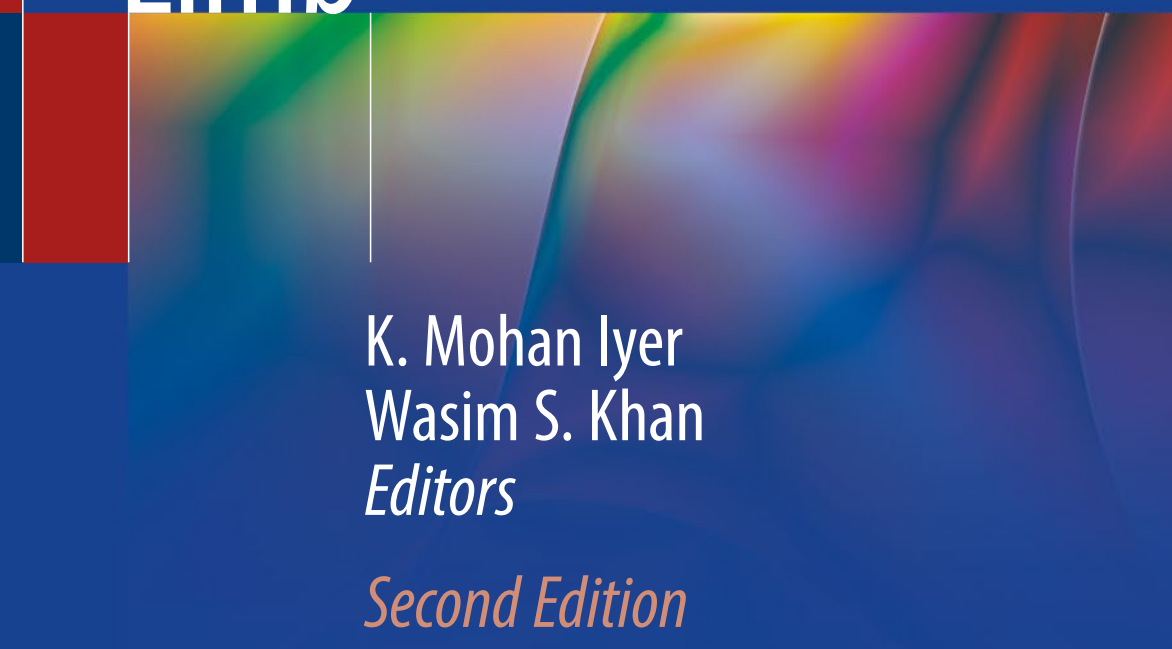
Orthopedics of the Upper and Lower Limb



K. Mohan Iyer
Wasim S. Khan
Editors

Second Edition

Springer

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K. Mohan Iyer • Wasim S. Khan
Editors

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I have written this dedication with a very heavy mind full of fond memories for my late respected teacher, Mr. Geoffrey V. Osborne, without whose constant encouragement and freedom I could not have written this book. Such teachers are extremely rare to spot these days where the turmoil of daily life overtakes one's ambitions, duties, and career aspirations. I have a remarkable store of personal and academic memories of him, with whom I had spent 4 long years at the University of Liverpool, UK, during which time I rarely looked upon him as my teacher, as he was more of a close friend and father to me.

To the memory of my respected teacher
(Late) Mr. Geoffrey V. Osborne
And loving thanks to
My wife, Mrs. Nalini K. Mohan
My Daughter, Deepa Iyer, MBBS, MRCP (UK), FAFRM(RACP)
My Son-in-Law, Kanishka B
My son, Rohit Iyer, BE (IT)
My Daughter-in-Law, Deepti B.U
My grandsons, Vihaan and Kiaan

Foreword

The principles of musculoskeletal medicine have been around for quite a while and date back to a time when we only had our five senses to rely on. The "look, feel, move" paradigm is engrained in the minds of us "simpleton orthopods," and allows us to be safe, efficient, and effective, guiding us to formulate accurate diagnosis and appropriate management, and to objectively assess whether our proposed management is working.

Techniques in trauma and orthopedic surgery are evolving, but the principles at the basis of our discipline remain unchanged. General principles should stay with us forever, and this book shows very clearly how we should proceed in the journey to examine our patients, formulate a diagnosis, plan management, and remember that it all started from the simple "look, feel, move" mantra.

Dr. K. Mohan Iyer and Mr. Wasim S. Khan have worked jolly hard on this second edition, especially considering that the first edition was not out of date. This second edition maintains the general feel of the first one and introduces the latest advances in our art. It is stimulating and allows the readers to open their eyes to new horizons: the chapters on knee resurfacing arthroplasty, navigations, and orthobiologics are just a few of the novel concepts in orthopedics.

The two editors have succeeded in improving on an already excellent work: Hats off to them and to the writing team, embracing several countries and a lot of teaching centers. Herculean task, great rewards, not necessarily bedtime reading!

Salerno, Italy
London, UK
Stoke on Trent, UK

Nicola Maffulli

Foreword for the First Edition/Orthopedics of the Upper and Lower Limb, by K. Mohan Iyer

Several decades ago, I was fortunate to hear Dr. K. Mohan Iyer speak about a limited, posterior greater trochanteric osteotomy as an adjunct to a posterior approach to the hip. Since then, I have used this method, as it allows easy access to the hip and joint replacement and then a secure posterior capsule and short external rotator muscle repair upon joint closure. Postoperative dislocations ceased to be an issue. As you might recognize, I have looked forward to additional contributions from Dr. Iyer, and here we have it—*General Principles of Orthopedics and Trauma*, *Orthopedics of the Upper and Lower Limb and Trauma Management in Orthopedics* (Springer). What a huge task to organize such books! Deciding on the material to be included, writing multiple chapters, and asking for skilled contributors who will embrace the challenge and have the talents to write either a general or subspecialty chapter. The text is aimed at the newcomer to this field of medicine, and it will serve that purpose quite well. I have always felt the best approach to learning orthopedic surgery is to read, cover to cover, a text such as this, aggressively study anatomy, read about the problems in the patients under one's care, read subspecialty texts, and read at least the abstracts in selected journals. By doing these things, one can be an educated person in the field—but it starts with the basic text!

In addition to the fundamentals, Dr. Iyer has added details about trauma and regional orthopedics. A cad has said only two types of doctors are necessary, and the others are optional. One of these is a physician who cares for broken bones. Details about fractures are essential to the field and to humanistic patient care. The regional

chapters serve as a transition to the later reading about each anatomic region in detail; this will be required to become an orthopedic surgeon.

So there you have it. An editor who is an energetic, dedicated scholar and teacher. Plus, the type of textbook most needed to jump into the field of musculoskeletal medicine and surgery. Learning is a joy. Lucky readers enjoy the intellectual journey.

Rochester, MN, USA Robert H. Cofield

Preface

I have written this preface dedicated to the memory of late Mr. G. V. Osborne, without whose constant support and encouragement I would never have written this book, which is in its second edition today. In addition to him, my family is now complete with son-in-law and daughter-in-law being the latest additions to the growing family together with two grandsons.

I have been religiously following the hip joint right from the days when I was with Mr. Osborne for more than 40 years till today. In fact, I am embarking on this book *The Hip Joint* (second edition) due to be published by Jenny Stanford Publishing (Singapore) which should be complete by this year, 2020. The first edition of this book was published by Pan Stanford Publishing (Singapore) in 2016 wherein it received two reviews by EJOST (European Journal of Orthopedic Surgery and Traumatology) (Springer) and Orthopedic News Update or Orthogate which prompted me to write the second edition of this book. This book *Orthopedics of the Upper and Lower Limb* (second edition) by K. Mohan Iyer and Wasim S. Khan which is in the second edition and published by Springer Nature, Switzerland, has totally 39 chapters in detail.

I am grateful to Jose Tharayil and his colleagues at Kochi, Kerala, for writing Chap.8 and Muhammad Zahid Saeed, UK, for Total Knee Replacement (Chap. 18) for their timely help , at short notice. I am very thankful to my colleague Dr. Kandiah Raveendran, MCh Orth (Liverpool, UK), FRCS who has written an original chapter not found in any other orthopedic books till today. He was the President of the International Society of Medical Shockwave Treatment (ISMST), Past Vice President of SICOT, Founder member of the sports medicine section of APOA, National Delegate of the APKS (Asia Pacific Knee Society), and a Consultant Orthopedic Surgeon, Ipoh, Malaysia. In addition, I also have an interesting chapter, Musculoskeletal Injuries of Lower Limb in Military, written by Ed Sellon and Duncan Goodall. I have a novel chapter, Pediatric Knee Injuries, by Humza T Osmani and Ioannis Pengas, who is a Consultant Orthopedic Surgeon at the Royal Cornwall Hospitals NHS Trust·Trauma and Orthopedics. There is also an interesting and stimulating chapter, Knee Surfacing Arthroplasty, by Sankalp Mutha, Mumbai, India.

I thank Dr. Maninder Shah Singh who could organize the 32nd Annual Conference of the Indian Foot & Ankle Society (IFASCON) 2019, New Delhi [IFASCON 2019].

He also presented a video on SCARF osteotomy on 31/8/2019 in addition to presenting a paper titled FHL transfer for degenerative acute achilles avulsions on 31/8/2019.

Above all, he wrote and supervised Chap. 30 for this book. The Foot and Great Toe, by Maninder Shah Singh, along with Darshan Mahesh Kapoor and Shyam Ashok Katekar.

In this chapter which is very well documented, Ritika Sharma, Bachelor of Physiotherapy, Third Year, Indian Spinal Injuries Centre, New Delhi, has been acknowledged for the 12 line diagrams which have been done with immense patience.

I give full credit to Maneesh Bhatia, Consultant Foot and Ankle Surgeon, University Hospitals, Leicester, UK; Education Secretary of British Indian Orthopedic Society (BIOS) Scientific Committee Member BOFAS, presented a paper titled Current concepts in syndesmotic injuries on 31/8/2019 in addition to being a moderator—Neuropathic Foot & Ankle on 31/8/2019. Above all, he presented an excellent paper titled Arthroscopic ankle arthrodesis on 1/9/2019—indications and techniques.

Above all he could write, supervise, and contribute three important chapters as follows for this book, namely:

1. Chapter 26. Clinical Examination of the Foot and Ankle (Basic and Surface Anatomy) with Special Tests, by Maneesh Bhatia and Sheweidin Aziz. In this chapter, he has made sure that the basics of clinical examination of the foot and ankle has been well documented by special clinical photographed taken by himself as an examiner specially for this chapter to make it very clear to the target audience the importance of this expanding subspecialty in orthopedics.
2. Chapter 27. Modern Management of Ankle Arthritis Including Primary Ankle Replacement by Maneesh Bhatia with Randeep Aujla who embarked on a one-year Fellowship on Soft tissue Knee and Sports Surgery Fellowship at Perth Orthopedic and Sports Medicine Centre, Australia, for the year 2020.
3. Chapter 37. Lisfranc Injuries, by Maneesh Bhatia and Lauren Thomson.

This book was slightly delayed mainly due to the sudden unexpected change of authors in the Table of Contents and rearranging them at the last minute though having been completed in time by the end of the year 2019.

Also I had a memorable personal event in its dedications of the marriage of my son during the build up to this book.

Acknowledgements

I am extremely grateful and obliged to Mr. Wasim S. Khan, MBChB, MRCS, Dip Clin Ed, MSc, PhD, FRCS (Tr & Orth), University Lecturer, University of Cambridge, Honorary Consultant Trauma and Orthopedic Surgeon, Addenbrooke's Hospital, Cambridge, UK, for joining me in this book as there are plenty of new and rare chapters added to this new second edition.

I am extremely grateful to Mr. Magdi E. Greiss, MD, MCh Orth, FRCS, Senior Consultant Orthopedic Surgeon, North Cumbria university Hospitals, UK, Former President, BOFAS, UK, for his invaluable snaps which he had preserved in his life during his training and early years after becoming a consultant in orthopedics.

I am also thankful to Dr. Rajesh Botchu, MBBS, MS (Orthopedics) MRCSI, MRCS Ed, FRCR, Consultant Musculoskeletal Radiologist, The Royal Orthopedic Hospital, Birmingham, UK, for his permission to use certain X-rays, MRI images, etc. for publication in this book.

I am particularly thankful to Michele Sparer and Shannon Collins for their permission to use certain images of Strykar for Chap. 31 in this book.

Above all, I am extremely thankful to Mr. Anand Shanmugam, Project Coordinator (Books) for Springer Nature for his help in formatting and publishing the entire book. I particularly extend my thanks to him for his patience in making a list of the contributory authors with corresponding authors for each chapter.

Also I would like to thank Mr. Mohan Kumar, Graphic Designer, Bangalore, India, for his immense help in line diagrams used in this book which have been sketched by using an illustrative tool.

Above all, I highly appreciate the help of my son, Mr. Rohit Iyer, in the presentation and publication of this book.

Bengaluru, Karnataka, India K. Mohan Iyer

Preface

I was honored and humbled when asked by Dr. K. Mohan Iyer to join him in the mammoth task of preparing a well-timed second edition to the hugely successful first edition of the textbook *Orthopedics of the Upper and Lower Limb*.

The management of orthopedics is based on well-established principles but the practice is continuing to evolve with newer developments and technologies. It is important that textbooks are updated to keep up with these changes. This 39-chapter second edition updates the previous text, the first edition, and covers a greater range of topics that have come to the fore of orthopedic practice over the years. The methodological layout, logical flow, easy-to-follow text, and use of figures should help the reader get the most out of this textbook and should appeal to all from medical students and trainees to allied health care professionals and subspecialists.

Acknowledgements

The task of completing the second edition was made easier by the strong unrelenting work ethics of Dr. K. Mohan Iyer and the guidance of Mr. Anand Shanmugam (Springer Nature), and I gratefully acknowledge their support. I am also grateful to one of my mentors, Prof. Nicola Maffulli for providing the Foreword to this second edition. I am grateful to all authors, including the Orthopedic Research Collaborative East Anglia (ORCA) members who have contributed 13 chapters. Above all, I would like to acknowledge our patients who we all strive to do the best for, and we hope this book goes some way in this noble quest.

Cambridge, UK

Wasim S. Khan

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Part I

The Upper Limb

Clinical Examination of the Shoulder (Basic and Surface Anatomy) with Special Tests

1

K. Mohan Iyer

The shoulder joint is made up of four bones, namely, the scapula, clavicle, humeral head, and posterior rib cage. It also consists of four joints, namely, the sternoclavicular, acromioclavicular, glenohumeral, and scapulothoracic joints.

The shoulder girdle is made up of two bones, namely, the scapula and the clavicle, along with three joints namely, the glenohumeral, acromioclavicular, and sternoclavicular joints. The sternoclavicular joint is of synovial and saddle variety. The acromioclavicular joint is of synovial and plane variety. The main glenohumeral synovial is multiaxial and of the ball and socket variety.

Ossification of the Bones of the Shoulder

1. Clavicle: It is the first bone to ossify. It has no medullary cavity. It occurs by intramembranous ossification. Secondary ossification centers via endochondral. The medial epiphysis ossifies at 12–19 years and fuses at 22–25 years. The lateral epiphysis ossifies and fuses at 19 years.
2. Scapula: The body and spine (posterior) ossify at birth. The coracoid process (anterior) atavistic epiphysis center at 1 year, base at 10 years. The acromion (lateral projection) fuses by 22 years via a 2–5-center form at puberty. The glenoid upper 1/4 ossifies at 10 years while the lower 3/4 appears at puberty and fuses by 22 years.
3. Proximal humerus: The humeral head ossifies at 6 months while the greater tuberosity ossifies at 1–3 years and the lesser tuberosity ossifies at 4–5 years. All physes close at 14–17 years in girls and 16–18 years in boys.

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The ligaments of the sternoclavicular joint are mainly the (1) capsular ligament, (2) sternoclavicular ligament (anterior and posterior), (3) interclavicular ligament, (4) costoclavicular ligament (consisting of the anterior lamina and the posterior lamina), and (5) articular disk (mainly fibrocartilaginous).

The ligaments of the acromioclavicular joint are made up of the (1) fibrous capsule; (2) acromioclavicular ligament; (3) coracoclavicular ligament which is made up of two parts, namely, conoid and trapezoid; and (4) coracoacromial ligament.

The ligaments of the shoulder joint (glenohumeral joint) are made up of the (1) fibrous capsule; (2) glenohumeral ligament consisting of the superior band, middle band, and inferior band; (3) coracohumeral ligament; and (4) transverse humeral ligament.

The glenohumeral joint is made up of 25% humeral head surface in contact with the glenoid, and this joint space thinning is seen with OA of the shoulder. The humeral head coverage is increased to 75% with the glenoid labrum. The glenoid labrum has a (1) fibrocartilaginous rim and is (2) triangular in cross section, which is (3) attached to the peripheral margin of the glenoid cavity except above. (4) It also deepens the glenoid fossa and forms a pliable cushion for ball to roll.

Relations of the Joint

1. Above—Deltoid, supraspinatus, subacromial bursa, and coracoacromial arch.
2. Below—Quadrangular space transmitting axillary nerve, posterior circumflex humeral vessels, and long head of the triceps.
3. In front—Subscapularis, coracobrachialis, and short head of the biceps.
4. Behind—Infraspinatus and teres minor.
5. Within capsule—Long head of the biceps.
6. The deltoid muscle covers the joint in front, behind, and laterally.
7. The long head of the biceps originates from the supraglenoid tubercle of the scapula. It is intracapsular but extrasynovial. The tendon passes through the shoulder joint and emerges below the transverse humeral ligament inside the joint; the tendon is surrounded by a separate tubular sheath of the synovial capsule (Fig. 1.1).

Rotator Cuff Muscles

1. Supraspinatus, infraspinatus, teres minor, and subscapularis which form a cuff around the humeral head.
2. Keep the humeral head within the joint (counteract deltoid).
3. Abduction, external rotation, and internal rotation.

Functions of the Rotator Cuff Muscles

1. The four major muscles of the rotator cuff rotate the humerus and properly orient the humeral head in the glenoid fossa (socket).

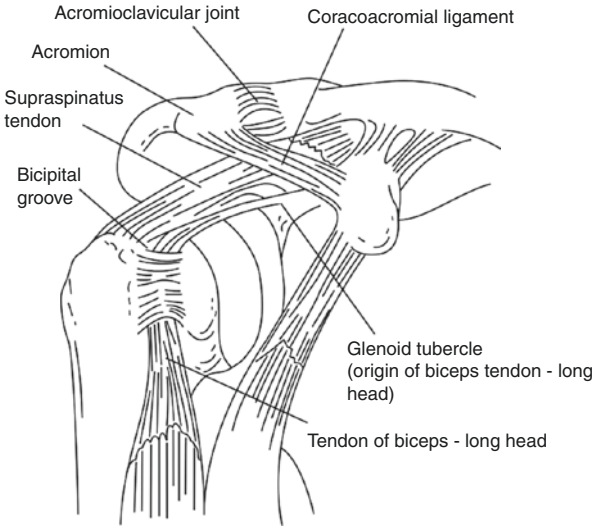

Fig. 1.1 Line diagram of the front of the shoulder

2. The tendons of these four muscles merge, forming a cuff around the glenohumeral joint.
3. Supraspinatus: abducts the humeral head and acts as a humeral head depressor.
4. Infraspinatus: externally rotates and horizontally extends the humerus.
5. Teres minor: externally rotates and extends the humerus.
6. Subscapularis: internally rotates the humerus.

Factors maintaining the stability of the shoulder joint are as follows:

(a) The glenoid labrum deepens the socket.
(b) The supraspinatus, tension of the upper part of the capsule, and coracohumeral ligament prevent its downward displacement.
(c) Tendons of the subscapularis, supraspinatus, infraspinatus, and teres minor blend with the fibrous capsule, form the musculotendinous rotator cuff, and act as guardians of the joint.
(d) The long head of the biceps and coracoacromial arch prevent upward dislocation of the humerus.

Bursae in Relation to the Shoulder Joint

(a) Communicating—Subscapular bursa and infraspinatus bursa.
(b) Noncommunicating—(1) Subacromial (largest bursa of the body), (2) above the acromion process, (3) between the capsule and coracoid process, (4) behind the coracobrachialis, (5) between the teres minor and long head of the triceps, and (6) in front of and behind the tendon of the latissimus dorsi.

Blood Supply and Nerve Supply

1. Vascular supply—Anterior and posterior circumflex humeral, suprascapular, and circumflex scapular vessels.
2. Nerve supply—The capsule is supplied by the suprascapular nerve (posterior and superior parts), axillary nerve (anteroinferior), and lateral pectoral nerve.

Movements of the Shoulder

They are (1) flexion, (2) extension, (3) abduction, (4) adduction, (5) external rotation, (6) internal rotation, and (7) circumduction.

The most important aspect is the plane of movement, such as (1) abduction and adduction occur at the plane of the scapula and (2) flexion and extension occur 90° to the plane of the scapula.

The three mutually perpendicular axes around which the principal movements of (a) flexion-extension, (b) abduction/adduction, and (c) medial and lateral rotation occur at the shoulder.

1. Flexion: It is a 90° movement and the muscles involved are the (a) deltoid (anterior fibers), (b) pectoralis major (clavicular fibers), (c) coracobrachialis, and (d) biceps.
2. Extension: It is of 45° and the muscles involved are the (a) deltoid (posterior fibers), (b) teres major, (c) latissimus dorsi, and (d) pectoralis major (sternocostal fibers).
3. Adduction: It is of 45° and the muscles involved are the (a) pectoralis major, (b) latissimus dorsi, (c) teres major, and (d) coracobrachialis.
4. Abduction: It is of 180° and the muscles involved are the (a) supraspinatus, (b) deltoid, (c) serratus anterior, (d) infraspinatus, and (E) trapezius.
5. External rotation: It is 80°–90° and the muscles involved are the (a) infraspinatus, (b) teres minor, and (c) deltoid (posterior fibers).
6. Internal rotation: It is 55° and the muscles involved are the (a) subscapularis, (b) pectoralis major, (c) latissimus dorsi, (d) teres major, and (E) deltoid (anterior fibers).
7. Circumduction: This is a movement in which the distal end of the humerus moves in circular motion while the proximal end remains stable. It is formed by flexion, abduction, extension, and adduction, successively.

Special Tests for the Shoulder

Stability of the Shoulder

1. Apprehension (Crank) test: With the patient seated, the shoulder is abducted 90° and externally rotated; a positive sign is one of pain and apprehension and

is indicative of traumatic instability problems. It is elicited by slowly applying lateral rotation on the shoulder.

2. Jobe relocation test (Fowler sign): With the patient supine, the shoulder is abducted 90° and externally rotated when a positive sign is seen as a relief of pain and apprehension and is indicative of posterior internal impingement/traumatic instability problems. It is elicited by applying a posteriorly directed force to the head of the humerus when further external rotation becomes possible. It is also called the Fowler sign.
3. Rockwood test: It is done with the patient sitting. A positive sign marks apprehension at 90° with some uneasiness and pain at 45° and 120°. It is indicative of anterior instability. It is elicited by stabilizing the elbow and humerus and then grasping the humeral head and lifting it forward.
4. Rowe test for anterior instability: It is done with the patient supine. The arm is abducted 80–120°, flexed 20°, and externally rotated 30°, with a hand on the patient's axilla when a positive sign of apprehension (pain) or a click sound is heard indicative of anterior instability and/or anterior labral tear. It is elicited by stabilizing the scapula, when pushing the spine forward using the index and middle fingers. Apply a counterpressure on the coracoid and then draw the humerus forward.
5. Rowe test for multidirectional instability: The patient stands forward flexed 45° at the waist with arms pointing to the floor. A positive sign is a sulcus sign which is indicative of multidirectional instability. It is elicited as follows: place the hand on the patient's shoulder with the index and middle fingers (anterior) and the thumb (posterior). The signs of multidirectional instability are seen anteriorly when the shoulder is extended 20–30° and then pushed anteriorly, posteriorly when the shoulder is flexed 20–30° and then pushed posteriorly, and inferiorly when the shoulder is flexed 20°–30° and then pushed posteriorly; apply traction [1].
6. Fulcrum test: With the patient supine, the shoulder is abducted 90° and externally rotated when a positive sign is pain and apprehension which is indicative of traumatic instability problems. It is elicited by placing a hand under the glenohumeral joint and then applying lateral rotation. It is also a modification of the crank test.
7. Anterior drawer test: It is done with the patient supine. The arm is abducted 80–120°, flexed 20°, and externally rotated 30°, with a hand on the patient's axilla when a positive sign of apprehension (pain) or a click sound is heard indicative of anterior instability and/or anterior labral tear. It is elicited by stabilizing the scapula, when pushing the spine forward using the index and middle fingers. Apply a counterpressure on the coracoid and then draw the humerus forward.
8. Posterior apprehension test: This is done with the patient supine, when a positive sign of apprehension is experienced when the shoulder is elevated to 90° indicative of posterior shoulder instability. It is elicited by applying a posterior force on the elbow and then horizontally adducting and internally rotating the shoulder. It is also called a stress test.

9. Feagin test: It is done with the patient standing when the shoulder is abducted 90° on the physiotherapist's shoulder. This is positive when there is a presence of sulcus on the coracoid process or apprehension which is indicative of multidirectional instability. It is elicited by closing hands over the humerus and pushing down and forward.
10. Clunk test: This is done with the patient supine when a positive sign is heard as a clunk/grinding sound indicative of a tear of the labrum (Bankart). It is elicited as follows: place one hand on the posterior aspect of the shoulder, while one hand holds the humerus above the elbow. Fully abduct the arm over the patient's head. Push anteriorly with the hand over the humeral head (place a fist under the glenohumeral joint) when the other hand rotates the humerus laterally.
11. Compression rotation test: With the patient supine and elbow flexed and abducted 20°, the examiner pushes up on the elbow and rotates the humerus medially and laterally. Snapping or catching is positive for labral tear.

Biceps Tests

1. Speed's test: This is done with the patient standing. A positive sign is pain on the bicipital groove with weakness which is indicative of bicipital tendonitis or superior labrum anterior to posterior (SLAP) II or a rupture of the biceps (Fig. 1.2). It is elicited as follows: resist shoulder extension by the patient first in supination and then in pronation with elbow extension. (The other names for this test are biceps test and straight arm test.)
2. Yergason's test: This done with the patient sitting/standing. The elbow is at 90° with the forearm pronated when a positive sign is indicated by pain/tenderness or popping out of the groove which indicates bicipital tendonitis or a torn transverse humeral ligament. It is elicited when there is resistance to supination while the patient externally rotates the shoulder (Fig. 1.3) [2].
3. Biceps saw test: Patient flexes elbow to 90° and places fist in the examiner's hand. The patient's fist is forced downward while making a saw motion FE to and from −30 and +30. Pain in the bicipital groove is suggestive of bicipital tendinitis.
4. Biceps tension test: This done with the patient standing, the shoulder abducted 90°, the elbow extended, and the forearm supinated when a positive sign is indicated by reproduction of symptoms and is indicative of a SLAP lesion. It can be elicited by applying an eccentric adduction force.
5. Biceps load test: This is done with the patient supine. The shoulder is abducted 90° and externally rotated, the elbow flexed 90°, and the forearm supinated; a positive test is indicated when the apprehension does not disappear. This is diagnostic of the integrity of the superior labrum. It is elicited as follows: fully externally rotate the shoulder until apprehension, stop external rotation, and hold the position. Then the patient resists elbow flexion at the wrist.

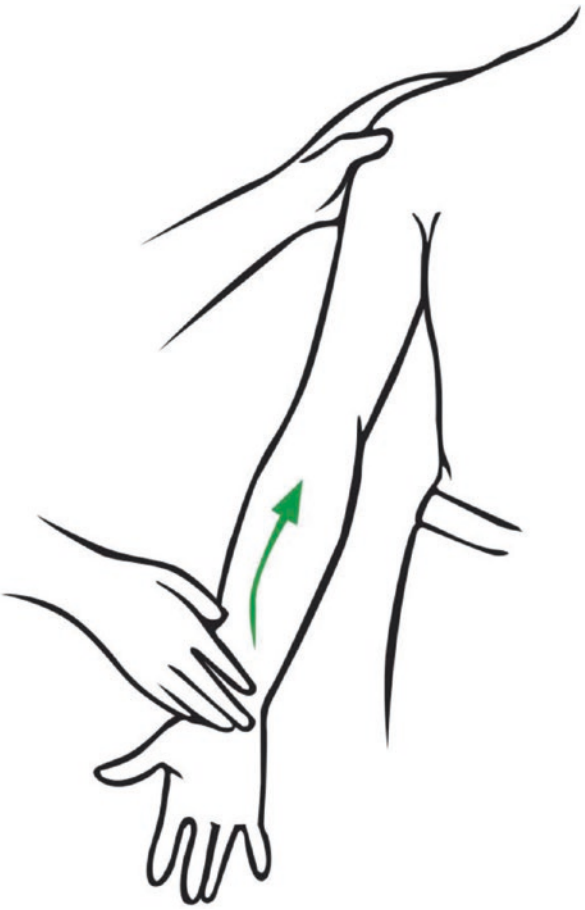

Fig. 1.2 Line diagram of speed's test

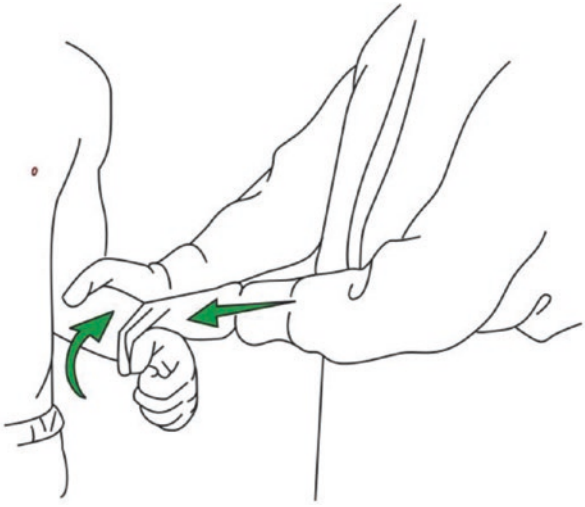

Fig. 1.3 Line diagram of Yergason's test

Impingement Tests

1. Neer impingement test: This is done with the patient sitting when a (+) sign is pain which indicates overuse injury to the supraspinatus muscle. It can be elicited when the patient's arm is passively and forcibly fully elevated and the shoulder is internally rotated (Fig. 1.4).
2. Hawkins-Kennedy impingement test: This is done with the patient standing or sitting, when a (+) sign is pain, which is indicative of supraspinatus tendonitis. It can be elicited by flexing the shoulder to 90° and then medially rotating the shoulder (Fig. 1.5).
3. Impingement test: The arm is abducted to 90° with a full lateral rotation; it is positive if painful.

Other Tests

1. Scapular thoracic glide test (lateral scapular slide test): This is done with the patient sitting/standing with arms at the side. The sign is positive when there is

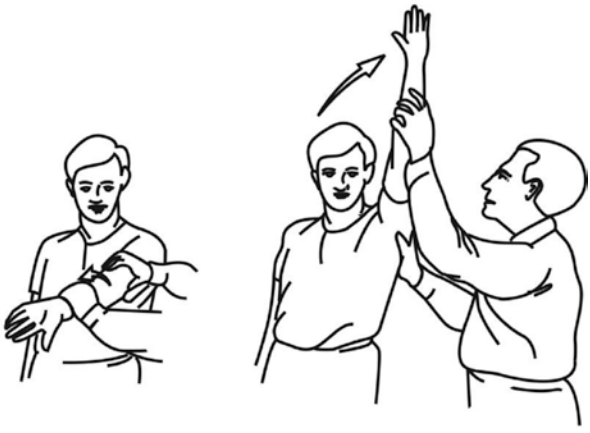

Fig. 1.4 Line diagram of Neer test

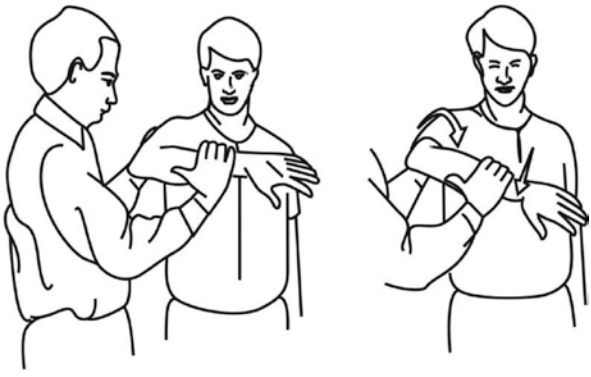

Fig. 1.5 Line diagram of the Hawkins-Kennedy test

>1–1.5 cm difference from the original measure which is indicative of a scapular instability. To elicit this, measure the distance from the spine to the scapula to T2/T3, inferior angle to T7–T9 or superior angle to T2. (Also test the patient with shoulder abduction: 45°, 90°, 120°, and 150°.)

2. O'Brien test: Patient flexes arm to 90° with the elbow fully extended and then adducts the arm 10–15° medially to the sagittal plane. Maximal pronation with FE against resistance—repeated in supination. Pain with pronation or no supination is AC or labral lesion, respectively.
3. Codman's (drop arm) test: This is done with the patient standing and the shoulder abducted 90° when a (+) sign is an inability to return the arm to the side slowly, indicative of a rotator cuff tear. It is elicited by asking the patient to slowly lower arms to the side with some arc movements. It is also known as Codman's test.
4. Military brace (costoclavicular syndrome) test: Palpate the radial pulse as the shoulder is drawn down and back. It is positive if the pulse is decreased which indicates possible thoracic outlet syndrome.
5. Adson maneuver: It is done with the patient sitting with the head on the ipsilateral side. A (+) sign: disappearance of pulse indicative of thoracic outlet syndrome. It can be elicited as follows: locate the radial pulse, externally rotate and extend the shoulder, and instruct the patient to take a deep breath and hold it.
6. Allen test: This is done with the patient sitting with the head on the contralateral side. A (+) sign: disappearance of pulse indicative of thoracic outlet syndrome. It can be elicited as follows: the elbow is flexed to 90°, the shoulder is extended and externally rotated horizontally, and palpate the radial side.
7. Halstead maneuver: This is done when the neck is hyperextended and rotated to the contralateral side. A (+) sign of disappearance of the radial pulse is indicative of thoracic outlet syndrome. It can be elicited as follows: find the radial pulse, and apply downward traction on the extremity.
8. Tinel's sign at the shoulder: It is done with the patient sitting. A (+) sign of tingling sensation is indicative of a peripheral nerve injury. It can be elicited by tapping on the scalene triangle in the area of the brachial plexus.
9. Yocum test: This is done with the patient standing or sitting. A (+) sign of pain is indicative of supraspinatus tendonitis. It can be elicited when the patient places a hand on the opposite shoulder and then the physiotherapist elevates the elbow. It is also a modification of the Hawkins-Kennedy test.
10. Coracoid impingement test: This is done with the patient standing or sitting when a (+) sign is pain, indicative of supraspinatus tendonitis. It can be elicited by flexing to 90°, horizontally adducting to 10–20°, and then medially rotating the shoulder. It is also known as a modification of the Hawkins–Kennedy test.
11. Lift-off sign: This is done with the patient standing and the dorsum of the hand on the back pocket. A (+) sign is an inability to lift the hand off back, which is indicative of a subscapularis lesion. It can be elicited by asking the patient to lift the hand away from the back.
12. Pectoralis major contracture test: This is done with the patient supine and his hands clasped behind the head, when a (+) sign is that the elbows do not reach

the table which is indicative of a tight pectoralis major. It is elicited by lowering the arm until elbows touch the table.

13. Teres minor test: This is done with the patient prone, with one hand on the iliac crest, when a (+) sign of pain and weakness is indicative of a teres minor strain. This can be elicited by asking the patient to extend and adduct the shoulder against resistance.
14. Infraspinatus test: It is done with the patient standing with the arm on the side with the elbow flexed to 90°, when a (+) sign is seen as pain with an inability to resist internal rotation. This is indicative of an infraspinatus strain. It can be elicited by applying an internal rotation force that the patient resists.
15. Hornblower's test: This is done with the patient standing, the shoulder flexed to 90°, and the elbow flexed to 90°, when a (+) sign is an inability to externally rotate the shoulder, which is indicative of a tear on the teres minor. It is elicited when the patient externally rotates with resistance.
16. Lateral rotation lag sign: It is done with the patient seated or standing and with the arms at the side. A (+) sign is when the patient cannot hold the position due to pain with an increased internal rotation on the affected side. This is indicative of a torn supraspinatus, infraspinatus, and subscapularis. This can be elicited as follows: when the patient passively abducts shoulder to 90°, the elbow is flexed to 90° and externally rotated when the patients holds the position. This is also called the infraspinatus "spring back" test.
17. Supraspinatus test: This is done with the patient standing when the shoulder is abducted 90°. A (+) sign: pain or weakness is felt indicative of a torn supraspinatus or neuropathy of the suprascapular nerve. It is elicited when the shoulder is internally rotated and angled forward 30° and the thumb is pointing to the floor, and then resist. It is also called the empty can test or Jobe test.
18. Heuter's sign: This is done with the patient sitting and the forearm pronated. A (+) sign is the absence of elbow supination which is indicative of a ruptured distal biceps tendon. It is elicited by resistance to elbow flexion with the forearm pronated.
19. Lippman's test: The patient is sitting/standing. A positive sign is a sharp pain on the bicipital groove indicative of bicipital tendonitis. It is elicited as follows: hold the patient's arm and flex to 90° with one hand, while the other hand palpates the biceps tendon 7–8 cm below the glenohumeral joint. Then move the biceps tendon side to side.
20. Gilchrest's test: It is done with the patient standing. A positive sign is pain on the bicipital groove indicative of bicipital paratendonitis. It is elicited as follows: ask the patient to lift 2–3 kg/5–7 lb of weight over the head with the arm in external rotation.
21. Ludington's test: This is done with the patient sitting and hands clasped behind the head. A positive sign indicated by no contraction evident/palpable is indicative of a torn long head of the biceps. It is elicited by asking the patient to contract the biceps.
22. Ellman's compression rotary test: This is done with the patient lying on the unaffected side, when a positive sign of pain reproduction is indicative of

glenohumeral arthritis. This can be elicited by compressing the humeral head while the patient rotates the shoulder medially and laterally.

23. Acromioclavicular shear test: This is done with the patient sitting. The sign is positive with an abnormal movement of at the AC joint indicative of acromioclavicular joint pathology. It is elicited as follows: cup hands over the deltoid, one on the clavicle and one on the scapula. Squeeze both hands together.
24. Wall push-up test: This is done with the patient standing, at arm's length on the wall. A positive sign is indicated by winging within 5–10 reps of push-up. This is indicative of weakness of scapular muscles. This is elicited by asking the patient to do 15–20 wall push-ups.
25. Closed kinetic chain upper extremity stability test: This is done with the patient prone, on the floor at arm's length with hands 36 in. apart. The positive sign is winging of the scapula which is indicative of weakness of scapular muscles. This can be elicited as follows: the patient touches the other hand and then returns to the original position. This is done for 15 s while the physiotherapist counts how many reps the patient is able to do.
26. SLAP prehension test: This is done with the patient sitting. The sign is positive when it is first painful, followed by second relief of pain, which is indicative of a SLAP lesion. It is elicited as follows: the patient actively abducts the shoulder 90°; the forearm is pronated and then horizontally adducted. Then the shoulder is abducted 90°; the forearm is supinated and horizontally adducted.
27. Inferior shoulder instability test: This is done with the patient standing relaxed. A positive sign is seen as a sulcus sign (+1 $\leq$ 1 cm, +2 = 1–2 cm, +3 $\geq$ 2 cm) which is indicative of inferior instability/glenohumeral laxity. It is elicited by grasping the patient's elbow and then pulling it distally. It is also known as a sulcus sign.
28. Jerk test: This is done with the patient sitting when the shoulder is flexed 90° and internally rotated, and a positive sign is seen as a sudden jerk or clunk, indicative of recurrent posterior instability. It is elicited as follows: grasp the patient's elbow and axially load the humerus proximally. Maintain the axial load and then move the arm to a horizontal arm to horizontal adduction with internal rotation.
29. Push-pull test: This is done with the patient supine. A positive sign of >50% translation and pain/apprehension is seen when the shoulder is abducted 90° and flexed 30°, and this is indicative of posterior instability. It is elicited by holding the patient's arm on the wrist and humerus with a pull on the arm at the wrist while pushing down on the humerus with the other hand.
30. Dugas test: This is done with the patient sitting. A positive sign is seen as pain/inability to do the command of the physiotherapist which is indicative of anterior dislocation. It is elicited by asking the patient to place one hand on the opposite shoulder and to lower the elbow to the chest [3].
31. Protzman test: It is done with the patient sitting. The arm is abducted 90°, supported on the patient's hip when a positive sign of pain is experienced indicative of anterior instability. It is elicited by palpating the anterior head with one hand, while the other hand is on the patient's axilla and the humerus is pushed anteriorly and inferiorly [4].

32. Surprise test: It is done with the patient supine. A positive sign of pain and forward translation of the humeral head when the shoulder is abducted 90° and externally rotated is indicative of traumatic instability problems. It can be elicited by performing "Fowler sign"; after further external rotation, release the posterior force. It is also called as anterior release test.
33. Load and shift test: This is done with the patient sitting relaxed on a chair.

    A positive sign may be as follows: (a) normal laxity = 1–25%, (b) grade 1 = head rides over the glenoid rim (25–50%), (c) grade 2 = head rides over the rim but reduces (>50%), and (d) grade 3 = head rides over the rim and remains dislocated.

    This test mainly indicates traumatic problems at the glenohumeral joint. It is done as follows: grasp the humeral head and stabilize the shoulder. Seat the humerus on the glenoid fossa and push it anteriorly and posteriorly to check for instability.
34. Tinel's sign: It is done with the patient sitting with the neck slightly flexed. A positive sign is indicated by localized pain indicative of a cervical plexus lesion. It is elicited by tapping the area of the brachial plexus with a finger along the nerve trunks [5, 6]. (It takes its name from French neurologist Jules Tinel [1879–1952].)
35. Brachial plexus compression test: This is done with the patient sitting. A positive test is indicated when the pain radiates into the shoulder and signifies mechanical cervical lesions having a mechanical component. This is done by applying firm compression to the brachial plexus by squeezing the plexus under the thumb or fingers.
36. Valsalva test (Valsalva maneuver): A positive sign of increased pain is indicative of increased intrathecal pressure. It is elicited when the patient takes a deep breath and holds it while bearing down, as if moving bowels [7]. (Physician Antonio Maria Valsalva first described the technique in the 1700s as a way to clear pus out of the ears.)
37. Scalene cramp test: This is also carried out with the patient sitting. A positive sign indicated by an increase in pain is indicative of thoracic outlet syndrome. It is elicited when the patient actively rotates the head to the affected side and pulls the chin down into the hollow above the clavicle by flexing the cervical spine [8].
38. Jackson's test: This is also done with the patient sitting. A positive sign indicated by pain radiating into the arm is indicative of cervical nerve root compression. It is done by rotating the patient's head to one side and applying downward pressure on the head.
39. Lhermitte's sign: This done with the patient in a long sitting position. A positive sign of pain radiating down the spine is indicative of dural or meningeal irritation. It is done by flexing the patient's head and one hip simultaneously while the leg is kept straight [9]. (Lhermitte's sign was first described by Marie and Chatelin in 1917. This sign is mostly described as an electric-shock-like condition by some patients of multiple sclerosis. This sensation occurs when the neck is moved in a wrong way or rather flexed. It can also travel down to the spine,

arms, and legs and sometimes the trunk. Demyelination and hyperexcitability are the main pathophysiological reasons depicted for Lhermitte's sign.)

40. Distraction test: This is done with the patient sitting. A positive sign indicated by relief of pain is indicative of pressure on the nerve roots. This is done by placing one hand under the patient's chin and the other around the occiput. Then slowly lift the head, by applying traction to the cervical spine.
41. Shoulder abduction test: This is also done with the patient sitting. A positive sign indicated by relief of symptoms is indicative of nerve root compression. This is done by abducting the patient's arm and then resting the hand or forearm on top of the head.
42. Shoulder depression test: This is done with the patient sitting. A positive test with increased pain is indicative of nerve root compression. This is done by side-flexing the patient's head on the unaffected side and then applying downward pressure on the opposite shoulder (affected side).
43. Naffziger's test: This is done with the patient sitting. A positive test is pain indicative of a nerve root problem or a space-occupying lesion. It is done by compressing the patient's jugular veins for 30 s and then asking the patient to cough [10].

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Shoulder Joint

2

K. Mohan Iyer

Dislocation/Recurrent/Habitual Dislocation of the Shoulder

Dislocation of the Shoulder

Shoulder dislocation is well documented in Egyptian tomb murals as early as 3000 BC, with depiction of manipulation for glenohumeral dislocation resembling Kocher's method. A painting in the tomb of Ipuy, 1300 BC, the sculptor of Ramesses II, shows a physician reducing a dislocated shoulder, using a similar technique.

Epidemiology

It occurs in approximately 1.7% of the population. It occurs in both sexes, men in 20–30 years (M:F 9:1) and women 61–80 years (M:F 1:3). It occurs less in children as their epiphyseal plate is weaker and tends to fracture before dislocating. It is seen more commonly in the elderly as their collagen fibers have fewer cross-links, hence a weaker capsule or tendons or ligaments.

Anatomy

Glenohumeral stability depends on both passive and active mechanisms:

1. Passive: such as joint conformity, vacuum effect of limited joint volume. Adhesion and cohesion owing to the presence of synovial fluid, scapular inclination of 0–30°, glenoid labrum.

   The bony restraints are the coracoid, acromion, and glenoid fossa, along with ligamentous and capsular restraints such as the joint capsule and superior, middle, and inferior glenohumeral ligaments along with the coracohumeral ligament.
2. Active: such as the biceps, long head, rotator cuff, and scapular stabilizing muscles.

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Pathoanatomy of Shoulder Dislocations

These may vary from (1) stretching or tearing of capsule, (2) avulsion of glenohumeral ligaments usually off the glenoid, (3) labral injury, (4) Bankart lesion, (5) impression fracture, (6) Hill-Sachs lesion, and (7) rotator cuff tear.

A Bankart lesion is usually (a) seen in anterior dislocation, with (b) stripping of the glenoid labrum along with the periosteum usually at the (c) anteroinferior surface of the glenoid and scapular neck.

The avulsion of the anteroinferior glenoid rim causes a bony Bankart lesion.

A Hill Sachs lesion is usually a (1) depression on the humeral head in its posterolateral quadrant caused (2) due to impingement by the anterior edge of the glenoid on the head as it dislocates.

Dislocation of the shoulder is usually seen as follows:

1. Mostly anterior in more than 95% of dislocations.
2. Posterior dislocation occurs in less than 5%.
3. True inferior dislocation (luxatio erecta) occurs in less than 1%.
4. Habitual: a nontraumatic dislocation may present as multidirectional dislocation due to generalized ligamentous laxity and is usually painless by nature.

An inferior dislocation is commonly referred to as luxatio erecta (Latin for "erect dislocation"), deriving its name from the classical presentation of an arm that is elevated and abducted and held overhead, unable to be lowered. The vast majority of cases of luxatio erecta will be successfully managed with closed reduction and postreduction immobilization. Cases of an irreducible inferior dislocation in the emergency room setting usually occur secondary to the humeral head buttonholing through the inferior capsule. Usually a beach chair position and a reduction utilizing a deltopectoral approach. Alternatively, one can use a strap incision to utilize both the deltopectoral interval and, if necessary, deltoid-splitting interval.

Mechanism of injury: The commonest is a fall on an outstretched hand with the shoulder abducted and externally rotated. Posterior dislocation is usually by a direct blow from the front of the shoulder or from epileptiform convulsions or electric shock.

Clinical Features

There is pain, and the patient holds the injured limb with the other hand close to the trunk. The shoulder is abducted and the elbow is kept flexed. There is a loss of the normal contour of the shoulder which appears as a step. The anterior bulge of the head of the humerus may be visible or palpable along with an empty glenoid socket.

Radiographic Evaluation

(a) Trauma series of the injured shoulder (Fig. 2.1a, b) includes:
   1. Anteroposterior (AP).
   2. Scapular-Y.
   3. Axillary views taken in the plane of the scapula. Velpeau axillary view (if a standard axillary cannot be obtained because of pain, the patient may be left

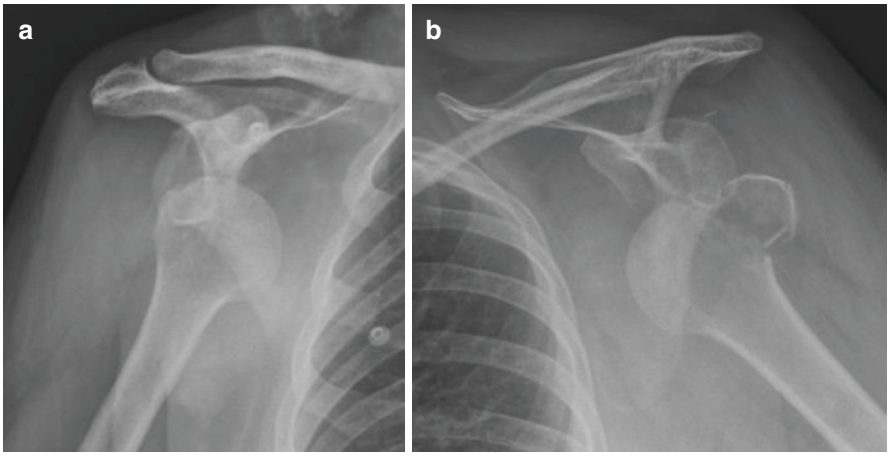

Fig. 2.1 (**a**) Anterior dislocation of the shoulder. (**b**) Anterior fracture dislocation of the shoulder. (Courtesy: Dr. Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopedic Hospital, Birmingham, UK)

in a sling and leaned obliquely backward 45° over the cassette. The beam is directed caudally, orthogonal to the cassette, resulting in an axillary view with magnification).

4. West Point axillary view (this is taken with the patient prone with the beam directed cephalad to the axilla 25° from the horizontal and 25° medial. It provides a tangential view of the anteroinferior glenoid rim).
5. Stryker notch view: Here the patient is supine with the ipsilateral palm on the crown of the head and the elbow pointing straight upward. The X-ray beam is directed 10° cephalad, aimed at the coracoid. This view can visualize 90% of posterolateral humeral head defects.

(b) Computed tomography may be useful in defining humeral head or glenoid impression fractures, loose bodies, and anterior labral bony injuries (bony Bankart lesion).
(c) Single- or double-contrast arthrography may be utilized to evaluate rotator cuff pathologic processes.
(d) Magnetic resonance imaging may be used to identify rotator cuff, capsular, and glenoid labral (Bankart lesion) pathologic processes.

Management

It is an emergency and should be reduced in less than 24 h or else AVN of the head of the humerus sets in. It is immobilized strapped to the trunk for 3–4 weeks and rested in a collar and cuff.

The reduction maneuvers available are as follows:

1. Traction–countertraction method
2. Hippocrates method

3. Stimson's technique
4. Kocher's technique
5. Milch technique
6. Scapular manipulation

- *Stimson's technique*: The patient is placed prone on the stretcher with the affected shoulder hanging off the edge. Weights (10–15 lb) are fastened to the wrist to provide gentle, constant traction (Fig. 2.2).
- *Kocher's maneuver*: It is carried out in four steps as follows: (1) traction with the elbow flexed at a right angle, with steady traction applied along the long axis of the humerus, (2) external rotation, (3) adduction, and (4) internal rotation.
- *Milch technique*: The arm is abducted and the physician's thumb is used to push the humeral head into its proper position. Gentle traction in line with the humerus is provided by the physician's opposite hand.
- *Scapular manipulation*: The patient sits upright and leans the unaffected shoulder against the stretcher. The physician stands behind the patient and palpates the

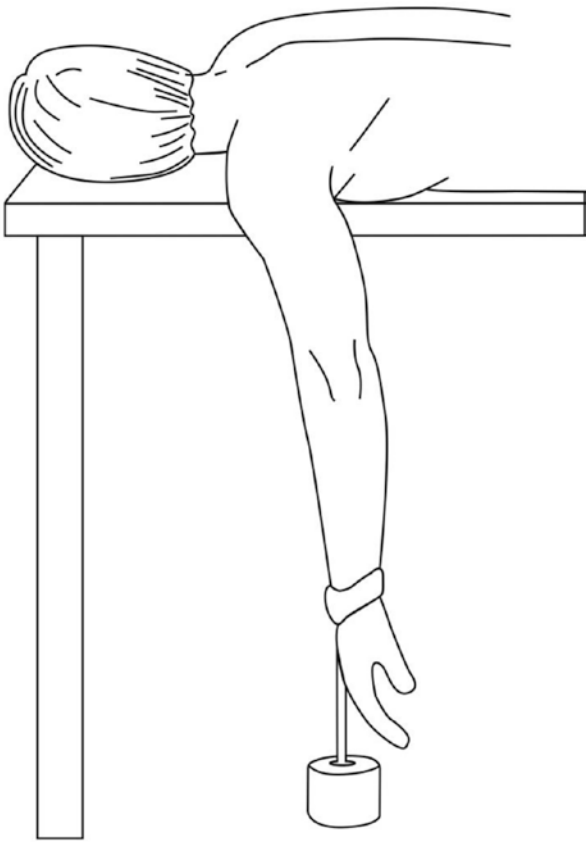

Fig. 2.2 Line diagram showing Stimson's technique

tip of the scapula with his thumbs and directs a force medially. The assistant stands in front of the patient and provides gentle downward traction on the humerus. The patient is encouraged to relax the shoulder as much as possible.

Postreduction: The orthopedic follow-up is roughly every week.

Complications

- Recurrent dislocation: It occurs in approximately 50–90% of patients under age 20 and approximately 5–10% of patients over age 40.
- Ways to prevent redislocation: The position of immobilization, increasing the duration of immobilization, physical therapy, and operative repair.
- Mobilization: <30, immobilize 3 weeks, and >30, begin mobilization after 1 week

Position: Internal rotation and adduction versus 10° external rotation (anatomically sound but evidence does not support benefit).

Recurrent Dislocation of the Shoulder

The shoulder is one of the most unstable and frequently dislocated joints in the body, because of its greatest range of motion at the expense of stability; it accounts for nearly 50% of all dislocations and has a 2% incidence in the general population. The factors that influence the probability of recurrent dislocations are age, return to contact or collision sports, hyperlaxity, and the presence of a significant bony defect in the glenoid or humeral head.

Normal Functional Anatomy of the Shoulder Joint

The glenoid fossa is a flattened, dish-like structure. Only one-fourth of the large humeral head articulates with the glenoid. The glenoid is deepened by 50% by the presence of the glenoid labrum, which increases the humeral contact up to 75%. Superiorly the biceps attaches to the supraglenoid tubercle which blends with the posterior part of the labrum. The labrum may serve as a "chock block" to prevent excessive humeral head rollback. The joint capsule is lax and thin and, by itself, offers little resistance or stability. Anteriorly, the capsule is reinforced by three capsular thickenings or ligaments that are intimately fused with the labral attachment to the glenoid rim.

Glenohumeral Ligaments

1. *Superior glenohumeral ligament*: it attaches to the glenoid rim near the apex of the labrum conjoined with the long head of the biceps. On the humerus, it is attached to the anterior aspect of the anatomical neck. The restraint to inferior, anterior, and posterior stress at 0° of abduction is a tightening of the rotator interval (which includes the superior glenohumeral ligament) that decreases posterior and inferior translation; external rotation also may be decreased.
2. *Middle glenohumeral ligament*: it has a wide attachment extending from the superior glenohumeral ligament along the anterior margin of the glenoid down

as far as the junction of the middle and inferior thirds of the glenoid rim. On the humerus, it also is attached to the anterior aspect of the anatomical neck. It limits external rotation when the arm is in the lower and middle ranges of abduction but has little effect when the arm is in 90° of abduction.

3. *Inferior glenohumeral ligament*: the glenoid margin from the 2 to 3 o'clock positions anteriorly to the 8 to 9 o'clock positions posteriorly to the humeral attachment is below the level of the horizontally oriented physis into the inferior aspect of the anatomical and surgical neck. The anterosuperior edge of this ligament usually is quite thickened. There are a less thick and distinct posterior part and a thin axillary recess which create a hammock-type model. With external rotation, the hammock slides anteriorly and superiorly. The anterior band tightens, and the posterior band fans out. With internal rotation, the opposite occurs.

   The anteroinferior glenohumeral ligament complex is the main stabilizer to anterior and posterior stresses when the shoulder is abducted 45° or more.

Muscles Around the Shoulder Joint

They dynamically position the scapula to place the glenoid opposite the humeral head as the shoulder moves. The ligaments work in a static fashion to limit translation and rotation; their stiffness and torsional rigidity are increased with concomitant muscle activity. The intrinsic and extrinsic muscle groups serve as fine-tuners of motion and power movers by working in "force couples." The force couples control and direct the force through the joint, contributing to stability.

Importance of Synchronous Movement of the Scapula

The glenoid has the ability to remain in the most stable position in relation to the humeral head with movement. Many studies show the importance of this dynamic balance to the appropriate positioning of the glenoid articular surface so that the joint reaction force produced is compressive rather than shear. Strengthening rehabilitation of the scapular stabilizers (serratus anterior, trapezius, latissimus dorsi, rhomboids, and levator scapulae) is especially important in patients who participate in upper-extremity-dominant sports.

The glenoid also has the ability to "recoil." This ability to "recoil" lessens the impact on the shoulder as the scapula slides along the chest wall. Scapular dyskinesia is an alteration of the normal position or motion of the scapula during coupled scapulohumeral movements and can occur after overuse of and repeated injuries to the shoulder joint.

Other stabilizing factors: Version of glenoid, cohesion of joint fluid, vacuum effect produced by negative intra-articular pressure, Ruffini end organs, and Pacinian corpuscles in the shoulder capsule.

Pathoanatomy

Acute shoulder dislocation occurs (1) when the humeral head is forced through the capsule where it is weakest or (2) when the humeral head is forced anteriorly out of the glenoid and tear labrum from almost entire half of the glenoid rim and also capsule and periosteum called Bankart lesion.

A Hill–Sachs lesion is an impaction fracture on the humeral head on the posterolateral aspect which can be produced as the shoulder is dislocated due to impaction of the humeral head against the glenoid rim. Instability results when the defect engages the glenoid rim in the functional arc of motion at 90° abduction and external rotation. Defects of 35–40% of the head were shown to decrease stability.

Capsular laxity—Excessive laxity can be caused by a congenital collagen deficiency, shown by hyperlaxity of other joints, or by plastic deformation of the capsuloligamentous complex from a single macro-traumatic event or repetitive microtraumatic events. The primary deficiencies and secondary deficiencies like erosion of the anterior glenoid rim, stretching of the anterior capsule and subscapularis tendon, and fraying and degeneration of the glenoid labrum all can occur with repeated dislocation.

An arthroscopic study of anterior shoulder dislocations found that 38% of acute injuries were intrasubstance ligamentous failures and 62% were disruptions of the capsuloligamentous insertion into the glenoid neck.

The "circle concept" of structural damage to the capsular structures was suggested by cadaver studies that showed that humeral dislocation does not occur unless the posterior capsular structures are disrupted in addition to the anterior capsular structures. Posterior capsulolabral changes associated with recurrent anterior instability often are identified by arthroscopy.

Classification

There are three types of shoulder instability as follows:

1. Direction of instability
   - Unidirectional
   - Bidirectional
   - Multidirectional
2. Degree of instability
   - Subluxation
   - Dislocation
3. Duration of instability
   - Acute
   - Subacute
   - Chronic if greater than 6 weeks

In addition to these types, there is an entity termed as (4) "habitual" dislocation of the shoulder.

In addition, one must take into consideration the following factors:

(a) Type of trauma
   - Macro-trauma
   - Microtrauma

  - Secondary trauma
(b) Age of initial dislocation
  - <20 years—90% recurrence
  - 20–40 years
  - >40 years—10% recurrence

Matsen's [1, 2] Simplified Classification System

1. Traumatic unidirectional Bankart surgery (TUBS)
2. Atraumatic, multidirectional, bilateral, rehabilitation, inferior capsular shift, and internal closure (AMBRII)

Microtraumatic or developmental lesions fall between the extremes of macrotraumatic and atraumatic lesions and can overlap these extreme lesions.

History

It mainly depends on the amount of initial trauma (high or low energy). Recurrence with minimal trauma in the midrange of motion—a/w with bony lesion. It also depends on the position in which the dislocation or subluxation occurs. If dislocations occur during sleep or with the arm in an overhead position, a/w with significant glenoid defect with the ease with which the shoulder is relocated is determined. There may be associated nerve injury or physical limitations caused by this instability.

Be careful with subluxation which is commonly overlooked by physicians because the symptoms are vague and there is no history of actual dislocation. The patient may complain of having a "dead arm" as a result of stretching of the axillary nerve or of secondary rotator cuff symptoms: posterior shoulder instability may present as posterior pain or fatigue with repeated activity (e.g., blocking in football, swimming, bench press, rowing, and sports requiring an overhead arm movement).

Mental status: Some patients with posterior instability learn to dislocate their shoulders through selective muscular contractions. Although voluntary dislocation does not indicate pathological overlay, some of these patients have learned to use voluntary dislocation for secondary gain, and in these patients surgical treatment is doomed to fail.

Physical examination: Both shoulders should be thoroughly examined, with the normal shoulder used as a reference for atrophy, asymmetry, tenderness, active and passive ROM, power of muscle, and winging or dyskinesia of the scapula.

Stability of the Shoulder Joint

1. *Shift and load test*: with the patient sitting with the arm slightly abducted placing one hand along the edge of the scapula to stabilize it by grasping the humeral head with the other hand and applying a slight compressive force. The amount of anterior and posterior translation of the humeral head in the glenoid is observed. Easy subluxation of the shoulder indicates loss of the glenoid concavity.
2. *Sulcus test*: with the arm in 0° and 45° of abduction. This test is done by pulling distally on the extremity and observing for a sulcus or dimple between the humeral head and the acromion that does not reduce with 45° of external rotation.

The distance between the humeral head and acromion should be graded from 0 to 3 with the arm in 0° and 45° of abduction, with 1+ indicating subluxation less than 1 cm, 2+ indicating 1–2 cm of subluxation, and 3+ indicating more than 2 cm of inferior subluxation, which does not reduce with external rotation. Subluxation at 0° of abduction is more indicative of laxity at the rotator interval, and subluxation at 45° indicates laxity of the inferior glenohumeral ligament complex.

3. *Apprehension test*:
   (a) In an anterior dislocation, the shoulder is in 90° abduction and the elbow in 90° flexion, resulting in external rotation with anterior stress applied.
   (b) In a posterior dislocation, the shoulder is in 90° and the elbow in 90° flexion resulting in a forward flexion, internally stress applied rotated with posterior stress.

   Thus in both types of dislocation (a) and (b), an apprehension or instability is produced.
4. *Drawer test*:
   (a) In an anterior dislocation, anterior stress is applied in various degrees of abduction and external rotation, whereas
   (b) In a posterior dislocation, posterior stress is applied 90° of abduction and in various degrees of flexion + IR.

   Combining the results of (a) and (b), Grade 1 means that the humeral head slips up to the glenoid rim, Grade 2 means that it slips over the labrum but then spontaneously relocates, and Grade 3 indicates dislocation.
5. *Jobe's relocation test*: it is used mainly for evaluating instability in athletes involved in sports requiring overhead motion.

A bony deformity of the glenoid or humerus is indicated by apprehension or instability at low ranges of motion (<90° of abduction).

The tests for inferior instability are as follows:

1. Sulcus test
2. Hyperabduction test
3. Beighton hyperlaxity scale

An external rotation of more than 85° at 0° of abduction is indicative of hyperlaxity.

It also rules out (1) scapular dysfunction, (2) primary/secondary rotator cuff impingement, and (3) neck problems.

A radiological evaluation includes X-rays—AP view, axillary view, Garth AP oblique view, West Point view, and Stryker view—wherein the simplest is AP view in internal rotation.

Other radiological investigations which may be helpful are (1) standard double-contrast arthrography, CT with 3D reconstruction, double-contrast CT arthrography, and gadolinium-enhanced MRI.

Examination using anesthetic and arthroscopy is very useful in anterior shoulder instability. There are more than 150 operations and many modifications but there is no single best procedure. For a successful result adequate exposure and accurate

surgical technique are needed. The pathological condition and a procedure that corrects this condition most anatomically should be defined.

(1) It has a low recurrence rate (2) and has a low complication rate. (3) It also has a low reoperation rate (4) and does no harm (arthritis). (5) It maintains motion and (6) is applicable in most cases. (7) It also allows observation of the joint. (8) It also corrects the pathological condition and (9) is not too difficult.

(a) *Modified Bankart procedure*: the incision is along the Langer lines, beginning 2 cm distal and lateral to the coracoid process and going inferiorly to the anterior axillary crease. Then develop the deltopectoral interval, retracting the deltoid and cephalic vein laterally and the pectoralis major muscle medially. Leave the conjoined tendon intact, and retract it medially.
(b) *Bankart operation*: the subscapularis and shoulder capsule are opened vertically with a lateral leaf of the capsule reattached to the anterior glenoid rim. A medial leaf of the capsule is imbricated, and the subscapularis is approximated. The procedure can be done through a subscapularis split; in larger, more muscular individuals, the subscapularis split can be extended superiorly approximately 1 cm medial to the biceps tendon in an L-shaped fashion.
(c) Anterior stabilization with associated glenoid deficiency (Latarjet procedure): it can be done by keeping the following steps in mind: (1) harvesting and preparation of the bone block; (2) division of the subscapularis, capsulotomy, and exposure; and (3) fixation of the bone block and closure. With the arm in external rotation, repair the remnant of the coracoacromial ligament to the lateral capsular flap with two interrupted absorbable sutures. Remove the sponge placed earlier in the subscapular fossa, and move the arm through all ranges of motion to evaluate mobility. Coat the cut surface of the coracoid with bone wax, place a suction drain, and close the superficial soft tissue layers.

Rehabilitation Program after Anterior Capsulolabral Reconstruction

1. Postoperative Period (0–3 Weeks): abduction pillow, passive/active ROM, abduction (90°), flexion (90°), and external rotation (45°); no extension, isometric abduction, horizontal adduction, and external rotation; elbow ROM, ball squeeze, and ice.
2. Phase I (3–6 Weeks): discontinue brace/pillow, modalities as needed, progressive passive and active ROM, protecting anterior capsule. Active internal rotation (full) and external rotation (neutral) using tubing and free weights. Prone extension (not posterior to the trunk). Shoulder shrugs and active abduction. Supraspinatus strengthening and ice.
3. Phase II (6 Weeks–3 Months): continue ROM, gradually increasing external rotation (the goal is full ROM by 2 months). Continue strengthening exercises, with emphasis on the rotator cuff and parascapular muscles. Add shoulder flexion and horizontal adduction exercises. Encourage joint mobilization. Begin upper-body ergometer for endurance at low resistance followed by ice.
4. Phase III (3–6 Months): continue capsular stretching and strengthening and ergometer. May include isokinetic strengthening and endurance exercises for

internal and external rotation. Add push-ups (begin with wall push-up with the body always posterior to the elbows).

Start chin-ups at 4–5 months. Total body conditioning. Advance to throwing program or skill-specific training as tolerated followed by ice.

Reconstruction of Anterior Glenoid Using Iliac Crest Bone Autograft

Glenoid bone loss approaching 40% of the anterior glenoid or posterior bone loss of 25% with recurrent posterior dislocation should be reconstructed with an autogenous iliac crest bone graft.

It is used occasionally for posterior lesions; the medial aspect of the acromion can be used as a graft. More recently, Provencher et al. [3] described using allograft from the lateral aspect of a distal tibia for reconstruction.

Unsuccessful Surgical Repairs for Anterior Instability

A failure of stabilization may result from failure to correct the pathology, failure to heal, or poor patient compliance. Hence all potential causes of failure must be fully evaluated.

If severe restriction of rotation (i.e., <15° of external rotation) is present, open coronal subscapularis lengthening should be considered.

Posterior Instability of the Shoulder

Posterior shoulder dislocations and recurrent posterior instability of the shoulder account for only 2–4% of all dislocations of the shoulder. Traumatic events that result in posterior dislocation often are associated with altered consciousness, such as with seizures, electrical shock, and intoxication. It also can be caused by a direct blow to the anterior shoulder or by a fall on a forward-flexed extremity.

Recurrence can be caused by atraumatic and repetitive microtrauma leading to a traumatic posterior dislocation. Repetitive overuse and microtraumatic injuries that result in posterior instability include sports requiring overhead motion, such as pitching, tennis, and swimming (especially backstroke and breaststroke), weightlifting (especially bench press), and blocking by offensive linemen. Some patients learn to subluxate their shoulders voluntarily with horizontal adduction and internal rotation; however, this does not mean there is a psychological overlay. A patient who has a bland type of affect and who is able to subluxate his shoulder with muscular contraction alone is more likely to have some psychological overlay and secondary gain. These patients rarely, if ever, should be surgically treated. In the past, glenoid version has been implicated in posterior instability; however, glenoid version contributes significantly to posterior instability only in patients with severe congenital dysplasia or traumatic disruption of the bony architecture.

Conservative Treatment

The initial management is (1) to avoid provocative activities and educate the patient to avoid specific voluntary maneuvers and (2) strengthen exercise programs aimed at the external rotators, and posterior deltoid is carried out, and normal motion should be obtained. Most patients with posterior instability respond to an aggressive

exercise program, especially patients with generalized ligamentous laxity and instability occurring as a result of repetitive microtrauma. Patients who have traumatic dislocations are less likely to be helped by an exercise program (athletes who have repetitive posteriorly directed forces to the shoulder, such as football linemen, hockey players, and platform divers).

Indications of Surgery

1. Failure of 4–6 months of an appropriate rehabilitation program.
2. Habitual dislocation has been ruled out.
3. Must be emotionally stable with pain and instability preclude adequate function of the involved shoulder.

Surgical Treatment

Many surgical procedures with varied results are described. The best results with any procedure with traumatic posterior dislocation. If an open technique is to be done, arthroscopic examination for these lesions to evaluate the rotator interval anteriorly, a greater than 1 cm gap between the superior and middle glenohumeral ligaments at the edge of the glenoid, and closure of the rotator interval should be done arthroscopically. If surgery is required, a procedure that is found most successful is the inferior capsular shift procedure through a posterior approach.

The capsular shift technique of Tibone or that of Neer and Foster [4] is used for atraumatic multidirectional instability in a patient who is not an athlete who uses throwing or overhead motions.

For an athlete with overhead movement, muscle-splitting technique with medial shift as described by Tibone et al. [5] is preferred.

The technique described by Hawkins and Janda [6] is best reserved for a laborer or an athlete involved in contact sports, such as football or ice hockey.

Neer Inferior Capsular Shift Procedure Through a Posterior Approach

In this procedure, the posterior capsule is split longitudinally, and the capsular attachment along the humeral neck is released as far inferiorly and anteriorly as possible. The superior capsule is advanced inferiorly, and the inferior capsule is advanced superiorly. The infraspinatus is cut so that it is overlapped and shortened, adding further buttress to the posterior capsule. This procedure obliterates the axillary pouch and redundancy. Postoperatively, immobilize the arm at the side in neutral flexion-extension and 10° of external rotation with the elbow bent 90°. Rigid external immobilization is needed to ensure that 10° of external rotation is maintained.

Tibone and Bradley technique: The capsule is shifted on the glenoid side to reduce the volume of the posterior capsule in a manner similar to that described by Neer and Foster.

Capsular shift reconstruction with posterior glenoid osteotomy: Posterior glenoplasty rarely is indicated, although it can be used if severe developmental or traumatic glenoid retroversion of more than 20° is confirmed on CT reconstructed films. High recurrence rates of up to 53% have been reported with this. Hawkins et al.

reported a complication rate of 29%, including osteonecrosis of the glenoid and degenerative arthritis of the glenohumeral joint, after this procedure. Currently, a similar but simpler procedure using a glenoid osteotomy is preferred for severe glenoid dysplasia, whether traumatic or congenital.

Causes of Failure in Surgical Repair of Posterior Shoulder Instability

1. Inadequate soft tissue healing
2. Ligamentous laxity
3. Deficient capsule
4. Deficient subscapularis
5. Deficient glenoid
6. Engaging Hill-Sachs lesion
7. Overconstrained joint
8. Nerve dysfunction

McLaughlin Procedure

McLaughlin [7] described transfer of the subscapularis tendon into the defect. Neer and Foster subsequently described transfer of the subscapularis with the lesser tuberosity into the defect and securing it with a bone screw. In a rare reverse Hill-Sachs lesion with involvement of 20–25% of the articular surface, transfer of the subscapularis with the tuberosity placed into the defect has been shown to produce satisfactory results.

Multidirectional Instability of the Shoulder

The primary abnormality in multidirectional instability is a loose, redundant inferior pouch.

It is important to distinguish multidirectional instability from routine unidirectional dislocation because the former problem is not correctable by standard repairs.

Surgery in these patients is not indicated unless disability is frequent and significant, an adequate trial of conservative treatment emphasizing muscular and rotator cuff rehabilitative exercises has failed, and the patient is not a voluntary dislocator.

Arthroscopic Procedure

Control of bleeding during arthroscopy: It is usually done by an arthroscopic electrocautery device. Arthroscopy pump for inflow, maintaining a constant fluid flow and pressure of 60–70 mmHg; add 1 mL of 1:1000 epinephrine to each 3000-mL bag of irrigant. Perhaps the most effective is to use hypotensive anesthesia with a systolic blood pressure of 90–100 mmHg.

Habitual Dislocation of the Shoulder

Habitual dislocation of the shoulder is extremely rare. Habitual dislocation of the shoulder is a phenomenon in youth, with the first dislocation occurring before 30 years of age, which is more common in males. It is very common in epileptics, with

the dislocations frequently occurring in fits, in traumatic falls, or by muscle spasms alone [8]. Treatment in epileptics is particularly difficult, with the tendency of recurrence being extremely great during the seizures.

Formerly 25–30% of the patients with habitual dislocation of the shoulder were epileptics, but in recent years the frequency of habitual dislocation in epileptics is decreasing; the dislocation occurs with every fit and is always anterior [9]. The dislocation in epileptics has decreased considerably on account of the fact that the modern treatment of epilepsy reduces the number of convulsions.

Milwaukee Shoulder Syndrome (MSS)

It is a rare destructive, calcium phosphate crystalline arthropathy. MSS occurs in elderly patients typically aged 60–90 years. There is a female preponderance in the ratio of 4:1; the higher life expectancy of women may be contributory. Unilateral shoulder joint involvement is more common and seen in the dominant side; however, in the case of bilateral shoulder disease, this is almost always more advanced on the dominant side.

Risk factors for MSS are as follows:

1. Trauma or overuse
2. Calcium pyrophosphate dihydrate crystal deposition
3. Neuroarthropathy
4. Dialysis arthropathy
5. Denervation
6. Female gender
7. Advanced age

Shoulder Dystocia (SD)

It (SD) still represents a huge risk of morbidity for both the mother and fetus. SD remains an unpreventable and unpredictable obstetric emergency (Table 2.1).

Risk factors for SD

Complications (Table 2.2) of SD

Table 2.1 Risk factors for SD

Maternal-fetal (pre-labor)	Intrapartum
Macrosomia	Prolonged active phase of first-stage labor
Diabetes (gestational or mellitus)	Prolonged second-stage labor
Maternal BMI greater than 30 kg/m^2	Assisted vaginal delivery (forceps or vacuum)
Short stature	Oxytocin augmentation
Previous SD	Secondary arrest
Abnormal pelvic anatomy	Inappropriate maneuvers (fundal pressure)
Postdate pregnancy	Epidural anesthesia
Advanced maternal age	
Male gender	
Male gender	

Table 2.2 Complications of SD

Maternal	Fetal
Postpartum hemorrhage	Brachial plexus palsy
III- or IV-degree episiotomy or laceration	Fetal death
Symphyseal separation or diathesis, with or without transient femoral neuropathy	Fetal hypoxia, with or without permanent neurologic damage
Rectovaginal fistula	Clavicle and humerus fracture

When SD occurs, umbilical cord compression between the fetal body and the maternal pelvis is a potential danger. Insult to the fetus from hypoxia results from compression of the neck and central venous congestion, as well as compression of the umbilical cord, and reduces placental intervillous flow from prolonged increased intrauterine pressure, and secondary fetal bradycardia occurs.

In conclusion, despite its infrequent occurrence, all healthcare providers attending pregnancies must be prepared with a high level of awareness and training to handle vaginal deliveries complicated by SD.

Acromioclavicular Joint Arthritis

Definition: Acromioclavicular joint arthritis (AC joint arthritis) is a progressively degenerative disease in which there is degeneration of the joint cartilage and the underlying bone which causes pain and stiffness.

Etiology: The combination of three factors underlies the frequency of problems of the AC joint.

1. Because it is a diarthrodial joint, it is vulnerable to the same processes affecting other joints in the body, such as degenerative osteoarthritis, infections, and inflammatory and crystalline arthritis.
2. Its superficial location and its relationship to the shoulder girdle predispose it to traumatic injury.
3. The biomechanics of the shoulder girdle require the AC joint to transmit large loads across a very small surface area, which can result in failure with repetitive activity or overuse.

Risk Factors

1. Age (>45 years)
2. History of previous injury to the AC joint (specially previous trauma and sports injury)
3. Weightlifting activities particularly those transmitting huge loads across the shoulder joint like bench press and military press.

Causes

There are three common causes of acromioclavicular joint arthritis:

1. Primary osteoarthritis
2. Posttraumatic osteoarthritis
3. Distal clavicle osteolysis

Primary Osteoarthritis

In comparison to the rate of occurrence in the knee and hip, primary osteoarthritis in the shoulder is relatively rare. However, primary involvement of the AC joint is a much more common primary involvement of the glenohumeral joint and is, in fact, the most common cause of pain in the AC joint.

Degenerative changes by the fourth decade in the majority of AC joint.

In one study, 54–57% of elderly patients demonstrated radiographic evidence of degenerative arthritis. In another study, magnetic resonance (MR) imaging demonstrated evidence of arthritic changes in 48% of the AC joints in over 300 older asymptomatic patients. Despite its seeming prevalence by radiologic criteria, symptomatic primary osteoarthritis is a relatively uncommon clinical entity.

Posttraumatic Arthritis

Acromioclavicular arthritis following trauma is even more common than primary osteoarthritis, due to the frequency of injury to this vulnerable joint. The incidence of posttraumatic arthritis symptoms after injury or surgery is highly variable and depends on the degree of injury and the type of operative procedure.

Arthritis also occurs, although less commonly, after distal clavicle fractures, particularly those with intra-articular extension. Operative procedures for AC joint dislocations in which the AC joint is preserved or transfixed have been associated with a higher incidence of arthritis than those in which the joint is sacrificed (i.e., Weaver–Dunn procedure).

Distal Clavicle Osteolysis

An increasingly recognized but still infrequent entity that causes AC joint symptoms is distal clavicle osteolysis. Osteolysis related to repetitive microtrauma has recently been receiving more attention, particularly among weightlifting athletes. This condition is thought to be growing in frequency due to weight training and its incorporation into fitness programs and training regimens for other sports.

The proposed mechanism of this form of osteolysis is that repetitive stresses to the subchondral bone of the distal clavicle lead to fatigue failure, which initiates resorption.

Grading of Osteoarthritis

Kellgren Lawrence Classification (Table 2.3) of the Severity of Osteoarthritis [10].

Table 2.3 Grades of osteoarthritis

Grade	Description
0	No radiographic features of osteoarthritis
1	Doubtful narrowing of the joint space with possible osteophytic lipping
2	Possible narrowing of joint space with definite osteophytes
3	Definite narrowing of the joint space, moderate multiple osteophytes, subchondral sclerosis, and possible deformity of the bone ends
4	Marked narrowing of joint space, large osteophytes, severe subchondral sclerosis, definite deformity of the bone ends

Shoulder Separation: Shoulder separation is not truly an injury to the shoulder joint. The injury actually involves the acromioclavicular joint (also called the AC joint).

The most common cause for the separation of the AC joint is from a fall directly onto the shoulder.

The fall injures the ligaments that surround and stabilize the AC joint.

Types of Separation

A mild shoulder separation involves a sprain of the AC ligament that does not move the collarbone and looks normal on X-rays. A more serious injury tears the AC ligament and sprains or slightly tears the coracoclavicular (CC) ligament, putting the collarbone out of alignment to some extent.

Severe Separation: The most severe shoulder separation completely tears both the AC and CC ligaments and puts the AC joint noticeably out of position.

Symptoms: Pain at the end of the collar bone. Pain may feel widespread throughout the shoulder until the initial pain resolves, following this it is more likely to be a very specific site of pain over the joint itself. Swelling often occurs.

AC joint injuries are graded from 1 to 6 using the Rockwood scale [11] which classifies injuries in relation to the extent of ligament damage and the space between the acromion and clavicle.

Shoulder Separations are Graded from Type I to VI

The Rockwood classification of acromioclavicular injuries in adults is as follows:

1. Type I: minor sprain of the acromioclavicular ligament, intact joint capsule, intact coracoclavicular ligament, intact deltoid and trapezius.
2. Type II: rupture of the acromioclavicular ligament and joint capsule, sprain of the coracoclavicular ligament but intact coracoclavicular interspace, minimal detachment of the deltoid and trapezius.
3. Type III: rupture of the acromioclavicular ligament, joint capsule, and coracoclavicular ligament; elevated clavicle (≤100% displacement); detachment of the deltoid and trapezius.
4. Type IV: rupture of the acromioclavicular ligament, joint capsule, and coracoclavicular ligament; posteriorly displaced clavicle into the trapezius; detachment of the deltoid and trapezius.
5. Type V: rupture of the acromioclavicular ligament, joint capsule, and coracoclavicular ligament; elevated clavicle (>100% displacement); detachment of the deltoid and trapezius.
6. Type VI (rare): rupture of acromioclavicular ligament, joint capsule, and coracoclavicular ligament; the clavicle is displaced behind the tendons of the biceps and coracobrachialis.

Evaluation and Diagnosis

1. Presentation:

   Isolated AC joint arthritis presents with discomfort or aching over the anterior and/or superior aspect of the shoulder. Pain is often brought on by activities of

daily living, such as washing the opposite axilla and reaching back to retrieve a wallet. Symptoms are often exacerbated by more demanding activities, such as pushing or overhead work in the case of laborers and weightlifting, golfing, swimming, or throwing in athletes. Patients may note pain at night, with nocturnal awakening when rolling onto the affected shoulder. There may be associated symptoms of popping, catching, or grinding.

2. Physical Examination:

Careful examination of the entire shoulder girdle combined with cervical spine examination is essential to rule out any contribution from cervical lesions.

Inspection of the affected extremity may reveal swelling, deformity, joint prominence, or asymmetry which may indicate AC joint instability. Palpation over the AC joint may elicit tenderness, which is anecdotally sensitive but nonspecific.

Dynamic stability of the AC joint can be assessed by placing the patient supine and the affected extremity in 90° of flexion. With one hand on the affected joint, the examiner assesses for the movement of the clavicle with respect to the acromion while applying a downward force on the patient's flexed arm.

Specific Tests

1. Provocative maneuvers, such as reaching across to touch the opposite shoulder or placing the hand behind the back, may elicit discomfort.

The provocative tests include:

(a) Cross-body adduction test: the most reliable provocative physical examination is the cross-body adduction test, in which the arm on the affected side is elevated 90° of forward flexion and the examiner then grasps the elbow and adducts the arm across the body. Reproduction of pain over the AC joint is suggestive of an AC joint lesion. This test may also be positive in patients with subacromial impingement and may cause discomfort posteriorly in patients with posterior capsular tightness.

The sensitivity is 77% and specificity is 79%.

(b) AC resisted extension test:

The patient is seated with the examiner standing behind him/her. The patient's shoulder is positioned into 90° of flexion and internal rotation, with the elbow placed into 90° of flexion. The examiner places his/her hand on the patient's elbow and asks him/her to horizontally abduct the arm against isometric resistance.

A positive test is pain at the AC joint. This test has a sensitivity of 72% and specificity of 85%.

(c) O'Brien active compression test:

In this test, the affected arm is brought into 90° of forward flexion and 10° of adduction. The patient then performs resisted shoulder flexion with the arm in maximum internal rotation and then in maximum supination. Pain with the former maneuver is consistent with a SLAP lesion and pain with the latter maneuver indicates AC joint abnormality.

Sensitivity of this test is 41% and specificity is 95%. The overall accuracy of these provocative tests in diagnosing AC joint arthritis is 93%.

2. Painful arc sign:

   In this test, the affected shoulder is abducted and if the patient experiences pain during the last 30° of abduction, it is consistent with AC joint arthritis. Sensitivity of this test is 50% and specificity is 47%.

3. Paxinos sign:

   With the patient sitting and the symptomatic arm by the side, the examiner's thumb is placed under the posterolateral aspect of the acromion and the index and middle fingers of the same (or contralateral) hand are placed superior to the mid-clavicle.

   If we are examining the left shoulder, the right hand is to be used for eliciting this sign, and vice versa.

   The examiner provides pressure to the acromion in an anterosuperior direction with the thumb while applying pressure in an inferior direction to the mid-clavicle with the index and middle fingers. If pain is elicited or increased in the region of the acromioclavicular joint, the test is considered positive.

Radiological Examination

X-Rays

1. Shoulder AP View: the AP projection is usually obtained with the patient in upright or supine position and with the coronal plane of the body parallel to the cassette. The beam is directed in a true AP direction relative to the body. This results in slight overlap of the glenoid rim and the humeral head as the glenohumeral joint is tilted anteriorly approximately 40°.
2. Zanca View:

   Zanca described a modified technique that provides a clear, unobstructed view of the distal clavicle and AC joint. This projection is obtained by angling the X-ray beam 10–15° superiorly and decreasing the kilovoltage to about 50% of that used for a standard glenohumeral exposure.

   Findings: Patients with primary or posttraumatic degenerative arthritis will have findings of arthritic changes which include (a) sclerosis, (b) osteophyte formation, (c) subchondral cysts, and (d) joint space narrowing.

3. Supraspinatus Outlet View:

   Supraspinatus outlet view is useful for evaluating the acromion process and subacromial abnormalities such as osteophytes that may cause impingement.

   It is similar to the Y-view but with caudal tube angulation. This view is taken with the patient turned as for the Y projection and the cassette perpendicular to the body of the scapula and parallel to the glenoid fossa. The X-ray is taken from a mediolateral projection along the axis of the scapular spine, with the X-ray beam angled 10–15° craniocaudally and centered on the acromioclavicular joint.

Ultrasonography

Ultrasonography can be used to assess joint space and detect osteophytes or other bony erosions, although the usefulness of this technique is dependent on the skill of the technician and is limited to superficial soft tissue.

MRI

It is very sensitive in identifying abnormalities of the AC joint, but these changes often do not correlate with physical findings.

In one study of asymptomatic volunteers, findings indicative of AC joint arthritis were present in 75% of shoulders. The nonspecificity of MRI precludes it from being useful in the evaluation of patients with AC joint symptoms. However MRI can be helpful in ruling out other causes of shoulder joint pain which can be concomitantly present with AC joint arthritis.

Joint Injection: Joint injection can be used both diagnostically and therapeutically. A combination of local anesthetic and corticosteroid is used.

Technique: Palpate the bony landmarks and mark the site of injection. Prepare the skin using sterile technique. A 23-gauge needle is directed into the joint from a superior approach. The needle is then slowly advanced perpendicular to the articulation while palpating for a tactile pop through the capsule. The mixture can then be easily injected and noted to flow freely into the joint. The joint can be injected under sonographic guidance.

Despite the subcutaneous nature of the joint, intraarticular injections can sometimes be difficult where the accuracy can be improved with the use of ultrasound guidance.

Elimination of pain within a few minutes of the injection confirms the AC joint as the source of the patient's symptoms and is considered by many authors to be the most valuable diagnostic tool.

Relief after an injection is also considered the most accurate prognostic indicator of success with distal clavicle resection.

Treatment: Whether treated conservatively or with surgery, the shoulder will require rehabilitation to restore and rebuild motion, strength, and flexibility.

The main treatment will depend on the type of injury. The initial treatment of a separated shoulder consists of controlling the inflammation and resting the joint.

1. Icing the injury: the inflammation from a separated shoulder can be controlled with ice placed on the joint every 4 h for a period of 15 min. Icing can be done for the first several days until the swelling around the joint has subsided.
2. Resting the AC joint: a sling to rest the joint can be worn until the pain has subsided and you can begin some simple exercises. Resting the joint will help minimize painful symptoms and allow healing to begin.
3. Anti-inflammatory medication: this will also help to minimize the pain and inflammation.
4. Grade I and II injuries: it has been underestimated and may lead to more chronic disability than previously recognized, especially in athletes and heavy laborers who stress their shoulders daily.

   Some late surgery as AC joint resection arthroplasty may be needed. However, more than 50% of the patients have a good or excellent shoulder 6 years after injury.
5. Surgery: it is not done in most cases. Type I and type II shoulder separations are by far the most common types of separated shoulders, and these types of injuries

need surgery rarely and only if there are problems with nonoperative treatment. Type IV, V, and VI shoulder separations almost always require surgery, but these are very uncommon injuries. Difficult decisions arise with patients with a type III shoulder separation. There is controversy among orthopedic surgeons as to how to best manage patients with a type III shoulder separation. As may be expected, there is no "right answer."

6. Surgical treatment: surgery can be considered if pain persists or the deformity is severe. A surgeon might recommend trimming back the end of the collarbone so that it does not rub against the acromion. Where there is significant deformity, reconstructing the ligaments that attach to the underside of the collarbone is helpful. This type of surgery works well even if it is done long after the problem started.
7. Nonsurgical treatment for type III shoulder separations: most evidence suggests that patients with type III shoulder separations do just as well without surgery and avoid the potential risks of surgical treatment. These patients return to sports and work faster than patients who have surgery for this type of injury.
8. Types III and IV: AC joint instability symptoms may persist, with impingement symptoms secondary to the drop down of the shoulder and the abnormal biomechanics. A patient may complain of severe deformity in the AC joint and traction symptoms with neck pain and neural brachial plexus symptoms. There is a significant decrease (24%) in horizontal abduction strength at fast speeds.

   However, overall 87% with type III dislocation showed satisfactory outcome with conservative treatment of "skillful neglect."
9. Surgery for type III shoulder separations: recent studies have suggested that some athletes and heavy laborers may benefit from early surgical treatment of type III shoulder separations. These include athletes who participate in sports that require overhead throwing such as baseball.

   The potential benefit of early surgical treatment for type III shoulder separations remains unproven.

Sternoclavicular Joint

The Sternoclavicular joint is a saddle type of synovial joint between the sternal end of the clavicle and the manubrium of the sternum with the articulation of the first costal cartilage (Fig. 2.3).

Capsule properties: It has a strong joint capsule which consists of the sternal end of the clavicle, the manubrium of the sternum, and part of the first costal cartilage. The articular surfaces are covered with fibrocartilage. The joint is separated into two compartments by a fibrocartilaginous articular disk (Fig. 2.4).

The capsule reinforced is mainly the subclavius muscle. It originates at the costal cartilage of the first rib and gets inserted into the inferior aspect of the clavicle.

Ligaments: The ligaments of the sternoclavicular joint provide much of its stability. There are four major ligaments:

1. Sternoclavicular ligaments (anterior and posterior)—these strengthen the joint capsule anteriorly and posteriorly.

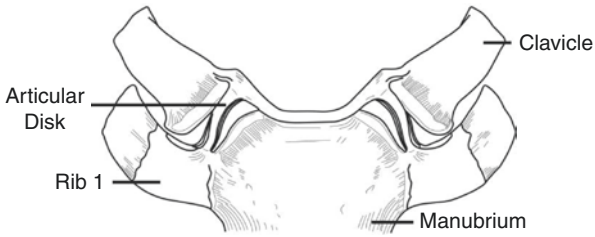

Fig. 2.3 Line diagram of the sternoclavicular joint.

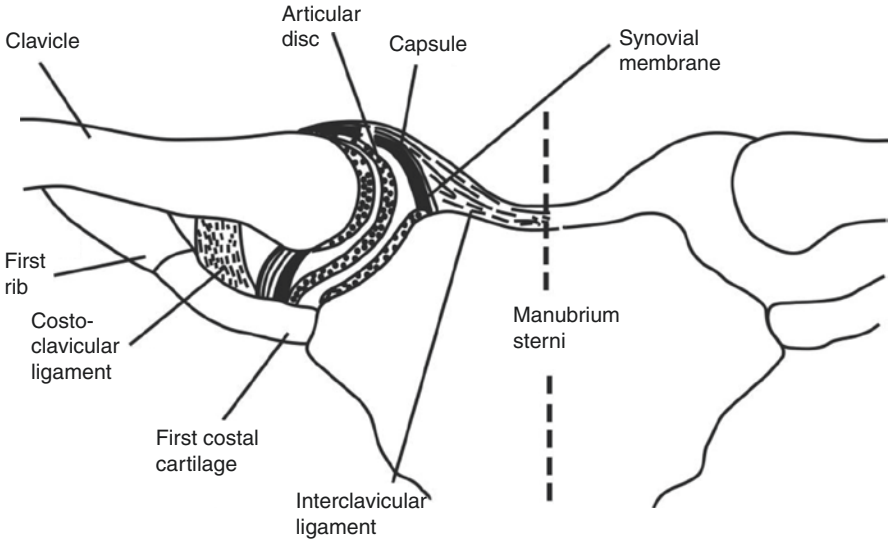

Fig. 2.4 Line diagram of the sternoclavicular joint showing capsule and articular disk

2. Interclavicular ligament—this spans the gap between the sternal ends of each clavicle and reinforces the joint capsule superiorly.
3. Costoclavicular ligament—the two parts of this ligament (often separated by a bursa) bind at the first rib and cartilage inferiorly and to the anterior and posterior borders of the clavicle superiorly. It is a very strong ligament and is the main stabilizing force for the joint, resisting elevation of the pectoral girdle.

Neurovascular supply (a): Arterial supply to the sternoclavicular joint is from the internal thoracic artery and the suprascapular artery. The veins of the joint follow the major arteries. The veins of the joint follow the major arteries. (b) The joint is supplied by the medial supraclavicular nerve (C3 and C4) and the nerve to the subclavius (C5 and C6).

Movements of the joint: The sternoclavicular joint has a large degree of mobility (Table 2.4). There are several movements that require this joint movement, namely, (1) elevation, (2) depression, (3) retraction, (4) protraction, and (5) rotation.

Table 2.4 Movements of the Sternoclavicular joint.

Movements	Muscles
(a) Elevation	Upper trapezius, levator scapulae
(b) Depression	Lower trapezius, pectoralis minor
(c) Adduction (retraction)	Middle trapezius, rhomboids
(d) Abduction (protraction)	Serratus anterior
(e) Upward rotation	Upper and lower trapezius, serratus anterior
(f) Downward rotation	Rhomboids, pectoralis minor, levator scapulae

Physiological importance: The Sternoclavicular joint (SCJ) is important because it helps to support the shoulder. The SCJ links the bones of the arms and shoulder to the vertical skeleton.

Pathologic abnormality of the sternoclavicular joint (SCJ) is rare. Those with osteoarthritis may benefit from local steroid injection to ameliorate the inflammation and subsequent pain associated with this process. For those who fail conservative treatment, surgical excision of the joint is curative in this noninfectious process.

SCJ septic arthritis is a rare clinical entity, accounting for only 1% of cases of septic arthritis in the general population. It can, however, result in life-threatening complications if not treated adequately. Although mild cases may respond to antibiotics and surgical debridement, more serious cases require SCJ resection.

Although the sternoclavicular joint is an unusual site for infection, thoracic surgeons may preferentially be called on to coordinate management of cases refractory to antibiotic therapy because of the anatomic relationship of this joint to major vascular structures. Most cases of early sternoclavicular joint infections will respond to conservative measures. However, when radiographic evidence of infection beyond the sternoclavicular joint is present, en bloc resection, although seemingly aggressive, results in immediate eradication of all infection with negligible functional morbidity.

The sternoclavicular joint (SCJ) is commonly affected by rheumatological conditions. The sternoclavicular joint (SCJ) is a true diarthrodial joint that can be involved during the course of RA; however, its clinical implications appear to continue to be underestimated by the rheumatology community. There is ultrasound evidence that reveals a higher prevalence of SCJ involvement in patients with RA than in age- and sex-matched healthy controls. The SCJ is capable of referring pain to areas distant from the joint.

Scapular Winging

Scapular winging is a rare debilitating condition that leads to limited functional activity of the upper extremity. It is the result of numerous causes, including traumatic, iatrogenic, and idiopathic processes that most often result in nerve injury and paralysis of the serratus anterior, trapezius, or rhomboid muscles. Diagnosis is easily made upon visible inspection of the scapula, with serratus anterior paralysis resulting in medial winging of the scapula. This is in contrast to the lateral winging generated by trapezius and rhomboid paralysis.

The serratus anterior is a muscle that originates on the surface of the first to eighth ribs at the side of the chest and inserts along the entire anterior length of the medial border of the scapula. The serratus anterior acts to pull the scapula forward around the thorax.

The muscle is inserted along the medial border of the scapula between the superior and inferior angles along with being inserted along the thoracic vertebrae. The muscle is divided into three named parts depending on their points of insertions:

1. The serratus anterior superior is inserted near the superior angle.
2. The serratus anterior intermediate is inserted along the medial border.
3. The serratus anterior inferior is inserted near the inferior angle.

The most common type of scapular winging is medial winging due to a long thoracic nerve (LTN) palsy leading to a dysfunctional serratus anterior muscle. This weakness of the serratus causes the scapula to move away from the ribs with flexion of the arm. Consequently the glenoid tilts downward and the arm cannot be raised into full flexion.

The presentation, exam findings, and treatment plans can vary based on the individual etiology responsible for the winging; however, this chapter will focus on LTN palsy leading to winging from a dysfunctional serratus anterior muscle.

Plain films are commonly normal, although subtle differences in scapular position may be detected.

A CT scan can be performed to better elucidate bony abnormalities and rule out scapular masses only if the patient has mechanical symptoms to suggest such deformities.

An EMG evaluation is required to confirm the diagnosis of LTN dysfunction but should not be performed until at least 6 weeks after the onset of symptoms.

Scapular winging is a cause of shoulder pain that is often overlooked, leading to unnecessary surgical procedures. Careful physical examination and appropriate diagnostic workup can verify the diagnosis, which is most commonly due to a long thoracic nerve palsy. Most patients benefit from a trial of conservative therapy focusing on periscapular strengthening with or without bracing. Surgery may be indicated for those patients who do not improve with conservative therapy and consists of the transfer of all, or a portion, of the pectoralis major tendon either with or without graft.

Frozen Shoulder

Frozen shoulder is defined as a glenohumeral joint with pain and stiffness that cannot be explained on the basis of joint incongruity. It is also known as adhesive capsulitis as the pathology involves the capsule of the joint. The pathology is similar to Dupuytren's with fibroblastic proliferation and is commonly associated with diabetes or Dupuytren's contracture. The incidence is 2%. It is more commonly seen in women than men during the fifth to seventh decade. Bilateral involvement occurs in 10–40% of the cases. It does not usually recur in the same shoulder, but some 20–30% develop the condition in the opposite shoulder.

Historical perspective: Duplay [12] referred to frozen shoulder in 1872 as "scapulohumeral periarthritis," a disorder he believed resulted from subacromial

bursitis. Pasteur later referred to the same condition as "tenobursite," which he attributed to bicipital tendinitis. In 1934, Codman [13] coined the term "frozen shoulder" but used it in association with tendinitis of the rotator cuff. In 1945, Neviaser [14] introduced the concept of adhesive capsulitis. He discovered that the capsule was tight, thickened, and stuck to the humerus in such a manner that it could be peeled off like an "adhesive plaster from the skin."

Muscles involved:

1. Flexion: anterior fibers of deltoid and pectoralis major
2. Extension: posterior fibers of deltoid and latissimus dorsi
3. Abduction: middle fibers of deltoid and supraspinatus
4. Adduction: pectoralis major and latissimus dorsi
5. Lateral/external rotators: infraspinatus and teres minor
6. Medial/internal rotators: subscapularis and latissimus dorsi

Etiopathogenesis

Lundberg [15] classified frozen shoulder into primary and secondary types.

1. Primary frozen shoulder: no inciting event, normal plain radiographs, and no findings other than loss of motion. No inciting event but intrinsic and extrinsic predisposing factors present.
   (a) Intrinsic factors include ages between 40 and 60 years, female sex, and diabetes mellitus.
   (b) Extrinsic factors may include immobilization and faulty body mechanics.
2. Secondary frozen shoulder: precipitant traumatic event.

Pathology

Lundberg evaluated the shoulder capsules of 14 patients. Histology showed an increase in fibrous tissue, fibroblasts, and vascularity.

Hazelman reported shoulder capsular tissue showed fibroblast and myoblast proliferation identical to that seen in Dupuytren's disease and vascular changes suggestive of diabetic microangiopathy.

This suggested that frozen shoulder may have abnormalities at the cellular level with malfunctions of fibroblast recruitment and cytokine growth factor production and release.

Hannafin (Table 2.5) and Chiaia [16] described three phases based on capsular biopsies on 15 patients with frozen shoulder.

Neviaser (Table 2.6) defined four stages of frozen shoulder based on arthroscopic changes observed [17].

Hannafin's stages of frozen shoulder

Neviaser's arthroscopic stages of frozen shoulder

Secondary Frozen Shoulder

1. Rotator cuff diseases
2. Fracture residuals

Table 2.5 Stages of frozen shoulder (Hannafin)

Phase	Clinical	Examination under anesthesia	Arthroscopy	Histology
1	Pain + decreased ROM	Full ROM	Vascular synovitis, diffuse	Vascular inflammatory synovitis, normal capsule
2	Pain + decreased ROM	Limited ROM	Vascular synovitis, diffuse	Hypervascular synovial hyperplasia, diffuse capsular fibroplasias
3	Marked pain + limited or loss of motion	Marked loss of motion	Minimal synovitis	Minimal synovial hyperplasia, capsular hyperplasia, scar formation

Table 2.6 Four arthroscopic stages of frozen shoulder (Neviaser)

Stage	Pathology	Physical symptoms	Treatment
1	Low-lying fibrinous synovitis, no capsular contracture	Pain + normal ROM	Physical therapy
2	Increased synovitis with fibrinous adhesions with capsular contracture	Pain + restricted ROM	Physical therapy followed by manipulation
3	Subsiding synovitis with increasing capsular contracture	Less with loss of motion	Physical therapy followed by manipulation
4	Only capsular contracture, no intraarticular process	LOM with pain on extremes of motion	Physical therapy followed by manipulation

3. Calcific tendinitis
4. Previous shoulder surgery
5. Osteoarthritis
6. Cervical spine lesions
7. Autoimmune disease
8. Chest wall tumors
9. Thyroid disorders
10. Parkinson's disease
11. CVA
12. Head injury
13. Myocardial infarction

Clinical Features

Natural History

- Stage 1: Freezing

  In the "freezing" stage, you slowly have more and more pain. As the pain worsens, your shoulder loses range of motion. Freezing typically lasts 6 weeks to 9 months.

- Stage 2: Frozen
  Painful symptoms may actually improve during this stage, but the stiffness remains. During 4–6 months of the "frozen" stage, daily activities may be very difficult.
- Stage 3: Thawing
  Shoulder motion slowly improves during the "thawing" stage. Complete return to normal or close to normal strength and motion typically takes 6 months to 2 years.

The clinical features can also be described in three stages as follows:

- Phase 1—Pain
  It is insidious/acute in onset and also present during activity and rest unlike other disorders. It is more at night affecting sleep and distributed vaguely over the deltoid muscle area. The only point of tenderness is the bicipital groove which may radiate over the C5 dermatome to the upper back due to compensatory use of shoulder girdle muscles.
- Phase 2—Stiffness
  Motion is guarded and a protective muscular spasm is a common feature. The patient may prefer wearing a sling to support the arm. Functional activities such as dressing or grooming which require reaching overhead or the back may be difficult. Loss of ROM is most prominent once the pain has subsided.
  "Girdle hunching maneuver" is used in order to substitute glenohumeral movements with scapulohumeral movements. There is an "empty end feel" at the end of the ROM. The internal rotation is lost initially followed by loss of flexion and external rotation. The hallmark is a terminally painful passive ROM (c.f. rotator cuff tendinitis and painful arc syndrome).
  Limitation of passive ROM shows a capsular pattern: external rotation is greater than abduction which is greater than internal rotation. The external rotation is lesser than 45°, abduction is lesser than 80°, and internal rotation is lesser than 70°.
- Phase 3—Thawing
  As motion increases, pain diminishes and usually occurs spontaneously over 4–9 months even without any treatment. It may not regain full range of motion, but may feel normal as a result of compensatory mechanisms and adjustments in activities of daily living.

Diagnosis

Campbell [18] describes the presence of three features to diagnose frozen shoulder:

1. Internal rotation restricted up to the point when the patient cannot touch beyond his sacrum
2. 50% loss of external rotation
3. Lesser than 90° of abduction

However, these criteria are not definitive and the presence of all three is not mandatory.

Investigations

1. X-rays—to exclude other mimicking conditions

   Plain X-ray is normal. However, it can be used to rule out other conditions. Commonly revealed conditions are osteoporosis, degenerative changes, decreased space between the acromion and humeral head, calcium deposits, and cystic changes.

   Differential diagnosis: Sudden onset of severe pain; causes may be adhesive capsulitis, calcific tendonitis, or PT syndrome; loss of ER may be adhesive capsulitis, OA, or locked post dislocation
2. Arthrography: this can either be done fluoroscopically or with the help of MRI. A 50% reduction in joint fluid volume and a boxlike appearance of the joint cavity are diagnostic. Joint volume capacity is only 5–10 mL (normal = 20–30 mL). A tight thickened capsule, loss of the axillary recess, subcoracoid folds, subscapular bursa and absence of dye in the biceps tendon sheath.
3. MRI: the normal inferior glenohumeral ligament measures less than 4 mm and is best seen on coronal oblique images at the mid-glenoid level. In adhesive capsulitis, the axillary recess may show thickening up to 1.3 cm or more; the joint capsule is also thickened. A classical "subcoracoid triangle sign is seen" in sagittal oblique T1 weighted images.

Treatment

Although frozen shoulder is a self-limiting condition, it imposes such morbidity and lengthy recovery time that patients and clinicians alike seek treatment interventions. No standard treatment regimen, however, is accepted universally.

Conservative treatment is the mainstay of therapy and only refractory cases are subjected to operative interventions.

1. Oral analgesics: salicylates, NSAIDs, and codeine compounds help to reduce pain and inflammation in the early stages.
2. Steroid: it is good for pain, not improving ROM. This, when accompanied by local analgesics and gentle active motion, helps in the freezing stage of frozen shoulder. Hollingworth [19] reported that injection of a corticosteroid directly into the anatomical site of the lesion produced pain relief and at least 50% improvement in ROM in 26% of the cases studied. Weiser et al. [20] injected prednisolone into the shoulder joints of 100 patients, then passively mobilized the joint, and gave the patients a vigorous active home exercise program; 78% obtained pain relief, and 61% regained normal function.

   In summary, local corticosteroid injections have been used with varying results, but, generally, they produce a greater gain in motion recovery if used in combination with exercises and heat therapy.
3. Infiltration debridement: this method consists of forcibly extending the joint capsule with the contrast material that is used for arthrographic procedures.

Local anesthetics and ROM exercises may be combined with infiltration debridement to facilitate restoration of motion.

4. Physiotherapy: role of the physiotherapist—Thermotherapy may be tried before resorting to passive mobilization; the thick and contracted capsule must be released and made more stretchable by deep heating using ultrasonic or other suitable modalities. The heating is carried out throughout the joint. Passive physiological exercise: motion in a range that usually is achieved actively.

   Accessory exercise: motion between joint surfaces, which cannot be achieved actively.

   (a) Phase 1 physiotherapy: it may be used when the patient has a painful joint. A physical therapist would apply accessory movement in a comfortable joint position, with the affected arm supported in a loose-packed position. The therapist administers slow, gentle oscillatory movements in anterior-posterior and cephalad-caudad directions if they do not increase pain or induce muscle spasm. The therapist provides a mechanical block to movement short of the painful, restricted range and continues to use gentle, low-amplitude oscillations.

   (b) Phase 2 physiotherapy: it is used to treat a stiff joint. As the condition progresses, the therapist may detect stiffness before or concurrently with the onset of pain. The therapist then should begin low-amplitude physiological and accessory oscillations at the limit of the restriction. To increase abduction, for example, the therapist with caudal glide performs more powerful oscillations at the end of the accessory range.

   Role of the Patient: Home Treatment Regimen (1) Pendulum exercises*:* in a forward stooping position, with one hand resting on a table or chair, the patient gradually swings the arm like a pendulum and later carries out a circumduction movement. This is done 5 times daily in 5–10-min sessions. (2) Shoulder elevation exercises: this is done with the normal hand supporting the affected one; the shoulder is gradually lifted to a position of flexion abduction and external rotation. (3) Hand to back position: patient carries the arm backward with the shoulder in a position of extension, adduction. (4) Shoulder wheel exercises: this is to be done by the patient himself at the physiotherapy center. (5) Pulley exercises: these can be done by the patient himself at home.

5. Manipulation: closed manipulation of the shoulder under general anesthesia. It is reserved for patients who have failed to gain ROM after physiotherapy and local injections. It is also recommended in patients who refuse to wait for long for the resolution of symptoms. There is a significant improvement seen in around 70% of patients. The shoulder is manipulated using a short arm lever and a fixed scapula. The acronym FEAR can be used as a safe sequence for shoulder manipulation-flexion extension, abduction and adduction, and external and internal rotation. An audible and palpable release of adhesions is a good prognostic sign.

   Post manipulation care: Immediate exercises to be started, emphasizing the need to move the arm continuously. Circumduction, overhead bar, and pulley

exercises are begun immediately (10–20 repetitions each hour). Constant reassurance for 3 months. Counseling that ROM will improve immediately but pain may persist for 3–6 weeks. Permanent loss of 20° of flexion; internal rotation and external rotation are usual. Abduction orthosis is worn at night for 3 weeks to prevent significant axial pouch adhesions from returning in the early phase.

Complications of MUA: (1) proximal humeral fractures, (2) shoulder dislocations, (3) fracture dislocation, (4) rotator cuff ruptures, and (5) traction nerve injuries.

These can be avoided by gentle, slow manipulation. If a firm end point to motion is felt, further manipulation should not be attempted.

6. Arthroscopic release: This is for patients in whom closed manipulation fails. Rotator interval: triangular area in the anterior and superior shoulder where no rotator cuff tendons are present. It is bounded by the supraspinatus superiorly, the subscapularis inferiorly, and the coracoids medially.

   Contents: The coracohumeral ligament, biceps tendon, and superior glenohumeral ligament.

   Arthroscopic pathology (Table 2.7) in frozen shoulder

   Selective arthroscopic releases may accomplish the following gains in motion [21]: rotator interval, external rotation; inferior capsule, external rotation, flexion, and internal rotation; and posterosuperior capsule, internal rotation.
7. Hydrodilatation: moderate ST relief—high patient satisfaction.
8. Laser: strong evidence that it may be beneficial.
9. SS nerve block: it may help in pain relief [22].
10. Operative treatment: about 89.5% can be treated nonoperatively. Only 10% of patients need release at 12 months from the onset of symptoms. MUA is technically easy but with risk of fractures. Arthroscopic capsular release is precise and safe in the right setup but with surgical risks.

Disorders of the Rotator Cuff (Impingement Syndrome)

It is basically an ill-defined term for a variety of shoulder disorders that manifest as anterolateral shoulder pain with (1) nonspecific diagnosis and (2) numerous types of shoulder impingement with only a small proportion of these necessitating decompression.

Table 2.7 Varieties of arthroscopic pathology found

Structure	Findings	Treatment
Anterior triangle	Diffuse synovitis, scar, and contracture of CH ligament	Synovectomy, excision of scar and CH ligament
Subscapularis bursa	Dense adhesions	Excise adhesions
Anterior capsule	Scarring of MGLH (middle glenohumeral ligament)/IGHL	Cut MHGH/IGHL (inferior glenohumeral ligament)
Posterior capsule	Scarring of IGHL	Divide IGHL and posterior capsule
Axillary recess	Diffuse synovitis and contracture	Synovectomy and divide capsule
Subacromial bursa	Chronic bursitis and adhesions	Excise inflamed tissue, subacromial decompression as needed

The first description of subacromial bursitis was in 1867 by Jarjavay [23]. Later Codman [24], in 1931, described an inability to abduct the arm with incomplete or complete ruptures of the supraspinatus tendon, rather than primary bursal problems. The concept of outlet impingement was first coined by Meyer in 1937 [25].

In 1972, Neer described the impingement syndrome [26] which is characterized by a ridge of proliferative spurs and excrescences on the undersurface of the anterior process of the acromion, apparently caused by repeated impingement of the rotator cuff and the humeral head with traction of the coracoacromial ligament. Anterior third of the acromion and its anterior lip seemed to be the offending structure in most cases of impingement tests. Neer eventually came to argue that a vast majority, if not all, of lesions of the rotator cuff were due to subacromial impingement. The debate of extrinsic impingement versus intrinsic degeneration as the etiology of rotator cuff tears continues to this day.

Etiology

1. Extrinsic: such as primary impingement as in outlet stenosis or secondary impingement resulting in instability
2. Intrinsic: such as degeneration seen in aging or avascularity

Neer [26] described impingement of the rotator cuff by the coracoacromial ligament and the anterior third of the acromion and undersurface of the AC joint. Spurs and bony changes on the undersurface of the anterior 1/3 of the acromion correlated with rotator cuff tears.

90% of patients treated by acromioplasty had significant pain reduction, full use of the shoulder, <20% of overhead limitation, and at least 75% normal strength had RC injury is a result of primary impingement.

Bigliani, in 1986 [27], popularized the theory of "extrinsic" impingement, stating that acromial changes were primary as opposed to secondary changes causing impingement.

And 70% of full-thickness RC tears in type III acromion have never been proven or peer-reviewed.

Jobe and Kvitne, in 1989 [28], stated that secondary impingement causes glenohumeral instability, which is common in the overhead-throwing athlete leading to cocking → strain on the static stabilizers in the early phase (45-abd, ER), MGHL in the late phase (90-abd, ER), and IGHL resulting in (1) anterior soft tissue deficiency and (2) anterior translation of the humeral head.

Walch, in 1992 [29], stated that internal impingement is very common in the overhead-throwing athlete and cocking giving rise to tension on the posterosuperior articular surface of the supraspinatus, and compression between the humeral head and adjacent glenoid rim resulted in posterior superior synovitis along with partial undersurface tears.

Morgan and Rajan (2004) [30] stated that glenohumeral internal rotation deficit (GIRD) is very common in the overhead-throwing athlete leading to a posteroinferior capsular contracture at the posterosuperior humeral head translation leading to a SLAP type of internal impingement.

Scapulothoracic dyskinesis results in functional scapular instability, with an increased distance from the spinous process to the medial border of the scapula with loss of acromial elevation.

Ozaky (1988) [31] and Ogata and Uhthoff (1990) [32] stated that some of the intrinsic factors result in tendon degeneration (and fiber failure) which is the most important etiologic factor of symptoms (and cuff tears) in the subacromial space and not the impingement syndrome; partial-thickness tears are most often on the articular side, and the articular side of the cuff is hypovascularized.

Burkhart (1995) [33] emphasized that the main problem is tendon degeneration and weakness.

Most mobile joint in the body: The cuff envelops and blends with the glenohumeral capsule on all sides except at the redundant inferior pouch. The tendon of the long head of the biceps—"fifth tendon" of the cuff-intraarticular but extrasynovial structure—passes deep in the "rotator interval."

Rotator interval: The superficial roof of the rotator interval (Fig. 2.5) is the coracohumeral (CH) ligament and the floor of the interval is the superior glenohumeral ligament (SGHL). It is also called the suspensory structure for the humeral head.

The subacromial bursa is a filmy synovium-lined sac which has been described as Matsen's concept of the "humeroscapular articulation." Its main function is

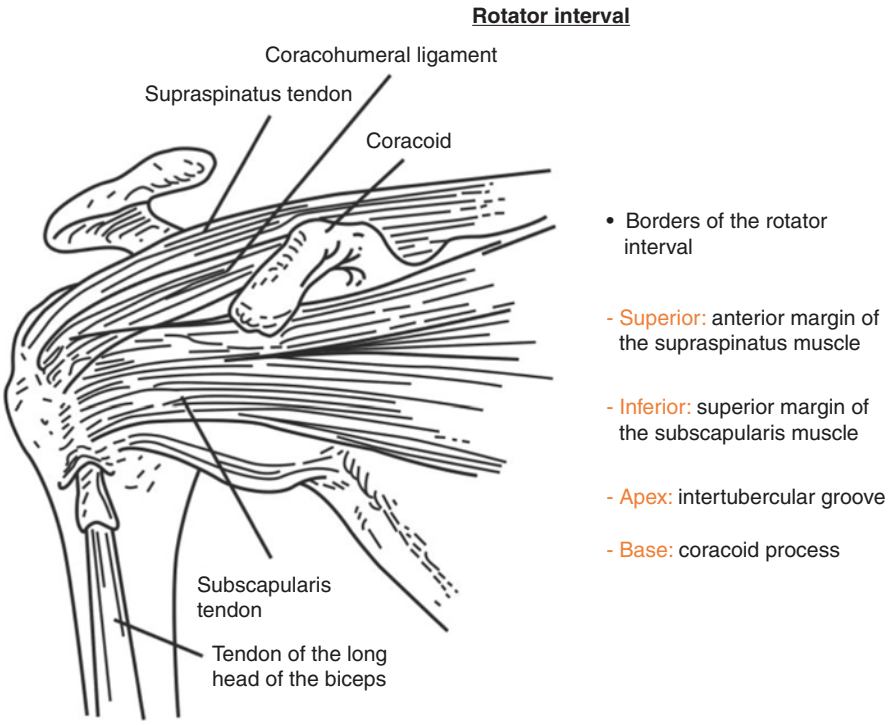

Fig. 2.5 Line diagram of the rotator interval

primarily as a stabilizer. The pain is of insidious onset which is exacerbated by overhead activities and lifting objects away from the body. It is a night pain which is a poor indicator of successful nonoperative management. The strength is usually normal.

A clinical assessment which is of a low specificity is carried out by the (1) Neer impingement test, (2) lidocaine injection test, and (3) Hawkins' impingement test. It always evaluates the AC joint by palpation, cross arm test, and injection. An imaging assessment is also carried out by plain X-rays (anteroposterior view, axillary lateral view, and supraspinatus outlet view) including axillary, outlet, and Zanca to complete this assessment. Further an MRI and a CT arthrogram are done. Radiographs may reveal exostoses of the greater tuberosity cysts or sclerosis, subacromial sclerosis (sourcil sign), which indicate chronic cuff tears.

Additionally, superior migration of the humeral head with narrowing of the acromiohumeral space to less than 7 mm suggests a rotator cuff tear, and a space less than 5 mm suggests a massive tear.

A Stryker notch view may be helpful in some cases when a small contour defect may be due to a Hill-Sachs impression fracture.

A West Point view done which is similar to the axillary view, when the patient is prone instead of supine, is helpful to determine glenoid fractures.

Arthrogram: Traditionally, an arthrogram has been used to document full-thickness rotator cuff tears. Leakage of contrast material into the subacromial and subdeltoid spaces after injection into the glenohumeral joint indicates a full-thickness tear. Arthrography is still useful for patients in whom MRI is contraindicated. An arthrography can be combined with MRI to improve the diagnostic accuracy.

MRI: A patient with symptoms of subacromial impingement may show increased signal in the supraspinatus tendon on T2-weighted MRI consistent with tendinopathy; increased fluid in the subacromial bursa also is a sign of subacromial impingement.

A dynamic ultrasound also can be useful in confirming shoulder impingement syndrome, assessing glenohumeral laxity, and identifying biceps tendon pathology.

There are three stages of impingement, namely, (1) inflammation and edema, (2) fibrosis and tendinitis, and (3) partial or complete tearing.

Developmental Stages of Impingement Syndrome

- Stage 1: edema and hemorrhage. Typical age of patient: <25 years old. Differential diagnosis: subluxation or AC joint arthritis. The clinical course is reversible and the treatment is conservative.
- Stage 2: fibrosis and tendinitis. Typical age of patient: 25–40 years old. Differential diagnosis: frozen shoulder or calcium deposits. The clinical course is recurrent pain with activity and treatment: consider bursectomy or division of coracoacromial ligament.
- Stage 3: bone spurs and tendon rupture. Typical age of patient: >40 years old. Differential diagnosis: cervical radiculitis or neoplasm. The clinical course is one of progressive disability and the treatment is anterior acromioplasty with/without rotator cuff repair.

There are four types of impingement, namely, (1) primary impingement, (2) secondary impingement, (3) subcoracoid impingement, and (4) internal impingement.

Primary impingement without any other contributing pathology may be intrinsic or extrinsic.

Subacromial impingement is thought to be a combination of (a) extrinsic compression due to the rotator cuff between the humeral head and anterior acromion, coracoacromial ligaments, and acromioclavicular joint and (b) intrinsic degeneration of supraspinatus.

Secondary intrinsic impingement causes instability of the glenohumeral joint translation of the humeral head, typically anteriorly contact of the rotator cuff against the coracoacromial arch.

Examples of secondary impingement are (1) thickening of the rotator cuff, (2) calcium deposits within the rotator cuff, and (3) thickening of the subacromial bursa.

Associated conditions may be (1) hook-shaped acromion, (2) os acromiale, (3) posterior capsular contracture, (4) scapular dyskinesia, (5) tuberosity fracture malunion, or (6) instability.

Subcoracoid impingement: This was first described in 1909 by Goldthwait [34]. It consists of a prominent coracoid which may be idiopathic and iatrogenic and is treated by Trillat osteotomy. Park et al. [35] in their series of 475 patients with rotator cuff tears comprising subcoracoid impingement in 13%. Among patients with subscapularis tears, 56% had subcoracoid impingement. Among patients with subacromial impingement but no rotator cuff tears, 41% had subcoracoid impingement. The physical findings were tenderness over the coracoids with a positive coracoid impingement test. An injection of lidocaine into the subcoracoid region similar to the Neer impingement test and a CT suggesting a distance of 6.8 mm between the coracoid tip and the closest portion of the proximal humerus indicates impingement.

Internal impingement: This occurs when the internal contact of the rotator cuff occurs with the posterosuperior aspect of the glenoid. The arthroscopic findings include (1) partial rotator cuff tears, (2) posterior and superior labral tears, and (3) anterior shoulder laxity.

Treatment

The primary goal is to restore the health of the rotator cuff: restoration of the neuromuscular balance of the shoulder girdle and the synchronous motion of the three joints about the shoulder to prevent subluxation.

Initially a nonoperative regimen consists of anti-inflammatory medications with one or at most two subacromial cortisone injections plus a physical therapy program focusing on stretching for full-shoulder motion and strengthening the rotator cuff.

If the patient fails to respond after 3–4 months of conservative therapy, then operative intervention may be indicated and should be directed to the specific lesion.

In short nonoperative treatment may be a modification of activity with cessation of overhead activity, NSAID or subacromial injection, and physical therapy (stretching/strengthening) and acute injury with profound strength loss: MRI of the rotator cuff is indicated.

Surgical Treatment: This can be considered with failed conservative treatment of longer than 6 months, acromial prominence/spurs/sclerosis, + impingement sign, arc of pain, relief from subacromial injection, no evidence of RCT (+/- MRI), or an arthroscopic subacromial decompression.

The surgical treatment of choice for impingement syndrome is arthroscopic or open acromioplasty.

The end-point assessment is as follows: (1) Is the CA ligament released? (2) Is the acromion flat in the AP plane? (3) Is the acromion flat in the ML plane? (4) Is the AC joint inspected? (5) Is the rotator cuff inspected?

Ellman, in 1987 [36], reported on the results of subacromial decompression stating that it is a technically demanding procedure and also adding that if a cuff tear is present, you cannot repair it.

Surgical Technique: It is carried out with the patient in a beach chair position. The bony landmarks of acromion, coracoid, and ACJ are identified. A posterior portal is used which is the same skin incision for GHJ arthroscopy, because it has the advantage of redirecting the cannula, to aim and advance the scope beneath the anterolateral corner. Room with a view shows clear visualization with four walls, floor, and ceiling. The CA ligament is landmark. The lateral portal skin marking bisects mid AC, 3 cm lateral to the acromion.

Spinal needle is placed underneath the anterior half of the acromion and parallel to it. Introduce a power shaver through the lateral portal and perform a bursectomy. Expose the undersurface of the acromion, release the CA ligament to define the anterior and lateral edges. Use an electrocautery to remove soft tissues from the undersurface of the acromion and from the distal clavicle to excise the CA ligament. Perform a "provisional" anterior acromioplasty. Perform the final acromioplasty using the "cutting block" technique. Flatten the acromion from posterior to anterior. Remember to watch the angle. Check acromioplasty in both planes post to ant and med to lat.

Distal Clavicle Resection: The scope lateral/instrument anterior is the best access to the distal clavicle with a 70° arthroscope and with 8–10 mm excision of the distal clavicle.

Perform End-point Assessment: (1) Is the CA ligament adequately released? (2) Is the acromion flat in the AP plane? (3) Is the acromion flat in the ML plane? (4) Is the AC joint inspected? (5) Is the rotator cuff inspected?

Odenbring et al. [37] reported the long-term outcomes of arthroscopic acromioplasty for chronic shoulder impingement syndrome.

A prospective cohort study with a minimum of 12 years' follow-up showed 31 patients 12–14 years' follow-up 29 patients (open acromioplasty) as a control group had no full-thickness cuff tears.

They summarized it as follows: Arth group: revision acromioplasty in six patients

Open group: revision acromioplasty in three patients; good and excellent results in 77% and better results with arthroscopic acromioplasty.

Stephens et al. [38]: Arthroscopic acromioplasty, a 6- to 10-year follow-up of 83 patients, mean follow-up of 8.3 years.

Stephens et al. [38] studied arthroscopic acromioplasty, a 6- to 10-year follow-up of 83 patients, mean follow-up of 8.3 years, and came to the conclusion that "Overall, 81% of patients in their series had good to excellent results after 6 to 10 years," and hence "To optimize the indications for the procedure, other causes of impingement, such as occult instability and degenerative joint disease, should be ruled out."

Kartus et al. [39] reported on long-term clinical and ultrasound evaluation after arthroscopic acromioplasty in patients with partial rotator cuff tears and came to the conclusion that "Arthroscopic acromioplasty and rotator cuff debridement in patients with partial tears does not protect the rotator cuff from undergoing further degeneration."

Chahal et al. [40] concluded that "On the basis of the currently available literature, there is no statistically significant difference in subjective outcome after arthroscopic rotator cuff repair with or without acromioplasty at intermediate follow-up."

Familiari et al. [41]: Conclusions: (1) "does not support the routine use of partial acromioplasty or CA ligament release in the surgical treatment of rotator cuff disease" and (2) "in some instances, partial acromioplasty and release of the CA ligament can result in anterior escape and worsening symptoms"

Clement et al. [42] concluded that "Functional outcome of patients with calcific tendonitis after arthroscopic bursectomy and debridement of the calcific deposit is not influenced if performed in combination with or without a subacromial decompression."

Inclusion: "The emphasis of treatment is shifting from that of decompression to restoring the health of the rotator cuff" [43].

Complications after acromioplasty which are not limited to infection are as follows:

1. Seroma formation
2. Hematoma
3. Synovial fistula
4. Biceps rupture
5. Pulmonary embolus
6. Acromial fracture

Shoulder Arthroplasty

Lesions of the shoulder requiring arthroplasty are far less common than lesions involving the weight-bearing joints of the body, commonly such as the hip and knee. Osteoarthritis of the hips is frequently seen in western countries while osteoarthritis of the knees is seen more often in eastern countries like India.

The characteristics of the shoulder are as follows: (1) the greatest ROM, (2) no inherent bony stability, (3) relies on soft tissues for stability, and (4) many injuries involving the soft tissues mainly such as the rotator cuff and labrum and has little glenoid bone stock.

Indications for Shoulder Arthroplasty

1. Rheumatoid arthritis
2. Osteoarthritis
3. Posttraumatic (complex fractures)
4. Rotator cuff tear arthropathy (reverse shoulder replacement)
5. Instability
6. Tumors

The common arthroplasty options are (1) hemiarthroplasty, (2) total shoulder replacement, and (3) reverse total shoulder replacement.

Pros and cons (Table 2.8) of various types of shoulder replacements

1. Hemiarthroplasty (Fig. 2.6) of the Shoulder: AP and Axial Views of Hemiarthroplasty of the Shoulder. (Courtesy: Dr Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopedic Hospital, Birmingham, UK.)
2. Total Shoulder Arthroplasty. AP and Axial Views of Total Shoulder Arthroplasty (Fig. 2.7). (Courtesy: Dr. Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopedic Hospital, Birmingham, UK.)
3. Reverse Total Shoulder Arthroplasty: AP and Axial Views of Reverse Total Shoulder Arthroplasty (Fig. 2.8). (Courtesy: Dr. Rajesh Boitchu, Consultant MSK Radiologist, Royal Orthopedic Hospital, Birmingham, UK.)

Surgical Approach

Deltopectoral Approach: The interval is between the deltoid and pectoralis major muscle. Cephalic vein is retracted laterally. The subscapularis muscle is then split preserving the most inferior fibers to protect the axillary nerve.

Deltoid Splitting Approach: In this approach, deltoid splitting is done up to 5 cm below the acromion to protect the axillary nerve traversing in the substance of the

Table 2.8 Various types of shoulder replacement

Shoulder replacement	Pros	Cons
Hemiarthroplasty	• Humeral head OA • Replaces humeral head	• Not useful if glenoid is also osteoarthritic
Total shoulder	• Replaces the glenoid and humerus head • Needs intact rotator cuff to function • Good bone stock needed • Normal biomechanics is maintained	• Not useful if rotator cuff is torn or deficient
Reverse shoulder	• Can be done in rotator cuff-deficient patients	• Ball and socket are reversed • Relies on deltoid function • Biomechanics of shoulder changed

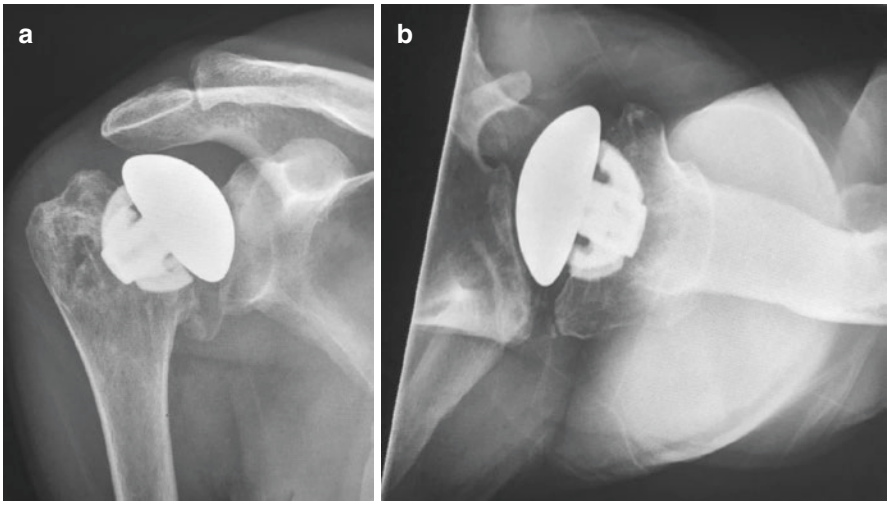

Fig. 2.6 X-rays showing hemiarthroplasty of the shoulder

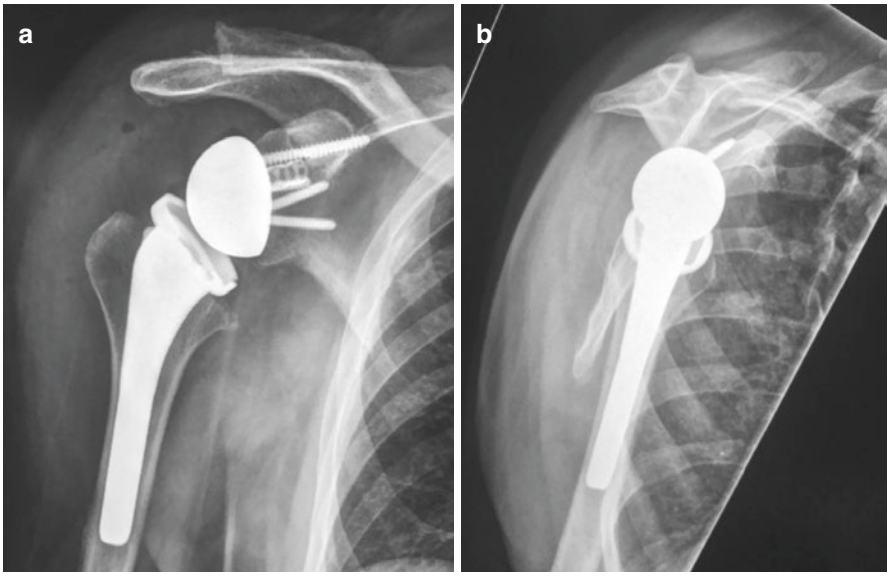

Fig. 2.7 X-rays showing total shoulder arthroplasty

muscle. Supraspinatus tendon is exposed and allows for repair of the rotator cuff and further exposure of the head for resurfacing or hemiarthroplasty.

Surface replacement is another option. It has evolved over the years. The surface of the head of the humerus is replaced with a hydroxyapatite-coated implant, and it ensures a more anatomical replacement as glenoid remains native without replacement. Survivorship is more than 90% at 10 years.

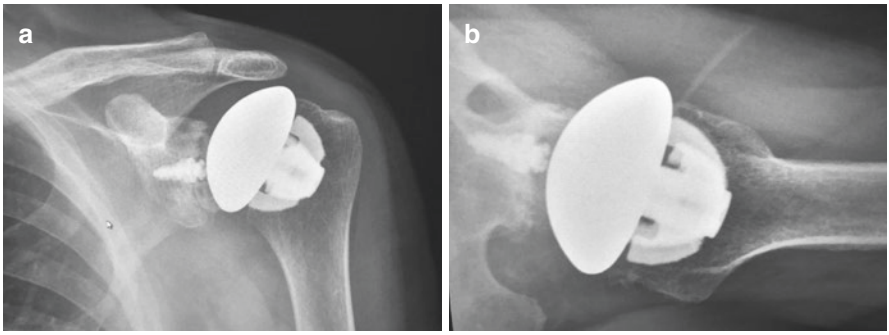

Fig. 2.8 X-rays showing reverse total shoulder arthroplasty

History of Replacement: In 1893, French surgeon Pean inserted platinum and rubber components to replace a shoulder joint destroyed by tuberculosis. In 1951, Neer I used vitallium hemiarthroplasty prosthesis which resulted in pain relief and good function compared to previous options. In 1974, Neer II prosthesis modified Neer I to conform to a glenoid component. In the 1970s, constrained components were popular, but follow-up reports demonstrated high rates of loosening, particularly of the glenoid component. In the 1980s, modular humeral components were developed, along with cementless glenoid fixation using polyethylene on a metal backing.

Other problems with metal-backed glenoid components were as follows:

1. Metal backing increased the thickness of the component and often led to overstuffing of the joint.
2. To avoid overstuffing of the joint, the polyethylene thickness had to be reduced, resulting in accelerated poly wear and failure.
3. Poly-metal disassociation occurred with unacceptable frequency.

As regards humeral components:

(a) Cemented: (1) it was good for an osteopenic bone, (2) with a lower risk of intra-operative fracture and (3) more stress shielding, and (4) hard to revise.
(b) Prox porous coated: it needed a good bone stock. There was a higher risk of intra-operative fracture. It was less stress shielding but easier to revise.
(c) Fully porous coated: it also needed a good bone stock. There was a higher risk of intra-operative fracture. They are more stress shielding and hard to revise.

Cemented vs. Press-Fit Humeral Components

At first Harris, Jobe, and Dai reported less micromotion with proximally cemented stems.

The fully cemented stems provide no additional benefit or stability over proximally cemented stems. It was then that Sanchez-Sotelo reported a low rate of stem loosening regardless of fixation, but press-fit prostheses developed more radiolucent lines in the first 4 years.

Need for Modularity
1. F-H offset.
2. B-C head thickness.
3. D-E = 8 mm top of the humeral head is higher than the greater tuberosity.
4. Reestablishing normal glenohumeral anatomic relationships is important to ensure optimal results.

Other Anatomic Variables to Consider
1. Glenoid: 2° anteversion to 7° retroversion.
2. Humeral head: 20–40° retroversion.
3. Axial CT of the glenohumeral joint is a valuable pre-op planning tool.

Contraindications to shoulder arthroplasty are as follows:

1. Active or recent shoulder joint infection.
2. Paralysis with complete loss of rotator cuff and deltoid function.
3. A neuropathic arthropathy.
4. Irreparable rotator cuff tear is a contraindication to glenoid resurfacing.

Osteoarthritis of the Shoulder

In addition to the universal features of osteoarthritic joints (joint space narrowing, cysts, osteophytes, etc.), the shoulder can also demonstrate (1) posterior glenoid erosion, (2) flattening of the humeral head, (3) enlargement of the humeral head, and (4) rotator cuff tears which are uncommon in OA.

Recommendation Based on Experience

Neer (1998) [44]: "When the articular surface of the glenoid is good, the results of hemiarthroplasty are similar to those of TSA. Wear on the glenoid has not been a problem if the articular surface was good at the time of surgery and glenohumeral motion was re-established".

Recommendations Based on Evidence
(a) Kirkley et al. (2000) [45]: Their results were on 42 patients with 3 surgeons (stratified) with a 1-year follow-up. There was no significant difference in WOSI, ASES, DASH constant score, or ROM. They trend toward better pain relief with TSA. They only noticed that two hemi patients crossed over to TSA after 1-year follow-up.
(b) Gartsman (2000) [46]: They had 51 shoulders with an average f/u of 35 months. There were no differences in ASES or UCLA scores. There was significantly better pain relief with TSA in their series. Three patients crossed over to TSA by 35 months.

Recommendations Based on Evidence
1. Similar improvement in pain and function in both groups if the rotator cuff was intact. Better results with hemi if with a rotator cuff tear.

2. Hemi: revision at mean of 1.5 years for glenoid pain
3. TSA: revision at mean of 4.5 years for glenoid loosening

Important Technical Issues to Consider

1. OA tends to result in posterior glenoid wear/erosion, which, if accepted, will lead to a retroverted glenoid component.
2. Compensate by anteriorly reaming or placing the humeral component in less retroversion.
3. Failure to do so will result in posterior instability.

Rheumatoid Arthritis

(1) Peri-articular erosions, (2) peri-articular osteopenia, (3) thin cortices, and (4) adjacent joint involvement are notable factors to be kept in mind.

In cemented short-stemmed prosthesis, Gill et al. recommend at least 60 mm between the cement mantles of the ipsilateral shoulder and elbow arthroplasties. If this cannot be achieved, join both cement mantles together.

Generally, TSA is performed due to destruction of the glenoid articular surface by the disease.

Glenoid erosion may require bone grafting; however, if the glenoid is eroded to the level of the coracoid process, glenoid resurfacing is contraindicated.

Rotator Cuff Arthropathy

It was initially described by Neer, Craig, and Fukada (1983) [47]. A distinct form of osteoarthritis is associated with a massive chronic rotator cuff tear. Generally, rotator cuff tears occur in less than 10% of shoulders with OA.

A function of the rotator cuff is to depress the humeral head and keep it centered on the glenoid fossa. Massive rotator cuff tears result in proximal migration of the humeral head.

This is a contraindication to glenoid resurfacing as it results in eccentric (superior) glenoid loading and early component loosening.

Surgical Options

1. Hemiarthroplasty with a large head
2. Repair of the rotator cuff and TSA
3. Reverse TSA

One must also take into consideration the “Clayton Spacer.”

Outcomes of Hemiarthroplasty

1. Rockwood: 86% satisfactory results after 4 years.
2. Zuckerman: 93% adequate pain relief and 90% improved function for ADLs.
3. Sanches-Sotelo: 75% modest improvements in ROM and strength for ADLs. Good pain relief.

4. Field et al. and Sanchez-Sotelo reported that impaired deltoid function and previous subacromial decompression (loss of coracoacromial ligament) were significantly associated with clinical shoulder instability post hemiarthroplasty.

Reverse Total Shoulder Arthroplasty

It lateralizes the center of rotation and places the deltoid at a mechanical advantage.

It has more inherent stability and prevents proximal migration of the humeral head.

Reverse total shoulder arthroplasty is hard to revise with little glenoid bone stock once the component is removed.

Osteonecrosis of the Shoulder

Causes: (1) corticosteroids, (2) alcoholism, (3) sickle cell disease, (4) lupus, and (5) idiopathic.

It usually involves young patients with adequate bone stock. Prefer proximally porous-coated, press-fit humeral prosthesis because it is less stress shielding and easier to revise if necessary. Only resurface glenoid in stage V osteonecrosis (glenoid erosion).

Posttraumatic Arthritis

It is usually due to fractures treated conservatively. It may also have malunion of tuberosities, distorting normal anatomic landmarks. Nearly 12% of patients have axillary nerve palsies (Neer) and many have soft tissue contractures and muscle weakness.

The choice of prosthesis depends on the patient's age and the condition of the glenoid surface and bone stock. Axillary nerve palsy is a relative contraindication to arthroplasty.

Complications

1. Instability: 1.2% due to excessive retro/anteversion, the head being too small or too low (post fracture), or subscap rupture.
2. Rotator cuff tear: 2% which results in superior migration of the humerus and glenoid loosening.
3. Glenoid loosening.
4. Infection: 0.5% usually due to *Staphylococcus aureus* and more common after revision surgery.
5. Heterotopic ossification: 10–45%. It is common in males with a Dx of osteoarthritis. It is low grade and nonprogressive and does not affect the outcome.
6. Stiffness which depends on the indication for arthroplasty. There is usually subscap shortening. Oversized components or inappropriate rehab.
7. Periprosthetic fracture: intra-op 1% and post-op 0.5–2%. It is most common in RA, 85% are women, and glenoid fractures are rare.
8. Axillary nerve injury: it is rare and has a higher risk during revision surgery which is usually a neuropraxia.

   Ultimate bail-out or salvage procedure is either an excision arthroplasty or shoulder arthrodesis.

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The Arm

3

K. Mohan Iyer

Arm

The arm extends from the shoulder to the elbow. Two types of movement occur between the arm and forearm at the elbow joint: flexion-extension and pronation-supination. The muscles performing these movements are clearly divided into anterior and posterior groups.

The skeletal support for the arm is the humerus. Most of the large muscles of the arm insert into the proximal ends of the two bones of the forearm, the radius, and the ulna and flex and extend the forearm at the elbow joint. In addition, the muscles predominantly situated in the forearm that move the hand originate at the distal end of the humerus.

Muscles of the arm: (a) The anterior compartment of the arm (Table 3.1) contains three muscles, namely, (1) the coracobrachialis, (2) brachialis, and (3) biceps brachii; (b) the posterior compartment (Table 3.2) contains one muscle, namely, the triceps brachii.

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Muscles of the Arm

Anterior Compartment (Table 3.1)

Posterior Compartment (Table 3.2)

Table 3.1 Muscles of the arm (anterior compartment)

Muscle	Origin	Insertion	Innervation	Action
1. Coracobrachialis	Tip of the coracoid process of the scapular bone	Linear roughening on mid-shaft of humerus on medial side	Musculocutaneous nerve [C5, C6, C7]	Flexor of the arm at the glenohumeral joint
2. Biceps brachii	Long head supraglenoid tubercle of scapula; short head—apex of coracoid process Musculocutaneous nerve [C5, C6, C7] Powerful	Radial tuberosity	Musculocutaneous nerve [C5, C6, C7]	Powerful flexor of the forearm at the elbow joint and supinator of the forearm; accessory flexor of the arm at the glenohumeral joint
3. Brachialis	Anterior aspect of humerus (medial and lateral surfaces) and adjacent intermuscular septa	Tuberosity of the ulna	Musculocutaneous nerve [C5, C6, C7]; a small portion of its lateral part may be innervated by the radial nerve	Powerful flexor of the forearm at the elbow joint

Table 3.2 Muscles of the arm (posterior compartment)

Triceps brachii	1. Long head: infraglenoid tubercle of scapula; 2. Medial head posterior surface of humerus; 3. Lateral head posterior surface of humerus	Superior surface of the olecranon of the ulna bone	Radial nerve [C6, C7, C8]	Extension of the forearm at the elbow joint Long head can also extend and adduct the arm at the shoulder joint

Arteries and Veins of the Arm

Brachial artery: It is the major artery of the arm and is found in the anterior compartment.

It begins as a continuation of the axillary artery at the lower border of the teres major muscle and terminates just distal to the elbow joint where it divides into the radial and ulnar arteries.

In the proximal arm, the brachial artery lies on the medial side. In the distal arm, it moves laterally to assume a position midway between the lateral epicondyle and the medial epicondyle of the humerus. It crosses anteriorly to the elbow joint where it lies immediately medial to the tendon of the biceps brachii muscle.

Branches of the brachial artery in the arm include those to adjacent muscles and two ulnar collateral vessels, which contribute to a network of arteries around the elbow joint.

Additional branches are the profunda brachii artery and nutrient arteries to the humerus, which passes through a foramen in the anteromedial surface of the humeral shaft.

Profunda brachii artery: It is the largest branch of the brachial artery and passes into and supplies the posterior compartment of the arm. It enters the posterior compartment with the radial nerve and together they pass through the triangular interval, which is formed by the shaft of the humerus, the inferior margin of the teres major muscle, and the lateral margin of the long head of the triceps muscle. They then pass along the radial groove on the posterior surface of the humerus deep to the lateral head of the triceps brachii muscle.

Veins: Paired brachial veins pass along the medial and lateral sides of the brachial artery, receiving tributaries that accompany branches of the artery. In addition to these deep veins, two large subcutaneous veins, the basilic vein, and the cephalic vein are located in the arm. The basilic vein passes vertically in the distal half of the arm, penetrates the deep fascia to assume a position medial to the brachial artery, and then becomes the axillary vein at the lower border of the teres major muscle. The brachial veins join the basilic, or axillary, vein. The cephalic vein passes superiorly on the anterolateral aspect of the arm and through the anterior wall of the axilla to reach the axillary vein.

Nerves of the arm: These four nerves are found within the anterior and posterior compartments of the arm: (1) musculocutaneous nerve, (2) median nerve, (3) ulnar nerve, and (3) radial nerve.

1. Musculocutaneous nerve (C5–C7): It is a large terminal branch of the lateral cord. It enters the arm by passing through the coracobrachialis muscle. It passes diagonally down the arm in the plane between the biceps brachii and brachialis

muscles. After giving rise to motor branches in the arm, it emerges laterally to the tendon of the biceps brachii muscle at the elbow, penetrates the deep fascia, and continues as the lateral cutaneous nerve of the forearm.

2. Other nerves (median, ulnar, and radial nerves): Both the median and ulnar nerves pass through the arm into the forearm with no branches to the arm. The radial nerve originates from the posterior cord of the brachial plexus; it enters the arm after crossing the inferior margin of the teres major muscle lying posterior to the brachial artery and accompanied by the profundal brachial artery to the posterior aspect of the arm.

Pathology

Pathology of the proximal biceps brachii tendon: (1) Proximal biceps brachii tendon is generally referred to as the long head of the biceps tendon (LHBT). (2) The LHBT is recognized as an important pain generator of the shoulder. (3) Its pathology is rarely an isolated entity and frequently is correlated with rotator cuff (RC) and labral pathology. (4) Trauma, degeneration, overuse, subluxation, and dislocation of the LHBT may occur simultaneously with RC pathology. (5) They need to be identified and treated at the same time.

Anatomy of the LHBT: It has a complex anatomy with relation to the RC of the shoulder. It has an intra-articular (intracapsular) and extra-articular (extracapsular) portion. The intracapsular portion is through the biceps pulley, and the extracapsular through the bicipital groove (Fig. 3.1).

The LHBT has a total length of 9–10 cm and a diameter of 5–6 mm. The LHBT arises from the supraglenoid tubercle of the scapula and partly from the superior glenoid labrum (biceps anchor) with a 50% anatomic variation from the posterior glenoid. The biceps anchor has a broad origin from the supraglenoid tubercle and the superior labrum at the 12 o'clock position. The supraglenoid tubercle is about 5 mm medial to the superior edge of the glenoid rim. It has a variable origin with some

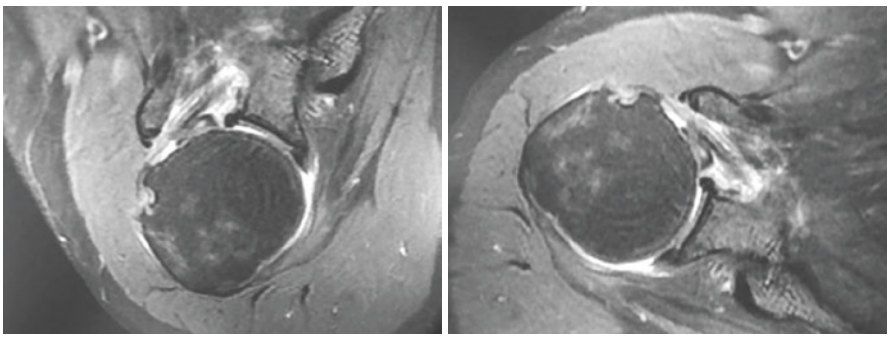

Fig. 3.1 Proton density fat saturated (PDFS) axial showing absence of LHBT in the bicipital groove—rupture of LHBT. (Courtesy: Dr. Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopedic Hospital, Birmingham, UK)

reports of 100% origin from the labrum alone. The intracapsular portion (flattened and larger) is stabilized by the biceps pulley (runs along the lateral half of the rotator interval). The biceps pulley is formed by the superior glenohumeral ligament (SGHL), coracohumeral ligament (CHL), and fibers of the supraspinatus (SSt) and subscapularis (SSc) tendons. The intracapsular portion undergoes an abrupt angulation of 30–40° fusing inferiorly to the bicipital groove; the extracapsular portion (rounded and smaller in caliber) is stabilized by the transverse humeral ligament and pectoralis major tendon; the bicipital groove is about 4 mm in depth with a 56° medial wall angle.

The arterial supply is described by Cheng et al. [1] from three potential sources:

1. Branches of the brachial artery
2. Thoracoacromial artery
3. A rare variant of the circumflex humeral artery

The tendon has a hypovascular zone at 1.2–3 cm from its origin and an avascular zone within the bicipital groove at its deeper sliding portion (composed of fibrocartilage).

The sensory and sympathetic innervation network described by Alpantaki et al. [2] is distributed asymmetrically and plays a role in the pathogenesis of shoulder pain during LHBT pathology.

Function: The LHBT, a multifunctional tendon at the shoulder and elbow level, has a close anatomic relation with the rotator cable of the shoulder (as described by Burkhart et al. [3], forming the suspension bridge model of the shoulder) and contributes to shoulder stability.

It is primarily a flexor and supinator of the elbow. At the shoulder level, it is a dynamic stabilizer of the humeral head and works as a (1) depressor (superior stabilizer) and compressor of the humeral head into the glenoid cavity which centers the head into the cavity, (2) limiter of external rotation, and (3) lifter of the glenoid labrum while in abduction.

Clinical Pathology

The LHBT has a wide range of patient groups and is correlated with RC and labral pathology in heavy laborers and weightlifters, overhead throwers, baseball pitchers, and discus and lance throwers.

Most lesions happen during the maximum external rotation and the deceleration phase of the throw.

LHBT lesions also appear in parachuters while trying to control the parachute slings; overuse, repetitive traction, friction, and glenohumeral rotation are the mechanical causes of lesion.

In ages greater than 45, the main cause is degenerative lesions.

Many authors tried to classify LHBT pathology based on anatomic location, inflammatory and degenerative changes, instability and dislocation, and arthroscopic real-time evaluation findings.

A simplified classification proposed by Cheng et al. includes six types of lesions, as follows:

- Type I: Tendinopathy
- Type II: Subluxation
- Type III: Dislocation
- Type IV: Partial tears
- Type V: Complete rupture
- Type VI: Superior labrum anterior posterior (SLAP) lesions

An alternative classification is intracapsular and extracapsular lesions, as follows:

- Intracapsular: SLAP tears, tendinopathy, subluxation, and dislocation
- Extracapsular: Lesions at the level of the bicipital groove (tenosynovitis, "hourglass" biceps)

The clinical presentation of the patient with LHBT pathology is similar to that of a patient with RC lesions.

Anterior shoulder pain and impaired function as a result of overuse or acute trauma are the most common symptoms. Pain may occur at rest or be worse at night and may radiate distally into the muscle. A "click" may be present in the case of instability and in the case of overhead throwers during the throw phase. The tendon may be palpable at the bicipital groove area.

Clinical evaluation and trials:

1. Yergason's test.
2. Speed's test.
3. Uppercut trial—Positive if pain presents to the bicipital groove area or unable to execute an uppercut punch under resistance. Indicates biceps tendinopathy and/or instability and possible SSc tear.
4. O'Brien's test (labral tear)—Arm forward to 90°. Elbow fully extended. Arm adducted to 10° across the body with the thumb down. Apply downward pressure against the patient's resistance. Repeat with the thumb up. A positive sign is suggestive of a labral tear if there is more pain with the thumb down.
5. Bear hug trial—Patient cannot hold the hand against the shoulder as the examiner applies an external rotation force. Indicates SSc tear and biceps instability.
6. Belly press trial—Hands on the abdomen, elbows out. Press in on the abdomen or keep elbows out while a posteriorly directed force is applied to the elbows. Positive test if unable to keep elbows out (inability to keep the humerus in internal rotation). Indicates SSc tear and biceps instability.

In conclusion:

Generally there's no absolute reliability to clinical tests. Authors compared the clinical test results to real-time arthroscopic evaluation and came to a conclusion: when the results are positive, it is quite reliable that some shoulder pathology is present.

The sensitivity and specificity for SLAP tears are as follows [4]:

1. Speed's test: The sensitivity test is 20% and the specificity test is 78%.
2. Yergason's test: The sensitivity test is 12% while the specificity test is 95%.
3. O'Brien's test: The sensitivity test is 67% while the specificity test is 37%

Type I: Tendinopathy

It is usually the result of degenerative changes: fibrosis, chronic inflammation, scar tissue, fibrotic thickening, and collagen disorganization. It may be the result of LHBT instability or partial tears. There is degeneration with age (over 40–45) with the youngsters involved mostly due to overuse and subacromial impingement. It can also be caused by mechanical causes (traction, friction, shearing forces) and bicipital groove lesions resulting in "hourglass" biceps. "Hourglass" biceps: hypertrophic tendinopathy results in entrapment of the LHBT within the bicipital groove. The "hourglass" biceps, unable to slide through, cause "locking" of the shoulder in abduction or forward flexion (authors compare it with a "trigger" finger at the shoulder level).

A rare case report of brachialis tendinopathy and/or paratendinitis should be considered in the differential diagnosis for the antecubital fossa and should be evaluated with axial fat-suppressed MRI. Recognition of this pathology can allow ultrasound-guided injection to be performed for therapeutic purposes [5].

Types II and III: Subluxation and Dislocation (Instability)

Subscapularis tears are most commonly associated with LHBT instability and rotator interval may also be correlated with LHBT instability. There is a traumatic injury mechanism or may be a fall on an outstretched arm with full internal or external rotation and backward fall on a fully extended arm with elbow impact and direct anterior shoulder impact which could lead to biceps tendinopathy. Hence Walch and Bennet proposed a four- and five-type pattern of LHBT instability, respectively; a recently modified pattern by Habermeyer proposes six types of instability (subluxation and intra- and extra-articular dislocation).

Types IV and V: Partial/Complete Rupture of the LHBT

A normal LHBT can endure high traction forces (667–890 N) and rupture of a healthy LHBT is rare. It can occur in weightlifters and males aged 50 or older and usually partial-/full-thickness ruptures are associated with underlying shoulder pathology (degeneration, impingement, RC and/or SLAP tears, tendon instability). The most common point of rupture is the hypovascular area (1.2–3 cm from the anchor) or proximal to the bicipital groove ("groove entry lesion"). All partial-thickness tears cause shoulder pain and dysfunction and complete rupture of a partial tear may actually relieve pain. The most characteristic sign of a complete rupture is "Popeye deformity" as the LHBT slides distally to the groove forming a muscle bulge of the biceps. A complete rupture on "hourglass" biceps may not present that sign.

*All Types I to VI with Figures

- Type I: Tendon displacement—subscapularis tendon (SScT) tear alone. Medial shift or minor subluxation of the biceps tendon secondary to a partial intrasubstance or anterior tear of the subscapularis tendon with intact medial ligament component of the biceps pulley (Fig. 3.2).

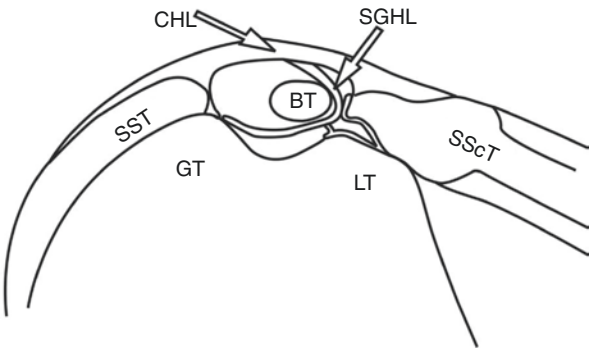

Fig. 3.2 Line diagram of Type I LHBT degeneration/tear (SScT—subscapularis tendon tear alone)

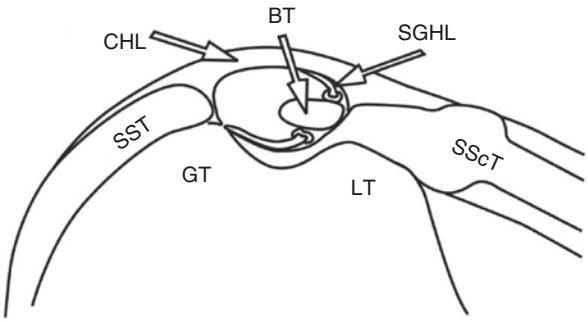

Fig. 3.3 Line diagram of Type II lesion (slightly greater medial subluxation of the biceps tendon through the torn portion of the ligaments)

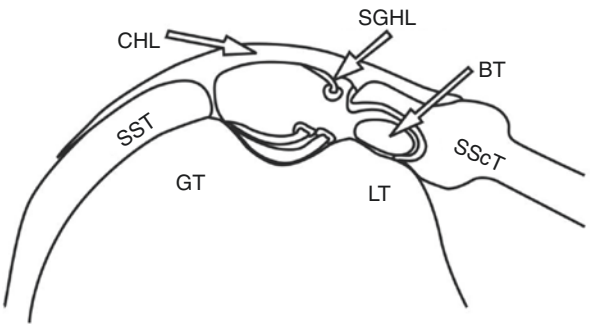

Fig. 3.4 Line diagram of Type III lesion

- Type II: Tendon displacement—medial ligament tears alone. Slightly greater medial subluxation of the biceps tendon through the torn portion of the ligaments, but the intact subscapularis tendon fibers prevent medial dislocation (Fig. 3.3).
- Type III: Extra-articular tendon dislocation—tears of the medial ligaments and subscapularis tendon. A partial intrasubstance tear of the subscapularis tendon allows the biceps tendon to dislocate medially without entering the joint because of the intact deep fibers of the subscapularis tendon (Fig. 3.4).
- Type IV: Extra-articular tendon dislocation—tears of the lateral limbs of the ligaments with an intact subscapularis tendon. The biceps tendon dislocates anteriorly

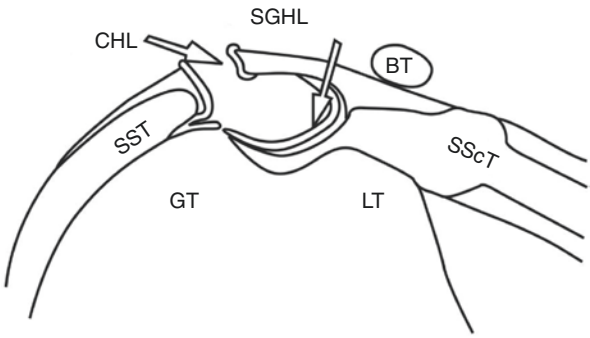

Fig. 3.5 Line diagram of Type IV lesion

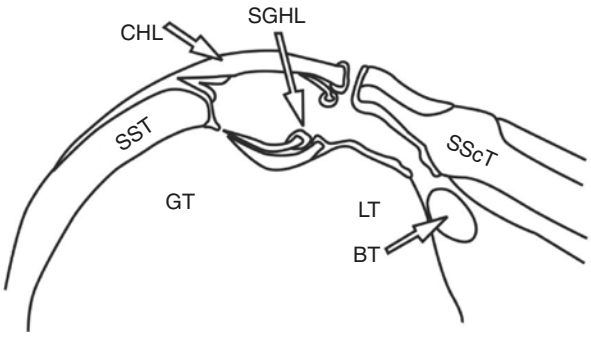

Fig. 3.6 Line diagram of Type V lesion

becoming located anterior to the intact subscapularis tendon. This pattern has a high association with partial- or full-thickness tears of the supraspinatus tendon. Although a full-thickness tear of the subscapularis is not required for this pattern, partial articular-sided, bursal-sided, and interstitial tears may be seen (Fig. 3.5).

- Type V: Intra-articular tendon dislocation—tears of the medial and lateral limbs of the coracohumeral and superior glenohumeral ligaments with a full-thickness tear of the subscapularis allow medial dislocation of the LHBT into the joint. Most commonly the superior subscapularis tendon demonstrates a full-thickness tear, while the lower portion of the tendon remains attached along the inferior portion of the lesser tuberosity and surgical neck. The course of the LHBT moves from a dislocated intra-articular position superiorly to an extra-articular location anterior to the intact subscapularis fibers inferiorly (Fig. 3.6).
- Type VI: Intra-articular tendon dislocation—tear of the medial limbs of the ligaments and detachment of the subscapularis from the lesser tuberosity. Bridging fibers from the subscapularis to the greater tuberosity remain intact. Medial dislocation of the biceps tendon remains deep to the subscapularis and enters the glenohumeral joint (Fig. 3.7).

Type VI: SLAP Tears

Up to ten types of SLAP lesions have been described. The biceps anchor is involved to a SLAP tear.

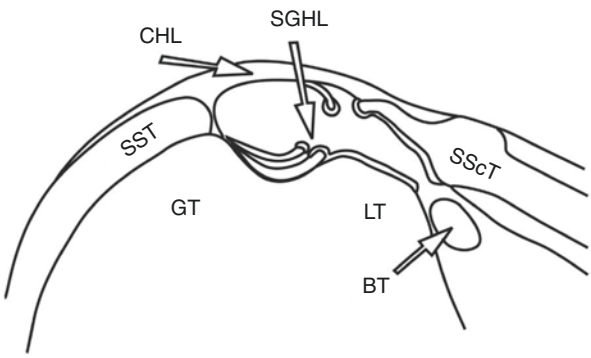

Fig. 3.7 Line diagram of Type VI lesion

- Snyder classification [6]: Original classification which includes Types I–IV.
- Mechanisms: Repetitive overhead activities (often seen in throwing athletes), fall on the outstretched arm with tensed biceps, and traction on the arm.
- Symptoms: Deep shoulder pain, mechanical symptoms of popping and clicking, weakness, easy fatigue, decreased athletic performance, instability, and biceps tendon tenderness.
- Imaging: Imaging should start with plain shoulder X-rays mainly to observe correlated glenohumeral and/or acromioclavicular bony pathology (arthrosis) that could cause symptoms (impingement, calcific tendonitis).
- Ultrasound: It is inexpensive. It also gives a good detection of IHB tendinopathy. It is very good for the detection of ruptures, subluxation, and dislocation. It has a low sensitivity for the detection of partial tears and is highly an operator-dependent technique with a long learning curve.
- Biceps tendinopathy: It shows an accumulation of fluid within the LHBT sheath.
- Biceps dislocation: The biceps groove is empty with the tendon visible medial to it under the SSc fibers.
- Biceps rupture: It shows an empty bicipital groove.

MRI and MRA

MRI allows very good visualization of the labrum, biceps tendon, bicipital groove, and bony osteophytes. It can also detect partial and complete tears of the LHBT, SLAP lesions, subluxation, and dislocation, associated.

RC pathology: MRA has high accuracy on partial-thickness tears and tendinopathy, biceps pulley lesions, and SLAP lesions. Both techniques are expensive. MRA needs the presence of a contrast substance but can raise sensitivity up to 90%.

Treatment Strategy

The methods of treatment include conservative (nonsurgical) and surgical management.

It usually helps if the management is not only targeted to symptoms but also age, activity, and cosmetic deformity.

Ages <40 and/or >40 with high activity demands or cosmetic deformity end up in surgical treatment. If the age is >40, low-demand patients could be managed with nonsurgical treatment, but we must keep in mind to repair any associated RC or labral lesions.

(a) Nonsurgical management:
   1. Activity modification.
   2. NSAID medication.
   3. Physical therapy.
   4. Steroid + local anesthetic injection to the bicipital groove (but not into the tendon) or subacromial space may relieve tendinopathy or impingement symptoms.

(b) Poor results of the treatment involve RC-associated pathology.
   1. Muscle spasm
   2. "Popeye" deformity (particularly in thin patients)
   3. 8–29% decrease in elbow flexion strength [7, 8]
   4. 21–23% decrease in forearm supination

(c) LHB rupture may not be completely benign with conservative treatment, resulting in possible painful symptoms and dysfunction, especially in younger patients.

(d) Surgical management:
   - In either patients <40, or failure of conservative treatment (repair of associated shoulder lesions). Then open surgery or arthroscopic surgery such as tenotomy or tenodesis. It is indicated when there exist partial (>25% of the tendon caliber) longitudinal tear with poor tendon gliding into the bicipital groove, SSc tear and LHBT subluxation/dislocation, complete LHBT rupture, SLAP tear, and biceps pulley/rotator interval lesion with LHBT instability.

Tenotomy Versus Tenodesis: Advantages and Disadvantages (Table 3.3)

Table 3.3 Advantages and disadvantages of tenodesis

Tenotomy	Tenodesis
Advantages	Advantages Length—tension relation maintenance
Simple procedure	Normal elbow flexion
Well tolerated	Normal supination power
Less rehabilitation protocol	Minimized cosmetic deformity
Faster return to activity	Minimized cosmetic deformity
Disadvantages	Disadvantages
Cosmetic deformity (Popeye sign)	Longer rehabilitation
Cramping	More demanding procedure
Fatigue pain	Low rates of failure fixation, humeral
Loss of supination strength	Shaft fractures, CRPS, infection

Tenotomy vs. Tenodesis

- Tenodesis: 40–100% of patients with good/excellent results, failure rate of 5–48%
- Tenotomy: 65–100% of patients with good/excellent results, failure rate of 13–35%
- Tenodesis post-op: bicipital pain in 24% of patients [9]
- Tenotomy post-op: bicipital pain in 17% of patients

Conclusions

1. Patients with LHBT lesions may present associated shoulder pathology.
2. A clinical evaluation must be followed by imaging to detect damaged structures.
3. If the first attempt with conservative treatment fails, then surgery is performed.
4. Tenotomy and tenodesis give good results.
5. The treatment and technique depend on the individual needs of the patient.

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Clinical Examination of the Elbow (Basic and Surface Anatomy) with Special Tests

4

K. Mohan Iyer

Clinical Examination of the Elbow (Basic and Surface Anatomy) with Special Tests

Is a synovial hinge joint formed by the articulation among the humerus, radius, and ulna? The approach to the clinical examination differs considerably in trauma cases and cold cases. It also differs from acute injury examination from old neglected cases.

It is very commonly seen in childhood injuries. It is prone to stiffness, in often neglected and inappropriately treated cases.

For effective use, our hands require stable and painless elbow joints.

Structures: (1) bones and joints, (2) ligaments and tendons, (3) muscles, (4) nerves, and (5) blood vessels.

1. Bones and joints: The elbow proper consists of three articulations which share one synovial cavity: (a) humeroulnar articulation, (b) humeroradial articulation, and (c) proximal radioulnar articulation.
2. Ligaments and tendons: The fibrous capsule is supported by three ligaments, namely, (a) annular ligament, (b) radial collateral ligament, and (c) ulnar collateral ligament.
3. Muscles (Table 4.1):
4. Bursae: (a) Subcutaneous olecranon bursa—It lies deep in the skin, but is superficial to the triceps brachii tendon and the olecranon, and lies in the subcutaneous olecranon bursa. (b) Subtendinous olecranon bursa—It reduces the friction between the olecranon and the triceps brachii muscle.

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Table 4.1 Muscles of the elbow joint

Anterior compartment arm: (a) Brachialis (b) Biceps brachialis	Flexion – Brachialis – Biceps brachii – Brachioradialis – Pronator teres (when flexion is resisted)
Posterior compartment arm: Triceps brachii	Extension – Triceps brachii – Anconeus (a minor contributor)
Anterior compartment forearm: (a) Pronator teres (b) Pronator quadratus	Pronation – Pronator teres – Pronator quadratus
Posterior compartment forearm: (a) Brachioradialis (b) Supinator (c) Anconeus	Supination – Biceps brachii – Supinator

Surface Landmarks of the Elbow (Figs. 4.1 and 4.2)

The elbow joint stability is (1) mainly due to the shape and fit of bones that make up the joint and the surrounding tissue such as capsular and collateral ligaments including the radial collateral ligament and annular ligament of the radius and the ulnar collateral ligament and transverse ligament. (2) Flexor–pronator muscles attached to the medial epicondyle brachialis biceps brachii, brachioradialis, and extensor supinator muscle and the triceps anconeus attached to the lateral epicondyle.

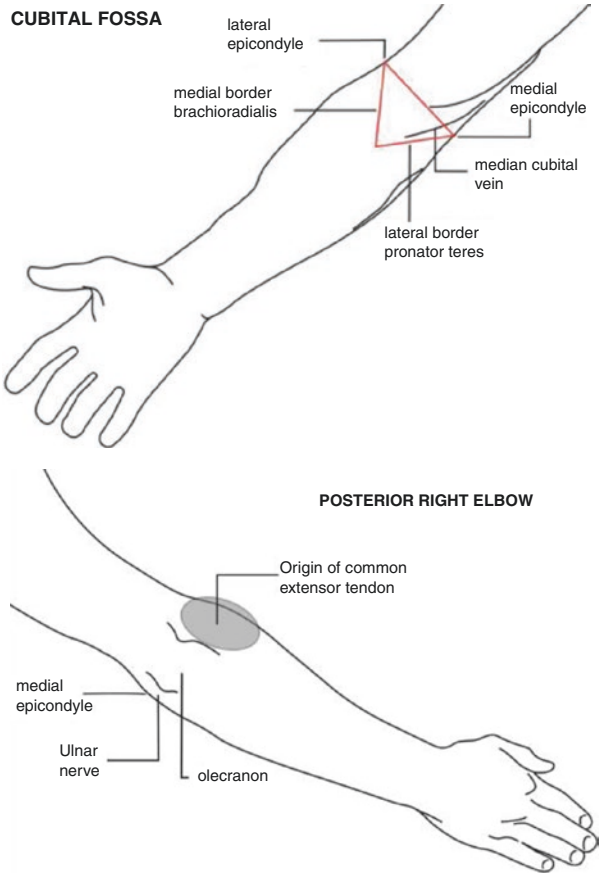
Nerves: (1) median nerve, (2) radial nerve, and (3) ulnar nerve

The common complaints are (1) pain, (2) swelling, (3) stiffness, (4) deformity, (5) instability, and (6) paraesthesias/neurological manifestations.

History of the following observations is made: (1) duration, (2) dominant limb (profession), (3) H/o injury/constitutional symptoms, (4) H/o polyarthralgia/urinary tract infection, (5) treatment received history, (6) H/o massage, (7) limitation of activities of daily living (ADLs), and (8) referred pain from neck/shoulder.

Physical Examination

1. Inspection for swelling, muscle wasting, edema, laceration, deformities, skin change, cubitus valgus, and cubitus varus
2. Palpation: soft tissue, posterior aspect, lateral aspect, and anterior aspect
3. Movements
4. Measurements
5. Distal neurovascular status
6. Regional lymph nodes
7. Thickening of the ulnar nerve
8. Special tests



Figs. 4.1 and 4.2 Surface landmarks of the elbow

Elbow fractures in children: Neuro-motor exam may be limited by the child's ability to cooperate because of age, pain, or fear; thumb extension, EPL (radial—PIN branch); thumb flexion, FPL (median—AIN branch); and cross fingers, interossei (ulnar).

Inspection: (1) Attitude and deformity; (2) carrying angle; (3) swelling such as para olecranon area, anconeus soft spot, radiocapitellar joint, and general diffuse swelling (effusion [semiflexed elbow]); (4) skin such as sinuses, scars, edema, and engorged veins; and (5) muscle wasting

Carrying angle: The physiological valgus ("carrying angle") of the elbow is increased when a load is being carried. Normally, the angle is between 9° and 14° when the elbow is extended and the forearm is supinated.

It is male 7–10° and female 10–15°. It disappears on pronation and flexion of the elbow.

Always make it a point to compare with the opposite side (Fig. 4.3).

Attitude and deformity: Either a Cubitus varus (gunstock deformity) or cubitus valgus

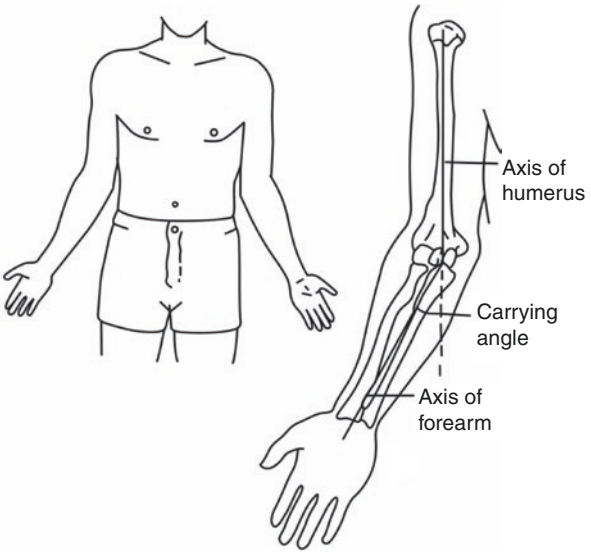


Fig. 4.3 Line diagram of the carrying angle of the elbow

Inspection: Olecranon bursitis or tuberculosis of the elbow (diffuse swelling, flexion deformity, and muscle wasting)

Palpation: (1) Local rise of temperature, (2) tenderness, (3) bony components, (4) soft tissue components, (5) ulnar nerve thickening, and (6) supratrochlear lymph node

1. Local rise of temperature:
   (a) Infective: Pyogenic or tubercular.
   (b) Inflammatory: Polyarthritis or acute myositis.
   (c) Traumatic: Fresh injury may be a hematoma or edema.
2. Tenderness:

   The maximum points of tenderness are (a) lateral epicondyle suggestive of tennis elbow, (b) medial epicondyle suggestive of golfer's elbow, (c) lower end of the humerus suggestive of supracondylar humerus, and (d) radial head suggestive of a fracture of the radial head or upper end of the ulna suggestive of an olecranon fracture.

Palpation

(a) Bony components: Look for irregularity, bowing, thickening, and steps. (1) Medial epicondyle, (2) lateral epicondyle, (3) olecranon, (4) supracondylar ridges, and (5) radial head and capitellum (springing of the forearm).
(b) Soft Tissue: Medial aspect, lateral aspect, posterior aspect, and anterior aspect.

It is important to observe the three bony point relationship which should always be compared with the opposite normal elbow in flexion and extension (medial epicondyle, lateral epicondyle, and olecranon). Palpation starts at the posterior aspect, with the patient standing with his or her shoulder braced backward. The three

palpation landmarks—the two epicondyles and the apex of the olecranon—form an equilateral triangle when the elbow is flexed 90° and a straight line when the elbow is in extension.

Flexing the elbow allows palpation of the olecranon fossa on either side of the triceps tendon.

Anatomical landmarks on the lateral aspect of the elbow: The radial head is palpated with the thumb, while the examiner's other hand is used to pronate and supinate the forearm.

Palpation of the medial aspect of the elbow. Above the medial epicondyle is the ridge on which the intermuscular septum inserts. Two centimeters above the epicondyle is the site used for lymph node palpation.

Palpation of supratrochlear node: Flex the elbow to a right angle to relax the surrounding structures. It is palpated on the anterior surface of the medial intermuscular septum 1 cm above the medial epicondyle. It is not palpable in the normal elbow, but in traumatic causes may be palpable: unilateral or bilateral (systemic).

The ulnar nerve is palpated behind the intermuscular septum. It may sometimes sublux or roll on the epicondyle. Ulnar nerve instability is more readily demonstrated if the elbow is flexed 60° and the upper limb is abducted and externally rotated.

Diagrammatic view of the pattern (Fig. 4.4) of the flexor-pronator group: The thumb represents the pronator teres; the index, flexor carpi radialis; the middle finger, palmaris longus; and the ring finger, flexor carpi ulnaris

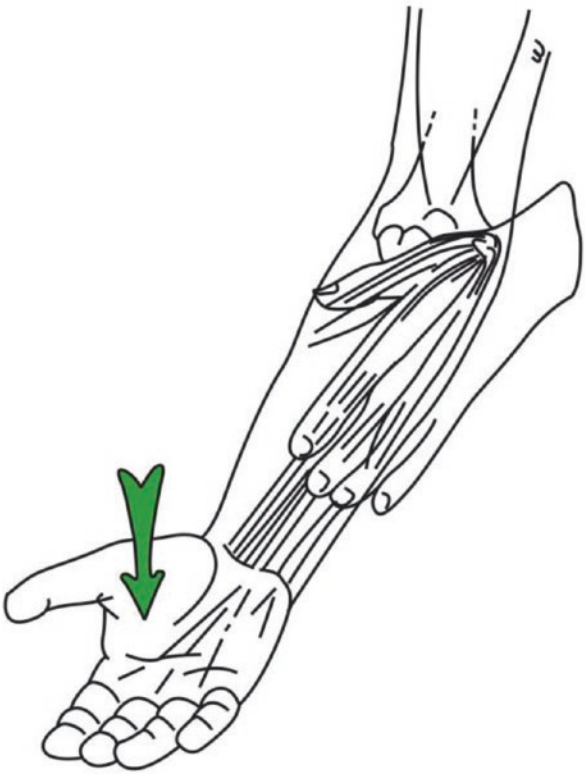


Fig. 4.4 Line diagram showing a diagrammatic view of the pattern of the flexor-pronator group: The thumb represents the pronator teres; the index, flexor carpi radialis; the middle finger, palmaris longus; and the ring finger, flexor carpi ulnaris

Palpation of medial biceps expansion (lacertus fibrosus), which courses over the brachial vessels and the median nerve.

Critical angle of flexion: The arc of flexion is 30–110°. In spite of some degree of morbidity with partial limitation of motion, a person will be able to perform day-to-day activities without much difficulty.

Pulled elbow: It is caused by subluxation of the head of the radius which occurs in infancy and early childhood. The mechanism of injury is a traction force applied to the elbow in pronation leading to subluxation of the head which becomes impacted in the orbicular ligament. This condition responds dramatically to quick movement of the forearm into full supination. Full supination for the management of pulled elbow.

Special Tests

1. Tennis elbow: It occurs mainly in 30–50 years of age. It is usually due to degeneration of the tendon fibers over the lateral epicondyle which are mainly involved in wrist extension. There is a severe burning pain outside the elbow which is worse when gripping or lifting objects and with direct pressure over the lateral epicondyle. This pain may also radiate on the forearm.
   (a) Cozen's test: The patient's elbow is stabilized by the examiner's thumb, which rests on the patient's lateral epicondyle. The patient is then asked to make a fist, pronate the forearm, and radially deviate and extend the wrist while the examiner applies resistance. A positive sign is when a sudden severe pain is experienced in the area.
   (b) Mill's test: While palpating the lateral epicondyle, the examiner passively pronates the forearm, flexes the wrist, and extends the elbow when a sharp pain is experienced over the area which indicates a positive test.
   (c) Tennis elbow test: The examiner resists the extension of the third digit of the hand distal to the proximal interphalangeal joint stressing the interosseous muscle and tendon when a positive test is indicated by pain over the area.
   (d) Chair test: Ask the patient to attempt to lift a chair with the elbow straight and the shoulders flexed to 60°. Difficulty to perform and complaint of pain over the lateral aspect are positive signs.
   (e) Thomson's test: Ask the patient to clench the fist, dorsiflex the wrist, and extend the elbow. A forced palmar flexion against the patient's resistance with pain over the area is a positive sign.
2. Golfer's elbow: It is also known as medial epicondylitis similar to tennis elbow. It is most common in men 20–50 years of age. There is pain over the medial aspect of the elbow radiating down the inner forearm which is worse when making fists or shaking hands.

   Golfer's elbow test: Flex the elbow, supinate the hand, and then extend the hand. Pain over the medial epicondyle is a positive sign.

3. Olecranon bursitis: It is usually caused by inflammation of the bursa. Its main causes are (a) trauma, (b) prolonged pressure, (c) infection and (d) medical conditions such as rheumatoid arthritis and gout.
4. Medial ligament injuries (MCL/UCL/little leaguer's elbow): It is caused by repetitive microtraumas, which may result in many growth disorders of the elbow. It is usually injured due to valgus trauma (acute) or repetitive overhead throwing activities (chronic). This can be evaluated by a valgus stress test by the elbow being flexed to 25°/30°, when an abduction or valgus force is applied to the distal forearm while the ligament is palpated when the examiner feels the ligament tense when stress is applied.
5. Lateral ligament injuries: These are less common than medial ligament injuries. If the LCL is damaged, varus opening is present with stress. This varus laxity increases with annular ligament injury due to the separation of the head of the radius from the ulna. This can be evaluated by varus stress test: the elbow is flexed 25°/30° and stabilized by the examiner's hand. An adduction force is applied by the examiner to the distal forearm and the examiner feels the ligament tense when stress is applied.
6. Lateral pivot-shift test: The patient is in a supine position when the wrist and proximal forearm are grasped securely, and a combination of supination, valgus, and axial compression is applied to the elbow. At 40° of flexion, the patient with posterolateral rotatory instability will note apprehension/pain as the radial head subluxates about the capitellum. With further flexion, a clunk or sense of relief occurs as the joint is reduced. This examination is most easily performed with the patient under sedation.
7. Provocative test for cubital tunnel syndrome (puts tension on the ulnar nerve at the elbow). Four provocative tests were included: Tinel's sign, elbow flexion, pressure provocation, and combined elbow flexion and pressure provocation. The most sensitive provocative test in the diagnosis of cubital tunnel syndrome was elbow flexion when combined with pressure on the ulnar nerve.
8. Special test for posterolateral instability: Posterolateral rotatory instability of the elbow is often caused by a complex injury to the lateral ulnar collateral ligament complex. This often leads to chronic mechanical symptoms of instability and pain. Surgical repair or reconstruction of the ligament is often required to stabilize the radiocapitellar joint.
9. Tests for neurological dysfunction (Tinel's sign): The area in the groove between the olecranon process and the medial epicondyle is tapped. A positive sign is indicated by a tingling sensation in the ulnar nerve distribution distal to the point of compression which indicates the point of regeneration of the sensory fibers. The most distal point at which abnormal sensation is felt represents the limit of nerve regeneration.
10. Wartenberg's sign: Sitting with hands on the table, the examiner passively spreads the fingers apart and asks the patient to bring them together. The inability to bring the little finger close indicates ulnar nerve neuropathy.

The Elbow

5

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Floating Elbow

It is an injury involving a fracture of the humerus and a fracture of the radius and/or the ulna in the same extremity. This may be associated with dislocation of the elbow in high-energy injuries. Here the elbow remains disconnected from the shoulder and wrist, hence the name "floating."

The term floating elbow was first coined by Stanitski and Micheli to describe an injury pattern in children involving simultaneous fractures of the forearm axis and supracondylar humerus in the same extremity [1]. The most likely mechanism of injury is a fall on the outstretched arm with the forehand pronated and wrist hyperextended [2]. Similar injuries may also be caused by direct trauma and other positions of the arm and forearm. These injuries may also be seen in adults with direct high-velocity trauma such as sideswipe injuries, severe crush injuries, or falls from extreme heights [3]. Ipsilateral fractures of the humerus and forearm are extremely rare in adults and their incidence may be anywhere between 2% and 13% with the majority of these injuries being due to high-energy injuries.

There is a lot of damage to the bones, muscles, nerves, and vascular which varies a lot from case to case. Pediatric injuries historically have had better results than those of their adult counterparts, largely because of children's ability to remodel skeletal deformity with time. In Stanitski's series of six patients, all had an excellent outcome with respect to range of motion (ROM) and carrying angle. Yokoyama et al. reviewed a series of floating elbow injuries in adults. They had 67% good or excellent results; nonunions were present in four cases. All of these were fractures treated with unlocked intramedullary fixation. In 2013, Ditsios et al. described a prognostic classification for adults with floating elbow injury. This classification

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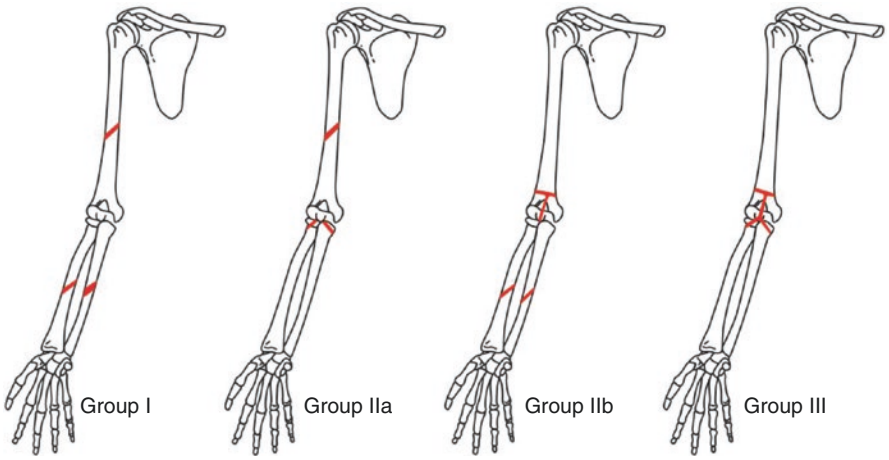

Fig. 5.1 Line diagram showing the Ditsios floating elbow prognostic classification

system focuses on the presence or absence of intra-articular elbow involvement associated with the fracture pattern (Fig. 5.1).

1. Group/type I—Humeral shaft fracture + radius and/or ulna shaft fracture
2. Group/type IIa—Humeral shaft fracture + intra-articular radial head and/or olecranon fracture
3. Group/type IIb—Intra-articular distal humerus fracture + radius and/or ulna shaft fracture
4. Group/type III—Intra-articular distal humerus fracture + intra-articular radial head and/or olecranon fracture

Clinical Features

Patients obviously complain of pain and deformity of the affected limb. The neurovascular deficit ranges from 25% to 40% and other injuries such as compound fractures, head injuries, and trauma to the thorax and abdomen vary depending on the severity of the injury.

This leads to a thorough detailed examination of the patient as a whole such as neurovascular status, fractures, compartment syndrome, etc., with all fractures being reduced and treated as appropriate.

A bare minimum of imaging studies are carried out as early as possible with X-rays in the AP and lateral views. Selective patients with a persistent pulseless extremity despite fracture reduction should undergo arteriography to rule out vascular injury if a delay in operative intervention is anticipated.

Management

Indications for treatment of the isolated humerus and forearm fractures vary greatly with patient age, location of the fracture, and injury to the soft-tissue envelope present at the time of injury. Appropriate soft-tissue management is very important to aid in fracture healing and may dictate the type of fixation used.

The need for fixation of the fractures in adults at the earliest is the rule with very little contraindications to this such as (1) Patients who have severe irreparable vascular compromise may need to undergo emergency amputation to facilitate patient resuscitation. (2) Patients who are very critically ill may require a delay in fixation to allow for recovery from the systemic inflammatory response that accompanies these injuries, but open reduction and internal fixation (ORIF) should still be undertaken when appropriate.

(a) Nonoperative therapy: Immobilization of the fractures and appropriate debridement of open fracture wounds. Administer intravenous (IV) antibiotics to patients with open fractures. The vascular status of the limb must be monitored carefully. If a disruption is present or suspected, surgical consultation and coordination of management needs for the combined injury should take place.
(b) Surgical therapy: The type of fracture management and soft-tissue coverage should be determined on a case-by-case basis. Treatment guidelines for children [4] differ slightly from those for adults. Studies by Moed et al. [5] and Grace et al. [6] have shown that immediate internal fixation of both bone forearm fractures in adults with early ROM provides the patient with a stable construct that allows for accelerated rehabilitation and return to function.

In children, supracondylar humerus fractures should be treated with closed (or open, if necessary) reduction and percutaneous pinning. In children, acceptable results have been achieved with closed reduction of the forearm fracture, with or without percutaneous pinning.

Complications

The incidence of some type of associated neurovascular injury in children and adults is 25–45%. A loss of range of movements in the elbow and forearm axis is not uncommon, even with anatomic restoration of all fractures.

Infection is an unavoidable complication in open fractures and hence a delay in definitive fixation until the soft tissues are in a condition is the best and ideal option.

Malunion and nonunion can result from a number of factors, including persistent infection, inadequate fixation, poor soft-tissue envelope, and poor technique.

In conclusion although the nature of floating elbow injuries is complex, the presence of nerve injury and intra-articular involvement predispose to worse clinical outcomes [7].

Nursemaid Elbow (in Children)

It is a common injury among preschool-aged children, in whom radial head subluxation when the normal anatomical alignment of two of the three bones that form the elbow joint is disrupted. It is commonly seen in girls and the left arm is more often injured than the right which is thought to be secondary to the likelihood of the parent being right-handed (and thus most frequently pulling their child's left hand). The injury can occur innocently from swinging a young child by the arms or pulling a child's arm while in a hurry. It takes relatively little force to pull the bones of a young child's elbow out of place. It is usually a temporary condition without permanent consequences.

The elbow joint involves the two bones of the forearm (radius and ulna) and the bone of the upper arm (humerus, hence the name "funny bone"). The ulna is the bone that is on the side of the forearm that has the baby finger. The radius runs the length of the forearm on the thumb side. The radius forms an individual connection with the capitellum, the far end of the humerus. The relationship between the ulna and the humerus is sturdy. However, the head of the radius requires a tight ligament (the annular ligament) to anchor the radial head into the proper region of the capitellum. If the radial head receives a sudden pull or is subjected to chronic traction, this ligament may partially tear and slip out of position, allowing the radius to lose its proper fitting in the "socket" at the end of the humerus.

Typically, this type of injury occurs in children 1–4 years of age but has occurred in infants 6–12 months of age as well. The peak incidence is 27 months of age. As children grow, their bones become larger and more defined. In addition, ligaments become stronger and thus provide a better support system. Nursemaid elbow is rarely seen in children older than 6 years of age unless the child is involved in prolonged hanging by the hands or prolonged lifting of heavy objects. (The appellation "nursemaid elbow" comes from a time when children of upper social class families commonly were cared for by a nursemaid or nanny.)

Nursemaid elbow usually results from a sudden pulling force applied to the extended arm of a child. Due to the relative strength of the adult in comparison to the weakness of the child's supportive annular ligament, the applied force may not seem strong to the parents, and they may not realize an injury has occurred.

The upper end of the radius that connects to the elbow joint is known as the radial head. The radial head has a shallow concave shape allowing it to fit over a complementary convex prominence at the end of the humerus (the capitellum). As the child grows, the radial head broadens and ultimately becomes wider than the portion of the radius (called the radial neck) adjacent to the head. In the young child, the radial head does not yet have a well-defined lip at its end. As such, in the toddler, the radial neck and radial head are similar in size.

The annular ligament holds the radius alongside the ulna, which is the other bone in the forearm. Besides stabilizing the radial head-humerus joint, the annular ligament permits the radius to twist when the hand changes position from palm down (prone) to palm up (supine). In young childhood, the annular ligament is still relatively loosely attached to the bone and can experience a small tear in some of its fibers.

This combination of these two things (shallow concave radial head and loose-fitting annular ligament which may easily partially tear) allows the loose portions of the ligament to slide over the radial head when a pulling force is applied to the elbow while the forearm is slightly rotated palm down (pronation). When this happens, this annular ligament tissue can become trapped between the radial head and the capitellum, resulting in the subluxation of the radial head or nursemaid elbow.

Symptoms

Typically after the injury, the child generally cries in pain and will refuse to use the involved arm. The child usually supports the painful arm with their other hand. When the forearm is turned with the thumb away from the body to show the palm upward (in supination), the child will resist and cry in pain.

Nursemaid elbow means the elbow (radial head) has slipped out of its normal place at the joint. It is sometimes referred to as "pulled elbow" because it occurs when a child's elbow is pulled and partially dislocated resulting in a "radial head subluxation" [8]. Because a young child's bones and muscles are still developing and pliable, it typically takes very little force to pull the bones of the elbow partially out of place, making this injury very common. It results from a sudden pulling force applied to the extended arm of a child, because of the relative strength of the adult in comparison to the weakness of the child's supportive annular ligament. The elbow joint has numerous ligaments which grow stronger and tighter as a child grows older. In little kids and babies, the ligaments are still loose which makes it easy for the radial head to slip out of place. It is also called a pulled elbow or radial head subluxation. As the child matures, the radial head broadens and ultimately becomes wider than the portion of the radius (called the radial neck) adjacent to the head. As such, in an infant, the radial neck and radial head are similar in size. The annular ligament holds the radius alongside the ulna, thus providing a stabilizing factor to the radial head-humerus joint. The combination of these two things, namely, a shallow concave radial head and loose-fitting annular ligament, which may easily partially tear thus allows the loose portions of the ligament to slide over the radial head when a pulling force is applied to the elbow while the forearm is slightly rotated palm down (pronation). When this occurs, this annular ligament tissue becomes trapped between the radial head and the capitellum, resulting in the subluxation of the radial head or nursemaid elbow. A potentially useful sonographic finding of a pulled elbow ("hook sign") using point-of-care ultrasound in the emergency department [9].

Various causes of nursemaid elbow may be as follows:

1. A tug or a jerk on a child's arm to make him or her walk faster.
2. Swinging a child by the arms or hands.
3. Lifting a child up by the hands or wrists.
4. Catching a child by the hands to stop a fall.
5. An infant rolls over onto the arm.

Symptoms of Nursemaid Elbow

The main symptom of a pulled elbow is pain when the child attempts to move the arm with its refusal to use this arm, which is usually held for support by the other hand. There may be no swelling, bruising, or other signs of a serious injury. The elbow may be slightly bent and the palm may be turned toward the body. Any movement to straighten the arm or move the elbow back into place will be met with resistance and may result in more serious damage.

Treatment of Nursemaid Elbow

An X-ray may be necessary to rule out any fracture. Any analgesic may be given for pain relief. Usually a "reduction maneuver" is performed to put the elbow (radial head) back into the correct position, when a click may be heard in some cases.

Elbow Arthritis

Elbow psoriatic arthritis is an inflammatory type of arthritis, seen in people who are having psoriasis, presented with patchy and shiny, scaly skin lesions.

1. Persons with a history of psoriasis and those with a family history with close family members or identical twins are at an increased risk of elbow psoriatic arthritis.
2. It is more common in people in their middle ages, ranging from 30 to 50 years, but can also occur at any age.
3. Men and women are equally affected, but involvement of the spine may be seen more in men.
4. People having autoimmune disorders or abnormalities of the immune system can be at increased risk of developing elbow psoriatic arthritis.
5. Some people with an injured joint or psoriasis developed after trauma to the skin can develop arthritis later on.
6. Some studies have suggested a link between certain infections like streptococcal infections or HIV and the possibility of development of elbow psoriatic arthritis; however, this may need more research.

7. Certain factors like increased stress and other triggers that can make symptoms of psoriasis worse can influence the development of worsening of elbow psoriatic arthritis.

- Joint complaints—Elbow joint pain, swelling, tenderness, warmth, and stiffness. Pain and stiffness can make the joint painful to move resulting in restricted movements and a decreased ROM. Pain and tenderness may also be present in the ligaments and tendons attached to the elbow joints. Morning stiffness of the affected joints is often present, which may last longer than 30 min.
- Along with joint inflammation and swelling, there may be inflammation of tendons attached to the bones and resulting swelling. Tendons around the elbows, heels, and pelvic bones are commonly involved. Elbow psoriatic arthritis can present with any of these joint symptoms depending on the pattern of the disease in that individual. Additionally, other joints, if involved, may also show similar signs and symptoms.
- Psoriasis—The skin on the elbow may show psoriatic lesions, which may also be present in other parts of the body. These are dry, silver-colored or gray-appearing scales in spots present over red patches on the skin area. They may have raised margins and can cause itching. These are usually present over the joints, ankles, heels, knees, and spine along with the elbows and even on the scalp.
- Inflammation of tissues—If finger- or toenails are involved, there can be swelling of joints in fingers that appear like sausages, medically termed as dactylitis, and they can even get detached. Pain and swelling in the heels due to inflammation of the tendon attached to the bone which is called Achilles tendon may be noted. Occurrence of pain and inflammation where the tendon attaches to the bone in other areas like the knees, chest, and hips is called enthesitis.
- Other symptoms of elbow psoriatic arthritis—Other parts of the body or joints may also be affected by joint inflammation, pain, stiffness, and skin lesions. Eye inflammation may be present, with redness and pain in the eyes. Nail abnormalities like crumbling, pitting, discoloration, and disfigurement may be seen. Sometimes swelling in fingers and toes may develop that involves not only the joints but also other tissues. Fatigue is also a commonly associated complaint, which may leave the person feeling weak with lack of energy.

Blood Investigations

Laboratory studies include blood tests that help to detect the presence of inflammation, infection, and related parameters. Some tests include complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Tests that aid in the detection of the presence of autoimmune disorders include ANA profile that includes various parameters, based on which the physician can get an idea about the immune system disorders. Rheumatoid factor (RF) is performed to rule out the possibility of rheumatoid arthritis (RA).

Joint Aspiration

Excess synovial fluid from the elbow joint may be aspirated for fluid analysis to rule out other forms of arthritis like gout, septic arthritis, and others.

Imaging Studies

Imaging studies include X-rays, ultrasound, MRI, and computed tomography (CT) scans that can help to detect changes in the bones and elbow joints which can help to distinguish elbow psoriatic arthritis from other forms of arthritis.

Treatment for elbow psoriatic arthritis aims to relieve the symptoms and control disease progression, thus minimizing joint damage as much as possible. Various treatment modalities suit different people and the most effective combination needs to be worked out for each individual case.

Medical Management of Elbow Psoriatic Arthritis

1. Analgesics or pain killers for elbow psoriatic arthritis.
2. Nonsteroidal anti-inflammatory drugs (NSAIDs) for elbow psoriatic arthritis. NSAIDs are also available in gel or cream form that can be applied locally to the affected joint to avoid stomach irritation.
3. Disease-modifying antirheumatic drugs (DMARDs) help to control disease progression and thus also limit deformities of joints. DMARDs help in slowing down an overactive immune system and delays damage to the joints of the body.
4. Immunosuppressant medications.
5. TNF-alpha inhibitors.
6. Steroid injections for elbow psoriatic arthritis—Steroid injections may be given into the elbow joint to reduce inflammation and pain and control the overactivity of the immune system for elbow psoriatic arthritis.
7. Skin creams—Skin creams, ointments, or light treatment may be considered to control skin symptoms in the case of psoriasis in elbow psoriatic arthritis. Skin creams containing vitamin D, retinol, or others may be prescribed depending on the individual case.

Surgical Treatment for Elbow Psoriatic Arthritis

Surgical treatment is considered only in cases where medical management and other conservative measures for treating elbow psoriatic arthritis fail to produce an effect. Surgery helps in relieving elbow joint pain, increasing ROM, and improving the physical look of the affected region.

(a) Synovectomy: It is performed on the elbow joint for restoring the joint function, or any diseased or damaged portion in the joint can be removed. Synovectomy is often performed as an arthroscopic procedure.
(b) Arthroscopy: This procedure helps to detect the abnormalities within the joint and helps to remove loose fragments in the joint, bone spurs, damaged lining, or any cysts present in the joint area.
(c) Osteotomy: It is performed for realigning the long bones of the arm in order to release the pressure off the elbow joint.
(d) Joint fusion: This procedure may not allow free movement at the joint.
(e) Joint replacement.

Liebenberg Syndrome (Brachydactyly with Joint Dysplasia with Dysplastic Elbow Joints)

It was first described by Dr. F. Liebenberg in 1973 [10]; it involves abnormal development of the arms, resulting in characteristic arm malformations that can vary in severity, and has three main symptoms, namely, (1) dysplasia (improper formation) of the bony components of the elbow, (2) abnormal shape of carpal bones, and (3) brachydactyly, a symptom where the fingers and toes are shorter than normal. It has an autosomal dominant mode of inheritance. Liebenberg (1973) described four males and six females in five generations of a white South African family with upper limb deformities affecting the fingers, wrists, and elbows.

Here the heterozygotes with this mutation express the disease phenotype. It is mainly the result of one of two different genetic mutations; the first one is a deletion upstream of the PITX1 gene on chromosome 5, which includes the H2AFY gene, which is responsible for suppressing an upstream enhancer element known as hs1473. When H2AFY is removed, the enhancer is brought closer to PITX1 and inappropriately enhances it in the forelimbs, causing them to adopt hindlimb morphology [11]. The second mutation that can cause the phenotype for Liebenberg syndrome is a translocation of chromosome 18 and chromosome 5. Translocation mutations are ones that switch parts of nonhomologous chromosomes with each other. This move introduces two enhancers from chromosome 18 to move to a position directly upstream of PITX1 on chromosome 5. The enhancers increase the transcription of the PITX1 gene and cause patients to develop the same phenotype described above.

Affected individuals also have joint deformities (contractures) that limit movement of the elbows, wrists, and hands. Development of the lower limbs is normal in people with this condition. They also have no other health problems related to this condition, and life expectancy is normal. Radiographs show a highly abnormal elbow joint. The olecranon is very small or missing, and the radius and ulna have a flattened joint surface.

In all, less than ten affected families have been described in the medical literature.

The inheritance pattern is characteristic of the condition as an autosomal dominant pattern, which means having a genetic change that affects the PITX1 gene on one copy of the chromosome in each cell is sufficient to cause the disorder. In most cases, an affected person has one parent with the condition.

This condition also has various other names as follows: (1) brachydactyly-elbow wrist dysplasia syndrome, (2) brachydactyly with joint dysplasia, and (3) carpal synostosis with dysplastic elbow joints and brachydactyly.

The patients have normal overall body structure, and the elbows are enlarged with abnormally large olecranons and radial heads. Patients have relatively fixed elbow positions and are therefore unable to pronate or supinate their forearms. The wrists are limited in their active movements and have very prominent ulnar sides of the joints. The fingers are short and abnormally bent and brachydactyly and camptodactyly are quite common and toe-like. Hence these fingers are small club-shaped appendages with a reduced range of movements in the hand. Treatment is mainly surgery to correct some of the morphological changes made by Liebenberg syndrome; carpectomy is used to correct the radial deviations and flexion deformities in the wrist.

Panner's Disease (Osteochondrosis of the Capitellum of the Elbow)

In 1927, a Danish orthopedic surgeon, Dr. Dane Panner, first described radiographic changes of the capitellum in young adults, subsequently known as Panner's disease [12]. The capitellum has a rich vascular supply prior to the age of 5 years. Afterward, the nucleus of the capitellum is mainly supplied by posterior vessels functioning as end arteries [13]. If those vessels are disrupted by repetitive stress (i.e., throwing), ischemia can develop. This may result in the disordered endochondral ossification called Panner's disease. Several experts believe that "abnormal valgus stress" after the age of 5 years is the most important factor in the development of Panner's disease. It is believed that Panner's disease mostly occurs in boys (90%) possibly because of the delayed appearance and maturation of the secondary growth centers [14]. The higher risk for traumatic injuries in boys could be another explanation for the increased prevalence of Panner's disease in boys who reported a precedent trauma. Valgus stress in throwing athletes and increased axial load to the radiocapitellar joint in gymnasts may typically result in lateral compression injuries of the elbow which lead to several lesions, including Panner's disease and OCD of the humeral capitellum. The complaints of several weeks of pain and stiffness in the elbow often include a history of valgus stress. These are increased by activity and relieved by rest in most patients. There may be a small effusion and swelling. Range of movements is usually reduced in the terminal loss of 20° of extension. Fragmentation of the capitellum, radiotranslucent areas, and sclerosis were also often documented in literature.

Synovial Chondromatosis of the Elbow

Primary synovial chondromatosis (PSC) is a rare, benign, proliferative disease of hyaline cartilaginous bodies within the synovium of joints. Synovial osteochondromatosis is a benign proliferative disorder with metaplasia of the synovial membrane that affects the fibroblasts of the synovial joints, tendons, and bursae. Very few cases have been reported in literature till today. PSC is generally associated with monoarticular involvement, while it is more frequently seen in large joints. It is extremely important to remember that synovial conditions of the elbow may involve the ulnar nerve even when a patient is asymptomatic. This condition is more commonly reported in the knee joint. This can be treated by either an open or arthroscopic synovectomy and the diagnosis is made evident by undergoing magnetic resonance imaging (MRI) preoperatively. It has also been reported in painful PSC diffusely affecting the ulnohumeral joint with extrusion into the cubital tunnel resulting in compressing the ulnar nerve but without any preoperative signs or symptoms of ulnar nerve compression [15]. The maximum number of cases reported on PSC is that in the elbows of 12 patients [16] which have been treated by removal of loose bodies and partial synovectomy in all patients. They had observed that the degree of osteoarthritis deteriorated after surgery in five of these cases and functional deficiencies were usually moderate, but most importantly it was noted that there was no recurrence of PSC. There was also a unique case of elbow synovial chondromatosis with sudden onset of severe loss of elbow extension and flexion ROM caused by mechanical block from the deposition of chondral fragments in the olecranon and coronoid fossae, respectively [17]. This was treated by successful arthroscopic surgical treatment of the synovial chondromatosis of the elbow which included arthroscopic removal of loose bodies and partial synovectomy. A rare case report has been described in literature giving rise to a mechanical snapping elbow [18]. Arthroscopic treatment of synovial osteochondromatosis of the elbow is an effective and safe therapeutic management with low morbidity and early return to activities [19]. Peripheral nerve palsy due to synovial osteochondromatosis of the elbow joint is extremely rare [20]. Three-dimensional reconstruction based on MRI shows the specific shape and location of the tumor and its relation to the posterior interosseous nerve.

Olecranon Impingement Syndrome

Elbow impingement is a condition characterized by compression and damage to soft tissue (such as cartilage) situated at the back of or within the elbow joint. When the elbow is straightened fully, the soft tissues are compressed at the back of the joint resulting in impingement. If these forces are excessive and repetitive, damage and inflammation of the cartilage and/or soft tissue at the back of the elbow joint may occur. At times bony spurs may develop within the elbow joint to add to the existing problem. This condition typically develops in activities associated with hyperextension or in combination with a valgus (sideways) force. It may occur as a

specific incident involving a hyperextension force or to repetitive strain due to overuse. This is commonly seen in a throwing athlete, in the "winding-up" phase of the throwing motion (just prior to throwing) which may hyperextend the elbow resulting in significant strain on the elbow joint giving rise to impingement which is frequently seen in baseball pitchers, cricketers, and javelin throwers. This may also be seen in martial arts due to repetitive punching, thus forcing the elbow into hyperextension. At times, it may also be seen in contact sports due to a forced hyperextension force at the back of the elbow.

Clinical Features

There is sudden excruciating pain at the back of the elbow which increases with forced hyperextension or it may develop gradually and progressively over a period of time. In minor cases such as due to overuse, the patients may continue with the activity along with swelling and stiffness at the elbow with rest. Progress of the condition results in a stage of discontinuing throwing activities along with some pain and stiffness and ultimately with inability to straighten the elbow fully.

Diagnosis

A complete history with clinical features typical of the condition assists in diagnosing this condition, though certain investigations like X-rays, ultrasound, CT, and MRI help to rule out other conditions.

Treatment

Majority of the patients respond to physiotherapy along with rest from any activity that increases their pain, like an orthosis or brace.

Any activities that place a tremendous amount of stress on the elbow should be decreased or avoided which ensures the body can begin the healing process in the absence of further tissue damage. Subsequently these activities may be resumed slowly within limits of pain.

The specific physiotherapy for elbow impingement may include the following:

1. Soft-tissue massage
2. Stretches
3. Joint mobilization
4. Electrotherapy (e.g., ultrasound)
5. Anti-inflammatory tablets
6. Taping, sports tape (for protective taping), or (a) slings or (b) elbow braces
7. Bracing
8. Ice or heat treatment

9. Exercises to improve flexibility and strength
10. Patient education
11. Activity modification advice
12. Sporting technique correction
13. Graduated return to the activity program

Cubital Tunnel Syndrome

Cubital tunnel syndrome is the second most common peripheral nerve entrapment syndrome in the human body. It is the cause of considerable pain and disability for patients. When appropriately diagnosed, this condition may be treated by both conservative and operative means.

Cubital tunnel syndrome is ulnar nerve entrapment neuropathy in the cubital tunnel of the elbow. This is the tunnel where the ulnar nerve is compressed. It can cause numbness, tingling, weakness, and/or pain in the arm and in the fourth and fifth fingers.

Relevant Applied Anatomy: Cubital tunnel is formed medially by the medial epicondyle of the humerus and laterally by the olecranon process of the ulna and the tendinous arch joining the humeral and ulnar heads of the flexor carpi ulnaris. The roof of the cubital tunnel is the cubital tunnel retinaculum which is also known as the epicondylo-olecranon ligament or Osborne band. The floor of the tunnel is formed by the capsule and the posterior band of the medial collateral ligament of the elbow joint. Along with other structures, the cubital tunnel contains the ulnar nerve. Compression of the ulnar nerve in this tunnel causes cubital tunnel syndrome.

The ulnar nerve is the terminal branch of the medial cord of the brachial plexus and contains fibers from the C8 and T1 spinal nerve roots. It descends the arm just anterior to the medial intermuscular septum and later pierces this septum in the final third of its length. Progressing underneath the septum and adjacent to the triceps muscle, it traverses the cubital tunnel to enter the forearm where it passes between the two heads of the flexor carpi ulnaris muscle. This anatomical arrangement has two implications for the nerve. As it lies some distance from the axis of rotation of the elbow joint, movement of the elbow, therefore, requires the nerve to both stretch and slide through the cubital tunnel. Sliding has the greatest role in this process, although the nerve itself can stretch by up to 5 mm. There is an increase in intraneural pressure with elbow flexion. In addition, the shape of the elbow becomes oval to ellipse, narrowing the canal by 55%. Wrist extension and shoulder abduction are further known to increase intraneural pressure.

Risk Factors for Cubital Tunnel Syndrome

1. Diabetes mellitus.
2. Works involving long periods of elbow flexion.
3. Post-trauma.

4. Marked varus or valgus deformity at the elbow.
5. Obesity.
6. Workers operating vibrating tools.
7. Bone spurs/arthritis of the elbow.
8. Swelling of the elbow joint.
9. Cysts near the elbow joint.
10. Fracture or dislocations of the elbow.
11. Sex: women are more likely to develop cubital tunnel syndrome than men.

Pathophysiology of Cubital Tunnel Syndrome

Compression, traction, and friction have been implicated in cubital tunnel syndrome.

Compression is the principal mechanism. It could be mechanical compression to the nerve substance or compression of the intrinsic blood supply leading to ischemia.

With previous injuries to the nerve, a tight tunnel may predispose the nerve to friction and compression. Diabetes may make a nerve more vulnerable to compression by causing local ischemia or by interfering with nerve metabolism.

There are many areas in the cubital tunnel in which the ulnar nerve can be compressed (Fig. 5.2).

The ulnar nerve controls most of the intrinsic muscles in the hand that help with fine movements. It also gives innervations to some of the extrinsic muscles in the

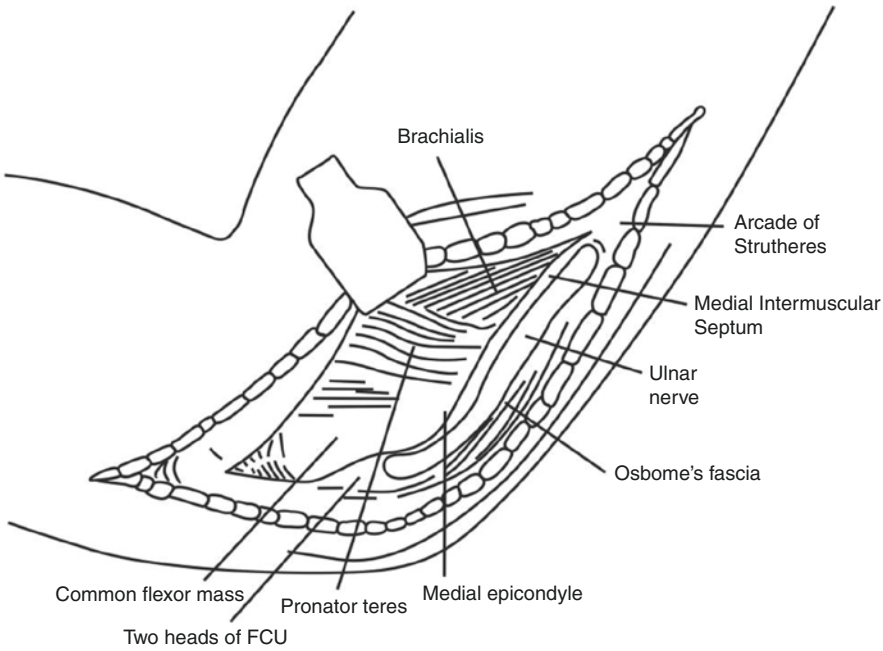

Fig. 5.2 Line diagram of the cubital tunnel

forearm that help you make a strong grip. At the elbow the ulnar nerve travels through a tunnel of tissues called the cubital tunnel that runs under the medial epicondyle of the elbow.

Causes of Cubital Tunnel Syndrome

1. Repetitive use of the arm or elbow (including leaning on the elbow).
2. Leaning on your elbow for long periods of time can put pressure on the nerve.
3. A direct blow to the inside of the elbow.

Clinical Features

(a) Altered sensation in the little and ring fingers.
(b) Sensory loss in the distribution.
(c) Clumsiness in the hand with the progression of the severity.
(d) Wasting of hypothenar muscles, small muscles of the hand, and muscles on the ulnar side of the forearm.
(e) Tenderness in the elbow joint at the medial epicondyle.
(f) Numbness, tingling, or decreased sensation in the palm or last two fingers. This may be worse at night while sleeping.
(g) Weakening of grip and difficulty with finger coordination.
(h) If the nerve is very compressed or has been compressed for a long time, muscle wasting in the hand can occur.
(i) Pain in the elbow, palm, and/or last two fingers. Activities that use the arm may increase the pain.
(j) Sensitivity to cold.

Diagnosis

1. The diagnosis is established by history and physical examination, along with the findings of nerve conduction tests and imaging.
2. Physical examination includes observation and inspection of the elbow and forearm.
3. Tapping the nerve at the elbow (Tinel's sign test).
4. A sensory examination that includes both light touch and a test of the ability to distinguish between sharp and dull stimuli and temperature.
5. Checking the strength of specific muscles of your hand.
6. Checking your pinching and gripping ability.
7. Nerve conduction test.
8. Imaging—X-rays of the elbow are taken to see if bone spurs or arthritis can cause compression.

9. There are *special tests* that can be done to confirm that someone has cubital tunnel syndrome:
   (a) Tinel's sign at the elbow: Tinel's sign should be positive over the cubital tunnel.
   (b) Elbow flexion test.
   (c) Froment's sign (Fig. 5.3).

PS: It must be noted that for the compression in Guyon's canal, which also causes ulnar nerve symptoms, the sensation is preserved over the dorsum of the hand because of the dorsal cutaneous branch of the ulnar nerve that comes off proximal to Guyon's canal.

McGowan Score: It grades the ulnar nerve neuropathy

1. Mild occasional paresthesia, positive Tinel's sign, subjective weakness
2. Moderate paresthesia, objective weakness, positive Tinel's sign
3. Severe constant paresthesia, weakness, overt muscle wasting

The elbow flexion test is a useful accurate provocative test for cubital tunnel syndrome.

Differential Diagnosis

1. Cervical radiculopathy C8-T1—Motor and sensory deficits in a dermatomal pattern including the fourth to fifth digits, associated weakness of intrinsic muscles of the hand, and associated painful and often limited cervical ROM
2. Thoracic outlet syndrome—Compression of the structures of the brachial plexus potentially leading to pain, paresthesias, and weakness in the arm, shoulder, and neck
3. Ulnar neuropathy at other places
4. Brachial plexus injury

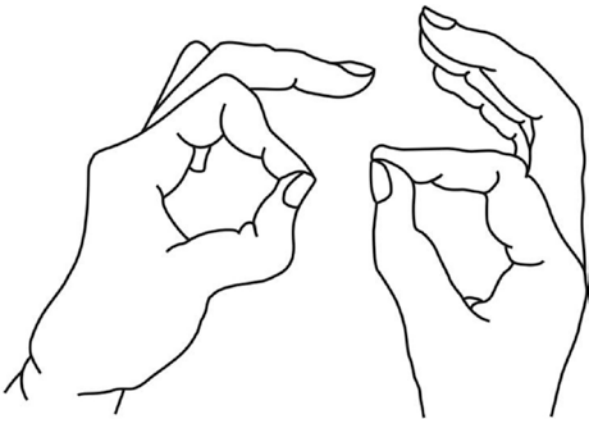

Fig. 5.3 Line diagram of Froment's sign

Investigations

The diagnosis of cubital tunnel syndrome is mainly clinical. Nerve conduction studies can be done to confirm the diagnosis.

Mild cases may have normal nerve conduction studies.

X-rays of the elbow could be done to rule out bony changes. X-rays may reveal osteoarthritis, cubitus valgus, or calcification in the medial collateral ligament.

Treatment

(a) Nonsurgical means include:
   1. NSAIDs—If the symptoms have just started, anti-inflammatory medicine such as ibuprofen is recommended which can be used to reduce any swelling around the nerve.
   2. Ice—This can help reduce the swelling.
   3. Brace or splinting—These can be worn in the night to help keep the elbow straight.
   4. Nerve gliding exercises—Ulnar nerve glides can help the nerve slide through the cubital tunnel at the elbow in which there can be an improvement of symptoms.

(b) Surgical treatment may be recommended if:
   1. Nonsurgical means have not improved the condition.
   2. The ulnar nerve is very compressed.
   3. The nerve compression has caused muscle weakness and damage.

Surgical means include:

1. Cubital tunnel release—In this operation, the ligament "roof" of the cubital tunnel is cut and divided and left free without any sutures (deroofing).
2. Ulnar nerve anterior transposition—The ulnar nerve is moved from its place behind the medial epicondyle to a new place in front of it.
3. Medial epicondylectomy—Another option to release the nerve is to remove part of the medial epicondyle.
4. Like ulnar nerve transposition, this technique also prevents the nerve from getting caught on the bony ridge and stretching when the elbow is bent.

Complications of Ulnar Nerve Release

1. Persistent dysesthesia
2. Reflex sympathetic dystrophy
3. Infection
4. Neuroma formation
5. Persistent sensory deficit and weakness

Summary

Cubital tunnel syndrome is when the ulnar nerve enters the cubital tunnel and is compressed in that area. There are five areas in the tunnel where the ulnar nerve can be compressed. Activities such as prolonged bending of the elbow and a direct blow to the elbow can be the causes of cubital tunnel syndrome. Special tests such as Tinel's sign at the elbow, elbow flexion test, and Froment's sign are used to diagnose the condition. Physical therapy management is geared toward reducing the tingling sensation, pain, and/or numbness, improving strength, and reducing swelling.

Little League Elbow (LLE) Syndrome

LLE syndrome is a valgus overload or overstress injury to the medial elbow that occurs as a result of repetitive throwing motions.

During the throwing motion, valgus stress is placed on the elbow. This valgus stress results in tension on the medial structures (i.e., medial epicondyle, medial epicondylar apophysis, medial collateral ligament complex) and compression of the lateral structures (i.e., radial head, capitellum). Repeated stress results in overuse injury when tissue breakdown exceeds tissue repair. A common elbow problem in children is medial apophysitis, commonly referred to by doctors as "little leaguer's elbow." Recurrent microtrauma of the elbow joint can lead to LLE, a syndrome which includes the following: (1) delayed or accelerated growth of the medial epicondyle (medial epicondylar apophysitis), (2) traction apophysitis (medial epicondylar fragmentation), and (3) medial epicondylitis.

The elbow consists of numerous ossification centers and cartilaginous physes. Becoming familiar with the chronologic order of the appearance and ossification of these growth centers is important. Consider the mnemonic CRITOE (i.e., capitellum, radius, internal epicondyle, trochlea, olecranon, external epicondyle).

Each of the ossification centers appears at a relatively predictable time starting around age 1–2 years, with 2-year intervals between the centers' appearances. Closure of each of the apophyses occurs from age 14 to 16 years, with the medial epicondyle specifically closing at approximately age 15 years. The elbow likely reaches full skeletal maturity by the late teen years, at which time injuries to the ulnar collateral ligament (UCL) are far more common. Until then, the young thrower is at risk for LLE syndrome.

Static stabilizers around the elbow include the bony articulations, the joint capsule, and the various ligament bundles.

The dynamic stabilizers primarily include the muscles that cross the elbow joint, such as the triceps, biceps, and brachioradialis.

(1) The flexor-pronator group stabilizes against valgus stress, while (2) the extensor-supinator group stabilizes against varus stress.

Elbow biomechanics include flexion/extension ROM and pronation/supination. Slight hyperextension 5–15° through flexion of approximately 150° is within normal limits.

Baseball pitchers with years of throwing experience often have relative 5–10° flexion contractures on their dominant side; however, in a young thrower, a flexion contracture can be a sign of injury. Pronation of 75° and supination of 85° are normal. Varus-valgus laxity of 3–4° is normal.

Sport-Specific Biomechanics

One should be familiar with the stages of throwing to understand the complexities of the biomechanical forces that contribute to a young thrower's risk of injury, such as in LLE syndrome. The pitching or throwing motion can be divided into six stages as given below. Medial elbow injuries are the most common type seen in throwers and occur most commonly in the cocking and acceleration phases of throwing, owing to the presence of maximum valgus extension or distraction forces.

Windup begins with the pitcher balancing his weight over his rear leg, with the elbow flexed and the forward leg flexed at least 90°.

Stride starts with the lead leg beginning to descend toward the plate and the two arms separate. The elbow moves from extension into flexion of 80–100°.

Cocking occurs when the humerus is in extreme abduction and external rotation and the elbow is flexed. The lead foot contacts the ground, the pelvis and trunk rotate, and elbow torque transfers valgus force across the elbow joint. During this phase, medial tension and lateral compression forces are applied to the elbow.

Acceleration is the shortest pitching phase, lasting from maximal external shoulder rotation to ball release. In this phase, the trunk rotates as the elbow extends. Maximum elbow angular velocity is comparable during fastballs, sliders, and curveballs, but is less during the change-up pitch. Velocity comes from rotation of the trunk, shoulder, and hips. Varus torque forces during this phase act to resist the valgus extension "overload" phenomenon and can contribute to posterior elbow (olecranon) impingement.

Deceleration is initiated at ball release and ends when the shoulder has reached full internal rotation. The body must decelerate the arm and dissipate forces in the elbow and shoulder.

Follow-through is the final phase of the baseball pitch and ends with the pitcher reaching a balanced fielding position with full-trunk rotation and the bodyweight fully transferred from the rear leg to the forward leg. During follow-through, the elbow flexes into a relaxed position and crosses the body.

LLE symptoms may include:

1. Pain in any part of the elbow (Fig. 5.4)
2. Swelling
3. Difficulty straightening the arm all the way
4. Sometimes, a bump appears on the inside of the elbow
5. A locked or stiff elbow

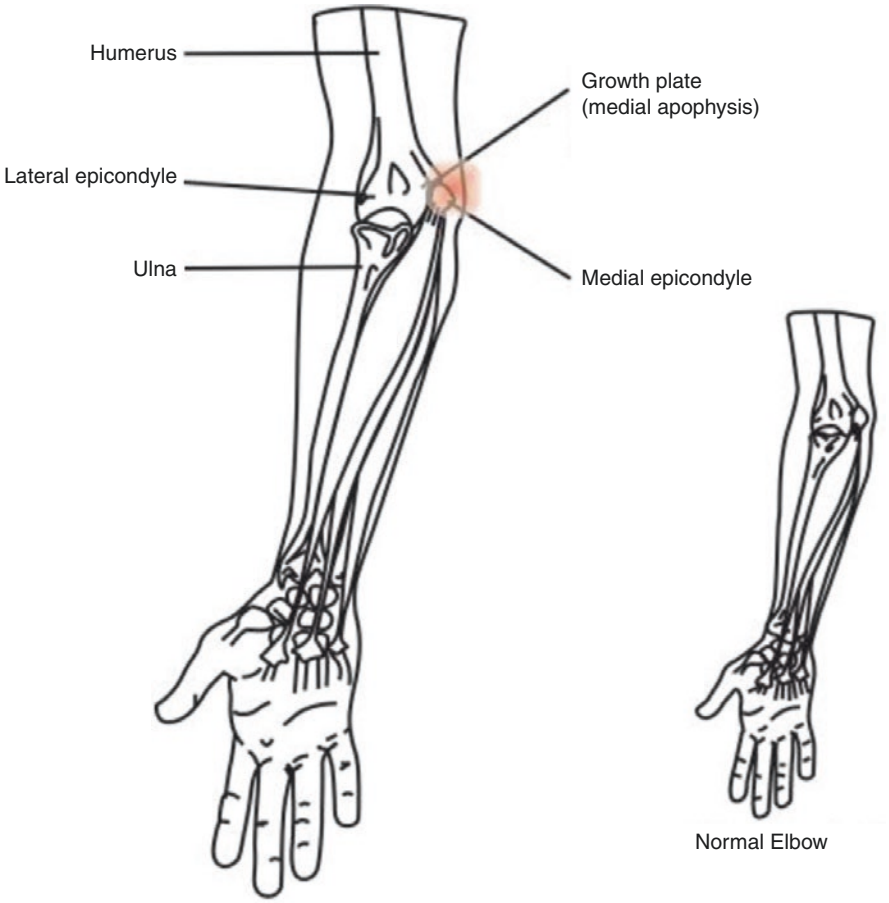

Fig. 5.4 Line diagram showing the sites of pain in LLE syndrome

Prevention of Medial Epicondylitis

Lots of pitching put repetitive stress on the medial epicondyle growth plate, which can weaken it and make it more prone to overuse injury.

1. The best way to prevent medial epicondyle apophysitis is to follow the attached guidelines for appropriate pitch count limits and proper rest between pitching appearances.
2. Do not throw through the pain. Pain is a sign of injury, stress, or overuse. Pushing through pain will only worsen the injury. Rest is required to allow time for the injured area to heal.
3. Do not throw breaking pitches (curveball, slider) before 14 years of age.

Clinical Examination

1. Inspection is important to note the carrying angle and any flexion contractures that may be present relative to the opposite side. During the initial examination, evaluate for muscle atrophy or hypertrophy, bony deformities, or the presence of swelling and ecchymosis.
2. Palpation of bony structures should include both epicondyles, the olecranon process, the capitellum, and the radial head. Soft-tissue palpation should include the UCL (felt best with the patient's elbow in 50–70° of flexion), the biceps tendon, the triceps tendon, and the flexor-pronator and extensor-supinator muscle complexes.
3. Strength testing of various muscles should be performed.
4. Neurologic testing should include evaluation of the ulnar nerve. Palpation for tenderness, stability testing, and Tinel's test via percussion over the ulnar groove for paresthesias consistent with ulnar neuritis constitute a thorough examination.
5. Special tests include valgus stress testing to evaluate injury to the UCL. The patient may be prone, supine, or upright. The stress test should be performed with the elbow in 20–30° of flexion with a valgus force exerted on the elbow. Opening up on the injured side, compared with the opposite uninjured side, is most reflective of an injury to the UCL. Pain without instability during valgus stress testing is more commonly seen with LLE syndrome.
6. Two special tests to note are the milking maneuver, which is performed with the patient seated, and the valgus extension overload test.
   (a) For the milking maneuver, the examiner grasps the thrower's thumb with the arm in the cocked position of 90° of shoulder abduction and 90° of elbow flexion. Then the examiner applies valgus stress by pulling down on the thumb.
   (b) For the valgus extension overload test, the examiner stabilizes the humerus from the outside and then pronates the forearm during extension while applying valgus stress. Pain is more likely associated with posterior impingement if this test result is positive.

Causes

1. Training errors, such as abrupt changes in intensity, duration, or frequency of throwing activity, are frequently associated with sports injuries. Poor coaching and lack of preseason conditioning can also contribute to an increased risk of injury.
2. Strength and flexibility imbalances can indirectly cause elbow injuries, as can injuries to other areas of the body (current and/or previous injuries).
3. Anatomic malalignment of the lower extremities can cause LLE syndrome.
4. Improper footwear or playing surface can result in an insecure platform for stability in throwing activities.
5. Associated disease states or preexistent injury can be causative.

6. Growth patterns are sometimes implicated as the cause of LLE syndrome. Physeal or growth cartilage at the epiphysis or apophysis is less resistant to repetitive trauma than fused adult bone at ligamentous and tendinous insertions; thus, skeletally immature athletes more commonly develop growth plate (apophyseal) injuries. In addition, rapid growth (growth spurts) causes increased muscle and tendon tightness around a joint, resulting in loss of flexibility, biomechanical imbalance, and an increased risk of injury.

Differential Diagnoses: Elbow Dislocation, Elbow and Forearm Overuse Injuries, Humeral Capitellum Osteochondritis Dissecans, Lateral Epicondylitis, Medial Epicondylitis, and UCL Injury.

Investigations

1. Laboratory studies are rarely needed in the evaluation of elbow pain in athletes. If ordered, an elevated ESR or CRP level may indicate an acute inflammatory condition such as septic bursitis, which is more commonly observed in patients with olecranon bursitis. This condition manifests as posterior elbow pain, swelling, and decreased ROM. Patients with olecranon bursitis should be referred to a hand specialist for incision and drainage, possible surgical excision, and antibiotic treatment.
2. Plain radiographs: They are useful for detecting fractures, calcified loose bodies, heterotopic ossification, growth plate irregularities, developmental stages of ossification centers (CRITOE), arthritis, tumors, and infectious conditions (e.g., osteomyelitis).
   (a) Plain radiographs are indicated for most cases of athletic elbow pain, particularly if symptoms have been present for more than 3 weeks, if an acute inciting injury is reported, or if significant bony tenderness with or without a joint effusion is present [1, 2].
   (b) Note that with LLE syndrome, the diagnosis is often a clinical one, and routine radiographs show no bony irregularities. Also important are comparison views of the unaffected elbow in young preadolescent and adolescent athletes in order to properly assess the developmental stages of the ossification centers. Some possible radiographic abnormalities, noting the normal ossification center, age of appearance, and age of closure, that affect one's initial management strategies are as follows:
      - Capitellum—Age of appearance, 1 year; age of closure, 14 years
      - Radius—Age of appearance, 3 years; age of closure, 16 years
      - Internal epicondyle—Age of appearance, 5 years; age of closure, 15 years
      - Trochlea—Age of appearance, 7 years; age of closure, 14 years
      - Olecranon—Age of appearance, 9 years; age of closure, 14 years
      - External epicondyle—Age of appearance, 11 years; age of closure, 16 years

3. Widening or distal displacement of the medial epicondyle is a worrisome radiographic finding seen in the setting of a medial epicondyle avulsion fracture and warrants a referral to a sports orthopedic surgeon for surgical consultation.
4. Valgus stress radiographs may be useful in a skeletally mature athlete. The findings are often subtle, but 2 mm of joint widening or more may indicate a UCL injury. Proximal UCL ossicles are sometimes seen as a result of repetitive microtrauma.
5. Osteochondritis dissecans manifests as a bony, craterlike defect in the capitellum and may possibly be associated with compression changes in the radial head. Osteochondritis dissecans lesions, when detected, should be referred to a sports-medicine specialist, and additional imaging with MRI is usually indicated. Outcomes vary, depending on the size of the lesion, the degree of displacement, the presence of any associated loose bodies, and the patient's skeletal maturity.
6. Osteochondrosis of the capitellum (Panner's disease) shows fragmentation of the capitellar ossification center and a smaller and irregular epiphysis. Severe cases may show advanced avascular necrosis of the capitellum. Interestingly, this disease is often self-limited in the 8- to 11-year-old athletes; these patients often do well with time and conservative management.
7. Osteophytes are sometimes seen in the olecranon on the lateral elbow radiograph and are often correlated with cases of posterior elbow impingement.
8. MRI provides great detail of the structural integrity of the articular cartilage surface, bone marrow, subchondral bone, muscles, tendons, ligaments, and nerves.
9. CT scanning has dramatically advanced with the advent of helical scanners. CT scanning is most useful for characterizing bony tumors, myositis ossificans, and fracture morphology. Contrast tomography can be used, but it is no longer favored except in certain individualized cases.
10. Ultrasonography can be useful for imaging soft tissues around the elbow. Instability with dynamic ultrasonography during valgus stress and ulnar nerve instability with dynamic motion have been studied, but these techniques are not routinely used in the United States.
11. Radionuclide bone scanning is a sensitive but nonspecific imaging modality to identify the presence of a bony injury. Bone scanning is rarely used for elbow injuries, because alternative imaging techniques are more likely to aid in diagnosis.
12. Arthroscopy of the elbow can be used as both a diagnostic and treatment procedure. Arthroscopy can be used to determine the size and location of the bony lesions intra-articularly. Arthroscopy can also help determine whether loose fragments are present in the joint. Sometimes, arthroscopy can be used for surgical excision or fixation of bony fragments. Most patients have some form of imaging studies performed before an arthroscopic evaluation; therefore, arthroscopy is primarily used as a form of treatment.

Treatment

If caught early enough and treated properly by a pediatric orthopedic physician, LLE will heal completely and not cause any permanent elbow damage. To ensure a proper diagnosis, the orthopedic physician will review the patient's symptoms, clinical examination results, and X-rays.

LLE treatment options are dependent on the extent of the growth plate injury. Left untreated, throwing injuries in the elbow can be very complex. However, younger children tend to respond better to nonsurgical treatments, such as:

1. Rest the affected area
2. Apply ice packs to bring down any swelling
3. Utilize NSAIDs.

If pain persists after a few days of complete rest of the affected arm or if pain recurs when throwing is resumed, it is recommended that the child stop the activity until cleared by a pediatric orthopedic physician. Based on the severity of the injury, a 6-week period of rest may be recommended. Upon approval from the physician, a slow progressive throwing program may be instituted over the next 6–8 weeks. While rare, surgery or casting is occasionally necessary to relieve pain symptoms.

Other Treatments (Injection, Manipulation, etc.)

Joint injections and manipulations are not appropriate forms of treatment in patients with LLE syndrome.

Surgical Intervention

Medial epicondylar fractures may require either closed reduction and casting or surgical reattachment with fixation if displacement, elbow instability, or failure of conservative treatment occurs.

Type II osteochondrotic lesions are treated surgically if the loose body interferes with motion or causes mechanical symptomatology (e.g., locking, buckling). Techniques of surgical treatment include removing loose bodies, drilling to stimulate active repair, bone grafting when architectural support is needed, or reattachment with absorbable or nonabsorbable Kirschner wires (K-wires). Type III lesions are usually treated with loose body removal, with or without drilling, curettage, or reattachment with K-wires.

When loose bodies or osteophytes are present in patients with olecranon injuries, surgical removal may be indicated in those who are symptomatic. Bone grafting may also be used in cases of olecranon nonunion when rest and immobilization have failed.

It is important to note that all throwers who have had surgical treatment for elbow pain require some form of progressive rehabilitation following the principles outlined above, including a thorough biomechanical pitching analysis. Pitchers should be counseled that many do not return to the previous level of throwing following surgical treatment of elbow injuries; however, outcomes vary based on individual circumstances.

Return to Play

Return to throwing activities in individuals with LLE syndrome should be carefully monitored by the patient and his or her family in conjunction with an educated trainer, the coach, and a pediatric sports medicine or orthopedic specialist.

Olecranon Bursitis

Olecranon Bursitis is an inflammation of the bursa overlying the olecranon process between the bone and the skin.

Other names for olecranon bursitis are student's elbow, miner's elbow, and draftsman's elbow.

Etiology

The common causes of olecranon bursitis are as follows:

1. Post-traumatic: such as acute hemorrhagic bursitis due to acute trauma
2. Chronic: such as repetitive rubbing on hard surfaces
3. Inflammatory: such as gout, pseudogout, uremia, RA, pigmented villonodular synovitis
4. Infection: such as pyogenic or tuberculosis

It is seen commonly in adults and less commonly in children. Normally the bursa prevents the soft tissue by providing a mechanism for the skin to glide freely over the olecranon process. Direct trauma in sports or from injury leads to an acute swelling because the bursa gets filled with blood and effusion. There may also be associated fractures of the olecranon spurs, hence its importance to rule out underlying fractures and ligamentous injuries in differential diagnosis. A chronic aseptic bursal swelling is the most common form of olecranon bursitis. It mainly occurs due to repetitive rubbing of the olecranon on hard surfaces and is usually asymptomatic. The inflamed or infected olecranon bursitis is the most difficult to treat as it is often difficult to differentiate septic from aseptic inflammatory arthritis since it requires microbiological confirmation.

Examination

Acute post-traumatic bursitis may present as a tender, fluctuant, fluid-filled swelling when aspiration yields a hemorrhagic fluid. Patients with chronic aseptic bursitis present with a painless swelling over the olecranon process and frequently give a history of repetitive rubbing of the elbows on hard surfaces.

Patients with systemic conditions like RA, gout, and uremia and medical conditions may present with painful swelling olecranon bursae. Septic olecranon bursitis may be primary (hematogenous spread) or secondary to local cortisone injections. Patients may have local signs of inflammation, with or without a discharging sinus, with some patients presenting with systemic signs of infection.

Differential Diagnosis

The differential diagnosis of acute bursitis must include (1) underlying fractures, (2) ligamentous injuries, and (3) rarely a synovial cyst that may mimic bursitis of the elbow.

Imaging

Radiographs are mandatory for post-traumatic arthritic onset bursitis to rule out fracture. Ultrasound studies may reveal an underlying abscess or fluid collection. MRI may reveal an underlying abscess or osteomyelitis which is not very evident on plain radiographs.

Treatment

Acute post-traumatic bursitis may be treated conservatively initially with rest, ice, a compression dressing, elevation, and NSAIDs to decrease pain and swelling. Excessive swelling may require to be aspirated. But patients who are not responsive to conservative treatment for a long time may require a surgical bursectomy.

Septic bursitis should be treated with aspiration, IV or oral antibiotics, rest, ice, compression, elevation, and occasional splinting. An incision and drainage may be required if aspiration alone fails to control the symptoms.

Patients with chronic, aseptic bursitis of idiopathic etiology or from repeated rubbing of the elbow can be given protective pads to the elbows, ice, instructions

regarding avoiding repetitive trauma, NSAIDs, and reassurance. Aspiration and corticosteroid injection have been shown to result in more rapid recovery at 6 months than only NSAID treatment.

RA of the Elbow Joint

RA is a chronic inflammatory disease. It causes inflammation, swelling, and pain to many joints of the body. When RA attacks the elbow, the joint may swell and become inflamed and there may even be visible bulging. The swelling in the joint can compress nerves which causes tingling and numbness. Over time, the actual structure of the joint is damaged and altered. There may even be an erosion of cartridge and bone. In fact, in the most severe cases, there can be dislocation of bones and even joint deformities. It is noticed to be frequently symmetrical and bilateral in nature.

Symptoms of RA in the Elbow

The common symptoms of RA in the elbow include: (1) pain, particularly on the outer side of the joint—the pain may worsen as you rotate the forearm, (2) swelling, (3) unstable joint, (4) locking or lack of full movement, (5) stiffness, and most importantly (6) pain in both elbows (symmetry).

Certain inflammatory and noninflammatory arthritis (Table 5.1) must be kept in mind as follows:

Table 5.1 Inflammatory and noninflammatory arthritis

Features	Inflammatory	Noninflammatory
History	Morning stiffness >1 h, low grade temperature, fatigue, and rash	Morning stiffness <1 h
Presentation	Erythema (rubor), warmth (color), swelling (tumor), tenderness (dolor), and loss of function	Bony proliferation in osteoarthritis
Laboratory	+ESR with a decreased CRP, anemia of chronic disease, +RF, and anti-CCP antibodies	Within normal limits
Radiographs	Erosions, periostitis, joint space narrowing	Joint space narrowing with osteophytes and subchondral sclerosis
Synovial fluid	Leucocyte count >2000/μL, predominantly neutrophils, leucocyte count >50,00/μL	Leucocyte count <2000/μL, <50% neutrophils

Autoimmune (Table 5.2)

Infectious (Table 5.3)

Post-infectious (Table 5.4)

Crystalline Arthropathies (Table 5.5)

Table 5.2 Types of autoimmune arthritis

	Symmetric	Asymmetric	Spine	Monoarticular	Oligoarthritis	Polyarthritis	Migratory
RA	++++	–	Cervical	–	+	++++	
AS			++++			++++	
Psoriatic arthritis	++	+++	+	++	+++	++	
Associated inflammatory bowel disease	++	+++	+	++	+++	++	

Table 5.3 Features of infectious arthritis

	Symmetric	Asymmetric	Spine	Monoarticular	Oligoarthritis	Polyarthritis	Migratory
Nongonococcal	–	++++	++	++++	+++	+	–
Disseminated gonococcal		++++			++++		++++
Date Lyme disease		++++			++++		++++

Table 5.4 Features of post-infectious arthritis

	Symmetric	Asymmetric	Spine	Monoarticular	Oligoarthritis	Polyarthritis	Migratory
Reactive arthritis		++++	++		++++	++	
Acute rheumatic fever		++++			++	+++	++++

Table 5.5 Features of crystalline arthropathies

	Symmetric	Asymmetric	Spine	Monoarticular	Oligoarthritis	Polyarthritis	Migratory
Gout	+	++++	+	+++	+++	+	+
Pseudogout	++	+++	–	+++	++	++	

Erythrocyte Sedimentation Rate (ESR)

It is usually seen in conditions that increase immunoglobulins. It is age/2 in normal males and age + 10/2 in normal females.

It is increased in (1) myeloma, Waldenstrom's, 1% amyloid; (2) SLE, Sjogren's; and (3) chronic liver disease.

"CRP"

Cytokine Response Protein (CRP)

When ESR and CRP are both elevated, it is suggestive of (1) infection, (2) inflammatory states, (3) trauma, and (4) cancer.

Serology

(a) RF: It is the titer of IgM against IgG. It is present in 80% of RA. Its sensitivity is 68% and its specificity is 85%.
(b) Anti-citrulline-containing peptide antibody (anti-CCP Ab): It is associated with the development of erosive disease. Its sensitivity is 67% and specificity is 95%.

Synovial Fluid

Categories of synovial fluid (Table 5.6) based on clinical and laboratory findings

Table 5.6 Types of synovial fluid

Measure	Normal	Noninflammatory	Inflammatory	Septic	Hemorrhagic
Volume, mL (knee)	<3.5	Often >3.5	Often >3.5	Often >3.5	Usually >3.5
Clarity	Transparent	Transparent	Translucent-opaque	Opaque	Bloody
Color	Clear	Yellow	Yellow to opalescent	Yellow to green	Red
Viscosity	High	High	Low	Variable	Variable
WBC per mm^3	<200	200–2000	2000–100,000	15,000–>100,000	200–2000
PMNs, %	<25	<25	>50	>75	50–75
Culture	Negative	Negative	Negative	Often positive	Negative
Total protein, g/dL	1–2	1–3	3–5	3–5	4–6
Glucose, mg/dL	Nearly equal to blood	Nearly equal to blood	>25, lower than blood	<25, much lower than blood	Nearly equal to blood

There is an increase in the clinical sensitivity and specificity of rheumatological disease.

1. In RA, there are synovial thickening of the metacarpophalangeal (MCP) joints, moderate MCP flexion, and swan neck deformities.
2. In ankylosing spondylitis (AS), the genetic predisposition for its development (Fig. 5.5) is as follows:

- The features are early sacroiliitis (Table 5.7) along with fusion of the sacroiliac joints in AS.

3. Psoriatic arthritis: It shows the following features: (a) asymmetric oligoarthritis (43%), (b) symmetrical polyarthritis (33%), (c) sacroiliitis (14%), (d) predominant spondylitis (4%), and (e) synovitis-acne-pustulosis-hyperostosis-osteomyelitis (SAPHO) syndrome (2%).

- Arthritis mutilans may also be seen in psoriatic arthritis with marked deformity and destruction of the digits (Fig. 5.6). Pencil-in-cup deformity with involvement of the DIP joints in psoriatic arthritis.

4. Infectious arthritis: Here septic arthritis is the presumed diagnosis in any patient with acute monoarthritis with fever. Here synovial fluid analysis is the diagnostic

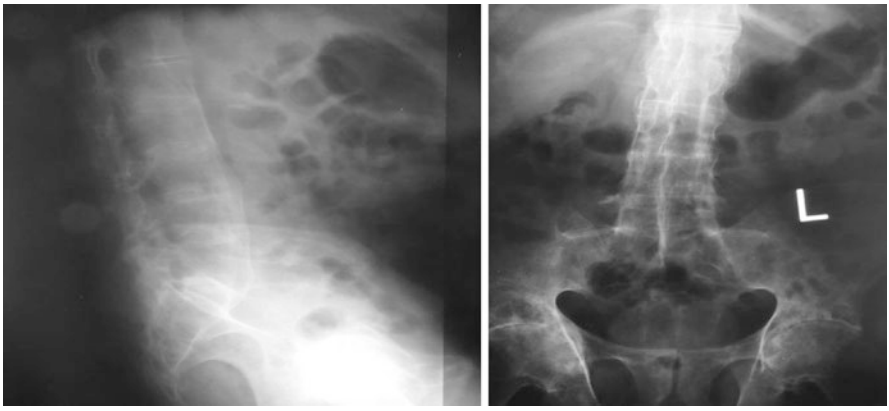

Fig. 5.5 AS: bamboo spine. (Courtesy: Figure reused with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

Table 5.7 Ethnic differences of sacroiliitis

	HLA-B27 positive	AS and HLA-B27 positive
West European Whites	8%	90%
African Americans	2–4%	48%

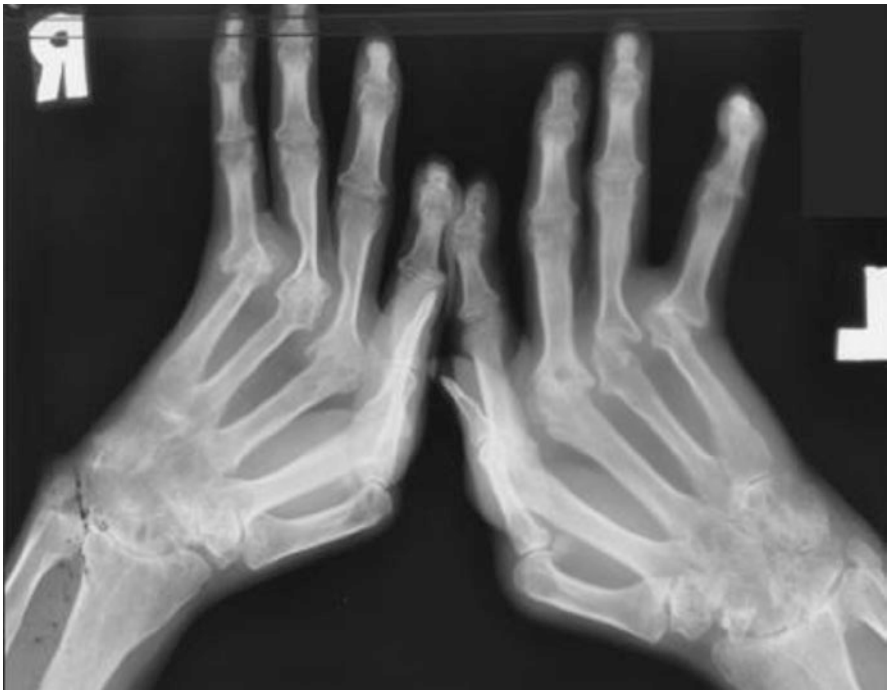

Fig. 5.6 Arthritis mutilans (RA). (Courtesy: Figure reused with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

Table 5.8 Clinical features of disseminated gonococcal arthritis

Symptoms/signs	Percentage of patients
Migratory polyarthralgia	70
Tenosynovitis	67
Dermatitis	67
Fever	63
Purulent arthritis: 1. One joint 2. More than one joint	42 32 10
Genitourinary symptoms	25

test (white cell count with a differential, with gram stain being sensitive in 29–50%. A crystal search is also done using polarized light microscopy and the culture is positive in majority of the patients with nongonococcal bacterial arthritis).

5. Disseminated gonococcal arthritis: Major symptoms and signs of disseminated gonococcal arthritis (Table 5.8).
6. Lyme arthritis: This is usually characterized by intermittent or persistent arthritis. Usually there is a monoarthritis of the knee joint which is very common.

There may be an abrupt presentation which may last several weeks to months. There is an erythema migrans along with large joint effusions of the knee joint which are usually warm and swollen.

7. Reactive arthritis (Table 5.9): It usually occurs preceding enteric infection, i.e., diarrhea (like salmonella of various serovars, shigella especially *Shigella flexneri* and also *Shigella dysenteriae* and *sonnei*, yersinia including *Yersinia enterocolitica* 0.3 and 0.9 and *Yersinia pseudotuberculosis*, campylobacter especially *Campylobacter jejuni*, and *Clostridium difficile*). There is preceding urethritis. Reactive arthritis commonly causes asymmetrical arthritis of the knees and a sausage-shaped toe in reactive arthritis.
8. Gout: It may present as a tophus of the knee or as an inflamed tophaceous gout of the PIP joints of the hand which ruptures spontaneously discharging a pasty material of urate crystals and inflammatory cells with no organisms.
9. Pseudogout: The definite diagnosis is a demonstration of CPPD crystals in tissue or synovial fluid by definitive means such as X-ray diffraction, etc. (Fig. 5.7).

Table 5.9 Clinical features of reactive arthritis

	Sensitivity (%)	Specificity (%)
Episode of arthritis for more than a month with urethritis and cervicitis	84.3	98.2
Episode of arthritis for more than a month with either urethritis or cervicitis with bilateral conjunctivitis	85.5	96.4
Episode of arthritis, conjunctivitis, and urethritis	50.6	98.8
Episode of arthritis for more than a month, conjunctivitis, and urethritis	48.2	98.8

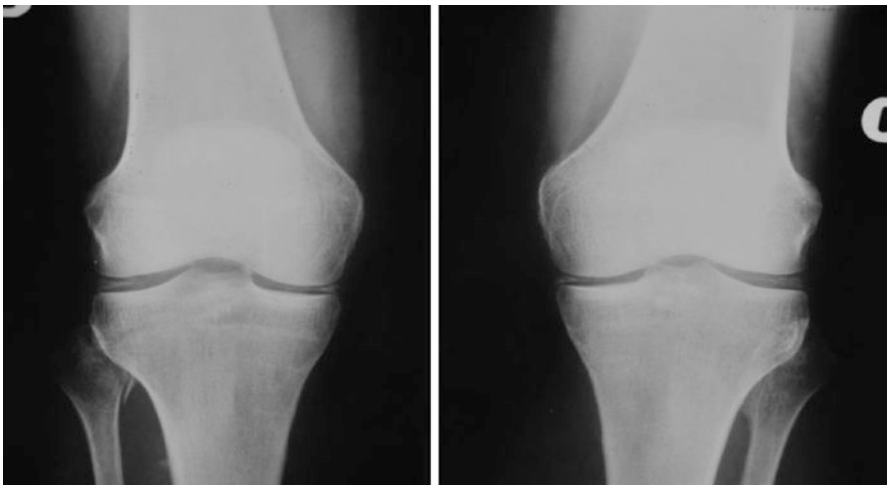

Fig. 5.7 Pyrophosphate arthropathy—pseudogout. (Courtesy: Figure reused with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

- Endocrine and metabolic disorders associated with CPPD crystal deposition disease (Table 5.10).

10. Crystal-induced arthritis (Table 5.11).

Table 5.10 Features of CPPD crystal deposition

Disorder	Probability of association
Hemochromatosis	Definite
Hyperparathyroidism	Definite
Hypophosphatasia	Definite
Gitelman's syndrome	Definite
Hypothyroidism	Probable
Gout	Possible
X-linked hypophosphatemic rickets	Possible
Familial hypocalciuric hypercalcemia	Possible
Hemosiderosis	Possible

Table 5.11 Features of crystal-induced arthritis

Characteristic	Gout	Pseudogout
Prevalence	Age: 58; men = 58/1000 and women = 11/1000	<1 case per 1000 individuals, increases with age
Crystal chemistry	Monosodium urate	Calcium pyrophosphate dehydrate
Appearance	Negative birefringent, needle shaped	Weakly positive birefringent; linear or rhomboidal
Articular involvement	Monoarticular > oligoarticular, Polyarticular <30%	Monoarticular > oligoarticular
Most frequently affected joints	First MTP joint, ankle, knees, others	Knee, wrist, others
Predisposing factors/risk factors	Hyperuricemia, obesity, hypertension, hyperlipidemia alcohol ingestion, lead ingestion, hereditary enzyme defect (rare)	Hypothyroidism, OA, chronic renal insufficiency, diabetes
Therapeutic options	Acute attacks-NSAIDs, colchicine Chronic management by urate-lowering agents like colchicine	Acute attacks-NSAIDs, colchicines Chronic management by NSAIDs+ colchicine

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The Forearm

6

K. Mohan Iyer

Pronator Teres Syndrome

Pronator teres syndrome is a compression neuropathy of the median nerve at the elbow. It is rare compared to compression at the wrist (carpal tunnel syndrome) or isolated injury of the anterior interosseous branch of the median nerve (anterior interosseous syndrome). Pronator teres syndrome (PTS), first described by Henrik Seyffarth in 1951, is caused by a compression of the median nerve (MN) by the pronator teres (PT) muscle in the forearm [1]. It is a compression of the median nerve as it passes between the two heads of the pronator teres muscle, bicepital aponeurosis. The compression is due to hypertrophy and imbalance of regional tissues resulting in the development of fibrous tissue due to the inflammatory processes from repetitive stress, decreasing range of motion, and increasing stresses.

Epidemiology

1. More common in women
2. Common in fifth decade
3. Has been associated with well-developed forearm muscles (e.g., weight lifters)

Pathoanatomy

There are five potential sites of entrapment, which include:

1. Supracondylar process residual osseous structure on distal humerus present in 1% of population

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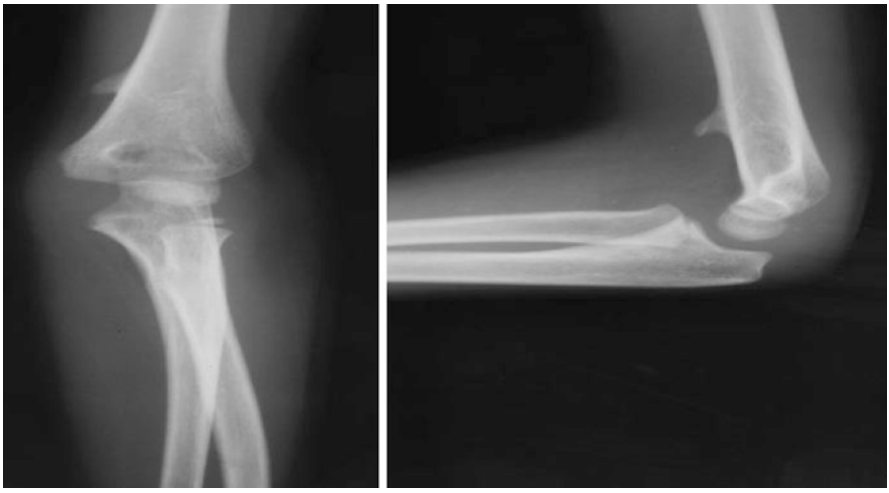

Fig. 6.1 Ligament of struthers. (Courtesy: Figure reused with the kind permission of Magdi E. Greiss Whitehaven, Cumbria, UK)

2. Ligament of struthers: It is a band that travels from tip of supracondylar process to medial epicondyle (Fig. 6.1).

   It should not be confused with arcade of Struthers, which is a site of ulnar compression neuropathy in cubital tunnel syndrome
3. Bicipital aponeurosis (a.k.a. lacertus fibrosus)
4. Between ulnar and humeral heads of pronator teres
5. FDS aponeurotic arch

It is also commonly associated with medial epicondylitis.

Signs and Symptoms

The presenting symptoms are paresthesias in thumb, index, middle finger, and radial half of ring finger as seen in carpal tunnel syndrome, but in pronator syndrome, paresthesias are often made worse with repetitive pronosupination. These symptoms are of an insidious onset without any history of trauma. There is an aching pain in the proximal, vola forearm with paresthesias radiating into the median nerve innervated fingers, which is worsened by repetitive pronation supination movements and wrist flexion.

Above all, it should have characteristics different from carpal tunnel syndrome (CTS):

1. Aching pain over proximal volar forearm.
2. Sensory disturbances over the distribution of palmar cutaneous branch of the median nerve (palm of hand) that which arises 4–5 cm proximal to carpal tunnel.
3. Lack of night symptoms.

Signs

There is a loss of sensation over the palmar cutaneous branch territory. The provocative tests are specific for different sites of entrapment

1. Positive Tinel sign in the proximal anterior forearm but no Tinel sign at wrist or provocative symptoms with wrist flexion, as would be seen in CTS
2. Resisted elbow flexion with forearm supination (compression at bicipital aponeurosis)
3. Resisted forearm pronation with elbow extended (compression at two heads of pronator teres)
4. Resisted contraction of FDS to middle finger (compression at FDS fibrous arch)

There could be a possible coexisting medial epicondylitis.

- Anterior Interosseous nerve syndrome: The site of compression is the same for both the Pronator Syndrome (PS) and the Anterior Interosseous Syndrome (AIN/Kiloh Nevin Syndrome).
- PS: It is vague volar forearm pain, median nerve paresthesias with a minimum of motor findings.
- AIN: Pure motor nerve palsy of any or all three, namely: (1) FPL, (2) FDP of the index and middle fingers, and (3) PQ.

It arises from the posterior surface of the median nerve 5–8 cm from the distal branch supplying the FDP, FPL, and PT.

Etiology: (1) Thickened fascia between the superficial and deep heads of the PT, (2) Tendinous origin of the deep head of PT and FDS, (3) An accessory tendon of FPL [Gantzer's muscle], (4) Thrombosed ulnar collateral vessels, and (5) Volkmann's ischemia.

Clinically, there is acute pain in the proximal forearm, with no sensory deficit seen usually, along with loss of pinching of the DIP of the index finger and the interphalangeal joint of the thumb (Spinner's sign). There is a deterioration of handwriting, along with weakness of FDP, FPL, and PT.

The pain may be present along the course of the nerve. Inability to make the `OK' sign when asked by the examiner. This exposes weakness of the FPL and the index finger FDP.

Differential diagnosis: (1) Brachial neuritis, (2) Viral neuritis (Parsonage-Turner syndrome), and (3) Rupture of FPL tendon.

Treatment: Initially, treatment is conservative; the surgical indications are if there are no relief of symptoms and severe symptoms. Surgical treatment is identification and division of the offending structure.

Imaging

Radiographs

The recommended views are the elbow films, which are mandatory. The findings may demonstrate a supracondylar process.

Diagnostic tests: EMG and NCV, which may be helpful if positive, but are usually inconclusive. It may exclude other sites of nerve compression or identify double-crush syndrome.

Treatment

(a) Non-operative to include rest, splinting, and NSAIDS for 3–6 months [indications being mild to moderate symptoms]. The technique is a splint should avoid forearm rotation.
(b) Operative: Surgical decompression of median nerve [indicated only when non-operative management fails for 3–6 months; the technique is decompression of the median nerve at all five possible sites of compression. The outcomes of surgical decompression are variable with 80% of patients having relief of symptoms.

A new Mini-invasive Decompression for Pronator Teres Syndrome has also been described, which considers the anatomic knowledge of this region. A 3.5-cm oblique skin incision is done 6 cm distal to the medial epicondyle, over the flexor/pronator muscle mass. The medial cutaneous nerve is atraumatically retracted. The superficial fascia of the flexor/pronator muscles is opened transversely. With the section of the septum between the pronator teres and the flexor carpi radialis, access to the deep fascia of the superficial head of the pronator teres is obtained. This structure is released. The median nerve is now easily visualized, and other types of possible compression causes are excluded. The overall results were 93% [2].

Congenital Radio-Ulnar Synostosis

It is a rare condition of upper limb skeletal malformation characterized by bony fusion of the radius and ulna. The condition is caused by a failure of segmentation between the radius and ulna. An initial descript of this rare condition was by Standifort in 1793, and Gros described the post-traumatic radio-ulnar synostosis in 1864 [3]. Synostosis between the radius and ulna can take two general forms: congenital and posttraumatic. Each form may be further classified into types. Embryologically, the upper limb bud arises from the unsegmented body wall at 25–28 days. The elbow becomes visible at 34 days, and the humerus, radius, and ulna become visible at 37 days. Initially, the three cartilaginous analogues of the

humerus, radius, and ulna are connected before segmentation. Therefore, for a short time, the radius and ulna share a common perichondrium. Abnormal events at this time can lead to a failure of segmentation. The duration and severity of the insult can determine the degree of subsequent synostosis. The skeletal anomaly includes varying degrees of proximal radial and ulnar fusion, with or without involvement of the radial head. If the radial head is involved, it may be dislocated anteriorly or posteriorly [4]. A fibrous synostosis may allow limited motion. Regional soft-tissue hypoplasia is often present in severe cases, including those in which atrophy and fibrosis of the brachioradialis, pronator teres, pronator quadratus, and supinator muscles occur. The interosseous membrane also may be abnormal. Although the condition is present at birth, it usually is not discovered until early adolescence, when the patient presents with a lack of pronation and supination.

Etiology: The most common cause of posttraumatic radioulnar synostosis is an operatively treated forearm fracture. Patients with high-energy comminuted open fractures appear to be more likely to develop this complication. Monteggia and proximal forearm fractures also appear to have a higher incidence of synostosis [5]. Patients with closed head injuries (skull/cranial trauma) appear to be more prone to this complication, presumably for the same reason that they develop heterotopic ossification [6].

Epidemiology: Because congenital radioulnar synostosis is caused by an in-utero insult, its association with other abnormalities is not surprising.

Sign and symptoms: Functional deficits associated with congenital radioulnar synostosis depend on the severity of the deformity and on whether or not it is bilateral. In cases involving severe, fixed forearm pronation deformity, the patient cannot compensate for the resulting functional limitations by using scapular and glenohumeral motion. The forearm usually lies in the pronated or hyperpronated position.

Hypermobility at the midcarpal and radiocarpal joints can disguise this lack of forearm rotation, particularly with neutral or mild pronation deformities. There is usually full or nearly full elbow range of motion, with flexion contractures rarely exceeding 30°. An abnormal carrying angle of the elbow or a shortening of the forearm may be observed.

Classification

Wilkie divided congenital synostosis into the following two types based on the proximal radioulnar junction [7].

- Type 1—Complete synostosis has occurred, with the radius and ulna fused proximally for a variable distance.
- Type 2—Less involved, and may exist as a partial union; this type involves the region just distal to the proximal radial epiphysis and is associated with radial head dislocation.

Cleary and Omer described four types of congenital synostosis, as follows [8]

1. Fibrous synostosis
2. Bony synostosis
3. Associated posterior dislocation of the radius
4. Associated anterior dislocation of the radius

Posttraumatic radioulnar synostosis has been classified into the following three types based on the location:

- Type 1—Least common; occurs in the distal forearm
- Type 2—Occurs in the midforearm
- Type 3—Occurs in the proximal forearm

Imaging Studies

Plain radiographic imaging in orthogonal (e.g., posteroanterior [PA] and (lateral) planes are recommended for the workup of patients with congenital or posttraumatic radioulnar synostosis (Fig. 6.2)

Management

1. Conservative: Observation.
2. Surgical: Osteotomy with fusion, surgical resection of synostosis with irradiation and indomethacin or proximal radial excision.

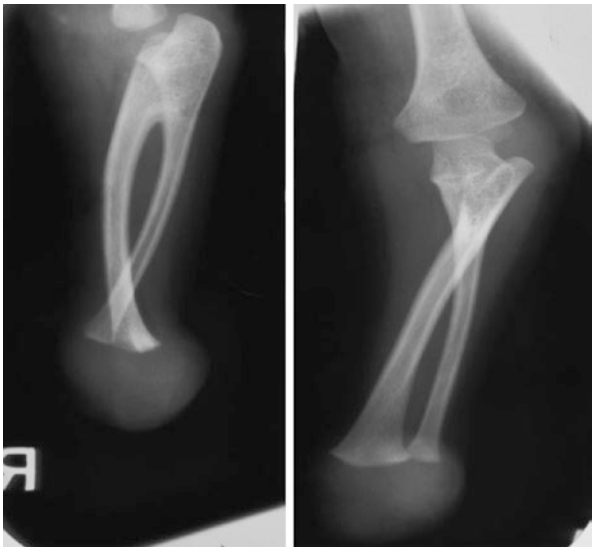

Fig. 6.2 Congenital radio-ulnar synostosis. (Courtesy: Figure reused with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

Post-operative rehabilitation: This is done by splinting in maximum pronation and supination between active and passive physiotherapy.

Biomechanics of the Elbow Joint

The elbow complex includes the elbow joint (Humeroulnar and Humeroradial joints) and the Proximal and Distal radioulnar joints. It is mainly considered as a modified or loose hinge joint. One degree of freedom is possible at the elbow joint (Flexion + Extension), which occurs at a saggital plane around a coronal axis. A slight bit of axial rotation and side-to-side motion of the ulna occur during flexion and extension and that is why the elbow joint is considered as a modified or loose hinge joint. The joint is associated with two major ligaments and five muscles directly associated with the elbow joint. Three of the muscles cross the anterior aspect of the joint, while two others are extensors that cross the posterior aspect of the elbow joint.

The proximal and distal radioulnar joints are linked and function as one unit. These two joints acting together produce rotation of the forearm and have one degree of freedom of motion. These radioulnar joints are diarthrodial uniaxial of the pivot type, and hence permit rotation (supination and pronation), which occurs in transverse plane around a vertical axis. Six ligaments and four muscles are associates with these joints, namely, two for pronation and two for supination.

Structure of the elbow (HU and HR) articulations: Articulation between the ulna and humerus at the humeroulnar joint primarily occurs as a sliding motion of the ulnar troclear ridge on the humeral troclear groove. Articulation between the radial head and the capitulum at the humeroradial joint involves sliding of the shallow concave radial head over the convex surface of the capitulum.

Joint capsule: The HU along with the HR and the superior radioulnar joint are enclosed in a single joint capsule. This capsule is large, loose, and weak anteriorly and posteriorly, and contains folds that can unfold to allow for a full range of elbow motion. The capsule is reinforced by collateral ligaments.

Ligaments

1. Collateral ligaments: Medial and lateral collateral ligaments.
2. Annular ligament.
3. Transverse ligament.

Function of the MCL: Also called the ulnar collateral ligament that stabilizes against valgus torque. It also limits extension at the end of elbow extension ROM. It helps to guide joint motion through flexion ROM. It provides resistance to longitudinal distraction of joint surfaces.

Function of LCL: Also called radial collateral ligament that stabilizes the elbow against varus torque. It also stabilizes against combined varus and supination torque.

It reinforces the HR joint and assists in providing some resistance to longitudinal distraction of the articular surfaces. It also prevents subluxation of the humeroulnar joint.

Muscles

1. The major flexors of the elbow joint are the brachialis, the biceps brachii and the brachioradialis.
2. The brachialis muscle arises from the anterior surface of the lower part of the humeral shaft and attaches by a broad thick tendon to the ulnar tuberosity and the coronoid process.
3. The biceps brachii arises from two heads, one long and the other short. The short head arises as a thick flat tendon from the coracoid process of the scapula, while the long head arises by a narrow tendon from the supraglenoid tubercle, and both the heads insert by way of a strong flat tendon on the rough surface of the posterior area of the tuberosity of the radius and into the bicipital aponeurosis.
4. The brachioradialis muscle arises from the lateral supracondylar ridge of the humerus and inserts into the distal end of the radius just proximal to the radial styoid.
5. The extensors of the elbow are the triceps and the anconeus. The triceps has three heads, namely, the long, medial, and lateral. The long head arises from the infraglenoid tubercle of the scapula by a flattened tendon that blends with the glenohumeral joint capsule, while the medial and lateral heads cross the elbow joint. The medial head arises from the entire posterior surface of the humerus, while the lateral head arises from only a narrow ridge on the posterior humeral surface. These three heads insert by a common tendon into the olecranon process. The anconeus is small triangular muscle that arises from the posterior surface of the lateral epicondyle of the humerus and extends medially to attach to the lateral aspect of the olecranon process and the proximal part of the posterior surface of the ulna.

Long Axes of the Humerus and the Forearm

When the upper extremity is in the anatomical position, the long axis of the Humerus and the long axis of the Forearm form an acute angle when they meet at the elbow. The angulation in the frontal plane is caused by the configuration of the articular surfaces at the humeroulnar joint.

The medial aspect of the trochlea extends more distally than does the lateral aspect, which shifts the medial aspect of the ulnar trochlear notch more distally to result in a lateral deviation (or valgus deviatrion) of the ulna with the humerus. This normal valgus deviation is called the carrying angle or cubitus valgus. The average angle in full elbow extension is about 15°.

An increase in the carrying angle beyond the average is considered to be abnormal, particularly when it is unilateral. A varus angulation of the elbow is referred to as cubitus varus and is usually abnormal. Normally, the carrying angle disappears when the forearm is pronated when the elbow is in full extension, and when the supinated forearm is flexed against the humerus in full elbow flexion.

Range of motion: The range of movement in the elbow is from 0° of elbow extension to 135–145° of elbow flexion. The stability of the elbow joint depends on the configuration of the joint surfaces, the ligaments, and the joint capsule. The elbow joint has inherent stability at the extremes of extension and flexion. In full extension, the humeroulnar joint is in a close-packed position. In this position, the bony contact of the olecranon process in the olecranon fossa limits the end of the extension range, and the configuration of the joint structures helps to provide valgus and varus stability. The bony components, the MCL, and the anterior joint capsule may contribute equally to resists valgus stress in full extension.

Radioulnar joint: The radius and ulna articulate with each other at their proximal and distal ends at synovial joints, called the proximal and distal radioulnar joints. These articulations are the pivot type of synovial joint that produces supination and pronation.

Proximal radioulnar joint: Here the radial head articulates with the radial notch of the ulna, the radial head is held strongly by the annular ligament.

Distal radioulnar joint: Here the head of the ulna articulates with the ulnar notch of the radius.

The Proximal and Distal radioulnar joints are mechanically linked, and hence motion at one joint is always accompanied by motion at the other joint. The ligaments involved are (1) Annular, (2) Quadrate, and (3) Oblique cord.

The muscles involved are pronator teres, pronator quadrates along with the biceps brachii and supinator.

Range of motion: In supination, the radius and ulna lie parallel to one another, while in pronation, the radius crosses over the ulna. The normal range of forearm pronation-supination averages from 71° of pronation to 81° of supination. Most activities are accomplished in this function range of 50° pronation to 50° supination.

Injuries around the Elbow Joint are (1) Compression injuries, (2) Distraction injuries, and (3) Varus/Valgus injuries.

1. Compression injuries: Falling on the hand when the elbow is in an extended position, which may involve transmission of forces through the bones of the forearm to the elbow. Transmission of forces from the hand to the elbow may occur through either the radius or ulna or both.
2. Distraction injuries: Nursemaid elbow: This is a common injury in children younger than five years and rare in adults. Lifting a small child hand up in the air by one or both hands or a pull on the hand creates tensile forces at the elbow and the radial head is pulled out of the annular ligament.

3. Varus/Valgus injuries: The application of a valgus stress on the forearm produces a compression on the lateral aspect of the elbow joint with tensile forces on the medial aspect of the joint. Similarly, the application of a varus stress to the forearm produces a tensile stress on the lateral aspect of the elbow joint and compression on the medial aspect of the joint.

Carpal Tunnel Syndrome

It is a nerve compression syndrome where the median nerve is frequently compressed at the wrist because of its vulnerable anatomic position. This syndrome is a pressure on the median nerve that supplies the feeling and movements to parts of the hand, which may lead to numbness, tingling, weakness, or muscle damage in the hand and fingers.

Several risk factors are involved in its production, such as (1) sewing, (2) painting, (3) driving, (4) writing, (5) use of tools (especially hand tools that vibrate), (6) sports such as handball, and (7) playing certain musical instruments.

Causes: The most common factor is idiopathic, where the exact cause is unknown. In general, anything that crowds, irritates, or compresses the median nerve in the carpal tunnel can lead to carpal tunnel syndrome.

1. Bone fractures or arthritis of the wrist.
2. Acromegaly.
3. Diabetes.
4. Alcoholism.
5. Hypothyroidism.
6. Kidney failure and dialysis.
7. Menopause, premenstrual syndrome (PMS), and pregnancy.
8. Infections.
9. Obesity.
10. Rheumatoid arthritis, systemic lupus erythematosus (SLE).
11. Synovitis.
12. Excessive hand exercise.
13. Odema or hemorrhage of the median artery.
14. Thrombosis of the median artery.

Incidence

- Prevalence: It occurs in approximately 50 cases per 1000 of the population.
- Race: The whites are probably at highest risk.
- Gender: The female-to-male ratio for carpal tunnel syndrome is 3–10:1.
- Age: The peak age range for the development of carpal tunnel syndrome (CTS) is 45–60 years with only 15% of patients with CTS are younger than 31 years.

Pathophysiology

The sequence of events are as follows: Due to etiologic factors → synovium swollen → pressure on the median nerve → temporary blockage of myelinated nerve fibers → numbness of the fingers → continued pressure causes → Ischemia, axonal death, muscular dystrophy, and pain.

Signs and Symptoms

1. Tingling.
2. Numbness or discomfort in the lateral 3, 1/2 fingers.
3. Intermittent pain in the distribution of the median nerve.
4. Symptoms get aggravated at night.
5. To relieve the symptoms, the patient often flick their wrists as if shaking down a thermometer (flick sign).

Motor Changes
1. Apelike thumb deformity.
2. Loss of opposition of the thumb.
3. Index and middle finger lag behind when making a fist.

Sensory Changes
1. Loss of sensation of lateral 3, 1/2 digits including the nail bed and distal phalanges on dorsum of the hand.
2. It is important to remember that there is no sensory loss over the thenar eminence in carpal tunnel syndrome as branch of the median nerve (palmar cutaneous branch) passes superficial to the carpal tunnel and not through it.

Vasomotor Changes: The skin area with sensory loss is warmer and the skin is dry.

Trophic Changes: (1) Long-standing cases lead to dry and scaly skin, (2) nails crack easily, and (3) Atrophy of the pulp of the fingers (thenar muscles).

Management

Physical assessment tests:

1. Less sensitivity to pain where the median nerve runs to the fingers.
2. Thumb weakness.
3. Inability to tell the difference between one and two sharp points on the fingertips.
4. Flick signal: The patient is asked ‘What do you do when the symptoms are worse?’

5. Phalen's test:
   The patient rests his elbows on the table and the wrists dangle (in flexion) with the fingers pointing down and the back of the hands pressed together. This test is positive (CTS) if symptoms develop with a minute.
6. Tinel's sign test: Here the examiner taps over the median nerve to produce a tingling or mild shock sensation.
7. Durkan Test (carpal compression test): With the wrist in neutral position and the forearm supinated, a pressure over the carpal tunnel for 30 s produce a tingling or shock over the median nerve.
8. Hand elevation test: This is positive when symptoms of CTS are reproduced by the patient holding his or her hand overhead actively against gravity without strain for 2 min.
9. Tourniquet test: This is positive when a tourniquet is inflated above the systolic pressure for one minute intensifies the symptoms of CTS.

Diagnostic evaluation is done by history, clinical examination, and nerve conduction study. A nerve conduction study (NCS), which is also called as nerve conduction velocity (NCV), is a test of the measurement of the speed of an electrical impulse. NCS can determine nerve damage and destruction. During this test, the nerve is stimulated with surface electrode patches attached to the skin.

Management: (1) General measures, (2) wrist splints, (3) oral medications, (4) local injection, and (5) ultrasound therapy.

1. General measures: Avoid repetitive wrist and hand motions that may exacerbate symptoms or make symptom relief difficult to achieve. Avoid the use of vibratory tools. Ergonomic measures to relieve the symptoms depending on the motion need to be minimized.
2. Wrist splints: They are probably most effective when applied within three months of the onset of symptoms. A wrist hand orthosis, cock-up wrist splint, or thumb spica splint helps manage the pain of CTS.
3. Oral medications: (a) Diuretics: NSAIDs or pryridoxine containing vitamin B6. Lastly, oral corticosteroids such as prednisolone (20 mg/day followed by 10 mg/day for 2 weeks).
4. Local injection: Local corticosteroid injection may be given.
5. Ultrasound therapy: Predicting the outcomes of conservative treatment.
6. Surgery: Surgery should be considered if more than two injections are needed. It should also be considered in patients with symptoms that do not respond to conservative measures. Patients with severe nerve entrapment as indicated by nerve conduction studies, thenar atrophy, or motor weakness are ideal candidates for surgery.

Open Release (cutting the flexor retinaculum): Here the surgeon makes an incision less than 5 cm long in the palm, and perhaps into the wrist, as well to expose the transverse carpal ligament, after which the surgeon cuts the ligament to reduce the pressure on the underlying nerve. After which, only the skin is closed.

Endoscopic release: Here the endoscope is guided through a single small incision at the wrist (single-portal) technique or at the wrist and the palm (two-portal technique).

This endoscope allows the surgeon to see the structures at the wrist such as the transverse carpal ligament without the need to open the entire area through a large incision.

Opioid consumption following CTS surgery is more commonly influenced by age and gender, and less influenced by the type of anesthesia, type of insurance, or the opioid prescribed. More and more opioids are prescribed than needed on an average of 5:1. Even many older patients do not require opioid analgesia after CTR [9].

Prognosis

Surgery is usually successful, although in some cases it does not completely relieve numbness in the hand and fingers. This may also be the case in long standing cases, as permanent damage may be cause by long-standing CTS or other health problems such as diabetes.

Complications following surgery:

The most common complications of carpal tunnel release are as follows:

1. Nerve damage with tingling and numbness (usually temporary)
2. Infection
3. Scarring
4. Pain
5. Stiffness
6. Loss of some wrist strength is a complication that affects 10% to a third of the patients

Prevention

1. Avoid or reduce the number of repetitive wrist movements whenever possible.
2. Use of tools or equipment properly designed to reduce the risk of wrist injury.
3. Ergonomic aids, such as split keyboards, keyboard trays, typing pads, and wrist braces may be used to improve posture during typing.
4. Take frequent breaks during typing and always stop when there is tingling or pain.

Manual physical therapy versus surgery for CTS: The surgery and physical manual therapies including desensitization maneuvers of the central nervous system were similarly effective at medium-term and long term follow-ups for improving pain and function [10].

Conclusion

Carpal tunnel syndrome is a disease of the median nerve that affects more white or females after the age of 30 years. It affects the neuro sensory function of the hand, and if left untreated can cause permanent loss of function of the affected area.

Volkmann's Ischemic Contracture

It is a condition characterized by ischemic necrosis of the structures contained in the volar compartment of the forearm associated with crippling contractures and varying degrees of neurological deficit.

In 1881, Volkmann stated that the paralytic contractures that develop with a few hours of injury are caused by arterial insufficiency of muscles [11]. In 1906, Hilderbrand [12] first used the term 'Volkmann's ischemic contracture' to describe the final result of any untreated compartment syndrome. In 1909, Thomas [13] found that paralytic contractures followed severe Hilderbrand contusions of the forearm without fractures. In 1914, Murphy [14] reported that increased internal pressures in the deep compartments of the forearm and effusion in the muscles resulted in ischemia. In 1928, Jones [15] concluded that Volkmann's contracture could be caused by pressure from within or without or both. Volkmann's ischemic contracture is a late sequela of untreated or inadequately treated compartment syndrome in which the necrotic muscle and nerve tissue are replaced with fibrous tissue.

Severe ischemic insult may have three outcomes, namely:

1. Complete recovery
2. Gangrene
3. Middle course (Contracture)

The difference between gangrene and ischemic contracture is not purely quantitative: gangrene involves all the tissues while contracture is selective (muscles and nerves).

Relevant anatomy: At the entrance of the flexor compartment of the forearm, lacertus fibtosus fans medially from the biceps tendon. Beneath the lacertus fibrosus, the brachial artery and the median nerve pass to enter the flexor compartment.

The brachial artery then divides into the radial and ulnar arteries. The radial artery courses superficially and is not crossed by any structure in the forearm. The ulnar artery passes beneath the pronator teres where it gives a branch, the common interosseous artery, which then further divides into the volar and dorsal interosseous arteries.

The median nerve accompanies the brachial artery beneath the lacertus fibrosus to enter the substance of pronator teres passing between its humeral and ulnar heads.

Compartments of the forearm are as follows:

1. Superficial volar compartment,
2. Deep volar compartment,
3. Dorsal compartment, and the
4. Mobile wad of Henry. The mobile wad (or mobile wad of Henry) is a group of the following three muscles found in the posterior compartment of the forearm: brachioradialis, extensor carpi radialis brevis, and extensor carpi radialis longus. It is also sometimes known as the lateral compartment or radial group of the forearm.

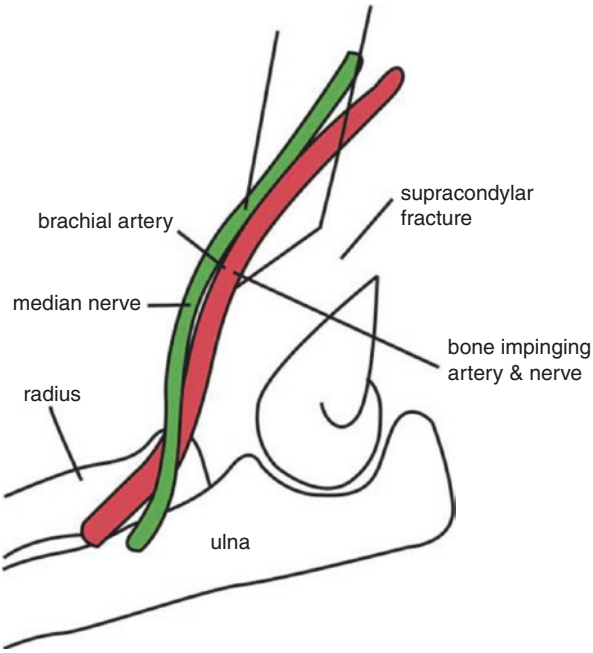

Fig. 6.3 Line diagram showing the common mechanism of injury in supracondylar fracture

Etiology

Supracondylar fractures in children are the most common precipitating injury. The brachial artery may be impinged on the sharp proximal fragment against which it is held by the lacertus fibrosus (Fig. 6.3).

Hemorrhage and odema may further compress the brachial artery and the median nerve in this region.

1. Crush injuries
2. Prolonged external compression
3. Internal bleeding (Hemophilia)
4. Burns
5. Snake bites
6. Intravenous regional anesthesia.

Tolerance of Tissue

1. Muscle: Functional impairment after 2–4 h of ischemia; Irreversible functional loss after 4–12 h.
2. Nerves: Functional impairment after 30 min of ischarmia; Irreversible functional loss after 12–24 h.

Clinical Picture

Acute compartment syndrome (impending Volkmann's contracture):

1. If local compression is the cause, then the pulses are intact (in early stages), paresis, stretch pain, paresthesia (median nerve sensory zone commonly), with good capillary filling.
2. If arterial injury is the cause, stretch pain, paresthesia, pulsenessless, pallor (cyanosis), and paresis.

Two-point discrimination is more than 1 cm in the sensory zone of the median nerve. Diminished perception of vibratory sense of 256 cycles/s stimulus.

Measurement of Intracompartment Pressure

1. Whitesides handheld pressure monitoring system
2. Wick catheter
3. Slit catheter

Evaluating the compartmental pressure: A range between 10 and 20 mm Hg below the diastolic pressure indicates that cessation of blood flow is imminent. A range of 40 – 50 mm Hg indicates that threatening compression and ischemia are present. A pressure of 30 mm Hg or greater is a criterion for fasciotomy.

Deformities in Volkmann's ischemic contracture are as follows:

1. Mild type: (a) The deep flexors are involved partially, particularly flexor digitorumprofundus. (b) Flexion contractures of one or more fingers that can be extended on hyperflexing the wrist. (c) Resistant pronation contracture involving the pronator teres or pronator quadrates.
2. Moderate type: (a). Involves most of flexor digitorum profundus, flexor pollucis longus, and part of flexor digitorum superficialis. (b) Neurological deficit involving median nerve more than ulnar nerve is present. (c) Deformity is an intrinsic minus deformity. (d) Diminished sensations over the median and ulnar nerves zones.
3. Severe type: (a) All the flexor muscles are involved. (b) Neurological deficit is severe. (c) Joint contractures are marked. (d) Wasing of the forearm muscles.

Management/Acute Compartment Syndrome

This is a surgical emergency.

1. Forearm fasciotomy: The incision is a volar curvilinear liberal one medial to the biceps tendon and crossing the elbow flexion at an angle carrying it distally to the palm to release the carpal tunnel.

2. The incision must extend deeply to explore the FDP and FDL. Necrotic tissue is excised. The median nerve is freed beneath the lacertus fibrosus and he ulnar nerve is freed and transplanted anteriorly. The brachial artery must be inspected and decompressed. The surgical wound is then left open for a secondary suture at a later date when the swelling subsides. The extremity is supported with a splint in the functional position.

Conservative treatment of established deformities: Robert Jones in the 1930 treated this using a wooden tongue depressor to correct the established deformities gradually from distal to proximal over a prolonged period. A Banjo splint was used with rubber bands fastened to adhesive tape on the fingers, which also permits exercises at all times and is most efficient.

Established Volkmann's ischemic Contracture Management

1. Muscle slide operation the flexors of the forearm.
2. Technique of Inglis and Cooper: The incision is on the medial aspect of the volar side of the arm 5 cm proximal to medial epicondyle and distally to the midpoint of the forearm over the ulna. The ulnar nerve is then identified and released from the cubital tunnel and protected. The tendinous origins of the muscles on the medial epicondyle are cut, with the flexor carpi ulnaris and the flexor digitorum profundus being completely released from the medial epicondyle and the ulna. The lacertus fibrosus is divided along with any remaining portions of the flexor muscle origin and the ulnar nerve is then transposed anteriorly.
3. Technique of Williams and Haddad: In this procedure, the incision is on the medial aspect of the arm and forearm anterior to the medial epicondyle of the humerus, beginning 5 cm proximal to the elbow and extending distally to 5 cm proximal to the wrist when the structures on the anterior and medial aspect of the elbow are exposed. The lacertus fibrousus is then divided and the origins of the superior flexor muscles are released from the medial epicondyle, and the origin of the flexor digitorum superficialis is released from the radius. The origin of the flexor carpi ulnaris is then released from the olecranon and the common origins of the flexor carpi ulnaris and flexor digitorum profundus are released from the ulna. The origin of the flexor digitorum profundus is released from the volar aspect of the ulna and the interosseous membrane. The origin of the flexor digitorum profundus to the index finger is then released from the radius. The ulnar nerve is transplanted anteriorly into the brachialis muscle. The sutures are removed after 3 weeks and an extension hand splint should be worn for 3 months. The occupation and physiotherapy should be continued until the desirable function has been achieved.

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Clinical Examination of the Wrist

7

Lynette Spalding and Wasim S. Khan

Introduction

The wrist is a complex anatomical region connecting the forearm and hand. It comprises multiple bones and their articulations, supporting ligaments, and joint capsules plus muscles acting on the wrist or traversing it via tendons to act in the hand, accompanied by neurovascular structures. As such, an examination of the wrist would not be complete without assessment of the joint above and the joint below, i.e., examination of the elbow and hand.

In this chapter, we aim to provide an overview of the clinical assessment of the wrist, with a structured sequence of examination following the Orthopedic mantra of 'look, feel, move, special tests'. In reality, this of course would be supplemented by a thorough history and subsequent investigations as indicated, which will be discussed further in the chapters regarding pathology of the wrist and hand.

Regarding special tests, there are multitudes available, and their use is dependent on the site of pathology identified in the initial examination. We will present a rationale of the use of special tests to confirm or refute a differential diagnosis based upon the location of symptoms in either the radial, ulnar, or central areas of the wrist (Table 7.1). Clearly, some diagnoses such as ganglion or fracture ± nonunion can present in any area. However, we will highlight the more common sites.

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Table 7.1 Wrist pathology by anatomical location

Radial	Central	Ulnar
Scaphoid fracture non-union	Central instability	DRUJ degeneration
Scapho-lunate instability	Lunotriquetral instability	TFCC tear
STT degeneration	Intersection syndrome	ECU instability
First CMCJ degeneration	Kienbock's disease	Pisotriquetral degeneration
De Quervain's tenosynovitis	Carpal tunnel syndrome	Hook of hamate non-union
Preiser disease		Ulna nerve

Anatomy of the Wrist

A detailed discussion of the anatomy of the wrist is outside the remit of this chapter. However, an overview is important in localizing the structure(s) causing the patients' symptoms.

The bony anatomy of the wrist comprises the distal radius, distal ulna, and the eight carpal bones. The articulations involved are the distal radioulnar joint, radiocarpal, and ulnocarpal joints and intra-carpal joints [1].

The eight carpal bones are arranged in two rows to make up the carpus. The proximal row comprises (from lateral to medial) the scaphoid, lunate, triquetrum and pisiform (a sesamoid bone within the tendon of extensor carpi ulnaris), and articulates with the distal radius and ulna/TFCC. The distal row is made up of (from lateral to medial) the hamate, capitate, trapezoid and trapezium, and articulates with the base of the metacarpals. This articulation allows mobility in the thumb, stability in the second and third carpo-metacarpal joints (CMCJ), and increased mobility in the fourth and fifth CMCJ [1]. The articulation between the proximal and distal carpal rows forms the S shaped midcarpal joint [2]. There is considerable variation in the size and articulation of the carpal bones from the smallest pisiform, which articulates with one bone to the largest capitates, which articulates with seven other bones [1].

The scaphoid and lunate articulate with the distal radius to form the radiocarpal joint, and the triquetrum articulates with the triangular fibrocartilage to form the ulnocarpal joint. Interosseous intrinsic ligaments connect the carpal bones in the proximal and distal carpal rows whilst extrinsic ligaments span from the distal radius and ulna across the carpal rows. There are both intrinsic and extrinsic ligaments on the volar and dorsal surfaces of the wrist [1], and these can be the site of pain or instability.

The distal radioulnar joint is formed between the sigmoid notch of the radius and the distal ulnar convexity [1], with the radius rotating around the stationary ulna. In an ulnar neutral wrist, only 20% of axial force is transmitted via the ulna, more if ulna positive [3]. The triangular fibrocartilage complex (TFCC) is interposed between the distal ulna and the triquetrum in the ulnocarpal joint. The TFCC originates from the sigmoid notch of the radius and inserts at the base of the ulna styloid. It is made up of a central disc with dorsal and volar radio-ulnar ligaments [3] plus many, which include the ulnar collateral ligament, meniscal homologue, extensor carpi ulnaris sheath, ulnolunate, and ulnotriquetral ligaments in its structure [1].

Overlying the bony structures on the dorsal aspect of the wrist lie the extensor tendons within six fibrosseous compartments, with the extensor retinaculum securing them to prevent bowstringing. Compartment 1 contains extensor pollicis brevis and abductor pollicis longus. Compartment 2 contains extensor carpi radialis brevis and extensor carpi radialis longus. Extensor pollicis longus lies within compartment 3 and travels around Lister's tubercle on the distal radius. Compartment 4 contains extensor digitorum and extensor indicis. Extensor digiti minimi runs in compartment 5, and compartment 6 contains extensor carpi ulnaris [3]. Only the contents of compartments 2 and 6 act on the wrist, the others traverse the wrist to act in the hand.

The volar aspect of the wrist is characterized by the carpal tunnel; bordered by the trapezium and scaphoid laterally plus pisiform and hamate medially with the carpus as its floor and flexor retinaculum as its roof. The carpal tunnel contains the four flexor digitorum superficialis, four flexor digitorum profundus, and one flexor pollicis longus tendons plus the median nerve [3]. Flexor carpi radialis runs in the groove on trapezium within a subcompartment of the carpal tunnel [2] to attach to the base of the second and third metacarpals [3]. The radial artery lies lateral to FCR at the wrist [3].

Medial to the carpal tunnel lies the ulnar tunnel/Guyon's canal with the transverse carpal ligament as its floor, volar carpal ligament as its roof, and bounded by the pisiform medially and hook of hamate laterally. The ulnar tunnel contains the ulnar nerve and ulnar artery [3]. Palmaris longus, if present attaches to the superficial flexor retinaculum/palmar aponeurosis [3]. The most medial tendon on the volar aspect of the wrist is flexor carpi ulnaris, attaching to/containing pisiform and attaching to the hook of hamate and fifth metacarpal [3].

Clinical Examination

With an appreciation of the anatomical structure of the wrist, attention can then be turned to clinical examination, following the standard protocol of 'look, feel, move'. In some conditions, symptoms and signs may be bilateral but often they are unilateral, therefore it can be very informative to compare the two sides. The arms should be exposed from elbow to fingertip to allow a thorough examination of the dorsal and volar surfaces of the limb. Both systemic and localized pathology should be considered. The examples given are by no means exhaustive but designed to demonstrate a thinking process during examination.

Look

Observe the overall posture of the limb. Are there any deformities that could suggest contracture, and therefore limited function? Is there any generalized wasting that may suggest a neurological lesion?

Observe the posture of the wrist. Are there deformities characteristic of rheumatoid arthritis such as radial deviation? Is the distal ulna prominent? Are there dropped fingers suggesting tendon ruptures? Previous trauma may have caused

deformity such as the dinner fork deformity seen with a Colles' fracture. Localized wasting may suggest a peripheral nerve lesion.

What condition is the skin in? Glossy skin with discoloration and increased hair and nail growth may suggest a pathology such as Complex Regional Pain Syndrome [4]. Bruising may suggest steroid use. Skin lesions such as psoriatic lesions or pitted nails could suggest psoriatic arthropathy. Lack of protective sensation with nerve pathology can lead to inadvertent injury. Skin condition must be noted, if considering operative intervention, as potentially impaired healing or increased risk of infection may be suggested.

Are there scars from previous surgery or trauma?

Is there any swelling present? This may represent trauma, especially in the presence of bruising. Diffuse swelling may represent infection or inflammation, especially if combined with erythema. Synovitis may present as a diffuse boggy swelling. More localized swelling may suggest ganglion, or tumor (benign or malignant).

Are there color changes such as erythema suggesting infection or pale/blue discoloration suggesting vascular spasm/pathology?

Feel

Assess the temperature of the wrist. Increased warmth suggests infection or inflammation. A cool limb may be consistent with vasospasm in Raynaud's.

Palpate any swelling to assess for discomfort and the character of the swelling. Is there an underlying bony deformity? Is a localized selling fixed or moves with a tendon. A soft ganglion may be balottable but a small tense one may not.

Palpate the bony structures of the wrist in a systematic fashion, assessing carefully for tenderness and crepitus. As with any bone, tenderness to palpation may indicate fracture or non-union.

The radial styloid is easily identifiable on the lateral aspect of the wrist. Bony tenderness here can be due to fracture non-union or degenerative change. The carpus can then be sequentially palpated for bony tenderness and intrinsic or extrinsic ligament tenderness suggesting possible injury. The scaphoid can be palpated dorsally in the anatomical snuffbox and the scaphoid tubercle can be palpated on the volar aspect of the wrist. Tenderness may be due to fracture or non-union. The lunate can be identified medial to the scaphoid or just proximal to the capitate [5]. It is a common site of pathology following trauma, being the most frequently dislocated and second most frequently fractured bone in the wrist [5]. Tenderness between the two bones may indicate scapholunate ligament pathology. The triquetrum can be palpated just distal to the ulna styloid by radial deviation of the wrist [6]. The pisiform can be easily palpated medial to the triquetrum. Pisotriquetral joint pathology may cause tenderness here. In the distal carpal row, the body of the hamate can be palpated dorsally, and the hook from the volar aspect, located 2 cm from the pisiform on a line between the pisiform and the head of the second metacarpal [5]. The capitate is palpable proximal to the base of the third metacarpal [7]. The remaining two carpal bones, the trapezoid and trapezium make up the

scapho-trapezio-trapezoid (STT) joint along with the scaphoid, and tenderness here can be due to STT degeneration. The distal ulna can be palpated for tenderness or increased mobility

Next, the TFCC can be palpated in the depression between the pisiform, flexor carpi ulnaris, and ulnar styloid [8, 9]. TFCC tear or degeneration may cause tenderness here.

Sequential palpation of the extensor tendon compartments may reveal tenosynovitis, known as de Quervain's tenosynovitis if affecting the first dorsal compartment. Intersection syndrome may be identified if pain elicited at the site of crossover of the first and second extensor compartments, proximal to Lister's tubercle [10].

Move

Assess both active and passive ranges of motion, comparing sides.

Test wrist flexion and extension from neutral. Normal range of 0–60° flexion and 0–50° extension [11].

Assess forearm pronation and supination with elbow flexed to 90°. Normal range of 0–90° pronation and 0–90° of supination [5].

Radial deviation of wrist has a normal range of 0–15° and ulnar deviation has a range of 0–50° [11].

If there is an increased range of movement, assessing the patient's Beighton score will test for any generalized joint laxity [12].

Special Tests

Returning to the table of differential diagnoses dependant on site of positive examination findings, there are special tests (Table 7.2) that can be employed to narrow the differential.

Table 7.2 Special tests useful for differential diagnosis

Radial	Central	Ulnar
Scaphoid fracture non-union Scaphoid tenderness	Midcarpal instability Lichtman test	DRUJ degeneration/instability Piano key sign/ulna compression
Scapho-lunate instability Kirk Watson test	Lunotriquetral instability Regan/Kleinman's test	TFCC tear TFCC stress test/fovea sign
STT degeneration Grind test	Intersection syndrome Tenderness at intersection	ECU instability ECU snap
First CMCJ degeneration Grind test	Kienbock's disease Lunate tenderness	Pisotriquetral degeneration Pisotriquetral grind test
De Quervain's tenosynovitis Finkelstein's test	Carpal tunnel syndrome Durkan/Phalen/Tinel test	Hook of hamate non-union Hamate tenderness
Preiser disease Scaphoid tenderness		Ulna nerve compression Tinel test

Assessment of carpal tunnel syndrome (median nerve compression), ulna nerve compression, and base of thumb degeneration are discussed in the chapter on examination of the hand.

Kirk Watson Test [13–15]

The Kirk Watson test is designed to assess for instability due to scapho-lunate ligament (SLL) deficiency. With radial deviation of the wrist, scaphoid flexion is initiated by the SLL and completed by pressure from the surrounding carpal bones. In the Kirk Watson test, external pressure is applied to the scaphoid as the wrist is moved into radial deviation. If the SLL is deficient, the scaphoid does not move until the other carpal bones are engaged, and then a clunk is felt. The original description included approaching the patient as if about to arm wrestle. The examiner's thumb is placed on the scaphoid tubercle, and index finger on the scapho-lunate ligament. The examiner's other hand moves the wrist into radial deviation. With an intact SLL, the scaphoid can be felt to flex against the thumb. In a positive test, there is no scaphoid movement initially then a clunk as the other carpal bones force the scaphoid into flexion. The patient may withdraw/resist due to apprehension.

The test should be repeated on the 'normal' side and the response compared as subsequent work by Kirk Watson found that 21% of asymptomatic people had a positive test unilaterally [14]. A gritty sensation when performing the test may represent scapho-lunate joint degeneration [15].

Finkelstein's Test [16–19]

Finkelstein's test is designed to stress the first extensor compartment, with provocation of pain suggesting a diagnosis of de Quervain's tenosynovitis. Often, the description is confused with Eichhoff's test, which has lower specificity [19].

To perform Finkelstein's test, place the patient's wrist on the edge of a table and ask them to actively ulnar deviate the wrist. The examiner then passively flexes the thumb into the palm to assess for provocation of pain.

In contrast, Eichhoff's test involved the patient grasping the thumb in their palm and the examiner passively moving the wrist into ulnar deviation.

Lichtman Test [20, 21]

Otherwise known as the midcarpal shift test, the Lichtman test is designed to assess for midcarpal instability. The wrist is held in pronation and 15 of ulnar deviation. Volar pressure is applied to the capitate by the examiners thumb as an axial load is applied to the metacarpals. A positive test provokes a painful clunk and reproduction of the patient's symptoms.

Reagan's Test [7, 22–24]

Also called the lunotriquetral ballotment test, this test assesses for lunotriquetral injury or instability. The examiner grips the lunate between thumb and index finger of one hand, and the triquetrum in the other. This allows the triquetrum

(and pisiform) to be pushed in a dorsal direction with respect to the lunate. A positive test provokes pain at the site of the lunotriquetral ligament.

Kleinman Shear Test [7, 23–25]
This test also assesses for lunotriquetral injury or instability, and is otherwise known as the shuck test. Dorsal translation force is applied to the pisiform and volar translation force to the lunate. This can be achieved with the examiner's thumb placed on the pisiform, and the examiner's index finger on the dorsum of the lunate, or with a thumb on each bone. Provocation of pain in the lunotriquetral joint is a positive test.

Piano Key Sign [6, 7]
To assess the stability of the distal radio-ulnar joint. With the wrist in pronation, pressure is applied to the ulnar head (pressing on a piano key). The test is positive if the ulna head returns to its resting position when the pressure removed (as a piano key does).

Ulna Compression Test [7, 26]
The ulna is compressed against the sigmoid notch. Provocation of pain is a positive test and suggests degeneration or instability of the distal radio-ulnar joint.

TFCC Stress Testing [1, 7, 11, 25–28]
There are a number of maneuvers described to stress the TFC (often and DRUJ), the simplest being to move the wrist into ulnar deviation and then flex and extend it.

The ulnocarpal stress test as described by Nakamura is sensitive for ulna sided wrist pain and will identify intra-articular pathology but is not specific. The wrist is placed in ulnar deviation, an axial load applied, and the wrist passively pronated and supinated.

Ulnar Foveal Sign [7, 29]
The ulnar soft spot is bordered by the ulnar styloid process dorsally, the flexor carpi ulnaris tendon volarly, the pisiform distally, and the volar surface of the ulnar head proximally. Tenderness and reproduction of symptoms on palpation here is considered a positive test and is sensitive for foveal disruptions and ulnotriquetral ligament pathology.

ECU Snap Test [7, 30–32]
The extensor carpi ulnaris tendon snapping over the ulnar head as the wrist is supinated in extension. This may be observed or palpated and is accompanied by pain and reproduction of symptoms.

Pisotriquetral Grind Test [7, 33, 34]
Thumb and index finger are used to compress the pisiform and move it in a radial and ulnar direction. Provocation of pain is considered a positive test suggesting pisotriquetral joint degeneration.

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8 Clinical Examination of the Hand and Fingers (Basic and Surface Anatomy) with Special Tests

Abhijeet Wakure, Akshay Omkumar, and Jose Tharayil

Introduction

Referral for hand injury forms a significant burden of any trauma service. In this chapter, we describe the surface anatomy of the hand. This chapter also deals with the history-taking and clinical examination of hand injuries as well as other common conditions affecting the hand. We also focus on some specific tests used to detect specific problems affecting the hand. It is a good practice to have a ready to use proforma for hand injuries so that important information is not missed out. This also helps with data collection and record keeping.

Surface Anatomy

Surface anatomy is of utmost importance in the hand due to presence of many structures packed into a tight space. It is important in the diagnosis and management of various pathologies. Knowledge of surface anatomy also helps in planning incisions for hand surgery.

Terminology to describe locations in the hand is different from other regions. "Radial" and "ulnar" refer to proximity to the respective bones. "Dorsal" is used to describe the posterior surface of the hand, and "palmar" or "volar" is used for the anterior surface. "Thenar" and "hypothenar" are used to describe the bulge of tissue on the radial and ulnar side of the palm, respectively.

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Palmar Surface of the Hand

On the palmar aspect of the fingers, skin creases correspond to the respective interphalangeal joints (IPJs). The crease at the base of the thumb corresponds to the first metacarpophalangeal joint (MPJ). However, the crease at the base of other fingers does not correspond to the respective MPJ but overlies the middle of the proximal phalanx. The distal palmar crease overlies the heads of the metacarpals of little, ring, and middle fingers. The proximal palmar crease lies over the head of metacarpal of index finger (Fig. 8.1).

The palmar surface consists of three regions, viz., thenar, mid-palmar, and hypothenar. Under the thenar and hypothenar eminences are the corresponding short muscles of the thumb and little finger. On the ulnar border of the hand, the fleshy mass felt is the abductor digiti minimi (ADM).

Web Spaces

There are four web spaces between the five fingers. The first web space is the widest, which permits independent movement of the first metacarpal in all planes, which is a unique feature of the human hand. The first dorsal interosseous muscle forms most of the bulk on the dorsal aspect of the first web space. The adductor pollicis is on the volar aspect of the same space.

Dorsal Surface of the Hand

On the dorsal aspect, the skin is thin and loose, which permits easy flexion of the fingers. This thin skin enables us to easily palpate the structures such as extensor tendons with the underlying metacarpals. The knuckles are seen on the dorsum and are mainly formed by the MPJs and the overlying extensor expansion.

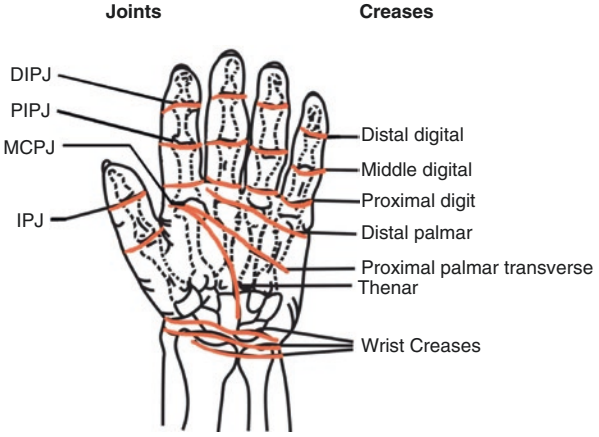

Fig. 8.1 Joints of the hand and their relationship to palmar creases

Surface Landmarks

On the volar aspect of wrist, the following structures can be felt from the radial to ulnar side—abductor pollicis longus (APL) tendon, radial artery, flexor carpi radialis (FCR) tendon, palmaris longus (PL) tendon, ulnar artery, and flexor carpi ulnaris (FCU) tendon. The median nerve is located between the tendons of FCR and PL. The ulnar nerve is located adjacent to the ulnar artery and radial to the FCU (Fig. 8.2).

At the base of the thenar eminence, a bony swelling can be felt, which is the tubercle of the trapezium. Proximal to this is the tubercle of the scaphoid, which is under the cover of the tendon of FCR. The distal wrist crease roughly overlies the area between the proximal part tubercle of the trapezium and distal part of tubercle of scaphoid. On the ulnar side in the hypothenar area, close to the distal wrist crease, the pisiform bone can be felt. If a line is dropped from the ulnar margin of the ring finger to the pisiform, the hook of hamate can be felt along this line just distal and radial to the pisiform.

Kaplan's Cardinal Line is drawn parallel to the proximal transverse palmar crease from the apex of the first web space (Fig. 8.2).

This corresponds to the level of the deep palmar arterial arch. The intersection point of a line drawn along the radial border of the middle finger with the Kaplan's

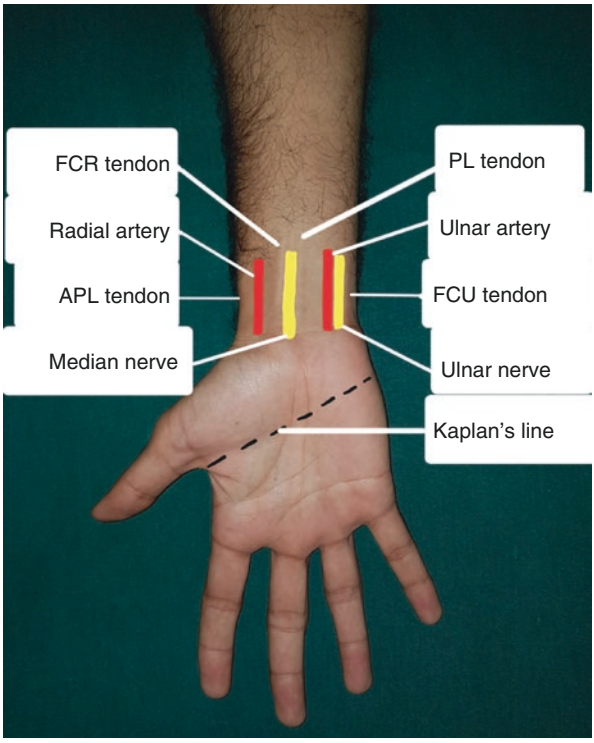

Fig. 8.2 Volar aspect of the hand showing surface anatomy of various structures (see text)

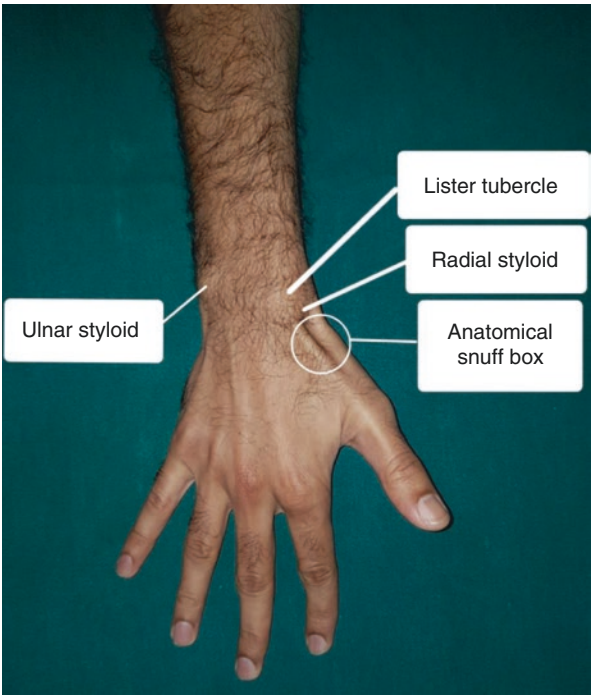

Fig. 8.3 Dorsum of the hand showing surface anatomy of various structures (see text)

line is the point of recurrent motor branch of median nerve that supplies the thenar muscles.

On the dorsal aspect of the wrist, the lateral-most tendon to be palpated is the APL. The anatomical snuffbox is bound anteriorly by Extensor Pollicis Brevis (EPB) together with APL and posteriorly by the Extensor Pollicis Longus (EPL). Within the snuff box the scaphoid (proximally) and trapezium (distally) can be palpated. The radial artery can also be palpated here. Radial styloid is felt on the lateral side of the distal end of the radius. The head of the ulna is felt as a round bony prominence, distal to which lies the ulnar styloid. Lister's tubercle is a bony prominence felt on the distal radius just radial to the EPL tendon (Fig. 8.3).

It serves as a pulley for the tendon of EPL and separates the second and third extensor compartments. Proximal to the base of the third metacarpal is the capitate. Lunate lies between the capitate and Lister's tubercle [1, 2].

History

Patient Demographics

The interview with the patient should always start with the basic information about the patient. Patient's name, age, sex, address, and level of education should be

noted. The information is important for record-keeping as well as to establish a rapport with the patient. In addition, this information is also useful to make important decisions regarding type of management. Occupation and hand dominance of the patient must be recorded since this has special relevance in management of conditions affecting the hand.

Present Complaints

Identifying the current problem of the patient, characterizing it further, and establishing chronology of the problem comes after demographics. Symptoms in the hand are generally classified as pain, paresthesia, numbness, weakness, swelling, discoloration, coldness, clumsiness, clicking, or snapping. Further characterization should be done by describing each symptom according to their location, intensity, frequency, radiation, and associated symptoms. Aggravating or relieving factors of each symptom should be noted.

In case of injury, it is important to note the mechanism, time, and place it happened. Details of mechanism can provide an insight into extent of the injuries and outcomes. For example, a small stab injury on the volar aspect of the hand may mean that a deeper flexor tendon or a nerve may be injured; injury to the flexor tendon with the finger flexed means that the distal end of the tendon is likely to have retracted; following replantation, a crush-amputation has a poorer outcome than a sharp cut amputation.

Past Medical History

Specific inquiry should be made into the pre-existing abnormality of the hand, including history of any previous injury or surgery to the hand.

Precise information about co-morbidities and the drugs that the patient is regularly taking can greatly influence diagnosis, treatment plan, as well as prognosis. Significant among these are diabetes and how well it is controlled; cardiac, pulmonary or renal disease; and rheumatologic, dermatological or neurological disease, and its possible effects on the hand. Medications affecting hemostasis (anti-platelets, anti-coagulants, etc.) or immunity (e.g., steroids) need to be considered because they might significantly affect the outcomes of surgery. Specific inquiry should be made about implanted devices like Pacemakers since electro-cautery may interfere in their functioning during the surgery.

It is vitally important to assess a patient with multiple co-morbidities in consultation with the medical specialist. Medications may need to be titrated, changed, or withheld. In a patient with multiple co-morbidities on many medications, overall risks versus benefits of surgical management for hand condition needs to be carefully assessed with the help of the medical specialist and needs to be discussed with the patient.

Allergies to specific drugs should be inquired. If present, they should be highlighted on the patient's case-sheets and medical records to avoid inadvertent administration of these medications.

Social History

It is important to note and quantify tobacco and/or alcohol use by the patient. This has implications on prognosis of certain surgeries like replantation and flap reconstructions. In addition, alcohol withdrawal can become a significant problem in the post-operative period if not already anticipated and acted upon. Any history of substance abuse must be noted as it has prognostic implications on the management plan.

Other Information

In case of trauma, time of the last meal should be noted as it influences administration of anesthesia. Inquiry about immunization against tetanus for general injuries and rabies for animal bites is necessary.

Examination of Hand

Introduction

Hand is a bilateral organ. Hence, normal hand should always be compared to the affected hand at every stage of examination of the hand. A lot of information can be gained by doing a close observation of the hand at rest and during spontaneous movements and comparing to the other hand. Good history taking combined with detailed examination is essential for the diagnosis of complex problems of the hand.

Inspection

In case of trauma, assessment should be made of the extent of injury, adequacy of skin cover, and loss of structures. Note should be made of lacerations and/or deep abrasions of the skin and the exposed injured underlying structures. It is important for the senior reconstructive surgeon to make this initial assessment as the plan of reconstruction can be made at this time [3].

Observation of pink color of the hand will indicate adequate vascularity of the hand. Any difference in color should be corroborated with other findings. Localized or diffuse swellings should be noted in detail. Any fusiform swellings or deformities of the joints such as angulations or contractures of the fingers must be looked out for. Scars and nail abnormalities should be noted. Dupuytren's contracture can be

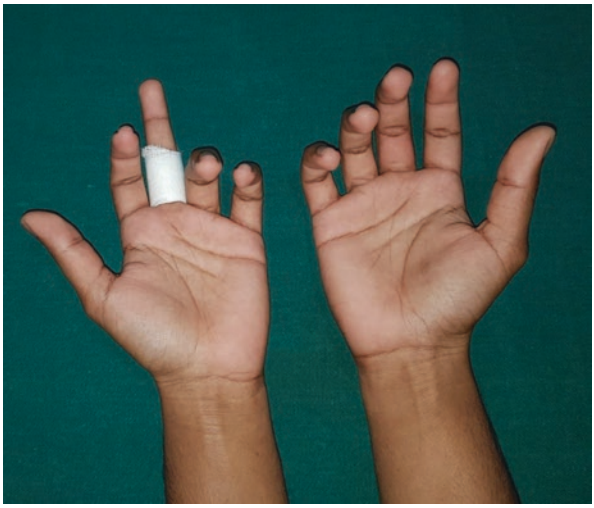

Fig. 8.4 Accidental stab injury to the left middle finger causing complete division of both flexor tendons has resulted in loss of normal cascade in the left hand. The right hand shows normal cascade of fingers

diagnosed when multiple palmar nodules are seen along with flexion contracture of PIP joints more commonly on the ulnar side of the hand.

The normal cascade of the fingers at rest (with wrist in cock-up position) consists of fingers in gradually increasing flexion of IPJs and MPJs from the ulnar to the radial side [4]. Abnormal cascade of the fingers would indicate some anatomical deformity with or without functional deficiency. Divided flexor tendon is diagnosed with abnormal extended position of the affected finger/s (Fig. 8.4).

Mallet finger deformity (flexion of the DIP joint) occurs due to rupture of extensor tendon at distal interphalangeal joint with or without associated swan neck deformity (hyperextension of the PIP joint). Boutonniere deformity (flexion of the PIP joint and hyperextension of the DIP joint) is a result of attenuation or rupture of the central slip of extensor apparatus at the PIP joint (Fig. 8.5).

Thenar and hypothenar eminences should be observed for any wasting of short muscles, which is indicative of median or ulnar nerve palsy. On the dorsum of the hand, intermetacarpal spaces should be observed for wasting of interrosseous muscles seen in ulnar nerve palsy or in old crush injuries. Ulnar palsy can also be suspected in case of characteristic clawing of the ulnar fingers. Trophic changes may be present in the radial fingers in case of median nerve palsy. Wrist drop associated with the inability to extend the metacarpophalangeal joints is indicative of radial nerve palsy. Intrinsic minus hand (seen in combined distal median and ulnar nerve palsy) can be diagnosed in cases with hyperextension at metacarpophalangeal joints and flexion of interphalangeal joints.

Intrinsic plus hand is seen in any condition characterized by spastic intrinsic and weak extrinsics (long flexor and extensor) muscles. This results in flexion of MCP joint and extension of IP joints. The most common conditions causing this may be trauma, which may be direct or indirect, arthritis, traumatic brain injury, or central neurological condition.

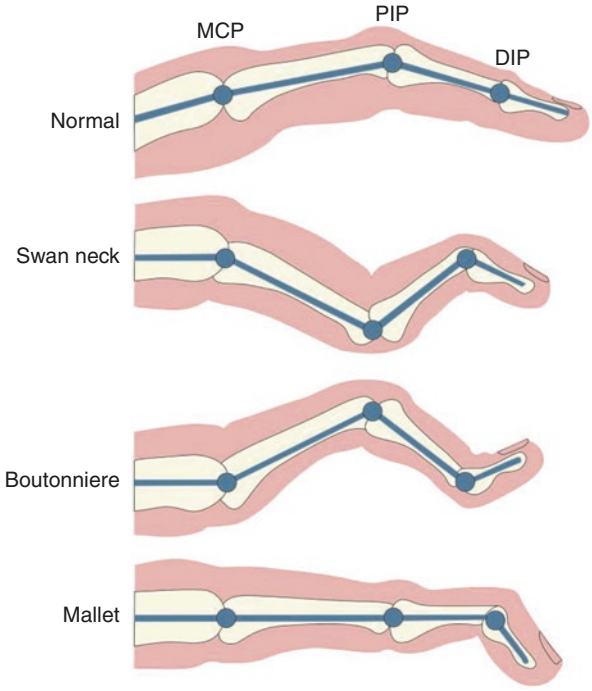

Fig. 8.5 Representative diagram showing position of the joints of the finger in various deformities

Palpation

The very first finding to be noted is local rise of temperature and tenderness compared to the other hand. Based on this, other findings will be prioritized. Temperature is best appreciated by running the dorsum of the examiners hand along the patient's hand from distal to proximal. Kanavel's cardinal signs are seen in patients with flexor tenosynovitis, which can cause loss of function of the affected finger and should be treated promptly [5]. These signs include—slight flexion of the finger, fusiform swelling over flexor tendon, tenderness over flexor tendon, and pain on passive extension of the finger.

Swellings should be examined in detail for size, site, extent, consistency, and tenderness, adherence to adjacent structures and relation to movements.

Blanching on pressure and the speed of capillary refilling should be observed in the finger tip or nail bed. A capillary refill of 2–3 s indicates normal vascularity. Radial pulse is examined by palpating the radial artery against the radius. Allen's test must be carried out to know the integrity of the radial or ulnar arteries as well as communications between the superficial and deep palmar arch [6]. The patient is asked to clench his fist and both the radial and ulnar arteries are occluded by digital pressure. The patient is then asked to clench and unclench his fist rapidly to achieve exsanguination. The radial artery is released and "pinking up" of the tips of the

fingers (especially the thumb) is noted. If it is delayed by more than 6 s, then it indicates that the radial artery is occluded. Similar procedure is repeated for the ulnar artery.

Movements

Hand is an organ with delicate movements and intricate and complex functions. Hence, in addition to detailed examination of active and passive range of motion of joints, other functions such as opposition, pinch, grip, etc., should also be tested.

Examination of the Joints

Active and passive range of motion of each joint of the hand should be examined using a goniometer.

Table 8.1 shows the range of motion of each joint of the hand [7]. This should be noted for each patient at the initial examination and at subsequent examinations to have an objective assessment of improvement following any intervention. However, one should remember that the functional range of motion necessary to obtain adequate function of the hand may be less than the full active range of motion [8].

Laxity or pain on stress testing of the collateral ligaments of the joints of the fingers indicates injury to these structures [9]. More than 20° of varus or valgus deformity of the PIP joints on stress testing indicates disruption of the collateral ligaments. The ulnar collateral ligament of the thumb is tested by flexing the first MCP joint in 30° and stabilizing the first metacarpal and applying valgus stress to the proximal phalanx. If the joint opens up by more than 30°, it indicates complete disruption while opening by less than 30° indicates partial disruption of the ulnar collateral ligament.

Tenderness on the volar aspect of the joint and pain on hyperextension of the joint indicate injury to the volar plate of the joint. Localized swellings of the joint should be noted and characterized as previously mentioned. An obvious deformity, tenderness, and loss of movement may indicate an underlying fracture or dislocation.

Table 8.1 Normal range of motion of thumb and fingers

Digit	Joint	Range of motion
Thumb	MCPJ	0° to 56°
	IPJ	−5° to 73°
Fingers	MCPJ	0° to 100°
	PIPJ	0° to 105°
	DIPJ	0° to 85°

Examination of the Musculotendinous Units [10, 11]

Finger Extensors and Flexors

Each and every musculotendinous unit should be examined precisely. Flexor digitorum profundus (FDP) is examined by holding PIP joint in extension and asking the patient to flex the tip of the finger (see Fig. 8.6).

Flexor digitorum sublimis (FDS) is examined by holding all other fingers in full extension and asking the patient to flex the free finger (see Fig. 8.7).

Flexor Pollicis Longus (FPL) and Extensor Pollicis Longus (EPL) are assessed by testing the flexion and extension, respectively, of the IP joint of the thumb against resistance while stabilizing the proximal phalanx of the thumb.

Trigger finger is due to impediment to the smooth motion of flexor tendon at the level of A1 pulley. De Quervain's tenosynovitis (of APL/EPB) is tested by eliciting pain on ulnar deviation of the wrist while the thumb is in the palm with finger wrapped around it.

Wrist Flexors

FCR tendon can be tested by palpating the tendon while the wrist is flexed against resistance in a radial-deviated position. FCU tendon can be tested by palpating the

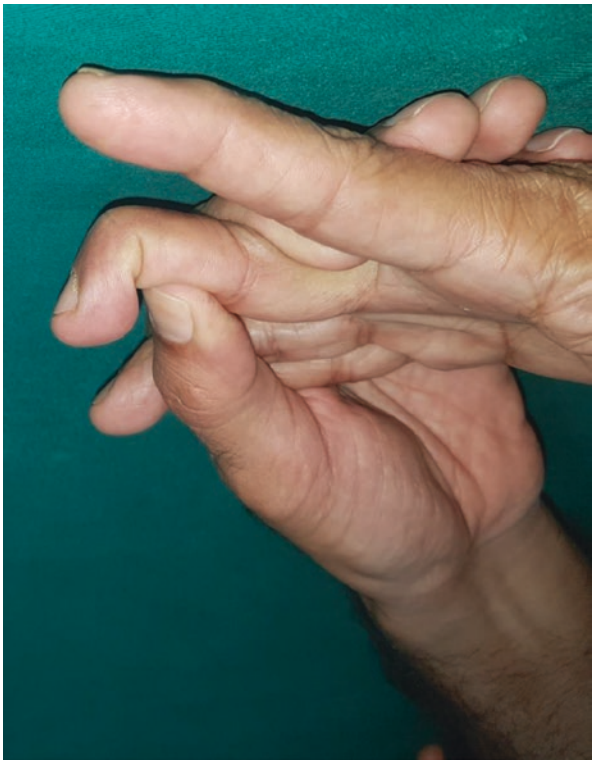

Fig. 8.6 Testing for FDS spans 2 finger joints

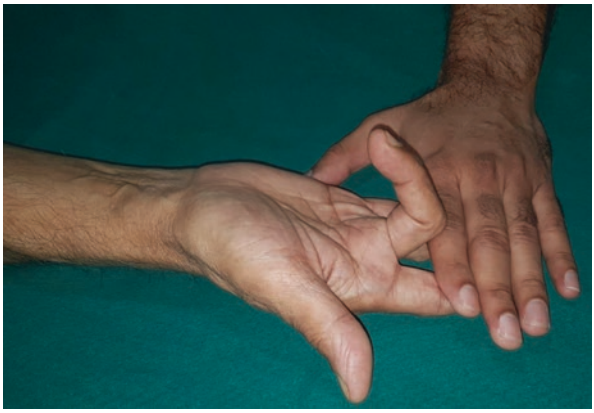

Fig. 8.7 Testing for FDS spans 2 finger joints

tendon while the wrist is flexed against resistance in an ulnar-deviated position. PL becomes taut when the wrist is flexed with the tips of the thumb and little fingers touching each other. Testing the PL is additionally important while planning for a tendon graft.

Wrist Extensors

Extensor Digitorum Communis (EDC) is tested by keeping the IP joints flexed (to nullify the intrinsic muscles) and extending the MCP joints. Extensor Indicis Proprius (EIP) and Extensor Digiti Minimi (EDM) are examined by asking the patient to extend the index and little finger, respectively, while holding the fist tightly. In case of division of EDC, only the index and little fingers can be extended and shows a Horn sign with middle and ring fingers remaining in flexed position. Extensor Carpi Radialis Longus (ECRL), Extensor Carpi Radialis Brevis (ECRB), and Extensor Carpi Ulnaris (ECU) are palpated while fully extending the wrist against resistance.

Examination of the Nerves [10, 11]

Three nerves supply the hand. Deficits in nerve function can be clearly evaluated by careful examination.

Motor Testing

Ulnar Nerve

Typical claw hand due to low ulnar nerve injury consists of hyperextension of MP joints and flexion deformity of IP joints resulting from the paralysis of the interrossei muscles. The clawing is less prominent in the index and middle finger because of the action of first and second lumbrical muscles supplied by the median nerve. FCU action is preserved in low ulnar palsy (see Fig. 8.8).

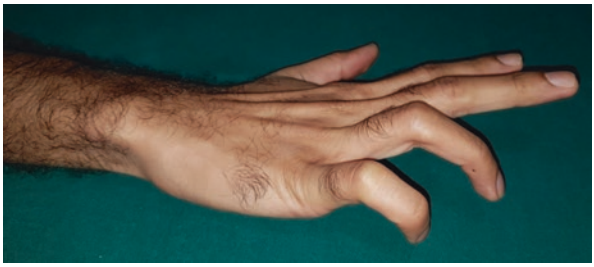

Fig. 8.8 Clawing of fingers seen in low ulnar nerve palsy

Fig. 8.9 Froment's sign

In high ulnar palsy, clawing is less prominent due to paralysis ulnar part of FDP muscles that reduces the flexion deformity in the IP joints of the ring and little fingers.

Adductor pollicis is tested by holding a card in the first web space tightly while the examiner tries to pull it away. If the muscle is paralyzed, the patient tries to flex the thumb to hold it. This is Froment's sign (see Fig. 8.9).

For testing the interrossei muscles, the middle finger is taken as the central point. Palmar interrossei adduct other fingers to the middle finger and dorsal interrossei abduct finger away from middle finger. These movements can be tested against resistance to assess ulnar nerve palsy.

Median Nerve

Palsy of Abductor Pollicis Brevis results in inability to raise the thumb perpendicularly to the palm. Further, the patient cannot oppose the thumb against the little finger due to palsy of Opponens Pollicis. A palsy of these two together will result in "ape thumb" deformity where the thumb lies supinated and in the same plane of the other fingers and cannot be brought into opposition.

Ochsner's test is when the patient is asked to clasp both palms. In high median nerve palsy, the IP joints of index finger remains extended—also known as pointing index. The connection of the FDP belly of the middle finger with that of the ulnar two fingers (supplied by ulnar nerve) enables flexion of the middle finger.

Ulnar deviation of the wrist is also seen on attempted flexion due paralysis of FCR.

Sensory Testing

Sensations in the hand can be objectively assessed using Semmes–Weinstein monofilament test, static two-point discrimination and dynamic two-point discrimination [12]. These tests are also useful for assessing worsening or improvement in the sensory function of the nerves. The required parameters are listed in Table 8.2.

Figure 8.10 shows the sensory distribution of the three nerves in the hand.

Ulnar Nerve

Absence of sensation in ulnar one and half digits on palmar and dorsal aspect confirms high ulnar palsy. Compression neuropathy involving ulnar nerve in the cubital

Table 8.2 Objective tests for sensory function of the hand

Static two-point discrimination scoring	
Interpretation	**Distance**
Normal	1–5 mm
Fair	6–10 mm
Poor	11–15 mm
Protective sensation only	One point perceived
Anesthetic	No points perceived
Semmes-Weinstein monofilament test	
Interpretation	**Filament color (size)**
Normal light touch	Green (1.65–2.83)
Diminished light touch	Blue (3.22–3.61)
Diminished protective sensation	Purple (3.84–4.31)
Loss of protective sensation	Red (4.56–6.65)

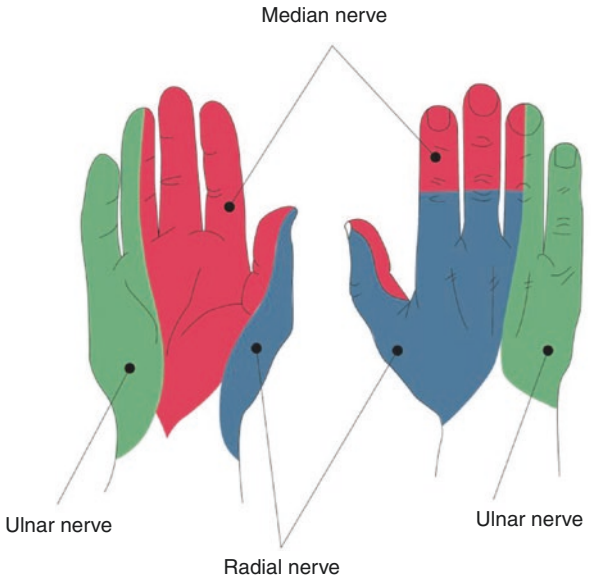

Fig. 8.10 Sensory distribution of the three nerves of the hand

tunnel can lead to paresthesias, tingling, and numbness in this region. Presence of sensation over the dorsum of these fingers indicates low ulnar palsy (due to sparing of the dorsal sensory branch). Similarly, compression neuropathy in the Guyon's canal leads to sparing of the dorsum.

Median Nerve

In median nerve palsy, absence of sensation on the palmar aspect in radial three and half fingers is characteristic.

Phalen's test: Keeping both wrists in full flexion by pressing the dorsum of both hands against each other for 1 min produces tingling and numbness in the median nerve distribution (Fig. 8.11).

Assessment (SODA), Arthritis Hand Function Test, Jebsen Hand Function Test, and Michigan Hand Outcomes Questionnaire [11].

Reverse Phalen's test: Keeping both wrists in full extension by pressing the palm of both hands against each other for 1 min produces tingling and numbness in the

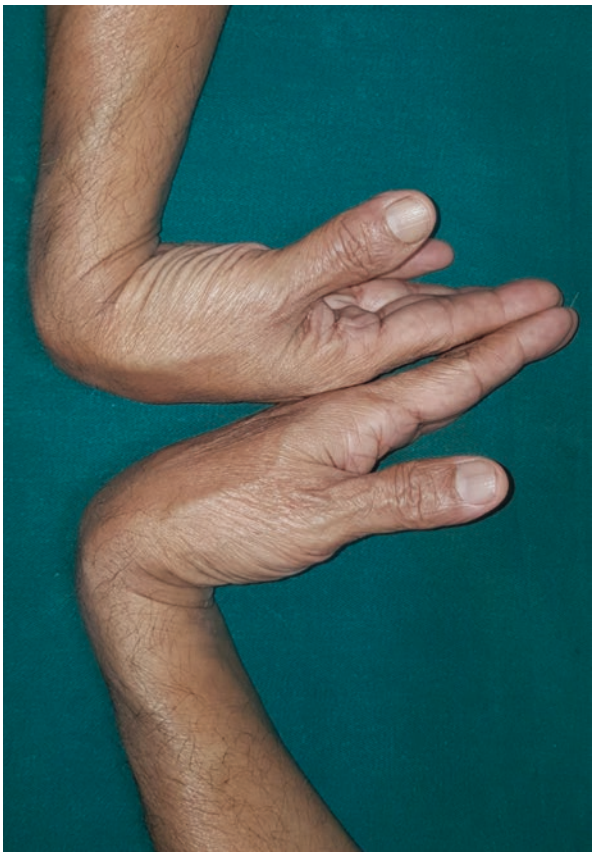

Fig. 8.11 Phalen's test

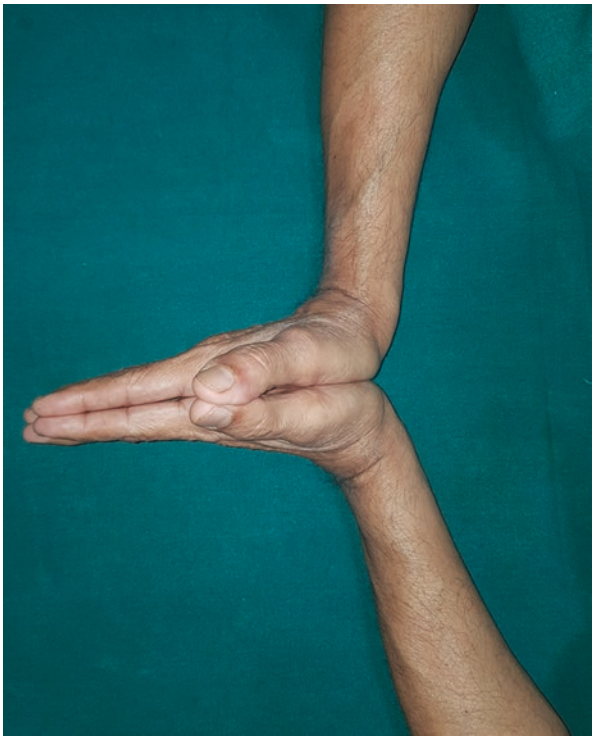

Fig. 8.12 Reverse Phalen's test

median nerve distribution. Both the tests increase the pressure in the carpal tunnel leading to these symptoms in early carpal tunnel syndrome (Fig. 8.12).

Radial Nerve

Lack of sensation on the dorsum of radial three and half digits up to the level of DIP joints confirms the high radial nerve palsy.

In posterior interosseous nerve palsy, the sensation may be preserved due to supply from superficial sensory branch of radial nerve. Wartenberg syndrome is due to compression neuropathy of the superficial radial nerve leading to numbness, tingling, or pain over the back of thumb and index finger.

Functional Tests

Functional tests of the hand can evaluate any deficiency in the intricate and complex functions of hand. They are useful for assessment of the performance status of the hand and to assess how much an intervention has helped the patient. One such score is Disability of Arm, Shoulder, and Hand Questionnaire (DASH). The score can be used to assess hand performance as a patient reported test. It has been validated for assessing utility of operative interventions as well [13]. Some other useful functional tests are Sequential Occupational Dexterity.

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The Hand and Fingers

9

Shabih Siddiqui and Muzamil Baba

Dupuytren's Disease

This condition is mostly seen in white northern Europeans and no medical student can escape without being asked about who Dupuytren was.

Baron Dupuytren accurately described the condition in a dissected cadaver hand and presented a detailed account in a paper in 1831. Sir Astley Cooper had earlier described the condition in 1822.

The disease involves a benign fibroproliferative disorder with a very unclear etiology. However, risk factors include tobacco and alcohol use, HIV, diabetes, epilepsy, chronic lung diseases, and tuberculosis. Sporadic cases are more common although an autosomal dominant inheritance has been recently suspected. The ring and little finger and ulnar half of the palm are most commonly involved (Fig. 9.1).

Flexion contracture of the fingers, web space contractures, hard and painful nodules in the palm, and gradual disuse of the hand and fingers develop slowly over the years. There is extensive fibromatosis of the palmar aponeurosis of the hand. Normal bands become diseased cords.

Dupuytren diathesis involves knuckle pads on the dorsum of the fingers, plantar fibromatosis of the sole of foot (Ledderhose disease) and contractures of the penis, which is also called Peyronie's disease.

The main anatomic structures involved are the longitudinal pretendinous bands, the spiral bands, and the natatory ligaments. The lateral digital sheaths, Grayson's ligaments, and typically sparing of Cleland's ligaments.

As these contractures increase fine motor skills, grasp and pinch are affected. Initially, the contractures can be passively corrected but as time goes on these contractures become fixed. Later, involvement of the muscle and fascia of abductor digiti minimi can contract the little finger further.

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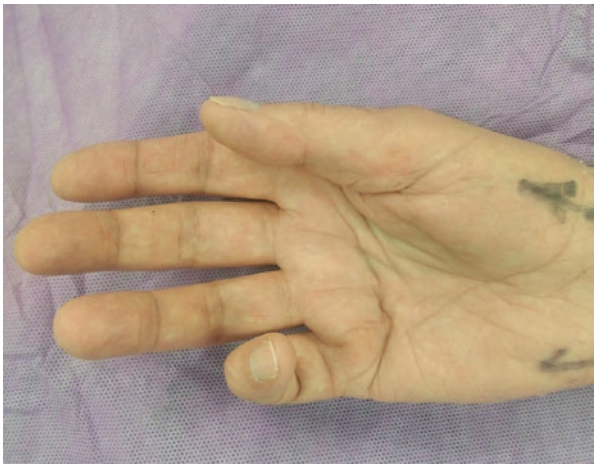

Fig. 9.1 Clinical picture of patient with Dupuytren's contracture involving right hand little finger and thumb. (Clinical photo S. Siddiqui/M. Baba)

Meyerding first described the histologic features in the 1940s. Since then, enormous work has been done by several workers explaining the histology, and the three main stages have been identified.

The initial stage, which is the proliferative stage, involves increase in the cellularity of the palmar fascia.

The second is the involutional stage in which there is dense myofibroblasts network and less cellular collagen bundles. In the last or residual stage, most myofibroblasts have disappeared and few fibroblasts remain within dense collagen cord.

Dupuytren's disease is more commonly seen in Northern Europe. Non-Caucasians are rarely affected. It is ten times more common in men than women. Early treatment may include splinting, steroid use, and physical therapy. Inevitably, surgical release is needed. Surgical principles are removal or debulking of contracted palmar fascia, preserving the neurovascular structures, correction of joint deformities, and aggressive physical therapy. Surgery is not curative but can salvage hand function significantly.

A tabletop (Hueston's test) in which the hand is held flat on the table and if the palm cannot be kept flat is an indication for surgery. If there is an MCP contracture of more than 30° or any degree of PIP contracture, then surgery is indicated.

Operative procedures include fasciotomy, limited fasciotomy, local fasciotomy, and total radical fasciectomy.

Fasciotomy can be considered for a single contracted palmar band only in experienced hands, as there is risk of damage to neurovascular structures. Total fasciectomy is rarely done due to high morbidity associated with it. Dermo fasciectomy with skin grafting is also done. This has the best chance of preventing recurrence.

Numerous incisions have been used. Skoog and McCash advocate transverse incisions as skin infection and hematoma chances are minimal. Zigzag incisions

and combinations of longitudinal and oblique incisions have been used. Patients are counseled regarding infection, damage to neurovascular structures, and 20–30% chance of recurrence.

Early postoperative flare reactions have been described, which are more common in women and are managed with a course of NSAIDS or short term oral steroids.

Trigger Finger

This condition is commonly seen in women who are older than 50 years.

Trigger finger—or stenosing tenosynovitis—is a common problem that interferes with the normal function of the fingers or thumbs. This usually affects the middle or ring finger.

It occurs when any digit of the hand gets stuck in a bent or flexed position and causes locking or catching. Overcoming this resistance results in the affected digit snapping straight. If the condition worsens, the finger may need to be forcibly straightened or may remain locked in a bent position.

Green has classified the condition into four stages with Stage I presenting as pain and tenderness around the A1 pulley, Stage II catching of the digit intermittently, Stage III causing locking, which is passively correctable and Stage IV with a fixed locked digit.

Causes and Risk Factors

The tendon sheath passes through a canal, which is the A1 pulley at the level of distal crease of the palm.

Trigger finger arises when a nodule, or knot, develops in one of the tendons at this site. Inflammation or scarring is believed to be responsible and is usually the result of repetitive use of the tendon in repeated gripping actions, or an inflammatory condition such as rheumatoid arthritis. There is disparity between the tendon sheath size and the canal through which it passes. Hence, the problem is mainly mechanical.

It is more likely to arise in people with medical conditions such as gout and diabetes. However, sometimes there is no explanation why a nodule has developed.

Diagnosis depends entirely on the history and clinical examination. Concomitant trigger finger with carpal tunnel syndrome may be present in 40–60% of cases.

Treatment

As the problem is mainly mechanical to overcome trigger finger, any obstruction to normal tendon movement needs to be removed. Specific treatment depends on how severe the symptoms are and how it is affecting an individual.

Mild symptoms may only need the hand to be rested and to avoid repetitive use of the finger.

In more serious cases, anti-inflammatory drugs or a steroid injection into the affected tendon sheath should solve the problem.

If these measures are not successful, surgical release, which means releasing the A1 pulley to remove the problematic narrowing, will be recommended. This is usually done as a day case under local anesthesia.

One recent study in the *Journal of Hand Surgery* suggests that the most cost-effective treatment is two trials of corticosteroid injection, followed by open release of the first annular pulley. Choosing surgery immediately is the most expensive option and is often not necessary for resolution of symptoms. Active and passive physiotherapy will be needed subsequently.

Trigger Thumb in Children (Pediatric Trigger Thumb)

The problem of triggering in children occurs usually in the thumb. It may be noticed soon after birth. Hence, it is also sometimes called congenital trigger thumb but a better diagnosis would be pediatric trigger thumb. In adults, it may accompany rheumatoid arthritis.

Children with trigger thumb rarely complain of pain. They are usually brought in for evaluation when aged 1–4 years, when the parent first notices a flexed posture of the thumb IP joint. These children often demonstrate bilateral fixed flexion contractures of the thumb by the time they present to the physician. The diagnosis is made instantaneously, as the appearance is classical. One is unable to passively straighten the fixed contracted thumb. A nodule can be felt at the stenosed site.

The cause of trigger thumb is the same as in the finger. There is mechanical obstruction of the tendon of flexor pollicis longus through tight A1 pulley. Apart from being congenital, there may be an element of unnoticed trauma. Treatment is usually surgical but one must not always rush into surgery as in some studies there is spontaneous recovery in 30% cases. Recovery is unlikely if the child is more than 2 years.

In children, the procedure is performed under a general anesthetic under a tourniquet. A transverse or curvilinear incision centered over the MCP joint of the thumb was made on the volar aspect. This is centered over the nodule. The A1 pulley is cleanly divided longitudinally. The tendon of the FPL is then delivered in the wound with a curved hemostat (Fig. 9.2). The edges of the pulley may be excised. Full excursion of the thumb is then observed to make sure no secondary adhesions are present. It is obvious to say that the neurovascular structures are protected. Skin is closed with absorbable sutures and thumb movements are encouraged as soon as possible.

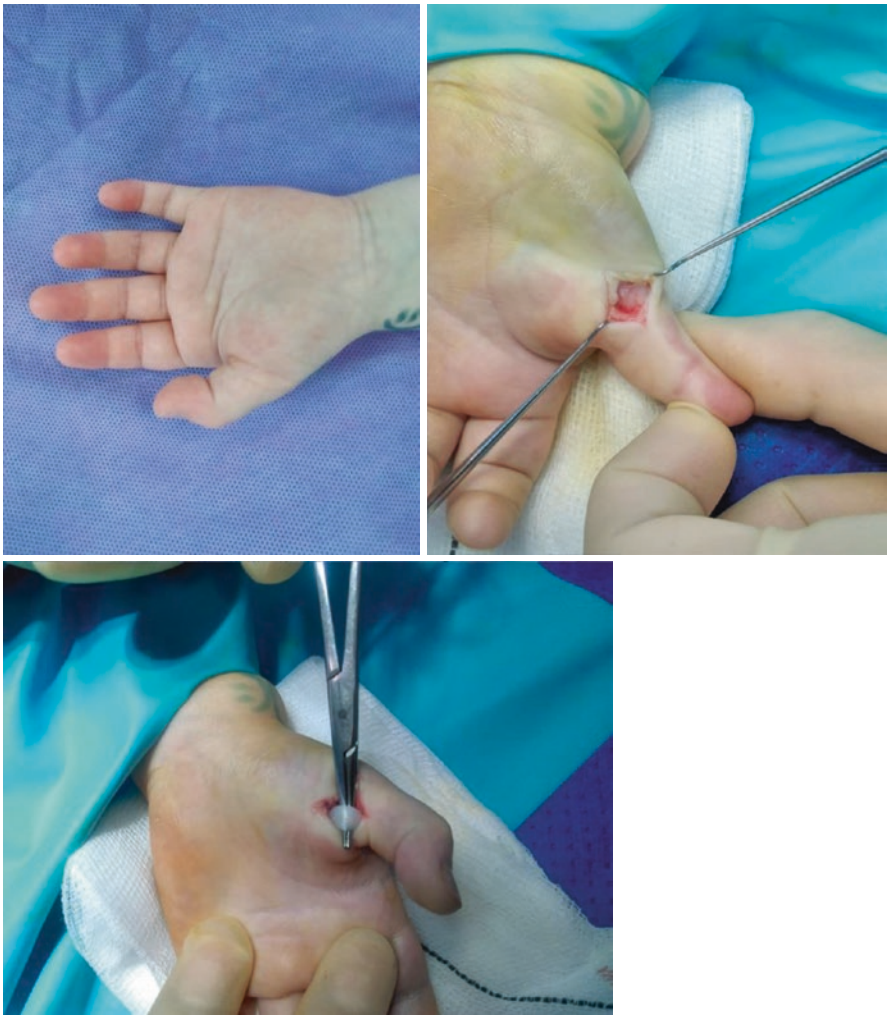

Fig. 9.2 Trigger Thumb deformity in a 3-year old child with surgical release of A1 pulley and complete correction of the deformity

The Thumb in Adults

S. Siddiqui and Muzamil Baba

Osteoarthritis of Thumb (CMC J)

This condition is seen common in those who have done a heavy physical job but can also present in the general population especially females. The carpometacarpal joint

is involved: the trapezium being commonly involved and sometimes the trapezium trapezoid complex is involved as a whole.

Previous trauma can also cause secondary OA at this joint. This is also thought to be caused by attenuation of the volar beak ligament (Pellegrini).

Patients with rheumatoid disease commonly have some degree of involvement of this joint. There may in addition be osteoarthritis of the proximal and distal interphalangeal joints with Herbeden and Bouchard's nodes. Patients can present with concomitant carpal tunnel syndrome in 50% of the cases.

Physical examination reveals pain, tenderness, and swelling at the joint site. There is reduction of joint movement. The grind and circumduction tests reveal sharp pain. Crepitus is common. Painful instability of the carpometacarpal joint is frequently seen. The scaphoradial joint may frequently be involved.

Early osteoarthritis of the thumb is treated conservatively by non-steroidal agents and splinting. Physical therapy can sometimes produce dramatic improvement. Steroid injections are frequently given in outpatients followed by hand therapy. Surgery is reserved for those cases that fail medical management.

Fusion of CMC joint is a viable option for younger patients who do not want to risk loss of strength. Relief of pain is good but fine motor function may be compromised.

Arthroscopic hemitrapeziectomy is a recent advance that can be used for early stage of the disease.

Joint Arthroplasty

Plastic or metal prosthesis is used to replace the worn-out joints. This is usually done to elderly or rheumatoid patients. However, the most commonly performed procedure continues to be trapezium excision with ligament reconstruction and tendon interposition.

1. Deformity of thumb before surgery.
2. Small incision at base of thumb to expose nodule and A1 pulley.
3. A1 pulley completely released and long flexor tendon of thumb exposed and free completely.

The Thumb

10

Shahrier Fazal Sarker and Wasim S. Khan

Pediatric Trigger Thumb

Trigger thumb is more common in the pediatric population. Although commonly called congenital trigger thumb, it is more of a postnatal problem as studies have shown almost no incidence of a trigger thumb in newborns.

Children usually present with a flexion deformity at the interphalangeal joint (IPJ) of the thumb. The Sugimoto classification divided it into four stages—stage 1: palpable nodule, stage 2: active triggering, stage 3: passive triggering and stage 4: fixed flexion deformity. Most patients present at stage 2 and later as children do not usually complain of a nodule [1].

The pathophysiology is the same as in adults and other trigger fingers—a thickening of the flexor tendon (Flexor Pollicis Longus (FPL) in the case of trigger thumb), a thickening of the tendon sheath or both. In all cases, a nodule at the A1 pulley can be felt.

In about 30% of cases, the triggering resolves spontaneously and many studies have shown that it is reasonable to continue with observation until the child is three, unless there is painful triggering or a fixed flexion deformity of the IPJ.

Management

Initial management consists of observation, active and passive motion exercises, and splinting at night.

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In my practice, surgical release is performed under a general anesthetic using an above elbow tourniquet. The surgeons wear loupes to allow better visualization. The child is positioned safely on the operating table with an arm board. After preparing the surgical field with chlorhexidine and an arm drape, a V-shaped incision is made along the volar surface of the A1 pulley. The neurovascular bundles, in particular the radial digital nerve, is found and protected. Soft tissue dissection using tenotomy scissors is done until the A1 pulley is clearly seen. A longitudinal incision is made to along the whole length of the A1 pulley. Thereafter, the FPL is mobilized to ensure good excursion. The wound is irrigated with normal saline. The tourniquet is deflated, and hemostasis performed with a bipolar diathermy. The skin is then closed with an absorbable suture such as Monocryl 3/0. The wound is covered, and a bulky dressing is applied. The patient is seen in 7–10 days and the dressings are removed and child is allowed to mobilize as able.

Base of Thumb Fractures

Bennett's and Rolando fractures are intra-articular fractures of the base of the thumb metacarpal.

Bennett's Fractures

Bennett's fractures (Fig. 10.1) are classically described as a two-part fracture of the base of the first metacarpal. It is essentially a fracture dislocation of the first carpometacarpal joint. The beak ligament, which originates from the trapezium and inserts into the ulnar and volar edge of the first metacarpal, holds the constant fragment in place and the rest of the metacarpal subluxes radially, proximally, and dorsally.

It is the most common fracture of the base of the first metacarpal and occurs with axial loading and partial flexion of the thumb. The deforming forces on the metacarpal shaft that subluxes are the pull of the Adductor Pollicis (adduction and supination), Abductor Pollicus Longus (radial, dorsal, and proximal displacement), and the Extensor Pollicis Longus (radial, dorsal, and proximal displacement). It is key to understand the deforming forces, as reversing these is the cornerstone of treating these fractures.

Bennett's fractures need to be anatomically reduced as best as possible, as any incongruity will predispose to first CMCJ arthritis. Studies have shown that the first CMCJ compressive forces are 12 times the force being produced. This, together with increased mobility of the first CMCJ and the constant daily use, increases the stress on the joint [1, 2].

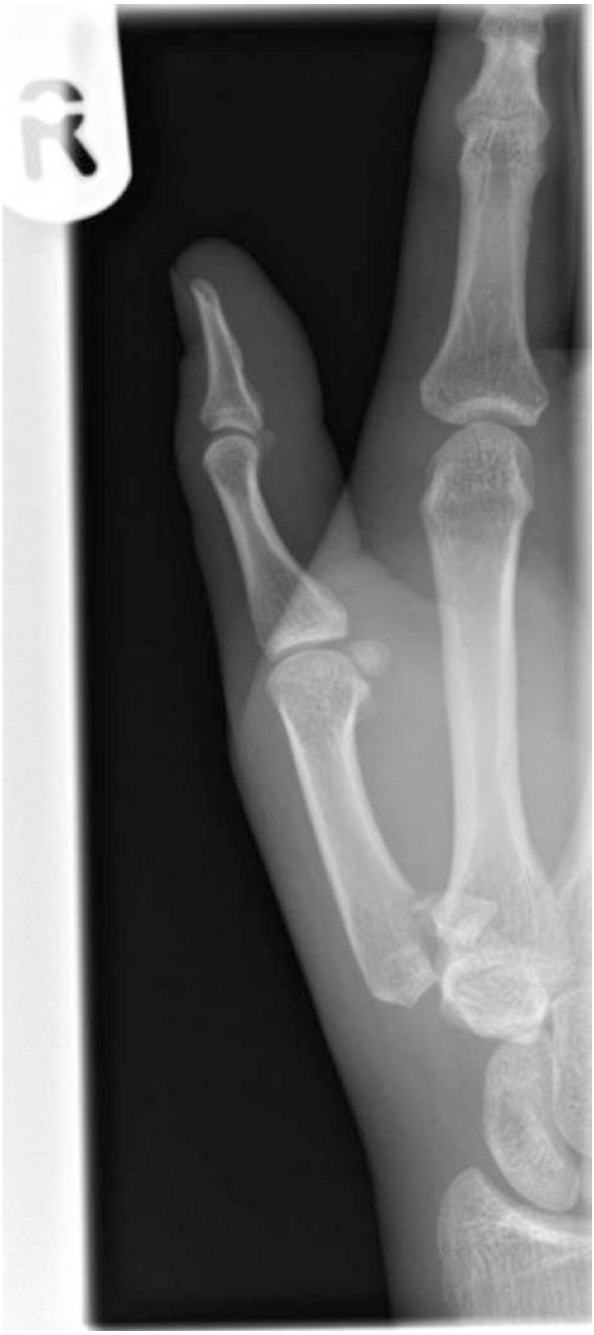

Fig. 10.1 Lateral view showing Bennet's fractures is due to pull of the adductor pollucis. (Image courtesy of S. Sarker)

Management

Non-surgical management of these fractures is very rare. Only in cases where the joint surface is completely restored that this can be considered, but the patient needs to have a good plaster and regular X-rays.

Reduction is performed by longitudinal traction, pronation of the metacarpal, and pressure at the metacarpal base. I would recommend positioning your index finger in the first web space, and then to grip the thumb and pronate it whilst applying axial traction. Simultaneously, your thumb should be pushing the base of the first metacarpal ulnarly towards the second metacarpal. When the cast is applied, these forces need to be maintained, which can be quite difficult that is why non-surgical management tends to be suboptimal [3].

Surgical management consists of the same manipulation technique and then either using K-wires or a plate (Figs. 10.2 and 10.3).

This can be done under either a general anesthesia or a brachial plexus block. A tourniquet is used, and the operation is performed under fluoroscopic guidance. The patient is positioned supine with an arm board. If the fracture can be reduced closed with a congruent joint surface, I use 2 × 1.6 mm K-wires. I make a small 1 cm dorsal longitudinal incision over the base of the thumb, and identify and protect the cutaneous branch of the radial nerve and the extensor tendons. One wire is driven from the shaft of the first metacarpal into the trapezium, and the other wire through the shaft of the first metacarpal through the constant fragment and into the base of the second metacarpal. Some surgeons transfix the shaft of the first metacarpal to the shaft of the second metacarpal with a transverse K-wire but I tend to avoid that, as there is a chance of injuring the ulnar neurovascular bundle. The K-wires are then bent, cut, and buried under the skin. The skin is closed with Ethilon 4/0. A below elbow thumb spica cast is then applied for 4 weeks and then converted to a splint under the care of the hand therapists. The sutures are removed in 2 weeks. The K-wires can then be removed under a local anesthetic in 6 weeks. Some surgeons prefer to leave the K-wires outside the skin, and these should be removed in 4 weeks and a cast reapplied. The advantage of burying the K-wires is that passive movement exercises can be started earlier.

If the fracture is very unstable and the joint surface is not congruent under closed reduction, then an open reduction and internal fixation should be considered. The incision mentioned earlier is extended and a similar approach is undertaken. The fracture is then reduced and held with K-wires. The joint surface can be directly visualized as well as confirmed fluoroscopically. Plate and screws are then used. I would recommend using any plating system the surgeon is familiar with or is available. Either a locking or non-locking system can be used depending on surgeon's choice and patient's factors. I prefer using a T-plate with non-locking screws so that compression at the fracture site can be achieved.

Rolando Fractures

Rolando fractures (Fig. 10.4) are comminuted fractures of the base of the first metacarpal and are classically described as T-shaped or Y-shaped.

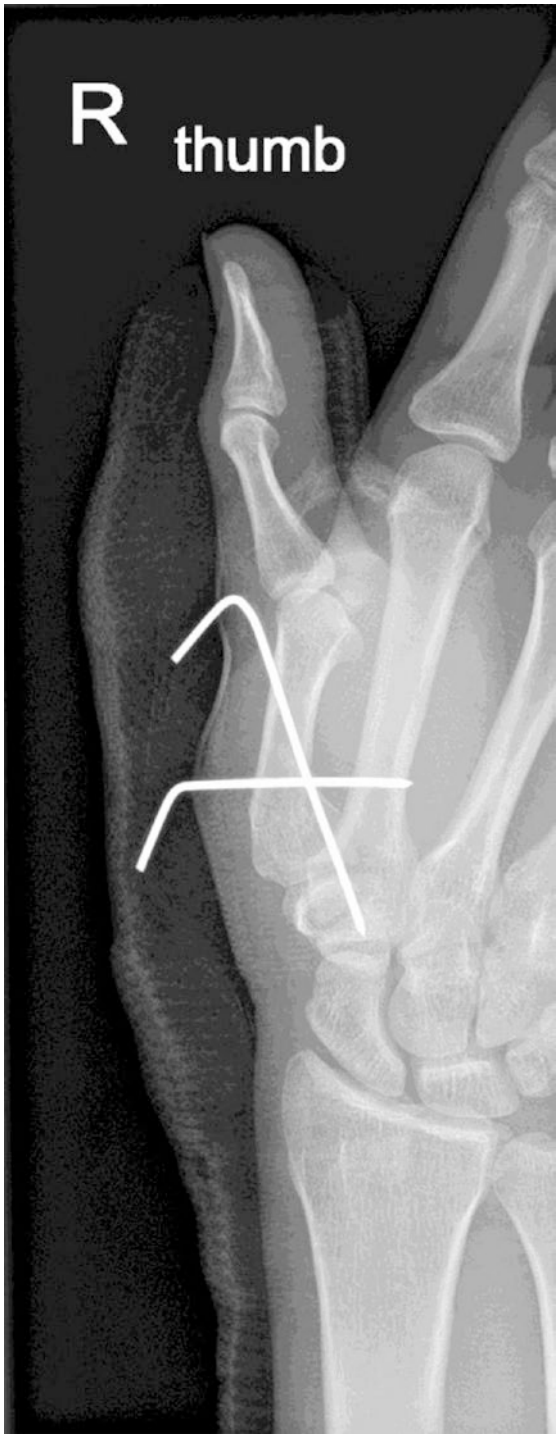

Fig. 10.2 K-wire fixation of Bennett's fracture. (Image courtesy of S. Sarker)

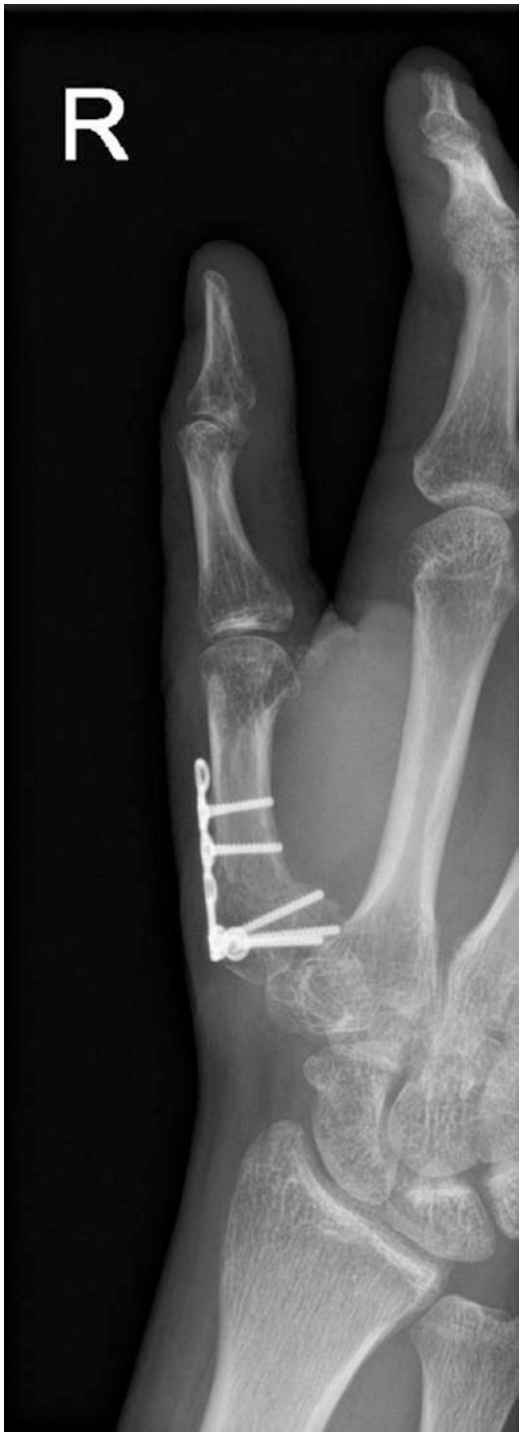

Fig. 10.3 ORIF of Bennett's fracture. (Image courtesy of S. Sarker)

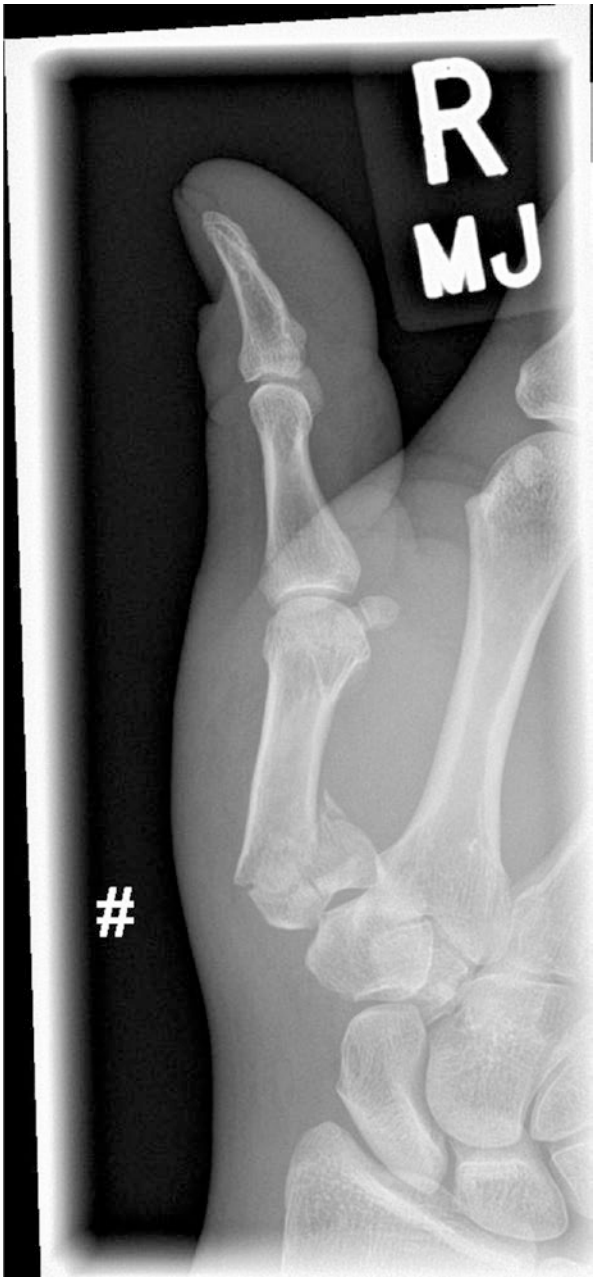

Fig. 10.4 Rolando fracture. (Image courtesy of S. Sarker)

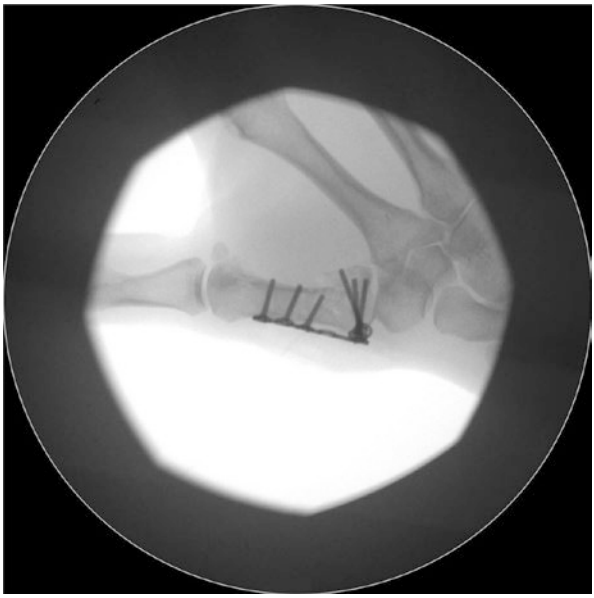

Fig. 10.5 Intra-operative images of ORIF of Rolando fracture. (Image courtesy of S. Sarker)

These fractures are usually treated with surgical management. The surgical management is the same as with the open reduction and internal fixation of Bennett's fractures as described above (Fig. 10.5).

Occasionally, the fracture may be so comminuted that plate and screw fixation cannot be done. In those cases, K-wires can be used to hold the metacarpal shaft in an acceptable position. External fixation can even be used.

The post-operative management plan is similar to that of Bennett's fractures.

Collateral Ligament Injuries

The radial and ulna collateral ligament (UCL) of the metacarpophalangeal joint (MCPJ) can be commonly injured.

Ulna Collateral Ligament

Acute UCL injuries are commonly referred to as Skier's Thumb and chronic UCL injuries are referred to as Gamekeeper's Thumb.

Acute UCL injuries usually happen due to a hyperabduction or hyperextension injury and are called Skier's Thumb as when skier's fall, their thumb often gets caught in the ski pole and have that mechanism of injury.

Patients present with pain at the ulnar aspect of the thumb MCPJ with occasional bruising and swelling. They also have symptoms of instability and find it difficult in grasping objects as well as a weakness in pinch grip.

The UCL is made up of the proper and accessory ligaments. The proper ligament is orientated in an oblique fashion and the accessory ligament in a transverse fashion. It is covered by the adductor aponeurosis.

On examination, patients have acute tenderness at the site of the MCPJ and the surgeon should perform a valgus stress test in 30° of flexion to test the proper UCL and in full extension to test the accessory UCL. With a sprain, there will be some opening of the joint but a firm end point and in completed ruptures, there is gross opening and no firm end point. Often clinically, it is difficult to test as the patient is in a lot of pain. The contralateral thumb should be examined as a comparison [1].

X-rays should be done and usually show a subluxed joint, and occasionally a bony avulsion fragment from the ulnar base of the proximal phalanx can be seen (Fig. 10.6).

Most surgeons would get an urgent ultrasound if they suspect a complete tear. They are looking for a Stener lesion—which occurs when the adductor aponeurosis becomes interposed between the distally avulsed ligament and its insertion into the base of the proximal phalanx. A Stener lesion is an indication for surgical repair, as the UCL will not heal with the soft tissue interposition.

Management of Acute UCL Injuries

Partial tears or complete tears without Stener lesion can be treated without surgery. A thumb spica cast or a thumb spica thermoplastic splint is recommended for 6 weeks followed by hand therapy.

For tears with a Stener lesion, a primary repair is recommended.

The patient is positioned supine with an arm board. Either a general anesthetic or brachial plexus block can be used. It is performed with tourniquet and fluoroscopic guidance. A lazy S incision is made over dorsoulnar portion of the thumb MCPJ. The branches of the superficial radial nerve are identified and protected. The adductor aponeurosis and Stener lesion are identified. The Stener lesion usually obscures the proximal border of the adductor aponeurosis. A longitudinal incision is made in the aponeurosis. The joint is then visualized and inspected for any damage. Midsubstance tears can be sutured using fiberwire or Ethibond. For distal avulsions a micro anchor system is used (Fig. 10.7). The anchor is placed into the insertion of the UCL on the proximal phalanx of the thumb and performed under fluoroscopic guidance. Small bony fragments can be excised. The proximal portion of the UCL is then sutured down onto the bone where the anchor has been placed with the MCPJ flexed at 45° to ensure adequate tensioning. UCL tears with large bony fragments can be repaired with mini fragment screws.

The stability of the repair is tested and then the capsule and aponeurosis are repaired with an absorbable suture such as Vicryl 3/0. The skin is closed with the surgeons preferred suture material. A thumb spica cast is then applied for 2 weeks, and it can be converted to a thumb spica splint for another 4 weeks. Radially directed stress on the thumb should be avoided for 12 weeks.

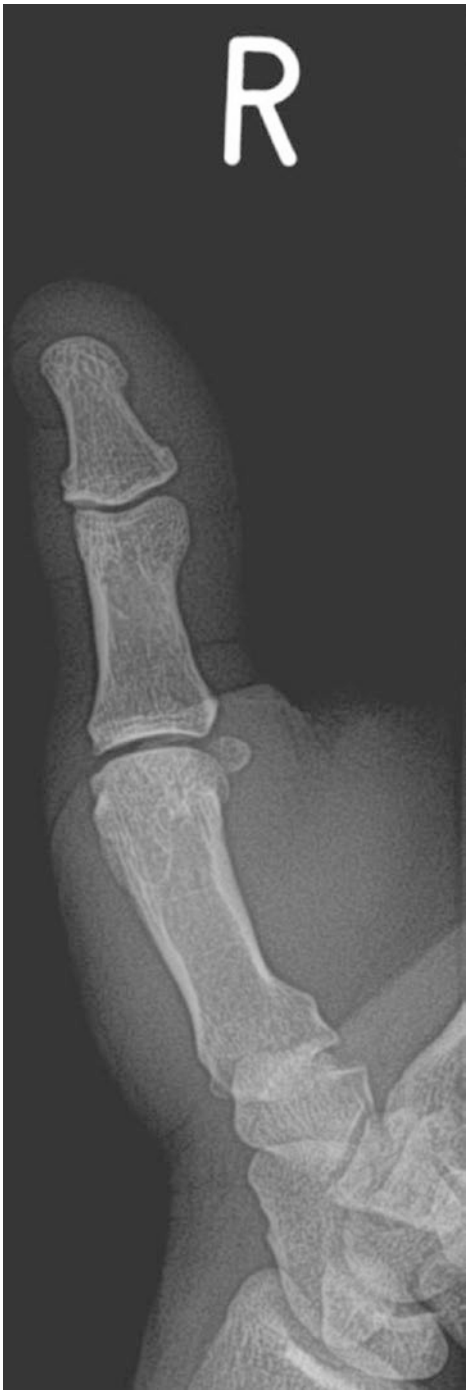

Fig. 10.6 UCL injury. X-rays can appear normal. (Image courtesy of S. Sarker)

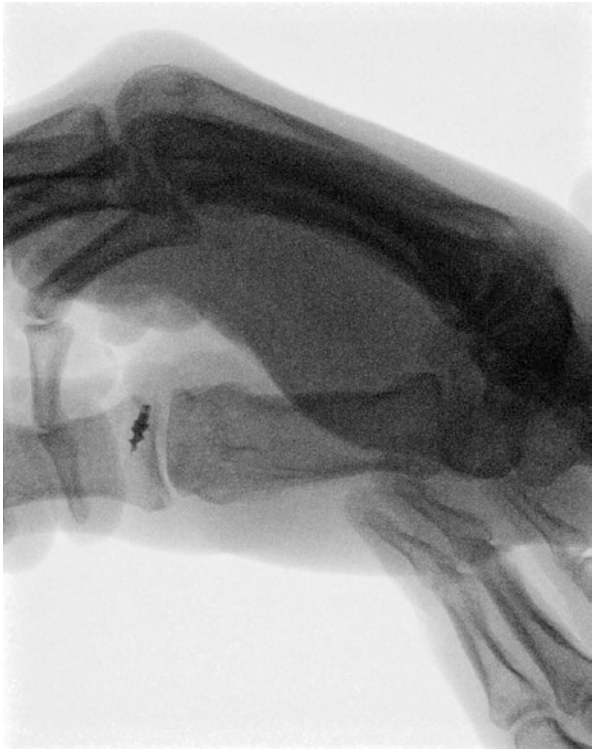

Fig. 10.7 Intra-operative X-ray showing position of suture anchor. (Image courtesy of S. Sarker)

Management of Chronic UCL Injuries

Chronic UCL ruptures are usually due to repetitive strain, failed non-surgical treatment of acute UCL ruptures, or missed Stener lesions. They are usually injuries more than 6 weeks old. The clinical presentation is similar to that in acute injuries and an ultrasound scan should be done to confirm the diagnosis.

For partial tears, the treatment is the same as above.

For surgical management, the approach is the same as for acute UCL ruptures. Intra-operatively, if the ligament is viable, then a repair with a suture anchor can be performed. If the ligament is attenuated, then I would suggest using an artificial ligament augmentation using material such as fibertape. An alternative is using a palmaris longus graft, which is looped through the base of the proximal phalanx and metacarpal neck.

Patients with significant MCPJ arthritis may need either an arthrodesis or arthroplasty instead of ligament reconstruction.

Radial Collateral Ligament (RCL) Injuries

The RCL is almost a mirror image of the UCL. The biggest difference is the broad abductor neurosis is less likely to get caught between the avulsed fragment and insertion of the RCL.

The mechanism of injury is usually forced adduction of a flexed MCPJ.

The management algorithm for RCL injuries are exactly the same as that for UCL injuries.

First Carpometacarpal Joint Dislocations

First CMCJ dislocations are rare but should be considered in traumatic injuries of the thumb.

The first CMCJ is a biconcave saddle joint, which is reciprocally opposed. The contour of the joint provides some primary stability but is further stabilized by the joint capsule and the following five ligaments—beak/volar oblique ligament, first intermetacarpal ligament, dorsoradial ligament, posterior oblique ligament, and the ulna collateral ligament. The first CMCJ participates in three planes of motion—flexion/extension, abduction/adduction, and pronation/supination [1, 2].

The dislocation is usually dorsal and happens with axial compression on a flexed thumb. The patient usually complains of pain at the base of the thumb and there can often be an obvious deformity.

Stress testing should be performed but can be very painful in the acute setting. Standard PA and lateral X-rays should be obtained. Complete dislocations are obvious but partial tears to the stabilizing structures mentioned before usually have normal X-rays.

If appropriate, the reduction can be performed in the Emergency Department with adequate analgesia and a thumb spica cast should be applied with the metacarpal in palmar, abduction, and extension. The spica cast should remain for 4–6 weeks.

If it remains unstable, then a closed reduction and K-wiring (in a similar fashion to Bennett‘s fractures) should be undertaken.

For fractures that cannot be reduced closed or for delayed presentations, it is recommended that an open reduction and ligament reconstruction is performed. There are a variety of methods described and it depends on which ligament needs to be reconstructed. There is considerable debate to which of the ligaments are more important.

First Carpometacarpal Joint Osteoarthritis

First CMCJ arthropathy is very common. It can happen to young patients with a heavy manual job, patients with previous trauma to the base of the first metacarpal, those with rheumatoid arthritis, or those with generalized osteoarthritis. It tends to affect females more than men.

Patients present with pain at the base of the thumb, which can often radiate distally and proximally. It is too important to exclude other causes of pain such as arthropathy of the thumb IPJ, arthropathy of the scaphotrapeziumtrapezoid joint (STTJ) or de Quervain’s tenosynovitis (tenosynovitis of the first Extensor Compartment).

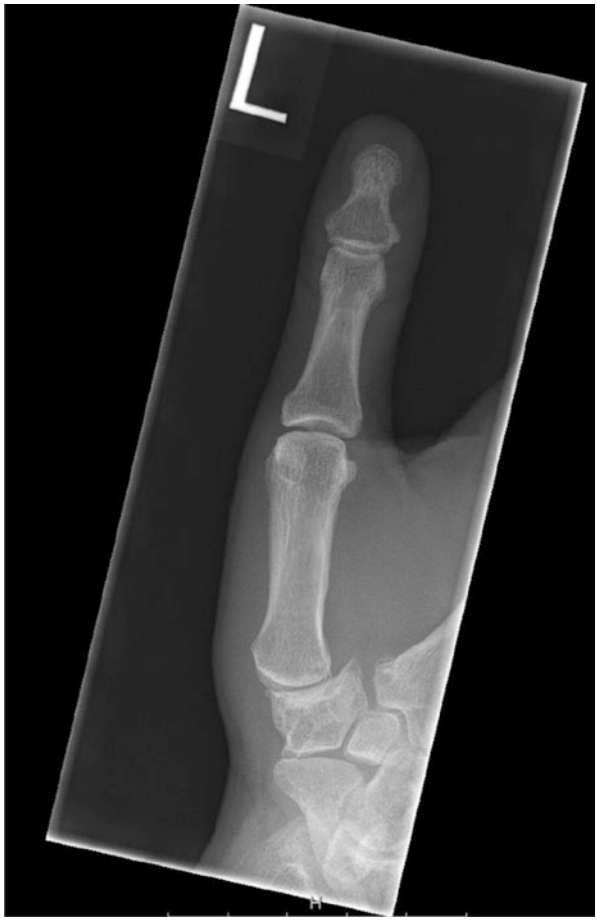

Fig. 10.8 DP view of thumb showing reduction in joint space and subchondral sclerosis. (Image courtesy of S. Sarker)

Patients often hold their thumbs in an adducted position and can be found wearing off the shelf thumb spica splints. They usually complain of difficulty twisting things, opening doors, and jars. Examination reveals tenderness at the base of the thumb and axial loading and grinding reproduces the pain.

Standard DP, lateral, and peritrapezial view X-rays (Figs. 10.8 and 10.9) are recommended to show the extent of the disease as well as the involvement of the STTJ. Usually, no further imaging or tests are required.

Initial management consists of activity modification, splints, and analgesia. Failing that, injection of the first CMCJ with steroid and local anesthetic can be done either under fluoroscopic guidance in theatre or under ultrasound guidance by a radiologist. I prefer to use 40 mg of Kenalog and 1 mL of 1% Lidocaine for the injections.

Some surgeons offer arthroscopy and debridement of the first CMCJ as the next step.

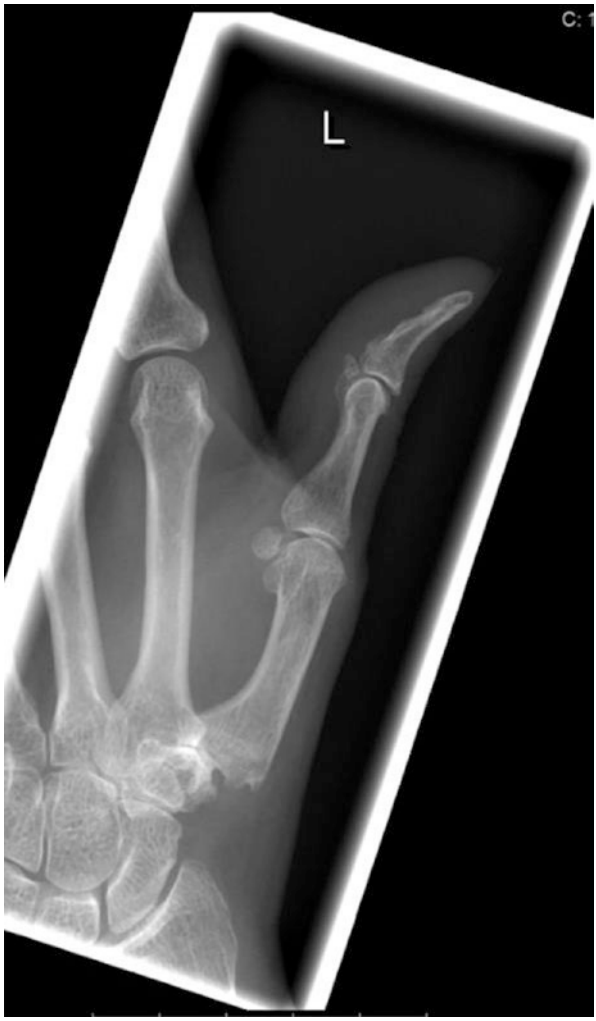

Fig. 10.9 Lateral view of thumb showing advanced first CMCJ OA. (Image courtesy of S. Sarker)

Trapeziectomy remains the gold standard of treatment. This can be done on its own or with a ligament reconstruction and tendon interposition. In my practice, I tend to use an Abductor Pollicus Longus (APL) sling. However, many studies have shown that there is no difference on short and long-term outcomes of the different methods used.

Under either a general anesthetic or a brachial plexus block, the patient is positioned supine with an arm board. This is done under tourniquet control. A longitudinal incision from the base of the first metacarpal towards the radial styloid is made on the dorsoradial surface of the wrist. The branches of the superficial radial nerve

and radial artery are identified and protected. The first CMCJ capsule is incised and the trapezium is identified. The trapezium is released circumferentially and broken into three or four pieces using an osteotome. The Flexor Carpi Radialis tendon is visible at the base of the wound after removal of the trapezium. The pieces are then removed. Bone nibblers are used to ensure no small pieces of bone remain, as this can cause pain. A slip of the APL is harvested through the same incision, looped around the FCR tendon, and sutured to itself using a suture such as fiberwire or Ethibond. The capsule is closed with Vicryl 3/0 and the skin closed either with an absorbable or non-absorbable suture according to surgeon's preference. The patient is placed in a thumb spica cast and this is converted to a splint in 2 weeks when the sutures are removed. Patients are warned about a slow recovery and loss of grip strength.

For younger patients, a fusion may be preferable, as they want to preserve grip strength as much as possible. In some cases, when only part of the joint is affected, an extension osteotomy can be done to offload the joint.

Some surgeons are now performing first CMCJ replacements (Fig. 10.10), as these can reduce the pain without sacrificing motion and grip strength. This can only be performed in cases of isolated first CMCJ OA where the STTJ is preserved. If the STTJ is involved, then a trapeziectomy remains the main surgical option. There are a variety of ball and socket prostheses in the market. The surgical approach is similar to trapeziectomies, and the implant is inserted according to the manufacturer's surgical technique guide. Postoperatively, patients are in a thumb spica cast for 2 weeks and then converted to a splint with motion started under the care of hand therapist.

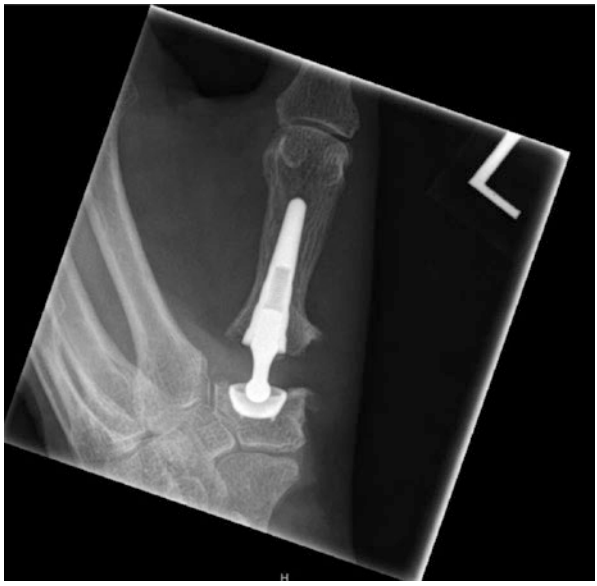

Fig. 10.10 Post-operative X-ray of first CMCJ replacement. (Image courtesy of S. Sarker)

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Hand Infections

11

Shabih Siddiqui and Muzamil Baba

Because of the recent changes in management strategies and early surgical intervention with better microbiology support and antibiotics, the long-term squeal as well as limb and life threatening infections are rarely seen these days.

The basic principles of management, which include early recognition, adequate and aggressive surgical intervention, and methodical timely rehabilitation, have been responsible for a favourable outcome in the majority of infections.

Overall, for any hand infection, *Staphylococcus aureus* continues to be the most common pathogen followed by *Streptococcus.* Recently, increased cases of Gram negative and anaerobic bacteria are reported in immunocompromised individuals including diabetic patients, intravenous drug abusers, and farmyard or fight bite injuries.

There has also been increased prevalence of MRSA, which is proving to be a challenge to recognize and treat at early stages.

The hand is expected to perform beyond expectations. From day to day activities, to be the breadwinner, and even to perform magic, such are the expectations of the hand. Untreated hand infections can lead such a vital and dexterous organ to turnout into a painful and useless appendage.

Paronychia/Eponychia

Paronychia involves infection of the nail fold and is the most common infection of the hand. Common and typical organism is *Staph aureus*, but can also be caused by group A streptococci, bacteroids, etc.

The infection starts as a swelling in the nail fold (Fig. 11.1), which can later develop into an abscess.

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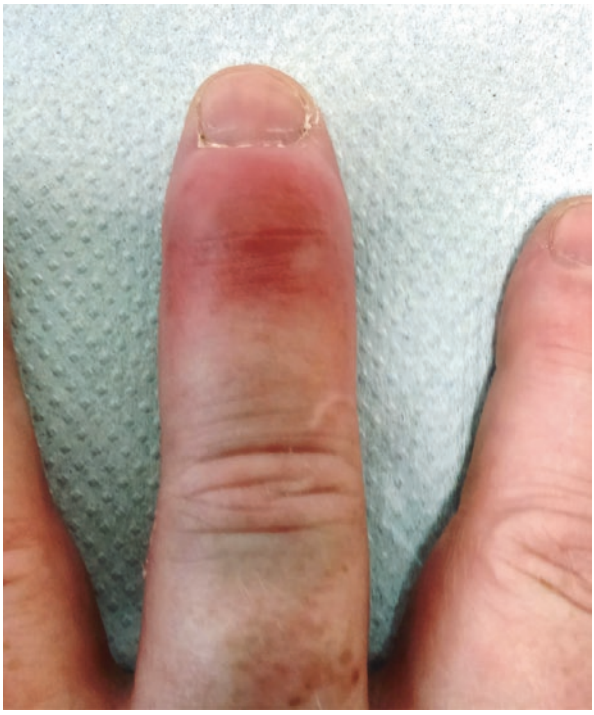

Fig. 11.1 Paronychia middle finger in elderly female with early stage of Cellulitis before abscess formation. (Clinical photo S. Siddiqui/M. Baba)

Initial treatment is by warm soaks, spitting, and antibiotics. Incision and drainage is needed if simple treatment fails and an abscess would need drainage. Part or total nail plate removal may be needed to get rid of the infection.

Rarely, a chronic paronychia caused by fungal infection may be resistant to conventional treatment and needs antifungals, and rarely, marsupialisation, which involves excision of the dorsal eponychium, may be required to completely eradicate the infection.

Topical methylprednisolone in combination with antifungals has also been used successfully to treat resistant infections.

Felon

A felon is an abscess of the terminal pulp space of the phalanx. There are 15–20 septa running from the periosteum to skin of distal phalanx. This explains why these closed space abscesses are so painful and if not treated adequately may cause osteomyelitis of the phalanx. Commonly caused by puncture wounds or foreign bodies, the causative organisms are same as for paronychia. Initially, there is cellulitis, swelling, and tenderness. If left untreated, abscess can form rapidly.

Complications include osteomyelitis, flexor tenosynovitis, or pyogenic arthritis of the terminal joint leading to long-term stiffness and pain. Early treatment includes

warm soaks, antibiotics, and limb elevation, and if not successful, incision and drainage is resorted to.

The incision and drainage is carried out with longitudinal central or a mid-lateral incision to decompress all the small compartments within the pulp. A longitudinal incision or a classic hockey stick incision is given and all septae are knocked down. Incisions are left open to heal by secondary intention.

Tendon Sheath Infections

Again, these are the results of foreign body implantation, puncture wounds, or lacerations. Pyogenic flexor tenosynovitis can present late after a minor penetrating trauma and can evolve rapidly and cause significant complications and spread proximally, causing destruction of pulley system or tendon absorption.

A thorough history and clinical examination including the well-known Kanavel's four classic signs clinch diagnosis. These include

1. tenderness over the involved tendon sheath,
2. flexed attitude of finger,
3. fusiform swelling of finger, and,
4. pain on passive extension of finger.

The index, middle, and the ring fingers are commonly involved.

Coagulase positive *Staph. aureus* is commonly involved but gram-negative organisms are also frequently involved.

Due to anatomical connections, the small finger infection can spread proximally to involve the ulnar bursa.

Early antibiotic therapy may be curative but if after 24–36 h, infection does not subside, incision and drainage should be performed.

Surgery involves either a standard open approach with Bruner incision or minimally invasive approach with small transverse incisions proximally and distally over the A1 and A5 pulleys.

Closed irrigation with a fine catheter, which was previously used, is now utilized less frequently due to risk of secondary infection and extreme swelling of the surrounding soft tissue of the involved finger.

Following a successful eradication of the infection, early involvement of the hand therapists is vital to regain back most of the hand flexibility and function.

Thenar and Midpalmar Infections

These are two potential closed spaces of the hand. The boundaries of the thenar space are as follows: on the ulnar side by the midpalmar oblique septum, volarly by index finger profundus tendon, and on the dorsal side by the adductor pollicis.

Infections in this area present with marked pain, swelling tenderness, and pushing the thumb into abduction.

The midpalmar space is bounded dorsally by the third, fourth, and the fifth metacarpals, volarly by the flexor tendons, radially by the midpalmar septum, and ulnarly by the hypothenar septum. Proximally, these spaces are until the distal limit of the carpal tunnel.

Midpalmar space infections present with swelling not only in the palm but also dorsally. Fluctuation, erythema, and tenderness are however more in the palm. Motion of the middle, ring, and little fingers is reduced.

Treatment of thenar and midpalmar space infections is incision and drainage. Initially, broad-spectrum antibiotics are started, followed by culture-specific antibiotics.

Herpetic Whitlow

This is a viral infection of the fingers caused by contact with herpes simplex virus, predominantly seen in health care workers because of their frequent contact with oral and genital secretions of patients.

Incubation period varies between 2 and 20 days. There is intense throbbing pain and erythema. Vesicles filled with clear fluid, which might coalesce to form bulla. Treatment is usually symptomatic and Acyclovir may be reserved for severe cases. Surgical drainage can lead to disastrous complications. Hence, it is important to distinguish this condition from paronychia, etc.

Human and Animal Bites

Any doctor working in the A&E department knows how frequent these injuries are, especially on a Saturday night. Apart from human bites, dog and cat bites are common. There is a mixture of inoculating organisms and some of the serious sequela are osteomyelitis, chronic stiffness of finger, amputation, and even death.

Tetanus and rabies must always be at the back of the mind of the treating physician.

These wounds need early and thorough irrigation and must be left open. They are secondarily closed if needed.

The common causative organisms for human bites include *Streptococcus*, *Staph. aureus*, *Eikenella corredens*, and *bacteroides* spp.

Dog bites are more frequent than cat bites. However, cat bites can cause more serious infections. Again, the the common causative organisms for animal bites include *Streptococcus*, *Staph. aureus*, and *Pasteurella multocida.*

These organisms are susceptible to a variety of antibiotics, and help from the microbiology department is well worth the effort. Ampicillin/sulbactam and Amoxicillin/Clavulanate are the empirical first line antibiotics with ciprofloxacin and doxycycline for patients who are allergic to penicillin.

Human bites mostly involve third or fourth MCP joints and commonly cause penetration of the metacarpophalangeal joint especially if assault is the mechanism of injury. Once again, thorough exploration and irrigation is needed along with appropriate antibiotics, which initially are parenteral and subsequently oral.

Necrotizing Fasciitis

The term Necrotising Fasciitis was first used by Wilson in 1952 and is used for a particularly extremely dangerous condition, the incidence of which has substantially increased in the last few decades.

It can be potentially limb and life threatening. It is a soft tissue infection manifested by rapidly progressive inflammation and necrosis of subcutaneous fat, fascia, skin, and muscle.

The most common organism involved is Group A *Streptococcus*. An early diagnosis and high index of suspicion is the key to success. Patients with reduced host defenses are particularly liable to infection. Most common group of patients include those with diabetes, malignancy, IVDA, alcoholics, and AIDS.

Sadly, the incidence of necrotizing fasciitis is increasing, especially in the poorer countries.

Early and radical surgical debridement with ruthless removal of necrotic tissue is the key to successful salvage of limb and life. Intra-operative findings are typical with liquefied subcutaneous fat, muscle necrosis, dishwater pus, and widespread thrombosis of the venous system. Patients need judicious use of antibiotics as well as intravenous resuscitation and medical support. In life threatening situations, amputation may be the only option to prevent death.

Tuberculous Infection of Hand

The infection may begin in the tenosynovium of the long flexor tendons of the hand and may be the only manifestation of the disease. It may spread to adjacent bones and joints. Metacarpals can be involved causing tuberculous dactylitis. Differential diagnosis includes rheumatoid arthritis, synovitis, sarcoidosis non-specific synovitis, and fungal infections.

A biopsy may show AFB or caseating granulomas with Langham giant cells. Tuberculosis of wrist may present as Carpal Tunnel Syndrome. Rice bodies may be found during Carpal Tunnel Release. Treatment includes synovial biopsy, surgical debridement, and multi-therapy anti-tuberculous drugs.

Infections by atypical non-tuberculous mycobacterial species can be difficult to diagnose and treat with average time to diagnosis of more than a year. *Mycobacterium marinum* is the most common pathogen involved and treatment involves multiple antibiotic therapy, such as Clarithromycin, Ethambutol, Trimethoprim-sulphamethoxazole, and tetracycline. Long-term antibiotics for 3–6 months are often needed.

Hand Soft Tissue Infections

12

Rachael Fisher and Wasim S. Khan

Principles of Management

In all potential hand infections, initial management must include: *marking the extent of erythema, elevation of the limb, and ensuring baseline laboratory investigations for infection (WCC, CRP, ESR) have been ordered.*

Antibiotics treatment should be tailored to the most likely underling pathogen: *in soft tissue infections, this is most commonly Staphylococcus aureus. In those who are immunocompromised or diabetic, polymicrobial infections should always be considered.*

In cases where there is potential penetrating source of infection*: wounds require urgent management with a thorough washout, debridement of soft tissues, and drainage of collection. Remove any foreign material and primary closure should not be attempted. It is pertinent to confirm immunization status against tetanus and a low threshold for vaccination should be used.*

Urgent Presentations

Necrotizing Fasciitis

Necrotizing fasciitis is a rapidly progressive soft tissue infection hallmarked by the necrosis of the affected tissues.

Mortality rates from necrotizing fasciitis range from 5.4 to 11.1%, with amputation rates in the extremities reaching ~25% [1]. Prompt diagnosis and treatment is paramount in achieving a good outcome, unfortunately, diagnosis is not always easy or clear.

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Table 12.1 LRINEC score for necrotizing fasciitis

Laboratory marker	Result	Score
C-reactive protein (CRP)	≥150 mg/L	4
White blood cell (WCC)	15–25 mm^3	1
	>25 mm^3	2
Hemoglobin (Hb)	11–13.5 g/dL	1
	<11 g/dL	2
Sodium (Na)	<135 mmol/L	2
Creatinine	>141 μmol/L	2
Glucose	>10 mmol/L	1

Score: ≤5 = low risk, 6–7, intermediate risk, >8 high risk (>75% chance) of necrotizing fasciitis [2]

Clinical signs of skin necrosis, crepitus, subcutaneous gas visible on XR or CT, and hemodynamic instability should be taken with high levels of suspicion. LRINEC scoring (Table 12.1) was developed to aid diagnosis (using six laboratory values); a score >8 indicates a 75% chance of having necrotizing fasciitis. However, it should always be used in the context of the clinical situation and index of clinical suspicion.

Necrotizing fasciitis can be either:

- *Monomicrobial*; *S. aureus* or a β-hemolytic streptococci are the most commonly identified bacteria in young healthy individuals.
- *Polymicrobial*; more often the case in the elderly, diabetic, or immunocompromised patient.

Management relies on a combination of prompt high dose, broad-spectrum antibiotics, and rapid and aggressive surgical debridement of affected tissues; often with multiple returns to theatre to ensure clear margins of tissue. Patients require high level of nursing and medical treatment with an often-protracted length of treatment.

Flexor Tenosynovitis

Flexor tendon sheath infections are often a result of a penetrating trauma to the finger; often a seeming innocuous incident. Due to the high potential for significant reduction in movement (due to tendon necrosis, pulley disruption, and adhesions) and potential for amputation, prompt diagnosis and management is key.

The diagnosis of flexor tenosynovitis is predominantly made clinically using the Kanavel's four cardinal signs [3] (Table 12.2);

Similar to the majority of soft tissue infections, the most common causative organism is *S. aureus*. However, polymicrobial infections involving *Streptococcus*, *Staphylococcus epidermidis*, and *Pseudomonas aeruginosa* are also frequently encountered [3].

Management again involves prompt high dose, broad-spectrum antibiotics, and local surgical control. Flexor sheath drainage and irrigation is required using either

Table 12.2 Kanavel's four cardinal signs of flexor tenosynovitis

1	Tenderness on palpation along the flexor sheath
2	Fusiform swelling of the digit
3	Pain on passive extension of the digit
4	Flexed attitude of the finger

an open technique or closed continuous irrigation. They have been described without consensus on superiority [4].

Tuberculosis infection of the hand; although infrequently encountered, flexor tenosynovitis may be the primary manifestation of tuberculosis infection in the hand. The potential for local spread causing tuberculous dactylitis is high, and diagnosis should be made through biopsy (AFB positive, caseating granulomas with Langham giant cells). It is also worth considering that tuberculous can present as carpal tunnel syndrome, with rice bodies found at the time of decompression. Tuberculosis of the hand should be managed in conjunction with the infectious disease department, with the commencement of pharmacological treatment along with any required surgical debridement.

Palmar Deep Space Infections/Abscess

Deep palmar space infections can present following spread from a flexor sheath infection—most notably from the little finger or thumb, or through direct penetrating trauma to the palm.

The palmar aspect of the hand is divided into three distinct areas; the thenar space (adductor pollicis tendon to the third metacarpal), the midpalmar space (third to fifth metacarpal bones), and the hypothenar space (a potential space between the fifth metacarpal and the deep hypothenar muscles). These three areas are separated by septa: the midpalmar and hypothenar septa.

The thenar and hypothenar spaces communicate with the space of Parona in the volar aspect of the forearm—theoretically creating a path for the spread of infection from the thumb flexor tendon directly to the forearm and back to the little finger leading to a "horseshoe" abscess [5].

Thenar abscesses are the most common deep space infection and present with localized swelling, erythema, and direct pain over the thenar eminence, exacerbated with abduction ± opposition of the thumb [1].

Midpalmar abscesses present with fullness of the palm and flexed posture of the middle and ring fingers.

Hypothenar abscesses are the least common deep space infection and are usually the result of penetrating trauma. They often present innocuously, often only with tenderness on direct pressure.

Dorsal abscess formation is rare and often a result of penetrating trauma. A collection on the dorsal aspect of the hand is often a secondary sign of an infection or pathology elsewhere in the hand.

Interdigital abscess often occurs through a fissure in the skin in the webspace between fingers. Limited space on the palmar aspect of the hand means extension to the dorsal aspect occurs quickly and creates a "collar button" abscess, leading to abduction of the fingers and is often seen in children.

Imaging with ultrasound or CT may be of benefit when there is an index of suspicion, although both have been shown to have low sensitivity and specificity for diagnostic purposes. MRI may offer a higher level of specificity but are often more difficult to obtain.

Management, irrelevant of the position of the collection, is with surgical incision and drainage, ensuring both the palmar and dorsal components are targeted. Often, thenar collections require a two-incision approach (palmar and dorsal), whereas midpalmar and hypothenar collections can be addressed with a single palmar incision.

Septic Arthritis

Septic arthritis presents with a warm, swollen joint, often with associated erythema. It can occur secondary to penetrating injury or spread from a soft tissue infection. Without treatment, bacteria can destroy the articular cartilage, causing stiffness and pain in the joint, before potentially spreading into the subchondral bone leading to osteomyelitis.

MCP joint infection is seen as a result of a "fight-bite," clenched fist injury. These patients require wound and joint washout along with antibiotic treatment. X-rays before theatre may aid the identification of retained chipped teeth within the joint before washout.

Bite Injuries

Most commonly caused by cats or dog, or as previously mentioned, humans.

Dog bites are the most common bite injury presenting to the emergency department [6]. Dog bites cause large gaping wounds and often, secondary to the high energy of the trauma, underlying fractures.

Cat bites tend to be much smaller in terms of the destructive superficial injury compared to dog bites, but create much deeper wounds due to the sharp nature of their teeth. They are more prone to causing tenosynovitis and care must be taken to ensure the path of penetration is fully explored.

Human bites, whilst less commonly encountered, pose a risk of joint contamination and the development of septic arthritis or extensor tendon injuries [6].

Animal bites have specific pathogens for infection and antibiotic cover should be commenced early (Table 12.3):

All patients should be protected against tetanus and counselled individually with regard to the risk of rabies.

Table 12.3 Common pathogens following animal and human bites

Animal	Pathogen
Human	1. *Staphylococcus aureus* 2. α-Hemolytic streptococcus 3. *Eikenella corrodens* 4. Bacteroides
Dog	1. *Pasteurella multocida* 2. Streptococcus viridians 3. Bacteroides
Cat	1. Pasteurella mutlocida

Management of wounds is required early with irrigation and should never be closed primarily.

Depending on the location of the bite the potential for deep space infection, flexor tenosynovitis and septic arthritis should be considered and managed accordingly.

Wound reviews should take place at regular intervals in the days following the injury.

Common Presentations

Cellulitis

Cellulitis of the hand is an infection in the subcutaneous tissues. It presents with the well-recognized combination of erythema, warmth, and pain. There may be a history of trauma and puncture wounds may be visible. The skin is usually tender to touch with evidence of induration but not fluctuant.

Depending on the severity of the infection, patients may be septic (tachycardic, pyrexial, and hypotensive) with evidence of a high white cell count and CRP.

The most common pathogen leading to cellulitis in the hand is *S. aureus* but, as with all infections, polymicrobial infections are more common in those with diabetes or immunocompromised.

Management of cellulitis is with antibiotics and elevation. Ensure the affected area is marked and any spreading cellulitis should be investigated with an index of suspicion for necrotizing fasciitis.

If left untreated, cellulitis can lead to the development of an abscess or rapidly progressive infection into other compartments of the hand [5].

Felon/Pulp Infections

Subcutaneous abscess in the distal pulp of a digit. The pulp is divided by multiple (~20) fibrous septa that in the presence of infection and inflammation can quickly

develop a build-up of pressure causing considerable pain. It is often caused by penetrating trauma and more common in diabetic patients.

Presentation is initially as a swollen, tender area in the pulp of the distal portion of the finger, but if left untreated can progress to a rapidly forming abscess, which can be complicated by skin expansion leading to necrosis or sinus tract formation. Similarly, due to the fibrous septa being adherent to the periosteum of the distal phalanx, osteomyelitis can rapidly progress and there is potential for flexor tenosynovitis or septic arthritis.

Early presentations can be managed with antibiotics, elevation, and soaks in warm water. In those presenting with a large collection, incision and drainage may be required, taking care to incise and disrupt all involved septations.

Paronychia

Paronychia is an infection of the tissue adjacent to the nail fold: initially beginning as a subcuticular abscess in the nail fold, the infection has the potential to spread under the nail plate and into the nail pulp. If the germinal matrix is involved, pressure necrosis can occur and nail growth arrest may result.

Frequently, it is the result of nail biting and dishwashing where the barrier to infection has been lost. The skin may appear erythematous, swollen, and tender.

Management, as with felons, depends of the presence of a drainable collection. If no drainable collection; warm soaks and antibiotics, if a drainable collection is present; incision and drainage to augment antibiotic therapy.

Whitlow

A cutaneous viral infection caused by herpes simplex virus. Clear, painful vesicles frequently resulting in bullae over the fingertip. This is frequently seen in healthcare workers exposed to the saliva of patients via herpes with an incubation period of 2–20 days. Self-limiting condition, which infrequently in severe cases requires antiviral treatment. Surgical drainage is never advocated.

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Congenital Deformities of the Upper Limb

13

Mira Pecheva, Kendrick To, Neil Rushton and Wasim S. Khan

Introduction

Congenital upper limb deformities may occur in isolation or they may be part of a syndrome. It is, therefore, important to take a full history, including antenatal, perinatal and postnatal history, and a family history.

Examination should include the whole limb and the contralateral upper limb, the lower limbs, and a high index of suspicion for associated malformations if there is a possibility that the deformity is part of a syndrome.

The management should be in conjunction with the pediatric team and should include support for the child and the family. The decision of operative timing should be carefully discussed. Staged reconstructions may be required.

Epidemiology

The reported incidence of congenital hand anomalies is 21.5 per 10,000 live births [1] according to 2010 data. This appears to be unchanged, with previous numbers of 11.4–19.7 per 10,000 live births according to 2001 [2].

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Number and Type	Example
1: Failure of formation: a. Transverse b. Longitudinal c. Central	a. Congenital transverse amputation b. Radial deficiency Ulnar deficiency c. Cleft hand
II: Failure of differentiation (separation)	Radioulnar synostosis Carpal coalition Syndactyly
III: Duplication	Polydactyly
IV: Overgrowth	Macrodactyly
V: Undergrowth	Symbrachydactyly
VI: Congenital Constriction band syndrome	
VII: Generalised	Skeletal dysplasias

Fig. 13.1 The classification of congenital hand anomalies by Swanson

Classification of Congenital Hand Deformity

Various classifications exist but the most widely used is the Swanson Classification (Fig. 13.1) [3], which has been modified by the International Federation of Societies for Surgery of the Hand (ISSH) and the American Society for Surgery of the Hand (ASSH) [3]. It was introduced as a descriptive classification, minimizing the use of Greek or Latin. Importantly, this classification does not describe the etiology of the congenital abnormality, and not all malformations can be classified according to this system.

Failure of Formation

Transverse Absence

Examination

This type of limb absence occurs when the limb stops forming past a certain level, most often at the proximal 1/3 forearm, transmetacarpal (Fig. 13.2), distal forearm (Fig. 13.3), and transhumeral. The absence is usually unilateral (83%) with no specific genetic cause [4].

Management

Prosthetic limbs, including 3D printed prostheses, can be used [5].

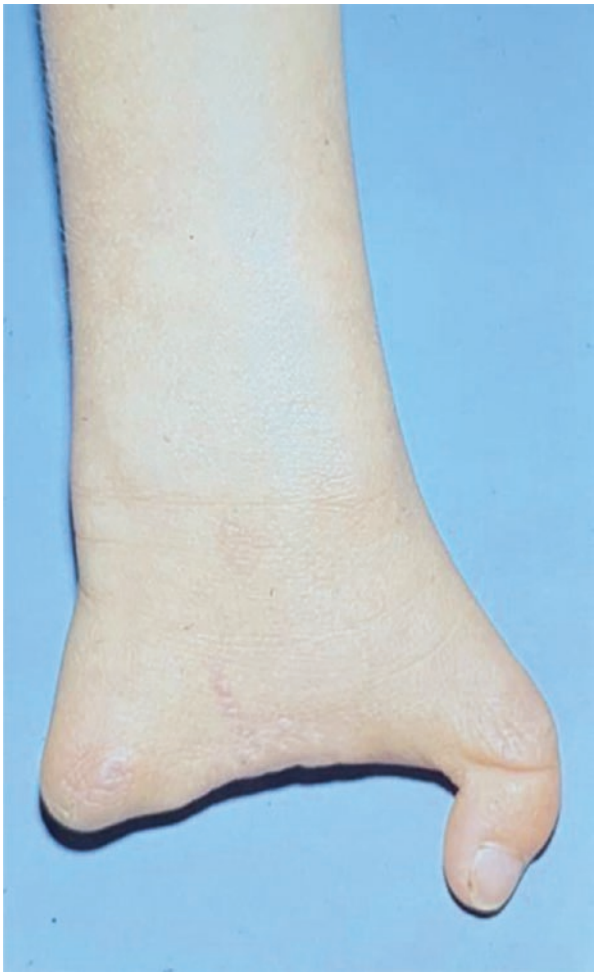

Fig. 13.2 Clinical photograph of transmetacarpal transverse absence of medial four digits in the left hand

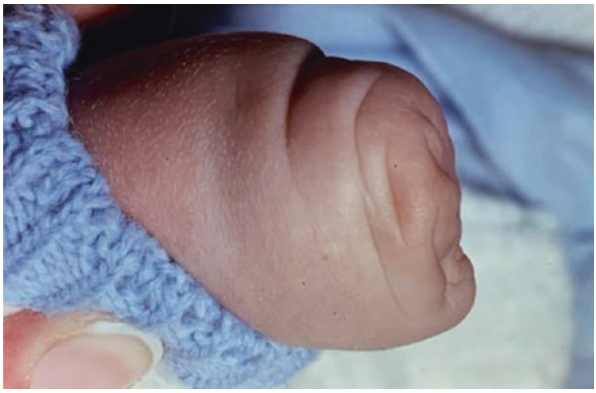

Fig. 13.3 Transverse absence of the hand

Longitudinal Absence

Radial Longitudinal Anomalies: Radial Hypoplasia, Aplasia

Radial longitudinal deficiency can affect a range of radial structures, including the radius, carpus, and thumb, to varying degrees. This includes the bony, ligamentous, and neurovascular structures and can range from hypoplasia to absence. It is a rare condition and affects 0.2–0.5/10,000 live births [1]. It is thought that most are due to spontaneous genetic mutations [6].

Examination

Depending on the structures affected, patients may present with a short or bowed forearm and hand, unstable wrist with/without radial deviation (Figs. 13.4 and 13.5), thumb absence or hypoplasia, or impaired thumb function and digits with a reduced range of movement and power [7]. It is important to look for skin creases to determine if the joint moves. More than 50% have bilateral involvement, which is often asymmetrical [7].

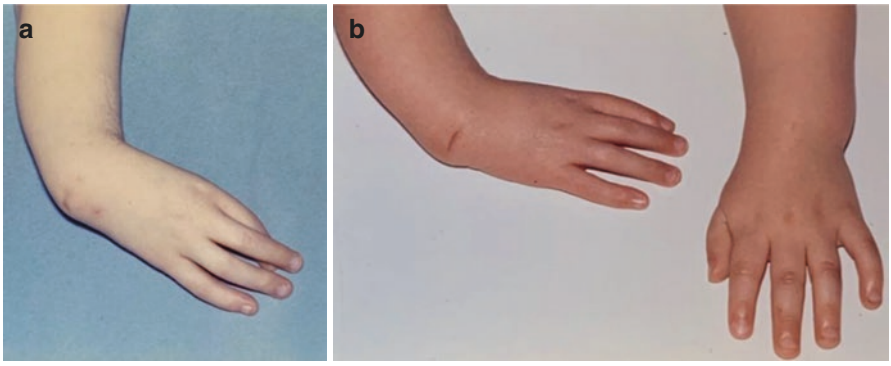

Fig. 13.4 (**a**, **b**) Clinical photographs of longitudinal distal radial deficiency of right hand

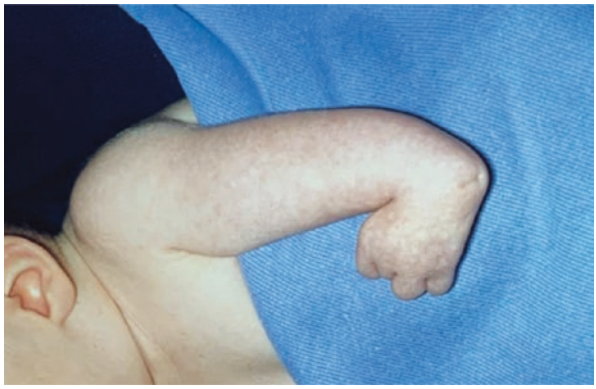

Fig. 13.5 Clinical photograph of longitudinal radial deficiency of the left arm with hypoplasia of the proximal radius

Associations

Radial hypoplasia is often associated with a syndrome, and it is important to look for "the big 4" as this may be the first presentation of a life-threatening syndrome, with appropriate multi-disciplinary management:

1. VACTERL syndrome—vertebral anomalies, anal atresia, cardiac anomalies, tracheoesophageal fistula, renal anomalies, limb anomalies.
2. Holt-Oram syndrome—autosomal dominant condition associated with cardiac defects.
3. TAR syndrome—thrombocytopenia, anemia, absent radius. Autosomal recessive inheritance. The thumb is often present.
4. Fanconi anemia—an autosomal recessive type of bone marrow failure syndrome characteristic phenotypic manifestations. Blood film: aplastic anemia.

Other syndromes such as trisomy 13 and 18 may also present with radial hypoplasia or aplasia.

Investigations

XR: Limb—assessment of affected structure, particularly, in children over 8 years old; Spine–vertebral assessment

Ultrasound: renal assessment

Echocardiography

Classification

Bayne and Klung classified radial longitudinal deficiency into four types based on the radiographic findings [8]. James and McCarroll [9] modified this classification to encompass radial, carpal, and thumb abnormalities (Fig. 13.6).

Management

Type 0, 1, or mild type 2 deficiency can be managed with stretching or splinting. Tendon transfers and soft tissue releases can help with significant radial deviation. A range of operative options exist for more severe deficiencies, including radial lengthening, carpal centralization on the distal ulna. This is particularly useful for type 3 and 4 deficiencies, where there is wrist instability and radial deviation. Soft tissue distraction with a ring external fixator may be necessary before centralization [10].

Initially, studies focused on outcomes following operative management. Retrospective case series have identified a high late deformity recurrence and impaired ulnar growth. Notably, active range of motion in the wrist and digits may be more important for activity and self-perceived disability than radial angulation of the wrist [7].

Ulnar Deficiency

A spectrum of deficiency varying from absent ulnar rays, carpal bones to ulnar absence with radial head dislocation or radiohumeral synostosis and shoulder

Type/Thumb	Carpus	Distal radius	Proximal radius
N: Hypoplastic or absent	Normal	Normal	Normal
0: Hypoplastic or absent	Absence, hypoplasia or coalition	Normal	Normal, radioulnar synostosis or congenital radial head dislocation
1: Hypoplastic or absent	Absence, hypoplasia or coalition	Radius > 2mm shorter than ulna	Normal, radioulnar synostosis or congenital radial head dislocation
2: Hypoplastic or absent	Absence, hypoplasia or coalition	Hypoplastic	Hypoplastic
3: Hypoplastic or absent	Absence, hypoplasia or coalition	Absent physis	Variable hypoplasia
4: Hypoplastic or absent	Absence, hypoplasia or coalition	Absent	Absent

Fig. 13.6 Classification of radial longitudinal deficiency by Bayne and Klung, modified by James and McCaroll

anomalies [11] affecting 1 in 5000 to 1 in 100,000 live births, and it is 4–10 times less common than radial longitudinal absence [12].

It is thought to be caused by deficiency of Sonic hedgehog protein (Shh) expressed in the zone of polarizing activity, the main controller of anteroposterior limb development [12, 13].

Examination

It is more commonly unilateral than bilateral and the entire affected limb is usually hypoplastic. Hand and carpal anomalies are always present [14]. There may be

absent ulnar digits, syndactyly of the remaining digits, first web space anomalies. There may be elbow anomalies such as radial head stiffness due to radiohumeral synostosis or ulnar deviation, due to a large fibrocartilaginous ulnar "anlage," which may be attached to the distal radial epiphysis or the ulnar side of the carpus [15]. The radial head may be dislocated.

Associations

There may be femoral deficiency, fibular deficiency, phocomelia, scoliosis or skeletal dysplasia. Ulnar deficiency is not commonly associated with syndromes [16, 17].

Investigations

XR—the fibrocartilaginous "anlage" may not be visible on XR.

MRI—to exclude synostoses and evaluate the joints.

Type	Thumb and first web space
A	Normal
B	Mild deficiency
C	Moderate – Severe deficiency: Loss of opposition; malrotation of thumb into plane of fingers; thumb / index finger syndactyly (Fig. 5); absent extrinsic tendon function
D	Absent thumb

Fig. 13.7 Classification of ulnar deficiency by Cole and Manske

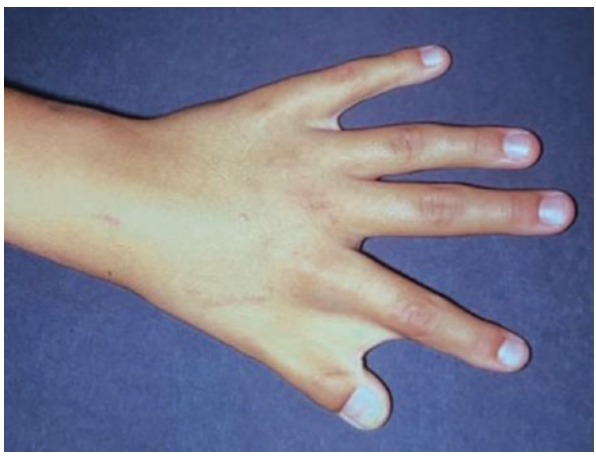

Fig. 13.8 Clinical photograph of syndactyly of the first web space in the left hand

Classification

Cole and Manske classified ulnar deficiency according to the thumb and first web space (Fig. 13.7) [14]. There is progressive involvement of the thumb and first web space. (A) Normal; (B) extrinsic tendon function is intact and thumb opposition is preserved; (C) there is varying involvement of the thumb and first web space (Fig. 13.8); and (D) complete thumb absence. The classification aimed to focus the surgeon on the deficiencies that are most important for the restoration of function [14].

Management

Splinting and stretching exercises may be helpful for mild deficiencies.

Thumb reconstruction with first web space deepening, opponensplasty and pollicization can help to improve function [12]. Ulnar anlage excision should consider the proximity of the ulnar neurovascular bundle, particularly on excision of the anlage from the carpals. Centralization can help to improve stability. The position of the forearm can be improved with conversion of one-bone forearm in the cases of severe instability and significant loss of distal ulna or proximal radius [18]. Excessive limb internal rotation, which can inhibit independent feeding, can be addressed with humeral transverse rotation osteotomy. Elbow procedures are rarely performed but can include radial head excision and synostosis osteotomy [12].

Central Deficiency: Ectrodactyly (Cleft Hand)

Absence of one or more of the central digits of the hand is uncommon. The incidence is 0.52 per 10,000 live births [19]. Inheritance is usually autosomal dominant, but spontaneous genetic mutations also occur. This occurs due to suppressed development of the central or medial apical ectodermal ridge are responsible for central clefting [20].

Examination

Deficiency or absence of one (Fig. 13.9) or more (Fig. 13.10) central hand fingers may be seen in the hand, with a central V-shaped cleft without digital remnants

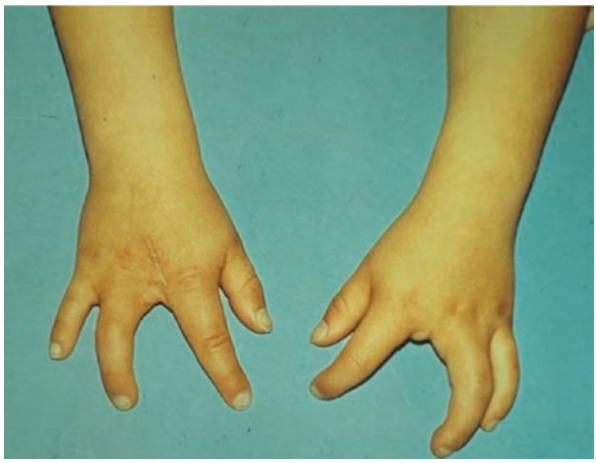

Fig. 13.9 Clinical photograph of bilateral ectrodactyly with absence of the middle digit

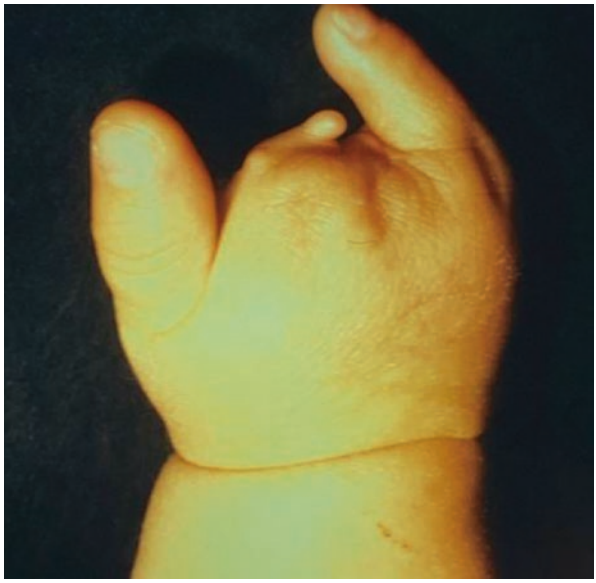

Fig. 13.10 Clinical photograph of ectrodactyly of the right hand with absence of multiple central digits

("nubbins"). The thumb may or may not be present. There may be a family history of cleft hand, which may be seen in the accompanying parent. It is important to differentiate cleft hand from an atypical cleft in the hand, as seen in Symbrachydactyly (see Type V anomalies—Undergrowth, Symbrachydactyly). In symbrachydactyly, the inheritance pattern is spontaneous, without lower limb involvement, a "U"-shaped cleft is seen and finger nubbins can be present.

Associations

A cleft may also be present in the feet, as in split hand / foot malformation (SHFM), inherited in an autosomal dominant pattern with variable penetrance, or as part of Ectrodactyly, Ectodermal dysplasia and Cleft lip/palate (EEC) syndrome.

Investigations

Referral to a geneticist can be useful in determining the likelihood of inheritance for future generations.

Classification

The Manske and Halikis classification is based on the presence of the first web space (Fig. 13.11) [21].

In Type IV, where there is a merged web and the index ray osseous elements are suppressed, the thumb web space is merged with the cleft and there is accompanying ulnar collateral ligament instability of the thumb metacarpophalyngeal joint.

In Type V, the thumb and radial osseous elements are completely suppressed and only the ulnar rays remain.

Management

Patients with a family history and those without may take a different view. It is imperative that function is preserved and not lost in reconstructive efforts. Surgery

Type	First web space	Characteristics
I	Normal web	Thumb web space is not narrowed
II A	Mildly narrowed web	
II B	Severely narrowed web	
III	Syndactylyzed web	Thumb and index rays syndactylyzed, obliterated web space.
IV	Merged web	Index ray osseous elements are suppressed, thumb web merged with cleft.
V	Absent web	Thumb elements completely suppressed, ulnar rays remain, thumb web space no longer present.

Fig. 13.11 Classification of central deficiency according to the thumb web space by Manske and Halikis

may be indicated in progressive cleft deformity. For Type I defects, where there is a normal thumb web space, cleft focus is the surgical goal. In Types IIA, IIB, and III, the thumb space is narrowed so reconstruction is aimed at widening it with local flaps or dorsal/volar transposition flaps from the cleft. In Type IV, the web space is merged, so the focus is on reducing the wide web space and stabilizing the metacarpophalyngeal joint. Toe-to-hand transfers may be considered in Type V deficiencies [22].

Failure of Differentiation (Separation)

Syndactyly

Congenital syndactyly has been linked to aberrations of apoptosis between the digits, with lack of interdigital cleft formation. Apoptosis is usually controlled by bone morphogenic proteins (BMPs), with subsequent reduction of fibroblast growth signalling in the apical ectodermal ridge [23] and it occurs in 1 in 2000–3000 live births [24]. The inheritance of syndactyly is usually autosomal dominant, but autosomal recessive types also exist [25].

Examination

The skin covering the syndactyly would be insufficient to cover each digit and the fascial covering consists of a thickened lateral digitals sheet.

Simple syndactyly consists of anomalous skin/soft tissue connections, usually with normal neurovascular anatomy and extensor tendons. Complex syndactyly has underlying bony anomalies, with evidence of nail fusion. In synonychia, with loss of the paronychial fold and flattening of the nail matrix. Complex complicated syndactylies have additional phalanges or digits in the anomalous web space. Syndactyly may be simple or multiple. Examination should include the full limb, contralateral limb, lower limbs, and chest wall.

Associations

Syndactyly can be syndromic, with over 300 anomalies identified [26], or non-syndromic, with nine distinct forms [25]. The syndactyly seen in Apert syndrome affects all five digits and can be extensive, involving the matacarpophalyngeal joints, classified by Upton [27].

Investigations

XR—to evaluate the bony anatomy and additional digits.

Management

Long-term studies have shown that delayed surgery after 18 months has superior outcomes, although early surgery may be necessary if there is progressive deformity. Interdigital commissure reconstruction can be achieved with local skin flaps, "Z"-plasty or a double-opposing "Z"-plasty "butterfly flap" for mild web deepening. Cronin combined palmar and dorsal triangular flaps and corresponding zigzag incision on the dorsal and palmar surface [28]. In cases of distal phalangeal fusion, it is necessary to reconstruct the paronychial fold. Various techniques exist using a skin flap from the fused finger pulp or a composite skin graft from subcutaneous fat [29].

Duplication

Polydactyly

Polydactyly occurs in 1 in approximately 700–1000 live births [24]. Syndactyly can occur as part of a syndrome (syndromic polydactyly) or as a separate manifestation (non-syndromic), with the latter classified as preaxial (radial), axial (central), and postaxial (ulnar) [30]. The inheritance is often autosomal dominant with variable penetrance due to defects in the anteroposterior patterning. Six genes at ten loci have been identified, including ZNF141, GLI3, MIPOL1, IQCE, PITX1, and the GLI1 [30]. Preaxial polydactyly in a Caucasian patient is often associated with an underlying syndrome. Postaxial polydactyly is more common in children of African descent.

Classification

Wassel [31] classified preaxial (radial) polydactyly (Fig. 13.12) and this was later modified by Wood [32] to expand on the description of the duplicated triphalangeal thumb.

The Wassel classification does not identify the origin of the additional digit and, therefore, has limited application in surgical planning. The Rotterdam classification, by Zuidam et al. [33], identifies the involved joint and has broader classification capabilities according to a recent multicenter study [34].

In postaxial (ulnar) polydactyly, the extra digit can be well developed (type A), with little finger osseous and soft tissue structures that articulate with the

Type	Duplicated element
I	Bifid distal phalanx
II	Duplicated distal phalanx
III	Bifid proximal phalanx
IV	Duplicated proximal phalanx
V	Bifid metacarpal phalanx
VI	Duplicated metacarpal phalanx
VII	Triphalangeal component

Fig. 13.12 Wassel classification of thumb duplication

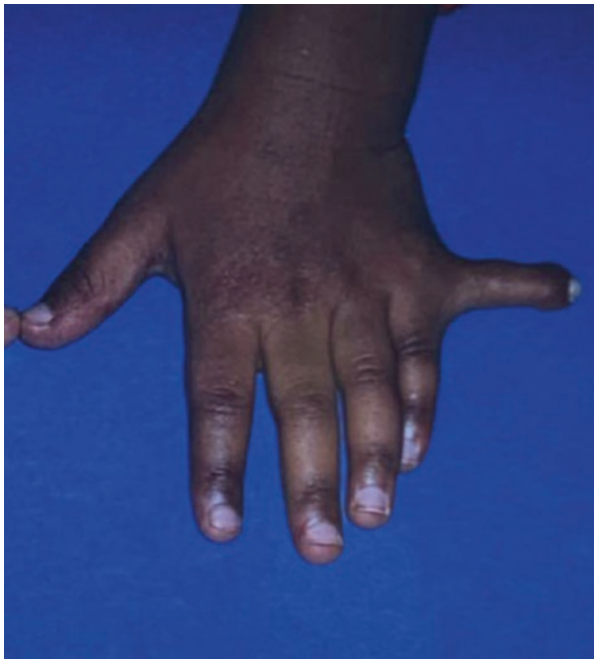

Fig. 13.13 Clinical photograph of polydactyly of the Fifth Digit in the left hand

fifth metacarpal (Fig. 13.13); or rudimentary (type B), existing as a pedunculated appendage attached to the little finger.

Management

Thumb duplications (Figs. 13.14a, 13.14b, and 13.14c) usually require resection and reconstruction including web space deepening. "On-top-plasty" involves the transfer of better developed distal components to better-developed proximal components [35].

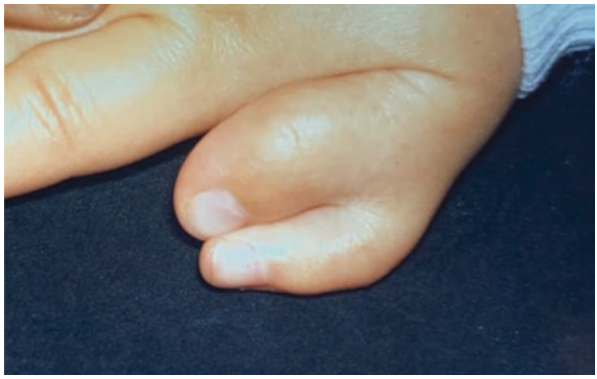

Fig. 13.14a Clinical photograph of thumb duplication of the right hand

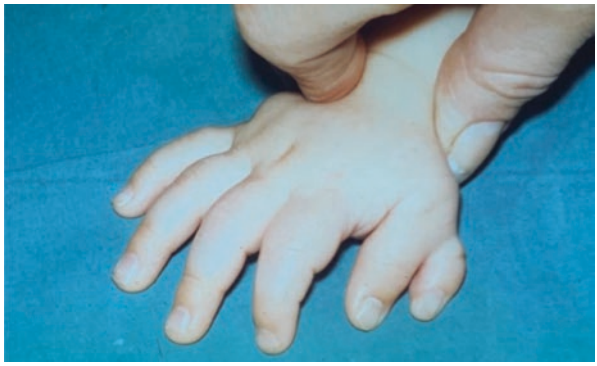

Fig. 13.14b Clinical photograph of thumb duplication of the right hand

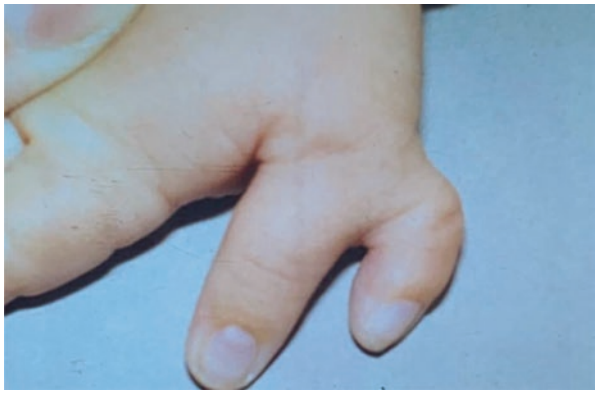

Fig. 13.14c Clinical photograph of thumb duplication of the right hand

Ulnar polydactyly remnants may be managed with string or ligature ligation in the neonate, but care must be taken to prevent painful neuromas.

Ulnar Dimelia (Mirror Hand)

This spectrum of anomalies is caused by ectopic expression of the Zone of Polarizing activity or the Sonic hedgehog gene, leading to aberrations in limb patterning on the anteroposterior axis [36].

Classification

Ulnar dimelia (mirror hand) is a spectrum of duplications ranging from a two ulnae with multiple fingers (Type 1) to complete duplication of the hand, (Type 5) with a normal forearm [37]. This is a descriptive classification.

Investigations

Radiographs may show humeral articulation with two ulnae. The radius may be absent. There may be eight triphalangeal digits.

Management

Early reconstruction, preferably before the age of two, involves selective digital ablation and pollicization and development of the first web space.

Overgrowth

Macrodactyly

Macrodactyly is a form of congenital limb enlargement affecting 0.08 per 10,000 neonates [38]. It is most commonly unilateral and affects multiple digits. It has been linked to PIK3CA mutations as part of the PIK3CA overgrowth spectrum as a form of somatic mosaicism [39]. Most cases are due to sporadic mutations.

Examination

Two growth patterns have been identified—static macrodactyly, where the rate of growth of the enlarged digit(s) is the same as the other non-affected digits; and progressive macrodactyly, where the affected digit(s) enlarge at a greater rate.

There may be associated lipofibromatosis.

Hyperostotic macrodactyly presents with osteocartilagenous masses around the joint(s) and can cause limitation in the range of movement.

There may be neurological compression.

Nerve–Territory-oriented macrodactyly presents with overgrowth along the nerve territory, most commonly in the median nerve distribution.

Associations

There may be segmental gigantism, affecting one part of the limb, or hemihypertrophy, with unilateral body involvement, in association with neurofibromatosis.

Investigations

MRI is useful to detect vascular malformations.

Genetic testing can detect PIK3CA mutations.

Classification

Flatt classified macrodactyly based on the pathogenesis: Type (I) lipofibromatosis; (II) neurofibromatosis; (III) polyostotic; and (IV) hemihypertrophy [40].

Management

Management options include limiting limb growth with epiphysiodesis, performed when the digit(s) reach the size of the digit of parent of the same gender; debulking procedures; osteotomies to correct deformity or amputation. Keloid scarring may be problematic, particularly with PIK3CA. Web creep (commissure growth), can be problematic and Brunner's lines are imperative in surgical planning.

Undergrowth

Thumb Hypoplasia

Underdevelopment of the thumb can manifest as an isolated entity complete absence. It affects 1 in 100,000 neonates [41].

Examination

Careful examination of the thenar muscles, creases, and first web space. There may be radial longitudinal anomalies.

Associations

Syndromes include VACTERL, Holt–Oram, TAR, and Fanconi anemia.

Classification

Blauth [42] classified thumb hypoplasia on the degree of hypoplasia based on Müller's work, with later modification by Manske and McCarroll to subdivide grades 3A and 3B depending on the presence or absence of the proximal metacarpal (Fig. 13.15) [43].

Management

Surgical options include metacarphophalangeal stabilization, ulnar collateral ligament reconstruction, opponensplasty to address absent intrinsic thenar muscles, extrinsic muscle exploration, pulley release, and pollicization. The first web space may need deepening. Importantly, the index finger may be anomalous, with reduced grip strength and range of movement. Reconstruction may need to be staged.

Symbrachydactyly (Fused Short Fingers)

This encompasses a range of anomalies from hypoplasia (Fig. 13.16) to aplasia. It can be attributed to failure of differentiation and formation and, therefore, difficult to classify according to the ISSH system. The incidence is 0.6 per 10,000 live births. It is thought that subclavian artery disruption distal prior to 42 days gestation causes limb growth arrest and interdigital tissue degeneration [44].

Examination

Rudimentary digits ("nubbins") with remnants of nail plate, bone, and cartilage are classic. There may be absence of the central digits with normal or hypoplastic border digits.

Type	Finding
I	Minimal shortening and narrowing
II	First web space narrowing Hypoplastic intrinsic thenar muscles MCP joint instability A: Uniaxial B: Multiaxial
III - A	Type II + extrinsic tendon abnormalities Hypoplastic metacarpal Stable CMC joint
III - B	Type II + extrinsic tendon abnormalities Partial metacarpal aplasia Unstable CMC joint
IV	Floating thumb
V	Absent thumb

Fig. 13.15 (Modified) Blauth classification of thumb hypoplasia

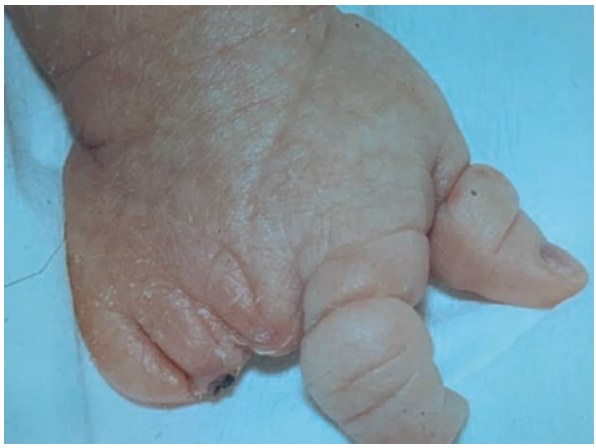

Fig. 13.16 Clinical photograph of symbrachydactyly of the right hand with hypoplasia of the medial three digits

Associations
Poland's syndrome, with an absence of chest wall muscles, thought to be due to subclavian artery disruption.

Classification
There are multiple descriptions with four main groups, originally described by Blauth [45]: (1) short finger type, where all four fingers are incompletely formed but the thumb is normal; (2) atypical cleft hand—with an aplastic central hand and less affected border digits; (3) monodactylic—absent fingers with a present thumb; and (4) peromelic, with a transverse line of developmental arrest the MCP joint level and rudimentary digits distally. Additional modifications include acarpia type, with absent digits and partial or complete absence of the carpals; and forearm amputation type, with an absent distal forearm with rudimentary digits.

Management
Rudimentary digits can be excised, as nail components can cause local inflammation and infection. The main goal is to achieve a functional hand, with a stable thumb, wide first web space, and at least one ulnar digit for opposition.

Constriction Ring Syndrome (Amniotic Disruption Sequence or Amniotic Band Syndrome)

This term "Syndrome" is a misnomer, as this spectrum of anomalies does not arise in any form of consistent pattern. Fibrous band constriction of the fetus leads to varying abnormalities, which most commonly affect the limbs, with 0.98–1.16 per 10,000 neonates affected, with a limb amputation rate of 0.62 per 10,000 [46, 47].

The intrinsic (intra-fetal) postulates that there is disruption of the germinal disc in early embryogenesis or vascular disruption, whereas the extrinsic theory suggests traumatic amnion disruption.

Association
Prematurity—34%.

Investigations
Antenatal ultrasound with amniotic bands.

Management
Neonatal limb salvage procedures may be required if there is proximal constriction. Amputation may be required. Fetoscopic in utero band release may be indicated in cases where blood flow is abnormal on Doppler, but has significant risk to the mother and fetus [48].

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Part II

The Lower Limb

Clinical Examination of the Hip Joint (Basic and Surface Anatomy) with Special Tests

14

Vidhi Adukia, Kunal Kulkarni, and Dipen K. Menon

Introduction

The hip (femoroacetabular) joint is a ball-and-socket synovial joint comprising of the articulation between the acetabulum and femoral head. It is the primary connection between the lower limb and the axial skeleton. The main functions of the hip joint are to bear the weight of the body during stance and gait. Static stability is provided by the osseous anatomy, alongside the labrum, capsule and ligamentous attachments. Dynamic stability is conferred through the contraction of the various muscle groups around the hip [1].

Anatomy of the Hip

Acetabulum

Named after a Roman 'vinegar cup', this is a hemispherical cavity formed by three bones—the ilium, ischium and pubis. Fusion occurs at the triradiate cartilage, a 'Y'-shaped growth plate, between 12 and 16 years. Radiographically, this point is known as the 'teardrop', which can be identified on an anteroposterior (AP) radiograph of the pelvis (Fig. 14.1). At the lip of the acetabulum, a horseshoe-shaped fibrocartilaginous ring called the labrum serves to deepen the acetabulum and provide a larger articular surface to aid the articulation and prevent dislocation [2]. This attaches to the edge of the transverse acetabular ligament (TAL), which is a useful

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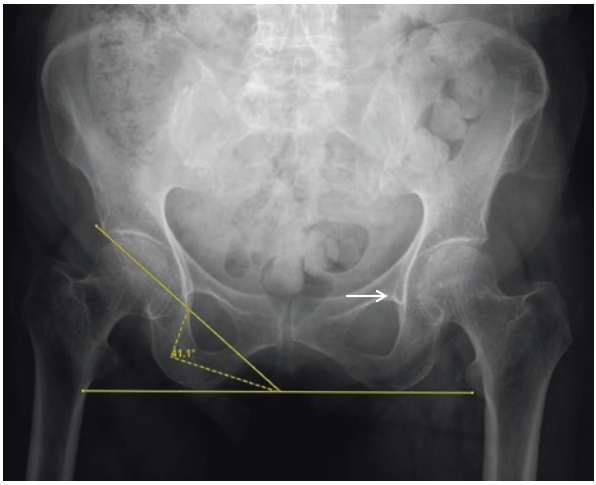

Fig. 14.1 AP radiograph of the pelvis indicating the 'teardrop' (white arrow) and acetabular inclination measuring approximately 41° in this example (yellow lines)

surgical landmark in guiding acetabular component orientation during a total hip arthroplasty (THA). Important reference angles to consider when planning a THA are as follows [3]:

1. Acetabular inclination—measured in the coronal plane; approximately 45°.
2. Acetabular anteversion—measured in the sagittal plane; approximately 15–20°.

Femur

The femoral head is spherical in shape and sits within the acetabulum. Distally, it is connected to the shaft by the neck. The medial portion of the femoral neck is partly formed by the *calcar femorale*, a dense plate of bone that ascends vertically from the posteromedial aspect of the shaft to the neck. This provides structural support and allows for the distribution of stresses from the femoral head to the shaft.

At the lateral aspect of the junction of the femoral neck and shaft, the greater trochanter (GT) projects superiorly, providing attachments to multiple muscle groups. The lesser trochanter (LT) is a projection from the posteromedial aspect of the femoral shaft. The intertrochanteric line, a ridge on the anterior surface of the femoral shaft, connects the two trochanters [2] (Fig. 14.2).

The femoral neck–shaft angle (NSA) usually ranges between 120 and 140° (average 127°) (Fig. 14.2). An increased NSA angle is termed *coxa valga*, whereas a reduced NSA angle is termed *coxa vara*. The femoral neck is itself anteverted by between 15° and 20° in relation to the femoral shaft. Excess anteversion or retroversion must be considered in both lower limb deformity analysis (as it may contribute to more distal signs such as in- or out-toeing gait) or other conditions (slipped capital femoral epiphysis) [3, 4].

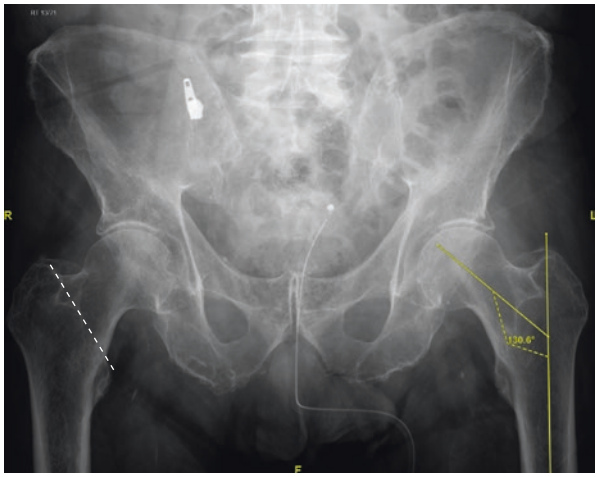

Fig. 14.2 AP radiograph of the pelvis demonstrating the intertrochanteric line (white dashed line) and the neck shaft angle (yellow lines)

Hip Joint Capsule

The capsule consists of circular and longitudinal fibres attached to the acetabulum, labrum and transverse acetabular ligament proximally. Distally, the fibres form a collar around the femoral neck, attaching to the intertrochanteric line anteriorly, and somewhat more proximally on the posterior aspect of the femoral neck.

Ligaments

Four main ligaments surround the hip joint [2]. Three are extracapsular (iliofemoral, pubofemoral and ischiofemoral); one is intracapsular (ligamentum teres). These ligaments play a role in the static stability of the hip.

1. Iliofemoral ligament: Also known as the ligament of Bigelow, this is one of the strongest ligaments in the body. It arises from the anterior inferior iliac spine (AIIS) and acetabular margin. It bifurcates, resulting in an inverted 'Y'-shaped appearance, inserting onto the intertrochanteric line.
2. Pubofemoral ligament: This is a triangular ligament that arises from the superior pubic ramus and obturator crest. It inserts onto the intertrochanteric line, reinforcing the hip capsule anteriorly.
3. Ischiofemoral ligament: Spirals from the ischium towards the posterior aspect of the femoral neck, where it attaches to the GT and merges with the hip capsule. It strengthens the capsule posteriorly.
4. Ligamentum teres: Triangular shaped, arising from the acetabular notch to insert onto a depression on the femoral head called the fovea. Although it confers some stability, its primary function is to provide a conduit for the artery of the ligamentum teres (more relevant in childhood).

Blood Supply

The femoral head and proximal femur have a complex blood supply system, which alters with age. This can broadly be divided into three groups (Fig. 14.3).

1. Intraosseous circulation: This comprises the nutrient artery of the femur, which most commonly arises from the second perforating branch of the profunda femoris. The profunda femoris (deep artery of the thigh) in turn arises from the femoral artery and subdivides into the medial femoral circumflex artery (MFCA), lateral femoral circumflex artery (LFCA) and three perforating branches. The MFCA is the primary supply to the adult femoral head [5]. The perforating arteries (three perforators) are so named because they penetrate the adductor magnus tendon. The nutrient artery is usually a branch of the second perforator or branches from the first and third perforator, if there are two nutrient arteries. The nutrient artery enters the medullary canal through the posterior cortex and then subdivides into ascending and descending blood vessels to form the femoral intraosseous circulation.
2. Retinacular circulation: This is formed by the retinacular arteries, which arise from an extracapsular arterial ring formed by the anastomosis of the MFCA

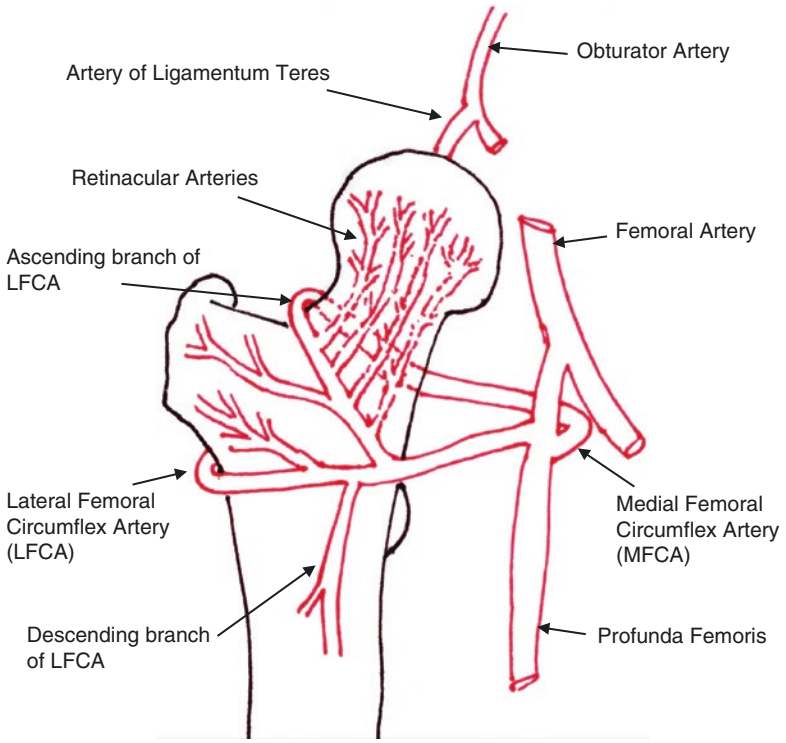

Fig. 14.3 An illustration of the blood supply of the proximal femur (not to scale)

posteriorly and LFCA anteriorly. Whilst the superior and inferior gluteal arteries also provide a small contribution towards this anastomosis, the majority of the blood supply is from the MFCA. The retinacular arteries pierce the hip capsule as they ascend the femoral neck to supply the femoral head, forming a sub-synovial intra-articular ring. An intracapsular neck of femur fracture can therefore result in disruption of this source of blood supply to the femoral head, resulting in osteonecrosis (commonly referred to as 'avascular necrosis', AVN).

3. Artery of ligamentum teres: This is derived from the posterior branch of the obturator artery, but can occasionally arise as a branch of the MFCA. As the name suggests, it travels with the ligamentum teres to supply the femoral head. However, its contribution diminishes with age, and it is therefore a much more important source of blood supply to the paediatric femoral head.

Muscles

There are numerous muscle groups acting on the hip joint, which are detailed in Fig. 14.4 [2].

Biomechanics of the Hip Joint

A functional hip joint is a key component of locomotion, as this joint must support the significant forces that are exerted upon it. For example, supine straight leg raise can result in joint reaction forces (JRF) two times the body weight, whereas running can result in a JRF that is ten times the body weight [6]. The JRF is a biomechanical concept that can help explain how forces are distributed across the joint through a combination of muscle pull and body weight (Fig. 14.5).

Consideration of the JRF plays an important role in understanding and managing pathology of the hip joint; a reduction in JRF can result in a concurrent decrease in a patient's symptoms. Strategies to reduce the JRF can therefore include [7]:

(a) Reducing body weight.
(b) Decreasing the lever arm (B in Figure) by medialising the axis of rotation or using a Trendelenburg gait.
(c) Increasing the abductor lever arm by increasing the offset.
(d) Using a walking stick in the opposite hand.

History Taking and Symptoms

A detailed history is key, as a diagnosis can often be reached from the patient's symptoms alone. Clinical examination and investigations can then be directed towards confirming or refuting the provisional diagnosis. Moreover, the history

Muscle	Origin	Insertion	Blood supply	Innervation	Primary action on hip
Psoas major	Transverse processes of T12–L5	Lesser trochanter	Anterior branch of iliolumbar artery	Ventral rami of lumbar spinal nerves L1–L3	Flexor
Iliacus	Iliac fossa		Iliac branch of iliolumbar artery	Femoral nerve (L2–L3)	
Gluteus medius	Outer surface of ilium between the posterior and anterior gluteal line	Greater trochanter	Superior gluteal artery	Superior gluteal nerve (L4–S1)	Abductor
Gluteus minimus	Outer surface of ilium between the anterior and inferior gluteal line				
Piriformis	Sacrum	Greater trochanter	Superior gluteal artery	Nerve to piriformis (S1, S2)	External rotator
Obturator internus	Ischiopubic ramus		Internal pudendal artery	Nerve to obturator internus (L5, S1)	
Gemellus superior	Ischial spine			Nerve to obturator internus (L5, S1)	
Gemellus inferior	Ischial tuberosity		MCFA	Nerve to quadratus femoris (L4–S1)	
Obturator externus	Obturator membrane	Trochanteric fossa	Obturator and MCFA	Obturator (L3, L4)	
Quadratus femoris	Ischial tuberosity	Trochanteric crest	Inferior gluteal, MFCA	Nerve to quadratus femoris (L5–S1)	
Rectus femoris	Straight head – AIIS Reflected head – Capsule of hip joint	Base of patella via quadriceps tendon	Artery of the quadriceps	Femoral (L2 – L4)	Knee extensor, Hip flexor
Vastus medialis	Intertrochanteric line, linea aspera		Femoral artery		
Vastus intermedius	Anterolateral surface of femoral shaft		Artery of the quadriceps		
Vastus lateralis	Intertrochanteric line, linea aspera		LCFA, artery of the quadriceps, profunda femoris		
Adductor longus	Pubis	Linea aspera	Profunda femoris	Obturator (L2–L4)	Adductor
Adductor brevis					
Adductor magnus					
Pectineus			MCFA	Femoral (L2–3)	
Gluteus maximus	Posterior gluteal line of ilium	Iliotibial tract and gluteal tuberosity	Inferior gluteal artery	Inferior gluteal (L5–S2)	Extensor
Semitendinosus	Ischial tuberosity	Medial proximal tibia	Perforating arteries	Sciatic (L5–S2)	
Semimembranosus		Horizontal line on posterior aspect of medial tibial condyle			
Biceps femoris	Long head–Ischial tuberosity Short head–Linea aspera	Head of fibula and lateral tibial condyle			

Fig. 14.4 Different groups of muscles acting on the hip joint

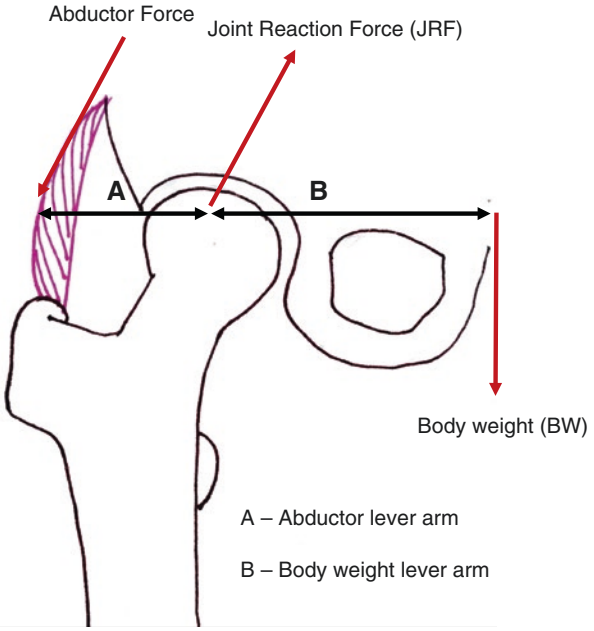

Fig. 14.5 Joint reaction force on the hip joint

will illustrate exactly how patient's symptoms are affecting their quality of life and help guide the management plan.

Patient Reported Outcome Measures

In recent years, particularly with the global advent of national arthroplasty joint registries, there is a growing emphasis on patient reported outcome measures (PROMS), following major joint replacement. The Oxford Hip Score (OHS) is a 12-question (maximum score 48, with 48 being best and 0 being worst) validated and commonly utilised questionnaire designed to ascertain the impact of hip pain on a patient's quality of life (QoL) [8]. A low score is strongly suggestive of hip disease severely affecting QoL, and these patients are therefore more likely to benefit from surgical intervention. The score can be repeated after such an intervention (e.g. a THA) to demonstrate any improvement or deterioration in symptoms.

Hip Pain

This is the most common symptom that patients present with. The location of the pain can be varied, ranging from the ipsilateral groin to the thigh, buttock, GT and knee. Buttock and thigh pain could be referred from coexisting lumbar spine pathology and present as 'hip pain'. Similarly, knee pain could be secondary to underlying knee OA or be referred from the hip joint above (via the obturator nerve).

Hip Pain History		
	Component	Hip relevance
S	Site	Where is the pain located (i.e. groin–Hip joint; GT–Bursitis)
O	Onset	What starts the pain (e.g. activity in young patient–Femoro-acetabular impingement; start-up pain with existing hip replacement – Loosening)
C	Character	History of trauma Sharp, burning, ache (mechanical vs neurological)
R	Radiation	Does it spread anywhere (e.g. buttock/leg–Co-existing spinal pathology; thigh–OA, loose arthroplasty femoral component; knee–Co-existing knee arthritis)
A	Associated symptoms	Weakness (sciatic nerve injury), dysesthesia, fevers (infected joint) Is it getting better or worse
T	Temporality	Any pain in childhood pain (dysplasia, SCFE, Perthes sequelae) Movements make it worse (e.g. psoas tendon - resisted flexion; internal rotation–Osteoarthritis) Rest pain Associated stiffness
E	Exacerbating and relieving factors	Worse towards end of the day Anti-inflammatories make it better Functional assessment: Able to climb stairs/ use transport /wash/dress independently/ do shopping/job/housework
S	Severity	Visual Analogue Scale: 0 to 10 (0; no pain, 10; worst imaginable pain)

Fig. 14.6 Pain history using the mnemonic 'SOCRATES'

Hip pain, especially when secondary to osteoarthritis (OA), tends to be exacerbated by movement, relieved with rest and is often associated with varying degrees of stiffness. Inflammatory arthritides commonly exhibit morning stiffness and more symmetrical patterns. Patients often complain of the pain gradually worsening over time that is accompanied by a steady decline in their ability to carry out their activities of daily living.

The mnemonic 'SOCRATES' can be helpful in ensuring that a thorough pain history is taken (Fig. 14.6). In conjunction, note the patient's analgesic requirements. In addition, patients need to be asked about any history of trauma, diseases

affecting the hip in childhood and any previous treatment/surgery. Remember to ask about any walking aids or adaptations. Relevant past medical history is important in assessing risk prior to considering any intervention.

Stiffness

Hip stiffness is common and often accompanies pain. It is often worse first thing in the morning, tending to ease with activity. Alternatively, patients may complain of difficulty getting in and out of cars or chairs or bending down (deep hip flexion) to put their shoes on. This occurs due to numerous reasons; for example, loss of joint space and osteophyte formation in OA results in the femoral head impinging on the acetabulum during hip movements. Furthermore, inflammation around the hip capsule causes contractures or fibrosis, resulting in hip stiffness.

Limp

This often occurs due to hip pain and stiffness. Pain causes patients to mobilise with an antalgic gait, which helps them reduce the JRF on the hip joint by reducing the time spent in the stance phase of gait on the affected side. Stiffness often results in an arthrogenic gait in which the pelvis on the affected side elevates significantly and is associated with a circumduction movement of the leg to clear the ground during the swing phase of gait. Similarly, a Trendelenburg gait (where the pelvis sags on the unaffected side) is observed by the healthy 'lifted leg lagging' due to abductor muscle weakness on the contralateral affected side, forcing patients to lean towards the affected side to maintain their balance (abductor lurch).

Deformity

Deformities in the hip can be masked by compensatory tilting of the pelvis. A fixed flexion deformity of one hip can be compensated by increased forward pelvic tilt resulting in an increase in the lumbar lordosis in the spine (sagittal plane compensation). Similarly, fixed adduction and abduction deformities of the hip can be compensated by pelvic tilt in the coronal plane resulting in apparent shortening or lengthening of the affected leg respectively. These deformities can be unmasked by performing special tests that will be discussed in detail later in this chapter.

Swelling

Swellings in and around the hip joint are difficult to detect due to the deep seated location of the hip joint. However, occasionally patients might present with lumps

around the trochanter or groin that could be indicative of an underlying abscess or tumour. Do not forget alternative (non-orthopedic) pathologies such as herniae, which may also present with groin pain!

Examination of the Hip Joint

This follows the basic sequence of 'look, feel and move', with a few additional special tests that can be applied to the examination of any joint. Practice is key to ensuring a slick and efficient examination, which is especially important for those preparing for postgraduate examinations. It is mandatory to keep the patient comfortable and avoid causing undue pain or distress—remember to look at the patient's face at all times and clearly explain every step. Adequate exposure of the lower back, down to the feet, is required (underwear may be kept on), albeit taking care to preserve the patient's dignity with the judicious use of sheets. A chaperone should be offered for examinations if appropriate [9].

Look

As discussed earlier, gait can be significantly altered in patients with underlying hip disease and should therefore be commented upon. Patients can try to alleviate their symptoms by using walking aids and/or a shoe raise, which should be noted.

The patient should then be asked to expose the hip and the knee joint, at which time leg length discrepancy, muscle wasting, thigh crease asymmetry, scars (indicating previous surgery or infections), swellings, erythema and sinuses may be observed. It is important to examine the patient from the front, side and back to ensure that such abnormalities are not missed. Remember that some scars may be well hidden, including beneath the underwear (e.g. pelvic surgery in the groin or the scar of a medial hip approach in the inner thigh).

Feel

Palpation of the greater trochanter may elicit tenderness indicating underlying trochanteric bursitis. Similarly, palpation around the groin may cause some tenderness; however it is difficult to truly palpate the hip joint simply due to its deep location.

Move

Active and passive hip movements should be checked both on the affected and unaffected side with the patient supine and include hip flexion, abduction, adduction, internal rotation and external rotation. Hip extension is best tested with the patient

Normal ranges of motion for the hip joint	
Flexion	0 – 125°
Extension	0 – 15°
Abduction	0 – 45°
Adduction	0 – 30°
External rotation	0 – 45°
Internal rotation	0 – 35°

Fig. 14.7 Approximation of normal ranges of motion for the hip joint

prone, although this is not routinely tested in the older patient in whom hip OA is suspected. Internal and external rotation of the hip can be tested with the hip extended or flexed. Figure 14.7 shows the normal ranges of motion of the hip joint. It is important to remember that age, sex and ethnicity can also have an impact on what the 'normal' range of movement is for the hip. Normal range of hip movement decreases with age, especially in men, and studies have shown that Asian populations can have an increased 'normal' range of motion of the hip joint for external and internal rotation. In patients with severe hip OA, internal and external rotation especially may be significantly limited by pain and stiffness.

Special Tests

1. Trendelenburg Test

   This test is performed to assess a patient's abductor mechanism [10]. The examiner should stand facing the patient, placing his/her hands on the patient's iliac wings with the thumbs on each anterior superior iliac spine ASIS (right thumb on the patient's left ASIS and the left thumb on the patient's right ASIS: see reason below). The patient needs to hold onto the examiner's forearms. The patient should then flex their knee in order to stand on one leg, first on the affected side, followed by the unaffected side. The abductors on the weight-bearing side will elevate the contralateral hemipelvis in order to maintain balance as shown in Fig. 14.8. The test is considered to be positive; i.e. the patient has a weakened abductor mechanism, when the pelvis on the unaffected side drops ('lifted leg lags') with the patient standing on the affected leg due to contralateral abductor weakness. This rise and fall of the hemipelvis can often be quite subtle, and we therefore advocate the examiner also places their hands lightly on both anterior superior iliac spines (ASIS) of the patient in order to improve the accuracy of the test (Fig. 14.9).
2. Leg length Measurement

   Hip disorders such as OA, SCFE or developmental dysplasia of the hip (DDH) can result in significant leg length abnormalities, which can have an impact on the surgical management of the disorder. It is important therefore to delineate the presence of leg length discrepancies, along with where the shortening has occurred.

Fig. 14.8 Clinical photograph showing the Trendelenburg's test in a patient with normally functioning hip abductors. The white dashed line represents the line joining the two ASIS. The picture on the right demonstrates the rising hemipelvis as the patient is asked to weight-bear on a single leg. In patients with weak abductors, when asked to bear weight on the affected leg, the hemipelvis on the 'sound' side will sag

With the patient lying supine on the examination couch, the examiner should place a forearm along both the patient's ASIS, ensuring that their forearm is perpendicular to the side of the couch (to ascertain that the pelvis is square) (Fig. 14.10). The next step is to measure the distance between each ASIS and ipsilateral medial malleolus to determine the true leg length (Fig. 14.11).

Any discrepancies in the true leg lengths should prompt the examiner to move onto Galeazzi's test in order to determine if the shortening is in the femoral or tibial segment of the affected leg (Fig. 14.12). For this, the patient is asked to flex their knees to 90° with both heels equidistant from the buttocks and the legs in contact with one another. Proximal positioning of the knee on the affected side indicates femoral shortening, whereas an inferior positioned knee indicates tibial shortening of the affected limb.

If the shortening is due to a femoral abnormality, further tests can be carried out to determine if the abnormality is proximal, i.e. above the level of the GT, or whether the abnormality lies in the femoral shaft below the GT.

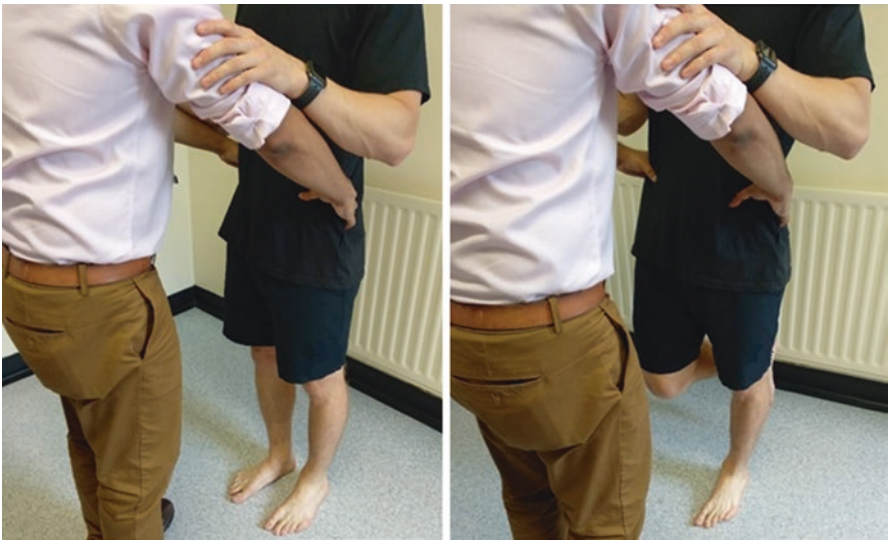

Fig. 14.9 Clinical photograph showing Trendelenburg's test with the examiner placing their arms lightly on the patient's ASIS bilaterally in order to feel the rise of the hemipelvis as the patient weight-bears on one leg

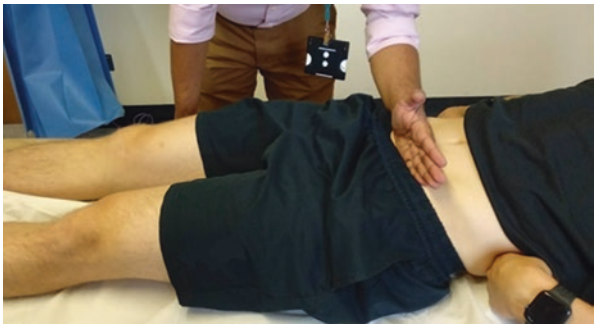

Fig. 14.10 The pelvis is squared using the examiner's forearm to line up both ASIS

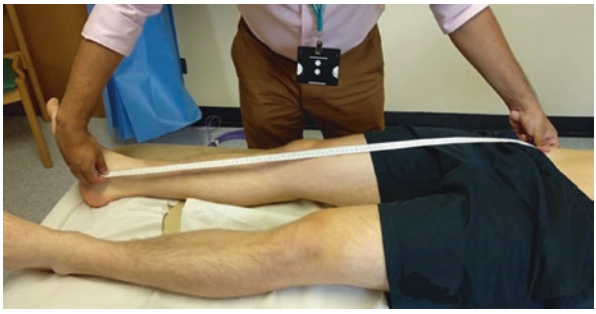

Fig. 14.11 Measurements are taken from the ASIS to the medial malleolus with a tape measure for each limb to determine the true leg length

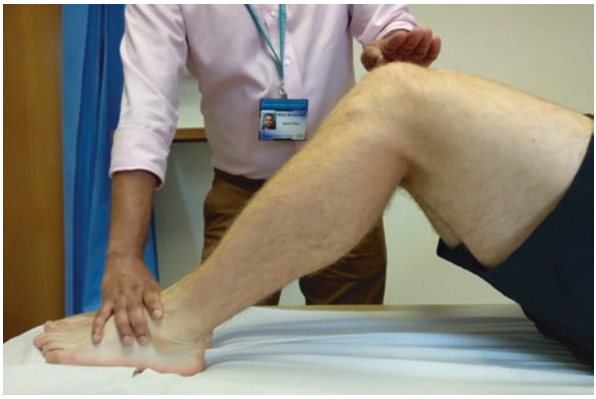

Fig. 14.12 Clinical photograph showing Galeazzi's test

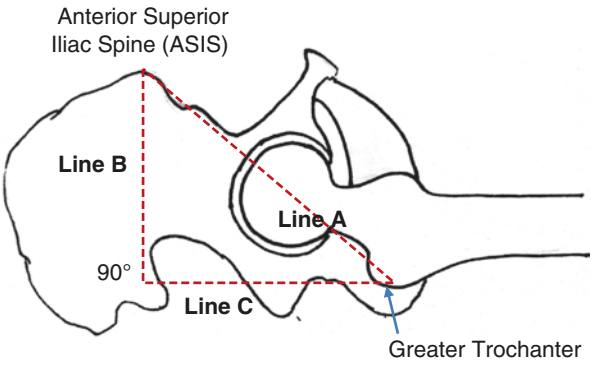

Fig. 14.13 Line diagram showing Bryant's triangle

One such test is drawing the iliofemoral triangle, also known as Bryant's triangle, with the patient supine, as illustrated in Fig. 14.13. The base of this triangle is formed by a line joining the ASIS and the top of the greater trochanter (line A). The two sides of the triangle are made up of a line dropped down perpendicular to the bed from the ASIS (line B), with the third line perpendicular to line b, going down to the greater trochanter (line C). Bryant's triangle is drawn on both the affected and unaffected sides, and the lengths of line C on the two sides are compared. Shortening on one side indicates that the femur has migrated proximally due to disease in the hip joint.

As this test compares the affected limb with the contralateral side, it cannot be used in patients who present with bilateral hip disorders. In these cases, Nelaton's line can be used (Fig. 14.14). This is a line that is drawn from the ischial tuberosity to the ASIS. The position of the greater trochanter is then noted in relation to this line. In patients without hip disease, the greater trochanter will lie on the line or distal to it, whereas in patients with femoral shortening due to underlying hip pathology, the greater trochanter will lie proximally, indicating migration of the femur proximally. In clinical practice, plain radiographs of the pelvis and femur are used to determine any leg length discrepancies.

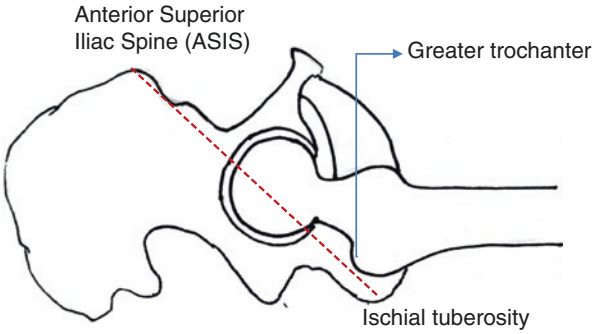

Fig. 14.14 Line diagram showing Nelaton's line

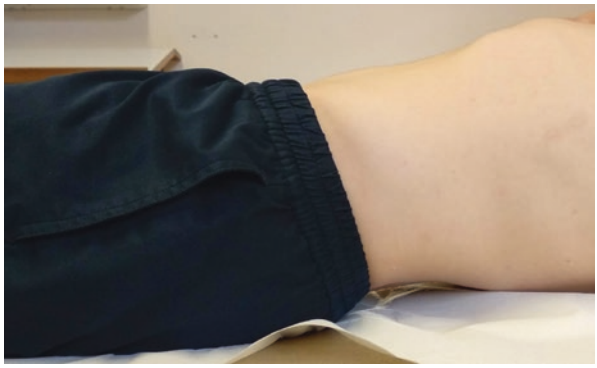

Fig. 14.15 Clinical photograph showing the lumbar lordosis present when the patient lays flat on the examination couch

3. Thomas' Test

   Patients with underlying hip disorders can develop a fixed flexion deformity (FFD), which can be difficult to elicit due to the patient compensating with a forward tilt of the pelvis and a resultant increase in lumbar lordosis. Thomas' test is performed to determine the degree of FFD present in the affected hip joint. With the patient supine, the examiner should place a hand underneath the patient's lower back, at the level of the lumbar lordosis (Fig. 14.15), and ask them to bring the unaffected leg close to their chest, by fully flexing at the hip and knee joint. This should result in complete obliteration of the lumbar lordosis (Fig. 14.16). The examiner should then inspect the straight (affected) limb to see if there is any residual flexion at the hip joint, indicating an FFD. This can then be repeated for the opposite leg.

4. Anterior Impingement Test

   This test, as the name suggests, is used to determine the presence of underlying intra-articular hip disease. The examiner should flex the affected hip to 90° with the patient supine. The hip is then passively adducted and internally rotated in this position of 90° of flexion (Fig. 14.17). The test is positive if the movement combination results in the patient experiencing pain in the groin. Classically, the FABER (flexion, abduction and external rotation) manoeuvre can be used to elicit femoro-acetabular impingement (FAI).

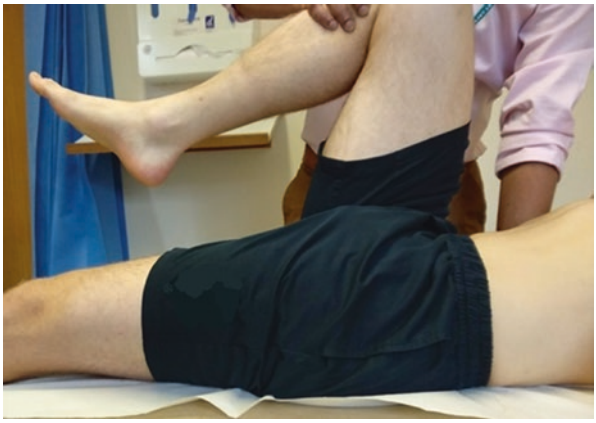

Fig. 14.16 Clinical photograph showing the obliteration of the lumbar lordosis as the patient flexes their hip and knee. The examiner checks this by placing their hand underneath the lumbar lordosis of the patient to ensure complete obliteration. Any flexion of the contralateral hip would be due to a fixed flexion deformity

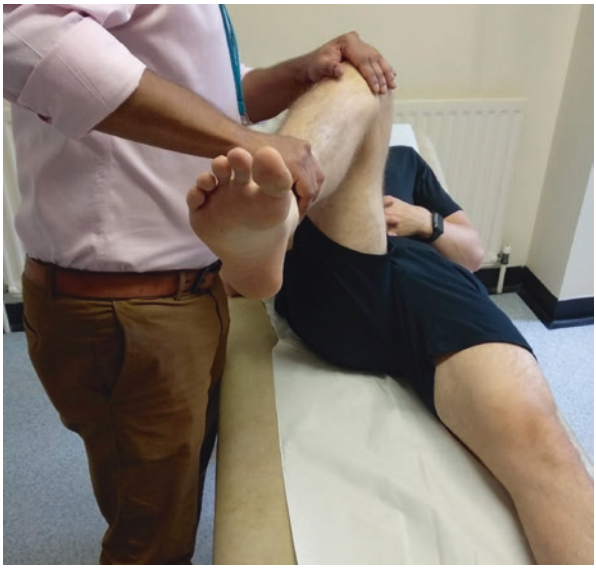

Fig. 14.17 Clinical photograph showing the anterior impingement test which involves hip flexion, adduction and internal rotation (FADIR)

Examination of the hip joint should always be completed by a) performing a neurovascular examination of the limb and b) examining the joint above (spine) and below (knee). Overall assessment of lower limb alignment is also of importance in practice, particularly when considering lower limb deformity. Figure 14.18 summarises the examination of the hip joint and the sequence in which we recommend that the tests be carried out.

Special Tests in the Paediatric Patient

The Ortolani and Barlow tests are special tests used most commonly to exclude DDH in the paediatric patient. In the UK, these tests are performed in the newborn child as part of a screening programme for DDH. They are not useful after the age

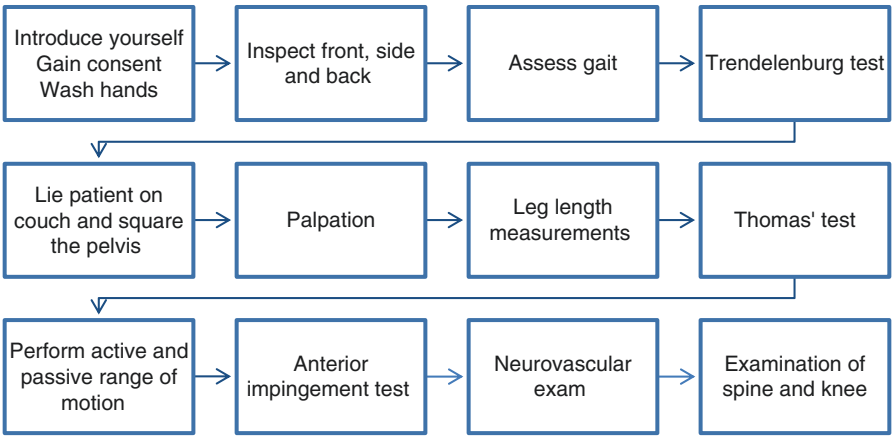

Fig. 14.18 Flowchart summarising the hip examination

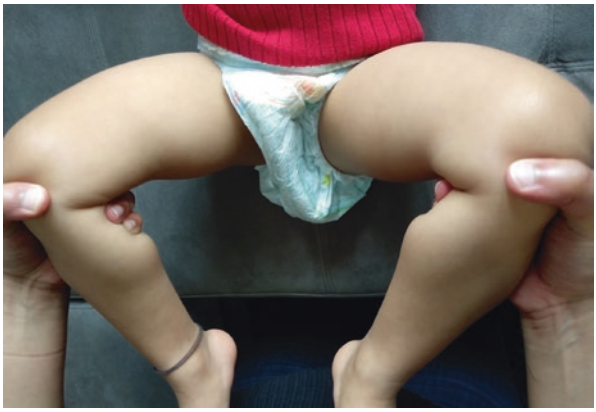

Fig. 14.19 Clinical photograph showing the Ortolani's test. The examiner places his/her fingers over the posterior aspect of the hip, with the thumbs anteriorly whilst attempting to reduce the dislocated hip

of 3–6 months, as hip contractures develop in the older patient which make eliciting the 'clunk' and the 'click' of the Ortolani's and Barlow's tests difficult. In this older age group, leg length discrepancy and loss of abduction are more helpful findings.

Ortolani's Test

This test attempts to reduce a dislocated hip and is performed with the newborn baby supine. The examiner passively flexes both the patient's hips to 90° and then abducts the hips all the whilst applying a gentle force over the hips, attempting to push them anteriorly and into the acetabulum (Fig. 14.19). The test is positive when a clunk is palpated, as the dislocated hip moves over the neolimbus and reduces into the acetabulum.

Barlow's Test

This test is performed after a positive Ortolani's test to check whether the hip is unstable and can be re-dislocated. With the newborn baby supine and the hips held in 90° flexion, the examiner adducts the hips and applies a gentle depressing force, attempting to dislocate the hips posteriorly (Fig. 14.20). The test is positive when the hip re-dislocates with a 'click'.

Investigations

Blood Tests

Following history taking and examination, basic investigations are conducted. Blood tests can be helpful in evaluating certain underlying pathologies. For patients with OA due to undergo arthroplasty, routine blood tests, including full blood count (haemoglobin, renal function, clotting), are a useful preoperative baseline. Depending upon local hospital policy, the patient may also require a group and save blood test prior to surgery.

If septic arthritis is the suspected diagnosis, in addition to the above blood tests, a C-reactive protein test (CRP) and erythrocyte sedimentation rate (ESR) are helpful. CRP is a reliable marker when monitoring the response of a patient to the treatment of an infection.

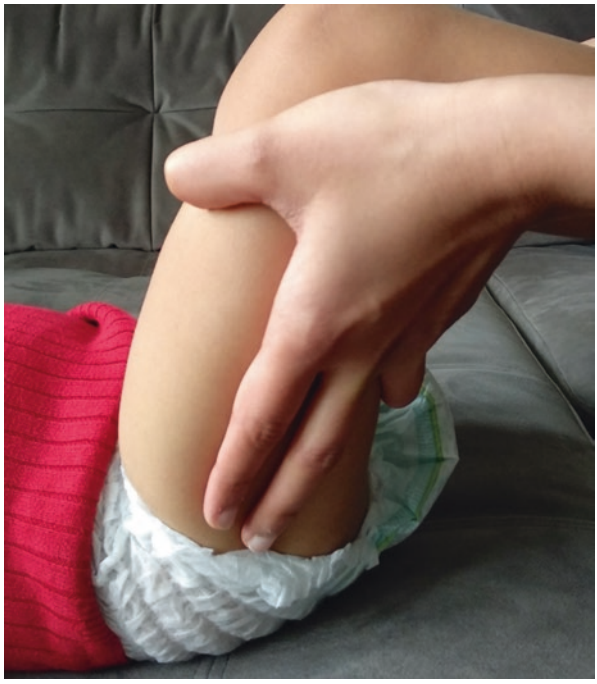

Fig. 14.20 Clinical photograph showing Barlow's test. This is the exact opposite of the Ortolani's test in essence. The examiner places his/her fingers over the posterolateral aspect of the flexed hip in the supine patient, with the thumbs over the proximal thigh anteriorly, whilst gently adducting and depressing the hip in order to re-dislocate the joint

Other blood tests used in patients presenting with hip pathologies also include adjusted serum calcium and phosphate levels, rheumatoid factor and serum electrophoresis, which are mainly used to screen for metabolic and neoplastic hip disorders.

Radiographs

Plain radiographs of the pelvis are usually the first line of imaging conducted for patients presenting with symptoms around the hip joint (Fig. 14.21). Commonly, anteroposterior (AP) pelvis and lateral views (frog lateral views in the paediatric patient) are obtained for the majority of hip pathologies, but special views such as Judet views may be helpful if the patient presents with a history of trauma and fractures of the acetabulum are suspected. Advantages of this imaging modality are that it is readily available, inexpensive and associated with low radiation exposure. However, a plain radiograph is a two-dimensional representation of a three-dimensional structure; if a patient remains symptomatic with a normal pelvic radiograph, then further investigations should be considered.

Ultrasound Scan (USS)

This is commonly used for paediatric patients with suspected DDH or septic arthritis. In infants below the age of 6 months, ultrasound is the mainstay of hip imaging for DDH. In infants and children above this age, an X-ray maybe more helpful, as the ossific nucleus appears. USS can demonstrate hip joint effusion that would raise the suspicion of an underlying hip disorder (including septic arthritis). It confers no radiation risk. However, it is an operator-dependent modality (due to inter-observer

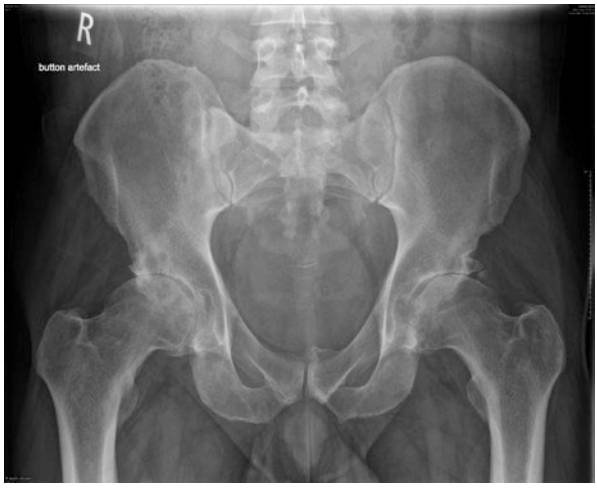

Fig. 14.21 Plain AP radiograph of the pelvis which typically includes both hips and sacroiliac joints. This radiograph demonstrates bilateral hip osteoarthritis (narrowing of joint spaces, osteophytes, subchondral sclerosis and cysts)

variability) and could be difficult to access in individual hospitals due to the lack of adequately trained personnel.

Bone Scintigraphy

Commonly referred to as a 'bone scan', scintigraphy of the hip joint involves the use of a radioactive tracer such as technetium or gallium, which is injected intravenously into the patient, after which images are taken using a gamma camera (Fig. 14.22). Uptake of the tracer tends to be directly proportional to osteoblastic activity, and therefore bone scans can help diagnose multiple conditions such as infections, fractures and osseous metastases. Whilst a bone scan is a sensitive test, it is not very specific and therefore the underlying diagnosis needs to be made on the basis of the history and examination taken by the clinician [11]. The disadvantage with a bone scan is its significant radiation exposure (6.3 mSv approximately: equivalent to about 120 chest X-rays).

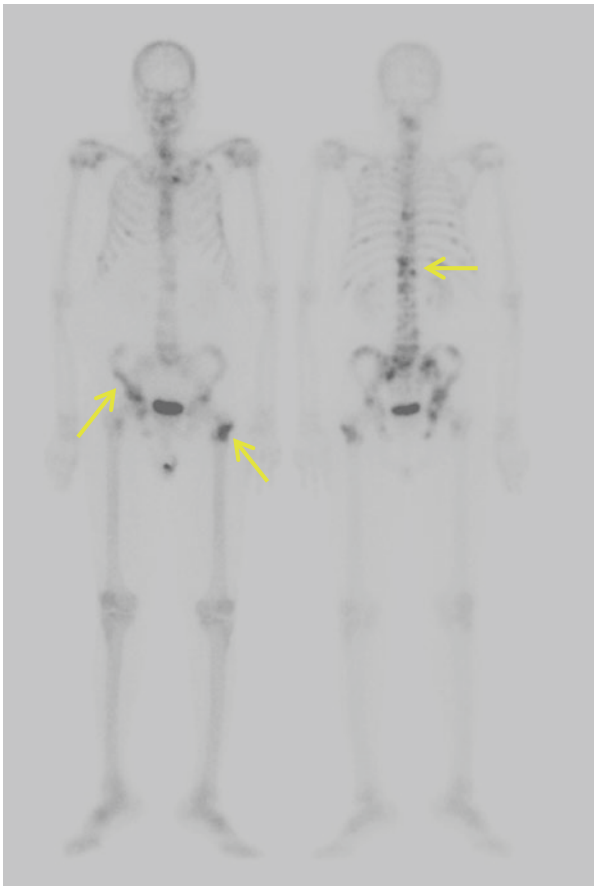

Fig. 14.22 A whole body bone scan in a patient diagnosed with prostate cancer demonstrating metastases to the spine, right ilium and left proximal femur (yellow arrows)

Computed Tomography (CT)

CT is an investigative tool that produces computer-generated cross-sectional images using multiple X-rays (Fig. 14.23). Currently, CT scans have become essential in everyday clinical practice. They are used routinely in trauma cases to demonstrate fractures, especially those involving the pelvis or acetabulum, as well as in the staging of cancers and monitoring response to treatment. Furthermore, three-dimensional reconstructive images obtained from modern day CT scanners can be used for preoperative planning of complex fracture patterns [12]. However, the disadvantage is that the patient is exposed to a higher dose of radiation, and therefore CT scans are associated with an increased lifetime relative risk of cancer [13]. It is particularly important to reduce the use of CT scans when possible, in infants and children.

Magnetic Resonance Imaging (MRI)

Another form of cross-sectional imaging used routinely in orthopedics is MRI. MRIs are useful in depicting soft tissue structures and bony oedema and can therefore help diagnose and stage multiple conditions such as AVN of the femoral head, SCFE and Perthes' disease [14]. They are even used to detect fractures of the femoral neck that are not visible on CT scanning (MRI is the gold standard for clinically suspected fractures with normal looking X-rays). Moreover, MRIs are better than CT scans in demonstrating intramedullary lesions and are therefore used to grade tumours around the hip joint. Whilst MRI scans do not expose the patient to any radiation, they are expensive and not as easily accessible as CT scanning.

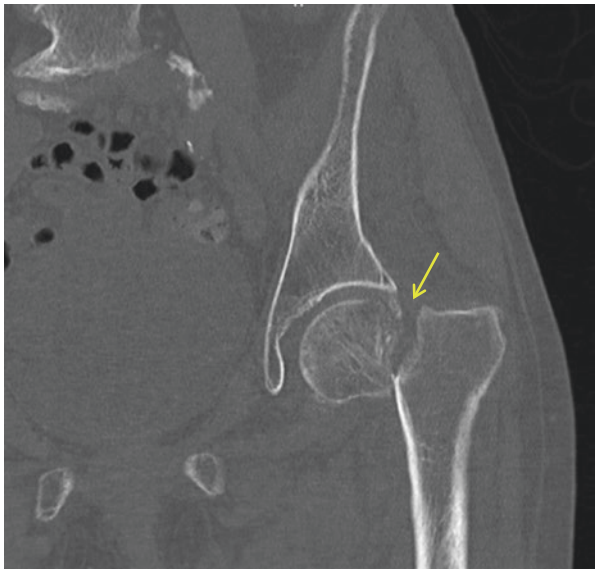

Fig. 14.23 A CT scan demonstrating a displaced left, intracapsular neck of femur fracture (yellow arrow)

Arthrogram

Commonly performed to investigate intra-articular hip pathologies such as femoroacetabular impingement (FAI) and labral tears, arthrograms are invasive procedures compared to CT and MRI scanning. They are two-stage procedures, usually involving the injection of contrast or air into the hip joint, followed by imaging in the form of either fluoroscopy, CT or MRI. Arthrograms allow the investigator to obtain both static and dynamic images, and this is useful in diseases such as Perthes' disease and FAI [15].

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The Hip Joint

15

Vidhi Adukia, Kunal Kulkarni, and Dipen K. Menon

Paediatric Disorders of the Hip

Developmental Dysplasia of the Hip (DDH)

DDH encompasses a spectrum of developmental disorders of the hip, ranging from acetabular deficiency to proximal femoral abnormalities. These result in dysplasia, subluxation and/or dislocation of the hip joint [1].

Epidemiology: The incidence of DDH ranges from 1:100 (dysplasia) to 1:1000 (dislocation). The incidence is lower in African Americans (possibly due to anatomically deeper acetabulae) but higher in cultures where infants are swaddled with the hips extended. DDH is more common in females (possibly due to greater ligamentous laxity) and tends to affect the left hip more (probably due to the commoner left occiput anterior intrauterine foetal position). Other risk factors associated with DDH are listed in Fig. 15.1 [1].

Pathophysiology: DDH is thought to arise due to a combination of maternal and foetal capsular laxity, genetics and postnatal malpositioning. Greater laxity allows the proximal femoral head to sublux out of the still developing, fairly shallow acetabulum. However, this alone does not explain the other associations of DDH (such as breech presentation or the left hip being affected more). A combination of adverse anatomical and biochemical factors may therefore also need to be present. Restricted foetal movement (seen in multiple pregnancies, increased birth weight, increased muscle tone in the uterus for the firstborn child), direct pressure from the sacrum,

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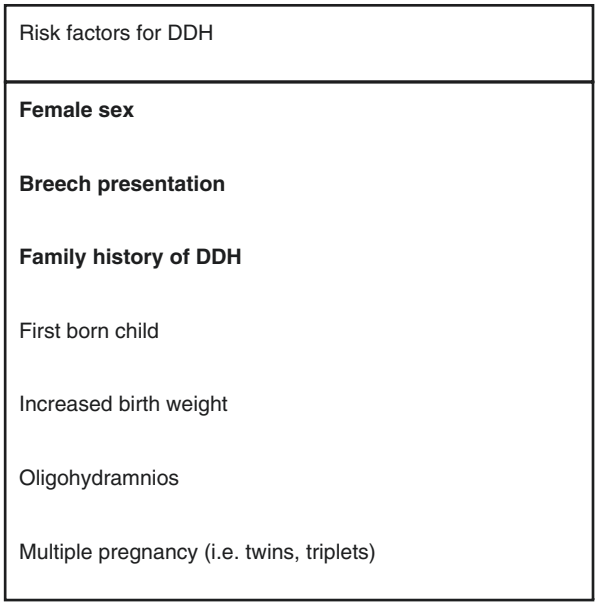

Fig. 15.1 Risk factors associated with DDH (bold = most important)

postnatal malpositioning and increased levels of the maternal hormone relaxin (induces ligamentous laxity) have all been implicated in DDH.

Pathoanatomy: The acetabulum relies on the spherical femoral head for its development. Eccentric pressure applied by the femoral head can result in the formation of a pathological lesion in the acetabulum called a neolimbus, a ridge of hypertrophied cartilage in the superolateral articular surface of the acetabulum. When the femoral head moves over this ridge and subluxes out of the acetabulum, it produces a 'clunking' sensation known as 'Ortolani's sign' [2]. Furthermore, recurrent subluxation of the femoral head leads to the development of a pseudoacetabulum with a hypertrophied ligamentum teres and pulvinar, along with iliopsoas shortening and capsular constriction, all of which act as barriers to the reduction of the hip joint, and can be identified on a hip arthrogram.

Screening programmes: Several countries adopt risk-based neonatal screening programmes for DDH, with expert consensus (albeit limited evidence) suggesting early diagnosis and treatment can prevent the development of irreversible anatomical changes that are likely to require surgical intervention. There is, however, significant debate surrounding the concept of 'universal' screening, as the natural history of the disease is that, in more 'physiological' cases, it stabilises within the first 3 months of life in over 85% of cases. However, there is currently no way of knowing whether an infant falls into this group, and so the criticism of universal screening is that such strategies result in a number of children being over treated and being exposed to the risks associated with DDH treatment (e.g. AVN, neurological injury).

Clinical presentation: The majority of patients with DDH are identified through screening programmes, such as neonatal birth checks, with clinical findings including asymmetrical buttock/upper thigh creases, leg length discrepancy (including

Galeazzi for unilateral or Klisic tests for bilateral dislocations), reduced abduction and positive Barlow and Ortolani's tests (although the latter are only reliable in children below the age of 6 months). A minority present later, with reduced abduction, or altered gait due to leg length discrepancy (e.g. toe walking).

Investigations: Depending on the age of the child, ultrasound (below 6 months) or plain radiographs of the hip (above 6 months, once the femoral head ossific nucleus is visible) can be used to confirm the diagnosis of DDH. Ultrasound measures the angles between the bony acetabulum and ilium (alpha angle) and the labrum and ilium (beta angle) and can be used to stage severity, for example, with the Graf classification. A decreased alpha angle and increased beta angle suggest DDH. Moreover, the ultrasound can also be used to make a dynamic assessment through the direct visualisation of femoral head dislocating from the acetabulum during Barlow's test. Figures 15.2 and 15.3 demonstrate the key lines and angles on the anteroposterior (AP) radiograph of the pelvis that are used to help diagnose DDH.

(a) Hilgenreiner's line—This is a horizontal line connecting the two tri-radiate cartilages (white dashed line). In normal cases, this line passes superior to the femoral head ossification centre.
(b) Perkin's line—This is a vertical line passing through the lateral margin of the sourcil of the acetabulum (yellow dashed line). It lies perpendicular to Hilgenreiner's line. In normal cases this line is lateral to the ossification centre of the femoral head. Together with Hilgenreiner's line, four quadrants are formed; the femoral head ossific nucleus should lie in the infero-medial quadrant.
(c) Acetabular index—This is the angle (Angle 1) formed by Hilgenreiner's line and a line drawn along the margins of the acetabular sourcil (red dashed line). It varies with age, but on average should be less than 25° in patients over the age of 6 months.

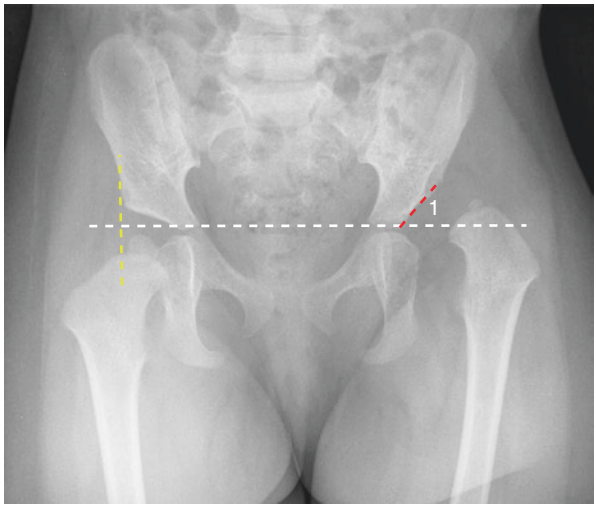

Fig. 15.2 AP pelvis radiograph demonstrating the key lines and angles used in the diagnosis of DDH. In this example, the patient has developmental dysplasia of the left hip

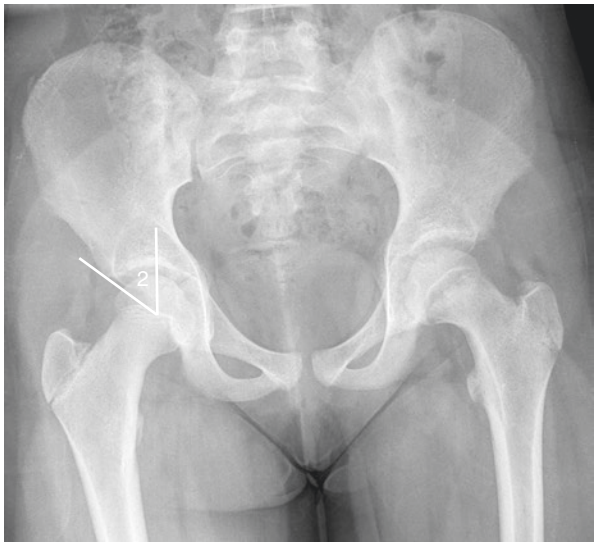

Fig. 15.3 AP pelvis radiograph demonstrating the centre-edge angle of Wiberg used in the diagnosis of DDH in an older patient

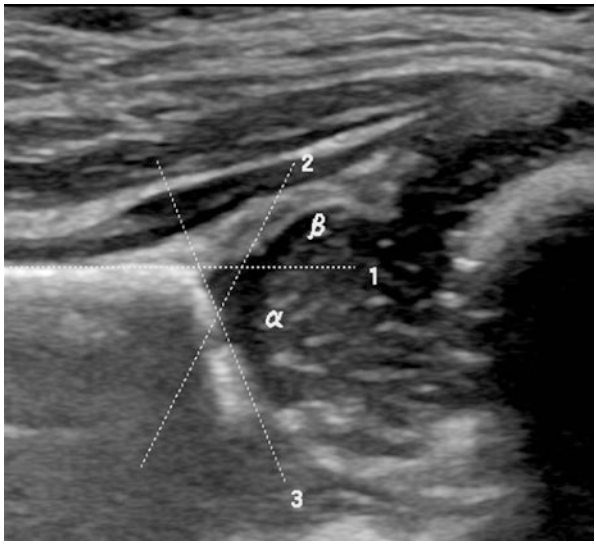

Fig. 15.4 An ultrasound of a patient's left hip demonstrating the alpha (α) and beta (β) angles. *Line 1*, ilium; *line 2*, labrum; *line 3*, acetabulum

(d) Centre-edge angle of Wiberg—This is the angle formed by a line parallel to Perkin's line, passing through the centre of the femoral head and a line drawn from the centre of the femoral head to the lateral edge of the acetabulum (Angle 2). It is useful after the age of 5 years, when an angle of less than 20° is considered abnormal [3].

Classification: Depending on the alpha and beta angles obtained from the hip ultrasound (Fig. 15.4), the Graf Classification for DDH divides patients into four main groups (Fig. 15.5). This can then be used to guide management [4].

Type	Alpha angle	Beta angle	Type of hip	Treatment
Ia	> 60°	< 55°	Normal	Nil
Ib	> 60°	> 55°	Still normal, good coverage of the femoral head	Nil
IIa	50 – 59°	55 – 77°	Immature	Observation
IIb	50 – 59°	55 – 77°	Delayed ossification	Pavlik harness
IIc	43 – 49°	< 77°	Dysplastic	Pavlik harness
D	43 – 49°	> 77°	Decentring, unstable	Pavlik harness / hip spica
III	< 43°	> 77°	Subluxed	Pavlik harness / hip spica
IV	< 43°	unmeasurable	Dislocated	Closed / open reduction

Fig. 15.5 Graf classification of DDH

Treatment: The goal of treatment is to reduce the hip and then to maintain a stable reduction to allow the acetabulum and proximal femur to develop normally. Non-operative management includes using the Pavlik harness and hip spica. First described by Arnold Pavlik in the 1940s, the Pavlik Harness is used to keep the hips in a flexed and abducted position. It has an over 80% success rate for treating DDH, with only a small percentage of patients having disease progression and requiring surgical management. Opinion remains divided, but infants generally need to be in the harness for anywhere between 3 and 9 months, with regular ultrasound follow-up to ensure that the hip joint remains reduced. However, using the Pavlik harness is not without complications and can be associated with femoral nerve palsy and osteonecrosis of the femoral head, especially if applied incorrectly. Moreover, it is mainly indicated for the treatment of patients under the age of 6 months, as it is difficult to maintain in the older, more active child, where closed reduction and application of a hip spica may be more appropriate [5]. The hip spica is based on a similar principle of keeping the hip in a flexed and abducted position in order to maintain hip joint reduction. Closed reduction of the hip joint is performed under general anaesthetic (often preceded by an arthrogram to identify barriers to reduction), followed by application of a hip spica cast. An adductor tenotomy is also

commonly performed to increase Ramsey's 'safe zone' range of stable hip abduction, with the hip placed into abduction in the middle of this range to minimise the risks of failed reduction and neurovascular complications.

Failure of non-operative treatment or late presentation often necessitates open reduction of the hip joint, sometimes in association with a pelvic or femoral osteotomy. Femoral derotational osteotomy and shortening helps achieve reduction of the hip joint, whereas a pelvic osteotomy (e.g. a Salter or Dega) is performed to ensure sufficient acetabular coverage of the femoral head while the child is still growing so that remodelling can take place. The goal of these treatments is to extend the age at which the child will develop degenerative changes as an adult [6].

Slipped Capital Femoral Epiphysis (SCFE)

This is a condition affecting the proximal femoral physis, commonly during the adolescent growth phase. The name is a misnomer; the femoral epiphysis does not slip—rather, this remains enlocated within the acetabulum while the metaphysis slips anterosuperiorly and externally rotates in relation to the epiphysis [1].

Epidemiology: The incidence of SCFE is around 10:100,000. Unlike DDH, it is two to three times more common in males than in females, with patients presenting around the age of 12–13 (i.e. at puberty). In approximately 20–50% of cases, the disease is bilateral. It is more common in African Americans and Hispanics. SCFE tends to affect the left hip more than the right. There is also a clear association between obesity and the rate of SCFE, with a rise in prevalence of SCFE reflecting the growing childhood obesity epidemic. Other conditions that are implicated in SCFE are hypothyroidism, metabolic bone disease (e.g. rickets) and radiotherapy treatment; endocrine abnormalities should always be considered in bilateral SCFE [7].

Pathoanatomy: It is postulated that the susceptible adolescent physis is less resistant to the shear forces acting on the proximal femur. This 'slippage' occurs through the zone of hypertrophy in the physis, possibly secondary to abnormally high mechanical stresses placed on the physis, further increased due to obesity or metabolic disorders [8].

Natural history: The epiphyseal 'slip' can cause disruption of the blood supply to the femoral head, resulting in avascular necrosis (AVN) of the femoral head. If untreated, the deformity can then cause the femoral head to impinge on the acetabulum, leading to OA, presenting later in adulthood [9].

Clinical presentation: Patients with chronic SCFE tend to present with vague hip, thigh and knee pain. However, patients with an acute or unstable epiphyseal slip can present with significant lower limb pain and difficulty weight-bearing on the affected limb. On examination, patients may have a shortened and externally rotated limb. Drehmann's sign may be seen wherein passive flexion of the affected hip joint results in external rotation of the leg, which occurs due to the femoroacetabular impingement.

Investigations: Due to the generalised lower limb pain it can often be difficult to differentiate SCFE from other hip pathologies such as septic arthritis and Perthes

disease. AP and frog lateral views of the pelvis can help in making a diagnosis of SCFE. Klein's line is a radiographic line that is drawn parallel to the superior border of the femoral neck on the AP pelvic radiograph and should intersect the femoral head; in SCFE, the line does not intersect the femoral head, a feature known as the Trethowan's sign (Fig. 15.6). Another sign is the metaphyseal blanch sign, where the overlap between the 'slipped' epiphysis and metaphysis results in a crescent-shaped area of increased density over the proximal femoral metaphysis. Other helpful investigations include an ultrasound of the hip which may demonstrate an effusion or an MRI which may reveal oedema in the metaphysis confirming the slip. Blood tests looking for inflammatory markers can further help differentiate SCFE from conditions such as septic arthritis [10].

Classification: SCFE can be classified according to the onset of symptoms, the ability of the patient to weight-bear on the affected limb (Loder classification), the extent to which the epiphysis has displaced in relation to the metaphysis (i.e. the difference in epiphyseal–diaphyseal or Southwick angle between affected and unaffected sides). Figure 15.7 highlights the various classification systems.

Treatment: The goal of treatment is to prevent the epiphysis from slipping any further, thereby minimising the risk of AVN and chondrolysis. This is generally achieved by fixing the epiphysis in place in situ. A single cannulated screw is most commonly used for fixation, with the ideal screw positioned centrally in the femoral neck and perpendicular to the physis (Fig. 15.8). Reduction of slips remains a controversial topic. Unfortunately, this fixation is not without risk. Penetration of the hip joint by the screw increases the risk of chondrolysis, while AVN can still occur as a result of kinking of the retinacular vessels supplying the femoral head. Remodelling of the physis occurs around the fixation, and if required any residual deformity can be corrected at a later date with a sub-trochanteric osteotomy. As one out of five patients have bilateral SCFE, close observation is recommended in

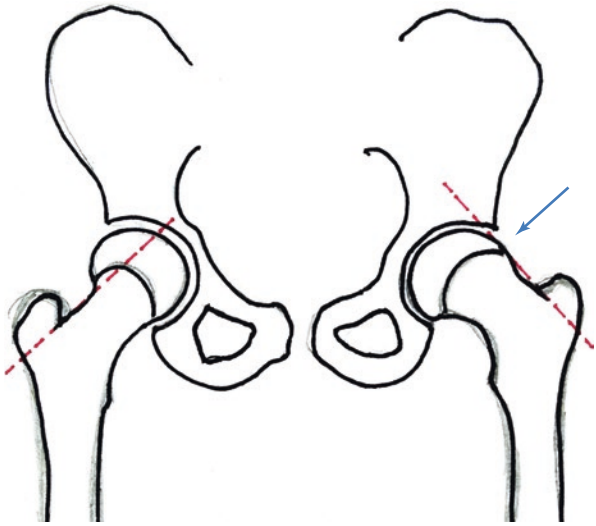

Fig. 15.6 Diagram (not to scale) demonstrating Klein's line (pink dashed lines) bilaterally and Trethowan's sign (blue arrow)

Temporal classification	
Acute	Symptoms ongoing for < 3 weeks
Chronic	Symptoms ongoing for > 3 weeks
Acute on chronic	Acute exacerbation of symptoms ongoing for > 3 weeks
Loder classification	
Stable	Patient able to weight bear on the affected limb
Unstable	Patient unable to weight bear on the affected limb
Southwick classification (based on AP radiograph)	
Grade I	0 – 33%
Grade II	34 – 50%
Grade III	> 50%

Fig. 15.7 Various classifications for SCFE

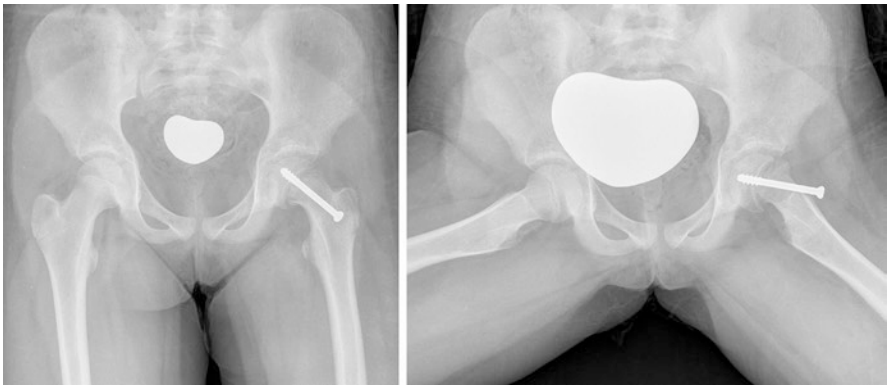

Fig. 15.8 AP and frog lateral plain radiographs demonstrating in situ fixation of a left-sided SCFE in a 12 year old

the first instance. Prophylactic fixation of the non-symptomatic hip can be considered in patients who are at high risk of developing bilateral SCFE, such as obese male patients with endocrine or metabolic bone disorders, those who present with SCFE at a younger age or in those likely to be less compliant with treatment and follow-up [11, 12].

Complications: A complication of SCFE is femoroacetabular impingement due to the mechanical deformity, which often results in patients developing earlier OA, requiring early intervention, such as total hip arthroplasty. The other significant complication is AVN which is most commonly seen in acute, unstable epiphyseal slips. Patients who are younger at the time of first presentation are at a higher risk of developing AVN. Other factors associated with an increased risk of AVN include a higher grade of epiphyseal slip and delay in presentation or diagnosis [12].

Legg–Calve–Perthes Disease

This is a non-inflammatory condition that affects the proximal femoral epiphysis, resulting in AVN, followed by remodelling of the femoral head.

Epidemiology: Perthes affects children between the ages of 4–8 years, with males being affected about 4–5 times more commonly than females. There is a clear genetic predisposition, with higher prevalence among Caucasians and is rare in African Americans. It also has a higher incidence in patients from lower socioeconomic groups. Bilateral Perthes is rare and often associated with an underlying metabolic disease.

Pathophysiology: In childhood, the majority of blood supply to the femoral epiphysis is from the MFCA, which has to pierce the hip joint capsule and then ascend the femoral neck as the lateral ascending cervical artery. It is theorised that impairment of this vulnerable blood supply leads to avascular necrosis of the femoral epiphysis. The first signs of AVN are therefore often seen in the antero-medial part of the epiphysis which is furthest away anatomically from the MFCA. AVN results in fractures of the bone, which then undergoes remodelling as revascularisation occurs. There remains uncertainty as to why these interruptions to the blood supply to the femoral epiphysis occur, with theories ranging from repetitive microtrauma to underlying coagulopathies [1, 13].

Clinical presentation: Common symptoms are generalised groin, thigh or knee pain, often without a preceding history of trauma. On examination, there may be an antalgic gait, with limited internal rotation and abduction of the hip joint.

Investigations: Perthes has characteristic radiological features depending upon the stage of progression of the disease process. First described by Waldenstrom in the early 1900s, Perthes can be divided into four distinct phases based on the radiological changes seen in the femoral head:

(a) *Initial (3–6 months):* In the early sclerotic phase, AP radiographs of the pelvis can often be normal aside from sclerosis of a smaller epiphysis. Later on, they may demonstrate a widened medial joint space.
(b) *Fragmentation (6 months–2 years):* As the lack of blood supply results in collapse of the epiphysis, there is loss of bony epiphyseal height on plain radiographs, subchondral lucency (‘crescent sign’) and fragmentation or loss of the femoral head, with patchy densities and lucencies.

(c) *Re-ossification (up to 18 months):* The third stage is when revascularisation takes place and the necrotic bone is resorbed and replaced with new bone, which appear radiologically as islands of 'fluffy' calcification (akin to callus formation).
(d) *Remodelling:* The last stage is the healing phase, where the femoral head heals and remodels, with the trabeculae returning. The final shape can be ovoid (bad) or spherical (good), with varying degrees of congruency of the joint (classified by Stuhlberg).

Other helpful investigations include ultrasound (which may detect a joint effusion), MRI (useful in confirming the diagnosis in unclear cases) and a radionuclide bone scan (may demonstrate decreased uptake in the ischaemic area of the epiphyseal bone).

Classification: There are multiple classification systems for Perthes as depicted in Fig. 15.9. The Herring classification, also known as the lateral pillar classification, is said to be the most reliable in being able to predict outcomes for patients and having less inter-observer variability. It is based on the height of the lateral pillar of the femoral epiphysis on AP radiographs of the pelvis and is usually measured at the onset of the fragmentation stage of the disease. Another classification system is the Catterall classification which depends on the degree of involvement of the femoral head. Finally, the Stuhlberg classification stratifies the final outcome based upon the femoral head deformity following the remodelling stage [14].

Prognosis: As expected, the younger the patient is at the time of diagnosis, the better the prognosis. Children under the age of 6 years are likely to do well, whereas older children are likely to have poor prognosis due to poorer remodelling/growth potential of the acetabulum around a variably deformed femoral head to maintain congruency of the hip joint. In older patients, the femoral head deformity results in an incongruent joint with poorer outcomes, as the acetabulum has already formed to match the femoral head, prior to it being deformed due to Perthes. Another factor that has an impact on the prognosis of a patient is the stage of the disease at the time of diagnosis. This is because early treatment prevents significant deformity and therefore has a better prognosis, in comparison to when diagnosis or treatment is delayed.

Treatment: Symptomatic relief must be provided by using analgesia, protected weight-bearing, splints or rarely surgery to encourage the development of a congruent hip joint. In the earlier stages of the disease, when the femoral head is still susceptible to being moulded, an abduction brace can be used to maintain reduction. In the anatomical resting position, the JRF on the hip joint results in the femoral head becoming flattened against the acetabulum, while the anterolateral part of the femoral head, which is not covered by the acetabulum, is pushed outwards, resulting in an incongruent joint. Abduction forces the entire femoral head to remain within the acetabulum, which causes the femoral head and (concave) acetabulum to mutually encourage each other to mould and form a congruent joint. This is also known as the

Herring (lateral pillar) classification		Prognosis
A	Lateral pillar is not involved	Good
B	Lateral pillar involved, but maintains > 50% of its height	Poor
B/C	Lateral pillar narrower (2–3mm) or poorly ossified, but maintains approx. 50% height	
C	Lateral pillar involved and is less than 50% of its original height	
Catterall classification		
I	Isolated involvement of anterior epiphysis	Good
II	Involvement of anterolateral epiphysis with central sequestrum	
III	75% of epiphysis involved	Poor
IV	All of epiphysis involved	
Stuhlberg's classification		
I	Spherical congruency with no radiological abnormalities	Good
II	Spherical congruency with some loss of femoral head shape	Good
III	Aspherical congruency with a non-spherical femoral head	Mild to moderate arthritis
IV	Aspherical congruency with a flat femoral head and acetabulum	Poor
V	Aspherical incongruency with a flat femoral head and normal acetabulum	Severe early arthritis

Fig. 15.9 Classification systems used most commonly for Perthes disease

'containment theory'. It can also be achieved by a proximal femoral varus osteotomy, which realigns the femoral head to ensure that it is covered by the acetabulum in its entirety. Other surgical procedures include pelvic osteotomy to augment the acetabular roof and encourage greater femoral head coverage.

In the later stages of the disease when the shape of the femoral head is fixed, the aim of treatment is to align the deformed (often flattened) femoral head in the best possible fit with the acetabulum and encourage maximum function. Typically, a valgus femoral osteotomy (±pelvic shelf osteotomy) is performed to lengthen the leg (shortening due to fixed adduction) and increase the range of motion of the hip joint before the lateral portion of the femoral head impinges on the lateral acetabulum causing pain. Such procedures, combined often later with hip arthroscopy, can help delay the need for a THA [15].

Septic Arthritis

This is an intra-articular infection within the hip joint that results in joint inflammation and cartilage destruction. It can affect both, adults and children. This is an orthopedic emergency necessitating prompt diagnosis and treatment to prevent femoral head destruction or deformity.

Epidemiology: Septic arthritis most frequently affects the hip or the knee joints. While it can affect patients of any age group, there are peaks at the extremes of ages. Other patients at risk of developing septic arthritis include premature babies, immunocompromised individuals and those who have had invasive procedures around the time of birth such as umbilical catheterisation [1].

Pathophysiology: Bacteria can inoculate the hip joint either directly through trauma or an invasive procedure or seed the joint haematogenously. Multiple bacteria have been implicated in septic arthritis, but the commonest remains *Staphylococcus aureus* in most age groups, with Group B Streptococcus (neonates) and *Neisseria gonorrhoeae* (adolescents) also prevalent. Articular surface damage is caused by the proteolytic enzymes produced by the organisms.

Clinical presentation: Patients present with symptoms localised to the hip joint such as acute pain, stiffness and difficulty in weight-bearing. Systemically, they may complain of feeling generally unwell with fevers, night sweats and loss of appetite. On clinical examination, they tend to mobilise with an antalgic gait or reluctance to weight-bear on the affected limb. There may be localised swelling and tenderness, with the hip joint resting in a flexed, abducted and externally rotated position (FABER), i.e. the position of maximum capsular volume. Passive hip movements elicit severe pain, and patients therefore resist moving the hip (pseudoparalysis). Systemic examination may reveal tachycardia and pyrexia, suggestive of sepsis.

Differentials: These include transient synovitis (a diagnosis of exclusion), osteomyelitis (may coexist in children) or other non-orthopedic pathology (e.g. appendicitis, gynaecological disorders). Differentiating between transient synovitis and septic arthritis can often be clinically challenging. Investigations may be useful in this regard.

Variable	Value	Probability of patient having septic arthritis
Non weight bearing	-	1 variable = 3% 2 variables = 40% 3 variables = 93% 4 variables = 99%
Fever	> 38.5 °C	
White blood cell count	> 12,000 cells / mm^3	
Erythrocyte sedimentation rate	> 40 mm / hr	

Fig. 15.10 Kocher's predictors of septic arthritis

Investigations: Plain radiographs may occasionally demonstrate an effusion, but are often unremarkable; their primary purpose is to exclude other underlying bony pathology. An ultrasound scan of the hip joint may better demonstrate a joint effusion. MRI can be a useful adjunct, especially if osteomyelitis is suspected.

Kocher et al. [16] described four variables which can be used to risk stratify the likelihood of septic arthritis; these are shown in Fig. 15.10. A modified version of Kocher's criteria is commonly used in practice, which utilises CRP in place of ESR.

If septic arthritis of the hip joint is suspected, the gold standard investigation is to obtain an aspirate from the hip joint (in a systemically well patient). This will usually need to be performed under general anaesthetic in paediatric patients, but can be done under local anaesthetic with X-ray or ultrasound guidance in adults. The aspirate is then sent to microbiology laboratory for gram staining, cultures and sensitivity, which may confirm the diagnosis and demonstrate the causative organism. Ideally, the aspirate should be taken prior to the patient receiving any antibiotics. In unwell patients, a diagnostic and definitive washout may be warranted to minimise damage to the articular cartilage.

Treatment: Once the diagnosis is established, antibiotics should be started based on the most likely causative organism, with the advice of a microbiologist. Often patients require antibiotics for 4–6 weeks after being diagnosed with septic arthritis of the hip joint, with regular monitoring of their inflammatory markers. Antibiotics are initially administered intravenously, although there is some evidence in children that earlier switch to oral routes may yield equivalent outcomes. The definitive treatment is a hip arthrotomy with thorough washout of the joint, followed by closure. This reduces the bacterial load and improves antibiotic penetration of the joint and may need to be repeated depending upon the response of the patient. The anterior Smith-Petersen) approach is most commonly employed for this (discussed in more detail in the surgical approaches section of this chapter).

Complications: If diagnosed and treated early, the prognosis for most patients is good, with a majority making a complete recovery. As expected, septic arthritis at

the extremes of age tends to be more severe and is associated with poorer prognosis. Complications occur due to destruction of the acetabular cartilage and femoral head, resulting in leg length discrepancy, dislocation or pseudoarthrosis. In adults, significant ankyloses and earlier OA may occur.

Tuberculosis (TB)

With an increasingly aging population and the evolution of multidrug resistant *Mycobacterium tuberculosis*, the prevalence of TB is on the rise. Estimates suggest that worldwide, over 20 million people are affected by TB. The hip is the second most common musculoskeletal site for TB infection after the spine, accounting for 15% of all musculoskeletal TB cases.

Epidemiology: Hip TB most commonly affects individuals under the age of 30 years. Risk factors for developing infection include being in close constant contact with individuals with TB (such as healthcare and prison workers), immunosuppression (either due to concurrent HIV infection, suffering from diabetes or being on steroids and/or immunosuppressants, e.g. after organ transplantation), renal failure and smoking.

Pathophysiology: TB infection of the hip joint tends to occur due to haematogenous spread of the bacterium, *Mycobacterium tuberculosis*, from the lungs or lymph nodes to the femur and synovium. Very rarely tuberculous osteomyelitis of the proximal femur can spread to the hip. An inflammatory process occurs, resulting in joint destruction and the formation of cold abscesses. Figure 15.11 depicts the natural history of the disease.

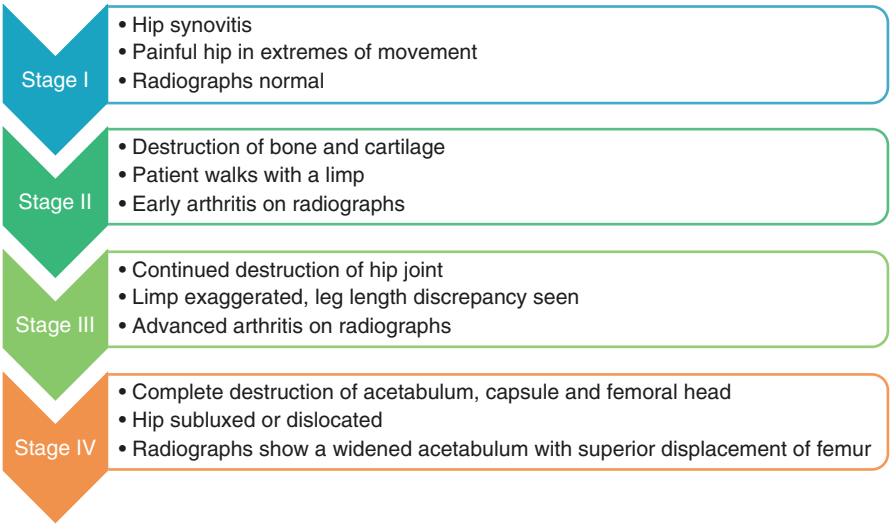

Fig. 15.11 Natural history of the disease with the four stages of progression

Clinical presentation: Patients with hip TB often do not present till the later stages of the disease when the hip joint has undergone significant destruction. Symptoms may be mild and non-specific. The majority of patients complain of groin pain radiating to the knee and altered gait, which may have a significant impact on their quality of life. Typically, the hip pain tends to be worse at night due to muscle spasms, often resulting in 'night cries' in the younger patient. On examination, leg length discrepancy and muscle wasting may be seen. Patients tend to hold their hip in a flexed, abducted and externally rotated position (FABER) for comfort and will resist passive hip movements due to pain. Other signs that might suggest underlying hip TB include presence of an abscess or a discharging sinus. Differential diagnosis for hip TB includes septic arthritis, osteomyelitis, Perthes disease and neoplasm [17].

Investigations: First-line investigations for hip TB include blood tests for inflammatory markers such as white cell count and C-reactive protein, which will be raised. Modern assays (e.g. QuantiFERON®-TB Gold) have over 90% sensitivity for diagnosis. Plain radiographs may not reveal any abnormalities in the early stages of the disease, but changes that can be seen in later stages of the disease follow three classical features known as the Phemister triad: joint space narrowing, bony erosion and articular osteopenia. An ultrasound of the hip may reveal a joint effusion or synovial thickening, whereas MRI may also be used to detect bony changes earlier on in the disease process. The next step in the investigative process is to obtain a hip joint aspirate. Fluid obtained at the time of the aspirate can be sent off to test for acid-fast bacilli and for culture and sensitivity.

Treatment: The aim is to halt further destruction of the joint by controlling the infection and restoring joint function. Anti-tuberculous chemotherapy forms the mainstay of treatment. Traction may help relieve muscle spasms in addition to analgesics. Surgical options to treat hip TB include synovectomy and debridement of the joint, aspiration of any cold abscesses. Other surgical procedures that are commonly recommended once the disease is under control are joint arthrodesis, THA or a Girdlestone excision arthroplasty depending on the severity of joint destruction and baseline function of the patient. There are concerns about performing a THA in patients with hip TB due to the recurrence rate of infection and the relatively high rate of post-operative complications. However, studies have shown that *Mycobacterium tuberculosis* does not form a biofilm and therefore classic triple/quadruple regime antibiotic treatment for 6–12 months post THA yields good results in terms of low infection recurrence [18, 19].

Adult Disorders of the Hip

Osteoarthritis (OA)

This is a degenerative condition that results in destruction of articular cartilage, resulting in pain and loss of function. The hip is the second most commonly affected joint, after the knee. Hip OA can result in significant functional disability, limiting

patients' ability to work, undertake activities of daily living (ADLs) and participate in recreational activities.

Epidemiology: Hip OA affects over 2.5 million people in the UK alone, with the majority being over the age of 65. Women are twice as likely to be affected than men and Caucasians populations tend to have a higher prevalence of hip OA than Asians. Other risk factor associations for developing hip OA include high BMI, manual labour or having an underlying intra-articular disease such as DDH or Perthes [20].

Pathophysiology: Studies suggest that hip OA develops due to a combination of factors which cause abnormal loading of the hip joint resulting in repetitive intra-articular microtrauma. This encourages an inflammatory response leading to synovitis, subchondral bone remodelling and osteophyte formation. Trauma, joint dysplasia and instability can all result in premature OA [21] (Fig. 15.12).

Clinical presentation: Patients typically present with hip or groin pain associated with joint stiffness. The pain may be exacerbated by weight-bearing and certain movements and may radiate down to the ipsilateral knee joint. On examination, these patients may have a Trendelenburg gait and loss of movement, especially internal rotation.

Differentials: Other diagnoses that may present with similar symptoms include knee OA, lumbar spine pathology, trochanteric bursitis and femoroacetabular impingement. Indeed, these may coexist with hip OA.

Investigations: Plain radiographs form the basis of investigating hip OA and classically depict the four characteristic features of (1) loss of joint space, (2) osteophyte formation, (3) subchondral sclerosis and (4) subchondral cysts (Fig. 15.13). However, it must be noted that these radiographic changes may not be present in all patients presenting with hip pain, in which case the history and examination will be of more importance. MRIs are of little use in investigating hip OA, although they may be useful in younger patients, where (combined with an arthrogram) they may help identify Femoro-acetabular impingement (FAI), see below.

Primary OA	Secondary OA
Increasing age Idiopathic	Trauma Congenital disorders – SCFE, Perthes, Congenital hip dislocation Metabolic disorders – Gout, Haemochromatosis, Gaucher's disease Endocrine disorders – Diabetes, acromegaly Miscellaneous – Osteonecrosis, infection, Paget's disease

Fig. 15.12 Causes of osteoarthritis

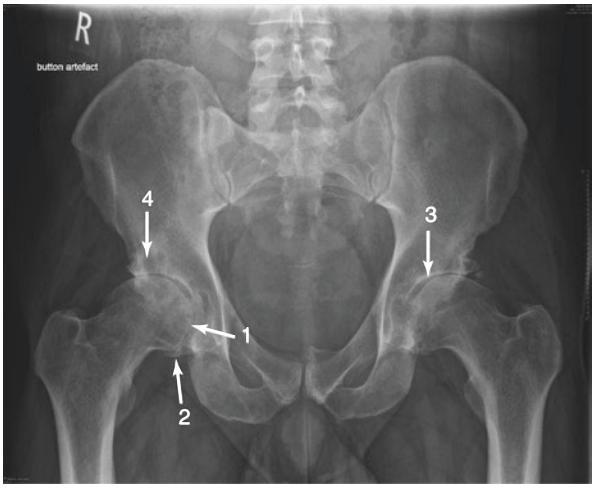

Fig. 15.13 AP plain radiograph of the pelvis demonstrating OA in the hips bilaterally. Radiographic changes can be remembered by using the mnemonic LOSS where: (*1*) L = loss of joint space, (*2*) O = osteophyte formation, (*3*) S = subchondral sclerosis, (*4*) S = subchondral cysts

Treatment: Early hip OA can be treated conservatively with the use of analgesia, walking aids, weight loss and physiotherapy. Reduction in weight and increase in muscle strength decreases the JRF through the hip joint, easing symptoms [22]. Intra-articular steroid injections are commonly used as they can provide excellent pain relief while at the same time helping to confirm that the hip is the source of the patient's pain. However, these often require image guidance to confirm appropriate needle position and do carry a small risk of infection. Ultimately, if a patient's symptoms are refractory to conservative and non-operative treatment options, surgery may yield excellent outcomes. This can either be joint preserving (i.e. an osteotomy to restore normal joint loading—uncommon) or joint replacing (i.e. THA—common). In rare cases, joint fusion or excision arthroplasty may be considered.

Osteonecrosis of the Hip

Also known as avascular necrosis (AVN) of the hip, this is characterised by a disruption of the blood supply to the femoral head and articular surface, resulting in osteocyte necrosis, subsequent remodelling and ultimately collapse of the femoral head.

Epidemiology: The incidence of AVN is variable, but studies suggest that it accounts for approximately 10% of patients undergoing THA. It tends to affect men three to five times more than women. Unlike osteoarthritis, patients with AVN often present under the age of 50 years with risk factors including excessive alcohol intake, prolonged use of high-dose glucocorticoids, pancreatitis and autoimmune disorders (e.g. systemic lupus erythematosus, SLE).

Pathophysiology: Causes of AVN can be divided into traumatic and non-traumatic. Traumatic causes include previous (subcapital) femoral neck fracture and dislocation, all of which can disrupt the blood supply to the femoral head, leading to collapse. Non-traumatic AVN is caused by a combination of local and systemic

factors that are hypothesised to predispose patients to increased intraosseous pressure, vasoconstriction and microemboli, ultimately reducing the femoral head's blood supply [23].

Clinical presentation: Patients typically present with hip or groin pain that is exacerbated on weight-bearing and movement of the hip. Clinical examination may elicit an abnormal gait, pain on axial loading of the hip and reduced range of movement.

Investigations: Plain radiographs are the first-line investigation for AVN of the hip and may demonstrate flattening of the femoral head in the later stages. Radiographs may be normal in early stages; MRI can be useful earlier in the disease process and has a high sensitivity and specificity for AVN. The modified Ficat and Arlet system is used to classify AVN (Fig. 15.14).

Treatment: In the early stages, non-operative treatment including restricted weight-bearing, supplemented by walking aids and physiotherapy, can provide symptomatic relief. In addition, bisphosphonates may occasionally be used to slow down osteoclastic activity. It is important to also attempt to address the underlying cause of the AVN. As the natural history of the disease is gradual progression leading to collapse of the femoral head, performing a core decompression early in the disease process may help restore vascularity and reduce the hip pain. This can be performed either as an open procedure or, more commonly, percutaneously and involves drilling several holes in the femoral head and removing any necrotic tissue to reduce intraosseous pressure and help restore perfusion. Following a core decompression, bone grafting may be necessary to provide structural support and stimulate bone healing. Core decompression is not indicated in the later stages of the disease as its success rate tends to dramatically drop after Ficat and Arlet stage 2 (i.e. once there is subchondral collapse). As patients with AVN tend to be younger, emphasis is on joint preservation and therefore, THA is reserved for those who fail medical treatment and/or core decompression [24, 25].

Fig. 15.14 Modified Ficat and Arlet classification system for AVN of the hip

Stage	Radiographs
0	Normal
I	Mild osteopenic changes
IIa	Presence of sclerosis / cysts
IIb	Crescent sign
III	Flattening of femoral head
IV	End stage disease with joint space narrowing

Meralgia Paresthetica

This condition occurs due entrapment of the (sensory) lateral femoral cutaneous nerve (LFCN). The LFCN arises from the lumbar plexus (dorsal branches of L2, L3), after which it crosses the iliopsoas, travelling inferolaterally towards the ASIS. The LFCN then passes underneath the inguinal ligament before innervating the skin over the anterolateral aspect of the thigh. It is therefore susceptible to injury at multiple levels, including extrinsic compression by retroperitoneal and pelvic tumours, or stretching due to hyperextension of the trunk or during anterior pelvic procedures, resulting in a neuropathy. The majority of cases are idiopathic in nature with no precipitating cause found. Patients usually present with pain, numbness and paraesthesia in the anterolateral thigh. Symptoms are exacerbated by tight belts, prolonged walking and standing. Diagnosis is primarily clinical [26], with imaging (e.g. USS) to help determine the cause of compression. Imaging may demonstrate a swollen LFCN and help confirm the diagnosis [27]. In the majority of cases treatment is conservative and includes simple oral analgesics (such as anti-inflammatories) and removal of the compressive agent (such as tight belts for weightlifting). Occasionally, an injection of local anaesthetic and corticosteroid is required, with surgery being reserved for patients with intractable symptoms. This involves either neurolysis or transection of the nerve; however there are no clear evidence to guide optimal surgical management.

Trochanteric Pain Syndrome

This encompasses a range of pathologies including trochanteric bursitis, gluteus medius or minimus tendinopathy and external coxa saltans (snapping hip). The exact cause behind this is unclear; however it is hypothesised that abnormal biomechanics of the hip result in gluteal tendinopathies, with the bursa overlying the greater trochanter becoming inflamed, likely secondary to repetitive microtrauma. Patients often present with lateral hip pain that is exacerbated by movement. On examination, there may be localised tenderness over the greater trochanter prominence on palpation, and minimal pain elicited on passive movement of the hip joint, which is in stark contrast with the classic examination findings in hip OA. When there is diagnostic doubt, USS or MRI can be useful in demonstrating inflammatory changes within the bursa. In majority of the cases, trochanteric bursitis settles spontaneously. For patients with recurrent or protracted symptoms, corticosteroid injections and physiotherapy can be used. Surgery is rarely required. Surgical options include bursectomy, iliotibial band release and gluteus medius tendon repair [28, 29].

Femoroacetabular Impingement (FAI)

As the name suggests, this is a condition in which anatomical abnormalities in either the acetabulum or the femoral head result in disruption of the normal concentric

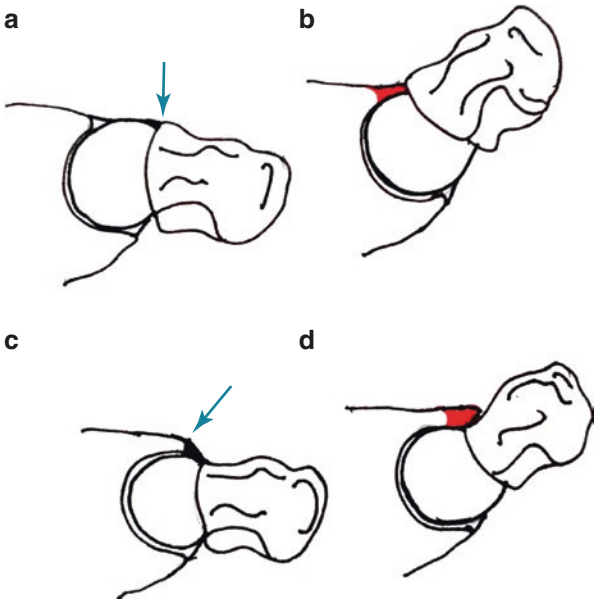

Fig. 15.15 Line diagram showing the cam and pincer deformities

movement of the hip joint, resulting in pain, joint stiffness and loss of function [21]. There are two main types of FAI described:

1. Cam: This is often the consequence of an abnormally shaped (non-spherical) femoral head in a normal acetabulum (diagrams A and B in Fig. 15.15).
2. Pincer: This is usually due to either excessive acetabular retroversion or an abnormally deep acetabulum, which results in the femoral head abutting it anteriorly. Over two-thirds of patients with FAI will have pincer-type anatomy (diagrams C and D in Fig. 15.15).

Diagram A demonstrates an abnormally shaped, bulging femoral head with a normal acetabulum, i.e. a cam deformity, which results in FAI as shown by Diagram B.

Diagram C demonstrates an abnormally shaped acetabulum with a normal femoral head, i.e. a pincer deformity, which results in FAI as shown by diagram D.

The lack of joint congruency in both types can result in damage to the acetabular labrum. Patients present with hip pain that is exacerbated by certain movements and loss of function. Presentation tends to be at a younger age and in more active patients than the typical group for OA. Examination findings are very similar to hip OA. Passive hip movements elicit groin pain, especially with a combination of hip flexion, adduction and internal rotation (FADIR or anterior impingement test). Plain radiographs may show more advanced abnormalities although MRA (MRI with intra-articular contrast) will better demonstrate the extent to which the acetabular labrum has been damaged. Hip arthroscopy is an increasingly popular therapeutic option, during which the acetabular labrum can be repaired, and joint incongruency improved by reshaping the femoral head with a burr [30].

Approach	Advantages	Disadvantages
Anterior	Muscle sparing Internervous planes Good femoral head exposure (e.g. DDH)	Limited access to acetabulum
Antero-lateral	Lower dislocation rate	Abductors can be damaged (Trendelenburg gait)
Lateral	Good exposure of the femur Commonly used (familiarity)	Abductors can be damaged
Posterior	Good exposure of acetabulum and femur Extensile	Higher dislocation rate compared to other approaches

Fig. 15.16 Advantages and disadvantages of the four most commonly used approaches

Surgical Approaches to the Hip Joint

The hip joint can be accessed via a number of classic approaches, each of which has multiple variations, advantages and disadvantages. The approach used depends upon patient and procedure factors. In this chapter, we will focus on the four commonest approaches. Figure 15.16 summarises the advantages and disadvantages of these.

Anterior (Smith-Petersen)

This was first described by Hueter in the late 1800s (Fig. 15.17) and later popularised by Smith-Petersen. As a muscle sparing approach, this follows intermuscular intervals to access the hip joint (Fig. 15.18). It is most commonly used in treating paediatric patients with DDH or for a hip arthrotomy for washout of septic arthritis. This approach can also be modified to perform minimally invasive hip surgery [1]. Advantages include a supine position (Fig. 15.19) (permitting bilateral hip surgery without repositioning the patient) and potentially faster initial recovery. However, operating times are often longer, and the approach is associated with a steep learning curve. It can be technically difficult in obese patients and provides limited access to the acetabulum and femur (Fig. 15.20). It is therefore not as popular as some of the other hip approaches for adult hip surgery [31, 32]. Figure 15.21 illustrates the anterior approach to the hip joint.

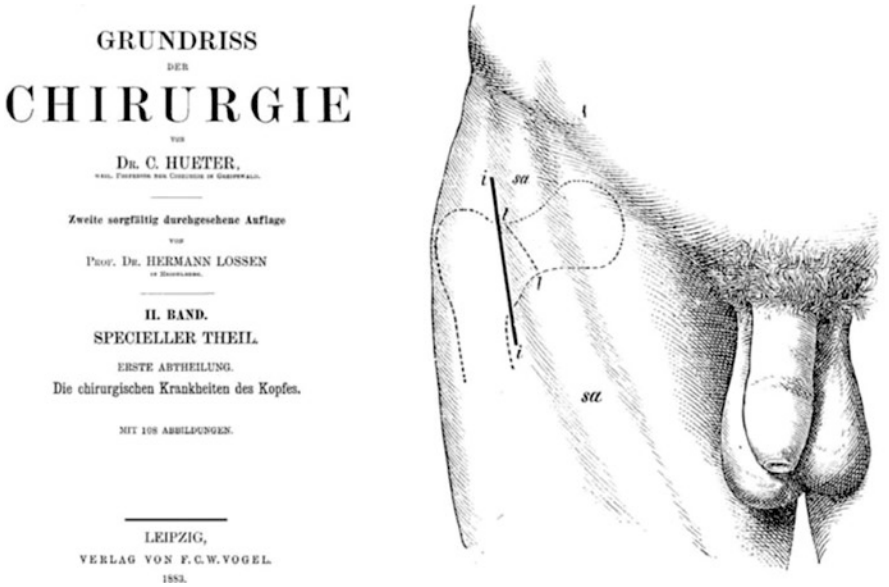
GRUNDRISS
DER
CHIRURGIE
VON
DR. C. HUETER.
Zweite sorgfältig durchgesehene Auflage
VON
PROF. DR. HERMANN LOSSEN
II. BAND.
SPECIELLER THEIL.
ERSTE ABTHEILUNG.
Die chirurgischen Krankheiten des Kopfes.
MIT 108 ABBILDUNGEN.
LEIPZIG,
VERLAG VON F. C. W. VOGEL.
1883.
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Fig. 15.17 The direct anterior approach to the hip joint was first described in *Der Grundriss der Chirurgie* (The Compendium of Surgery) in 1883 by German surgeon Dr. Carl Hueter. Image reproduced courtesy of Medacta

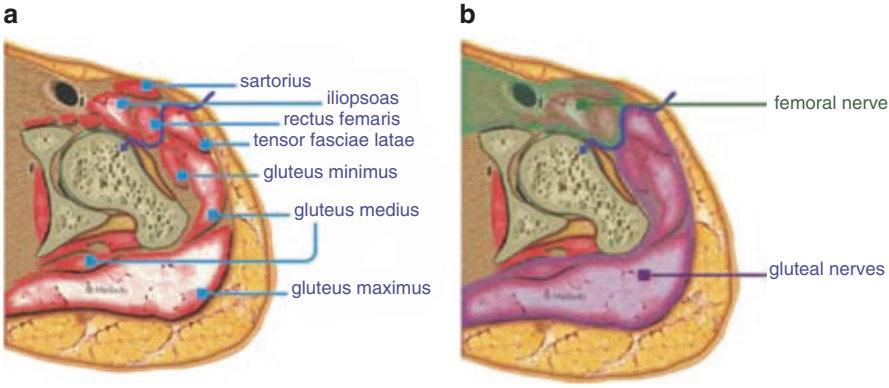

Fig. 15.18 The anterior approach is intramuscular (image **a**) and interneural (image **b**). The dark blue line represents the approach, passing between the rectus femoris and sartorius, innervated by the femoral nerve, and the TFL and gluteal muscles, innervated by the gluteal nerves. Image reproduced courtesy of Medacta

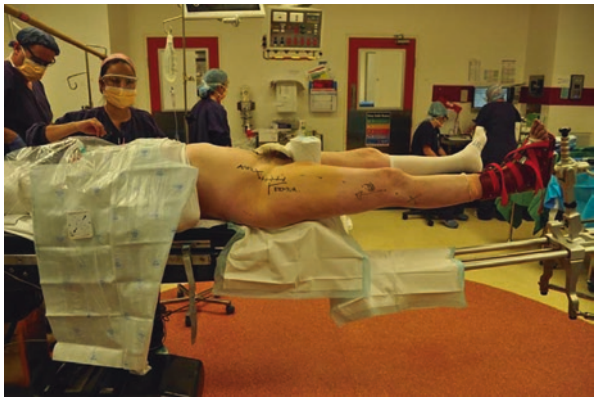

Fig. 15.19 Photograph showing a patient positioned on a fracture table for a right total hip replacement using the anterior approach. Image reproduced courtesy of Medacta

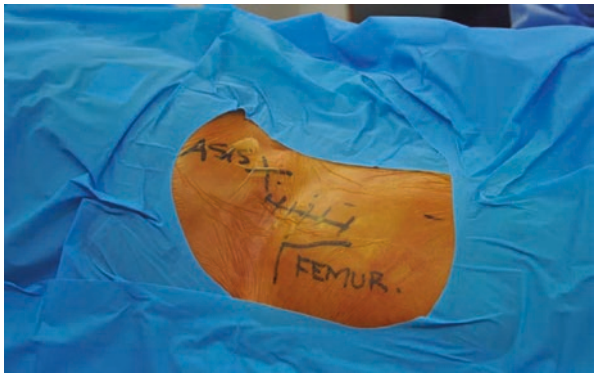

Fig. 15.20 Photograph showing the line of incision (cross-hatched line) of the anterior approach. The 'X' represents the ASIS, whereas the dotted line represents the interval between the rectus femoris anteriorly and the tensor fascia lata posteriorly. Image reproduced courtesy of Medacta

Anterolateral (Watson-Jones)

Described by Watson and Jones in the mid-1900s, this approach utilises the interval between tensor fasciae latae and gluteus medius (both supplied by the superior gluteal nerve). It involves detaching the abductor mechanism of the hip by either incising the tendons inserting onto the greater trochanter or performing a trochanteric osteotomy. It tends to be used for patients who are at high risk of hip dislocations (e.g. those suffering from neuromuscular disorders) as this approach spares the gluteus maximus and short external rotators and therefore results in a lower dislocation rate following THA. Some evidence also suggests that this approach can cause less pain, leading to faster initial recovery. However, this comes at the cost of disrupting the abductor mechanism, which could lead to the patient developing a Trendelenburg gait post-operatively. Moreover, if

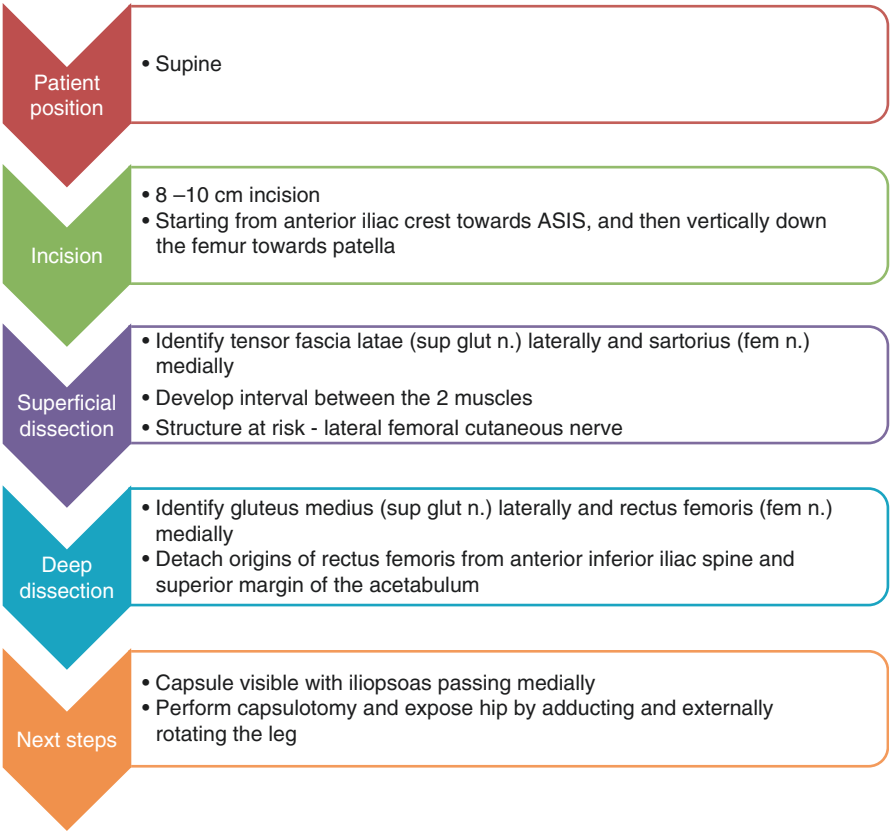

Fig. 15.21 Anterior approach to the hip joint

a trochanteric osteotomy is performed, there is a theoretical risk of developing a non-union or trochanteric bursitis. Structures at risk when performing this approach are the femoral nerve, artery and vein that are most commonly injured by retractors placed medially into the psoas muscle while gaining access to the anterior hip capsule [1]. Figure 15.22 illustrates the anterolateral approach to the hip joint.

Lateral (Hardinge)

This approach, popularised by Hardinge in the late 1900s, is similar to the antero-lateral approach, in that it also involves detaching the abductor mechanism. After dissecting down to the gluteus medius, the direct lateral approach involves incising the abductor tendons (gluteus medius and minimus, superior gluteal n.) off the greater trochanter while leaving a cuff of tendon still attached to the greater trochanter posteriorly in order to facilitate tendon repair. The incision is extended by 2–3 cm proximally into the medial third of the gluteus medius muscle itself, often giving the

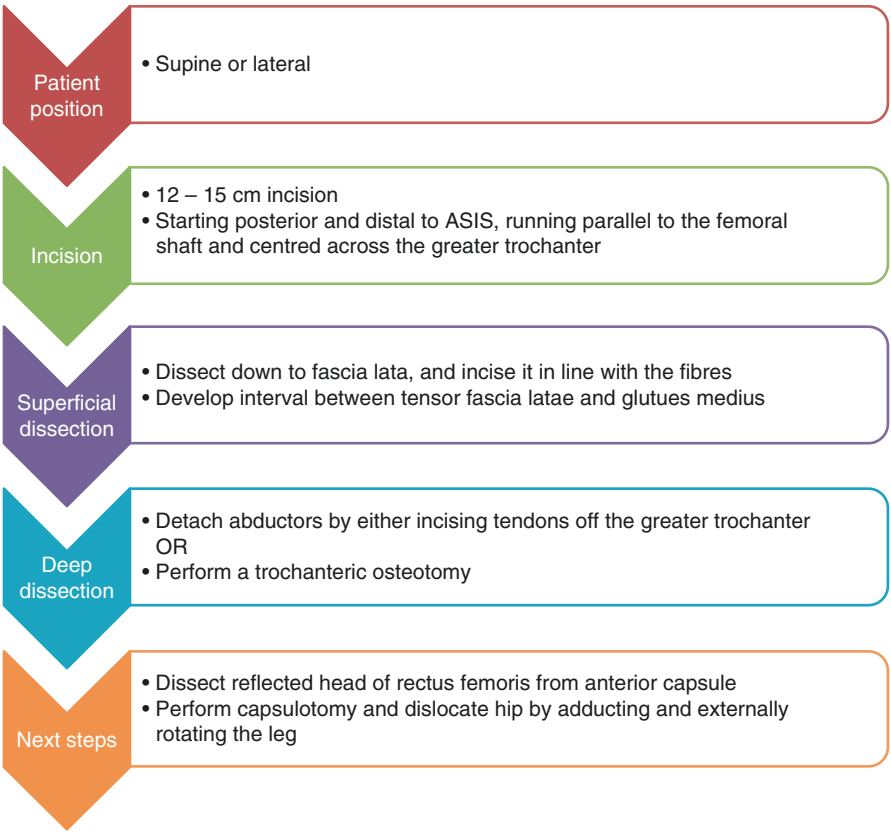

Fig. 15.22 Anterolateral approach to the hip joint

incision an 'omega' shape. This exposes the anterior hip capsule, allowing the surgeon to perform a capsulotomy. This approach has the advantage of providing good exposure to the femur; however, views of the acetabulum may be limited as the approach cannot be extended proximally due to risk of damaging the superior gluteal nerve [1]. Figure 15.23 illustrates the direct lateral approach to the hip joint.

Posterior (Moore, Southern)

Described by Moore in the mid-1900s, this is another muscle-splitting approach akin to the direct lateral approach in that it involves making a split in the gluteus maximus muscle (inferior gluteal n.). Following this, with the leg internally rotated, the tendons of the short external rotators of the hip are divided. This exposes the underlying joint capsule, allowing the surgeon to perform a capsulotomy. Alongside the direct lateral, this is a popular approach for THA, as it provides excellent exposure of both the acetabulum and the femur. It can also be extended both proximally

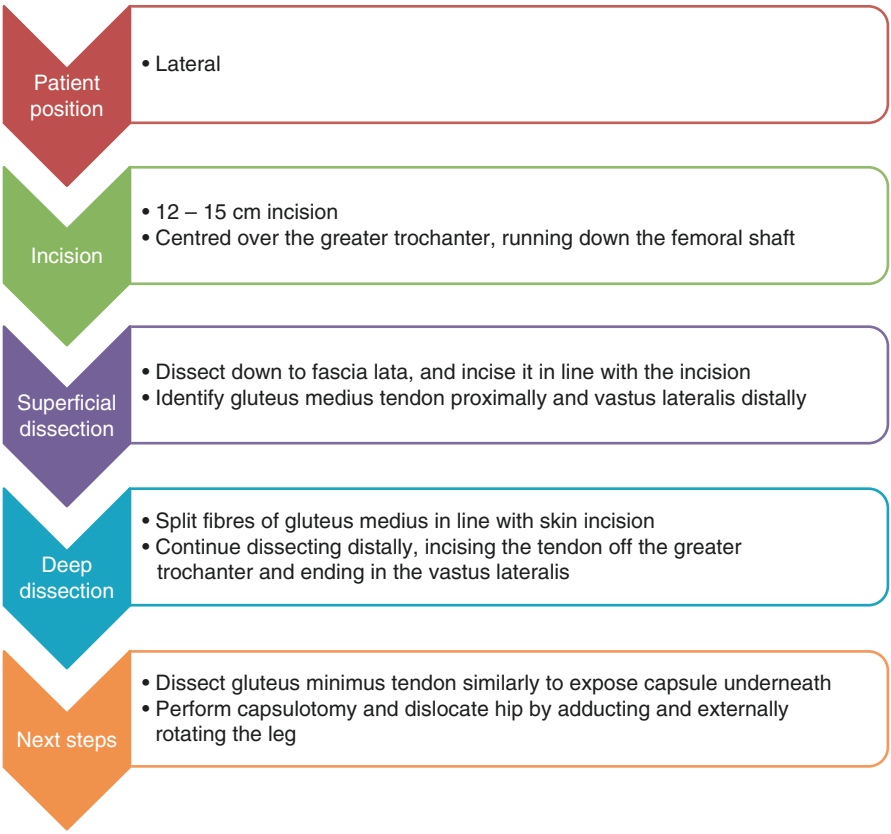

Fig. 15.23 Lateral approach to the hip joint

(Kocher-Langenbeck) and distally and is therefore useful especially in acetabular or revision hip surgery (femoral osteotomy), respectively. The main disadvantage of this approach is the relatively higher dislocation rate compared to other approaches, although the incidence can be reduced by meticulous repair of the hip joint capsule and the short external rotator tendons. The main structure at risk during this approach is the sciatic nerve which can be found passing inferolaterally across the posterior surface of the short external rotators (quadratus femoris) [1]. Figure 15.24 illustrates the posterior approach to the hip joint.

Total Hip Arthroplasty

Introduction

With the global increase in life expectancy, the number of total hip arthroplasty (THA) procedures performed worldwide is increasing yearly [33]. Introduced in the

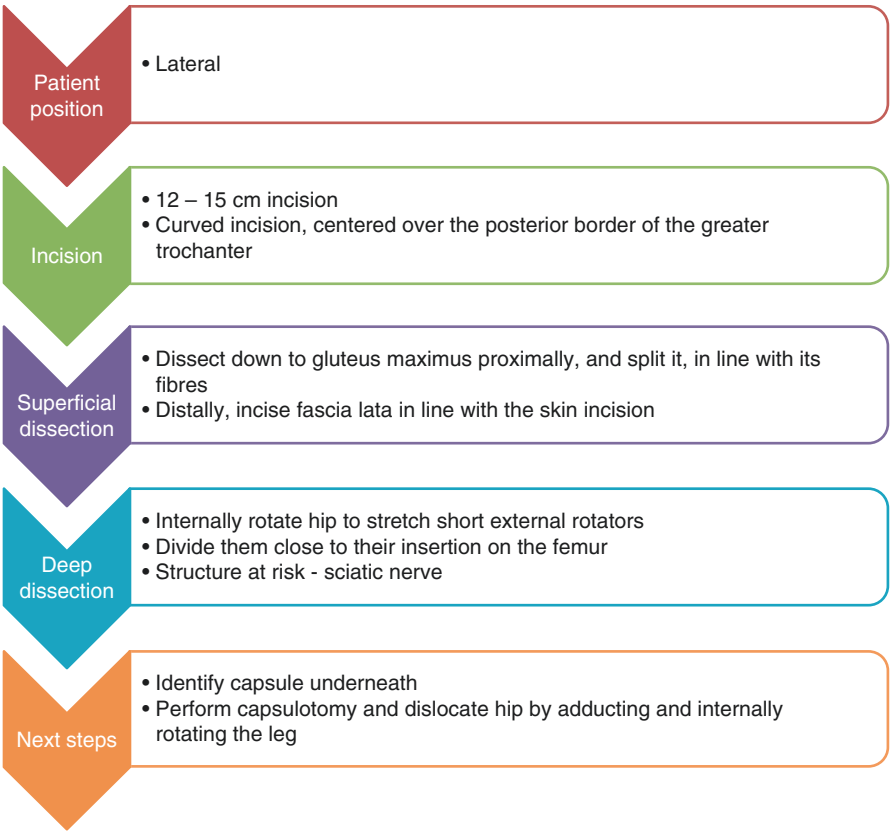

Fig. 15.24 Posterior approach to the hip joint

late seventeenth century as a treatment for hip TB, and subsequently pioneered by John Charnley in the 1960s through his 'low friction arthroplasty', THA prostheses and techniques have evolved considerably and are widely regarded as one of the most successful orthopedic procedures in terms of improving patient-reported outcomes and health-related quality of life. The primary aim of the procedure is to provide relief from hip joint pain, with the secondary aim being to improve function (i.e. maintain mobility). Figure 15.25 summarises the indications and contraindications of performing a THA.

Preoperative Planning

This involves taking a thorough history from the patient to ascertain the nature of the pain and extent of functional disability. A detailed past medical and drug history will identify comorbidities that may put patients at higher risk of perioperative complications and allow the clinician to make adjustments such as stopping

Indications	Contraindications
• Pain not relieved by conservative measures • Functional impairment: loss of ability to work or undertake activities of daily living • Gait abnormality (inc. difficulty climb stairs)	• Absolute: Any active infection (local or systemic) • Relative: Significant medical comorbidity resulting in perioperative risks outweighing than potential benefit, significant cognitive impairment, poor bone stock, marked deformity

Fig. 15.25 Table with Indications and contraindications for a THA

anticoagulants or biological rheumatological agents preoperatively. The patient's spine and ipsilateral knee should also be examined alongside the hip to ensure that the pathology is indeed in the hip joint and to exclude other related/adjacent causes of pain. If there is any doubt as to the source of the pain, then a diagnostic intra-articular hip steroid (and more importantly local anaesthetic) injection can be trialled prior to proceeding with arthroplasty [1, 34].

Patients should be advised on the risks, benefits, alternatives, recovery and outcome following THA, with input from other key team members (e.g. anaesthetists, physiotherapists, rheumatologists) to optimise a patient medically and psychologically before the decision to proceed with arthroplasty is made. Non-operative measures (analgesia, physiotherapy, offloading walking aids, activity modification, weight loss, injections) should always be tried in the first instance. A growing number of centres enrol patients in some form of preoperative physio-led 'joint school' where patients are educated regarding the surgery and post-operative rehabilitation. Such support networks also allow any specific support needs that an individual patient will require (e.g. support with washing/dressing/meals post-operatively) to be identified and addressed early. Combined with 'enhanced recovery' pathways, such measures can help reduce the length of hospital stay.

Once the decision to proceed with arthroplasty is made, preoperative plain radiographs of the pelvis and proximal femur guide selection of the femoral and acetabular components most suited for the patient and ensure that any anatomical deformities are taken into consideration when planning surgery. With the mainstream use of electronic medical imaging, alongside growing availability of templating software, technology should be used to plan the procedure (e.g. estimate the extent of bony cuts, sizing and positioning of the implants required) [35, 36].

Risks and Complications of THA

Figure 15.26 lists the common and significant risks and complications of THA that need to be discussed with the patient as part of gaining informed consent [37].

Common	2 – 5%	Pain Bleeding Leg length discrepancy Prosthesis wear/loosening/dislocation Deep vein thrombosis (DVT)
Rare	1 – 2%	Infection Periprosthetic fracture
Very Rare	Less than 1%	Neurovascular injury Fracture Wound healing problems (including hypertrophic or keloid scarring) Pulmonary embolism (PE) Fatality Other medical complications

Fig. 15.26 Table indicating the risks and complications of THA

Some specific complications are discussed below:

1. *Leg length discrepancy:* Patients may present with discomfort and difficulty walking. Generally leg length discrepancies of more than 2 cm are noticeable and require intervention (e.g. shoe raises). Preoperative radiological templating, implant positioning and intraoperative assessment of leg lengths will help reduce any significant leg length discrepancy.
2. *Infection:* Factors predisposing patients to developing infection in the THA include immunosuppression (diabetes, steroids) and obesity, with the commonest organisms being *Staphylococcus aureus* and *Staphylococcus epidermidis*. Superficial infections are commoner than deep, although the latter has far graver consequences. Numerous strategies can be put in place to reduce the rate of infection, starting with screening patients preoperatively and treating them for any active infection. Perioperative antibiotics and laminar flow theatres should be used, alongside meticulous haemostasis. There is also evidence to support measures such as wearing surgical exhaust suits, reducing unnecessary theatre traffic and appropriate skin preparation. Post-operatively, patients should be educated regarding the symptoms and signs of an infected prosthesis and advised to seek urgent medical attention.
3. *Dislocation:* This can occur due to either modifiable surgical factors (approach, implant positioning, technique, adequate soft tissue repair) or non-modifiable patient factors (neuromuscular disorders, compliance with hip precautions). A posterior approach is often associated with higher dislocation rates than the other approaches, although with good soft tissue repair techniques the incidence of dislocation can be minimised, particular when post-operative ‘precautions’ are followed. Moreover, optimising surgical technique is key (i.e. using larger femoral heads and constrained liners when needed, ensuring correct component orientation and performing a good soft tissue repair) to increasing stability. Most

dislocations can be treated with closed reduction; however, patients presenting with recurrent dislocations may require open reduction and revision surgery to address the underlying cause [38, 39].

4. *Venous thromboembolism (VTE):* Post-operative anticoagulation is commonly used for around one-month post THA. However, there remains a lot of controversy surrounding the evidence base, and there is no clear consensus on the ideal chemical prophylaxis agent and the duration of prophylaxis. While some advocate antiplatelet therapy (aspirin), others prefer low-molecular weight heparin (LMWH) or the newer, direct oral anticoagulants (DOACs). Moreover, some surgeons forego drug therapy in favour of compression stockings alone, especially in patients who are at risk of developing haematomas, which in turn increases their risk of getting an infection. Ultimately, each patient should be individually assessed for their risks of developing venous thromboembolism and treatment tailored accordingly, within the remit of clearly defined protocols in each elective arthroplasty unit [40–42].
5. *Periprosthetic fracture:* The incidence of a periprosthetic fracture following a THA varies depending on the implants used and patient factors. Periprosthetic fractures are more frequent in women, especially those with osteoporosis, and are associated with the use of uncemented femoral stems [43]. Figure 15.27 depicts the Vancouver classification of periprosthetic femoral fractures.
6. *Neurovascular injury:* Nerve injuries are a rare occurrence after a THA, with the most likely injured structure being the sciatic nerve (usually the peroneal division). Patients commonly present with foot drop due to common peroneal nerve injury. Other nerve injuries include the femoral and superior gluteal nerves (approach dependent) and tend to occur due to excessive traction on the nerve or from compression due to haematoma/cement. Major vascular injuries are rare; patients may present with oozy wounds (infection should always be a consideration), excessive pain or ischaemic limbs. Structures at risk include the common femoral and external iliac vessels. Mechanism of injury is similar to nerve injuries and includes excessive traction or direct iatrogenic injury.

Obturator nerve and vessel injuries are rarer and occur due to incorrect placement of acetabular screws. To prevent this, surgeons should be aware of the quadrant system described by Wasielewski et al. for the safe placement of acetabular screws. The acetabulum can be divided into four quadrants by drawing an imaginary line from the ASIS through the centre of the acetabulum to the posterior fovea, with a second line perpendicular to this (Fig. 15.28). The posterosuperior and posteroinferior quadrants tend to be the safest for screw placement, with screws in the anterosuperior and anteroinferior quadrants being most likely to injure the obturator vessels and nerve respectively [44].

Implant Fixation

Figure 15.29 shows the components of a THA. These can be inserted cemented or uncemented. Bone cement, polymethylmethacrylate (PMMA), acts as a grout (not an

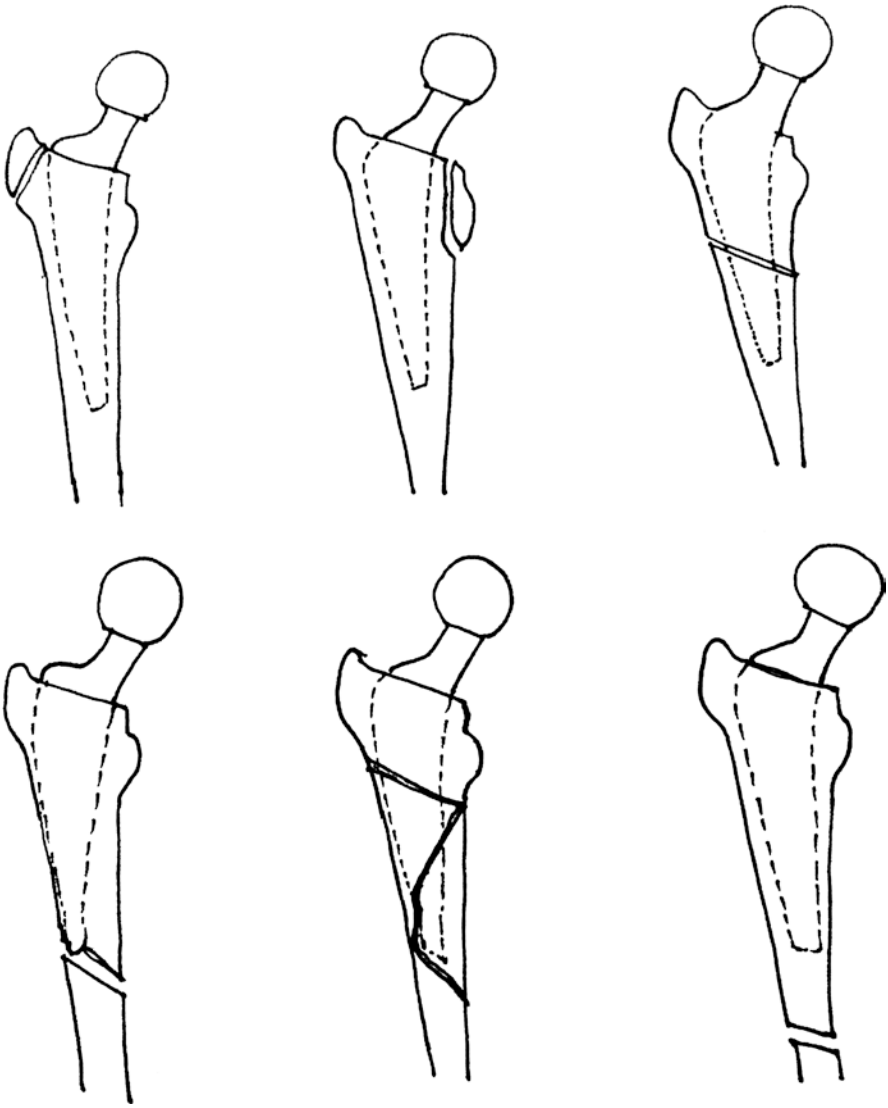

Fig. 15.27 Line diagram showing the Vancouver classification of periprosthetic femoral fractures. A_G—fracture through greater trochanter, A_L—fracture through lesser trochanter, B1—fracture around a well fixed stem, B2—fracture around a loose stem, B3—fracture around a loose stem with poor bone stock, and C—fracture well below the stem and cement mantle

adhesive) and an even cement mantle with a centralised stem is paramount to balance stresses. Cement preparation involves mixing a powder (polymer) and liquid (monomer), which contain additives such as activators, stabilisers, antibiotics, chlorophyll and radiopacifiers (Fig. 15.30). When mixed, an exothermic reaction occurs, resulting in polymerisation of the PMMA monomers, which in turn causes the bone cement to harden.

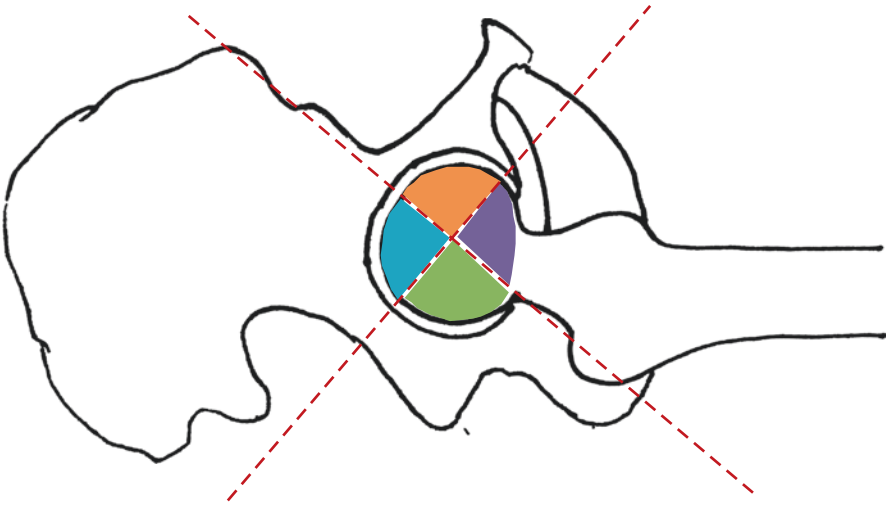

Fig. 15.28 Line diagram showing four quadrants of the acetabulum.
Key:

	Quadrants	Zone safety	At risk structures
	Posterior – superior quadrant	Safest	Sciatic nerve Superior gluteal nerve and vessels
	Posterior – inferior quadrant	Safe	Sciatic nerve Inferior gluteal nerve and vessels
	Anterior – superior quadrant	Danger	External iliac artery and vein
	Anterior – inferior quadrant	Danger	Obturator nerve and vessels

Mixing and cementing have evolved considerably from the first-generation technique of hand mixing and finger packing to the current third-generation techniques wherein mixing takes place in a vacuum (to minimise porosity), and the resultant mixture is injected into the femoral canal in a retrograde fashion using a cement gun. The canal is then pressurised to ensure an even cement mantle, with the optimum thickness being 2 mm around the stem. A cement restrictor, placed prior to inserting the cement, prevents the cement from being pushed further down the femoral canal during the cementing process.

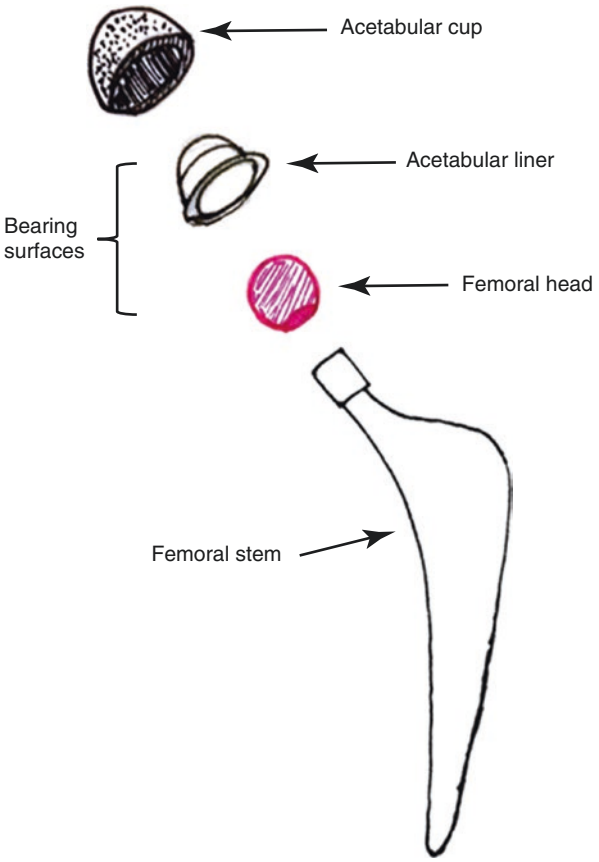

Fig. 15.29 Line diagram showing the components of a THA

Liquid	Powder
Monomer	Polymer
Activator	Opacifier
Catalyst	Dye (Chlorophyll)
Stabiliser	Antibiotics
Dye (Chlorophyll)	

Fig. 15.30 Components of bone cement

Uncemented prostheses rely on press-fit fixation and osseointegration. They tend to have porous coatings, which encourage bony ingrowth and promote the formation of a stable bone-implant construct. There remains considerable debate on the merits of cemented vs uncemented stems, with no consensus on the optimal strategy. For example, while uncemented stems remain popular in the USA, cemented stems are increasingly utilised in the UK.

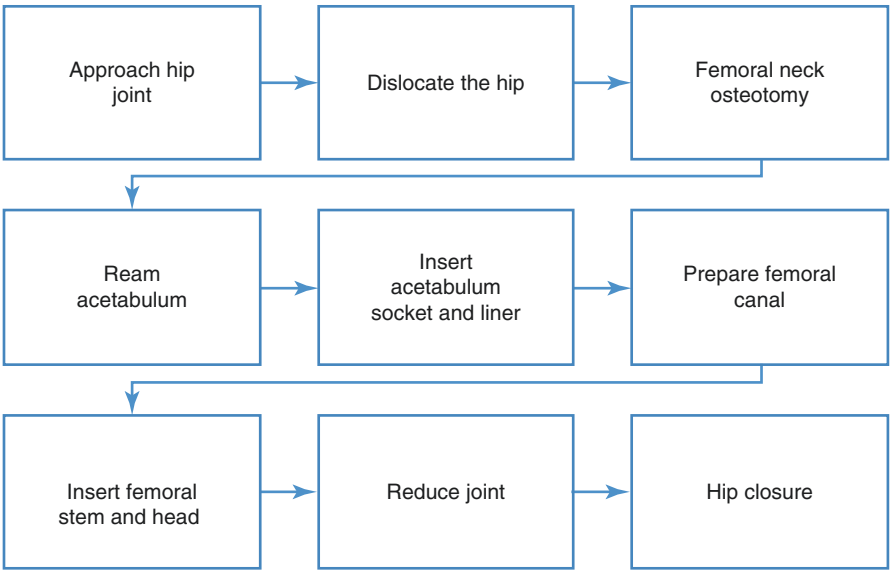

Fig. 15.31 Basic surgical steps for a THA

Figure 15.31 describes the basic surgical steps for a THA.

Acetabular Component

Prior to inserting the acetabular cup, the surgeon needs to ream the acetabulum down to the 'true floor' for two reasons. Firstly, the subchondral bone must be reached to improve fixation, and, secondly, to restore the centre of rotation of the hip joint and improve the abductor lever arm. Next, the acetabular cup should ideally be placed in 35–45° of abduction and 15–25° of anteversion (depending on the approach), to ensure optimum stability.

Cemented acetabular components are generally made up of ultra-high molecular weight polyethylene (UHMWPE) and can have spacers which are 2–3 mm in height. These spacers prevent the cup from bottoming out and aid in getting an even cement mantle around the implant. Other modifications include having a larger posterior wall or a posterior lip to prevent hip dislocation. While cheaper than their uncemented counterparts, there is some concern that cemented acetabular cups have lower survivorship and therefore tend to be used mainly for older patients or in revision surgery in the elderly.

Inserting uncemented acetabular components requires under reaming of the acetabulum by 1–2 mm to aid the press-fit fixation of these implants. They tend to have a metal shell with a porous coating (often hydroxyapatite) to encourage osseointegration [45–47].

Femoral Component

The femoral head component of most modern stems is available in different sizes. Changes in size not only affect the vertical height of the prosthesis but also have an impact on the offset of the stem. The diameter of the femoral head is directly correlated to the range of motion of the femur before impingement occurs and the distance the head needs to travel after impingement occurs to escape the acetabulum (termed as the 'jump distance'). Therefore, larger femoral heads result in more stable prostheses. However, this stability comes at the cost of increased component wear [48, 49].

Three main considerations must be made when sizing the femoral stem:

(a) *Height:* This refers to the vertical height of the prosthesis, which is determined by the stem length, neck height, height of the head (not applicable in monoblock stems) and the depth to which the stem is inserted into the femoral canal.
(b) *Offset*: This is defined as the distance between the centre of rotation of the femoral head and to a line through the long axis of the stem. The offset has a direct correlation with the abductor lever arm and muscle strength; increasing or decreasing this will affect the joint reaction force through the hip joint, making it more or less stable. Moreover, offset also has an impact on soft tissue reconstruction and therefore needs to be accurately restored.
(c) *Version*: This refers to the orientation of the component in the femoral neck in the coronal plane. Normally, the femur is 10–15° anteverted. Excessive anteversion or retroversion will increase the likelihood of dislocation.

Figure 15.32 shows the components of a femoral stem. A centraliser is used to guide central positioning of the implant in the femoral canal.

Cemented femoral stems, like cemented acetabular components, tend to be cheaper than their uncemented counterparts. They can be roughly divided into composite beam and taper slip stems. Composite beam stems have a rough surface and rely on strong bone–cement and cement–implant interfaces, whereas taper slip stems tend to be polished, double or triple tapered, and subside within the cement mantle utilising the creep property of cement. In current practice, taper slip stems (e.g. the Exeter design) are more commonly used, with composite beam implants associated with an increased revision risk. Cemented stems are also associated with a lower risk of periprosthetic fractures. However, cementing can potentially result in 'bone cement implantation syndrome' (BCIS), a phenomenon arising secondary to embolisation of fat into the pulmonary and coronary circulations, resulting in hypotension and cardiac arrhythmias.

Uncemented femoral stems can be straight or curved and like their uncemented acetabular counterparts, tend to have porous coatings to facilitate bony ingrowth. As these stems rely on a strong bone–implant interface, they can have collars which rest on the calcar of the femur, providing additional stability and preventing subsidence. While uncemented implants do not carry the risk of BCIS, they are associated

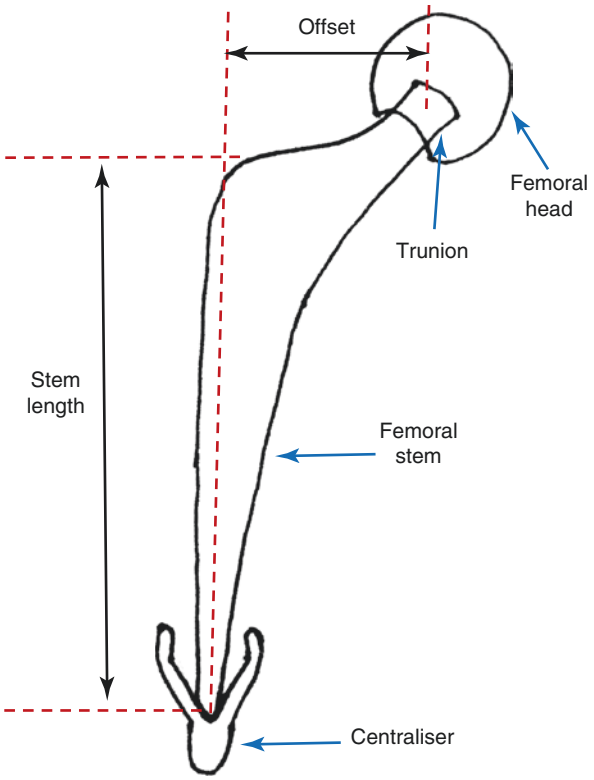

Fig. 15.32 Line diagram showing the components of a femoral stem

with a higher risk of intraoperative periprosthetic fractures as well as increased post-operative thigh pain. This is due to the uncemented implants being larger than their cemented counterparts to fill the femoral canal and achieve stable fixation. Larger implants increase the stiffness of the stem, and this mismatch between bone and implant stiffness results in proximal femoral stress shielding which itself can be a cause of post-operative thigh pain and later failure. Some uncemented designs may therefore have longitudinal grooves and flutes added to reduce stiffness and increase rotational stability [50, 51].

Bearing Surfaces

Bearing surfaces undergo wear, resulting in loose micro-particulate debris. These particles (usually UHMWPE) generate an inflammatory response by macrophages, which can further lead to cytokine mediated osteoclastic activity and ultimately loosen the implant via osteolysis.

As discussed, one strategy to reduce wear is to use a smaller femoral head, albeit at the cost of reduced stability. On the acetabular side, highly cross-linked UHMWPE, made by gamma irradiation and heat treatment, may be used, which is

Bearing type	Advantages	Disadvantages
Metal on metal	Low wear Low dislocation rate	Systemic toxicity Pseudotumour formation
Metal on polyethylene	Safe Cost effective	Aseptic loosening
Ceramic on polyethylene	Low wear	Aseptic loosening Fracture risk
Ceramic on ceramic	Low wear Scratch resistant Inert particles	Fracture risk Intolerant of malpositioning Squeaking

Fig. 15.33 Bearing surfaces for a THA

increasingly resistant to wear. Figure 15.33 describes the different bearing surface types currently available for use in a THA.

Ceramic bearings, while associated with low wear due to their hydrophilic surfaces, have a higher fracture risk due to their inherent brittleness. This may be mitigated by adopting a ceramic-on-polyethylene approach. Metal on metal bearing surfaces also have low wear, although they can raise metal (cobalt and chromium) ion levels, which can result in aseptic lymphocytic vasculitis-associated lesions (ALVAL) and tissue necrosis. They have therefore recently fallen out of favour, although they can work very well in carefully selected patients. Patients with metal on metal implants in the UK now undergo planned lifelong monitoring for signs of systemic toxicity [52].

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Clinical Examination of the Knee Joint (Basic and Surface Anatomy)

16

Abhishek Vaish and Raju Vaishya

Introduction

Knee problems are common in every age group. Younger patients usually have sports injuries, whereas older patients have more problems related to degenerative joint disease or osteoarthritis (OA) [1]. The success of treatment of a knee problem depends on accurate diagnosis and timely management. History and physical examination are the best tools for a clinician to reach a diagnosis. The radiological investigations such as plain radiographs and magnetic resonance imaging (MRI) are reserved in doubtful cases to confirm the diagnosis, to document injury, and to assess the severity and planning of treatment.

The clinical examination of the knee comprises of the following:

A. Look: The clinical examination begins as the patient walks into the clinic.
   - It is essential to assess the walking style (gait) of the patient. The clinician should observe if the patient is comfortable walking unaided or needs assistance with a stick or walker.
   - The facial expression of the patient speaks a lot if the patient winces in pain and avoids loading on the affected limb. The clinician should comfortably sit the patient down on an examination table and make sure that he/she stands on the side, which is affected and is examined. The part to be examined should be fully exposed, keeping in mind the patient's privacy and comfort. As a rule, one should leave the most painful part of the test reserved for the last as it may lead to falsely positive tests.
   - When the patient is comfortably lying down on the examination table, look at the attitude of the affected limb (compared to the opposite limb with both limbs parallel to each other). Appreciate any fixed flexion deformity,

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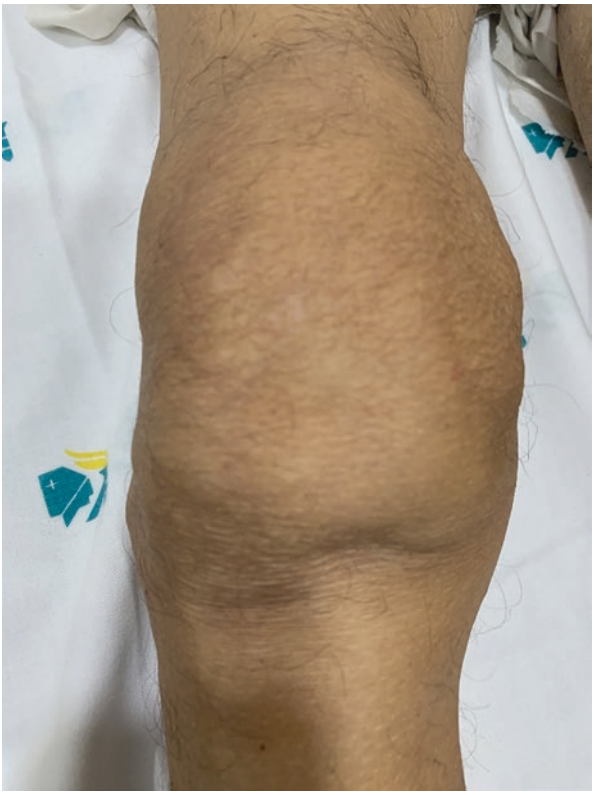

Fig. 16.1 Knee swelling, with obliteration of all the normal depressions around the knee and muscle wasting of the thigh

rotational deformities, and gross muscle wasting as comparable to opposite thigh/calf at mid-level. Also, look for suprapatellar pouch fullness (Fig. 16.1) which may be suggestive of effusion/hemarthrosis. Also, one should rule out any abrasions/lacerations and prominent blood vessels around the injured part.

- One should also appreciate the mechanical alignment of lower limbs in coronal and sagittal planes. The most common sagittal deformities are varus (Fig. 16.2) and valgus deformities.

B. Feel: The clinician should make sure that his examining palms are not too cold as this may take the patient by surprise and cause unwarranted muscular contractions/guarding.

- Compare the local temperature of the affected knee with the opposite side. In cases of inflammation, there is a rise in local temperature.
- Site of tenderness around the knee joint is an excellent indicator to find the pathology (Fig. 16.3). Check the medial and lateral joint lines.
- The overlying skin should be examined to see if there are any signs of puckering, skin adherence to the underlying structures, blisters, or dilated veins. Presence or absence of skin wrinkles is crucial in extensive internal degloving around the knee joint. It is estimated to be the best predictor for the timing of surgical intervention without causing further harm to the soft tissues around.

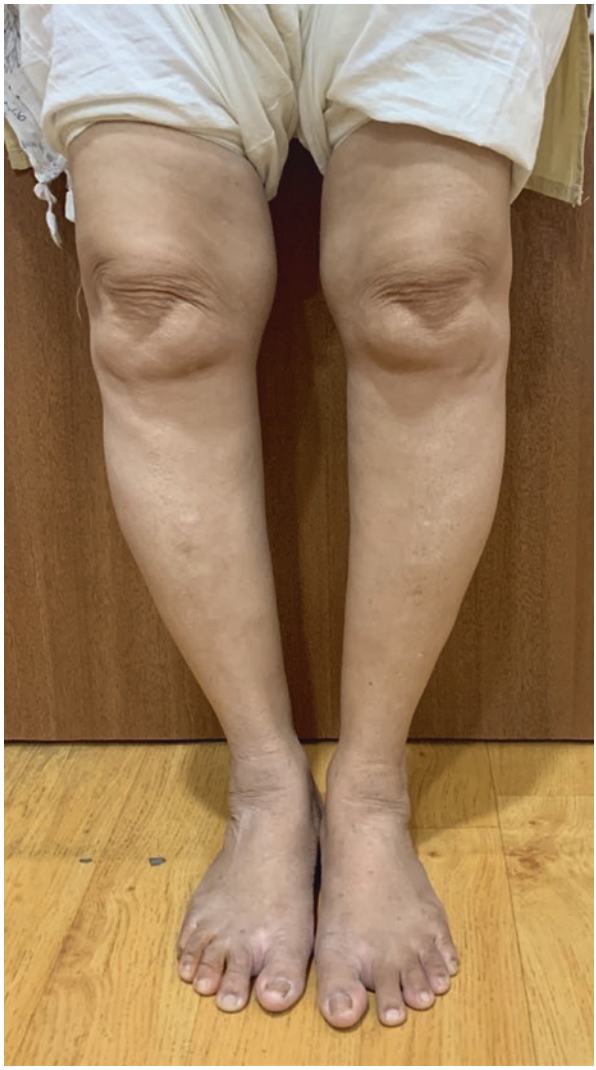

Fig. 16.2 Bilateral genu varum (bow leg) deformity of the knees

C. Move: The knee joint should be examined for range of motion (ROM) and compared with the opposite limb. It gives an idea about the deficit in the range for that patient. The movements possible around the knee joints are flexion-extension, lateral, and medial rotations. These movements should be checked both actively and passively.
   - If the passive movements are restricted, then that indicates a possible mechanical block or arthrofibrosis.
   - If passive movements are full, but active movements are restricted, then this is suggestive of muscle inhibition or weakness. These patients may need physiotherapy or muscle stimulation.

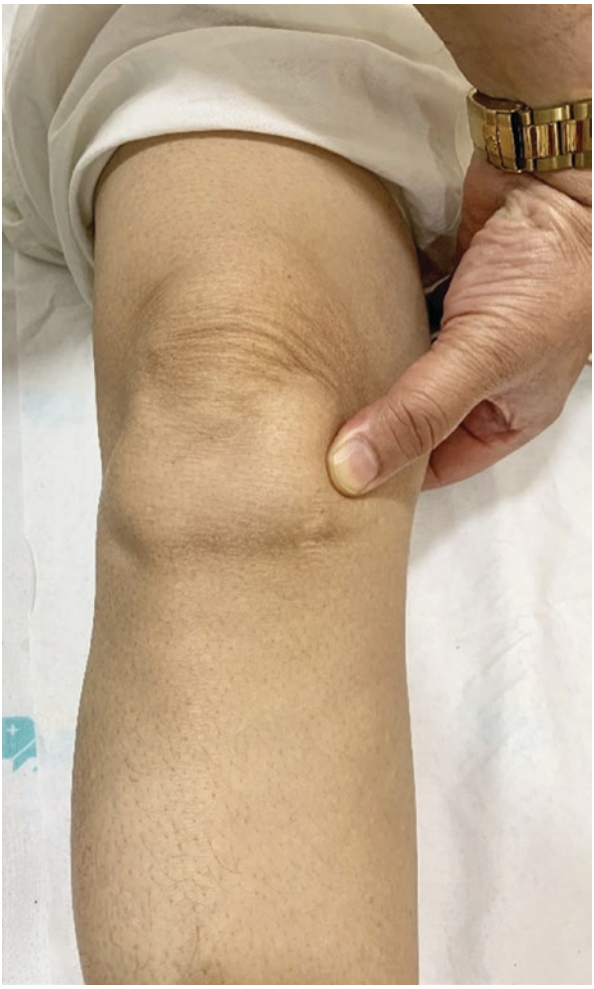

Fig. 16.3 Eliciting joint line tenderness

D. Measure:
   - Thigh and calf girth can be measured and compared with the opposite limb (measure from bony prominence as a reference on both sides). If there is a difference of more than 2.5 cm, then it is significant muscle wasting (Fig. 16.1). Limb length and segmental lengths may be necessary to assess in cases such as nonunion, congenital deformities.
   - Use a goniometer to measure the ROM and angular deformities.

E. Special Tests: There are several special tests for clinical diagnosis of the knee. Some most commonly performed tests are as follows:
   1. Tests for isolated ACL injuries:
      - Anterior Drawer Test—It is performed with the knee flexed to 90° (Fig. 16.4). It is crucial to feeling the relaxation of the hamstrings with two fingers to avoid false-negative tests. With both the hands placed

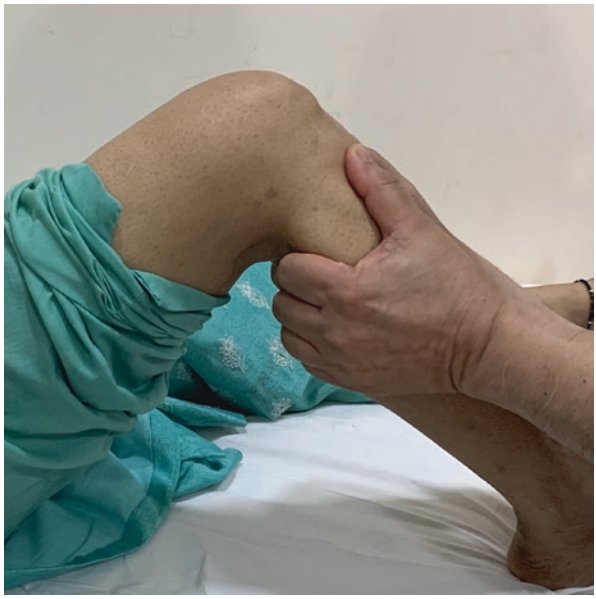

Fig. 16.4 Anterior drawer test

behind the tibial condyles, the tibia is pulled forward [2]. In a positive test, there is an increased translation of the upper tibia anteriorly, compared to the opposite knee. Translation can be graded into three kinds of grading:

1. Grade I—Translation less than 5 mm compared to the opposite limb.
2. Grade II—Translation between 5 and 10 mm compared to the opposite limb.
3. Grade III—Translation more than 10 mm compared to the opposite limb.

The drawback with anterior drawer test is that in an acute setting when the knee is swollen and painful, it is often difficult for the patient to flex the knee to 90°. Also, a mechanical block due to a torn meniscus may cause a "door stopper" effect leading to a false-negative test.

- Lachman test—It is performed with 30° flexion of the knee (Fig. 16.5); there is increased anterior excursion compared to the opposite knee. In acute injuries, there is a soft mushy endpoint.

  This test has the benefit that it can be performed easily in acute settings and also with concomitant meniscal injuries.
- Pivot shift test—It is a dynamic test which is more diagnostic of ACL tear than the other described tests. However, intactness of medial collateral ligament and cooperation of the patient with full muscle relaxation are its prerequisites to perform this test. In this test, the patient lies in the supine position, and a combination of an axial load and valgus force is applied by the examiner, during a knee flexion from an extended position. In a positive test (ACL tear), there is a subluxation of the lateral tibial condyle [3].

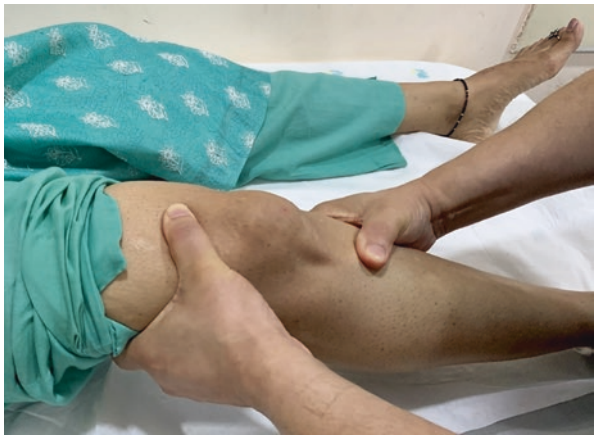

Fig. 16.5 Lachman test

2. Tests for isolated PCL injuries:
   - Posterior Sag test—Place both knees at 90° flexion on the examination table and look tangentially from the sides to appreciate a posterior sag of the tibia compared to the unaffected opposite side. A definite posterior sag is suggestive of insufficiency in the posterior cruciate ligament.
   - Posterior Drawer Test—This test is also referred to as the reverse anterior drawer test. It is performed with the knee in 90° flexion, thumbs supporting the medial and lateral tibial condyles and the rest of the three fingers posteriorly on the hamstrings to feel the relaxation. Compare the posterior excursion of tibia more than 5 mm compared to the opposite side. It is essential to check the start point in this test. In cases where both anterior and posterior cruciate ligaments are insufficient, it is easy to make clinical error between positive anterior and posterior drawer tests.
   - Quadriceps Active Test—This test is performed to assess the integrity of the PCL. With the patient in the supine position, the patient's involved limb is placed in a position of 45° of hip flexion and 90° of knee flexion [4]. Look for the tibia to "sag" compared to the position of the femur. The examiner should then sit on the foot of the affected limb to stabilize. Next, have the patient actively contract their quad muscle. A positive test occurs if the patient's tibia shifts forward. The importance of this test is that posterior cruciate ligament is responsible for resisting against the excessive posterior translation of the tibia on the femur, due to its attachments posteriorly on the tibial plateau and anteriorly on the lateral side of the medial femoral condyle. In the position of 45° of hip flexion and 90° of knee flexion, gravity places a force on the tibia that pulls the tibia posteriorly but is blocked by an intact PCL. In the absence of a PCL, the tibia appears to "sag." When the quad contracts, an anterior translation of the tibia on the femur occurs due to the attachments of the quadriceps muscles. The most common mechanism for PCL injury is a posterior translation at 90° of knee flexion. While the PCL can be ruptured through hyperextension

and hyperflexion as well, it is unlikely that it is the only ligament torn in these injuries. The ACL is stressed more than the PCL in both hyperflexion and hyperextension.

3. Tests for menisci:
   - McMurray's test—With the patient supine, place the patient's tested leg in maximal hip and knee flexion. While palpating the joint line, apply a valgus force to the knee while simultaneously externally rotating and extending the knee completely. Place the tested leg back in maximal hip and knee flexion. While palpating the joint line, apply a varus force to the knee while simultaneously internally rotating and extending the knee completely. A positive test occurs when pain or clicking/thudding is produced [5].

     The importance of this test is that the menisci are crescent-shaped structures that help increase the concavity of the tibia for acceptance of the femoral condyles. They attach anteriorly and posteriorly to the intercondylar area of the tibia. Laterally, they adhere to the tibia loosely via the coronary ligaments (this allows some sliding of the menisci). With its concave shape, the meniscus acts to decrease compressive forces of the knee by increasing the force distribution of the femoral condyles onto the tibia. Due to decreased blood supply to the inner aspects of each meniscus, an injury in this area is less likely to heal. This test stresses each meniscus by adding a rotary force to a flexion/extension pattern. When the tibia is externally rotated, the medial meniscus is primarily being assessed, while the posterior portion of the lateral meniscus may be assessed as well. When the tibia is internally rotated, the lateral meniscus is being tested. These motions stress the structure, and when combined with palpation of the joint line, clicking can be felt. The meniscus is usually injured through twisting motions on a slightly bent knee or sometimes through muscular contractions of the semimembranosus, quadriceps, or popliteus, due to their attachments to the menisci. Whenever an injured meniscus is present, or a meniscus is removed, the force distribution characteristic is loss and increased compressive forces are placed on the knee. These individuals have been found to have increased development of knee osteoarthritis.
   - Apley's Grinding test—With the patient lying down on the stomach, flex the knee under examination to 90° and with one hand press the foot down toward the examination table but at the same time giving internal and external rotation forces [6]. Notice if the patient winces in pain or complains of sudden catch suggestive of a meniscal tear.
   - Thessaly test—Have the patient stand on the test leg with the knee bent to 20° of flexion (the opposite leg is flexed behind the patient). The patient may place his/her hands on the hands of the examiner for balance during the test. The patient then rotates the knee medially and laterally three times each direction. A positive test occurs when the patient experiences joint line discomfort or if locking/catching occurs.

4. Tests for patellofemoral joint:
   - Patellar tap/fluctuation—The patient is lying in supine with the leg extended. The examiner puts pressure on the proximal side of the knee to squeeze the fluid out of the suprapatellar pouch (Fig. 16.6). The fluid can be moved under the patella while maintaining the pressure on the suprapatellar pouch; the examiner uses his/her other hand to press upon the medial and lateral recesses forcing the fluid under the patella [7]. Tap down the patella with the finger/thumb to create an upward and downward movement and a palpable "click" as the patella hits the underlying femur (Fig. 16.7). If the test is negative, the femur and the patella are already in contact. A positive test is when the patella can be felt to move down through the fluid and rebound on the patella. The test can be falsely positive; therefore, we must always test both the knees to compare.

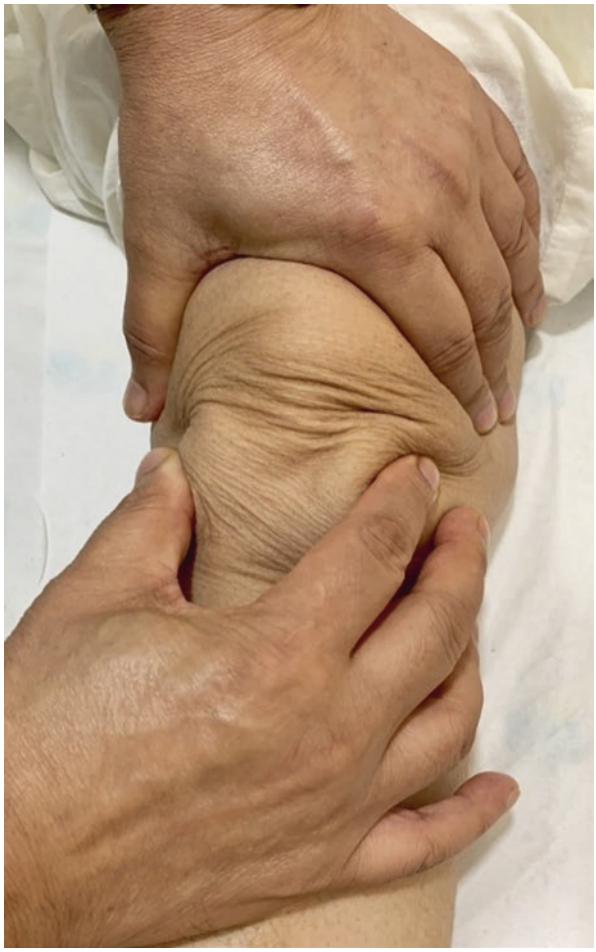

Fig. 16.6 Cross-fluctuation test for knee effusion

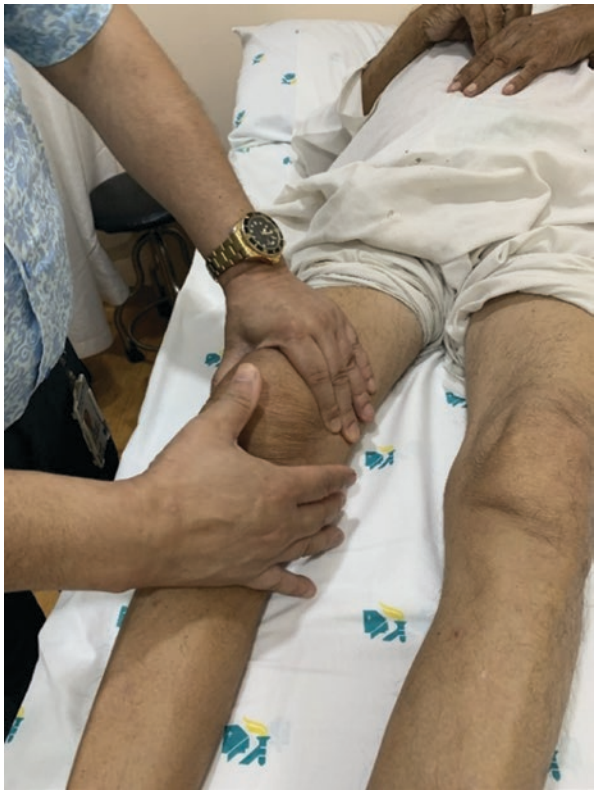

Fig. 16.7 Patellar tap test for knee joint effusion

- Apprehension test—This is a test that is designed for the clinical identification of lateral patellar instability. The moving patellar apprehension test is performed in two parts. Part 1 is a provocation oriented test. The examiner places the knee to be examined into full extension. A lateral force is applied to the patella with the examiner's thumb (Fig. 16.8). The examiner then moves the knee from full extension to 90° of flexion and then returning to full extension while maintaining the laterally applied force on the patella. The second aspect of the test (part 2) consists of a symptom alleviation maneuver. The examiner repeats the part 1 of the test with a medially applied force on the patella. The examiner places the knee to be examined into full extension. A medial force is applied to the patella with the examiner's index finger. The examiner then moves the knee from full extension to 90° of flexion and then returning to full extension while maintaining the medially applied force on the patella. A positive test consists of orally expressed apprehension or apprehensive quadriceps recruitment on the provocation test (part 1) and alleviation of these symptoms with normal ROM within the test ROM in part 2 of the test [8].
- Patellar grind test—This test is performed to elicit signs of patellofemoral arthritis, patella femoral syndrome, and chondromalacia. The patient is

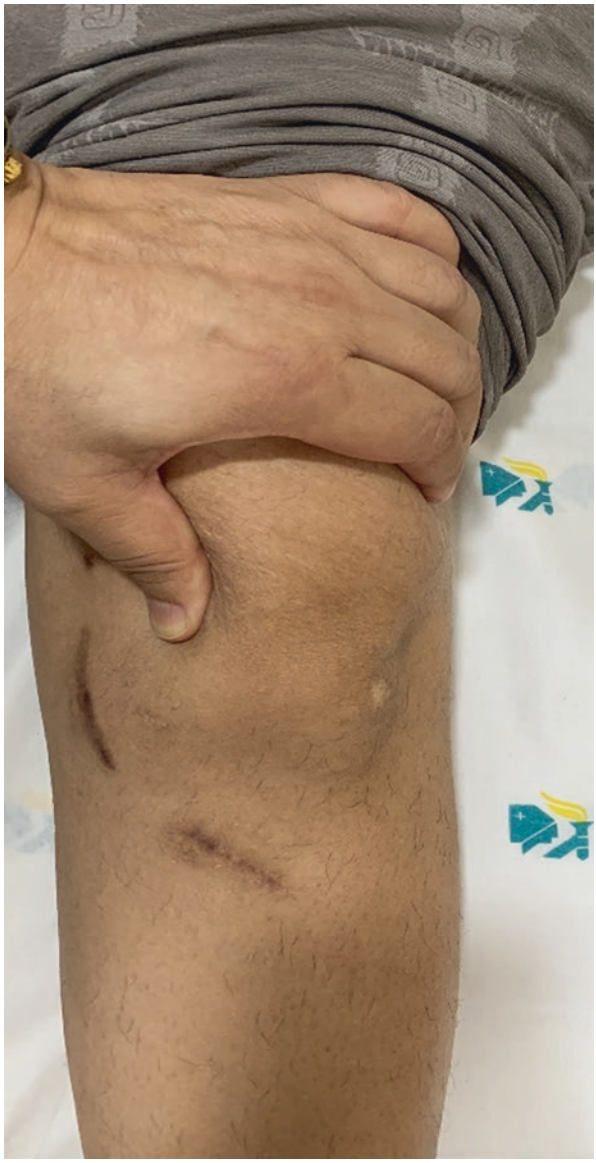

Fig. 16.8 Patella Apprehension test

positioned in supine or long sitting with the involved knee extended. The examiner places the webspace of his hand just superior to the patella while applying pressure (Fig. 16.9). The patient is instructed to gently and gradually contract the quadriceps muscle. A positive sign on this test is a pain in the patellofemoral joint.

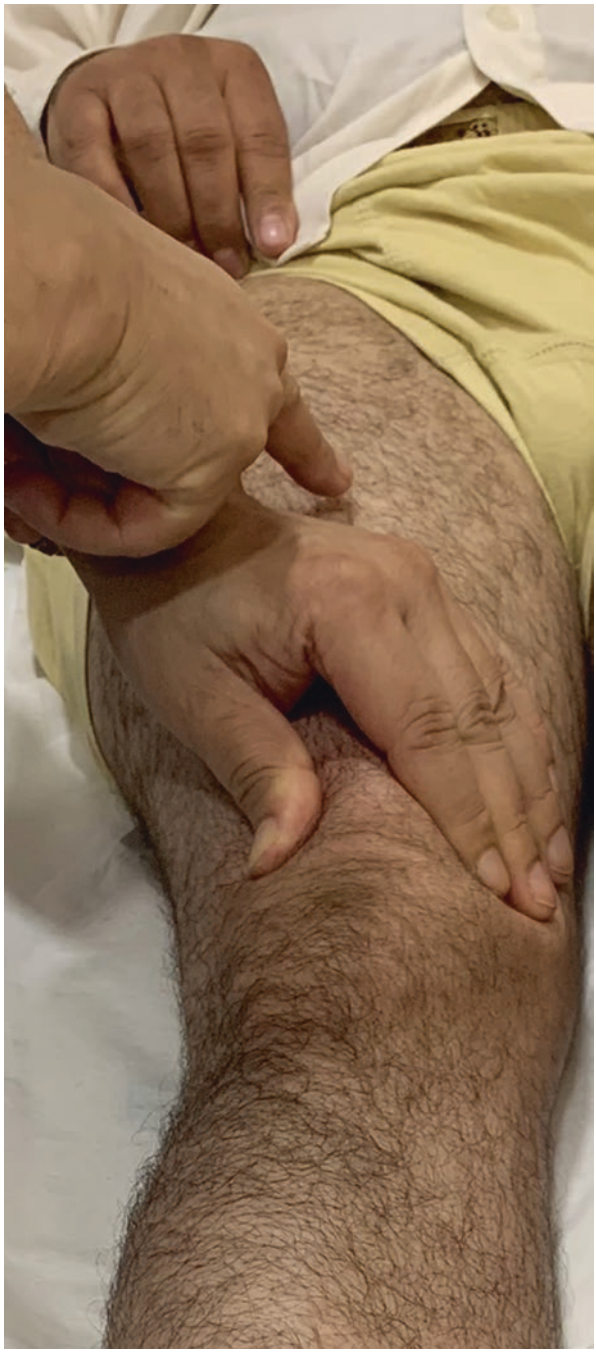

Fig. 16.9 Patella grind test

5. Tests for mediolateral stability:
   - Varus stress test—Performed to assess the integrity of the LCL. The patient's leg should be relaxed for this test. The examiner should passively bend the affected leg to about 30° of flexion. While palpating the lateral joint line, the examiner should apply a varus force to the patient's knee. A positive test occurs when pain or excessive gapping occurs (some gapping is normal at 30°). Be sure to not include rotation of the hip in your application of force. Next, the examiner should repeat the test with the knee in neutral (0° of flexion). A positive test occurs when pain or gapping is produced. There should be no gaping at 0°. The lateral collateral ligament is vital for resisting varus force at the knee due to its attachments along the femur and fibular head. With the fibular nerve also located around the fibular head, any injury with a mechanism of a varus force to the knee could potentially stress the fibular nerve as well. Other tissues at risk with these injuries include the PCL and arcuate complex, especially if the injury varus force is combined with extension. At 0°, there is usually no gapping that occurs when a varus stress is applied, so if gapping occurs during the test, severe injury is suspected, i.e., ACL, PCL, LCL, and capsule. In the position of 30°, some gapping occurs, because the LCL and other structures are no longer stressed maximally. The LCL is a very thick, fibrous ligament that can be palpated with position stress in the Fig. 16.4 position (think of the attachments!). Due to the difficulty of varus forces being an injury mechanism (because of the shielding by the opposite lower extremity), isolated LCL injuries are relatively rare [9].
   - Valgus stress test—This test helps to assess the integrity of the medial collateral ligament (MCL). The patient's leg should be relaxed for this test. The examiner should passively bend the affected leg to about 30° of flexion. While palpating the medial joint line, the examiner should apply a valgus force to the patient's knee (Fig. 16.10). A positive test occurs when pain or excessive gapping occurs (some gapping is normal at 30°). Be sure to not include rotation of the hip in your application of force. Next, the examiner should repeat the test with the knee in neutral (0° of flexion). A positive test occurs when pain or gapping is produced. There should be no gaping at 0°.

     The medial collateral ligament is vital for resisting valgus force at the knee due to its attachments along the femur, meniscus, and tibia. The MCL also plays a significant role in restraining tibial external rotation. The surgical severing of the superficial portion of the MCL was shown to increase tibial external rotation at 90° by about three times (Ellenbecker, 2000). According to Neumann, the MCL attaches to the medial epicondyle proximally and posterior to the distal attachment of the pes anserinus distally on the anteromedial tibia. The deeper fibers of the MCL are shorter than the superficial fibers and also attach to the posteromedial capsule, meniscus, and semimembranosus tendon. Because the deeper fibers are shorter than the superficial fibers, they are more likely to be

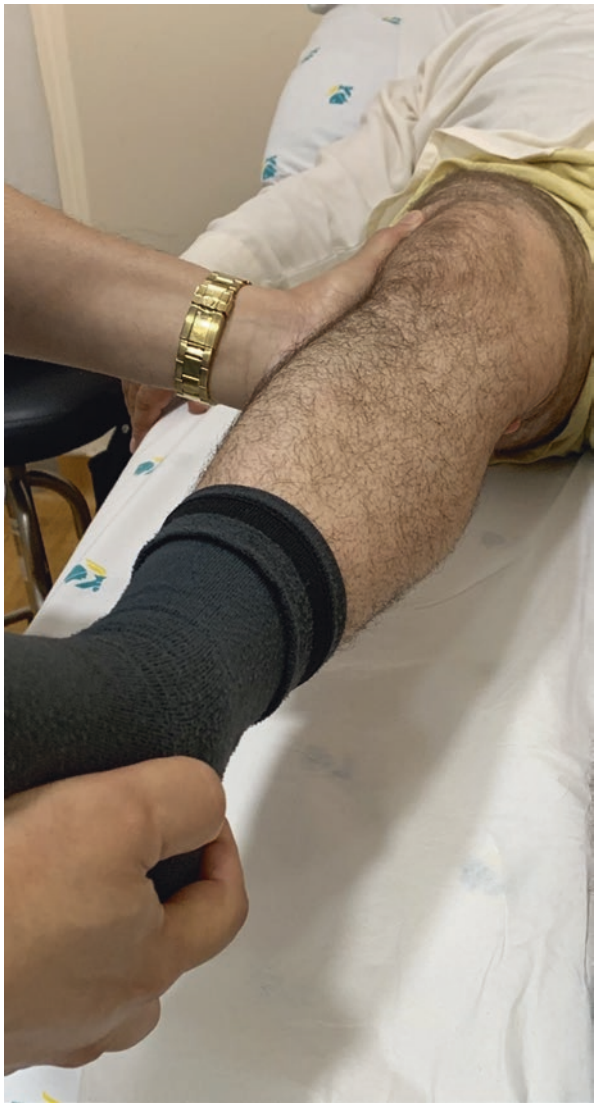

Fig. 16.10 Valgus stress test

injured when stressed with a valgus force, even though the superficial fibers provide the primary resistance to valgus force. The superficial fibers, on the other hand, are more likely to be stressed with external rotation of the tibia on the femur (or internal rotation of femur on tibia). With the attachment of the MCL to the meniscus, whenever the mechanism of injury affects the MCL, be sure to check the meniscus for injury as well. At 0°, there is usually no gapping that occurs when a valgus stress is applied, so if gapping occurs during the test, severe injury is suspected, i.e., ACL, PCL, MCL, and capsule (no gapping because the

MCL, posteromedial capsule, hamstrings, oblique popliteal ligament, and parts of the ACL are most taut in full extension). In the position of 30°, some gapping occurs, because the MCL and other structures are no longer stressed maximally, but the MCL is the primary stabilizer in this position. The MCL overall is one of the most essential ligaments for the stability of the knee. With a hypermobile knee, due to a sprained MCL, it is essential to take extra precautions to decrease the risk of further injury. With a lax MCL, the ACL becomes increasingly stressed with valgus forces, especially at 45° of flexion [10]. Remember the MCL is the primary valgus restraint in the flexed knee; without it, the ACL is prone to injury.

6. Tests for posterolateral instability:
   - External rotation recurvatum test—In this test, the examiner determines if there is an increased amount of knee hyperextension compared to the contralateral side. The test is performed while applying a stabilizing force to the distal thigh while one lifts the great toe to assess the amount of knee recurvatum present. This is usually measured by the amount of heel height in cm. In general, it is measured on the medial aspect of the foot and compared to the healthy contralateral knee. Studies have demonstrated in the face of a posterolateral knee injury; an increased amount of recurvatum is usually indicative of a combined anterior cruciate ligament tear.
   - Dial test in 30° and 90°—The test can be done with the patient either in the prone or supine position. The aim of the test is to inspect the external rotation (foot-thigh angle, best measured in a clinical setting at the knee joint while the knees are at 30° and 90° of flexion). The clinician flexes the patient's knees to 30° and places both hands on the feet of the patient, cupping his heels. A maximal external rotation force is then applied, and the foot-thigh angle is measured and compared with the other side. The knees are then flexed to 90°, and again an external rotation force is applied (Fig. 16.11), and the foot-thigh angle is measured again. The test is positive when there is more than 10° of external rotation in the injured knee compared to the uninjured knee [11]. Two types of different injuries can be identified:
     (a) An isolated injury to the PLC: more than 10° of external rotation in the injured knee is present at 30° of flexion, but not at 90° of flexion.
     (b) Instability of the PCL: more than 10° of external rotation in the injured knee is present at 90° of flexion, but not at 30° of flexion.

   A combined injury: more than 10° of external rotation in the injured knee is present at 30° and 90° of flexion. This is an injury of the PCL and the PLC.
   - Reverse pivot shift test—The reverse pivot shift test helps to diagnose acute or chronic posterolateral instability of the knee. A significantly positive reverse pivot shift test suggests that the PCL, the LCL, the arcuate complex, and the popliteal fibular ligament are all torn. The reverse pivot shift test begins with the patient supine with the knee in 90° of flexion. Valgus stress is then applied to the knee with an external rotation force.

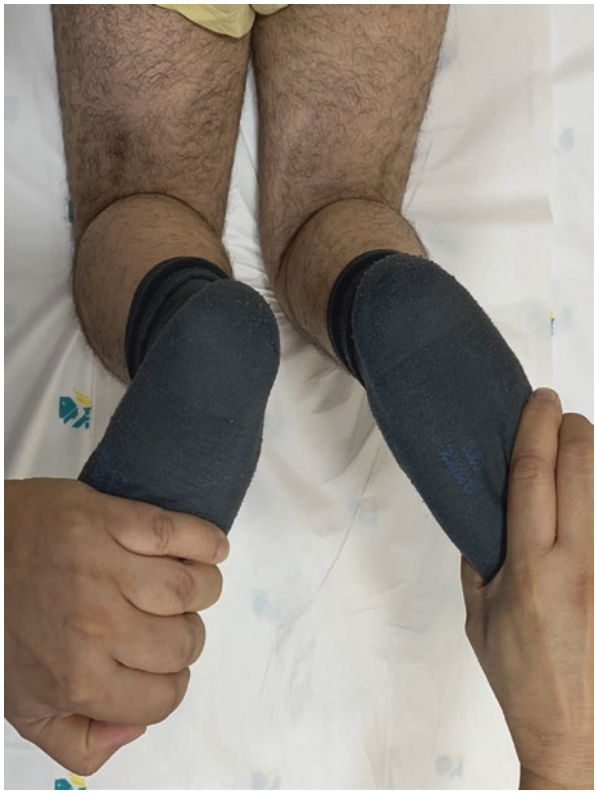

Fig. 16.11 Dial test in 90° flexion

Bring the knee from 90° of flexion to full extension. The tibia reduces from a posterior subluxed position at about 20° of flexion. A shift and reduction of the lateral tibial plateau can be felt as it moves anteriorly from a posteriorly subluxed position. A "clunk" occurs as the knee is extended. This is called reverse pivot shift because the shift of the lateral tibial plateau occurs in the opposite direction of the real pivot shift (seen in ACL tears). If the tibia is posterolaterally subluxed, the iliotibial band will reduce the knee as the IT band transitions from a flexor to extensor of the knee. It is imperative to compare this test to the contralateral knee.

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ACL Injuries of the Knee Joint

17

Abhishek Vaish and Raju Vaishya

Introduction

Sports injuries in the younger population are rising due to the increasing popularity of sports and an increase in awareness about health. An anterior cruciate ligament (ACL) is the most commonly injured ligament of the knee. It may be associated with other internal structural injuries of the knee, such as the meniscus, cartilage, and other ligaments. As ACL injuries mostly produce a dramatic and significant instability of the knee and affect the younger population, these have to be dealt in time to restore the knee function and to restore their activities of daily living. We shall discuss the importance of ACL injuries, its burden, biomechanics of this ligament, and the management of its injuries, in this chapter. The importance of rehabilitation after an ACL injury is crucial for an excellent functional outcome and to avoid knee stiffness. As these injuries involve young and active individuals, reinjury or failure of previous ligament reconstructions is also known and hence the rate of revision ACL surgeries has seen an increase recently. Chronic ACL insufficiency can cause damage to the articular cartilage and thus early degenerative arthritis, meniscal tears, and stretching of secondary stabilizers such as collateral ligaments.

Incidence

The ACL injuries are much more common than the PCL injuries. The age- and sex-adjusted annual incidence of ACL tears was 68.6 per 100,000 person-years [1], whereas the annual incidence of isolated, complete PCL tears was 1.8 per 100,000 [2]. There has been a steady increase in the interest and understanding of ACL injuries in the last four decades [3].

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Mostly there is complete tear of an ACL after a twisting injury of the knee and the partial tears of ACL are relatively rare and their incidence ranges from 10 to 28%. It is reported that there is a 15–66% chance of this lesion to progress to a complete tear and hence the partial tear must be managed appropriately [4].

Anatomy and Biomechanics

The ACL is composed of collagen fibers which are longitudinally oriented. It is an extra synovial but an intra-articular structure of the knee. Its blood supply comes primarily from the middle geniculate artery, and its nerve supply is through the posterior articular nerve (a branch of tibial nerve). The approximate length of an ACL is 35 mm and has two principal bundles comprising of an anteromedial (AM) bundle and a posterolateral (PL) bundle. The AM bundle provides anteroposterior stability to the knee whereas the PL bundle provides rotational stability to the knee. The AM bundle is tight in flexion, and the PL bundle is tight in extension. The AM and PL bundles are parallelly aligned in extension and cross each other in the flexion. At 30° flexion, the tension is least on ACL. The ACL can bear an ultimate load of 1700 ± 250 N, according to biomechanical studies.

Risk Factors

Risk factors for ACL tears include excessive demands on unconditioned knees, knee flexion angle during landing, limb alignment, notch size, and hormonal fluctuations. Joint hypermobility is also considered a contributor. Its prevalence is higher in females and in Africans (than Caucasians) and decreases with age. Joint hypermobility is reported to be more common in patients with ACL injury compared to normal population and hence predispose them to ACL injuries [5].

Clinical Presentation

History

Most commonly a patient presents with a history of an injury after playing sports. There is a history of twisting of the knee, classically as a sudden deceleration or cutting movements leading to hyperextension. The patient describes hearing or feeling a pop sound, followed by an inability to stand up immediately after this injury. There is immediate swelling of the knee and inability to weight-bear fully, on the injured knee. In chronic cases, the typical history of giving way of the knee and intermittent locking of the knee is seen.

Examination

The injured knee is swollen with hemarthrosis, after acute injuries, and the knee may be in an attitude of flexion. In chronic cases, there is often wasting of the thigh muscles, with or without knee swelling. Palpation of the knee may reveal the local temperature is mildly raised, and the signs of fluid in the knee (e.g., cross-fluctuations, ballottement, and patellar tap) may be positive. The knee movements are restricted in acute injuries and usually not in chronic cases.

Special Tests

To diagnose an ACL tear clinically requires some special tests, which are being described below:

- Lachman test: It is performed with 30° flexion of the knee (Fig. 17.1); there is increased anterior excursion compared to the opposite knee. In acute injuries, there is a soft mushy endpoint.
- Anterior drawer test: It is performed with the knee flexed to 90° (Fig. 17.2). With both the hands placed behind the tibial condyles, the knee is pulled forward. In a positive test, there is increased translation of the upper tibia anteriorly, compared to the opposite knee.
- Pivot shift test: It is a dynamic test which is more diagnostic of ACL tear than the other described tests. However, intactness of medial collateral ligament and cooperation of the patient with full muscle relaxation are its prerequisites to perform this test. In this test, the patient lies in the supine position, and a combination of an axial load and valgus force is applied by the examiner, during a knee flexion from an extended position (Fig. 17.3). In a positive test (ACL tear), there is a subluxation of the lateral tibial condyle.
- Objective tests: The laxity related to ACL tears can be objectively tested and recorded by various instruments like a knee ligament arthrometer (KT-1000/KT-2000, Fig. 17.4), which can precisely quantify the ACL injuries and grading.

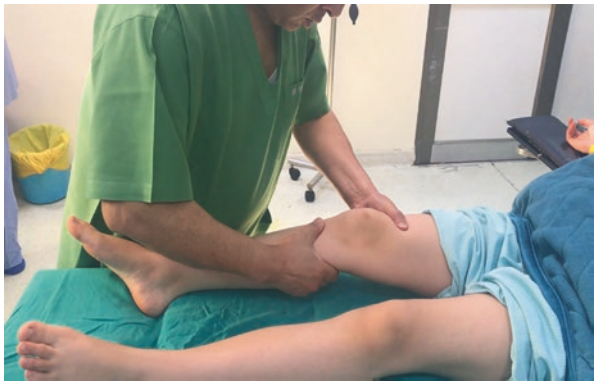

Fig. 17.1 Clinical photograph demonstrating the Lachman test

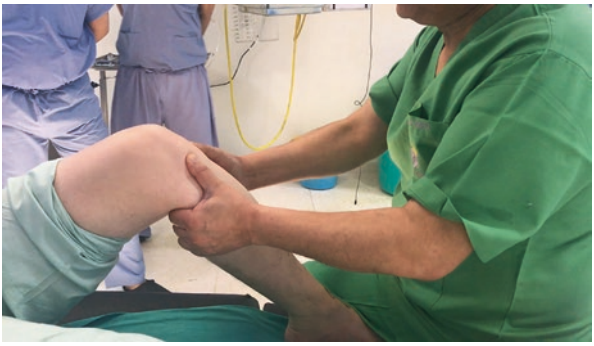

Fig. 17.2 Clinical photograph demonstrating the anterior drawer test

Fig. 17.3 Clinical photograph demonstrating the Pivot Shift Test

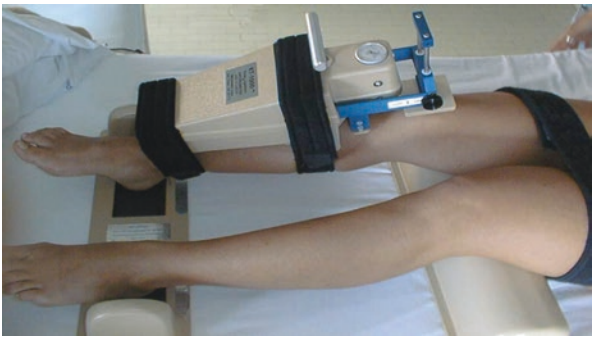

Fig. 17.4 Clinical photograph demonstrating the knee ligament arthrometer

Radiological Investigations

Plain radiographs are usually not helpful in the diagnosis of an ACL injury. However an associated fractures (avulsion, lateral femoral condyle, and Segond fractures) could be diagnosed and may provide a clue to the tear of ACL.

Computed tomography (CT) is very useful and accurate in the diagnosis of avulsion bone fragment and an undisplaced fractures of the condyles.

Magnetic resonance imaging (MRI) is the most accurate investigation tool to diagnose an ACL injury, with a specificity and sensitivity of more than 90%. It is considered as a gold standard test. Several primary and secondary signs to diagnose an ACL tear on the MRI have been described [6]. A discontinuity in the ligament and an abnormal contour of the ACL, along with an empty notch sign, are characteristics of an ACL tear (Fig. 17.5). Mucoid degeneration and the ACL sometimes resemble an ACL tear on the MRI and hence must carefully be evaluated [7] .

Natural History

The intra-substance tear of the ACL rarely heals on its own, and the symptomatic instability often requires ACL reconstruction using a graft. However, the bony avulsion injuries, especially in adolescent, may heal spontaneously. The course of healing in ACL injuries is determined by many factors such as age, activity level, the extent of the injury, physiotherapy, and time since injury. People who do not give these injuries adequate time and environment to heal usually suffer from having concomitant injuries (Figs. 17.6 and 17.7) to other structures in due time, such as

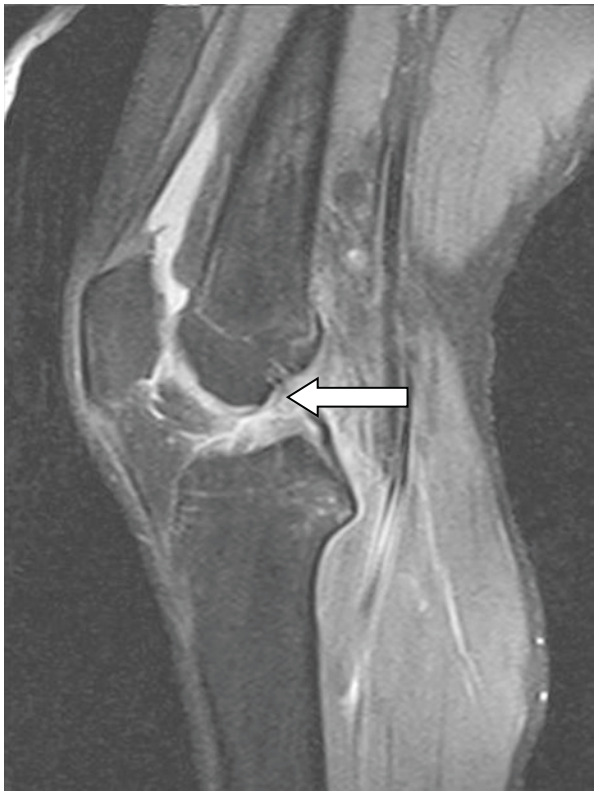

Fig. 17.5 An MRI image showing complete disruption of the ACL

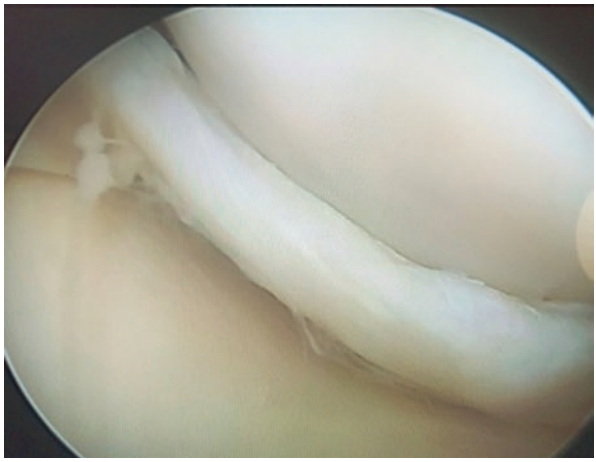

Fig. 17.6 Arthroscopic view of a bucket handle tear of the medial meniscus in an untreated case of ACL tear

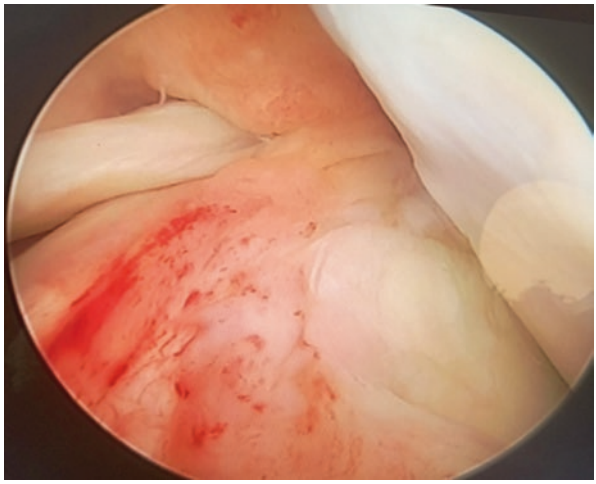

Fig. 17.7 Arthroscopic view of a posterior capsular tear in a chronic ACL tear

cartilage (21–31%) and menisci (50–70%). The other commonly associated injuries with ACL tears are O'Donoghue's unhappy triad, Segond fracture, posteromedial corner injury of the knee, and meniscocapsular separation [6].

Treatment

The treatment of an ACL injury depends on the preinjury activity level of the patient, age, and amount of instability. The treatment may range from a conservative to operative management.

A. Conservative Treatment

It includes aggressive rehabilitation, pain management, and counseling about activity level. Patient's motivation regarding physiotherapy should be assessed

Fig. 17.8 Photograph of ACL knee brace

beforehand, and compliance should be checked on regular follow-up. Various specialized braces (Fig. 17.8) are available to provide stability to the knee.

B. Operative

The ACL reconstruction is an accepted and established surgical technique for ACL injuries and is now considered as a gold standard in the management of symptomatic ACL tears. ACL reconstruction has been shown to restore the joint stability and improve the functional outcome.

1. Repair (Isolated ± Augmentation)
   - Mid-substance tear: The results of a direct repair procedure are not very good, because the healing capacity of ACL is limited. Hence, some people have tried to augment the healing of a partially torn ACL by using an injection of mesenchymal cells (MSCs) and platelet-rich plasma (PRP).
   - Bony avulsion: Most commonly bony avulsion injuries of ACL occur in adolescent individuals, with an open physes, through the tibial attachment. These

can be repaired using screws, staples, and sutures with good functional outcomes. This procedure can either be performed by an open or arthroscopic procedure.

2. Extra-articular Reconstruction Procedures

There are various extra-articular procedures (McIntosh, Andrew, Losee). However, these procedures are not very popular nowadays. These are reserved mainly for children with ACL insufficiency, where an intra-articular procedure cannot be done due to open physes.

3. Intra-articular Reconstruction Procedures

Surgeon preferences in ACL reconstruction differ considerably among the arthroscopic surgeons. There is majority consensus for using hamstring autograft (single bundle) with a suspensory fixation on the femoral side and an aperture fixation on the tibial side. Transportal technique of making the femoral tunnel and preservation of amputation stump are the preferred methods. However, differences exist over the timing of surgery, rehab after surgery, pain management, etc. [8, 9]

- Timing of Surgery

  It was earlier believed that the functional outcomes of ACL reconstruction are best achieved if the surgery is performed after the resolution of initial inflammation and achievement of the full range of motion. However, nowadays, there has been a change in this trend, and many ACL reconstructions are being performed soon after the initial injury. The only important factor which determines an excellent functional outcome is proper rehabilitation after an ACL reconstruction. It avoids the development of knee stiffness.

- Graft selection

  Autografts have better chances of incorporation, revascularization, and healing compared to allografts and therefore preferred. The synthetic grafts which were popular in the 1980s have mostly gone into disrepute and only being used infrequently. The most commonly used autografts are hamstring (Fig. 17.9) and bone-patellar-tendon-bone graft (BPTB). Peroneal longus, tibialis anterior, quadriceps, and Achilles tendon grafts are the other lesser commonly used options. It has been documented that BPTB grafts have lesser chances of failure and laxity. However, this graft is associated with a high incidence of anterior knee pain, and there could be a fracture of the patella during its harvesting. Allografts are excellent alternatives; however, their availability is still not universally present and is expensive, and there could be a risk of disease transmission.

- Single versus double bundle reconstruction

  The majority of ACL reconstructions are being done using a single bundle of quadrupled hamstring grafts (Fig. 17.10). However, some surgeons prefer to recreate the two native bundles of ACL (AM and PL bundles) by using double bundle of the hamstring grafts for their reconstruction.

- Graft placement

  Accurate graft placement is crucial in ACL reconstruction (Fig. 17.11) surgery to achieve satisfactory functional outcomes and to avoid failure. If the femoral tunnel is too anteriorly placed, it reduces flexion and causes early

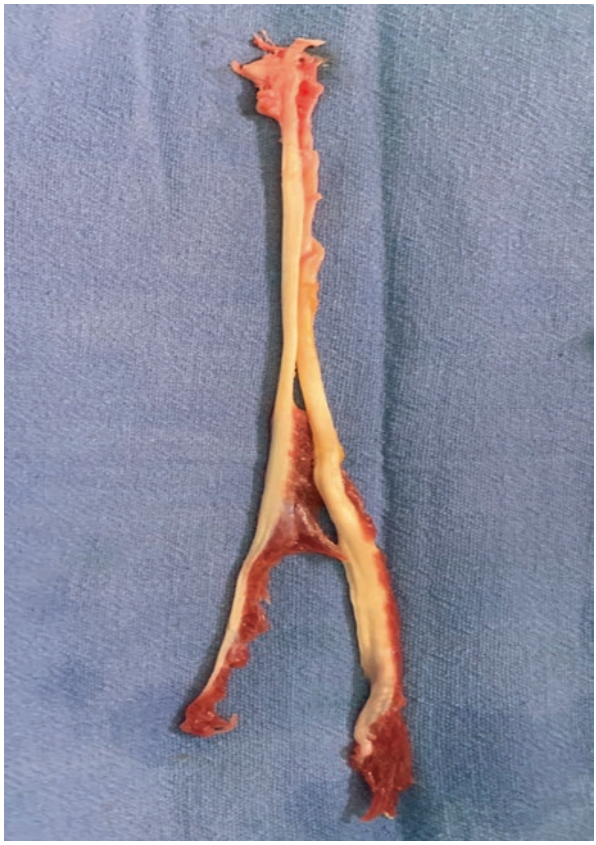

Fig. 17.9 Clinical photograph of Harvested Hamstring tendons for ACL Reconstruction

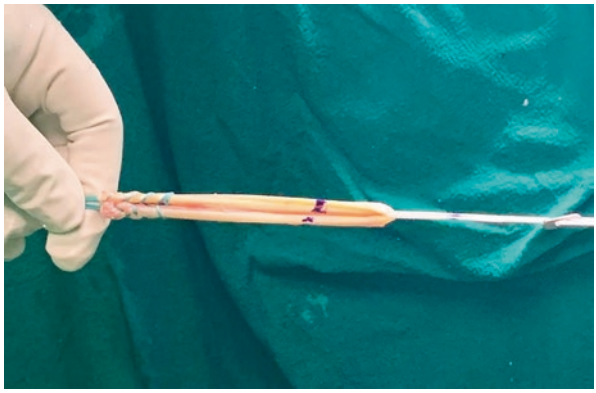

Fig. 17.10 Clinical phoograph of Quadrupled Hamstring tendon graft for ACL reconstruction

failure of graft and if made too posteriorly placed, it causes the graft to be taut in extension and lax in the flexion leading to poor stabilization of the knee. Hence, an isometric placement of the graft provides the best outcomes. Most surgeons advocate placement of graft posterior to native ACL tibial insertion (near PL bundle) as the maximum stress on ACL is on the extension.

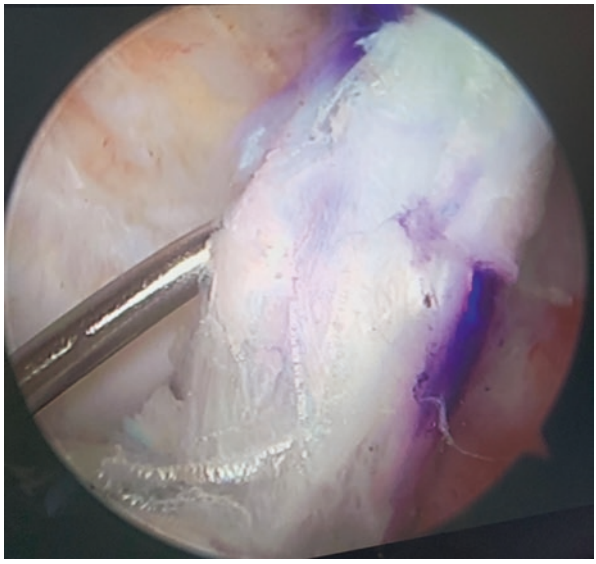

Fig. 17.11 Arthroscopic view of reconstructed ACL using Hamstring tendons

- Graft tensioning

  Graft tensioning is important to avoid graft failure and for better joint kinematics. The tension is graft specific. However, this tension should be just sufficient to negate the Lachman test. If the graft is not cyclically preconditioned, it has shown to have a reduction in force by approximately 30% after fixation.
- Graft fixation

  A sound fixation of the ACL graft is essential during its reconstruction surgery. It can be achieved either by:
  (a) Direct fixation using: Interference screws, staples, washers, cross pins.
  (b) Indirect fixation using: Polyester tapes, suture post, titanium button.
- Graft failure

  A reconstructed ACL graft can fail through several mechanisms and reasons:
  (a) Fixation device failure (e.g., screw divergence, graft tunnel length mismatch, and slippage)
  (b) Auto-/allograft failure: Creep, reduced strength, immune response
  (c) Impingement
  (d) Nonanatomical location of tunnels
  (e) Too aggressive rehabilitation causing stretch-out
  (f) Infection

Rehabilitation

Functional outcomes after ACL surgery depend primarily on the postoperative rehabilitation. The goal in the first 2 weeks after surgery is to attain full extension, attain

flexion up to 90°, and attain good quadriceps strength. Next 2 weeks are reserved to attain full flexion. Further 2 weeks are for attaining isotonic and isometric strengthening of quadriceps. Return to sports is usually aimed at 6 months.

Results

The results of ACL reconstruction surgery depend on numerous factors such as obesity, concomitant injuries, and timing of surgery. Obesity leads to increased loading over the medial compartment leading to excessive shearing along with rotations. The articular cartilage wears out quicker as well as the reconstructed graft may stretch out faster. The ACL injuries are often associated with medial meniscal injuries. Although most patients get good to excellent results in the short term after ACL reconstruction, its consequences in the long term in prevention or acceleration of knee osteoarthritis (OA) are not yet well-defined. Most surgeons therefore prefer not to wait long after an ACL injury to do an ACLR, as delayed reconstruction is associated with secondary damages to the intra- and periarticular structures of the knee [10].

Chronic ACL insufficiency may sometimes be associated varus malalignment of the knee due to coexisting osteoarthritis (OA). A combined ACL reconstruction with tibial osteotomy (HTO) needs to be done in these cases to achieve success [11].

An ACL reconstruction should be performed either in an acute setting or after the edema subsides and the patient gains a full passive range of motion for the best results. Chronic tears, after ACLR, have shown poor results compared to acute reconstructions as the later as seldom associated with secondary injuries and degeneration.

Complications

Intraoperative:
- Inadequate length of the graft.
- Blowout of posterior femoral condyle.
- Graft amputation.
- Incorrect tunnel placement.

Postoperative:
- Extension lag.
- Anterior knee pain (mainly with BTB graft).
- Arthrofibrosis (joint stiffness).
- Recurrent or residual instability of the knee.

Revision ACL Surgery

Revision ACL reconstruction may be required if the primary ACL reconstruction fails early or late due to various reasons. Early failure has been reported in

about 10% cases, within 6 months of the surgery, and the late failures occur after 1 year, mainly due to recurrent or repeat injury.

Recent Advances in ACL Reconstruction

Biologics are the way ahead in medicine. There are various new modifications which have been tried along with the regular ACL reconstruction to enhance healing. This augmented ACL reconstruction has not shown significantly better results. Various newer substances used are bio-scaffolds such as hyaluronan, platelet-rich plasma, and mesenchymal stem cells. All these theoretically have proven to deliver growth factors such as platelet-derived growth factor (PDGF), transforming growth factor (TGF), and vascular endothelial growth factor (VEGF) to the injury site.

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Total Knee Replacement

18

Muhammad Zahid Saeed

Introduction

There is no cure for osteoarthritis of the knee joint [1]. The gold standard surgical intervention at present is cemented total knee replacement with a survival rate of 95% at 14 years [2]. The best survival of knee replacement is observed in the old patient who is 70 years of age or older, the surgery is performed for rheumatoid arthritis, the activity level is low, knee joint is well balanced, mechanical axis is maintained and all components are fixed using the cement. The decreased survivorship of knee joint is observed in young patients, aged 55 years or less, TKR is performed for osteoarthritis and the patient's activity level is high [3–26].

According to the British National Joint Registry (NJR) the majority (97%) of procedures have been performed for osteoarthritis (Fig. 18.1), with more women undergoing the procedure than men, and a mean age at implantation of total knee joint was 68.9 years. The knee replacement is likely to last the rest of their life without the need for revision in the cohort of patients around the average age of undergoing surgery (68.9 years) [2].

Indications of Total Knee Replacement [1, 3]

- Incapacitating pain affecting, walking, activities of daily living, sleep and recreational activities
- Exhaustion of conservative measures to control the pain

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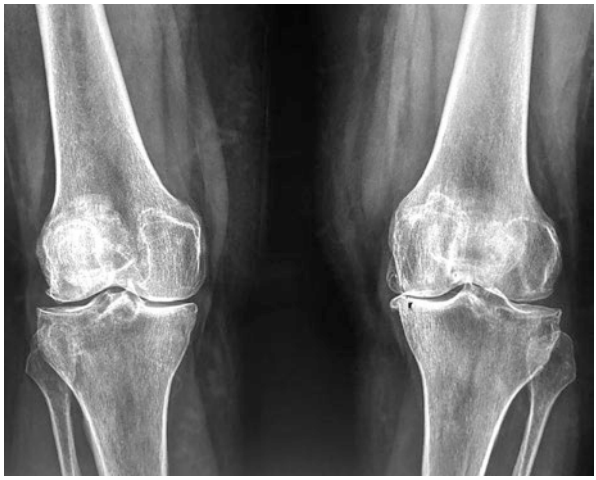

Fig. 18.1 Advanced osteoarthritis of both knee joints (reduced joint space, osteophyte formation, cyst formation and sclerosis of the bones)

- Medically fit for surgery and anaesthetics
- Absence of active infection—anywhere

Principles of Total Knee Replacement [3]

- Restoration of mechanical axis of the affected limb
- Restoration of the joint line
- Balance ligaments with equal flexion and extension gaps
- Maintenance of normal Q angle

Preoperative Planning-Risk Calculator

The British National Joint Registry (NJR) has introduced a "risk calculator" that is a useful tool to predict the likelihood for revision, mortality or patient-reported outcome measures (PROMs) by simply recording the individual patient data such as age, gender, BMI, etc. It is predicted that "risk calculator" will be utilised in the future in the outpatient setting by the surgeon and patient to make "shared decision-making" [2].

Preoperative Planning for Total Knee Replacement

Preoperative X-rays must include [2]:

- Bilateral weight-bearing (standing) AP (anteroposterior) view of knees (Fig. 18.1)
- Extension and flexion lateral views

- Skyline/sunrise (merchant view)
- Standing full-length AP hip to ankle in the following scenarios
- Angular bony deformity in both coronal and sagittal planes
- Very short individuals (152 cm or less)
- Very tall individuals (190 cm or more)

Preoperative Planning-Special Circumstances [1–20]

- Bone defects.
- Joint subluxation. Check Beighton score. Plan the amount of constraint required.
- Medial and lateral collateral ligaments laxity. Plan the amount of constraint required.
- Weakness (reduced muscle power in the legs).
- Compromised vascular status (Absent distal pulses).
- Impaired common peroneal nerve function due to any cause.
- Demented patients.

Anatomical and Mechanical Axis of Femur and Tibia

Under normal conditions, a vertical line drawn downwards from an individual's symphysis pubis in the standing position is called the *vertical axis*. A line drawn from the centre of the femoral head to the centre of the ankle joint, which passes through the knee just medial to the tibial spine, is known as the *mechanical axis* of the lower limb [1]. The mechanical axis is not parallel to the vertical axis and usually makes an angle of 3° with the vertical axis. The mechanical axis can vary in different individuals depending on the height and the width of the pelvis [20].

The *anatomical axis* is a line drawn parallel to the length of the intramedullary canal of either the femur or the tibia. When looking from the front, in standing posture, the anatomical axis of the femur (AAF) makes an angle of 5–7° with the mechanical axis of femur (MAF). Usually the anatomic axis of the tibia (AAT) and mechanical axis of tibia (MAT) are coincident. In the situation where a tibial angular deformity is present, the AAT and MAT are different. The tibial and femoral cut angles are cautiously measured to make a tibia end cut perpendicular to the mechanical axis of tibia (MAT) and femoral end cut perpendicular to the mechanical axis of femur (MAF) [20–24].

The femoral anteroposterior cut is made parallel to the epicondylar axis, perpendicular to the anteroposterior axis—defined as the centre of trochlear grove and intercondylar notch (white side line) and in 3° of external rotation to the posterior condylar axis [4, 5, 12, 16, 20–24] (Fig. 18.2).

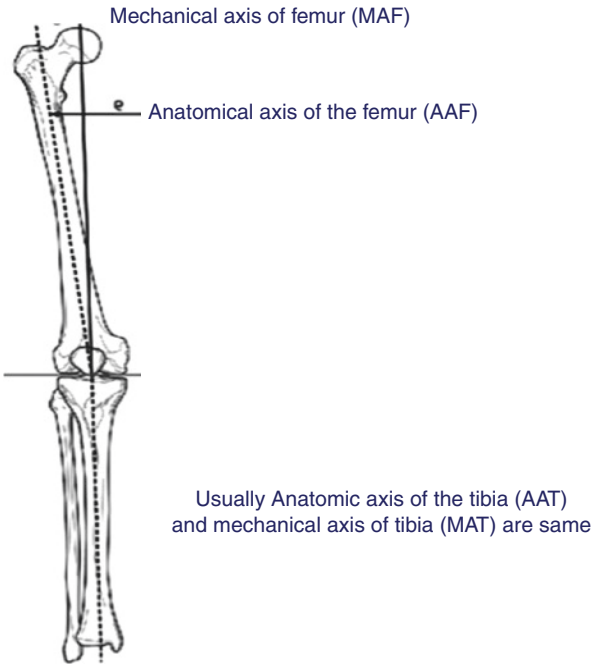

Fig. 18.2 Line diagram showing the anatomical and the mechanical axis of femur and tibia

Total Knee Replacement Prosthesis Design

There are three types of total knee joint replacement prosthesis design [2–24].

1. Cemented Total Knee Replacement
   (a) Unconstrained
   (b) Constrained-nonhinged
   (c) Constrained-hinged
2. Uncemented Total Knee Replacement
3. Hybrid Total Knee Replacement

1. Unconstrained Design
   (a) Posterior Cruciate Retaining [7, 17, 19]
   - PCL is taught during the knee flexion and acts as a safety valve for the anterior dislocation of femur on tibia.
   - Flexion instability is reduced.
   - Femoral rollback (posterior femoral-tibial contact point in sagittal plane) is created. It helps the posterior femur to flex further on the posterior tibia without impingement.
   - Increased contact stresses on the PE leads to rapid PE wear.

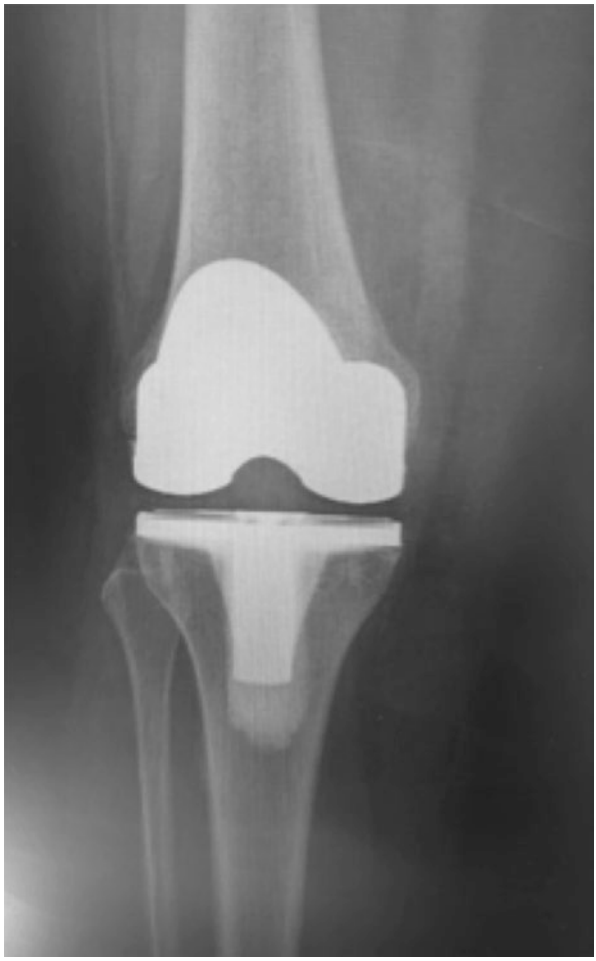

Fig. 18.3 Posterior cruciate substituting total knee replacement: note the "cam" in the femoral component. The tibial component has a "post" which is not visible on the X-rays

(b) Posterior Cruciate Substituting (Posterior Stabilised) [8]

The hallmark of posterior cruciate substituting design is as follows:

- Cam and post (Fig. 18.3)
- Rotating or fixed tibial platform

2. Constrained-No Hinged Design [2–24]
   - The constrained-no hinged implant has a large central post that substitutes for medial collateral ligament and lateral collateral ligament function.
3. Constrained-Hinged Design [2–24]
   - The femur and tibia are mechanically linked with at connecting bar and bearing.

Current Evidence [2]

- According to the British National Joint Registry (NJR) of all knee replacement prostheses used, 63% were cemented unconstrained prostheses and 23% were cemented posterior stabilised, with mobile-bearing implants not frequently used.
- The cemented TKR is the most widely used knee replacement in the UK, representing 85% of all primary prostheses logged in the registry.
- The numbers of uncemented or hybrid total knee implants used has decreased, to 2.2% of all primary knee replacements.

Principles of Bone Cuts in Total Knee Replacement [2–24]

- Controlled resection—replace with what has been lost.
- Replace bone and cartilage with implants which are of the same thickness and size.
- Maintains joint line that is imperative for accurate ligament function.
- Maintains tension in the medial and lateral collateral ligaments.

Ligament Balancing in Total Knee Replacement [2–24]

1. Coronal Plane
   - Correction of varus deformity
   - Correction of valgus deformity
   - Correction of extraarticular coronal plane deformity
2. Sagittal Plane
   - Correction of flexion contractures

1. Correction of Coronal Plane Deformity
   (a) Correction of Varus Deformity
      - Convex side is lateral which is loose.
      - Concave side is medial so medial compartment release is required.
      - The sequences of medial compartment release are as follows:
        – Osteophytes.
        – Deep medial collateral ligament (MCL) including medial knee capsule.
        – Posterior medial corner including capsule and semimembranosus.
        – Superficial MCL is the key structure for stability.
          Release posterior oblique portion of superficial MCL for medial extension tightness
          Release anterior oblique portion of superficial MCL for medial flexion tightness

(b) Correction of Valgus Deformity
- Convex side is medial side, which is loose.
- Concave side is lateral side so lateral compartment release is required.
- The sequences of lateral compartment release are as follows:
  - Osteophytes
  - Lateral capsule
  - Iliotibial band release is required for lateral extension tightness
  - Popliteus releases is needed for lateral extension tightness
  - Lateral collateral ligament (last structure to release)

(c) Correction of Extraarticular Coronal Plane Deformity
- McPherson's rule guides us that corrective osteotomy procedure and total knee replacement should be performed at the same time when coronal deformity is within the distal one fourth of the femur or proximal one fourth of the tibia and deformity is 20° or more [11, 14].
- The closing wedge osteotomy of the femur or tibia is preferred over the open wedge osteotomy. The additional fixation at osteotomy site can be avoided by using the diaphyseal press-fit stem with splines.
- The deformities within the distal one fourth of the femur or proximal one fourth of the tibia are the most challenging to correct.
- When the extraarticular coronal bone deformity is closer to the knee joint, the mechanical malalignment at the joint line is greater and vice versa.
- When the deformity is farther away from the knee, the smaller intra-articular bone cut is needed to correct the mechanical alignment and vice versa.

(d) Correction of Coronal Plane Deformity Without Collateral Release

Mullaji and Shetty [43] recommended that the release of collateral ligaments during TKR can be avoided in most cases of severe deformity by the following steps:
- Determining whether the deformity is intra- or extra-articular
- Individualizing the bone resection and valgus cut angle
- Balancing the extension gap first with thoughtful releases
- Using additional procedures in the presence of extra-articular deformity

2. Correction of Sagittal Plane Deformity—Flexion Deformity (Flexion Contracture) [3, 17–24]
- The Concave side is posterior knee so the structures at the posterior aspect of the knee joint require a release.
- It is performed with the knee flexed (usually at 90° of flexion) to reduce the risk of injuring the popliteal artery.
- The sequences of posterior knee release are as follows:
  - Osteophytes
  - Posterior capsule
  - Gastrocnemius muscle origin

Sagittal Plane Balancing in Total Knee Replacement [3, 17–24]

- McPherson's rule
  - Adjust the tibia first for symmetric gap problems.
  - Adjust femur first for asymmetric gap problem.

(A) Symmetric Sagittal Plane Gaps (Table 18.1)
(B) Asymmetric Sagittal Plane Gaps (Table 18.2)

Table 18.1 Symmetric sagittal plane gaps—total knee replacement

Scenarios	Reason	How to address the problem
Tight in both extension and flexion	Symmetric gap Not enough tibial bone was cut	Take off more proximal tibia
Loose in both extension and flexion	Symmetric gap Extra-tibial bone was cut	Consider using thicker polyethylene insert Consider using metallic tibial augmentation

Table 18.2 Asymmetric sagittal plane gaps—total knee replacement

Scenarios	Reason	Tackle the problem
Tight in extension only Flexion is decent	Asymmetric gap Not enough posterior capsule was release Did not cut enough distal femur	Release posterior capsule Cut more distal femur bone, 1–2 mm at a time
Tight in flexion only Extension is decent	Asymmetric gap Not enough posterior femur was taken off Tight PCL scarred No posterior tibial slope was cut	Cut more posterior femur bone, 1–2 mm at a time Recess PCL Recut posterior slope of tibia, if no posterior tibial slope was cut (only anterior slop was cut)
Loose in flexion and extension is decent	Asymmetric gap Extra posterior femur was cut	Increase size of femoral component Use cement or metal augmentation to fill posterior gap Consider using thicker polyethylene insert and manage as tight extension gap
Loose in extension and flexion is decent	Asymmetric gap Extra distal femur was cut Anteroposterior size too large	Use distal femoral augmentation Use thicker tibial polyethylene inset, and manage as tight flexion gap Reduction in size of femoral component from anterior to posterior

Computer Navigation Versus Conventional Total Knee Replacement

There have been no research studies to show an increase in functional outcome or rates of wear with the use of computer navigation. However, the research studies have revealed that the functional outcome between the computer-navigated and conventional total knee replacement appears to be no different despite the better alignment achieved using the computer navigation technique [25–29].

Robotic Total Knee Replacement

We are entering in a new era of robotic surgery. There is a greater scope for wider and more meaningful robotic applications. It is credible that this new innovation will one day become the benchmark of care for a variety of surgical procedures. We do not have sufficient long-term follow-up study results to make a solid conclusion about the robotic use in surgery. The high level prospective randomised studies will help us to make sure that robotic surgery is safe for our patients [30–42].

The perceived advantages of robotic surgery are execution of the surgical plan with accuracy and precision, accurate placement of the implants, less pain, early recovery, excellent outcomes, early return to work, and patient safety. The disadvantages include increased risk of complications during the surgeon's learning curve, high blood loss, malalignment of the prosthesis, costs, long operation times and unpredictable results [30–42].

Costs are an important aspect for running any healthcare system in the world. Initial equipment costs for robotic surgery are more than £1 million and ongoing operational costs are noteworthy [30–42].

The learning curve associated with robotic surgery can have a considerable impact on training of junior doctors [30–42].

The robots can be used for total knee replacement, but its efficacy and advantages are still not clear. Results in knee arthroplasty are more unpredictable. The evidence base for robotic assistance in total knee replacement is still in infancy and no strong conclusion can be made on its implications for clinical practice at present [30–42].

A national registry may define, improve and maintain the quality of care of individuals receiving robot-assisted total knee replacement across the globe for common and highly successful operations that bring many patient relief from pain, improved mobility and quality of life [30–42].

Total Knee Replacement: Complications

- Pain.
- Periprosthetic joint infection (PJI) [13].
- Altered wound healing.
- Wound breakdown.
- Unsightly scar/keloid formation.
- Surgical scar tenderness.
- Bleeding.
- Stiffness.
- Arthrofibrosis and scar tissue formation.
- Postoperative flexion contracture due to hamstring tightness and spasm.
- Complex regional pain syndrome.
- Injury to medial and lateral collateral ligaments.
- Injury to extensor mechanism.
- Injury to quadriceps and patellar tendons.
- Injury to neurovascular structures, vascular injury, common peroneal nerve injury [15].
- Lateral superior genicular artery is at risk with lateral retinacular release and transection of this artery increases risk for osteonecrosis of the patella.
- Joint dislocation [10].
- Thromboembolism (deep vein thrombosis, pulmonary embolism).
- Leg length discrepancy.
- Osteolysis, prosthesis wear/aseptic loosening leading to revision of the prosthesis.
- Periprosthetic fracture of tibia and femur.
- Patella fracture [18].
- Heterotopic ossification.
- Symptoms may not settle and pain in the knee joint may persist.
- Malalignment and malrotation of the prosthesis.
- Incorrect sizing of the implants.
- Femoral notch [9].
- Heterotopic ossification.
- Complex regional pain syndrome.
- Anaesthetic risks.
- Morbidity and mortality.
- Medical problems including chest infection, urinary tract infection, urinary retention, stroke, myocardial infarction.

Total Knee Replacement: Survival

The British National Joint Registry (NJR)'s 15th Annual Report which was published in 2018 has revealed that the Kaplan-Meier revision rate at 14 years for all cemented total knee replacements is approximately 4.47%. The revision rate for unconstrained fixed bearing implants is 4.07%. The revision rate was 5.39% for

posterior stabilised implants. The best survival rates were observed for cemented TKR with monoblock tibial components [2]

The knee replacement has a revision rates of nearly 15% at 14 years in female patients less than 55 years at the time of surgery, compared to just 2.5% in female patients over 75 years [2].

Over 80% of revision operations recorded in the registry were single stage procedures. The most common indications for surgery were aseptic loosening, other indication, instability and pain. The NJR data revealed that the risk of re-revision following a first revision was greater than the risk of revision after a primary total knee replacement [2].

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Surface Arthroplasty of the Knee

19

Sankalp C. Mutha

Epidemiology

Knee OA is a common form of arthritis characterized by loss of articular cartilage within synovial joint, subchondral bone sclerosis and thickening of capsule.

It affects 9.6% men and 18% women over age of 60 years [1].

Among persons with a potential need for TKA, only 12.7% of women and 8.8% of men were "definitely willing" to have a procedure [2].

Introduction

"Arthroplasty of the knee has as yet, no standard technique. It is still an open chapter in joint surgery and one which offers much promise" (Albee 1928). F.H. Albee made this remark in 1928 [3].

His observations hold true even today while attempting effectively to replace one of the most complex joints in the body.

Knee resurfacing has a long history paralleling the advances of total knee replacement and frustrations associated with developing perfect implant can just bring back thoughts reflecting Albee's statement again.

Knee resurfacing had been despised for long time due to various reasons ranging from faulty implant designs to not so good reproducible results even in experienced hands.

Although myth around failure of some of implants has been disappearing in recent times but the best anatomical design and perfect biomechanical interplay to get the best functional outcome are still a goal remained not completely fulfilled

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History

In 1743 William Hunter stated, "an ulcerated cartilage is a troublesome problem and once destroyed, it never repairs". In spite of new advances in the field of tissue engineering, this statement holds true even three centuries later [4, 5].

Alterations in cartilages in osteoarthritis and the morphologic changes in joints affected by osteoarthritis were identified in writings of the Hunter brothers over 200 years ago.

In 1942, Bennett, Waine and Bauer presented their classic monograph, which described the progression of changes seen in the development of osteoarthritis of the knee, and 7 years later, Collins established a classification system for the joint changes on which much of the current work is based [6].

Pathoanatomical Overview

Articular cartilage is a specialised connective tissue present in the knee.

The early degenerative changes in articular cartilage go through various phases like cartilage softening, fissuring and diminished thickness which progress over time when cartilage eventually thins out leading to total destruction.

However, changes in the knee joint start from subchondral bone expansion, bone marrow changes and meniscal tear which along with cartilage loss leads to osteoarthritis of the knee.

Scientific evidence indicates that the menisci, ligaments, periarticular muscles and joint capsule are also involved in osteoarthritis of the knee.

Arthroplasty

Arthroplasty is defined in the broadest sense as a reconstructive procedure that alters the structure or function of a joint.

Perhaps the earliest indication of knee arthroplasty comes from those who studied Egyptian mummies in which it appeared that a primitive type of arthroplasty involving the distal femur and proximal tibia had been performed.

Hemiarthroplasty is an alternative to knee replacement for young, active patients specially with early osteoarthritis involving one compartment, as it allows preservation of more bone stock and anatomical structures.

Historical Milestones

Resection Arthroplasty

In 1861, Ferguson reported on his experience with the resection of the entire knee in patients with ankylosis or significant deformities usually related to tuberculosis

or other infections. When sufficient amounts of bone were resected, a satisfactory range of motion (ROM) was achieved, but the knees were unstable. With less bony resection, the joints often became ankylosed. These results generally were considered poor and the procedure essentially was abandoned [7].

Soft Tissue Interposition Arthroplasty

Verneuil is credited as being the first to describe an excision arthroplasty supplemented by the interposition of soft tissues to prevent bony ankylosis. In 1860, he reported on his results using a flap of fascia lata with subcutaneous fat. Only limited success was achieved [7].

Despite numerous variations in technique and in the material used for interposition over the next 100 years, results were unsatisfactory and soft tissue interpositional arthroplasty lost its relevance slowly over decades.

The last seven decades heralded the beginning of an era where numerous implants ranging from metal on bone to metal on ceramic found space in surgical armamentarium for treatment of OA knee.

Literature Review of Past Implants

Many papers report data on four very similar designs developed in the 1950s, 1960s and 1970s.

They all required some minimal bone cuts on the tibia, for fit and fixation.

They all needed a 15–20 cm medial parapatellar incision for joint preparation and implant insertion [7].

Early Designs of Knee Resurfacing

McKeever Device

In the early 1950s, Duncan C. McKeever put forth idea that osteoarthritis could be isolated to only one compartment of the knee joint and replacement of the complete knee might not be necessary if only one knee compartment had osteoarthritic changes.

Elliot presented Dr. McKeever's results of 40 patients in 1985. The patients had all been operated on during the 1950s by Dr. McKeever using the McKeever device.

In this report, 39 out of 40 patients were reported to have had a good result [8].

MacIntosh Device

MacIntosh device in a way followed principles which McKeever proposed and took the same concept forward albeit with limited success.

In this series of 130 knees operated using the MacIntosh device, there were 89 knees with rheumatoid arthritis and 41 knees with osteoarthritis.In the osteoarthritis patients, a good result was obtained in 80.5% of the cases.In the patients with rheumatoid arthritis, 69% of patients had a good response [9, 10].

Sbarbaro Device

Sbarbaro implant followed up on the work and concept of Duncan and MacIntosh and was used in 1960s onward till early 1970s.

It was one of biggest series published.

Two hundred forty-nine OA knees in 219 patients were operated. Of these, 224 were medial OAs and 25 were lateral OAs.

Fifty-six percent of patients rated the results to be excellent, while 29% showed some improvement and 15% were failures [11].

Swanson Device

Swanson et al. reported the use of the Swanson device in 1985 on 32 patients. These patients were followed for 2–14 years. Of these, ten underwent unicompartmental surgery and 22 underwent bicompartmental surgery.

All unicompartmental procedures showed good results [12].

UniSpacer Device

Hallock and Fell reported the initial UniSpacer experience in 2003 with 71 knees in 67 patients.

They reported that there was a 21% (15 of 71) revision rate at 1 year, mainly due to dislocation or pain [13].

Hinged Knee Prosthesis

In the 1950s, Walldius developed a hinged prosthesis that replaced the joint surfaces of the femur and tibia [14].

In 1971, Gunston developed polycentric knee arthroplasty. This was done by using the concepts of low friction hip arthroplasty developed by Charnley. Gunston's knee arthroplasty retained the collateral and cruciate ligaments to help absorb stress and consisted of relatively flat tibial interposition of high-density polyethylene and a round femoral prosthesis, which replaced the posterior portion of femoral condyles [15].

In the early 1970s, three types of condylar prostheses were developed, which formed the bedrock of modern knee arthroplasty.

In early 1976 Ranawat et al. with duocondylar prosthesis followed by Coventry with geometric prosthesis and Townley with anatomic prosthesis led the way for new surgical implants for knee arthroplasty [16].

However, the geometric and anatomic types were discontinued soon due to early loosening of fixation.

The Oxford unicompartmental knee replacement (Biomet Orthopedics, Inc, Warsaw, Ind) was designed by John Goodfellow and John O'Connor. It was first used in a patient as a unicompartmental device in 1982. It brought back focus on unicompartmental arthroplasty and knee resurfacing arthroplasty [17].

Since then more designs have been created and tested to address unicompartmental osteoarthritis with the aim of preserving anatomy. This involved reducing the size of surgical trauma with minimal bone cut and more muscle sparing to improve rehabilitation post-surgery.

What Does Knee Resurfacing Involve?

Knee resurfacing involves placing a functional glide path made of a low friction material onto the tibial plateau.

It is designed to replace the function of worn articular cartilage without damage to ligaments and soft tissue in and around the knee.

It involves procedures in two stages:

1. First stage is arthroscopic removal of meniscus and soft tissue from one compartment.
2. Second stage involves preparation of bed for implant with removal of cartilage followed by insertion of implant with small arthrotomy.

Who Are Ideal Candidates for Knee Resurfacing Surgery?

Patients with:

1. Early osteoarthritis of medial compartment
2. Age group of 40–60 years where patients are unwilling to undergo total knee replacement
3. Mobile patients with no deformities of joint
4. Patients ready to accept less than complete pain relief in return for less destructive surgery of knee joint
5. Those who are ready for bridge surgery to postpone total knee replacement
6. Medically unfit patients for total knee replacement who need relative pain relief in severe osteoarthritis of knee

Metal on Bone Quagmire?

Dr. Scott's article documented that a metallic CoCr spacer could give long-term pain relief with the McKeever [18]. The highlights of the article were as follows:

1. Average follow-up of 16.8 years.
2. Longest patient follow-up was 29 years.
3. Some patients were very active.
4. Significant improvement in pain and function scores.

OrthoGlide knee resurfacing device is one of the better innovations for knee resurfacing in recent times.

OrthoGlide

OrthoGlide was introduced in the United States in 2006 and has been more recently been used in Canada, Germany and India. It is constructed from a well-accepted orthopedic implant material, i.e. cobalt chrome. It utilizes normal anatomic structures and forces to keep implant in place on the tibia. This implant seems to combine learning acquired from the prior five predicate implant experiences.

Top Surface View (Fig. 19.1)

Bottom Surface View (Fig. 19.2)

What Is OrthoGlide Knee Resurfacing?

The OrthoGlide system precisely aligns the surface of the implant to the contours of the patient's tibial articular surface, restoring a smooth and continuous joint surface for normal gliding mechanism (Fig. 19.1).

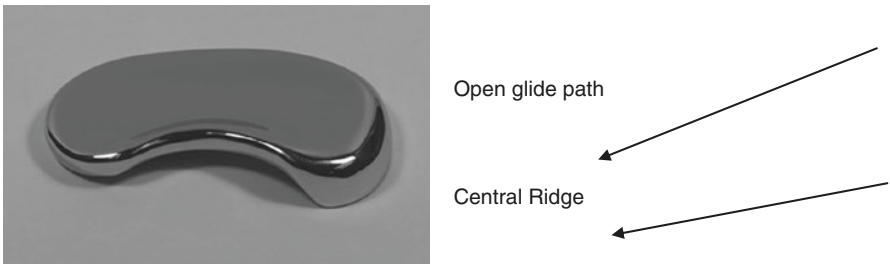


Fig. 19.1 Clinical photograph showing the OrthoGlide® Medial Knee Implant

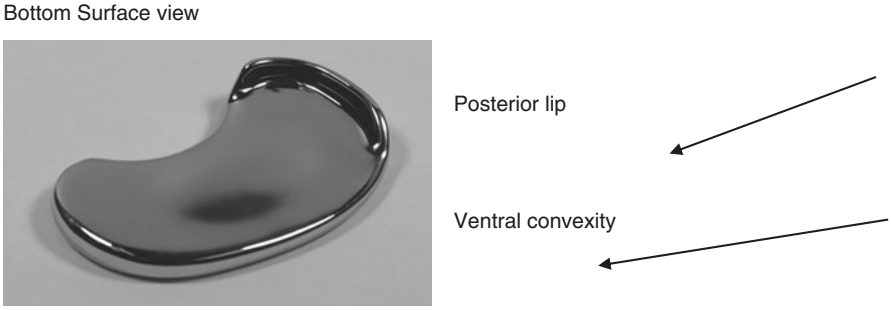
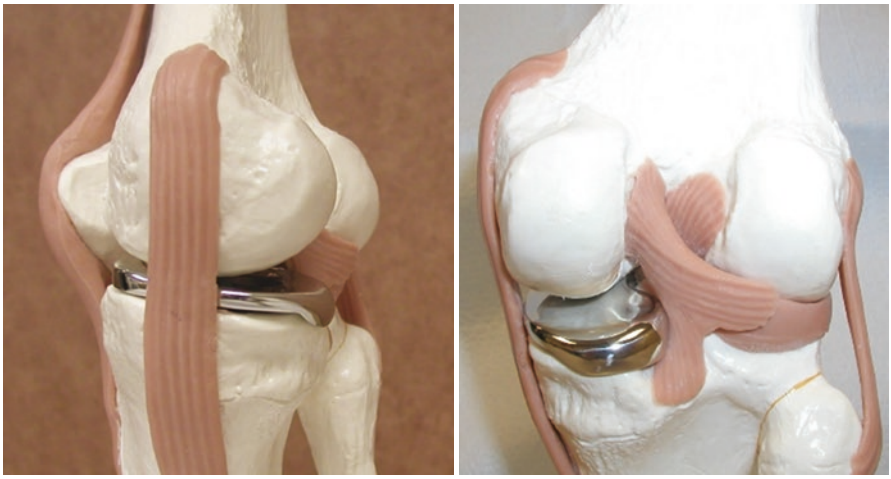


Fig. 19.2 Clinical photograph showing the OrthoGlide® Medial Knee Implant

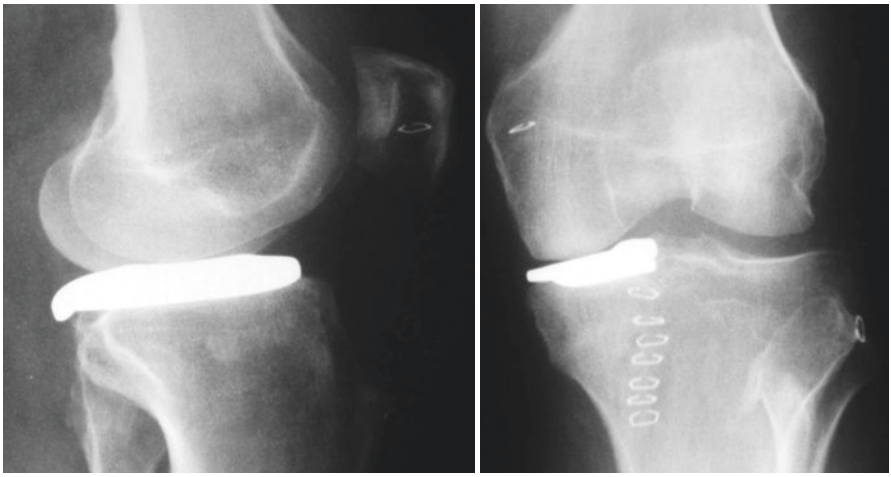


Figs. 19.3 and 19.4 Clinical photograph showing the bone model and its stability

The implants are designed to match the surface and contours being restored (Figs. 19.1 and 19.2).

Design Rationale

The posterior lip of the implant is designed to prevent significant AP movement. The medial ridge of the implant stops rotation. It has an open glide path for unconstrained motion of the femoral condyle. No bone cuts are required for the OrthoGlide resurfacing implant (Figs. 19.5 and 19.6) and it is inherently stable (Figs. 19.3 and 19.4).



Figs. 19.5 and 19.6 Post-operative rays showing no bone cuts or cement use

Radiographic Images Post-implant Insertion (Figs. 19.5 and 19.6)

Indications for OrthoGlide Knee Resurfacing

The device is primarily indicated for uncemented use in the treatment of moderate degeneration of the medial compartment of the knee (grades II–IV chondromalacia) with no more than minimal degeneration (grades I and II chondromalacia, no significant loss of joint space) in the lateral and patellofemoral compartments in patients with osteoarthritis.

Exclusion Criteria

- Lateral compartment disease
- Symptomatic patellofemoral disease
- Patients with chronic large symptomatic effusion and inflammatory arthritis
- Absent ACL
- Collapse or loss of contour of the tibial plateau
- Varus > 10° and FFD >10°

Flow Chart for Surgical Options for Media OA Knee (Fig. 19.7)

Medial osteoarthritis of the knee can be treated with knee resurfacing if ligaments are intact and no significant varus and fixed flexion deformity present.

Patients with more than 10° of varus and fixed flexion deformity or with patellofemoral arthritis are better suited for unicondylar knee arthroplasty (Fig. 19.7).

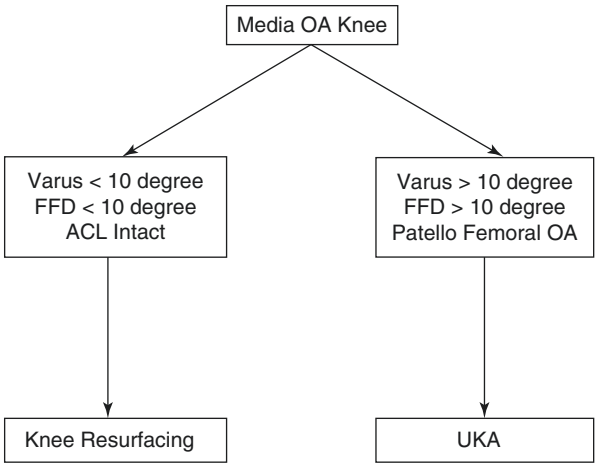


Fig. 19.7 Flow for treatment of medial osteoarthritis of knee. *OA* osteoarthritis, *FFD* full flexion deformity, *ACL* anterior cruciate ligament, *UKA* unicondylar knee arthroplasty

Pre-operative Planning involves

- Evaluation of pre-op X-rays and MRI for eligibility criteria including cartilage loss, joint space, ligament integrity, anatomic axis and osteophytes that could interfere with implantation and other bony or soft tissue abnormalities. Osteophytes on the posterior tibia, medial femur and tibia, anterior femur and tibial/femoral notch that can interfere with implant placement in the medial compartment are identified. This is followed by evaluation of the other compartments and the distal portion of the femur for abnormalities and disease.
- A diagnostic arthroscopy is performed of all three knee compartments for inspection of lateral and patellofemoral compartments to rule out advanced OA. Integrity of ACL, PCL, MCL and lateral complex ligaments of the knee is evaluated. Collapse or loss of contour of the tibial plateau is looked for.

All patients with full-thickness cartilage loss ("bone-on-bone") on both the tibial and femoral condyle in the lateral compartment (grade III/IV) are excluded. Patients with full-thickness cartilage loss ("bone-on-bone") on both the patella and trochlear groove (grade III/IV) are also excluded.

Surgery

The procedure involves preparing the knee joint for insertion of the implant.

Potential osteophytes that may need to be addressed are as follows:

- Notch (Tibial/Femoral)—Possible impingement with the central ridge of the implant
- Anterior Femoral—Possible impingement preventing full extension
- Medial Tibial/Femoral—Reduces tension on the MCL to allow joint space restoration

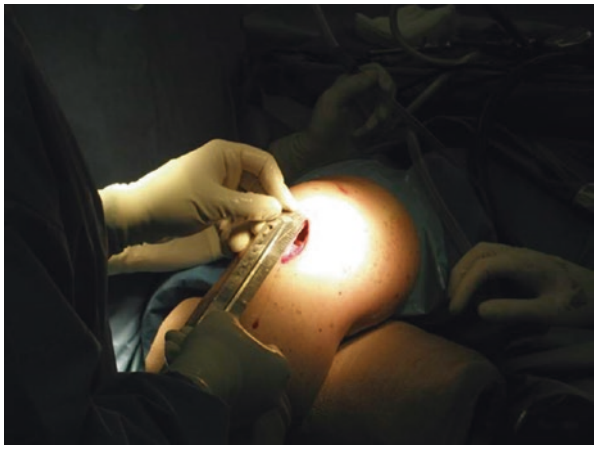


Fig. 19.8 Medial arthrotomy of knee

Medial Portal Arthrotomy and Fat Pad Removal

A mini-arthrotomy (Fig. 19.8) is done by extending the medial arthroscopic portal vertically 1.5–2 in. (5–7 cm). A portion of the infrapatellar fat pad is removed for improved visualization.

Anterior Meniscectomy

The meniscectomy of the anterior portion of the medial meniscus is completed. Care must be taken not to disturb the MCL.

Take Down Intercondylar Eminence

The cartilage on the medial aspect of the intercondylar eminence (ICE) on the tibial spine area is removed down to subchondral bone (typically with a high-speed shaver, not burr). This allows a rounded right angle at the medial spine. This is followed by flattening the cartilage anterior and posterior to the spine to the level of the tibial plateau.

Posterior Menisectomy

The posterior horn attachment of the medial meniscus is released. A partial meniscectomy of the posterior and medial portion of the medial meniscus to a 1–2 mm meniscal rim is performed.

Osteophyte Removal

All osteophytes from the anterior, posterior and medial rim of the tibial plateau and femoral condyle are removed followed by a smoothening with a manual or power rasp. Removal of these "MCL tenting" osteophytes allow a greater opening of the medial joint.

Posterior Cartilage Removal

The necessary level of cartilage on the surface of the tibial plateau is removed to match the point of maximal wear. If the maximal wear is to bone, then the entire plateau is smoothened to the calcified cartilage level. The goal is a flat surface of uniform thickness onto which the implant will be seated (Figs. 19.9 and 19.10).

Posterior Osteophyte Removal

Osteophytes are not uncommon in this region. In some patients, early osteophytes may not be visible on radiographs, but would still interfere with posterior OrthoGlide lip insertion and these need to be identified and removed.

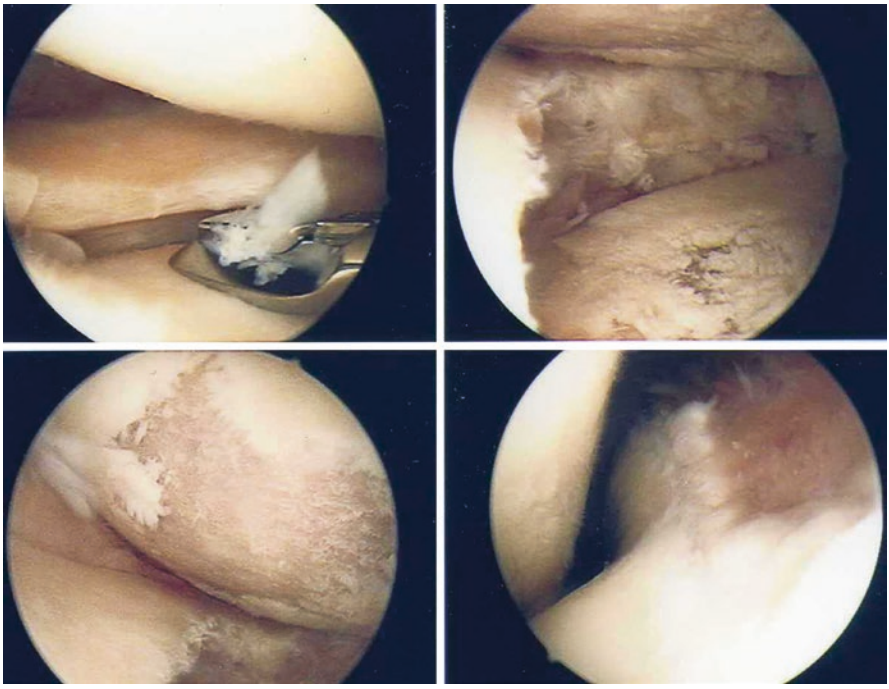


Fig. 19.9 Arthroscopic view of removal of the posterior one-third of the medial meniscus

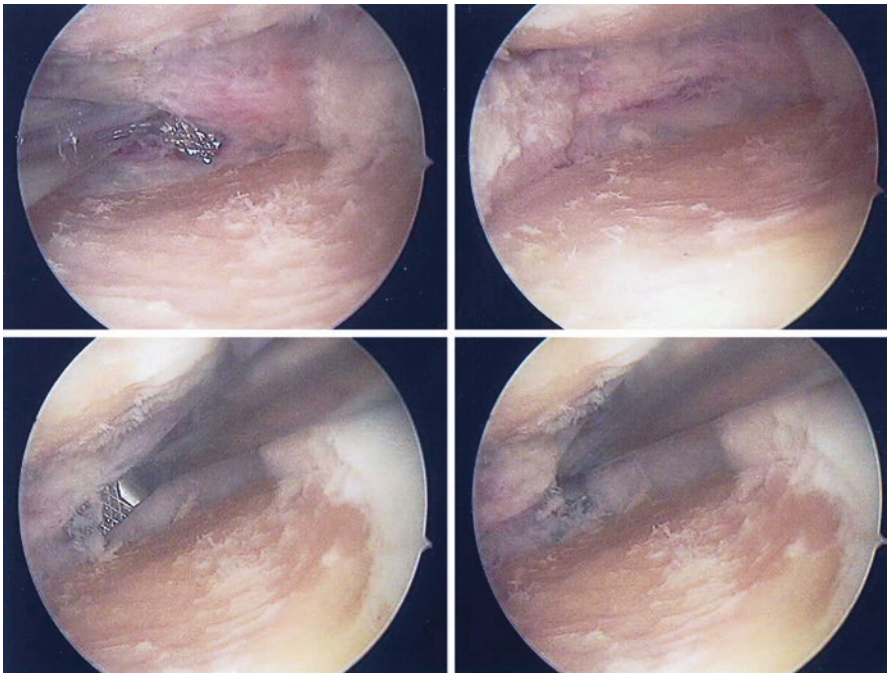


Fig. 19.10 Arthroscopic view of cartilage removal

Tibial Cartilage Smoothening

Tibial shaper in a compatible reciprocating instrument is used to remove the cartilage to a uniform level throughout, typically near the calcified cartilage level.

Congruency Rasp Insertion

Femoral congruency rasp is used to smoothen femoral surface and remove any osteophytes from femoral condyle.

Femoral Condyle Smoothening

With the femoral smoother or femoral congruency rasp in place on the tibia, the knee is fully flexed and extended while placing varus stress on the lower leg to achieve optimal result.

Flexion and Extension Gap Analysis

The gap gauges (3 mm, 4 mm) are used to measure the extension gap and select the trial device thickness most appropriate for the patient. The gap gauges should be used with the knee in the neutral position and the gauge should slip in without forcing it in both the extended and flexed positions.

Balanced extension and flexion gaps are important for natural ligament tension through range of motion. With the OrthoGlide, the goal is to have balanced flexion and extension gaps.

Tibial Plateau Sizing

The appropriate AP length 3-mm-thick trial device is placed on the tibial plateau with the posterior lip towards the medial tibial plateau. External rotation and valgus stress on the lower leg is used and then moved to ~30° of flexion while exerting posteriorly directed horizontal force on the trial device to accomplish insertion.

Trial Device Insertion

A trial device based on femoral congruency rasp size and the gap gauge thickness is chosen.

Tibial Spine Spacing

The tibial spine gauge is placed in the prepared area along the intercondylar eminence at full extension and at deep flexion to assess the space available for the medial ridge of the implant. This helps prevent any impingement of the implant on the lateral aspect of the medial femoral condyle. The knee is moved with the gauge in position through a full range of motion to look for impingement on the medial femoral condyle. If impingement is found, a more extensive preparation of the intercondylar eminence and/or the medial femoral condyle is done.

Proper Sizing

The trial device of proper size covers the entire prepared medial tibial joint surface so that the anterior edge of the trial device is at least even with or slightly longer than the tibial plate.

OrthoGlide Insertion Technique

The device is inserted with the knee at 25–40° flexion, valgus rotation and tibial external rotation. On insertion, the implant is rotated towards PCL tibial attachment (Fig. 19.11).

Surgical Pointers

Remove as much of the fibrillated cartilage and hypertrophied synovium as possible with shaver or a bipolar-type instrument.

Special attention is given to sizing—longer is better.

Congruency of implant with femoral condyle is checked.

Copious irrigation is done.

Since arthritic knees "flare up" and result in painful recurrent effusions longer sizes are used. The implant must span the entire femoral condyle anteriorly in full extension while allowing for potential for slight posterior migration and for the stress of weight-bearing. This is best determined via direct vision with the trial through ROM. The procedure requires patience and reminders that knee preservation is always subject to multiple additional treatment(s).

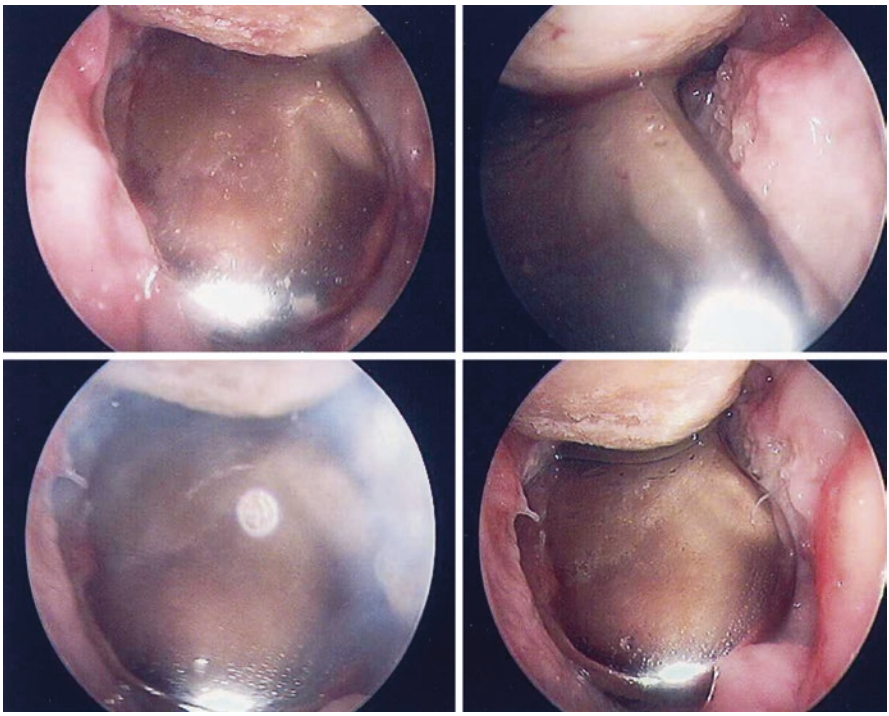


Fig. 19.11 Arthroscopic view of OrthoGlide implant

Post-op Management

Aggressive physical therapy is to be avoided.

Knee brace should be locked in extension or knee immobilizer should be used.

Ambulate the patient as tolerated. Continuous passive motion can be considered.

All effusions should be treated aggressively to prevent contractures with ice and limb elevation. Ace brace plus topical steroids or iontophoresis, steroid injections (Aristospan and Decadron) and HA injections can also be used. Judicious use of anti-inflammatory medicines has to be done.

If effusions persist, then arthroscopic debridement can be considered.

The patient is usually discharged home the same or next day with no formal physiotherapy. However, daily ROM exercises with weight-bearing as tolerated are advised. Gradual return to full function is advised.

Early short-term results of OrthoGlide knee resurfacing are comparable to TKA; however, it offers the following advantages over TKA:

1. Day surgery/outpatient procedure.
2. Less cost as compared to conventional TKA.
3. Allows squatting for patients.
4. Involves no bone cuts.
5. Uncemented procedure.
6. Conversion to UKA/TKA+PF/TKA is easy.

Post-operative Comparison with UKA/TKA

Most of the expected improvements in pain and function after UKA were achieved within 6 months of surgery.

In comparison to UKA, significant improvement was achieved within 3 months in OrthoGlide knee resurfacing.

Also, both UKA/TKA were associated with significant knee swelling and pain within 1–5 days of surgery.

No complaints of knee swelling were seen after OrthoGlide knee surgery. However, anterior knee pain was more frequent in post-operative period.

Literature Review of Quadriceps Strength Post-Surgery Comparison with UKA/TKA

Mizner et al. (2005) found that 1 month after TKA, quadriceps muscle strength decreased by 62% relative to the pre-operative level [19].

OrthoGlide spares quadriceps and soft tissue during surgery and hence does not affect muscle strength.

Mancher et al. (2002) found an increase in strength of the quadriceps muscle 18 months after UKA in both operated and non-operated legs [20].

UKA takes longer time to restore quadriceps function compared to OrthoGlide knee resurfacing.

Quadriceps muscle strength will vary post-operatively depending on the type of surgery done (Fig. 19.12).

Type of surgery	Post-op day 1	Post-op-1 month
Knee resurfacing	Unchanged	Unchanged
UKA	Reduced	Regained
TKA	Reduced	Reduced

Fig. 19.12 Quadriceps strength post-surgery. *Post-op* postoperative, *UKA* unicondylar knee arthroplasty, *TKR* total knee arthroplasty

Early Brief Experience and Results of the Author in OrthoGlide Knee Resurfacing

Material and Methods

This study examined early clinical results of patients treated with OrthoGlide Medial Knee Implant in India done by the author.Eleven knees of OrthoGlide knee resurfacing performed between 2012 and 2015 (six males and five females) were studied.The mean age of patient was 57.8 years (range 50–70); mean follow-up period was 12 months with range from 6 to 24 months.Pre-operative diagnosis of unicompartmental medial osteoarthritis was made in all the patients.

Results

The mean improvement in Oxford Knee Score was from 15 (range from 10 to 16) to 40 (range from 35 to 46) and mean WOMAC score improved from 30 (range from 20 to 36) to 85 (range from 80 to 92) at mean follow-up of 222 days (Figs. 19.13 and 19.14).

Good to excellent results are obtained in all of the knees with one case of persistent anterior knee pain managed by viscosupplementation and knee brace over 3 months.

Conclusion

Newer designed, minimally invasive knee resurfacing implant like appears to be safe and can provide effective pain relief along with rapid recovery and excellent functional result suitable for active lifestyle as it allows squatting and sitting cross-legged.

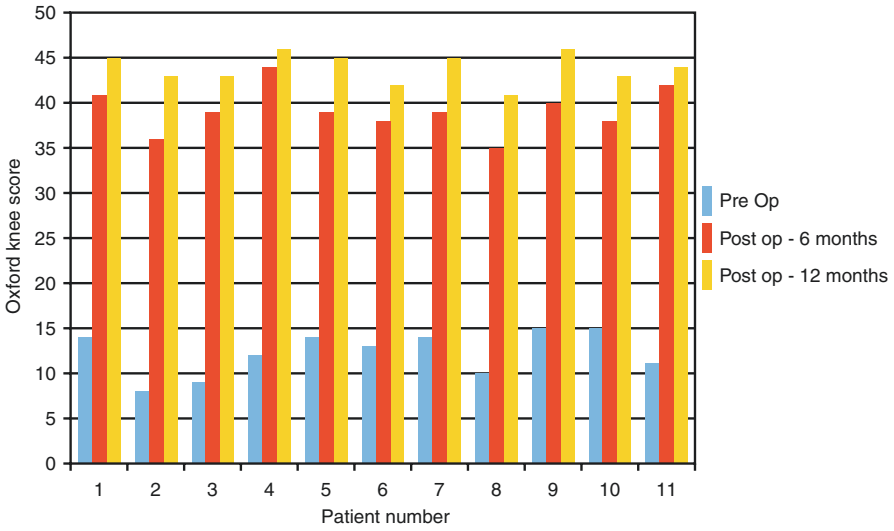


Fig. 19.13 Oxford Score

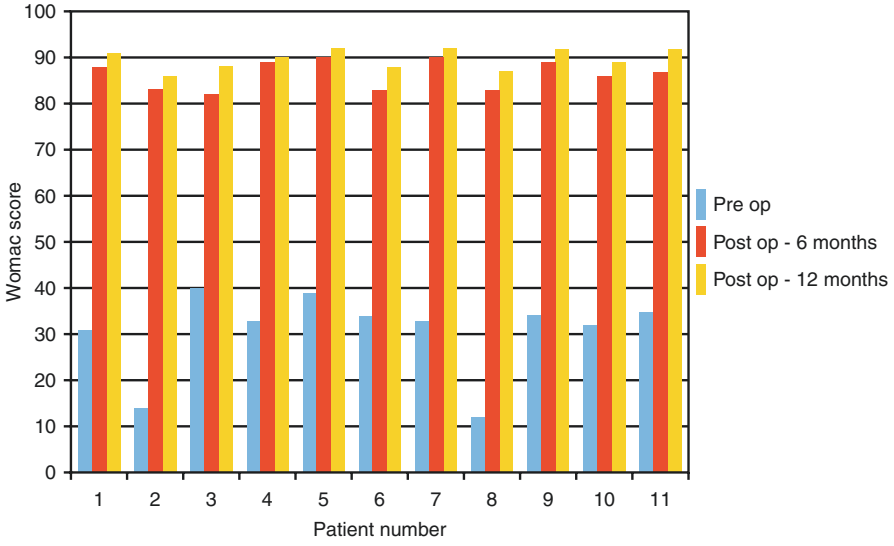


Fig. 19.14 WOMAC Score

This uncemented, arthroscopic, muscle-sparing day care surgery provides excellent option in our hands in surgical treatment of knee osteoarthritis patients [21].

First OrthoGlide knee Resurfacing Hemiarthroplasty in India by Author

Pre-operative X-ray: Medial Osteoarthritis of Knee

Post-operative X-ray Day One: OrthoGlide Implant In Situ

Post-operative X-ray of first OrthoGlide knee resurfacing done successfully by author in India (Figs. 19.15 and 19.16).

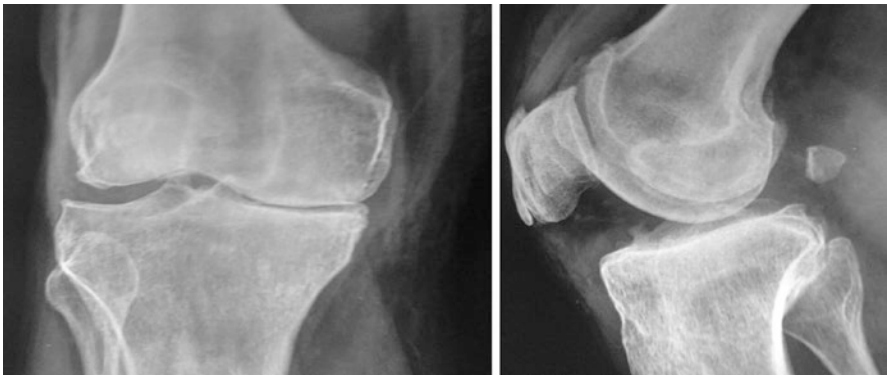


Fig. 19.15 X-rays taken pre-operatively showing medial OA of the knee joint

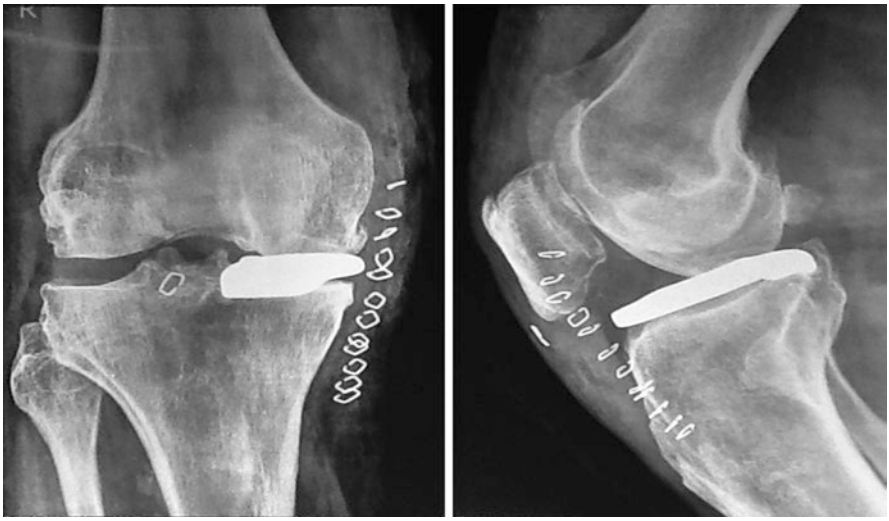


Fig. 19.16 X-rays taken postoperatively showing the implant in situ

Use of Robotics in Knee Resurfacing

Technically knee resurfacing is considered a demanding procedure as minimal invasive techniques pose challenges to surgeons' skills. The development of robotic-assisted technology makes it less demanding for surgeons.

Robotic-assisted systems improve a surgeon's ability to achieve the desired component alignment, optimize soft tissue balance, control the joint line and restore normal knee kinematics in UKA [22].

Various studies have reported improved radiological results with robotic knee resurfacing. But it is still unclear whether more favourable radiographic outcome measures correlate with greater functional improvement in the patients.

Robotic systems use templates prepared with CT scans. The robot provides both tactile and haptic feedback during surgery in order to assist the surgeon to follow the pre-operative plans [23].

Newer Implants in Knee Resurfacing

Newer implants using 3D computer imaging and patient-specific jigs which can provide more anatomical fit and better functional outcome are on horizon.

Patellofemoral resurfacing implants for isolated patellofemoral arthritis are also becoming available [24].

Clearly the last word has not been cast in stone in knee resurfacing yet.

Knee Resurfacing Future Ahead

Newer designed minimally invasive knee resurfacing hemiarthroplasty appears to be safe and can provide effective pain relief along with rapid recovery and excellent functional result, especially with optimal pre-operative and ongoing post-operative patient education.

Refinement in implant design and materials, techniques and indications is on the horizon.

Open communication within the orthopedic community will be required to assess the relative merits of various established and emerging technologies regarding ever-changing advances in metallurgy, robotics and the potential for optimal patient outcomes.

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The Knee Joint

20

J. Sibbel and W. Khan

Introduction

This chapter will discuss the knee, the largest synovial joint in the body. It will cover the normal bony anatomy and geometry of the knee and how this gives rise to its complex biomechanics which go beyond a simple hinge. The factors stabilising the knee during its motion will be discussed and how these stabilisers have both primary and secondary restraint functions. It will also discuss our current understanding of pathophysiology of the most common degenerative disease affecting the knee, osteoarthritis as well as its clinical and radiological features, with a brief discussion of the various management options available.

Osseous Anatomy

The knee is comprised of three bones: the femur, tibia and patella and the two joints formed by their articulations, the tibiofemoral and patellofemoral joints. The distal femur is composed of medial and lateral condyles, the former being the larger of the two. These articulate with two corresponding tibial facets: a medial concave facet which is ovoid in shape and a lateral convex facet which is more circular and smaller than the medial side. The difference in the size and shape of these two facets has important effects on the kinematics of knee which will be discussed later in this chapter. The patella is the largest sesamoid bone in the body, which is invested in the retinaculum of the knee, receiving direct insertions from the quadriceps tendon proximally and patellar tendon distally. The articular surface is divided by a vertical ridge into a broader lateral facet which articulates with the correspondingly broad lateral aspect of the femoral intercondylar groove or trochlear groove. The primary function of the patella is to act as a fulcrum for quadriceps mediated knee extension.

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Biomechanics

Tibiofemoral Joint

The knee is far from a simple hinge joint. Through the range of motion of the knee from extension to flexion, the point of contact between the femur and tibia moves posteriorly in what's known as *femoral rollback*. Rollback increases the clearance of the posterior surface of the femur on the tibia and thus allows deeper flexion to occur.

It was formerly thought that this occurred to the same extent both medially and laterally as part of a four-bar linkage system [1]. A four-bar link is the simplest moveable closed-chain mechanism in mechanics. These are efficient at transferring motion and power. In the knee's case this link would be formed by the anterior and posterior cruciate ligaments and the bony bridges between their femoral origins and tibial insertions. However, MRI studies have since shown that the contact point in the lateral compartment moves posteriorly by 20 mm from −5° to 120° of flexion, whereas the medial contact point hardly moves at all, resulting in internal rotation of the tibia in relation to the femur during flexion [2]. The corollary of this is that at the extremes of extension the tibia externally rotates on the femur in what's known as the *screw home mechanism* as it results in the tightening of both the ACL and PCL, 'locking' the knee into place in a position of efficient stability while standing. This is unlocked by the action of popliteus which internally rotates the tibia and is therefore responsible for the initiation of flexion from full extension.

The reason this asymmetrical rollback and subsequent rotation occurs is due to both the bony and meniscal anatomy. The medial femoral condyle's increased size, the concavity of the medial tibial plateau and the relatively fixed medial meniscus result in little movement medially. In contrast, the lateral meniscus is much more mobile, to the extent that at full flexion the posterior horn of the lateral meniscus actually shares half the load of the lateral femoral condyle with the tibia. This, in conjunction with the femoral condyle's decreased size and convex tibial plateau, laterally results in the increased movement and the kinematics described above.

Knee Stability

Static stability in the knee is provided by the alignment and geometry of the articulating bones and the tension of the connecting ligaments. There are four main ligaments which contribute: the anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), medial collateral ligament (MCL) and lateral collateral ligament (LCL). The posteromedial and posterolateral structures and the iliotibial band also contribute. Each of these stabilisers acts as *primary and secondary restraint* to the different translational, rotational and varus/valgus stresses the knee undergoes [3, 4].

Anterior Cruciate Ligament

This arises from the lateral femoral condyle at the posteromedial aspect and inserts onto the tibia in a broad footprint between the intercondylar eminences. It acts as a primary restraint to both *anterior translation* and *internal rotation* and a secondary restraint to both varus and valgus forces. At 30° of knee flexion it provides approximately 85% of resistance to anterior translation. It is composed of two functional bundles, the anteromedial and posterolateral bundles, which are named for their tibial insertion and divided at their origin by the bifurcate ridge. In flexion, the anteromedial bundle is tighter and resists anterior translation. The posterolateral bundle is tighter in extension and also contributes to resisting rotation [5].

It was previously proposed that more anatomic ACL reconstructions using two bundles rather than one would yield more physiological results; however recent meta-analyses of randomised controlled trials comparing the two treatment methods have shown no significant difference in knee stability, clinical function, graft failure rate or osteoarthritic changes at mid- to long-term follow-up [6].

Posterior Cruciate Ligament

This arises from a broad crescent-shaped footprint on the anterolateral aspect of the medial femoral condyle and inserts on the tibial sulcus below the articular surface of the knee. The PCL acts a primary restraint to *posterior tibial translation*, providing 95% of the resistance at 90° of knee flexion, and secondary restraint to external rotation, varus/valgus stress and hyperextension. Again, it is composed of two bundles, in this case the anterolateral and posteromedial bundles which are tight in flexion and extension, respectively. These bundles act reciprocally and resist posterior translation at all degrees of knee flexion.

Note: it is therefore the more anterior bundle in both the ACL and PCL which is tighter in flexion and the more posterior bundle which is tighter in extension.

Medial Collateral Ligament

The MCL is composed of superficial and deep fibres with different functions. The superficial component originates from medial epicondyle of the femur and inserts directly into the proximal tibia, running deep to the tendons of gracilis and semitendinosus. It is the primary restraint to *valgus stress* at all angles of flexion, but has its least effect at full extension where the posteromedial capsule becomes tight and contributes more resistance. It has its greatest effect at 25–30° of knee flexion and therefore applying valgus stress at this point isolates the superficial MCL clinically. It also acts as a secondary restraint to external rotation and anterior translation. The deep MCL is a thickening of the medial joint capsule which attached to the medial meniscus. It acts only as a secondary restraint to valgus stress, mainly at full knee extension.

Lateral Collateral Ligament

It originates on the lateral femoral condyle posterior and superior to the femoral insertion of the popliteus tendon, runs superficial to popliteus and inserts onto the

anterolateral surface of the fibular head. It acts as the primary restraint to varus at all degrees of flexion and a secondary restraint to posterior translation and external rotation.

The management of injuries to these ligaments will be discussed in another chapter.

Patellofemoral Joint

The patella is a sesamoid bone embedded in the quadriceps tendon and patellar tendon, which acts as a lever arm to increase the power and efficiency of the quadriceps extensor mechanism. The forces applied to the patella during knee flexion are extremely high, approximately six times a person's body weight at 90° of flexion, which is reflected in the fact that it has the thickest articular cartilage in the body. The patella first engages the trochlea of the distal femur at 20° of flexion, when the distal pole of the patella makes contact. This contact point moves proximally as the knee flexes, and after 120° of flexion only the medial facet of the patella remains in contact with the corresponding femoral condyle [7, 8].

Patellofemoral joint (PFJ) stability is determined by several factors:

1. Q angle: This is the angle formed by the intersection of a line from the anterior superior iliac spine to the centre of the patella and from there to the tibial tubercle. The normal range is 5–20°. Higher Q angles than this increase the propensity for lateral dislocation and lateral facet degenerative disease.
2. Trochlear geometry: The trochlea is normally a concave groove which confers significant inherent stability once the patella has engaged it. The normal sulcus angle on a Merchant view radiograph is 138 ± 6° [9]. When this angle is increased, i.e. the trochlear is more shallow, it is said to be dysplastic and causes instability. This can be seen on a plain lateral radiograph in what's known as the *crossing sign*, whereby the trochlear groove lies in the same plane as the anterior border of the lateral femoral condyle.
3. Patellar height: As mentioned above, the patella normally engages the trochlea in early flexion; however this is dependent on normal patellar height, and therefore in *patella alta* this early engagement does not occur [10]. This can be diagnosed on a plain lateral radiograph with the knee flexed to 30° using multiple indices, the most widely used being the Insall-Salvati ratio [11]. This is the ratio of the patellar tendon length to the length of the patella, the normal range being 0.8–1.2, higher than this indicates patella alta.
4. Medial structures: The medial patellofemoral ligament (MPFL) is a condensation of medial capsular fibres which originates from the medial femoral condyle and inserts onto the superomedial aspect of the patella. It is the primary static restraint to lateral patella displacement. It is in continuity with the deep retinacular surface of vastus medialis obliquus (VMO) which provides significant dynamic medialising stability to the patella. These medial structures are often injured in acute patellar dislocations and can lead to recurrent instability. Physiotherapy to strengthen them is therefore part of first-line treatment.

When conservative measures fail, surgical management options for recurrent patellofemoral instability aim to address one or several of the factors mentioned above, either through pure soft tissue procedures such as MPFL reconstruction, bony work such as trochleoplasty or tibial tubercle transfer to decrease the Q angle or a combination of both [12].

Osteoarthritis of the Knee

Osteoarthritis (OA) is a chronic degenerative joint disorder which is characterised by the progressive breakdown of articular cartilage. It can affect any joint in the body, but most significantly affects the weight-bearing joints of the lower limb, causing pain, swelling and stiffness leading to impaired function. OA of the hip and knee is associated with substantial disability worldwide; according to the World Health Organisation Global Burden of Disease Study 2010 hip and knee OA is the 11th leading cause of disability worldwide and the number one leading cause of lower limb disability in older adults. Knee OA in particular affects over 70 million Europeans with a direct economic cost of over €2 billion. Its prevalence increases with age and obesity, and due to increasing life expectancy and obesity across Europe and North America, the global burden of OA will continue to increase.

Risk factors can be divided into modifiable (obesity, joint trauma, sedentary lifestyle, occupational activities) and non-modifiable (gender (females > males), increased age, genetics and possibly race).

Pathophysiology of OA

The pathology underlying development of OA is still not fully understood but certain key factors are becoming clearer. The initiation and progression of OA are increasingly seen as two distinct pathophysiological processes, i.e. an early and late phase. Understanding the different events involved in the initiation of articular cartilage damage and the progression of this damage to complete cartilage loss and severe OA will help to explain why everyone does not inevitably develop OA when they get older even though everybody will have accumulated articular cartilage damage by the time they reach a certain age. Two important aspects to the disease process appear to be the reactivation of secondary ossification centres causing subchondral bone remodelling and the action of proteolytic enzymes and inflammatory cytokines at the cell-matrix interface in the articular cartilage itself.

Subchondral bone has important mechanical effects on the overlying articular cartilage. Increased stiffness in subchondral cortical bone has been shown to increase mechanical stress in the deep zones of hyaline cartilage by up to 50% [13]. The classic clinical sign of radiological *subchondral sclerosis* is a consequence of decreased bone remodelling and increased subchondral plate density in late-stage OA, which subsequently contributes to overlying cartilage breakdown through increased mechanical stress. In early-stage OA, however, endochondral ossification

centres reactivate and subchondral remodelling occurs at a much higher rate than normal in response to factors that are not fully understood [14], but which results in increased vascularisation and subsequent disinhibition by vascular growth factors of matrix metalloproteinases (MMPs). This causes cartilage degradation and a positive feedback loop of increased bone remodelling as the joint attempts to adapt to altered force loading, manifesting as another classical radiological sign of *osteophyte formation* [15, 16]. The late-stage sign of *subchondral cyst formation* is another consequence of this abnormal bone remodelling process due to decreased bone mineralisation despite increased subchondral density, an apparent paradox which appears to be explained by findings that although osteoblasts in late-stage OA produce increased amounts of type-1 collagen, its collagen chain structure differs to normal in a way that reduces its affinity for calcium [17]. There is some evidence that these different early- and late-stage processes are both necessary for OA progression, as experimental induction of subchondral stiffness alone without the precursor phase of increased remodelling does not result in cartilage degradation [13].

Cartilage damage also alters the interaction between chondrocytes and their surrounding cellular matrix in adult hyaline cartilage, normally mediated by mechanosensors like Indian hedgehog (Ihh) and connexin 43 [18, 19]. This leads to abnormal cell signalling which further promotes the activation of MMPs as well as inflammatory mediators like IL-1β, TNF-α and IL-8 which can themselves increased the synthesis of MMPs [20]. These cause further breakdown of the cartilage matrix as well as inflammation and vascularisation of the surrounding synovium, which in turn further disinhibits proteolytic enzyme action via the loss of synovial B cells [16].

The articular cartilage degradation that takes place in OA is therefore different to the normal ‘wear and tear’ of ageing, and this can be seen in the different structural characteristics of normally ageing cartilage and in cartilage of joints with OA [21]. This is summarised in Fig. 20.1.

Clinical Features and Management

Patients with osteoarthritis of the knee complain of pain on weight-bearing which can severely affect their function, including their mobility distance and their ability to use stairs if their patellofemoral compartment is involved. They can also complain of swelling aggravated by activity, stiffness which is worse later in the day, night pain which wakes them and mechanical symptoms such as locking or giving way of the knee.

Clinical assessment must include determining whether a fixed flexion deformity is present, overall limb alignment including varus/valgus deformity, and assessing ligamentous integrity, as these will all affect surgical management options [22]. Plain radiographs of the weight-bearing knee in both AP and lateral views are essential, and sunrise views can be obtained to assess the patellofemoral compartment. The classical radiological signs of osteoarthritis are shown in Fig. 20.2: (1) joint

	Normal ageing	Osteoarthritis
Water content	Decreased	Increased
Collagen	Unchanged	Disorganised
Proteoglycan content	Decreased	Decreased
Proteoglycan synthesis	Unchanged	Increased
Chondrocyte number	Decreased	Unchanged
Chondrocyte size	Increased	Unchanged
Elastic modulus	Increased (i.e. more stiff)	Decreased (i.e. less stiff)

Fig. 20.1 Changes in articular cartilage in normal ageing compared to osteoarthritis

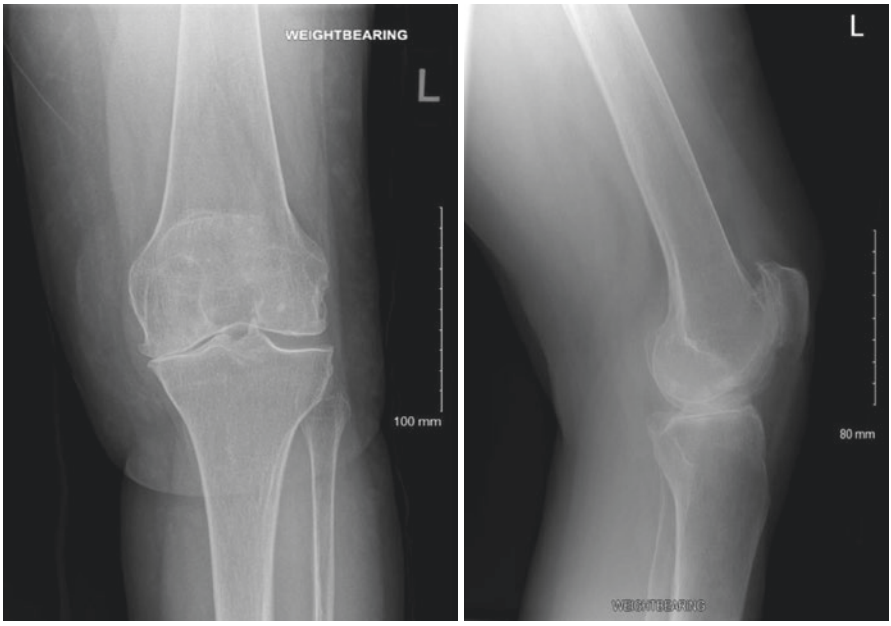

Fig. 20.2 Plain AP and lateral radiographs of a left knee showing osteoarthritis

space narrowing, (2) osteophyte formation, (3) subchondral cyst formation and (4) subchondral sclerosis. There is also a varus deformity present which is common in advanced OA, as well as marked patellofemoral joint involvement. When examining this patient it would be important to specifically assess the laxity of their lateral collateral ligament. In more severe varus or valgus deformities the collateral ligaments may become incompetent which would have important implications in their surgical management and the degree of constraint in the implant chosen.

Management of symptomatic knee osteoarthritis must always begin with non-operative options. These include the use of NSAIDs, physiotherapy focusing on quadriceps strengthening, activity modification and weight loss when BMI is above 25. When these fail, there are multiple surgical options depending on the patient demographic. These include high tibial osteotomy for patients below the age of 50 with unicompartmental disease; unicompartmental arthroplasty for patients with isolated disease, most often the medial compartment; and total knee arthroplasty. These will be further discussed in a separate chapter.

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Cartilage Injuries in the Knee

21

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Cartilage defects of the knee affect up to 66% of patients undergoing knee arthroscopies and 16–46% of patients with acute ACL injuries, with 20% of patients having chondral defects [1, 2]. Other associated injuries include medial meniscus tear of 37% which should be inspected for during arthroscopy [1, 2]. The treatment of articular cartilage is crucial in preventing progressive damage and degeneration of the knee joint. The challenge lies in the lack of blood supply and poor healing, which involves scarring and overgrowth of fibrocartilage and type 1 collagen in place of hyaline cartilage [3]. This is crucial to the function of cartilage to maintain low-friction lubrication when forces are generated during joint loading and weight bearing to reduce wear and degradation.

It can be broadly classified into traumatic and nontraumatic lesions (such as osteochondritis dissecans or OCD), and depth is graded using the International Cartilage Repair Society (ICRS) grading system [4]. The Outerbridge classification (Fig. 21.1) provides a similar grading system from grades 1–4 [5]; however, it should be noted that the difference between Outerbridge grades 2 and 3 is the size rather than the depth.

It can also be described according to the location, most commonly affecting the patella (36%) followed by medial femoral condyle (34%) [1, 2]. Isolated cartilage lesions account for 30% of cartilage defects compared to non-isolated of 70% which bears a poorer prognosis [1, 2]. These lesions should also be defined and managed by the size, degree of containment, and other associated knee pathologies which will be discussed further in the management section. Osteochondritis dissecans (OCD) poses its own prognoses and management strategies, which will be discussed separately to this chapter.

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Grades	ICRS	Outerbridge
Grade 0	Normal	Normal
Grade 1	Superficial lesions	Softening and swelling
Grade 2	Lesion extends <50% of depth	Fragmentation and fissuring < 0.5 inch in diameter
Grade 3	Severe (lesion extends >50% of depth)	Fragmentation and fissuring > 0.5 inch in diameter
Grade 4	Through the subchondral bone	Erosion of cartilage down to bone

Fig. 21.1 ICRS grading system and Outerbridge classification

Pathophysiology

The normal articular cartilage is divided into the superficial, middle, and deep zone [6]. The tide mark forms the boundary between calcified cartilage and superficial layers [6]. This is crucial as injuries crossing the tide mark lead to formation of hematoma, stem cell migration, and vascular ingrowth, which leads to fibrocartilage formation [6].

Sixty-eight percent of cartilage lesions are a result of trauma, from major events or minor repetitive trauma. Biomechanically during impact loading, the force produces a nonuniform force in the form of increased hydrostatic pressure, stress, shear, and compression through all layers of the cartilage [3]. Repeated trauma or presence of acute damage to the joint introduces synovial inflammation in the joint, upregulating arachidonic acid, IL-1, TNF-α, and metalloprotease [3, 6]. Loading on a cartilage defect can lead to matrix metalloproteinase production, which damages surrounding cartilage [3, 6]. Factors such as age of patient, size of lesion and containment, ligamentous and meniscal integrity, and overall alignment are factors that need to be considered in the surgical planning to prevent early onset of osteoarthritis [1–6].

Symptoms

An accurate record of the history of injury should be detailed prior to the physical examination. Patients commonly present after a traumatic event; usually a rotational force through the knee and often cartilage defects are overlooked. It typically presents with pain, swelling, and decreased range of motion.

In chronic presentations, note from the history of associated pathology that can cause knee pain, such as locking, instability, and clicking. Special tests such as McMurray's and anterior drawer test are useful in assessing concomitant injuries such as meniscal tear and ACL injuries. Other associated pathologies and past medical history should also be evaluated as this can affect the management plan, which includes history of rheumatoid arthritis, collagen disorders, HLA B27, and systemic lupus erythematosus. Within the adolescent group of patients, recurrent patella dislocation or signs of patella instability are associated with a high incidence of cartilage defect of up to 70–93% primarily involving the lateral femoral condyle and medial patella facet [7].

Investigation

Plain radiographs can be obtained to rule out any acute fractures and assessing the presence of lipohemarthrosis or tendon injuries. A skyline view is useful in assessing for patellofemoral diseases. Other causes of knee pain can also be investigated including osteoarthritis, bony spurs, cyst, and osteochondral defect. However, in cases of OCD, plain radiographs are unable to describe the stability of the lesion. Similarly, CT can be obtained to assess the osseous structures, especially to evaluate subchondral involvement and in measuring TT–TG distance as a cause for patellofemoral cartilage lesions. However, the dose of ionizing radiation should be put into consideration especially in the adolescent group of patients [8].

MRI remains the gold standard in assessing the cartilage lesions and other pathologies in the knee joint, typically meniscal and ligamentous. It can also visualize the depth of the lesion, bone bruising, as well as subchondral zones. MRI provides a multiplanar assessment in cartilage-sensitive sequences without ionizing radiation. On the MRI scan, the cartilage injury is best interpreted in fat-suppressed T2 sequence [9]. Arthrogram and dGEMRIC (delayed gadolinium-enhanced MRI of cartilage), 3D mapping, and needle arthroscopy can also be utilized for evaluating cartilage defects in specialist centers [6, 9].

For patients with previous surgical repairs of cartilage lesion, MRI can also be utilized as a noninvasive method to assess response to surgery, cartilage repair, and growth during the postoperative phase. Features that can be quantified include the following [9]:

1. Morphological features (incorporation of graft or quality of cartilage growth).
2. Bony overgrowth or hypertrophy of graft.
3. Periosteal detachment.
4. Percentage of filling.
5. Presence of fissures.

MRI can quantify the intensity of grafts compared to hyaline cartilage in the cases of ACI and extent of hypertrophy. It is also able to quantify whether the

periosteal graft is intact, detached, or fully incorporated [10]. MR imaging is also able to provide longitudinal quantification of the cartilage, which when correlated clinically can be used to decide if revision surgery is required to address potential complications. MRI can also assess whether the graft or cartilage growth is flush, proud, or defective, which is crucial during weight bearing for equal load distribution in the knee joint [10, 11]. In cases of microfracture and marrow stimulation techniques, bony overgrowth can be evaluated along with the quality of the subchondral plate [11]. In specialist centers, MR arthrogram utilizing intravenous gadolinium can be used to visualize presence of fluid within the repair tissue that can be used to positively predict traumatic chondral delamination [11]. However, when evaluating repair cartilage in MRI, the signal can be brighter than hyaline cartilage due to the heterogenous nature, usually with mixture of hyaline and fibrocartilage. In serial MR imaging for patients receiving microfracture and marrow stimulation techniques, intermediate signal intensity within the defect in asymptomatic patients has shown progressive filling in the defect with improvement of bone marrow edema [11].

Treatment

The main strategies of treatment are to attempt at restoring hyaline cartilage, preserving a smooth lubricated joint to allow equal loading, and, ultimately, reducing the risk of degenerative joint disease. Several methods of treatment have been described including debridement, microfracture, osteochondral autologous transplantation (OAT), and autologous chondrocyte implantation (ACI).

The treatment algorithm should be tailored to various factors including [1–6, 8–11]:

1. Patient factors.
2. Isolated or associated meniscal/ligamentous pathologies.
3. Presence of chondral defect/bone less.
4. Number of lesion(s).
5. Size of lesion(s).
6. Site of lesion(s).

Besides surgical strategies in addressing the cartilage injury, we should always consider the patient holistically including occupational and functional status, BMI, physical demand, age and skeletal maturity, limb–leg alignment, comorbidities, and ability to tolerate rehabilitation program. Primary arthroplasty and osteotomies can be considered in patients with severe degenerative joint disease or osteoarthritis. In general, large (>2.5 cm [2]), multifocal lesions as well as patellofemoral joint lesions have technically more challenging treatment options and poorer outcomes [1–6].

Conservative

For young patients with isolated cartilage defects, a period of observation can be utilized along with analgesia and physiotherapy. Other conservative measures include biological injections, such as corticosteroids, hyaluronic acid, and platelet-rich plasma (PRP). These can also be utilized in association with surgical management.

Hyaluronic acid provides chondroprotection properties, which prevents inhibition of collagen synthesis [12]. It can provide temporary relief to patients with osteoarthritic changes and can delay the need for replacement, especially when paired with corticosteroids [12].

PRP has seen recent trends as a treatment option. PRP is obtained through autologous blood products after centrifugation—it is broadly defined by whether it is leucocyte rich or leucocyte poor [13]. Generally, leucocyte-rich PRP is used in tendon pathologies and produces a higher local inflammatory response. In leucocyte-poor PRP, the leucocyte concentration is three times lower than the basal concentration and technically requires shorter centrifugation time and less blood product [13]. It confers expression of type 2 collagen and anabolism of chondrocytes [13]. Other biologics include cell therapy in the form of mesenchymal stem cells.

Surgical Management

1. Debridement—In patients with symptomatic chondral defects, the presence of cartilage will increase the levels of metalloproteinase, producing damaging effects on existing cartilage. Therefore, simple excision remains a viable surgical option and can provide symptomatic relief of 5 years or more in patients with local tenderness and coexisting isolated chondral defect [4, 6, 8]. The aim is to debride any unstable cartilage and calcified cartilage to promote healing and new tissue formation at the base of the chondral defects.
2. Microfracture and marrow stimulation technique—The method of microfracture was first described by Steadman et al., which combines debridement of unstable and damaged cartilage [6, 14]. Microfracture differed from drilling first described by Pridie as it aims to expose the subchondral plate to pluripotent marrow cells, reduce thermal damage from drilling, and produce a layer of rough surface for new tissue to adhere to [6, 14]. The procedure involves debridement to edge of healthy cartilage with vertical walls and producing multiple holes using arthroscopic awls that are 3–4 mms apart while ensuring the subchondral plate is intact [14]. Patients are generally restricted in weight bearing post procedure, with continuous passive motion forming the early part of the rehabilitation program. Postoperative histological findings from 20 biopsies show that 11.4% had mainly hyaline cartilage, while 17.1% mixed fibrocartilage and hyaline cartilage [11, 14].

3. Osteochondral autologous transplantation (OAT)—OAT or osteochondral cylinder transplantation is also known in literature as mosaicplasty, where it utilizes a cylindrical device to cut and transplant an osteochondral plug to the defect [4]. This method confers advantages as the defect is filled with mature hyaline cartilage. The usual sites for harvesting osteochondral cylinders are from the lateral and medial margins of the femoral trochlea as well as intercondylar notch. It is recommended that the lesion should be between 1 and 4 cm [2]. A press fit donor transplant to lesion (ideally slightly larger by 0.1 mm) with similar thickness is recommended to reduce the risk of lack of lateral integration [3, 4, 6]. This can result to cyst formation from synovial fluid penetration. Besides that, the graft should lie flush, as sunken grafts can cause necrosis and proud grafts can cause fissuring and fibroplasia [3, 4, 6]. OAT is reserved for larger lesions and patients with normal alignment as excessive pressure can cause cartilage cell death [3, 4, 6]. In chondral defects in athletes, OAT and ACI provide better outcomes than microfractures with faster return to sports [3, 4, 6, 14].
4. Autologous chondrocyte implantation (ACI)—ACI techniques have evolved since it was first described and involve arthroscopically harvesting cartilage. Chondrocytes are subsequently released from the matrix, culture-expanded and injected underneath the periosteum sewn over the defect on second surgery [15]. Like OAT, ACI can produce hyaline cartilage and can be utilized in large defects [4, 15]. Techniques have improved in recent years to involve culturing chondrocytes on collagen membranes and scaffold (matrix-induced ACI or MACI™ [4, 6, 8–15]), which can produce less hypertrophy than using the periosteum. MACI has been described to reduce surgical time and the ability for implantation with minimally invasive techniques such as mini-arthrotomy or arthroscopically [15]. The SUMMIT trial recently concluded that MACI provided better patient outcomes compared to microfracture in large lesions >4 cm2 in patients with symptomatic articular defects at 2 years [15].

Complications and Prognosis

The mainstay of complications to include during the consent process includes those of knee arthroscopy, whose main features include infection and pulmonary thromboembolism, and revision surgery occurs for reasons discussed below, persistent pain and anterior knee pain.

The potential modes of failures of grafts and marrow stimulation techniques are outlined in investigations, where MR imaging can evaluate presence of fissures, tissue hypertrophy or bony overgrowth, delamination, and presence of incorporation [9–11]. In the presence of patients who are symptomatic with MRI suggesting cartilage hypertrophy, bony overgrowth, or periosteal hypertrophy, debridement is viable surgical management [9–11]. Lack of filling of cartilage in microfracture patients can affect functional outcome significantly. The revision rates for microfractures are 2.5% in less than 24 months and 2–31% after 24 months from a recent evidence-based systematic analysis [11]. In a recent

case series of patients receiving ACI, 52 out of 309 (15%) of patients require revision surgery, for symptomatic hypertrophy, delamination, graft failure, and disturbed fusion of cartilage [10].

Osteochondritis Dissecans (OCD)

OCD refers to a very specific disease that affects primarily adolescence and is a common cause of knee pain, most commonly affecting those who are still skeletally immature [16]. OCD involves the subchondral bone leading to delamination of cartilage and instability of the fragment. It affects males more than females. OCD is estimated to be approximately 2% in all knee arthroscopies [1].

As per cartilage injuries, it is quantified morphologically, by site, stability, and skeletal maturity of the patient. Most commonly it affects the posterolateral aspect of the medial femoral condyle (70%) and inferolateral condyle (20%) [8, 16]. The term "OCD" most commonly in literature describes an idiopathic lesion that should not be confused with other diagnoses or osteochondral lesions due to avascular necrosis secondary to chemotherapy or steroids [8, 16]. Various causes of OCD have been postulated and disregarded such as genetics, inflammatory, and avascular necrosis; however, repetitive trauma causing stress fracture of the subchondral bone seems prevalent as the prevalence of OCD in young female athletes increases. Nonetheless, the distinction and etiology of juvenile and adult de novo OCD remains controversial, for which both confer different management strategies and provide different prognoses. The onset of OCD in adults is associated with greater instability and is often not amendable to conservative management with a length of protective weight bearing. The age of onset affects the healing process where 9% of children develops osteoarthritis in contrast to 81% of those in their adulthood [6, 8, 16].

Presentation and Investigation

Patients typically present with nonspecific knee pain, which can cause mechanical symptoms following an insidious onset if the lesion is unstable and loose. Mechanical symptoms include locking, catching, pain on active motion, and crepitus on physical examination. It is crucial to illicit from the history of trauma and other concomitant soft tissue knee injuries such as ACL and meniscal injuries. Up to 25% of cases involve the contralateral knee, which is crucial to enquire and examine bilateral knees [8]. Quadriceps atrophy can be present to indicate the chronicity of the pathology. A Wilson's test can be used to elicit discomfort when internally rotating the tibia during extension of the knee between 30° and 90° and relieved with internal rotation of the tibia as this corresponds to the most common site of the OCD, which is the lateral aspect of the distal medial femoral condyle [8, 16].

As with cartilage injuries to the knee, it is important to obtain baseline investigations such as plain film radiographs, and MRI remains the gold standard modality to illustrate other associated pathologies. Plain film radiograph views include AP,

Stage	Description
1	Small change of signal without clear margins of fragment
2	Osteochondral fragment with clear margins but without fluid between fragment and underlying bone
3	Fluid invisible partially between fragment and underlying bone
4	Fluid is completely surrounding the fragment, but the fragment is still in situ
5	Fragment is completely detached and displaced (loose body)

Fig. 21.2 MRI classification for juvenile OCD [8]

lateral, and notch views which can be useful to visualize the posterior aspect of the femoral condyles.

MR imaging can be used to classify juvenile OCD (Fig. 21.2). It has been demonstrated that patients with the presence of high signal line behind the fragment on MRI had the highest rate of failed nonoperative management, which is best visualized in gadolinium-enhanced scans [8]. Besides that, size and site, extent of bony edema, presence of loose bodies, and involvement of subchondral bone can be assessed from the MR images [8].

Treatment and Prognosis

Conservative treatment can be advocated in 50% of juvenile OCD with open physis and compliance to restricted weight bearing, and it has been shown that the OCD heals in 10–18 months. A rehabilitation program should be agreed and protocolized with interval and serial imaging with an initial phase of protected weight bearing in a cast or immobilizer. This is followed by low-impact exercise and gradual return to sports after the patient becomes asymptomatic.

Young patients with closed physis and adult OCD should be considered for surgical management [8, 16]. OCD can be classified intraoperatively according to their grade as per the Guhl classification [8, 16]. Stage 1 involves the lesion as stable and intact, where stage 2 involves fragmentation in situ and early separation. In stage 3, there is partial detachment of the lesion, and in stage 4, the lesion is completely detached and often loose bodies are present.

Treatment options should be guided by patient's age, site of lesion, stability, MRI findings, and skeletal maturity. The goal is to restore subchondral interface and preserve articular cartilage, which includes [8, 16]:

1. Fragment excision—Excising the OCD fragment can be utilized in young patients with healing potential. However, fragment excision provides short-term relief and poor functional outcome long term, with a poor return to sports and activities for patients [8, 16].
2. Repair—Drilling provides a viable treatment option for patients with low-grade [1 and 2] lesions with open physis. It can be used as an adjunct to improve blood supply and healing to repair options. Grade 3 and 4 lesions, which are generally more unstable, with associated loose bodies and flaps can be treated with reattachment [8, 16]. It involves debridement of the base and bony surface of the flap, with microfracturing to improve subchondral blood supply. The lesion can then be reduced bioabsorbable fixation or standard screws. Once union is achieved, metallic screws can be removed.
3. Restore—Surgical management involving restoration of articular cartilage has been previously described, which includes OAT, microfracture, and ACI. Restoration methods are generally preserved for patients that have failed conservative or reparation techniques or if the lesion is large, difficult to be reduced, unstable, and within the weight-bearing regions. Similarly, microfracture and marrow stimulation techniques should be reserved for smaller defects with OAT and ACI for larger lesions (>2 cm [2]). [8, 16]

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Meniscus Tears

22

Nadim Tarazi, Kendrick To, and Wasim S. Khan

Introduction

Till this day, meniscal tears remain to be one of the most common problems we deal with in knee surgery. In addition, arthroscopic meniscectomies are one of the most frequent procedures performed in orthopedics.

The history of meniscal surgery began when Thomas Annandale performed the first open meniscal repair in 1885. This hype did not last for long and in 1975 Smillie advocated meniscal resection. However, the discovery that removing the meniscus from the knee joint resulted in the deterioration of articular cartilage and the gradual development of arthritis radically changed the approach for treating meniscus-related problems. In 1982, partial meniscectomy was proposed to be an alternative to complete meniscectomy, and this soon became the sole technique for treating meniscal tears. It is only recently that meniscal preservation has received significant recognition. This current era of meniscal preservation is based on three pillars: repair of the torn meniscus whenever reasonable, nonsurgical treatment of asymptomatic meniscal pathologies despite a meniscal tear noted on an MRI, and partial meniscectomy with resection of as much as necessary and as little as possible. In fact, in 2013, a review of arthroscopic procedures found a doubling in the number of meniscal repairs performed in the past 5 years, without a concomitant increase in meniscectomies.

Structure and Function of the Knee Meniscus

Historically the menisci were described as a functionless embryonic remnant and hence commonly resected. The menisci are now known to play a key role in the normal biomechanical function and long-term health of the knee joint.

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The menisci are highly hydrated and composed of 70% water and 30% organic matter. In general, collagen constitutes the majority (75%) of the organic matter. The remainder of the dry matter is made of non-collagenous proteins such as glycosaminoglycan, as well as DNA and elastin. Type I collagen fibers provide the primary meniscal structural scaffolding; this predominance of type I collagen is one of the major differences between the menisci and hyaline, or articular cartilage, which is composed of predominantly type II collagen [1].

Three collagen fiber layers are specifically arranged to convert compressive loads into circumferential or "hoop" stresses. In the superficial layer, the fibers travel radially, serving as "ties" that resist shearing or splitting. In the middle layer, the fibers run parallel or circumferentially to resist hoop stress during weight bearing. Lastly, there is a deep layer of collagen bundles that are aligned parallel to the periphery. The morphology of this load-bearing structural complex determines its vital functions: load bearing, joint stability, joint congruity, increasing joint contact area, decreasing joint contact stresses, protection of articular cartilage, shock absorption, lubrication, limitation of extreme movement, and proprioception. The structural degeneration or deterioration of the menisci due to senescence, trauma, or surgery eventually leads to osteoarthritis of the knee joint, independent of meniscus morphology [1].

Both menisci are C-shaped and have concave upper surfaces and flat undersides that match their respective interfaces with the femoral condyles and tibial plateau.

The medial meniscus is more crescent in shape as the posterior horn of the medial meniscus is always larger than the anterior horn. It is also slightly smaller than the lateral meniscus and has a stronger capsular attachment which makes it less mobile and therefore more prone to injury. The lateral meniscus is usually longer, wider, and more mobile, with a variable shape, size, and mobility, ranging from C-shape to almost discoid shape. The medial meniscus covers 50–60% of the medial plateau, and the lateral meniscus covers 70–80% of the lateral plateau [2].

Menisci are stabilized by several ligaments and attachments. Anterior and posterior roots are attached to the tibia. Meniscotibial union is an enthesis, which is much stronger than capsular attachments. Anterior roots are joined by the anterior intermeniscal (transverse) ligament. Attachment to the capsule is provided by the coronary ligament and, in the case of the medial meniscus, by expansions of the medial collateral ligament also. Finally, meniscofemoral ligaments (Humphrey anteriorly and Wrisberg posteriorly) attach the posterior part of the lateral meniscus to the lateral wall of the internal femoral condyle, surrounding the posterior cruciate ligament (PCL). These attachments and ligaments are important as they act like a "belt" sustaining and facilitating menisci biomechanics [2].

Blood Supply of the Meniscus

Blood vessels arise from the lateral, middle, and medial geniculate arteries and penetrate through the joint capsule to form a perimeniscal capillary plexus, where radial branches enter the menisci and supply the peripheral quarter of the menisci (red

zone). In cadaveric studies, Arnoczky et al. [3] and Day et al. [4] found that radial branches penetrate the menisci to a depth of 2–3 mm, with the most consistent blood supply occurring at the anterior and posterior horns. Both studies found that the posterolateral aspect of the lateral meniscus, adjacent to the popliteus tendon, was avascular as was the inner 70–75% of the menisci (white zone). Cooper described these zones by dividing the meniscus into three radial sections (zones A, B, and C) from posterior to anterior and the width into three from peripheral to central.

In addition, the blood supply to the menisci varies with age. Petersen and Tillmann reviewed cadaveric specimens of 20 human menisci ranging in age from birth to 80 years old and found that at birth the whole meniscus was vascularized. By the second year, they had an avascular area on the inner circumference. By the age of 20, blood vessels were only present in the peripheral third, which further regressed to a quarter at the age of 50 [5].

Meniscal Tear Classification

Meniscal tears can be classified based on shape, size, chronicity, vascularity, and whether the tear is degenerative or traumatic. However, one increasingly used classification system is that of the ISAKOS classification of meniscal tears which has shown to provide sufficient interobserver reliability among the surgeons. It evaluates tears based on the following criteria [6]:

1. Tear depth: A partial tear involves either the superior or inferior surface of the meniscus. A full tear extends through both the superior and inferior meniscal surfaces.
2. Location/rim width (zones): Tears can involve more than one zone. Such tears should be graded based on how far the tear extends into the meniscus. For instance, a tear that involves zones 3, 2, and 1 should be graded as a zone 1 tear:
   - Zone 1 (red zone): Have a rim of less than 3 mm from the outer border of the meniscus.
   - Zone 2 (red-white zone): Have a rim of 3 to less than 5 mm.
   - Zone 3 (white zone): Have a rim width of more than 5 mm.
3. Radial location: Indicate whether the tear is posterior, mid-body, or anterior in location.
4. Relationship to popliteal hiatus: A tear of the lateral meniscus that extends partially or completely in front of the popliteal hiatus should be graded as central to the popliteal hiatus.
5. Tear pattern.
   - Horizontal tears: The most common tear pattern and usually occurs in the older population and can be associated with meniscal cysts. The tear lies parallel to the tibial plateau and separates the meniscus into upper and lower parts.
   - Vertical tears (longitudinal and radial): The most common to be repaired. They are classified into either longitudinal or radial tears. It is important to note that longitudinal and radial tears may both appear vertical on sagittal cuts

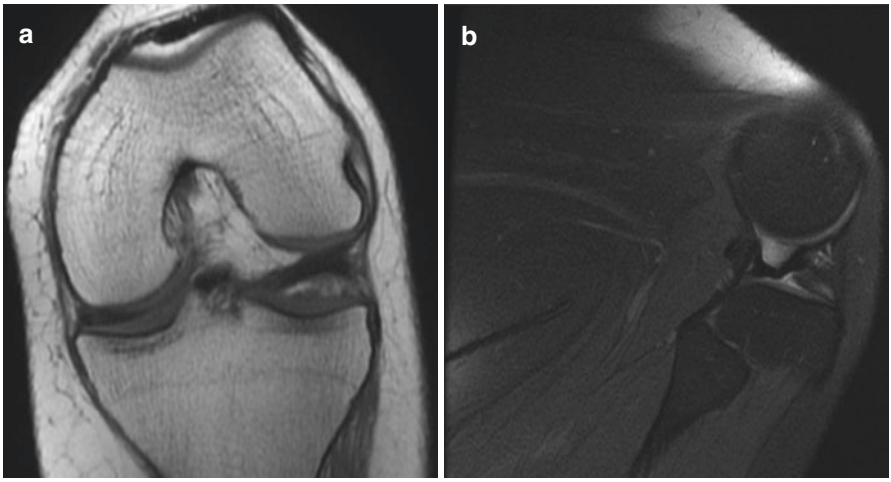

Fig. 22.1 Coronal (**a**) and sagittal (**b**) MRI demonstrating a flipped lateral meniscus with the knee in a locked position

of a MRI. However, in the setting of a longitudinal tear, the lesion extends parallel to the c-shaped circumference of the meniscus, whereas radial tears lie perpendicular to the meniscal circumference. As the two tears differ in prognosis and treatment approach, "vertical" is not the preferred term to describe a tear unless paired with the proper category (longitudinal vs radial). Bucket hand tears (Fig. 22.1) are a form of longitudinal tears in which subsequent displacement of the inner rim of the tear results in a configuration that resembles the handle of a bucket. They are unstable and are often associated with ACL ruptures.

- Oblique Tears (Parrot Beak): Such tears combine features of radial and longitudinal tears. They lie perpendicular to the free edge of the meniscus but then curve such that a portion of it lies parallel to the c-shaped fibers of the meniscus.
- Meniscal root tears: A form of radial tear that involves the central attachment of the meniscus and usually seen at the posterior horn. They are often large radial tears that extend through the entire AP width of the meniscus. The loss of the central attachment of the posterior horn may allow extrusion of the body of the meniscus relative to the joint. In addition, the large tear dramatically undermines the ability of the meniscus to distribute hoop stresses. Both of these factors increase contact forces across the joint, leading to accelerated osteoarthritis and predisposing the patient to the development of subchondral insufficiency fractures.
- Flap Tears: Where the meniscus tears within its midsubstance, usually in a predominantly horizontal pattern, and then the upper or lower component of the torn meniscus becomes displaced from its site of origin. These tears can be challenging to recognize on MRI but are important to diagnose since they

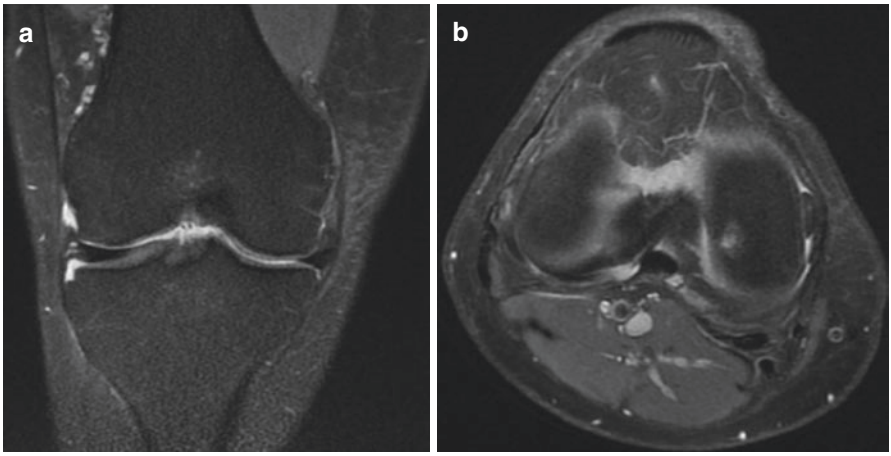

Fig. 22.2 Coronal (**a**) and axial (**b**) MRI views of a superiorly displaced flap tear of the medial meniscus

are often highly symptomatic due to a reactive synovitis. Arthroscopic treatment is typically required for adequate symptom relief in patients with displaced meniscal flap tears (Fig. 22.2).

6. Quality of tissue.
   - Degenerative.
   - Nondegenerative (traumatic).
   - Undetermined.

   Degenerative tears: Typically occur in middle-aged and older people. Degenerative tears occur due to repeated loads which in long term begin to ear out the meniscus as a result of years of microtraumas and aging of the meniscus. After the age of 40, the cellularity of menisci decreases and present cells suffer a process of senescence. It is believed that this could lead to a higher risk of tears and decreased potential ability to repair. The most common degenerative tear patterns are usually horizontal, radial, or complex, mostly affecting the posterior horn of the medial meniscus. Vertical tears are almost nonexistent in this group. It is very common to find osteoarthritis affecting the tibiofemoral compartment where the meniscal tear is. The prevalence of degenerative meniscal tears increases with age, ranging from 16% in the knees of women aged 50–59 years to over 50% in the knees of men aged 70–90 years [7]. These epidemiological data are important from two aspects: First, they demonstrate the remarkably high prevalence of meniscus lesions in the general population which may be considered part of normal aging. Second, most of these meniscus lesions do not directly cause knee symptoms as over 60% of tears were completely pain-free. Most degenerative cases respond adequately to conservative treatment with physical therapy and painkillers [8].

   Traumatic tears: Are due to an acute overload, generally associated with an adequate knee injury, which surpasses the normal resistance of meniscus tis-

sue, leading to a rupture. Vertical longitudinal tears are frequent and associated ligament injuries are not rare. Radial tears, flap tears, and most posterolateral root tears also commonly belong to this group.

Traumatic tears more commonly affect young patients who may benefit from surgical treatment more frequently. A traumatic tear can be defined as "stable" or "unstable" according to its mobility.

7. Length of tear in mm.
8. Indicating the amount of the meniscus that was excised and drawing it on a diagram.
9. Percentage of medial meniscus excised.

Clinical Evaluation

History taking should always be the first step in the clinical evaluation of meniscal tears as it gives important diagnostic clues. Younger patients frequently describe a sensation or hearing a "pop" during injury which commonly occurs after a sudden change in direction or pivoting with or without contact with another individual. Other symptoms include pain, subacute swelling, mechanical symptoms such as catching or locking, and limitation in range of movement. Physical examination should be done in a systematic fashion beginning from inspection to evaluation of ligament stability. Although special provocative signs for meniscal tears such as McMurray's test as well as joint-line tenderness are commonly present, their accuracy, specificity, and sensitivity are highly variable and questionable. In addition, the above mechanical symptoms and signs can be found to be secondary to arthritic changes or ligamentous injuries. It is reported that knee joint-line palpation may produce equivocal results, with medial joint-line specificity of 34.5% and sensitivity of 44.9% for predicting medial meniscal tears and lateral joint-line specificity of 49.1% and sensitivity of 57.6% for predicting lateral meniscal tears in subjects with acute ACL injuries [9]. However, among subjects with a non-impaired ACL, knee joint-line tenderness is more accurate, with a 77% clinical accuracy for meniscal tear identification [10, 11].

Therefore, if a meniscal tear is suspected following a thorough history and physical examination, radiographic assessment with an MRI remains to be the gold standard noninvasive method for the evaluation of meniscal tears. It is important to note, however, that plain radiographs should be the first-line imaging tool in the assessment of middle-aged and older patients as it can assess for the presence of osteoarthritis as well as for malalignment, concomitant bony pathology, etc.

Management

Nonoperative Management

Nonoperative management is useful for the initial management of traumatic meniscal tears and a first-line treatment method in degenerative meniscal tears. In the

setting of acute traumatic tears, the "PRICE" (protection, rest, ice, compression, elevation) protocol is applied. However, in the setting of degenerative meniscal tears, nonoperative management (anti-inflammatory and analgesic medications, quadriceps strengthening, activity modification, unloader bracing and intra-articular injections, etc.) rather than surgical methods should be tried for at least 3 to 6 months, if the mechanical symptoms do not dominate the clinical picture. If the patient's symptoms persist, then surgical treatment can be considered.

Recently there has been an ongoing debate relating to the management of degenerative meniscal tears in middle-aged and older people, comparing nonoperative treatment with arthroscopic meniscectomy. Based on recent data, the ESSKA Meniscus Consensus Project developed a decision algorithm (Fig. 22.3). In the painful knee affecting middle-aged or older people, plain X-rays should be taken as first line. MRI is not indicated at this stage, unless a diagnosis requiring complementary examination is suspected. Nonoperative treatment is initiated, comprising physiotherapy and possibly intra-articular injections. In case of failure at 3 months, an MRI is performed, to confirm the diagnosis of a degenerative meniscal lesion (Fig. 22.4), although it is still necessary to check that the lesion matches the symptoms. If radiography and MRI show no signs of advanced osteoarthritis, arthroscopy may be considered. In the setting of advanced OA, arthroscopic debridement shows no superiority than nonoperative measures.

The ESSKA consensus project for degenerative meniscal tears stated the below main messages:

1. APM (arthroscopic partial meniscectomy) should not be considered as the first-line treatment choice.
2. APM should only be proposed after a proper standardized imaging protocol.
3. APM can be proposed after 3 months of persistent pain/mechanical symptoms or earlier in case of considerable mechanical symptoms.
4. No APM should be proposed with advanced osteoarthritis.

Operative Management

Broadly speaking, there are three main surgical options for meniscal tears. This includes meniscectomy, meniscal repair, and meniscal reconstruction.

Meniscectomy

Meniscectomy can be performed totally or partially, open or arthroscopically. In the past when the meniscus was viewed as a vestigial structure, along with the technical challenge of meniscal repairs, total meniscectomy was the preferred option. Considering that early OA has been shown to be the long-term outcome following a total meniscectomy, there has been a change in the way we manage meniscal tears. In the modern era, total meniscectomy is almost never performed or advised as a primary procedure for meniscal tears. Partial meniscectomies have since replaced this along with other surgical options such as repair and reconstruction/transplant. In

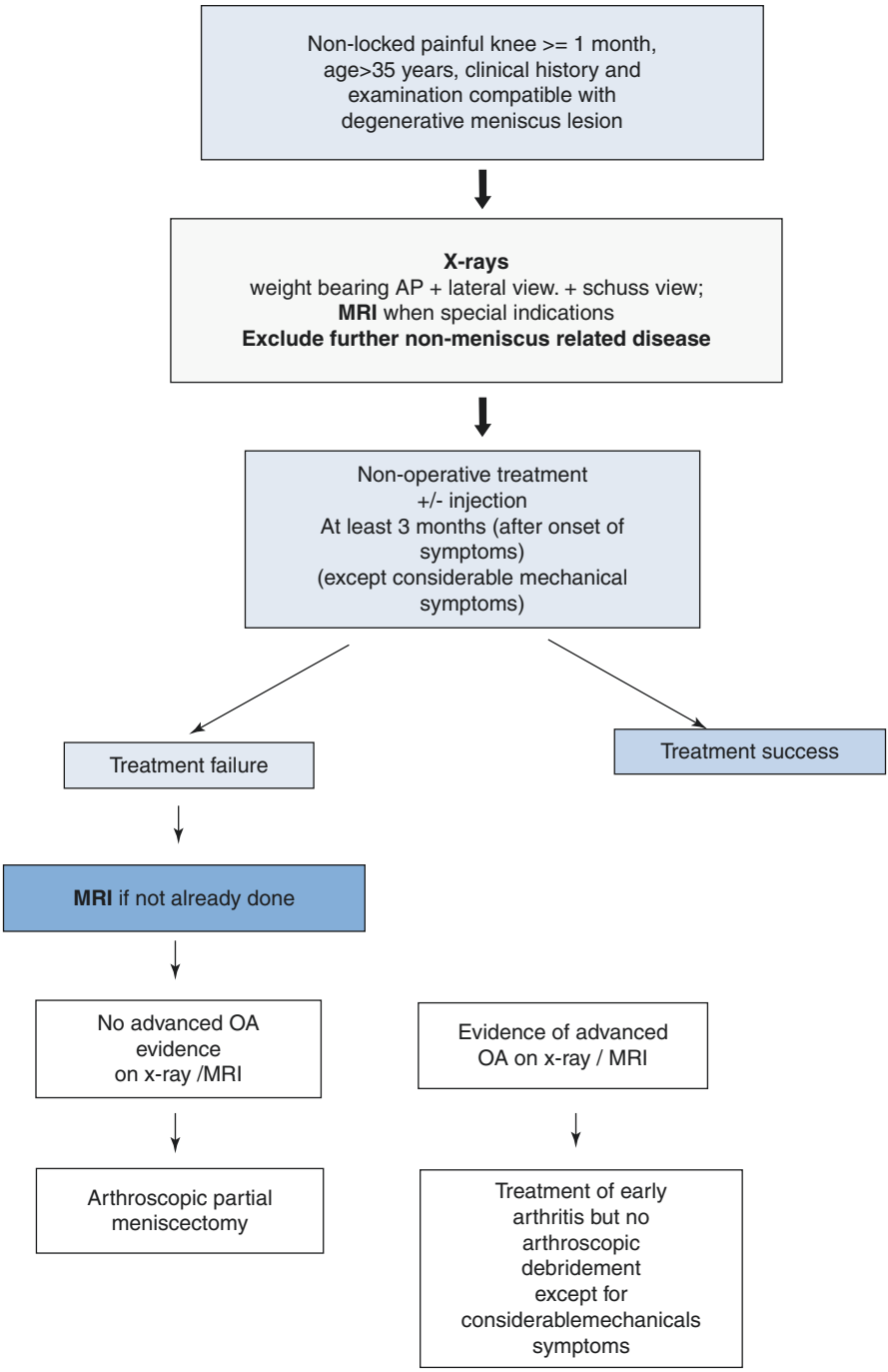

Fig. 22.3 Line diagram showing the ESSKA consensus project proposed the above algorithm for treatment of degenerative meniscal tears [6]

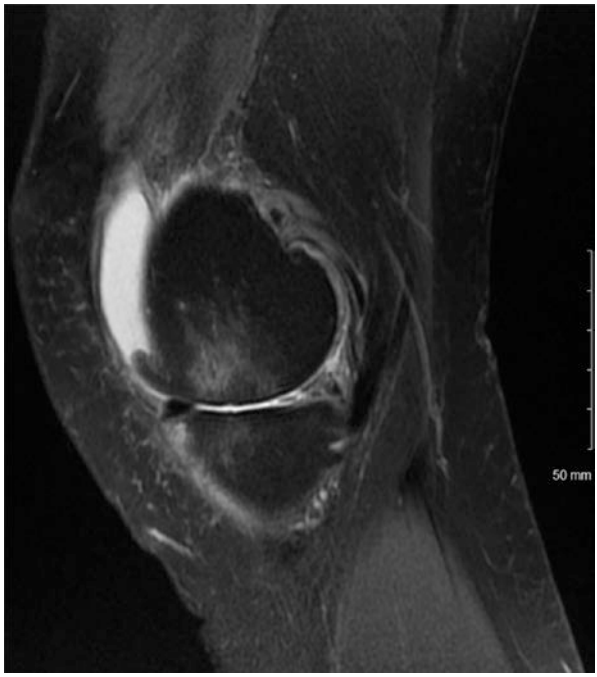

Fig. 22.4 MR image of a degenerative medial meniscal tear

fact, arthroscopic partial meniscectomy (APM) has become the most common surgical procedure performed for the treatment of meniscal tears. This is due to the rapid speed of the procedure, good short-term results, and low complication rate. Over the last decade or so, there have been multiple studies comparing the results of partial meniscectomy with that of nonoperative management or sham surgery. It is generally accepted that following APM, patients can expect an improvement in functional outcomes with good short-term results. However, most of the RCTs found no difference in terms of improvement of clinical outcomes in the long term after surgery compared to nonoperative treatment. Studies have also shown a high rate of progression to osteoarthritis at long term. Due to the above findings, APM should be the last resort for most meniscal tears. However, it may be applicable in carefully selected patients, such as those who have persistent mechanical symptoms from degenerative tears or symptomatic traumatic complex tears with a damaged meniscus and non-repairable tears in middle-aged patients with a non-symptomatic ACL injury.

It is certainly time to change the paradigm in meniscus management. Meniscectomy should not be the first-line choice. Meniscus repair in traumatic tears and nonoperative treatment in degenerative meniscal tears are the first answers.

Meniscal Repair

Introduction

Meniscal repair was initially performed by Scottish surgeon Sir Thomas Annandale in 1883. Following this breakthrough in the history of meniscus surgery, the first arthroscopic repair was performed by Ikeuchi (1969).

The main aim of meniscal repair is to achieve meniscal healing, avoiding the adverse effects of meniscectomy. Meniscal repair procedures are divided into two major types, open and arthroscopic. Although the open procedure is associated with greater tissue trauma with larger surgical incisions, there have been good results reported 10 years after open medial meniscal repair. Despite the good results, there has been increasing evolution from open to arthroscopic repair, and currently the latter predominates for meniscal tears. Indications for open meniscectomy are limited to posterior medial meniscal tears with a very tight medial compartment, if percutaneous release of medial collateral ligament does not provide enough visualization of the compartment. An open meniscal repair is performed through an incision posterior to the collateral ligaments, through the capsule and synovium to allow direct exposure to the torn meniscus.

Indications

Not all meniscus tears are amenable to repair, and several factors of both the meniscal tear and the patient must be considered to determine repair suitability and healing potential. The final determination of partial meniscectomy versus repair and method of repair is frequently made at the time of arthroscopy. The surgeon must therefore be prepared for several options.

Firstly, tear location is an important factor to consider. Based on the blood supply pattern, tears in the vascular periphery of the meniscus have the best ability to heal, whereas tears in the central white-white zone demonstrate poorer healing rates and are less amenable to repair. Meniscal tear orientation and complexity must also be considered. Longitudinal vertical tears, bucket handle tears, and meniscocapsular separations are most amenable to repair. Conversely, complex tears may be better managed with partial meniscectomy. These tears include degenerative tears, radial tears, and tears with horizontal cleavage planes or multiple flaps [12]. Oblique, undersurface tears can also be problematic because they often extend from the vascular zone to avascular zone.

Moreover, the type of the lesion (degenerative vs traumatic), age of the patient, and chronicity are also essential criteria to be assessed. Degenerative meniscal tears display poor repair potential due to the insufficient tissue integrity of both the lesion site and the adjacent meniscal tissue. Some authors have also recommended partial meniscectomy or repair for patients older than 30 years. Finally, many traumatic meniscal tears are associated with an ACL rupture. It is well known that meniscal repair has a higher success rate when performed in conjunction with ACL reconstruction in comparison to isolated meniscal repair.

Having taken the above into account, the most amenable tears to be repaired are acute, traumatic tears within the peripheral well-vascularized red-red zone which are longitudinal-vertical in orientation in young patients.

Techniques

Arthroscopic-Assisted Inside-Out and Outside-In Techniques

Traditionally, the inside-out technique was considered to be the gold standard as it allows more a more consistent suture placement, perpendicular to the tear.

In the inside-out technique, the sutures are introduced from inside the knee, engaging both fragments of the tear and passing through the capsule with them being knotted onto the capsule in a vertical mattress fashion. The sutures are recovered outside the joint. For this, an open approach should be used, increasing surgical time and potential complications. Tears of the posterior and middle thirds of the meniscus are suitable for this technique [2].

Outside-in techniques are more suitable for repair of the anterior and middle thirds of the meniscus where a perpendicular trajectory related to the tear can be achieved. The suture is introduced from outside the capsule through the joint, engaging the two fragments of the meniscal tear and again through to the outside of the capsule. Knots are tied on the external surface of the capsule [2].

With the above techniques, the structures at risk depend on location of the meniscal tear; lateral meniscal repairs risk lateral genicular artery and branches of peroneal nerve. With medial meniscal repairs, the saphenous vein and nerve are at risk.

All-Inside Meniscal Repair

All-inside repairs were first introduced in 1991 and since grew in popularity, benefited from improvements in device and technique. An intact meniscal rim is required as an anchor for repair devices; therefore, meniscocapsular separations are preferentially repaired with an alternate technique. Anterior horn tears (Fig. 22.5) are also a relative contraindication due to difficulty in access and may be better managed with outside-in suture techniques [13].

The early generations consisting of a rigid device while now the more recent fourth-generation devices are suture based, flexible, and lower profile, and they allow for variable compression and re-tensioning across the meniscal tear.

They consist of an anchor component and a sliding knot, which allows compression of the torn meniscal segments together.

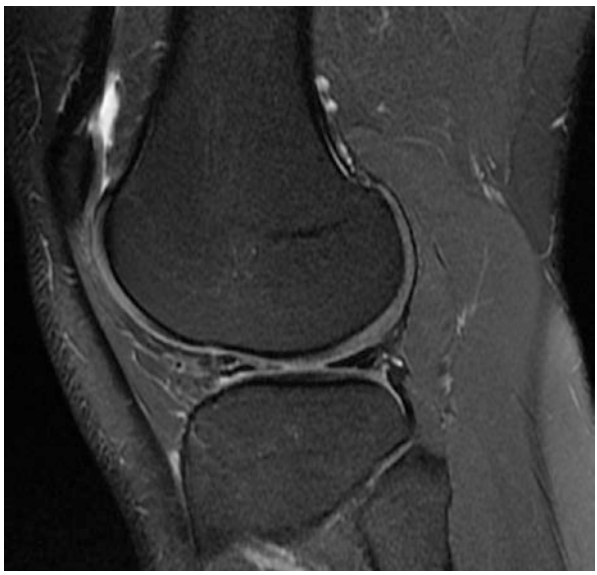

Fig. 22.5 MR image of an anterior horn lateral meniscal tear

The suture fixator is introduced from inside the joint through the capsular fragment of the tear until it rests over the capsule. The second implant is introduced through the central fragment of the tear and the capsule. It also rests over the external wall of the capsule, but the knot is tied from inside the joint. Its main advantages are that no open approaches are needed and that tears on the posterior horn are easier to repair, because the suture is perpendicular to the tear [14].

A recent systematic review conducted by Grant et al. showed there were no differences in rate of failure when comparing inside-out techniques (17%) with all-inside techniques (19%) for repair of isolated meniscal injuries [15].

Adjuncts to Meniscal Repair

Regardless of the technique used for meniscal repair, there are several adjuncts that may be used to enhance meniscal healing:

Meniscal stimulation: Meniscal rasping or abrasion is used to stimulate enhanced blood supply as well as releasing growth factors generating a healing response. In addition, neovascularization has been observed. Less commonly, trephination may be used which aims to allow communication between the vascular peripheral third and avascular zone.

Bone marrow stimulation: This is designed to recreate the environment of an ACL reconstruction to enhance healing of isolated meniscal tears. While in ACL reconstruction drilling tunnels release growth factors, blood, and platelets from the bone, perforations or microfractures at the intercondylar notch are used to try to emulate the same effect [2].

Fibrin clot: Enhances the local healing environment by placing factors found in the peripheral blood, such as growth factors, fibrin, and platelets, at the site of repair.

Stem cell therapy: Used to enhance the ability of healing and regeneration of the meniscus. However, more studies are needed to provide enough evidence to support the routine use of stem cells in meniscal pathology.

Meniscal Reconstruction

The main aims of meniscal reconstruction procedures are to place safely a functional meniscus that is anatomically and structurally similar to the native meniscus, to restore the knee and lower extremity biomechanics, to improve joint function, to relieve pain, and eventually to prevent or delay the degenerative process in the knee joint. Meniscal reconstruction consists of two main procedures: meniscal scaffolds and meniscal allograft transplantation (MAT).

Meniscal Scaffolds

The concept of meniscal replacement by means of using a scaffold was introduced in the 1990s. Highly porous, cell-free, and biodegradable meniscal scaffolds are used to fill the defect in the previously partially resected meniscus and to develop the meniscal tissue by allowing migration and growth of vascular channels and precursor cells into the scaffold.

It necessitates that the meniscal roots and peripheral rim remain conserved.

These requirements are not needed for MAT, which enables complete replacement of the meniscus. In clinical practice, there are two main types of meniscal scaffolds: the collagen meniscus implant (CMI) and polyurethane-based scaffold. Good mid-term and long-term clinical outcomes of polyurethane-based scaffolds and CMI were recently reported. However, overall the chondroprotective effects of these implants are still controversial, and long-term higher level of evidence comparative studies are required to clarify the clinical efficacy of these implants [16].

Meniscal Allografts

Meniscus allograft transplantation (MAT) is a potential biological solution for the symptomatic, meniscus-deficient patient who has not yet developed advanced OA. The meniscal allograft is donated from a cadaver and transplanted into an injured knee. However, prior to perusing meniscal transplantation, there are important steps that need to be undertaken. Firstly, it is important to assess for malalignment. A corrective osteotomy is considered the golden standard treatment for a malaligned knee with significant meniscus loss. For instance, a lateral meniscus transplantation should not be performed in a valgus-aligned lower limb. Secondly, ligamentous injuries ex ACL tear should be addressed surgically prior to or in combination with meniscal transplant. Ligament reconstruction will protect MAT, as much as MAT will protect the ACL reconstruction. The "ideal candidate" for MAT is a young patient with a history of symptomatic femorotibial compartment symptoms having undergone a previous meniscectomy, with a stable knee, neutral alignment, and no severe chondral damage or arthritis [16].

The most appropriate technique to preserve menisci allografts in an acceptable condition until transplantation is still a matter of debate. There are four types of meniscal allograft preparations: fresh, deep frozen, cryopreserved, and freeze dried.

Overall, MAT has proven its effectiveness in reducing pain and improving function and quality of life when proper indications are followed. However, there are still some questions to address regarding the integration and longevity of the graft, the efficacy of MAT in prevention of osteoarthritis, and the possibility of returning to high-demand activities.

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Collateral Ligament Injuries in the Knee

23

Jaison Patel and Wasim S. Khan

The collateral ligaments of the knee in simple terms are the primary restraints to valgus and varus stress in the knee. The recognition and appropriate treatment of these injuries is important in avoiding long-term instability of the knee. The medial collateral ligament (MCL) and the lateral collateral ligament (LCL) are the primary structures responsible for valgus (MCL) and varus (LCL) stability. However it is best to think of these structures as being a part of a more complex anatomical arrangement involving a combination of structures.

In this chapter, I will discuss the anatomy, biomechanics, clinical assessment, and treatment options for these complex ligamentous injuries in a native knee. Each patient is individual as is their knee injury. With a good understanding of the anatomy and biomechanics of the knee, you will be able to manage these complex injuries better. It is important to recognize that insufficiency of collateral ligaments in a post-TKR knee due to iatrogenic injuries or from progressive degenerative deformity can also occur, but will not be discussed here.

The Medial Side of the Knee

Anatomy and Biomechanics

The medial side of the knee serves to provide static stabilization of the knee in translation, valgus stress, and rotation. Medial collateral ligament (MCL) injuries are the most common ligamentous injuries to the knee accounting for up to

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	Anterior	Middle	Posterior
Superficial layer	Fascia	Fascia	Fascia
Middle layer	No distinct structures	sMCL	Posteromedial capsule
Deep layer		dMCL	

Fig. 23.1 A table adapted by Robinson et al. simplifying the structures of the medial knee [5]

40% of all knee injuries [1]. MCL injuries commonly occur in athletes from traumatic events or due to sudden abnormal movement in the knee, typically involving a valgus force. The MCL is part of the capsuloligamentous complex of the medial side of the knee and is often described as having three layers; the superficial, middle, and deep. The superficial is made up of the sartorius and investing fascia; the middle of semimembranosus, superficial medial collateral ligament (sMCL), and medial patellofemoral ligament (MPFL); and the deep compromising of deep medial collateral ligament (dMCL), posterior oblique ligament (POL), and meniscotibial ligament.

Figure 23.1 is a simplified summation of this by Robinson et al. [5]. The three main structures involved in providing stability to the medial side of the knee are the sMCL, POL, and dMCL [2, 3]. The POL is not thought to be a discrete ligament by some as anatomical studies have described this structure to be a capsular condensation running posteromedially from the femur to tibia [4]

Superficial Medial Collateral Ligament (sMCL)

The sMCL has a femoral attachment that is located just posterior (4.8 mm) and proximal (3.2 mm) to the prominence of the adductor tubercle and attaches to the tibia approximately 46–60 mm distal to the joint line at the level and just posterior to the pes anserinus insertion [2, 6]. The attachment of the ligament at the femur is oval in shape, and the ligament is more rounded. At its distal insertion, the attachment is broader. At the tibial attachment, the ligament fibers anteriorly are parallel and posteriorly are more oblique and reinforce the posterior capsule. The length of this ligament is 10–12 cm. The sMCL is the primary constraint to valgus stress to the knee. sMCL has a proximal and distal divisions that have differing roles in providing stability as described by Griffith et al. [7]. The proximal component serves as a static stabilizer against valgus at all degrees of flexion up to 90°. It is also a secondary external rotational stabilizer at 90° flexion and internal rotation at all degrees of flexion [7]. The distal component is a primary stabilizer to external rotation at 30° of knee flexion [7]. Numerous studies have demonstrated that repair/reconstruction of both the proximal and distal components of the sMCL are needed to restore primary valgus and rotational stability of the medial knee [7, 8].

Deep Medial Collateral Ligament (dMCL)

The dMCL is also known as the mid-third capsular ligament and is a secondary stabilizer to anterior translation of the knee and contributes to a lesser extent to valgus stability. The dMCL can be thought of as a thickening of the joint capsule and is made up of distinct meniscotibial and meniscofemoral ligaments. The dMCL meniscotibial portion provides secondary valgus stability at 60° and secondary internal rotation at all levels of flexion. The meniscofemoral portion of the dMCL provides secondary valgus stability at all levels of knee flexion and internal rotation at 0° and 30° of knee flexion. This is similar to the meniscotibial portion. The meniscofemoral ligament however also provides primary stability of internal rotation at 20°, 60°, and 90° of flexion and secondary external rotation at 30° and 90° of knee flexion. Biomechanical studies have shown that the dMCL has the lowest load to failure rate of the three important medial structures and suggests that it plays only a small role in medial-sided stability [8].

The Posterior Oblique Ligament (POL)

The POL has many differing descriptions. Laprade described it on anatomical studies to have three arms to the structure: superficial, central (tibia), and capsular. It is thought that the central arm is most crucial as it is the largest in size. The POL originated off the distal insertion of semimembranosus. POL is a primary restraint to internal rotation and is a secondary restraint to valgus and external rotation [9]. The restraint to internal rotation occurs at all degrees of flexion but is at the greatest load at full extension [10]. The POL works with the sMCL in sharing the load to anterior and posterior translation of the knee. The POL also provides a secondary restraint to abnormal valgus force in knees with an isolated MCL injury [12]. Combined sMCL and POL/PMC injuries have shown to cause the greater valgus instability when compared to isolated sMCL injuries. This shows the importance of this structure and therefore suggests that reconstruction of this at full extension (the knee position at which the POL is under the greatest load) can help restore stability near to that of a native knee [9, 11].

Injuries, Clinical Assessment, and Classification

Injuries to the medial side of the knee usually occur due to a valgus stress being applied to a stationary knee and are therefore common in those who participate in sport.

Clinical assessment should begin with basic tests of the knee and include an accurate assessment and documentation of the neurovascular status. Palpation along the course of the MCL and noting points of tenderness (femoral or tibial). Based on the information above, we know that we should examine for valgus instability at 0° and 30° of flexion to decide on whether the POL is involved. An anteromedial drawer test can be performed (anterior drawer with the foot externally rotated). It is important to examine the other ligamentous structures as well, as medial-sided knee injuries can be a part of a more complex multiligament injury.

Grade 1	Few fibers disrupted Associated pain	No laxity
Grade 2	More fibers disrupted With more generalized	No laxity
	pain	
Grade 3	Complete disruption	Laxity +1 3-5mm +2 6-10mm +3 more than 10mm

Fig. 23.2 Classification of injuries and laxity [12]

In 1976 with further clarification in 1994, Hughston classified injuries of the MCL [12]. Apart from grading the degree of laxity, no other classification system has been proposed and been subsequently validated (Fig. 23.2).

The MCL has a high healing capacity owing to its extra capsular location. Grade 1 injuries are almost exclusively treated nonoperatively.

MCL injuries are usually associated with an anterior cruciate ligament tear. Studies have shown that ACL repair without concurrent repair of grade 2 and 3 MCL injuries leaves patients with residual valgus laxity, but others have shown that a grade 3 injury treated nonoperatively does not show any laxity. This shows that there is still some controversy as to which patients would benefit from repair/reconstruction. In the presence of instability related to MCL injuries, a recent systematic review has shown improved outcomes after reconstruction of the ligament in both isolated and multiligament injuries [13].

Repair of the medial collateral ligament in the acute setting is possible. This is more likely to be considered in the setting of an avulsion-type injury from the distal femur or distal tibia with retraction of the fragment under pes anserinus the so-called “Stener”-type lesion.

Most surgical treatment however is in reconstruction of the MCL. Unless there is a clear indication for an urgent reconstruction or repair, such as in open injuries and multiligament or other intra-articular derangement, most patients are treated due to residual laxity from a period of nonoperative treatment. Grade 3 injuries, if isolated, have shown to have good results if treated nonoperatively. However poorer outcomes have been described in the presence of concomitant ligament injuries, the ACL being the most commonly associated ligament injury.

Various reconstruction techniques have been described and continue to be described every year. Patients can undergo reconstruction of the sMCL using either allograft or autograft. No evidence so far has suggested either to be superior. Both anatomic and nonanatomic methods have been described. Before considering ligament reconstruction, patients with constitutional valgus should have this boney abnormality corrected first.

Bosworth/modified Bosworth is a technique that only reconstructs the sMCL. The semitendinosus is used in the reconstruction by harvesting it but leaving it attached to the pes anserinus. The proximal tendon is then wrapped around a post (screw and washer) at an isometric point on the medial femoral condyle. The tendon is then either stitched to itself distally or to the capsule [14].

The Lind reconstruction is similar to the Bosworth technique, but attempts to reconstruct the POL as well. The semitendinosus is harvested and left attached distally on pes anserinus. The tendon in this case is fixed to the femur using an interference screw, and the free end is passed through a tibial tunnel from posterior to anterior to recreate the POL. The tibial tunnel is created by drilling anterior to posterior and is approximately 10 mm below the joint line. The sMCL limb is tension with the knee at 10° flexion and neutral rotation and the POL limb tension at 60° flexion and neutral rotation [15].

Laprade describes an anatomical reconstruction. He recreates the sMCL and POL using two separate grafts. To perform this reconstruction, the tibial insertion and subsequent tunnel of the sMCL is identified. This is usually just posterior to the pes anserinus and 6 cm from the joint line. The POL tunnel is made from a point just anterior to semimembranosus toward Gerdy's tubercle. The femoral attachments for the sMCL and POL are then identified around the medial femoral condyle and tunnels drilled. The grafts are fixed into the femoral tunnels. They pass under the sartorius and are fixed to the tibial tunnels. Laprade tensioned and fixed the POL in full extension and neutral rotation and the sMCL at 20° flexion with a slight varus force being applied [16].

The Lateral Side of the Knee

Anatomy and Biomechanics

The lateral side of the knee is inherently unstable due to the convexity of the lateral tibial plateau and femoral condyle and the mobile lateral meniscus. The posterolateral corner (PLC) of the knee is a complex arrangement of ligaments that have varying function. PLC injuries can occur as an isolated injury, but more commonly present in patients with multiligamentous knee injuries. They can occur as a result of knee dislocations. Understanding the anatomy and biomechanics of the posterolateral corner is crucial in being able to diagnose the injury and its subsequent management. As with the medial side of the knee, a three-layered structure has been proposed and is commonly used to describe the anatomy [17].

Superficial Layer

The iliotibial band is a superficial structure that inserts onto Gerdy's tubercle. Tenodesis of this is commonly used in reconstruction of the knee; in particular the Lemaire procedure or variations in this are commonly used to supplement ACL reconstructions in those patients with additional anterolateral rotatory instability.

Through its course distally, the ITB is attached to the lateral femoral condyle region via Kaplan fibers and distally attaches to the patella and patella tendon anteriorly. This contributes to lateral-sided stability.

Middle Layer

Vastus lateralis, deep ITB fibers, patellofemoral and patellomeniscal ligaments.

Deep Layer

Lateral collateral ligament (LCL) or fibula collateral ligament (FCL), fabellofibular ligament, arcuate ligament, popliteus muscle-tendon complex (PMTC), and the popliteofibular ligament (PFL) are found in this layer. The arcuate ligament is a thickening of the capsule that travels over the popliteus ligament attaching to the posterior fibula. The fabellofibular ligament if present is a ligament that runs from the fibula head to the sesamoid bone that is the fabella.

The LCL, popliteus, and PFL are the main structures involved in providing stability.

The Lateral Collateral Ligament (LCL)

The LCL origin is located posterior to the lateral femoral condyle, just proximal to the groove for the popliteus. Distally it attaches to the anterolateral portion of the fibula head approximately 28 mm distally [18]. The LCL is the primary restraint to varus stress to the knee, especially in the low degrees of flexion. It is the tightest at 30° of flexion. It is a secondary restraint to varus stress beyond 30° of flexion. The LCL also provides primary restraint to external rotation in extension [19, 20].

Popliteus Muscle-Tendon Complex (PMTC)

Popliteus tendon is located in the popliteus sulcus at the distal femur and travels distally and posteriorly lateral to the lateral meniscus where its muscle belly is located posteriorly at the proximal tibia. As it passed the lateral meniscus, fibers attach to it providing the meniscus with some stability. It is these fibers to where the PFL attaches. The broad attachment of the popliteus tendon to the femur has been consistently shown to be 18 mm anterior to the LCL insertion [18]. This is an important factor when considering how you plan your reconstruction. The PMTC is a dynamic and static stabilizer of the knee. The PMTC unlocks the knee from full extension by internally rotating the tibia around the femur. It prevents posterior translation of the tibia on the femur along with external rotation from low degrees of flexion to 90° of flexion [21].

Popliteofibular Ligament

The PFL attaches to the styloid of the fibula head and lies deep to the arcuate ligament. It is present in over 94% of knees. Unlike the LCL, the PFL and popliteus has been shown to be isometric throughout the range of flexion and provides external rotation constraint at all points, but less so at extension, but this is where the LCL will take over this role.

Injuries, Clinical Assessment, and Classification

Posterolateral corner injuries can occur as a result of direct injury, varus strain to a flexed knee, external rotation injuries, and knee dislocations. Isolated injuries occur in an approximately a quarter of cases, with the majority of PLC injuries being a part of combined injuries with PCL or ACL.

No direct classification system has been proposed, much like the medial side, and often the modified Hughston classification has been used to classify the severity of the injury.

Without causing too much repetition, the initial basic assessment of the knee should be the same as any other acute injury. It is important to note that injuries to the common peroneal nerve have been reported in up to 25% of posterolateral corner injuries [22]. Varus laxity should be tested at full extension and 30° of flexion. Full extension instability suggests LCL and cruciate ligament injury. The dial test in an isolated PLC injury will show >10° of extra external rotation at 30° only. If >10° of extra external rotation compared to the unaffected side is present at 30° and 90°, this suggests a PLC and PCL injury. Posterolateral drawer test can be performed with the knee flexed and the foot externally rotated. A posterolateral and external rotation force is applied to the tibia and is positive if there is increased translation. A reverse pivot shift can be performed but is important to assess this against the unaffected side, as laxity can be present in normal knees. The test is performed with a flexed knee, much the same as a pivot shift, but with valgus and external rotational force to subluxation of the tibia posterolaterally. The iliotibial band will reduce it as the knee extends.

Surgical Management

Like the medial side, low-grade PLC injuries can be treated nonoperatively with early mobilization, but high-grade injuries don't do as well long term with ongoing instability [23]. The early nonoperative treatment is with a brace locked in extension for 2–4 weeks and with progressive weight bearing as tolerated [24].

Surgical treatment should be considered in high-grade injuries, those with concomitant ACL/PCL injuries due to the high risk of failure if not and also those who fail nonoperative treatment.

When considering repair procedures, the same basic principles apply to the lateral side as they do with the medial side. Midsubstance tears are difficult to repair and have poor outcomes. Those with boney avulsions can do well but must be done within 2–3 weeks. After 3 weeks, the tendons retract and scar tissue forms, making it impossible to repair the ligament [25]. It is rare in reality to be presented with the option to repair a PLC, and studies have shown that delayed reconstruction has a better outcome when compared to acute repair [26].

Various anatomical and nonanatomical reconstruction techniques have been described.

The Clancy procedure is a biceps tenodesis. The procedure involves taking a strip of the biceps tendon and rerouting around a post on the lateral condyle to recreate a LCL [27].

Larson's technique involved taking a graft from the fibula head to the lateral femoral condyle. A second oblique graft is then routed from the posterior aspect to the lateral femoral condyle. The femoral tunnel is placed in an isometric not anatomical point. Variations of this technique have been described by Fanelli and Arciero, to try and make this technique more anatomical [28, 29].

Stannard produced a technique different to the above, in that it recreated the three major ligaments of the posterolateral corner (FCL, popliteus, and popliteofibular ligament). This technique was isometric and nonanatomical. Tunnels were created anterior to posterior on the tibia and anterolateral to posteromedial on the fibula head. A fixation point was then created on the lateral femoral condyle at an isometric point. A graft is then fixed into the AP tibial tunnel at one end, the tendon routed around the femoral post, back down to the fibula, passed posterior to anterior through the fibula tunnel and then back to the femoral post where it is secured.

Laprade describes an anatomical reconstruction of the posterolateral corner which is commonly used in our practice. An exposure to the lateral knee is performed using a hockey-shaped incision from lateral femoral condyle distally to a point between Gerdy's tubercle and fibula head. The superficial layer of the ITB and biceps femoris are identified. The common peroneal nerve is identified and protected throughout. The insertion of the LCL and popliteofibular ligament onto the fibula is identified along with the musculotendinous junction of the popliteus. A tunnel is created in the fibular head laterally aiming posteromedially. A tibial tunnel is then created at an area just distal to Gerdy's tubercle; this point is 1 cm proximal and medial to the fibula tunnel. The femoral attachments of the LCL and popliteus are identified by making a longitudinal incision through the anterior portion of the ITB. At the LCL insertion, a tunnel is created in the anteromedial direction, and a second tunnel at the popliteus tendon insertion is created in a similar direction. The distance between these two tunnels should be 18.5 mm as this is consistently the distance seen in anatomical studies [18]. Laprade's preference is to use a split Achilles tendon allograft with the calcaneus bone block split into two. A bone block is then placed and fixed into each femoral tunnel. One graft is passed under the ITB and through the fibula head in a posteromedial direction. This is tensioned and fixed at 20° flexion and valgus. The other tendon graft is passed through the popliteus hiatus to the posterior knee. The two free ends of the graft are then passed through a tibial tunnel posterior to anterior and fixed after cycling the knee.

Conclusion

The complex anatomy of the medial and lateral side is difficult to understand and the biomechanics complicated. Further studies into the biomechanics of the knee will further advance our knowledge of this subject. The importance of recognizing collateral injuries as not single ligament injuries but an injury of complex

arrangements of various structures is highly stressed. The aim of surgical management should be to restore normal knee biomechanics. This can only be possible if normal knee biomechanics are understood.

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Lower Limb Muscle Strains

24

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Classification of Muscle Strains

In a chapter exploring lower limb muscle strains, we should first discuss the definition and classification of the term "muscle strain" [1]. Many different classification systems of the grading of muscle strains have existed, including anatomical details, clinical signs, or imaging features of strains. Commonly, most classifications included forms of "mild," "moderate," and "severe" types of strains (Fig. 24.1).

These may either be by anatomy, radiological findings, or clinical presentation. A model incorporating all three is from the British Athletic Classification of Grading. This classification describes a classification of muscle strain/tear severity, including anatomy (involvement of muscle, musculotendinous junction, and/or tendon involvement), symptoms, and MRI findings (Fig. 24.2).

Sports Hernia

Athletic pubalgia, or "sports hernia," is a common injury that has first been described in the 1980s and is increasingly recognized as a cause of chronic groin pain in young patients [2]. Unlike the traditional sense of the term, this

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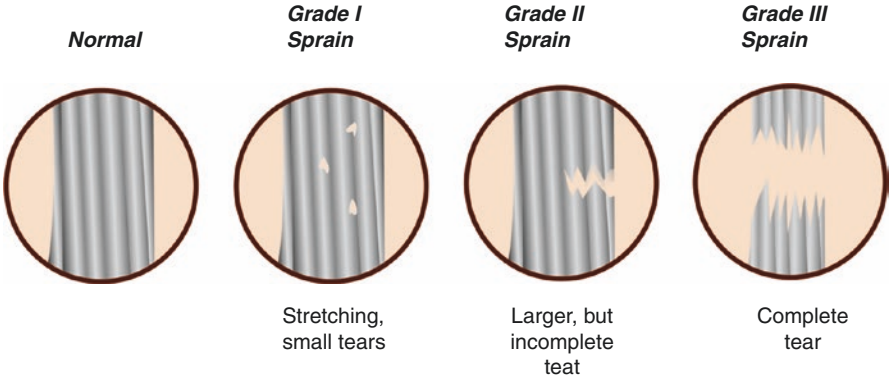

Fig. 24.1 Line diagram showing classification of muscular strains

type of "hernia" does not involve a herniation of soft tissue. Rather, it refers to an injury to the abdominal wall and/or injury to the adductor longus that consequently produces a pain in the anterior pelvis or groin pain [2]. Sports hernias are estimated to be present in 5–28% of athletes. Cases of sports hernias have primarily documented young males actively involved in sport, while young females comprise a lower percentage of patients (3–15%). These patients present with vague unilateral groin pain that is difficult to localize. The pain exacerbates on exertion, such as sprinting, twisting, sidestepping, and kicking, especially involving the combined movements of abdominal hyperextension and thigh hyperabduction. On examination, the patient is pain-free. Other sources report tenderness on palpation of the pubic ramus [3]. Imaging in forms of MRI and ultrasound can be used to exclude other causes of pain such as acetabular labral tears, iliopsoas tendonitis, or intra-abdominal sources of pain (such as appendicitis or ovarian cyst) [4].

The management of sports hernias may be nonoperative or operative. Nonoperative options include a combination of physiotherapy, nonsteroidal anti-inflammatories drugs, and rest. This trial of conservative management may last between 6 and 12 weeks [1, 2]. The operative management of sports hernias includes open or laparoscopic (preferred) repair or reinforcement of the abdominal wall and, in lesser cases, decompression of the genital branch of the genitofemoral nerve [2]. Laparoscopic repairs involve reinforcement of the abdominal wall with mesh as either a total extraperitoneal mesh repair (TEP) or a pre-peritoneal polypropylene mesh repair followed by a course of postoperative analgesia and physiotherapy [1]. Browns et al. (2013) report a postoperative return to sports rate of 96% from laparoscopic repairs, thereby making this option the preferred management among athletes while conservative management with patients who are not professional athletes [1].

Grade	Symptoms	MRI Findings
0a- Focal neuromuscular soreness 0b- General muscle soreness	Focal muscle soreness General muscle soreness	Nil Nil to high signal
1a–Extending from fascia, <10% cross-sectional area	No frank fiber disruption	Hematoma
1b–Muscle involvement, <10% cross-sectional area	No frank fiber disruption	Hematoma
2a–Extends from fascia, 10-50% cross sectional area	Less strength reduction	Periphery high signal
2b– Muscle involvement, 10–50% cross sectional area	Strength reduction	High signal at musculotendinous junction
2c–Tendon involvement, <50% cross-sectional area	Loss of tendon tension	High signal at tendon
3a–Extends from fascia, >50% cross sectional area	Sudden onset, fall to ground	Periphery high signal
3b–Muscle or musculotendinous involvement, >50% cross sectional area	Sudden onset, fall to ground	High signal at musculotendinous junction
3c–Tendon involvement, >50% cross sectional area	Sudden onset, fall to ground	High signal at tendon
4a–Complete tear extending from fascia	Sudden, fall to ground, palpable gap	Periphery defect
4b–Complete tear involving muscle or musculotendinous junction involvement	Sudden, fall to ground, palpable gap	Defect at musculotendinous junction
4c–Complete tear with tendon involvement	Sudden, fall to ground, palpable gap	Defect at tendon

Fig. 24.2 Table as advocated by the British Athletic Association for Muscular Strains

Hamstring Strain

Typically referred to as a "pulled hamstring," hamstring strains are the most common injury in sports and most common in recurrence rates citing 12–48% in professional football [1]. The injury involves a sudden stretch of one of the hamstrings, involving musculotendinous junction during a sprint. The pain presents as nonspecific and general pain in the posterior compartment of the thigh. Hamstring injuries also differ in rehabilitation. For example, biceps femoris strains (long head) are strains from high-speed running. In contrast, the semimembranosus is involved in extensive lengthening of the hamstrings. These movements are involved in high kicking and slide tackling and involve the proximal tendon of the semimembranosus [2]. Risk factors for hamstring injuries include lack of muscle flexibility, insufficient warm-up exercise, fatigue, previous hamstring injury, and increasing age [3, 5].

Clinically, patients with these injuries are examined and imaged with findings of strain, tears, contusions, and inflammation appearing on MRI and ultrasound. Management of these injuries is mainly supportive, and rehabilitation has been found effective, particularly in the form of eccentric strengthening exercises [3, 6].

The role of platelet-rich plasma continues to draw contradicting evidence with double-blind trials demonstrating finds of no effect of PRP in outcome measures, versus other studies demonstrating lower pain severity scores in patients with hamstring injuries managed with rehabilitation and PRP injections versus rehabilitation alone [3, 4, 7, 8].

Quadriceps Strain

Quadriceps strain can most commonly occur in competitive sports such as football and rugby, where eccentric contraction of the muscles is required for knee flexion and hip extension. This injury occurs when higher forces across the musculotendinous junction are applied with eccentric contraction. The most common quadriceps injury is of the rectus femoris [3]. Patients present with anterior thigh pain that is sharp in nature, and this is followed with loss of function of the quadriceps. Classically, this is described along the distal portion of the rectus femoris at the musculotendinous junction. This pain may be associated with inflammation, swelling, and bruising 24 h post injury. On examination, there is pain on resisted knee extension and hip flexion. Imaging in the forms of MRI or ultrasound may provide further evidence to support the history and examination; however, the diagnosis of quadriceps injury is primarily a clinical diagnosis.

In addition to quadriceps strain, quadriceps contusions may occur as a result of muscular strain. These contusions are areas of localized hematomas that can cause direct rupture to the muscle fibers adjacent to the area of impact.

Management of quadriceps strain is in keeping with rehabilitation – with particular focus on physiotherapy, ice, NSAIDs, and strengthening. Knee flexion for the first 24 h will aid in limiting hematoma formation in the knee, and this may be done

in a hinged knee brace set to 120°. Return to sports may only occur if the patient is pain-free, normal knee range of movement, and demonstrates near to normal strength as to that of the contralateral side [3, 9]. *Myositis ossificans*, a nonneoplastic proliferation of bone, may become a complication from quadriceps contusion, with an incidence of 9–17% post contusion [3, 9]. The diagnosis of myositis ossificans is aided from radiographs that demonstrate new bone formation in the area of contusion [3].

"Calf Strains"

Much like the aforementioned strains, the gastrocnemius strain is common and has its own common lay term of "calf strains," and much like "quads," and "hamstrings," the calf, or triceps surae, refers to a group of muscles. This group of muscles includes the gastrocnemius, the soleus, and the plantaris. The insertion site of all three muscles is the Achilles tendon.

Gastrocnemius Strain

Gastrocnemius injuries, or "tennis leg," are most commonly isolated to the medial gastrocnemius head. The presentation of this type of injury is sudden extension with a dorsiflexed foot, resulting in pain, swelling, and inability to weight bear. As the gastrocnemius involves two joints and possesses a high number of fast-twitch muscle fibers, it is more likely to injure and strain [3].

On examination, gastrocnemius strains present with tenderness on palpation of the medial muscle belly and musculotendinous junction. Pain on maximal extension from a flexed knee while the ankle is dorsiflexed is localized to the gastrocnemius. It is also important to note that gastrocnemius and soleus tears may occur together, with a prevalence of both tears found to be in 17% of calf strains [3, 10, 11].

Soleus Strains

Soleus injuries can be differentiated from gastrocnemius injuries clinically by tenderness on palpation of the lateral aspect of the calf. If there is pain in the calf while the knee is in full flexion, then the pain is of origin of the soleus [11].

Plantaris Strain

The plantaris muscle is a vestigial muscle and is rarely involved in calf strains. Isolated plantaris calf strains are difficult to distinguish without imaging [3, 11, 12].

To investigate and aid in the diagnosis of injuries to the gastrocnemius, soleus, or plantaris injuries through imaging, MRI and US are the modalities of choice.

Management of gastrocnemius, soleus, and plantaris injuries will all involve management of the hematoma that initially presents immediately after the injury. The first 3–5 days are managed with supportive treatment using ice and compressive wrap or tape. Rehabilitation follows the acute stage, and this requires exercises that isolate the soleus and gastrocnemius by varying the degree of knee flexion and allow the muscle to strengthen. Surgical intervention may be considered for a grade III or grade IV strain of a medial gastrocnemius strain [3, 11].

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The Leg

25

K. Mohan Iyer

Congenital Pseudoarthrosis of the Tibia (Osteogenesis Imperfecta)

Pseudoarthrosis is a false joint associated with abnormal movements at the site.

Congenital pseudoarthrosis of the tibia (CPT) refers to nonunion of a tibial fracture which develops spontaneously or after trivial trauma in a dysplastic bone segment of the tibial diaphysis.

Three types of tibial bowing exist in children:

1. Anterolateral bowing (Fig. 25.1) (this topic)
2. Posteromedial bowing (physiologic)
3. Anteromedial bowing (fibular hemimelia)

CPT is rare and usually develops in the first 2 years of life. Its aetiology is not clear. The reported incidence of congenital pseudoarthrosis of the tibia (CPT) varies between 1:140,000 and 1:250,000, and bilateral forms are extremely rare.

There is a strong association of neurofibromatosis type 1 with CPT. Six percent of the patients with neurofibromatosis have the deformity, and nearly 55% of the cases with anterolateral bowing and pseudoarthrosis are associated with neurofibromatosis. The presence of neurofibromatosis does not affect the outcome of tibial pseudoarthrosis.

Fibrous dysplasia is seen in about 15% of cases with anterolateral bowing.

Tibial bowing is often obvious and is present at birth. The foot is usually dorsiflexed to such a degree that it makes contact with the anterior aspect of the distal tibia. The posterior bow of the tibia is less obvious but can be easily palpated. A dimple may be present in the skin posterior to the apex of the bow.

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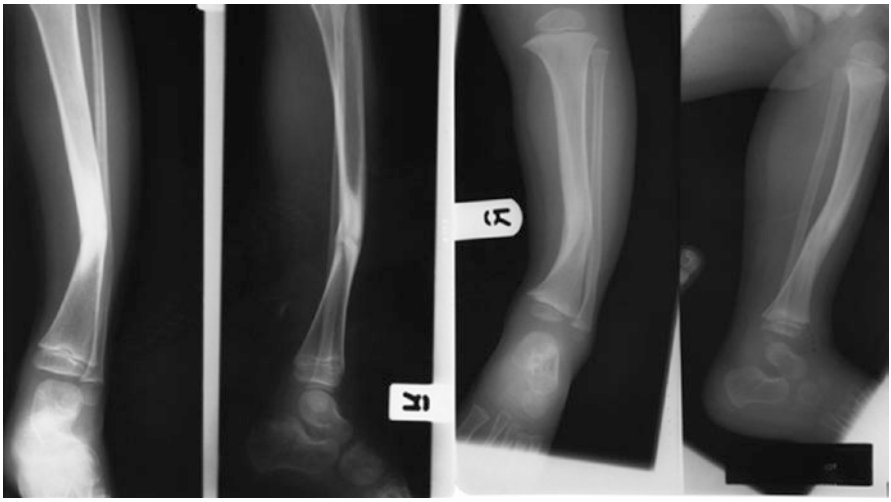


Fig. 25.1 Congenital pseudoarthrosis of the tibia (Courtesy: reused with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

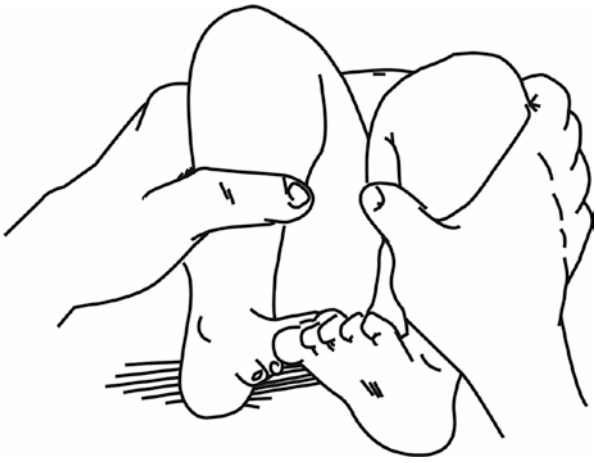


Fig. 25.2 Line diagram to show posteromedial tibial bowing. The Galeazzi test. Note the difference in the height of the flexed knees

The Galeazzi test (Fig. 25.2), typically used to assess hip dislocation, can also be used to assess any congenital disorder that results in a significant limb-length inequality. The examination is performed with the patient supine and the hips and knees flexed. The result is considered positive if knee height is asymmetrical. It is also helpful to assess whether the limb-length inequality is primarily from the femur or from the tibia and to assess limb length in someone with knee or hip flexion contractures.

Differential diagnoses include the other types of tibial bowing, such as anterolateral and anteromedial bowing. Intrauterine fracture or osteogenesis imperfecta may also result in tibial bowing [1, 2]. These are often easily differentiated on the basis of physical examination findings in which the direction of the tibial bowing and the associated foot deformity are noted.

Anteromedial bowing is often associated with congenital loss of the lateral rays of the foot and fibular deficiency. Anterolateral bowing is associated with a pseudoarthrosis of the tibia that may be obvious radiographically at birth or may develop with growth. Approximately 50% of children with anterolateral bowing are eventually diagnosed with neurofibromatosis.

The main problems are as follows:

1. Primary:
   (a) Obtaining union.
   (b) Shortening of the limb which may be (1) present at birth, (2) pseudoarthrosis remains ununited, or (3) repeated unsuccessful operations.
   (c) The tendency to refracture diminishes with skeletal maturity.
2. Secondary:
   (a) Abnormal inclination of the proximal tibial epiphyses.
   (b) Posterior bowing of the proximal tibial epiphyses.
   (c) Proximal migration of the lateral malleolus.
   (d) Fibular hypoplasia.
   (e) Fibular pseudoarthrosis.
   (f) Ankle valgus and calcaneus deformity.

Neurofibromatosis

NF-1 occurs due to mutation of the gene coding for NEUROFIBROMIN on chromosome 17. Neurofibromin is expressed in a broad range of cells and tissue type. It negatively regulates Ras activity (cell function and proliferation). Its deficiency leads to increased Ras activity. It affects Ras-dependent MAPK (mitogen-activated protein kinase) activity which is essential for osteoclast function and survival.

Diagnostic criteria for neurofibromatosis (Fig. 25.3):

1. Six or more café au lait macules (>5 mm before puberty and > 15 mm after puberty).
2. Axillary or inguinal freckling.
3. Two or more neurofibromas or one plexiform neurofibroma.
4. Two or more Lisch nodules.
5. Optic glioma.
6. A distinctive osseous lesion such as sphenoid dysplasia or thinning of long bone cortex with or without pseudoarthrosis.
7. A first-degree relative with NF-1.

Pathology

The exact pathology is still not clear. Recent studies have shown that there is hyperplasia of fibroblast along with the formation of dense fibrous tissue. This invasive fibromatosis is located within the periosteum and broken bone ends causing compression, osteolysis, and persistence of pseudoarthrosis.

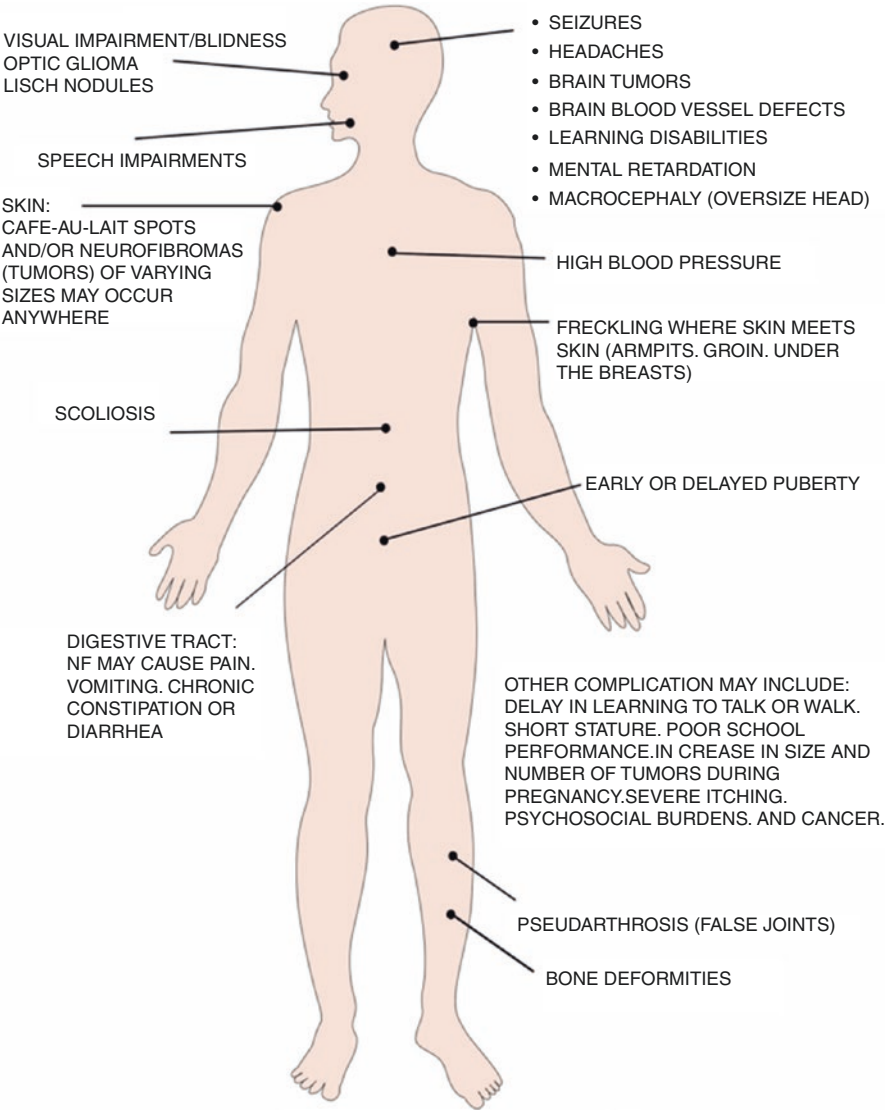


Fig. 25.3 Line diagram showing the signs of neurofibromatosis

Paley et al. [1] theorized that the pathology of pseudoarthrosis is not bony but is periosteal in origin. This theory was reconsidered by Codavilla [2] a century ago, which is supported by the following observations:

1. Thickening with hamartomatous transformation of the periosteum.
2. Appearance of strangulation of bone with atrophic changes followed by avascular changes.
3. Failure of Remodeling of Pin Tracts Leading to Stress Fractures

Histopathology

1. Fibrous hamartoma is the key pathology.
2. A soft tissue at the pseudoarthrotic site which is composed of a variable mixture of fibrous tissue, fibrocartilage, and hyaline cartilage with evidence of endochondral ossification.
3. The marrow spaces are devoid of ossification.
4. The invasive fibromatosis is located in the periosteum and between broken bone ends and surrounds the tibia causing compression, osteolysis, and persistence of pseudoarthrosis.

Classification

There is no universally agreed classification based on clinical and radiological findings, but the classification by Boyd (1982) [3] is the commonly used one.

This classification is divided into six types:

1. Type 1: It has the following features: (a) pseudoarthrosis with anterior bowing, (b) a defect in the tibia which is present at birth, and (c) other congenital anomalies may be present which may affect the management of pseudoarthrosis.
2. Type 2: It has the following features: (a) pseudoarthrosis with anterior bowing with an hourglass constriction of the tibia which is present at birth; (b) spontaneous fractures after minor trauma; (c) commonly seen before 2 years of age; (d) also known as high-risk tibia; (e) tibia is tapered, round, sclerotic with obliteration of the medullary canal; (f) it is the most common type; (g) associated with NF-1; and (h) it has the poorest prognosis.
3. Type 3: It has the following three features: (a) pseudoarthrosis which develops in a congenital cyst usually near the junction of the middle and distal third of the tibia, (b) anterior bowing may precede or follow the development of a fracture, and (c) recurrence of fracture is less common after treatment.
4. Type 4: It has the following features: (a) originates in a sclerotic segment of the bone, (b) it is without narrowing of the tibia, (c) the medullary canal is partially or completely obliterated, (d) an insufficiency or stress fracture develops in the cortex of the tibia and gradually extends through the sclerotic bone, and (e) the prognosis in this type is good.
5. Type 5: It has the following four features: (a) The pseudoarthrosis in the tibia develops with a dysplastic fibula. (b) Pseudoarthrosis of both the bones may develop. (c) The prognosis is good if the lesion is only confined to the fibula. (d) If the lesion progresses to the tibia, then the natural history usually resembles type 2.
6. Type 6: It mainly occurs as an interosseous neurofibroma or schwannoma, and this variety is extremely rare.

Anderson Classification [4]

It is divided into six types as follows:

1. Club foot.
2. Cystic.

3. Late.
4. Fibular.
5. Dysplastic.
6. Angulated.

Crawford Classification

This is broadly divided into two types, namely:

1. Non-dysplastic type: There the anterolateral bowing is with increased sclerosis and density of the medullary canal
2. Dysplastic type: This is characterized by three features, namely, (a) anterolateral bowing with failure of tubularization, (b) cystic changes, and (c) frank pseudoarthrosis (Figs. 25.4 and 25.5).

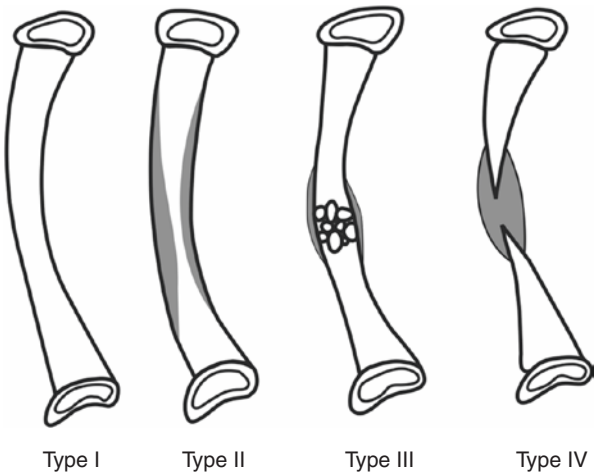


Fig. 25.4 Line diagram showing the types

Table 1 El-Rosasy-paley Classification for CPT

Type I	Atrophic bone ends (based on radiographic examination) Mobile pseudarthrosis No previous surgical intervention
Type II	Atrophic bone ends (based on radiographic examination) Mobile pseudarthrosis
Type III	Previous surgical intervention with or without retained hardware Borad bone :ends (based on radiographic examination) Stiff pseudarthrosis With or Without previous surgical intervention

Fig. 25.5 Line diagram showing the El-Rosasy–Paley classification [5]

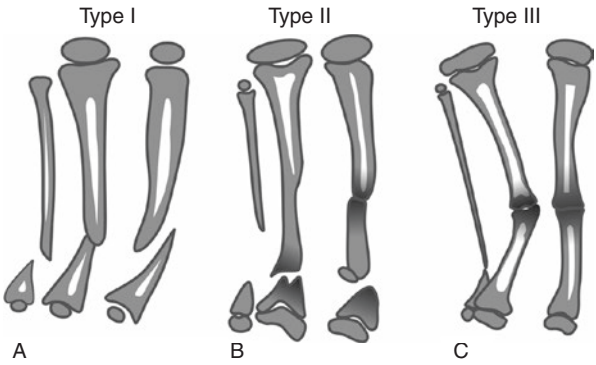


Fig. 25.6 Line diagram showing types of the El-Rosasy–Paley classification [5]

Classification by Paley (Figs. 25.5 and 25.6)

Clinical Features

1. Always unilateral.
2. Associated with anterolateral bowing of the tibia.
3. Bowing usually occurs between the middle and distal third.
4. The deformity may be associated with skin dimple, limb shortening, dysplasia of the fibula, and ankle valgus.
5. If cutaneous signs of neurofibromatosis are present, the diagnosis is readily apparent

Imaging

1. An MRI (magnetic resonance imaging) shows the following:
   (a) The extent of the disease.
   (b) Preoperative planning so that the borders for resection can be defined more accurately.
   (c) The area of pseudoarthrosis is hyperintense on fat-suppressed and T2-weighted images and slightly hypointense on T1-weighted images with contrast enhancement after administration of gadolinium.
2. Computed tomography scan confirms these findings.
3. Total bone scintigraphy shows the level of pseudoarthrosis.

PS: Regardless of the treatment method, there is general pessimism regarding the quality and longevity of the union attained.

The aims of treatment may be summarized as follows:

1. Achieve union.
2. Prevent refracture.
3. Correct limb-length inequality.
4. Correct associated growth abnormalities.
5. Prevent ankle deformity and arthritis.

1. Achieving union:
   (a) Microvascular free fibular transfer.
   (b) The Ilizarov technique.
   (c) Bone grafting with intramedullary nailing.

   Of all these things, the most important aspect is excision of the pseudoarthrosis.
2. Strategies to minimize the risk of refracture:
   (a) Splint the limb in an orthosis till skeletal maturity.
   (b) Retain an intramedullary nail till skeletal maturity.
3. Strategies to deal with shortening of the limb:
   (a) Minimize the extent of shortening by obtaining union of the pseudoarthrosis as early as possible.
   (b) Treatment to establish shortening by limb equalization procedures.
4. Strategies for minimizing valgus deformity of the ankle:
   (a) Make sure of union of the fibular pseudoarthrosis.
   (b) Retain the intramedullary rod which crosses the ankle joint as it can also prevent ankle deformity through the motion lost.

Prophylaxis

Once the diagnosis of a non-resolving anterolateral bowing of the tibia has been made, then the first step is to prevent refracture if possible. In an infant of the walking age, no specific treatment is needed other than education of the caretakers. Once the child begins walking, weight-bearing prophylaxis should be attempted though there is no documentation that such program can prevent a fracture. A clam shell orthosis which provides circumstantial support is usually recommended. Protection of the unfractured tibia should be continued indefinitely until skeletal maturity or till the patient reaches skeletal maturity.

Treatment

It is mainly divided into two types, namely:

1. Prophylactic: It includes (a) decreased activity, (b) cast or an orthotics, and (c) curettage and bone grafting.
2. Active: Surgical treatment/options.

Surgical Options

1. Vascularized fibular grafts.
2. External fixation.
3. Intramedullary rod.
4. BMP (bone morphogenetic protein).
5. Electrical stimulation.

Williams Technique: The procedure of choice for the first attempt to gain union entails resection of the pseudoarthrosis, shortening, and fixation with an intramedullary rod along with autogenous bone grafting. This procedure can be performed at

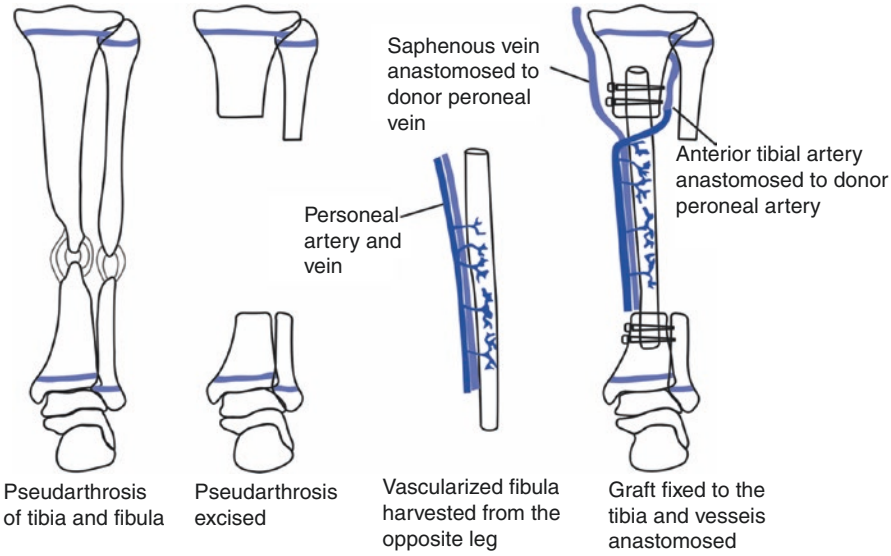


Fig. 25.7 Line diagram showing use of a vascularized fibular graft with an intramedullary rod for treatment of CPT

any age, and the rates of union have been reported as around 85% although solid long-lasting union without deformity is another matter.

Williams Technique: Williams conceived a novel technique [6] of threaded male and female components of the rod that when joined can be placed antegrade through the site of and out of the bottom of the foot. After retrograde insertion back in the proximal intramedullary in the tibia, the male end is unscrewed and removed from the bottom of the foot with the female threaded rod left intraosseously in the tibia or across the ankle/talus (Fig. 25.7).

The undesirable effect of ankle immobilization and IM fixation is thought to be a necessary evil to adequately immobilize the distal fragment. As the tibia grows, the foot and the ankle may eventually grow off the distal end of the IM rod, thereby allowing the ankle to regain motion.

The possibility of an ankle valgus is almost inevitable especially if there is a fibular pseudoarthrosis despite the achievement of solid union.

Limb-length discrepancy is yet another untoward event with shortening at maturity averaging as much as 5 cm.

Weak and stiff ankle and subtalar joint secondary to cross-ankle fixation results in a poorly functioning foot.

The need for fibular surgery still remains controversial.

Vascularized fibular graft: This procedure involves harvesting a long segment of opposite fibula along with its vascular pedicle. This is then transferred into the gap created after radical excision of the pseudoarthrotic segment. The vessels of the transferred fibula are then anastomosed with the local vessels. The transferred fibula is then fixed securely to the tibia (Fig. 25.7).

Intramedullary fixation of the donated fibula is contraindicated theoretically because of the possibility of disturbance of blood supply of the microvascular graft. Because the transfer brings tissue with its own blood supply, free fibular vascularized transfer has been recommended as the procedure of choice for gaps >3 cm after resection of the pseudoarthrosis.

The success rate for free fibular transfer is around 92–95% if union alone is considered. Refracture has been reported in a third of the patients probably a direct result of not being able to apply a permanent intramedullary fixation.

Morbidity of Donor Leg

The distal end of the fibula must be synostosed with the tibia (Langenskiold procedure), or the fibula of the donor site must be reconstructed with a bone graft to prevent ankle valgus. In addition weakness may ensue in the donor leg due to resection of origins of the flexor muscles.

Bone Morphogenetic Protein

BMP2 is extremely useful to speed up the union rates in CPT. BMP7 seems to be effective in the absence of an actively differential osteoblastic cell line.

Electrical Stimulation: Electrical stimulation does not correct existing deformities, and hence its application is probably limited to the earlier phases of pseudoarthrosis treatment when union is the primary goal.

Amputation: The final function in a patient who has undergone multiple operations but must still protect the leg with an orthosis may well be worse than if an earlier amputation and prosthetic fitting had been performed.

Complications

1. Refracture.
2. Malalignment of the tibia.
3. Limb-length discrepancy.
4. Ankle valgus.
5. Ankle stiffness.

1. Refracture: Its incidence is around 14–60%. The anatomic alignment of the tibia and fibula minimize the risk of refracture. Intramedullary rod and external bracing must be continued as external protection against refractures.
2. Malalignment of the tibia: Diaphyseal malalignment of the tibia (procurvatum or valgus deformity) are progressive and do not remodel.
3. Limb-length discrepancy: Residual length discrepancy following successful union is a major problem. Growth abnormalities of the tibia and fibula and the ipsilateral femur abnormalities are also seen with CPT which include inclination of the proximal tibial epiphyses, posterior bowing of the proximal third of the tibial diaphysis, and proximal migration of the lateral malleolus.
4. Ankle valgus: This compromises the functional outcome. A progressive ankle valgus is a problematic postoperative donor site of a vascularized fibular graft in children. Tibiofibular metaphyseal synostoses (the Langenskiold procedure).

5. Ankle stiffness: It progressively regresses once the intramedullary rod is removed from the ankle. The pain which is secondary degenerative changes at the ankle can be treated by limitation of activity and shoe modification. Any severe pain may require ankle arthrodesis.

The follow-up is till skeletal maturity to identify and treat any residual problems.

Pseudoarthrosis of the Fibula

Pseudoarthrosis of the fibula often precedes or accompanies the same condition in the ipsilateral tibia. A progressive valgus deformity develops. Several grades are seen such as:

1. Bowing of the fibula without pseudoarthrosis.
2. Pseudoarthrosis without ankle deformity.
3. With ankle deformity.
4. Fibular pseudoarthrosis with latent tibial pseudoarthrosis.

Osteochondritis of the Ankle

This clinical entity was first described in the literature by Franz König in 1888. Osteochondral lesions of the talus may be caused by trauma or repetitive microtrauma with no history of trauma. It is bilateral in 10% of cases. Most cases of osteochondritis dissecans of the talus occur around the time of skeletal maturity (10–14 years of age for girls and 12–16 years of age for boys). There is an increasing prevalence of this condition in athletic teenage girls. These ankle lesions tend to be smaller, more difficult to diagnose, and less likely to heal despite treatment efforts.

Clinical Features

Swelling with inflammation and soreness in the ankle joint. There may be a sense of locking or catching in the joint during movements. There may be a decreased range of movements and a feeling of crepitus along with weakness of the joint. There may be limping or pain and stiffness after activity.

There are various causes of this condition as follows:

1. Ischemia: The restricted blood supply is usually caused by some problem with blood vessels or vascular problems. The bone undergoes avascular necrosis, a deterioration caused by lack of blood supply. Ischemia usually occurs in conjunction with a history of trauma.

2. Genetic factors: There are chances of another family member also being involved which indicates inherited genetic susceptibility.
3. High demand impact sports or competitive sports indicate repeated stress to the bone or joint.
4. Miscellaneous factors such as weak ligaments or meniscal lesions in the knee.

Classification and Staging

There are three types of classifications as follows:

1. Berndt and Harty (1959) [7]

Berndt and Harty radiographic classification	
Stage 1	• Small area of subchondral compression
Stage 2	• Partial fragment detachment
Stage 3	• Complete fragment detachment but not displaced
Stage 4	• Displaced fragment

2. Anderson (1989) [8]

   Stage I due to injury—Subchondral trabecular compression. No visible injury seen on X-ray. Bone scan will demonstrate an injury. If so, then MRI will help define the injury, showing marrow edema.

   Stage II—Incomplete separation of the osteochondral fragment. Can readily be seen on computerized tomography (CT scan) or magnetic resonance imaging (MRI scan).

   Stage III—Unattached, but undisplaced, fragment. An attachment to the talus cannot be seen, but the fragment is not displaced. T2 image may demonstrate synovial fluid around a fragment.

   Stage IV—Displaced fragment.

   The displacement can at times.

   Kappis also described similar lesions of the talar dome in 1922 [9].
3. Hepple MRI staging system [10] (Figs. 25.8, 25.9, and 25.10)

Stage 1	Articular cartilage edema [8]
Stage 2a	Cartilage injury with underlying fracture and surrounding bony edema
Stage 2b	Stage 2a without surrounding bone edema [9]
Stage 3	Detached but nondisplaced fragment [10]
Stage 4	Displaced fragment
Stage 5	Subchondral cyst formation

Diagnosis and Investigations

A detailed history with clinical examination to include medical history, family history, and lifestyle, including sporting activities.

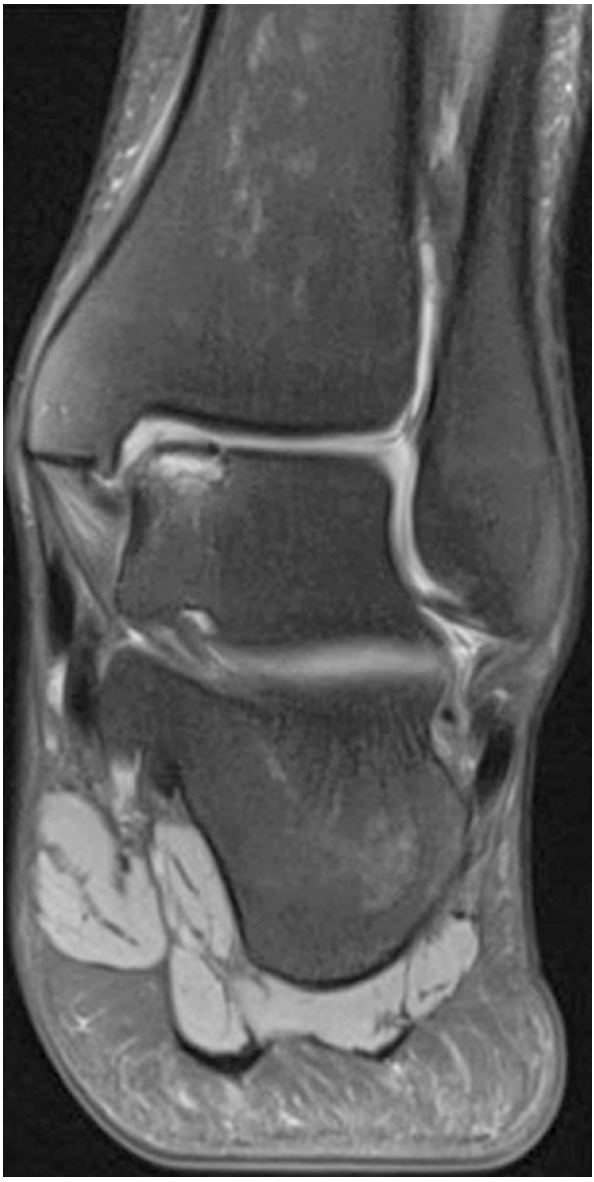


Fig. 25.8 MRI: Coronal PDFS showing osteochondral lesion of the superomedial talar dome (Courtesy: Dr. Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopaedic Hospital, Birmingham, UK)

Investigations

(a) Imaging: Plain X-rays may be normal and may show a subtle lucency or fragment of bone.
(b) CT: It helps evaluating the bony lesions seen on plain radiographs.
(c) MRI: It is indicated in ankle sprains.

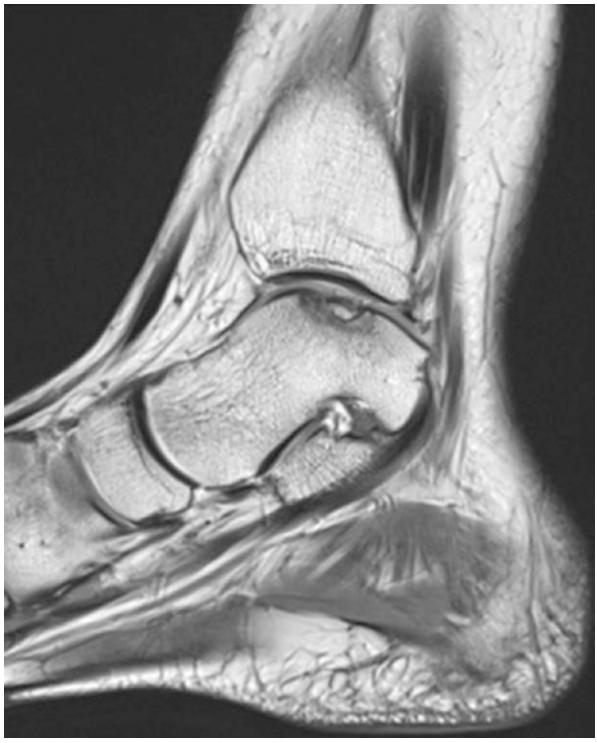


Fig. 25.9 MRI: Sag PD showing osteochondral lesion of the superomedial talar dome (Courtesy: Dr. Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopaedic Hospital, Birmingham, UK)

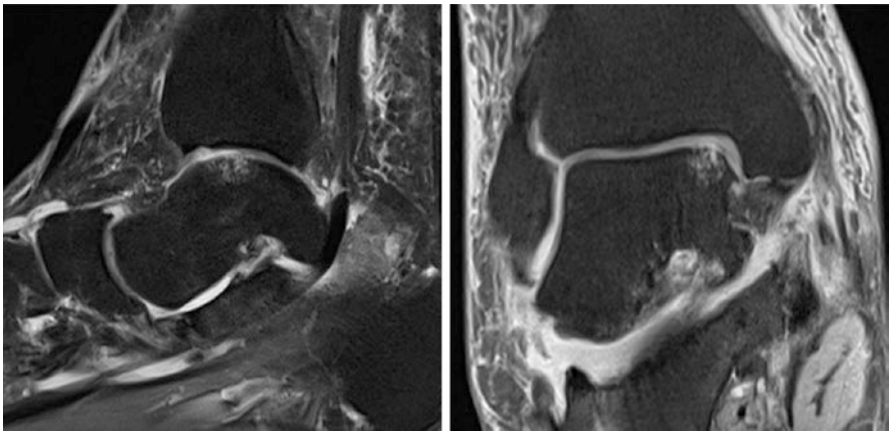


Fig. 25.10 MRI: Sag PDFS and coronal PDFS showing osteochondral lesion of the superomedial talar dome (Courtesy: Dr. Rajesh Botchu, Consultant MSK Radiologist, Royal Orthopaedic Hospital, Birmingham, UK)

Treatment

1. Nonoperative:
   A short leg cast and non-weight bearing for 6 weeks.
   The main indications for this are:
   (a) Acute injury.
   (b) Undisplaced fragment with incomplete fracture.
2. Surgical/Operative:
   (a) Arthroscopy with removal of the loose fragment with microfracture (marrow stimulation or antegrade drilling of the base). This is mainly indicated in chronic fractures, fragment size being <1 cm, or when the displaced smaller fragment with minimal bone in the osteochondral fragment.
   (b) Retrograde drilling and/or bone grafting: It is indicated when the fragment size >1 cm with intact cartilage cap.
   (c) ORIF vs. osteochondral grafting: It is mainly indicated when the fragment size >0.5 cm and displaced with an emphasis on rehabilitation, particularly peroneal strengthening and range of motion.

Rupture of the Peroneus Tertius

The muscle arises from the lower third of the anterior surface of the fibula (anterior compartment of the lower leg), from the lower part of the interosseous membrane, and from an intermuscular septum between it and the peroneus brevis muscle. The tendon, after passing under the superior extensor retinaculum of the foot and inferior extensor retinaculum of the foot in the same canal as the extensor digitorum longus, is inserted into the dorsal surface of the base of the metatarsal bone of the fifth digit. It is innervated by the deep fibular nerve, unlike the other peroneal muscles which are innervated by the superficial fibular nerve, since the peroneus tertius is a member of the anterior compartment. Its action is that of weak dorsiflexion of the ankle joint and to evert the foot at the ankle joint. The muscle is uncommonly found in humans. Magnetic resonance (MR) imaging demonstrates a short-segment longitudinal split tear adjacent to the tendinous insertion of the peroneus tertius muscle.

Peroneal tendon ruptures are often the result of an inversion ankle sprain. The peroneus tertius muscle arises in the anterior compartment of the leg and demonstrates significant morphologic variation. There has been a report in a child of an isolated rupture of this tendon giving rise to lateral-based ankle pain which was treated by surgical reconstruction with relief [11]. Injury to the PT tendon is rare with virtually no cases reported in the literature. As a consequence of the rarity of this injury, there is little clinical information regarding injury or rupture of the peroneus tertius muscle and tendon. Magnetic resonance (MR) imaging demonstrates a short-segment longitudinal split tear adjacent to the tendinous insertion of the peroneus tertius muscle [12].

Compartment Syndrome of the Leg

A compartment syndrome is a clinical condition characterized by an increased tissue pressure with a closed anatomical space that compromises the circulation and function of that space.

It is a true orthopedic emergency which is typically seen in the leg, but can also occur at other places like the shoulder, arm, forearm, hand, gluteal region, iliopsoas, thigh, and foot.

Historical Aspects

In 1881, Richard von Volkmann [13] published an article in which he attempted to describe the condition of irreversible contractures of the flexor muscles of the hand to the ischemic process occurring in the forearm.

In 1906, Hildebrand [14] first used the term Volkmann ischemic contracture to describe the final result of any untreated compartment syndrome and was first to suggest that elevated tissue pressure may be related to ischemic contracture.

In 1909, Thomas [15] reviewed 112 published cases of Volkmann ischemic contracture and found fractures to be the predominant cause. Also he noted that tight bandages, an arterial embolus, or arterial insufficiency could lead to this problem.

Murphy in 1919 [16] was the first to suggest fasciotomy might improve the contracture. He also suggested that tissue pressure and fasciotomy were related to the contracture.

Seddon, Kelly, and Whitesides [17] in 1967 demonstrated the existence of four compartments in the leg and the need to decompress more than just the anterior compartment. Since then, compartment syndrome has been shown to affect many areas of the body including the hand, foot, thigh, and buttocks.

Aetiology

Decrease in compartment size, due to (1) tight dressing and bandage/cast, (2) localized external pressure like lying on the affected limb, and (3) closure of fascial defects.

Increase in the compartment contents: (1) bleeding-fractures, vascular injury, and bleeding disorders and (2) increased capillary permeability/ischemia/trauma/burns/exercise/snake bite/drug injection/IVF.

Fracture

It is the commonest cause with an incidence of accompanying compartment syndrome of 9.1%. The incidence is directly proportional to the degree of soft tissue and bone [18].

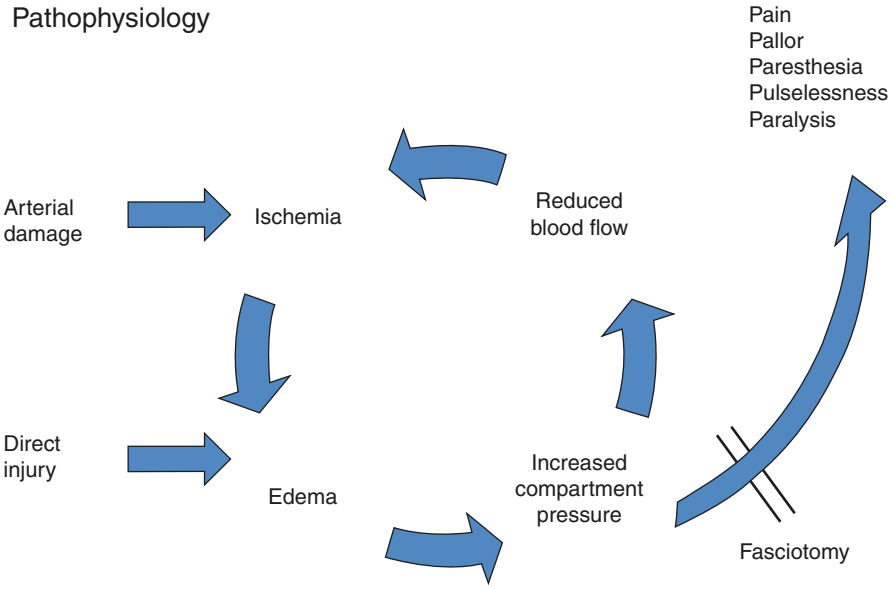


Fig. 25.11 Line diagram of the sequence of events in compartment syndrome

Treatment of Compartment Syndrome

Acute Compartment Syndrome: It is a medical emergency which requires urgent intervention to correct. It can lead to permanent muscle or nerve damage and can also result in the loss of function of the limb [11].

Chronic Compartment Syndrome: It is also known as exertional compartment syndrome. It is not a medical emergency. It is most often caused by athletic exertion (Fig. 25.11).

Pathophysiology

A. Normal tissue pressure: It is normally 0–4 mmHg, while with exertion is 8–10.
B. Absolute pressure theory: It is 80 mmHg (Mubarak) and 45 mmHg (Matson).
C. Pressure gradient theory: <20 mmHg of diastolic pressure (Fig. 25.12) (Whitesides et al. [19]).

Delta pressure, currently used to diagnose acute compartment syndrome, is less than or equal to 30 mmHg (P = DBP–ICP).

Rowand described AV gradient theory: Local blood flow as per the arteriovenous gradient theory which can be expressed as LBF = Pa–Pv/R.

Pathophysiology

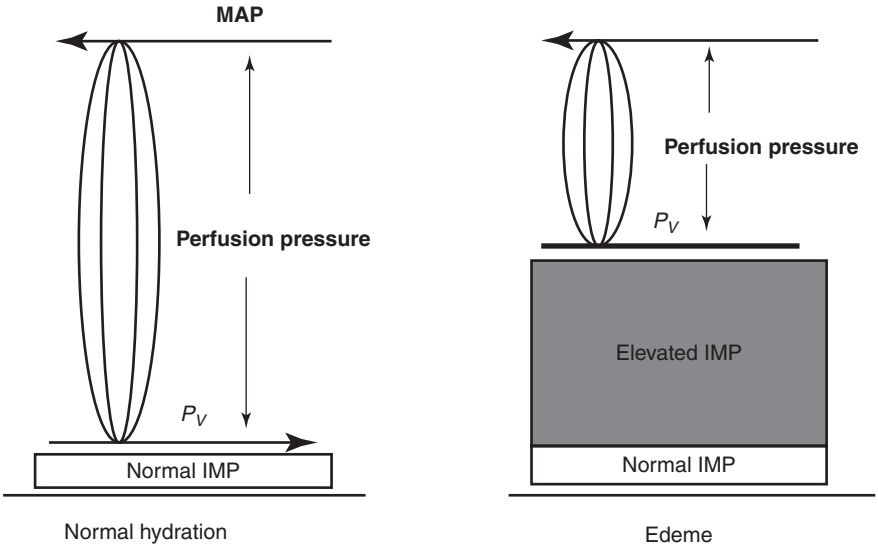


Fig. 25.12 Line diagram showing the AV gradient theory of pathophysiology

Stage	Pathophysiology	Clinical Symptoms
I	Increased intramuscular pressure	-swelling -decreased range of motion
II	Decreased muscle Blood flow	-Ischaemic pain at rest -Pain at passive stretch
IIII	Impaired function	Sensory and motor dysfunction.

Fig. 25.13 Table showing the pathophysiology of compartment syndrome

Tissue Survival

Muscle: 3–4 h = reversible changes, 6 h = variable damage, 8 h = irreversible damage.

Nerve: 2 h = loss of nerve conduction, 4 h = neuropraxia, 8 h = irreversible changes.

Diagnosis: Pain with the aggravation of pain by passive stretching of the muscles in question are the most sensitive clinical finding before the onset of ischemic dysfunction in the nerves and muscles.

Development of the syndrome may be described in three clinical stages (Fig. 25.13) that are related to the pathophysiological stages as follows:

History: Tightness of compartment, local pain and duration.

Clinical examination (the P's—the first three are most reliable):

1. Pain out of proportion.
2. Palpably tense compartment.
3. Pain with passive stretch.
4. Paresthesia/hypoesthesia.
5. Paralysis.
6. Pulselessness/pallor.

Compartment pressures = objective parameter.

Lab Tests: CPK, urine myoglobin.

Pulse Oximetry: helpful in identifying limb hypoperfusion. It is not sensitive enough to exclude compartment syndrome.

Clinical Parameters

1. Pain: Usually the first symptom. Classically out of proportion to injury. Exaggerated with passive stretch with involved muscles in the compartment. It is the earliest symptom, but inconsistent. It is minimal in the deep posterior compartment. It is not applicable in an unconscious patient.
2. Tense Compartment: It is an early finding as compared to the other side.
3. Paresthesia: The peripheral nerve tissue is more sensitive than muscle to ischemia. It is difficult to interpret. It will progress to anesthesia if the pressure is not relieved.
4. Paralysis: This is a very late finding when irreversible nerve and muscle damage are present. The paresis may present early but may be difficult to evaluate because of pain. If motor deficit develops, then full recovery is very rare.
5. Pallor and Pulselessness: It is rarely present. It indicated direct damage to vessels rather than compartment syndrome (hence arteriography is indicated). Vascular injury may be more of a contributing factor to the syndrome than the result.

Clinical Evaluation

Beware of epidural anesthesia, long-acting nerve blocks, and controlled intravenous opiate analgesia.

Compartment Pressure Monitoring

Raised tissue pressure is the primary event in compartment syndrome, and changes in the ICP may precede the clinical signs and symptoms.

When to do compartment monitoring. The ideal time is (1) when compartment syndrome is suspected, (2) equivocal or unreliable findings, (3) obtunded patient

with tight compartments, (4) vascular injury, (5) regional anesthesia, and (6) clinical adjunct.

It is contraindicated in clinically evident compartment syndrome.

Pressure Measurements

(a) Infusion technique: Designed by Whitesides [20]. It basically is a simple technique which can be used for intermittent recordings. The disadvantages are as follows: (a) it is not suitable for continuous monitoring and (b) required injection of saline into the compartment which can aggravate impending syndrome (Fig. 25.14).

(b) Slit and wick catheters: It requires bubble-free column of saline. The transducer dome should be level with the insertion site. It is more accurate and can also be used for continuous monitoring. Its disadvantages are that the tip of the catheter may become blocked by a blood clot (Fig. 25.15).

(c) Stryker STIC catheter system: It is a handheld device which is easy to use. It measures pressures quickly and can be carried in the pocket. It is more accurate and can be used for continuous monitoring (Fig. 25.16).

(d) Near-infrared spectroscopy (NIRS): It is a noninvasive method of detecting variations in the level of muscle hemoglobin and muscle myoglobin. It has a good predictive power in chronic exertional CS. The diagnostic value in acute compartment syndrome is limited since changes in relative oxygenation may have already occurred (Fig. 25.17).

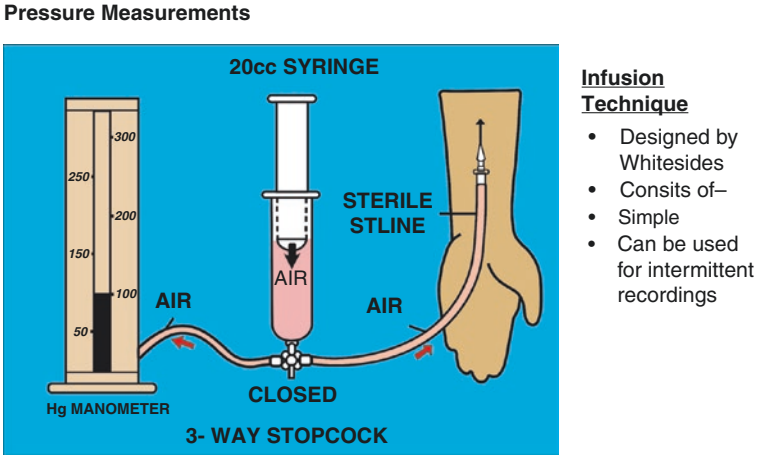
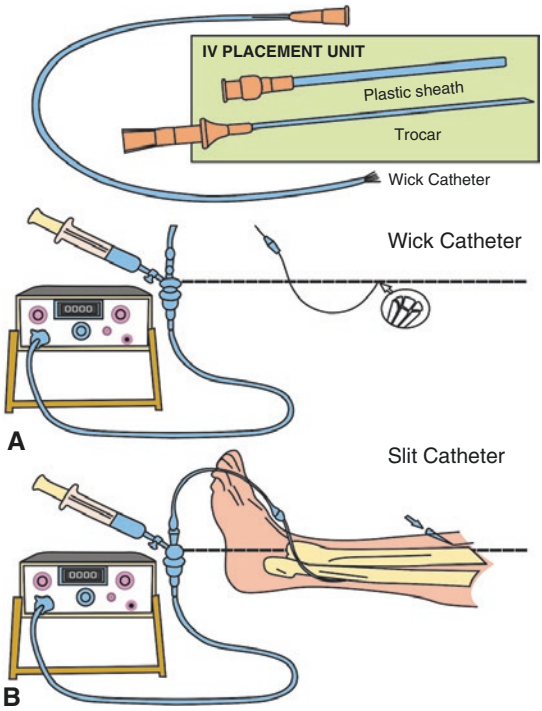


Fig. 25.14 Line diagram showing the infusion technique of measurement of pressure



Slit and wick Catheters

- Requires bubble free column of smile
- The transducer dome should be level with the insertion site
- More accurate
- Can be used for continuous monitoring

Disadvantage –Tip of the catheter may become blocked by a blood clot.

Fig. 25.15 Line diagram showing the slit and wick catheters

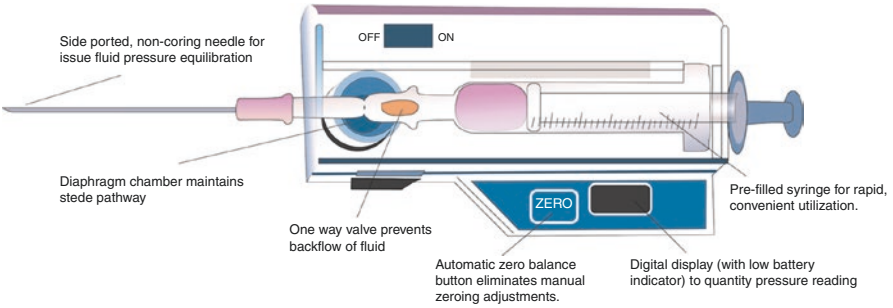


Fig. 25.16 Line diagram showing the Stryker STIC catheter system

(e) Scintigraphy: It is used to evaluate regional perfusion, in particular myocardial perfusion. Edwards et al. investigated 99Tcm-methoxyisobutyl isonitrile (MIBI) scintigraphy in patients with suspected CECS and found predictive results in 89%. It is unlikely to be of value in acute trauma as it cannot be used for continuous monitoring of perfusion.

(f) Electronic transducer-tipped catheter system: It is easy to use and it is a non-infusion technique which is highly precise (Fig. 25.18). It is free of hydrostatic pressure artifacts and provides dynamic responses to changes in the intramuscular pressure.

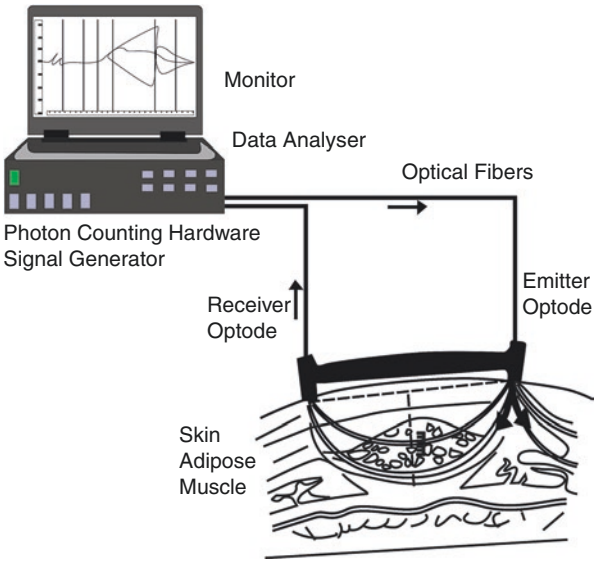


Fig. 25.17 Line diagram showing NIRS

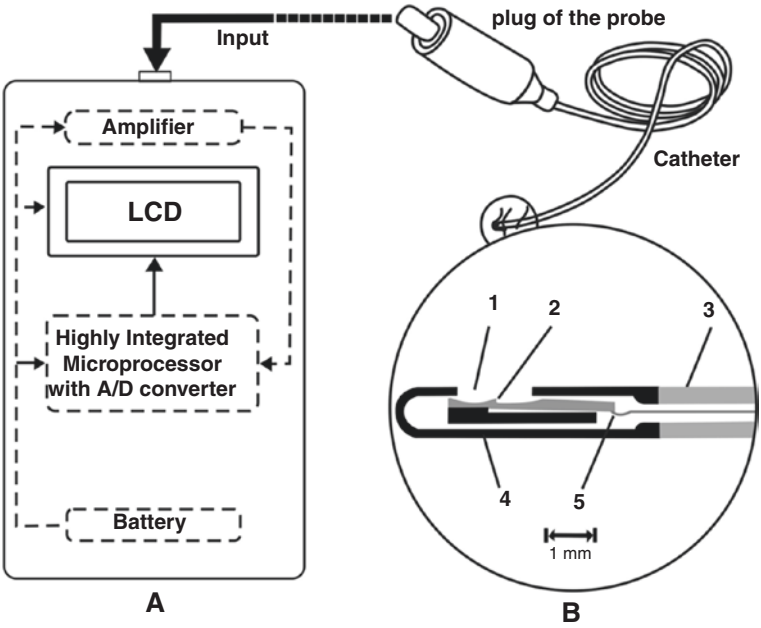


Fig. 25.18 Line diagram showing the electronic transducer catheter-tipped system

Pressure Measurements

1. The measurement must be made in all the compartments.
2. The highest pressure is seen in the anterior or deep posterior compartment.
3. The measurements are made within 5 cm of fractures.
4. Marginal readings must be followed up with repeat physical examination and repeat compartmental pressure measurement.
5. The highest figures must be used in deciding the need for fasciotomies [21]

Pressure Threshold Theory for Fasciotomy

McQueen, Court-Brown JBJS (Br) concluded that decompression should be performed if the differential pressure level drops to under 30 mmHg [22] (Fig. 25.19).

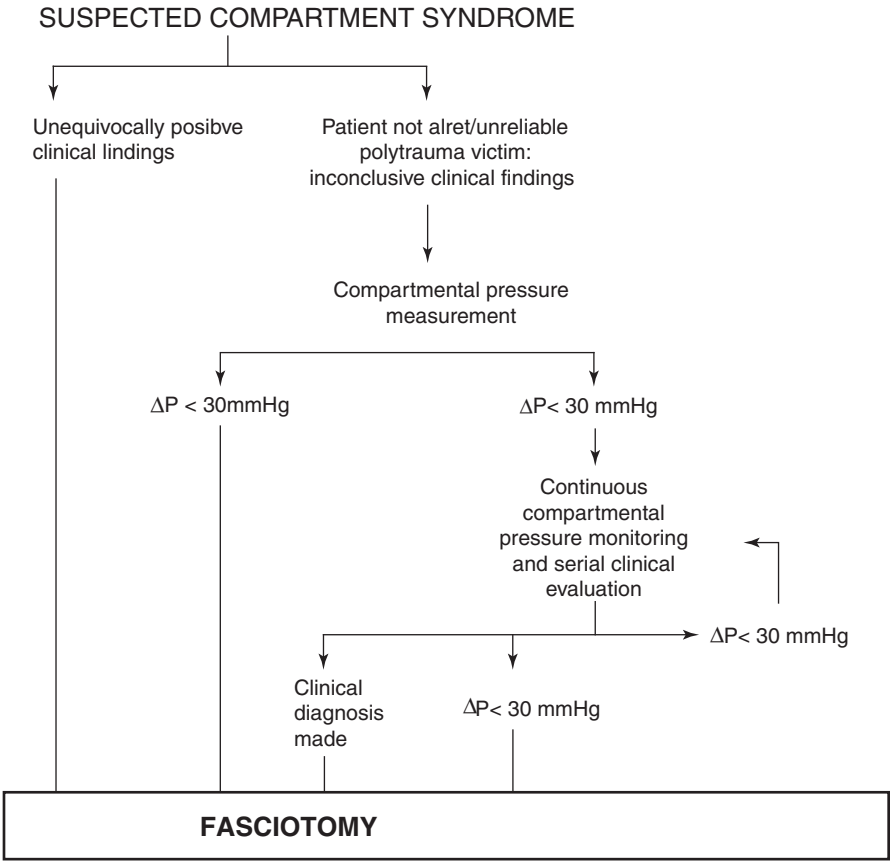


Fig. 25.19 Line diagram showing importance of suspected compartment syndrome

Emergency Treatment

1. Remove cast or dressing.
2. Place at the level of the heart. (NB: Do not elevate as elevation reduces the arterial inflow and the arteriovenous pressure gradient on which perfusion depends [perfusion pressure = Pa −Pv].)
3. Alert or anesthesia.
4. Medical treatment: Supplement oxygen administration.
5. Ensure that the patient is normotensive, as hypotension reduces perfusion pressure and facilitates further tissue injury.

Removal of Circumferential Casts

1. Compartment pressure falls by 30% when the cast is split on one side.
2. Compartment pressure falls by 65% when the cast is spread.
3. Splitting the padding reduces it further by 10% and complete removal of the cast by another 15%.
4. Total of 85–90% reduction by just taking off the plaster.

Surgical treatment is by "fasciotomy" of all the three compartments.

1. Prophylactic release of pressure before permanent damage occurs.
2. It does not reverse the damage present but can prevent secondary sequelae of CS.
3. Always look for direct injury to vessels and nerves, which should be repaired if they exist.
4. Fracture care: Stabilize by plating, intramedullary mailing, or external fixator

Indications for Fasciotomy

1. Unequivocal positive clinical findings.
2. Pressure within 15–20 mm of Hg of DBP.
3. Rising tissue pressure.
4. Significant tissue injury or high-risk patient.
5. >6 hours of total limb ischemia.
6. Injury at high risk of compartment syndrome.

Contraindication

Missed compartment syndrome (>24–48 h).

(Sheridan and Matsan reported an infection rate of 46% and an amputation rate of 21% after late fasciotomy [23].)

Principles of Fasciotomy

Make an early diagnosis, make long extensile incisions, release all fascial compartments, preserve neurovascular structures, debride necrotic tissues, coverage within 7–10 days.

Lower Leg: It has four compartments, namely, (1) lateral having the peroneus longus and brevis; (2) anterior having the EHL, EDL, tibialis anterior, and peroneus tertius; (3) superficial posterior having the gastrocnemius, plantaris, and soleus; and (4) deep posterior having the tibialis posterior, FHL, and FDL.

Compartments of the Leg

There are three decompression techniques, namely, (1) fibulectomy, (2) perifibular fasciotomy, and (3) double-incision fasciotomy.

1. Fibulectomy: It was described by Patman and Thompson. It is unnecessary and too radical. It is obsolete now.
2. Perifibular fasciotomy: It was described by Matson et al. (1980). It consists of a single lateral incision just posterior to the fibula, which extends proximally from the head of the fibula to the ankle. Then it exposes and protects the common peroneal nerve proximally. It is more difficult to decompress the deep posterior compartment.
3. Double-incision fasciotomy: In most instances it affords a better exposure of all the four compartments. It consists of two vertical incisions extending from the knee to the ankle separated by a minimum of 8 cm [20] (Fig. 25.20).

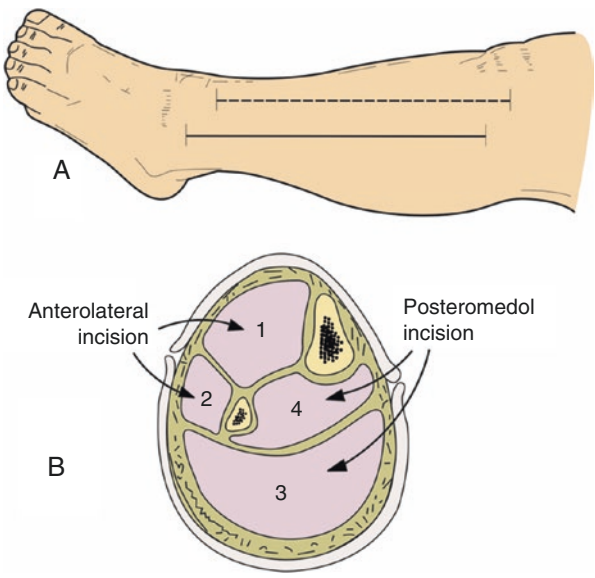


Fig. 25.20 Line diagram showing cross-sectional view of double-incision fasciotomy

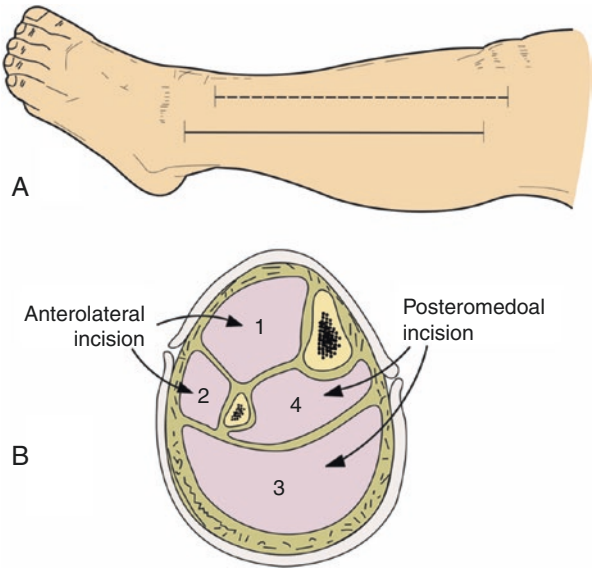


Fig. 25.21 Line diagram showing lateral view of double-incision fasciotomy

One incision over the interval between the anterior and lateral compartments is taking care to preserve the superficial peroneal nerve. The other incision located 1–2 cm behind the posteromedial aspect of the tibia is taking care to avoid injury to the saphenous nerve and vein.

The use of the generous incisions (Fig. 25.21) is supported. Lengthening the skin incisions to an average of 16 cm decreases the intra-compartmental pressure significantly.

Wound management: After fasciotomy the wound must be debrided of all devitalized tissue. The wound is not closed at initial surgery. A bulky compression dressing and a splint are applied. "VAC" (vacuum-assisted closure) can be used. The incision for fasciotomy is closed after 3–5 days. After 48 h, the wound is inspected and any further necrotic tissue is removed. Usually the wound requires a skin graft. The goal is to obtain a definite coverage within 7–10 days for which several techniques are available: (1) progressive closure by wire sutures or tape, (2) ETE (external tissue extender) tissue bands, (3) dermatotraction, (4) rod-tensioning device, and (5) skin grafting.

Complications related to fasciotomies:

1. Altered sensation within the margins of the wound (77%).
2. Dry, scaly skin (40%).
3. Pruritus (33%).
4. Discolored wounds (30%).
5. Swollen limbs (25%).
6. Tethered scars (26%).
7. Recurrent ulceration (13%).

8. Muscle herniation (13%).
9. Pain related to the wound (10%).
10. Tethered tendons (7%).

Complications of CS:
1. Volkmann's contracture.
2. Weak dorsiflexors.
3. Claw toes.
4. Sensory loss.
5. Chronic pain.
6. Amputation.

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Clinical Examination of the Foot and Ankle (Basic and Surface Anatomy) with Special Tests

26

Maneesh Bhatia and Sheweidin Aziz

Gait

The simple definition of gait is "the person's manner of walking." It is the progression of the body from one point to another in an energy-efficient process involving synchronous joint and muscle movements. The bipedal gait has enabled humans to use their hands and be stable in an upright position. Gait is divided into stance and swing phases which comprise 60% and 40%, respectively.

The stance phase is further divided into (1) initial contact (2) loading response (3) mid-stance (4) terminal stance and (5) pre-swing. The swing phase is divided into (1) initial swing (2) mid-swing and (3) terminal swing. These subdivisions of the gait cycle are difficult to differentiate in the clinical setting; however, the focus should be on the three rockers. There are five prerequisites of normal gait described by Perry in 1985 [1] and they include (1) stability in stance (2) adequate foot clearance in swing (3) adequate step length (4) appropriate pre-positioning of the foot in terminal swing and (5) energy conservation.

Perry further described muscle actions during the stance phase by combining the initial contact and loading response as the first rocker. During the first rocker, the fulcrum and ground reaction force (GRF) is at the heel and progressively moves anteriorly by eccentric contraction of the anterior compartment muscles of the leg. The second rocker is the mid-stance phase and the GRF moves forward to become anterior to the knee; there is eccentric contraction of the plantarflexors. The third rocker is the terminal stance. As the heel begins to lift as a result of concentric contraction of the gastrocnemius and soleus complex, the pivot point moves forward toward the metatarsal heads.

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Start by asking the patient to walk in their shoes with walking aid in their usual manner, and note foot, knee and hip movements. Pay attention to Perry's rockers and note changes in normal gait phases. Ask the patient to walk on their tiptoes, heels, and inner and outer borders. Inability to walk in this fashion may correlate to various pathologies which will be described later in the chapter.

In this chapter we will discuss the common types of gait:

1. Antalgic: This is by far the most common gait pattern seen in orthopedic clinics. It is secondary to pain. There is shortening of the stance phase on the affected side and shortened swing phase on the contralateral side.
2. Equinus: Walking on tiptoes, e.g., plantarflexion contracture.
3. Trendelenburg: Observed on the coronal plane. The hip abductors are either inactivated due to pain at the hip joint or are weak resulting in a drop of the pelvis on the contralateral side. This could further be confirmed by performing the Trendelenburg test.
4. Steppage/high step: The forefoot is the initial contact point, and there is usually as a slap as there is no eccentric contraction of the anterior leg compartment muscles resulting in uncontrolled plantarflexion of the foot, e.g., foot drop secondary to peroneal nerve palsy or tibialis anterior tendon rupture.
5. Short leg: Pelvic drop: The pelvis droops on the affected side.
6. Scissoring: Legs cross over each other, e.g., adductor contracture in spastic cerebral palsy.

Walking Aid

The type of walking aid and which side it is held should be noted. Gait should be assessed with and without walking aid if possible. You should also observe whether the walking aid is the right height for the patient and maintains patient's coronal and sagittal balance.

Shoes

Assess whether shoes are standard off the shelf or customs made. Look for pattern of wear which is normally on the lateral aspect of the heel. Abnormal wear patterns including wear over the metatarsal heads may suggest an abnormal initial contact, e.g., leg length discrepancy or tight Achilles tendon. Other abnormal pattern may be medial wear where there is excessive pronation, e.g., pes planus deformity. Always compare to the other side. Look inside shoes for insoles, ascertain whether they are patient bought or health professional recommended.

Orthoses

Note the type of orthoses and whether it is corrective or accommodative. Describe the orthoses as seen, noting any hinges.

Inspection

Always look for scars, note their locations and how well they have healed. Assess the quality of skin for any signs of active infection or skin irritation. Look for discharging sinuses or weeping skin.

From the Front

1. Assess the alignment of the lower limbs, any pelvic obliquity is suggestive of leg length discrepancy. If there is a leg length discrepancy, then screen the spine for deformities followed by Galeazzi test to determine site of discrepancy.
2. Symmetry: Is the deformity bilateral and symmetrical? This is suggestive of a systemic or global pathology or is it unilateral suggestive of previous trauma.
3. Swelling and skin changes: If symmetrical, could be secondary to systemic diseases such as rheumatoid arthritis or peripheral vascular disease. If unilateral changes are present, could be due to trauma, venous thromboembolism (VTE), infection, stress fracture, or stress response (usually confirmed on MRI scan).
4. Callosities: Note the site and whether it is unilateral or bilateral. It is indicative of areas of abnormally increased pressure.
5. Observe for ecchymosis especially on the plantar surface which is suggestive of Lisfranc injuries.
6. Look for signs of peripheral vascular disease: loss of hair, shiny skin, discoloration, or dusky toes.
7. Look for active or healed ulcers noting the depth and size as well as clinical signs of infection which may be present in patients with diabetic neuropathy.
8. Inspect between the toes for areas of moisture, skin breakdown or active infection suggestive of poor hygiene, vitamin D deficiency, fungal infections and peripheral neuropathy.
9. Hallux varus or valgus: Note the position of the great toe in relation to the lesser toes. Note whether there is an overlying bunion and whether the overlying skin is irritated or breaking down.
10. Assess the overall width of the forefoot: This is increased in bunion and bunionette deformities.
11. "Peek-a-boo sign": Looking from the front, the medial heel pad is not normally seen; when seen it is a feature of cavo-varus deformity.
12. Look for increased intermetatarsal space especially between the second and third toes indicating an enlarged bursa.

From the Side

1. Lesser toe deformities: Make a note whether there is overriding of the second toe over the great toe. Evidence of under-riding or overriding fifth toe suggesting tightness of the extensor or flexor tendons, respectively. Look for a hammer deformity (flexion of the PIPJ), mallet toe (flexion of the DIPJ), or claw toe (hyperextension of the MTPJ, flexion of PIPJ and DIPJ). It is of vital importance

to note whether the deformities are flexible or rigid as this will impact whether a bony procedure will need to be added to a soft tissue procedure.
2. Medial longitudinal arch: Flattening would suggest pes planus or a high arch which could be a feature of pes cavus (Fig. 26.1).
3. Note the position of the first ray: A plantarflexed ray could be driving hindfoot varus as in cavo-varus deformity.

From the Back

1. Note whether the calves are of equal size suggestive of muscle atrophy in cases of neglected Achilles rupture or history of clubfoot.
2. First note the heel position which is normally neutral. When the heel is in varus, an assessment of flexibility should be performed using the Coleman block test (described below under cavo-varus foot). If the hindfoot is in valgus, ask the patient to stand on tiptoes holding onto wall for support; normally the heel should go into varus (Fig. 26.2) [2].

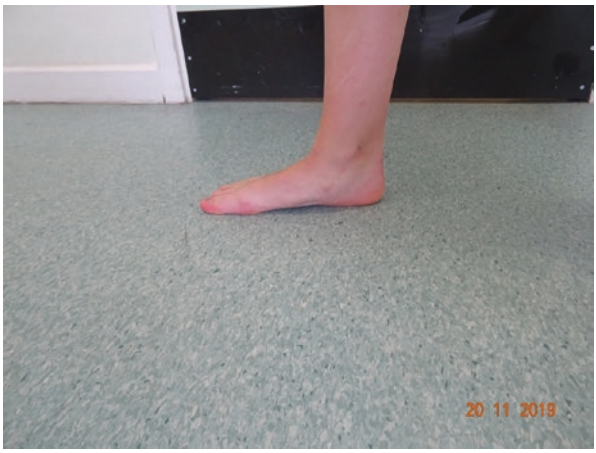

Fig. 26.1 Clinical photograph showing medial longitudinal arch flattening—pes planus

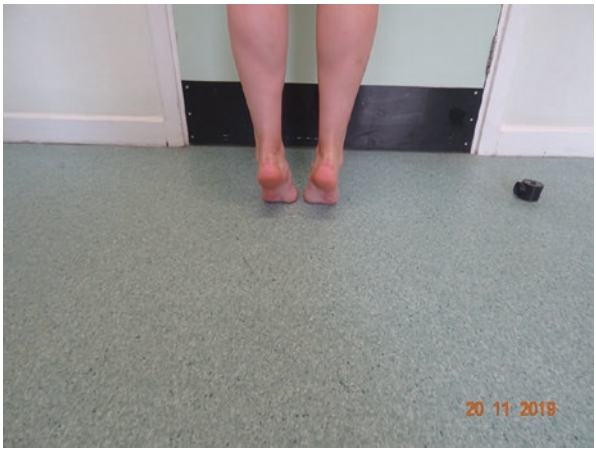

Fig. 26.2 Heel rise—clinical photograph showing the heel levels swing into varus in physiological flat feet

3. Too many toes sign (Fig. 26.3):
   Normally the lateral 1 ½ toes are seen from behind; however, when more toes are seen, this is indicative of an increased heel valgus angle as seen in acquired tibialis posterior insufficiency.
4. Metatarsus adductus: Increased lateral curvature of the foot.

Palpation

Palpation starts with assessment of temperature, always comparing to the other side. Temperature is increased in Charcot arthropathy and decreased in peripheral vascular disease.

The clinical photographs 26.4, 26.5, 26.6 depict the main bony landmarks for palpation.

Fig. 26.3 Too many toes—clinical photograph showing the right foot with five toes

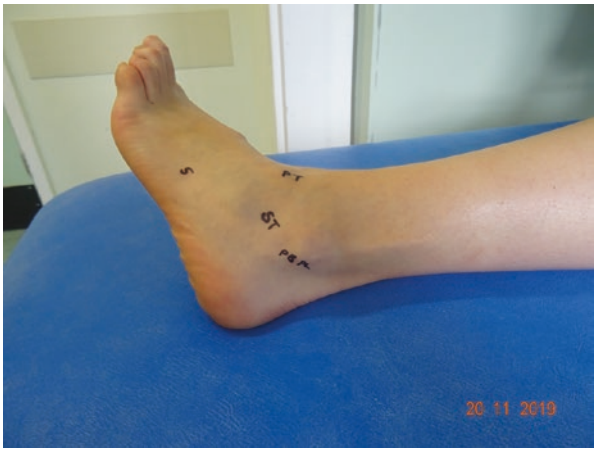

Fig. 26.4 Anatomical landmarks: clinical photograph of the lateral side showing *5* fifth tarsometatarsal joint; *PT* peroneus tertius, *ST* sinus tarsi, *PB* peroneus brevis, *PL* peroneus longus

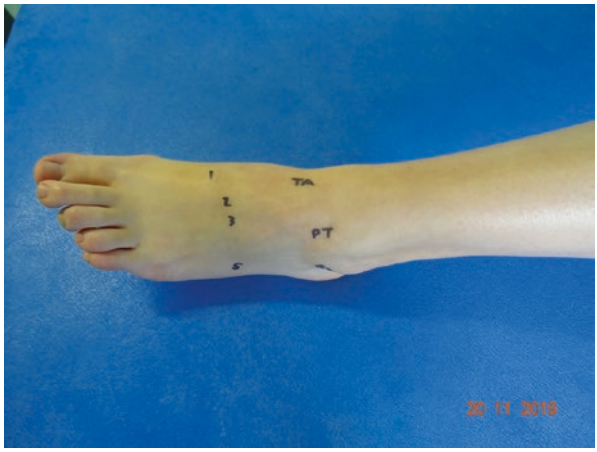

Fig. 26.5 Anatomical landmarks: clinical photograph of the dorsum of the ankle and foot showing *1* TMTJ, *2* TMTJ, *3* TMTJ; *TA* tibialis anterior, *PT* peroneus tertius

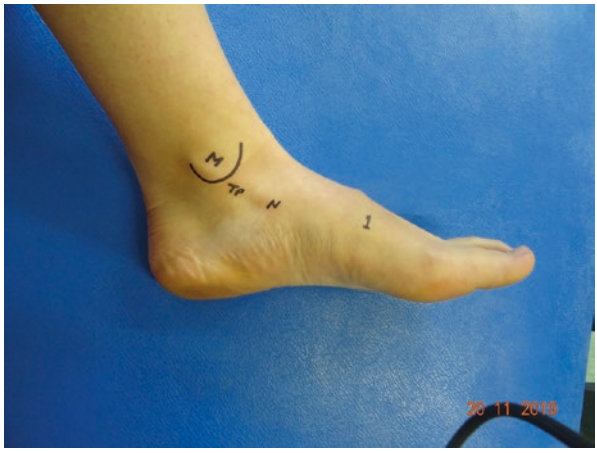

Fig. 26.6 Anatomical landmarks: clinical photograph of the medial side showing *M* medial malleolus, *TP* tibialis posterior, *N* navicular, *1* first metatarsal

Start palpation away from the area of maximum tenderness in an orderly manner ensuring all parts of the foot and ankle are examined. Having a system that is reproducible and organized is important in foot and ankle examination and comes with practice and experience.

Range of Movement

Both active and passive movements are important to note. It is a good practice to compare them to the contralateral side as normal range of motion varies between individuals, but these are broad ranges of reference. Ankle dorsiflexion is usually about 0–20°; this value is reduced in cases of tight Achilles tendon or anterior ankle impingement secondary to osteophytes. Ankle plantarflexion is usually

about 0–45°; this is usually reduced secondary to anterior capsule tightness seen in osteoarthritis.

Inversion and eversion normally take place at the subtalar joint. Inversion has been reported as a wide range of 0–35°; this could be increased in lateral ligament laxity or reduced in cases of stiffness following injury or surgery. Eversion of 0–15°, on the other hand, could be increased in medial ligament (deltoid) injury. Both inversion and eversion may be reduced in cases of tarsal coalition or advanced hindfoot arthritis.

To test for passive ankle dorsiflexion and plantarflexion, hold the lower leg just above the ankle joint with one hand, hold the heel in the other palm, and place the foot on the forearm and push upward and downward (Fig. 26.7).

The measurements are from neutral position and should always be compared to the other side. To passively assess subtalar movements, place the index and thumb over the Tatar neck while cupping the heel with the other hand, and move the heel in the coronal plane to assess for inversion and eversion (Fig. 26.8).

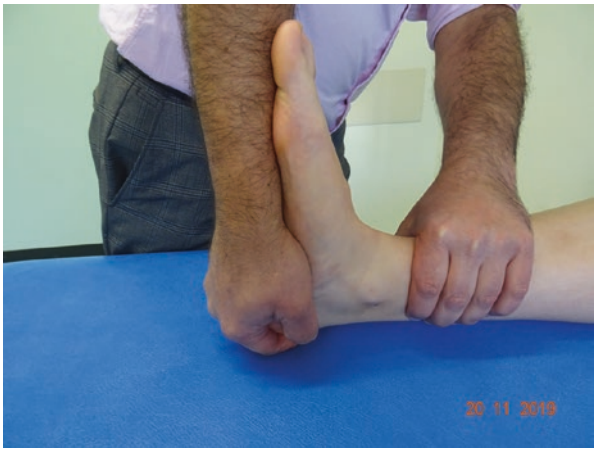

Fig. 26.7 Clinical photograph showing passive dorsiflexion of the ankle

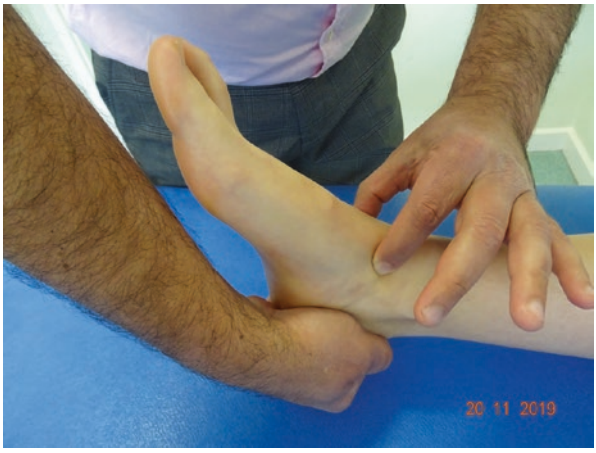

Fig. 26.8 Clinical photograph showing passive subtalar joint movement

Movement of the first ray MTPJ should be assessed in cases of suspected arthritis and compared to the other side. Make a note of whether plantarflexion and dorsiflexion is painful through range of movement or only at the extremes suggesting dorsal osteophytes or early degenerative disease. Assess the strength of the tibialis anterior by palpating the tendon and asking the patient to dorsiflex and invert against resistance (Fig. 26.9).

Tibialis posterior: palpate the tendon behind the medial malleolus, and ask the patient to invert foot in plantarflexion against resistance (Fig. 26.10).

Peroneal tendon: with the ankle in neutral position, ask the patient to evert against resistance (Fig. 26.11).

Power of the muscles should be documented using the Medical Research Council (MRC) grading system: Grade 0, no contraction or muscle movement; Grade 1, trace of contraction, but no movement at the joint; Grade 2, movement at the joint with gravity eliminated; Grade 3, movement against gravity, but not against added resistance; Grade 4, movement against external resistance with less strength than usual; and Grade 5, normal strength.

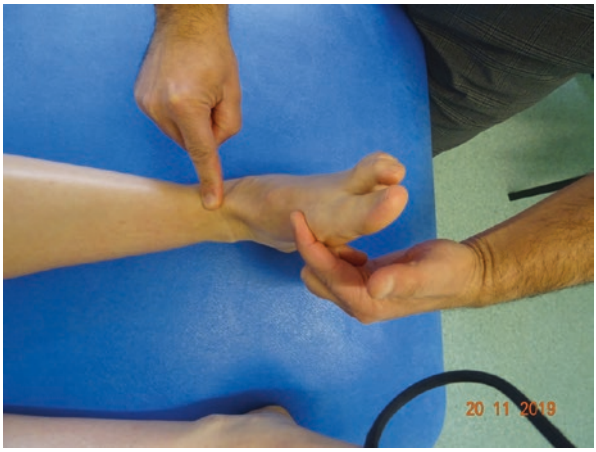

Fig. 26.9 Clinical photograph showing the power of the tibialis anterior tendon

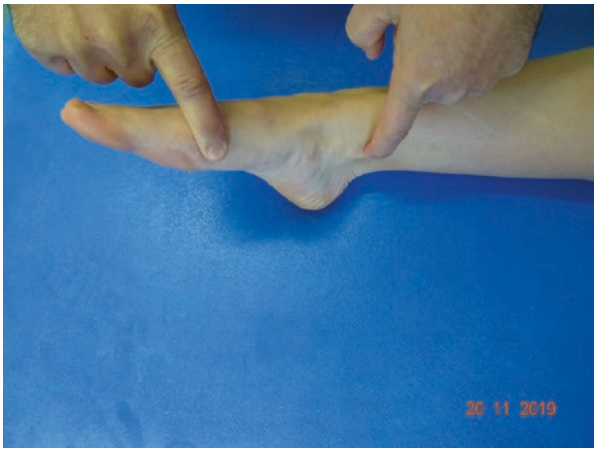

Fig. 26.10 Clinical photograph showing the power of the tibialis posterior tendon

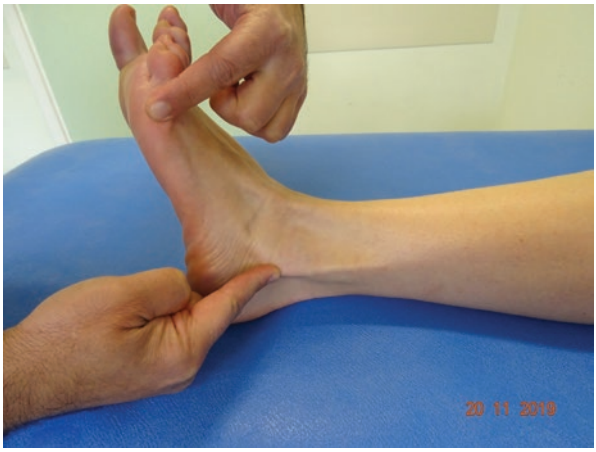

Fig. 26.11 Clinical photograph showing the power of the peroneal tendon

Vascular Examination

A thorough vascular examination is required to assess extent of potential risk secondary to peripheral vascular disease or diabetes. The presence of current or healed ulcers are poor prognostic features. Clear documentation of distal pulses must be made. If the dorsalis pedis or posterior tibial pulses cannot be palpated, then a Doppler should be employed to assess flow (triphasic waveform is normal). The dorsalis pedis pulse can be palpated on the dorsum of the foot just lateral to EHL or medial to EDL. Posterior tibial artery is located just posterior to the medial malleolus. ABPI should be performed where normal is 0.9–1.3, a reading of higher than 1.3 is indicative of calcified vessels and poor flow, and <0.5 is indicative of ischemia. When ABPI is >1.3, toe pressures are more reliable and a systolic >40 mmHg is a predictor of healing.

Neurological Examination

It is of vital importance to assess the neurological status of the foot with clear documentation before and after any intervention. Loss of protective sensation, in diabetic neuropathy, can lead to devastating ulceration that could become infected and lead to loss of limb. Loss of sensation that does not follow a dermatomal distribution and instead a stocking distribution is more in keeping of diabetic or alcoholic neuropathy. Inability to feel the Semmes-Weinstein monofilament 5.07 is consistent of peripheral neuropathy [3]. Dull aches across the medial aspect of the foot with associated loss of sensation or tingling could be a sign of tarsal tunnel syndrome requiring further investigation. The five main sensory nerves to test are superficial peroneal, deep peroneal, tibial, sural, and saphenous.

Forefoot

1. Medial and lateral sesamoiditis: Localized tenderness over the medial and lateral sesamoids. Medial sesamoiditis may be a result of overload due to excessive pronation which might resolve with orthotics.
2. Morton's neuroma (Fig. 26.12):

   Thumb index squeeze test: There is tenderness of the third and/or second intermetatarsal spaces when pressed using the index and thumb. **Mulder's click test**: although commonly performed, it is not very reliable (Fig. 26.13).

   Dorsiflex the foot and squeeze the metatarsal head; a positive test is an audible click [4]. Look for associated conditions such as lesser toe deformities, callosities and tenderness on palpation of the MTPJ indicating synovitis. It is important to check for sensations at the tip of toes and on the plantar aspect of foot as this could be diminished in Morton's neuroma.

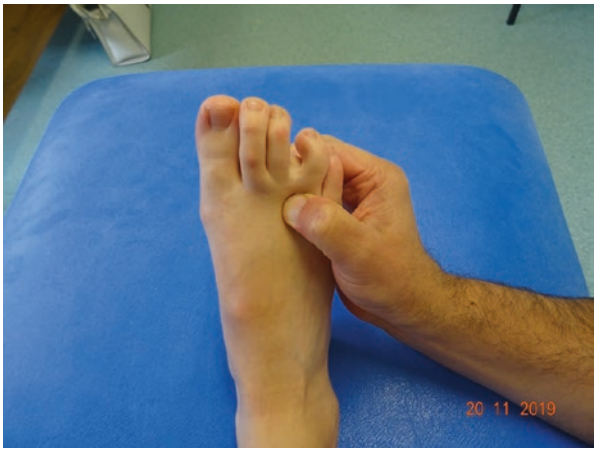

Fig. 26.12 Clinical photograph showing how to test for Morton's neuroma by squeezing the third web space

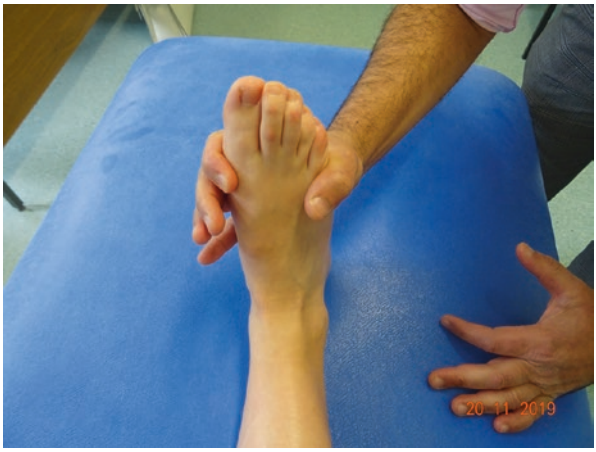

Fig. 26.13 Clinical photograph showing how to elicit a Mulder's click

3. Hallux valgus: There is lateral deviation of the big toe. Assess for medial prominence and state of overlying skin changes from shoe pressure resulting in erythema or bursa. The big toe could be over- or under-riding the second toe. Callosities at the IPJ indicate hyperpronation of the big toe when pushing off. Assess whether the deformity is correctable. Look for deformities of the lesser toes.
4. Hallux varus: This could be idiopathic or iatrogenic. There is medial deviation of the big toe. Look for scars which may indicate overcorrection of previous hallux valgus. Range of movement is usually painless unless there is associated degenerative joint disease.
5. Hallux rigidus: Assess for dorsal prominence suggestive of dorsal osteophytes. Note the range of movement, crepitus, and pain. Pain could be throughout all range of motion or at the end of dorsiflexion or plantarflexion. Normal range of motion can be as high as 65–75° and plantarflexion 5–15°. It is very important to always compare to the contralateral side. **Grind test**: Axial compression and rotation of the big toe will result in pain.
6. Freiberg's disease: Although not very common, this condition should be suspected in young females complaining of forefoot pain centered on the second metatarsal head. Pain worsens on weight bearing or on squeezing the second MTPJ (Fig. 26.14).

   In early stages of the disease, pain is worsened with distraction of the second MTPJ, while in late stages it is worsened with compaction.
7. Lesser toe deformities: You should firstly identify the deformity and then assess whether it is flexible or fixed. Flexible deformities can be managed with tenotomies, fixed deformities require fusion, and subluxed MTP joint requires osteotomies to reduce pressure of the metatarsal head. Dorsiflex the foot and reassess flexibility of the toe deformities in order to relax the extrinsic muscles of the foot:
   (a) Mallet: MTPJ and PIPJ are normal, DIPJ flexion. This is indicative of a tight flexor tendon.

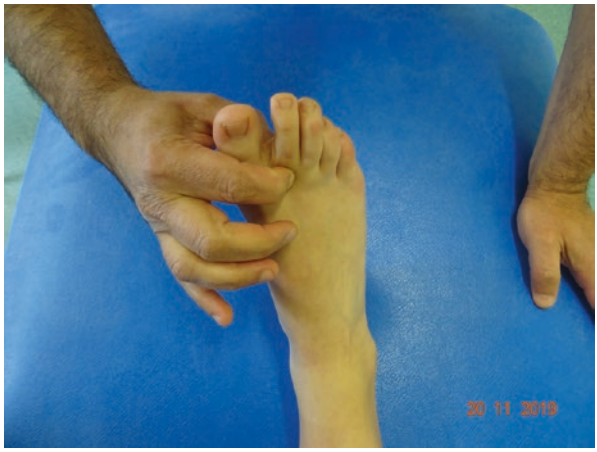

Fig. 26.14 Clinical photograph showing tenderness in Freiberg's disease

(b) Hammer: MTPJ normal, PIPJ flexion, and DIPJ extension.
(c) Claw: MTPJ hyperextension, PIPJ and DIPJ flexion. **Anterior draw or digital Lachman test for plantar plate**: over time the plantar plate becomes insufficient and should therefore be assessed. Hold the proximal phalanx in 20–25° of dorsiflexion relative to the metatarsal head; apply dorsal translation in an attempt to sublux the MTPJ. A positive test is defined as 2 mm of dorsal displacement or 50% joint subluxation.

Midfoot

1. Arthritis: This includes tarsometatarsal joints, naviculocuneiform joint, talonavicular joint, and calcaneocuboid joint. Patient will have tenderness overlying the respective joints with movement. To assess the midfoot, grasp the ankle and proximal foot still with one hand and supinating and pronating the distal metatarsals with the other hand (Fig. 26.15).

   Alternatively move the metatarsal shafts up and down in the coronal plane while stabilizing the hindfoot and ankle.
2. Accessory navicular: Tenderness over the navicular bone in young females which becomes inflamed at the site of tibialis posterior insertion site.

Hindfoot

1. Ankle: Look for antalgic gait, effusion, and pain on range of movement. In late stages or when secondary to previous trauma, there may be associated deformity. **Anterior impingement test**: ask the patient to squat; a positive test will result in reduced ankle dorsiflexion compared to the contralateral side.

   Posterior impingement test: Pain on the posterior aspect of the ankle on passive plantarflexion is a positive test (Figs. 26.16 and 26.17).
2. Plantar fasciitis: Palpate for tenderness over the plantar medial aspect of the calcaneum. It is very important to assess for gastrocnemius complex tightness. Silfverskiold test (Figs. 26.18 and 26.19):

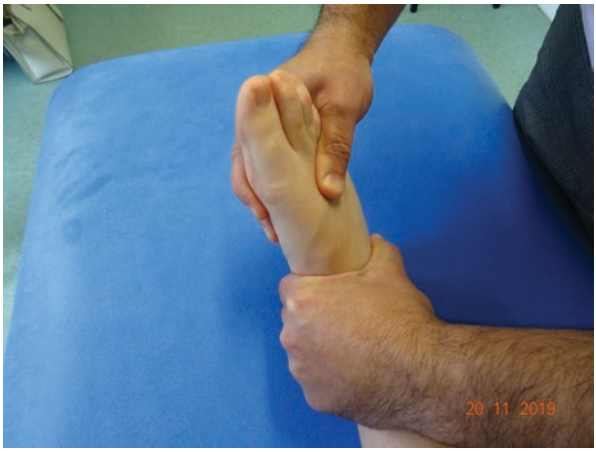

Fig. 26.15 Clinical photograph showing how to test for the ROM (range of movement) of the midfoot joint

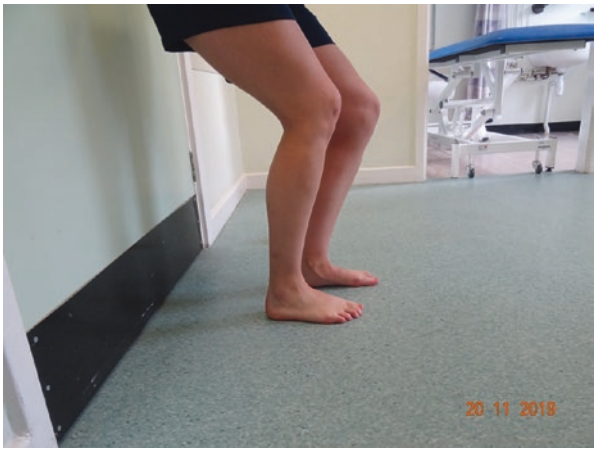

Fig. 26.16 Clinical photograph showing anterior impingement

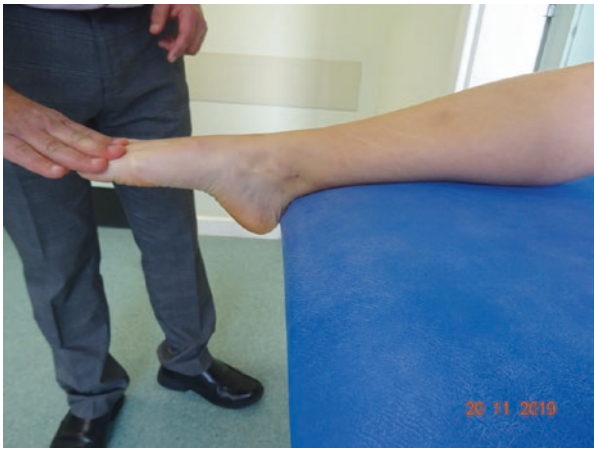

Fig. 26.17 Clinical photograph showing passive dorsiflexion of the ankle will result in ankle pain posteriorly

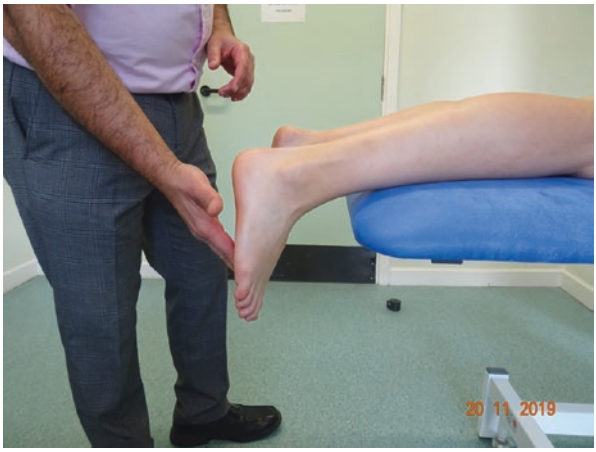

Fig. 26.18 Clinical photograph showing Silfverskiold test to assess ankle dorsiflexion with the knee extended

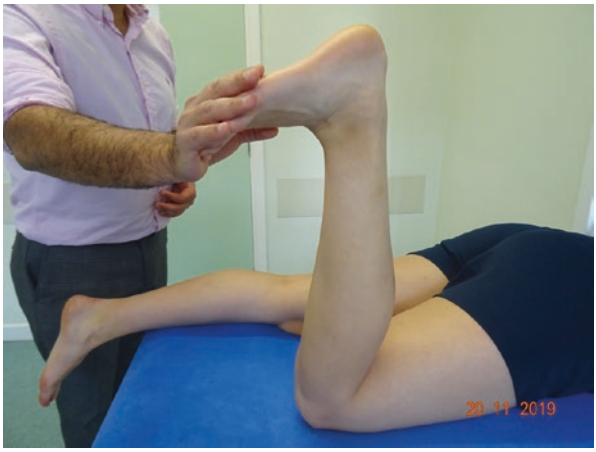

Fig. 26.19 Clinical photograph showing Silfverskiold test with the knee flexed to 90°

3. This test can be done with the patient supine or prone, although if patient is able to lie prone, it is easier to compare to other side. Note the degree of ankle dorsiflexion with knees extended and flexed. If there is reduction in ankle dorsiflexion with knee extended and flexed, then the gastrocnemius and soleus are both tight, and Achilles tendon release is required to regain motion. If dorsiflexion is limited with knee extended but improves with knee flexion, then the gastrocnemius only is tight [5].
4. Cavo-varus: Look for tibialis anterior weakness during swing phase of the gait assessment. Look for toe deformities and recruitment of EHL resulting in cock-up toes. Assess the strength of the muscles (weak tibialis anterior and peroneal muscles). Shoes would have excessive lateral wear. Look for hindfoot alignment from behind which will be in varus and "peek-a-boo" sign. Look for plantarflexion of the forefoot by examining the foot with the patient supine (Fig. 26.20).

Coleman block test: with the patient standing, a one inch block is placed on the lateral aspect of the foot freeing up the plantarflexed first ray and eliminating contribution of forefoot pronation. A flexible hindfoot will correct to neutral or valgus. A spine examination is also required to be completed to look for scoliosis and signs of spine dysraphism.

1. Pes planus: From the side (Fig. 26.21):

   The medial longitudinal arch is reduced, inability to introduce the first palmar crease of the index finger under the arch. From behind: "Too many toes sign" more than the normal 1 ½ toes (Fig. 26.22). The hindfoot will be in valgus, ask the patient to stand on tiptoes while holding onto wall, and see if the hindfoot corrects to neutral or varus position (Fig. 26.23). If the patient is able to stand on both tiptoes, then ask them to stand on each tiptoe of each foot to assess the strength of the tibialis posterior tendon. Inability to perform single heel rise is indicative of tibialis posterior insufficiency. Patients may be able to stand on tiptoes but are unable to repeat more than five times in quick succession which

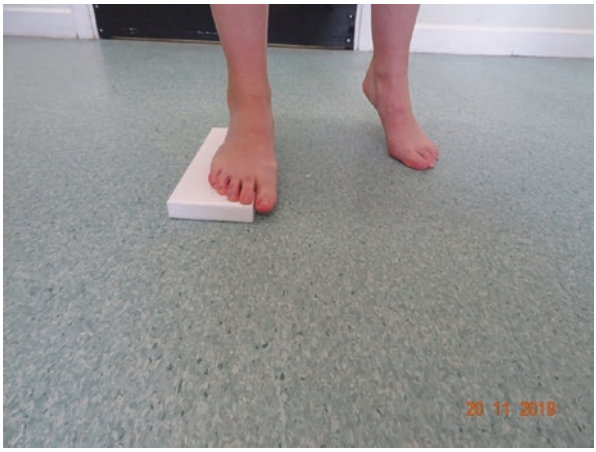

Fig. 26.20 Clinical photograph showing Coleman block test

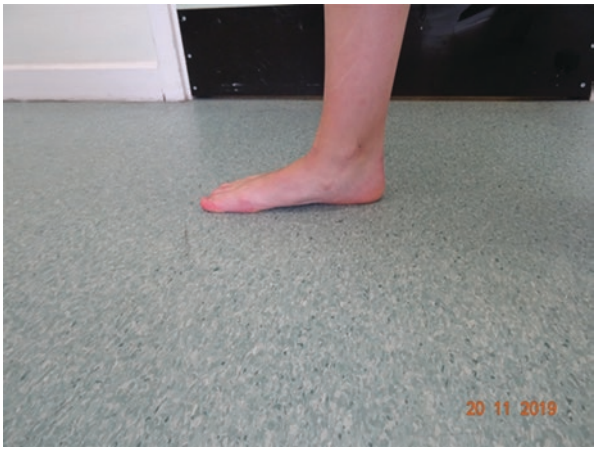

Fig. 26.21 Clinical photograph showing medial longitudinal arch flattening—pes planus

Fig. 26.22 Too many toes—clinical photograph showing the right foot with five toes

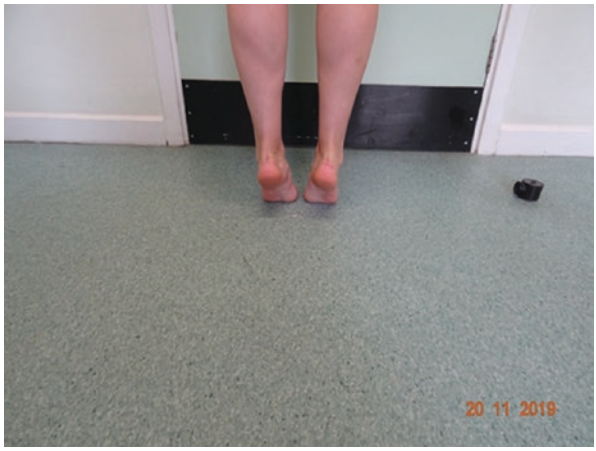

Fig. 26.23 Heel rise—clinical photograph showing the heel levels swing into varus in physiological flat feet

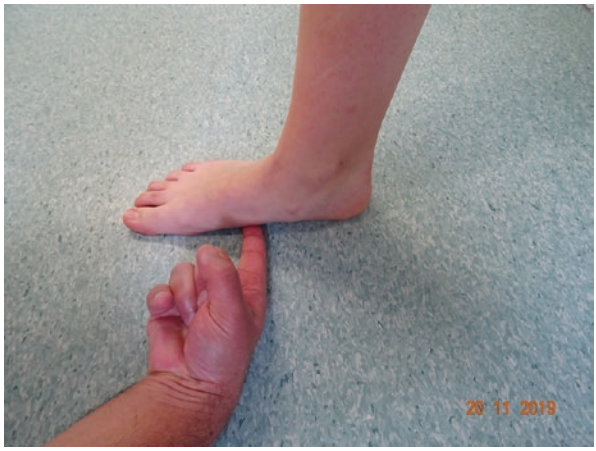

Fig. 26.24 Clinical photograph showing Jack's test which demonstrates the inability to pass more than the tip of the index finger under the medial arch of the foot

would mean weak tibialis posterior muscle. **Jack's test**: assess the flexibility of pes planus. With the patient weight bearing, passive dorsiflexion of the big toe MTPJ will result in an increase in the medial longitudinal arch concavity enabling you to insert the distal phalanx of the index finger under the medial arch (Figs. 26.24 and 26.25). Perform **Silfverskiold test** (described above) to look for Achilles tendon tightness.

2. Achilles rupture: Feel for a palpable gap and check for bruising. In chronic ruptures active ankle plantarflexion will be weak and passive ankle dorsiflexion will be increased. With the patient in prone position, assess for increased resting ankle dorsiflexion and perform Thompson test. **Thompson test**: is performed with the feet off the edge of the couch, the calf is squeezed halfway up the leg, and lack of ankle plantarflexion indicated a positive test and rupture [6] (Fig. 26.26).

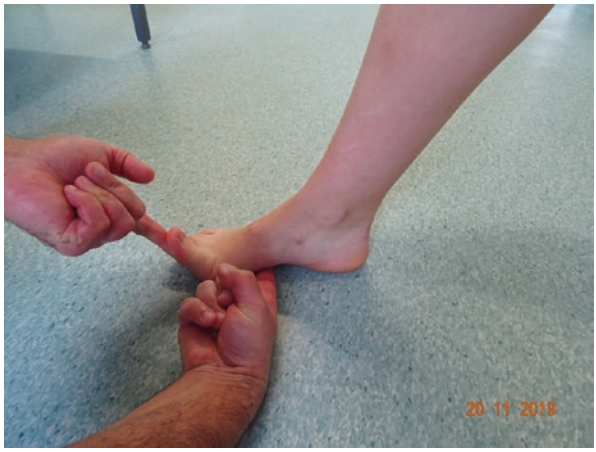

Fig. 26.25 Clinical photograph showing Jack's test by dorsiflexing the big toe the examiner is able to pass distal phalanx under the medial arch of the foot

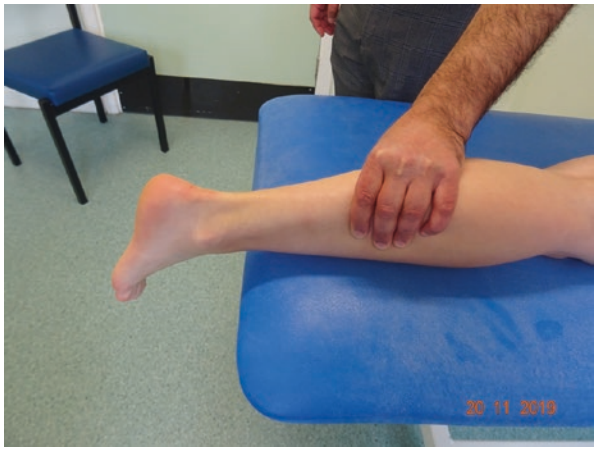

Fig. 26.26 Clinical photograph showing how to elicit the Thompson test

3. Tarsal coalition: Loss of internal rotation of the subtalar joint resulting in rigid movement during the gait cycle. Patients are unable to walk on the outer border of their feet. From the side look for flattening of the medial longitudinal arch. From behind look for hindfoot alignment (valgus) and "too many toes" sign. There is reduction of subtalar joint movement. Jack's test: arch does not reconstitute. Hindfoot does not correct on tiptoeing. Perform Silfverskiold test to look for contracture of the Achilles tendon.

Ankle Instability

Assessment should start by performing the Beighton score to assess for generalized laxity: score of 5 or more out of 9 is considered a sign of generalized laxity. A point is awarded for each of the following: (1) passive dorsiflexion and

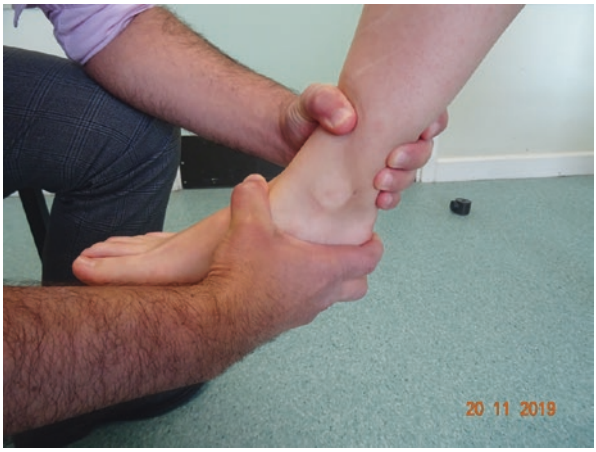

Fig. 26.27 Clinical photograph showing the anterior draw test

hyperextension of each fifth MCP joint beyond 90° (2) passive apposition of each thumb to the flexor aspect of the forearm (3) passive hyperextension of the elbow beyond 10° (4) passive hyperextension of each knee beyond 10° and (5) active forward flexion of the trunk with the knees fully extended so that the palms of the hands rest flat on the floor.

Ankle sprains may be low or high based on injured structures:

1. Low ankle sprain: Look for an antalgic gait, bruising, and swelling. Look for tenderness on the lateral aspect of the ankle at the base of the lateral malleolus. **Anterior draw test** (Fig. 26.27):

   Ask the patient to flex their knee to 90° to relax the gastrocsoleus complex. To test the competency of the ATFL (anterior talofibular ligament), plantarflex the ankle to 10°; grip the distal tibia with one hand to stabilize and grasp the heel with the other hand; while stabilizing the tibia, pull the foot forward and note degree of forward translation. A positive test is when there is no firm end point or increased anterior translation compared to the uninjured side. A dimple sometimes is seen (dimple or suction sign) indicating ATFL compromise. To test the competency of the CFL (calcaneofibular ligament), repeat the test with the ankle in dorsiflexion position. **Talar tilt test**: Ensure the gastrocsoleus complex is relaxed by flexing the knee to 90°, tilt the talus from side to side and compare to the uninjured side. Absence of a firm end point is indicative of a combined injury to ATFL and CFL.
2. High ankle sprain: Look for bruising and swelling. There are multiple tests to assess for a syndesmosis injury. **Cotton's test**: There is excessive translation of the talus from medial to lateral in the ankle mortise. **Crossed-leg test** (Fig. 26.28): Ask the patient to sit with the middle of the injured leg across the top of the opposite knee. Pressure is applied to the medial aspect of the proxi-

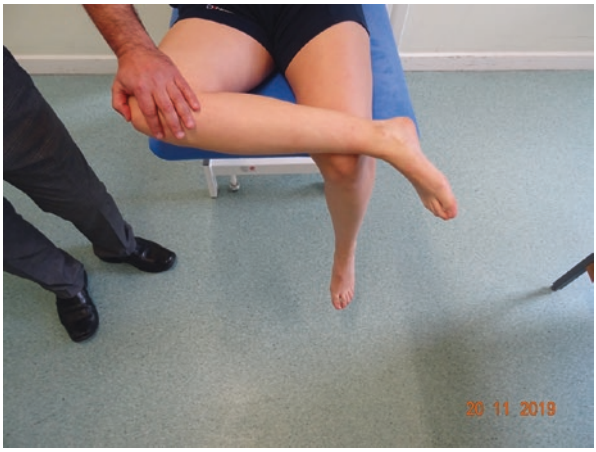

Fig. 26.28 Clinical photograph showing the crossed-leg test

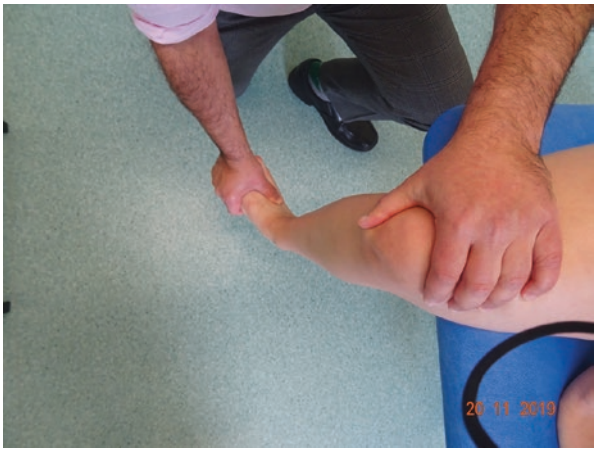

Fig. 26.29 Clinical photograph showing the external rotation test

mal tibia and fibula at or near the knee, to apply shear strain to the distal syndesmosis ligaments. A positive test will result in pain at the ankle. **External rotation test** (Fig. 26.29): Ask the patient to sit at the edge of the couch with the knee and hip flexed to 90°. With one hand stabilize the leg halfway down the shin and with the other hand externally rotate the ankle. A positive test will result in pain at the ankle. **Fibula translation** (Fig. 26.30): With the patient in supine position, hold the fibula with the thumb and index finger while translating anteriorly and posteriorly in the sagittal plane. **Squeeze (Hopkin's) test**: with the patient supine, squeeze the middle of the lower leg between the palms of both hands (Fig. 26.31).

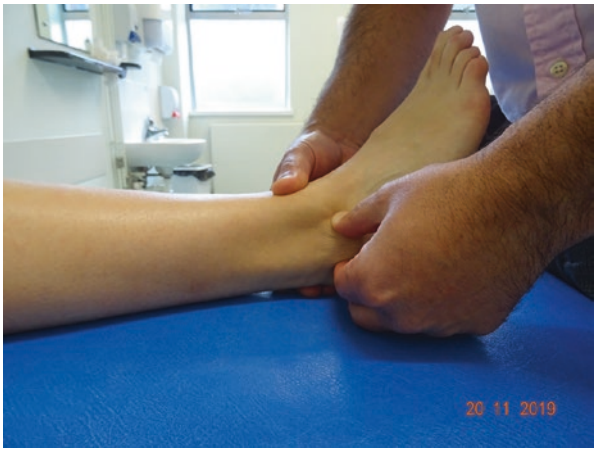

Fig. 26.30 Clinical photograph showing fibular translation

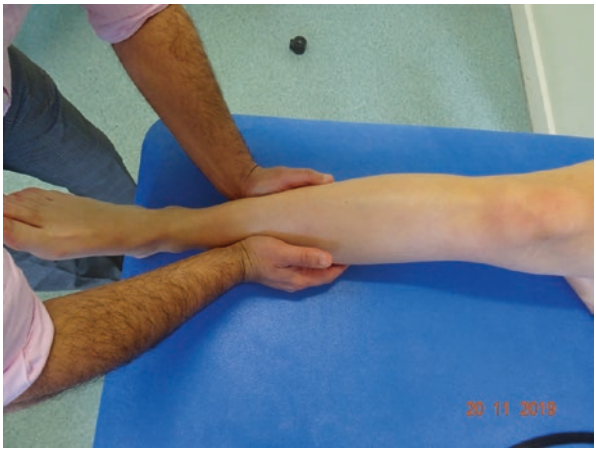

Fig. 26.31 Clinical photograph showing squeeze (Hopkin's) test

Systemic Conditions

1. Diabetic neuropathy: It is of vital importance to thoroughly assess the neurovascular status of the foot and look for ulcerations. In the presence of an ulcer, you must note any clinical signs of infection and depth. Look for asymmetrical deformities suggestive of Charcot arthropathy.
2. Rheumatoid arthritis: Start the examination from the ankle first and proceed distally. Assess skin condition for fragility and presence of rheumatoid nodules. Look for symmetrical deformities, hindfoot alignment (valgus) and forefoot pronation and abduction. Medial and lateral malleoli swelling secondary to tenosynovitis of the tibialis posterior and peroneal tendons. Note the presence of hallux valgus and lesser ray deformities, e.g., hammer or claw toes and any callosities over the joints or on the plantar aspect. Assess movement of the ankle and subtalar joints noting any restrictions.

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Modern Management of Ankle Arthritis Including Primary Ankle Replacement

27

Maneesh Bhatia and Randeep Singh Aujla

Introduction

Etiology

It is said that 1% of the world's population is affected by ankle osteoarthritis (OA) [1]. Its effect on health-related outcomes is at least as severe as hip arthritis [2]. However, it is nine times less prevalent than hip and knee OA [3]. In a study of 864 participants (68% female, 34% African American, mean age 72, mean BMI 31), there was found to be a 7% incidence of radiographic evidence of ankle arthritis [4].

The majority of ankle OA is secondary to trauma [5]: 1.6% of hip and 9.8% of knee OA are post-traumatic in nature, whereas ankle OA is secondary to trauma in 79.5% of cases [6]. Traumatic injuries that may lead to ankle OA include fractures of the malleoli/tibial plafond/talus, osteochondral injuries of the talar dome, and ankle ligamentous injuries.

The reasons why ankle trauma has such a profound effect on causing ankle OA has good biomechanical evidence. The ankle has a relatively low contact area for the given load across it. The ankle has a contact area of around 350 mm^2 as compared to 1100 mm^2 and 1120 mm^2 for the hip and knee, respectively [7]. Also the articular cartilage thickness is lower, at 1–2 mm, compared with the knee at 1–6 mm [8]. In vitro models have shown that a step defect in articular cartilage of the ankle can increase contact stress by 30%, and up to 60%, in the presence of steps and instability [9].

In distinction to the hip and knee, primary OA has a much lower incidence in the ankle. Primary OA accounts for around 90% of cases of hip and 98% of knee

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OA, whereas primary ankle OA only makes up around 7% of cases. Other etiologies for ankle degeneration include inflammatory arthritis in 12% of cases [10]. Rarer causes of secondary arthritis of the ankle include hemochromatosis, hemophilia, gout, neuropathic diseases, avascular necrosis of the talus, and postinfective arthritis.

Clinical Presentation

Patients with ankle arthritis present with pain, diminished quality of life, and limited physical function as severe as patients with hip arthritis [2]. They may also describe stiffness or loss of range of motion. Swelling may be noted, particularly in inflammatory arthritis. As arthritis becomes more advanced, there is increasing pain at rest and during the night. This may awaken patients from their sleep. In more advanced cases, deformity may be noted by the patient.

Clinical inspection can demonstrate valgus or varus hindfoot position that is often fixed or only partially correctable. Tenderness may be felt at the tibiotalar joint line with encroaching osteophytes. Tibiotalar range of motion would be reduced in dorsi- and plantarflexion. Gait assessment is an essential part of the examination. Care should be taken to assess footwear and insoles and look for walking aids. Neurovascular status should always be assessed prior to embarking of surgery to the foot and ankle.

Classifications

Takakura Classification [11]

The Takakura classification (Table 27.1 and Fig. 27.1) was described in 1986 and developed in 1995. It was later modified in 2006 by Tanaka et al. [12].

Modified Cedell Classification [13]

Cheng et al. (2001) modified a classification (Table 27.2) from 1965 created by Cedell et al. [14].

Table 27.1 Modified Takakura classification

Stage I	Early sclerosis and osteophyte formation, no joint space narrowing
Stage II	Narrowing of medial joint space (no subchondral bone contact)
Stage IIIa	Obliteration of joint space at the medial malleolus, with subchondral bone contact
Stage IIIb	Obliteration of joint space over roof of talar dome, with subchondral bone contact
Stage IV	Obliteration of joint space with complete bone contact

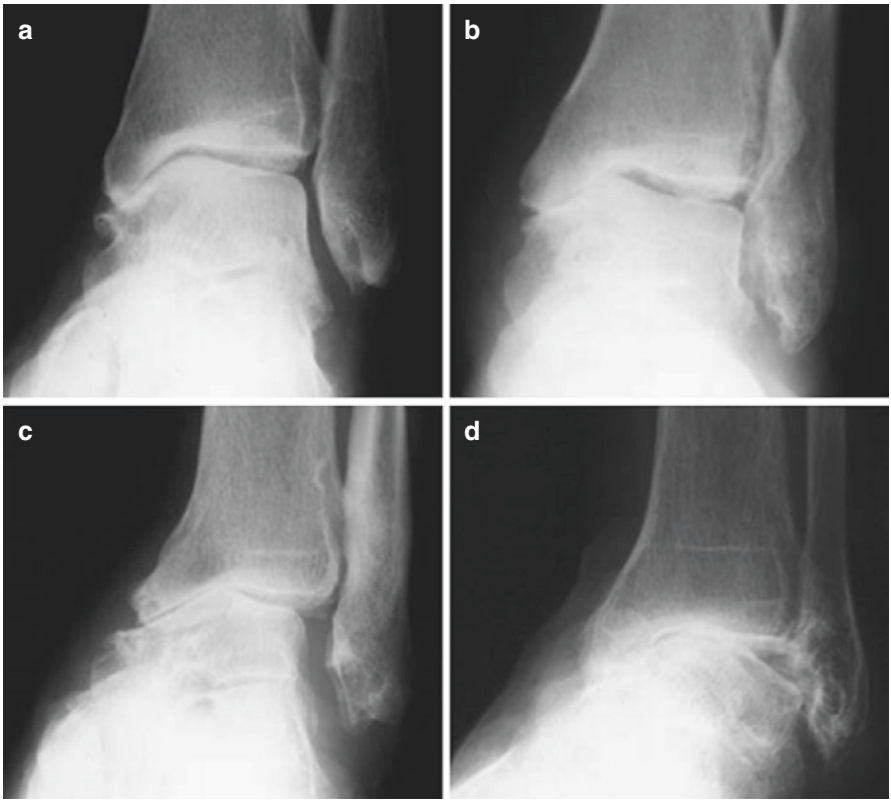

Fig. 27.1 Modified Takakura classification [11]

Table 27.2 Redrawn. Modified Cedell classification [Cedell C-A. 11 Outward Rotation-Supination Injuries of the Ankle. Clin Orthop Relat Res 1976–2007. 1965;42:97–42:9

Stage 0	Normal alignment	No reduction of the joint space
Stage 1	Normal alignment	1. Slight reduction of the joint space
		2. Slight formation of deposits at the joint margins
Stage 2	Mild malalignment	1. More pronounced change than mentioned above
		2. Subchondral osseous sclerotic configuration
Stage 3	Obvious varus/valgus alignment	1. Joint space reduced to about half the height of the uninjured side
		2. Rather pronounced formation of deposits
Stage 4	Joint space has completely or practically disappeared	

Table 27.3 The COFAS classification for end-stage ankle arthritis

Type 1	Isolated ankle arthritis
Type 2	Ankle arthritis with intra-articular varus or valgus deformity or a tight heel cord or both
Type 3	Ankle arthritis with hindfoot deformity, tibial malunion, midfoot ab- or adductus, supinated midfoot, plantarflexed first ray, etc.
Type 4	Types 1–3 plus subtalar, calcaneocuboid, or talonavicular arthritis

Canadian Orthopaedic Foot and Ankle Society Classification for End-Stage Ankle Arthritis [15].

A Canadian group of fellowship-trained foot and ankle surgeons developed a scoring system containing clinical and radiological elements. The Canadian Orthopaedic Foot and Ankle Society (COFAS) classification (Table 27.3) has a good inter- and intra-observer reliability (kappa = 0.62 and 0.72) [15]. The COFAS classification includes adjacent soft tissue and joint/bony issues.

Nonsurgical Treatment

Lifestyle Modification

Initial treatment of ankle OA should involve lifestyle modification. This involves weight loss, which we know plays a large role in off-loading the ankle joint and alleviating pain [16]. It has been shown that there is a fourfold reduction in load exerted through the knee for each kilogram that is lost [17]. A similar effect at the ankle can be extrapolated. Along with weight loss, minimizing activities that aggravate the symptoms and avoiding high-impact activities can help relieve pain. Physical therapy can be used to increase flexibility and range of motion while strengthening surrounding musculature. Using a walking aid will also help off-load the affected ankle joint by 11–25% [18, 19].

Bracing, Orthotics, and Footwear

The goal of physical aids, such as braces and orthotics, in relieving pain is achieved by maintaining talar alignment and limiting tibiotalar movement during gait and activities.

Bracing can alleviate pain, improve quality of life, and prolong the time until ankle arthrodesis is needed in patients [20]. Braces range from over-the-counter lace-up-type ankle braces to custom-molded orthosis. The Arizona Brace (Fig. 27.2) is a lace-up leather brace which is popular. It provides a comfortable support without the restrictions, and complications, of harder materials such as plastic.

Fig. 27.2 Arizona Brace®

An ankle-foot orthosis (AFO) can also limit ankle movement to improve pain [21]. The use of custom-made carbon fiber energy storing AFOs has shown improved function in military personnel [22].

Modification of footwear can be utilized to improve pain. In particular a rocker sole (Fig. 27.3) has been found to decrease ankle motion and aid the transition from heel strike to push-off [23].

Overall these methods have advantages of being cheap, easy to use and have a low risk of complications. However, they do not treat the underlying pathology, is dependent upon patient compliance, and can cause pressure sores from continued use.

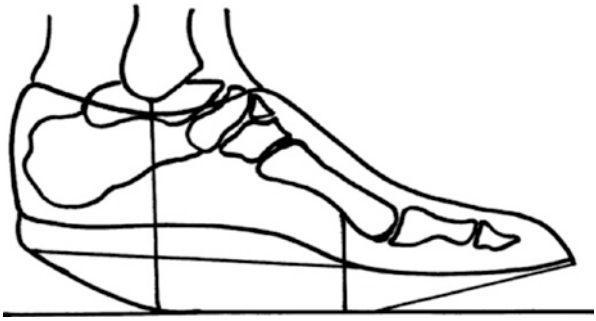

Fig. 27.3 Diagram of a rocker bottom sole shoe

Injection Therapy

Literature around injection therapy for ankle arthritis is generally limited. Options include corticosteroid, visco-supplementation, or platelet-rich plasma injections.

Corticosteroid

Corticosteroids have an anti-inflammatory and immunosuppressive effect. The manner in which this is affected is complex. Within joints they reduce vascular permeability and synovial blood flow that subsequently releases inflammatory modulators. It also increases the concentration of hyaluronic acid within the joint [24]. This ultimately leads to pain relief.

Corticosteroids are generally safe with low complication rates. The most common complication is a postinjection steroid flare, which can occur in 2–10% of cases. Systemic side effects include vasovagal reaction, transient hyperglycemia, and facial flushing. Rare complications are fat atrophy and skin depigmentation. Very unusual, but significant, risks include septic arthritis (0.03%) and tendon rupture.

A retrospective review of 365 patients demonstrated that 86% of patients reported a significant improvement in symptoms from foot and ankle conditions [25].

Overall in ankle arthritis, corticosteroids are used as an adjunct to allow a period of pain relief in order to facilitate other good practices such as weight loss and physiotherapy.

Hyaluronic Acid Injection

Hyaluronic acid is a naturally occurring polymer. It aims to restore viscoelasticity through the replacement of dysfunctional synovial fluid and provide analgesia through its effect on early inflammation [26, 27]. It has been shown to be chondroprotective by aiding in the synthesis of proteoglycans and glycosaminoglycans. It is an established treatment for osteoarthritis in other joints such as the knee [28–31].

A recent meta-analysis evaluated the effect of hyaluronic acid injections for ankle osteoarthritis [32]. They reviewed 9 studies inclusive of 354 participants. They found that hyaluronic acid can significantly reduce pain from ankle arthritis

and it is superior to other reference treatments (saline injection, exercise therapy, and arthroscopy). A 2015 Cochrane review concluded that there was unclear benefit or harm for the use of hyaluronic acid for ankle osteoarthritis compared to placebo at 6 months based on the low-quality evidence it reviewed [33].

Platelet-Rich Plasma

Platelet-rich plasma (PRP) is made up of the noncellular component of blood. The aim is to have a platelet concentration that is 3–5× higher than baseline. Platelets are a large part of hemostasis, inflammatory modulation, and tissue repair. This has been demonstrated in animal and in vitro studies [34].

Complications involve all the potential dangers of any injection; the risk of infection, bleeding, and damage to surrounding structures.

Clinical studies have shown a small improvement in symptoms which can last over 12 months [35–37].

Surgical Treatment

Chondral Regeneration Techniques

Fresh Osteochondral Allograft

Fresh osteochondral allograft uses transplant techniques to remove large areas of chondral damage and replace with donated tissue. Grafts will usually need to be implanted between 4 and 6 weeks postharvesting. This requires an open procedure and the allograft will need to be fixed in situ. A period of immobilization is required to protect the healing process.

It is indicated in young patients with large chondral defects who are deemed too young for ankle arthroplasty, have an excellent range of motion, low body mass index, and normal radiographic alignment and who refuse arthrodesis [38].

Fresh osteochondral total ankle allograft transplantation has been reported to have survival rates of between 50% and 92% at 1- to 12-year follow-up [39]. The most recent study containing 86 patients had a 10-year survival of 56% [40].

It is a technically demanding procedure that requires access to fresh allograft that can be processed in a timely manner. Specific complications include nonunion, disease transmission, arthrofibrosis, and failure. Subsequent revision surgery to arthrodesis or arthroplasty has a higher revision and reoperation rate than primary procedures [41].

Microfracture

Microfracture relies on marrow stimulation in the treatment of chondral lesions in joints. The procedure is conducted by debriding articular cartilage to create a stable shoulder. Following this the subchondral bone is perforated with a drill or microfracture pick to allow the extrusion bone marrow mesenchymal stem cells and cytokines to form a fibrin clot. This fibrin clot is maintained at the base of the chondral defect and will ultimately become fibrocartilage.

Studies have reported positive outcomes in small cohorts, with improvements in function and satisfaction rates [42, 43]. This is supported with radiological evidence that the defect is filled with tissue similar to articular cartilage [44].

Autologous Chondrocyte Implantation

Autologous chondrocyte implantation (ACI) is the use of cultured host chondrocytes being reimplanted within a scaffold. This technique has been shown to have good outcomes for osteochondral lesions with a size less than 1.5 cm^2 [45–47]. However, its use is not advocated in degenerative disease and should be reserved for focal lesions.

Arthroscopic Surgery

Arthroscopic debridement of the ankle joint aims to treat unstable chondral defects, synovitis and remove fibrous tissue/loose bodies. Therefore, it is useful only in early ankle arthritis. It can also be used to treat concurrent issues such as anterior/posterior impingement.

It results in improvements in pain and swelling via a minimally invasive method that can be followed by aggressive uninhibited rehabilitation. Short postoperative recovery time is required.

Arthroscopic ankle debridement can provide short-term pain relief. A 5-year follow-up study demonstrates that this is most effective in patients with ankle impingement [48]. However, 28% of cases required a major procedure at 5-year follow-up.

Despite this there is trepidation about performing arthroscopic debridement for degenerative conditions as seen over the last decade with the knee. As a result, arthroscopic debridement of the knee as a procedure for degenerative disease has dwindled based on literature. There are such studies for the ankle also. Martin et al. demonstrated a 12% good/excellent result for ankle arthroscopy for degenerative disease [49]. However, this study which is from 1989 included only eight patients with degenerative joints. A further critical literature review in 2009 found poor evidence (grade C against) for the use of ankle arthroscopy for arthritis [50].

Osteotomy

Joint preservation has become a key element of modern orthopedic surgery as an increasing number of younger patients seek treatment for degenerative conditions. As 63% of patients with severe ankle arthritis have a malalignment of the hindfoot, realignment osteotomy has become an important option for these patients [51]. It improves symptoms by redistributing load away from degenerative areas of articular cartilage and onto intact areas. This has even been shown to stimulate articular cartilage regeneration [52–54].

Osteotomy can be used to treat post-traumatic deformity and asymmetric degenerative conditions. Post-traumatic deformity is corrected at the center of rotation angle (CORA), which should be assessed by thorough preoperative planning. Takakura et al. first described distal tibial osteotomy to treat varus ankle arthritis in 1995 [11]. It aimed to treat patients with intermediate ankle arthritis defined at stages 2, 3a, and 3b according to the modified Takakura system (Table 27.1).

Contraindications include neuropathic conditions, such as Charcot ankle, and patients with peripheral neuropathy. Factors that can affect wound healing can also contraindicate osteotomy, including peripheral vascular disease, tobacco use, scars, and local infections. Poor bone quality can also be a relative contraindication. This can be caused by advanced age, medications, osteopenia, and rheumatological conditions.

Traditionally extra-articular, or supra-malleolar, osteotomy aimed to improve overall limb mechanical alignment in varus ankle arthritis. It does this by creating a distal tibial medial opening wedge osteotomy to create a valgus mechanical alignment. This can be either horizontal or oblique in nature (Fig. 27.4).

The addition of a fibular osteotomy is controversial, but recent studies suggest it is not essential [55].

An unpublished systematic review and meta-analysis demonstrated that distal tibial osteotomy, for varus ankle OA, can improve pain visual analogue scores by 4.5 points and American Orthopaedic Foot and Ankle Score (AOFAS) improves by 25 points. These results included 242 osteotomies in 235 patients with a mean follow-up of 45 months.

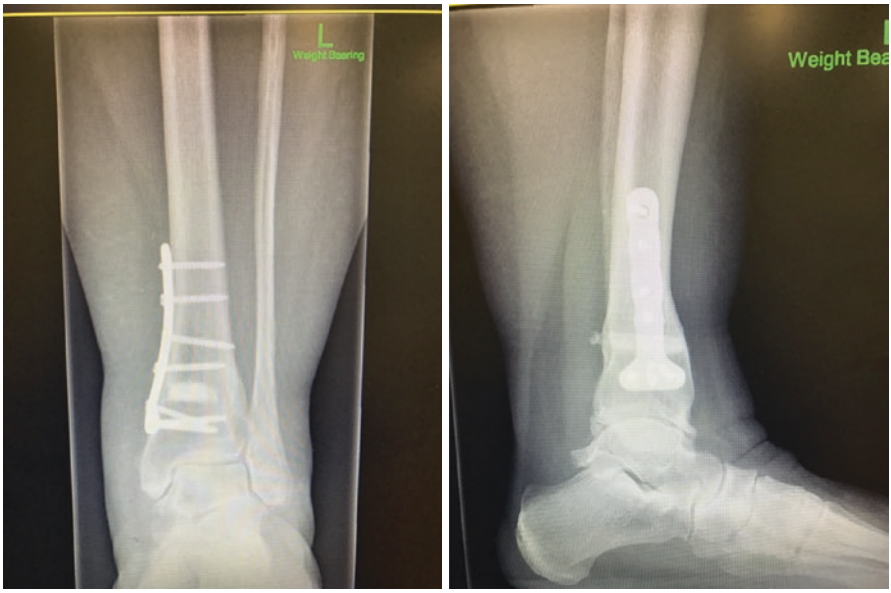

Fig. 27.4 AP and lateral postoperative radiographs following horizontal distal tibial osteotomy

Table 27.4 Advantages and disadvantages for distal tibial osteotomy for ankle arthritis

Advantages	Disadvantages
Native joint preservation	Disease progression can make future surgery more complex
Correct malalignment	Protected/non-weight-bearing postoperative period
Can be used post-trauma	Concurrent procedures such as calcaneal osteotomy or ligament reconstructions may be needed
Maintained range of motion	High implant removal rate
Can treat instability symptoms without ligament reconstruction	

Immediate postoperative complications include infection, venous thromboembolism, delayed/nonunion, stiffness, metalwork irritation, sub-fibular pain due to overcorrection, loss of correction leading to persistent symptoms, and progression of arthritis (Table 27.4).

Distraction Arthroplasty

Distraction arthroplasty is indicated for young patients with ankle arthritis. The theory behind its use is that by applying an off-loading force to articular cartilage that it can repair and regenerate. Generally, this is achieved by applying a ringed external fixator for a period of 8–12 weeks.

The exact physiology is not yet fully understood. Animal studies have shown that distraction will reduce mechanical force on articular cartilage. As a result, chondrocytes intra-articular blood flow improves and greater nutrition is found. This provides the theory extrapolated to a human population.

Complications include pin site infections, traction nerve injury, stiffness, and failure.

Smith et al. in a 2012 review stated that there was "inadequate evidence based literature" [56]. More recent studies have been more supportive [57]. An international randomized controlled trial in 2019 comparing distraction arthroplasty to debridement alone found that both treatments improved functional outcomes with a higher further surgery rate in the debridement group (51% vs. 26% at 3 years) [58]. Ikuta et al. performed distraction arthroplasty and microfracture in seven patients with only one being converted to fusion by 4.2-year follow-up [59].

Arthrodesis

Ankle arthrodesis has long been the gold standard surgical treatment of end-stage ankle arthritis. The goal of ankle arthrodesis is to provide a pain-free plantigrade foot for weight-bearing activities [60]. Optimal position is neutral dorsiflexion, 5–10° external rotation, 0–5° hindfoot valgus, and 5 mm of posterior talar translation [61].

Ankle arthrodesis can be used in a variety of pathologies including osteoarthritis, inflammatory arthritis, post-traumatic arthritis, gout, postinfective arthritis, Charcot

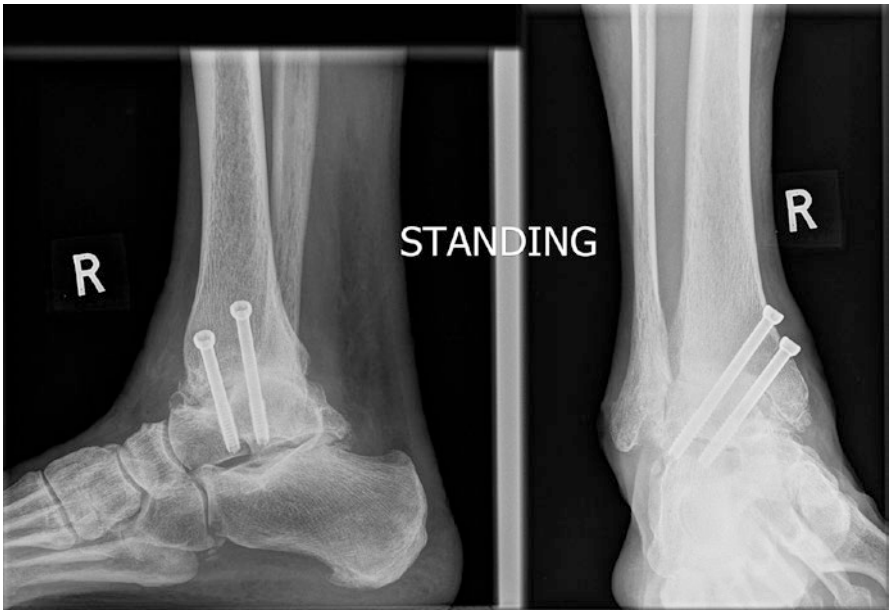

Fig. 27.5 Postoperative radiographs of arthroscopic ankle arthrodesis

arthropathy, osteonecrosis of the talus, failure of total ankle arthroplasty, and neuromuscular conditions.

General contraindications for ankle arthrodesis are few but include the presence of acute or chronic infection and complex regional pain syndrome.

There are multiple methods for achieving arthrodesis including open or arthroscopic techniques and stabilized with internal fixation (screws, plates, nails) or external fixation (circular frames). Many factors can affect this decision including etiology, patient comorbidities, presence of smoking or poor vascularity, and surgical preference and skills.

Open arthrodesis can be performed via trans-fibular or fibular sparing (anterior) approaches. Once the joint has been exposed, the surfaces are prepared using osteotomes, high-speed burrs, and bone rongeurs. Once prepared the fixation can be performed using screws, a plate, or external fixators. Open arthrodesis techniques are reserved for severe deformity (>15° of varus/valgus coronal plane deformity) in patients with healthy skins and adequate vascularity.

Arthroscopic ankle arthrodesis is a minimally invasive procedure that allows minimal soft tissue dissection, devascularization of bone/skin, and more cosmetically acceptable scars. Arthroscopic arthrodesis is performed via anterolateral and anteromedial portals. The joint is debrided and prepared using a bone burr. Once the joint surfaces are prepared, the fixation is usually performed using percutaneous partially threaded cannulated screws (Fig. 27.5). This method is reserved for patients with mild easily correctable deformities. It has benefits for patients with abnormal/poor wound healing or scar tissue present from previous injury. Contraindications to arthroscopic arthrodesis include severe deformities, large bone defects that need an

open approach to graft, previously failed arthrodesis, and neuropathic disorders such as Charcot arthropathy.

No randomized controlled trial has compared open versus arthroscopic techniques. A systematic review highlights the advantages of arthroscopic surgery to be better clinical scores, fewer complications, shorter hospital stay, and less blood loss when compared to open surgery [62]. Union rates between open and arthroscopic techniques have been found to be comparable [63, 64].

Other general limitations of arthrodesis, of either technique, include difficulty in walking on uneven surfaces [65] and aching around the foot and ankle with prolonged walking or standing. Functionally patients demonstrate reduced cadence and stride length with diminished range of motion of the hindfoot and midfoot during walking [66].

Arthrodesis has a lower complication and reoperation rate when compared to arthroplasty [67, 68].

Nonunion occurs in approximately 12% (range; 3–23%) of foot and ankle fusions [69]. Nonunion is more common in patients who have previously had sub-talar arthrodesis and patients with varus deformities (Table 27.5) [70].

It has always been postulated that ankle arthrodesis will lead to adjacent joint arthritis due to altered biomechanics of the foot and ankle. Prevalence of subtalar joint arthritis ranges between 24 and 100%. The prevalence of talonavicular and calcaneocuboid arthritis ranged between 18 and 77%. Overall there is no true consensus in the literature as to the effects of ankle arthrodesis on biomechanics and impact on adjacent joint disease [72] (Table 27.6).

Table 27.5 Risk factors for nonunion in foot and ankle fusion surgery [71]

Risk factor		Grade of evidence
Patient factors	Smoking	B
	Diabetes	B
	Soft tissue envelope	B
	Nonsteroidal anti-inflammatory drugs	C
	Compliance with weight bearing	C
	Obesity	I
	Osteoporosis	I
	Alcohol	I
	Age	I
	Vascularity	I
	Local infection	I
Surgical factors	Construct stability	I
	Interfragmentary gaps	I
	Surgeon volume: High vs. low	I

Grade A indicates good clinical evidence from at least one randomized controlled trial and other good-quality studies. Grade B indicates fair clinical evidence from well-conducted clinical studies but no randomized controlled trials. Grade C indicates conflicting clinical evidence. Grade I indicates insufficient clinical evidence

Table 27.6 Advantages and disadvantages for arthrodesis for ankle arthritis [73]

Advantages	Disadvantages
Predictable outcome	Protected/non-weight-bearing postoperative period
Deformity correction	Nonunion risk approximately 10%
Close to normal gait if isolated tibiotalar disease	Potential adjacent joint degeneration
Infection uncommon and easy to treat if it does occur	
Cheap	
No risk of instability	

Total Ankle Arthroplasty

History

The first documented attempt at tibiotalar joint replacement was performed in 1962 by Carrol Larson. He utilized a "cup arthroplasty" design, inspired by Smith-Petersen, using a vitallium talar dome resurfacing [74]. In 1970 Lord and Morrotte inserted 25 total ankle replacements using a reverse hip design with a stem implanted in the tibia and a cemented acetabular cup into the calcaneum, after the talus was excised [75]. A two-component cemented design was introduced at Imperial College of London Hospital between 1972 and 1981 [76]. Long-term satisfaction was obtained in only 11 out of 62 ankles. These first-generation prostheses were very constrained and required extensive bony resection. They frequently failed due to osteolysis and loosening. Early constrained and unconstrained prosthesis continued to have poor results with a high conversion to arthrodesis [77–79]. This has been found in 72% at 10 years [80].

Prosthesis design developed through the 1980s with the use of fixed and mobile bearings. These second-generation prostheses required less bony resection and had less constraint.

Third-generation designs (Fig. 27.4) were semi-constraint made of three components. This allowed for a mobile-bearing polyethylene insert. Cobalt chromium alloy tibial and talar components can be cemented or uncemented.

Indications

The classic teaching has been for ankle arthroplasty to be used for patients who were elderly, low-demand, and with inflammatory arthroplasty [81]. They would ideally be thin and nonsmokers and have a retained ankle range of motion with minimal deformity. However, demand in younger and more active patient will increase as failure rates fall. Also post-traumatic arthritis is becoming a leading indication as surgeons push the boundaries of ankle arthroplasty [82].

Absolute contraindications:
- Heavy smoker
- Uncontrolled diabetes
- Peripheral neuropathy
- Peripheral vascular disease
- Significant bone loss
- Osteonecrosis/avascular necrosis
- Active or previous infection

Relative contraindications:
- Age younger than 50
- Angular deformity >15°
- Obesity
- Heavy industrial laborer
- Sporting aspirations

Modern Implants

Modern designs include:

Outcomes

A recent systematic review of modern prosthetic design identified a failure (revision, arthrodesis, or amputation) rate of 9.8% at 5 years [82]. However, survivorship does vary depending upon implant (Tables 27.7 and 27.8).

Table 27.7 Varieties of modern implants

Brand	Manufacturer	Components	Bearing
Infinity™	Wright Medical (Arlington, TX)	Two	Fixed UHMWPE
Topaz/INBONE/ INBONE II	Wright Medical (Arlington, TX)	Two	Fixed UHMWPE
TNK	Nara (Kyoto, Japan)	Two	Fixed Ceramic
Buechel-Pappas™	Endotec (South Orange, NJ)	Three	Mobile UHMWPE
HINTEGRA®	Newdeal (Lyon, France)	Three	Mobile UHMWPE
STAR	WaldEmar Link (Hamburg, Germany)	Three	Mobile UHMWPE

Table 27.8 Survivorship of implants

Prosthesis	Failure rate	Survivorship analysis pooled data
Infinity™	3–10%	95% at 2 years
TNK	4%	80% at 10 years (95% CI; 71–90%)
Buechel-Pappas™	0–8%	92% at 12 years (95% CI; 89–95%)
HINTEGRA®	9%	84% at 10 years
STAR	4–24%	89% at 5 years (95% CI; 74–99%)

The Infinity (Wright Medical (Arlington, TX)) (Fig. 27.6) have shown to have a 3–10% early revision rate [83, 84]. However, it is a widely used prosthesis in the United States of America and the United Kingdom.

The HINTEGRA ankle replacement (Fig. 27.7) has a 10-year survivorship of 84% [85].

The Scandinavian Total Ankle Replacement (STAR) is into its fourth generation and remains a highly popular ankle replacement implant (Fig. 27.8). It consists of a three-piece mobile-bearing prosthesis. Implant survival is 73–76% at 15 years; however more than half needed additional surgical procedures [86, 87].

Overall it is felt that an ankle arthroplasty would be a more cost-effective alternative to arthrodesis in a 60-year-old cohort with end-stage ankle arthritis [88].

Complications

Failures can result in revision surgery (62%), arthrodesis (36%), or amputation (1%). Salvage arthrodesis can expect a 90% union rate with a lower reoperation rate when compared to revision arthroplasty [89].

Superficial wound healing complications occur in 8% of cases with a deep infection rate of 0.8%. Residual pain is present in between 23% and 60% of patients.

Overall satisfaction is between 79–97% [90, 91]. Sixteen patients who underwent total ankle replacement on one ankle and arthrodesis on the contralateral limb had similar satisfaction with both procedures [92].

The presence of osteolysis and cysts on postoperative radiographs is common. One study noted a rate of 86% with only 14% developing component migration or subsidence.

Other complications include progressive arthritis in adjacent joints, component malalignment, fracture, venous thromboembolism, heterotrophic ossification, or syndesmotic nonunion (Table 27.9).

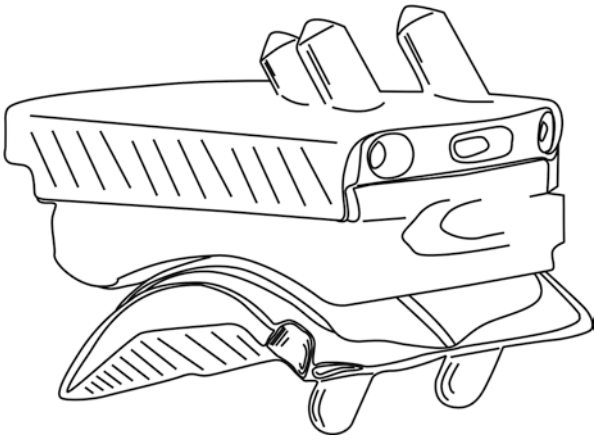

Fig. 27.6 Line diagram showing the Infinity Total Ankle System (marketed by Wright Medical; Arlington, TX)

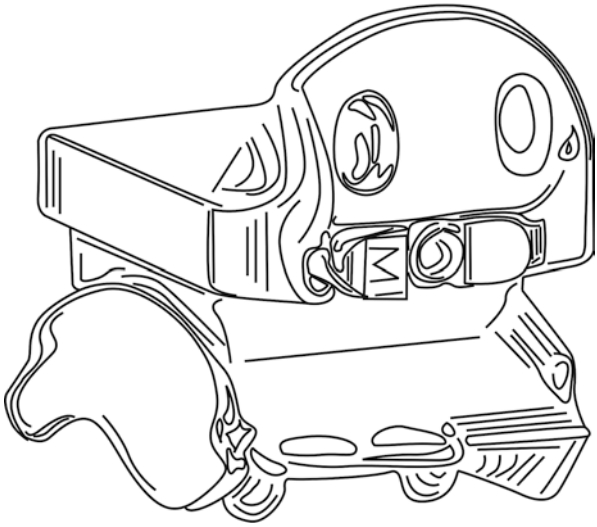

Fig. 27.7 Line diagram showing the HINTEGRA ankle replacement (marketed by Newdeal; Lyon, France)

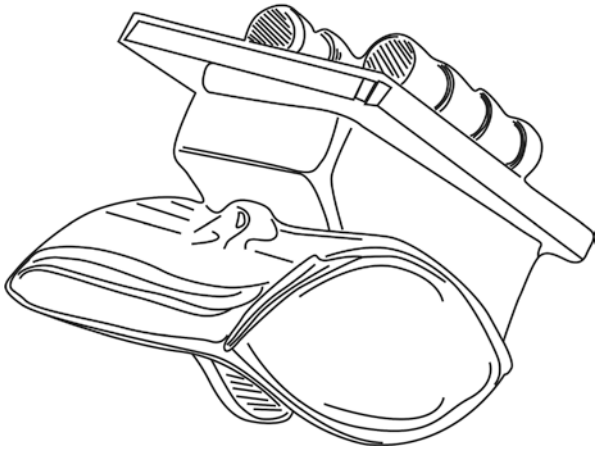

Fig. 27.8 Line diagram showing the STAR (marketed by Stryker/ Waldemar Link; Hamburg, Germany)

Table 27.9 Advantages and disadvantages for arthroplasty for ankle arthritis [73]

Advantages	Disadvantages
Slight improvements in range of motion	10% failure at 5 years
Immediate full-weight bearing	Loss of bone stock for future surgery
Reduced risk of adjacent joint disease	No standardized implant design/philosophy established yet
Improved mobility on uneven surfaces and stairs	Deep infection (0.8%) can be catastrophic

Take-Home Messages

- Ankle arthritis is predominantly post-traumatic in nature with less than 10% of cases occurring primarily.
- Nonsurgical treatment options include weight loss, lifestyle changes, footwear modification, braces, and injection therapies.
- Joint-preserving techniques involve cartilage regeneration, arthroscopic debridement, distal tibial osteotomy, and distraction osteogenesis.
- Ankle arthrodesis remains the gold standard, but with fourth-generation total ankle replacements, results are improving and hence indications are expanding.

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The Ankle

28

Luckshmana Jeyaseelan and Kesavan Sri-Ram

Ankle Fractures

Introduction

Over five million ankle injuries occur in the United States each year, with an ankle fracture incidence of approximately 187 per 100,000 people per year [1]. As well as the obvious bony involvement, syndesmotic stability and deltoid ligament incompetence should be considered. The most common underlying mechanism of injury is rotation. Anatomic reduction is essential as only 1 mm of lateral talar shift results in a 42% decrease in tibiotalar contact area [2].

Classification

The English surgeon, Percivall Pott, is thought to have first described ankle fractures according to their malleolar involvement, namely, uni-, bi-, and trimalleolar. This description continues to be useful to describe the overall extent of the injury.

The Danis-Weber Classification refers to the fibula fracture alone and its relative position to the syndesmosis.

- Weber A: Below the level of the syndesmosis
- Weber B: At the level of the syndesmosis
- Weber C: Above the level of the syndesmosis

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The Weber classification, as it is more commonly referred to, is a simple description of the fibula fracture but does not always reliably predict syndesmotic injury or describe injury elsewhere in the ankle.

The Lauge-Hansen classification is a detailed description of sequential injury of structures around the ankle that leads to four classical injury patterns. These are named such they refer to the position of the foot, followed by the foot's motion relative to the leg.

Supination Adduction (SA)
1. Transverse distal fibula fracture
2. Vertical medial malleolar fracture

Supination External Rotation (SER)
1. Anterior inferior tibiofibular ligament sprain
2. Spiral or short oblique fibula fracture (anteroinferior to posterosuperior)
3. Posterior tibiofibular ligament rupture or avulsion of posterior malleolus fracture
4. Medial malleolus transverse fracture or disruption of deltoid ligament

Pronation Abduction (PAD)
1. Transverse fracture of medial malleolus OR deltoid ligament injury
2. Anterior inferior tibiofibular ligament injury
3. Comminuted/transverse fibular fracture (proximal to tibial plafond)

Pronation External Rotation (PER)
1. Fracture of medial malleolus OR deltoid ligament injury
2. Anterior inferior tibiofibular ligament injury
3. Spiral (or oblique) fracture of the fibula (aspect proximal to tibial plafond)
4. Posterior inferior tibiofibular ligament injury OR posterior malleolus avulsion fracture

Although not initially included in the original classification, a fifth type of injury is recognized as pronation dorsiflexion. The talus is driven upward with axial loading and initiates the sequence below. Pilon fractures are a pronation dorsiflexion variant.

Pronation Dorsiflexion
1. Fracture of the medial malleolus
2. Fracture of the anterior lip of the tibia
3. Fracture of the supramalleolar fibula
4. Rupture of posterior inferior tibiofibular ligament or posterior malleolus fracture

Haraguchi Classification (Posterior Malleolar Fracture)

This describes three types of fracture, originally described by Haraguchi in a CT study of 57 patients [3].

- Type 1: The most common; is a single posterolateral fragment (67%).
- Type 2: There is extension to the posteromedial side of the distal tibia (19%). There may be more than one fragment.
- Type 3: Fractures are a thin shell of bone (14%).

Eponymous Ankle Fractures

A Bosworth fracture-dislocation is an eponym for rare ankle injury in which the proximal fibular fragment is entrapped (fixed displacement) behind the tibia, frequently irreducible by closed methods.

A Maisonneuve fracture is a spiral fracture of the upper third of the fibula associated with a tear of the distal tibiofibular syndesmosis and the interosseous membrane. There is an associated fracture of the medial malleolus or rupture of the deep deltoid ligament.

A Pott fracture is an eponym for fracture of the distal fibula, 2–3 in. proximal to the ankle joint. It is effectively a fracture-dislocation of the ankle, involving a fracture of the fibula and disruption of the deltoid ligaments with an intact tibiofibular ligament resulting in lateral displacement of the talus.

The Tillaux fracture is a fracture of the anterolateral tibial epiphysis commonly seen in adolescents (Salter-Harris III tibial fracture).

Wagstaffe-Le Fort fracture is a vertical avulsion fracture of the anteromedial aspect of the distal fibula due to avulsion of the anterior tibiofibular ligament attachment (ATFL).

History and Examination

A detailed generic history is required to identify comorbidities, which may influence treatment and outcomes, such as diabetes mellitus, and factors that may influence operative management of the injury, e.g., respiratory and cardiac conditions that may affect anesthesia, anticoagulant use, etc.

A focused history/examination includes identifying/examining for:

Open versus closed injury

Exact location of tenderness, e.g., syndesmotic involvement

Neurovascular status

Compartment syndrome

Associated/distracting injuries
Pre-injury baseline including level of mobility and home situation
Associated conditions that may have adverse effect on outcome:

– Diabetes mellitus/peripheral neuropathy/Charcot
– Rheumatoid arthritis and use of steroids/biologics

Examination of the knee joint to ensure no Maisonneuve-type injury.

Imaging

Ottawa Ankle Rules

These were created to help in identifying those patients that require an ankle x-ray in the setting of injury and thus prevent unnecessary radiation exposure.

The rules state x-rays should be taken in those presenting with:

Tenderness of the bone at the posterior edge or tip (within 6 cm) of either the lateral or medial malleolus

Patient unable to bear weight at the time of injury AND on arrival to the emergency department. Weight-bearing is determined by the patient's ability to take four steps.

Plain Radiographs

1. AP/lateral/mortise in all patients
2. External rotation/gravity stress views to assess stability of syndesmosis/competency of deltoid
3. Full-length tibia, or proximal tibia, to rule out Maisonneuve-type fracture

CT scans are useful in fracture-dislocations as they can help identify comminution of fracture fragments, syndesmotic instability, and articular impaction injuries.

Radiographic Measurements/Findings

Syndesmotic Injury

- Decreased tibiofibular overlap/increased clear space
- Increased medial clear space with lateral talar shift

Lateral Malleolus

- Talocrural Angle
  - Measured by bisection of line along the line of the tibial plafond and another line through the tips of the malleoli
  - Normal angle is 79–87°.
  - An increased angle is suggestive of fibula shortening (compare injured and uninjured sides).
- Dime Sign
  - An unbroken curve between the lateral process of the talus and the recess in the distal tip of the lateral malleolus

Treatment

General principles for the management of ankle fractures include:

- Anatomic reduction of the tibiotalar articulation
- Syndesmotic reduction and stabilization
- Conservative treatment reserved for stable, minimally displaced fractures with no talar shift
- Surgical fixation for all unstable fractures with displacement and/or talar shift, including bi- and trimalleolar fractures

Management by Fracture Type

Fibula Fractures

A variety of surgical techniques are commonly used for fixation of fibula fractures. The most frequent fixation takes the form of lag screw fixation with semitubular neutralization plate. In poor bone quality, or very distal fibula fractures, anatomic fibula locking plates are an invaluable resource. In circumstances of poor soft tissue or where open procedures are contraindicated, the intramedullary fibula nail is becoming a more commonly used implant.

Weber A/Subsyndesmotic Fibula Fracture

- The majority of these injuries can be managed nonsurgically.
- Patients can be weight-bearing in a walker boot as pain allows.
- Surgical fixation is reserved for the significantly displaced fractures to prevent sequelae of malunion/nonunion.

Weber B Fractures

- Up to 40% of Weber B fractures will demonstrate syndesmotic injury (essentially differentiating a Lauge-Hansen SER II and SER II).
- Weight-bearing radiographs have been shown to be extremely valuable in assessing syndesmotic stability in these injuries [4].
- In those fractures deemed stable, conservative management in a walking boot with weight-bearing as pain allows is safe.
- In unstable Weber B fractures, including displaced fractures with talar shift, surgical fixation is recommended.
- Once the fibula is stabilized, on-table assessment of the syndesmosis is essential with external rotation stress views and subsequent syndesmotic stabilization if indicated.

Weber C Fractures

- Syndesmotic injury in Weber C fibula fracture patterns is high.
- These are considered to be unstable injuries with a low threshold for surgical fixation.

- Fixation of the fibula with syndesmotic stabilization and syndesmotic stabilization alone are both supported in the literature.
- Fixation of high Weber C fractures can be technically more challenging than standard ankle fracture fixation.
- However, with syndesmotic stabilization alone, care must be taken to ensure no significant malrotation or shortening of the fibula is present at time of stabilization, as this will lead to malreduction of the syndesmosis.

Medial Malleolar Fractures

As is the general principle, undisplaced medial malleolar fractures can be managed nonsurgically, although the literature reports nonunion rates of up to 11%. One of the suspected causes of this is interposition of periosteum and/or soft tissue. With those requiring fixation, two medial malleolar screws are the most common mode of fixation, with evidence supporting non-cannulated, cannulated, partially, and fully threaded cancellous screws. The vertical shear-type fractures seen in the supination adduction ankle fracture subtype are commonly treated with antiglide medial semitubular plating for additional biomechanical support.

Posterior Malleolar Fixation

Classical opinion and evidence suggest fixation of posterior malleolar fragments in the setting of more than 25% articular surface. Similarly, there is the arbitrary "one-third rule" which was introduced by Nelson and Jensen who, in 1940, reported on a very small series of patients. However, fixation of the posterior malleolus has been found to carry several benefits including restoration of the articular surface of the tibia; accurate restoration of length of the fibula, helping to avoid malunion; and restoration of stability of the syndesmosis with fewer patients requiring its fixation [5, 6].

The posterolateral approach to the ankle is straightforward and provides safe access to the most commonly placed posterolateral malleolar fragment, as well as allow posterior fixation of the fibula fracture [7].

Syndesmotic Stabilization

As previously discussed, restoring the length and rotation of the fibula are essential to ensuring satisfactory reduction of the syndesmosis.

Outcomes are strongly correlated with anatomic reduction of the syndesmosis.

The two most common techniques for reducing the syndesmosis are:

1. A reduction clamp on medial tibia and the fibular ridge at the level of the syndesmosis. Must be cautious not to overtighten syndesmosis, and hence some surgeons prefer method two.
2. Applying thumb pressure onto the fibula—to allow even distribution of force onto the fibula and allow it to find its anatomic resting place at the syndesmosis.

Typically, one or two screws are used at the syndesmosis. There is no evidence that supports an optimal technique when assessing one versus two screws, 3.5 mm versus 4.5 mm screws, three cortices versus four cortices, and tight rope versus syndesmosis screws.

There remains no clear evidence supporting postoperative removal of syndesmosis screws. A widely quoted paper demonstrated that the best patient outcomes were reported in those patients in whom the screw broke after weight-bearing [8]. This was a relatively small study but nevertheless supports retention of the syndesmotic metalwork. Another school of thought is that removal of the syndesmosis fixation may allow those slightly malreduced syndesmoses to settle into a better position.

Fracture Fixation in Diabetics

Ankle fractures in diabetic patients pose significantly higher risks, including high risk of soft tissue complications including infection and higher risk of hardware failure.

A multidisciplinary team approach is vital including a diabetologist to optimize diabetic control, careful monitoring by plaster technicians for soft tissues issues and need for total contact casting, as well as the orthopedic surgical team. The general rule for this subgroup of patients is more fixation, e.g., multiple quadricortical syndesmosis screws and longer immobilization, typically a minimum of 8–10 weeks. These patients are at risk of developing Charcot arthropathy, especially if peripheral neuropathy is present from the outset.

In those with multiple medical comorbidities and multi-organ involvement and failure with poor soft tissues, a tibiotalocalcaneal intramedullary nail can be used to stabilize the ankle, often combined with arthroscopic preparation of the ankle and/or subtalar joints.

Summary of Indications for Surgical Fixation

- Any unstable ankle fracture with significant displacement or talar shift
- Open ankle fractures—require coordinated approach with plastic surgery
- Bosworth fractures

Complications of Surgery

- Superficial wound infection
- Deep infection
- Thromboembolic events
- Malunion/nonunion
- Reoperation for hardware prominence
- Post-traumatic ankle arthritis

Posterior Tibial Dysfunction and Adult-Acquired Pes Planovalgus

Introduction

Adult-acquired flatfoot (often referred to as pes planovalgus or posterior tendon dysfunction) is a common presentation in foot and ankle clinics and is a significant cause of pain, deformity, and disability.

It is characterized by four key features:

- Pes planus
- Hindfoot valgus
- Forefoot supination (also referred to as forefoot varus)
- Forefoot abduction

In order to fully appreciate the nature of this disease, it is essential to understand some key anatomic and biomechanical principles.

Anatomical Considerations

The relevant anatomy here refers to those structures that maintain the medial longitudinal arch of the foot, the loss of which is a key aspect of pes planovalgus. The arch is maintained by a combination of both static and dynamic stabilizers.

Static Stabilizers

Spring Ligament

– The plantar calcaneonavicular ligament, composed of three bands

Deltoid Ligament

– The deltoid ligament has a deep and superficial layer.
– The deep layer is comprised of deep anterior and posterior tibiotalar ligaments.
– The superficial component is made up of the talocalcaneal, talospring, and talonavicular bands.

Dynamic Stabilizer

Tibialis Posterior Tendon

– A muscle arising from the interosseous membrane and adjacent tibia and fibula
– Tendon that passes most anteriorly behind the medial malleolus
– Anterior, middle, and posterior limbs that insert broadly into the sole of the foot including the plantar navicular, the cuneiforms, the metatarsal, the sustentaculum tali, and the cuboid
– It inverts and plantarflexes the foot at the ankle and is a dynamic stabilizer of the medial longitudinal arch.
– It is also a hindfoot inverter.

The static and dynamic stabilizers combine to create a medial sling which supports the talar head and is a key relationship for maintenance of the longitudinal medial arch. The failure of this mechanism begins with the failure of tibialis posterior due to tendonitis and dysfunction. Initially, the spring ligament complex maintains the talar head, but in turn, this fails. The talar head falls plantar and medial

with nothing to hold it up. Unopposed peroneus brevis, the antagonist to tibialis posterior, pulls the foot further into abduction. The hindfoot drifts further into increasing valgus with the loss of tibialis posterior and the consequent gastrocnemius contracture. Deformities that begin as flexible and correctable changes become fixed and rigid.

The exact etiology of tibialis posterior failure is unknown; however, theories include acute injury causing initial tenosynovitis versus a long-standing degenerative process. There is also a vascular watershed theory whereby there is poor intrinsic blood flow in the part of the tendon between the medial malleolus and the navicular insertion. This may make it prone to undergoing a degenerative process and ultimately fail.

History and Examination

Risk Factors
- Obesity
- Hypertension
- Diabetes
- Increased age
- Corticosteroid use
- Seronegative inflammatory disorders

Symptoms
- Medial ankle pain—along the course of tibialis posterior
- Lateral ankle pain—due to sinus tarsi or subfibular impingement
- Change of foot shape—loss of medial longitudinal arch and forefoot abduction
- Plantar fasciitis—related to a tight gastrocnemius

The key feature to establish from the examination is whether the deformities are flexible or rigid, as this will determine features of management.

Features to assess include:

- Loss of medial arch
- Hindfoot valgus—Flexible or rigid
- Double leg heel rise—Do both heels swing in as would occur normally?
- Single heel raise—Checks tibialis posterior function
- Too many toes (forefoot abduction)—Greater than one and a half toes is abnormal.
- Assessment of forefoot supination—Passively correct hindfoot valgus and assess the foot end on.
- Feel tibialis posterior for tenderness and tendon thickening.
- Silfverskiold Test—to assess for gastrocnemius tightness

Imaging

Plain WEIGHT-BEARING Radiographs of the Ankle and Foot

Radiological Parameters

As well as assessing for degenerative change in the joints, must also look for features of tarsal coalition as this can cause a rigid flatfoot. The calcaneonavicular coalition is the most common and often identified on plain oblique films by the anteater sign (a bony projection from the anterior process of the calcaneus to the navicular). A talocalcaneal coalition, where there is coalition across the medial facet of the subtalar joint, is typically identified in a lateral ankle x-ray by the C-sign that is sometimes visible.

A number of radiographic parameters can be identified on weight-bearing radiographs of the foot [9, 10]:

Weight-Bearing AP Radiograph of the Foot

Talonavicular Uncoverage Angle/Percentage

- Can be expressed as an angle or percentage uncoverage
- Represents degree of forefoot abduction, namely, how much the navicular has drifted off the talus
- Two lines are drawn, one connecting the edges of the articular surface of the talus and one connecting the edges of the articular surface of the navicular. The angle formed by these two lines is the talonavicular coverage angle.
- An angle of more than 7° indicates lateral navicular subluxation.
- Can also be described as a percentage of uncovered talar surface

Kite Angle

- An ankle between the long axis of the talus and a line along the lateral border of the calcaneus
- Normal range for adults is 15–30°.
- An angle greater than 30° represents hindfoot valgus.

Simmon Angle

- The talar-first metatarsal angle
- A line drawn between the long axes of the talus should be in line with the first metatarsal shaft.
- The presence of an angle indicates forefoot abduction.

Weight-Bearing Lateral Radiograph of the Foot

Meary's Angle

- The lateral talar-first metatarsal angle
- This is an angle formed between the long axes of the talus and first metatarsal on a weight-bearing lateral view.
- A normal angle is between 0 and 5°.

Calcaneal Pitch

- An angle between the plantar surface of the calcaneus and the transverse plane. The transverse plane is usually identified by a line from the calcaneus to the base of the fifth metatarsal head.
- The normal angle is 17–32°.
- An angle less than the lower range indicates pes planus and loss of the longitudinal arch height.

A CT scan can help further delineate bony, structural pathology.

An MRI can be useful to assess the condition of the tibialis posterior tendon and patency of spring ligament, as well as identify more subtle degenerative changes in the joints.

Classification

The most widely used classification is that by Johnson and Strom, later modified by Myerson.

Stage 1:	Mild medial ankle pain/swelling
	Tibialis posterior synovitis
	No deformity
	Normal single heel rise
Stage 2:	Medial ankle pain/swelling +/− sinus tarsi impingement pain
	Tibialis posterior tenosynovitis and degeneration
	IIA: hindfoot valgus only
	IIB: hindfoot valgus and forefoot abduction
	Abnormal single heel rise
Stage 3:	Medial ankle pain/swelling and sinus tarsi impingement pain
	Subfibular abutment pain
	Degenerate tibialis posterior
	Osteoarthritic changes with FIXED deformity
	Forefoot abduction/valgus fixed hindfoot/no single heel raise
Stage 4:	Stage 3 + deltoid insufficiency and valgus talar tilt
	Stage 4a—talar tilt no ankle OA
	Stage 4b—talar tilt with ankle OA

Treatment

Treatment regimens differ based on whether any deformity present is flexible or rigid. In short, flexible deformities can be corrected—by corrective orthotics and corrective, joint-sparing surgery. Fixed, rigid deformity requires accommodative orthotics and joint-sacrificing surgery.

Nonsurgical Options

Physiotherapy

The goal is for early detection of the dysfunction and conservative management to prevent chronicity [11].

- Strengthening of tibialis posterior
- Range of motion exercises
- Gastrocnemius stretching and strengthening
- Improving stability and gait

Orthotics

- Orthotics can be used at all stages of adult flatfoot treatment. In early and flexible deformities, corrective orthotics can be used. These typically involve medial hindfoot posting and medial longitudinal arch support. Add medial forefoot posting if forefoot supination is present.
- Corrective orthotics typically used in stages 1 and 2
- With rigid deformity (stages 3 and 4), accommodative orthotics are required to support existing deformity. These will often be custom molded to match patient's deformity and pad pressure areas.
- With stage 4 involvement, can add ankle bracing to support failing joint.

Surgical Options [9–12]

Flexible Deformity

- Calcaneal Osteotomy
- Medializing calcaneal osteotomy and/or Evans osteotomy
- MCO used to realign hindfoot valgus. Always used in pes planovalgus correction
- Evans osteotomy is a lateral column lengthening procedure with an opening wedge osteotomy around 10 mm proximal to the calcaneocuboid joint. It is indicated in patients with forefoot abduction deformities (talar uncoverage >30%).
- MCO and Evans can be combined in the same correction.
- Medial Column Procedures
  - These are used to bring down the medial column to treat forefoot varus.
  - Cotton osteotomy
    - Dorsal opening wedge osteotomy of the medial cuneiform
    - Used in a setting of a stable medial column
  - Plantarflexion osteotomy of the first metatarsal
    - Basal, dorsal opening osteotomy to bring down first ray
    - Used in stable medial column
  - Lapidus procedure (first TMTJ fusion)
    - Fusion procedure used in the setting of first TMTJ instability
    - Serves the same purpose, to bring down the first ray
- Flexor Digitorum Longus Tendon Transfer/Spring Ligament Repair
  - FDL is synergistic with tibialis posterior and is commonly used to augment/replace the action of tibialis posterior.

  - Deformities must be flexible and reducible.
  - FDL is detached proximal to the knot of Henry and reattached through the medial navicular (plantar to dorsal).
  - Some toe flexion remains due to remaining attachment at knot of Henry.
  - FDL transfer often paired with spring ligament repair to augment medial structures
- Tendoachilles Lengthening or Gastrocnemius Recession
  - In the setting of equinus contracture

Fixed Deformity

Options in the surgical treatment of rigid deformity are limited. In fact, the mainstay of treatment for stage 3 dysfunction is triple arthrodesis (subtalar, talonavicular, and calcaneocuboid fusions). The deformity itself can be corrected with a double hindfoot fusion (subtalar and talonavicular), and often the calcaneocuboid joint is spared, particularly in the absence of arthritic change. The allows for a medial approach double hindfoot fusion and reduces potential lateral wound complications with large corrections.

In stage 4A dysfunction where the talar tilt is not associated with arthritic change, a triple/double hindfoot fusion can be combined with a deltoid ligament reconstruction. This may spare further degeneration in the ankle but also opens the opportunity for a total ankle replacement should the need arise, as there would be ligamentous stability. The alternative way to treat the stage 4B would be to perform a tibiotalocalcaneal fusion using a hindfoot intramedullary nail.

Ankle Arthritis

Introduction

Osteoarthritis of the ankle is a major cause of disability, with an impact on quality of life comparable to end-stage heart failure and hip arthritis [13–15]. Its incidence has been estimated to be 47.7/100 000, affecting approximately 1% of the world population [13–15].

Etiology of Ankle Arthritis

The majority of ankle arthritis is post-traumatic or related to ankle instability. Saltzman evaluated 639 patients presenting with painful end-stage ankle OA. In this cohort, 445 patients (70%) had post-traumatic OA, 76 (12%) had rheumatoid OA, and 46 (7%) had primary ankle OA [16]. While rotational ankle fractures were identified as the most common reason for post-traumatic ankle OA (164 patients), previous ligamental injuries were also found to be a cause of ankle OA in 126 patients [16].

Repetitive ankle sprains in sports are the main cause of ligamentous post-traumatic ankle OA with concomitant varus hindfoot deformity. The incidence of

ankle sprains and fractures is increasing, with arthritis typically setting in within 2–22 years of injury. Consequently, an increasing number of young adults, still in their working lives, are developing ankle arthritis.

Secondary OA has also been associated with a variety of underlying diseases or disorders, such as rheumatoid disease, hemochromatosis, hemophilia, gout, neuropathic diseases, avascular talus necrosis, osteochondral lesions, and postinfectious arthritis.

In contrast to the hip and knee, the ankle joint is most commonly affected by secondary degenerative changes. It is highly congruent under load and has articular cartilage that, although thinner than that in the knee and hip, has a higher compressive modulus. These biomechanical differences may protect the joint from primary osteoarthritis but may well predispose the joint to post-traumatic changes.

Horisberger identified the latency time between injury and end-stage post-traumatic ankle OA as 20.9 years, with a range between 1 and 52 years, in a clinical study including 257 patients (270 ankles) with painful end-stage ankle OA [17]. The major influencing factors were the type of fracture, complications arising in the healing phase, age at the time of injury, and a varus malalignment of the hindfoot [17].

History and Examination

Presenting Features

- Pain on weight-bearing
- Joint stiffness
- Altered gait
- Ankle swelling/deformity

Examination Findings

- Ankle joint effusion
- Deformity
- Osteophytes can cause palpable tenderness, especially on the anteromedial and anterolateral aspects of the ankle joint.
- Reduced range of movement in the joint
- Check for hindfoot malalignment.

Imaging

Weight-bearing AP/lateral and mortise views. Non-weight-bearing images can be misleading and cannot show true extent of pathology. Weight-bearing images of the foot are obtained to assess adjacent joint degenerative change especially in the subtalar and talonavicular joints.

Basic radiological features of osteoarthritis are seen:

- Joint space narrowing
- Subchondral sclerosis and cysts
- Osteophyte formation
- In the setting of instability, subluxation of the talus on the tibia may be seen.

Assessment of the radiographs for deformity with assessment of supramalleolar and intra-articular regions. In the supramalleolar region, it is important to ensure there are no malunited tibial fractures that have a secondary effect on the ankle joint. Weight-bearing long leg films are useful to assess anatomical and mechanical alignment. Must assess for the presence of deformity at the ankle joint, namely, varus or valgus, and if this is congruent or not. A difference of more than 4° is considered to represent noncongruency.

Both CT and MRI can be valuable in further assessment of the arthritic ankle. CT can identify and delineate bone cysts as well as adjacent joint osteoarthritis, whereas MRI is useful for assessing articular cartilage within the ankle and adjacent joints.

Classification

Takakura used weight-bearing radiographs to classify ankle OA into four stages [18]. For clinical use, the investigators simplified the classification, describing stage 1 as early, stages 2 and 3 as intermediate, and stage 4 as late.

Tanaka slightly modified the Takakura classification as follows:

- Stage 1: Early sclerosis and formation of osteophytes without narrowing of the joint space
- Stage 2: Narrowing of the medial joint space
- Stage 3A: Obliteration of the medial joint space with subchondral bone contact limited to the medial malleolus
- Stage 3B: Subchondral bone contact extending to the roof of the dome of the talus
- Stage 4: Obliteration of the entire joint space, resulting in bone contact throughout the ankle

Treatment

Nonoperative options include:

- Analgesia
- Activity modification
- Orthotics/bracing to support the ankle
- Patella tendon-bearing orthosis has been shown to be especially effective.
- Physiotherapy to work on lower limb strengthening, gait training, and stability
- Intra-articular joint injection
  - Can be used for diagnostic and therapeutic purposes
  - Classically, local anesthetic + steroid is cheapest but with varying duration of action (between 30 and 80% efficacy improved to 30–97% with use of ultrasound for more accurate placement of needle).

- Some evidence to support viscosupplementation, in the form of hyaluronic acid injection, suggests good efficacy but no better than other nonoperative modalities.
- No evidence supporting the injection of protein-rich plasma, fat-derived, or bone marrow-derived stem cell therapy.

Surgical Options

1. Arthroscopic Ankle Debridement and Microfracture

   Largely reserved for those with well-defined lesion less than 15 mm in diameter

   Can be used to improve those with anterior impingement due to anterior osteophytes

   Can also be effective in those with mechanical symptoms due to loose bodies or cartilage flaps
2. Joint Distraction Arthroplasty

   Controversial

   Involves application of an external fixator/frame with sequential, often daily distraction of the joint

   Indicated in:
   - Highly motivated patients
   - Refractory pain
   - Appropriate joint alignment
   - Preservation of ankle joint motion
   - Patients who do not want to proceed with arthrodesis of ankle arthroplasty

   Joint distraction is thought to optimize the body's own regenerative capacity and function via mechanical unloading of the joint. However, the exact biological mechanism remains poorly understood, and the literature has shown varying results.

   Decrease in joint reactive forces, an increase in proteoglycan synthesis, recruitment of mesenchymal stems cells, and decrease in subchondral sclerosis are all thought to occur with this technique.

   Outcomes following joint distraction arthroplasty have shown variable results.
3. Supramalleolar Osteotomy

   Supramalleolar osteotomy is a joint-preserving procedure reserved for eccentric cartilage loss secondary to excessive varus or valgus malalignment. It is well documented that changes in pressure and force transfer across the ankle joint occur in response to ankle malalignment, occurring in both the coronal and sagittal plane [19].

   Supramalleolar osteotomies are performed to realign the mechanical axis and thus redistribute the joint-loading force in the ankle, with the goal of delaying or stopping the degenerative cascade [19].

   Indications for supramalleolar osteotomy are:

- Asymmetric valgus or varus osteoarthritis
- At least 50% preserved tibiotalar joint surface

Contraindications include:

- Elderly patients with hindfoot instability not correctable with ligament reconstruction
- Severe vascular or neurologic conditions of the affected extremity
- Inflammatory arthritides
- Active infection

Varus Ankle Joint

- Medial opening wedge osteotomy is the ability for gradual correction, with simultaneous correction of the sagittal plane deformity via distraction and subsequent allograft implantation.
- May need an additional approach should the patient need a fibular osteotomy.
- The lateral closing wedge offers the ability to readily access the fibula, circumvent the need for allograft insertion, increase the inherent stability of the construct, and avoid medial soft tissue compromise.

Valgus Ankle Joint

- For valgus ankle correction, most recommend a medial closing wedge osteotomy with the aim again of 2–4° of varus overcorrection of the joint surface. The addition of a fibular osteotomy through a separate lateral incision is required.

Ankle Fusion Versus Ankle Arthroplasty

These two procedures have represented the mainstay in treatment of end-stage ankle arthritis. Ankle fusion has, however, been the gold standard for the treatment of end-stage ankle arthritis for many years [20]. Open approaches, including anterior plating and transfibular ankle arthrodesis, were initially popularized with excellent union results and relatively low complications. As arthroscopic techniques developed and evidence grew for the benefits of arthroscopic over open procedures, arthroscopic ankle fusion became a main treatment, with open cases being reserved for complex deformity cases.

However, it is well documented that tibiotalar fusions lead to deficits in function and may cause or exacerbate adjacent joint arthritis, particularly in the subtalar joint. Ankle arthroplasty preserves motion at the ankle joint while still achieving the primary goal of pain relief [20]. However, the results from the first generations of total ankle replacement were disappointing due to poor clinical results and high rates of aseptic loosening [21, 22]. The first-generation prostheses were non-modular with all-polyethylene tibial components and solid metallic talar components using bone cement for the bone prosthesis interface [20–22]. Current three component mobile-bearing implants have much improved early to midterm results, resulting in ankle arthroplasty being an effective option in certain patient populations [20–22].

Patient selection in ankle arthroplasty is very important, and there are a number of contraindications:

- Severe deformity
- Severe osteoporosis
- AVN of the talus
- Charcot arthropathy
- Ankle instability
- Obesity
- Young, active patients who have increased risk of failure and revision
- Previous septic arthritis

The current prostheses have ten-year survival rates of more than 90%, and therefore, total ankle arthroplasty is a valuable asset in the careful chosen candidate.

Risks/complications in arthrodesis/total ankle arthroplasty:

Ankle Arthrodesis
- Infection
- Nonunion (4–6% reported in literature)
- Malunion
- Development of adjacent joint OA (especially subtalar and talonavicular joints)

Ankle Arthroplasty
- Infection (superficial and deep)
- Aseptic loosening requiring revision surgery

Secondary Causes of Ankle Arthritis

Rheumatoid Arthritis

Involvement of the foot and ankle in patients with rheumatoid arthritis is common, with over 90% foot pain during the course of their disease. Ankle arthritis usually occurs in the later stages of rheumatoid arthritis.

Ankle fusion has long been considered to be the gold standard in rheumatoid-driven ankle arthritis. The relief of pain and decrease of deformity give rise to the potential gain of walking ability. However, the loss of ankle motion and the resultant stress on other joints, especially in RA patients, may increase degenerative lesions in the mid- and forefoot. There is evidence to support total ankle arthroplasty as a safe and effective alternative to fusion. Despite concerns over bone quality and osteointegration of components, the lower demand of patients with long-standing rheumatoid, in whom ankle arthritis is usually seen, may confer some protection against loosening.

Stengel performed a meta-analysis that included ten studies on third-generation ankle replacements and presented a weighted average improvement of 45.2 points

(maximum score 100 points). Gougoulias et al. reviewed 13 studies and focused on clinical failure and survival rates of both second- and third-generation implants that showed 9.8% (95% CI 3.1–16.5) implants failure after 5 years.

In rheumatoid arthritis patients, preoperative assessment plays an important role. As well as the effects of the general manifestations of rheumatoid, e.g., cervical involvement when considering manipulation of the neck for general anesthesia, assessment of medications is vital. Many pharmacological agents used in the treatment of rheumatoid arthritis are immunosuppressive and may, therefore, increase the risk of wound complications and infections.

Commonly used medications include:

Nonsteroidal Anti-inflammatories

Theoretical risk of impaired bone healing (only proven in animal studies)

Steroids

- Patients on chronic steroid therapy may develop secondary adrenal insufficiency that can manifest as full-blown adrenal crisis in the perioperative period. Patient may need perioperative stress-dose steroids to mitigate risk. Some evidence shows that steroids can lead to delayed healing, inhibit collagen synthesis, and increase risk of infection.

Disease-Modifying Antirheumatic Drugs (DMARDs)

- Common examples include methotrexate, sulfasalazine, and hydroxychloroquine—all three of which can be continued. Leflunomide can be continued for minor procedures but stopped 1–2 days prior surgery and restarted 2 weeks after.

Tumor Necrosis Factor (TNF) Antagonists

- Etanercept must be stopped 2 weeks prior to major surgery. With infliximab and adalimumab, surgery should be lined up at the end of a dosing interval, prior to a new dose. All can be restarted at 2–4 weeks. Evidence shows a great risk of surgical site infection if taking TNF antagonists, but this risk is reduced with pausing treatment.

Biologic Agents

- The most commonly used are rituximab, abatacept, and TCZ. Evidence of increased risk of wound healing complications and infection. Rituximab must be stopped 3–6 months prior to surgery. TCZ should be stopped weeks prior to surgery. For most other biologics, aim to stop between three and five half-lives prior to surgery. Biologics can be recommenced after surgery once there is good wound healing, usually no earlier than 2 weeks.

It is worth mentioning that although evidence suggests an increased complication rate while on these medications, a full assessment must be performed with the support of the patient's rheumatologist, and considerations must be given to severity and the disability caused by a potential acute flare-up.

Gout

Gout is the most common form of inflammatory arthritis in men. It is caused by the deposition of monosodium urate crystals in joints and soft tissue. The disease is characterized by painful acute flares, and it may progress to a chronic disease with tophus and deformity. Tophi, mainly found in articular, periarticular, bursal, bone, auricular, and cutaneous tissues, are a pathognomonic feature of gout. In most patients, the first gout attack occurs in the first metatarsophalangeal joint. However, in >20% of all patients, the ankle is the site of the first and subsequent gout attacks.

Gout is a rare cause of severe ankle arthritis. The role of gout in the development of degenerative joint changes due to bone erosion is unclear. On view is that gout may cause chronic arthropathy, especially in patients with prolonged hyperuricemia. On the other hand, joints affected by gout often have previous arthritic changes, which may predispose to monosodium urate crystal deposition.

Initial treatment of gouty flares is pharmacological, including NSAIDs and colchicine for pain relief and allopurinol for longer-term prevention of flares.

The two surgical methods to treat end-stage gouty ankle arthritis that have been discussed in the literature are total ankle replacement and ankle arthrodesis. Ankle arthrodesis has long been the gold standard. However, as has been the case with inflammatory arthropathies generally, the midterm results following total ankle replacement in patients with gouty ankle arthritis are encouraging. Patients have significant pain relief, and recent studies have observed substantial improvement in ankle function and quality of life. Surgery around the first metatarsophalangeal joint has been associated with increased rate of wound healing complications and infection. However, infection and wound complication after surgery around the ankle are comparable to non-gout patients and may be explained by the better-quality soft tissue envelope around the ankle compared to the big toe.

Tendonitis and Tendinopathy Around the Ankle

Introduction

Tendons are dense, highly structured connective tissues that produce joint motion by transferring forces from muscle to bone. They are composed primarily of type I collagen arranged in parallel fibers, with additional constituents including proteoglycans, glycosaminoglycans, and other collagens constituting the remaining 20–30% of dry weight. Although the structure and inherent characteristics of tendons give them great tensile strength and allow them to transfer force from muscle to bone, many of these same characteristics also result in poor healing potential.

Although the prevalence and incidence are variable, chronic tendon disorders comprise a significant portion of all sport-related injuries. Studies suggest that chronic tendon problems represent 30% of all running-related injuries [21].

Many chronic tendinopathies are a result of excessive mechanical load across a tendon. That is to say that the tendon was not preconditioned for the load that it is exposed to. For this reason, it is not necessarily the absolute force, duration, or

frequency of load a tendon is exposed to across a period of time but instead that these factors were in excess of the tendon's usual conditioning. The high intensity or frequency the tendon is exposed to is believed to injure the structure of the tendon.

To a certain point, spontaneous healing generally occurs; however, if the regenerative capabilities of the tendon are exceeded, clinical symptoms of tendinopathy occur. Inflammation is a common result, and for this reason, tendonitis is frequently the diagnosis under these circumstances.

Tendinitis is inflammation of a tendon proper, whereas tenosynovitis is inflammation of the tendon and its sheath. Tendinosis is degeneration of the collagen bundles forming the tendon tissue without significant inflammation.

Around the ankle, the common problems are the Achilles, the tibialis posterior, and the peroneal tendon. The tibialis posterior tendon has been discussed in detail in the adult-acquired flatfoot section of this book, and the others are discussed in more detail below.

The Achilles Tendon

Achilles tendinopathy can be subdivided into disorders of the Achilles insertion, including insertional tendinopathy (IAT), Haglund's deformity, and retrocalcaneal bursitis, and disorders of the midportion of the Achilles (2–6 cm above the insertion), including non-insertional Achilles tendinopathy [23–25].

In a meta-analysis by Vlist et al. [26], limited evidence was identified for the following nine risk factors:

1. Prior lower limb tendinopathy or fracture
2. Use of ofloxacin antibiotics
3. Increased time between heart transplantation and initiation of quinolone treatment for infectious disease
4. Moderate alcohol use
5. Training during cold weather
6. Decreased isokinetic plantarflexor strength
7. Abnormal gait pattern with decreased forward progression of propulsion
8. More lateral foot rollover at the forefoot flat phase
9. Creatinine clearance of <60 mL/min in heart transplant patients

Insertional Achilles Tendinopathy

Insertional tendinopathy is a degenerative process resulting in disorganized collagen and mucoid degeneration [23–25]. Microtears within the tendon occur as a result of repetitive mechanic stress from overuse. This causes the collagen degeneration, fibrosis, and finally calcific metaplasia [23–25]. Haglund's deformity, an enlargement of the posterosuperior tuberosity of the calcaneus, and retrocalcaneal bursitis, inflammation of the bursa between the anterior

aspect of the Achilles and posterior aspect of the calcaneus, are both associated exacerbating factors. They cause mechanical bony impingement and chemical irritation [23–25].

Patients present with pain and tendon thickening at insertion of Achilles tendon, with a classical "pump bump" deformity—so called due to rubbing in footwear ("pumps"). Footwear is an irritant and patients will prefer backless shoes.

Imaging

Weight-bearing lateral radiograph may demonstrate:

- Intra-tendinous calcification
- Insertional spurring
- Haglund's deformity
  - Haglund's deformity can be evaluated by Fowler and Philip posterior calcaneal angle and parallel pitch lines.

USS can be a valuable dynamic imaging modality, in particular to assess neovascularization. MRI gives details of extent of tendon involvement, retrocalcaneal bursitis, calcaneal edema at the tendon-bone interface, and calcification within the tendon.

Treatment

Initial management includes:

- Rest
- Ice
- NSAIDS
- Activity and footwear modification
- Physiotherapy including gastrocnemius and Achilles stretching
- Night splinting
- Extracorporeal shockwave therapy

Guided injections into the retrocalcaneal bursa, to reduce inflammation, can be therapeutic. The risk is of tendon weakening and ultimately potential rupture due to corticosteroid effects.

Surgical Intervention

Improvement rates of up to 90% have been reported in the literature following non-surgical treatment, but recurrence of symptoms is common.

Refractory or recurrent insertional Achilles tendinopathy is treated by:

- Achilles debridement, Haglund's excision, retrocalcaneal bursa excision, and reattachment of the Achilles
- If more than 50% of the tendon is involved/requires debridement, FHL tendon transfer is recommended.
- A Zadek (dorsal closing wedge calcaneal) osteotomy has some evidence to show consistent symptomatic improvement by altering the biomechanics of the tendon.

Midsubstance Achilles Tendinopathy

Chronic midportion Achilles tendinopathy occurs as a result of overuse. Fusiform thickening of the midportion of the Achilles is thought to be caused by abnormal vascularity and ischemic degeneration. There is a failed healing response with disorganized proliferation of tenocytes, disruption of collagen fibers, and subsequent increase in the noncollagenous matrix, mainly proteoglycans. The tendon thickening increases over time, and physical activity triggers pain. It usually affects an area 2–6 cm proximal to the Achilles insertion.

Patients present with pain, swelling, and discomfort as described. Discomfort is activity related.

Imaging

Similar to insertional Achilles tendinopathy, plain radiographs may show areas of calcification. This can be confirmed on ultrasound or MRI, which will help delineate the extent of involvement of the Achilles.

Treatment

Initial management follows the same protocol as for insertional Achilles tendinopathy. Orthotic use includes correcting hindfoot deformity with insoles, heel raises, night splints, and, in severe cases, plaster casting for total rest.

Targeted injections can play a role, but again the risk of corticosteroid use and tendon rupture is significant. High-volume saline injections under ultrasound guidance have shown some benefit in the literature.

Surgical Treatment

- Some evidence for gastrocnemius lengthening/recession to reduce forces through Achilles
- Achilles debridement +/− tubularization is recommended for moderate to severe disease. If a significant proportion of the tendon requires debridement, with less than 50% good tendon remaining, a tendon transfer, usually FHL, is required.

The Peroneal Tendons

Peroneal tendon injury/pathology most commonly presents as three forms, which can occur concurrently:

- Tendon subluxation/dislocation
- Tendon tears
- Tendonitis/tendinopathy
- Painful os peroneum

Review of relevant anatomy and function enables a better understanding of treatment protocols.

Peroneus longus and peroneus brevis tendons run along the posterolateral surface of the fibula in lateral compartment of leg. The peroneus longus tendon travels along the posterior aspect of the peroneus brevis tendon, inferior to the peroneal tubercle on the lateral wall of the calcaneus, and then turns medially around the lateral border of the cuboid in the cuboid notch toward the first metatarsal.

The peroneus brevis runs directly posterior to fibula.

The os peroneum is a fibrocartilaginous sesamoid in the tendon on the peroneus longus. It is fully ossified in 20% of feet and exists in the tendon at the point where it wraps around cuboid in the cuboid tunnel. The os peroneum provides an increased mechanical advantage, which allows the peroneus longus to plantarflex the first ray.

The peroneus longus and brevis tendons share a synovial sheath starting 2.5–3.5 cm proximal to the tip of the fibula and then separate into their own, separating sheaths at the level of the peroneal tubercle. In 15% of patients, there is a shared peroneal tendon sheath.

The peroneus brevis has a long musculotendinous junction that may extend inferior to the ankle joint and occupy space in tendon sheath, leading to pathologic conditions such as tenosynovitis, superior peroneal retinaculum (SPR) damage, and chronic tears.

The tendons lie laterally to the subtalar joint line and function to plantarflex the ankle and evert the foot. Almost 28% of hindfoot eversion power comes from the peroneus brevis, and 35% of power comes from the peroneus longus.

In addition, the peroneus brevis is the primary abductor of the forefoot, while the peroneus longus also plantarflexes the first metatarsal. Both muscles are active stabilizers in inversion-supination ankle sprains, and the peroneus longus is a passive stabilizer of the ankle during inversion-supination.

At 15–25 of plantarflexion, the tendons are perched along the distal fibula and prone to injury with inversion.

Both muscles are innervated by the superficial peroneal nerve proximally in leg and get their blood supply from the posterior peroneal artery.

Both tendons have a hypovascular zone where they wrap around the tip of the fibula, with the peroneus longus tendon having an additional hypovascular zone at the cuboid notch; not surprisingly, these are common areas of injury.

The peroneus quartus is an anomalous muscle that exists in up to 22%.

This muscle originates from the peroneus brevis and travels through the shared tendon sheath to insert onto the peroneal tubercle and has been implicated in several peroneal tendon disorders. This muscle is the most common accessory muscle found in the ankle.

There are multiple stabilizers of the peroneal tendons as they wrap around the lateral malleolus that serve to counteract the forces that would work to sublux or dislocate the tendons anteriorly during plantarflexion.

The superior peroneal retinaculum (SPR) extends from the posterolateral surface of the fibula 2 cm above its tip to the lateral wall of the calcaneus and/or Achilles and helps to confine the peroneal tendons within the retrofibular groove.

The calcaneofibular ligament, which lies deep to the tendons, also helps to stabilize the tendons in the retromalleolar groove.

A fibrocartilaginous ridge is located along the posterolateral border of the distal fibula, which acts to deepen the retrofibular/retromalleolar groove by 2–4 mm and also functions as a bumper to prevent subluxation.

Presentation, History, and Examination

Peroneal pathology must be considered with any patient presenting with lateral ankle pain.

It should always be considered in those presenting with ankle sprain/instability as the mechanism of injury is similar to the injuries often concurrent.

A detailed history of previous injuries can be relevant, with patients presenting post ankle sprain and ankle or calcaneal fracture.

Peroneal tendonitis is of more gradual-onset pain and presents with posterolateral ankle swelling.

Patients with peroneal tendon subluxation often complain of a painful clicking sensation.

Peroneus brevis tears are often characterized by persistent swelling along the course of the peroneals, whereas peroneus longus tears can present with pain in or around the cuboid groove and extending into the plantar aspect of the foot near its insertion.

Use of ciprofloxacin and other fluoroquinolones has been associated with tendon rupture and should be included in the history.

Inspection and palpation with active firing of the tendons are important parts of the physical examination.

Warmth and swelling along the course of the peroneal tendons should be identified, which can indicate peroneal tendonitis.

Assessment of hindfoot and forefoot alignment is fundamental because a varus hindfoot can predispose the peroneals to injury.

Palpation along the course of the peroneals should be performed, and any areas of tenderness should be noted; in some cases, one may be able to feel thickening of the tendon.

Strength testing of the peroneals should be performed; weakness, pain, or both with resisted ankle eversion and/or resisted plantarflexion of the first ray may indicate pathology.

Ligamentous stability should be assessed.

Peroneal subluxation is tested by flexing the knee and having the patient actively plantarflex and dorsiflex the ankle with resisted eversion. The test yields positive result when the peroneal tendons can be felt or seen to subluxate anterior to the lateral malleolus.

Intrasheath subluxation should be suspected if the tendons are noted to translate relative to one another without truly subluxating anterior to the lateral malleolus.

Imaging

- Radiographs are often normal but can be useful in the presence of previous bony injury.
- Also, the "fleck sign" on radiograph is pathognomonic of SPR avulsion.
- CT may be helpful to investigate the anatomy of the posterior fibula groove.
- US, being a dynamic assessment, usually picks up the subluxation, and MRI can be used to assess the fibula groove and assess for a peroneal tendon injury and intra-articular pathology.

Peroneal Tendon Instability: Subluxation/Dislocation

This is where the peroneal tendons dislocate and then repetitively sublux from behind the lateral malleolus.

It most commonly occurs in young, active patients.

The mechanism is usually a sudden, rapid dorsiflexion of an inverted foot and can also be caused by sudden, rapid contraction of the tendons themselves.

Subluxation leads to recurrent trauma to the tendon and the development of longitudinal tears, usually in brevis.

Another common causative factor can be lateral wall calcaneal malunion after calcaneal fracture.

Can be associated with tears to both peroneals and due to a similar mechanism, with ankle instability.

Treatment

Nonoperative Treatment [27, 28]

- All acute peroneal tendon subluxation/dislocation injuries, in nonathletes, can be managed with cast immobilization, ensuring that the tendons are reduced at the time of casting.
- Period of 6 weeks of immobilization in cast

Surgical Options [27, 28]
- Indicated in athletes with acute injuries, looking for a rapid return to sport or in the setting of an acute longitudinal tendon tear
- In the acute situation, a repair of the superior peroneal retinaculum and fibular groove deepening is performed, with successful outcomes of 96% success.
- In the chronic setting, repair of the SPR is more challenging due to the attenuation of the soft tissue, although still attempted. This is combined with a fibular grove deepening and addressing any hindfoot varus with, typically, a lateral calcaneal osteotomy.
- Management of tendon tears is discussed in the next section, but careful attention should be paid to exploring each tendon for injury.

Peroneal Tendon Tears

These most commonly occur in the peroneus brevis tendon with tears and peroneus longus relatively rare and concurrent tears even less common.

Chronic tears are more commonly seen than traumatic rupture.

Complete ruptures can lead to recurrent sprains, pain, and instability, and in the case of peroneus longus ruptures, the potential development of a cavovarus foot.

Partial tear of the peroneus brevis is most commonly seen at the area of vascular watershed in the retromalleolar sulcus.

Peroneus longus tears are more commonly observed in its watershed areas, namely, the peroneal tubercle or distal to the os peroneum in the cuboid tunnel.

Treatment

Nonoperative Treatment [27, 28]
– Nonsteroidal anti-inflammatories
– Activity restriction in modification
– Resting in a walking boot
– Conservative management has a high failure rate which has been documented as much as 80%.

Surgical Treatment Options [27, 28]

Simple tears can be managed with debridement/core repair and tubularization. These typically involve between 30 and 50% of the tendon.

Complex tears involving more than 50% of the tendon require debridement of the tears and either:

1. Tenodesis to the peroneus longus
2. Interposition allograft, if sufficient muscle excursion
3. FHL tendon transfer, if insufficient muscle excursion

Consideration of calcaneal osteotomy in those with hindfoot varus

Peroneal Tendonitis/Tendinopathy [27, 28]

Nonoperative Treatment

- NSAIDs
- Activity modification
- Ankle brace to reduce subtalar motion and rest the tendons
- Physiotherapy

Surgical Treatment

In cases of peroneal tendonitis that have been resistant to conservative management, surgical treatment is based on pathology:

- Tenosynovectomy
- Debridement of diseased tendon and tubulization
- Repair/debridement of peroneal tendon tears, again most commonly peroneus brevis
- Tenodesis is severely degenerate tendons or complete rupture.
- Treat any tendon instability as a causative factor.
- Consider lateralizing calcaneal osteotomy in those with varus hindfoot.

Painful Os Peroneum Syndrome

Surgical treatment of painful os peroneum generally involves open debridement of the tendon and tenosynovectomy. An incision is made along the course of the peroneus longus to the level of the lateral border of the foot. The os peroneum is carefully shelled out, and the tendon debrided and/or repaired via tubularization or an end-to-end repair, depending on the size of the defect. If greater than 50% of the tendon has been compromised, tenodesis of the longus tendon to the peroneus brevis is generally recommended.

Painful Conditions Around the Ankle

Ankle Instability/The Low Ankle Sprain

Ankle sprains are one of the most common musculoskeletal injuries. In all sports injuries, the rate of ankle sprains ranges from 15 to 20%. It is also commonly seen in dancers.

Anatomy

The ankle joint capsule is reinforced by the lateral ligament complex laterally and by the deltoid ligament medially.

The lateral ligament complex comprises of three main structures: the ATFL, CFL, and posterior tibiofibular ligament (PTFL).

The ATFL is a capsular thickening that runs anteromedially, originating from the anterior aspect of the distal fibula and inserting on the talar body/neck.

The CFL originates on the anterior border of the fibula, confluent with the ATFL. It courses deep to the peroneal tendons and inserts obliquely and posteriorly along the calcaneal tubercle.

The PTFL arises from the posterior aspect of the distal fibula and courses posterolaterally to insert into the posterior aspect of the talus.

The most common injury mechanism is a combination of inversion and adduction of the foot in plantarflexion. This injury mechanism can cause damage to the lateral ankle ligaments. Injury of the anterior talofibular ligament with intact medial ligaments leads to anterolateral rotary instability. Additional transection of the calcaneofibular ligament adds a tilting of the talus. Recurrent ankle sprains can cause function instability.

Associated injuries/conditions include:

- Subtle cavovarus foot, in particular hindfoot valgus
- Peroneal tendon injuries
- Osteochondral defects
- Deltoid ligament injury
  - Superficial deltoid: limits talar abduction
  - Deep deltoid: limits external rotation
- Fractures
  - Fifth metatarsal base
  - Anterior process of calcaneus
  - Lateral or posterior process of the talus
- Complex regional pain syndrome

History

- History of rolling or twisting the ankle during activity or sports
- A "pop" or crack may be heard at the time of injury.
- Inability to weight-bear
- Repeated episodes of the ankle giving way
- Pain, clicking, or locking may be associated with intra-articular pathology (e.g., osteochondral defect (OCD).
- Patients may describe a lack of confidence with the ankle and don't "trust" it.

Examination

- Pain, swelling, and bruising
- There may be associated hindfoot pathology (cavovarus foot).
- Examination for hypermobility is necessary.
- Tenderness at the site of the ligament injury
- Increased anterior drawer and inversion compared to the unaffected side indicates a mechanical instability.

Grade	Haematoma/swelling pain on palpation	Anterior drawer	Hindfoot inversion (talar tilt test)	Injury	Stability
I	Yes	Negative	Negative	Incomplete ATFL	Stable
II	Yes	Positive	Negative	Complete ATFL	Unstable
III	Yes	Positive	Positive	Complete AFL/CFL	Unstable

Fig. 28.1 Table summarizing the classification of ankle instability and clinical relevance

– Crepitus or pain in the ankle joint suggests intra-articular pathology.
– Hindfoot instability may be displayed by cupping and rotating the heel and comparing to contralateral side.

Classification (Fig. 28.1)

Imaging

Weight-bearing ankle x-rays (AP/lateral and mortise) and foot x-rays

To rule out ankle and fifth metatarsal fractures
May be anterior translation of the talus

Ultrasound (USS) has a 91% accuracy in detecting an acute ATFL tear compared with 97% for MRI [29].

MRI is useful is the diagnosis of ligament injury and assessment of intra-articular pathology and surrounding tendons, including peroneals.

Treatment

Nonsurgical [30–33]

- Indicated for all acute injuries
- May require an initial immobilization
  – Short period of weight-bearing immobilization in a walking boot, but early mobilization facilitates a better recovery
- Grade III sprains may benefit from 10 days of non-weight-bearing
- Physiotherapy
  – Initial Phase
    Early functional rehabilitation begins with motion exercises and progresses to strengthening, proprioception, and activity-specific exercises.

- Strengthening Phase
  Begins once swelling and pain have subsided and patient has full range of motion
  Neuromuscular training with a focus on peroneal muscles strength and proprioception training
  Functional brace that controls inversion and eversion during the strengthening period and used as prophylactic treatment during high-risk activities thereafter

Early functional rehabilitation enables the quickest return to physical activity, with supervised physical therapy showing a benefit in early follow-up.

Surgical Treatment [30–33]

Indications: Ongoing pain and instability with failure of nonoperative management
Reconstruction of the lateral ligaments can be:

- Anatomic versus nonanatomic
- Current gold standard is the Brostrom-Gould Stabilization, which is an anatomic technique.
  - Shortening and reinsertion of the ATFL and CFL
  - The Gould modification describes reinforcing the repair with inferior extensor retinaculum and fibular periosteum.
- Tendon transfer and tenodesis represent nonanatomic procedures.
  - Watson-Jones—peroneus brevis passed through fibula into neck of talus
  - Chrisman-Snook—reinforcing the ATFL and CFL by using one-half of the peroneus brevis tendon

The surgical procedure is usually followed by 2 weeks of immobilization in a non-weight-bearing cast, followed by a further 4 weeks in a walker boot as a minimum.

Osteochondral Lesions (OCL) of the Talus

The term “osteochondral lesion” is used to describe a number of pathological lesions to the talus including osteochondral defects, osteochondritis dissecans, and true osteochondral fractures.

Articular cartilage has poor regenerative capacity, and the osseous blood supply to the talus is tenuous. Both of these issues make the treatment of osteochondral lesions of the talus particularly challenging.

OCLs of the talus can be divided into idiopathic (osteochondritis dissecans) and traumatic, as a result of ankle instability and an ankle sprain, or an ankle fracture. The most common cause, by far, is trauma, accounting for around three quarters of all talar lesions.

Pathophysiology

Idiopathic

Osteochondritis Dissecans

- Originally described by Berndt and Harty as a "transcondylar fracture"
- Now more accurately described as an idiopathic and focal lesion involving subchondral bone that leads to an increased risk of cartilage softening, loosening, and eventual disruption
- Can cause premature onset of osteoarthritis
- Thought to be a primary dysfunction of the subchondral bone that affects the overlying cartilage
- Dysfunction of bone thought to be secondary to ischemic events
- Localized osteonecrosis of the subchondral plate, resulting from either repetitive microtrauma or the disruption of the anastomosing vessels between the epiphyseal cartilage and bone during development, has been implicated in the development of osteochondritis dissecans.
- Early symptoms are the result of softening and changes of the mechanical properties of the affected cartilage, in the absence of radiographic changes.
- As the cartilage detaches from the underlying pathological subchondral surface, cartilage flaps and loose bodies can develop, leading to eventual synovitis, mechanical symptoms, and progressive pain.

Trauma

- An axial load along with plantarflexion, external rotation, and inversion leads to traumatic impaction of the medial talar dome.
- Similarly, an axial load combined with inversion and dorsiflexion of the foot causes traumatic impaction of the lateral talar dome.
- A history of trauma is more commonly associated with lateral lesions than with medial lesions.

History and Examination

Common findings on history and physical examination include:

- Pain
- Swelling
- Mechanical symptoms such as catching or locking
- Ankle joint effusion
- Stiffness of the ankle
- Evidence of ankle instability
- Diminished range of motion
- Pain during inversion or dorsiflexion
- Hindfoot malalignment

Imaging

Weight-Bearing Ankle Radiographs—AP/Lateral/Mortise

May visualize a radiolucent area at medial/lateral talar dome

MRI

Will not only identify lesion but will pick up underlying bone edema and possible association with ankle sprain

- CT: Can be useful for true osteochondral lesions to evaluate extent of bony involvement, as well as subchondral cystic change.
- Many advocate all three modalities in assessing OCDs.

Classification

The most commonly known is the Berndt and Harty classification based on plain radiographs:

- Stage 1—A small area of subchondral compression
- Stage 2—Partial fragment detachment
- Stage 3—Complete fragment detachment
- Stage 4—Displaced fragment

There are also CT-based and MRI-based classifications summarized below.

Ferkel and Sgaglione CT Staging System

- Stage 1—Cystic lesion within dome of talus with an intact roof on all view
- Stage 2a—Cystic lesion communication to talar dome surface
- Stage 2b—Open articular surface lesion with the overlying nondisplaced fragment
- Stage 3—Nondisplaced lesion with lucency
- Stage 4—Displaced fragment

Hepple MRI Staging System

- Stage 1—Articular cartilage edema
- Stage 2a—Cartilage injury with underlying fracture and surrounding bony edema
- Stage 2b—Stage 2a without surrounding bone edema
- Stage 3—Detached but nondisplaced fragment
- Stage 4—Displaced fragment
- Stage 5—Subchondral cyst formation

Management

Nonoperative [34–37]

Nonoperative treatment for acute nondisplaced osteochondral lesions of the talus and cystic lesions has been associated with successful clinical results in about 50% of cases.

- Short leg cast and non-weight[bearing for 6 weeks
- Indications:
  - Acute injury
  - Nondisplaced fragment with incomplete fracture

Typical modalities of activity modification, bracing, nonsteroidal anti-inflammatory drugs (NSAIDs), physical therapy, and protected weight-bearing in a walking boot may alleviate symptoms.

Surgical

Indication: Acute injury with large osteochondral fragment

No improvement despite 3–6 months of nonoperative treatment

Surgical intervention on more chronic lesions can be subdivided into three basic principles [34–37]:

- Cartilage repair
- Cartilage regeneration
- Cartilage replacement

Cartilage Repair

Marrow Stimulation Techniques

- Often performed as first-line surgical treatment
- Ankle arthroscopy + microfracture
  - Perforation of the subchondral bone allows progenitor cells to infiltrate the lesion and stimulate healing.
  - Microfracture leads to the formation of fibrocartilage (mainly type I collagen as opposed to native hyaline cartilage).
  - Although thought to be structurally inferior to hyaline cartilage, microfracture is evidenced to reliably produce pain relief and improvement in function.
- Outcomes influenced by:
  - Size of lesion
  - Age of patient
  - Chronicity
  - Location
  - Containment of the lesion
  - Presence of subchondral cysts
  - Presence of joint degenerative changes

- Larger lesions do poorly:
  - Best for lesions of mean diameter <15 mm
- Procedure associated with debridement of loose chondral margins that may cause mechanical symptoms

Retrograde Drilling

Best for lesions with intact cartilage but subchondral cysts

Can be combined with autologous bone graft or injectable synthetic graft/bone substitutes

Cartilage Regeneration

Autologous Chondrocyte Implantation (ACI)

- Two-stage operation
- Hyaline cartilage harvested from anterior talus on non-weight-bearing femoral condyle
- Cartilage then cultured for 3–8 weeks to allow growth of chondrocytes
- Second procedure involves delivery of cells into the lesion in the talus, with a periosteal patch from the tibia sewn to cover the defect.
- Encouraging clinical results

Matrix-Induced Autologous Chondrocyte Implantation (MACI)

- Second generation of ACI
- Periosteal harvesting is eliminated with use of a matrix.
- Saves time and reduces complications from periosteal harvest
- Mixed results but generally show improvement in functional outcome

Cartilage Replacement

Osteochondral Allograft Transfer (OAT)

- Replace osteochondral lesions of the talus with hyaline cartilage
- Main benefit is thought to be retention of type II collagen.
- Cartilage plug usually from ipsilateral knee but can also use talus and calcaneus
- Significant improvement in outcome scores
- However, up to 50% of patients in some studies complained of knee pain.

Osteochondral Allografting

- Typically used for large lesions, usually on the shoulder of the talus
- Cadaveric human graft
- Fresh allografts have the advantage of chondrocyte viability compared with fresh-frozen allografts.
- Donor talus is size-matched, typically on the basis of a CT scan of the recipient talus.
- Advantages—ability to treat large cystic lesions and restore structural integrity
- Disadvantages—possible nonunion of bony interfaces and use of intra-articular fixation

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Ankle Sprains

29

Maria Loredana Popescu and Wasim S. Khan

Definition

Ankle sprains are common musculoskeletal injury in which the ligaments of the ankle are partially or completely torn due to sudden stretching.

Osseo-Ligamentous Anatomy of the Ankle

The ankle joint is formed by the tibia, fibula, and talus. The distal tibial metaphysis flares up to form the plafond and the posterior and medial malleolus. The distal fibula forms the lateral malleolus and rests within the incisura (a groove on the posterolateral aspect of the tibia). The medial and lateral malleoli together with the plafond form a mortise to fit the talar dome.

The intrinsic stability of these congruent articular surfaces is reinforced by three main ligamentous structures (see also Table 29.1):

1. The lateral collateral ligament complex comprises of three separate ligaments connecting the distal aspect of the fibula to the talus and calcaneus. Anterior talofibular ligament (ATFL) passes anteriorly to the talar neck and is very often ruptured in ankle sprains. The calcaneofibular ligament (CFL) passes inferiorly to insert into the calcaneus (the second most common ruptured ligament in low

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Table 29.1 Ligaments involved in the stability of the ankle joint

Lateral ligament complex	Anterior talofibular ligament (ATFL) Calcaneofibular ligament (CFL) Posterior talofibular ligament (PTFL)
Syndesmotic ligament complex	Anterior inferior tibiofibular ligament (AITFL) Interosseous membrane (IOM) Interosseous ligament (IOL) Inferior transverse ligament (ITL) Posterior inferior tibiofibular ligament (PITFL)
Medial ligaments	Deltoid ligament – Deep component – Superficial component

ankle sprains after ATFL). The posterior talofibular ligament (PTFL) passes backward to insert on the talus.

2. The syndesmosis is a complex of ligaments in between the distal tibia and fibula. This constitutes of anterior inferior and posterior inferior tibiofibular ligaments (AITFL, PITFL), interosseous membrane (IOM), interosseous ligament (IOL), and inferior transverse ligament (ITL). The AITFL originates from the tubercle of Chaput on the tibia and inserts onto the tubercle of Wagstaffe on the fibula. Rotational ankle injuries can cause rupture of this ligament or avulsion injuries of one of its insertions (Tillaux fracture in children). The PITFL originates from posterior tubercle of tibia (Volkmann's tubercle) and inserts onto the posterior malleolus. This is the strongest component of syndesmosis, but high-energy rotational injuries can cause ruptures of this ligament or Volkmann's fractures of the posterior malleolus. The syndesmosis resists axial, rotational, and translation forces and widens 1 mm during the gait.
3. The deltoid ligament is a strong, fan-shaped ligament situated on the medial aspect of the ankle. It originates from the tip of the medial malleolus and fans inferiorly. The deep component inserts into the talus, while the superficial one inserts into the talus and calcaneus.

The role of the deltoid ligament is to stabilize indirectly the medial ankle mortise.

The body of the talus is semicircular in sagittal plane and has a rectangular cross section in coronal plane broader anteriorly than posteriorly. Therefore, the talus fills the mortise fully in dorsiflexion but is looser in plantar flexion, allowing some inversion and eversion. This knowledge is useful when performing and interpreting ankle radiographs.

Epidemiology

Acute ankle sprains are among the most common musculoskeletal injuries [1]. They account for 3–5% of all emergency department visits in the UK, equating to approximately 5600 incidences per day [2]. They have high socioeconomic costs associated with the diagnosis, treatment, and loss of work productivity contingent with the severity of injury. Twenty-five percent of all people who sustain an ankle sprain are unable to attend school or work for more than 7 days following the initial injury [3]. These injuries are common in dancers, athletes, and military population. Female sex, lower age, and athletes competing in indoor and court sports are the subgroups most at risk of ankle sprain [4].

Classification

1. Low ankle sprains are the most common type (>90%)

   They are typically caused by inversion injuries of the ankle affecting the integrity of the lateral ligaments: ATFL (via plantar flexion and inversion), CFL (via dorsiflexion and inversion), and very rare PTFL.

   They can be classified according to the severity, taking into consideration the severity of structural damage, associated clinical features, and the functional impairment generated (as detailed in Table. 29.2). Recurrent ankle sprains can lead to functional instability.
2. High ankle sprains are rare (up to 10%) but more severe; missed injuries may result in end-stage osteoarthritis. These are external rotation-type injuries associated with syndesmotic damage and significant ankle instability.
3. Deltoid ligament sprains are uncommon; they often result from forced eversion of the foot and can be associated with Maisonneuve injury. The Maisonneuve injury is a spiral fracture of the proximal third of the fibula associated with disruption of the distal tibiofibular syndesmosis and the interosseous membrane. There is an associated fracture of the medial malleolus or rupture of the deep deltoid ligament. This type of injury can be difficult to detect.

Ankle sprains are often associated with other foot and ankle injuries: osteochondral defects, peroneal tendon injuries, ankle and foot fractures (fifth metatarsal base, lateral or posterior process of talus, anterior process of calcaneus, deltoid ligament injury, loose bodies), and complex regional pain syndrome. They should be carefully considered when performing clinical examination and requesting further imaging as their presence might change the patient management.

Table 29.2 Table showing the classification of low ankle sprains

Severity of low ankle sprains	Ligament disruption	Clinical appearance	Pain on weight bearing
Grade I	Tearing of the fibres, no disruption	Mild swelling Tenderness over ATFL No laxity on examination No restriction in ROM	None/Mild
Grade II	Tear of the ATFL, partial tear of CFL	Localised swelling, ecchymosis Tenderness antero-lateral aspect of the ankle Mild laxity but stable	Moderate
Grade III	Complete disruption of ATFL and CFL	Severe swelling and ecchymosis Ligamentous laxity Functional loss	Severe

Clinical Presentation

History is an important part of clinical examination and can provide useful information regarding diagnosis, patient functional status, physiological reserve, and expectations. The proposed management should take into consideration all these factors.

Establishing the mechanism of injury can provide useful clues regarding the nature of the injury. Very often simple twisting injuries with the foot inverting or everting under the loaded tibia are described by the patients.

Time of injury is relevant when assessing swelling and establishing a framework for surgical treatment.

Patient age, mobility, level of function pre-injury, and past medical and drug history should all be considered when discussing management options (nonoperative vs. operative).

The most common complaints are ankle pain, swelling, bruising, difficulty or inability to weight-bear, recurrent instability, as well as catching/popping sensation in recurrent sprains.

Clinical examination should be thorough following the orthopedic mantra of look, feel, move technique with added special tests for each category of ankle sprains.

1. Low ankle sprains:

   The clinical examination will reveal swelling and bruising over the lateral malleolus and localized tenderness over the affected ligaments (anterior and inferior to the lateral malleolus) with restricted range of movement due to pain.

Anterior drawer test performed with ankle in plantar flexion for ATFL and dorsiflexion for CFL will show excessive anterior displacement of talus relative to tibia.

Talar tilt test will show excessive ankle inversion compared to contralateral side, suggestive of ATFL and CFL injury.

2. High ankle sprains:

The clinical examination will reveal inability or difficulty weight-bearing, swelling and bruising over the lateral aspect of the ankle, and tenderness on palpation of the anterolateral aspect of the ankle, proximal to AITFL and sometimes medial malleolus.

Squeeze test is performed by compressing the tibia and fibula at midcalf level. The presence of ankle pain at the syndesmotic level is an indicator of a high ankle sprain.

External rotation stress test is performed with the patient in sitting position (hips and knees 90 degrees of flexion) and foot dorsiflexed. Application of external rotation will elicit pain over the syndesmosis.

Fibula translation test is performed with tibia stabilized; application of anterior and posterior drawer forces will cause pain and excessive fibular translation.

Cotton test produces widening of the syndesmosis when pulling fibula laterally.

Imaging

1. Ankle radiographs should be requested as per Ottawa ankle rules if there is any pain in the malleolar zone and bone tenderness:
   (a) Along the distal 6 cm of the posterior edge of the tibia or tip of the medial malleolus
   (b) Along the distal 6 cm of the posterior edge of the fibula or tip of the lateral malleolus
   (c) Inability to bear weight both immediately and in the emergency department for four steps.

The recommended radiographs are weight-bearing AP/lat/mortise (20° of internal rotation) of the ankle, AP/lat of the leg (to exclude a Maisonneuve injury), and AP/lat/oblique views of the foot if any foot injuries are suspected.

The radiological findings in low ankle sprain are limited, and standard ankle radiographs are mainly useful for identifying associated injuries.

An ATFL injury can be suggested by an anterior talar translation on the lateral view. A CFL injury can be identified by performing varus stress views which will assess the degree of talar tilt.

Standard ankle radiographs are very useful in assessing high ankle sprains, and the most common findings are:

(a) Reduced tibiofibular overlap

Tibiofibular overlap is the overlap of the posterior tibia on fibula at the level of incisura (normal >6 mm on AP, >1 mm on mortise).

(b) Increased medial clear space

Medial clear space is the space between the medial malleolus and medial border of the talus, 1 cm below the joint line (normal <4 mm).

(c) Increased tibiofibular clear space

Tibiofibular clear space is the distance between the lateral cortex of the tibia and the medial cortex of the fibula, 1 cm above the joint line (normal <6 mm on AP and mortise).

External rotation stress views can be used to assess the existence and degree of talar shift in high ankle sprains (medial clear space >4 mm, tibiofibular clear space >6 mm).

Contralateral ankle radiographs can be considered when suspecting a normal anatomical variant.

2. CT is not routinely used in the ankle sprains assessment. It can be useful when there is a clinical suspicion of syndesmotic injury with normal radiographs, to screen ankle for associated injuries (osteochondral defects, loose intra-articular bodies, fractures) or postoperatively to assess reduction of syndesmosis after fixation. It is more sensitive than radiographs for detecting minor degrees of syndesmotic injury.
3. MRI has limited value in acute low ankle sprains assessment. It is indicated when pain persists more than 6–8 weeks post injury despite conservative measures. It is more helpful in high ankle sprains with normal ankle radiographs (having high sensitivity and specificity) and also in evaluation of other associated injuries (osteochondral defects, peroneal tendon injury, medial collateral injuries, loose intra-articular bodies).

Management of Ankle Sprains

Low Ankle Sprains

Conservative Management

The primary goals are to manage pain, control inflammation, and protect the joint. The management consists of RICE (rest, ice, compression bandage, and elevation), analgesia (NSAIDs), and early ROM exercises followed by rehabilitation program (ROM exercises, peroneal tendon straightening, proprioception, and activity-specific training). The benefits of functional rehabilitation vs. cast immobilization are supported by multiple RCT and meta-analysis [5, 6]. Nonoperative management has good results in most patients (90%), athletes included [1]. If nonoperative treatment is unsuccessful (persistent symptomatic ankle instability), late reconstruction of the ligaments gives a good outcome [7].

Acute operative management should be considered in patients with an unstable ankle occurring in association with osteochondral fracture, peroneal tendon injury, or evidence of syndesmotic injury.

Chronic operative management is reserved for patients with failed conservative management with persistent symptomatic ankle instability.

There are two types of chronic instability: mechanical and functional.

Patients with mechanical instability complain of ankle giving way and have objective hypermobility of tibiotalar joint on clinical examination. Patients with functional instability complain of ankle giving way, but clinical examination cannot reproduce ankle instability. The patients with mechanical ankle instability are the ones who are likely to benefit from surgery the most.

Surgical management falls in two basic categories: anatomic repair and tenodesis stabilization.

The goal of anatomical repair is to restore the normal anatomy and joint biomechanics.

In Brostrom procedure, suture anchors are used to reattach the shortened and re-tensioned ATFL to its fibular insertion point.

The Gould modification is the reinforcement of the repair by reefing the extensor retinaculum.

Tenodesis stabilization is performed when the lateral ligaments are irreparable. Tendon grafts are to be used to reconstruct the ankle ligaments. Most commonly used tendon is peroneus brevis tendon (Watson-Jones, Evans, Chrisman-Snook procedures). Sacrificing peroneus brevis may result in altered ankle kinematics which could lead to degenerative changes in ankle and subtalar joints. Semitendinosus tendon allograft showed promising results in ATFL and CFL reconstruction without interfering with ankle stability and subtalar motion with decreased predisposition to talar arthritics in short-term follow-up [8].

Common postoperative complications include wound problems, nerve injuries, recurrent ankle instability, stiffness, and impingement. They should all be discussed with patient while gaining informed consent for surgery.

High Ankle Sprains

Sprain without diastasis or instability are best managed nonoperatively using RICE, NSAIDs, and immobilization in a brace of booth with protected weight-bearing as per patient symptoms followed by ankle rehabilitation (range of movement exercises, strengthening, and neuromuscular training).

Sprains with instability but no diastasis should be managed in a non-weight-bearing cast for minimum of 4 weeks with serial weight-bearing radiographs to check position and then progress with WB stats in a protective boot, aiming for FWB at 8 weeks.

Sprains with diastasis of the syndesmosis require surgery.

Aim of surgery is to accurately reduce the fibula within the incisura (the most important factor in successful syndesmotic fixation) [9].

Technique

Using an open lateral incision, incisura is exposed, and fibula is anatomically reduced and rigidly fixed using metallic screws. Placing the screws 2 cm above the tibiotalar joint provides a better stability than placing them more proximally [10]. But even when using quality operative fluoroscopic imaging, postoperative CT scan showed malreduction of the syndesmosis up to 24% [11]. These screws can be removed or left in situ (in which case osteolysis and breakage are likely without affecting significantly patient outcome).

Bioabsorbable screws are increasing in popularity, offer excellent stability, don't require removal, but can cause a local inflammatory process (sterile abscess or cyst) [12].

Semirigid fixation using suture secured with buttons across the syndesmosis has the advantage of maintaining some movement. There are studies showing that semirigid fixation was at least as good as rigid screw fixation [13]. Other advantages associated with their use are less complication, shorter rehabilitation, and faster return to work [14].

Chronic ankle instability causes ankle arthritis, and the salvage procedure remains ankle arthrodesis. Despite being a debilitating procedure, it has been shown to produce good long-term pain relief.

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The Foot and Great Toe

30

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Hallux Valgus

Introduction

Hallux valgus deformity (Fig. 30.1) is characterized by malpositioning of the first metatarsophalangeal (MTP) joint caused by a lateral deviation of the great toe and an apparent medial deviation of the first metatarsal bone.

It is frequently is accompanied with deformity and symptoms in the lesser toes.

Etiology

Multiple Proposed Etiology Includes:

1. Congenital or familial/cultural predispositions and individuals wearing unaccommodative footwear. About 84% of adult patients with hallux valgus have a positive family history with a female:male ratio of 15:1 [1, 2]. Exact etiology remains unclear and may be multifactorial. Other factors which can contribute to the development of hallux valgus deformity are as follows:
2. TMT (tarsometatarsal joint) mobility. As discussed by Doty et al. [3], the first TMT joint mobility is necessary for the increase in intermetatarsal and hallux valgus angle. "Hypermobility" may not be the cause but rather the outcome of hallux valgus deformity.
3. Pes planus may lead to hallux valgus because of increased forefoot abduction that creates a non-physiologic load on the plantar-medial aspect of the great toe during heel rise. The association between pes planus and hallux valgus is controversial.

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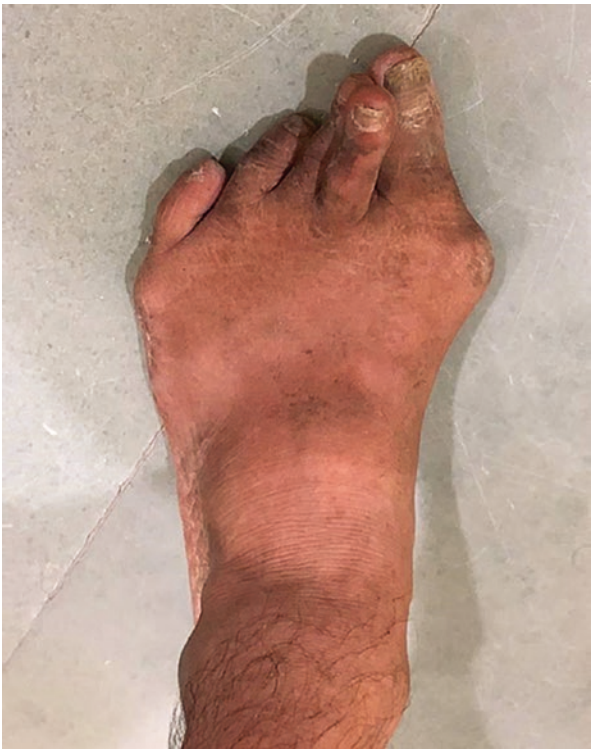


Fig. 30.1 Clinical photograph showing the deformities of valgus at first metatarsophalangeal joint, pronation of the great toe, prominent bunion, widening of forefoot, and lesser toe deformities

4. Shape of first metatarsal head. A "squared" or flattened configuration of the MTP joint may resist valgus forces and limit development of hallux valgus; in contrast, a rounded, concentric shape of the MTP joint may predispose to hallux valgus if a valgus stress is consistently maintained on the hallux.

Pathomechanics

The deviation of the proximal phalanx laterally over the metatarsal head leaves the medial joint surface of the metatarsal head exposed, leading to the bony prominence of the medial aspect of the MTP joint. This bony prominence along with the inflamed medial bursa is commonly known as the "bunion" (Latin word bunion, meaning a turnip).

Repetitive valgus stress at the first MTP joint with weight-bearing leads to laxity of the medial joint capsule and contractures of lateral joint capsule, suspensory ligament of the lateral sesamoid, and adductor tendon. Unbalanced corrective muscular forces particularly the abductor hallucis brevis are negated. As the hallux deviates further laterally, neutral muscle forces such as EHL and FHL now become deforming forces, pulling the toe further into valgus.

Clinical Features

Not all patients with hallux valgus deformity are symptomatic. Pain is located around the medial aspect of MTP joint which is aggravated by motion and tight footwear. Severity of deformity and associated pes planus is evaluated while standing, whereas medial eminence tenderness, joint range of motion, and first ray hypermobility are checked with patient seated. Restricted movement and crepitus suggest degenerative changes in the first MTP joint. Examination should also include other toes, midfoot, subtalar, and ankle joints.

Components of a Hallux Valgus Deformity (Fig. 30.2)

1. Valgus at the first MTP joint
2. Pronation of big toe
3. Medial deviation of the first metatarsal
4. Bunion over the medial aspect of the first metatarsal head
5. Lesser toe deformity including hammertoe, mallet toe, and crossover toe deformities

Imaging

Weight-bearing radiographs in anteroposterior (Fig. 30.3) and lateral views of the foot are recommended for measuring the extent of deformity.

The intermetatarsal (IMA) angle is also seen—the angle between the long axis of first and second metatarsal.

Note that both the angles are increased in hallux valgus deformity, as in this case

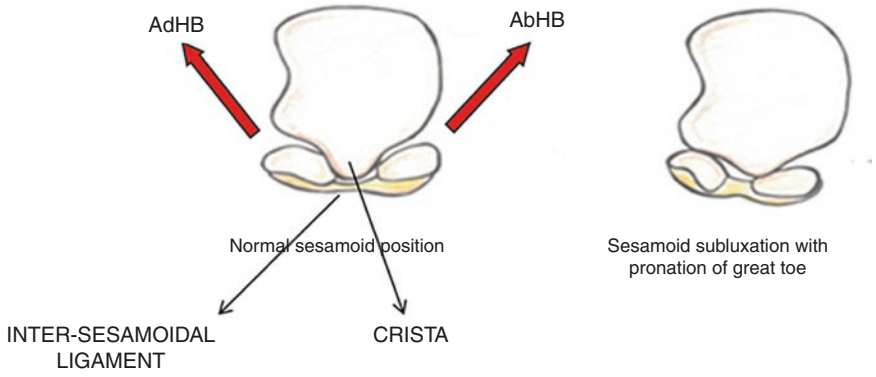


Fig. 30.2 Schematic representation of the axial cut at the level of the plantar sesamoids of first metatarsophalangeal joint showing deformities including pronation and sesamoid subluxation in hallux valgus (*AdHB* adductor hallucis brevis, *AbHB* abductor hallucis brevis). (Diagram credits: Ritika Sharma)

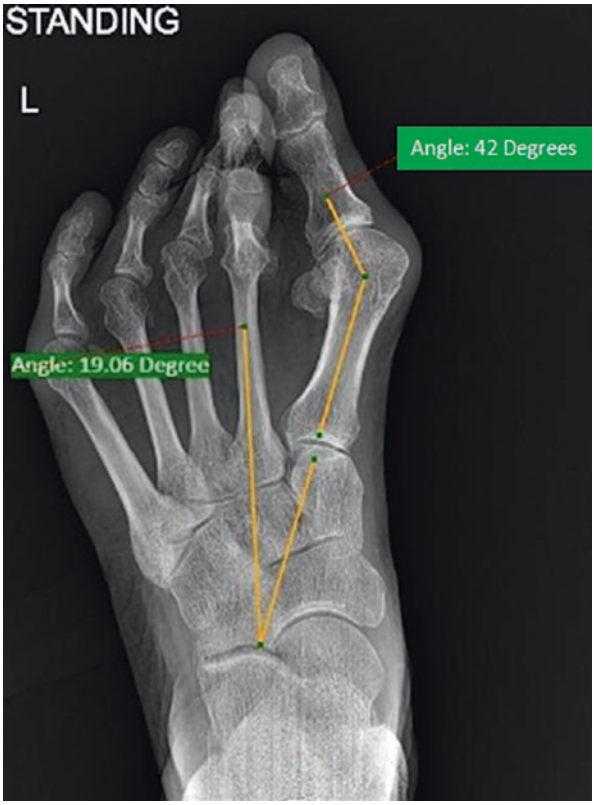


Fig. 30.3 Weight-bearing X-ray of foot anteroposterior view showing the hallux valgus angle (HVA) [the angle between the long axis of the first metatarsal and the proximal phalanx of the big toe] and the Inter-metatarsal angle (IMA) [The angle between the long axis of first and second metatarsal]. Note that both the angles are increased in Hallux valgus deformity, as in this case

An increase in the intermetatarsal angle (IMA), the angle between the long axis of the first and second metatarsal, signifies widening of the forefoot and denotes the severity of the deformity in conditions such as hallux valgus and metatarsus primus varus. The normal IMA measures less than 10°.

The hallux valgus angle (HVA) between the long axis of the first metatarsal and the proximal phalanx is the most commonly used denominator to quantify the severity of the hallux valgus deformity as below. The normal HVA measures less than 15°.

These two angles can also be used to evaluate correction of the deformity in the postoperative X-rays.

Grades of deformity (as per the hallux valgus angle (HVA) [4]

1. Mild: HVA < 20°.
2. Moderate: HVA 20–40°.
3. Severe: HVA > 40°.

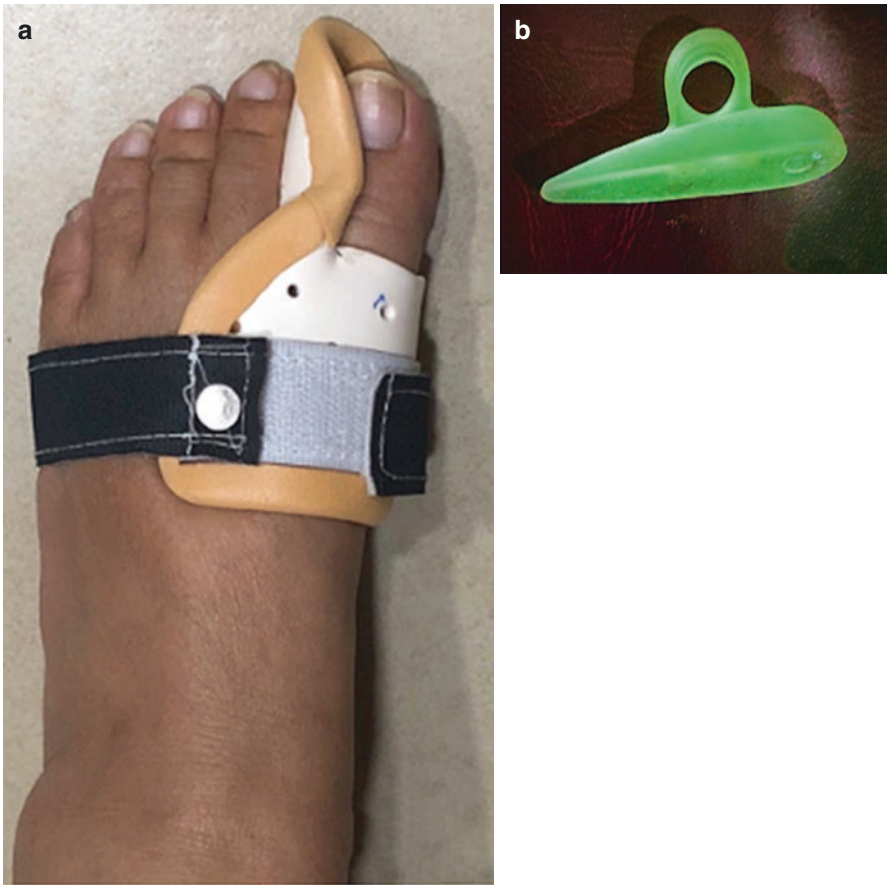


Fig. 30.4 Some of the corrective splints available for hallux valgus. (**a**) Custom moldable splint. This can be used postoperatively as well. (**b**) Silicone toe separator

Management

Conservative Management

Nonoperative management can relieve symptoms and reduce deformity progression but will not produce deformity correction. This includes footwear modifications (wide toe box), medial padding, toe separators, night splints, and insoles (Fig. 30.4a, b).

Operative Management

The progressive and severe deformity benefits the most from surgical treatment. Mild to moderate deformities often can be corrected by distal procedures, whereas severe deformities usually need proximal procedures or arthrodesis. There are a huge variety of procedures described for correction of the hallux valgus deformity, confirming the fact that there is a lack of consensus on the best procedure to be done for a certain patient. Presented below are the most commonly performed procedures for hallux valgus deformity. This is by no means an exhaustive list or description.

Simple Bunionectomy

This basically involves excision of the medial eminence with medial capsular plication. It is sometimes done in symptomatic patients with very early flexible deformity. However, this procedure tends to have a high risk of recurrence, and a McBride procedure or a bony corrective procedure is more frequently used.

Modified McBride Bunionectomy

This procedure consists of medial capsulotomy and plication and a lateral release of the first metatarsophalangeal joint. The lateral release involves release of the tight adductor hallucis and lateral capsule including release of the suspensory ligament of the lateral sesamoid. Occasionally, an excision of the lateral sesamoid is carried out.

This surgery is indicated in symptomatic patients showing no improvement with conservative treatment. It is preferred in cases with mild deformity (HVA of 15–25°, IMA less than 13°), without any degenerative changes of the MTP joint.

Distal Metatarsal Chevron Osteotomy

It was described by Corless in 1976 [5] as a modification of the Mitchell procedure to correct mild to moderate hallux valgus deformity. The procedure consists of:

1. V-shaped osteotomy in the sagittal plane through the metatarsal head and neck (Fig. 30.5) followed by lateral shift of the distal fragment and trimming of the proximal fragment.Medial plication of the capsular flap stitched with the abductor hallucis tendon.

Initially, the osteotomy was described as requiring no fixation because of inherent stability; however, with time, it was noted that fixation with a single screw afforded more stability and allowed earlier weight-bearing and joint mobilization.

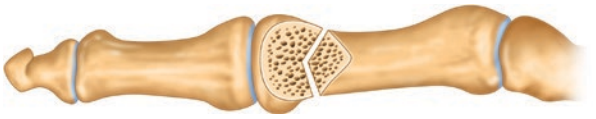


Fig. 30.5 Schematic representation of the lateral view of the big toe showing a distal metatarsal chevron V-shaped osteotomy of the head and neck of the first metatarsal. (Diagram credits: Ritika Sharma)

The chevron osteotomy is generally recommended for patients with a hallux valgus angle of less than 40° and an intermetatarsal angle of less than 20° [5]. A hallux deformity including significant pronation of the big toe is also not suitable for correction with the chevron osteotomy. For all these deformities, the scarf osteotomy is a more suitable option.

Scarf Osteotomy

The scarf osteotomy is a sagittally placed displacement Z-osteotomy involving the metatarsal head, neck, and shaft (Fig. 30.6a, b).

The bony correction is combined with a soft tissue realignment similar to that described in McBride bunionectomy. This biomechanical configuration has a high level of stability, particularly in the horizontal plane, allowing immediate weight-bearing and faster rehabilitation. It also provides a broad surface area for healing of the osteotomy resulting in lower rates of nonunion.

Cadaver studies have confirmed that with weight-bearing the scarf osteotomy is almost twice as stable as a distal chevron osteotomy [6]. It is a very versatile osteotomy, and the angle and length of the cuts can be modified to achieve optimal correction in a wide variety of hallux valgus deformities. It allows lateral displacement of the metatarsal head fragment to correct the IMA, medial displacement of the proximal fragment to correct hallux varus, plantar displacement to increase

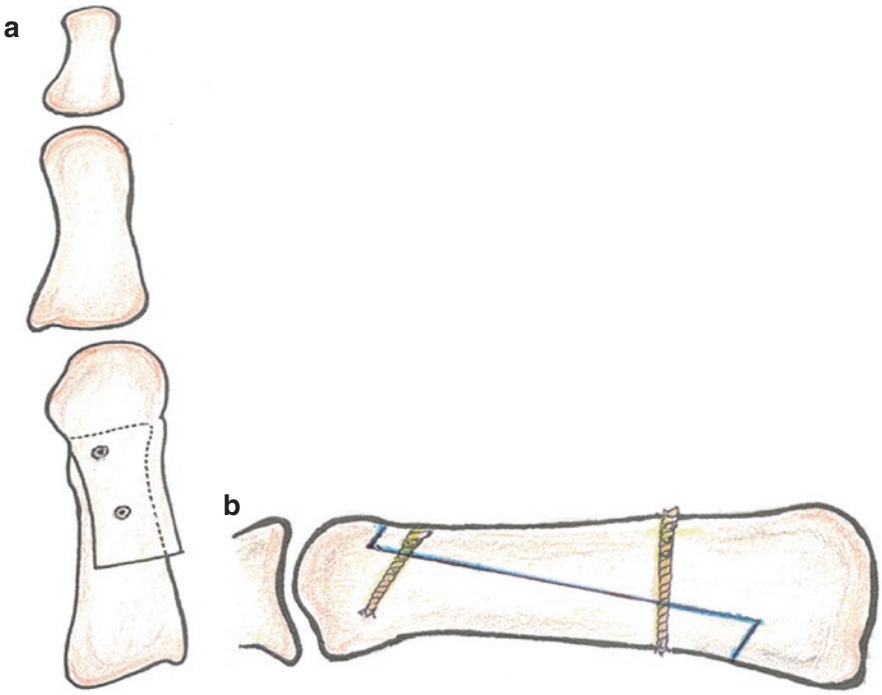


Fig. 30.6 (**a**, **b**) Schematic representation of the Z-shaped osteotomy cut of the scarf osteotomy. (Diagram credits: Ritika Sharma)

weight-bearing of the first ray, and lengthening or shortening of the first metatarsal. A correction of the distal metatarsal articular angle is also possible with additional cuts.

Figure 30.7 shows the surgical technique of the scarf osteotomy.

The scarf osteotomy is combined with a lateral soft tissue release, excision of the medial bony eminence, and medial capsular plication, and occasionally with an Akin's proximal phalangeal basal osteotomy. The Akin's osteotomy allows derotation correction of the pronation deformity as well.

This procedure has a high success rate [7, 8] and is indicated for patients with moderate to severe hallux valgus deformity (HVA 20° and above, IMA 15° and above) and without significant joint degeneration.

Akin's Osteotomy

Akin's osteotomy (Fig. 30.8) is a medial closing wedge osteotomy of the proximal phalanx of the big toe.

It is as previously mentioned, usually done in combination with osteotomy of the first metatarsal in the correction of hallux valgus deformity.

It is a transverse osteotomy done at the base of the proximal phalanx, usually using a small oscillating saw taking care not to penetrate the far cortex (Fig. 30.9). A small medial wedge is removed, and correction of the valgus and pronation is possible. The osteotomy is fixed using a small staple or headless screw (Fig. 30.10a, b).

First Tarsometatarsal Joint Arthrodesis

Also popularly known as the Lapidus procedure, this procedure has gradually come of age as the procedure of preference for patients with severe hallux valgus associated with generalized ligamentous laxity or tarsometatarsal arthritis. It also may be used as a salvage procedure after previously failed hallux valgus correction. One of the most common indications for which it has been used is for first ray hypermobility. However, this indication has been recently challenged as it has been shown that the first ray hypermobility is the consequence and not the precursor of a hallux valgus deformity. Correcting the hallux valgus with a distal osteotomy, such as chevron, significantly reduces the first tarsometatarsal hypermobility [9].

The procedure was popularized by Lapidus, hence the name [10, 11]. It allows a powerful correction of the hallux valgus in the sagittal plane as well as allows significant rotational correction. It is usually combined with a distal soft tissue correction.

It has been shown to return good to excellent results in 71–80% of patients with a low complication rate [12]. The common complications are nonunion, transfer metatarsalgia, and incomplete correction.

First Metatarsophalangeal Joint Arthrodesis

Arthrodesis of the MTP joint is preferred in cases of joint degeneration, failed prior surgical correction, or neuromuscular disorders. This is combined with correction of the lesser toe deformity as indicated. The surgical technique is described in hallux rigidus (Fig. 30.11).

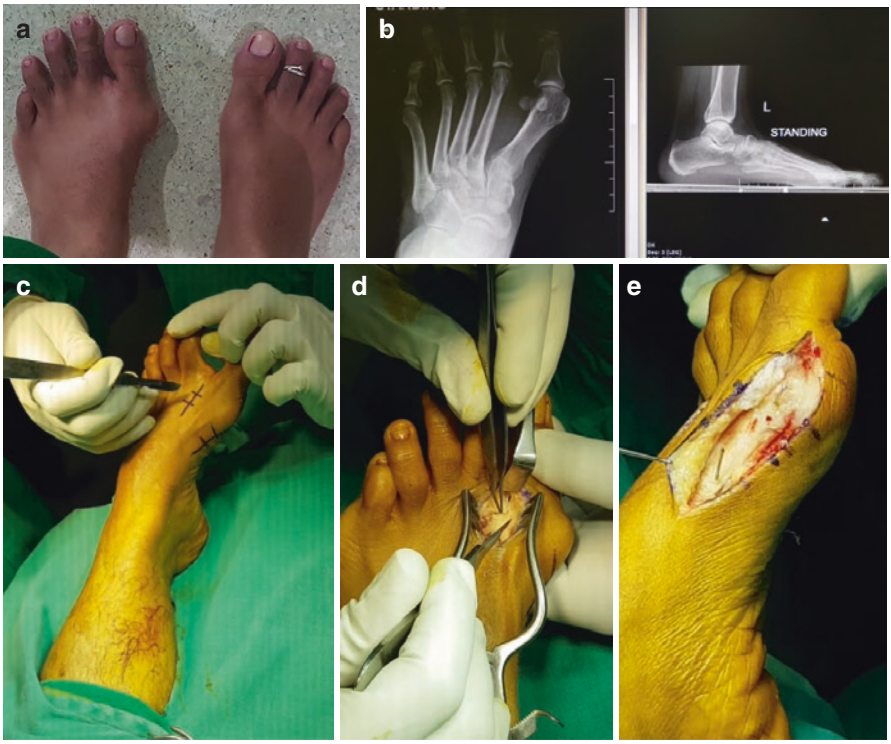


Fig. 30.7 (**a**–**l**) Scarf osteotomy for hallux valgus—surgical steps. (**a**) Preop deformity showing Valgus at MTP joint, prominent bunion and pronation of big toe (**b**) Weight bearing x-rays of the foot, note the subluxation of the sesamoids, the first metatarsal looks adducted and the foot looks widened (**c**) two incisions are typically used (**d**) The first incision in the first webspace at the level of the MTP joint, the sesamoid suspensory ligament and adductor hallucis tendon are released, taking care to avoid injuring the collateral ligament (**e**) Medial incision and sharp dissection through capsule to expose the medial aspect of head and shaft of the first metatarsal. 2 K-wires are inserted to guide the osteotomy and to prevent over-progression of the saw blade (**f**) Metatarsal osteotomy with oscillating saw, taking care that the whole horizontal cut is done in a single plane. Scarf osteotomy is a very versatile osteotomy and various modifications of these cuts have been described to correct varying deformities (**g**) Metatarsal osteotomy completed, note the wide surface area available for healing (**h**) the osteotomy is displaced with the distal fragment moved medially and sometimes rotated, the fragment can be displaced up to 50% of the width of the metatarsal without significantly compromising stability. The osteotomy is fixed with 2 headless compression screws (**i**) The protruding medial eminence is shaved off leaving a smooth profile (**j**) Final intraoperative correction achieved, weight bearing simulation using a flat surface on the plantar aspect should be used to check position on weight-bearing (**k**) Post op x-ray, note the sesamoids are back in place, widening of the foot is corrected. In this case an additional osteotomy of the base of the proximal phalanx (Akin osteotomy) was added to correct residual angulation and pronation (**l**) Final clinical correction at 3 years post-surgery, along with the pre-operative position

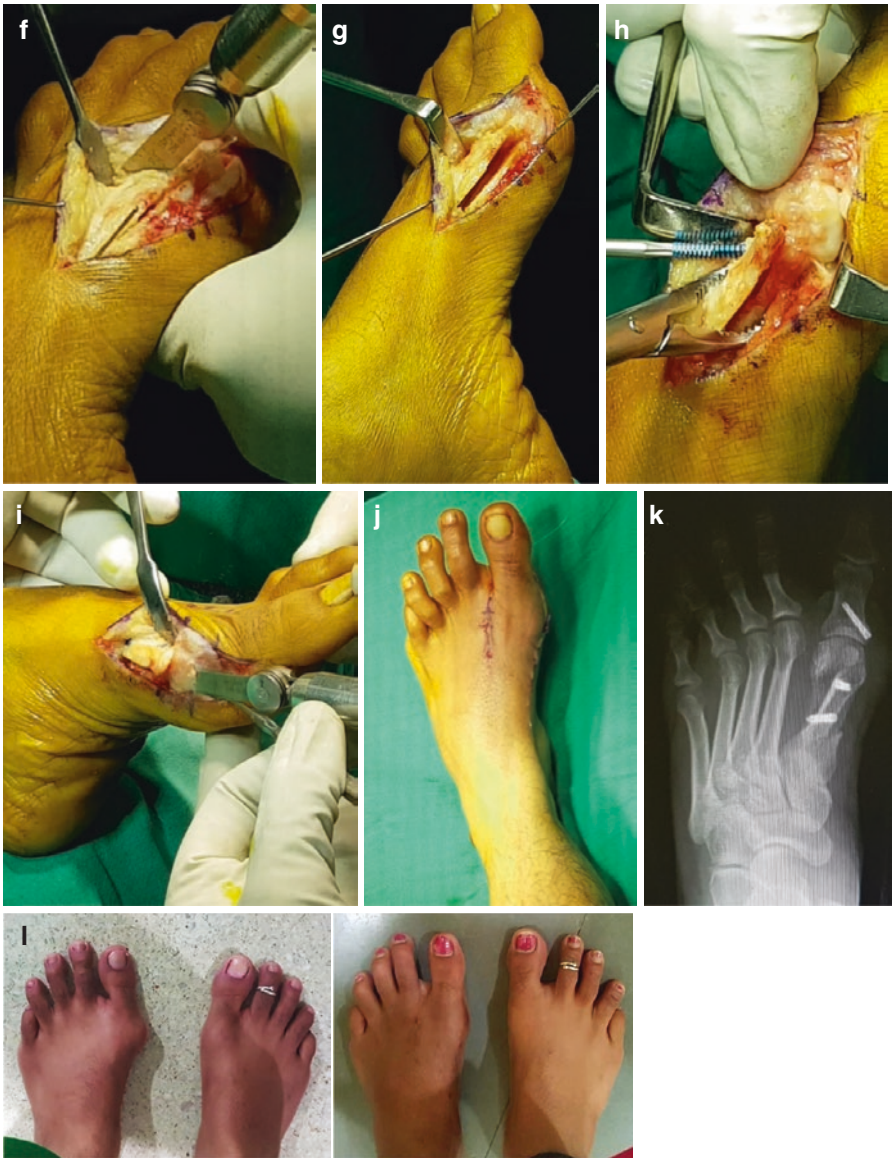


Fig. 30.7 (continued)

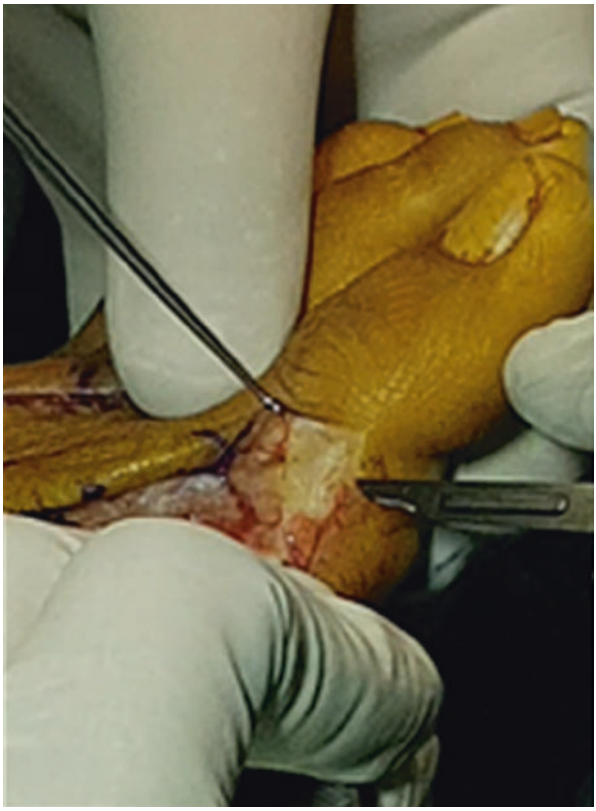


Fig. 30.8 Akin's proximal phalanx base corrective (medial closing) osteotomy: exposure of medial aspect base of phalanx

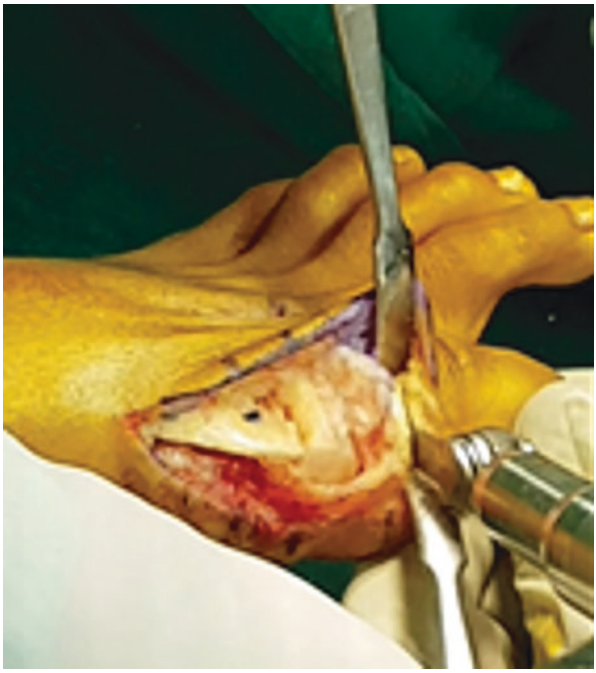


Fig. 30.9 Medial closing osteotomy at base

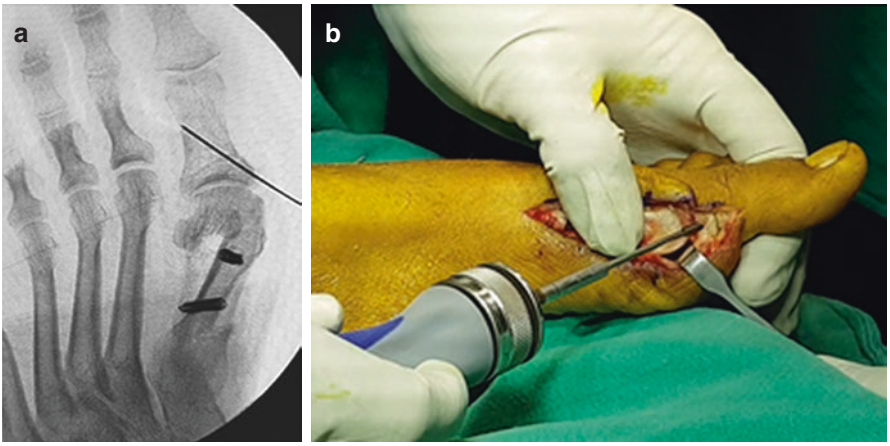


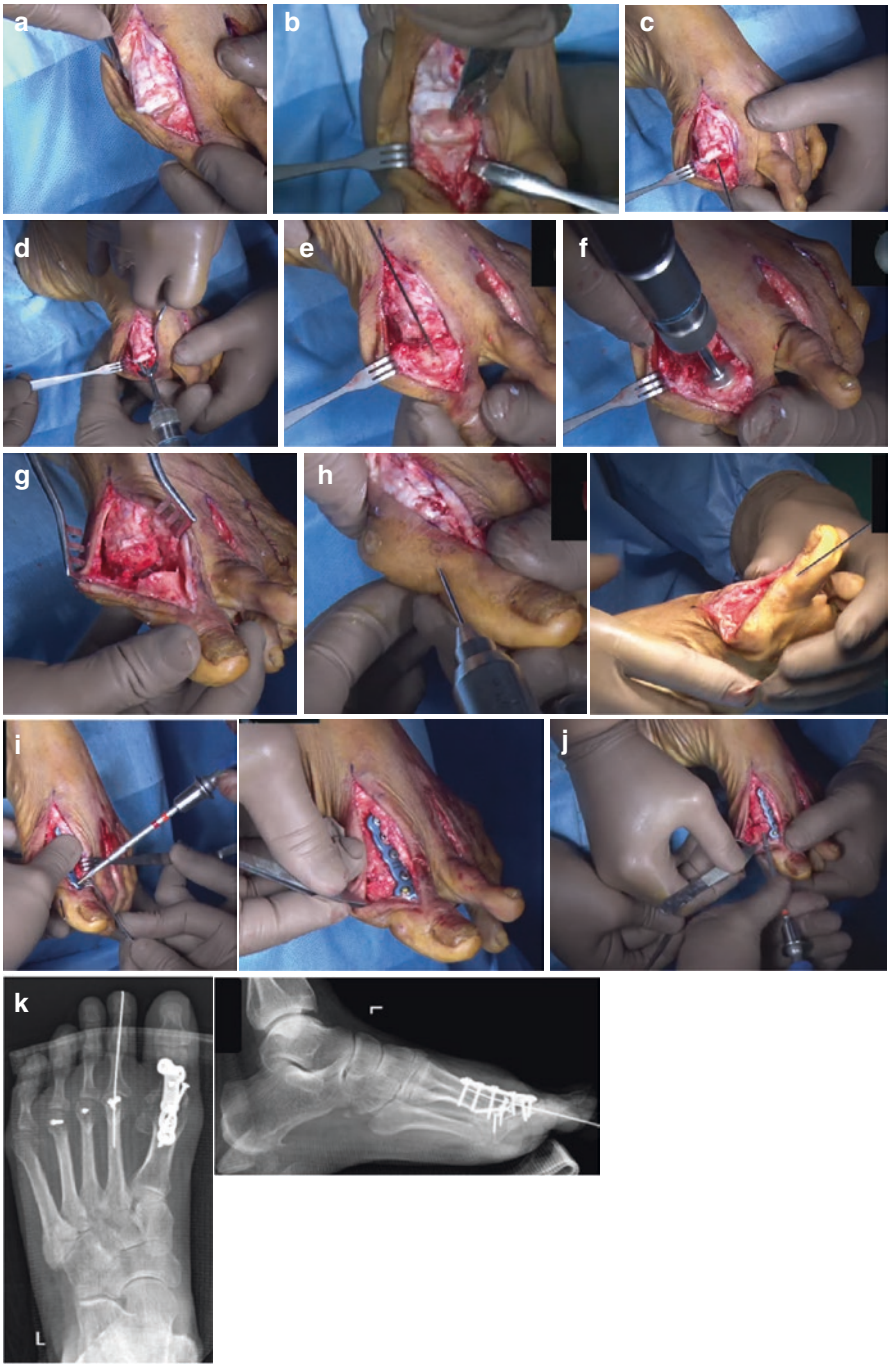
Fig. 30.10 (**a**) Temporarily fixed with K-wire. (**b**) Fixed with compression screw

Minimally Invasive Surgery for Hallux Valgus

Recently, minimally invasive surgery for hallux valgus and forefoot correction is being used as an alternative to open surgery. Various techniques have been described [13–20]. The most popular ones are the minimally invasive chevron and Akin's osteotomies usually fixed with one to two screws and the Reverdin-Isham technique which consists of an intra-articular medial wedge closing osteotomy of the metatarsal. It is usually performed with exostectomy of the medial eminence, Akin's osteotomy, and distal soft tissue release without any internal fixation.

These techniques have shown promise with a recent systematic review evaluating 2279 procedures showing an overall complication rate of 13% [21].

Fig. 30.11 (**a**-**k**) First MTP joint arthrodesis for hallux rigidus—surgical steps for locking plate fixation. (**a**) Dorsal midline incision, dorsal capsulotomy and exposure of the first metatarsophalangeal joint (**b**) Excision of degenerated cartilage from the metatarsal and phalangeal joint surfaces (**c**) Preparation for reaming – Intramedullary k-wire inserted along the long axis of the first metatarsal bone (**d**) Reaming of the distal metatarsal joint surface with a convex reamer to create a dome like end which helps to correct the deformity in sagittal and coronal planes (**e**) Preparation for reaming – K wire inserted along the long axis of the first proximal phalanx (**f**) Reaming of the proximal phalanx base joint surface with a matching concave reamer (**g**) Prepared surfaces for arthrodesis (**h**) Temporary fixation with k wires, alignment of the toe is neutral horizontal plane alignment with 5–10 degrees of dorsiflexion (**i**) Fixation with a pre-contoured locking plate, this has inbuilt dorsiflexion (**j**) Supplementary trans-articular oblique screw fixation (**k**) Post-operative x-ray showing the locking plate fixation for first MTP joint arthrodesis for Hallux rigidus. Lesser toe corrections in the form of Weil's osteotomies of 2–4 metatarsals and PIPJ fusion of the second toe were also required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Hallux Rigidus

Introduction

Latin—hallux—great toe; rigidus—rigid

It refers to the arthritis of the first metatarsophalangeal (MTP) joint of the big toe characterized by progressive pain and stiffness.

It may be bilateral and is commonly a problem of the middle aged or elderly.

Etiopathology

Multiple etiologies have been proposed which include post-traumatic, inflammatory, or metabolic conditions such as rheumatoid arthritis and gouty arthritis.

Clinical Presentation

Pain and swelling over the plantar aspect of the first MTP joint are usually the presenting symptoms with progressive disease. With time, the patient prefers to not take weight over the first MTP joint. The nonfunctional first MTP joint leads to the lesser metatarsal heads progressively taking more weight and collapse of the transverse metatarsal arch, also called as transfer metatarsalgia. This leads to formation of calluses on the plantar aspects of the lesser MTP joints.

Tenderness, crepitus, and terminal restricted range of motion may be observed in the initial stages. As the disease progresses, the joint gets progressively stiff and painful. Gait may be antalgic with reduced push off.

Management

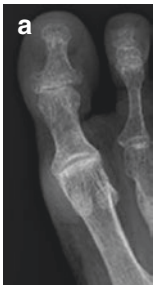
Investigations

Plain radiographs (Fig. 30.12a, b): Weight-bearing AP and lateral views and oblique views of the foot are the standard radiographs to assess the deformity and degeneration of the joint.

Aim of treatment is to relieve pain and improve function of the patient.

Conservative Management

Conservative treatment is attempted in all newly diagnosed cases and early stages of the condition. It includes activity restriction, shoe wear modification, foot orthoses, and local steroid injections. Almost half of patients improve with conservative measures.



b Classification – Based on radiology
1- Mild osteophyte formation with joint space preservation.
2- Moderate osteophyte formation with narrowed joint space and sclerosis.
3- Marked osteophyte formation with severe loss of joint space and subchondral cysts.

Fig. 30.12 (**a**) Plain radiograph of foot AP view showing reduced joint space with subchondral sclerosis of the MTP joint in a case of hallux rigidus. (**b**) Table showing radiological classification of hallux rigidus [22]

Operative Management

This includes two broad categories: joint salvage procedures (dorsal cheilectomy, metatarsal/phalangeal osteotomies) and joint destructive procedures (arthrodesis, resection arthroplasty, interpositional arthroplasty, and implant arthroplasty).

Choice of correct surgery depends upon age, extent of disease, activity level, and associated comorbidities.

Cheilectomy

This procedure entails resection of dorsal osteophytes from the base of the proximal phalanx and metatarsal head, removal of loose bodies, synovectomy, and release of medial and lateral capsular ligaments. This surgery debulks to improve joint motion without compromising stability.

It has been shown to give good results, especially in the early stages of the disease when the main complaint of the patient is loss of dorsiflexion of the big toe. However, a cheilectomy in the later stages of the disease is not as fruitful and may even increase the symptoms in some cases.

Osteotomies

Proximal Phalangeal Osteotomy

Dorsal closing wedge osteotomy of the proximal phalanx places the MTP joint in dorsiflexion, shifting the arc of MTP joint motion dorsally, improving the functional arc of movement. It may be combined with cheilectomy but has insufficient evidence.

Metatarsal Osteotomy

This aims to decompress the joint by shortening and realigning the articular surface leading to functional improvement of pain and stiffness.

Arthroplasty

Keller Resection Arthroplasty

This involves the resection of the base of the proximal phalanx for the treatment of hallux valgus with associated osteoarthritis of the first MTP joint. This is a radical surgery which decompresses the joint with the drawback of instability. At present, the only indication for this procedure is a severely painful joint in low-demand patients. Transfer metatarsalgia and cock-up deformity are common complications.

Interpositional Arthroplasty

This procedure combines resection of the joint and placement of a biological spacer, e.g., extensor hallucis brevis tendon, hamstring tendons, and fascia lata [23]. The procedure is known to give partial relief of symptoms and may be used in low-demand patients.

Total Prosthetic Replacement Arthroplasty

Various types of prosthetic replacement implants have been used over the years with gradually improving long-term results. However, the problems of implant loosening, synovial inflammation, and residual pain are significant and may require conversion to arthrodesis of the MTP joint as a salvage procedure. The various implants which have been used are Silastic, double-stemmed prosthesis, silicone implants, and metallic uncemented prosthesis.

Arthrodesis

Arthrodesis of the first metatarsophalangeal joint is the operative procedure of choice for severe hallux rigidus, for failures of prior hallux valgus or rigidus correction, and for young active patients. The main indications for surgery are pain [22] and antalgic gait, and the aim is to restore pain-free mobility and to maintain stability of the first ray [23].

The surgical technique of first MTP joint arthrodesis using a pre-contoured locking plate is described in Fig. 30.11. Note that any deformities of the lesser toes are to be corrected at the same time, keeping in mind that there may be some shortening of the first ray. Locking plate fixation is preferred in osteoporotic patients and allows early weight-bearing.

Other options for fixation of the arthrodesis are cross 3.5 cortical or headless screws.

Complications of this procedure are nonunion in up to 10–13% and malalignment. Delayed complications like interphalangeal joint arthritis and metatarsalgia may also be seen [22–24]. Overall, it is a very reliable procedure and remains the gold standard for treatment of hallux rigidus.

Metatarsalgia

Introduction

Generalized or localized forefoot pain around the metatarsal heads is called metatarsalgia. It usually arises from either mechanical or iatrogenic causes. Identifying the cause is the key toward successful treatment.

Biomechanics

Repetitive abnormal loading of the lesser metatarsal is the fundamental pathomechanics. This overload of weight-bearing forces may affect entire forefoot or an isolated (e.g., a metatarsal head) when the foot is plantigrade.

Primary Metatarsalgia

This refers to symptoms arising from intrinsic abnormalities in the foot biomechanics leading to overload of the affected metatarsal. This includes disproportionately long or plantarflexed metatarsal relative to the remaining rays of the forefoot. Other conditions associated with this are:

- Insufficiency or painful abnormalities of the first ray
- Plantarflexed metatarsal
- Metatarsal length discrepancy
- Equinus deformity or isolated gastrocnemius tightness

Secondary Metatarsalgia

Systemic conditions that can lead to secondary metatarsalgia include the following:

- Inflammatory arthropathy (gout, rheumatoid arthritis).
- Trauma
- March fracture
- Freiberg infarction
- Neurological disorders, i.e., peripheral neuropathy
- Morton's neuroma
- Tarsal tunnel syndrome

Iatrogenic Metatarsalgia

It results secondary to hallux valgus or other forefoot/midfoot surgery which results in increase in pressure over the lesser metatarsal heads which includes:

1. Excessive shortening of the second metatarsal
2. Excessive elevation of the first metatarsal during hallux valgus surgery
3. Metatarsal head excision

Common pathologies causing metatarsalgia are described in the following sections:

1. Morton's neuroma
2. March fracture
3. Freiberg infarction

Morton's Neuroma

Introduction

Morton's neuroma is entrapment neuropathy of the interdigital nerve of the forefoot. It occurs due to the compression of the nerve at the transverse intermetatarsal ligament. It is associated with perineuritis, fibrosis, and thickening and is not exclusively a neuroma.

It is frequently present in females in fourth and fifth decade and in younger athletes around twenties.

Patho Anatomy

The four plantar common interdigital nerves emerge from branches of the medial and lateral plantar nerves. The transverse intermetatarsal ligament attaches to the plantar plates of the adjacent MTP joints. The interdigital nerve can be possibly compressed under the intermetatarsal ligament, leading to the formation of neuroma.

The third common interdigital nerve is potentially larger since frequently it has a dual innervation from the medial and lateral plantar nerves (Fig. 30.13). The third web space is the most common site of development of a neuroma.

Etiology

Exact etiology is unknown; however, possible theories proposed include the following:

- Compression underneath the distal edge of transverse metatarsal ligament exaggerated with toe dorsiflexion
- Repetitive microtrauma or bursitis leading to fibrosis and nerve entrapment

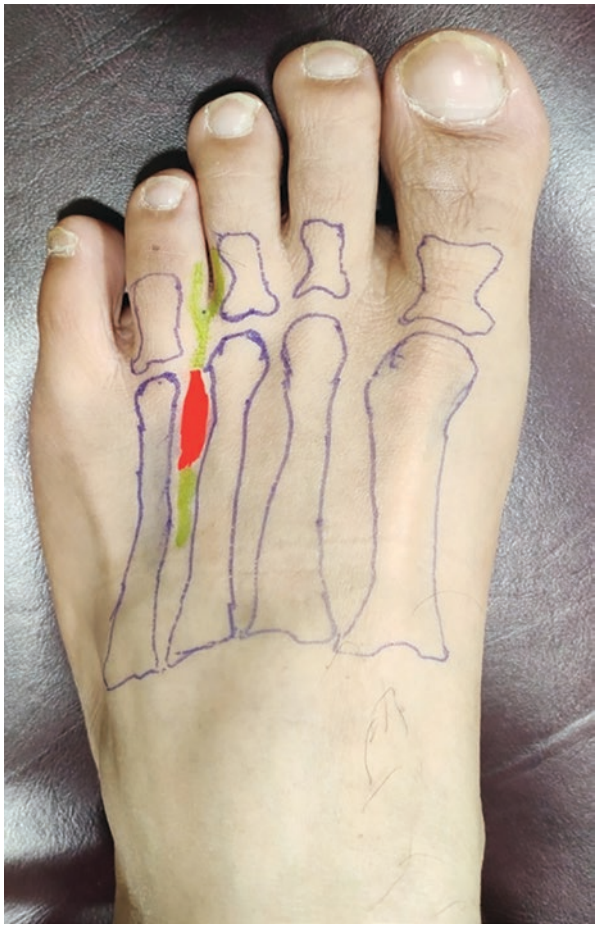


Fig. 30.13 Schematic drawing showing the anatomic location of Morton's neuroma

- Subluxation of MTP joint and nerve stretching
- Compression from a space-occupying lesion such as a ganglion or a cyst is an important differential diagnosis.

Clinical Features

Pain along the specific web space which may be associated with tingling and burning sensation or numbness.

Symptoms aggravated with wearing tight shoes.

On Examination

Tenderness and paresthesia with plantar pressure directed in between metatarsal heads—precipitating the symptoms.

Reduced skin sensation over the affected toe tips.

Compression test—applying dorsal and plantar pressure over the web space with thumb and index finger with squeezing of the metatarsal heads at the same time recreates the symptoms. This confirms the diagnosis and location regarding which web space is involved.

Investigations

MRI has a limited role to check size and extent of large neuromas and exclude other pathology.

Ultrasonography is helpful to locate an oblong mass, although it is operator dependent.

Local lignocaine Injection. Injection of lignocaine around 2 cm proximal to metatarsal head under the transverse intermetatarsal ligament causing resolution of symptoms may confirm the diagnosis. Improvement of symptoms following an injection indicates a potential positive outcome following nerve resection.

Nerve conduction studies may be helpful to rule out other neuropathies.

Treatment

Initial treatment should be conservative and consist of the following measures:

Footwear modifications—Shoes with a wide toe box or customized off-loading insoles (Fig. 30.14) may be tried.

Anti-inflammatory medications frequently help.

If the conservative measures fail to improve symptoms, further intervention may be required as follows:

- Radiofrequency ablation (RFA) is a minimally invasive intervention with good results.
- Neurectomy with/without intermuscular burial of nerve ends with the release of intermetatarsal ligaments (Fig. 30.15).

This has success rates of around 80% [25–27].

March Fracture

Introduction

Repetitive stresses over normal bone result in fatigue/stress fractures. Metatarsal stress fractures are commonly seen in military personnel and known as march fractures.

The usual location of these stress fractures is diaphyseal, although it can be at the metaphyseal-diaphyseal junction also, especially in the first and fifth metatarsals (Fig. 30.16a) [28, 29].

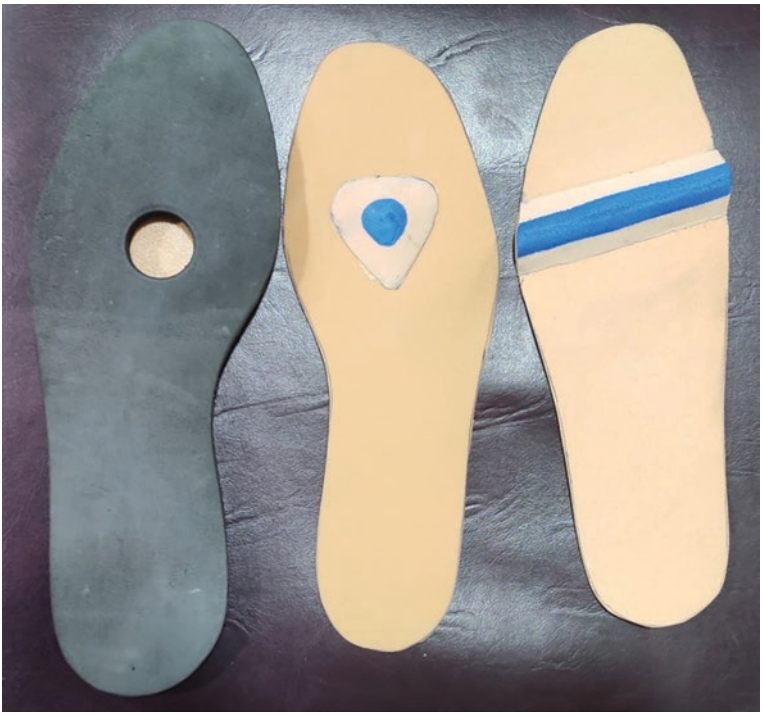


Fig. 30.14 Photograph showing the various custom-made foot insoles used for off-loading the painful area in various conditions causing metatarsalgia-like Morton's neuroma, March fracture etc.

Incidence

Metatarsal stress fracture comprises 15–20% of all lower limb stress fractures.

The second metatarsal is most commonly affected (Fig. 30.16b). Recurrence of these fractures is rare.

Risk Factors

The following conditions have been shown to be associated with a higher incidence of metatarsal stress fractures:

- Malalignment of the foot such as cavus foot or flatfoot. Cavus foot usually leads to overloading of the lateral border of the foot and is associated with stress fractures of the fourth and fifth metatarsals.
- Shorter first metatarsal.
- Barefoot walking.

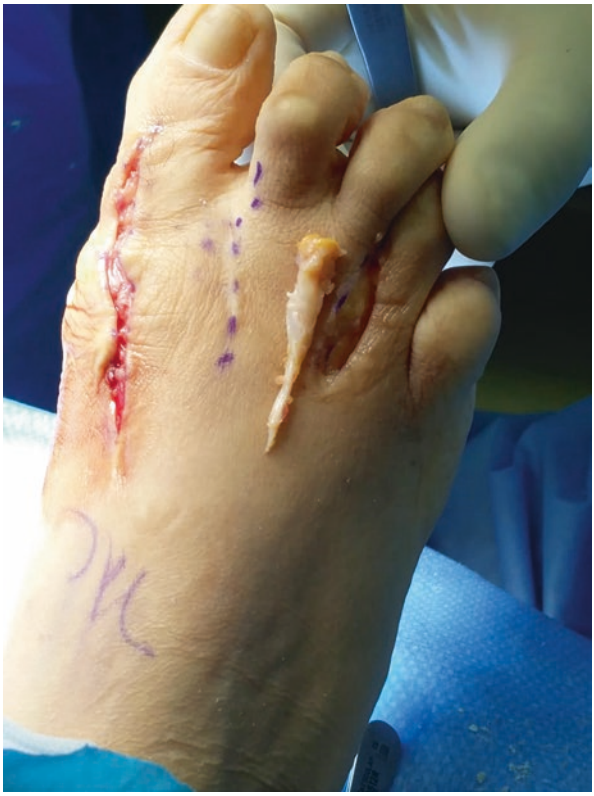


Fig. 30.15 Photograph showing the excised Morton's neuroma from the third web space. Note the enlarged and bulbous neuroma (Courtesy: Dr. Ajoy SM, Bangalore, India)

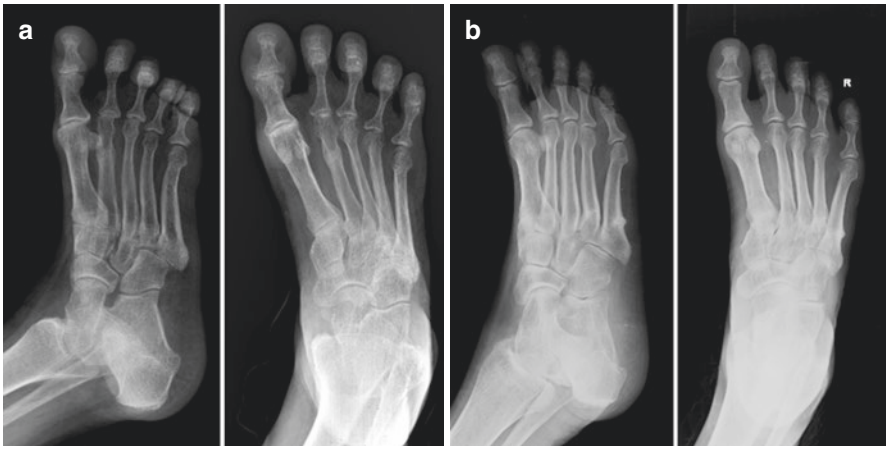


Fig. 30.16 (**a**) A hallux rigidus associated with stress fracture of the second metatarsal. (**b**) X-rays of foot showing sclerosis and periosteal reaction at the proximal metaphyseal area of the fourth and fifth metatarsals indicating healing stress fractures

- Occupation involving high-impact activities such as military personnel and ballet dancers.
- Osteoporosis, endocrine disorders, and inflammatory arthritis are other risk factors.

Pathology

Baker suggested that repeated stress over bone leads to microdamage to the bone and stimulates the periosteum which increases bone turnover causing remodeling of bone over time. Continued stress which overpowers the remodeling process leads to a fatigue fracture [30].

Clinical Presentation

The patient usually presents with pain over midfoot/forefoot which aggravates on activity. A swelling or a lump over the dorsal part of the foot is usually evident after the first 3 weeks. Progressive pathology leads to difficulty in walking or running associated with limping.

Investigations

X-rays are usually normal in the early stages and only show changes once the callus is evident. This happens after 3–4 weeks of onset of symptoms (Fig. 30.16c).

Stress fractures in the first metatarsal usually present with sclerosis surrounding the fracture in linear manner. Fractures involving the lesser metatarsals usually show periosteal reaction with callus formation.

If there is clinical suspicion of a stress fracture but the radiographs are normal, MRI can help delineate the exact pathology. A CT scan can help in diagnosis and is mainly used for follow-up to evaluate healing of the fracture (Fig. 30.17).

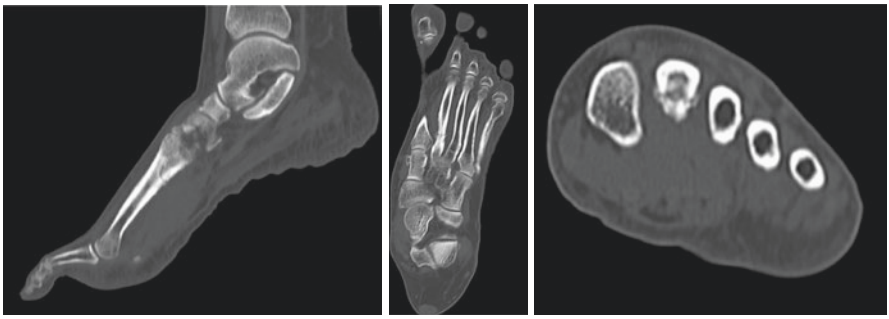


Fig. 30.17 Sagittal, coronal, and axial images of CT scan of foot showing a healing stress fracture of the base of the second metatarsal

A video-analyzed running assessment with gait analysis can give useful information regarding impact forces and give clues regarding possible mitigating interventions.

Treatment

Treatment includes rest and limitation in activity. As patients improve clinically and radiologically, a gradual return to activity can be planned.

Wearing of well-padded shoes such as sports shoes and off-loading insoles are useful to reduce impact. Occasionally, immobilization in a CAM walker boot is helpful in the earlier stages. There is no clear evidence to support the use of calcium supplements, bisphosphonates, or vitamin D in these cases.

Surgical Management

There are limited indications for surgical intervention in these cases. Significant dorsiflexion deformity at the level of the fracture is one of the indications for early surgery. Surgery avoids potential complications of malunion, transfer metatarsalgia, and recurrence. Other indications for surgery include malunion causing a plantarflexed toe and nonunion.

Freiberg Infraction

Introduction

This condition is also known as osteochondrosis of the metatarsal head. It is more common in the adolescent age group, with a male to female ratio of 1:5. The second metatarsal is the most commonly affected. The phases of osteochondrosis signifying the progression of the disease are shown in Fig. 30.18.

Etiopathogenesis

Etiopathogenesis of the condition is still unclear. Freiberg suggested the concept of repetitive microtrauma, especially in cases with long second metatarsal leading to ischemic necrosis of the metatarsal head [31]. High-heeled shoes are implicated to cause repeated forced dorsiflexion and impingement of MTP joints dorsally [32]. Another theory suggested is the possibility of vascular compromise of the metatarsal head leading to ischemia and collapse of the articular surface. Risk factors associated with this condition are trauma, systemic disorders, and genetic predisposition.

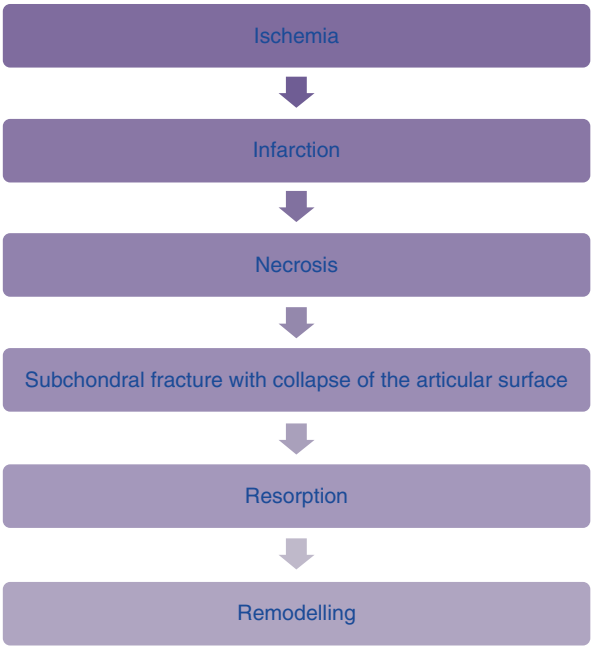


Fig. 30.18 Phases of osteochondrosis of second metatarsal head

Clinical Features

Patients usually present with pain and swelling over the affected joint which are aggravated with activities. Examination reveals swelling and synovial thickening along with painful restricted movement of involved MTP joint. Bony deformity is seen due to remodeling [33].

Radiology

Plain AP and oblique X-rays of the foot show sclerosis of the metatarsal head in the early phase and collapse in the later phases (Fig. 30.19).

Smillie's radiological staging classifies the progressive disease into five stages [34]:

Stage 1: Ischemic epiphysis progresses to a fissure with subchondral sclerosis. Increased joint space due to synovitis is seen.

Stage 2: Subchondral bone collapse occurs due to resorption of the metatarsal head associated with changes in the articular surface.

Stage 3: Central collapse further increases with peripheral cartilage irregularities, and the plantar articular cartilage of head of metatarsal remains intact.

Stage 4: Complete damage of articular surface, fracture, and loose body formation.

Stage 5: Deformity and arthritis.

Nuclear imaging and magnetic resonance imaging (MRI) are helpful to confirm the diagnosis.

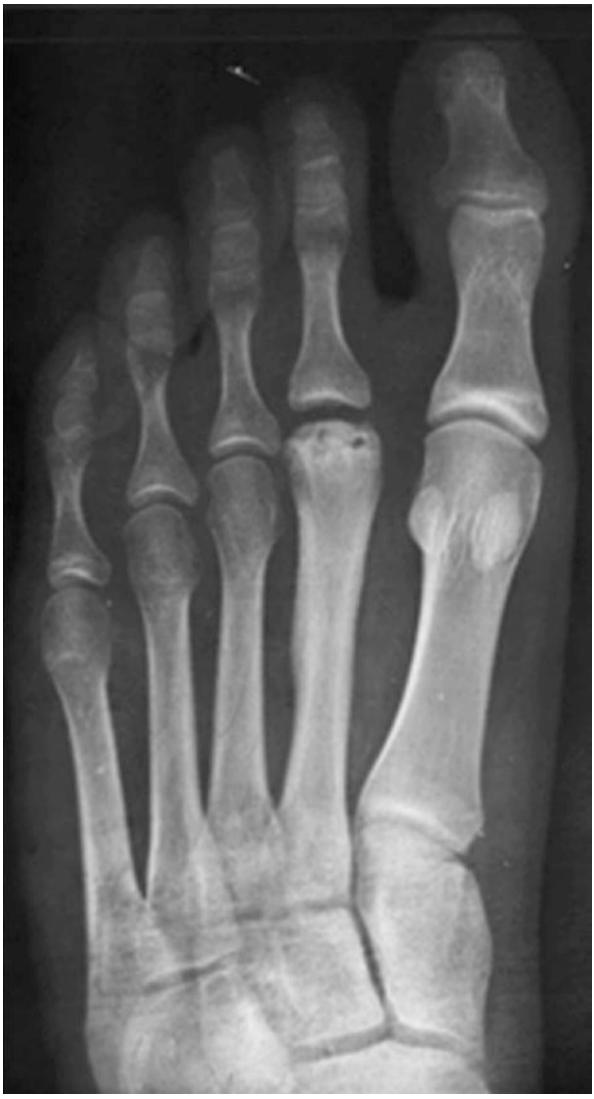


Fig. 30.19 X-ray of foot AP view showing subchondral sclerosis of head of second metatarsal along with osteopenia and loss of contour of second metatarsal head, features suggestive of Freiberg Infraction. (Image taken from your book. Courtesy: Magdi E. Greiss, Whitehaven, Cumbria, UK)

Management

Conservative Treatment

In the initial stages, the aim is to reduce the stress over metatarsal head and MTP joint with activity modification; protection in a CAM walker boot or short-leg walking cast with analgesics are sometimes required.

Surgical Management

Surgical intervention is indicated in a symptomatic patient with joint collapse. In early stages (stage 2), debridement of the joint and excision of the

proliferative bone usually give satisfying results. In stage 3–4 disease, the joint is more involved and the options are:

- Core decompression.
- Osteochondral plug transplantation with or without external fixation to unload the joint.
- Metatarsal osteotomies—Two types of osteotomies are described:
  - Shortening osteotomies reduce stress over the metatarsal head and the articular surface.
  - Dorsal closing wedge osteotomy allows the normal plantar articular surface to be redirected toward the joint [35].

For stage 5 disease, all the above procedures are options, and for severe disease, arthrotomy and debridement with tendon interpositional arthroplasty can also be done.

Sesamoids of the First Metatarsophalangeal Joint

Introduction

Sesamoids of great toes are present under the first metatarsal head located within the flexor hallucis brevis tendons connected to each other with the intersesamoidal ligament.

The stability of sesamoid motion is guided by articulation with grooves over plantar aspect of first metatarsal separated by a central crista (Fig. 30.20a, b).

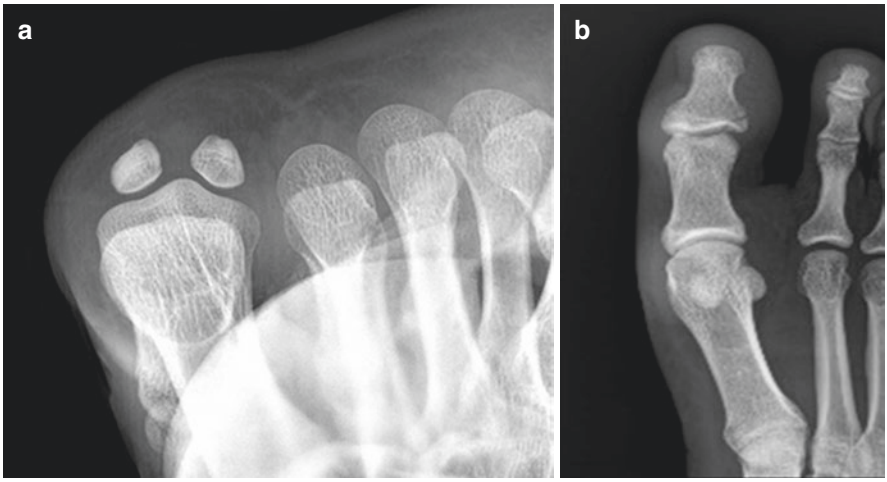


Fig. 30.20 (**a**) X-ray sesamoid view of first MTP joint showing tibial and fibular sesamoids with intervening crista. (**b**) X-ray of foot oblique view showing normal position of sesamoids

Abductor hallucis and adductor hallucis muscles attach over medial and lateral sesamoid, respectively. Sesamoids are held in position medially and laterally by the collateral ligaments of the MTP joint and the sesamoid suspensory ligaments [36].

The sesamoids ossify within 6–7 years of age with multiple ossifying centers.

Functions of the sesamoids of the first MTP joint are as follows [37]:

- Absorb weight and pressure over the first ray.
- Protect the flexor hallucis longus tendon.
- Give mechanical stability to the first ray by altering the direction of muscle pull and reducing friction.

Pathologies Associated with Sesamoids

Absence of the Sesamoids

Absence of both the sesamoids is extremely rare and usually asymptomatic [38, 39].

Bursitis

Around one-third of the normal population have a bursa under the first metatarsal head. Inflammation of the bursa is common in a few conditions like arthritis of first MTP joint, cavus foot, and excessive standing [40]. Treatment usually focuses on off-loading the first metatarsal head by means of custom insoles or orthotic devices.

Bipartite Sesamoids

Around 20–30% of sesamoids are bipartite, and of these, around 90% are bilateral [41, 42].

Bipartite sesamoids (Fig. 30.21) have intervening articular cartilage between the two pieces; this usually serves as a source of symptoms because of increased chances of overuse and traumatic injury. The medial sesamoid is more commonly bipartite and is a common site of the injuries. Usually, these patients ambulate with weight-bearing on the lateral aspect of the foot to avoid motion and loading of the sesamoid complex.

Radiology: Increased gap between the two sesamoid fragments can be a confirmatory finding in a disruption of bipartite sesamoid (Fig. 30.21). MRI usually shows increased marrow edema and raised vascularity which indicates disruption of a bipartite sesamoid [41].

Fracture of the Sesamoids

Sesamoid fractures are a rare entity. A symptomatic bipartite sesamoid is often common differential diagnosis. The mechanism of injury is usually a direct impact either due to crush injury or fall, leading to sudden loading of the forefoot and MTP subluxation causing the fracture.

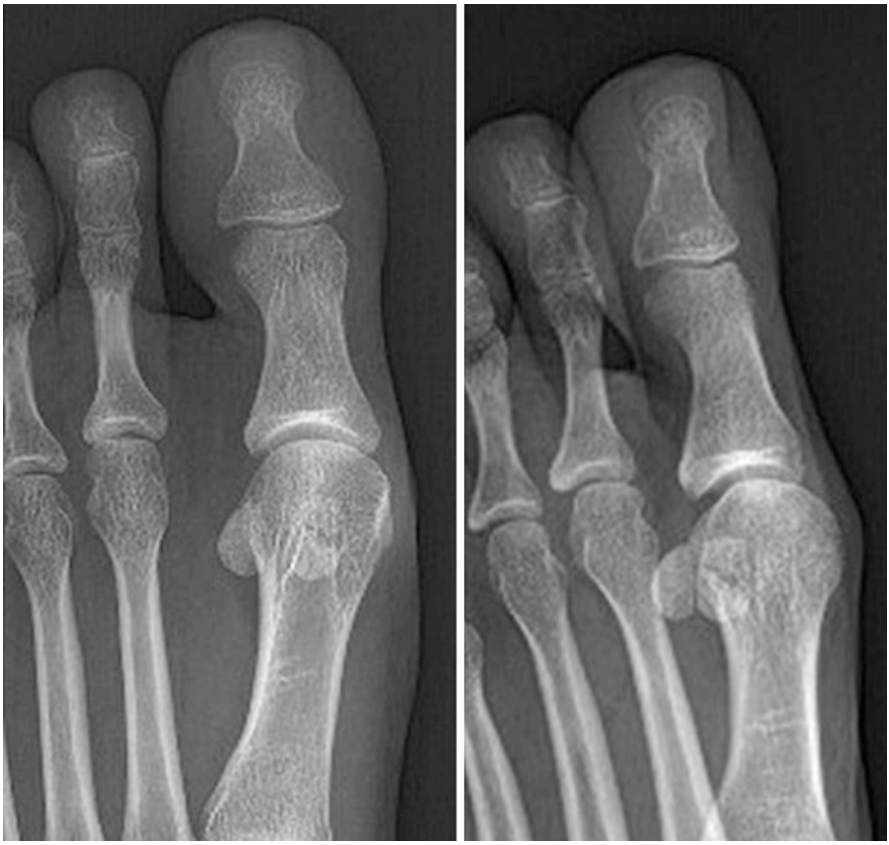


Fig. 30.21 Foot X-rays AP and oblique views showing radiolucent transverse line with smooth edges in the medial sesamoid suggestive of a bipartite sesamoidPatients usually present with painful dorsiflexion of the first MTP joint

X-rays show a sharp radiolucent line which differentiates sesamoid fractures from a bipartite sesamoid. The bipartite fragments usually have more rounded edges.

Delay in diagnosis of a sesamoid fracture is therefore common because of the difficulty of confirming the fracture by radiography.

Treatment is non-weight-bearing in boot cast/plaster until the sesamoid fracture has healed, which usually occurs in 6–8 weeks [43]. Symptomatic tibial sesamoid nonunions may require treatment with bone grafting. Sesamoidectomy is also a valid option for symptomatic patients with articular fracture.

Biedert et al. [44] suggest excision of the medial sesamoid in stress fractures found in athletes with 6–8 weeks of immobilization improves symptoms and allows return to sports activity.

Arthroscopic excision [45, 46] has the benefits of less postoperative pain, reduced hospital stay and duration of postoperative pain and stiffness, better cosmetic results, and early rehabilitation.

Ferkel and Scranton [47] suggest arthroscopic exploration of the metatarsophalangeal joint is a feasible option for symptomatic patients failing conservative treatment.

Sesamoid Dislocation

Sesamoid subluxation (Fig. 30.22a) is commonly seen with hallux valgus deformity.

The medial deviation of the first metatarsal head is mainly responsible for sesamoid subluxation.

Patho Anatomy

As the deformity of hallux valgus increases, apparent medial deviation of the first metatarsal along with pronation of the great toe occurs. This leads to increased weight-bearing over the tibial sesamoid further exacerbating the deformity and pushing the sesamoid laterally [48].

Over time, the sesamoid crista gradually erodes, and sesamoids dislocate out of the place (Fig. 30.22b).

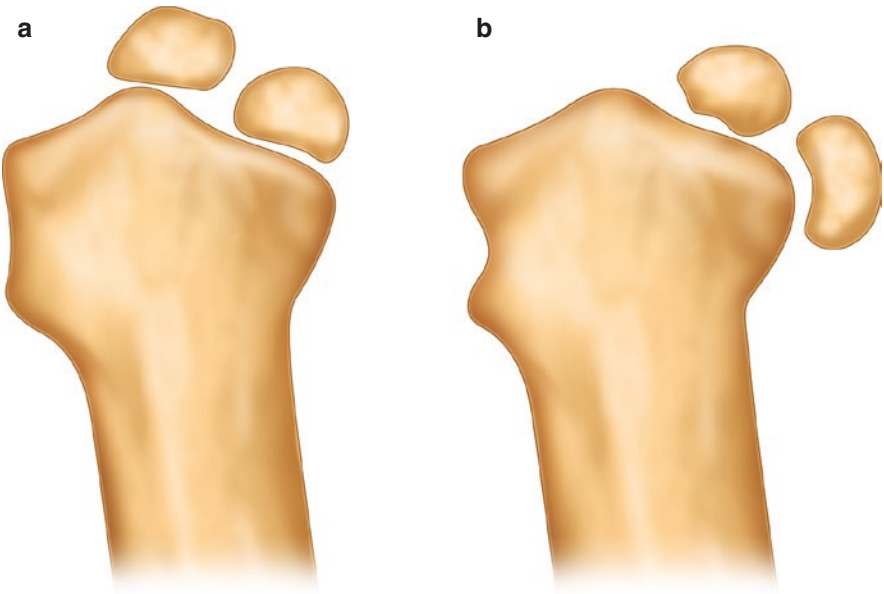


Fig. 30.22 Schematic representation of sesamoid subluxation (**a**) and sesamoid dislocation with erosion of the central crista (**b**). (Diagram credits: Ritika Sharma)

Treatment

The mainstay of treatment is to realign the sesamoids with articular plantar facets of first metatarsal. This can be done by treating the primary pathology, i.e., hallux valgus and its corresponding deformity [48].

Hammertoe

Introduction

Hammertoe is an abnormal flexion deformity of the proximal interphalangeal (PIP) joint of one or more lesser toes, with or without a hyperextension deformity of metatarsophalangeal (MTP) joint. The distal interphalangeal (DIP) joint usually remains supple, although it can sometimes develop a flexion or an extension deformity [49].

MTP and DIP joint deformities are secondary to long-standing proximal interphalangeal joint flexion deformity.

Flexion deformity of the PIP joint is usually not supple except in the very early stages; this is unlike in claw toes where the PIP joint deformity is usually supple [50].

Incidence

It is the most common deformity of the lesser toes seen in one-third of the general population. Females tend to have a higher incidence [51]. Hammertoe deformity usually involves one or two toes with the second toe most commonly involved.

Predisposing Factors

The etiology is multifactorial, and some commonly associated conditions are as follows:

1. Inflammatory arthritis like rheumatoid arthritis and psoriatic arthritis.
2. Wearing shoes with a narrow toe box.
3. Hallux valgus leading to secondary deformities of lesser toes.
4. Neuromuscular diseases like cerebral palsy, Friedreich ataxia, multiple sclerosis, Charcot-Marie-Tooth disease, etc.
5. Old age.
6. Trauma.
7. Peripheral neuropathy, i.e., diabetes and leprosy.

Clinical Features

Pain usually occurs due to the dorsal prominence of the flexed PIP joint rubbing against the shoes. This causes secondary pain over plantar aspect of head of metatarsal due to hyperextension of MTP joint (Fig. 30.23a, b).

Differentiation needs to be done with a similar condition called claw toe which is due to intrinsic muscle weakness. Here, the extrinsic muscles (extensor digitorum longus and flexor digitorum longus) overpower the intrinsic muscles (interossei and lumbricals) causing a resultant hyperextension at MTP joint and flexion deformity of the PIP joint and DIP joint. This results in dorsal subluxation of interossei, which now become extensors of the MTP joint resulting in further exacerbation of the deformity (Fig. 30.24).

Radiology

Gun barrel deformity may be seen on AP view of the foot X-rays.

Management

Conservative treatment is appropriate for young patients with flexible deformity and elderly patients with multiple comorbidities. The modalities consist of:

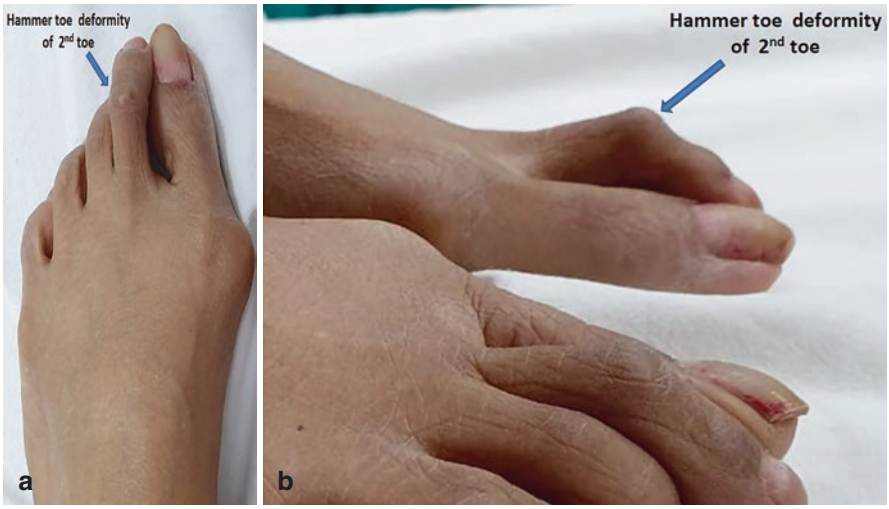


Fig. 30.23 (**a**) and (**b**) Clinical images showing hammertoe deformity involving the PIP joint of the second toe

- Stretching and manipulation of the toes—helps in keeping the feet flexible.
- Corrective taping may be helpful.
- Modifications of shoe wear—well-fitting shoes with a high, roomy, wide toe box with padding over bony prominences.
- Orthotics for transfer metatarsalgia—metatarsal bar, metatarsal pad, and off-loading insoles.

Surgical treatment (Fig. 30.25) helps in improving function and pain in symptomatic patients.

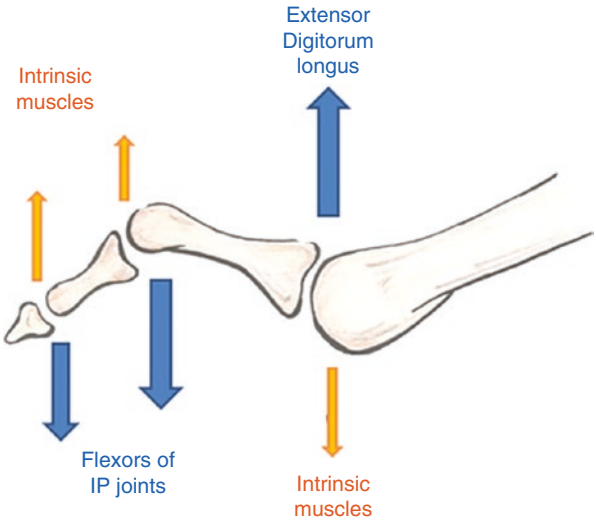


Fig. 30.24 Diagram showing forces acting over MTP, PIP, and DIP joints by intrinsic and extrinsic muscle forces. In a claw toe, strong extrinsics overpower weak intrinsics, causing hyperextension at MTP joint and flexion at PIPJ and DIPJ. As the deformity progresses, the interossei sublux dorsal to the MTP joint, therefore becoming extensors, further aggravating the deformity. Once this happens, the lumbricals are at mechanical disadvantage, and cannot counterbalance the forces. Credits—Ritika Sharma

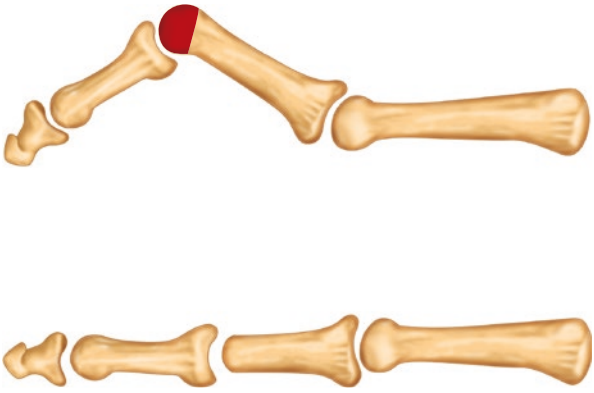


Fig. 30.25 Schematic representation of DuVries resection arthrodesis for hammertoe deformity. The head of the proximal phalanx of the deformed toe is resected, the cartilage of the middle phalanx is not denuded, and the joint is stabilized with a K-wire. This leads to a fibrous union across the joint, allowing a jog of movement which is usually pain-free. (Diagram credits: Ritika Sharma)

Principles of Deformity Correction

1. For flexible hammertoe deformity [52], a flexor to extensor tendon transfer works well. Kuwadas [53] described a popular technique for flexor to extensor tendon transfer.
2. For rigid hammertoes, DuVries arthroplasty/condylectomy is usually the procedure of choice. This involves resection of the head of the proximal phalanx which leads to fibrous union allowing 10–15° pain-free range of motion at PIP joint.
3. Another option especially for severe cases and failed DuVries arthroplasty is formal fusion of the PIP joint.

Ingrown Toenail

Synonyms: Onychocryptosis, "unguis incarnatus," "ungualabial hypertrophy," "embedded toenail".

Introduction

Ingrown toenail (Fig. 30.26) occurs when the sharp corner or edge of the toenail digs into the nail groove on the medial or lateral aspect causing local inflammation, pain, and sometimes local cellulitis. The skin flap on that edge usually hypertrophies, increasing chances of further injury and inflammation.

It is more common in teenagers and commonly seen in the great toe.

The common causes are excessively or improper trimmed nails, trauma, tight shoes, or patient on isotretinoin therapy [54].

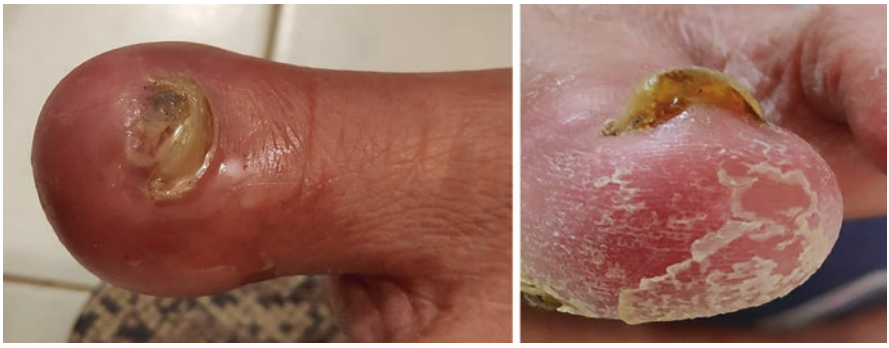


Fig. 30.26 Clinical images of ingrown big toenail with acute paronychia. (Courtesy: Dr. Ajoy SM, Bangalore, India)

Classification

Frost classified it into three types [55].

Type 1: Fish hook-shaped spur—due to improper trimming of the nail; develops in the lateral nail groove (Fig. 30.27).

Type 2: Incurved nail—distortion of nail plate at the margin.

Type 3: Soft tissue hypertrophy of the lateral border around nail with normal nail plate [56].

Management

1. Treatment in acute phase involves
   (a) Warm water fomentation to soften surrounding tissue.
   (b) Antibiotics can be helpful for infected cases and paronychia.
   (c) Regular dressing and packing with cotton wool bud, gently separating the hypertrophic skin from the nail edge, is usually the most successful intervention.
2. Once the acute phase settles, surgical treatment can be considered for recurrent cases.

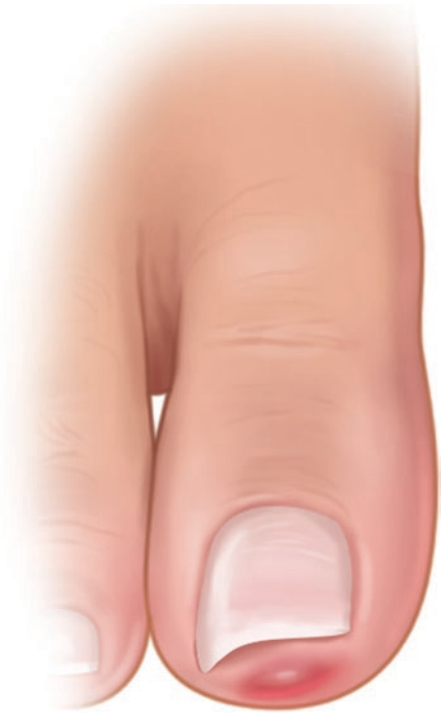


Fig. 30.27 Schematic representation showing ingrown nail with a fish-hook spur. (Diagram credits: Ritika Sharma)

The commonly performed surgical procedures are the Zadik procedure and nail plate avulsion.

1. Zadik/Fowler procedure (Fig. 30.28a–e) is a reliable method of treating the condition where skin margin and root are incised using a crescentic incision to expose lateral nail spur and germinal matrix along with the loose or infected tissue. This is removed and it can be combined with chemical germinal matrix ablation which has been shown to have a lower chance of recurrence [57].

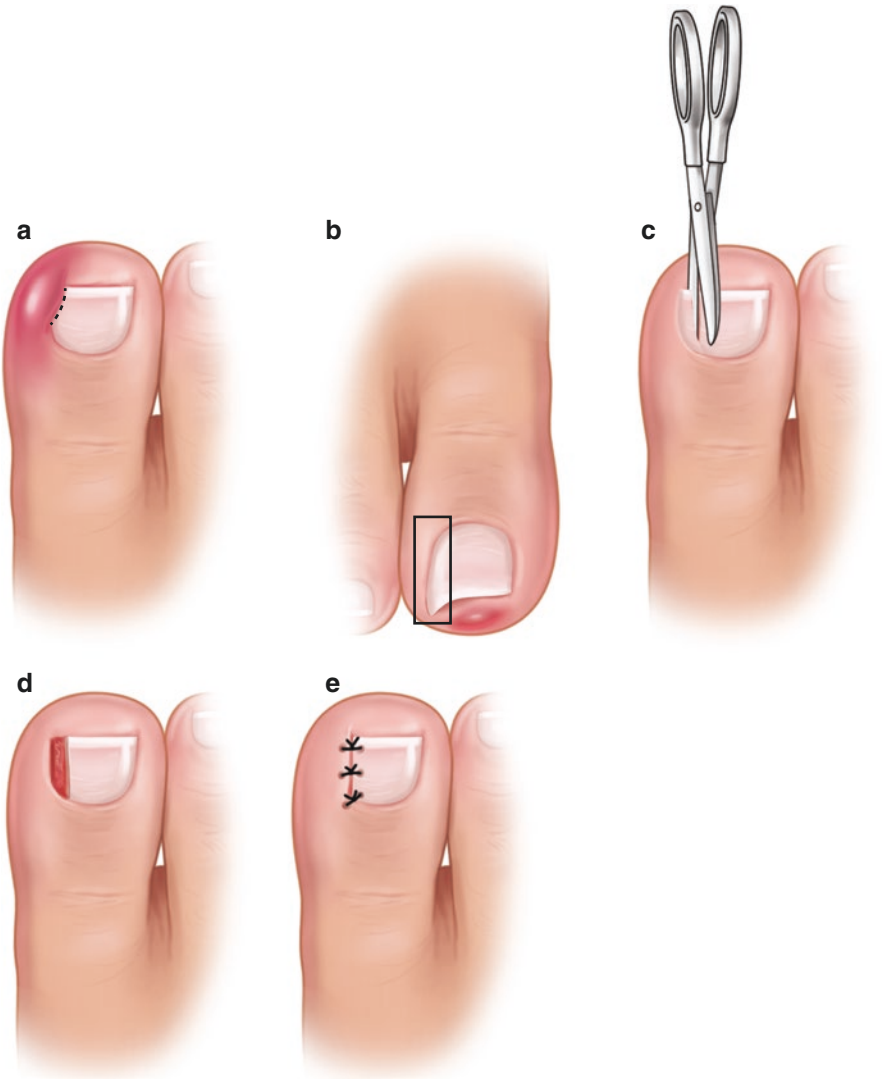


Fig. 30.28 (**a**–**e**) Line diagrams showing the steps involved in surgical excision of an ingrown toenail. The green rectangle in (**b**) shows the amount of tissue which needs to be resected. (Diagram credits: Ritika Sharma)

2. Complete avulsion of the nail plate can be done for significant nail deformity; however, it is usually avoided due to the possibility of clubbing of nail and deformation of toe.

Prevention: Prevention of an ingrown toenail from arising or recurring usually involves keeping a good foot hygiene, using shoes with wider toe box, and conservative nail trimming in a straight border.

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Part III

Additional Chapters

Advances in Navigation and Robot-Assisted Surgery

31

James Corbett and Wasim S. Khan

Introduction

Navigation is defined as the process of accurately ascertaining one's position and planning and following a route. Just as a GPS system gives a car driver visual feedback of their journey along a route, navigation in orthopedics gives a surgeon real-time feedback while performing a surgical step. Visual feedback on a screen orientates the surgeon in the surgical field to more accurately make a bony cut or position an implant.

Navigation has been in use in orthopedics for over 20 years. First reported uses were in navigation for pedicle screw placement during spinal surgery [1]. Current aims of navigation are to more accurately make bony cuts, more accurately position implants, and reduce damage to surrounding structures. The potential benefits are clear, with improved accuracy and reduced variability of implant placement, and the goal is to improve implant survivorship while at the same time minimizing soft tissue damage. Concerns remain about the potential for increasing operating time and cost without objective improvement in outcomes.

In this chapter, we will discuss different types of navigation systems available, discuss a technique for robot-assisted surgical navigation for hip arthroplasty, and review the current evidence available.

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Classification of Navigation Systems

There are numerous systems available to provide intraoperative assistance to the orthopedic surgeon: accelerometer-assisted surgery, computer-assisted orthopedic surgery (CAOS), and robot-assisted surgery. The main focus of this chapter is developments in computer-assisted orthopedic surgery, but accelerometer-based designs provide a cheaper alternative that does not require a separate console in the operating theater. They function by attachment of a device which measures dynamic movements to conventional jig systems. In accelerometer-assisted knee arthroplasty, the device is attached to the jig and, after a series of calibration movements, identifies the center of rotation of the hip and knee and subsequently is used to align the cuts relative to the mechanical axis.

Computer-assisted orthopedic surgery can be divided into active, semi-active, or passive systems [2]. The earliest and most complicated were active robotic systems. Semi-active systems do not perform the surgical tasks but limit the placement of surgical tools; they allow a surgeon to move freely within set limits but provide resistance if the operator's actions approach the boundaries of a defined "safe zone." Passive systems display information such as limb alignment on a monitor [3].

Composition of a Navigation System

There are three main components to a computer navigation system: the computer platform, a tracking system, and rigid markers. In optic-based systems, multiple cameras placed on a console in the operating theater visualize the markers, and their movements are tracked and processed by the computer system in 3D space. Markers must therefore be attached to the bones and the surgical instruments to allow them to be tracked.

Computer System

This receives input from the tracking system and interprets the data forming a 3D coordinate grid with *x, y,* and *z* components. The output from the computer system is on a monitor in the theater, displaying to the surgeon the shape and size of the instrument and its relation to the target object.

Tracking System

Tracking can be performed using optical cameras, electromagnetic coils, or ultrasonic probes.

Optical systems use two or three cameras to pick up infrared light from three to five marker arrays attached to the target object. This introduces line of sight issues—the camera must be able to "see" the markers or its accuracy decreases.

Electromagnetic systems do not use reference arrays or cameras—avoiding line of sight issues—but the field is distorted by metallic objects and trackers must be linked to the computer by wires which can prove difficult in practice [4].

Markers

These can be active or passive. Active markers emit light like a bulb. The majority, however, are passive and reflect infrared light. The tracking system must triangulate the position of each marker. A marker attached to a bone forms the "dynamic reference base" [3].

Referencing

Now we have an understanding of the components of a navigation system, the next step is how these components relate and produce real-time feedback in the operating theater.

We can consider the three components: the therapeutic object (the target of the treatment), the virtual object (3D-mapped virtual representation of the therapeutic object), and the navigator (which relates the objects) [5]. The navigator uses the 3D coordinate system to map the object and end effector, and the relation between the two can be computed. The end effector can be a different tool depending on the purpose of the system, for example, a cutting jig or the reamer of an acetabular component. In robotic devices, the robot is the navigator. For computer-assisted optic-based surgical navigation, it is the tracking device [5].

To establish a coordinate system, the effector must be calibrated; the size and shape of the effector have to be defined to the coordinate system. For image-based systems, the next step is registration of the therapeutic object with respect to the virtual object to display the end effector's location in respect to the virtual object. This is correlating the 3D model created from preoperative imaging to the patient's anatomy on the operating table by taking points in specific locations on the patient. The last step is referencing, which aims to account for movement of the navigator or the therapeutic object during the surgical procedure by either attaching a "dynamic reference base," holding optical markers to established landmarks on the therapeutic object, or immobilizing the object with respect to the navigator.

Types of Referencing in Computer Navigation

You can further categorize navigation systems by the type of referencing they use: CT based, fluoroscopy based, or imageless [3]. In CT-based systems, a 3D model is created from preoperative CT scan with the advantage of being specific to the patient's own anatomy but increasing cost and with increased radiation exposure.

Fluoroscopy-based systems use an image intensifier, and while taking the images, a marker is used and a computer relates the position of the marker to the image captured.

Imageless systems require no preoperative scans and do not use fluoroscopy. They use pooled data from CT scans of a large number of patients to create a database. During the operation, multiple predetermined landmarks are identified and used to determine center of rotation of joints. A "best fit" model is then created by the computer system using the database. The advantage of imageless is that it does not require a CT scan, reducing delay and radiation exposure. There is, however, potential for inaccuracy based on the surgeon's ability to identify accurately the landmarks with the bony marker and the reliance on the database of "normal patients" which could prove inaccurate for patients with severe deformity or unusual anatomy.

Once referencing has been performed, we have defined a coordinate system, the target object, and effector's position within it and can see how they relate on the screen. The next section will consider the addition of a robot into this system.

Robot-Assisted Surgery

One of the first documented uses of robotics in surgery was in 1985 using the PUMA 560 to place a needle for CT-guided stereotactic brain surgery [6]. The first robot used in orthopedics was ROBODOC (Integrated Surgical Systems, California, USA) in 1992 to prepare the femur in total hip arthroplasty [7]. Since then, numerous other systems have been designed and used in hip and knee arthroplasty, spinal surgery, foot and ankle surgery, arthroscopy, and trauma, and the two most common in current use are the MAKO (Stryker®) system and the NAVIO (Smith and Nephew®). Most evidence is available for hip and knee procedures.

The setup for robot-assisted orthopedic surgery is similar to computer-assisted surgery and will be described in more detail later. The main difference is the presence of the robot in the operating theater in addition to the camera and display console. Further, unlike conventional navigation where a tracking device is the navigator and guides an effector with its reflective markers to the target, the robot acts as the navigator. Its arm is connected to the tool and functions in a semi-active way; it can be maneuvered in the "safe zone" in the surgical field but will only allow you to perform the task according to the preoperative plan. Actions outside of the plan are restricted.

Robot-Assisted Hip Surgery

The method described here is the MAKO (Stryker®) CT-based robotic navigation system [8]. The patient requires preoperative CT imaging following specific protocols defined by the manufacturer. Reconstruction of this CT imaging then forms the basis for the "virtual object."

Fig. 31.1 MAKO hip: example of a pre-op plan for THA

A surgical plan is then formulated based on this imaging (Fig. 31.1). In accordance with personal preferences, CT landmarks are also established.

Operative Steps

Distal landmark placement: A distal landmark is identified, for example, over the patella.

Pelvic array placement: An array of markers is placed into the pelvis either through the wound in the supra-acetabular bone or in the iliac crest.

Femoral cortical screw placement: A uni-cortical screw is placed in the proximal femur and a femoral array attached to this. The array should be visible by the camera.

Femoral registration: Femoral landmarks are identified using probe. These are the landmarks identified preoperatively on the CT scan. They orient the femur with respect to the virtual image. Registration is verified by identifying further landmarks and if inaccurate, repeated.

The femur is now registered with the system, and the planned femoral cut can be marked out with cautery and the neck resected. This allows access to the acetabulum. The femur can be broached using the robot as a guide at this stage or later in the procedure.

Pelvic registration can then be performed mapping out the orientation of the native acetabulum and registering it with the virtual acetabulum formed from the pre-op CT images.

The robotic arm then controls movement of the acetabular reamer (Fig. 31.2) and executes reaming based on the final position of the cup determined during the

Fig. 31.2 Assembled MAKO acetabular reamer

preoperative plan, and the display will turn red if a surgeon reams 1 mm past the operative plan, and if greater than 2.3 mm, the power drill will turn off. The cup can then be seated using the robotic arm as the guide.

Outcomes in Navigated Hip Surgery

Computer-Assisted Total Hip Arthroplasty (THA)

A recent review and meta-analysis comparing navigation and conventional hip arthroplasty found navigated hips had improved accuracy for anteversion and inclination than freehand placement, but there is a lack of evidence to support improved functional outcomes or reduction in complications or revision [9]. Intraoperative navigation has been demonstrated to accurately assess acetabular component position intraoperatively in the direct anterior approach [10] where fluoroscopy has previously been used.

Robot-Assisted Hip Surgery

Early work in robot-assisted hip surgery focused on correct orientation of uncemented femoral prostheses but no sustained improvement in clinical outcomes and a higher dislocation rate limited its use [11]. Others found no difference between conventional THA and robot-assisted THA, but costs and technical complications relating to the robot were greater [12]. More recent work by Domb et al. in 100 patients in a matched study assessed acetabular cup positioning and accuracy of

placement in the safe zone as described by Lewinnek et al. [13]. One hundred percent of robot-assisted surgeries were within Lewinnek's safe zone compared with 80% in the conventional THA group. The impact on dislocation, wear, and revision remains unclear [14].

Advances in Navigated Knee Surgery

Limb alignment and soft tissue balancing are important for the success of knee surgery. Component malalignment is a major cause of aseptic loosening of knee arthroplasty, and navigation aims to improve accuracy of implant placement, reduce malalignment, and reduce rates of loosening. In the following section, we will consider current evidence of different techniques for navigation in knee surgery.

Accelerometer-Assisted Total Knee Arthroplasty (TKA)

Numerous studies have demonstrated improved mechanical alignment and component positioning with accelerometer-based systems compared to conventional jig methods [15–17]. As yet, this does not seem to translate to improvement in clinical outcomes.

Computer-Assisted TKA

A randomized trial from Hong Kong compared conventional instrumentation with computer-assisted TKA and demonstrated improvement in mechanical alignment and component positioning in terms of femoral component rotation but did not demonstrate any improvement in clinical outcomes at average follow-up of 5 years [18]. Similar findings have been reported in previous reviews [19]. Data from the Australian registry found non-navigated knees had a stronger association with revision compared to computer-navigated knees, with the tibial component revised more frequently than the femoral component [20].

Robot-Assisted Total Knee Arthroplasty

A retrospective study of 84 knees recently published in arthroplasty today demonstrated no statistically significant differences between the group of robot-assisted and conventional arthroplasty patients in terms of clinical or radiographic outcomes. There was a trend toward fewer radiographic outliers in the robot-assisted group, but this did not achieve statistical significance [21]. Other studies support this finding with improved accuracy at the expense of increased operative time and cost [22].

Robot-Assisted Unicompartmental Knee Arthroplasty (UKA)

A recent review in the BJJ published in 2019 [23] found that implant positioning with robotic UKA was more accurate, but there was insufficient evidence at this stage to determine whether this had any effect on mid- to long-term survivorship. This supports a similar finding by Fu et al. previously [24].

Improved gait kinematics in robot-assisted UKA compared with conventional Oxford UKA was reported by Motesharei et al.; however, they did not evaluate patient-reported clinical outcomes [25].

Navigation and Osteotomies

Osteotomies around the knee are well-established treatments that rely on accuracy and reliability of correction angles in coronal and sagittal planes [26]. To increase precision of these corrections, navigation can be a useful tool. A recent meta-analysis showed that the risk of outliers in navigated high tibial osteotomies (HTOs) was lower in navigated surgery compared with all conventional HTOs but was not significant when compared with the gap-measurement method subgroup of conventional HTOs. Tibial slope maintenance was better in navigated HTOs but no difference in patient-reported clinical outcome measures. Navigated HTO also increased operative time by approximately 10 min [27].

Navigation and Shoulder Arthroplasty

Accurate glenoid component placement is vital to prevent failure in total shoulder arthroplasty. A prospective case-control study of 60 patients demonstrated increased rates of augmentation of navigated glenoid prosthesis with final alignment more likely to be "neutral" with reduced variability. No data were presented on survivorship [28].

Navigation and Fracture Fixation

While navigation and robotics in arthroplasty are rapidly evolving areas, navigation in trauma surgery is in its infancy. Navigation offers the promise of real-time guidance of fracture reduction which could be particularly beneficial in fixation of complex intra-articular fractures. Further work is being done on potential use of robotics in application of external fixators [29]. One study reports promising results on the use of robotic surgery for wire placement during scaphoid fracture surgery [30].

Numerous systems exist to navigate the surgeon to accurately perform distal locking of intramedullary nails without the use of fluoroscopy. Higher accuracy with shorter operating time and no radiation has been reported with magnet-based

navigation systems [31]. Increased costs associated with this instrumentation should be taken into account.

Other Uses

Young adult hip surgery: The potentials for planning and more accurate treatment of femora-acetabular impingement using computer-assisted hip arthroscopy are highlighted in a review by Nakano et al. [32]. Studies on clinical outcomes are lacking, but navigation can certainly be useful in what are technically depending surgeries, particularly in assessing sphericity of the femoral head arthroscopically.

Navigated tumor resection surgery: Clear resection margins are fundamental to successful tumor resection surgery, and surgical navigation clearly offers potential benefits in helping to achieve this particularly in patients where freehand resection is challenging [33]. A recent paper demonstrates effective resection and reports reduced setup time using skin markers for registration [34].

Simulation training for orthopedic surgeons: Wire placement in hip fracture surgery simulation models has typically struggled to gain widespread acceptance due to the requirement for fluoroscopy. Simulation-based training with a navigation model has eliminated the need for fluoroscopy while providing real-time feedback on wire placement. This tool has been expanded to placement of ilio-sacral screws, allowing training to occur in a simulated environment [35].

Conclusion and Future

Computer-assisted orthopedic surgery demonstrates improvements in surgical accuracy, but this does not always translate to improved functional outcomes for patients. One explanation for this is that we have still not identified what the "optimal" positions are for our implants. CAOS and robot-assisted surgery show great promise, and there are many examples where they have been demonstrated to improve surgical accuracy. Whether improvements in functional outcomes are borne out in longer-term studies remains to be seen.

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Stem Cell Therapy in Orthopedics

32

Benjamin M. Davies and Wasim S. Khan

Introduction

Trauma and orthopedic surgery has been, and continues to be, an area in which there is significant interest in the development of cellular therapies to treat both traumatic and atraumatic conditions.

Cellular therapies encompass a wide variety of biological treatments from the simple transfer of bone marrow from one anatomical location to another, up to, and including the use of laboratories to select, grow, and prepare cells prior to use.

Orthopedics has, in fact, been making use of cellular therapies for a long time. Microfracture of cartilage, as described by Steadman in 1997 [1], is a form of cell therapy. Bone grafting, which can also be viewed as a type of cell therapy, has been taking place for almost 200 years [2].

The promise of cell therapies is that we will be able to move from a treatment model that frequently relies on simply replacing damage, such as with total joint arthroplasty, to a model where we repair and regenerate damaged tissue to restore the body's normal function. Stem cells represent the latest area of research and clinical development in this field.

Stem Cells

Stem cells are defined by their properties [3], namely:

- They can self-renew.
- They can differentiate into all, or a subset of, cellular phenotypes.

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A number of different types of stem cells exist:

- Embryonic stem cells (ESCs)
- Umbilical cord blood stem cells
- Induced pluripotent stem cells (iPSCs)—cells with stem cell properties created in a laboratory by a process of dedifferentiation of fibroblasts [4].
- Adult stem cells:
  - Mesenchymal stem cells (MSCs)[1]
  - Hematopoietic stem cells (HSCs)
  - Neural stem cells

ESCs are not encountered in clinical practice due to their scarcity and, more significantly, the ethical issues that would be involved in their use. iPSCs are similarly rarely used due to the difficulties, and expense involved, in their creation.

Stem Cells Used in Orthopedics

Cellular therapies in orthopedics revolve, almost entirely, around the use of MSCs. The chief variable is the source of these MSCs. Depending on the source of the extracted MSCs, they may have slightly different names (Fig. 32.1).

Properties of MSCs

MSCs have a number of fundamental properties that make them suitable for use in orthopedics:

- They can differentiate into chondrocytes, osteoblasts, and adipocytes.
- They are immune-modulatory.
- They can divide multiple times.

Tissue source	MSC name	MSC containing fraction name
Bone marrow	Bone marrow MSC (BM-MSC)	Bone marrow concentrate (BMC)
Synovium	Synovial stem cell (SSC)	n/a
Adipose tissue	Adipose-derived stem cell (ADSC)	Stromal vascular fraction (SVF)

Fig. 32.1 Table showing the different types of MSCs

[1] Sometimes referred to as mesenchymal stromal cells.

Identification of MSCs in both research and clinical scenarios is not straightforward. Traditionally, they have been identified by possession of the following characteristics [5]:

- Adherence to plastic in cell culture
- Tri-lineage differentiation (cartilage, bone, adipose tissue)
- Characteristic cell surface antigen expression[2]

The complexity of isolating and identifying MSCs, combined with their extreme scarcity, is one of the major issues in both studying them and developing clinical applications, especially those based on autologous sources.

BM-MSC

These are the most commonly used type of MSC. They underpin the biological component of bone grafting as well as providing the regenerative component of microfracture treatment used to repair cartilage defects. They are, however, relatively rare, forming <0.01% of the cells found in bone marrow.

ADSC

ADSCs are becoming a more commonly used cell type in orthopedics. This is due to the relative ease of obtaining large volumes of fatty tissue (by way of lipoaspiration) and the higher proportion of ADSCs in adipose tissue (3–4%). These cells do, however, have a greater propensity to differentiate into adipocytes when compared with MSCs sourced from other tissues such as bone marrow or synovium.

SSC

SSCs are difficult to obtain due to the scarcity, difficultly in harvesting, and associated morbidity with obtaining synovial tissue. They do, however, demonstrate the greatest ability to differentiate into chondrocytes of all the different types of MSC discussed here.

Autologous Versus Allogeneic

MSC-based treatments can be undertaken using autologous (patient's own) or allogeneic (donor) cells. At present, the vast majority of treatments available use

[2] CD73/CD90/CD105 positive and CD11b/CD14/CD19/CD34/CD45/CD79α/HLA-DR negative.

autologous cells as, although MSCs do not provoke a full foreign body reaction, they are not completely immune privileged [6].

Single-Sitting Versus Laboratory-Based Treatments

MSC treatments can be, broadly, broken into two groups.

- Single-Sitting Procedures
- These treatments make use of concentrated MSCs obtained from tissue such as bone marrow or adipose tissue. The concentrated fraction is usually formed by a centrifugation process. These treatments have the benefit of being relatively simple to implement, cheap, and with low regulatory burdens. However, they rely on purely concentrating the native MSC population which can be extremely variable between different individuals and cannot, usually, be prospectively assessed at the time of treatment.
- Laboratory-Based Procedures
- These treatments involve the harvesting of MSCs from tissue and then expanding them in a laboratory setting to vastly increase the number of cells available for reimplantation (in a similar manner to how chondrocytes are harvested and expanded for use in autologous chondrocyte implantation (ACI)). The manipulation of cells outside of the body means that there is a significant regulatory burden associated with these treatments in addition to the costs of laboratory processing of cells. Products produced in this way are usually classed as Advanced Therapy Medicinal Products (ATMPs) and may be subject to different regulatory procedures compared with other licensed medications and treatments.

Mechanism of Action of Stem Cells

There are two broad mechanisms of action that have been put forward as underpinning the process by which MSCs produce their reparative effects.

Initially, it was felt that the primary mechanism of action was differentiation of MSCs into the required tissue in order to affect a tissue repair—so MSCs placed in a cartilage defect would differentiate into chondrocytes and thereby generate new cartilage to fill the defect. This theory has been partially undermined by evidence that the majority of regenerative tissue that forms in a defect following MSC implantation is not derived from the implanted cells [7].

Over time, greater understanding of the functions of MSCs has led to the development of new proposed mechanisms by which they repair damaged tissue [8]. This paradigm shift is based on MSC abilities of:

- Immunomodulation
- Paracrine and cell-to-cell interactions
- Homing to sites of injury

This change in the understanding of how MSCs function has allowed for an expansion in cellular therapy techniques as there is no longer a logical requirement to place a large number of cells into a specific area of damage in the hope that they will directly differentiate into specific cell types and create new, undamaged tissue.

MSC Treatments in Orthopedic Practice

The MSC-based regenerative medicine treatments that are available vary by the type of tissue that one wishes to treat. It should be remembered that many of these treatments are not yet in mainstream use and so may not be available in all locations or health-care provider systems.

Bone

As previously mentioned, any autologous bone graft could be considered as an MSC-based procedure, although the structural and bone-inducing effects of bone matrix must not be discounted.

BMC has also been used in some settings in order to augment and stimulate the healing of nonunions, and this may be viewed as a form of augmented bone grafting.

Cartilage

The use of MSCs in the treatment of cartilage damage can be broken down into the treatment of isolated osteochondral defects (OCDs) and the treatment of more global, joint-wide arthritis.

Osteochondral Defects

When thinking about the use of MSCs in the treatments of OCDs, it is worth thinking of the treatments available as a hierarchy.

1. Microfracture

   This procedure involves debriding an area of cartilage damage and then using an awl to puncture the subchondral bone plate and allow the MSCs resident in the intramedullary canal to leak into the defect and form a clot. This technique has been used successfully for many years, and there is evidence that it provides good improvement in symptoms for up to 5 years when used in defects ≤ 2 cm^2 [9, 10].
2. BMC

   A number of manufacturers provide methods of concentrating bone marrow aspirate so that it can be injected into osteochondral defects and other areas of tissue damage. There is some variation in how these techniques work. Some use a patch to cover the area, in a similar way to ACI. Others use the addition of

fibrin to stabilize the forming clot and so hold it in position. The evidence base for these procedures is relatively small, but they benefit from a relatively low morbidity and cost when compared to other tissue regeneration procedures.

3. Scaffold

   The use of scaffolds in chondrocyte-based procedures—named Matrix-Assisted Chondrocyte Implantation (MACI)—overcomes some of the technical issues regarding the procedure, theoretically improves chondrocyte formation, and has similar results to ACI [11]. This has led to the development of similar procedures using MSCs, where they are placed on a three-dimensional scaffold prior to implantation.

Arthritis

Stem cell injections to treat joint-wide osteoarthritis have become more and more prevalent since the demonstration of their effectiveness in an animal model [12]. The exact nature of these treatments varies widely with the use of different cell sources (fat, bone marrow, umbilical cord), preparation methods (concentrated vs. expanded), delivery agents (platelet-rich plasma, hyaluronic acid), and dose [13].

The wide variety of ways in which stem cells have been used and the lack of robust study data means that this form of treatment is not widely available due to a deficiency in the evidence base. Single-sitting procedures form the majority of treatments available (due to their lower regulatory burden). Until the results of early studies [14] are built upon adequately, the use of these treatments will, most likely, fail to enter widespread clinical use.

Tendon

The use of stem cells in tendon pathology is not as advanced as in cartilage disorders. The principles of what treatments are available are the same as for any other musculoskeletal tissue.

The outcome of studies looking at the use of stem cells in tendons may well provide more evidence for their use [15, 16], but until that time, they are unlikely to become widely available in the majority of health-care settings.

Regulatory Issues

As alluded to above, the two primary groups of treatments—single-sitting versus laboratory-based—lead to two very different set of regulatory issues.

Cell-based treatments that simply harvest the cells and reimplant them with very little processing apart from concentration of the sample are usually referred to as "*minimally manipulated*" cells [17]. These treatments are typically subject to relatively light levels of regulation and little need for extensive clinical trials.

Treatments that involve cells (or structural tissues) that have been manipulated to any significant degree, including expansion in a laboratory, are regulated in a

manner much more closely akin to that used for drug regulation. There is a significantly greater requirement to demonstrate regulatory compliance at all stages of product development and a requirement for extensive clinical trial data. The use of *adaptive pathways* where products are assessed as they go through the development pathway and may be released for public use at an earlier stage is helping to speed up this process [18].

Future Developments

As of mid-2019, there is only one ATMP approved by the US Food and Drug Administration (FDA) for orthopedic use—MACI® by Vericel which makes use of autologous, cultured chondrocytes rather than stem cells. The future may well see the approval of MSC-based treatments that are able to achieve similar results without the need to harvest chondrocytes.

One allogeneic treatment using umbilical cord blood-derived MSCs (Cartistem® by Medipost) has received approval for use in cartilage defect repair in South Korea but has not been approved for use more widely as of yet.

Future research and clinical treatment development will enable the development of appropriate definitions and criteria for MSCs that reflect their actions more closely and enable easier development and comparison of treatments.

Laboratory research is also looking into the function and potential therapeutic uses of extracellular vesicles (EVs) [19]. These are small particles released from cells, including MSCs, that contain proteins and other biologically active components to facilitate cell-to-cell communication. These may represent the final mechanism of action of MSCs, and further study may enable us to replicate their actions directly without the need to implant living cells into damaged tissue.

Conclusions

Stem cells are a promising area of treatment development for use in the field of orthopedics. At present, the lack of robust evidence and the high regulatory and financial burden (especially of laboratory-based procedures) have prevented their widespread penetration into clinical practice.

The future of orthopedic surgery will, surely, involve the development of regenerative strategies that will enable surgeons to address musculoskeletal disorders at an earlier stage, prior to patients becoming significantly disabled by their disease.

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Shock Wave Therapy in Orthopedics

33

Kandiah Raveendran

History

ESWT was first used in patients for the disintegration of renal and ureteric calculi in 1980 followed shortly by its use for the disintegration of gallbladder calculi in 1985.

In 1988, the first successful treatment for nonunion of the bone was performed in Bochum in Germany.

In the nineties, shockwave treatment was used for the treatment of plantar fasciitis, lateral epicondylitis (tennis elbow), and calcific rotator cuff tendinitis.

In the last two decades, the use of ESWT has grown rapidly, and it has now expanded beyond the field of orthopedics into the treatment of skin ulcers, burns, cellulite, cardiac ischemia, erectile dysfunction, neurological disorders, and osteoarthritis.

In this chapter, I will only discuss the use of ESWT in orthopedics.

Physics of Shockwaves

Shockwaves are acoustic waves like ultrasound waves. Focused shockwaves (F-EWST) (Fig. 33.1) differ from ultrasound waves by being uni-phasic with a high-pressure amplitude (up to 150 MPa).

There are three different shockwave generators used today.

1. Electrohydraulic Source

   A high-voltage electric spark is generated between the two tips of an electrode in a water-filled semi-ellipsoid reflector. This produces a focused shockwave front which occurs due to the reflection of the spherical wave at the reflector.

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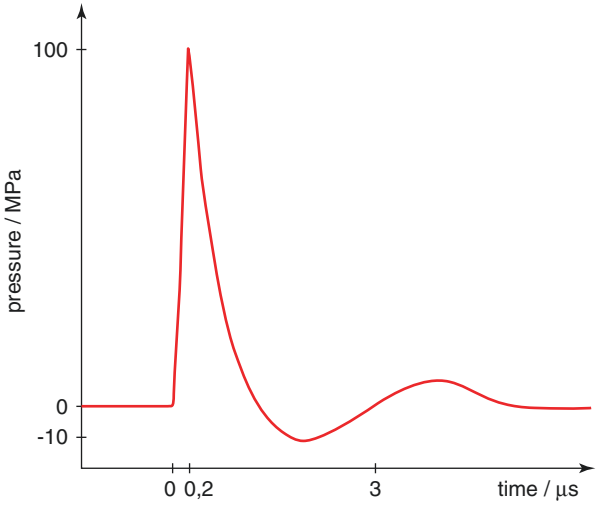

Fig. 33.1 Pressure profile of a focused shockwave

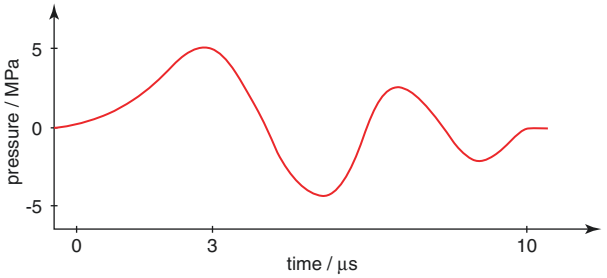

Fig. 33.2 Pressure profile of a radial pressure wave

2. Electromagnetic Source

   There are two types of generators. The first is a flat coil and a metal membrane opposite it. The coil produces a magnetic field resulting in a sudden deflection of the membrane creating a flat pressure wave in a fluid focused by an acoustic lens.

   The second type uses a cylindrical coil which creates a cylinder-shaped wave which is focused by reflection of the wave at a hyperbole-shaped metal reflector.

3. Piezoelectric Source

   The third type of generator uses high-voltage current applied to a few hundred piezoelectric crystals mounted on a spherical surface. This produces a pressure wave in the water, and in contrast to the other two systems, this is self-focusing.

In contrast to the focused ESWT (F-ESWT), there is a second type of wave which is the radial pressure wave (RPW) (Fig. 33.2). This is usually referred to in the older literature as radial shockwaves.

There are two types of generators. The first one uses compressed air to accelerate a projectile within a guiding tube producing a wave which propagates in a radial fashion.

The second generator uses electromagnetic acceleration.

These devices do not produce shockwaves, but they may also induce acoustic cavitation in the tissues.

The shockwave or radial wave transducer must be coupled with liberal application of gel over the patient's skin; otherwise, the therapeutic effect may be reduced.

F-ESWT and RPW may have different effects due to the different waveforms. Focused shockwaves can penetrate deeper suitable to treat the bone, while radial pressure waves are suitable for treating large superficial areas like the skin.

Mechanism of Action

The mechanism of shockwave treatment is not fully understood in spite of extensive basic research. In lithotripsy, shockwaves disintegrate urinary calculi, but in orthopedics, shockwaves cause interstitial tissue and extracellular responses leading to tissue regeneration. It stimulates cell functions and maintains physiological tissue homeostasis. This has been termed mechanotransduction, producing biological responses and healing by mechanical stimulation of bone or soft tissues [1, 2]. Shockwaves do not damage tissues.

Shockwave Treatment for Tendinopathy

Shockwaves enhance neovascularization at the tendon-bone junction of the Achilles tendon in dogs. Shockwave treatment releases angiogenic growth and proliferating factors improving the blood supply. This mechanism also may relieve pain and initiate repair of the chronically inflamed tissues by tissue regeneration [3].

Shockwave Treatment for Bone Healing

In 1997, Haupt et al. in an experimental model in rats confirmed a positive effect of shockwaves on fracture healing. High-energy shockwaves produce a significantly higher BMD (bone mineral density). The effects of shockwaves on bone appear to be time and dose dependent. Shockwaves promote bone stromal cell growth, differentiation of osteo-progenitors, recruitment of mesenchymal stem cells, and expressions of TGF-β1 and VEGF [4, 5].

New functional proteins induced by F-ESWT have a chondroprotective, neovascularization, anti-inflammatory, anti-apoptosis, and tissue and nerve regeneration effect giving pain relief and improved function.

Clinical Indications

The International Society for Medical Shockwave Treatment (ISMST) has a list of approved clinical indications and contraindications which are regularly updated. This is on the ISMST Web page (www.shockwavetherapy.org). The ISMST recommends that only a qualified physician who has passed a certification course be permitted to use focused ESWT.

The indications are divided into four sections:

1. Approved standard indications
2. Common empirically tested clinical uses
3. Exceptional indications
4. Experimental indications

The treatment of each patient should be individualized including a comprehensive posttreatment program including to rest the affected limb and a specific physical therapy regime. The patient should be told that improvement may take 2–4 weeks, and repeated treatments may be necessary with excellent and good results in 60–90% of the patients.

The contraindications are as listed in Fig. 33.3 below.

Approved Standard Indications*

1.1. **Chronic Tendinopathies**
- 1.1.1. Calcifying tendinopathy of the shoulder
- 1.1.2. Lateral epicondylopathy of the elbow (tennis elbow)
- 1.1.3. Greater trochanter pain syndrome
- 1.1.4. Patellar tendinopathy
- 1.1.5. Achilles tendinopathy
- 1.1.6. Plantar fasciitis, with or without heel spur

1.2. **Bone Pathologies**
- 1.2.1. Delayed bone healing
- 1.2.2. Bone nonunion (pseudarthroses)

Fig. 33.3 Table showing ESWT contraindications

ESWT contraindications
High-energy focused shockwaves
1. Lung tissue in the treatment area 2. Malignant tumor in the area 3. Epiphyseal plate in the area 4. Brain or spine in the area 5. Severe coagulopathy 6. Fetus in the treatment area
Low-energy focused and radial pressure waves
1. Malignant tumor in the treatment area 2. Fetus in the treatment area

1.2.3. Stress fracture
1.2.4. Avascular bone necrosis without articular derangement
1.2.5. Osteochondritis dissecans (OCD) without articular derangement

1.3. **Skin Pathologies**
1.3.1. Delayed or nonhealing wounds
1.3.2. Skin ulcers
1.3.3. Non-circumferential burn wounds
∗Following ISMST

These standard indications have been researched extensively over the last two decades, and the level of evidence is from RCTs or prospective trials.

Shoulder Tendinopathies

Calcifying Tendinopathy of the Shoulder (CTS)

Current knowledge of evidence-based F-ESWT for shoulder pathology emphasizes the excellent results for calcific shoulder tendinopathy. This is one of the two main indications for the use of ESWT in orthopedics, the other being plantar fasciitis. The success rate ranges from 78% to 91%, and complete elimination of calcification occurred in 57.6%, partial elimination in 15.1%, and unchanged in 27.3% of the patients in one study.

ESWT should always be used before the option of surgery [6].

Noncalcifying Tendinopathy of the Shoulder

This treatment is still controversial, and there is a lack of compelling evidence. The same applies to frozen shoulder, bicipital tendinitis, and postsurgical shoulder stiffness.

Lateral Epicondylopathy of the Elbow (Tennis Elbow)

Thiele et al. in their paper "Lateral epicondylitis: this is still a main indication for extracorporeal shockwave therapy" document the historical use of shockwaves for tennis elbow since 1996 with mixed results in earlier studies. Later studies showed more positive results, and the authors concluded that repeated applications of ESWT should be performed before resorting to surgery [7].

There are many ways to treat lateral epicondylopathy, and present evidence does not support one technique over the other. However, ESWT has been approved by the US Food and Drug Administration (FDA) for the treatment of tennis elbow since 2002. The use of radial or focused ESWT technologies is recommended when conventional treatments fail.

Greater Trochanteric Pain Syndrome

There is no evidence-based protocol for the management of greater trochanteric pain syndrome (GTPS). Conservative treatment for GTPS can achieve 90% success using ice packs, nonsteroidal drugs, physical therapy, and steroid injections.

In order to determine the best treatment protocol for GTPS, the exact pathology needs to be defined.

Rompe et al. have shown that radial pressure wave (RPW) was more effective at 4 months compared with steroid injection and home training, and this matched home training at 15 months [8].

Patellar Tendinopathy (Jumper's Knee)

This is a frequent injury associated with jumping sports like volleyball, basketball, and soccer. Acute cases can be treated by conservative measures like ice, rest, and eccentric exercises followed by a physical therapy schedule with 80% good results.

It is the 20% who develop a dysplastic insertional tendon overuse syndrome will be recalcitrant to the above treatment. Various forms of treatment have been proposed including surgery.

ESWT, both F-ESWT, and RPW have produced good results for this chronic patellar tendinopathy. ESWT must be seen as part of a treatment protocol including eccentric exercises and standardized physical therapy. At this stage, it is not possible to recommend F-ESWT over RPW or vice versa [9].

Achilles Tendinopathy

Achilles tendinopathy is a fairly common disorder affecting athletes as well as the sedentary population. It is usually divided into insertional and non-insertional or midportion tendinopathy.

The mainstay of treatment is conservative including nonsteroidal anti-inflammatory drugs, heel lifts, eccentric exercises, and physical therapy. Newer modalities include platelet-rich plasma (PRP) injections, laser therapy, and radiofrequency.

Both F-ESWT and RPW have been used, and there are several studies including Level 1 studies showing significant improvement in comparison to placebo. Gerdesmeyer et al. concluded that shockwave treatment is the most effective modality of treatment for chronic Achilles tendinopathy.

This is usually combined with eccentric training and should be offered before surgery [10].

Plantar Fasciitis

Plantar fasciitis is the most common cause of heel pain. Conservative treatment usually results in a high recurrence rate. Early studies found no evidence to support the use of ESWT, but since then, several studies have shown significant improvement with a low recurrence rate.

Wang et al. showed excellent or good results in 82.7% of the patients compared to 55% in the control group with low recurrence at a follow-up between 60 and 72 months.

A prospective multicenter, double-blind, randomized, and placebo-controlled FDA study was done on 250 subjects at five centers in the United States in 2015. The study using F-ESWT confirmed significant superiority compared to the placebo, with 35% pain reduction between groups [11].

Rompe et al. used RPW on 73 patients (group 1) and on another 79 patients (group 2) who also underwent an 8-week plantar fascia-specific stretching program. At 2 months and 4 months, group 2 showed a significant improvement in the Foot Function Index (PS-FFI). At 24 months, both groups were comparable in their improvement [12].

Since 2010, the American College of Foot and Ankle Surgeons has recommended ESWT as the treatment of choice for plantar fasciitis when conservative treatment has failed.

Delayed Bone Healing and Nonunion

Valchanou and Michailov were probably the first to use ESWT for fracture nonunion way back in 1991. Wang et al. showed that shockwaves generate expression of pro-angiogenic and pro-osteogenic growth factors stimulating bone healing.

In 2001, Schaden et al. reported a success rate of 85% in the treatment of 115 delayed and nonunions. Wang et al., also in 2001, reported a success rate of 82.4% of bony union at 6 months follow-up [13]. The results of high-energy ESWT in nonunion of long bones appear to be comparable to surgery with no postsurgical pain and complications.

Schaden et al. believe that ESWT is the first choice of treatment for nonunion after a long personal experience and an extensive review of the literature. A nonunion gap of more than 5 mm in long bones is a negative factor for a successful outcome. ESWT should be used following nonunion after internal fixation or with the addition of a plaster cast, orthosis, or an external fixator if there is no internal fixation [14].

Stress Fractures

Stress fractures are relatively uncommon and difficult to diagnose. A high index of suspicion is necessary to make an early diagnosis. They are classified as low, medium, and high risk.

Low-risk stress fractures usually respond to conservative treatment. However, one-third may not respond and can evolve into high-risk fractures.

Hotzinger reported the first case of a stress fracture treated with ESWT in 1999. Generally, a focused device is indicated using mid- or high-energy levels and may necessitate general anesthesia. ESWT should be tried before resorting to surgery [15].

Avascular Bone Necrosis

Avascular necrosis (AVN) is a disease where there is cellular death and bone infarction due to interruption of the blood supply. The etiology is multifactorial and is most common in the head of the femur. It also occurs in the humerus, tibia, talus, scaphoid, and lunate.

Conservative treatment is usually unsuccessful, and surgery has been recommended for symptomatic cases depending on the stage of the AVN. Core decompression with or without bone grafting (fibular or cancellous chips) is the gold standard to preserve the femoral head. Rotational femoral neck osteotomy is another option.

ESWT has been used since 2001 for ARCO (Association Research Circulation Osseous) stage 1–3 AVN of the femoral head.

Wang et al. compared ESWT and core decompression with 8–9 years follow-up. Clinical outcomes showed good or excellent results in 76% and fair or poor in 24% in the ESWT group, as compared to 21% good or excellent and 79% fair or poor in the surgical group [16].

Bone ischemia is also an indication for the use of ESWT and early diagnosis using MRI, and early treatment is imperative to try and prevent bony collapse and the onset of osteoarthritis [17].

There is at present no good evidence for the use of ESWT in bone ischemia and necrosis in the humerus, tibia, talus, scaphoid, and lunate.

Osteochondritis Dissecans

Osteochondritis dissecans (OCD) has probably a repetitive stress etiology and is divided into the juvenile and adult forms. Spontaneous healing occurs in 70% of the juvenile form.

OCD is usually in the knee but can also occur in the ankle.

Case reports of ESWT in OCD of the knee with arthroscopy have shown good results with return to sport.

No evidence-based treatment recommendations can be made at this stage [18].

Skin Ulcers

ESWT has been used for delayed wound healing and chronic diabetic and nondiabetic ulcers for several years.

ESWT for skin wound healing started in Vienna in 2004 after observing healing of a sinus during ESWT treatment of a fracture nonunion of a tibia. As of 2013, 700 patients have been treated at the same center in Vienna with 70% wound healing [19].

Moretti et al. have shown in a prospective randomized study on neuropathic diabetic foot ulcers (DFU) that 53% achieved complete healing after ESWT in 60.8 days.

Wang et al. compared ESWT to daily hyperbaric oxygen in DFU and achieved 31% healing as compared to 22% in the hyperbaric group.

ESWT has been also used in delayed healing of traumatic and postsurgical wounds including flaps with good results. ESWT should be started after wound debridement. It should be used together with appropriate dressings and antibiotics.

As more Level 1 studies are done on the above indications, physicians will be more confident to use ESWT for these approved indications. The techniques of both focused shockwaves and radial pressure waves are being improved.

ESWT has few side effects and is generally a safe treatment in the hands of qualified physicians. It should be considered and used before any surgery is planned on the patient.

The next section is empirically tested clinical uses where there are no strong evidence-based studies.

Common Empirically Tested Clinical Uses∗

2.1. **Tendinopathies**
- 2.1.1. Rotator cuff tendinopathy without calcification
- 2.1.2. Medial epicondylopathy of the elbow
- 2.1.3. Adductor tendinopathy syndrome
- 2.1.4. Pes anserinus tendinopathy syndrome
- 2.1.5. Peroneal tendinopathy
- 2.1.6. Foot and ankle tendinopathies

2.2. **Bone Pathologies**
- 2.2.1. Bone marrow edema
- 2.2.2. Osgood-Schlatter disease: apophysitis of the anterior tibial tubercle
- 2.2.3. Tibial stress syndrome (shin splint)

2.3. **Muscle Pathologies**
- 2.3.1. Myofascial syndrome
- 2.3.2. Muscle sprain without discontinuity

2.4. **Skin Pathologies**
- 2.4.1. Cellulite

∗Following ISMST

Tendinopathies

The evidence for rotator cuff tendinopathy without calcification is not as strong as with calcification. It is used by physicians and also for other shoulder pathologies like frozen shoulder, bicipital tendinitis, and postsurgical stiffness.

The other tendinopathies include medial epicondylopathy (golfer's elbow), adductor tendinopathy, pes anserinus tendinopathy, and foot and ankle tendinopathies.

Bone Pathologies

Bone marrow edema is an excess of fluid in the bone marrow due to several causes. The pathogenesis is unknown, but it can be confirmed by the use of MRI. It may represent an early stage of avascular necrosis.

Prospective studies have shown early pain relief and functional improvement with focused shockwaves.

Osgood-Schlatter is another indication for the use of ESWT.

Tibial stress syndrome (shin splits) is also another empirical indication for the use of ESWT.

Muscle Pathologies

ESWT has been used in muscle injuries and sprains with good results.

Myofascial syndrome is a pain of muscular origin that arises from trigger points. It is characterized by regional pain, palpable taut muscular band, and referred pain along the muscle.

It is usually refractory to conventional treatment and has attracted the use of ESWT both focused and radial for its treatment [20].

The common areas treated are the upper trapezius, shoulder, lumbosacral muscles, quadriceps, biceps femoris, and gastrocnemius.

Skin Pathologies

ESWT was first used in 2005 for the treatment of cellulite in females. Knobloch did a meta-analysis of 12 studies including 5 RCTs in 2017. Focused as well as radial and combinations have been effective in the treatment of cellulite of the lower limbs with follow-up from 3 to 12 months. This is a very promising indication for cosmesis [21].

The third category of indications is exceptional indications, and it is recommended that they be handled by expert ESWT practitioners.

Urological pathologies like erectile dysfunction have caught the attention of ESWT physicians but will not be discussed in this chapter.

Exceptional Indications: Expert Indications∗

3.1. **Musculoskeletal Pathologies**
- 3.1.1. Osteoarthritis
- 3.1.2. Dupuytren's disease
- 3.1.3. Plantar fibromatosis (Ledderhose's disease)
- 3.1.4. De Quervain's disease
- 3.1.5. Trigger finger

3.2. **Neurological Pathologies**
- 3.2.1. Spasticity
- 3.2.2. Polyneuropathy
- 3.2.3. Carpal tunnel syndrome

3.3. **Urological Pathologies**
- 3.3.1. Pelvic chronic pain syndrome (abacterial prostatitis)
- 3.3.2. Erectile dysfunction
- 3.3.3. Peyronie disease

3.4. **Others**
- 3.4.1. Lymphedema

∗Following ISMST

Musculoskeletal Pathologies

Experiments by CJ Wang et al. in rats have proven beneficial effects in osteoarthritis of the knee. This has stimulated interest in the use of ESWT for this common problem of osteoarthritis in humans.

ESWT has an anti-fibrotic action mediated by TGF-β interaction and has shown to be beneficial in early Dupuytren's and Ledderhose's diseases. It can reduce pain and slow down disease progression.

It has been used for De Quervain's tenovaginitis and trigger fingers as a noninvasive procedure.

Neurological Pathologies

Carpal tunnel syndrome is treated by both neurologists and orthopedic surgeons. Studies have shown good results in mild and moderate carpal tunnel syndrome using either focused shockwaves or radial pressure waves.

Experimental Indications∗

4.1. Heart muscle ischemia [22]
4.2. Peripheral nerve lesions
4.3. Pathologies of the spinal cord and brain
4.4. Skin calcinosis
4.5. Periodontal disease
4.6. Jawbone pathologies

4.7. Complex regional pain syndrome (CRPS)
4.8. Osteoporosis
∗Following ISMST

In orthopedics, ESWT has been used by some physicians for complex regional pain syndrome (CRPS) and osteoporosis.

It is, however, too early to claim any good results in these two conditions.

Complications

ESWT has been always assumed to be safe. Pain during treatment should not be considered as a complication, but efforts should be done to reduce treatment pain. High-energy treatment for bone pathologies can be done under general anesthesia in selected patients.

Reported complications like transient redness, pain immediately following treatment, headache, dysesthesias, swelling, ecchymoses and/or petechiae, bruising, and a throbbing sensation are all rare and not serious.

Conclusions

In 1997, Haupt wrote, "In patients in whom conservative treatment has failed, surgery used to be the only choice, but its success rate barely exceeds that of shock wave therapy and surgery can still be done if shock wave therapy fails" [23].

Twenty years after Haupt wrote this, sadly many surgeons, physicians, and allied medical personnel are unaware of ESWT or believe it to be a form of alternative medicine.

It has become established in the treatment of several tendinopathies and bone pathologies [24–26]. It should be universally taught in medical colleges and physical therapy institutes.

The treatment protocols are variable and individualized, and further research is necessary to standardize the protocols.

It is not only a science but an art. All practitioners should be certified by national or international organizations.

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Basic Imaging in Orthopedics

34

E. McLoughlin, E. M. Parvin, S. L. James, and R. Botchu

Introduction

Radiological imaging is a routinely used diagnostic tool in the evaluation of the orthopedic patient. Imaging modalities including conventional radiography, ultrasound (US), computed tomography (CT), magnetic resonance imaging (MRI), and nuclear medicine (NM) are routinely used in the assessment of musculoskeletal abnormalities. The choice of imaging modality used should be tailored to the individual, but patient factors, the experience and expertise of the referring clinician and the radiologist, as well as the availability, safety, cost, and invasiveness of the technique should be considered.

In this chapter, we review the basic physics of each imaging modality. The role of each modality as well as its advantages and limitations in the orthopedic setting will also be highlighted.

Radiation Types and Radiation Safety

Imaging modalities are divided into two types based on whether they use ionizing or non-ionizing radiation.

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Ionizing radiation is electromagnetic radiation that has enough energy to detach electrons from atoms or molecules inside the cells of the body, a process known as ionization; this process can cause cell damage or death. By contrast, non-ionizing radiation has sufficient energy only to move electrons to a higher energy state, known as excitation, and is a much safer type of radiation.

Conventional radiography, CT, and fluoroscopy are all X-ray techniques, which use ionizing radiation. Nuclear medicine uses radioactive tracers that emit gamma rays and are another form of high-energy electromagnetic ionizing radiation. By contrast, US and MRI both involve non-ionizing radiation. US use high-frequency sound waves, and MRI low-energy electromagnetic radio waves and a strong magnetic field.

The harmful consequences of ionizing radiation are categorized as deterministic or stochastic effects:

Deterministic effects occur shortly after exposure to a radiation dose above the acceptable threshold dose and cause cell malfunction or death. The severity of deterministic effects increases with the radiation dose and can be mild, moderate, or severe, resulting in either local damage to the exposed tissues (e.g., skin erythema/necrosis, cataracts, hair loss, or infertility) or whole-body effects leading to bone marrow damage, radiation sickness, or death. These effects are very unlikely to occur as a result of radiation doses used in routine diagnostic radiology.

Stochastic effects occur following a latent period of years after radiation exposure and are caused by cellular DNA damage, resulting in the possibility of cancer induction and teratogenesis. The risk of stochastic effects increases with dose, but in contrast to deterministic effects, the severity is independent of dose.

In view of the fact that ionizing radiation is harmful, consideration must be given to the radiation dose to the patient when imaging using ionizing radiation is requested. In all cases, the radiation exposure should be justified, and the radiation dose kept "as low as reasonably practicable" to acquire a diagnostic examination, in line with the Ionizing Radiation (Medical Exposure) Regulations 2017 (IR(ME)R 2017) [1]. Imaging children and pregnant women requires special consideration over the normal adult population because their tissues are more radiosensitive, and safer non-ionizing techniques should be considered if clinically appropriate. In addition, all staff working with ionizing radiation should by adequately protected against the detrimental effects of radiation through the provision of personal protective equipment and radiation monitoring devices, in line with the Ionizing Radiation Regulations 2017 (IRR 2017) [2].

Choosing the Appropriate Imaging Modality

Different imaging modalities are used to assess the existence, type, and severity of bone, joint, and soft tissue disorders. It is important that the radiologist and orthopedic surgeon have an understanding of the indications, advantages, and

Modality	Advantages	Disadvantages
Radiography	• Rapid image acquisition • Fluoroscopy allows for dynamic real-time assessment • Cheap and widely available	• Ionizing radiation • Poor soft-tissue resolution
Computed Tomography	• Rapid image acquisition • Multiplanar and 3D volume rendering capabilities • High resolution, detailed images • Excellent bony detail	• Ionizing radiation • Poor soft-tissue resolution • Expensive
Ultrasound	• Non-ionizing radiation • Dynamic real-time assessment • No patient contraindications • Cheap and widely available	• Operator dependent • Poor bone resolution • Risk of thermal damage to tissues with prolonged use • Limited views in patients with raised body mass index
Magnetic resonance imaging	• Non-ionizing radiation • High resolution, detailed images • Multiplanar capabilities • Excellent soft-tissue detail	• Slow image acquisition • Patient contraindications (e.g., cardiac pacemakers, aneurysm coils, metallic foreign bodies) • Expensive and no longer applies
Nuclear medicine	• High sensitivity in detecting bone abnormalities by identifying changes in bone metabolism which precede changes in bone morphology • Whole body imaging can be performed	• Ionizing radiation • Low specificity • Low spatial resolution • Expensive and low availability

Fig. 34.1 Table showing advantages and disadvantages of each imaging modality

limitations of each imaging technique. Plain radiography is recommended as the first-line imaging tool to detect bone or joint abnormalities or traumatic injuries, with more detailed assessment achieved using CT or MRI. Abnormalities arising in soft tissues including ligaments, tendons, muscles, and subcutaneous tissues are evaluated using MRI and US. An understanding of the basic physics and limitations of each imaging modality is important and can help the clinician determine the most appropriate imaging technique to reach the diagnosis. Figure 34.1 is a table that outlines the advantages and disadvantages of each imaging modality.

Imaging Modalities

Radiography

Physics

X-rays are produced by an X-ray tube and pass through the body where they are variably absorbed by the different body tissues; high-density bone absorbs more X-rays than low-density soft tissues such as muscle and fat. The image is then captured on a detector placed behind the patient, and the resulting two-dimensional image demonstrates excellent contrast between the different tissues within the imaged field.

Radiographic Views

Two radiographic projections perpendicular to each other (anteroposterior (AP) and lateral) are acquired. This optimizes visualization of the area of interest while reducing potential misinterpretation of overlapping structures (Fig. 34.2). Additional views may be helpful in in assessing complex joints. Weight-bearing

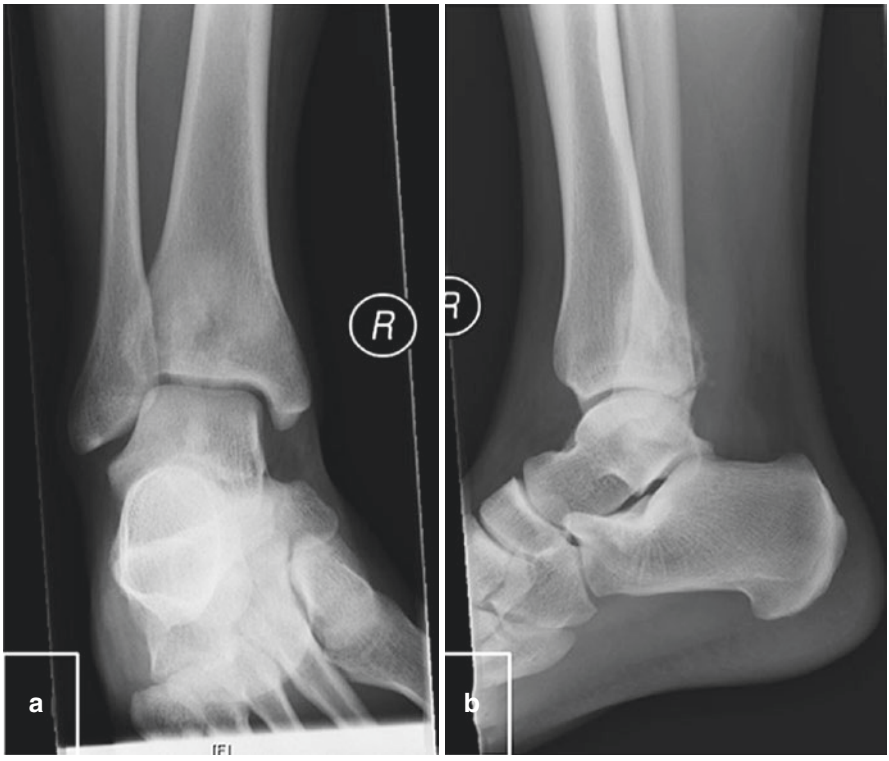

Fig. 34.2 AP (**a**) and lateral (**b**) radiographs of the ankle demonstrating a mixed sclerotic/lytic lesion in the distal tibia with periosteal reaction and extraosseous extension. Biopsy confirmed an osteosarcoma

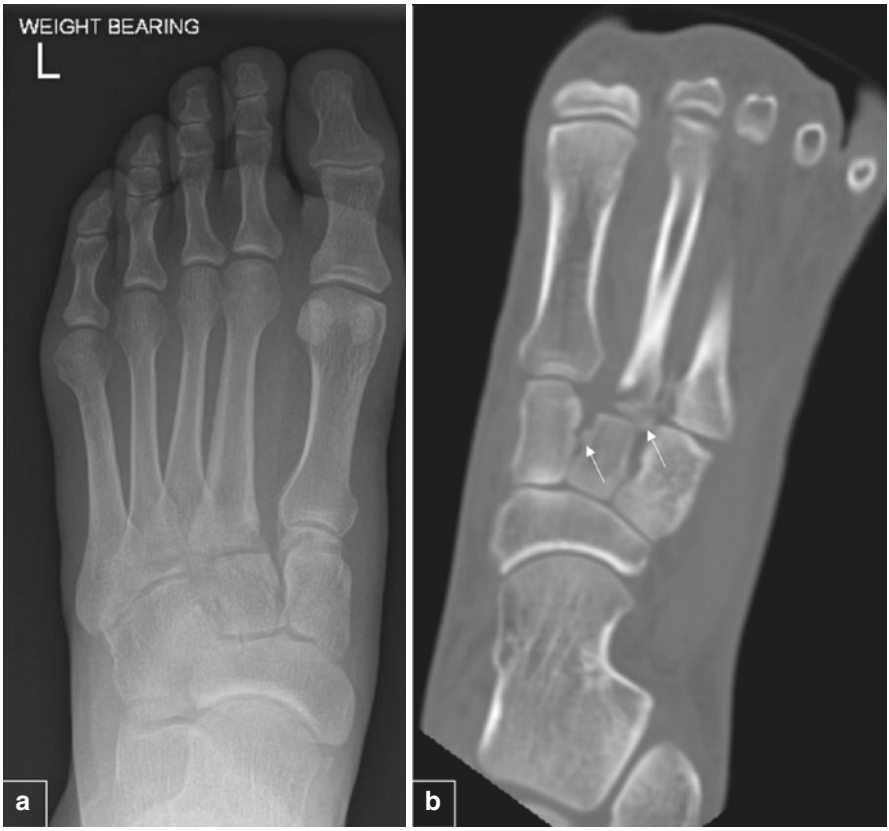

Fig. 34.3 AP weight-bearing radiograph (**a**) of the foot demonstrating widening of the interval between the first and second metatarsal highly suspicious for Lisfranc ligamentous injury. Coronal CT (**b**) demonstrates fractures of the medial cuneiform and base of the second metatarsal (arrows) in keeping with Lisfranc fracture dislocation. (Images courtesy of Dr. Ganesh Retnasingam MBChB, MRCSEd, PGCME, FRCR, Consultant MSK Radiologist, Liverpool University Hospitals NHS Foundation Trust)

or stress views allow for dynamic assessment of the joint space using under the weight of the body and can be useful if there is concern for a ligamentous injury (Fig. 34.3), although cross-sectional imaging (such as CT or MRI) is usually required in this setting and allows for more accurate evaluation of the involved structures [3].

Indications

Radiography is the most widely used imaging modality in the orthopedic setting. This technique uses X-rays to visualize the internal structures of the body and is the first-line imaging tool in the assessment of bone and joint disorders and traumatic injuries. Bone lesions, fractures, infection, loose and radio-opaque foreign bodies, and degenerative joint disease can be identified on plain radiographs. Fracture healing and joint prostheses can be assessed with serial radiographs.

Limitations

The poor resolution of soft tissue structures on plain radiographs is a known limitation of radiography. However, subtle changes can be identified and used as indirect signs of bony injury, for example, elbow joint effusion with elevation of the posterior fat pad indicating an underlying occult radial head fracture as seen in Fig. 34.4.

A further limitation of conventional radiography is that a diagnosis of acute bone or joint infection may be missed on early plain radiographs as the early changes associated with these conditions will not be apparent until 10–21 days after the onset of infection, leading to a delay in diagnosis (Fig. 34.5) [4]. In addition, subtle stress or un-displaced fractures may be missed on plain radiographs, and cross-sectional imaging may be required to reach a diagnosis (Fig. 34.6).

Fluoroscopy

This technique allows dynamic, real-time images to be obtained by using a continuous X-ray beam. This has a number of important uses in orthopedics and is commonly used to assess needle placement in minimally invasive procedures such as vertebroplasty and joint/spinal steroid injections and to assess fracture reduction (Fig. 34.7), joint stability, and hardware placement intraoperatively.

Arthrography/Myelography

Arthrography and myelography involve the injection of a contrast agent into the area of interest (joint space in arthrography or subarachnoid space in myelography). Needle placement is performed under fluoroscopic guidance to ensure accurate needle positioning and injection of contrast. Once contrast has been injected into the

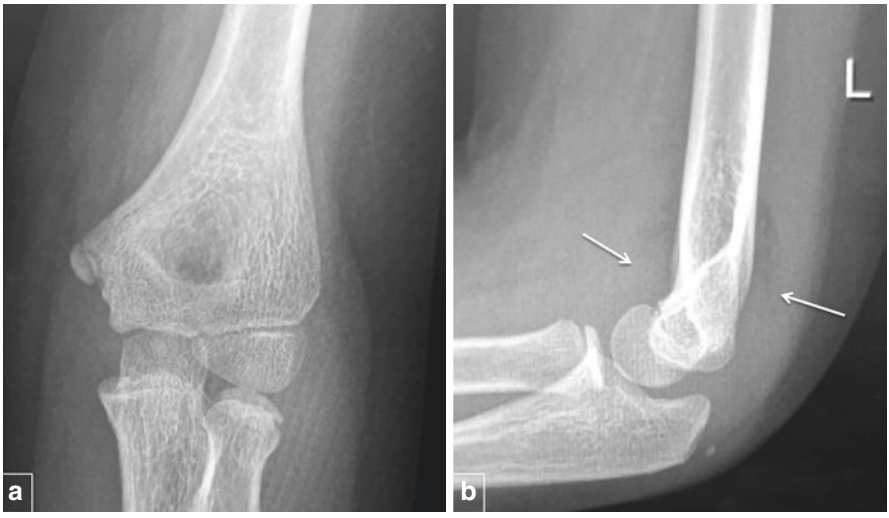

Fig. 34.4 AP (**a**) and lateral (**b**) views of a pediatric elbow demonstrating an elbow joint effusion with elevation of the anterior and posterior fat pads (arrows), highly suggestive of an underlying occult supracondylar fracture

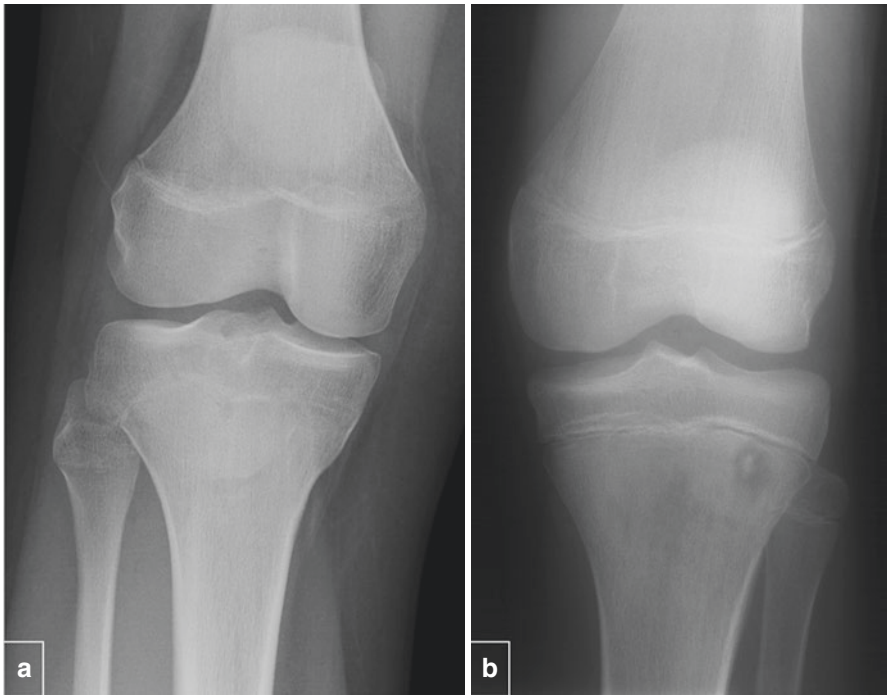

Fig. 34.5 AP radiographs of the knee in a pediatric patient with knee pain performed 6 weeks apart. No focal abnormality was seen on the initial radiograph (**a**). The radiograph performed 6 weeks later demonstrates an oval lucency in the tibial metaphysis with central sclerotic focus in keeping with osteomyelitis with sequestrum formation (**b**)

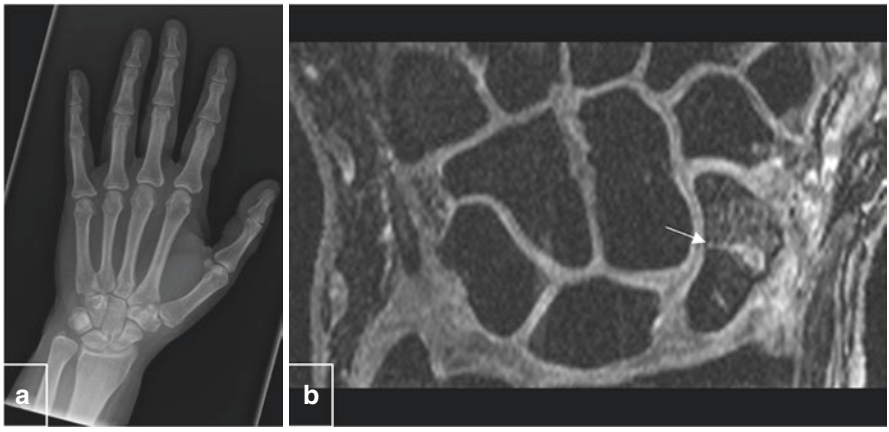

Fig. 34.6 AP radiograph of the wrist (**a**) showing a normal scaphoid in a patient with anatomical snuffbox pain following a fall. Coronal MRI image of the wrist (**b**) demonstrates an un-displaced fracture of the scaphoid waist (arrow), not visualized on the preceding radiograph. (Images courtesy of Dr. Ganesh Retnasingam MBChB, MRCSEd, PGCME, FRCR, Consultant MSK Radiologist, Liverpool University Hospitals NHS Foundation Trust)

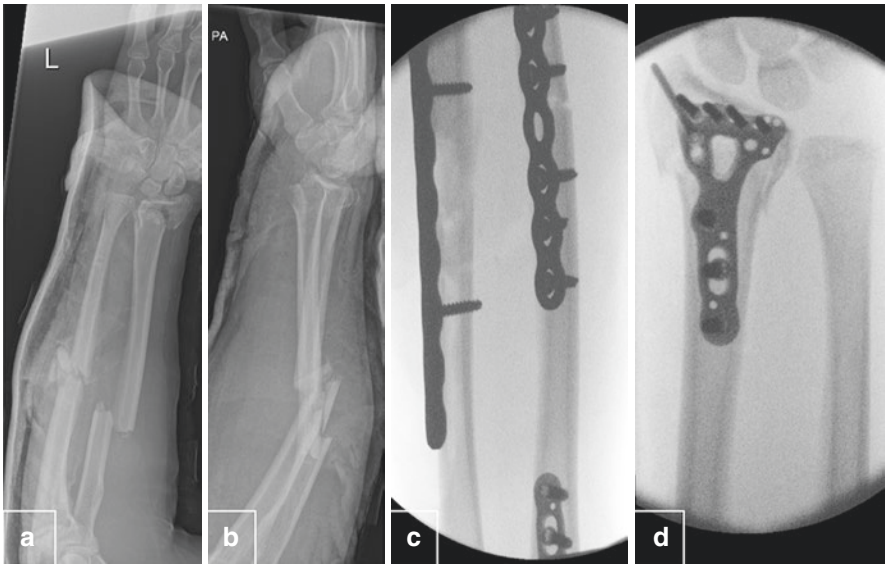

Fig. 34.7 AP and lateral radiographs (**a**, **b**) of the forearm demonstrating fractures of the midshaft of the distal radius and ulna and distal radial fracture. Intraoperative fluoroscopic images (**c**, **d**) assessing open fracture reduction and internal fixation with satisfactory fracture alignment. (Images courtesy of Dr. Ganesh Retnasingam MBChB, MRCEd, PGCME, FRCR, Consultant MSK Radiologist, Liverpool University Hospitals NHS Foundation Trust)

target site, cross-sectional imaging with CT or MRI is performed to allow detailed assessment of the joint space in arthropathy or movement of the contrast column in the subarachnoid space in myelography (Fig. 34.8).

Computed Tomography (CT)

Physics

CT imaging uses X-rays to produce cross-sectional images of the body. The patient is positioned supine on a CT table, which moves perpendicularly through the CT gantry. An X-ray tube is located in the CT gantry. As the gantry rotates 360° around the patient, the X-ray beam is projected from all directions and transmitted through the patient. At the same time, the table is moved through the gantry. The tissues attenuate the X-ray beam to varying degrees based on the density of the tissue they pass through, and the unattenuated bean is then detected by detectors on the opposite side of the gantry. Complex computing techniques are then used to process the data from the detectors, converting the X-ray beam attenuations of the various tissues into attenuation coefficients (measured as Hounsfield units (HU)) based on their density relative to water (Fig. 34.9). The resulting dataset is used to create a gray-scale axial CT image. The technique of 'windowing' allows the viewer to

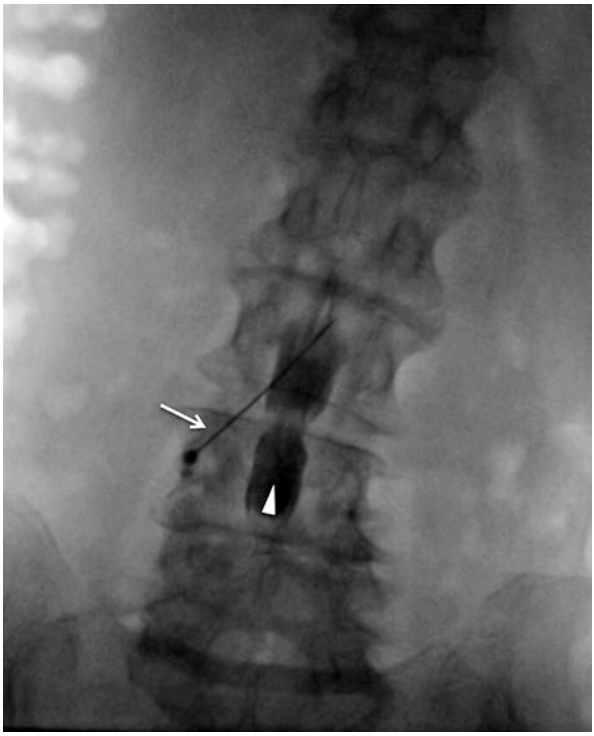

Fig. 34.8 Myelogram of the spine with contrast instilled into the subarachnoid space (triangle) using a 22G needle (arrow) placed under fluoroscopic guidance

Fig. 34.9 Table with HU values for different structures in the body

Structure	Hounsfield units (HU)
Water	0
Air	–1000
Fat	–50 to –200
Muscle	25–40
Bone	200 to 1000

achieve greater contrast over part of the range of Hounsfield units and thereby to examine certain tissues in more detail.

The slice thickness is determined by the speed of movement of the table; in the musculoskeletal setting, a slice thickness of 0.75 mm is typically used. From the dataset, two-dimensional reformatting of the axial slices into coronal and sagittal planes can be performed which allows for more detailed, accurate assessment of complex anatomy or subtle pathology. Three-dimensional volume-rendered images can also be acquired and are extremely useful in surgical planning of complex fractures (Fig. 34.10).

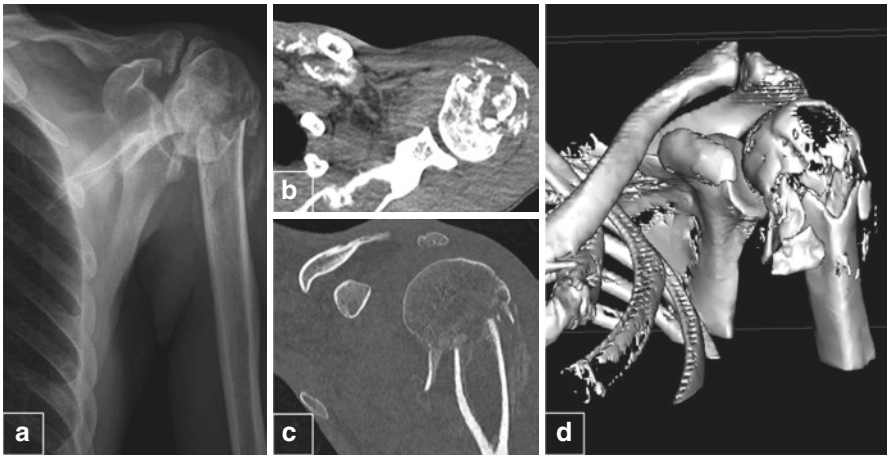

Fig. 34.10 AP radiograph (**a**), axial CT soft tissue window (**b**), coronal CT bony window (**c**), and 3D volume-rendered CT (**d**) of comminuted left proximal humeral and scapular fractures with dystrophic callus formation in a patient in intensive care. (Images courtesy of Dr. Ganesh Retnasingam MBChB, MRCEd, PGCME, FRCR, Consultant MSK Radiologist, Liverpool University Hospitals NHS Foundation Trust)

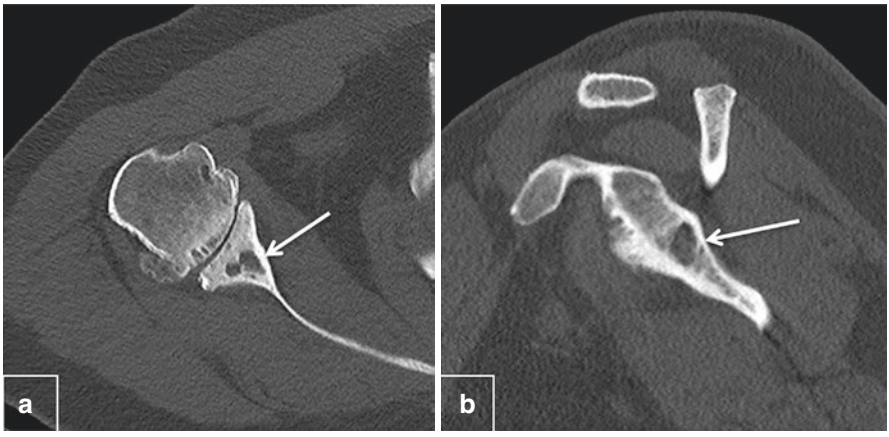

Fig. 34.11 Preoperative planning CT of the shoulder prior to shoulder replacement demonstrating severe glenohumeral joint degeneration with subchondral cysts in the glenoid on axial (**a**) and sagittal (**b**) views

Indications

CT is a valuable imaging technique and commonly used to evaluate bones and joints. It allows for more in-depth evaluation of the musculoskeletal system than radiography owing to its multiplanar capability and excellent contrast resolution. CT is useful in characterizing complex fractures, assessing fracture healing and the positioning and integrity of hardware, evaluating joints and their suitability for prosthetic placement (Fig. 34.11), and as a problem-solving tool to identify

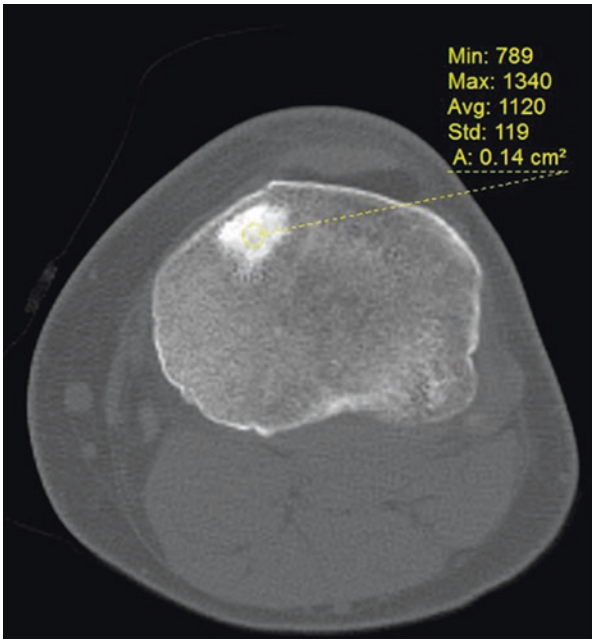

Fig. 34.12 Axial CT demonstrating a sclerotic bone lesion in the distal femur with an HU value of 1120 in keeping with a benign bone island. CT can be used to confirm the diagnosis of a sclerotic bone lesion by measuring the HU of the lesion. An HU value of >885HU confirms the diagnosis of a benign bone island, and a sclerotic bone metastasis can be reliably excluded

radiographically occult pathology such as un-displaced fractures and loose bodies. CT can also be used in the assessment of equivocal bone lesions because of its ability to measure accurately the tissue attenuation coefficient of lesions (Fig. 34.12) [5]. In patients with malignant bone and soft tissue tumors, CT is important in the initial staging, treatment planning, and follow-up of disease. It is commonly used by orthopedic surgeons in surgical planning of complex fractures and joint replacement surgery.

Contrast-Enhanced CT

Intravenous iodine-based contrast agents may be used in the musculoskeletal setting, providing additional information on a variety of disease processes over conventional CT imaging. Iodine enhances CT images by attenuating more X-rays and increasing the contrast between tissues. This can be used to evaluate the vascularity of bone or soft tissue tumors, to assess for involvement of the neurovascular bundle in the trauma setting (Fig. 34.13) or in patients with tumors, and to identify hyperemia in inflammatory and infectious process. The most serious adverse effects associated with the use of iodine-based contrast include anaphylaxis, renal failure, and extravasation resulting in local soft tissue necrosis.

Limitations

The poor differentiation of soft tissues structures is a limitation of CT.

In addition, CT imaging is susceptible to a number of artifacts, which can impact on the quality of images and limit interpretation of the study (Fig. 34.14).

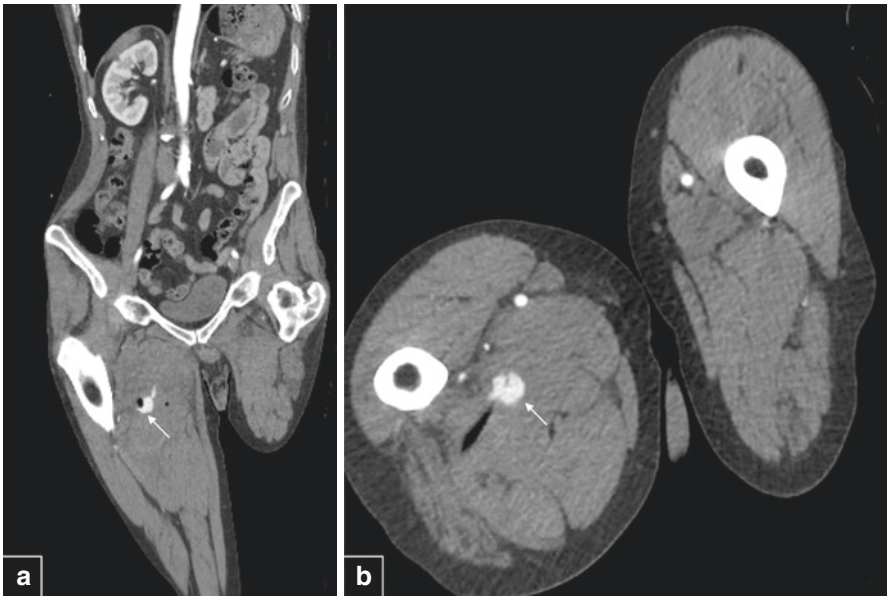

Fig. 34.13 Coronal (**a**) and axial (**b**) images demonstrating active contrast extravasation (arrow) from the medial branch of the right profunda femoris following wooden implement injury. (Images courtesy of Dr. Ganesh Retnasingam MBChB, MRCEd, PGCME, FRCR, Consultant MSK Radiologist, Liverpool University Hospitals NHS Foundation Trust)

CT artifact	Causes and amelioration
Motion	• Patient motion during scanning from movement, breathing, or cardiac pulsation results in blurring or streak artifact on the CT images. This can cause problems in interpretation
Streak	• Hyperdense objects including dental amalgam and metallic implants/prostheses can cause streak artifact. • Metal artifact reduction software (MARS) uses complex computing algorithms to improve image quality by reducing this artifact
Partial volume effect	• Causes loss of contrast between adjacent tissues when parts of these tissues are contained in a single voxel and measured tissue attenuation coefficients are averaged • Results in reduced spatial resolution and erroneous signal intensity • Reduced by using small slice thickness
Beam hardening	• Occurs when the x-ray beam, which is comprised of radiation of varying energies, passes through a structure resulting in selective attenuation of lower-energy radiation • Because the remaining higher-energy radiation is not so easily attenuated the result is low-attenuation streaks on the image.

Fig. 34.14 Table showing common artifacts seen in CT imaging

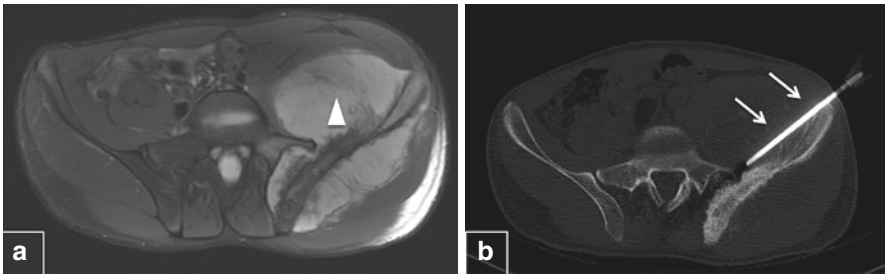

Fig. 34.15 Axial MRI of the pelvis (**a**) demonstrating a soft tissue mass (triangle) arising from the left iliac wing. A CT-guided bone biopsy (**b**) demonstrates the biopsy needle (arrows) within the extraosseous component of the lesion. Histology confirmed Ewing's sarcoma

Applications

CT-Guided Intervention

In addition to its diagnostic capabilities, CT is useful in guiding needle placement in therapeutic interventional procedures including steroid injections and cement vertebroplasty/sacroplasty, as well as in diagnostic bone biopsies (Fig. 34.15).

CT Arthrography

CT arthrography of a joint can provide additional information on the internal joint structures over conventional CT. It has a number of uses in orthopedics including assessment of joint structures including the articular cartilage, menisci, and labrum [6, 7].

Ultrasound (US)

Physics

US use high-frequency sound waves of 1–20 MHz to visualize the internal soft tissue structures of the body. The sound waves used are much higher than the sound waves within the range of human hearing (20 Hz–20 kHz).

The US transducer is comprised of piezoelectric crystals, which produce pulses of US waves by converting electric energy into sound energy. These waves traverse through tissue and change direction (reflect or deflect) depending on the relative density of the tissues at tissue interfaces. The reflected waves are then converted from sound energy to electric energy by the crystals in the transducer. Measurement of the timing and intensity of the reflected pulses allows an ultrasound image of the scanned area to be visualized on the US monitor.

US Transducers

A number of different US transducers are available, but high-frequency (9–18 MHz) linear array transducers are most commonly used in the musculoskeletal setting (Fig. 34.16). The limited depth of penetration of linear transducers can make

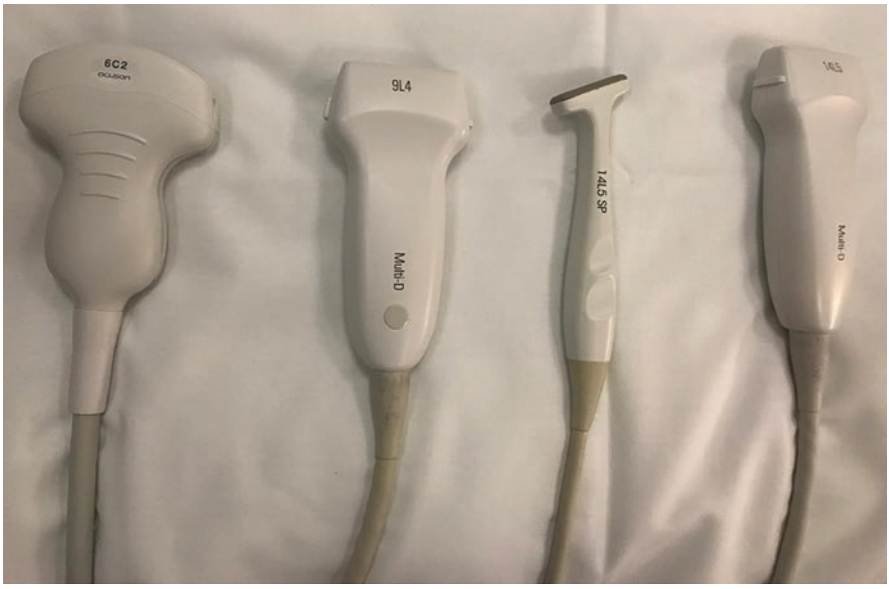

Fig. 34.16 US transducers used in the musculoskeletal setting (L–R: curvilinear, linear, hockey stick, and musculoskeletal linear probes)

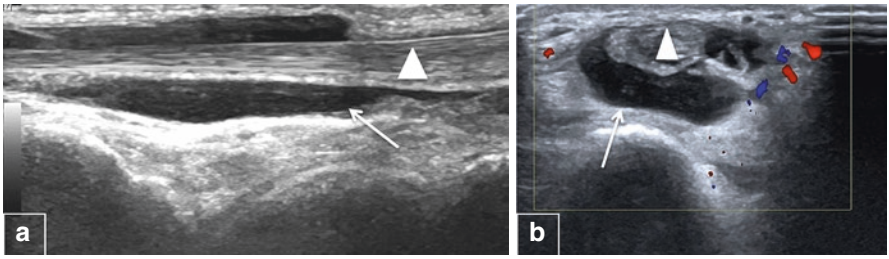

Fig. 34.17 Longitudinal (**a**) and transverse (**b**) images of the tibialis anterior tendon (triangle) with fluid seen in the tendon sheath (arrow) in keeping with tenosynovitis

scanning of patients with raised BMI or deep structures/lesions challenging. Furthermore, the small static field of the linear US transducer can make assessment of large structures extending outside of the static field difficult; however, extended field of view imaging can be employed in these cases to generate a panoramic image.

Indications

US is a safe and cost-effective imaging technique allowing high-resolution, dynamic evaluation of musculoskeletal soft tissues with real-time correlation of imaging findings with patient symptoms. US is particularly useful in the assessment of tendons, ligaments, nerves, and muscles (Fig. 34.17). It has the advantage of allowing dynamic assessment of these structures including ligament laxity and tendon

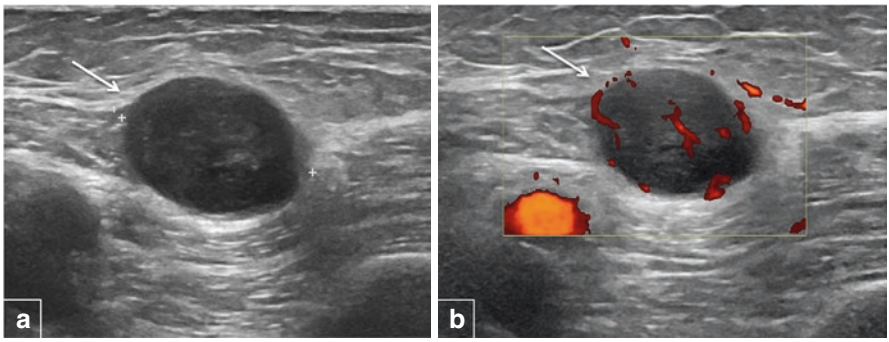

Fig. 34.18 US image demonstrating a heterogenous, predominately hypoechoic lesion (**a**) (arrow) with increased vascularity on power Doppler (**b**) (see US applications below). Biopsy confirmed a soft tissue sarcoma

Ultrasound artifacts	
Anisotropy	• Occurs in muscles and tendon when the US beam is oblique to the imaged structure • Causes loss of reflectivity of the imaged structure, which appears hypoechoic • Can result in the erroneous diagnosis of tendinopathy or tear
Acoustic shadowing	• Causes loss of signal (signal void) behind structures that strongly absorb or reflect US waves, for example, bone, calculi, metal implants, and bowel gas • Results in poor visualization of the tissues deep to these structures

Fig. 34.19 Table showing common artifacts seen in ultrasound imaging

rupture or subluxation. Joints can be assessed for the presence of a joint effusion, synovitis, and erosions/degeneration. In patients with metal-on-metal hip replacements, US is the first-line imaging tool in the assessment of pseudotumor formation (metallosis) [8]. Ultrasound is also useful in characterizing soft tissue masses (Fig. 34.18).

Limitations

US is highly operator dependent and requires an in-depth knowledge of normal musculoskeletal anatomy so that pathology can be easily differentiated from normal anatomical appearances. An understanding of the different ultrasound artifacts is also required to optimize images and prevent misinterpretation of findings (see Fig. 34.19).

A further limitation of US is the poor visualization of bone; however, evaluation of the bone-soft tissue interface, joint space, and periosteum is usually possible in areas where the bone/joint is superficial.

Applications

Doppler Imaging

The Doppler effect, whereby the frequency of sound waves from a source is increased or decreased based on whether the source is moving toward or away from the observer, can be used in US to detect and measure blood flow in tissues. The shift in frequency can be displayed in color on a standard gray-scale image (color Doppler). This is particularly useful in the musculoskeletal setting in detecting neovascularity in tendons and joints, which indicates active inflammation (Fig. 34.20). The vascularity of soft tissue lesions can also be assessed using color Doppler (Fig. 34.21). An alternative method of displaying the Doppler signal is power Doppler (Fig. 34.18) where the intensity of the color relates to the volume of the blood rather than its velocity.

US-Guided Intervention

US can be used to guide needle placement in diagnostic interventional procedures including US-guided soft tissue biopsy and in performing therapeutic interventions such as dry needling, barbotage (Fig. 34.16), steroid injections, joint/cyst aspiration (Fig. 34.17), and trigger finger release [9].

Magnetic Resonance Imaging (MRI)

Physics

Hydrogen (H) nuclei form the basis of MRI. H nuclei are positively charged (protons) and present in most body tissues, in particular water and fat. MRI involves a magnetic field and radio waves. MRI uses the interaction between hydrogen protons and a

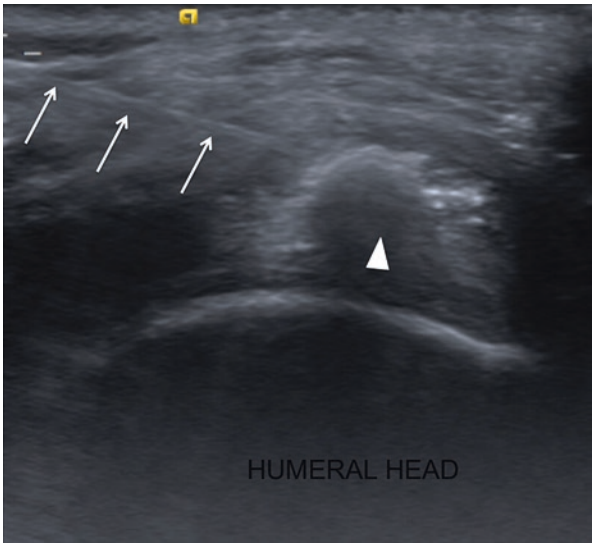

Fig. 34.20 US image demonstrating US-guided barbotage of calcific tendinopathy (triangle) with the supraspinatus tendon using a 21G needle (arrows)

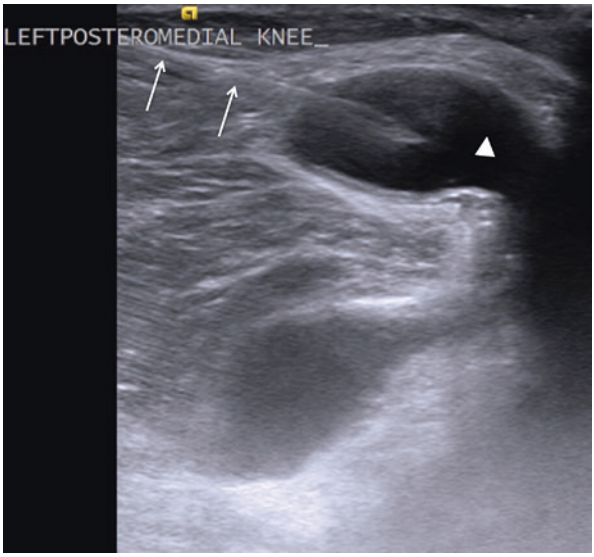

Fig. 34.21 US image demonstrating US-guided aspiration of a large Baker's cyst (triangle) in the medial popliteal fossa using a 21G needle (arrows)

strong magnetic field to create images. The patient is placed inside a uniform magnetic field, typically of field strength 0.5–3.0 T, although field strengths of up to 7.0 T are now available in the research setting. The magnetic field causes the protons in the region of interest to align with the main magnetic field, and their magnetic axes rotate around the field direction (precession). A radiofrequency pulse is then sent through the area of interest via a radiofrequency coil, and, if the frequency of the pulse is the same as the precessional frequency of the protons, they absorb energy and their alignment is changed (resonance). When the radiofrequency pulse is removed, the protons realign with the main magnetic field. This occurs at different speeds depending on the tissue in which the hydrogen protons are located. There are two key relaxation processes with relaxation times known as T1 and T2. T1 and T2 are different for different tissues. The radiofrequency signals emitted during relaxation are detected by the radiofrequency coil, or by surface coil on the area of interest, and form the basis from which the MRI image is generated. There are many complex sequences used in MRI, but a study of these is beyond the scope of this chapter. The interested reader is referred to other sources for further study [10]. The most common MRI sequences used in the musculoskeletal setting and their uses are outlined in Fig. 34.22.

Indications

MRI is a popular imaging technique in the musculoskeletal setting owing to its ability to provide images with excellent anatomical detail and unparalleled soft tissue contrast, as well as its multiplanar capability. It is the primary imaging tool in detecting soft tissue pathology involving ligaments, tendons, and muscle and is particularly useful in the assessment of intra-articular soft tissue structures such as the menisci, articular cartilage, and labrum which are poorly visualized using other imaging techniques (e.g., US). MRI is invaluable in the characterization and local staging or complications of soft tissue and bone tumors (Fig. 34.23). It may also be

MRI sequence	Uses
T1	Excellent at delineating anatomical detail and detecting hemorrhage and bone marrow-replacing lesions
T2 (with fat saturation)	Excellent for detecting fluid and edema
PD and PD with fat saturation	Good for anatomical detail and for assessing cartilage, menisci, ligaments, and tendons
Gradient echo	Excellent in detecting small foci of haemosiderin, gas, or metallic foreign bodies
STIR	Excellent for detecting fluid and edema

Fig. 34.22 Table outlining the most commonly used MRI sequences in the musculoskeletal setting

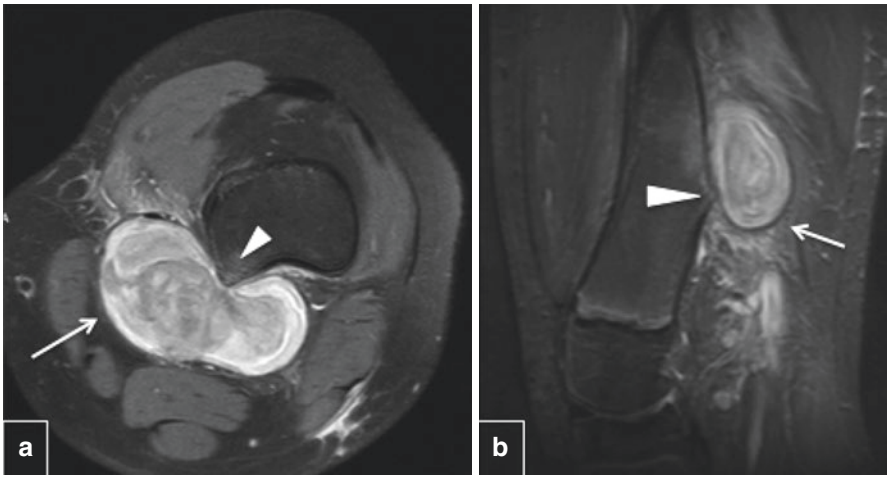

Fig. 34.23 Axial (**a**) and sagittal (**b**) MR images demonstrating an osteochondroma (triangle) arising from the distal femur abutting the popliteal artery and causing pseudoaneurysm formation (arrow)

used to identify occult inflammatory and infectious bone disorders such as osteomyelitis and chronic recurrent multifocal osteomyelitis and to identify and grade traumatic bone conditions including stress fractures/response and osteochondral injuries as well as avascular necrosis.

Contrast-Enhanced MRI

Gadolinium-based contrast agents may be used in MRI to augment images; however, this is not routinely required in the musculoskeletal setting. The most common indications for use of contrast include characterization of musculoskeletal tumors and postoperative assessment of the tumor bed for possible recurrence [11],

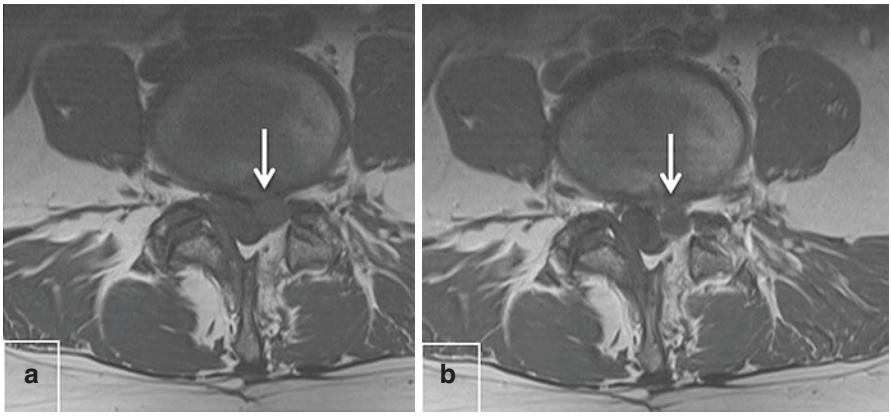

Fig. 34.24 Axial T1 (**a**) and T1 post contrast (**b**) image of the lumbar spine in a patient with previous micro-discectomy. There is soft tissue seen in the left lateral recess, which does not enhance post contrast in keeping with a recurrent disc herniation. This is easily differentiated from postoperative epidural fibrosis on contrast-enhanced MRI as the recurrent disc is avascular and does not enhance post contrast

identification of abscesses/sinus tracts in patients with infection [12], and in postoperative spinal imaging to differentiate between avascular recurrent disc herniation and vascular postoperative epidural scarring/fibrosis (Fig. 34.24) [13]. It is important to consider the significant complications associated with the use of gadolinium contrast. Patients with impaired renal function are at increased risk of developing nephrogenic systemic fibrosis (NSF), a systemic disease which causes irreversible fibrosis of skin, joints, organs, and eyes. There is increasing evidence that gadolinium can cross the blood-brain barrier potentially leading to neurotoxicity in patients who have had multiple contrast-enhanced studies, and judicious use of contrast is advised [14, 15].

Limitations

MRI is contraindicated in patients with MRI-incompatible devices including cardiac pacemakers, metallic foreign bodies, and aneurysm coils. MRI is not suitable for patients with claustrophobia due to the enclosed nature of standard MRI scanners. MRI is also subject to artifacts which can reduce the quality of images and lead to problems with interpretation (Fig. 34.25).

Applications

MR Arthrography

The visualization of joints and intra-articular structures such as ligaments and the labrum can be enhanced by injecting intra-articular contrast prior to MRI. This can be achieved using a direct approach, i.e., instillation of contrast directly into the

MRI artifact	Cause and amelioration
Motion	• Occurs due to voluntary and physiologic patient movement including breathing/ cardiac and arterial pulsation. Pulsation artifact produces multiple images of the vessel which appear as “ghosts” on the image
Susceptibility	• Occurs at the interface of tissues with markedly different magnetic properties (e.g., metal, air, haemosiderin). The magnetic field is distorted resulting in local low signal change at the interface, for example, around a foreign body or prosthesis. This can be reduced using metal artifact reduction sequence (MARS). This artifact can also be exploited to make certain lesions more conspicuous (blooming artifact from hemosiderin deposition in PVNS)
Aliasing (“wrap around”)	• Occurs when the field of view is smaller than the body part being imaged and signal from the tissues outside the field of view is superimposed on the image. Can be eliminated by increasing the field of view or using a surface coil
Magic angle	• Predominately occurs in tendons orientated obliquely to the main magnetic field causing them to appear spuriously hyper-intense which can be misinterpreted as tendinopathy

Fig. 34.25 Table outlining common artifacts seen in MRI imaging

joint under fluoroscopic guidance prior to MRI (direct arthrogram) (Fig. 34.26) or after administering intravenous contrast and performing delayed imaging of the target joint (indirect arthrogram).

Whole-Body MRI (WB-MRI)

WB-MRI is increasingly used as an alternative imaging modality in the staging and posttreatment monitoring of pediatric patients with bone and soft tissue tumors [16]. It can also be used in the screening of chronic recurrent multifocal osteomyelitis in the pediatric population [17]. Systemic muscle diseases such muscular dystrophy and dermatomyositis can also be detected and evaluated with WB-MRI [18].

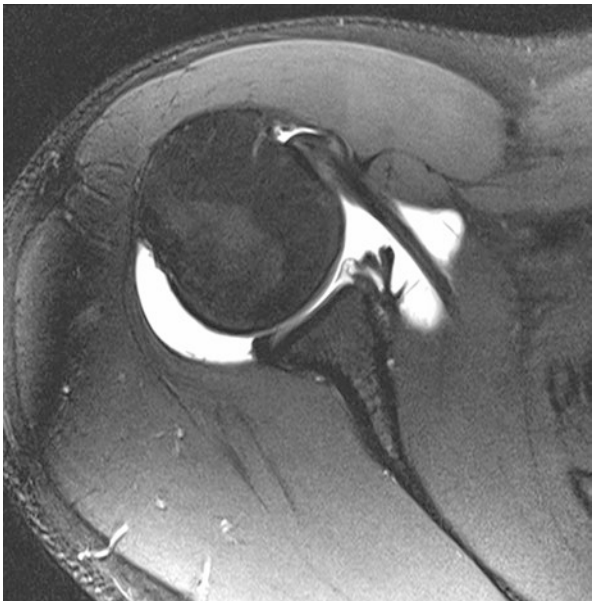

Fig. 34.26 Axial image of an MR arthrogram with contrast in situ in the glenohumeral joint demonstrating a tear of the anterior labrum

Nuclear Medicine

Physics

A radioactive material is administered to the patient in the form of a radiopharmaceutical—a substance made up of a radioactive isotope attached to a biologically active molecule designed to target the organ of interest. The radioactive isotope emits gamma radiation (high-energy electromagnetic radiation), and this radiation is detected by a suitable detector and displayed on a monitor.

There are two distinct types of NM imaging: single-photon emission (SPE) imaging with a gamma camera and positron emission tomography (PET) with a PET scanner. Both techniques work by producing two- or three-dimensional images of the bio-distribution of the radiopharmaceutical in the body. SPE imaging typically uses technetium 99m (99mTc) as its radiopharmaceutical isotope; there are a wide variety of radiopharmaceuticals that can be used to target different tissue. PET imaging most commonly uses fluorine-18 (18F) which is introduced into the body on a biologically active molecule, usually a glucose analogue such as fluoro-deoxyglucose (FDG).

SPE imaging can produce either two-dimensional images using a planar camera (e.g. bone scintigraphy) or three-dimensional images by rotating the cameras around the patient and reconstructing the acquired set of images; this is known as single-photon emission computed tomography (SPECT).

In PET imaging, the radionuclide accumulates in tissues with high metabolic activity and emits positrons (antimatter particles with the same mass as electrons but with opposite charge). Each positron will only travel a very short distance in the body before colliding with an electron. When this happens, the two particles are annihilated, and their energy converted into two high-energy gamma rays that are emitted in opposite directions. The detectors in the PET scanner detect these coincident pairs of gamma rays.

Indications

Nuclear medicine is a functional imaging modality which has a number of uses in the orthopedic setting. It is highly sensitive in identifying bone abnormalities by detecting changes in bone metabolism which predate changes in bone morphology. Different imaging techniques are used to evaluate a variety of disease processes (see Applications section). Nuclear imaging can be used to identify osteoblastic bone lesions, radiographically occult fractures, aseptic loosening of joint prostheses, bone infection, metabolic bone disease, and reflux sympathetic dystrophy.

Limitations

The major limitation of nuclear medicine is its low specificity. A variety of disease processes including infection, fractures, or bone tumors can cause increased uptake; however, differentiating the underlying cause of increased metabolic bone activity is not possible on nuclear imaging alone, and correlation must be made with clinical history and biochemical tests to reach the correct diagnosis. Furthermore, the spatial resolution of nuclear medicine imaging is low; however, the anatomical localization of areas of increased metabolic activity can be improved through the use of hybrid techniques such as SPECT-CT, PET-CT, and PET-MR in which CT or MRI images are taken at the same time.

It is important to note that bone scintigraphy is only useful in disease processes where there is increased bone turnover (osteoblastic bone activity), for example, sclerotic bone metastases and osteoblastic tumors. Osteolytic bone lesions with limited osteoblastic reaction (e.g., lytic renal cell carcinoma metastases or myeloma) will have low or absent uptake leading to a false-negative result [19].

Applications

Bone Scintigraphy

This nuclear medicine technique is performed as either a single- or dual-phase study. It uses 99mTc-labelled methylene diphosphonate (MDP) which is injected with images acquired using a gamma camera. The radiopharmaceutical accumulates at sites of increased bone metabolism and can readily detect osteoblastic bone lesions (Fig. 34.27), fractures, infection, metabolic bone disease, and joint prosthesis loosening or infection. In single-phase imaging, a whole-body technique is used, and images are acquired between 2 and 4 h after the radiopharmaceutical is injected. In dual-phase imaging, a target area is usually imaged, and images are acquired

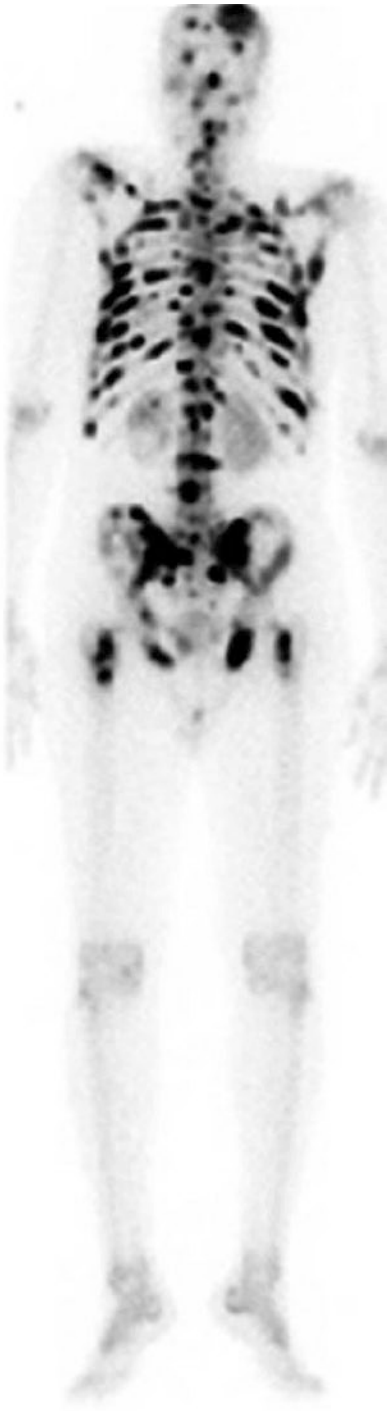

Fig. 34.27 99Tc-MDP bone scan demonstrating multifocal areas of uptake in a patient with known prostate cancer, in keeping with widespread osteoblastic sclerotic metastases

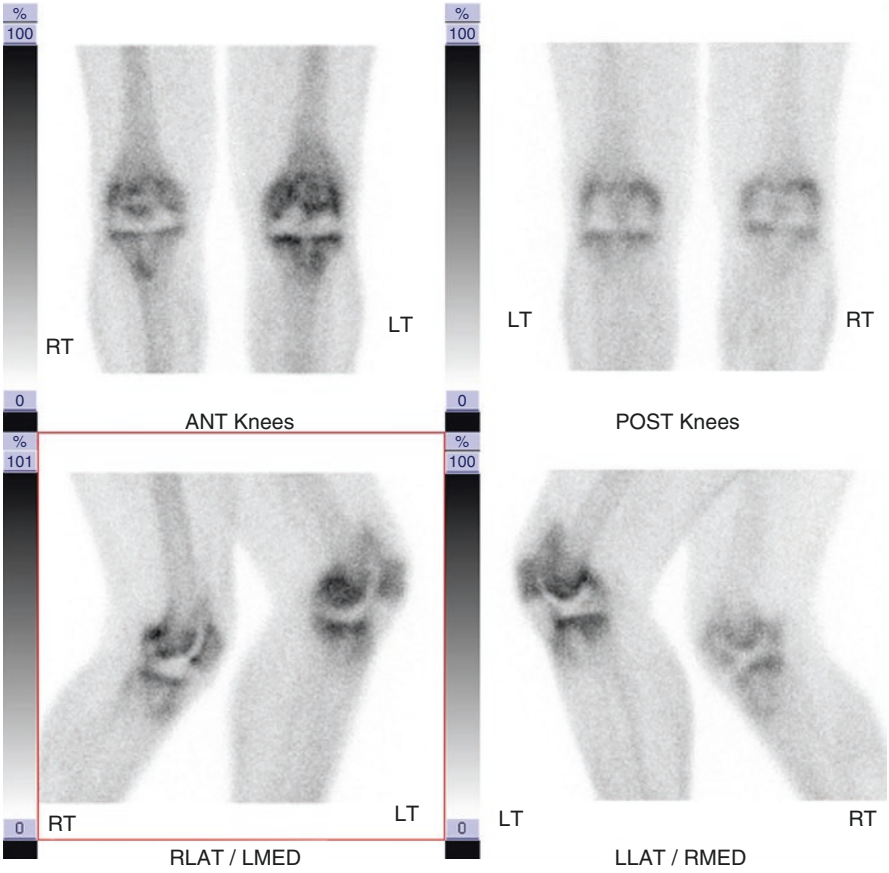

Fig. 34.28 99mTc-MDP bone scan of both knees with bilateral knee prostheses in situ. Increased radiopharmaceutical accumulation is seen around the left knee prosthesis in keeping with joint loosening

5–10 min (blood pool phase) and 2–4 h after radiopharmaceutical injection (delayed phase). If tracer uptake is seen in the blood pool phase, this suggests the presence of infection, fracture, or septic loosening of a prosthesis (Fig. 34.28).

White Blood Cell (WBC) Scan

This technique involves removing white blood cells from the patient, tagging them with a radioactive tracer (Indium-111), and reinjecting them into the patient. The tagged WBCs localize to areas of infection.

SPECT-CT

SPECT can be used in conjunction with CT (SPECT-CT) to identify bone metastases. The use of hybrid SPECT-CT provides both functional and anatomical imaging and increases the localization of bone metastases over standard bone scintigraphy.

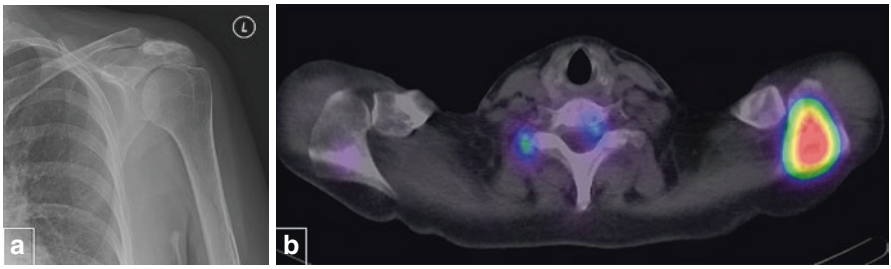

Fig. 34.29 AP radiograph (**a**) of the left shoulder demonstrating a sclerotic focus in the acromion in a patient with a history of previous breast cancer. This demonstrated intense FDG uptake on the PET-CT (**b**). Biopsy confirmed sclerotic bone metastases

PET-CT

PET imaging is invariably integrated with low-dose CT (PET-CT) which allows for anatomical localization of areas of increased activity. PET-CT can be used as a problem-solving tool to identify clinically occult bone infection, characterize equivocal bone lesions (Fig. 34.29), and differentiate between pathological and non-pathological fractures [20]. PET-CT is also occasionally used in the staging of patients with bone and soft tissue tumors and in treatment response monitoring; however, it is more widely used in patients with non-sarcomatous malignancy.

PET-MR

Integration of PET imaging with MRI (PET-MR) is possible and can be used to stage bone and soft tissue malignancies; however, the use of this technique in the musculoskeletal setting has yet to be fully established in the clinical setting [21].

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Advanced Imaging in Orthopedics

35

Moomal Rose Haris and Harun Gupta

Ultrasound Advanced Imaging

B-mode ultrasound (US) is widely used as a first-line imaging modality for a multitude of musculoskeletal conditions as it is readily available, relatively low cost, utilizes nonionizing radiation, and has no patient contraindications. It also allows the ultrasound operator to perform dynamic real-time assessment for correlation with patient symptoms. These advantages are the basis of ongoing research within the ultrasound imaging field with the aim of making it more sophisticated and of widening its applicability to various musculoskeletal areas.

Sonoelastography

The biomechanical properties of soft tissues can to some extent reflect pathophysiology of the musculoskeletal disorder [1]. Noninvasive qualitative assessment of soft-tissue elasticity and stiffness can be performed using sonoelastography [2].

Elasticity of soft tissues is defined as tissue deformability, which is determined by structure and composition [1]. Malignant soft tissues are found to be harder than normal tissues such as subcutaneous fat, muscle and connective tissues, which are used as a reference level. Elasticity of soft tissues can also be affected by other pathological processes such as tendinosis, neuromuscular diseases, and wound healing [1].

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In sonoelastography, repeated compression is applied to an area of interest using the ultrasound transducer and then tissue displacement is measured [3]. There is greater displacement of tissues that are soft and less displacement of tissues that are stiff. Recent studies have shown great promise in sonoelastography as a technique to identify Achilles, patellar, and common extensor tendinopathy of the elbow [4]. The diagnostic sensitivity of tendinosis can be increased through the combined use of sonoelastography and conventional ultrasound techniques [4].

There are three main types of sonoelastography:

1. Compression elastography: this is also known as static strain elastography [5]. It is based on the principle that compression of tissues produces strain. Tissue displacement is calculated in real time by applying manual compression to the tissue over a certain area repeatedly with an ultrasound transducer. The displacement is then converted to a color-coded distribution map, which is often superimposed over the B-mode image or displayed next to it.
2. Shear wave elastography: this is also known as dynamic elastography (Fig. 35.1). It is based on the measurement of the propagation velocity distribution of the directional shear wave formed by an ultrasound pulse [4]. It gives a quantitative assessment of tissue elasticity and does not rely on the ultrasound operator to apply manual compression to the target site. The velocity of the shear waves can

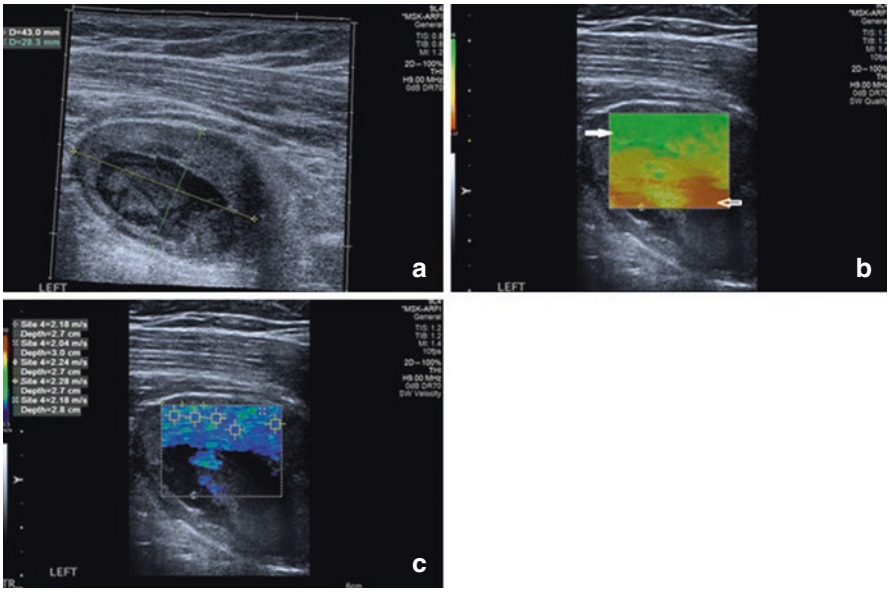

Fig. 35.1 Glomus tumor in a 62-year-old. (**a**) Transverse B-mode image demonstrating an oval mass with solid vascular peripheral component and necrotic/cystic center. (**b**) Corresponding shear wave velocity quality map with the solid area in green (good quality reading) (solid arrow) and cystic/necrotic area in red (poor quality reading). (**c**) Corresponding shear wave qualitative map, which is predominantly blue/cyan with five quantitative readings

be measured and used to assess tissue elasticity. Abnormal (harder) tissues have a higher shear wave velocity than normal (softer tissues).

3. Transient elastography: this is also known as pulsed elastography [4, 5]. It uses a short tone burst of vibration to avoid reflected interferences from forward propagation waves using a pulse-echo system. This technique can provide quantitative measurements of regional elasticity but is limited by tissue depth [6].

The limitations of sonoelastography include operator dependence and reliance on skilled, repetitive compression to obtain high-quality images.

Contrast Enhanced Ultrasound (CEUS)

Ultrasound contrast agents enhance blood flow signals and allow assessment of microcirculation. These agents are well used in the assessment of liver, pancreatic, and cardiac lesions but are now showing to be of value in musculoskeletal radiology through the assessment of inflammatory arthritidies, soft tissue lesions, and muscle degeneration [7].

The contrast agent contains gas microbubbles, which are injected intravenously and enhance the Doppler signal [7]. This allows the detection of low-speed blood flow, which is difficult to identify using standard color or power Doppler imaging.

CEUS can delineate microvasculature within inflamed joints and so detect early arthritis with high sensitivity and specificity [7–9]. Studies have shown that CEUS imaging has shown good correlation with histopathological changes of inflammatory arthritis and plays a significant role in the therapeutic monitoring of the disease [7–9].

CEUS makes it possible to assess changes in the vascularity of focal lesions and parenchymal tissue. The vascularity of tumor tissue is different from that of normal tissue. CEUS uses this premise to detect such lesions, resulting in improved sensitivity and accuracy. CEUS also has the ability to assess muscle perfusion, making it a promising diagnostic and research tool to assess muscle vitality and function, especially in the pre and postsurgical setting, for example, to assess rotator cuff muscle integrity [10].

Despite all the advantages associated with CEUS, it is still in its early stages. Therefore there is a lack of current awareness, and so image interpretation is limited. There is currently a lack of agreed clinical standards as research is still ongoing [7–9].

Fusion Imaging

Fusion imaging involves combining two imaging modalities [11]. This is achieved by preloading the data set obtained from cross-sectional imaging (computed tomography (CT) or magnetic resonance imaging (MRI)) on to the ultrasound machine. This enables real-time spatial registration of the ultrasound images with the

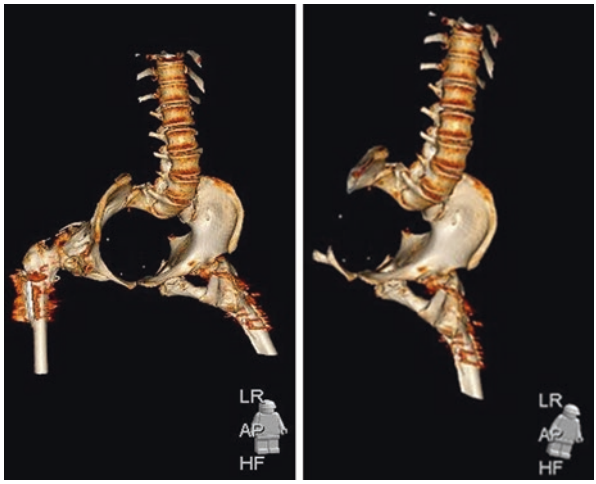

Fig. 35.2 3D volume rendering of the pelvis post trauma

previously acquired cross-sectional images [11]. This improves anatomic localization of lesions on ultrasound and has positive effects in terms of targeted image-guided injections and biopsies [11].

CT Advanced Imaging

Multislice CT was introduced in the early 1990s and has made significant improvements in bone and joint imaging [12]. CT allows extended anatomic coverage and better detail when compared to radiographs. This is of particular importance when assessing for bone union, ligamentous disruption, and prosthetic-related complications. With multislice CT imaging multiplanar reformatted images and three-dimensional (3D) rendering techniques (Fig. 35.2) can be applied and allow the clinician to view reduced images that are more relevant to the patient and their management [13]. Continued research into new CT imaging parameters has led to new significant developments in the musculoskeletal imaging field.

Weight-Bearing and Cone Beam CT Imaging

Weight-bearing (standing) radiographs are now commonly performed for the evaluation of lower limb musculoskeletal complaints with improved detection rates of joint space loss [14]. However, with regard to the hip, the Osteoarthritis Research Society has stated that while there is a theoretical advantage of imaging the hip in the standing position, in real terms supine imaging of the hip can accurately assess for joint space loss and hip morphology [15]. However, in patients with hip dysplasia, weight-bearing radiographs have been shown to be more accurate in the assessment of degenerative change than supine films [15].

Weight-bearing radiographs of the foot and ankle are standard practice when assessing for a variety of conditions. This includes osteoarthritis, hallux valgus and longitudinal foot arch abnormalities such as pes planus, pes cavus and ligamentous disruption.

Despite providing more detailed anatomic information than radiographs, due to the conventional CT scanner design lower limb imaging is predominantly limited to the supine position; thereby losing the aforementioned advantages of identifying pathology exaggerated on weight-bearing imaging. To tackle this issue previously cross-sectional weight-bearing extremity imaging was attempted by applying axial loads to joints during conventional CT scanning in the supine position. However, recent advances in CT scanning technology with the use of cone beam CT (CBCT) has enabled imaging of the lower limb to take place in the normal weight-bearing (standing) position [16, 17].

CBCT utilizes a cone-shaped X-ray beam and a flat-panel detector, which rotate around the patient. Single projection images are obtained at preset intervals, and a volumetric data set is then generated. This data set is used to reformat images in the axial, coronal, and sagittal planes. 3D-rendered imaging can also be produced from the data set [17, 18]. The advantages of CBCT includes producing images with higher spatial resolution with a faster acquisition time and also generation of a lower radiation dose than when compared to conventional CT. CBCT also has increased accuracy in the assessment of radiographically occult fractures, degenerative joint disease, and sequestrum identification in chronic osteomyelitis. CBCT can be used in combination with arthrography to assess articular cartilage, osteochondral lesions, intra-articular bodies, and synovial tumors [19].

Dual-Energy CT (DECT)

DECT is a currently evolving imaging technique. The main advantages of DECT over conventional CT in the musculoskeletal setting is that it provides additional information with regard to soft-tissue composition and reduces metallic artifact and so allows for better image quality [20, 21].

In DECT, there is simultaneous acquisition of images of one area using two different X-ray energies so different tissue composition/contrast is highlighted. Most commonly, this is attained by rapid kilovoltage switching of a single X-ray source where a single X-ray tube rapidly switches between two energy levels to obtain two data sets [22]. Dual-source DECT uses a similar rapid kilovoltage-switching technique but has two X-ray tubes and detector plates, which are positioned at right angles to each other in the gantry to obtain the two data sets. Although DECT involves two energy levels, the radiation dose administered to the patient is divided between the two, resulting in dose neutrality when compared with conventional CT [21]. Spatial resolution of DECT should be equivalent to conventional CT (Fig. 35.3).

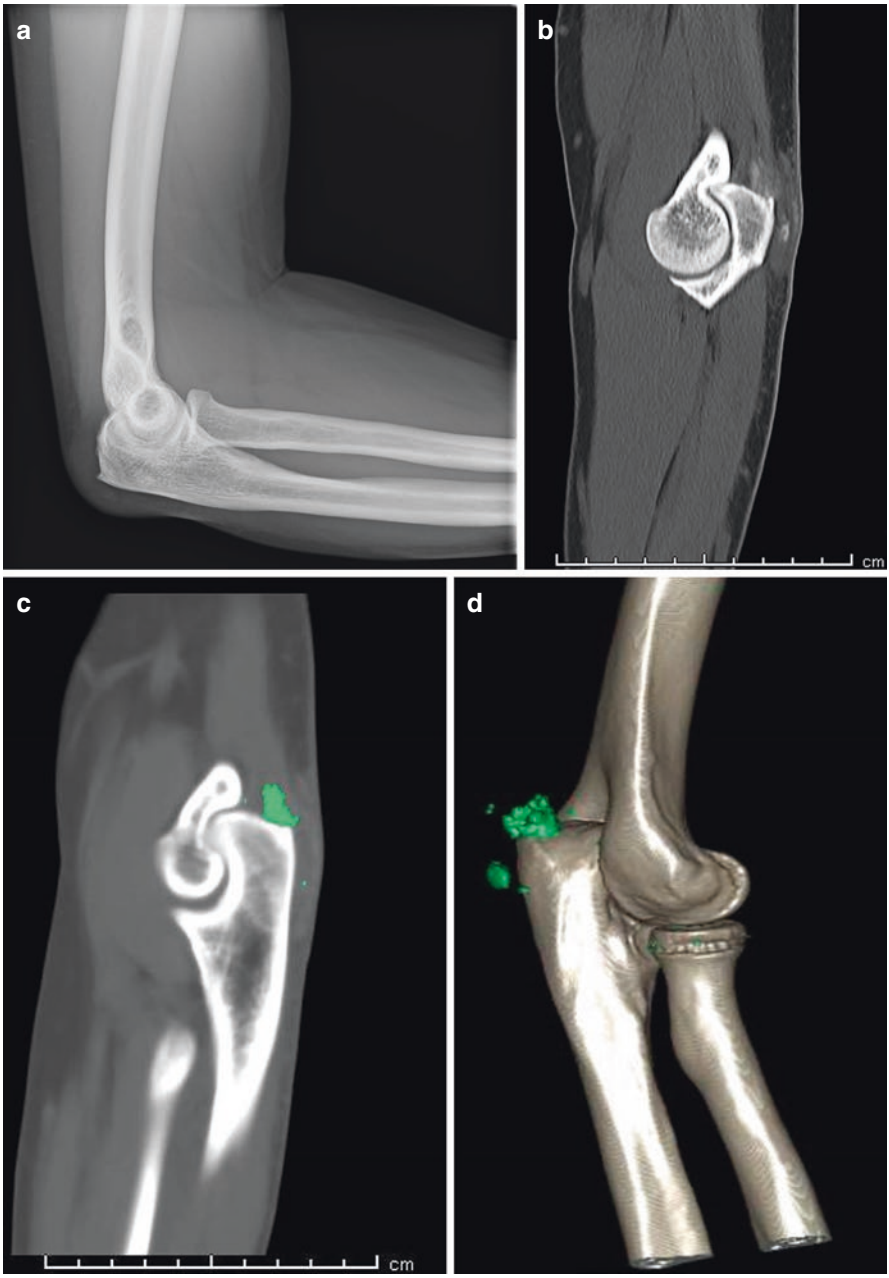

Fig. 35.3 Gouty tophi in elbow. (**a**) Radiograph showing a small olecranon spur and an olecranon bursa. (**b**) Conventional CT showing crystal deposition at the triceps insertion and olecranon bursa. (**c**) DECT highlighting the crystal/tophi deposition. (**d**) 3D volume rendering DECT highlighting the crystal tophi deposition. (Image courtesy: Dr. Jaspreet Singh, Consultant Musculoskeletal Radiologist. The Robert Jones and Agnes Hunt Orthopaedic Hospital NHS Foundation Trust)

The specific advantages of DECT are detailed below:

1. Identification of ligaments and tendons: one of the main advantages of DECT is that it produces soft-tissue contrast/composition by imaging at different X-ray energy levels. Recent studies have commented on the ability of DECT to assess for tendinosis and tendon tears and also evaluate the integrity of the stabilizing ligaments of the knee [23, 24].
2. Metal artifact reduction software (MARS): image degradation due to streak artifact from metal prosthesis results in significant image degradation and reduced diagnostic ability to detect prosthesis-related complications. Using specific post-processing software, DECT can reduce streak artifact and contrast-to-noise ratio by creating data sets at energy levels that have lower susceptibility to this artifact [25]. This allows for better assessment of the adjacent bone and soft tissues to look for complications such as osteolysis, peri-prosthetic fractures and adjacent collections.
3. Gout imaging: the current reference standard for the diagnosis of gout is considered to be the detection of monosodium urate (MSU) crystals in aspirated joint synovial fluid. However, joint aspiration can fail to demonstrate the presence of MSU crystal in up to 25% of patients with acute gout, and up to 42% of patients with gout show no laboratory findings of elevated urate levels, making this a diagnostic conundrum [26]. However, DECT has the ability to detect MSU crystals in a noninvasive manner using tissue decomposition techniques [27].
4. Detection of bone marrow edema: magnetic resonance imaging (MRI) is generally thought to be the imaging modality of choice to detect bone marrow edema. Bone marrow edema is very difficult to identify on conventional CT as it only produces very small changes in the Hounsfield attenuation values due to the presence of fine trabecular bone, which is essentially inseparable from marrow on CT [28]. However by using material decomposition protocols in DECT, the highly attenuating calcium signal of the fine trabecular bone can be removed and allows superior imaging evaluation of bone marrow [29].
5. Detection of bone metastases: it can be very difficult for radiologists to differentiate bone metastases from incidental and indeterminate bone lesions in the oncology setting. Differentiating between these lesions is important as it can make the difference in deciding whether a patient has a stable, improving, or progressive disease. There are two main ways by which DECT can differentiate between metastatic and benign bone lesions [30]:
   - Quantitative tissue decomposition: in metastatic lesions, bone content decreases due to osteolysis, with fatty marrow being replaced by soft tissue. Water content in these lesions increases due to increased vascular permeability of malignant tissue. DECT using a two-tissue decomposition algorithm has been used to show the ability of this imaging technique to differentiate between Schmorl's nodes (which also have increased water content) and vertebral body metastases and produced positive statistically significant results [30, 31].
   - Qualitative tissue decomposition: DECT can be used to define and color code cortical and trabecular bone and can make metastases more obvious [32].

MRI Advanced Imaging

MRI has revolutionized musculoskeletal imaging since its introduction in the 1970s [33]. It has the ability to produce high spatial resolution and multiplanar imaging with excellent soft-tissue contrast. Improvements in current MR imaging parameters and advancements in technology continue to evolve to meet the increasing imaging demands in the musculoskeletal field [34].

High-Field-Strength MRI

High-field-strength (3.0T) MRI is experiencing more widespread use. The major advantage of 3.0T MRI of the musculoskeletal system, when compared to the most commonly used 1.5T MRI field strength, is the doubled signal-to-noise ratio (SNR). This increase in SNR can be used to improve spatial resolution or reduce scanning time. Improved spatial resolution obtained from high-field-strength (3.0T) MRI has been shown to increase the visualization of articular cartilage abnormalities and ligamentous pathology when compared to standard strength MRI [34, 35].

Studies have also highlighted that the increased sensitivity of high-strength MRI to magnetic susceptibility changes between tissues may be advantageous to detect certain pathologies such as chondrocalcinosis and calcific deposition within tendons [36].

Moreover, 3.0T MRI has been compared to MR arthrography (MRA) at 1.5T systems, and research has suggested that 3.0T MRI is equivalent to 1.5T MRA [37] for the diagnosis of hip labral tears and cartilage delamination (Fig. 35.4). It was found that 3.0T MRI is superior to 1.5T MRA in the diagnosis of acetabular cartilage defects [37–39].

There are limited, published cost-efficiency analyses comparing 3.0T imaging of the musculoskeletal system to 1.5T imaging with varied results. It was indicated that 3.0T MRI has the ability to shorten scan time, improve diagnostic confidence in detecting pathology, as well as potentially remove the need to perform contrast studies at the 3.0T strength. However, there are multiple studies that show that although 3.0T MRI produces superior image quality [40], 1.5T imaging does perform diagnostic quality images, and so the increased cost for 3.0T is not justified.

Closed, Open, Upright, and Extremity MRI Scanners

Most institutes use the conventional closed MRI scanner with a cylindrical bore magnet. The patient lies supine and then passes through a narrow tubular tunnel which is enclosed by the magnet. In patients who suffer from claustrophobia, this process can be difficult to tolerate [41]. To deal with this, there has been ongoing development of closed scanners with shorter tubes and more internal space and open, upright, and extremity scanners to increase patient comfort.

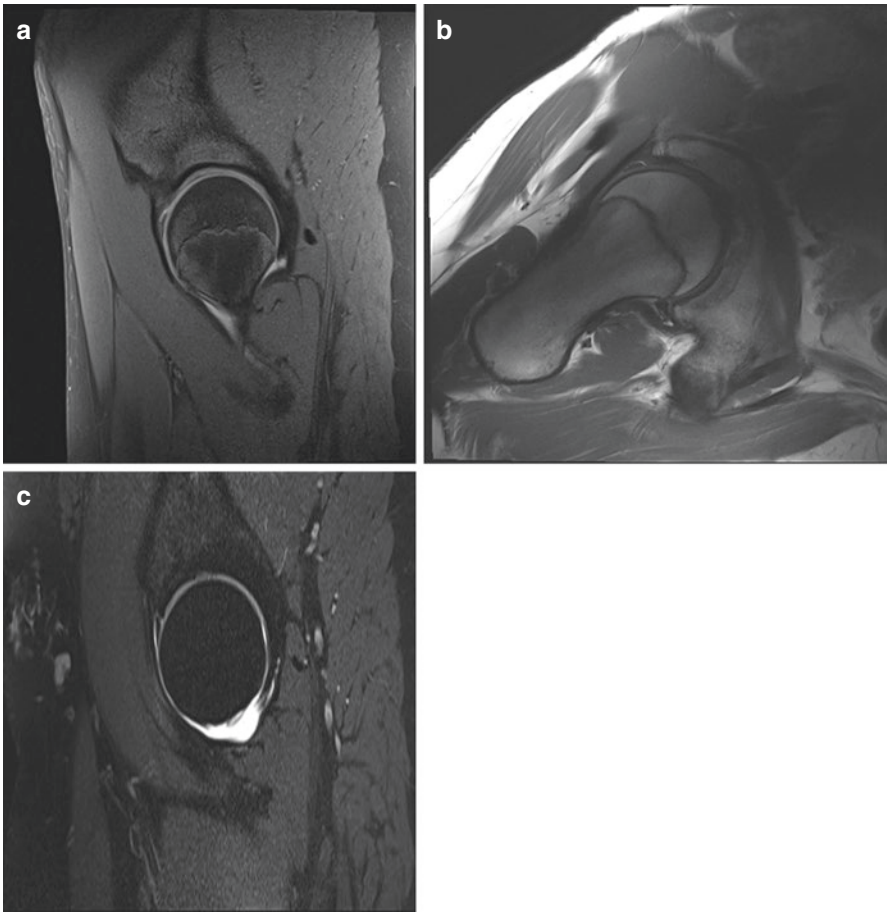

Fig. 35.4 Anterior labral tear. (**a**) 3.0T MRI of the left hip. Sagittal oblique plane. Linear high signal at the anterior labrum is in keeping with a tear. (**b**) 3.0T MRI of the left hip. Axial oblique plane. Linear high signal at the anterior labrum is in keeping with a tear. (**c**) 1.5T MR arthrogram. Axial oblique plane of the left hip in a different patient demonstrating an anterior labral tear

Open MRI: this scanner consists of two horizontally orientated metal discs, which are the poles of the magnet. The patient lies supine between the two discs. Open scanners do not have sides and are therefore more comfortable for claustrophobic or obese patients [42].

Upright MRI (uMRI): this is a type of vertically orientated MRI scanner that is open at the front and top with magnetic poles placed on each side of the patient to allow for weight-bearing (standing) and horizontal (sitting) positions. Current uMRI scanner use low-medium field magnets going up to 0.6T compared to conventional MRI scanners, which can have field strengths of up to 4.0T [41]. The lower strength magnets utilized in uMRI scanners reduces image resolution and results in longer scanning times. This can cause difficulties in patients who are

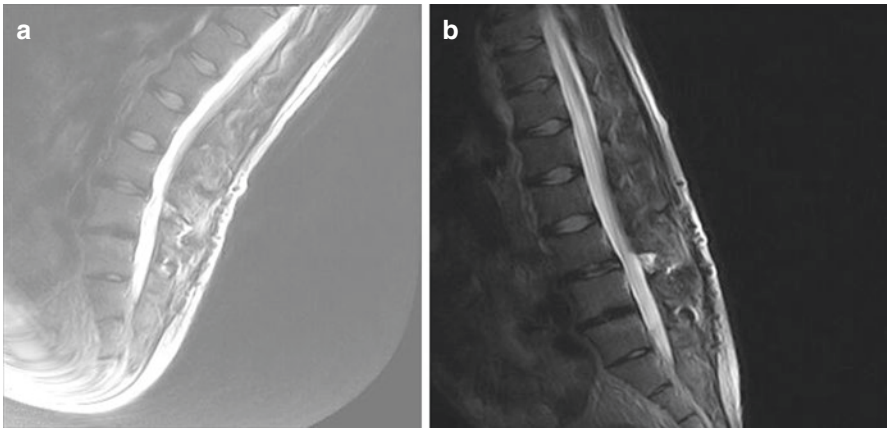

Fig. 35.5 Small extruded disc at L4–L5 levels. (**a**) Patient in lumbosacral extension position MR imaging. (**b**) Patient in lumbosacral flexion position MR imaging

unable to stand or sit for prolonged periods of time and is also not currently cost efficient.

Extremity MRI: this type of scanner has been in use since the 1990s to image peripheral body parts such as the knee and ankle. Extremity MRI scanners can have field strengths of up to 1.5T. These scanners were previously thought to be markedly inferior to equivalent conventional MRI systems; but recent studies have shown them to be somewhat equivalent [43, 44]. However, the increased cost associated with these scanners and their limited use in patient groups has meant that they are not in widespread use.

Dynamic MRI

Certain musculoskeletal disorders can be exacerbated and appear more conspicuous on patient movement. Examples include positional-related sciatica (Fig. 35.5), pelvic floor pathology, and hernias. Dynamic MRI is already in use for cardiac studies, but it remains in the early stages in musculoskeletal radiology due to the wide variability in imaging parameters and postprocessing techniques used [45, 46].

Diffusion-Weighted Imaging (DWI)

DWI imaging is an advanced MRI imaging technique that derives its image contrast from differences in water molecule movement/diffusion between tissues. DWI provides qualitative and quantitative information that reflects cellular level changes and provides insights into tumor cellularity and cell membrane integrity. In general, malignant tumors have greater cellularity than benign tumors, and infectious and inflammatory processes have increased cellular membrane permeability compared

to normal tissues. DWI is used in conjunction with an apparent diffusion coefficient (ADC) map, which acts as a reference level. Highly cellular lesions and lesions with increased membrane permeability will demonstrate high signal intensity on DWI and corresponding low signal intensity on the ADC imaging map [47].

The use of DWI is already well established in neuroradiology. However, its use in musculoskeletal and other areas of radiology is rapidly increasing.

Studies have demonstrated DWI to be more effective in detecting metastatic osseous lesions when compared to scintigraphy and positron emission tomography [48]. Diffusion-weighted whole-body imaging with background body signal suppression (DWIBS) has been shown to be of excellent tool when assessing and monitoring treatment response to solid tumors such as lymphoma in young patients [49]. This not only has the advantage of no radiation dose but also negates the use of radiopharmaceuticals, which can be in short supply.

DWI has also proven to be as or more effective than short tau inversion recovery (STIR)-based sequences in the detection of metastatic lesions in prostate and breast cancer, as well as in myeloma [50].

There are limitations to DWI, including its difficulty to differentiate between malignant and infectious processes, as well as malignant soft tissue tumors and soft tissue infection using just DWI due to overlapping imaging features [51]. Despite these limitations, DWI is a very valuable tool in modern-day musculoskeletal imaging.

Chemical Shift Imaging (CSI)

CSI is being more commonly used in musculoskeletal radiology to assess for bone marrow pathology [52, 53].

Bone marrow is of vital importance in hematopoiesis. The bone marrow broadly contains yellow (fatty) and red (hematopoietic) marrow. At birth, almost the whole skeleton is occupied by red marrow, which, as one grows older, converts to yellow marrow in a predictable fashion from distal appendicular skeleton to the axial skeleton in a bilateral and symmetrical manner. Bone marrow in the vertebral column is typically the last to convert to yellow, inactive marrow. Reconversion from yellow to red marrow can occur in response to increased hematopoietic demand. This increased demand can be secondary to several factors such as but not limited to anemia, obesity, chemotherapy, malignant infiltration, and hematological disorders.

The resonant frequencies of protons in different tissues are different. This shift in resonant frequency is known as a chemical shift. There is only a small difference between the resonant frequencies of protons in fat and water, and this is what is utilized in CSI. In CSI one acquisition four images will be produced:

1. In-phase images (similar to nonfat suppressed images)
2. Out-of-phase images: typical chemical shift imaging
3. Water-only images: similar to fat-suppressed images
4. Fat-only images: water-suppressed signal intensity images

When a voxel contains both fat and water, there is an additive effect on the signal in the in-phase images with loss of signal on the out-of-phase images. Processes that replace the fat will be seen to have a greater quantity of water within the voxel and therefore have no significant loss of signal on the out-of-phase imaging compared with the in-phase imaging. Signal loss can be assessed both qualitatively and quantitatively using the signal intensity ratio (SIR). Signal loss of greater than 20% on out-of-phase imaging is suggestive of benign processes, while a signal loss of less than 20% on out-of-phase imaging is suggestive of a malignant process [52].

CSI imaging can therefore differentiate between malignant and benign processes such as benign osteoporotic fractures versus malignant pathological fractures.

MR Spectroscopy (MRS)

MRS works on a similar basis to CSI. By detecting the signal of water, fat, and other metabolites, MRS can give metabolic information with regard to the lesion and help with characterization and assessment of treatment [54, 55].

The two main applications for which MRS is being trialed in the musculoskeletal system are for the assessment of musculoskeletal tumors and evaluation of muscle physiology and disease [54].

MRS can detect choline, which is an established marker of malignancy. Choline-containing compounds are part of the cell membrane make-up, and when there is damage to the cell membrane or increased cell turnover, choline levels increase. MRS can detect these raised choline levels to identify malignancy and also monitor changes to assess treatment response [56].

MRS has also been used to detect creatinine levels when assessing patients with potential myopathies, especially inflammatory in nature where standard MRI has shown no abnormalities. This is currently in its initial stages, but it is known that there are substantial differences in creatinine metabolism of muscles in patients with chronic myopathies when compared with healthy patients [55, 56].

Further research is still needed to determine the validity of MRS in musculoskeletal applications.

MR Elastography (MRE)

MRE is an emerging imaging technique for noninvasive evaluation of the biomechanical properties of musculoskeletal soft tissues. MRE is already commonly used in the detection and staging of liver fibrosis [57].

MRE works on a similar concept to sonoelastography, where changes in soft-tissue stiffness, for example, increased stiffness in neuromuscular disease and decreased stiffness in myositis, can be used to differentiate these tissues from normal soft tissues [55].

In MRE, a mechanical wave is produced in the soft tissues and then a modified phase-contrast MR sequence is used to image the wave motion. Finally, an inversion algorithm is applied to convert the wave image into an elastogram.

MRE looks promising, but it remains in its initial stages and needs further research to understand its pitfalls and explore further uses within the musculoskeletal field.

MR Neurography (MRN)

MRN has shown rapid improvements over the past two decades. High resolution imaging using high field strength MRI has shown excellent anatomic capability to detect nerve anatomy and pathology [58].

MRN can directly assess nerve pathology by looking at nerve contour and signal and indirectly by looking for muscle denervation. Studies have shown MRN to be as sensitive and specific as electromyography in the detection of median and ulnar nerve entrapment. It can also be of use in piriformis syndrome, ischial tunnel syndrome and nerve compression from tumors [59].

Further developments in MRN will focus on diffusion-based nerve and muscle imaging and using nerve-specific MR contrast images. These ongoing developments will not only help us detect nerve-based pathology but will also broaden our understanding of peripheral neuromuscular disease [60].

Dynamic Perfusion Imaging (DPI)

DP MRI is a functional imaging technique where enhancement patterns of soft tissue tumors are evaluated following an intravenous injection of gadolinium. It is best known for its use in the assessment of breast lesions. DPI provides important information regarding the vascularization, perfusion, and capillary permeability of a tumor, and its use can therefore be expanded for musculoskeletal soft-tissue tumor evaluation [61].

The intravenous injection is monitored for approximately 5 min within a defined region of interest (ROI) appropriate for the size of the tumor. To evaluate perfusion, three ROIs of identical size are chosen: one at the site of early tumor enhancement, the second within an artery, and the third in a healthy muscle. Time-intensity curves are then generated [62] and fall into five categories:

- Type 1: No enhancement
- Type 2: Faint and gradual enhancement
- Type 3: Rapid early enhancement followed by a plateau
- Type 4: Rapid early enhancement followed by a washout phase
- Type 5: Rapid early enhancement followed by slower enhancement

Soft-tissue lesions that are benign tend to have types 1 and 2 time-intensity curves; both benign and malignant lesions can be seen with type 3 time-intensity curves. Type 4 time-intensity curves are most often associated with malignant soft tissue, while type 5 time-intensity curves are seen with posttreatment soft-tissue lesions and lesions with a large interstitial compartment such as a myxoid tumor.

There is also a growing recognition for DPI in the assessment of epiphyseal perfusion in cases of potential avascular necrosis in the setting of slipped upper femoral epiphysis [61].

Nuclear Medicine (NM) Advanced Imaging

Nuclear medicine is a functional imaging technique that has multiple established applications in musculoskeletal radiology. Imaging involves intravenous injection of a radiopharmaceutical, which emits gamma radiation. This radiation is emitted from the patient and is detected and displayed in either a two-dimensional (2D) or a 3D format. NM images use either single-photon emission (SPE) imaging with a gamma camera or positron-emission tomography (PET) using a PET scanner.

Early identification of musculoskeletal conditions leads to improved management and patient outcomes. The assessment of multiple tissues and functional processes are often necessary to study the complex pathology of musculoskeletal disorders. The role of multimodality molecular imaging has become more important. Advances in NM imaging are therefore primarily focused on hybrid imaging where combining NM imaging with CT or MRI helps improve localized tracer uptake.

Single-Photon Emission Computed Tomography CT (SPECT-CT)

SPECT-CT is the fusion of SPECT imaging with simultaneous low-dose non-contrast CT to improve the localization of tracer uptake. It is commonly used in indeterminate cases of bone lesions on whole-body planar imaging where the concern is for metastases. SPECT-CT results in increased diagnostic confidence compared to planar scintigraphy when differentiating malignant from benign bone lesions. Whole-body SPECT-CT has demonstrated significantly higher sensitivity than targeted SPECT-CT to detect bone and extra-axial metastases [63], though in practical terms this may be difficult to apply due to time constraints.

SPECT-CT also has value in localizing the site of greatest tracer uptake in degenerative joint disease with a special focus on the ankle and foot to guide intra-articular joint injections and improving the clinical success of the procedure (Fig. 35.6). A study looking into this area demonstrated a high clinical success rate for SPECT-CT-guided joint injections [64].

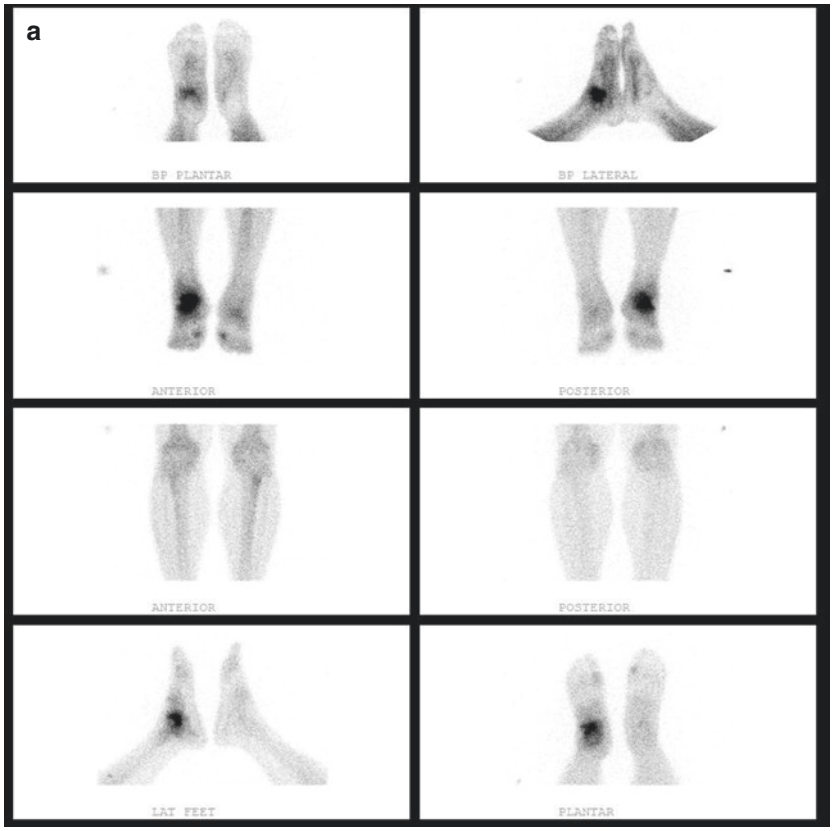

Fig. 35.6 Sinus tarsi syndrome. (**a**) Planar spot images from blood pool and delayed phase imaging of both lower legs showing increased tracer uptake at the right ankle. (**b**) SPECT-CT images with additional volume-rendered image demonstrate tracer uptake localizing to the subtalar joint and sinus tarsi. (Image courtesy of Dr. Fahmid U. Chowdhury, Consultant in Radiology and Nuclear Medicine)

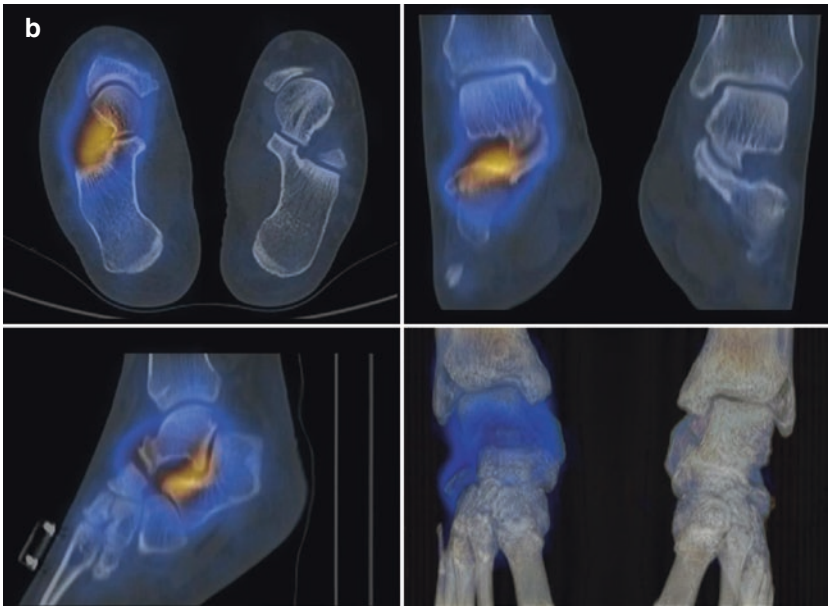

Fig. 35.6 (continued)

PET-CT

Whole-body PET-CT imaging is often performed for oncological purposes, and the presence or absence of metastasis is of significant concern as it can affect the staging of a patient's malignancy.

The most commonly used tracer in PET-CT is fluorine-18 (F-18) fluorodeoxyglucose (FDG), which is a glucose analog.

FDG uptake is sensitive but not specific to metastatic/malignant disease. Numerous benign sources of FDG uptake can occur in the musculoskeletal system and be mistaken for aggressive or malignant processes such as fractures, degenerative disc disease, osteophytes, postoperative appearances, and muscle overuse [65]. Confusion and misinterpretation can occur if the bone is not evaluated on the CT portion of the PET-CT (Fig. 35.7).

FDG PET has also been considered for the early diagnosis of musculoskeletal infections and may also play an important role in the evaluation and follow-up of chronic osteomyelitis and spinal infection [66]. However, FDG-PET-CT results in patients with suspected prosthetic joint infection are controversial, and as such, combined indium-111-labeled leukocyte and technetium-99m-sulfur colloid marrow scintigraphy is still the gold standard.

FDG-PET-CT can be used in the initial staging of bone and soft-tissue sarcomas and also in the follow-up stages to assess response to treatment. Tumors can be graded based on radionuclide uptake. High-grade tumors generally demonstrate

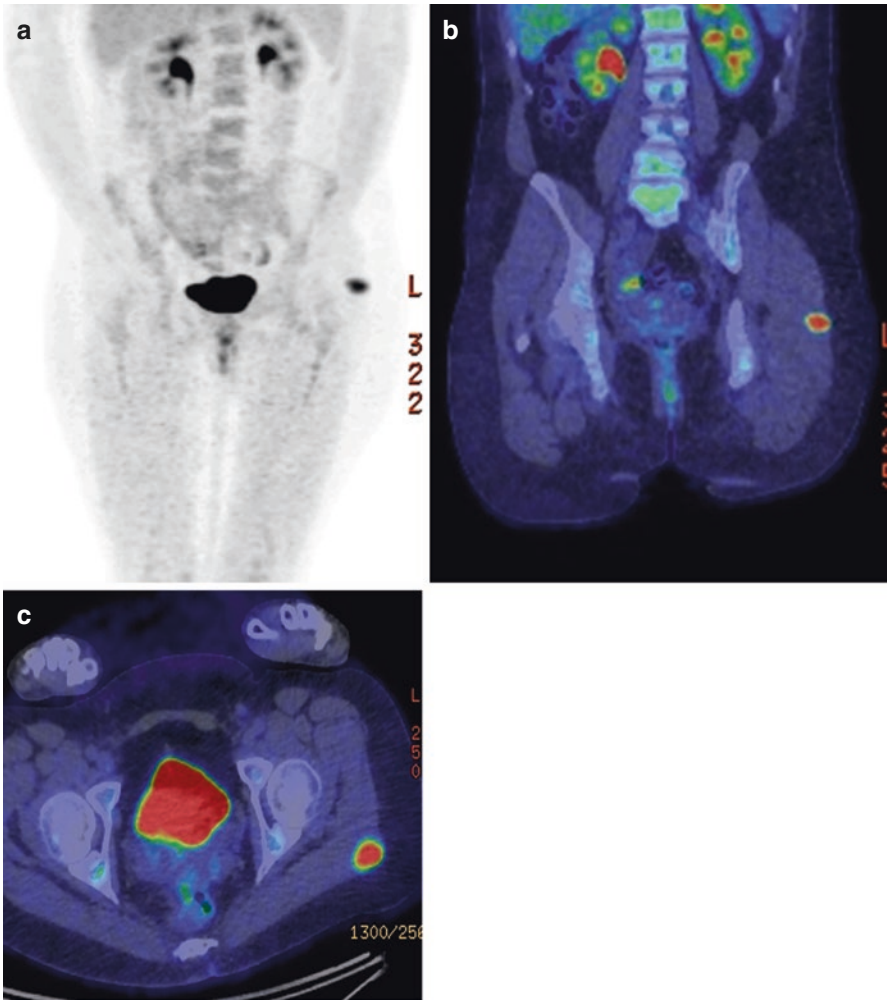

Fig. 35.7 Twenty-five-year-old female with left gluteal lesion. MRI and ultrasound (not shown) demonstrated a lesion concerning for a peripheral nerve sheath tumor. Biopsy has proven malignant peripheral nerve sheath tumor (MPNST). First presentation of NF1 in this patient. (**a**) Attenuation-corrected PET image in coronal plane shows solitary focus of ^{18}F FDG tracer uptake in the left gluteal lesion. (**b**) Fused ^{18}F FDG PET/CT image in the coronal plane showing uptake in the left gluteal lesion. (**c**) Fused ^{18}F FDG PET/CT image in axial plane showing uptake in the left gluteal lesion

increased uptake, as measured by the standardized uptake value (SUVmax) compared, and this information can be used to guide biopsy location. A recent study showed PET-CT to be of value in high-grade bone and soft-tissue sarcoma staging, where it resulted in the upstaging of cancer in approximately 12% of patients who had also undergone conventional CT or MRI imaging [67].

PET-MRI

PET MRI (Fig. 35.8) is an emerging imaging modality that aims to combine high-resolution MRI with simultaneous molecular information from PET to analyze the multifaceted processes involved in numerous musculoskeletal disorders such as osteoarthritis, rheumatoid arthritis, metabolic bone diseases, and neuropathic peripheral pain [68].

PET is the only molecular imaging modality with high sensitivity to several processes that precede structural and biochemical changes at the tissue level. PET-MRI gives the additional advantage of producing good soft-tissue resolution and reduced ionizing radiation compared to PET-CT; this is of particular importance in the pediatric setting [68, 69].

There are huge complexities when combining the hardware for multiple imaging modalities, and this holds true for PET-MRI, especially when considering which MR sequences to employ that will combine well with PET images, and further refinement of these MRI techniques is needed to maximize its clinical usefulness.

Despite the aforementioned difficulties, PET-MRI offers the potential to obtain metabolic, morphologic, and functional data from all tissues to aid the identification and follow-up of musculoskeletal disorders.

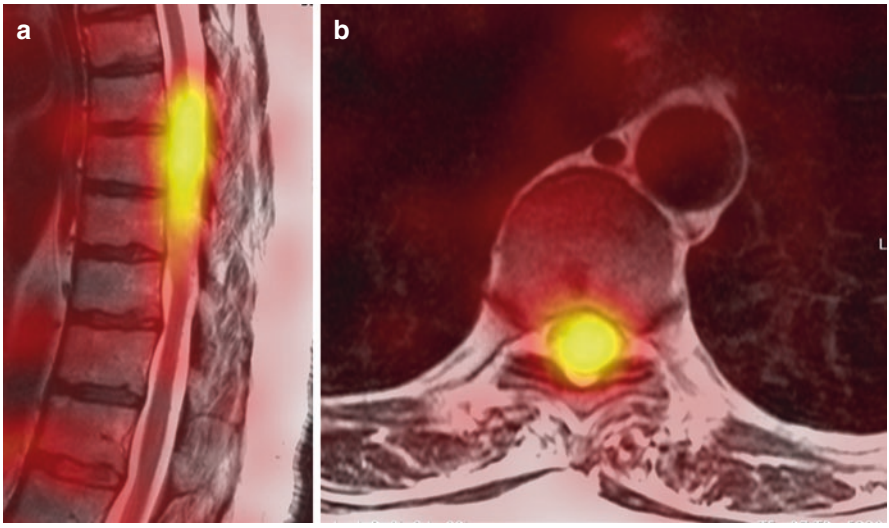

Fig. 35.8 PET-MRI showing an intradural intramedullary expansile soft-tissue mass, which demonstrates an increased metabolic activity. Histology was in keeping with an ependymoma. (**a**) Sagittal T2-weighted fused ^{18}F FDG PET-MRI image. (**b**) Axial T2-weighted fused ^{18}F FDG PET-MRI image

Conclusion

We hope that by completing this chapter you have become more familiar with the new and ongoing developments in the musculoskeletal imaging field and will keep up to date with changes in these imaging techniques as with more research and utilization, they may become standard practice in the assessment of musculoskeletal problems in the near future.

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Musculoskeletal Overuse Injuries of Lower Limb in Military

36

Edward Sellon, Duncan Goodall, Rob Barker-Davies, Alexandra Crick, and Alex Ashburner

Introduction

Overuse injuries are generally defined as those arising from long-term energy exchanges resulting in cumulative microtrauma over time [1]. There is a high incidence of overuse injury in military personnel, particularly among infantry recruits, due to accelerated increases in physical activity volume and load carriage intensity. The need to meet fixed training requirements, such as deploying on a field exercise at set times, can pressure rest and recovery requirements for some individuals. This can result in loss of training time, reduced performance, and permanent discharge [2]. Musculoskeletal injuries (MSKI) lie behind around 60% of occupational downgradings in the British military and a similar proportion of medical discharges [3]. It is thought that overuse injuries account for approximately 65% of these [4]. Similar MSKI incidence is reported in militaries worldwide, and parallels are seen in civilian runners and professional dancers [5]. When considering the burden of lower limb overuse injuries, it is worth considering incidence and recovery times together in order to understand the relative accumulated rehabilitation time. This gives a better indication of the occupational burden of the injury. By this measure, stress fractures and medial tibial stress syndrome have the greatest burden, followed by tendinopathies of the lower limb

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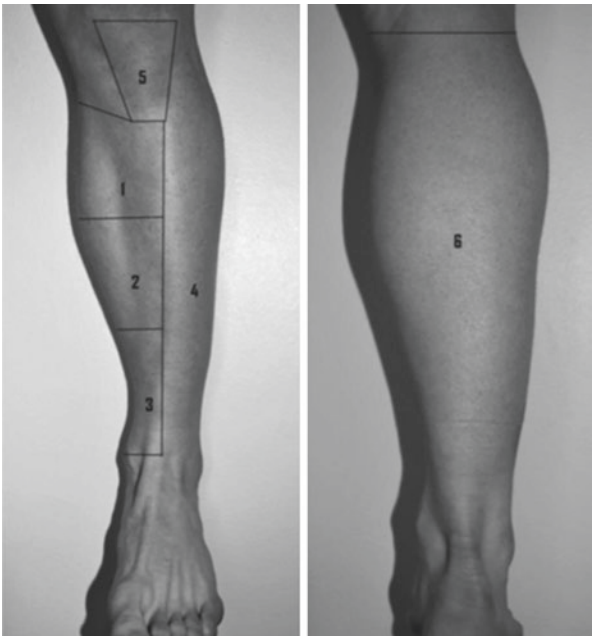

Fig. 36.1 Clinical photograph showing surface markings on the lower leg to help delineate and communicate the area of pain in exercise-induced leg pain (EILP). Zones 1–6

[5]. This chapter will concentrate on areas such as these, which confer the greatest occupational impact, and also on other less common causes of exercise-induced leg pain (EILP), such as chronic exertional compartment syndrome and popliteal artery entrapment syndrome, that are relatively overrepresented in the military population. These are usually triaged on the basis of symptom location (see Fig. 36.1) and severity.

Lower Limb Tendinopathy

Tendinopathies are characterized by activity-related pain and dysfunction. In the military population, this is usually due to excessive loading and chronic overuse. Patellar and Achilles tendinopathies are among the conditions associated with greatest accumulated rehabilitation time in the British military [5]. The biomechanics are multifactorial but predominantly associated with repetitive microtrauma leading to a number of degenerative changes [6]. The more chronic cases result in tenocyte and collagen necrosis and microtears [7]. Imaging has an important role in the diagnosis and can be used to guide percutaneous intervention [8].

Achilles Tendinopathy

Achilles tendinopathy resulting from unaccustomed or excessive mechanical loading is typically located either in the midportion of the tendon where it is relatively hypovascular or at the distal insertion [9]. Achilles tendinopathy is particularly common in military populations with incidence over a 6-month period of initial training in UK recruits around 1.4% [10]. Many of these cases respond well to an initial period of reduced training load with mean time spent in rehabilitation between 10 and 20 days [10]. Unfortunately, these cases often present late from fear of being re-coursed or "back-squadded" and separated from their cohort [6, 11]. The continued application of overload to these tendons can then drive further degenerative changes [6, 11]. Tendon compression in the military setting from poorly fitting boots can also perpetuate the problem [12]. Without addressing these factors initially, the tendon is at risk of developing further pathological changes irrespective of treatments applied.

A summary of the UK military treatment guideline is included in Table 36.1. The majority of cases can be managed in primary care by a general practitioner and a physiotherapist, with some facilities benefiting from the additional resource of exercise rehabilitation instructors. Diagnosis can be made clinically based on an insidious onset of symptoms with an inflammatory pattern. The main finding on examination is usually well-localized pain to the affected tendon, which is elicited by loading and tenderness to palpation. Optimal loading through a pain-guided progressive loading program is the mainstay of treatment. A progressive sequence of loading tests is used to gauge symptoms and provides a helpful starting point to

Table 36.1 Defence lower limb tendinopathy guideline summary

History	Examination	Common sites	Initial management
Morning stiffness Warm-up pain Pain after immobility	Pain over tendon Provoked by loading	Achilles Patella Gluteals Hamstrings Tibialis posterior Peroneal	Exclude other causes Manage predisposing conditions (DM, obesity) Do active rest Consider short course of NSAID therapy
Progressive loading (keep pain <4/10) • Isometrics • Isotonics • Eccentrics/heavy slow resistance • Plyometrics (Do not do phases 3 or 4 on concurrent days. Add a phase 1 or 2 day in-between to optimize recovery)		Take-home messages • Do not jump in with eccentric/heavy slow resistance • If not improving at 6 weeks, reconsider diagnosis and management • If not improving at 12 weeks, get an US scan to confirm diagnosis and consider shockwave therapy	

Table redrawn showing the UK Defence Lower Limb Tendinopathy Guideline Summary (2019)
PCRF Primary Care Rehabilitation Facility, *RRU* Regional Rehabilitation Unit

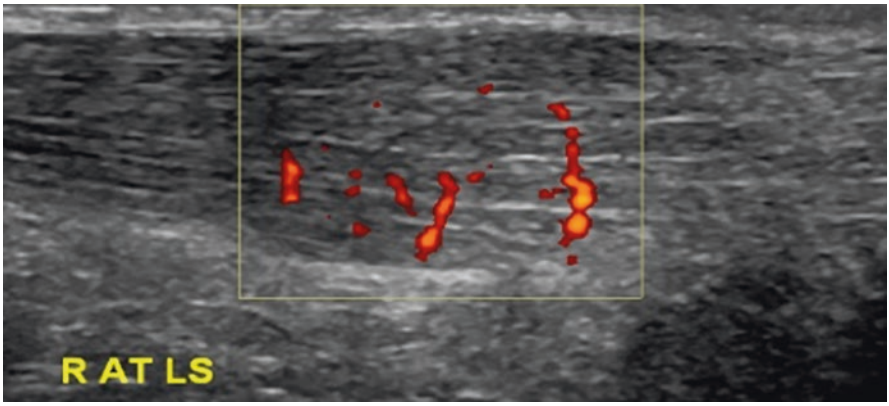

Fig. 36.2 Longitudinal ultrasound image of the Achilles tendon (proximal on the left, distal to the right). There is severe, chronic midportion Achilles tendinopathy with fusiform thickening, hypoechoic fibrillar echotexture, and neovascularity. This represents grade 4 on the modified Ohberg scale with at least four vessels running through the substance of the tendon [17]

guide the optimal tendon load for prescription [13]. A typical sequence for the Achilles tendon would be to initially examine a double-leg calf raise, followed by a single-leg calf raise, then progressing to an initially low then high hop. Pain is used to titrate load, and patients are told to avoid exercises into a pain score above 4/10 [14]. A sequence of phases should be worked through (see Fig. 36.2) and with key points being that eccentric loading or heavy slow resistance exercises sit at phase 3 and are not used as the sole or initial treatment. The higher load exercises are also microcycled through the week when introduced rather than practiced daily, with the lower level exercises from the preceding phases used to provide relative rest on the days in-between.

UK Defence has set a target of making a decision on treatment pathway after 6 weeks of initial load-based therapy in primary care. If improvements are not being made or a question remains over the diagnosis at this stage, referral to one of the Regional Rehabilitation Units (RRUs) is indicated. This will include a sport and exercise medicine consultant review with diagnostic ultrasound. Regional Rehabilitation Units may consider the use of adjunctive therapies such as extracorporeal shock wave therapy, alongside pain-guided progressive loading. A small number of refractory chronic cases may be considered for injectable therapy at tertiary level (Defence Medical Rehabilitation Centre), the options for which are discussed further below (Table 36.1).

Imaging

Ultrasound is the preferred imaging modality in the assessment of tendinopathy or a tear. This allows dynamic assessment and guided intervention when needed.

Ultrasound

Ultrasound assessment using a high-frequency probe with the patient prone allows ready assessment of the normal echogenic fibrillar pattern. Fusiform midportion or

insertional tendon thickening with a darker "hypoechogenic" appearance is the hallmark of tendinopathy (Fig. 36.2). This is due to the tightly packed collagen becoming separated by increased ground substance in a maladaptive response to training. While a healthy response to training increases the number of small proteoglycans, an increase in the number of large proteoglycans is associated with an expansion in extracellular matrix [7]. This explains why thickening is not a feature of healthy tendon adaptation [15]. Tendons are usually relatively avascular and devoid of nerve fibers [16]. Power Doppler imaging can be used to demonstrate tendon neovascularity emanating from the underlying Kager's fat in chronic disease. Dynamic assessment can be useful when there is suspicion of a tear. Paratenonitis is also associated with hypoechoic thickening and Doppler activity and may occur in isolation.

MR Imaging

Fusiform swelling of the tendon is seen with focal or diffuse increases in T1 and T2 signals. There may also be edema of the underlying Kager's fat pad. Insertional tendinopathy may be associated with retrocalcaneal bursitis and reactive bone marrow edema in the posterior calcaneus. Paratenonitis produces a posterior rim of high T2 or STIR signal around the Achilles and is best assessed on axial sequences.

Patellar Tendinopathy

Patellar tendinopathy is also common in military populations. The approach to clinical assessment and management is similar to that for Achilles tendinopathy outlined above. Pain is usually well located in the inferior patella pole on loaded examination and palpation. The area of pathological tendon frequently develops initially in the deep proximal fibers. In order to elicit this clinically, it may be necessary to tilt the patella when examining by depressing the upper pole with one hand so that the lower pole tilts anteriorly, allowing palpation of these deep fibers. The main alternative diagnosis to exclude or to consider the coexistence of is patellofemoral pain syndrome (PFPS). It is therefore important to question the patient as to their presenting pain (or the main pain) in the process of examination. PFPS also occurs insidiously with an inflammatory type pattern. The pain tends to be located around or posterior to the patella, associated with knee flexion beyond 60° and can cause knee joint effusion [18]. PFPS is also associated with dynamic knee valgus, as can be demonstrated on a single-leg squat, which responds to functional stabilization training [19]. The initial treatment for patellar tendinopathy, which may include a single-leg isometric hold in a squat position, may therefore coincidentally provide a degree of functional stability and be helpful for coexistent PFPS (Table 36.1). However, if the diagnosis of PFPS is missed in the context of treating coexistent patellar tendinopathy, progression through the more demanding phases of loading will be hampered.

Typical progression of pain-guided progressive loading would be phase 1 isometric holds consisting of a double-leg or a single-leg squat position limited to 90° knee flexion. Phase 2 body weight double-leg squats progressed to single-leg squats, followed by

phase 3 of either eccentric or weighted (heavy slow resistance squats) squats with a barbell [20]. Finally, plyometric activities and return to running are introduced at phase 4. As for Achilles tendinopathy, the higher level activities are microcycled with lower level activities on days in-between, and exercises are titrated to keep pain scores below 4/10.

Imaging

The structural changes associated with patellar tendinopathy can be demonstrated using ultrasound or MR imaging. Both modalities are associated with excellent accuracy (ultrasound, 0.63–0.83; MRI, 0.68–0.70) but varying sensitivity (ultrasound, 0.68–0.87; MRI, 0.50–0.57) [21]. Ultrasound, however, has become the preferred technique over recent years due to rapid technological improvements.

Ultrasound

The superficial anatomy allows a high-resolution view of the fibrillar tendon structure with ultrasound and the assessment of neovascularization using power Doppler. The patient is examined supine with the knee supported in slight flexion to extend the tendon. A high-frequency probe (around 15 MHz) is used with a light probe pressure to examine the full length and width of the tendon. The knee is then extended in order to assess for neovascularization using power Doppler. Tendon relaxation enables potential flow within the small vessels.

The structural changes seen in patellar tendinopathy are usually focal, involving the proximal central or medial deep surface fibers, near the attachment with the patella. As with the Achilles, early changes include tendon thickening and loss of the normal fibrillary pattern with hypoechogenicity (Fig. 36.3). As with the Achilles

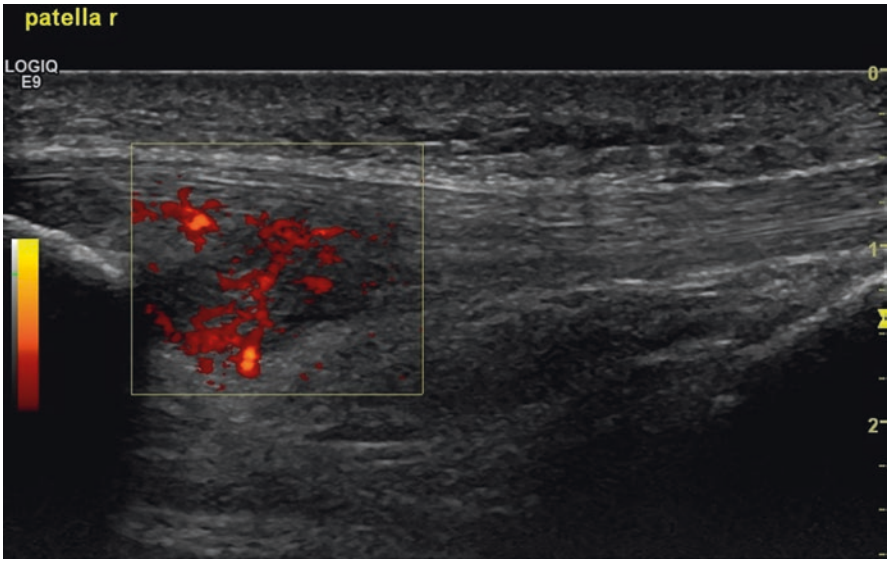

Fig. 36.3 Longitudinal ultrasound image of the patella tendon (proximal on the left, distal to the right)

tendon, there is thickening of the tendon, in this case of the proximal aspect. This must be distinguished from the presence of partial tears. Tendon ruptures are uncommon in the context of overuse tendinosis. Patellar paratenonitis is also uncommon, more typically seen in the Achilles. Patellar tendinopathy can occasionally be diffuse, involving the entire tendon, but this suggests an underlying metabolic cause (e.g., obesity, type 2 diabetes, mellitus, dyslipidemia, or hypercholesterolemia) rather than simple overuse.

Doppler activity as a marker for vascular ingrowth in chronic tendinopathy remains the subject of ongoing research with recent literature revisiting the idea that there may be an inflammatory component of the chronic injury-repair response [22]. Inflammatory mediators may be responsible for neural and vascular ingrowth and have been implicated as a potential source of pain [23]. Most radiologists consider the presence of Doppler signal on ultrasound to represent active and clinically significant tendinopathy. However, Doppler activity can also be seen in asymptomatic athletes [24], particularly after strenuous exercise [22, 25, 26].

There is severe, chronic patellar tendinopathy (Fig. 36.3) with proximal tendon thickening, hypoechoic deep fiber echotexture, and neovascularity (modified Ohberg grade 4) of the tendon and underlying Hoffa's fat.

MR Imaging

MR imaging of patellar tendinopathy is characterized by focal tendon thickening and intermediate to high signal change on all sequences. It can also demonstrate focal high T2 signal areas of cystic degeneration or focal tearing. Healthy tendons should appear uniformly dark on all sequences. The changes can be subtle if only a small area of tendon is involved, but edematous change in the adjacent soft tissues can be a useful indicator.

Elastography

While the changes outlined above are generally helpful in diagnosing tendinopathy, they are of limited value as an outcome measure. Ultrasound appearances in terms of either maximum tendon diameter or neovascularity do not generally correlate well with clinical outcomes [24, 25]. It may be that tendon thickness and neovascularity take longer to change in response to treatment as correlation has been demonstrated with functional outcome at a 3- to 5-year follow-up [26]. Changes in hypoechogenicity have been shown to occur much earlier, within 6 months, and to be significantly related to pain with a functional test [27]. However, measuring hypoechoicity is more subjective and consequently less reliable than tendon thickness or neovascularity [28]. Hypoechogenicity likely represents a reduced signal from type 1 collagen and therefore, if reliably measurable, would be a candidate for a gold standard outcome in the short to medium term (6 weeks to 6 months). Ultrasound tissue characterization (UTC) [29–32] and, more recently, shear wave elastography (SWE) have been proposed as more sensitive image-related outcome measures [33, 34].

SWE is a fairly recent development in the field of ultrasound and can be used to assess the stiffness (or rigidity) of soft tissues. A high-intensity pulse is

transmitted, producing shear waves that pass through a tissue causing deformation. The degree of deformation caused by the pulse is tracked with further low-intensity pulses, determining the shear velocity, and elastic modulus (stiffness) of the tissue. The elastic modulus of the Achilles tendon has been shown to decline with tendinopathy. Studies have shown that SWE is more sensitive than ultrasound alone in detecting early Achilles tendinopathy. This is less certain for other tendons, such as the patella tendon, but it presents a potential quantitative assessment tool for examining the mechanical integrity of tendons. This may be of interest in the military environment as a screening tool for tendinopathy in soldiers during basic training.

Tendon Intervention

Corticosteroid injections (CSI) for tendinopathy have reduced in popularity over the last decade as evidence has grown of their temporary effects on pain and function and the possible detrimental long-term effects on tendon strength [35, 36]. They can be thought of as long-acting anesthetics only, to reduce symptoms in the short term to allow better compliance with physiotherapy, but caution is advised particularly in the presence of tendon damage. A large systematic review demonstrated short-term benefits, high relapse rate, and risk of tendon rupture [37]. Where there is ultrasound evidence of tendinosis, a peritendinous CSI should be accompanied by clear information about the risk of rupture and the importance of a 2-week rest post procedure. In the context of elite sport, where there can be pressure to return the athlete to the field of play, there may be advantages in the short term to such an approach. In the military context, CSI are generally not used unless the target is the paratenon in the presence of a normal tendon. The tendinosis is usually chronic, and a longer term therapeutic solution is preferred.

Dry needling (also known as fenestration and percutaneous tenotomy) has better long-term effectiveness in the treatment of focal tendinopathy. It is used in both the patella and Achilles tendons to promote bleeding in order to stimulate a healing inflammatory cascade, when conservative measures have failed. The entheses are the areas best treated, particularly the proximal patella tendon. It involves passing a needle multiple times through the area of damaged tendon and is sometimes supplemented by an injection of autologous blood or platelet-rich plasma (PRP). It inevitably causes short-term tendon weakening, and rest should be advised for 2 weeks post procedure with relative rest for a further 2–4 weeks.

High-volume image-guided injections (HVIGI) have grown in popularity since they were initially championed [8]. This involves an ultrasound-guided injection of 10 ml bupivacaine and up to 40 ml of normal saline deep to the patella or Achilles tendon in order to strip off the vascular and neural ingrowth and/or any fibrous adhesions. Whether or not disruption of these nerve fibers can lead to reduced pain and better outcome in the treatment of tendinopathy is a focus of ongoing research for both Achilles and patellar tendinopathies [13].

Medial Tibial Stress Syndrome

Medial tibial stress syndrome (MTSS) is a term used to encompass diagnoses such as shin splints, periosteal inflammation, and periostitis. When assessing a service person with MTSS, the clinician should always have a high index of suspicion for potential stress fractures. As with all incidents of EILP within the military, MTSS can be prevalent with service personnel involved in weighted marches. This is common across the tri-services but is particularly common in the Royal Marines and the British Army.

The importance of understanding an individual's history and training is necessary with regard to considering load management. For example, a "phase 1" recruit during initial basic training may present as a result of a sharp increase or spike in their exposure to load, depending on their physical condition and preparation prior to joining the military. The same is true of service personnel who have been in the military for a longer period when increasing the frequency, volume, or intensity of training in preparation for a course, training exercise, or deployment.

Signs and Symptoms

Pain is usually reported in the mid to lower part of the tibia. Symptoms are mechanical in nature, and pain is typically brought on by impact, such as with running, and settles with rest, with little neurogenic or vascular presentation.

Management

Pain on palpation or percussion of the tibia will usually elicit symptoms. MR imaging is more sensitive than radiographs in demonstrating medullary bone stress or a stress fracture. Conservative management consists of assessing the individual's lower limb biomechanics and evaluating their training program. Assessing the service person's stance, gait, and running (if appropriate) will aid in assessing lower limb biomechanics. A navicular drop, overpronated foot type, excessively high or flat arches, tibia varum, and genu valgum or varum are common observations that may contribute to MTSS. Podiatry input may well be worth considering if an overriding biomechanical component to the presentation is suspected. However, there are frequent examples of what would be considered "suboptimal" biomechanics in service persons who are completely asymptomatic. Therefore, building a detailed clinical picture of whether or not the soldier has appropriate levels of physical conditioning, alongside a balanced training program, is necessary when ascertaining the appropriate management. Assessing proximal control at the hip, single-leg control and functional tasks can also be beneficial in developing the clinical picture.

Depending on the extent of the injury, a period of off-loading may be necessary. Cross-training with non-weight-bearing exercise is useful to maintain

cardiovascular fitness. Utilization of a bike or rowing ergometer and swimming should not elicit symptoms.

Graded return to impact, incorporating education and training program design to avoid a boom-and-bust approach to rehabilitation, is paramount. A graded return to impact program, including the completion of strength milestones to ensure relative strength to body weight and limb symmetry, should be completed prior to a graded walk to run program. Within a military context, this is necessary as the soldier will be returning to load marches that require carrying body weight in addition to a Bergen, webbing, helmet, and rifle. An antigravity treadmill can be a good adjunct when gradually returning to running, allowing slow progressive loading.

Imaging

Radiographs have a poor sensitivity for detecting MTSS or a stress fracture, particularly on initial examination. However, they remain useful if only to show overt fractures and to help exclude other conditions such as osteomyelitis or osteosarcoma [38]. Radiographic signs include endosteal or periosteal callus formation, periosteal reaction, or an incomplete transverse fracture line through one cortex.

MR imaging has surpassed bone scintigraphy as the imaging tool for bone stress reactions and fractures with 90% sensitivity and nearly 100% specificity in the tibia [39]. Additionally, unlike other modalities, it can demonstrate edema within 3 days of symptom onset (Brewer and Gregory). The other advantage of MR imaging is that it can be used to predict the time of healing using the Arendt and Griffiths classification (see Table 36.2) [40]. Signs of bone stress on MR imaging include periosteal or adjacent soft-tissue edema, marrow edema, and cortical edema or fracture line.

Fractures with any displacement or with a fracture line that crosses >50% of the femoral neck width should be considered high risk of displacement. Additionally, stress fractures that involve the lateral femoral neck, patella, anterior cortex of the tibia, talus, navicular, fifth metatarsal, or hallux sesamoids should be considered high risk due to tension forces, with greater rates of displacement or delayed union or nonunion. It is therefore a combination of clinical symptoms, osseous location (high-risk or low-risk site), and imaging features on MR imaging that indicates prognosis and treatment.

Table 36.2 Table showing the Arendt and Griffiths classification of stress fractures [40]

Grade	XR	MR imaging	Treatment
Grade 1	Normal	Positive STIR image	3-week rest
Grade 2	Normal	Positive STIR and T2 image	3–6-week rest
Grade 3	Discrete line or periosteal reaction	Positive T1 and T2 but images without definite cortical break	12–16-week rest
Grade 4	Fracture or periosteal reaction	Positive T1 and T2 with fracture line	16+ weeks of rest

STIR short-time inversion recovery

Chronic Exertional Compartment Syndrome (CECS)

Unlike with acute compartment syndrome, which is usually associated with a traumatic event, chronic exertional compartment syndrome (CECS) manifests gradually over time.

Within the military setting, servicemen and women are required to march at very specific speeds and cadences, as a collective troop. This does not allow for the variance in soldier stature, size, or height. This is thought to be a contributing factor to the relatively high prevalence of CECS within the military. For example, a shorter individual will need to keep the same pace as a taller individual with a longer stride. This, coupled with the need to maintain a fast pace and resist the urge to break into a run, increases the likelihood of excessively stressing the anterior compartment muscles of the lower leg, from both a biomechanical and a loading perspective [41].

Signs and Symptoms

The patient will usually describe a crescendo pain, with descriptors such as "swelling," "tightness," or a "bursting" sensation. In turn, this leads to immediate cessation of activity as they can no longer continue [42]. It is also often accompanied by a foot slap or a lack of eccentric control of dorsiflexion on heel strike during ground contact. This can be accompanied by paresthesia or altered sensation down the lower limb and on the dorsum of the foot and the toes. Activity cessation will bring symptom relief [43].

Management

If CECS is thought likely, individuals will be put through a dynamic stress test (DST) with intramuscular compartment pressure measurement (ICPM) catheters. This involves taking the individual through an incremental march over three 5-min stages carrying 15 kg of weight in a Bergen at a speed of 6.5 km/h on a slight incline to replicate a forced march scenario. If the DST is positive and replicates the patient's symptoms, surgical intervention through a fasciotomy is considered. Within the British military, 46% of people who had a fasciotomy went on to improve their occupational medical grading, with secondary outcome measures of symptom severity also improving [44].

Fasciotomy is undertaken under general anesthesia under tourniquet control. Regional anesthesia in the form of ultrasound-guided local anesthetic block of the common peroneal or sciatic nerve may additionally be undertaken to provide early postprocedural analgesia. Usually, a single longitudinal incision is made through the skin and subcutaneous fat overlying the anterior compartment. The deep fascia is then incised proximally, confirming exposure of the anterior compartment muscles before extending the incision distally to the extensor retinaculum (Fig. 36.4). Shorter skin incisions and endoscopic procedures have been described, but these

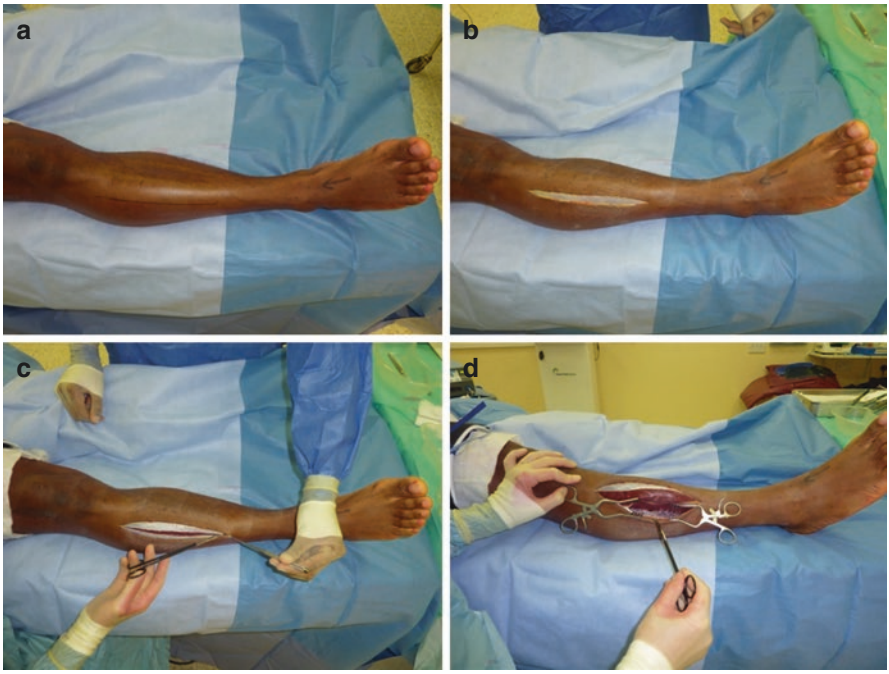

Fig. 36.4 Clinical photographs showing (**a**) skin marking, (**b**) incision exposing the deep fascia, (**c**) incision of the deep fascia, (**d**) forceps indicating the septum between the anterior and lateral compartment

should not compromise the completion of the incision within the deep fascia over the anterior compartment or inadvertent injury to the superficial peroneal nerve adjacent to the intercompartmental fascia. Hemostasis is achieved following deflation of the tourniquet. A wound drain may be inserted. The skin incision is closed. Some form of wound dressing is required during healing for at least 2 weeks. Elevation of the leg to minimize edema when at rest is important. Protected weight-bearing during this period is also advised.

Early postoperative intervention should focus on pain and swelling management. Scar management is integral in managing postoperative paresthesia or hypersensitivity in combination with encouraging a healthy mobile scar. Patients should be educated and encouraged to do this independently as part of a daily routine. If the pain is well tolerated, rehabilitation can focus on progressively increasing standing tolerance and early exercises to restore full ankle and knee ranges of movement.

If the wounds have healed and there are no signs of infection, patients can start hydrotherapy to encourage gentle load bearing on standing and also swimming for cross-training. Exercise bikes are also useful in allowing a graded return to load bearing while preventing excessive end-of-range ankle movement. Once patients are able to walk reciprocally and independently without pain, a graded return to impact can begin on the treadmill (ideally antigravity). Static weight-bearing

gastrocnemius and soleus stretches can be included in the program. Once the pain has settled during early return to impact, patients can progress through mid and late stages of rehabilitation, as appropriate.

Imaging

MR imaging is often used to help exclude medial tibial stress syndrome or a stress fracture. The same is true for the lumbar spine if there is suspected centrally driven radicular pain or radiculopathy. There is no established role for MR imaging in diagnosing CECS. There is, however, a growing body of evidence, supporting the use of MR imaging with in-scanner or out-of-scanner exercise protocols to elicit diagnostic T2 signal ratios, but the clinical significance of this is yet to be fully established [45, 46]. Some authors have advocated the use of ultrasound to identify anterior CECS by measuring anterior compartment thickness [47]. While promising, these less-invasive, radiological techniques remain largely inconclusive. Occasionally, MR imaging or ultrasound may identify a defect in the deep fascia overlying the anterior compartment. This may have developed following previous blunt trauma. Muscle herniation through this defect on exertion may contribute to exercise-induced leg pain.

Popliteal Artery Entrapment Syndrome

Popliteal artery entrapment syndrome (PAES) refers to a group of conditions that are caused by the compression of the popliteal artery by adjacent musculoskeletal structures, sufficient to cause vascular and neurogenic symptoms (Table 36.3). Anatomic anomalies are usually found in the position of the popliteal artery (sometimes with the popliteal vein and tibial nerve) in relation to the medial head of gastrocnemius, popliteus, or fibrous bands. However, in the young military or sporting population, neurovascular compression may be acquired through muscular hypertrophy [44–46].

Table 36.3 Table showing popliteal artery entrapment syndrome classification (Popliteal Vascular Entrapment Forum) [45]

Type 1	The popliteal artery passes medially and then deep to the normal medial head of gastrocnemius
Type 2	The medial head of gastrocnemius inserts more laterally than usual. The popliteal artery descends normally but passes medially to the muscle
Type 3	The medial head of gastrocnemius has an accessory slip arising more laterally, which compresses the popliteal artery
Type 4	The popliteal artery is compressed by running deep to the popliteus muscle or by an anomalous fibrous band
Type 5	Primary popliteal vein entrapment
Type 6	Functional entrapment with no anatomical abnormality

Precise incidence is unknown in the military population, with a reported overall incidence range of 0.17–3.5% in the general population [47]. It is seen more commonly in men than women (ratio 9:1), with 80% presenting under the age of 30 years, in keeping with the military demographic.

Signs and Symptoms

Like with all of the other exercise-induced leg pain diagnoses, a thorough history and examination is crucial. Symptom duration can be over 24 months prior to diagnosis if the condition is not considered, and so this will affect the military personnel's ability to be deployed on duties. Symptoms can be unilateral or bilateral (30%) and classically present with claudication calf pain on exertion. Military personnel can also complain of a feeling of "tired legs" but may also report symptoms of paresthesia or numbness and later cold feet, pallor, and discoloration. Claudication symptoms resolve quickly on stopping the march or exercise.

On examination, there may be evidence of hypertrophy of the calf complex, and on active plantar flexion and passive dorsiflexion, there may be diminished foot pulses. There may also be evidence of popliteal bruit and an ankle brachial pressure index (ABPI) drop of >0.5 on stress test.

Imaging

Duplex ultrasound is usually the initial imaging strategy and can elicit popliteal artery compression. This can be done dynamically using an exercise protocol with repeated active plantarflexion and dorsiflexion. It should be understood that popliteal artery occlusion can be noted in asymptomatic individuals, with studies reporting a range of 7.1–80% [48, 49]. This again emphasizes the importance of the clinical workup prior to vascular review.

If US is abnormal, MR or direct angiography can be used to further delineate the underlying anatomical subgroup of PAES. If functional entrapment is suspected, US-guided injection of botulinum toxin type A (BTX-A) can be performed as an alternative to surgery, although the long-term outcomes are unknown [50].

Management

Given its rare presentation, this condition is best managed with a close interdisciplinary team working with the rehabilitation team and a vascular surgical team with experience of the condition. Other differential diagnoses considered during the clinical workup include other vascular, musculoskeletal, and neurological causes of leg pain. Imaging and investigations will be tailored to rule out such conditions.

If there is an anatomical variant noted from imaging, then surgical treatment is advocated. More commonly in the military population, a functional PAES is

diagnosed and then management decision also requires consideration of the occupational status of the individual and what their role is in the military. Botox injection may aid diagnosis in where the military personnel can have the injection to invoke atrophy of the muscle compartment and assess symptoms on exertion.

Superficial Peroneal Nerve Entrapment Syndrome

Superficial peroneal nerve entrapment syndrome (SPNES) typically occurs at the site where the nerve traverses the deep fascia within the distal leg passing from the lateral compartment proximally to become subcutaneous distally. There may be an associated defect within the deep fascia at this site, identified clinically or on imaging. This may develop spontaneously or following a specific injury, usually involving ankle inversion. The ankle inversion may itself have caused nerve injury due to undue temporary neural tension. Recurrent muscle herniation through this defect within the deep fascia during exercise will repeatedly compress the nerve. For this reason, it can often be overlooked as chronic ankle instability or a persistent pain presentation. A detailed history looking out for this mechanism, alongside descriptors linked to neural mechanisms such as "burning or "tingling" pain, with reports of paresthesia, can help in distinguishing between a mechanical and a neurogenic presentation.

Signs and Symptoms

Symptoms are elicited especially while running, with neuropathic "burning" type pain and sensory disturbance extending from the lateral ankle to the dorsum of the foot. If more severe, the sensory disturbance can be persistent and not only triggered by activity. A discrete bulge laterally, proximal to the lateral malleolus, may be seen, enlarging with active eversion of the ankle. A defect within the deep fascia may also be palpable. A positive Tinel's test with paresthesia experienced over the dorsum of the foot indicates the site of compression.

Management

MR imaging can assist in developing a working diagnosis, particularly to exclude possible radiculopathy. Nerve conduction studies can be beneficial, alongside a presentation with loss of motor function to exclude a significant lesion of the common peroneal nerve. Sensory conduction may be affected in more severe SPNES, and this will inform prognosis. Normal nerve conduction studies do not exclude the diagnosis. Other than modifying the aggravating factors, conservative treatment is limited in a military population, and this condition usually requires surgical intervention. Decompression of the superficial peroneal nerve can reduce symptoms, and an early postoperative plan of scar and pain management, education, and desensitization exercises are necessary.

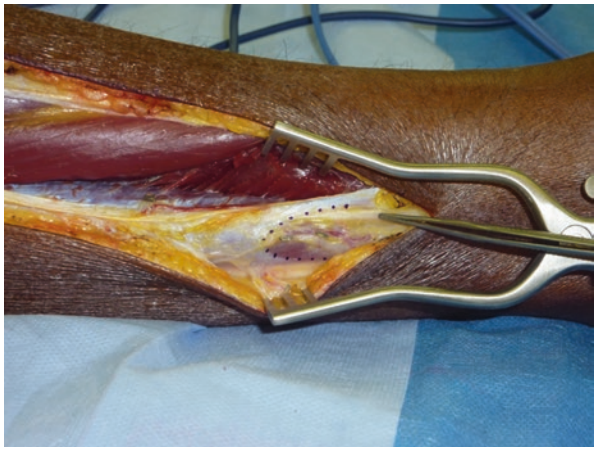

Fig. 36.5 Clinical photograph showing a fasciotomy exposing the superficial peroneal nerve (SPN) with fascial defect distally. The SPN is indicated with forceps

Surgically, the superficial peroneal nerve is exposed via a single incision fasciotomy of the anterior and lateral compartments. The anterior compartment fasciotomy is undertaken, as previously described (see CECS). The lateral compartment is then accessed via incision of the septum between the two compartments starting proximally and extending distally (Fig. 36.5). The SPN is immediately posterior to the septum in the mid part of the leg and is vulnerable to surgical injury here. It is not unusual to be undertaking this procedure in combination with fasciotomy for CECS. Clinically, it may be felt that CECS is affecting both the anterior and the lateral compartments, but there are also variations in the anatomy of the SPN (unpublished) so that part or all of the SPN is found within the anterior rather than the lateral compartment. Using this approach should reduce the risk of injury to the nerve, allow simultaneous fasciotomy, and allow a standard approach for fasciotomy subsequently via the same scar if ever needed. The SPN is exposed from within the lateral compartment proximally to its subcutaneous course distally.

Imaging

MR imaging can be used to follow the CPN and SPN on the axial T1 images between the knee and the ankle. The CPN winds around the fibular neck and divides deep to peroneus longus at the lateral margin of the fibula neck. The SPN is the more posterior of the two divisions and continues deep and then anterior to peroneus longus, before piercing the fascia approximately 10–15 cm above the ankle joint to enter the subcutaneous fat. This is the point where focal fascial thickening, or a defect such as hernia, can lead to nerve entrapment, point tenderness, and distal sensory disturbance. Ultrasound is also very useful for assessing the distal SPN as it approaches and pierces the fascia and can be used to guide a diagnostic local anesthetic injection around the nerve. Guided steroid injections and radiofrequency denervation of the nerve are further therapeutic options if surgery is to be avoided.

Conclusion

Overuse injuries of the lower limb are common among young service personnel and can prove a diagnostic and management challenge. They typically have a long recovery time, and as such there is a large economic and operational cost. Service personnel injured during training, while doing sport, or on operations may never return to full active duty. As diagnostic techniques improve for the many potential causes of exercise-related leg pain, in the future, it may become possible to intervene at an earlier stage to reduce the overall injury burden.

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Lisfranc Injuries

37

Lauren Thomson and Maneesh Bhatia

Introduction

The Lisfranc tarsometatarsal articulation is a very stable construct, an osteologic Roman arch keystone configuration, allowing for significant loading during the gait cycle. Any injury to this joint can lead to significant disability from pain, midfoot arthritis, and an overall decreased quality of life.

The articulation is named after a French Napoleonic field surgeon, Jacques Lisfranc de Saint-Martin (1790–1847) [1], who described a trans-tarsometatarsal amputation common among mounted infantry and not the actual anatomy.

Injury at the Lisfranc joint are uncommon, accounting for 0.2% of all fractures; this is an incidence of 1:55,000. The larger proportion of these injuries are closed 87.5% [2].

Males are two to four times more likely to sustain an injury; however, this is based on a single institutional review and will vary worldwide; with up to 1/3 of injury missed on initial assessment, this is likely to be an underestimation [3–5].

Anatomy of the Ligament

The Lisfranc anatomy can be conceptualized as two anatomical areas—the Lisfranc joint and the Lisfranc ligament.

The osteological structure of the foot can be divided into three columns anatomically: medial, middle, and lateral columns. The medial column comprises the first metatarsal, medial cuneiform, and navicular bones. The middle column is made up of the second and third metatarsals and the intermediate and lateral cuneiforms, and finally, the lateral column contains the fourth and fifth metatarsals and the cuboid.

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The middle column is the least mobile of these and is subject to the highest forces during heel rise in the stance phase of the gait cycle [6]. These columns are an integral part of the longitudinal and transverse arches, which are the main static osseous stabilizer of the Lisfranc joint (Fig. 37.1).

The Lisfranc joint is the tarsometatarsal joint, comprised of the cuneiforms and cuboid, proximally and distally as the five metatarsals. The second metatarsal base is the key to the stability of this construct as the base is recessed into a mortise, with the middle cuneiform acting as the arch "Keystone" [7].

To accompany this bony architecture is a series of ligamentous attachments. All tarsometatarsal joints have dorsal and plantar ligament attachments (with the dorsal attachments being weaker). The second to fifth metatarsal bases also have an intertarsal ligament, except for the first and second bases; instead, the medial cuneiform and the second metatarsal base are linked by a plantar interosseous ligament (Fig. 37.2).

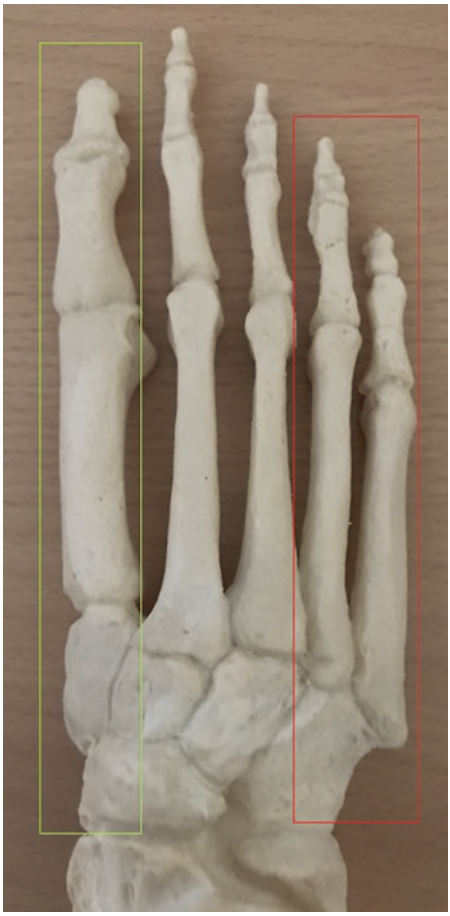

Fig. 37.1 Anatomy of the columns

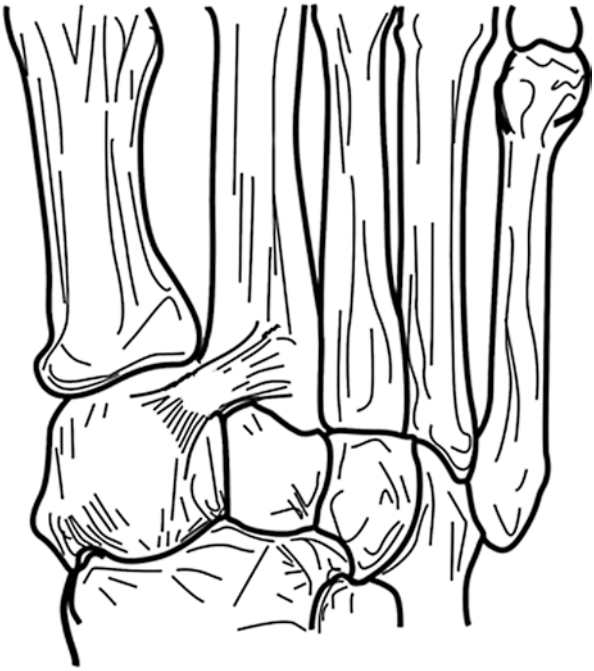

Fig. 37.2 Lisfranc ligament

This ligament is orientated so that the lateral aspect of the medial cuneiform and the lateral base of the second tarsometatarsal are linked—this is the Lisfranc ligament complex [7, 8]. This is a thick, strong ligament, an important dynamic stabilizer, and is vital to help maintain the midfoot arch. A cadaveric biomechanical study by Solan et al. showed that the Lisfranc ligament is three times stronger than the plantar ligaments; these were both stronger and stiffer than the weaker dorsal ligaments [9].

Mechanisms of Injury

The Lisfranc joint can be injured by both low- and high-energy mechanisms. Indirect injuries occur from axial loading onto a plantar-flexed foot, i.e., fall from height and athletic injury. Higher energy injury can result from crush injuries or motor vehicle collisions. During initial assessment, the more subtle injuries can be missed in 20% of patients [10, 11].

This injury is associated with a spectrum of different injury patterns, from sprains (the commonest type in the athletic population), pure ligamentous injury, through to fractures of the whole of the Lisfranc joint (metatarsals, cuneiforms, and the cuboid) [12]. Indirect injury is more common with midfoot twisting and bending moments causing ligamentous disruption [13, 14].

Table 37.1 Myerson classification of Lisfranc injury

Incongruity	Subtype	Description
Type A: complete	–	Dislocation of all the metatarsals in the same direction (lateral or dorsoplantar)
Type B: incomplete	B1	Medial dislocation involving the first metatarsal only
	B2	Lateral dislocation involving any of metatarsals two to four
Type C: incomplete/ complete	C1	Divergent, incomplete dislocation involving the first metatarsal and some lateral metatarsals
	C2	Divergent, complete dislocation involving the first metatarsal and some lateral metatarsals

Stavlas et al. noted in their systematic review that the majority of cases higher energy injuries were more common than lower energy injuries (58% vs. 42%), the injuries were also more likely to be closed 87.5% [2].

The lower energy injury pattern is often limited to the first, second, and third metatarsal bases, with widening of the first intermetatarsal space with or without dorsal subluxation of the base of the second metatarsal (weaker ligaments). These purely ligamentous injuries are often subtle and therefore missed, so they require a higher index of suspicion given the context of the injury [15].

Higher energy injury is often more overt in clinical presentation and can be associated with significant soft-tissue envelope injury. These injury types are associated with compartment syndrome and, therefore, neurovascular injury—requiring emergent treatment.

There are a number of classification systems for categorizing the Lisfranc injury; some focus on purely ligamentous and others on fracture dislocations. The radiological classifications comment on the direction of the injury—homologous, isolated, or divergent—in the original paper by Quenu and Kuss; this has been updated by Harcastle, which grouped the radiographic features into congruity of the tarsometatarsal joint, Types A, B, and C; this was updated by Myerson, who subdivided the B and C categories depending on the number of metatarsal involvement. This classification allows for accurate communication but provides little information with regard to treatment [11] (Table 37.1).

History and Examination Findings

During the initial patient history, eliciting mechanism is important, especially if there is a subtle ligamentous injury; the bending and twisting on the plantar-flexed foot should alert the clinician to the possibility of a Lisfranc injury. The patient may also present partial weight-bearing to the accident and emergency department. In general, the patient will present with significant midfoot pain, non-weight-bearing and consider plantar ecchymosis as pathognomonic of a tarsometatarsal or Lisfranc injury [16].

Clinical examination may show plantar ecchymosis, edema, skin lacerations or blistering, and foot deformity during the physical examination; note the neurovascular status of the foot to enable rapid identification of an impending compartment syndrome.

Radiology

Plain film radiograph remains the mainstay of imaging for the initial diagnosis of a Lisfranc injury; as the disruption is a 3D disruption, three radiographs need to be obtained: the anteroposterior view (ideally weight-bearing, but this is often not pragmatic due to excess pain), the 30° oblique view, and the lateral view (to identify dorsal subluxation). If any uncertainty remains, plain film radiographs of the contralateral foot for comparison may be required [17] (Fig. 37.3).

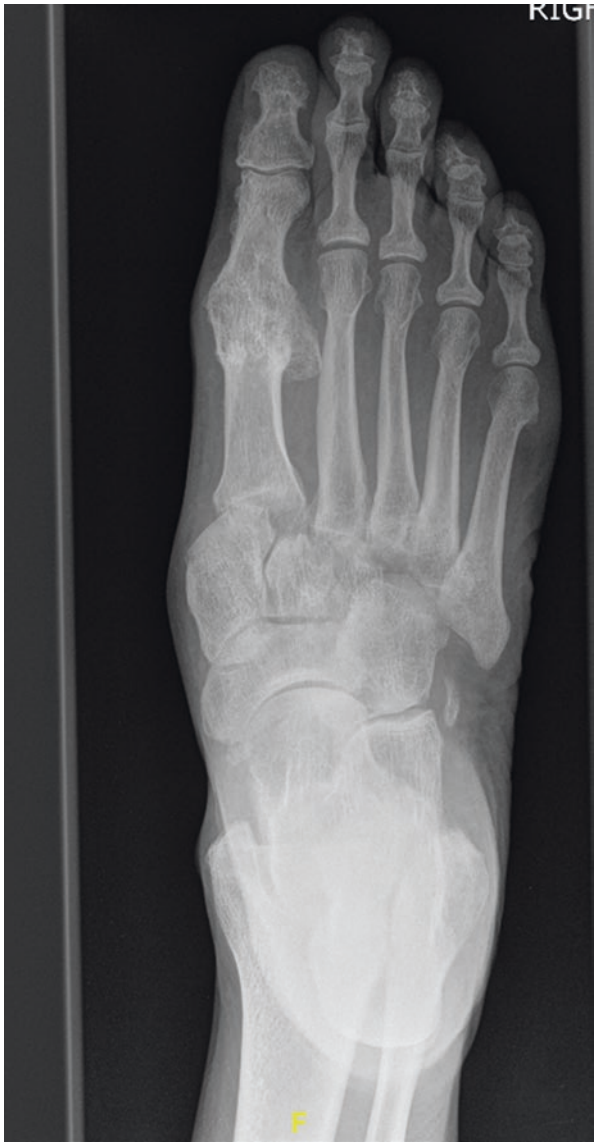

Fig. 37.3 Type C2 Myerson injury

The clinician can then identify the loss of alignment between the second metatarsal translation laterally relative to the middle cuneiform, of 2 mm or more (anteroposterior radiograph), the medial borders of the second metatarsal base and middle cuneiform shoulder be aligned (Fig. 37.4). The oblique view allows the clinician to review the alignment between the medial border of the cuboid and the medial border of the fourth metatarsal [7]. In some cases, the "Fleck" sign, a bony avulsion of the second metatarsal base, can be seen in the intermetatarsal space (Fig. 37.5). The lateral view will show a degree of dorsal subluxation of the second metatarsal base.

If there is still doubt regarding the diagnosis, CT (computerized tomography) and MRI (magnetic resonance imaging) are two further modalities for Lisfranc review. The CT will image any subtle fracture missed on the previous radiographs and will have an accurate review of articular alignment [18].

MRI is more useful if a purely ligamentous injury, including sprains, is suspected; soft-tissue edema is more apparent on this imaging modality. MRI has been shown to have a sensitivity and positive predictive value of 94% for correctly diagnosing unstable midfoot Lisfranc injuries [19].

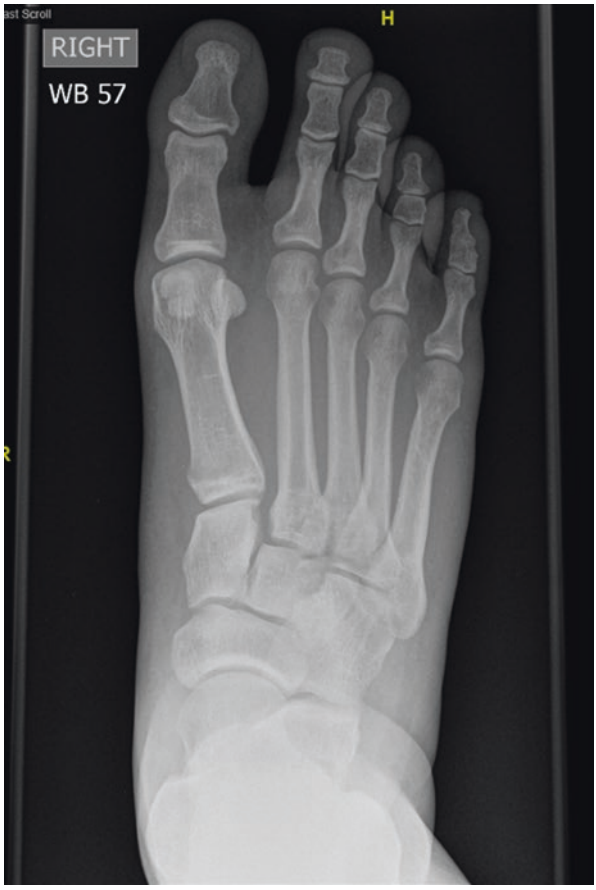

Fig. 37.4 Metatarsal translation

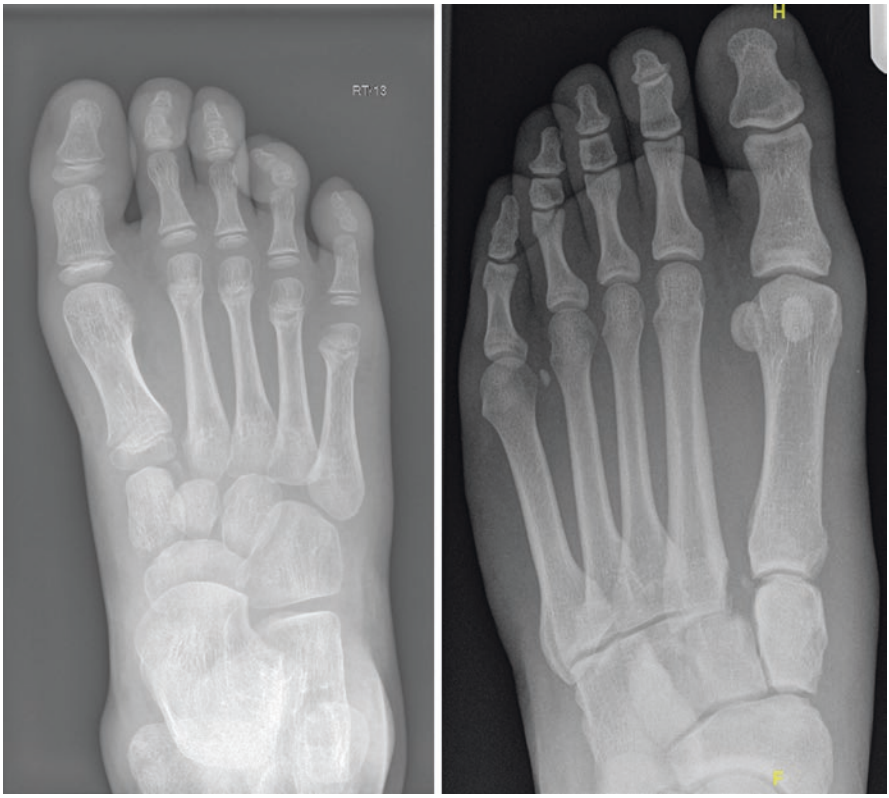

Fig. 37.5 Fleck sign

Management

Management depends on the type of injury; those fractures that are open and contaminated will require washout, reduction, and temporizing fixation (dependent on the patient's physiological status); those with a midfoot sprain can be managed with non-weight-bearing. Identification of the injury type will guide management.

Stable, undisplaced injuries can be managed nonsurgically with cast immobilization and non-weight-bearing on the affected limb for 6 weeks. Dependent on the pain at 6 weeks, if ongoing, a further 4 weeks of weight-bearing in a walker boot can commence [17].

If there is a proven ligamentous injury without diastasis, then 12–16 weeks of immobilization is required. To prevent stiffness within the ankle joint, physiotherapy-guided range of movement exercise should begin at week 6–8 [20].

On presentation, any displaced injury 2 mm or more must be reduced, if possible, to lessen the threat to the skin and the amount of soft-tissue swelling and to enable earlier fixation; however, there may be a delay of 1–2 weeks before the soft-tissue

envelope is able to undergo surgical intervention. The patient should have their foot elevated to decrease swelling.

Anatomic reduction is key to a good surgical outcome; stabilization should proceed in a proximal to distal, medial to lateral direction [21]. Stiffening the medial and middle columns does not have a significantly detrimental outcome to foot stability; however, stabilization of the fourth and fifth metatarsals should not be rigid; K-wires are commonly used for this, then removed at week 4–6.

There are multiple ways of approaching the fixation of a Lisfranc injury; one or two intermetatarsal web space (with a wide skin bridge) incisions allow for a wide surgical field, addressing all injuries; fluoroscopic fixation percutaneously is performed only if anatomical reduction (closed) is achievable. A dorsal approach is the most commonly used incision, the key being to identify extensor hallucis brevis, under which the neurovascular bundle is located and can therefore be protected (Figs. 37.6, 37.7, and 37.8) [7].

Two methods are commonly described in the literature to stabilize the medial and middle columns: cannulated screw fixation and dorsal plating. A cadaveric review by Alberta et al. found no significant difference between loading and unloading joint stress, suggesting that screw fixation and dorsal plating/tubular playing (tarsometatarsal fixation) are equivalent methods of stabilization [22, 23]. The screws will require removal, but there is no consensus within the literature as to when this should occur [2, 8].

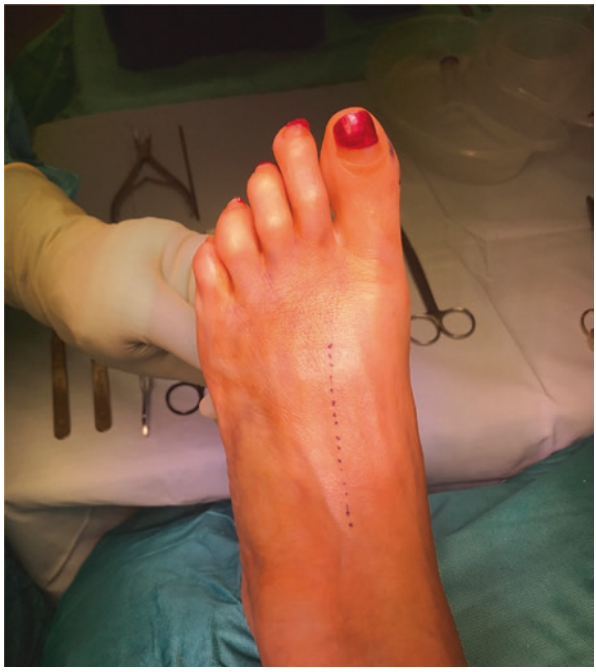

Fig. 37.6 Dorsal incision

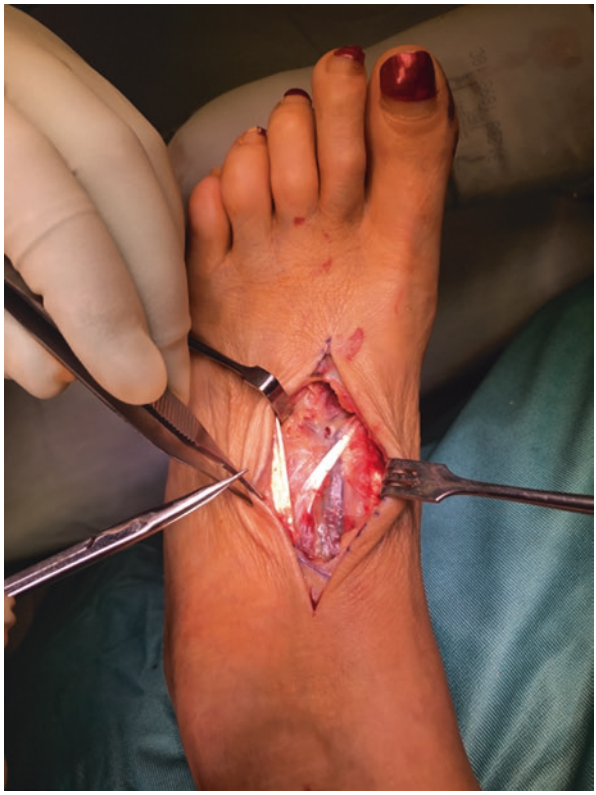

Fig. 37.7 Extensor hallucis brevis and neurovascular bundle

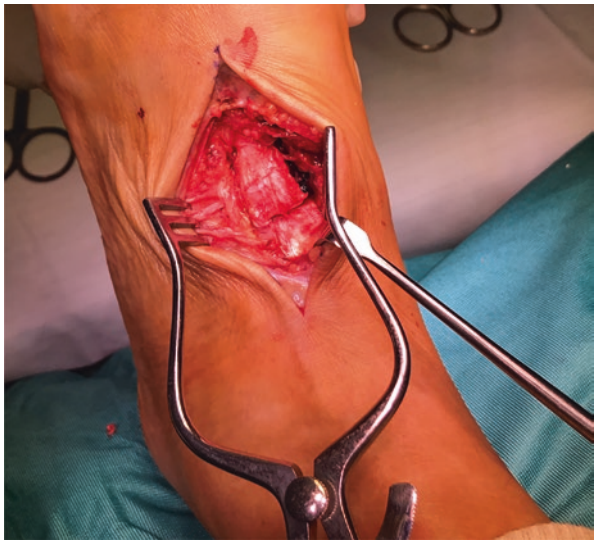

Fig. 37.8 Lisfranc disruption

The advantage of dorsal plating is that the cartilage remains virgin and is not disrupted, unlike with percutaneous screw fixation, nor do the plates have the same rate of fracture as the screws alone (Figs. 37.9 and 37.10) [23].

The majority of screw fixations are undertaken with small fragment screws (3.5–4.0 mm), and stabilization of the fourth and fifth metatarsals is only performed if there is lateral column instability with K-wires (1.6–2 mm) [2].

Lee et al. noted in their cadaveric study that screw fixation provided a more stable anatomical construct than K-wiring alone and therefore posed a reduced risk of posttraumatic osteoarthritis [24].

Primary arthrodesis, although uncommon as a procedure, can be used in nonreconstructible cases, significant intra-articular injury, or multidirectional instability [7, 20].

A prospective cohort study by Coetzee et al. compared the outcomes of primary arthrodesis with open reduction and screw fixation in purely ligamentous injuries. The study noted that patient-reported outcome measures were better in the primary arthrodesis, as was anatomic reduction. Their patient cohort also stated a 92% return to preinjury levels, compared to open reduction and internal fixation.

Henning et al., however, noted that although primary arthrodesis resulted in significantly fewer further procedures—i.e., metalwork removal and salvage arthrodesis—there was no significant difference in their patient outcomes.

This was corroborated by a systematic review and meta-analysis by Smith et al. Their analysis of 101 Lisfranc injuries from three studies found that the risk of hardware removal for open reduction and internal fixation was significantly higher than for primary arthrodesis, but patient-reported outcome measures for both were

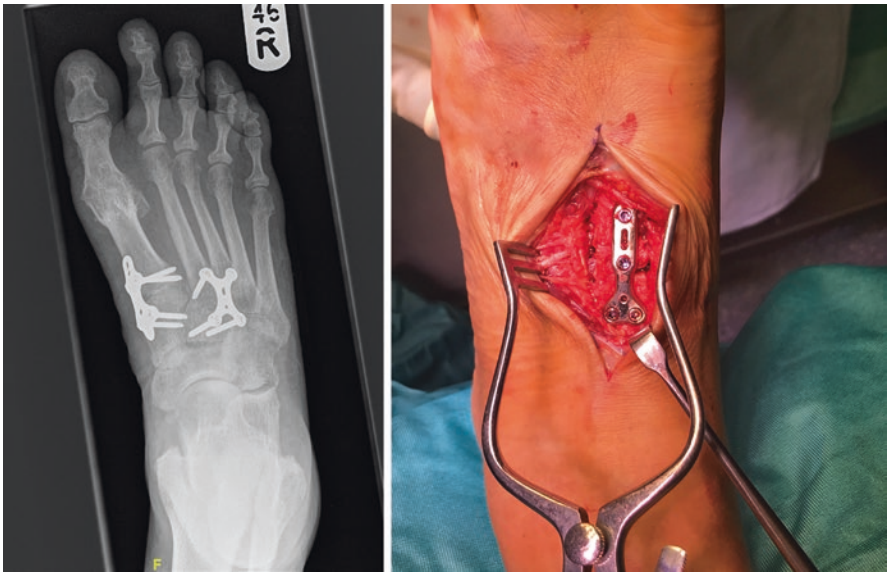

Fig. 37.9 Dorsal plating: two columns, single column

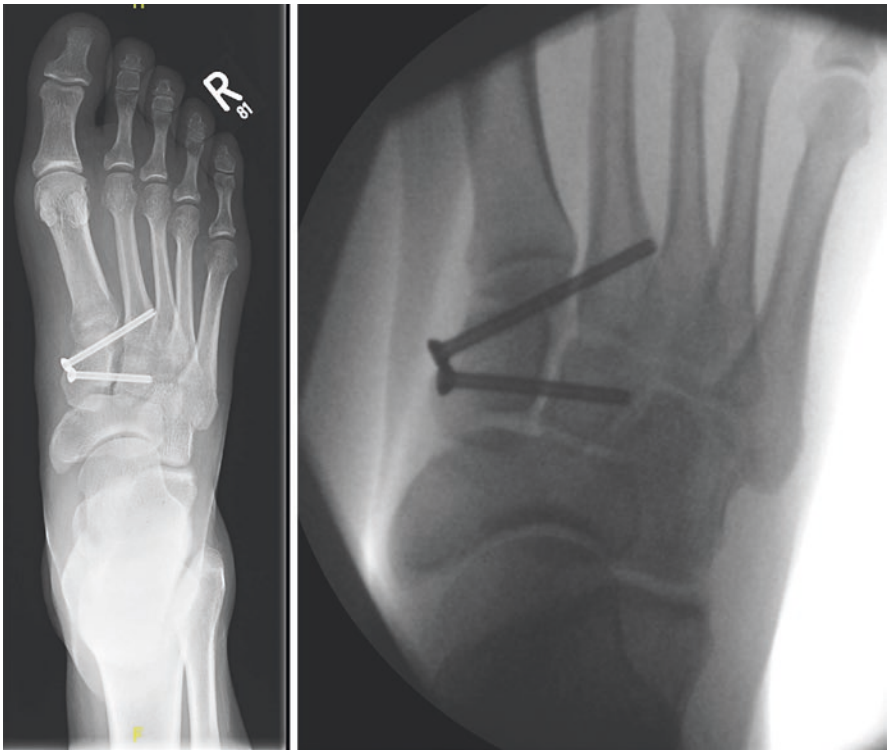

Fig. 37.10 Percutaneous screw fixation

similar. Also, neither ORIF nor fusion had a significant risk of nonanatomic alignment [25–27].

The authors do not perform primary arthrodesis for purely ligamentous injuries and reserve this option for cases with significant articular damage.

In the authors experience, we consider fusion once we ascertain that there is significant cartilaginous injury.

Finally, if there is significant bony destruction, vascular compromise, and/or soft-tissue loss, leaving the foot nonviable, amputation must be considered as an option.

Postoperative

A systematic review by Stavlas et al. studied 11 articles, and the common consensus for postoperative management of Lisfranc fixation was a short leg plaster, with or without a toe plate; ankle in plantigrade (to prevent gastrocnemius shortening and stiffness within the ankle); and appropriate thromboprophylaxis. On average, the majority of published literature has a toe-touch regimen of 8 weeks, before

progressing to full weight-bearing. However, this does not have to remain in plaster of Paris; conversion to a removable boot has also been noted in the literature. With more extensive reconstruction, a longer period of up to 12 weeks of protected weight-bearing has also been documented [2, 27].

Outcomes of Lisfranc Injuries

A missed Lisfranc injury will result in significant consequences for the patient. This will lead to posttraumatic midfoot arthritis (often quoted as high as 94%) and traumatic planovalgus deformity and will result in salvage arthrodesis surgery for the patient [15]. Poor anatomical reduction after open reduction and internal fixation will also increase the probability of developing posttraumatic arthritis, up to 60% [28].

Postinjury arthrodesis is as high as 20% following posttraumatic arthritis; despite a much higher radiographic reported incidence of arthritis following Lisfranc injury, radiographic evidence of arthritis does not automatically indicate surgery [14]. Overall, for the significance of the injury, complications are often minor, but this should not negate the fact that a Lisfranc injury can be associated with amputation [2, 29].

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Pediatric Knee Injuries

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Humza T. Osmani, Wasim S. Khan and Ioannis Pengas

Introduction

Pediatric knee injuries have rapidly increased over the past few decades due to the steady rise in participation and intensity in school sports [1, 2]. These injuries are of particular importance in the skeletally immature patients, often resulting in growth disturbance and earlier degenerative changes with profound long-term consequences. In this review, we discuss the diagnosis and management of common and uncommon but significant pediatric fractures around the knee. The following injuries/fractures are discussed due to their prevalence and/or significance on the pediatric population with regard to consequences: (1) physeal fractures around the knee (distal femur and proximal tibia), (2) tibial spine fractures, (3) patella sleeve fractures, (4) metaphyseal fractures, (5) tibial tubercle fractures, (6) bipartite patella, and (7) stress fractures of the patella.

Physeal Fractures Around the Knee

The physis of the distal femur and proximal tibia is responsible for 70% of the overall length of the lower limb with an average growth of approximately 9 mm and 6 mm per year, respectively [3]. In addition, the tibial tubercle apophysis contributes to appositional and angular growth and the proximal fibula to angular and longitudinal growth. Disturbance to any one of these physes can result in angular deformity, growth arrest, and leg length discrepancy. The Salter-Harris classification can be used to categorize physeal fractures around the knee [3].

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Distal Femoral Fractures

Distal femoral fractures make up 2–5% of physeal fractures [4]. They result from a varus or valgus force across the knee resulting in failure of the periosteum on the tension side of the physis and a metaphyseal fragment on the compression side. Anterior displacement of a fracture is due to a hyperextension mechanism and posterior displacement due to posterior force with a flexed knee [5].

For nondisplaced, stable fractures, patients can be treated with long leg casting as long as they are followed up closely. There should be <2 mm of physeal displacement to reduce the risk of growth disturbance [6]. Salter-Harris Type 1 and 2 fractures can be reduced and treated with closed reduction and pinning. If the physis needs to be crossed, this should be performed with smooth K-wires, which ideally should be removed at 4 weeks to prevent pin site infection. A Salter-Harris type 2 fracture with a large metaphyseal, Thurston-Holland fragment can be treated using a metaphyseal lag screw avoiding the physis. Salter-Harris type 3 and 4 fractures that are nondisplaced and stable can be treated with immobilization in a long leg cast. However, as there is a propensity for late displacement, most authors recommend internal fixation [7]. These intra-articular fractures need reduction of the articular surface prior to internal fixation. If anatomic reduction is not possible via closed techniques, an incision over the physis is necessary to remove the interposed periosteum. Depending upon the size of the patient, 4.5–7.3-mm cannulated screws can be used, placed parallel to the physis to compress the fracture [7]. Patients are then immobilized in a hinged knee brace for 4 weeks to allow movement and prevent arthrofibrosis [6].

The risk of growth arrest is 40–90% [8]. This is much higher than in other locations because of the large amount of growth and amount of energy required to cause the fracture, as well as the undulating pattern of the physis. Physeal bar excision may be required if it is more than 50% with at least 2 years of growth remaining [8].

Proximal Tibial Fractures

These fractures are uncommon, accounting for <1% of all physeal fractures [6]. A significant force is required to produce this sort of fracture as the superficial medial collateral ligament (MCL), lateral collateral ligament (LCL), tibial tubercle, and fibula provide a structural support at preventing displacement [8, 9]. Nondisplaced Salter-Harris type 1 and 2 fractures can be immobilized in a long leg cast for 4–6 weeks [5]. Displaced fractures often can be reduced with closed manipulation and treated with a long leg cast [5]. For displaced fractures with <2-mm displacement, open reduction should be performed and held in situ using 2–3.5-mm transphyseal smooth pins. These can be placed in an antegrade or retrograde fashion. Nondisplaced Salter-Harris type 3 and 4 fractures can be treated conservatively in a long leg cast; however, their propensity for displacement is high, and therefore percutaneous screws are recommended [5]. For displaced fractures, open reduction internal fixation (ORIF)

should be performed. Screws or pins are placed parallel to the joint surface in the epiphysis or metaphysis, allowing for compression at the fracture site [6].

Tibial Spine Fractures

These avulsion fractures are a well-recognized injury affecting the pre- adolescent and early adolescent populations and represent an anterior cruciate ligament (ACL) injury with associated instability, loss of function, and long-term disability. It is a rare injury, most commonly seen in 8–14-year-olds [10]. It occurs because of the rapid deceleration or hyperextension of the knee and is associated with other conditions like meniscal injury, osteochondral defects, collateral ligament injury, and capsular damage. Bony avulsion, rather than ligamentous rupture, has been postulated to occur as an end result in this younger age group, either because the physeal bone is weaker and hence more susceptible to injury or the ligament itself allows for greater elasticity, resulting in a bony avulsion rather than a ligament rupture [11, 12]. Tibial spine fracture is a knee injury, which was first classified by Myers and McKeever in 1959, with an additional grade added in 1979 by Zaricznyj [13, 14].

Initially, all grades of avulsion fractures were managed nonoperatively, but as surgical techniques advanced, Grade 3 and 4 fractures started to be surgically managed; more recently, Grade 2 fractures have also been managed surgically, while nonoperative management is reserved for patients with Grade 1 fractures with immobilization in a plaster or knee brace [15, 16]. Ten to twenty degrees of knee flexion is recommended as full extension may place excessive tension on the ACL and popliteal artery [10]. Immobilization is then recommended for approximately 6 weeks, depending on the age of the patient, healing rate, and radiological findings. Prolonged immobilization may lead, however, to arthrofibrosis and an extensor lag [10].

There are multiple well-described techniques for the surgical treatment of tibial spine avulsion fractures, including antegrade or retrograde screw or K-wire fixation, and suture anchor or suture fixation [17, 18]. Suture fixation techniques have usually been described through tibial drill holes and tied either through a suture disc or over a tibial cortical bone bridge [15, 18]. Where screw fixation is used as a fixation technique, one or more cannulated cancellous screws are typically used. In a skeletally immature population, it has been suggested that screw threads should not cross the physeal scar line, or there is the risk of resultant growth disturbance [15, 19]. Where physeal sparing techniques are used, biomechanical studies suggested that screw fixation is inferior to suture technique [15, 20]. Recently, it has been recognized that insertional injuries, both proximal peel-off and tibial spine are amenable to repair if treated acutely, inferring the benefit of earlier rehabilitation and maintained proprioception while preserving full reconstructive options if required later. Formal rehabilitation is crucial as it encourages a faster recovery and prevents the development of secondary complications [21].

Patella Sleeve Fractures

The incidence of patellar fractures in skeletally immature patients is low, comprising only about 6.5% of all such fractures [22]. Patella sleeve fractures are the most common type in skeletally immature children. The fracture is caused by an acute injury due to powerful contraction of the quadriceps with the knee flexed, usually during sports. This fracture differs from straightforward avulsions because of the "sleeve" of periosteum, which is pulled off the patella. It can be easily missed on plain radiographs, especially in fractures with a very small avulsed bony fragment; however, it can also present as patella alta on plain radiographs and diagnosis confirmed with ultrasound. Where a bony fragment is present, it is accompanied by a separation of a large fragment of articular cartilage, which can be detected on MRI [22, 23].

In a severely displaced sleeve fracture, open reduction and internal fixation are mandatory in order to achieve a good outcome [24–26]. For undisplaced fractures, conservative management usually achieves good results; however, it may result in weakness of the extensor mechanism associated with patella alta and ossification of the patellar tendon if the diagnosis is missed or inappropriately treated [27].

Metaphyseal Fractures

These fractures, although rare, are significant as they can result in the development of a late valgus deformity, known as "Cozen's phenomenon" [28, 29]. This fracture commonly occurs in children between the ages of three and six and can be due to a direct or indirect force. Indirect forces are more common, whereby "the lower leg is forced into abduction, adduction, or hyperextension against a knee fixed in space" [9].

Nondisplaced fractures can be treated in a long leg cast with a varus mold. Patients can start weight-bearing at 3 weeks and require 6–8 weeks in the cast with serial radiographs. Displaced fractures should be reduced and thus require sedation or general anesthesia, followed by a long leg cast with a varus mold. If there is inability to adequately reduce a displaced fracture, then open reduction is mandated. This would involve a limited medial approach to the proximal tibia and removing periosteum or the tendons of pes anserinus from the fracture site that may be preventing reduction. Internal fixation is rarely required for these fractures [9].

The subsequent valgus deformity that may ensue should be observed for 12–24 months with the expectation of eventual resolution, and this should be communicated with the parents and the patient beforehand. If the deformity fails to resolve, then bracing, as well as surgical interventions, including a medial hemiepiphysiodesis [29] and osteotomy, may need to be considered [28].

Tibial Tubercle Fractures

Tibial tubercle fractures are a relatively uncommon injury, with an incidence between 0.4% and 2.7% [30–32]. They are usually seen in adolescent males nearing skeletal maturity with well-developed quadriceps musculature. These fractures present with marked displacement of the entire proximal apophysis, with or without intra-articular extension. The majority of these injuries are incurred during athletic activity involving jumping, most commonly basketball, and are the result of one of two possible mechanisms of injury: violent knee flexion against tightly contracting quadriceps (landing from a jump) [33] or violent quadriceps contraction against a fixed foot (when jumping) [34].

The tibial tuberosity develops from a secondary ossification center in the proximal aspect of the tibia. In contrast to the proximal tibial epiphysis, which develops in compression, the tibial tuberosity is an apophysis and develops in traction [35]. Closure of the proximal tibial epiphysis, which extends distally toward the tubercle apophysis, may leave a mechanically vulnerable period that predisposes the tuberosity to avulsion injury [36]. The classification of tibial tubercle fractures is as follows: type I is an avulsion of a small part of the tibial tubercle, distal to the proximal tibial physis. Type II extends across the physis but does not enter the knee joint. Type III is an avulsion that extends proximally to the physis into the knee and is the most common type [37]. This classification was modified to more accurately define specific fracture patterns and to establish treatment for different fracture types by including displacement and comminution [31].

The treatment goal for these fractures is to restore the extensor mechanism and, where disrupted, the joint surface. Closed treatment generally entails closed reduction and immobilization in a long leg or cylinder cast for approximately 4 weeks. Open reduction and internal fixation entail fixation with screws, as well as tension band wiring [38] or suture repair of periosteum, followed by casting for 3–4 weeks. Arthroscopic-assisted open fixation is also preferred by some surgeons [9]. A systemic review by Pretell-Mazzini et al. [37] has shown that 98% return to preinjury activity, regardless of the type of fracture, after open reduction and internal fixation. Fracture consolidation was achieved in 99.4% of cases with an overall complication rate of 28.3%, the most common being bursitis, leading to the removal of metalwork and implant (55.8%), with refractures occurring in 6.3%.

Bipartite Patella

This is more common in males. It is often bilateral and is a normal variant representing a failure of fusion. There are three sites at which it is found, and each has an important soft-tissue attachment. It is classified depending on the location of the separate fragment into: type 1 involves the distal pole of the patella with attachment of the patellar tendon (5%); type 2 involves the lateral margin of the patella with attachment of the lateral retinaculum (20%), and type 3 involves the superolateral

corner, the insertion of vastus lateralis. This is the most common site for symptoms (75%) [39, 40].

Most patients are asymptomatic, and the diagnosis is made incidentally. If symptomatic, patients complain of anterior knee pain from direct or indirect trauma, which is aggravated by jumping or squatting. This disrupts the synchondrosis, causing symptoms similar to fractures, including localized tenderness over the accessory fragment, hematoma, unusual patella prominence, or a larger than normal patella [40].

Conservative management involves rest, immobilization, NSAIDs, and physiotherapy. Occasionally, symptoms are severe enough and warrants surgery. The simplest and most effective procedure is excision of the superolateral fragment through an incision in line with the fibers of vastus lateralis [40] and, alternatively, limited detachment of vastus lateralis from the fragment, which removes the stress on the fragment, allowing union [41].

Stress Fracture of the Patella

Incidence of these fractures is approximately 1%. Stress fractures occur when there is repeated trauma or overuse related stress to a bone, without sufficient time for it to heal and remodel [42]. Microscopic fractures can subsequently propagate into macroscopic fractures. They tend to occur due to two different mechanisms, leading to different patterns of stress fractures: firstly, longitudinal fractures due to a lower force at high frequency, for example, from running, and secondly, transverse fractures due to jumping and/or kicking, which causes repeated muscular contractions [42, 43]. Patients with cerebral palsy with flexion contractures have also been reported as another group in which this has been reported [44].

Diagnosis can be difficult due to their rarity and delay in considering this as a differential. Furthermore, they can be misinterpreted as bipartite patella or Sinding-Larsen-Johansson disease [41, 43]. Initial radiographs will be sensitive in only a third of the cases. More accurate and sensitive investigation tools are bone scan or magnetic resonance imaging [43].

Due to a small number of reported series, there is a lack of definitive treatment recommendations for patella stress fracture; however, in those with cerebral palsy, management includes release of the hamstrings and correction of the flexed knee deformity [44]. In athletes in whom anterior pain persists despite conservative management, stress fractures should also be considered as the underlying pathology [42].

Conclusion

Pediatric knee injuries are common and are rapidly increasing due to the steady rise in sport participation. These injuries can be challenging to spot and are of particular importance in skeletally immature patients as early degenerative changes potentially have profound consequences in the long term. Controversies regarding

operative versus nonoperative treatment exist; varying opinions of the type of operative intervention, due to risk of complications, make the management of these injuries difficult too. Ensuring that patients and their parents are correctly counseled from the beginning remains vital.

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The Foot

39

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The foot has two key functions:

1. Providing stability and balance to the body when standing to support the weight of the body
2. Acting as a rigid lever to push off when a person is walking or running, as well as to allow one to be mobile enough to adapt to uneven ground and absorb shock during mobilization

Brief Discussion on Arches of the Foot

There are four main arches in the foot to consider. Two are longitudinal arches running proximally to distal and two run from medial to lateral. The arches of the foot act as a spring, enabling the foot to bear weight, as well as absorb shock, during movement:

1. The medial longitudinal arch, which runs from the calcaneus to the head of the first metatarsal, includes the bones in between; the talus; the navicular; the first, second, and third cuneiforms; and the first, second, and third metatarsal heads.

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The summit is the midtarsal joint. It is the higher of the two longitudinal arches and confers significant flexibility to the foot. The plantar calcaneonavicular ligament (spring ligament) confers significant support. The main supporting tendon is the tibialis posterior.

2. The lateral longitudinal arch runs from the calcaneus to the fourth and fifth metatarsal heads and includes the cuboid and the fourth and fifth metatarsal bones. This arch is flatter than the medial one. In the standing position, it lies flat to the ground. It is supported by the long plantar and plantar calcaneocuboid ligaments.
3. The metatarsal arch extends in the coronal plane from the first to the fifth metatarsal heads with the summit at the second and third metatarsals.
4. The transverse arch of the foot is also located in the coronal plane. It is an incomplete arch consisting of the metatarsal bases, the cuboid, and the three cuneiform bones. Its peak is the base of the second metatarsal acting as the keystone. This arch is disrupted as part of traumatic Lisfranc disruptions.

Biomechanics and Movements of the Foot

The biomechanics of the foot and ankle are important to the normal functioning of the lower extremity. The main function of the foot is mechanical, i.e., to transfer loads from the leg to the ground. With appropriate neuromuscular coordination, the human foot, as a final link, remarkably adapts both in static and dynamic situations to a variety of ground terrains. Inadequate distribution of these forces could lead to abnormal stress and the eventual breakdown of soft tissues. The foot has got both dynamic and static supports. The static structures include the bones, joint surface congruity, ligaments, and fascia. The dynamic components include the tendons and muscles.

Muscle activity is not necessary to support the fully loaded foot at rest. The maintenance of the arch in the static foot is attributed to ligamentous and osseous support. In the static stance position, the plantar aponeurosis takes up approximately 60% of the stress of weight-bearing and the beam action of the metatarsals approximately 25%. The ability of the plantar aponeurosis to absorb stress increases as the aponeurosis becomes taut with toe extension. This mechanism has been described as the windlass effect [1].

The foot is made up of 28 bones and approximately 44 articulations. Consequently, most movements of the foot involve a large number of joints, and the movement of the individual joints in each movement is difficult to describe. The movement of the ankle joint is triplanar (occurring simultaneously in the sagittal, coronal, and horizontal planes) with predominant movement in the sagittal plane, i.e., plantar flexion (extension) and dorsiflexion (flexion). Subtalar is part synovial and part syndesmosis. The subtalar joint is part and parcel syndesmotic. The anterior syndesmotic part is separated from the posterior syndesmotic part by a funnel-shaped channel called the sinus tarsi, which is more or less horizontal and runs in a posteromedial to anterolateral direction. The subtalar joint is often regarded as a major contributor to the pronation and supination range of

motion, but results show that the contribution of the subtalar joint (24%) is less than that of the talonavicular joint (44%).

Pronation involves simultaneous abduction, dorsiflexion, and eversion. Similarly, supination involves simultaneous adduction, plantar flexion, and inversion. Pronation and supination are rear-foot movements when the foot is not weight-bearing [2]. During weight-bearing, these movements are constrained, depending on the magnitude and distribution of the ground reaction force acting on the plantar part of the foot.

When the whole foot is rotated internally about a longitudinal axis such that the sole faces medially, the foot is inverted; the opposite is eversion, which occurs in the subtalar joint. When the anterior part of the foot moves on the posterior part on a vertical axis, the foot is said to be adducted, while the opposite movement is called abduction. Both adduction and abduction occur at the talonavicular joint. The standard range of movements for dorsiflexion is 30°, and plantar flexion is 50°. At the subtalar joint, there is 20° of inversion and 10° of eversion.

Flatfoot

When discussing about flatfeet, it is best to take an approach broadly, dividing the topic into congenital and acquired pathologies. It also works to divide the topic into a younger population and an older/acquired population.

Three main congenital flatfoot pathologies exist: congenital vertical talus, flexible pes planus, and calcaneovalgus:

Congenital Vertical Talus

Congenital vertical talus is a rare foot deformity wherein patients present with an irreducible dorsal dislocation of the navicular on the talus. Half of the cases are bilateral with an unknown etiology, and 50% have associations with neurologic/neuromuscular conditions, congenital abnormalities, or chromosomal/genetic defects. Common conditions of association include arthrogryposis and myelomeningocele. There may be a positive family history in a fifth of cases with inheritance in an autosomal dominant fashion.

Congenital vertical talus manifests at birth with a rigid, fixed equinus hindfoot deformity. The hindfoot is in valgus due to the pull of a tight tendo Achilles, as well as tight posterolateral capsular structures of the ankle and the subtalar joint. The midfoot is pulled into dorsiflexion by tight anterior tendinous structures. The posterior tibialis tendon and the peroneus tertius are dislocated and act as dorsiflexors rather than plantar flexors. This gives the foot a convex/rocker bottom plantar surface, as well as deep creases in the dorsum. The talar head is palpable in the medial plantar arch, and the navicular is dislocated laterally and gives the midfoot fixed dorsiflexion.

A thorough family history may be positive for congenital vertical talus. A neurological exam is important as the patient may require imaging of the neurological axis. Plain radiographs have shown a vertical talus, the calcaneum in equinus, and a dorsally dislocated navicular. The lateral talocalcaneal angle is increased (Fig. 39.1).

Three lateral views are necessary (maximum dorsiflexion, maximum plantar flexion, and neutral) to show a rigid deformity. The anteroposterior (AP) radiograph demonstrates an increased talocalcaneal angle greater than 40°. A less severe form exists in the shape of an obliquely orientated talus, and imaging (forced plantar flexion radiograph) distinguishes it from a vertical talus (Fig. 39.2).

Management has trended away from surgery to serial manipulation and casting in the form of reverse Ponseti technique, followed by percutaneous Achilles tenotomy and fixation of the talonavicular joint [3]. Initially, the foot is pushed into plantar flexion and inversion/adduction. This is done progressively, with weekly cast changes with the final cast placing the foot into extreme equinovarus. K-wire stabilization of the talonavicular joint and Achilles tenotomy follows. Percutaneous capsulotomy may be necessary to achieve full reduction prior to K-wire fixation. A period of bracing follows as per the normal Ponseti regimen. More invasive

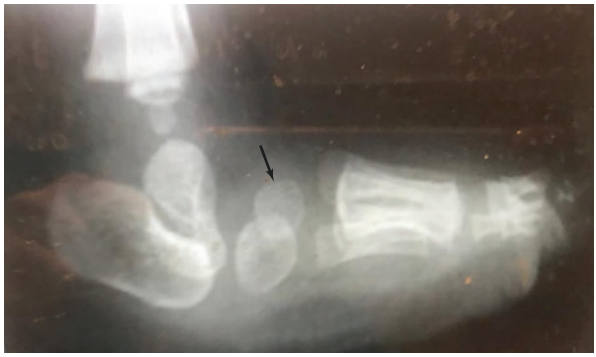

Fig. 39.1 Lateral view of the foot showing a vertical position of the talus and an increased talocalcaneal angle. The arrow is pointing at the navicular, which is dorsally dislocated

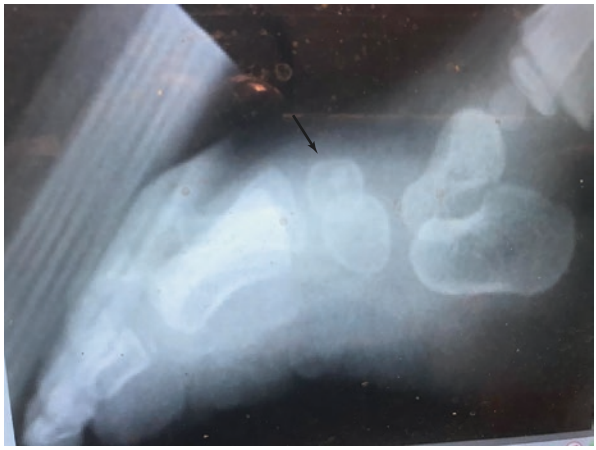

Fig. 39.2 Forced plantar flexion lateral view showing the navicular (arrow), which is still dorsal dislocated, distinguishing it from an obliquely oriented talus

procedures may be necessary and are performed at 6–12 months old, including tendon lengthening, spring ligament reconstruction, and tibialis anterior tendon transfer to the talar neck. Recalcitrant cases need more invasive open procedures, with fusion being an option in later life.

Calcaneovalgus

Calcaneovalgus is a congenital flexible deformity that is due to intrauterine malpositioning. Here the foot is in extreme dorsiflexion at the hindfoot, commonly with the dorsum of the foot flat, touching the anterior aspect of the tibia (Fig. 39.3).

The foot is also everted and abducted. It is passively correctable and thus can be differentiated from congenital vertical talus. It has preponderance in firstborn children and females. Plain radiographs, taken on the foot in plantar flexion, will show a line drawn through the talus lining up with the first metatarsal to differentiate it from congenital vertical talus. Management involves passive stretching with the addition of serial casting if necessary. Parent reassurance is important in these cases.

Flexible Pes Planus

Flexible pes planus is a relatively common finding in the pediatric population. But a lack of understanding may lead to these patients engaging with an orthopedic surgeon. In this group of patients, during standing, the foot is flat with the heel in valgus. Upon tiptoeing, the arch reconstitutes, hence the term flexible flatfoot.

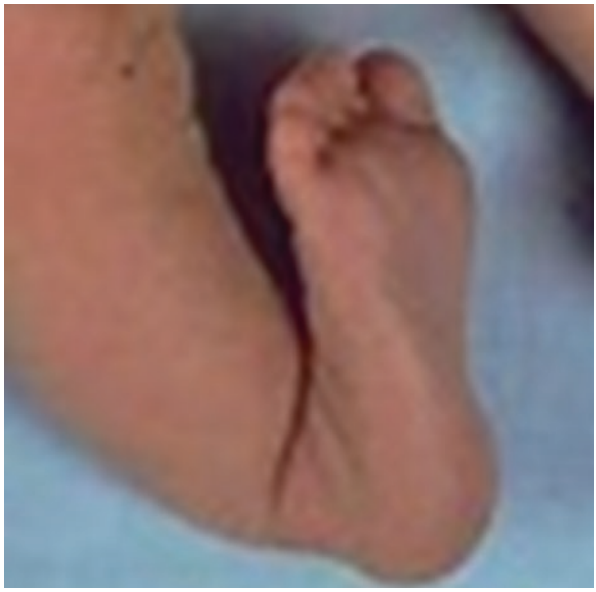

Fig. 39.3 Calcaneovalgus. Showing dorsum of the foot flat almost touching the anterior aspect of the tibia

Children are born with flexible flatfeet and develop the medial longitudinal arch during the first decade of life. The development of the medial longitudinal arch is a combination of osseous, ligamentous, and neuromuscular interplay, and inappropriate or pathological processes in each of these have theorized the development of flexible flatfoot. By the age of two, the medial arch has formed, and by the age of ten, the flexible flatfoot usually disappears. It is deemed a normal variant of childhood development. This condition has been found to be familial and bilateral. Unilateral flatfoot should warrant further investigation.

The majority of patients are asymptomatic and are managed with reassurance to parents and patients of its normal variant nature, as well as its resolution with time. Symptomatic patients need to be investigated to rule out underlying pathology, which may require further intervention. Commonly if the deformity is flexible, there is a complaint of planteromedial pain and occasionally sinus tarsi pain, both of which may be activity related. Orthotics can be trialed in these patients, although the improvement in this group over time may be natural progression to normal feet with maturation. Indeed, there is no strong evidence to suggest benefit with orthotics. A proportion of these patients have a flexible flatfoot with a tight Achilles heel cord (pes equinovalgus). This may benefit from physiotherapy or rarely surgery if conservative measures fail. Rigid flatfoot should bring about concern regarding tarsal coalition (Fig. 39.4a and b).

Clinical examination should demonstrate that the foot is flat on standing but an arch reconstitutes when the foot is hanging down or on tiptoes. Jack's test would reveal medial arch reconstitution (Fig. 39.5).

On standing, the hindfoot is in valgus (Fig. 39.6), and a Silfverskiold test may reveal a tight heel cord. Assess subtalar motion to confirm if the deformity is rigid.

Asymptomatic flatfeet are managed by reassurance and watchful waiting. However, painful flatfeet not responding to rest, activity modification, physiotherapy, and simple over-the-counter pain killers may need operative intervention. Releasing a tight heel cord may help (orthotics may actually cause more pain in this subset of patients). Further operative intervention may be needed in combination with soft tissue and osteotomy procedures. A medial displacement calcaneal osteotomy corrects a valgus heel. A lateral calcaneal lengthening osteotomy is also a viable alternative option. Arthroereisis has some

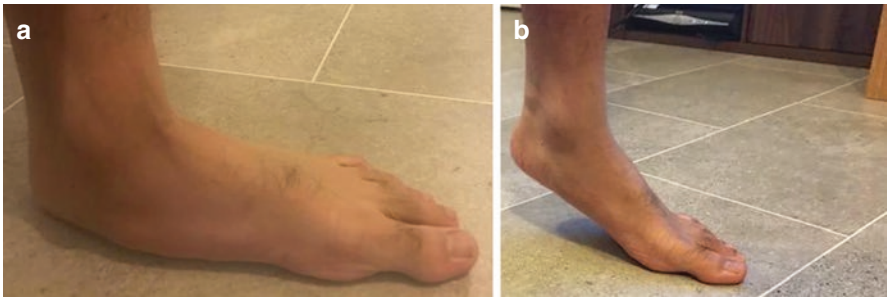

Fig. 39.4 (**a**) Showing fixed flatfoot. (**b**) Fixed flatfoot does not develop a medial arch of tiptoeing

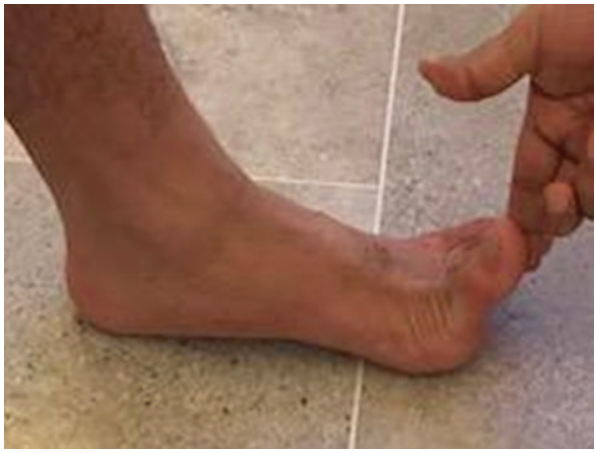

Fig. 39.5 Jack's test. Showing medical arch reconstitution on hyperextending the big toe, indicating a flexible flat foot

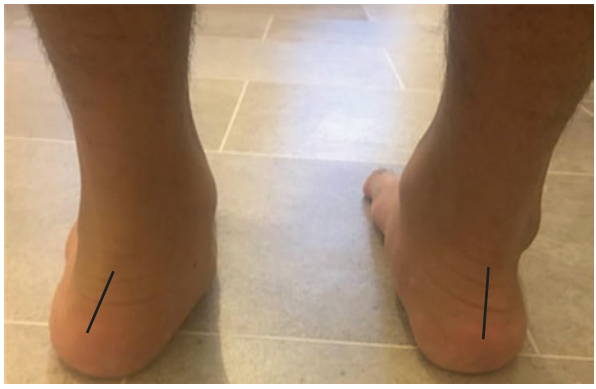

Fig. 39.6 Showing left heel valgus on standing

advocates—limiting motion by an implant in the sinus tarsi to avoid midfoot collapse [4]. Late or salvage options would include fusion.

Acquired flat foot disorders develop later in life. Examples of this include posterior tibialis tendon insufficiency (PTTI) or tarsal coalition.

Posterior Tibialis Tendon Insufficiency

Adult flatfoot deformity is most commonly caused by posterior tibialis tendon (PTT) insufficiency. This is a complex and progressive condition that commonly presents late. It mainly affects women between their fourth and sixth decades. Risk factors include obesity, diabetes, age, and inflammatory disorders. There are a proportion of patients who experience a traumatic event, but in the majority of patients, it is a degenerative process.

To understand the process, we must revisit the anatomical and biomechanical functions of the foot in relation to PTT. The muscle originates in the interosseous

membrane and adjacent tibia and fibula to become tendinous in the distal part of the calf. The tendon passes around the medial malleolus to the plantar surface of the foot. It eventually goes on to insert into multiple different parts—the navicular tuberosity and medial cuneiform, the middle three metatarsals and the plantar aspect of the remaining tarsal bones and the anterior portion of the sustentaculum tali.

The tendon is the primary dynamic support for the medial arch of the foot. Biomechanically, the tendon pull lies medial and inferior to the subtalar joint axis and posterior to the ankle joint axis. Thus, it functions to supinate and adduct the hindfoot to cause inversion and to plantar flex the ankle. During normal gait, the muscle stabilizes the foot in stance. It locks the transverse tarsal joint producing a rigid lever for toe off. There is a well-documented watershed zone of relative hypovascularity proximal to the navicular insertion, which may be a cause of degeneration and failure. This may also become pathological in patients with inflammatory disorders or those suffering from overload in obesity. The spring ligament is the primary dynamic stabilizer of the medial longitudinal arch and the talonavicular joint. It may become affected in late disease and become incompetent. It may also be involved in acute trauma. This may need to be addressed as part of operative management.

Patients present initially with medial ankle or midfoot pain in the distribution of the tibialis posterior tendon occasionally into the calf. Progressive deformity of the foot with loss of the medial longitudinal arch then occurs with lateral-sided pain due to subfibular impingement, which is a late symptom.

Physical examination is key in appreciating the progression of the disease to appropriately stage as well as to guide intervention. Inspection in early disease may be entirely normal, with only subtle swelling medially along the course of the tendon during the initial synovitis stage. Subsequently, there is progressive collapse of the medial longitudinal arch and forefoot abduction. Inspection from behind would show a valgus hindfoot with the abducted forefoot showing ‘too many toes’ when viewed from behind.

The patient is then asked to perform a single heel raise—watching for movement into eversion and reconstitution of the arch. This is not possible with an incompetent tendon and indicates middle to later stages of the disease.

The patient can be seated in the next part of the assessment. The tibialis posterior tendon is palpated along its length, and strength can be assessed by measuring resistance against inversion and plantar flexion. The tendo Achilles can be tight in the later stages of the disease as it shortens with increasing hindfoot valgus. This can be tested through the Silfverskiold test. Placing the heel into neutral alignment assesses the degree of fixed forefoot varus. The lateral side of the ankle should be palpated for signs of subfibular impingement.

The radiographic imaging series should consist of a weight-bearing AP and a lateral of the foot and a weight-bearing view of the ankle mortise. A lateral radiograph demonstrates the collapse of the medial arch with a negative tarso-first metatarsal angle (normally 0–10°) and a decreased calcaneal pitch (normal 17–32°) and a decreased medial cuneiform-floor height. An AP radiograph shows progressive uncovering of the talar head (greater than 15%). Late-stage disease will demonstrate

subtalar arthritic changes on the lateral radiograph and ankle arthritic changes and talar tilt on the AP mortise view. Magnetic resonance imaging (MRI) is the gold standard in assessing tendon pathology.

Treatment and management depend on the stage of disease at presentation with late presentation necessitating operative intervention being common. Johnson and Strom [5] classified PTT pathology, and Myerson [6] added a fourth stage. The classification system helps guide treatment.

In stage 1 disease, there is tenosynovitis without any deformity as tendon length remains the same. There may be pain on stressing the tendon and subtle swelling along the course of the tendon. The patient is able to perform a single heel raise. Immobilization in walking plaster cast or boot is first-line treatment. After a period of immobilization, conversion to custom orthosis (medial heel lift and longitudinal arch support) is required. Physiotherapy for tendon strengthening can also be started. Failure of nonoperative management would require tenosynovectomy of the tendon.

Stage 2 disease describes progression of the pathology to a flatfoot deformity with irreversible tendon elongation and dysfunction. The key to this stage is the flexibility of the deformity. The flatfoot deformity couples with a flexible hindfoot deformity. In the later parts of this stage, the forefoot becomes abducted. The patient is unable to do a single heel raise. There may be mild lateral sinus tarsi pain, as well as medial pain. Initial management, as with all later stage disease, is use of an ankle-foot orthosis, which includes a medial post to stop the collapse of the valgus hindfoot. A full length longitudinally supporting orthotic is also necessary.

Surgical intervention for stage 2 deformities involves tendon transfer of either flexor hallucis longus (FHL) or flexor digitorum longus (FDL) into the navicular to reconstruct the defunctioned PTT. The flexor digitorum longus may be preferred as it is immediately adjacent to the PTT, as well as synergistic in its line of pull and firing at similar times in the gait cycle. If an equinus contracture is present, it is corrected with a gastrocnemius recession. Spring ligament reconstruction or plication is increasingly being used by surgeons as an adjunct to treatment.

In stage 2, there are progressive deformities that need management on a case-by-case basis. Stage 2A presents with hindfoot valgus without significant forefoot abduction. After soft-tissue management, protection of the reconstruction involves bony osteotomy. This commonly is a calcaneal osteotomy with medial displacement to reconstruct the excess valgus and realign the tendo Achilles. Stage 2B disease involves significant uncoverage of the talar head and forefoot abduction. This may also require a lateral column lengthening procedure such as an Evans opening wedge osteotomy. In stage 2C disease, forefoot supination means a loss of the tripod mechanism of the foot with a relative dorsiflexion of the first ray. This can be managed with plantar flexion opening wedge osteotomy to plantar flex the first ray or with a first tarsometatarsal joint fusion.

Stage 3 disease involves rigid deformities of the hindfoot and the forefoot with degenerative changes of the subtalar joint. Accommodative orthotics are needed for the fixed deformity. Fusion is the mainstay of treatment at this stage, but this may be done on a case-by-case basis, ranging from isolated subtalar fusion to

double (talonavicular and subtalar) or triple arthrodesis. Fusion may need to be corrective in severe deformity or on rare occasions be combined with a corrective osteotomy.

Stage 4 disease involves tibiotalar degenerative disease and deltoid ligament compromise. Again with fixed deformities, fusion operations are required. All stage 3 operative interventions are available in order to correct the deformity if ankle pain and degeneration are not significant. This would be coupled with tendo Achilles lengthening and deltoid ligament reconstruction. Despite this, the disease may progress, in which case a tibiotalocalcaneal arthrodesis will be necessary.

Peroneal Flatfoot/Tarsal Coalition

The term peroneal flatfoot is a historical misnomer and is used to describe patients with a rigid flatfoot deformity. This group of patients commonly present in adolescence. Due to the rigid deformity, there is a shortening of the peroneal muscles and an eversion deformity, hence the term peroneal/spasmodic flatfoot, although this is not a neurological or neuromuscular problem. This is mostly a congenital issue, although posttraumatic/degenerative or infectious causes may be an underlying etiology. Its incidence may be underreported.

There are two main types of congenital tarsal coalition:

1. Calcaneonavicular—this is a connection between the anterior process of the calcaneus and the lateral aspect of the navicular. It commonly presents at 8–12 years old.
2. Talocalcaneal—this is a connection of varying areas between the talus and calcaneus. It may affect all or one of the calcaneal facets (commonly the middle). It presents at 12/15 years old.

Pathophysiologically, this is believed to be a disorder or a failure of mesenchymal segmentation manifesting as a coalition at birth. This is initially a fibrous coalition and may remain so. A proportion will proceed to a cartilaginous coalition (synchondrosis) or ossification to a synostosis. Patients present with rigid deformities, including flattening of the longitudinal arch, abduction of the forefoot, and a valgus hindfoot. Subtalar motion is hindered during gait.

The pathognomonic radiographic findings are as follows: calcaneonavicular (Fig. 39.7) 45° internal oblique view demonstrates the coalition.

Lateral view of the foot shows an elongated anterior process of the calcaneum or an “Ant eater nose sign” (Fig. 39.8).

Talocalcaneal—Lateral Radiograph demonstrating talar beaking and the or a “C” sign due to the medial outline of the talar dome and the posteroinferior aspect of the sustentaculum tali (Fig. 39.9).

A Harris view may show the coalition or an irregularity of the middle facet. Computed tomography (CT) and 3D reconstruction may be of benefit to further visualize the size and extent of the coalition and plan operative management. It may

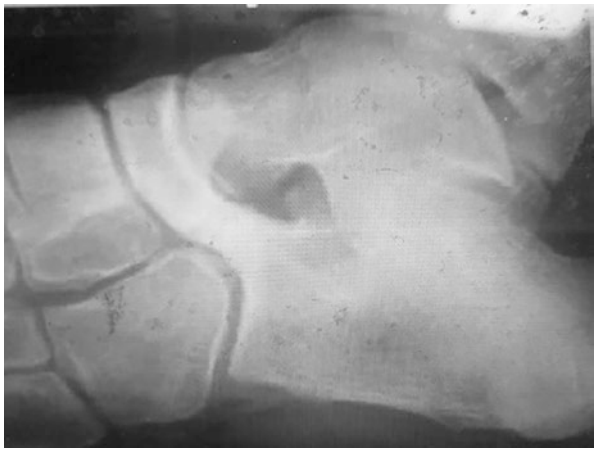

Fig. 39.7 Oblique view of the foot showing a bony bridge between the calcaneus and the navicular (calcaneonavicular coalition)

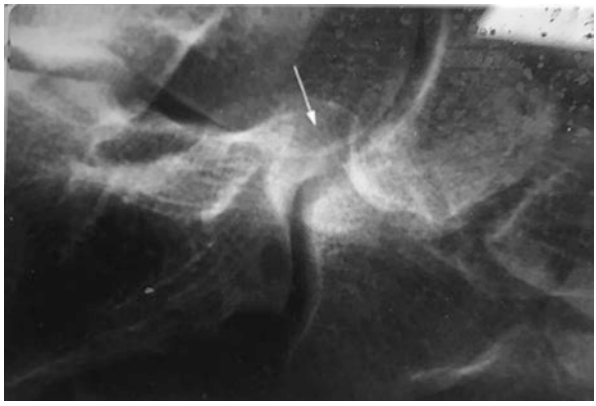

Fig. 39.8 Oblique view of the hindfoot, the arrow pointing toward the elongated process of calcaneum "Ant eater nose sign"

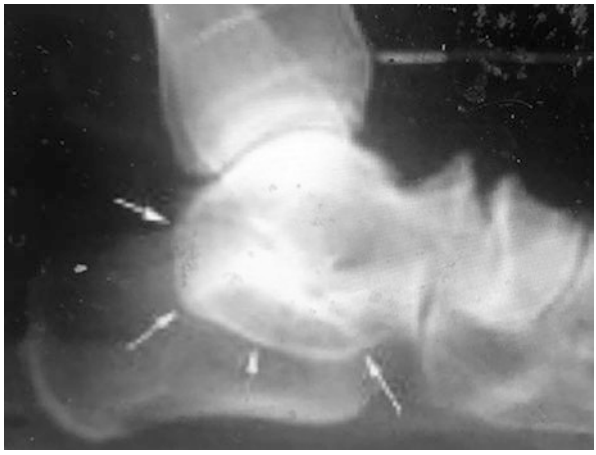

Fig. 39.9 Showing talar beaking and the "C" sign of talonavicular coalition (bony continuity of the inferomedial border of the talus with the sustentaculum tali)

also be useful in diagnostic uncertainty. MRI has recently gained popularity in 3D imaging as it demonstrates fibrous coalitions (synostosis) or cartilaginous coalitions. It also has the benefit of not exposing adolescents to ionizing radiation. Both MRI and CT may show multiple coalitions at different points in up to 20% and can be bilateral in 25–50% [7, 8].

Management of tarsal coalition is initially nonoperative. Immobilization with orthotics or casts for up to 6 weeks is successful for a proportion of patients. There is an unknown role for medial arch support of in-shoe orthotics as they may cause pain in these rigid flatfeet. Surgical management in case of failure of nonoperative management or refractory cases begins with coalition resection and interposition graft. Interposition material can be local (extensor digitorum brevis in calcaneonavicular and split flexor hallucis longus in talocalcaneal), fat graft, or bone wax. There may be a need to correct any underlying deformity commonly to correct any hindfoot valgus (calcaneal osteotomy +/− lengthening). Talocalcaneal coalitions of greater than 50% (involving the posterior facet) may do better with primary arthrodesis than with resection. Triple arthrodesis is reserved for advanced cases with associated degenerative changes or failed resections.

Miscellaneous Causes for Heel Pain

Heel pain can have multiple causes, and on occasions multiple etiologies may exist. Careful history and examination are needed to diagnose and offer appropriate treatment. In this subsection, we look at some of the common causes for heel pain and will go into greater depth in later sections come larger topics.

Plantar Fasciitis

The deep fascia of the sole, the plantar aponeurosis, is arranged in three divisions, which cover and provide partial origin to the superficial muscles of the sole. The middle division is much thicker than the others and ends distally in five digital processes. Passive hyperextension of the toes therefore renders it taut (windlass mechanism) [1]. In plantar fasciitis, inflammation of the plantar fascia, usually in the central to medial subcalcaneal region, occurs and patients present with heel pain. The nature of the lesion is obscure, and the discomfort starts with no obvious reason and is aggravated by weight-bearing. In some, it can be incapacitating to such an extent that they hobble around with the heel off the ground. Plantar fasciitis can be associated with metabolic diseases such as hypothyroidism, gout, inflammatory conditions like rheumatism, or ankylosing spondylitis. Many patients are overweight, and their work involves prolonged standing.

Clinically, apart from tenderness at the attachment of the plantar fascia to the calcaneus, the foot looks normal. The point of tenderness is just forward to the medial plantar tubercle of the calcaneum over the attachment of the central and medial plantar fascia and the flexor digitorum brevis.

A lateral radiograph of the calcaneus may show a calcaneal spur. In the absence of symptoms, the spur is left well alone.

Management

The patient should be advised that they have a chronic soft-tissue condition and that recovery may be protracted. Empirically, it appears that most cases will respond to physiotherapy, nonsteroidal anti-inflammatory drugs (NSAIDs), night splints, and local steroid injections. Static daily exercises are performed to stretch the tendo Achilles, and night splints are worn to keep the ankle in 10° of dorsiflexion and to keep the tendo Achilles stretched while in bed [9, 10]. Usually, the condition becomes tolerable in a couple of months and may take anywhere from 6 months to a year to resolve. Weight reduction in the obese is recommended, as well as treating general conditions like hypothyroidism and gout. Extracorporeal shock wave therapy (ESWT) may be of benefit [11]. Local platelet-rich plasma (PRP) injections can be tried [12].

If nonoperative measures fail, then partial central and medial plantar fasciotomy is considered. If the tendo Achilles is tight, then the gastro soleus fascia is released in the calf.

Calcaneal Stress Fracture

Although metatarsal bones are the commonest sites of stress fractures, one must consider calcaneal stress fractures in the differential for heel pain in active patients or those at risk of insufficiency. Pain worsens during weight-bearing and diminishes during rest. MRI may be needed if radiographs are inconclusive. Management involves protected weight-bearing and rest.

Subtalar Arthritis

The key in subtalar arthritis is diagnosing using a pertinent history from the patient:

1. Previous trauma to the talus or calcaneum can progress to talocalcaneal joint degeneration.
2. Progressive foot pathologies such as acquired flatfoot due to PTT pathology eventually progress to hindfoot arthritis.
3. Inflammatory arthropathies like rheumatoid arthritis could involve subtalar joint.

Nonoperative management involves orthotics to limit movement, NSAIDs, and targeted injections. Subtalar arthrodesis may be needed in some circumstances and may be done using minimally invasive techniques (arthroscopic subtalar fusion).

Sever's Disease

This condition is also known as calcaneal apophysitis and presents in adolescent boys between the ages of 10 and 14 years. It normally occurs just prior to physeal closure and is an overuse injury. Those who undertake running or jumping sports are more affected. It may be associated with tight tendo Achilles (especially gastrocnemius). This is a self-limiting condition, and management is supportive with activity modification, physiotherapy and stretching exercises, heel cups/pads, and NSAIDs. Further imaging is rarely needed but may be used if the diagnosis is doubtful. Lateral radiograph may show sclerosis or fragmentation of the calcaneal apophysis.

Bursae Around the Heel

Two bursae exist around the heel, which may become pathological and when inflamed may cause bursitis. The subcutaneous calcaneal bursa develops on the posterior heel pad and may become inflamed from rubbing against ill-fitting footwear.

The retrocalcaneal bursa is commonly inflamed in conjunction with insertional Achilles tendinopathy and is related to calcaneal Haglund deformity (best visible on a plain lateral radiograph) (Fig. 39.10).

These patients present with posterior heel pain and fullness on examination in the heel area. Pain on palpation of either side and anterior to the Achilles tendon is

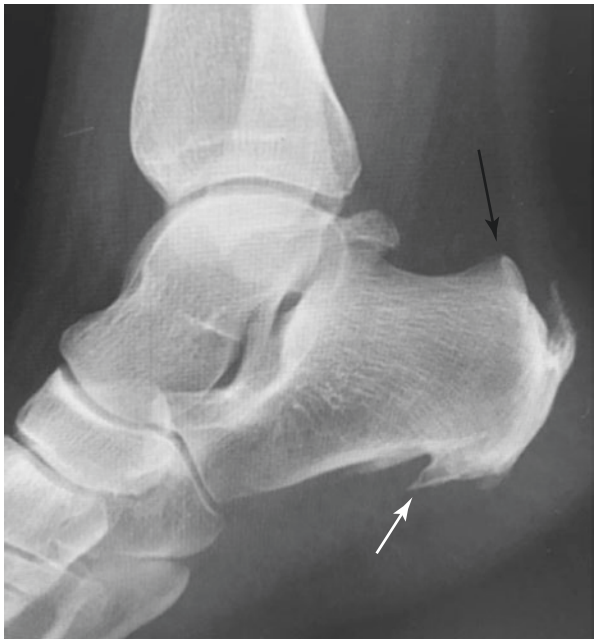

Fig. 39.10 Lateral radiograph: the black arrow points to a prominence at the posterosuperior aspect of the calcaneus (Haglund) and the white arrow to the calcaneal spur

present. Dorsiflexion of the ankle compresses the bursa inflamed by the Achilles tendon, causing pain. Nonoperative management involves NSAIDs, appropriate footwear, and possible orthotic support. There may be a role for consideration of a steroid injection under ultrasound guidance if initial conservative management fails. If associated with an insertional Achilles tendinitis, there is a risk of infiltration into the tendon and subsequent increased risk of Achilles tendon rupture. Surgical management aims to debride and remove the inflamed bursal tissue. Haglund's deformity is excised at the same time. This can all be done through a lateral approach anterior to the Achilles tendon or a direct posterior approach if managing any Achilles pathology with tendon detachment. A gastrocnemius slide should be considered, if tight.

Tendo Achilles Pathology

The tendo Achilles (TA) is a shared tendon comprising the aponeurosis confluence of the gastrocnemius and soleus muscles and the plantaris muscle. It is the largest tendon in the body, both anatomically and functionally (considering the forces that pass through it both at rest and during exercise). The tendon fibers of the soleus and the gastrocnemius rotate 90° in the Achilles tendon to allow the fibers of the gastrocnemius to insert poster laterally and the tendons of soleus to insert anteromedially. The plantaris tendon normally inserts anteromedially. The Achilles tendon inserts 2 cm distal to the posterosuperior calcaneal prominence in a crescent-like shape. When considering pathology of the TA, we differentiate between noninsertional Achilles tendinopathy (pathology 2–6 cm from the insertion) and insertional Achilles tendinitis (pathology within the last 2 cm).

Noninsertional Achilles Tendinopathy

Pathologically, this can be subdivided into inflammation of the paratenon, primary degeneration of the Achilles in the form of Achilles tendinosis due to chronic overuse or peri-tendonitis, and Achilles tendinosis, all of which are coexisting. It is frequently seen in the athletic population, especially runners or those involved in push-off sports.

Etiologically, there are both intrinsic (biomechanics, age, gender, etc.) and extrinsic factors (medication, footwear, sports, etc.). Multiple theories exist on the underlying causes, including mechanical and vascular. Mechanical injury theory suggests that physiological overload of the tendon or an accumulation of multiple overloads/microtearing with inadequate healing may occur. These would lead to tendon sheath overload (tendinitis) and the production of a degeneration spiral within the tendon itself (tendinosis). Vascular theory suggests abnormal vascularity to this are of the tendon predispose to pathology. Contradictory evidence exists with some studies demonstrating that the area is hypovascular in normal tissue but in pathological tissue there is increased blood flow.

Typically, these patients present with pain localized between 2 and 6 cm from the calcaneal insertion. Commonly, there is start-up pain during mobilization or exercise or pain at the end of exercise sessions. On examination, they can have a fusiform swelling of the Achilles tendon at this level. There is decreased range of motion due to pain with reduced plantar flexion power.

Plain weight-bearing lateral radiographs may show intrasubstance calcification and soft-tissue swelling at the level of the pain. MRI is the imaging of choice as it can demonstrate tendon thickening and disorganized tissue with intermediate high signal intensity and is useful in determining the percentage cross-section of the tendon involved.

Management initially begins with conservative strategies to manage inflammation. Rest and activity modification are augmented with eccentric strengthening exercises that show level 1 evidence of benefit. These are the mainstay of tendinopathy management. Night splints may also be of benefit, offering no load tensile stretch of the tendon. Heel lifts may be of use in these patients to reduce the tensile stretch on the pathological tendon.

Other treatments include cryotherapy, platelet-rich plasma injections to deliver growth factors and cytokines to enhance tissue repair processes, and PRP with stem cells. Extracorporeal shock wave therapy is also in use.

The aim of operative intervention is to induce mild trauma to the pathological tendon to enhance the healing response or to remove diseased tendon. Percutaneous longitudinal tenotomy may be appropriate in mild diseases. This may be augmented by stripping the anterior portion of the Achilles tendon with a large suture to reduce paratendinous adhesions. Open excision and debridement (+/− tubularization) of diseased tendon may be necessary with the flexor hallucis longus tendon being needed for transfer in patients who have a greater than 50% involvement or MRI evidence of diffuse disease.

Insertional Achilles Tendinopathy

Pain within the final 2 cm of the TA is referred to as insertional Achilles tendinopathy. Tendon degeneration occurs with loss of parallel collagen fiber structure, fatty infiltration, and loss of fiber integrity with mucoid degeneration histologically leading to tendon thickening. Mechanical overload and microtrauma are thought to be key mechanisms in the disease process. This may be different at different parts of the TA with the superficial portion of the tendon undergoing more tensile force due to the pull of gastrocnemius and soleus, while the deep portion of the tendon exhibits more compressive forces as it is impinged against the calcaneum.

Patients present with posterior heel pain and swelling. Footwear is troublesome due to compression on the pathological tendon at that level. There is swelling, which may be bony in nature to palpate. Examination demonstrates a painful range of motion with plantar flexion loss of power due to pain and pain at terminal dorsiflexion as pathological fibers are stretched.

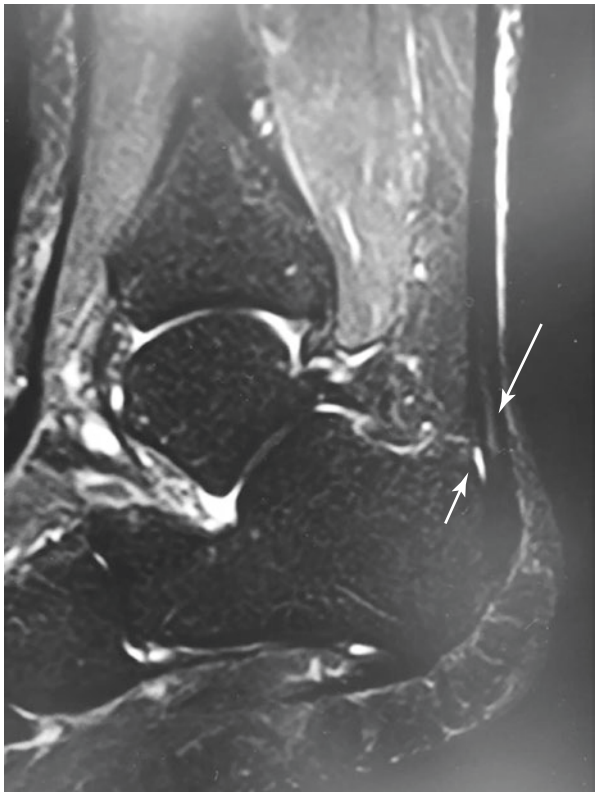

Fig. 39.11 STIR MRI images demonstrating retrocalcaneal bursitis (short arrow), along with degeneration in the tendo Achilles (long arrow), as well as Haglund prominence and calcaneal spur

Plain lateral weight-bearing radiograph imaging should be assessed for the presence of Haglund's deformity (as previously discussed). There may be calcification within the tendon and enthesophyte changes. MRI scan again is the gold standard imaging of choice to demonstrate this, as well as to signify if retrocalcaneal bursitis is occurring at the same time (Fig. 39.11).

Nonoperative management again involves eccentric exercises and progressive strengthening. This must be coupled with activity modification and rest, if appropriate. Footwear modification in the form of heel pads and heel lifts may also be used, as may night splints. ESWT has been shown to be effective at reducing pain, but intolerable pain during intervention may make this a difficult modality to adhere to and may require this to be coupled with anaesthesia. There has long been reluctance to use injections of steroid into pathological tendon for fear of promoting tendon rupture, and it is not recommended. Patients with a significant part of their pain profile arising from retrocalcaneal bursitis may benefit from focused bursal injections and from certain nonoperative management. Platelet-rich plasma has low levels of evidence showing efficacy and, along with other emerging nonoperative treatments, requires more research.

Operative treatment involves open debridement and decompression of pathological tendon. As discussed in a previous section, management of retrocalcaneal bursa and Haglund's deformity may be needed either as an initial stand-alone operation or at the

same time. The midline approach remains popular as it is extensile and allows excellent visualization of the structures. It also minimizes risk to the sural nerve and the vascular supply. A lateral approach has the benefit of not leaving the patient with a midline scar that is easily irritated by footwear. The amount of detachment of the tendon depends on the amount of pathological tendon to debride, and consideration must be taken into planned reattachment methods—suture/suture anchor and postoperative immobilization. FHL augmentation may be necessary as in noninsertional tendinopathy. Some patients may have a gastrocnemius contracture, thus necessitating gastrocnemius recession.

Calcaneal Spurs

Calcaneal spur is an outgrowth of the calcaneal tuberosity on the plantar posterior surface of the calcaneus normally arising from the medial process. It has variable anatomical associations with the plantar fascia and the muscles of the first plantar layer of the foot. It is commonly found in patients with increasing weight, elderly patients, and patients with osteoarthritis. There is also an association with patients with plantar fasciitis. Some theories exist suggesting that calcaneal spurs are an adaptive response to pathological processes and there is a relationship between heel pain and the presence of a calcaneal spur. There remain, though, patients with calcaneal spurs who exhibit no symptoms, so it is appropriate to say that they may be part of a normal spectrum/anatomical variant. Histological analysis has shown that the trabecular pattern within cadaveric spurs is vertical and perpendicular to the weight-bearing surface, which would indicate their occurrence in those who are overweight. Spurs may cause pain due to their size, fractures, and compression of the inferior calcaneal nerve, a branch of the lateral plantar nerve, and may be additive to concurrent pathologies such as plantar fasciitis. Excision is a surgical option that may be required.

Pes Cavus

Pes cavus is a broad term used to describe a foot with an abnormal high arch, which does not flatten on weight-bearing (Fig. 39.12).

It can be a sign of an underlying neurologic disorder in around 66% of cases [13]. So until otherwise proven, an underlying neuromuscular disorder has to be ruled out. Pes cavus can be present in 10% of the normal population and, in some, can be familial [14].

Biomechanics

The foot provides a stable base that helps in balance, shock absorption, load distribution and, propulsion during locomotion. The foot acts as a rigid lever in supination and a flexible coupling in pronation during various stages in the gait cycle.

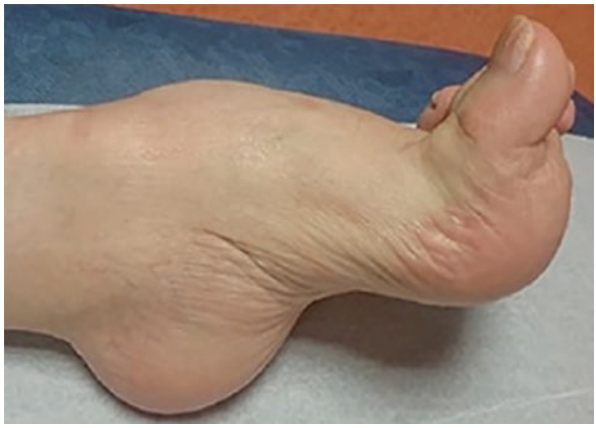

Fig. 39.12 Showing high longitudinal arch, plantar flexion of the forefoot, and cock-up deformity of the big toe

During stance, the weight-bearing stresses are transferred evenly or uniformly across the entire foot to the three points of contact (tripod), i.e., the heel, head of first metatarsal, and head of fifth metatarsal. However, in people with abnormally high arches, the size of the footprint is reduced, which increases the plantar pressure in the tripod areas for a greater proportion of the gait cycle than in normal feet, causing metatarsalgia and callus formation.

Location of the Deformity

Cavus can be located in the forefoot (anterior cavus) and posterior (hindfoot foot cavus) based on the location of the apex of the deformity or may be combined or global type [15].

Etiological Classification of Pes Cavus

1. Neuromuscular:
   (a) Muscle: dystrophies
   (b) Spinal roots and peripheral nerves: Charcot-Marie-Tooth (CMT) disease, spinal dysraphism, polyneuritis
   (c) Anterior horn cell: poliomyelitis
   (d) Central and long tract disease: Friedreich's ataxia, cerebral palsy
2. Congenital:
   (a) Idiopathic
   (b) Associated with other conditions: residual clubfoot, arthrogryposis
3. Traumatic:
   (a) Soft tissue: sequalae of compartment syndrome, burns
   (b) Bone: malunited fractures, crush injuries

Patterns of Pes Cavus

Pure pes cavus is uncommon. Here there is forefoot equinus relative to the hindfoot, which increases the height and the curvature of the medial arch. This is due to metatarsal drop (plantaris).

Pes cavo varus is more commonly seen as an abnormal high arch that is accompanied by a varus heel (medially angulated).

Equino cavo varus involves a case wherein when there is no active dorsiflexion, the ankle plantar flexes due to strong triceps surae, then the calf muscles and posterior joint capsule contract, and equinus follows.

Calcaneus varus is a dynamic deformity that is caused by the paralysis of the calf muscles, leading to excessive ankle (calcaneal) dorsiflexion and compensatory forefoot equinus (e.g., poliomyelitis).

Development of the Deformity in Neuromuscular Cavus

The development of cavus deformity is due to weakness of the tibialis anterior and peroneus brevis, overpowered by strong a peroneus longus and posterior tibialis, resulting in plantar flexion of the first ray. With the first metatarsal plantar flexed, the forefoot pronates and the medial forefoot strikes ground first. Then the subtalar joint supinates to bring the lateral forefoot to the ground. To maintain a three-point contact (Rang's tripod analogy), the hindfoot drifts into varus. Initially, the subtalar joint is flexible, and this becomes rigid with time. Recruiting extensor hallucis longus (EHL) and extensor digitorum longus (EDL) as secondary ankle dorsiflexors will lead to cock-up deformity of the hallux and claw deformity of the lesser toes due to weakness of the interossei.

Diagnosis

An accurate diagnosis is important because the underlying cause of cavus foot largely determines its future course. If the high arch is due to a neurologic disorder or other medical condition, it is likely to progressively worsen.

Clinical Presentation

Patients with pes cavus present with recurrent ankle sprains and lateral ankle pain and fifth metatarsal stress fractures due to excessive weight-bearing by the lateral foot (lateral overload). In addition, they have painful plantar calluses under metatarsal heads and plantar fasciitis.

Clinical Examination

The foot is characterized as follows:

1. Forefoot: clawing of toes and cock-up deformity of the big toe
2. Midfoot: high arch with midfoot pain and tenderness
3. Hindfoot: varus heel (initially flexible and fixed in late stages), tight gastrocnemius

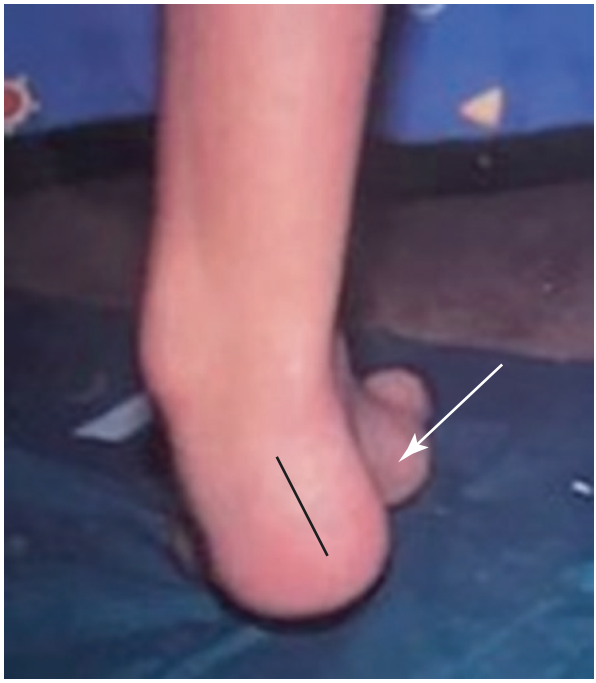

Fig. 39.13 Photograph showing the heel in varus (black line) and the white arrow pointing toward the cavus deformity of the foot

Coleman's block test [16] determines whether the hindfoot varus is forefoot driven and also establishes if the subtalar joint is mobile (Fig. 39.13).

Patient is made to stand on a raised block. If the hindfoot varus is corrected by shifting the big toe off the block (taking the first metatarsal out of the equation), then the subtalar joint is mobile (Fig. 39.14).

Peek-a-boo heel: looking from the front in the standing position shows the varus heel "peeking" around the medial ankle. Positive Silfverskiold test indicates tight gastrocnemius.

Gait is altered due to unstable base of support, increased double limb stance, and decreased single limb stance. Examination of the hands and spine may show wasting of the first dorsal interosseous muscle and scoliosis (especially in cases of CMT).

Imaging Studies

Plain Lateral weight-bearing radiographs are a key assessment as the dorsoplantar view can appear normal.

Calcaneal inclination angle (calcaneal pitch) is more than 30°.

Lateral talo-first metatarsal angle (Meary's angle) >5° with dorsal apex with break in Meary's line caused by plantar flexion of the first ray.

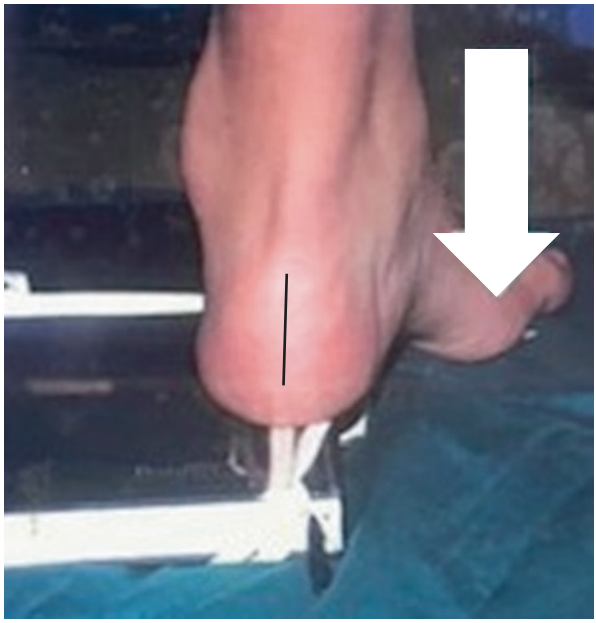

Fig. 39.14 Coleman block test showing flexible cavovarus. When the patient is made to stand on a raised block and the big toe is shifted off the block (arrow), the varus hindfoot (black line) corrects indicating a mobile subtalar joint

Cavus angle—angle of longitudinal arch is less than 150°—is formed between the calcaneal inclination axis and a line drawn along the inferior edge of the fifth metatarsal.

Management

The aim of management is to relieve pain, correct deformity, and prevent recurrence.

Nonoperative Management

Accommodative footwear includes shoes that would accommodate the high-arched foot with a wide toe box to prevent pressure over the dorsum of the toes, lateral heel wedge to prevent heel varus, and a cutout under the first metatarsal head to prevent early loading of the metatarsal. In cases of significant muscle weakness, an AFO (ankle-foot orthosis) may be needed to balance muscle forces.

Operative Management

Due to the progressive nature of the condition, surgical management is preferred to ankle foot orthosis.

Soft-Tissue Procedures

Cavus deformity: plantar fascia release (Steindler's stripping releases short flexors of calcaneus) is the first procedure in the surgical management of pes cavus, along with other soft-tissue or bony procedures. Tendon transfers for plantar-flexed first ray to balance the muscle forces. For example, transfer of the peroneus longus to brevis decreases plantar flexion force on first ray without weakening eversion. For

cock-up deformity of the big toe, EHL is transferred to the neck of the first metatarsal (Jones transfer) with fusion or tenodesis of the interphalangeal joint of the big toe.

Key Points on Pes Cavus

- Pes cavus can be familial in 10% of cases.
- Rule out underlying neurological condition.
- If unilateral, then rule out spinal cord tumor/dysraphism.
- Idiopathic pes cavus is not associated with any muscle imbalance and is not progressive.
- Accommodative footwear is recommended in idiopathic/nonprogressive cases.

Congenital Talipes Equinovarus (CTEV) (Clubfoot)

CTEV, aka clubfoot, is a congenital deformity in which an infant's foot is turned inward, and in severe cases, the sole of the foot faces sideward or even upward (Fig. 39.15). Although diagnosed at birth, many cases are first detected during perinatal ultrasound. Clubfoot is more common in males. Boys are twice more affected

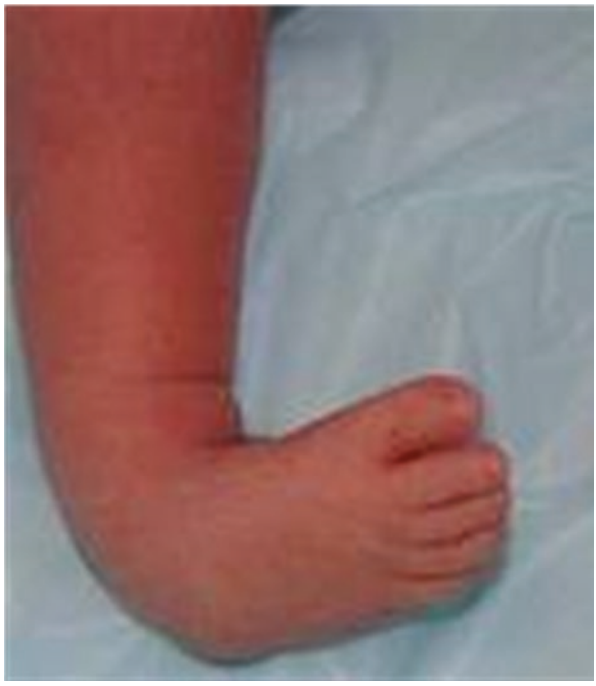

Fig. 39.15 Photograph of CTEV showing forefoot adduction and supinated, hindfoot varus, and equinus

than girls, and half of the cases are bilateral. The right foot is affected more than the left. Congenital talipes equinovarus or clubfoot deformity occurs in 1–2/1000 births.

Etiology

The etiology is unknown, but a few theories have been proposed:

1. Mechanical theory: it is one of the packaging disorders; e.g., oligohydramnios forces the foot into a deformed posture in the uterus. As the fetal position of presentation is left occipito-anterior, the right foot is more commonly forced into a deformed position.
2. Neuromuscular theory: it is due to neuromuscular imbalance, and the bony deformity is merely secondary.
3. Germ plasm defect theory: the talus never develops into normal size and shape, and all other deformities are secondary to the talar deformity.

Pathologic Anatomy

Clubfoot deformity is due to the talar neck being deviated medially and plantarward with medial rotation of the calcaneus and medial displacement of the navicular and the cuboid. The posterior and medial soft-tissue structures, i.e., Achilles tendon, tibialis posterior, flexor hallucis longus, flexor digitorum longus, joint capsules, ligaments, and fascia, are contracted. There is atrophy of the calf muscles, and the foot on the affected size is smaller.

Clinically

The mnemonic "CAVE" identifies the major findings of **C**avus, forefoot **A**dduction and supinated, hindfoot **V**arus, and **E**quinus. Clubfoot can be mild to severe, with the more severe (complex or atypical) forms being associated with other conditions (e.g., arthrogryposis, diastrophic dwarfism, prune belly, tibial hemimelia, myelomeningocele, and those derived from improper casting methods).

Management

The treatment of CTEV has evolved with time. It is divided into two main approaches: conservative and surgical.

Conservative technique primarily achieves the correction of clubfoot by slowly stretching the tight structures (viscoelasticity), allowing time for soft tissue and cartilaginous remodeling. This is achieved by serial casting.

Casting usually is started early (or until the foot reaches a length of at least 8 cm) but not later than the first month of life to make casting easier. In premature babies,

treatment is deferred for several weeks until the infant is stabilized and the foot has grown. There are numerous manipulation techniques used in CTEV (Ponseti, Kite, and French methods).

Dr. Ponseti, an orthopedic surgeon at Iowa university, in the 1940s described his "Ponseti" method following extensive anatomical studies and understanding of the flexibility of the foot, which has become popular since the 1990s. His technique involves a very specific series of short and gentle manipulation (to stretch the soft-tissue structures) and casting (to maintain the amount of correction achieved). The principles based in manipulative correction in the Ponseti method can be considered as the creep; on the other hand, the cast immobilization and the FAO wearing can be considered as stress relaxation [17–19].

In serial casting, in the first cast, the first metatarsal must be raised, which means supinating the forefoot to align the forefoot with the hindfoot and to decrease the cavus. Thereafter, abducting the foot, with counterpressure on the talar head, as a fulcrum corrects forefoot adduction and the hindfoot varus simultaneously. The calcaneus should not be touched as this might block the motion of the calcaneus, which must be free to swing out from underneath the talus and thereby to abduct, evert, and dorsiflex ("Kite's error" of manipulation, as mentioned by Ponseti, is applying counterpressure over the calcaneocuboid joint and correcting the deformities step by step, i.e., forefoot varus first, inverted hindfoot second, and the ankle equinus last rather than simultaneous correction of the hindfoot varus, inversion, and adduction). The foot must be abducted to 60–70° before correcting the equinus. Attempts to correct equinus before heel varus and foot supination are corrected will result in rocker bottom deformity.

Percutaneous Achilles Tenotomy (PAT)

Ponseti recommended tenotomy when dorsiflexion was less than 15–20°, after foot had been abducted to at least 60°. The prerequisites are as follows: talar head must be covered, the heel should be in slight valgus, and it should be possible to abduct the foot to 60°. He recommended performing PAT under local anesthesia, which can be performed as an outpatient procedure and is safe and effective. Tenotomies are usually performed between 4 and 12 weeks, the average being around 9 weeks.

Light sedation is a safe alternative to general anesthesia (GA) and is recommended when treating older infants who might struggle with local anesthesia leading to an incomplete tenotomy. PAT is followed by a further 3 weeks in a cast. After the deformity is fully corrected, next is a Denis Browne splint or a foot abduction brace, which consists of a bar equal to the length of the child's shoulders with shoes at both ends. These shoes are externally rotated to 70° (on the CTEV side) and 40° (on the normal foot, in unilateral clubfeet) with 10–15° of dorsiflexion. The brace is worn full time for 3 months and then at night and during nap times (at least 10 h a day) for 4 years.

Common Errors (Red Flags) in Manipulation Techniques Leading to Complex Deformities

Incorrect counterpressure on the talus (either too low or on a wider area). The applied abduction force applied then acts on the mid foot joints, resulting in a spurious correction.

Improperly applied cast: cast application is a skilled process. Ponseti recommended a thin cast from the groin with the knee in at least 90° of flexion and with the cast molded well behind the knee and around the heel.

Accelerated Ponseti is when the casts are changed every 5 days (rather than weekly) [20].

When Ponseti's method is used correctly, there is a 98% chance of achieving full correction. Thus, this method has become the treatment of choice for clubfoot and is now a gold standard in the management of clubfoot. Pirani's score gives a significant correlation between initial severity of the foot and outcomes [21].

Other manipulative methods described that are less commonly used are Kite and French methods.

Refractory or Resistant Clubfeet

Surgery is favored in resistant feet, usually at 6–9 months. The principle of surgery is to align the talonavicular joint and the calcaneus. Surgical release of tight structures follows an "a la Carte" approach where tight structures are released systematically. This could include Z-plasty of the Achilles tendon; fractional lengthening of FDL, FHL, or section of the tibialis posterior and abductor hallucis; posterior capsulotomies of the posterior tibiotalar joint, talocalcaneal joint, and talonavicular joint; and resection of the fibulocalcaneal ligament. The soft-tissue release is followed by casting for several months postoperatively [22, 23].

Relapse

Signs of a relapse include tightness of the heel, loss of dorsiflexion, toe walking, and forefoot turning in.

Ponseti method, followed by tibialis anterior transfer and/or tendo Achilles tenotomy or lengthening when indicated, has been successful in older children with recurrent deformity or prior surgery [24].

Other methods may be needed in treating a relapse; depending on the age of the child (3–10 years), methods like osteotomies, cuboid decancellation, wedge tarsectomy, and metatarsal osteotomy should be carefully planned. Triple arthrodesis is used for recurrent clubfoot salvage surgeries after 12 years to relieve pain and correct deformity. For neglected clubfoot, salvage procedures like triple arthrodesis/talectomy may be considered.

Key Points
- Clubfoot is a common congenital deformity that affects one in 100 live births.
- Most are idiopathic and not associated with other conditions.
- Babies should be referred early for treatment.
- The current best treatment is serial casting and bracing according to the Ponseti method.
- Results are better with manipulative methods than with surgical release.
- Recurrences can occur and are normally caused by noncompliance with bracing.

Painful Conditions of the Ankle

Sprains of Ankle Joint

Ankle sprains occur following a sudden sideways or twisting movement of the foot. An ankle sprain is an injury to the ligaments that support the ankle. When an ankle sprain happens, the ligament is stretched too far and is either partially or completely torn. The injury is common in athletes but also occur in routine daily activities such as stepping off a curb or slipping on ice.

Differentiating between a sprained ankle and an ankle fracture can be difficult, and sometimes radiographs are needed. Ottawa rules indicate radiographs only in patients if there is any pain in the malleolar zone and bony tenderness at the posterior edge of the tip of lateral malleolus, bone tenderness at the posterior edge or tip of the medial malleolus, or inability to bear weight both immediately and in the emergency department for four steps [25].

There are two broad categories of ankle sprain:

1. **Inversion ankle sprain**: the most common type of ankle sprain (90%) occurs when the foot is inverted, twisting inward. In this type of ankle sprain, the lateral ligaments are stretched too far and commonly involve the anterior talofibular ligament (ATFL) and occasionally the calcaneofibular ligament (CFL). The posterior talofibular ligament is rarely involved.
2. **Eversion ankle sprain**: the other type of sprained ankle is called an eversion injury, where the foot is twisted outward. When this occurs, the medial deltoid ligament is stretched too far. Patients will have pain on the medial side of the ankle joint.

High Ankle Sprain (Syndesmotic Injury)

These injuries have a more complex recovery as they involve the ligaments above the ankle connecting the tibia and the fibula (syndesmosis), as well as on the side of the joint. Patients suffering from high ankle sprain require recovery periods of almost twice those for patients with common ankle sprains.

Grades of Sprain

The degree of symptoms tends to correlate well with the severity of ligament injury and the prognosis for recovery:

A. **Grade I ankle sprain** causes stretching of the ligament. The symptoms tend to be limited to pain and swelling. Most patients can walk without crutches but may not be able to jog or jump.
B. **Grade II ankle sprain** involves a more severe partial tearing of the ligament. There is usually more significant swelling and bruising caused by bleeding under the skin. Patients usually have pain with walking but can take a few steps (Fig. 39.16).
C. **Grade III ankle sprain** is a complete tear of the ligaments. The ankle is usually quite painful, and walking can be difficult. Patients may complain of instability or a giving-way sensation in the ankle joint. This bruising will move down the foot toward the toes in the days after the ankle sprain because of gravity pulling the blood downward into the foot.

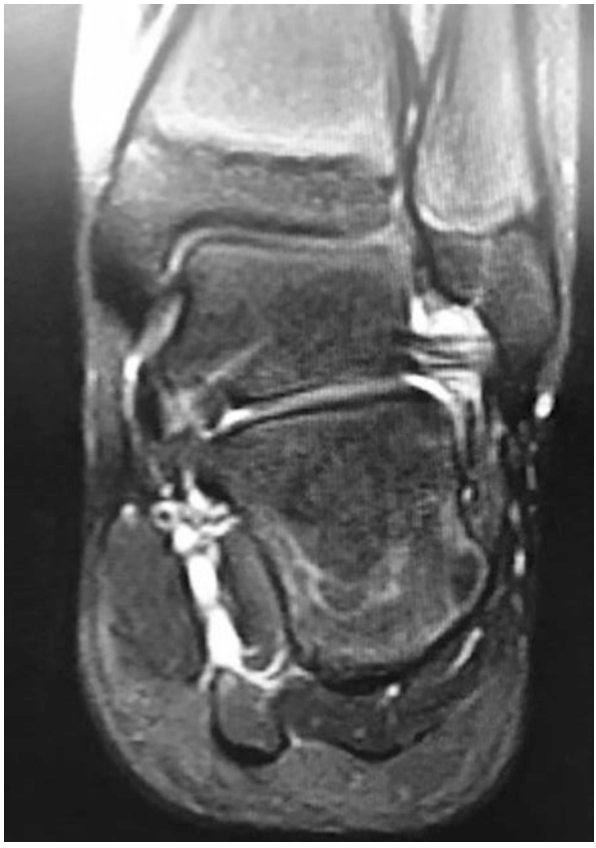

Fig. 39.16 Showing grade 2 sprain, the arrow pointing to the posterior talofibular ligament with peri-ligamentous edema

Treatment of sprained ankles is important because returning to normal activities in a timely manner is important for most patients. Rest, ice, compression, and elevation (RICE) treatments are important but should quickly progress to rehabilitation and strengthening.

Surgical treatment is reserved for recurrent symptomatic ankle instability with excessive tilt and a positive anterior drawer sign on exaination/postive stress radiographs. Anatomic procedures (modified Brostrom procedure) are usually successful.

Traumatic Arthritis of the Ankle (Anterior Impingement, Footballer's Ankle)

Anterior ankle impingement is caused by repeated strain on the anterior capsule due to repeated holding and kicking the ball onto the dorsum of the foot in plantar flexion. The classic form of impingement is referred to as "footballer's ankle." Despite the name, this can happen in many different types of sports, including soccer, football, and basketball, and in dancers. Soft tissues get entrapped between exostoses or calcified spurs, which form on the anterior lip of the tibia and the neck of the talus from repetitive traumatic avulsions of the anterior ankle capsule. The impingement incites inflammation of the surrounding synovial and capsule tissues, limiting range of motion and causing pain.

Patients present with pain and decrease in overall ankle range of motion, mostly affecting dorsiflexion, and in some an effusion may be present. Radiographs show spurs arising from the anterior distal tibia or the talus. Occasionally, an MRI is also utilized to evaluate other structures of the ankle. Management includes physiotherapy to help break down scar tissues and improve range of motion and anti-inflammatory medications to relieve pain and swelling. Steroid injection can be tried for inflammation. If patients present with effusion, then ankle immobilization may be necessary for 3 weeks. If conservative measures fail, then either arthroscopy or open surgery is performed to remove the bony spurs or exostoses that are causing the impingement.

Rupture of Tendo Achilles

Introduction

The Achilles tendon is the largest, strongest, and thickest tendon in the body, connecting the gastrocnemius, soleus, and plantaris to the calcaneus. It is approximately 15 cm long and begins near the middle portion of the calf. Contraction of the gastrosoleus causes plantar flexion of the ankle, enabling such activities as walking, jumping, and running. The Achilles tendon receives its blood supply from its musculotendinous junction and its innervation from the tibial nerve. Occasionally, the tear may be partial and usually occurs where the tendon joins the calf muscle. This

injury is managed slightly differently and usually involves resting the ankle in a boot for a few weeks. Most common site of rupture is around 2–6 cm above its insertion due to poor vascularity in this region.

Causes

The incidence of Achilles rupture is 7 out of 100,000 in the general population and 12 out of 100,000 in competitive athletes [26]. It can occur at any age but is most common in 30–50 years with a male-to-female ratio of nearly 20:1. Activities that cause forceful push off (strong contraction of the calf muscles) or lunging type movement with sudden hyperdorsiflexion can stress the Achilles tendon excessively, causing rupture. Other mechanisms by which the Achilles can be torn involve sudden direct trauma to the tendon or sudden activation of the Achilles after atrophy from prolonged periods of inactivity. Fluoroquinolone antibiotics, e.g., ciprofloxacin; previous steroid injections; chronic Achilles tendonitis; rheumatological disease; and kidney disease are known to increase the risk of tendon rupture.

Symptoms

When a rupture of the Achilles tendon occurs, one experiences a sudden pain in the heel or calf, sometimes associated with a snapping or popping sound. Patients often describe the feeling as if someone has hit them or has shot them in the back of the leg, only to turn around and find that no one is there. After rupture, there may be some swelling and bruising, and it is usually difficult to walk as it is difficult to push off the ground properly on the affected side. A partial tear of the Achilles tendon is rare, so any acute injury to the Achilles tendon should be assumed to be a complete rupture.

Diagnosis

It is usually possible to feel a gap in the tendon, usually 4–5 cm above its calcaneal insertion. The tear can occur higher up, about 10 cm above the insertion into the heel, at the site where the muscles join the tendon; this is known as a musculotendinous tear.

Special tests: Thompson (Simmonds) test, which is a calf squeeze test, will be performed. Normally if the Achilles tendon is intact, this causes the foot to point downwards, but if it is ruptured, it causes no movement. Ultrasound scan helps to confirm the diagnosis and the exact site of the rupture.

Treatment options: there are two treatment options available for Achilles tendon ruptures—nonoperative (conservative) and operative (surgical).

Nonoperative management (functional bracing), as evidence suggests, has similar results to surgery without the associated complications. Occasionally, surgery may be considered, especially in cases of delayed presentation or atypical ruptures.

The principle of functional bracing is the use of a specialized boot to keep the ankle in a fixed position while allowing the tendon to heal and the patient to try to

function normally. There is a set regime that involves initially being in a below-knee plaster cast with the foot in maximum equinus position while the patient can briefly bear weight on the toes for a couple of weeks. This then changed to a specialist boot (e.g., Vacoped), which is a below-knee boot with the ankle in 30° of equinus with Velcro straps. The boot should be worn at all times, including in bed, to ensure that your tendon is protected throughout the healing process. During the weeks following the injury, the boot will be gradually adjusted to allow the degree of equinus to be corrected from 30° (full) to 15° (mid) and 0° (neutral). Full weight-bearing is commenced at 3 weeks, and the boot is removed at 8 weeks.

As the calf pump is not effective and the patients are at a high risk for venous thromboembolism (VTE), thromboprophylaxis is advised for about 6 weeks from the injury. The total treatment time will be approximately 9 weeks. Following this, a flat shoe with a single heel raise is worn for 4 weeks.

The risks of conservative treatment include risk of rerupture (8%), decreased tendon strength, risk of clot in leg veins (deep vein thrombosis)/lungs (pulmonary embolus).

Surgical Treatment

This is not usually the preferred treatment option as the risks of complications may outweigh the benefits. Surgery may allow earlier weight-bearing with reduced rerupture rate (4%), but wound-healing problems and sural nerve injury are potential risks. However, surgery may be considered for certain patient presentations; delayed presentation (more than 2–3 weeks following injury), reruptures of the Achilles tendon with tendon gap and elite athletes for (some evidence of slightly increased push off strength).

There are two different types of surgeries: open surgery and percutaneous surgery.

During an open surgery, an incision is made in the back of the leg and the Achilles tendon is repaired. There are different techniques, and some surgeons harvest the plantaris, which is spread out and wrapped around the Achilles tendon repair, preventing adhesions and adding to the strength of the repaired tendon.

In percutaneous or minimally invasive surgery, the surgeon makes several small incisions, rather than one large incision, and sews the tendon back together through small incisions. Surgery may be delayed for about a week after the rupture to let the swelling go down. For sedentary patients and those who have vasculopathy or risks for poor healing, percutaneous surgical repair may be a better treatment choice than open surgical repair.

Chronic ruptures of the Achilles tendon are those that present 6 weeks from the injury. These are managed with open repair if the gap between the two ends are less than 2.5 cm. If the gap is more than 2.5 cm, then the surgical options are V-Y plasty of the proximal part of the tendon, turndown flap of the aponeurosis, use of peroneus brevis, flexor hallucis longus, or fresh-frozen allografts.

Outcome

The general outcome of Achilles tendon rupture is good. Kotnis et al. [27] elected to manage conservatively only those patients whose gap in full equinus was less than 5 mm. All others were managed operatively. They observed no statistically significant difference in rerupture rates between the groups. The tendon usually heals around 2 months or more and will take several more months to regain strength and flexibility. If patients have more physical work, then it may take 3–4 months to return to work. Driving a manual car should be avoided for at least 9 weeks following your injury. Return to sport is between 4 and 12 months, depending on the sport that the patient wishes to return to and depending on one's strength and ability to perform the necessary skills.

Fusion of the Ankle Joint

The goal of ankle fusion (also commonly known as ankle arthrodesis) is to relieve pain and improve function for a patient with painful end-stage ankle arthritis. Asymmetrical joint surfaces along with severe pain, swelling, instability, and stiffness due to various causes, i.e., trauma, infection, inflammatory conditions, and osteochondral defects. Advanced or end-stage ankle arthritis is degeneration of the articular cartilage of the lower tibia and the talus with exposed underlying subchondral bone and osteophytes formation. Pain typically is made worse with movements of the arthritic ankle. The goal of arthrodesis is to fuse the tibiotalar joint into one bone, which eliminates joint movements and reduces pain from the arthritic joint.

Indications

Patients who have advanced ankle arthritis that is symptomatic and who have exhausted all other options, like activity modification, analgesics, anti-inflammatories, corticosteroid injections, walking aids, special ankle braces, or ankle foot orthoses to stabilize the ankle and restrict its movements, and have failed arthroscopic ankle debridement may be a candidate for ankle arthrodesis.

Relative contraindications are insufficient quantity or quality of bone for fusion, poor blood supply of the ankle, impaired nerve function, medical conditions that increase the risk of anaesthesia, and severe limb deformity.

Surgical Techniques

Ankle arthrodesis can be done either arthroscopically or by an open technique. Two important factors for successful fusion are as follows:

1. The position of the foot in relation to the leg
2. The method of fusion

The ankle joint is responsible for the majority of plantar flexion and dorsiflexion movements. Ankle fusion decreases this movement, but the movement of the subtalar joint and the other joints of the foot remains unaffected. If the patient's ankle is fixed at 90°, then some transverse tarsal movements will provide an average of 10° of plantar flexion. In women, the ankle is held in 100° to accommodate for a heel.

With regard to the method of obtaining sound fusion, arthroscopic techniques involve making small incisions around the ankle, and any remaining cartilage within the ankle joint is removed so that there is good apposition of the bony surfaces of the tibia and the talus. The ankle is held in the most functional position with percutaneous screws, which allow the bones to fuse together.

Open Techniques

Many open techniques have been described either by the lateral or anterior approach. In the lateral approach to the ankle, the distal fibula is excised and the tibial and talar surfaces are cleared of cartilage remnants to expose the underlying bone. Tibio-talar contact surfaces are held with two screws passed obliquely from the tibia to the talus. Bone grafts may be used to aid fusion. In the anterior approach, the joint is approached from the front, and the tibio-talar surfaces are prepared, which are then compressed using screws, plates, or staples.

Postoperatively, patients are kept non-weight-bearing in a cast for 6 weeks, followed by partial weight-bearing in a walking boot. After 10–12 weeks, the ankle fusion is typically sturdy enough to allow walking out of the plastic boot and a gradual return to more vigorous activity.

Complications

Apart from the general complications of any surgery the (the risks associated with anesthesia, infection, damage to nerves and blood vessels, and bleeding or blood clots), a specific risk associated with ankle arthrodesis is nonunion (10%). Loss of motion in the ankle after a fusion causes premature wear and tear in the subtalar and other joints of the foot as these joints have to compensate for the ankle fusion. However, the literature suggests no difference in fusion times for open and arthroscopic fusion; wound complications and return to function are quicker in the latter [28].

Peroneal Tenosynovitis

The peroneal tendons act by everting and plantar flexing the foot. The peroneal tendons also provide lateral stability to the ankle during weight-bearing and protects it from sprains. They also help to pronate and stabilize the arch when walking. Peroneal tendinitis is related to the pulley action of the lateral malleolus on the peroneal tendons. The peroneal tendons and their tendon sheaths become inflamed due to various conditions like overuse, faulty footwear, inappropriate training methods, and trauma. Peroneal tendonitis is particularly common in athletes and especially runners as they are more likely to roll their feet outward,

causing friction between the tendon and bone. Other risk factors are pes cavus and muscle imbalance.

Peroneal tendonitis can be either acute, meaning it comes on suddenly, or chronic, i.e., it develops over time. Patients present with pain in the retrofibular region with tenderness over the course of the tendons. Stressing the tendon against resistance reproduces the pain overlying the tendons. Other presentations could be pain at the back of the ankle, pain that worsens during activity and lessens during rest, swelling at the back of the ankle, and instability of the ankle when bearing weight.

MRI or sonography can assist in the diagnosis of tenosynovitis, focal degeneration, and attenuation of tendons.

Treatment

Nonoperative treatment includes a rest for the foot and ankle, such as a boot or support. Anti-inflammatory medication such as ibuprofen can help relieve pain and swelling. Physiotherapy in the form of ultrasound therapy can reduce pain and swelling. and once symptoms improve, exercises that strengthen the muscles and improve proprioception can be performed. A brace for use during activities that involve repetitive ankle motion can be tried. If symptoms persist, then a local corticosteroid injection can be considered. Surgery (peroneal tenosynovectomy) is rare when treating peroneal tenosynovitis, and it is only usually considered as a last resort if nonsurgical methods fail in reducing pain.

Dislocation of Peroneal Tendons

The peroneal tendons (peroneus longus (PL) and peroneus brevis (PB)) are located on the lateral side of the leg, and their tendons are enclosed in a fibrous osseous tunnel that runs behind the lateral malleolus. The retrofibular groove is a shallow sulcus within which the peroneus brevis lies. Although PL shares the same synovial sheath with PB, it does not contact the sulcus and distally separates into its own sheaths. A tough connective tissue known as the superior peroneal retinaculum holds the peroneal tendons in place.

Traumatic Subluxation

Activities involving forced dorsiflexion inversion mechanism resulting in powerful reflex muscular contraction of the peroneal tendons results in injury to the superior peroneal retinaculum (e.g., skiing, dancing, playing basketball, playing soccer, ice skating), then the peroneal tendons move out of their place and slip over the lateral malleolus on the lateral aspect of the ankle. This condition is known as peroneal

tendon subluxation or dislocation. The peroneal tendons continue to remain in the dislocated position, or they may return to their previous position on their own. Diagnosis is confirmed by observing the subluxation or dislocation of the peroneal tendons during eversion and dorsiflexion. The superior retinaculum may be avulsed with a fleck fracture at its insertion along the lateral border of the lateral malleolus [29]. Ankle instability usually accompanies subluxation, and the ankle mortise should be checked. Acute traumatic subluxation does require a non-weight-bearing cast held in 90° for at least 4 weeks. Inadequate treatment results in chronic subluxation.

Chronic subluxation of the tendon will be accompanied by an audible click or pop as the tendon displaces and relocates into the grove. Treatment option includes repair of superior peroneal retinaculum, posterior fibular groove reconstruction using periosteal flap, rerouting the tendon below the calcaneofibular ligament, and bone block reconstruction procedures [29].

Rupture of Plantaris Tendon

The plantaris muscle is the smallest component of the tendo Achilles complex. The plantaris muscle and tendon sit roughly in the center of the calf, between the two heads of the gastrocnemius.

Around 10–20% of the population is born without plantaris muscles, but this does not affect mobility, either in the long or short term. Injuries to the plantaris muscle can either be a muscle strain or, more commonly, a plantaris muscle rupture. Plantaris muscle ruptures have also been called “tennis leg” as this injury is seen in athletes who are lunging forward, such as tennis players. Typical symptoms of a plantaris muscle rupture include sudden pain in the back of the calf, swelling or bunching of the calf muscle, bruising in the back of the leg, and cramping and spasm sensations of the calf muscles.

Diagnosis

Plantaris muscle tears can be differentiated from an Achilles tendon tear by a Simmonds test, which shows that the foot can be pointed downward following a plantaris rupture, on squeezing the calf, which is not the case with Achilles tear. Plantaris ruptures can also be confused with deep vein thrombosis (DVT). If the diagnosis is unclear, then either an MRI or an ultrasound can be helpful, which can also look out for other possible causes of calf pain.

Treatment of a plantaris muscle injury is almost always nonsurgical. The initial treatment of a plantaris injury is with the usual RICE (rest, ice, compression, elevation) treatment. If the pain is significant, patients may require a brief time of immobilization or crutch use to allow the pain to subside. Gradual increases in mobility and strength can be obtained with the assistance of a physiotherapist. With conservative treatment, symptoms will gradually resolve over the course of several

weeks, although a full recovery may take up to 8 weeks, depending on the severity of the injury.

Peroneal Palsy Due to Hematoma

Compression of the common peroneal nerve due to a traumatic hematoma within its nerve sheath following spiral fractures of the distal part tibia or inversion sprains of the ankle usually after skiing injuries is known but rare. Traction is transmitted along the nerve, which seems to be vulnerable, from the ankle to mid-thigh, where the vasa nervorum of the common peroneal nerve rupture below the sciatic bifurcation, resulting in a hematoma between the epineurium and perineurium [30]. Persistent pain, numbness, and weakness in peroneal nerve distribution, after reduction and immobilization of an ankle injury, occurring for hours or even days after the injury should raise suspicion and should alert the surgeon to investigate either with an ultrasound or MRI.

Early exploration of the nerve and its branches with prompt evacuation of the hematoma with ligation of the ruptured vasa nervorum is followed by swift and dramatic recovery [29].

Foot Drop in Leprosy

Leprosy (Hansen's disease) is a chronic infectious tropical disease (neglected tropical disease (NTD)) caused by mycobacterium leprae that attacks the nerves but is curable. There are two main forms: lepromatous, which is progressive, and tuberculoid, which is generally self-limiting because the host had reacted defensively and successfully against the bacillus. Most patients with tuberculoid will have tender and thickened nerves, which will ultimately make them lose their function with time, which can resolve spontaneously (with years) or progress to other rare types of leprosy such as borderline or lepromatous leprosy. Neuropathy in tuberculoid leprosy is an inherent effect of the host response and is not related to the proliferation of the bacilli. In lepromatous leprosy, the hematogenous spread of the bacilli and unchecked proliferation cause nodules throughout the skin, which is widespread due to poor immune response. Here the nerve damage is slower than in tuberculoid form [31]. The first sign of leprosy is usually a patch of discolored numb skin. The disease is curable but, if left untreated, causes life-changing permanent damage to the hands, feet, and eyes, leading to paralysis, blindness, ulcers, and amputations. Penetrating ulcers are treated by total contact casts, i.e., plaster boot with a rocker [32]. The common paralytic deformities of the foot seen in leprosy patients are foot drop, inversion of the foot, and claw toes due to paralysis of the dorsiflexors, namely the tibialis anterior, and the extensors and evertors, namely the peronei. Long-standing foot drop results in secondary contractures mainly of the tendo Achilles and instability of the talonavicular joint. A foot drop deformity should be corrected by a tibialis posterior transfer before

complications occur. If the tendo Achilles is tight, then the tendon is lengthened by Z-plasty.

Conclusion

The foot and ankle is a complex conglomerate of bones (28 bones), joints (31 joints), ligaments, and fasciae, which work in unison to provide a stable, functional, and effective lever arm to the body for energy-efficient locomotion. It can be affected by a multitude of pathologies, some of which have been illustrated above. To discuss all the pathologies is outside the scope of this chapter.

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