Orthopedic Board and FRCS Examination

The Top 100 Viva Topics

Aiman Mudawi
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Shamsi Abdul Hameed
Mohd Al-Ateeq Al-Dosari
Editors

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Foreword

It is a great pleasure and honor to be asked to write the foreword for *Orthopedic Board and FRCS Examination: The Top 100 Viva Topics*. From experience, I know that books like these are difficult to write, and the editors must be congratulated on compiling an excellent text that is likely to be widely used by candidates sitting postgraduate exams worldwide.

The topics covered are all "high yield" meaning that they are those that are most importantly asked in examinations. The questions are arranged in a way that it encourages the reader to think in a logical manner. The answers are in bullet points that make them easier to understand. Most questions start with a visual prompt and include clear illustrations. Relevant and current references are also included where appropriate. It is particularly pleasing to see the way that the authors integrate clinical application into each topic. This is something that is difficult to do in written format, and the editors must again be congratulated for making this possible.

I am involved with postgraduate orthopedic examinations throughout the world, and it makes me excited to believe that this book will have a positive impact on the exam preparation and general orthopedic knowledge of surgeons in training wherever and whenever it is used!

Sheffield, UK — Fazal Ali

Preface

The FRCS (Fellowship of the Royal Colleges of Surgeons) Orthopaedic Part 2 exam stands as the ultimate challenge in the journey of an orthopedics trainee. Renowned for its formidable difficulty, it represents the culminating test of knowledge and expertise in this specialized field.

Preparation for the Part 2 exam demands not only dedication but also a strategic approach. It necessitates focused reading, meticulous planning, and a comprehensive grasp of a vast array of topics. It is with this imperative in mind that we have developed this book, tailored specifically for the final-year resident.

This resource has been carefully crafted to address the unique needs of those preparing for the FRCS Orthopaedic Part 2 exam. It acknowledges the time constraints faced by residents and the need to cover a wide range of topics in depth. Our approach draws upon the foundational knowledge found in core textbooks, as well as the invaluable insights gathered from our own experiences and the contributions of fellow authors who have attended various review courses.

It is important to clarify that this book does not aspire to serve as a definitive textbook on Orthopedics. Instead, it is designed as a targeted revision aid, specifically tailored for the viva portion of the Part 2 exam. We envision it as a companion to your in-depth reading from core and evidence-based resources, reinforcing your understanding of the topics that commonly surface during FRCS Orthopaedic examinations.

The content within these pages is a product of our collective experiences, reflecting the very topics we encountered during our own FRCS journeys. By sharing our insights and knowledge, we hope to provide you with a valuable resource that will enhance your preparedness, bolster your confidence, and facilitate your success in this challenging examination.

As you embark on this journey toward achieving your FRCS Orthopaedic Part 2 certification, consider this book not merely as a reference but as a trusted guide. We extend our best wishes to you, knowing that your dedication and our shared insights will propel you toward excellence in the field of orthopedics.

Doha, Qatar — Aiman Mudawi
Doha, Qatar — Mazhar Fuad
Doha, Qatar — Shamsi Abdul Hameed
Doha, Qatar — Mohd Al-Ateeq Al-Dosari

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Editors and Contributors

About the Editors

Aiman Mudawi, MBBS, FRCS (Tr. & Orth) is an Orthopedic Specialist at HMC (Hamad Medical Corporation) with a passion for treating orthopedic trauma cases, particularly those involving the pelvis, acetabulum, and peri-articular areas. He completed his residency and trauma fellowship in orthopedic surgery at HMC. Subsequently, he successfully achieved the Joint Surgical Colleges Fellowship in Trauma and Orthopedic Surgery (FRCS). In addition to his clinical work, Dr. Mudawi has had the privilege of contributing as both an author and coauthor to numerous research projects and abstract publications in prestigious international journals.

Mazhar Fuad, D' Ortho, MS Ortho, FRCS completed his Orthopedic residency at Calicut Medical College and subsequently pursued a fellowship in upper limb surgery in Belgium and France. He holds an FRCS (Tr & Ortho) degree from the Royal College of Edinburgh. With a strong commitment to education, he has taken on teaching responsibilities for undergraduate students and Orthopedic residents. Furthermore, he serves as a member of the teaching faculty at both Weill Cornell and Qatar University. Currently, he is dedicated to his role in the Upper Limb Unit at HMC (Hamad Medical Corporation).

Shamsi Abdul Hameed, D' Ortho, MS Ortho, FRCS is a renowned orthopedic surgeon and educator. He completed his residency at Manipal University in India and pursued fellowships in adult reconstruction at NUH Singapore's and Oxford university.

His primary interests are in arthroplasty and sports medicine, where he has made significant contributions. Dr. Hameed is also passionate about education, holding positions as an Associate Professor at Manipal University and serving as the Associate Program Director for Orthopaedics Residency at Hamad Medical Corporation in Qatar.

Currently, he is a Consultant Orthopedic Surgeon at Hamad Medical Corporation, providing exceptional patient care and contributing to the advancement of orthopedic medicine. His dedication to both patient care and education makes him a valuable figure in the field.

Mohd Al-Ateeq Al-Dosari, MD, FRCS (Tr & Orth) is a distinguished healthcare professional with expertise in orthopedics and sports medicine. He completed his Orthopedics training followed by Fellowship in Sports Medicine and Arthroplasty at the University of Alberta, Canada.

Dr. Al Ateeq is at present the director of the Bone and Joint Centre and the Head of the Orthopedics department at HMC. He is also a dedicated educator, holding faculty positions at Weill Cornell University and Qatar University.

His commitment to academic excellence is evident through his role as a member of the Scientific Committee in the *European Journal of Trauma and Orthopaedics*. He also holds the esteemed position of President of Association of Sports Medicine Qatar and at present Vice president of Arab Board Council for Orthopedic training.

In the field of orthopedics and sports medicine, Dr. Mohd. Al Ateeq is a respected figure known for his clinical and academic leadership.

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Part I

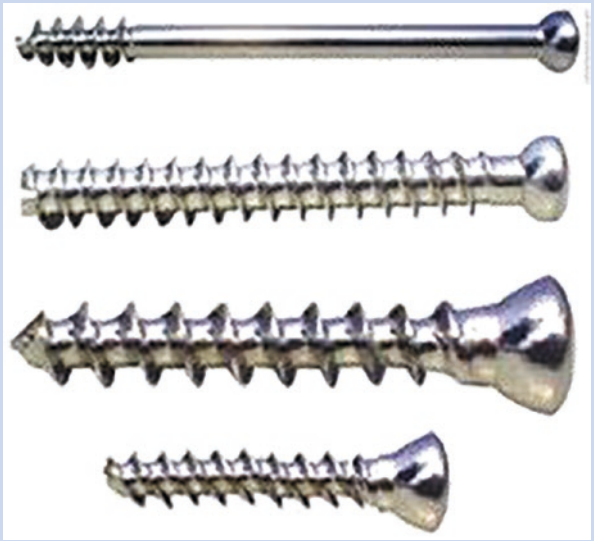
Basic Science

Applied Basic Science

1

Shamsi Abdul Hameed and Naveen Joseph Mathai

1.1 Viva 1



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- **Define a screw**
- **Describe the different parts of the screw.**
- **Describe the difference between cancellous and cortical screw.**
- **What are the factors that increase resistance to pull out?**
- **Describe lag screw principles.**

Define a screw.
A screw is a device that converts a rotational movement into a longitudinal/translational movement along the axis of movement.

Describe the different parts of the screw.
- **Head:** It serves as an attachment for the screwdriver and acts as a stop when the head comes in contact with the bone surface. It comes in various designs.
- **Countersink:** It is the undersurface of a screw head. It increases compression and can be flat/conical or threaded.
- **Shaft/shank:** It is a smooth link between the head and the threads. Screws with long shaft used as a lag screw. The smooth shaft has no purchase in the proximal hole.
- **Thread**: Compression screws may be designed partially threaded to ensure they grip only distally.
- **Tip**: Most of the screws have a cutting flute at the tip. But there are mainly three types of screws.
- **Run out:** The distance between the shank and the start of the threads. It is commonly an area of stress riser where the screw breaks.
- **Pitch:** The distance between adjacent threads (cancellous > cortical > locking).

Describe the difference between cancellous and cortical screw.

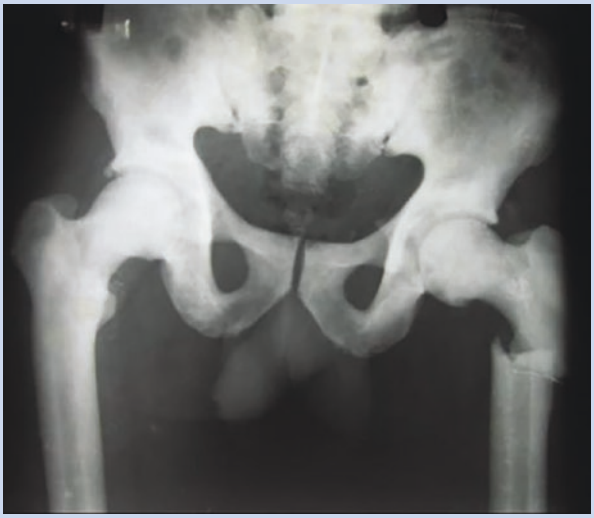
Feature	Cortical screw	Cancellous screw
Thread	Shallow	Deep
Outer diameter	Small	Large
Core diameter	Larger	Smaller
Pitch	Small	Large
Pull out strength	Low	High
Screw strength	High	Low

What are the factors that increase resistance to pull out?
- Increase outer diameter.
- Decrease inner diameter.
- Decrease pitch.

Describe lag screw principles.

- Anatomical reduction of the fracture.
- Trajectory of the screw should be at the centre of the fragment and at right angle to the fracture plane to achieve maximal compression.
- Over drilling of the proximal fragment to create a glide hole. For example, if a 3.5 mm cortical screw is used, the proximal fragment is drilled with a 3.5 mm drill bit.
- The drill sleeve for the core diameter is inserted into the gliding hole.
- Drill the far cortex. 2.5 mm if using 3.5 m. 3.2 mm if using 4.5 mm. The screw will engage with the far cortex not the proximal hole causing fracture compression.
- Countersink the near cortex.
- Measure the depth.
- Insert and tighten the screw.

1.2 Viva 2



- **Describe the X-ray**.
- **What is osteopetrosis?**
- **What are the types of osteopetrosis and what is the pathophysiology?**
- **What are the clinical features of osteopetrosis?**
- **How will you treat this patient?**

Describe the X-ray.

This is a radiograph of an individual which shows a fracture in the subtrochanteric area of the femur. There is an increase in overall bone density throughout with loss of cortico-medullary differentiation. The features are suggestive of osteopetrosis.

What is osteopetrosis?

Osteopetrosis is a congenital bone disease caused by defective osteoclastic resorption of immature bone. It is characterized by long bone fractures, cranial nerve palsies and low back pain.

What are the types of osteopetrosis and what is the pathophysiology?

- Genetic inheritance (three types)
  - Malignant autosomal recessive
  - Intermediate autosomal recessive
  - Benign autosomal dominant (most common)
- Pathophysiology: there is decrease or defective osteoclast dysfunction leading to dense bone and obliterated medullary canals. It is mainly due to carbonic anhydrase II dysfunction or chloride channel dysfunction.

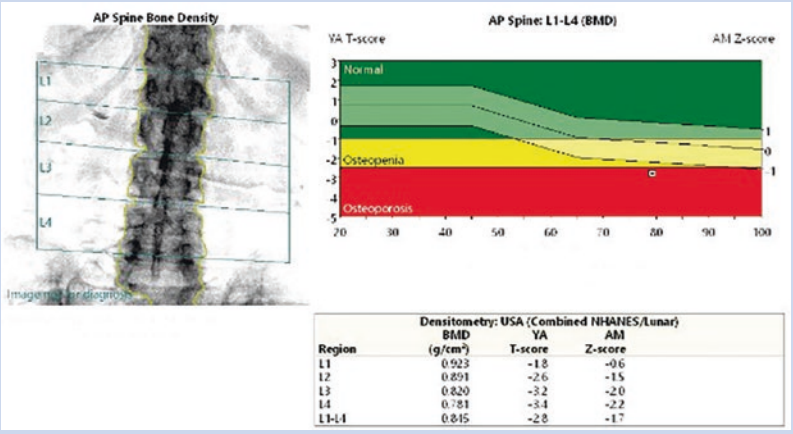
What are the clinical features of osteopetrosis?

The commonest symptoms are frequent fractures, progressive deafness and blindness in severe cases and severe anaemia in autosomal recessive types. However, the most common benign type which is autosomal dominant is usually asymptomatic.

How will you treat this patient?

- The recommended treatment approach is a multidisciplinary one. In terms of medical management, options include bone marrow transplant and the administration of high-dose calcitriol, particularly in cases of autosomal recessive types.
- Surgical options include avoiding intramedullary nail and to use a plate instead. Slow steady drilling and constant cooling and change of drill bit are required. There is an increased risk of hardware failure and higher union rates compared to non-operative management.

1.3 Viva 3



- **Can you interpret this DEXA scan?**
- **Define osteoporosis.**
- **But define osteoporosis as per WHO.**
- **What is the pathophysiology behind it?**
- **What is a DEXA scan?**
- **Could you provide an explanation of *Z*-scores and *T*-scores?**
- **What are the guidelines for treatment of osteoporotic fractures?**
- **What is FRAX score?**

Can you interpret this DEXA scan
The BMD measured at A/P spine is 0.845 g/cm^2, with a *T*-score of −2.8. This patient is considered osteoporotic according to World Health Organization (WHO) criteria. Pharmacological treatment (bisphosphonates) should be considered.

Define osteoporosis.
It is a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration of bone tissue.

But define osteoporosis as per WHO.
It is defined by the World Health Organization (WHO) as a bone mineral density that is 2.5 standard deviations or more below the mean peak bone mass of an average young (30 years), healthy adult as measured by the DEXA scan (dual-energy X-ray absorptiometry).

What is the pathophysiology behind it?
Bone resorption is always followed by bone formation, a phenomenon referred to as coupling. In osteoporosis, this coupling mechanism is thought to be unable to keep up with the constant microtrauma to trabecular bone.

What is a DEXA scan?
Dexa Scan (Dual-Energy X-ray Absorptiometry)

- The technique involves simultaneous measurement of the passage of X-rays through the body with two different energies in order to minimize the effect of soft tissue (particularly the fat) on the result. It is usually performed in the lumbar spine and measures BMD from L2-L4 and compiles scores.
- Causes of false negative in the spine: osteoarthritis.
- Causes of false positive in the spine: previous laminectomy-vertebral fractures.

Could you provide an explanation of Z-scores and T-scores?

- *Z*-score is the number of standard deviations (SDs) that a patient's BMD is above or below the mean value for the person of the patient's age, sex and race.
- *T*-score is the number of standard deviations above or below the mean peak bone mass for a sex and race-matched healthy normal young adult population.

What are the guidelines for treatment of osteoporotic fractures?
NICE guidelines recommend bisphosphonates (alendronate, etidronate and risedronate) as treatment options for the secondary prevention of osteoporotic fragility fractures:

1. In women aged 75 years and older, without the need for prior DEXA scan.
2. In women aged 65–75 years, if the presence of osteoporosis is confirmed by DEXA scanning.
3. In post-menopausal women younger than 65 years of age, if they have a very low bone mineral density, or if they have confirmed osteoporosis plus, or more additional age-independent risk factors.
   (a) Low body mass index.
   (b) Family history of maternal hip fracture before the age of 75 years.
   (c) Untreated premature menopause.
   (d) Certain medical disorders independently associated with bone loss such as chronic IBD, RA, hypothyroidism, or coeliac disease—condition associated with prolonged immobility.

What is FRAX score?
FRAX (World Health Organization Fracture Risk Assessment Tool) calculates the 10-year risk of fracture based on the following variables: age, sex, race, height, weight, BMI, history of fragility fracture, parental history of hip fracture, use of oral glucocorticoids, secondary osteoporosis and alcohol use to calculate 10-year risk of fracture.

1.4 Viva 4

- **How do bisphosphonates act?**
- **What are the types of bisphosphonates?**
- **How does osteoclast work?**
- **In what conditions do you use bisphosphonates?**
- **What are the complications of bisphosphonates?**

How do bisphosphonates act?
It is the first line in the management of osteoporosis.

- It prevents loss of bone mass.
- It inhibits the formation of osteoclast Ruffled border.

What are the types of bisphosphonates?
- Non-nitrogen containing (simple phosphonate) which works by creating a non-functioning analogue of adenosine triphosphate (ATP) which competes with ATP and leads to osteoclast apoptosis. It directly stabilizes the hydroxyapatite crystals making it more resistant to resorption.
- Nitrogen containing (such as alendronate, risedronate and zoledronate) directly inhibits the enzyme farnesyl diphosphate synthase preventing prenylation (formation of brush border) and functioning of signalling proteins required for protein prenylation and prevents osteoclastic bone resorption.

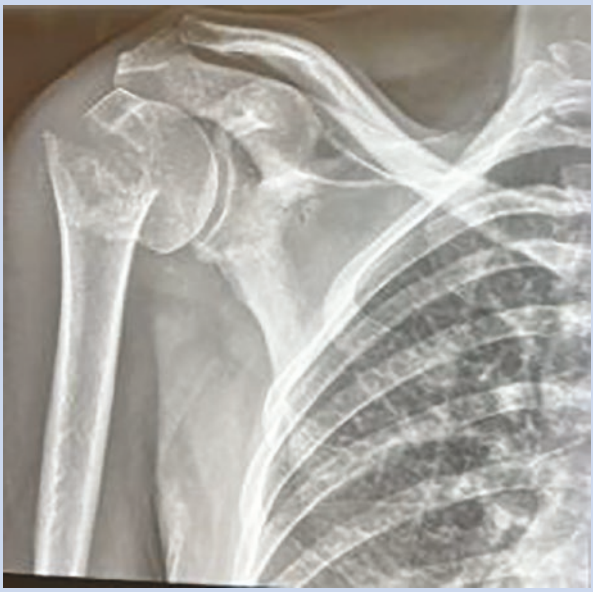
How does osteoclast work?
- Active osteoclast is derived from inactive form which is a mononuclear precursor. Osteoclasts reside in howship's lacunae (pits in endosteal or periosteal bone).
- On the surface, it expresses RANK which helps to bind onto the RANK ligand which is expressed by the osteoblast. Once it attaches to each other it activates osteoclast to become a multinucleated cell. Osteoclasts have ruffled border which increases surface area for resorption to take place.
- It releases hydrochloric acid and hydrogen ions via carbonic anhydrase system which is ATP-driven making the area more acidic. The reduced acidity dissolves the majority of the inorganic matrix of the bone (hydroxyapatite).
- To resorb the organic matrix, proteolytic enzymes such as TRAP (tartaric acid phosphatase) and cathepsin K which are produced by lysozymes and mitochondria dissolve the organic matrix (*such as proteoglycans, Collagen 1*).

In what conditions do you use bisphosphonates?
Bisphosphonates are used clinically to treat post-menopausal and glucocorticoid-induced osteoporosis, Paget's disease of the bone, malignant hypercalcaemia and CRPS.

What are the complications of bisphosphonates?
Oral bisphosphonates are associated with gastric and oesophageal irritation. They should be taken after food. Atypical femoral fractures with long-term use. Intravenous bisphosphonates can cause osteonecrosis of the jaw. Intravenous bisphosphonates are more effective and last longer. Oral bisphosphonates are less well absorbed.

1.5 Viva 5

A 51-year-old female sustained a fall from a standing height.



- **Describe the radiograph.**
- **What is the most appropriate definitive treatment?**
- **Could you walk me through the steps of the surgical approach you are going to use?**

Describe the radiograph.
This is an AP view of the proximal humerus. It shows displaced proximal humerus fracture.

What is the most appropriate definitive treatment?
Surgery—Open reduction and internal fixation

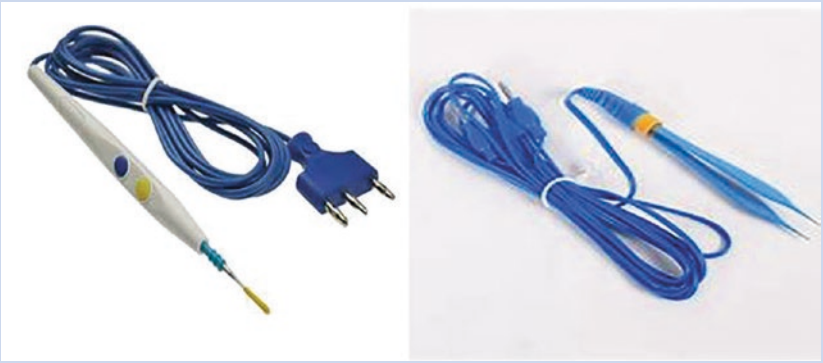
Could you walk me through the steps of the surgical approach you are going to use?
Assuming full informed consent has been taken, all relevant case notes and radiographs have been obtained, the surgical site has been marked, patient has been suitably anaesthetized and preoperative antibiotic was administered.

- **Position and Set up:** I would utilize the beach chair position. This would include sitting the patient up at 45°, placing pillows under the knees, and formal head

support. I would confirm the ability to get image intensifier films, I would do a WHO checklist time out and proceed with routine preparation and draping.

- **Landmarks:** I would identify and mark the landmarks which include coracoid.
- **Incision:** Line from the coracoid process, along the deltopectoral groove to the lateral arm at the level of axilla.
- **Superficial Surgical Dissection:** I would identify the cephalic vein which lies in a layer of fat and is used to identify the interval between the deltoid and pec major. I would retract the cephalic vein (usually laterally as the tributaries come from the lateral side) and divide the deltopectoral fascia of the arm.
- **Deep Surgical Dissection:** Identify the coracoid process. I would divide the clavipectoral fascia from the coracoid process down along the lateral border of conjoint tendon. I would retract conjoint tendon medially, with care taken to avoid excessive retraction and damage to the musculocutaneous nerve and retract the deltoid laterally. This will allow for identification of the subscapularis tendon. I would externally rotate the arm to put the subscapularis under tension. I would place stay sutures in the lateral part of the subscapularis tendon and divide in tendinous portion and take off capsule under tension. I would perform a vertical capsulotomy to enter the joint.
- **Structures at Risk:**
  - Musculocutaneous nerve: This enters the coracobrachialis muscle as close as 2.5 cm distal to the tip of the coracoid. Vigorous retraction can lead to damage to this nerve.
  - Axillary nerve: This is at risk during the incision in the subscapularis tendon, where it lies just distal to the tendon as it wraps around the humerus from lateral to medial.
  - Cephalic vein: This can cause bleeding if damaged intraoperatively and will increase surgical oedema if damaged and ligated.

1.6 Viva 6



- **Explain the mechanism of action of the device shown in the picture.**
- **What are the different waveforms of this device?**
- **What are the two common ways of diathermy that we use routinely?**
- **What are the precautions when using this device?**

Explain the mechanism of action of the device shown in the picture.
Diathermy works by passage of high-frequency alternating current at high local intensities through the patient to generate heat with temperature up to thousand degrees. The neuromuscular tissue is not stimulated at frequency above 50 MHz. Diathermy uses a frequency above 100 MHz.

What are the different waveforms of this device?
- **Cut:** uses a continuous low voltage current to generate high local temperatures.
- **Coagulation:** uses intermittent current, doesn't cause tissue damage but it does allow coagulum to form.
- **Fulguration:** coagulation over a wider area.
- **Blend:** has longer 'on' time than coagulation.

What are the two common ways of diathermy that we use routinely?
Monopolar: uses high power units producing a power of at least 400 W to generate high-frequency local current at the active electrode, which is held by the surgeon, passes through the patient and then to the return electrode. The return electrode must be placed in well-vascularized shaved skin with no scars, way from bony prominences and away from metallic implants, to allow dissipation of heat and avoid burns.

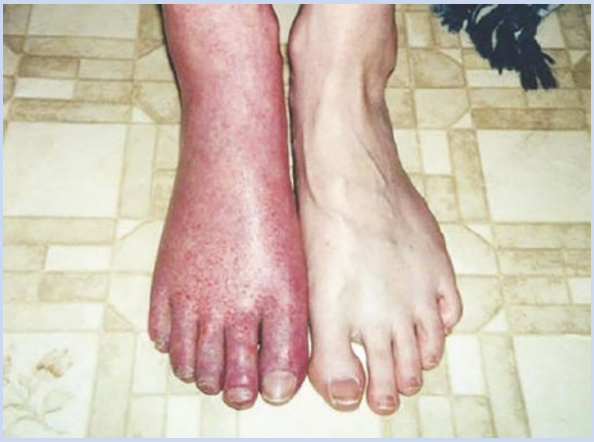
Monopolar allows both modes, cutting and coagulation.

Bipolar: uses low power units of 50 W to generate current between the two tips of diathermy forceps which is held by the surgeon. These act as active and return electrodes, there is no plate on the patient, it allows on coagulation.

What are the precautions when using this device?
- Active electrode should be in contact with target tissue and for the lowest time possible.
- Return electrode should be on well-vascularized shaved skin with no scars, away from metallic implants.
- Avoid in patients with pacemaker, if you must use, try bipolar with ECG monitoring.
- Avoid use when there is pooling of chlorhexidine.

1.7 Viva 7

A 31-year-old woman has a history of right foot trauma from 3 months ago. She is currently experiencing pain over the dorsum of the foot and withdraws her foot when any touch is attempted.



- **What is the most likely diagnosis?**
- **What is CRPS?**
- **What are the predisposing factors of complex regional pain syndrome?**
- **What are the types of CRPS?**
- **What are the clinical criteria for diagnosing CRPS?**
- **How do you treat CRPS?**

What is the most likely diagnosis?
The presented history, symptoms, along with the clinical photo strongly suggest complex regional pain syndrome (CRPS).

What is CRPS?
Abnormal reaction that is characterized by pain, swelling, stiffness, skin discoloration and vasomotor disturbances of the affected part.

What are the predisposing factors of complex regional pain syndrome?
The exact cause is unknown. There are many precipitating factors which have been identified:

- Trauma (usually minor)
- Cerebral lesion
- Spinal cord disorder

- Infection
- Surgery
- Prolonged immobilization and tight cast
- Emotional distress

What are the types of CRPS?

- CRPS type 1—Common, formerly known as reflex sympathetic dystrophy (RSD), does not exhibit demonstrable nerve lesions.
- CRPS type II was formerly known as causalgia. It **has evidence of obvious nerve damage.** It is more painful and difficult-to-control symptoms. The four cardinal signs are pain out of proportion, swelling, stiffness and discoloration.

What are the clinical criteria for diagnosing CRPS?

Diagnosing CRPS is mainly clinical-Budapest Criteria

Symptoms

- **Sensory**—Hyperaesthesia (an abnormal increase in sensitivity), and/or **allodynia**, which is pain caused by stimuli which shouldn't trigger a painful response. Examples of allodynia include lightly touching the affected limb, perhaps by moving a bed sheet or by a gentle tap on the wrist.
- **Vasomotor—**Skin colour changes and/or changes in temperature of the limb.
- **Sudomotor/oedema—**Swelling of the limb and/or an excess of sweat from the affected limb, or changes in sweating swelling of the limb.
- **Motor/trophic—**Decreased range of motion and/or motor dysfunction, including weakness, spasms, tremors or wasting. Trophic changes include changes to the hair, nails or skin on the affected limb.

Signs

At the time of clinical examination, at least one sign must be present to the person carrying out the examination in two or more of the categories listed below, they vary slightly from the list above.

- **Sensory**—Hyperalgesia (to pinprick), a heightened sensitivity to pain, and/or allodynia to light touch or deep somatic (physical) pressure and/or joint movement.
- **Vasomotor**: temperature differences between the limb, and/or skin colour changes and/or skin colour changes between the limb.
- **Sudomotor/oedema:** oedema and/or sweating changes and/or sweating differences between the limbs.
- **Motor/trophic:** decreased range of motion and/or motor dysfunction (i.e. weakness, tremor, or muscle spasm) and/or trophic changes (hair and/or nail and/or skin changes).

How do you treat CRPS?
Non-operative:

- Adequate pain control.
- Physiotherapy: active ROM and mirror therapy.
- Alpha blocker agents (Phenoxybenzamin), Antidepressant, Anti-convulsant—GABA agonist.
- Bisphosphonates for 8 weeks—once weekly in case of bone resorption.
- Nerve stimulation—TENS and peripheral nerve stimulator.
- Nerve blockade:
  - Chemical sympathectomy—stellate ganglion block for upper limb.
  - Lumbar spinal—for lower limbs.

Operative

- Sympathectomy.

Vitamin C (500 mg daily × 50 days) has been shown to decrease the incidence of CRPS (type I) following foot and ankle surgery and in distal radius fracture treated conservatively.

1.8 Viva 8

- **Give a definition of bone graft.**
- **What are the three main properties of bone graft?**
- **What are the types of bone graft that you are aware of?**
- **Explain the term graft incorporation and what are the stages of graft healing.**
- **What are the steps and processes involved in bone banking?**

Give a definition of bone graft.
Bone graft is a material that assists with bone healing through its osteoconductive, osteoinductive and osteogenic properties.

What are the three main properties of bone graft?
- **Osteoconduction—**process by which graft provides scaffold or matrix enabling blood vessel ingrowth for a new bone to form. Examples—Calcium phosphate, Synthetic polymers.
- **Osteoinduction**—provides a biologic stimulus that has capacity to activate differentiation of mesenchymal stem cells into osteoprogenitor cells capable of forming new bone. Examples—Bone Morphogenetic Proteins (BMP), Transforming growth factor-β, Platelet-derived growth factor (Fresh frozen allograft).
- **Osteogenic** bone graft provides living cells that will produce bone including primitive mesenchymal stem cells, osteoblasts and osteocytes (Fresh allograft).

What are the types of bone graft that you are aware of?
Autograft: involve utilizing bone obtained from the same individual receiving the graft.

- Cancellous (Less structural support—Greater osteoconduction—Rapid incorporation via creeping substitution).
- Cortical (Provides more structural support—Slower incorporation due to need to remodel existing Haversion canals).
- Corticocancellous.
- Vascularized bone graft (quicker union and cell preservation—Examples include free fibula strut graft (peroneal artery), free iliac crest (deep circumflex iliac arteries), distal radius used for scaphoid fractures (1–2 intercompartmental superior retinacular artery branch of radial artery).

Allograft: bone from another individual, same species, mainly osteoconductive, can be osteoinductive.

- Fresh (Unprocessed-Highest risk of disease transmission and immunogenicity—BMP preserved—provide osteogenic properties)
- Fresh frozen (Unprocessed-Stored at −70 °C for minimum of 180 days—Less immunogenicity than fresh—BMP preserved and therefore osteoinductive).
- Freeze dried (processed to remove soft tissues and washed to deplete cell and marrow content followed by freeze drying—Least immunogenic—Least structural integrity—BMP depleted (purely osteoconductive)—Lowest likelihood of viral transmission).

Xenograft (different species)

- As allograft but greater potential for rejection.

Isograft (genetically identical)

Explain the term graft incorporation and what are the stages of graft healing.

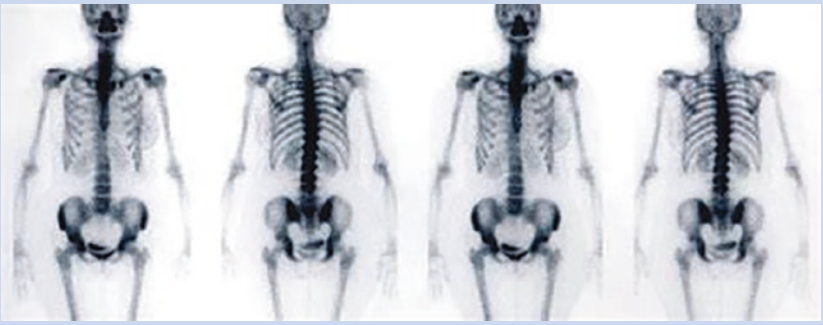
1. Graft incorporation: the process by which invasion of the graft by host bone occurs. There are five stages of graft healing:
   (a) Inflammation: necrosis of the graft occurs, and inflammatory response is established with ingrowth of capillary buds bringing macrophages and mesenchymal cells. The graft is penetrated by osteoclast, which initiates the resorptive phase and incorporation.
   (b) Osteoblast differentiation from precursors.
   (c) Osteoinduction: osteoblast and osteoclast function.
   (d) Osteoconduction: new bone forms over scaffold.
   (e) Remodelling.
2. The earlier stages are similar for both cortical and cancellous bone, but osteoconduction and remodelling are different.
3. In cancellous bone: the osteoblast and osteoclast work simultaneously by a process called **creeping substitution** (it refers to osteoblasts laying down new bone over the old grafted bone, which is subsequently resorbed by osteoclast).
4. In cortical bone: the initial inflammatory response is slower, and the osteoclast resorption occurs by cutting cones entering the graft. Mechanical strength is lost in the first 3–6 months and returns over 1–2 years.

What are the steps and processes involved in bone banking?

1. Donor Consent:
   - Living Donor: Individual provides consent themselves.
   - Cadaveric Donor: Consent is obtained from the relatives of the deceased.
2. Donor Screening:
   - Infection Check: Screening for HBV, HCV, HIV and syphilis.
   - Ensuring the absence of malignancies.
3. Preparation: This involves six distinct phases:
   - Debridement: Removal of all tissue from the bone.
   - Ultrasonic Washing: Thorough cleansing using ultrasonic methods.
   - Ethanol Treatment: Application of ethanol to eliminate bacteria.
   - Antibiotic Soaked Treatment.
   - Irradiation: Process to eliminate microbes.
   - Demineralization: Partial removal of proteins to reduce immunogenic reactions.

4. Preservation:
   - Fresh: Unprocessed bone with osteogenic properties. Highest risk of disease transmission and immunogenicity.
   - Fresh Frozen: Unprocessed bone stored at −70 °C for a minimum of 180 days. Lower immunogenicity compared to fresh bone.
   - Freeze Dried: Most commonly used in practice. Least immunogenic and highest likelihood of viral transmission. Lower structural integrity and lacks bone morphogenetic proteins (BMP), making it purely osteoconductive.

1.9 Viva 9



- **What is bone scan?**
- **What are the phases of bone scintigraphy?**
- **What are the identifiable pathologies on bone scans?**
- **What are the limitations of bone scan?**
- **What is the duration for which a bone scan can be positive following a fracture?**
- **What advice will you give the patient after a bone scan?**

What is bone scan?

Nuclear medicine imaging that detects distribution of an injected radio-labelled tracer followed by gamma camera recording of photoemissions.

The most commonly used radiolabelled tracer:

- Technetium-99m—affinity for osteoblasts; t1/2—6 h.
- Gallium-67—affinity for inflammatory proteins; t1/2—3.2 days.
- Indium-111—autologous leucocytes obtained and labelled with Indium-111 then re-injected; t1/2—2.8 days.

What are the phases of bone scintigraphy?

- Flow phase—images immediately (1–2 min) post-injection; demonstrates arterial flow and areas of hyperperfusion.
- Blood pool phase—images 5 min post-injection; demonstrates relative vascularity where areas of inflammation result in 'pooling' of blood due to stagnant flow in dilated capillaries (soft tissues and bone).
- Static phase—images 3 h post-injection; demonstrates bone activity.

What are the identifiable pathologies on bone scans?

- Infection (hot in all three phases).
- Trauma (stress fractures-non-union).
- Tumours (osteoid osteoma-osteoblastoma).
- Some metastases.
- Loosening in arthroplasty (bone scan shows increased uptake in the delayed static bone phase. The blood flow and blood pool phase are normal).
- Paget's disease.
- CRPS.

What are the limitations of bone scan?

- High sensitivity but low specificity for increased bone activity.
- Two-dimensional imaging—poor localization of small lesions; alternative is single photon emission computer tomography (SPECT) imaging which obtains the scan with an arc of 360°around the patient.
- High radiation dose—advice and encourage fluid intake following investigation to aid excretion of radiolabelled tracers. Example—Technetium-99m 3.5 mSv (Chest radiograph 0.02 mSv).

What is the duration for which a bone scan can be positive following a fracture?

The bone scan usually becomes positive within 24 h to 3 days following a fracture. The dynamic flow component can remain positive for as long as 2–3 weeks following the fracture before returning to normal. The blood pool scan remains positive for approximately 8 weeks. The delayed static scan is usually positive for 6 months to 2 years due to continuing bone healing and remodelling. In cases of malunion, it can remain positive indefinitely due to continued bone remodelling.

What advice will you give the patient after a bone scan?

- Drink plenty of fluids for the rest of the day.
- Avoid contact with pregnant women.
- If travelling abroad in the 7 days following the scan, to take a doctor's note along as ports and airports have very sensitive radiation detectors which may pick up tiny amounts of radioactivity remaining after the scan.

1.10 Viva 10

- **What are the basic principles of the device shown in this picture?**
- **How do you decide on inflation pressures in upper and lower limb surgery?**
- **What are the complications following the use of a tourniquet?**
- **What do you understand by the term post-tourniquet syndrome? How is it treated?**
- **What details would you record in the operative record after surgery? (BOAST guidelines)**
- **What is the upper limit of tourniquet time? (BOAST guidelines)**

What are the basic principles of the device shown in this picture?
Tourniquets help to provide a bloodless field during surgery, by eliminating arterial flow distal to the tourniquet. They can be non-pneumatic (used for digits) or pneumatic which can be automatic (operate from an air line or electric pump) or non-automatic (hand-operated pump).

They should be well padded, of appropriate size and shielded from the surgical prep, which could lead to a burn.

How do you decide on inflation pressures in upper and lower limb surgery?
- Recommended pressures for upper Limb—50–75 mmHg above systolic blood pressure.
- Lower Limb—100–150 mmHg above systolic blood pressure.

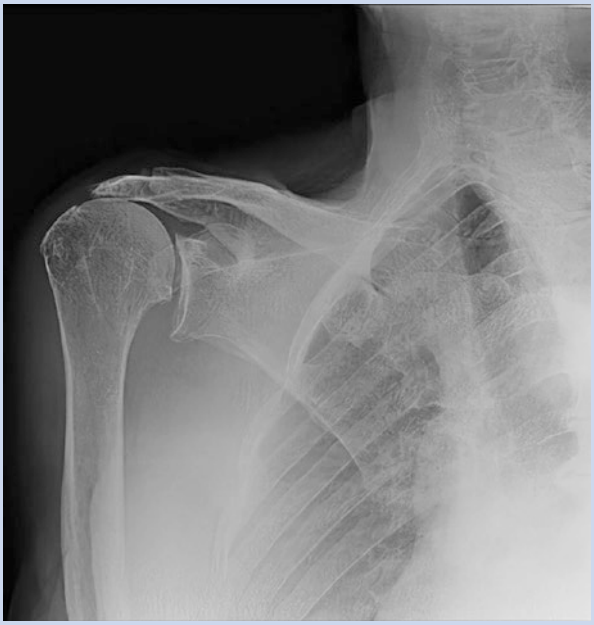
What are the complications following the use of a tourniquet?
- Compression neurapraxia.
- Skin and soft-tissue necrosis.
- Post-operative swelling/stiffness.
- Delayed recovery of muscle power.
- Alterations in acid–base balance.

What do you understand by the term post-tourniquet syndrome? How is it treated?
This is a reperfusion injury due to ischaemia, oedema, stiffness, pallor, weakness and numbness can be noted. It is treated with removal of the tourniquet and supportive measures such as ensuring that the patient is well hydrated and haemodynamically stable.

What details would you record in the operative record after surgery? (BOAST guidelines)
- The condition of the tourniquet site prior to and at the end of the procedure.
- The method of isolation is used to exclude skin preparation fluids from seeping under the tourniquet.
- The method of exsanguination.
- Compressive exsanguination should not be used in the presence of infection, history of malignancy or risk of DVT.

What is the upper limit of tourniquet time? (BOAST guidelines)
- It should ideally be less than 120 min and only extended beyond this after a clinical assessment of the relative risks and benefits, by the operating surgeon.
- Audible reminders must be given to the operating surgeon every 10 min beyond 120 min, and tourniquet use beyond 150 min is rarely justified.

1.11 Viva 11



- **What does the radiograph reveal?**
- **What are the static and dynamic stabilizers of the shoulder?**
- **What are the surgical options for the patient?**
- **What about balloon interposition arthroplasty?**
- **What is the prerequisite for reverse shoulder arthroplasty?**
- **What is the biomechanical principle of reverse shoulder arthroplasty?**

What does the radiograph reveal?
This is a plain radiograph of the shoulder AP view which reveals features of glenohumeral arthritis and proximal migration of the humeral head. This would be consistent with a clinical picture of rotator cuff arthropathy.

What are the static and dynamic stabilizers of the shoulder?
The static stabilizers are

- Bony congruity between the humeral head and glenoid
- Glenoid labrum
- Negative pressure
- Ligaments (superior, middle and inferior)

The dynamic stabilizers are the rotator cuff and extrinsic muscles of the shoulder including the pectoralis major, latissimus dorsi, deltoid, pectoralis minor, biceps and triceps.

In functional ranges of movement, dynamic stabilizers are the principal stabilizers.

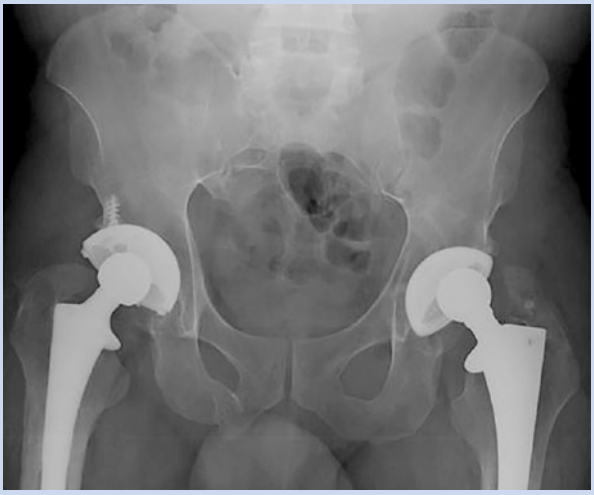
What are the surgical options for the patient?
This would require reverse shoulder arthroplasty.

What about balloon interposition arthroplasty?
Balloon interposition arthroplasty may not be an option in established rotator cuff arthroplasty. It can be considered in individuals with massive irreparable cuff tear to alleviate the pain and prevent superior migration of the proximal head with minimal functional improvement.

What is the prerequisite for reverse shoulder arthroplasty?
An intact and functioning deltoid muscle is prerequisite for reverse shoulder arthroplasty.

What is the biomechanical principle of reverse shoulder arthroplasty?
Delta reverse prosthesis employs a large glenoid hemisphere with no neck and humeral cup with a horizontal position resulting in medialization of the centre of rotation. This results in more of anterior and posterior fibres of the deltoid becoming abductors. The humerus is also lowered resulting in increased tension in the deltoid.

1.12 Viva 12



- **What can you see?**
- **What is wear?**
- **What are the different mechanisms of wear?**
- **What are the modes of wear in artificial implants?**

What can you see?
This is an AP pelvis with bilateral total hip arthroplasties. The left hip shows eccentric femoral head placement within the acetabulum indicative of eccentric polyethylene wear.

What is wear?
It is a progressive unwanted loss of bearing substance from the material as a result of chemical or mechanical action.

What are the different mechanisms of wear?
Wear is usually either mechanical or chemical.

Chemical mechanisms involve corrosion.

Mechanical wear mechanisms include abrasive, adhesive and fatigue (Delamination) wear.

Abrasive wear: Two-body abrasive wear occurs when a soft material comes into contact with a significantly harder material (e.g. metal).

Adhesive wear: occurs when two surfaces come into contact, and the bonds between the surfaces are stronger than the bonds within the materials (cohesive strength), material fragments of the softer material become adherent to the harder material.

Fatigue (Delamination) wear: occurs due to repetitive cyclical loading, so the material fails below its yield strength. It is mainly a problem in knee replacements that accelerated by:

1. Low joint conformity—leads to stress concentration.
2. Low poly thickness—surface stress concentration
3. Malalignment—stress concentration
4. Subsurface faults or oxidization—manufacturing and storage

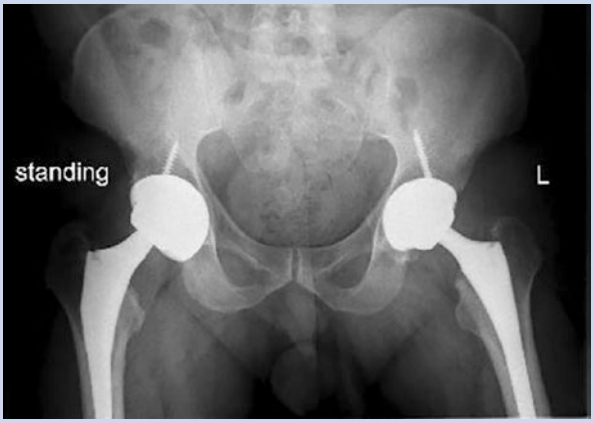
What are the modes of wear in artificial implants?
Mode 1: Generation of wear debris that occurs with motion between the two primary bearing surfaces as intended by the designers.

Mode 2: A primary bearing surface rubbing against a secondary surface in a manner not intended by designers, e.g. a femoral head articulating with an acetabular shell following wear-through of the polyethylene.

Mode 3: Two primary bearing surfaces with interposed third body particles, e.g. bone, cement or metal.

Mode 4: Two non-bearing surfaces rubbing together, e.g. back sided wear of an acetabular liner, fretting of the morse taper, stem-cement fretting or neck of femoral component impinging on rim of cup.

1.13 Viva 13



- **What are the common types of bearing couples utilized in total hip arthroplasty?**
- **What is ceramic?**
- **What are the properties of ceramic which make it to be used as femoral heads?**
- **What are the disadvantages of ceramic?**
- **What are the generations of ceramic?**
- **What are the complications of using ceramic head in THR?**

What are the common types of bearing couples utilized in total hip arthroplasty?

This is an A/P pelvis radiograph with bilateral total hip arthroplasties**.**

The four main bearing couples currently in use are metal/UHMWPE, ceramic/UHMWPE, metal on metal and ceramic on ceramic. The first two can be described as hard on soft and the latter two as hard on hard.

What is ceramic?

Ceramic are compounds of metallic and non-metallic elements. It may be bioinert (alumina and zirconia) or bioactive (hydroxyapatite, glass ceramic).

What are the properties of ceramic which make it to be used as femoral heads?

- Very hard materials with good wear resistance.
- Stiff (high Young's modulus).
- Biocompatible.
- Bioinert (chemically inactive, less risk for cancer).
- Smooth surface.
- High wettability.
- Low friction and wear and low coefficient of friction.

What are the disadvantages of ceramic?

- Brittle (little or no plastic phase before failure).
- Relatively weak under tension.
- Expensive.
- Squeaking due to edge loading from malpositioning leading to loss of fluid film lubrication.
- Stripe wear—caused by contact between femoral head and rim of cup during partial subluxation, result in crescent-shaped line on femoral head. Clinical significance unknown.

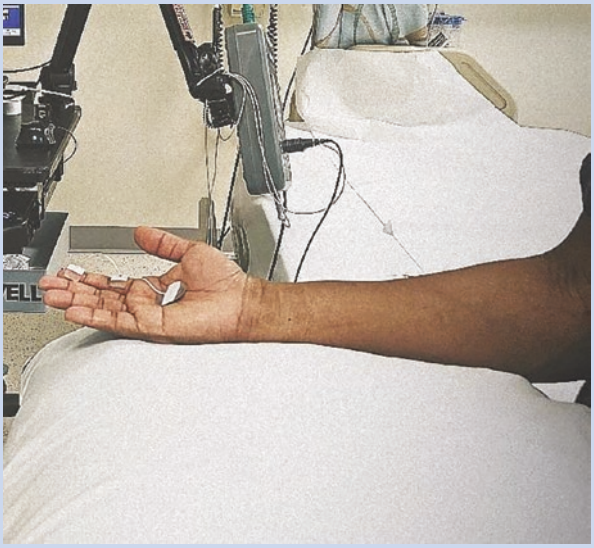
What are the generations of ceramic?

- First generation of ceramic was made of alumina and was processed from industrial-grade materials using high-temperature sintering.
- Second generation of ceramics added molecules such as calcium and magnesium oxide to decrease grain size and control phase transformation.
- Third generation of ceramics used a different process called hipped. The ceramic underwent hot isostatic pressing with the use of pressurized gas. An example of this is the BIOLOX forte that has substantially reduced the rate of fracture.
- Fourth generation has been found to have significantly better wear properties in the lab than previous generations. BIOLOX delta, an example of a fourth-generation ceramic, composed of ceramic consisting of approx. 82% alumina and 17% zirconia, has even higher grain uniformity and smaller grain size than previous generations.

What are the complications of using ceramic head in THR?

- Fracture of the head
- Squeaking
- Stripe wear
- Liner dissociation

1.14 Basic Science: Viva 14



- **What can you see?**
- **Explain the structure of nerve fibre.**
- **How can you classify nerve injuries?**
- **What is nerve conduction study?**
- **Explain the following terms: latency, amplitude and conduction velocity.**
- **What factors adversely affect recovery of a nerve after repair?**

What can you see?
This clinical photograph shows a nerve conduction study of the upper extremity, assessing the sensory function of the median nerve.

Explain the structure of nerve fibre.
- Neuron: the functional unit of the nerve, made up of a cell body and an axon.
- Cell body (perikaryon): chemical factory of the nerve, gives rise to the axons and dendrites.
- Dendrites: receive messages from the other cell bodies and conduct impulses toward cell body.
- Axons: Conduct electrical signals via action potentials.
- Individual axons are surrounded by a connective tissue layer called the endoneurium. Axons are grouped together in motor or sensory bundles called fascicles which are separated by the perineurium. Groups of fascicles are contained within

a peripheral nerve surrounded by a connective tissue layer called the epineurium which is the outermost layer of dense connective tissue surrounding a peripheral nerve (covers individual fascicles and provides tensile strength).

How can you classify nerve injuries?
Seddon Classification 1943:

- **Neuropraxia:** Nerve contusion with reversible conduction block and without Wallerian degeneration.
- **Axonotmesis:** Conduction block with axonal degeneration. Axon and myelin sheath degenerate but endoneural tubes remain intact.
- **Neurotmesis:** All layers of nerve are disrupted and there is Wallerian degeneration.

Sunderland Classification 1951:

- **First degree**—Same as neuropraxia.
- **Second degree—**Same as axonotmesis.
- **Third degree—**Axonal injury associated with damaged basal lamina and endoneural damage. However, perineurium is intact.
- **Fourth degree—**In continuity, both perineurium and endoneurium are disrupted, but continuity of nerve is maintained by epineurium.
- **Fifth degree—**Same as neurotmesis.

Birch and Bonney (Clinical classification)

- Conduction block (same as neuropraxia)
- Degenerative lesion

Degenerative lesions correspond to axonotmesis or neurotmesis.

What are the factors determining the prognosis following nerve injury?
- Violence of injury
- Delay between injury and repair
- Age
- Gap between nerve ends
- Level of injury
- Condition of nerve ends
- Association with arterial/bony injury
- Type of nerve which is injured

What is nerve conduction study?
Test conduction of action potential along large diameter myelinated motor and sensory nerves**.**

It is used to determine:

1. The presence and severity of peripheral nerve dysfunction.
2. Type of injury. Whether it is axonal or demyelinating.
3. Localization and distribution.
4. Clues to the underlying aetiology.
5. Prognosis.

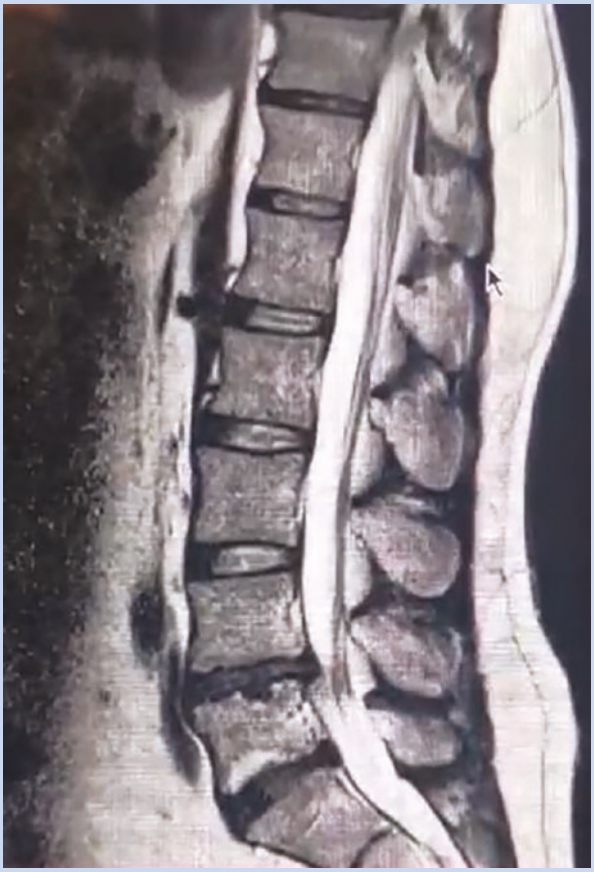
Explain the following terms: latency, amplitude and conduction velocity.

- **Latency** measures the time between onset of stimulus and the response in milliseconds.
- **Amplitude:** Measures the size of the response in microvolts or millivolts. It denotes the quantity of the axons contributing to the action potential. For sensory nerves, this is much smaller measured in microvolts (millionths of a volt) and for motor nerves this is in millivolts (thousandths of a volt).
- **Velocity**: Distance between stimulating and recording electrodes divided by the time. measured in metres per second. The latency and velocity indicate the quality of conduction along the axons.

What factors adversely affect recovery of a nerve after repair?

- Patient factors:
  - Older age.
  - Systemic factors. Diabetes, alcoholism, smoking, rheumatoid arthritis, neuropathies, etc.
- Injury factors:
  - High energy of initial injury. Crush or traction injury.
  - Associated vascular or bony injury.
  - More proximal injury (increased time to reach target organ).
  - Nerves that supply multiple sites with sensory and motor components.
  - Large gap.
- Surgical factors:
  - Delay in repair (increased time for end-plate degeneration).
  - Repaired under tension. Excessive tension can cause breakdown at the area of repair.
  - Quality of repair. It is important to try to suture only the epineurium and not pass the needle and suture through the fascicles, as this can create more damage and scarring, yielding a poorer result. Preservation of blood supply and accurate apposition of the fascicle.
  - Infection.

1.15 Basic Science: Viva 15



- **What can you see?**
- **What is the embryology of the intervertebral disc?**
- **Describe the structure of the intervertebral disc.**
- **How does the disc derive its nutrition?**
- **What are the functions of the intervertebral disc?**
- **What happens to the intervertebral disc when patients age?**

What can you see?
This is a lumbar spinal sagittal T2-weighted MRI showing herniated discs at the level of L4-L5 and L5-S1.

What is the embryology of the intervertebral disc?
- Embryological development of the vertebral column and associated discs occurs at approximately week 4 of gestation.
- The notochord regresses in the region of the vertebral bodies, enlarges in the region of the disc and forms the nucleus pulposus, which is then surrounded by the circular fibres of the annulus fibrosus.

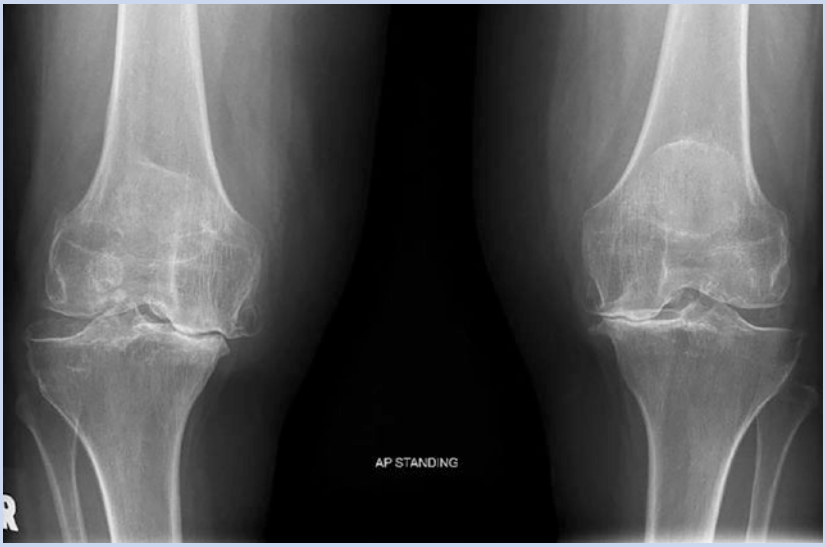
Describe the structure of the intervertebral disc.
- The fibrocartilaginous annulus fibrosus is attached to both the anterior and posterior longitudinal ligaments of the spine as well as the vertebrae on either side.
- Outer annulus fibrosus—Type 1 collagen fibres arranged obliquely in lamellae that insert into the adjacent vertebral bodies via Sharpey's fibres.
- Inner annulus fibrosus—Type 2 collagen.
- Nucleus pulposus—Relatively highly proportion of proteoglycans and low proportion of collagen compared with annulus fibrosis (particularly outer annulus)

How does the disc derive its nutrition?
It is through diffusion through the avascular disc material from the vascular plexus around the annulus fibrosus and cartilaginous end plates.

What are the functions of the intervertebral disc?
- Disc must be able to resist compression, bending, shear and torsional forces.
- Proteoglycans (PG) resist compressive forces (resulting from body weight above disc and action of paraspinal muscles).
- Collagen fibres resist tensile forces. Under compression nucleus pulposus resists the force by converting it into radial forces, which are resisted by circumferential hoop stresses in the annulus fibrosus.

What happens to the intervertebral disc when patients age?
- PG content decreases (reducing resistance to compression).
- Water content decreases. Uniformity of nucleus pulposus decreases causing fibrous areas and softer areas. Collagen content within the intervertebral disc increases as well.

1.16 Basic Science: Viva 16



- **What is the pathogenesis of this condition?**
- **What are the functions of articular cartilage?**
- **Can you explain the histological appearance of articular cartilage?**
- **Describe the biomechanical changes of ageing and osteoarthritis in cartilage.**

What is the pathogenesis of this condition?

This standing A/P radiograph of both knees shows features of osteoarthritis, evident by joint space narrowing, subchondral sclerosis and the formation of osteophytes.

In the initial stages of osteoarthritis (OA), an increase in water content disrupts the collagen network, while a decrease in proteoglycan levels reduces the stiffness of the cartilage matrix, altering the Young's modulus of elasticity. Consequently, the cartilage becomes more susceptible to damage. Chondrocytes, responding to this tissue damage, release mediators that aim to increase cell proliferation as a reparative mechanism.

As osteoarthritis progresses, the initial proliferative response diminishes. Over time, the levels of proteoglycans decrease significantly, leading to a softening of the cartilage and loss of elasticity. This further compromises the integrity of the joint surface, contributing to the progression and severity of the disease.

What are the functions of articular cartilage?

- Shock absorber: resist the compressive forces encountered across the joint under loading.
- It has very low coefficient of friction 0.0002, 30 times smoother than modern bearing surfaces.
- Lubrication.

Can you explain the histological appearance of articular cartilage?

Microscopically: articular cartilage composed of 25% cells and 75% extracellular matrix (ECM).

- Cells: Chondrocytes from mesenchymal stem cells. Produce extracellular matrix.
- ECM:
  - Water: 65–80% of weight.
  - Collagen fibres: Most abundant is **type II.** Provide tensile strength.
  - Proteoglycans: Provide compression strength.
  - Glycoproteins.
  - Degradative enzymes.

Macroscopically: articular cartilage has a layered structure that shows decreased water and collagen and increase in proteoglycans from superficial to deep.

Superficial tangential (10–20% thickness)

- It is the thinnest layer.
- It has the highest water and collagen content.
- Almost entirely collagen fibres, lie horizontally parallel to joint surface to resist shear stress.
- Flat chondrocytes parallel with lamina splendens.
- The most superficial part is called the lamina splendens, providing a very low-friction lubrication surface. It contains no cells.

Intermediate "Transition zone" (40–60% thickness)

- The thickest layer.
- Collagen fibres are obliquely oriented.
- More rounded chondrocytes.
- Represent transition of shear to compression loading.

Deep basal "Radial zone" (30% thickness)

- Collagen fibres lie longitudinally perpendicular to joint surface to resist compression.
- Rounded spherical and more concentrated chondrocytes.
- Arranged in columns.

Tidemark: It is the boundary between calcified and uncalcified cartilage. It is cell free and represents calcified cartilage to provide structural stability for articular cartilage on subchondral bone.

Calcified zone: Hydroxyapatite crystals anchor articular cartilage to subchondral bone. Forms a barrier to blood vessels supplying subchondral bone.

Describe the biomechanical changes of ageing and osteoarthritis in cartilage.
Osteoarthritis

- Water content is increased, with a decrease in the proteoglycan content.
- The chondroitin/keratin sulphate ratio is increased.
- There is increased cell synthesis, increased degradation. Overall, the collagen content is maintained.
- Modulus of elasticity is decreased, becomes more elastic.

Ageing

- Water content is decreased, with a decrease in the proteoglycan content.
- The keratin/chondroitin sulphate ratio is increased.
- Decreased cell synthesis, increased degradation. Overall, the collagen content is maintained.
- Modulus of elasticity is increased.

1.17 Viva 17

You have recently joined the department as a hip consultant and have noticed an increased number of surgical site infections?

- **What are you going to do?**
- **How would we reduce the risk of surgical site infections?**
- **Are you familiar with any scientific papers or studies that specifically explore or discuss the maintenance and assessment of sterility within operating theatres?**

What are you going to do?
I would perform a root cause analysis using an MDT approach to identify the source or cause of these infection situations and find out the underlying cause for the infection and find solutions.

I would stop the surgical operations and investigate and gather data by performing an audit at first. I would ensure patients who are infected are treated appropriately.

How would we reduce the risk of surgical site infections?
In the *preoperative phase* or period, we can advise patients to shower using soap on the day of surgery.

Nasal decolonization: Consider nasal mupirocin in combination with a chlorhexidine body wash before procedures in which Staphylococcus aureus is a likely cause of surgical site infection.

- Hair removal: Do not use hair removal routinely to reduce the risk of surgical site infection. If hair has to be removed, use electric clippers with a single-use head on the day of surgery.
- Think of cell saver if high risk of bleeding is expected intraoperatively.
- Optimization of blood sugar.
- Prophylactic antibiotics to be given at induction.

Intraoperative
Theatre design—Laminar airflow and theatre design (separate topic)

- Hand decontamination: The operating team should wash their hands prior to the first operation on the list using an aqueous antiseptic surgical solution, with a single-use brush or pick for the nails and ensure that hands and nails are visibly clean.
- Skin preparation—Skin prep and paint as per NICE guidelines.
- Diathermy: If diathermy is to be carried out, use evaporation to dry antiseptic skin preparations and avoid pooling of alcohol-based preparations. Do not use diathermy for surgical incision to reduce the risk of surgical site infection.
- Maintain patient haemostasis: Maintain patient temperature—Maintain optimal oxygenation—Maintain adequate perfusion during surgery.
- Apply antiseptic or antibiotic to the wound before closure as part of a clinical research trial.

Postoperative

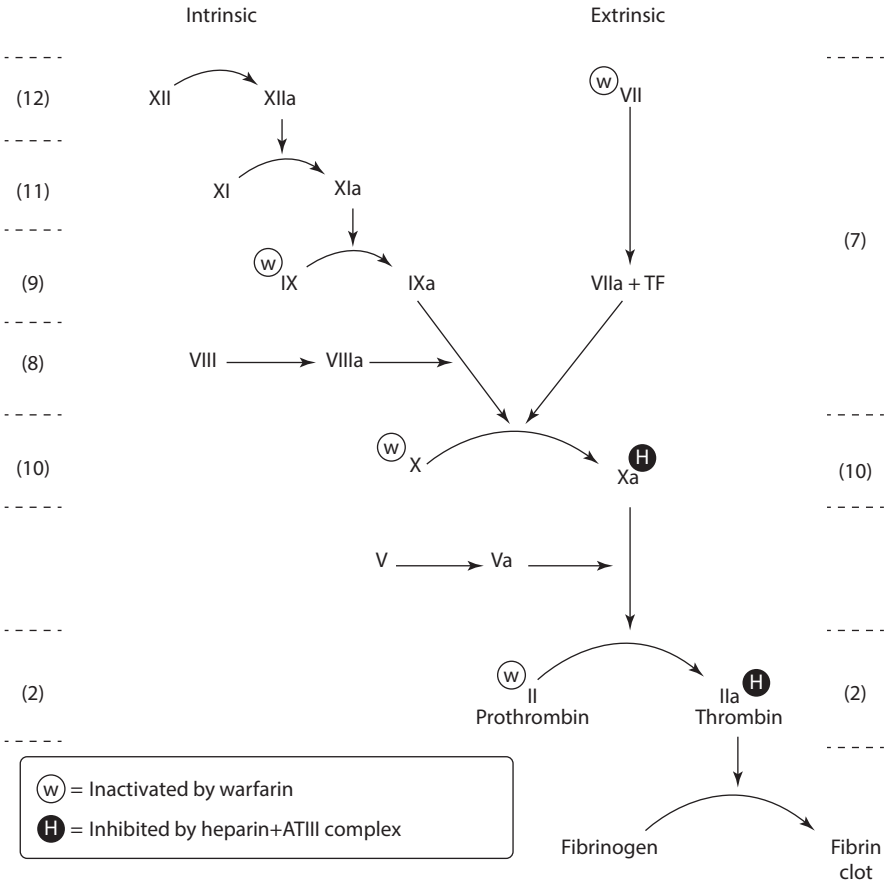
- Changing dressing: Use an aseptic non-touch technique for changing or removing surgical wound dressings. Avoid repeated change of dressing.
- Use sterile saline for wound cleansing up to 48 h after surgery.

Are you familiar with any scientific papers or studies that specifically explore or discuss the maintenance and assessment of sterility within operating theatres?
The classic paper on theatre sterility was published by Lidwell. This was MRC randomized study which showed a decrease in infection rates following joint replacements carried out in ultraclean theatres.

1.18 Viva 18

- **Can you draw the intrinsic and extrinsic clotting pathway?**
- **Can you show me the steps inhibited by low molecular weight heparin?**
- **What are Apixaban, Rivaroxaban and Dabigatran and what are their specific mechanisms of action?**
- **What are the current guidelines/recommendations to offer VTE prophylaxis to people undergoing elective hip/knee replacement?**
- **What is the mechanism of action of Aspirin?**
- **What are the NICE guidelines for initiating VTE for arthroscopic knee procedures?**

Can you draw the intrinsic and extrinsic clotting pathway?



Can you show me the steps inhibited by low molecular weight heparin?
The steps inhibited by low molecular weight heparin are inhibiting activated factor Xa and activated factor IIa.

What are Apixaban, Rivaroxaban and Dabigatran and what are their specific mechanisms of action?
These are newer oral anticoagulants. Dabigatran is a direct thrombin inhibitor whereas both Rivaroxban and Apixabanare are both factors Xa inhibitors.

What are the current guidelines/recommendations to offer VTE prophylaxis to people undergoing elective hip/knee replacement?
Hip replacement

- LMWH (for 10 days) followed by aspirin (for 28 days)
- LMWH (for 28 days) combined with antiembolism stockings (until discharge)
- Rivaroxaban for 5 weeks

Knee replacement

- Aspirin (for 14 days)
- LMWH (for 14 days) combined with antiembolism stockings (until discharge)
- Rivaroxaban for 5 weeks

What is the mechanism of action of aspirin?
Aspirin is a cyclo-oxygenase inhibitor and inhibits prostaglandin production thereby preventing platelet aggregation. It also prevents formation of thromboxane A2, which is a prothrombotic agent secreted by platelets.

What are the NICE guidelines for initiating VTE for arthroscopic knee procedures?
VTE prophylaxis is not required for people undergoing arthroscopic knee surgery where total anaesthesia time is less than 90 min and person is low risk of VTE. Consider LMWH 6–12 h after surgery for 14 days for people undergoing arthroscopic knee surgery only if anaesthesia time is more than 90 min or person's risk of VTE outweighs their risk of bleeding. Consider VTE prophylaxis for people undergoing other knee surgery whose risk of VTE outweighs their risk of bleeding.

Further Reading

BOAST-The-Use-of-Intraoperative-Tourniquets.pdf. https://www.boa.ac.uk/asset/9E51C69D%2DF53A%2D4D1C%2D9212726226AA9A5C/.

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Part II

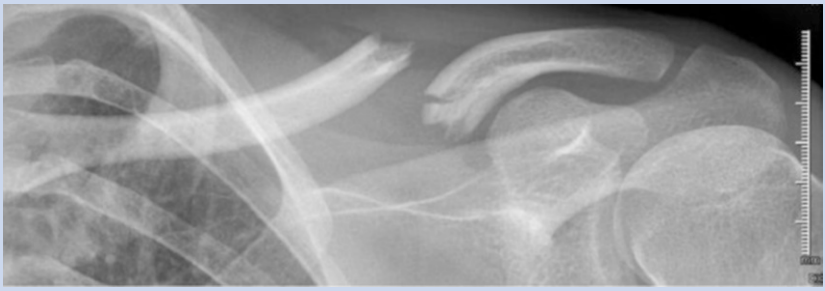
Trauma

Upper Limb Trauma

2

Maamoun Abousamhadaneh and Elhadi Babikir

2.1 Viva 19



- **Describe this radiograph of a 26-year-old male who was involved in a motorcycle accident.**
- **How would you assess this injury?**
- **What are the management options for this patient? What would you do?**
- **What are the indications for surgical intervention?**

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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_2

Describe this radiograph of a 26-year-old male who was involved in a motorcycle accident.
This is a plain radiograph of a skeletally mature patient showing a displaced fracture of left clavicle shaft. Rest of bony landmarks seem intact.

How would you assess this injury?
After conducting a comprehensive assessment of the patient following ATLS protocol and confirming the absence of additional injuries, I would proceed to obtain a full history paying close attention to the hand dominance, occupation, hobbies, sports participation, smoking, and check for any associated medical conditions that may impact on the overall management of this patient.

In terms of clinical evaluation, it is imperative to meticulously assess both skin integrity and neurovascular status. Additionally, I would conduct a careful examination to identify any clinical or radiographic indicators of associated fractures involving the scapula, glenoid, or acromion.

I would provide the patient adequate pain control while ensuring that the upper extremity is adequately immobilized using a sling to promote rest.

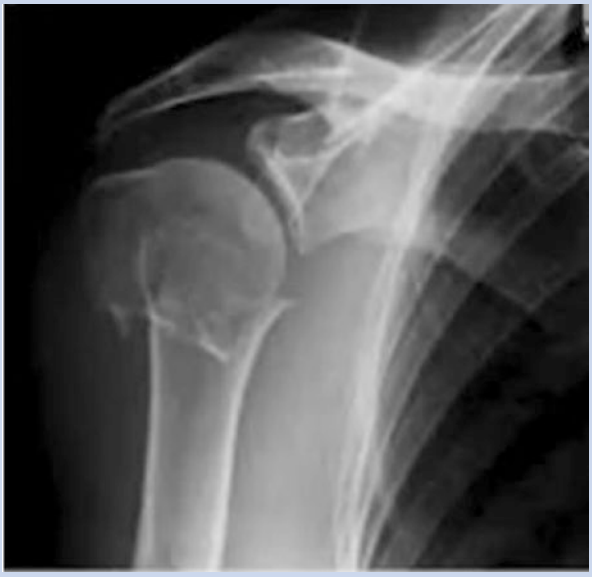
What are the management options for this patient? What would you do?
Treatment is controversial, it can be nonoperative or operative based on several factors such as patient's activity level, the condition of the surrounding soft tissues, the extent of radiographic displacement, and the degree of shortening observed.

Involving the patient in the decision-making process is vital, especially when there are no absolute indications for surgery. I would engage in a detailed discussion with the patient, presenting both treatment options along with their respective advantages and drawbacks. I would explain to him that most of these fractures will heal, and patients will return to full function. However, there are risk factors for non-union in non-operative treatment of midshaft clavicle fractures (roughly 15%) that include advanced age, female gender, smoking, fracture displacement, and comminution. On the other hand, open reduction and internal fixation of clavicle fracture is associated with improved results when there are 100% displacement and >2 cm shortening in young active individuals as per COTS and the clavicle trials. However, surgical treatment is associated with some complications such as hardware prominence (30%), neurovascular injury (3%), infection (4%), non-union (3%), and fixation failure.

What are the indications for surgical intervention?
Indications for surgical fixation of clavicle fractures include open fractures, underlying neurovascular injury, or impending open fracture from internal bony pressure causing skin compromise. Relative indications for fixation include greater than 2 cm of shortening, greater than 100% displacement (no bony contact), highly comminuted fractures, floating shoulder, and polytrauma patients.

2.2 Viva 20

A 67-year-old male sustained a fall to his right shoulder



- **Describe the radiograph.**
- **How would you manage this patient?**
- **Are there any research papers or evidence that support your perspectives?**
- **What factors would make you consider surgical intervention?**
- **What are the complications of surgical fixation of proximal humerus fractures?**

Describe the radiograph.

This is an A/P radiograph of right shoulder showing proximal humerus fracture involving the surgical neck.

I would like to establish several patient and fracture-related factors which are important for decision making. These include mechanism of injury, functional demands, and co-morbidities. Fracture-related factors include open fractures, associated neurovascular deficit, skin compromise, and ipsilateral limb or shoulder girdle fractures. To gain a more comprehensive understanding of the fracture, my initial step would involve requesting additional radiographs in the form of axillary and scapula-Y views. These additional views will provide a clearer visualization of the fracture.

How would you manage this patient?
Assuming this an isolated, closed, and low-energy injury with no associated neurovascular deficit, I would offer the patient non-operative treatment of this fracture. I would ensure that the patient has adequate analgesia and is comfortable in a collar and cuff which helps to reduce and maintain the fracture by the effect of gravity. I would review the patient in the outpatient clinic after 2-weeks with further radiographs. I would refer the patient to physiotherapy to start gentle pendulum exercises from the 3-week point post-injury followed by a gradual progressive rehabilitation which includes passive and assisted active range of motion.

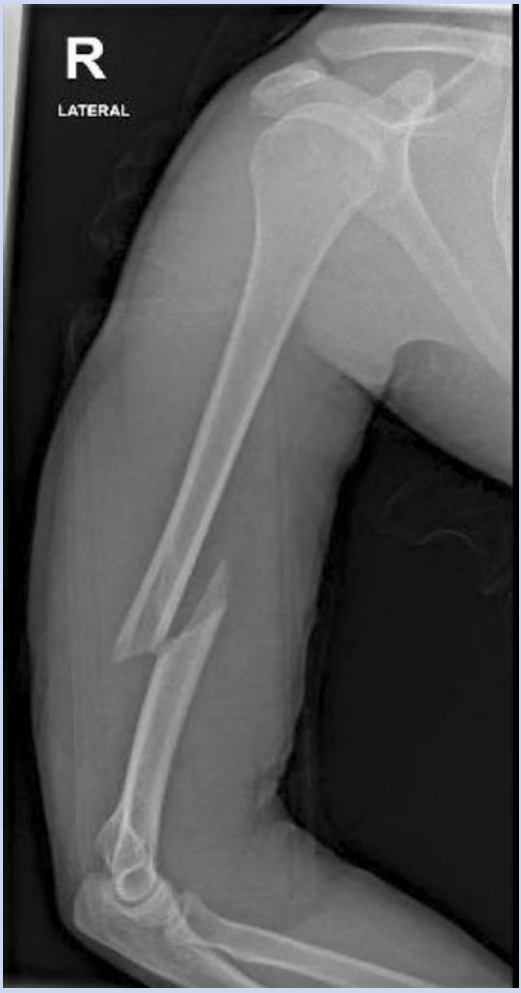
Are there any research papers or evidence that support your perspectives?
There is a multi-center randomized clinical trial (the PROFHER trial) published in 2015 with 2-years follow-up found that no significant difference in the functional outcomes between surgical and nonsurgical treatment among patients >65 years with displaced proximal humeral fractures involving the surgical neck. Then another study was conducted to determine the long-term treatment effects beyond the 2-year follow-up. This study confirmed that the main findings of the PROFHER trial over 2 years were unchanged at 5 years.

What factors would make you consider surgical intervention?
I would consider surgical intervention for injuries complicated by an open wound, tenting of the skin, vascular injury, fracture dislocation, or a split of the humeral head.

What are the complications of surgical fixation of proximal humerus fractures?
Starting with early complications, these would include nerve injury and infection, particularly in open fractures. Late complications include AVN, varus collapse with subsequent screw cut out, and fixation failure.

2.3 Viva 21

A 20-year-old male sustained this injury after a fall from 6 feet.



- **Describe this radiograph.**
- **What factors would lead you to manage a humerus shaft fracture conservatively?**
- **How does Sarmiento brace work?**
- **How might your treatment change in the case of a radial nerve palsy after manipulation?**
- **If you decide to proceed with surgical intervention. What procedure and surgical approach would you choose?**

Describe this radiograph.
This is a plain radiograph of right humerus showing a displaced spiral fracture of the humerus shaft.

I would like to take a detailed history from the patient and examine the entire limb to assess the neurovascular status and the skin integrity.

I would align and immobilize the fracture in a U-slab plaster, repeat neurovascular assessment, and obtain radiographs. Assuming that a satisfactory fracture alignment was achieved, I would have a discussion with the patient and discuss the pros and cons for operative and non-operative treatment options, I would plan to treat this fracture nonoperatively with early functional brace. I'm aware about Cochrane review that has shown equal outcomes between surgical and nonsurgical treatment for humerus shaft fracture.

What factors would lead you to manage a humerus shaft fracture conservatively?
Conservative management, involving functional bracing, is typically recommended for the majority of isolated closed humeral shaft fractures. Generally accepted criteria for non-surgical treatment encompass specific parameters, including less than 30° of varus/valgus angulation, 20° of anterior/posterior angulation, and a shortening of less than 3 cm.

However, it's important to note that certain fracture patterns, such as those characterized by fracture site distraction, a transverse or short oblique morphology, or fractures occurring in the proximal 1/3rd of the humeral shaft, have been documented in the literature as having a higher incidence of non-union when managed non-operatively.

How does Sarmiento brace work?
The brace is typically applied once an initial satisfactory alignment of the fracture has been achieved. It typically consists of plastic sleeves that can be adjusted using straps to accommodate any swelling. The compression of soft tissues aids in maintaining proper fracture alignment while also providing sufficient immobilization to facilitate an early return to functional use. Additionally, the brace allows for some movement of the elbow and shoulder, reducing the risk of unnecessary joint stiffness.

How might your treatment change in the case of a radial nerve palsy after manipulation?
After initially presenting with normal radial nerve function and experiencing a noticeable deterioration following manipulation or the application of a plaster cast, I would discuss with the patient that the radial nerve injury likely occurred during the manipulation, typically due to a stretching injury referred to as "neuropraxia." (In cases involving high-energy or open fractures, neurotmesis may be suspected.)

My next step would be to review the post-manipulation radiographs. If I find that the alignment is satisfactory, with no signs of radial nerve entrapment at the fracture site, I would propose conservative treatment. A systematic review on radial nerve palsy in humeral shaft fractures has reported an overall recovery rate of 88.1%. Notably, there was no significant difference in final outcomes when comparing groups initially managed expectantly with those explored early.

Around the 4 to 6-week mark, I would consider conducting electromyography/nerve conduction studies (EMG/NCS). During this period, I would continue monitoring the patient, paying special attention to the sequence of recovery. Typically, the brachioradialis muscle is the first to recover, while the extensor indicis proprius (EIP) is the last. (Spontaneous recovery is observed in approximately 70% of cases within 4 months.)

In the event of fibrillations (indicating Wallerian degeneration) and a lack of recovery by the 3-month mark, I would refer the patient to a specialized nerve injury unit for further evaluation and management.

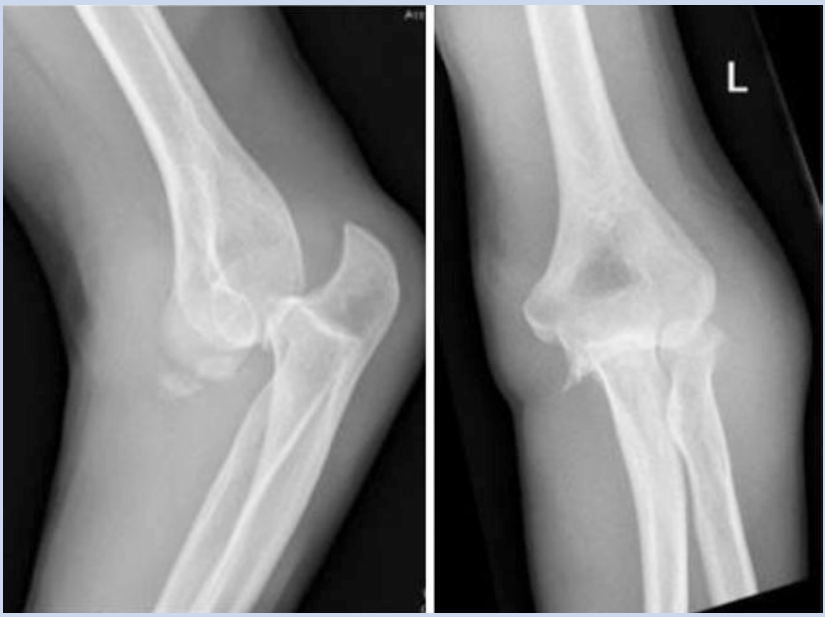
If you decide to proceed with surgical intervention. What procedure and surgical approach would you choose?

I would explain to the patient that there are two methods to fix humerus mid-shaft fracture, either closed reduction and intramedullary nailing or open reduction and internal fixation. I'm aware about Cochrane review that showed no significant differences in functional outcomes and union rate. However, I would advise the patient to go for an open procedure to explore the nerve and fix the fracture. I would choose a posterior approach which allows a good visualization of the nerve on the posterior surface of the humerus. The evidence showed that most nerve injuries involve a neurapraxia in closed fractures. However, I would be prepared to undertake nerve repair or grafting with the assistance of a plastic surgery colleague If the nerve was found to be lacerated.

My preference would be to use a compression plating 4.5 mm with interfragmentary screw for fracture fixation.

2.4 Viva 22

A 50-year-old male presented with left elbow pain and deformity after sustaining a ground-level fall.



- **Can you describe the radiographs and how would you manage this patient?**
- **What is the mechanism of injury and what structures you would expect to be injured?**
- **How can you classify coronoid fractures?**
- **What treatment would you advise for this patient?**
- **Could you please walk me through the operative sequences?**

Can you describe the radiographs and how would you manage this patient?
This is an elbow radiograph of a skeletally mature patient showing a posterior dislocation of the elbow with associated coronoid fracture and possibly radial head fracture. Features are consistent with Terrible triad injury.

I will complete a full clinical assessment and document any neurovascular deficit, then reduce the elbow dislocation under sedation in the emergency room by flexing the elbow to 30° in supination to unlock the olecranon from the olecranon fossa then applying longitudinal traction and gently levering or pushing the olecranon over the distal humerus. I would then confirm adequate reduction using image

intensifier, and I would place the patient in an above elbow back-slab. If closed reduction is unsuccessful, I would arrange to perform this in the operating theatre under general anesthesia.

What is the mechanism of injury and what structures you would expect to be injured?

Terrible triad injury typically occurs with fall on extended arm with a combined mechanism of axial loading, supination of the forearm, and a posterolateral valgus force.

This injury results in a radial head fracture, coronoid fracture, and posterolateral dislocation of the elbow.

Elbow dislocation results from complete or near complete disruption of circle of capsuloligamentous stabilizers and bone (Horii circle). This occurs sequentially from lateral to medial in three stages. Stage 1 (posterolateral rotatory instability) from partial or complete LCL failure (usually epicondyle avulsion and less commonly a midsubstance tear); stage 2 (perched ulna) with additional anterior and posterior capsular structure injury; and stage 3 (dislocation) with MCL failure depending on the degree of injury (often intact).

How can you classify coronoid fractures?

The coronoid fractures are classified according to the system of Regan and Morrey which is based on fragment size. It has three types:

Type I: coronoid process tip fracture

Type II: fracture of 50% or less of height

Type III: fracture of more than 50% of height

Another classification called the O'Driscoll system comprises three fracture types (I: tip, II: anteromedial, and III: basal) defined based on their location in the 3D anatomy.

What treatment would you advise for this patient?

This is an unstable injury, and I would advise operative treatment. I would specifically aim to restore ulnohumeral joint stability by reducing and fixing the coronoid fracture + repairing the collateral ligament.

A preoperative CT scan would be helpful in determining the location of fractured fragments, their degree of comminution and displacement.

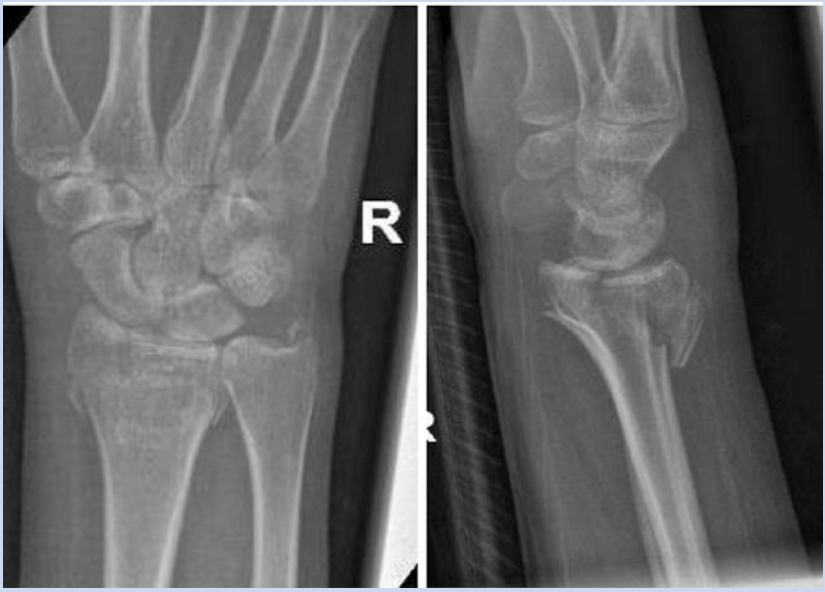
Could you please walk me through the operative sequences?

Having prepared the patient for surgery, I would position the patient in the supine position with placing the affected upper extremity on a radiolucent arm board. I would confirm I could achieve fluoroscopic imaging before scrubbing. I would ensure that preoperative antibiotics were administered, and I would use a narrow sterile tourniquet. First, I will address the radial head through "Kocher approach" utilizing the interval between anconeus and extensor carpi ulnaris. I would remove radial head fragment which would give me access to the coronoid and anterior capsule. Depending on coronoid fragment size I would reduce and fix the coronoid

fracture with a single screw, or I would suture the anterior capsule down to the coronoid footprint using suture anchors. Then prepare to either fix radial head or replacement it. Ideally, I would try and fix this fracture, but I would have a radial head replacement available (if more than three fragments). I would take care not to "overstuff" the joint. Then I would repair LCL using non-absorbable braided sutures and assess the elbow stability. If still unstable I would repair the MCL through the same medial approach. If the elbow still unstable despite all the above I would apply elbow spanning external fixator as a last resort.

2.5 Viva 23

A 35-year-old man, involved in MVC



- **Describe the radiographs.**
- **What are the radiographic findings that indicate instability?**
- **How would you manage this patient in the emergency department?**
- **How would you manage this patient definitively?**

Describe the radiographs.

These are AP and lateral views of a skeletally mature patient showing the right wrist and hand. The most obvious pathology is comminuted, displaced, and intra-articular fracture of distal radius associated with shortening, loss of normal radial inclination, and dorsal comminution.

What are the radiographic findings that indicate instability?
Radiographic features indicating instability include dorsal angulation >5° or >20° of contralateral distal radius, volar or dorsal comminution, displaced intra-articular fractures >2 mm, radial shortening >5 mm, associated ulnar fracture, severe osteoporosis, articular margin fractures (dorsal and volar Barton's fractures). Pre-reduction radiographs are the best predictor of stability.

How would you manage this patient in the emergency department?
This is a high-energy injury in a young adult patient that has to be managed as per ATLS protocol to rule out systemic or life-threatening injuries. Assuming it is an isolated injury, my management would be dictated by BOAST guidelines for distal radius fractures. I would take an AMPL history, and I would assess his limb for any open wounds, the extent of neurological compromise, specifically in the median nerve distribution, as well as looking for vascular compromise and for signs of compartment syndrome. I would document my findings.

My next priority after assessment is to give analgesia and perform closed reduction under regional block to align the fracture as much as possible to reduce the pressure on the soft tissues.

I would splint his wrist and elevate his arm, reassess his neurological status, document the results, and repeat the x-rays.

I would proceed with admission and provide adequate pain control. I would elevate the limb to reduce the swelling and carefully watch for any signs of compartment syndrome.

I would request CT to plan my surgical treatment.

How would you manage this patient definitively?
This is an unstable fracture as indicated by the presence of radial shortening and dorsal comminution in a young male patient. I would treat this injury definitively with open reduction and internal fixation. My goals of treatment would be restoration of intra-articular congruity, rigid maintenance of reduction, and achieve bone union with good function.

I would choose to treat this fracture with locked volar plating through a modified Henry's approach which will allow early mobilization and avoid the need for prolonged cast treatment.

Further Reading

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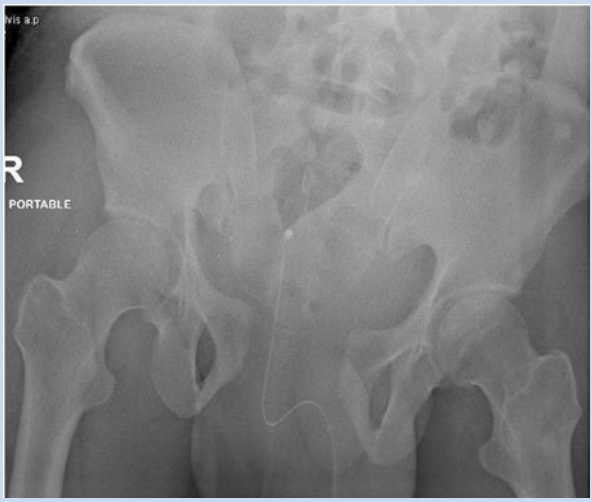
Pelvis and Lower Limb Trauma Viva

3

Aiman Mudawi and Yousef Abuodeh

3.1 Viva 24

A 34-year-old gentleman, pedestrian hit by a car.



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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_3

- **Describe the radiograph.**
- **How would you manage this patient?**
- **What are the radiological signs of rotational and vertical instability in pelvic fractures?**
- **What are the prognostic factors associated with mortality in pelvic fractures?**
- **What is the role of tranexamic acid for control of bleeding in trauma patients?**

Describe the radiograph.

This anteroposterior (A/P) pelvis radiograph reveals an anterior-posterior compression-type pelvic ring injury, evident by the widening of both the symphysis and sacroiliac (SI) joints. This injury places the patient at risk of life-threatening hemorrhage.

How would you manage this patient?

In the emergency department, I would adhere to ATLS guidelines to ensure the identification and prioritization of all life- and limb-threatening injuries. In this specific case, my primary concern would be the patient's circulation.

I would begin by ensuring a patent airway with cervical spine control and assessing breathing. Moving on to circulation with hemorrhage control, I would make sure that pelvic binder is properly applied prehospital to reduce movement and tamponade blood loss. I would make sure that tranexamic acid had been administered. I would gain IV access with two large-bore cannulas into the antecubital fossa, taking blood for FBC, U&Es, LFTs, glucose, lactate, coagulation, and cross-match for at least four units of bloods. Subsequently, I would initiate appropriate resuscitation following my hospital's major hemorrhage protocol. Simultaneously, I would search for any obvious sources of bleeding in the chest, abdomen, pelvis, and long bones.

I would administer 2 L of warmed Ringer's lactate. O-negative blood would be used as necessary, as it is typically available in A&E. For group-specific blood (ABO + RhD grouping), the wait time is usually 15–20 min, while fully cross-matched blood takes 30–40 min. I would adhere to a 1:1:1 protocol (1-unit red cell concentrate (RCC), 1 unit of fresh frozen plasma (FFP), and 1 unit of pooled platelets), with the guidance of rotational thromboelastometry (ROTEM) where available.

During this critical phase, I would coordinate with the theater team, informing them of the patient's unstable pelvis and the potential need for surgery. Additionally, I would communicate with the radiology department, notifying them of the case and the possible requirement for their intervention.

Once test results become available, I would implement target-driven resuscitation with the following goals: Hb > 8, PT and APTT within normal range (transfuse 4 units FFP if APTT or PT ratio > 1.5), Fibrinogen >1 g/L (transfuse 2 units of pooled cryoprecipitate if <1), and Platelets >75 (transfuse 1 unit of platelets if <75, 2 units if <30).

After initial resuscitation and temporary stabilization (either pelvic binder or external fixator) definitive management would involve stabilization of anterior symphyseal (open reduction and internal fixation with a symphyseal plate) and posterior sacroiliac diastases (sacroiliac screws).

What are the radiological signs of rotational and vertical instability in pelvic fractures?

Radiographic signs of instability include sacroiliac displacement of greater than 0.5 mm in any plane, avulsion of the fifth lumbar transverse process, avulsion of the lateral border of the sacrum (implying a tear in the sacrotuberous ligament), and avulsion of the ischial spine (implying a tear in the sacrospinous ligament).

What are the prognostic factors associated with mortality in pelvic fractures?

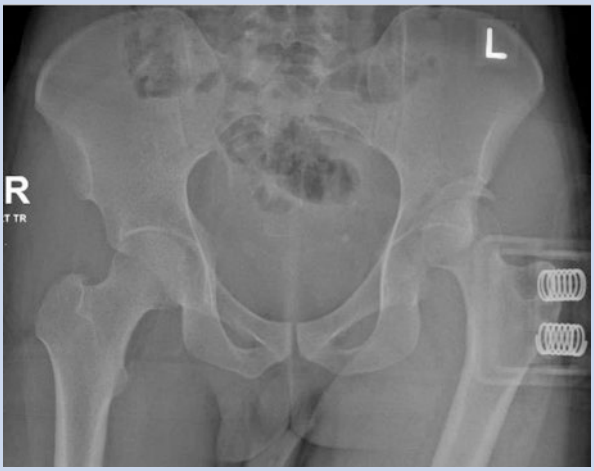
1. Hemodynamically unstable patient. Systolic BP < 90 on presentation.
2. Open fracture.
3. Old age > 60.
4. High Injury Severity Score.
5. APC III and vertical shear injury.

What is the role of tranexamic acid for control of bleeding in trauma patients?

Tranexamic acid has been shown to reduce the risk of death in bleeding trauma patients if administered within 3-h from injury (CRASH 2 trial). It is given as a 1 g bolus over 10 min followed by a 1 g infusion over 8 h.

3.2 Viva 25

A 27-year-old motorcyclist comes into A&E after being thrown from his motorbike in a collision.



- **What does this radiograph show and how would you manage this patient in the A&E?**

- **How can you classify this injury?**
- **If it is not possible to achieve a closed reduction, how would you proceed?**
- **What are the structures that can block closed reduction of posterior hip dislocation?**
- **Can you please walk me through the surgical technique for acetabular posterior wall fixation?**

What does this radiograph show and how would you manage this patient in the A&E?

This AP pelvis radiograph reveals a left hip posterior fracture dislocation, a potentially high-energy injury. In line with ATLS guidelines, my initial priority would be to ensure that all life- and limb-threatening injuries are promptly identified and addressed.

Concerning the hip injury itself, immediate closed reduction is imperative. Prior to attempting reduction, I would thoroughly assess and document the neurovascular status of the affected limb, with a specific focus on the sciatic nerve. Subsequently, I would proceed with closed reduction under sedation, while carefully evaluating hip stability through maneuvers involving flexion, adduction, and axial loading. I would apply skin traction or skeletal traction if hip joint stability remains a concern. Following reduction, I would confirm the satisfactory joint reduction by obtaining a repeat AP pelvis radiograph and repeating and documenting the neurovascular assessment. To gain a more comprehensive understanding of the injury, I would arrange for a CT scan of the hip. This CT scan would serve multiple purposes, including verifying joint reduction and congruity, identifying associated fractures such as those in the acetabulum and femoral head, assessing for marginal impaction, and identifying any intra-articular fragments.

How can you classify this injury?

Traumatic hip dislocation can be broadly classified into simple (with no associated fractures) and complex (associated with fractures involving acetabulum or proximal femur). 90% of hip dislocations are posterior. Thompson-Epstein classification describes five types of posterior hip dislocation defined by associated acetabular fracture or fracture of the femoral head.

- Type I: With or without a minor fracture
- Type II: With a large single fracture of the posterior acetabular rim
- Type III: With comminution of the acetabular rim, with or without a major fragment
- Type IV: With fracture of the acetabular floor
- Type V: With fracture of the femoral head

If it is not possible to achieve a closed reduction, how would you proceed?

If closed reduction proved unsuccessful, I would arrange to take the patient to the operating theater to undergo a closed reduction with a formal general anesthetic and muscle relaxant. I would arrange with acetabular reconstruction service, and I would arrange for a preoperative CT scan, but this should not introduce unnecessary

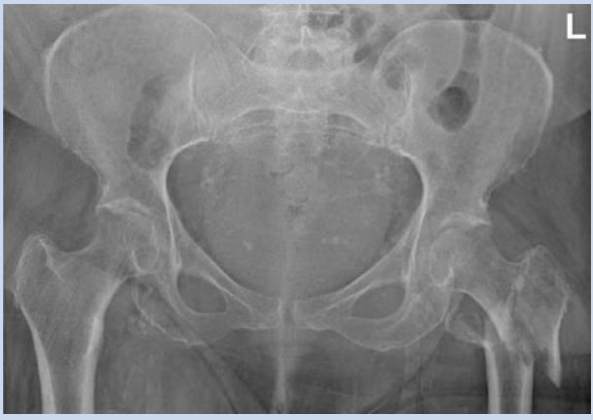
delay in moving to the operating theater. If a closed reduction fails, then open reduction is carried out with surgical fixation of acetabulum if required.

What are the structures that can block closed reduction of posterior hip dislocation?
Labrum, capsule, ligamentum teres, femoral head, or posterior wall bone fragments have all been found to block reduction of a posterior dislocation.

Can you please walk me through the surgical technique for acetabular posterior wall fixation?
In an appropriately marked and consented patient, I would position the patient lateral on a radiolucent table. I would use Kocher–Langenbeck approach, I would start the skin incision distal and lateral to posterior superior iliac spine and curve it distally over the greater trochanter toward the lateral aspect of femoral shaft. After dissection of subcutaneous tissue and fat, I would split the maximus proximally along its fibers to expose the piriformis and short external rotators. I would place a retractor between the piriformis and gluteus medius muscle, and I would incise the short external rotators at least 1.5 cm from their insertions to again avoid damage to the MCFA. I would take particular care to protect the sciatic nerve. I would carefully place additional retractors in the greater and lesser sciatic notches. Capsulotomy may be required if still intact to expose the posterior wall fragments which are further displaced to allow articular visualization and loose bodies removal. If there is a marginal impaction, then the acetabular articular surface elevated and molded to the femoral head. Gaps are filled with autograft or bone substitute. I would reduce the fractured fragments directly using reduction forceps or a ball spike pusher and hold it with K-wires. Pre-bent, under-contoured reconstruction plate 3.5 mm is used to compress the fracture with cortical screws at either end.

3.3 Viva 26

A 62-year-old lady sustained a fall from a standing height.



- **Describe the radiograph.**
- **Can you describe the deforming forces on the proximal femur in the setting of fracture?**
- **How would you manage this patient?**
- **Why did you suggest a nail to treat this fracture?**

Describe the radiograph.
This is an A/P view of the pelvis, both hips and proximal femur showing an intertrochanteric fracture with a reverse obliquity.

Can you describe the deforming forces on the proximal femur in the setting of fracture?
The proximal fragment is pulled into flexion by the forces of the iliopsoas attaching on the lesser trochanter. Additionally, the proximal fragment can be deformed into an abducted and externally rotated position as a result of the pull on the greater trochanter by the abductor muscles. However, if the lesser trochanter is detached from the proximal fragment, the classically flexed proximal fragment will not be present.

The distal fragment is pulled into an adducted position by the adductor's attachment on the femoral shaft.

These muscular-deforming forces make obtaining good reduction more difficult and challenge the fixation construct.

How would you manage this patient?
I would ensure the patient is stable hemodynamically and has received adequate analgesia. I would conduct full history and examination, paying particular attention to the mechanism of injury, comorbidities, and pre-injury functional level. I would ask specifically about previous history of cancer, as there is a high incidence of these fractures occurring secondary to pathological lesions of the bone. I would assess the patient for cognitive impairment. My examination would be focused on the identification of dehydration, neurovascular status and the soft tissue condition of the limb, check for cardiac and respiratory pathologies, and I would screen for common cancers that metastasize to bone.

I would obtain further images including lateral view and full-length femur radiographs. I would then arrange simple investigations. These would include a chest radiograph, ECG, urine dipstick, and blood tests. Blood tests would include full blood count, urea and electrolytes, liver function tests, erythrocyte sedimentation rate, bone profile, and a crossmatch for two units of blood.

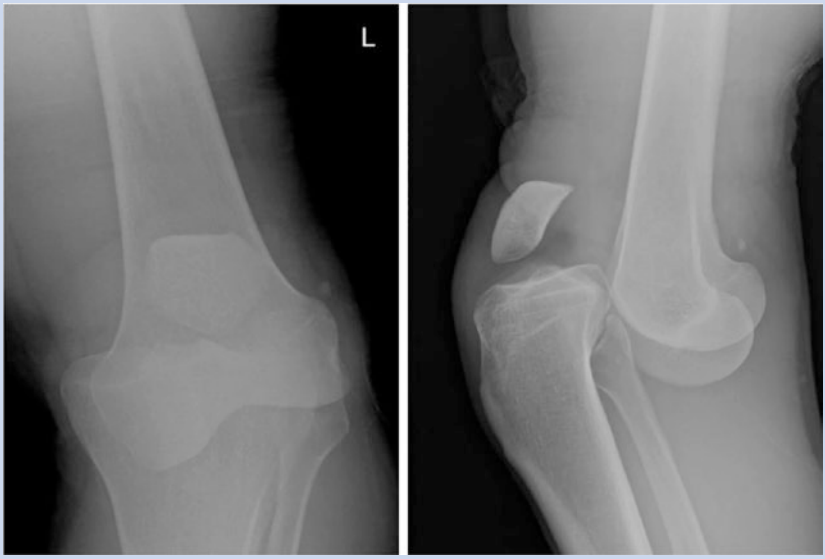
I would begin rehydration with intravenous crystalloids and ask the orthogeriatrician and anesthesiologist to assess the patient pre-operatively. Having done this, I would recommend surgical stabilization of the fracture with the use of a cephalomedullary nail and consent of the patient. I would organize the surgery to be performed on the next routine trauma list (ideally within 24 h).

Why did you suggest a nail to treat this fracture?
This is an intertrochanteric femur fracture with reverse obliquity which is considered highly unstable. Fixation with intramedullary device provides better mechanical fixation stability with reduced risk of secondary displacement.

Patients with transverse/reverse oblique trochanteric and subtrochanteric fractures operated with a SHS were found to have a higher reoperation rate compared to those operated with an IM nail as per the paper by Matre and colleagues from 2012, based on the Norwegian Hip Fracture Database.

3.4 Viva 27

A 20-year-old male athlete sustained a fall from a significant height at 19:30



- **Can you describe the radiographs?**
- **How are knee dislocations classified?**
- **How would you manage this patient?**
- **How would you measure ABI?**
- **The nurse called you at 23:30, pulses are absent. How would you proceed?**
- **What is your definitive management of this injury?**

Can you describe the radiographs?
These are AP and lateral radiographs showing an anterior knee dislocation.

Anterior knee dislocations are the most common of all directional dislocations. They are usually produced by a hyperextension mechanism.

How are knee dislocations classified?
Knee dislocation can be classified based on the direction of tibial displacement (Kennedy classification):

- Anterior: most common, due to hyperextension injury. It has the highest rate of peroneal nerve injury.
- Posterior: second most common, due to axial load to flexed knee. It has the highest rate of vascular (popliteal) injury.
- Lateral.
- Medial.
- Rotational: posterolateral is the most common one and usually irreducible.

Another classification proposed by Schenk. It is based on the number of injured ligaments. It has five types:

KD I—One cruciate ligament is involved (ACL or PCL).
KD II—Both cruciate ligaments ruptured, but no other ligamentous injury.
KD III—Both cruciate ligaments ruptured, plus either the medial collateral ligament (MCL) or lateral collateral ligament (LCL) (three ligaments injured).
KD IV—Both cruciate ligaments and both collateral ligaments ruptured (four ligaments injured).
KD V—Multiligamentous injury with periarticular fracture.

How would you manage this patient?
I would assess the patient according to ATLS guidelines. I would assess and document the skin integrity and neurovascular status of the limb.

I would perform a closed reduction of the knee through gentle traction-countertraction with the patient under conscious sedation. I would stabilize the limb in a long leg splint followed by assessment of vascular status and repeat radiographs to confirm the reduction. if pulses are present after reduction, I would measure ABI. Even in the presence of a normal neurovascular examination, the patient will require serial examinations as an intimal injury may progress to critical ischemia. I would admit the patient for observation of neurovascular status.

How would you measure ABI?
With the patient in a supine position, a blood pressure cuff is applied proximal to the ankle of the injured limb. Systolic pressure is determined using a Doppler probe,

either at the posterior tibial artery or the dorsalis pedis artery. The same measurement is taken on the ipsilateral uninjured arm. The Ankle-Brachial Index (ABI) is then calculated by dividing the systolic pressure of the injured limb by the systolic pressure of the upper limb.

Mills et al. reported a remarkable 100% specificity, sensitivity, and positive predictive value for detecting significant arterial injury when the pressure difference is less than 0.9. If there is a change in the clinical examination or if the ABI, which was previously normal, drops below 0.9, I would consider arranging an urgent arteriogram and seeking advice from a vascular surgeon.

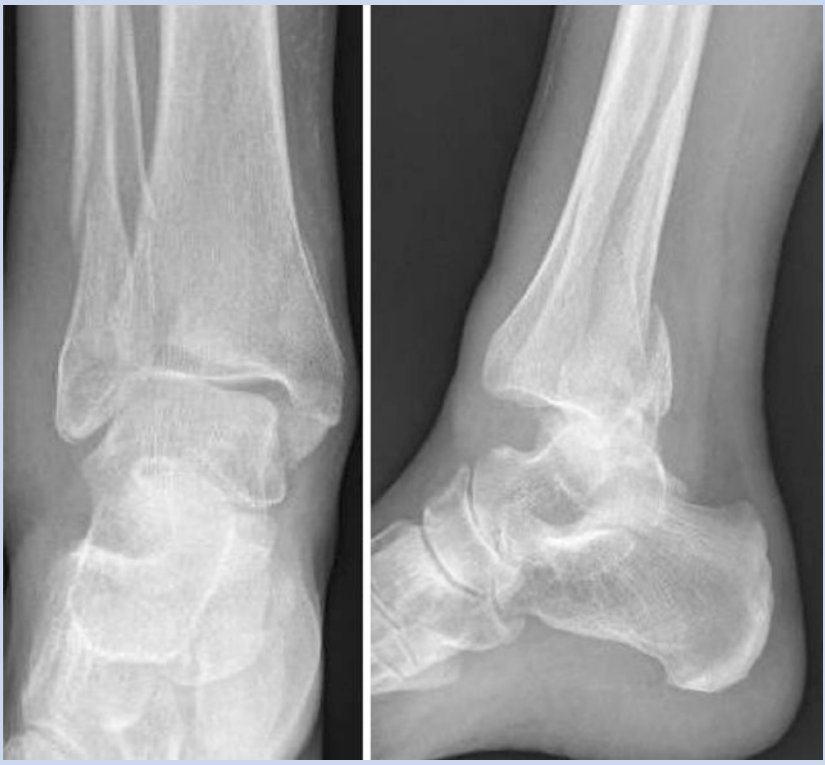
The nurse called you at 23:30, pulses are absent. How would you proceed?
First, I would confirm that the knee joint is reduced. I would perform immediate reduction and reassessment if knee joint is dislocated. Immediate surgical exploration is considered if the pulses are still absent following reduction. Ischemia time >8 h has amputation rates as high as 86%.

To ensure a secure reduction and provide additional protection to the vasculature, I would consider the application of a spanning external fixator. If the ischemia time is particularly critical, a vascular shunt may be performed before the external fixator is applied to rapidly restore blood flow. Fasciotomy is required after revascularization.

What is your definitive management of this injury?
I would arrange for this patient to have a preoperative MRI to confirm the nature of the ligamentous injury as well as rule out associated meniscal or osteochondral pathology. The management of multiligamentous knee injuries has not been well studied and remains controversial. Most authors currently recommend surgery for acute knee dislocation. Others advocate that surgical intervention should be delayed until adequate perfusion of the limb is ensured. Controversies persist regarding surgical timing, surgical technique, graft selection, and rehabilitation. No consensus exists in the literature regarding the optimal time to proceed with surgical intervention.

I would inform the patient this can be done as a single or staged procedure. I would recommend reconstruction of ACL and/or PCL. Mid-substance tears of the LCL and popliteus should be reconstructed. Fibular avulsions of the LCL/biceps femoris complex should be repaired. MCL avulsions are directly repaired using suture anchors. Mid-substance tears are treated initially in a hinged-knee brace with early motion.

3.5 Viva 28



- **Describe the radiographs.**
- **How do you classify these injuries?**
- **This is an isolated injury. How would you proceed?**
- **CT scan showed a displaced posterolateral fragment. What would be your operative plan for this patient? Describe the sequence of surgery.**
- **How would you manage and follow the patient postoperatively?**

Describe the radiographs.
These are AP and lateral radiographs of a skeletally mature right ankle demonstrating trimalleolar fracture. There is a transverse fracture of medial malleolus with a comminuted fracture of lateral malleolus. There is a fracture involving the posterior malleolus as well with posterior dislocation.

How do you classify these injuries?
Ankle fractures can be classified based on either the mechanism of injury (Lauge-Hansen) or the level of fibula fracture (Weber).

In Lauge-Hansen classification system, the first term represents the position of foot and the second to the force placed upon the foot and talus. Four major patterns are described which include: Supination adduction (SAD)—Supination external rotation (SER)—Pronation abduction (PAB)—Pronation external rotation (PER). In this case, the fibula has a spiral pattern, indicating a rotational force, so this could be SER or PER. However, the fibula fracture runs from anterior inferior to posterior superior direction which would be more in keeping with a SER pattern. In a PER pattern, the fracture runs from anterior superior to posterior inferior.

This is an isolated injury. How would you proceed?

My management would be dictated by BOAST guidelines for ankle fractures. I would take a history, asking specifically about the mechanism of injury, pre-injury mobility status, and smoking. I would check for comorbidities that may influence the treatment choice and outcomes such as diabetes, peripheral neuropathy, and osteoporosis. I would perform a circumferential examination of the limb, looking for any evidence of open injury and documenting the degree of skin damage, contusion, or fracture blistering. I would assess the circulation and sensation. I would perform an urgent closed reduction under sedation in the emergency department to avoid compromise of the overlying soft tissue.

I would apply a below knee back slab keeping the fracture aligned and all toes exposed. I would obtain radiographs to confirm adequate reduction and then proceed with admission to the ward for elevation +/− ice. I would request a CT scan for preoperative planning and discuss operative intervention with the patient.

CT scan showed a displaced posterolateral fragment. What would be your operative plan for this patient? Describe the sequence of surgery.

Given no contraindications to surgery. I would perform ORIF, either early within 1 day before swelling has occurred or allow swelling to settle down, as long as the fracture is in a satisfactory position. The principles of the definitive surgery are to restore anatomical reduction of the articular surface, restore fibular length and rotation, and achieve stability.

Using a tourniquet, and with antibiotic prophylaxis, I would perform the surgery in the prone position. I would fix the posterior malleolus and the lateral side through a posterolateral approach which gives good access to the Volkmann's fragment and optimal access to the lateral malleolus. I would fix the fibular fracture with a lag screw and an antiglide plate. I would use one-third tubular plate. I would contour the plate by narrowing the corners at its distal end to fit closely over the posterior border of the distal fibula. This contour ensures that it will not impinge on the peroneal tendons.

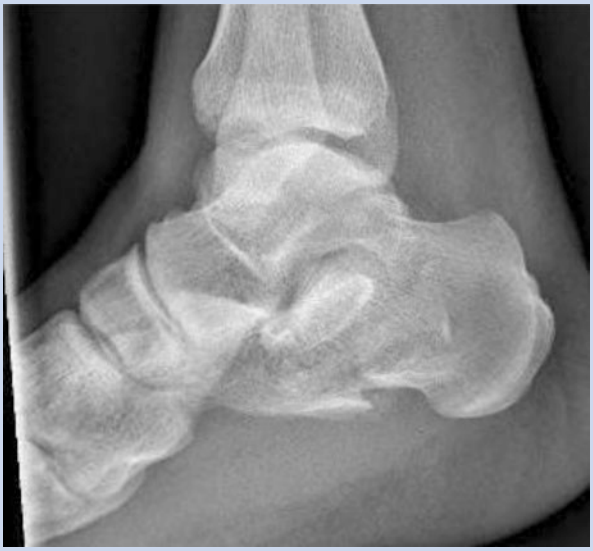
I would fix the posterolateral fragment, depending on its size, with screws from the small or mini fragment set. A small buttress plate can also be used to supplement fixation.

Lastly, the medial malleolus can be addressed through a standard medial incision by using two short (40 mm) partially threaded cancellous screws.

How would you manage and follow the patient postoperatively?
Postoperatively, I would place the patient in a below knee back-slab. I would inspect the wounds at 2 weeks and allow the patient to bear weight as tolerated in a below knee cast unless there are specific concerns regarding the stability of fixation. I would see the patient again within 6 weeks of surgery to check the reduction and fixation on radiographs, and I would replace the cast with a removable boot until walking independently.

3.6 Viva 29

A 30-year-old male patient fell from 3 m height.



- **Describe the radiograph.**
- **Are you aware of any classification system for this fracture?**
- **How would you manage this patient initially?**
- **Assuming that the patient is a smoker, how would you manage this patient definitively?**

Describe the radiograph.
This is a lateral radiograph of the ankle and hindfoot. The most obvious pathology is intra-articular comminuted fracture of right calcaneus with reduced Bohler angle.

Are you aware of any classification system for this fracture?
Calcaneal fractures can be broadly classified into intra- and extraarticular fractures based on subtalar joint involvement. Intra-articular fractures account for 75%. It can be classified by the Essex–Lopresti system as tongue type or joint depression type according to whether the fracture line on the lateral radiograph exits posterior (tongue) or anterior (joint depression) to the proximal insertion of the Achilles tendon at the posterior tuberosity. Joint depression types are further classified into four types by the Sandaers system which is based on the number of articular fragments seen on the coronal CT image at the widest point of the posterior facet.

How would you manage this patient initially?
I would begin by ensuring that the patient receives adequate analgesia for pain management. Next, I would conduct a comprehensive patient history, paying close attention to factors such as the patient's general health, including diabetes, neuropathy, peripheral vascular disease, and smoking status. Additionally, I would assess the patient's reliability and compliance with treatment recommendations. I would perform a full circumferential examination of the limb paying attention to the state of the soft tissues to identify the degree of soft tissue swelling, any open wounds or blistering. I would also perform a neurovascular examination and specifically look for signs of compartment syndrome.

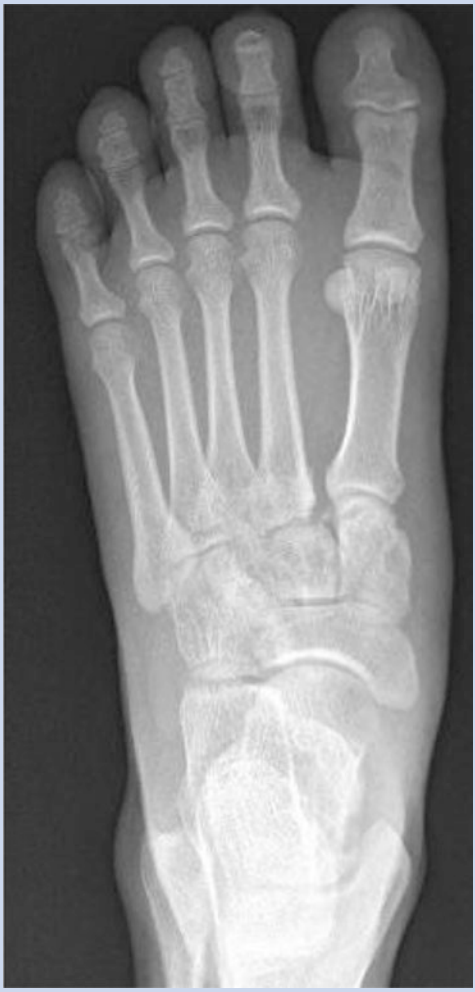
Calcaneal fractures are generally sustained from an axial load injury, I would be careful to look for associated injuries to the contralateral calcaneus as well as ankle, knee, and spine.

Assuming that this is an isolated injury without other life-threatening concerns, I would proceed to splint the limb in a below-knee back-slab.

Assuming that the patient is a smoker, how would you manage this patient definitively?
I would have a discussion with the patient explaining the nature and complexity of this injury. It's essential for the patient to understand that this type of injury can be life-changing, often leading to persistent foot problems. During our conversation, I would carefully weigh the pros and cons of both operative and non-operative treatment options. In this specific case, I would recommend non-operative treatment. My decision is based on the findings of the UK Heel Trial, which compared surgical and non-operative management of closed intra-articular calcaneal fractures. The trial demonstrated that there was no significant difference in functional outcomes between the two groups, while the surgical group experienced a higher incidence of surgical site infections and reoperations.

3.7 Viva 30

A 26-year-old man sustained an injury to his left foot while he was going down the stairs.



- **What can you see?**
- **What are the clinical and radiographic features of this injury?**
- **Are you aware of any classification system of this injury?**
- **How would you treat this injury?**
- **How will your treatment change in the case of purely ligamentous injury?**

What can you see?
This is an AP radiograph of left foot showing widening of the interval between the first and second rays. There is also a small chip of bone seen in the first intermetatarsal space. Features are suggestive of Lisfranc injury, but I would like to see further imaging in the form of oblique and lateral views.

This injury usually results from an axial load through hyper-plantarflexed forefoot.

What are the clinical and radiographic features of this injury?
Clinical features include medial plantar ecchymosis and swelling throughout midfoot.

Radiological signs include:

Widening of the interval between the first and second ray, discontinuity of a line drawn from the medial base of the second metatarsal to the medial side of the middle cuneiform and bony fragment (fleck sign) in first intermetatarsal space that represents avulsion of Lisfranc ligament from the base of second metatarsal which are all seen on the AP view.

Medial side of the base of the fourth metatarsal does not line up with medial side of cuboid on the oblique view.

Dorsal displacement of the proximal base of the first or second metatarsal on the lateral view.

Are you aware of any classification system of this injury?
Quenu and Kuss divided these injuries into three patterns: type A, indicating total incongruity; type B, partial incongruity; and type C, divergent. In 1986, Myerson further divided types B and C, with type B1 indicating partial incongruity with medial displacement; type B2, partial incongruity with lateral displacement; type C1, divergent pattern with partial displacement; and type C2, total displacement.

How would you treat this injury?
This injury represents an unstable articular injury. In the absence of any obvious contraindication I would advise that this patient should be treated operatively. I would have a careful discussion with the patient that this can be a life-changing injury and patients might end up with residual pain.

Once the patient is adequately prepared for surgery, including proper marking, informed consent, and necessary anesthetic assessments, I would position them in the supine orientation. The procedure would be conducted with the assistance of a tourniquet and image intensifier.

I would begin by making a longitudinal incision in the first inter-metatarsal space. The reduction process would proceed from the medial to the lateral direction. This would involve reducing the first cuneiform-metatarsal joint using K-wires and securing it with non-cannulated 3.5-mm screws, extending from the first metatarsal to the first cuneiform. Next, I would reduce the space between the first and second metatarsals. To achieve this, I would apply a reduction clamp between the medial zone of the first cuneiform bone and the lateral zone of the second metatarsal. This

reduction would be stabilized using a non-cannulated 3.5-mm screw, extending from the first cuneiform to the base of the second metatarsal.

Finally, I would conduct an examination under anesthesia (EUA) of the lateral column. If there is injury to the fourth and fifth rays, I would stabilize them with K-wires positioned between the metatarsal and the cuboid bone. These wires would be removed after 6 weeks.

How will your treatment change in the case of purely ligamentous injury?
There is controversy regarding the treatment of purely ligamentous Lisfranc injuries, a level 1 investigation of 41 patients with primarily ligamentous Lisfranc dislocation treated with ORIF or primary arthrodesis of the medial column of midfoot showed that primary arthrodesis has a better short and medium-term outcome than ORIF.

Further Reading

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Part III

Adult Pathology

Hip

4

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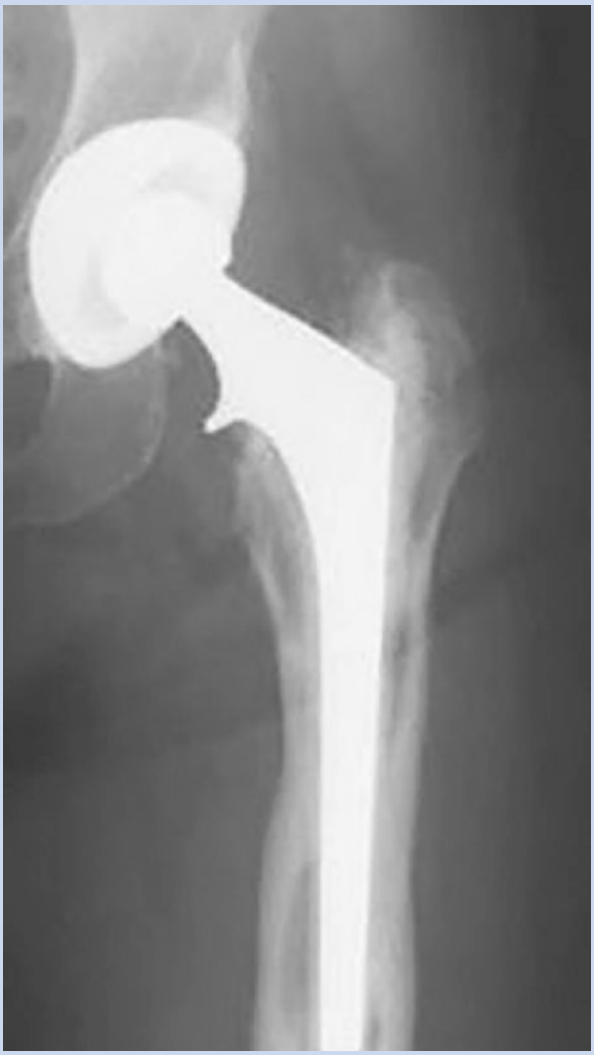
A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_4

4.1 Viva 31

A 72-year-old man reports persistent, progressively worsening pain in his hip after undergoing a total hip arthroplasty



- **What can you see?**
- **How are you going to evaluate this patient?**
- **How are ESR & CRP going to help you in the diagnosis of PJI?**

- **Are you aware of any diagnostic criteria used for the diagnosis of PJI?**
- **What is alpha-defensin?**
- **How do you classify periprosthetic hip infection?**
- **How would you treat this patient assuming that the diagnosis of chronic PJI is made?**
- **Tell more about the two-stage revision arthroplasty.**

What can you see?
This A/P radiograph of the left hip, date & name are not displayed, shows hybrid Charnley THR, there are radiolucencies in Gruen zones 1, 2, 4, 5, and 7. There is periosteal bone formation about the meta-diaphyseal region of the femur with scalloping resorption. Radiographic features are highly suggestive of infection until proven otherwise.

How are you going to evaluate this patient?
I would like to take a full history from this patient paying attention to the following points:

- The date of index operation? What was the indication for THR? And what was his expectation from the surgery?
- How was the postoperative course? Any perioperative complications?
- How was the wound condition postoperatively? Any wound healing problems?
- Any history of urinary catheterization?
- Any history of repeated courses of antibiotics?
- Was there any period of pain-free interval post THR?
- The nature of the pain should be carefully analyzed, including its onset (timing), character, location, radiation, aggravating, and relieving factors. (Pain from an infected prosthesis is typically non-mechanical and unrelated to physical activity and not relieved by rest.)
- I would enquire if there was a history of inciting event: bacteremia from a UTI, chest infection or dental extraction – colonoscopy.
- I would also like to explore more fully host risk factors for infection such as DM, rheumatoid arthritis, and immunosuppression (steroids, DMARDs). Lifestyle factors such as heavy alcohol intake and smoking.
- Medical condition of the patient and fully assess his fitness for anesthesia and surgery.

Having taken a full history, I would perform a comprehensive clinical examination

- The wound should be inspected for any signs of infection (sinus, erythema, discharge, warmth, and tenderness).

- I would also like to examine the abdomen, knee, and lumbar spine to exclude other potential sources of hematogenous infection/cause for the hip pain.
- I would perform routine blood tests including CRP and ESR.

How are ESR & CRP going to help you in the diagnosis of PJI?

- ESR (peaks 5–7 days after surgery returns to normal 90 days) and CRP (peaks 2–3 days after surgery, returns to normal at 21 days) are the baseline screening test even when there is a low suspicion of infection.
- They have high sensitivity and low specificity as they are nonspecific markers of inflammation.
- When both ESR and CRP are negative, periprosthetic infection is unlikely, however when both tests are positive PJI must be considered.
- Their combined use is a very good "rule out" test.
- However, normal ESR and CRP don't always exclude the presence of PJI.

Are you aware of any diagnostic criteria used for the diagnosis of PJI?
Musculoskeletal Infection Society (MSIS) analyzed the available evidence to propose a new definition for prosthetic joint infections. it includes major criteria and minor criteria; the minor criteria were subdivided into preoperative and intraoperative sections. The diagnosis of PJI can be made if one major criterion is present (two positive cultures of the same organism or a sinus tract communicating with the joint). If no major criterion is present, the minor criteria are scored. Minor criteria include serum ESR and CRP, synovial WBC count, PMN, CRP, and alpha defensin. A different score is assigned to each test, on the basis of pretest probability, and a score of 6 indicates infection. A score of 1 indicates the absence of infection. For patients with a score between 2 and 5 (a possible infection), additional tests and intraoperative findings should be incorporated.

What is alpha-defensin?
It is an antimicrobial peptide released by activated neutrophils. It performs well in challenging situations like culture-negative PJI, systemic inflammatory conditions, and concurrent antibiotic. However, alpha-defensin has limitations, with low positive predictive value (PPV) and specificity in the setting of metallosis or adverse local tissue reaction.

How do you classify periprosthetic hip infection?
A classification system for PJI was proposed by **Coventry** in 1975 and then modified by **Fitzgerald**. It is divided into:

- Acute postoperative infections occurring within three months of the surgery.
- Delayed infections that appear between three months and two years after the surgery.
- Late infections that occur more than two years after the surgery

How would you treat this patient assuming that the diagnosis of chronic PJI is made?
The choice of treatment of chronic PJI depends on the following factors:

- Physiological status of the patient and the overall health characteristics.
- History of PJI in the current joint and all other joints.
- Characteristics of the infection organism.

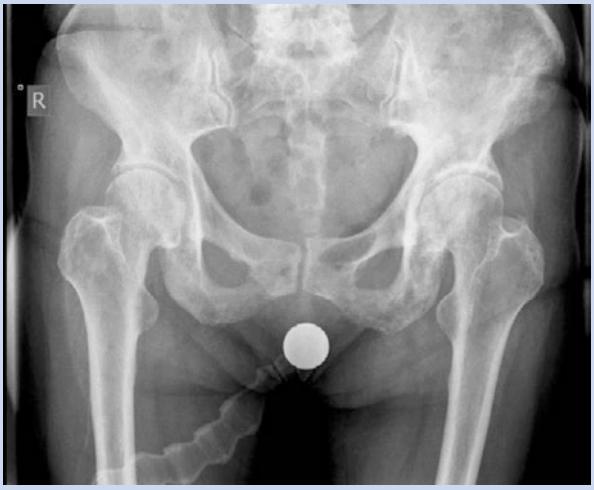
Single-stage revision arthroplasty could be considered if the infective organism was of low virulence with known antibiotics susceptibility or if the patient had multiple comorbidities and the surgical stress of having a two-stage procedure might compromise the patient. On the other hand, two-stage revision should be considered in case of more severe infections or virulent organisms, as the success rate of a single-stage procedure is much less in these situations.

Tell more about the two-stage revision arthroplasty
The first stage involves complete removal of all prosthesis and foreign materials, debridement of surrounding infected soft tissues, and placement of an antibiotic-impregnated cement spacer to maintain the integrity of soft tissue. The second stage involves removal of the spacer and any additional necrotic tissues, thorough irrigation, and placement of new prosthetic implants.

The duration of antibiotic treatment between the two stages remains controversial. Current practice suggests delaying the second stage for at least 6 weeks depending on good clinical progress with antibiotics and wound healing. A number of surgeons re-implant at 3 months, treating the patient with 6 weeks antibiotics and then a further 6 weeks without antibiotics, regularly monitoring the CRP/ESR for any signs of elevation and checking clinical progress for any signs of reoccurrence of infection such as sinus discharge or increasing hip pain.

4.2 Viva 32

These are the radiographs of a 78-year-old female who is complaining of predominantly left-sided hip pain.



- **What is Paget's disease?**
- **What are the clinical and radiological manifestations of Paget's disease?**
- **What are the preoperative considerations and technical issues of performing THA in Paget's disease?**
- **What type of hip replacement would you use?**

What is Paget's disease?

Paget's disease is a metabolic bone disorder of unknown etiology characterized by an increase in osteoclastic bone resorption and compensatory disorganized osteoblastic new bone formation. There is accelerated but chaotic bone remodeling in which the bone is biomechanically weak and prone to deformity and fracture.

The disease can be divided into three major phases, lytic, mixed lytic/sclerotic, and sclerotic, each of which is associated with distinctive clinical, radiological, and pathological features.

What are the clinical and radiological manifestations of Paget's disease?

The clinical features of Paget's disease include:

- An enlarged and deformed skull can lead to increased intracranial pressure and hydrocephalus. Progressive closure of skull foramina can lead to cranial nerve

deficits such as facial palsy (narrowing of neural foramina), hearing loss or blindness (pressure on optic nerve), headache, and vertigo.
- High cardiac output secondary to increased bone vascularity (rare).
- Compression fractures of the vertebral body.
- Pagetic spinal stenosis, defined as compression of the spinal cord, cauda equina, or spinal nerves by expanded pagetic bony tissue of the spine.
- Insufficiency fractures.
- Bowing deformities of long bones occur due to softening. Sabre tibia (forward bowing of the tibia).
- Paget's sarcoma less than 1%: Osteosarcoma, chondrosarcoma, malignant fibrous histiocytoma, and giant cell tumors all have been reported with Paget's disease.

Radiological features:

- ***The skull*** is involved in 29–65% of cases: most commonly involving the frontal bone, producing well-defined geographic lytic lesions in the skull. It is seen in the early or lytic phase. At a later stage a "cotton wool appearance" represents mixed lytic and plastic pattern of thickened calvarium.
- Lateral radiographs of the ***lumbar spine*** demonstrate a "picture-frame" vertebral body that is secondary to severe osteoporosis centrally and a thickened, sclerotic cortex or Ivory vertebra (increased density).
- Advanced disease in the ***long bones*** is characterized by coarsened trabecula, bony sclerosis, loss of corticomedullary differentiation, bony enlargement, and deformity. The femur develops a lateral curvature whilst the tibia develops an anterior curvature that may result in fracture. Fine cracks may appear (stress fractures) which resemble Looser zones but occur on the convex bone surface.
- ***Protrusio deformity*** of the pelvis is a common occurrence with advanced Paget's disease – Both iliopectineal (**Brim sign**) and ilioischiatic lines are thickened.

What are the preoperative considerations and technical issues of performing THA in Paget's disease?
It needs **Multidisciplinary approach:**

1. As bone pain is common in Paget's disease and does not necessarily improve with THA a diagnostic local anesthetic injection to rule out concurrent bone pathology may be indicated. It is also important to exclude referred pain from spinal stenosis or radiculopathy and other causes of musculoskeletal pain.
2. I would make a referral to one of my rheumatoid colleagues for a Pamidronate (Aredia) injection (6 weeks to 3 months). This is a bisphosphonate, which is a potent inhibitor of osteoclastic activity, and hence bone resorption. Preoperative treatment with bisphosphonate or calcitonin has been used to reduce the incidence of intraoperative bleeding, HO, and loosening. It lowers bone turnover by decreasing activity and number of osteoclasts.

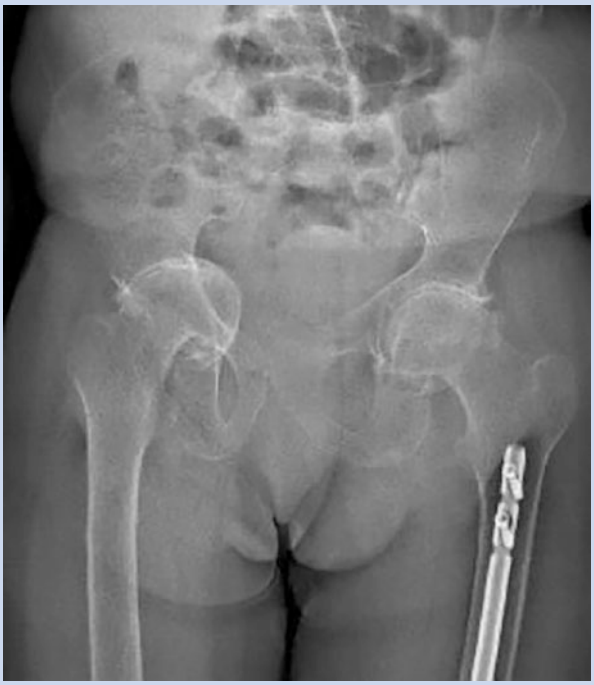
3. I would make referral to anesthetic clinic for preop assessment—ECHO (they have high cardiac output)
4. There is a potential for significant intraoperative bleeding from hypervascular and osteoporotic bone which may require additional cross-matching of blood–cell saver.
5. Proper preoperative templating and planning is necessary to size an enlarged medullary canal and determine the correct component size.
6. There is increased risk of HO in this condition, therefore prophylaxis should be considered.
7. Trochanteric osteotomy may be required for adequate exposure because of a broad spectrum of deformities.
8. Marked deformity of the proximal femur with coxa vara or anterolateral bowing of the femoral shaft may require corrective osteotomy and the use of modular stems prior to THA.
9. The presence of dense sclerotic bone may make reaming and bone preparation difficult, sharp reamer will be required to shape the femoral canal. Burrs may be needed to enter the bone prior to reaming and/or broaching.
10. If protrusio acetabulum exists: ream to expand the periphery without deepening the socket to avoid causing added protrusion—dislocation of the hip can be extremely difficult, and the neck may need to be cut in situ.
11. As Paget's bone is brittle there is a higher risk of both intraoperative and postoperative fracture.

What type of hip replacement would you use?

I prefer cementless components especially when bone is very sclerotic, or a concurrent osteotomy is done. Extremely sclerotic bleeding bone will make interdigitation of cement difficult and cement extravasation into the fracture gaps may occur after osteotomy. If using a cementless cup the use of adjuvant acetabular screws is recommended.

Parvizi et al. reported on 21 cementless THA implanted against pagetoid bone; all were stable and demonstrated radiographic evidence of ingrowth at a 7-year follow-up.

4.3 Viva 33



- **What can you see?**
- **What is Protrusio acetabuli?**
- **What are the types of Protrusio acetabuli?**
- **How do you grade protrusio?**
- **What is the difference between Coxa profunda and Protrusio acetabuli?**
- **What are the technical considerations in THA?**

What can you see?
The is an A/P radiograph of the pelvis, both hips and proximal femur show right protrusio acetabuli which is noted by: sunken acetabulum, there is medial displacement of both the medial wall of the acetabulum and femoral head, femoral head is medial to ilio-ischial line (a line that connecting the most lateral aspect of the pelvic rim and the lateral border of the obturator foramen). There are osteoarthritic changes of left hip. The bones appear osteopenic.

What is Protrusio acetabuli?
It is medial intrapelvic displacement of the medial wall of the acetabulum. Normally on an AP radiograph the medial wall of the acetabulum lies 2 mm lateral to the ilioischial line in a male and 1 mm medial to this line in a female. Protrusio is present if the medial wall of the acetabulum is 3 mm or more medial to the ilioischial line in a male or >6 mm medial to it in a female.

What are the types of Protrusio acetabuli?
Primary:

- It is termed Otto's pelvis.
- More common in females 10:1 ratio.
- Idiopathic. Attributed to changes in triradiate cartilage ossification.
- Secondary (due to softening of the bone):
- Osteogenesis imperfecta.
- Osteomalacia.
- Rickets.
- Rheumatoid disease.
- Marfan's disease.
- Ankylosing spondylitis.
- Paget's disease.
- Infection. TB.
- Trauma. Iatrogenic fracture during surgery.

How do you grade protrusio?
Hirst grade (based on the distance of the medial wall of the acetabulum to Kohler's line)

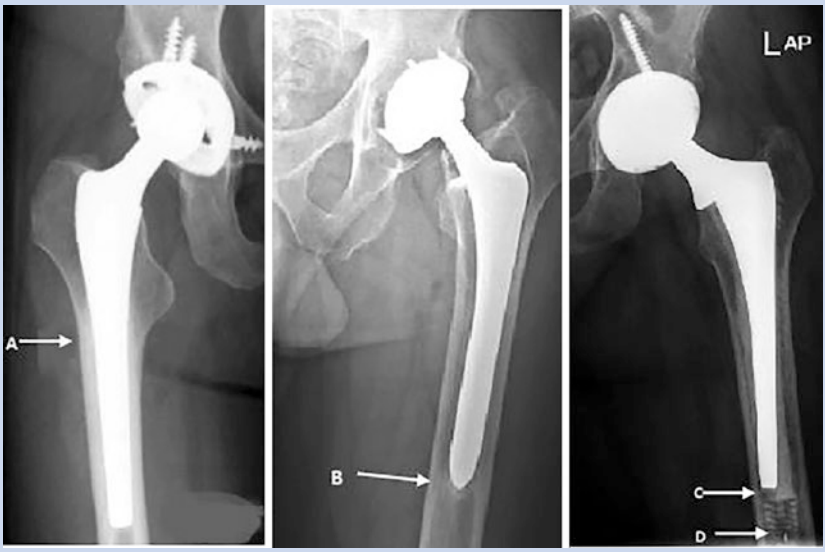
- Grade I: mild. 5–10 mm.
- Grade II: moderate. 10–15 mm.
- Grade III: severe >15 mm.

What is the difference between Coxa profunda and Protrusio acetabuli?
Coxa profunda is the mildest form of the protrusion acetabuli, in which the medial wall of the acetabulum touches the ilio-ischial line or slightly medial to it.

What are the technical considerations in THA?
- Because of the medial migration of the femur, the sciatic nerve is often nearer to the joint than normal and should be identified early and protected.
- Dislocation of the hip is difficult, femoral neck should be osteotomized in situ to avoid fracture of the femur, and the head is then removed from the acetabulum in a retrograde manner.

- Avoid over reaming of the acetabulum, the medial wall is thin. Bone grafting of acetabulum socket is needed for the medial wall to restore the offset.
- The cup to be placed in more lateral position and fixed with acetabular screws to avoid medial migration.
- Femoral stem with increased offset reduces the risk of femoral–pelvic impingement especially if short neck is used to equalize leg length.

4.4 Viva 34



- **What do these letters represent on the shown radiographs?**
- **What is the purpose of placing centralizer and cement restrictor in cemented femoral prosthesis?**
- **Discuss the characteristic features and biomechanical principles of the cemented femoral stems, Exeter vs Charnley.**
- **What are the types of fixations of cementless femoral stem?**
- **What type of total hip arthroplasty would you prefer to use and why?**

What do these letters represent on the shown radiographs?

A. spot weld and cortical hypertrophy
B. early distal pedestal formation
C. femoral stem centralizer that appears as a radiolucent bubble distal to the stem tip
D. cement restrictor

What is the purpose of placing centralizer and cement restrictor in cemented femoral prosthesis?

Femoral stem centralizer guides the femoral stem to a neutral position within the cement and improves the quality of the distal cement mantle.

Cement restrictor is a part of third generation cementing technique that helps to increase the pressurization and penetration of cement into the cancellous bone proximally which enhances prosthetic stability.

Discuss the characteristic features and biomechanical principles of the cemented femoral stems, Exeter vs Charnley

The Exeter stem is collarless, highly polished, and tapered in two or three planes. The stem is allowed to subside initially (about 1.6mm/year) because of the cement creep, radial compressive forces are created in the adjacent cement sealing the stem-cement interface and transferred to the bone as hoop stress. This taper means that the hoop stresses which are perpendicular to the long axis of the implant remain applied to the bone as the hip subsides.

Charnley stem has a collar to prevent subsidence, works in a composite beam manner, (1) shape closed prosthesis, (2) works as a single unit between stem, cement and bone relies on friction to maintain the position of the stem within the cement mantle, (3) exhibits a 3-point fixation, and (4) exhibits a stress shielding passing the calcar going distally.

What are the types of fixations of cementless femoral stem?

Fixation of cementless stem is classified as:

Bone ingrowth which characterized by:

- No subsidence.
- No reactive line adjacent to the porous-coated portion.
- Endosteal hypertrophy (spot welds) at the distal limit of the porous coating.
- Variable amount of proximal bone resorption due to stress shielding.

Stable fibrous:

- No progressive implant migration.
- No endosteal hypertrophy noted at the distal limit of the porous coating.
- There may be a reactive line adjacent to the porous-coated portion, which is parallel to the surface and separated from it by a narrow interval, but it does not widen with time.

Unstable:

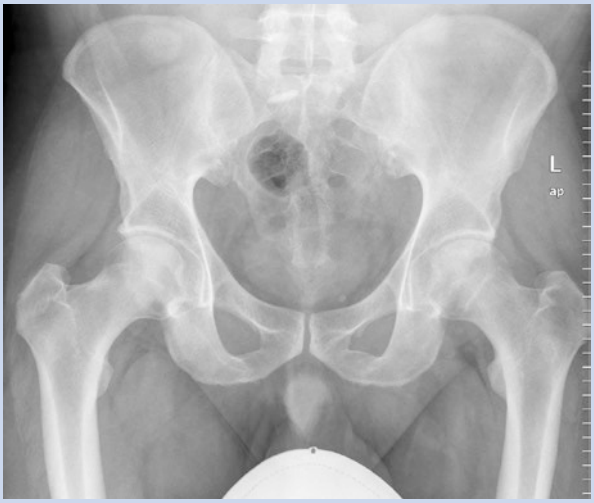
- Progressive component migration.
- Formation of a distal pedestal at the stem tip (is an endosteal new bone formation below the distal end of the stem and it usually extends over 50% of the canal).
- Calcar hypertrophy. (Distal pedestal formation and calcar hypertrophy imply prosthesis-to-bone stress transfer away from the porous coating metaphyseal part of the implant and are associated with instability.)
- The presence of reactive lines that tend to diverge from the component surface and progressively widen with time.

What type of total hip arthroplasty would you prefer to use and why?
I would use hybrid THR "Exeter cemented stem"

- It is a hip system that provides me with the ability to deal with anatomical variants and achieve all the primary technical goal of THR.
- It is a system which allows me to incorporate my philosophy.
- My training has been with Exeter, and I am familiar with instruments.
- It is a system with a proven track record for long-term survival studies and patient-reported outcome measure. It has 13A* ODEP.

4.5 Viva 35

A 37-year-old male patient with 6-week history of left hip pain. No history of trauma.



- **What can you see?**
- **What is the etiology and what risk factors are associated with this condition?**
- **How is AVN classified?**
- **What is crescent sign?**
- **What is the Kerboul necrotic angle and its importance?**
- **What are the factors that determine decision-making for treatment?**
- **What are the goals of treatment and what are the available treatment options?**

What can you see?
A/P pelvis radiograph showing subarticular cystic-like lesion in the superolateral aspect of both femoral heads surrounded by bony sclerosis, more obvious in the left femoral head. Features suspicious of bilateral hip AVN. However, there is no evidence of subchondral collapse.

What is the etiology and what risk factors are associated with this condition?
The etiology of AVN remains unclear and is likely to be multifactorial. Factors thought to contribute to the disruption of the microcirculation include:

- Trauma.
- Caisson disease (dysbaric osteonecrosis, also known as divers' disease, caused due to release of nitrogen gas bubbles that impinge the blood vessels).
- High alcohol intake.
- Autoimmune disease: systemic lupus erythematosus (SLE).
- Corticosteroid usage.
- Ionizing radiation.
- Haemoglobinopathy (sickle cell anemia).
- Gaucher's disease.
- Hypercoagulation disorders (Thrombophilia—low protein C or S).
- Idiopathic (40%).

How is AVN classified?
Several classification systems for AVN exist.

- Radiographic staging of AVN was first proposed by Ficat and Arlet in 1968 and is one of the simplest to use, includes 4 stages:
  - Stage 1: no bony changes seen on plain X-ray. Known as the silent hip.
  - Stage 2: sclerotic and cystic changes within the femoral head. A: no collapse. B: crescent sign (subchondral collapse).
  - Stage 3: flattening of the femoral head.
  - Stage 4: secondary osteoarthritis with decreased joint space and articular collapse.

It doesn't grade the severity of the disease among certain phases in disease progression.

It doesn't quantify the size and extent of the lesion.

- Steinberg expanded the staging system into seven stages and quantified the amount of involvement of the femoral head:
  - Mild A (<15%)
  - Moderate B (15–30%)
  - Severe C (>30%)

It is considered more useful than Ficat because it grades the severity and extent of the involvement, both of which are thought to affect prognosis.

What is crescent sign?
It is a linear subcortical lucency, situated immediately beneath the subcortical bone, representing a fracture line and impending femoral head collapse.

What is the Kerboul necrotic angle and its importance?
The Kerboul necrotic angle is used to calculate the size of the necrotic segment. It is the sum of the angle of the necrotic segment as measured on both the anteroposterior and frog-lateral radiographs. Patients with a Kerboul angle >200 more commonly have poor results with certain bone-preserving procedures.

Modified Kerboul necrotic angle is measured on mid-sagittal and mid-coronal MRI.

What are the factors that determine decision-making for treatment?
The choice of management depends on:

- Patient age
- Cause
- Extent of the disease (Kerboul angle)
- Rate of progression

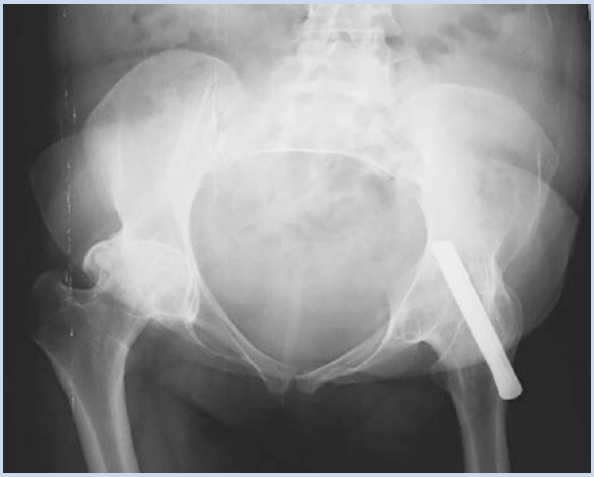
What are the goals of treatment and what are the available treatment options?
Goals of treatment:

- Address and eliminate (if possible) underlying risk factors (if present)
- Relieve pain
- Improve function
- Prevent or delay progression
- Minimize morbidity

In the absence of level I evidence, it is not possible to make definitive treatment recommendations. However, based on the available data, treatment recommendations can be summarized as follows:

- Patients with small asymptomatic lesions → follow up with serial clinical and radiographic examination.
- Patients with symptomatic osteonecrosis with small lesions in pre-collapse hips should be treated with → head-sparing procedure (Core decompression—Rotational osteotomies).
- Large lesions in pre-collapse hips → may be treated with a head-sparing procedure in younger patients, but it is reasonable to consider THA in older patients.
- Patients with collapse of the femoral head should not have a femoral head-saving procedure → THA is the most reliable option for these patients.

4.6 Viva 36



- **What position would you choose to arthrodese a hip?**
- **What are the indications for this procedure?**
- **What are the indications for conversion to THA?**
- **What are the preoperative considerations in conversion to THA?**
- **What are the principles and considerations of surgical technique?**
- **What are the results and outcomes of hip arthroplasty following arthrodesis?**
- **What effect does hip arthrodesis have on ipsilateral TKR?**

What position would you choose to arthrodese a hip?
Position of fusion:

- 0–5° adduction. Abduction creates pelvis obliquity.
- 5°–10° external rotation.
- 20°–30° flexion. Insufficient flexion makes sitting extremely difficult and excessive flexion may lead to LLD and lumbar lordosis.

This position is designed to minimize excessive lumbar spine motion and opposite knee motion which help minimize pain in these regions.

What are the indications for this procedure?
1. Severe unilateral hip disease in young adults: Hip arthrodesis can be considered for young individuals who engage in physically demanding work and have end-stage unilateral hip disease.

2. Last resort after failed total hip arthroplasty (THA): When total hip arthroplasty (THA) has been attempted and has not yielded successful outcomes, hip arthrodesis can be considered as a final option.

What are the indications for conversion to THA?

- Sever persistent low back pain (make sure that the pain is not caused by other pathology).
- Increasing ipsilateral knee pain.
- Contralateral hip disease.
- Painful pseudoarthrosis of the hip.
- Difficulty in performing ADL mainly involving hip flexion such as bending, putting shoes and socks on, and tying shoelaces.

What are the preoperative considerations in conversion to THA?

It is a technically demanding procedure due to disturbed anatomy. Preoperative work up includes:

- The origin of pain should be accurately defined, and the functional demands and expectations of the patient should be explored.
- Clinical assessment of hip abductor function (firing).
- Pre-operative investigation of abductors—MRI + EMG studies. If non-functional → constrained prosthesis.
- CT scan can be helpful for identifying bone stock and hip abductor muscle mass.
- The original reason why the arthrodesis was performed should be sought. If the arthrodesis was performed following infection, the potential for reactivation of dormant infection must be considered and appropriate biopsies taken pre- or intra-operatively.
- Meticulous preoperative planning for acetabular position abductor moment arm restoration and leg length restoration.
- Prophylactic management for HO (NSAIDs).

What are the principles and considerations of surgical technique?

- Exposure is difficult because prior incisions, distortion anatomic planes, medialization hip center, and soft-tissue contractures.
- Identify and preserve hip abductor muscles.
- Sciatic nerve often embedded in scar tissue. Line of neck resection should be carefully identified, avoid cutting into greater trochanter or dividing the posterior acetabular wall.
- Identification orientation acetabulum can be difficult. Careful acetabular reaming to preserve anterior and posterior columns. Perform concentric reaming of the acetabulum to achieve medialization and sizing of the component.
- Optimize leg length and restore ideal femoral offset to avoid impingement and instability
- Adductor tenotomy, iliopsoas muscle release, and anterior capsulectomy often required to correct severe contractures.

What are the results and outcomes of hip arthroplasty following arthrodesis?
CORR (2011) retrospective comparative: Conversion of hip arthrodesis to THA provides improvement of hip function, good survival, and high level of patient satisfaction comparable primary THA.

What effect does hip arthrodesis have on ipsilateral TKR?
TKA in patients with an ipsilateral hip fusion leads to a reduced ROM and the frequent need for manipulation under anesthesia (MUA) because of stiffness. Moreover, these artificial joints function under abnormal overstress leading to early failure. The only exception to performing a TKA before converting the fused hip would be a patient with a satisfactorily positioned hip in whom abductor muscle function was questionable. In these patients, the results of THA are known to be inferior, with poor gait patterns and a decreased likelihood of adequate knee pain relief. If the hip is fused in a poor position and the patient has significant knee pain, the conversion THA is preferable because of the notably inferior results of a TKA in that setting.

Further Reading

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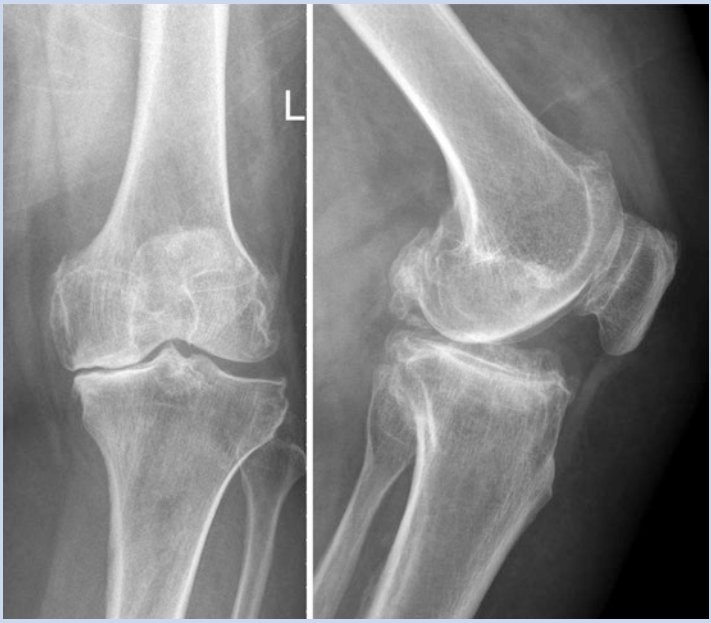
Knee

5

Shamsi Abdul Hameed and Shanmugam Karthikeyan

5.1 Viva 37



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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_5

- **Describe the radiograph.**
- **What is the pathogenesis of this condition?**
- **How are you going to manage this patient?**
- **What are the technical goals for TKR?**
- **Which knee system would you use and why?**
- **Would you use cemented or uncemented TKA?**
- **Would you do patella resurfacing?**

Describe the radiograph

These are A/P & lateral radiographs of left knee demonstrating features of degenerative changes noted by medial joint space narrowing—subchondral sclerosis and osteophytes formation. The leg is in overall varus alignment.

What is the pathogenesis of this condition?

In the early stage of OA, there is ↑ water content which leads to disruption of the integrity of the collagen network and ↓ proteoglycan level that leads to ↓ stiffness of the matrix & the young's modulus of elasticity → cartilage tissue becomes more susceptible to further damage → chondrocytes respond to tissue damage by releasing mediators to increase proliferation. As osteoarthritis progresses, however, the proliferative response decreases and the level of proteoglycans eventually drops very low, causing the cartilage to soften and lose elasticity and thereby further compromising joint surface integrity.

How are you going to manage this patient?

Firstly, I would like to obtain a detailed history from this patient and conduct physical examination.

I would like to establish from the history:

- The main complaint of the patient. The real source of the pain must be displayed (spinal disease-ipsilateral hip pathology-peripheral vascular disease-bursitis)
- Disability & ADLs
- What has been done for the patient?
- Patient's medical condition
- Any source of infection (dental-dermatological-urological-RTI)
- Patient's expectation (patient satisfaction rates were reported 80% after TKA)

I would like to establish from the physical examination:

- Weight (BMI)
- Skin condition & previous surgical incision
- Alignment of lower limbs
- Patellofemoral tracking
- Range of motion
- Stability
- Neurovascular status
- Examination of hip joint and spine

The management of OA of the knee can be divided into non-surgical and surgical options. Non-surgical alternatives should be exhausted before surgical treatment is offered. Conservative measures include *(As per the NICE guidelines):*

- Acetaminophen, NSAIDs, and Tramadol should be the first analgesics offered.
- Weight reduction: Prospective study (BJJ, 2008) from Scotland conducted in morbidly obese patients Vs pt. BMI <30, followed up for 3 years revealed inferior Knee Society Scores—higher incidence of radiolucent lines on radiographs—higher rate of complications—Inferior survivorship.
- Physio: Low-impact aerobics and quad strengthening to optimize knee kinematics
- Intra-articular injection:
  - Steroids: Reduce inflammation; increase viscosity of synovial fluid—Methylprednisolone (Depo-Medrone) Low solubility—Risk of peri-prosthetic infection if done within 3 months of TKR.
  - Hylan (Synvisc): 3 injections of 2 ml each over 3 weeks or 1 injection of 6 ml, 2007, RCT, JBJS: No differences between patients treated with intra-articular injections of Hylan and those treated with corticosteroid with respect to pain relief or function at 6 months.

Assuming failure and exhaustion of all non-operative measures, TKA would be the treatment of choice.

What are the technical goals for TKR?

- Restoration of mechanical alignment by making cuts perpendicular to mechanical axis
- Restoration of joint line
- Coronal and sagittal planes balancing
- Restoration and preservation of Q angle

Which knee system would you use and why?

- I need a knee system which can be used for all anatomic variants (varus, valgus…). I need a system that incorporates stems/augment/offset trays for all complex knees.
- I need a system which allows me to incorporate my philosophy of balancing the knee.
- I need a system with a proven track record for long-term survival studies and patient-reported outcome measures.
- High ODEP (Orthopedic Data Evaluation Panel) rating. It provides an approved list of prostheses that meet the revision rate standard at 10 years set out in NICE guidance.
- I would use PFC Sigma: 13* with 2.7% revision rate at 10 years.

Would you use cemented or uncemented TKA?

At present, the gold standard is cemented TKA, supported by evidence.

I will use standard viscosity cement.

Meta-analysis (BJJ, 2009) about survival and clinical function of cemented and uncemented prostheses in total knee replacement: Improved survival of the cemented compared to uncemented implants, with no statistically significant difference in the mean Knee Society Score.

NJR data 10-year revision risk is 3.5% for cemented and 5% for uncemented.

Would you do patella resurfacing?

This is a controversial topic, however, because I practice evidence-based medicine, I do know at the present time long-term results showed no difference in outcomes between resurfaced and nonresurfaced knees (multicenter RCT JBJS am 2011—showed no difference in 5-year Oxford scores)

I know that the patella is important for quadriceps function and the height of the patella is important to optimize that.

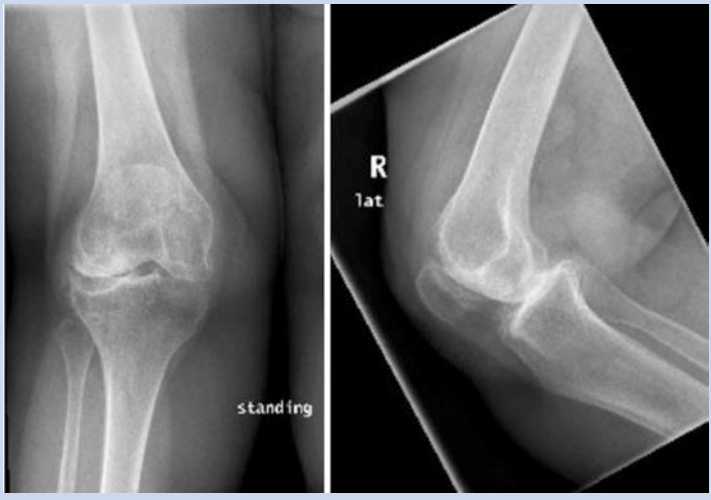
I don't routinely resurface the patella, I would do patelloplasty with diathermy.

I would consider resurfacing in case of:

- Advanced osteoarthritis of patellofemoral joint with preoperative patellofemoral pain
- Rheumatoid arthritis
- Patella maltraking after excluding all other factors.

5.2 Viva 38

This is a radiograph of a 69-year-old lady complaining of long-standing right knee pain. She has been referred to your clinic to be considered for total knee arthroplasty.



- **Describe the radiograph.**
- **What conditions are associated with this pattern of joint disease?**
- **How would you evaluate a patient with a valgus knee?**
- **How can you classify valgus knee?**
- **What are the problems and the intraoperative technical considerations associated with knee valgus deformity?**
- **What form of knee replacement would be appropriate? What approach would you use?**
- **Describe the method of lateral ligament complex release and balancing the knee for valgus knees.**

Describe the radiograph
These are weight-bearing A/P and lateral radiograph of right knee showing advanced osteoarthritis noted by loss of joint space, osteophyte formation, subchondral sclerosis, and cysts associated with valgus deformity

What conditions are associated with this pattern of joint disease?
Valgus deformity of the knee is commonly seen in:

- Inflammatory arthritis
- Primary osteoarthritis—most common
- Women (physiological valgus)
- Overcorrection from a high tibial osteotomy for a preexisting varus deformity
- Metabolic disorder:
  - Rickets
  - Renal osteodystrophy
- Post-traumatic:
  - Tibial malunion
  - Physeal arrest
  - Tibial plateau fracture

How would you evaluate a patient with a valgus knee?
This can be divided into clinical and radiographic evaluation.
Clinical evaluation includes:

- History:
  - Pain: onset, duration, nature, aggravating and relieving factors, radiation, involvement of other joints—associated symptoms (stiffness)
  - Impact on activities of daily living: ability to go up and down stairs—walking distance—walking time without support—walking time with support
  - What treatment the patient received: analgesics, injection, physiotherapy
  - Chronic illnesses and previous surgical procedures

- Physical examination:
  - Alignment and gait (assess the dynamic instability—recurvatum)
  - Deformity (measure the valgus deformity and check if correctable—look for any recurvatum—FFD)
  - ROM
  - Check the competency of ligaments (collaterals).
  - Check NV status (peroneal nerve)
  - Examine other joints
  - Look for any systemic features of inflammatory arthropathy
- Radiographic evaluation:
  - Standing long-leg films
  - A/P weight bearing (look for lateral femoral condyle hypoplasia—medial joint opening—tibial plateau bone defects
  - Lateral (look for posterior condyle hypoplasia—patellar height (baja associated with valgus knee)
  - Skyline views (look for PF arthritis—patellar maltracking)
- If osteotomy or UKA is considered
  - Rosenburg (to assess medial/lateral cartilage wear)
  - Varus stress (assess if deformity is correctable to neutral)

How can you classify valgus knee?

Valgus knee deformity can be classified according to Ranawat:

- Grade I is <10° valgus deformity (normal valgus angle is ~6°), correctable alignment with stress, and intact MCL, and this type accounts for >80% of all valgus knees.
- Grade II is an angle 10°–20°, MCL is attenuated but has a firm endpoint, and this type accounts for 15% of valgus knees.
- Grade III is a valgus angle >20° and absent or severely attenuated MCL. This grading scale helps to determine the type of implants and correction that is required.

What are the problems and the intraoperative technical considerations associated with knee valgus deformity?

In valgus knee:

- The medial structures are stretched while lateral and posterior structures are contracted. The release of lateral and posterior structures may result in increased extension gap requiring a thicker insert which may elevate the joint line. Excessive PCL release usually requires cruciate sacrificing implants to balance the knee.

- The lateral femoral condyle is deficient; therefore, the standard posterior condylar referencing can result in internal rotation of the component due to lateral condyle hypoplasia. AP axis (Whiteside line) or epicondylar axis are used to prevent malrotation in the form of internal rotation.
- There is a risk of patellar maltracking which can be addressed through lateral release using outside in technique.
- With correction of significant valgus deformity, one has to watch for peroneal nerve palsy in the postoperative period which can be due to traction of the nerve or direct injury while performing pie-crusting technique.

What form of knee replacement would be appropriate? What approach would you use?

The type of knee implant required depends on the extent of the soft tissue release and the competence of the ligaments following the release. In the valgus knee, the competence of the medial collateral ligament (MCL) is a factor that should be assessed preoperatively.

- If the MCL and the lateral collateral ligament (LCL) are competent after the soft tissue releases and bone cuts have been made, an unconstrained TKR prosthesis may be used.
- If the PCL is attenuated and/or if the joint line is significantly altered, a posterior stabilized design is recommended (PCL substituting option should always be available at the time of surgery).
- If the MCL is attenuated, or the LCL has to be released, then a more constrained prosthesis will be required.

In my practice, I would use the medial parapatellar approach because that's what I know and I'm comfortable with. It gives good access to the whole knee and better soft tissue cover. I may add rectus snip if there is a difficulty to evert the patella. I'm aware about the principles behind using lateral parapatellar approach which gives easier access and preserves the neurovascular supply to the extensor mechanism. It may be indicated in case of severe valgus deformity.

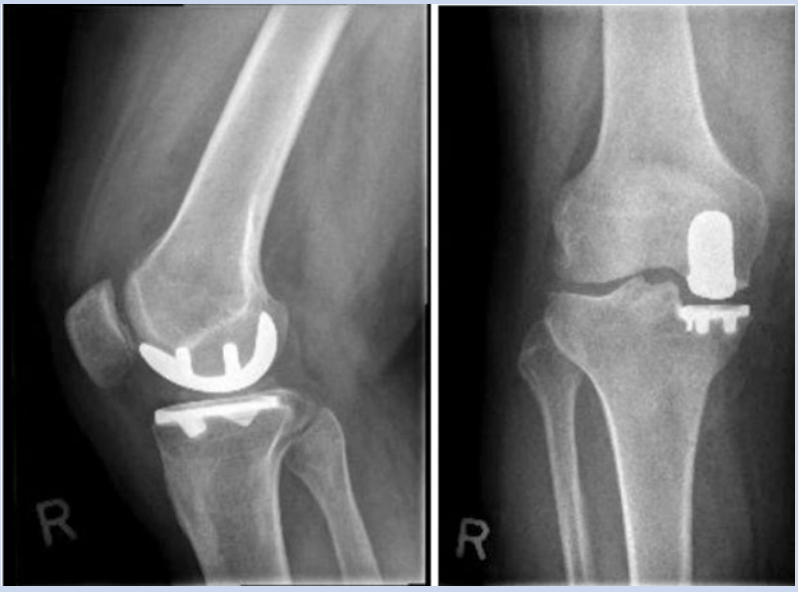
Describe the method of lateral ligament complex release and balancing the knee for valgus knees

I would perform sequential release of lateral structures (more on the femoral side). This is called inside-out technique (Ranawat's). Release is to be done in flexion, so popliteal artery falls posteriorly.

Lateral release should begin with the removal of the lateral osteophytes off the femur and tibia, meniscus and release of the lateral capsule off the tibia → If the knee is tight laterally in extension, the iliotibial band can be released either subperiosteally off Gerdy's tubercle, or by Z-lengthening or "pie-crusting" → The next

structure to be released is popliteus tendon, which is released sub-periosteally off the lateral condyle of the femur. In a large > 15° valgus deformity these releases will usually be necessary. → If the knee remains tight laterally the next step would be to release the LCL sub-periosteally off the lateral condyle of the femur. At this stage a constrained knee prosthesis should be considered, and one should be aware of the possibility of stretching the peroneal nerve when correcting large valgus deformities. If the gap remains unbalanced after releasing the lateral structures, MCL advancement can be considered. The epicondyle origin of MCL is advanced proximally and secured with sutures anchor or staples.

5.3 Viva 39



- **What can you see?**
- **Which patients would you offer UKA?**
- **What are the advantages of UKA?**
- **What are the outcomes of UKA?**

What can you see?
AP and lateral radiographs showing a right medial UKA. The components look well fixed and aligned. There are no signs of loosening or periprosthetic fractures. The lateral compartment and PFJ look relatively normal.

Which patients would you offer UKA?
The indications and prerequisites for UKA include:

- Unicompartmental diseases (non-inflammatory).
- Intact ligaments (ACL and PCL). ACL deficiency is a controversial contraindication with some surgeons considering it a viable option in the case of a medial fixed-bearing UKA.
- No signs of medial-lateral subluxation or instability.
- Correctable Varus deformity <10, valgus deformity <5.
- FFD <15.
- ROM >90.
- Classically reserved for elderly >60, low physical demands, and non-obese.

What are the advantages of UKA?

- Smaller incision.
- Less time/blood loss/dissection/morbidity.
- Preservation of normal knee kinematics by retaining the cruciate ligaments and remaining healthy joint surfaces.
- There is also evidence to suggest better range of movements.
- Early rehabilitation/return to work.
- Avoids patellar related complications (maltraking-fracture-dislocation).
- Maximizes the longevity of TKA.

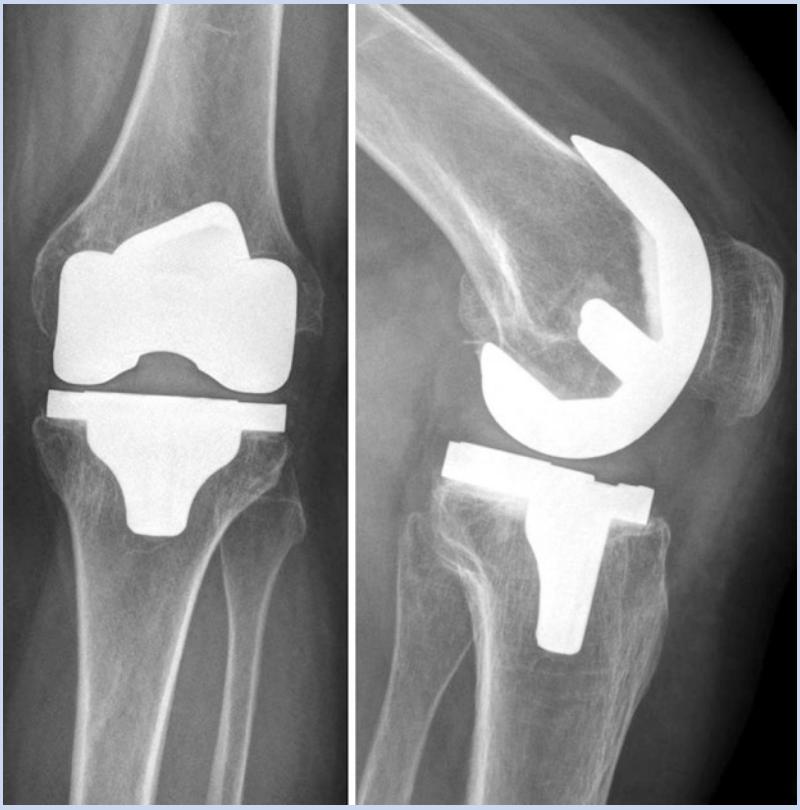
What are the outcomes of UKA?
NJR 16th annual report for UKA—cumulative revision rates at 15-year follow-up range from 31.7% (in <55 years age group) to 8.3% (in 75+ years age group).

Results of revision are less satisfactory than primary TKR (4 times higher—from New Zealand NJR).

Disease progression is the most common cause for revision.

5.4 Viva 40

A 62-year-old lady has been referred to your clinic with ongoing pain in her knee following a primary uncomplicated TKA.



- **Describe the radiograph.**
- **What are the most common causes of pain following TKA?**
- **How would you evaluate a patient with painful TKA?**

Describe the radiograph

These are anteroposterior and lateral radiographs showing a primary left TKA with cruciate retaining prosthesis. The components look well aligned and fixed. There are no obvious signs of loosening or periprosthetic fractures.

What are the most common causes of pain following TKA?
The predominant causes of pain following TKA include:

- Infection—may not be common but is the most important to exclude
- Patellofemoral problems (the most common)
- Component mal-position (overhang, mal-alignment, poor cementing)
- Loosening
- Instability
- Periprosthetic fractures
- Complex regional pain syndrome (CRPS)
- Dual pathology (hip arthritis)

How would you evaluate a patient with painful TKA?
I would start by taking a careful history from the patient, my history will be divided into 3 parts including preoperative, intraoperative, and postoperative

Preoperative: I would like to know:
- The indication for arthroplasty. Pain following TKR may be due to other underlying pathology, such as Paget's disease, pigmented villonodular synovitis, or RA, rather than problems with the knee replacement itself.
- Pain severity: prospective study by Lingard of 860 patients with TKR showed that those who had marked functional limitation and severe pain before operation were more likely to have a worse outcome after one and two years
- What were the patient's expectations: sometimes patients have TKA because they can't kneel while praying, so it's very important to explain to the patients that post TKA pts won't be able to kneel.

Intraoperative:
- I would like to identify which prosthesis was used and whether the patella was resurfaced or not. Were the components cemented or uncemented and was the prosthesis stemmed? Any navigation used should be noted as the sites for insertion of the pins can be a source of stress fracture and pain.

Postoperative:
- Immediate postoperative care, including wound healing, length of stay, and any reported complications. The rehabilitation and physical therapy course are also noted.
- The temporal course of the postoperative symptoms is noted. (Patients with either loosening or a hematogenous infection usually have an asymptomatic period with good knee function before becoming symptomatic. Conversely, patients with improper balancing and instability or with complex regional pain syndrome (CRPS) or dual pain typically do not report an asymptomatic interval.)

- Analyze the nature of the pain, including its character, location, radiation, and aggravating and relieving factors. Start-up pain is suggestive of loosening of tibial component, pain on weight bearing is usually due to loose components or instability, pain with full flexion can be associated with impingement or overstuffing of the flexion space, chronic pain in full extension is commonly caused by an overstuffed extension space, pain associated with stair climbing or descent can be attributed to dysfunction of the extensor mechanism, rest pain, and continuous postoperative pain that never improves are associated with infection or CRPS.

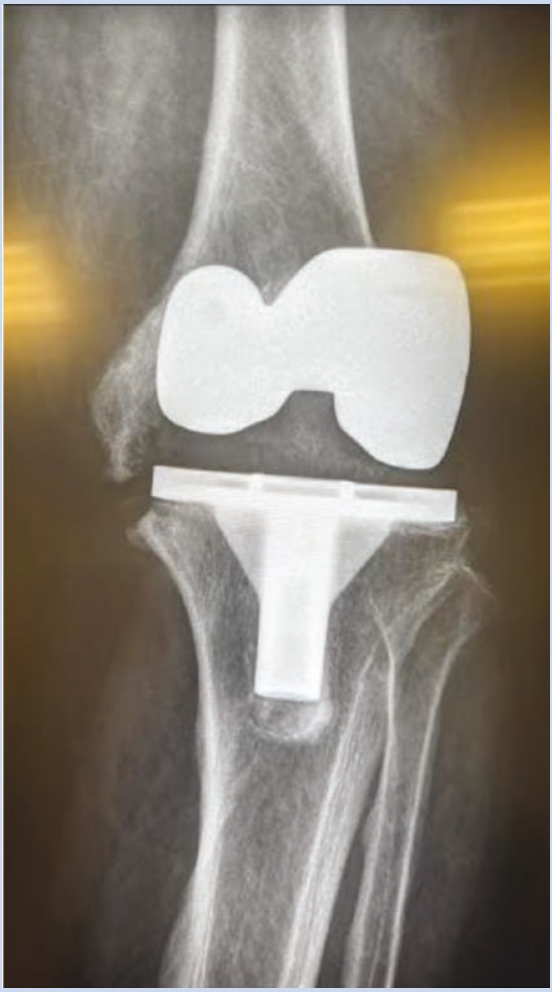
I also would like to know:

- Any other associated symptoms: associated symptoms such as instability or stiffness are more likely to have an intrinsic rather than an extrinsic problem
- Are there any co-morbidities? Systemic illnesses, such as inflammatory arthropathy and neuropathy, can affect joint function. Diabetes can affect the outcome of TKA.
- Any previous operations or procedures in the same knee. Any dental, colonoscopic, urologic procedures.
- Psychosocial history (mental health status): patients with preoperative depression and anxiety are less likely to be satisfied after knee arthroplasty.

Physical examination:

- **Gait**
  - Gait with thrust → instability.
  - Excessive external or internal rotation of the foot may be indicative of rotational malposition of the tibial component.
- **Inspection**
  - Describe the previous incision.
  - Erythema, warmth, swelling, or wound drainage → infection.
  - Swelling + recurrent hemarthrosis → PVNS—coagulation disorder.
  - Doughy dusky skin with limited painful ROM → CRPS.
- **Feel**
  - Tenderness over medial side → impingement of the medial collateral ligament or pes anserinus (overhanging of tibial component).
  - Tenderness over posterolateral aspect → impingement of the popliteus tendon by protruding prosthetic components—fabella.
  - Tenderness just medial to medial tuberosity → cutaneous neuroma.
- **Move**
  - Palpable and painful pinching of soft tissue in the PF joint in extension ➔ patellar clunk syndrome.
- Assess instability: collateral ligaments.
- Lumbar spine, hip, foot, and ankle, as well as a neurovascular status (common peroneal nerve), should be included in the analysis of painful TKA.

5.5 Viva 41



- **Are you aware of any classification system for the bone defect in TKA?**
- **What are the main goals of revision TKA?**
- **What are the implant choices for revision TKA?**
- **How would you optimize your exposure in revision knee surgery?**
- **What are the management options for extensive bone loss?**

Are you aware of any classification system for the bone defect in TKA?
The Anderson Orthopedic Research Institute (AORI) classification is the most practical and frequently used system which predominantly depends on the size of the bone defect originated from the tibia (T) and femur (F). In type 1 (T1 and F1) defects, there is a minor bone defect without compromising the stability of a revision component. The development of posterior condyles remains normal. In type 2 (T2 and F2) defects, metaphyseal bone damage and cancellous bone loss occurred in one femoral condyle/tibial plateau (type 2A: T2A and F2A) or both femoral condyle/tibial plateau (type 2B: T2B and F2B). The development of the posterior condyles and/or tibial component is reduced. In type 3 (T3 and F3) defects, there is significant cancellous metaphyseal bone loss compromising the ligamentous instability of a major portion of the condyle or plateau.

What are the main goals of revision TKA?
- Extraction of knee components with minimal bone and soft tissue destruction
- Restoration of cavitary and segmental defects
- Restoration of the joint line
- Balanced knee ligaments
- Achieve well-fixed and stable knee components

What are the implant choices for revision TKA?
The constraint ladder within knee implant design includes:

Unconstrained prosthesis (PCL retaining)
↓
Unconstrained prosthesis (PCL substituting)
↓
Constrained, non-hinged prosthesis
↓
Constrained, hinged prosthesis

How would you optimize your exposure in revision knee surgery?
- Using longer skin incision, this allows identification of the normal tissue planes and allows the scar tissue to be mobilized and subsequent wound closure.
- Extended approach through:
  - Extended medial para-patellar approach with rectus snip allows better visualization.
  - Tibia tubercle osteotomy in the very stiff knee with patell baja. It gives the best view but technically demanding, increases wound problems and stemmed tibial component is required to bypass the osteotomy site.
  - Patellar turndown gives good exposure but slower rehab.
- Synovectomy.
- PCL sacrifice can help in exposure.

What are the management options for extensive bone loss?

- The use of cement, either alone or combined with screws and mesh.
- The use of bone grafting with structural or morselized graft.
- The use of modular augmentation of the components with wedges or blocks of metal.
- The utilization of custom-made, tumor, or hinge implants.

5.6 Viva 42

A 19-year-old female is referred to your clinic by her GP due to recurrent right patella dislocation.

- **What is the function of the patella?**
- **Explain the anatomy & biomechanics of patellofemoral joint?**
- **What is the etiology of patellofemoral joint instability?**
- **How would you evaluate a patient with patellar instability?**
- **How would you investigate this patient?**
- **What are the treatment principles and options?**

What is the function of the patella?

- It increases the efficiency of Quads by providing long lever arm for Quads.
- It decreases the coefficient of friction "cartilage on cartilage".
- It centralizes the divergent forces of Quads.

Explain the anatomy and biomechanics of patellofemoral joint?

The patella is the largest sesamoid bone in the body, the posterior surface has 25% inferior non-articular part and 75% superior articular part; it composed of two facets, a medial and lateral facet, separated by a vertical ridge. The medial facet is convex, and the lateral facet is concave. The patella articulates with the trochlear groove of the anterior femur.

As the knee joint ranges from extension to flexion, the contact of the articular surface of the patella with the femur changes. In full extension, the patella has little to no contact with the trochlear groove and, therefore, this position is of higher risk for instability. From 10 to 30° of flexion, the patella engages the trochlear groove with the contact area being the inferior most portion of the medial and lateral facets. As the knee progresses through more flexion, the contact surface becomes more proximal on the patella.

The stability of the patella is dependent on both osseous anatomy and the integrity of longitudinal and transverse soft tissue stabilizers. The transverse stabilizers include the medial and lateral retinaculum, the vastus medialis and lateralis

muscles, the ilio-tibial band, and the medial patellofemoral ligament (MPFL). The longitudinal stabilizer is the extensor mechanism itself, which is comprised of the quadriceps tendon proximally and the patellar tendon distally

What is the etiology of patellofemoral joint instability?
The etiology of PFJI is multifactorial as it involves:

Acute traumatic instability
- Usually on noncontact twisting injury with the knee extended and foot externally rotated.
- Direct blow: less common.

Habitual
- *Painless*—pathology is proximal (ITB and vastus lateralis).

Chronic
- Bony factors (Patella alta—Trochlea dysplasia—á Q-angle—á TT-TG).
- Soft tissues (weak VMO—tight lateral—Generalized hypermobility).
- Malalignment (Increased femoral anteversion—External tibial torsion—Foot hyperpronation)

How would you evaluate a patient with patellar instability?
I would start by taking a detailed history followed by a physical examination.
In the history, I would like to address the following points:

- Age: Those patients who experience a first-time dislocation under the age of 20 are at high risk for recurrence.
- Gender: Acute dislocation occurs equally in males and females. However, females have a higher incidence of patellofemoral instability due to their increased Q-angle.
- Mechanism of injury.
- Number and circumstances of instability: (A prior instability episode is one of the best predictors of recurrent instability) risk of recurrent instability is 20% following first dislocation—50% following second dislocation.
- Physical activity level and sports participation.
- Systemic laxity such as Ehlers Danlos or family history of laxity can similarly put patients at risk for dislocation.

Physical examination:

- **Inspection:** I would check for *effusion, angular/rotational deformities, gait* (*foot and patella progression), and measure Q-angle* (it is measured using the anterior superior iliac spine (ASIS), center of the patella and center of the tibial tuberosity as anatomical landmarks. With normal values estimated between 8°–17° in males and 12°–20° in females).

- **Feel*:*** check for patellar height—medial-sided tenderness over medial femoral epicondyle (Bassett sign), J-sign (refers to the inverted 'J' track the patella takes from extension to early flexion (or vice versa) in a maltracking patella. The laterally subluxated patella in extension suddenly shifts medially as it engages the trochlear groove of the distal femur during flexion)—crepitus.
- **Move:** increased passive patellar translation in knee flexion 20° (lateral translation of medial border of patella to lateral edge of trochlear groove is considered "2" quadrants and is considered abnormal amount of translation)—lateral patellar apprehension -Lateral position: Ober test for iliotibial band tightness—hip motions &femoral anteversion (Prone position).
- **Beighton score**

How would you investigate this patient?
I would perform the following imaging:

- **Radiographs:**
  - Full leg length: to evaluate overall lower extremity alignment and Q-angle.
  - A/P: to rule out fracture or loose body from medial patellar facet (most common)-lateral femoral condyle.
  - Lateral views with 30 degrees flexion of the knee useful in detecting osseous morphologic features associated with patellar maltracking such as patella alta and useful to assess trochlear dysplasia by the presence of so-called crossing sign.
  - Merchant view of the patella: the knee is flexed 45 over the end of the table and the x-ray beam is inclined 30 downward. This view is used to assess for patellar tilt (lateral patellofemoral angle), patellar subluxation (congruence angle), and trochlear dysplasia (sulcus angle).
- **CT:**
  - Tibial tubercle–trochlear groove (TT-TG) distance is measured by (A) first drawing a line through the trochlea perpendicular to the line connecting the posterior condyles. These lines are superimposed onto an image through the tibial tubercle (B), and the TT-TG distance is measured as that between the above-described line and the tibial tubercle (distance AB). A distance between the tibial tuberosity and the trochlear groove exceeding 20 mm is nearly always associated with patellar instability.
  - To assess for rotational profile and femoral anteversion.
- **MRI:**
  - Useful in showing MPFL disruption and trochlear dysplasia.

What are the treatment principles and options?
The goal of patellar instability treatment is to achieve a stable, functional, and pain-free knee and ultimately to halt or slow the development of osteoarthritis.

It is divided into nonoperative and operative management.

Nonoperative treatment:

- Indicated in acute first-time dislocators without osteochondral fracture or loose bodies.
- NSAIDs.
- Immobilization (3–6 weeks).
- Progressive physiotherapy (closed chain exercises, and vastus medialis obliquus Strengthening).

Operative treatment options:

- **Arthroscopic debridement (removal of loose body) vs fixation + MPFL reconstruction** which is indicated in the presence of displaced osteochondral fractures or loose bodies consider.
- **MPFL reconstruction** which is best indicated in isolation in the setting of recurrent instability with minimal underlying osseous malalignment (normal TT-TG, minimal trochlear dysplasia). Gracilis or semitendinosus commonly used.
- **Tibial tubercle transfer:**
  - Medialization of the tubercle restores a normal TT-TG distance while anteriorization of the tubercle unloads patellofemoral contact forces.
  - Elmslie–Trillat procedure involves open lateral retinacular release, medial displacement of the tibial tuberosity hinged distally with periosteum, and medial capsular reefing, this is usually indicated in recurrent instability with an excessive tibial tuberosity-trochlear groove distance.
  - Fulkerson osteotomy, involves anteromedialization transfer of tibial tubercle by using more oblique cut, leading to unload the lateral and distal articular surface, and can decrease overall patellofemoral contract stress by shifting the contact area proximally and medially, it is indicated in patients with grade-3 or 4 chondromalacia of the lateral patellofemoral join.
- **Trochleoplasty** This procedure involves removal of cancellous bone beneath the trochlea followed by repositioning of the cortical part of trochlea and fixation of the articular surface. However, this technique is best reserved for patients with severe dysplasia who have previously undergone failed attempts to provide patellofemoral stability with other techniques in the absence of osteoarthritic changes.
- **Derotation osteotomy** indicated in rotational pathology.

Further Reading

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Drapeau-Zgoralski V, Swift B, Caines A, Kerrigan A, Carsen S, Pickell M. Lateral patellar instability. J Bone Joint Surg. 2023;105(5):397–409. https://doi.org/10.2106/JBJS.22.00756.

UK National Joint Registry (NJR). www.njrcentre.org.uk.

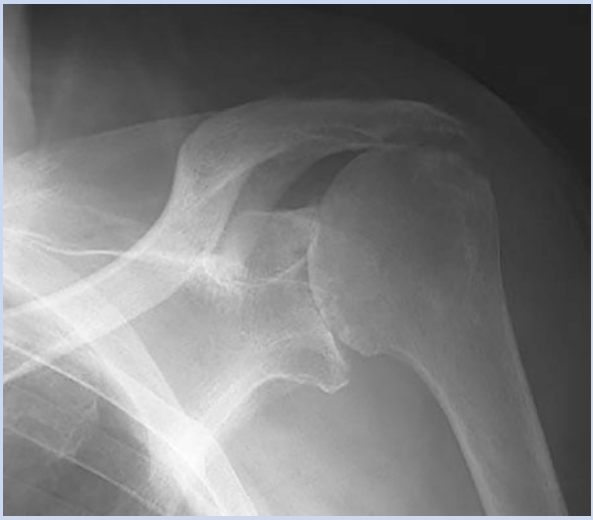
Shoulder and Elbow

6

Aiman Mudawi and Mazhar Fuad

6.1 Viva 43

This is a 74-year-old gentleman who has been referred by his GP with shoulder pain and reduced function.



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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_6

- **What can you see?**
- **What would you expect to find when you examine this patient?**
- **After non-operative treatments have been thoroughly explored without success, what surgical intervention would you recommend for this patient?**
- **How does a reverse shoulder prosthesis work biomechanically?**
- **What potential complications can arise following reverse shoulder arthroplasty?**

What can you see?
This is an anteroposterior X-ray of the left shoulder, which reveals several notable findings including proximal migration of the humeral head, along with joint space loss, subchondral sclerosis, and an absence of osteophytes. These observed features are indicative of rotator cuff arthropathy.

It is shoulder arthritis in the setting of rotator cuff dysfunction.

What would you expect to find when you examine this patient?
I would expect several clinical findings when examining a patient with rotator cuff arthropathy:

1. Supraspinatus/Infraspinatus Atrophy.
2. Subcutaneous effusion from loss of fluid from capsule.
3. Limitations in active and passive range of motions. Pseudoparalysis: which manifests as the inability to effectively abduct the shoulder.

After non-operative treatments have been thoroughly explored without success, what surgical intervention would you recommend for this patient?
Considering the compromised state of his rotator cuff, my recommendation would be to consider a reverse total shoulder replacement. However, before proceeding, I would conduct an assessment to ensure the adequate function of the deltoid muscle.

How does a reverse shoulder prosthesis work biomechanically?
It is a semi-constrained fixed fulcrum device. It uses convex glenoid hemispheric ball and a concave humerus articulating cup. It shifts center of rotation medially and inferiorly resulting in:

- Increased deltoid tension, effectively extending the lever arm of the deltoid muscle.
- Improved range of motion (ROM) for the shoulder joint.
- Decreased shear forces on the glenoid component and surrounding structures.

What potential complications can arise following reverse shoulder arthroplasty?

- Scapular notching due to inferior impingement of humeral cup on the scapular neck which occurs due to superiorly placed glenoid component or medialization of the center of rotation.
- Limited external rotation.
- Decreased deltoid efficiency due to inferior shift of COR which alters pull of deltoid.
- Dislocation, typically occurs with hyperextension and external rotation and it is anterior.
- Glenoid loosening.

6.2 Viva 44

GP's letter: A 47-year-old female with a past medical history of diabetes mellitus (insulin-controlled) and hypothyroidism presents to the office complaining of 3 months of shoulder pain and progressively worsening stiffness.

- **What is frozen shoulder?**
- **What are the classical stages described in the "natural history" of frozen shoulder?**
- **Discuss the various treatment options available for frozen shoulder.**
- **Are you aware of any relevant literature in this area?**

What is frozen shoulder?

It is a condition that is characterized by gradually resolving pain and global limitation of glenohumeral motion with no specific underlying cause and normal X-ray.

It is a disease of the joint capsule, which is thickened and shows increased vascularity. The picture is strikingly similar to Dupuytren's disease, with proliferation of fibroblasts, transformation to myofibroblasts, and increased collagen deposition. However, the clinical picture in both these conditions is different, with Dupuytren's being a progressive and pain-free deformity of the fingers and frozen shoulder being painful and self-limiting condition.

What are the classical stages described in the "natural history" of frozen shoulder?

Phase 1—Painful (Freezing) Phase: During this phase, patients experience a gradual onset of severe, diffuse pain that is unrelated to physical activity. Pain tends to worsen at rest, often disturbing sleep. This phase typically lasts from 6 weeks to 9 months.

Phase 2—The Frozen Phase: In this stage, the intense pain transforms into end-of-range pain. There is a notable decrease in the range of motion, which can significantly impact daily activities. Phase 2 usually spans from 4 to 12 months.

Phase 3—Thawing Phase: During this phase, there is a gradual return of shoulder motion over 12–42 months. Complete recovery may not always be achieved.

Discuss the various treatment options available for frozen shoulder.

Non-operative treatment is generally effective if initiated in the first 4–6 months, it involves:

1. NSAIDs.
2. Intra-articular injection.
3. Physical therapy: program of gentle, pain-free stretching and moist heat. should be supervised and last for 3–6 months.
4. Some advocate hydrodilatation: done by interventional radiologist, doesn't require GA, the capsule is injected with a mixture of local anesthetic, radio-opaque dye and saline to cause stretching and controlled tearing of the capsule, then to be followed by aggressive PT.

Operative treatment is generally reserved for cases that are unresponsive to non-operative:

1. Manipulative therapy to release adhesions. A safe sequence for shoulder manipulation—Flexion, extension, abduction and adduction, external and internal rotation (FEAR).
2. Arthroscopic surgical release treatment. It involves release of the anterior capsule, the rotator interval, middle glenohumeral ligament, and coracohumeral ligament. It is usually sufficient, although the posterior capsule can also be released if required.

Are you aware of any relevant literature in this area?

The UK FROST study, multi-center, pragmatic, three-arm, superiority RCT

It involved three treatment modalities:

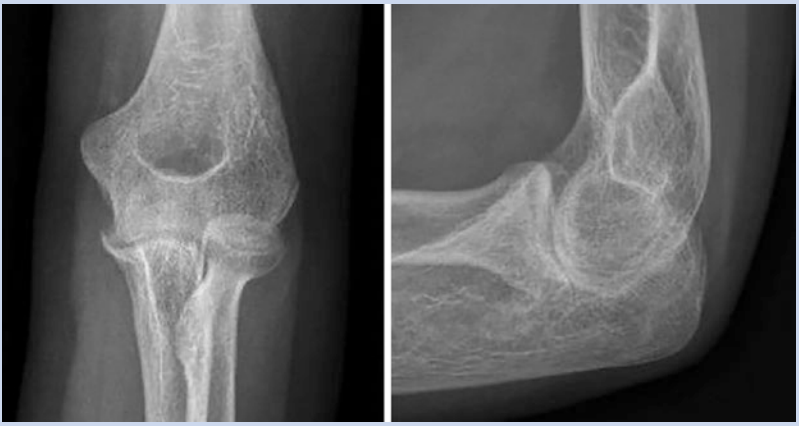
1. Early structural PT.
2. MUA.
3. Arthroscopic release.

Results:

1. None of the 3 interventions were clinically superior.
2. MUA was the most cost-effective.
3. Arthroscopic capsular release carried higher risks.

6.3 Viva 45

A 60-year-old lady with limited daily activities complains of chronic elbow pain.



- **Describe this radiograph.**
- **How can you distinguish between inflammatory and degenerative elbow arthritis radiographically?**
- **Are you aware of any classification systems used for this condition?**
- **How would you manage the condition?**
- **How would you prepare her for surgery?**
- **What are the types of total elbow arthroplasty?**
- **How much weight can you lift after total elbow arthroplasty?**
- **When will you consider interposition arthroplasty?**

Describe this radiograph

These are anteroposterior and lateral elbow radiographic views showing significant joint space narrowing and erosion of the articular surface. Periarticular osteopenia is also evident, involving both the radio-capitellar and ulnohumeral articular surfaces. No osteophytes or sclerosis are observed. These findings are indicative of inflammatory arthritis affecting the elbow.

How can you distinguish between inflammatory and degenerative elbow arthritis radiographically?

In radiographs of inflammatory arthritis, you typically observe uniform joint space narrowing, absence of sclerosis, and no osteophytes. There's often periarticular osteopenia. In contrast, osteoarthritis is characterized by asymmetric joint involvement, subchondral sclerosis, and the presence of osteophytes.

Are you aware of any classification systems used for this condition?
Mayo Classification of Rheumatoid Elbow

1. Osteopenia, subchondral cysts, and synovitis without marked joint line space narrowing.
2. Joint line space narrowing with preservation of the overall bony architecture.
3. Moderate bone loss affecting 1 (IIIA) or both (IIIB) humeral columns.
4. Complete disintegration of the elbow joint with dysfunctional instability (mutilans rheumatoid arthritis).
5. Ankylosis secondary to juvenile rheumatoid arthritis.

How would you manage the condition?

For this patient with limited household activity and chronic elbow pain related to rheumatoid arthritis, the management will involve a multidisciplinary approach. I would collaborate with a rheumatologist and involve an occupational therapist for assessment.

I would initiate a discussion with the patient to understand her current symptoms, their progression, past treatments, medication history (including disease-modifying or biological agents), functional status, and expectations. A thorough clinical assessment of the affected joint would include evaluating range of motion, joint stability, adjacent joints, tendons and assessing for potential nerve damage, especially involving the radial and posterior interosseous nerves. Cervical spine examination would also be considered.

The initial treatment approach would consist of non-operative measures, which may involve the use of a brace and occupational therapy with a focus on improving range of motion and muscle group balancing, as well as strength-building exercises.

If non-operative measures are ineffective or not well-tolerated, surgical options would be considered. This may include a synovectomy to address inflammation. In cases where radiocapitellar arthritis is clearly identified as a source of symptoms, excising the radial head may be considered.

Total elbow arthroplasty is typically reserved for patients with severe joint destruction or those who have failed previous interventions like synovectomy and debridement. In such cases, a linked or semi-constrained total elbow arthroplasty may be offered.

How would you prepare her for surgery?

Preparation for surgery in a rheumatoid patient involves several critical steps:

1. Assessment of Anesthesia Suitability: I would confirm the patient's fitness for general anesthesia.
2. Cervical Spine X-ray: I would perform a cervical spine X-ray to evaluate for any cervical spine instability or other relevant issues, given the potential involvement of the cervical spine in rheumatoid arthritis.

3. Anesthesia and Rheumatology Review: I would conduct a thorough preoperative review by an anesthesiologist and a rheumatologist to ensure a comprehensive understanding of the patient's health and the best approach for surgery.
4. Blood Investigations: I would perform blood investigations to assess the disease activity.
5. Management of Medications: If the patient is on biologics, it is generally advisable to discontinue these medications before surgery. Surgery should be scheduled at the end of the dosing cycle. Medications can typically be resumed at least 14 days post-surgery, assuming there are no complications such as wound healing problems, surgical site infections, or systemic infections.
6. DMARDs (Disease-Modifying Antirheumatic Drugs): For patients on DMARDs like methotrexate, sulfasalazine, hydroxychloroquine (HCQ), or leflunomide, it is generally recommended to continue these medications throughout the surgical period. However, individual cases may require adjustments based on the patient's specific health condition and the advice of the rheumatologist.

What are the types of total elbow arthroplasty?

Constrained
- High level of failure due to bone prosthesis loosening.

Semi constrained
- Sloppy hinge.
- Allows 5+ degrees of varus/valgus movement.
- Reduces stress on bone implant interface.

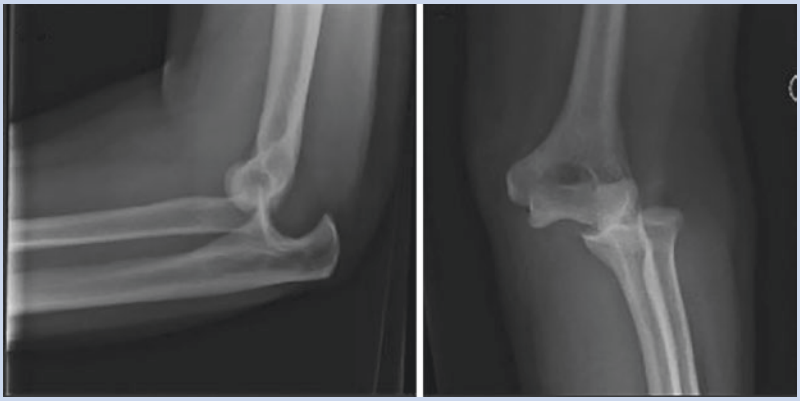
Unconstrained
- Must have intact collateral ligaments.
- Needs good bone stock.
- Decreased stress on bone implant interface.
- Decreased PE wear.
- Instability is the main concern.

How much weight can you lift after total elbow arthroplasty?
No more than 5 pounds.

When will you consider interposition arthroplasty?
This procedure involves using some sort of soft-tissue material or synthetic equivalent interposed at the joint surface after removal of any remaining articular cartilage and recontouring of the articulating surface. Interposition arthroplasty offers a solution for rheumatoid elbows not responding to synovectomy in patients considered not ideal candidates for elbow arthroplasty due to concerns related to implant failure (young age, active patients).

6.4 Viva 46

A 25-year-old male patient with history of a fall from 2m height.



- **What do these radiographs show?**
- **What are the stabilizers of elbow joint?**
- **How are these injuries classified? What is the pathophysiology?**
- **How would you manage this patient?**

What do these radiographs show?
These are AP and lateral radiographs of a skeletally mature individual of left elbow showing a posterolateral dislocation without obvious associated fractures.

What are the stabilizers of elbow joint?
Elbow stability is maintained by both static and dynamic stabilizers.

Static (primary) stabilizers
1. Ulno-humeral joint
   (a) Primary stabilizer for varus stress.
   (b) Coronoid is essential to resist post dislocation.
   (c) Loss of 50% or more of coronoid height results in elbow instability.
   (d) Olecranon provides stability in extension, at 25 degrees of flexion, the olecranon is unlocked from fossa.
2. Anterior oblique ligament of MCL
   (a) MCL has 3-components: anterior—posterior—transverse bands.
   (b) Anterior band originates from medial epicondyle and inserts in the anteromedial facet "sublime tubercle" – Resists valgus stress—Tight throughout ROM (isometric).

(c) Posterior band of MCL resists valgus in flexion, it forms the floor of cubital fossa.
(d) Transverse ligament connects coronoid to the olecranon, its role in elbow stability is unclear.

3. Lateral ulnar collateral ligament
   (a) Lateral collateral lig is composed of lateral ulnar collateral ligament (LUCL)—radial collateral ligament (RCL)—annular lig.
   (b) LUCL is the primary stabilizer to varus stress, originates from lateral epicondyle and inserts on crista supinator.
   (c) RCL inserts on annular ligament.

Static (secondary) stabilizers
- Radial head (radio-capitellar joint): functions as a secondary stabilizer to valgus stress particularly in MCL-deficient elbow.
- Capsule: provides stability in extension.

Dynamic stabilizers
- Muscles crossing the joint: anconeus—brachialis—triceps—biceps, they provide compressive stability.

How are these injuries classified? What is the pathophysiology?
Anatomic classification
- Based on the position of olecranon relative to humerus, posterolateral is the most common type accounting for 80%.

Simple vs Complex:
- Simple: no associated fractures.
- Complex: associated with fractures.
  - Terrible triad injury: elbow dislocation (posterolateral)—LUCL injury—radial head fracture—coronoid tip fracture.
  - Varus posteromedial rotatory instability: LCL injury—coronoid fracture (anteromedial facet or comminuted)—radial head fracture is unlikely.
  - Pathoanatomic cascade described by O'Driscoll: progression of injury is from lateral to medial known as circle of Hori. LCL fails first (primary lesion) by avulsion of the lateral epicondylar origin, midsubstance LCL tears are less common but do occur → the radial head dislocates or fractures → Then anterior capsule fails → MCL fails last depending on degree of injury.

How would you manage this patient?
Having taken a brief history, completed my clinical assessment and documented the neurovascular status of the patient, I would plan to reduce the elbow dislocation. I would usually perform this under sedation in the emergency department. I would reduce the elbow by flexing it to 30° to unlock the olecranon from the olecranon fossa, in supination to shift coronoid under trochlea, applying longitudinal traction

to improve coronal displacement and gently levering or pushing the olecranon over the distal humerus. Occasionally this cannot be achieved under sedation, and in this situation, I would arrange to perform this in the operating theatre under general anesthesia. Having reduced the elbow, I would take it through a gentle range of movement to check if a congruent reduction has been obtained and can be maintained. I would then place the patient in an above elbow backslab. I would reassess the neurovascular status, I would confirm reduction with further X-rays in plaster and would review the patient in the fracture clinic at 1 week. At that time, I would remove the backslab before repeating my examination and taking further X-rays. I would plan to mobilize the patient actively from this time.

Further Reading

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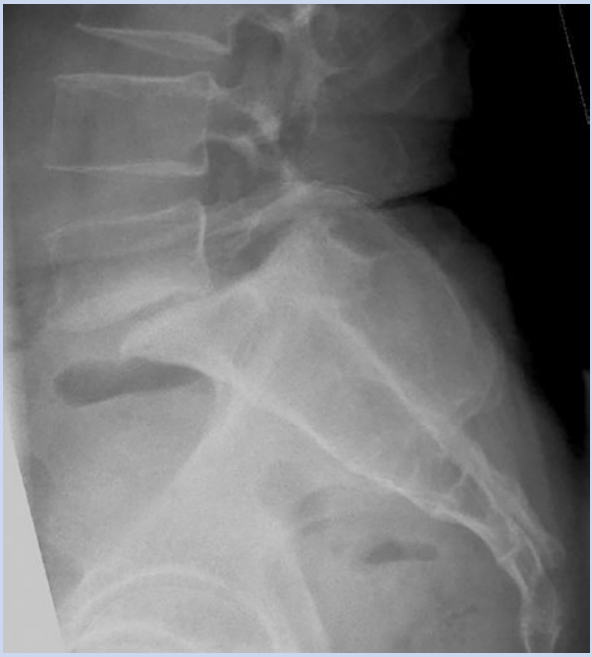
Spine

7

Saeed Ahmed Qaimkhani and Nasser Khan

7.1 Viva 47



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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_7

- **Describe the above radiograph.**
- **How do you classify spondylolisthesis?**
- **How do you grade spondylolisthesis?**
- **How do you treat patients with spondylolisthesis?**
- **What kind of surgery is being done for spondylolisthesis?**

Describe the above image

This is a lateral view radiograph of lower lumbar and sacrococcygeal region showing anterolisthesis/spondylolisthesis of L5 vertebral body over S1.

How do you classify spondylolisthesis?

Commonly used classification is Wiltse Classification which is basically etiological classification and is as follows:

1. Congenital
2. Isthmic
3. Degenerative
4. Traumatic
5. Pathological
6. Iatrogenic

How do you grade spondylolisthesis?

It depends on the degree of slippage of one vertebra over the other as follows (Fig. 7.1):

Grade 0: 0% slippage
Grade 1: 0–25%
Grade 2: 26–50%
Grade 3: 51–75%
Grade 4: 76–100%
Grade 5: more than 100%/spondyloptosis

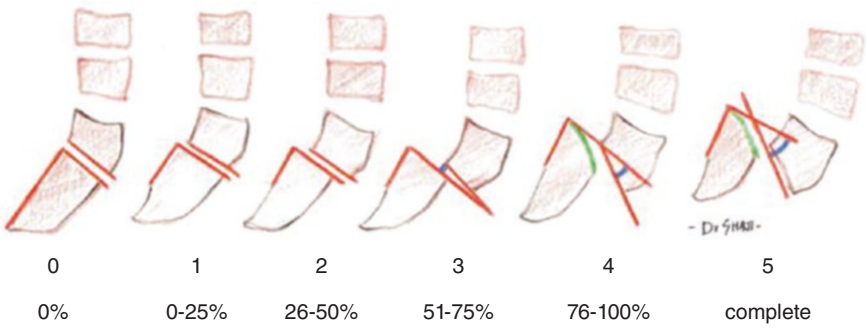


Fig. 7.1 Meyerding classification

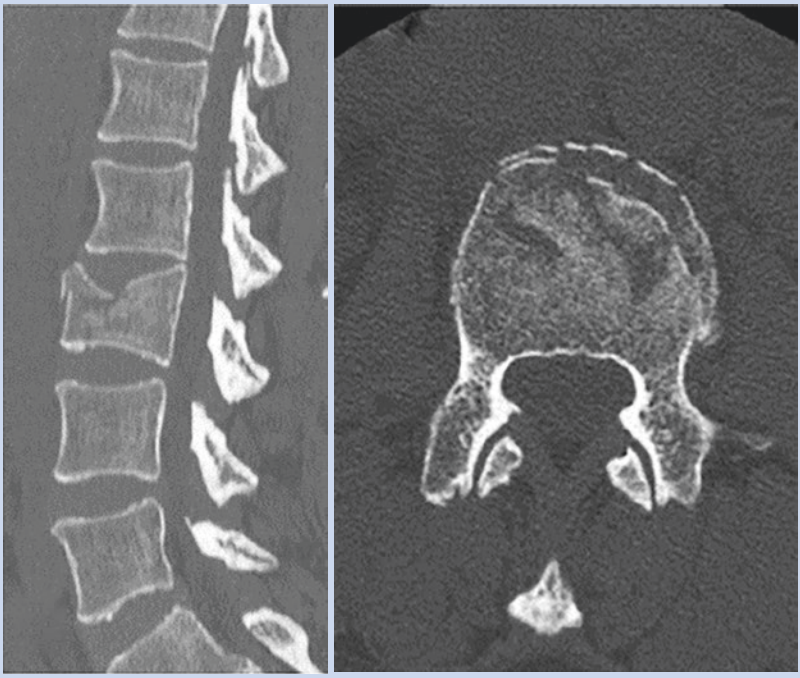
How do you treat patients with spondylolisthesis?
It all depends on the severity of the spondylolisthesis and on the clinical findings.

For example, the Grade 0 and 1 most of the time are incidental findings and are treated non-operatively. On the other hand, spondylolisthesis of higher grades may need surgical intervention.

What kind of surgery is being done for spondylolisthesis?
The surgical management of spondylolisthesis depends on various factors, including the severity of the condition, the grade of the slip, the patient's comorbidities, and the surgeon's preference. Among the surgical approaches, the most commonly utilized method is posterior decompression combined with instrumented fusion.

7.2 Viva 48

A 40-year-old male fell from a height of 8m and presented to Accident and Emergency complaining of low back pain. He is neurologically intact.



- **Can you describe the images?**
- **How would you manage this patient?**
- **Do you know any classification system of thoracolumbar spine fractures?**

Can you describe the images?
This CT scan includes sagittal (A) and axial (B) cuts of the lower lumbar vertebrae. Based on the imaging, there is a compression fracture of L1 vertebral body. The axial cut through the fractured vertebra (L1) does not reveal any involvement of the posterior wall.

How would you manage this patient?
I would follow the ATLS protocol for managing trauma patients, which includes assessing and securing the patient's airways, cervical spine, breathing, circulation, and neurologic status. Once I've cleared other potential injuries and ensured the patient's stability, I would initiate treatment with a thoracolumbar orthosis (TLSO brace).

Do you know any classification of thoracolumbar spine fractures?
There are many classifications of thoracolumbar spine fractures but the most commonly used one is TLICS Classification which consists of three parameters as follows:

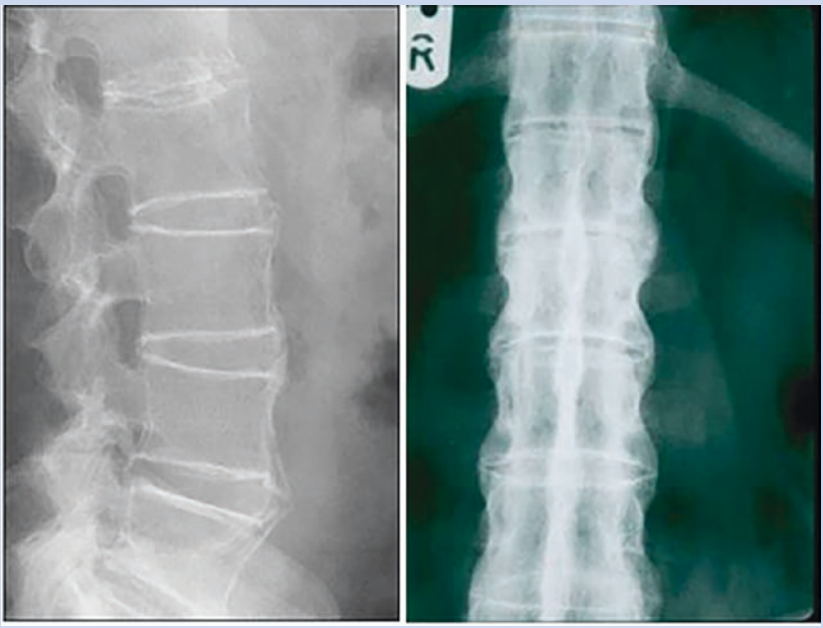
1. Injury morphology
2. Integrity of the posterior ligamentous complex (PLC)
3. Neurologic status.

A parameter can be scored 0–4 points, and the total score is the sum of these parameters with a maximum of 10 points.

The total score predicts the need for surgery. A total of more than 4 points indicates surgical treatment.

The integrity of the posterior ligamentous complex is a crucial factor in the TLICS. While PLC injury can sometimes be determined using a CT scan, an MRI may be necessary in some cases for a more detailed assessment.

7.3 Viva 49



- **Describe the radiographs.**
- **What is ankylosing spondylitis?**
- **How does the patient present clinically?**
- **What are the extraskeletal manifestations of ankylosing spondylitis?**
- **What are the diagnostic criteria for ankylosing spondylitis?**
- **What are the radiological findings of ankylosing spondylitis?**
- **What are the treatment options?**
- **Any surgical intervention?**

Describe the radiographs

Radiographs of the lumbar spine; AP view shows features of bamboo spine as seen in ankylosing spondylitis, marginal syndesmophites are seen bilaterally. Lateral view shows fusion of the entire lumbar spine anteriorly due to ossification of ALL.

What is ankylosing spondylitis?

It is a systemic chronic autoimmune seronegative (inflammatory) spondyloarthropathy of unknown etiology that primarily affects axial spine, characterized by positive HLA B27 in 90% of cases.

How does the patient present clinically?
Insidious onset of low back pain, neck, and upper thoracic pain (occurs later in life), morning stiffness, shortness of breath caused by costovertebral joint involvement, leading to reduced chest expansion and ocular pain (anterior uveitis).

Physical examination:

- Early: Hang-dog posture (rounding of the shoulders and slight dorsal kyphosis).
- Later: stoop develops with limitation of forward vision with thoracolumbar kyphosis and flexion deformities of the hip (question mark posture). Chin-brow to vertical angle: used to measure chin-on-chest deformity, useful for preoperative planning for osteotomy. Occiput to wall distance. Unable to stand with his back flush against wall. Fixed flexion deformity of hip joints. Reduced chest expansion. Measure at the level of the nipples from full expiration to full inspiration, it is reduced to 3 cm compared to a normal expansion of 7 cm and positive Schober's test for reduced lumbar spine movements.

What are the extraskeletal manifestations of ankylosing spondylitis?
Heart disease (carditis–aortic valve disease), pulmonary fibrosis, renal amyloidosis, uveitis, Klebsiella pneumoniae synovitis. HLA-B27 individuals are more susceptible to Klebsiella pneumoniae synovitis.

What are the diagnostic criteria for ankylosing spondylitis?
Bilateral sacroiliitis, HLA-B27 positive, ± uveitis.

What are the radiological findings of ankylosing spondylitis?
Usually, whole spine radiographs (standing full-length A/P and lateral) are needed. The radiographic findings typically progress through several stages:

Early stage:
1. Squaring of the vertebral body: This occurs due to flattening of the normal anterior concavity and ossification of the anterior longitudinal ligament (ALL).
2. Erosion and fuzziness of the SI joints: Early signs include erosion and a fuzzy appearance of the sacroiliac (SI) joints, especially on the iliac side of the joint.
3. Joint narrowing: Progressive narrowing of the SI joints.
4. Bony ankylosis: Ultimately, the disease may lead to bony ankylosis and obliteration of the SI joints.

Late stage:
1. Ankylosis of the facet joints.
2. Ossification of annulus fibrosis.
3. Marginal syndesmophyte formation (bamboo spine).

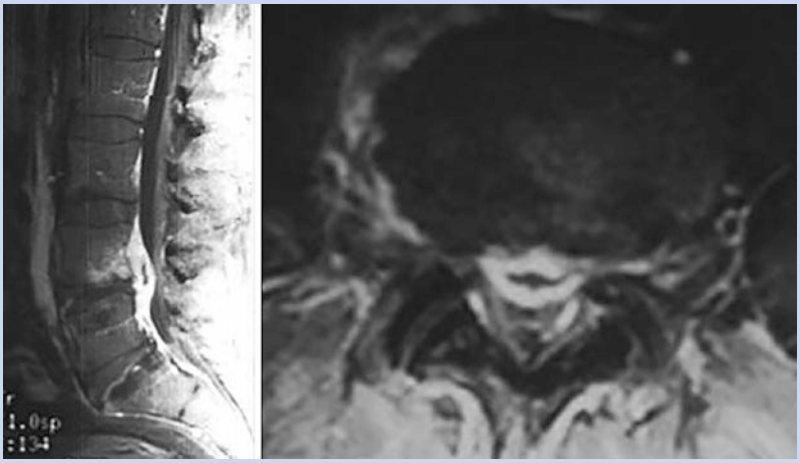
What are the treatment options?
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)—Disease-Modifying Antirheumatic Drugs (DMARDs)—Biologic Therapies (TNF-Alpha Blocking Agents)—Physical therapy.

Any surgical intervention?
It depends on the severity of the disease and involved joints; patients may need simple non-operative measures like splintage, physiotherapy, injections or in later stages may require Joint replacement surgeries or spinal decompression and fusion.

7.4 Viva 50

You received a call from ER physician about 55 years old male known case of diabetes with back pain radiating to both lower limbs. He had caudal epidural injection 1 week ago for his lumbar canal stenosis.
On Examination: Temperature is 38.5 °C, he has tenderness in lower lumbar area. There is generalized motor weakness 3/5 in all muscle groups in lower limbs bilaterally.
Sensations altered globally. PR normal.
Investigations: WBC 15, ESR 120, CRP 20 radiographs are normal.

- **How would you manage this patient?**
- **Here is the MRI. What is your diagnosis, and how would you proceed?**



- **How would you define cauda equina syndrome?**
- **How urgent should surgical decompression of cauda equina be done and why?**
- **What are the other causes of cauda equina syndrome?**

How would you manage this patient?
The most likely diagnosis is infection with epidural abscess which is causing neurological compromise secondary to cauda equina compression. This is serious emergency. I would send blood cultures, emergency MRI and make arrangements for urgent decompression and washout.

Here is the MRI. What is your diagnosis, and how would you proceed?
The MRI findings in both views; sagittal and axial suggest (in view of the history and physical exam) epidural abscess behind L4 and L5 vertebral bodies causing compression of the thecal sac (cauda equina).

The treatment of the epidural abscess with neurological deficits is surgical decompression which should be done on urgent basis without any delay.

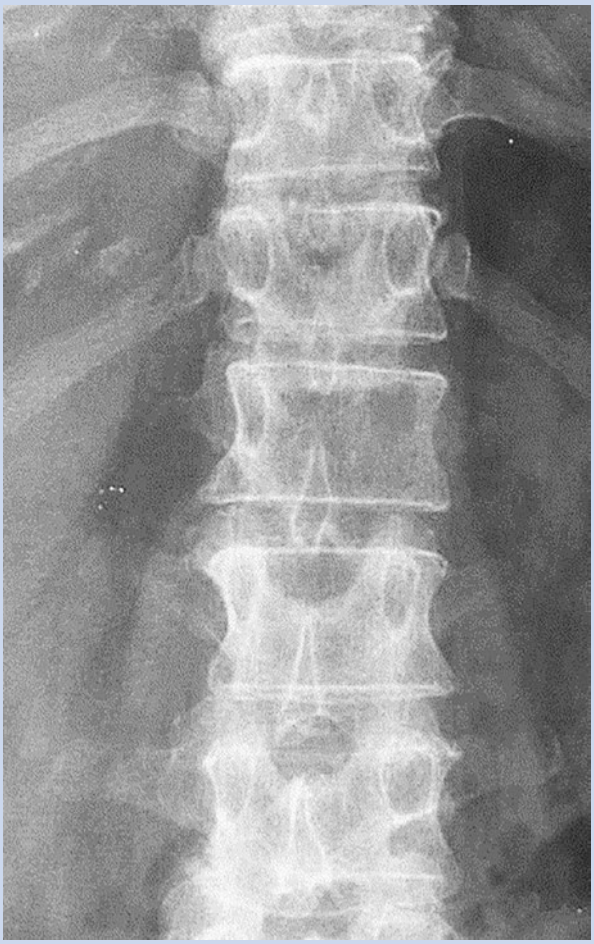
How would you define cauda equina syndrome?
It is a spectrum of symptoms including saddle anesthesia and/or bilateral lower limb numbness and or bilateral motor weakness and or bladder (urinary retention with overflow incontinence) and bowel symptoms. Residual bladder urinary volume of 200 ml combined with other symptoms and signs is pathognomonic of cauda equina compression and emergency MRI scan should be obtained.

How urgent should surgical decompression of cauda equina be done and why?
According to recent studies urgent decompression within 24 h has good prognosis. Once the sphincteric symptoms are established, then the prognosis is poor.

What are the other causes of cauda equina syndrome?
There are several causes including large disc herniation, metastatic compression, spinal canal stenosis secondary to advanced degenerative changes, postoperative epidural hematoma.

7.5 Viva 51

A 65-year-old lady presented with 3 months history of back pain.



- **Can you comment on the radiograph?**
- **What are the primary carcinomas that spread to the bones?**
- **What are the most common sites of metastasis?**
- **What is the pathogenesis of metastasis?**
- **What is the associated condition with bone metastasis which can lead to medical emergency?**

- **How do you treat hypercalcemia secondary to bony metastasis?**
- **Are you aware of any classification systems used for spine metastasis?**
- **What imaging modalities will you order to reach your diagnosis?**
- **How would you manage a patient with spine lesion?**
- **Would you use steroid in patients with metastatic spinal cord compression?**

Can you comment on the radiograph?
This is an AP view radiograph of thoracolumbar region showing absence of pedicle on the left side of L1 vertebral body. The sign is called Winking owl sign and it is pathognomonic for spine lesion mostly seen in spine metastasis.

What are the primary carcinomas that spread to the bones?
Carcinomas that commonly spread to bone include breast (almost one-third of breast cancer patients develop spine mets), lung, thyroid, renal, and prostate.

What are the most common sites of metastasis?
Lung is the most common site of metastasis followed by liver and bone. Common sites of bone metastatic lesions include spine followed by proximal femur and lastly humerus.

What is the pathogenesis of metastasis?
It involves four processes
- Intravasation: entry to blood vessels
- Avoidance of immune surveillance
- Localization of the target tissue
- Angiogenesis

Tumors typically spread via Baston's vertebral plexus (it is valveless veins that allow direct spread of the tumor to axial skeleton involving spine—shoulder girdle—skull and pelvis)

Effect of tumor on bone
- Lysis (thyroid–lung–renal): Through IL-1–IL-6–PTH-rP → activate osteoblasts → activate osteoclasts through RANK-L pathway.
- Sclerosis (breast–prostate): Produces Endothelin-1 → activate osteoblasts through ET receptors → inhibits Wnt gene → increases osteoblast activity causing sclerosis.

What is the associated condition with bone metastasis which can lead to medical emergency?
Metastatic hypercalcemia can lead to medical emergency. The symptoms include confusion, muscle weakness, polyuria & polydipsia, nausea/vomiting, and dehydration.

How do you treat hypercalcemia secondary to bony metastasis?
By proper hydration (volume expansion), loop diuretics and bisphosphonates.

Are you aware of any classification systems used for spine metastasis?
The Tokuhashi score is a prognostic tool specific to metastatic spinal disease. It assesses six elements: general condition, extraspinal bony metastasis, number of vertebral bodies with metastasis, visceral metastasis, primary tumor, and neurologic compromise. The score ranges from 0 to 15, with lower scores (0–8) indicating a shorter life expectancy (<6 months), moderate scores (9–12) suggesting a prognosis of more than 6 months, and higher scores (12–15) indicating a prognosis of over 1 year.

What imaging modalities will you order to reach your diagnosis?
1. Radiographs including AP and lateral views of involved area of spine (preferably whole spine). We have to look at the nature of lesions, e.g., purely lytic or mixed lytic/blastic lesions. As lung, thyroid, and renal are primarily lytic, whereas 60% of breast CA is blastic and 90% of prostate CA is blastic.
2. CT scan: helpful to identify metastatic lesions to the spine.
3. MRI: useful to show neurologic compromise of the spine.

How would you manage a patient with spine lesion?
We treat the patient using general considerations as NOMS framework: Neurologic, Oncologic, Mechanical instability, and Systemic illness.

- Neurologic: measure of epidural spinal cord compression (ESCC)—0–1 low grade, 2–3 high grade.
- Oncologic: responsiveness to radiation.
- Mechanical instability: spinal instability neoplastic score (SINS) SINS: 0–6 no surgical consultation required; 7–18 surgical consultation advisable.
- Systemic illness: formulation of prognosis from disease burden, medical comorbidities, functional status.

Nonoperative
- Mainly palliative care in patients where life expectancy of <6 months. Tokuhashi scoring system can be used to determine life expectancy.
- Radiation alone. It is indicated in patients who are not surgical candidate. No signs of neural compression, neurologic deficit, or instability.

Operative In the form of neurologic decompression, spinal stabilization, and postoperative radiation. Surgery is indicated in metastatic lesions to spine with neurologic deficits in patients with life expectancy of >6 months.

Preoperative embolization indicated in metastatic renal and thyroid CA to spine.

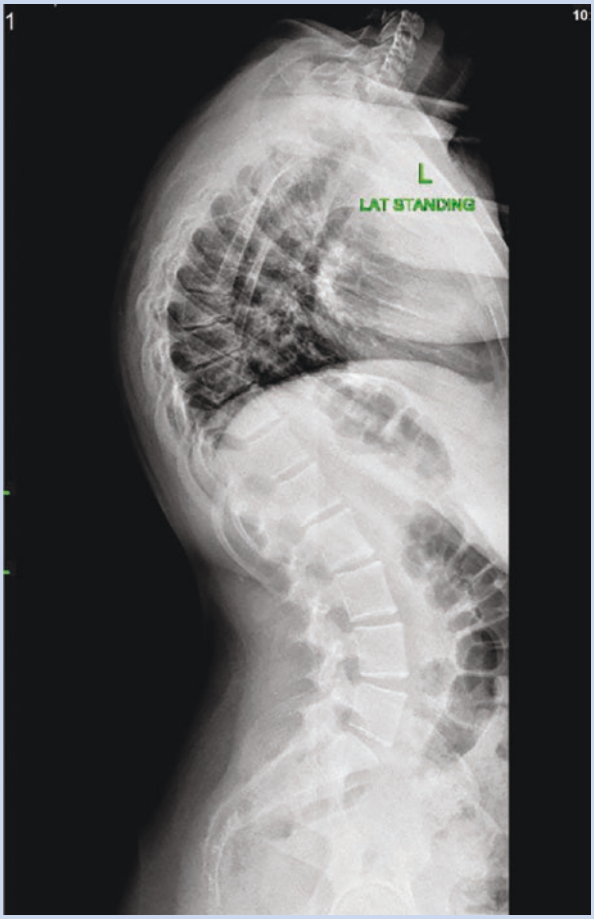
Would you use steroid in patients with metastatic spinal cord compression?
Steroids can help in relieving compression, but it is also associated with systemic toxicity.

Evidence: systematic review in 2017 showed metastatic spinal cord compression is one of the indications to give steroid, with high dose bolus then wean down.

- Recommend loading dose of 16 mg Dexamethasone as soon as possible after assessment, followed by short course of 16 mg daily.
- Dose should be reduced gradually over 5–7 days and stopped (cover with PPI and monitor blood glucose).

7.6 Viva 52

A 17-year-old girl presented with back pain.



- **Can you comment on the radiograph?**
- **Define scheuermann kyphosis.**
- **How many types of scheuermann kyphosis are there?**
- **How do you manage scheuermann kyphosis patients?**
- **What kind of surgery is commonly done for scheuermann kyphosis?**

Can you comment on the radiograph?
This is a standing lateral view radiograph of the entire spine, which reveals an increased kyphosis of the thoracic spine with anterior wedging primarily affecting vertebrae T8, T9, and T10. The sagittal balance appears to be well maintained.

Define scheuermann kyphosis
Scheuermann kyphosis is defined as a rigid thoracic hyperkyphosis of greater than 45 degrees caused by anterior wedging of >5 degrees across three consecutive vertebrae. It is differentiated from postural kyphosis by its rigid curve.

How many types of scheuermann kyphosis are there?
There are mainly two types of scheurmann kyphosis as follows:

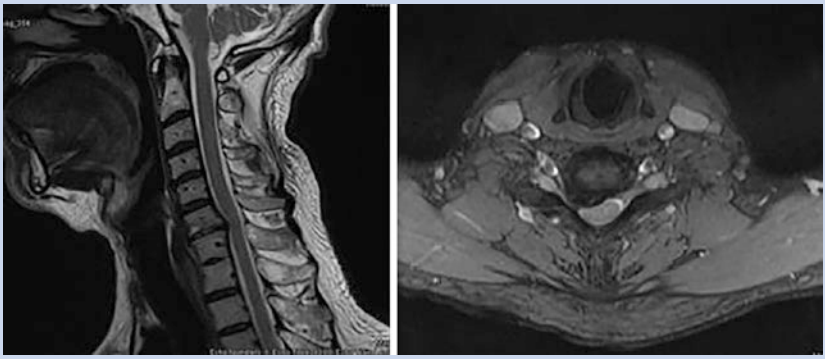
1. Thoracic scheuermann kyphosis (most common).
2. Thoracolumbar/lumbar scheuermann kyphosis. Less common, associated with increased back pain, more irregular endplates noted on radiographs, no vertebral wedging involved.

How do you manage patients with scheuermann kyphosis?
The approach to treating scheuermann kyphosis depends on the severity of the curve. For kyphosis measuring less than 60° and for asymptomatic individuals (or those with mild pain), observation alone may suffice. Surgical treatment is typically recommended for kyphosis exceeding 75°, particularly if it is rigid and occurs in skeletally mature patients. Surgical intervention is also considered in cases where there is a neurological deficit due to cord compression or in patients experiencing severe pain**.**

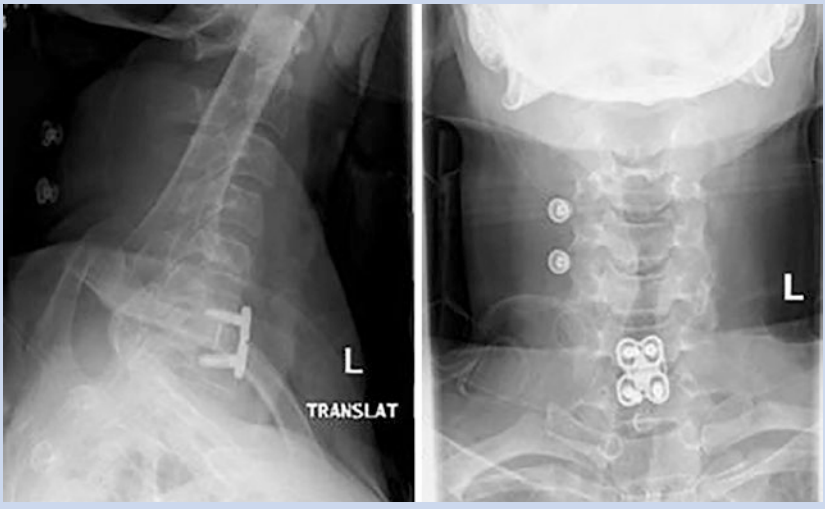
What kind of surgery is commonly done for scheuermann kyphosis?
The most common surgical procedure for treating scheuermann kyphosis involves posterior instrumentation, which often includes multiple osteotomies using pedicle screws and dual rod instrumentation.

7.7 Viva 53

A 43-year-old male presented with neck pain and right upper limb radiculopathy.



- **What can you see?**
- **Which nerve root is affected at this level, and what are the clinical manifestations?**
- **How do you examine the C7 nerve root?**
- **How would you manage this case?**
- **Can you describe the following radiographs?**



What can you see?
This is a T2-weighted MRI scan of the cervical spine including sagittal (left) and axial (right) sections, revealing degenerative disc disease at the C6/C7 level. There is a primarily posterolateral disc protrusion, which is impinging on the right-sided exiting nerve root.

Which nerve root is affected at this level, and what are the clinical manifestations?
At the C6/C7 level, the affected nerve root is C7. Clinical manifestations typically include numbness and decreased sensation in the C7 dermatome, primarily affecting the palmar aspect of the middle finger, along with weakness in elbow extension.

How do you examine the C7 nerve root?
To examine a specific nerve root, the affected dermatome, myotome, and tendon reflex should be examined. In this case the examination of C7 nerve root will be as follows**:**

1. Dermatome: Decreased sensation over the tip of the middle finger.
2. Myotome: The triceps muscle will be affected and is usually tested by resisting elbow extension.
3. Deep tendon reflex: Tested by triceps tendon reflex.

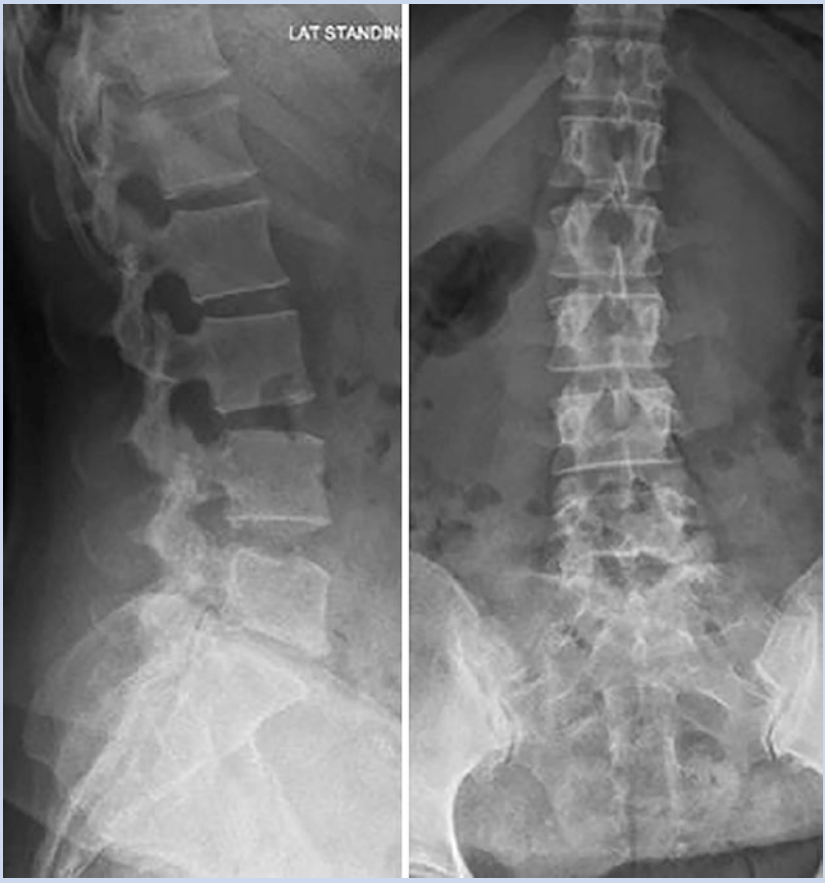
How would you manage this case?
Typically, treatment begins with conservative management, which includes pain control using NSAIDs and neuropathic medications for upper limb radiculopathy. Additionally, I would refer the patient for physiotherapy. In cases where conservative management is unsuccessful, I would have a discussion with the patient about the possibility of surgical treatment, such as anterior cervical decompression and fusion at the C6/C7 level.

Can you describe the following radiographs?
The radiograph on the left is called swimmer view, which is usually done to see the cervicothoracic junction. It shows anterior instrumentation at the level of C6/C7 with interbody cage plus plates and screws. The X-ray on the right shows AP view with well-aligned plate and screws.

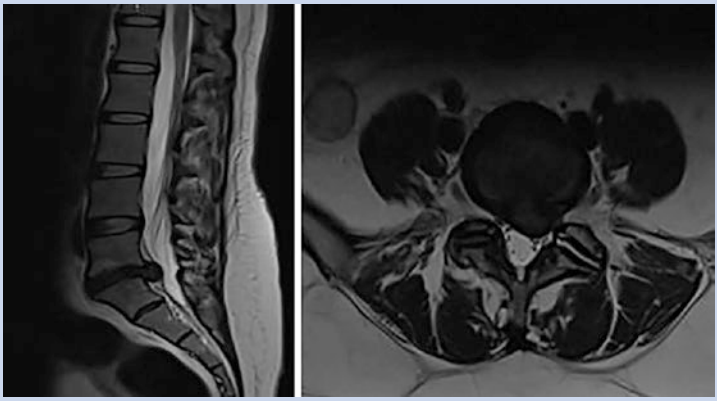
7.8 Viva 54

A 33-year-old female presents with low back pain and left lower limb radiculopathy. She reports that her leg pain is more bothersome than her back pain and is affecting her daily activities.

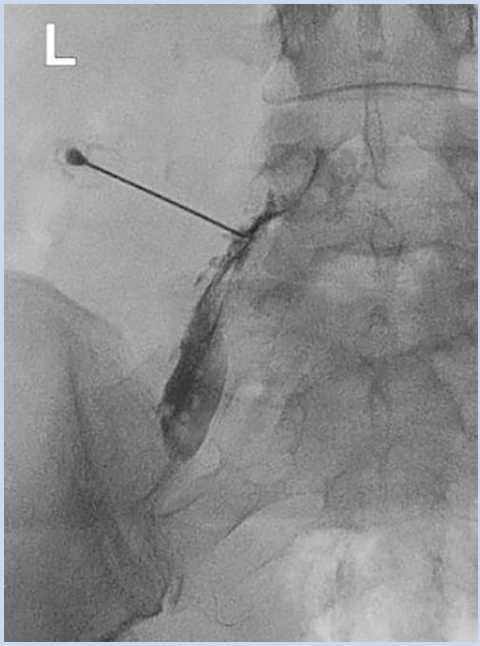
- **Can you describe the radiographs**



- **MRI scan of the patient was done. Could you describe the findings**



- **Which nerve root is affected at this level, and what are the clinical manifestations?**
- **How would you manage this patient?**
- **The patient appears to be hesitant about surgical intervention. Are there alternative treatment options available that we can discuss with her?**
- **Can you comment on the image below**



- **What surgical procedure would you recommend after exhausting all nonsurgical options?**

Can you describe the radiographs?
These are anteroposterior (A/P) and lateral radiographs of the lumbosacral spine. The lateral standing view reveals a reduction in lumbar lordosis and a decrease in the intervertebral disc space between L5 and S1. The A/P view shows mild scoliosis at the L5/S1 level.

MRI scan of the patient was done. Could you describe the findings?
The MRI scan includes T2-weighted images, including sagittal (on the left) and axial (on the right) views of the lumbosacral spine. In the sagittal view, there is evidence of degenerative disc disease at the L5/S1 level, along with a posterolateral disc protrusion causing impingement on both the exiting and traversing nerve roots, as observed in the axial cuts.

Which nerve root is affected at this level, and what are the clinical manifestations?
At this level, compression affects both the exiting and traversing nerve roots on the left side, resulting in the involvement of both the L5 and S1 nerve roots. The patient will experience radiculopathy in the left lower limb, leading to pain and altered sensation, primarily in the dorsum and plantar aspect of the foot. Weakness may also occur, affecting the extension of the big toe (L5) and plantar flexion of the foot (S1).

How would you manage this patient?
I would start with conservative treatment, including physical therapy and pain management. Surgical intervention would be considered if conservative measures failed to alleviate the patient's symptoms.

The patient appears to be hesitant about surgical intervention. Are there alternative treatment options available that we can discuss with her?
Transforaminal epidural selective nerve root block is an option. It serves both diagnostic and therapeutic intervention.

Can you comment on the image below?
This is an intraoperative fluoroscopic image illustrating a left L4/L5 transforaminal epidural block, which provides both diagnostic and therapeutic purposes.

What surgical procedure would you recommend after exhausting all nonsurgical options?
I would recommend a microscopic decompression and discectomy for the left L5/S1 disc protrusion.

Further Reading

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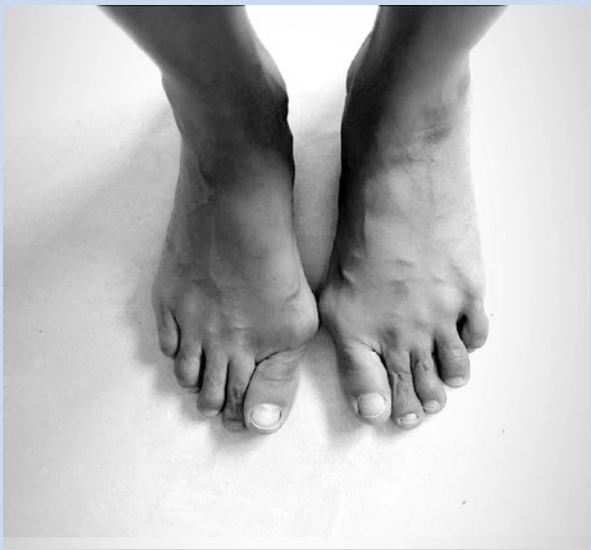
Foot and Ankle

8

Behrooz Mostofi and Jassim Al Saei

8.1 Viva 55



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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_8

- **What can you see?**
- **What is hallux valgus?**
- **What are the risk factors for this condition?**
- **What is the pathoanatomy of this condition?**
- **What causes pain in this condition?**
- **How would you manage this patient?**
- **What are the possible complications following surgical treatment?**

What can you see?
This is a clinical photograph, a weight-bearing frontal view of both feet showing hallux valgus deformity with the hallux over-riding the second toes. Valgus alignment is accompanied by a pronation deformity of the hallux.

What is hallux valgus?
Hallux valgus (HV) is defined as a lateral deviation of the proximal phalanx on the 1st metatarsal head that is frequently associated with medial deviation of the 1st MT.

What are the risk factors for this condition?
- Genetic predisposition (70% of patients with hallux valgus have a family history)
- 2nd toe deformity—amputation
- Pes planus
- Metatarsus primus (rotation and angulation of the first metatarsal away from the second metatarsal)
- Rheumatoid arthritis
- Cerebral palsy
- Ligamentous laxity
- Narrow-toed, high-heeled footwear

What is the pathoanatomy of this condition?
The sequence of events leading to deformity in the first metatarsophalangeal (MTP) joint is as follows:

1. The proximal phalanx deviates laterally.
2. The first metatarsal (1st MT) deviates medially.
3. The sesamoid complex shifts to a lateral position relative to the 1st MT head.
4. The medial capsule of the 1st MTP joint weakens progressively, while the lateral capsule contracts.
5. The abductor hallucis muscle becomes more plantar on the medial aspect of the 1st MTP joint.
6. This leaves the adductor tendon unopposed, exerting a deforming force laterally by attaching to the proximal phalanx and the lateral sesamoid.

7. Finally, the flexor hallucis brevis, flexor hallucis longus, and extensor hallucis longus increase their valgus moment on the MTP joint, further deviating the first ray. With time, the windlass mechanism is lost, resulting in a loss of weight-bearing under the 1st MT and its transfer to the lesser metatarsals (transfer metatarsalgia).

What causes pain in this condition?
- Pain from inside the MTP joint: because of joint incongruity, synovitis, or arthritis.
- Pain from outside the joint: from prominence of the bunion.
- Pain at the soles of foot: from 2nd metatarsalgia, or pressure on the 2nd toe.

How would you manage this patient?
Assuming a complete evaluation that includes a detailed history, physical examination, and radiographs (specifically, standing x-rays of both feet to measure key parameters like HVA, IMA, DMAA, and PPAA) was obtained, the management of hallux valgus typically follows these steps:

Non-surgical treatment:
- Shoe wear modifications including low heeled shoes-wide toe box.
- Bunion pads and toe spacers.
- Metatarsal padding.
- Medial arch support insoles to limit mid and forefoot pronation.

If non-surgical measures do not yield satisfactory results, surgical options can be considered.

The goals of treatment:
- Alleviate pain.
- Correct the deformity.
- Regain the function of the first ray.

Surgical decision depends on:
- 1st MTP arthritis
- 1st TMT hypermobility
- Radiographic parameters of HV (HVA, IMA, DMAA) (Table 8.1).

Table 8.1 Radiographic measurements in hallux valgus

Angle	Normal	Mild	Moderate	Severe
HVA	<15	<20	<40	>40
IMA	<9	10–12	13–15	>15

Surgical management principles:

1. If the MTP joint displays arthritis or severe hallux valgus with joint subluxation, or if neuromuscular disease is present, consider MTP fusion.
2. Check TMT joint; if it is hypermobile or arthritic, the Lapidus procedure (TMT fusion) may be necessary.
3. The choice of surgical procedure depends on the severity of hallux valgus:
   (a) For mild deformities, a lateral release and distal osteotomy are suitable.
   (b) Moderate to severe deformities may require a lateral release and either a Scarf or proximal osteotomy or even a Lapidus procedure.
   (c) In cases of severe deformity with altered DMAA, a lateral release and Scarf/double osteotomy may be needed.
   (d) If there is hallux valgus interphalangeus, an Akin procedure may be considered.
   (e) All bony procedures should be combined with soft tissue procedures; neither bony alone nor soft tissue alone is recommended.

The algorithm of surgical management is provided in the form of Fig. 8.1.

What are the possible complications following surgical treatment?

1. Recurrence: most common cause of failure is insufficient preoperative assessment and failure to follow indications.
2. Avascular necrosis.
3. Dorsal malunion with transfer metatarsalgia: associated with shortening of MT.
4. Hallux varus: overcorrection of 1st IMA—excessive lateral capsular release with overtightening of medial capsule—over resection of medial first metatarsal head—lateral sesamoidectomy.
5. Cock up toe deformity: due to injury of FHL—most severe complication with Keller resection. It can happen as a result of tight EHL in severe deformity corrections.

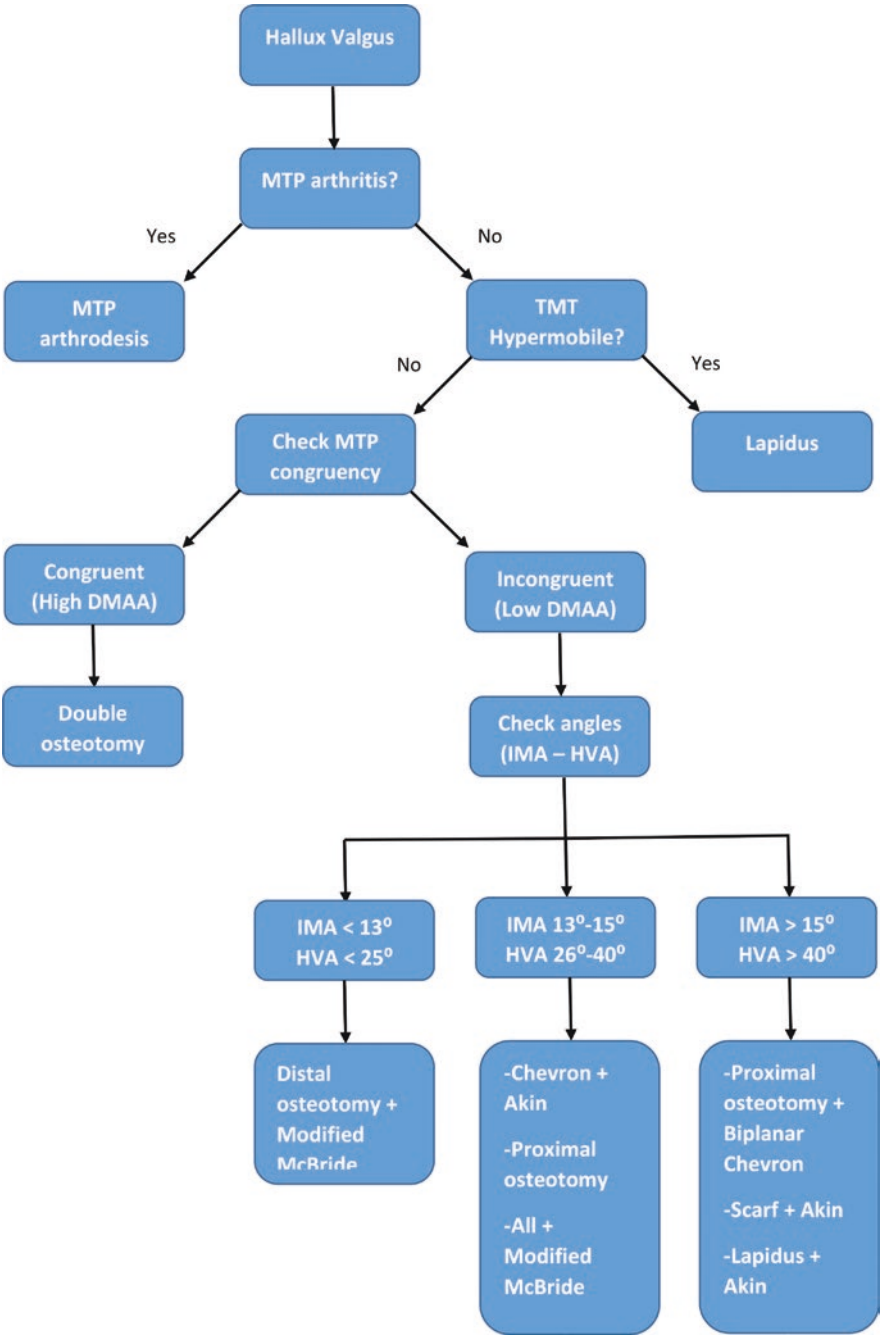
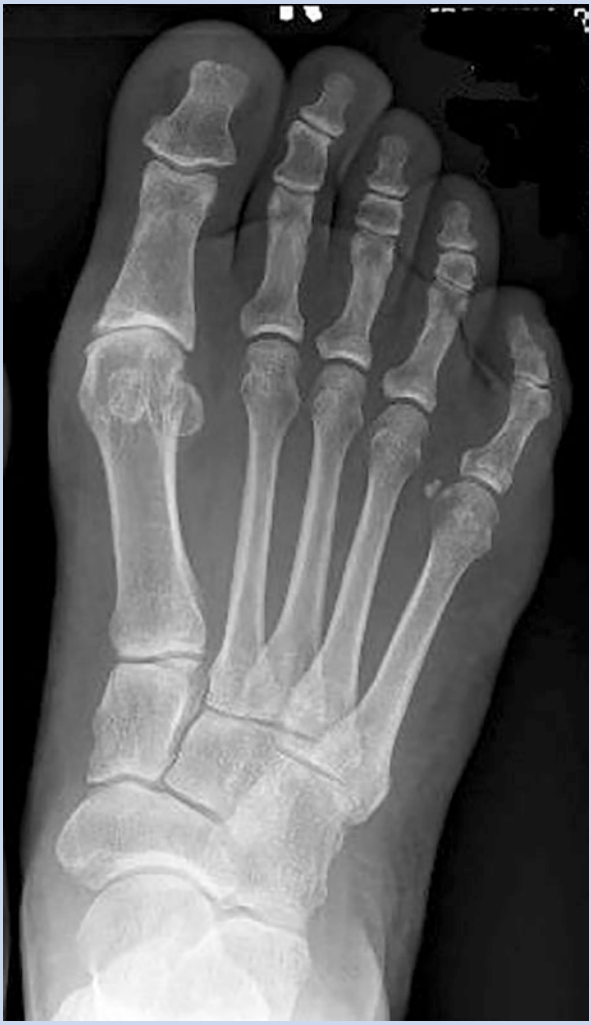


Fig. 8.1 The algorithm of surgical management for hallux valgus

8.2 Viva 56



- **Describe the radiograph.**
- **What is the aetiology of this condition?**
- **Are you aware of any classification system for this condition?**
- **How would you manage this condition?**

Describe the radiograph
This is a radiograph of a right foot showing osteoarthritis of the first metatarsophalangeal joint (MTPJ) with loss of joint space, osteophytes, and sclerosis. This condition is called hallux rigidus which is a degenerative arthritis disease of the first MTP joint. It leads to significant limitation in the range of motion of the first MTP joint. Like other degenerative arthritis processes, the formation of osteophytes is quite common. These osteophytes can lead to a mechanical hindering of MTP dorsiflexion.

What is the aetiology of this condition?
No primary aetiology of hallux rigidus has been defined. However, it is believed that repetitive microtrauma or an acute traumatic event can be the cause.

Are you aware of any classification system for this condition?
Coughlin and Shurnas' classification system for hallux rigidus is divided into four stages, which describe the severity of the condition:

Grade 1: Mild:
- Mild pain at the extremes of motion.
- Minimal joint degeneration.
- Full range of motion.

Grade 2: Moderate:
- Moderate pain with range of motion.
- Reduced range of motion, especially during dorsiflexion.
- Moderate dorsal osteophyte, <50% joint space narrowing.

Grade 3: Severe:
- Pain at the extremes of motion. No pain at mid-range.
- Restricted range of motion, both actively and passively.
- Severe dorsal osteophyte, >50% joint space narrowing.

Grade 4: End-stage:
- Pain at the extremes of motion and at mid-range.
- Restricted range of motion, both actively and passively.
- Advanced joint degeneration with large bone spurs.
- Severe dorsal osteophyte, >50% joint space narrowing.

This classification helps in determining the stage of hallux rigidus and guides the selection of appropriate treatment options.

How would you manage this condition?
I would start with non-operative management:

- Reassurance: The radiological stage is not always related to clinical symptoms, which may progress slowly.
- Activity modification.
- NSAIDs.
- Orthotic devices that increase the rigidity of the forefoot portion of the shoe to limit MTP dorsiflexion (Morton extension) or rocker bottom insoles or shoes. There are plenty on the market now (MBT or Sketchers brand).
- MUA and intra-articular steroid injection may provide relief of symptoms in mild/moderate cases. Not proven to be effective if severe changes are present.

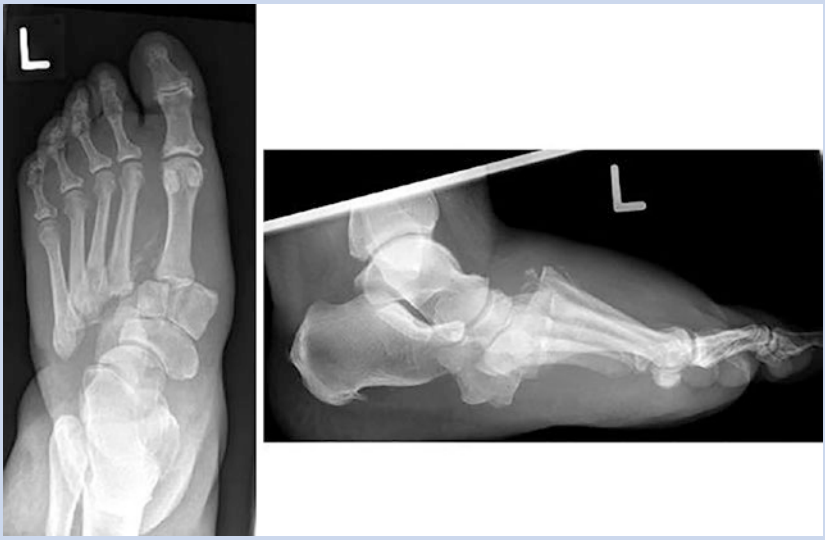
Surgical treatment depends on the grade of the disease.

Joint preserving procedures if the grind test is negative
- Joint debridement and synovectomy in acute chondral or osteochondral injuries.
- Cheilectomy (resection of the dorsal osteophyte along with removal of 25–30% of the dorsal aspect of MT head). Pain at the extremes of motion of MTP joint is an indicator of good prognosis. Cheilectomy does not work when hallux rigidus is severe, if degenerative changes present, then increased ROM can lead to more symptoms.
- Dorsal closing wedge osteotomy (Moberg) of the proximal phalanx is used to increase dorsiflexion of the MTP joint. Usually combined with cheilectomy and is indicated if cheilectomy doesn't provide at least 30–40 degrees of dorsiflexion. Moberg is rarely done with good results from 1st MTPJ fusion.

Joint sacrificing procedures:
- Resection arthroplasty (Keller procedure) involves removal of the base of the proximal phalanx. It can destabilize the joint leading to cock up deformity, weakness during push off and transfer metatarsalgia. Used in elderly or sedentary patients Again very rarely done. It should be last on the list just for completion sake.
- MTP joint replacement (hemi/total): Limited long-term evidence to support use.
- Arthrodesis of the MTP. The most commonly used procedure. The preferred alignment is 10–15° valgus-15 degrees dorsiflexion-neutral rotation to ensure an effective plane of motion of the IPJ, using two cannulated screws. The IP joint should be mobile (accelerates IP joint arthritis).

8.3 Viva 57

A 75-year-old diabetic lady presented with right foot pain—swelling and redness; no trauma was recalled.



- **What do you see?**
- **What is Charcot arthropathy?**
- **How can you differentiate Charcot foot from infection?**
- **What are the stages of Charcot arthropathy?**
- **Are you aware of any classification system?**
- **What are the principles of treating this condition?**

What do you see?
These are anteroposterior and lateral view radiographs of a severely deformed foot. Multiple dislocations are evident at the TMT (tarsometatarsal) and intercuneiform joints, along with lateral and dorsal displacement. Additionally, there are multiple areas of sclerosis. Based on the patient's presenting symptoms and radiographic findings, Charcot arthropathy is suggested as possible diagnosis.

What is Charcot arthropathy? Charcot arthropathy is the progressive destruction of bone and soft tissue that leads to loss of bony architecture, fracture, dislocation, and deformity. Up to 7.5% of patients with diabetes and neuropathy develop Charcot arthropathy.

How can you differentiate Charcot foot from infection?

- Erythema will decrease with elevation in Charcot (for 10 min).
- Lack of significant elevation of fever and WBC count.
- Blood glucose levels usually fluctuate with infection, so normal blood glucose level should discount infection in the differential diagnosis.

What are the stages of Charcot arthropathy?

Eichenholtz has staged this process:

- Stage 0: acute inflammatory phase: the foot becomes painful, swollen (oedematous), and warm (erythematous). X-rays may show periarticular soft tissue swelling and varying degrees of osteopenia. This stage can be difficult to differentiate from an acute infection.
- Stage 1: fragmentation stage. Over the following weeks the oedema and erythema settle, although the foot can continue to change shape (unless protected) as the bone continues to fragment.
- Stage 2: Coalescence: the foot continues to settle and starts to stiffen up and the deformities become fixed. X-rays show resorption of bone debris.
- Stage 3: Consolidation: over many months the oedema and erythema completely settle. X-rays show consolidation and remodelling of fracture fragments. (As a rough guide: forefoot 6 months, midfoot 12 months, hindfoot 18 months)

Are you aware of any classification system?

Brodsky anatomic classification system (Fig. 8.2):

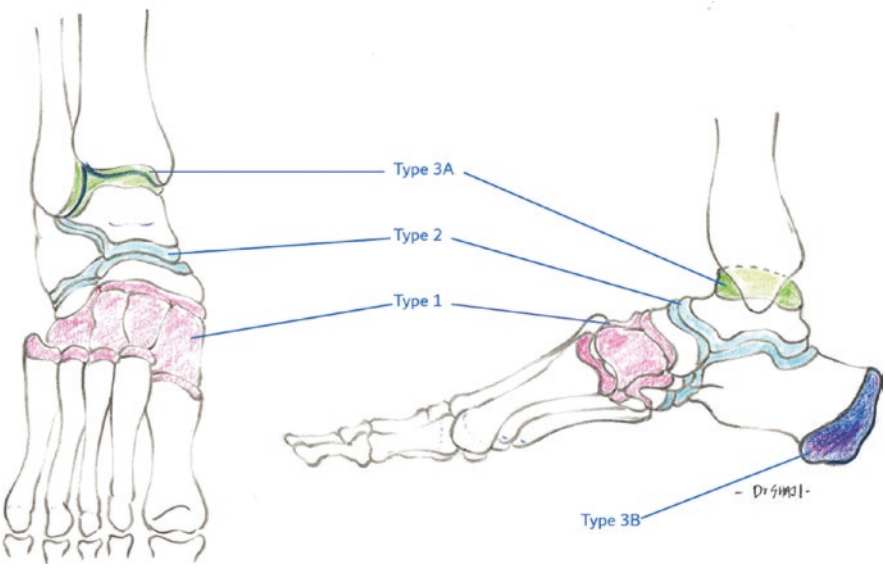


Fig. 8.2 Brodsky's anatomic classification of the Charcot foot

- Type I: involves tarsometatarsal and naviculocuneiform joints. It is the most common type.
- Type II: involves subtalar, talonavicular, calcaneocuboid.
- Type III: involves tibio-talar joint. A: tibio-talar. B: fracture of calcaneal tuberosity.
- Type IV: combination of area.
- Type V: forefoot involvement.

What are the principles of treating this condition?
- Prevention: optimum management of co-morbidities (diabetes).
- Goals of treatment are: plantigrade, stable foot that can fit into a shoe and the prevention of recurrent ulceration.
- TCC is the gold standard of treatment. It permits an even distribution of the foot pressures across the plantar surface of the foot. Weight bearing should be restricted. Casts should be changed (every week in presence of ulcer or swelling or every 2 weeks if there is no ulcer or swelling) until erythema and oedema have resolved and the temperature has reduced. TCC is commonly continued for up to 4 months. Once the active phase has ended, the patient can be fitted with a Charcot restraint orthotic walker and later with custom shoe with orthosis.
- Surgery performed in the inflammatory phase has a high rate of non-union, infection, wound complications, late deformity and amputation.
- Indications for surgical intervention: If a plantigrade weight-bearing surface cannot be achieved, recurrent ulcers and instability.
- Surgical options: exostectomy, reconstruction with osteotomy and fusion, amputation.

8.4 Viva 58

What do you see?
This is a clinical photograph of left foot with a cavus deformity. The hindfoot is in varus and there is elevated medial longitudinal arch.

What are the causes of this condition?
The causes of a cavus foot may be broken down:

- Congenital: idiopathic (most idiopathic cases are simple cavus without varus deformity)-arthrogryposis-sequela of clubfoot.
- Acquired: traumatic or neuromuscular. The neuromuscular causes may be grouped into central nervous system disease such as cerebral palsy or Friedrich's ataxia, spinal cord lesions such as spina bifida or spinal dysraphism, peripheral nervous system lesions such as an HSMN or muscular causes such as muscular dystrophy, crush injury or compartment syndrome.

What is the pathoanatomy of this condition?
The pathoanatomy of Pes cavus results from an imbalance in foot musculature. The

- **What do you see?**
- **What are the causes of this condition?**
- **What is the pathoanatomy of this condition?**
- **What is Coleman Block test?**
- **What are the principles of management?**

primary structural issue involves forefoot plantar flexion, especially in the 1st ray. This can result from the relative weakness of the tibialis anterior compared to the peroneus longus, but more commonly, it is due to intrinsic weakness and contracture. This imbalance leads to an increase in arch height and forefoot pronation.

Several factors contribute to the development of hindfoot varus deformity:

1. Compensatory Hindfoot Varus: The hindfoot assumes a compensatory varus posture to counterbalance the forefoot pronation.
2. Weakened Peroneus Brevis: Hindfoot varus develops because the weakened peroneus brevis cannot counteract the intact tibialis posterior.

The elevation of the arch occurs due to the tightening of the windlass mechanism, resulting from the imbalance between weakened intrinsic and extrinsic muscles. Additionally, clawing of the toes develops as a result of the loss of intrinsic function (MTP flexion and IP extension), leading patients to compensate by using the toe extensors, resulting in MTPJ hyperextension and IPJ flexion. The extensor hallucis longus (EHL) serves as an accessory dorsiflexor in the absence of the tibialis anterior.

Over time, the plantar fascia contracts, and the hindfoot varus deformity becomes more rigid.

What is Coleman Block test?

The Coleman block is a diagnostic clinical test used to assess the flexibility of hindfoot deformities, particularly in cases of cavus feet. In cavus feet, the first ray is often in a plantarflexed position, which necessitates the hindfoot to move into varus for the foot to be stable on the ground.

During the Coleman block test, the patient's foot is positioned so that the lateral border of the foot and the heel rest on a block, while the medial forefoot hangs off the edge of the block. The test is considered positive if, when viewed from behind, the heel assumes a physiological alignment of neutral to 5 degrees of valgus. This finding indicates that the hindfoot deformity is both flexible and primarily driven by the forefoot.

What are the principles of management?

The goal of treatment:

- Pain relief.
- Preserve function.
- Protect the foot & ankle from further deformity.

Non-operative treatment indicated in flexible mild cases

- Physiotherapy: eversion & dorsiflexion strengthening program—Achilles Tendon stretching exercises.
- Orthotic: semi-rigid insole with depression for 1st ray – heel lift & lateral heel wedge.
- In mild rigid deformities, accommodative shoes can be used.

Operative treatment: (Depends on aetiology)
Flexible: (hindfoot corrects in Coleman block test):
Bony procedures:

- Dorsiflexion osteotomy of the 1st MT.

Soft tissue procedures:

- Plantar fascia release.
- PTT transfer to the dorsum of the foot through the interosseous membrane (to decrease the varus moment.
- ± transfer of peroneus longus tendon to peroneus brevis, to eliminate its deforming effect.
- ± Achilles lengthening.

Rigid: (hindfoot doesn't correct in Coleman block test):
- Add lateralizing calcaneal osteotomy (Dywer).

Rigid deformities with arthritis:
- Triple arthrodesis.

Claw Toes:
- Girdlstone-Taylor procedure (transfer of FDL to EDL) in flexible claw lesser toes.
- Modifies Jones procedure (EHL transfer to 1st MT neck + IP joint arthrodesis) in flexible claw hallux.

8.5 Viva 59

Describe what you see
This is a clinical photograph showing a posterior view of feet in weight-bearing position. There is marked heel valgus and too many toes are visible. The medial longitudinal arch is not visible.

Clinical findings are consistent with flatfoot or pes planus.

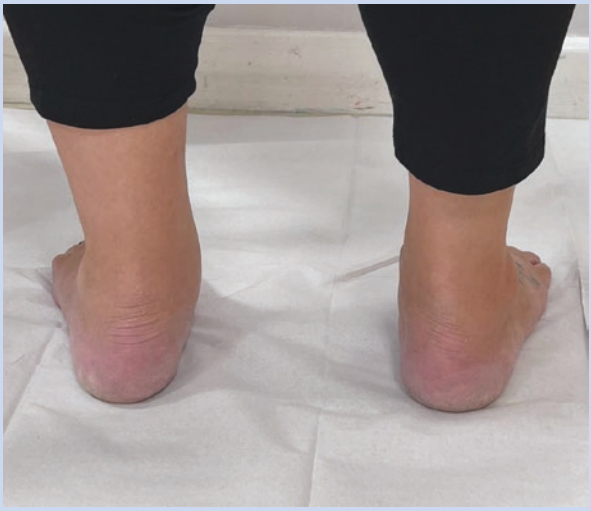
In adults, it is usually acquired and commonly due to tibialis posterior dysfunction.

Other causes include inflammatory arthritis, Charcot arthropathy, osteoarthritis and trauma.

It is commoner in females and the incidence increases with age.

What is the function of posterior tibial tendon PTT?
The PTT lies in an axis posterior to the ankle joint and medial to the axis of the subtalar joint.

It acts as an invertor of the hindfoot and adducts and supinates the forefoot during the stance phase of the gait. It acts as a secondary plantar flexor at the ankle joint.



- **Describe what you see.**
- **What is the function of posterior tibial tendon PTT?**
- **What is the classification system used for this condition?**
- **What are the treatment options?**

Activation of the PTT allows locking of the transverse tarsal joints creating a rigid lever arm for the toe-off phase of the gait.

The major antagonist of the PTT is peroneus brevis.

What is the classification system used for this condition?

Johnson classification

Stage I: painful stage, PTT tenosynovitis.

Stage II: flexible deformity. Subdivided into:

- IIA: <40% talonavicular uncoverage.
- IIB: >40% talonavicular uncoverage.
- IIC: 1st TMT instability.

Stage III: rigid deformity.

Stage IV: fixed deformity with valgus instability of the ankle joint (deltoid insufficiency).

What are the treatment options?

Stage I

Non-operative

- Initial period of immobilization followed by shoe orthosis (UCBL with medial posting) + NSAIDs
- Physiotherapy

Operative:

- Tenosynovectomy

Stage II
Non-operative

- Similar to stage I

Operative:

- IIA: medializing calcaneal osteotomy + FDL transfer to navicular bone or to intact PTT ± ETA ± spring ligament reconstruction. ± Cotton osteotomy or Naviculocuniform fusion (create the medial arch)
- IIB: add lateral column lengthening (Evan's osteotomy)
- IIIC: if the 1st TMT is unstable or arthritic >> fusion

Stage III
Non-operative

- AFO or Arizona

Operative:

- Triple arthrodesis

Stage IV
Non-operative

- Similar to III

Operative:

- If tibiotalar is not arthritic >>triple arthrodesis + deltoid reconstruction.
- If tibiotalar is arthritic >>pantalar arthrodesis (triple + tibiotalar).

8.6 Viva 60

Describe the radiograph

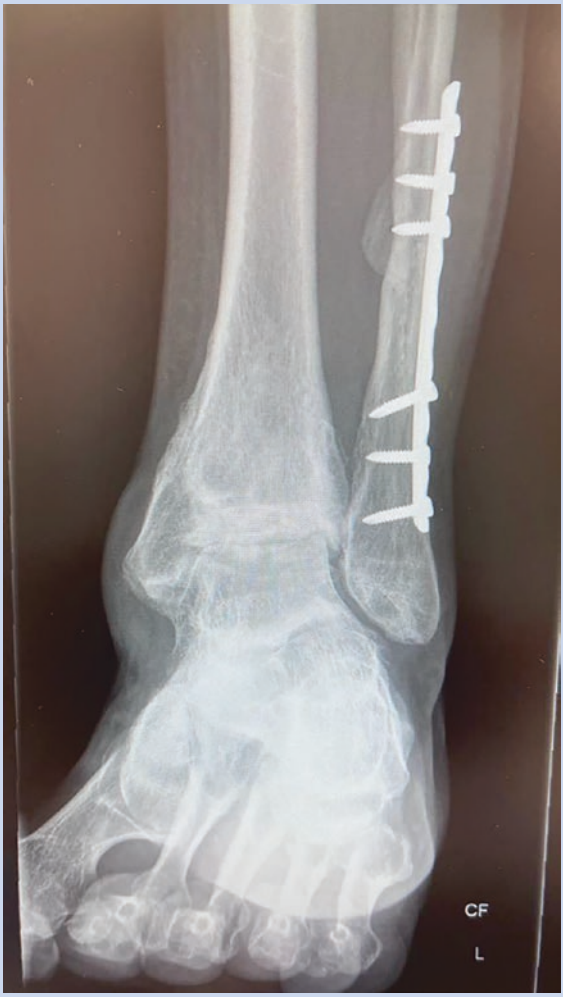
This is an AP weight-bearing radiograph of a left ankle showing narrowing of the joint space and some subchondral sclerosis consistent with post-traumatic arthritis. There is evidence of a previous fibula fracture superior to the syndesmosis which was fixed using a plate. The Fibula looks shortened with a lateral alignment of the ankle.

What are the most common causes of end-stage arthritis of the ankle?

Post-traumatic arthritis, the most common aetiology, accounts for greater than 2/3 of all ankle arthritis.

Primary osteoarthritis accounts for less than 10% of all ankle arthritis.

Other aetiologies include rheumatoid arthritis, osteonecrosis, neuropathic, septic, gout, and haemophiliac.



- **Describe the radiograph.**
- **What are the most common causes of end-stage arthritis of the ankle?**
- **Are you aware of any classification system?**
- **What are the management options available for ankle arthritis?**

Are you aware of any classification system?
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.

What are the management options available for ankle arthritis?
Non-operative:
- Pain medications (NSAIDS).
- Footwear modification with cushioned single rocker-bottom shoe to limit motion in the ankle during gait, increase propulsion at toe-off, decrease pressure on heel strike.
- Activity modification.
- corticosteroid injection to help decrease pain.

Operative:
- Indicated upon failure of conservative treatment in a patient with radiographic evidence of ankle arthritis.
- Ankle debridement and anterior tibial/talar exostectomy can help relieve impingement and improve symptoms in mild cases with pain during push off with positive anterior impingement signs.

Arthrodesis:
- Still considered the gold standard.
- Provides reliable relief of pain and return to activities of daily living.
- 50% of patients demonstrated subtalar arthrosis 10 years following ankle arthrodesis.
- There is a 10% nonunion rate, which can be negatively influenced by such factors as smoking, diabetes, avascular necrosis, and prior arthrodesis.
- Can be done arthroscopically assisted or open arthrodesis through trans-fibular approach, this is typically used when deformity is present. Options include screws fixation or plate and screws (associated with risk of superficial nerve injury) – other option: Tibiotalocalcaneal (TTC) fusion with retrograde

intramedullary nail (load-sharing device with improved bending stiffness and rotational stability compared to plate-and-screw constructs) however, there is a risk of lateral plantar nerve injury.
- The optimum position of arthrodesis of the ankle appears to be neutral flexion, slight (0–5°) valgus angulation, and approximately 5–10 degrees of external rotation, 5 mm of posterior talar translation.

Arthroplasty:
- Patient selection is crucial.
- Indicated in inflammatory arthritis, elderly patients.
- Contraindications: uncorrectable deformity, severe osteoporosis, talus osteonecrosis, Charcot joint, ankle instability, obesity, and young labourers increase the risk of failure and revision.
- Outcomes: recent 5–10-year outcome studies demonstrate up to 90% good to excellent clinical results, long-term studies are still pending on the newest generation of ankle. arthroplasty.
- Increased stride length, cadence, and stride velocity as compared to ankle arthrodesis.
- Principles: use long incision—Develop full skin flap—Minimize use of retractors.
- Types:
  - 1st generation: cemented—highly constrained—poor prognosis.
  - 2nd generation: agility—porous two components—all poly-tibia—poor prognosis.
  - 3rd generation: mobile bearing: Scandinavian Total Ankle Replacement STAR (Tibial and talar component are cobalt chrome—talar component has ridge to guide PE—tibial component has two anchors to improve fixation)
- Prospective Controlled Trial—Foot and ankle international—2009—STAR Vs Ankle Fusion By 24 months, ankles treated with STAR had better function and equivalent pain relief as ankles treated with fusion. NJR Eight-year cumulative revision risk was 8%.

8.7 Viva 61

Describe what you can see
This is a clinical photograph that shows ankle foot orthosis (AFO). It is used for patients who have drop foot.

Describe dropped foot gait pattern
The patient cannot dorsiflex or evert the foot.

As a result, the patient tends to walk with an exaggerated flexion of the hip and knee to prevent the toes from catching on the ground during the swing phase (foot clearance)

At initial heel contact, the foot slaps on the ground due to loss of eccentric contraction of the foot dorsiflexors (referred to as steppage gait).

- **Describe what you can see.**
- **Describe dropped foot gait pattern.**
- **What are the causes of drop foot?**
- **Describe the anatomy of sciatic, peroneal, and tibial nerves.**
- **What are your management options?**

What are the causes of drop foot?

- Neurodegenerative disorder of the brain-multiple sclerosis, stroke, cerebral palsy.
- L4–L5 disc herniation-the herniated disc compressing nerve root this is by far the most common cause in clinical situations.
- Lumbosacral Plexus injury—due to pelvic fracture.
- Sciatic nerve injury-hip dislocation.
- Injury to the knee-knee dislocation—fibular head fracture.
- Motor neuron disorder-polio and amyotrophic lateral sclerosis.

Describe the anatomy of sciatic, peroneal, and tibial nerves.

Sciatic nerve:

- It is the thickest and largest nerve in the body. Arises from the ventral rami of L4–S3.

- In most (>80%) patients, the sciatic nerve lies anterior to the piriformis as it exits the pelvis through the greater sciatic notch and then runs through the interval between the piriformis and the superior gemellus to continue its course posterior to the remainder of the short external rotators. Other variations include passing superior to or piercing the piriformis.
- It ends by dividing into tibial and common peroneal nerve just above the popliteal fossa.

Tibial nerve:
- The tibial division of the sciatic nerve provides innervations to all of the hamstring muscles in the thigh (semitendinous-semimembranous—long head biceps femoris) with the exception of the short head of the biceps femoris which receives its innervations from the common peroneal branch of the sciatic nerve. Both heads of the gastrocnemius muscle are innervated by the tibial division of the sciatic nerve.
- The short head of biceps femoris was developed in the extensor compartment but migrated to the flexor compartment.

Common peroneal nerve:
- It is the smaller terminal branch of the sciatic nerve. It begins just above the apex of the popliteal fossa and descends underneath the medial border of biceps femoris, it passes inside the knee joint capsule, then passes into peroneus longus where it divides into superficial and deep peroneal nerves.
- Before dividing, the nerve gives of five branches (3 cutaneous and 2 articular):
  - Lateral sural cutaneous nerve
  - Peroneal communicating nerve
  - Recurrent genicular nerve (skin over the patella)
  - Superior and inferior genicular nerves

What are your management options?
Initially (<1 year):
- Ankle-foot orthosis (AFO)
- Physical therapy

This will maintain passive dorsiflexion, i.e. prevent Achilles shortening

Continued deficient nerve function (>1 year):
- If nerve function fails to return during the course of conservative management and the patient demonstrates intact posterior tibialis muscle strength, posterior tibial tendon transfer to the dorsum of the foot has been shown to improve

functional outcomes and eliminate the need for continued bracing. In severe cases, hindfoot arthrodesis may be needed to achieve a plantigrade foot.

8.8 Viva 62

- **Describe what you can see.**
- **Describe the anatomy of the lateral ankle ligamentous complex.**
- **How would you manage a patient with chronic ankle instability?**

Describe what you can see.
This clinical photo shows the anterior drawer test of the ankle. This test is done to assess the integrity of lateral ankle ligaments, namely anterior and posterior talofibular ligaments, and calcaniofibular ligaments. This test is positive when anterior movement of the calcaneus is appreciated in relation to tibia which is observed by a depression of skin at the anterior ankle joint line. This test can also be performed under X-ray imaging, where a positive result is characterized by an anterior displacement of talar dome in relation to its articular surface of tibia by more than 10 mm compared to the normal contralateral ankle. It is typically conducted in conjunction with the varus stress test (talar tilt test).

The talar tilt test (radiologically) examines the integrity of the calcaneofibular ligament and the anterior talofibular ligament. It is performed by grasping the foot and heel while attempting to invert the talus on the tibia. It is considered positive if there is an absolute talar tilt >10°, or when it is >5° compared with the contralateral ankle.

Describe the anatomy of the lateral ankle ligamentous complex
The lateral ligamentous complex is made up of three ligaments (Fig. 8.3):

- Anterior talofibular ligament (ATFL): extends from the anterior aspect of distal fibula (10 mm proximal to the tip of fibula) to the body of the talus. The most commonly injured ligament in ankle sprain. the weakest ligament. Resists anterior translation of the talus. Strain in the ATFL increases with plantar flexion 20° and inversion. (ATFL is perpendicular to the long axis of the tibia, with ankle plantar flexion, ATFL becomes more parallel to the tibia.)
- Calcaneofibular ligament (CFL): extends from the tip of the fibula to the lateral wall of the calcaneus. Strain in CFL increases with dorsiflexion and inversion. (CFL is parallel to the tibia in neutral ankle flexion and on average at 105 degrees from ATFL.)
- Posterior talofibular ligament (PTFL): originates from the posterior fibula and inserts in the posterolateral tubercle of the talus. It is the strongest ligament. Under strain in dorsiflexion.

How would you manage a patient with chronic ankle instability?

- Non-operative management can be started with peroneal muscle strengthening and proprioception training as this can help is stabilizing the ankle.

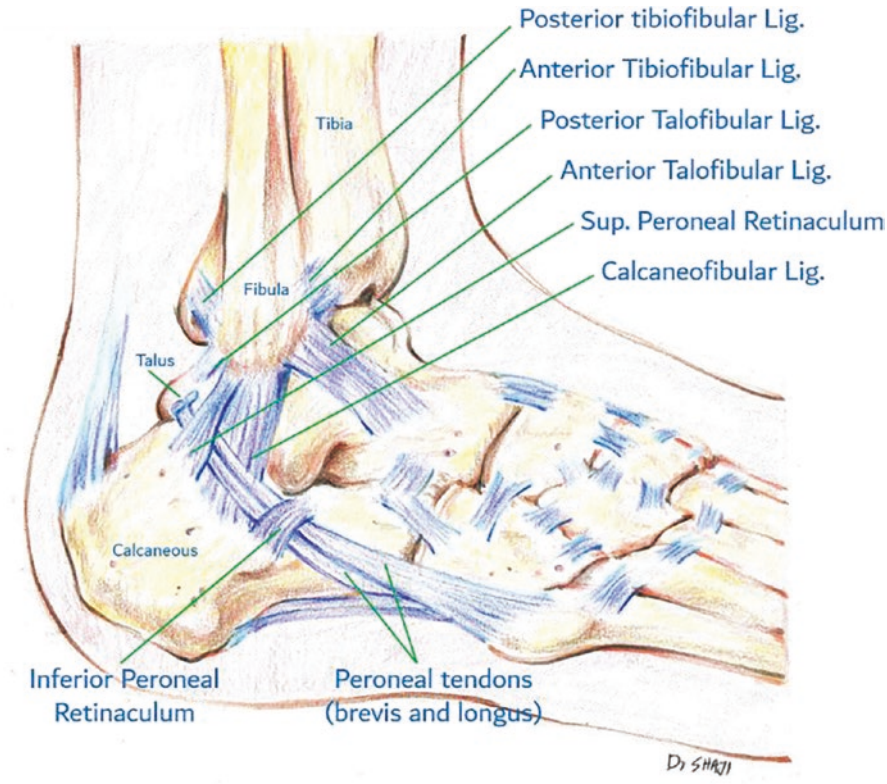


Fig. 8.3 Anatomy of the lateral ankle ligamentous complex and related structures

- Surgical treatments are broadly grouped into anatomical repair of the ligaments and non-anatomical repairs.

Anatomical repair
Brostrom Gould:

- Original Brostrom: direct repair of attenuated ligaments.
- Gould modification: direct repair of attenuated ligaments (ATFL—CFL) + augmentation using inferior extensor retinaculum.
- Good results in 90%.

Non-anatomical repair
Evans:

- Utilizes the entire peroneus brevis tendon with tenodesis to the fibula from the tip emerging posteriorly 3cm proximal, suturing its muscle belly onto peroneus longus.
- It limits inversion, but doesn't restrict anterior translation

Watson–Jones:

- Peroneus Brevis (PB) through fibula from posterior to anterior then into talus.
- Limits anterior translation and inversion.

Chrisman–Snook:

- Utilizes a split of PB through talus, fibula from anterior to posterior, then to calcaneus.
- If there is varus malalignment, consider correction with Dwyer lateralizing calacanel osteotomy if the malalignment is fixed, or a 1st metatarsal (MT) osteotomy if it is found to be flexible, which can be determined through the Coleman block test.

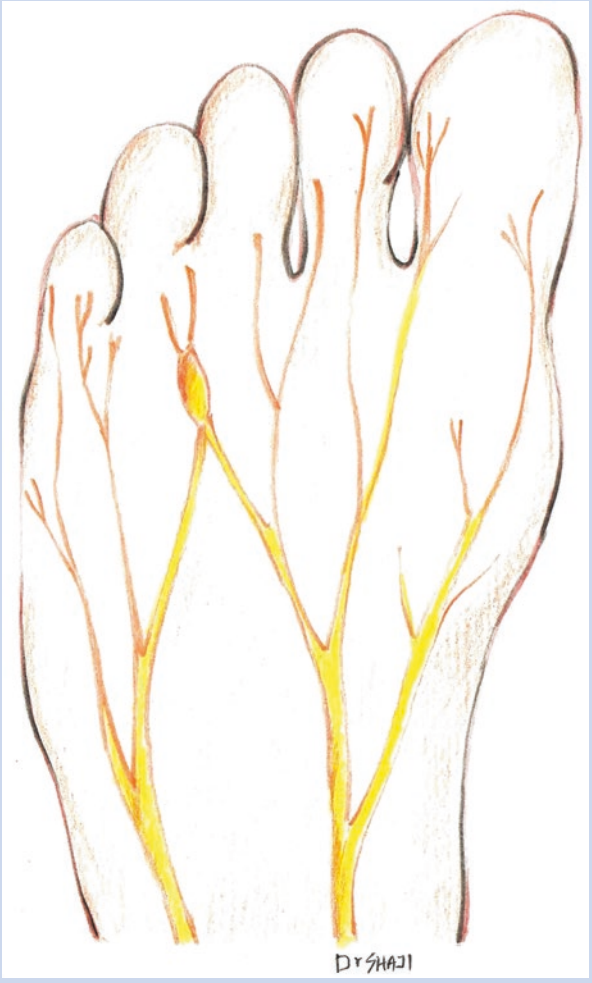
8.9 Viva 63

What is Morton's neuroma (interdigital neuroma)?
Morton's neuroma is a perineural thickening of the common digital nerve at the webspace of the foot.

It is most commonly seen in women (4:1, female-to-male ratio).

It is most commonly found between the third and fourth toes followed by 2nd and 3rd toes. It receives branches from both medial & lateral plantar nerves.

What causes this condition?
The aetiology is uncertain.



- **What is Morton's neuroma (interdigital neuroma)?**
- **What causes this condition?**
- **What is Mulder's sign?**
- **What diagnostic and/or radiographic tests are helpful in diagnosing Morton's neuroma?**
- **How would you treat it?**

Several causative factors have been suggested, although none is universally accepted:

- Anastomosis between the medial and lateral plantar nerves in third webspace
- High-heeled shoes with narrow toebox: Forced toe dorsiflexion
- Compression by the transverse intermetatarsal ligament
- Bursal hypertrophy

What is Mulder's sign?
The Mulder sign is elicited by squeezing the foot while palpating the web space. A tangible "click" can be felt that indicates the presence of a Morton's neuroma.

What diagnostic and/or radiographic tests are helpful in diagnosing Morton's neuroma?
The diagnosis of a Morton's neuroma is based on history and physical examination.

An injection of local anaesthetic into the webspace between the metatarsal heads causing resolution of the patient's symptoms favours a diagnosis of Morton's neuroma also helps with symptoms in case of bursitis of metatarsal heads). If no relief is obtained, a second injection can be administered to the MTP joint, with a repeat exam noting any improvement in the patient's symptoms.

Standing AP and lateral weight-bearing films to exclude other forefoot pathology.

MRI (preferred over ultrasound scan but has false positive rate).

U/S is another modality, it is however operator dependent.

How would you treat it?
Approximately 80% of patients will have complete resolution of symptoms with non-operative treatment**.**

The goals are to alleviate pressure on the nerve by decreasing tension on the inter-metatarsal ligament and to reduce compression of the forefoot, which may be accomplished through:

- Use of shoes with a wide toe box, firm sole, and a more rigid arch support.
- Metatarsal pads also may help to relieve pressure on the nerve.

Anti-inflammatory medication rarely offers any benefit.

Local corticosteroid injection may be helpful in relieving symptoms, but repeated injections are contraindicated.

Surgery should only be reserved for patients who fail non-surgical treatment. It is important to inform the patient that surgical excision does not always provide relief and recurrence is possible. (Recurrence mainly due to leftover plantar branches).

Dorsal incisions typically recommended for resection of primary neuroma.

Plantar incisions are used for recurrent neuromas. (it has better visualization as nerve plantar to deep transverse ligament and, hence, easier exposure with plantar exposure but results in painful scar).

Surgical principles:

- Divide transverse inter meta-tarsal ligament.
- Excise nerve 2–3 cm proximal to the bifurcation and then both branches.
- Send specimen to histology for confirmation of diagnosis (Shows perineural fibrosis with thickened walls, demyelination, and degeneration of nerve fibre).

Further Reading

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Part IV

Hand and Paediatrics

Hand

9

Behrooz Mostofi and Mazhar Fuad

9.1 Viva 64

Image 1

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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_9

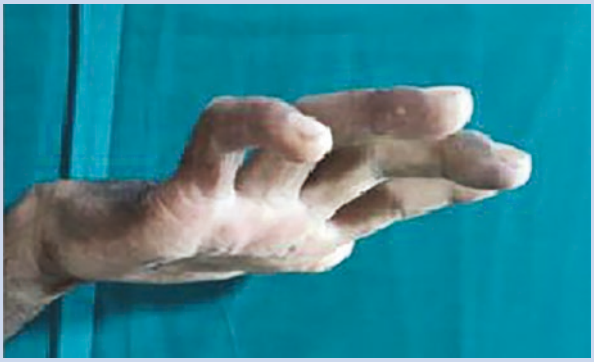


Image 2

- **Describe the images.**
- **What is the underlying pathology?**
- **What are the problems with intrinsic weakness of hand?**
- **What are the factors to be considered in management of claw hand?**

Describe the images

Image 1 shows a hand with flexion at the MCP joints and extension of PIPJs and DIPJs. Features of intrinsic plus hand.

Image 2 shows wrist flexion, MCPJs extension, flexion of the PIP and DIP joints. Features of intrinsic minus hand.

What is the underlying pathology?

- The deformity is produced by an imbalance between the intrinsic and extrinsic muscles of the hand.
- If the extrinsic muscles are weakened or paralyzed, the spastic intrinsic muscles → MCPJs flexion and extension of the IPJs. Causes: CVA, cerebral palsy, brain injury.
- If the intrinsic muscles are weakened or paralyzed, there is unopposed pull of the normal long extensors → hyperextension of the MCPJs, while the long flexors cause flexion of the PIP and DIP joints (intrinsic minus). Causes: combined median and ulnar nerves palsy, brachial plexus injuries, Volkmann's ischemic contracture, compartment syndrome of the hand, leprosy, Neurological (CMT, Polio, amyotrophic lateral sclerosis).

What are the problems with intrinsic weakness of hand?

The three main problems caused by intrinsic weakness of the fingers are:

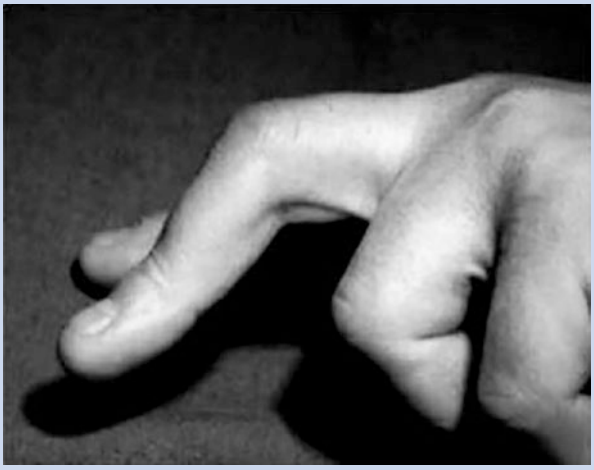
1. Clawing with loss of synchronistic finger flexion (MCP flexion occurs only after IP joint flexion) rather than simultaneously inhibiting the grasp of large objects.

2. Inability to abduct and adduct the digits (decreasing pinch strength).
3. Weakness of grip.

What are the factors to be considered in management of claw hand?

- Patient commitment and compliance.
- Identifying the motor deficits.
- Determine the active and passive motion of the digits.
- Is there adequate sensibility? The outcomes of surgery can be improved if sensibility can be restored.
- Can the claw deformity be passively corrected?
- Bouvier's test. (Can blocking hyperextension of MCP improve IP extension?)
- Joint stiffness and contractures need to be addressed before considering any tendon transfer procedures.
- What are the expendable and appropriate motor donors?
- What is the quality of the soft tissues along the path of the tendon transfer?

9.2 Viva 65



- **What can you see?**
- **What is the underlying pathoanatomy?**
- **Describe Elson test.**
- **What is the management of this condition?**

What can you see?
This clinical photograph demonstrates flexion of PIP joint and extension of the DIP joint of the middle finger. Clinical picture of Boutonniere deformity.

What is the underlying pathoanatomy?
The mechanism underlying this deformity is dysfunction of the central slip (inserting into the base of the middle phalanx). Weakening or disruption of the central slip leads to loss of active PIPJ extension. Subsequent attenuation of the triangular ligament results in volar migration of the lateral bands. The lateral bands fall volar to the axis of rotation of the PIPJ, becoming a flexor of the PIPJ. The lateral bands then transmit their force to the extension of the DIPJ.

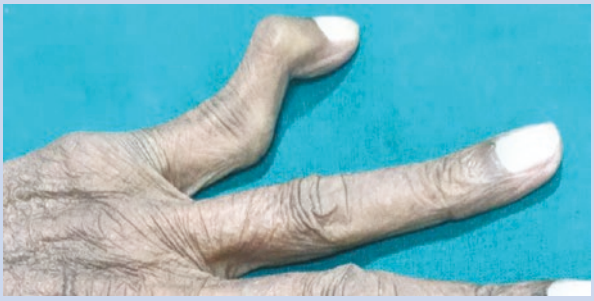
The mechanism of injury: laceration over the dorsum of the PIPJ; palmar dislocation of the PIPJ.

Describe Elson Test
It is the most reliable way to diagnose a central slip injury before the deformity is evident. Bend PIP 90° over edge of a table and extend middle phalanx against resistance. In the presence of central slip injury there will be weak PIP extension and the DIP will go rigid due to recruitment of the lateral bands.

In the absence of central slip injury DIP remains floppy because the extension force is now placed entirely on maintaining the extension of the PIP joint, the lateral bands are not activated.

What is the management of this condition?
- In acute injury <4 weeks.
  - Surgery for avulsion fracture and open injuries. Consider re-inserting central slip with mini anchor.
  - Closed injuries: PIPJ splint for 6 weeks followed by dynamic splinting (Capener) for 4–6 weeks with encouragement of DIPJ ROM to avoid contracture of the oblique ligament.
- In chronic injury
  - Flexible: Lateral band relocation or terminal tendon tenotomy (modified Fowler) or central slip reconstruction using lateral band (Matev or Littler procedures).
- In painful stiff joint
  - Arthrodesis.

9.3 Viva 66



- **What is the pathology shown in this photograph?**
- **What is the underlying pathoanatomy and the causes of this condition?**
- **What is the treatment of this condition?**

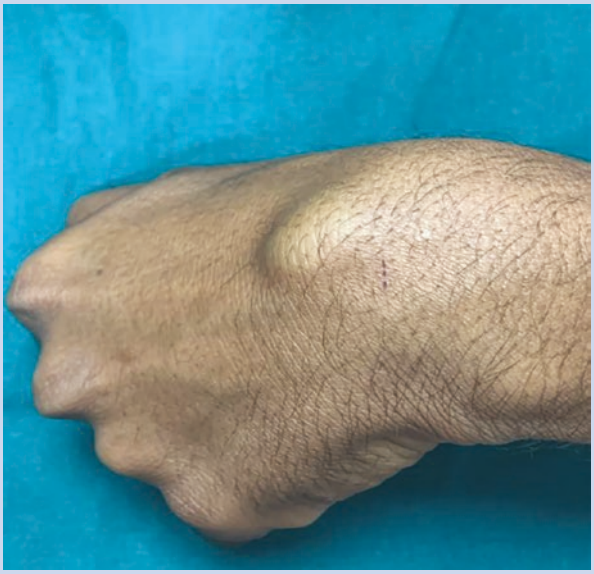
What is the pathology shown in this photograph?
This photograph demonstrates a swan neck deformity, characterized by hyperextension of the PIP joint with flexion of the DIP joint.

Unlike Boutonniere, this condition can be secondary to problems in MCP and DIP.

What is the underlying pathoanatomy and the causes of this condition?
- PIPJ
  - FDS rupture
  - Volar plate dysfunction (trauma—ligamentous laxity—rheumatoid arthritis).
- MCPJ
  - Volar subluxation in rheumatoid arthritis.
- DIPJ
  - Mallet injury. Transfer of DIP extension force into PIPJ extension.

What is the treatment of this condition?
- Splinting with double ring splint.
- In progressive deformity: volar plate advancement + central slip tenotomy. FDS tenodesis in FDS rupture.

9.4 Viva 67



- **Describe the photograph.**
- **What is the differential diagnosis?**
- **Give a definition of this condition.**
- **What are the common locations of this condition?**
- **How would you confirm the diagnosis?**
- **How would you manage this condition?**

Describe the photograph

This is a clinical photograph of the left hand and wrist demonstrating a swelling over the dorsoradial aspect of the wrist. This is the typical site for a dorsal wrist ganglion.

What is the differential diagnosis?

1. Pigmented villonodular synovitis (giant cell tumour of the tendon sheath).
2. Epidermal inclusion cyst.
3. Neurilemmoma.
4. Foreign body granuloma.
5. Fibroma of tendon sheath.

Give a definition of this condition

Ganglion is a mucin-filled synovial cyst lined by compressed collagen and a few cells. No epithelial or synovial lining.

It contains mucin, which is a mixture of glucosamine—albumin—globulin and hyaluronic acid.

It is the most common hand mass.

What are the common locations of this condition?

- Dorsal carpal (70%).
  - Originate from SL articulation.
- Volar carpal (20%).
  - Originate from radiocarpal or STT joint.
- Volar retinacular (10%).
  - Originate from herniated tendon sheath fluid.

(Volar ganglions have higher recurrence rate)

How would you confirm the diagnosis?

- Take a history specifically asking about any fluctuation in size. Clinically this is a firm, smooth swelling attached to deep structures, but not to the skin and are commonly translucent to light illumination.
- Radiographs of a ganglion will be normal, although a T2-weighted MRI axial image of the wrist will show increased signal where the cyst is located.

How would you manage this condition?

- Counsel the patient this is a benign condition. If there is no history of increasing size or pain this may be the only treatment necessary. If there is pain affecting function especially with forced wrist extension, then surgical excision may be considered. Small occult dorsal ganglions maybe more symptomatic than larger ones. The recurrence rate is 5% (requires adequate exposure to identify origin and allow resection of stalk and a portion of adjacent capsule; at dorsal DIP joint: must resect underlying osteophyte).
- A brief period of immobilization recommended postoperatively.
- Aspiration: higher recurrence rate (50%) than surgical resection but minimal risk so reasonable to attempt.
- Complications of surgery:
- Wound infection, neuroma formation, hypertrophic scar, median nerve, and radial artery damage.
- Dias et al. [1] reported residual pain in 27%, restricted movement in 15% and reduced grip power in 34% of operated cases.
- Arthroscopic resection has the potential advantages of minimizing the surgical scar and permits evaluation of any intra-articular pathologic condition of either midcarpal or radiocarpal joint. Initially thought to be superior to open approach but recent RCT refutes this claim.

9.5 Viva 68

A 65-year-old lady presents with numbness over the thumb, index, and middle finger, with bilateral thenar eminence wasting.

- **What is the likely diagnosis and what are the potential causes?**
- **What is the anatomy of carpal tunnel and what is the pathoanatomy of CTS?**
- **Are you aware of any variations in the motor supply of median nerve in the palm. How is it significant?**
- **How may the diagnosis of this condition be confirmed?**
- **What are the treatment options?**
- **What are the surgical treatment options?**
- **Following a revision of carpal tunnel release due to a previous incomplete release, what percentage of pain relief would the patient experience?**

What is the likely diagnosis and what are the potential causes?
The most likely diagnosis is carpal tunnel syndrome (CTS), caused by compression of the median nerve in the carpal tunnel. It is more common in females, ratio 6:1 and bilateral in 40%.

Most of the cases of CTS are idiopathic (pathological synovium), although there are many recognized predisposing factors:

1. Fluid retention: diabetes, pregnancy, hypothyroidism, renal failure, congestive cardiac failure.
2. Obesity.
3. Inflammatory arthropathies.
4. Space occupying lesions: lipoma, ganglion.
5. Congenital anomalies (persistent median artery, higher origin of lumbrical muscles).
6. Alcoholism.
7. Traumatic (Colle's fracture).

What is the anatomy of carpal tunnel and what is the pathoanatomy of CTS?
It's a fibroosseous tunnel formed by the concavity of the anterior surface of the carpus and roofed over by the flexor retinaculum transverse carpal ligament. It is defined by the scaphoid tubercle and trapezium radially, the hook of hamate and the pisiform ulnarly. It contains 9 flexor tendons, FDS—lies on the profundus tendons arranged 2by2 (middle and ring lie superficial to index and little). Remember, 34 (third and fourth) over 25 (second and fifth). FDP—all together on a deeper plane (lie side by side on the floor of the carpal tunnel).

- The palmar cutaneous branch of the median nerve arises on the radial side of the forearm immediately proximal to the wrist crease, it usually runs over the carpal tunnel to supply the skin on the thenar eminence.

- Synovial biopsy from the carpal canal from idiopathic CTS shows fibrous tissue and variable oedema with scattered lymphocytes.

Are you aware of any variations in the motor supply of median nerve in the palm: how is it significant?
There are three main variations to the motor branch in the palm:

- **Extraligamentous branch** (50%) arises from the volar radial aspect of the median nerve distal to the transverse carpal ligament and recurrent to the thenar muscles.
- **Subligamentous branch** (30%) arises from the anterior surface of the nerve within the carpal tunnel, emerging distal to the flexor retinaculum and recurrent to the thenar muscles.
- **Transligamentous branch** (20%) arises from the anterior surface of the nerve within the carpal tunnel and pierces the flexor retinaculum. This is prone for iatrogenic injury especially in percutaneous/arthroscopic release.

How may the diagnosis of this condition be confirmed?
The diagnosis of CTS is usually made on clinical grounds:

- Pain and paraesthesia over the median nerve distribution area.
- Night pain.
- Thenar atrophy.
- Simian thumb (Ape-hand deformity): the thumb is lying in the plane of the palm (flexion and adduction) due to loss of opponens pollicis muscle function.
- Provocative tests (Tinel's, Phalen, Reverse Phalen's, Durkin's tests). Durkan's test is the most sensitive test to diagnose carpal tunnels syndrome.
- If the diagnosis is in doubt, NCS is indicated. General parameters for NCS diagnosis of carpal tunnel syndrome include a distal motor latency of >4.5 ms, and distal sensory latency of >3.5 ms.
- EMG test the electrical activity of individual muscle fibres and motor units, potential pathologic findings:
  - Increased insertional activity.
  - Sharp waves.
  - Fibrillations.
  - Fasciculations.

What are the treatment options?
Treatment may be non-surgical or surgical. Conservative options include:

- NSAIDs.
- Splintage (night semi-extension splints).
- Activity modification.
- Steroid injection (controversial). Failure to improve after injection is poor prognosis for surgery.

What are the surgical treatment options?

- Surgical treatment is indicated after failure of conservative treatment.
- Surgery should be undertaken prior to development of permanent motor or sensory deficit.
- Surgery release is normally undertaken through an open or endoscopic approach.

Open Approach Technique

- Incision: longitudinal distal to the distal wrist crease and just ulnar to palmaris longus, in line with the radial border of the ring finger and proximal to Kaplan's cardinal line to avoid injury to deep palmar arch (Fig. 9.1).
- Incise the superficial palmar fascia.
- Use self-retainer to clear palmar fat exposing flexor retinaculum.
- Underneath transverse carpal ligament and insert Mcdonald elevator gently. Divide the ligament over the Mcdonald.
- Cut transverse ligament ulnar to avoid cutting recurrent motor branch.
- Decompress proximally up to proximal flexor crease and distally until fully released.
- Neurolysis—no difference in outcome (perform if nerve adherent to adjacent structures).
- Palmar cutaneous branch is at risk if injured repair should not be attempted. It can cause painful neuroma.
- Complications: incomplete release of the transverse carpal ligament.

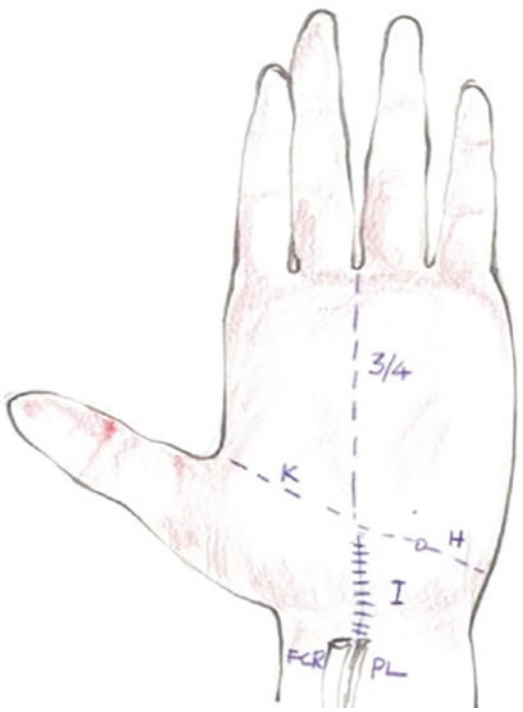


Fig. 9.1 Kaplan's cardinal line

Endoscopic

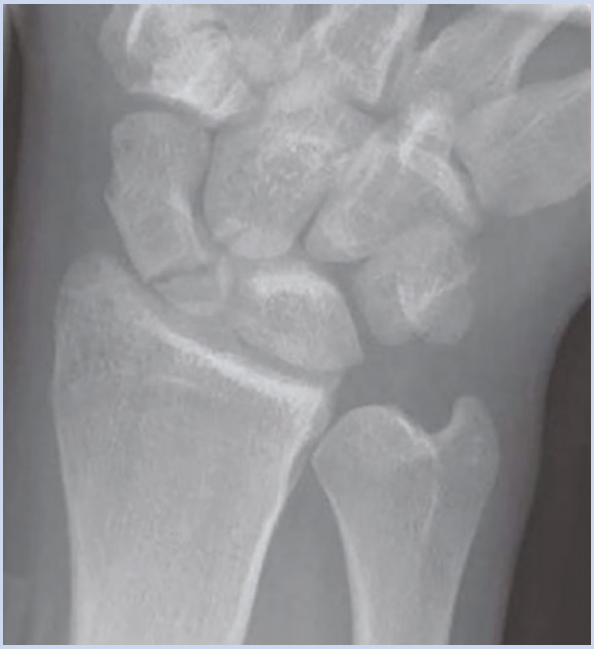
- Introduced to reduce the incidence of pillar pain but this has not been demonstrated. Use either the Agee (one incision) or Chow (two incisions) technique.
- Complications include incomplete release of the transverse carpal ligament, ulnar nerve injury and laceration of the superficial arch.

Kaplan described an anatomical guideline for locating the recurrent motor branch of the median nerve. Kaplan's line is a line drawn from the distal border of the abducted thumb to the hook of hamate. The recurrent motor branch of the median nerve entering the thenar muscle mass is estimated by the intersection of Kaplan's line and a vertical line from the radial border of the middle finger. The hook of hamate lies at the intersection of the ulnar border of the ring finger and Kaplan's line. The deep palmar arch lies 7 mm distal to Kaplan's cardinal line. The superficial palmar arch lies 15 cm distally, deep to the distal transverse palmar crease.

Following a revision of carpal tunnel release due to a previous incomplete release, what percentage of pain relief would the patient experience?

- 25% of patients will have no relief.
- 25% of patients will have complete relief.
- 50% of patients will have partial relief of symptoms.

9.6 Viva 69



- **What can you see?**
- **What are the anatomical and biomechanical considerations?**
- **What is the mechanism of injury?**
- **What are the clinical findings?**
- **What are the recommended radiographic views?**
- **What is the best imaging for diagnosis of occult fracture?**
- **Are you aware of any classification system?**
- **How would you manage this patient with isolated scaphoid fracture?**
- **What are the indications for operative fixation?**
- **What surgical approaches are commonly used?**

What can you see?

This is P/A radiograph of a wrist that demonstrates proximal pole scaphoid fracture. There is minimal displacement. I do not see any sclerosis. I would like to get lateral and oblique views to assess the fracture pattern and displacement in more detail and assess the other carpal bones. Fractures of the proximal third of the scaphoid account for 20% of scaphoid fractures, those of the middle portion account for 60%, and fractures of the distal part make up the remaining 20%.

What are the anatomical and biomechanical considerations?

- The scaphoid may be divided into proximal, middle (termed the waist), and distal third.
- 80% of the scaphoid surface is cartilage, thus leaving a small area for arterial blood supply to enter the bone. The major blood supply is dorsal carpal branch (branch of the radial artery), enters scaphoid in a nonarticular ridge on the dorsal surface, and supplies 80% of scaphoid via retrograde blood flow.
- Minor blood supply from superficial palmar branch (branch of volar radial artery) enters distal tubercle and supplies 20% of scaphoid. A proximal 1/5 fracture has a non-union rate of 80–100% when treated non-operatively.
- The scaphoid can be palpated on the dorsal surface in the anatomical snuffbox. The distal tubercle can also be palpated on the palmar surface at the base of the thenar eminence.
- The scaphoid flexes with wrist flexion and radial deviation and it extends during wrist extension and ulnar deviation, these factors make immobilization of scaphoid fractures difficult, with scaphoid fracture, distal scaphoid tends to flex, and proximal scaphoid extends with the proximal carpal row, because of this, angulation occurs at fracture site, which gradually leads to humpback deformity.
- They are rare in the elderly and in children. (Scaphoid fracture is uncommon in children because the physis of the distal radius usually fails first, resulting in Salter type I or II fractures of the distal radius. Similarly, in elderly patients, the distal radial metaphysis usually fails before the scaphoid can fracture.) In children, the fracture occurs within the distal pole of the scaphoid because it ossifies before the proximal pole.

What is the mechanism of injury?
The most common mechanism of injury is axial load across hyper-extended and radially deviated wrist.

What are the clinical findings?
Wrist effusion—Anatomic snuffbox tenderness dorsally—Scaphoid tubercle tenderness volarly—Reduced range of motion (but not dramatically)—Pain at extremes of motion, Pronation followed by ulnar deviation will cause pain.

What are the recommended radiographic views?
Four standard radiographs:

1. PA in ulnar deviation (extends scaphoid). The scaphoid view is a PA picture taken with the wrist in 30° extension, full pronation, and ulnar deviation 20°. This view shows the scaphoid in its longitudinal axis without superimposed shadows from the distal radius, as wrist rotates from neutral to ulnar deviation, proximal row dorsiflexes and X-ray profile of the scaphoid appears longer, and in radial deviation, proximal carpal row volar flexes and scaphoid appears foreshortened.
2. Lateral.
3. Two oblique views. Pronated oblique (45° pronated oblique), and ulnar deviated oblique (also described as 45° supinated oblique).

What is the best imaging for diagnosis of occult fracture?
- MRI is the most sensitive for diagnosis of occult fracture <24 h.
- Bone scan is effective at 72 h.
- CT with 1 mm cuts is less effective than MRI and bone scan to diagnosis occult fracture, but can be used to evaluate location of fracture, size of fragments, extent of collapse, and progression of non-union or union after surgery.

Are you aware of any classification system?
Mayo classification based on location of fracture line:

Type I Tubercle.
Type II Distal articular fracture.
Type III Distal third.
Type IV Middle third.
Type V Proximal third 20%. The more proximally located the fracture plane is, the greater the risk of delayed union, non-union, and AVN.

Russe classification

According to the plane of fracture with respect to the long axis of the scaphoid, predicts the tendency of the fracture to heal.

(A) Horizontal oblique.
(B) Transverse.
(C) Vertical oblique.

Increased shear forces in vertical oblique fractures may prolong the time for fracture healing.

Herbert classification: It is based on the stability of the fracture.

Type A. Stable.
Type B Unstable fractures are those with a displacement of more than 1 mm or an angulation of more than 15° between the fragments. Additional fractures, trans-scaphoid-perilunate dislocations, multi-fragment fractures and proximal pole fractures are also classified as unstable.
Type C. Delayed union.
Type D. Non-union.

How would you manage this patient with isolated scaphoid fracture?
I would like to see full scaphoid views to assess the fracture and its displacement. This fracture is minimally displaced and being proximal pole, I would discuss with patient regarding operative versus non-operative means. Non-union rates are higher in proximal pole fractures. I would prefer screw fixation through dorsal approach for this fracture.

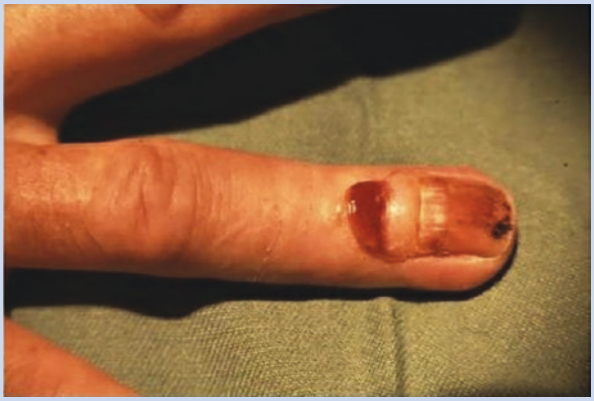
What are the indications for operative fixation?
- Proximal pole fractures.
- Displacement >1 mm.
- 15° scaphoid humpback deformity
- Intrascaphoid angle of >35°.
- Scapholunate angle >60°.
- Scaphoid fractures associated with perilunate dislocation.
- Comminuted fractures.
- Unstable vertical or oblique fractures.
- In non-displaced waist fracture, to allow decreased time to union, faster return to work/sport, and similar total costs compared to casting (studies have shown better early outcome scores, grip strength, and range of motion (ROM) with fixation but no difference after 12–16 weeks).

What surgical approaches are commonly used?
- Undisplaced or minimally displaced fractures can be fixed percutaneously. Waist and distal fractures are more easily treated through a volar approach as it allows preservation of the dorsal blood supply. Proximal pole fractures are more readily accessed via a dorsal approach.
- **Volar approach**: Surface landmarks are the scaphoid tuberosity and FCR tendon. Skin incision is longitudinal along the radial border of FCR curving radially at the distal wrist crease. Divide the superficial branch of the radial artery and

dissect through the bed of the FCR tendon sheath. Distally the thenar muscles are elevated to allow exposure. Incise and reflect the capsule and the radioscaphocapitate and radioscapholunate ligaments. Screws are placed distal to proximal, 45° to the horizontal and 45° to the long axis of the forearm. A piece of trapezium may need to be excised to gain access to the distal pole of the scaphoid.

- **Dorsal approach**: Use for proximal pole fractures as it provides access with the wrist hyperflexed. Care is needed to avoid damage to the dorsal blood supply. The incision is distal to Lister's tubercle. The approach is between the third and fourth extensor compartments (EPL and EDC). EPL is reflected radially while EDC taken ulnarly. Flex the wrist 90° to expose the proximal pole and reduce the fracture with flexion and traction. The entry point for the guidewire is just radial to the scapholunate ligament and aims along the thumb metacarpal.

9.7 Viva 70



- **What is the diagnosis?**
- **Describe the different parts of injured structure?**
- **How would you manage this patient?**
- **What would you explain to the patient?**

What is the diagnosis?

This is a clinical photograph of a nail bed injury; it may seem innocuous but must be treated with care to avoid later nail deformities.

Describe the different parts of injured structure?

- Perionychium: the entire area that includes the nail, nailbed, and the surrounding skin**.**
- Paronychium: the lateral nail fold.

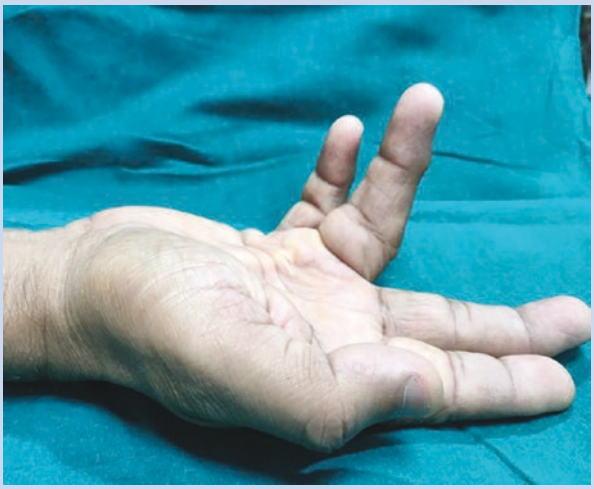
- Eponychium: the dorsal nail fold, proximal to nail plate.
- Hyponychium: the skin immediately distal and palmar to the nail, at the junction of the sterile matrix and fingertip.
- Lacuna: the white portion of the proximal nail.
- Sterile matrix: the soft tissue deep to the nail, distal to the lacuna.
- Germinal matrix: the soft tissue that deep to the nail proximal to the sterile matrix. It is responsible for the development of the nail (the periosteum of the distal phalanx lies immediately volar to the sterile and germinal matrices).

How would you manage this patient?
- I would take a relevant history, including handedness; occupation; mechanism of injury; and comorbidities.
- Examine for a visible nail bed laceration.
- I will provide tetanus prophylaxis (if indicated) and antiseptic (betadine) dressing.
- I would obtain radiographs to exclude an underlying fracture.
- Discuss with the patient the possibility of long-term nail deformity.

Definitive Treatment
- Digital block of the digit with local anaesthetic (without adrenaline) and digital tourniquet.
- Remove the nail plate carefully.
- Inspect the nail bed and wash thoroughly.
- Copious lavage of any underlying fracture.
- Ensure there is no soft tissue or nail bed entrapment in the fracture site, as non-union may result.
- Reduce fracture if necessary and stabilize with K-wire (small tuft fractures do not require treatment, but diaphyseal fractures can cause deformity with angulation and malalignment).
- Ensure there is no flexor or extensor tendon avulsion.
- Repair nail bed with a 6–0 absorbable suture.
- Wash and replace nail plate. Figure-of-eight stitch (or equivalent) to hold the nail plate in place.

What would you explain to the patient?
I would explain that the nail plate will fall off and be gradually replaced by a new one, which may initially appear disfigured. There is a risk of some long-term nail deformity and discomfort in the region of the nail bed and some distal interphalangeal joint stiffness.

9.8 Viva 71



- **Describe the image.**
- **How can you define this condition?**
- **What is the aetiology of this condition?**
- **Describe the anatomical structures affected in this condition?**
- **What histopathological changes are seen in this condition?**
- **What is Dupuytren's diathesis?**
- **Are you aware of any classification system used for hand contracture?**
- **How would you treat this patient?**
- **What are the factors increasing the risk of recurrence of surgically treated conditions?**

Describe the image

This is a clinical photograph that shows flexion of the little finger at the level of PIP and DIP joints, although it is difficult to comment without palpation, there appear to be cords present at the palm extending to little finger, ring finger. There are no obvious surgical scars. The appearance is suggestive of Dupuytren's disease.

How can you define this condition?

Dupuytren's disease is a benign proliferative disorder of the palmar and digital fascia forming nodules and cords with secondary flexion contracture of the finger.

The male to female reported ratios vary from between 4:1 and 10:1, over 60 years. It occurs in fifth to seventh decades. Earlier onset more aggressive disease.

What is the aetiology of this condition?
The exact aetiology is unknown. There are some theories:

1. Genetic link is autosomal dominant with variable penetrance.
2. Neoplastic.
3. Traumatic.
4. Inflammatory.

Describe the anatomical structures affected in this condition?
The structures that are affected in this condition are:

- **Palmar:**
  - Pretendinous bands (overlying the flexors in the palm of the hand).
- **Palmo-digital:**
  - Spiral bands: emerge from the either side of the pretendinous band along both sides of the flexor sheath passing under the neurovascular bundle.
- **Digital:**
  - Lateral digital sheet: formed by merging fibres from the spiral band, running lateral to the neurovascular bundle.
  - Natatory ligament: crosses the palm just proximal to the distal palmar crease (DPC).
  - Grayson's (G for ground) ligament runs along fingers **palmar** to neurovascular bundle.
  - Cleland's (C for ceiling) ligaments which pass from side of phalanges to the skin, **dorsal** to the neurovascular bundle are spared in Dupuytren's disease.

The diseased cords are:

- **Spiral cord**, composed of pretendinous band, spiral band, lateral digital sheath and Grayson's ligament. It displaces the neurovascular bundle superficially and at the midline where it is at risk of damage during the distal palmar dissection. Most commonly seen in the little finger. Results in PIPJ contracture.
- **Central cord:** results from involvement of the pretendinous band. MCPJ contracture.
- **Retrovascular cord:** DIP joint contracture.
- **Natatory cord:** webspace contracture.
- **Abductor digiti minimi cord:** little finger contracture.

What histopathological changes are seen in this condition?
The disease is staged by Luck into three phases:

1. Proliferative.
2. Involutional.
3. Residual.

- The cell type seen in stages 1 and 2 is the myofibroblast which is replaced by fibrocytes in stage 3.
- There is a large amount of type III collagen.

What is Dupuytren's diathesis?
It is a more aggressive form of the disease characterized by:

- Young age of onset <40 years.
- Rapid progression.
- Bilateral disease.
- Involvement of more radial digits.
- High recurrence rat.
- Involvement of extra-palmar sites: penis (Peyronie's disease), plantar fibromatosis (Ledderhose disease) Garrod's pads (Calluses on the dorsal aspect of the MCP and IP joints).

Are you aware of any classification system used for hand contracture?
Dupuytren's Classification (Woodruff 1998)
- Stage I: Palmar disease. No contracture → surveillance/discharge.
- Stage II: One finger. MPJ contracture → surgery +++.
- Stage III: One finger. MPJ + PIPJ contracture → difficult surgery.
- Stage IV: More than one finger → difficult surgery.
- Stage V: Severe (finger-in-palm) deformity → consider amputation.

TUBIANA
- Stage 0—Nodule but no contracture.
- Stage I—<45° flexion.
- Stage II—45–90°.
- Stage III—90–135°.
- Stage IV—>135°.

How would you treat this patient?
I would like to take full history from the patient asking about:

- Age—Hand dominance—occupation.
- The main complaints. (Pain is not always a feature of Dupuytren's but may be present in the initial presentation of nodules due to local pressure effects.)
- Rate of progression.
- ADLs: Difficulty washing face, combing hair, putting the hand in a pocket or a glove. Decreased manual dexterity at work.
- Involvement of other sites. Ectopic lesions. (penile and foot involvement).
- What have been done for the patient?
- Specific risk factors and associated abnormalities: Increasing Age, Male Sex, Diabetes, Epilepsy, HIV, COPD, Alcoholic/smoker (controversial), Family history, Past surgery, Patient's expectations.

Physical Examination

- Any previous scars suggesting previous surgery.
- Palmar nodules and digits involved.
- Skin pits.
- Garrod's pads over the dorsal PIP join. Ledderhose disease (nodules in the sole of the feet).
- Hueston's tabletop test: ask the patient to place his palm on the table, to look for MCP and PIP joint contracture (inability to do this test confirms the diagnosis and it is a relative indication for surgery).
- Functional assessment (power: squeeze my fingers—Span; hold a cup—Tripod: hold a pen—Key pinch: pulp to pulp—precision: tip to tip, pick up a coin).
- PIP angle (measure with MCPJ fully flexed). The combination of PIP joint in a fixed flexion deformity with the MCP joint in flexion signifies a severe deformity and a poor prognosis. Measure MCPJ angle with hyperflexion of PIP joint and measure PIPJ angle with flexion of MCPJ.
- Neurovascular assessment: it is very important to test for sensation distally for any site of proposed surgery (1.5% risk of digital nerve injury for the first time surgery, up to 20% for recurrent surgery). Assess the circulation with capillary refill and digital Allen's test.

Non-operative Treatment

- Patient needs counselling, that conservative treatment may be attempted but condition will not spontaneously resolve and there is chance of progression and greater functional impairment:
- Occupational therapy (range of motion exercises).
- Injection. Collagenase Clostridium injection into Dupuytren cords to produce lysis and rupture, followed by MUA after 24 h. Can be performed in patient with early symptoms, MCPJ <20°. Although early results have been encouraging, definitive word on the efficacy of this method is still pending. CORDLESS trial showed similar recurrence rates to surgery (47%), it is safe and effective method in certain groups.
- If findings are mild without significant difficulty with daily activities, it is reasonable to follow the patient at repeated intervals to check for progression.

Operative Treatment

- Patient needs counselling that surgery is not curative, and disease can recur, and it is unlikely to achieve full correction of the deformity.
- Relative indications include:
  - Abnormal Hueston's table test.
  - MCP contracture >30°.
  - PIP contracture >15°.

Surgical Options

1. Needle fasciotomy: outpatient procedure. Indicated in patients with poor medical condition or for a well-defined pretendinous palmar cord causing an MCP joint contracture. High recurrence rate.
2. Limited fasciectomy: removal of all diseased tissue. Most widely used surgical treatment overlying skin is preserved.
3. Radical fasciectomy: release and/or excision of all palmar fascia including portions that do not appear involved in the disease process. Decreased utilization due to high complications rate.
4. Dermatofasciectomy with full-thickness skin graft coverage. Indicated in recurrent disease or
5. Dupuytren's diathesis.
6. Amputation of the involved digits.

In this patient if the symptoms are mild and not interfering with his daily routine, I would discuss with the patient regarding the conservative options and the need for regular follow-up.

If the symptoms are more severe and interfere with daily life activities with a positive tabletop test I would discuss surgical options after thorough counselling of the patient. My choice of procedure would be partial fasciectomy in this patient.

Surgical Approaches

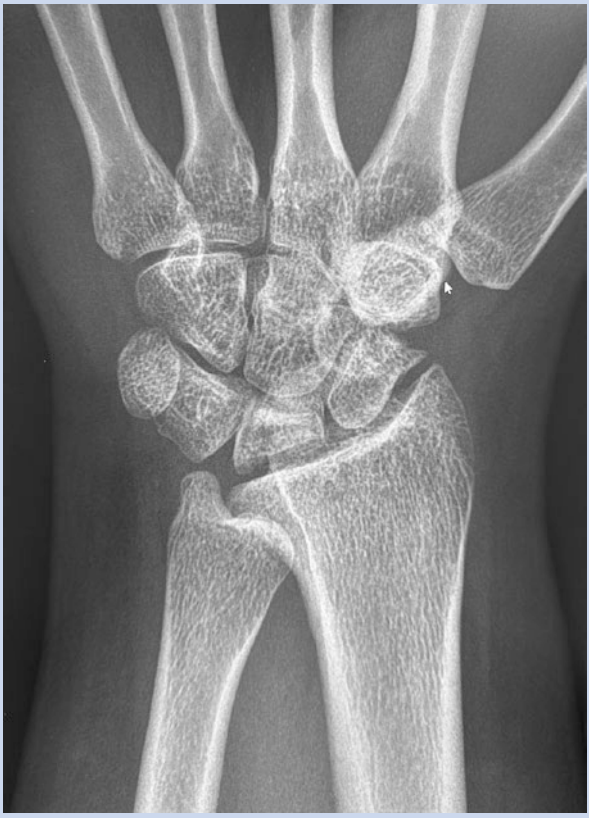
- Brunner zigzag incisions. Multiple V-Y incisions with the apices in palmar skin creases with angles >60°.
- Straight incision with Z plasty to lengthen the skin.
- McCash open technique. Transverse palmar incision with digital extension, either Bruner or Z plasty, along the digit. The wound provides excellent access to the diseased tissue and is left open at the end of the procedure. Good technique in elderly patients, as it causes less oedema and haematoma formation, it is useful if you are short of skin, and it causes less pain and stiffness. However, it takes a relatively long time to heal (6–8 weeks).

What are the factors increasing the risk of recurrence of surgically treated conditions?

- The patient is below the age of 50 years old.
- Positive family history.
- Both hands are affected.
- Ectopic lesions (Peyronie's disease, Knuckle pads and Ledderhose disease).

9.9 Viva 72

A 39-year-old male presents with longstanding wrist pain.



- **Describe the radiograph.**
- **What is the aetiology of this condition?**
- **How do you classify Kienbock's disease?**
- **What are the treatment options for this condition?**
- **How would you manage this case?**
- **How can you measure the ulnar variance and carpal height?**

Describe the radiograph
This is a plain radiograph of left wrist, shows increased density of the lunate with partial collapse, with no obvious arthritic changes in the surrounding joints. There is appearance of negative ulna variance in DRUJ. Features are consistent with Kienbock's disease, which is avascular necrosis of lunate. I would like to see lateral view to assess the scapholunate and radio lunate angles.

What is the aetiology of this condition?
The aetiology of Kienbock's disease is unknown. Several theories have been put forward and thought to be due to multiple factors.

Biomechanical Factors
- Ulnar negative variance. Leads to increased radial-lunate contact stress.
- Repetitive trauma or single traumatic incident.

Lifestyle Factors
- Hard manual workers (increases shear and compression forces across lunate).

Anatomic Factors
- Vascular blood supply to lunate. I—pattern arterial blood supply—high risk. There are three variations (Y 60%-I 30%-X 10%).

How do you classify Kienbock's disease?

Lichtman Classification (Radiological)
- **Stage I**: normal X-ray. Findings seen on MRI (decreased T1 signal intensity).
- **Stage II**: sclerosis of lunate.
- **Stage IIIA**: fragmentation and collapse of the lunate without fixed scaphoid rotation (carpal height is preserved).
- **Stage IIIB**: fragmentation and collapse of the lunate with fixed scaphoid rotation (there is loss of carpal height).
- **Stage IV**: Degenerated adjacent intercarpal joints.

What are the treatment options for this condition?
This depends on the patient's age, symptoms, functional demand, the stage of the disease, ulnar variance, the carpal height, and the degenerative changes.

- **Stage I**: immobilization + analgesia. If failed, surgical treatment same as stage II and IIIA.
- **Stage II**: in cases of ulnar −ve variance → joint levelling procedures. Radial shortening or ulnar lengthening, more evidence on radial shortening (↓ risk of non-union and ↓ need for bone grafting). In cases of neutral variance → (1) radial wedge osteotomy. (2) Distal radius core decompression to create a local vascular

healing response to facilitate recovery before collapse. (3) Vascularized bone graft (transfer of pisiform—transfer of distal radius on a vascularized pedicle of pronator quadratus) early results promising, but long-term data lacking.

- **Stage IIIA**: same as stage II.
- **Stage IIIB**:
  - STT fusion.
  - Proximal row carpectomy. Some studies have shown superior results of STT fusion over PRC for stage IIIB disease.
- **Stage IV**: wrist fusion—total wrist arthroplasty (long-term results are not available).

How would you manage this case?

My concern with Kienböck's disease is that radiographic appearances do not always correlate well with clinical symptoms and the natural history of this condition is still uncertain and it doesn't always progress from stage I to stage IV. Surgery has not been shown conclusively to prevent the progression of the disease, so I would need to fully assess this patient with regard to the symptoms, functional disability, expectations from treatment and careful counselling of the patient.

I would confirm these were length films for ulnar variance, I would like to request for other views +/− CT which is the most sensitive tool to determine the early lunate collapse in Kienbock's disease.

I would start with conservative treatment → rest, splintage and analgesia. Surgery is indicated if symptoms do not settle. In this case with partial collapse, if no fixed scaphoid rotation on further evaluation and no obvious degenerative changes and a negative ulnar variance, I would go for radial shortening osteotomy. I would counsel the patient preoperatively for any additional procedures which could be a salvage procedure like proximal row carpectomy or a limited/total wrist fusion.

How can you measure the ulnar variance and carpal height?

1. **Ulnar variance** (Fig. 9.2).

   Wrist PA view with the shoulder flexed 90°, elbow 90° and the wrist in neutral position (pronation leads to ↑ variance—supination leads to ↓ variance).

   Measure the distance between two lines, one line is drawn through the articular surface of the ulna, second line through the articular surface (lunate fossa) of the distal radius. Compare it with the contralateral side.

   The position of ulnar styloid will help determine which way film was obtained.
   - In standard PA view, ulnar styloid is peripheral.
   - In standard AP view, ulnar styloid points centrally.
2. **Carpal height** (Fig. 9.3).

   By measuring the distance between the distal articular surface of the capitate and the distal articular surface of the radius divided by the length of the third metacarpal.

   The average ratio ranges between 0.45 and 0.6.

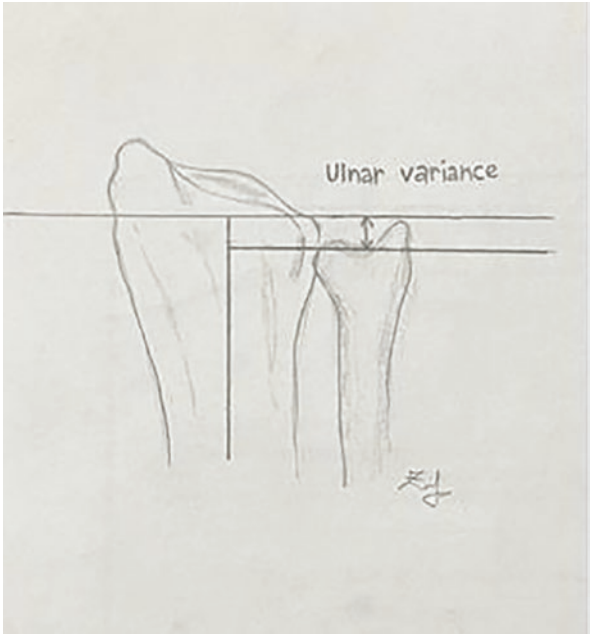


Fig. 9.2 Ulnar Variance

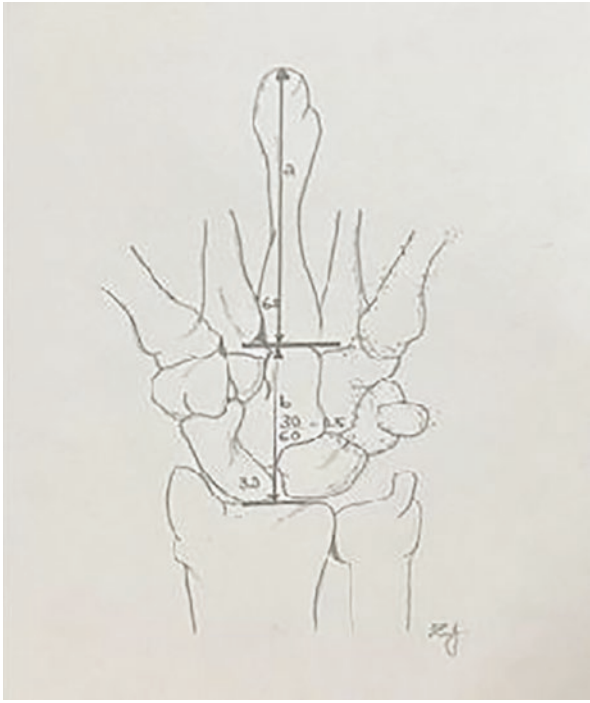
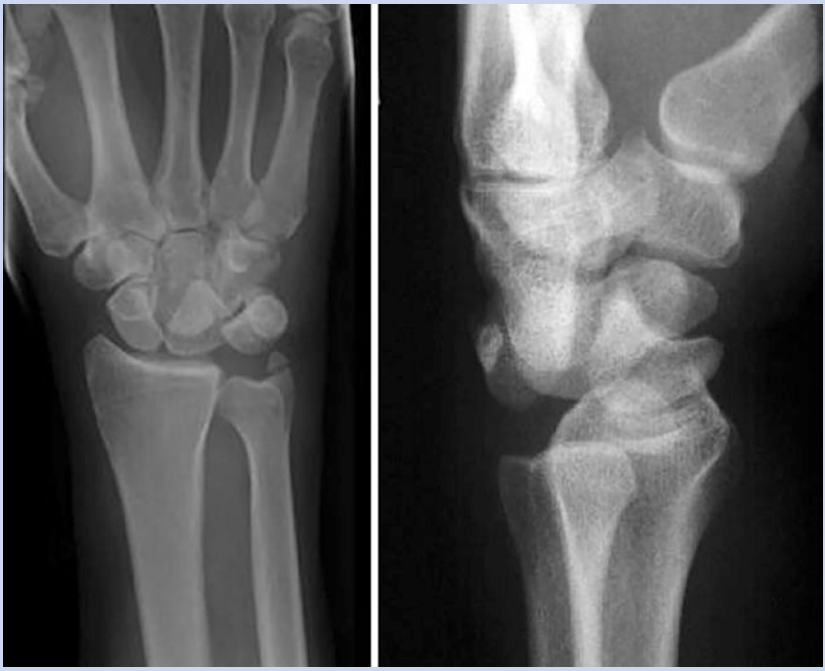


Fig. 9.3 Carpal Height

9.10 Viva 73

A 35-year-old professional football player complains of severe wrist pain after making a tackle. He reports paraesthesia in his thumb and index finger.



- **What can you see?**
- **What is Gilula's arc?**
- **What is the difference between perilunate and lunate dislocation?**
- **What are the greater and lesser arc injuries?**
- **What is the mechanism of injury?**
- **What classification system do you know of for such an injury?**
- **How will you manage this patient?**

What can you see?
These are P/A and lateral radiographs of the wrist shows:

- Break in Gilula's arc. Luno-capitate overlap, Triangular shape of lunate (piece of pie sign) and Ulnar styloid fracture.
- Loss of the collinearity of the radius, lunate and capitate.
- Features are suggestive of peri lunate dislocation.

What is Gilula's arc?
- They are three arcs drawn on a PA radiograph of the wrist used to assess the alignment of the carpus.
- First arc is running along the proximal convexity of the scaphoid, lunate and triquetrum.
- Second arc is running along the distal concavity of the scaphoid, lunate and triquetrum.
- Third arc is running along the proximal curvature of the capitate and hamate.

What is the difference between perilunate and lunate dislocation?
- Perilunate dislocation: lunate stays in position while carpus dislocates.
- Lunate dislocation: lunate forced volar or dorsal while carpus remains aligned.

What are the greater and lesser arc injuries?
- Greater arc—injuries through carpal bones (scaphoid, capitate, hamate, triquetral, pisiform).
- Lesser arc—purely ligamentous (scapholunate, capitolunate, lunotriquetral ligaments).

What is the mechanism of injury?
Forced wrist dorsiflexion with ulnar deviation. The injury begins at the radial side and progresses towards the ulnar side through the midcarpal space.

What classification system do you know of for such an injury?

Mayfield classification	
Stage 1	Scapholunate dissociation
Stage 2	+ lunocapitate disruption
Stage 3	+ lunotriquetral disruption, perilunate
Stage 4	Lunate dislocated

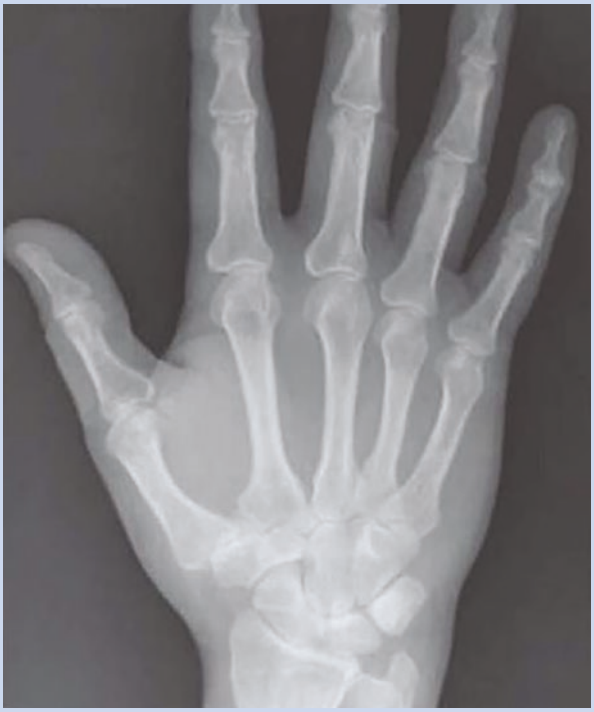
- Stage IV associated with spilled teacup sign on X-ray.

How will you manage this patient?
- Assuming this is an isolated injury, I will assess the patient clinically and look for neurovascular impairment particularly median nerve. This will need to be reduced as an emergency procedure under either conscious sedation or anaesthesia. Emergent closed reduction leads to ↓ risk of median nerve damage and ↓ risk of cartilage damage. It is reduced by hyperdorsiflexion of the wrist, and then apply pressure on the lunate to reduce and flex the wrist. If failed → open reduction in theatre. I will take the patient the same night to the theatre in the presence of median nerve compression symptoms. If no median nerve compression symptoms, the patient can be left to the next daylight hours with close observation of neurological function (median nerve).

- In theatre, I will try another attempt of closed reduction by putting the affected extremity on Chinese finger trap for 5 min followed by hyperdorsiflexion of wrist, and then apply pressure on the lunate to reduce and flex the wrist.
- If failed, then open reduction through a dorsal approach + volar carpal tunnel approach for decompression or an extended carpal tunnel approach in case of lunate dislocation, reducing the lunate by pushing it into the space of porier.
- I would stabilize the reduction temporarily with k-wires, followed by closure and splint immobilization.
- Ligament repair should be protected with 2K-wire fixation. Spanning ex.fix if remains unstable.
- Thumb spica for at least 6 weeks.
- Proximal row carpectomy is indicated in missed injuries >8 weeks.

9.11 Viva 74

A 50-year-old female patient presents with right hand radial-sided pain.



- **Describe radiograph.**
- **What is the differential diagnosis of radial-sided wrist pain?**
- **Do you know any X-ray view which may better show the thumb CMC joint?**
- **What is the pathoanatomy of this condition?**
- **Do you know any classification system for this?**
- **How would you manage this patient?**

Describe this radiograph

- This is a P/A radiograph of the hand. The most obvious abnormality is complete loss of joint space of the thumb carpometacarpal (CMC) joint and subchondral sclerosis**.**
- Features consistent with basilar thumb arthritis, which is the second most common OA of the hand (DIP > thumb CMC > PIP > MCP).

What is the differential diagnosis of radial-sided wrist pain?

- De Quervain's disease.
- Intersection syndrome.
- Wartenberg syndrome.
- Basilar thumb arthritis.
- STT osteoarthritis.
- Radio-scaphoid OA.
- Scaphoid fracture, Non-union scapholunate disruption.

Do you know any X-ray view which may better show the thumb CMC joint?

- A Robert's view—this is a true AP view of the thumb CMC joint, and is taken with the elbow extended, the forearm fully pronated and the thumb abducted and hyper-pronated.
- Lateral view should be obtained as well to exclude DISI deformity which is a contraindication to perform trapeziectomy.
- Check for MCP joint arthritis (contraindication for CMC fusion).
- Check for STT arthritis.

What is the pathoanatomy of this condition?

Theorized to be due to attenuation of anterior oblique ligament **(Beak ligament**) leading to instability subluxation, and arthritis of CMC joint.

Do you know any classification system for this?

Eaton and littler classification of basilar thumb arthritis	
Stage 1	Slight joint space widening (pre-arthritis)
Stage 2	Slight narrowing of CMC joint with sclerosis <2 mm
Stage 3	Marked narrowing of CMC joint with osteophytes >2 mm
Stage 4	Pantrapezial arthritis (STT involved)

How would you manage this patient?
History

- Constant dull pain at the base of the thumb aggravated by activities like (undoing screw, doing up buttons, writing).
- Pain at the MCP joint from compensatory hyperextension.
- Night pain.
- Features of CTS (50%).
- Difficulties with ADLs (pinching—grasping—dropping objects).

Physical Examination

Look
- Squaring-off of the base of the thumb (shoulder sign which is a prominence at the base of the thumb due to dorsal subluxation of the thumb's metacarpal base).
- Adduction contracture of the first webspace.
- Thenar muscle wasting.
- Compensatory hyperextension of the MCP joint to compensate for the loss of motion due to CMC arthritis and stiffness and the adduction contracture leading to Z deformity.
- Secondary flexion deformity of IP joint.
- Look for trigger fingers and signs of CTS.

Feel
- Tenderness localized to the base of the thumb, Swelling, Crepitus.
- Move and special tests:
- Grind test—pain with axial loading of the thumb metacarpal and rotation of the CMC joint, considered positive if pain disappears with repeat test with distraction of the joint.
- Crank test—pain with axial loading of the thumb with passive flexion and extension.

In this case I would start with non-operative measures including NSAIDS, splinting, occupational therapy.

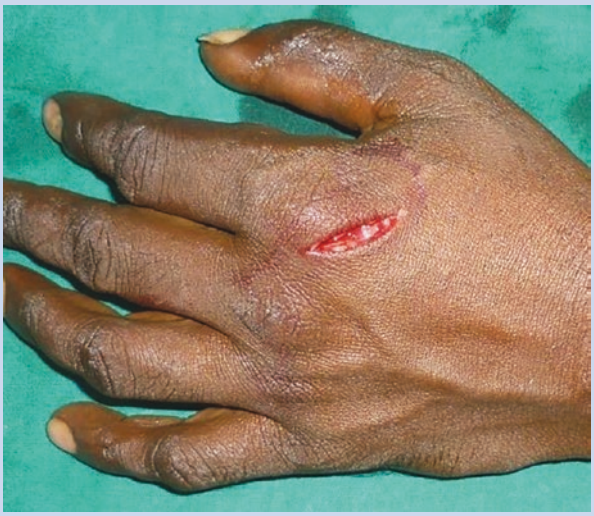
I will consider intraarticular injection if noninvasive measures fail. If the patient still is symptomatic then I would discuss the surgical options. There are numerous surgical options. The choice depends on:

- The stage of the disease. Whether there is isolated CMC joint disease or pantrapezial disease.
- The presence of MCP joint hyperextension deformity.
- The patient's activity level.
- Surgeon experience.
- (Dias—J Hand Surgery, 2004): At 3 months and 1 year—all procedures had same improvement in pain and grip.
- There is no hard evidence that one technique is superior to another. Dias—J Hand Surgery, 2004.

My preferred option would be Trapezectomy + LRTI.

9.12 Viva 75

A 28-year-old man presented 6 h after sustaining an injury to his left hand. He appeared hesitant to share his medical history but eventually acknowledged being involved in a fight.



- **Describe what you see.**
- **How would you evaluate the patient in the emergency department?**
- **Could you elaborate on the operative management steps for this injury?**

Describe what you see

This clinical photograph shows a wound over the metacarpophalangeal joint of the index finger of the left hand. It raises suspicion of a 'fight bite' wound over the MCPJ caused by a penetrating tooth injury. The wound may extend down to the underlying joint, increasing the risk of septic arthritis. There is a high likelihood of an extensor tendon injury and potentially accompanying osteochondral injuries or fractures.

How would you evaluate the patient in the emergency department?

- I would take a thorough history with high suspicion of fight bite injury. I would enquire about handedness, occupation, past medical and surgical history, and immunization status.
- Look for signs of sepsis in general examination. (Fever, tachycardia, hypotension.)
- On local examination, I would assess the wound for signs of infection, erythema, and swelling. I would check for tendon injury by tenodesis effect (with passive wrist flexion and extension).
- X-rays to assess for underlying fractures and retained tooth fragments.
- I would enquire about Tetanus immunization status and give prophylaxis.
- I would start antibiotics after attaining wound swab. Start with broad spectrum group. The infection is typically polymicrobial, but the most common organism cultured from a fight bite is *Staphylococcus aureus*. Gram-negative bacteria should also be covered with the antibiotics as Eikenella corrodens is often implicated in a fight bite.
- I would take photographs of the wound and apply saline-soaked non-adherent sterile dressing.

Could you elaborate on the operative management steps for this injury?

I would take the patient to the operating room for debridement under general anaesthesia, using an arm tourniquet. I would extend the wound after positioning the hand in a clenched fist to align the skin with the tendon and joint. In this position, I would perform a careful exploration, checking for any tendon injuries. I'd examine the underlying tendons and assess joint penetration. If the tendon is ruptured, I would not attempt primary repair but would tag the ends at that time. Deep tissue samples should be collected during the surgery, and I'd initiate intravenous antibiotic therapy.

I would then inspect the joint and irrigate it with a generous amount of normal saline. I would partially close the wound by suturing the wound extensions and apply a sterile, non-adherent dressing. Following surgery, I would advise the patient to keep the arm elevated using a sling while in the ward. I'd reevaluate the wound 48 h post-op in the ward and determine the need for further surgery, including tendon repair.

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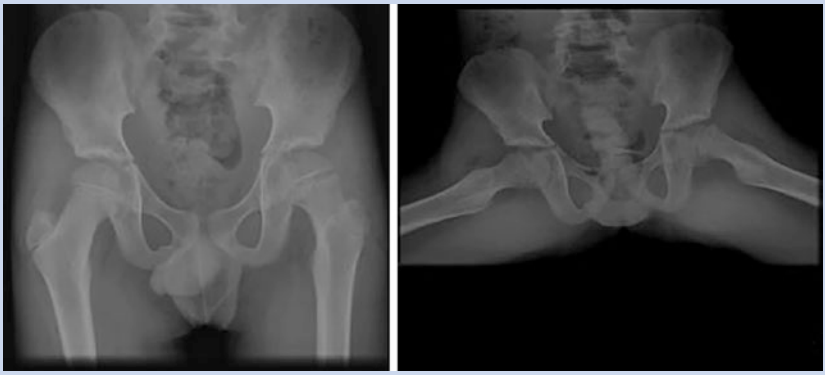
Paediatrics

10

Stanley Jones and Mazhar Fuad

10.1 Viva 76

An obese 14-year-old patient presents to your office. He has been limping for several months, and recently has been unable to walk without significant pain.



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A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_10

- **Describe the radiographs.**
- **Describe the aetiology of this condition.**
- **How would you assess this patient further?**
- **Describe the radiographic features of this condition.**
- **Are you aware of any classification systems for this condition?**
- **How would you manage this patient?**
- **Would you pin the contralateral non-slipped, asymptomatic side?**

Describe the radiographs

These are A/P and frog-leg lateral views of the pelvis in a skeletally immature child. The A/P view reveals a widened physis on the left hip, while the frog-leg lateral view confirms the diagnosis of slipped capital femoral epiphysis.

Describe the aetiology of this condition

The exact aetiology is unknown, but it is thought to be the result of mechanical insufficiency of the proximal femoral physis to resist load, which can occur due to either physiological loads across an abnormally weak physis or abnormally high loads across a normal physis. It occurs through the hypertrophic zone. Renal SUFE occurs through secondary spongiosa. The femoral neck displaces anteriorly and superiorly relative to the femoral.

How would you assess this patient further?

My detailed history would include:

- Duration of the symptoms.
- Any limp, presence of knee pain (referred pain).
- Ability to bear weight with or without crutches.
- Any history of trauma.
- Past history (endocrine abnormalities, renal osteodystrophy, radiotherapy).

On examination I would assess his ability to weight bear with or without support, Shortening of the limb, Hip ROM, Loss of Internal Rotation, External rotation on flexion of hip.

Describe the radiographic features of this condition

The radiographic features include:

- Widening and Irregularity of the physeal line.
- Trethowan's sign; a line (referred to as Klein's line) drawn on the superior border of the femoral neck on the AP view passes over the head rather than through the head.

- Steel's blanch sign which is a crescent-shape dense area in the metaphysis due to superimposition of the neck and the head.
- Decreased epiphyseal height as the head slipped posteriorly behind the neck.
- Increased distance between the tear drop and the femoral neck metaphysis.
- Remodelling changes of the neck with sclerosis, and callus formation.

Are you aware of any classification systems for this condition?
Loder classification
- Stable SUFE. If the patient is able to bear weight on the involved extremity.
- Unstable SUFE. If the patient is unable to bear weight on the involved extremity with or without crutches.

Temporal based on the duration:

- Acute <3 weeks.
- Chronic >3 weeks. It is the most common type (85%).
- Acute on chronic.

Severity of the slip based on the percentage of the epiphyseal displacement relative to metaphyseal width of femoral neck on A/P or lateral radiographs (Wilson):

- Mild 0–33%.
- Moderate 33–50%.
- Severe >50%.

Carney classification

Depending on the difference in the Southwick angle between the involved and uninvolved side:

It is measured in frog leg view. It is formed by a line perpendicular to the physis and diaphyseal line.

Slip is graded based on the difference between the affected and unaffected hip.

- Mild 0–30%.
- Moderate 30–50%.
- Severe >50%.

How would you manage this patient?
The aim of treatment is to:

- Prevent further slippage.
- Facilitate closure of the physis.

Pinning in situ without reduction with a single cannulated screw is the method of choice for treatment of SUFE.

Other methods of fixation

- Primary osteotomy.
- Sub-capital (Dunn), Basal trochanteric, Intertrochanteric.
- Bone graft epiphysiodesis.

After the appropriate consent for the procedure, I would undertake in situ pinning under GA. I would not attempt any forceful reduction manoeuvres. Reduction is often obtained with positioning. I would start my drill on the anterior surface of the neck to cross perpendicular to the physis and enter the central portion of the femoral head. I would aim for a minimum of three threads to cross the physis. I would make sure the screw tip is at least 5 mm from subchondral bone in all views.

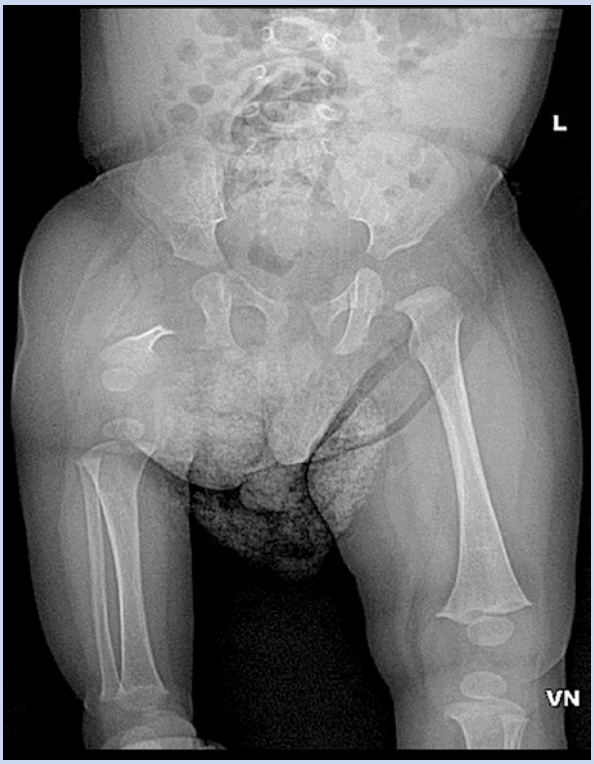
Stable slips can bear weight after in situ pinning. Unstable slips are made non-weight bearing.

Would you pin the contralateral non-slipped, asymptomatic side?
This is controversial but my indications for contralateral prophylactic pinning include:

- Obese male.
- Endocrine disorder or renal osteodystrophy.
- Young patient age <10 years.
- Poor family compliance.

So, I would not pin the opposite side in this case,

10.2 Viva 77



- **What is the diagnosis?**
- **Are you aware of any classification systems for this condition?**
- **What are the treatment principles?**

What is the diagnosis?

This a radiograph of both lower limbs of a skeletally immature child showing absence of right proximal femur and a dysplastic acetabulum. The right knee is proximally located. The features are in keeping with proximal femoral focal deficiency.

Are you aware of any classification systems for this condition?

The Aitken Classification

Class A: This is the mildest form of PFFD. In Type I PFFD, the femoral head and acetabulum are relatively normal. There may be some shortening of the femur, but the hip joint is usually well-formed.

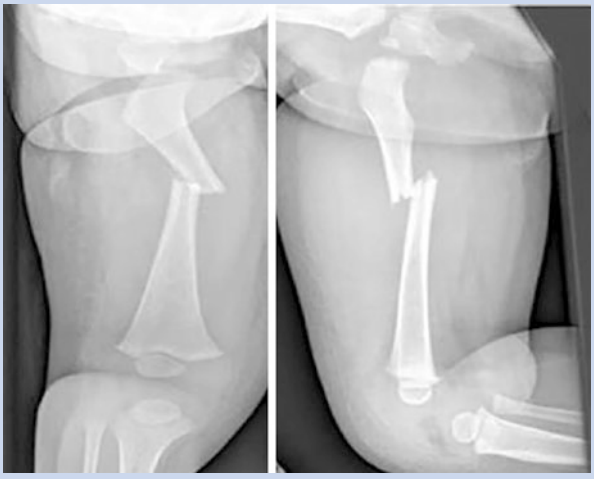
Class B in Type II PFFD, there is a partial absence of the proximal femur, and the femoral head may be abnormal. However, the acetabulum is dysplastic.
Class C Type III PFFD is characterized by the complete absence of the femoral head, neck, and proximal shaft. The acetabulum is severely dysplastic.
Class D: This is the most severe form of PFFD. In this type, the entire femur is absent, and the hip joint is usually non-existent.

What are the treatment principles?
Treatment is challenging and is undertaken in a specialized centre with interest and expertise. NICE guidelines were released in 2009 for bony and soft tissue reconstruction.

Treatment is individualized based on ultimate expected LLD, Hip and Knee Stability, Proximal Musculature, Condition of foot, Availability of Expertise and Patient and family motivation.

Treatment Options
- Non-operative (extension prosthesis) → in bilateral.
- Lengthening: indicated when predicted LLD at maturity <20 cm with stable hip and functional foot.
- Knee arthrodesis with ablation of the foot: when the foot on the affected side is at the level of contralateral knee or higher with non-functional foot.
- Van Ness rotationplasty: when the foot on the affected side is at the level of contralateral knee or higher with stable functional ankle.

10.3 Viva 78



- **Describe the radiograph.**
- **If this child were 15 months old, what potential concerns would you have?**
- **Tell me about non-accidental injury and what are the risk factors?**
- **What are the features that raise the suspicion of NAI?**
- **What are the most common injuries in NAI?**
- **What are some conditions that can be mistaken for NAI, and how can they be detected?**
- **How will you manage this 15-month-old child?**

Describe the radiograph
These are AP and lateral radiographs of a skeletally immature femur showing an oblique fracture of the proximal third.

If this child were 15 months old, what potential concerns would you have?
I would place non-accidental injury at the top of my list.

Tell me about non-accidental injury and what are the risk factors?
It is an injury that is caused by a parent or a caregiver.

It may be difficult to suspect a parent or caretaker of abuse, but we have a duty of care as professionals ensure care and protection of children.

Risk factors include unplanned pregnancy, firstborn, premature babies, stepchildren, family history of abuse, and parental IV drug abuse.

What are the features that raise the suspicion of NAI?
- Multiple injuries of different ages.
- Injury that is not consistent with the history stated or the developmental age of the child.
- Delayed presentation, reluctance to seek help and fear of medical examination.
- Child is brought to different surgeries/departments (to avoid detection of repeated injuries).
- Unexplained denial or aggression.
- No explanation for the injuries, a story that changes on repetition, or child's story differs from carers.
- Bruises at sites where accidental bruising is unusual: face, eyes, ears (bruising around the pinna may be subtle), neck and top of shoulder, anterior chest, abdomen.
- Petechiae (tiny red or purple spots) not caused by a medical condition—may be due to shaking or suffocation.
- Fractures: fractures of different ages, skull fractures, any scapular fracture, posterior rib fractures, outer end of clavicle digital fractures, metaphyseal corner fractures, bilateral or multiple diaphyseal fracture, transphyseal separation.

What are the most common injuries in NAI?

Bruises and skin lesions are the most common injury.

Fractures are the second most common.

What are some conditions that can be mistaken for NAI, and how can they be detected?

- Osteogenesis imperfecta: blue sclera, poor dentition, family history of multiple fractures, biochemical testing.
- Rickets: prematurity, vitamin D deficiency, metabolic and biochemical testing, wide physes, bowlegs, "Rachitic rosary".
- Kidney disease: history of dialysis, laboratory tests, urinalysis.

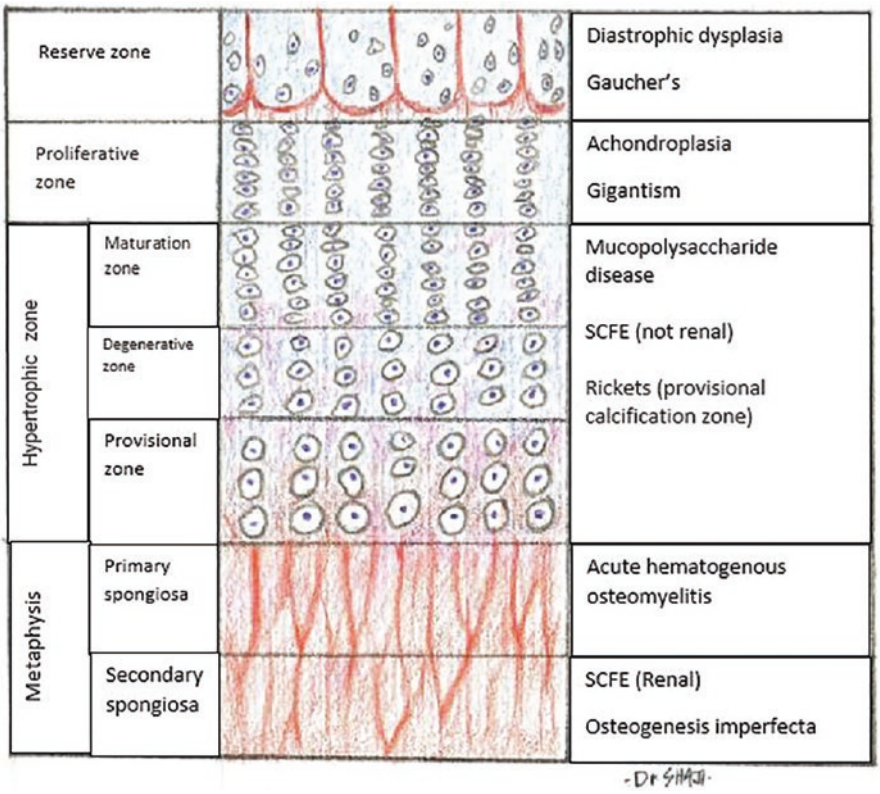
How will you manage this 15-month-old child?

- Firstly, it is important to get the child into a safe environment.
- Keep patient comfortable with pain medication.
- Treat the traumatic injuries appropriately in the same way as for an accidental injury, according to Advanced Trauma Life Support (ATLS) guidelines and being mindful that there may be other more life-threatening injuries (subdural haematoma and 'shaken baby' syndrome).
- A detailed history from the parents and examined the child fully from head to toes.
- I would inform the paediatricians and child medical protection team of my concern about a possible NAI and make arrangements for the child to be admitted.
- Skeletal survey: (1) Anteroposterior (AP) skull. (2) Lateral skull. (3) AP chest. (4) Oblique left ribs. (5) Oblique right ribs. (6) AP abdomen and pelvis. (7) Lateral spine. (8) Dorsopalmar both hands. (9) Dorsoplantar both feet. (10) AP left humerus. (11) AP right humerus. (12) AP left forearm. (13) AP right forearm. (14) AP left femur. (15) AP right femur. (16) AP left tibia/fibula. (17) AP right tibia/fibula.
- I would treat this fracture in gallows traction (overhead Bryant skin traction) is useful for children younger than 2 years who weigh 10–12 kg for fracture shaft of femur. Vascular compromise is the biggest danger. Check the circulation twice daily. Other complications: compartment syndrome-peroneal nerve palsy-skin break down. The buttocks should be just off the bed, with a radiograph at 2–3 weeks, to show callus formation, and then gentle mobilization as comfort allows. A hip spica is sometimes used.

10.4 Viva 79

- **Draw the different layers of the growth plate (physis) and give an example of a disease that affects each layer?**
- **How does longitudinal and circumferential bone growth occur?**
- **What classification do you use for physeal fractures?**
- **What is the significance of this classification?**

Draw the different layers of the growth plate (physis) and give an example of a disease that affects each layer?



How does longitudinal and circumferential bone growth occur?

Longitudinal bone growth occurs at the growth plate by endochondral ossification.

Circumferential growth by two mechanisms:

- Along the length of diaphysis, appositional ossification within the inner layer of the perichondrium leads to new bone formation by osteoblasts.
- Around the physis, the groove of Ranvier contains chondrocytes that are responsible for circumferential growth.

The perichondrial ring of LaCroix lies outside the groove of Ranvier, anchoring the epiphysis and metaphysis.

What classifications do you use for physeal fractures?
The most widely used system today is the Salter–Harris classification:

- Type I Injury through the physis without fracture through the metaphysis (5%).
- Type II injuries involve separation of a portion of the physis with the fracture progressing out of the metaphysis (75%).
- Type III injuries involve a fracture that runs through a portion of the physis and out through the epiphysis (10%).
- Type IV fractures are longitudinal splits through the epiphysis, physis and metaphysis (10%).
- Type V fractures involve a crush injury of the growth plate and are not evident on radiographs at the time of injury. Rare.

Peterson added another two types:

- Type VI with metaphyseal fractures extending to the physis.
- Type VII with loss of the physis (VIIa for central and VIIb for peripheral).

Ogden added another three:

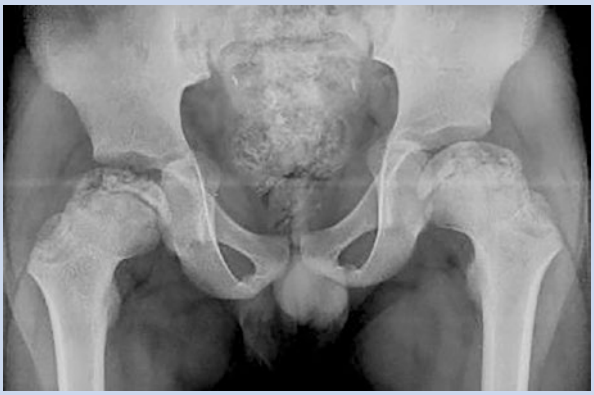
- Type VII: Epiphyseal fractures not involving physis.
- Type VIII: Metaphyseal fractures affecting later growth.
- Type IX: Periosteal damage affecting later growth.

What is the significance of this classification?
- The higher the grade the worse the prognosis (Table 10.1).
- The location of the physeal fracture is also an important prognostic factor.
- Undulant physis or irregular physis (such as the distal femur and distal tibia) have the worst prognosis as the fracture is more likely to affect several layers of the physis.

Table 10.1 Physeal injuries and growth disturbances

Type	Risk of growth disturbance (%)
I	7
II	2–3
III	14
IV	18

10.5 Viva 80



- **Tell me what you can see. What is the differential diagnosis?**
- **What is LCPD?**
- **How do they present?**
- **How do you classify this condition?**
- **What factors affect prognosis?**
- **What are the goals and principles of the management of this condition?**
- **What is hinge abduction and how do you treat it?**
- **What are the long-term sequelae of this condition?**
- **What are technical difficulties with THA in this condition?**

Tell me what you can see. What is the differential diagnosis?
This is an AP pelvic radiograph of a skeletally immature patient showing flattening, fragmentation, and deformity of both femoral heads. There is also lateral extrusion of both heads. At the top of my list is Legg-Calvé-Perthes disease (LCPD), but other diagnoses such as infection need to be excluded.

Differential diagnosis

Bilateral LCPD is not common and requires skeletal survey and blood tests to exclude:

1. Hypothyroidism.
2. Multiple epiphyseal dysplasia.
3. Spondyloepiphyseal dysplasia.
4. Meyer's dysplasia.
5. Sickle cell.
6. Gaucher's disease.

Unilateral involvement:

1. Septic arthritis (usually the child is unwell, fever with high inflammatory markers).
2. Sickle cell (history, sickling test, Hb electrophoresis).
3. Eosinophilic granuloma (other lesions particularly in skull, radiological features, biopsy).
4. Transient synovitis.

What is LCPD?

It is an idiopathic avascular necrosis of the femoral capital epiphysis in children. The cause is unknown. It is thought to be multifactorial.

It is affecting children between 4 and 9 years old. It is bilateral in 15–20% of cases but involvement is usually asymmetrical and never simultaneous (in contrast to multiple epiphyseal dysplasia).

It is more common in boys than girls by about 4:1.

How do they present?

- Onset is insidious, they commonly have a limp and pain in the groin, thigh, or knee.
- Antalgic/Trendelenburg gait.
- Leg length discrepancy.
- Decreased abduction and internal rotation.
- Flexion and adduction contracture may occur in advanced disease.

How do you classify this condition?

There are many classifications used for Perthes' disease.

Waldenström classified it into pathological stages:

- Initial (sclerotic/necrotic) stage: It lasts 6–12 months. Ischaemia leads to subchondral bone death and necrosis (dead bone looks dense on plain radiograph).
- Fragmentation (resorption) stage: In this stage, revascularization has started bringing osteoblasts and osteoclasts. The latter remove dead and necrotic bone causing radiolucent fissures among dead fragments. This stage usually lasts from 12 to 24 months.
- Reossification (healing) stage: Osteoblasts form new bone which is soft and pliable. It starts peripherally and progresses centrally. Last up to 18 months.
- Remodelling (residual) stage: The head is considered to have healed when there is no avascular bone visible on the radiographs; however, it continues to remodel until skeletal maturity. The head becomes large (coxa magna) and hard with residual deformity of head according to the shape at the end of the fragmentation phase.

Catterall (1971) Based on extent of head involvement on A/P and lateral views at fragmentation phase. Catterall advised four stages:

- Catterall I: 0–25% head involvement. Only anterior epiphysis (therefore seen only on the frog lateral film).
- Catterall II: 25–50% head involvement. Anterior and central segments.
- Catterall III: 50–75% head involvement. Anterior segment involved. Lateral head—also fragmented. **Only the medial portion** is spared.
- Catterall IV: the entire epiphysis is involved.

Salter and Thompson (1984) recognized that Catterall's first two groups and second two groups were distinct and therefore proposed a two-part classification; this is often referred to as modified Catterall's classification. Salter and Thompson Group A: Less than 50% of the head is involved. Salter and Thompson Group B: More than 50% of the head is involved. Again, the main difference between these two groups is the integrity of the lateral pillar.

(Herring) lateral pillar 1992. This is based specifically on the integrity of the lateral pillar on the AP film only, at the beginning of the fragmentation phase. The femoral head is divided into medial, central, and lateral (Pillar) segments, the central segment is the largest representing 50% of the width with the medial and lateral segment representing 20–30% each.

- Group A: Normal height of the lateral one-third of the head is maintained. Fragmentation occurs in the central segment of the head.
- Group B: More than 50% of the original lateral pillar height is maintained.
- Group C: Less than 50% of the original lateral pillar height is maintained.
- Group B/C: This group has been added recently to increase the prognostic accuracy of the lateral pillar classification. In this group the lateral pillar is narrow to 2–3 mm wide or poorly ossified or exactly 50% of lateral pillar height is maintained.

This classification has the best interobserver agreement. Limitation is that final classification is not possible at initial presentation due to the fact that the patient needs to have entered into the fragmentation stage radiographically.

Stulberg classification showed that a lack of sphericity and congruency were both predictors for poor outcome. A modified version of the Stulberg classification is becoming more popular. It consists of three groups:

- Group A hips (Stulberg I and II) have a spherical femoral head.
- Group B (Stulberg III) have an ovoid femoral head.
- Group C (Stulberg IV and V) have a flat femoral head.

What factors affect prognosis?

- Gender. Females have poor prognosis; they mature earlier with less remodelling potential.
- Age. The most important prognostic factor. Children older than 8 years at the onset of diagnosis have a guarded prognosis.
- Obesity. Poor prognostic factor.
- Bilateral involvement.
- Stiffness with progressive loss of ROM.
- Catterall classification. Type III and IV have poor prognosis.
- Herring classification. Groups B and C have poor prognosis.
- Sphericity of femoral head and congruency at skeletal maturity (Stulberg classification).
- Catterall head at risk signs:
  - Gage sign: radiolucency in the shape of V in the lateral portion of the epiphysis indicating a soft lateral pillar +/− the adjacent metaphysis.
  - Calcification lateral to the epiphysis—implies loss of lateral support and head extrusion.
  - Lateral subluxation of the femoral head.
  - Horizontal physis—implies a lateral growth arrest phenomenon and deformity.
  - Metaphyseal cyst: part of physis is damaged with infiltration into the metaphysis.

What are the goals and principles of the management of this condition?

The Goals of Treatment

- Alleviate the symptoms.
- Maintain range of motion.
- Achieve and maintain dynamic containment of the femoral head in the acetabulum to facilitate healing of the femoral head in a spherical geometry.
- Avoid complications such as AVN and degenerative disease.

Principles

- Group A lateral pillar → no treatment.
- Group B or B/C in children >6 years → better outcomes with surgical treatment (femoral or pelvic osteotomy).
- Group B or B/C in children <6 years → have good outcomes unrelated to treatment.
- Group C → poor prognosis regardless of the treatment.
- For patients with a good prognosis, with containable hips → symptomatic treatment, NWB and supportive measures are usually adequate (pain medications-PT to improve and maintain ROM-activity restriction).
- Literature does not support use of orthotics.
- All patients require periodic clinical and radiographic follow-up until completion of disease process.

What is hinge abduction and how do you treat it?
It is intraoperative finding (EUA arthrogram) in which there is lateral extrusion of the femoral head resulting in impingement of the femoral head on the edge of the acetabulum with abduction. It is treated with valgus-extension proximal femoral osteotomy.

What are the long-term sequelae of this condition?
- Enlargement of the femoral head (Coxa magna).
- Flattening of the femoral head (Coxa plana) → The mushroom femoral head.
- Damage of the femoral physis that result in shortening and widening of the femoral neck (Coxa breva).
- Hinge abduction.
- Coxa vara.
- Degenerative disease.

What are technical difficulties with THA in this condition?
- Anteverted femoral neck may lead component malpositioning and increased risk of dislocation or proximal femur fracture.
- A previous femoral osteotomy may cause difficulties in reaming the femoral canal.
- Increased risk of HO if prior hip surgery has been performed.
- Distorted anatomy.

10.6 Viva 81

A 4-year-old presents with a 24-h history of limping and progressive inability to bear weight. The parents recount no history of trauma but note that he recently had an upper respiratory tract infection.

- **Outline the initial steps in investigating and managing this child in line with your thoughts.**
- **Should X-rays be obtained to evaluate for septic arthritis?**
- **ESR 57, CRP 44, WBC 11.1. What information is provided by these results? What is the supporting evidence from the literature?**
- **What are the next steps in your management of this child? Describe exactly what you would do?**
- **What are the different ways bacteria can enter the joint?**
- **What organism is most implicated in septic arthritis in neonates? In children less than 2 years old? In children greater than 2 years old?**
- **What is the difference in the synovial fluid analysis between infection and non-infectious conditions?**

Outline the initial steps in investigating and managing this child in line with your thoughts

This is a clinical picture of a child with a flexed, abducted and externally rotated attitude of left lower limb. This scenario mandates a high index of suspicious for septic arthritis.

My clinical assessment would start with a detailed history from the parents. On examination, I would assess the general condition of the child and request a temperature measurement and vital signs. I would assess the resting posture and range of motion of the hips. I would also examine the whole lower limb, chest, abdomen, and spine. My aim is to differentiate Septic arthritis from Transient synovitis.

Urgent blood test will be done (CRP, ESR, CBC and blood cultures). I would also request pelvis X-ray. Antibiotics should be withheld until I have a microbiological sample.

Should X-rays be obtained to evaluate for septic arthritis?

Yes, X-rays of the symptomatic joint should be obtained. They can help narrow the differential diagnosis as it may reveal fractures or bone lesions that are sources of joint pain. Radiographs can also increase the suspicion for septic arthritis in the presence of a joint effusion, increased joint space, localized soft tissue swelling, or evidence of osteomyelitis. However, a normal X-ray does not rule out septic arthritis (or an occult fracture).

ESR 57, CRP 44, WBC 11.1. What information is provided by these results? What is the supporting evidence from the literature?

Kocher's landmark paper (JBJS-Am 1999) set out four criteria based on a retrospective review that was used in the differentiation between septic arthritis and transient synovitis in a child presenting with a hip pain:

- Inability to weight bear on the affected limb.
- Fever >38.5.
- ESR >40.
- WBC >12.

Based on the number of these criteria that are met, the likelihood of underlying septic arthritis can be predicted. In this case three of four criteria are met, suggesting a 93% likelihood of hip sepsis (Table 10.2).

Table 10.2 Kocher criteria to determine risk for pediatric septic joint

Kocher's four criteria	Significance
Inability to weight bear	Four criteria met: 99% septic arthritis
ESR >40 mm/h or CRP >20	Three criteria met: 93% septic arthritis
Fever >38.5	Two criteria met: 40% septic arthritis
WBC >12,000/mm	One criterion met: 3% septic arthritis

What are the next steps in your management of this child? Describe exactly what you would do?

The safest intervention in this case would be to proceed directly to open surgical drainage to avoid destruction of the articular surface by lytic enzymes from both bacteria and neutrophils (within 8 h). This is undertaken via an anterior approach (Smith-Peterson) approach. Inter-nervous plane between the femoral and superior gluteal nerves. Superficial dissection between the Sartorius and tensor fascia lata, at which point the ascending branch of lateral femoral circumflex artery must be identified and ligated. The dissection then continues in the same plane between the rectus femoris and the gluteus medius. The capsule is then identified. Once the joint capsule is opened, fluid should be taken and sent for culture and sensitivity. Copious lavage should be undertaken, using normal saline. The capsule should then be closed, and the wound repaired in layers. There is no evidence to support leaving a drain.

Following surgery, empirical antibiotics should continue until culture results are available, at which point the antibiotic can be adjusted as required depending on C/S. Clinical and haematological parameters should be closely monitored to assess the need for further washout. The child should be allowed to weight bear as tolerated.

What are the different ways bacteria can enter the joint?

Bacteria gain access to the joint via hematogenous dissemination, by local spread of disease (e.g., osteomyelitis), or via direct inoculation from trauma or surgery. The metaphysis is intra-articular. So direct spread from a metaphyseal osteomyelitis is a high risk in children up to 12–18 months of age. The pattern of circulation then changes and the physis forms a more effective barrier to spread.

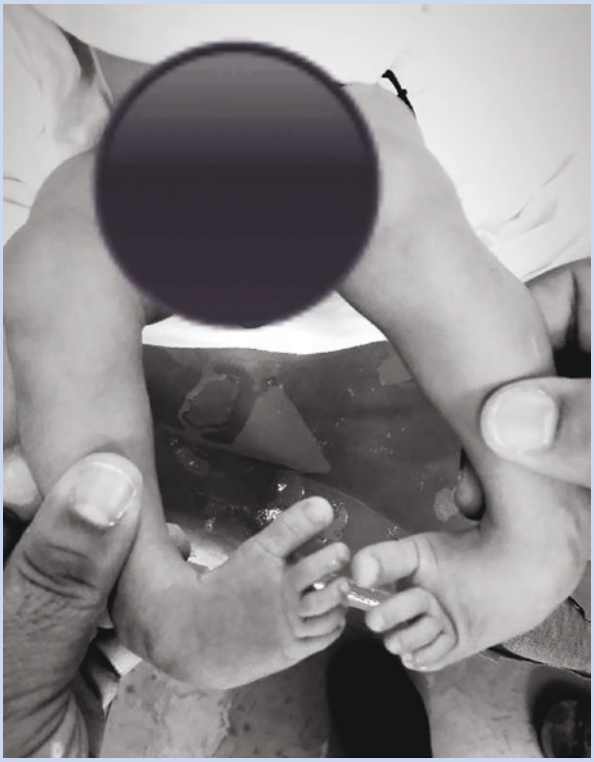
What organism is most implicated in septic arthritis in neonates? In children less than 2 years old? In children greater than 2 years old?

- *Staphylococcus aureus* in all these age groups.
- Haemophilus influenza in children less than 2 years old.
- *Neisseria gonorrhoeae* is a common cause in adolescents.

What is the difference in the synovial fluid analysis between infection and non-infectious conditions?

Conditions	WCC (per mm^3)	PML (%)	Other characteristics
Non inflammatory	200	25	Joint aspirate glucose and protein equal to serum values
Inflammatory	2000–75,000	50	↓ Joint aspirate glucose, low viscosity, yellow-green, friable mucin clot. Synovial complement in low in RA but normal in AS
Infectious	>80,000	>75	Thick, cloudy fluid +Cultures ↓ Joint aspirate glucose ↑ Joint aspirate protein

10.7 Viva 82



- **Describe the clinical photo.**
- **What is the aetiology of this condition?**
- **What are the associated conditions?**
- **How do you classify this condition?**
- **How would you manage this child?**

Describe the clinical photo

This clinical photograph displays deformities in both feet of a young child, including hindfoot equinus and varus, midfoot cavus deformity, and forefoot adduction. These findings are indicative of Congenital Talipes Equinovarus (CTEV).

What is the aetiology of this condition?

The cause in the majority of cases is unknown (idiopathic). A few theories have been introduced to explain the aetiology:

1. The neuropathic theory. 25 Biopsies were taken from the posteromedial and peroneal muscle groups in 60 patients mostly under the age of 5 years. Evidence of neurogenic disease was seen in most instances and was more obvious in the older patients.
2. The myopathic theory. 26 histochemical analysis was made of 103 muscle biopsies taken from 62 patients with idiopathic club feet. The authors noticed the muscles in patients aged under 6 months contained 61% Type 1 fibres in the affected legs, compared with 44.3% in normal legs.
3. Arrest of development of the growing limb bud.
4. Congenital constriction of the annular band.
5. Viral infection.
6. Mechanical moulding theory.

What are the associated conditions?
These include the following (FAT PANDAS):

1. Fetal alcohol syndrome.
2. Tibial hemimelia.
3. Prune belly.
4. Arthrogryposis.
5. Neurological causes: spina bifida (myelomeningocele), polio, cerebral palsy.
6. Down syndrome (may include vertical talus).
7. Amniotic constriction band syndrome (Streeter's dysplasia).
8. Sacral agenesis.

How do you classify this condition?
Pirani scoring system, based on the severity clinical findings and the correctability of the deformity. It composed of two main scores, the midfoot contracture score and the hindfoot contracture score, which are combined to give a possible maximum total score of 6. (A high score correlates to a more deformed foot.) Each of these scores is made up of 3 separate components which are graded as 0, 0.5, or 1. The individual components of the deformity assessed are: severity of medial crease, coverage of the lateral head of talus, and curvature of the lateral border used in the midfoot score and rigidity of equinus, severity of the posterior crease, and degree of emptiness of heel for the hindfoot score (Table 10.3).

Table 10.3 Pirani score system

Parameters	Mild	Moderate	Severe
Midfoot			
Curved lateral border	0	0.5	1
Medial foot crease	0	0.5	1
Talar head coverage	0	0.5	1
Hindfoot			
Posterior crease	0	0.5	1
Rigid equinus	0	0.5	1
Empty heel	0	0.5	1

How would you manage this child?
Having established the diagnosis of idiopathic club feet, my management is serial casting by the Ponseti method. Aim of treatment: painless functional plantigrade foot without need of orthotic shoe in the first year. The treatment should be started as early as possible; serial casting weekly for up to 3 months. Sequence of deformity corrections (CAVE):

- Cavus is corrected first by supination of the forefoot via dorsiflexion of the first MT (supination of the forefoot will make the foot look worse after the first cast, so I have to warn the parents).
- Adductus and varus are simultaneously corrected by abducting the foot at mid-foot level using the uncovered head of the talus laterally as a fulcrum. Above-knee casts with the knee at 90° (to prevent cast falling off and control tibia rotation) are applied with moulding into the corrected position and then each week the old cast is removed, the foot is scored, and then subsequent casts are applied. Foot supination is slowly decreased during each casting.
- The midfoot usually corrects well after four or five casts. If there is residual equinus (or less than 20° of dorsiflexion) of the hindfoot then this can be addressed by performing an Achilles tenotomy under a local or general anaesthetic.
- A final cast is applied for a further 3 weeks while the tenotomy heals.
- Babies then go into Denis Browne boots with a bar (23 h a day for 3 months then just at night and naptime until the age of 5 years). This holds the affected foot externally rotated at about 70°. 40° to the normal side. The vast majority of patients do very well and avoid the need for extensive surgical release 90%. However, approximately 25% will require a tibialis anterior transfer laterally (SPLATT) for residual deformity or inversion in swing after the age of about 4–5 years.
- In cases of recurrence.
  - <2 years: repeat Ponseti.
  - 2 years: Casting + soft tissue +/− bony procedures.

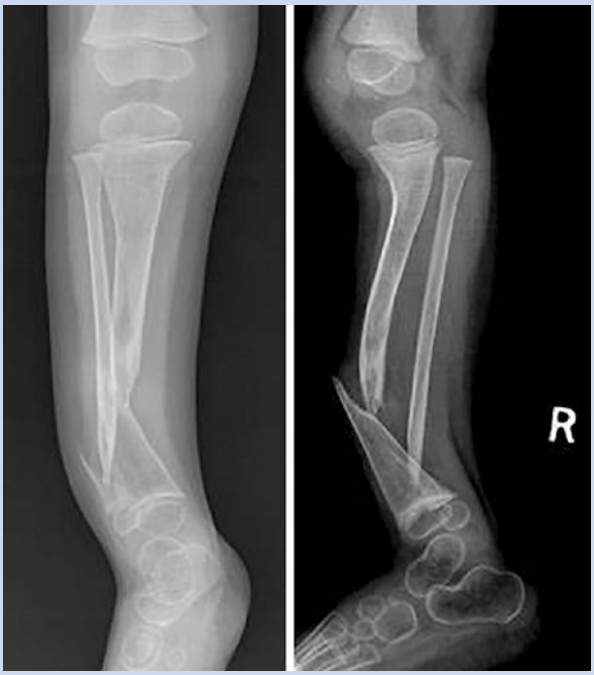
  Surgical options for relapse.

Soft Tissue Release
- Medial release of everything except the deltoid.
- Posterior release of capsule and Achilles (Cincinnati incision).

Bony Procedures
- Calcaneal slide (lateral slide to correct varus/medial slide to correct valgus).
- Cuboid osteotomy: to allow the forefoot to rotate around talo-navicular joint.

Tendons Transfer
- SPLATT or whole tendon transfer if the patient is unable to evert their foot actively.

10.8 Viva 83



- **Describe the radiograph.**
- **Are you aware about any classification for this condition?**
- **What are the principles of management of this condition?**

Describe the radiograph

This is an A/P radiograph showing anterolateral bowing of the tibia with pseudoarthrosis, there is tapering of the tibia at the defective site.

50% of patients with anterolateral bowing have neurofibromatosis.

10% of patients with neurofibromatosis have anterolateral bowing.

Are you aware about any classification for this condition?

Boyd and Crowford Classification

- Type I: anterolateral bowing with increased cortical density, narrow but normal medullary canal.
- Type IIA: anterolateral bowing with widened medullary canal.
- Type IIB: anterolateral bowing with cystic lesion before fracture.
- Type IIC: frank pseudoarthrosis with tapering of the tibia at the defective site.

What are the principles of management of this condition?
The primary goal of treatment is the prevention of pseudoarthrosis. Several methods can be employed to manage anterolateral bowing and its associated pseudoarthrosis:

Prevention of Pseudoarthrosis:

- Utilize a total contact brace.

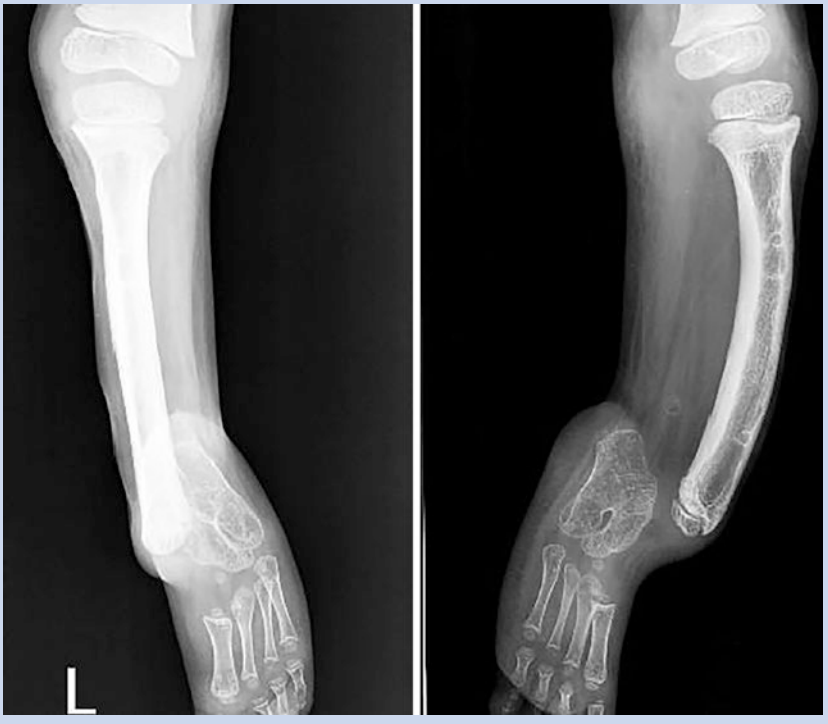
Treatment of Pseudoarthrosis: In cases where pseudoarthrosis has already developed, there are limited options available. Treatment methods include:

- Intramedullary rod placement with bone grafting.
- Circular fixator with bone transport.
- Vascularized fibular graft.

Amputation Indication:

In cases of persistent pseudoarthrosis that do not respond to the above treatments, amputation may be indicated as a last resort.

10.9 Viva 84



- **What is the diagnosis?**
- **What are the associated abnormalities of this condition?**
- **Any classification for this condition?**
- **What are the principles of treatment?**

What is the diagnosis?

This is a radiograph of left lower limb of skeletally immature child, showing anteromedial bowing of the tibia with entire absence of fibula. Known as fibular hemimelia.

It is the most common long bone deficiency.

What are the associated abnormalities of this condition?

- Shortening of the femur or tibia.
- Genuvalgum deformity from lateral femoral condyle hypoplasia.
- Cruciate ligament deficiency.
- Anteromedial bowing of the tibia.
- Ball and socket ankle.
- Equinovalgus foot deformity.
- Tarsal coalition.
- Absence of lateral rays.

Any classification for this condition?

Johnson Classification

- Type I: Shortened fibula with Ia: normal foot. Ib: equinovalgus deformity.
- Type II: complete absence of the fibula with foot deformity.
- Type III: bilateral.

What are the principles of treatment?

The management depends on several factors, including the functional status of the foot and the degree of limb length discrepancy (LLD):

Nonfunctional Foot

If the foot is nonfunctional, amputation may be considered, with options like Syme or Boyd amputation.

Functional Foot with LLD <5 cm or ≤10%

When the foot is functional, and the LLD is less than 5 cm or less than 10%, limb lengthening procedures can be a viable option.

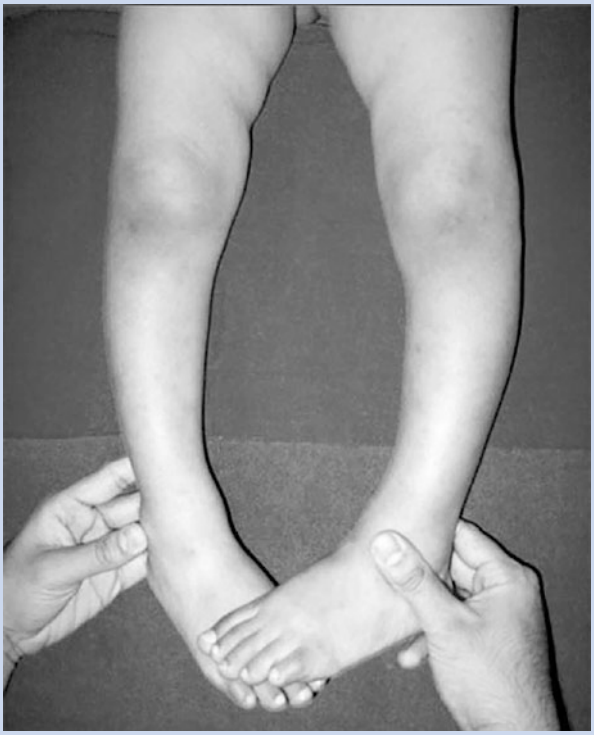
Functional Foot with LLD 5–10 cm or 10–30%

In cases where the foot is functional, but the LLD is between 5–10 cm and 10–30%, the decision may be limb lengthening procedures.

Functional Foot with LLD >30%

If the foot is functional, but the LLD exceeds 30%, amputation may be the recommended course of action.

10.10 Viva 85



- **What can you see?**
- **What are the causes of bowing?**
- **How can you distinguish between physiological and pathological bowing?**
- **What is Blount's disease?**
- **Outline the radiographic measurements of alignment and angles used in lower limb angular deformity**
- **Are you aware of any classification system of this condition?**
- **What are the principles of treatment?**

What can you see?
The photograph shows bilateral genu varum in a child.

What are the causes of bowing?
The causes of bowing are summarized in Table 10.4.

Table 10.4 Differential diagnosis for genu varum and genu valgum

Cause	Genu varum	Genu valgum
Congenital	Fibular hemimelia	Tibia hemimelia
Acquired		
Dysplasia	Skeletal dysplasia	Skeletal dysplasia
Developmental	Primary genu varum (Blount's)	Primary tibia Valga
Infection	Growth plate injury	Growth plate injury
Trauma	Partial physeal arrest	Partial physeal arrest
Tumour	Osteochondroma	Osteochondroma
Metabolic	Vit D deficiency Osteogenesis imperfecta	Renal osteodystrophy

How can you distinguish between physiological and pathological bowing?
Genu varum is more likely to be pathological if it is:

- Present after 2 years.
- Unilateral or with asymmetry.
- Associated with shortening of the limb LLD (or stature).
- Severe (beyond 2 SD of the mean as per Salenius chart; SD = 8°).
- In a child with obesity.
- Associated progressive deformity.
- Associated apex at the proximal tibia or lateral thrust.

Genu valgus is more likely to be pathological if it is:

- Severe (intermalleolar distance >10 cm at 10 years or >15 cm at 5 years).
- Unilateral.

What is Blount's disease?
It's a developmental disorder that characterized by disordered growth of the medial aspect of proximal tibia physis resulting in progressive LL deformity.

There are two recognized types: (1) Infantile (0–3). (2) Late (juvenile 4–10. Adolescent >10 years).

- In infantile tibia vara, patients generally start to walk early (9–10 months); it is more severe, more prevalent in females, blacks, and those with marked obesity. It is bilateral in approximately 80% of cases. The deformity is painless.
- In the adolescent type, patients complain of pain at the medial aspect of the knee. These patients are overweight, and involvement is unilateral in 80% of cases with LLD.

Outline the radiographic measurements of alignment and angles used in lower limb angular deformity

1. Mechanical axis of limb: a line drawn from the centre of femoral head to centre of ankle should pass through the centre of knee (usually about 8 mm medial to the centre).

2. Tibio-femoral angle: the angle between longitudinal axis of femur and tibia (should be within the normal range presented by Salenius).
3. Metaphyseal diaphyseal angle (Drennan): the angle between a transverse line connecting the metaphyseal beak and a line perpendicular to the longitudinal axis of the tibia. <10° → 95% chance of resolution. >16° → 95% chance of progression.
4. Epiphyseal-metaphyseal angle (EMA): the angle between a line through the proximal tibia physis and a line connecting the base of the epiphyseal ossifical centre to the most distal point on the medial beak of proximal tibial metaphysis. EMA >20° → greater risk of developing Blount.

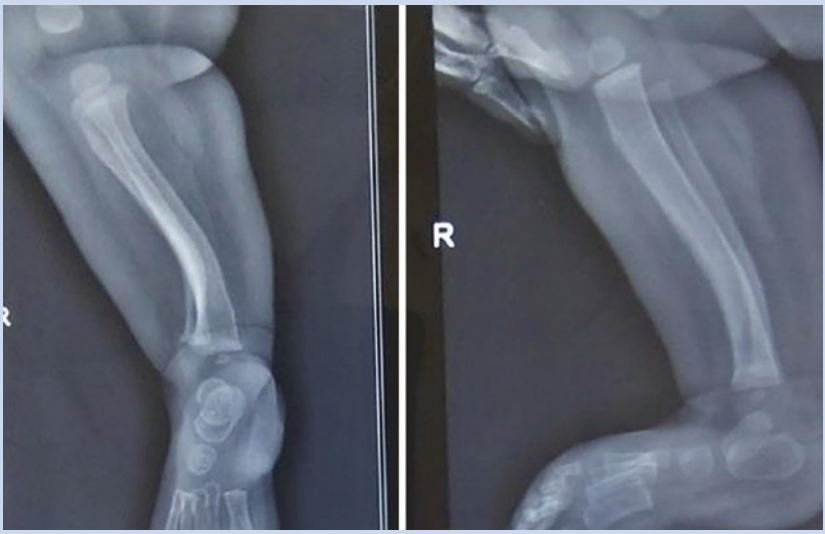
Are you aware of any classification system of this condition?
Langenskiold classification of Blount's disease:

- Medial metaphyseal beaking.
- Medial epiphyseal wedging.
- Medial epiphyseal irregularity.
- Epiphyseal filling of metaphyseal depression.
- Double epiphyseal plate.
- Medial physeal closure.

What are the principles of treatment?
Infantile
- Non-operative treatment with (KAFO): indicated in stage I&II. Must be continued until resolution of bony changes (2 years).
- Proximal tibia/fibula valgus osteotomy: indicated in > stage II—failure of non-operative management. Technique:
- Performed below tibia tubercle.
- Overcorrect into 10–15° because the medial physeal growth abnormalities persist.
- The distal fragment is fixed in slight valgus, lateral translation, and external rotation with pins.
- Anterior compartment fasciotomy to decrease the risk of post-op compartment syndrome.

Adolescent
- Non-operative treatment associated with poor outcomes.
- Surgical treatment depends on the age of the patient (status of physis) and the severity of the deformity.
- Guided growth: principles of Heuter Volkmann (compression slows growth while tension speeds up growth), Lateral tibia/fibula Epiphysiodesis, indicated mild to moderate cases. It corrects 1°/month. Apply fixation on tension side. Options: temporary (staples or plates) needs to be removed—permanent (drilling/curettage physis).
- Proximal tibia/fibula osteotomy in sever deformity and less growth left.

10.11 Viva 86



- **What is the diagnosis?**
- **What is the underlying cause of this condition?**
- **What advice can you offer to the child's parents regarding the prognosis of this condition?**

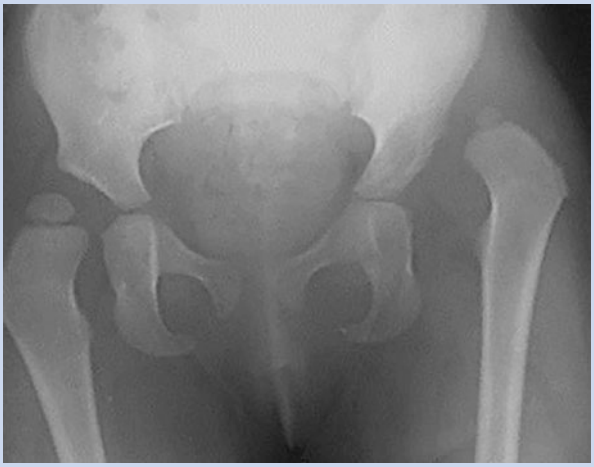
What is the diagnosis?
These are AP and lateral radiographs of a skeletally immature right lower extremity showing posteromedial tibial bowing.

It is a congenital condition that presents at birth and usually involves the middle and distal third of the tibia.

What is the underlying cause of this condition?
It is thought to be a result of intrauterine positioning which usually involves the middle and distal third of the tibia. It is commonly associated with calcaneovalgus foot, another intrauterine positioning condition. Initial management of this condition involves a period of observation, as the bowing usually resolves by 5–7 years of age.

What advice can you offer to the child's parents regarding the prognosis of this condition?
Bowing deformity is likely to resolve by the age of 5 (~80% of patients). However, patients need to be monitored long-term since the majority of patients have a residual leg length difference (LLD) of 2–5 cm. Greater than 50% of patients will require surgery at some point in the future to address the leg length difference.

10.12 Viva 87



- **Describe the radiograph.**
- **What is the aetiology of this condition?**
- **How is this condition detected on physical exam?**
- **How would you treat a child with this condition?**
- **What are the different types of femoral and pelvic osteotomy?**

Describe the radiograph

This is an anteroposterior (AP) pelvic radiograph of a skeletally immature child, revealing a dislocated left hip and a dysplastic acetabulum. There is an asymmetric appearance of the femoral nucleus, and the femoral head is situated in superolateral quadrant (made by the intersection of Perkin's and Hilgenreiner's lines). These findings strongly suggest the presence of Developmental Dysplasia of the Hip (DDH).

What is the aetiology of this condition?

DDH is a developmental disorder that describes a spectrum of pathologic conditions involving the developing hip, ranging from acetabular dysplasia to hip subluxation to irreducible hip dislocation.

The exact cause is largely unknown but is thought to be multifactorial. These include (6Fs):

1. **F**irst baby (the uterus is tighter and less elastic-less room for baby motion).
2. **F**emale (6:1. lax ligament due to maternal hormones).

3. **F**amily history (may be genetic predisposition. No parent involvement + one affected child → 6%. One parent involved + no sibling involved → 12%. Parent and sibling involved → 36%).
4. **F**oetal malposition (breech presentation with knee in extended position).
5. **F**oetal packaging disorders (oligohydramnios, twins, neck torticollis, congenital knee dislocation, metatarsus adductus).
6. Le**F**t side (60% left hip, 20% right and 20% both). May be related to the foetal position.

Family history and breech presentation are probably the most important factors.

How is this condition detected on physical exam?
The clinical presentation varies with age:

In the neonatal period: Ortolani and Barlow tests are very important in the early weeks of life, but their value becomes less as the child gets older. Ortolani test identifies a dislocated hip that can be reduced. By flexing the infant's hip and knee to 90°, the thigh is then gently abducted with the middle finger over the greater trochanter to feel for the reduction of the dislocated head as it comes from the dislocated position to the socket. With time, it becomes more difficult to reduce the femoral head into the acetabulum, and the Ortolani test becomes negative. The Barlow manoeuvre attempts to dislocate a hip that is reduced; the hip is flexed and adducted, and posterior pressure is exerted to push the hip out of joint. A Barlow positive involves feeling the femoral head clunk out of the acetabulum. Barlow test is rarely positive after 10 weeks.

In infants older than 6 months: limitation of motion and apparent LLD.

In walking child: in addition to the previously mentioned findings, you may observe lumbar lordosis, pelvic obliquity, a Trendelenburg gait, and a positive Galeazzi test, which is typically only observed in unilateral cases.

How would you treat a child with this condition?
The principles of treating DDH are:

- Achieve a concentric reduction of the hip joint.
- Maintain stability in the concentric reduction.
- Promote normal hip growth and development.
- Minimize complications.

Treatment Approaches:

For children younger than 6 months:

I would start with the Pavlik Harness. Close monitoring is essential, with weekly checks for any potential femoral nerve issues. If, after 3 weeks, the hip remains dislocated, I would opt to remove the Pavlik Harness. I'd schedule a follow-up at

6 months for an examination under anaesthesia (EUA), an arthrogram, adductor tenotomy, and Spica cast application. A subsequent CT scan would ensure the hip is adequately reduced. At 6 weeks, I'd review the patient for change of Spica.

For children aged 6–18 months:

In this age group, I would consider a closed reduction performed under general anaesthesia. Preliminary traction, which used to be common, is now less frequently used due to potential complications. Adductor tenotomy is often required. Intraoperative hip arthrography would be required to confirm the adequacy of reduction. During the procedure, I'd establish safe zones for hip positioning, ensuring that hip abduction remains below 60° to minimize the risk of avascular necrosis (AVN). I would apply a Spica cast to maintain reduction. Confirmation of the reduction through CT would be followed by cast changes at 6 weeks.

For children older than 18 months:

I would opt for an open reduction approach. Femoral osteotomy would be considered in children under 4 years of age, especially when dealing with unstable hips in adduction or abduction. In older children over 4 years, I would more commonly consider a pelvic osteotomy, which is indicated for hips unstable with rotation.

What are the different types of femoral and pelvic osteotomy?

Femoral Osteotomy

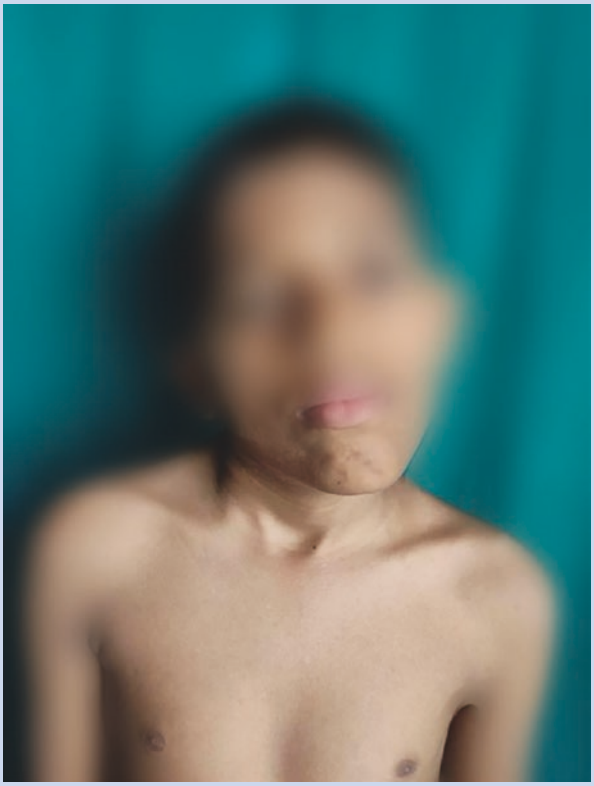
- It provides shortening (to decrease pressure on the femoral head and decrease the risk of osteonecrosis) derotation (external rotation to address the excessive femoral anteversion) and varus.

Pelvic Osteotomy

- *Reconstructive osteotomies*: redirect or reshape the roof of acetabulum with its normal hyaline cartilage into a more appropriate weight-bearing position. A prerequisite to a reconstructive pelvic osteotomy is a hip that can be reduced concentrically and congruently. The hip must be near-normal ROM.
- Redirectional osteotomies include Salter (single innominate), Steel (triple innominate), Ganz (periacetabular).
- Reshaping (Volume reducing): Pemberton and Dega. Indicated when the acetabulum is in a wrong shape (shallow and deficient superiorly), e.g.: CP.
- *Salvage osteotomies*: indicated in adolescents with severe dysplasia. Weight-bearing coverage is increased by using the joint capsule between the femoral head and the bone above it. These osteotomies rely on fibrocartilaginous metaplasia of the interposed joint capsule to provide an increased articulating surface.
  - Shelf: corticocancellous bone graft is added to the lateral weight-bearing part of the acetabulum which acts as extra-articular buttress. Healing by fibrous metaplasia.
  - Chiari: cut at supraacetabular area, the distal part is shifted medially.

10.13 Viva 88

A 7-year-old boy presented with the neck deformity and palpable mass.



- **What is the most likely diagnosis?**
- **What are the causes of this condition?**
- **How would you manage a child with this condition?**

What is the most likely diagnosis?
The most likely diagnosis is torticollis as the clinical photograph shows lateral tilting of the head with chin directing to the opposite side.

What are the causes of this condition?
Congenital
- Congenital muscular torticollis: the most common cause of infantile torticollis, occurs due to abnormal contracture of the sternocleidomastoid (SCM), associated with other packaging disorders (DDH—Metatarsus adductus), clinically

there is a painless palpable neck mass (the contracted SCM) is noted within the first 4 weeks of life, gradually subsides, becoming a tight band as the patient ages.
- Vertebral anomalies: Failure of segmentation—occipitalization of C1.

Acquired (Usually Painful)
- Traumatic: C1 fracture.
- Inflammatory: atlantoaxial rotatory subluxation—juvenile rheumatoid.
- Tumours: osteoid osteoma—eosinophilic granuloma—posterior fossa—acoustic neuroma—cervical cord.

How would you manage a child with this condition?

I would like to perform a thorough evaluation. This should include an inspection for any signs of asymmetry, such as differences in ear level, hairline, eye alignment, and temporal bones. Features suggestive of Klippel-Feil Syndrome should be noted, including a low posterior hairline, a short, webbed neck, and limited cervical range of motion.

It's essential to evaluate eye motion, which may not be easy before the age of 6 months. Visual disturbances can sometimes cause pseudo-torticollis, where a child tilts their head during gaze.

I would palpate the neck, paying attention to the sternocleidomastoid (SCM) muscle, lymph nodes, and the presence of any lumps. It's crucial to determine if the child experiences pain during palpation. A comprehensive evaluation of range of motion (ROM) is necessary, particularly in terms of rotation and lateral tilt. In cases where the child presents with head rotation and tilting similar to torticollis but with spasticity in the SCM on the contralateral side, I would consider the possibility of atlantoaxial rotatory instability (AARD). This is different from congenital torticollis, where the spasticity is on the same side.

If the history dates back to birth, and there's a palpable 'tumour' in the sternomastoid muscle that disappears by 4 months of age, and the sternomastoid feels tight, a diagnosis of congenital muscular torticollis can be made. The initial treatment typically involves passive stretching of the sternocleidomastoid. Encouraging the child to adopt a lateral head tilt away from the affected side and chin rotation toward the affected side is important (opposite of the deformity). In approximately 90% of cases, passive stretching of the sternocleidomastoid during the first year of life is effective in improving the condition.

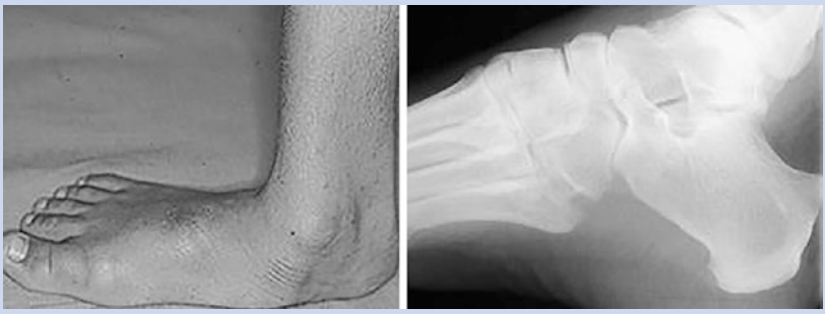
When stretching fails to improve the condition, surgical intervention may be indicated. The surgical options include unipolar (distal release of clavicular head and z-lengthening of the sternal head) or bipolar (as for unipolar, with proximal release just below mastoid attachment) release. While bipolar release often produces better results, the proximal release carries a risk of accessory nerve injury.

In cases where there is no palpable mass, imaging may be indicated to rule out other conditions.

If necessary, a dynamic CT scan of the cervical spine, particularly at the C1–2 level, may be conducted. This scan is essential for ruling out atlantoaxial rotatory subluxation. (Scan at the C1–2 level with head straight, then in maximum rotation to the right, and then in maximum rotation to the left, will see fixed rotation of C1

on C2 which does not change with dynamic rotation.) Treatment includes a soft collar initially. If conservative measures fail, head halter traction, benzodiazepines, and a hard collar may be considered. In the event of continued failure, C1–C2 fusion may be considered.

10.14 Viva 89



- **Describe what you see.**
- **What are the most common types of this condition?**
- **What is the pathoanatomy of this condition?**
- **What are the clinical features?**
- **What is the role of CT and MRI?**
- **What are the treatment options?**

Describe what you see

The clinical photograph shows a foot with loss of the medial longitudinal arch. The oblique radiograph of the foot shows an elongated anterior process of the calcaneus. The Features are consistent with a tarsal coalition—a calcaneo-navicular bar.

What are the most common types of this condition?

Calcaneonavicular	Talonavicular
More common	Less common
Age 8–12 years	Age 12–25 years
Radiology:	*Radiology*:
• Elongated anterior process of calcaneus (anteater sign). Oblique view	• C shaped arc formed by the medial outline of talar dome and posteroinferior aspect of sustentaculum tali. Lateral view
• Broad mediolateral dimension of the navicular bone (wider than Talar head) on AP view	• Talar beaking because of limited motion of subtalar joint. Lateral view
	• Brick sign. Distorted and curved subtalar joint

What is the pathoanatomy of this condition?
It occurs due to the failure of the primitive mesenchymal cells to differentiate and form normal articular separation between bones.

What are the clinical features?
- 75% of children are asymptomatic.
- History of recurrent ankle sprain and difficulty with uneven ground.
- Pain in sinus tarsi and inferior fibula suggests calcaneonavicular—pain distal to medial malleolus or medial foot suggests talocalcaneal.
- Pain worsened by activity.
- Hindfoot in valgus—forefoot in abduction (if adducted think of skew foot)—pes planus (rigid). Double heel raise fails to correct the hindfoot valgus.
- Jack's test fails to correct the Pes planus deformity.
- Limited subtalar motions.
- Heel cord contracture.

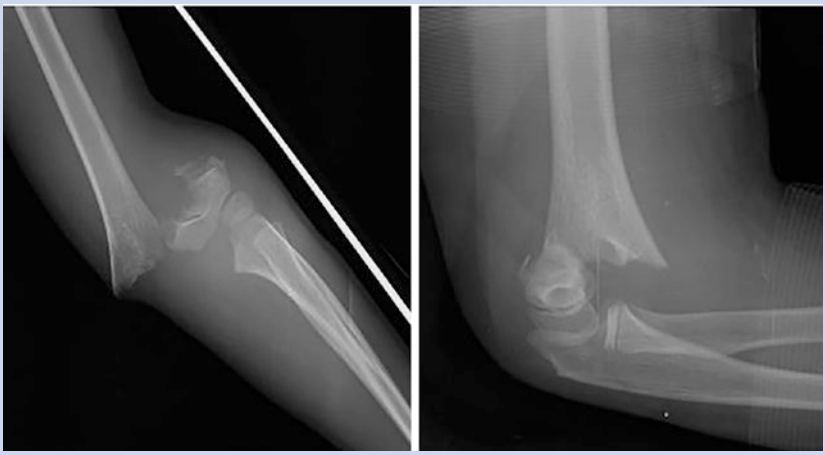
What is the role of CT and MRI?
CT Scan
- To exclude additional coalitions, incidence approx. 5%.
- Determine size, location, and extent of coalition.
- Size of talocalcaneal coalition based on size of posterior facet using coronal slices.

MRI
- Helpful to visualize a fibrous or cartilaginous coalition.

What are the treatment options?
- The initial treatment for symptomatic cases → NSAIDs—activity modification—shoe orthoses—immobilization in severe cases.
- In cases of persistent symptoms despite prolonged period of nonoperative management and coalition <50% → coalition resection with interposition graft, +/− correction of associated foot deformity.
  - Interposition material: extensor digitorum brevis (calcaneonavicular coalition)—split flexor hallucis longus tendon (talocalcaneal coalition)—interposed fat graft—bone wax.
  - Calcaneal osteotomy for hindfoot valgus.
  - Heel cord lengthening if intraoperative ankle dorsiflexion is not past neutral.
- If coalition involves >50% of the joint surface of a talocalcaneal coalition, consider subtalar arthrodesis.
- In advanced coalitions that fail resection—diffuse associated degenerative changes affecting calcaneocuboid and talonavicular joints, consider triple arthrodesis.

10.15 Viva 90

This 6-year-old child fell out of a tree onto his arm. Patient arrived middle of the night.



- **Can you describe the radiograph?**
- **What are the types of this injury? How can be classified?**
- **What does a fat pad sign typically indicate in a child?**
- **How would you manage this child?**
- **Which techniques would you utilize to minimize the risk of iatrogenic ulnar nerve injury when planning to place a medial pin?**
- **You reduce and pin the fracture under anaesthetic, but on post-operative review in recovery you are unable to feel a pulse. What would you do now?**

Can you describe the radiograph?
These are A/P and lateral views in a skeletally immature child that showed an 'off-ended' Gartland 3 supracondylar fracture of the distal humerus.

What are the types of this injury? How can be classified?
- Supracondylar fractures of distal humerus are divided into Extension and flexion types.
- Extension fractures account for approximately 98% of these injuries, and they usually occur as the result of a fall on an outstretched hand with the elbow in full extension.
- The Gartland classification is the most used scheme to describe supracondylar fractures. Type I fractures are nondisplaced, type II fractures have an intact

posterior hinge, and type III fractures involve complete displacement. Wilkins in 1984 modified the Gartland classification by dividing type II fractures into subtypes A and B. Type IIA fractures are extended but have no rotational abnormality. These fractures are frequently stable following a flexion reduction manoeuvre and can be held in a cast. In addition to extension deformity, type IIB fractures involve some degree of rotational element. These fractures are generally unstable after reduction.

- Leitch et al. recently proposed the addition of a type IV fracture to the Gartland classification. Type IV fractures are unstable in both flexion and extension because of complete loss of a periosteal hinge.

What does a fat pad sign typically indicate in a child?

An anterior fat pad sign is often physiologic. When a posterior fat pad sign is present, it is generally indicative of an elbow effusion. This most commonly is due to a non-displaced supracondylar humerus fracture (SCH). Approximately 80% of positive posterior fat pad signs are the result of occult fractures.

How would you manage this child?

I would manage this child as per BOAST guidelines.

- I would assess the child for the presence of an open injury and evaluate for signs of compartment syndrome.
- I would assess the limb on presentation and immediately before surgical treatment. I would assess and document the status of radial pulse, digital capillary refill time and the individual function of the radial, median (including anterior interosseous) and ulnar nerves. In patients with extension-type supracondylar fractures, anterior interosseous nerve injury is most common, this nerve is tested by flexion of the interphalangeal joint of the index finger and thumb, followed by median, radial, and ulnar nerve injuries. The ulnar nerve is most commonly injured in flexion-type fractures.
- I would organize for the child to have analgesia and a temporary backslab splint.
- Surgical management should be carried out on the day of injury. Night-time operating is not necessary unless there are indications for urgent surgery which include an absent radial pulse, clinical signs of impaired perfusion of the hand and digits, open injury, or evidence of threatened skin viability (Puckering).
- A perfused limb does not require exploration of the brachial artery, regardless of presence/absence of the radial pulse. Most vascular impairments associated with supracondylar fractures resolve with fracture reduction.
- An ischemic limb, however, needs discussion with the vascular team before reduction. If remains ischemic after reduction, then needs exploration by a surgeon trained in small vessel repair.
- In theatre: The technique for reduction of these injuries is to
  - 1. Apply good longitudinal continuous traction (in 20° of flexion) for several minutes.
  - 2. Correct any valgus/varus and rotational deformity, when a supracondylar fracture is displaced posteromedially, pronation in flexion may facilitate

reduction by placing the medial periosteum under tension and closing the lateral gap. Conversely, when the fracture is displaced posterolaterally, supination in extension may facilitate reduction.
  - 3. Flex elbow to lock it in place.
  - 4. Assess rotation by obtaining Jones view.
- In case of failed closed reduction → The anterior approach to the elbow provides the best exposure of the neurovascular structures and the soft-tissue obstacles anteriorly that prevent reduction. This approach is performed through either a transverse or an oblique incision made across the elbow flexion crease.
- In case you find brachial artery is in spasm you can use local anaesthesia or Papaverine or stellate ganglion sympathetic block to produce arterial vaso-dilatation.
- If these failed so it is intimal tear need excision of injured segment and reversed venous graft with prophylactic fasciotomy.
- Pins configuration:
  - 3-lateral pins biomechanically stronger in bending and torsion than 2-pin constructs.
  - No significant difference in stability between three lateral pins and crossed pins, risk of iatrogenic nerve injury from a medial pin makes three lateral pins the construct of choice.
  - Cross pins biomechanically strongest to torsional stress. (Highest risk if placed with elbow in hyperflexion as ulnar nerve subluxates anteriorly over medial epicondyle in some children.)
  - I would use 2 mm diameter wires where possible, to achieve stability. Divergent and 4-cortical fixation. Don't cross wires at fracture site.
- I would check for stability under fluoroscopy in external and internal rotation.
- I would bend and cut the wires for ease of removal in the clinic in 3–4 weeks' time. I would splint the arm in a backslab in semi flexion (<90°).
- I would reassess the perfusion of the hand and watch for compartment syndrome. (If the limb remains ischaemic after fracture reduction, then exploration of the brachial artery is required).
- Check X-rays in 1 week and remove wires in 4 weeks in clinic. It will take 6 months for 95% of normal elbow range of motion to return.

Which techniques would you utilize to minimize the risk of iatrogenic ulnar nerve injury when planning to place a medial pin?
- I would place medial pin with elbow in extension to allow the nerve to relax.
- I would use small medial incision (rather than percutaneous pinning).

You reduce and pin the fracture under anaesthetic, but on post-operative review in recovery you are unable to feel a pulse. What would you do now?
If I found no pulse in my post-operative review of the patient, I would make an assessment of the rest of the vascularity of the hand in terms of its colour and warmth and also the capillary refill time.

If the hand appeared pink, felt warm, and exhibited adequate capillary refill in the fingertips, I would closely monitor the situation with regular reviews. In such cases, the absence of a pulse might be due to arterial spasm, and it could take a day or two for the pulse to recover.

However, if the hand appeared white, and the capillary refill was reduced, I would release the back slab and extend the arm to observe if this improved the situation. If there was no improvement, I would promptly contact vascular or plastic surgeons for an urgent review. This is because the artery may have become entangled in the fracture, leading to occlusion during the reduction. In such cases, open exploration, typically through an anterior approach, would be necessary.

Scannell—JBJS 2013 After 20 months of follow-up, children with perfused, pulseless supracondylar humeral fracture treated with closed reduction, percutaneous pinning, and observation demonstrated palpable distal radial pulse, normal growth of arm, and good/excellent functional outcomes.

Further Reading

BOAST supracondylar fractures in the humerus in children. https://www.boa.ac.uk/asset/A240155A%2DF0DD%2D4BE7%2D8C8AF7B6CC4DA795/.

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Part V

Miscellaneous

Miscellaneous

11

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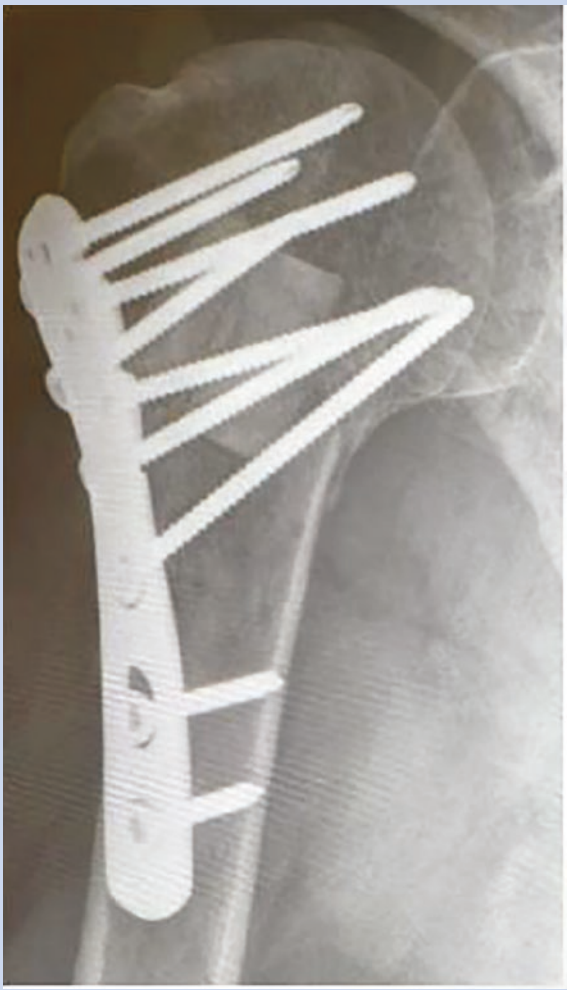
A. Mudawi et al. (eds.), *Orthopedic Board and FRCS Examination*,
https://doi.org/10.1007/978-3-031-54402-6_11

11.1 Viva 91

A 70-year-old gentleman who is a diabetic with history of trauma to the right shoulder 3 weeks ago. Over the past 2 days he has had increasing pain over the right shoulder. This is an X-ray of his affected shoulder.



- **What can you see?**
- **Would you like to know anything else from the patient?**
- **How will you proceed with evaluation of this patient?**
- **What is C-reactive protein (CRP) and what is its role in diagnosis/management of bone infection?**
- **Enumerate one other acute phase reactant that may be useful in implant-related infections.**

What can you see?
This is a plain X-ray of the shoulder joint with a proximal humeral fracture that has been treated with open reduction and internal fixation with proximal humeral plate and screws. There is a rectangular radio-opaque shadow in the metaphyseal area suggestive of bone graft or bone graft substitute. The shoulder joint appears normal.

Would you like to know anything else from the patient?
I would like to know if the fracture was open or closed; if the perioperative period was uneventful or if there were any postoperative wound issues or deep infection which had to be treated. I would also like to know if the current onset of pain was associated with any fever, night pain or swelling/redness at the site of pain. Was it preceded by any reinjury or aggressive manipulation?

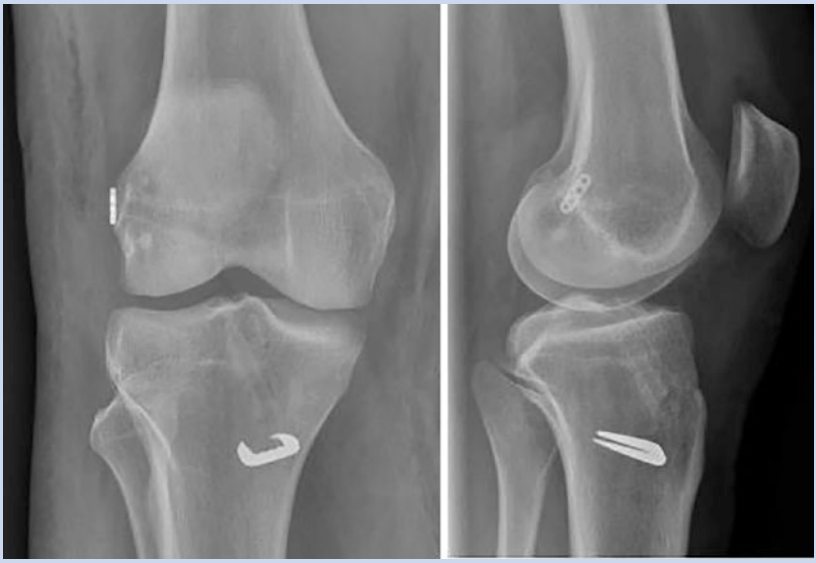
How will you proceed with evaluation of this patient?
I would request blood tests including FBC, ESR, C-reactive protein, and serum procalcitonin to exclude possible infection.

What is C-reactive protein (CRP) and what is its role in diagnosis/management of bone infection?
C-reactive protein is an acute phase reactant that usually peaks in 48 h after surgical intervention and returns to normal in 2–3 weeks. A CRP value of <10 mg/mL is considered normal. Elevated CRP contributes to a diagnosis of infection in this setting. It has a reported sensitivity of 96% and specificity of 92% in bone infections.

Enumerate one other acute phase reactant that may be useful in implant-related infections
IL-6 is produced by activated macrophages and monocytes. IL-6 is a better predictor of periprosthetic infections.

11.2 Viva 92

A 20-year-old university football player sustained an injury to his knee during a pre-season friendly match. Following the injury, he had acute effusion of the knee and was carried off the field as he was unable to continue the game. This is his knee X-ray following treatment.



- **Comment on the radiograph.**
- **What are the injuries associated with ACL tear and how would you diagnose them?**
- **What are the stages of rehabilitation of isolated ACL reconstruction?**

Comment on the radiograph

A/P and lateral radiographs of the knee show cortical suspensory fixation implants in place on the femoral side and ligament staple on the tibial side following ACL reconstruction. The femoral and tibial tunnels appear to be properly positioned. There are no other bony injuries.

What are the injuries associated with ACL tear and how would you diagnose them?

Medial collateral ligament, medial meniscus, lateral meniscus, and osteochondral lesions are the common associated injuries. I would request for plain MRI of the affected knee with routine T1, T2, and fat suppression sequences and oblique ACL cuts to confirm diagnosis and rule out associated injuries.

What are the stages of rehabilitation of isolated ACL reconstruction?
Ideally, I would like to commence with pre-habilitation and preoperative assessment including general health status and workplace/leisure activities. I would expect functional range of movement prior to surgical intervention. Postoperative expectations and patient role would also need to be discussed. Initial rehabilitation or inpatient rehabilitation would aim to

- Make the patient independently ambulant with weight bearing as tolerated with walking aids.
- Knee flexion to 90 and full extension.
- Independent with home exercise program.

0–2 weeks rehabilitation program

- Knee movements from 0° to 120°.
- Full weight bearing with walking aid.
- Knee effusion control.
- Good quadriceps activation.

2–6 weeks rehabilitation program

- Full knee movements.
- Well-controlled knee effusion after activities.
- Weaning off walking aids.
- Symmetry in stair climbing.

Recovery rehabilitation phase 6–12 weeks

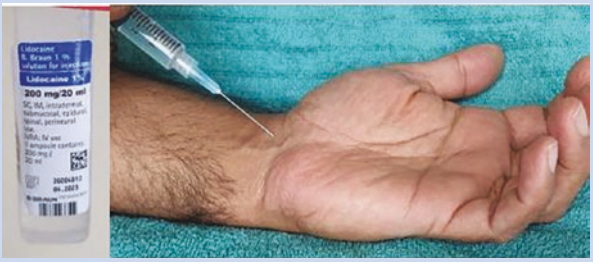
- Equal proprioception on single-leg stance on both sides.
- Adequate hamstring, hip abductors, hip adductors, and gastrocnemius strength.

Final rehabilitation phase 12 weeks to 1 year

- 85–100% leg symmetry index of knee extensors.
- Single-leg press relative strength index >125%.
- Symmetry on hop test.
- Graded return to sport if that was identified as a final goal.
- No contact sports for 6 months.
- Long-term maintenance program.

11.3 Viva 93

A 55-year-old gentleman presented with burning pain in the hand and fingers especially on holding objects for prolonged periods and at night.



- **What is the probable diagnosis and what is the definitive treatment?**
- **What are the types of anesthesia for upper limb surgical procedures?**

What is the probable diagnosis and what is the definitive treatment?
Carpal tunnel syndrome. If the patient fails non-operative treatment such as night splints, open/endoscopic carpal tunnel release would be indicated.

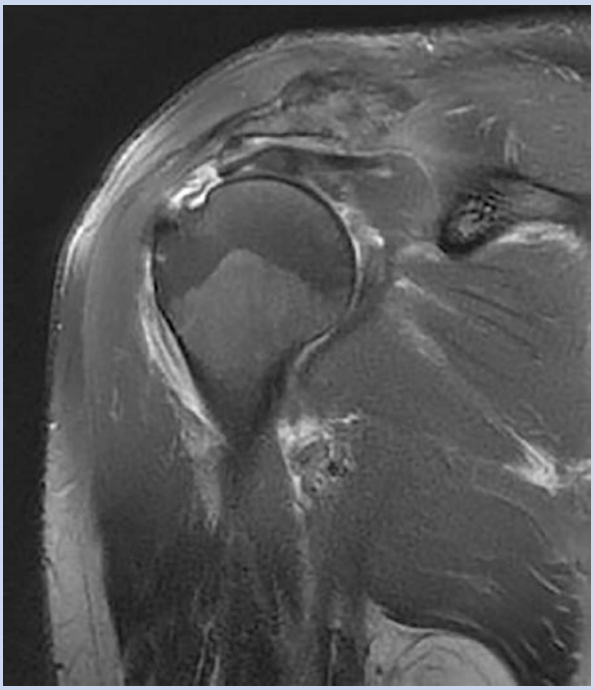
What are the types of anesthesia for upper limb surgical procedures?
General and regional anesthesia.

Regional anesthesia can be further categorized as

- Interscalene block.
  - Commonly for shoulder, upper arm, and elbow surgeries.
- Supraclavicular block.
  - Commonly for surgeries of lower arm to the hand.
- Infraclavicular block.
  - For surgeries of lower arm to the hand.
- Axillary block.
  - Usually for post-operative analgesia from elbow to hand.
- Bier block.
  - For short procedures of the forearm to hand, not exceeding 60 min.
  - Requires use of Esmarch exsanguination followed by inflation of arm tourniquet and administration of intravenous lidocaine.
  - Tourniquet should be released only after 30 min to avoid potential LAST (local anesthetic systemic toxicity).

11.4 Viva 94

A 58-year-old gentleman presents to clinic with pain in the shoulder predominantly over the deltoid insertion. He has no history of trauma to the shoulder. Read the MRI image given below.



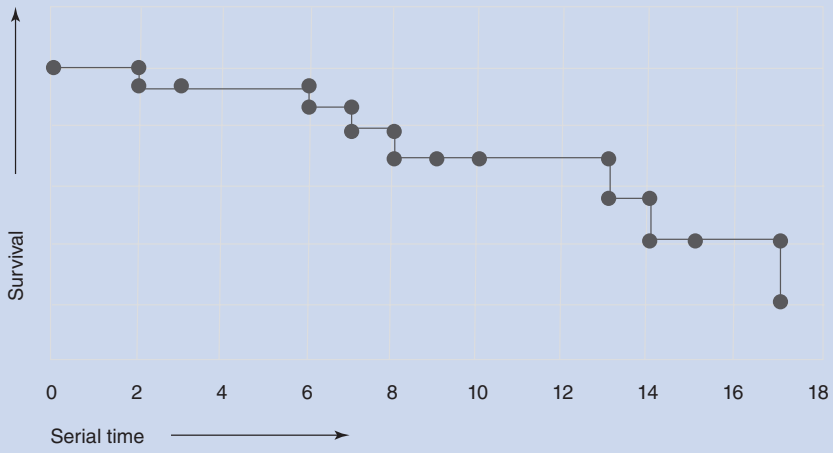
- **How would you manage this patient?**
- **Proceeding with operative management, what is your plan? Justify.**
- **Elaborate on UKUFF trial.**

How would you manage this patient?
This is a T2-weighted MRI image featuring a coronal cut of the shoulder joint, revealing a rotator cuff tear. I would review the indications for non-operative management versus operative management in this patient considering age, risk of tear propagation, healing potential on repair and quality of muscle.

Proceeding with operative management, what is your plan? Justify
After a detailed discussion with the patient regarding the pros and cons of operative management, I will proceed to arthroscopic repair of the torn rotator cuff. Open repair is also equally acceptable according to UKUFF trial.

Elaborate on UKUFF trial
It was a multicentric trial involving 19 hospitals in the UK. The objective was to evaluate the clinical and cost-effectiveness of arthroscopic and open rotator cuff repair for degenerative rotator cuff tendon tear in patients who are 50 years or older. It was a randomized trial with two parallel groups. Oxford shoulder score was the primary outcome measure at 24 months. An MRI was done at 24 months to assess the integrity of the repair. The study concluded that there was no clinical or cost benefit in choosing one treatment over the other. However, the re-tear rate was quite high in both open and arthroscopic groups at 38% and 46% respectively.

11.5 Viva 95



- **What does the above graph convey?**
- **Give an example of its use in orthopedics.**

What does the above graph convey?
This is an example of a Kaplan-Meier survival analysis plot. It shows the relationship between a predefined event to time.

Give an example of its use in orthopedics
This type of analysis is typically used in survival analysis of prosthetic implants. What is the meaning of censored data and how is it represented on this type of graph? When a subject in the study has either completed the duration of the study without having exposure to the "event" under analysis or has dropped out of the study due to other reasons, such data is called censored data. This is represented as tick marks/dots on the horizontal line.

Discussion:

Kaplan-Meier plots and survival analysis: Kaplan-Meier analysis is usually used to analyze the "time-to-event" in real-world situations where the entry point and follow-up may be different for different subjects and all the subjects may not complete the study. The "event" that is being looked at as the endpoint should be defined in the beginning of the study. For example, the "event" in survival analysis for a hip prosthesis may be revision of the implant for any indication. Each subject in the study may meet one of three outcomes.

1. The subject may complete the study and the event may not have occurred. (Terminal censoring).
2. The subject may not complete the study and drop out due to reasons not linked to the event being studied. Example: Death due to other causes, lost to follow up, study data not available. (Within study censoring).
3. The event has occurred in the subject.

   Total survival time to the event for subjects in 1 and 2 given above cannot be determined and these subjects are "censored" from the study. They do not have a bearing on the analysis of survival. However, it is important to consider them in the final evaluation. Hence, the censored patients are marked with a tick mark on the K-M plot.

   The subjects in a K-M survival analysis have three characteristic variables which are studied.

   (a) The time duration that was spent in the study until the event occurred or the subject was censored. This is called the serial time.
   (b) Status at the end of the study—whether event occurred, or subject was censored.
   (c) Study group in which the subject is placed.

   Reading the K-M Plot

   The lengths of horizontal lines indicate the duration of survival prior to the occurrence of a predefined event.

   Once an event occurs, the interval is terminated (vertical line), and the next interval begins. Censored data is marked with a tick mark on the plot, but it does not terminate the interval. The vertical distances indicate the change in cumulative probability as the curve progresses.

   Comparison between K-M curves of two interventions to study differences in outcomes between the two is usually done using log rank test. This method calculates chi-square for each event time and the summed chi-square gives a comparison of the whole graphs of both groups. An alternative method for comparison of K-M curves is the use of hazard ratio. This method also compares the two whole graphs and gives a relative probability of the event between groups of intervention. For example, comparison of K-M plots of two hip implants with endpoint of revision as the pre-defined event, will give a comparison of the likelihood of the event occurring with either implant.

11.6 Viva 96

A 45-year-old male patient, presented with open fracture right femur following motor vehicle accident. His blood pressure was 90/69 mmHg with pulse rate of 120/min.

- **How would you manage this patient?**
- **The patient responds to fluid resuscitation and his initial hemoglobin is 9 mg/dL. However, after 2 h, his blood pressure dropped again, and his hemoglobin is now 7 mg/dL. What would you do?**

- **The nurse noticed that the patient has a card indicating his affiliation with the Jehovah's Witness faith and that he denies consent for transfusion of blood and blood products. She raises this issue with you when you request blood transfusion. How would you manage this patient?**
- **The patient then loses consciousness and becomes unresponsive to fluid resuscitation and his hemoglobin has dropped to 4 mg/dL. How would you address this situation?**

How would you manage this patient?
I would manage this patient according to ATLS protocol proceeding with primary survey checking the Airway, Breathing, Circulation, Disability and Exposure [ABCDE].

I noticed that the patient is hypotensive and tachycardic indicating a class 3 hemorrhagic shock status according to ATLS classification. I would ensure two wide-bore intravenous cannulae for resuscitation, start crystalloid infusion and send blood for type and cross-match.

The patient is responding to fluid resuscitation and his hemoglobin returned to 9 mg/dL. However, after 2 h his blood pressure dropped again, and his hemoglobin dropped to 7 mg/dL. What would you do?
I would check locally for any ongoing bleeding, arrange for blood transfusion, and prepare the patient for surgical intervention.

The nurse noticed that the patient has a card indicating his affiliation with the Jehovah's Witness faith and that he denies consent for transfusion of blood and blood products. She raises this issue with you when you request blood transfusion. How would you manage this patient?
Firstly, I would check the card reviewing the patient's identifiers to make sure that it is his card. I would then go through the details of products he refuses to receive and if the refusal is valid even in life-threatening situations. Acknowledging the patient's desire, I would withhold the blood transfusion and discuss the situation with him and convey the need for blood transfusion in this particular situation.

The patient then loses consciousness and becomes unresponsive to fluid resuscitation and his hemoglobin drops to 4 mg/dL. How would you address this situation?
Since this is potentially life-threatening, I would discuss the need for blood transfusion with the patient's relatives.

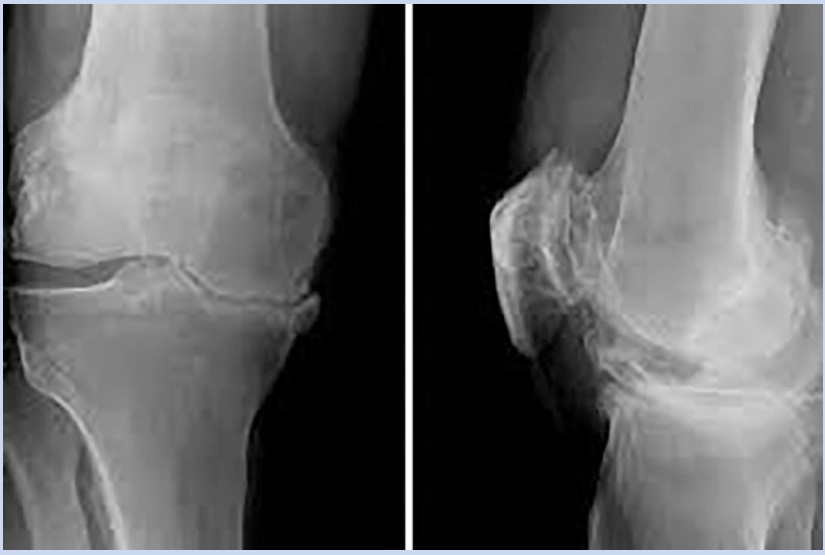
If relatives are not present in life-threatening conditions, a decision can be taken based on the clinical judgment of the responsible physician based in the best interest of the patient.

The local Hospital Liaison Committee for Jehovah's Witnesses (HLC) may be able to guide patients on appropriateness of certain treatment options and help in decision-making.

General Medical Council guidelines clearly state that emergency treatment to save life or prevent deterioration can be provided even in the absence of consent. The Royal College of Surgeons guidelines maintain that in such circumstances, "Every effort should be made to avoid the use of blood and blood products. However, in serious or life-threatening situations the use of blood and blood products should be based on the judgment of the clinician responsible."

11.7 Viva 97

A 65-year-old female patient presents with complaints of right knee pain affecting her activities of daily living and not responding to conservative measure for the last 18 months.



- **Describe the radiograph and what is the suggested treatment?**
- **How would you consent this patient for a total knee replacement?**
- **You failed to inform the patient about the risk of allergic reaction to metal. Despite being a rare complication, the patient developed metal allergy. How would you interpret this considering the Montgomery case?**

Describe the radiograph and what is the suggested treatment?
The radiograph shows advanced right knee tricompartmental osteoarthritis. This patient would benefit from total knee replacement.

How would you consent this patient for a total knee replacement?
I would obtain written informed consent from this patient. The following should be explained giving the patient sufficient time to understand the procedure and to address any questions:

- The nature of the disease should be explained in a language the patient understands:
  Osteoarthritis is a degenerative disease that involves the articular surface of the joints, namely the lining of the bone ends which we call hyaline cartilage, leading to pain and deformity affecting the activities of daily living of the patient.
- The available options of treatment should be discussed:
  Trial of pain control medications along with physical therapy sessions aimed at muscle strengthening and range of motion exercises along with lifestyle modifications are the first line of treatment. Following this, invasive measures such as intra-articular injections can be tried. Since these measures failed to reach acceptable function and pain control in your case, surgical option should be considered.
- Explain the suggested treatment:
  - What we intend to undertake and achieve:
    Total knee replacement is a surgical procedure where we shave the damaged cartilage from the bone surface along with part of the underlying bone and replace that with metal, fixed to the bone with medical cement. A polyethylene piece is then inserted between the two metal surfaces. The aim of this procedure is to give you a stable painless knee with functional range of motion.
  - Implant specifications.
    The metal we use is made of Chrome-Cobalt alloy. The spacer we use is made of a medical plastic called ultra-high molecular weight polyethylene. This is treated to withstand wear.
    The brand used will be according to the trust policy and its track record in NJR.
    Cost according to the trust policy.
  - Preoperative preparation.
    Before surgery we need to make sure that your body is fit to undergo the surgery. In order to ascertain that we need to do:
    - Blood investigations (blood counts, renal function).
    - Have the anesthetist assess you for fitness.
    - Dental checkup.
    - Preoperative physiotherapy to prepare for postoperative rehabilitation.

- Postoperative course:
  Expected number of days of stay in hospital.
  Removal of sutures.
  Clinic follow-up.
  Postoperative rehabilitation.
- Risks and complications: (with percentages).
  Surgical complications.
  - Intra-operative (excessive bleeding, fracture, neurovascular injury, cement complications).
  - Postoperative (stiffness, malunion, deformities, metal/plastic allergies).

  Death—This rare complication can happen due to any of the mentioned complications during anesthesia, during surgery, or in the postoperative period.
  Anesthesia.
  - Type.
  - Alternatives.
  - Anesthetist identifiers (name, second anesthetist, qualifications).
  - Complications.

  Aborting the procedure/surgery.
  - Unexpected medical complication.
  - Technical issues: Instruments, OT machines.
  - Surgeon related mishaps.

You failed to inform the patient about the risk of allergic reaction to metal. Despite being a rare complication, the patient developed metal allergy. How would you interpret this considering Montgomery case?

Before Montgomery, a doctor's duty to warn patients of risks was based on whether they had acted in line with a responsible body of medical opinion. This was known as the Bolam test.

Now, the doctor is under a duty to take reasonable care to ensure that the patient is aware of any material risks involved in any recommended treatment and of any reasonable alternatives or variants of the suggested treatment.

The test of materiality is whether, in the circumstances of the particular case, a reasonable person in the patient's position would be likely to attach significance to the risk, or the doctor is or should reasonably be aware that the particular patient would be likely to attach significance to it.

Points to consider.

- *Whether a risk is material doesn't only depend on how frequently it occurs.*
- *Your advisory role involves talking to the patient to make sure* ***they*** *understand the risks and benefits of their treatment, so that they can make an informed decision.*
- *Simply providing the information or getting a signature on a consent form may not be enough to evidence proper consent but can be helpful as part of the consent process.*

"Nadine Montgomery's son was born with cerebral palsy as a result of shoulder dystocia during birth.

Mrs. Montgomery was around five feet tall, and was also diabetic, which often results in a larger fetus. She had raised concerns that her baby might be too big to be delivered vaginally but had not asked about 'exact risks'.

Evidence showed a 9–10% risk of dystocia where a diabetic woman gives birth via *vaginal delivery, but Mrs. Montgomery wasn't warned of the risk of shoulder dystocia or offered a caesarean section as an alternative.*

The treating obstetrician felt that if Mrs. Montgomery was told of the risk she would opt for a caesarean and didn't believe this was in her best interest.

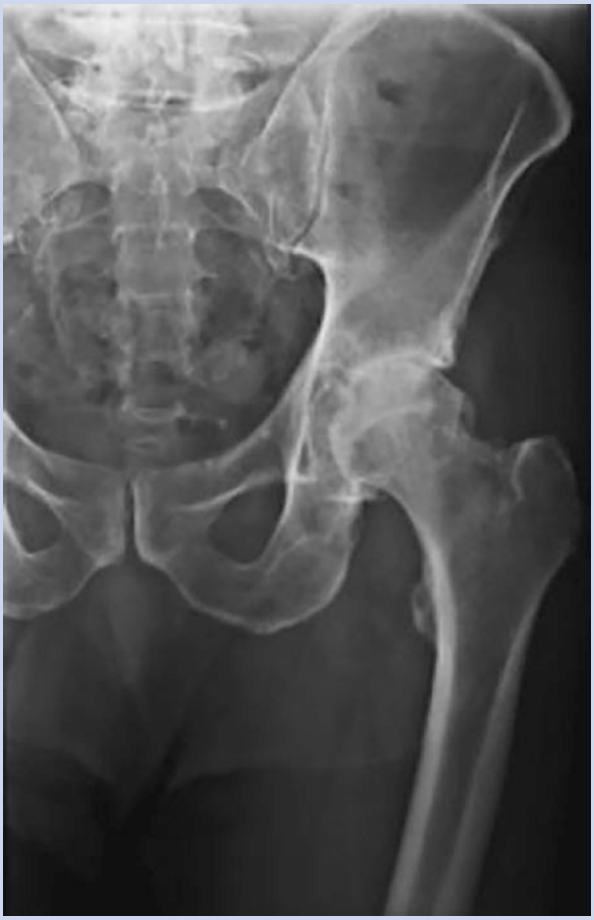
It was accepted that shoulder dystocia can cause serious complications for mother and baby but also accepted that the risk of cerebral palsy was low, at around 0.1%.

Mrs. Montgomery claimed for negligence, arguing she should have been told of all the risks.

She was awarded over £5 million in damages, after an appeal went to the Supreme Court."

11.8 Viva 98

A 64-year-old male patient presented with left hip pain affecting activities of daily living.



- **Knowing he failed his conservative measures, what would you offer him?**
- **What type of total hip replacement would you offer him and why?**
- **In the case of total knee replacement, which knee system would you use and why?**

Knowing he failed his conservative measures, what would you offer him?
This is an anteroposterior radiograph of the left hip showing advanced osteoarthritis. I think this patient would benefit from total hip replacement.

What type of total hip replacement would you offer him and why?
I would use a hybrid total hip replacement with "Exeter cemented stem and Trident uncemented cup".

- It is a hip system that provides me with the ability to deal with anatomical variations and achieve all the primary technical goals of THR.
- My training has been with Exeter, and I am familiar with the instrumentation.
- It is a system that allows me to incorporate my philosophy.
- It is a system with a proven track record for long-term survival studies and patient-reported outcome measures. It has a 15A* ODEP rating. It is also widely used in the UK according to the current NJR data (NJR 2023). In terms of bearing surfaces, I would like to use ceramic on polyethylene due to significant better outcome on long-term studies.
- The design rationale of Exeter is as follows.
  - Exeter femoral stem is highly polished, collarless, and double taper. This allows the femoral component to subside with cement creep, maintaining hoop stresses along femoral bone and maintaining bone stock by avoiding stress shielding. The centralizer/subsidizer aids in this.

ODEP = Orthopaedic Data Evaluation Panel
Independent panel of experts in UK.

ODEP provides the NHS with an approved list of prostheses that meet the revision rate standard at 10 years as set out in NICE guidelines and are suitable for use in primary hip and knee replacement.

The ***number*** represents the number of **years** for which the product's performance has been evidenced.

13: ***13*** years of evidence.
10: 10 years of evidence—**full compliance with NICE benchmark**.
7: 7 years of evidence—product is on-track to achieve the 10-year benchmark, but has not yet got sufficient data to evidence performance at 10 years.
5: 5 years of evidence—product is on-track to achieve the 10-year benchmark, but has not yet got sufficient data to evidence performance at 10 years.
3: 3 years of evidence—product is on-track to achieve the 10-year benchmark, but has not yet got sufficient data to evidence performance at 10 years.

The letter represents **the strength of evidence (data)** presented by the manufacturer.

A strong evidence—generally higher numbers of patients (giving greater confidence in the results presented), with all patients being subject to follow-up (their outcomes recorded).
B acceptable evidence—smaller numbers of patients than the A rating (giving less confidence in the results than A), but sufficient data to demonstrate compliance.

The ***star*** has been added to the rating system following revised guidelines from **NICE in February 2014, in which a benchmark replacement rate of less than 1 in 20 (5%) at 10 years** was defined. The star is awarded where products are evidenced to comply with this benchmark. A* represents very strong evidence above A and B. Ratings without a star signify compliance to the prior NICE guidance of a replacement rate of less than 1 in 10 (10%) at 10 years.

Pre-entry—Products that are registered with ODEP for which there is insufficient data to evidence the 3-year entry benchmark have been listed as "pre-entry", showing products currently undergoing clinical evaluation. Products not under Beyond Compliance, but which are registered with the National Joint Registry.

Pre-entry A*—Products are being evaluated through the Beyond Compliance initiative, a post-market surveillance service supported by ODEP.

Beyond Compliance: A service to support the safe and stepwise introduction of new or modified implants such as joint replacements.

Uses available data to monitor the early performance of new brands of THR/TKR that come within the Beyond Compliance service.

Any potential problems relating to a product, or its surgical use can be identified before large numbers have been implanted and with the agreement of the manufacturer, to reduce the likelihood of wide dissemination of implants before any weaknesses are identified in real-world use.

In the case of total knee replacement, which knee system would you use and why?

- I would use a knee system which
  - Covers anatomical variations (varus, valgus).
  - Incorporate stem/augment trays for complex knee problems.
  - Allow to incorporate my philosophy of balancing the knee.
  - Has proven track record for long-term survival studies and patient-reported outcome measures.
  - Has a high ODEP rating.

- I would use Triathlon, CR fixed bearing cemented knee system.
  - ODEP 15*A with 15-year revision rate of 3.72 at 15 years (NJR 2023).
  - Or PFC Sigma—ODEP 15*A with 3.17% revision rate at 15 years (NJR 2023).

11.9 Viva 99

- **Some patients have mentioned to you that your colleague smelt alcohol in the clinic more than once in the last 2 weeks. Today you noticed him coming drunk to work. What would you do?**

Some patients have mentioned to you that your colleague smelt alcohol in the clinic more than once in the last 2 weeks. Today you noticed him coming drunk to work. What would you do?

My first concern would be the safety of the patient. However, I have a duty of care to my colleague and the hospital too.

Patient safety—I will talk to my colleague and send him home (if difficult, I would enlist the help of another consultant or clinical director). I will review all the patients seen by him and complete the ward round. I will also recall all the patients discharged by him. I will ensure that appropriate cover is arranged for him, if needed.

Duty of care to colleague—I will arrange a taxi for him to go home and check on him later, to ensure that he has reached home safely. I will have a discussion with him (if a consultant, I will pass on the incident to the clinical director) and try to help. Occupational health referral is another option. I would insist that his behavior was not appropriate.

Duty of care to the hospital—Keep accurate records and inform your consultant or clinical director.

Where concerns about performance have arisen, it may be helpful, at any stage of the process, to consider why this has happened. THINK ABOUT:

The individual's health and other factors:

- Does the individual have a physical or mental illness?
- Is the individual depressed or suffering other mental illness?
- Might alcohol or substance misuse be involved?
- Has there been a recent major life event?

Knowledge, skills, and behavior

Is there a difficulty with clinical knowledge and skills?

Might a deficiency in education, supervision, or continuing professional education be contributing to the problem?

- Was the practitioner's induction appropriate or sufficient?
- Does the individual have difficulty understanding the limits of their competence?

- Is the problem predominantly one of the practitioner's behavior or attitude?
- Is this new behavior or is it an exacerbation of long-standing problems?

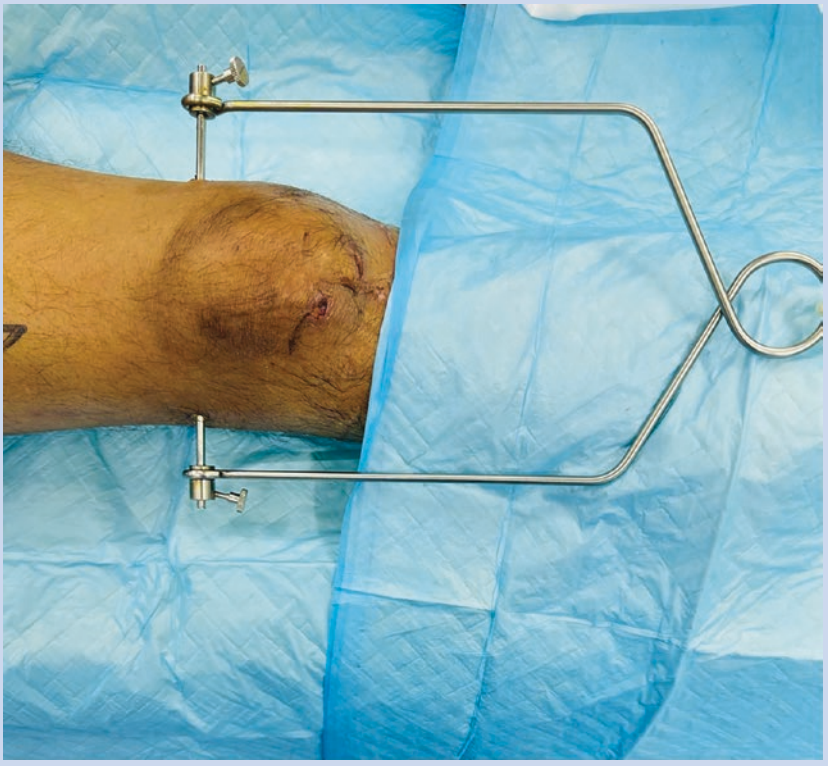
The job

- Have work factors changed?
- Is there a problem with technological advances or techniques?

The work environment

- Are there team difficulties?
- Have there been major organizational changes?
- Could issues relating to equality and diversity be a problem?
- Could bullying or harassment be a problem?
- Are there any systems issues that contributed to the performance difficulty?

11.10 Viva 100



- **What can you see?**
- **What are the principles of skeletal traction?**
- **What are the common sites for skeletal traction application and its anatomical marking?**
- **What are the possible complications of skeletal traction?**

What can you see?
This is a clinical photo showing the patient with left distal femur skeletal traction.

What are the principles of skeletal traction?
Skeletal traction is a form of traction used in orthopedics by driving a metal pin or wire through the bone to counteract the effect of the deforming forces produced by the muscle spasm and gravity. It aims to reduce and realign the deformity. Moreover, it helps to control movements, relieve pain, and aid in bone healing.

Instruments used:

- Steinmann pin (smooth pin) 4–6 mm.
- Denham pin (threads in the middle) used in cancellous or osteoporotic bone.
- Bohler stirrup to attach to the pins. It allows the weight to be connected to the pin in different directions.
- K-wire (Kirschner wire) attached to K-wire strainer. The strainer is used to tighten the wire so that it can hold traction weight. This is used more in the upper limbs.
- Screw eye.

What are the common sites for skeletal traction application and its anatomical marking?
Olecranon:

3 cm distal to the tip of the olecranon perpendicular to the axis of the ulna. Avoid ulnar nerve—K.wire or screw eye.

Second and third metacarpals:

2–2.5 cm proximal to the distal end of the second metacarpal, through the second and third metacarpal perpendicular to the axis of the radius—K.wire.

Proximal femur (greater trochanter):

2.5 cm on the lateral surface of the femur, distal to the most prominent part of the greater trochanter midway between anterior and posterior femur surfaces. A cancellous screw or screw eye can be used.

Distal femur:

3 cm proximal to the tibiofemoral articulation, at the intersection of the line joining the superior pole of the patella with the line drawn proximally from the anterior edge of the fibula head. Avoid entering the knee joint.

Proximal tibia:

2 cm posterior and 2 cm distal to the tibial tuberosity, the pin driven from lateral to medial to avoid injury to the common peroneal nerve.

Note: prolonged distal femoral traction causes knee stiffness and fibrosis of the extensor mechanism. Hence it should be changed to proximal tibial traction after 2–3 weeks.

Distal tibia:

5 cm proximal to the ankle joint midway between the anterior and posterior surfaces of the tibia.

Calcaneus:

2 cm posterior and distal to the lateral malleolus or 3 cm posterior and distal to the medial malleolus. Avoid entering the subtalar joint.

Note: calcaneal traction can cause subtalar joint stiffness and possible bone infection. Use the distal tibial traction when possible.

What are the possible complications of skeletal traction?

- Infection of the bone.
- Incorrect placement of the pin/wire can result in:
  - Cut-out through the bone and pain.
  - Improper rotational alignment and control.
  - Difficult splint application.
  - Uneven pull on the pin/wire leading to movement in the bone, skin ischemia or bone necrosis which might predispose to infection.
- Fracture distraction.
- Ligament damage.
- Epiphyseal plate damage.
- Depressed scar at pin site.

Skin Traction

Skin traction: Max 6–7 kg—adhesive traction.

Skin traction: Max 4–5 kg—non-adhesive traction.

Skin traction contra-indications:

- Abrasions/Lacerations/Dermatitis.
- Impaired circulation.
- Significant shortening where skin traction weights are not enough. In this situation skeletal traction should be considered.

Complications of skin traction:

- Allergic reactions/Excoriations.
- Pressure sores.
- Nerve palsy—as in common peroneal nerve palsy in below knee skin traction.

Further Reading

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