6. Reconstruction :

A. Oral

Osteolysis : histiocytic response to wear debris including the following steps
or :
Osteolysis is a particle-induced biological process occurring at the bone-metal or bone-cement interface around total joints resulting in rapidly expanding focal lesions that may or may not cause loosening

Hip Osteonecrosis :
bone cell death caused by interruption of blood supply to femoral head leads to subchondral bone collapse and morphological / arthritic change

Idiopathic Transient Osteoporosis of the Hip (ITOH)
Rare self-limiting condition of unknown cause that leads to temporary bone loss in proximal femur

Saddle point
most distal part of the junction between the superior aspect of the femoral neck and the greater trochanter

THA Trunnionosis : wear of the femoral head-neck junction

(ALVAL), aseptic lymphocyte-dominant vasculitis-associated lesion is a mass-forming tissue reaction caused by metal-on-metal wear

Squeaking : a high pitched audible sound occurring during hip movement

"screw home" mechanism
tibial externally rotates 5 degrees in the last 15 degrees of extension

because medial tibial plateau articular surface is longer than lateral tibial plateau
leads to "lock" knee decreasing the work performed by the quadriceps while standing

The HHS is a measure of dysfunction so the higher the score, the better the outcome for the individual. Results can be recorded and calculated online. The maximum score possible is 100. Results can be interpreted with the following[1]: <70 = poor result; 70–80 = fair, 80–90 = good, and 90–100 = excellent.

Haris hip score

Variable	Points
Pain	
None or ignores it	44
Slight, occasional	40
Mild pain, rarely moderate	30
Moderate pain	20
Marked pain	10
Totally disabled, pain in bed	0
Function	
Limp	
None	11
Slight	8
Moderate	5
Severe	0
Support	
None	11
Cane, long walks	7
Cane, most of the time	5
One crutch	3
Two canes	2
Two crutches	0
Not able to walk	0
Distance walked	
Unlimited	11
Six blocks	8
Two to three blocks	5
Indoors only	2
Bed and chair	0
Stairs	
Normally without railing	4
Normally with railing	2
In any manner	1
Unable to do	0
Shoes and socks	
With ease	4
With difficulty	2
Unable	0
Sitting	
Ordinary chair for 1 h	5
High chair for 1 h	3
Unable to sit in any chair	0
Public transport	
Able to use	1
Unable to use	0

Knee Society Score -- OrthoToolKit

About the score ^

Originally published in 1989 in *Clinical Orthopaedics and Related Research*, the Knee Society Clinical Rating System (KSS) was designed to provide a simple and objective scoring system to rate the knee and patient's functional abilities before and after total knee arthroplasty.

The original KSS has a "Knee Score" section (7 items) and a "Functional Score" section (3 items). Both sections are scored from 0 to 100 with lower scores being indicative of worse knee conditions and higher scores being indicative of better knee conditions.

Supporting literature v

About the score developer v

Part 1 - Knee Score

Knee Society Score: 100 / 100

Pain

- [x] None
- [ ] Mild / Occasional
- [ ] Mild (Stairs only)
- [ ] Mild (Walking and Stairs)
- [ ] Moderate - Occasional
- [ ] Moderate - Continual
- [ ] Severe

Flexion Contracture

[x] None	[ ] 5-10°	[ ] 10-15°	[ ] 15°-20°	[ ] >20°

Extension lag

☑ None	☐ < 10°	☐ 10-20°	☐ > 20°

Total Range of Flexion

☐ 0-5°	☐ 6-10°	☐ 11-15°	☐ 16-20°	☐ 21-25°
☐ 26-30°	☐ 31-35°	☐ 36-40°	☐ 41-45°	☐ 46-50°
☐ 51-55°	☐ 56-60°	☐ 61-65°	☐ 66-70°	☐ 71-75°
☐ 76-80°	☐ 81-85°	☐ 86-90°	☐ 91-95°	☐ 96-100°
☐ 101-105°	☐ 106-110°	☐ 111-115°	☐ 116-120°	☑ 121-125°

Alignment (Varus & Valgus)

☐ 0°	☐ 1°	☐ 2°	☐ 3°	☐ 4°	☑ 5-10°
☐ 11°	☐ 12°	☐ 13°	☐ 14°	☐ 15°	☐ Over 15°

Antero-posterior stability (maximum movement in any position)

☑ < 5mm	☐ 5-10mm	☐ 10+mm

Mediolateral stability (maximum movement in any position)

☑ < 5°	☐ 6-9°	☐ 10-14°	☐ 15°

Knee Society Score: 100 / 100

Part 2 - Function

Walking

☑ Unlimited
☐ > 10 blocks
☐ 5-10 blocks
☐ < 5 blocks
☐ Housebound
☐ Unable

Stairs

Stairs

- [x] Normal up and down
- [ ] Normal up, down with rail
- [ ] Up and down with rail
- [ ] Up with rail, down unable
- [ ] Unable

Walking aids used

- [x] None used
- [ ] Use of Cane/Walking stick
- [ ] Two Canes/sticks
- [ ] Crutches or frame

Knee Society Score (Function): 100 / 100

Knee Society Score

Clinician's name (or ref) ..... **Patient's name (or ref)** .....

During the past 4 weeks..... **Click here for part 2 - FunctionScore**

Part 1 - Knee Score

Pain

- ○ None
- ○ Mild / Occasional
- ○ Mild (Stairs only)
- ○ Mild (Walking and Stairs
- ○ Moderate - Occasional
- ○ Moderate - Continual
- ○ Severe

Flexion Contracture (if present)

- ○ 5°-10°
- ○ 10°-15°
- ○ 16°-20°
- ○ >20°

Extension lag

- ○ <10°
- ○ 10-20°
- ○ >20°

Total Range of Flexion

○ 0-5	○ 6-10	○ 11-15	○ 16-20	○ 21-25
○ 26-30	○ 31-35	○ 36-40	○ 41-45	○ 46-50
○ 51-55	○ 56-60	○ 61-65	○ 66-70	○ 71-75
○ 76-80	○ 81-85	○ 86-90	○ 91-95	○ 96-100
○ 101-105	○ 106-110	○ 111-115	○ 116-120	○ 121-125

Alignment (Varus & Valgus)

○ 0	○ 1	○ 2	○ 3	○ 4
		○ 5 - 10		
○ 11	○ 12	○ 13	○ 14	○ 15
		○ Over 15°		

Stability (Maximum movement in any position)

Antero-posterior

- ○ <5mm
- ○ 5-10mm
- ○ 10+mm

Mediolateral

- ○ <5°
- ○ 6-9°
- ○ 10-14°
- ○ 15°

Print page Close Windo Reset

To save this data please print or Save As CSV

Nb: This page cannot be saved due to patient data protection so please print the filled in form before closing the window.

Final Knee Score is 0

(NB: consider a negitive outcome as zero)

Click here for part 2 - FunctionScore

Grading for the knee Society Score

Score 80-100 Excellent **Score 70-79** Good **Score 60-69** Fair **Score below 60** Poor

Reference for score: Insall JN, Dorr LD, Scott RD, Scott WN. Rationale of the Knee Society clinical rating system. Clin Orthop Relat Res. 1989 Nov;(248):13-4. link to pubmed. Link SF36, SF12

Reference for Grading: Asif S , Choon DS . Midterm results of cemented Press Fit Condylar Sigma total knee arthroplasty system. J Orthop Surg (Hong Kong). 2005 Dec;13(3):280-4.

hyaluronate molecules in the synovial fluid.

OSTEOARTHRITIS

Osteoarthritis (OA) is a chronic disorder of synovial joints in which there is progressive softening and disintegration of articular cartilage accompanied by new growth of cartilage and bone at the joint margins (osteophytes), cyst formation and sclerosis in the subchondral bone, mild synovitis and capsular fibrosis. It differs from simple wear and tear in that it is asymmetrically distributed, often localized to only one part of a joint and often associated with abnormal loading rather than frictional wear.

In its most common form, it is unaccompanied by

osteoarthritis.

a. Grade 0 – Normal appearances
b. Grade 1 – Osteophytes with normal joint space
c. Grade 2 – Less than 50% joint space reduction
d. Grade 3 – More than 50% joint space reduction
e. Grade 4 – Bone-on-bone contact

Kellgren & Lawrence (based on AP weightbearing XRs)	
Grade 0	• no joint space narrowing (JSN) or reactive changes
Grade 1	• possible osteophytic lipping + doubtful JSN
Grade 2	• definite osteophytes + possible JSN
Grade 3	• moderate osteophytes + definite JSN + some sclerosis + possible bone end deformity
Grade 4	• large osteophytes + marked JSN + severe sclerosis + definite bone end deformity

Or

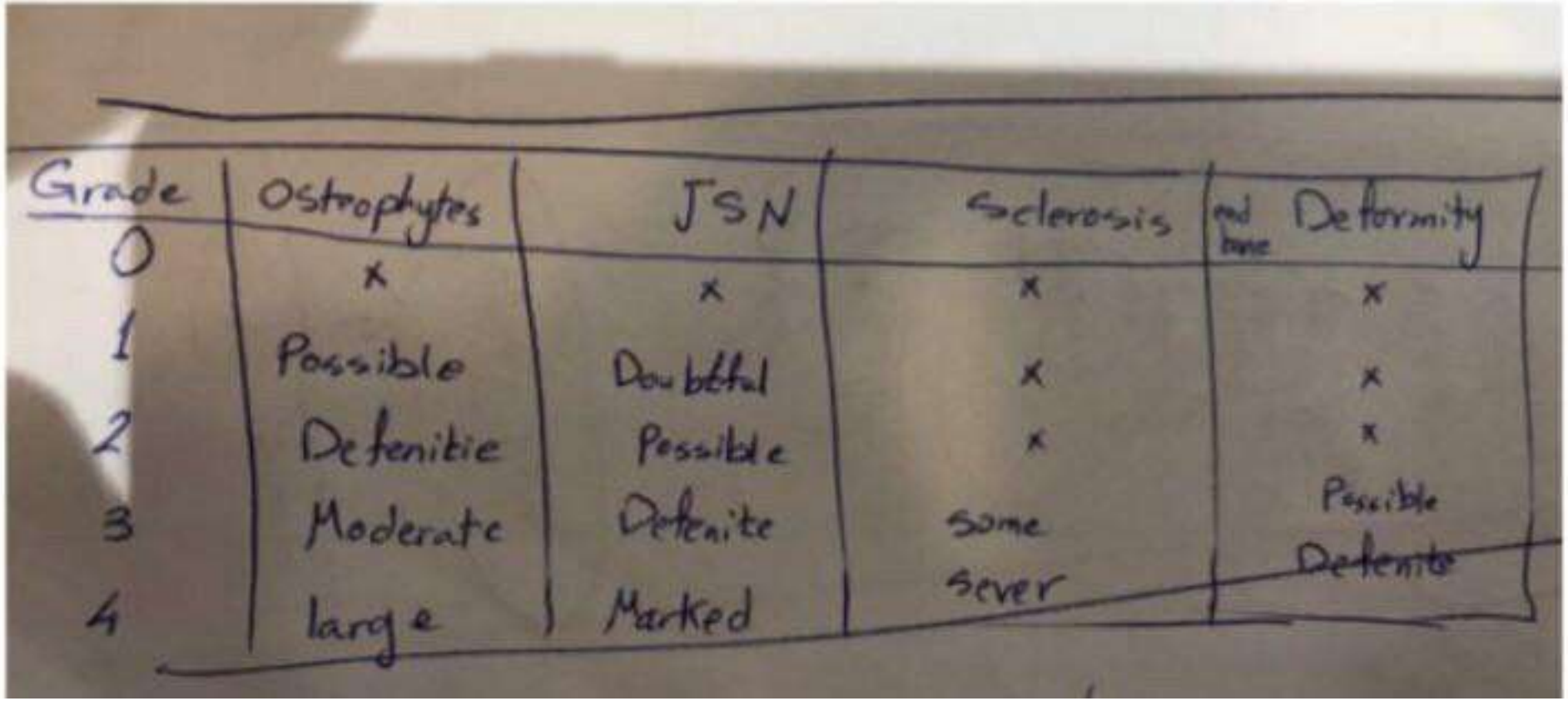


Table 22.6 Radiographic features of osteoarthritis versus rheumatoid arthritis

Osteoarthritis	Rheumatoid arthritis
Loss of joint space	Loss of joint space
Osteophytes	No osteophytes
Subchondral cysts	Marginal erosions
Bony sclerosis	Osteoperosis
Deformity and malalignment	Deformity and malalignment
Loose bodies	Loose bodies uncommon
Asymmetrical	Symmetrical
Normal soft tissue	Soft-tissue swelling

Generally OA can be subcategorized as:

1: Primary (unknown causes) and
Secondary (known causes, posttraumatic, Metabolic, etc)

2: Non-Inflammatory; Inflammatory and Infective arthritides

3: Mon-Articular and Poly-Articular

Risk Factors

Modifiable

- **Obesity (central obesity)**
- **Trauma**
- **Occupation (hard labor)**
- **Muscle weakness**
- **Metabolic syndrome**
- **Others** (Hypertension, Cardio-vascular; Diabetes ; Dyslipidemia…)

Non-modifiable

- **Gender**
- **Females at increased risk**
- **Age**
- **Genetics (Family hx)**
- **Race (some Asian populations at lower risk)**

Radiographic Findings in OA

Subchondral sclerosis

Narrowing of the joint space

Marginal osteophytes

Subchondral cysts

Squaring of the gliding surfaces; increased bone density & joint enlargement with deformity.

	Aging	Osteoarthritis
Water Content	Decreased	Increased
Collagen	Same	Disorganized
Proteoglycan Content	Decreased	Decreased
Proteoglycan Synthesis	Same	Increased
Chondrocyte Size	Increased	Same
Chondrocyte Number	Decreased	Same
Modulus of Elasticity	Increased	Decreased

Effect of Osteoarthritis

water
chondroitin sulfate
proteoglycan synthesis
proteoglycan degradation
(more degradation than synthesis)

collagen content
proteoglycan content
keratin sulfate
modulus of elasticity

Effect of Aging

keratin sulfate
modulus of elasticity
chondrocyte size

water
proteoglycan content
chondrotin sulfate
chondrocyte number

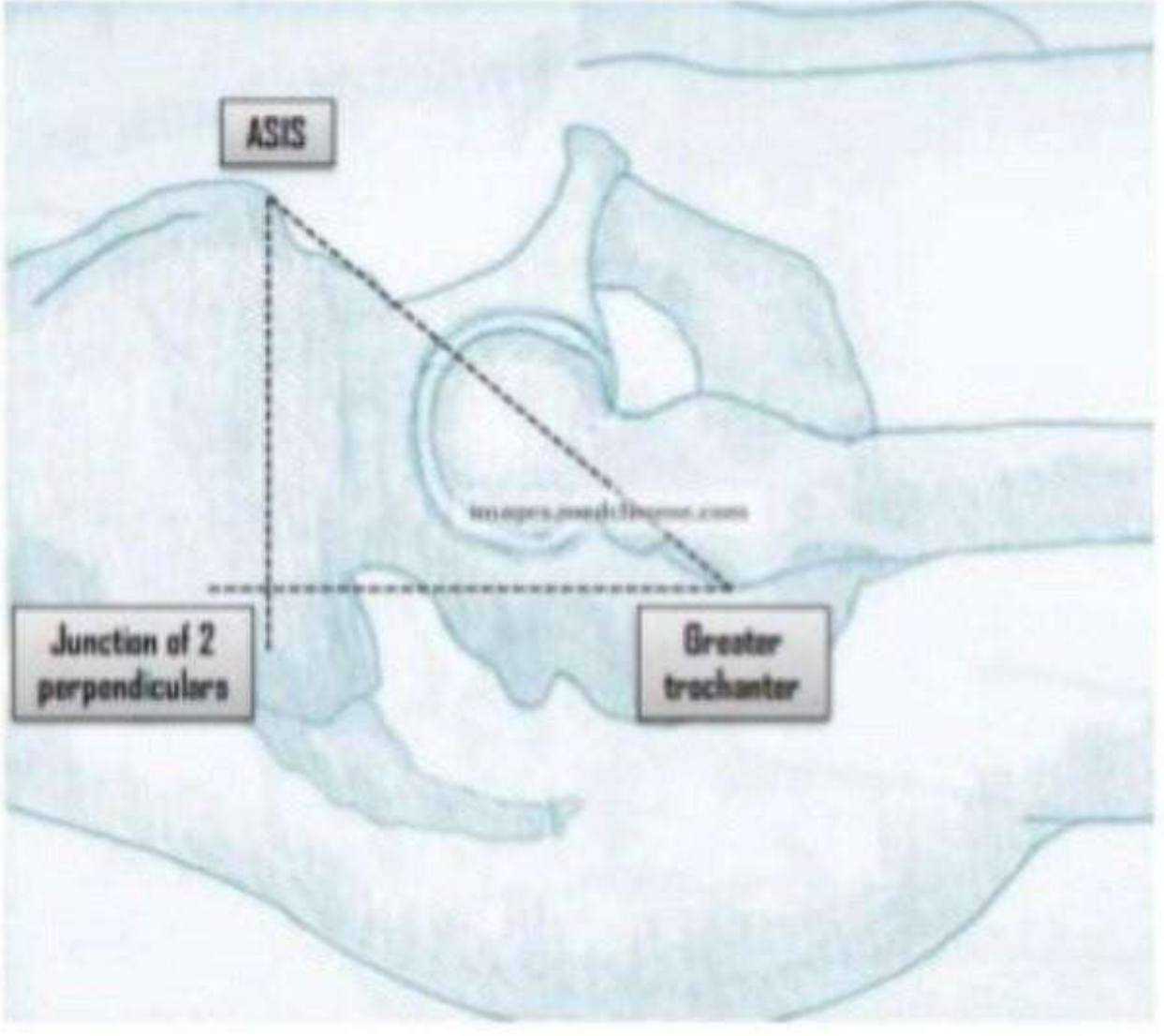
With aging : (Increase keratin)

An increase in the production of chondroitin 6 sulphate

A decrease in the production of **chondroitin 4** sulphate

BRYANT'S TRIANGLE.

- The patient lies in the dorsal position.
- A line is drawn vertically downwards from the ASIS.
- Another from the tip of the same spine to the tip of the greater trochanter and lastly a horizontal line is drawn from the tip of the greater trochanter to the first line.



Radiographic Analysis

- Step 1
  - assess the mechanical axis
    - draw a line of the hip-to-ankle view that shows the overall mechanical axis
      - neutral mechanical axis should bisect the center of knee
- Step 2
  - estimate magnitude of coronal deformity
    - measure the tibiofemoral angle
- Step 3
  - determine the femoral resection angle
    - difference between mechanical and anatomic axis of the femur
- Step 4
  - determine tibial bone cut
    - perpendicular to mechanical axis
- Step 5
  - assess bony defects and osteophytes
    - easiest to do on AP weight-bearing view
- Step 6
  - assess tibial slope
    - completed on lateral radiograph
- Step 7
  - assess patellar height
    - completed on lateral radiograph
    - assess for patella baja
      - this will make exposure more difficult
- Step 8
  - assess patellar shift/tilt
    - completed on skyline view of patella

Template the Femur

- Steps
  - choose appropriate implant size on lateral radiograph
    - restore posterior condylar offset
    - avoid notching
  - assess this size component on the AP to determine medial/lateral positioning and en… no overhang
    - if there is significant overhang, may have to consider downsizing

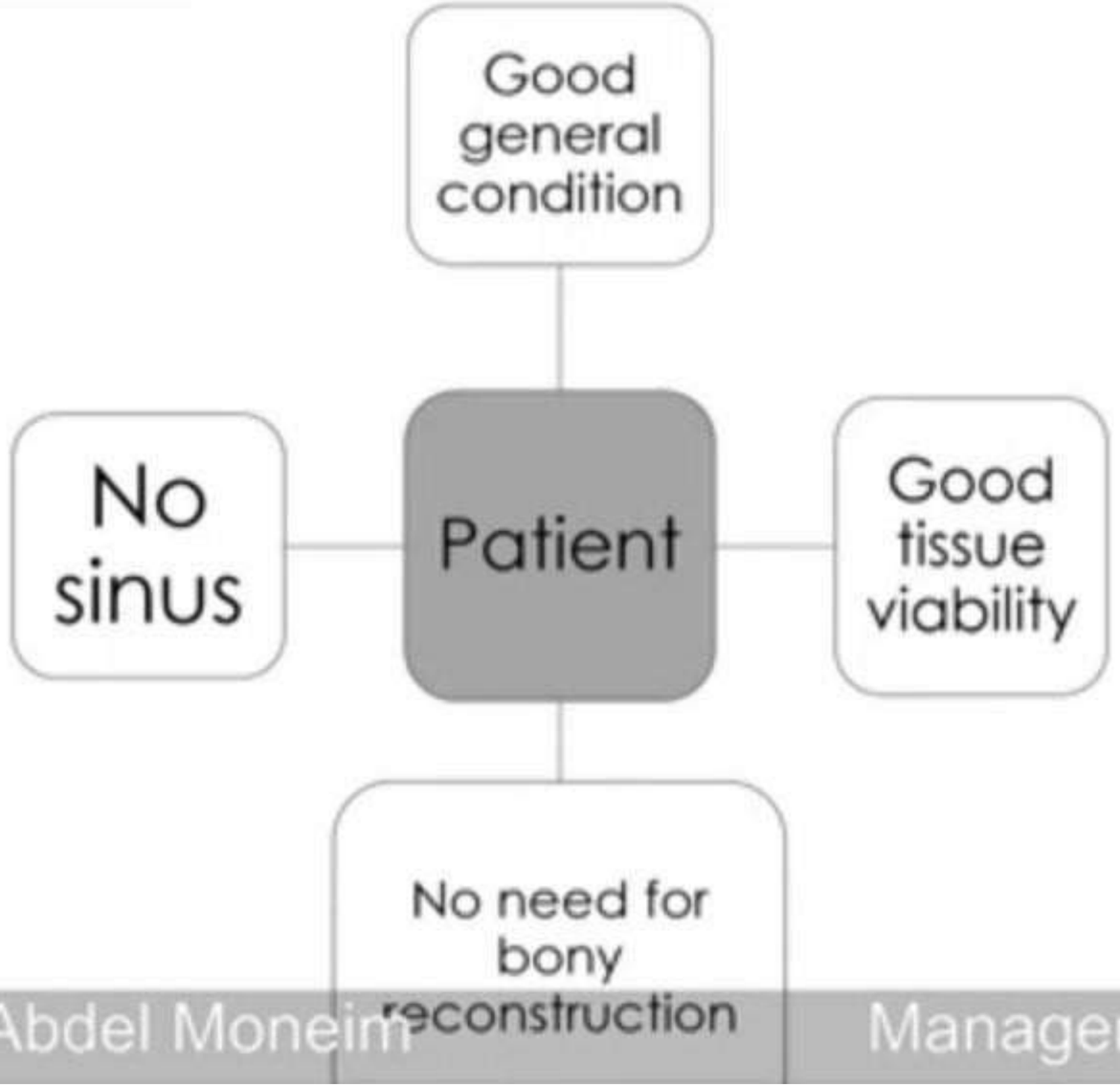
Major criteria (at least one of the following)	Decision
Two positive cultures of the same organism	Infected
Sinus tract with evidence of communication to the joint or visualization of the prosthesis	

Preoperative Diagnosis	Minor Criteria	Score	Decision
Serum	Elevated CRP *or* D-Dimer	2	≥6 Infected **2-5 Possibly Infected [a]** 0-1 Not Infected
Serum	Elevated ESR	1	
Synovial	Elevated synovial *WBC count or LE*	3	
Synovial	Positive alpha-defensin	3	
Synovial	Elevated synovial PMN (%)	2	
Synovial	Elevated synovial CRP	1	

Intraoperative Diagnosis	Inconclusive pre-op score *or* dry tap [a]	Score	Decision
	Preoperative score	-	≥6 Infected **4-5 Inconclusive [b]** ≤3 Not Infected
	Positive histology	3	
	Positive purulence	3	
	Single positive culture	2	

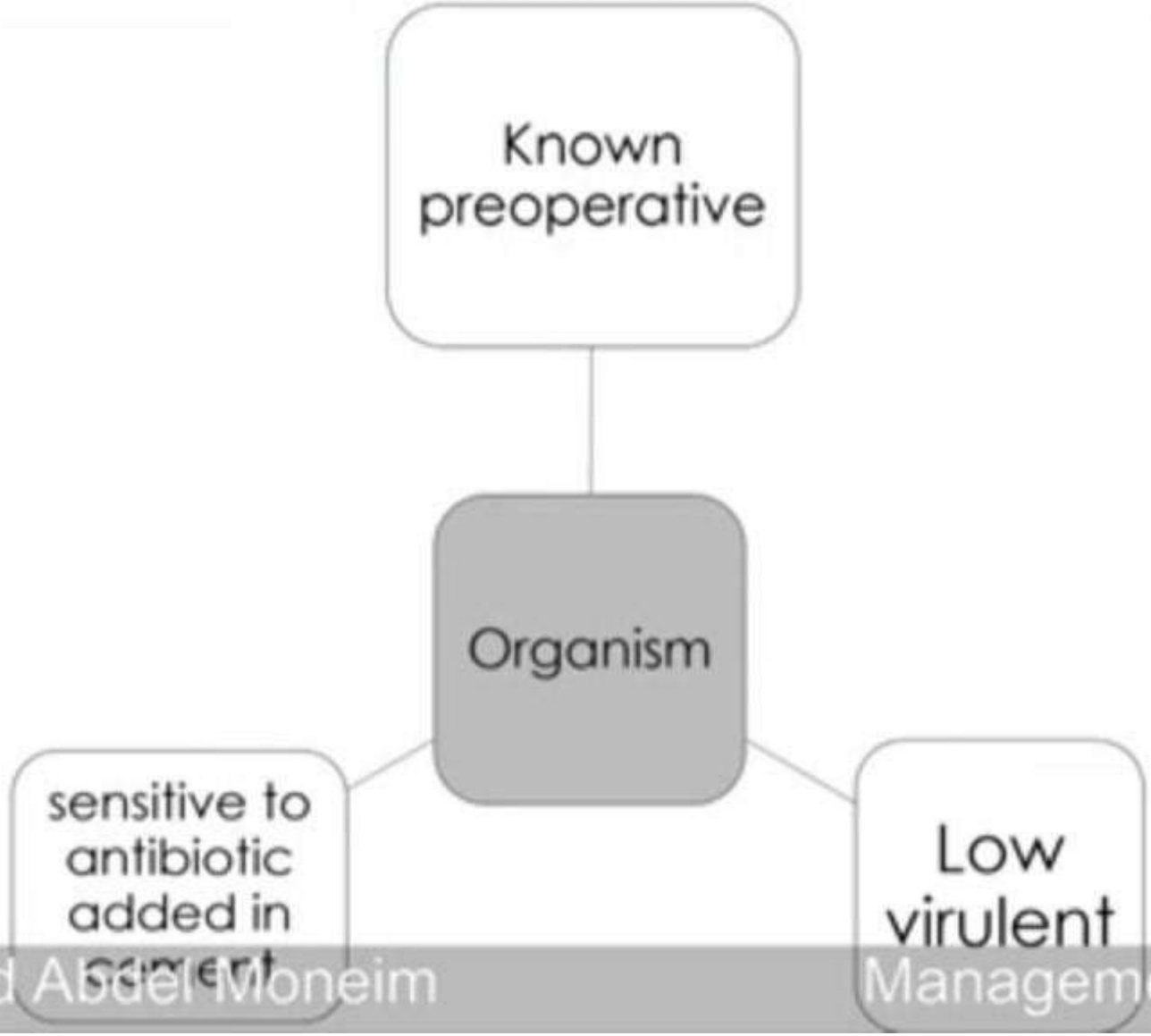
KASR ALAINY

ONE STAGE EXCHANGE ARTHROPLASTY



KASR ALAINY

ONE STAGE EXCHANGE ARTHROPLASTY



Injury severity score (ISS)	High ISS is a risk	11%
Traumatic brain injury (TBI)	Higher incidence in the spastic limbs of the patient	11%
Spinal cord injury	Complete SCI produces more HO than incomplete SCI. Cervical and thoracic SCI produces more HO than lumbar SCI. Younger age produces more HO (20-30yo). Higher incidence in the spastic limbs of the patient.	20%
Neurologic compromise	Prolonged coma in young patient (20-30yo), and prolonged ventilator use	
Other diseases	Burns (both locally under burn and remotely; more common with >20% body surface area), DISH, ankylosing spondylitis, hypertrophic osteoarthritis (prominent osteophytes)	
Decubitus ulcers	Worse with concomitant decubitus ulcers and SCI or TBI	70% (with concomitant SCI)
Antegrade femoral nail entry site	Worse with piriformis fossa entry point.	25%
Distal femur traction pins	HO in distal quadriceps. Higher incidence in patients with other concomitant injuries, use of large diameter Steinmann pins (5mm) because of hematoma, soft tissue injury from percutaneous insertion.	rare
Amputation through zone of injury	Worse with blast mechanism	63%
Surgical approaches	Extended iliofemoral > Kocher-Langenbeck > ilioinguinal approach (acetabular fracture). Anterior approach > posterior approach for femoral head fracture fixation.	25% (acetabular fracture fixation)
Total hip arthroplasty	THA Complications ▸ Increased risk with psoas tenotomy and cementless components (more particulate debris and marrow spillage, muscle trauma from difficult broaching). Smith-Petersen and Hardinge > transtrochanteric > posterior (posterior has lowest risk of HO).	53% (significant in only 5%)
Total knee arthroplasty	TKA complications ▸ Increased risk with notching anterior femur, surgical trauma to quadriceps, distal femur exposure and	

Orth Laith Trawnah.

Forwarded Message

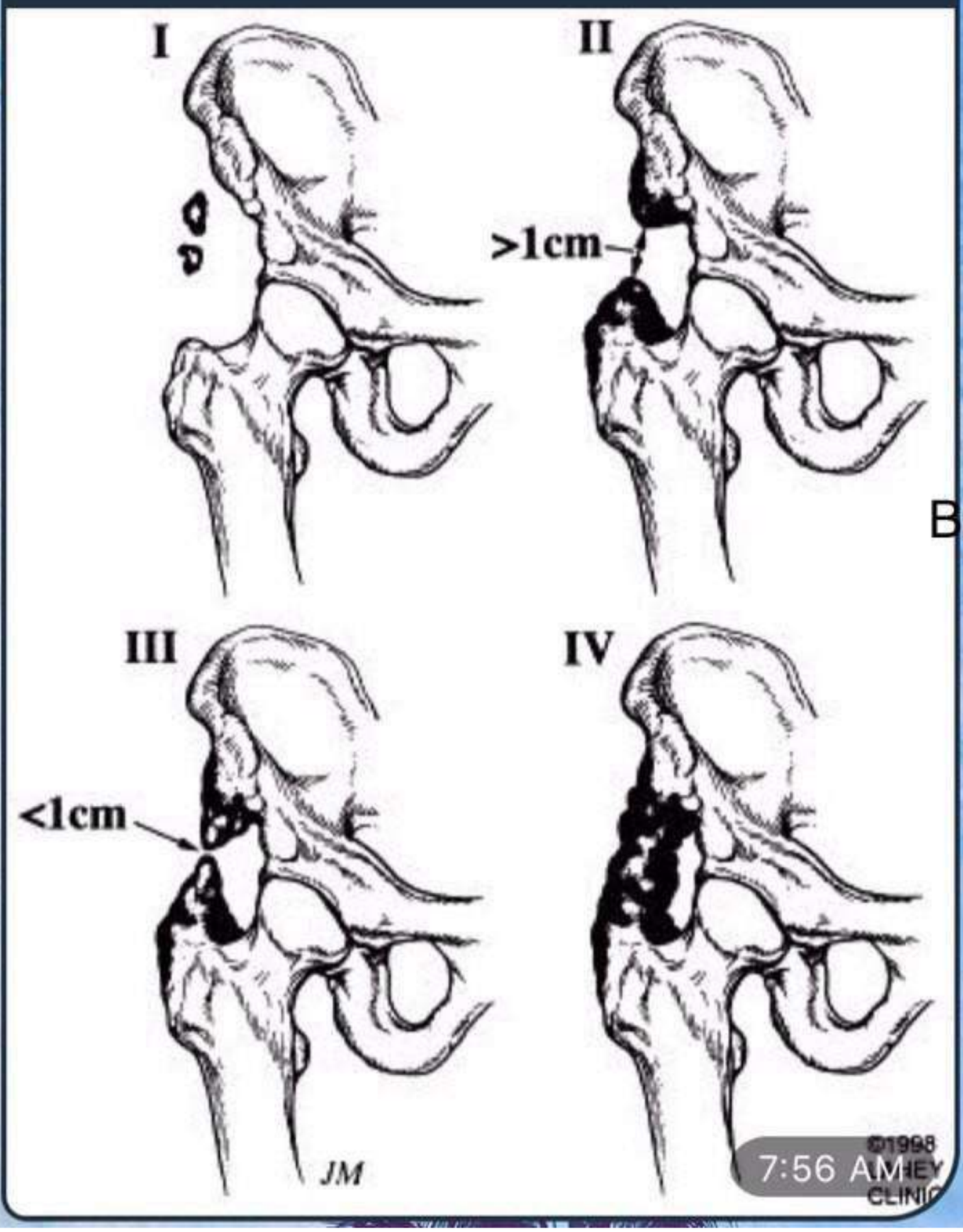
From: Orth Laith Trawnah.

Grade I	Ossification islands around the hip
Grade II	Bone projection of pelvis or proximal femur with at least 1 cm away from the opposite surface
Grade III	Bone projection of pelvis or proximal femur reducing space between opposite surface lower than 1 cm
Grade IV	Hip ankylosis

7:56 AM

Forwarded Message

From: Orth Laith Trawnah.



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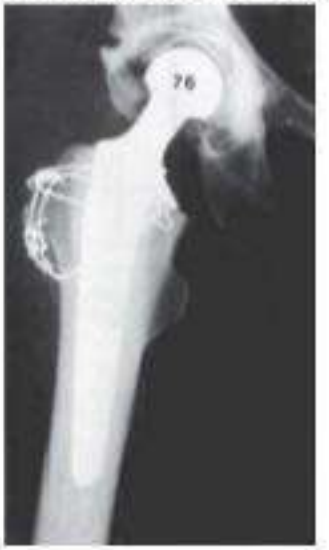
Brioker Classification

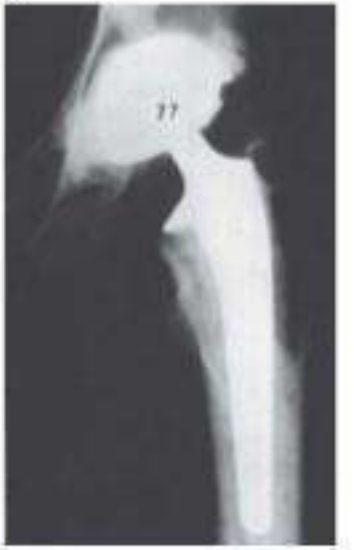
Barrack, Mulroy, and Harris grading for femoral cement mantle

1. Grade 1 :Complete filling of the medullary canal without radiolucencies ("**white-out**")
2. Grade 2 : Slight radiolucency at the bone-cement interface **(<50%)**

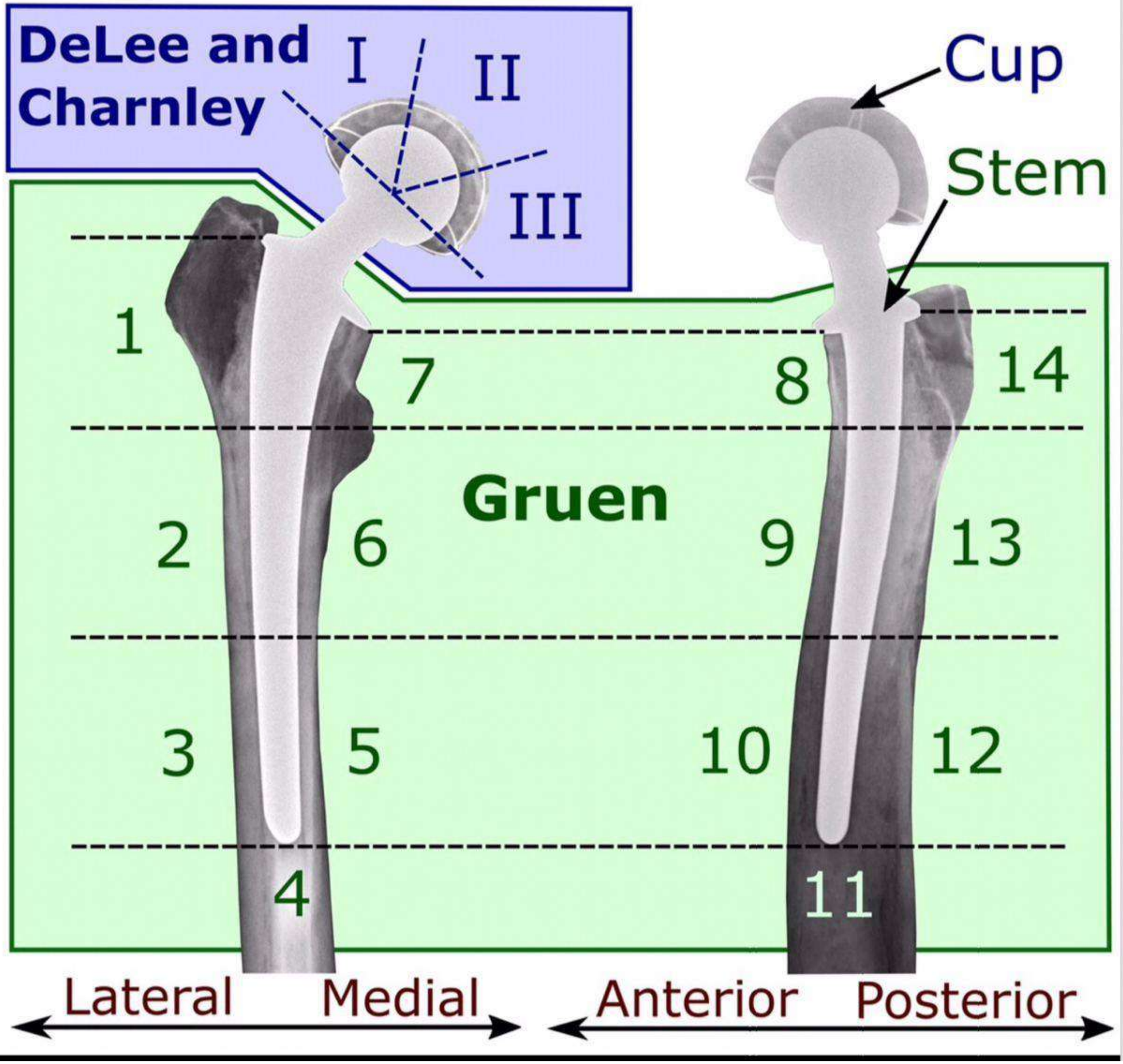
3. Grade 3 : Lucency surrounding **50% to 99%** of the interface

4. Grade 4 :**Complete lucency** & a **defect** of the mantle **at the tip of the stem**



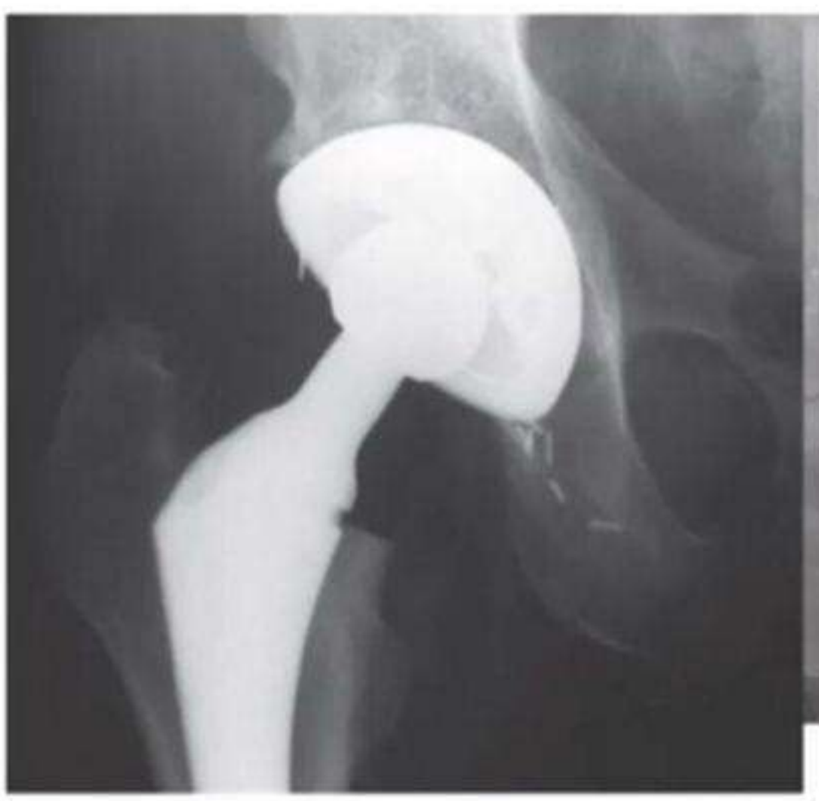


Hip prosthesis zones

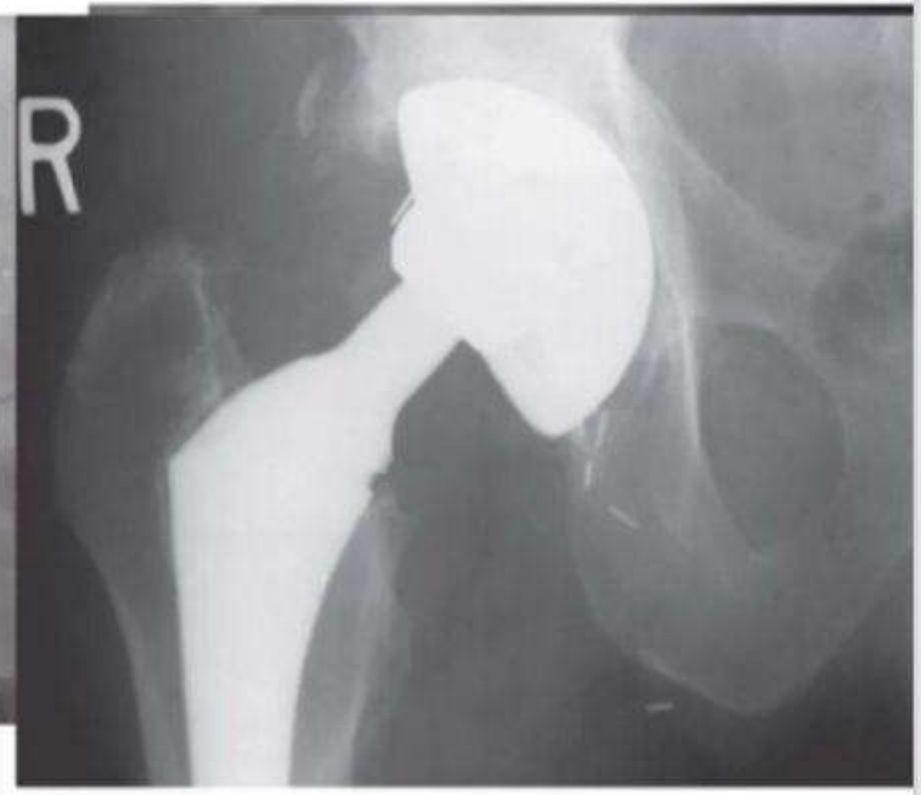


Loosening Cementless-Acetabulum

1. Migration > 2-5mm
2. Broken screw
3. RLL around cup (3 zones)
4. RLL >2mm, progr. >2yrs
5. Change inclination or version



- Postoperative



- 4 years

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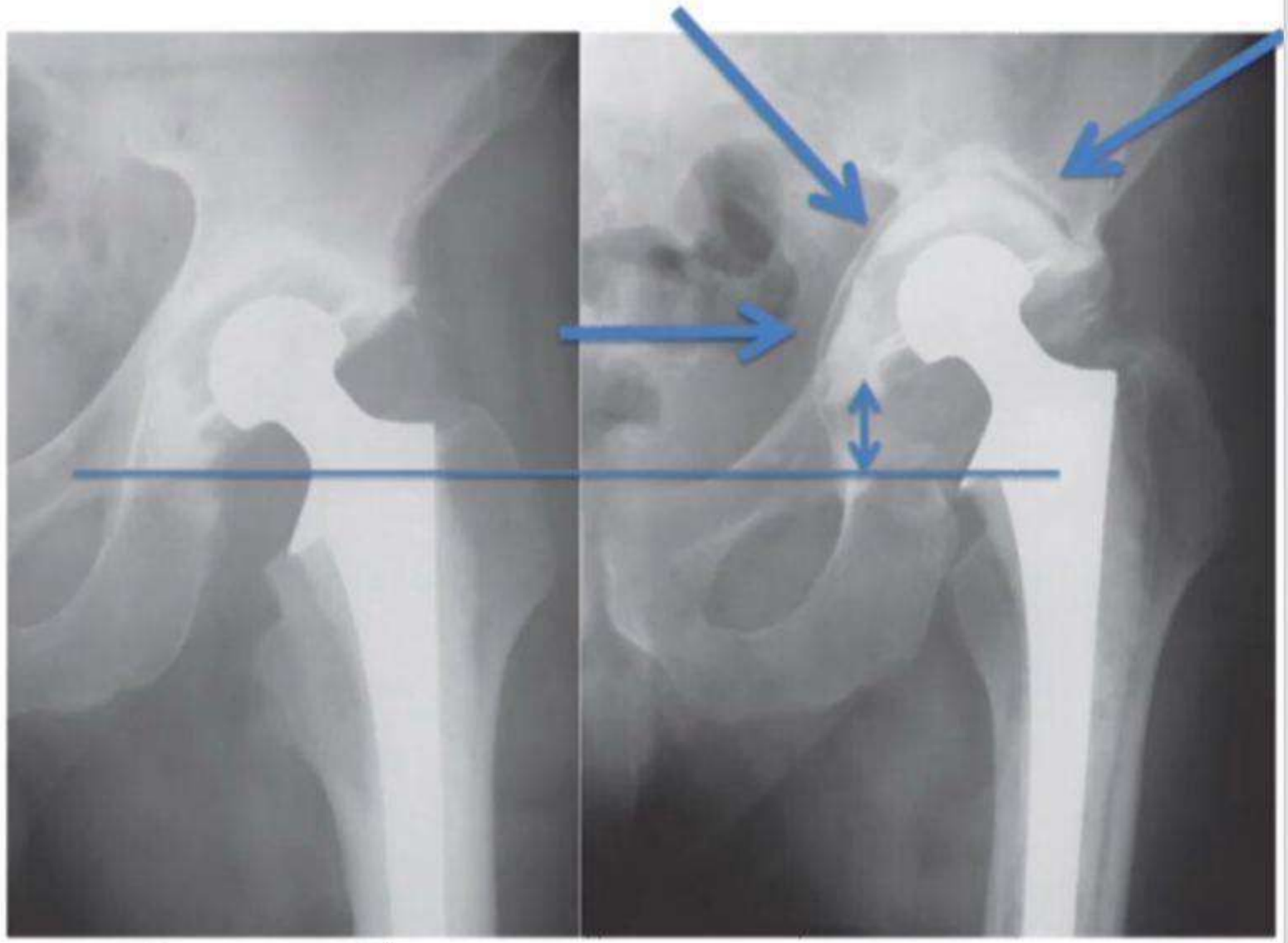
Loosening Cemented Cup

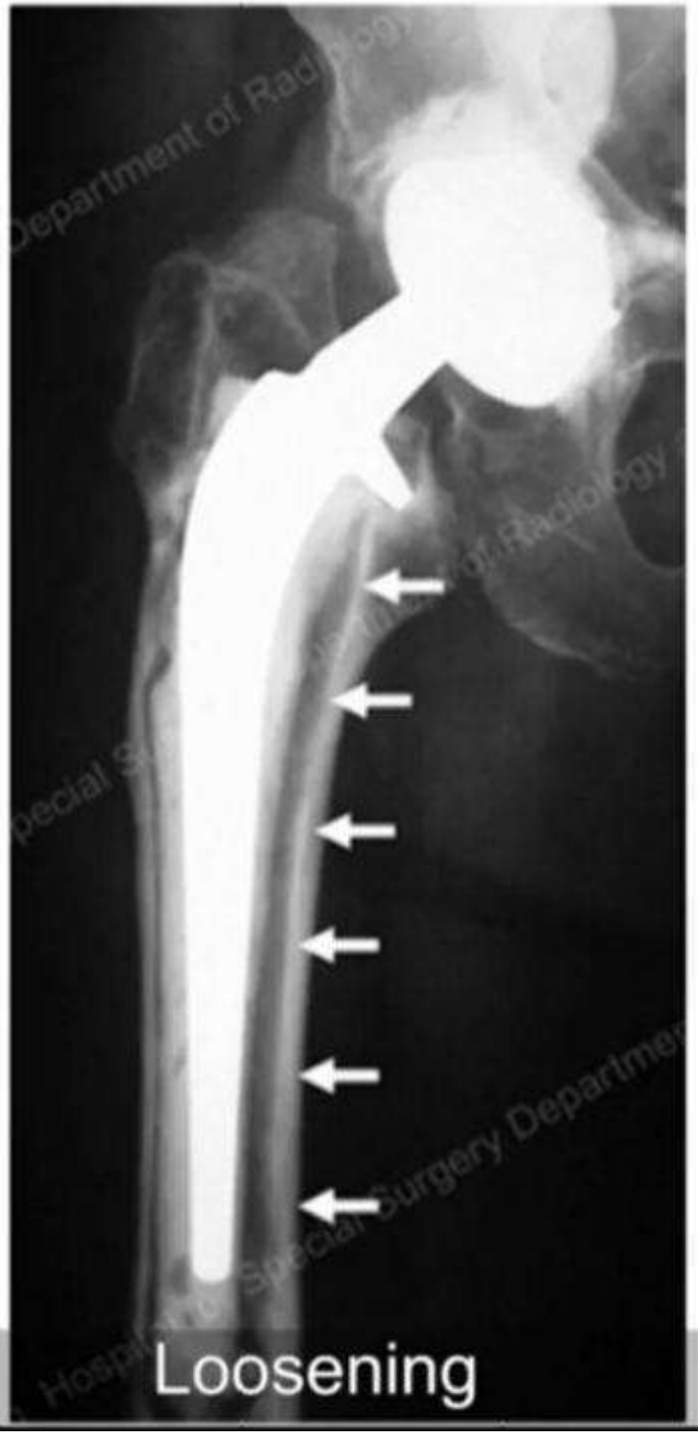
1. Radiolucency (bone resorption) around cup: > 2 mm wide, progressive > 6m
2. Migration or Protrusio: Superior or Medial migration
3. Fracture of medial wall
4. Change in Inclination or Anteversion of cup
5. Wear of the cup
6. Fracture of cup or cement: Rare



Postoperative 7 years

Loosening Cemented Stem

1. Possible
2. Probable
3. Definite



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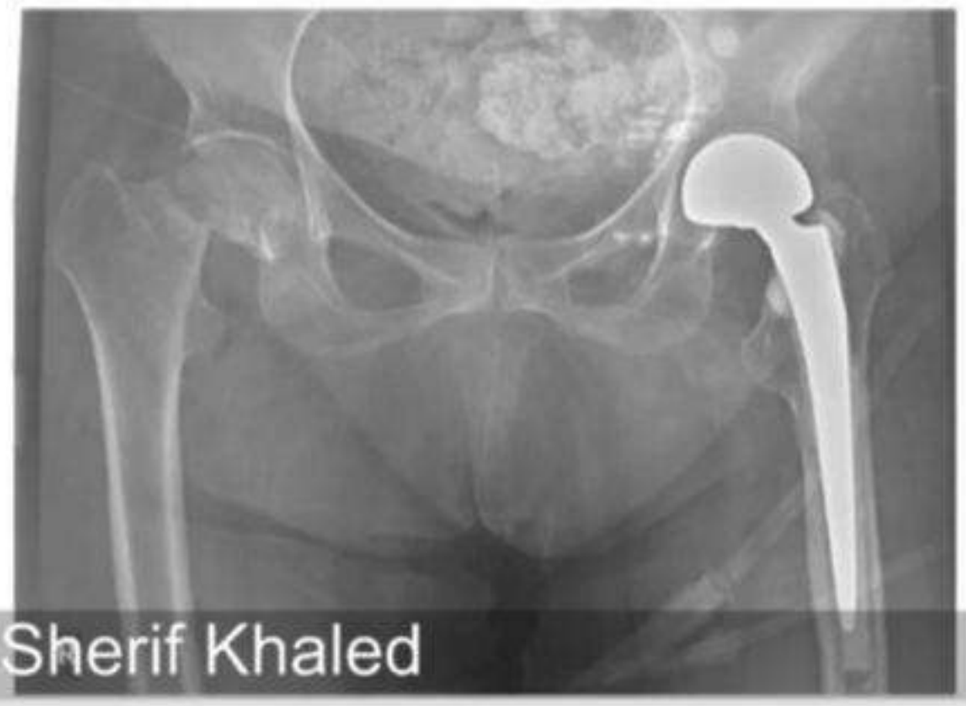
Possible Loosening

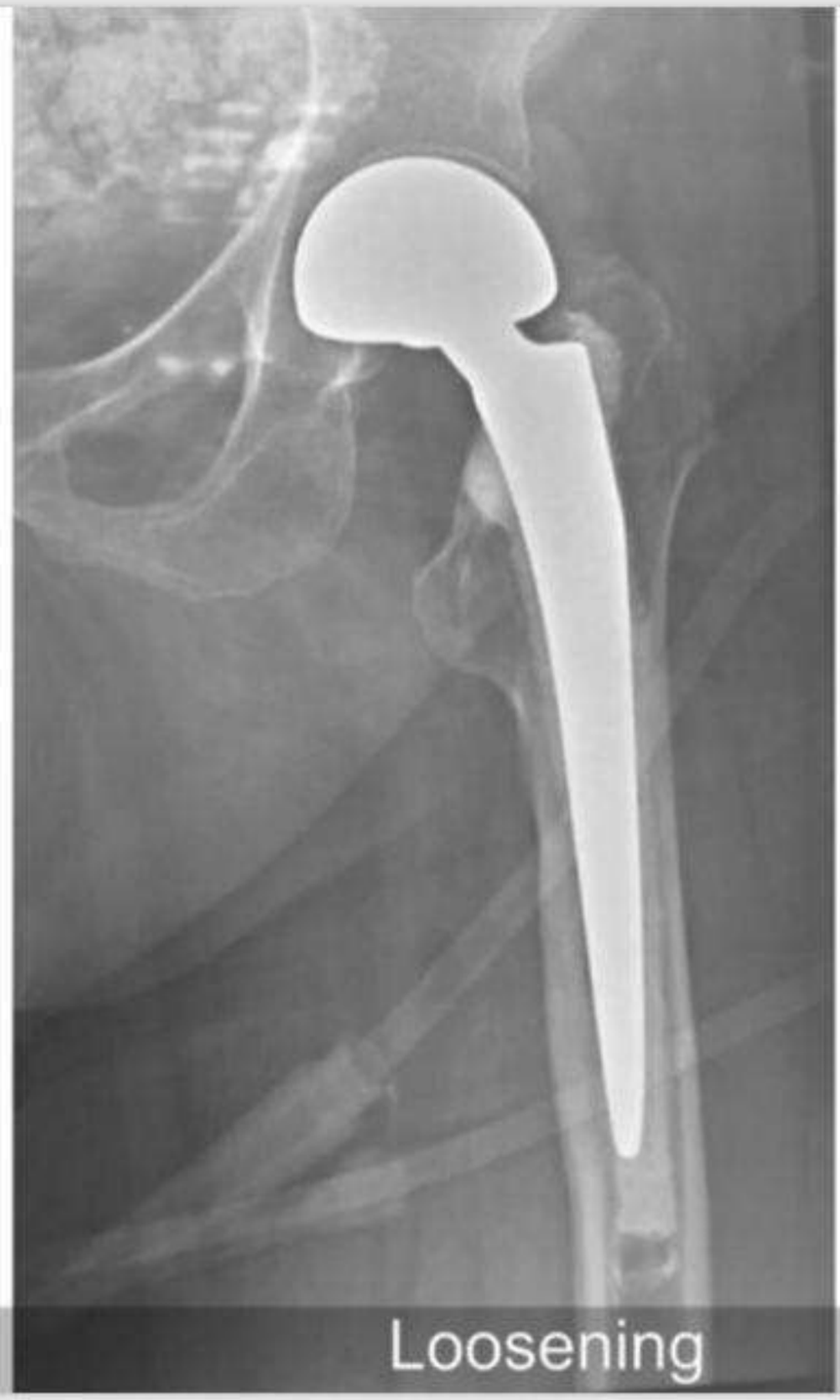
- RLL 50-100% total

  Bone/Cement interface

- Loose if progressive



Sherif Khaled



Loosening

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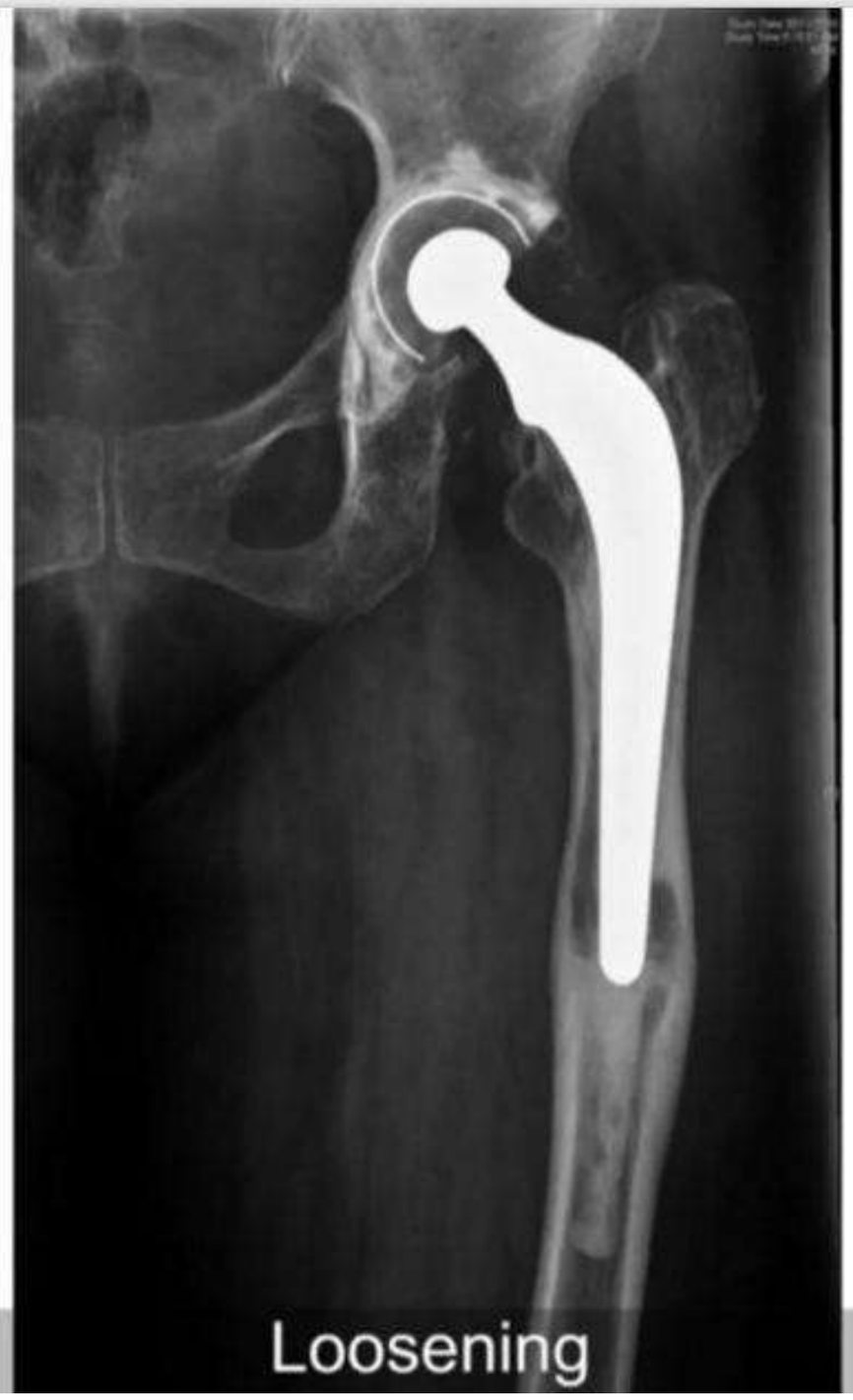
Probable Loosening

- RLL continuous B/C interface
- >2mm wide Progressive
- Surrounded by ↑ density
- Endosteal Cavitation (linear/ focal Osteolysis)

Sherif Khaled

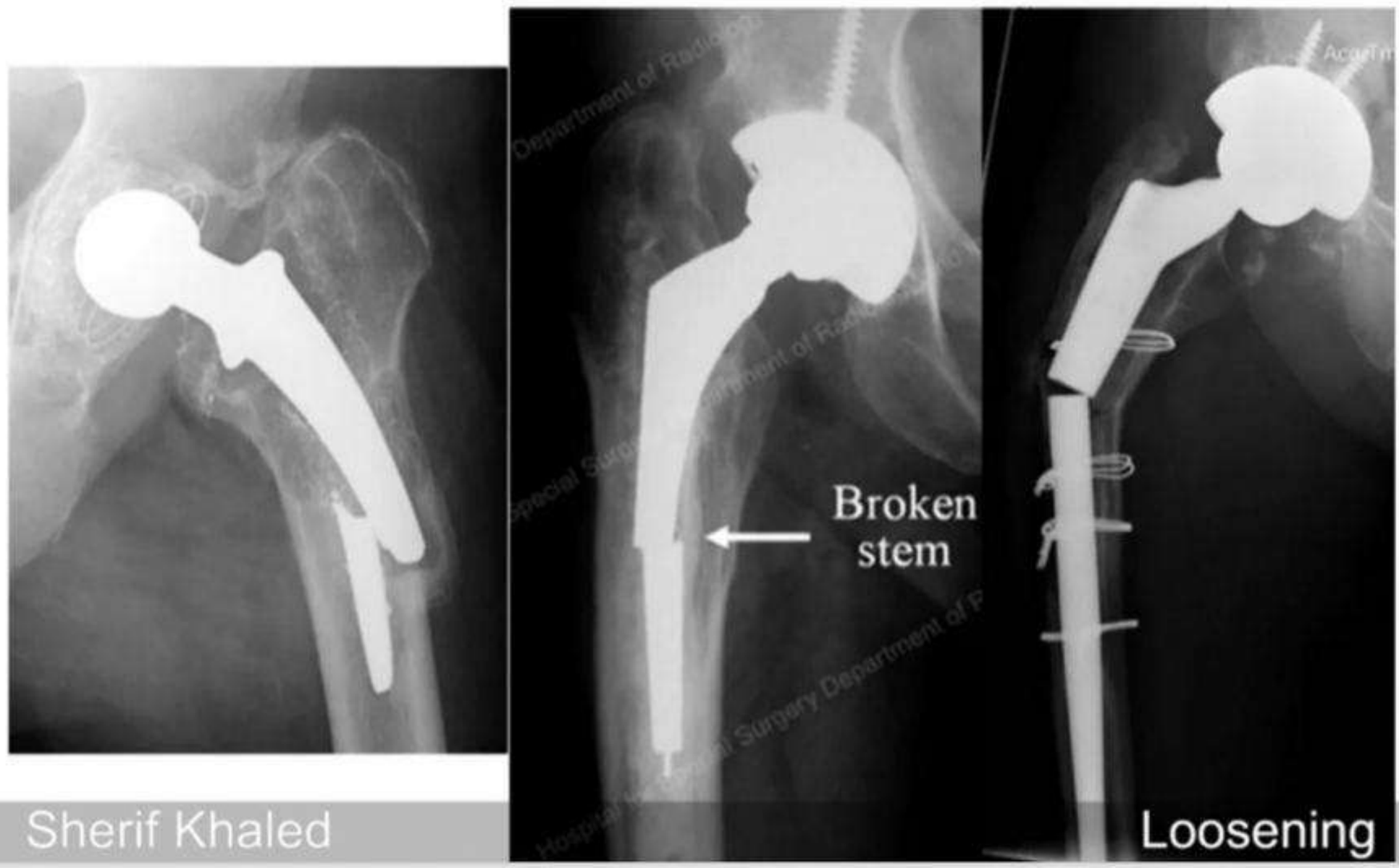


Loosening

Definite Loosening

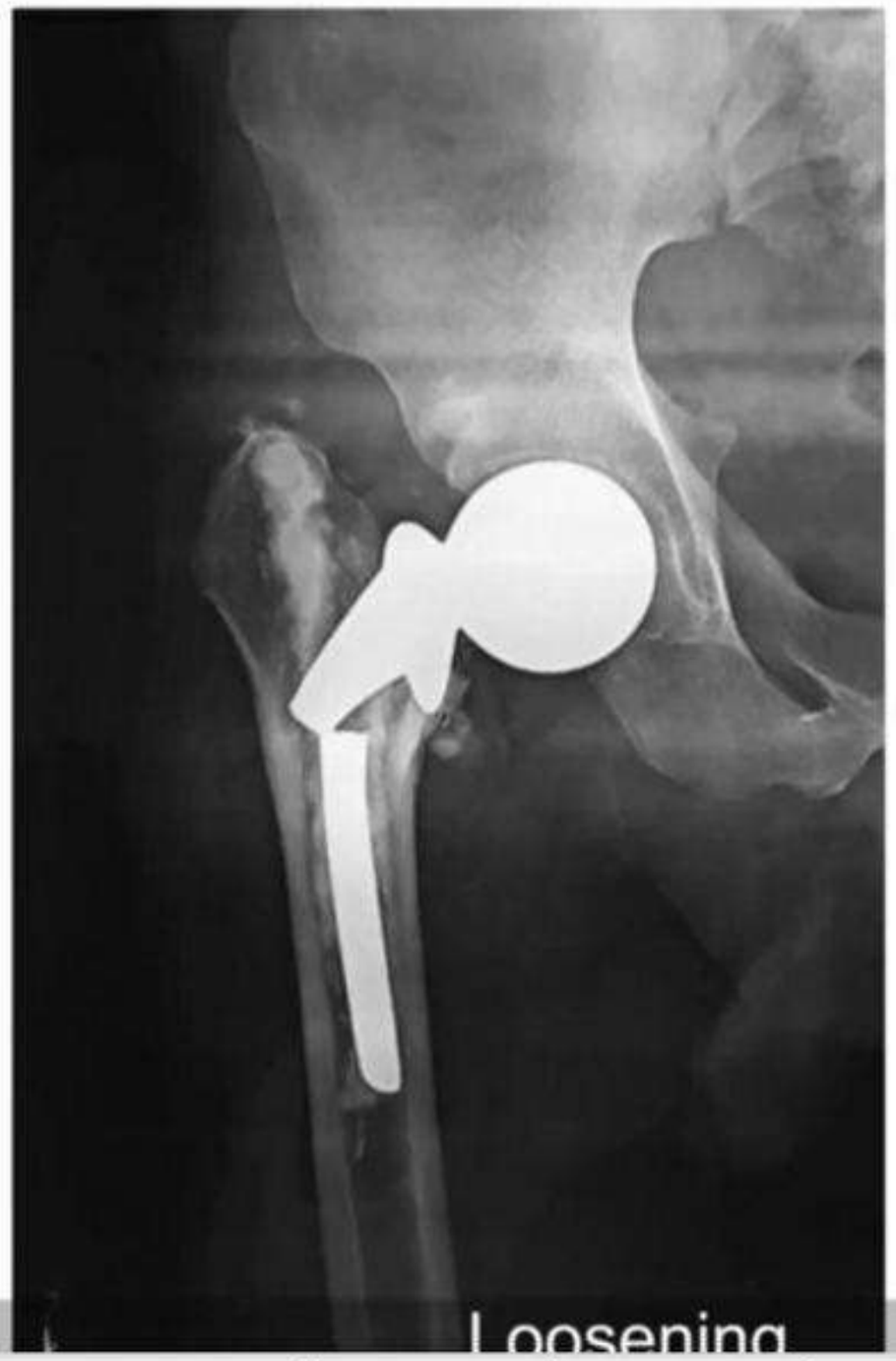
1. Stem fracture
2. Cement fracture
3. Lucency cement-implant interface
4. Stem position Changes:
   I. Subsidence
   II. Medial midstem Pivot
   III. Calcar Pivot
   IV. Distal Pivot

1. Stem Fracture

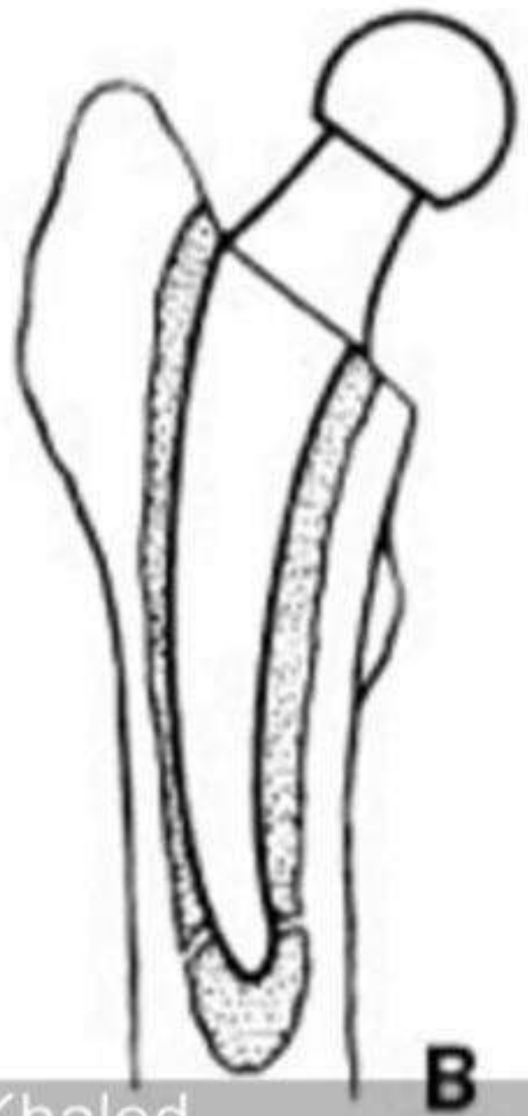


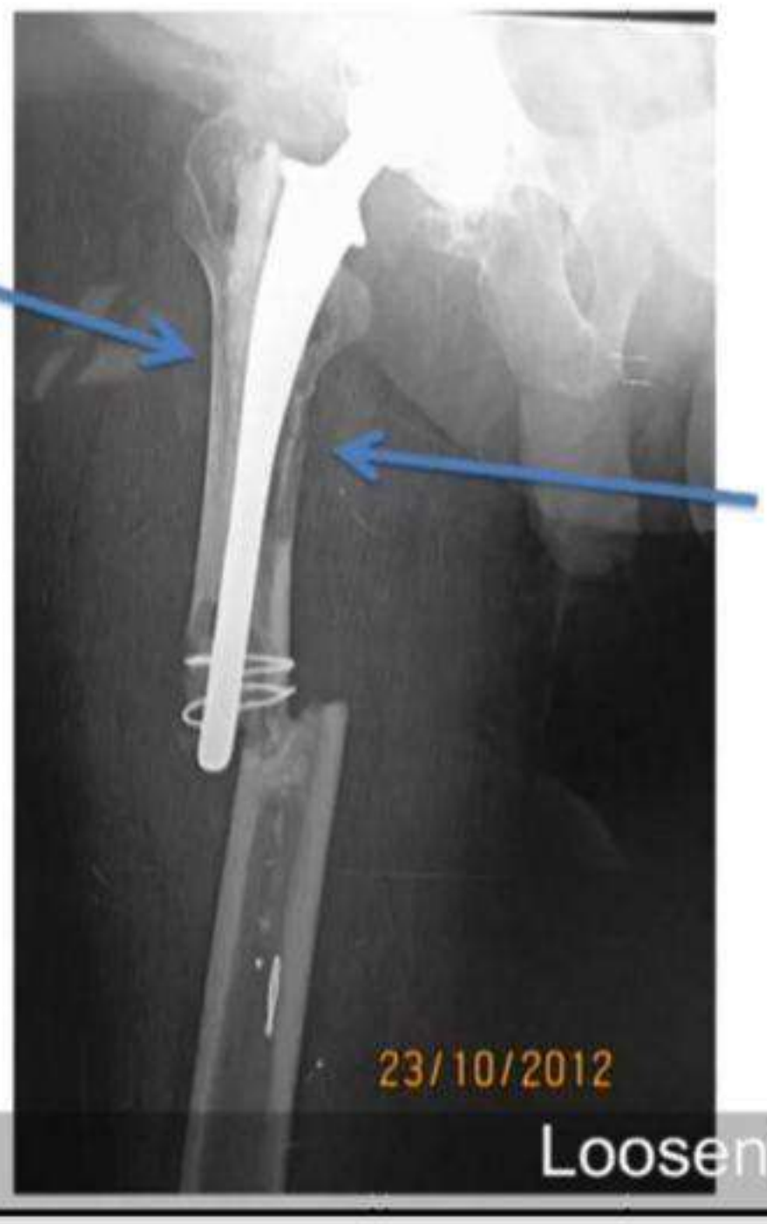
Bending Cantilever

- Distal fix strong, but proximally loose
- Breakdown of proximal cement
- Bone destruction
- Stem Fracture



2. Cement Fracture





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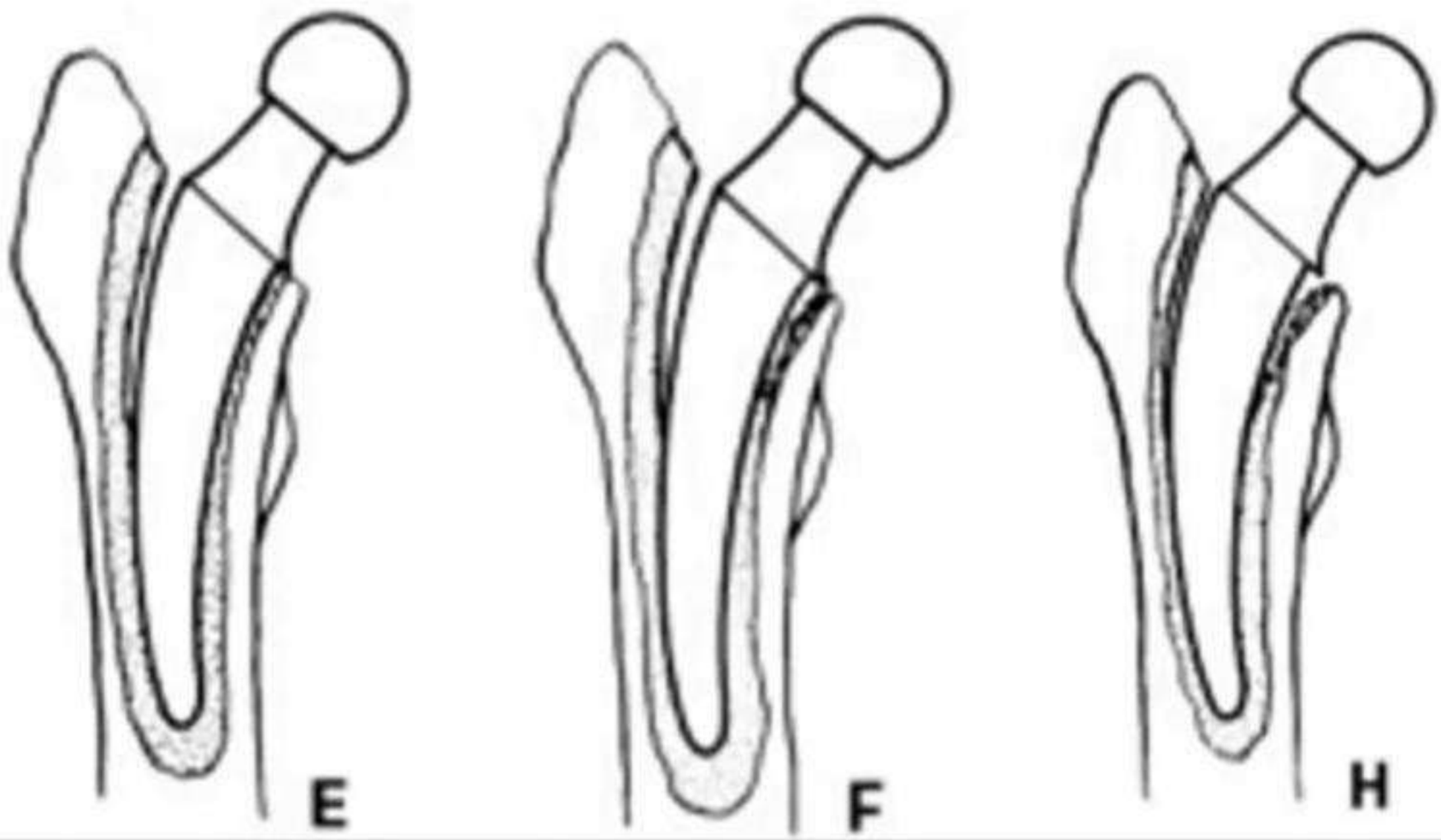
3. RLL Cement-Implant



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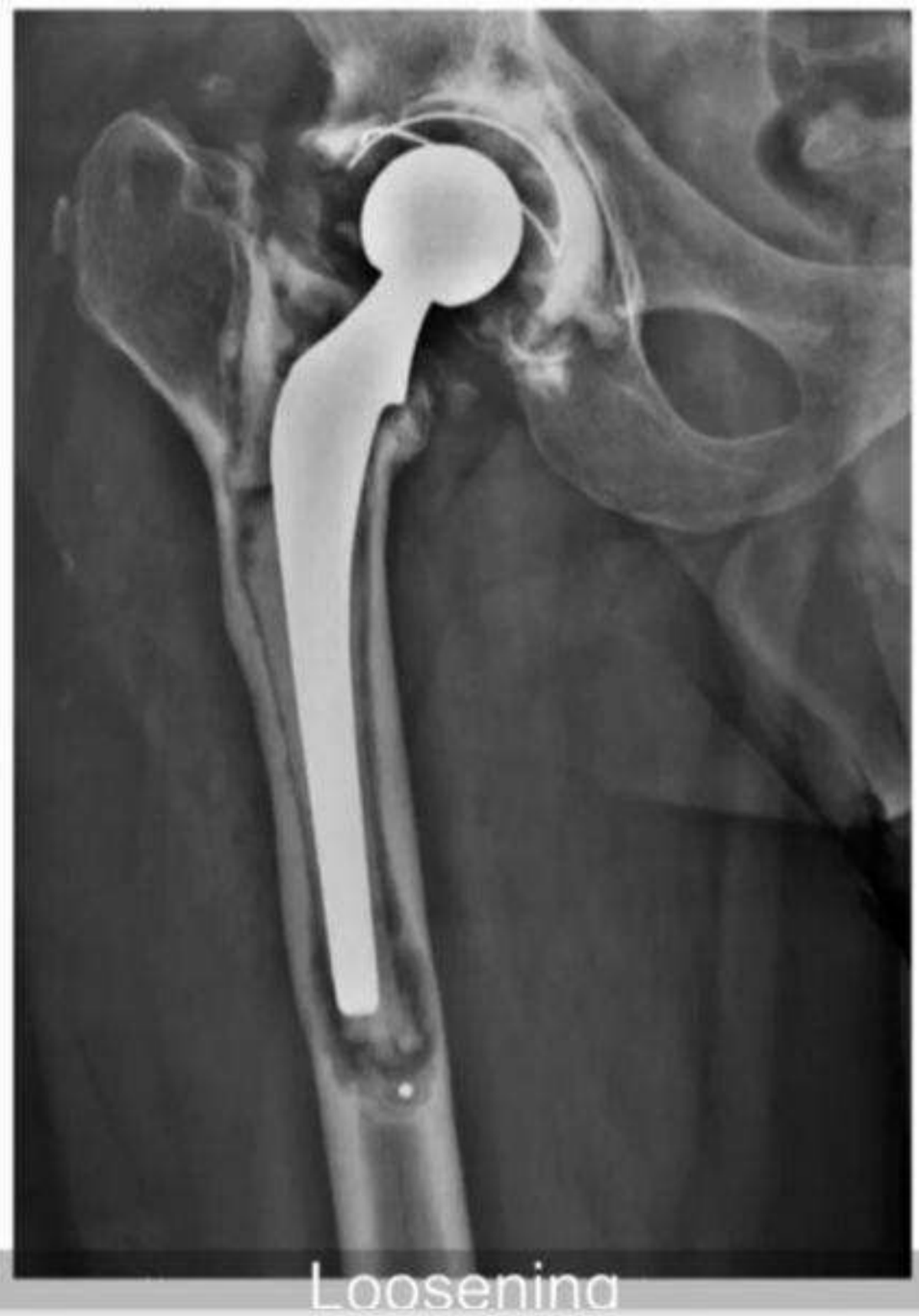
Stem Position Changes

4i. Subsidence

- 1-2 mm normal in 1st year
- 2-5 mm abnormal
- From tip GT to head neck junction (or tip of stem)



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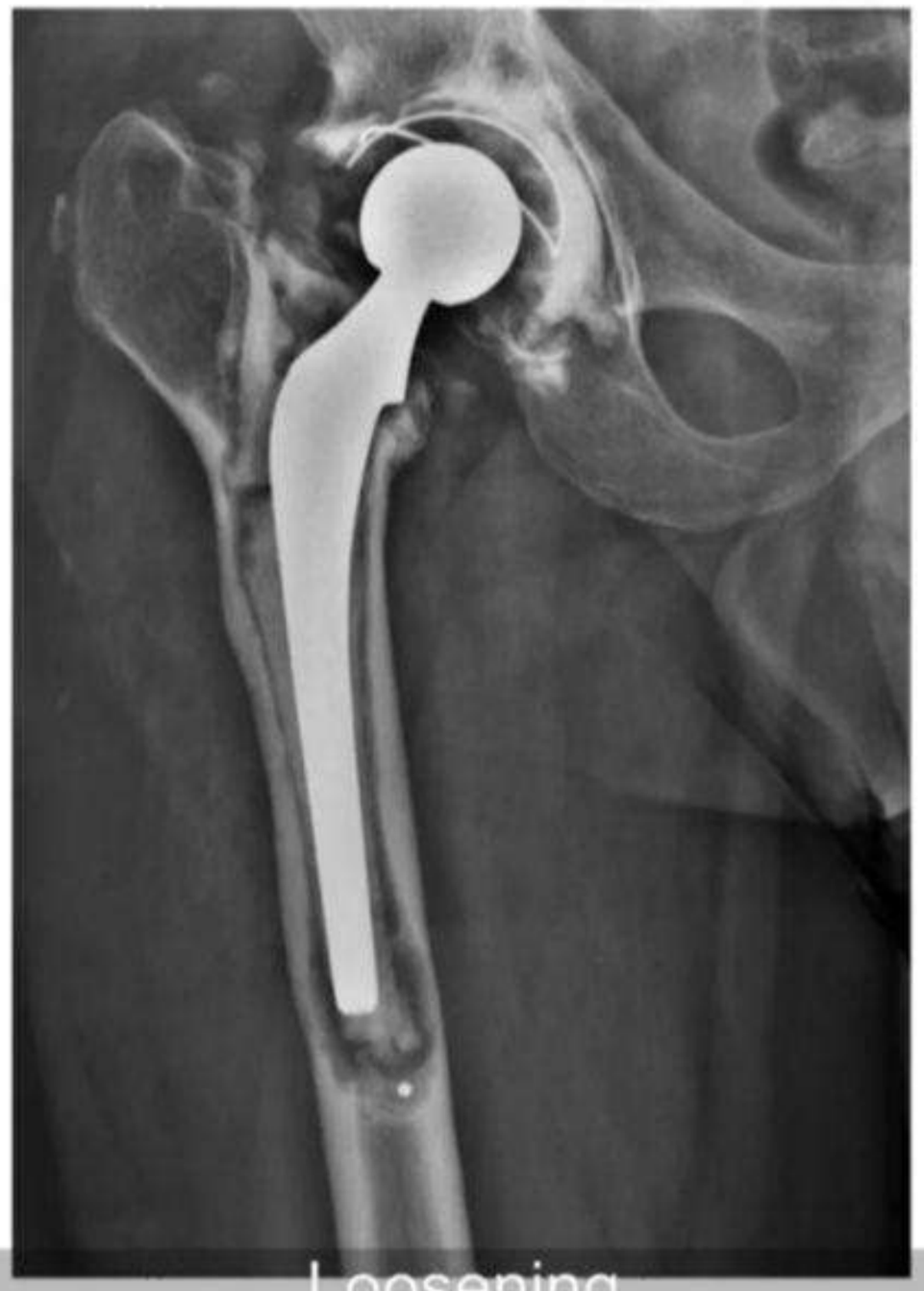
Stem Position Changes
4i. Subsidence

- 1-2 mm normal in 1st year
- 2-5 mm abnormal
- From tip GT to head neck junction (or tip of stem)
- Types:

1. Cement & Stem
2. Stem in Cement mantle (Pistoning)



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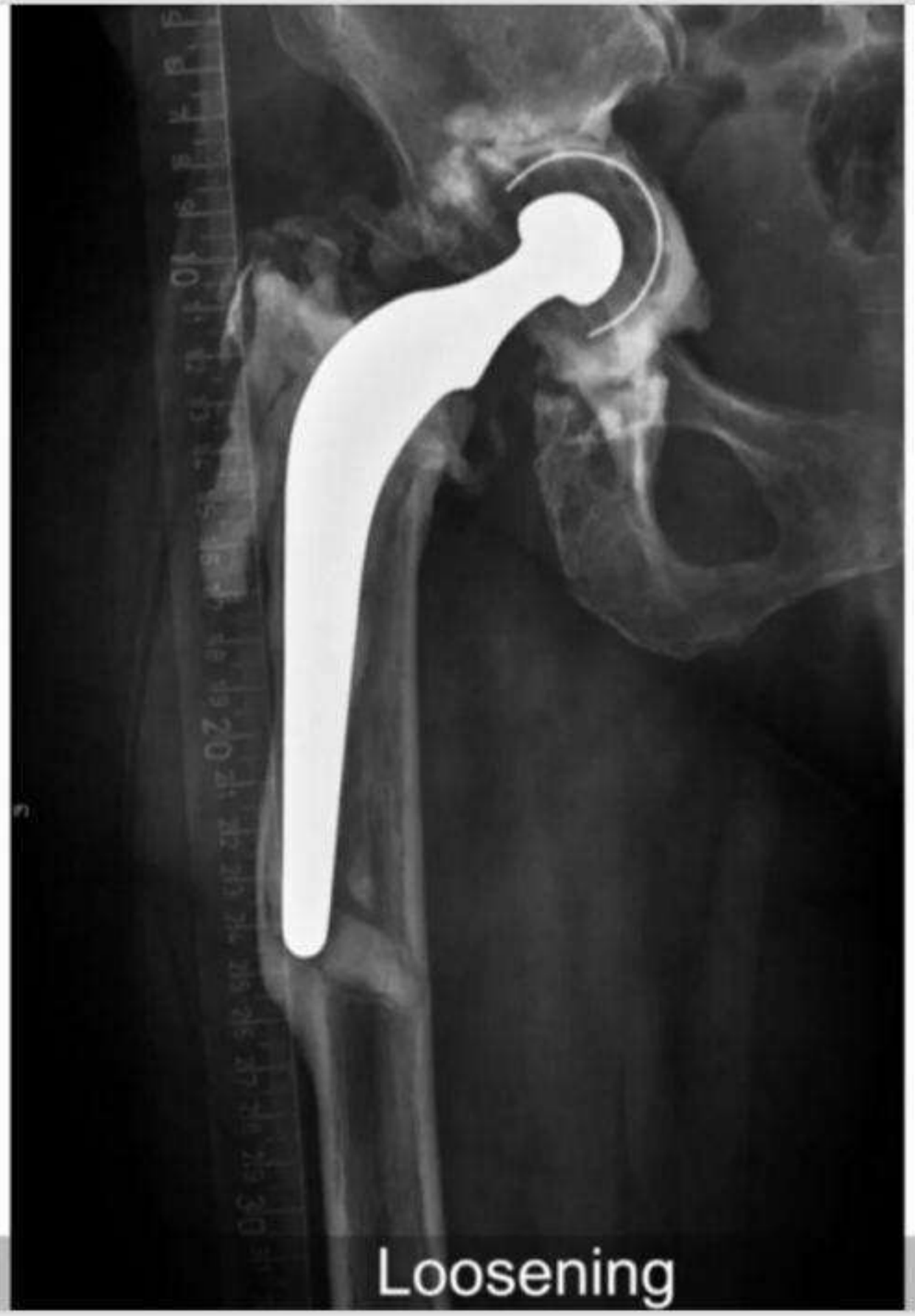
4ii. Medial Midstem Pivot

- Pivots about midstem
- Proximal medial, distal lateral
- Poor cement superomedial or inferolateral

4iii. Calcar Pivot (Distal Toggle)

- Windshield wiper effect
- Distal Loose
- Proximal support adequate
- Subsequent bone reaction



Loosening

3. What are the other modes of failure that you know of?
Answer: Gruen described four modes of failure of a cemented femoral implant:[3]

Mode	Mechanism	Cause
1a	Stem pistoning within the cement	Subsidence of stem within cement
1b	Cement-embedded stem pistoning with the femur	Subsidence of cement mantle and stem within bone
2	Medial midstem pivot	Lack of superomedial and inferolateral cement support
3	Calcar pivot	Medial and lateral toggles of distal stem
4	Bending (fatigue) cantilever	Loss of proximal cement support with distal stem still fixed

EXAMINER: What do you mean by AVN?

CANDIDATE: Avascular necrosis occurs due to interruption of the blood supply to the femoral head leading to ischaemia and cellular death.

EXAMINER: What is the aetiology of AVN?

CANDIDATE: A number of conditions are associated with AVN. The most common cause is trauma secondary to fracture and/or dislocation of the femoral head. Other conditions include:

- Corticosteroid use.
- Alcohol abuse.

Trauma
Gout, Gaucher's
Rheumatoid/radiation
Infection/increased lipids/inflammatory arteritis
Pancreatitis/pregnancy
SLE/sickle cell/smoking
CRF/chemotherapy/Caisson disease
In approximately 10–20% of cases no cause can be identified.

EXAMINER: What is the pathophysiology of AVN?

CANDIDATE: Aetiological factors in AVN are usually related to underlying pathological conditions that alter blood flow, leading to cellular necrosis and ultimately to collapse of the femoral head. This damage can occur in one of five vascular areas around the femoral head: arterial extraosseous, arterial intraosseous, venous intraosseous, extravascular intraosseous and extravascular extraosseous.

1. Extraosseous arterial factors are the most important. The femoral head is at increased risk because the blood supply is an end-organ system with poor collateral development. Blood supply can be interrupted by trauma, vasculitis (Raynaud's disease), or vasospasm (decompression sickness).
2. Intraosseous arterial factors may block the microcirculation of the femoral head through circulating microemboli. These can occur in sickle cell disease (SCD), fat embolization or air embolization from dysbaric phenomena.
3. Intraosseous venous factors affect the femoral head by reducing venous blood flow and causing stasis. These factors may accompany conditions such as Caisson disease, SCD or enlargement of intramedullary fat cells.
4. Intraosseous extravascular factors affect the hip by increasing the pressure, resulting in a femoral head compartment syndrome. For example: fat cells hypertrophy after steroid administration or abnormal cells, such as Gaucher and inflammatory cells, can encroach on intraosseous capillaries, reducing intramedullary circulation and contributing to compartment syndrome.
5. Extraosseus extravascular (capsular) factors involve the tamponade of the lateral epiphyseal vessels located within the synovial membrane, through increased intracapsular pressure. This manifests as trauma, infection and arthritis, causing hip effusion that may affect the blood supply to the epiphysis.

cells within the vasculature. This may result in interruption of the venous drainage from the femoral head, leading to blood stasis, an increase in intraosseous pressure and AVN.[6] Other studies suggest primary osteocyte cell death without any other features. This is seen with steroid use, in transplant patients and those who consume significant amounts of alcohol.

EXAMINER: How common are steroids as a cause of AVN?

CANDIDATE: High-dose corticosteroids are the most common cause of non-traumatic AVN accounting for 10–30% of cases. However only 10% of patients exposed to corticosteroids may develop AVN. Dosage is typically steroids > 2 g of prednisone, or its equivalent, within a 2–3-month period.

The period from the start of corticosteroid treatment to the diagnosis of AVN ranges from 1–16 months (mean 5.3 months), and the majority of patients are diagnosed within 1 year.

EXAMINER: You mentioned the crescent line, what is its significance?

CANDIDATE: Therapeutic interventions are less likely to halt progression of the disease once this sign appears.

EXAMINER: How does AVN of the hip present?

CANDIDATE: Although AVN can be clinically silent typically a patient complains of pain, usually localized to the groin area but occasionally to the ipsilateral buttock and knee. It is usually a deep intermittent, throbbing pain, with an insidious onset that eventually occurs at rest and may be

What dislocation rate do you quote when consenting a patient for a total hip replacement?

Dislocation following hip arthroplasty is one of the most common complications. Large studies have shown the incidence of dislocation following primary hip arthroplasty to be 3–5% over the life of the implant. The dislocation rate more than triples after revision hip surgery. The majority of dislocations occur in the first month (approximately 1%) and first year (approximately 2%). Over 50% of hips re-dislocate after initial closed reduction. Dislocation produces significant cost implications—both in terms of patient morbidity and the financial costs of treatment. It has been estimated that the cost of re-operation for a primary dislocation is 150% that of the original surgery.

What causes a hip to dislocate?

Causes of dislocation are multifactorial and can broadly be divided into surgical factors, patient factors, and implant design factors.

Surgical factors

- Component mal-position (most common)
- Soft tissue imbalance or failure of reattachment
- Soft tissue impingement (osteophytes/capsule)
- Retained debris (cement) in acetabular component

Patient factors

- Previous hip surgery or arthroplasty
- Female gender (relative risk 2.1)
- Acute fracture of proximal femur (relative risk 1.8)
- Inflammatory arthropathy
- Generalized soft tissue laxity
- Patient non-compliance (dementia, learning difficulties, drug/alcohol addiction)

Implant design factors

- Small head/neck ratios—leading to greater impingement risk
- Small head size (relative risk 1.7 with size 22 mm heads compared with 32 mm)
- Loosening of components leading to rotation and mal-alignment
- Wear of acetabular component leading to head subluxation

What measures can you take to prevent re-dislocation?

Prevention of re-dislocation can be attempted using conservative or operative methods. Assessment of joint stability should be made at the time of reduction. If the hip dislocates in the patient's normal functional range then it is likely that surgical intervention will be required.

Conservative methods

- Patient education, carer advice
- Physiotherapy and occupational therapy input
- Bracing of joint in an attempt to 'remind the patient' and prevent a position of instability

Surgical methods

Soft tissue laxity correction:

- Reattachment of avulsed soft tissues or trochanter
- Increasing neck offset using modular components

- Increasing acetabular lateral offset (lateralized liner)
- Trochanteric advancement

Increasing range of motion:

- Increase head–neck ratio (larger femoral head)
- Excision of osteophytes or soft tissues
- Increase excursion distance to dislocation (larger femoral head)
- Revision of mal-aligned components

Increase constraint:

- Augmentation of acetabular liners
- Constrained or captured liners

What are the anatomical issues associated with DDH?

CANDIDATE: The anatomical differences are divided into acetabular, femur and soft tissue issues. The acetabulum is shallow and anteverted; the femur has a small deformed head and short anteverted valgus neck.

EXAMINER: That's not all the differences. There are some you have missed. Do you know any more?

CANDIDATE: Muscles around the hip are usually shortened and er, er . . .

EXAMINER: The greater trochanter is small and posteriorly displaced, the femoral canal narrow, the acetabulum is usually small with poor bone quality, hip capsule elongated and redundant, psoas tendon hypertrophied and abductors orientated transversely as a result of the superior migration of the femoral head. The femoral and sciatic nerves may be shortened and therefore more vulnerable to injury during arthroplasty surgery.

EXAMINER: What is the role of a CT scan in planning an operation for DDH?[1]

CANDIDATE 1: CT scans can be used to determine the available acetabular coverage and to estimate the degree of femoral anteversion.

CANDIDATE 2: CT scans are useful in assessing available bone stock, and the morphology, dimensions and orientation of both the acetabulum and femur.

Answer: c. 4%

Vivas

Adult Pathology

A 45-year-old female underwent a total hip replacement for significant end stage arthritis secondary to dysplasia. An anterolateral approach was used for the surgery with cemented implants in the femur and acetabulum. On the ward round the day after surgery she was noted to have a painless drop foot and loss of sensation in the lower leg.

1. What is the diagnosis and how common is this?
   Answer: Sciatic nerve palsy. This is likely to be a traction injury due to excessive lengthening rather than any direct trauma in this case (antero-lateral approach). Incidence of nerve palsy is 1% to 2% of primary total hip replacement, 3% to 4% after revision, and 5% to 6% in THR for congenitally dislocated hips.
2. Is this patient at higher risk of the complication than a standard patient? What are the other risk factors?
   Answer: Yes the complication rate is higher in patients with hip dysplasia. Due to both alterations in the anatomy[1] and also due to lengthening. The maximum acceptable lengthening (before shortening osteotomy should be undertaken) is 4 cm.[2] Other risk factors include revision surgery, limb lengthening, female gender, anticoagulation and vascular insufficiency.
3. What specific features should be looked for on the post-operative radiograph of the hip?
   Answer: I would want to assess the amount of lengthening. I would also want to ensure that the reduction was congruent with no evidence of soft tissue interposed between femoral head and acetabulum. Finally I would assess for cement extrusion (and possible thermal damage).
4. What other features should be examined for and what investigations should be considered?
   Answer: I would want to carry out a full lower limb neurological examination. The most common branch injured is the peroneal division of the sciatic nerve and I would examine specifically for that. I may consider arranging an USS of the hip to assess for haematoma or a CT scan which may help identify both haematoma and the presence of extruded cement. EMG studies may be used to assess the level of the lesion and also to monitor recovery but may not show any abnormalities for several weeks. The acute management is to detension the sciatic nerve by extending the hip and flexing the knee. I would also provide an ankle foot orthosis (foot-drop splint).
5. When should exploratory surgery be undertaken?
   Answer: Surgery should be undertaken acutely if the nerve may be entrapped or compressed (by haematoma or cement). Similarly if there is a risk the nerve has been surgically transected or ligated with an ill placed suture.
6. Patients may present with a painful drop foot. Would your management differ in this instance?
   Answer: A painful neuropathy is more likely to represent ongoing compression and therefore I would be much more likely to offer surgical exploration.
7. What further investigations should be considered in the outpatient setting?
   Answer: Nerve conduction studies may be used to monitor renervation and referral to a Peripheral Nerve Injury Unit.

Contributors:
Mr Emmet Griffiths, Specialty Registrar, East of England Rotation

2. What is the course of radial nerve in this region?
   Answer: The radial nerve courses over the spiral groove posteriorly between lateral and medial heads of triceps. 14 cm proximal to the lateral epicondyle, 20 cm proximal to the medial epicondyle. It passes through the Lateral intermuscular septa never less than 7.5 cm above the distal articular surface amd then runs between brachialis and brachioradialis (anterior to lateral epicondyle)
3. What is the incidence of radial nerve injury with humeral fractures?
   Answer: The reported incidence is between 8% and 15% of closed fractures. This is increased in distal one-third fractures (the Holstein lewis lesion). A neuropraxia is the most common injury in closed fractures, whereas in open fractures a neurotmesis is more likely.
4. What is the prognosis for radial nerve injury with these fractures and what are the prognostic factors?
   Answer: 85% to 90% of patients improve with observation over three months, spontaneous recovery found at an average of seven weeks, with full recovery at an average of six months. I would want to get EMG at three to four months to ensure that renervation was occurring (fibrillations indicative of denervation would be an indication for surgical exploration). I would expect wrist extension in radial deviation is to be regained first and extensor indicis to be the last. Prognosis is improved in younger patients and in those with a neurapraxia.
5. What are the indications of surgery in patients with humeral fractures?
   Answer: Absolute indications include open fracture, vascular injury requiring repair and brachial plexus injury. Relative indications include ipsilateral forearm fracture (floating elbow), bilateral humerus fracture, polytrauma or associated lower extremity fracture (to allow early weight bearing

B. Recon & Sport Viva : Long & Short

Infected THR

- When presented with a case of possible infection say 'I am concerned about infection and would take history and examine wound and investigate further'
- 0.5 % in OA and 1 % in RA, double the risk in revision surgery
- **ROH, Prospective study (BJJ, 2006)**: Deep infection developed in 0.57% hip replacements and 0.86% knee replacement
- Most common infecting micro-organism was coagulase-negative staphylococcus, followed by Staph. Aureus, Enterococci and Streptococci

Prevention

1. **Pre-op** (Same day admission):
   - Separation of elective from trauma patients, ring-fencing of beds (GIRFT)
   - Treat septic lesions (feet, urinary, dental, and respiratory)
   - Shave in anaesthetic room
   - Nasal decolonisation
   - Avoid intra-articular corticosteroids
   - Glycaemia control
2. **Intra-op**
   - Antibiotic-loaded cement
   - Broad spectrum Antibiotic on induction
   - Ultraclean air & laminar flow
   - Body exhaust suit
   - Avoid unnecessary movement of theatre personnel
   - Surgical Technique -
     - gentle handling of tissues, length of surgery, wound lavage, careful haemostasis, avoid tissue necrosis, prevent hematoma,
   - Face masks, modern weaved gowns, sterile disposable non-woven drapes.
   - Catheterization covered with Antibiotic (Incidence of bacteremia increases from 0.5% to 1.0% with single catheterization to 10-30% for catheters left for 4 days)
3. **Post-op**
   - Antibiotic for 24 hours
   - Minimise dressing changes
   - Early but safe discharge

History and examination:

1. **History**
   - Date of the index operation, revision surgery, any signs/symptoms of delayed wound healing, hematoma, persistent discharge after the index procedure, any hip debridement and washout following the index procedure, prolonged antibiotics usage
   - Or if the patient had a pain-free period and recurrence of pain following recent insertion, indwelling urinary catheter and any recent history of UTI, dental extraction or URTI

2. **Pain**
   - Site, duration and progression of pain
   - Septic osteolysis, where the pain has persisted from the time of operation, is continuous, relentlessly progressive and presents with rest pain or night pain, or
   - It can occur in a well-functioning arthroplasty from a distant source of infection usually from dental, respiratory, or urinary tract infection through the haematogenous route
3. **Risk factors**
   - Skin conditions increasing permeability of bacteria - Psoriasis, venous disease
   - Co-morbidities reducing immunity - DM, CKD, Liver failure, malnutrition, HIV
   - Inflammatory arthroplasty (RA, Psoriasis, AS)
   - Life style: Morbid obesity (BMI >50 has 18.3% increased risk of PJI – **Parvizi JBJS Am 2010**), smoking, excessive alcohol consumption, IV drug abuse, and poor oral hygiene
   - Can do serum Cotinine test for smokers
   - Recent bacteremia
4. **Examination**
   - Gait, hip kept in a position of ease, previous surgical scar, circumferentially look for signs of inflammation, induration, fluctuance and sinuses, ROM and any associated deformity of contractures

Classification

➢ **Coventry/Fitzgerald classification**

- **Acute**
  - Post-operative - within 3 weeks
  - Superficial infection / infected hematoma
  - Not invaded bone prosthesis interface
  - Haematogenous - Secondary to another infection
- **Chronic**
  - Has been present for > 3 weeks
  - Low-grade intraoperative infection
  - Biofilm

➢ **Musculoskeletal Infection Society (MSIS) criteria 2018, Parvizi et.al**

- *Major criteria*
  - Presence of sinus tract is diagnostic
  - 2 positive cultures from separate samples
- *Minor criteria* (≥6 infected, 2-5 inconclusive, 0-1 not infected)
  - Serum
    - CRP >10mg/L or D-dimer >860ng/mL – 2 points
    - ESR > 30mm/hr – 1 point
  - Synovial
    - WBC >3000 cells/uL – 3 points
    - Positive alpha-defensin – 3 points
    - Synovial PMN > 80% - 2 points
    - Synovial CRP > 6.9mg/L – 1 point

- **Intra-operative diagnosis** (≥6 infected, 5 inconclusive, ≤3 not infected)
  - Positive histology – 3 points
  - Positive purulence – 3points
  - 1 positive culture – 2 points

Biofilm

- Produced by bacteria (staph) formed on implant within 3 weeks of infection
- Polysaccharide (Glycocalyx) layer protects bacteria from host immune system (WBC and Abx)
- Adhere to foreign material, inhibit antimicrobial activity & reduce ABx penetration
- Anti-biofilm agents: Rifampicin for Gram +ve and Ciprofloxacin for Gram –ve

Organisms:

- Staphylococcus Aureus - 20%
- Staphylococcus Epidermidis - 30%
- Coagulase-negative Staphylococcus
- Peptostreptococcus: following dental procedure
- Most common fungal organism is Candida
  Requires antifungal drugs for 6-12 months

Investigations

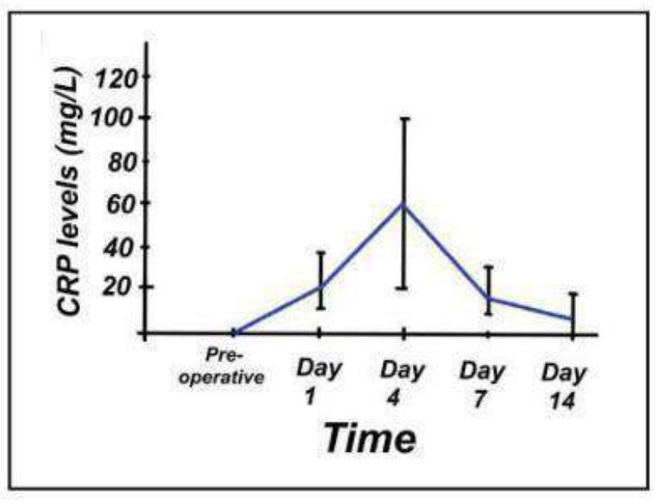
- **Blood tests: Berbari (JBJS, 2010 – meta-analysis)**

1. **CRP:**
   - Normally as high as 250 in 1st week post THR
   - Peaks at day 3; normalize in 2 weeks
   - Sensitivity 88 % and specificity 74%
2. **ESR:**
   - Takes up to 6 months to go back to normal
   - Sensitivity 75 % and specificity 70%
3. **IL 6:**
   - Produced by stimulated monocytes and macrophages
   - Peaks at 2 days and rapidly return to normal value. Sensitivity 97% and specificity 91%
   - Expensive

   (**Berbari et al JBJS Am 2010** – IL-6 more accurate than ESR, CRP, or WBC count)
4. **Pro-calcitonin**
5. **Synovasure (Zimmer Biomet):** Biomarker
   - Alpha Defensin test for peri-prosthetic joint infection
   - 97% sensitivity and 96% specificity & Only 62% in low grade infection
   - Antimicrobial peptide released by neutrophils in response to pathogens
   - False +ve with metallosis

(Courtesy of F Arnaout)

6. **Polymerase chain reaction (PCR)** of aspirate, amplifies bacterial DNA



- Contamination leads to false-positive result

7. **Synovial fluid WBC**
   - > 3000 cell/uL
   - >80% PMN
   - Saline injection & Re-aspiration provides accurate diagnostic information in event of dry tap
   - AAOS guidelines:
     - Aspirated fluid for microbiologic culture & synovial fluid WBC
     - Patients should be off-antibiotics for minimum of 2 weeks prior to obtaining intra-articular culture

➢ **Histopathology**
- **Intraoperative tissue culture**
  - 7 samples (2 or more should grow same organism)
- **Intra operative frozen section**
  - 10 WBC/high power field

➢ **Radiology**
- **X-rays:**
  - Osteolysis (focal), cortical destruction, new bone formation (periosteal reaction) in chronic infection
- **Tech bone scan**
  - Sensitive, useful if –ve
  - Can remain +ve 2 years following THA
- **White cells scan**
  - High specificity (100%) but low sensitivity (45%)
- **Positron Emission Tomography**
  - PET identifies areas of high metabolic activity
  - Sensitivity & specificity 98%

Management

➢ Can use Ethylene blue to delineate area of infection in soft tissue in presence of sinus

➢ Poly exchange & Abx for 6 weeks (Debridement, antibiotic, Implant retention (**DAIR)**
- Early post-op or acute haematogenous (less than 3 weeks) –If implants are stable, change modular components
- No communicating sinus or purulence in joint
- Low virulence and known micro-organism
- Patient not immunocompromised
- Success rate 16-89%

➢ Prosthesis removal & Abx spacer
- Abx for 6 weeks for chronic infection
- Aim of inserting spacer
  - Maintenance of joint space and limb length
  - Stabilization of the joint to allow some mobilization
  - Reduce dead space
  - In-situ release of high local antibiotic dosage with reduced systemic effects
- Increase porosity by hand mixing
- Heat stable Abx for spacer
  - Gentamycin Maximum 10 g (6 weeks elution time)
  - Tobramycin 2.4 - 3.6 g /40 g package of cement 2 weeks elution time
  - Vancomycin 1-4 grams per 40 grams of cement -Maximum 10 g

Chapter 2

Hip structured oral questions

Paul A. Banaszkiewicz and Rahul Kakkar

All viva questions outlined here are examples of actual questions asked in the FRCS (Tr & Orth) exam. Currently each viva question lasts 5 minutes and examiners are advised against switching to another topic earlier even if a candidate knows the subject well. Therefore to give a more realistic 5-minute viva question some topics have been combined from several smaller disjointed accounts from the old-style viva.

We have aimed the candidates' answers for a 7–8 score so they are significantly more detailed than what would be required for a bare pass. Aiming for the minimum to pass will generally be unsuccessful and is not recommended. On a few occasions we have answered questions in a less detailed manner so as to allow readers to gauge the differences in potential scores.

Structured oral examination question 1

EXAMINER: This is a radiograph of a 77-year-old woman who sustained a displaced intracapsular fractured neck of femur 3 years earlier managed with a hemiarthroplasty of the hip (see Figure 2.1). She was admitted onto the orthopaedic ward last night because of increasing left hip pain and difficulty mobilizing.

CANDIDATE: This is an anteroposterior (AP) radiograph of the pelvis taken on the 11/5/11 demonstrating a cemented Thompson's hemiarthroplasty of the left hip. The neck cut is straight down on to the lesser trochanter. The prosthesis seems to have sunk below the lesser trochanter and there are radiolucencies in Gruen zones 1, 4, 5 and 7. There appears to be a faint rim of calcification in the soft tissues, adjacent to the lateral cortex of the femur. The femoral head size would seem to match the acetabulum so it is not under or oversized and the femoral stem orientation appears neutral, neither excessively anteverted nor retroverted. I would like to see immediate postoperative radiographs to confirm whether there has been a change in stem position from the time of the original surgery and would also like to see an up-to-date lateral radiograph of the hip.

EXAMINER: Here is a lateral radiograph of the left hip that was taken on admission. (Figure 2.2.)

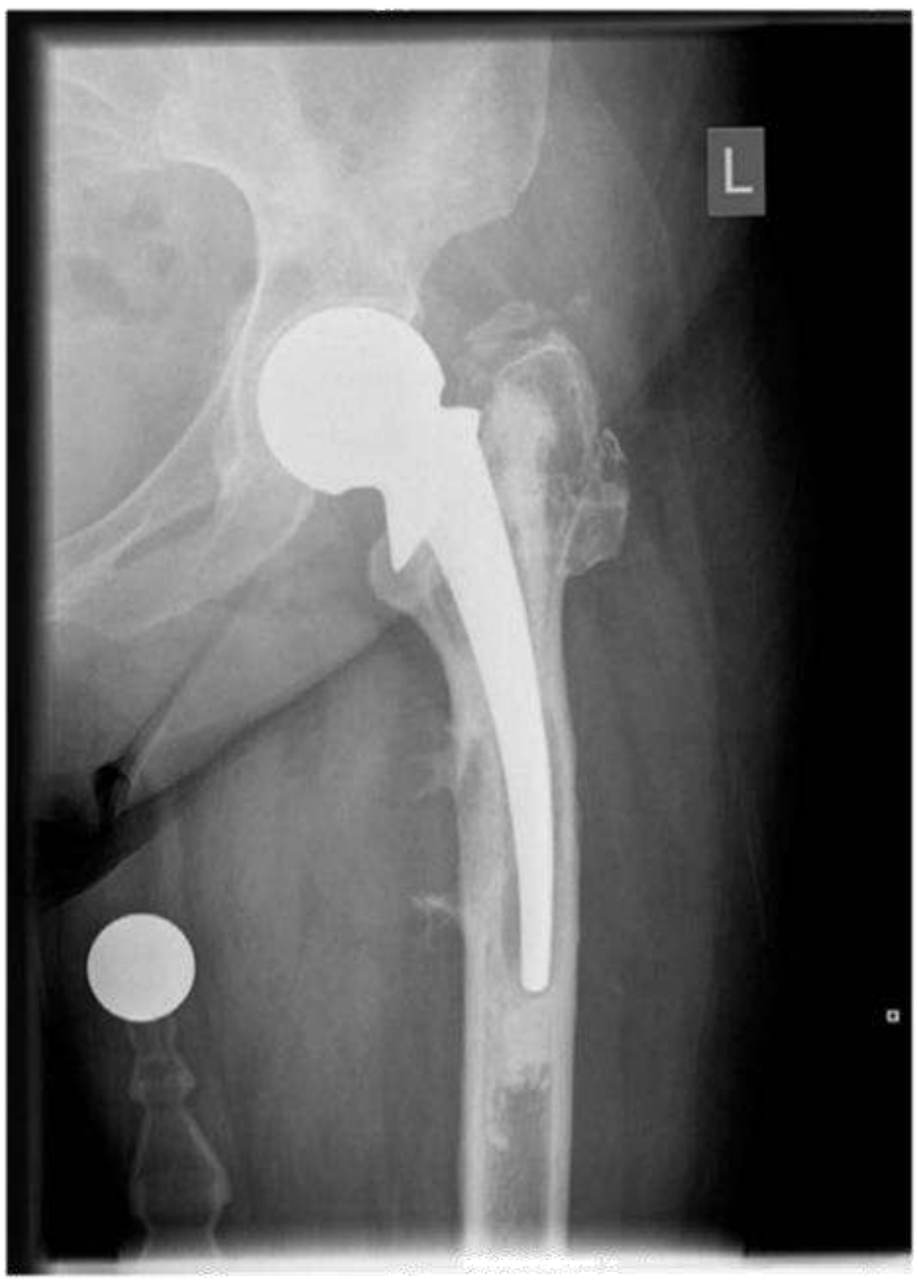


Figure 2.1 Anteroposterior (AP) radiograph of loose left cemented Thompson's hemiarthroplasty hip.

Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination, ed. Paul A. Banaszkiewicz and Deiary F. Kader. Published by Cambridge University Press.

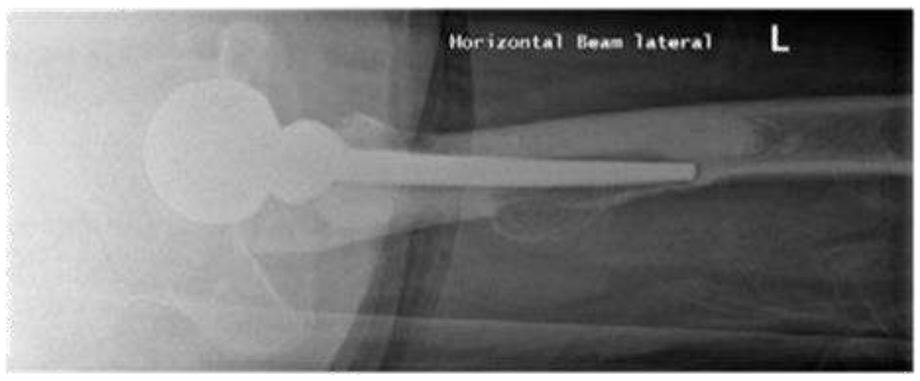


Figure 2.2 Lateral radiograph of loose left cemented Thompson's hemiarthroplasty hip.

CANDIDATE: The lateral radiograph demonstrates loosening of the Thompson's prosthesis with a large cortical lytic lesion surrounding the stem. There appears be reactive bone formation along the posteromedial diaphysis of the femur and a suggestion of a possible soft tissue mass. There is lateral cortical destruction at the tip of the stem.

EXAMINER: What do you think is going on?

CANDIDATE: The stem appears loose. The prosthesis has only been inserted for 3 years. I think the radiographs are highly suggestive of infection until proven otherwise.

EXAMINER: How would you investigate this patient?

CANDIDATE: I would perform routine blood tests including CRP and ESR to see if there are raised inflammatory markers.[1]

EXAMINER: How helpful are these?

CANDIDATE: They have relatively low sensitivity and low specificity as markers of prosthetic joint infection. Berbari *et al.* (Level II) published a systematic review in the JBJS American edition in 2010 on the use of inflammatory markers for diagnosis of prosthetic joint infection.[2] They concluded that IL-6 is a much more sensitive test for infection.

EXAMINER: What do you mean by sensitivity and specificity?

CANDIDATE: Sensitivity is the ability of a test to pick up truly infected cases and specificity the ability of the test to exclude appropriately those cases which are not infected.

Or in more general terms sensitivity is the proportion of individuals with the disease (or condition) who are correctly identified by the test. Specificity is the proportion of individuals without the disease who are correctly identified by the test.

Positive predictive value is the probability that a patient with a positive result genuinely has infection and the negative predictive value is the probability that a patient with a negative result has genuinely avoided infection.[4]

EXAMINER. The paper actually reported that IL-6 was more accurate than CRP or ESR rather than sensitive. What do we mean by accuracy?

CANDIDATE: The accuracy of a test is defined as the proportion of tests that have given the correct result (true positives and true negatives).

EXAMINER: So how are you going to proceed with this patient?

CANDIDATE: I would want to take the patient to theatre and perform an aspiration of the hip to rule out infection.

EXAMINER: You are jumping in a bit fast. Is there anything else you might want find out beforehand?

CANDIDATE: I would want to take a full history from the patient. A number of patients who develop infection have early wound problems such as prolonged redness, induration, swelling or discharge. There may be a history of repeated courses of antibiotics. The wound may have become frankly infected requiring washout in theatre.

Onset of hip pain following a problem-free interval and an episode of sepsis is suggestive of haematogenous seeding of infective organisms from elsewhere. I would enquire if there was a history of bacteraemia from a UTI, chest infection or dental extraction.

Pain from an infected prosthesis is typically non-mechanical and unrelated to physical activity and not relieved by rest.

EXAMINER: The wound was oozy postoperatively but settled down. A large part of picking up periprosthetic infection is obtaining a good history and examination along with a high index of clinical suspicion.

How useful is a hip aspiration in diagnosing infection?

CANDIDATE: Spangehl *et al.* (level I) demonstrated a sensitivity of 0.86, a specificity of 0.94, a positive predictive value of 0.67 and a negative predictive value of 0.98 with initial image-guided aspiration in 180 patients undergoing revision hip arthroplasties.[5] They reported that aspiration alone is not sufficient for the diagnosis because of the risk of false-positive and false-negative results. They suggested in low-probability cases with a normal ESR and CRP that aspiration was not necessary. Aspiration would be indicated if pretest probability for infection was high (acute onset of pain, systemic illness, sinus formation) particularly if the CRP/ESR was normal or in all cases where the CRP or ESR was high.

EXAMINER: Joint aspiration was negative. How are you going to manage this patient?

CANDIDATE: I would need to get more history from the patient, most importantly what her symptomatic complaints are and also fully assess her fitness for anaesthesia and surgery.

EXAMINER: She has a 2-year history of intermittent progressively worsening hip pain worse with activities such as walking or rising from a chair. She is not the fittest patient for surgery; she developed pseudo-bowel obstruction and aspiration pneumonia postoperatively after the hemiarthroplasty requiring HDU admission.

CANDIDATE: I would need to sit down with the patient and fully discuss what her expectations from surgery were. We would need to reach an agreement on whether she would wish to proceed with revision hip surgery taking on board/taking into account/accepting the potential risks and complications of the surgery weighed against the probable benefits of the procedure.[6]

EXAMINER: She can't live with her pain – she wants you to do something!

CANDIDATE: I would still be very suspicious that the hemiarthroplasty has a low-grade infection and perform a two-stage hip revision operation for her.

EXAMINER: Are there any other tests you might want to perform that could diagnose infection before going ahead with surgery?

CANDIDATE: The use of nuclear imaging (technetium-99 triple-phase bone scan, gallium imaging, labelled-leukocyte scans or FDG-PET imaging) for the detection of periprosthetic joint infection is worth considering but controversial. The recent AAOS clinical practice guidelines summary from 2010 reported a weak recommendation for their use.[7]

EXAMINER: How do you classify periprosthetic hip infection?

CANDIDATE: Tsukayama *et al.* proposed a 4-stage system consisting of early postoperative, late chronic and acute haematogenous infections, and positive intraoperative cultures of specimens obtained during revision of a presumed aseptically loose THA.[8,9]

Early postoperative infection presents less than 1 month after surgery with a febrile patient and a red swollen discharging wound. With late postoperative infection the patient is well, the wound has healed well, there is a worsening of hip pain and a never pain-free interval. Acute haematogenous infection can occur several years after surgery with a history of bacteraemia (UTI or other source of infection) and severe hip pain in a previously well-functioning hip. Positive intraoperative culture (at least three samples from different locations taken with clean instruments) occurs when a preoperative presumptive diagnosis of aseptic loosening was made.

McPherson *et al.* have also developed a staging system for periprosthetic hip infections that included three categories: infection type (acute versus chronic), the overall medical and immune health status of the patient, and the local extremity (wound) grade.[10]

EXAMINER: Why are you discounting a one-stage procedure?

CANDIDATE: Although there are advantages to performing a one-stage procedure such as low treatment cost and preservation of patient function it is a controversial option as the success rate is less than a two-stage procedure. The procedure involves removal of the prosthesis, thorough debridement and re-implantation at a single sitting.

EXAMINER: What are the prerequisites for a one-stage procedure?

CANDIDATE: There may be a case for performing a single-stage revision in a specialist centre with a large experience in dealing with infected hips.

EXAMINER: That's not quite the question I asked.

CANDIDATE: Prerequisites include a known organism sensitive to antibiotics, no pus present, elderly patients or patients with multiple medical problems. It is also indicated in healthy individuals devoid of re-infection risk who have adequate bone and soft tissue for reconstruction and a low virulence pathogen.

EXAMINER: What are the reported success rates for a single-stage revision?

CANDIDATE: Buchholz *et al.* who pioneered one-stage revisions at the Endo-Klinic in Hamburg reported a success rate of 77% in 583 revisions, but only after extensive bone and soft-tissue resection, which compromised long-term function.[11] These results were published in 1981 and can be viewed as somewhat historic now. Raut *et al.* from Wrightington reported a success rate of 86% in 57 cases at average follow-up of 7 years despite many discharging sinuses.[12,13] Hanssen and Rand summarized the results of single-stage exchange and found a cumulative success rate of 83% when antibiotic-loaded cement was used but only 60% when it was not.[14]

EXAMINER: What are the advantages to performing a two-stage procedure?

CANDIDATE: It is particularly important to perform a two-stage revision with more severe infections or virulent organisms, as the success rate of a single-stage procedure is much less in these situations.

EXAMINER: That's not what I asked.

CANDIDATE: It is more versatile for reconstruction allowing the use of either cemented or cementless components and bone allograft in patients with severe bone loss. It allows clinical assessment of the response to antibiotics prior to re-implantation.

EXAMINER: What are the disadvantages of a two-stage procedure?

CANDIDATE: It can be difficult to nurse patients between stages and the second-stage surgery can be difficult due to soft tissue scarring, limb shortening, disuse atrophy, loss of bone density and distortion of anatomy. If a PROSTALAC spacer is used it can dislocate or fracture and it is more costly to perform a two-stage procedure.

EXAMINER: So you perform the first-stage revision, how long will you keep the patient on antibiotics? (Figure 2.3.)

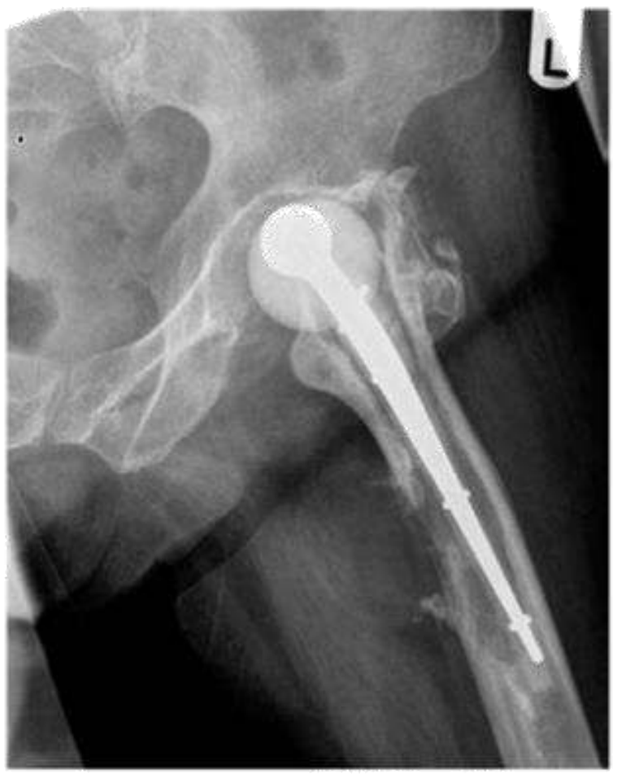


Figure 2.3 Anteroposterior (AP) radiograph of first stage PROSTALAC spacer.

CANDIDATE: Duration of antibiotic treatment and timing between stages remains controversial. Current practice suggests delaying the second stage for at least 6 weeks pending good clinical progress with antibiotics and wound healing. A number of surgeons re-implant at 3 months treating the patient with 6 weeks of antibiotics and then 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. Some surgeons would routinely re-aspirate the hip to exclude any residue infection before going ahead with the second stage.

EXAMINER: Five of my last six THAs have become infected – what should I do?

CANDIDATE: Stop operating and investigate.

EXAMINER: Go on.

CANDIDATE: I would want to know if the same organism had been identified in the five cases particularly if the organism was *Staphylococcus aureus* as this may suggest a nasal carrier in theatre. Nasal swab cultures would need to be taken of relevant theatre staff and appropriate treatment started.

We would want to investigate for a breakdown in theatre sterility. I would involve microbiology and investigate the laminar flow system to see if it was working correctly.

There may be issues with the preparation of the instruments set such as packaging integrity and expiry date. A sterilization indicator should be present and the packaging must be dry.

There may be a breakdown in the precautions that must be taken by the scrub practitioner during the procedure such as the sterile field not being constantly observed and too much movement around the sterile field, including the opening and closing of doors and a wide space not being observed between scrubbed staff.

Taylor and Bannister showed that sets opened outside the confines of the laminar hood have significantly higher colony forming unit (CFU) counts during and after surgery.[15] Very few centres follow Sir John Chamley's technique of opening the instrument sets under the canopy at each stage of the operation.

Madhavan *et al.*'s paper from Bristol in the *Annals of the Royal College of Surgeons England* specifically looked at breakdown in theatre discipline during total joint replacement.[16] They noted a slackness had crept into theatre protocol such as corridor from changing room to theatre and theatre personnel attire.

EXAMINER: Do you know any papers that have looked at theatre sterility?

CANDIDATE: The classic paper on theatre sterility was published by Lidwell *et al.* in 1982.[17] This was an MRC randomized study which showed a decrease in infection rates following joint replacements carried out in ultra-clean theatres.

The deep infection rate was 3.4% in conventional theatres, 1.7% with ultraclean air and body exhaust and 0.2% when this was combined with prophylactic antibiotics.

EXAMINER: That's fine. Let's move on.

Alternative scenario

Differential diagnosis of the lesion would be granulomatous reaction to wear debris from Thompson's

hemiarthroplasty. This is much more likely with a metal-on-polyethylene bearing THA interface. Other important differentials include metastatic disease and soft tissue sarcoma.

The examiner could lead you down the path of investigation of a possible tumour mass. A bone scan and MRI would need to be ordered for further investigation. A computed tomography-guided fine-needle aspiration of the mass could be performed. See references 18 and 19 for a similar type of scenario.

Endnotes

1. The candidate has got out of sync with the examiner and flow of the question. Not a disaster. The candidate should have answered how they would manage the patient with the standard default answer of history, examination and investigations etc.
2. Berbari E, Mabry T, Tsaras G *et al.* Inflammatory blood laboratory levels as markers of prosthetic joint infection: a systematic review and meta-analysis. *J Bone Joint Surg Am* 2010;**92-A**:2102–2109.
3. These are double-bullet questions fired at the candidate from a high-powered rifle and the candidate has to give a precise, correct answer back and then the oral continues on.
4. In the oral exam it is better to explain these terms to the examiners by drawing a table but in this particular question it doesn't quite fit together with the interactions to do this.
5. Spangehl MJ, Masri BA, O'Connell JX *et al.* Prospective analysis of preoperative and intraoperative investigations for the diagnosis of infection at the sites of two hundred and two revision total hip arthroplasties. *J Bone Joint Surg Am* 1999;**81-A**:672–682.
6. Waffly answer but can't be helped – it is what needs to be said by the candidate to the examiners. A standard, safe, nondescript response.
7. It may be enough just to mention the uncertainties with nuclear imaging or one may have to quantify your answer a bit more fully. It is a judgement decision but don't persist with your answer if the examiners want to move on. ^{99m}-Technetium bone scans are sensitive but not specific. Some investigators have found that a negative scan rules out infection while others have reported that a scan can occasionally be negative in the presence of infection if there is inadequate blood supply to the bone. A ^{99m}-technetium bone scan identifies areas of increased bone activity through preferential uptake of the diphosphonate by metabolically active bone. Increased uptake occurs with loosening, infection, heterotopic bone formation, Paget's disease, stress fractures, modulus mismatch of a large uncemented stem, neoplasm, reflex sympathetic dystrophy, and other metabolic conditions. In the uncomplicated THA, uptake around the lesser trichinae and shaft is usually insignificant by 6 months, but in 10% of cases, uptake may persist at the greater trochanter, prosthesis tip and acetabulum for more than 2 years. The pattern of uptake has not been found to consistently reflect the presence or absence of infection. Gallium imaging likewise has a poor sensitivity and accuracy. The use of leukocyte scans is generally preferred, having a higher sensitivity (88–92%) and specificity (73–100%), but their usefulness for the diagnosis of infection continues to be debated. FDG-PET is expensive, limited to a few institutions and although very sensitive does not allow differentiation between an inflamed aseptically loosened prosthesis and an infected one.

8. Tsukayama DT, Estrada R, Gustilo RB. Infection after total hip arthroplasty. A study of one hundred and six infections. *J Bone Joint Surg Am* 1996;**78-A**:512–523.
9. This is sometimes referred to as Gustilo's classification. With due respect to the first author Gustilo is easier to remember.
10. McPherson EJ, Woodson C, Holtom P *et al.* Periprosthetic total hip infection. Outcomes using a staging system. *Clin Orthop Relat Res* 2002;403:8–15.
11. Buchholz HW, Elson RA, Engelbrecht E *et al.* Management of deep infection of total hip replacement. *J Bone Joint Surg Br* 1981;**63-B**:342–353.
12. Raut VV, Siney PD, Wroblewski BM. One-stage revision of infected total hip replacements with discharging sinuses. *J Bone Joint Surg Br* 1994;**76-B**:721–724.
13. With due respect although Raut is the first author I think 'Wroblewski from Wrightington has shown' is easier to remember. There is enough to learn already without making things difficult for yourself!
14. Hanssen AD, Rand JA. Evaluation and treatment of infection at the site of a total hip or knee arthroplasty. *J Bone Joint Surg Am* 1998;**80-A**:910–922.
15. Taylor GJS, Bannister GC. Infection and interposition between ultraclean air source and wound. *J Bone Joint Surg Br* 1993;**75-B**:503–504.
16. Madhavan P, Blom A, Karagkevrakis B *et al.* Deterioration of theatre discipline during total joint replacement – have theatre protocols been abandoned? *Ann R Coll Surg Engl* 1999;**81**:262–265.
17. Lidwell OM, Lowbury EJ, Whyte W *et al.* Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement: a randomised study. *Br Med J* 1982;**285**:10–14.
18. Hanna MW, Thornhill TS. Thigh mass and lytic diaphyseal femoral lesion associated with polyethylene wear after hybrid total knee arthroplasty. A case report. *J Bone Joint Surg Am* 2006;**88-A**:2473–2478.
19. Patterson P, Grigoris P, Raby N *et al.* A thigh mass associated with a total hip replacement in a 69-year-old woman. *Clin Orthopaed Related Res* 2002;**404**:373–377.

Structured oral examination question 2

EXAMINER: This is an anteroposterior (AP) radiograph of a 52-year-old woman who presents to your clinic with non-specific right hip pain. She had a right metal-on-metal hip resurfacing procedure performed 3 years ago. (Figure 2.4.)

CANDIDATE: The anteroposterior (AP) radiograph demonstrates a higher abduction angle (lateral opening) than normal. The current recommendations are for an acetabular abduction angle of 40°. Several studies have demonstrated the importance of optimal cup positioning with regard to wear, metal ion levels and the revision rate. High cup angle has been consistently reported to lead to greater wear and higher serum metal ion levels. The head size appears small; the current recommendations are that unless a minimum 46 mm head size can be used the procedure should not be performed because of the risks of ALVAL and pseudotumours. There is no radiolucency about the metaphyseal stem, no obvious narrowing of the neck and no divot sign.

EXAMINER: What do you mean by a divot sign?

CANDIDATE: A divot sign is a depression in the neck contour just below the junction with the femoral component often associated with a reactive exostosis. It is believe to be caused by repetitive bone-to-component abutment due to impingement.

EXAMINER: What is a pseudotumour and what is the difference between ALVAL and pseudotumour?

CANDIDATE: ALVAL (aseptic lymphocyte-dominated vasculitis-associated lesion) is caused by metal particulate debris. Patients present with localized hip pain and a localized osteolytic reaction. A more severe inflammatory reaction is termed a pseudotumour.

Several studies have described an association between pseudotumours and increased wear of retrieved components. Influencing factors include implant size and implant design (clearance and cover [arc angle]). In addition acetabular component positioning and femoral head–neck offset influence the risk of impingement and edge loading usually associated with high wear rates.[2] Despite this Campbell *et al.* reported that in 32 THA revised due to pseudotumor several patients demonstrated minimum wear features suggesting a hypersensitivity cause.[3]

Therefore the origin of pseudotumours is probably multifactorial caused either by excessive wear, metal hypersensitivity, a combination of the two, or as yet an unknown cause. Pseudotumor-like reactions have also been reported in non-metal-on-metal bearings. In these cases, the histological findings showed accumulations of macrophages and giant cells, again suggesting an excessive wear origin.

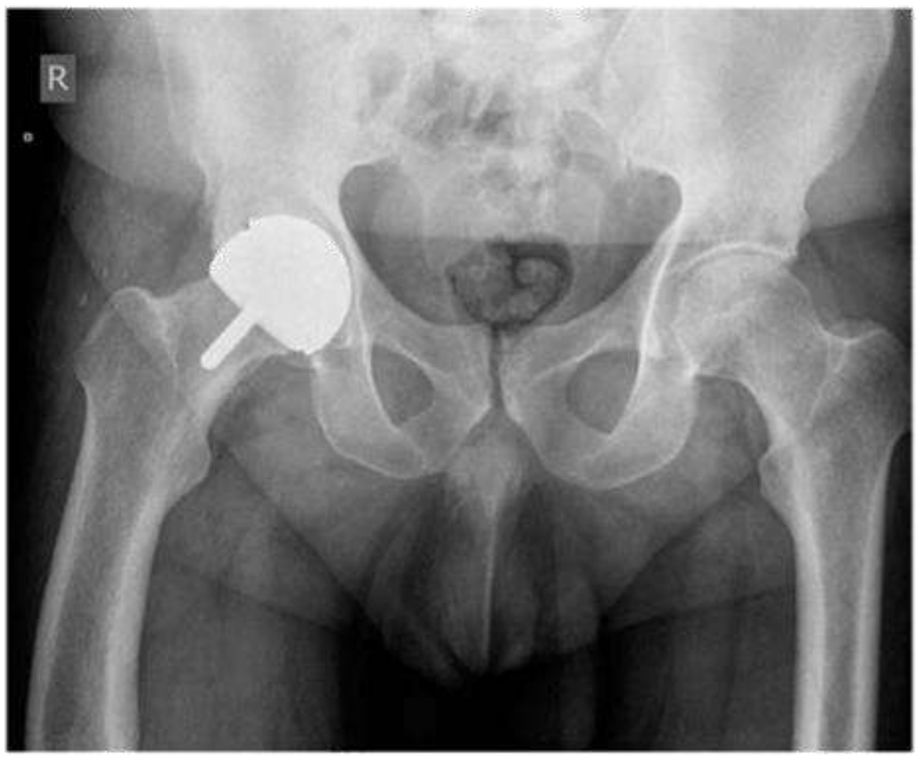


Figure 2.4 Anteroposterior (AP) radiograph right metal-on-metal hip resurfacing implant.

EXAMINER: What are the risk factors for pseudotumours?

CANDIDATE: Significant risk factors for the development of pseudotumor include female sex, age less than 40 years, small component size, hip dysplasia and specific implant designs (ASR).

EXAMINER: How are you going to investigate this patient?

CANDIDATE: A careful history and examination of the patient is required. It is crucial to determine if the pain is arising from intrinsic (indicating hip pathology) or extrinsic sources (referred pain).

Extrinsic sources would include referred pain from the spine or pelvis, peripheral vascular disease, stress fracture, tendinitis or bursitis about the hip.

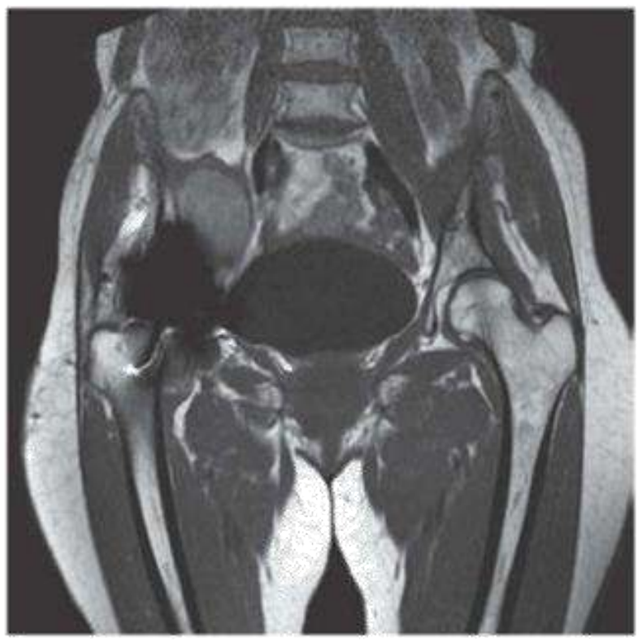
Intrinsic causes include aseptic loosening, avascular necrosis, infection [Long pause].

EXAMINER: What does the British Hip Society recommend [Prompt]?

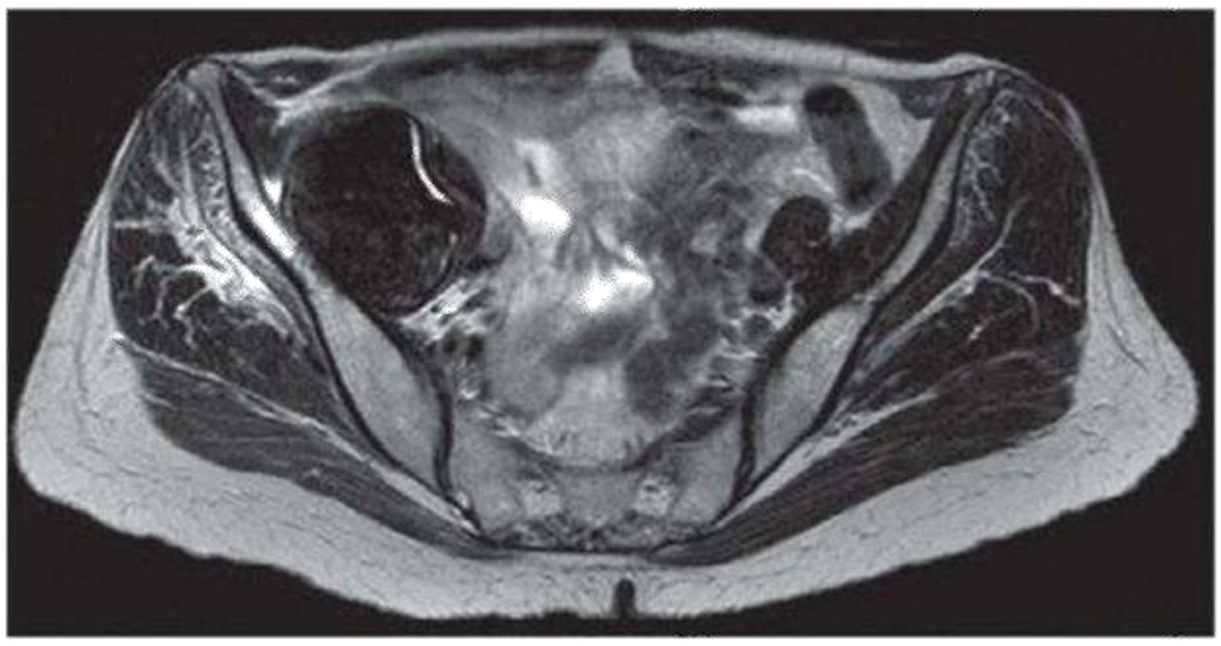
CANDIDATE: Blood cobalt and chromium ions should be measured, as these are indicators of surface wear. If levels are raised the patient will require close observation. If levels are rising and the hip is painful it may be sensible to consider revising the implant.

I would also order an MRI scan with metal artifact reduction sequences (MARS). This is operator dependent but

(a)



(b)



Figures 2.5a and 2.5b MRI of right MOM hip resurfacing implant demonstrating ALVAL mass.

can give clear images of fluid collections or solid lesions (pseudotumours) around the hip.

EXAMINER: This is the MRI scan obtained. What does it show? (Figure 2.5.)

CANDIDATE: The MRI is a T2-weighted image coronal view, which demonstrates an intra-pelvic mass.

EXAMINER: This was a pseudotumour. In fact the mass could be felt clinically when examining the abdomen.

EXAMINER: What are you going to do?

CANDIDATE: This patient requires urgent revision surgery to the hip.

EXAMINER: She is very scared of surgery and would prefer to avoid it.

CANDIDATE: I would stress the important of early revision surgery as the longer the MOM resurfacing implant is left in place the more extensive the soft tissue destruction will most likely be.

EXAMINER: What are the principles of surgery for pseudotumours?

CANDIDATE: The pseudotumour needs to be managed with aggressive debridement of all involved soft tissue. It is important to do a thorough debridement of the abnormal tissue similar to the treatment of infection. The surgery should be preformed by an experienced hip surgeon.

Although she is still relatively young I would use a metal-on-polyethylene bearing surface. A ceramic bearing surface has the potential for catastrophic fracture. We are already revising for a rare complication and we don't want anything to go wrong again. However I would use an uncemented implant. I would keep the option of using a constrained cup open as the soft tissues may be so poorly compromised that the hip is unstable but obviously would prefer to avoid this, as components will loosen early in this situation.

It would be sensible to get a second opinion from an experienced hip surgeon as per British Hip Society guidelines to confirm and support the appropriateness of the management plan.

EXAMINER: Why bother with MOM hip resurfacing procedures? The old Charnley cemented hip replacement with trochanteric osteotomy works equally well with excellent long-term results reported from the surgeons at Wrightington.

CANDIDATE: Advantages of MOM hip resurfacings include better restoration of hip biomechanics, improved proprioceptive feedback, improved wear characteristics with no PE-induced osteolysis, increased levels of postsurgical activity, greater range of movement, reduced risk of dislocation, improved femoral bone stock mass because the neck and most of the head are retained and ease of conversion to a THA if the implant should fail.

EXAMINER: What are the contraindications for resurfacing?

CANDIDATE: These include severe osteoporosis, insufficient bone stock in the femoral head, large cysts at the femoral neck or head, a narrow femoral neck, notching of the femoral neck and severe obesity (BMI $> 35\,kg/m^2$).

Other contraindications include a history of chronic renal disease, metal hypersensitivity, those with anatomical abnormalities in the acetabulum or proximal femur and certainly caution in women of childbearing age.

EXAMINER: Is resurfacing contraindicated in women of childbearing age?

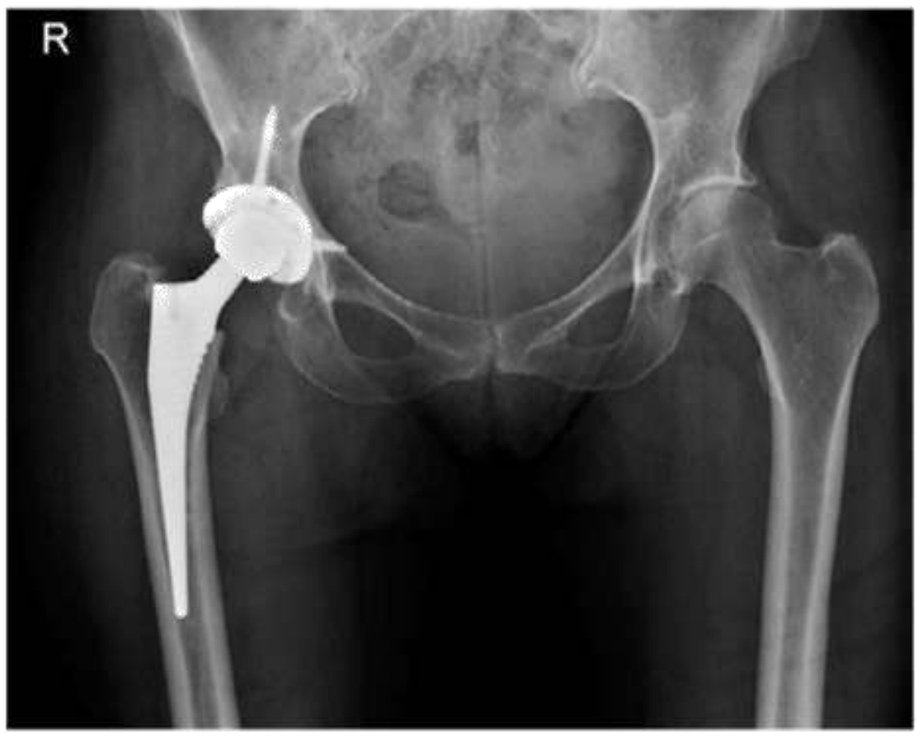


Figure 2.6 Anteroposterior (AP) radiograph of revised hip demonstrating uncemented THA with screw fixation into acetabulum.

CANDIDATE: No, although a recent annotation by De Smert in the JBJS British Edition reported that two-thirds of surgeons would exclude patients of childbearing age. Most surgeons (89%) believed that women should not be excluded.[4]

EXAMINER: I think most hip surgeons would now avoid a resurfacing procedure in a female regardless of whether they were of childbearing age or not.[5]

These are her postoperative radiographs. We kept her non-weightbearing for 6 weeks as there was quite an extensive anterior wall defect in the acetabulum but she has done very well. The hip pain has settled and the abdominal mass resolved. We were very lucky as the extensive soft tissue destruction that sometimes can be seem with this condition was absent. (Figure 2.6.)

EXAMINER: What are the outcomes of hip resurfacing compared with conventional THA?

CANDIDATE: Several recent studies report identical Harris hip scores but a greater percentage of patients with resurfacing involved in high demand activities. There is a higher revision rate in hip resurfacing compared with conventional THA.[6]

EXAMINER: What factors are associated with higher revision rates for hip resurfacing procedures?

CANDIDATE: These would include AVN, hip dysplasia, female sex, inflammatory arthritis, increased age, a small femoral implant and specific implant designs.

Endnotes

1. Occasionally if an examiner doesn't know what a candidate is discussing they will enquire further. Equally the examiner may let it pass so as not to reveal their own knowledge gap. Skilful, wily candidates may be able to bait and tempt the examiner into asking for clarification so as to then appear very studious and knowledgeable. Be careful however as there is a very real danger you may irritate the examiners by coming across as a 'know it all'.
2. An indirect way of letting the examiners know that you have read the various guidelines.
3. Campbell P, Ebramzadeh E, Nelson S *et al.* Histological features of pseudotumor-like tissues from metal-on-metal hips. *Clin Orthop Relat Res* 2010;**468**:2321–2327.
4. De Smet K, Campbell PA, Gill HS. Metal-on-metal hip resurfacing: a consensus from the Advanced Hip Resurfacing Course, Ghent, June 2009. *J Bone Joint Surg Br* 2010;**92**:335–336.
5. MOM hip resurfacing implants are being used much less now than previously. Whether this is an over-reaction to the ASR or not time will tell. However, from the exams perspective be very careful with what you are going to say or recommend to the examiners. Know the current guidelines and literature! Large MOM Jumbo hip replacements are now contraindicated as a primary procedure due to metal wear and corrosion at the trunnion.
6. Huo MH, Stockton KG, Mont MA *et al.* What's new in total hip arthroplasty? *J Bone Joint Surg Am* 2010;**92-A**:2959–2972.

Structured oral examination question 3

EXAMINER: This is an anteroposterior (AP) radiograph of a 78-year-old man presenting with increasing right hip pain. He had a THA performed 17 years ago.

CANDIDATE: The AP radiograph demonstrates severe osteolysis of both femoral and acetabular components. There are radiolucent lines at the bone–cement interface located circumferentially around all seven DeLee and Charnley zones in the acetabulum. The femoral component has separated from the femoral cement with lucencies in all seven Gruen zones.

The femoral implant is a Stanmore prosthesis and no cement plug has been used in the femur, so-called first generation cementing techniques.

EXAMINER: What do you mean by first-generation cementing techniques?

CANDIDATE: First-generation cementing techniques involved hand mixing of cement and finger packing of bone cement in the doughy phase into an unplugged, unwashed femoral canal. Clinical results with first-generation cementing have been variable and in general have produced some disappointing results due to the inability to produce a consistent cement mantle.

Second-generation techniques involved plugging the medullary canal, cleaning the canal with pulsed lavage and inserting cement in a retrograde manner using a cement gun. This reduced the incidence of gross voids and filling defects in the mantle.

Third-generation techniques involved porosity reduction via vacuum mixing or centrifugation and cement pressurization.

Fourth-generation cementing techniques include stem centralization both proximally and distally to ensure an adequate and symmetrical cement mantle. This is important as uneven and excessively thin cement mantles are associated with early failure and revision.

EXAMINER: How is cementing technique graded?

CANDIDATE: The quality of the cement mantle has been described by Harris and Barrack using a scale of A to D.[1]

Complete filling of the medullary cavity by cement, a so-called 'white-out' at the cement–bone interface is graded 'A'. Slight radiolucency of the cement–bone interface is defined as 'B'. Radiolucency involving 50% to 99% of the cement–bone interface or a defective or incomplete cement mantle is graded 'C'. Grade 'C2' is given to a defect where the tip of the stem abuts the cortex with no intervening cement. Radiolucency at the cement–bone interface of 100% in any projection, or a failure to fill the canal with cement such that the tip of the stem is not covered, is classified 'D'.

EXAMINER: What are you going to do?

CANDIDATE: I would want to take a full history from the patient. I would enquire about pain.

I would also want to exclude the possibility of infection (septic loosening) and would ask about problems with the hip postoperatively such as a wound infection requiring washout or a prolonged course of antibiotics. A history of fever, chills or a sinus tract suggests infection. Night pain, rest pain or constant pain would also suggest infection.

With aseptic loosening typically the pain is aggravated by weightbearing. Pain is significant with the first few steps of walking (start-up pain) which improves slightly with further walking only to worsen again with more walking. The pain is always improved with rest and rarely constant.

With aseptic loosening of a THA examination may reveal a shortening of the affected limb, antalgic gait and positive Trendelenberg sign. Pain at the extremes of movement suggests loosening.

It is important to exclude other causes of intrinsic hip pain such as trochanteric bursitis, tendinitis or impingement. Extrinsic sources of hip pain should also be excluded, particularly the lumbar spine especially if the pain has neurogenic features such as radiation below the knee, numbness, paraesthesia or dysaesthesias. Pulses and skin temperature should be checked to rule out a vascular cause for pain.

EXAMINER: Assume there is no infection in the hip and referred causes of pain have been ruled out. What are you going to do?

CANDIDATE: I would assess the patient. Find out how bad the pain is and whether the hip should be revised or whether symptoms are manageable and the patient can be reviewed regularly at the orthopaedic follow-up clinic.

EXAMINER: The patient can only walk about 200 yards before severe pain.

CANDIDATE: I would offer him revision hip surgery provided co-morbidity issues have been optimized and the risks of surgery had been discussed and understood. Both components would need to be revised.

EXAMINER: What are the complications that you would need to mention to the patient when consenting for surgery?

CANDIDATE: I would mention

- Infection.
- Dislocation. Usually component malpositioning or laxity of soft tissues around the hip.
- Fracture/perforation of femoral shaft.
- Nerve palsy (peroneal, sciatic, femoral) 2–7%.
- Vascular injury (femoral, iliac, obturator).
- Leg-length discrepancy.
- Heterotopic ossification.
- Death (cardiac/pulmonary).
- DVT/PE.

In addition the patient is going to require an extended trochanteric osteotomy (ETO) to remove the cement distally and this will increase operating time and blood loss. There is always the concern that the osteotomy site will go on to either malunion or non-union. Osteotomy migration or fracture can also occur.

EXAMINER: What about the bone loss? How do you plan for this?

CANDIDATE: Bone loss can be classified on the femoral side by using either the AAOS (Table 2.1) or the Paprosky classification system (Table 2.2).

The Paprosky classification evaluates the femoral diaphysis for its ability to support an uncemented, fully porous coated prosthesis. It is less detailed than the AAOS classification but is more useful in decision making if an uncemented revision is to be performed.

Table 2.1 AAOS classification system for femoral defects.

I Segmental defect
Proximal (partial or complete)
Intercalary
Greater trochanter
II Cavitary defect
Cancellous
Cortical
Ectasia (dilatation)
III Combined segmental and cavity defect
IV Malalignment
Rotational
Angular
V Femoral stenosis
VI Femoral discontinuity

Table 2.2 Paprosky classification system for femoral defects.

I Minimal metaphyseal cancellous bone loss/normal intact diaphysis
Type I defects are seen after removal of uncemented component without biological ingrowth on surface. Usually seen with Austin Moore type prosthesis or resurfacing procedures. The diaphysis and metaphysis are intact and there is partial loss of the calcar and anteroposterior (AP) bone stock
II Extensive metaphyseal cancellous bone loss/ normal intact diaphysis
Often seen after removal of cemented prosthesis. Calcar deficiency and major AP bone loss
IIIA Metaphysis severely damaged/ > 4 cm diaphyseal bone for distal fixation
Grossly loose femoral component First-generation cementing techniques
IIIB Metaphysis severely damaged/ < 4 cm diaphyseal bone for distal fixation
Type IIIB defects extend slightly further than type IIIA, however reliable fixation can be achieved just past the isthmus of the femur Cemented with cement restrictor Uncemented with substantial distal osteolysis
IV Extensive metaphyseal and diaphyseal bone loss/isthmus non-supportive
Extensive defect with severe metaphyseal and diaphyseal bone loss and a widened canal that cannot provide adequate fixation for a long stem

Table 2.3 AAOS classification system for acetabular defects.

Type I Segmental defects
Peripheral – superior/anterior/posterior
Central – medial wall absent
Type II Cavitary defects
Peripheral – superior/anterior/posterior
Central – medial wall intact
Type III Combined segmental and cavitary bone loss
Type IV Pelvic discontinuity
Separation of anterior and posterior columns
Type V Arthrodesis

Table 2.4 Gross and associates classification system for acetabular bone defects.

Type	Description
I	No substantial loss of bone stock
II	Contained loss of bone stock (columns and/or rim intact)
III	Uncontained loss of bone stock (< 50% acetabulum)
IV	Uncontained loss of bone stock (> 50% acetabulum)
V	Contained loss of bone stock with pelvic discontinuity

Acetabular bone loss

Acetabular defect classification systems are used to predict the extent of intraoperative bone loss and guide reconstructive options.

Several classification systems exist; the three most commonly used are the American Academy of Orthopaedic Surgeons (AAOS) system (Table 2.3), the Gross and associates system (Table 2.4) and the Paprosky classification system (Table 2.5).

Gross and associates classification system (Table 2.4)

This classification is based on the nature of the bone graft needed for reconstruction on standard preoperative AP and lateral radiographs. A bone defect is considered uncontained if morselized bone graft cannot be used to fill the defect.

Table 2.5 Paprosky classification of acetabular bone defects.

Type	Radigraphic finding	Intraoperative finding	Trial stability
I	No cup migration	Intact rim and no distortion. No major osteolysis. Bone loss minimal	Full
	No substantial bone loss	Small focal areas contained bone loss	
		Columns intact	
IIA	Superior (or superomedial) migration of < 3 cm	Superomedial bone loss	Full
	No substantial ischial lysis	Columns supportive and rim intact	
	No substantial teardrop lysis	Migration into defect under thin superior rim	
		Host-bone contact of > 50%	
IIB	Superior (or superolateral) migration of < 3 cm	Uncontained superior rim defect < 1/3	Full
		Columns supportive	
		Host-bone contact of > 50%	
IIC	Medial wall defect	Uncontained medial wall defect	Full
	Cup medial to Kohler line	Rim intact and rim columns supportive	
IIIA	Superolateral cup migration	Unsupportive dome	Partial
	Moderate ischial lysis	Columns intact	
	Partial teardrop destruction	Host-bone contact of 40–60%	
	Kohler line intact		
IIIB	Superomedial migration	Risk of pelvic discontinuity	None
	Severe ischial destruction	Bone contact of < 40%	
	Teardrop loss	Rim defect of > 50%	
	Migration medial to Kohler line		

Paprosky acetabular bone loss classification

This classification is based on information that can be obtained from AP radiographs. Four radiographic criteria are assessed:

1. **Superior migration of the hip centre**
   - Indicates damage to anterior and posterior columns.
   - Superomedial indicates greater damage to anterior column.
   - Superolateral indicates greater damage to posterior column.
2. **Ischial osteolysis**
   - Bone loss inferior posterior column and posterior column.
3. **Teardrop osteolysis**
   - Inferior anterior column and medial wall.
4. **Position of the implant relative to Kohler's line**
   - Deficiency of anterior column and/or medial wall deficiency.

A trial component with full inherent stability does not change position when the surgeon pushes its rim or performs a trial reduction. A trial component with partial inherent stability does not change position with removal of the inserter, but does not withstand the force of pushing on the rim or performance of a trial reduction. A trial component with no inherent stability changes position with the simple act of removing the inserter.

The Paprosky classification (Table 2.5) is often used clinically in preference to the AAOS classification as it not only predicts bone loss encountered intraoperatively but also assists in determining reconstructive options.

EXAMINER: How would you plan for surgery?

CANDIDATE:

- I would get an anaesthetic review to make sure the patient was fit enough for surgery and risks acceptable and also so they could order any special tests such as echocardiogram or pulmonary function tests etc.
- I would cross match for four units and make sure the cell saver was available.
- I would order one femoral head frozen allograft and have freeze dried allograft available if required.
- Implant removal kit which would include curved and straight osteotomes for the cemented cup and femur, ultrasonic tools, high speed burrs.
- Accurately template the revision implants required taking into account the level of the ETO.
- I would use uncemented components as generally they are preferred if previously cement was used. Cement would be relatively contraindicated if using an ETO as it may get into the osteotomy site and prevent healing.
- A long stem femoral implant, multihole revision (tantulum) acetabular shell and a metal-on-polyethylene bearing surface. I would attempt to use at least 32 mm head but preferably a 36 mm head as this significantly reduces the risk of postoperative dislocation.
- I would need Dall–Miles cables grip system to rewire the ETO back into place.
- I would need a flexible light source for visualizing the medullary canal of the femur.
- I would generally prefer a posterior approach with ETO unless the risk of dislocation was deemed high in which case I would use an anterolateral approach with ETO.

EXAMINER: How do you remove the cemented femoral component?

CANDIDATE: It is important to clear the shoulder of the prosthesis removing any cement or bone overhanging the proximal aspect of the greater trochanter as either stem removal will be obstructed or a greater trochanter fracture will occur with stem removal.

Flexible osteotomes and a small burr can then be used to further disrupt the cement/implant interface.

The ETO will greatly simplify implant and cement removal. I would use cement splitters to remove cement along with ultrasonic tools. Cement is split radially and then removed.

EXAMINER: What about the acetabular component?

CANDIDATE: The safest way is to disrupt the PE cup from the cement using curved gouges. This prevents inadvertent damage to the bone of the acetabulum bed. After removal of the cup the cement is removed piecemeal. Sometimes a threaded extractor through a drill hole in the PE can be used. High-speed burrs are sometimes needed to debulk cement within acetabular anchoring holes.

Endnote

1. Barrack RL, Mulroy RD Jr, Harris WH. Improved cementing techniques and femoral component loosening in young patients with hip arthroplasty: a 12 year radiographic review. *J Bone Joint Surg Br* 1992;74:385–389.

This is a classic hip paper. You should know the key message, relevance and why it is important.

Structured oral examination question 4

EXAMINER: These are the radiographs of a 78-year-old lady who has been referred to the orthopaedic clinic by her GP because of increasing pain in her right hip. Would you care to comment on the radiographs? (Figure 2.7.)

CANDIDATE: This is an AP radiograph, demonstrating lower lumbar vertebrae, both hips and proximal femur. The most obvious features in the right hip are loss of joint space, osteophytes, sclerosis and bone cysts. The radiographic features are highly suggestive of osteoarthritis (OA) of the hip.

EXAMINER: How is osteoarthritis classified?

CANDIDATE: OA is classified into primary OA when obvious cause can be identified and secondary OA caused by such conditions as avascular necrosis, DDH, post traumatic, Paget's disease, slipped capital femoral epiphysis, protrusio acetabuli, Perthes' disease.

EXAMINER: What are the percentages of each type of OA?

CANDIDATE: Various studies have suggested that almost 90% of cases of OA are secondary.

EXAMINER: How are you going to manage this patient?

CANDIDATE: I would take a full history and examination from the patient, specifically I would want to know the location of pain, exclude referred pain from the spine. Hip pain is

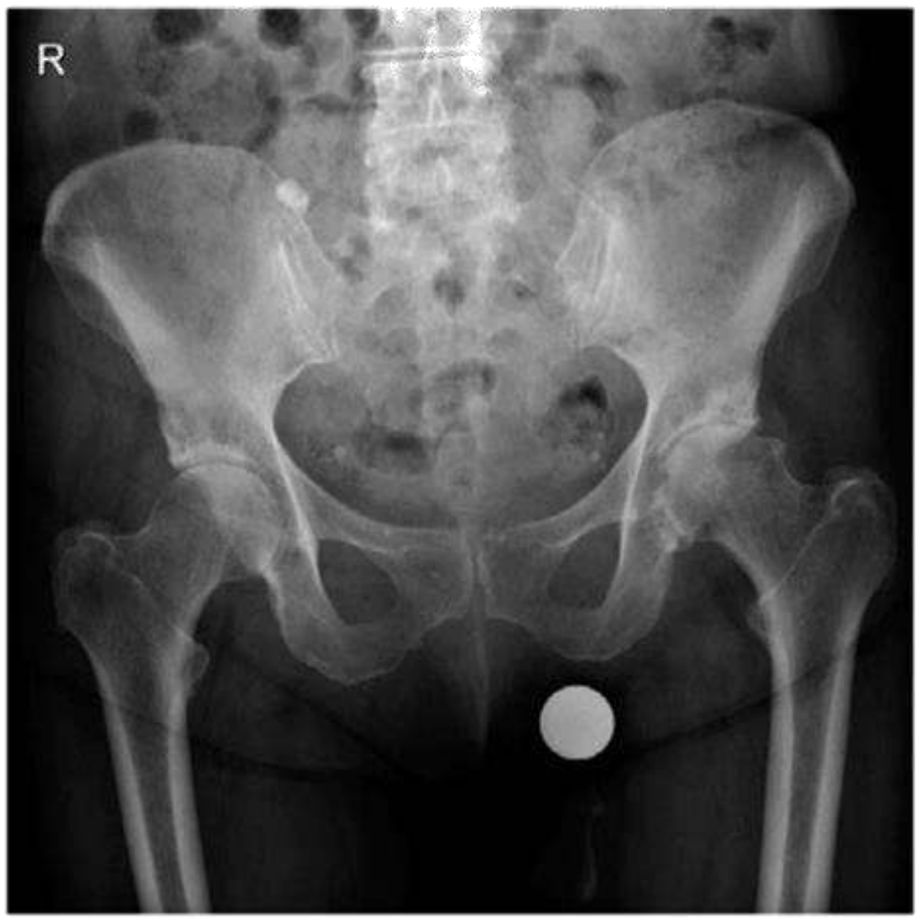


Figure 2.7 Anteroposterior (AP) radiograph demonstrating severe osteoarthritis of the left hip.

classically located in the buttock or groin radiating to the knee. Pain radiating below the knee to the foot is strongly suggestive of radicular type pain from the spine. I would inquire whether the patient had difficulty putting shoes and socks on, tying shoe laces, bending to pick up an object from the floor, getting in and out of a car [Examiner interrupts]

EXAMINER: The patient struggles to walk a quarter of a mile. She has typical symptoms of advanced OA. What are you going to do?

CANDIDATE: Assuming that all conservative options had been tried and have been unsuccessful I would offer her THA.

EXAMINER: What type of hip arthroplasty would you perform?

CANDIDATE: I would perform a cemented Exeter THA.

EXAMINER: Why this particular implant?

CANDIDATE: The Exeter THA has excellent peer-reviewed long-term data. It has a 10A ODEP rating for clinical use. It is an implant that I am very comfortable using, I have been trained to use this implant by my consultants, the instrumentation is straightforward and simple to use, the neck cut is not critical and the introducer allows for even pressure when inserting the implant.

EXAMINER: What are the design principles of the Exeter Stem?

CANDIDATE: The Exeter implant is a loaded taper model and becomes lodged as a wedge in the cement mantle during axial loading, reducing peak stresses in the proximal and distal cement mantle. The stem is allowed to subside initially until radial compressive forces are created in the adjacent cement and transferred to the bone as hoop stresses.

EXAMINER: What approach would you use to the hip?

CANDIDATE: I am happy to use either the Hardinge or posterior approach to the hip.

EXAMINER: Make up your mind. Which one are you going to do?

CANDIDATE: For the majority of cases I would prefer to use the posterior approach to the hip. In rare instances I would use a Hardinge anterolateral approach if the risk of dislocation was high such as neurological or muscular weakness around the hip (Parkinson's disease/CVA), early dementia or substance abuse.

The posterior approach is considered easier to perform and is generally a quicker procedure, limiting operative complications such as blood loss and anaesthetic issues.

The abductor muscles are not disturbed significantly so there is generally no gait abnormality but the acetabulum is more difficult to see and can make prosthesis positioning difficult, possibly causing an increased dislocation rate due to component malpositioning. The sciatic nerve is at slightly more risk of being injured as well.

EXAMINER: There is approximately double the risk of sciatic nerve injury using the posterior approach. Most surgeons would say that there is no significant difference in surgical time between the two approaches; the posterior approach can take just as long as the anterolateral approach. The posterior approach is marginally technically easier than the anterolateral approach but this also depends on surgeon training, experience with using either approach and personal preference. I would argue about the acetabulum being less easy to visualize posteriorly as most surgeons believe the posterior approach provides better acetabular visualization especially for revision cases. The pelvis tends to tilt more and so the degree of cup anteversion is usually underestimated leading to an increased risk of dislocation. Where I think the posterior approach does make a difference is a reduced incidence of Trendelenberg gait postoperatively and improved Harris hip scores compared with the anterolateral approach. Whilst results have been a bit contradictory the risk of posterior dislocation is slightly higher posteriorly even with a careful repair of the soft tissues. Larger head sizes are being used now so this is becoming less of an issue.

EXAMINER: Talk me through the posterior approach to the hip.

CANDIDATE: Assuming full informed consent has been obtained, all relevant case notes and radiographs have been obtained, the leg has been marked and she has been suitably anaesthetized I would position the patient laterally, affected leg uppermost, with hip supports. I would then prepare and drip the patient and make an incision centred over the greater trochanter, approximately 15 cm in length.

I would cut through the skin, subcutaneous tissue, and open up the fascia lata, splitting the gluteus maximus along the line of muscle fibres, and then release the short external rotators from the greater trochanter. Finally, I would perform a capsulectomy and then dislocate the hip.

I would protect the sciatic nerve being aware of its position and avoid dissecting too near to it.

EXAMINER: What are the pathological processes involved in the development of osteoarthritis of the hip?

CANDIDATE: Disruption of the integrity of the collagen network occurs early in OA allowing hyperhydration. The increased water content of cartilage causes softening, decreases Young's modulus of elasticity and reduces its ability to bear load.

Initial changes in OA involve damage to the tangential zone immediately below the articular surface, with disorganization of the collagen network, loss of proteoglycans and swelling. This leads to a hypertrophic repair response with increased synthesis and accumulation of proteoglycan. However the repair process fails with loss of surface integrity, and fibrillation parallel to the surface. In regions of severe damage, there is a loss of cellularity and sporadic formation of cell clusters or clones.

Normal cartilage metabolism is a highly regulated balance between synthesis and degradation of the various matrix components. With OA the equilibrium between anabolism and catabolism is weighted in favour of degradation.

Cartilage catabolism results in release of breakdown products into synovial fluid, which then initiates an inflammatory response by synoviocytes.

These breakdown products include: chondroitin sulphate, keratan sulphate, PG fragments, type II collagen peptides and chondrocyte membranes.

Activated synovial macrophages then recruit PMNs establishing a synovitis. They also release cytokines, proteinases and oxygen free radicals (superoxide and nitric oxide) into adjacent synovial fluid. These mediators act on chondrocytes and synoviocytes modifying synthesis of PGs, collagen, and hyaluronan as well as promoting release of catabolic mediators.

Cartilage changes in osteoarthritis are characterized by increases in:

- Water content.
- Chondrocyte activity and proliferation.
- Stiffness of articular cartilage.
- Interleukin-1.
- Metalloproteinase levels.
- Cathepsins B and D levels.

and decreases in

- Quality of collagen.
- Proteoglycan quality and size.

Histology classically demonstrates:

- Loss of superficial chondrocytes.
- Replication and breakdown of the tidemark.
- Fibrilation.
- Cartilage destruction with eburnation (polished, shiny smooth with an appearance like ivory) of subchondral bone.

EXAMINER: Is OA simply an ageing process of cartilage?

CANDIDATE: Several differences between ageing cartilage and OA cartilage have been described suggesting a separate disease entity. For example OA and normal ageing cartilage differ in the amount of water content and in the ratio of chondroitin sulphate to keratin sulphate constituents.

EXAMINER: [Interrupting] That's fine that's okay.[1] What molecules are responsible for degrading the cartilage matrix?

CANDIDATE: The primary enzymes responsible for the degradation of cartilage are the matrix metalloproteinases (MMPs). These enzymes are secreted by both synovial cells and chondrocytes and are categorized into three general categories: (a) collagenases, (b) stromelysins and (c) gelatinases.

In OA, synthesis of MMPs is greatly enhanced and the available inhibitors are overwhelmed, resulting in net degradation. Interestingly, stromelysin can serve as an activator for its own proenzyme, as well as for procollagenase and prostromelysin, thus creating a positive feedback loop of pro-MMP activation in cartilage.

EXAMINER: What factors are responsible for inducing metalloprotease synthesis?

CANDIDATE: IL-1 is a potent pro-inflammatory cytokine that, in vitro, is capable of inducing chondrocytes and synovial cells to synthesize MMP. In addition IL-1 suppresses the synthesis of type II collagen and proteoglycans. Therefore in OA, IL-1 actively promotes cartilage degradation and may also suppress attempts at repair.

Endnote

1. Know the biochemical differences between ageing and osteoarthritis in cartilage as your examiners may want you to continue answering the question.

Structured oral examination question 5

EXAMINER: This is the anteroposterior (AP) radiograph of a 48-year-old man who presents to your clinic with several weeks' history of progressively worsening bilateral hip pain. What do you think of the radiograph? (Figure 2.8.)

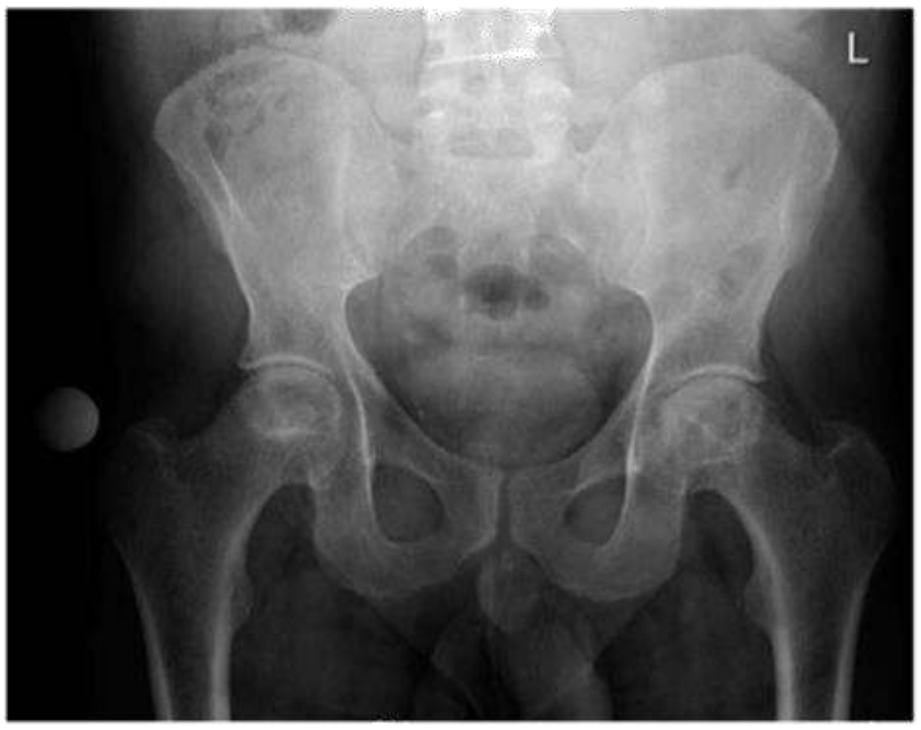


Figure 2.8 Anteroposterior (AP) radiograph of bilateral avascular necrosis.

CANDIDATE 1: This is an anteroposterior (AP) view of the pelvis. The most obvious abnormality is patchy diffuse sclerosis with increased density in the superolateral aspect of the right femoral head (Ficat 2).

The left femoral head has a possibly minimal osteoporosis and/or blurring and poor definition of the bony trabeculae (Ficat 1). The radiograph is suspicious of bilateral AVN. I would like to obtain a frog-leg lateral radiograph of both hips. I would look for the crescent sign, indicating subchondral fracture, a feature of AVN that is more obvious on a frog-leg lateral than AP projection. This is because the anterior and posterior margins of the acetabulum on the AP projection are superimposed over the superior portion of the femoral head, the usual location of the sign. When AVN is bilateral, it usually occurs in each hip at different times, and the staging of disease in each hip is often different. [Candidate score 7–8]

CANDIDATE 2: This is an AP pelvic radiograph showing both hips. There is nothing very obvious staring at me. There are no features of osteoarthritis such as joint space narrowing, osteophytes or sclerosis.[1] [Candidate score 4]

EXAMINER: What do you mean by AVN?

CANDIDATE: Avascular necrosis occurs due to interruption of the blood supply to the femoral head leading to ischaemia and cellular death.

EXAMINER: What is the aetiology of AVN?

CANDIDATE: A number of conditions are associated with AVN. The most common cause is trauma secondary to fracture and/or dislocation of the femoral head. Other conditions include:

- Corticosteroid use.
- Alcohol abuse.
- Smoking.
- Coagulopathies.
- Sickle cell anaemia.
- Caisson disease.
- Hypercholesterolaemia.
- Organ transplantation.
- Systemic lupus erythematosus.
- Gaucher's disease.
- Hypertriglyceridaemia.
- Intramedullary haemorrhages.
- Chronic pancreatitis.

AS IT GRIPS 3Cs (mnemonic)

Alcohol
Steroids
Idiopathic
Trauma
Gout, Gaucher's
Rheumatoid/radiation
Infection/increased lipids/inflammatory arteritis
Pancreatitis/pregnancy
SLE/sickle cell/smoking
CRF/chemotherapy/Caisson disease

In approximately 10–20% of cases no cause can be identified.

EXAMINER: What is the pathophysiology of AVN?

CANDIDATE: Aetiological factors in AVN are usually related to underlying pathological conditions that alter blood flow, leading to cellular necrosis and ultimately to collapse of the femoral head. This damage can occur in one of five vascular

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areas around the femoral head: arterial extraosseous, arterial intraosseous, venous intraosseous, extravascular intraosseous and extravascular extraosseous.

1. Extraosseous arterial factors are the most important. The femoral head is at increased risk because the blood supply is an end-organ system with poor collateral development. Blood supply can be interrupted by trauma, vasculitis (Raynaud's disease), or vasospasm (decompression sickness).
2. Intraosseous arterial factors may block the microcirculation of the femoral head through circulating microemboli. These can occur in sickle cell disease (SCD), fat embolization or air embolization from dysbaric phenomena.
3. Intraosseous venous factors affect the femoral head by reducing venous blood flow and causing stasis. These factors may accompany conditions such as Caisson disease, SCD or enlargement of intramedullary fat cells.
4. Intraosseous extravascular factors affect the hip by increasing the pressure, resulting in a femoral head compartment syndrome. For example: fat cells hypertrophy after steroid administration or abnormal cells, such as Gaucher and inflammatory cells, can encroach on intraosseous capillaries, reducing intramedullary circulation and contributing to compartment syndrome.
5. Extraosseus extravascular (capsular) factors involve the tamponade of the lateral epiphyseal vessels located within the synovial membrane, through increased intracapsular pressure. This manifests as trauma, infection and arthritis, causing hip effusion that may affect the blood supply to the epiphysis.

EXAMINER: Specifically how do steroids cause AVN?[2]

CANDIDATE: The mechanism postulated for steroid-induced AVN is still unclear.

Johnson proposed that fat cell hypertrophy within the bone marrow increases femoral head pressure resulting in sinusoidal vascular collapse and necrosis of the femoral head.[3] The exact mechanism of fat cell hypertrophy remains obscure but a disorder in fat metabolism is implicated.

Jaffe *et al.* believe patients undergoing steroid treatment are in a hyperlipidaemic state, which can increase the fat content within the femoral head and raise intracortical pressure producing sinusoidal collapse and finally necrosis.[4] Other investigators have proposed that this hyperlipidaemic state leads to fat embolism occluding the femoral head microvasculature, which initiates the pathophysiological process.[5] A recent study in rabbits suggests that the use of steroids can also damage endothelial and smooth muscle cells within the vasculature. This may result in interruption of the venous drainage from the femoral head, leading to blood stasis, an increase in intraosseous pressure and AVN.[6] Other studies suggest primary osteocyte cell death without any other features. This is seen with steroid use, in transplant patients and those who consume significant amounts of alcohol.

EXAMINER: How common are steroids as a cause of AVN?

CANDIDATE: High-dose corticosteroids are the most common cause of non-traumatic AVN accounting for 10–30% of cases. However only 10% of patients exposed to corticosteroids may develop AVN. Dosage is typically steroids > 2 g of prednisone, or its equivalent, within a 2–3-month period.

The period from the start of corticosteroid treatment to the diagnosis of AVN ranges from 1–16 months (mean 5.3 months), and the majority of patients are diagnosed within 1 year.

EXAMINER: You mentioned the crescent line, what is its significance?

CANDIDATE: Therapeutic interventions are less likely to halt progression of the disease once this sign appears.

EXAMINER: How does AVN of the hip present?

CANDIDATE: Although AVN can be clinically silent typically a patient complains of pain, usually localized to the groin area but occasionally to the ipsilateral buttock and knee. It is usually a deep intermittent, throbbing pain, with an insidious onset that eventually occurs at rest and may be present or even worsen at night. Physical examination reveals pain with both active and passive range of motion, especially with passive internal rotation. Range of motion is important as this helps determine the extent of the disease. In general, more limited flexion and abduction indicate more extensive articular damage, whereas limited rotation alone may indicate less destruction. A careful examination of the contralateral hip should always be undertaken as AVN is bilateral in 40–80% of cases.

EXAMINER: How is AVN classified?

CANDIDATE: Several classification systems for AVN exist. Ficat and Arlet is the most commonly known and consisted of four stages.[7] Hungerford and Lennox later added a fifth stage (Stage 0) when MRI became available.[8]

Stage 0 (preclinical). Suspected disease in the contralateral hip when the index joint has definitive findings. No clinical symptoms. MRI non-diagnostic.

Stage I (pre-radiological). Normal findings on radiographs and positive findings on MRI or bone scan. The MRI shows a double-line sign, consistent with a necrotic process.

Stage II (pre-collapse). Osteopenia, demineralization, sclerosis or cysts. A late finding is the crescent sign, a linear subcortical lucency, situated immediately beneath the subcortical bone, representing a fracture line and impending femoral head collapse.

Stage III (collapse). The femoral head is flattened and collapsed with the presence of sequestration manifested by a break in the articular margin without acetabular involvement.

Stage IV (progressive degenerative disease). Severe collapse and destruction of the femoral head, acetabular osteophytes. Osteoarthritis superimposed on a deformed femoral head.

EXAMINER: Any other classification systems?

CANDIDATE: Steinberg (Table 2.6) expanded the staging system into seven stages and quantified the amount of involvement of the femoral head into mild (< 15%), moderate (15–30%) and severe (> 30%), based on radiographs.[9] 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.

EXAMINER: Any others?

CANDIDATE: Other classification systems include the ARCO (Association Research Circulation Osseous) classification, University of Pennsylvania system and the Mitchell MRI classification.

EXAMINER: What is the Kerboull necrotic angle and its importance?

CANDIDATE: The Kerboull 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 Kerboull angle > 200° more commonly have poor results with certain bone-preserving procedures.

EXAMINER: How are you going to manage this patient?

CANDIDATE: I would perform bilateral core decompression. The AVN is still at an early stage where it may be successful (Ficat stage I and II AVN). The procedure has no role in the management of Ficat stage III or IV disease. Results have been satisfactory when core decompression is combined with either non-vascularized or vascularized fibular grafts in patients with Ficat stage II lesions.

EXAMINER: What are the prerequisites for performing a free vascularized fibular graft (VFG)?

Table 2.6 Staging system of Steinberg *et al.*

Stage	Radiographic feature
0	Normal X-ray findings; normal bone scan and MRI. Diagnosed on histology
I	Normal X-ray findings; abnormal bone scan and/or MR findings IA: Mild (< 15% of femoral head affected) IB: Moderate (15–30% of femoral head affected) IC: Severe (> 30% of femoral head affected)
II	Cystic and sclerotic changes in the femoral head IIA: Mild (< 15% of femoral head affected) IIB: Moderate (15–30% of femoral head affected) IIC: Severe (> 30% of femoral head affected)
III	Subchondral collapse (crescent sign) without flattening IIIA: Mild (< 15% of femoral head affected) IIIB: Moderate (15–30% of femoral head affected) IIIC: Severe (> 30% of femoral head affected)
IV	Flattening of femoral head IVA: Mild (< 15% of surface and < 2-mm depression) IVB: Moderate (15–30% of surface or 2- to 4-mm depression) IVC: Severe (30% of surface)
V	Joint narrowing and/or acetabular changes (this stage can be graded according to severity)
VI	Advanced degenerative changes

CANDIDATE: VFG for AVN is a major operative procedure with a long rehabilitation time and therefore patient selection to minimize the potential for an unsuccessful operation is critical.

McKee from Toronto suggests the operation should be limited to patients:[10]

1. With 2 mm or less of femoral head collapse as measured on plain radiographs.
2. Who are 45 years of age or younger (and have a reasonable life expectancy).
3. Have had withdrawal of an identified aetiological agent.
4. Have no contractures about the hip.
5. Have a supple joint.

These are obviously general guidelines that may be adjusted somewhat depending on the individual patient.

EXAMINER: What are the advantages of performing a free vascularized fibular graft (VFG)?

CANDIDATE: Advantages of vascularized fibular grafting include:

- Being able to perform a core decompression of the femoral head.
- The ability to perform curettage and removal of the osteonecrotic focus.
- Impaction of autogenous cancellous graft to fill the defect created by removal of the osteonecrotic bone.
- The structural support of the subchondral surface provided by the fibular graft.
- The addition of vascularized bone and blood supply to the area of osteonecrosis enhances the revascularization process.

EXAMINER: What complications can occur with a free vascularized fibular graft?

CANDIDATE: Gaskill *et al.* from a tertiary centre in North Carolina performing a large volume of VFG reported a 16.9% complications rate, 4.3% of complications require reoperation or chronic pain management.[11,12]

Donor site morbidity

- Great-toe flexion contracture (4.3%). Majority asymptomatic noticeable only on clinical examination with the ankle fully dorsiflexed. Occasionally requires z-lengthening of the FHL tendon at the level of the medial malleolus. Flexion contracture of the second and third toes may co-exist in a small number of patients.
- Persistent weakness in the operated extremity (0.6%) either long toe flexors or peroneal group.
- Mild persistent pain and tenderness at the ankle or distal osteotomy site (4.1%) usually after prolonged standing or moderate activity such as jogging.
- Sensory deficits (1.7%). The sensory deficit was not always consistent with peripheral nerve or dermatomal distributions.
- Superficial infection.

Graft site complications

- Symptomatic lateral pin migration (2.4%). A Kirschner wire was used routinely to secure the fibular graft in its final position after placement in the femoral head.
- Symptomatic heterotopic ossification (1.4%).
- Femoral fracture (0.7%). All occurred in the intertrochanteric and subtrochanteric region after a fall.
- Deep venous thrombosis (0.3%).
- Superficial infection (4%).
- Deep infection (4%).
- Haematoma (1%).
- Trochanteric bursitis (1%).

EXAMINER: What are the other techniques that can be used to manage AVN hip?

CANDIDATE: The trapdoor procedure is performed with an arthrotomy to dislocate the hip anteriorly, followed by curettage of the necrotic segment of the head and packing of the defect with iliac crest bone graft through a cartilage window in the femoral head. This can be used for Ficat stage III and early Ficat stage IV and reasonable results reported.

EXAMINER: You have to be more specific than that – what do you mean by reasonable results?[13]

CANDIDATE: Michael Mont reported on a series of 30 hips Ficat stage III/IV at 5 years with 73% having good to excellent results.[14]

EXAMINER: Any other options?

CANDIDATE: Osteotomy has been used to treat Ficat stage III and IV disease but results have been variable because it is difficult to rotate the necrotic segment out from the weightbearing area, especially when the lesion is large. Sugioka *et al.* reported good to excellent results at 3 to 16 years of follow-up in 78% of 229 hips treated with the transtrochanteric anterior rotational osteotomy.[15] Their results with this technically demanding procedure have not been reproduced by others.

A success rate of approximately 30% at 5 years is common, with the best results reported in patients whose lesions do not result from trauma and who have less than 30% of the head involved.

EXAMINER: Any new technique that has emerged in the last 2 or 3 years?

CANDIDATE: Stem cells have been used to manage AVN.

EXAMINER: Go on – do you know about the technique or results?

CANDIDATE: Sorry that's all I know.

EXAMINER: Two techniques are being promoted. One is a three-stage procedure and the other is a single-stage procedure.

The first method is by stem cell culture in the lab to multiply the number of cells several million fold. These cultured stem cells are reinjected into a previous core decompression site.

In the second method, bone marrow obtained from the pelvis is centrifuged in the operating room to yield a bone marrow concentrate rich in stem cells. The patient is supine on a traction table with a C arm image intensifier. Percutaneous core decompression drilling with a Kirschner wire (diameter 2.7 mm) is performed to perforate the interface between the necrotic lesion and healthy bone. Following this concentrated

autologous bone marrow aspirate is slowly transplanted into the necrotic area under fluoroscopic control. This is still an experimental procedure but early results seem promising for early disease.

EXAMINER: The patient had surgery on both hips. These are his postoperative radiographs.

CANDIDATE: The AP radiograph demonstrates a metal core rod in the right hip. (Figure 2.9.)

EXAMINER: What do we call this?

CANDIDATE: The patient has had a tantalum rod inserted into the femoral head. The implant achieves decompression, supports the subchondral plate of the necrotic areas and probably induces bone regeneration.

EXAMINER: Anything else?

CANDIDATE: The use of a trabecular metal 'AVN rod' has a number of attractive theoretical advantages, including no donor site morbidity, improved rehabilitation, structural support of the femoral head and the potential for 'osseointegration' of the biologically friendly material.

EXAMINER: The patient had core decompression performed on the left hip and a core decompression with tantalum rod inserted in the right hip. He initially got good pain relief from the procedures for about a year or so but he returns to the orthopaedic clinic complaining both hips are now painful. The left side is worse than the right. What do you think of the radiographs?

CANDIDATE: The AP radiograph suggests AVN has progressed.

EXAMINER: What will you do?

CANDIDATE: I would offer him bilateral hip arthroplasty, the left one being more symptomatic first.

EXAMINER: What type of hip replacement would you use?

CANDIDATE: In view of his relatively young age I would perform an uncemented THA with a ceramic bearing surface.

EXAMINER: What are the advantages of using a ceramic bearing surface?

CANDIDATE: The advantages of using a ceramic bearing surface include superior lubrication, friction, and wear properties compared with other bearing surfaces in clinical use. Specifically it is an extremely hard material, very resistant to wear, with a low coefficient of friction, excellent abrasive resistance and excellent wettability properties for

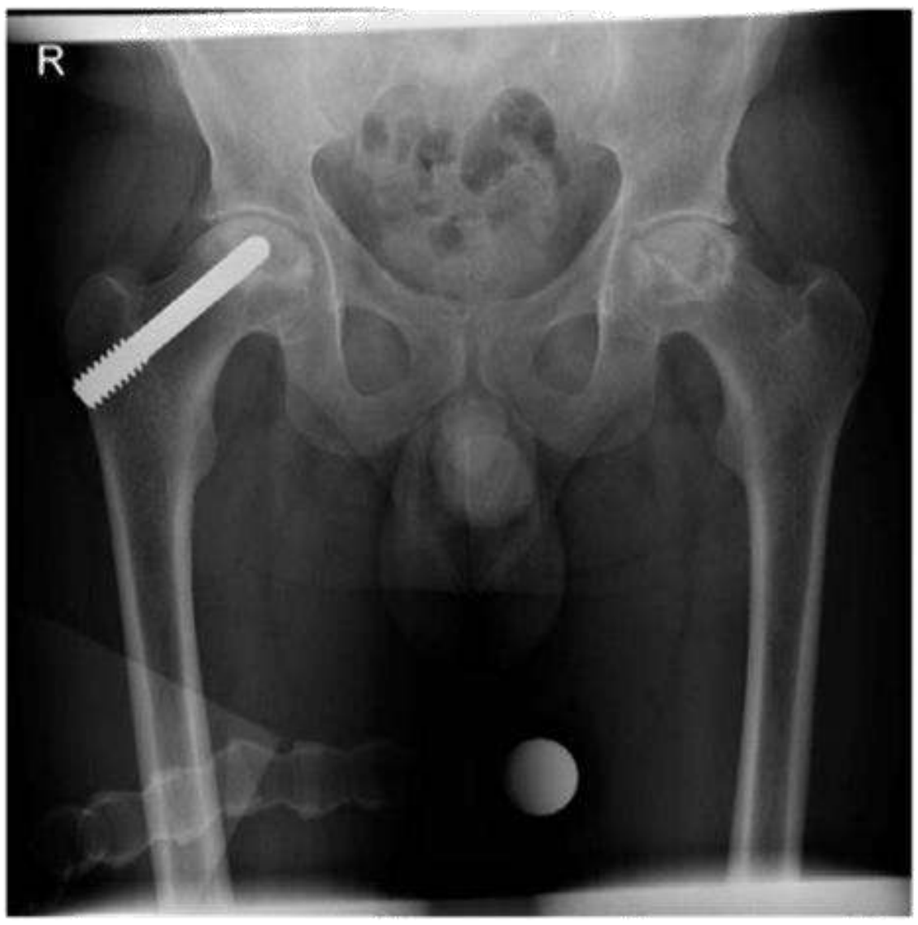

Figure 2.9 Anteroposterior (AP) radiograph pelvis with tantalum rod inserted into the right hip.

improved lubrication. It is presumed that the lower wear rates lead to a lower rate of aseptic loosening and the need for revision surgery.

Disadvantages include potential for catastrophic fracture, squeaking, chipping on insertion and reduced range of implant sizes.

EXAMINER: What is the incidence of squeaking?

CANDIDATE: The reported incidence of squeaking with alumina ceramic bearings varies widely from 0.45% in a series of 2716 ceramic implants to 7.0% in a series of 159 ceramic implants. Most reported series note that squeaking is rare and without clinical significance; however, on rare occasions, major squeaking has led to revision surgery.[16]

EXAMINER: Will there be any special issues removing the tantalum rod and performing THA?

CANDIDATE: I would contact the manufacturers of the implant as there is a special implant removal kit. Otherwise not using the removal kit makes the surgery much more difficult.
I would use a Gigli and reciprocating saw to section the head, implant removal corer to take out the tantalum rod and then perform a conventional uncemented THA.

EXAMINER: Are there any worries with tantalum material?

CANDIDATE: Studies suggest a trend towards an poorer outcome in patients following conversion of tantalum rod to THA.[17]

3. Marrow stromal cells from pagetic lesions have increased RANKL expression.
4. Osteoclast precursor recruitment is increased by interleukin (IL) 6, which is increased in the blood of patients with active Paget's disease and is over-expressed in pagetic osteoclasts.
5. The antiapoptotic oncogene *Bcl-2* in pagetic bone is over expressed.
6. Expression of the proto-oncogene *c-fos*, which increases osteoclastic activity, is increased.
7. Numerous osteoblasts are recruited to active resorption sites and produce large amounts of new bone matrix. As a result, bone turnover is high and bone mass is normal or increased, not reduced.

Endnotes

1. Parvizi J, Schall DM, Lewallen DG, Sim FH. Outcome of uncemented hip arthroplasty components in patients with Paget's disease. *Clin Orthop Relat Res* 2002;**403**:127–134.
2. Lusty PJ, Walter WL, Walter WK, Zicat B. Cementless hip arthroplasty in Paget's disease at medium-term follow-up (average of 6.7 years). *J Arthroplasty* 2007;**22**(5):692–696.
3. Alexakis PG, Brown BA, Howl WM. Porous hip replacement in Paget's disease: an 8–2/3-year follow-up. *Clin Orthop Relat Res* 1998;**350**:138–142.
4. Ludkowski P, Wilson-MacDonald J. Total arthroplasty in Paget's disease of the hip: a clinical review and review of the literature. *Clin Orthop Relat Res* 1990;**255**:160–167.

Structured oral examination question 8

When reviewing various hip topics to include in this chapter DDH was the most common viva question that was regularly asked in the oral viva examination in the last 15 years. We would guess this is because it is a fairly common hip condition with a lot to talk about. The story can go in many different directions.

EXAMINER: These are the anteroposterior (AP) radiographs of a 66-year-old woman with bilateral hip pain. (Figure 2.13.) Would you like to pass comment on them?

CANDIDATE 1: The AP radiographs demonstrate a severely dysplastic hip on the right side with secondary OA changes. On the left side again there is dysplasia but to a lesser degree with again secondary OA changes present. [Candidate score 5]

CANDIDATE 2: This is an AP radiograph of the hips and pelvis of a 66-year-old woman taken on the 16/5/11, which demonstrates severe bilateral dysplasia.[1] There is a high dislocation on the right side, Crowe IV or Hartofilakidis III hip. There is no contact between the true and false acetabulum. The femoral head appears poorly developed and probably absent with the femoral neck articulating against the iliac crest. The view of the proximal portion of the femoral canal on the right side suggests a very narrow medullary canal. On the left side there is a Crowe III hip or Hartofilakidis II hip. There is a low dislocation and secondary osteoarthritis.[2] [Candidate score 6–7]

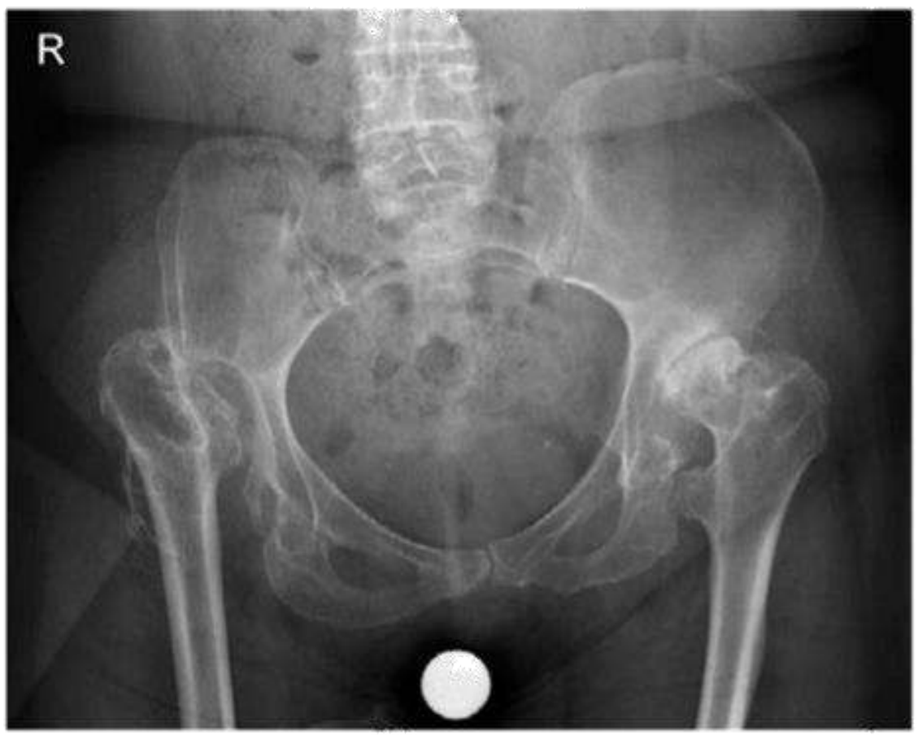

Figure 2.13 Anteroposterior (AP) radiograph of the pelvis of severe bilateral developmental dysplasia of the hip (DDH).

EXAMINER: The left side is a Hartofilakidis I hip as the femoral head is still contained within the original acetabulum. With a low dislocation the femoral head is in contact, at least in part, with the true acetabulum and in this situation this is the most severe deformity. In high dislocation, the femoral head and acetabulum make no contact and the head has migrated superiorly and posteriorly. Often in this situation, the true acetabulum is reasonably well preserved although underdeveloped and osteoporotic.[3]

EXAMINER: What do you mean by dysplasia?

CANDIDATE: Dysplasia is lack of coverage of the femoral head, whether it is subluxed or dislocated.

EXAMINER: How do you classify dysplasia?

CANDIDATE: Crowe classified dysplasia radiographically into four categories based on the proximal migration of the femoral head. The migration is calculated on an AP radiograph by measuring the vertical distance between the inter-teardrop line and the junction between the femoral head and medial edge of the neck.

Crowe I is less than 50% subluxation, Crowe II hips have between 50% and 75% subluxation.

EXAMINER: [Interrupting] That's fine. That's okay. Any other classification systems that you know?

CANDIDATE: [Sharp intake of breath, shaking of head and then silence.] No.

EXAMINER: Have you heard of the Hartofilakidis classification?

CANDIDATE: I have but I can't remember the specifics.

EXAMINER: The Hartofilakidis classification system divides DDH in adults into three types: dysplasia, low dislocation and high dislocation. Many surgeons prefer this system, as it is more practical and simpler to use.

What are the anatomical issues associated with DDH?

CANDIDATE: The anatomical differences are divided into acetabular, femur and soft tissue issues. The acetabulum is shallow and anteverted; the femur has a small deformed head and short anteverted valgus neck.

EXAMINER: That's not all the differences. There are some you have missed. Do you know any more?

CANDIDATE: Muscles around the hip are usually shortened and er, er . . .

EXAMINER: The greater trochanter is small and posteriorly displaced, the femoral canal narrow, the acetabulum is usually small with poor bone quality, hip capsule elongated and redundant, psoas tendon hypertrophied and abductors orientated transversely as a result of the superior migration of the femoral head. The femoral and sciatic nerves may be shortened and therefore more vulnerable to injury during arthroplasty surgery.

EXAMINER: What is the role of a CT scan in planning an operation for DDH?[4]

CANDIDATE 1: CT scans can be used to determine the available acetabular coverage and to estimate the degree of femoral anteversion.

CANDIDATE 2: CT scans are useful in assessing available bone stock, and the morphology, dimensions and orientation of both the acetabulum and femur.

Any leg length discrepancy can be precisely evaluated and allow for design of custom femoral implants.

Various measurements include: femoral neck shaft angle, anteversion of the femoral neck, medial head offset, position of the isthmus and height can be measured.

The AP size of the acetabulum as measured by CT is often different from the supero-inferior size evaluated on plain radiographs.

Proximal femoral anteversion is calculated by measuring the angle between the posterior bicondylar axis and the mediolateral dimensions of the medullary canal 20 mm above the lesser trochanter.

EXAMINER: These measurements are useful to know but how are they actually going to help you to plan surgery?

CANDIDATE: In the acetabulum following the abnormal anatomy too closely might lead to anterior instability if the cup is overanteverted. It is important to recognize that a substantial amount of acetabular anteversion and deformity can be present with a relatively normal-looking AP pelvic radiograph.

In addition femoral anteversion may be difficult to recognize. Even in normal-looking AP radiographs a significant amount of anteversion may be present. Attempting to implant an uncemented stem in a deformed anteverted femur may result in a proximal femoral fracture.

EXAMINER: What are the technical difficulties in performing a THA in a DDH patient?

CANDIDATE: Crowe type II and III hips have a marked superolateral rim deficiency and anterior wall defect. Bulk autografting of the superolateral acetabulum with bone from the femoral head can be used to increase the cover and stability of the acetabular component. The graft and its bed need careful preparation, stable fixation and precise positioning. Graft resorption can occur leading to cup migration and loosening.

Although it is technically difficult for anatomical placement of the acetabular component the forces on the THA are significantly reduced. Linde *et al.* found a 42% rate of loosening of cemented Charnley components after a mean of 9 years if the component was positioned outside the true acetabulum compared with 13% if placed inside.[5,6]

EXAMINER: Any other options to deal with deficient superior coverage of the cup?

CANDIDATE: A small, uncemented cup can be placed in a high hip centre location. In this position the cup is completely covered with host bone and avoids the need for grafting. Disadvantages include decreased polyethylene thickness associated with a small acetabular component, difficulties with correction of leg length inequality and altered hip biomechanics. Hip instability is increased due to

the use of a small femoral head component along with the risk of femoral–pelvic impingement either in flexion or extension.

EXAMINER: What do we mean by cotyloplasty?

CANDIDATE: Cotyloplasty involves a deliberate fracture of the medial wall of the acetabulum in order to place the acetabular component within the available iliac bone. The acetabulum is advanced medially by the creation of a controlled comminuted fracture of the medial acetabular wall. Mixed results have been reported but there is a worry that future revisions may be difficult because issues with restoration of bone stock have not been addressed.[7]

EXAMINER: How do you preoperatively plan for DDH surgery?

CANDIDATE: On the acetabulum side the position of the true acetabulum should be identified and a decision made whether to restore the acetabulum to its true position or not. The degree of anteversion of the acetabulum should be defined as well as the adequacy of bone stock for satisfactory cup fixation and coverage.

Preoperative planning would also include an estimation of the acetabular component size, the preferred method of fixation (cement/uncemented) and need for bone graft.

On the femoral side the size of the femoral canal and the need for special or custom implants should be assessed.

The need for femoral shortening should be made pre-operatively. The method and amount of femoral shortening needs to be worked out beforehand.

Preoperative planning should also include the surgical approach to be used, solutions to deal with the hypoplastic acetabulum and femur, management of LLD and restoration of abductor function.

EXAMINER: What is the effect of anteversion of the femoral stem on THA?[8]

CANDIDATE: When there is more than 40° of anteversion, a corrective rotational osteotomy or a modular implant in which the version of the femoral neck can be varied may be necessary.

EXAMINER: That's not really the question I asked.

CANDIDATE: A large amount of femoral anteversion increases the risk of dislocation.

EXAMINER: That's correct but not the whole story. You have already partly answered the question earlier on.

CANDIDATE: I am sorry, I don't understand.[9]

EXAMINER: Attempting to implant an uncemented stem in a deformed femur may result in a proximal femoral fracture. In this situation you may want to use either a cemented or modular stem that allows control of anteversion. Also excessive anteversion of the femoral component can lead to internal rotational contracture of the hip.

EXAMINER: How do you correct length inequality in DDH?

CANDIDATE: With Crowe type III and IV hips if the cup is placed in the anatomical position femoral shortening is required. Without femoral shortening it is very difficult to reduce the prosthetic head into the acetabular component because of soft tissue contractures.

If one attempts to fully correct a significant leg length discrepancy a sciatic nerve palsy may occur. If permanent this can be a disabling complication from surgery and which patients are less willing to accept these days. The exact amount of lengthening that results in sciatic nerve palsy is not known. Acute limb lengthening of more than 2–4 cm during arthroplasty is associated with an increased risk of neural injury. Therefore as a general rule the hip should be lengthened the minimum amount required to re-establish reasonable function and hip stability. Any lengthening more than 4 cm becomes very risky for a sciatic nerve injury and is generally not advised.

Shortening is performed either by sequential resecting of the proximal femur or by performing a shortening subtrochanteric osteotomy.

Sequential proximal resection results in a small straight femoral tube with a small metaphyseal flare which is usually unsuitable for an uncemented femoral implant. Typically a small cemented DDH stem needs to be used with a straight proximal medial geometry and without a metaphyseal flare.

Advantages of a subtrochanteric shortening osteotomy include preservation of the metaphyseal femoral region (which provides most of the rotational stability of the implant) and allowing concomitant correction of angular and anteversion deformities. It is technically difficult and there is a risk of non-union.

EXAMINER: How do you reduce the risk of non-union?

CANDIDATE: Different subtrochanteric osteotomy geometries can be used. These include transverse, oblique, stepcut and double Chevron osteotomies. A transverse osteotomy is simplest and the resected bone can be used as an onlay graft.

Avoiding the use of a cemented stem prevents the risk of the cement interfering with healing of the osteotomy site. A press fit achieves distal fixation of the prosthesis. Strut allograft and circlage cables may also be needed for support.

EXAMINER: What are the principles of revision hip surgery with DDH?

CANDIDATE: Two major concerns are deficient acetabular bone stock and the position of the acetabular cup particularly if the centre of the hip has not been restored during the primary procedure.

Several surgeons have advocated the use of a high hip centre in order to take advantage of the remaining bone stock and to avoid the use of a structured graft.

However, a high hip centre does not correct leg length discrepancy, does not provide good bone stock for revision hip surgery and is associated with early acetabular loosening and a higher rate of dislocation because of ischial impingement.

The pattern of bone loss associated with DDH is a reduced AP diameter combined with poor superior support. This loss is further increased by surgical bone loss at the time of the index operation, migration of the cup and osteolysis.

Bone graft would need to be ordered along with special equipment such as universal screwdrivers, screw extractors, high speed burrs and metal cutters.

Endnotes

1. It is not unreasonable to mention the patient's age and when the radiograph was taken to the examiners with the first radiograph shown in the viva exam. Just like the trauma viva and the 'I would initially manage the patient with the ATLS protocol' if you keep repeating the catch phrase it will severely annoy the examiners. Once is reasonable to let the examiners know it is part of your standard practice. Any more is irritating and wastes time.
2. The score is 6–7, as the candidate didn't classify the left side correctly. If the candidate had correctly identified a Hartofilakidis I hip it would be more towards a 7–8 mark. The candidate would have correctly used two classification systems to grade the severity of the DDH. He or she has already pre-empted questions on DDH classification.
3. The examiners have the answers in front of them so it is easy for them to point out the differences between a Hartofilakidis I hip and Hartofilakidis II hip. Unless they had a hip interest the examiners probably wouldn't be familiar with this depth of knowledge in everyday clinical practice. Expanding on this it would then be fair to say this level of knowledge is probably not needed as the viva attempts to standardize answers to a newly qualified day one consultant in a DGH. These are guidelines but unfortunately in real life the exam is an artificial situation and never quite that straightforward.
4. This is probably one of the pre-agreed oral viva questions that the examiners need to ask. The examiners have a set standard answer with various bullet points provided so as to be able to mark candidates accordingly.
5. Linde F, Jensen J, Pilgaard S. Charnley arthroplasty in osteoarthritis secondary to congenital dislocation or subluxation of the hip. *Clin Orthop* 1988;**227**:164–171.
6. The candidate's answer isn't particularly well structured.
7. Candidates can either volunteer this extra information or perhaps wait for the examiners to ask it!
8. Technically the candidate hasn't really answered the question.
9. The candidate is not quite getting what the examiner wants and has just gone a bit blank in the stress of the moment.

Chapter 3

Knee structured oral questions

Michael Maru and Deiary F. Kader

Structured oral examination question 1: TKR in valgus knee

EXAMINER: This is a radiograph of a 72-year-old lady complaining of pain and gradual deformity of both knees. She has been referred to your clinic to be considered for total knee arthroplasty. What can you see? (Figure 3.1.)

CANDIDATE: These are weightbearing anteroposterior (AP) views of a 72-year-old lady demonstrating narrowing of joint spaces with bone-on-bone contact in the lateral compartments of both knees. There is early arthrosis affecting the medial compartments of both knees. There is moderate valgus deformity.

EXAMINER: What conditions are associated with this pattern of joint disease?

CANDIDATE: The valgus deformity of the knee with arthritis is commonly seen in women and in inflammatory joint conditions such as rheumatoid arthritis. It can also occur in primary osteoarthritis, overcorrection of high tibial osteotomy (HTO), post-traumatic arthritis following lateral meniscectomy and osteonecrosis.

EXAMINER: What are the perioperative considerations for total knee arthroplasty in valgus knee?

CANDIDATE: The preoperative assessment should include a thorough history and examination to establish if there are any predisposing factors such as rheumatoid arthritis and the success of non-surgical management. The competency of the knee collateral ligaments and degree of deformity correction should be assessed in order to plan on type of implants.
I would use a medial parapatellar because this gives good access to the whole knee and better soft tissue cover. I am aware that a lateral approach can also be used.

EXAMINER: What is the theoretical advantage of a lateral approach?

CANDIDATE: It is a direct approach providing easier access and preserves the neurovascular supply to the extensor mechanism.

EXAMINER: Tell me more about the intraoperative considerations.

CANDIDATE: In valgus knees the lateral femoral condyle is deficient, therefore the femur is internally rotated and tibia is externally rotated. The medial structures are stretched while lateral and posterior structures are contracted. The vastus lateralis acts as a subluxing or dislocating force to the patella. In mild valgus deformity (7–10°) a distal femoral cut of 7° can improve patella tracking and avoid the need for lateral retinacular release. Due to the posterior femoral condyle deficiency, the standard 3° posterior condylar referencing can result in internal rotation of the component. In this situation,

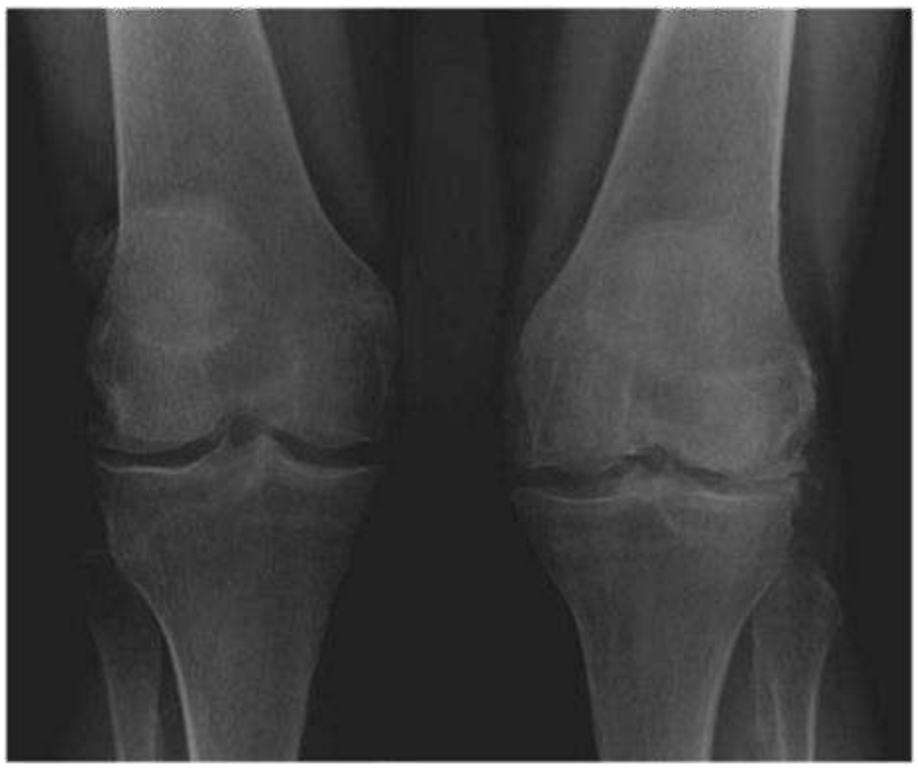

Figure 3.1 Anteroposterior (AP) radiograph bilateral knees.

Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination, ed. Paul A. Banaszkiewicz and Deiary F. Kader. Published by Cambridge University Press.

synthesize and maintain extracellular matrix. The blood supply to the meniscus comes from the lateral, middle and medial geniculate vessels with 20–30% of the peripheral portion being vascular. The main functions of the menisci are load transmission with estimated 50% in extension and 85% in flexion, joint conformity and articular congruity, distribution of synovial fluid aiding nutrition and joint lubrication. The menisci also have proprioceptive function,

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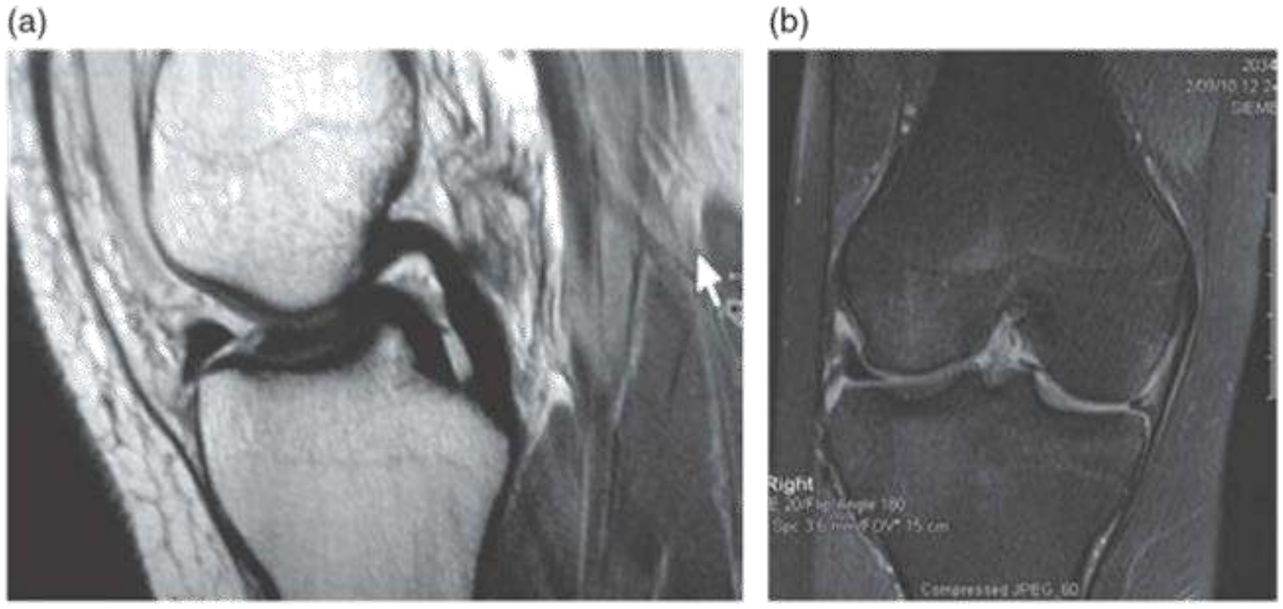
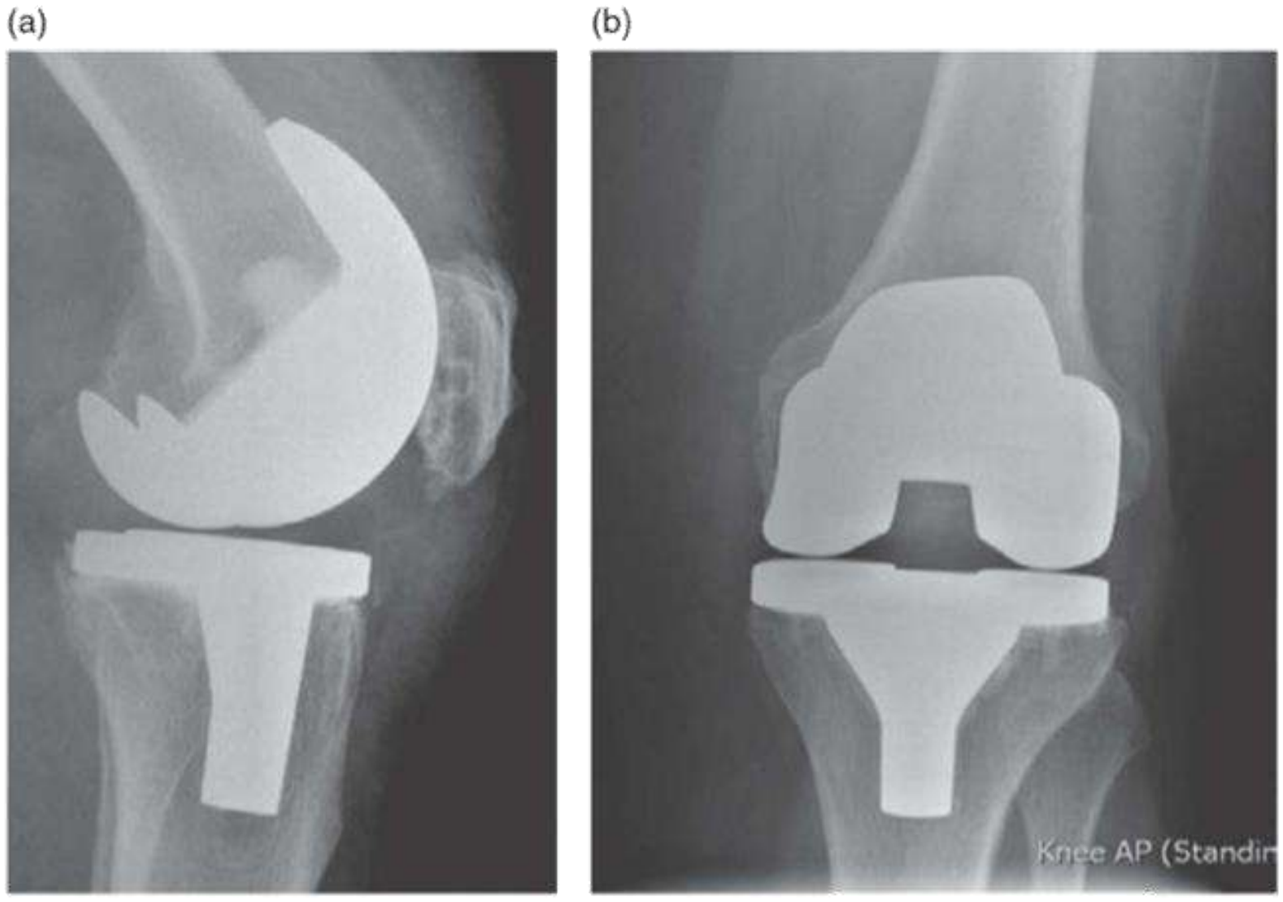
Structured oral examination question 3: Infected total knee arthroplasty (TKA)

EXAMINER: A 78-year-old lady who underwent left TKA 2 years ago is referred to your Painful Arthroplasty Clinic because of increasing pain, stiffness and recurrent swelling of the left knee for 4 months. Prior to onset of symptoms, she was very active



Figures 3.2a and 3.2b T2-weighted sagittal and coronal MRI scan images of knee.



Figures 3.3a and 3.3b Anteroposterior (AP) and lateral radiographs of left TKA.

and enjoyed long-distance walking. She is systemically well. These are the plain radiographs. (Figure 3.3.)

CANDIDATE: This is an AP and lateral radiograph showing a cemented cruciate sacrificing total knee arthroplasty taken on 16/8/11. There is an area of subchondral radiolucency underneath the medial side of the tibial component. There is no obvious periosteal reaction. Both components appear to be well fixed. I would like to see the initial postoperative radiograph and compare it with the most recent radiograph.

EXAMINER: This is the most recent radiograph and there are no other postoperative radiographs available! What would you like to do for this patient?

CANDIDATE: I would start by taking a detailed history of the perioperative events, general health as well current problem. I would like to know the date of index operation, if there was prolonged discharge from the wound, redness or persistent swelling in the immediate postoperative period. A pain-free interval after the operation followed by sudden deterioration may be suggestive of haematogenous spread precipitated by bacteraemia from UTI, URTI or dental procedure. I would also like to know the pattern of pain: mechanical or non-mechanical and whether it's relieved by rest. The clinical examination should be focused on identifying instability and localizing the problem.

EXAMINER: So you think this joint is infected?

CANDIDATE: My working diagnosis is infected TKA. My main differential diagnoses are aseptic loosening, inflammatory arthropathy in a prosthetic joint, instability and malalignment.

EXAMINER: How would you investigate this patient?

CANDIDATE: I would start with routine blood investigations including CRP and ESR.

EXAMINER: How sensitive and specific are these?

CANDIDATE: In a recent systematic review in the American JBJS by Berbari *et al.*, the pooled sensitivities for ESR and CRP were 75% and 88% respectively while the pooled specificities were 70% and 74% respectively. The study also reported that interleukin 6 (IL-6) level assay was more sensitive and specific at 97% and 91% respectively. If the blood inflammatory markers are elevated, I would proceed with radioisotope bone scan and arrange for alignment check under image intensifier and joint aspiration in theatre.

EXAMINER: Are you aware of any guidelines regarding diagnosis of periprosthetic joint infections?

CANDIDATE: I am aware of the AAOS clinical guideline practice summary for diagnosis of periprosthetic joint infection of the knee. The working group strongly recommend:

- Testing ESR and CRP.
- Joint aspiration.
- The use of intraoperative frozen sections.
- Obtaining multiple intraoperative cultures.
- Against initiating antibiotic treatment until after cultures.
- Against the use of intraoperative Gram stain.

Nuclear imaging was weakly recommended as an option in patients in whom diagnosis of periprosthetic joint infection has not been established and who are not scheduled for re-operation.

Berbari E, Mabry T, Tsaras G *et al.* Inflammatory blood laboratory levels as markers of prosthetic joint infection: a systematic review and meta-analysis. *J Bone Joint Surg Am* 2010;**92**-A:2102–2109.

Della Valle C, Parvizi J, Bauer TW *et al.* Diagnosis of periprosthetic joint infections of the hip and knee. *J Am Acad Orthopaed Surg* 2010;**18**(12):760–770.

EXAMINER: Let's say the aspiration yields heavy growth of *Staphylococcus aureus*. How would you proceed from here?

CANDIDATE: With raised inflammatory markers and a positive bone scan and aspiration, I would offer this patient two-stage revision total knee replacement. I have opted for two-stage procedure because the investigations show severe infection caused by a virulent organism. The first stage would be extraction of the implants, debridement of joint and bone followed by application of antibiotic-loaded spacer. Antibiotic treatment depending on sensitivity is started after the first stage usually for a period of 4–6 weeks with close monitoring of CRP and ESR as well as clinical progress. The timing of the second stage depends on achieving normal CRP and ESR, healing of wounds or sinus and general well-being of the patient. Recent studies have shown that two-stage revision has better infection eradication rate and no difference in clinical outcome (knee scores, range of motion) compared with single stage (Jämsen *et al.*). Some of the disadvantages of two-stage revision are soft tissue scarring, dislocation of spacers, disuse atrophy and loss of bone density which makes the second-stage procedure difficult. I am aware that some surgeons have reported encouraging results from single-stage revision such as Buechel *et al.* who reported infection eradication rate of 90.9% over an average follow-up of 10.2 years. This compared favourably with the results of two-stage revision surgery while remaining cost-effective. However, I believe that single-stage revision should be reserved for cases where the organism and its sensitivities are known and it is of low virulence; in the very elderly patients and those with multiple medical problems.

[Debrief: The examiner has allowed the candidate to talk about the topic without interrupting.]

Jämsen E, Stogiannidis I, Malmivaara A *et al.* Outcome of prosthesis exchange for infected knee arthroplasty: the effect of treatment approach. *Acta Orthop* 2009;**80**(1):67–77.

Buechel FF, Femino FP, D'Alessio J. Primary exchange revision arthroplasty for infected total knee replacement: a long-term study. *Am J Orthop* (*Belle Mead NJ*) 2004;**33**(4):190–198; discussion 198.

Structured oral examination question 4: Unicondylar knee arthroplasty (UKA) versus high tibial osteotomy (HTO)

EXAMINER: This is a radiograph of a 42-year-old man who is a bricklayer. He complains of pain over medial aspect of knee which has failed non-surgical management. He has come to your clinic for a consultation. What can you see? (Figure 3.4.)

CANDIDATE: This is a weightbearing AP radiograph of left knee demonstrating moderate medial compartment osteoarthritis. The lateral compartment appears normal. There is a varus deformity of less than 10°. I would like to take a history and examine the patient. The examination is focused mainly on localizing the tenderness, range of motion, if the varus deformity is correctable and stability of knee.

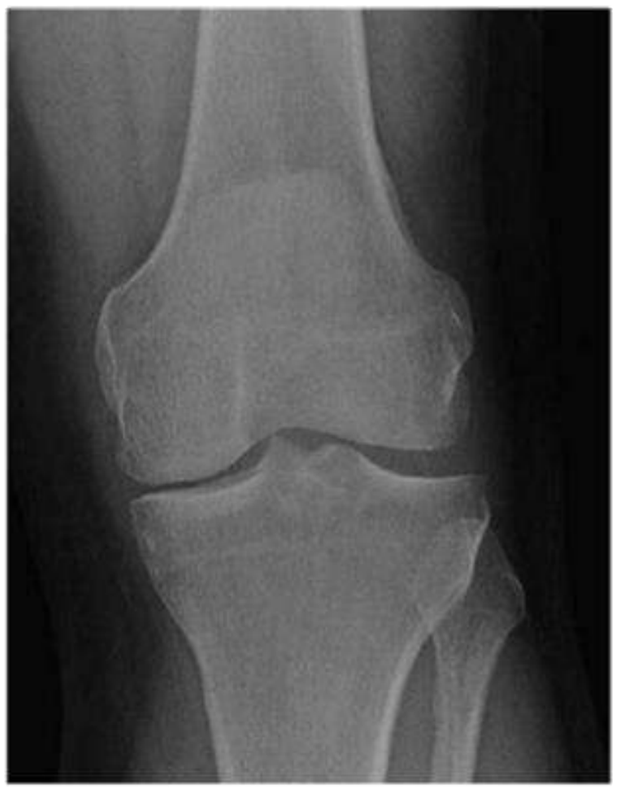


Figure 3.4 Anteroposterior (AP) radiograph left knee.

EXAMINER: The patient is fit and well, states that the pain is affecting his job and he would like to consider a surgical option. What would you offer him?

CANDIDATE: The options of surgical management once conservative measures have failed include HTO, unicondylar knee arthroplasty or total knee replacement. Since this patient has a high-demand physical job, I would offer him HTO.

EXAMINER: What are the prerequisites of HTO?

CANDIDATE: A physiological age of < 60 years, fixed varus deformity < 15° or valgus deformity < 12°, fixed flexion deformity of < 15°, > 90° flexion.

EXAMINER: Are you aware of any contraindication for HTO?

CANDIDATE: The main contraindications are inflammatory arthropathy such as rheumatoid arthritis and psoriatic arthropathy, incompetent medial collateral ligament or ACL, large varus thrust with coronal subluxation of > 1 cm, severe OA of medial compartment or lateral compartment/PFJ and more than 20° of correction. Obesity is also a contraindication because valgus knee is poorly tolerated due to medial thigh contact.

EXAMINER: The patient tells you that he has heard about partial knee replacement and is keen to consider the option. How do you proceed?

CANDIDATE: I would explain to the patient that UKA is an option; however, I would not recommend UKA for this particular patient because the highly physically demanding job could result in accelerated wear of UKA.

EXAMINER: So which patients would you offer UKA?

CANDIDATE: The indications and prerequisites for HTO and UKA are more or less the same. However women prefer the UKA because they do not tolerate the angular deformity created by HTO very well. In addition, patients who have low physical demand may benefit from UKA.

EXAMINER: Are you aware of any comparative studies of HTO versus UKA?

CANDIDATE: Yes. A recent review by Dettoni *et al.* reported that a few studies show slightly better results for UKA in terms of survivorship and functional outcome. Nevertheless, the differences are not remarkable, the study methods are not homogeneous and most of the papers report on closing wedge HTOs. They concluded that with the correct indications, both treatments produce durable and predictable outcomes in the treatment of medial unicompartmental arthrosis of the knee. There is no evidence of superior results of one treatment over the other.

Dettoni F, Bonasia DE, Castoldi F *et al.* High tibial osteotomy versus unicompartmental knee arthroplasty for medial compartment arthrosis of the knee: a review of the literature. *Iowa Orthop J* 2010;**30**:131–140.

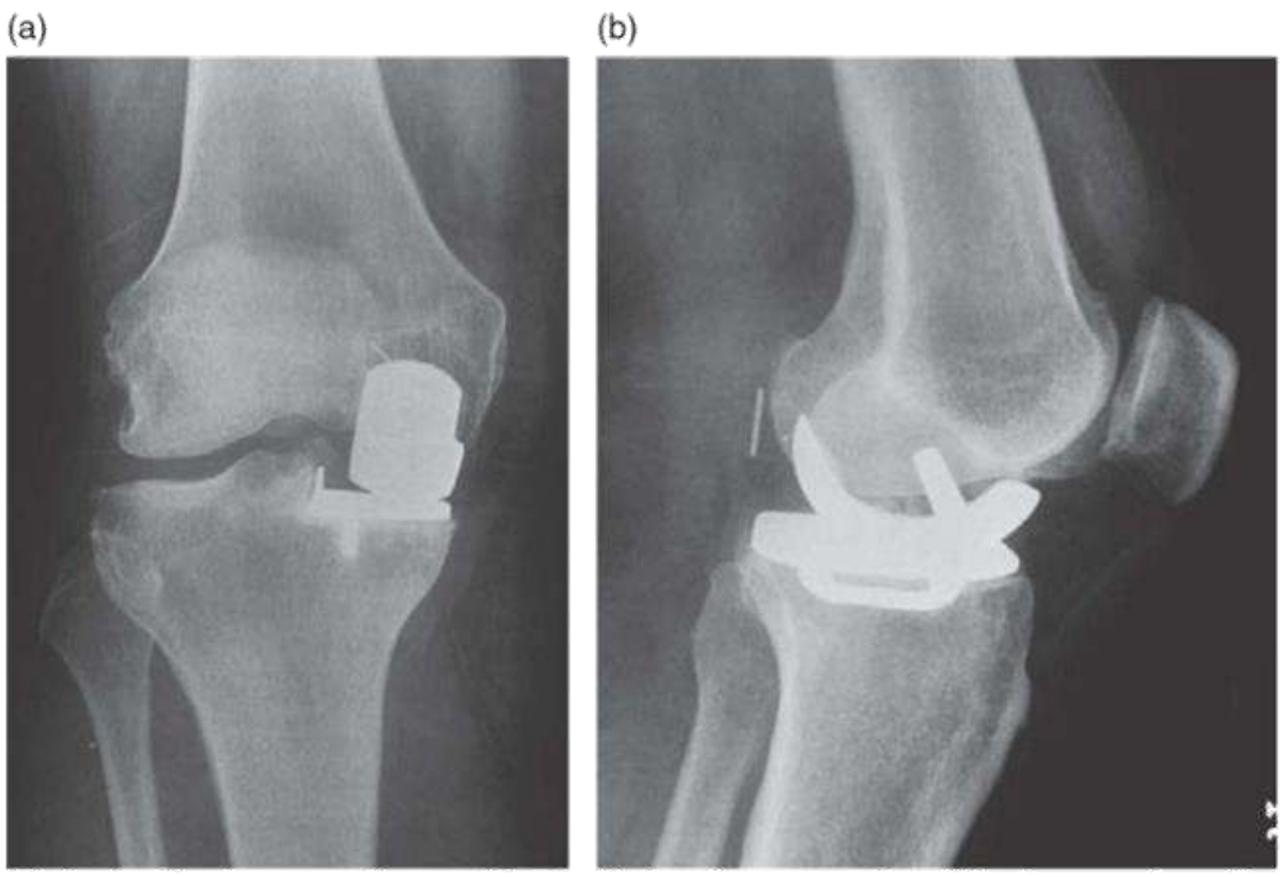
EXAMINER: Let's say this patient has decided to go ahead with HTO. What type of HTO would you perform and why?

CANDIDATE: I am conversant with closing wedge osteotomy. This was considered the gold standard in the past and may entail proximal fibular osteotomy or disruption of tibial–fibular joint. It has the risk of peroneal nerve injury, there is also loss of bone stock making it technically difficult to perform TKA. Due to these reasons, the open wedge osteotomy has become popular recently even though it has the disadvantage of having to use bone graft and late collapse with loss of correction. No conclusions can be drawn on which techniques are to be preferred when comparing between closing wedge with opening wedge as none has shown significantly better outcome over the other.

Amendola A, Bonasia DE. Results of high tibial osteotomy: review of the literature. *Int Orthop* 2010;**34**(2):155–160.

EXAMINER: You mentioned difficulty with conversion of HTO to TKA. Tell me more about this.

CANDIDATE: Before the introduction of internal fixation and early motion in HTO, cast immobilization was part of the postoperative treatment and this resulted in patella baja following a lateral closing wedge osteotomy. This complication was probably due to contracture of the patellar tendon during



Figures 3.5a and 3.5b Anteroposterior (AP) and lateral radiographs UKA.

cast immobilization. More recent studies show that closing wedge osteotomy increases patellar height, whereas opening wedge osteotomy lowers patellar height and this can have implications following TKA. Van Raaij *et al.* performed a systematic review and reported prolonged surgical time, extra-operative procedures and less postoperative knee range of motion (ROM), but no increase in revision surgeries for patients receiving TKA after prior HTO compared with patients receiving primary TKA.

van Raaij TM, Reijman M, Furlan AD, Verhaar JAN. Total knee arthroplasty after high tibial osteotomy. A systematic review. *BMC Musculoskeletal Disord* 2009;**10**:88.

http://www.wheelessonline.com/image3/i1/knee6.jpg

Structured oral examination question 5: Unicondylar knee arthroplasty (UKA) versus total knee replacement (TKR)

EXAMINER: Have a look at these radiographs. What can you see? (Figure 3.5.)

CANDIDATE: Non-weightbearing AP and lateral radiographs of 54-year-old man showing a left medial UKA in situ. The components look well fixed and aligned. There are no obvious periprosthetic fractures. The lateral compartment and PFJ look relatively normal.

EXAMINER: What else can you see?

CANDIDATE: (A bit hesitant and moves closer to the computer screen. This is followed by period of silence before the examiner prompts.)

EXAMINER: The patient tells you that he fell while coming down the stairs sustaining injury to the left knee. He complains of global pain and swelling of the left knee and inability to flex it. What's going through your mind?

CANDIDATE: There is a faint radio-opaque line behind the femoral component. I would like to compare this with previous radiographs. The history and radiographs are suggestive of dislocation of mobile bearing spacer.

EXAMINER: Good. What are the advantages of UKA?

CANDIDATE: Some of the advantages of UKA are:

- Preservation of bone stock.
- Faster recovery and return to normal function.
- Prevention of PFJ overload.
- Retention of knee kinematics and increased flexion.
- Less blood loss, infection rate and reduced risk of thromboembolism.
- Easier revision to TKA than HTO.

EXAMINER: Does UKA perform as well as TKA?

CANDIDATE: Careful patient selection for UKA is critical if consistent and reliable results are to be obtained. In the early 1980s UKA became gradually unpopular mainly because of poor results due to poor patient selection, operative technique and polyethylene wear. With improvement in patient selection, operative technique and prosthesis design, the results of UKA became comparable to TKA. Latest reports show highly satisfactory survival rate and patient satisfaction for UKA particularly in activities requiring ROM such as going down stairs and kneeling. In a recent report from the Finnish

Arthroplasty Registry, Koskinen *et al.* published a 10-year survival rate of between 53% and 81% depending on prosthetic model implanted. The UK National Joint Registry (NJR) 8th Report showed an overall 5-year revision rate of TKA and UKA of 3% and 9.4% respectively.

Koskinen E, Paavolainen P, Eskelinen A, Pulkkinen P, Remes V. Unicondylar knee replacement for primary osteoarthritis. A prospective follow-up study of 1,819 patients from the Finnish Arthroplasty Register. *Acta Ortho Scand* 2007;**78**(1):128–135.

UK National Joint Registry (NJR) 8th Report. 2011; www.njrcentre.org.uk

Structured oral examination question 6: Anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) reconstruction

EXAMINER: These images belong to a 26-year-old rugby player. He gives a history of falling awkwardly on to his left knee after a heavy tackle. What can you see? (Figure 3.6.)

CANDIDATE: These are plain radiographs and MRI of the right knee. The most obvious abnormality is cortical disruption at the site of PCL insertion with displaced avulsed fragment. The lateral radiograph shows this is a large fragment which is displaced into the joint.

EXAMINER: How would you treat this patient?

CANDIDATE: I would offer this patient reattachment of the PCL avulsion through open procedure.

EXAMINER: What approach would you use?

CANDIDATE Posterior approach.

EXAMINER: Tell me about posterior approach to the knee.

CANDIDATE: The indications include removal of popliteal cysts and neoplasms, posterior synovectomy, open reduction and internal fixation of posterior tibial plateau shear fractures, fixation of bone avulsions associated with a posterior cruciate ligament (PCL) injury, repair of posterior vascular injuries, and more recently, posterior inlay PCL reconstructions. The patient is usually positioned prone with tourniquet high up in the thigh. The lazy S-shaped incision is made starting posterolaterally along the border of biceps femoris tendon crossing the popliteal fossa and ending posteromedially at the posterior border of semitendinosus tendon. The deep fascia is incised in the midline. The small saphenous nerve is identified with accompanying sural nerve that must be preserved. The sural nerve is traced proximally where it pierces deep fascia from the tibial nerve trunk. At the apex of the fossa, the common peroneal nerve separates from tibial nerve. The tibial nerve lies posterior to the popliteal vein which in turn is superficial to popliteal artery. Popliteal vessels are displaced laterally and this usually requires ligation of middle geniculate and superior medial geniculate vessels. The medial head of gastrocnemius is identified, traced proximally and can be detached from its origin then retracted towards midline to expose the medial joint capsule. Similarly the lateral head of gastrocnemius can be detached to expose the posterolateral corner of the joint. The main structures at risk are the popliteal vessels, small saphenous vein and common peroneal nerve and tibial nerve.

EXAMINER: Have you been involved in any arthroscopic PCL reconstruction?

CANDIDATE: Yes (despite never having seen one!)

EXAMINER: What is the optimum tunnel placement?

CANDIDATE: The tunnel placement in PCL reconstruction depends on whether it is single-bundle or double-bundle reconstruction . . .

EXAMINER: Tell me about the one you have seen.

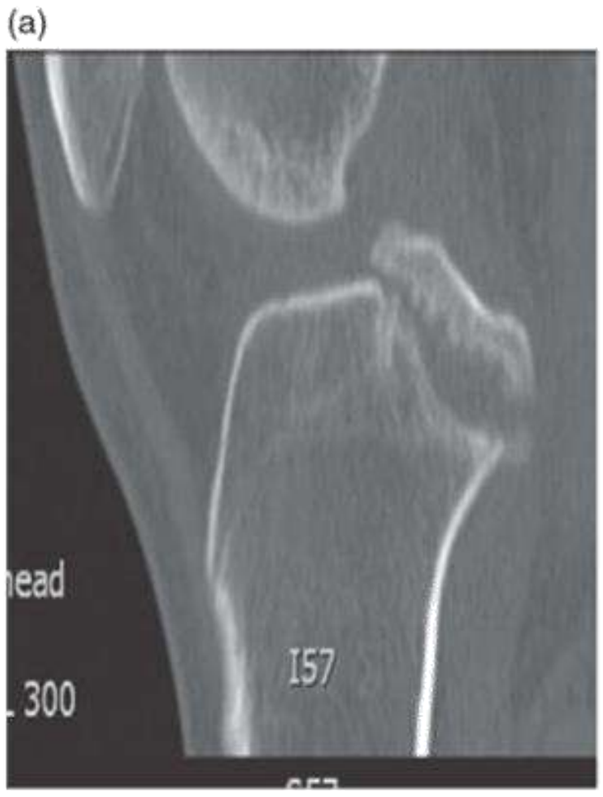
CANDIDATE: The optimum placement of PCL tunnel is controversial. The literature shows that the femoral tunnel for posterolateral bundle reconstruction should be placed at 1.30 o'clock . . .

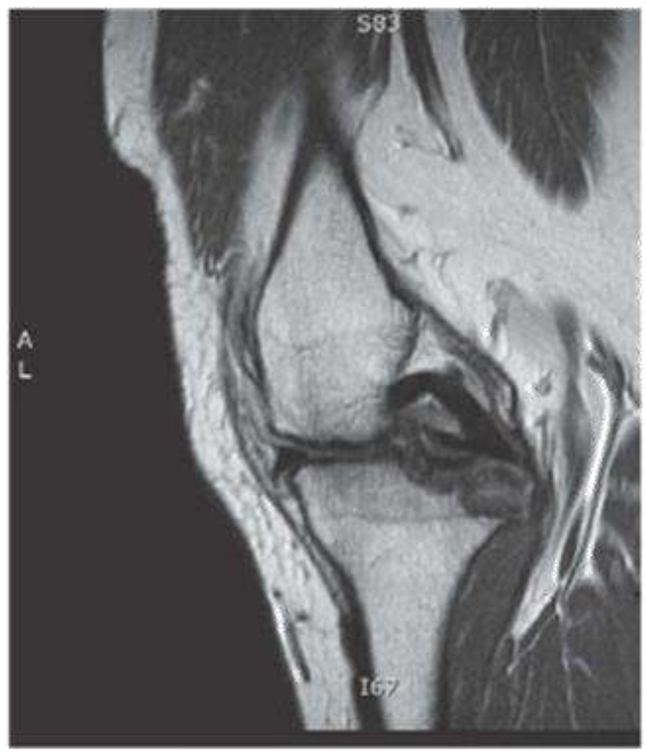
EXAMINER: Are you sure? (Realizing that the candidate is bluffing.)

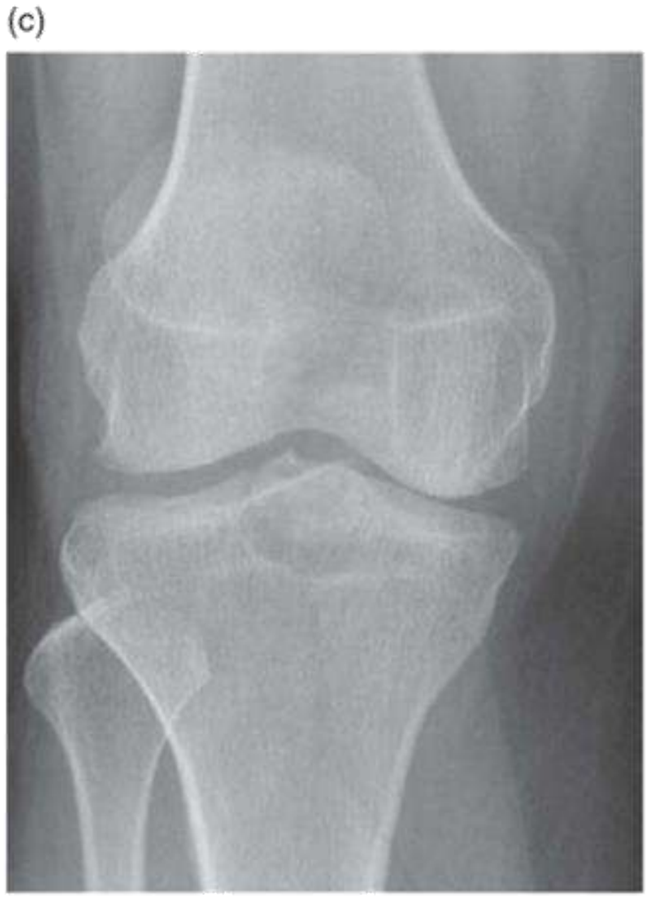
CANDIDATE: To be honest I have not seen many of these but I will check on it.

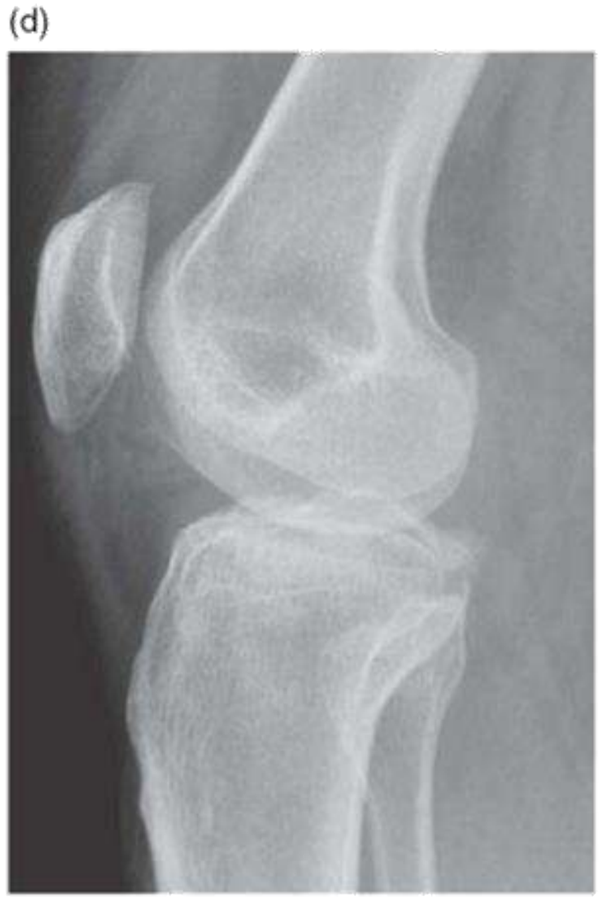
EXAMINER: Let's move on. Now tell me about the optimum tunnel placement for single bundle ACL reconstruction.

CANDIDATE: The principles of ACL reconstruction are placement of tunnel anatomically and isometrically, using biologically active grafts which are adequately tensioned to allow early rehabilitation. In single-bundle reconstruction, the aim is to place tunnel at the footprint of the posterolateral bundle of ACL. The anteromedial bundle is thought to be the most isometric but most surgeons feel that it's important to replace the posterolateral bundle. For the femoral tunnel the isometric point lies at about 10 to 10.30 o'clock for right knee and 1.30 to 2 for left knee. The most common mistake is to









Figures 3.6a, 3.6b, 3.6c and 3.6d CT, MRI and plain radiographs of left knee.

place femoral tunnel too anterior or 'resident's ridge'. This restricts flexion of the knee and may result in elongation of graft. Similarly, too posterior tunnel placement results in excessive tightening of graft when knee is extended. It's been shown that abnormally narrow intercondylar notch correlates directly with increased incidence of ACL tears. Careful assessment of notch should be done prior to graft insertion using a pin to ensure no impingement on lateral femoral condyle. The presence of impingement with correct placement of tunnels necessitates notchplasty of the anterior portion of lateral femoral condyle.

EXAMINER: Which graft would you use and why?

CANDIDATE: I would use a hamstring four-strand autograft. The two main biological autografts used in ACL reconstruction are hamstring and bone patella tendon bone (BPTB) graft. The BPTB graft has the advantage of being easy to harvest, rigid fixation and faster integration as it uses bone to bone healing. However, it has donor site morbidity which includes anterior knee pain in 30–50%, patellar tendonitis 3–5%, patellar fracture and patella baja. The hamstring graft on the other hand has less donor site morbidity, can be harvested from a small incision and can be passed relatively easily. However it has slow healing because of tendon to bone incorporation which takes 8 to 12 weeks. It can also result in hamstring

weakness and saphenous nerve injury. There are several studies comparing outcome of BPTB versus hamstring graft. Most studies show arthroscopic reconstruction with either graft results in similar functional outcome but increased morbidity in BPTB in form of early OA and increased knee laxity with radiographic femoral tunnel wide in hamstring graft.

Feller JA, Webster KE. A randomized comparison of patellar tendon and hamstring tendon anterior cruciate ligament reconstruction. *Am J Sports Med* 2003;**31**:564–573.

Howell SM, Taylor MA. Failure of reconstruction of the anterior cruciate ligament due to impingement by the intercondylar roof. *J Bone Joint Surg Am* 1993;**75**-A:1044.

Pinczewski LA, Deehan DJ, Salmon LJ, Russell VJ, Clingeleffer A. A five-year comparison of patellar tendon versus four-strand hamstring tendon autograft for arthroscopic reconstruction of the anterior cruciate ligament. *Am J Sports Med* 2002;**30**:523–536.

Debrief: With a thorough understanding of ACL reconstruction, the candidate has recovered from a bad start of this viva. Candidates should be honest and be prepared to say they have not seen some operations.

Structured oral examination question 7: Revision knee replacement

EXAMINER: Have a look at these images and tell me what you can see. (Figure 3.7.)

CANDIDATE: These are AP and lateral radiographs of failed left total knee replacement. The implants appear to be loose with widespread osteolysis and bone loss in the femur and tibia. The tibial base plate is in varus and extended. There is notching of the anterior cortex of femur. There is calcification of soft tissues including the popliteal vessels. I would like to see immediate postoperative and most recent radiographs for comparison. The radiographs are suggestive of infection until proven otherwise.

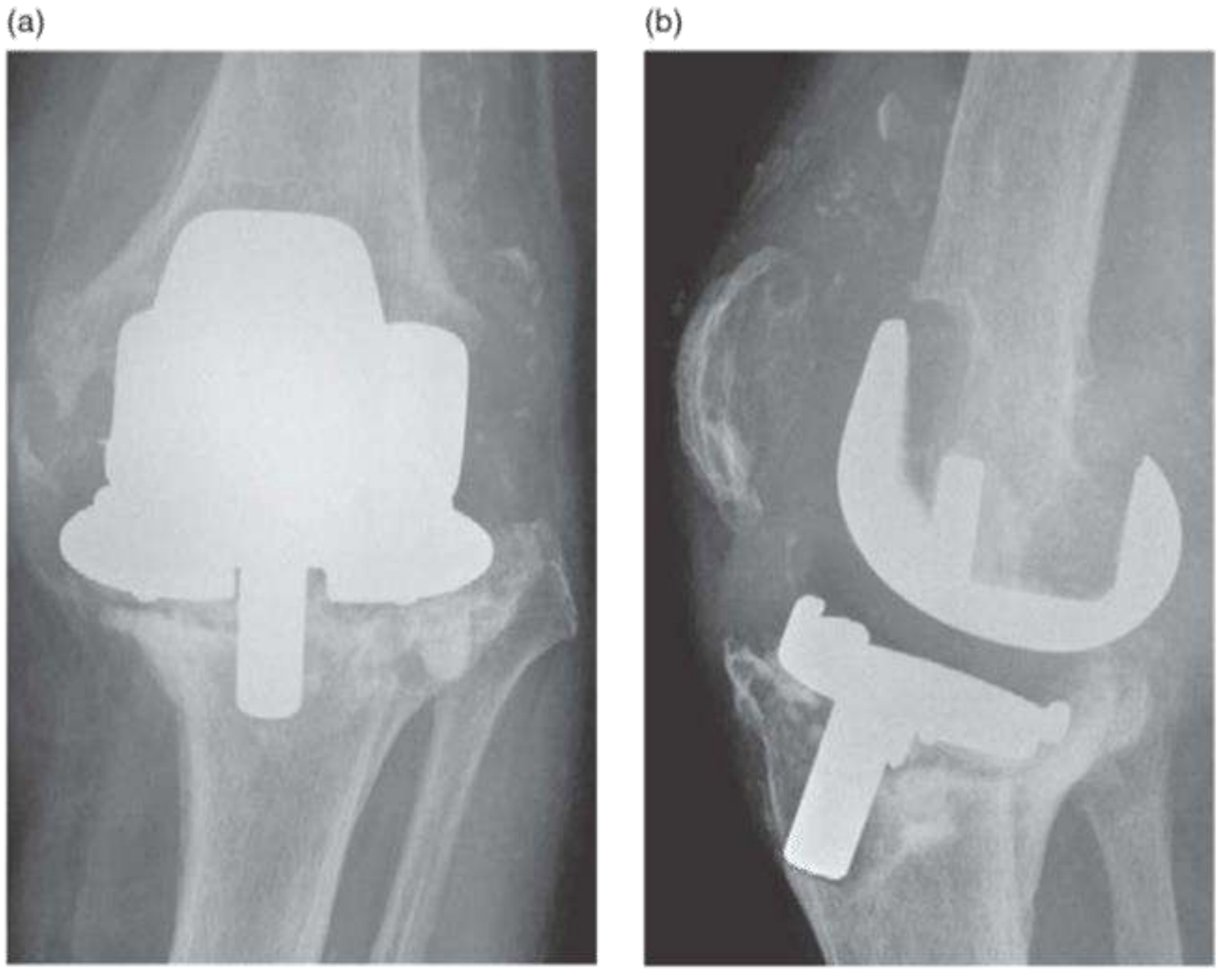
EXAMINER: Good. You investigate this patient and come up with a diagnosis of aseptic loosening. The patient is keen to consider single-stage revision surgery. What are your concerns with regards to these radiographs?

CANDIDATE: I am concerned about several factors, namely:

- The state of the collateral ligaments (stability).
- Soft tissues and vascular status of the limb.
- The extensive bone loss.

The collateral ligaments are likely to be dysfunctional and especially the MCL therefore a constrained knee replacement may be required. The soft tissues appear contracted and calcified which may lead to wound complications. The bone loss will require bone graft, augmented or stemmed implants.

EXAMINER: Are you aware of any classification system for bone loss around knee arthroplasty?



Figures 3.7a and 3.7b Anteroposterior (AP) and lateral radiographs of left TKA.

CANDIDATE: The most commonly used classification system is that of the Anderson Orthopaedic Research Institute (AORI) which classifies the femur (F) and tibia (T) separately as follows:

Type 1 – Intact metaphyseal bone with minor defects which will not compromise the stability of a revision component.

Type 2 – Damaged metaphyseal bone. Loss of cancellous bone in the metaphyseal segment which will need to be filled with cement, augments or a bone graft at revision in order to restore the joint line. Defects can occur in one femoral condyle or tibial plateau (2A) or in both condyles or plateaux (2B).

Type 3 – Deficient metaphyseal bone. Bone loss which comprises a major portion of either condyle or plateau. These defects are occasionally associated with detachment of the collateral or patellar ligaments and usually require long-stemmed revision implants with bone grafts or a custom-made hinged prosthesis.

Engh G. Bone defect classification. In GA Engh, CH Rorabeck (Eds), *Revision Total Knee Arthroplasty*. Baltimore, MD: Lippincott Williams and Wilkins, 1997, pp. 63–120.

EXAMINER: You mentioned that a constrained implant may be required. What are the levels of constraints?

CANDIDATE: The constraint ladder within knee implant design includes:

PCL retaining (cruciate retaining or CR). Rotating platform more constrained due to conformity.

↓

PCL substituting (posterior stabilized or PS).

↓

Unlinked constrained condylar implant (varus–valgus constrained or VVC) provides anteroposterior and varus–valgus stability (substitute for deficient collaterals), e.g. constrained condylar knee (LCCK, NexGen), TC3.

↓

Linked, constrained condylar implant (rotating-hinge knee or RHK). Rarely indicated. Used for global instability (total collateral disruption/recurvatum) and severe distal femoral bone loss, osteolysis/fracture.

EXAMINER: What are the indications of PCL substituting posterior stabilized (PS) implants?

CANDIDATE: Some of the indications of PCL sacrificing implants are:

- Previous patellectomy.
- Rheumatoid arthritis.
- Post-traumatic osteoarthritis with stiffness.
- Previous HTO and large deformity.
- Over-released PCL.

EXAMINER: What are the advantages of PS over CR (cruciate retaining) design?

CANDIDATE: The advantages are:

- Conforming surfaces allowing roll-back.
- No component slide.
- Provides a degree of VVC.
- The cam–post mechanism improves anterior–posterior stability.
- Facilitates any deformity correction.
- Uses more congruent joint surfaces than CR, which reduces wear.
- Better range of motion.
- Technically easier than CR and reproducible.
- Higher degree of flexion.

EXAMINER: Are you aware of any current literature regarding performance of PS and CR implants?

CANDIDATE: There are limited studies in the literature comparing the outcomes of the two designs. Most of the studies are characterized by a small number of patients, different outcome measures, poor randomization and comparing designs of different manufacturers. Range of motion appears to be the only common outcome parameter. A meta-analysis by Jacobs *et al.* showed a difference in range of motion and reproduction angle favouring posterior stabilized designs over PCL retention designs 1 year postoperatively. However, it is uncertain whether this observation is of clinical relevance. It seems that in patients with functional PCL the decision as to which design is chosen depends largely on the favour and training of the surgeon.

Jacobs WC, Clement DJ, Wymenga AB. Retention versus removal of the posterior cruciate ligament in total knee replacement. *Act Orth* 2005;**76**(6):757–768.

EXAMINER: (Going back to the radiographs.) What are the principles of management of bone loss in revision knee replacement in this patient?

CANDIDATE: The options of management of the extensive bone loss are:

1. The use of cement, either alone or combined with screws and mesh.
2. The use of bone grafting with structural or morsellized graft.
3. The use of modular augmentation of the components with wedges or blocks of metal. Recent studies show modular

porous-coated press-fit metaphyseal sleeves may be used to fill AORI type 2 and 3 defects and provide for stable ingrowth.

4. The utilization of custom-made, tumour or hinge implants.

The method of reconstruction and the materials for revision surgery are largely dependent on the potential for future further revision and the life expectancy, functional demand and comorbidities of the patient. In this patient who is reasonably young restoration of bone stock is preferable, because of likelihood of further revision surgery.

Structured oral examination question 8: Patellar instability

EXAMINER: A 17-year-old lady is referred to your Patella clinic by the GP due to recurrent bilateral patellar dislocation. How would you assess this patient?

CANDIDATE: I would start by taking a detailed history followed by clinical examination. In the history, I would enquire about age at first dislocation, frequency of dislocations, traumatic or atraumatic, any associated syndromes such as bone or connective tissue dysplasia and generalized joint laxity. I would also enquire about any mechanical symptoms, the presence and localization of pain.

EXAMINER: What are risk factors for patellar instability?

CANDIDATE: The risk factors for patellar instability are:

1. Bony factors (static)

   Trochlear dysplasia.
   Hypoplastic femoral condyle.
   Patellar shape.
   Patella alta.

2. Malalignment

   Patellar malalignment is an abnormal rotational or translational deviation of the patella along any axis.
   External tibial torsion/foot pronation.
   Increased femoral anteversion and increased genu valgum.
   Increased Q angle or abnormal tibial tuberosity–trochlear groove (TT–TG) distance.

3. Soft tissue (dynamic)

   Ligamentous laxity (medial patellofemoral ligament rupture/ insufficiency).

4. Abnormal gait

   Walking with valgus thrust.

5. Genetic factors such as connective tissue disorder syndromes.

EXAMINER: Tell me about the most important static stabilizer of the patella.

CANDIDATE: The primary static restraint to the lateral patellar displacement is medial patellofemoral ligament. It provides 50% of the total medial restraining force. MPFL sectioning can lead to substantial changes in patellar tracking. It originates from the area between the medial epicondyle and adductor tubercle and inserts onto the proximal two-thirds of the patella. The average length of the ligament is 5.5 cm. During acute patellar dislocation there is a 90–95% incidence of damage to the MPFL. Femoral attachment is commonly affected. In the past 10 years, MPFL reconstruction has become a popular procedure for treatment of recurrent patellar dislocation.

EXAMINER: How would you investigate this patient?

CANDIDATE: I would perform the following investigations:

1. A lateral radiograph is the most helpful view for assessment of patellar tilt, height and trochlear depth.
2. Axial radiographs (Merchant's view) to assess patellar tilt angle (normal $< 10^{\circ}$), congruence, sulcus angle (normal 138°) and trochlear dysplasia.
3. MRI for articular lesion and state of MPFL.
4. CT scan to assess:
   - Femoral anteversion (normal 5–15)
   - Tibial torsion.
   - TT–TG distance more than 15–20 mm is significant.
   - Patellar tilt.
   - Trochlear depth.

EXAMINER: What are the principles and methods of distal realignment procedures?

CANDIDATE: The three main groups of realignment procedure as determined by direction of tibial tubercle (TT) transfer are:

- Medial transfer to treat malalignment.
- Anteromedial transfer for malalignment and PFJ chondrosis.
- Anterior when there is distal PFJ chondrosis.

The methods of realignment are:

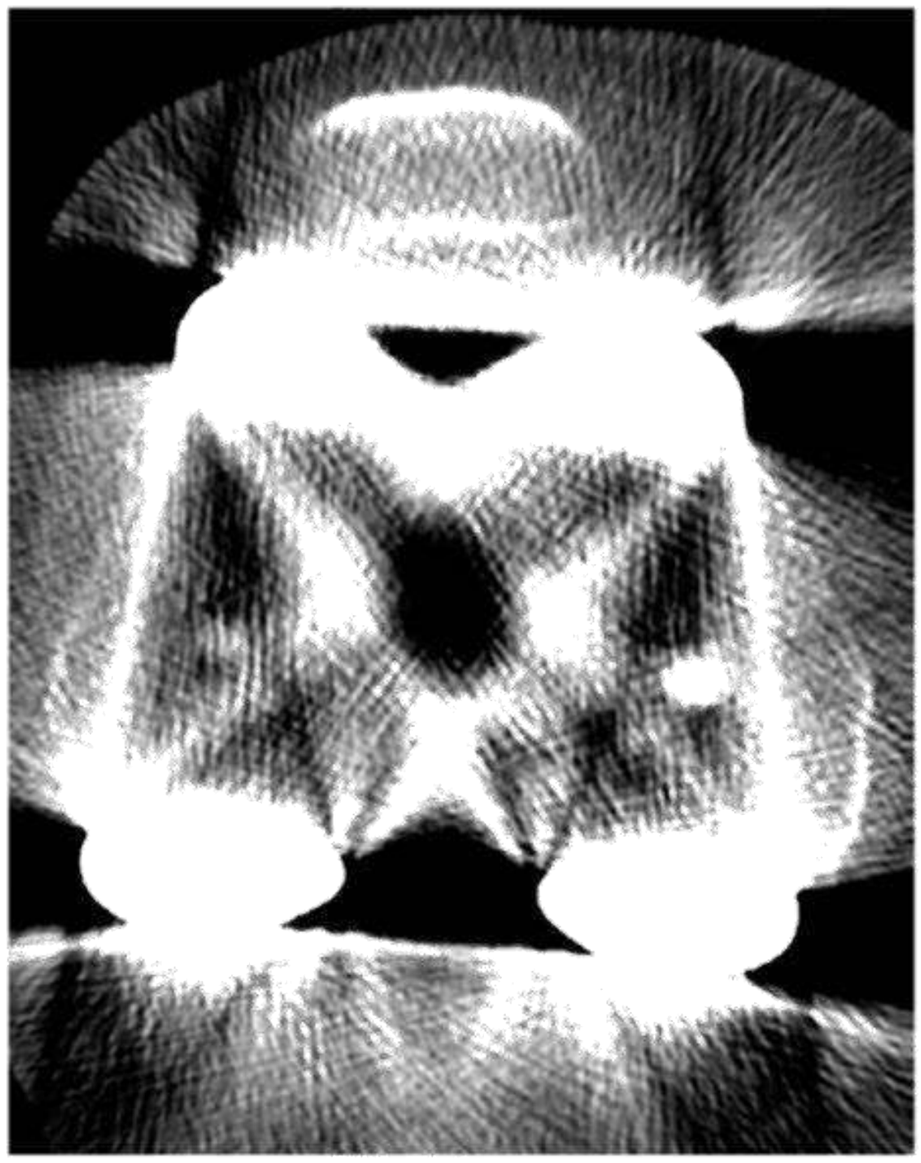
Elmslie–Trillat: Medialization without posteriorization of the tibial tubercle.

Fulkerson: Medialization with anteriorization of the tibial tubercle in the arthritic patella.The obliquity of the cut depends on the degree of malalignment and arthrosis.
A steep cut up to a 60° angle maximizes anteriorization and is useful in patients who have more arthrosis than malalignment.

Hauser: Transfer of the tibial tubercle to a medial, distal and posterior position. This has been abandoned. It increases the PFJ reaction force and causes patellofemoral degenerative joint disease.

Goldthwait 1899–Roux 1888: Medial transposition of the medial half of the patellar tendon, lateral release/medial reefing. Now the lateral half is placed under the medial half and medially (historical procedure) .

Maquet: Anterior transportation of tibial tubercle, which decreases patellofemoral contact forces. Not performed nowadays (historical) as it has a high incidence of skin necrosis, compartment syndrome and no effect on the Q angle.



Figures 3.8 CT axial view of TKR.

Structured oral examination question 9: Malalignment of total knee replacement (TKR) components

EXAMINER: Have a look at this image. What can you see? (Figure 3.8.)

CANDIDATE: This is a CT scan of the distal femur showing an axial view of the femoral component of TKR. There is a lot of metal artifact.

EXAMINER: Why do you think a CT scan was done for this patient?

CANDIDATE: CT scan can be performed following TKR to check for loosening or malalignment of the components.

EXAMINER: What do you think of the alignment of this femoral component?

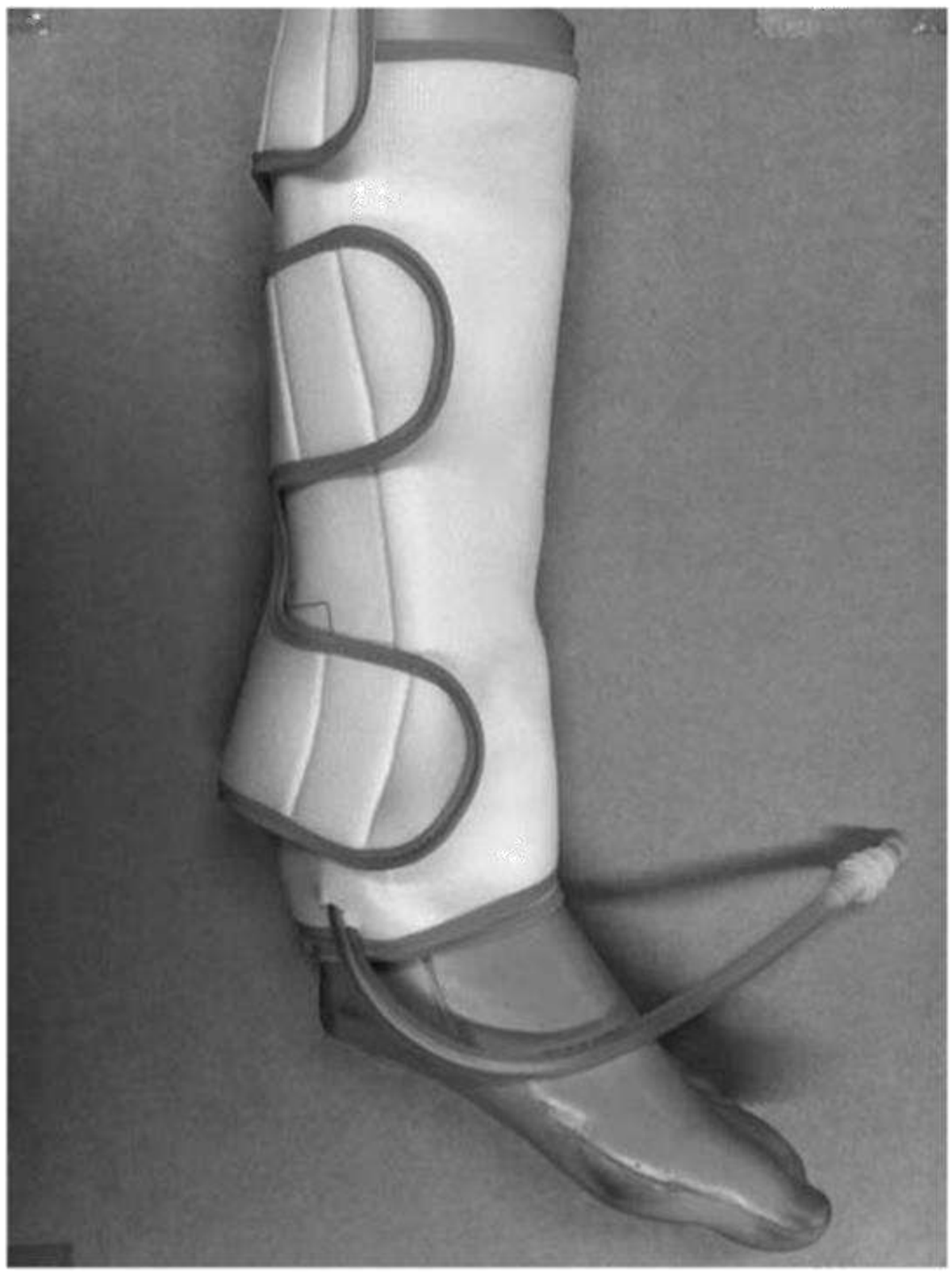
CANDIDATE: The angle formed by the surgical transepicondylar axis and the posterior condylar axis show the femoral component is internally rotated.

EXAMINER: Good. What problems can arise from internal rotation of the femoral component?

CANDIDATE: Rotational alignment of the tibial and femoral component plays an important role in TKR. Once correct frontal alignment and proper soft tissue balancing have been achieved, the rotational placement of the components represents the 'third dimension' in knee TKR. Femoral component malposition has been implicated in patellofemoral maltracking following TKR, which is associated with anterior knee pain, subluxation, fracture, wear, and aseptic loosening. It has been suggested that rotating-platform mobile bearings compensate for malrotation between the tibial and femoral components and may, therefore, reduce any associated patellofemoral maltracking. Internal rotation of femoral component by resection of excessive amounts of posterior lateral femoral condyle or insufficient resection of the posterior medial femoral condyle moves the anterior femoral patellar groove portion of the femoral component medially, making it more difficult for a relatively laterally placed patella to be captured by the patellofemoral groove. In addition, internal rotation of the femoral component results in tight flexion gap on the medial side of the knee.

Nicoll D, Rowley DI. Internal rotational error of the tibial component is a major cause of pain after total knee replacement. *J Bone Joint Surg Br* 2010;**92**-B:1238–1244.

Viva 27



Do you use this device in your clinical practice?

Describe Virchow's triad and the risk factors for formation of a deep vein thrombosis (DVT).

What risk levels do you quote to patients undergoing total hip replacement (THR) and total knee replacement (TKR)?

What is your DVT prophylaxis policy for THR in a 70-year-old man with no significant additional risk factors?

Do you use this device in your clinical practice?

Yes, this is a mechanical calf pump that we use intra-operatively to prevent venous thrombosis.

Describe Virchow's triad and the risk factors for formation of a deep vein thrombosis (DVT).

Virchow's triad includes:

1. Hypercoagulable state
2. Stasis of vascular flow
3. Damage to the vascular endothelium

What risk levels do you quote to patients undergoing total hip replacement (THR) and total knee replacement (TKR)?

Forty to 60 per cent of THR patients who do not receive prophylaxis will get a DVT (dependent on imaging method). With chemical and mechanical prophylaxis asymptomatic DVT occurs in 10% of THR and 20% of TKR patients. Symptomatic DVT occurs in 1.3% of TKR patients and 2.81% of THR patients.

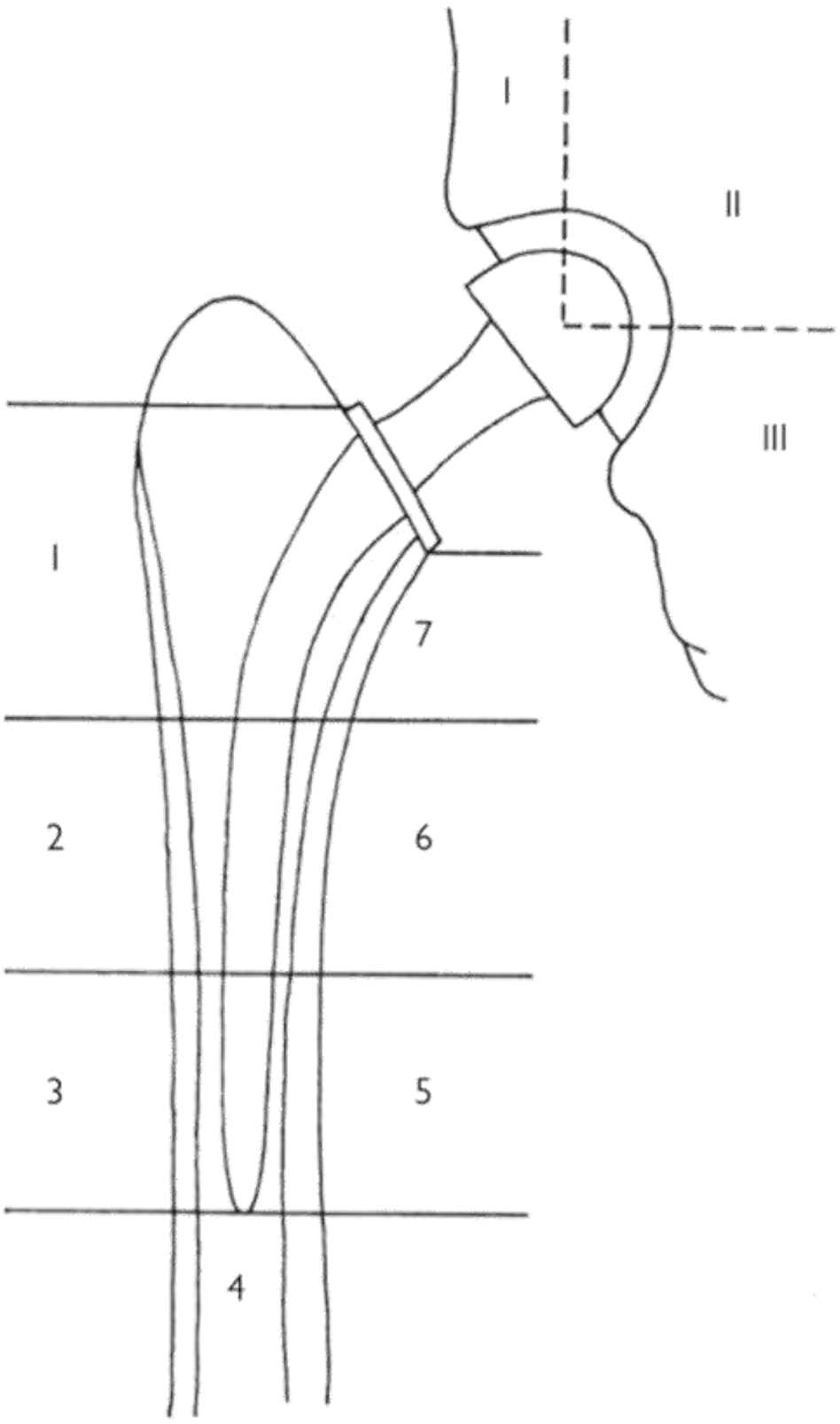
What is your DVT prophylaxis policy for THR in a 70-year-old man with no significant additional risk factors?

The two main strategies for prevention are:

1. Non-pharmacological interventions. These include anti-DVT stockings and foot or calf pumps
2. Pharmacological interventions. These include one or more of the following:
   - Low-molecular-weight heparin (LMWH): heparin and LMWH are equivalent in preventing DVT, although LMWH has greater bioavailability, longer duration of anticoagulant effect in fixed doses, and little requirement for laboratory monitoring, and is thus more cost-effective
   - Fondaparinux sodium (Arixtra)—a synthetic pentasaccharide. When used at 2.5 mg subcutaneously (SC) four times a day post-operatively, it significantly improves the risk-to-benefit ratio for the prevention of post-operative venous thromboembolism
   - Warfarin—an effective but cumbersome DVT prophylaxis regimen is achieved with either a fixed or an adjusted dose
   - Aspirin—however, there is not much evidence of its efficacy

You should be able to quote your local policy on what they use in this situation.

Viva 33



Reproduced from C. Bulstrode et al., Oxford Textbook of Trauma and Orthopaedics second edition, 2011, figure 7.10.3, p. 586, with permission from Oxford University Press..

What does this diagram represent?

What is the pathological process behind aseptic loosening?

What does this diagram represent?

The diagram shows the potential areas of lucency around the femoral and acetabular components of a total hip replacement according to Gruen (femur) and DeLee and Charnley (cup).

What is the pathological process behind aseptic loosening?

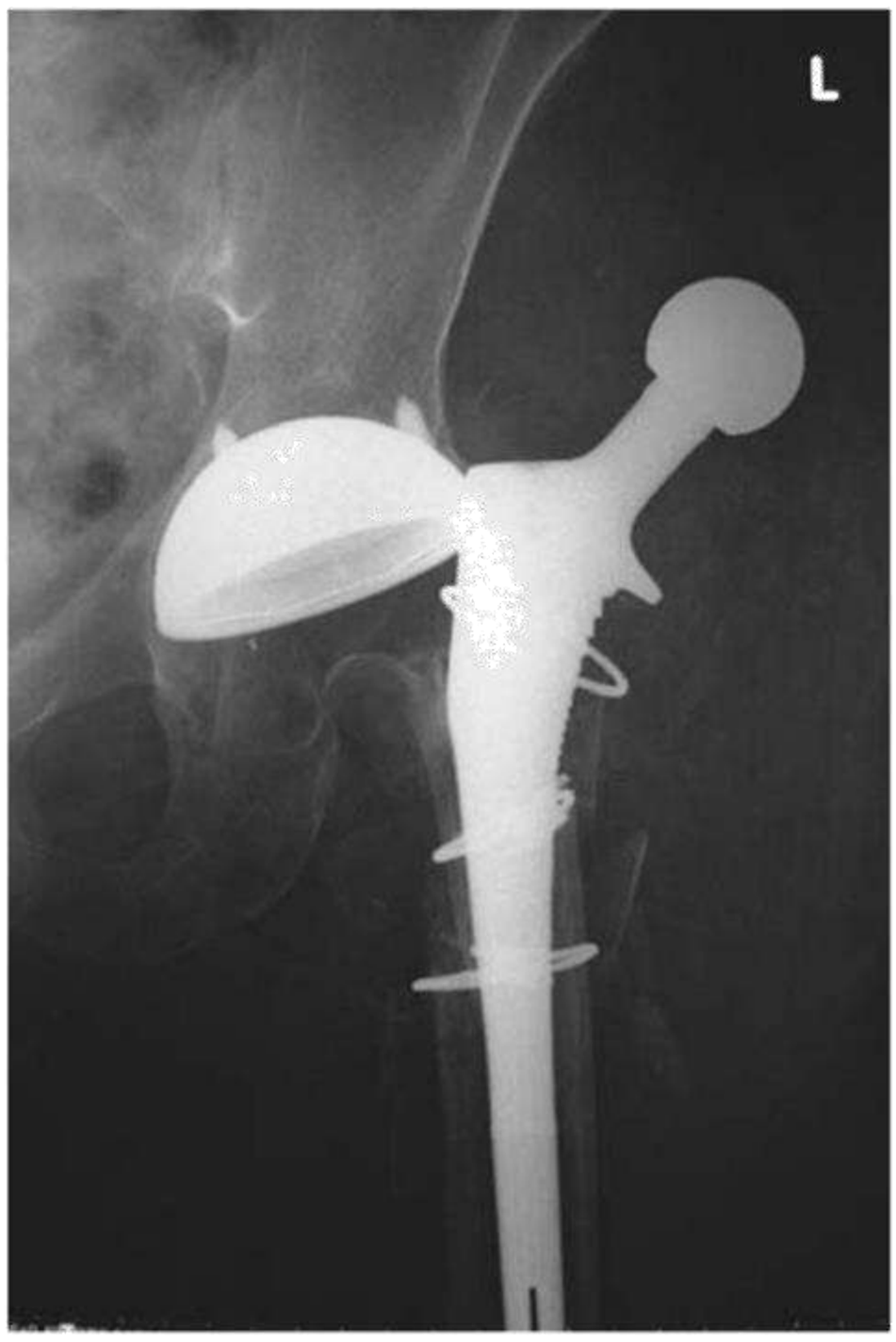
Osteolysis in total joint replacement is thought to occur as a result of resorption of bone by osteoclasts at the bone–cement interface and is associated with aseptic implant loosening. Polyethylene wear debris produced at the bearing surfaces combined with cement debris formed from movement at the interfaces is thought to induce osteolysis. *In vitro* studies suggest that the activated macrophage is a key intermediary in this process.

Peri-prosthetic bone resorption involves a series of complicated interactions between macrophages and osteoclasts. Osteolysis occurs due to both the direct resorption of bone, as a consequence of osteoclast stimulation and, to a lesser extent, the secretion of enzymes from other cells (such as metalloproteinases from fibroblasts). Macrophages are thought to be pivotal in the osteolysis process.

Cell culture studies have demonstrated that particulate wear debris from prosthetic materials are *phagocytosed* by macrophages, which subsequently respond in one of two ways:

- Firstly they secrete numerous cellular mediators some of which [tumour necrosis factor-alpha (TNF-α), interleukin (IL)-6, IL-1, and prostaglandin 2 (PGE2)] are able to induce cell proliferation and bone resorption in osteoclasts
- Secondly, *in vitro* studies have demonstrated that activated macrophages are able to differentiate into osteoclasts via two distinct pathways (fibroblast RANKL activated and TNF-α activated)

Viva 69



Reproduced from C. Bulstrode et al., Oxford Textbook of Trauma and Orthopaedics second edition, 2011, figure 7.10.4, p. 586, with permission from Oxford University Press.

What dislocation rate do you quote when you consent a patient for a total hip replacement?

What causes a hip to dislocate?

What measures can you take to prevent re-dislocation?

What dislocation rate do you quote when consenting a patient for a total hip replacement?

Dislocation following hip arthroplasty is one of the most common complications. Large studies have shown the incidence of dislocation following primary hip arthroplasty to be 3–5% over the life of the implant. The dislocation rate more than triples after revision hip surgery. The majority of dislocations occur in the first month (approximately 1%) and first year (approximately 2%). Over 50% of hips re-dislocate after initial closed reduction. Dislocation produces significant cost implications—both in terms of patient morbidity and the financial costs of treatment. It has been estimated that the cost of re-operation for a primary dislocation is 150% that of the original surgery.

What causes a hip to dislocate?

Causes of dislocation are multifactorial and can broadly be divided into surgical factors, patient factors, and implant design factors.

Surgical factors

- Component mal-position (most common)
- Soft tissue imbalance or failure of reattachment
- Soft tissue impingement (osteophytes/capsule)
- Retained debris (cement) in acetabular component

Patient factors

- Previous hip surgery or arthroplasty
- Female gender (relative risk 2.1)
- Acute fracture of proximal femur (relative risk 1.8)
- Inflammatory arthropathy
- Generalized soft tissue laxity
- Patient non-compliance (dementia, learning difficulties, drug/alcohol addiction)

Implant design factors

- Small head/neck ratios—leading to greater impingement risk
- Small head size (relative risk 1.7 with size 22 mm heads compared with 32 mm)
- Loosening of components leading to rotation and mal-alignment
- Wear of acetabular component leading to head subluxation

What measures can you take to prevent re-dislocation?

Prevention of re-dislocation can be attempted using conservative or operative methods. Assessment of joint stability should be made at the time of reduction. If the hip dislocates in the patient's normal functional range then it is likely that surgical intervention will be required.

Conservative methods

- Patient education, carer advice
- Physiotherapy and occupational therapy input
- Bracing of joint in an attempt to 'remind the patient' and prevent a position of instability

Surgical methods

Soft tissue laxity correction:

- Reattachment of avulsed soft tissues or trochanter
- Increasing neck offset using modular components

- Increasing acetabular lateral offset (lateralized liner)
- Trochanteric advancement

Increasing range of motion:

- Increase head–neck ratio (larger femoral head)
- Excision of osteophytes or soft tissues
- Increase excursion distance to dislocation (larger femoral head)
- Revision of mal-aligned components

Increase constraint:

- Augmentation of acetabular liners
- Constrained or captured liners

KNEE VIVAS

Mr Paul Allen
Consultant Knee surgeon
Princess Alexandra Hospital

1. Shown clinical picture of infected Total Knee Replacement
How will you proceed

HISTORY
1. Wound Infection
2. Post op drainage/antibiotics
3. Co morbidities- Diabetes/smoking/obese/steroids

EXAMINATION
1. Swelling/warm/pain/reduced range of movement/Sinus

Investigations

1. Bloods –White cell count /CRP/ESR
2. Scans –White cell scan
3. Aspirate (after stopping antibiotic for two weeks)
- sent for gram stain and culture
-if first aspirate negative and clinical features continue – send two more aspirates
-Arthroscopic synovial tissue biopsy

Classification

1. Positive intra-operative culture in revision
2. Early –less than four weeks–superficial/deep
3. Acute haematogenous
4. Late chronic-more than four weeks

Treatment protocol

1. Early superficial

-no arthrotomy
-debride/irrigate
-Close wound
-Antibiotics for six weeks

2. Early deep
-do arthrotomy/synovectomy
-change polyethylene
-debride/irrigate

-Close/Antibiotic beads medial and lat gutter
-Antibiotics for six weeks

3. Acute haematogenous –less than four weeks – same as early deep

4. Chronic- late more than four weeks

Options are
-Two stage revision (gold standard)
-One stage revision

Two stage revision

First stage
-Remove all components and thorough debridement
-Antibiotic cement and spacer
-Antibiotics for six weeks or more
- Repeat blood markers/CRP
-Wait two weeks after CRP return to normal/clinically normal
-Second stage revision /re implant after six weeks or more

Single stage revision option if organism known before hand/patient cannot withstand two stage

2. Shown MRI of knee with osteochondral defect. How will you manage

Definition – Avasclar necrosis occurring at osteochondral junction in second decade males most commonly around knee (also ankle/elbow)

Most common site – lateral aspect of medial femoral condyle

Etiology

1. Repetetive micro trauma
2. Genetic
3. Altered micro vascularity

Clinical features

1. Pain/ crepitus/loose body/Quadriceps wasting/effusion
2. Wilson test –flex knee 90 degree and internally rotate- extend- pain at 30 degrees is Wilsons test positive

Investigations

1. Radiograph – tunnel view
2. MRI

Classification

Guhl arthroscopic

1. Softening of cartilage
2. Undisplaced
3. Displaced but attached
4. Loose body

Treatment

Depend on

1. Age- juvenile heal better
2. Size
3. Area- weight bearing
4. Guhl stage

Non operative

1. Juvenile (less than 12 years) OCD, painless or painful with non displaced lesion-Reduced weight bearing plus quadriceps strengthen for 3-6 months/follow up with MRI to assess healing

Surgical

Surgical

Indications

1. Failed non operative in skeletally immature individuals or adults
2. Unstable/loose body
3. Avascular segment
4. Weight bearing area with large lesion

Goals

1. Pain relief/prevent osteoarthritis/better function/promote healing

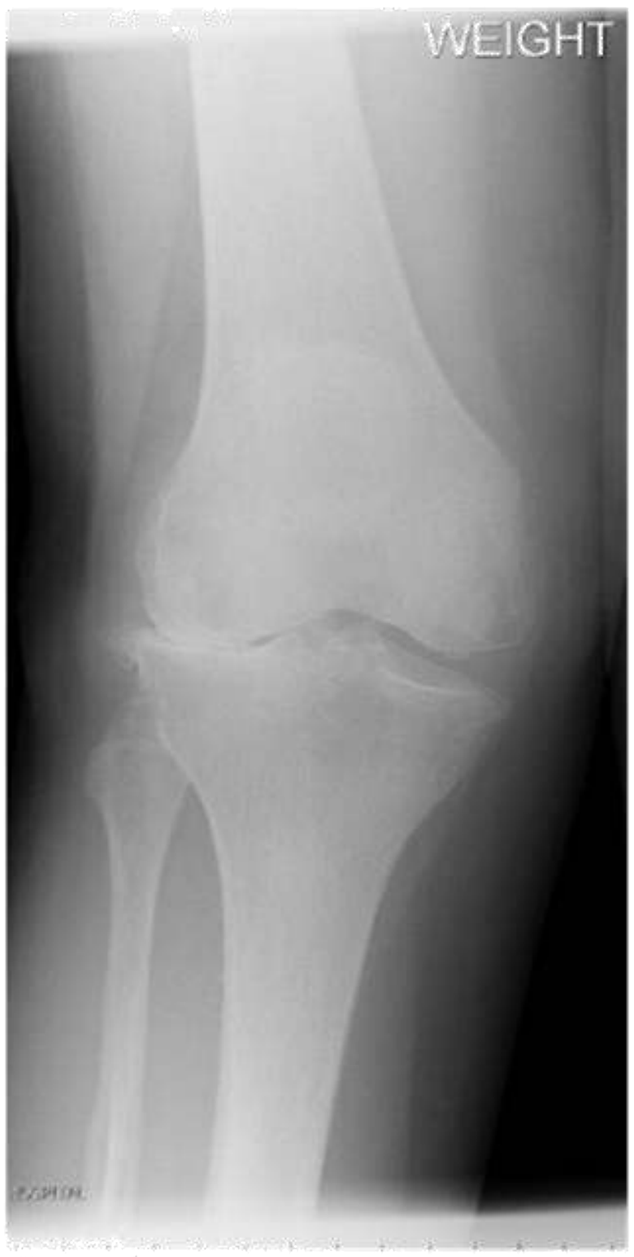
Treatment options

1. Arthroscopic drilling
2. Curettage+ drill+Bone graft if big defect
3. Unstable segment –screw fixation after curettage and drilling
4. Osteo chondral allografts /autografts

- Mosaic plasty
(recommend by NICE with special consent/audit/research.IPG162-march 2006)
-Autologous chondrocyte implantation(ACI)
(not recommended by NICE .TA89-issued may 2005-for review in 2013)

4. Valgus knee

Radiograph of valgus knee with osteoarthritis. How will you manage. What will you do different from a routine knee replacement?



Examine

1. Correctable deformity?
2. Fixed flexion deformity?
3. Valgus or varus malalignment
4. Common peroneal nerve neurology
5. Patella tracking

Evaluation

1. Radiographs
-Weight bearing AP-lateral in 30 degree/sunrise, skyline views/Pelvis
-Ascertain the presence of osseous defects, and quantify their extent
-Assess amount of bone resection-template is a must

Assess severity of valgus

1. Mild –less than 10 degree
2. Moderate -10-20 degree - soft tissue stretch
3. Severe –more than 30 degree with ligament imbalance

Problems with valgus knee

1. Femoral hypoplasia

1. Femoral hypoplasia
2. Posterior femoral condyle erosion/lateral side bone defects
3. Unusual femoral neck shaft angle
4. External rotation deformity of distal femur
5. Osseous defects
6. Ligament imbalance-medial laxity
7. Patellar maltracking
8. Fixed flexion deformity
9. Common peroneal nerve involvement

Goals of TKR

Restore alignment
Restore joint line
Restore range of movement (ROM) and patellofemoral tracking
Ligament balancing

TKR method (Ranawat paper 2005)

Implant –PCL substituting/posterior stabilised

Why?

1. More stable- cam stabilised
2. Allow greater lateralisation of femoral and tibial components- better patella-femoral tracking/less soft tissue release laterally

Technique

Tourniquet-medial parapatellar approach
-First resect tibia- then femur
-Sequential release of lateral structures (more on the femoral side)
This is called inside out technique

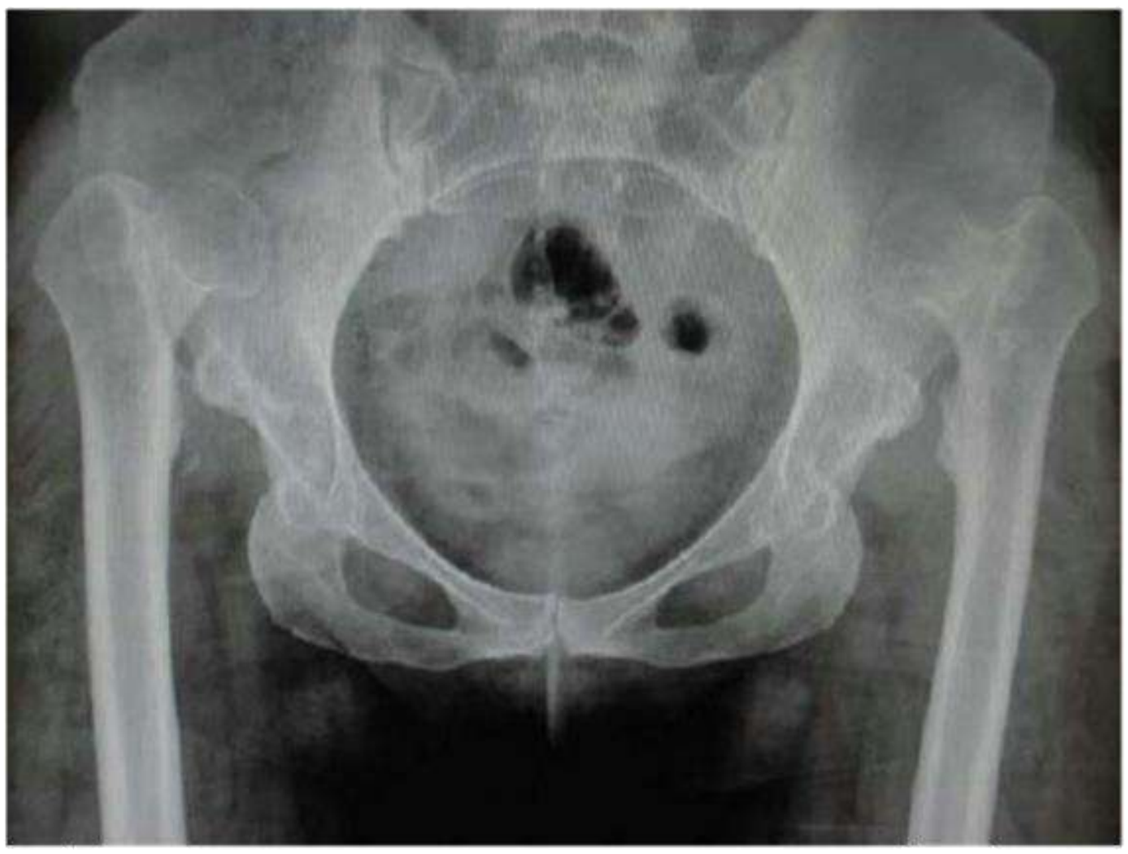
Progressive release of

-osteophytes
-capsule released with cautery (PCL already sacrificed)
-Iliotibial band (Pie crusting with multiple stab incisions)
-Popliteal tendon tenotomy if tight in flexion
-Lateral collateral ligament
-Lateral head of gastrocnemius
2. Balance extension gap before flexion gap
3. Cut distal femur in 3 degree (not 5-7) to avoid overcorrection
4. Resect less bone from femur/tibia for better soft tissue balancing
5. Use bone cuts to balance flexion gap

Hip vivas

Hip vivas

1. Radiograph of dysplastic hip. Describe findings. Investigations and management. Surgical options?



Describe
Crowe classification/Hartofilakidis classification

1. Less than 50 percent subluxation (Dysplasia)
2. 50-75 (Low Dislocation)
3. 75-100 (High Dislocation)
4. More than 100

History

1. Pain
2. Instability

Examination

1. Stiffness? – need 90 degree flexion and 15 degrees rotations for osteotomy

2. Limb Length discrepency – may need lengthening/shortening

3. Stability

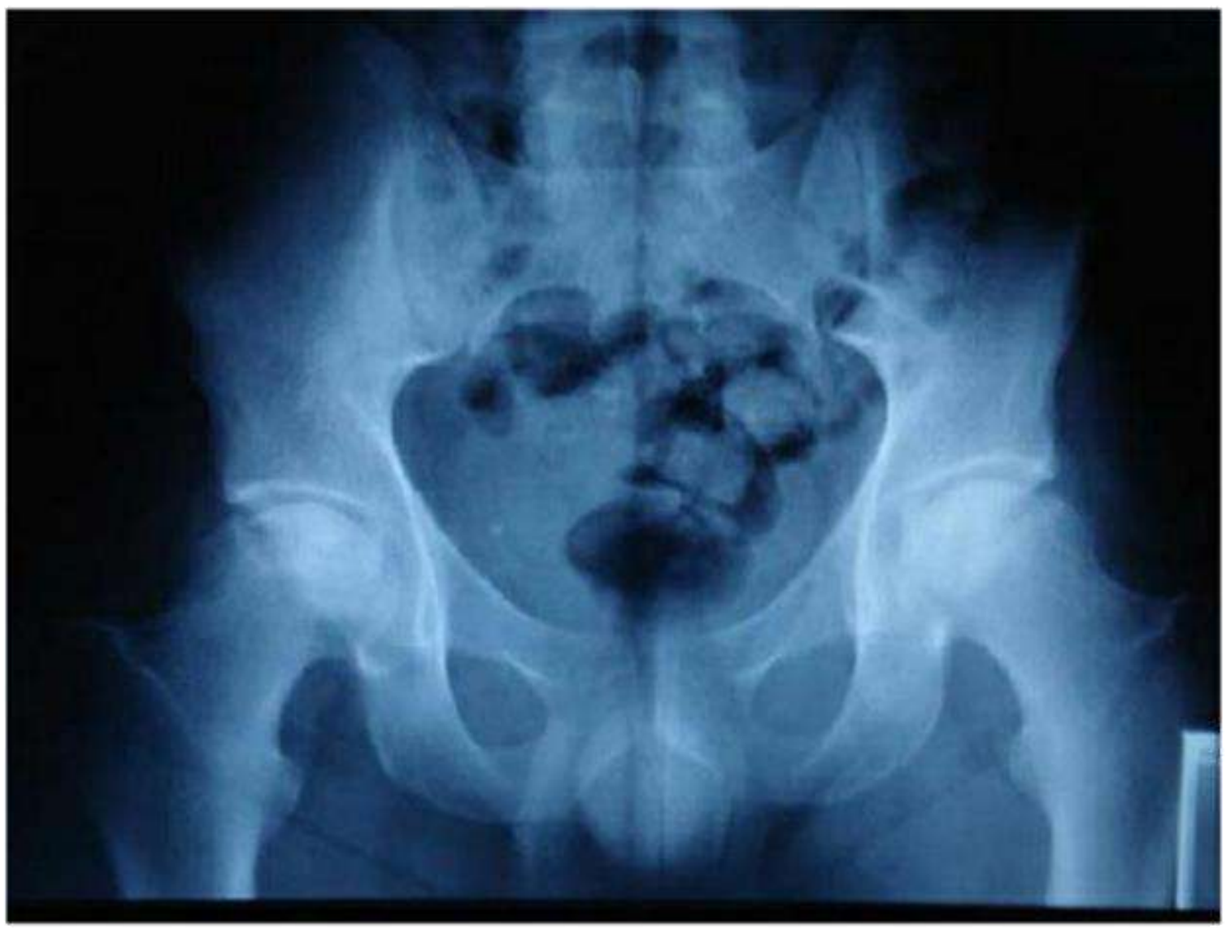
Investigations

1. Radiographs
- AP –coccyx point to symphysis
-Functional radiograph in abduction and adduction
-False profile view to know anterior coverage

2. AVASCULAR NECROSIS

2. AVASCULAR NECROSIS

Shown radiograph of avascular necrosis hip. classify and discuss management



Etiology - Direct/indirect/rare causes

1. Trauma, Genetic
2. Coagulopathies – (new theory) sickle cell anemia
3. Steroids/Alcohol/smoking/Radiotherapy
4. Diseases- Systemic lupus erythematosus/ Renal/Inflammatory bowel disease/gout/hypercholesterolemia/ Gaucher's disease/Acquired immunodeficiency syndrome

Radiologic assessment

1. Ficat classification

a-MRI positive
b-Sclerosis /cysts
c- Collapse
d- Osteoarthritis

2. lesion-Pre collapse or post collapse/size/degree of collapse
3. Osteoarthritis

Treatment

No role for conservative -80% progress

Medical treatment
-bisphosphonates
-anticoagulants
-vasodilators

-lipid lowering agents

Surgical

1. Core decompression—Pre-collapse –small to medium size

Success rates

Stage 1-84%
Stage 2 – 65%
Stage 3 – 40%

2. Vascular fibular grafts

-good results in stage 3-small to medium size lesions, but difficult/time consuming procedure

3. Osteotomy

Two types-intertrochanteric/rotational

Inter trochanteric- varus/valgus osteotomies

Rotational – Sugioka

Problem with osteotomy

1. Difficult hip replacement later on

So osteotomy used only in patients with
-Stage 1 and 2(small lesion with no collapse)
-Unilateral
-good range of movement
-Not in corticosteroid induced

4. Bone graft- vascularised/non vascularised

Methods –through core decompression/trap door/femoral neck window

-Good results stage 1,2 and 3

Accepted treatment?

Ficat stage 1 and 2- core decompression+ drilling +_ bone grafting

Ficat stage 3 and 4 – Total Hip Replacement

Any role for resurfacing?

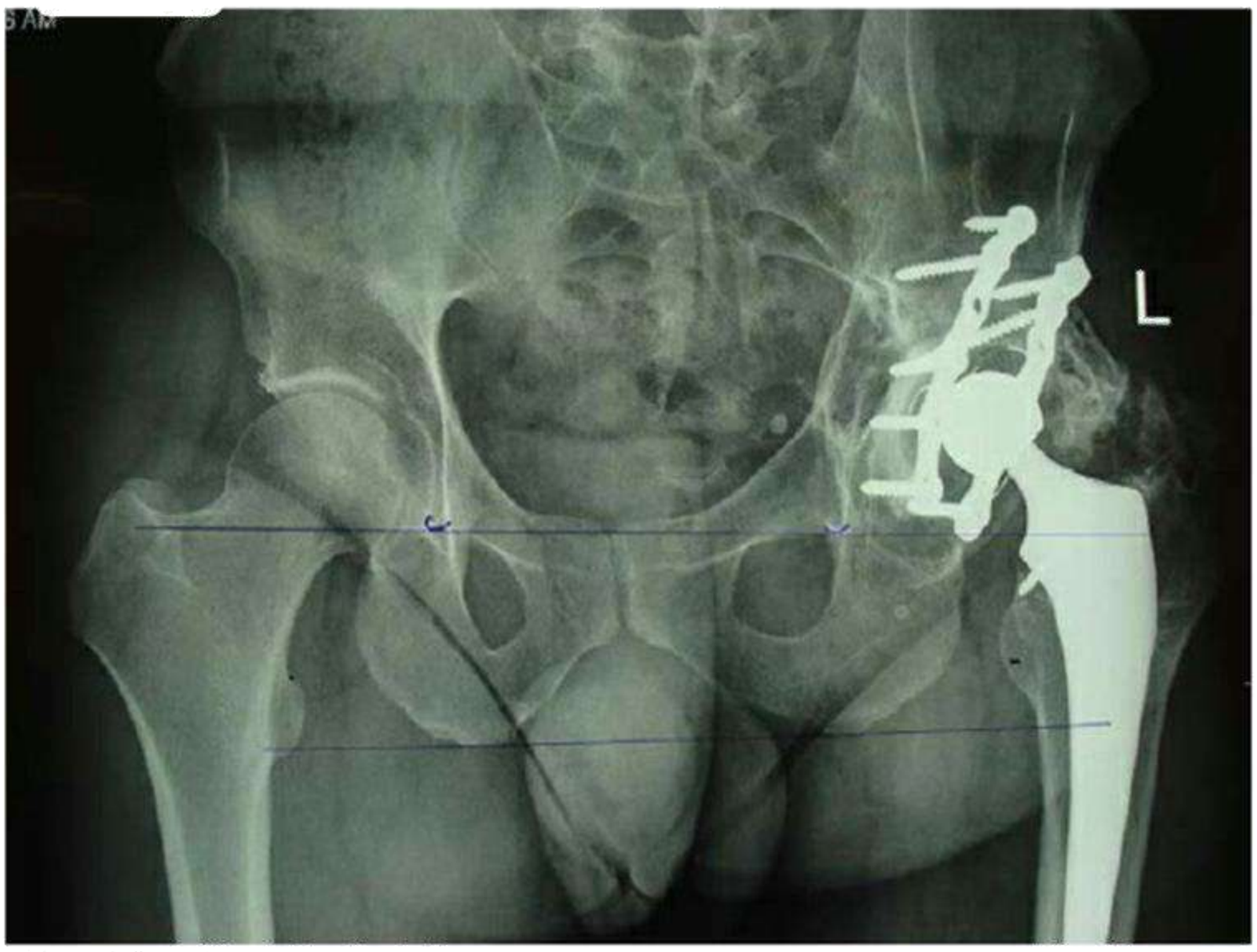
No- Contraindicated in large area of necrosis and cysts

References

4. Heterotrophic ossification

Radiograph of heterotopic ossification oh hip

10 of 11



Classification

Brooker

1. Islands of ossification
2. More than 1 cm gap
3. Less than 1 cm gap
4. Ankylosis

Contributing factors

1. More common in men
2. Ankylosing spondylitis
3. Diffuse idiopathic skeletal hyperostosis(DISH)
4. Head injury
5. Post traumatic osteoarthritis
6. Previous arthrodesis

Histology –as in myositis ossificans, with mature periphery and immature centre

Treatment

Observe- if minimal pain and reasonable range of movement

Surgery

-If very stiff

-If very stiff
-After one year of maturation

1. Excise bone and radiotherapy

Prevention in high risk cases

1. Indomethacin: 75mg for six weeks
2. Radiotherapy (800 cgy)

References-
1. Shehabetal. Heterotopic Ossification. JNucMed. 2002. 43(3). 346. 353.

7.Pediatrics :

A. Oral

Sprengel's Deformity :

The most common congenital shoulder anomaly in children , with a small and undescended scapula often associated with scapular winging hypoplasia omovertebral connection between medial angle of scapula and cervical spine

Developmental Dysplasia of the Hip (DDH) :

Disorder of **abnormal development** resulting in dysplasia, subluxation, and possible dislocation of the hip secondary to **capsular laxity** and **mechanical factors**

Developmental Coxa Vara

A decreased neck-shaft angle that is associated with an **ossification defect** in inferior femoral neck physis or ossification center leads to :

1. **decreased** proximal femoral neck-shaft angle (**Varus**)
2. **vertical** position of the proximal femoral **physis**

Legg-Calve-Perthes Disease

idiopathic avascular necrosis of the proximal femoral epiphysis in children
occurs secondary to disruption of blood supply to femoral head followed by **revascularization** with subsequent **resorption** and later **collapse**

SCFE :

condition of the proximal femoral physis that leads to slippage of the **metaphysis** relative to the epiphysis in which metaphysis translates anterior and externally rotates while epiphysis remains in the acetabulum, lies posterior to the translated metaphysis
and is most commonly seen in **adolescent** obese males ,

Infantile Blount's Disease (tibia vara)

progressive **pathologic** genu **varum** centered at the tibia divided into Infantile Blount's & Adolescent Blount's

Neurofibromatosis

an autosomal dominant disorder of neural crest origin characterized by

SCH

Nerve injury in SCH #

1. Posteromedial displacement of the fracture is more common (75%) and associated with radial nerve injury

2. Posterolateral displacement is associated with brachial artery injury and median nerve injury

BUT the most common nerve injury with sch fx is AIN (5%) followed by radial nerve injury (4%)

Ulnar nerve injury with flexion type fx

important technical points for fixation of SCH # with lateral entry pins :

1. Maximize separation of the pins at the fracture site

2. Engage the medial and lateral columns proximal to the fracture.

3. Engage sufficient bone in both the proximal segment and the distal fragment

4. Maintain a low threshold for use of a third lateral entry pin

Verify points to check for a good reduction SCH #:

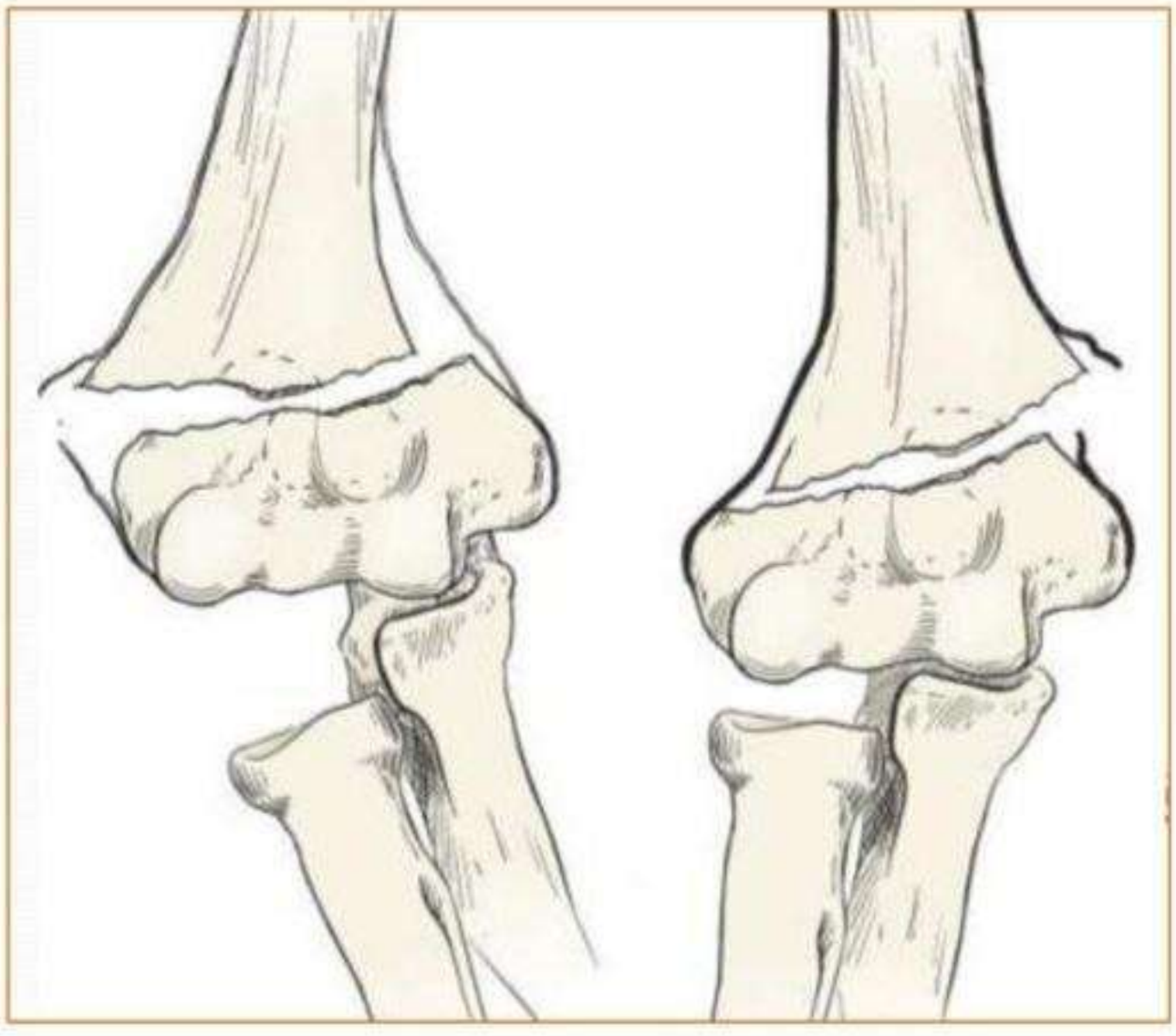
(1) the AHL intersects the capitellum

(2) Baumann's angle is greater than 10 degrees

(NL : 9-26)

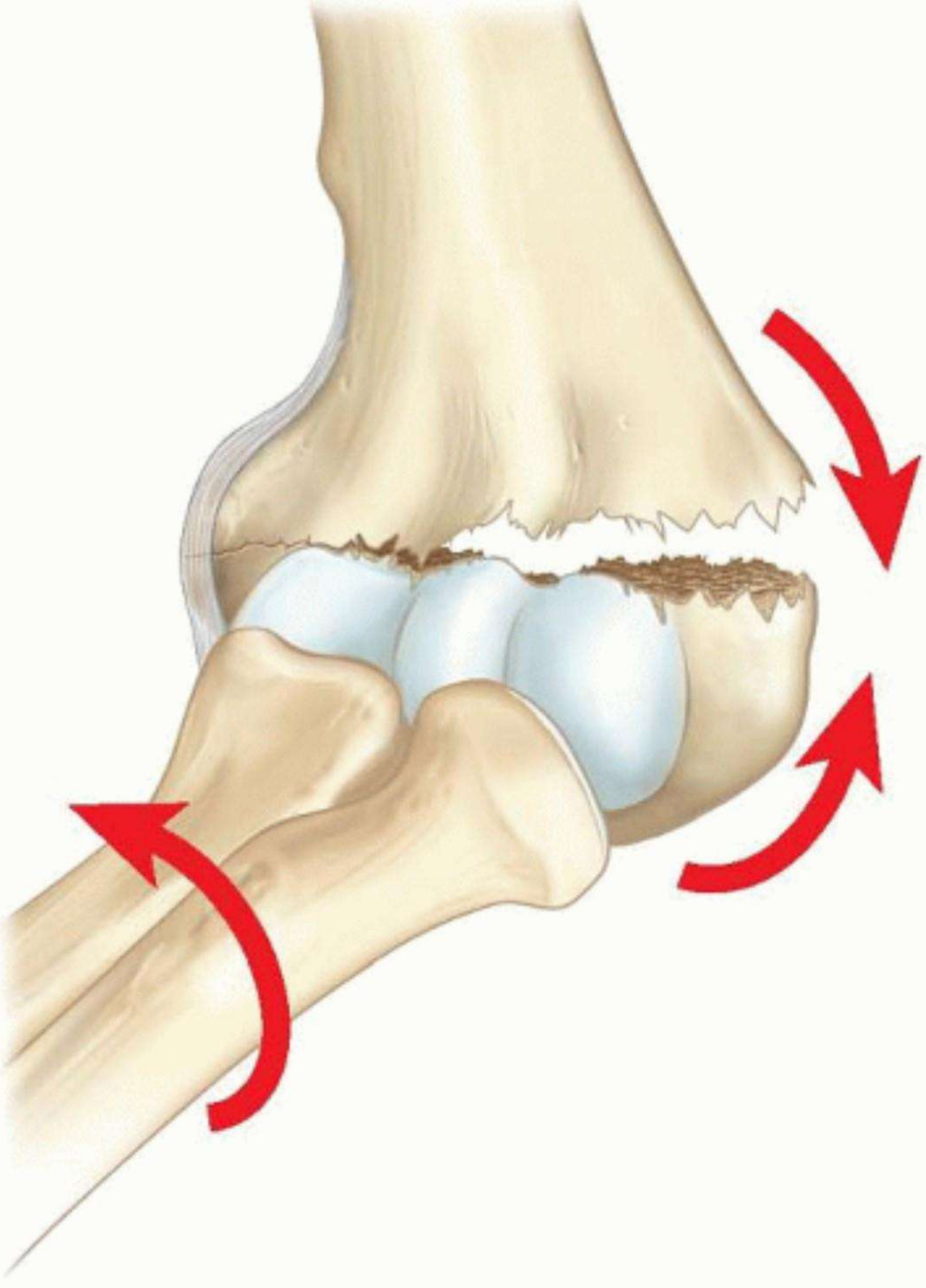
(3) the medial and lateral columns are intact on oblique views

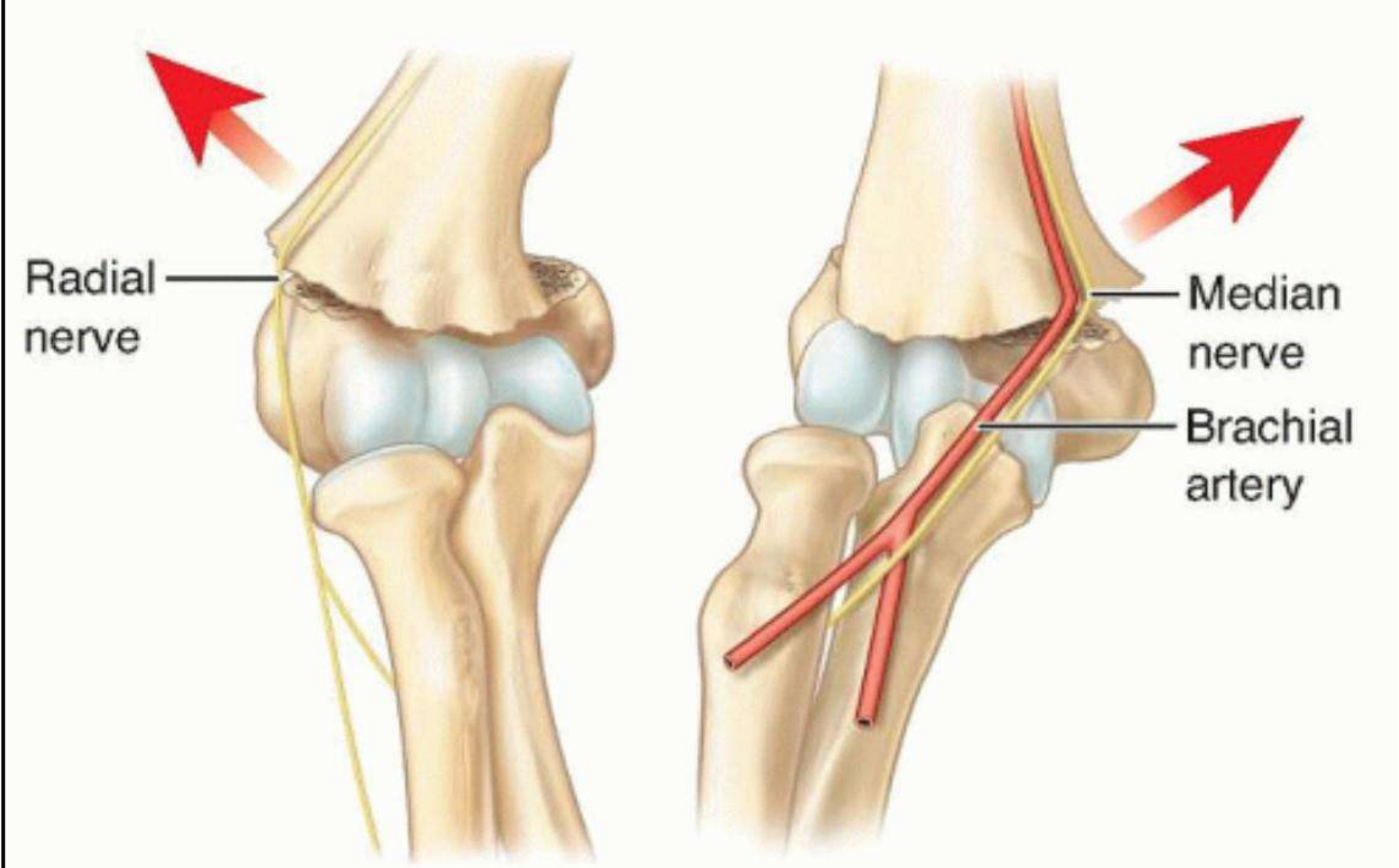
Supracondylar #- Gartland Classification



Type-III Complete displacement (extension type) may be:

- Posteromedial (75%), or
- Posterolateral (25%)




Radial nerve
Median nerve
Brachial artery

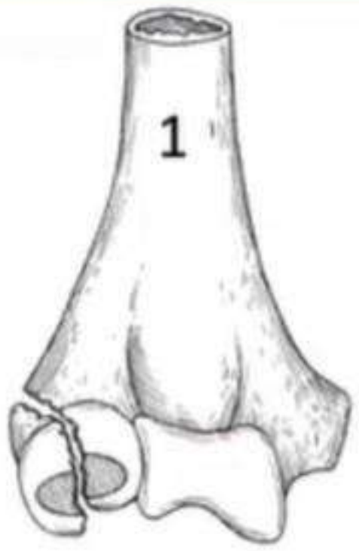
Surgical Approach

- direct anterior approach in cases of neurovascular compromise
- lateral approach
- medial approach in flexion type .

Milch Classification 1946

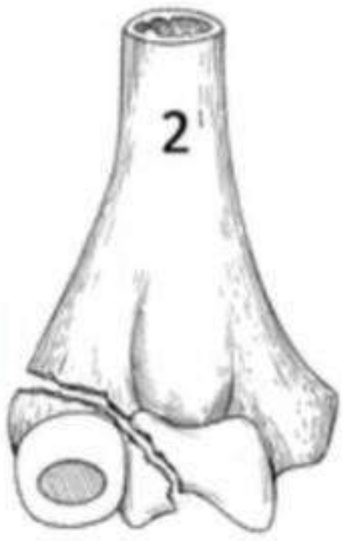
- **Type 1**
  - Rare
  - Impaction/Shear force
  - S&H type IV

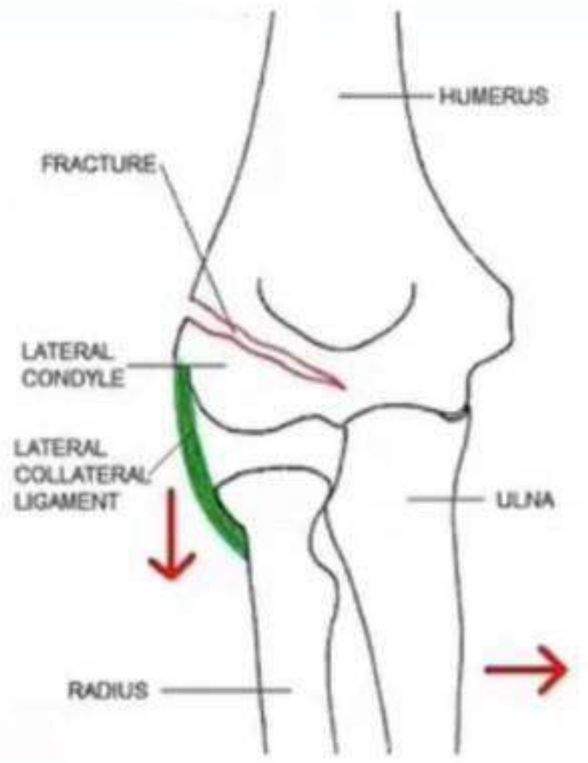
SH 4



- **Type 2**
  - Avulsion
  - S&H II

SH 2

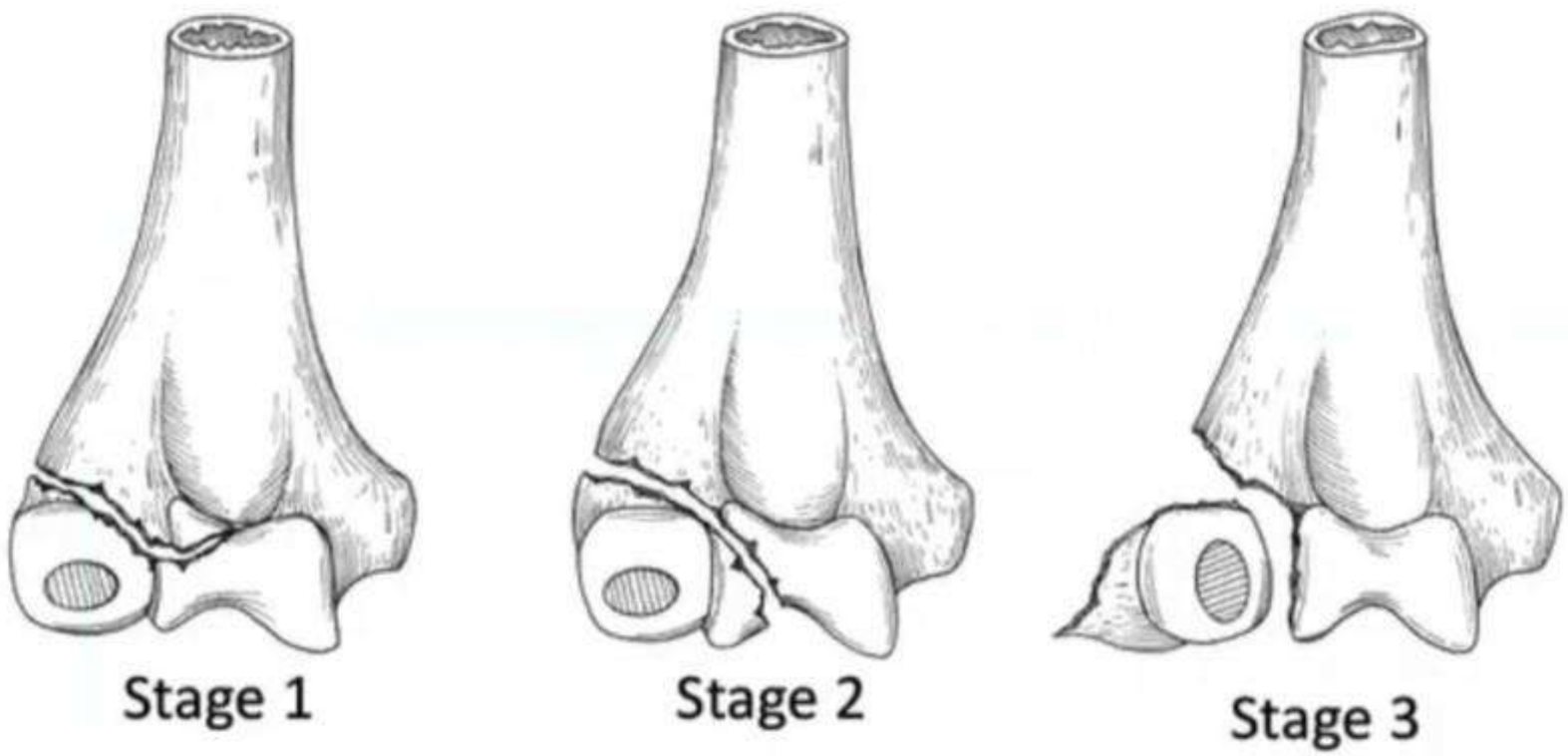




Mechanistic classification, not of much help in TTT

Jacob Classification 1975

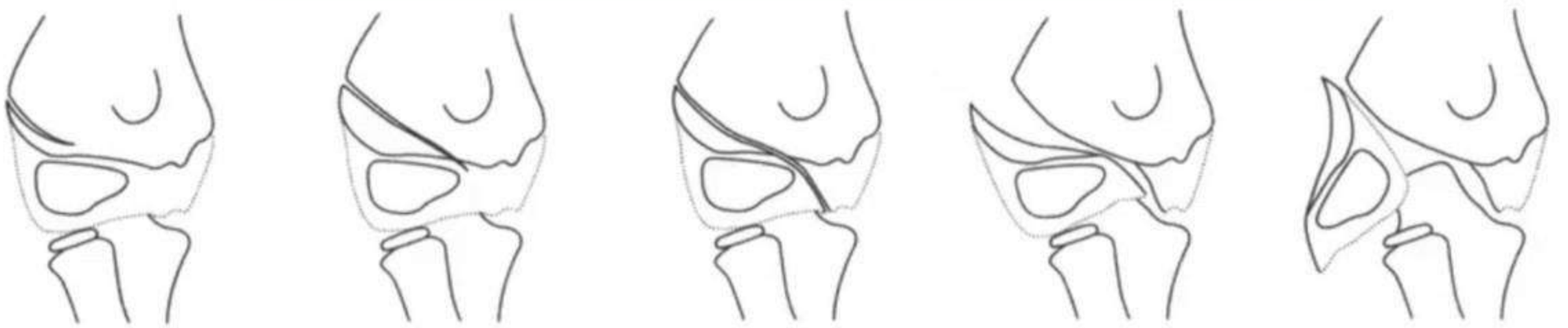
- Stage 1 > Intact articular surface <2 mm (non-displaced, stable)
- Stage 2 > Disrupted articular surface (minimally displaced, potentially unstable
- Stage 3 > Displaced >4mm, Rotated fragment



Song et al., JBJS, 2008

Stage	Degree of Displacement	Fracture Pattern	Radiograph Views Used as Basis	Stability
1	≤2 mm	Limited fracture line within the metaphysis	All 4 views	Stable
2	≤2 mm	Lateral gap	All 4 views	Indefinable
3	≤2 mm	Gap as wide laterally as medially	Any of 4 views	Unstable
4	>2 mm	Without rotation of fragment	Any of 4 views	Unstable
5	>2 mm	With rotation of fragment	Any of 4 views	Unstable

AP and Internal oblique view



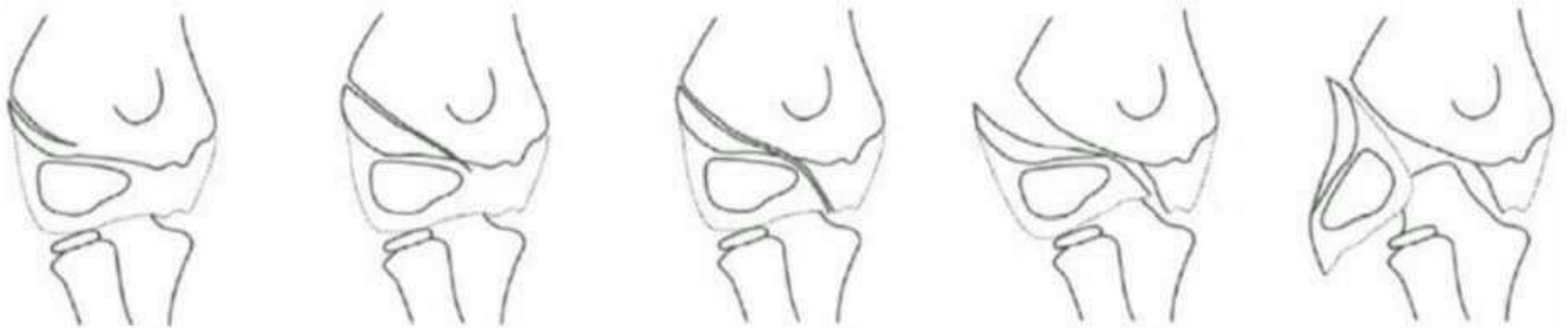


FIGURE 19-9 Stages of displacement of fractures of the lateral humeral condyle in children. Stage I, stable fracture with 2 mm or less of displacement and fracture line limited to within the metaphysis. Stage II, indefinable fracture with 2 mm or less of displacement and fracture line extending to the epiphyseal articular cartilage; there is a lateral gap. Stage III, unstable fracture with 2 mm or less of displacement and a gap that is wide laterally as medially. Stage IV, unstable fracture with displacement of more than 2 mm. Stage V, unstable fracture with displacement of more than 2 mm with rotation. (Reproduced with permission from: Song KS, Kang CH, Min BW, et al. Closed reduction and internal fixation of displaced unstable lateral condylar fractures of the humerus in children. *J Bone Joint Surg Am.* 2008;90:2673–2681.)

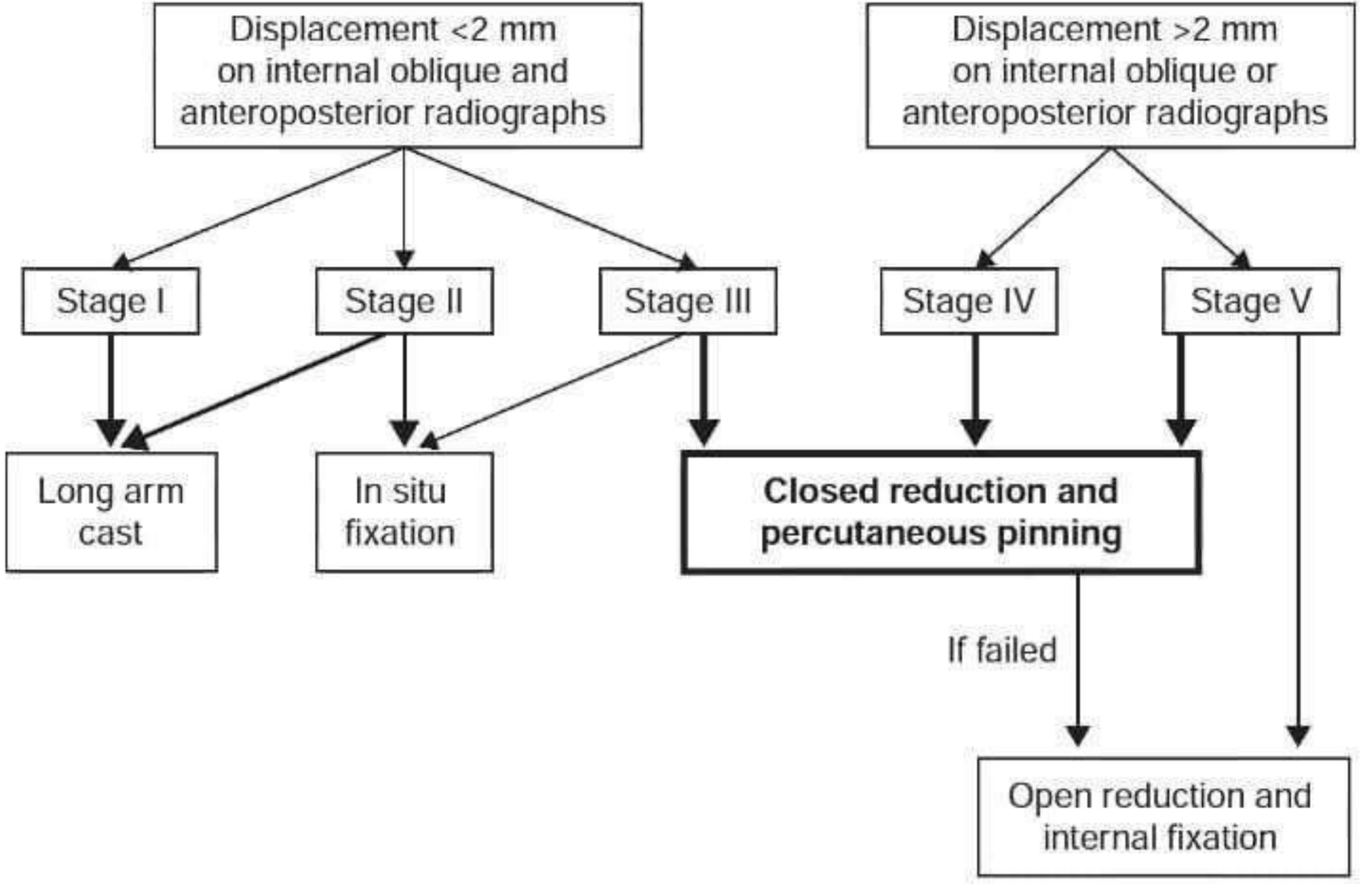


FIGURE 19-10 Treatment algorithm based on stage of fracture displacement described in Fig. 19-9. (Reproduced with permission from Song KS, Kang CH, Min BW, et al. Closed reduction and internal fixation of displaced unstable lateral condylar fractures of the humerus in children. *J Bone Joint Surg Am.* 2008;90:2673–2681.)

Difference Between Condyle and Epicondyle

October 18, 2017 • by Lakna • 4 min read

Main Difference – Condyle vs Epicondyle

Bones play a critical role in providing support and aiding the movement of animals. Among the different types of bone in the body, long bones such as femur, tibia, ulna, and humerus are especially involved in the movement of the body. Some surfaces of the long bones are soft and the other surfaces can be rough. Some surfaces may contain hills and valleys. Condyle and epicondyle occur at the end of the long bones. The condyle is more prominent than the epicondyle. The condyle is smooth and round whereas epicondyle is rough. Epicondyle is a projection on the condyle. The **main difference** between condyle and epicondyle is that **condyle forms an articulation with another bone. whereas epicondyle provides sites for the attachment of muscles**.

CONDYLE
VERSUS
EPICONDYLE

CONDYLE	EPICONDYLE
Condyle refers to a rounded protuberance at the end of a bone, forming an articulation with another bone	Epicondyle refers to a protuberance on the condyle of a long bone
A large structure at the end of the long bones	A small structure on top of the condyle
A smooth, rounded structure	A rough projection
Forms an articulation with another bone	Provides sites for the attachment of muscles
Medial and lateral condyle are examples	Medial and lateral condyle of the humerus and femur are examples

The **apophysis** is a site of tendon or ligament attachment, as compared to the **epiphysis** which contributes to a joint, and for that reason, it is also called 'traction **epiphysis**'. When unfused, **apophyses** can easily be mistaken for fractures.

CANDIDATE: The risk factors for patellar instability are:

1. Bony factors (static)

   Trochlear dysplasia.

   Hypoplastic femoral condyle.

   Patellar shape.

   Patella alta.

2. Malalignment

   Patellar malalignment is an abnormal rotational or translational deviation of the patella along any axis.

   External tibial torsion/foot pronation.

   Increased femoral anteversion and increased genu valgum.

   Increased Q angle or abnormal tibial tuberosity–trochlear groove (TT–TG) distance.

3. Soft tissue (dynamic)

   Ligamentous laxity (medial patellofemoral ligament rupture/ insufficiency).

4. Abnormal gait

   Walking with valgus thrust.

5. Genetic factors such as connective tissue disorder syndromes.

Risk factors include:

1. Female,
2. Q angle $> 20°$ (normal is 10° in boys and 15° in girls),
3. Genu valgus,
4. Rotational abnormalities, such as increased femoral anteversion or external tibial torsion,
5. Patella alta (Figure 4.17),
6. A shallow patella-femoral sulcus angle (ABC $> 144°$ is abnormal) (Figure 4.17),
7. Abnormal congruence angle of Merchant (OBX is normally $-6°$ to $-8°$). This angle is abnormal if it is more than +16°. Positive (+) means lateral while negative (−) means medial (Figure 4.17),
8. Vastus medialis obliquus hypoplasia,
9. Generalized ligamentous laxity,
10. Pes planus,
11. Lateral mobility greater than 3/4. (Medial mobility less than 1/4 indicates a tight lateral reticulum.)

Several classification systems have been developed for physeal fractures. The most widely used system today is the Salter–Harris classification (Figure 12.55).

- Type I injuries involve complete separation of the epiphysis through the physis without fracture through the metaphysis.
- Type II injuries involve separation of a portion of the physis with the fracture progressing out of the metaphysis.
- Type III injuries involve a fracture that runs through a portion of the physis and out through the epiphysis.
- Type IV fractures are longitudinal splits through the epiphysis, physis and metaphysis.
- Type V fractures involve a crush injury of the growth plate and are not evident on radiographs at the time of injury.

This classification has been modified by Peterson, Rang and Ogden. Peterson added another two types: Type VI with metaphyseal fractures extending to the physis and Type VII with loss of the physis (VIIa for central and VIIb for peripheral). (Figure 12.56.)

Ogden from his series of 443 physeal fractures has added another three (Figure 12.57):

Type VII: Epiphyseal fractures not involving physis.
Type VIII: Metaphyseal fractures affecting later growth.
Type IX: Periosteal damage affecting later growth.

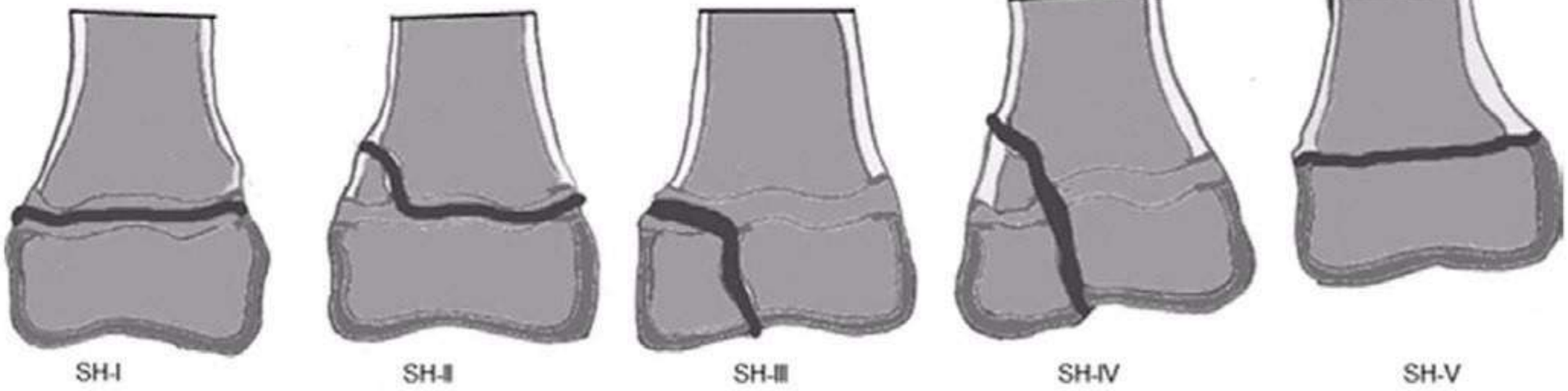


Figure 12.55 Salter–Harris classification.

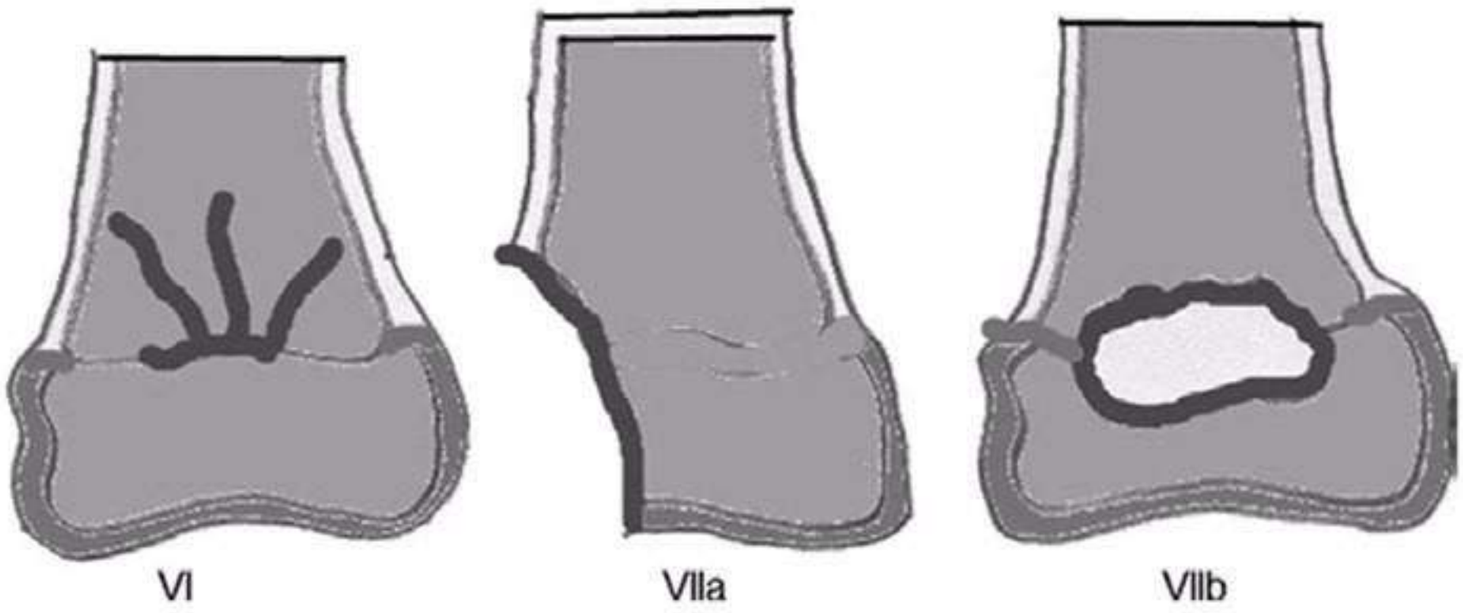


Figure 12.56 Peterson modification of Salter–Harris classification.

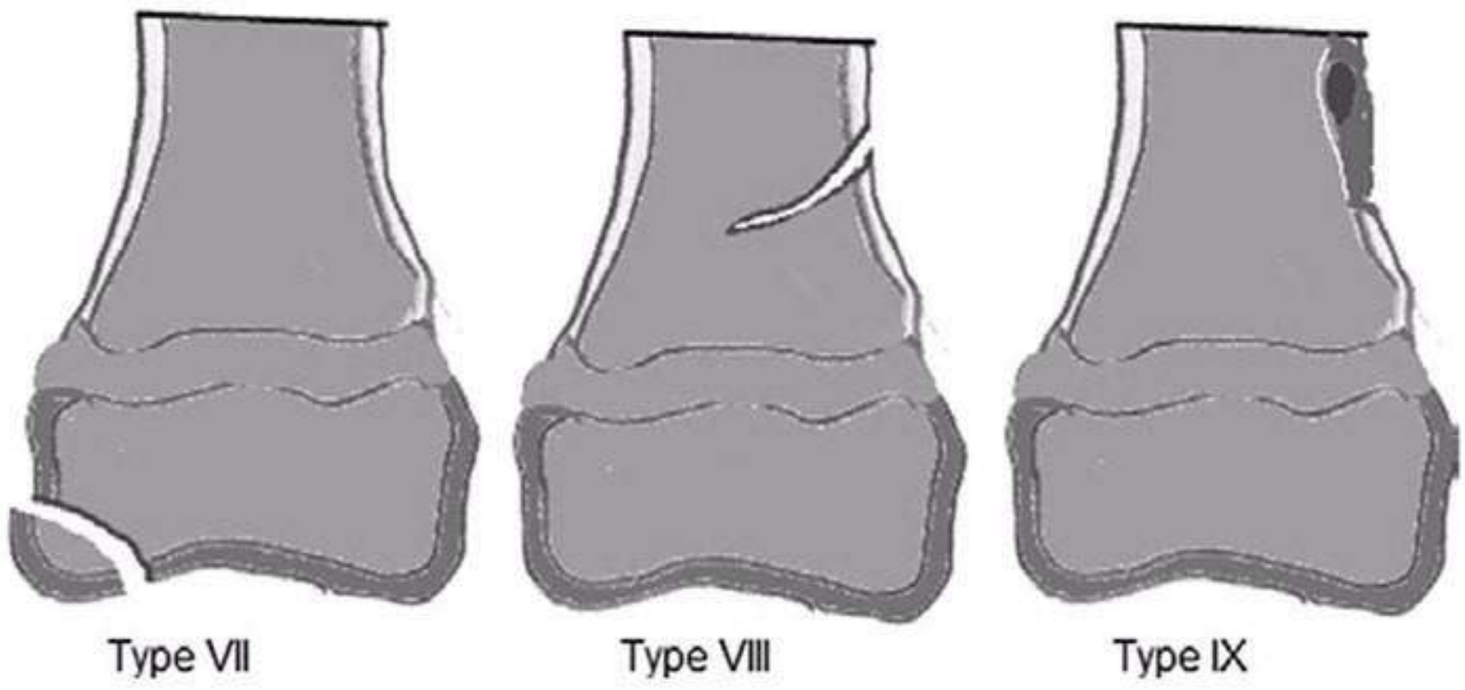


Figure 12.57 Ogden modification of Salter–Harris classification.

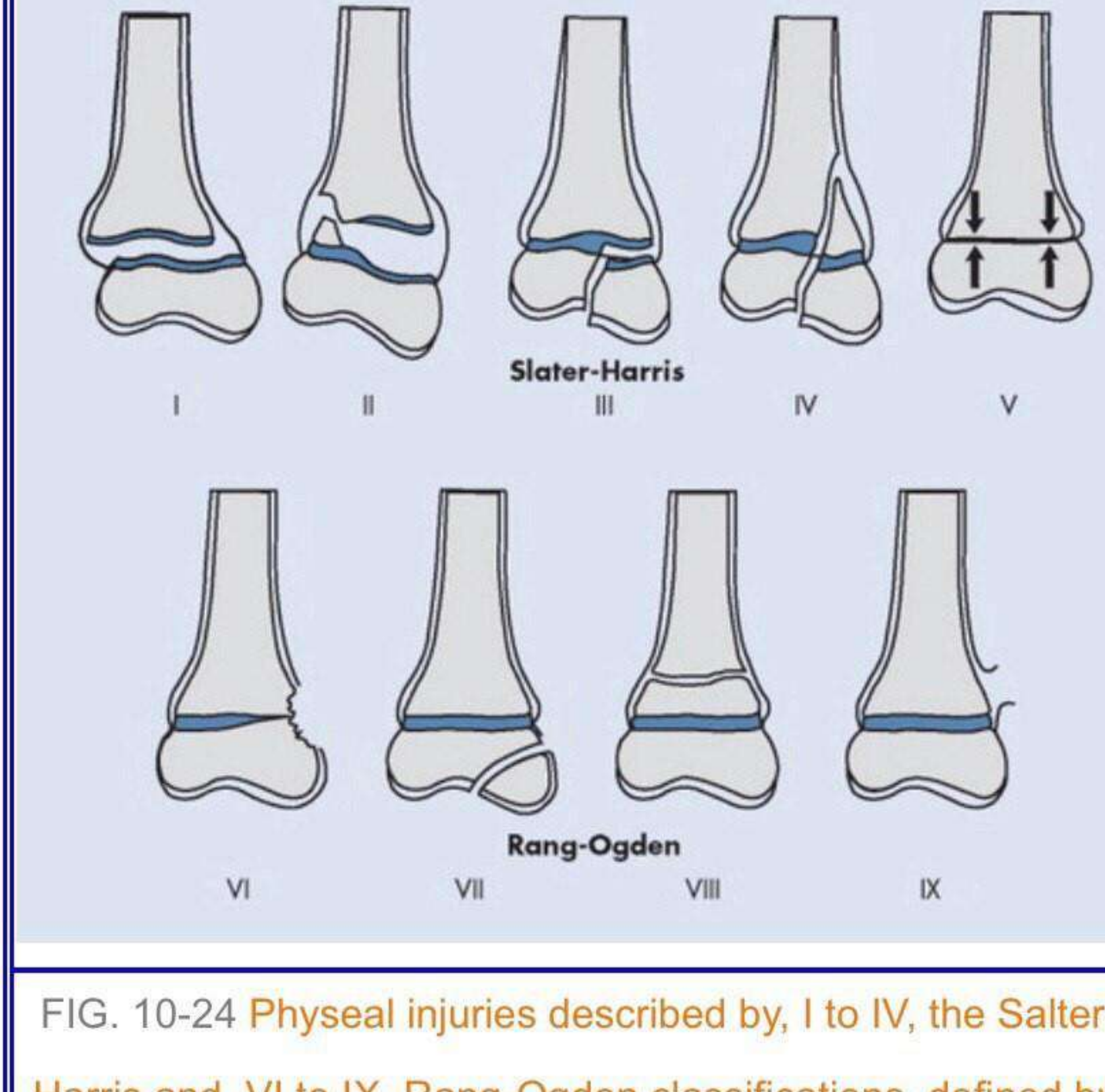
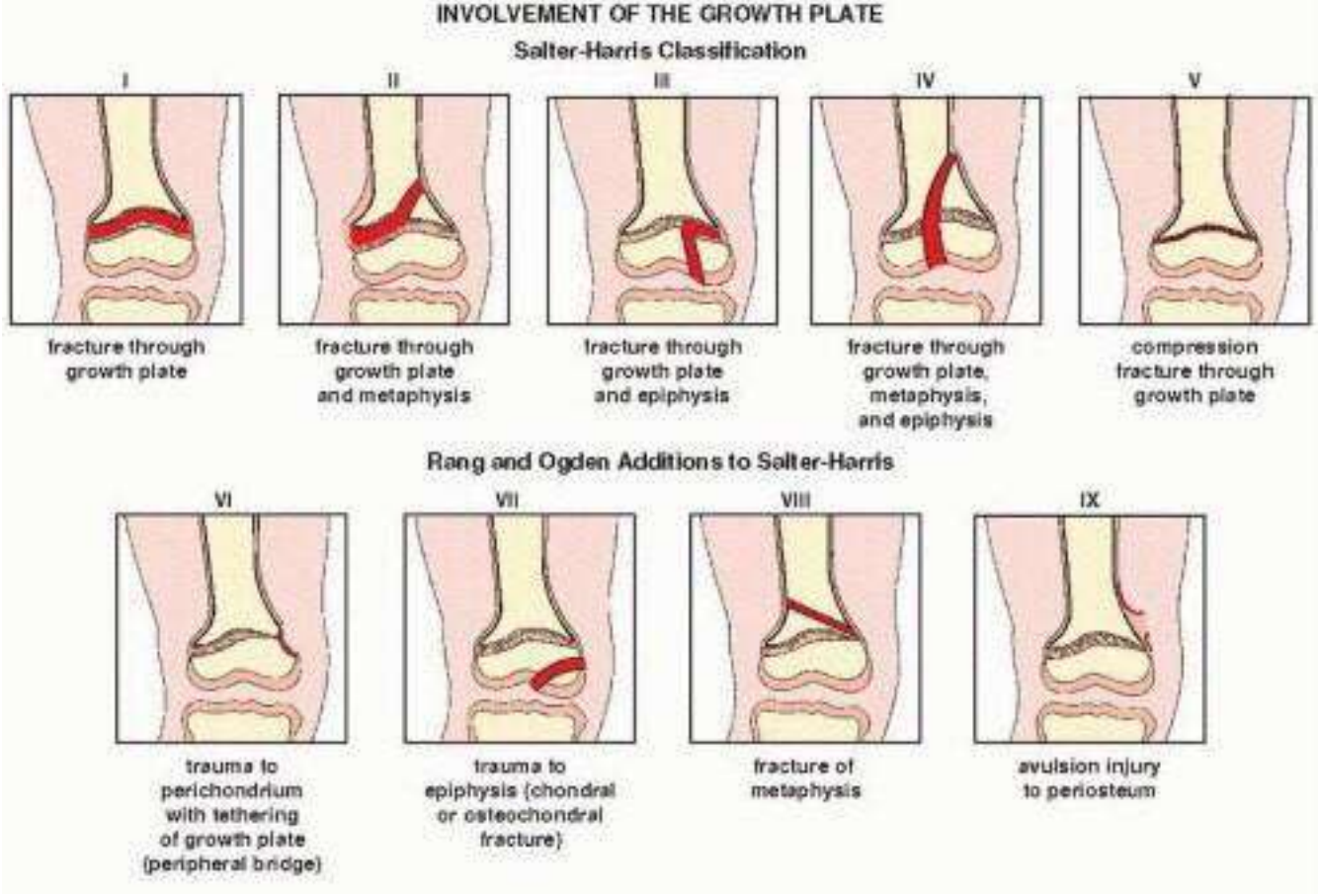
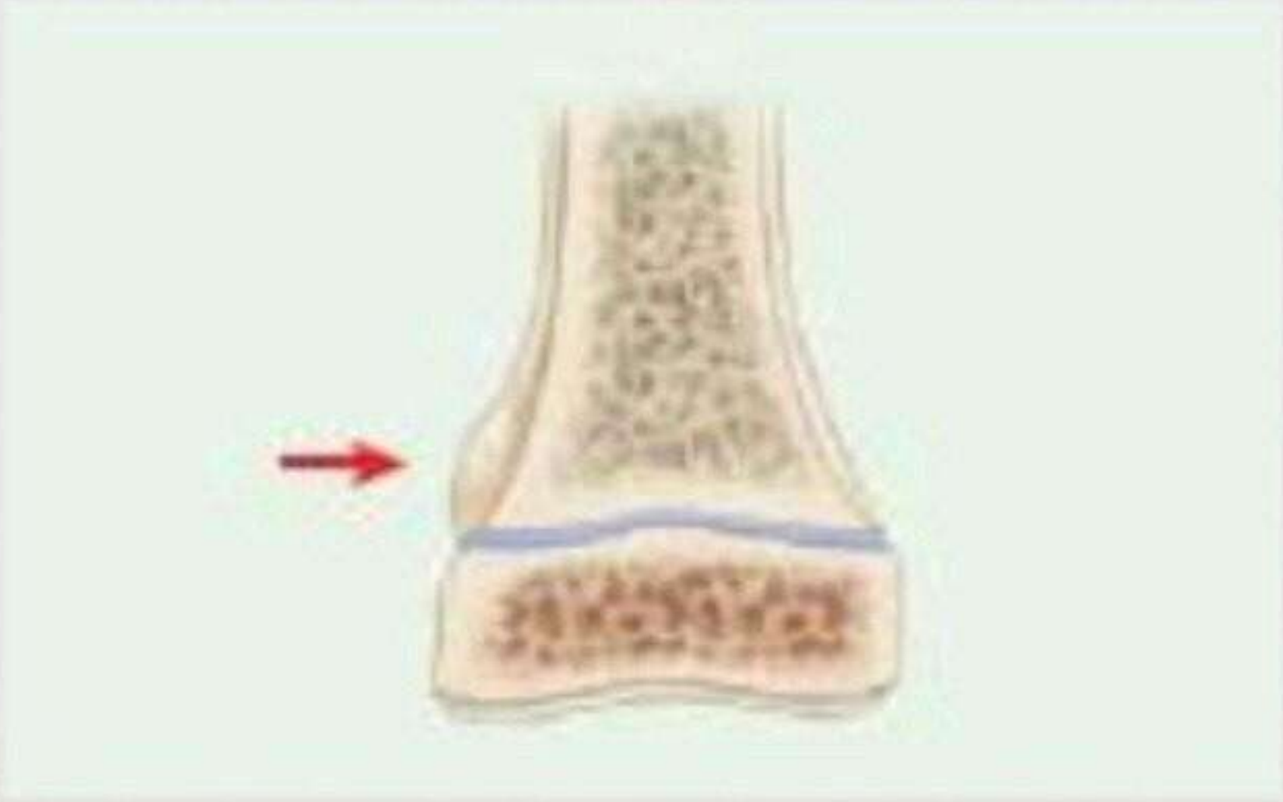


FIG. 10-24 Physeal injuries described by, I to IV, the Salter-Harris and, VI to IX, Rang-Ogden classifications, defined by, I, fracture through the physis; II, fracture through the physis and metaphysis; III, fracture through the physis and epiphysis; IV, fracture through the metaphysis, physis, and epiphysis; V, compression injury of the physis; VI, injury to the perichondrium; VII, osteochondritis dissecans; VIII, injury to the metaphysis; and, IX, injury to the periosteum.


INVOLVEMENT OF THE GROWTH PLATE
Salter-Harris Classification
I
II
III
IV
V
fracture through growth plate
fracture through growth plate and metaphysis
fracture through growth plate and epiphysis
fracture through growth plate, metaphysis, and epiphysis
compression fracture through growth plate
Rang and Ogden Additions to Salter-Harris
VI
VII
VIII
IX
trauma to perichondrium with tethering of growth plate (peripheral bridge)
trauma to epiphysis (chondral or osteochondral fracture)
fracture of metaphysis
avulsion injury to periosteum

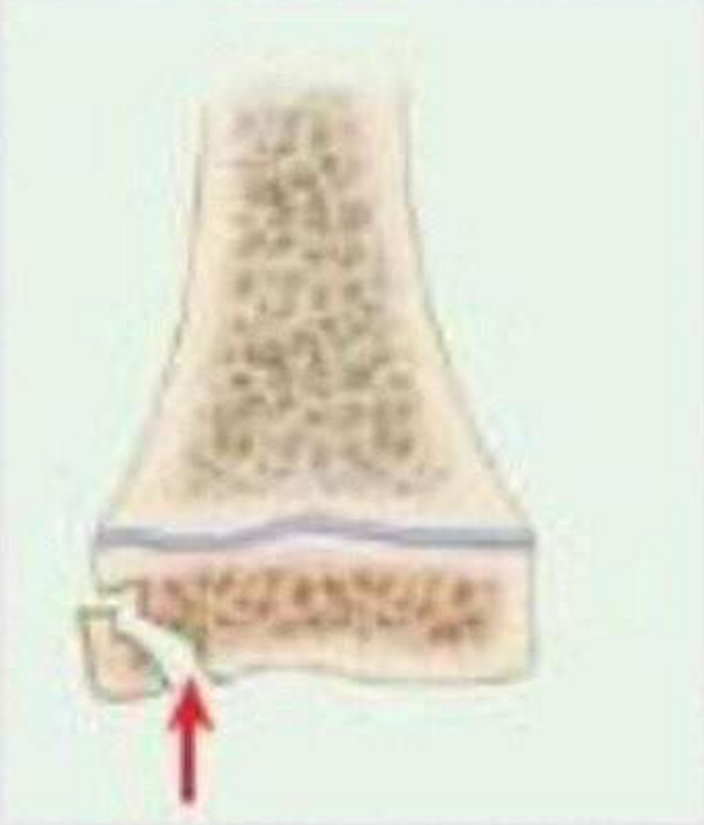
Type 6 injury RANG

- Injury to the perichondrial ring



Type 7 ogden

- Trauma to epiphysis (chondral to osteochondral)
- Isolated injury of the epiphyseal plate

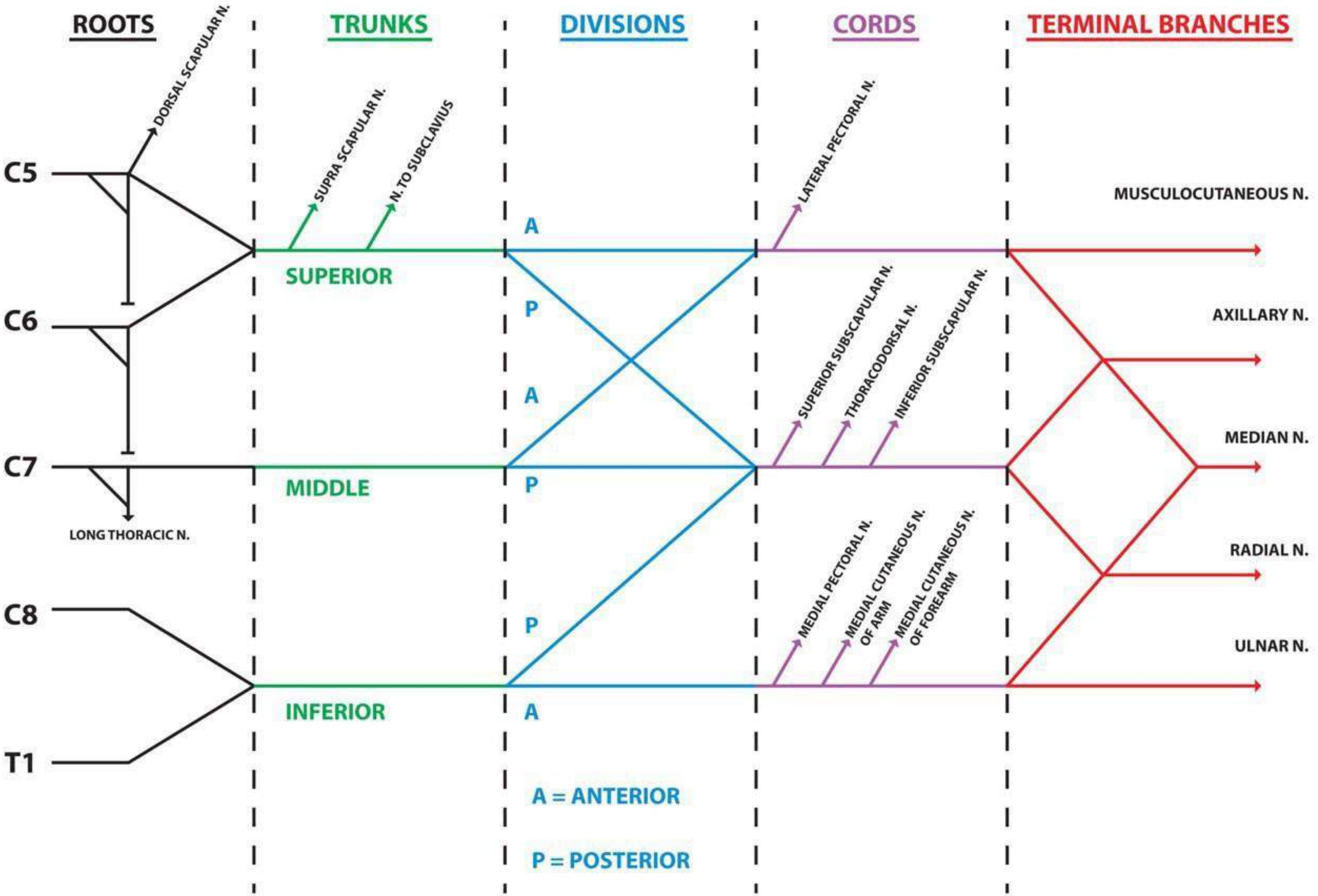


Type 8

- # of metaphysis
- Isolated injury of the metaphysis with possible impairement of enchondral ossification

Type 9

- Avulsion injury to periosteum
- which may impair intramembranous ossification


ROOTS
TRUNKS
DIVISIONS
CORDS
TERMINAL BRANCHES
DORSAL SCAPULAR N.
C5
C6
C7
C8
T1
LONG THORACIC N.
SUPRA SCAPULAR N.
N. TO SUBCLAVIUS
SUPERIOR
MIDDLE
INFERIOR
A
P
A
P
P
A
LATERAL PECTORAL N.
SUPERIOR SUBSCAPULAR N.
THORACODORSAL N.
INFERIOR SUBSCAPULAR N.
MEDIAL PECTORAL N.
MEDIAL CUTANEOUS N. OF ARM
MEDIAL CUTANEOUS N. OF FOREARM
MUSCULOCUTANEOUS N.
AXILLARY N.
MEDIAN N.
RADIAL N.
ULNAR N.
A = ANTERIOR
P = POSTERIOR

Physiologic Classification	
Spastic (most common)	Velocity-dependent increased muscle tone and hyperreflexia with slow, restricted movement due to simultaneous contraction of agonist and antagonist muscles. Most amenable to operative treatments.
Athetoid	Characterized by constant succession of slow, writhing, involuntary movements
Ataxic	Characterized by inability to coordinate muscle movements. Results in unbalanced, wide based gait.
Mixed	Usually mixed spastic and athetoid features and involves the entire body
Hypotonic	Usually precedes spastic or ataxic for 2-3 years
Anatomic Classification	
Quadriplegic	Total body involvement and nonambulatory
Diplegic	Legs more than arms but usually still ambulatory. IQmay be normal (injury in brain is midline) ?
Hemiplegic	Arms and legs on one side of the body, usually with spasticity; will eventually be able to walk, regardless of treatment
Gross Motor Function Classification Scale (GMFCS)	
Level I	Near normal gross motor function, independent ambulator
Level II	Walks independently, but difficulty with uneven surfaces, minimal ability to jump
Level III	Walks with assistive devices
Level IV	Severely limited walking ability, primary mobility is wheelchair
Level V	Nonambulator with global involvment, dependent in all aspects of care

- children are more likely to have extrapulmonary involvement
- biopsy with stains and culture for acid-fast bacilli is diagnostic
- Salmonella
- more common in sickle cell patients
- pathoanatomy
- acute osteomyelitis
- most cases are hematogenous
- initial bacteremia may occur from a skin lesion, infection, or even trauma from tooth brushing
- microscopic activity
- sluggish blood flow in metaphyseal capillaries due to sharp turns results in venous sinusoids which give bacteria time to lodge in this region
- the low pH and low oxygen tension around the growth plate assist in the bacterial growth
- infection occurs after the local bone defenses have been overwhelmed by bacteria
- spread through bone occurs via Haversian and Volkmann canal systems
- purulence develops in conjunction with osteoblast necrosis, osteoclast activation, the release of inflammatory mediators, and blood vessel thrombosis
- macroscopic activity
- a subperiosteal abscess develops when the purulence breaks through the metaphyseal cortex
- septic arthritis develops when the purulence breaks through an intra-articular metaphyseal cortex (hip, shoulder, elbow, and ankle) (NOT KNEE)
- Infants <1 year of age can have infection spread across the growth plate via capillaries causing osteomyelitis in the epiphysis and septic arthritis
- chronic osteomyelitis
- periosteal elevation deprives the underlying cortical bone of blood supply leading to necrotic bone (sequestrum)
- sequestrum
- the necrotic bone which has become walled off from its blood supply and can present as a nidus for chronic osteomyelitis
- an outer layer of new bone is formed by the periosteum (involucrum)
- involucrum
- a layer of new bone growth outside existing bone seen in osteomyelitis
- chronic abscesses may become surrounded by sclerotic bone and fibrous tissue leading to a Brodie's abscess

- Prognosis

Classification

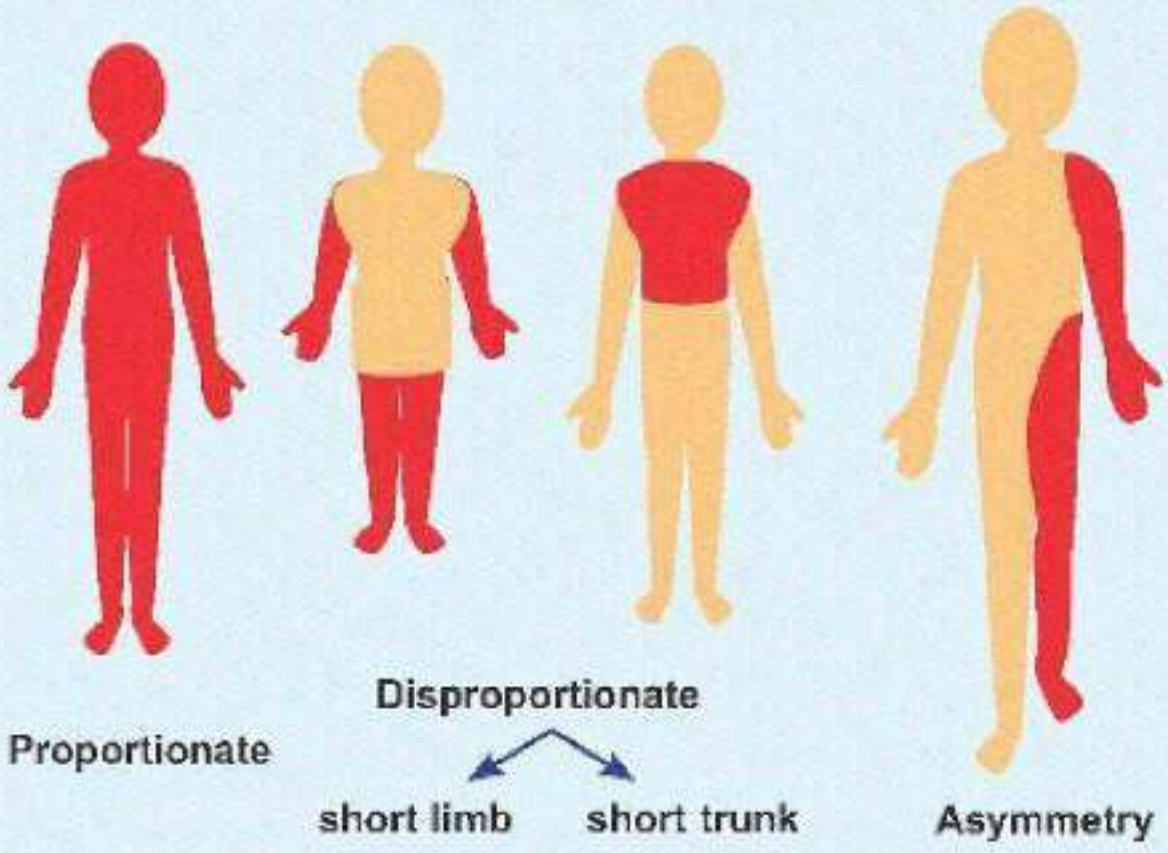
- Timing classification
  - acute
    - within 2 weeks
  - subacute
    - within one to several months
  - chronic
    - after several months
- Cierny-Mader classification

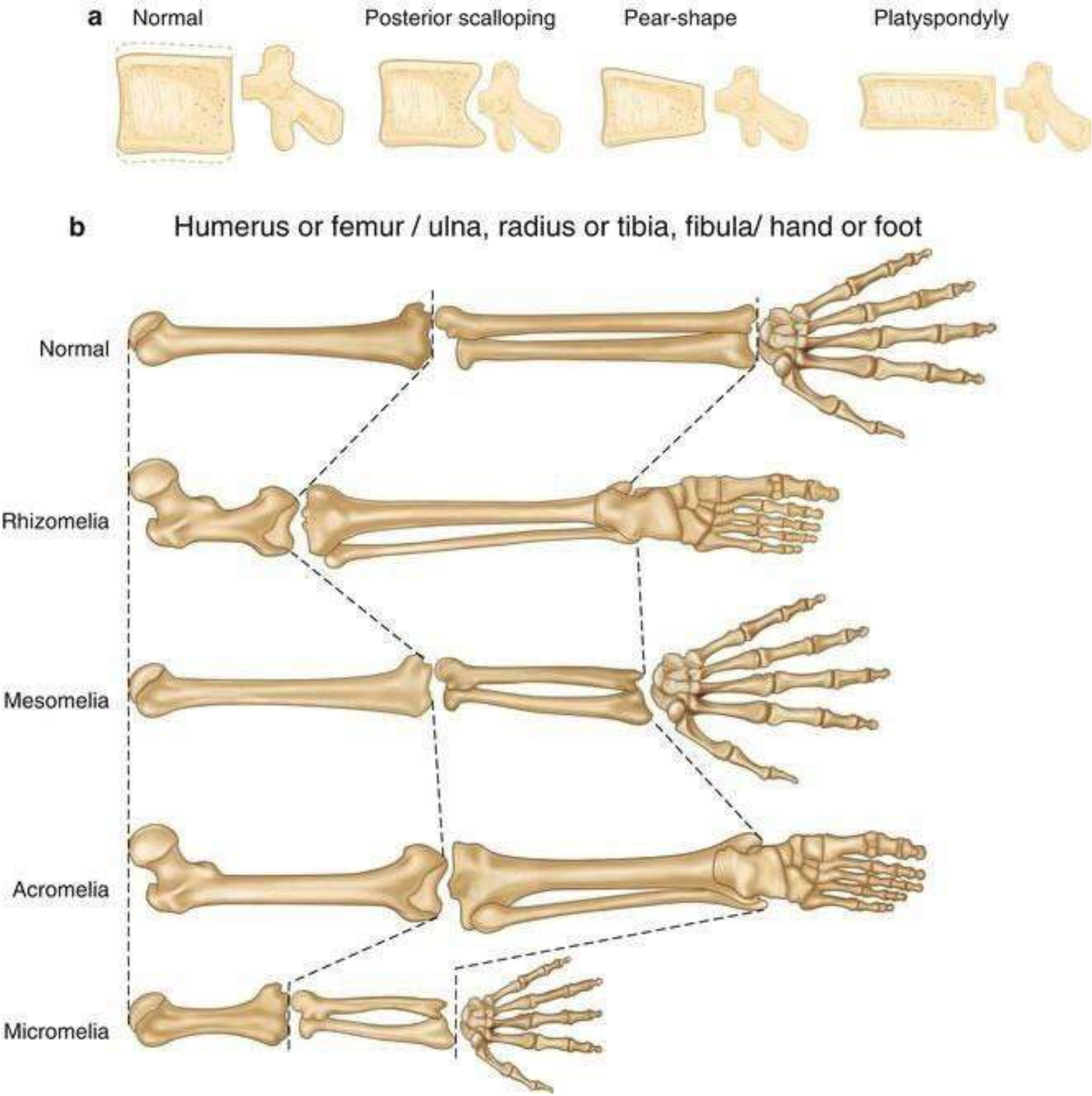
Cierny-Mader Classification of Osteomyelitis (describes anatomic involvement, host, treatment, prognosis)		
Anatomic Location		
Stage I	Medullary	
Stage 2	Superficial	
Stage 3	Localized	
Stage 4	Diffuse	
Host Type		
Type A	Normal	
Type BL	Locally compromised	
Type BS	Systemically compromised	
Type C	Treatment is worse to the patient than infection	

Table 2.2 The Beighton score

Criteria	Note
Little finger dorsiflexion	1 if >90°, 2 if bilateral
Thumb to forearm (wrist flexion)	1 if thumb tips touch the forearm skin, 2 if bilateral
Elbow extension	1 if hyperextension >10°, 2 if bilateral
Knee extension	1 if hyperextension >10°, 2 if bilateral
Trunk flexion with knees full extended	1 if palms can rest flat on the floor

The Beighton score is a nine-point score: the higher the score, the greater the laxity. The threshold for joint laxity in a young adult ranges from 4 to 6.


Disproportionate
Proportionate
short limb
short trunk
Asymmetry


a
Normal
Posterior scalloping
Pear-shape
Platyspondyly
b
Humerus or femur / ulna, radius or tibia, fibula/ hand or foot
Normal
Rhizomelia
Mesomelia
Acromelia
Micromelia

Rhizomelic

Mesomelic

Acromelic

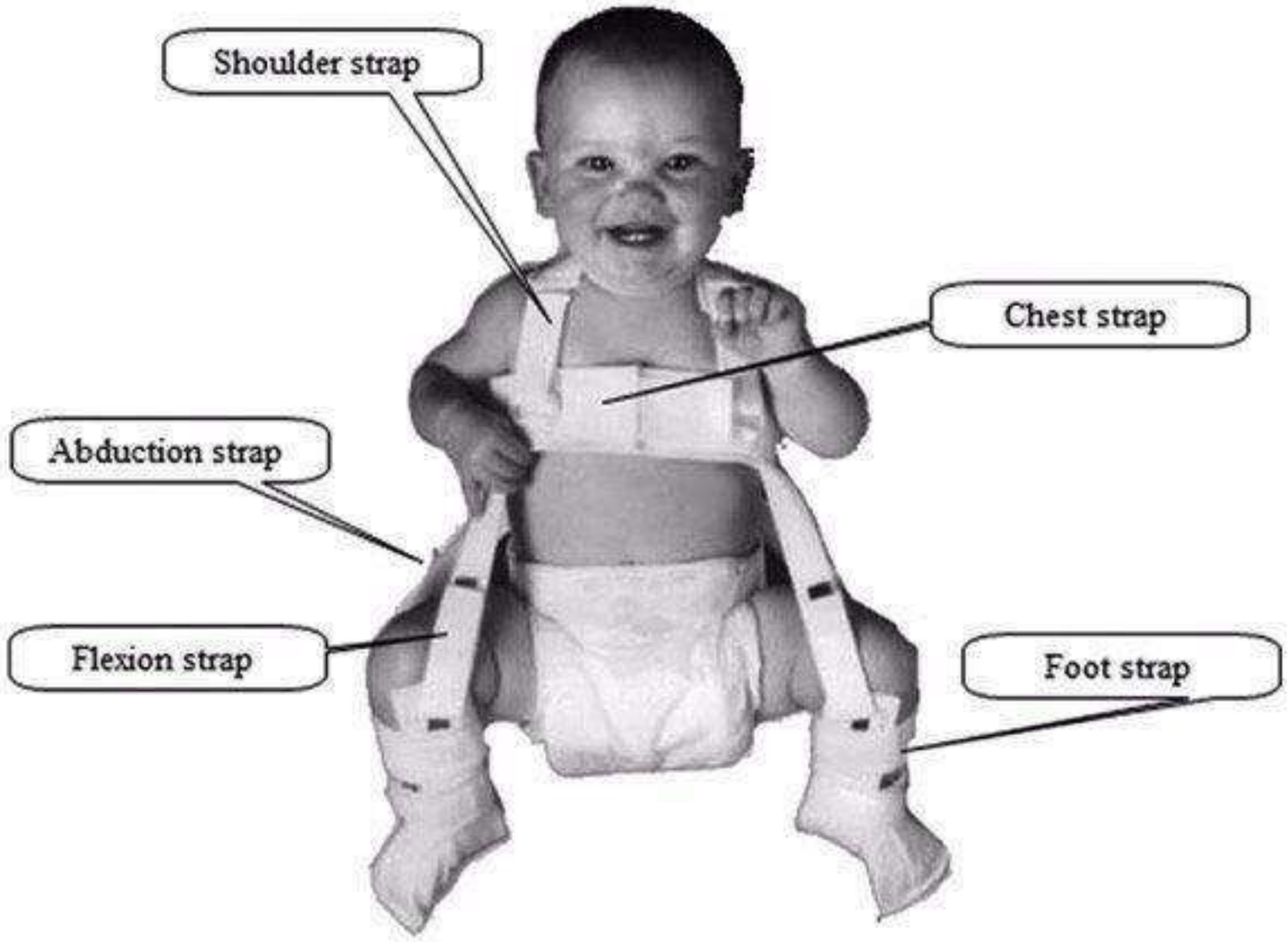


Figure 3.3.19 Pavlik harness (picture courtesy of Wheaton Brace Co.).

may cause the hip to re-dislocate. Forced abduction may lead to avascular necrosis of the femoral head.

It is essential to check a child with a Pavlik harness frequently (initially weekly then every 2 to 4 weeks) for reduction, ultrasound progression and fitness (as the child may outgrow the harness) and to document active knee extension (functioning femoral nerve). Pavlik harness treatment failure is predicted for children older than 7 weeks, bilateral hip dislocation and failure of reduction (negative Ortolani test).

Complications

1. Failure of reduction,
2. Damage to the posterior acetabular wall when there is a persistent posterior dislocation,
3. Avascular necrosis of the head of the femur (AVN) 2.4% (range, 0–15%),
4. Femoral nerve palsy,
5. Skin damage,
6. Brachial plexus injury,
7. Knee dislocation.

Murnaghan *et al.* [10] reported a femoral nerve palsy incidence of 2.5% (30 cases) in 1218 patients treated with a Pavlik harness, with 87% presenting within one week of harness application. Femoral nerve palsy was more likely in older, larger patients in whom the hip dysplasia was of greater severity. Patients whose femoral nerve palsy resolved within 3 days had a 70% chance of successful treatment using the Pavlik harness, whereas those who had not recovered by 10 days had a 70% chance of treatment failure. All patients had eventual complete return of full quadriceps function, with no clinically evident long-term motor or sensory deficit. The success rate associated with treatment with a Pavlik harness was 94% in the control group and 47% in the palsy group.

Contraindication of Pavlik harness

1. Major muscle imbalance, such as myelomeningocele,
2. Major stiffness as in arthrogryposis,
3. Ligamentous laxity, as in Ehlers–Danlos syndrome,
4. Severe respiratory compromise (a Craig splint may be useful),
5. Irreducible hip,
6. Age >6 months.

Children from 6 to 18 months

The goals of the treatment are to obtain and maintain reduction of the hip without damaging the femoral head. This can be achieved by either closed or open reduction. Closed reduction is the preferred treatment option.

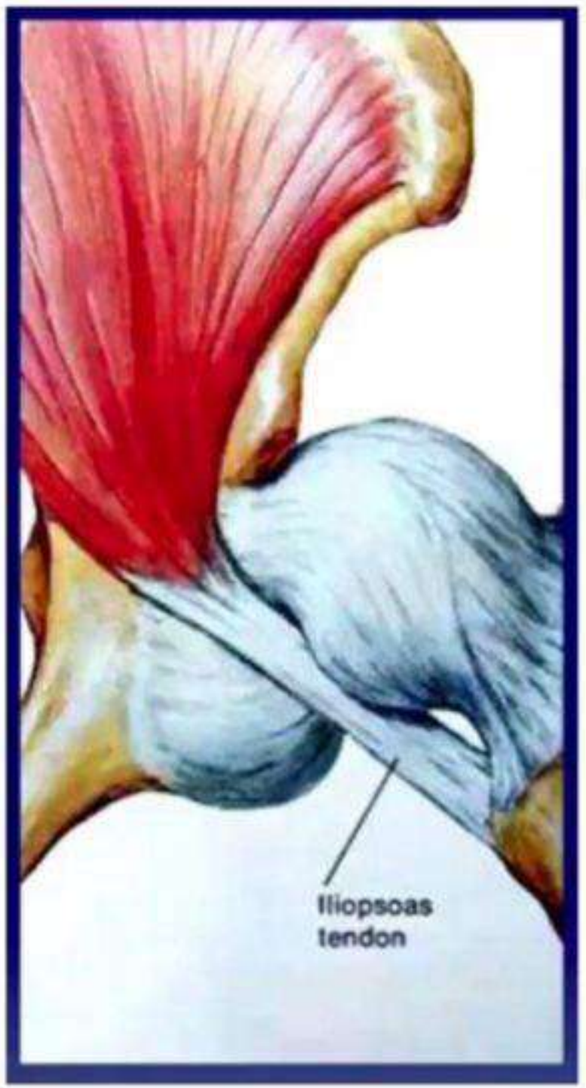
Two controversial issues worth discussing are:

1. A preliminary period of traction before closed reduction,
2. The timing of reduction of the hip in relation to the appearance of the ossific nucleus of the femoral head.

Proponents of preliminary period of traction claim that it reduces the AVN rate and the need of open reduction. To reduce costs, they recommend a portable home traction

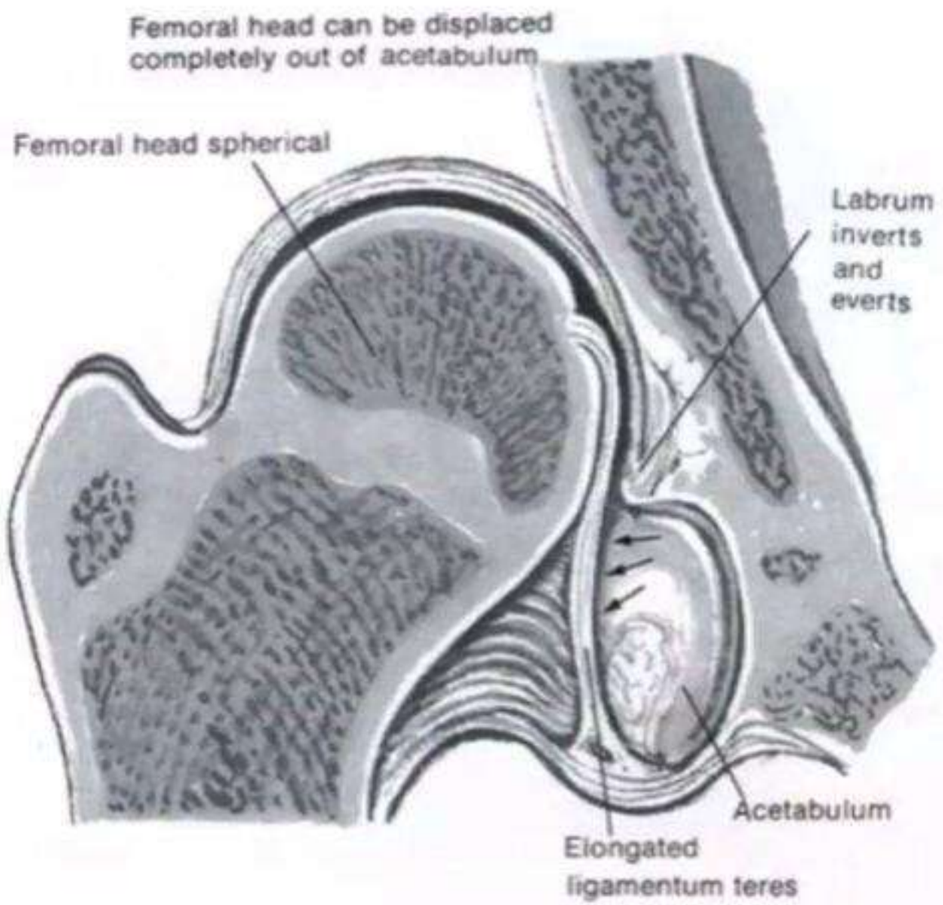
Obstacles for reduction

- Extra articular
  - Contracted add longus, iliopsoas



Obstacles for reduction

- Intra articular
  - Hypertrophied ligamentous teres
  - **Transverse acetabular ligament**
  - **Inferomedial constriction of the capsule**
  - Hypertrophied or inverted labrum ??? Iatrogenic
  - Pullviner tissue ???



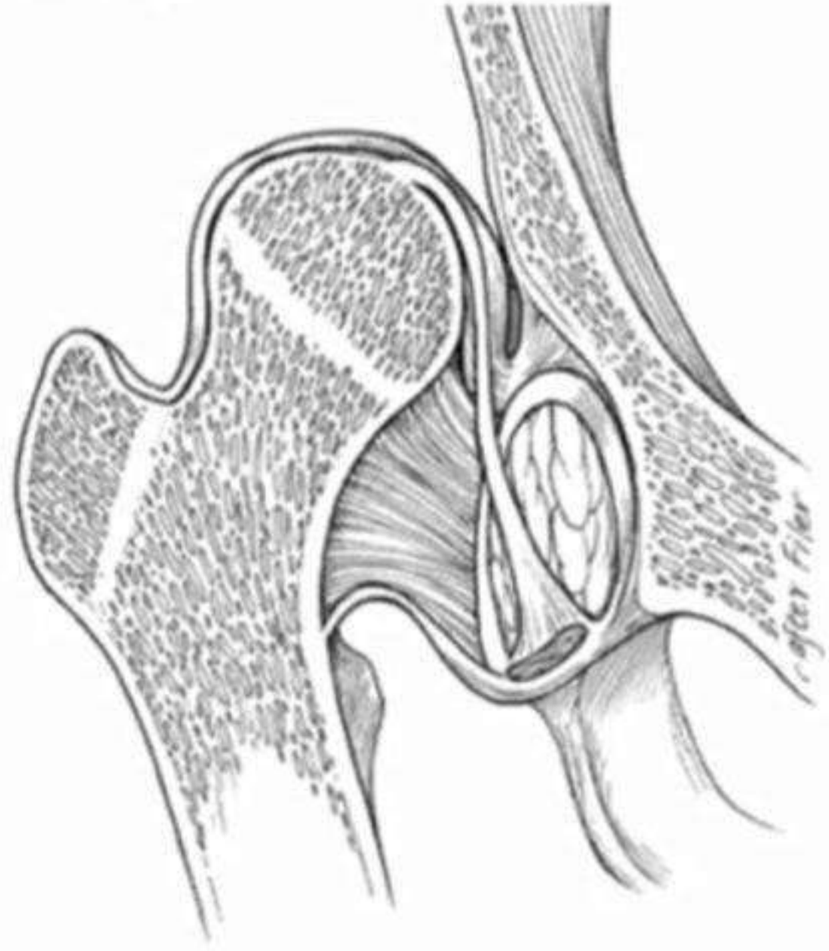
Obstacles to reduction

Extra-articular

- Tight iliopsoas and adductors
- Pre-cephalic insertion of the capsule

Intraarticular

- Ligamentum teres hypertrphied
- Transverse acetabular ligament
- Pulvinar
- Redundant capsule (hourglass)
- inverted limbus



Ant-lat

Lat

Post-Med

Medial

F

L

UVB | Pect | AL / AB | G / AM

CLASSIFICATION

Pelvic Osteotomy

1- Redirection Osteotomies

- Salter
- Steel (triple)
- Ganz

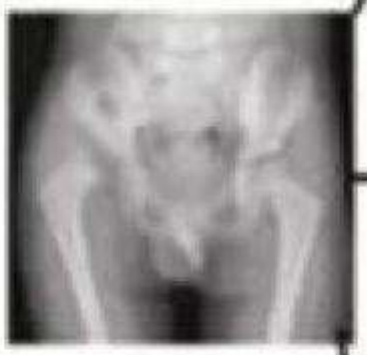
2- Reshaping Osteotomies

- Pemberton
- Dega
- Dail

3- Salvage Osteotomies

- Chiari
- Shelf

Pelvic Osteotomies for DDH



<7 y/o

- Salter (redirection)
- Pemberton (re-shaping, increased anterior coverage)
- Dega osteotomy, San Deigo ostoetomy (increase lateral coverage, re-shaping)

>7 y/o

(Opened triradiate cartilage)

- Dega (increase lateral coverage, re-shaping)
- Triple innominate osteotomy (Steel, Tonnis, etc.)

(Closed triradiate cartilage)
Ganz =Peri acetabulum osteotomy

- Triple innominate osteotomy (Steel, Tonnis, etc.)
- Ganz (Bernese) osteotomy
- Spherical osteotomy

Cannot acheive concentric reduction → Salvage procedure

- Shelf procedure (Staheli)
- Chiari medial displacement osteotomy

Q 8: Why does it happen?

There are several theories to explain the aetiology of SUFE. Some are more convincing than others; but none is perfect. These can be summarized as follows:

- The biomechanical theory: There are several anatomical features that lead to increase the shear forces across the physis and lead to slip:
  1. Increased weight (> 80th centile).
  2. Femoral retroversion (> 10°).
  3. Increased physis height due to widened hypertrophic zone.

SCFE

4. More vertical slope of the physis.
5. Trauma.

- Structural defect of the physis theory:
  1. Hormonal theory: several endocrine disorders have been implicated with causation of SUFE. Bilateral slip is more common with endocrine diseases.
     i. Hypothyroidism.
     ii. Growth hormone deficiency.

iii. Sex hormone (more common in boys 3 : 1. Boys age 12–14, girls age 11–13).

iv. Renal osteodystrophy. It is associated with the highest risk (90%) of bilaterality. By contrast, idiopathic SUFE has a 20% risk of bilaterality initially and a further 10% to 20% risk until maturity. It is also associated with the highest risk (43%) of progressing to grade III slip. The slip goes through the metaphysis rather than the hypertrophic zone of the physis.

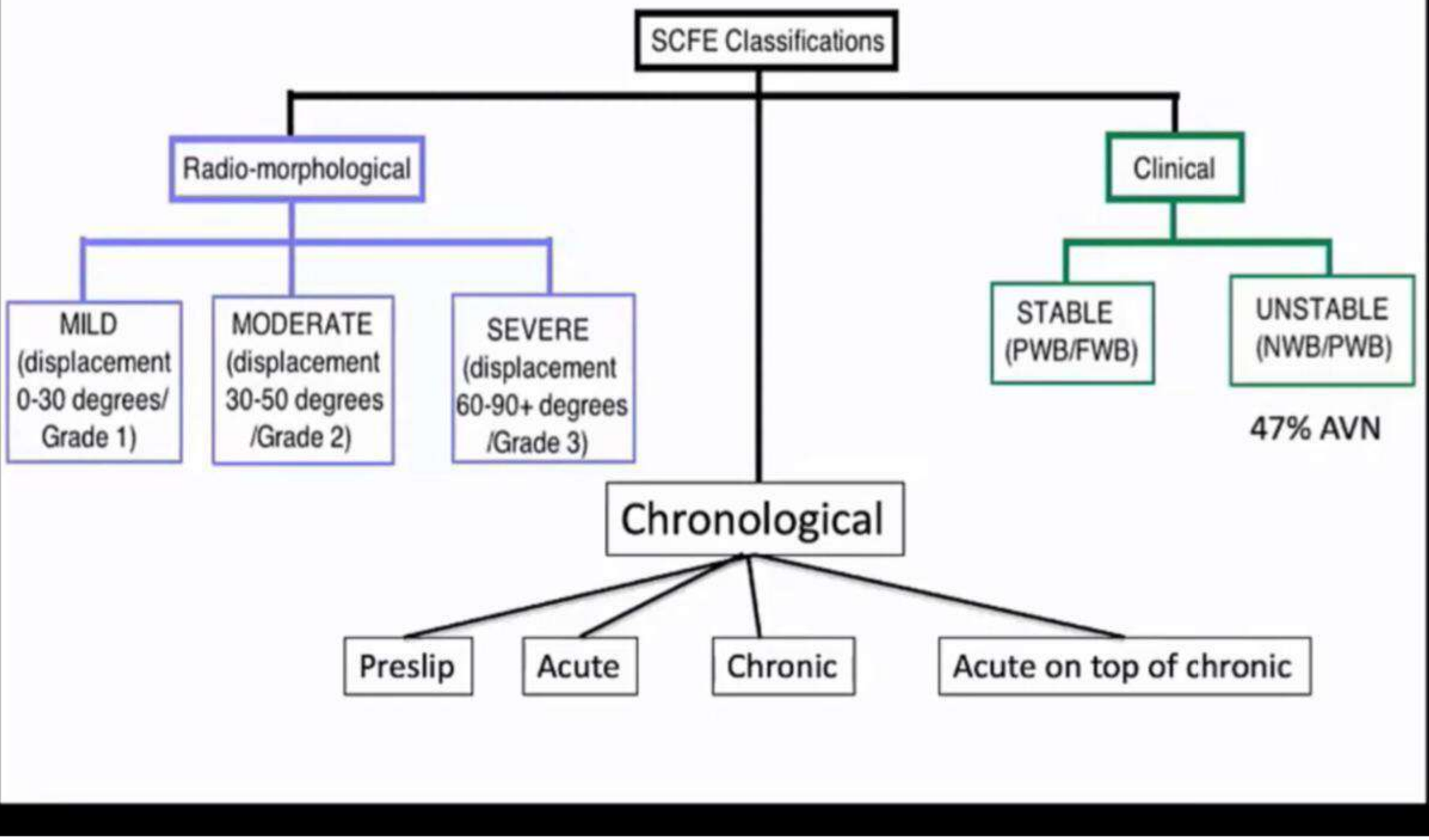
2. Radiotherapy.
3. Racial/ethnic (more common in blacks).
4. Idiopathic.

- Combined: increased sheer forces on abnormal physis.

Q 9: How would you treat this child?

This child has a grade II slip and I would treat him with pinning in situ (PIS). The primary aim of the treatment is to stabilize the slip and prevent further progression until physis closure. There is almost a universal agreement that grade I and grade II slips

Classification



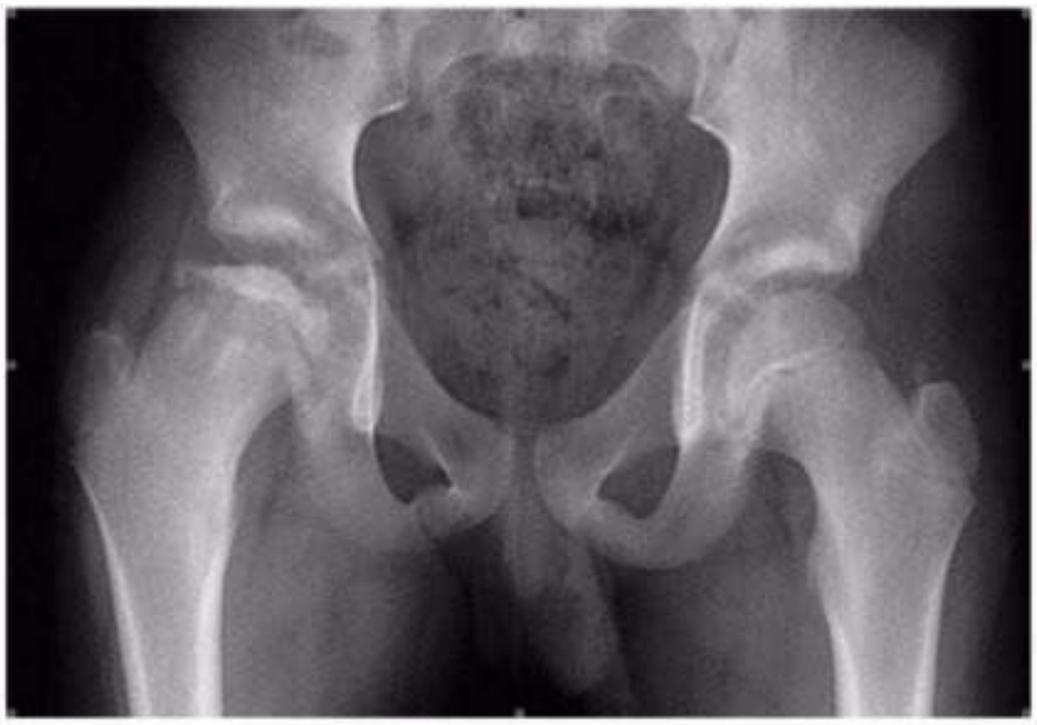


Figure 12.8 Pelvis X-ray of a child with right hip pain and limping.

Q 17: This is a pelvic X-ray of a 9-year-old boy who has been limping over the last 3 months. What can you see? (Figure 12.8.)

This is an AP view of the pelvis in a skeletally immature patient. The most obvious feature is the collapse of the right femoral head with increased bone density. There is a widening of the joint space on the right side. At the top of my list is Legg–Calvé–Perthes disease (LCPD), but other diagnoses such as infection need to be excluded.

Q 18: What is Perthes' disease?

It is a non-inflammatory AVN of the femoral head in a growing child caused by interruption of blood supply. The condition was first described by Waldenstorm, but he attributed it to tuberculosis; it was then described more accurately by Arthur Legg (1874–1939), Jacques Calvé (1875–1854) and George Perthes (1869–1927) almost at the same time; hence the name Legg–Calvé–Perthes disease (LCPD).

It is not common (1\10 000), affecting children between 4–9 years old of low socioeconomic class. Child is often small with delayed bone age by usually 2 years. It is bilateral in 15% of cases but involvement is usually asymmetrical and never simultaneous (in contrast to multiple epiphyseal dysplasia). There may be a family history.

Q 19: What does it happen?

The aetiology is unknown; however, several theories have been put forward to explain it:

1. The anatomical theory.

The blood supply to the femoral head changes as the child grows (Table 12.1). The change over to adult pattern may be affected comprising the blood supply to the femoral head leading to ischaemic necrosis.

Table 12.1 Blood supply of the femoral head.

Age	Birth–4 years	4 years–adult	Adult
Source	Medial and lateral circumflex arteries from profunda femoris artery Ligamentum teres with posterior division of obturator artery	Posterosuperior and posteroinferior retinacular from medial femoral circumflex artery Negligible lateral circumflex artery Minimum ligamentum teres	Medial femoral circumflex to lateral epiphyseal artery

2. Hydrostatic pressure theory.

This theory attributes the reduction in blood supply to the femoral head to the increase in the intraosseous venous pressure which has been noticed in several cases.

3. Thrombophillic theory.

There is evidence of association of LCPD with various forms of thrombophilia. In one study, 72 patients with LCPD were compared with 197 matched healthy controls.[5] The factor-V Leiden mutation was more common in LCPD (8/72) than in the controls (7/197) (chi-square = 5.7, p = 0.017). A high level of anticardiolipin antibodies was found in 19 of the 72 LCPD compared with 22 of the 197 controls (chi-square = 9.5, p = 0.002). Other studies showed association of LCPD with protein S and C abnormalities.[6,7] It is important to remember that association does not always mean causation.

Q 20: What is your differential diagnosis?

In this scenario where the right side only is involved, my differential diagnoses are:

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.

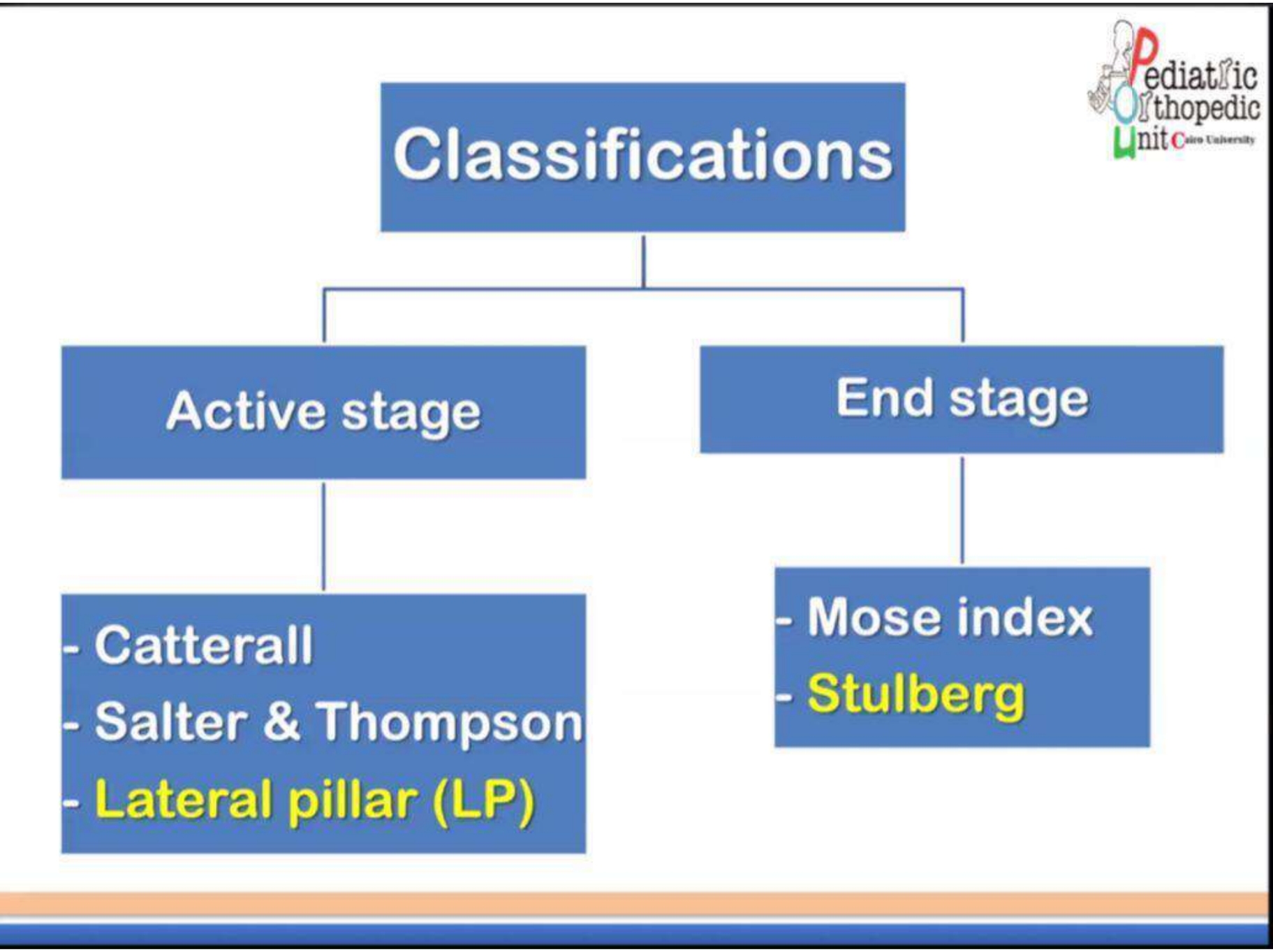

Classifications
Active stage
End stage
- Catterall
- Salter & Thompson
- Lateral pillar (LP)
- Mose index
- Stulberg

Table 3.2.6 LCPD poor prognostic signs

Poor clinical prognostic signs (FOOBS)	Poor radiological prognostic signs
Female	Gage's sign (V shape lucency at lateral epiphysis (Figure 3.2.5)
Older age	Horizontal growth plate – implies a growth arrest phenomenon and deformity
Obesity	Lateral calcification (lateral to the epiphysis – implies loss of lateral support and head extrusion)
Bilateral	Lateral subluxation – implies loss of lateral support Uncovering of the femoral head >3 mm in excess of opposite side (measured as the horizontal distance between a vertical line through the outer lip of acetabulum and lateral edge of femoral head physis) or >20% of extrusion
Stiffness	Metaphyseal rarefaction or cyst

Table 3.2.5 Stulberg grading of LCPD (see also Figure 3.2.4)

Head	Class	Description	Risk of future OA
Spherical and congruent	I	Normal spherical head	No increased risk of arthritis
	II	Spherical head, coxa magna/breva, steep acetabulum	
Aspherical but congruent	III	Ovoid or mushroom-shaped head	Mild to moderate arthritis develops in late adulthood
	IV	Flat head on flat acetabulum (may hinge on abduction)	
Aspherical and incongruent	V	Flat head but normal acetabulum	Severe arthritis before 50

Class	Description	Features	Prognosis
I	Spherical congruency	Completely normal hip joint	Good
II	Spherical congruency; less than 2 mm loss of head shape	Spherical femoral head with a concentric circle on anteroposterior and frog-lateral radiographs, with 1 or more of the following abnormalities: • Coxa magna • Short femoral neck • Abnormally steep acetabulum	Good
III	Aspherical congruency; greater than 2 mm loss of head shape	Non-spherical but not a flat femoral head. Ovoid, mushroom-shaped head with 1 or more of abnormalities: • Coxa magna • Short femoral neck • Abnormally steep acetabulum	Mild-to-moderate arthritis
IV	Aspherical congruency	Flat femoral head with abnormal femoral head, neck and acetabulum.	Mild-to-moderate arthritis
V	Aspherical incongruency	Flat femoral head with a normal-shaped femoral neck and acetabulum.	Severe early arthritis

Table 4.1 Causes of genu varus (bow leg) and genu valgus (knock knees)

Bow leg	Knock knees
1. Physiological	1. Physiological
2. Tumours such as osteochondroma	2. Tumours such as osteochondromas
3. Skeletal dysplasia	3. Skeletal dysplasia
4. Blount's disease	4. Primary tibia valga
5. Infection	5. Infection
6. Trauma	6. Trauma
7. Metabolic (vitamin D deficiency, fluoride poisoning, osteogenesis imperfecta)	7. Renal osteodystrophy
8. Focal fibrocartilaginous dysplasia	8. Neuromuscular disorders (polio) and tight iliotibial band

Table 4.3 Langenskiold classification of Blount's disease

Stage	Description	Treatment
I	Medial beaking, irregular medial ossification with protrusion of the metaphysis	Orthotic for <3 years old
II	Cartilage fills depression Progressive depression of medial epiphysis with the epiphysis sloping medially as disease progresses	Failure of full correction or progression to Type III → surgery
III	Ossification of the inferomedial corner of the epiphysis	Surgery around the age of 4
IV	Epiphyseal ossification filling the metaphyseal depression	
V	Double epiphyseal plate (cleft separating two epiphysis)	
VI	Medial physeal closure	

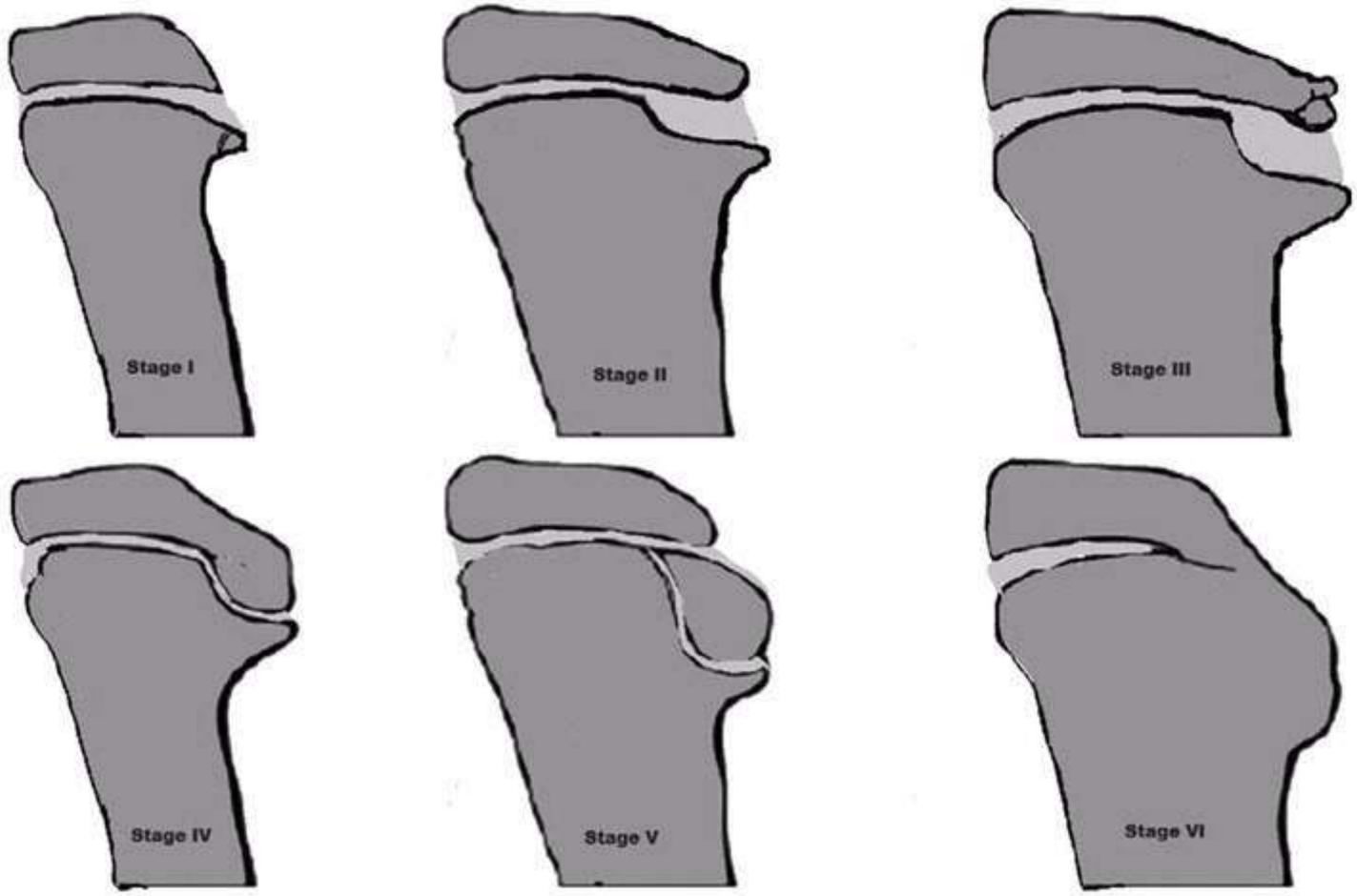
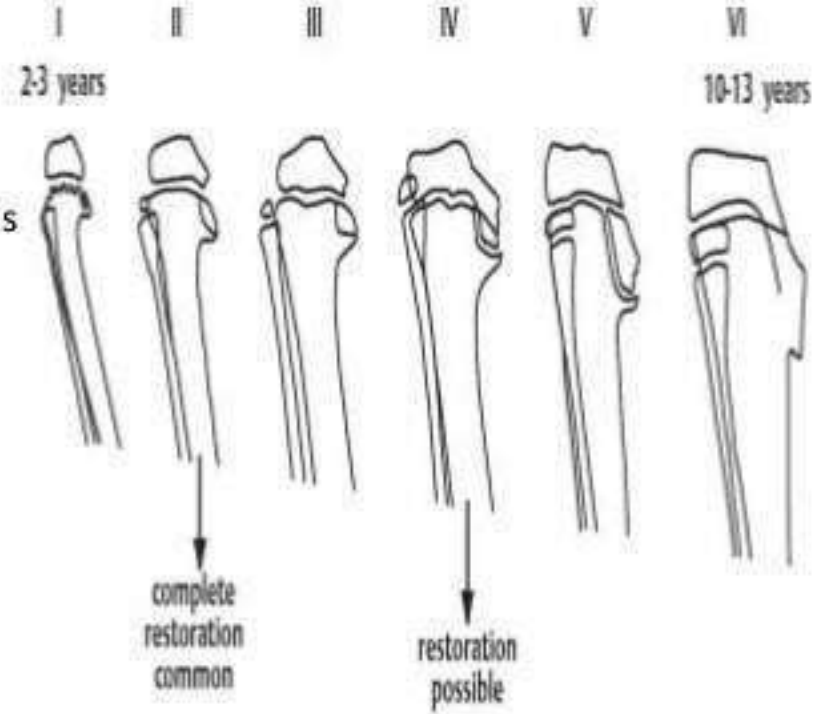


Figure 4.6 Langenskiold classification of Blount's disease.

classification

- Stage I
  - medial metaphyseal beaking
- Stage II
  - Saucer-shaped defect of medial metaphysis
- Stage III
  - Saucer deepens into step
- Stage IV
  - Sloping of epiphysis over medial beak
- Stage V
  - Double epiphysis
- Stage VI
  - Medial physeal bony bar



CANDIDATE: The risk factors for patellar instability are:

1. Bony factors (static)

   Trochlear dysplasia.

   Hypoplastic femoral condyle.

   Patellar shape.

   Patella alta.

2. Malalignment

   Patellar malalignment is an abnormal rotational or translational deviation of the patella along any axis.

   External tibial torsion/foot pronation.

   Increased femoral anteversion and increased genu valgum.

   Increased Q angle or abnormal tibial tuberosity–trochlear groove (TT–TG) distance.

3. Soft tissue (dynamic)

   Ligamentous laxity (medial patellofemoral ligament rupture/ insufficiency).

4. Abnormal gait

   Walking with valgus thrust.

5. Genetic factors such as connective tissue disorder syndromes.

Risk factors include:

1. Female,
2. Q angle $> 20°$ (normal is 10° in boys and 15° in girls),
3. Genu valgus,
4. Rotational abnormalities, such as increased femoral anteversion or external tibial torsion,
5. Patella alta (Figure 4.17),
6. A shallow patella-femoral sulcus angle (ABC $> 144°$ is abnormal) (Figure 4.17),
7. Abnormal congruence angle of Merchant (OBX is normally $-6°$ to $-8°$). This angle is abnormal if it is more than +16°. Positive (+) means lateral while negative (−) means medial (Figure 4.17),
8. Vastus medialis obliquus hypoplasia,
9. Generalized ligamentous laxity,
10. Pes planus,
11. Lateral mobility greater than 3/4. (Medial mobility less than 1/4 indicates a tight lateral reticulum.)

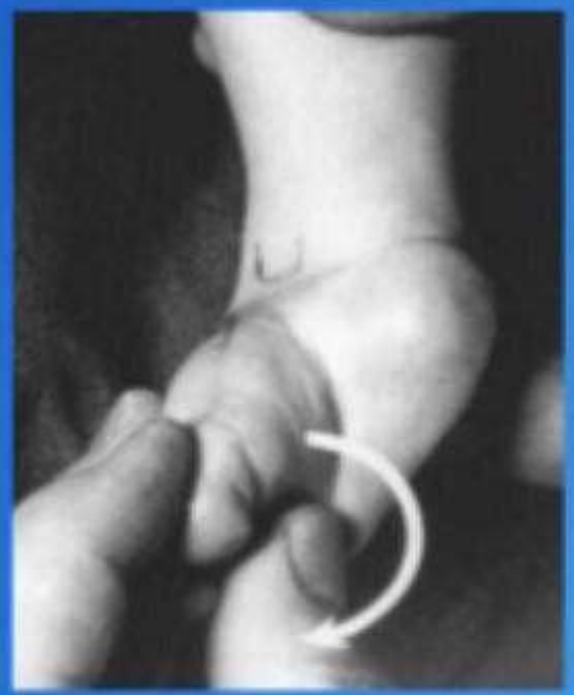
- Neuromuscular disorders
  - Muscular dystrophy
  - Charcot Marie Tooth disease
  - Friedriech's ataxia
  - Roussy-Levy syndrome
- Central nervous system disorders
  - Cerebral palsy
  - Poliomyelitis
- Spinal pathology
  - Spinal dysraphism
  - Diastematomyelia
  - Spina bifida
  - Myelomeningocele
  - Syringomyelia
  - Spinal cord tumour
- Muscle pathology
  - Crush injury
  - Burns
  - Compartment syndrome

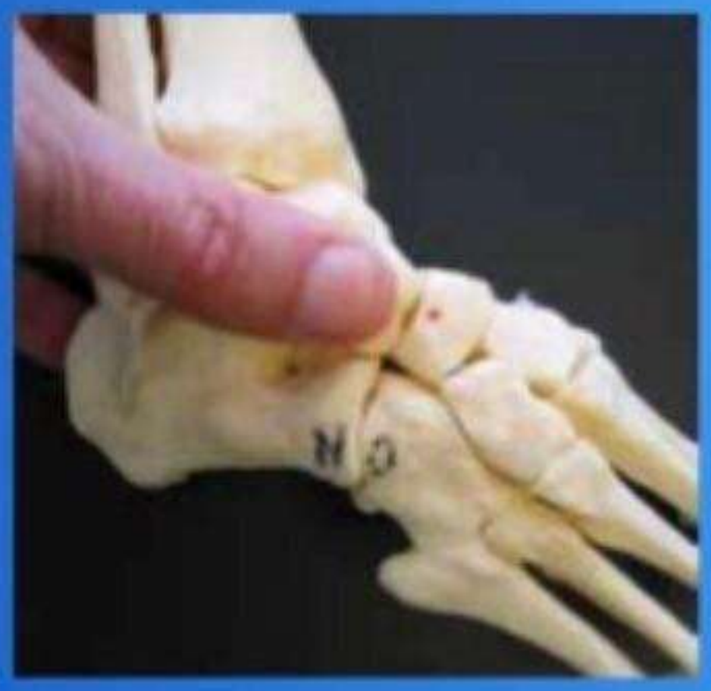
Definition

- Contractural malalignment of the bones and joints of the foot and ankle.

- Congenital dysplasia of all musculoskeletal tissues (musculotendinous, ligamentous, osteoarticular, and neurovascular structures) distal to the knee.

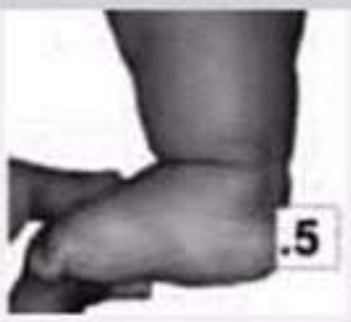
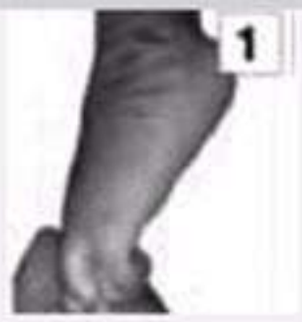
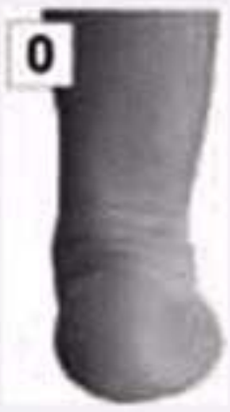
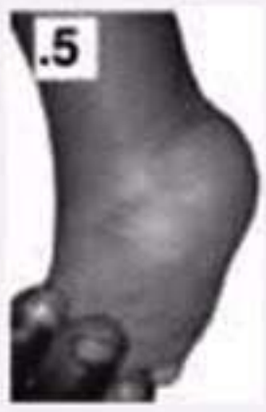
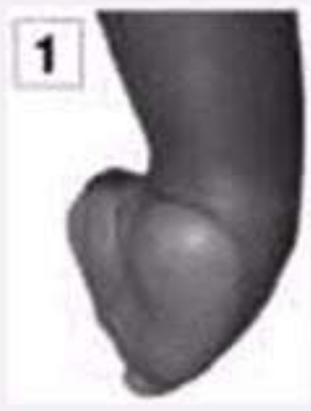
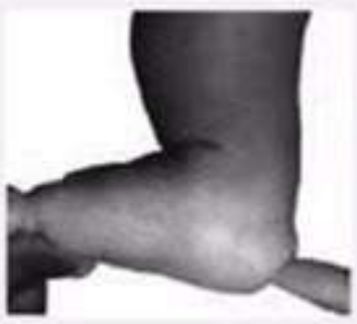
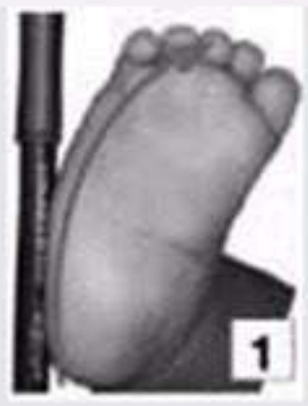
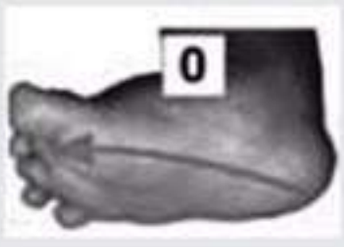
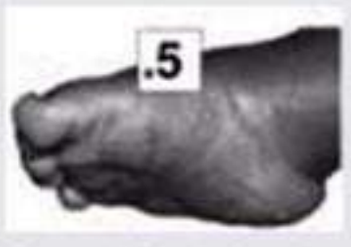
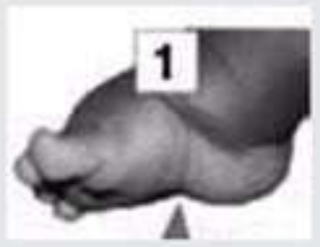
Common errors(Kite errors)





- Pronation/eversion of 1st metatarsal
- Premature dorsiflexion of heel
- Counterpressure at calcaneocuboid joint
- External rotation
- Below knee casts
- Short splints

Table 5.1 Pirani score for club feet (pictures courtesy of Dr Lynn Staheli and Global Help Publication): total Pirani score, 0–6

	Clinical sign	0	0.5	1.0
Hindfoot contracture score (0–3)	Equinus	 Dorsiflexion	 Comes to neutral	 Cannot reach neutral
	Deep posterior crease	 Multiple fine creases (normal)	 Superficial single crease, which is obliterated on dorsiflexion	 Deep persistent crease
	Empty heel	 Easily palpable calcaneum	Deep calcaneum	Not palpable
Midfoot contracture score (0–3)	Curved lateral border	 Straight	 Mildly curved	 Severely curved
	Medial crease	 Multiple fine creases (normal)	 Superficial single crease, which is obliterated on dorsiflexion	 Deep persistent crease
	Lateral head of talus	 Fully covered with the navicular bone	 Partially covered	Not covered at all

CONGENITAL DEFORMITIES OF THE UPPER LIMB

- 1 in 600 children born with a congenital upper limb deformity
- **Swanson's Classification**
  - Failure of formation
    - Transverse: Phocomelia (Patient can have remnants of digits)
    - Longitudinal: Radius, Ulna, Fibula, Tibia hemimelia, Cleft hand, longitudinal tibia deficiency, PFFD
  - Failure of differentiation: Syndactyly, Clinodactyly, Arthrogryposis
  - Duplication - Polydactyly
  - Overgrowth – Macrodactyly, Congenital hemi-hypertrophy
  - Undergrowth - Thumb hypoplasia
  - Constricting band syndrome
  - Complex – Intercalary

Type	Radiological Description	No. of limbs
1a	• Tibia not seen • Hypoplastic lower femoral epiphysis	6
1b	• Tibia not seen • Normal lower femoral epiphysis	12
2	• Distal tibia not seen	5
3	• Proximal tibia not seen	2
4	• Diastasis	4

Fig. 1

Congenital aplasia of the tibia. The four types of deformity as shown radiologically

B.Viva : Long & Short

Section 3 Exam-related material

Chapter 19

Viva and clinical practice

Mohamed O. Kenawey and Paul A. Banaszkiewicz

Topic 1: Club foot

Viva practice 1

Congenital talipes equinovarus (CTEV), or club foot, is a fairly common topic for the viva. There is more than enough material to cover in a 5-minute slot with room to spare. The treatment method has significantly changed in the last 10 years with the introduction of the Ponseti technique. This is something that you must be familiar with and in an ideal world have seen performed. That way you should be able to confidently describe the technique to the examiners, leaving them with the impression that you have actually seen the procedure being done.

In the clinical examination you might be shown a case of old club foot with residue deformities in a young adult. This can be challenging, as you will have to piece together what surgery has been performed previously and what further surgical options are available.

Examiner: You have been called to the paediatric ward because the doctors are unhappy with the appearance of this baby's right foot. What do you see? What is the diagnosis?

Candidate: These are clinical photographs of a newborn child, showing a deformed right foot: the heel is turned in (varus), the forefoot is deviated toward the midline (adductus), and there are posterior and medial creases. Although it is not very clear from these pictures, the ankle appears to be in equinus with the first metatarsal pointing downward (plantar flexion) [1]. The diagnosis is suggestive of congenital talipes equinovarus (club foot).

Examiner: What else could it be?

Candidate: Occasionally, a severe metatarsus varus can be confused with club foot. The heel is in a neutral position (unlike club foot) and there is no equinus. Positional calcaneovalgus in which there is dorsiflexion of the whole foot such that it may touch the tibia can sometimes be mistaken for a club foot deformity. (See Chapter 5 for more detail.)

Examiner: What are the deformities in this condition?

Candidate: The deformities in this condition can be remembered by the mnemonic CAVE: cavus (midfoot), abductus (forefoot), varus (hindfoot) and equinus (hindfoot). From proximal to distal, there is usually wasting of the calf muscles; the hindfoot is in equinus and varus, the talar head points laterally and downward (causing a bump on the outside of the foot, which is visible in the photograph). Although X-rays are not necessary in managing club feet, they would show that the talus and the calcaneum are parallel instead of at

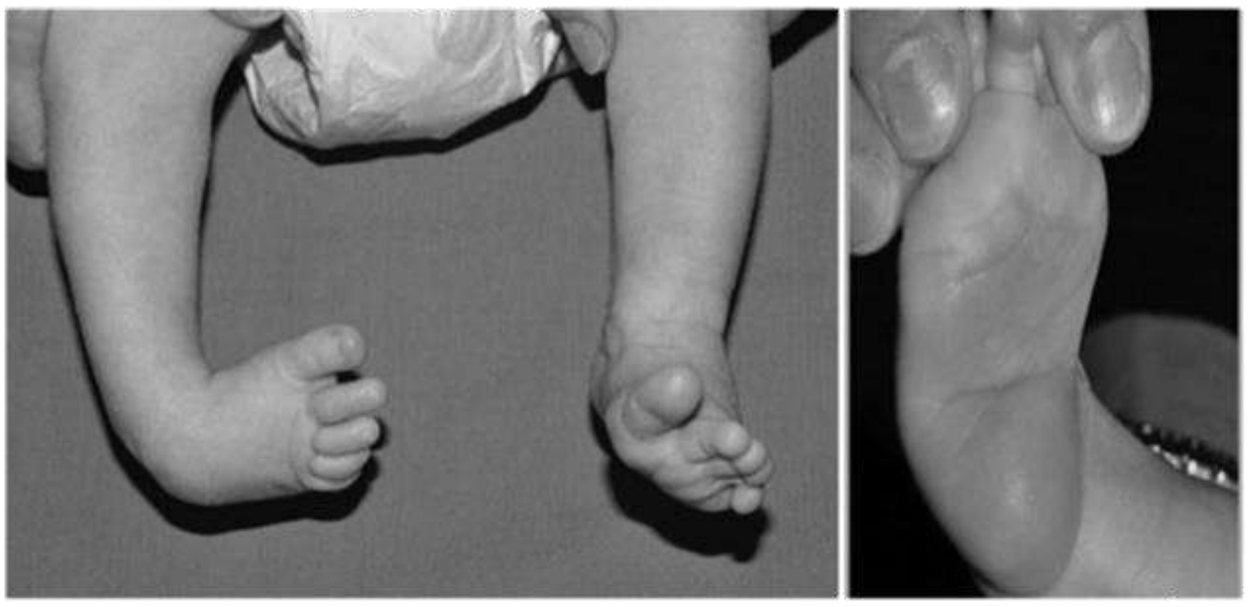


Figure 19.1 Club foot.

Postgraduate Paediatric Orthopaedics, ed. Sattar Alshryda, Stan Jones and Paul A. Banaszkiewicz. Published by Cambridge University Press.

Chapter 19: Viva and clinical practice

Table 19.1 Diméglio scoring system

Rating	4	3	2	1	0	Scores
Equinus	45–90° Plantar flexion	20°–45° Plantar flexion	20°–0° Plantar flexion	0°–20° Dorsiflexion	>+20° Dorsiflexion	
Varus	45–90 Varus	20°–45° Varus	20°–0° Varus	0°–20° Valgus	>20° Valgus	
Supination	45–90 Supination	20°–45 Supination	20°–0° Supination	0°–20° Supination	>–20° Supination	
Adductus	45–90° Adduction	20°–45° Adduction	20°–0° Adduction	0°–20° Abduction	>20° Abduction	
Posterior crease				Yes	No	
Medial crease				Yes	No	
Cavus				Yes	No	
Deviant muscle function				Yes	No	
					Total	

an angle (the talocalcaneal angle of Kite is 20°–40°). The midfoot is deviated toward the midline (adductus), and the first metatarsal points downward (plantar flexion).

Examiner: How are you going to treat this patient?
Candidate: I would take a full history and examination. I would want to know if there has been a family history of club foot. There is a genetic component but not a recognizable pattern of inheritance. If one child has club foot, the risk of club foot in a subsequent child is increased 20-fold. With examination I would want to exclude any associated conditions, such as spina bifida (4.4% of children with club foot), cerebral palsy (1.9%) or arthrogryposis (0.9%). I would want to access the severity of the condition. Deep creases behind the heel or on the medial side of the foot tend to be associated with a more severe condition.

Examiner: How are the deformities usually corrected, and in what order?
Candidate: The disorder is normally corrected by the Ponseti method of cast manipulation using the head of the talus as a fulcrum. Cavus is corrected first by dorsiflexing the first ray and unlocking the forefoot and midfoot. With the cavus corrected, the forefoot is abducted and the heel goes into valgus by the coupling on the subtalar joint. Finally, the equinus is corrected. Serial above-knee casts (with the knee at 90°) are changed weekly with the old cast removed, the deformity scored and the new cast applied. Residual equinus (or less than 20° of dorsiflexion) requires Achilles tendon release in the majority of patients. This can be performed under local or general anaesthetic and a final cast is applied for a further 3 weeks while the tenotomy heals.

Examiner: What do we want to achieve?
Candidate: The aim of treatment is to achieve a painless, plantigrade foot with good mobility, with no need for special or modified shoes.

Examiner: What should we tell the parents?
Candidate: Club foot is a common problem and affects about 1 in 1000 babies. In most cases, there are no obvious causes although it can run in families. Treatment has changed dramatically in the last 10 years and instead of extensive surgical correction, most babies are treated with serial casting and bracing.

Examiner: How do you access the severity of foot deformity?
Candidate: Two grading systems are used: the Pirani score (see Chapter 5 for more detail) and the Diméglio score. I am familiar with the Diméglio score. It consists of eight items. Scorings for four of the items range from 0–4 (best to worst). The other four items only score 0 or 1. The total score ranges between 0 and 20: very severe, 16–20; severe, 11–15; moderate, 6–10, and postural 0–5) (Table 19.1).

Examiner: The parents want to know if everything will be fine.
Candidate: I would tell the parents that treatment with the Ponseti technique for an idiopathic club foot deformity usually has a good result; 90% of children will have painless feet with good functions. However, the foot and the calf will not be as normal as the other side. The affected foot and calf are usually smaller in size and the child may need different sized shoes. Extensive surgery is rarely needed, but minor surgery is often required (tendo Achillis tenotomy in 85% of patients and tibialis anterior transfer in about 15%). The child should wear Denis Browne boots with a bar on a full-time basis for 3 months after finishing serial casting, then at night and nap times for 3–4 years. This holds the affected foot externally rotated at around 70°. Compliance and tolerance of treatment is essential for success and avoidance of recurrence. Most children tolerate treatment very well and avoid the need for extensive surgery. Recurrence or persistence of a small degree of the deformity is not

uncommon; most children can be treated with a repeat of serial casting; however, some may need further surgery.

Examiner: What are the published results for the Ponseti method?
Candidate: Jowett *et al.*, in a recent systematic review, found that the Ponseti method provides excellent results, with an initial correction rate of around 90% in idiopathic feet. Non-compliance with bracing is the most common cause of relapse; this occurs in about 15% of patients [2].

Examiner: What are the complications of the Ponseti technique?
Candidate: Complications include recurrence of deformity, pressure damage due to casting, neurovascular injury with Achilles tendon release and overcorrection of deformity.

Clinical practice 1

This is a potentially difficult short case; possibly an intermediate case in an adult, especially if there is co-existing LLD.

The child is likely to be about 5 years old. In a paper by Lampasi *et al.* [3], the mean age for revision surgery was 4.8 years (2 to 10.1). As the Ponseti method continues to gain acceptance across the world, relapsing deformity after initial complete correction is becoming more prevalent. This could also be an adult intermediate or short clinical case with post club foot syndrome. There would be a hypoplastic limb with a short leg gait, possibly several scars in the foot from previous

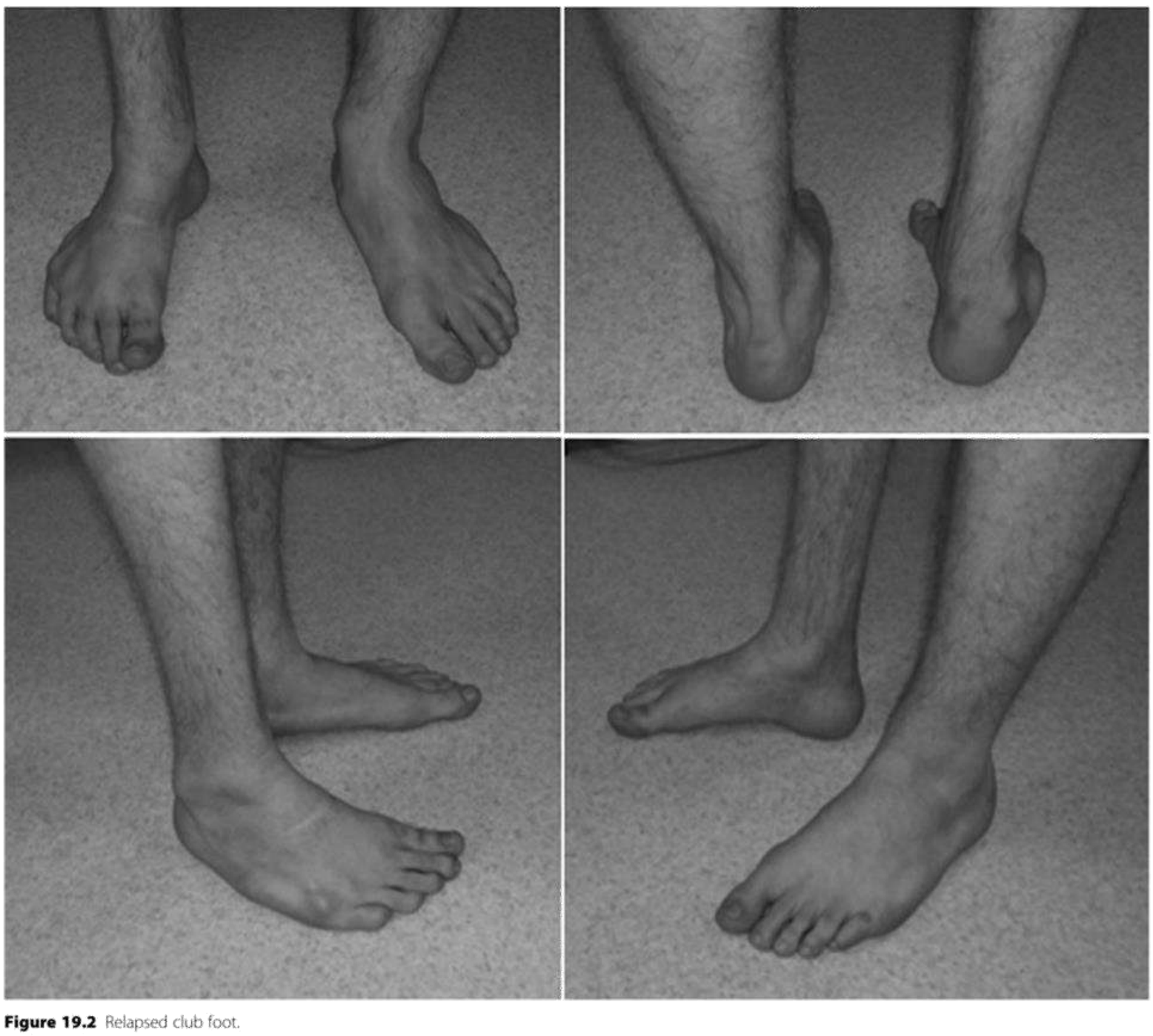


Figure 19.2 Relapsed club foot.

surgery and an LLD with shortening present in both the tibia and femur.

History

- Age at initial presentation,
- Treatment before relapse,
- Number of casts and tenotomies,
- Length of brace wear,
- Shoe wear.

Clinical examination

The appearance of the feet at follow-up can be classified according to Garceau and Palmer [4]. The paper is old – it was published in 1967 – but it does provide a framework that you can use. Not all components of the club foot tend to relapse to the same degree.

The relapse of the cavus deformity is rare and usually mild. The most important relapses occur in the hindfoot, first in the equinus, and then in the heel varus. In some relapsed club feet, the heel varus is very severe, while in others it is mild. Rarely, the heel in equinus may go into valgus, resulting in a calcaneovalgus deformity. This is a frequent occurrence in surgically treated club feet and is often referred to as over-corrected club foot. The following framework is useful:

- Describe the residue deformity to the examiner and in particular look for residual metatarsus adductus, heel varus and equinus. You can use Pirani or Diméglio scores to structure your description. Is it a correctable deformity?
- The affected foot may have several scars from previous surgery.
- Tendons: the strength of the peroneal and tibialis anterior tendons needs to be assessed. The direction of pull is important. If the tibialis anterior tendon pulls the foot into dorsiflexion and supination, it may need to be transferred to the midfoot.
- Check for LLD on the affected side (if unilateral).
- There have been several complicated residue club foot cases where epiphyseodesis of the normal contralateral femur and tibia at the knee has been performed.
- Gait:
  - No visible gait deformity,
  - Simultaneous heel-toe strike or mild genu recurvatum (due to equinus deformity),
  - Moderate genu recurvatum or lateral instability of the ankle,
  - Marked limp, failure of the heel to touch the floor, severe recurvatum or pivoting of the foot during step off.

Investigations

Weight-bearing AP, lateral and Saltzman's views of the foot (see Chapter 5 for more detail).

Management

The surgical technique for relapsed congenital club feet can be divided into three broad categories: namely soft-tissue releases, bony procedures and tendon transfers.

Surgical options for the correction of relapsed club foot could include:

- Medial release (almost every medial structure can be released or lengthened but avoid damaging the deltoid ligament),
- Posterior release (Achilles tendon and ankle posterior capsule),
- Tendon transfer,
- Bony procedures to correct alignment:
  - Calcaneum slide (lateral to correct varus, or medial to correct excessive valgus).
  - Cuboid osteotomy (to swing the forefoot around the talonavicular joint): a closing wedge corrects forefoot

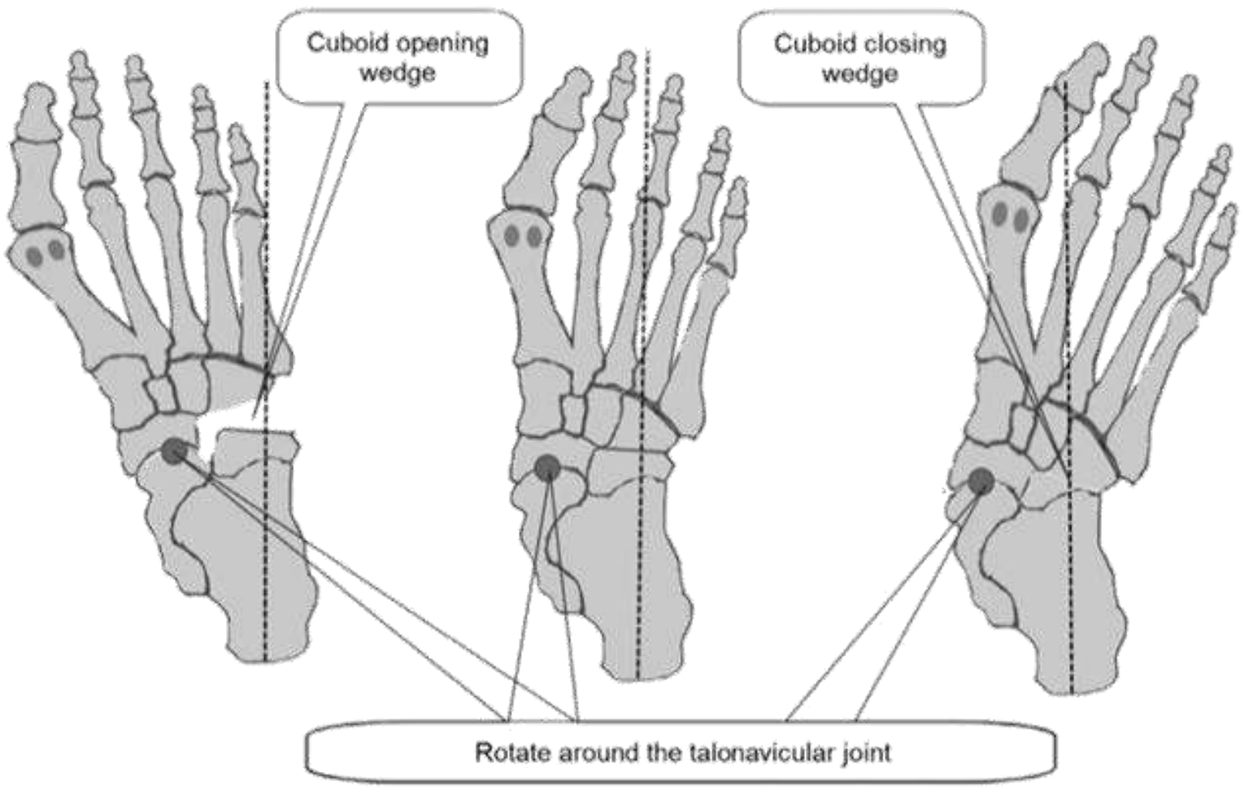
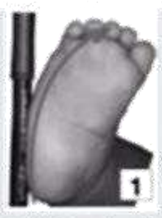


Figure 19.3 Cuboid osteotomy.

adduction, while an opening wedge corrects forefoot abduction.
  - Dorsal closing wedge of the first metatarsal to elevate the first ray.
- Triple arthrodesis (or selective fusion) as a salvage for advanced and uncorrectable deformity.

The one thing to avoid with relapsed club foot surgery is overcorrection. Whereas a small degree of undercorrection is often well tolerated in adulthood, overcorrection is less acceptable and difficult to treat.

Table 5.1 Pirani score for club feet (pictures courtesy of Dr Lynn Staheli and Global Help Publication); total Pirani score, 0–6

	Clinical sign	0	0.5	1.0
Hindfoot contracture score (0–3)	Equinus	 Dorsiflexion	 Comes to neutral	 Cannot reach neutral
	Deep posterior crease	 Multiple fine creases (normal)	 Superficial single crease, which is obliterated on dorsiflexion	 Deep persistent crease
	Empty heel	 Easily palpable calcaneum	Deep calcaneum	Not palpable
Midfoot contracture score (0–3)	Curved lateral border	 Straight	 Mildly curved	 Severely curved
	Medial crease	 Multiple fine creases (normal)	 Superficial single crease, which is obliterated on dorsiflexion	 Deep persistent crease
	Lateral head of talus	 Fully covered with the navicular bone	 Partially covered	Not covered at all

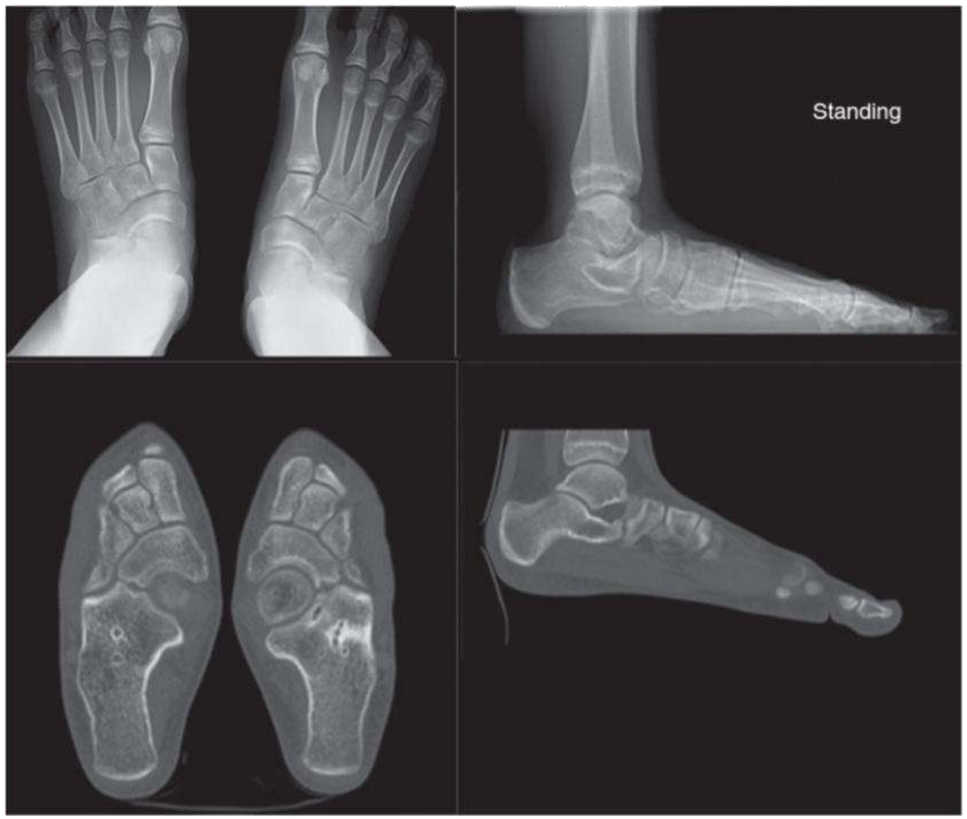


Figure 19.10 Child with bilateral foot pain.

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Topic 4: Rigid flat foot

Viva practice 4

Examiner: These are the radiographs of a 9-year-old boy who came to the clinic complaining of foot pain.
Candidate: These are plain AP and lateral radiographs of the feet and coronal and sagittal CT scans. They demonstrate a calcaneonavicular coalition. This is the most common tarsal coalition. It occurs in approximately two-thirds of cases. There is usually an elongated anterior process of the calcaneum (the anteater nose sign).

Examiner: What do you mean by a coalition?
Candidate: A coalition is a fusion between tarsal bones caused by an embryological failure of segmentation of mesenchymal tissue in the hind and midfoot. The fusion could be fibrous, cartilaginous or osseous. A solitary coalition can range from a minimal fibrosis to complete bony synostosis.

Examiner: How do patients present?
Candidate: A large number of coalitions can be asymptomatic and never present to an orthopaedic surgeon. Symptoms usually appear by the age of 10–12 years, when the coalition ossifies or becomes stiffer. These include calf pain due to peroneal spasticity, flatfoot and limited subtalar motion.
A child with a stiff hind foot may give a history of recurrent ankle sprains or fractures. Lateral heel pain is a common finding from fibular impingement on the valgus calcaneum. Subtalar movement is grossly reduced or absent.

Examiner: What investigations would you order?
Candidate: Computed tomography (CT) and magnetic resonance imaging (MRI) are generally not necessary to make the diagnosis of calcaneonavicular coalition. Most surgeons would prefer to order these image modalities to identify multiple coalitions and to assess coalition location and percentage of joint involvement. A talocalcaneal coalition can be difficult to diagnose on plain radiographs.

Examiner: What are the aims of management?
Candidate: These would be relief of pain, improvement of joint motion, correction of deformity and avoidance of degenerative joint disease.

Examiner: So what would you offer this patient?
Candidate: My initial treatment is to control symptoms using pain killers, shoe modifications and maybe a short period of immobilization (casting) or orthoses.
Persistence of symptoms is an indication for resection. The extensor digitorum brevis muscle is interposed between the resected bar to prevent recurrence. Raw bone areas are also covered with bone wax to further reduce the risk of recurrence.

Examiner: How successful is resection?
Candidate: This depends on the size of the coalition and whether degenerative joint disease is present.

Examiner: So what figure would you tell the patient?
Candidate: Cohen reported a series of 17 adult patients with symptomatic tarsal coalition in which three patients improved with conservative measures. Twelve patients had symptoms severe enough to require surgery.
Of these patients, 10 of the 12 obtained pain relief at 3-year follow-up [7].

Examiner: Are any other options available?
Candidate: A subtalar arthrodesis or triple arthrodesis is usually reserved for failed resections with persistent pain and deformity.

Clinical practice 2

A GP letter refers an 8-year-old boy to the orthopaedic clinic; there is several months' history of left foot pain.
Examiner: Would you examine this young man's left foot please?
Candidate: On inspection, I note a planovalgus attitude of the left foot. The forefoot is abducted relative to the hindfoot. From behind, the lateral toes are more visible but it is not a too-many-toes sign, as more than two toes should be seen outside the heel for this sign to be positive.

Can you stand up on your tiptoe for me please?

The patient is unable to perform a double heel raise. The medial longitudinal arch does not form and is absent. There is failure to reconstitute the medial longitudinal arch when the patient attempts to adopt a weight-bearing equinus position. The heels fail to go into varus. The feet appear to be as a rigid flat foot. I am thinking about tarsal coalition as a diagnosis.

Examiner: What test would you like to perform to confirm your diagnosis?
Candidate: I would like to perform the Coleman block test.

> I have no idea why I said this. This was the wrong test. The examiner quickly moved me onto a new case. I still, however, passed that particular short case.
>
> I cannot remember if I asked the patient whether the foot was painful or not.

This condition is sometimes referred to as peroneal spastic flatfoot but the peroneal muscles are not truly in spasm. Equinovarus deformity or forefoot inversion might occur in some forms of massive and multiple tarsal coalitions.
With the 5-minute format, a candidate would have only used up about 3 minutes worth of time and so would be expected to perform a more detailed examination of the foot.

Examiner: Would you like to examine the range of movement of the feet?
The severity and limitation of joint movement depends on the site and extent of the tarsal coalition. A talocalcaneal coalition causes a marked restriction of subtalar movement, while a calcaneonavicular coalition causes some restriction of both

subtalar and midtarsal joint movement. Children with multiple tarsal coalitions have no movement at all in the subtalar and midtarsal joint [8].
Therefore, as a candidate, you would need to slickly examine the range of movement of the ankle, subtalar and midfoot joints and pick up a reduction in movement of the subtalar or midfoot joints.
Remember examination of subtalar joint movement; this is a differentiator between candidates. It is quite obvious to examiners if you are well practised in examining foot and ankle movements with this test or have just learnt it for the exam.

Examiner: What are the causes of a rigid flat foot?
Candidate: Causes include:

1. Congenital vertical talus: this can occur in association with other congenital anomalies such as myelomeningocele, arthrogryposis, DDH.
2. Neuromuscular foot: this can occur in association with other neuromuscular conditions, such as cerebral palsy, Duchenne muscular dystrophy and poliomyelitis.
3. Skew foot: this is known as Z foot, serpentine foot. There is hindfoot valgus, midfoot valgus (often called lateral shift) and forefoot adduction (metatarsus adductus).
4. Trauma.
5. Seronegative arthritis.
6. Iatrogenic to treatment, such as club foot.

Examiner: These are the patient's radiographs. What do you see?
Candidate: The radiograph is a lateral weight-bearing X-ray of the left foot, which confirms the presence of a calcaneonavicular coalition. The anteater sign is present and I can see talar beaking.

It is unlikely that you will be shown a young patient with secondary degenerative arthritis, but in this case there would be clinical features of reduced painful range of movement in the foot, possibly with secondary deformity.

Section 4

Hand and Upper Limb/Children's Orthopaedics

Chapter

12

Children's orthopaedics

Sattar Alshryda and Akinwande Adedapo

Introduction

The aims of the FRCS exam are to see if you have enough knowledge to practise as a consultant orthopaedic surgeon safely; not to test you as a paediatric orthopaedic consultant. Hence, the depth of knowledge required is not huge. Nevertheless, a substantial number of candidates fail this section.

This is partly because this section is not well covered by most exam books. Reading paediatric orthopaedic textbooks for the exam is not practical and can be confusing for the inexperienced. Most candidates, particularly those who could not have a paediatric placement, rely on a few good courses to consolidate this area of knowledge.

With this in mind, we used a different approach to cover this section in which we married actual exam questions gathered over the past few years with comprehensive and expanded answers. This keeps the theme of a viva book, provides comprehension of the topics and extra knowledge that may help high fliers to score high marks. Hence, you may find candidate answers with diagrams, X-rays and graphs; these are for your benefit rather than expected answers.

We stuck to the exam principle, where a simple question is asked concerning a clinical picture, X-ray or video clip, followed by increasingly difficult questions to explore the candidate depth and breadth of knowledge. Some of the questions are deliberately difficult and beyond average candidate level, some are easy and the majority are average.

We wish you the best of luck.

Station 1: Paediatric hip

Q 1: You have been referred a 12-year-old boy who presented with left knee pain for the last 3 weeks. No history of trauma. How would you approach this child?

My approach is to take a detailed history, perform thorough examination and order the appropriate investigations guided by my examination and provisional diagnosis.

Q 2: What goes through your mind when you face such a scenario?

Although my aim is to reach the correct diagnosis, I do not want to miss or delay diagnosing conditions that require immediate attention.

Am I dealing with septic arthritis, or juvenile arthritis?

Is it the knee or the hip? Could it be a slipped upper femoral epiphysis (SUFE)?

Q 3: How does a SUFE patient present?

The classic presentation is an overweight child presenting with groin, thigh and/or knee pain (referred pain, obturator nerve) and limping. There may be a history of minor trauma. The age is usually between 11 and 14 years old. It is more common in boys (boys 3 : 1. Boys age 12–14, girls age 11–13). The child may be able to weight bear and ambulate (stable slip) or may not be able to do so even with crutches (unstable). If he can walk, there is an external rotation of the involved limb and he cannot sit comfortably without keeping his leg straight (as he cannot bend the hip). There is usually restriction in the flexion, abduction and internal rotation of the affected hip.

Q 4: How can you confirm your diagnosis?

By radiological test; pelvis AP and cross-table lateral views of both hips. I do not prefer frog lateral as it may worsen the severity of the slip.

Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination, ed. Paul A. Banaszkiewicz and Deiary F. Kader. Published by Cambridge University Press.

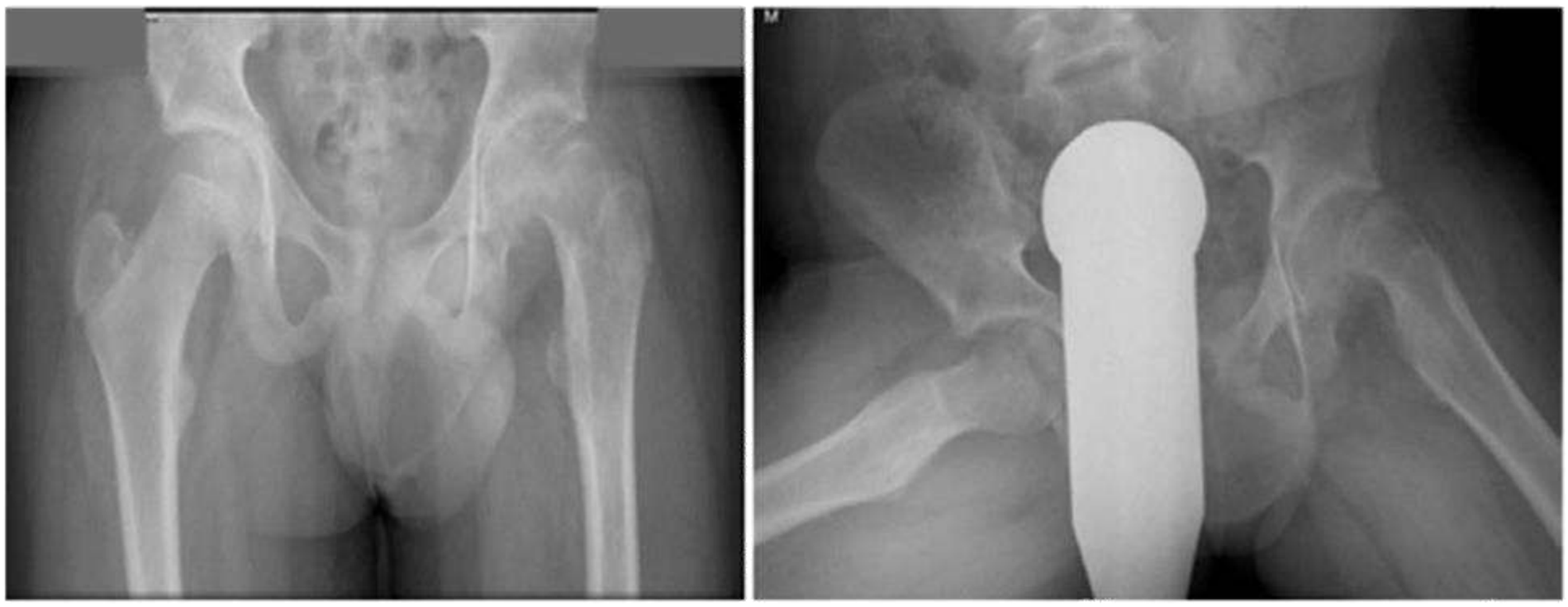


Figure 12.1 Pelvis X-ray of 12-year-old child with knee pain.

Q 5: These are his pelvis X-rays. What can you see? (Figure 12.1.)

These are AP and lateral X-rays of both hips. The most obvious abnormality is the slipped upper femoral capital epiphysis on the left side:

- Trethowan's sign is positive; a line (often referred to as Klein's line) drawn on the superior border of the femoral neck on the AP view should pass through the femoral head. In SUFE, the line passes over the head rather than through the head (compare left and right hip).
- Decreased epiphyseal height as the head is slipped posteriorly behind the neck.
- Remodelling changes of the neck with sclerotic, smooth superior part of the neck and callus formation on the inferior border. This may not be seen in acute slip.
- Increased distance between the tear drop and the femoral neck metaphysis.
- There are other radiological signs of SUFE which are not present on this radiograph such as widening and irregularity of the physeal line (early sign) and Steel's blanch sign which is a crescent-shape dense area in the metaphysis due to superimposition of the neck and the head.

Q 6: Can you grade the severity?

There are two radiological grading systems:

a. Severity of the slip by Wilson.
b. Grading using the lateral epiphyseal–shaft angle of Southwick.

I consider this grade II or moderate slip, although the AP gives a false impression of mild slip. (Figures 12.2, 12.3.)

Q 7: Are you aware of any other grading or classifications system?

Randall Loder[1] in his classic paper evaluated the presenting symptoms and radiographs of 54 patients (55 hips) and reclassified the slipped epiphyses as unstable or stable, rather than acute, chronic or acute-on-chronic.

I. Stable slip: child is able to weight bear and ambulate with or without crutches.
II. Unstable slip: child is not able to weight bear and ambulate.

Thirty of these were unstable and 25 were stable. All slips were treated with internal fixation. Avascular necrosis developed in 14 (47%) of the unstable hips and in none of the stable hips. Fourteen (47%) of the 30 unstable hips and 24 (96%) of 25 hips had a satisfactory result.

It is important to notice that a reduction occurred in 26 unstable hips and in only two of the stable hips. He was not able to demonstrate an association between early reduction and the development of AVN.

Q 8: Why does it happen?

There are several theories to explain the aetiology of SUFE. Some are more convincing than others; but none is perfect. These can be summarized as follows:

- The biomechanical theory: There are several anatomical features that lead to increase the shear forces across the physis and lead to slip:
  1. Increased weight (> 80th centile).
  2. Femoral retroversion (> 10°).
  3. Increased physis height due to widened hypertrophic zone.

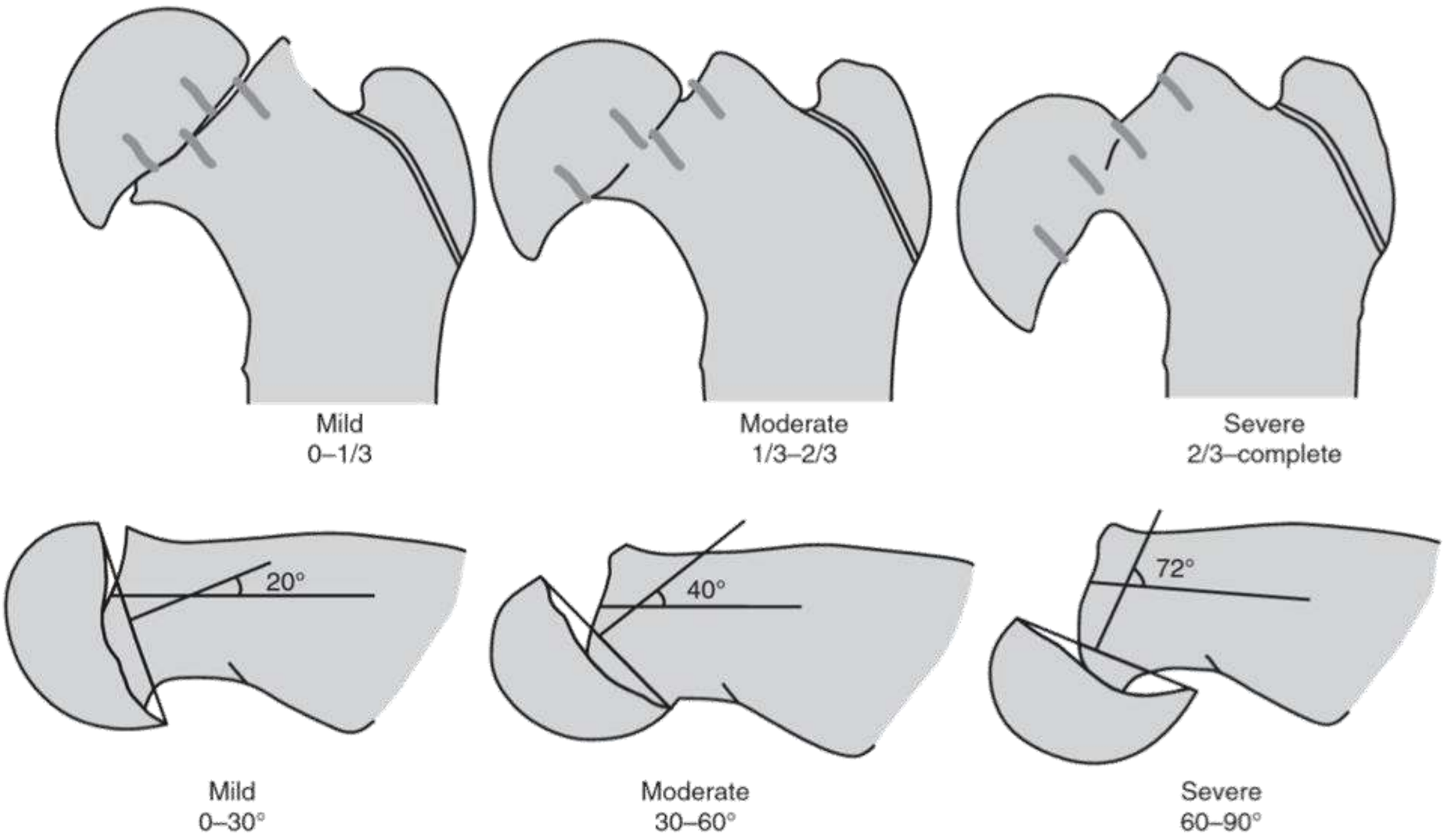


Figure 12.2 Slipped upper femoral epiphysis (SUFE) radiological grading.

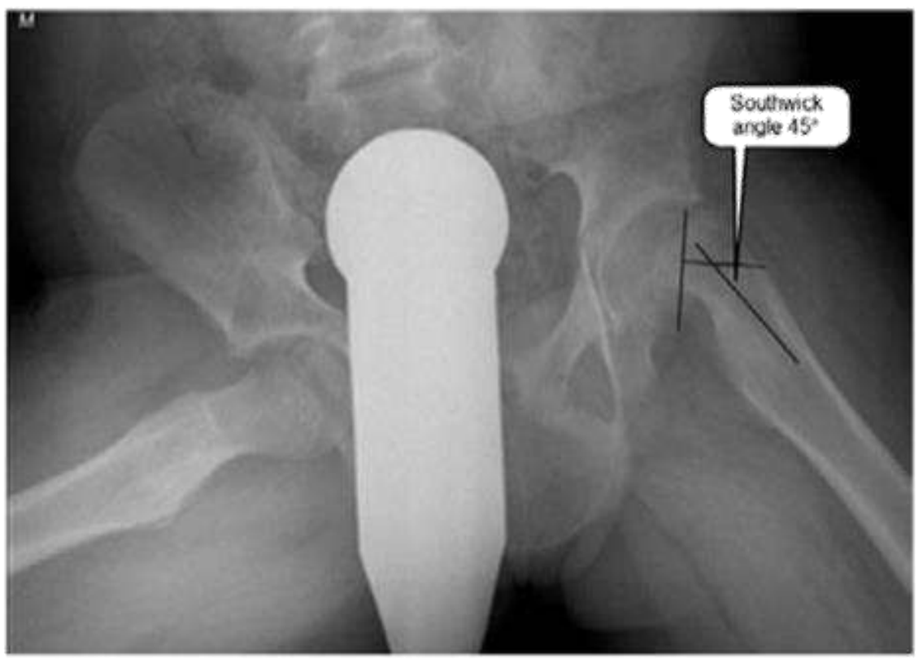


Figure 12.3 The lateral epiphyseal–shaft angle of Southwick.

4. More vertical slope of the physis.
5. Trauma.

- Structural defect of the physis theory:
  1. Hormonal theory: several endocrine disorders have been implicated with causation of SUFE. Bilateral slip is more common with endocrine diseases.
     i. Hypothyroidism.
     ii. Growth hormone deficiency.
     iii. Sex hormone (more common in boys 3 : 1. Boys age 12–14, girls age 11–13).
     iv. Renal osteodystrophy. It is associated with the highest risk (90%) of bilaterality. By contrast, idiopathic SUFE has a 20% risk of bilaterality initially and a further 10% to 20% risk until maturity. It is also associated with the highest risk (43%) of progressing to grade III slip. The slip goes through the metaphysis rather than the hypertrophic zone of the physis.
  2. Radiotherapy.
  3. Racial/ethnic (more common in blacks).
  4. Idiopathic.
- Combined: increased sheer forces on abnormal physis.

Q 9: How would you treat this child?

This child has a grade II slip and I would treat him with pinning in situ (PIS). The primary aim of the treatment is to stabilize the slip and prevent further progression until physis closure. There is almost a universal agreement that grade I and grade II slips

can be treated with PIS using a single cannulated screw. Some advise multiple smooth pins in a younger age group (< 8 years) to allow some growth.

However, there is controversy on the best treatment for grade III. Some advocate pinning in situ and a re-alignment procedure later if the remodelling is not optimum. There are several re-alignment procedures recommended such as subtrochanteric, intertrochanteric and neck osteotomies. Others recommend acute open reduction and fixation using Dunn or Fish osteotomy and more recently using flip osteotomy and surgical dislocation (Ganz technique).

Q 10: I agree, PIS is a reasonable option here. How soon do you want to do it?

The timing of operation is still controversial. Given the rarity of the condition (incidence 3/100 000), most studies that looked at the timing of surgery and outcome are suboptimum. Peterson *et al.*[2] showed early stabilization within 24 hours was associated with less AVN (3/42 = 7%) in comparison with those stabilized after 24 hours (10/49 = 20%). Kalogrianitis and colleagues[3] showed that AVN developed in 50% (8/16) of the unstable SUFE in their series. All but one were treated between 24 and 72 hours after symptom onset. They recommend immediate stabilization of unstable slips presenting within 24 hours; if this is not possible, delaying the operation until at least a week has elapsed.

Q 11: Would you pin the other non-slipped, asymptomatic side?

This is also controversial. The quoted risk of contralateral slip varies from 18 to 60%. Prophylactic PIS is not free of risk and it should be weighed against the benefit. The proponents and opponents have some evidence to support their views.[4] The following factors play a role in decision making:

1. Age of the child (< 10 years is associated with a higher risk of bilaterality).
2. The aetiology of the slip (renal osteodystrophy and endocrine disorders have a high incidence of bilaterality).
3. The ability and the compliance of the child and family.
4. The nature of current slip (very bad slip occurring over a very short period of time may justify pinning the other side).

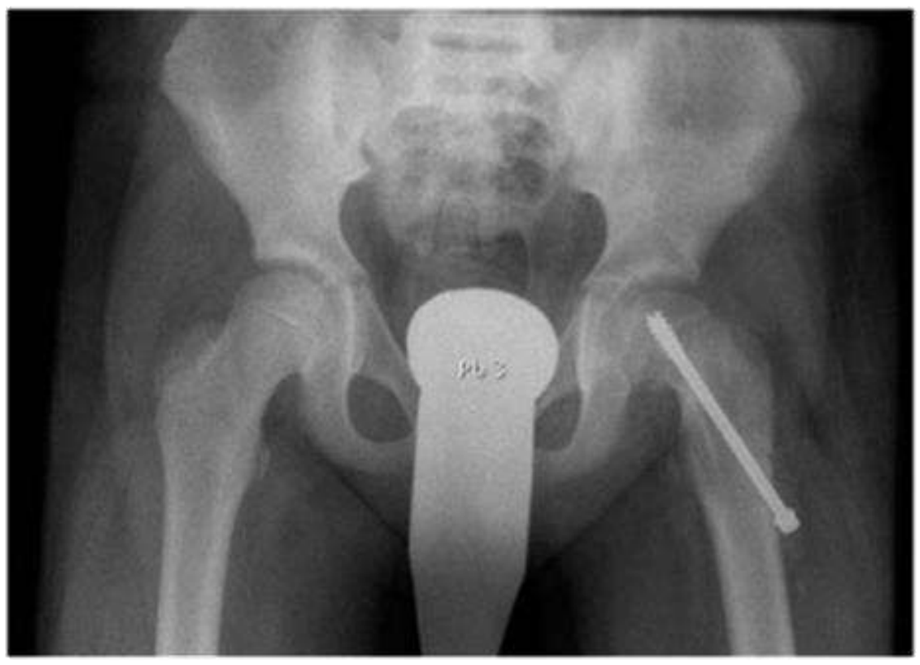


Figure 12.4 Postoperative X-ray after pinning in situ of left SUFE.

Q 12: This is the postoperative X-ray of the child. Any comments? (Figure 12.4.)

This is an AP pelvis plain X-ray with a single cannulated screw in situ. It is centred in the neck and epiphysis. There are about three threads in the epiphysis. Ideally, I would like to see five threads, but without seeing the lateral view, I cannot criticize this.

Q 13: A few months later, your secretary received a phone call from his general practitioner (GP) because he develops pain in the other hip. The GP asks if you could see him in your next available clinic.

Contralateral slip is not uncommon and I always warn my patients to seek urgent medical advice if they develop pain in the other hip. I would bring this child to hospital for urgent X-ray rather than waiting to see him in the routine clinic. Some of these slips progress rapidly to severe slip.

Q 14: This is the X-ray you requested. What do you see? (Figure 12.5.)

As I expected, there is a contralateral slip (probably mild) and I would consider pinning in situ.

Q 15: I agree. What do you think of the other side?

I think he is outgrowing the screw. There is a single thread of the screw left in the epiphysis and the physis is still open. I will exchange this for a longer screw. It should be relatively easy. I will pass the guide wire through the cannulated screw and exchange it for a longer screw.

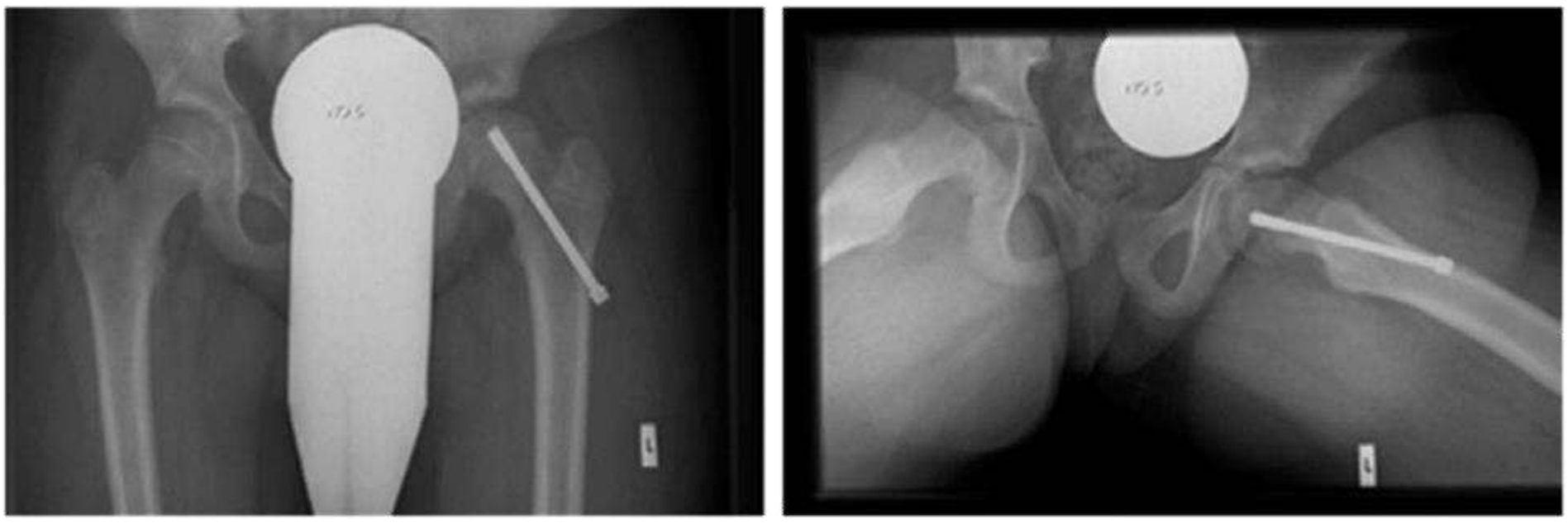


Figure 12.5 Pelvis X-ray (AP and lateral) of a child with right hip pain.

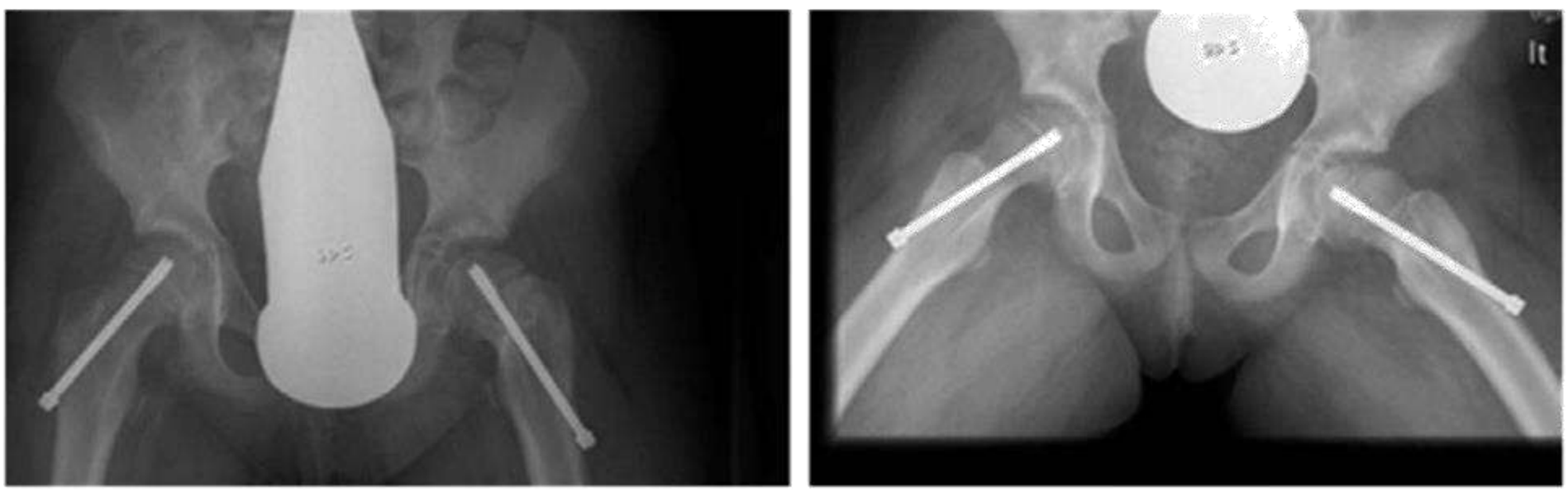


Figure 12.6 Postoperative plain X-ray of the pelvis after the second operation.

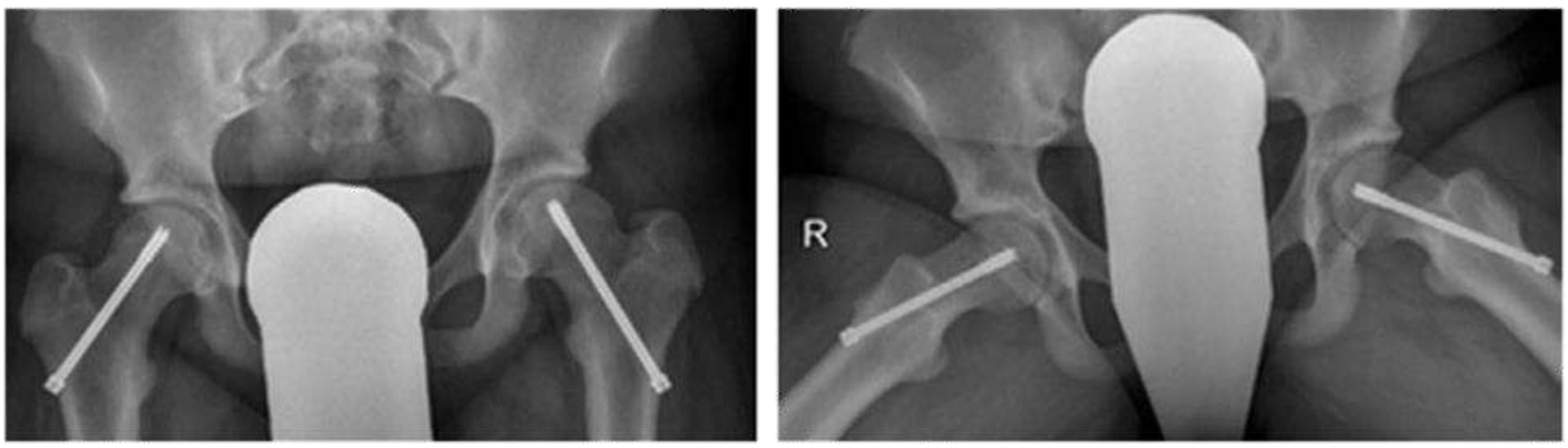


Figure 12.7 Pelvis X-rays 7 years after pinning in situ.

Q 16: This is exactly what was done. (Figure 12.6.) A few years later, the patient asks whether you would leave the screws or remove them. He is asymptomatic. His X-ray is shown in Figure 12.7.

The X-rays shows the physes have been closed, hence the screws become redundant. Interestingly there is a reasonable remodelling of the neck and the joints are not arthritic. I usually offer my patients removal of the screws and my justification is that this would make future hip replacement (if it is needed) more difficult; particularly if the screw heads are fully covered with bone. Of course, removing the screws is associated with risk and the patient has to be informed.

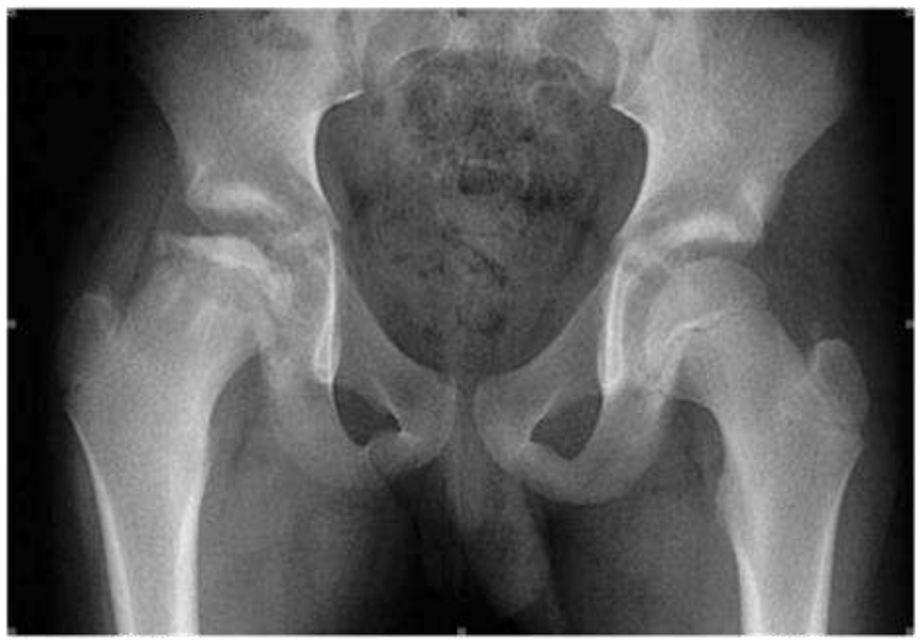


Figure 12.8 Pelvis X-ray of a child with right hip pain and limping.

Q 17: This is a pelvic X-ray of a 9-year-old boy who has been limping over the last 3 months. What can you see? (Figure 12.8.)

This is an AP view of the pelvis in a skeletally immature patient. The most obvious feature is the collapse of the right femoral head with increased bone density. There is a widening of the joint space on the right side. At the top of my list is Legg–Calvé–Perthes disease (LCPD), but other diagnoses such as infection need to be excluded.

Q 18: What is Perthes' disease?

It is a non-inflammatory AVN of the femoral head in a growing child caused by interruption of blood supply. The condition was first described by Waldenstorm, but he attributed it to tuberculosis; it was then described more accurately by Arthur Legg (1874–1939), Jacques Calvé (1875–1854) and George Perthes (1869–1927) almost at the same time; hence the name Legg–Calvé–Perthes disease (LCPD).

It is not common (1\10 000), affecting children between 4–9 years old of low socioeconomic class. Child is often small with delayed bone age by usually 2 years. It is bilateral in 15% of cases but involvement is usually asymmetrical and never simultaneous (in contrast to multiple epiphyseal dysplasia). There may be a family history.

Q 19: What does it happen?

The aetiology is unknown; however, several theories have been put forward to explain it:

1. The anatomical theory.

The blood supply to the femoral head changes as the child grows (Table 12.1). The change over to adult pattern may be affected comprising the blood supply to the femoral head leading to ischaemic necrosis.

Table 12.1 Blood supply of the femoral head.

Age	Birth–4 years	4 years–adult	Adult
Source	Medial and lateral circumflex arteries from profunda femoris artery Ligamentum teres with posterior division of obturator artery	Posterosuperior and posteroinferior retinacular from medial femoral circumflex artery Negligible lateral circumflex artery Minimum ligamentum teres	Medial femoral circumflex to lateral epiphyseal artery

2. Hydrostatic pressure theory.

This theory attributes the reduction in blood supply to the femoral head to the increase in the intraosseous venous pressure which has been noticed in several cases.

3. Thrombophillic theory.

There is evidence of association of LCPD with various forms of thrombophilia. In one study, 72 patients with LCPD were compared with 197 matched healthy controls.[5] The factor-V Leiden mutation was more common in LCPD (8/72) than in the controls (7/197) (chi-square = 5.7, p = 0.017). A high level of anticardiolipin antibodies was found in 19 of the 72 LCPD compared with 22 of the 197 controls (chi-square = 9.5, p = 0.002). Other studies showed association of LCPD with protein S and C abnormalities.[6,7] It is important to remember that association does not always mean causation.

Q 20: What is your differential diagnosis?

In this scenario where the right side only is involved, my differential diagnoses are:

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.

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.

Q 21: Do you know any grading or classification system?

There are several classifications to address different aspects of LCPD. These include the following.

Classification addressing chronological radiographic stages (Waldenstrom)

I. Initial (sclerotic/necrotic) stage

It lasts 6–12 months. Ischaemia leads to subchondral bone death and necrosis (dead bone looks dense on plain radiograph). There is joint space widening due to continuous cartilage growth (nutrient from synovial fluid). This can be subdivided into early (no loss in epiphysis height) or late where there is some loss of epiphyseal height but the epiphysis is still in one piece.

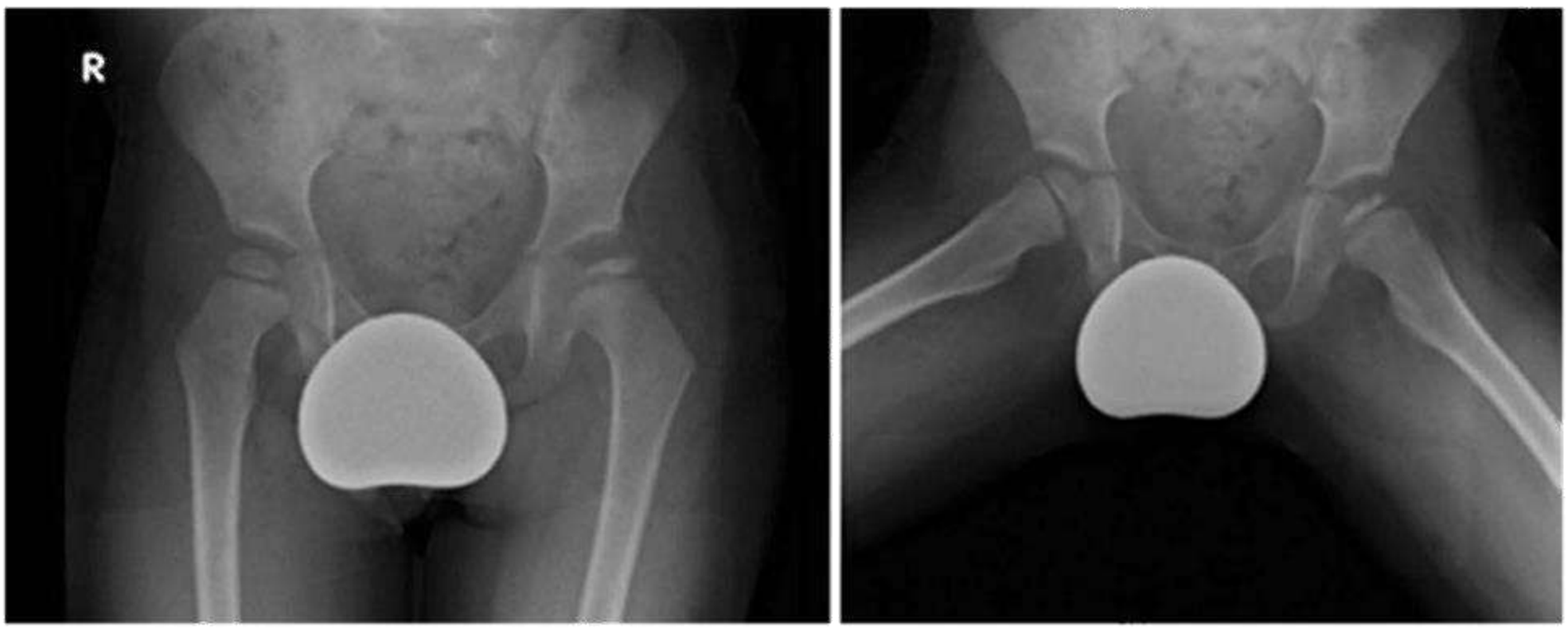


Figure 12.9 Legg–Calvé–Perthes disease (LCPD): initial (sclerotic/necrotic) stage.

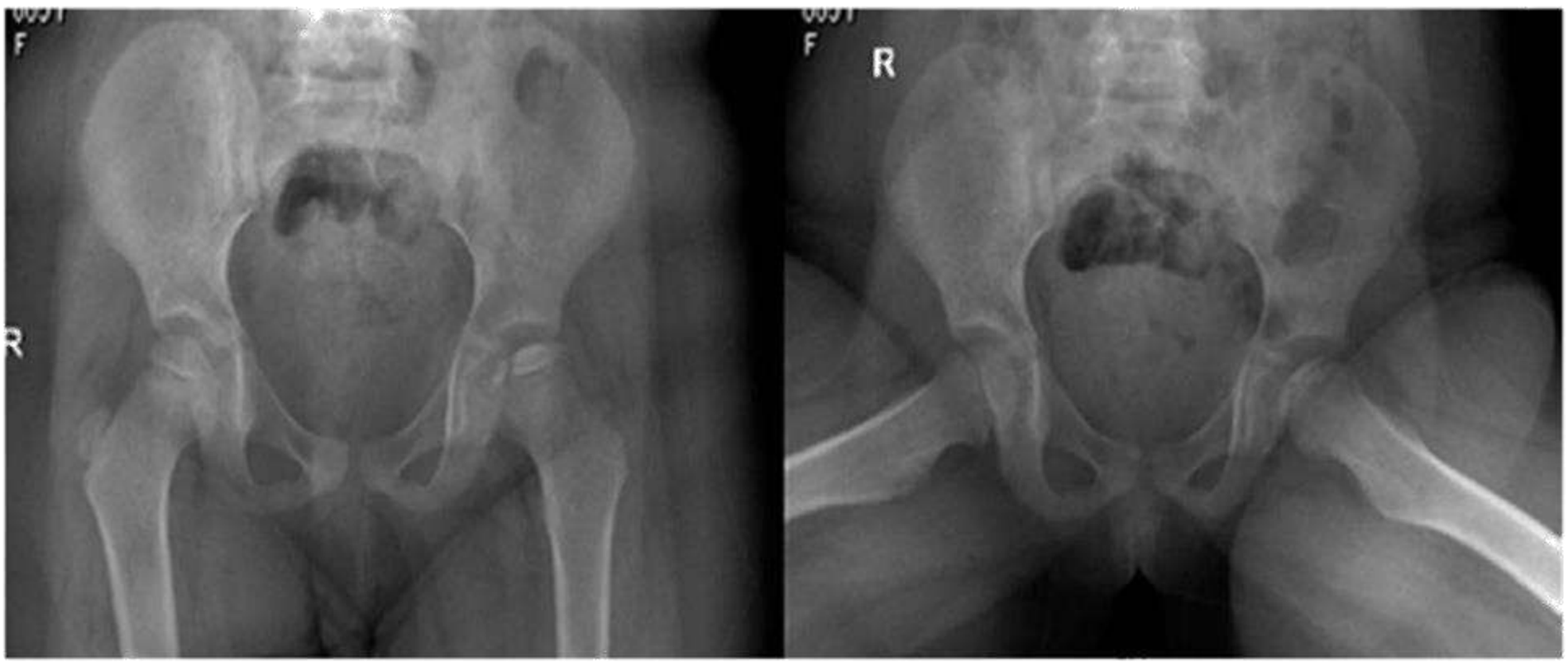


Figure 12.10 Legg–Calvé–Perthes disease (LCPD): early fragmentation stage.

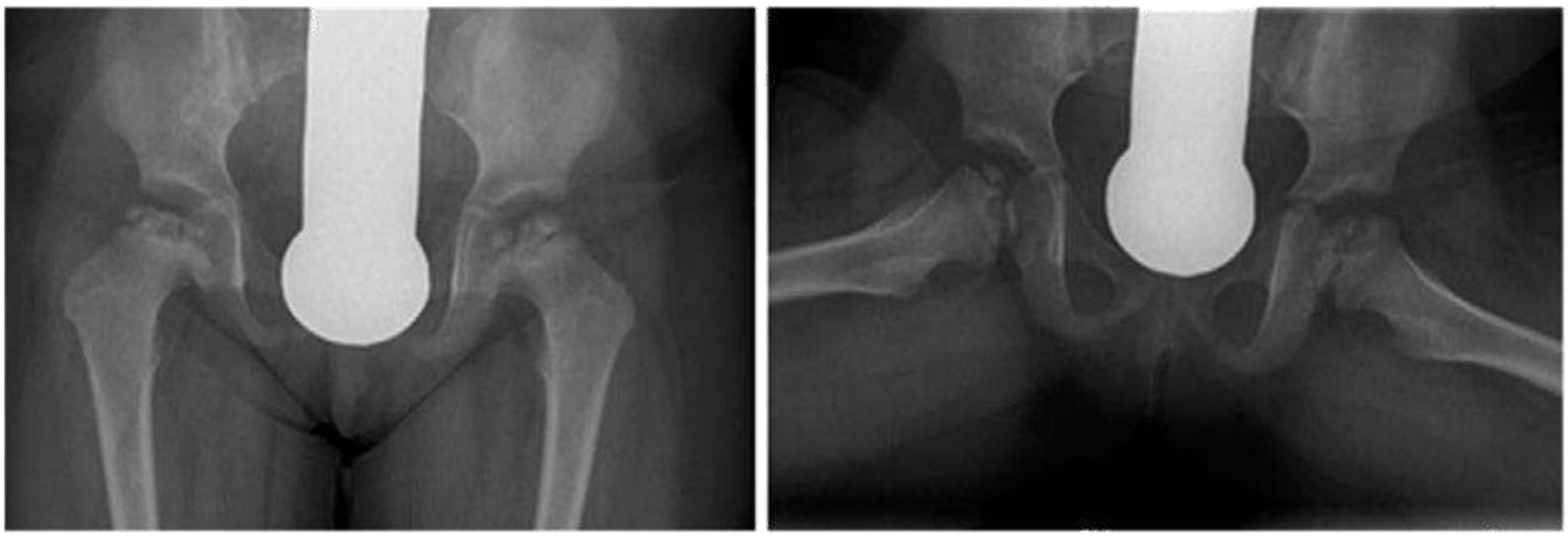


Figure 12.11 Legg–Calvé–Perthes disease (LCPD): late fragmentation stage.

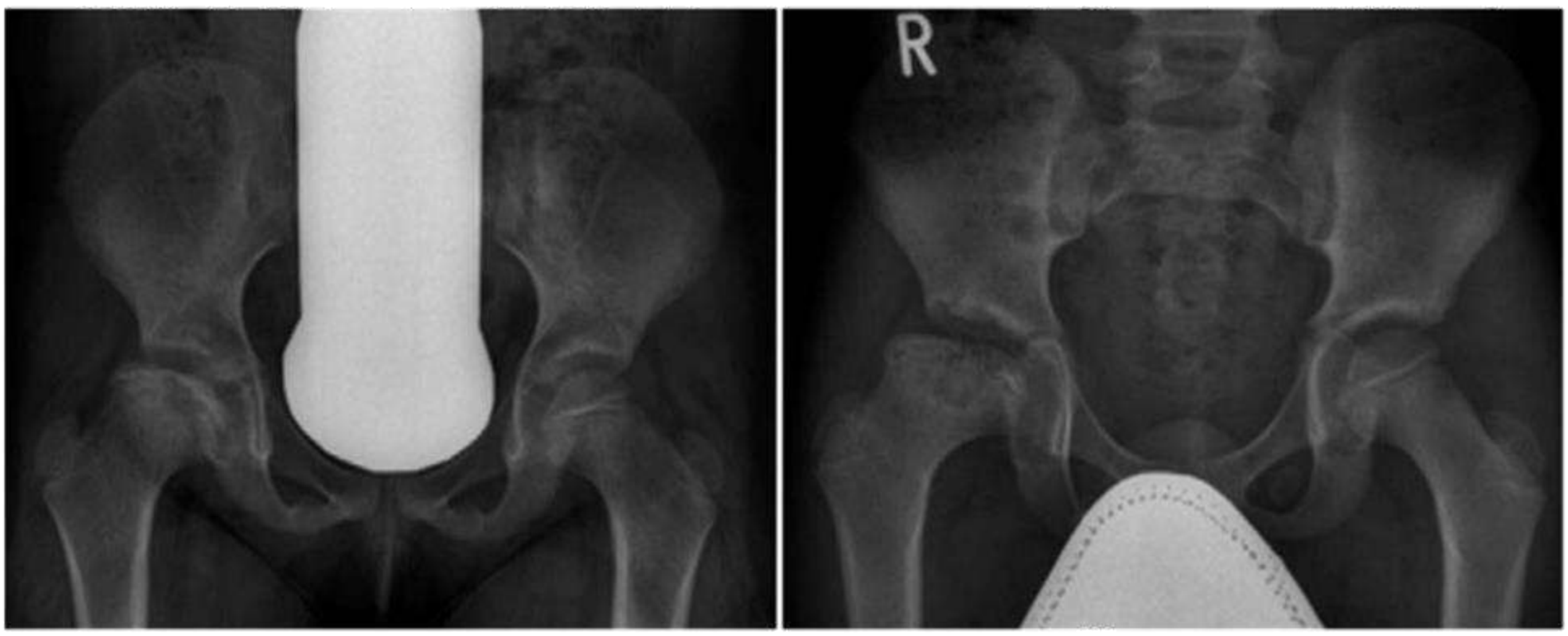


Figure 12.12 Legg–Calvé–Perthes disease (LCPD): reossification stage; IIIa (left) and IIIb (right).

II. 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 12–24 months. This stage can be further divided into early (1–2 fissures only) or late when the head is in several fragments.

III. Reossification (healing) stage

Osteoblasts form new bone which is soft and pliable. It starts peripherally and progresses centrally. This usually appears as a small and expanding fragment on the lateral part of the epiphysis marking the beginning of this stage IIIa. This soft bone matures and covers more than a third of the epiphysis in stage IIIb. This stage usually lasts 6–24 months.

It is critical to keep the soft head within acetabulum for natural moulding in order to maintain its sphericity. If uncontained the soft head will be extruded, collapse and lose its sphericity leading to early OA of head and acetabulum. This is the basis of containment treatment.

IV. 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.

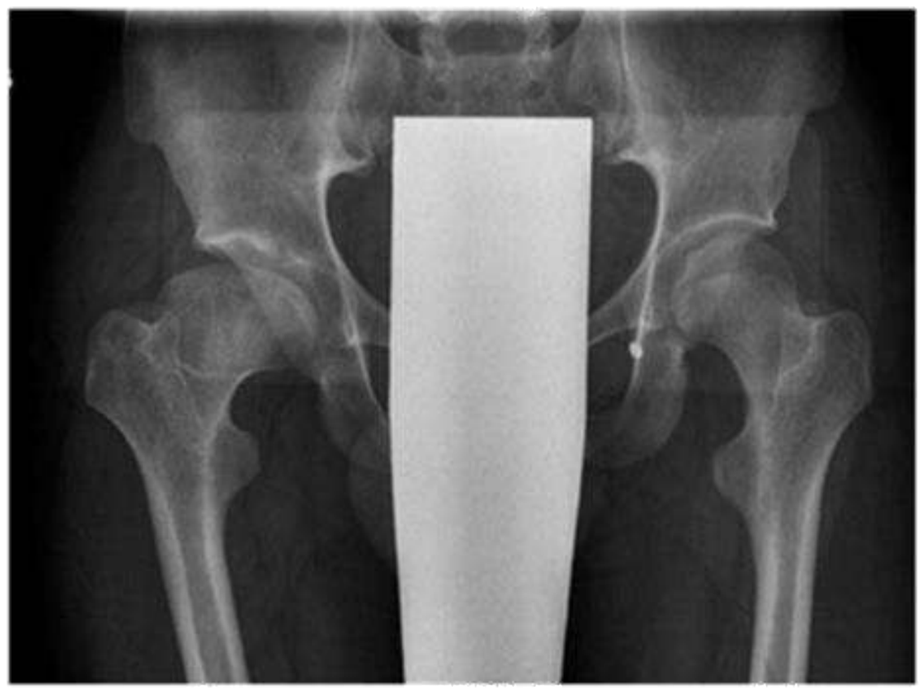


Figure 12.13 Legg–Calvé–Perthes disease (LCPD): remodelling (residual) stage.

Classification addressing severity of head involvement

Catterall 1971 (Figure 12.14)

Based on extent of head involvement 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 segment – fragmentation (sequestrum).
- Lateral part/rim is intact (protects the central involved area).
- Junction – clear.
- Metaphyseal reaction present – anterior.
- Subchondral fracture – anterior.

Catterall III

- 50–75% head involvement.
- Anterior segment involved. Lateral head – also fragmented.
- Only the medial portion is spared.
- Loss of lateral part/support worsens the prognosis.
- Junction – sclerotic.
- Metaphyseal reaction present – anterior and lateral.

Catterall IV

- >75% head involvement.

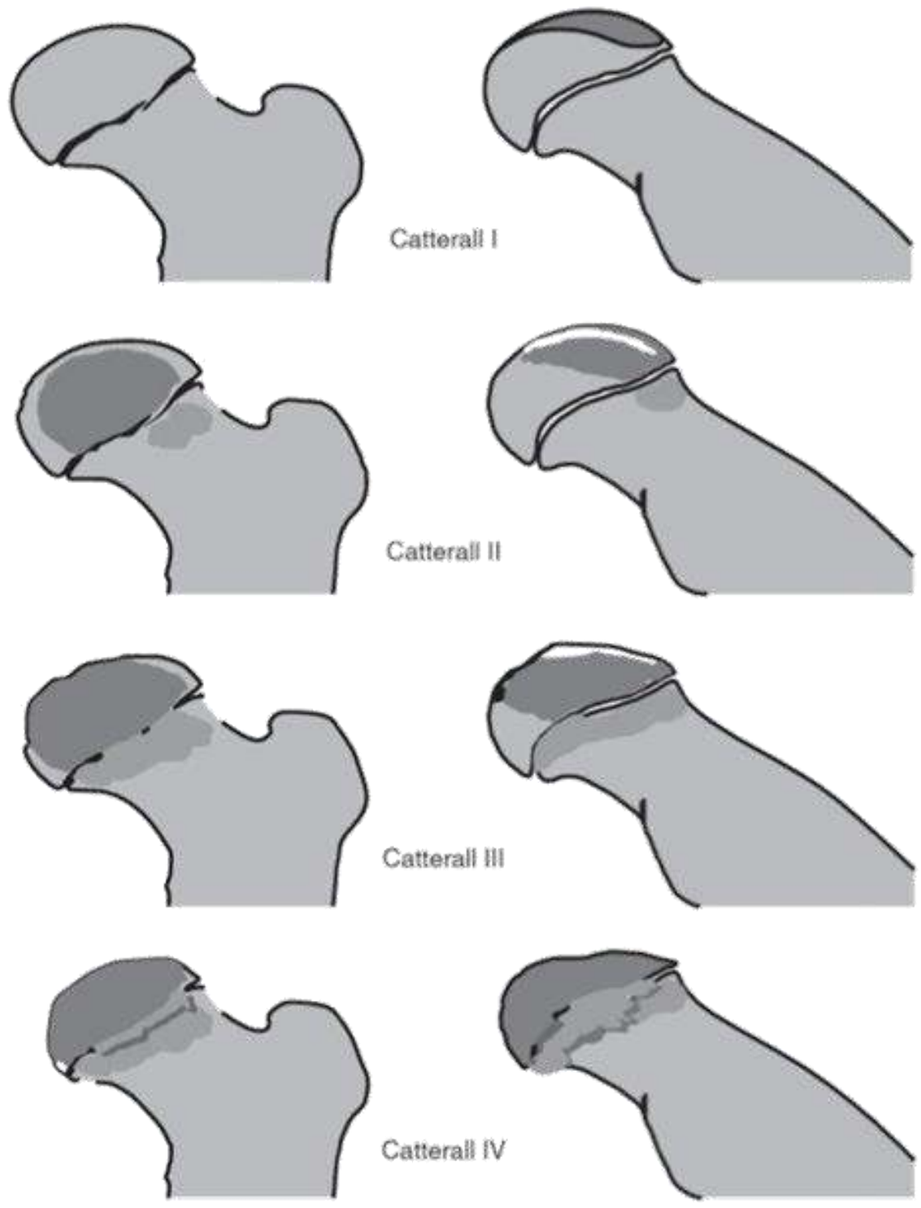


Figure 12.14 Legg–Calvé–Perthes disease (LCPD): Catterall classification.

Salter and Thompson 1984

Salter and Thompson 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.

Group A

- Normal height of the lateral one-third of the head is maintained.
- Fragmentation occurs in the central segment of the head.

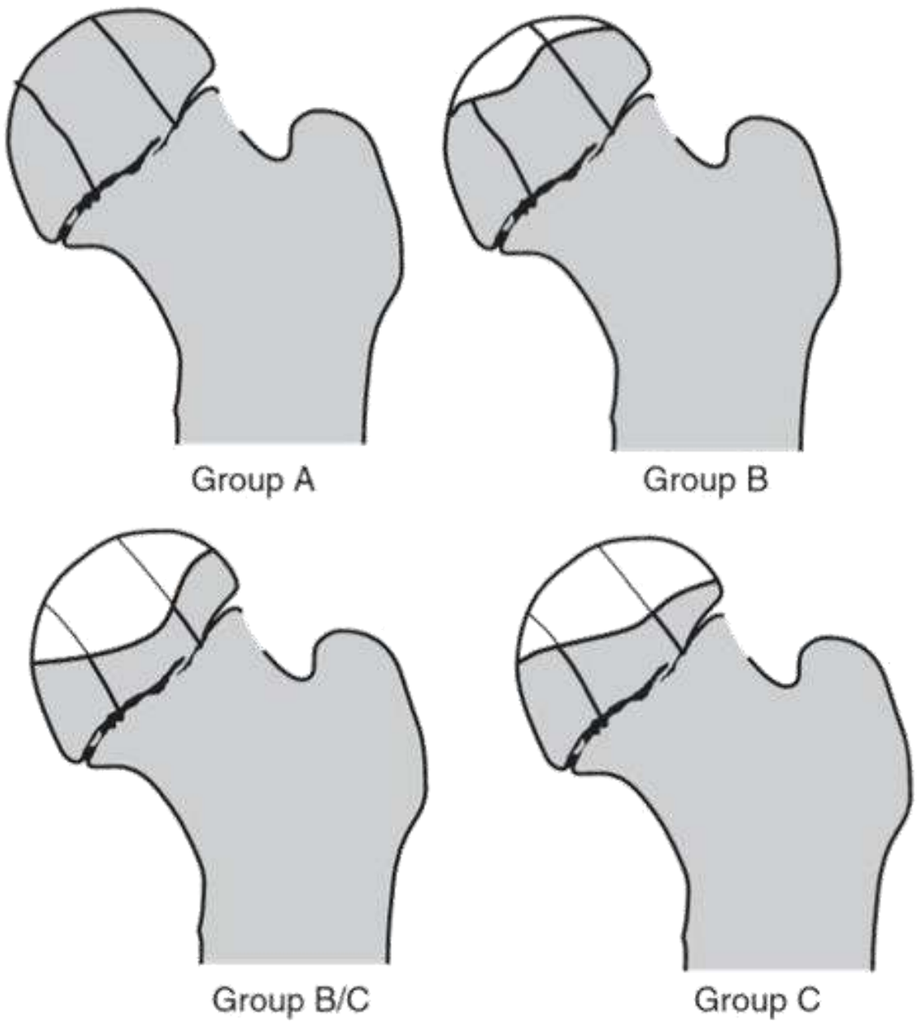


Figure 12.15 Legg–Calvé–Perthes disease (LCPD): Herring classification.

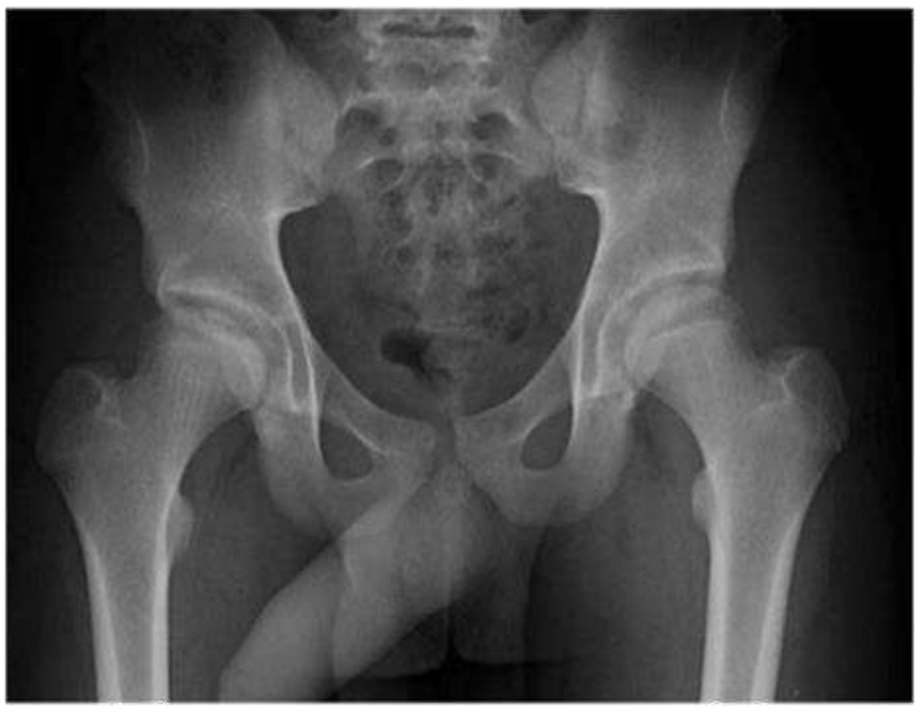


Figure 12.16 Legg–Calvé–Perthes disease (LCPD): Herring A.

Group B

- More than 50% of the original lateral pillar height is maintained.
- There may be some lateral extrusion of the head.

Group C

- Less than 50% of the original lateral pillar height is maintained.
- The lateral pillar is lower than the central segment early on.

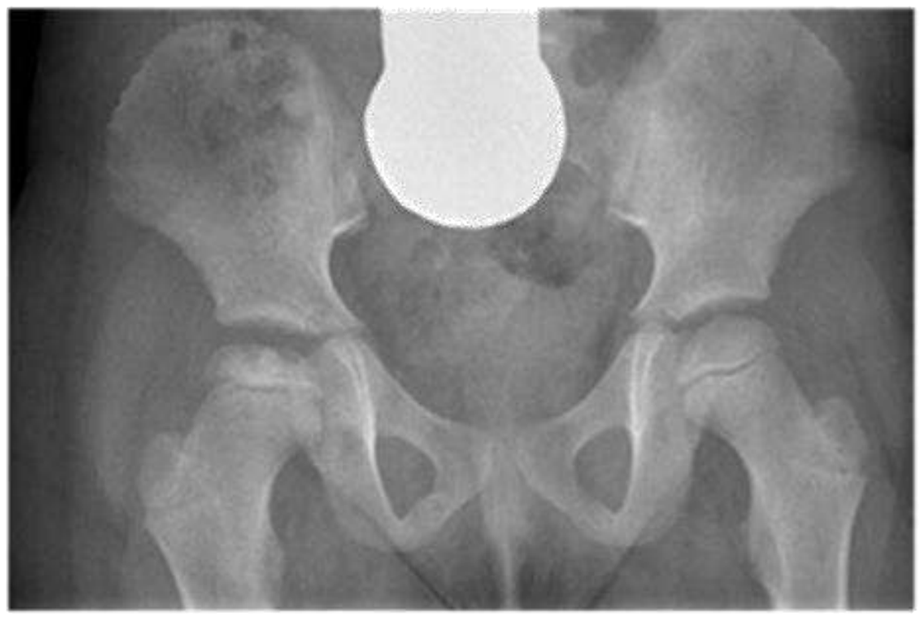


Figure 12.17 Legg–Calvé–Perthes disease (LCPD): Herring B.

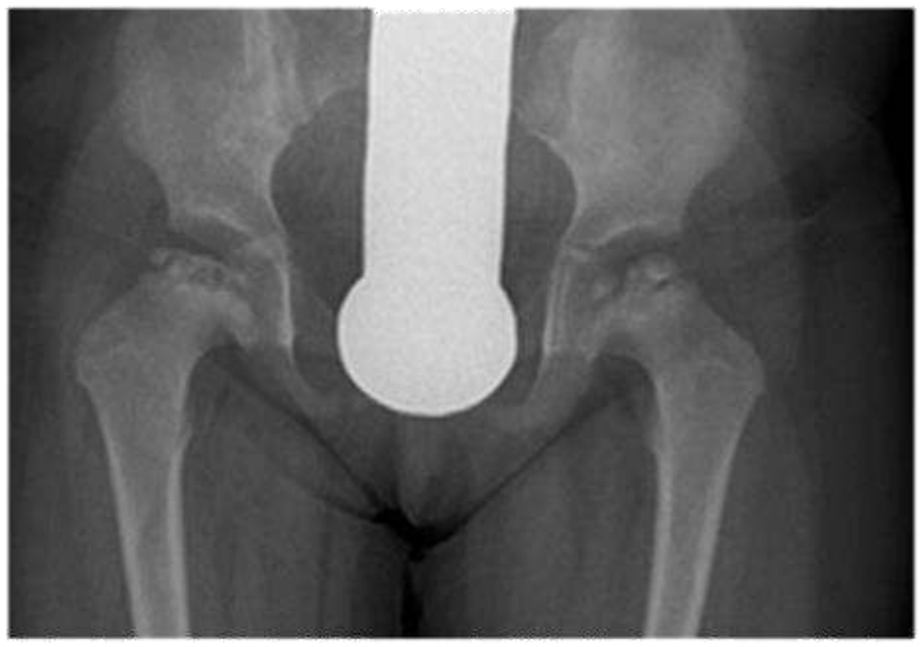


Figure 12.18 Legg–Calvé–Perthes disease (LCPD): Herring C (right) and Herring B/C left.

Group B/C

- Less than 50% of the original lateral pillar height is maintained.
- The lateral pillar is higher than the central segment.

Classification addressing outcome

Mose classification

Using the concentric circle technique to compare and classify the final outcome in PD at the end of growth. The final shape of the head may be compared with a perfect circle using the Mose template and both AP and lateral images.

Given that a congruous but aspherical head can perform well suggests that the Mose criteria are too strict and impractical.

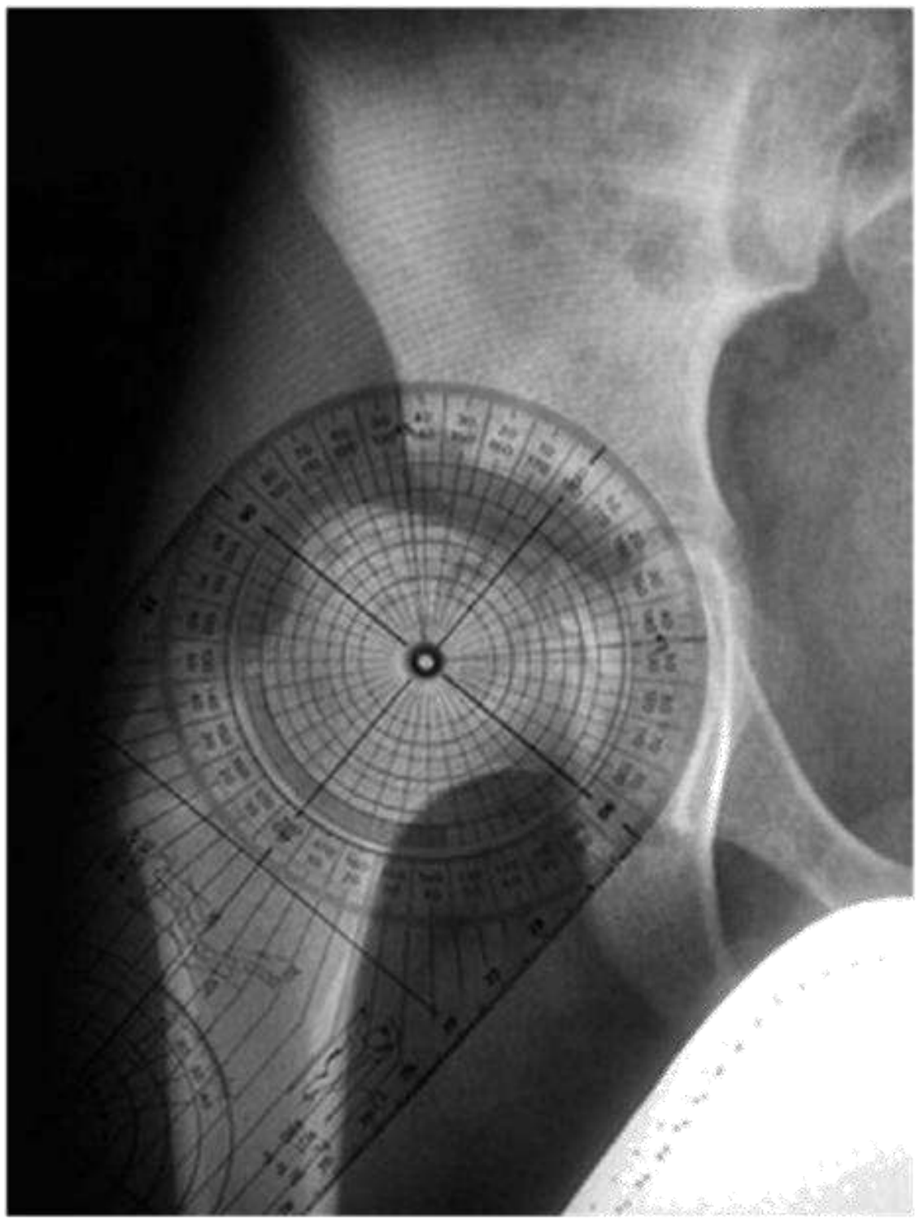


Figure 12.19 Legg–Calvé–Perthes disease (LCPD): Mose grading.

Table 12.2 Mose grading.

Outcomes	Description
Good outcome	Aspherical head contour is within 1 mm of a given circle on both views
Fair outcome	Aspherical head contour is between 1–2 mm
Poor outcome	Aspherical head contour is >2 mm

Stulberg classification

Stulberg showed that a lack of sphericity and congruency were both predictors for poor outcome.[8] Table 12.3 summarizes Stulberg's classification. 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; and group C (Stulberg IV and V) have a flat femoral head.

Table 12.3 Stulberg grading.

Congruency	Class	Description
Spherical congruency	I	Normal spherical head
	II	Spherical head, coxa magna/breva, steep acetabulum
Aspherical congruency	III	Ovoid or mushroom-shaped head
	IV	Flat head on flat acetabulum (may hinge on abduction)
Aspherical incongruency	V	Flat head but normal acetabulum

Table 12.4 Legg–Calve–Perthes disease: poor prognostic signs.

Poor clinical prognostic signs (FOOBS)	Poor radiological prognostic signs
Male	Gage's sign (V-shape lucency at lateral epiphysis; Figure 12.20)
Older age	Horizontal growth plate – implies a growth arrest phenomenon and deformity
Obesity	Lateral calcification (lateral to the epiphysis – implies loss of lateral support and head extrusion)
Bilateral	Lateral subluxation – implies loss of lateral support Uncovering of the femoral head >3 mm in excess of opposite side (measured as the horizontal distance between a vertical line through the outer lip of acetabulum and lateral edge of femoral head physis)
Stiffness	Metaphyseal rarefaction/cyst

Q 22: What do you think about this child's prognosis?

Unfortunately, this child already has three poor prognostic signs. He is a boy, 9 years old and the plain X-ray showed Herring C. There are other clinical and radiological prognostic signs which I would look for in my assessment (Table 12.4).

Q 23: How would you treat this patient?

I would start treating this child symptomatically with rest, analgesia, anti-inflammatory medication, temporary

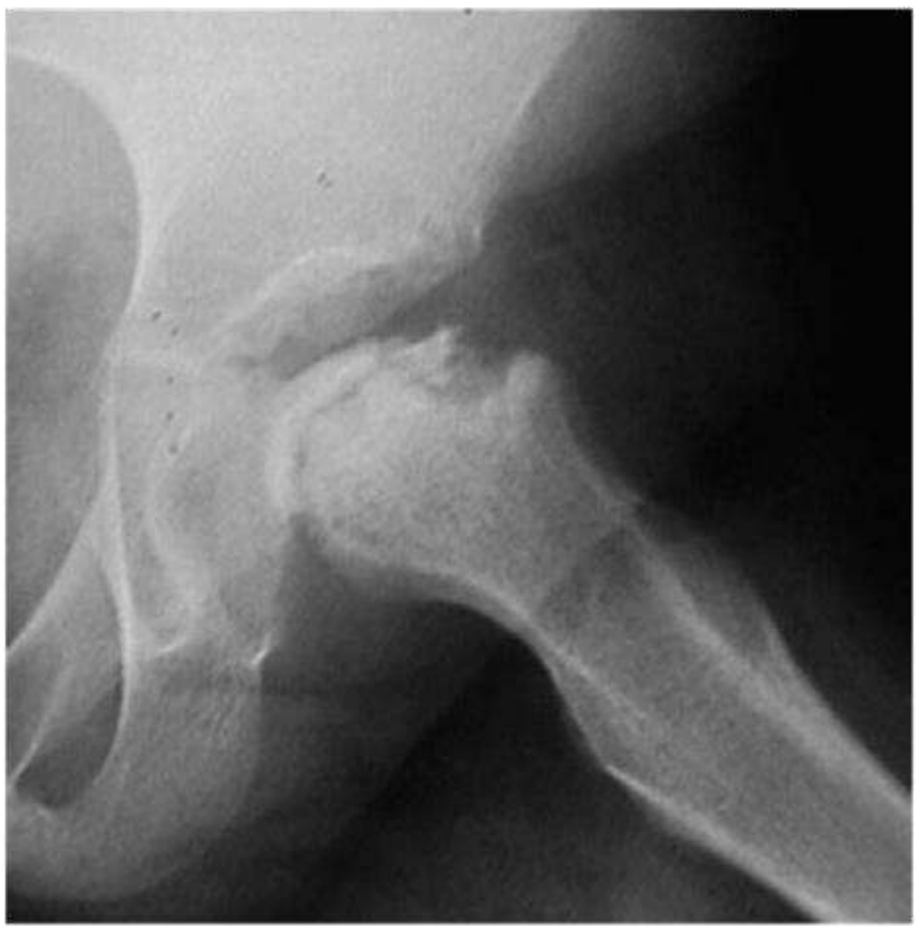


Figure 12.20 Legg-Calvé-Perthes disease (LCPD): Gage sign.

non-weightbearing with crutches and I may consider admitting for a short period of gentle traction. Physiotherapy plays an important role in improving range of motion. Several types of braces have been advocated but their values have been heavily questioned and compliance is a real issue in this age group.

Q 24: Would you consider surgery?

It depends which surgery you are referring to. I would not recommend containment surgery (femoral varus osteotomy or Salter osteotomy) for this boy. There is evidence that containment surgery is beneficial in certain subgroups of LCPD. Patients with lateral pillar B and B/C involvement and aged 8 years or older are likely to benefit from surgery.[9,10]

Unfortunately, studies have not shown benefit of containment surgery for lateral pillar C hips patients. However, one study suggests that early distraction with hinged external fixation may be of value in such patients.[11]

Q 25: Which studies are you referring to?

Herring *et al.* report on the results of the Legg–Perthes Study Group.[9] Thirty-nine surgeons from 28 centres took part in a prospective study. Each surgeon agreed to apply a single treatment method to each patient who met the study criteria. All patients were between 6 and 12 years of age at the onset of the disease, and none had had prior treatment. The treatment groups were no treatment, range of motion treatment in which the patient did exercises once a day, Atlanta brace treatment, femoral varus osteotomy, and Salter osteotomy.

The study showed that age, lateral pillar grading and treatment methods were significantly related to outcome.

In group B hips with an age at onset of more than 8 years, 73% of the operated hips had a Stulberg I or II result compared with 44% of the non-operated hips ($p = 0.02$). For the group B hips with onset at 8 years or younger, there was no advantage demonstrated for the surgical group. The group C hips were not shown to benefit from surgical or non surgical treatments.

Wiig *et al.* reported on a nationwide prospective study.[10] Twenty-eight hospitals in Norway were instructed to report all new cases of LCPD over a period of 5 years.

A total of 368 with unilateral disease were included in the study. For patients over 6 years of age at diagnosis with more than 50% necrosis of the femoral head (152 patients), the surgeons at the different hospitals had chosen one of three methods of treatment: physiotherapy (55 patients), the Scottish Rite abduction orthosis (26) and proximal femoral varus osteotomy (71). The study showed that the strongest predictor of poor outcome was femoral head involvement of more than 50% (modified Catterall classification) followed by age at diagnosis, then lateral pillar grades. In children over 6 years at diagnosis with more than 50% of femoral head necrosis, proximal femoral varus osteotomy gave a significantly better outcome than orthosis or physiotherapy. There was no difference in outcome after any of the treatments in children under 6 years.

Other small studies showed the benefit of shelf acetabuloplasty as a salvage operation for extruded head;[12,13] valgus osteotomy in hinged abduction;[14] and trochanteric growth arrest or advancement when there is overgrowth.

Q 26: This becomes confusing. Can you simplify it?

I agree it is confusing and this reflects the current state. However, for the sake of simplicity:

< 6 years old

- Prognosis is good for the majority.
- Bed rest, traction, pain-relieving anti-inflammatory medication and rest.

- No evidence that abduction splints or surgical intervention is warranted in the majority of these younger patients.

> 8 years old (think of this as a range rather than absolute number)

- Herring lateral pillar classification A may do well and do not need surgery.
- Herring lateral pillar classification B and B/C; containment of the head within the acetabulum seems to be warranted.
- Herring lateral pillar classification C is associated with poor outcome and containment surgery does not improve outcome. However, shelf acetabuloplasty may be useful in the early stage of the disease. Valgus osteotomy if patient develops hinged abduction late in the disease.

For the best result, surgery should be performed in the earliest stage of the disease; maybe even before being able to classify the severity. If all children older than 8 were to be offered surgical treatment, the group A and C hips would not likely benefit. These groups combined represent only 13% of hips presenting at age older than 8, and this approach may be justified.[15]

Q 27: **What do these three clinical photographs demonstrate in this 6-month-old child?** (Figure 12.21.)

The top left photo shows that the left thigh is shorter than the right (Galleazzi test is positive). The other two pictures show an abnormal skin crease (top right) and limitation of the left hip abduction (bottom). My suspicion is that this child has a dislocated left hip.

Q 28: **How would you manage this child?**

I always take a detailed history and perform a thorough examination. In the history, risk factors for developmental dysplasia of the hip (DDH) are important. These include (6 Fs):

1. First baby (the uterus is tighter and less elastic).
2. Female (lax ligament due to maternal hormones).
3. Family history (may be genetic predisposition).
4. Fetal malposition (breech presentation).
5. Fetal packaging disorders (oligohydramnios, twins, feet and neck torticollis).
6. LeFt side (60% left hip, 20% right and 20% both). May be related to the fetal position.

There are other risk factors of less importance and even the above risk factors vary in their importance.

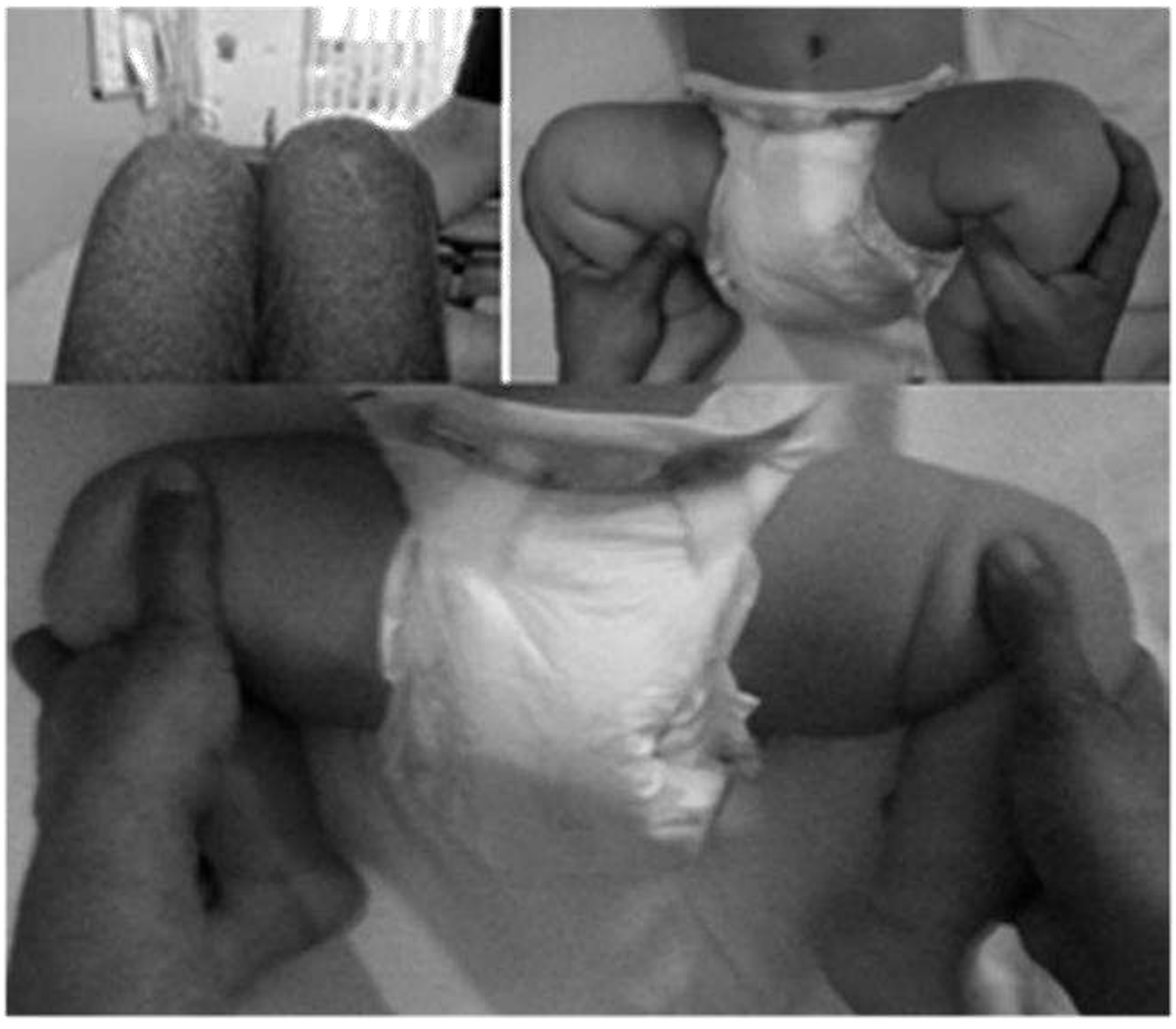


Figure 12.21 Clinical photographs of a 6-month-old child.

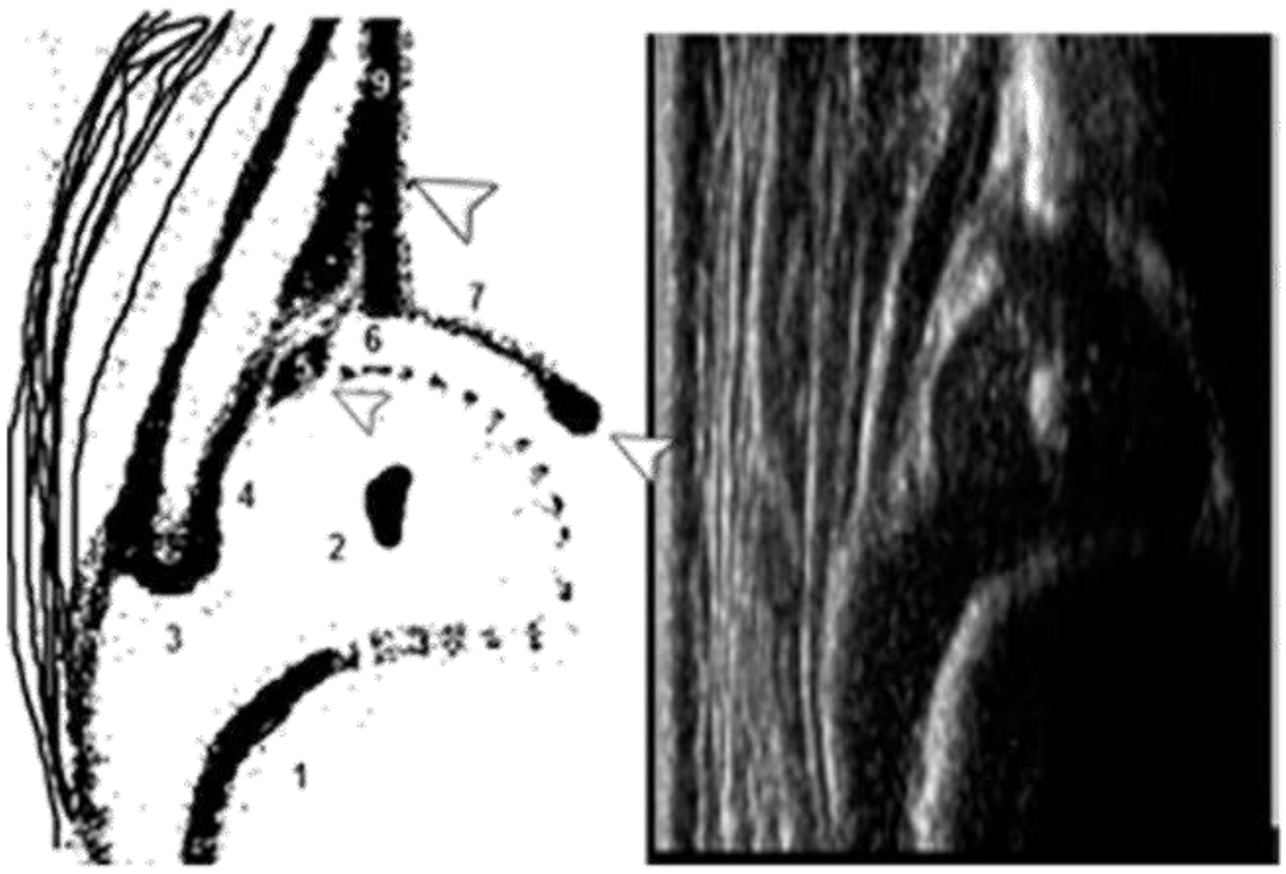


Figure 12.22 Infant hip ultrasound.

Family history and breech presentation are probably the most important.

Usually a child of this age is asymptomatic with no discomfort or pain. There may be asymmetrical skin fold (not very specific) or leg asymmetry as in Figure 12.21.

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. Barlow test is rarely positive after 10 weeks.

Q 29: How accurate are these tests?

Unfortunately, all these tests have limitations and the examiner should be aware of these. Bilateral limitation of abduction, asymmetrical skin crease and LLD are inaccurate in neonates. However, unilateral limited abduction has 70% sensitivity and 90% specificity for DDH in infants > 3 months.[16]

Ortolani and Barlow tests have a 60% sensitivity and 100% specificity in expert hands in comparison with ultrasound which has 90% sensitivity and specificity.[17]

Q 30: How does ultrasound help your diagnosis?

The role of ultrasound is well established in diagnosing and grading the DDH. There are two different techniques in use:

1. Graf: Static alpha and beta angles.[18]
2. Harcke: Dynamic provocation test.[19]

With a bit of experience, it is easy to identify the anatomical structures of infant hips and decide whether they are normal or not, and the severity of the dysplasia and dislocation.

The anatomical landmarks of normal infant hips in ultrasound include (Figure 12.22–12.27):

1. Chondro-osseous junction.
2. Femoral head.
3. Synovial fold.
4. Joint capsule.
5. Labrum.
6. Cartilage part of the roof. This is pliable and can be deformed with dislocation. Labrum and the cartilage part of the roof are sometimes collectively called limbus.
7. Bony part of the roof.
8. Bony rim (or the turning point between concavity and convexity of the roof).
9. Ilium.

These landmarks should be identified in the same sequence every time to enhance reproducibility.

Chapter 12: Children's orthopaedics

Table 12.5 Graf sonographic grading for developmental dysplasia of the hip.

Type	Alpha angle (α)			Beta angle (β)	Descriptions
I	> 60°			< 55° Ia > 55° Ib	Normal hip (at any age). This grade is further divided into Ia (β < 55°) and Ib (β > 55°). The significance of this subdivision is not yet established
II	50–59°	IIa		< 77°	If the child is < 3 months. This may be physiological and does not need treatment
		IIb		< 77°	>3 months, delayed ossification
	43–49°	IIc	Stable Unstable	< 77°	Critical zone, labrum not everted. This is further divided into stable and unstable by provocation test
D	43–49°			> 77°	This is the first stage where the hip becomes decentred (subluxed). It used to be called IId, but for the above reason, it is a stage on its own
III	< 43°	IIIa IIIb			Dislocated femoral head with the cartilaginous acetabular roof pushed **upwards**. This is further divided into IIIa and IIIb depending on the echogenicity of the hyaline cartilage of the acetabular roof (usually compared with the femoral head) which reflects the degenerative changes
IV	< 43°				Dislocated femoral head with the cartilaginous acetabular roof pushed **downwards**

Q 31: How would you treat a child with DDH?

The principles of treating DDH are:

1. Achieve a concentric reduction.
2. Maintain stability in the concentric reduction.
3. Promote the normal growth and development of the hip.
4. Minimize complications.

There are different methods to achieve the above principles depending on the child's age, reducibility of the dislocation and the availability of resources and expertise. There are accepted guidelines on managing children with DDH, who are grouped into the following age ranges:

1. Children from birth up to 6 months. (Figure 12.28.)
2. Children from 6 months to 18 months. (Figure 12.29.)
3. Children from 18 months to 30 months. (Figure 12.30.)
4. Children above 30 months. (Figure 12.31.)

The charts in Figures 12.28–12.31 summarize the treatment of DDH in different age groups.

Q 32: This is the sonogram of a 7-week-old child's hip? What can you see? (Figure 12.32.)

This is a sonogram of a newborn's hip. I can easily identify the chondro-osseous border and the femoral head and less easily the synovial fold. The capsule, labrum and the cartilaginous roof seem to be pushed down by the decentred femoral head (hence it is Graf IV). The bony roof is steep and shallow. In the picture on the right, the sonographers tried to quantify the dislocation by measuring α and β angles but I have a reservation on doing so for several reasons. The picture is not in the standard plane, and the cartilage roof line was not drawn correctly as it should pass through the labrum.

Q 33: What are your treatment options?

The options are:

1. Splinting the hip.
   i. Rigid such as Craig splint (Although user-friendly, it is out of favour. It may be useful in certain situations such as respiratory compromise.)
   ii. Dynamic splint such as Pavlik harness. (Table 12.6, Figure 12.33.)
   iii. Hip spica.

A Pavlik harness is my preferred option. It is sized by measuring the chest circumference. There are five sizes (premie, small, medium, large and extra-large). The harness has shoulder and leg straps. The anterior leg straps are to keep the hip flexion more than 90°

Section 4: Hand and Upper Limb/Children's Orthopaedics

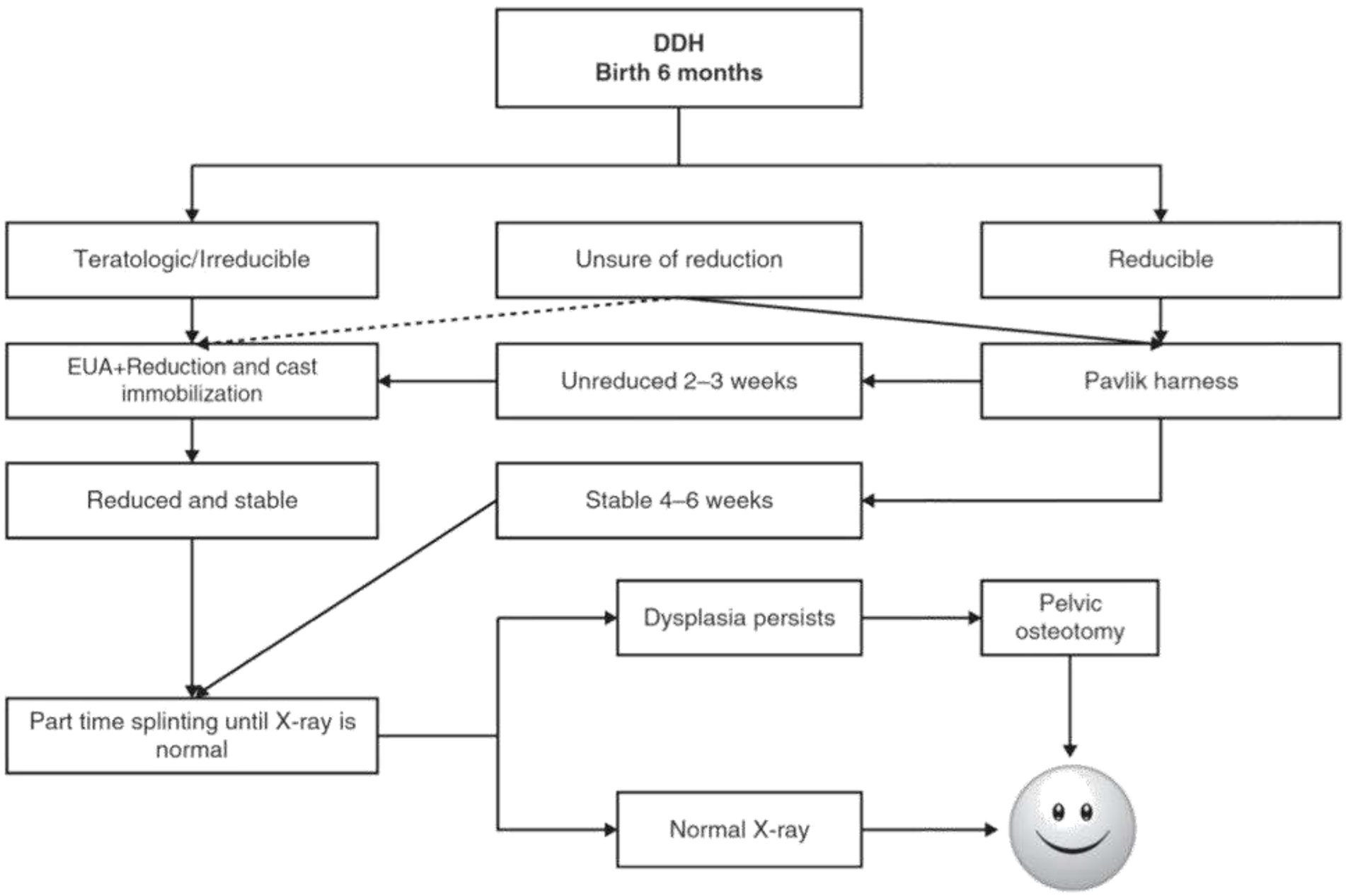


Figure 12.28 Developmental dysplasia of the hip (DDH) flow chart management (birth to 6 months).

while the posterior leg straps are to keep hip abduction in the safe zone. It allows motion within the range of stability. This motion is essential for stimulating the growth of the acetabulum. It is essential to do frequent checking of the child with a Pavlik harness (2–4 weeks) for the reduction, ultrasound progression, fitness (as the child may outgrow the harness) and to document active knee extension (functioning femoral nerve).

Q 34: Apart from DDH, are you aware of any other indication for a Pavlik harness?

Yes, femoral shaft fracture in infants. A retrospective study of 40 patients by Podeszwa *et al.* compared application of the Pavlik harness versus spica casting for the treatment of infant femoral shaft fracture.[21] No difference was found in radiographic outcomes, but approximately one-third of all spica patients experienced development of a skin complication. The authors conclude that all children younger than 1 year with a femoral shaft fracture should be considered for treatment with a Pavlik harness.

Q 35: This is a plain pelvis X-ray of a 7-month-old child. Her mother noted stiffness of the right leg when she applies a nappy. Can you tell me what might be wrong? (Figure 12.34.)

This plain radiograph of the pelvis shows the right acetabulum is shallow and there may be a false acetabulum a bit higher. Both femoral heads are not visible. They normally start ossifying by 8 weeks; however, there is normal variation. I would like to draw Hilgenreiner's line and Perkins' lines. I suspect the right femoral head (which is at the top of the femoral neck) lies in the upper lateral quadrant consistent with right hip dislocation.

Q 36: I agree, he has a dislocated right hip. How would you treat this child?

Provided there is no contraindication, I would take this child to theatre for EUA and try to reduce closed. If I am successful and the hip reduces satisfactorily, I would immobilize the hip in a hip spica. However, if I am not certain about the reduction, I will perform a hip arthrogram.

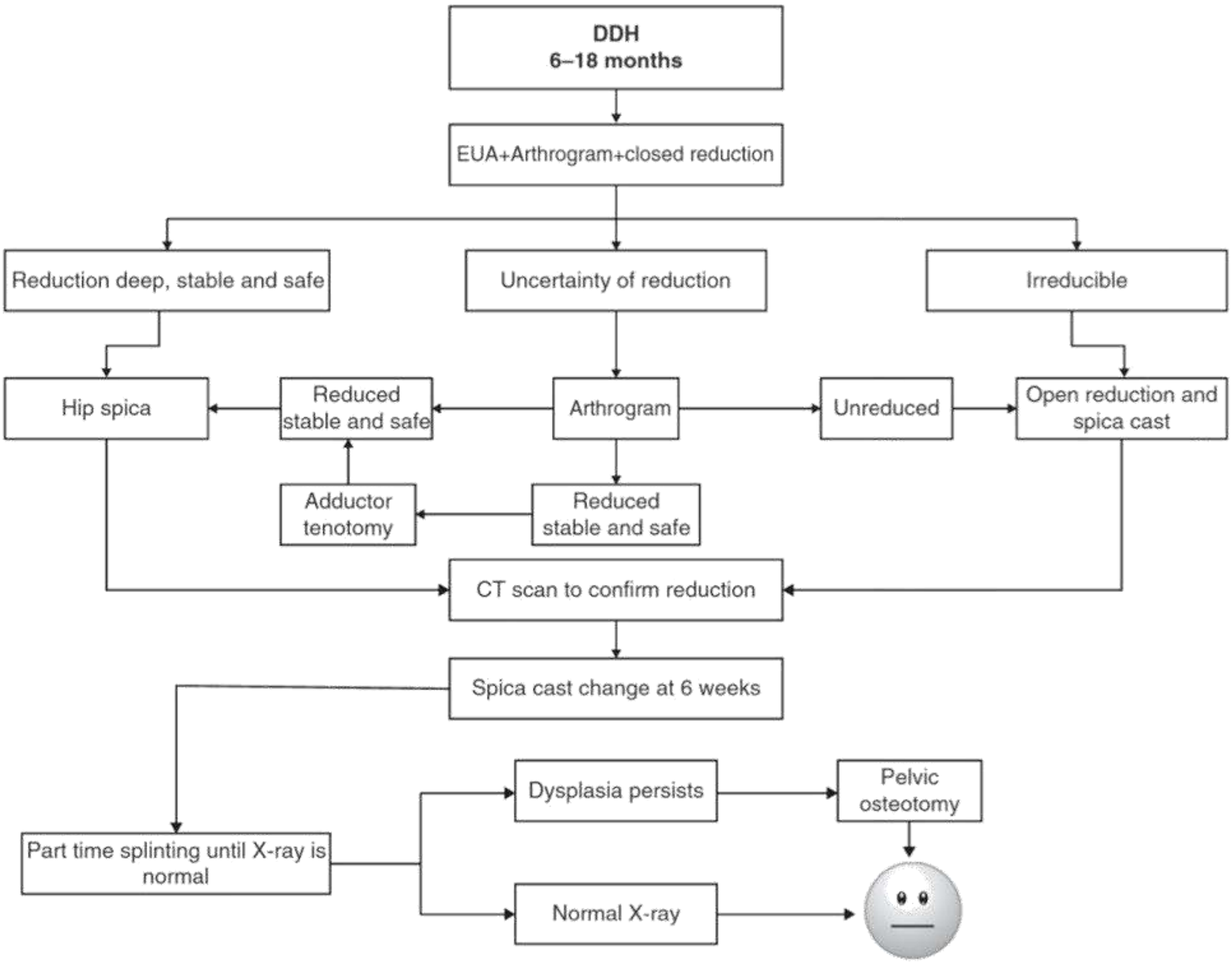


Figure 12.29 Developmental dysplasia of the hip (DDH) flow chart management (6–18 months).

Q 37: **These are the intraoperative hip arthrogram pictures. Do you want to comment on the findings?** (Figure 12.35.)

This arthrogram nicely shows the outline of the cartilaginous part of the femoral head. The capsule is distended. Pictures 2 and 3 show the hour-glass shape caused by the iliopsoas tendon (not visible) compressing the capsule in the middle. The top four pictures confirm hip dislocation where the head is not sitting in the acetabulum and there is significant medial dye pooling (> 7 mm). The ligamentum teres is thickened and elongated (best seen in picture 3). The surgeon seems to evaluate the hip reductions in different positions; I think the hip is shown to be reduced in pictures 6 and 7. Pictures 6 and 7 show the effect of rotation on reduction, where external rotation led to dislocation. If a satisfactory reduction (as shown in pictures 6 and 7) has been achieved within the safe zone and without extreme abduction or flexion, then the hip can be immobilized in a hip spica.

Q 38: **What would you have done if you could not have reduced the above hip closed?**

This will be an indication for open reduction (OR). Open reduction is indicated:

1. If the reduction is not possible.
2. If the reduction is not concentric.
3. If the hip reduced but remains unstable (narrow safe zone).
4. If stability can only be achieved by holding the hip in extreme abduction or internal rotation (high risk of AVN).

There is controversy on the best time to do the OR. Some delay it until the ossification nucleus of the

3. Interposed iliopsoas tendon.
4. Pulvinar (fibro-fatty tissues filling the acetabulum).
5. Capsule constriction.
6. Contracted transverse ligament giving the acetabular cartilage classic horseshoe shape.

Q 43: What is the limbus?

There is not a normal anatomical structure called limbus, but it is the name given to the labrum with the attached cartilaginous roof of the acetabulum moulded and pushed into the acetabulum preventing the femoral head reduction.

Q 44: So what would you do in the above situation?

I would proceed to OR through the anterior hip approach (Smith Petersen).

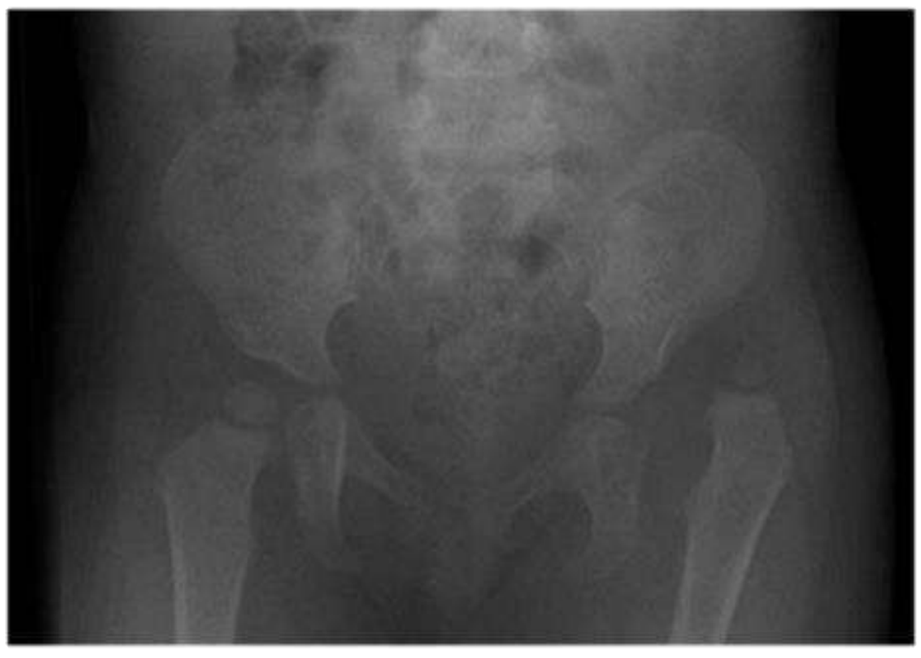


Figure 12.36 Plain pelvis X-ray of the hip of a 19-month-old child.

Q 45: This is a pelvis X-ray of a child with cerebral palsy. Can you describe the main problem and how you would treat it? (Figure 12.39.)

There is bilateral subluxation of the hips with Reimer's migration index (RIM) of more than 75% (Figure 12.40); bilateral coxa valga and femoral anteversion. Although the acetabuli look reasonable there might be deficient posterior wall which is common in these patients. Ideally, this should have been dealt with before it reached this advanced stage.

Q 46: How would you manage this child?

Patients with cerebral palsy (CP) should be managed by a multidisciplinary team including a paediatrician, physiotherapist, orthotist and paediatric orthopaedic surgeon as well as the family. Thorough history and assessment are essential with particular focus on current symptoms, mobility and other comorbidities that may preclude general anaesthesia.

This child needs substantive tissue release (adductors and iliopsoas muscles) with exploration of the hip joint. If the articular cartilage is healthy and not damaged, the hip should be reduced. Femoral (varus derotation osteotomy; VDRO) and pelvis osteotomy (Dega) are often necessary to achieve concentric stable reduction. Table 12.7 summarizes general recommendations for treating neurogenic hip dislocation. There is some controversy about certain aspects.

Q 47: A 6-year-old boy is referred to you with a painful right hip and limping. No history

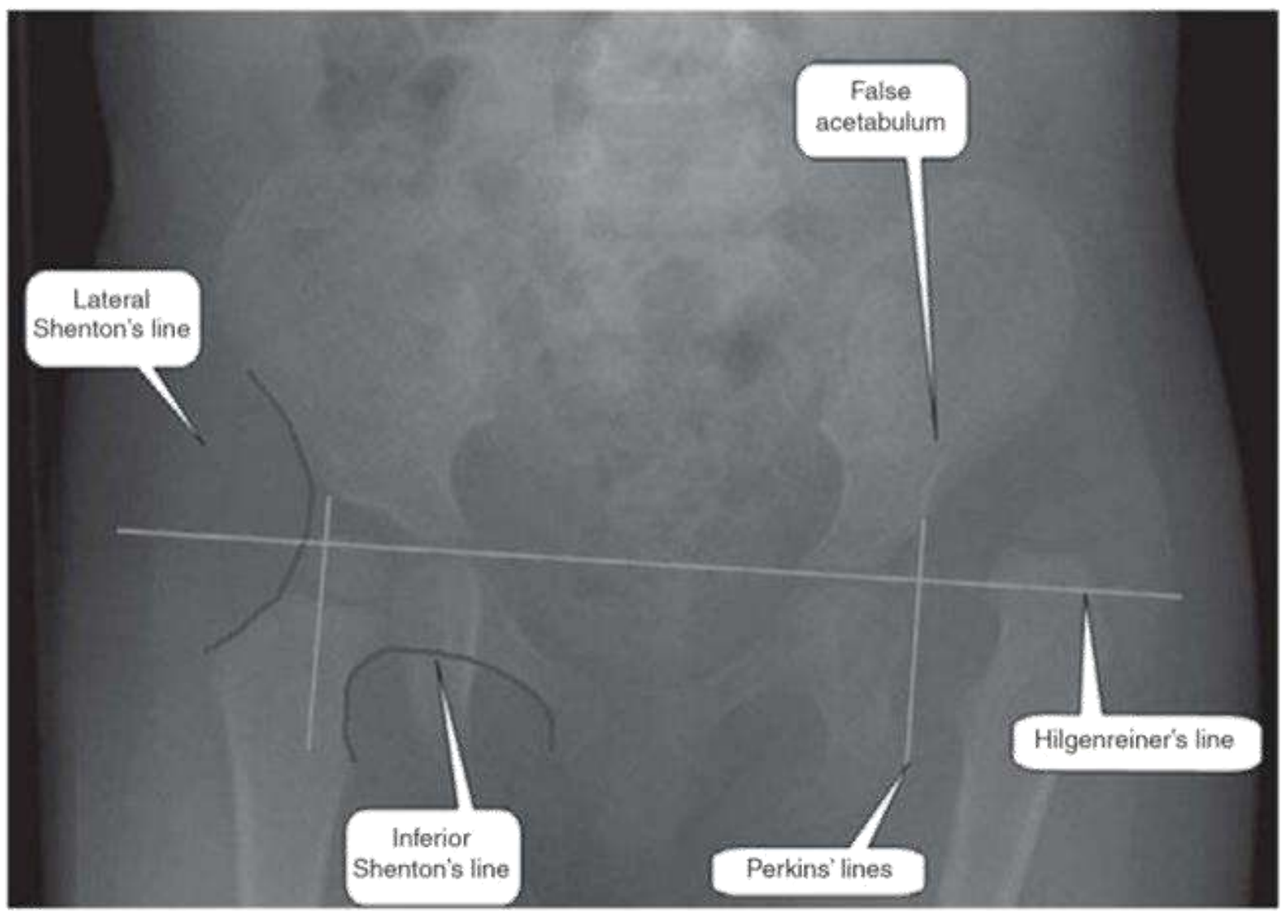


Figure 12.37 Pelvis X-ray of the hip of a 19-month-old child.

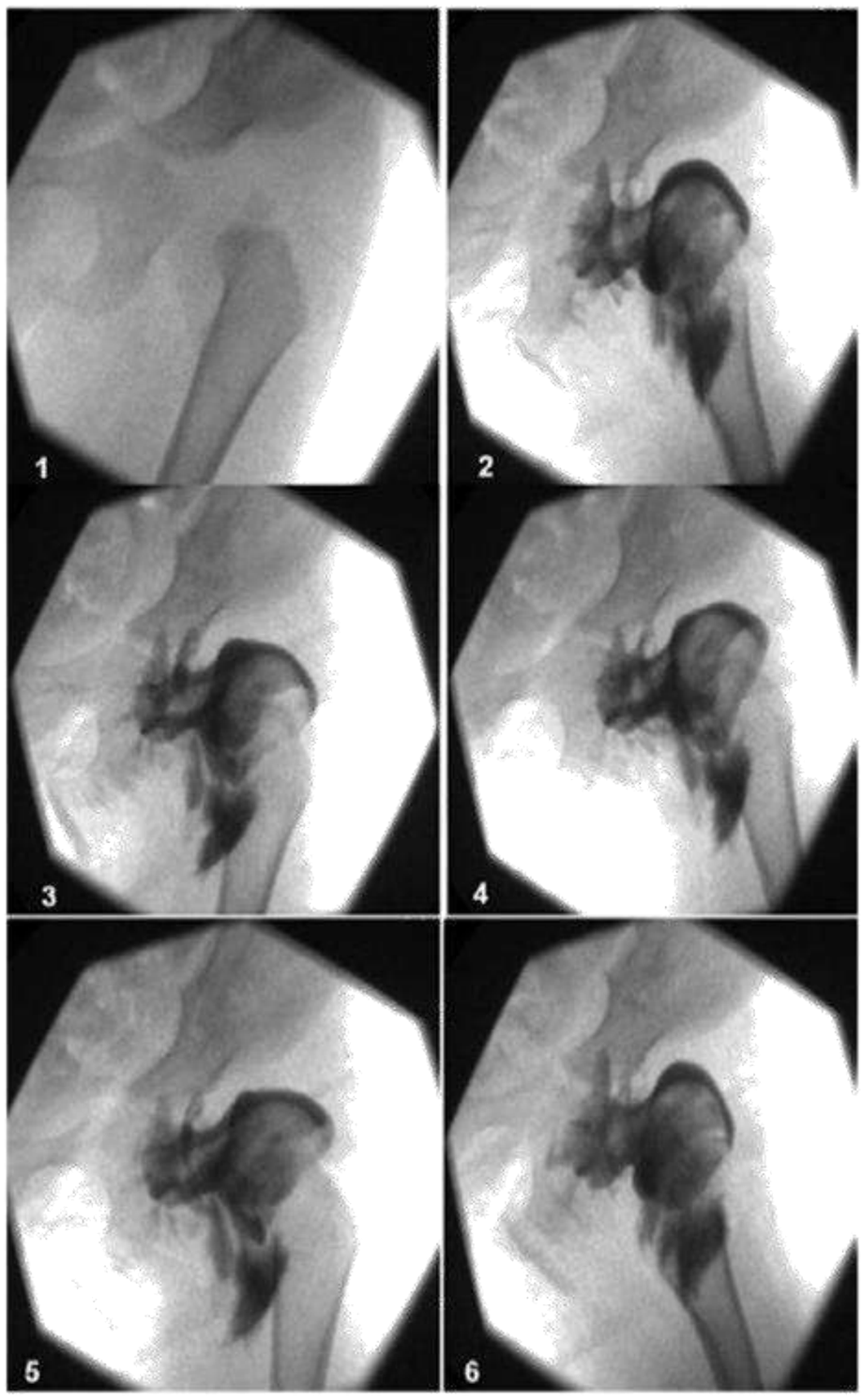


Figure 12.38 Hip arthrogram of a 19-month-old child.

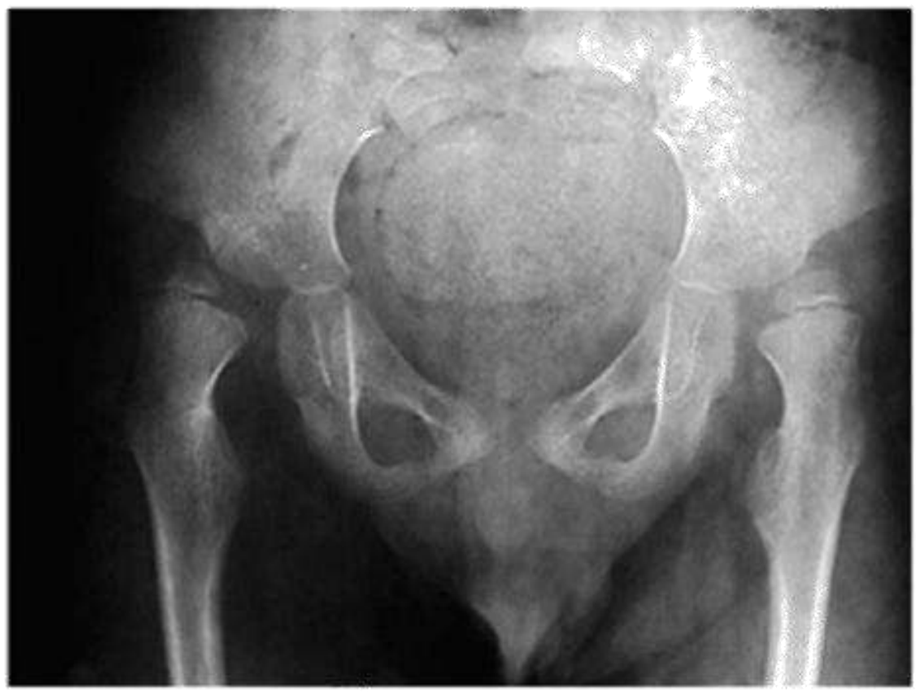


Figure 12.39 Pelvis X-ray of a child with CP.

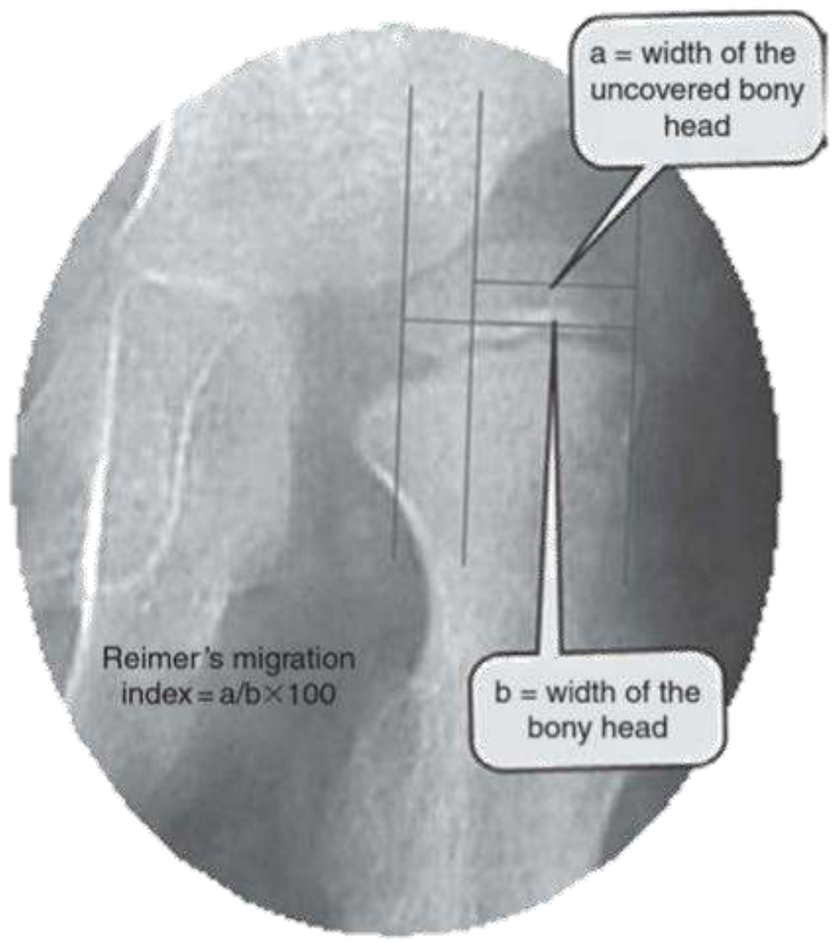


Figure 12.40 Reimer's migration index.

of trauma and he was previously fit and healthy. How would you approach such a patient?

I would like to know more (any constitutional symptoms, recent upper respiratory infection, previous similar episodes, involvement of other joints, contact with unwell children, etc.). Then I would perform a thorough examination (temperature, how unwell the child looks, walking, the involved limb and joints and other limbs and joints etc.). I would arrange for appropriate investigations. In such a situation, I usually request blood tests (FBC, CRP, ESR and blood culture if there is fever or the child looks unwell) and pelvis X-ray (AP and lateral).

Q 48: What goes through your mind?

This is quite a common scenario and most of the time, the diagnosis is irritable hip or transient synovitis which is a benign condition; however, I do not want to miss serious conditions such as septic arthritis, osteomyelitis or tumours.

Q 49: The child does not have a temperature, WCC is 10.4, ESR and CRP are 20 and the X-ray did not show anything abnormal. What would you do?

This is reassuring result and fits in with irritable hip. According to Kocher's criteria (Table 12.8), infection is very unlikely (but not impossible). My treatment will be symptomatic with analgesia, anti-inflammatory (not aspirin due to the possible link with Reyes' syndrome), antihistamine but no antibiotic. Depending on few logistic considerations, I may discharge this child home with 48-hour open access to come back to the ward if things deteriorate, otherwise I will review

Table 12.7 Management of neurogenic hip dislocation.

Severity	Passive hip abduction < 30°	Subluxation (RIM 20–40%) No coxa valga No acetabular dysplasia	Subluxation (RIM 40–60%) ± Coxa valga No acetabular dysplasia	Subluxation (RIM 40–60%) ± Coxa valga + Acetabular dysplasia	Subluxation (RIM > 60%) OR Dislocation No pain	Severe painful subluxation or dislocation
Interventions	Adductor release + Physiotherapy	Adductor release + Intramuscular release of psoas at pelvic brim + Physiotherapy	Adductor release + Intramuscular release of psoas at pelvic brim (in ambulant child) OR from lesser trochanter in non-ambulant child + Femoral osteotomy + Physiotherapy	Adductor release + Intramuscular release of psoas at pelvic brim (in ambulant child) OR from lesser trochanter in non-ambulant child + Femoral osteotomy + Acetabular augmentation if acetabulum is small or Dega osteotomy if acetabulum is large + Physiotherapy	Arthrotomy + If articular cartilage is healthy, then open reduction and treat as in column 3 or 4	Excision of the femoral head with valgus subtrochanteric osteotomy and muscle interposition

Table 12.8 Kocher's four criteria.[22]

Kocher's four criteria	Significance
Non-weightbearing ESR > 40 mm/hour (or CRP >20)[23] Fever (> 38.5) WBC > 12 000/mm^3	Four criteria met: 99% septic arthritis Three criteria met: 93% septic arthritis Two criteria met: 40% septic arthritis One criterion met: 3% septic arthritis

him in clinic in 1 week. For example, if the parents are sensible, do not live far away and have a means of transport, this is a practical approach.

Q 50: **What is the difference in the synovial fluid analysis between infection and non-infectious conditions?**

Table 12.9 Joint fluid analysis.

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 is low in RA but normal in AS
Infectious	> 80 000	> 75	Thick, cloudy fluid + Gram stain + Cultures ↓ Joint aspirate glucose ↑ joint aspirate protein

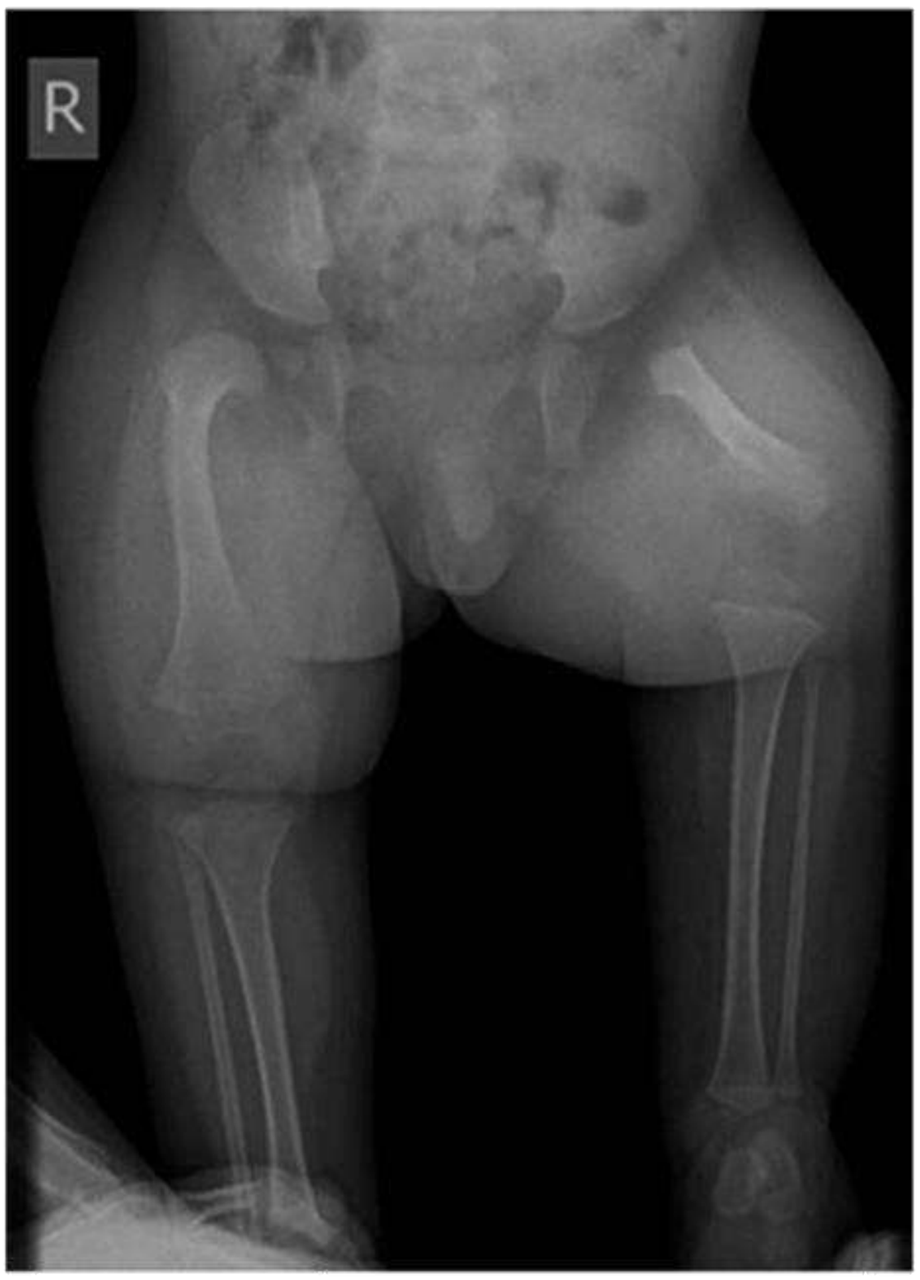


Figure 12.41 A child with short limb.

Q 51: **This is an X-ray of a child with a short lower limb. Describe what you see.** (Figure 12.41.)

This X-ray shows the left femur is short and dysplastic. The proximal parts (head and trochanter) are not visible; features consistent with proximal focal femoral deficiency (PFFD). There is coxa vara of the right side indicating the right side may be affected as well (bilateral involvement in 15%). The left fibula seems to be shorter than the right side raising the possibility of fibular hemimelia (two-thirds of patients). It is difficult to comment on the state of the hip and knee joints from a single plain film and further assessment is required.

Q 52: **How would you approach such a patient?**

Treatment of this condition is a challenge and it should be undertaken in specialized centres with interest and experience in its treatment. The National Institute for Health and Clinical Excellence (NICE) has issued guidance to the NHS hospitals on combined bony and soft tissue reconstruction for PFFD (IPG297 on 22 April 2009). Treatment must be tailored to individual patients based on LLD, hip and knee stability, femoral rotation, proximal musculature, foot condition, availability of the expertise and patient and family motivation. The two broad options are reconstructive surgery or amputation and prosthetic replacement.

A few classifications have been advised to aid assessment and treatment:

1. **Gillespie and Torode**
   a. Group A: congenital short femur by about 20%. The child can weight bear on the affected leg.
   b. Group B: The LLD is around 40% and there is anterior projection of the thigh and flexed knee.
   c. Group C: The thigh is short and bulbous and the leg is externally rotated with the foot at or near the level of the other knee.
2. **Aitken's classification**

Treatment recommendations: Reconstruction and lengthening if femoral length $>50\%$ (usually grade A and B where there is a head). Amputation, fusion, Van Ness rotational arthroplasty if the femoral length $<50\%$.

3. **Paley classification**

This is the most recent and comprehensive classification, however it is still not widely adopted.

Type I: Intact femur with mobile hip and knee.

a. Normal ossification proximal femur.
b. Delayed ossification proximal femur.

Type II: Mobile pseudarthrosis (hip not fully formed, a false joint) with mobile knee.

a. Femoral head mobile in acetabulum.
b. Femoral head absent or stiff in acetabulum.

Type III: Diaphyseal deficiency of femur (femur does not reach the acetabulum).

a. Knee motion $>45°$.
b. Knee motion $<45°$.

Type I is further subclassified into:

0. ready for surgery; no factors to correct before lengthening.
1. One factor to correct before lengthening.
2. Two factors to correct before lengthening.
3. Three factors to correct before lengthening.

4+. etc.

- Patient's perception of discrepancy.
  - How tall is the child? Is the child taller or shorter than average?
  - How tall are the parents or family members?

Station 2: Paediatric knees

Q 1: This is the clinical photograph of a 2-year-old child who was referred because of bowed legs. How would you approach such a child? (Figure 12.42.)

Bowed legs and knocked knees are common referrals to children's orthopaedics. Most are physiological (i.e. normal for the age of the child), however, this must be differentiated from pathological causes (Table 12.12).

The average child is born with genu varum of 15° which decreases through infancy. The legs are straight at some point in the second year then go into progressive valgus reaching maximum valgus of average 10° at around 3–4 years of age. Valgus then gradually decreases reaching the adult value (5° of valgus) at around age 8 (see Selenius curve; Figure 12.43).

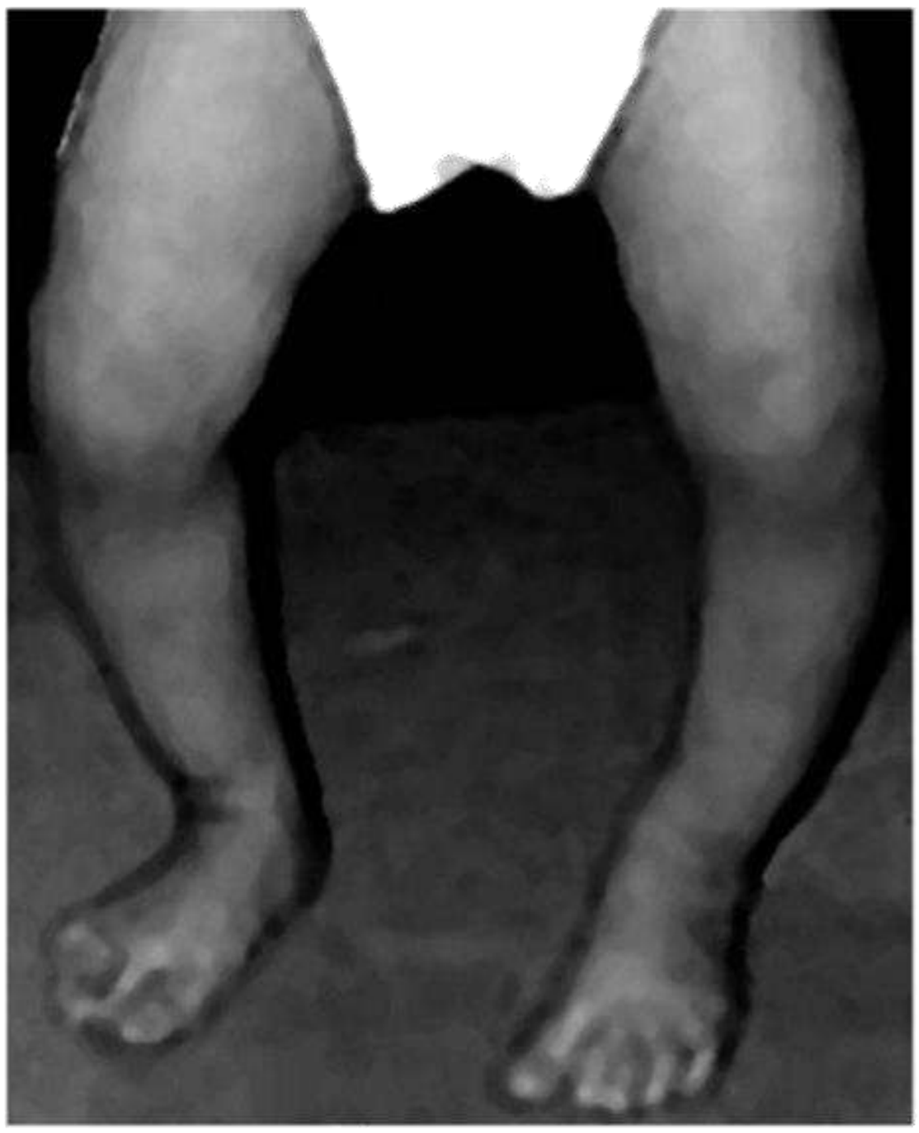


Figure 12.42 A child with bowed legs.

Q 2: So how can you tell whether the bowing is physiological or pathological?

In most cases, history, examination and appropriate investigations are adequate to differentiate between pathological and physiological bowing. However, sometimes it is not possible to be certain whether it is physiological or pathological and follow-up becomes necessary. It is important to win parent confidence. Reassuring them without adequate explanation and a future plan may not be enough.

Table 12.12 Causes of genu varum (bow legs) and genu valgus (knocked knees).

Bowed leg	Knocked knees
Physiological	Physiological
Tumours such as osteochondromas	Tumours such as osteochondromas
Skeletal dysplasia	Skeletal dysplasia
Blount's disease	Primary tibia valga
Infection	Infection
Trauma	Trauma
Metabolic (vitamin D deficiency, fluoride poisoning)	Renal osteodystrophy
Osteogenesis imperfecta	Neuromuscular disease (polio) and tight iliotibial band

Genu varum is more likely to be pathological if it is:

1. Present after 2 years.
2. Unilateral or with asymmetry of more than 5°.
3. Associated with shortening of the limb (or stature).
4. Severe (beyond 2 SD of the mean as per Selenius chart; SD = 8°).
5. In a child with obesity.

And genu valgus is more likely to be pathological if it is:

1. Severe (intermalleolar distance > 10 cm at 10 years or > 15 cm at 5 years).
2. Unilateral.

The following radiological assessment can be very helpful:

1. Tibiofemoral angle as per Selenius curve.
2. Metaphyseal–diaphyseal angle of Levine and Drennan (normal < 11°, abnormal > 16°).
3. Metaphyseal–epiphyseal angle (normal < 20°).

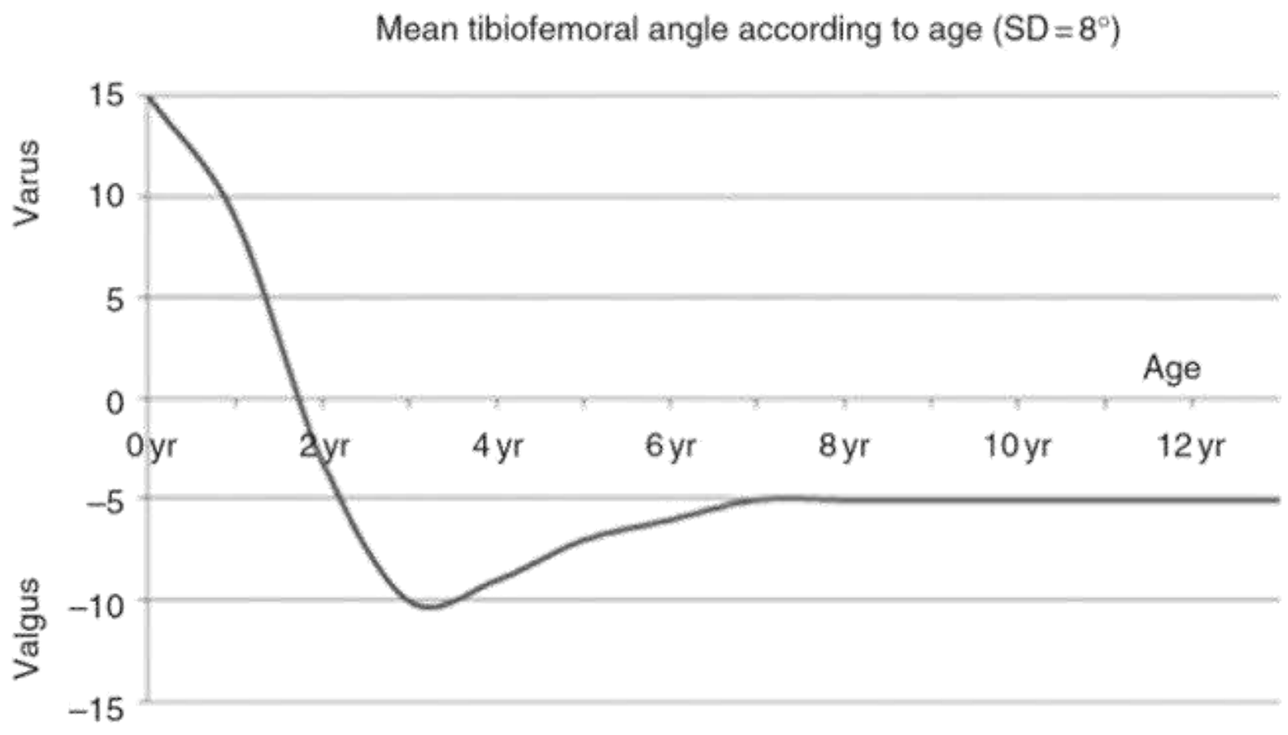


Figure 12.43 Selenius curve.

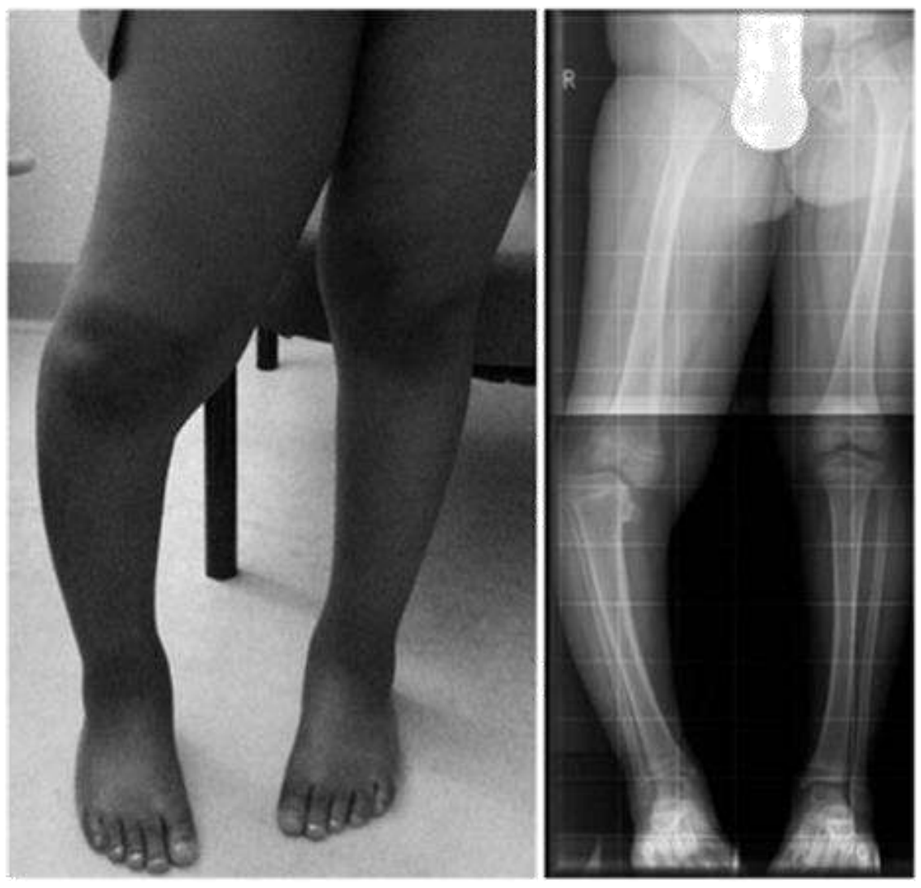


Figure 12.44 Clinical photograph and X-ray of a 7-year-old African child.

Q 3: **This is a clinical photograph and X-ray of a 7-year-old African child with right knee deformity. What do you think the diagnosis is?** (Figure 12.44.)

There is an obvious deformity of the right leg, specifically around the knee. The child looks big. The X-ray shows classic features of Blount's disease (idiopathic tibia vara) which is medial physeal closure of the proximal tibia with varus (and internal rotation) deformity – Type VI.

Q 4: **What is Blount's disease?**

Blount's disease is an uncommon growth disorder characterized by disordered ossification of the medial aspect of the proximal tibial physis, epiphysis and metaphysis. This is probably caused by a combination of excessive compressive forces on the proximal medial metaphysis of the tibia and altered enchondral bone formation. There are two recognized types:

1. Infantile (0–4).
2. Adolescent (> 10 years).

In infantile tibia vara, patients generally start to walk early (9–10 months); it is more prevalent in females, blacks, and those with marked obesity. It is bilateral in approximately 80% of cases and associated with a prominent metaphyseal beak, internal tibial torsion, and LLD. The deformity is usually 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.

Q 5: **Can you draw the following angles? And tell me the significance of these angles?**

1. Tibiofemoral angle.
2. Metaphyseal–diaphyseal angle of Levine and Drennan.
3. Metaphyseal–epiphyseal angle.

The tibiofemoral angle is 20° varus. In a 7-year-old child, the tibiofemoral angle approaches the mean adult value which is 5° valgus; hence there is 25° total varus deformity. This is an extreme deformity even after allowing for the quoted standard deviation of 8°. Drennan angle is 21° (normal value is 11° and above

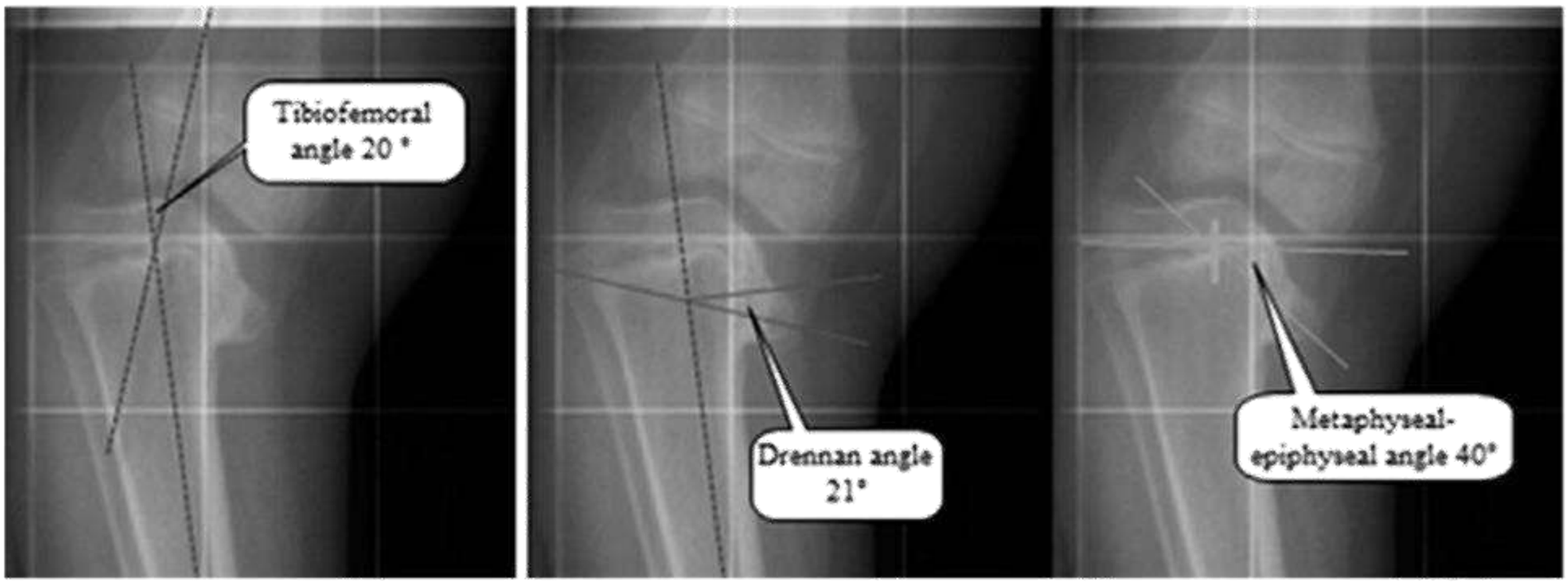


Figure 12.45 Radiological measurement in Blount's disease.

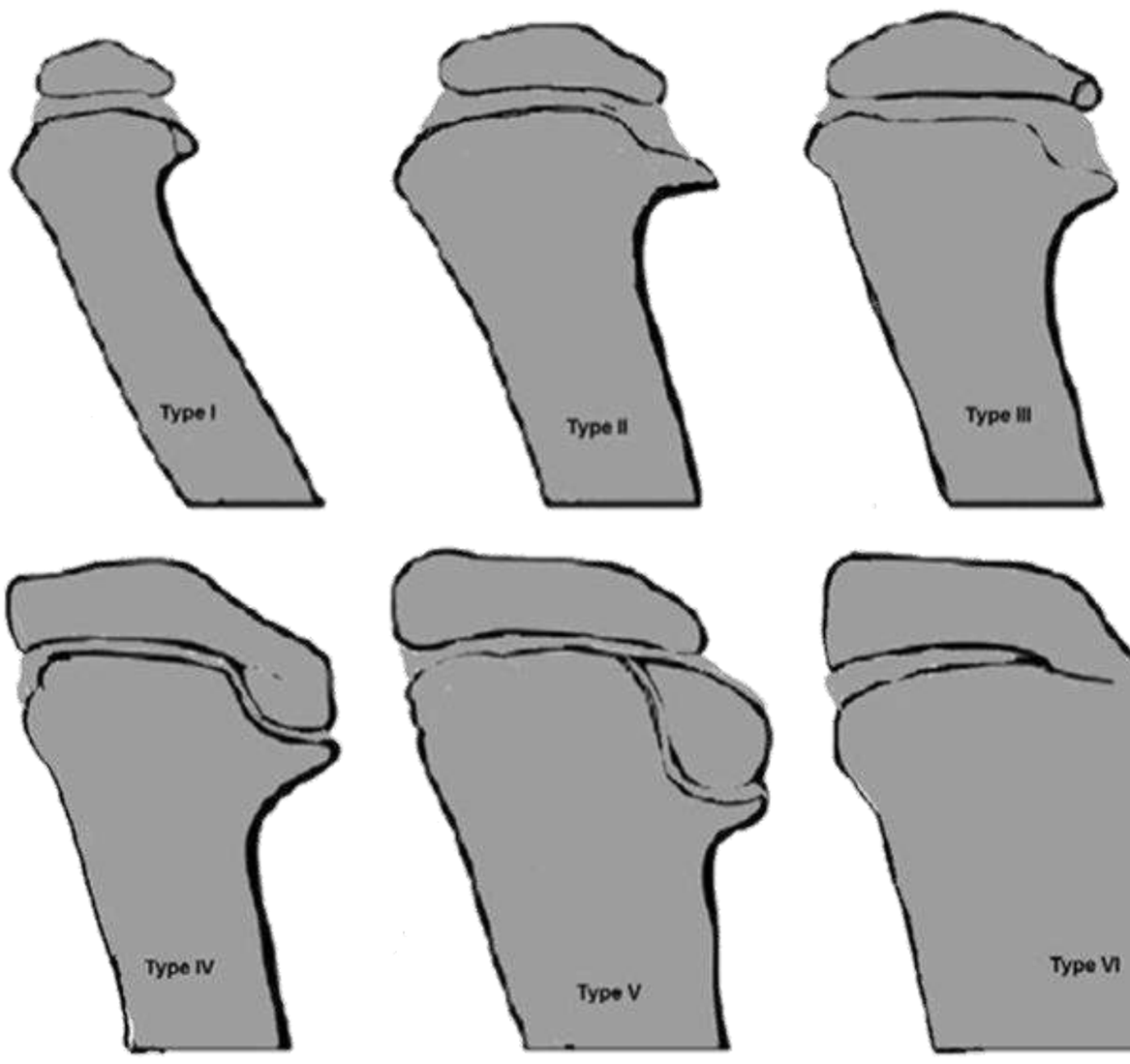


Figure 12.46 Langenskiold classification of Blount's disease.

16° is considered abnormal) which reflects varus deformity of the proximal tibia rather than the knee joint. The same can be said for the metaphyseal–epiphyseal angle.

Q 6: Are you aware of any classification of this condition?

Langenskiold proposed a six-stage radiological classification (Figure 12.46). This classification was not intended for use in determining the prognosis or the most desirable type of treatment. However, it is generally accepted that surgical treatment commonly is needed for any child with stage 3–6 changes (Table 12.13).

Q 7: So what would you offer this girl?

This girl has stage 6 so surgical treatment is indicated. She has 25° of varus deformity so epiphysiodesis alone will not be adequate (usually correct 1°/month); moreover, the medial physis has been closed so you cannot

Table 12.13 Langenskiold classification of Blount's disease.

Stages	Descriptions	Treatment
Stage 1	Medial beaking, irregular medial ossification with protrusion of the metaphysis	Orthotic for < 3 years
Stage 2	Cartilage fills depression. Progressive depression of medial epiphysis with the epiphysis sloping medially as disease progress	Failure of full correction or progression to type III → surgery
Stage 3	Ossification of the inferomedial corner of the epiphysis	Surgery
Stage 4	Epiphyseal ossification filling the metaphyseal depression	
Stage 5	Double epiphyseal plate (cleft separating two epiphyses)	
Stage 6	Medial physeal closure	

rely on medial growth even after epiphysiolysis. So the only option left is proximal tibial osteotomy and either acute correction using internal fixation or gradual correction using an external fixator.

Station 3: Paediatric feet

Q 1: This is a clinical photograph of a newborn's feet. What can you see? (Figure 12.47.)

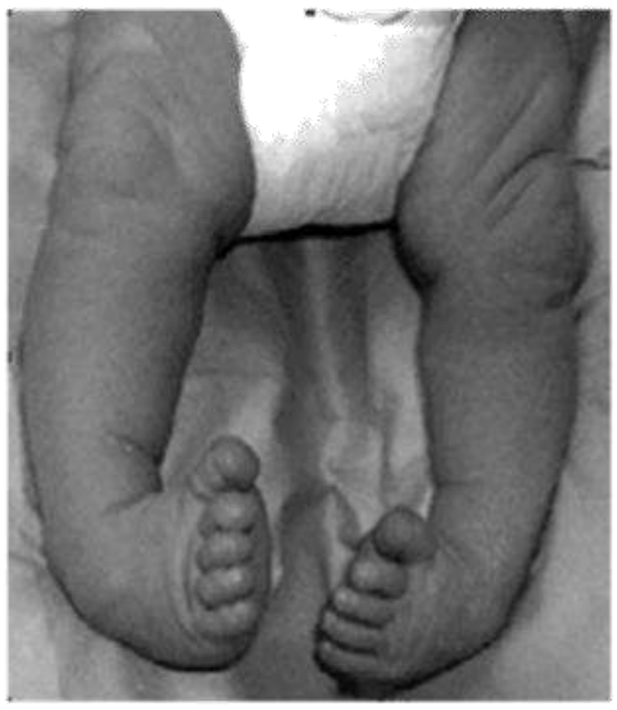


Figure 12.47 A clinical photograph of a newborn's feet (picture courtesy of Dr Lynn Staheli and Global Help Publication).[24]

This photograph shows the classic feature of bilateral club feet. The deformity is quite complex involving the ankle, subtalar and midtarsal joints. The ankles are usually in equinus. The hind feet are in varus. This is produced by a coupled inversion and adduction deformity at the subtalar joints. The forefeet are adducted and plantar-flexed in relation to the hindfoot.

Q 2: Tell me about the causes of club feet. Why was this child born with club feet?

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] A 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.
7. Multifactorial:
   a. Common in Polynesian and rare in the Japanese race.
   b. Not more common in consanguinity.
   c. 10% risk if first-degree relative affected – combination of environmental/genetic factors.[27]
   d. 25% have a family history.

Although less common than idiopathic, there are identifiable causes for club feet which need to be excluded. These include the following:

1. Neurological causes: spina bifida (myelomeningocoele), polio, cerebral palsy.
2. Sacral agenesis.
3. Fetal alcohol syndrome.
4. Congenital myopathy.

5. Down syndrome (may include vertical talus).
6. Arthrogryposis.
7. Hand anomalies (Streeter's dysplasia/ constriction band syndrome).
8. Diastrophic dwarfism.
9. Prune belly.
10. Tibial hemimelia.

Q 3: How would you manage this child?

Having established the diagnosis of idiopathic club feet, my management is serial casting by the Ponseti method. The treatment should be started as early as possible; the severity of the deformity is quantified using Pirani score, then serial casting weekly for up to 3 months.

Sequence of deformity corrections (**CAVE**):

1. **C**avus.
2. **A**dduction of the forefeet and **V**arus of the heel. This is achieved concomitantly using the talus head as a fulcrum.
3. **E**quinus of the heel. Tendo-Achilles tenotomy is required in 90% of the patients to correct the heel equinus.

This can be repeated if necessary and can be utilized up to the age of 1 year. Successful correction is followed by a regime of using Denise-Browne boots continuously for 3 months, after which they will be used at nap and night time for 3 years.

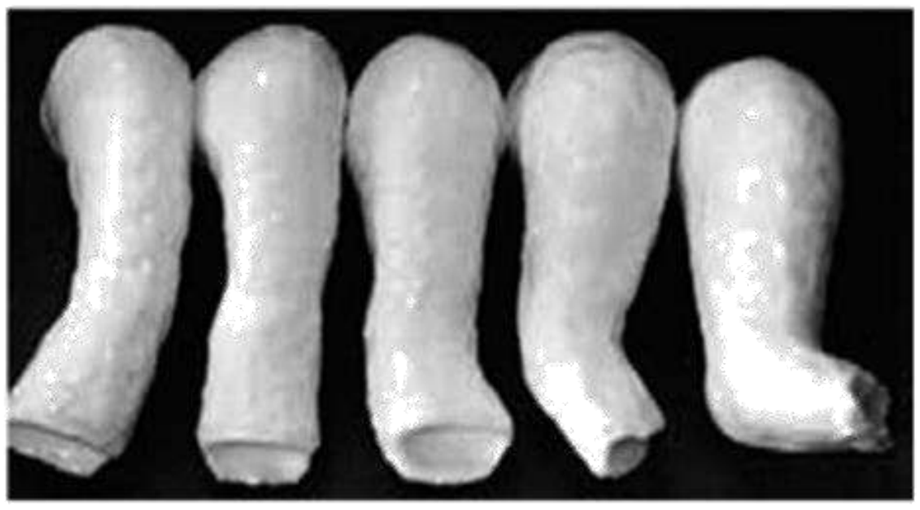


Figure 12.48 Ponseti's weekly serial casting (picture courtesy of Dr Lynn Staheli and Global Help Publication).[24]

Q 4: These are Denise–Browne boots. Can you describe the different parts and their functions? (Figure 12.49.)

The brace consists of open-toe high-top straight-last shoes attached to a bar. The bar should be of sufficient length so that the heels of the shoes are at shoulder width. This can be adjusted using the sliding clamp in the middle. The bar should be bent 5–10° to hold the feet in dorsiflexion.

For unilateral cases, the brace is set at 60–70° of external rotation on the clubfoot side and 30–40° of external rotation on the normal side. In bilateral cases, it is set at 70° of external rotation on each side.

Q 5: Explain the Pirani score for club feet.

The Pirani score is simple and reproducible. It uses six clinical signs to quantify severity of each component of the deformity. Each component is scored as 0 (normal), 0.5 (mildly abnormal) or 1 (severely abnormal) (Table 12.14).

The six clinical signs Pirani used are divided equally between the hindfoot and midfoot and then added for a total score of 0–6:

Hindfoot contracture score (HFCS) 0–3

1. Equinus.
2. Deep posterior crease.
3. Empty heel.

Midfoot contracture score (MFCS) 0–3

1. Curved lateral border.
2. Medial crease.
3. Lateral head of talus.

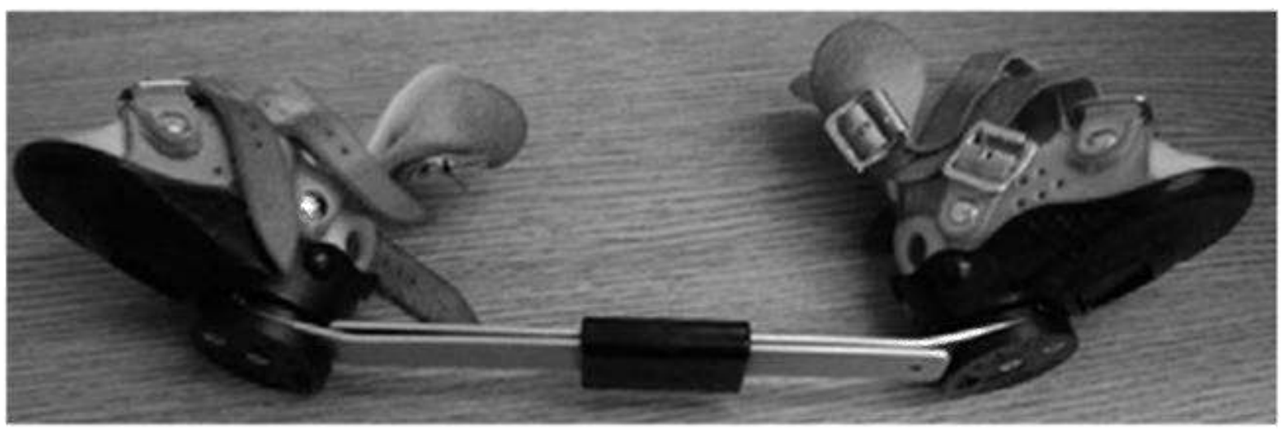


Figure 12.49 Denise–Browne boots.

Table 12.14 Pirani score for club feet. (Pictures courtesy of Dr Lynn Staheli and Global Help Publication.)

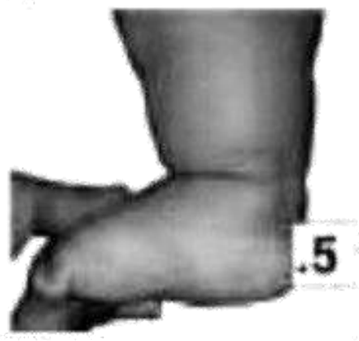
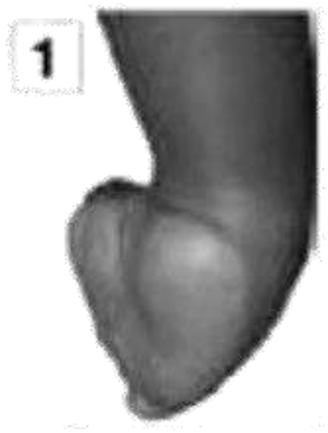
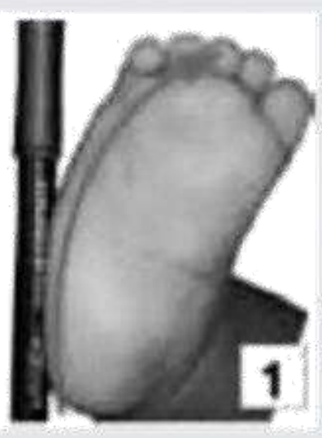
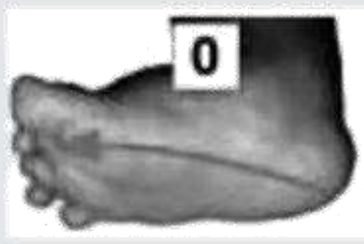
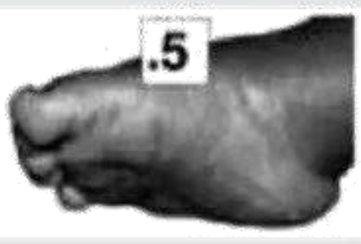
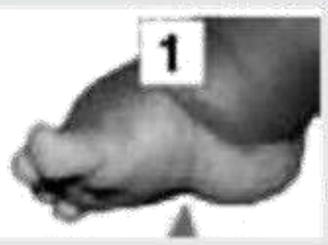
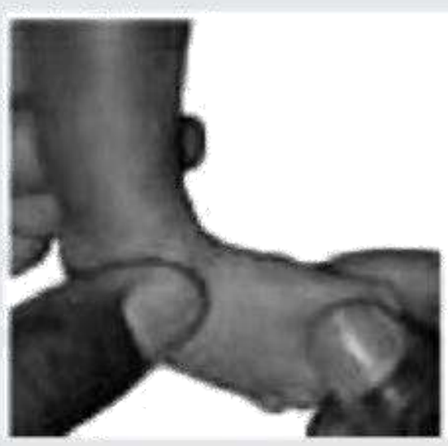
		0	0.5	1.0
Hindfoot contracture score	Equinus			
	Deep posterior crease			
	Empty heel		**Empty Heel** **Easily Palpable 0** **Palpable Deep .5** **Not Palpable 1**	
Midfoot contracture score	Curved lateral border			
	Medial crease			
	Lateral head of talus			**Talar Head** **None 0** **Partial .5** **Full 1**

Table 12.15 Dimeglio scoring system.

Rating	4	3	2	1	0
1. Equinus	45°–90° Plantar flexion	20°–45° Plantar flexion	20°–0° Plantar flexion	0°–20° Dorsiflexion	>+20° Dorsiflexion
2. Varus	45°–90° Varus	20°–45° Varus	20°–0° Varus	0°–20° Valgus	> 20° Valgus
3. Supination	45°–90° Supination	20°–45° Supination	20°–0° Supination	0°–20° Supination	>20° Supination
4. Adductus	45°–90° Adduction	20°–45° Adduction	20°–0° Adduction	0°>–<20° Adduction	> 20° Abduction
5. Posterior crease			Yes		No
6. Medial crease			Yes		No
7. Cavus			Yes		No
8. Deviant muscle function			Yes		No

Q 6: Do you know any other scoring system for club feet?

Yes, there is the Dimeglio classification which consists of eight items. Scorings for four items range from 0–4 (best to worst). Four items only score 0 or 1. Total score ranges between 20–0 (very severe: 16–20, severe 11–15, moderate 6–10, and postural 0–5). (Table 12.15.)

Q 7: This is another child who was born with bilateral club feet. Have a look at the picture and tell us how you would treat him. (Figure 12.50.)

The striking feature of this child is the lack of skin creases over the joints (hips, knees and elbows) which is very suggestive of arthrogryposis. As far as the club feet are concerned, I would still treat them as idiopathic club feet. Researchers from SickKids hospital in Toronto studied 40 non-idiopathic club feet and compared them with 249 idiopathic club feet and showed the success rate of Ponseti serial casting in the former group is less successful (10% failure rate; 4/40) and recurrence rate is higher (44%; 16/36), and more patients needed additional operations.[28] Nevertheless, correction was achieved and maintained in most patients.

Q 8: What other complications of Ponseti treatment are there?

In contrast to the above example, some patients may develop excessive heel valgus and external tibial torsion while using the brace. In such instances, the external rotation of the shoes on the bar should be reduced from approximately 70° to 40°.

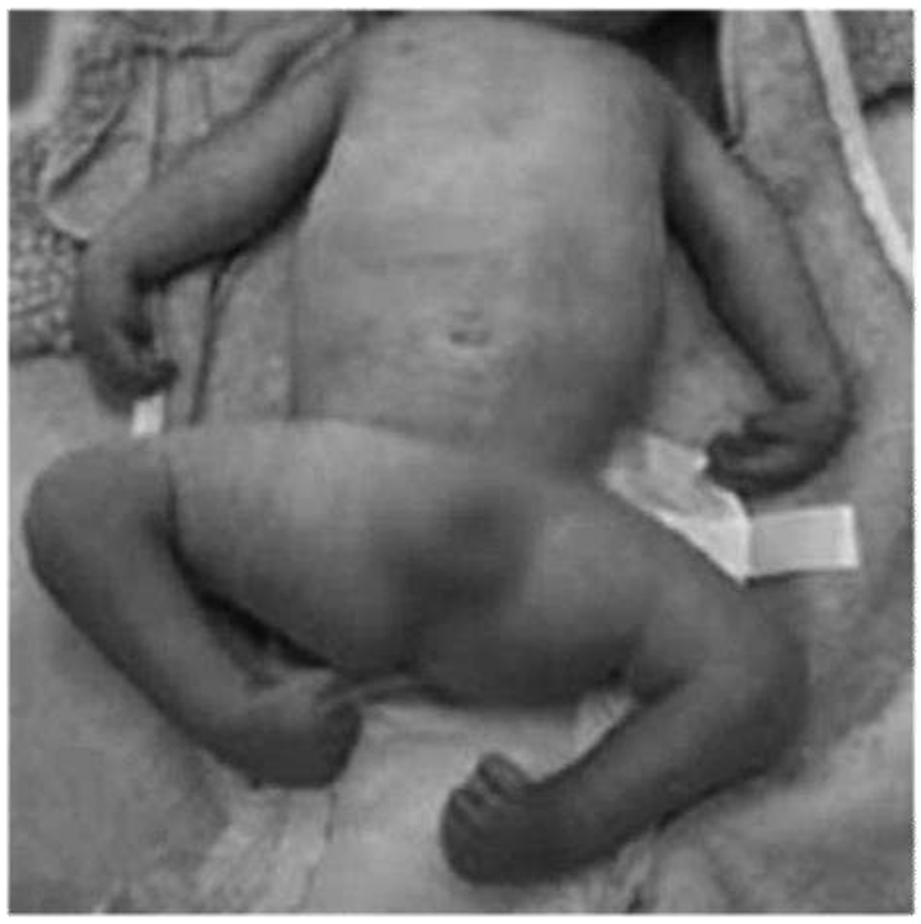


Figure 12.50 Bilateral club feet (picture courtesy of Dr Lynn Staheli and Global Help Publication).[24]

Q 9: These are lateral views of the left and right feet of a child who was brought to your clinic with a right foot deformity. What can you see? (Figure 12.51.)

These are weightbearing views of both feet. There is a vertical orientation (i.e. the talus is plantar flexed) of the talus of the right foot and the long axis of the first metatarsal is not in line with the navicular bone and the long axis of the talus. In comparison, the talus of

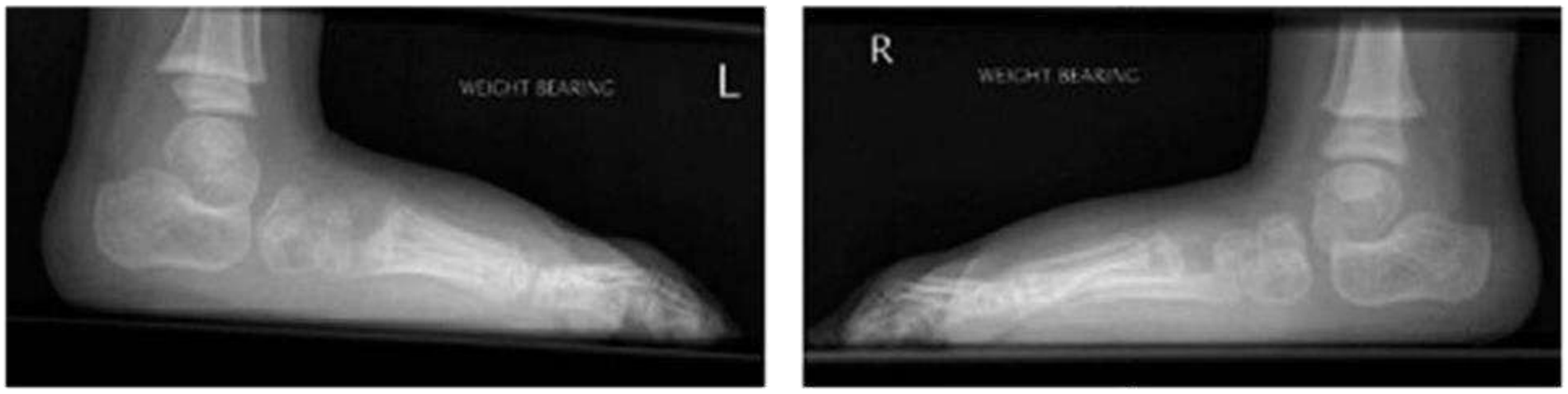


Figure 12.51 Lateral foot and ankle X-rays of a child with foot deformity.

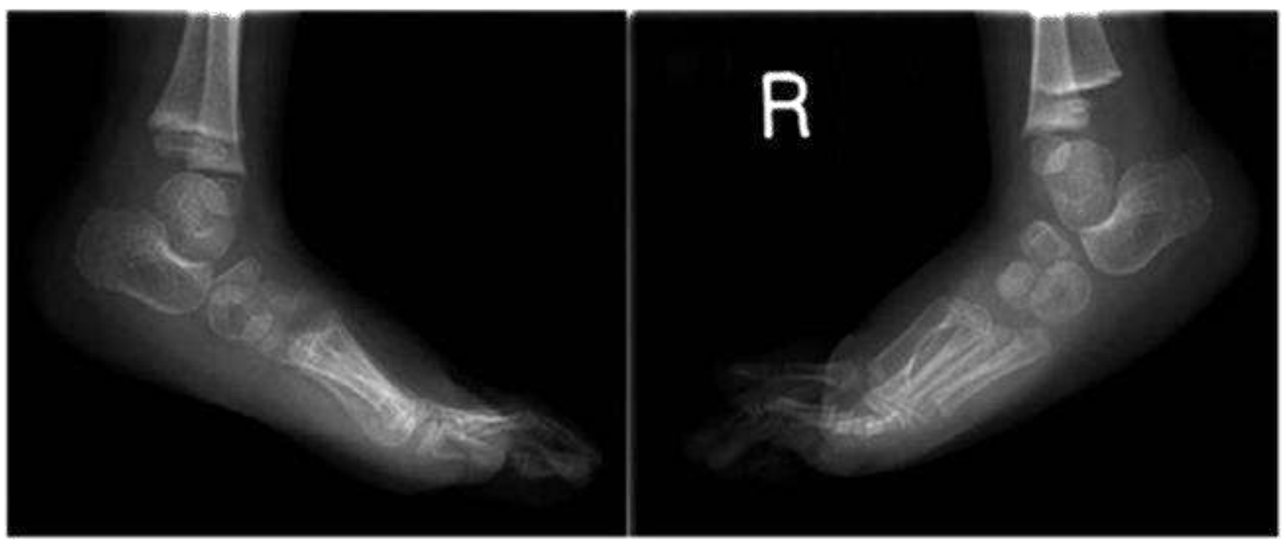


Figure 12.52 Plantar flexion lateral foot and ankle X-rays of a child with foot deformity.

the left foot seems to be pointing to the navicular bone; however, it is not perfectly aligned with the first metatarsal. I suspect there is a congenital vertical talus on the right-hand side. I would like to see lateral views with the ankle joint in hyperextension and hyperflexion.

Q 10: These are what you requested. How can they help? (Figure 12.52.)

These views help me differentiate between the congenital vertical talus and flexible oblique talus. In the former, the hind foot is rigid and the calcaneum and the talus remain in plantar flexion (right foot) and the alignment of the three bones (talus, navicular and first metatarsal) is not restored while it is not the case on the left side. Also, the soft tissue shadow shows a convex contour to the sole (rocker bottom).

Q 11: So, what is a vertical talus?

It is a rare congenital foot deformity characterized by a dislocation of the talonavicular joint. The navicular dislocates dorsally and the head of the talus points towards the plantar. The heel in equinus and valgus and the forefoot in dorsiflexion and abduction produce the classic rocker-bottom appearance. It is usually associated with other conditions such as:

1. Arthrogryposis.
2. Multiple pterygium syndrome.
3. Neuromuscular diseases (poliomyelitis, CP).
4. Spinal muscular dystrophy.
5. Genetics (trisomy 13, 15, 17, 18 and Turner syndrome).
6. Sacral agenesis.
7. Neural tube defects (myelomeningocoele, diastematomyelia).
8. Visceral anomalies.
9. Malformation syndromes (Marfan syndrome, nail patella syndrome, Freeman–Sheldon syndrome).

Q 12: How would you treat this child?

The aims of the treatment are:

1. Reduction of joint dislocation.
2. Maintenance of the joint reduction and restoration of normal foot biomechanics.
3. Identify any associated diseases and treat them (need multidisciplinary input).

There are a few techniques to achieve and maintain reduction depending on the age of presentation, severity of the condition and other associated disorders.

1. Serial casting and percutaneous surgery.[29,30]

This is sometimes called a reversed Ponseti technique. The abducted forefoot is gently plantar flexed and inverted with one hand while the other hand is pushing the talar head upward to correct the plantar-flexed position. This procedure is repeated at weekly intervals. Four or five casts are usually required until the plantar-flexed talus is in line with the first metatarsal.

A K-wire is introduced from the dorsum of the forefoot in line with the first metatarsal, through the navicular bone and talus so that it keeps the talo-navicular joint reduced. Percutaneous tendoachilles tenotomy is performed to correct the hind foot equinus and valgus. The foot is then put in cast in 5° dorsiflexion for 4 weeks. The K-wire is removed at 4 weeks; the foot is recast for another 4 weeks in 15° of dorsiflexion. Then a solid AFO to keep the foot in 15° planter flexion and 15° adduction is used until the age of 2 years.

2. Open soft tissue release.

In older children and severe cases, the closed reduction can be unsuccessful by the above method. Hence, open talonavicular joint reduction with soft tissue release is required. It is usually performed around 9–12 months. All contracted tissues are released or lengthened such as tendoachilles, posterior capsule, dorsal tendons (extensor digitorum longus, peroneus tertius). Rarely, excision of the navicular may be necessary.

3. Tibialis anterior transfer to the talar neck to prevent the talus from plantar flexing.
4. Triple fusion as a salvage procedure.

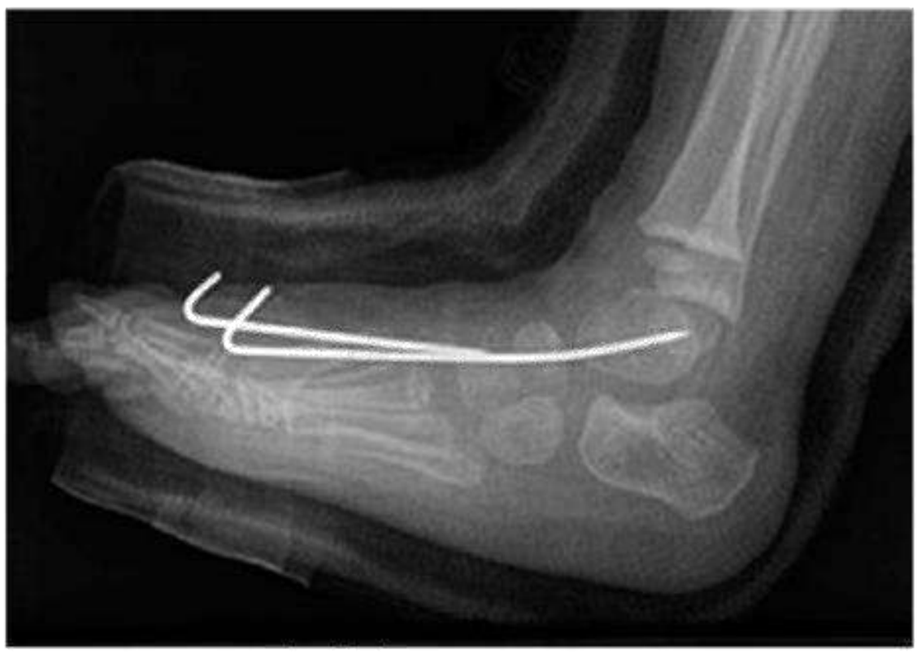


Figure 12.53 Postoperative vertical talus correction X-ray.

Station 4: Miscellaneous

Q 1: Can you draw me the different layers that the growth plate (physis) consists of and give me an example of a disease that affects each layer? (Figure 12.54.)

Q 2: What classifications do you use for physeal fractures?

Several classification systems have been developed for physeal fractures. The most widely used system today is the Salter–Harris classification (Figure 12.55).

- Type I injuries involve complete separation of the epiphysis through the physis without fracture through the metaphysis.
- Type II injuries involve separation of a portion of the physis with the fracture progressing out of the metaphysis.
- Type III injuries involve a fracture that runs through a portion of the physis and out through the epiphysis.
- Type IV fractures are longitudinal splits through the epiphysis, physis and metaphysis.
- Type V fractures involve a crush injury of the growth plate and are not evident on radiographs at the time of injury.

This classification has been modified by Peterson, Rang and Ogden. Peterson added another two types: Type VI with metaphyseal fractures extending to the physis and Type VII with loss of the physis (VIIa for central and VIIb for peripheral). (Figure 12.56.)

Ogden from his series of 443 physeal fractures has added another three (Figure 12.57):

Type VII: Epiphyseal fractures not involving physis.
Type VIII: Metaphyseal fractures affecting later growth.
Type IX: Periosteal damage affecting later growth.

Q 3: What is the significance of this classification?

This classification predicts the impact of injury on the growth plate function; the higher the grade the worse the prognosis (Table 12.16). The location of the physeal fracture is also an important prognostic factor. Undulant physis or irregular physis (such as distal femur and distal tibia) have the worst prognosis as the fracture is more likely to affect several layers of the physis.

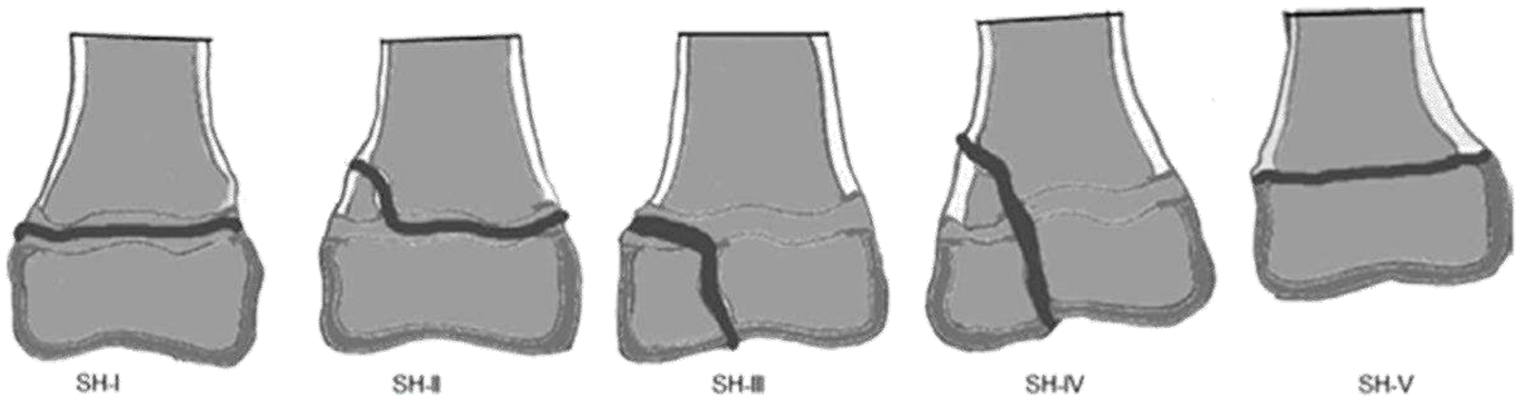


Figure 12.55 Salter–Harris classification.

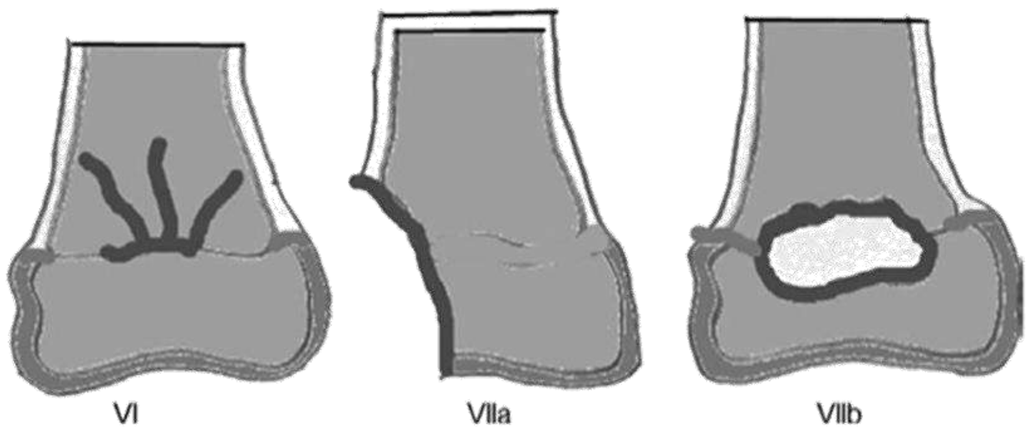


Figure 12.56 Peterson modification of Salter–Harris classification.

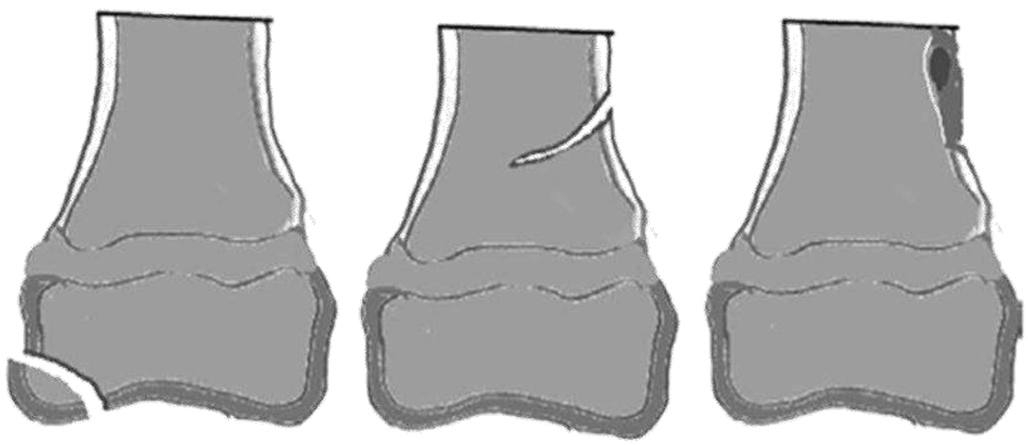
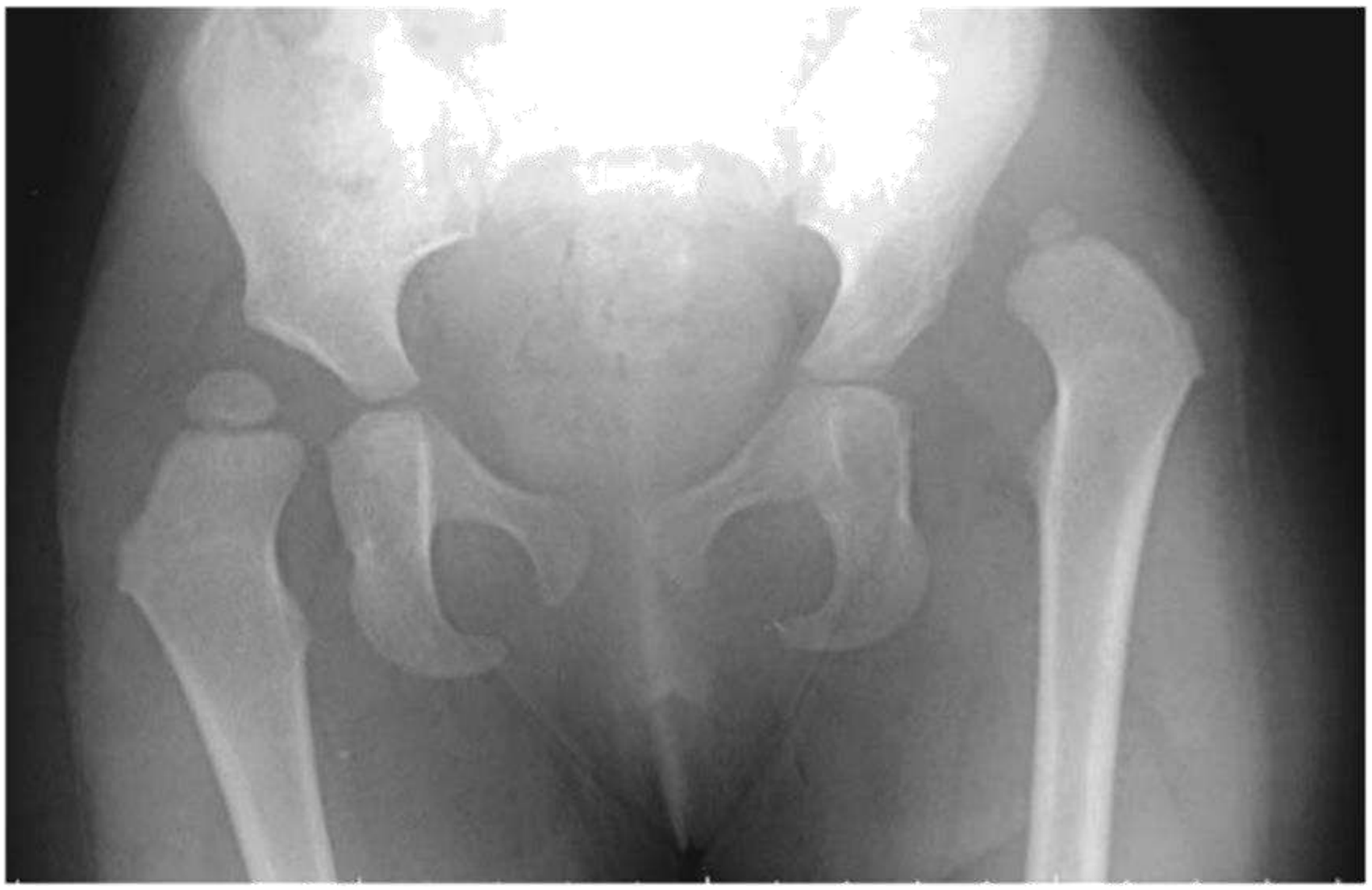


Figure 12.57 Ogden modification of Salter–Harris classification.

Viva 11

This 2 1/2-year-old girl is referred to your clinic with a limp.



Describe the radiographic findings.

How would you proceed in your management from here?

What open operative approaches would you use to reduce this hip?

Describe the radiographic findings.

This is an AP pelvic radiograph showing a dislocated left hip and dysplastic acetabulum. Shenton's line is broken and the femoral head lies lateral and superior to the inferiomedial quadrant (made by the intersection of Perkin's and Hilgenreiner's lines).

How would you proceed in you management from here?

I would take a full history and examine the child. There may be risk factors for developmental dysplasia of the hip (DDH) including positive family history and/or decreased intrauterine space [first born, breech, oligohydramnios (associated packaging problems)].

More importantly I would be looking to see if there were any underlying neuromuscular conditions such as spina bifida, arthrogryphosis, or cerebral palsy. Examination may reveal a Trendelenberg gait, leg length discrepancy, fixed flexion deformity as well as reduced abduction of the left hip, which is the most consistent and reliable clinical sign of this condition.

I would organize an examination under anaesthesia (EUA) and arthrogram to delineate the anatomy of the acetabulum, soft tissues, and proximal femur. It would be unlikely that this hip would reduce closed. Blocks to reduction would include: an inverted limbus; elongated ligamentum teres; hour-glass constriction of the capsule; psoas tendon and pulvinar. Indications for open reduction include: failure of closed reduction; an unstable reducible hip, or soft tissue interposition preventing a congruent reduction.

What open operative approaches would you use to reduce this hip?

I would use a modified anterior (ilio-femoral) approach to the hip. I would place my skin incision parallel and distal to the iliac crest, passing 2 cm distal to the anterior superior iliac spine (ASIS) and extending medially within the groin skin crease.

I would identify and protect the lateral cutaneous nerve of the thigh and then distally I would develop the internervous plane between tensor fascia lata (superior gluteal nerve) and sartorius (femoral nerve). Splitting the iliac crest apophysis I would elevate the muscles *en masse* on both sides of the pelvis down to the sciatic notch and the superior border of the acetabulum. I would divide the straight head of rectus femoris and then make a T-shaped capsular incision to enter the hip joint and clear the acetabulum of pulvinar and redundant ligamentum teres (not the labrum). Any inverted labrum will require to be everted and one or more radial cuts may be necessary to allow this. The inferior capsule may also require release, with care not to damage the blood supply to the femoral head. It is likely there would be tightness in the iliopsoas and its tendon will need releasing to be able to reduce the hip.

I would then assess the need for: (1) a shortening femoral osteotomy and/or (2) pelvic osteotomy (e.g. Salter) to give more cover.

I would then perform a double-breasted capsular repair, close in layers and apply a hip spica cast with the hip in approximately 30° of abduction and internal rotation. The spica would need changing at 6 weeks for a total of 3 months. Post-operatively I would watch carefully for spica syndrome and organize an MRI scan to check that the hip remains enlocated.

The patient would require long-term follow-up to check that the hip develops normally.

Viva 12

This is a photograph of a 7-year-old girl sitting in a comfortable position. Her mother is concerned because she walks with her feet turned in.

Photograph courtesy of Paul Thornton-Bott FRCS (Tr&Orth).

How would you proceed with your assessment?

You find on your examination that the child has extremely lax ligaments and increased internal rotation of both hips.

How do you grade ligamentous laxity in children?

The mother has asked about surgical treatment for this condition. What would you offer her?

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w would you proceed with your assessment?

This clinical photograph shows a child sitting in the 'W' position.

Important questions in the history would include enquiry about the pregnancy and birth, developmental milestones, family history, and any significant past medical history. I would ask the child and the parents about current symptoms and concerns. The common causes of an in-toeing gait include metatarsus adductus, internal tibial torsion, and persistent femoral anteversion.

In the examination it is important to rule out asymmetry in the lower legs or any neurological signs, which could indicate an underlying spinal abnormality or neurological problem.

I would examine the gait (with shoes on and barefoot), looking specifically at the foot progression angle (negative in this case: normal is –5° to +20°)

With the child lying prone I would assess the torsional profile, looking for:

- Metatarsus adductus: foot shape in relation to heel bisector line
- Tibial torsion: thigh–foot angle (normal range 0–20°) if foot shape is normal or transmalleolar axis (normal range 0–45°) if foot is abnormal. Tibial torsion is defined as the angle between the transcondylar axis of the proximal tibia and the bi-malleolar axis (normal range 10–25° external)
- Femoral anteversion: range of motion, internal rotation (IR) > 60° (normal range 20–60°) > external rotation (ER) < 20° (normal range 30–60°); Ruwe's method, measure angle from vertical [finger on greater trochanter (GT), most lateral point; normal is about 8–14°]

I would also examine the spine and lower limb neurology as well as assess the degree of ligamentous laxity.

How do you grade ligamentous laxity in children?

I would use the Beighton score (out of 9):

Increased finger hyperextension—2 points
Increased thumb hyperextension—2 points
Increased elbow hyperextension—2 points (1 point for each side)
Increased knee hyperextension—2 points
Ability to place palms on floor with legs straight—1 point

The mother has asked about surgical treatment for this condition. What would you offer her?

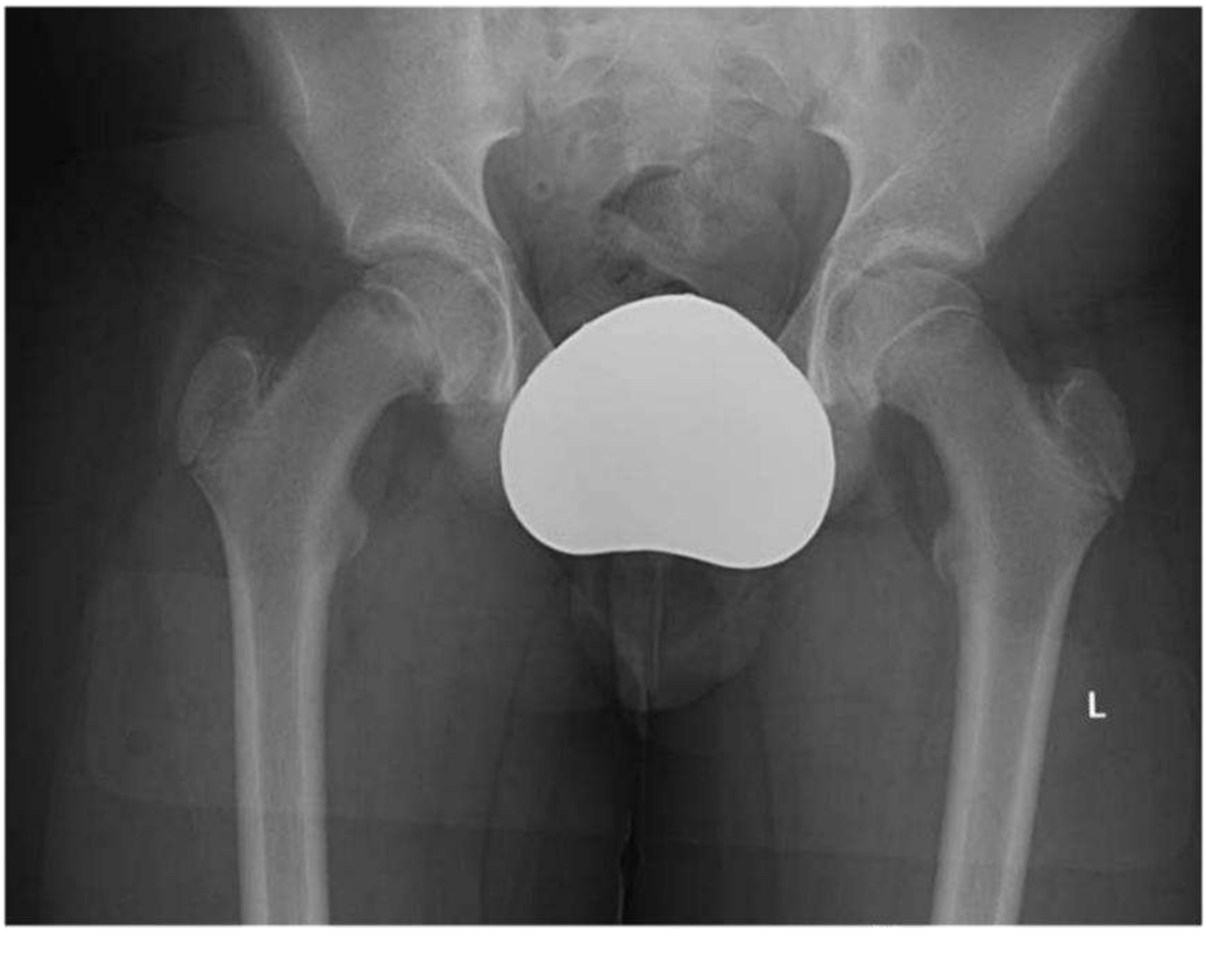
This child's in-toeing gait is most likely due to persistent femoral anteversion which is a common cause in children older than 3 years.

I would reassure the mother that her daughter is physiologically normal but just at one end of the normal spectrum for children of her age. She may be interested to learn that the only effective treatment is to cut the femora, rotate them, and then fix them, which is a major surgical procedure, with risks, for essentially a cosmetic problem.

I would also explain the natural history of the condition that it tends to improve over the first decade but she may well be left in-toeing as an adult. As muscle balance improves into adulthood it rarely presents a functional problem.

Viva 13

This 13-year-old boy presented with pain in his right knee.



Describe the radiograph.

How do you classify this condition?

What is your management plan now with the right hip?

What is your management plan now with the left hip?

Describe the radiograph.

This is an AP radiograph of the pelvis in a skeletally immature child. There is a mild slip of the right upper femoral epiphysis (SUFE) with a positive Trethowan's sign. This is shown by drawing Klein's line up the lateral border of the femoral neck and noting it does not intersect the epiphysis.

How do you classify this condition?

I would use Loder's classification which divides SUFEs into stable and unstable based on the patient's ability to bear weight secondary to pain, and is important for predicting the risk of AVN. Other classifications grade the slip into mild (<33%), moderate (33–50%), or severe (>50%) the degree of which corresponds to the slip angle; this is useful when working out which are pinnable or not *in situ*.

What is your management plan now with the right hip?

I would take a full history for the patient and parents and examine the child. I am looking for any underlying cause for this SUFE such as endocrinopathy.

Examination findings would reveal classically a hip that externally rotates and abducts with flexion.

My management plan would consist of pinning this slip *in situ* with a single cannulated screw—use of more than one screw increases complications including AVN and chondrolysis.

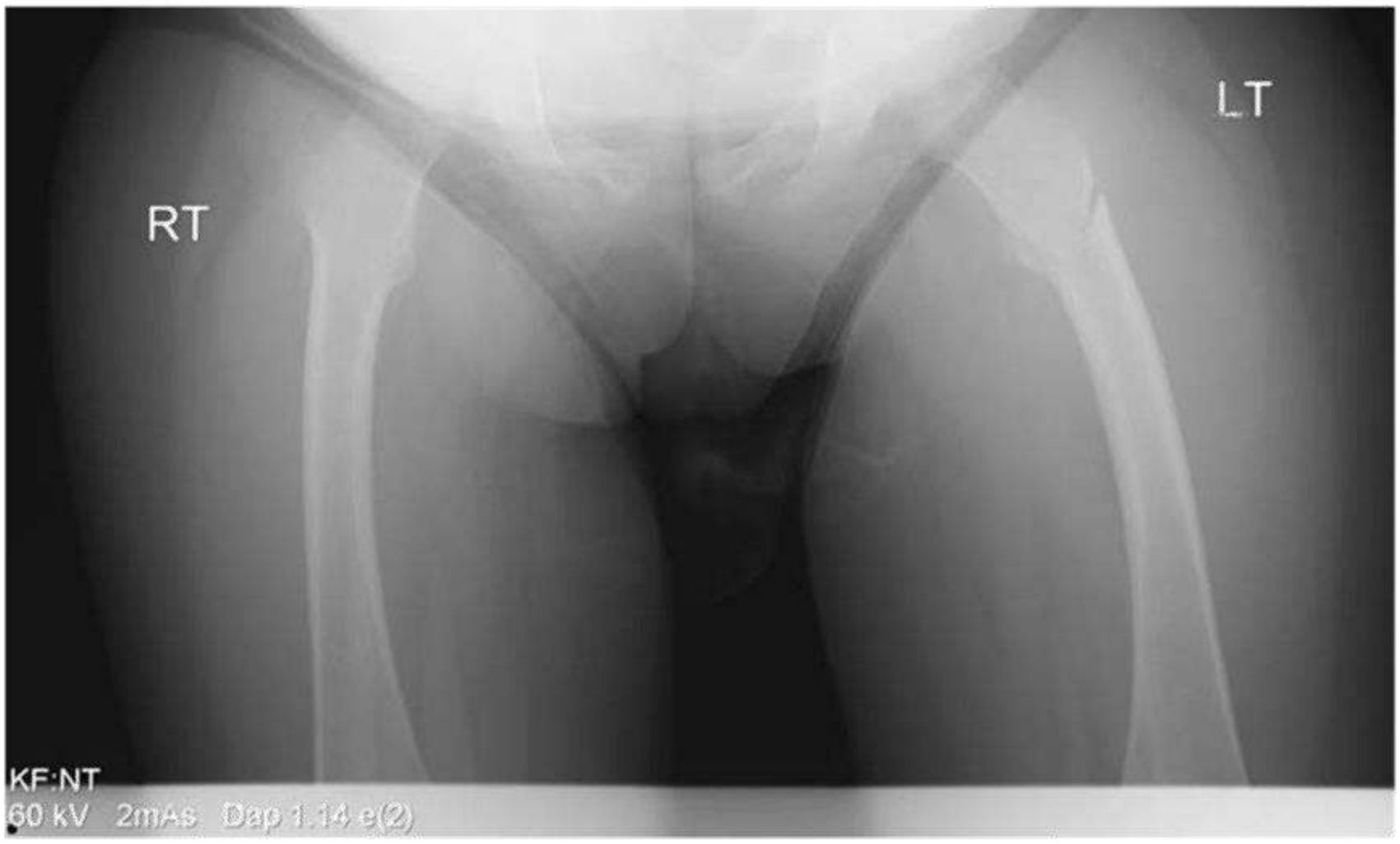
I would perform this under GA on a fracture table, but would not use a forced reduction manoeuvre or traction which could increase the risk of AVN. I would use a triangulation technique to define the appropriate location for the skin incision. The thread of the screw should be in the centre of the epiphysis passing through perpendicular to the physis (this avoids perforating the femoral head). As the slip is usually postero-medial this technique usually requires an anterior femoral neck entry point. A minimum of two to three screw threads should pass into the epiphysis, depending on the size of the child and the instrumentation used.

What is your management plan now with the left hip?

Contralateral prophylactic screw fixation to prevent slip in the future remains a controversial topic. Options are to treat every case with contralateral fixation, versus pinning only those children thought to be at higher risk of contralateral slip or significant leg length discrepancy (less than 10 years old, underlying endocrinopathy).

Viva 14

This 8-month-old baby was brought to casualty with the above injury.



What are your thoughts?

Tell me about non-accidental injury and what you would do if you suspected it?

How would you treat this fracture?

What are your thoughts?

This is a AP view radiograph of a child's lower pelvis and femurs showing an oblique fracture at the left subtrochanteric level. I would like to take a detailed history from the parents or carer, as a fracture of the femur in a non-ambulant child could be a non-accidental injury (NAI). **You must pick this possibility up—it is reasonable for this to be a pass/fail type question.**

Tell me about non-accidental injury and what you would do if you suspected it.

Non-accidental injury is an injury deliberately inflicted by a parent or a caregiver. It may be difficult to suspect a parent or carer of abuse but we have a duty of care as professionals to ensure care and protection of children.

Child abuse itself can take different forms (physical, neglect, sexual, emotional, Munchausen's by proxy—rare): most are in combination. It is the second most common cause of death in young children (after trauma). Risk factors include first born, premature babies, stepchildren, family history of abuse, and parental IV drug abuse.

Firstly it is important to get the child into a safe environment and 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). Having taken a detailed history from the parents and examined the child fully (with a chaperone), I would document my findings carefully in the notes. Clues suggesting NAI in the clinical assessment include a history that doesn't fit the injury, inconsistent explanations and delayed presentation, bruising patterns on the child, and retinal haemorrhages. I would inform the paediatricians of my concern about a possible NAI and make arrangements for the child to be admitted.

Radiographic clues for NAI include metaphyseal bucket handle corner fractures (virtually pathognomonic), rib fractures, and fractures of varying ages evidenced by different stages of healing.

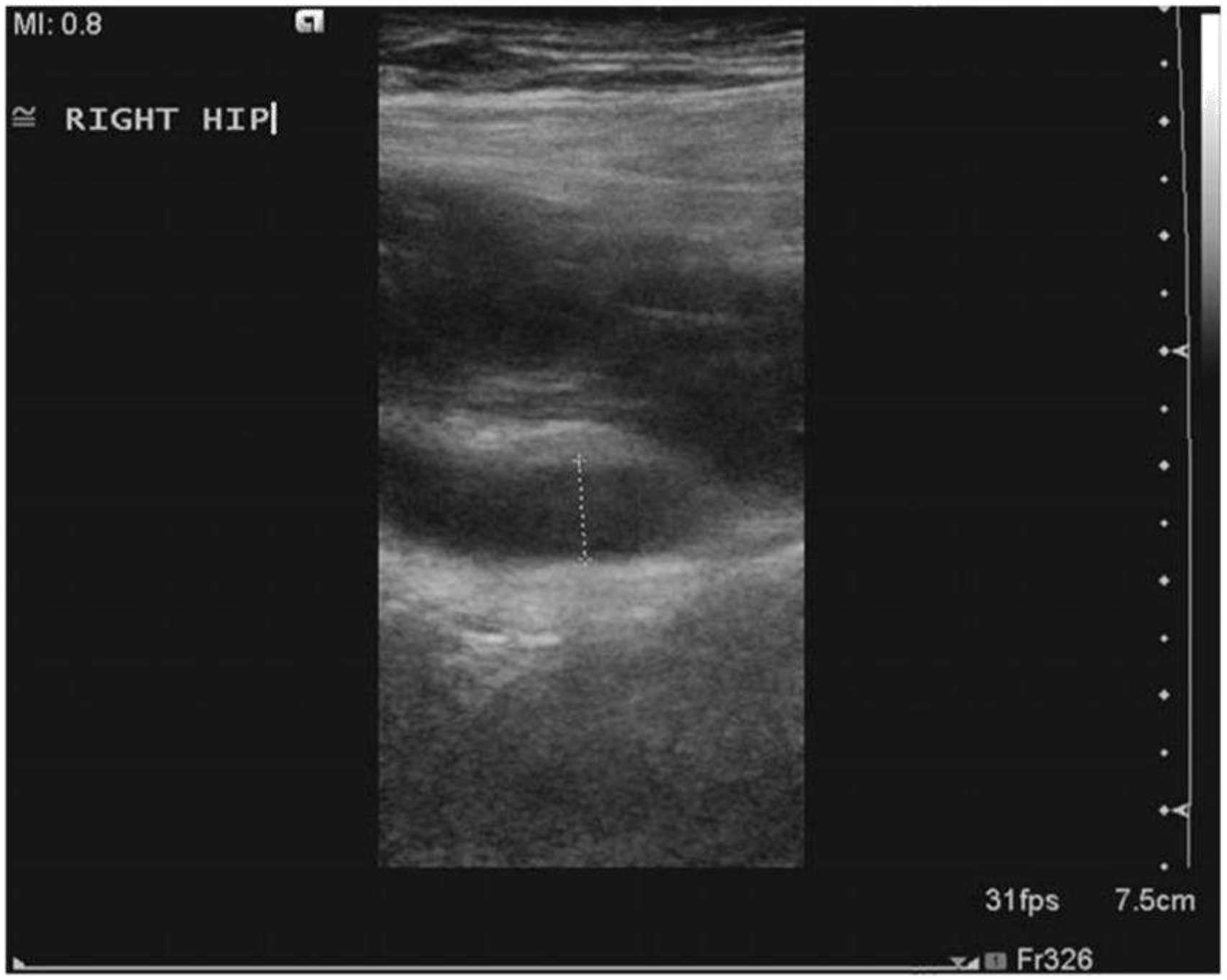
How would you treat this fracture?

I would treat this fracture in gallows traction with a radiograph at 2–3 weeks, to show callus formation, and then gentle mobilization as comfort allows. A hip spica is sometimes used.

Viva 15

A 3-year-old child is referred from casualty with a 24-h history of fever, malaise, and reluctance to bear weight on his right leg.

What is your approach to this patient?



The casualty officer has sent some routine bloods and organized an ultrasound scan (USS) of his right hip which is shown above.

How would you assess the child and what is your management now based on the hip scan?

What is your approach to this patient?

I would want to assess the child to exclude an infectious cause for his symptoms such as a septic arthritis or osteomyelitis.

How would you assess the child and what is your management now based on the hip scan?

My clinical assessment would start with a detailed history from the parents.

On examination I would make an assessment of whether the child was well or unwell, and request a temperature measurement. I would assess the resting posture and range of motion of the hips. I may find the hip to be in a position of most comfort (flexed, abducted and externally rotated). I would also examine the whole lower limb, chest, abdomen, and spine.

The ultrasound shows an effusion around the hip which has been measured as 7 mm.

I would review the blood investigations taken.

I would organize AP and lateral radiographs of any affected part to rule out any underlying structural abnormality or fracture.

Four predictive markers of hip septic arthritis include:

1. Temperature > 38.5°C
2. White blood cells (WBC) > 12,000 cells/mm^3
3. ESR > 40
4. Non-weight bearing

The chance of there being a septic arthritis increases with the number of positive factors present [×1= 3%, ×2= 40% (treat as septic arthritis), ×3= 93%, /×4= 99.6%].

I would arrange an aspirate of the hip joint

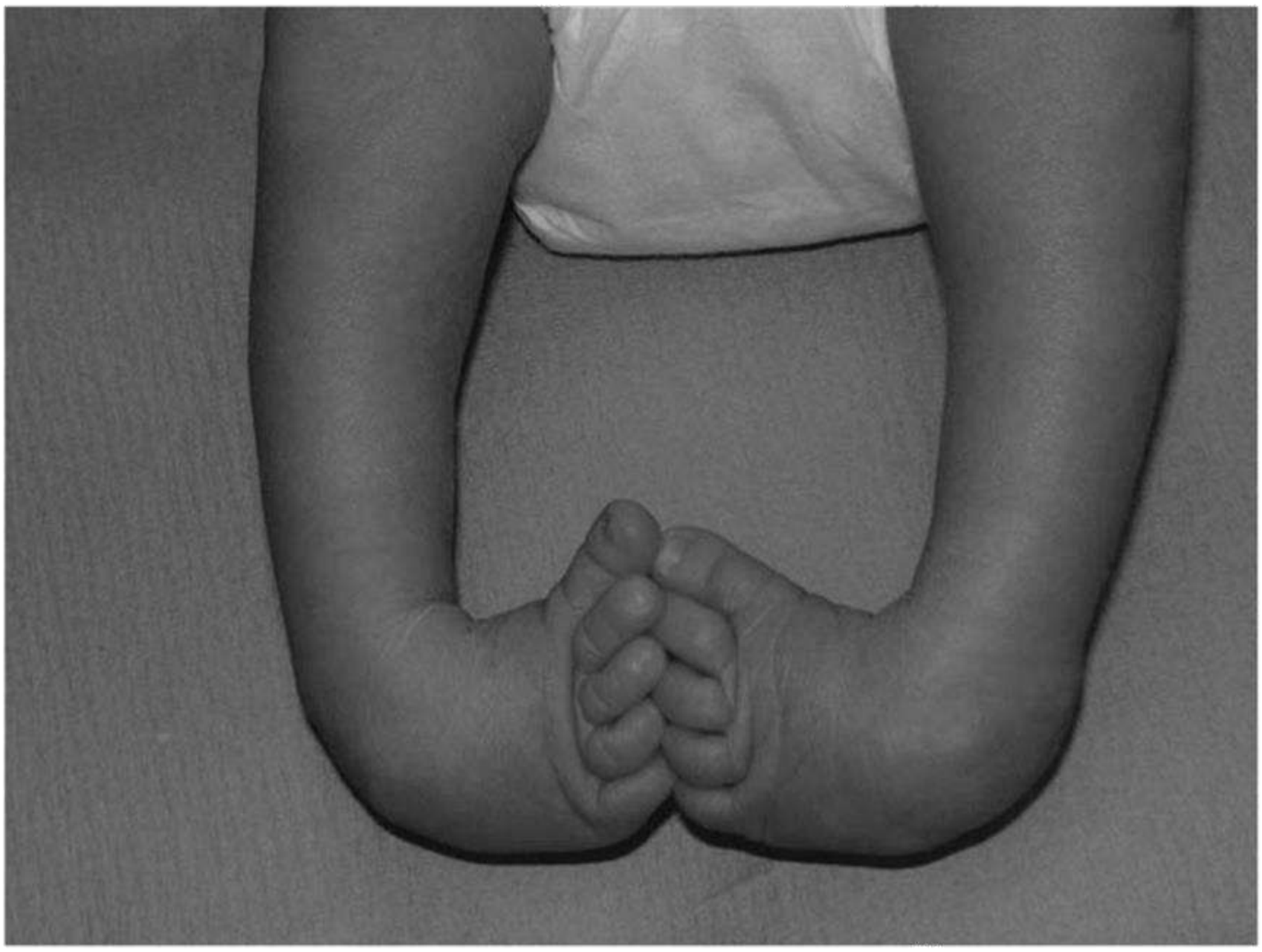
If the aspirate revealed pus I would organize an open washout of the hip as soon as possible (urgent case < 6 h)

I would approach the hip through an anterior approach and remove an ellipse of capsule to allow free drainage after taking some deep tissue samples to send to microbiology and a copious washout with normal saline. I don't routinely use a hip spica post-operatively but recognize the risk of secondary subluxation and dysplasia that may develop in this condition. I would discuss appropriate antibiotics with the microbiologist, usually starting with broad spectrum and then adjusting, guided by the culture and sensitivities.

The child would require daily clinical and serial biochemical (inflammatory markers) review to make sure they improved. A prolonged course of antibiotics is advised. The child would also require longer-term follow-up to check the development and the growth of the hip joint.

Viva 16

You are asked to go and assess a newborn chid on the maternity ward.



Describe the clinical photograph and the components of this deformity.

How do you classify the severity of this condition?

Describe the clinical photograph and the components of this deformity.

This is a clinical photograph of a newborn child with a clubfoot deformity (congenital talipes equino-varus). This is a complex three-dimensional deformity seen at birth with cavus and adductus of the midfoot and forefoot and varus and equinus of the hindfoot.

How do you classify the severity of this condition?

There are different scoring systems described to grade the severity of the deformity. In my institution we use the Pirani scoring system. It comprises 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 three 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.

How would you take your management from here?

I would manage this patient by taking a good history from the parents and examining the child to make sure they did not have any associated congenital anomalies or features which may suggest that this is a 'syndromic' club foot as opposed to an idiopathic clubfoot. I would then explain and start the Ponseti treatment programme, which is now recognized worldwide as an appropriate mainly non-operative approach to club foot treatment.

It starts with manipulation and serial casting.

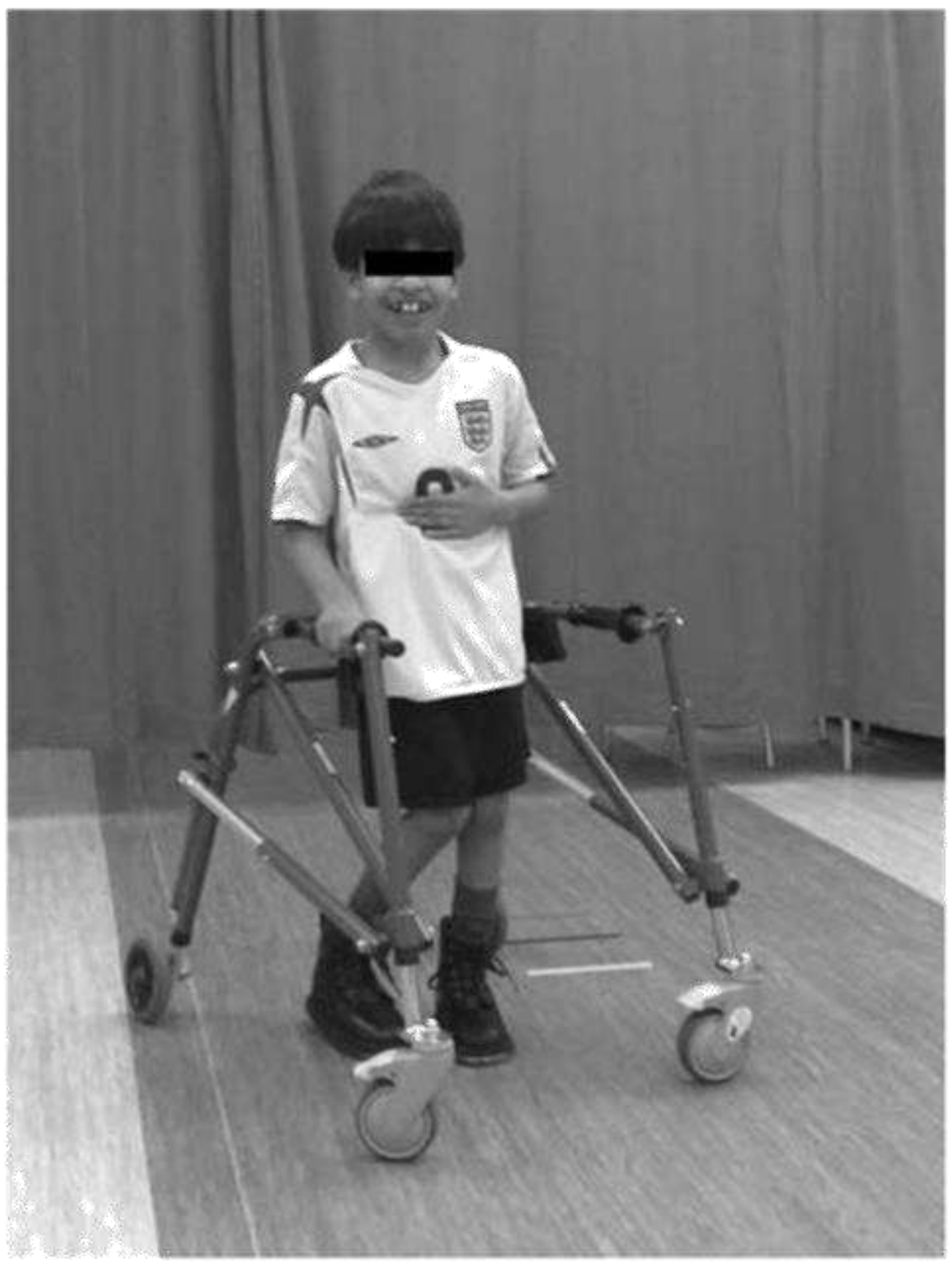
The first key manoeuvre is to reduce the cavus deformity by dorsiflexing the first ray and unlocking the forefoot and midfoot. Elevation of the first ray produces supination so I warn the parents the foot may look worse after the first cast. The second important manoeuvre is to abduct the forefoot at midfoot level using the uncovered head of the talus laterally as a fulcrum.

Above-knee casts (with the knee at 90°) 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. 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°. The vast majority of patients do very well and avoid the need for extensive surgical release. However, approximately 25% will require a tibialis anterior transfer laterally for residual deformity or inversion in swing after the age of about 4–5 years.

Viva 17

Here is a child with cerebral palsy.



What is cerebral palsy and what different types do you know?

What is spasticity?

In what different ways do we manage spasticity?

Often ambulant children with cerebral palsy are assessed by gait analysis. What does that involve?

What is cerebral palsy and what different types do you know?

Cerebral palsy is a neuromuscular disorder caused by a non-progressive lesion to the immature developing brain (before the age of 2 years). Although the neurological injury is non-progressive, the musculoskeletal features evolve as the child grows.

Types of cerebral palsy are:

Anatomical—hemiplegia (40%)/diplegia (30%)/total body involvement (30%)
Physiological—spastic (60%)/dystonic (20%)/ataxic (10%)/hypotonic (10%)
Functional—classified by the Gross Motor Function Classification System (GMFCS)

What is spasticity?

Spasticity is the velocity-dependent increase in the tone of muscles (represents an increased response to stretch reflex).

In what different ways do we manage spasticity?

Principles include a multidisciplinary approach involving family and patient in goal planning, decisions about treatments, and exploring expectations.

Non-operative treatment is based around a physiotherapist who often acts as the main link between specialists. Adjuncts can be used to control spasticity including: botulinum toxin injections or baclofen (tablets or intrathecal pump).

- Botulinum toxin A (derived from *Clostridium botulinum*) is injected locally (dose is weight dependent) into spastic muscles. It works by preventing release of acetylcholine at the neuromuscular junction of those tight muscles and is effective for 3–6 months. It is often used in combination with plasters and targeted physiotherapy and/or orthotics to maintain an improved stretch
- Baclofen is a gamma-aminobutyric acid (GABA) agonist (an inhibitory neurotransmitter) which acts both centrally and peripherally to decrease spasticity. If administered intrathecally, via an infusion pump it allows an increased local dose with decreased systemic side-effects

Surgery is often needed, and appropriate planning and timing is crucial when performing multilevel surgery to avoid 'birthday syndrome'. Options can include bony surgery as well as soft tissue lengthening (without weakening) of tight muscles, as well as transfers of muscles to augment weak muscles.

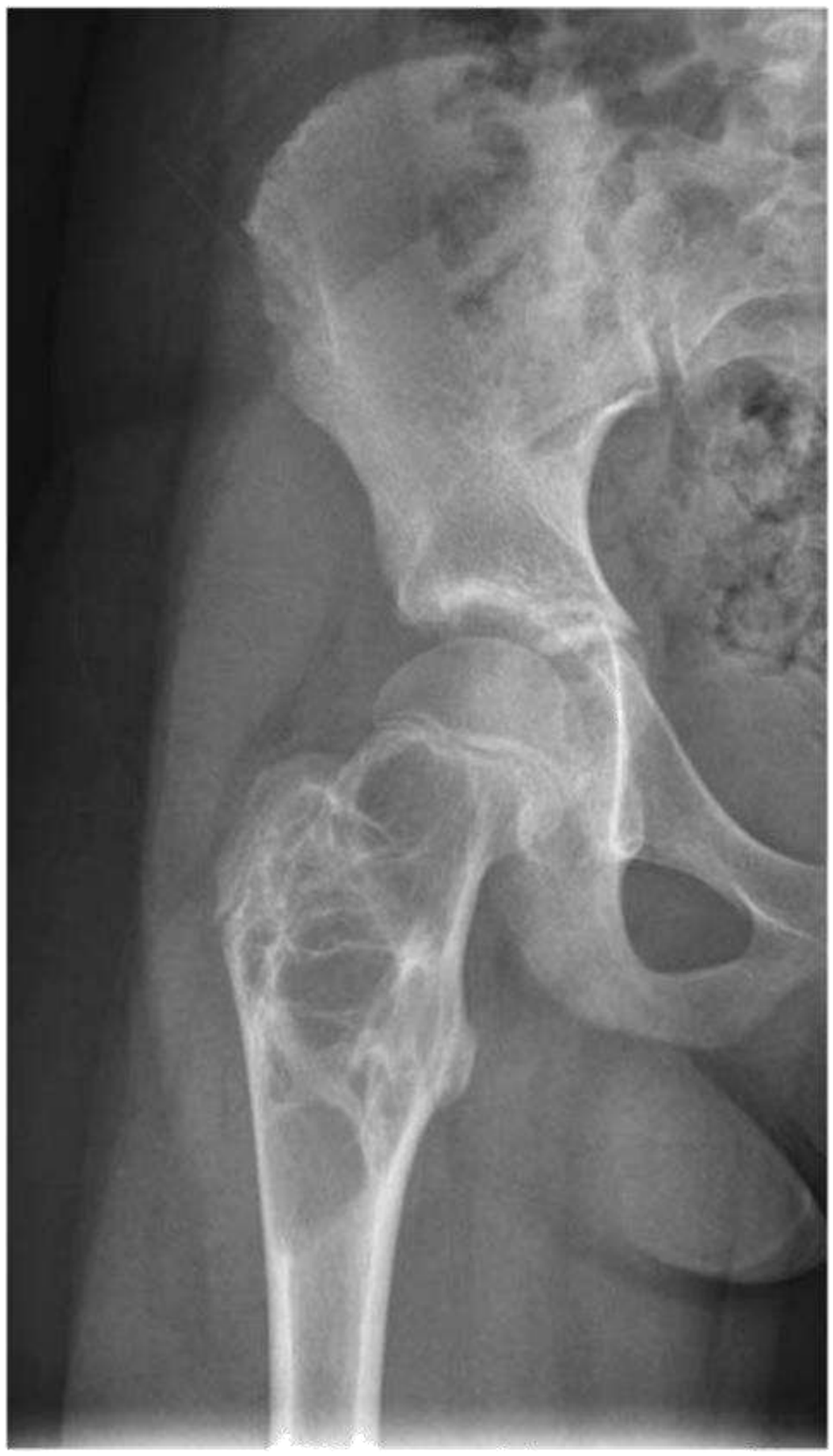
Often ambulant children with cerebral palsy are assessed by gait analysis. What does that involve?

Gait analysis is the systematic description, assessment, and measurement of the quantities that characterize human locomotion. It involves the study of kinematics (the movements of the individual parts of the body) and kinetics (the forces and how they interact to produce those movements) as well as electromyography and energy consumption.

There is no defined standard; however, most gait laboratories will have two-dimensional video analysis and three-dimensional computer analysis, using specialized markers stuck onto specific bony landmarks. The computer program then breaks down the individual movements of anatomical parts into graphic form. Further detailed analysis involves the use of force plates, measuring ground reaction force, and electromyography, looking at muscle firing patterns.

It is important that the results of gait analysis are looked at in conjunction with a static detailed physical examination.

Viva 18



Can you describe this radiograph? What do you think the diagnosis is?

This patient presents with new-onset pain in the upper thigh. What do you think has happened?

How would you manage this patient now?

You treat expectantly, but unfortunately the lesion remains. How would you proceed now?

Can you describe the radiograph? What do you think the diagnosis is?

This is an AP radiograph showing a multiloculated lytic lesion in the proximal metaphysis of an immature individual. The zone of transition is sharp indicating this is likely to be a benign lesion with no associated periosteal reaction. Top of my diagnosis would be a loculated simple bone cyst, but an aneurysmal bone cyst is also a possibility.

This patient presents with new-onset pain in the upper thigh. What do you think has happened?

A lot of these lesions are found incidentally on radiographs taken for another reason, but new-onset pain in that area would suggest a pathological fracture through the weakened bone. A fallen fragment sign (cortex that has fallen into the cystic cavity) is pathognomonic of this.

How would you manage this patient now?

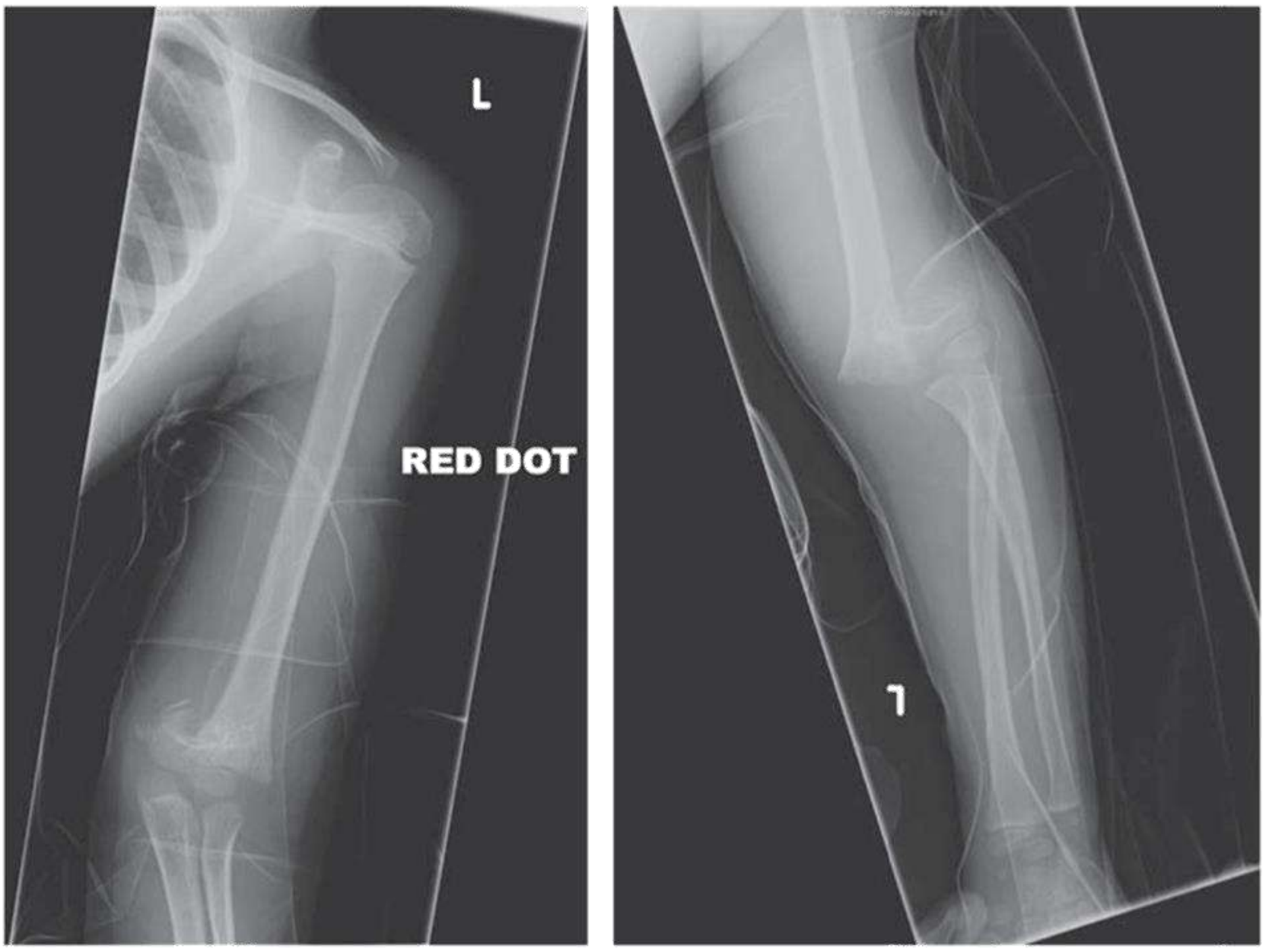
Having taken a thorough history and examined the patient, I would probably manage this expectantly as in a good percentage of patients the fracture actually stimulates new bone formation and with time the cyst fills in.

You treat expectantly but unfortunately the lesion remains. How would you proceed now?

If an expectant non-operative approach failed I would, under image guidance, aspirate the cyst and inject some steroid or bone graft/marrow to try and stimulate new bone formation. If this failed, a repeated attempt is worthwhile; however, the definitive surgical treatment would involve curetting out the lining of the cyst through a small cortical window and stabilizing the bone to prevent fracture. I would use flexible intramedullary nails across the lytic area. If the cavity is adjacent to the growth plate it is important not to damage it and risk growth arrest.

Viva 19

This 6-year-old child fell out of a tree onto their left arm.



Can you describe the radiograph?

How would you manage this child?

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?

There is an 'off ended' Gartland 3 supracondylar fracture of the distal humerus.

How would you manage this child?

Assuming this was an isolated injury I would assess the child for the presence of an open injury, and also assess the distal neurovascular status (colour of hand, capillary refill of the fingertips, radial pulse, sensation in the specific dermatomes, and motor function of ulnar, radial, median, and anterior interosseus nerves).

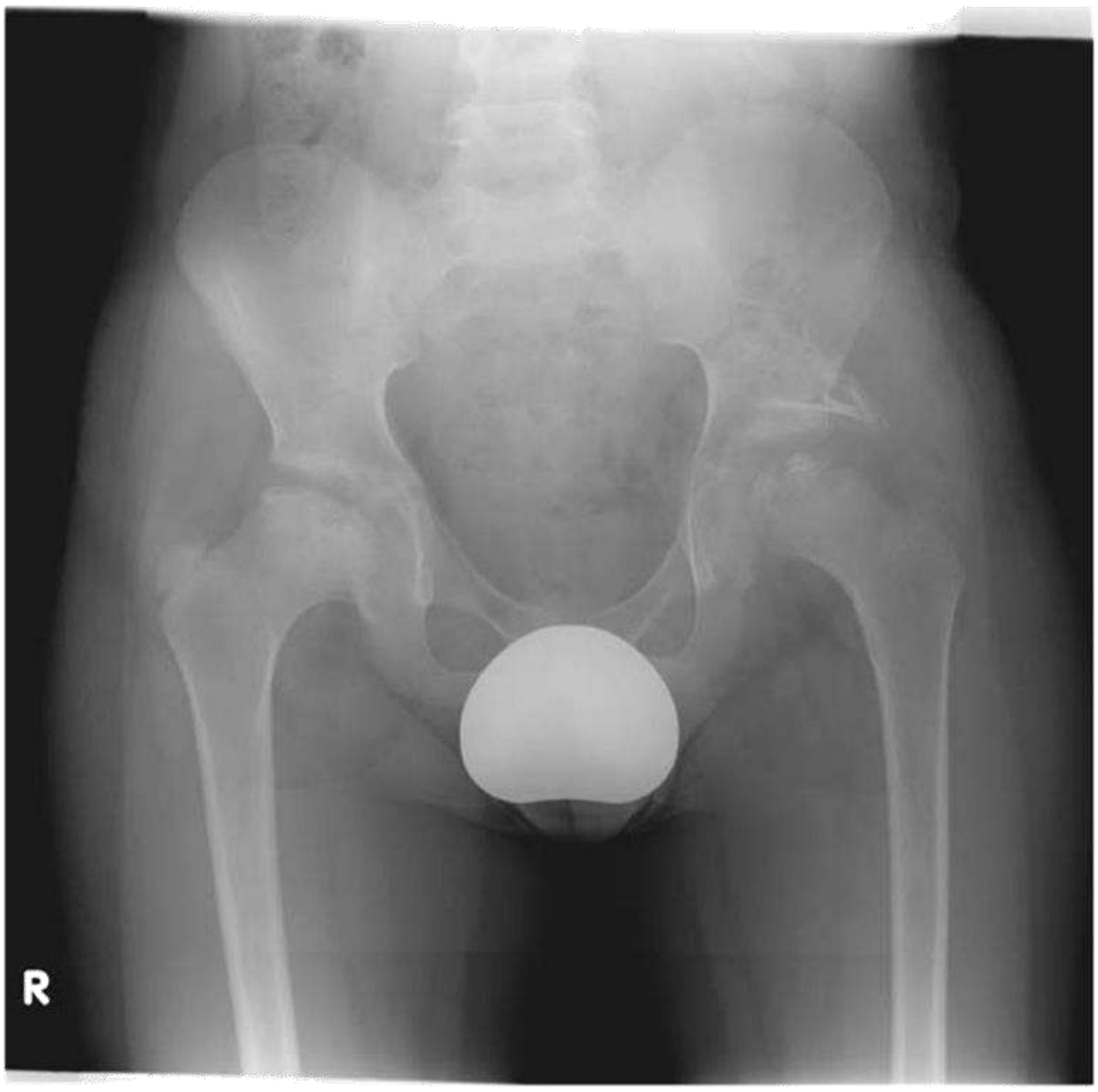
I would organize for the child to have analgesia and a temporary backslab splint to stop the arm from moving and then mobilize my theatre team, as this child needs to go to theatre as soon as possible for closed reduction and percutaneous pinning.

In theatre, the set-up of the image intensifier and the help of a good assistant is key. The technique for reduction of these injuries is to apply good continuous traction (in 20° of flexion) for several minutes, then correct any valgus/varus and rotational deformity, before flexing up and hooking the distal fragment back on to the shaft. The forearm can be pronated to lock the fragments. I would insert a lateral wire first (1.6 mm), making sure I was through the far cortex. With that wire giving some stability, it is possible to extend the arm a little to plan a mini open approach to the medial side, allowing protection of the ulna nerve prior to inserting the cross wire. 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 near extension. I would reassess the perfusion of the hand and watch for compartment syndrome.

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 was pink and warm with adequate capillary refill of the fingertips, then I would monitor the situation with regular review. The artery is likely to be in spasm and the pulse can take a day or two to recover. If the hand was white and capillary refill reduced, I would release the backslab and allow the arm to extend to see if this improved the situation. If not I would contact the vascular/plastic surgeons for an urgent review as the artery may have been caught up in the fracture and has now been occluded by the reduction. If so this would now require open exploration, usually via an anterior approach.

Viva 20



This radiograph shows a post-operative view of a boy's pelvis. What do you think the underlying diagnosis is and what procedure has he had?

What is the underlying disease and who gets it?

How do you classify this condition?

What are the principles of management?

This radiograph shows a post-operative view of a boy's pelvis. What do you think the underlying diagnosis is and what procedure has he had?

This is an AP pelvic radiograph of a skeletally immature patient showing flattening and deformity of both femoral heads in keeping with Perthes' disease. (Legg–Calve–Perthes disease). On the left side this patient has had a shelf procedure, which is a salvage type of acetabular procedure. It is an operation that redistributes the weight-bearing load of the femoral head through a larger surface area of pelvic cover.

What is the underlying disease and who gets it ?

Perthes' disease is idiopathic AVN of the proximal femoral epiphysis in childhood. It remains a controversial topic in orthopaedics because of its unknown aetiology and uncertain optimal treatment. It is more common in boys than girls by about 4:1 and it is bilateral in about 20% of cases.

How do you classify this condition?

There are many classifications used for Perthes' disease. Waldenström classified it into pathological stages:

1. Initial avascular event (crescent sign—representing a subchondral fracture)
2. Fragmentation (Herring's pillar classification)
3. Resolution—re-ossification
4. Remodelling—healed

The Herring classification of severity is based on the lateral pillar height on an AP radiograph during the fragmentation stage:

- B—more than 50% maintained
- C—less than 50% maintained

(A B/C border category was subsequently added.)

Catterall's classification contains four groups depending on the amount of femoral head involved on the lateral radiograph.

Catterall also added clinical and radiological 'head at risk signs' which he used to guide his management:

Clinical	Radiological
Obese	Horizontal physis
Progressive decreased ROM	Lateral subluxation of epiphysis
Abduction contracture	Lateral calcification
ER with flexion	Diffuse metaphyseal reaction
	Gage sign—inverted V-shaped lucency in lateral metaphysis

ROM, range of motion; ER, external rotation.

Stulberg's classification assessed the shape of femoral head at skeletal maturity and is used to predict who will do poorly in terms of early onset degenerative change:

I—normal
II—head spherical (magna/breva) and fits in socket which is congruent
III—mushroom head congruent
IV—flat head and flat socket congruent
V—flat head incongruent

What are the principles of management?

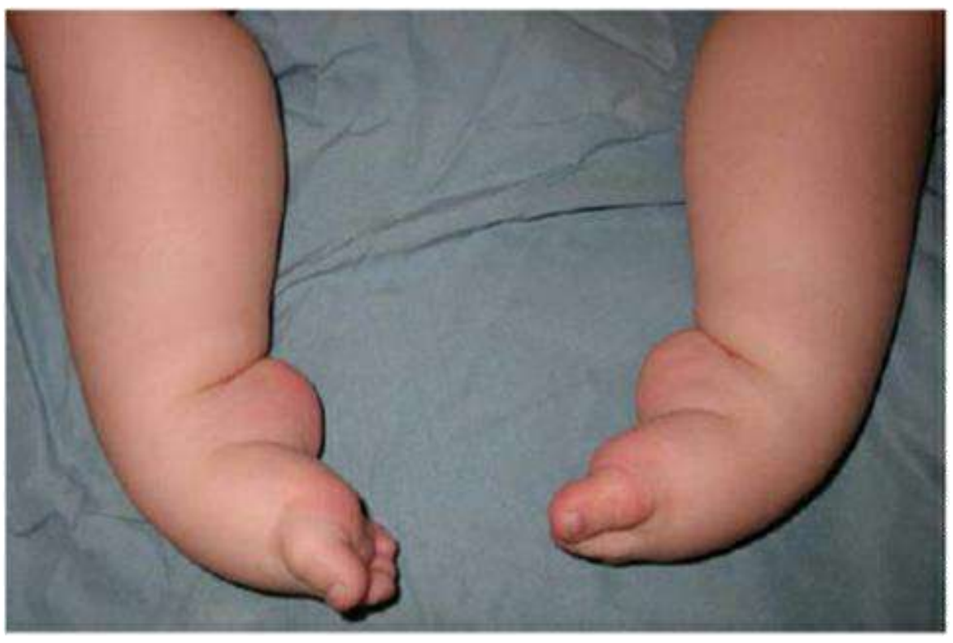
Goals of treatment are:

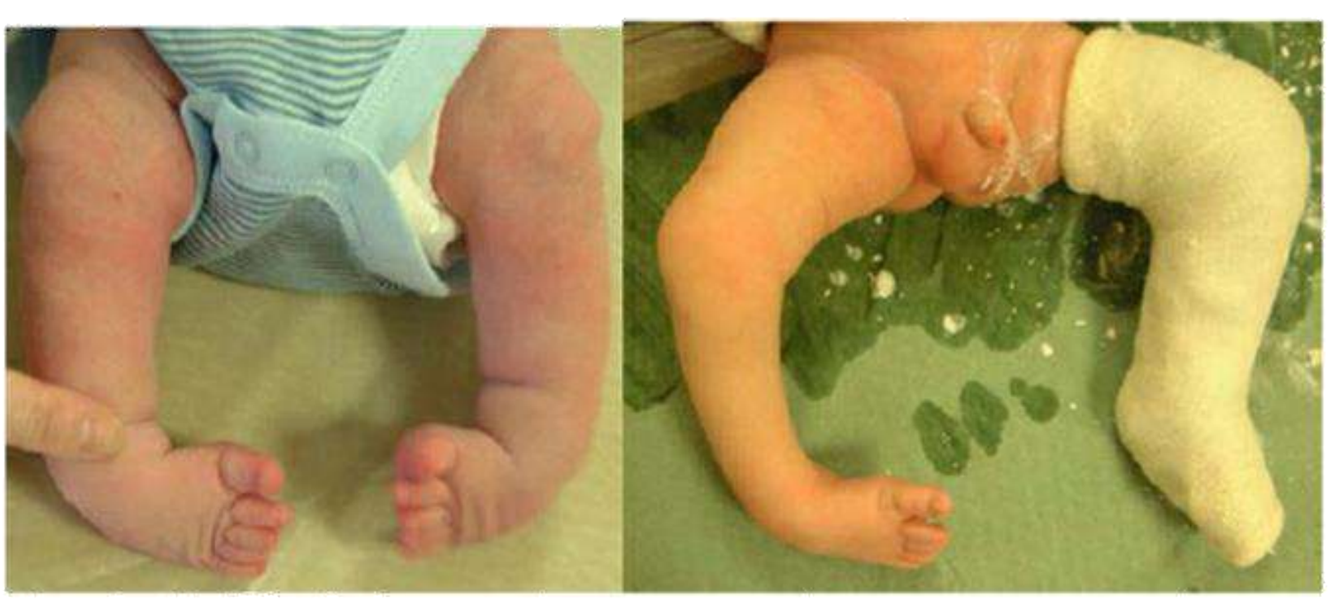
1. Symptomatic relief
2. Containment of the head and hence correct development
3. Restoration of ROM

These goals can be achieved by various non-operative and operative treatments which still are debated around the world.

Each patient should be managed on an individual basis taking into account their age, clinical signs, and radiological appearances on X-ray.

2. Congenital Taliped Equino Varus (CTEV)





Which are the deformities?

Deformities

1. Hindfoot equinus
2. Hindfoot varus
3. Midfoot/forefoot cavus
4. Forefoot adduction

How will you manage- at birth and late stage.

The Ponseti method is the preferred treatment. It starts soon after birth and consists of:

1. Weekly serial casting with above knee plasters for about 6

How will you manage- at birth and late stage.

The Ponseti method is the preferred treatment. It starts soon after birth and consists of:

1. Weekly serial casting with above knee plasters for about 6 weeks.
2. Percutaneous Achilles tendon release in about 80% of patients at about 6 weeks.
3. Further post-operative casting for about 3 weeks (a cast change during this period might be necessary).
4. Boots on a bar 23 hours a day for 3 months.
5. Boots on a bar for during the night up to the age of 4/5 years.

Ponseti treatment is also effective in older children in correcting all or part of the deformity. Depending on the severity of the deformity additional surgery is required.

Ponsetti method

Order of correction

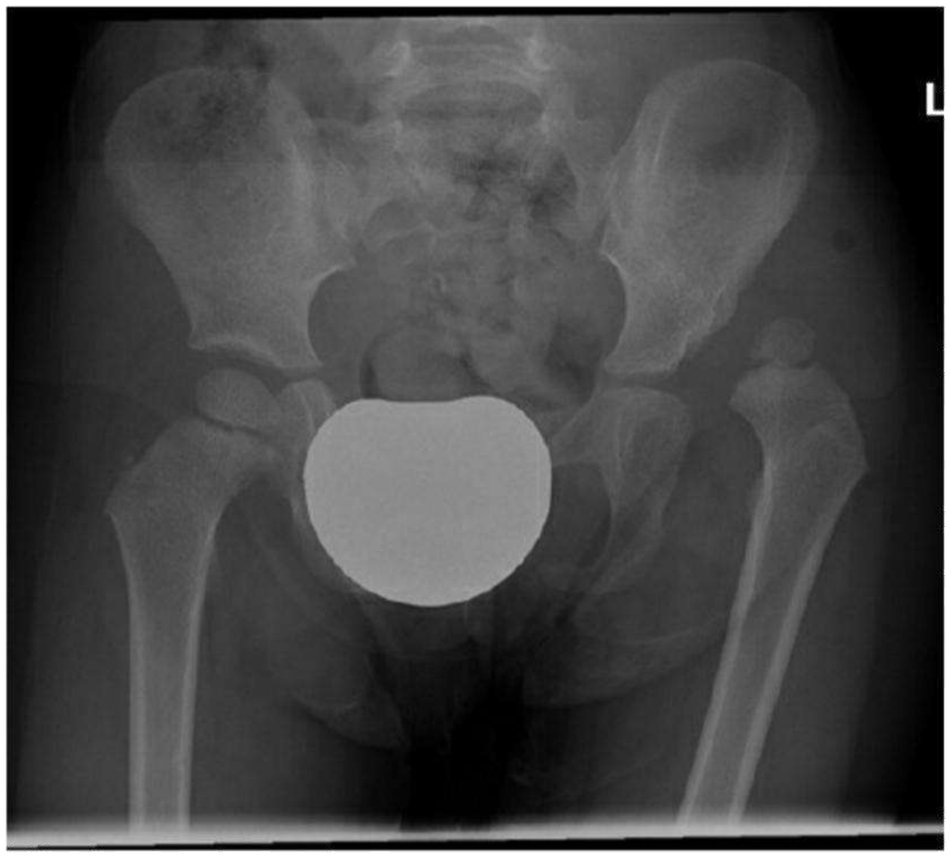
1. Simultaneous correction of cavus, fore/midfoot adduction (aim for 60°-70° abduction) and hindfoot varus.
2. Equinus correction once cavus, fore/midfoot adduction and hindfoot varus are corrected.

About 20% of patients need a tibialis anterior tendon transfer for dynamic supination deformity between the age of 3 to 5 years. Transfer is into the lateral cuneiform (the ossification centre must be visible).

Outcome of ponseti?

Boden et al reported a significant reduction in the need for radical surgical release with the Ponseti technique in comparison to a stretch and strap technique. Gray et al performed a metaanalysis of the literature to evaluate interventions for clubfeet. Evidence was limited because of limited use of outcome measures and lack of available raw data. From the data available they concluded that the Ponseti technique may produce better short-term outcomes compared with the Kite technique. Jowett et al performed a systematic review of the literature of the results of the Ponseti method and concluded that the original Ponseti method is the current best practice for the treatment of clubfeet with an initial correction rate of around 90%. Halanski et al performed a prospective comparative study comparing the Ponseti method with below knee casting followed by surgical release. They concluded that both had a relatively high recurrence rate but that the Ponseti cohort had significantly less operative interventions and required less revision surgery. Therefore they adopted the Ponseti

3. Developmental Dysplasia of the Hip (DDH)

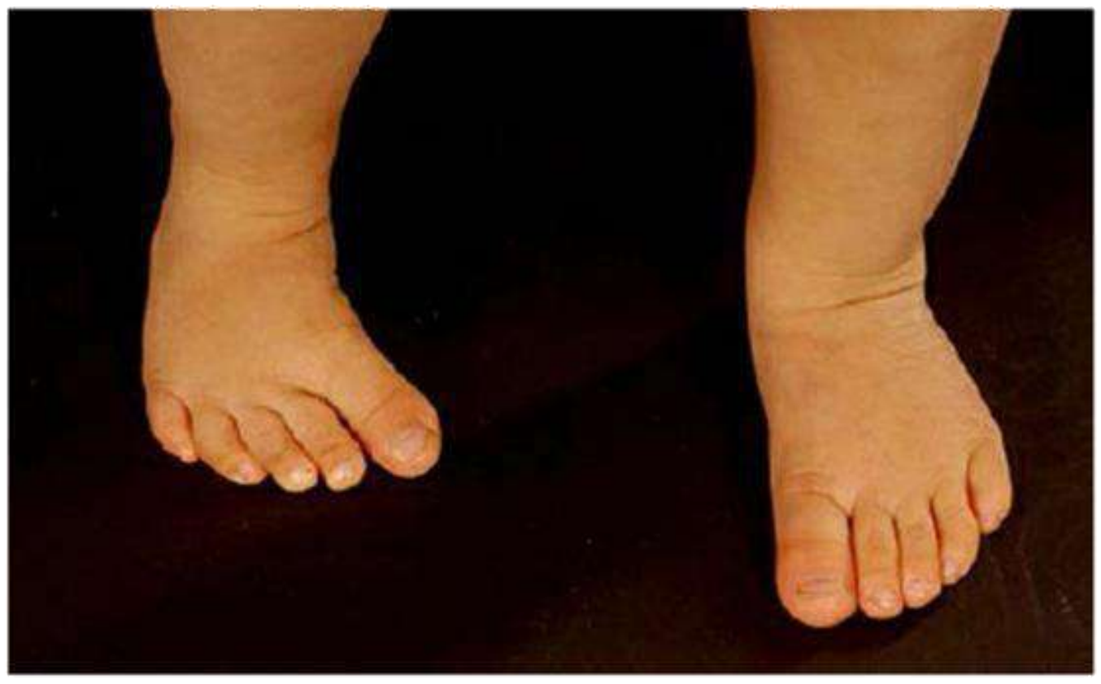


Dislocated hip in 3 years old girl.

What would be the clinical findings?

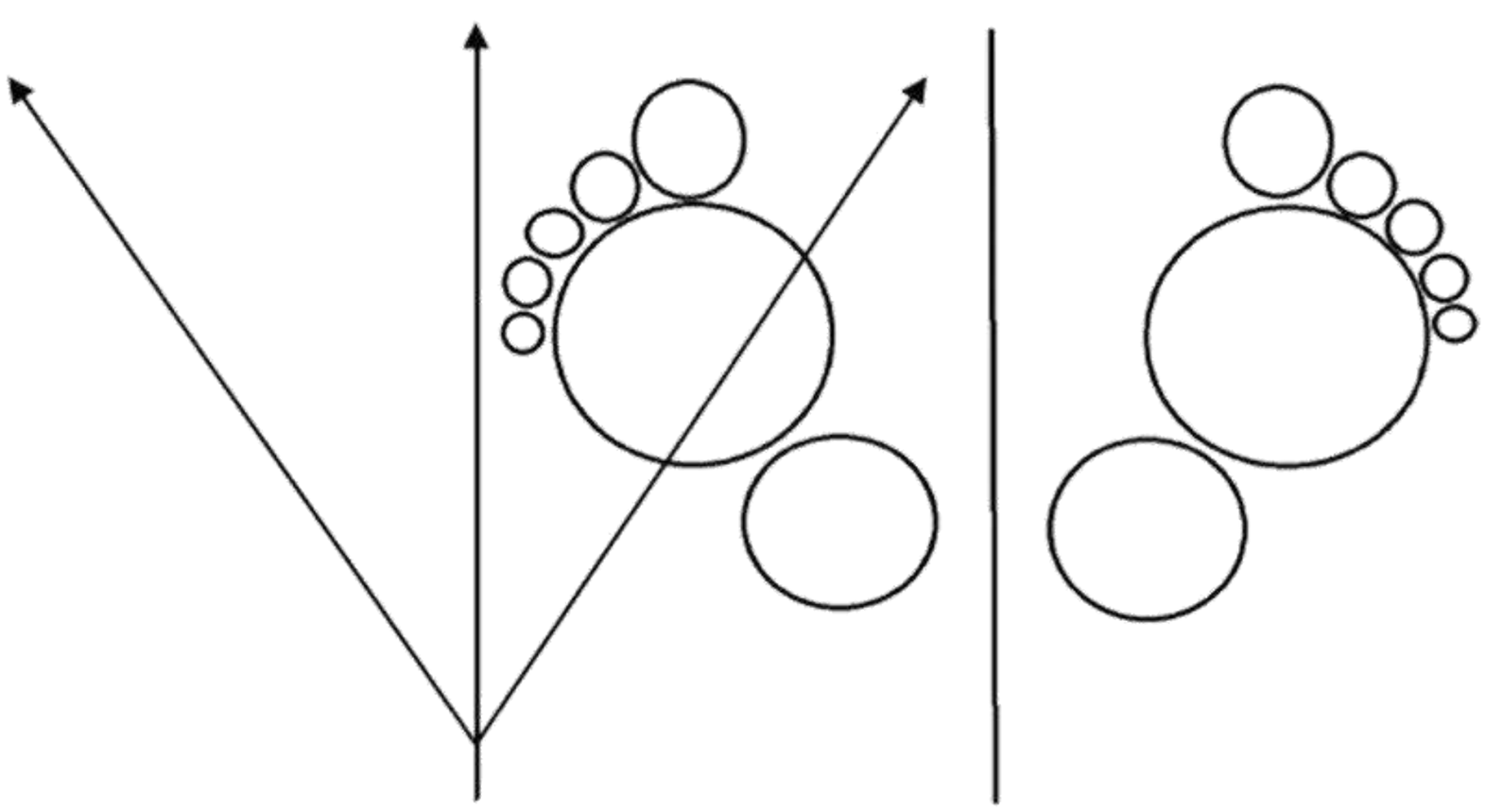
1) Apparent shortening and external rotation of the leg.
2) Reduced abduction of the affected hip.
3) There might be asymmetry of the thigh and/or buttock skin creases (dislocated hips can have symmetric looking creases and many normal hips have asymmetric creases).
4) The femoral head is most likely palpable in the buttock.
5) Barlow and Ortolani test are most likely negative since dislocated hips in older patients are usually irreducible. It is most likely possible to feel the femoral head moving within the buttock.

5. Picture of intoeing in 3 yr old.



Differential diagnosis of intoeing:
-foot deformity (metatarsus adductus, clubfoot)
-internal tibial torsion (normal in newborns [average of 4t internal rotation], usually better by 2 years of age and generally corrects by the age of 4 years). The medial and lateral malleolus lie in the same plane.
-femoral anteversion (adult angle of 10° to 20°).
-cerebral palsy
-hip dysplasia

Clinical examination
-Staheli rotational profile



1. Foot progression angle
-out-toeing = +
-in-toeing = -
-4 to 16 years: -8° t0 +16°
Lösel S, Burgess-Milliron M, Micheli L, Edington C. A simplified technique for determining foot progression angle in children 4 to 16 years of age. J Pediatr Orthop 1996;16(5):570-574

progression angle in children 4 to 16 years of age. J Pediatr Orthop 1996;16(5):570-574.

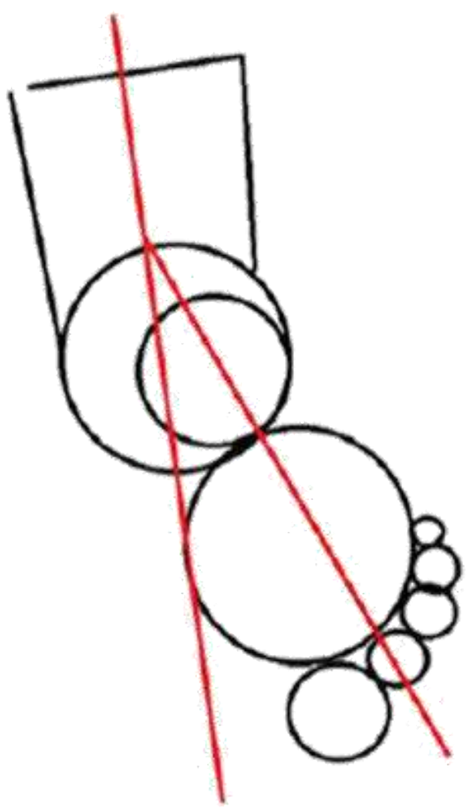
2. Hip/femoral rotation.

Test for femoral neck anteversion:patient positioned prone, one knee flexed to 90° at a time. Lower leg rotated from side to side. The examiner's other hand feels the greater trochanter. The prominence of the greater trochanter changes on the lateral side with the position of the lower leg. The leg is stopped in the position where the greater trochanter feels the most prominent. The angle between the vertical and the axis of the lower leg is the amount of femoral neck anteversion.

Age (years)	**Anteversion (degrees)**
Birth – 1 year	**30-50**
2	**30**
3-5	**25**
6-12	**20**
12-15	**17**
16-20	**11**

Internal rotation of >70° and limited external rotation are suggestive of increased femoral neck anteversion.

3. Thigh foot angle

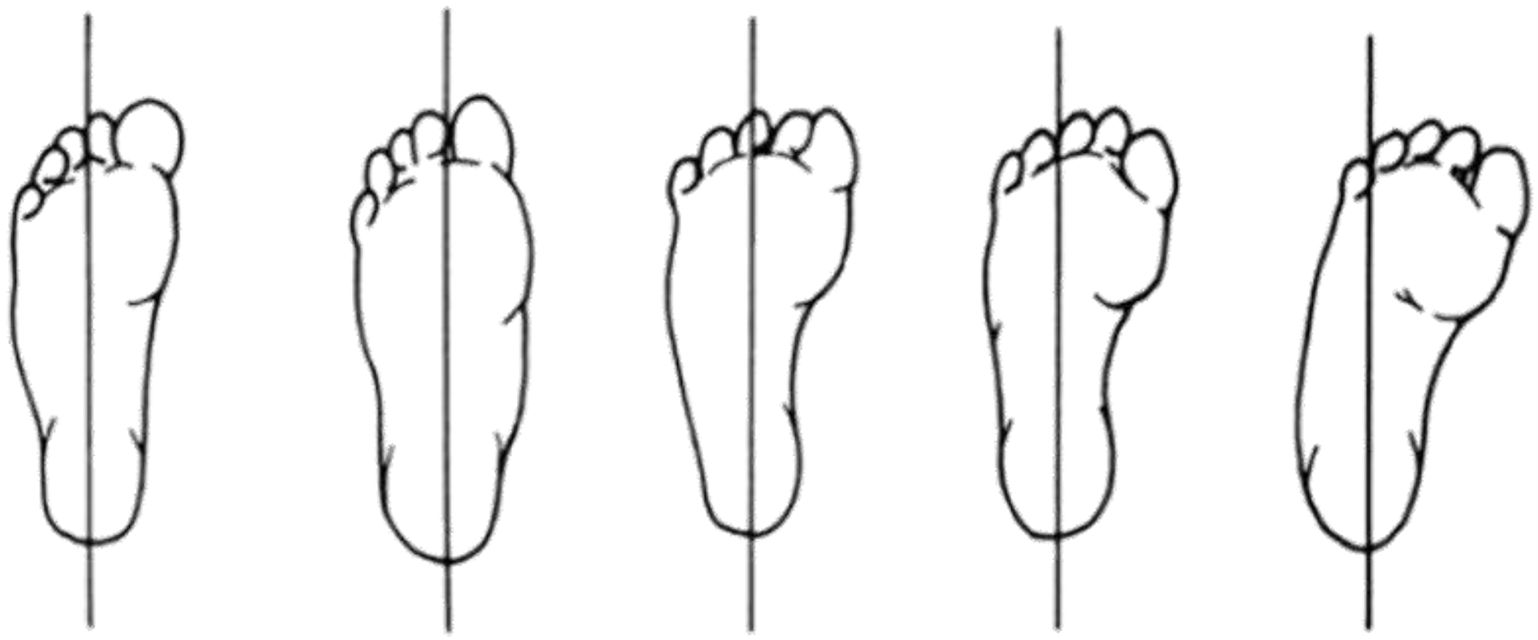


Age 1: -27 to +20°
Age 3: -15 to +25°
Age 5: -5 to +30°
Age 7: 0 to +30°
Age 9: +3 to +33°

The angle changes very little after the age of 8 years.

The angle changes very little after the age of 8 years.

4. 6 of 14 ades for metatarsus adductus:



-based on heel bisector line.
-mild: line lies along 3rd toe.
-moderate: line lies between 3rd and 4th toe.
-severe: line lies between 4th and 5th toe.

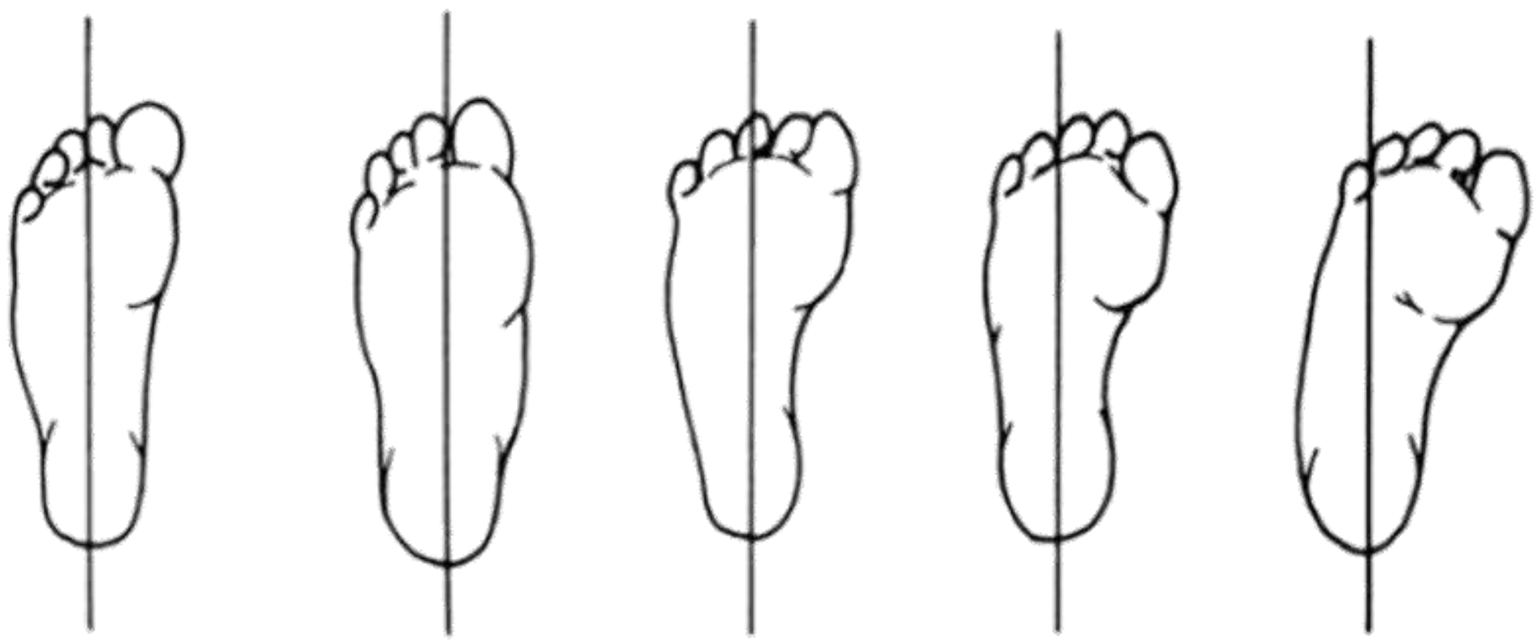
Bleck EE. Metatarsus aductus: classification and relationship to outcomes of treatment. J Pediatr Orthop 1983;3(1):2-9.

Metatarsus adductus angle:
-angle between longitudinal axis of the lesser tarsus (cuboid, navicular, cuneiforms) and axis of 2nd metatarsal
-mild deformity: 15-20°
-moderate deformity: 21-25°
-severe deformity: >25°

Investigations
CT scanogram:
-horizontal cuts through the hips, knees and ankles.

The angle changes very little after the age of 8 years.

4. Bleck grades for metatarsus adductus:



NORMAL VALGUS MILD MODERATE SEVERE

-based on heel bisector line.
-mild: line lies along 3rd toe.
-moderate: line lies between 3rd and 4th toe.
-severe: line lies between 4th and 5th toe.

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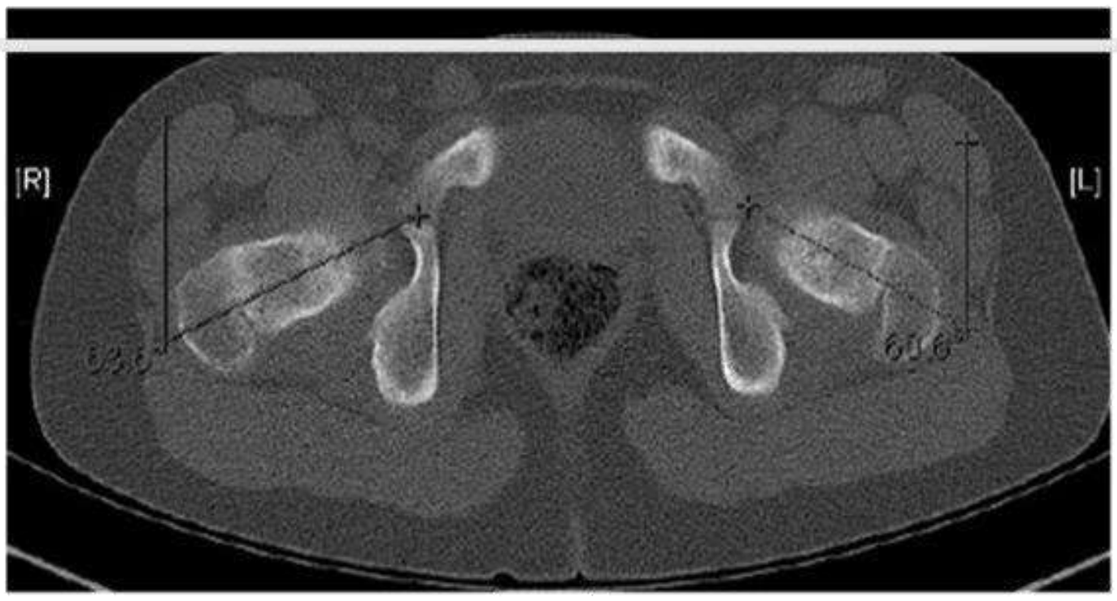
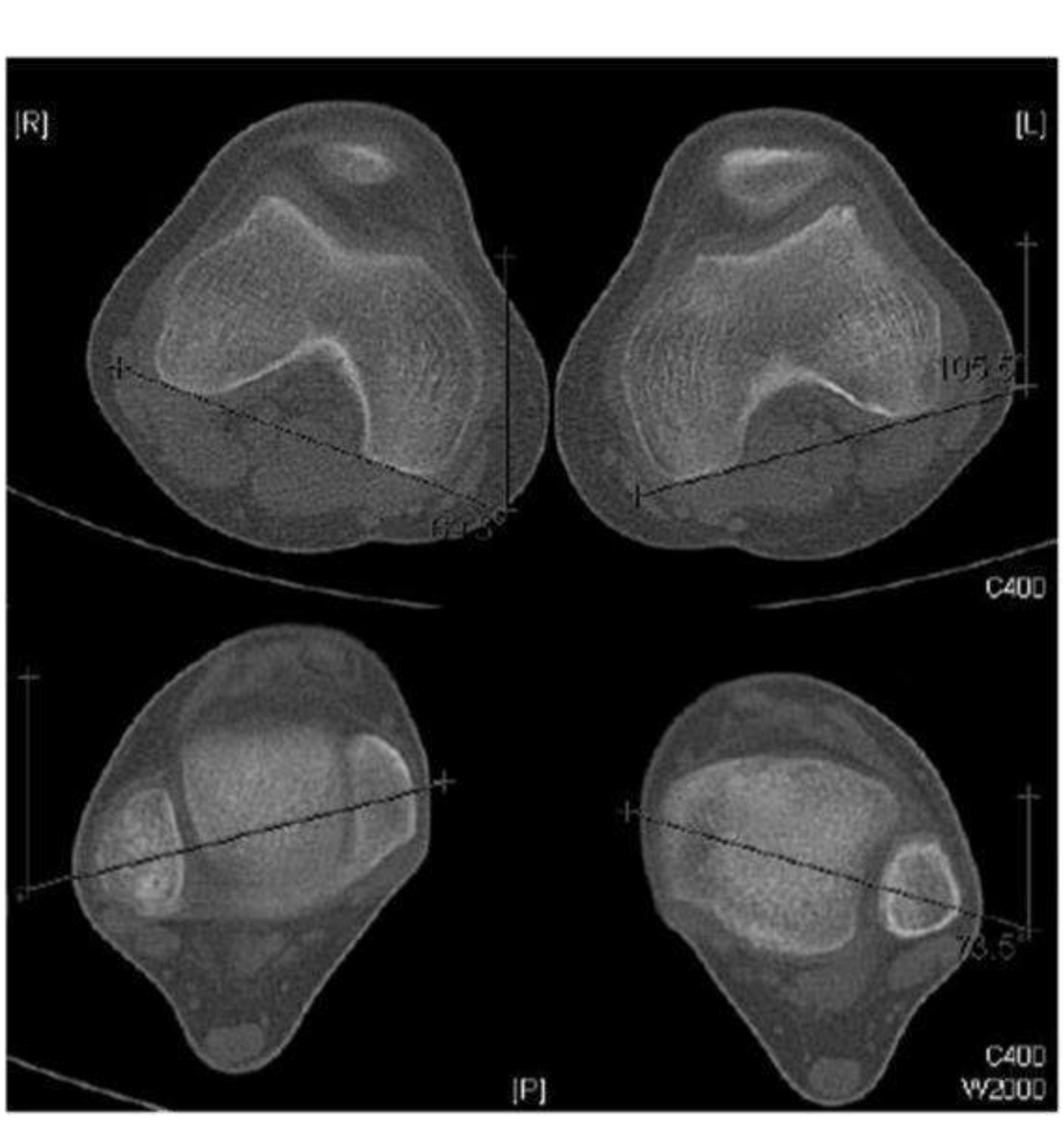




Management

Increased femoral neck anteversion and internal tibial torsion:

-Parent education. It needs to be explained that there are many children with similar findings.

-There is no evidence that non-operative management affects the rotational alignment. Management is therefore observation or surgical correction. The vast majority of patients are managed without surgery.

-Staheli proposed 5 indications for surgery in 1980: 1) age >8 years, 2) significant cosmetic and functional disability, 3) anteversion >50°, 4) medial hip rotation >85° and lateral rotation <10°, 5) family must be aware of the risks of surgery.

-The femoral osteotomy can performed at a subtrochanteric or supracondylar level.

-The tibial osteotomy can be performed distal to the tibial tuberosity or in the supramalleolar region (+/- fibular osteotomy). A prophylactic subcutaneous decompression of the anterior compartment is advisable to reduce the risk of compartment syndrome.

-Combined femoral and tibial correction might be necessary and parents/patients need to be informed about the possibility.

-In Tachdjian's Pediatric Orthopedics it is written: "Rotational osteotomies of the proximal tibial

-In Tachdjian's Pediatric Orthopedics it is written: "Rotational osteotomies of the proximal tibial region are contraindicated in a child younger than 8 years because of the possibility of a late valgus deformity occurring after the osteotomy in this region, similar to the Cozen fracture". No literature evidence is provided for this statement other than referring to the occurrence of valgus deformities following tibial fractures.

Herring JA, Moseley C. Posttraumatic valgus deformity of the tibia. J Peditr Orthop 1981;1:435.

Jordan SE, Alonso JE, Cook FF. The etiology of valgus angulation after metaphyseal fractures of the tibia in children. J Pediatr Orthop 1987;7:450.

Disorders of the leg. In: Tachdjian's Pediatric Orthopedics. 4th Ed. Saunders Elsevier: Philadelphia;2008:973-1033.

-Cozen's phenomenon: late valgus deformity following proximal tibial metaphyseal fractures.

Most surgeons reserve surgery for patients with significant cosmetic and functional disability and wait with surgery until the patient is about 8 to 10 years of age. There are exceptions. Surgery can be indicated in younger children where the intoeing results in the feet catching and the patients falling over frequently sustaining potentially serious injuries.

Staheli L. Medial femoral torsion: Experience with operative treatment. Clin Orthop Relat Res 1980; 146:222.

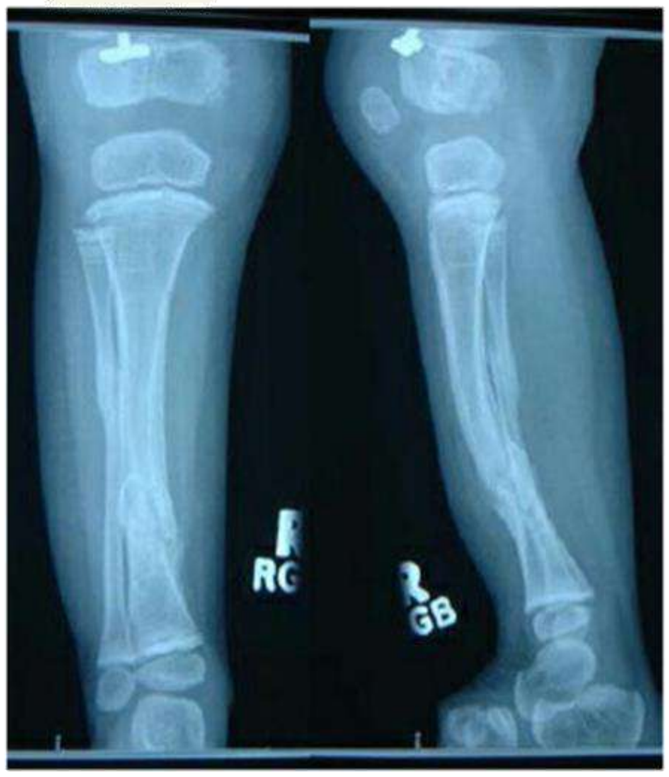
Metatarsus adductus:
-newborns with a metatarsus adductus deformity need to have a hip screening ultrasound because of there being an association with hip dysplasia.
-the majority of children with mild flexible deformities do not need any intervention since the deformity generally improves spontaneously. Sometimes stretching exercises and foot stimulation into active eversion are indicated. Serial casting should be performed very early if the deformity is resistant. Walkers can be provided with anti-varus shoes.

Treatment of the stiff metatarsus adductus can be very difficult without there being any consensus. If the deformity persists after the age of 6 to 8 years it is unlikely that a soft tissues procedure alone will be successful. A number of treatment options have been described:
-abductor hallucis release and capsulotomy of the first tarsometatarsal joint if the foot is resistant to casting.
-Heyman-Herndon release: release of the tarsometatarsal Joints in younger patients.
-metatarsal osteotomies.
-midfoot osteotomies: lateral column shortening (calcaneal or cuboid closing wedge osteotomy) and medial column lengthening (medial cuneiform opening wedge osteotomy).
-others.

6. X-ray of pseudarthrosis of tibia

7 .X ray of osteogenesis imperfect (OI).
-frequently called "Brittle Bone Disease".

9 of 14



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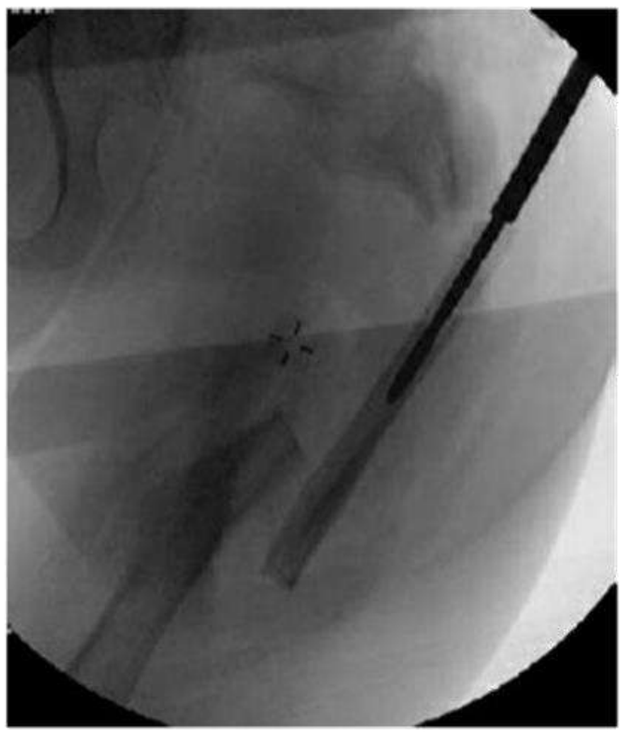
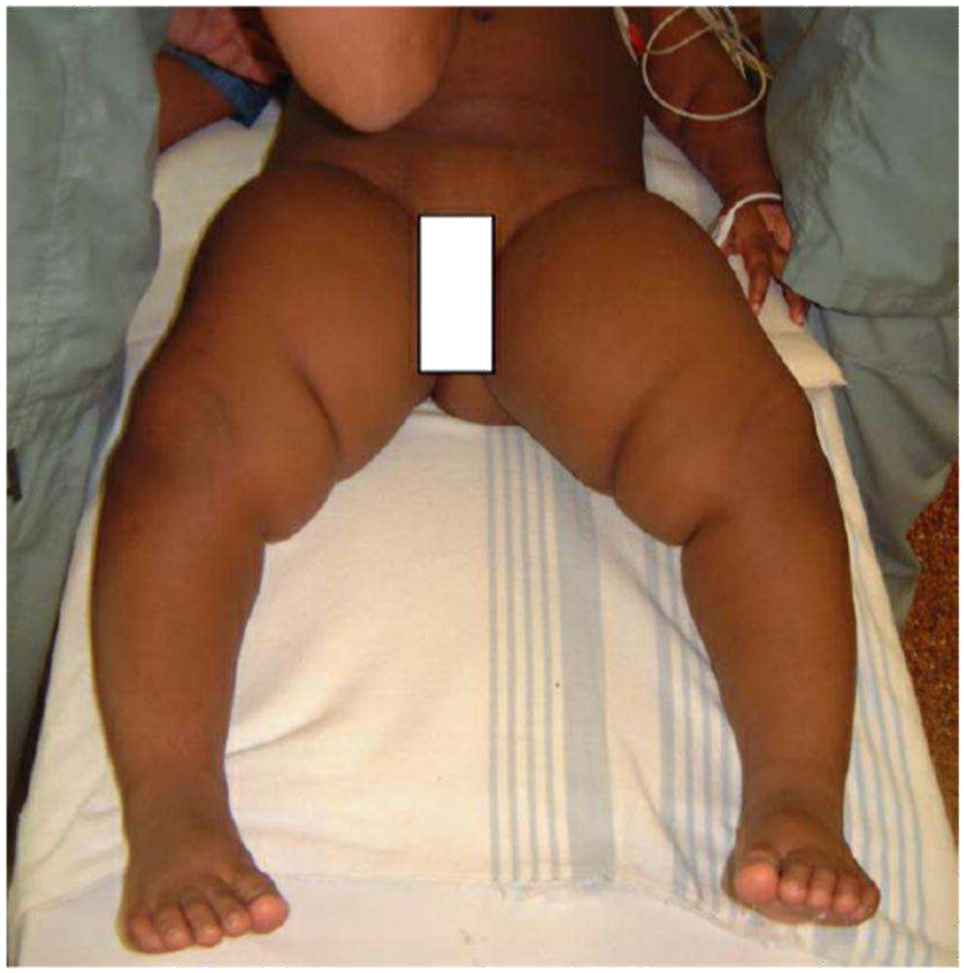
How will you manage.

The treatment involves:
1. Bracing
2. Bisphosphonates. Reduce fracture risk and increase bone mineral density.
3. Deformity corrections. Multilevel osteotomies are sometimes necessary (Sofield procedure / "sheek kabab technique" using intramedulalry rods) to correct severe deformities of IM rods.



10. Shown clinical picture of bow legs.



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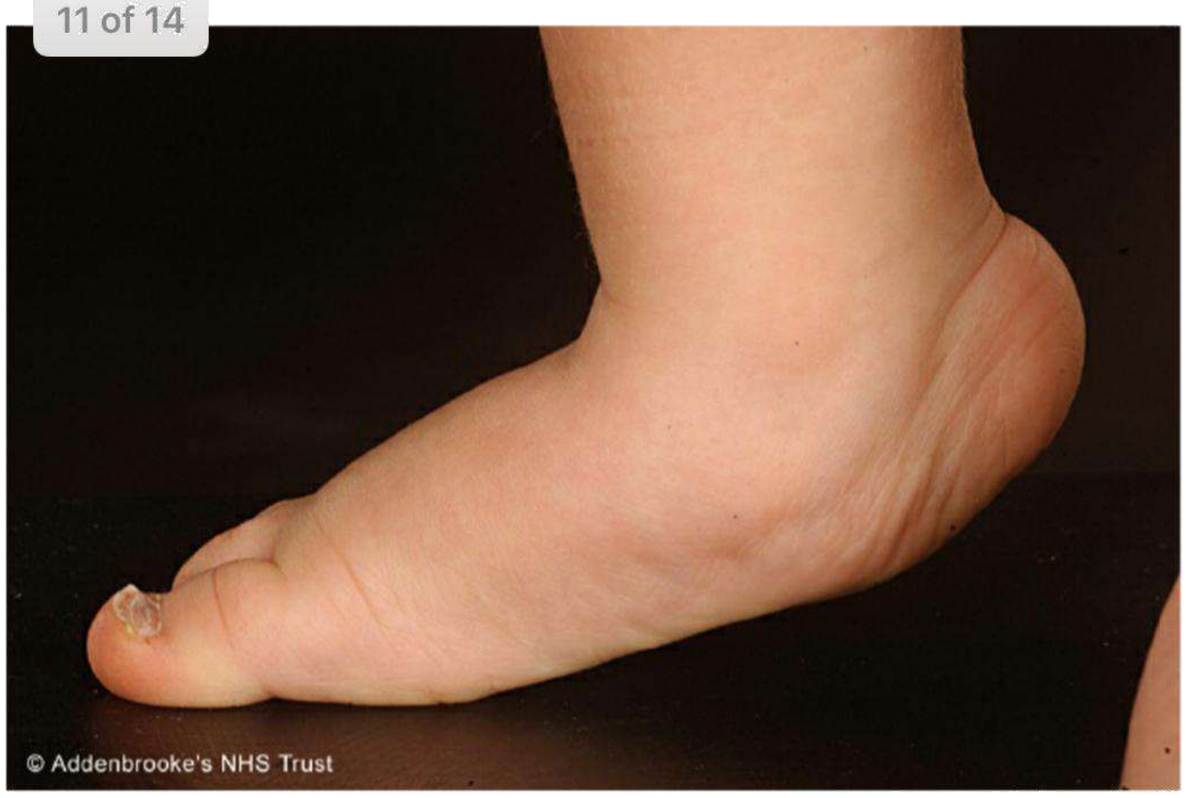
Differential Diagnosis:

-Physiological (tibio-femoral angle of at least 10°). Most newborns have a varus angle of 10°-15°. Maximal varus at 6-12 months. Neutral alignment by 18-24 months. Maximal valgus angle of 8° at 4 years. 6° by 11 years. A varus angle after 2 years of age is considered abnormal.
-Non-physiologic tibia vara (Blount's disease).
-metabolic bone diseases (Vitamin D deficiency – Rickets; the Department of health recommends Vitamin D supplements for children between 6 months and 5 years of age because of the increased incidence of Vitamin D deficiencies in the UK).
-physeal disturbance secondary to trauma or infection
-skeletal dysplasias
-fibrocartilagenous dysplasia
-Neurofibromatosis

11. Clinical picture of rocker bottom foot.



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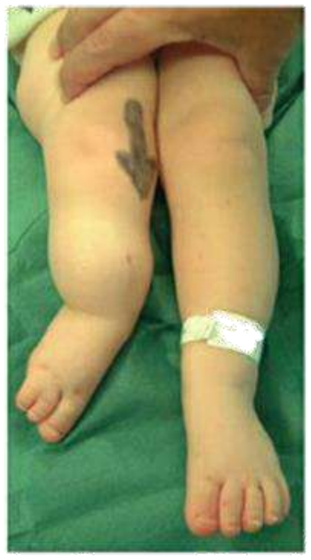
What do you see?
A foot with a rocker bottom shape. The hindfoot is in equinus (and also valgus), the fore- and midfoot are in extension.

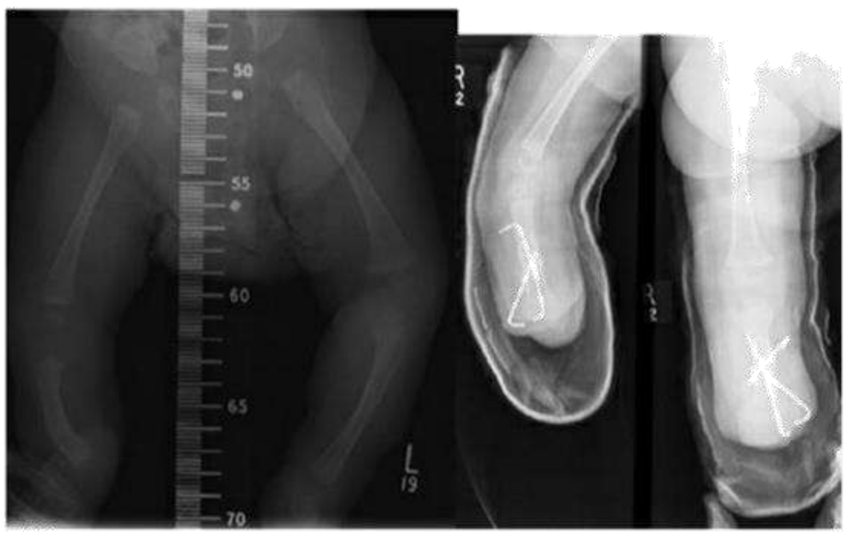
What is the differential diagnosis?
-congenital vertical talus (rigid flatfoot)
-incorrect treatment of clubfoot deformity
-calcaneo-valgus foot (flexible flatfoot)
-the foot is in extreme hyperextension

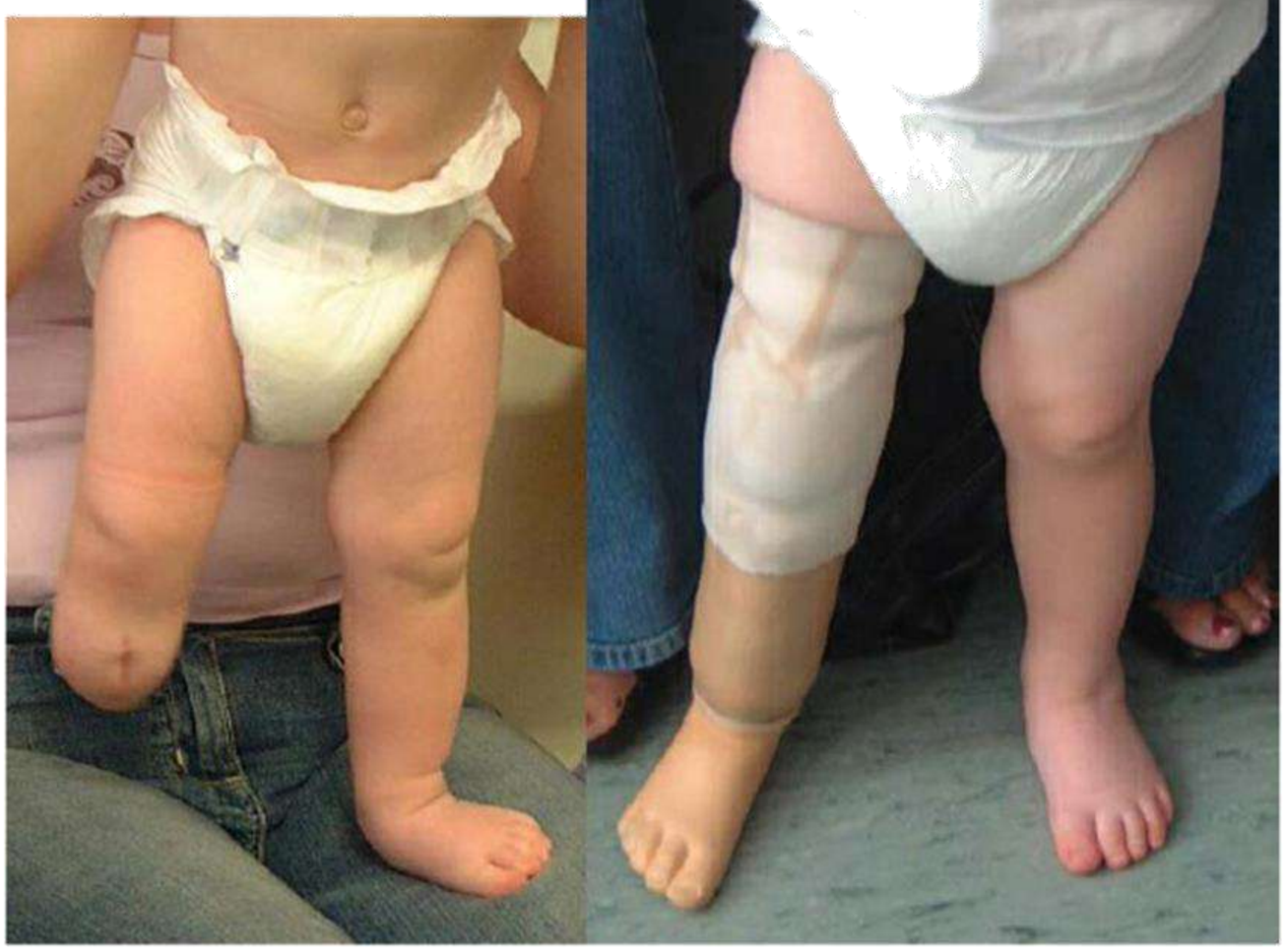
How do you differentiate congenital vertical talus from calcaneo-valgus foot?

-The vertical talus foot is rigid with a rigid dorsal dislocation of the navicular bone. The calcaneovalgus foot is flexible, responds well to stretching and the heel can be dorsiflexed.

12. Fibular hemimelia







-The term postaxial hypoplasia has been suggested.
-Minimal shortening to complete absence of the fibula.
-Most common congenital longitudinal lower limb deficiency.
-1 in 40000 live births.

Associations:
1. Proximal Femoral Focal Ddeficiency
2. Hypoplastic lateral femoral condyle
3. Femoral hypoplasia with external rotation
4. ACL and PCL deficiency
5. Patella subluxation
6. Genu valgus
7. Short and/or bowed tibia (anteromedial bow)
8. Ankle valgus
9. Absent lateral rays
10. Ball and socket ankle joint
11. Tarsal coalitions
12. Coxa vara
13. Absent foot rays

Classification:
Achterman and Kalamchi:
Type I: part of fibula present.
Type II: fibula is absent.

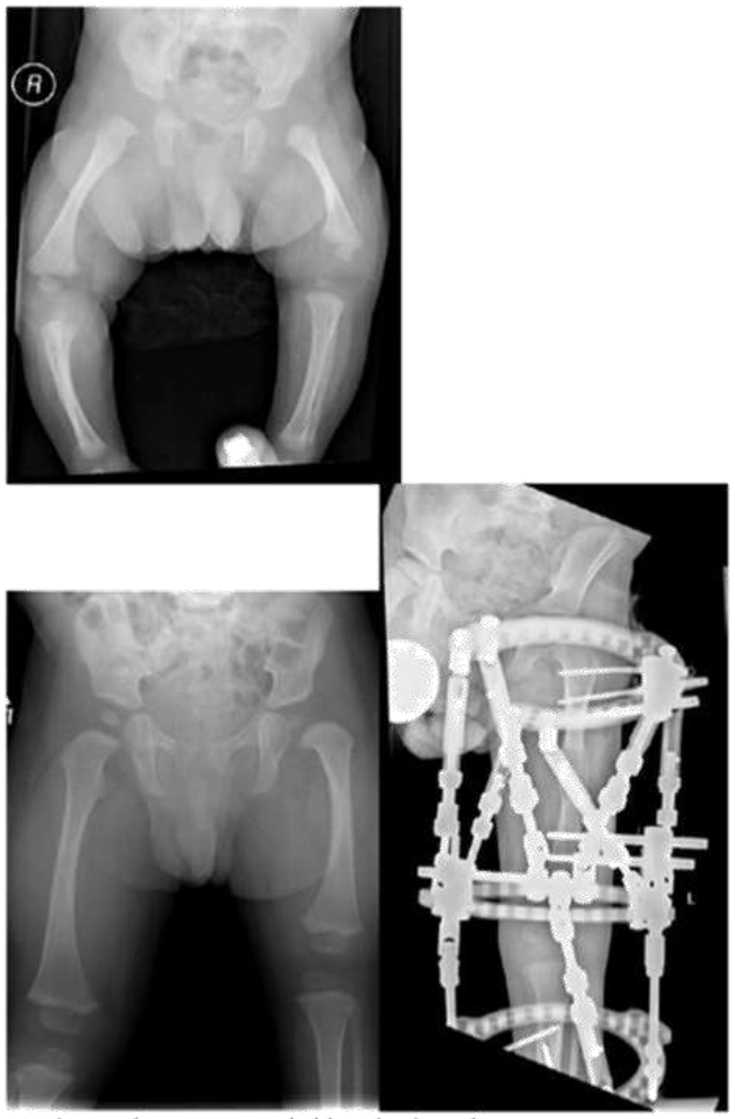
Achterman C, Kalamchi A. Congenital deficiency of the fibula. J Bone Joint Surg-Br 1979;61(2):133-7.

Stanitski:

Fibula:
I=nearly normal fibula; II=small or miniature fibula;III=complete absence of the fibula.
Tibiotalar joint and distal tibial epiphyseal morphology:H=horizontal; V=valgus (triangular distal tibial epiphysis; S=spherical (ball and socket).
Presence of a tarsal coalition = “c”.
Number of foot rays: 1-5

Stanitski DF, Stanitski CL. Fibular hemimelia: a new classification system. J Pediatr Orthop 2003;23(1):30-4.

13. Proximal Femoral Focal Deficiency (PFFD)



-The shortened limb is the most apparent functional deficit.
-The femur is flexed, abducted and externally rotated (secondary to femoral neck retroversion). The knee therefore remains flexed.
-Varying degrees of hip deficiencies (coxa vara to no hip joint).
-Generalised deficiency of the hip muscles even in stable hips.
-Varying degrees of knee instability (deficient ACL/PCL).
Patella may be absent or small and high riding.
Patellofemoral joint may be laterally subluxed or dislocated.
-Flexion and valgus deformity of the knee.
-Foot function depends on if there are associated deficiencies such as fibular hemimelia.
-It is thought that the congenitally short femur is part of the PFFD spectrum.

Aitken Classification:
A: Coxa vara; short femur with lateral bowing of upper third of femur. Adequate acetabulum that contains the head. A pseudoarthrosis develops at the subtrochanteric region that usually fuses with the shaft at maturity. B: Ossification of femoral head epiphysis is delayed. Acetabulum mildly dysplastic. Upper end of femoral shaft lies above femoral head. The junction between femoral head and shaft is by defective cartilage that fails to ossify by skeletal maturity. C: Markedly dysplastic acetabulum. The femoral head never ossifies. The femoral shaft is very short and its upper end tapers sharply to a point. The hip is very unstable D: Femoral head and acetabulum are absent. The femur is represented

8.Foot & Ankle :

A.Oral :

Dorsal Layer

- Extensor digitorum brevis

First Plantar Layer

- Most superficial of all the layers
- Muscles
  - abductor hallucis
  - flexor digitorum brevis (FDB)
  - abductor digiti minimi

Second Plantar Layer

- Muscles
  - quadratus plantae
  - lumbrical muscles
- Tendons
  - flexor digitorum longus (FDL)
  - flexor hallucis longus (FHL)
- Neurovascular structures
  - medial and lateral plantar arteries

Third Plantar Layer

- Muscles
  - flexor hallucis brevis
  - oblique and transverse heads of the adductor hallucis
  - flexor digiti minimi brevis

Fourth Plantar Layer

- Deepest layer
- Muscles
  - dorsal interosseous
  - plantar interosseus
- Tendons
  - peroneus longus
  - tibialis posterior
    - both tendons travel to their insertion point via fibro-osseus tunnels

- Neuromuscular disorders
  - Muscular dystrophy
  - Charcot Marie Tooth disease
  - Friedriech's ataxia
  - Roussy-Levy syndrome
- Central nervous system disorders
  - Cerebral palsy
  - Poliomyelitis
- Spinal pathology
  - Spinal dysraphism
  - Diastematomyelia
  - Spina bifida
  - Myelomeningocele
  - Syringomyelia
  - Spinal cord tumour
- Muscle pathology
  - Crush injury
  - Burns
  - Compartment syndrome

- Foot drop
  - Inability to dorsiflex at the ankle and/or toes
  - Commonly result from peroneal nerve palsy
    - Multiple etiologies
      - central nervous system (brain, spinal cord, nerve roots)
      - peripheral nervous system (sciatic nerve, peroneal nerve)
      - traumatic (knee dislocation, laceration, blunt trauma)
      - compressive (compressive mass, deformity correction)
      - systemic (diabetic polyneuropathy, mononeuropathy)
      - Iatrogenic (laceration, casting, positioning, surgical injury)
      - Mechanical (muscle debridement, tumor excision, etc.)
  - Presentation - variable depending on location of nerve injury
    - Motor
      - Loss of ankle/toe dorsiflexion (DPN)
      - Loss of ankle eversion (SPN)
    - Sensory
      - Loss of first dorsal webspace sensation (DPN)
      - Loss of lateral leg/dorsal foot sensation (SPN)
  - Treatment
    - Nonoperative
      - Observation
      - AFO bracing
      - Therapy - stretching and supple joints
    - Operative
      - Acute injury
        - Laceration - repair, grafting, or nerve transfer
      - Chronic injury
        - Tendon transfer
          - Posterior tibial tendon transfer to lateral cuneiform +/- gastroc or Achilles tendon lengthening ? ?

Classification

Wagner Classification and Treatment		
	Description	*Treatment*
Grade 0	Skin intact but bony deformities lead to "foot at risk"	Shoe modifications with serial exams
Grade 1	Superficial ulcer	Office debridement and contact casting
Grade 2	Deeper, full thickness extension	Operative formal debridement and contact casting
Grade 3	Deep abscess formation or osteomyelitis	Operative formal debridement and contact casting
Grade 4	Partial Gangrene of forefoot	Local vs. larger amputation
Grade 5	Extensive Gangrene	Amputation

Brodsky Depth-Ischemia Classification and Treatment		
Depth		
Classification	*Definition*	*Treatment*
0	At risk foot, no ulceration	Patient education, accommodative footwear, regular clinical examination
1	Superficial ulceration, not infected	Off-loading with total contact cast, walking brace or special footwear
2	Deep ulceration, exposing tendons or joints	Surgical debridement, wound care, off-loading, culture-specific antibiotics
3	Extensive ulceration or abscess	Debridement or partial amputation, off-loading, culture-specific antibiotics
Ischemia		
A	Not ischemic	
B	Ischemia without gangrene	Non-invasive vascular testing and vascular reconstruction with angioplasty/bypass
C	Partial forefoot gangrene	Vascular reconstruction and partial foot amputation
D	Complete gangrene	Complete vascular evaluation and major extremity amputation

Clinical evaluation

Cosmetic

"Do nothing"

Shoe wear problems

"modify shoes"

Pain

"evaluate and treat"

Pain

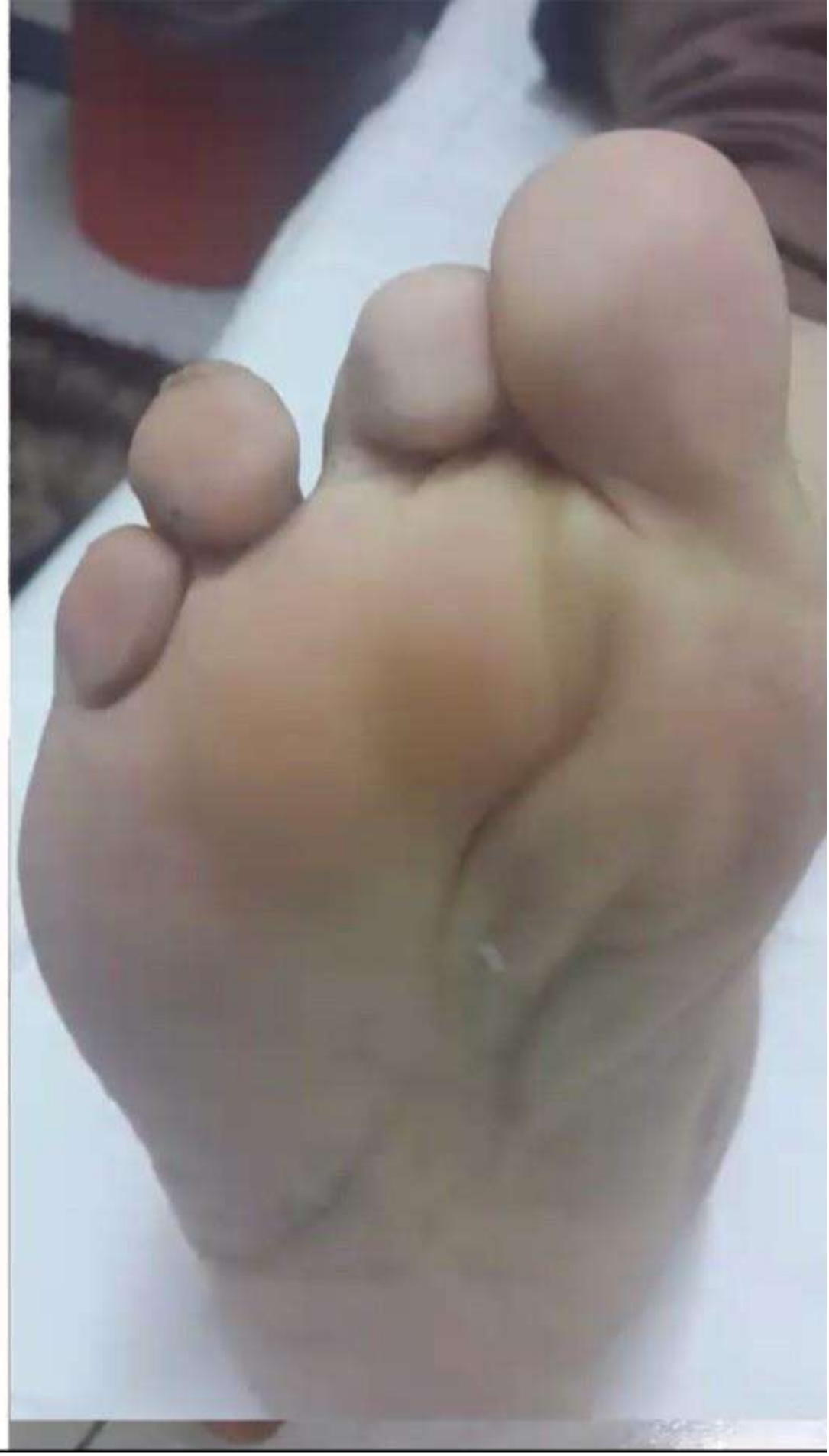
Causes:

Inflamed bunion

Metatarsalgia

Painful callosities

Osteoarthritis of MP joint



Grading

	HV angle	IMT angle
Normal	<15deg.	<9deg.
Mild	15-20deg.	9-11deg.
Moderate	20-40deg.	11-18deg.
Severe	>40deg.	> 18deg

Each grade is classified into :

- Congruent or non congruent according to PASA
- Arthritic / non Arthritic

You have to know the principles of these procedures

Modified Mc BBride
Bunionectomy
Chevron osteotomy (DMO)
Scarf osteotomy (Diaphyseal osteotomy)
Basal osteotomy
Lapidus procedure
Akin osteotomy
1st MTP fusion
Keller's procedure

Treatment algorithm

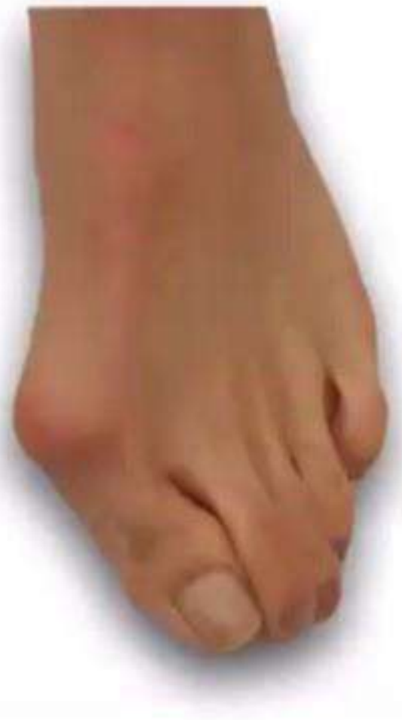
Adjuvant procedures
- McBride release
- Akin osteotomy

Non arthritic joint

Mild
DMO : chevron

Moderate
Shaft Osteotomy : Scarf
PMO

Severe
Double osteotomy
Lapidus



Hypermobile first ray
- Lapidus

Arthritic joint

old inactive
Keller procedure

Young active
MP fusion
Arthroplasty

B.Viva : Long & Short :

Section 2 Adult Elective Orthopaedics and Spine

Chapter 4

Foot and ankle structured oral questions

N. Jane Madeley and Neil Forrest

Structured oral examination question 1: Lateral ligament instability of the ankle

EXAMINER. Tell me what this diagram represents and name the structures labelled 2, 3 and 5. (Figure 4.1.)

CANDIDATE. This diagram is a representation of the lateral aspect of the ankle showing the bony and ligamentous structures. Structure 2 is the anterior talofibular ligament, structure 3 is the calcaneofibular ligament and structure 5 is the posterior distal tibiofibular ligament.

EXAMINER. What structures are injured in a lateral ligament injury?

CANDIDATE. The mechanism is usually a rotational injury with sequential failure of the ligaments from front to back, hence the anterior talofibular ligament or ATFL is most commonly injured followed by the calcaneofibular ligament or CFL and the posterior talofibular ligament is the least frequently injured.

EXAMINER. How would you go about diagnosing a lateral ligament injury to the ankle?

CANDIDATE. In the acute setting I would expect the patient to give a history of an episode of a twisting incident resulting in significant pain and swelling. There may be a history of recurrent sprains and instability. Acutely the lateral side of the ankle would be swollen and tender anterior and inferior to the tip of the fibula but discomfort may make it difficult to elicit a definite sign of instability.

In a patient with a more chronic history the clinical sign of instability would be a positive anterior drawer test or talar tilt test.

EXAMINER. Tell me more about those two tests.

CANDIDATE. The patient is examined sitting with their legs over the edge of the couch or sitting in a chair to relax the gastrocnemius soleus complex. For the anterior drawer test the distal tibia is grasped in one hand and the other hand grasps the heel and the foot is drawn anteriorly in relation to the talus. Pain or excess anterior translation or a sulcus sign developing at the anterolateral corner of the ankle are signs of an ATFL injury. The other ankle must be examined for comparison. The talar tilt test involves inversion of the ankle whist palpating the anterolateral corner of the joint to feel for movement of the talus within the mortise.

A lack of firm end point or tilt in excess of the normal side would represent instability and the CFL is considered to have been injured if this test is positive.

EXAMINER: What other clinical findings may be positive in a patient with recurrent ankle sprains?

CANDIDATE: Ankle sprains are more common in patients with a cavus foot deformity or hypermobility.

EXAMINER: If you suspect a lateral ligament injury how will you proceed in managing this patient?

Figure 4.1 Diagram of the lateral ankle ligaments. 1, Anterior inferior tibiofibular ligament; 2, Anterior tibiofibular ligament; 3, Calcaneofibular ligament; 4, Posterior talofibular ligament; 5, Posterior inferior tibiofibular ligament; 6, Achilles tendon.

Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination, ed. Paul A. Banaszkiewicz and Deiary F. Kader. Published by Cambridge University Press.

CANDIDATE: The first step in management would be rehabilitation with physiotherapy, concentrating on peroneal strengthening and proprioceptive training. If the dynamic stabilizers of the ankle are well conditioned the majority of patients recover well from a ligament injury. Bracing may be of benefit.

EXAMINER: . . . and if the patient continues to have significant symptoms despite adequate rehabilitation?

CANDIDATE: A patient that fails to recover would need more investigation. I would begin with simple weightbearing radiographs of the ankle. Stress X-rays of the ankle may be diagnostic for diagnosing a ligament injury, however if the patient is still having significant pain and swelling I would request an MRI scan to look for additional pathology.

EXAMINER: What other conditions would you be looking for?

CANDIDATE: My differential diagnosis for an ankle sprain that doesn't get better, in addition to incomplete recovery or rehabilitation would be peroneal tendon pathology such as a split tear or subluxing tendons, intra-articular pathology such as an osteochondral defect of the talus or loose body, or non-union of an anterior calcaneal process fracture in addition to the presumed diagnosis of lateral ligament injury.

EXAMINER: What are the surgical options for management of an isolated lateral ankle ligament complex injury in a young patient who has failed to respond to non-operative treatments?

CANDIDATE: The options would be a lateral ligament repair or reconstruction of the lateral ligaments.

EXAMINER: Do you know any methods of surgical repair?

CANDIDATE: Yes, the Broström repair.[1]

EXAMINER: What are the principles of that operation?

CANDIDATE: It is an anatomical repair of the lateral ligaments. The ATFL and CFL are imbricated to re-tension them. The extensor retinaculum may then be sutured over the top of the repair for additional strength. Consideration should be given to performing an ankle arthroscopy first at the same sitting to diagnose and address any associated intra-articular pathology.

EXAMINER: Are intra-articular lesions common in this group?

CANDIDATE: Various studies have found chondral injures in a significant proportion of chronic ankle instability. In one study associated intra-articular pathology amenable to arthroscopic treatment was identified in 83% of patients undergoing Brostrom repair.[2]

EXAMINER: A patient asks how successful a ligament repair will be, what will you tell them?

CANDIDATE: I would expect a successful result in over 80% of patients.

EXAMINER: Tell me about the options available for lateral ligament reconstruction.

CANDIDATE: There are several operations described for lateral ligament reconstructions. The majority of these involve sectioning the anterior half of the peroneus brevis proximally, leaving the distal end attached and routing the free end to reconstruct a lateral ligament. The reconstruction can be anatomical or non-anatomical. I have experience of the modified Chrisman–Snook procedure which transfers the anterior half of the peroneus brevis through bone tunnels in the distal fibula and lateral calcaneum to form an anatomical reconstruction of both the ATFL and CFL. I understand that many surgeons are moving towards reconstruction using a free hamstring graft in athletes to avoid harvesting one of the dynamic ankle stabilizers.[3]

EXAMINER: Thank you.

1. Broström L. Sprained ankles VI: Surgical treatment of 'chronic' ligament ruptures. *Acta Chirurgica Scand* 1966;**132**(5):551–565.
2. Kibler WB. Arthroscopic findings in ankle ligament reconstruction. *Clin Sports Med* 1996;**15**(4):799–804.
3. Boyer DS, Younger AS. Anatomic reconstruction of the lateral ligament complex of the ankle using a gracilis autograft. *Foot Ankle Clin* 2006;**11**(3):585–595.

Structured oral examination question 2: Ankle arthritis

EXAMINER: Describe the findings on this X-ray. (Figure 4.2.)

CANDIDATE: This is an AP weightbearing 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 and varus angulation of the ankle.

EXAMINER: Excellent, what are the most common causes of end-stage arthritis of the ankle?

CANDIDATE: Primary osteoarthritis is thought to be relatively uncommon and the most common cause of ankle arthritis is probably post-traumatic arthritis. Other causes are inflammatory arthritis and septic arthritis.

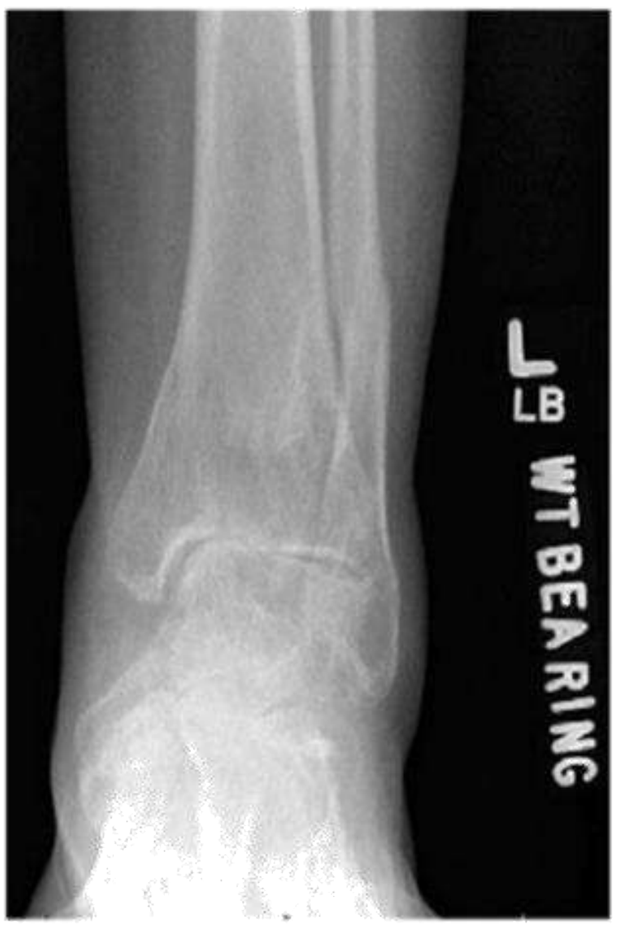


Figure 4.2 X-ray showing ankle arthritis.

EXAMINER: How is this patient likely to present?

CANDIDATE: They are most likely to complain of pain, however they may also present with restriction of movement, deformity and difficulty in performing activities of daily living (ADLs).

EXAMINER: Are you aware of any classification systems for arthritis of the ankle?

CANDIDATE: No, I am not aware of any classification systems specific to the ankle. The Kellgren and Lawrence Radiographic Criteria can be used.[1]

EXAMINER: The X-ray you have been shown belongs to a 42-year-old manual worker who had an ankle fracture 7 years ago which was managed non-operatively. Describe your management strategy for this patient.

CANDIDATE: I would first want to perform a full history and examination, and obtain a lateral radiograph.

EXAMINER: Absolutely. Tell me about the management options available for ankle arthritis.

CANDIDATE: I would start with conservative measures and optimize the patient's analgesia adding in NSAIDs, and suggest activity modification. He could try footwear modification with a cushioned sole and rocker-bottom shoe which may improve his symptoms as may use of an ankle brace or AFO. Similarly an injection of intra-articular steroid or viscosupplementation may be of symptomatic benefit. Physiotherapy could be an adjunctive treatment in patients with symptoms of instability or weakness but may aggravate symptoms.

EXAMINER: What surgical options are available?

CANDIDATE: There are two types of surgical option available, those aimed to 'buy time' or provide temporary relief and definitive treatments. The temporizing measures are debridement of the joint which can be performed arthroscopically or open depending on the extent of disease and should be aimed at treating identifiable causes of symptoms such as removing loose bodies, trimming anterior osteophytes which may give impingement symptoms, or debriding loose areas of articular cartilage and areas of synovitis. The other option is distraction arthroplasty.[2] The definitive surgical options are ankle fusion or ankle replacement.

EXAMINER: Isn't fusion an outdated treatment now that ankle replacements are available?

CANDIDATE: No, total ankle replacements are not suitable for every patient and ankle fusion is still considered the 'gold standard'.

EXAMINER: So which patients should be considered for ankle replacement surgery?

CANDIDATE: Ankle replacement surgery could be considered in low-demand patients over the age of 60 years who have inflammatory arthritis or osteoarthritis. Bilateral disease or arthritis affecting adjacent joints is a relative indication. Contraindications would include younger, more active patients, significant ankle instability, particularly deltoid ligament insufficiency, significant deformity, especially varus or valgus of more than 10°, peripheral vascular disease, a poor soft tissue envelope, marked osteoporosis or avascular necrosis of the tibial plafond or talar dome.

EXAMINER: Do you know anything about the types of ankle replacement available?

CANDIDATE: The earlier designs involved a two-component design such as the Agility total ankle replacement, which required fusion of the distal tibiofibular joint. Most modern designs are three-component uncemented mobile bearing prostheses.

EXAMINER: A patient wants to know how long an ankle replacement will last. What will you tell them?

CANDIDATE: The 10-year survival is approaching 85% but there are fewer data available than for knee and hip replacements. Many series are small.[2–5]

EXAMINER: The 42-year-old patient we began by discussing wants an ankle replacement. What would you tell him?

CANDIDATE: He is a young patient, in a manual job. He wouldn't be a candidate for total ankle replacement and I would explain to him that if his symptoms have failed to be controlled by non-operative measures and he requires definitive surgical treatment then an ankle fusion would be a better option for him.

EXAMINER: He still wants a replacement, as he is keen to get back to hill walking and sports and doesn't want a stiff ankle. What will you tell him now?

CANDIDATE: He would be at risk of early failure with an ankle replacement due to his young age and level of activity. A fusion would provide a stable pain-free ankle that would allow him to return to the majority of activities that he wishes to do. I would explain that many patients return to sports after ankle fusion. I would also explain that an ankle fusion would only sacrifice the residual movement that he has at his ankle joint and that his subtalar, midfoot and forefoot movements would still be present.

EXAMINER: What position should his ankle be fused in?

CANDIDATE: The foot should be plantigrade with a physiological 5° of hindfoot valgus and 5° of external rotation.

EXAMINER: What complications will you warn him about?

CANDIDATE: Non-union, malunion, delayed union, infection, wound-healing problems, nerve or vessel damage, DVT/PE, risk of exacerbating or developing arthritis in other joints.

EXAMINER: Thank you.

1. Kellgren JH, Lawrence JS. Radiological assessment of osteoarthrosis. *Ann Rheum Dis* 1957;**16**:494–501.
2. van Valberg AA, van Roermund PM, Marijnissen AC *et al.* Joint distraction in treatment of osteoarthritis: a two-year follow-up of the ankle. *Osteoarthritis Cartilage* 1999;7:474–479.
3. Wood PLR, Prem H, Sutton C. Total ankle replacement: medium term results in 200 Scandinavian total ankle replacements. *J Bone Joint Surg Br* 2008;**90-B**:605–609.
4. Bonnin M, Gaudot F, Laurent JR *et al.* The Salto total ankle arthroplasty: survivorship and analysis of failures at 7 to 11 years. *Clin Orthop Relat Res* 2011;**469**:225–236.
5. Mann JA, Mann RA, Horton E. STAR ankle: long-term results. *Foot Ankle Int* 2011;**32**(5):473–484.
6. Labek G, Klaus H, Schlichtherle R *et al.* Revision rates after total ankle arthroplasty in sample-based clinical studies and national registries. *Foot Ankle Int* 2011;**32**(8):740–745.

Structured oral examination question 3: The rheumatoid foot

EXAMINER: Please have a look at this radiographic print and tell me what you see. (Figure 4.3.)

CANDIDATE: This is an AP radiograph of a forefoot. There is hallux valgus with displacement of the second toe and destructive change of all the metatarsophalangeal joints. I cannot say whether this is a weightbearing film or not as it is not labelled. The intermetatarsal angle appears increased and I would normally measure this on a weightbearing film. There may be deformities of the lesser toes and I would like to see a lateral view to clarify this.

EXAMINER: Good. A lateral view would be very helpful. What do you think is the underlying diagnosis?

CANDIDATE: The destructive changes suggest that this is an inflammatory polyarthropathy such as rheumatoid arthritis.

EXAMINER: Could it be anything else?

CANDIDATE: The appearances could be secondary to a neuropathic process.

EXAMINER. What might be the commonest neuropathic process that could cause these appearances?

CANDIDATE: A peripheral neuropathy such as that associated with diabetes mellitus would be commonest.

EXAMINER: How would you confirm your diagnosis?

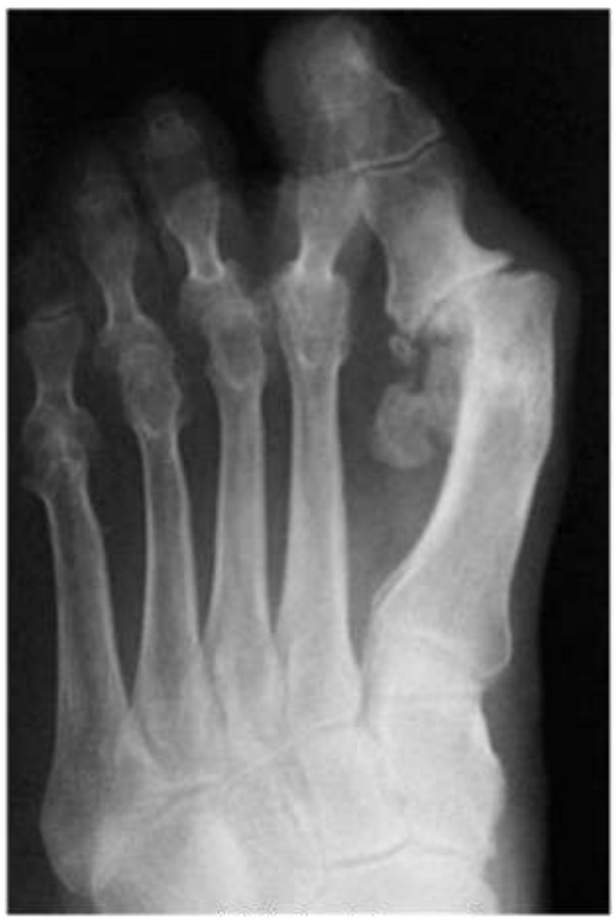


Figure 4.3 Anteroposterior (AP) radiograph of rheumatoid forefoot.

CANDIDATE: A detailed history would be most informative. Specifically, I would enquire about pain, swelling, sensory alteration and medical history.

EXAMINER: OK. This lady gives a clear history of progressive, painful, bilateral small joint swelling and post-immobility stiffness. She has great difficulty finding comfortable shoes and describes walking as if on pebbles. She is not aware of any diabetes or sensory loss. What are your thoughts at this stage?

CANDIDATE: This appears to be an inflammatory arthropathy.

EXAMINER: Yes. Her feet are making her life pretty miserable and she would like you, as an orthopaedic surgeon, to do something to make them better. Your examination finds marked active synovitis and plantar tenderness under the metatarsal heads as well as a minimally correctable hallux valgus. There is some hammering of the lesser toes with a cock-up deformity of the second toe. Sensation and perfusion appear good. What are you going to do?

CANDIDATE: First, I would want to know if she is known to a rheumatology service and has had any attempt at non-operative intervention.

EXAMINER: She has never seen a rheumatologist and has never sought help for her feet other than from you via her GP.

CANDIDATE: I would advise her that operations may be very helpful but that she should be formally assessed by a rheumatologist for diagnosis and disease control first. I would also advise review by the local podiatry and/or orthotics service as simple footwear modification may be all that is necessary to control her symptoms.

EXAMINER: I think that is appropriate advice at this stage. However, she returns to you a year later. Her synovitis is controlled by biologic agents but she has not found insoles and modified shoes helpful. How would you manage her at this point?

CANDIDATE: I would offer her a forefoot reconstruction consisting of excision of the lesser metatarsal heads, correction of lesser toe deformities and excision or fusion of the first metatarsophalangeal joint.

EXAMINER: Why?

CANDIDATE: This is a proven intervention with good results.

EXAMINER: How good?

CANDIDATE: More than 80% of patients report significant improvement.

EXAMINER: Would you fuse or excise the first metatarsophalangeal joint?

CANDIDATE: I would be guided by the age and functional demand of the patient in combination with the quality of the soft-tissue envelope. I would prefer to arthrodese the joint as I believe this aids maintenance of gait but, in a low-demand patient, excision is associated with reduced complications and more rapid rehabilitation.[1]

EXAMINER: If we say this lady is 45 years old, what would you do?

CANDIDATE: I would plan arthrodesis.

EXAMINER: How would you secure the arthrodesis?

CANDIDATE: I would use an oblique compression screw augmented by a dorsal locking plate, as biomechanical and clinical studies have shown this to be the most reliable method.

EXAMINER: Would you excise the lesser metatarsal heads in a patient of this age who now appears to have their disease under control?

CANDIDATE: If the joint surfaces were well preserved but with subluxation of the joints it might be appropriate to perform shortening metatarsal osteotomies such as Weil's osteotomies to preserve the metatarsal heads and allow reduction of the joints with soft tissue releases.

EXAMINER: Surely that just prolongs the procedure and increases the risk of complication?

CANDIDATE: Yes, but it is very difficult to salvage a rheumatoid foot without metatarsal heads if the disease progresses in subsequent years and this patient is young.

EXAMINER: Tell me about the principles of surgery in rheumatoid arthritis.

CANDIDATE: Surgery is indicated when symptoms or deformity are uncontrolled or unbraceable. The overall objective is to produce a stable, plantigrade foot. Aim for a single operation with a high rate of success. Arthrodesis is the predominant procedure. There is a high risk of complication due to osteopenia, dysvascularity, soft tissue fragility and immunosuppression.

EXAMINER: I agree. What steps can a surgeon take to minimize the risk of complication?

CANDIDATE: Biologic agents should be stopped in the run up to surgery and not resumed until there is good evidence of postoperative healing. It should go without saying that meticulous handling of soft tissues is necessary.

Incisions must be planned with care, both to maintain skin bridges and to ensure closure if significant deformities are being corrected.

EXAMINER: How long would you stop biological agents for?

CANDIDATE: Two weeks preoperatively and two weeks postoperatively.[2,3]

EXAMINER: What about other disease-modifying anti-rheumatic drugs? Which other ones would you stop?

CANDIDATE: Studies have shown that there is generally no need to stop other drugs such as methotrexate or leflunomide.

EXAMINER: I would like to backtrack a bit. Would you alter your management if she also had signs and symptoms of hindfoot arthritis?

CANDIDATE: Generally, I would plan to address the most symptomatic area first. However, a less symptomatic fixed hindfoot deformity should be corrected before proceeding to the forefoot. Flexible hindfoot deformity could be left until more symptomatic.

EXAMINER: Which hindfoot joints are most commonly affected in rheumatoid arthritis?

CANDIDATE: The talonavicular joint is most commonly affected, followed by the subtalar and calcaneocuboid joints.

EXAMINER: Can you outline the arguments for and against isolated talonavicular fusion in RA?

CANDIDATE: Isolated talonavicular fusion is a lesser procedure than triple fusion for both patient and surgeon and effectively eliminates hindfoot motion. Historically, a non-union rate of up to 37% has been reported although more recent studies suggest the non-union rate using contemporary fixation is much less. A triple arthrodesis is more reliable and allows greater deformity correction.

EXAMINER: Thank you.

1. Rosenbaum D, Timta B, Schmiegel A *et al.* First ray resection arthroplasty versus arthrodesis in the treatment of the rheumatoid foot. *Foot Ankle Int* 2011;**32**(6):589–594.
2. Lee MA, Mason LW, Dodds AL. The perioperative use of disease-modifying and biologic therapies in patients with rheumatoid arthritis undergoing elective orthopedic surgery. *Orthopedics* 2010;**33**(4):257–262.
3. Howe CR, Gardner GC, Kadel NJ. Perioperative medication management for the patient with rheumatoid arthritis. *J Am Acad Orthop Surg* 2006;**14**:544–551.

Structured oral examination question 4: Cavus foot

EXAMINER: These are pictures of the left foot of a 20-year-old man. Describe them. (Figure 4.4.)

CANDIDATE: These photographs show the anterior, medial and posterior views of a left foot with a cavus deformity. The hindfoot is in varus and there is a high arch. There doesn't appear to be any significant clawing or abnormality of the toes.

EXAMINER: What is the likely underlying cause?

CANDIDATE: A cavus foot develops a high arch as the result of imbalance in the musculature of the foot. It can be caused by a plantar flexion deformity of the forefoot or by a dorsiflexion deformity of the hindfoot known as calcaneocavus. The causes of a cavus foot may be broken down into congenital or acquired. The most common causes of congenital deformities are idiopathic, a sequela of clubfoot or due to arthrogryposis. The acquired deformities may be due to trauma or neuromuscular conditions. 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.

EXAMINER: HSMN?

CANDIDATE: Hereditary motor–sensory neuropathy. These are a group of inherited neurological conditions. Charcot–Marie–Tooth (CMT) is the most common group of these conditions.

EXAMINER: Can you go into more detail? How do these conditions lead to a cavus foot deformity?

CANDIDATE: The hereditary motor sensory neuropathies are a group of related conditions that may lead to cavus foot deformity due to muscle imbalance. The conditions are generally diagnosed by the pattern of deformity and a positive family history. The most commonly recognized is the Charcot–Marie–Tooth disease group which affects approximately 1 in 2500 people. These patients commonly have weakness of the intrinsic muscles, tibialis anterior and peroneus brevis. Type I will tend to present in the second decade, it is an autosomal dominant inheritance and patients have peroneal weakness, slow nerve conduction and absent reflexes. Type II presents later, in the third or

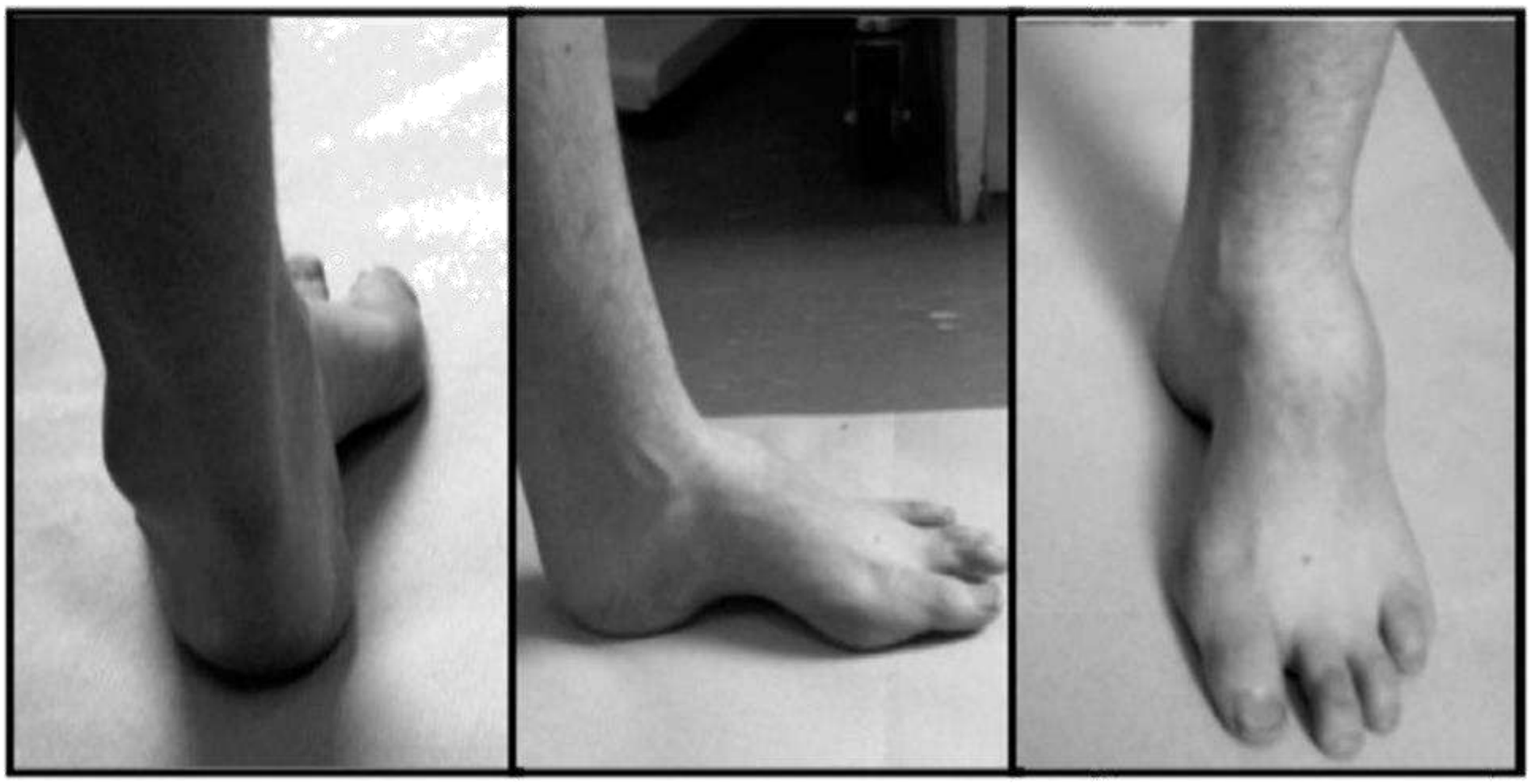


Figure 4.4 Cavus foot deformity.

fourth decade, and reflexes and nerve conduction are normal, however the foot signs may be more pronounced. Genetic analysis is able to diagnose and group these conditions more accurately and at least 17 types of CMT have been described.

EXAMINER: How would you assess this foot?

CANDIDATE: My assessment would have two components. I need to determine any underlying cause of the cavus and also evaluate the deformity itself. I would establish whether this is a unilateral or bilateral deformity and then I would begin by taking a thorough patient history. A cavus deformity is often secondary to a neurological cause so I would ask whether the foot had always been this shape and whether the deformity was progressive. I would ask what symptoms the foot causes and how it affects their function. I would also ask about any previous medical or surgical history, family history, and any previous surgical or non-surgical treatment the patient had received.

EXAMINER: What symptoms is this patient likely to complain about?

CANDIDATE: Common complaints in cavus feet are of pain, particularly forefoot pain, lateral foot pain under the metatarsal heads, or arch pain, instability of the ankle with a history of frequent ankle sprains. They may also have problems with fitting of footwear or alteration of gait.

EXAMINER: What are the main findings you would look for in the examination of a cavus foot?

CANDIDATE: On first, general inspection I would be looking to see if the deformity was bilateral and whether there were stigmata of a generalized condition such as wasting within the hands. With the patient standing I would look to see whether the heel was in varus, neutral or valgus alignment, assess the height of the longitudinal arch by inspection and also see whether I could pass more than two fingers underneath.
I would look to see the posture of the toes. This would be to assess the degree of deformity. While the patient was standing I would also look at the spine for any stigmata of an underlying abnormality such as a hairy patch or scoliosis.

With the patient sitting I would inspect the soles of the feet for callosities or areas of ulceration. I would look to see whether the cavus was due to plantarflexion of the first ray or the whole forefoot. I would assess sensation, deep tendon reflexes and power of the major muscle groups, particularly the tibialis anterior and posterior and the peroneal tendons. I would assess lateral ankle ligament competence with an anterior drawer and talar tilt test and look at the active and passive range of movement and see whether the deformities were flexible or fixed.

EXAMINER: What is shown in the following two diagrams?

CANDIDATE: These diagrams show the Coleman block test. (Figure 4.5.)

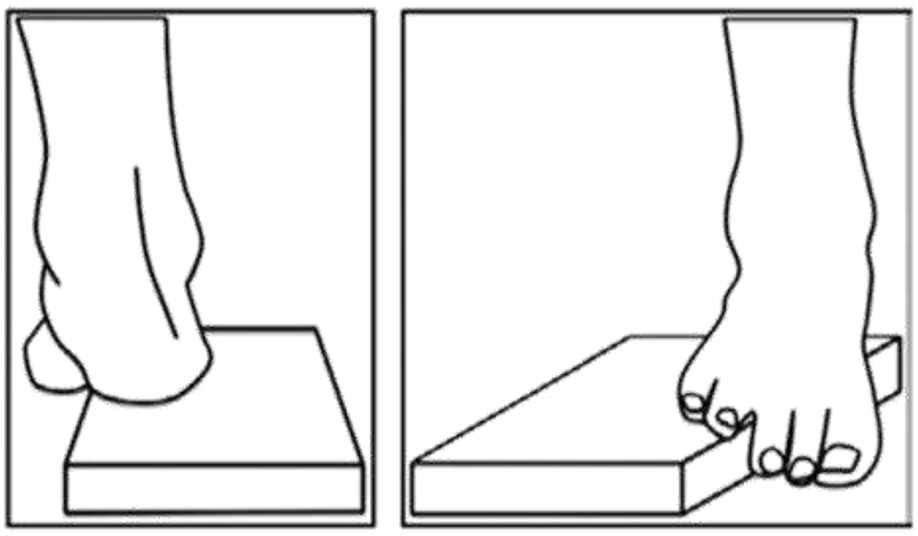


Figure 4.5 Coleman block test.

EXAMINER: And what is that?

CANDIDATE: The Coleman block test looks for flexibility of the hindfoot deformity by eliminating the deforming drive of the forefoot. In a cavus foot the first ray is plantarflexed so to place the foot stably on the ground the hindfoot has to move into varus. In the Coleman block test the foot is positioned so that the lateral border of the foot and the heel are placed on a block and the medial forefoot is allowed to hang off the edge of the block. If the heel then assumes a physiological alignment of neutral to 5° valgus when viewed from behind the hindfoot deformity is both flexible and driven by the forefoot.[1]

EXAMINER: What investigations would you use to evaluate this foot further?

CANDIDATE: In terms of evaluating the foot itself I would first obtain a series of weightbearing radiographs, a lateral of the foot and ankle, a hindfoot alignment view and an AP of the foot. If the patient had any signs or history suggesting an underlying spinal cause then radiographs or MRI scan of the spine should be considered.

EXAMINER: What information does the lateral X-ray tell you?

CANDIDATE: The magnitude of the cavus deformity can be quantified using Meary's angle, the angle between the long axis of the talus and the first metatarsal shaft. Normally this lies between +5° and −5°. Hibb's angle is the angle between the long axis of the first metatarsal shaft and the long axis of the calcaneum. This angle is normally 150° but decreases as the cavus worsens. The calcaneal pitch angle, the angle between the floor and the undersurface of the calcaneum, should be less than 30° but may be elevated in a cavus foot. The radiographs can also be used to look for signs of degenerative changes and the bones themselves may be abnormal in shape in a deformity that began early in childhood.

EXAMINER: What are the principles of managing this condition?

CANDIDATE: Firstly it is important to identify and if necessary address the underlying cause of the cavus. The patient should be examined for neuromuscular causes and investigated and referred for a neurological opinion if appropriate. The patient's current symptoms need to be understood as well as the likelihood of progression. Management can be non-operative with the use of orthotics to try and offload pressure areas and improve stability. Surgical treatment needs to be tailored to the individual patient's underlying pathology, risk of progression, level of deformity and muscular imbalance. Correction of deformity without addressing the muscular imbalance will not be successful.

EXAMINER: Thank you.

1. Coleman S, Chestnut W. A simple test for hindfoot flexibility in the cavovarus foot. *Clin Orthop Relat Res* 1977;**123**:60–62.

Structured oral examination question 5: Acquired adult flatfoot

EXAMINER: I would like you to look at this clinical photograph and tell me what you see. (Figure 4.6.)

CANDIDATE: This shows a posterior view of feet in a weightbearing stance. There is marked heel valgus and too many toes are visible. The medial longitudinal arch is not visible.

EXAMINER: How do you think the medial longitudinal arch may appear?

CANDIDATE: I would expect marked flattening of the arch.

EXAMINER: What term is used to describe this situation?

CANDIDATE: Pes planus or flatfoot.

EXAMINER: Yes. In adults, what are the causes of this condition?

CANDIDATE: Presentation in adults is usually acquired. The commonest cause is tibialis posterior dysfunction. Other causes include inflammatory arthritis, Charcot arthropathy, osteoarthritis and trauma.

EXAMINER: Good. How common is adult flatfoot?

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

EXAMINER: Okay. Let's stick with tibialis posterior dysfunction for just now. Describe a typical patient presentation.

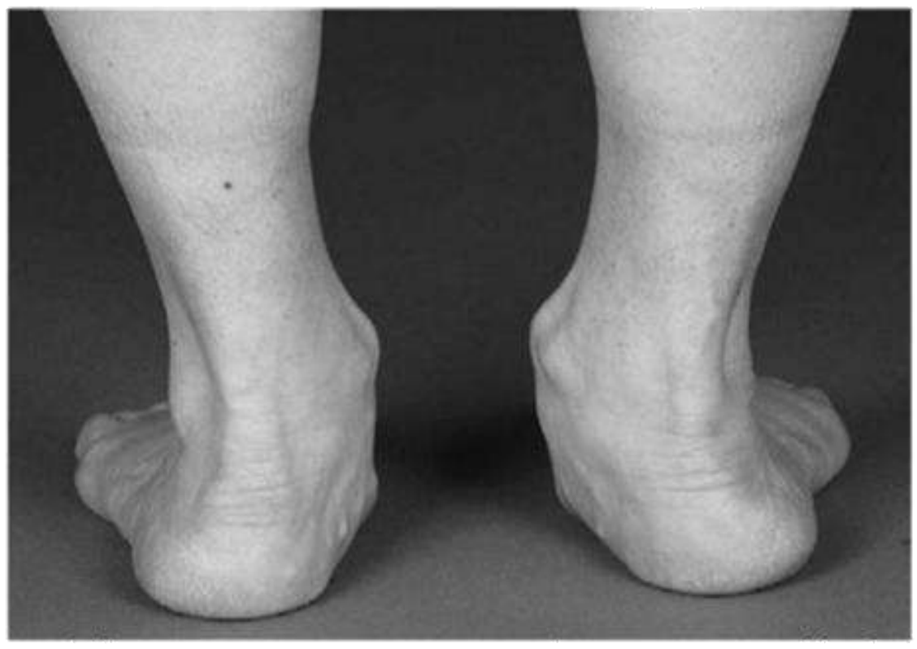


Figure 4.6 Acquired adult flatfoot.

CANDIDATE: The classic patient would be a female aged between 45 and 65 years of age. She would describe initial pain along the course of the tibialis posterior tendon. There is likely to be later development of increasing planovalgus deformity with medial deltoid ligament pain and sometimes lateral impingement pain.

EXAMINER: What are the key examination points you would look for?

CANDIDATE: I think the most useful test is the ability to perform a single heel raise. In conjunction with assessment of hindfoot flexibility, this would allow classification and guide treatment.

EXAMINER: As you have mentioned classification of tibialis posterior dysfunction, could you tell me any more about this?

CANDIDATE: Yes. Johnson and Strom proposed a three-stage classification in 1989. Myerson and Corrigan later added a fourth stage.[1] In stage I disease, there is no deformity but pain from the tendon. A single heel raise is usually possible but painful. In stage II disease, there is a flexible planovalgus deformity and weakness of single heel raise. In stage III disease, the deformity has become fixed and in stage IV, there is additional tilting of the talus in the ankle mortise. There are recommended procedures for each stage of the disease.

EXAMINER: Good. After your examination, how would you investigate this patient?

CANDIDATE: Weightbearing AP and lateral radiographs of both the foot and ankle would help to assess structural change and exclude other causes of flatfoot. They could also show associated degenerative change. The arch index could also be measured.

EXAMINER: Would the arch index influence your management?

CANDIDATE: No. I think it is mainly used as a research tool.

EXAMINER: Coming back to your classification, you suggested that there are recommended interventions for each stage of the disease. Please tell me about these.

CANDIDATE: For stage I, I would offer debridement of the tendon followed by 6–8 weeks of casting or splintage followed by provision of a definitive arch support orthosis.[2] For stage II disease I would offer either a lateral column lengthening or a medializing calcaneal osteotomy in conjunction with a FDL transfer to augment or replace the tibialis posterior.[3] In stage III disease, triple arthrodesis is recommended.[4] For stage IV disease, the management depends upon the flexibility of the ankle deformity. If it is flexible, then a triple arthrodesis combined with ankle bracing or deltoid ligament reconstruction may be adequate otherwise a triple arthrodesis combined or followed by ankle arthrodesis would be indicated.

EXAMINER: You seem very clear about surgical options. What about non-operative treatment?

CANDIDATE: I should have mentioned that. It is appropriate to offer analgesia and orthotic treatment to most patients initially. An orthotic providing medial arch support with a heel cup to control heel valgus can be helpful. There are two aims of orthotic treatment. First, this may offer adequate symptom relief. Second, it may control progressive heel valgus and flattening of the medial arch.

EXAMINER: You spoke about an FDL transfer. Tell me about this procedure.

CANDIDATE: After obtaining informed consent, anaesthesia, supine positioning, thigh tourniquet and skin prep and drape, I would make an incision over the line of the posterior tibial tendon, starting posterior to the medial malleolus. I would debride or resect the tendon according to the clinical appearances. The flexor digitorum longus sheath lies directly posterior to the tibialis posterior tendon and would be opened longitudinally as far distally as possible before the FDL tendon is divided. If there is a decent distal stump of tibialis posterior, then the FDL tendon could be sutured to this but it is probably better to pass it through a hole drilled in the navicular and suture it back to itself.

EXAMINER: In what direction would you pass FDL through the navicular?

CANDIDATE: From plantar to dorsal.

EXAMINER: What is the aim of a medializing calcaneal osteotomy?

CANDIDATE: The calcaneal osteotomy directly reduces the heel valgus and brings the weightbearing axis closer to the long axis of the leg. In addition it displaces the Achilles tendon insertion medially which stops it acting as an everter of the hindfoot.

EXAMINER: When obtaining consent, what would you advise about flexion of the toes after harvesting flexor digitorum longus?

CANDIDATE: I would expect flexion of the lesser toes to be maintained by flexor hallucis longus via the knot of Henry.

EXAMINER: Can you tell me a little more about the knot of Henry?

CANDIDATE: Flexor digitorum longus crosses flexor hallucis longus on the plantar aspect. There are a number of fibrous interconnections between the two tendons that afford a degree of cooperation in movement. This means that flexion of the digits can continue after harvest of FDL or FHL.

EXAMINER: One final question. What approach would you use for a triple arthrodesis to correct significant, fixed valgus heel deformity?

CANDIDATE: This is a potentially difficult situation. The joint preparation is most straightforward if a lateral utility approach or similar is combined with a dorsal incision over the talonavicular joint. If a significant deformity is being addressed there can be difficulty in closing the lateral incision once the deformity is corrected. There are advocates of triple arthrodesis via a single medial approach but this is difficult and not always possible.

EXAMINER: Thank you.

1. Myerson MS, Corrigan J. Treatment of posterior tibial tendon dysfunction with flexor digitorum longus tendon transfer and calcaneal osteotomy. *Orthopedics* 1996;**19**:383–388.
2. Teasdall RD, Johnson KA. Surgical treatment of stage I posterior tibial tendon dysfunction. *Foot Ankle Int* 1994;**15**(12):646–648.
3. Myerson MS, Badekas A, Schon LC. Treatment of stage II posterior tibial tendon deficiency with flexor digitorum longus tendon transfer and calcaneal osteotomy. *Foot Ankle Int* 2004;**25**(7):445–450.
4. Kelly IP, Easley ME. Treatment of stage 3 adult acquired flatfoot. *Foot Ankle Clin* 2001;**6**:153–166.

Structured oral examination question 6: Hallux valgus

EXAMINER: Please have a look at these clinical photographs and tell me what you see. (Figures 4.7 and 4.8.)

CANDIDATE: These show a frontal view of a pair of feet and an oblique view of the left foot. There is hallux valgus with the hallux over-riding the second toes. I can only count three lesser toes on the left foot and there is a scar in the webspace lateral to the hallux. The toenails appear friable and there is some excoriation around the lesser toes on the right foot. There is also a small area of scab on the dorsum of the right foot. I don't see any scars on the right foot but I think there is also a medial longitudinal scar over the left metatarsophalangeal joint.

EXAMINER: Absolutely. This 65-year-old lady had her left second toe removed some years ago for a presentation similar to that which she now has on the right. Her left-sided symptoms have also recurred. How would you assess her further?

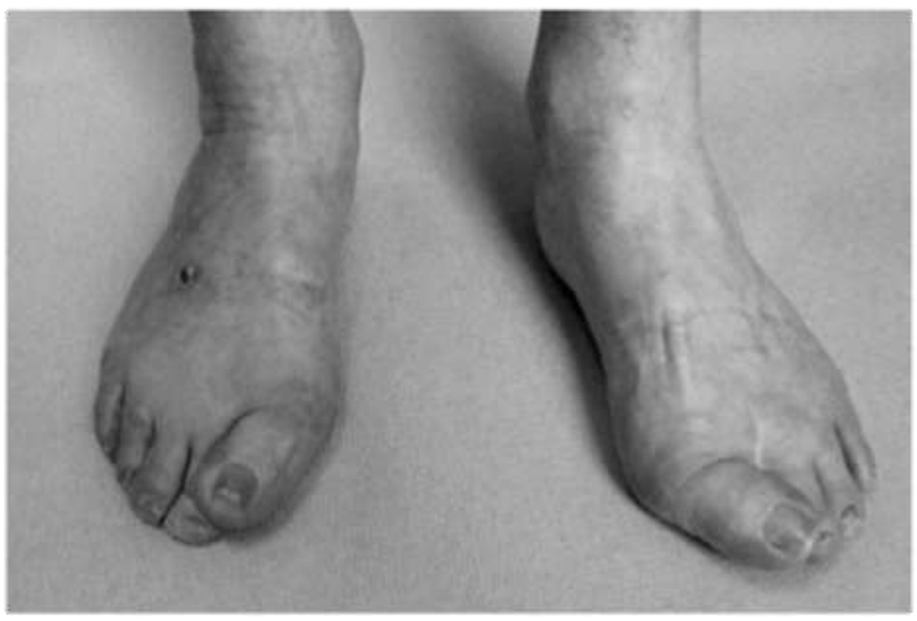


Figure 4.7 Anteroposterior (AP) view of hallux valgus.

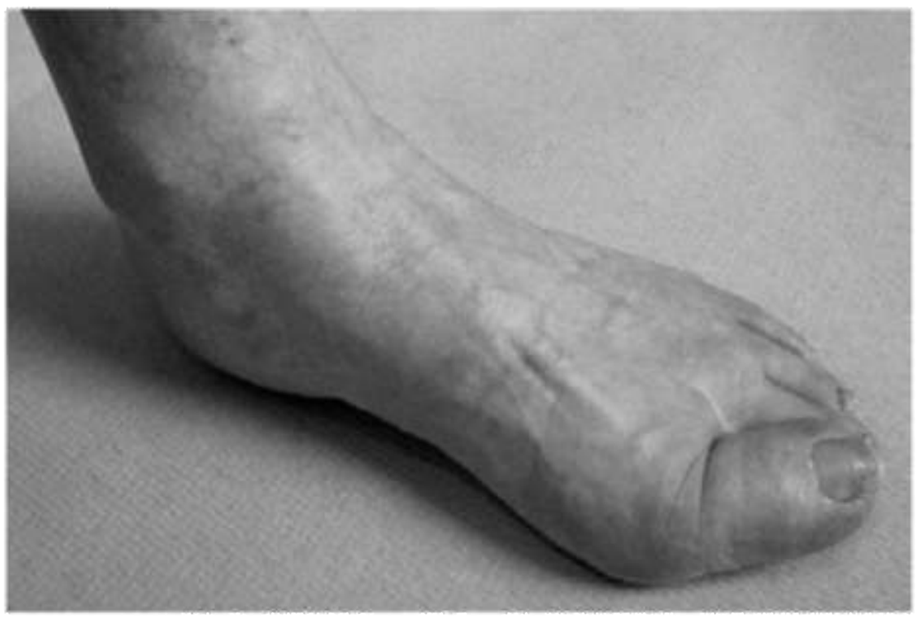


Figure 4.8 Oblique view of hallux valgus.

CANDIDATE: A detailed history should be obtained, looking to clarify the main source of her symptoms. Can I ask what symptoms she has?

EXAMINER: What do you think they are likely to be?

CANDIDATE: I would expect she has pain from her bunions and toes caused by rubbing on footwear and each other. I would be concerned to find out about symptoms suggestive of arthritic change at the MTP joint or metatarsalgia of the lesser rays.

EXAMINER: Let's say she has all these symptoms to varying degrees. Tell me about your further assessment.

CANDIDATE: I would complete the history, including questioning about relevant conditions such as diabetes, inflammatory arthritis, vascular disease and neuropathy, and proceed to examination. I would examine the gait and the posture of the weighted foot as hallux valgus is often associated with a planus foot. I would palpate for areas of tenderness, paying particular attention to the hallux MTP joint and lesser metatarsal heads. I would assess the degree of active and passive correction possible and the range of movement of the involved joints and look for gastrocnemius tightness. I would also perform a grind test to assess pain from loading the MTP joint. Neurovascular status must also be assessed.

EXAMINER: You spoke about assessing the range of movement of the involved joints. Can you be more specific?

CANDIDATE: I would want to assess the range of plantarflexion and dorsiflexion of the hallux MTP joint. It is also important to assess the movement at the first tarsometatarsal joint as excessive mobility here will influence surgical options.

EXAMINER: Okay, we might come back to that. Outline the value of plain radiographs in the management of hallux valgus.

CANDIDATE: I would routinely obtain weightbearing AP, oblique and lateral radiographs of the foot. This would allow me to objectively measure the angles, assess uncovering of the sesamoids and look for evidence of arthritic change.

EXAMINER: Keep going. What angles?

CANDIDATE: I would measure the intermetatarsal angle, hallux valgus angle and the distal metatarsal articular angulation.

EXAMINER: What is the normal range of these angles and how would these influence your management?

CANDIDATE: The intermetatarsal angle is normally less than 9°. The hallux valgus angle should be less than 15°. The distal metatarsal articular angle is normally a maximum of 15° from perpendicular to the axis of the first metatarsal. The degree of deformity largely determines the surgical management.

EXAMINER: If this lady had an intermetatarsal angle of 15° on the right with a hallux valgus angle of 35° and minimal passive correction of the hallux, what surgery would you plan?

CANDIDATE: If the first tarsometatarsal joint is normal, I would plan a scarf osteotomy combined with a lateral release and an Akin osteotomy of the proximal phalanx if necessary.

EXAMINER: Why would you choose a scarf osteotomy?

CANDIDATE: It is a very versatile procedure with stable fixation allowing postoperative mobilization without a cast. It maintains length of the metatarsal but allows translation, angulation and depression of the metatarsal head as necessary. It can also be used to shorten or even lengthen the metatarsal.[1]

EXAMINER: How would you secure the osteotomy?

CANDIDATE: With two headless compression screws.

EXAMINER: Why not use a simpler procedure such as a chevron or Mitchell osteotomy?

CANDIDATE: For the degree of deformity described, combined with the lack of passive correction of the hallux, I believe the correction that could be achieved with a distal osteotomy would be inadequate. A further disadvantage of a Mitchell osteotomy is that it produces shortening of the first metatarsal, which could lead to transfer metatarsalgia.

EXAMINER: For your proposed management, what complications would you discuss when seeking consent?

CANDIDATE: Firstly, I would advise that whilst early weightbearing is possible with a scarf osteotomy it takes up to a year for the foot to fully settle after such surgery but that typically 85% of patients are pleased with the outcome. I would advise a 1% risk of deep infection and a slightly higher risk of superficial infection. Recurrence is possible with time although the risk of this is greatest in adolescent cases. A minority of patients will have significant stiffness of the MTP joint afterwards and there can be sensory loss if the dorsomedial sensory nerve is injured. I would mention the possibility of hallux varus as a complication as this is difficult to treat. I would also mention the possibility of intraoperative and postoperative metatarsal fracture.

EXAMINER: How would you treat hallux varus?

CANDIDATE: A subtle varus may improve as the patient returns to normal foot wear. Whilst soft tissue procedures such as abductor hallucis and medial capsular release or transfer of a

slip of EHL are described for flexible deformity, arthrodesis of the first MTP joint is a reliable option in the presence of significant stiffness or arthrosis.

EXAMINER: So you have successfully treated this lady's right foot and she is pleased with the result. Would you go ahead and do the same on the left?

CANDIDATE: No. The absence of the second toe predisposes to recurrence and I would propose arthrodesis of the hallux MTP joint.

EXAMINER: Thank you.

1. Barouk LS, Toullec ET. Use of scarf osteotomy of the first metatarsal to correct hallux valgus deformity. *Techniques Foot Ankle Surg* 2003;**2**(1):27–34.

Structured oral examination question 7: Hallux rigidus

EXAMINER: This 45-year-old male patient has presented with pain and stiffness of his right big toe. Describe the X-ray findings. (Figure 4.9.)

CANDIDATE: This is a radiograph of a right foot showing osteoarthritis of the first metatarsophalangeal joint (MTPJ) with loss of joint space, osteophytes and sclerosis. There is also a mild hallux valgus deformity. There is no other obvious deformity.

EXAMINER: So what is this commonly called in orthopaedics?

CANDIDATE: Hallux rigidus.

EXAMINER: Tell me the range of movement of a healthy first MTPJ.

CANDIDATE: The joint should be able to dorsiflex between 70° and 90° and plantarflex between 24° and 40°.

EXAMINER: How would you go about managing this patient?

CANDIDATE: First of all I would need to perform a full history and clinical examination on the patient. I would also obtain a weightbearing lateral and an oblique X-ray of the foot in addition to the AP view we have here.

EXAMINER: Very good. If we concentrate on the clinical examination what specific findings are you looking for to help with your management decision?

CANDIDATE: I would need to assess the integrity of the skin and the neurovascular status of the foot. I would palpate for large osteophytes and assess the range of movement of the first MTPJ and look to see whether the patient had pain limited to the extremes of movement or throughout the arc of motion. A grind test of the joint would be informative. I also need to evaluate the motion and look for any sign of degenerative change at the interphalangeal joint (IPJ).

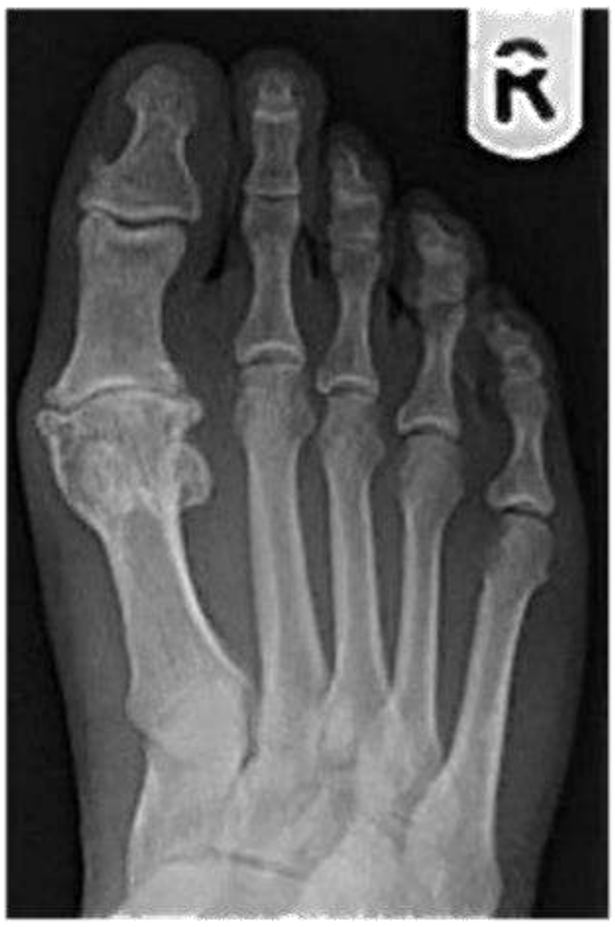


Figure 4.9 X-ray showing hallux rigidus.

EXAMINER: What is the importance of the IPJ?

CANDIDATE: A fusion of the first MTPJ may accelerate degeneration in the surrounding joints so if the IPJ is already symptomatic a motion-preserving procedure at the MTPJ may be more appropriate.

EXAMINER: Right so talk me through the management options for a patient with hallux rigidus.

CANDIDATE: In the first instance, unless there is a pressing indication for surgery such as impending skin compromise, I would maximize non-operative treatment. Options here include activity modification, footwear with a stiff sole and a rocker sole to reduce MTPJ motion. NSAIDs may be useful and in some cases an intra-articular injection may provide relief.

EXAMINER: And the operative options?

CANDIDATE: That choice would depend on the grade of the disease.

EXAMINER: Can you expand on that? Are you aware of any grading systems for this condition?

CANDIDATE: The most widely used is a radiographic grading by Hattrup and Johnson in which Grade 1 has a well-preserved

joint space with mild to moderate osteophytes, Grade 2 has a reduced joint space with moderate osteophytes and Grade 3 has a complete loss of joint space, marked osteophytes and there may be subchondral cysts within the metatarsal head.[1]

EXAMINER: So then, back to the operative options for treatment.

CANDIDATE: In Grade 1 or 2 disease a cheilectomy, in which the osteophytes and the dorsal 25–30% of the articular surface are resected, is widely used and gives good relief of symptoms. If there is good plantarflexion, restriction of dorsiflexion and no mid-range pain a Moberg dorsal closing wedge osteotomy of the proximal phalanx can be used to shift the arc of movement further into the dorsiflexion range to reduce symptoms.[2] For patients with severe disease and no ligamentous instability total joint replacements do exist but early loosening has been a common problem. Good results have been reported with hemiarthroplasty of either the metatarsal head or the base of proximal phalanx but few large series exist and neither is commonly used in the UK.[3,4] Arthrodesis of the first MTPJ is still the mainstay of treatment for severe disease and joint preparation with dome-shaped reamers and a lag screw and dorsal plate construct is the most biomechanically sound fixation.[5] Keller's arthroplasty is a possibility in elderly, low-demand patients, however cock-up deformities and transfer metarsarsalgia may develop.

EXAMINER: So, back to arthrodesis. What is the optimal position for fusion?

CANDIDATE: Dorsiflexion of 25° across the MTPJ, valgus of 10–15° and neutral rotation to ensure an effective plane of motion of the IPJ.

EXAMINER: How will you consent a patient for arthrodesis of the first MTPJ?

CANDIDATE: I will explain that the intentions of the surgery are to relieve pain and optimize mobility. The risks and complications include wound-healing problems, infection, damage to the medial cutaneous nerve, non-union, malunion, delayed union, metalwork irritation and accelerated degeneration in surrounding joints.

EXAMINER: If we return to the patient we started discussing. He is a 45-year-old male who is a keen walker. He has significant stiffness and pain on mobilization and dorsiflexion but a grind test is negative. He has exhausted non-operative measures. What treatment will you offer him?

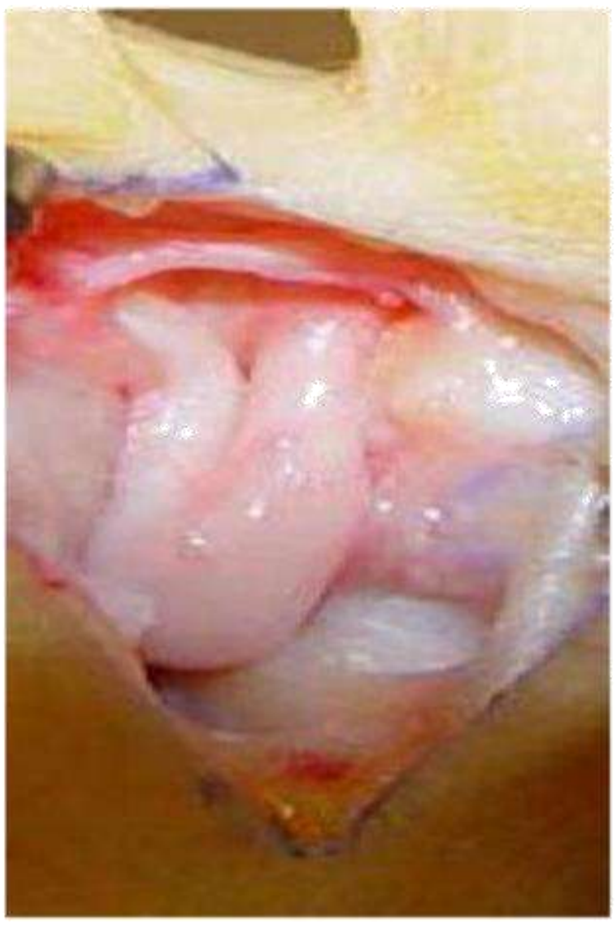
CANDIDATE: His X-ray shows joint space narrowing and peripheral osteophytes, but as he is an active individual and his grind test is negative I would offer him a cheilectomy. I would also discuss fusion with him and explain to him that this may become necessary if a thorough cheilectomy failed to provide sufficient relief or he had later progression of disease.

1. Hattrup SJ, Johnson KA. Subjective results of hallux rigidus following treatment with cheilectomy. *Clin Orthop Relat Res* 1988;**226**:182–191.
2. Moberg E. A simple operation for hallux rigidus. *Clin Orthop Relat Res* 1979;**142**:55–56.
3. Taranow WS, Moutsatson MJ, Cooper JM. Contemporary approaches to Stage II and Stage III hallux rigidus: the role of metallic hemiarthroplasty of the proximal phalanx. *Foot Ankle Clin N Am* 2005;**10**:713–728.
4. Carpenter B, Smith J, Motley T *et al*. Surgical treatment of hallux rigidus using a metatarsal head resurfacing implant: mid-term follow-up. *J Foot Ankle Surg* 2010;**49**:321–325.
5. Politi J, Hayes J, Njus G *et al*. First metatarsal-phalangeal joint arthrodesis: a biomechanical assessment of stability. *Foot Ankle Int* 2003;**24**(4):332–337.

Recommended reading

1. Maffulli N, Ferran NA. Management of acute and chronic ankle instability. *J Am Acad Orthop Surg* 2008;**16**:608–615.
2. Easley ME, Adams SB Jr, Hembree WC *et al*. Current concepts review: results of total ankle arthroplasty. *J Bone Joint Surg Am* 2011;**93**:1455–1468.
3. Courville XF, Hecht PJ, Tosteson ANA. Is total ankle arthroplasty a cost-effective alternative to ankle fusion? *Clin Orthop Relat Res* 2011;**469**:1721–1727.
4. Gougoulias N, Khanna A, Maffulli N. How successful are current ankle replacements? A systematic review of the literature. *Clin Orthop Relat Res* 2010;**468**:199–208.
5. Saltzman CL, Mann RA, Ahrens JE *et al*. Prospective controlled trial of STAR total ankle replacement versus ankle fusion: initial results. *Foot Ankle Int* 2009;**30**(7): 579–596.
6. Chou LB, Cougllin MT, Hansen S Jr *et al*. Osteoarthritis of the ankle: the role of arthroplasty. *J Am Acad Orthop Surg* 2008;**16**(5):249–259.
7. Jeng J, Campbell J. Current concepts review: the rheumatoid forefoot. *Foot Ankle Int* 2008;**29**: 959–968.
8. Trieb K. Management of the foot in rheumatoid arthritis. *J Bone Joint Surg Br* 2005;**87-B**:1171–1177.
9. Younger ASE, Hansen ST Jr. Adult cavus foot. *J Am Acad Orthop Surg* 2005;**13**:302–315.

3. Mortons neuroma:shown clinical pictureDemonstrate signs, treatment options? Chances of success with surgery?



Causes:
-Traumatic
-Tethering of digital nerve at 3rd web space
-Associated with hallux valgus

Symptoms
-pain
-swelling
-burning sensation
-reduced sensation
-difficulty in shoe wear

Signs
-Swelling
-Tenderness
- Mulders click

Investigation:
-Radiographs –Osteoarthritis of metatarsophalangeal joint/bony erosion
-Ultrasound scan
-MRI

Management:

Non-operative:
-Shoe wear
-Orthotics

-Injection

Operative:
Dorsal approach-release intermetatarsal ligament- do neurectomy 3 cm proximal to ligament

Evidence:
Gannis; F&A INT. 2004 Feb; 79-84
78% excellent to good
19% fair
3%poor

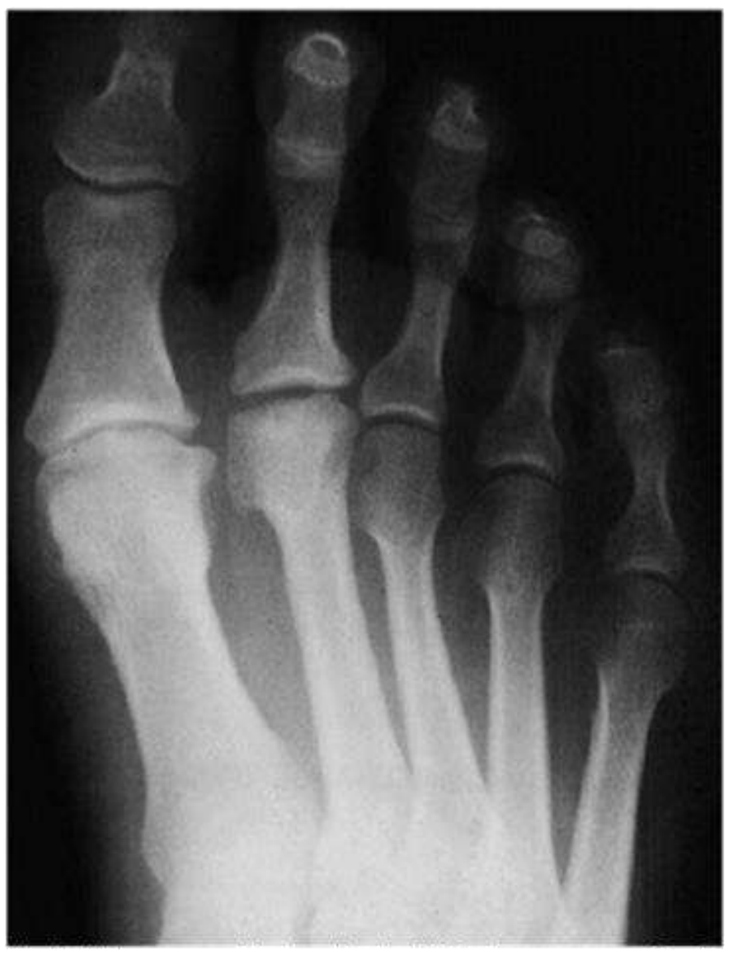
Chances of success

Reccurence after primary surgery-15-20%
JohnsonJE;JBJS;Am. June;70(5);651-7

Reasons for recurrence

-Inadequate excision
-Preserving intermetatarsal ligament
-Too distal

4. Radiograph of Freiberg disease. Discuss radiograph and management



Features
- occurs during the puberty
- most common in female
- Leads to avascular necrosis of the metatarsal head
- May be from repetitive stress with microfractures at the junction of the metaphysis and the growth plate

Associated factors:
- disease is more common in patients whose first metatarsal is shorter than second metatarsal, which increases weight imposed on the head of the second metatarsal
-Juvenile Arthritis

- Clinical Manifestations:
- pain localized to head of the second metatarsal
- wearing of high heeled shoes makes condition worse
- localized swelling at second metatarsophalangeal joint
-reduce range of movements of second metatarsophalangeal joint

Investigation
-Radiographs
-MRI

- Radiologic Findings:
-initially the epiphysis becomes sclerotic, joint space is widened. Later, the epiphysis narrows with irregular bony surfaces, sclerosis, and bone spurs at margins with the appearance of osteoarthritis
- epiphysis becomes fragmented followed by osteolysis and reconstitution of bony architecture
- fragmentation and osteolytic phases
- metatarsal head becomes irregular, widened, and flattened at articular surface;

Differential diagnosis:

Differential diagnosis:
- Ewing's Sarcoma
- Stress Fracture
- Osteosarcoma

- Treatment:
Conservative:
- Orthosis/ foot wear with metatarsal bar or pad placed beneath the involved bone
- limit activity for four to six weeks
- if patient have severe symptoms consider immobilizing foot in short leg walking cast for four weeks

Surgical indications:
-Very rare
- failure of conservative treatment
-partial or complete removal of metatarsal heads

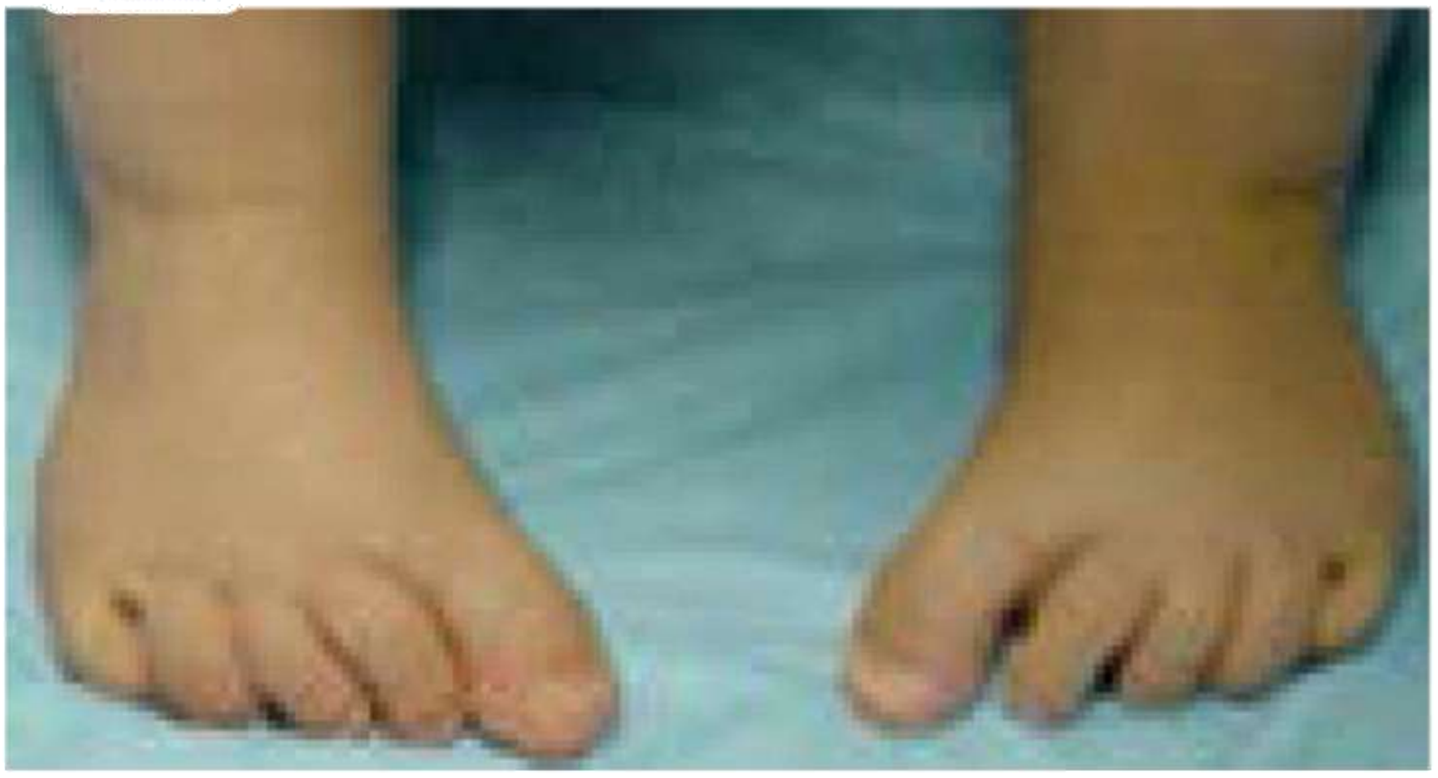
Another procedure is dorsally based closing wedge osteotomy.

5. Photograph of metatarsus adductus. What is this? What is it associated with? How is it

5. Photograph of metatarsus adductus. What is this? What is it associated with? How is it

5. Photograph of metatarsus adductus. What is this? What is it associated with? How is it staged? What treatments would you recommend

5 of 11



Associations

-Developmental dysplasia of hip
-Torticollis

Stages

Bleck- based on heel bisector line
Normally line passes between 2nd and 3rd toes

Mild—line goes through 3rd toe
Moderate – between 3rd and 4th toes
Severe –between 4th and 5th toes

Berg types

1. Simple metatarsus adductus
2. Complex metatarsus adductus- with lateral shift of midfoot
3. Skew foot- Metatarsus adductus with hindfoot valgus
4. Complex skew- Above with lateral shift midfoot

Management

Non operative

-Stretching
-Manipulation and casting

Operative (refractory cases)

Soft tissue/bony procedures

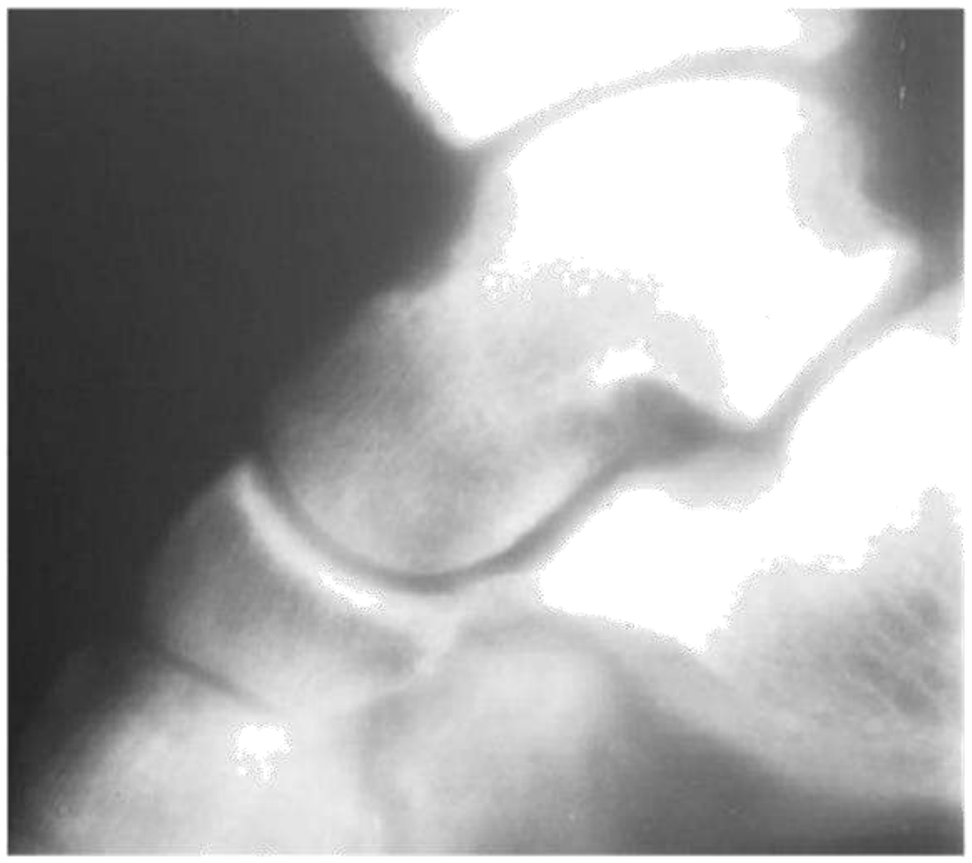
Soft tissue/bony procedures

Options

1. Abductor hallucis longus recession
2. Medial capsule release+ lateral column shortening osteotomy (calcaneum)

3. Medial opening osteotomy- cuneiform
Lateral closing osteotomy- cuboid+_ metatarsal osteotomy

6. Tarsal coalition. Shown radiograph. Discuss



Describe radiograph- oblique xray, beaking talus, ant eaters foot

Other radiograph views- Harris axial view
-CT is the investigation of choice

Features

1. Calcaneo navicular is commonest (8-12 years)
2. Talo navicular (12-16 years)
3. 50 percentage bilateral
4. Etiological theories- family history/Autosomal dominance?
5. Can be fibrous, cartilaginous, bony

Clinical features

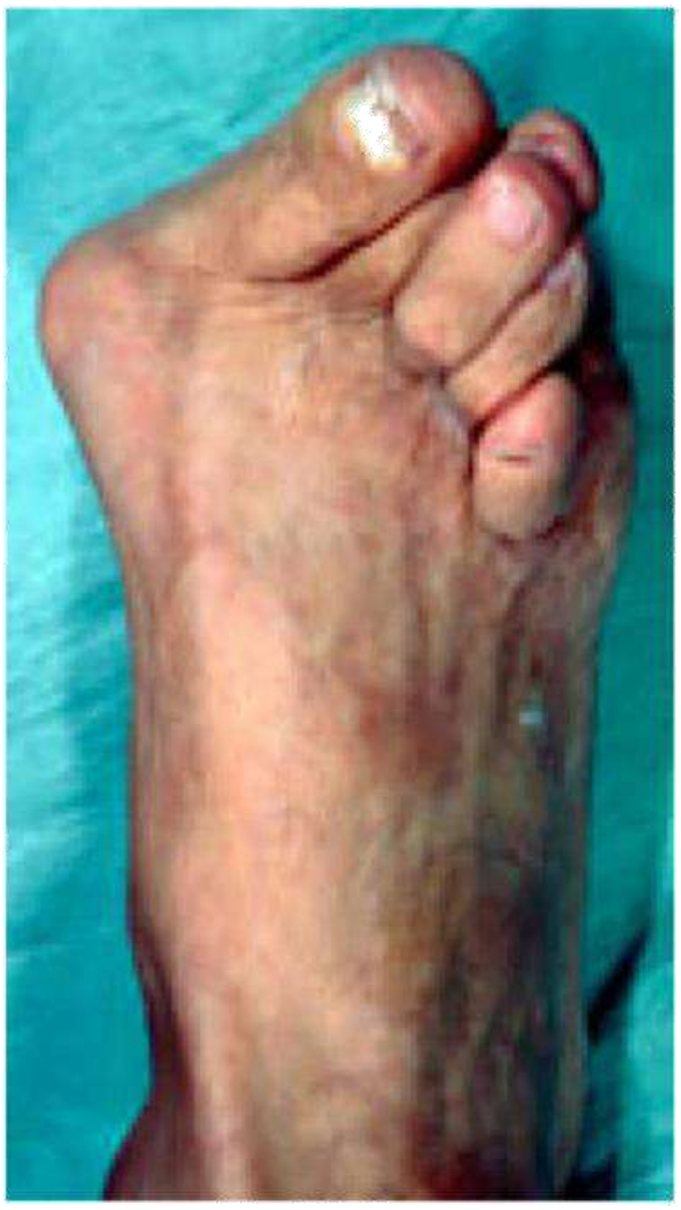
Symptoms

1. Diffuse pain and stiffness
2. Difficulty walking on uneven surfaces
3. Present as sprained ankle

Signs

1. Pes planus- rigid
2. Hind foot valgus
3. Peroneal spasticity

8. Clinical picture of rheumatoid foot. Discuss management



Deformities in rheumatoid foot
Fore foot
1. Hallux valgus
2. Claw toes-loss of muscle imbalance
3. Forefoot pronation?
4. prominent metatarsal heads- dislocate volar and plantar
5. Varus little toe

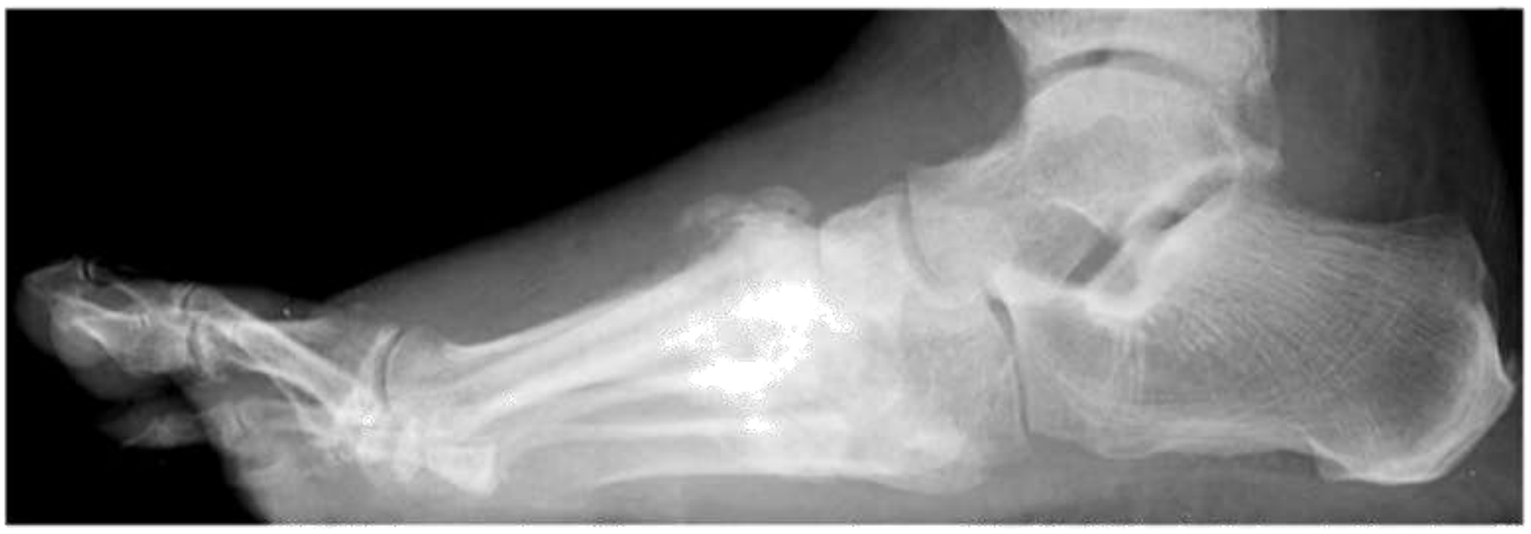
Midfoot-Pes planus
Hindfoot- valgus

Principles of rheumatoid surgery
1. Avoid bilateral surgery/hand +foot- for walking
2. If forefoot +hindfoot involved- operate on hindfoot first
3. Avoid simultaneous hindfoot and forefoot surgery- massive swelling
4. Most common surgery- forefoot
5. Cervical spine xray before surgery- Atlantoaxial instability
6. Continue Disease modifying anti rheumatic drugs

Management of forefoot problems

Conservative
- Foot orthosis/custom shoes

9. Radiograph of Charcoat foot. Causes. Assessment and management



Describe x ray
-Destruction of bone with rocker bottom feet/pes planus

Causes

1. Diabetes mellitus
2. Syringomyelia
3. Alcoholism
4. Syphilis
5. Leprosy
6. Spinal cord injury
7. renal dialysis

Assessment
1. History
2. Examination
3. Nerve studies –Semmes-Weistein testing, nerve conduction studies
4. Vascular- Doppler/angiogram
5. Infection – bloods tests (white cell count/erythrocyte sedimentation rate/blood culture)

Investigations:
Radiographs
Bone scan (indium)
MRI

Pathophysiology/Stages
Stage1: Developing stage-hot swollen erythrema and painless foot.
Stage2: Coalesent stage- collapsed foot, flat arch, rocker bottom appearance
Stage3:Reconstruction stage-osteopenic and disorganized joints and bone healing.

Management
Principle of management:
1. Prevention
2. Diabetic control
3. Eliminate infection

4. Improve blood supply

4. Improve blood supply
5. Ulcer management
6. Total contact casting for neuropathic ulcers
7. Resect bone prominences to reduce the risk of ulcer

Treatment
Conservative
Immobilization and off loading:
- In early and mid stages, the patient needs to stay off the affected foot
- Custom foot wear or patellar tendon bearing / custom orthosis
- Total contact casting helps distribute weight bearing forces more evenly than regular casts
-With either midfoot or ankle neuropathic changes, prolong immobilization is usually necessary (often 1-2 years)

References
J Bone Joint Surg Am. 2008 Apr;90(4):754-9. doi: 10. 2106/JBJS. F. 01523. **Charcot arthropathy and immobilization in a weight-bearing total contact cast.**
de Souza LJ

Surgical:
-If skin ulcers over a midfoot exostosis (cannot be managed with shoe/orthotics modifications) then surgical excision of the exostosis is indicated (do not turn a stable midfoot into an unstable midfoot)
-In general, surgical fixation should be avoided during the early fragmentation stage of the process: wait for the consolidation phase
- If surgery is necessary, keep full thickness incisions and do not attempt to avoid cutting cutaneous nerves since the foot is often already insensate
- Often surgical fixation includes joint arthrodesis

Tendon transfers:
- Chronic diabetic ulcers under the first metatarsal head treated by staged tendon balancing.

Arthrodesis and Reconstruction:

-Hindfoot valgus collapse: tibio talar calcaneal arthrodesis

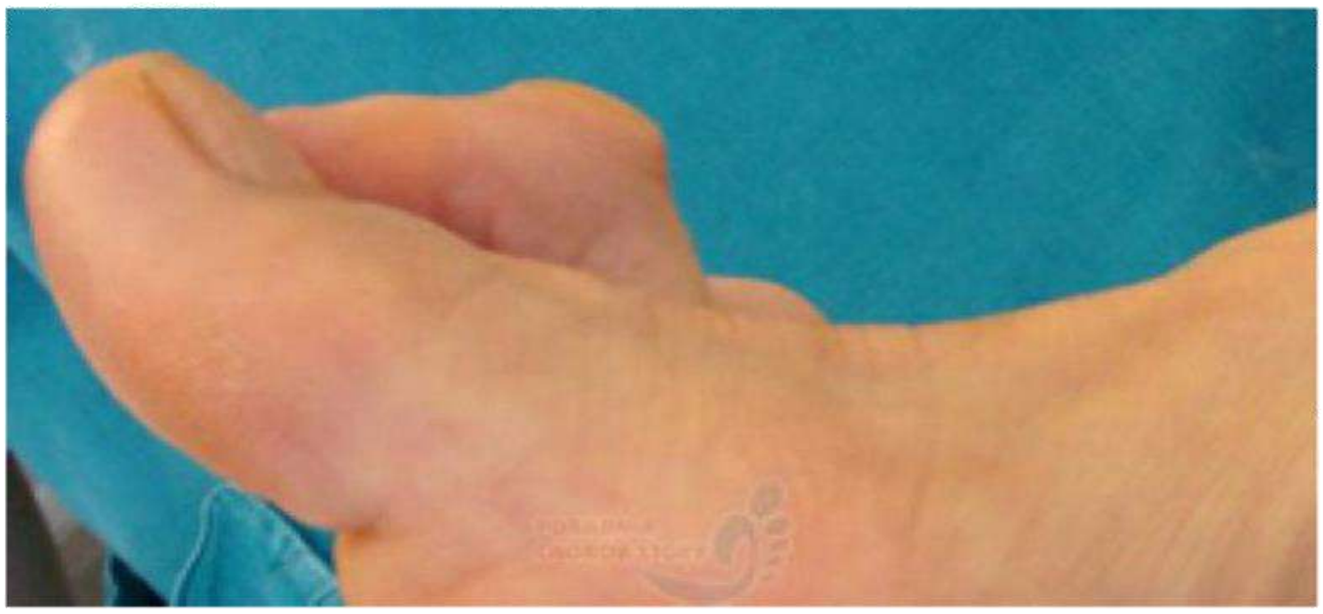
- Midfoot Charcot:
- high infection rate (25%) when arthrodesis is performed with an open plantar ulcer
- with midfoot collapse, reduction is often carried out by bone resection in addition to soft tissue release;

Complication
Wound breakdown
Metal work
Ulceration/gangrene
Amputation

10. Clinical picture of claw toes. What is the difference between claw and hammer? How will

10. Clinical picture of claw toes. What is the difference between claw and hammer? How will you manage?

11 of 11



Claw toe- Hyperextension of Metatarsophalangeal joint (MTPJ) and flexion proximal and distal interphalangeal joint (PIP+DIPJ)
Hammer toe- Extension Metatarsophalangeal joint (MTPJ) and flexion Proximal interphalangeal joint (PIPJ) only
Mallet toe- Flexion of distal interphalangeal joint (DIPJ) only
Curly toe- Flexion of all the distal three joints(MTPJ+PIPJ+DIPJ)

Pathology of claw

-repetitive trauma-stretching of plantar plate-extension of proximal phalanx

Management

Non operative- orthotics/wide shoes/metatarsal bar/padding

Operative
Flexible claw

1. Release dorsal structures- capsule/lengthen extensor tendon/release collateral ligaments

2. Flexor to extensor transfer (Flexor digitorum longus)

Rigid claw – arthrodesis

Trauma

9.Hand :

A. Oral :

Space of Poirier

central weak area of the wrist in the floor of the carpal tunnel at the level of the proximal capitate between the volar radioscaphocapitate ligament and volar long radiolunate ligament (radiolunotriquetral ligament)

Sagittal Band Rupture

Sagittal band rupture leads to leads to dislocation of the extensor tendon caused by **trauma** or by a chronic inflammatory process such as **rheumatoid arthritis**.

Quadriga Effect

The quadriga effect is characterized by an active flexion lag in fingers adjacent to a digit with a previously injuredor repaired flexor digitorum profundus tendon

Lumbrical Plus Finger

paradoxical extension of the IP joints while attempting to flex the fingers

Dupuytren's Disease

A benign proliferative disorder characterized by fascial nodules and contractures of the hand

Ulnocarpal Abutment Syndrome

Syndrome cause by excessive impact stress between ulna and carpal bones (primarily lunate) / positive ulnar variance

Ulnar Styloid Impaction Syndrome

impaction between ulnar styloid tip and triquetrum that is seen in patients with excessively long ulnar styloidsor ulna positive wrists

Kienbock's Disease

Avascular necrosis of the lunate leading to abnormal carpal motion ulnar negative variance (leads to increased radial-lunate contact stress)

Preiser's Disease (Scaphoid AVN)

A condition caused by AVN of scaphoid

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A condition caused by AVN of scaphoid

SLAC (Scaphoid Lunate Advanced Collapse)

A condition of progressive instability causing advanced arthritis of radiocarpal and midcarpal joints due to chronic SL ligament injury.(which is a consequence to a DISI deformity)

initially affects the radioscaphoid joint and progresses to capitolunate joint

CIND (carpal instability nondissociative)

Defined as instability between rows (either radiocarpal or midcarpal)
radiocarpal instability (between radius and proximal row)
midcarpal instability (between proximal and distal row)

Radial Clubhand (radial deficiency)

A longitudinal deficiency of the radius likely related to sonic hedgehog gene in which thumb usually deficient

Ulnar Club Hand

A congenital upper extremity deformity characterized by deficiency of the ulna and/or the ulnar sided carpal structures (absent ulnar sided digits)

Congenital Radial Head Dislocation

can bedifferentiated from a traumatic dislocation by:
bilateral involvement
hypoplastic capitellum
convex radial head
other congenital anomalies
lack of history of trauma
difficult to reduce
almost always posterior dislocation of radial head
often combined with bowing and shortening of radius

Madelung's Deformity

congenital dyschondrosis of the distal radial physis (ulnar volar physis of the distal radius) that leads to
partial deficiency of growth of distal radial physis
excessive radial inclination and volar tilt
ulnar carpal impaction (+UV)

Congenital Radial Ulnar Synostosis

bony bridge between the proximal radius and ulna results due to failure of differentiation in proximal aspect of the forearm

- Cleft Hand

absence of 1 or more central digits of the hand or foot (also known as lobster-claw deformity)

Symphalangism

Congenital digital stiffness that comes in two forms hereditary & nonherediatry

Camptodactyly :

Congenital digital flexion deformity that usually occurs in the PIP joint of the small finger

Clinodactyly

Congenital curvature of digit in radioulnar plane found in 25% of children with Down's syndrome , most commonly affected middle phalanx of small finger

Poland Syndrome

A congenital disorder characterized by
unilateral chest wall hypoplasia
hypoplasia of the hand and forearm
symbrachydactyly and shortening of middle fingers
result of absence or shortening of the middle phalanx
simple complete syndactyly of the short digits

Constrictive Ring Syndrome (Streeter's Dysplasia)

Amniotic band syndrome occurs when loose fibrous bands of ruptured amnion adhere to and entangle the normal developing structures of the fetus.

Giant Cell Tumor of Tendon Sheath

A benign nodular tumor that is found on the tendon sheath of the hands and feet

- PL
- FDS.

In the forearm (anterior interosseous branch[2])

- FPL
- Radial half of FDP
- PQ.

In the hand

Motor – the 'LOAF' muscles of the thenar eminence

- Lateral two lumbricals
- Opponens pollicis
- Abductor pollicis
- Flexor pollicis brevis.

Sensory

Flexor surfaces and nails of the radial 3½ digits:

- Skin thenar eminence supplied by the palmar cutaneous branch, which is given off 5 cm above the wrist
- Abnormal connections
- Martin–Gruber (17%) – median to ulnar nerve in forearm
- Riche–Cannieu (77%) – deep branch ulnar to median in hand
- Clinically may present as ulnar nerve lesion but no intrinsic deformity, or as severe carpal tunnel syndrome but with no muscle weakness.

Carpal tunnel anatomy

Fibro-osseous tunnel formed by the concavity of the anterior surface of the carpus and roofed over by the flexor retinaculum. Knowledge of the anatomy of the carpal tunnel is essential to undertake carpal tunnel decompression.

Boundaries of the carpal tunnel

- Radial wall: tubercle scaphoid, ridge of trapezium
- Ulnar wall: pisiform, hook of hamate
- Floor: carpus, proximal metacarpals
- Roof: flexor retinaculum.

Contents (ten)

Nine flexor tendons and the median nerve:

- FPL – the most radial structure
- Median nerve (just deep to the flexor retinaculum and lateral to FDS)
- FDS – lies on the profundus tendons arranged 2-by-2 (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 FCR tendon lies in a separate fibro-osseous tunnel deep to the flexor retinaculum.

Flexor retinaculum

Attachments

The flexor retinaculum is attached to the pisiform and hamate on the ulnar side and to the ridge of the trapezium and the tuberosity of the scaphoid on the radial side. There is also a deep slip, which is attached to the medial lip of a groove on the trapezium.

Functions

- Prevents bowstringing of the long flexor tendons
- It gives partial insertion to some muscles (PL, FCU)
- It gives partial origin to some muscles (thenar and hypothenar muscles).

Variations of the motor (recurrent) branch of the median nerve

A key surgical landmark and major surgical danger in carpal tunnel release. Surface landmark is the intersection of the flexed middle finger tip with Kaplan's line.

There are three main variations to the motor branch in the palm and several other much less common variations:

1. **Extraligamentous branch** (50%) arises distal to the transverse carpal ligament and recurrent to the thenar muscles. The nerve hooks radially and upwards to enter the thenar muscle mass between the FPB and APB muscles.
2. **Subligamentous branch** (30%) arises within the carpal tunnel, emerging distal to the flexor retinaculum and recurrent to the thenar muscles.
3. **Transligamentous branch** (20%) arises from the nerve within the carpal tunnel and pierces the flexor retinaculum.

Patients with rare variations usually have a large palmaris brevis muscle.

Variants of the palmar cutaneous branch of the median nerve

The course of the palmar cutaneous branch of the median nerve may vary in four important ways:

1. Normally the nerve is given off 5 cm proximal to the wrist and runs along the ulnar side of FCR before crossing the flexor retinaculum. The nerve divides into two major branches, medial and lateral, whilst crossing the flexor retinaculum.
2. There are two distinct branches, which travel separately across the wrist.
3. Within the carpal tunnel and penetrates flexor retinaculum.
4. Nerve may be replaced by a branch from the radial or ulnar nerve.

Kaplan's cardinal line

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 is estimated by the point where the middle finger tip flexes onto Kaplan's line. The deep palmar arch lies deep to Kaplan's cardinal line. The superficial palmar arch lies 2 cm distally, deep to the distal transverse palmar crease.

Examination corner

Hand oral 1

Clinical photograph of a recent surgical scar over the thenar crease suggestive of carpal tunnel decompression. The scar, however, was placed far too radial, over the thenar muscle mass, and extended straight across the wrist, cutting perpendicular to the flexor crease.

Candidate 1

EXAMINER: This woman has had recent surgery to her hand. What do you think of the scar and its position?

CANDIDATE: There is a recent longitudinal mid-palmar scar over the thenar crease, which extends proximally across the distal wrist crease into the distal forearm. [Long pause. The scar looked like one used for a carpal tunnel release but slightly atypical and the candidate was not entirely sure if he/she should mention carpal tunnel release.]

EXAMINER: [Who was expecting the candidate to continue talking and therefore ended up prompting the candidate.] What do you think of the scar itself?

CANDIDATE: The scar extends straight across the wrist.

EXAMINER: What surgery do you think she has had?

CANDIDATE: Carpal tunnel decompression.

EXAMINER: Do you normally extend the incision for decompression proximally above the wrist joint?[3]

CANDIDATE: [Who was aware of the controversy but was unsure what to say, hesitant, and trying to second-guess the examiner.] No, I generally do not go above the wrist with my initial skin incision for a straightforward carpal tunnel.

EXAMINER: I can't think of any particular reason why you should extend the skin incision above the distal wrist crease. What do you think of the scar?[4]

CANDIDATE: The scar looks as though it is still maturing, suggesting that surgery was only recently performed.

EXAMINER: Don't you think the scar is a bit too medial and also crossing the flexion crease at 90°? With this incision, important structures may be damaged and, in addition, a contracture may develop over the wrist joint.

CANDIDATE: Yes.

EXAMINER: Show me on my hand the incision you would use when performing a carpal tunnel release. [Hands candidate a pencil. The candidate draws the incision on the examiner's palm.]

EXAMINER: What structures pass through the carpal tunnel?

CANDIDATE: The carpal tunnel contains the flexor digitorum superficialis and profundus tendons to all fingers, flexor pollicis longus and the median nerve.

EXAMINER: [Not particularly happy with the candidate.[5]] What structures can be damaged during a carpal tunnel release?

CANDIDATE: The palmar cutaneous branch of the median nerve and the recurrent motor branch to the thenar muscles.

EXAMINER: Where are these nerves given off?

CANDIDATE: The palmar cutaneous branch of the median nerve is given off 5 cm above the wrist joint and the recurrent motor branch to the thenar muscles just distal to the flexor retinaculum.

EXAMINER: Show me where exactly these nerves travel – trace them out on my hand. [Offers outstretched hand to candidate.

On reflection the candidate thought that they should have picked up straight away that the scar was way too far radially into the thenar muscle bulk and then gone on to mention the structures placed at risk with this incision; this, despite numerous promptings by the examiner as to what they wanted the candidate to say.

The vital components to this oral scenario were immediate recognition of the misplaced surgical incision and the surface anatomy of various anatomical structures at risk from carpal tunnel decompression.]

[*Candidate*: I never recovered from not immediately recognizing the fairly obvious misplaced surgical scar. I also thought there was some catch in the questions being asked. Normally when performing carpal tunnel decompression I extend the skin incision slightly above the distal wrist crease. I was unaware that this was slightly controversial. Instead of defending my practice to the examiner I second-guessed what they wanted me to say but I didn't sound convincing in my reply. I was only vaguely familiar with Kaplan's cardinal lines, which I should have mentioned when discussing the surface anatomy of the various nerves at risk.]

[Fail]

Candidate 2

The candidate immediately picked up that the position of the scar was suboptimal and explained the structures at risk.

CANDIDATE: I am concerned that the scar has been placed too radially for a carpal tunnel decompression.

[The case was fairly straightforward; the crucial element was that you had to spot immediately that the incision was far too radial.]

[Pass]

Carpal tunnel syndrome

Incidence

- One per cent in the general population (14% in diabetics).

Causes

The acronym ICRAMPS helps you to remember the causes of carpal tunnel syndrome:

- I – Idiopathic
- C – Colles', Cushing's
- R – Rheumatoid

- – Acromegaly, amyloid
- – Myxoedema, mass, (diabetes) mellitus
- P – Pregnancy
- S – Sarcoidosis, systemic lupus erythematosus.

Symptoms

- Paraesthesia in radial 3½ digits
- Worse at night
- Weakness in the hand, dropping things
- Pain
- 40% bilateral involvement
- M:F 1:6
- Not always classical.

Signs

- Examine neck movements
- Swelling over volar aspect of the wrist
- Wasting of thenar muscles
- Sensation (light touch, two-point discrimination)
- Power (APB)
- Provocative tests:

  Median nerve compression test (Durkin's) – apply direct pressure with your thumb over the nerve, with the patient's elbow extended and wrist flexed 60° (86% sensitivity, 83% specificity)

  Tinel's sign (74% sensitivity, 90% specificity)

  Phalen's test (positive if signs <60 s) (61% sensitivity, 83% specificity)
- **Sensitivity**: the proportion of patients with carpal tunnel syndrome testing positive
- **Specificity**: the proportion of patients without carpal tunnel syndrome testing negative (this assesses how effective the test is at excluding those patients without carpal tunnel syndrome).

Nerve conduction studies (NCS)

Abnormal if:

- Sensory conduction is prolonged >3.5 ms (demyelination)
- Increased distal motor latency >4.0 ms
- Decreased amplitude (axonal loss)
- Accurate 85–90%
- False-negative rate 10–15%.

On which patients do you perform NCS? – Safe answer is to say all patients in whom carpal tunnel syndrome is suspected but who have negative carpal tunnel provocation tests.

Differential diagnosis

- Cervical disc disease
- Peripheral neuropathy – alcohol, diabetes
- Pronator syndrome
- Spinal cord lesions – tumour, syrinx, MS
- Thoracic outlet syndrome

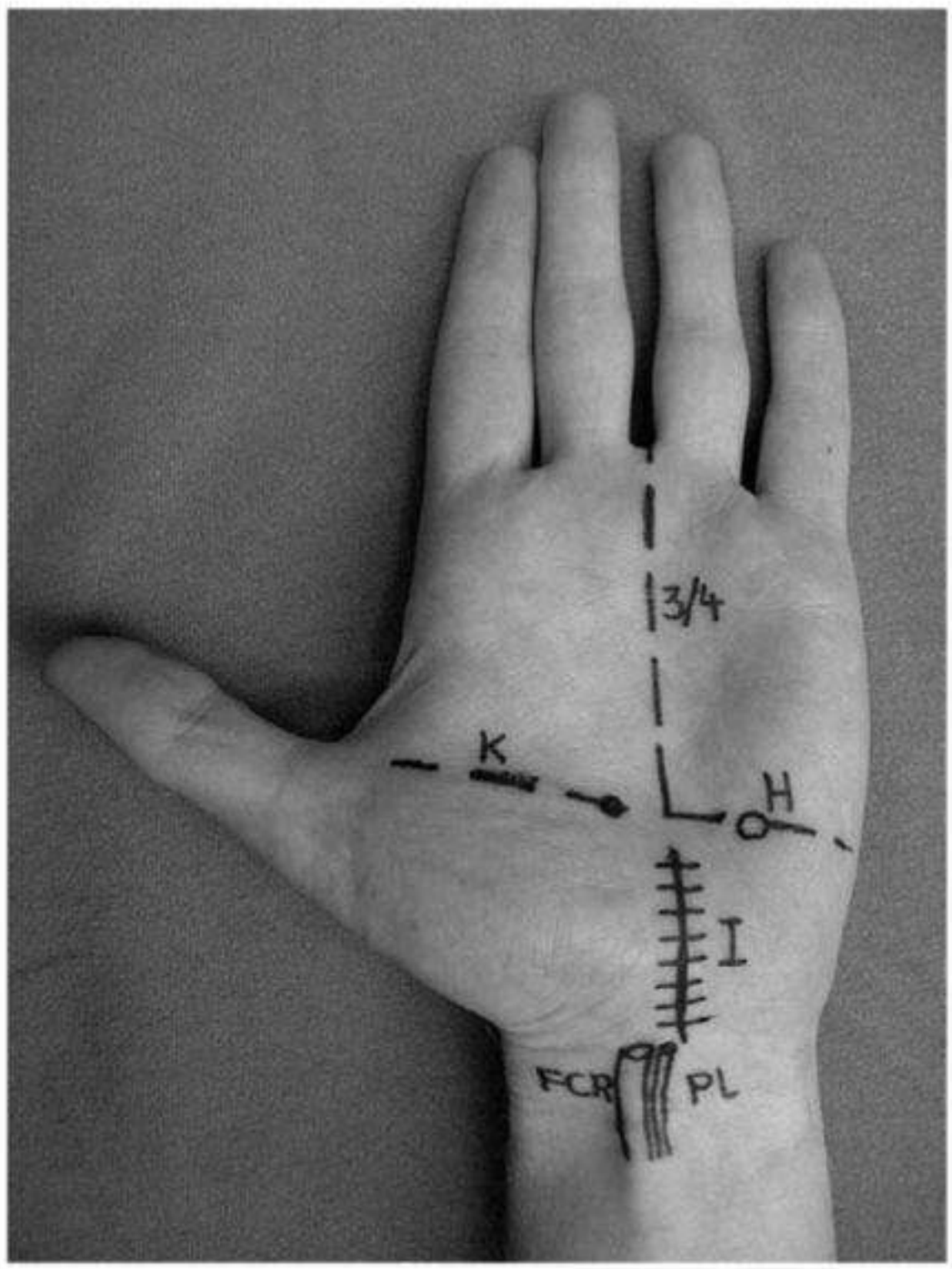

Figure 19.1 Landmarks for open carpal tunnel decompression. I, Incision; H, hook of hamate; K, Kaplan's line; black spot, motor branch; PI, palmaris longus; FCR, flexor carpi radialis.

- Collagen vascular disorders
- Raynaud's disease
- CRPS.

Management

Non-operative

Indicated in those with mild intermittent symptoms without neurological impairment, who have had symptoms <1 year, and who have no muscle wasting. The classic indication is a temporary, reversible carpal tunnel syndrome (pregnancy). Splintage in neutral (extension increases tunnel pressure), NSAIDs, short-term diuretics, steroid injections (80% transient relief, 20% symptom-free at 12 months).

Surgical

Indicated in those with progressive persistent symptoms with neurological defects.

1. **Open carpal tunnel decompression**

   95% good or excellent results. Allows good visualization of the median nerve and the contents of the tunnel. The physiological state of the nerve can be assessed.

   Incision – a safe incision is offered (Figure 19.1): longitudinal from the distal wrist crease and just ulnar to

palmaris longus,[6] in line with the radial border of the ring finger, and distally to Kaplan's line. It is not routinely recommended to cross proximal to the wrist crease.
Adjunctive surgical procedures – no demonstrable benefit of additional synovectomy or internal neurolysis following carpal tunnel release and may lead to adhesions.

2. **Endoscopic carpal tunnel release**
Introduced to reduce the incidence of pillar pain but this has not been demonstrated. Use either the Agee (one-incision) or Chow (two-incision) technique. Steep learning curve with increased early complication rate, including actual injury to the median nerve. Pain less at 3 months compared to open release, but no difference at 1 year.[7]

Complications of carpal tunnel release

- **Infection**: <1%
- **Tender scar**: owing to division of very fine terminal branches of the palmar cutaneous nerve
- **Haematoma**: major bleeding is rare if safe anatomical landmarks are observed
- **Dehiscence**: sutures removed too early
- **Damage to nerves**: recurrent motor branch of the median nerve, palmar cutaneous branch median nerve, ulnar nerve
- **Pillar pain**: pain felt when pushing down on the base of the hand following carpal tunnel release. Aetiology uncertain, possible because of gradual stretching of intercarpal ligaments, which are no longer de-tensioned by the flexor retinaculum. Others suggest that division of the flexor retinaculum disturbs the alignment of the pisotriquetral joint, which is the source of pillar pain
- **Complex regional pain syndrome**: rare but always mention in consent
- **Weakness of grip**: returns to preoperative levels in 3 months
- **Bowstringing of flexor tendons**: more a theoretical complication than a practical one.

Failed carpal tunnel release

Recurrent or persistent symptoms in up to 20%. Most problems are caused by inadequate release. Other differential diagnoses include cervical radiculopathy at C5/6, compression of the upper trunk brachial plexus and proximal median nerve compression. Double crush phenomenon relates to more than one site of compression that, if in isolation, would not cause symptoms, but together cause symptoms. Perform NCS after at least 3 months.

- **Symptoms unchanged**: wrong diagnosis, inadequate decompression, postoperative fibrosis, double crush phenomenon
- **New symptoms**: normal structures damaged at surgery, new diagnosis.

Re-exploration is indicated if:

- Marked symptoms
- Other causes of symptoms ruled out
- Positive Phalen's test
- Positive NCS after 3–6 months
- ?Relief of symptoms after steroid injection.

Examination corner

Hand oral 1: *Carpal tunnel syndrome*

- Symptoms
- Signs
- Tests
- NCS (principles, findings)
- Open versus endoscopic.

Hand oral 2: *Carpal tunnel syndrome*

EXAMINER: Who would you perform nerve conduction studies on?

EXAMINER: Show me in my hand how you would do a carpal tunnel decompression. [Examiner wearing a glove.]

EXAMINER: What nerves are in danger?

EXAMINER: If you extend your incision distally, what structure is in danger [deep palmar arch]?

EXAMINER: What are the surface markings of the deep palmar arch?

EXAMINER: What would you do if you found a space-occupying lesion of the nerve at an open carpal tunnel release?

CANDIDATE: I would close the wound as the decompression has been performed, and refer the patient to a surgeon with microscopic skills.

EXAMINER: How would you manage a patient with thenar wasting and weakness? [Explain to the patient that muscle will not return, and consideration for tendon transfers – see later.]

Pronator syndrome

Background

Essentially a high median nerve entrapment. The median nerve is vulnerable to compression at a variety sites around the elbow.[8] This is a rare clinical condition.

Sites of entrapment

- Ligament of Struthers – 1% population (supracondylar process)
- Bicipital aponeurosis (lacertus fibrosus)
- Origin of the pronator teres (abnormal anatomy, tight fibrous bands)
- Proximal arch of FDS.

Causes of Carpal Tunnel Syndrome

Mnemonic : DONALD TRUMP

D - Diabetes

O - Obesity, overuse injury

N - Neoplasm

A - Acromegaly

L - Low Thyroid (Hypothyroid)

D - Dialysis-related Amyloidosis

T - Trauma (e.g. Colles' fx)

R - Rheumatoid Arthritis

U - Use of Aromatase inhibitor

M - Menopause, Myxoedema

P - Pregnancy

Double crush syndrome is a distinct compression at two or more locations along the course of a peripheral nerve that can coexist and synergistically increase symptom intensity. In addition, dissatisfaction after treatment at one site may be the result of persistent pathology at another site along a peripheral nerve.

Physiology

- The normal resting pressure at the carpal tunnel in neutral position

  WITHOUT CTS is

  2.5 mmgh

Physiology

- The normal resting pressure at the carpal tunnel in max flexion or extension position

  WITHOUT CTS is

  30 mmgh

Physiology

- The normal resting pressure at the carpal tunnel in neutral position

  WITH CTS is

  30 mmgh

Physiology

- The normal resting pressure at the carpal tunnel in max flexion or extension position

  WITH CTS is

  90 - 110 mmgh

Physiology

- Elevation of carpal tunnel pressures of more than 20 to 30 mm Hg impedes epineurial blood flow, and nerve function is impaired .

Causes

- Idiopathic “most common”.
- Anatomical
- Physiological
- External

CAUSES

ANATOMICAL

PHYSIOLOGICAL

EXTERNAL FORCES

Decrease in Size of CT

Increase in Contents of

Neuropathic Conditions

Inflammatory Conditions

Alterations of Fluid Balance

Anatomy

Decrease in Size of Carpal Tunnel

- Bony abnormalities of the carpal bones
- Acromegaly
- Flexion or extension of wrist

Increase in Contents of Canal

- Forearm and wrist fractures (Colles fracture, scaphoid fracture)
- Dislocations and subluxations (scaphoid rotary subluxation, lunate volar dislocation)
- Posttraumatic arthritis (osteophytes)
- Musculotendinous variants
- Aberrant muscles (lumbrical, palmaris longus, palmaris profundus)
- Local tumors (neuroma, lipoma, multiple myeloma, ganglion cysts)
- Persistent medial artery (thrombosed or patent)
- Hypertrophic synovium
- Hematoma (hemophilia, anticoagulation therapy, trauma)

Anatomy

Decrease in Size of Carpal Tunnel

- Bony abnormalities of the carpal bones
- Acromegaly
- Flexion or extension of wrist

Increase in Contents of Canal

- Forearm and wrist fractures (Colles fracture, scaphoid fracture)
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- Persistent medial artery (thrombosed or patent)
- Hypertrophic synovium
- Hematoma (hemophilia, anticoagulation therapy, trauma)

External Forces

- Vibration
- Direct pressure

STEROID injection

- Temporarily relief
- Improvement is good prognostic factor
- Blind or U/S guided
- Under aseptic technique

- Site : 1 cm proximal and 1 cm ulnar to intersection of distal palmer crease with PL.
- Direction : 45◦ to skin, towards the base of thumb.
- 25 gauge needle 1 ml steroid with 1 ml 1% lidocaine.
- Slowly
- Stop if patient feels parasthesia, numbness or sever pain.

SURGERY

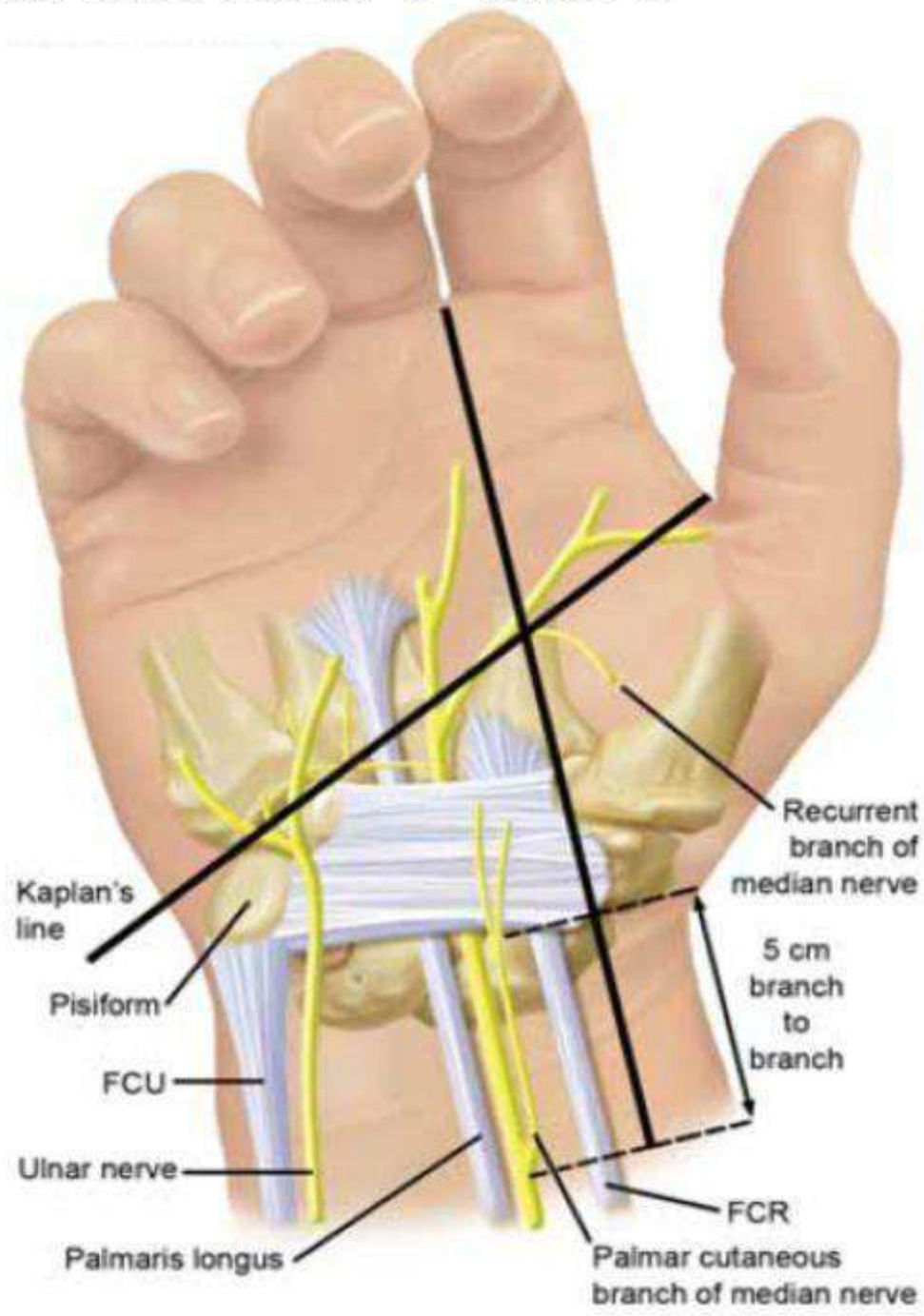
- Day case surgery
- Arm outstretched on a hand table.
- Pneumatic tourniquet.
- Loupe magnification is recommended.
- Anesthesia can be by general, regional or local.

Surgical Technique

- Scrubbing and toweling .
- Local anesthesia injected at site of incision deep and superficial ,proximal and distal
- Tourniquet inflated 100 mmgh above SBP

Kaplan Cardinal Line

- A line drawn from apex of the interdigital fold between thumb and index finger, toward ulnar side of hand, parallel with proximal palmar crease, & passing 4-5 mm distal to pisiform bone



Land marks

- Intersection of the Kaplan cardinal line and a line drawn along the radial border of the fourth ray, and ending at the wrist flexion crease

CTS

palmar hand layers :

1.Skin

2. Subcutaneous tissue

3. Fat

4. Volar Carpal Ligament

(obliquely oriented)

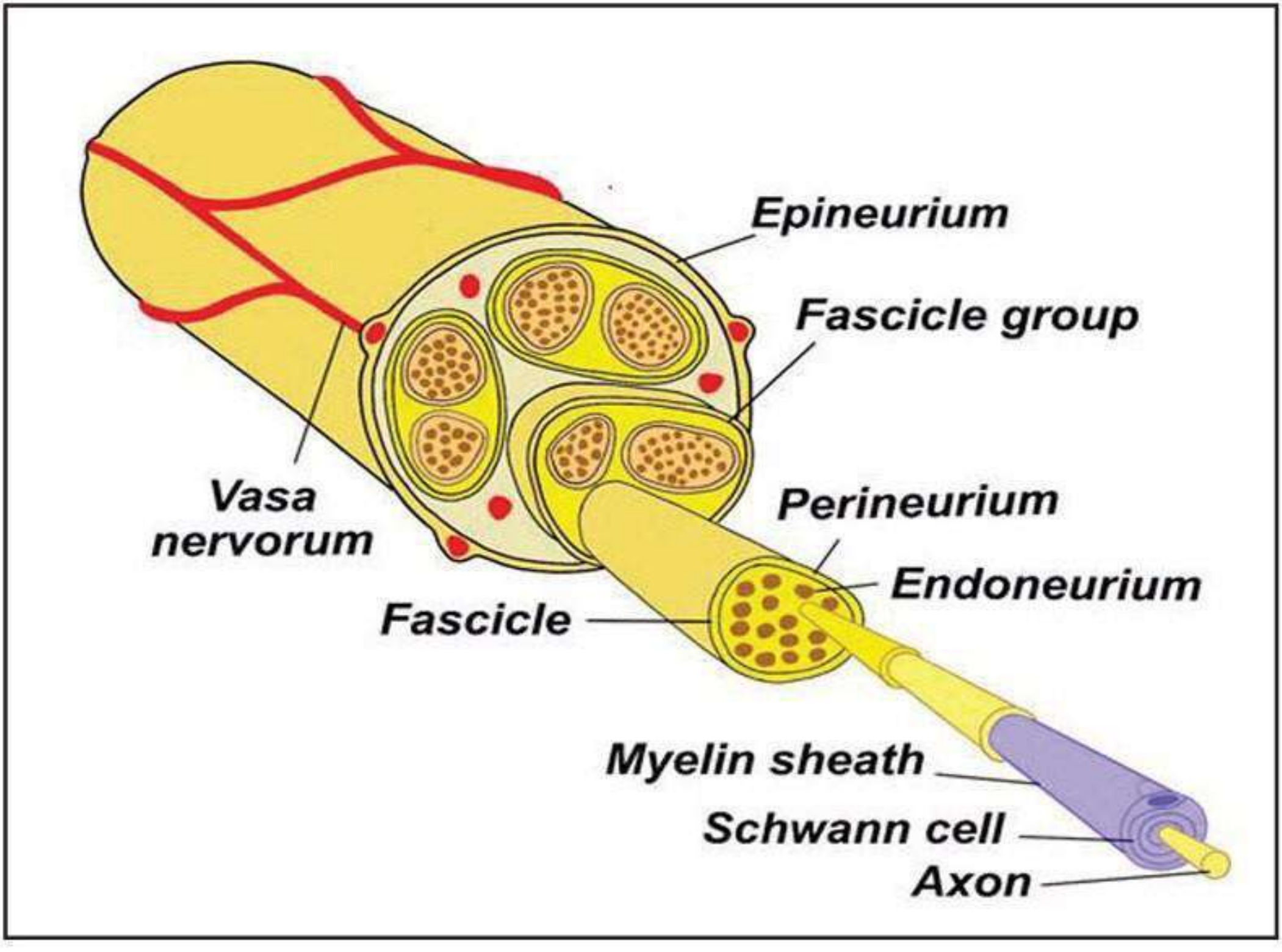
5. Palmaris brevis (innervated by ulnar nerve) could be found just above TCL

6. Tranverse carpal ligamnet

(longitudinal oriented)

- Musculocutaneous nerve
  - origin
    - lateral cord of the brachial plexus
  - anatomy at elbow
    - it exits laterally, distal to the biceps tendon
    - it will terminate as the LABC (forearm), which is found deep to the cephalic vein
  - innervation at elbow
    - it supplies the biceps and brachialis
      - the nerve runs between these muscles
- Radial nerve
  - origin
    - posterior cord of the brachial plexus
  - anatomy at elbow
    - it leaves the triangular interval (teres major, long head of triceps and humeral shaft)
    - found in spiral groove 13 cm above the trochlea
    - pierces lateral intermuscular septum 7.5 cm above the trochlea
      - this is usually at the junction of the middle and distal third of the humerus
      - lies between the brachialis and the brachioradialis
    - distally it is located superficial to the joint capsule, at the level of the radiocapitellar joint
- Median nerve
  - origin
    - medial/lateral cords of the brachial plexus
  - anatomy at elbow
    - it courses with brachial artery, running from lateral to medial
    - lies superficial to brachialis muscle at level of elbow joint
  - innervation at elbow
    - it gives branches to elbow joint
    - it has no branches in upper arm
- Ulnar nerve
  - origin
    - medial cord of brachial plexus
  - anatomy at elbow
    - runs medial to brachial artery, pierces medial intermuscular septum (at the level of the arcade of Struthers) and enters posterior compartment
    - it traverses posterior to the medial epicondyle through the cubital tunnel
  - innervation at elbow
    - it gives branches to elbow joint
    - it has no branches in upper arm
    - first motor branch to FCU is found distal to the elbow joint

b form fascicle



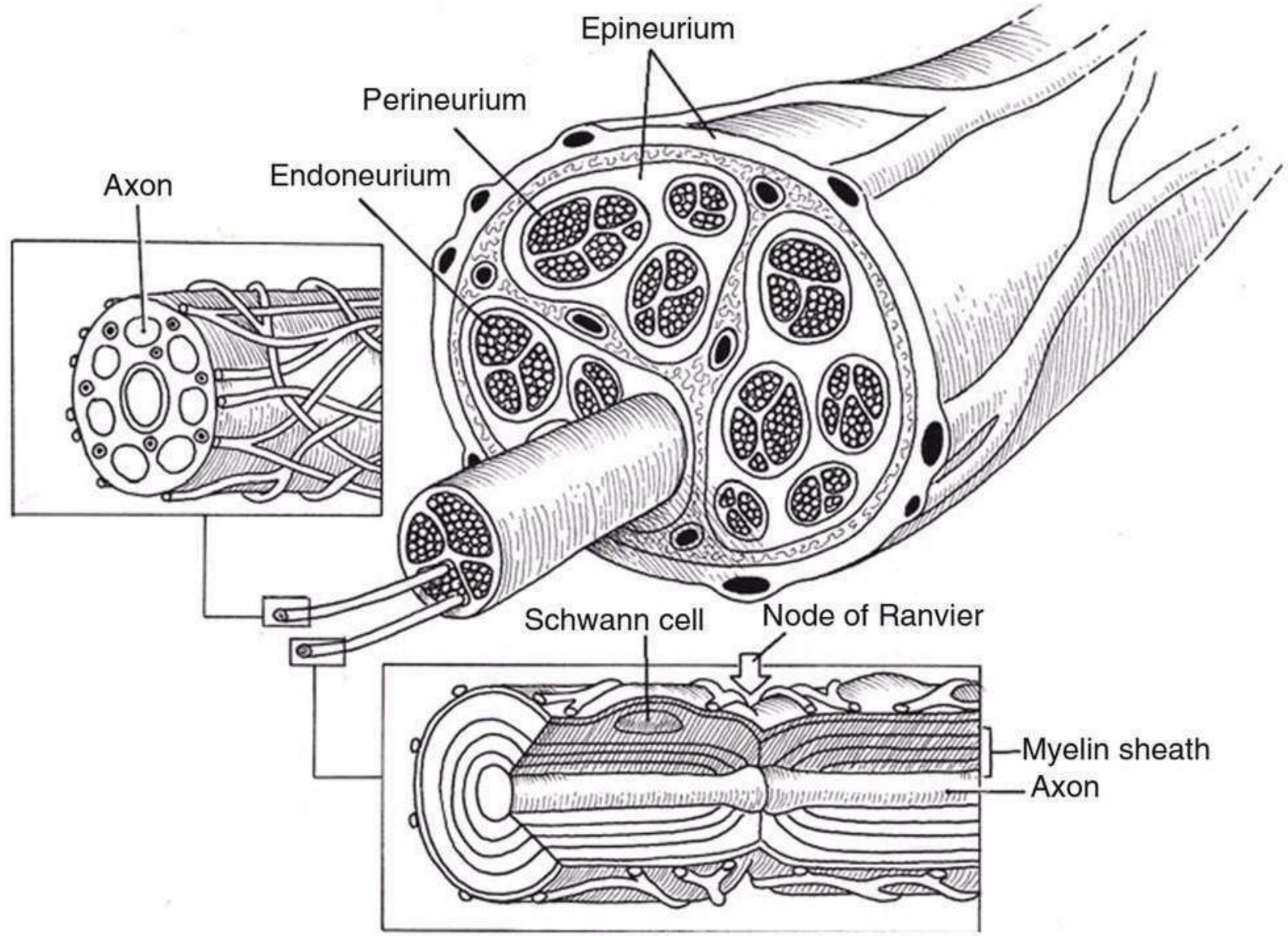


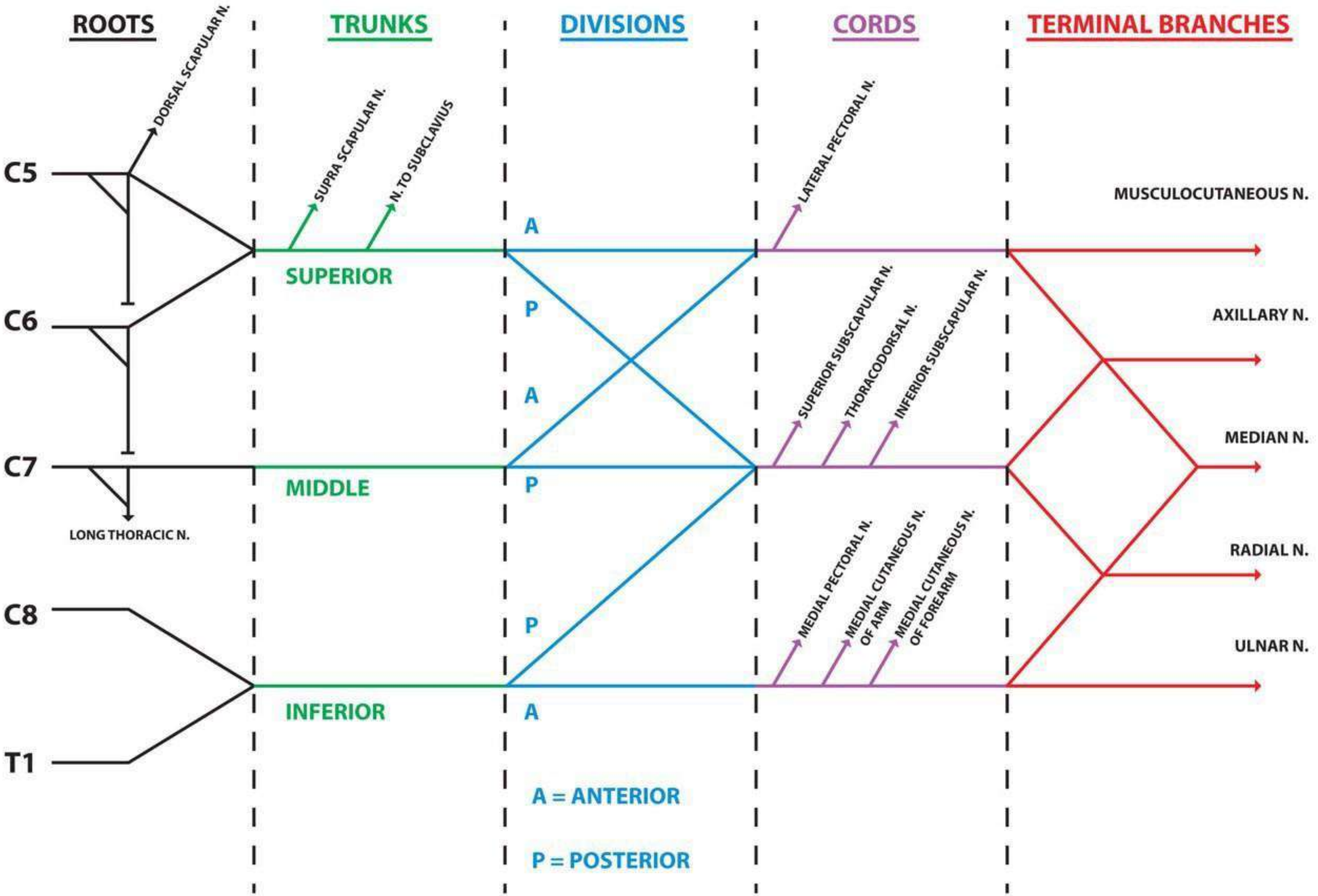
Fig. 3 Cross-sectional anatomy of the peripheral nerve (Adapted with permission from Lundborg G. Nerve injury and repair. New York: Churchill Livingstone; 1988. p. 33.)

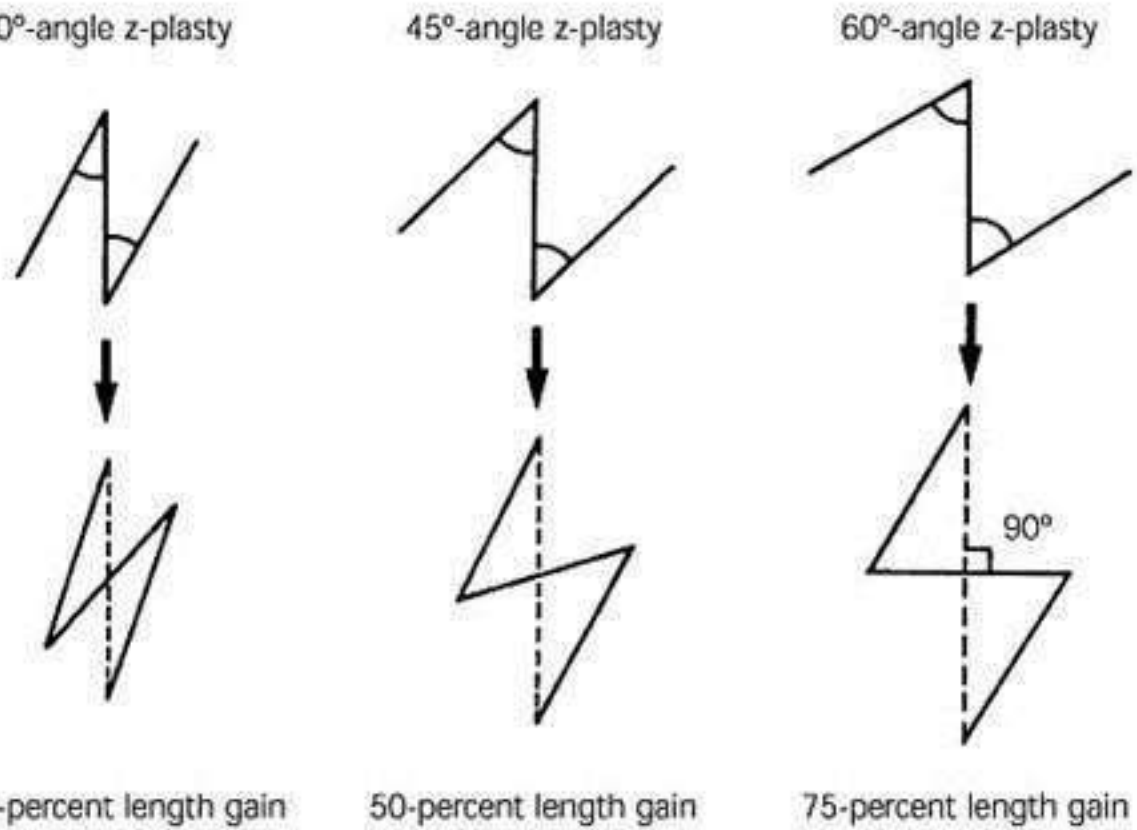
Functional structures

- epineural sheath
  - surrounds peripheral nerve
- epineurium
  - surrounds a group of fascicles to form peripheral nerve
  - functions to cushion fascicles against external pressure
- perineurium
  - connective tissue covering individual fascicles
  - primary source of tensile strength and elasticity of a peripheral nerve
  - provides extension of the blood-brain barrier
  - provides a connective tissue sheath around each nerve fascicle
- fascicles
  - a group of axons and surrounding endoneurium
- endoneurium
  - fibrous tissue covering axons
  - participates in the formation of Schwann cell tube
- myelin
  - made by Schwann cells
  - functions to increase conduction velocity
- neuron cell
  - cell body - the metabolic center that makes up < 10% of cell mass
  - axon - primary conducting vehicle
  - dendrites - thin branching processes that receive input from surrounding nerve cells

Seddon Type	Degree	Myelin Intact	Axon Intact	Endoneurim Intact	Wallerian Degen.	Reversible
Neurapraxia	1st	No	Yes	Yes	No	reversible
Axonotmesis	2nd	No	No	Yes	Yes	reversible
Neurotmesis	3rd	No	No	No	Yes	irreversible

Sunderland Grade	Myelin Sheath	Axon	Endoneurim	Perineurium	Epineurium
I	Disrupted	Intact	Intact	Intact	Intact
II	Disrupted	Disrupted	Intact	Intact	Intact
III	Disrupted	Disrupted	Disrupted	Intact	Intact
IV	Disrupted	Disrupted	Disrupted	Disrupted	Intact
V	Disrupted	Disrupted	Disrupted	Disrupted	Disrupted


ROOTS
TRUNKS
DIVISIONS
CORDS
TERMINAL BRANCHES
DORSAL SCAPULAR N.
C5
C6
C7
C8
T1
LONG THORACIC N.
SUPRA SCAPULAR N.
N. TO SUBCLAVIUS
SUPERIOR
MIDDLE
INFERIOR
A
P
A
P
P
A
LATERAL PECTORAL N.
SUPERIOR SUBSCAPULAR N.
THORACODORSAL N.
INFERIOR SUBSCAPULAR N.
MEDIAL PECTORAL N.
MEDIAL CUTANEOUS N. OF ARM
MEDIAL CUTANEOUS N. OF FOREARM
MUSCULOCUTANEOUS N.
AXILLARY N.
MEDIAN N.
RADIAL N.
ULNAR N.
A = ANTERIOR
P = POSTERIOR



Boundaries of Guyon's canal	
Floor	Transverse carpal ligament, hypothenar muscles
Roof	Volar carpal ligament
Ulnar border	Pisiform and pisohamate ligament, abductor digiti minimi muscle belly
Radial border	Hook of hamate

Zones of Guyon's canal			
	Location	*Common Causes of Compression*	*Symptoms*
Zone 1	Proximal to bifurcation of the nerve	Ganglia and hook of hamate fractures	Mixed motor and sensory
Zone 2	Surrounds deep motor branch	Ganglia and hook of hamate fractures	Motor only
Zone 3	Surrounds superficial sensory branch	Ulnar artery thrombosis or aneurysm	Sensory only

2. How would you classify this condition?
The condition is classified using the Lichtman[7] classification
This is a radiographic classification.

Stage	Description
Stage I	Normal radiographs or linear fracture.
Stage II	Lunate sclerosis. No lunate or carpal collapse.
Stage III	Lunate collapse.
Stage IIIA	No carpal collapse – carpal height normal.
Stage IIIB	Carpal collapse – carpal height diminished.
Stage IV	Lunate collapse with osteoarthritic changes present – midcarpal joint, radiocarpal joint or both.

Stage	Surgical treatment
Stage II or IIIA with negative or neutral ulnar variance	Radius shortening osteotomy. Ulnar lengthening. Capitate shortening.
Stage II or IIIA with positive ulnar variance	Direct revascularisation and external fixation or temporary scapho-trapezio-trapezoid (STT) pinning (stage II only). Radial wedge or dome osteotomy. Capitate shortening with or without capitohamate fusion. Combination of joint levelling and direct revascularisation procedures.
Stage IIIB	STT or scaphocapitate fusion with or without lunate excision and palmaris longus autograft. Radius shortening osteotomy. Proximal row carpectomy (PRC).
Stage IV	PRC. Wrist arthrodesis. Wrist denervation.

Watson Stages	
Stage I	Arthritis between scaphoid and radial styloid
Stage II	Arthritis between scaphoid and entire scaphoid facet of the radius
Stage III	Arthritis between capitate and lunate
note: radiolunate joint spared	

SLAC

Question 12 of 100

At which joint do degenerative changes occur first in a patient with chronic, untreated scapholunate dissociation?

A. Radioscaphoid
B. Radiolunate
C. Scapholunate
D. Capitolunate

Correct answer: A

- Discussion

Stage I of scapholunate advanced collapse (SLAC) is characterized by the presence of radioscaphoid arthritis. A predictable pattern exists of the progression of degenerative changes for SLAC wrist, including stage I (radial styloid involvement at the scaphoid fossa), stage II (scaphoid and entire scaphoid facet involvement), stage III (degeneration between the capitate and lunate), and stage IV (pancarpal involvement). The radiolunate joint is often spared.

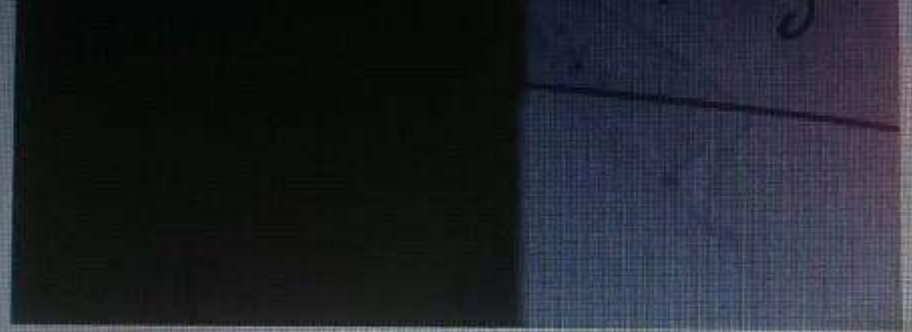
Mayfield Classification		
Stage I	• scapholunate dissociation	
Stage II	• + lunocapitate disruption	
Stage III	• + lunotriquetral disruption, "perilunate"	
Stage IV	• lunate dislocated from lunate fossa (usually volar) • associated with median nerve compression	

Perilunate

3. How would you stage this condition?

Answer: I would use the Woodruff classification system:[10]

Stage 1 - early palmar disease with no contracture (as in this case)
Stage 2 - one finger involved with MCPJ contracture
Stage 3 - one finger MCPJ + PIPJ
Stage 4 - stage 3 + more than one finger involved
Stage 5 - finger in palm deformity

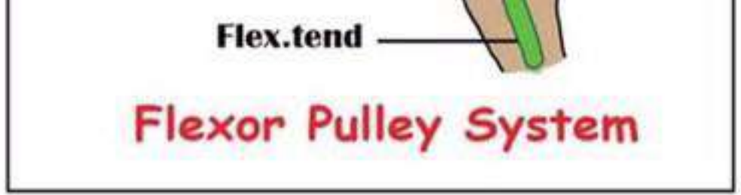


NCSR :

- Websapce : Natatory
- MCP : Central
- PIP : Spiral
- DIP : Retrovascular

** Poland's Syndrome, as demonstrated by the absent sternoclavicular head of major. **Syndactyly and symbrachydactyly** is often seen, in addition and shortening of the fingers

** A 40-year-old male sheet metal worker sustained a crush injury to his hand. **Intrinsic minus deformity**. What pathoanatomic process is responsible for his



Flexor Pulley System

➢ **Principles of repair**

- Direct repair if >60%.
- Within 2 weeks to avoid pulleys collapse.
- 4-6 core strands provide adequate strength for early active ROM.
- Cross flexion crease transversely or obliquely to avoid contractures.
- Core suture to purchase 10mm from cut edge, minimum of 4 size 4/0. Biomechanically strong repair with TiCron (Polyester coated with Si), Fiber-wire, stainless steel suture.
- Move the repaired tendon at end of operation to make sure it is strong enough and doesn't catch on pulleys.
- Circumferential epitendonous suture to improve gliding and strengthen repair using 5/0 or 6/0 mono-filament.
- Preserve/reconstruct A2 & A4 & oblique pulleys to prevent bowstringing causing weakness of flexion.
- Mobilize early to prevent adhesions and allow increased tendon excursion.

5. What are the principles of a tendon transfer?

Answer: Principles when deciding on tendon transfers are:

- Match muscle strength
- Force should be proportional to cross sectional area
- Amplitude should be proportional to length of the muscle
- Work capacity = force × amplitude
- Motor strength will decrease one grade after transfer
- Appropriate tensioning
- Appropriate excursion (can adjust with pulley or tenodesis effect)

Requirements also include patient compliance, no joint contractures, no active infection and grade 5/5 power (this will drop one grade).

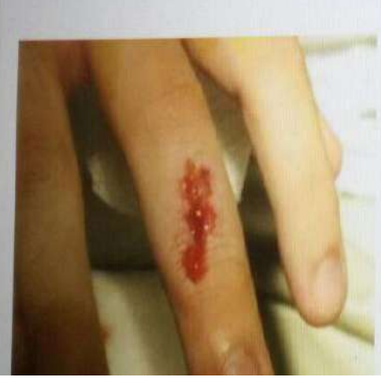
- basic principles
  - donor must be expendable and of similar excursion and power
  - one tendon transfer performs one function
  - synergistic transfers rehabilitate more easily
  - it is optimal to have a straight line of pull
  - one grade of motor strength is lost following transfer

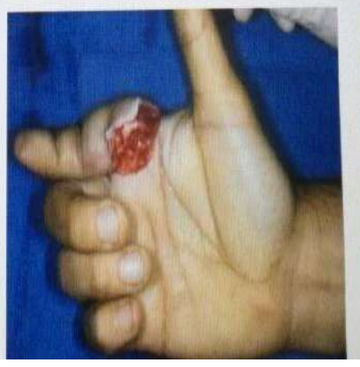
Classification

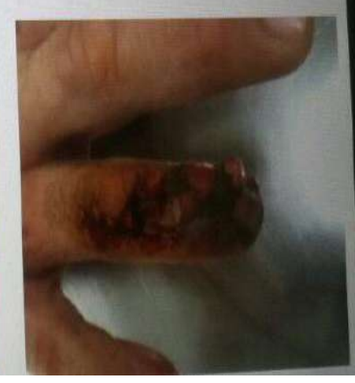
The McLain et al[34] Modified Gustilo Classification for Open Hand Fractures

Type	Size	Description
1	<1 cm	Clean wound without contamination, soft-tissue crush, or fracture comminution
2	>1 cm	Clean wound with no periosteal stripping, soft-tissue envelope intact, no fracture comminution
3	>1 cm	Contaminated wound, fracture with significant comminution and periosteal stripping, soft-tissue crush injury, farm injuries, blast injuries

Reprinted from McLain et al[34] with permission from "The American Society for Surgery of the Hand".







B. Viva : Long & Short :

Section 4 Hand and Upper Limb/Children's Orthopaedics

Chapter 11

Hand and upper limb

John Harrison and Santosh Venkatachalam

Structured oral examination question 1: EPL tendon rupture

EXAMINER: What does the photograph show? (Figure 11.1a.)

CANDIDATE: This is a clinical photograph of the hand with the thumb in an abnormally flexed posture at the IP joint.

EXAMINER: The patient has recently come out of plaster for a distal radius fracture. What is a likely pathology?

CANDIDATE: This is usually caused by an ischaemic rupture of the extensor pollicis longus tendon (EPL) in the third dorsal extensor compartment at 4–6 weeks following an undisplaced distal radius fracture.

EXAMINER: How do you manage this?

CANDIDATE: This is surgically reconstructed using a transfer of extensor indicis (EI) tendon.

EXAMINER: How many incisions would you use?

CANDIDATE: Three incisions are needed – a 1 cm transverse incision over the index finger metacarpal head (EI lies ulnar to the EDC index finger), a 3 cm midline dorsal incision proximal to the wrist to bring the divided EI tendon proximal to extensor retinaculum, a zigzag incision over the thumb metacarpal to identify EPL tendon distal to the rupture. (Figure 11.1b.)

EXAMINER: How do you test for extensor indicis preoperatively?

CANDIDATE: Point the index finger with the middle to little fingers flexed fully (this prevents EDC acting).

Structured oral examination question 2: Enchondroma

An 11 year old reports an injured hand in a fall.

EXAMINER: What do you see in this photograph? (Figure 11.2a.)

CANDIDATE: This is a clinical photograph of a hand showing a swelling at the base of the middle finger with widening of the interspace between the index and middle fingers. No bruising is seen.

EXAMINER: The child has a full range of movements in the hand. Any investigations you would do?

CANDIDATE: I would order a radiograph. The radiograph shows a cystic lesion affecting the proximal phalanx of the middle finger. The proximal radial cortex is markedly thinned and expanded to the radial side causing widening between the middle and index fingers. (Figure 11.2b.)

(a) (b)

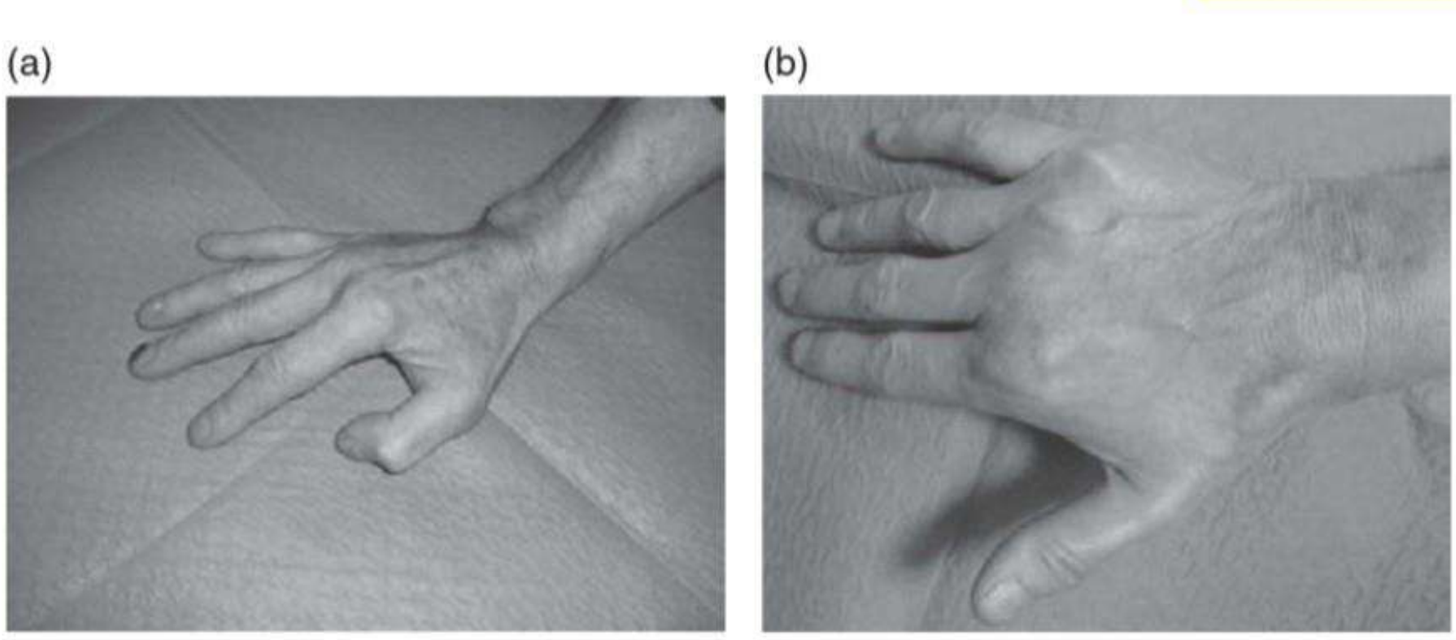
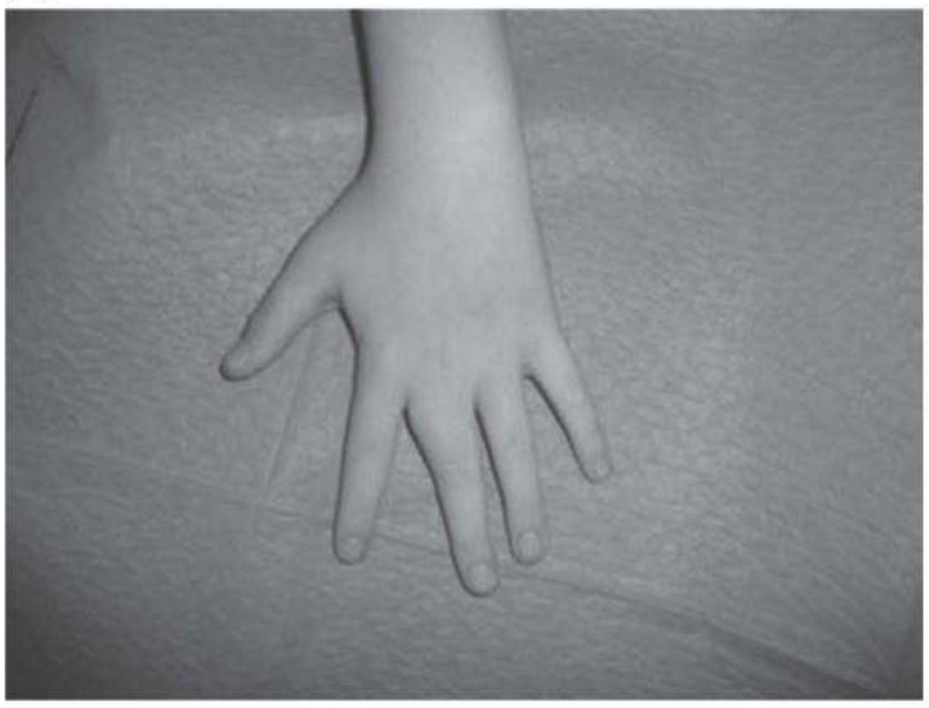


Figure 11.1a Clinical picture of a hand. **11.1b** Showing thumb retropulsion at 6 weeks postoperatively. The three incisions can be seen.

Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination, ed. Paul A. Banaszkiewicz and Deiary F. Kader. Published by Cambridge University Press.

(a)



(b)

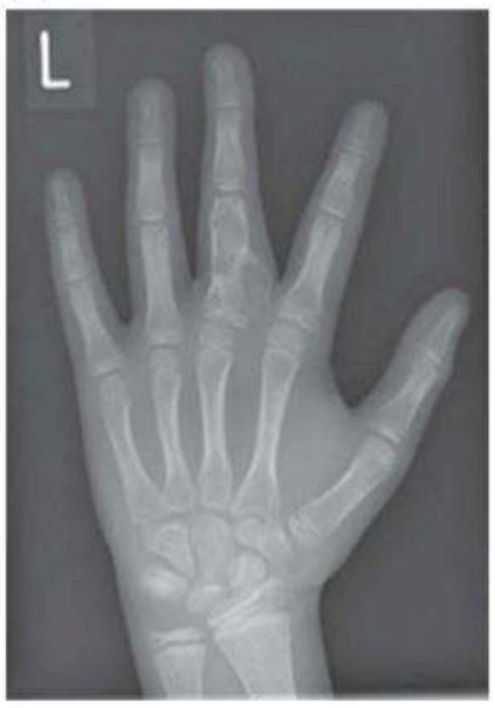


Figure 11.2a Clinical picture of an 11-year-old's hand. **11.2b** Radiograph of the child's hand.

EXAMINER: What is the likely diagnosis?

CANDIDATE: An enchondroma. This is a cartilage tumour of bone.

EXAMINER: How would you manage this lesion?

CANDIDATE: I would take a further history asking about any pain or rapid change in size of the finger. I would look for any bony swellings elsewhere (Ollier's disease – multiple enchondromatosis) or haemangiomas (Mafucci's syndrome). I would explain the tumour has been present prior to the injury and is benign. I would review the patient at 6 weeks and then annually. If it continued to increase in size, I would refer to a hand surgeon for partial excision and bone grafting.

EXAMINER: What is the risk of malignant change?

CANDIDATE: It is very rare for single tumours. The risk is 20–30% for Ollier's disease and near 100% for Mafucci's syndrome.

Structured oral examination question 3: Trans-scaphoid perilunate fracture dislocation

EXAMINER: Tell me what you see here. (Figure 11.3a.)

CANDIDATE: The radiograph demonstrates a trans-scaphoid perilunate fracture dislocation. I would like to see a lateral view.

EXAMINER: This is the lateral view. (Figure 11.3b.) What is the mechanism of injury?

CANDIDATE: Forced wrist dorsiflexion, ulnar deviation, and intercarpal supination. The injury begins at radial side and progresses towards ulnar side through the midcarpal space.

EXAMINER: What classification system do you know of for such an injury?

CANDIDATE: Mayfield has divided it into four sequential stages.

I. Rupture of SL ligament or fracture of the scaphoid.
II. Midcarpal dislocation (dorsal).
III. Lunotriquetral ligament injury.
IV. Lunate dislocation (usually volar).

A greater arc injury goes through the radial styloid, scaphoid and capitate.

EXAMINER: What is the 'spilled tea-cup' sign?

CANDIDATE: Stage IV Mayfield wherein the lunate is dislocated and faces the palm on X-rays. (Figure 11.3c.)

EXAMINER: What is the normal scapholunate angle?

CANDIDATE: Average is 45° (abnormal if $< 30°$ or $> 60°$).

EXAMINER: How will you manage this patient?

CANDIDATE: I will assess the patient clinically and look for neurovascular impairment particularly median nerve. This will need to be reduced as an emergency procedure in theatre under image intensifier and anaesthesia. The scaphoid needs to be fixed and the dorsal ligaments – scapholunate and lunotriquetral – need to be repaired through a dorsal approach. This can be done at a later stage by a hand surgeon. (Figure 11.3d.)

Structured oral examination question 4: Wrist ganglion

EXAMINER: What do you see? (Figure 11.4a.)

CANDIDATE: There is a swelling over the dorsoradial aspect of the wrist. This is typical site for a dorsal wrist ganglion.

EXAMINER: How would you confirm this?

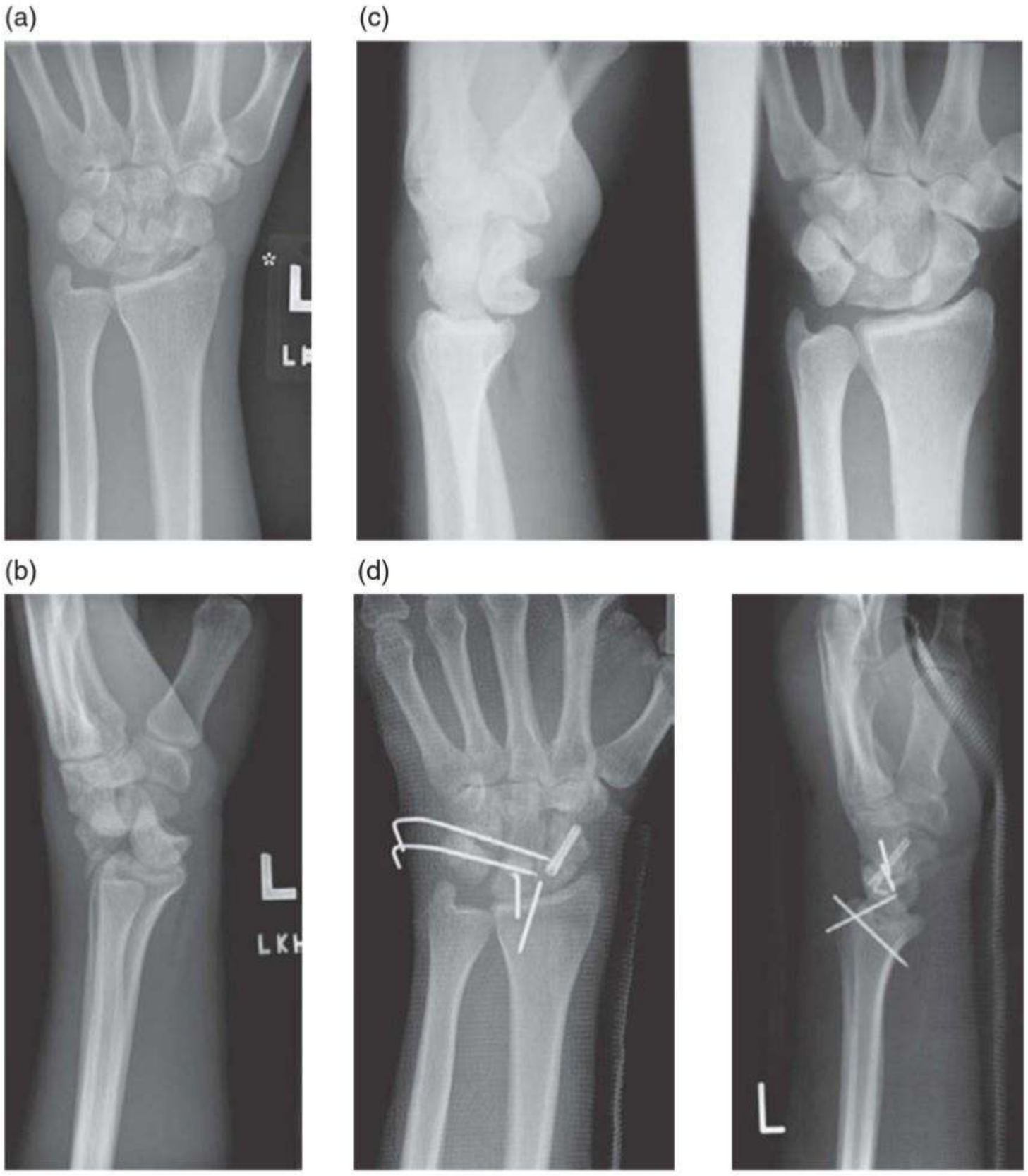


Figure 11.3a Anteroposterior (AP) radiograph of a wrist. **11.3b** Lateral radiograph of the same wrist. **11.3c** AP and lateral radiographs demonstrating a lunate dislocation. **11.3d** Postoperative radiographs showing ORIF of the scaphoid, repair of capitolunate and lunotriquetral ligaments (note the bone anchor in the lunate) and temporary K-wire stabilization of the carpus all done through a dorsal approach.

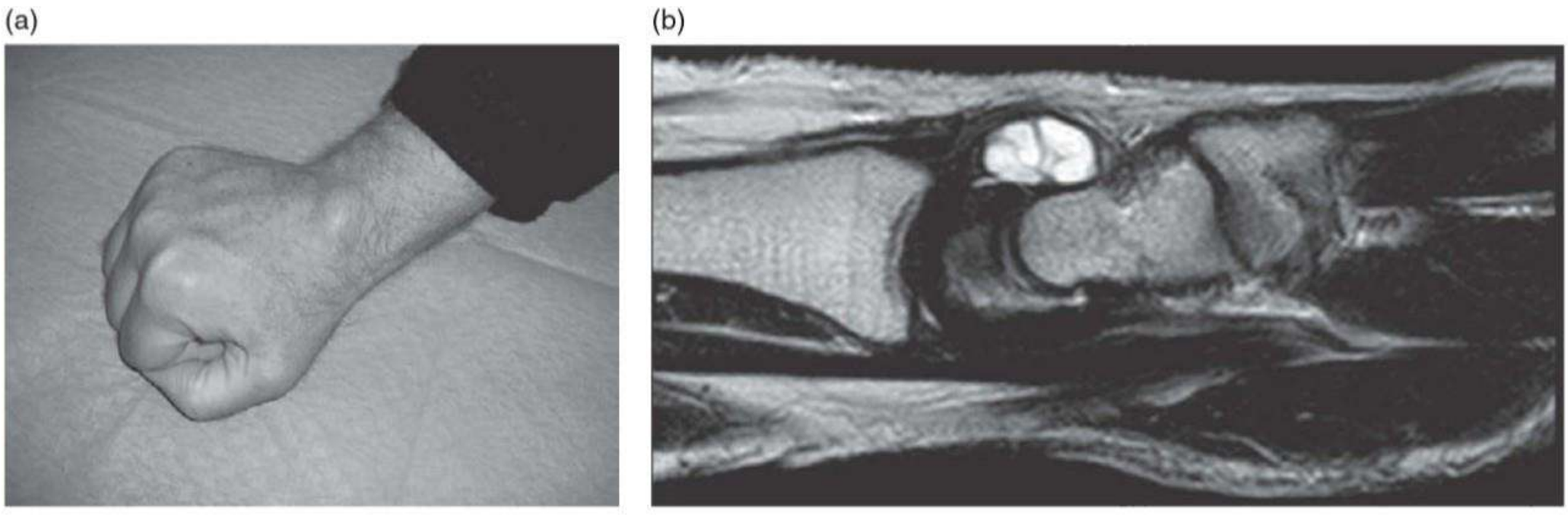


Figure 11.4a Clinical picture of a wrist. **11.4b** Imaging of the same wrist.

CANDIDATE: Take a history specifically asking about any fluctuation in size. Clinically this is a firm, smooth swelling attached to deep structures which classically transilluminates.

EXAMINER: What does this show? (Figure 11.4b.)

CANDIDATE: This is a T2-weighted MR scan showing a well-circumscribed, focal, multiloculated lesion overlying the lunate and capitate dorsally. The ganglion is likely to have arisen from the scapholunate ligament.

EXAMINER: How would you manage this?

CANDIDATE: Counsel the patient this is a benign condition. As long as 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 offered. The recurrence rate is 5%.

Structured oral examination question 5: Scaphoid fracture (proximal pole)

EXAMINER: An apprentice joiner has fallen on his hand at work. What does this show? (Figure 11.5a.) What is the diagnosis?

CANDIDATE: Proximal pole scaphoid fracture. The fracture is undisplaced.

EXAMINER: What is the relevant anatomy?

CANDIDATE: The proximal pole blood supply is from distal to proximal. A proximal 1/5 fracture has a non-union rate of 80–100% when treated non-operatively.

EXAMINER: How do you manage this injury?

CANDIDATE: Open reduction and internal fixation with a screw placed from a proximal entry point and through a dorsal incision.

EXAMINER: In this case, the fracture was treated non-operatively. This is an X-ray at 4 weeks after immobilization in a plaster cast. (Figure 11.5b.)

CANDIDATE: The fracture ends appear sclerosed with some cyst formation around the edges suggesting this is progressing to a non-union. At this stage, the proximal segment appears to be normal density suggesting no loss of vascularity.

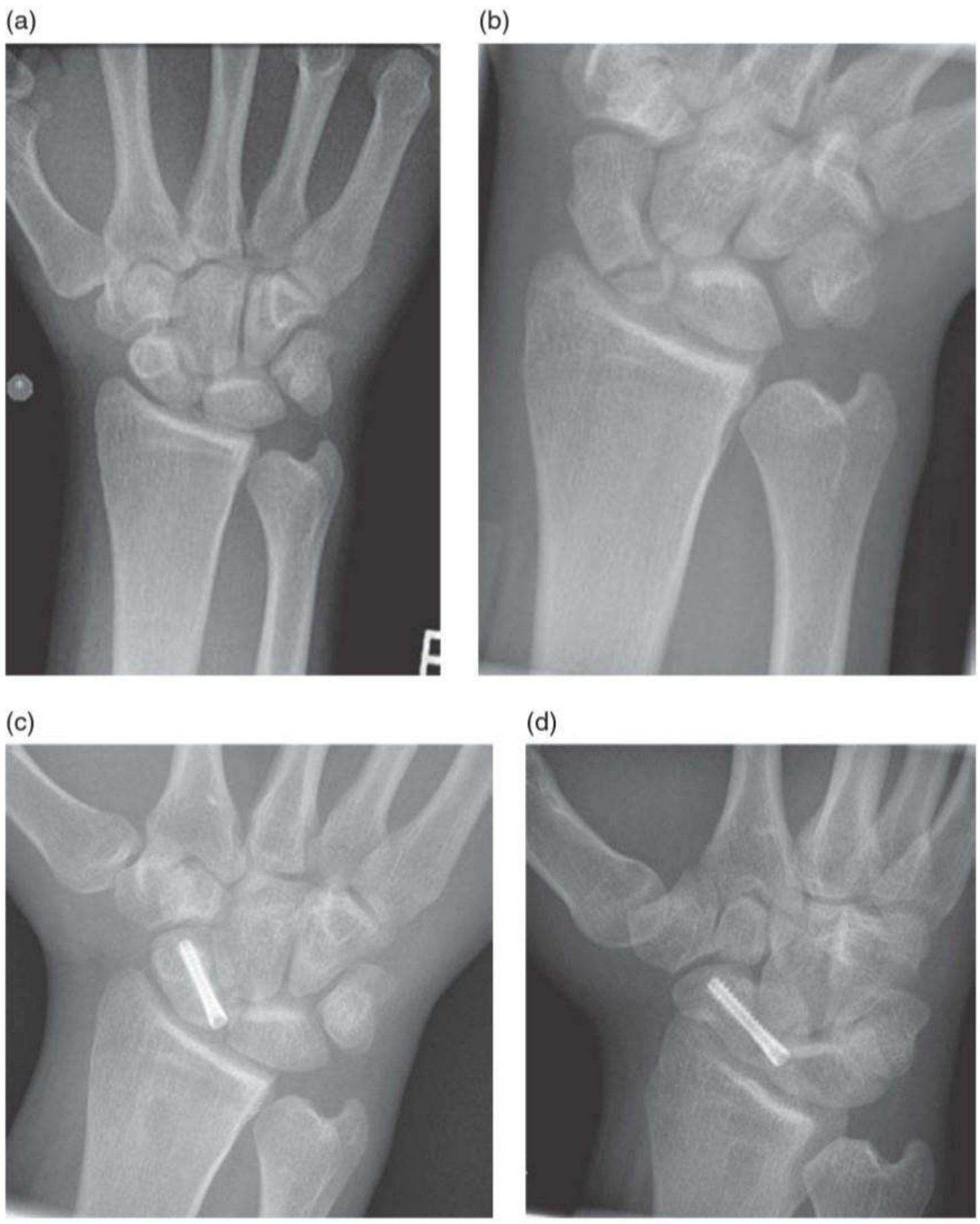


Figure 11.5a AP radiograph of a wrist. **11.5b** AP radiograph of the same wrist at 4 weeks. **11.5c** and **11.5d** Anteroposterior (AP) and oblique radiographs showing union at 8 weeks postoperatively (note the lucency in the distal radius where the graft has been taken from).

EXAMINER: How would you manage this now?

CANDIDATE: Open reduction through a dorsal approach and internal fixation with a vascularized bone graft using a 1,2-intercompartmental supraretinacular artery (1,2-ICSRA) pedicle.[1] (Figure 11.5c, d.)

EXAMINER: What is the natural history of a scaphoid non-union?

CANDIDATE: This will progress to a scaphoid non-union advanced collapse (SNAC) wrist.

I. Arthritis between radial styloid and distal scaphoid.
II. Radioscaphoid fossa involvement.
III. Capitolunate arthritis.
IV. Generalized wrist arthritis.

EXAMINER: Why does the arthritis affect the radial styloid and distal scaphoid initially?

CANDIDATE: With a non-union, the distal scaphoid typically flexes leading to incongruity between the distal scaphoid and radial styloid, whereas the proximal pole of the scaphoid behaves as a ball and socket joint and is not affected by the scaphoid being flexed.

1. Zaidemberg C, Siebert JW, Angrigiani C. A new vascularized bone graft for scaphoid nonunion. *J Hand Surg Am* 1991;**16**(3):474–478.

Structured oral examination question 6: Base of thumb arthritis

EXAMINER: Describe what this radiograph shows. (Figure 11.6a.)

CANDIDATE: This is a radiograph of the hand. The most obvious abnormality is complete loss of joint space of the thumb carpometacarpal (CMC) joint and subchondral sclerosis.

EXAMINER: Do you know any X-ray view which may better show the thumb CMC joint?

CANDIDATE: 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. (Figure 11.6b.)

EXAMINER: Do you know any classification system for this?

CANDIDATE: Eaton classification of thumb CMC joint OA. This is a radiological classification.

I. Widening of the joint.
II. Joint space narrowing.
III. Complete loss of CMC joint space.
IV. Pantrapezial arthritis.

EXAMINER: This is a 56-year-old plaster technician with night pain. How would you treat?

CANDIDATE: I would take a detailed history asking specifically for pain and loss of function. I would examine to see if his pain is localized to the thumb CMC joint by looking for squaring-off of the thumb metacarpal base and performing a grind test.

EXAMINER: Show on my hand how you would do the test.

CANDIDATE: I would stabilize your wrist with my left hand then hold your thumb metacarpal with my right hand and compress and rotate the metacarpal base at the CMC joint. The test is positive if this causes pain and then the pain goes with rotation and distraction. Treatment options include painkillers, activity modification, splints and surgery.

EXAMINER: But what would you do?

CANDIDATE: I would offer him a trapeziectomy. There are no randomized controlled studies to show benefit of this procedure over a ligament reconstruction, fusion or joint replacement.[1] I would explain preoperatively that he is likely to have some loss of grip strength on a permanent basis. (Figure 11.6c.)

1. Davis TR, Brady O, Barton NJ, Lunn PG, Burke FD. Trapeziectomy alone, with tendon interposition or with ligament reconstruction? *J Hand Surg Br* 1997;**22**(6): 689–694.

Structured oral examination question 7: Kienböck's disease

EXAMINER: A 30-year-old electrician presents with wrist pain. What does this show? (Figure 11.7a.)

CANDIDATE: Radiograph shows increased density of the lunate with some cyst formation and partial collapse. There are no obvious arthritic changes in the surrounding joints.

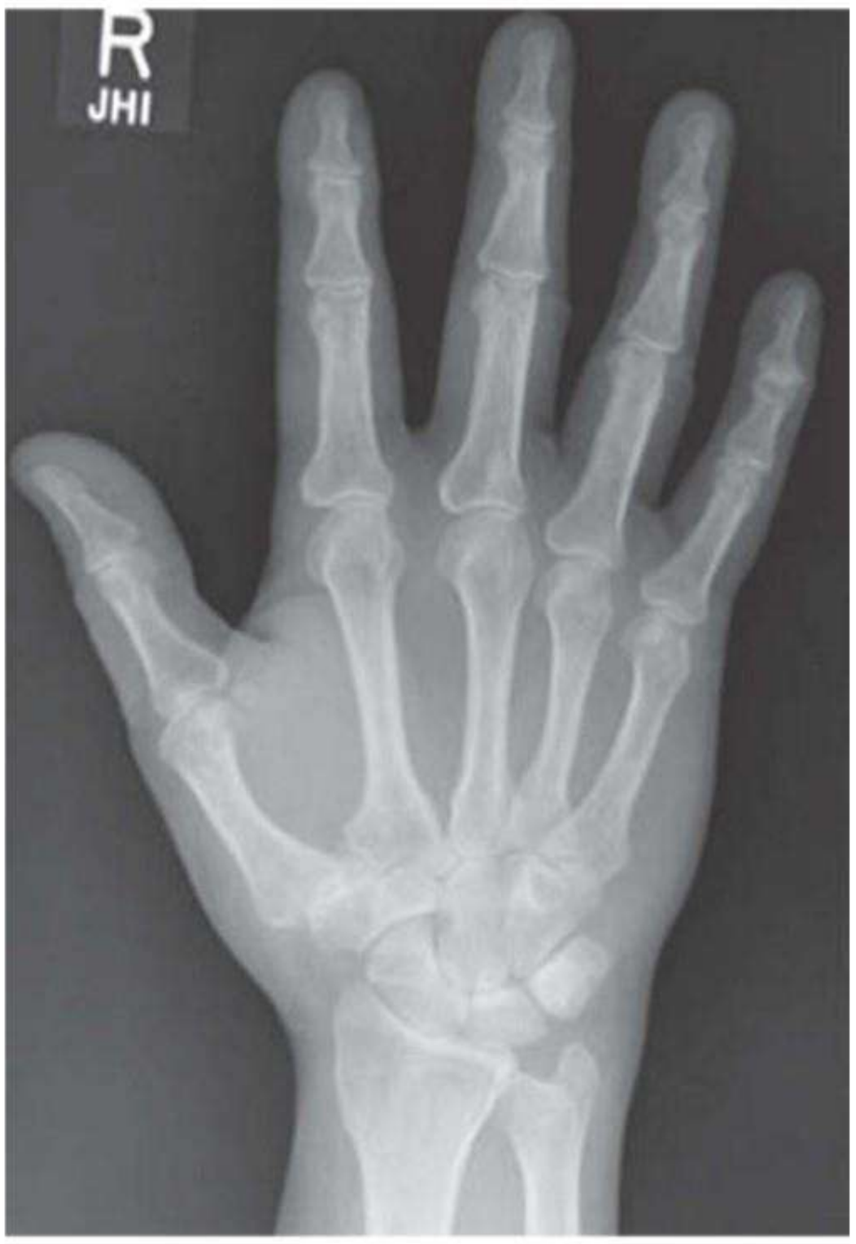
EXAMINER: What is the diagnosis?

CANDIDATE: Kienböck's disease – avascular necrosis of the lunate.

EXAMINER: How do you classify Kienböck's disease?

CANDIDATE: Lichtman classification (radiological).[1]

I. Normal X-rays (changes seen on MRI). II. Lunate sclerosis. IIIA. Fragmentation and collapse of lunate without fixed scaphoid rotation. IIIB. Fragmentation and collapse of lunate with fixed scaphoid rotation. IV. Radiocarpal and midcarpal arthritis.



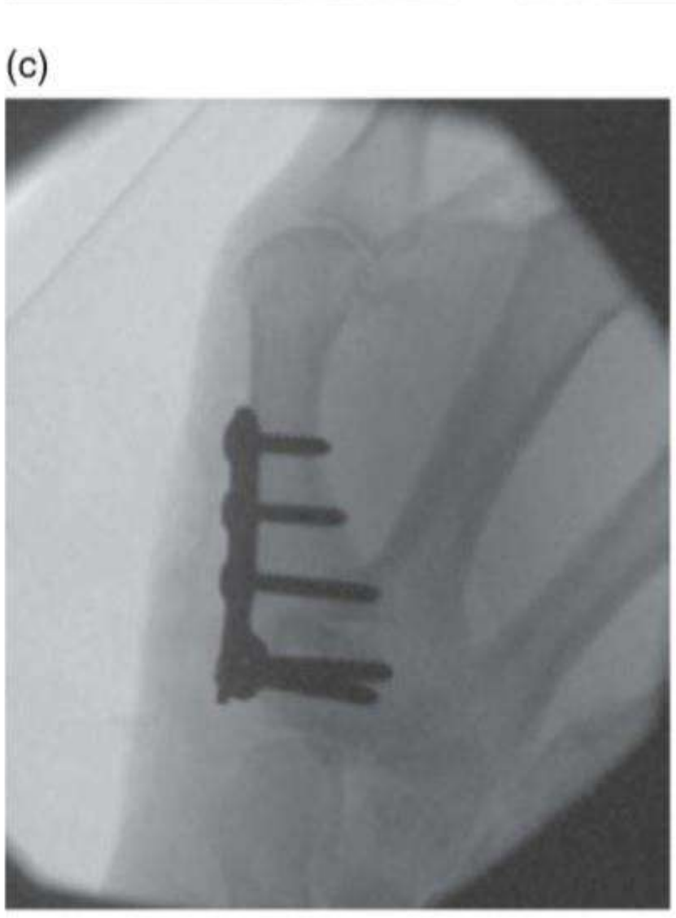


Figure 11.6a Anteroposterior (AP) radiograph of a hand. **11.6b** Robert's view (true AP view of the thumb CMC joint). **11.6c** In this case a fusion was done as this possibly allows improved grip strength in a manual worker but has a higher incidence of complications such as non-union.

EXAMINER: Any predisposing factors?

CANDIDATE: These include ulnar minus variant – this is thought to lead to increased loading on the lunate, poor intraosseous anastomosis and a single extraosseous nutrient vessel.

EXAMINER: What are patterns of intraosseous blood supply?

CANDIDATE: These were described by Gelberman who described a Y pattern (60%), I pattern (30%) and an X pattern (10%).

EXAMINER: What are the treatment options for this condition?

CANDIDATE: This depends on the patient's symptoms and functional demands. Conservative treatment with a period of time in splintage can be discussed. For symptomatic patients with Stage I/II/IIIA disease and if ulnar minus, I would offer a joint levelling procedure, either a radial shortening or an ulnar lengthening. I would prefer a shortening osteotomy since the incidence of non/delayed union is less. If ulnar neutral or plus, consider a procedure aiming to reduce loading on the lunate – either a partial carpal arthrodesis (STT or scapho-capitate) or a

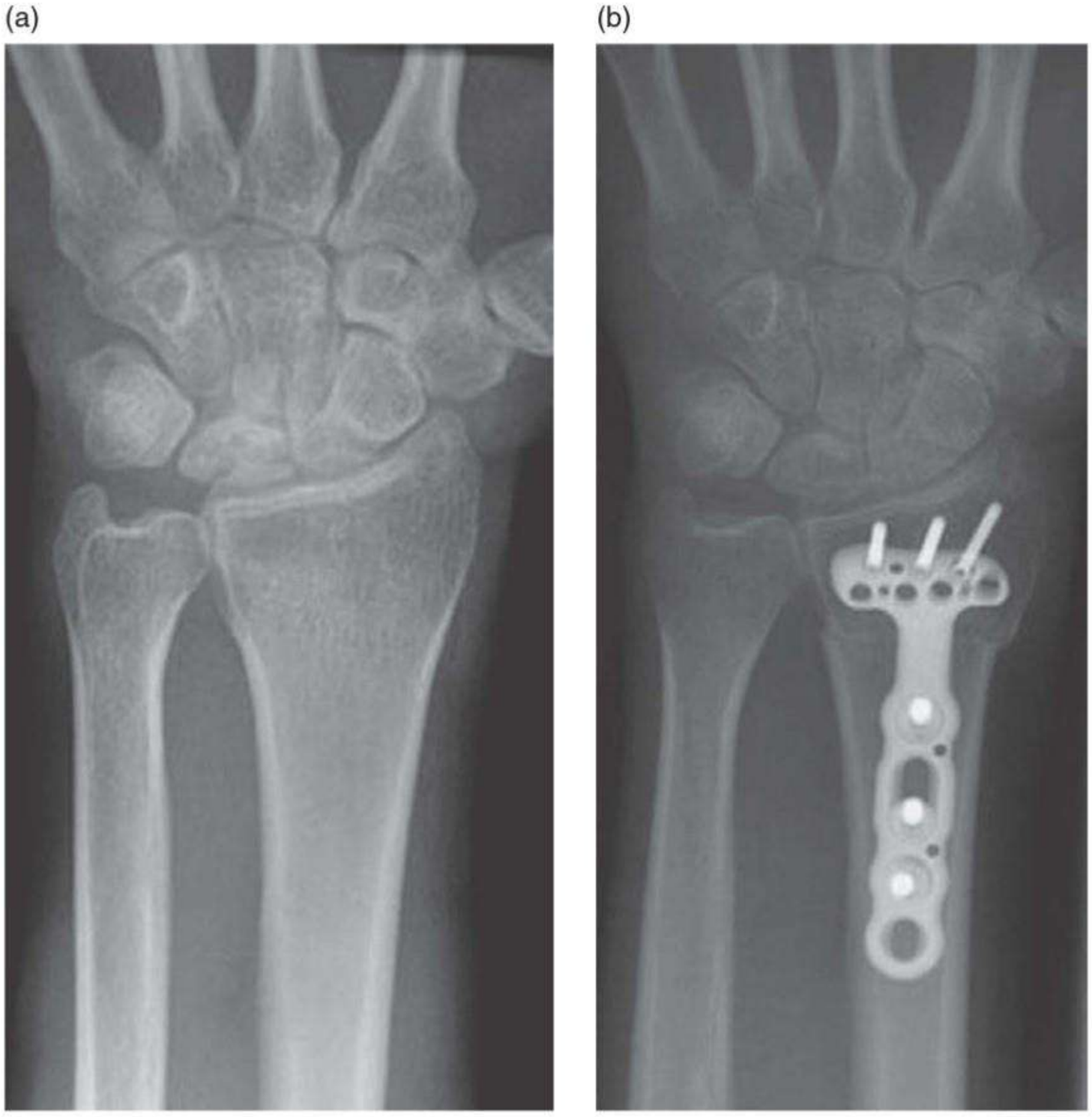


Figure 11.7a Anteroposterior (AP) radiograph of a wrist. **11.7b** Radiograph demonstrating a joint levelling procedure (radial shortening).

capitate shortening. Other options are a vascularized bone graft with 4,5-ICSRA or a distal radial osteotomy.

For Stage IIIB/IV disease, surgical options include a neurectomy, or salvage procedures such as a proximal row carpectomy or a wrist arthrodesis.

EXAMINER: How would you manage this case?

CANDIDATE: I would confirm these were length films for ulna variance, i.e. a wrist PA view with the shoulder flexed 90°, elbow 90° and forearm midprone. If these were, the wrist appears to be ulnar minus and I would offer a joint levelling procedure with a radial shortening. (Figure 11.7b.)

1. Allan CH, Joshi A, Lichtman DM. Kienbock's disease: diagnosis and treatment. *J Am Acad Orthop Surg* 2001; **9**(2):128–136.

Structured oral examination question 8: Dupuytren's disease

EXAMINER: What do you see? (Figure 11.8a.)

CANDIDATE: This is a clinical photograph of a hand showing a cord across the first web space causing a contracture. There is also a cord affecting the index finger causing fixed flexion of the MCP and PIP joints.

EXAMINER: What is the most likely diagnosis?

CANDIDATE: Dupuytren's contracture.

EXAMINER: What is the primary cell involved?

CANDIDATE: Myofibroblasts. They contain smooth muscle actin and lead to contracture of the cord.

EXAMINER: What is Dupuytren's diathesis?

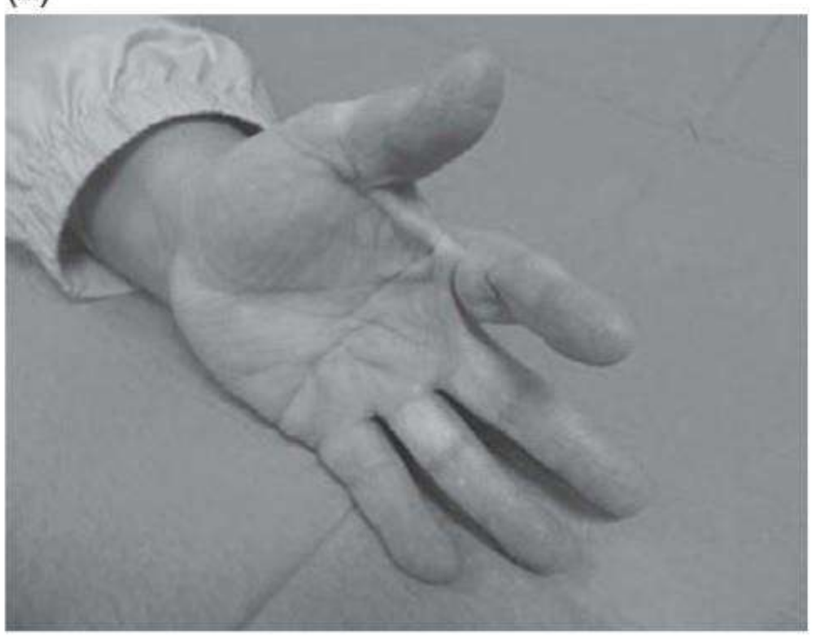
CANDIDATE: This is an aggressive form of the disease with a high recurrence rate. It includes bilateral disease, onset < 40 years, radial side hand involvement, involvement of feet/genitalia, and presence of Garrod's pads.

EXAMINER: What are the predisposing factors?

CANDIDATE: Family history, male, diabetes, epilepsy, alcoholism, smoking, COPD.

EXAMINER: What structures make up a spiral cord and how does it affect the neurovascular bundle?

(a)



(b)

Figure 11.8a Clinical picture of a hand. **11.8b** Illustration of a Z plasty. Mark a perpendicular (white dotted line) to main incision. Mark skin flaps with 60º angles. Make incisions and cross over skin flaps.

CANDIDATE: The pretendinous, lateral and spiral bands, and Grayson's ligament. A spiral cord pulls the neurovascular bundle midline.

EXAMINER: What is a thumb web space contracture called?

CANDIDATE: Commissural cord.

EXAMINER: What are the surgical options for treatment?

CANDIDATE: These are fasciotomy (division of the cord), fasciectomy (excision of the cord) and dermofasciectomy (cord and overlying skin excised).

EXAMINER: What is a Z-plasty? Can you draw it?

CANDIDATE: Technique to manage skin deficiency. Angles should be made at 60° to the incision to achieve a 75% increase in length. (Figure 11.8b.)

EXAMINER: How would you consent me for a fasciectomy?

CANDIDATE: The operation will be carried out under general anaesthetic (you will be put off to sleep) and as a day-case procedure. The aim is to restore lost movement. You will wake up with your hand in a heavy bandage and let home once you are comfortable. You will be seen at 10 days for removal of your sutures. You will then have physiotherapy to help with scar management and regaining finger movement and may need a splint. Complications include the following: early – infection, bleeding and haematoma formation, arterial or nerve injury, amputation and delayed wound healing, or late – recurrence (50% recur but most do not require further surgery) and complex regional pain syndrome.

EXAMINER: Do you know any new treatments for a Dupuytren's contracture?

CANDIDATE: Collagenase (Xiapex®) injections are now licensed in Europe for a Dupuytren's contracture. Two randomized controlled studies of 374 patients comparing Xiapex® to placebo have shown benefit with 60% showing correction to 5° of full extension.[1]
The patient has an injection at three points along the cord and returns the next day for a finger-extension procedure.

1. Hurst LC, Badalamente MA, Hentz VR *et al.* Injectable collagenase from *Clostridium histolyticum* for Dupuytren's contracture. *New Engl J Med* 2009; **361**(10):968–979.

Structured oral examination question 9: Ulnar collateral ligament injury thumb

EXAMINER: What is being tested? (Figure 11.9a.)

CANDIDATE: Clinical photograph showing stressing of the ulnar collateral ligament of the thumb. This shows opening of $>20°$ but should be compared with the opposite side.

EXAMINER: What is shown in this intraoperative photograph? (Figure 11.9b.)

CANDIDATE: This is an intraoperative photograph showing an approach to the medial side of the thumb MCP joint possibly for an ulnar collateral ligament repair.

EXAMINER: What structure has been divided to give this view?

CANDIDATE: The adductor pollicis tendon attachment to the EPL tendon.

EXAMINER: What lesion is shown in the photograph?

CANDIDATE: A Stener lesion. The torn proximal end of the ligament is retracted proximal to the adductor pollicis tendon and prevents the ligament from healing.

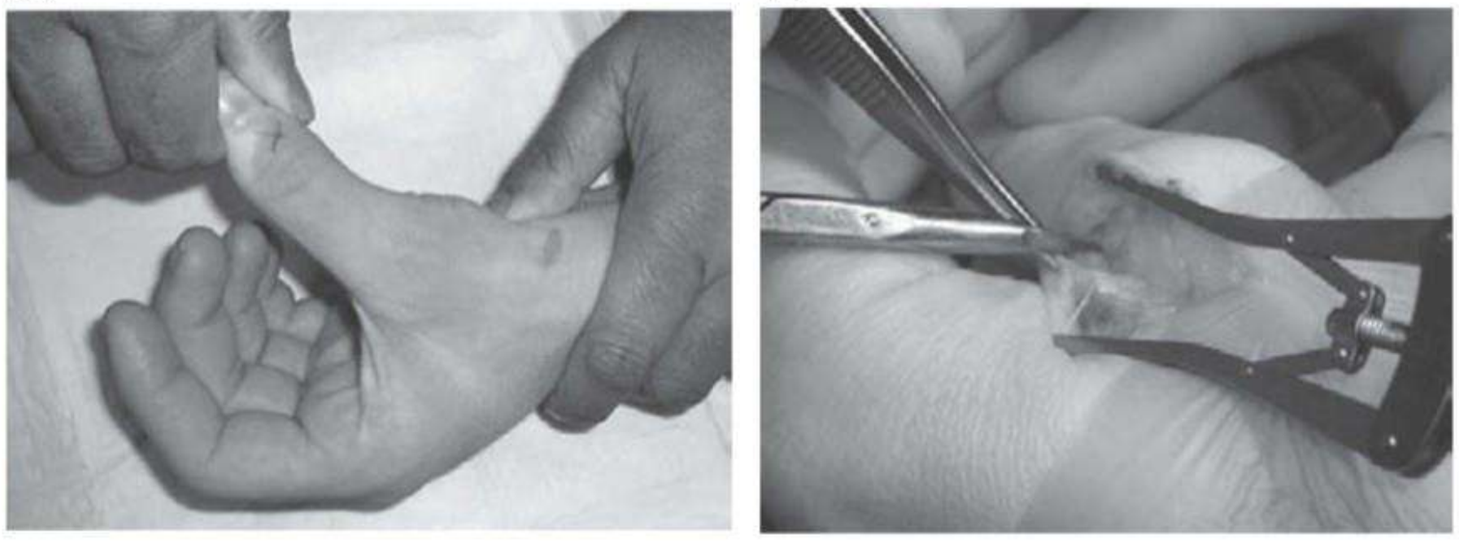


Figure 11.9a Clinical picture of a thumb. **11.9b** Intraoperative picture of a thumb during a UCL repair. The scissor tips are pointing at the bare insertion of the ligament to the base of the proximal phalanx. This is a chronic case and the scarred proximal ligament is lying bunched up over the metacarpal head.

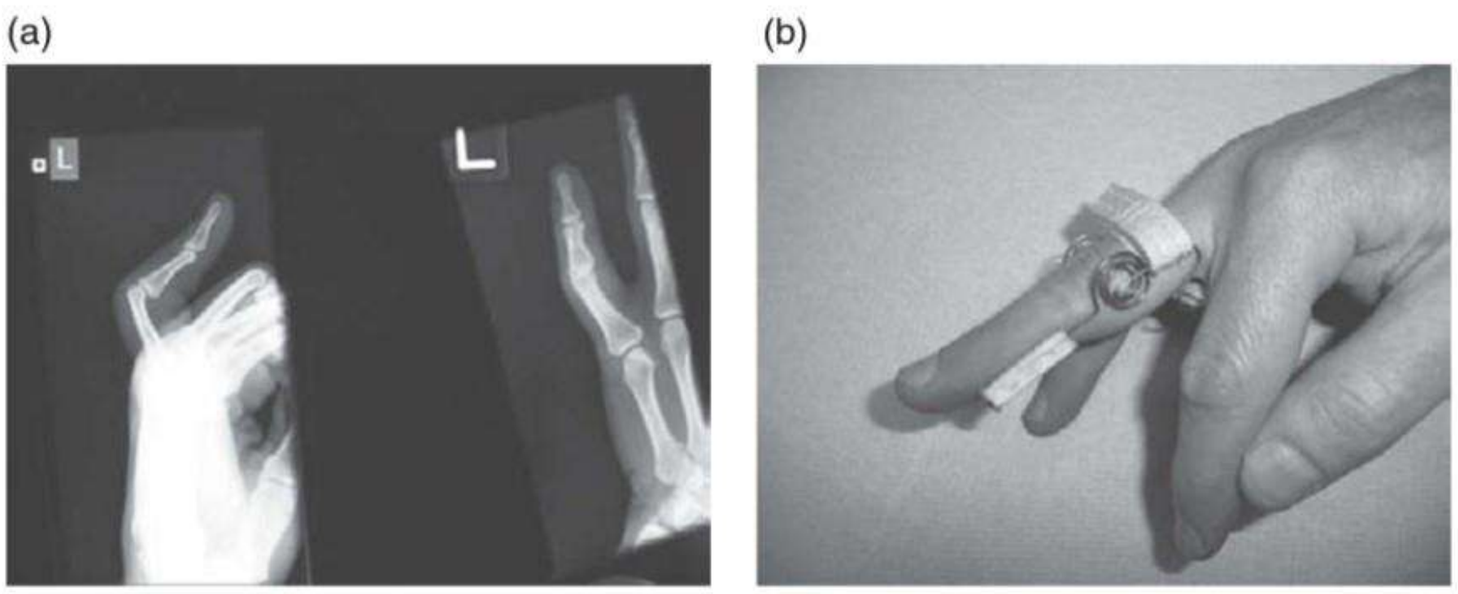


Figure 11.10a Anteroposterior (AP) and lateral radiographs of a little finger. **11.10b** Picture of a splint used to manage a Boutonnière deformity.

Structured oral examination question 10: Boutonnière deformity

EXAMINER: A rugby player has injured his finger during a game. He has pain and swelling and attends casualty where X-rays are taken. Describe what you see and the likely diagnosis. (Figure 11.10a.)

CANDIDATE: The radiographs show flexion at the PIP joint of the little finger and hyperextension at the DIP joint.

EXAMINER: How would you manage this?

CANDIDATE: I would test the central slip of the extensor mechanism by Elson's test – the PIP joint is flexed to 90° over the edge of a table, and the patient is asked to extend the finger against resistance (the examiner presses on the middle phalanx). A positive test shows weakness of extension of the PIP joint with hyperextension of the DIP joint due to recruitment of the lateral bands.

CANDIDATE: A closed central slip rupture if left may lead to a Boutonnière deformity. I would offer a dynamic splint – a Capener – to keep the PIP joint passively extended but allow active flexion. (Figure 11.10b.) This needs to be worn for 6 weeks. I would explain a mild flexion deformity is likely even with treatment.

Structured oral examination question 11: Rheumatoid arthritis

EXAMINER: What does this picture show? (Figure 11.11a.)

CANDIDATE: Clinical photograph of a hand with swelling of the MCP joints and ulnar drift of the digits. These are features of rheumatoid arthritis.

EXAMINER: How would you treat this patient?

CANDIDATE: I would ask about pain and any functional loss asking specifically about ADLs such as doing up buttons, writing, handling coins, etc. The patient may also be concerned about cosmesis. If the patient is struggling with pain I would offer her MCP joint replacements using silastic implants. I would explain surgery aims to correct the deviation of the fingers and would improve any pain from those joints, the appearance of the hand and the ability to pinch (the range of movement and grip strength are unlikely to improve).

EXAMINER: Any medications you would ask about pre-operatively?

CANDIDATE: I would ask about any steroid medication which would need covering perioperatively. Newer anti-TNF alpha treatments such as Infliximab and Etanercept should not be given within a couple of weeks of surgery and until after the

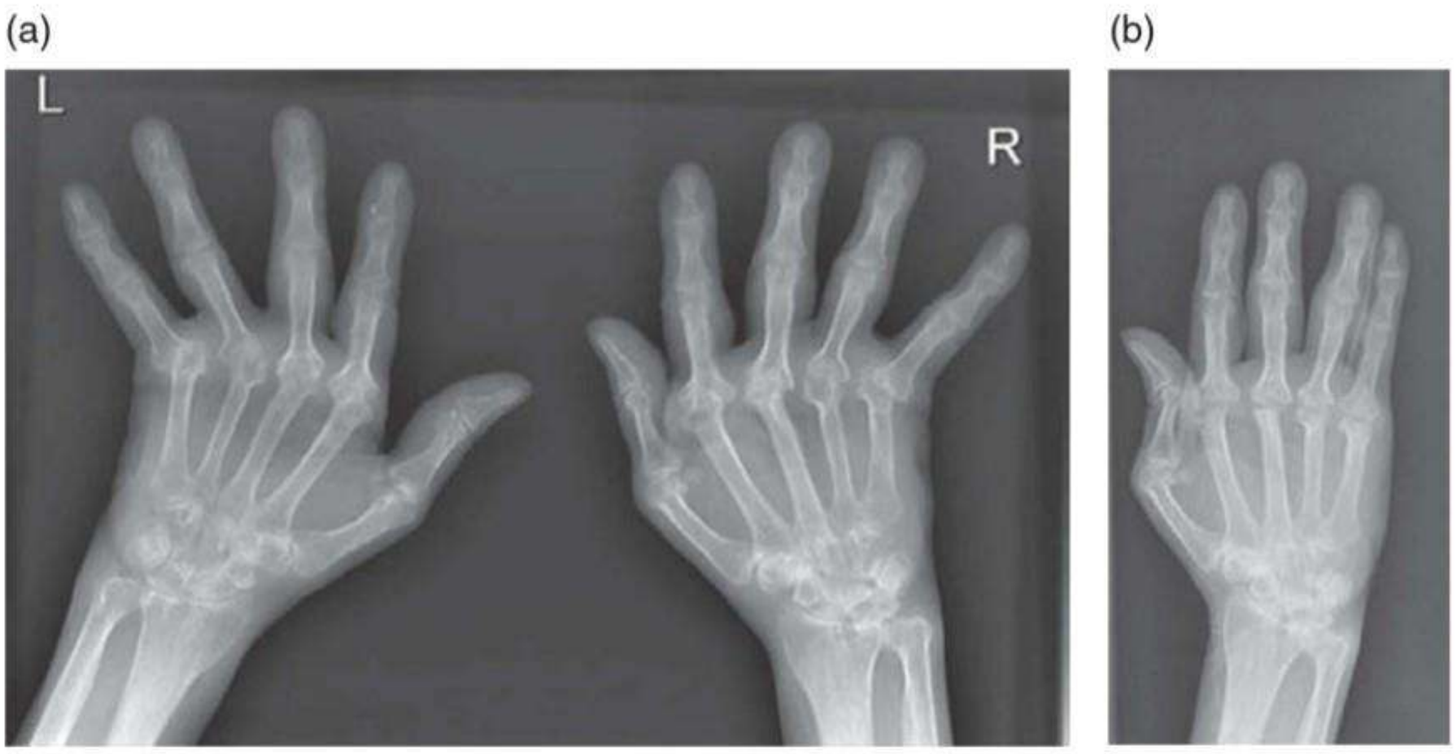


Figure 11.11a Radiographs of a patient's hands. **11.11b** Postoperative anteroposterior (AP) radiograph of a hand.

wound has healed. Methotrexate can be continued as there is no evidence to suggest it increases the risk of infection.

EXAMINER: What are the possible complications of MCP joint replacements? (Figure 11.11b.)

CANDIDATE: Early – infection, dislocation prosthetic stem. Late – recurrence ulnar drift, prosthesis wear/breakage, silicone synovitis.

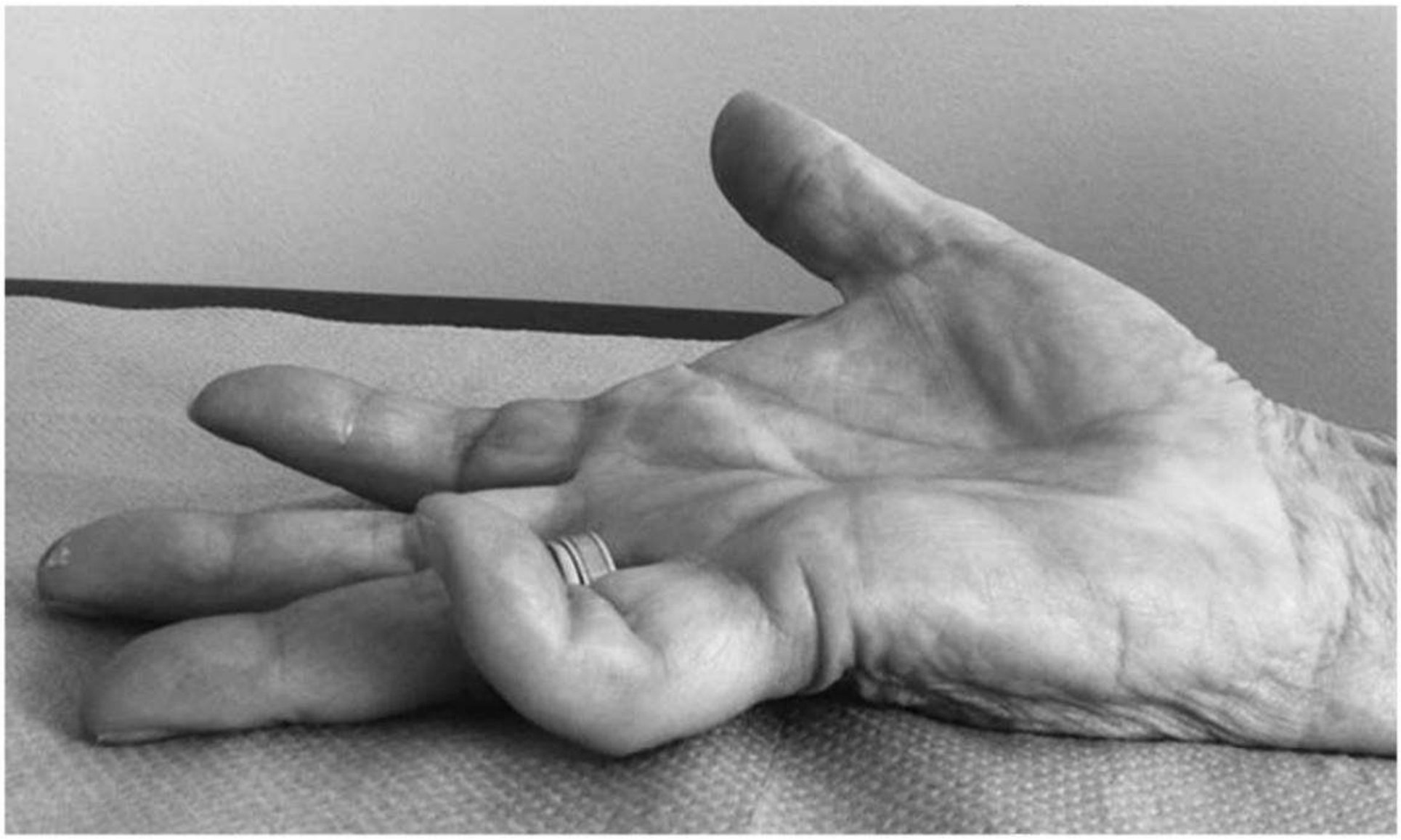
EXAMINER: What are the three most common rheumatoid hand operations?

CANDIDATE: Wrist fusion, MCP joint replacement and thumb MCP joint fusion.

Other potential cases

- Distal radius fracture.
- Infection.
- Flexor tendon injury.

Viva 1



What is the likely diagnosis?

What are the risk factors for this condition?

What are the two main components seen in the histology of mature tissue from this condition?

What are the management options?

What are the risks of surgical treatment?

What is the likely diagnosis?

The clinical photograph shows flexion of the right little finger metacarpophalangeal (MCP) joint, sug-

What is the likely diagnosis?

The clinical photograph shows flexion of the right little finger metacarpophalangeal (MCP) joint, suggestive of Dupuytren's contracture.

What are the risk factors for this condition?

The risk factors associated with Dupuytren's disease include: positive family history; liver disease; high alcohol intake; diabetes mellitus, and epilepsy.

What are the two main components seen in the histology of mature tissue from this condition?

Classic histological appearance is the presence of myofibroblast cells (probably derived from fibroblasts) and thick collagen fibres.

What are the management options?

The management options are non-operative measures and operative procedures.

Non-operative options include observation (and possibly splintage, especially at night).

Some authors have reported that steroid injections to early palmar nodules may reduce local tenderness. Although promising short-term results have been reported with collagenase injection, no long-term results are available.

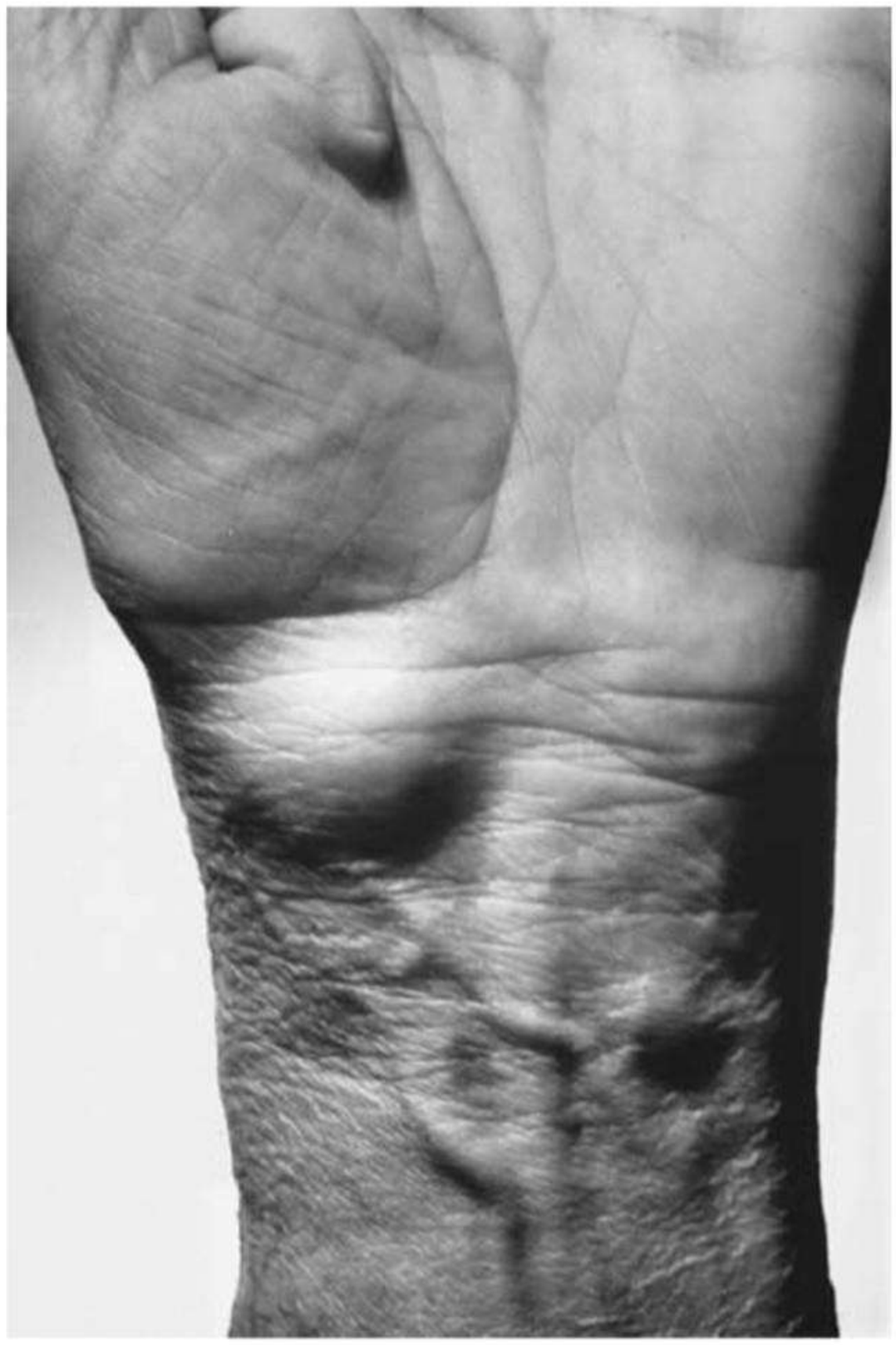
Surgical options include:

- Percutaneous fasciotomy, especially for mild contractures affecting MCP joint contractures
- Segmental/palmar fasciectomy
- Regional fasciectomy (and Z-plasty closure or skin grafting)
- Dermo-fasciectomy and skin grafting
- Proximal interphalangeal (PIP) joint arthrodesis (for severe or recurrent disease)
- Occasionally, amputation of the digit (for severe or recurrent disease)

What are the risks of surgical treatment?

Specific surgical risks include:

- Delayed wound healing, infection
- Tendon, nerve, and vessel injury
- Temporary or permanent numbness
- Necrosis of the digit and amputation
- Incomplete correction
- Recurrence and re-operation
- Joint stiffness
- Reduced flexion/extension especially at the PIP joint
- Pain, swelling, and tenderness; occasionally chronic regional pain syndrome



Reproduced from C. Bulstrode et al., Oxford Textbook of Trauma and Orthopaedics second edition, 2011, figure 6.13.2, page 512, with permission from Oxford University Press.

What is the likely diagnosis of the cystic, soft-tissue lump shown in the photograph? From what structure does it commonly arise?

What clinical test, outpatient procedure, and simple imaging investigation can be performed to confirm the diagnosis?

Give a histological definition of this condition.

What are the other sites for these cystic swellings in the wrist and hand?

How would you manage this condition in a 26-year-old woman who works as a secretary and presents to you for the first time?

What is the risk of recurrence post-excision?

What is the likely diagnosis of the cystic, soft-tissue lump shown in the photograph? From what structure does it commonly arise?

The appearance of the lump at the wrist is suggestive of a ganglion cyst. Approximately two-thirds of such cysts originate in the radiocarpal joint. The remaining third arise from the scapho-trapezoid joint.

What clinical test, outpatient procedure, and simple imaging investigation can be performed to confirm the diagnosis?

Clinical test = compressible lump which trans-illuminates
Outpatient procedure = aspiration of the ganglion under local anaesthetic
Simple imaging investigation = ultrasound scan

Give a histological definition of this condition.

A ganglion cyst is a fluid-filled cavity lined by compressed collagen and a few cells.

What are the other sites for these cystic swellings in the wrist and hand?

1. Ganglia in the hand are commonly seen over the dorsum of the wrist where they commonly arise from scapholunate ligament
2. Cysts arising from the distal interphalangeal (DIP) joint present as dorsal cysts and are called dorsal distal ganglia, mucoid, or mucous cysts
3. Smaller, firmer cysts may be found in relation to the flexor tendon sheath in the region of the A2 pulley. These are called palmar digital ganglia, flexor sheath ganglia, or pearl ganglia

Interosseous ganglia are uncommon, but when present are often in the lunate bone.

How would you manage this condition in a 26-year-old woman who works as a secretary and presents to you for the first time?

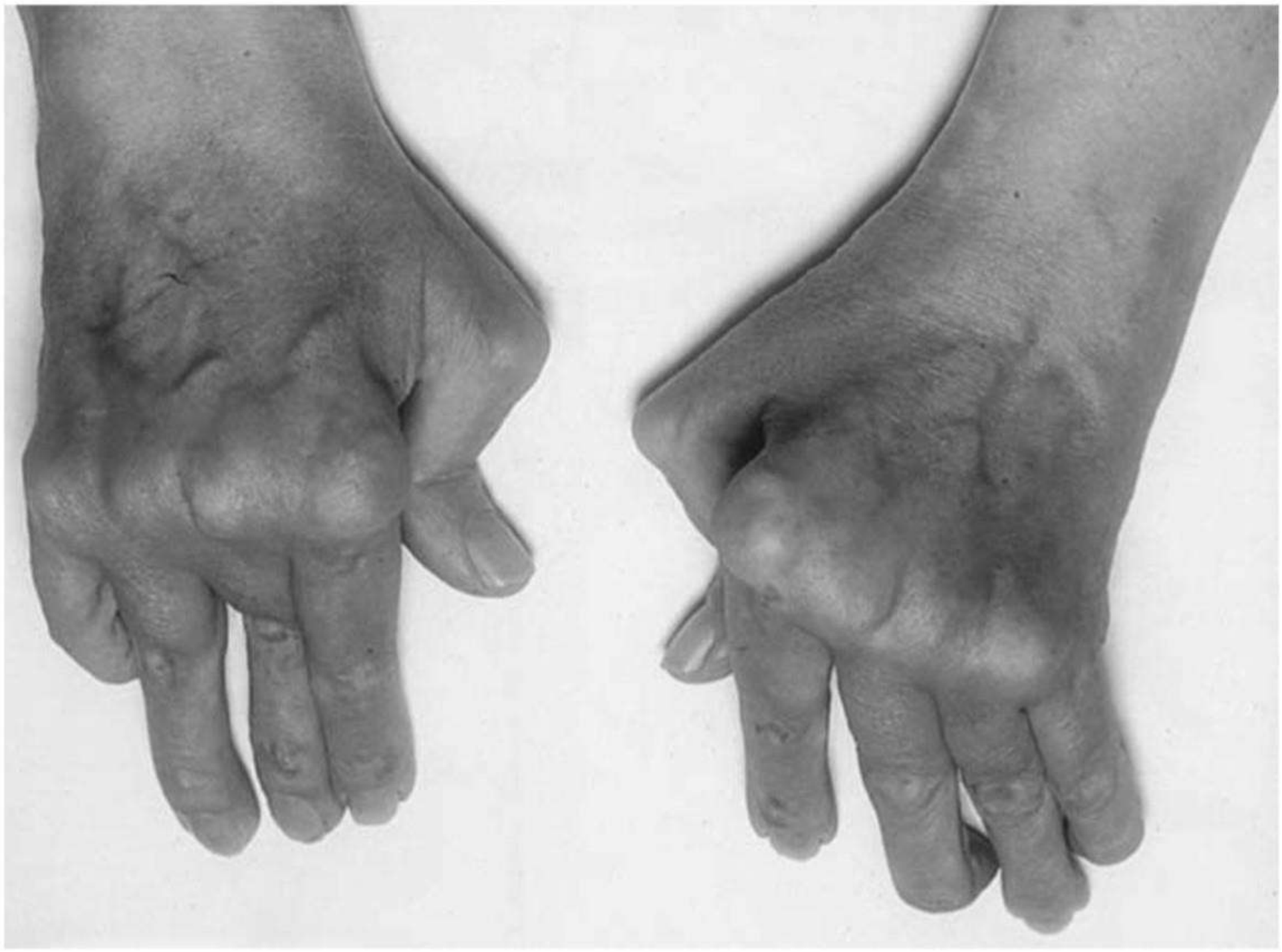
I would explain:

1. That a ganglion is a common benign cyst
2. That during the natural history of a ganglion it can often fluctuate in size periodically and may resolve spontaneously if simply observed
3. Treatment options and their potential risks include:
   - Simple observation (few if any risks)
   - Aspiration of the cyst (small risks of haematoma or infection, radial artery damage, and recurrence)
   - Surgical excision of the ganglion, open or arthroscopic (risks of anaesthesia and surgery including nerve, vessel, and tendon injury, haematoma and infection, pain, swelling, tenderness, stiffness, and recurrence)

What is the risk of recurrence post-excision?

The risk of recurrence in some published series is similar for all three treatment options above, so observation is the safe course of action.

Viva 3



Describe the picture.

How would you grade the thumb condition radiologically?

Why does subluxation occur in this condition?

How could you explain the hyperextension deformity of the MCP joint?

What are the management options?

How would you treat this patient who has unremitting carpo-metacarpal (CMC) joint pain despite full non-operative treatment and who is fit for surgery?

What are the advantages and disadvantages and surgical risks with simple excision arthroplasty?

Describe the picture

This is a clinical photo of the dorsal aspect of the hands showing a symmetrical, deforming, polyarthropathy consistent with rheumatoid arthritis. There are bilateral Z-shaped thumbs, swan-necking of the right middle finger, and marked ulnar deviation of all fingers at the MCP joints.

How would you grade the thumb condition radiologically?

I would use the Eaton and Littler system to stage this condition.

- Stage I—joint space widening, normal articular contours
- Stage II—up to one-third subluxation (on stress radiographs, thumbs resting on plate and pushing against each other): osteophytes < 2 mm, scapho-trapezio-trapezoidal (STT) joint is normal
- Stage III—marked narrowing of joint space, more than one-third subluxation: osteophytes > 2 mm
- Stage IV—pan-trapezial arthritis

Why does subluxation occur in this condition?

The palmar oblique ligament (also known as the 'beak' ligament) is a very strong ligament extending from the trapezium to the base of the first metacarpal. Degenerative attenuation and rupture of this ligament results in dorsal subluxation of the first metacarpal.

How could you explain the hyperextension deformity of the MCP joint?

Dorsal subluxation of the CMC joint causes metacarpal adduction, a thumb in the palm deformity, and reduction in thumb span. This leads to a secondary compensatory hyperextension at the MCP joint in an effort to increase the thumb span.

What are the management options?

Non-operative options include: oral analgesia; activity modification; use of splints; physiotherapy; and intra-articular steroid injection which could be performed in the outpatient clinic or under fluoroscopic guidance.

Operative options include:

1. Excision of the trapezium offers satisfactory pain relief in most cases and preserves movement. Results are reliable. However, pinch grip may be weakened
2. The addition of a suspension procedure and tendon interposition has been shown to offer no extra benefit
3. Implant arthroplasty has failed to offer good long-term results and early implant failure has made this procedure less popular
4. Rarely, CMC arthrodesis is performed in young adult manual workers as this procedure offers a stable thumb with good pinch grip. However, the manoeuvrability of the thumb is affected
5. First metacarpal-basal osteotomy may be considered, especially in earlier stages of the disease

How would you treat this patient who has unremitting CMC joint pain despite full non-operative treatment and who is fit for surgery?

I would offer this patient excision of the trapezium and fusion of the MCP joint under general anaesthetic (GA)/regional block. I would perform the procedure as a day-case.

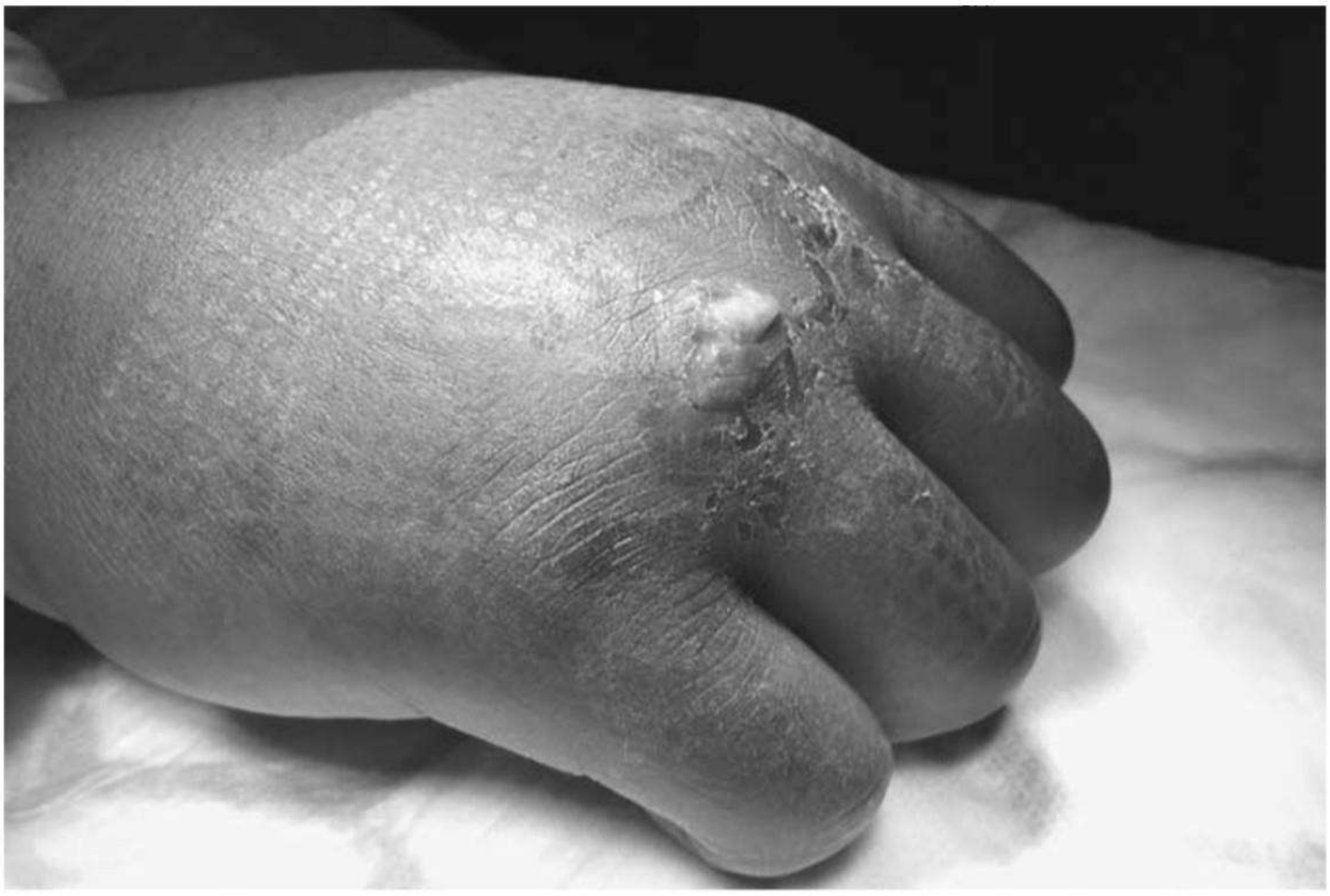
What are the advantages and disadvantages and surgical risks with simple excision arthroplasty?

Trapezium excision results in good pain relief and consequently improved function, but slight shortening of the thumb can cause reduced power of pinch. Risks specific to the procedure include painful scar, infection, nerve damage (superficial branch of the radial nerve), blood vessel damage (radial artery), incomplete relief of symptoms (especially if adjacent joints are affected by osteoarthritis), a relatively slow recovery of function and attainment of maximal pain relief, and instability of the carpus.

Viva 4

Viva 4

A 20-year-old man presents 48 h after he was involved in a fight with another person, when he sustained a punching injury shown in the photograph below.



What is the likely nature of this injury?

How would you assess this patient?

How would you treat this injury?

Which organisms commonly cause infection with this type of injury?

Which antibiotics would you use to cover these organisms?

What is the likely nature of this injury?

There is a 'fight-bite' puncture wound over the right ring finger MCP joint that may have been caused by a penetrating human tooth and may extend into the joint causing cartilage injury, bony fracture, and associated joint infection ± osteomyelitis.

How would you assess this patient?

I would take a full history including the circumstances of the injury, past medical history, and tetanus immunization status. I would look for systemic signs such as fever and tachycardia. On local examination, I would look for signs of cellulitis, tendon sheath infection, tendon rupture, and septic arthritis. I would request plain radiographs to exclude the presence of a foreign body and a fracture. I would also request baseline blood tests [full blood count (FBC), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP)].

How would you treat this injury?

My initial treatment would be to provide tetanus prophylaxis if indicated and apply sterile dressing to cover the wound. I would withhold antibiotics, if systemically well, until tissue samples are obtained.

I would take the patient to theatre for urgent debridement under GA with a tourniquet around the arm. During surgery, I would obtain pus swab and tissue samples for histology and microbiological examination. I would extend the wound and look for tendon damage (re-create fist by flexing the MCP joint). If the tendon is ruptured, I would tag the tendon ends and not attempt primary repair. I would inspect the joint. I would then wash the wound with copious amounts of fluid. I would leave the wound open and apply a splint over non-adhesive dressing. I would commence broad-spectrum antibiotics, pending culture results and arrange for a further look after 48 h.

Which organism commonly causes infection with this type of injury?

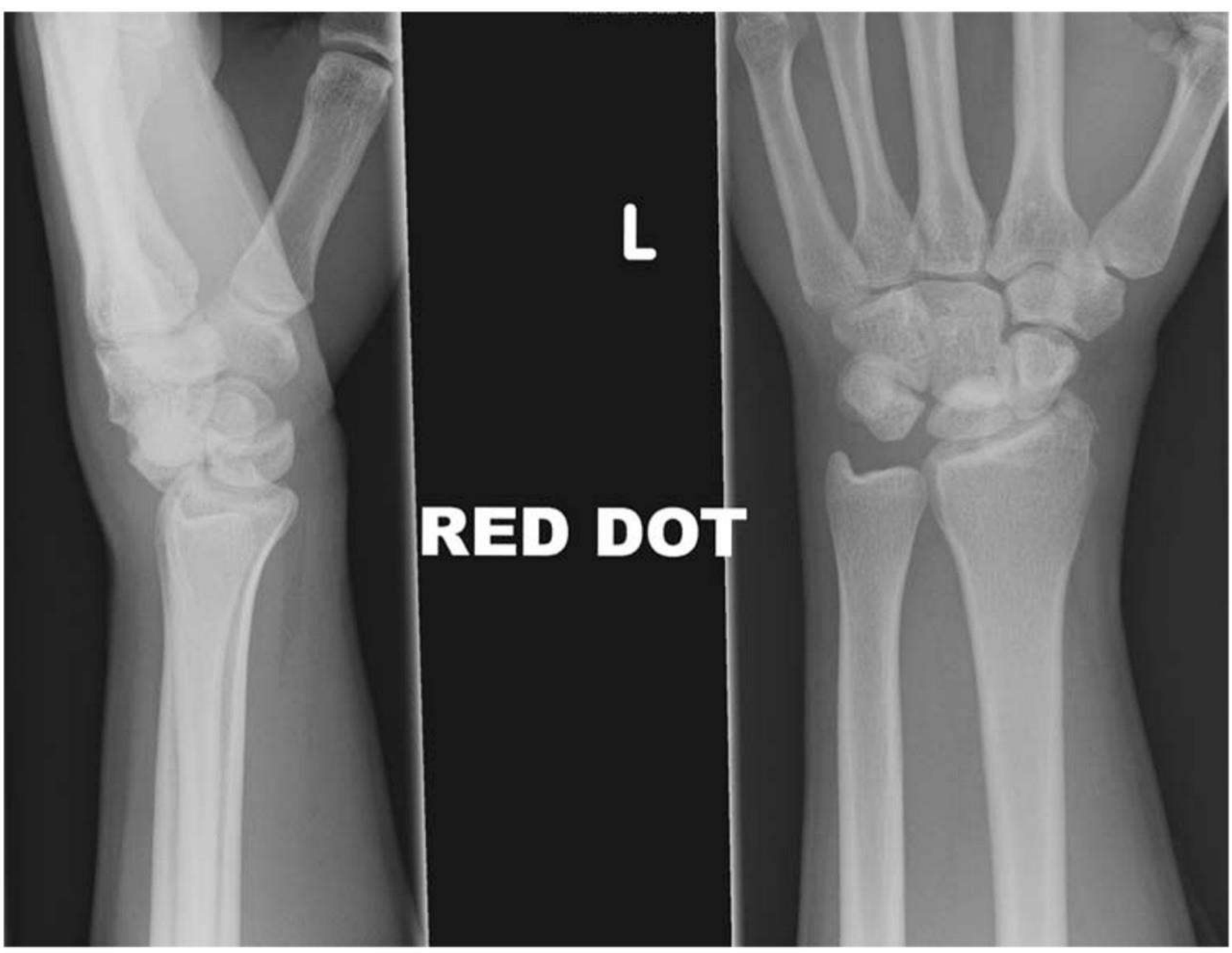
Although *Eikenella corrodens* is peculiar to this injury, *Staphylococcus aureus* is the commonest organism; anaerobic bacteria may also be implicated.

Which antibiotics would you use to cover these organisms?

I would prescribe broad-spectrum antibiotics according to local microbiological protocols such as co-amoxiclav, cephalosporin, and metronidazole.

Viva 5

A 24-year-old male cyclist has been knocked off his bicycle sustaining an isolated injury to his left wrist.



Describe the appearances of these radiographs.

How would you classify this injury?

How would you assess this patient's isolated injury?

How would you manage this injury initially and definitively?

Describe the appearances of these radiographs

Postero-anterior (PA) and lateral radiographs of the left wrist showing a perilunate dislocation.

PA view shows:

1. Disruption of the 'Gilula's' smooth carpal lines that join the proximal joint surfaces of the proximal row of carpal bones at the radiocarpal joint and the distal joint surfaces of the proximal row of carpal bones and the proximal joint surfaces of the distal row of carpal bones (at the mid-carpal joint). The Capitate appears to overlap the lunate
2. Hyperflexion of the scaphoid (scaphoid signet ring sign)
3. Abnormal triangular appearance of the lunate, but lunate located in the lunate fossa of the radius
4. Overlapping of the lunate and triquetrum [unable to visualize lunotriquetral (LT) joint]
5. No obvious fractures of radial styloid, scaphiod, capitate, triquetrum, hamate, or ulnar styloid.

Lateral view shows:

1. Dorsal dislocation of the capitate head from its articulation with the lunate at the mid carpal joint and dorsal translation of distal carpal row and metacarpals relative to the long axis of the radius.

How would you classify this injury?

Perilunate injuries often follow a typical pattern as described by Mayfield. Assuming there are indeed no fractures, this is a 'lesser arc' ligament-rupturing perilunate dislocation. 'Greater arc' injuries also include one or more fractures, typically of the radial styloid, scaphoid, capitate, hamate, triquetrum (± ulnar styloid). A typical lesser arc perilunate injury follows the 'Mayfield sequence' of ligament failures in sequential defined stages:

Check:

Stage I: failure of the radiocarpal ligament
Stage II: failure of the scapholunate ligament
Stage III: failure of the LT ligament and dorsal mid-carpal dislocation
Stage IV: palmar dislocation of lunate at the radiocarpal joint

Therefore this patient's injury is a Mayfield stage III lesser arc perilunate dislocation.

How would you assess this patient's isolated injury?

I would take a detailed history including handedness, occupation, mechanism of injury, co-morbidities, and time since last meal. I would examine carefully for abnormal wrist contour, pain and swelling, and signs of median nerve compression, and document median nerve function, sensory, and motor function.

How would you manage this injury initially and definitively?

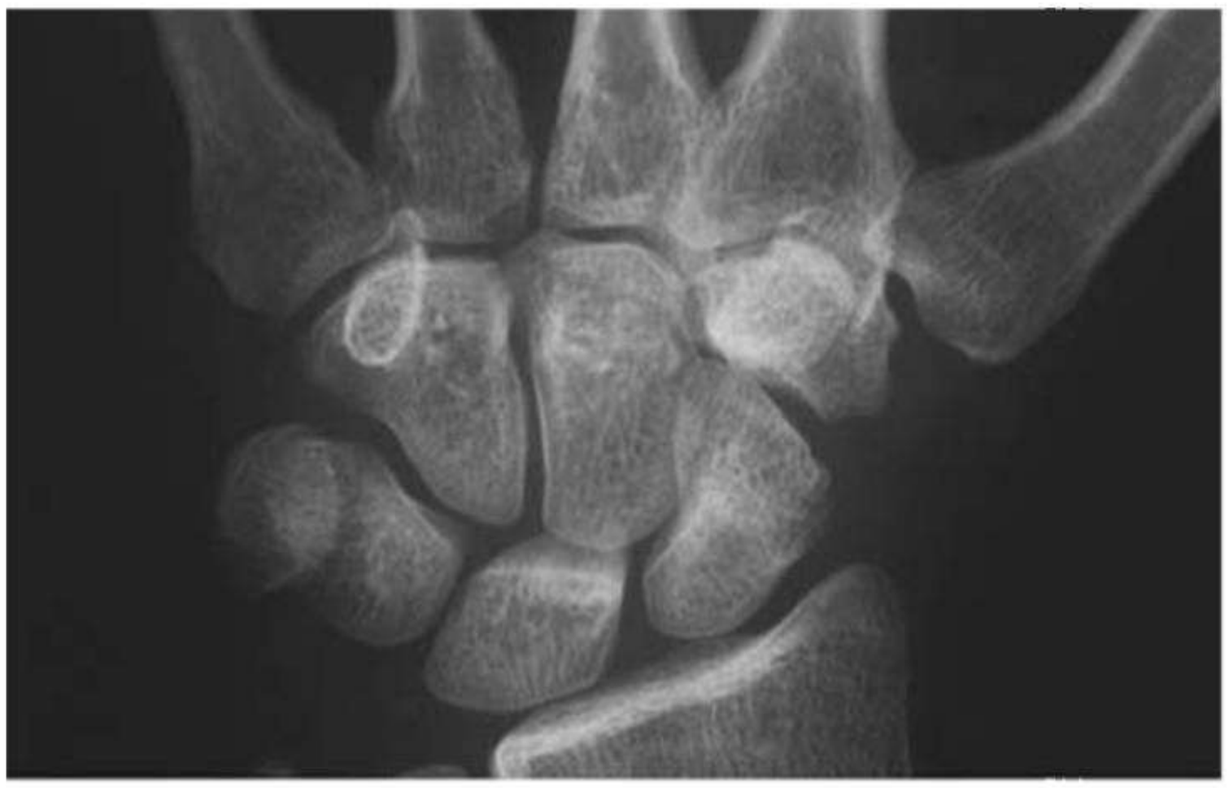
Initial management

- Exclude another injury. Provide analgesia. Regular neurovascular observations
- Keep nil-by-mouth [± intravenous (IV) hydration if necessary]
- Splintage [e.g. padded plaster of Paris (POP) slap + loose bandage]
- High elevation (Bradford sling or Chinese finger traps)
- Explain severity of injury to patient
- Prepare and consent patient for urgent theatre
- Minimum initial intervention requires closed (± open) reduction of the dislocation using image intensifier control (± carpal tunnel decompression) + POP slab stabilization

Definitive intervention (± specialist hand surgery advice)

- Would include closed but anatomical restoration of carpal alignment using joystick k-wires (arthroscopically and image intensifier controlled) + buried k-wire stabilization of scapholunate, LT, and mid-carpal joints
- OR open dorsal anatomical carpal reduction, buried k-wire stabilization, and repair of scapholunate, LT, dorsal and palmar radiocarpal, ligaments
- Post-operatively: high elevation and careful neurovascular observation. Full POP for 2 weeks. Removal of wires at 8 weeks and mobilization
- Risks of post-traumatic carpal instability or stiffness ± osteoarthritis

Viva 6



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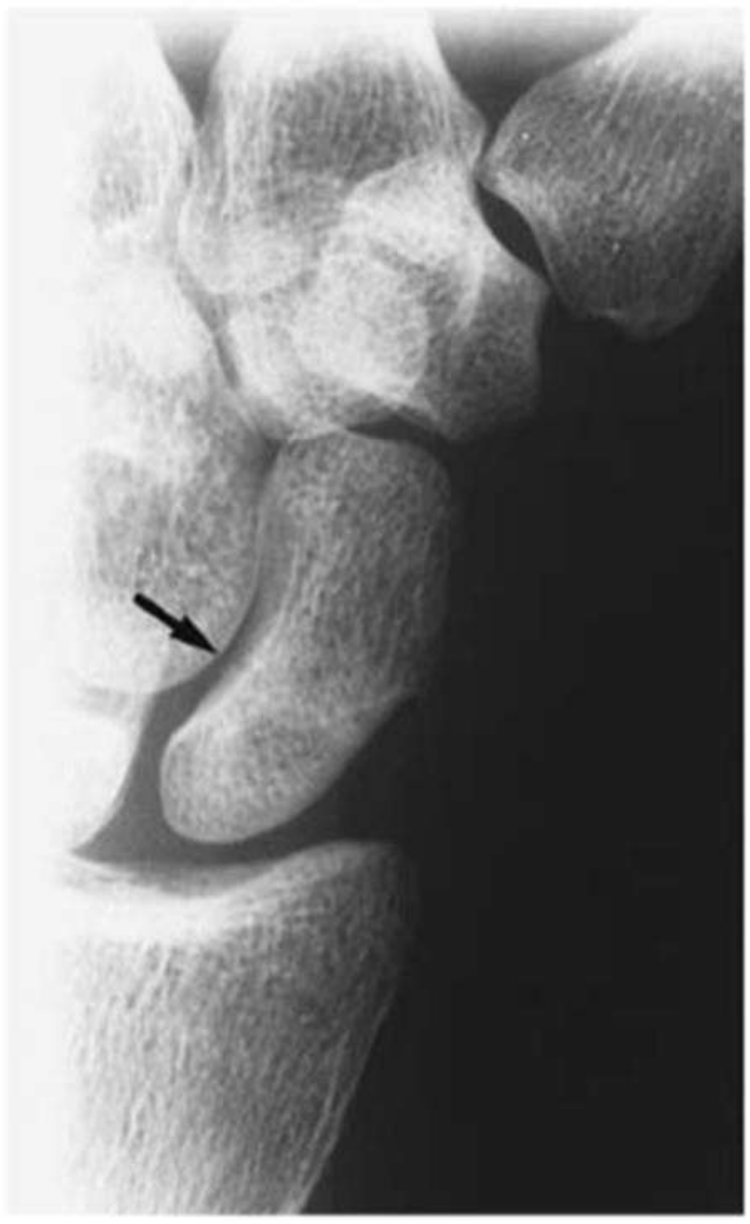


B

Reproduced from C. Bulstrode et al., Oxford Textbook of Trauma and Orthopaedics second edition, 2011, figure 1.10.2, p. 75, with permission from Oxford University Press.

Describe these radiographs and explain the diagnosis.

What are the indications for internal fixation of scaphoid fractures?

Should acute non-displaced fractures be fixed?

What are the complications of this injury?

How would you plan the management of an established non-union of a scaphoid fracture?

Describe these radiographs and explain the diagnosis

The standard PA radiograph of the wrist does not show any obvious fractures; however, there is a subtle, non-displaced fracture of the scaphoid visible on the scaphoid view. This view is obtained by putting the hand and wrist in ulnar deviation, along with 15° of cephalad angulation of the X-ray tube.

What are the indications for internal fixation of scaphoid fractures?

Indications for internal fixation of a scaphoid fracture are:

1. If the displacement is more than 1 mm
2. Or the scapholunate angle > 60°
3. Lunocapitate angle >15°
4. Intrascaphoid angle > 20° (dorsal humpback)
5. Proximal pole fractures, fractures associated with a peri-lunate dislocation
6. Delayed union

Should acute non-displaced fractures be fixed?

The overall rate of non-union scaphoid fractures treated in POP is 10%.

Not all acute non-displaced fractures need fixation, although there are some advantages. Studies have shown better early outcome scores, grip strength, and range of motion (ROM) with fixation but no difference after 12–16 weeks. The rate of delayed union has been shown to be less with early fixation. Patients should be advised to avoid cigarette smoking to optimize their potential for bone healing.

What are the complications of this injury?

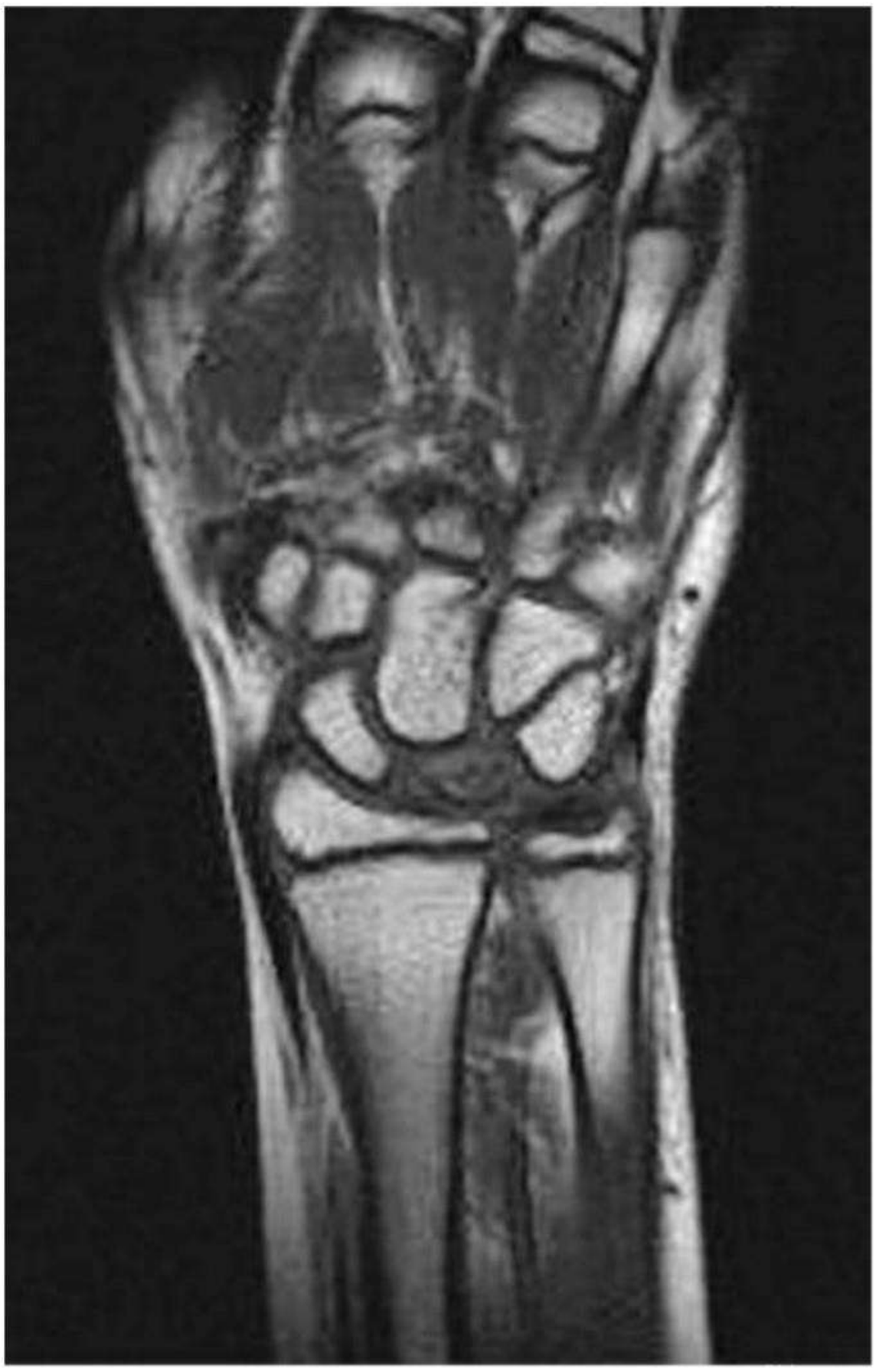
The two major complications related to this injury are avascular necrosis (AVN)of the proximal pole and non-union.

How would you plan the management of an established non union of a scaphoid fracture?

If arthritic changes are not present on the radiographs, fixation with bone graft should be attempted in an effort to get the fracture to unite. Zaidemberg and colleagues have reported excellent (100% union in 11 patients) results with a vascularized distal radial bone graft based on the 1,2 intermeta-carpal branch of the radial artery. More recent studies have reported a success rate of around 70%. If arthritic changes are present and patient is symptomatic, salvage procedures such as radial styloid-ectomy, proximal row carpectomy, scaphoid excision, and four-corner fusion and arthrodesis of the wrist should be considered.

Viva 7

This adolescent man comes to your clinic complaining of non-specific wrist pain and a magnetic resonance imaging (MRI) scan he has obtained privately.



Reproduced from C. Bulstrode et al., Oxford Textbook of Trauma and Orthopaedics second edition, 2011, figure 13.23.7, p. 1617, with permission from Oxford University Press.

Describe what you see in the image.

What is the cause of this condition?

What is the staging system for this condition?

What else should you look for on the radiographs?

What are the management options?

Describe what you see in the image

This T1 magnetic resonance image shows low signal density in the lunate, suggestive of Keinbock's disease.

What is the cause of this condition?

Kienbock's disease is AVN of the lunate bone in the wrist.

What is the staging system for this condition?

The Lichtmann classification, which recognizes four stages:

Stage I: normal radiographs, possible stress fractures
Stage II: sclerosis of the lunate, no collapse
Stage IIIA: fragmentation and early collapse
Stage IIIB: IIIA + scapholanate dissociation and fixed rotation of the scaphoid
Stage IV: IIIB + degenerative changes in the wrist joint

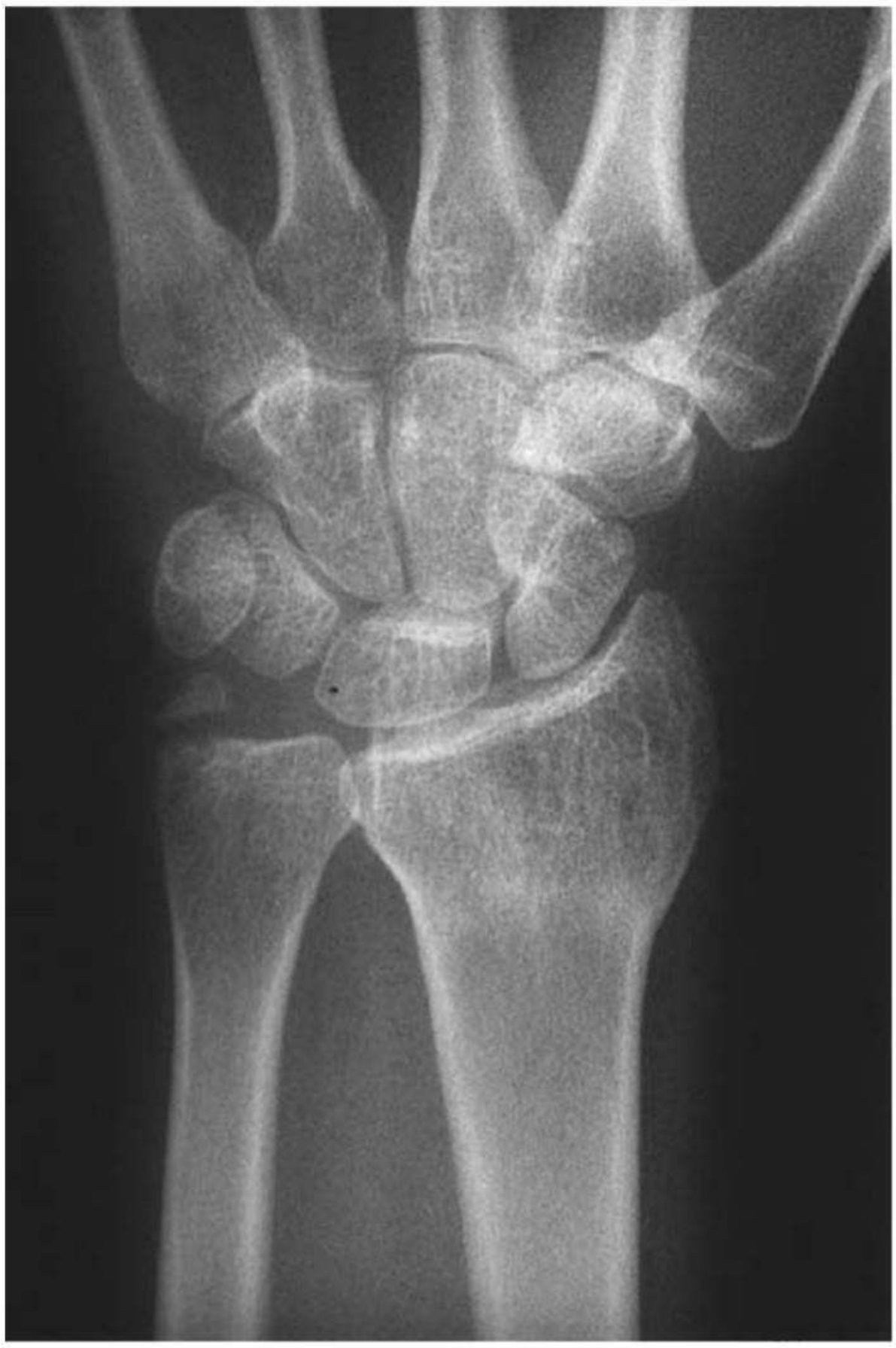
What else should you look for on the radiographs?

I would look for negative ulnar variance on antero-posterior (AP) radiographs taken with the forearm in mid-prone position.

What are the management options?

The condition can be managed non-operatively with analgesia and splintage. Operative options include joint levelling procedures (radius shortening), wrist denervation, partial or total wrist fusion, and proximal row carpectomy. Choice of treatment depends on the stage of the disease, degree of symptoms, and patient factors.

Viva 8



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Describe what you see on this radiograph of a 22-year-old with ulnar-sided wrist pain.

Which soft tissue structure would you expect to be involved?

Can you simplify the anatomy of this complex structure?

What are the management options for this condition?

Describe what you see on this radiograph of a 22-year-old with ulnar-sided wrist pain

This AP radiograph of the wrist shows ulnar positive variance. This appearance is typical of ulnar abutment syndrome.

Which soft tissue structure would you expect to be involved?

Triangular fibrocartilage complex (TFCC) tears are frequently associated with this condition (Class 2 lesion).

Can you simplify the anatomy of this complex structure?

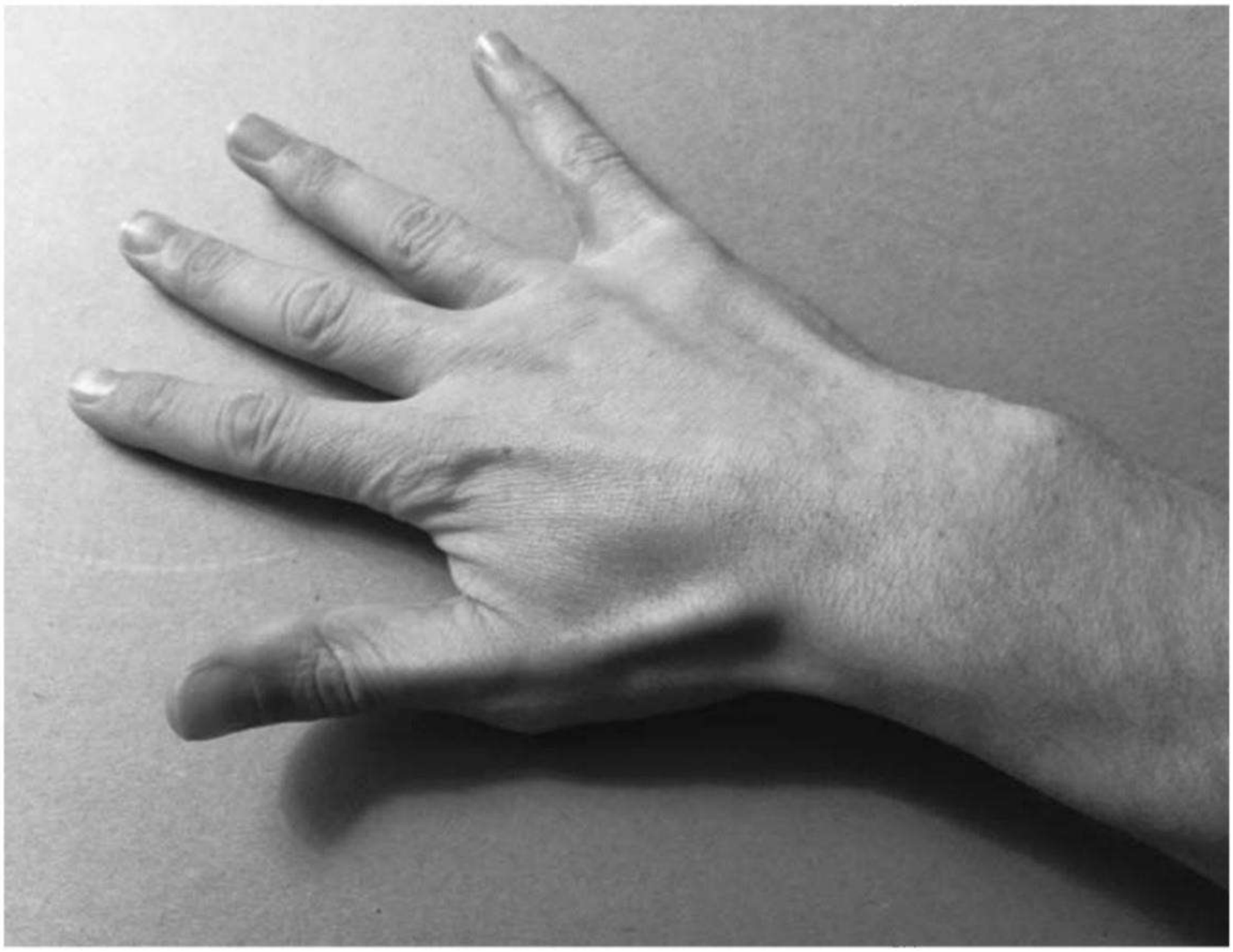
The TFCC is a pyramid-shaped fibrocartilagenous ligamentous structure found at the distal aspect of the ulna.

It comprises a fibrocartilagenous disc (a meniscus-like structure) and a sling of ligaments and acts as a key stabilizer of the distal radioulnar (DRU) joint and the ulnocarpal joint.

What are the management options for this condition?

Non-operative measures are splint, analgesia, and avoidance of aggravating activities. Operative options include arthroscopic wafer resection of ulna, or open ulnar shortening osteotomy.

Viva 9



Tell me about the dorsal compartments at the wrist joint.

What is de Quervain's syndrome?

What are the clinical signs of de Quervain's syndrome?

What are the management options for de Quervain's syndrome?

What are the adverse effects of local steroid injection?

What are the pitfalls of surgery?

Tell me about the dorsal compartments at the wrist joint

There are six compartments in which the extensor tendons traverse the dorsum of the wrist

1. APL, EPB (abductor pollicis longus, extensor pollicis brevis)
2. ECRL, ECRB (extensor carpi radialis longus, extensor carpi radialis brevis)
3. EPL (extensor pollicis longus)
4. EI, EDC (extensor indicis, extensor digitorum communis)
5. EDM (extensor digiti minimi)
6. ECU (extensor carpi ulnaris)

What is de Quervain's syndrome?

It is a painful condition affecting the first compartment tendons of the wrist joint. It is commoner in females, especially post-partum.

What are the clinical signs of de Quervain's syndrome?

There is localized tenderness and/or swelling along the radial aspect of the wrist over APL/EPB. The Finkelstein test is considered positive if pain is elicited on holding the thumb and quickly ulnar-deviating the wrist. Pain may also be elicited on ulnar-deviating the wrist with fingers flexed over the thumb held in the palm.

What are the management options for de Quervain's syndrome?

Non-operative options are splintage, analgesia, and local steroid injection. If non-operative measures fail, surgical release could be considered. The procedure is done under GA or regional anaesthetic and with a upper arm tourniquet. Release may be achieved thorough a longitudinal or transverse skin incision.

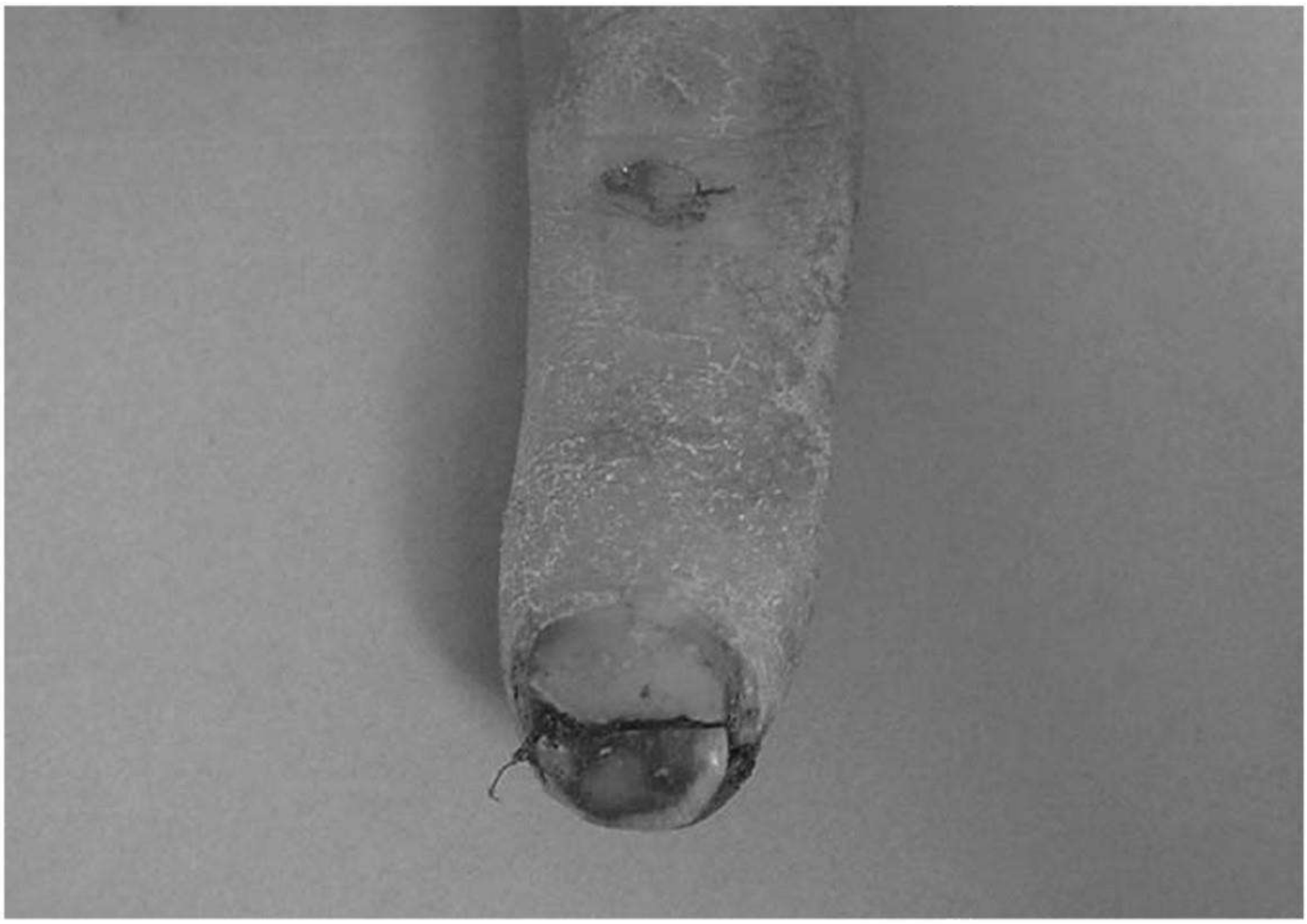
What are the adverse effects of local steroid injection?

Adverse effects of local steroid injection are infection, skin atrophy and depigmentation, subcutaneous fat atrophy at the site of injection, injury to the superficial branch of radial nerve (painful neuroma), and tendon rupture.

What are the pitfalls of surgery?

Failure to recognize anatomical variation (EPB may lie in a separate compartment) may lead to incomplete relief of symptoms. Injury to the sensory branch of the radial nerve could result in a painful neuroma.

Viva 10



Reproduced from C. Bulstrode et al., Oxford Textbook of Trauma and Orthopaedics second edition, 2011, figure 14.7.1, p. 1675, with permission from Oxford University Press.

How would you manage this crush injury?

What would you explain to the patient?

How would you manage this crush injury?

I would take a relevant history, including: handedness; occupation; mechanism of injury; and co-morbidities. I will provide tetanus prophylaxis (if indicated) and antiseptic (betadine) dressing.

I would obtain radiographs to exclude an underlying fracture.

As definitive management, I would explore and repair the nail bed under local anaesthesia (digital block) and digital tourniquet.

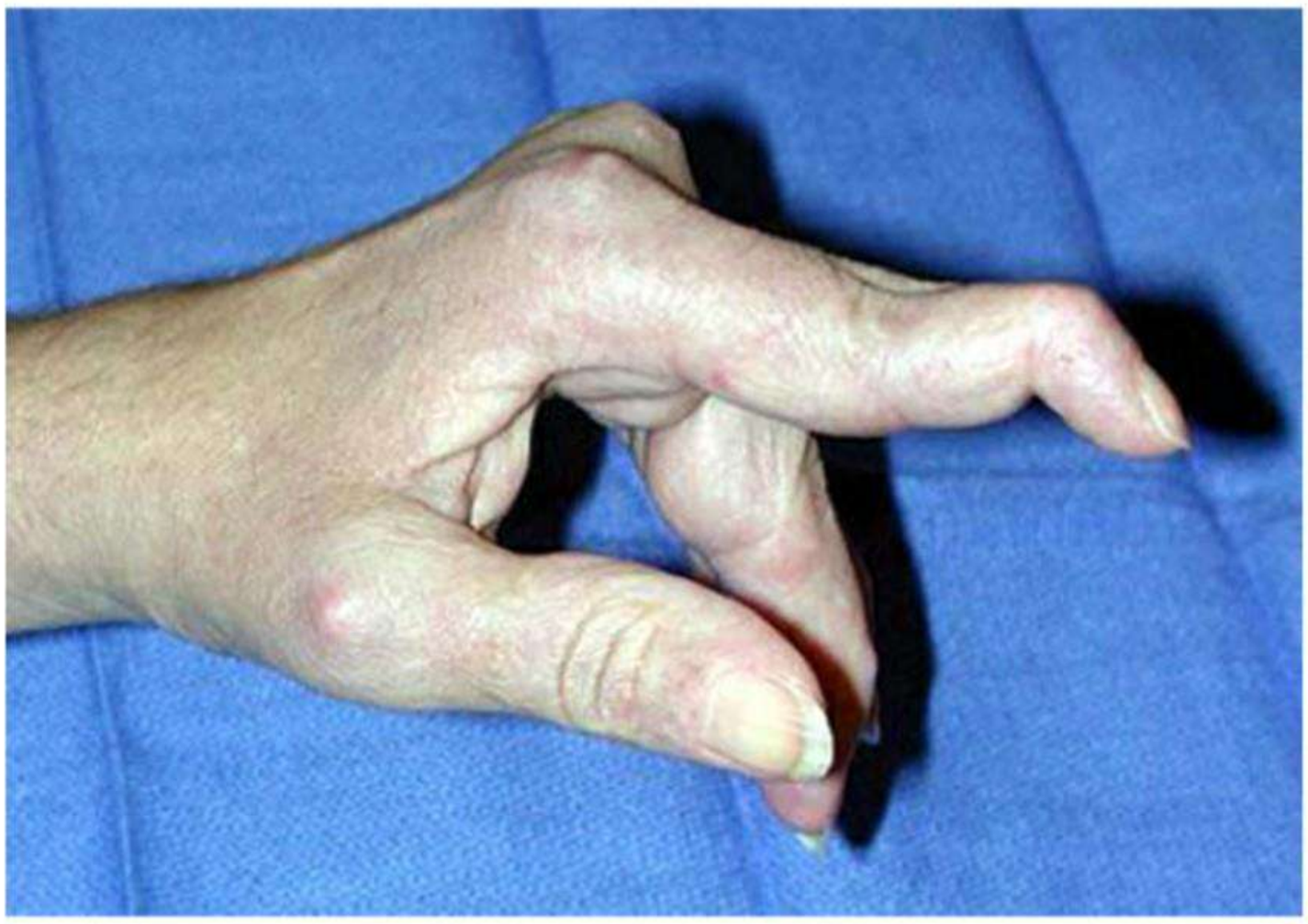
The salient steps of the procedure are:

1. Remove the nail plate carefully
2. Inspect the nail bed and wash thoroughly
3. Copious lavage of any underlying fracture
4. Reduce fracture if present and stabilize (axial k-wire; remove after 3–4 weeks) if necessary
5. Repair nail bed with a 6-0 absorbable suture (VICRYL Rapide)
6. 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.

1. Clinical picture of a swan neck deformity.
Describe the deformity, the underlying pathology and its classification.
How will you manage this patient?



Describe:
- Hyperextension at the PIPJ
- Flexion of the DIPJ

Pathoanatomy:
One of the common primary lesions is a lax volar plate, allowing producing hyperextension at the PIPJ from
- Trauma
- Rheumatoid arthritis
- Generalised joint laxity

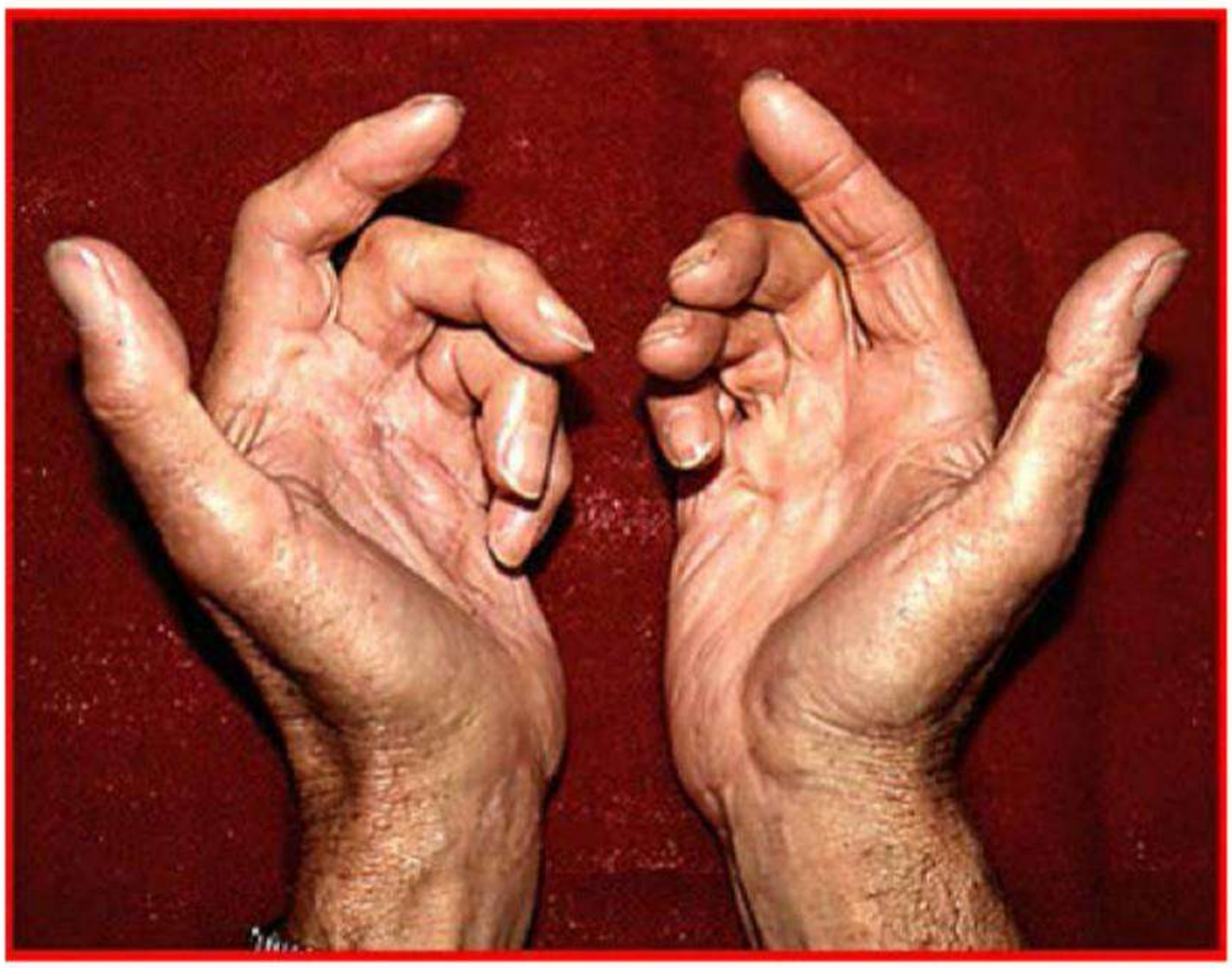
Secondary lesions arise from an imbalance of forces at the PIPJ, from
- volar subluxation of the MCPJ
- Laceration or transfer of FDS (unopposed extension at PIPJ)
- Intrinsic contracture (excessive extensor forces through the PIPJ), assess with Bunnell test
- Mallet finger (extension of DIPJ extension force to the PIPJ)

Classification (Nalebuff) and Treatment:

Type	Description	Treatment options
I	PIPJ completely flexible	Splinting with double ring splints DIPJ fusion if mallet deformity significant Consider spiral oblique retinacular ligament reconstruction (SORL)
II	PIPJ flexion limited in certain positions	Lateral band translocation (Littler) Intrinsic release if necessary +/- MCPJ
	(tight intrinsics)	reconstruction

2. Clinical picture of a hand of a patient with Dupuytren's. The ring and little fingers are involved

D ne deformity: what is the diagnosis?
What are the associations?
Name the different types of cord.
How would you manage this patient, and which incision(s) would you use?



Clinical picture of Dupuytren's:

- Describe deformities, bands and cords

Epidemiology/associations:

- Northern European and Celtic descent (likely autosomal dominant with variable penetrance)
- Diabetes mellitus
- Epilepsy: controversial
- Alcoholic/smoker (controversial)
- Hypercholesterolemia
- HIV

Aetiological theories and associations:

- Genetic- increased incidence in relatives
- Traumatic: micro trauma
- Neoplastic
- Inflammatory

Associations:

- Ledderhose disease (plantar fascia, 5% of patients)
- Peyronie's disease (Dartos fascia of the penis, 3% of patients)
- Garrod's disease (knuckle pads)
- Dupuytren's diathesis relates to certain features of Dupuytren's disease and indicates an aggressive form. These factors are patient aged below 50 years, positive family history, bilateral disease, and ectopic lesions.

- Fascial involvement (bands) become pathologic cords
- Myofibroblast is the offending cell
- 4 of 27 levels of cytokines- IL/TGF/PDGF/FGF etc seen

Cords:
- Pre-tendinous cord: flexes MCPJ
- Central cord
- Lateral cord
- Spiral cord: from pre-tendinous band/spiral band/lateral digital sheet/Greyson's ligament (pass deep to NV bundle- chance of injury during surgery, it displaces the neurovascular bundle)
- Natatory cord: cause web space contractures
- Abductor digiti minimi cord
- Commisural cords: dorsal/palmar- 1st web contractures

Clinical features:
- Cords – MCP flexed by pretendinous cord, PIP by central, spiral and lat cord
- Pits
- Nodules
- Garrod's pads dorsally
- MC in ring and little fingers
- Inability to lay hand flat on a table i.e. failure of Huestons table top test.

Management:
Non-operative:
- Observe if good function or minimal contracture
- Nice Guidelines from 2010 have described radiotherapy but this is not a common treatme... method

Operative:
Indications:
- Reduced function
- MCP >30 degrees
- PIP >15 degrees

Treatment of choice:
Partial fasciectomy
- 15% recurrence
- PIP not as successful as MCP
- Can result in residual contracture or fixed flexion deformity
- If >70 degree – very unlikely to achive full correction, and may need variable releases
- Incisions: Brunner, longitudinal with a Z-plasty

- Wounds in the palm can be left open: McCash open palm technique

Complications:

- Wounds in the palm can be left open: McCash open palm technique

Complications:

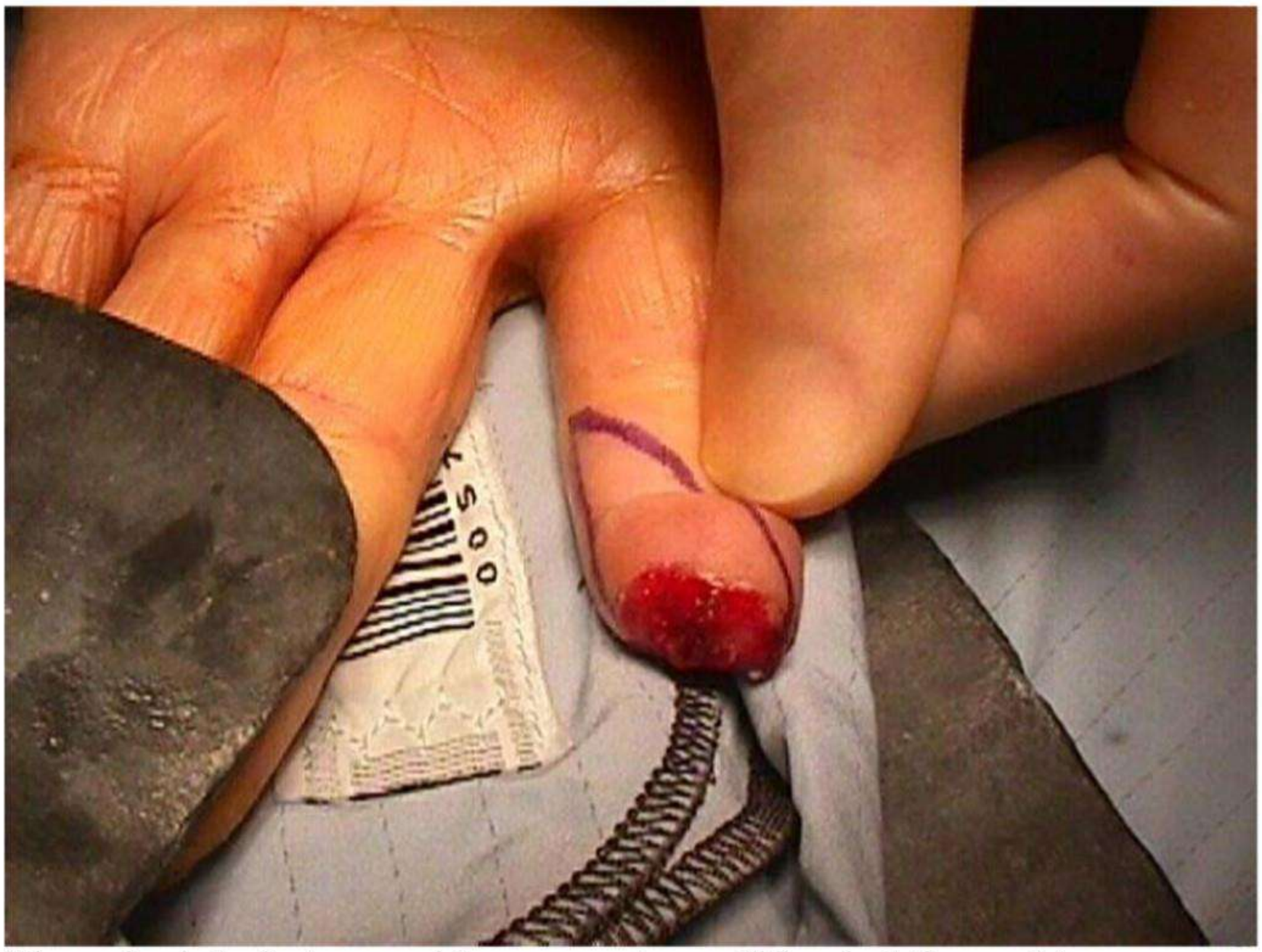
- Wounds in the palm can be left open: McCash open palm technique

Complications:
- Infection
- Delayed wound healing
- Incomplete correction
- Recurrence (30-50% at 10 years, (Bulstrode et al., 2005, Tonkin 1984)
- Neurovasular injury
- CRPS
- Amputation

Postoperative Management:
- Carefully applied bulky dressing providing compression
- Elevation
- Some advocate avoiding finger extension immediately from excessive tension on the wound
- Early wound check and initiation of active range of movement exercises
- May need extension splint at night
- Follow up for recurrence or involvement of other digits

Other procedures:
- Collagenase injections - sited directly into the cord and manipulation takes place the following day
- Percutaneous fasciotomy - in simple pretendinous palmar cord (45% recur)
- Segmental fasciectomy
- Total fasciectomy - not advocated at present
- Dermofasciectomy + skin graft - if recurrence with skin involvement. The full thickness skin graft is taken from the forearm or the groin (hair free) after excision of the diseased tissue and skin ('fire-break'). Some surgeons advocate this as their main method of treatment.
- McCash open palm technique - the wound is left open and allowed to heal by second intention

5. Clinical picture of finger tip injury.
How will you manage this patient?
Discuss the options for soft tissue flaps.



The principle is to provide a sensate, well healed tip, and to preserve length (allowing bony support for the nail).

Can be divided into:

1) Soft tissue loss without bone exposure:

- Fingertip lacerations or avulsions of the substance smaller than 1 cm^2 will heal by second intention, especially good in children. Some argue that wound contraction will lead to a hook nail deformity, but tight primary closure wound closure (drawing the nail bed over the tip) will lead to the same outcome and must be avoided.
- For wounds extending for more than 1 cm^2, a local flap may be needed to cover the defect

2) Soft tissue loss with bone exposure:

- Exposed bone is not a satisfactory bed for healing. Even if they heal, skin at the tip is poor quality and easily broken down, leading to a chronic ulcer.
- If bone is exposed, the prominence must be shortened with excision back to rounded edges and primary, tension free, soft tissue closure performed. Bear in mind the need for bony support of the nail bed.
- If excessive bony debridement is required to achieve closure, soft tissue coverage is indicated.

Soft Tissue Coverage Choices:

Local flap coverage:

- Advancement flap from the same finger has the advantages of rapid healing, maintenance of sensation and limited morbidity.

- The aim is to free the skin from the septae anchoring it to bone, whilst retaining the nerves and

Soft Tissue Coverage Choices:
Local flap coverage:
- Advancement flap from the same finger has the advantages of rapid healing, maintenance of sensation and limited morbidity.

- The aim is to free the skin from the septae anchoring it to bone, whilst retaining the nerves and vessels, allowing a tension free advancement.

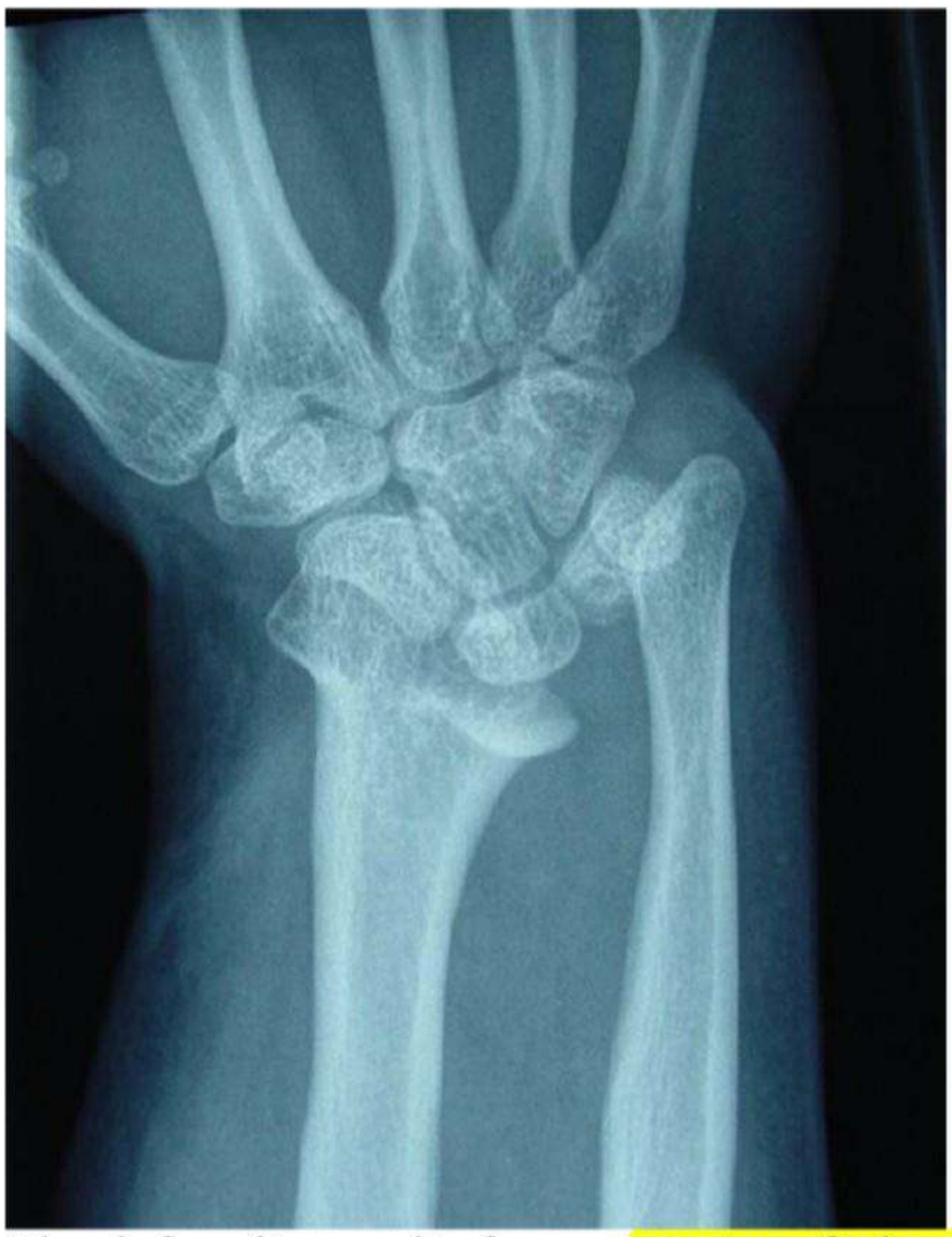
1) Options for straight or more dorsal tissue loss:
- Volar V-Y advancement (Atasoy)
- Double lateral V-Y advancement flap (Kutler)

2) Options for more volar tissue loss:
- Cross finger flap, but scars the adjacent finger
- A rectangular flap is raised from the three sides of the dorsal surface of the middle phalanx of the adjacent digit.
- The flap is normally based on the lateral side of the adjacent digit and swung on its pedicle and sutured in place.
- The flap is divided within two weeks to avoid stiffness of the fingers.
- Thenar flaps can be considered, are well tolerated in children, but can lead to PIP joint stiffness in adults.

3) There are more complex advancement flaps but these should be performed by experienced surgeons

Terminalisation:
- Indicated for a severely crushed distal phalanx
- The level is through the DIPJ, trimming the condyles of the middle phalanx
- Aim to preserve the FDS and achieve tension free primary closure
- Digital nerves are transected as proximally as possible
- A volar skin flap is created and the wound is closed dorsally

6. Plain radiographs of Madelung deformity of the distal radius.
What is the diagnosis, and how does the condition arise?
D e management of this condition.



The deformity results from premature fusion of the ulnar and volar aspect of the distal radial epiphysis. As the child grows, the distal radius develops increased radial inclination and volar tilt, and ulnar-carpal impaction.
Presents more frequent in females, those with a positive family history and is often bilateral.

Causes:
- Usually idiopathic
- Congenital dysvhondrosis
- Post traumatic
- Dysplastic (Ollier's disease, achondroplasia, multiple epiphyseal dysplasias)
- Genetic (association with Turner's syndrome and Leri-Weill dyschondrosteosis).

Clinical manifestations:
- Insidious onset of wrist pain (radioulnar impaction)
- Development of prominence of the dorsal ulnar head
- Bowing of the distal radius
- Limitation of forearm rotation.

Management:
- Determined by the degree of deformity, the degree of closure of the growth plate (? progressive) and the severity of the symptoms.

Nonoperative management:

Nonoperative management:
Monitor patients without pain

Operative management:
Aims to either correct deformity or relieve pain and improve function (or both):

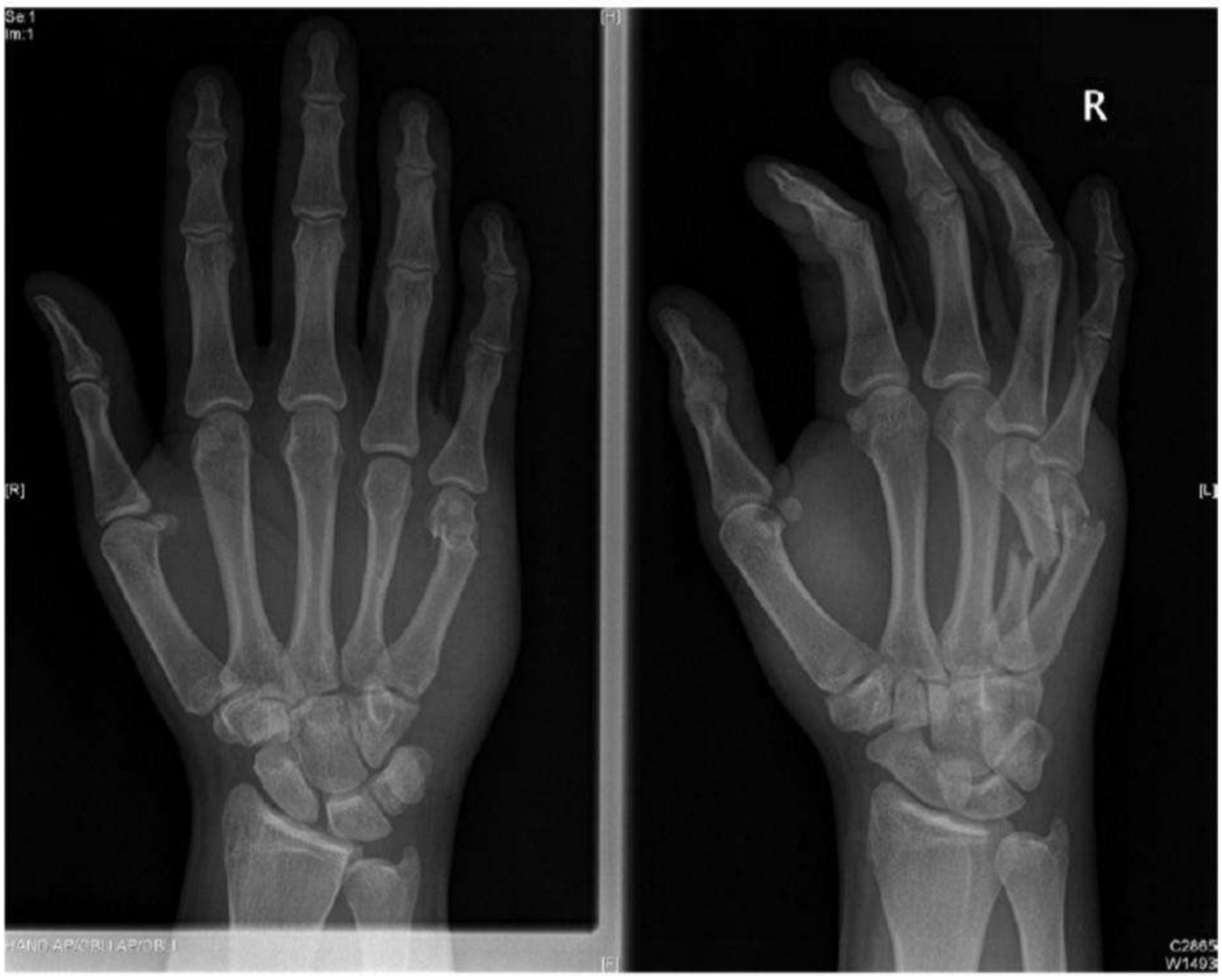
- Prevent further deformity if the growth plate is open, with epiphysiodesis, and a closing wedge osteotomy to correct the deformity
- Release of the Vickers Ligament, a tethering structure
- Include ulnar shortening if it is long and sypmtomatic and distal ulnar epiphysiodesis if the growth plate is open
- Alternatively one can include radial lengthening.

- For symptomatic relief, a Sauve-Kapandji procedure will decrease ulnar sided wrist pain and increase range of movement, or consider Darrach's procedure (though risks instability).

- For adults with severe pain and instability, a wrist arthrodesis is suitable.

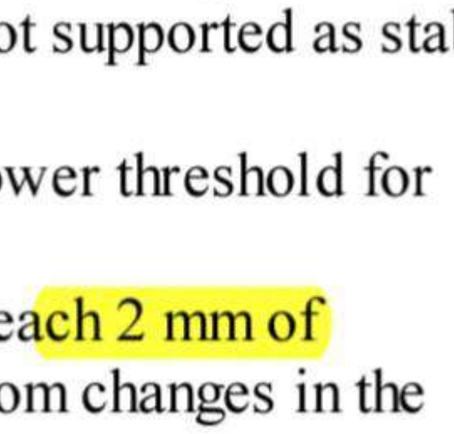
7. Plain radiographs of fractures of the 4th and 5th metacarpal shafts.

7. Plain radiographs of fractures of the 4th and 5th metacarpal shafts.
What is the management of these fractures?
H u undertake open reduction and internal fixation?
What is your approach?



- The displacement of metacarpal fractures is normally reduced by the intermetacaral ligaments, the intrinsic muscles and the adjacent metacarpal, but the border metacarpals are not supported as stably as the middle and ring metacarpals.
- This anatomical splinting is lost in this fracture pattern; therefore, there is a lower threshold for fracture fixation.
- In addition, with shortening of the metacarpals comes loss of extension (with each 2 mm of shortening there is a 7 degree extensor lag at the MCPJ (Strauch et al., 1998) from changes in the relationship between flexors and extensors.

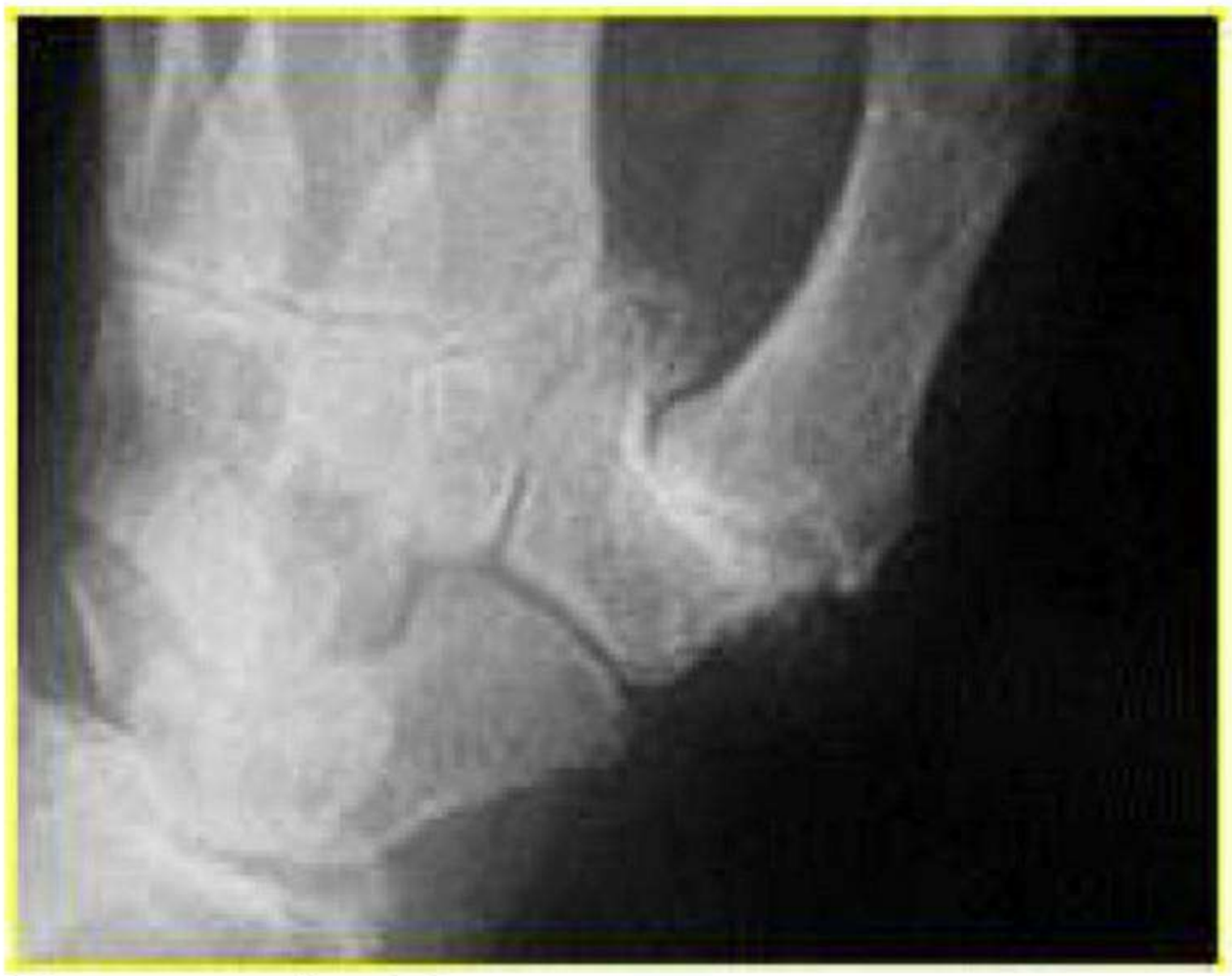
Ensure:
- There are no associated soft tissue injuries (fight bite) that need to be addressed
- The soft tissues overlying the fractures are intact
- Any rotational deformity is noted preoperatively
- There is no dislocation of the 4th or 5th CMCJs, clinically or radiologically
- There is no other associated phalangeal fracture



Management:
Open reduction and internal fixation with dorsal plating is the method of choice:
- Dorsal longitudinal incision between 4th and 5th metacarpals
- Soft tissue dissection; avoid the dorsal cutaneous branch of the ulnar nerve, then through the juncturae tendinum interconnecting the common extensor tendons.

- Retract the tendons over either fracture

9. Plain radiographs of osteoarthritis of the CMC joint.
What is the management of this condition?
What is the evidence for excision vs arthrodesis vs arthroplasty?



Non-operative Management:
- For mild symptoms this is the first line treatment
- Splints can be used (thumb spica)
- NSAIDS
- Activity modification
- Injections – Heyworth (2008) showed in a prospective, randomised, double blinded trial that steroid, hylan and saline were all equally effective at reducing pain and increasing thumb function at three months

Operative Management:
Trapeziectomy +/- further procedure:

-Trapeziectomy (excision arthroplasty)
Concerns with thumb weakness from shortening
Modifications to this technique were developed:
-Trapeziectomy with palmaris longus interposition (interposition arthroplasty)
-Trapeziectomy with ligament reconstruction and tendon interposition (50% FCR) (LRTI). The aim is to support the base of the first metacarpal and prevent thumb shortening.

The evidence:
- Davis et al. (2004) radomised 162 women with 183 procedures over 9 years with Eaton Grade II to IV to one of the above three procedures.
- Three months postoperatively, pain had significantly improved in all groups, with further improvement by one year, with no difference between groups.
- No difference in thumb-key and tip-pinch strength or grip strength between groups
- However, all patients were immobilised with a K-wire for four weeks, which is not standard practice, and may have increased the stability of the pseudarthrosis in the trapeziectomy alone group.
- Longer term outcomes may be different.

Arthrodesis:

11. Plain radiograph of mallet finger.
What is the management of this injury?

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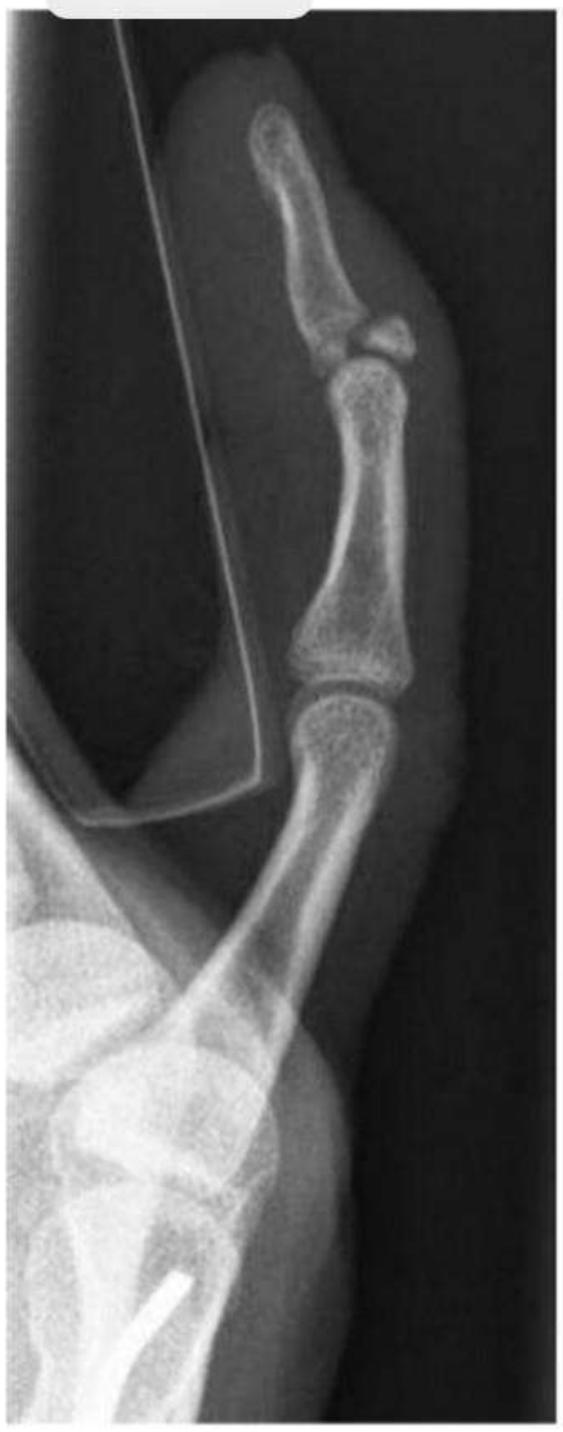


-The mallet finger deformity is caused by loss of extension at the DIPJ.
- The mechanism of injury is forced flexion of the extended DIPJ and is often a sporting injury (netball, basketball)
-The disruption to the extensor mechanism may be tendinous or bony

Tendinous:
- The vast majority of acute injuries (<12 weeks) should be treated conservatively
- The DIPJ is splinted continuously for six to eight weeks, but allow PIPJ movement
- An off the shelf Stack splint can be used, but be wary of dorsal skin maceration (a short Zimmer splint is an alternative)
- Strict instructions should be given for how to clean the finger out of the splint without flexion.
- Begin progressive flexion exercises at 6 weeks
- Usually maintain night splintage for another six weeks
- Surgery can lead to a loss of flexion and is avoided if possible
- An extensor lag after treatment is common, with around 30-40% of patients regaining full flexion

Bony Mallet:
The mechanism of the injury is a bony avulsion at the insertion of the extensor tendon.

Non operative management:
- As per tendinous mallet
- If there is displacement, get a plain radiographin a mallet splint to observe if it reduces.

- Check for fracture displacement at one week post injury with a good quality lateral radiographs.

Non operative management:
- As per tendinous mallet
- If there is displacement, get a plain radiographin a mallet splint to observe if it reduces.

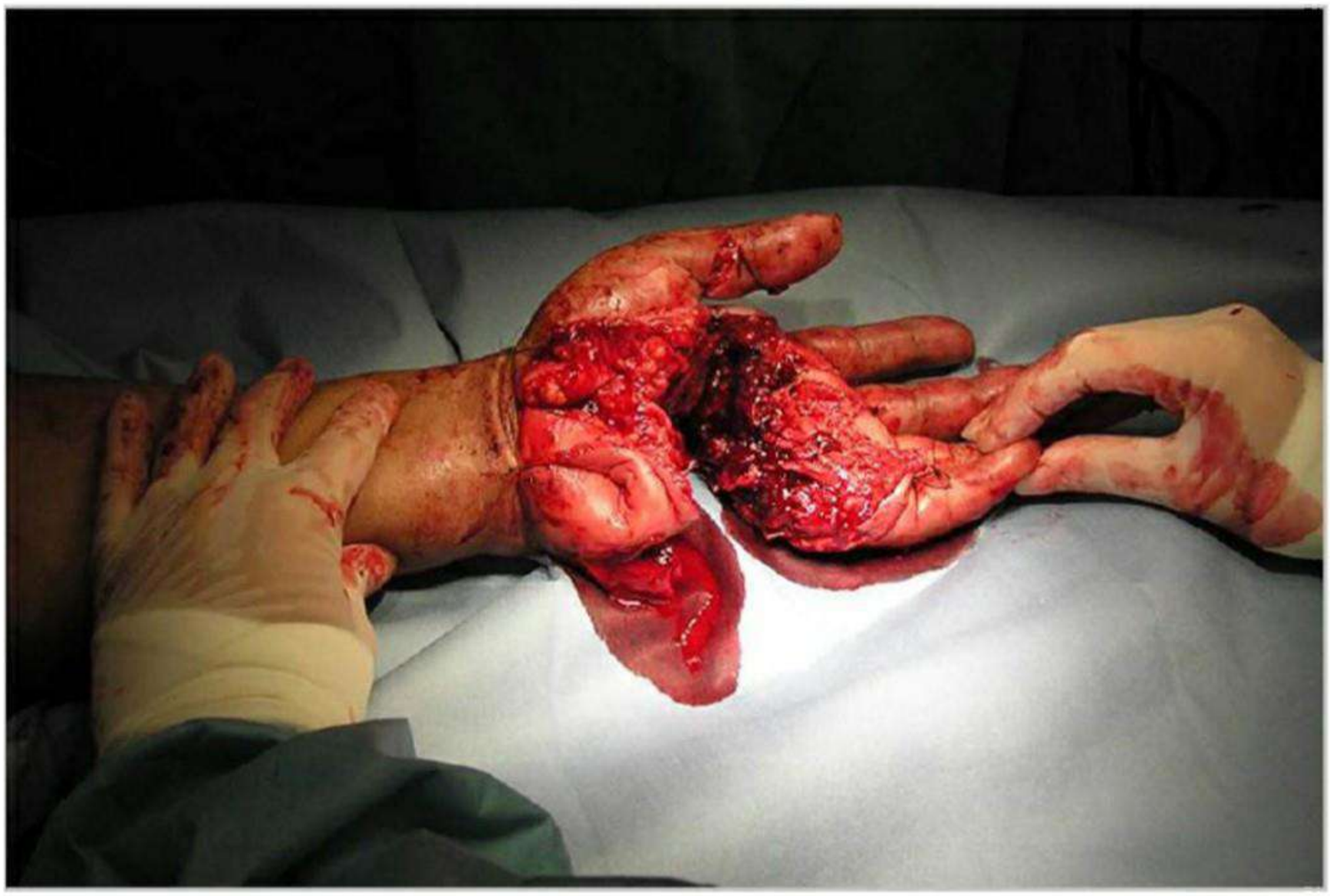
- Check for fracture displacement at one week post injury with a good quality lateral radiographs.

Operative management:
- Is reserved for subluxed DIPJs or large or displaced bony fragments
- Absolute indication – volar subluxation of the distal phalanx
- Relative indications – the bony fragment >40% of the articular surface, or there is >2 mm displacement

Techniques:
- For subluxation include reducing and holding with a K-wire across the joint for 4 weeks.
- The bony fragment can be difficult to control given its size. Fixation methods include a single screw, or a dorsal blocking K-wire

Chronic patients:
- Look for an associated Swan-neck deformity
- Treatment in a mallet splint for 6 weeks, or reconstruction of the terminal tendon (tendon advancement or Fowler central slip tenotomy if Swan neck deformity is present).
- Consider tenodermodesis (Sorene and Goodwin, 2004)
- Consider DIPJ fusion if the joint is painful, stiff or has post-traumatic OA

12. Clinical picture of a mangled hand.
How will you manage this patient?
What approach will you use and what are your priorities?
What are the principles of management of traumatic amputation at fingers What are the prognostic factors?



Maintain a structured approach:
- Manage the patient according to ATLS guidelines
- May require multidisciplinary approach with support from Plastic/Vascular surgery team

Decision making for any hand injury relies on:

1. Evaluation of the patient – mechanism, past medical history, site and pattern of injury. ATLS protocol must be followed

Priorities include recognition of nerve/vessel/ tendon/muscular injuries

2. Plan treatment and discuss the options with the patient
3. Restore bony anatomy and provide stability
4. Soft tissue care with good debridement +/- reconstruction
5. Early mobilisation

The principles of wound coverage:
Early coverage of traumatic wounds is important:
- Leads to to reduced flap failure rates, reduced effects of fibrosis, easier surgery for flap planning and vascular anastomoses, less vascular spasm, lower infection rate and fewer returns to theatre
- Wounds covered within 6 days had fewer infections than those covered between 6 days and 3 months (0.7% vs 17.5%).

The reconstructive ladder guides management using the simplest effective methods whilst restoring the hand to useful function and reducing morbidity.
- From simple to complex – primary wound closure, secondary wound closure, skin graft, local flap,

regional flap, free flap. The flap treatment options are determined in part by the location of the lesion.

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- From simple to complex – primary wound closure, secondary wound closure, skin graft, local flap,

regional flap, free flap. The flap treatment options are determined in part by the location of the lesion.

Principles of traumatic amputation at the fingers:
- Discuss with local centre - ? for replantation
- Care of the patient: nil by mouth, intravenous antibiotics, adequate analgesia, clean and dress the stump, plain radiographof amputated part
- Care of amputated finger: wrap in saline moistened gauze, place in a sealed plastic bag, and place the bag in a container with ice and normosaline

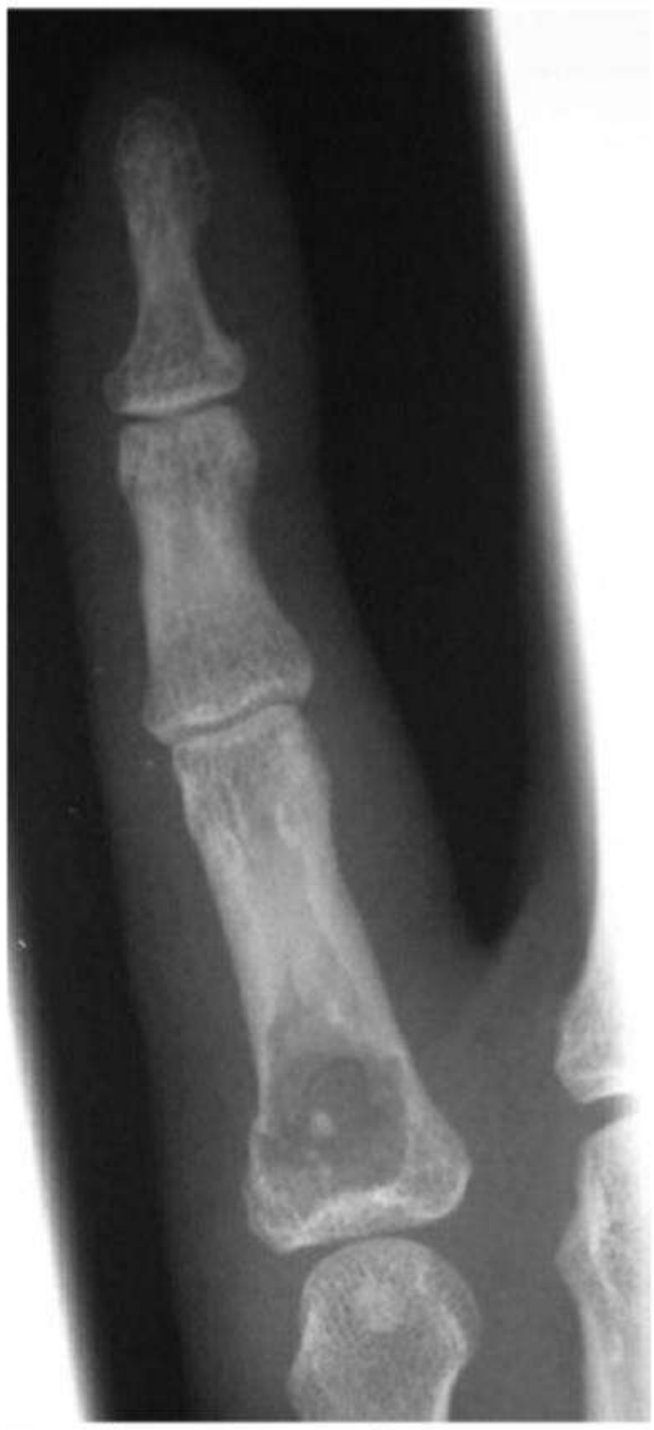
Is the digit(s) suitable for replantation?
Indications for replantation:
- Thumb
- Multiple digits
- Individual digits distal to insertion of FDS
- Children do well with most parts

Contraindications for replantation (Pederson, 2001):
- Single digit Zone II
- Mangled parts
- Multiple levels
- Prologued ischaemic time (for digit cold ischaemic time <24 hours, warm ischaemic time <12 hours).

Complications from replanted digits include infection, cold intolerance and stiffness.

If replantation is not possible, then the principles of phalangeal ampuation are:
- A mid-axial skin incision on both sides of the digit
- Sharp proximal transection of nerves
- If the tendon insertion site is absent, debride tendon and allow it to retract
- Do not suture flexors to extensors
- Volar flaps rather than dorsal

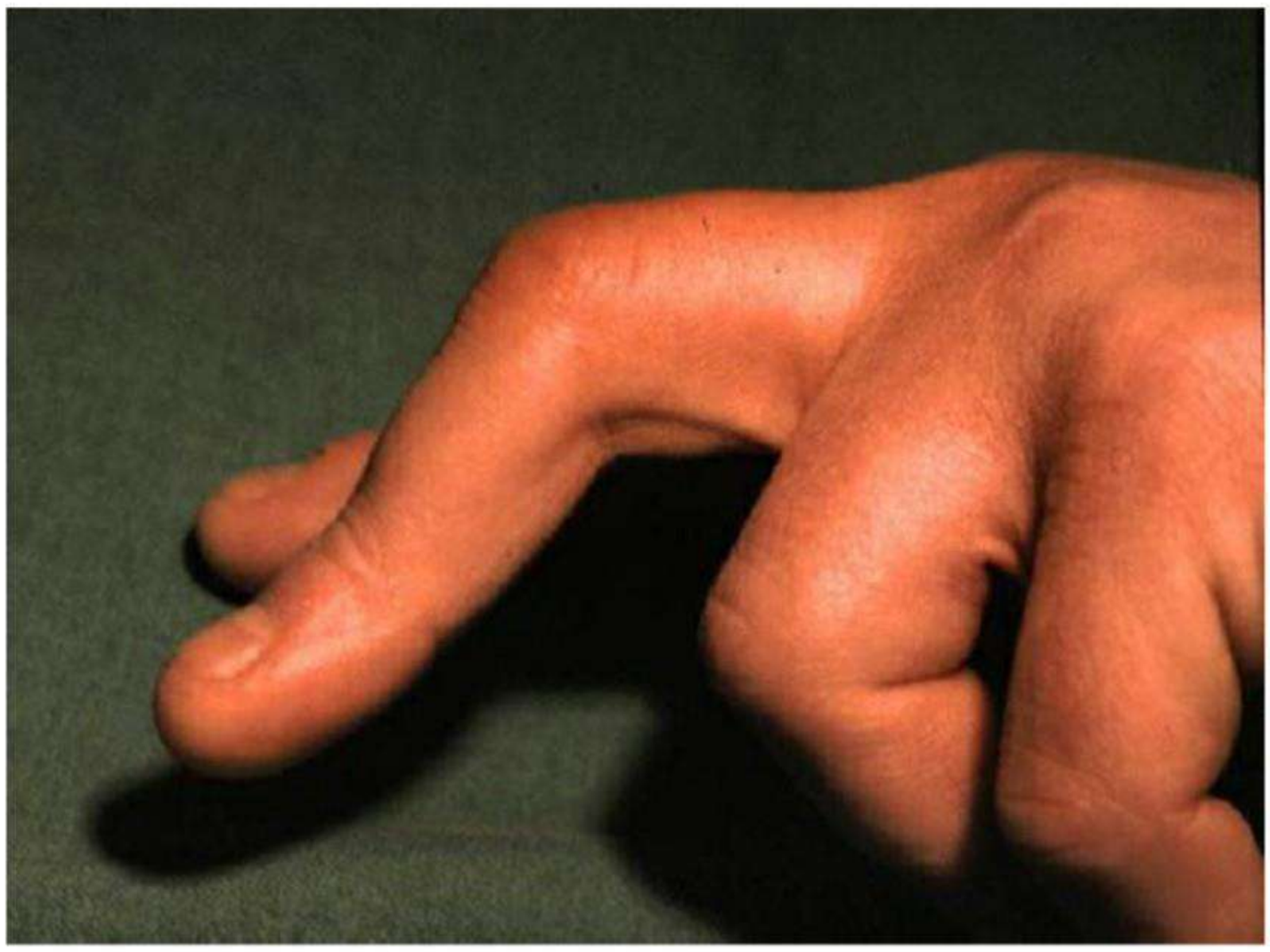
13 Plain radiograph of enchondroma proximal phalanx with fracture.
How would you manage this patient?



Define the lesion:
- An enchondroma is a benign lesion caused by an abnormality of chondroblast function in the physis
- It is the second most common benign cartilage tumour after osteochondroma
- They are solitary intramedullary tumours (diaphysis and metaphysis)
- The hand is the most common site (60%), with the proximal phalanges most common, followed by the metacarpals and middle phalanges
- Often present as pathological fractures
- Radiographic appearance of a well defined lucent lesion with a short zone of transition, a lobulated contour, thinning of the cortices and can have an area of 'pop-corn' calcification
- Malignant transformation is rare (around 1%)
- Differential diagnosis for this lesion includes bone infarct, chondrosarcoma and chondroblastoma.

Management (Review - O'Connor and Bancroft, 2004):
- Further imaging is seldom required for these lesions in the hand
- Allow the fracture to heal with a period of immobilization
- Surgical management then follows with open biopsy through a small incision removing tissue for frozen section. If the diagnosis is confirmed, the surgeon can proceed with enlargement of the bony lesion and curettage of the lesion. - - The margin of the excsion can be extended with a high speed burr where sufficient bone exists.
- Chemical cauterisation of the cavity can be made with phenol. The defect can then be packed with bone graft.
- Follow up of the patient with surveillance radiographs is at 6 months, 1 year and 2 years. The

14. Clinical picture of Boutonniere deformity.
What is the underlying pathology?
What is the classification and the management of this condition?



This is an aquired deformity of the extensor mechanism characterised by a flexion deformity at the PIPJ and an extension deformity at the DIPJ

They are caused by:
- Rupture of the central slip of the extensor mechanism, from attenuation (eg. secondary to capsular distension in rheumatoid arthritis), laceration or traumatic disruption
- Volar subluxation of the lateral bands because of to disruption or incompetence of the triangular ligament
- 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 extension of the DIPJ

Classification into four stages:

Stage	Description
I	The deformity is totally correctable passively, and there is full flexion of the DIP joint when the PIP joint is fully extended
II	Flexion of the DIP joint is limited when the PIP joint is passively corrected.
III	Stiffness of the PIP joint without joint destruction.
IV	Stiffness of the PIP joint with joint destruction

Management:
Nonoperative:
- Splint the PIPJ in extension for 6 weeks if the injury is less than 4 weeks old
- Encourage active DIPJ flexion and extension to avoid contraction of the oblique retinacular

- Encourage active DIPJ flexion and extension to avoid contraction of the oblique retinacular ligamemt

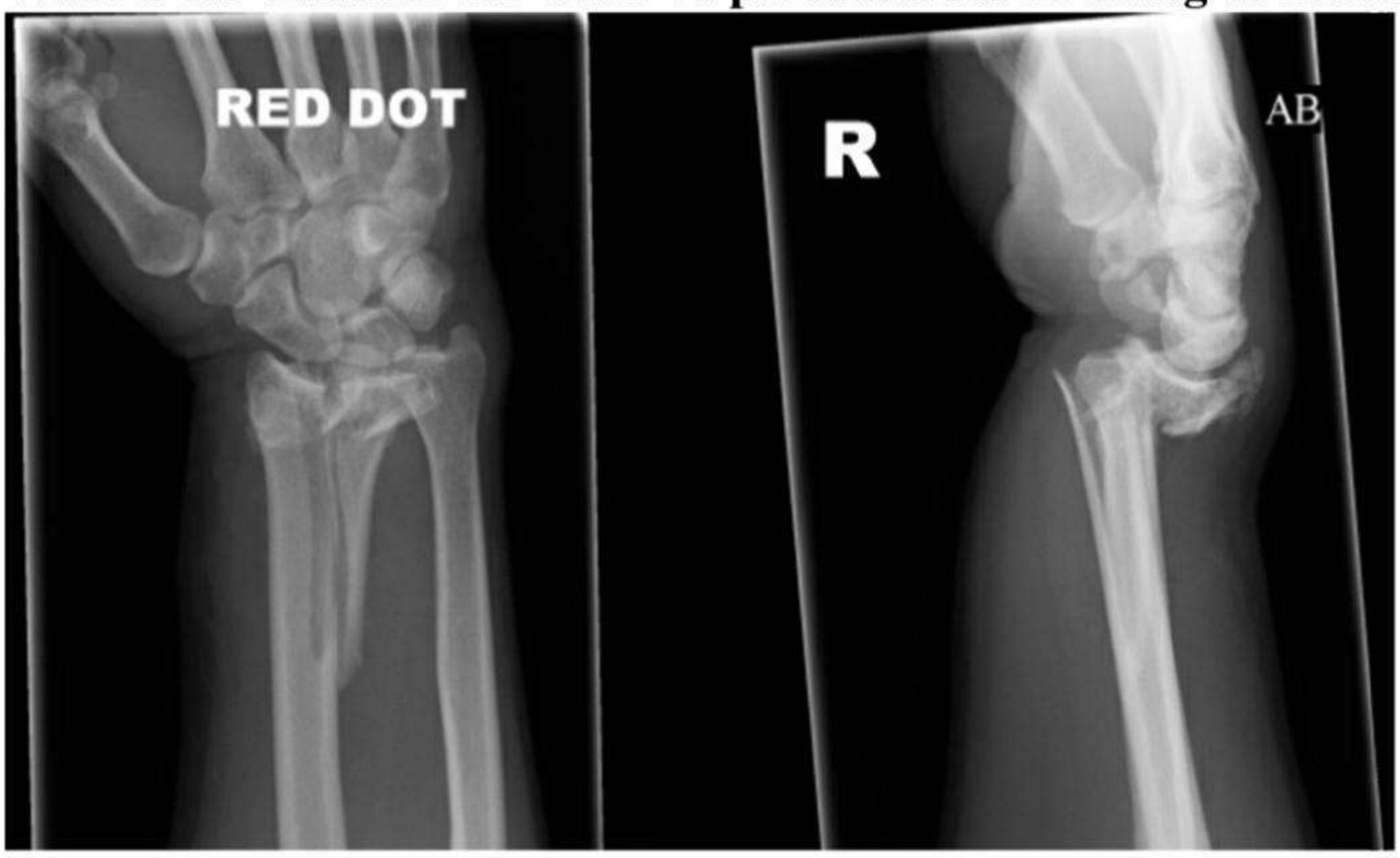
Operative (Stanley, 2004):
Choosing the most appropriate surgical procedure will depend on the severity of the anatomical deformities which need to be corrected:
- Improving passive PIPJ extension with serial casting or a Capner splint is worthwhile before surgery commenced
- Lateral band relocation - correction of PIPJ flexion with mobilization of the lateral bands posterior to the axis of rotation of the PIPJ
- Improving active DIPJ flexion with lengthening of the conjoined lateral bands over the middle phalanx
- PIPJ arthroplasty if the joint is destroyed but movement maintained
- Arthrodesis if there is gross uncorrectible deformity of the DIPJ or PIPJ

18. Plain radiographs of comminuted distal radius fracture.

D eradiographs.

How would you manage this patient?

What is the evidence for ORIF vs percutaneous K-wiring vs External fixation?



This is an AP and lateral radiographof the wrist in a skeletally mature patient. There is a comminuted, intra-articular distal radius fracture involving the radiocarpal joint and the distal radioulnar joint. There is dorsal translation and angulation of the metaphyseal fragments, with extension of the fracture proximally into the radial shaft.

Management:

- History to include age, occupation, handedness, mechanism of injury (low vs high energy) and activities of daily living
- Examination to assess skin quality and integrity, distal neurovascular status (in particular median and ulnar nerves), clinical deformity and continuity of extrinsic extensor and flexor tendons (especially EPL).

- Radiological Assessment:

View	Measurement	Normal	Acceptable healed position (Rockwood and Green, 2010)
Lateral	Volar tilt	11 degrees	Neutral
AP	Radial height	22 mm	Within 2-3 mm of contralateral wrist
	Radial inclination	11 degrees	<5 degrees loss
	Articular congruence	No step	<2 mm step-off

Non-operative management:

Closed reduction and a below elbow cast if the patient is medically unfit for surgery or previously

Non-operative management:

- Closed reduction and a below elbow cast if the patient is medically unfit for surgery or previou severely limited in completing ADLs.

Operative management:

1) Open reduction internal fixation:

Benefits:
- Accurate fracture reduction, volar locking plates allow direct fixation of comminuted fractures, bone grafting possible if bone loss or depressed fractures and early wrist mobilisation is possibl
- A prospective randomised trial of 144 intra-articular fractures found internal fixation produced superior results to bridging external fixation supplemented with percutaneous pinning, both radiographically and clinially (Leung et al., 2008).

Disadvantages:
- Risks of infection, nerve and tendon injury

2) Closed reduction and percutaneous K-wiring:

Benefits:
- Simple, minimally invasive and inexpensive
- Evidence to show that younger patients with extra-articular and simple articular fractures with l dorsal comminution can be treated successfully (Trumble et al., 1998)

Disadvantages:
- in patients with osteoporosis, a randomised controlled trial showed no advantage using percutaneous pins over cast alone (Stoffelen and Broos, 1999)
- Concerns about loss of position in a fracture with dorsal comminution with this technique
- Risk of infection of the pin sites and continued treatment in a cast causing stiffness.

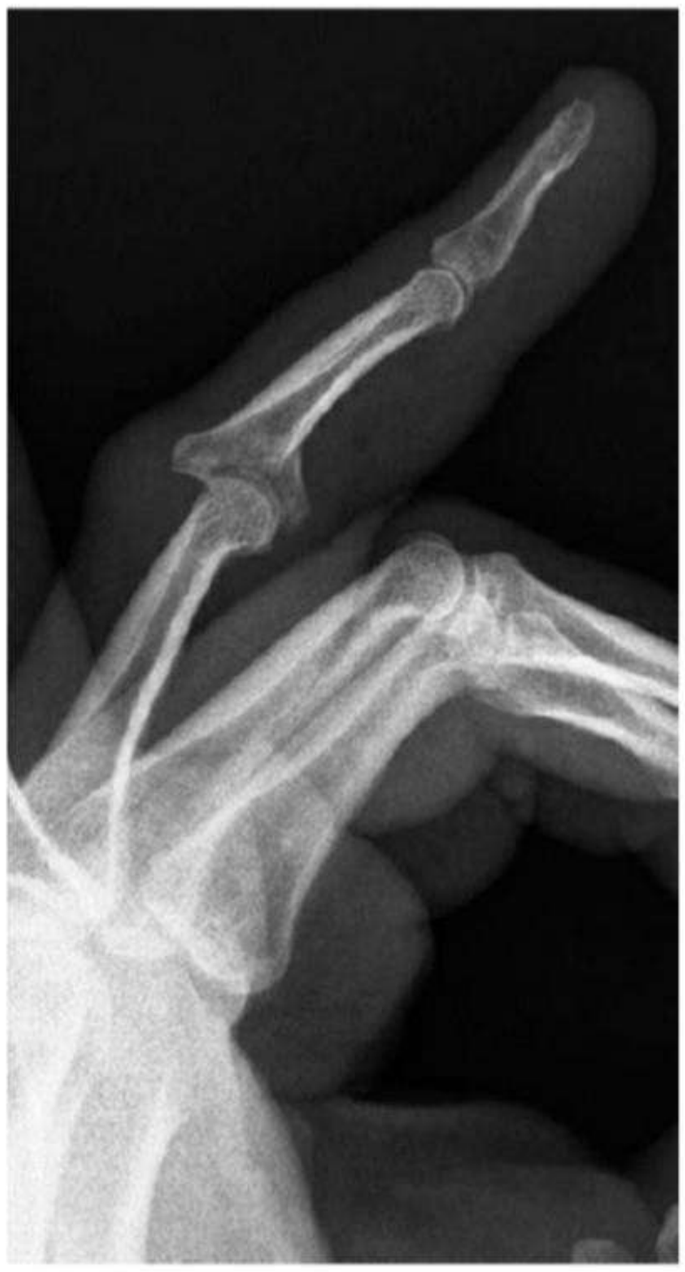
3) External Fixation:

- Includes bridging for intra-articular fractures (static or dynamic) or non-bridging for extra-artic fractures.
- Adjunctive fixation with K-wires for intra-articular fragments may be required.
- Non-bridging external fixation requires at least 1cm of intact volar cortex for the distal pins so would not be possible in this fracture
- Bridging external fixation relies on ligamentotaxis for fracture reduction. Studies have shown th this technique alone may not be sufficiently rigid to prevent some collapse and some loss of volar during healing (McQueen, 1998)

Benefits:
- Avoiding the zone of injury and damage control fixation

Disadvantages:
- Loss of reduction, stiffness if a bridging fixator is used, and possibility of superficial radial nerve

19. Plain radiographs of a dorsal PIPJ fracture-dislocation.
Describe the radiographic appearance.
What is your immediate assessment and treatment?
How would you splint the patient? How would you follow up this patient?

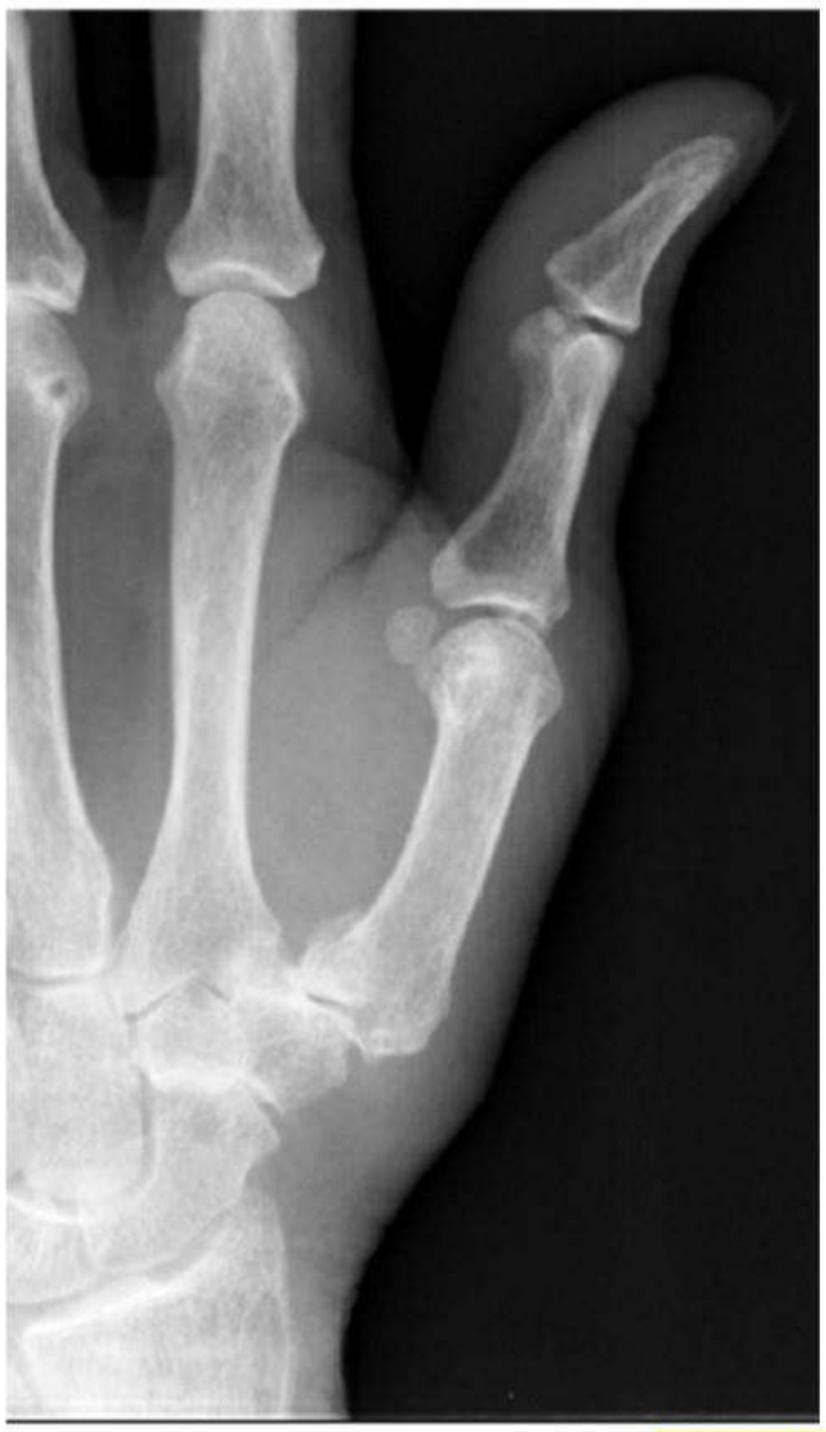


This is fracture dislocation of the base of the middle phalanx. The fracture involves more than 50 f the volar joint surface, with dorsal subluxation of the phalanx at the PIPJ. The fracture is not multifragmentary.

Immediate Assessment:
- History including age, occupation, handedness, mechanism of injury
- Examination including neurovasular and soft tissue status

Management:
- Reduce the fracture with traction and flexion at the PIPJ, then splintage in an extension block sp... at 40 degrees flexion with a repeat radiograph to check the position of the reduction
- Follow up in fracture clinic on a weekly basis, with reduction in the extension block by 10 to 1... degrees each week for the first three weeks
- The greater the proportion of the joint surface involved, the higher the angle of flexion that the extension blocking must begin, and the longer it takes to reach full extension. There is a greater risk of a permanent fixed flexion deformity in these patients.
- With more than 40% of the joint surface involved, the instability precludes non-operative management. ORIF or a dynamic fixator may be needed.

20. Plain radiograph of Bennett fracture. What is the management of this injury?



Bennett's fracture is an intra-articular fracture dislocation of the base of the 1st metacarpal.

The configuration of the fracture:

- A small volar fragment continues to articulate with the trapezium from the strong volar anterior oblique ligament (palmar beak ligament)
- Lateral retraction of the rest of the metacarpal because of the pull of abductor pollicis longus. The metacarpal head is displaced into the palm by the action of the adductor pollicis

Management:

Non-operative:

- The fracture can be reduced using thumb traction, abduction and extension with pronation
- The fracture can be held in position with a Bennett's cast (hitchhiker position) with moulding at the fracture site to maintain the position
- Accept up to 2 mm of fracture displacement (well tolerated at this joint

Operative:

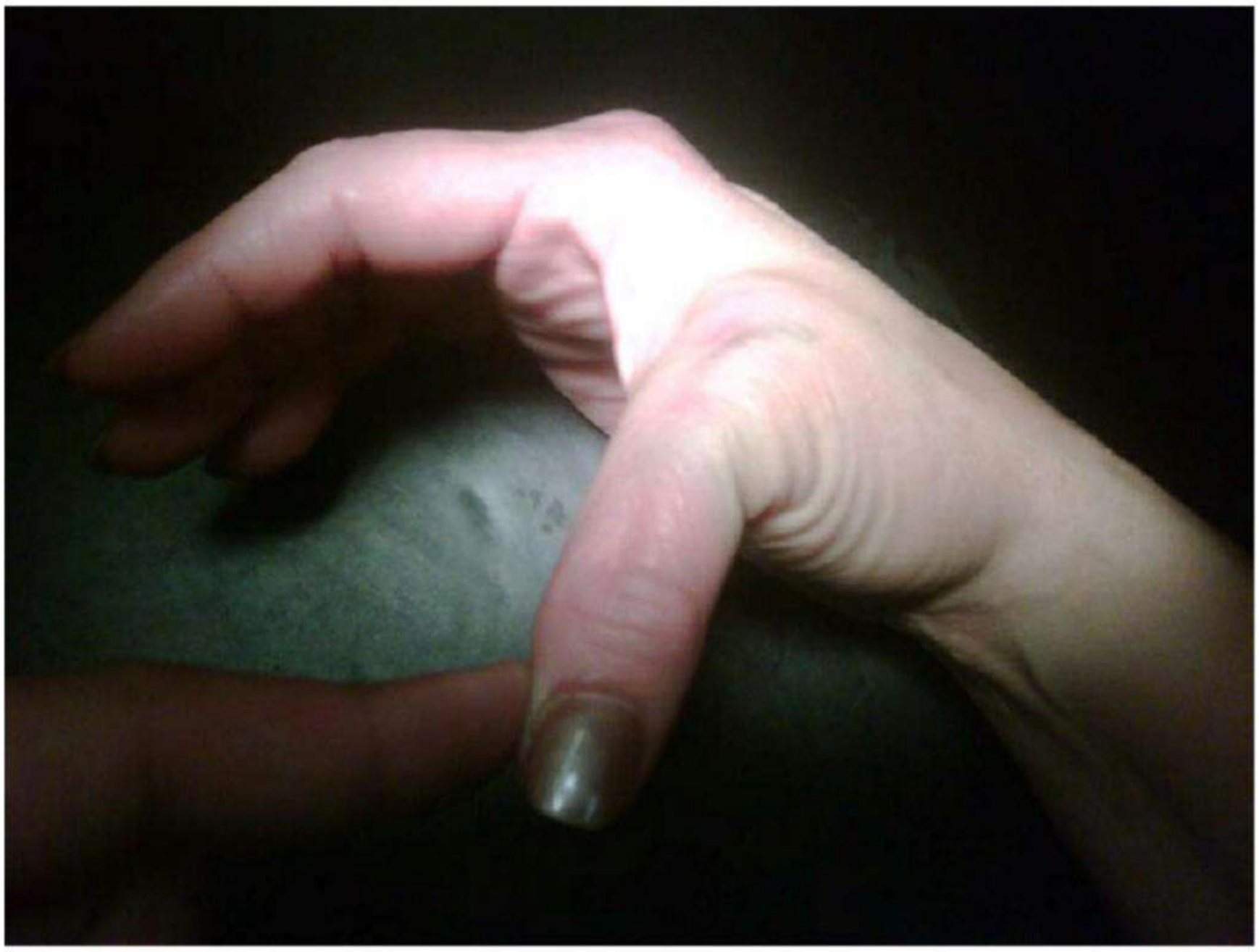
1) Closed reduction and percutaneous K-wire fixation:

- Consider when there is less than 3 mm displacement and volar fragment occupies less than 50% of the articular surface
- The wires stabilise the 1st metacarpal to the trazepium or 2nd metacarpal, and do not attempt to fix the volar fragment
- Complete in a spica cast for 6 weeks

2) Open reduction and internal fixation:

- Consider when there is more than 3 mm fracture displacement
- Use a limited incision to control the fracture fragments then stabilise with K-wires or, rarely, lag screw fixation and a T-shaped neutralisation plate with early mobilisation.

2. Clinical picture of a thumb with stress of the MCPJ in flexion.
What is the diagnosis?
How would you manage this injury?
What is a Stener lesion and where does it get stuck?



Diagnosis:
- This is a stress view radiograph of the thumb MCPJ. The opening at the ulnar aspect of the joint indicates an ulnar collateral ligament (UCL) injury. There is no associated avulsion fracture of the base of the proximal phalanx.

Management:
History:
- Includes age, occupation, handedness, mechanism of injury and chronicity of the injury.

Examination:
- Reveals swelling and tenderness over the ulnar apect of the MCPJ
- The clinical stress examination of the joint in flexion and extension, with laxity and no end point, is the gold standard for diagnosis (Tsiouri, 2009).
- Laxity in flexion of over 35 degrees (or 15 degrees more than the other side) indicates a rupture of the proper collateral ligament; laxity in extension indicates a rupture of the accessory collateral ligament. If the diagnosis of instability is uncertain, stress radiographs can be performed (Tsiouri, 2009).

Operative Management:
Anatomical basis:
- In acute, unstable injuries, acute repair is advocated because of the high likelihood of a Stener

lesion (Stener, 1962)

Operative Management:

Anatomical basis:

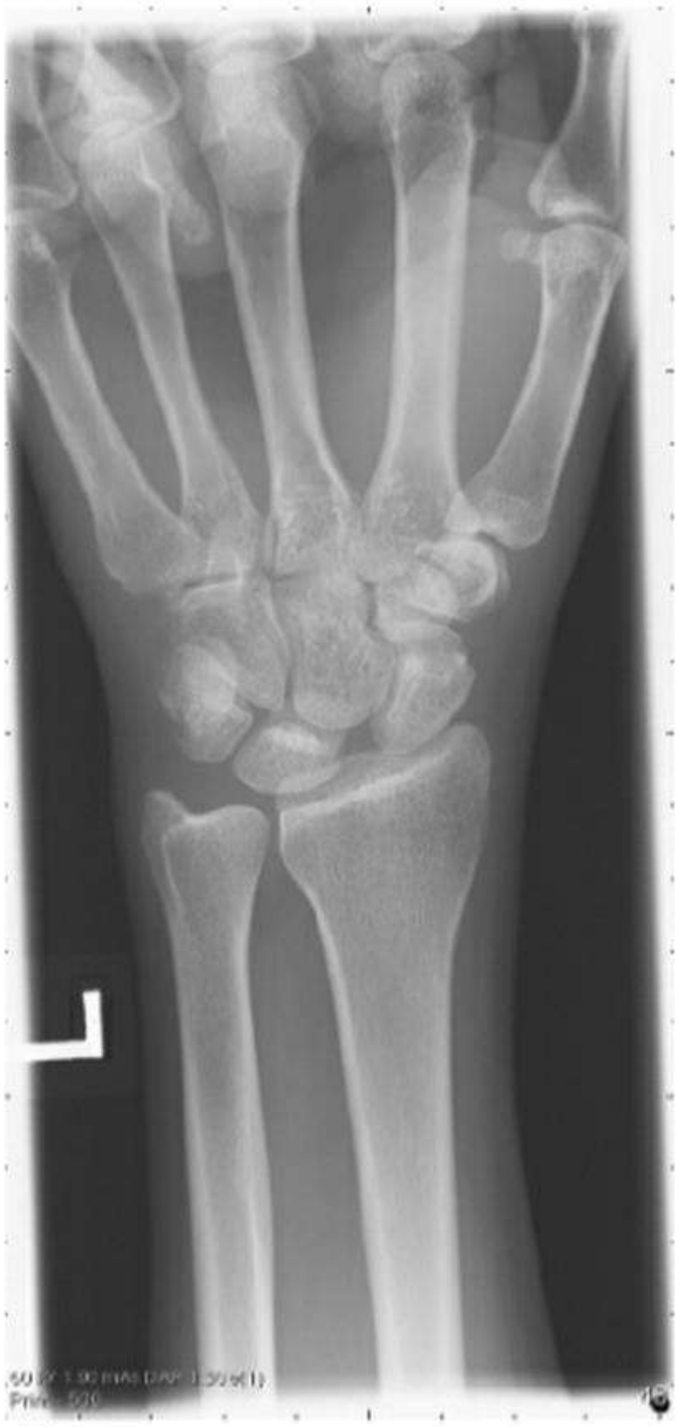
- In acute, unstable injuries, acute repair is advocated because of the high likelihood of a Stener lesion (Stener, 1962)
- A Stener lesion can prevent healing of the ulnar collateral ligament and can lead to chronic collateral ligament instability if treated non-operatively
- The anatomic basis of the Stener lesion is the proximal edge of the adductor aponeurosis
- The UCL usually tears at its attachment to the base of the proximal phalanx, and the torn stump comes to lie dorsal to the aponeurosis
- It is therefore prevented from healing back to its anatomic insertion at the volar, ulnar base of the proximal phalanx

Acute repair of the UCL:

A 'lazy-S' incision is made over the ulnar aspect of the base of the thumb.

- Care is taken to identify and protect the dorsal branches of the superficial radial nerve
- The adductor aponeurosis is identified and incised longitudinally, and the dorsal capsule and collateral ligaments are assessed
- The most common method of repair is the use of bone anchors, which give good results of loss of only 10 degrees of MCPJ and 15 degrees of IPJ motion (Weiland, 1997).
- Care must be taken in tensioning the repair to avoid stiffness

22. Plain radiograph showing an increased scapho-lunate gap.
What are the causes of this appearance?
How would you confirm the diagnosis?
Discuss SLAC wrist and its treatment?



Causes:
- Scapholunate (SL) dissociation because of a scapholunate ligament rupture
- Differential diagnosis of ulnar translocation, a reduced perilunate dislocation or a physiological scapholunate separation in lunotriquetral coalition

Radiological signs of scapholunate dissociation:
- SL gap of >3 mm on a clenched fist view (Terry Thomas sign)
- Cortical ring sign (given by cortical outline of the distal pole of the scaphoid in volar flexion)
- Scapholunate angle of >70 degrees from dorsal tilt of lunate and flexion of the scaphoid (normally around 45 degrees) on the lateral view

Confirm the diagnosis:
- History of fall onto outstretched hand with stress loading onto carpus in extension
- Examination findings of tenderness just distal to Lister's tubercle and a positive Watson's test (with the elbow resting on a tabletop and the forearm raised, with pressure over volar aspect of scaphoid and deviating the wrist from ulnar to radial, a clunk secondary to dorsal subluxation of the scaphoid over the doral rim of the radius is produced)

Investigations:
- MRI has low sensitivity but good specificity
- Wrist arthroscopy is the gold standard for diagnosis

Management:
Non-operative:
- In medically unfit patients or those who are not self-caring

Operative:
The options are divided into reconstructive and salvage

Acute:
- Scapholunate ligament repair is undertaken either directly or with bone anchors. The repair is protected with K-wires to the scapholunate and scaphocapitate joints

Chronic:
- Soft tissue procedures include a modified Brunelli (FCR tenodesis)
- Bony (salvage) procedures include a stabilisation with wrist fusion (e.g. STT) for rigid and irreducible dorsal intercalated segment instability (DISI)

SLAC wrist
- SLAC stands for 'scapholunate advanced collapse', with progressive instability causing arthritis of the radiocarpal and midcarpal joints from chronic dissociation between the scaphoid and the lunate

Management:
Non-operative:
- If symptoms are minor then advice, analgesia and splintage may be suitable

Operative:
Watson classification of SLAC wrist (radiographic appearance)

Type	Description	Operative Management
I	Arthritis between scaphoid and radial styloid	Radial styloidectomy and scaphoid stablisation (STT fusion) +/- PIN denervation PIN = posterior interosseous nerve
II	Arthritis between scaphoid and entire scaphoid fossa of the distal radius	Scaphoid excision and four corner fusion or proximal row carpectomy for relative preservation of strength and motion
III	Stage II + arthritis between lunate and capitate (eventually with proximal migration of the capitate)	Scaphoid excision and four corner fusion or wrist fusion (better pain relief and grip stregth at the expense of motion)

10.Tumors :

A.Oral :

- benign lesions are defined using Arabic numbers (1,2,3)
  - 1 = latent lesion
    - e.g. non-ossifying fibroma
    - enchondroma
  - 2 = active lesion
    - e.g. ABC, UBC
    - chondromyxoid fibroma
    - chondroblastoma
  - 3 = aggressive lesion
    - e.g. giant cell tumor of bone

Stage	Grade	Site (1)	Metastasis
IA	Low Grade	T1 - intracompartmental	M0 (none)
IB	Low Grade	T2 - extracompartmental	M0 (none)
IIA	High Grade	T1 - intracompartmental	M0 (none)
IIB	High Grade	T2 - extracompartmental	M0 (none)
III	Metastatic	T1 - intracompartmental	M1 (regional or distant)
III	Metastatic	T2 - extracompartmental	M1 (regional or distant)

Stage	Grade	Size	Depth	Node	Metastasis	5 yr. survival
IA	Low Grade	< 8cm	any	none	none	98%
IB	Low Grade	> 8cm			none	
IIA	High Grade	< 8cm	any	none	none	82%
IIB	High Grade	> 8cm	superficial	none	none	82%
III	any	discontinuous (skip) lesion	deep	none	none	52%
IVA	any	any		none	lung	
IVB	any	any	any	present	other than lung	30%

Harington's criteria

- > 50% destruction of diaphyseal cortices
- > 50-75% destruction of metaphysis (> 2.5 cm)
- Permeative destruction of the subtrochanteric femoral region
- Persistent pain following irradiation

Mirels' criteria

Mirels' criteria ?

score >/ 8 suggests prophylactic fixation

Score	*1*	*2*	*3*
Site	upper limb	lower limb	peritrochanteric
Pain	mild	moderate	functional
Lesion	blastic	mixed	lytic
Size	< 1/3	1/3 to 2/3	> 2/3

- **General principles**

1. Surgeon who performs Bx should be same surgeon who performs surgery
2. Center in which Bx performed should be same one to do surgery in & has oncology unit
3. Biopsy has to be evaluated by 2 general histopathologist or 1 musculoskeletal pathologist

- **Incision**

4. use longitudinal incision in the extremities (< 2 cm)

- **Approach**

5. Do not expose neurovascular structures (considered **contaminated** with tumor)
6. Take Bx away from NVB
7. maintain meticulous hemostasis by :
   1- Preventing post-op hematomas (considered contaminated with tumor)
   2- release tourniquet **prior** to wound **closure**
   3- Closure layer by layer

- **Biopsy**

8. perform through the involved compartment of the tumor (keep in **intra muscular** not inter muscular approach) (Bx through a single compartment)
9. For bone lesions with a soft tissue mass, it is ok to perform the biopsy using the soft tissue mass
10. Bx the **soft tissue component** when present
11. Take Bx form **Periphery** of the mass (Center of mass is necrotic)
12. Bx has to be **cultured** as well (each Bx has to be cultured & each tissue culture has to be histopathologically studied)
13. Each Bx should be taken by **different instruments** to prevent contamination

- **Closure**

14. if using a drain, bring drain out of the skin **in line with** surgical incision

(allows drain site to be removed with definitive surgical extensile incision)

(**1 cm distal** to wound or from wound it self)

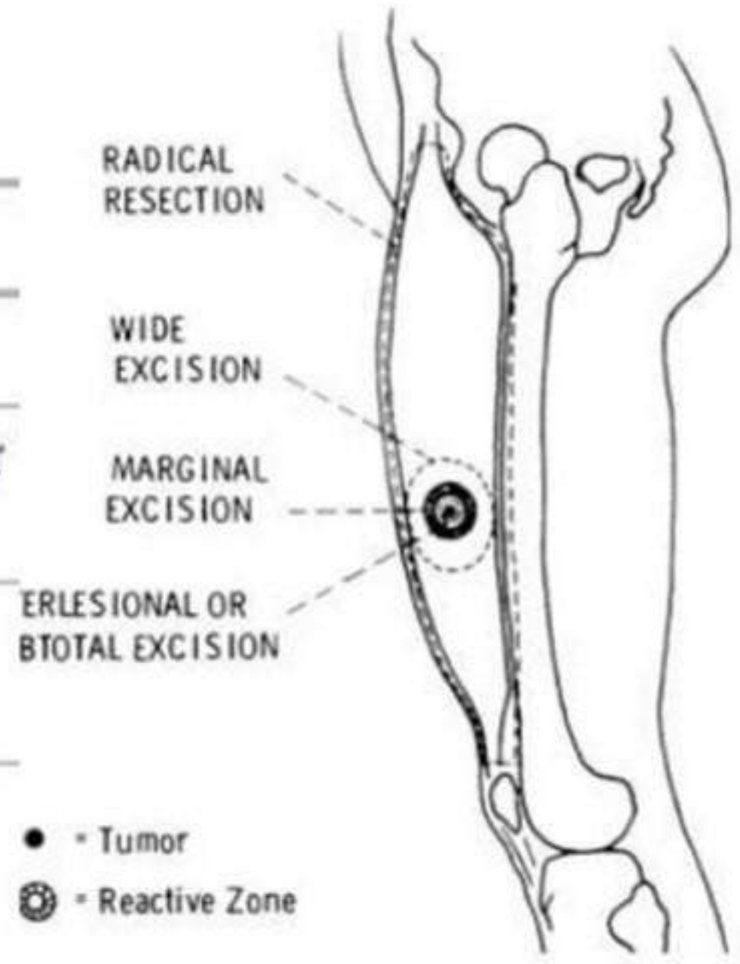
15. Site of Bone Bx should be **closed** with Bone cement or bone wax to **prevent hematoma**
16. **Subcuticular** continuous closure is better than interpreted simple closure

(interrupted will increase contamination at site of interrupted sutures & decrease stitch marks)

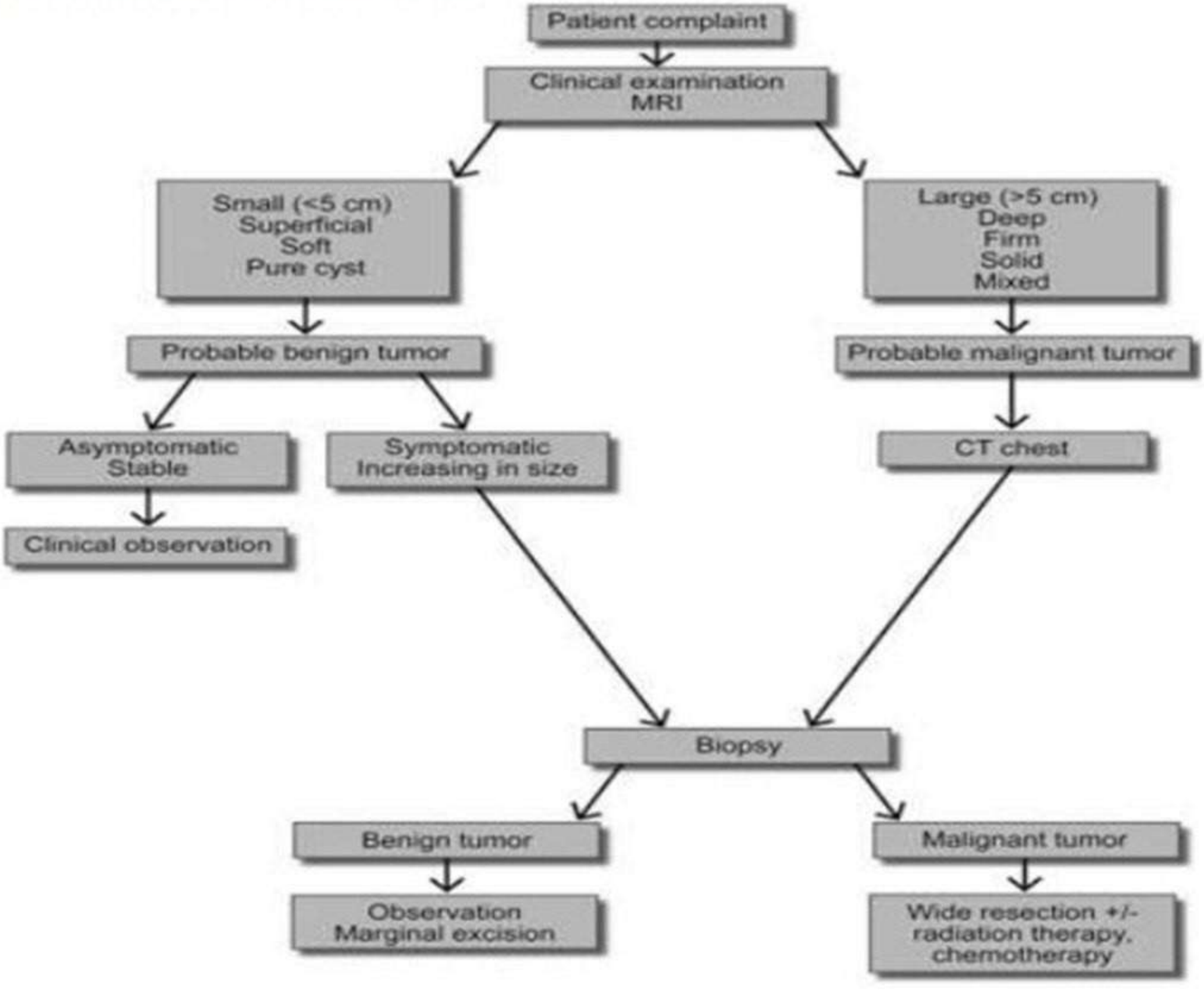
17. **No wash** should be done
18. Biopsies have to be **labeled** for orientation & site of specimen

Table 3 Surgical margins [30]

Type	Plane of dissection	Result
Intralesional	Piecemeal debulking or curettage	Leaves macroscopic disease
Marginal	Shell out *en bloc* through pseudocapsule or reactive zone	May leave either "satellite" or "skip" lesions
Wide	Intracompartmental *en bloc* with cuff of normal tissue	May leave "skip" lesions
Radical	Extracompartmental *en bloc* entire compartment	No residual



In summary…


Patient complaint
Clinical examination
MRI
Small (<5 cm)
Superficial
Soft
Pure cyst
Large (>5 cm)
Deep
Firm
Solid
Mixed
Probable benign tumor
Probable malignant tumor
Asymptomatic
Stable
Symptomatic
Increasing in size
CT chest
Clinical observation
Biopsy
Benign tumor
Malignant tumor
Observation
Marginal excision
Wide resection +/-
radiation therapy,
chemotherapy

Complications

- Soft tissue complications
  - early effects
    - delayed wound healing
    - infection
    - desquamation
  - late effects
    - fibrosis
    - joint stiffness
    - secondary sarcoma (below)
    - fractures (below)
- Post-radiation sarcoma
  - defined by the development of a sarcoma in a region previously radiated for malignancy
  - incidence is ~13%
    - more frequent in patients with prior chemotherapy
  - overal patient prognosis is very poor
- Post-radiation fractures
  - approximately 25% incidence following soft tissue sarcoma resection and external beam irradiation
    - risk factors for post radiation fracture
    - radiation dose ≥ 59Gy
    - weight bearing bones esp. femur
    - female
    - volume of bone receiving it
    - anterior femoral compartment resection
    - age
    - periosteal stripping
      - some advocate prophylactic fixation if periosteal stripping is performed
    - osteoporosis

B.Viva : Long & Short :

Chapter 7

Orthopaedic oncology

Thomas B. Beckingsale

Definitions

As in all other areas of the viva examinations, knowing basic definitions gives you an easy starting point when answering questions and gives the impression to the examiners that you have both a logical and clear thought process, and are in command of the subject matter.

Neoplasm/tumour: A growth or swelling, which enlarges by cellular proliferation more rapidly than surrounding normal tissue and continues to enlarge after the initiating stimuli cease. Usually lacks structural organization and functional coordination with normal tissues and serves no useful purpose to the host.

Malignant tumour: Malignant tumours have a predisposition to invasive and destructive local growth, and to distant metastasis usually via the vascular or lymphatic systems.

Benign tumour: Benign tumours do not metastasize, but can still exhibit locally aggressive behaviour.

Sarcoma: A diverse and rare group of malignant tumours of mesenchymal/connective tissue origin. Tumours of peripheral nerves are often included in this group.

Generic structured oral examination question 1: Biopsy

EXAMINER: So how would you obtain a tissue diagnosis?

CANDIDATE: A tissue sample can be obtained by biopsy. In general terms this can be performed by excisional, incisional or percutaneous means, but I would not perform a biopsy without first having discussed the case with a bone tumour multidisciplinary team (MDT).

EXAMINER: Good. Let's suppose you are the bone tumour surgeon now. When might you perform an excision biopsy?

CANDIDATE: The indications for an excision biopsy are narrow. The entire lesion is removed and the margins are often marginal. Hence, this type of biopsy is really only applicable to benign lesions where the imaging has been diagnostic, for example lipomas, or where the lesion is small and superficial such that excision biopsy would not compromise later re-excision. However, if there is any doubt about the diagnosis I would perform a percutaneous or incisional biopsy first.

EXAMINER: Ok, tell me how you would perform an incisional biopsy.

CANDIDATE: I would perform the procedure through a short longitudinal incision. I would plan the incision using the imaging, and position it such that the entire biopsy tract could be excised *en bloc* during the definitive resection, and such that it does not contaminate more than one compartment or key neurovascular structures. I would pay close attention to haemostasis and use minimal tissue dissection in order to minimize local tissue seeding.

EXAMINER: We perform most of our biopsies percutaneously now. Do you know any advantages or disadvantages to doing it this way?

CANDIDATE: I've seen biopsy performed by Tru-Cut needle. The procedure can be performed easily in clinic under local anaesthetic, which removes delay and the requirement for theatre time. Welker *et al*. have shown that it is safe, has a low complication rate and reliably provides enough tissue for diagnosis and treatment planning.[1] Other advantages are that it can be combined with imaging modalities, for example ultrasound for soft tissue lesions and CT for bony lesions. The disadvantage is that necrosis and mitotic rate is less reliable on core needle but this rarely affects management, and an incisional biopsy can always be performed subsequently if more information is required.

Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination, ed. Paul A. Banaszkiewicz and Deiary F. Kader. Published by Cambridge University Press.

1. Welker JA, Henshaw RM, Jelinek J, Shmookler BM, Malawer MM. The percutaneous needle biopsy is safe and recommended in the diagnosis of musculoskeletal masses. *Cancer* 2000;**89**(12):2677–2686.

Generic structured oral examination question 2: Margins

EXAMINER: What do you understand by a marginal margin?

CANDIDATE: A marginal margin, as described by Enneking, is when the resection line passes through the reactive zone of the tumour being excised.[1]

EXAMINER: Explain to me what you mean by the reactive zone.

CANDIDATE: Tumours grow in a centifugal fashion and this leads to compression and subsequent atrophy of the surrounding tissue forming a pseudocapsule. Outside the pseudocapsule is an area of oedema where inflammatory cells and micronodules of tumour are present. This is the reactive zone. Hence, if a resection line passes through this reactive zone, as in a marginal margin, then micronodules of tumour are likely to be left behind, increasing the risk of a local recurrence.

EXAMINER: So what other margins did Enneking describe and what do you understand by them?

CANDIDATE: Enneking described three other possible margins. He described intra-lesional margins, where the resection line passes through the tumour leaving macroscopic deposits of tumour in the surgical wound. He described wide margins, where the resection line passes outside the reactive zone and the tumour is excised with a surrounding cuff of normal tissue. In wide margins it is still possible that tumour will remain in the form of skip lesions. Finally, he described the radical margin, where the entire compartment in which the tumour resides is excised *en bloc*, in theory removing the entire tumour.

1. Enneking WF, Spanier SS, Malawer MM. The effect of the anatomic setting on the results of surgical procedures for soft parts sarcoma of the thigh. *Cancer* 1981;**47**(5):1005–1022.

Generic structured oral examination question 3: Staging

EXAMINER: So what stage is this tumour?

CANDIDATE: I would stage this tumour using the Musculoskeletal Tumour Society staging system as described by Enneking.[1] We have discussed that it is a high-grade osteosarcoma, which makes it at least Stage II. It's an intramedullary tumour that has invaded the surrounding soft-tissues making it extracompartmental, and upstaging it to IIB. We've not discussed whether there is any evidence of metastasis yet, but, if there is, that would immediately make it a Stage III, regardless of the other features we've talked about.

Table 7.1 Enneking/MSTS staging system.[2]

Stage	Description	Grade	Site	Metastases
IA	Low-grade, intracompartmental	G_1	T_1	M_0
IB	Low-grade, extracompartmental	G_1	T_2	M_0
IIA	High-grade, intracompartmental	G_2	T_1	M_0
IIB	High-grade, extracompartmental	G_2	T_2	M_0
III	Any grade, metastatic	G_{1-2}	T_{1-2}	M_1

General advice: This question will usually follow a discussion about a malignant tumour, for example osteosarcoma as in this example. The Enneking system (Table 7.1) is the easiest to remember and can be applied equally to bony and soft-tissue sarcomas.[2] The other commonly used system is the American Joint Committee on Cancer (AJCC) system, which is more complicated. The AJCC also have separate systems for bony and soft-tissue tumours.

1. Enneking WF, Spanier SS, Goodman MA. Current concepts review. The surgical staging of musculoskeletal sarcoma. *J Bone Joint Surg Am* 1980;**62**-A:1027–1030.
2. NCCN. *National Comprehensive Cancer Network Clinical Practice Guidelines in Oncology: Soft Tissue Sarcoma. V.2.2008*. National Comprehensive Cancer Network, 2008.

Structured oral examination question 1: Osteochondroma

EXAMINER: This young lad has been referred to you urgently by his GP after his mum brought him in with a firm lump on the front of his left thigh. Tell me about his X-ray. (Figure 7.1.)

CANDIDATE: This is a lateral radiograph of his left femur including the knee joint but not the hip joint. There is a bony

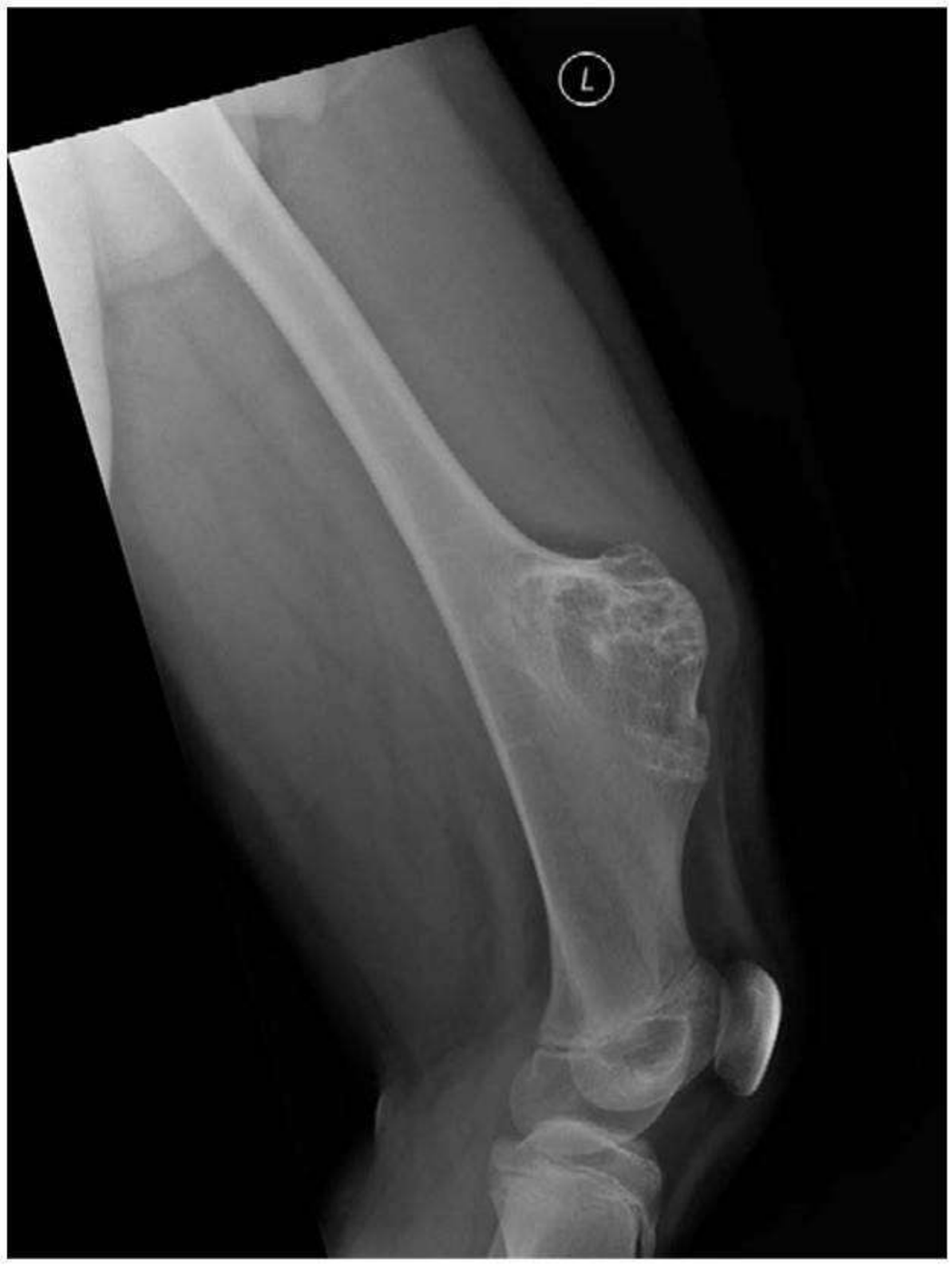


Figure 7.1 Osteochondroma.

growth on the anterior aspect of the femur, which looks like a large osteochondroma.

EXAMINER: What makes you think it's an osteochondroma?

CANDIDATE: Well, the cortices are in continuity with the bone as is the medullary cavity, and the lesion is extending out from the metaphyseal region of the distal femur, which is the most common site for these (25%). This is a sessile lesion rather than the pedunculated variety and appears to be a solitary lesion, although I'd want to examine the child to look for other lumps. It is quite a large lesion and there is some slightly atypical sclerosis within it so I would definitely get an MRI scan.

EXAMINER: OK, so you get an MRI, which shows a nice thin cartilage cap and no worrying features. How are you going to treat it?

CANDIDATE: First I'd take a history and examine the child. I'd want to know if it is tender or symptomatic before I decide what to do.

EXAMINER: It's not tender and it only bothers him occasionally if he knocks it, but his mother is adamant she wants it removed.

CANDIDATE: I would suggest a period of watchful waiting to see if it continues to grow or becomes more symptomatic. Removing it would carry risks of recurrence and neurovascular damage. There is also a chance of fracture, during the operation and afterwards as it's a large sessile lesion and removing it will weaken the anterior cortex of the femur considerably.

EXAMINER: His mum still wants it removed and she's worried that it's going to become cancer.

CANDIDATE: If this is a solitary lesion then malignant change is very rare indeed. If the child has multiple hereditary exostoses the risk is a bit higher. The textbooks often quote figures of 10% but it is probably more like 1–5%.

General advice: Examiners may show an example of a solitary osteochondroma in an area that is difficult to access for the purposes of excision, but then insist that the patient wants it removed, e.g. posterior, proximal tibia. The resultant discussion is then used to assess knowledge of anatomy and approaches, e.g. posterior approach to the knee. If the MRI has shown no sinister features, and the lesion is asymptomatic, then you can have a reasoned discussion with the examiner about watchful waiting versus removal, i.e. both answers are perfectly acceptable.

Other points:

- Continued growth after physeal closure raises the suspicion of malignant transformation.
- EXT gene mutation is the genetic abnormality in multiple hereditary exostoses. It is an autosomal dominant condition.

Structured oral examination question 2: Enchondroma

EXAMINER: Tell me about these radiographs of this chap's right foot. (Figure 7.2.)

CANDIDATE: Well they're AP and oblique views and they show an expansile, lytic lesion in the proximal phalanx of his second toe.

EXAMINER: What do you think it is?

CANDIDATE: The radiographs show features consistent with an enchondroma. It has a short zone of transition and appears quite well defined. There's also some stippled calcification within the substance of the lesion, which suggests a chondroid matrix.

EXAMINER: How would you treat this lesion?

CANDIDATE: Well, I would want to get more information so I would take a full history and examination. I would also want to get more imaging of the lesion with an MRI and discuss the pictures with a bone tumour MDT. If there's any doubt about the diagnosis they may want to do a biopsy, but in general the surgical treatment of an enchondroma is with curettage, with or without grafting.

General advice: Even if the diagnosis appears obvious and is of a benign lesion, don't be rushed into offering surgical treatment. Always work through history, examination and imaging. You will never be criticized for discussing the diagnosis with a bone tumour MDT, but you will end up in a very tricky discussion with the examiners and fail if you have made the wrong diagnosis, it turns out to be malignant, and you've not discussed it with an MDT first.

Other points:

- 50% of solitary enchondromas arise in the hands.
- Malignant transformation is very rare, but when it does occur it is usually in large lesions of long bones.
- Enchondromatosis = Ollier's disease (risk of bone malignancy is 10%, but if visceral and brain malignancies are included then the overall risk is 25%).
- Enchondromatosis + haemangiomas = Maffucci syndrome (risk of malignancy approaching 100%).

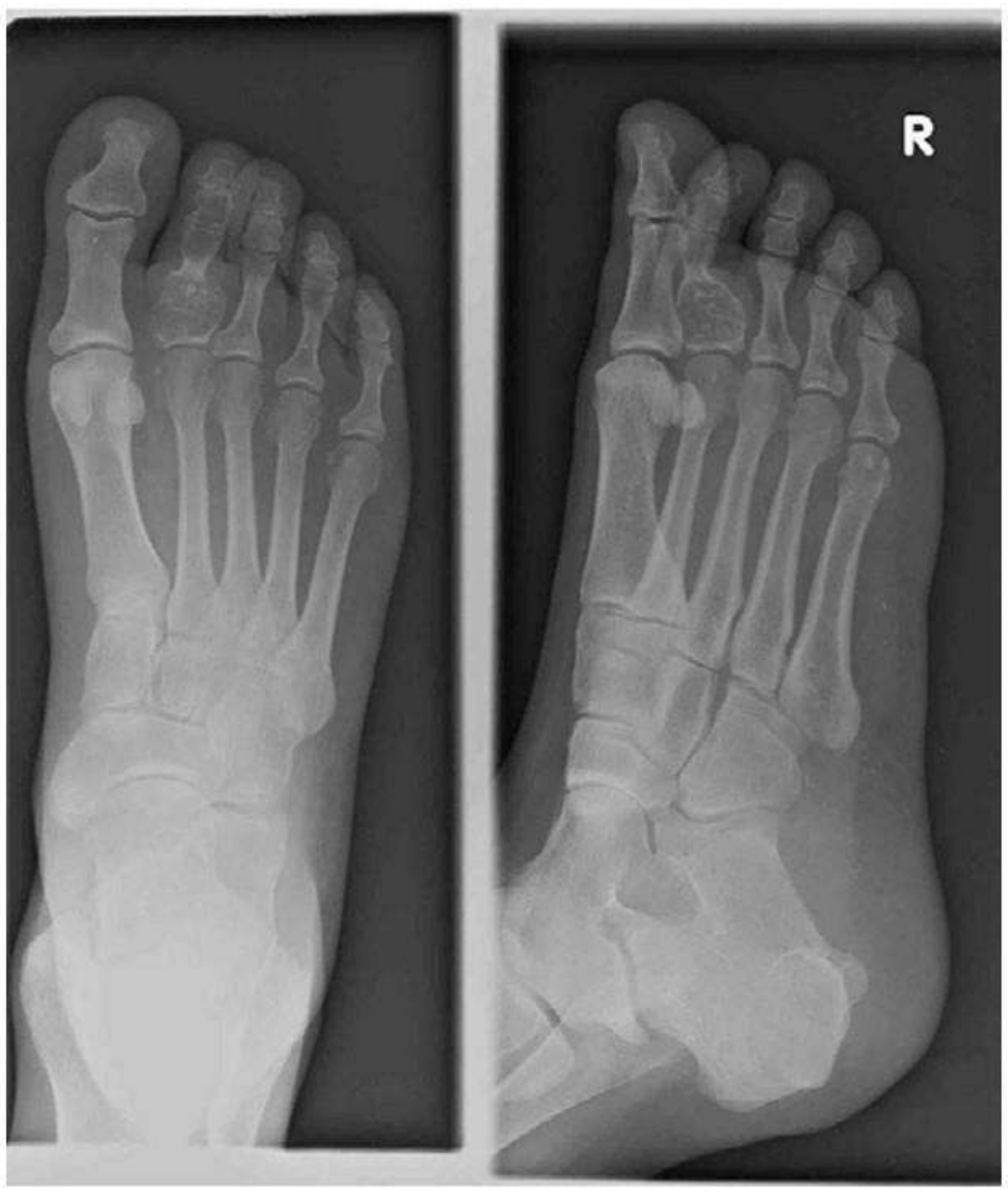


Figure 7.2 Enchondroma.

Structured oral examination question 3: Non-ossifying fibroma

EXAMINER: Tell me about this radiograph. (Figure 7.3.)

CANDIDATE: This is an AP radiograph of a left lower leg of a child, which includes both the ankle joint and the knee joint. There is a lucent lesion, eccentrically placed in the metaphyseal region of the tibia. The lesion is well-demarcated and its margin is slightly sclerotic. These features are typical of a non-ossifying fibroma.

EXAMINER: Good. What else can you tell me about this lesion?

CANDIDATE: Non-ossifying fibromas are developmental or hamartomatous lesions. They are actually very common and some have suggested an incidence of up to 35% in normal

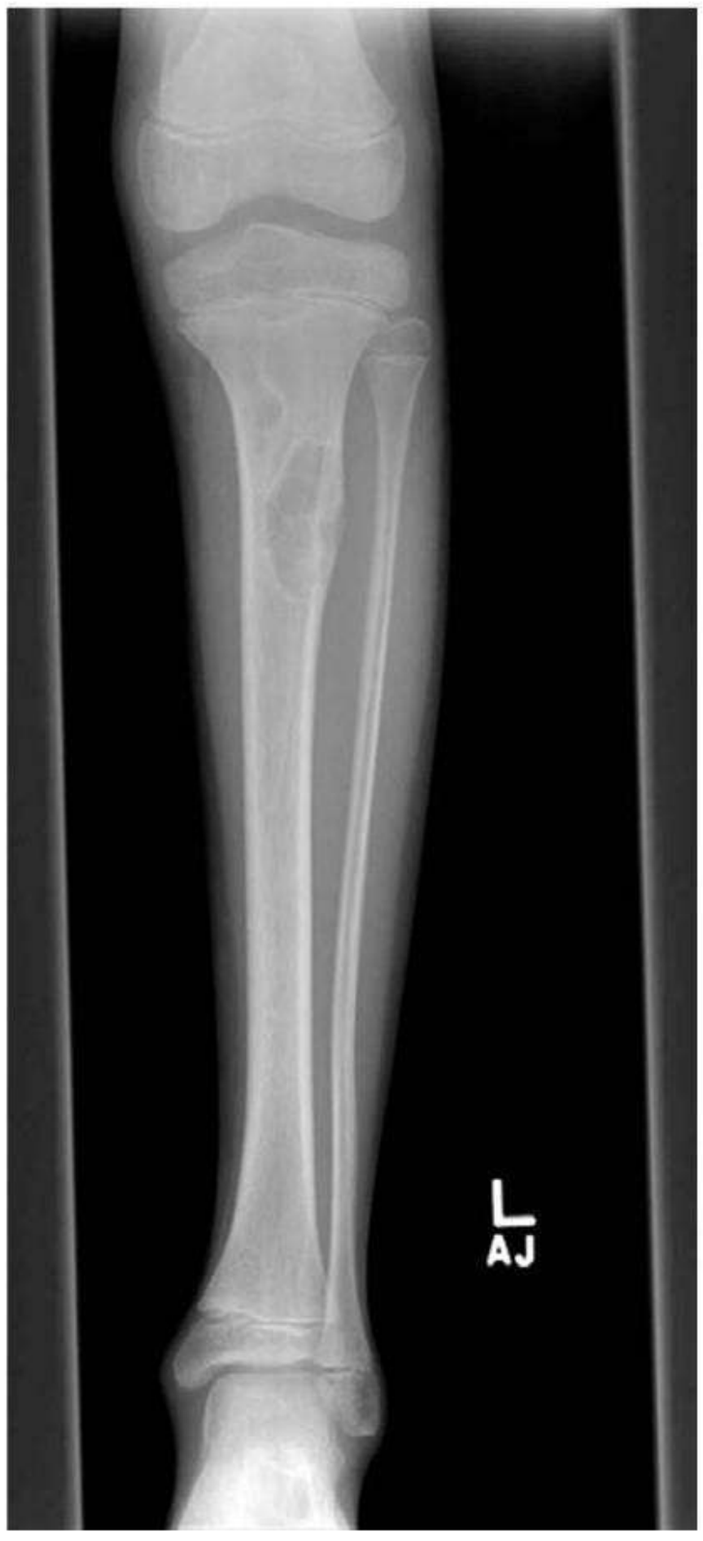


Figure 7.3 Non-ossifying fibroma.

children. They are usually asymptomatic and are often discovered as an incidental finding. Occasionally they can present after a pathological fracture, after which they tend to heal up.

EXAMINER: How would you treat this lesion?

CANDIDATE: I can't see any evidence of fracture. I would take a history and examine the patient to ascertain whether the lesion is painful or symptomatic and I would discuss the images with our local tumour MDT to make sure that they were in agreement with the diagnosis. That being the case this can be treated with observation only as these lesions normally resolve by adulthood. I would plan to keep the patient under review with surveillance radiography.

General advice: Again, you will not be criticized if you say that would take advice from the bone tumour MDT. You will, however, be in a very difficult situation if you have not stated that you would take their advice and your diagnosis is wrong.

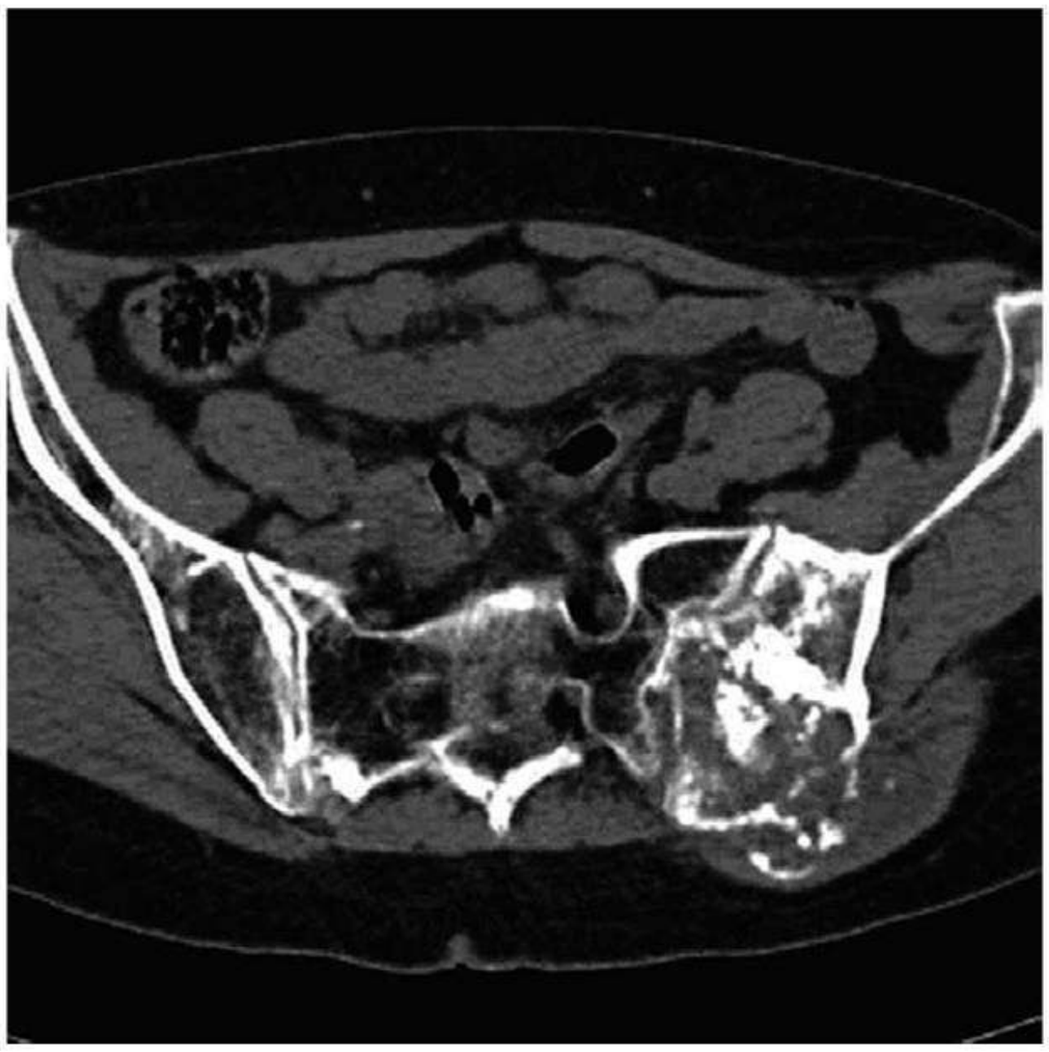


Figure 7.4 Chondrosarcoma.

Structured oral examination question 4: Chondrosarcoma

EXAMINER: This 60-year-old lady presented with pain and swelling around her lower back. What can you see on this CT scan? (Figure 7.4.)

CANDIDATE: This is an axial section showing the sacrum and iliac wings. There is an expansile lesion in the left iliac wing, which has extended into the soft tissues. The lesion has both lytic and sclerotic elements to it.

EXAMINER: What do you think the diagnosis might be?

CANDIDATE: The expansile nature, as well as the permeative margin and local invasion suggest a malignant process. Malignant tumours of bone can then be broken down into primary, metastatic, or immunohaematopoietic lesions. Metastatic and immunohaematopoietic tumours tend to produce lytic lesions within bones, whereas this lesion has areas of sclerosis and is much more expansile. Primary bone tumours can be classified according to their matrix as either bone-producing, cartilage-producing, fibrous tissue-producing or non-matrix producing. The patchy sclerosis within this lesion is in keeping with either a bone- or cartilage-producing primary tumour, although I would not rule out other diagnoses without further investigations.[1]

EXAMINER: You're right to suggest a primary lesion in this case. You've suggested bone- or cartilage-producing as the likely matrix. Which do you think is more likely here?

CANDIDATE: It is most likely to be a chondrosarcoma. The incidence of chondrosarcoma increases with age. This lady is 60 and although there is a second peak in the incidence of osteosarcoma in elderly patients, the majority of cases occur in adolescents around the growth spurt. The site of the tumour also makes chondrosarcoma the more likely diagnosis. Only around 5% of osteosarcomas occur in the pelvis, whereas up to 30% of chondrosarcomas are pelvic in origin. Finally, there is the appearance on the CT. It is not the clearest image but I'm trying to convince myself that there's stippled calcification, which would indicate a chondroid lesion.

EXAMINER: Very good. What treatment options are there for an aggressive-looking chondrosarcoma like this is?

CANDIDATE: Chondrosarcomas are poorly chemo- and radiosensitive so the only treatment option is wide local excision plus or minus reconstruction. However, despite surgical excision, longer-term survival is dependent on the presence or absence of metastases.

EXAMINER: So what do you think the prognosis is for this high-grade lesion?

CANDIDATE: The key is the presence or absence of metastasis and the patient needs staging investigations. In general, low-grade, or grade I, lesions are rarely metastatic and have a better than 90% 5-year survival. High, Grade III, lesions, as you've

intimated this one is, are metastatic in over 70% of cases and have only a 30% 5-year survival.

1. Bullough PG. *Orthopaedic Pathology*. Fourth Edition. Edinburgh: Mosby, 2007.

Structured oral examination question 5: Chondrosarcoma

EXAMINER: This is a very fit and well 50-year-old chap, who has come into A&E after falling down the stairs at home, sustaining this injury to his left leg. Tell me how you are going to manage this. (Figure 7.5.)

CANDIDATE: I would manage this patient initially using the principles of ATLS [Airway and protect cervical spine, Breathing, Circulation, Disability, Exposure and environment control].

EXAMINER: Fine. No issues with ABC and the patient is alert and orientated.

CANDIDATE: Moving on I want to assess whether the patient has any other injuries, and regarding this injury I want to know whether it is open or closed and whether the limb is neurovascularly intact.

EXAMINER: Okay. This is his only injury. It's an open fracture with a 1 cm wound on the lateral thigh. The limb is neurovascularly intact. How are you going to manage this?

CANDIDATE: If it's an open injury then I would take a picture of the wound and cover it with a betidine-soaked swab. The patient needs IV antibiotics and coverage for tetanus, depending on their vaccination history. Some form of immobilization is also important for patient comfort and nursing care. In this case I can see that a Thomas splint has been applied.

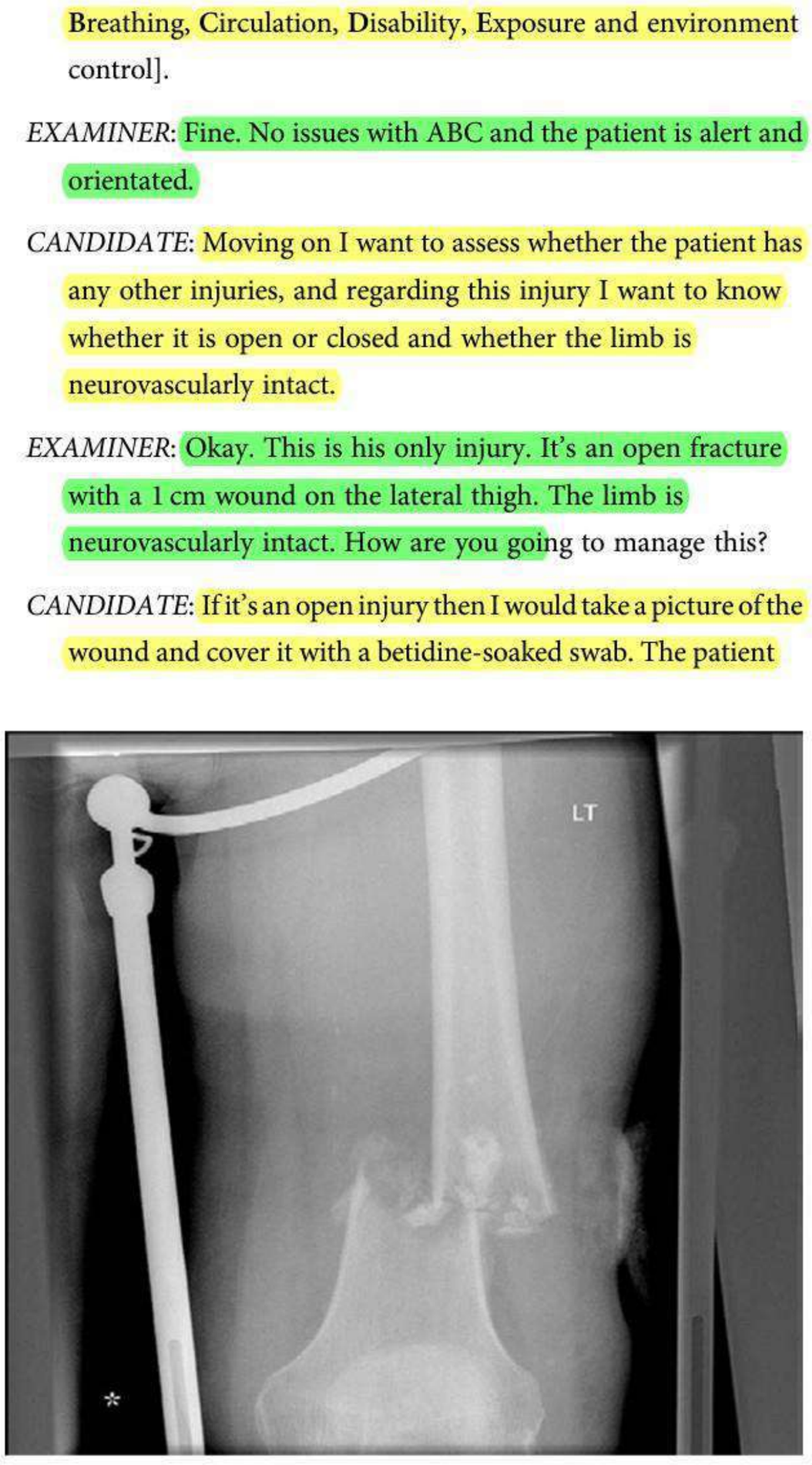


Figure 7.5 Chondrosarcoma.

EXAMINER: Good. So shall I book this patient for theatre with a plan to perform a debridement of the wound and nailing of the fracture?

CANDIDATE: Well, I know it's an open fracture but I have some concerns about the X-ray. There's some odd calcification within the medullary cavity, so I'm worried that this is a pathological fracture through a bony lesion.

EXAMINER: Why does that make a difference?

CANDIDATE: It's a rare situation, but if this is pathological fracture through a bone tumour, and we open up the fracture site and nail it, we would spread tumour the length of the femur and might convert a resectable tumour into one that is unresectable.

EXAMINER: But doesn't the open fracture need washing out?

CANDIDATE: It's a small puncture wound and I've put the patient on IV antibiotics so I think the infection risk is low. In this case I would arrange some urgent investigations, get more information and discuss the case immediately with the bone tumour MDT before rushing the patient to theatre.

General advice: Always look at the available evidence carefully and look out for any atypical features. If in doubt, say so! You will never be criticized for taking advice, but you will fail if you have blazed on with treatment and taken this patient to theatre for wash-out and nailing. If there is no threat to life or limb, then there is always time for further investigations, and to seek further opinions.

Always take time to look carefully before answering, especially if a question on a fracture comes up in the adult and pathology viva station.

Structured oral examination question 6: Osteosarcoma

EXAMINER: This young lad presented with a painful knee and a lump after a football injury. What do you think of the X-ray? (Figure 7.6.)

CANDIDATE: [*When I was shown the X-ray I immediately thought that the diagnosis was an osteosarcoma and described the X-ray changes that supported my initial*

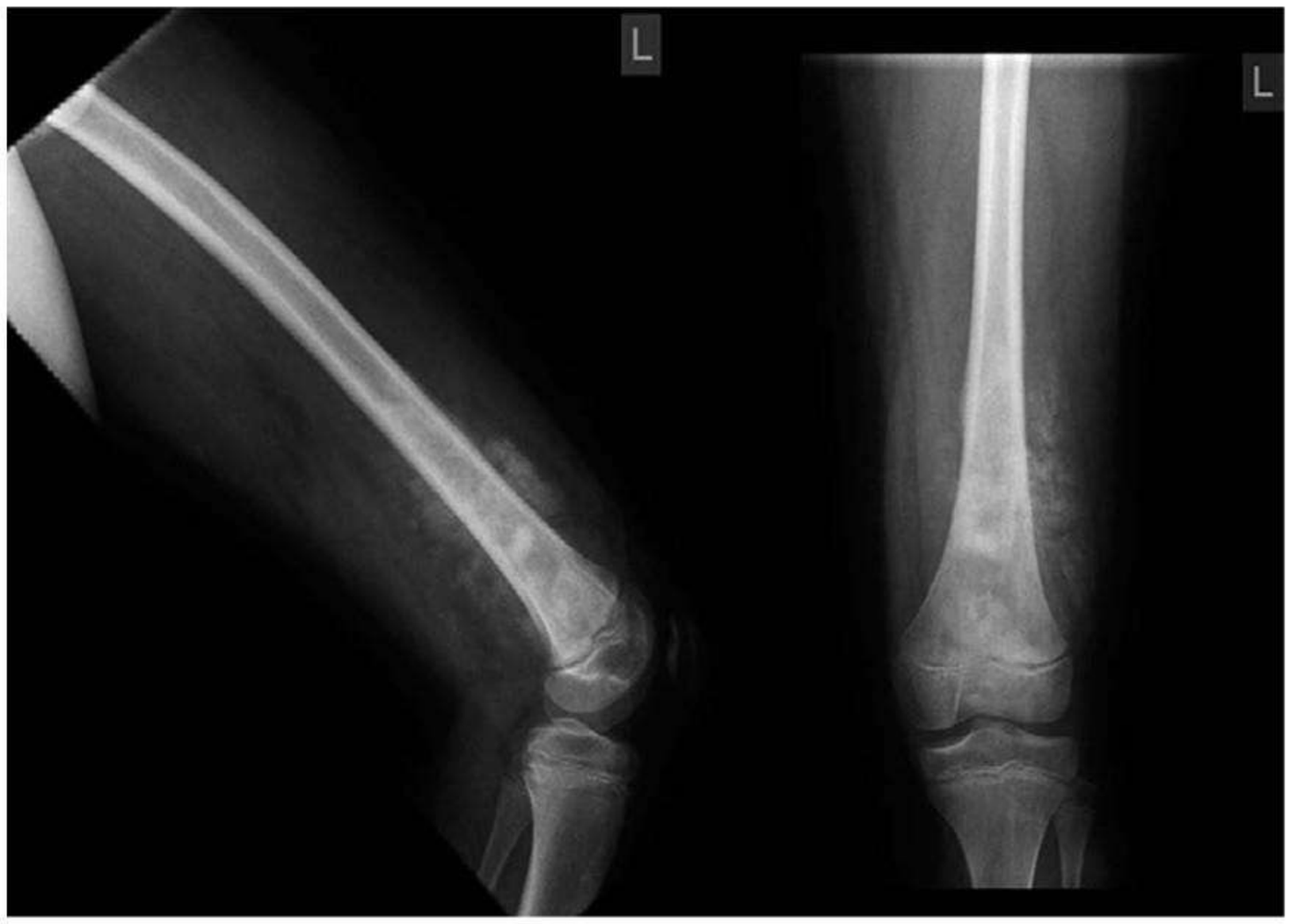


Figure 7.6 Osteosarcoma.

reaction.] There is an intramedullary sclerotic lesion with a wide zone of transition and there is extension through the cortices and into the soft tissues. There is sunray spiculation but at this resolution I can't see an obvious Codman's triangle.

EXAMINER: What's a Codman's triangle?

CANDIDATE: [*I knew what a Codman's triangle was but I did not have a clear definition at my fingertips* (*a triangle of reactive bone at the edge of the tumour where the periosteum is elevated*). *I struggled for a few seconds but managed to explain that it is indicative of a periosteal reaction.*]

EXAMINER: So what do you think the diagnosis is?

CANDIDATE: I think the diagnosis is an osteosarcoma.[1] The imaging shows an osteogenic tumour in an adolescent male. It is also in a classical position in the metaphyseal region of the distal femur, where about 35% of these tumours occur.

EXAMINER: So how would you investigate it further?

CANDIDATE: I would take a history and examine the patient. I would refer the child on to a bone tumour MDT immediately rather than delay the process by organizing more investigations locally.

EXAMINER: Okay, so you're working for the bone tumour MDT, what further investigations would you request?

CANDIDATE: I would request investigations to further delineate the tumour itself and I would arrange tests to assess for metastatic disease.

An MRI scan is the best modality for investigating the tumour itself and will delineate the local extent of the tumour, its relationship to key neurovascular structures, and the presence or absence of skip lesions. A CT scan can also be helpful as these lesions are osteogenic and therefore show up well on CT. These investigations can also be used to plan a biopsy.To stage the tumour one might initially get a chest X-ray but CT scan of the chest is mandatory to look for metastases and these are sadly found in about 30% at diagnosis. Other investigations you might use are blood tests, for example alkaline phosphatase and lactate dehydrogenase, which, if elevated, are associated with a poorer prognosis.

EXAMINER: Tell me about the general principles of treatment in cases like this.

CANDIDATE: Before commencing treatment, a confirmatory tissue diagnosis is made by biopsy and staging investigations are completed. Treatment for osteosarcoma then follows four distinct phases: neo-adjuvant chemotherapy, surgical excision and reconstruction, adjuvant chemotherapy and follow-up with clinical examination and imaging to look for recurrent disease or distant metastases.

EXAMINER: Why does the treatment start with neo-adjuvant chemotherapy? Why don't we start by excising the tumour and then start chemotherapy?

CANDIDATE: There are three main reasons for beginning treatment with neo-adjuvant chemotherapy rather than primary surgery. Firstly to treat occult micrometastases, which

are likely to be present in a much greater proportion of patients than the 30% who present with radiologically detectable metastases at diagnosis; secondly to reduce the inflammation around the primary tumour, aiding later surgical resection; and finally to allow assessment of response to the neo-adjuvant chemotherapy, determine prognosis and direct adjuvant chemotherapy.

EXAMINER: You mentioned assessment of response to neo-adjuvant chemotherapy. Why is this important?

CANDIDATE: Response of the tumour to chemotherapy treatment is measured as a percentage necrosis on histology of the resected specimen. A greater than 90% necrosis is considered a good response and this carries a better prognosis than poor or non-responders. The reason for this is that if the tumour has a good response to chemotherapy then occult, but clinically undetectable, micrometastases are more likely to be eliminated by treatment, reducing the risk of them enduring and developing into detectable metastases, and ultimately fatal disease.

EXAMINER: Do you know of any novel treatments?

CANDIDATE: I have read about muramyl-tripeptide. It is not directly tumouricidal but works by stimulating the immune system, causing macrophages to exhibit cytotoxic anti-tumour activity. In a randomized trial, Meyers *et al.* showed that, when MTP was added to the standard chemotherapy regime of cisplatin, doxorubicin and methotrexate, 6-year overall survival improved from 70% to 78%.[2] At the current time, NICE have not permitted its use for osteosarcoma, but this decision is under further discussion and appraisal.

1. Beckingsale TB, Gerrand CH. Osteosarcoma. *Orthopaed Trauma* 2010;**24**(5):321–331.
2. Meyers PA, Schwartz CL, Krailo MD *et al.* Osteosarcoma: the addition of muramyl tripeptide to chemotherapy improves overall survival – a report from the Children's Oncology Group. *J Clin Oncol* 2008;**26**:633–638.

Structured oral examination question 7: Aneurysmal bone cyst

EXAMINER: This is a 20-year-old man who presents with pain in his proximal left tibia. What do you make of his MRI scan? (Figure 7.7.)

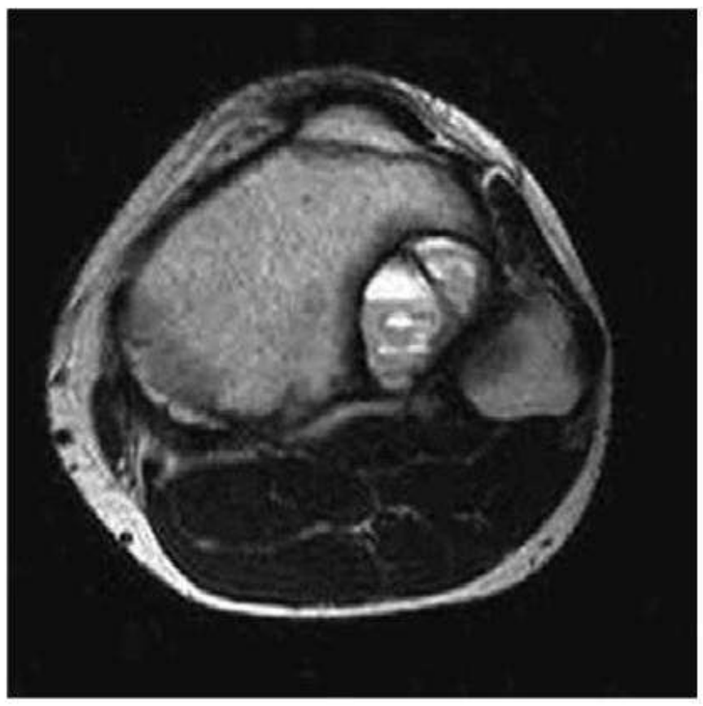


Figure 7.7 Aneurysmal bone cyst.

CANDIDATE: This is an axial T2 image, which shows a lesion in the postero-lateral tibia. It appears well circumscribed with a sclerotic margin, is eccentrically placed, and there are multiple septations and loculations with fluid levels. These appearances would be in keeping with an aneurysmal bone cyst.

EXAMINER: That's right. What's the normal management for these?

CANDIDATE: Firstly it's important to confirm the diagnosis and I would always discuss bony lesions of this type with a bone tumour MDT. Aneurysmal bone cysts can often form as a reactive change to another benign lesion, for example an osteoblastoma or giant cell tumour, which needs to be ruled out. The differential diagnosis of an aneurysmal bone cyst also includes a telangiectatic osteosarcoma, which would require very different management. In general, treatment of aneurysmal bone cysts is with curettage and grafting, but the recurrence rate can be as high as 50%.

Structured oral examination question 8: Ewing's sarcoma

EXAMINER: This is a histology slide taken from a biopsy of a tumour in the femoral diaphysis of a 16-year-old boy. What does this slide show? (Figure 7.8.)

CANDIDATE: This picture shows a magnified view of a stained histology slide. I'm no expert at histology, but I would describe the cells' appearance as small, round and blue, and given the brief history you provided I suspect this may represent a Ewing's sarcoma.[1]

EXAMINER: Excellent. What other features might this patient have presented with?

CANDIDATE: Patients usually present with pain and swelling related to the tumour. They usually present around the knee, with 25% occurring in the distal femur. Frequently, erythema, systemic pyrexia, a leukocytosis and a raised ESR are also

presenting features, which can incorrectly lead the unwary to a diagnosis of infection. Hence, it is mandatory to obtain radiographs when patients present with any unexplained pain or swelling. Patients can occasionally also present with pathological fracture through the lesion or with symptoms related to metastatic disease, such as bone pain in other sites or respiratory symptoms.

EXAMINER: And what would be the characteristic features you'd look for on an X-ray?

CANDIDATE: Ewing's sarcoma leads to a lytic, moth-eaten appearance to the bone. The classic finding, described as onion peel, is seen as a laminated periosteal reaction and probably reflects phases of tumour growth.

EXAMINER: How would you investigate this further?

CANDIDATE: I would start with a full history and examination. MRI scan is essential to delineate the local extent of the tumour and any involvement of key neurovascular structures. It can also be used to plan the biopsy. Other investigations aim to root out any evidence of metastatic disease. A CT chest is required to look for lung metastases, but, in Ewing's sarcoma, a bone scan and bone marrow biopsy are also required to look for widespread bony metastases. Distant bone marrow involvement carries a significantly poorer prognosis.

EXAMINER: In general terms, what is the management for Ewing's sarcoma?

CANDIDATE: There is a national videoconference MDT for all cases of Ewing's sarcoma, which recommends on management. In broad terms, Ewing's sarcomas are both chemo- and radio-sensitive and hence these modalities form part of the management protocol. Neo-adjuvant chemotherapy is the first-line treatment and usually precedes surgery, which involves wide excision and bony reconstruction where required. Occasionally lesions are treated solely with chemotherapy and radiotherapy, usually in surgically inaccessible lesions around the pelvis. The response to chemotherapy, like in osteosarcoma, is key to prognosis. The 5-year survival is 75% with a good response but only 20% with a poor one.

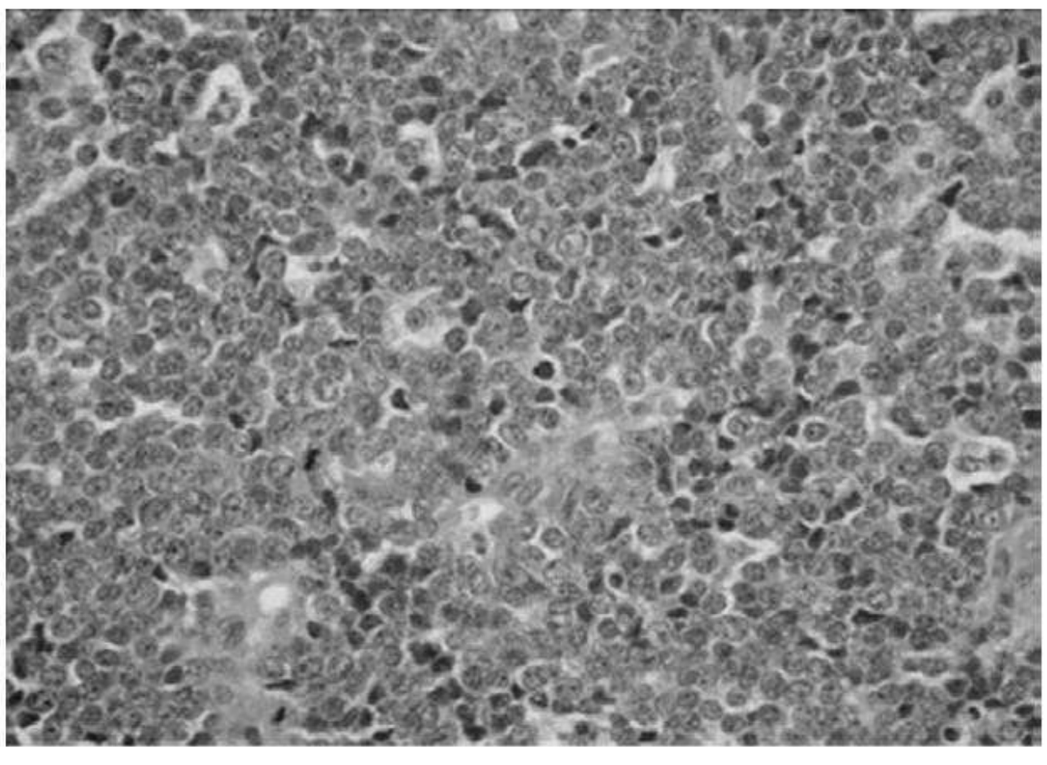


Figure 7.8 Ewing's sarcoma. See colour plate section.

1. Bullough PG. *Orthopaedic Pathology*. Fourth Edition. Edinburgh: Mosby, 2007.

Structured oral examination question 9: Lipoma

EXAMINER: This is an MRI of a patient who has presented with a painless mass on the lateral aspect of his right elbow. To orientate you the round structure (labelled A) is the radial head. Tell me about the lesion adjacent to it, which is labelled B. (Figure 7.9.)

CANDIDATE: There is an intramuscular mass in the extensor compartment adjacent, and lateral, to the radial head. The mass itself appears bland and is of the same intensity as the subcutaneous fat, suggesting a diagnosis of an intramuscular lipoma.

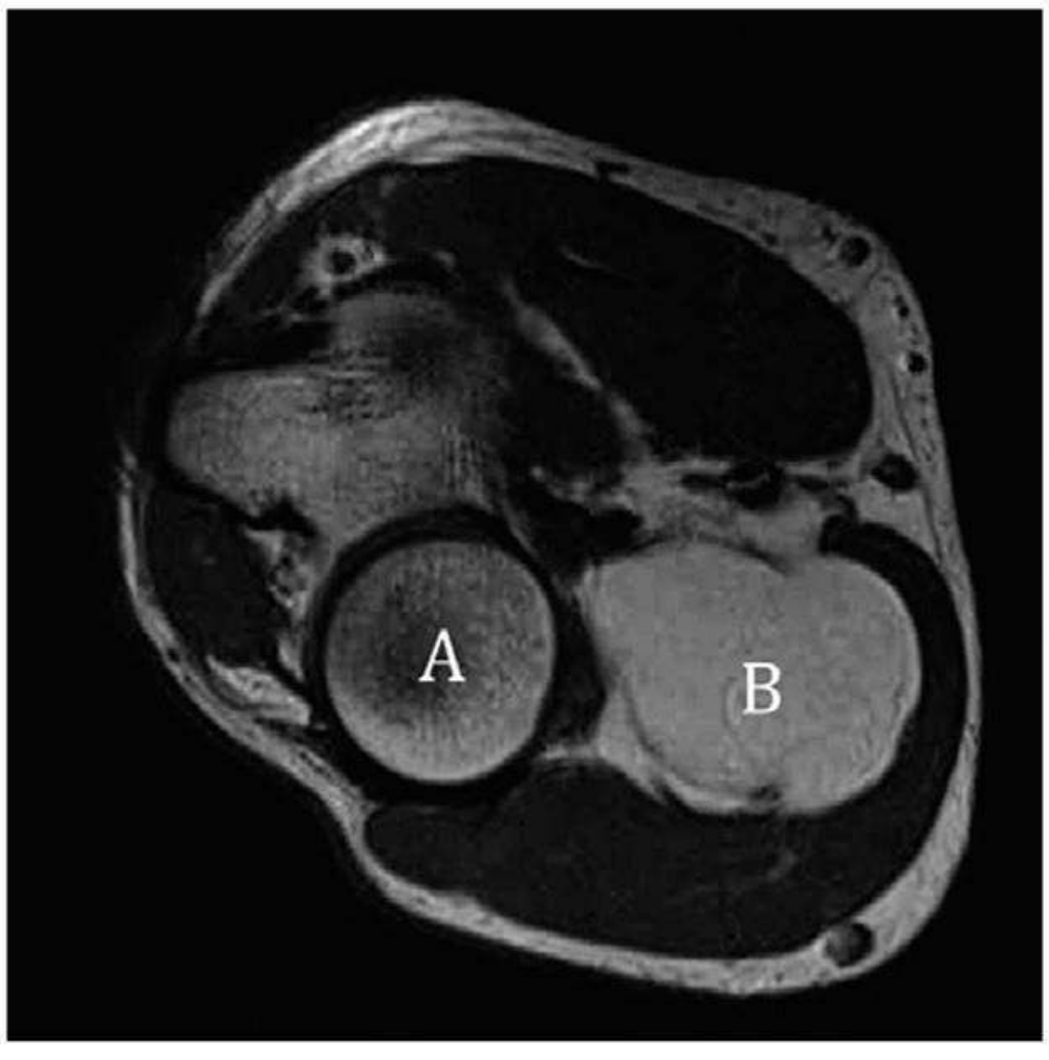


Figure 7.9 Lipoma.

EXAMINER: What is a lipoma?

CANDIDATE: A lipoma is a benign tumour of mature adipocytes, identical to the surrounding adipose tissue, and showing little variation of cell size or shape.

EXAMINER: And how would you treat this lesion?

CANDIDATE: I would want to start by taking a history and examination, in particular looking for any abnormal features like pain or distal neural compromise, which might suggest a more aggressive lesion than a simple lipoma, and alter my management. Also, this is only a single image of the lesion and I would want to see the rest of the scan and discuss it with the sarcoma MDT. Bland, innocent-looking lesions are usually treated with excision biopsy with a marginal margin. If there is any doubt then a biopsy should be taken prior to excision. Histology of lesions below the fascia, like this one, often come back labelled as atypical lipomas by the histologist, despite very bland appearance on MRI.

EXAMINER: What do you mean by an atypical lipoma?

CANDIDATE: The term is quite controversial and the literature often refers to them as lipoma-like liposarcomas. In essence, an atypical lipoma is a lipoma with some slightly atypical features but no evidence of malignancy. The histology of such lesions shows variation of adipocyte size, in contrast to the bland adipocytes of a simple lipoma, and nuclear atypia, as well as the presence of lipoblasts. These lesions are benign and management is still with marginal excision but they do have a low rate of local recurrence.[1]

1. Beckingsale TB, Gerrand CH. The management of soft-tissue sarcomas. *Orthopaed Trauma* 2009; **23**(4):240–247.

11.Approaches & Techniques

Shoulder

Deltopectoral : FRCS
Posterior shoulder : FRCS
Lateral :FRCS
Scope: OB

Humerus

Anterior humerus: FRCS
Post humerus : FRCS
Distal humerus Anteriolateral :FRCS
Lateral distal humerus: OB

Elbow

Posterior : OB
Kaplan : OB
Kocher : FRCS
Anterior :FRCS
Anteriolateral: FRCS

Hand & Forearm

Scaphoid: AO
Dorsal wrist : OB
FCR: OB
Henery : FRCS
Thompson : FRCS
Ulna: FRCS

Hip

Medial: OB
Anterior: OB +- AO
Anteriolateral: OB
Lateral: OB
Posterior: OB
THA: AO
Bipolar: AO

Femur

Lateral approach (split vastus lateralis) :OB
Posterio lateral,Lateral Distal femur(subvastus): AO

Distal femur Ant Lateral: OB
Posterior approach: OBv
Nail: AO

Knee

Arthroscopy : OB
TKR : OB + Belal
Anter lateral : FRCS + AO
Posterior: FRCS
Post medial : AO
Medila /Post medial : AO
Hamstring harvest: FRCS

Leg:

Tibia anter: OB
Fibula lateral: OB
Tibia Nail : AO

Ankle :

Posterio laterl: AO - OB
Lateral: AO - OB
Ant lateral: AO - OB
Ant: OB
Ant medial: AO
Medial: AO
Post med: AO

Calcaneal: AO + OB

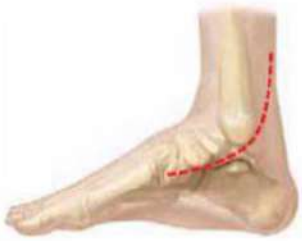
Posteromedial release for Club foot :

TECHNIQUES

INCISION

Turco Posteromedial Incision

- The Turco incision allows for access to the medial and posterior portions of the foot (TECH FIG 1).
- The technique begins with a medial incision at the first metatarso-medial cuneiform joint.
- The cut is extended proximally until it is just distal to the tip of the medial malleolus.
- Care is taken to curve the incision in a vertical direction, up the calf to expose the Achilles tendon.
- To reach the lateral side, the subtalar joint must be opened like a book, or a separate lateral incision must be made.



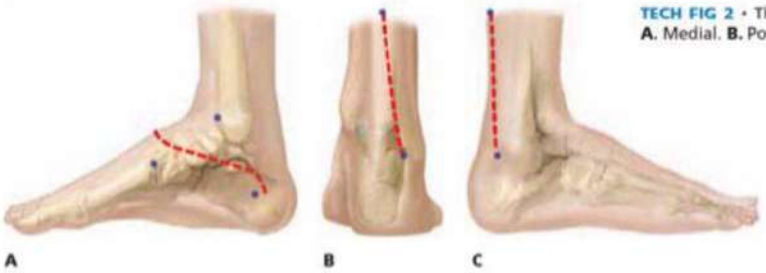
TECH FIG 1 • The medial Turco incision.

Carroll Medial and Posterolateral Incisions

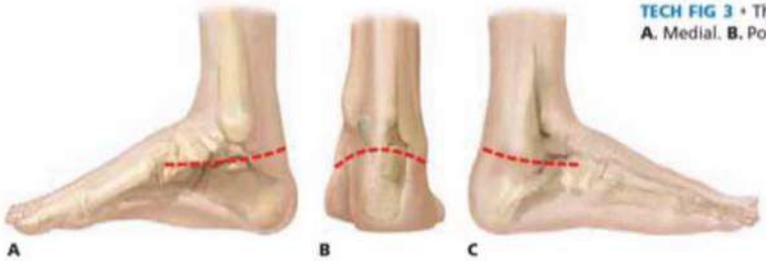
- The Carroll types of incisions allow medial or more posterolateral access (TECH FIG 2).
- For the medial incision, a triangle is cut that is demarcated by the center of the os calcis, the front of the medial malleolus, and the base of the first metatarsal.
- The incision is made parallel with the base of the triangle, then curved proximal-plantar, and then curved distally over the dorsum of the foot.
- For the posterolateral incision, an oblique incision is created that runs from the midline of the distal, posterior calf to a point between the tendo Achilles and the lateral malleolus.
- A lateral incision may be required to reach the lateral talonavicular joint.

Cincinnati Incision (Author's Preferred Incision)

- The Cincinnati incision provides the most extensive access to the foot, including medial, posterior, and lateral access (TECH FIG 3).
- The incision begins medially over the talonavicular joint, extending posteriorly at the level of the subtalar joint. It is continued distally to the talonavicular joint laterally and may be extended distally on both the medial and lateral sides.
- The Cincinnati incision is most easily performed with the patient prone. Flexing the knee provides excellent access to the Achilles tendon for Z-lengthening.
- For severely deformed feet (equinus), closure may be difficult.



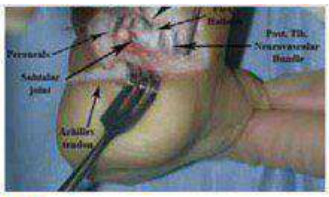
TECH FIG 2 • The Carroll incision. **A.** Medial. **B.** Posterior. **C.** Lateral.



TECH FIG 3 • The Cincinnati incision. **A.** Medial. **B.** Posterior. **C.** Lateral.

reduce the varus force. Fibrotic bands and tendon sheath should also be released.

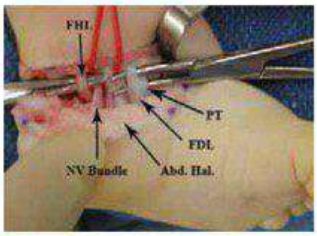
- If the Achilles lengthening is not sufficient to restore the anatomy, the posterior aspects of the subtalar and ankle joints are sequentially released.
- The first step is to identify and protect the sural nerve and vessels laterally and the posterior tibial neurovascular bundle medially. The flexor hallucis is then identified posteromedially and protected. The peroneal tendons are also identified and protected (TECH FIG 4).
- The ankle capsule is noted and incised from the posteromedial to the posterolateral corners to allow dorsiflexion of the talus in the mortise.



TECH FIG 4 • Posterior portion of the Cincinnati incision. The Achilles tendon has been cut for lengthening and retracted. (Copyright Richard S. Davidson.)

MEDIAL SOFT TISSUE RELEASE

- Medial release is undertaken if the posterior release as described above does not correct the anatomy.
- First, the posterior portion of the Cincinnati incision is extended medially to the medial aspect of the navicular.
- The posterior tibial neurovascular bundle is protected while releasing any thickened fascia as well as the flexor hallucis, which may have been lengthened through the posterior part of the incision.
- The posterior tibial tendon is located just distal to the flexor digitorum tendon and is lengthened in notch fashion as necessary.
- The abductor hallucis muscle is lengthened proximally or distally. The flexor digitorum tendon is identified just anterior to the posterior tibial neurovascular bundle and lengthened in notch fashion as necessary (TECH FIG 5).
- Deciding whether to lengthen the anterior tibialis tendon can be difficult. If the anterior tibialis tendon appears contracted on anatomic correction, it should be lengthened in a Z-lengthening. Occasionally, the anterior tibialis tendon remains overactive and will need to be lengthened at a future time.
- A helpful hint for the lengthening of the tendons on the medial side of the foot: Each of the ends of the lengthened tendons should be tagged with suture, which is then held in a color-coded bulldog clamp. Each group of the proximal and distal sets of clamps can then be held in proper order by a safety pin. This will avoid confusion when it is time to repair the tendons after anatomic realignment of the foot is accomplished.
- Release of the plantar fascia has been recommended in the past but is currently avoided since it can contribute to later pes planovalgus. Do not release the plantar fascia in cases of rocker-bottom deformity during the casting.
- Care should be taken to avoid injury to the medial plantar vessels and nerve.
- If lengthening of these tendons does not permit anatomic alignment, follow this addendum:
  - Identify and release the talonavicular joint. The navicular is medially displaced on the talar head, making



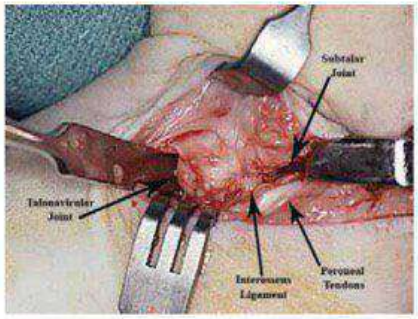
TECH FIG 5 • Medial portion of Cincinnati incision, superficial. (Copyright Richard S. Davidson.)

the talonavicular joint obliquely, rather than transversely, oriented.

- Follow the distal stump of the notch-lengthened posterior tibial tendon to its insertion on the navicular.
- The capsule is released medially, plantarly, and dorsally and as far laterally as can be reached safely. Be careful not to cut the talar neck, as this may lead to avascular necrosis or growth disturbances!
- Release the subtalar capsule from the talonavicular joint to the interosseous ligament medially, including the spring ligament. Be careful not to damage the deep deltoid ligament. A Freer elevator placed into the ankle joint posteriorly can help identify the ankle and subtalar joints.
- Reach the medial aspect of the calcaneocuboid joint by carefully dissecting the soft tissues from the plantar aspect of the talar neck. Release of this capsule will allow a wedge opening of the calcaneocuboid joint to straighten the lateral column. Another landmark to the calcaneocuboid joint from the medial side of the foot is the peroneus longus tendon crossing from lateral to plantar.
- Many authors have described release of the interosseous ligament through this incision. It is important to preserve this ligament as a pivot axis and to preserve its associated blood supply to the talus.

LATERAL SOFT TISSUE RELEASE

- A problem often occurs when the calcaneus has rotated under the talus on the interosseous membrane and is tethered by a stiff, fibrotic lateral capsule. If the above posterior and medial releases do not permit anatomic alignment, a lateral release may be needed.
- The posterior portion of the Cincinnati incision, made for the posterior release, is extended laterally at the level of the subtalar joint to the talonavicular joint.
- The extensor digitorum brevis is identified over the sinus tarsi. Its plantar edge is divided from the lateral calcaneus and the muscle is elevated to expose the sinus tarsi and neck and head of the talus.
- The lateral capsule of the talonavicular joint is exposed and released. A circumferential release of the talonavicular joint is thus completed (TECH FIG 6).
- The beak of the calcaneus is then palpated. From the lateral aspect of the talonavicular joint, cut the lateral subtalar capsule, between the beak of the calcaneus and the talar neck, proximally to the interosseous ligament, completing circumferential release of the subtalar capsule.



TECH FIG 6 • Lateral portion of the Cincinnati incision, showing the lateral structures. (Copyright Richard S. Davidson.)

12. Implants

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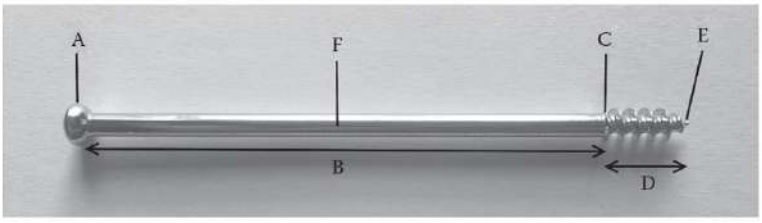
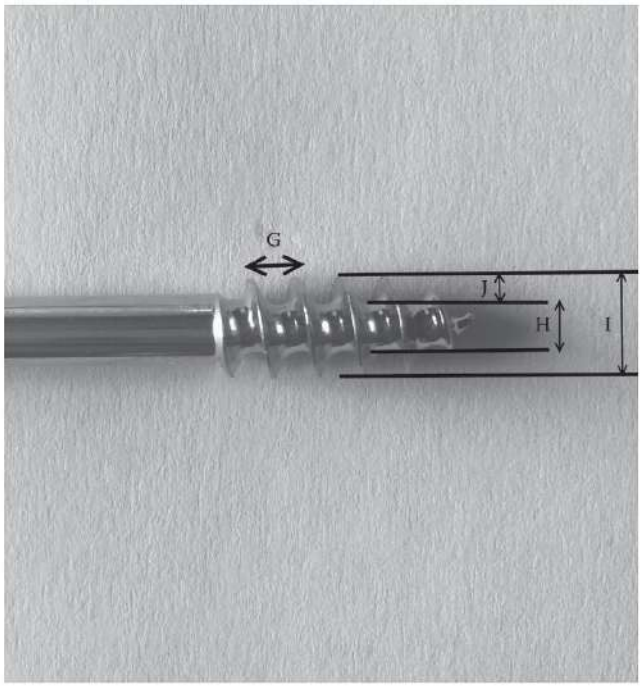
SCREWS





1. **What is a screw and what is the function of a screw?**
   A screw is a mechanical device which converts a rotational force (torque) into a linear movement. Its function is to connect two or more objects by compressing them together.

2. **What are the names of the different parts labelled above?**

   - A – Head
   - B – Shank
   - C – Run out (this is the area where screws tend to break)

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- D – Thread
- E – Tip
- F – Shaft
- G – Pitch
- H – Core diameter/minor diameter
- I – Thread diameter/major diameter
- J – Thread depth

3. Can you tell me the function of the following parts of a screw?

- Head – This provides an area for attachment of a screwdriver and is designed to prevent slip and to improve directional control. It also acts to prevent forward motion of the screw when it is fully seated against bone. Hexagonal head recess design is the most common.
- Flutes – These are present in self-tapping screws and they provide a route for the removal of swarf (bone debris).
- Pitch – This is the distance between threads. It is the same as the distance advanced for every single (360 degrees) turn of the screw.
- Core – The size of the core determines the strength of screw and its fatigue resistance. The size of the drill bit used is equal to the core diameter.
- Threads – Thread depth is half of the difference between thread diameter and core diameter. The thread depth determines the amount of contact with the bones, which in turn determines the resistance to pull out.

4. What is the difference between tensile strength and pullout strength in relation to screws?

The tensile strength of a screw is its resistance to breaking. This is proportional to the core diameter squared.

The pullout strength of a screw depends on the outer diameter of the threads and the area of the threads in contact with the bone. This is the effective thread depth (J in the picture above) and this is proportional to the pullout strength.

5. What type of screw is pictured here?

This is a partially threaded, cancellous screw.

6. How does the pictured screw differ from a locking bolt used with an intramedullary nail?

A locking bolt has superior rotational stability to a screw owing to its wide core diameter. It is particularly useful in osteoporotic bone of the distal femur to prevent the 'broomstick in a trashcan' phenomenon.

7. What drill sizes are required to insert a small fragment cortical screw and a large fragment cortical screw in order for them to act as lag screws?

A lag screw requires two different drill sizes in order to create a gliding hole (near cortex) and a threaded hole (far cortex) as per AO techniques.

A small fragment screw has a major diameter/thread diameter of 3.5 mm and a minor diameter/core diameter of 2.5 mm. This therefore requires:

- 3.5 mm drill bit (silver) for the gliding hole
- 2.5 mm drill bit (gold) for the threaded hole

A large fragment screw has a major diameter/thread diameter of 4.5 mm and a minor diameter/core diameter of 3.2 mm. This therefore requires:

- 4.5 mm drill bit for the gliding hole
- 3.2 mm drill bit for the threaded hole

AO teaching suggests using a standard screw (i.e. not self-tapping) when inserting a lag screw as self-tapping screws can readily angle incorrectly and they will cut a new path and destroy already cut thread. The taps that are used are the same size as the drill bit for the gliding hole.

8. What is the difference between a cortical screw and a cancellous screw?

Cortical screws:

- Smaller pitch
- Greater number of threads
- Thread diameter to core diameter ratio is less
- Designed to have better purchase in cortical bone
- Fully threaded
- Blunt tip

Cancellous screws:

- Greater thread depth
- Larger pitch
- Thread diameter to core diameter ratio is greater
- Designed to have better purchase in the cancellous bone
- Fully or partially threaded
- Corkscrew tip

9. What is the working length of a screw?

This is the length of bone traversed by a screw. In very osteoporotic bones, which typically present a thin cortex or a bone segment under high torsional loading, the use of bicortical screws is mandatory to enhance the working length of the screws, which in turn increases their torsional stiffness. (NB: Despite similar terminology which can lead to confusion, this is an entirely different concept from working length of a nail.)

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PLATES

1. **How is plate strength determined?**
   Plate strength is defined by the formula BH^3. B is the width; H is height (or thickness). Therefore, the rigidity (bending stiffness) of the plate is proportional to the thickness of the plate to the power of 3.

2. **What different functions can a plate provide?**

   - Bridging: In a comminuted fracture, the plate can bridge the fragment to allow restoration of length, rotation and alignment.
   - Buttress: Generally used in the periarticular region. When placed at the apex of the fracture, they prevent displacement of the fracture by shear forces.
   - Compression: This can be achieved in several manners:
     1. A lag screw through the plate.
     2. An eccentrically placed screw through the hole of a dynamic compression plate (DCP). There is a potential of 1.8 mm of glide when two holes are compressed, producing up to 600N of compression.
     3. An external compression device, for example, a Verbrugge clamp or AO articulated compression device.
     4. Overbending the plate so there is a small gap between the plate and the bone at the level of the fracture will achieve compression of both the near and far cortex and produce absolute stability.
   - Neutralisation: This protects a lag screw from torsional, shear and bending forces.
   - Tension band: A plate may be placed on the tension side of the bone to act as a tension band. When the bone is loaded, the plate converts tension into compression at the far cortex.

3. **What type of bone healing will occur when a lag screw and neutralisation plate has been used?**
   Where there has been anatomical reduction and interfragmentary compression, this can achieve absolute stability (no motion between fracture surfaces under functional load). This will lead to direct bone healing (also known as primary bone healing) if the gap between bony fragments is less than 0.01 mm and interfragmentary strain is less than 2%.

   Under these conditions, cutting cones are formed at the ends of the osteons closest to the fracture site. The tips of the cutting cones consist of osteoclasts, which cross the fracture line, generating longitudinal cavities at a rate of 50–100 μm/day.

   These cavities are filled with blood vessels and osteoblasts, which lay down lamellar bone in the form of new osteons. This process may take many months and is difficult to see on a radiograph due to the lack of callus formation.

4. **What type of bone healing will occur when a plate is used as a bridging plate?**
   When a plate is used as a bridging plate, bone healing will take the form of indirect (secondary) bone healing. This same form of bone healing occurs with cast treatment, IM nails and external fixation. There are four stages.

Stage 1 – Haematoma and Inflammation – Week 1

Haematoma from the ruptured blood vessels forms a fibrin clot. The clotting cascade and complement system are both activated. Macrophages, neutrophils and platelets release several cytokines, including PDGF, TNF-Alpha, TGF-Beta, IL-1, 6, 10, and 12.

There is angiogenesis and recruitment of fibroblasts, mesenchymal cells and osteoprogenitor cells as the haematoma is replaced by granulation tissue, which can tolerate the greatest strain before failure. Necrotic bone ends are resorbed by osteoclasts and other devitalised tissue is removed by macrophages.

Stage 2 – Soft Callus – Weeks 2–4

The granulation tissue formed during stage 1 is replaced by fibrous tissue due to the action of fibroblasts and chondroblasts which lay down cartilage (type II collagen). The mechanical environment drives differentiation of either osteoblastic (stable environment) or chondryocytic (unstable environment) lineages of cells. Cartilage production provides provisional stabilisation.

Stage 3 – Hard Callus – 1–4 months

Soft callus is resorbed by chondroclasts and osteoblasts produce osteoid, which is then mineralised to form woven bone (hard callus). The conversion of soft callus to hard callus is called endochondral ossification.

Stage 4 – Remodelling – Up to several years

Once the fracture has united, the hard callus (woven bone) is replaced with hard, dense lamellar bone by a process of osteoclastic resorption followed by osteoblastic bone formation. This is the same process seen during routine skeletal turnover. The bone assumes a configuration and shape based on stresses acting upon it (Wolff's law).

Electric fields may play a role in Wolff's law. The compression side is electronegative and stimulates osteoblast formation; the tension side is electropositive and simulates osteoclasts.

NAILS AND EXTERNAL FIXATORS

1. **How do IM nails and external fixators differ with regard to load bearing and their moment arm?**
   This depends on the fracture type. For AO type A and B fractures with cortical contact, the IM nail or external fixator is load sharing as there is some cortical contact between the main fracture fragments. In a type C fracture, the nail or external fixator is load bearing.

   Although a plate has a short moment arm when fixed to bone, an IM nail is placed down the centre of the intramedullary canal, such that the anatomical axis of the bone is collinear to the long axis of the IM nail. This results in a negligible moment arm and therefore increased stability. An external fixator has the longest moment arm of all three devices given its distance away from the bone.

2. **What different designs of IM nail do you know and what effect do these differences have on performance and stiffness?**
   IM nails come in many different designs. They can be cannulated or solid, slotted or non-slotted, cylindrical or clover leaf-shaped. Cannulated nails are less stiff than solid nails, but can be inserted over a guide wire and allow for deformation during insertion, which makes them less likely to lead to a blow out. Also, micro-motion at the fracture site will encourage secondary/indirect bone healing, which is how fractures heal when treated with IM nails.

   Slotted nails decrease the torsional stiffness as well as bending stiffness and are rarely used these days. Clover leaf-shaped nails are of increased stiffness compared to cylindrical nails.

3. **What is the working length of an intramedullary nail and what is the importance of working length?**
   The working length is the length of nail between the most distal point of fixation in the proximal fragment and the most proximal point of fixation in the distal fragment. In other words, it is the unsupported portion of the nail between the bone fragments.

   Torsional stiffness is inversely proportional to working length. Bending stiffness is inversely proportional to the working length to the power of 2.

4. **How does the radius of the nail affect rigidity?**
   For a solid nail, the rigidity/stiffness (to both bending and torsion) is proportional to the radius to the power of 4. For a cannulated nail, rigidity/stiffness is proportional to the radius to the power of 3.

5. **What is the difference between a reamed and an unreamed nail?**
   There are many differences between reamed and unreamed nails.

   Biomechanically, reaming can have several theoretical advantages. It can decrease the working length of the nail in addition to allowing for the insertion of a larger diameter nail. The result of both of these is an increase in the rigidity/stiffness of the construct.

   Unreamed nails theoretically minimise disruption of the endosteal blood supply. However, with reamed nails, there is a six-fold increase in the periosteal blood supply and

the direction of the blood flow can reverse from centrifugal to centripetal. Furthermore, there is reconstitution of the endosteal blood supply after 6 weeks. More importantly, the clinical relevance of reaming is such that reamed nails have been shown to have a positive effect on bone union in terms of both rates of union and time to union. This is particularly true in femoral fractures (both open and closed) and closed tibial fractures. The benefits of reaming appear to be less in open fractures of the tibia in clinical trials.

Finally, there is no good evidence to suggest an increased rate of compartment syndrome or pulmonary complications, for example, fat emboli (which occurs with both techniques), when using a reamed nail over an unreamed nail.

6. What are the indications for an external fixator?

I – Temporary fixation
- Damage control orthopaedics
- Periarticular fractures: 'Span – scan – plan'
- Pelvic ring injuries (rarely used these days due to pelvic binders)

II – Definitive treatment
- Significant soft tissue injuries with associated fractures
- Paediatric injuries (since they heal faster than adult injuries and the frame is less bothersome to a child)

III – Reconstruction
- Deformity
- Infection
- Non-union
- Lengthening

7. How does one increase the stiffness of an external fixator?

The most important aspect to an external fixator for increasing stiffness (other than good cortical opposition at the fracture site) is the diameter of pins. The bending stiffness of pins is proportional to the radius of the pins to the power of 4. However, as a rule, no pin should be greater than one-third of the diameter of the bone due to the risk of fracture. Other techniques to increase stiffness include:

- Pins in different planes (circular > multiplanar > uniplanar)
- Increasing the number of pins
- Increasing the space between pins
- Placement of pins near fracture site
- Decreasing the distance of rods from bone (this reduces the moment arm)
- Increasing the diameter of rods
- Increasing the number of rods

FURTHER READING

Bhandari M, Guyatt GH, Tong D et al. Reamed versus nonreamed intramedullary nailing of lower extremity long bone fractures: A systematic overview and meta-analysis. *J Orthop Trauma.* 2000 Jan;14(1):2–9.

Reichert IL, McCarthy ID, Hughes SP. The acute vascular response to intramedullary reaming. Microsphere estimation of blood flow in the intact ovine tibia. *J Bone Joint Surg Br.* 1995 May;77(3):490–493.

Names of the different screw functions

- Lag/interfragmentary compression screw
- Compression screw
- Position screw
- Locking head screw
- Buttress/antiglide screw
- Anchor screw
- Push-pull screw
- Reduction screw
- Poller screw

24

Small Fragment Set

Screw Type	Cortical	Cortical	Cancellous	Locking	Locking
Thread diameter	2.7	3.5	4.0	2.7	3.5
Drill	2.0	2.5	2.5	2.0	2.8
Tap	2.7	3.5	4.0	Self tap	Self tap

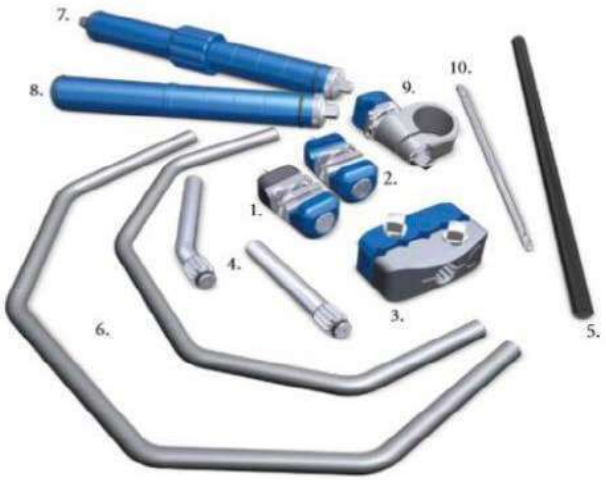
Large Fragment Set

Screw Type	Cortical	Cortical	Cancellous	Locking	Locking
Thread diameter	4.5	5.5	6.5	4.0	5.0
Drill	3.2	4.0	3.2	3.2	4.3
Tap	4.5	5.5	6.5	Self tap	Self tap

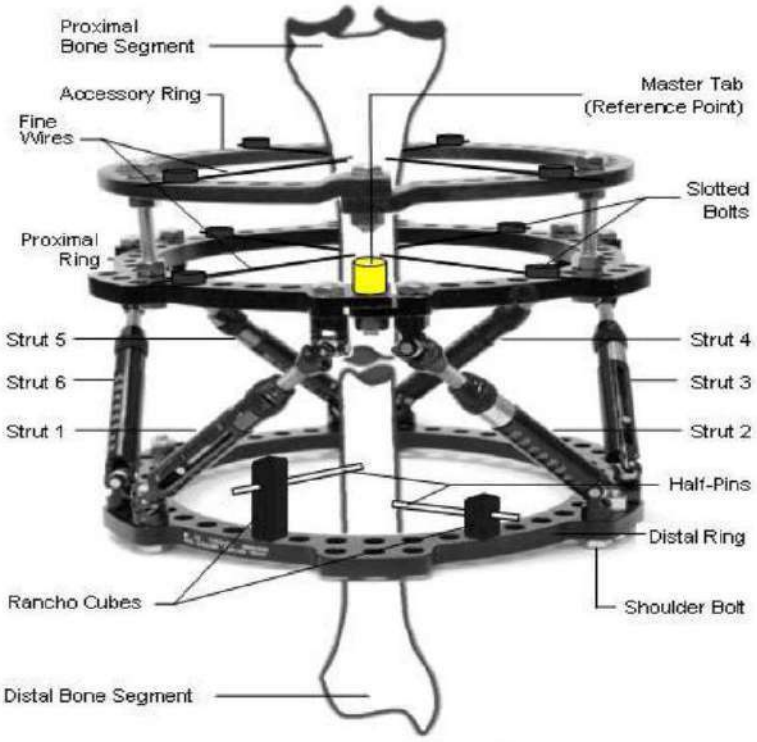
Six step lag screw technique

Using 4.5 mm cortex screw:

- Drill 4.5 mm gliding hole
- Drill 3.2 mm threaded hole
- Countersink
- Measure depth
- Tap
- Screw

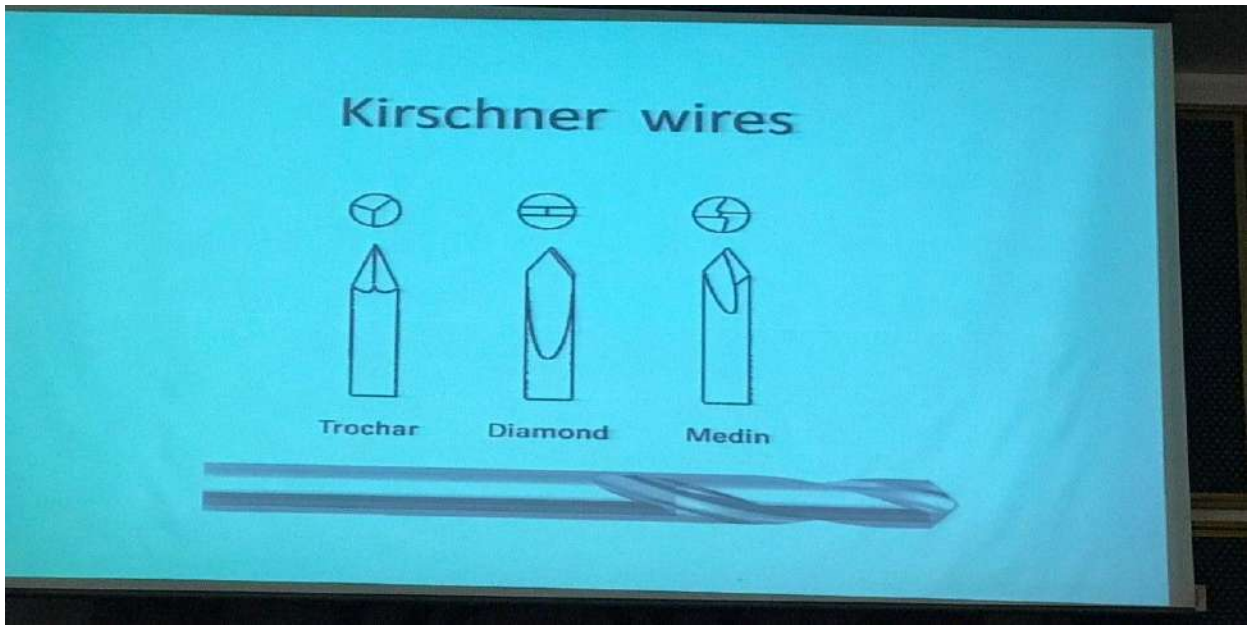


1. Pin to Rod Coupling
2. Rod to Rod Coupling
3. 5-Hole Pin Clamp
4. Straight Post / 30° Angled Post
5. ø8mm Connecting Rods
6. Semi-Circular Aluminum Connecting Rods
7. Dynamization Tube
8. Compression/Distraction Tube
9. Tube to Rod Coupling
10. Apex® Self-Drilling Pins



External fixators

- Factors that increase stability of conventional external fixators
  - contact of ends of fracture
  - larger diameter pins (most important)
  - additional pins
  - decreased bone to rod distance
  - pins in different planes
  - increasing size or stacking rods
  - rods in different planes
  - increased spacing between pins
- Factors that increase stability of circular (Ilizarov) external fixators
  - larger diameter wires
  - decreased ring diameter
  - olive wires
  - extra wires
  - wires cross perpendicular to each other
  - increased wire tension
  - placement of two central rings close to fracture
  - increased number of rings


Kirschner wires
Trochar
Diamond
Medin

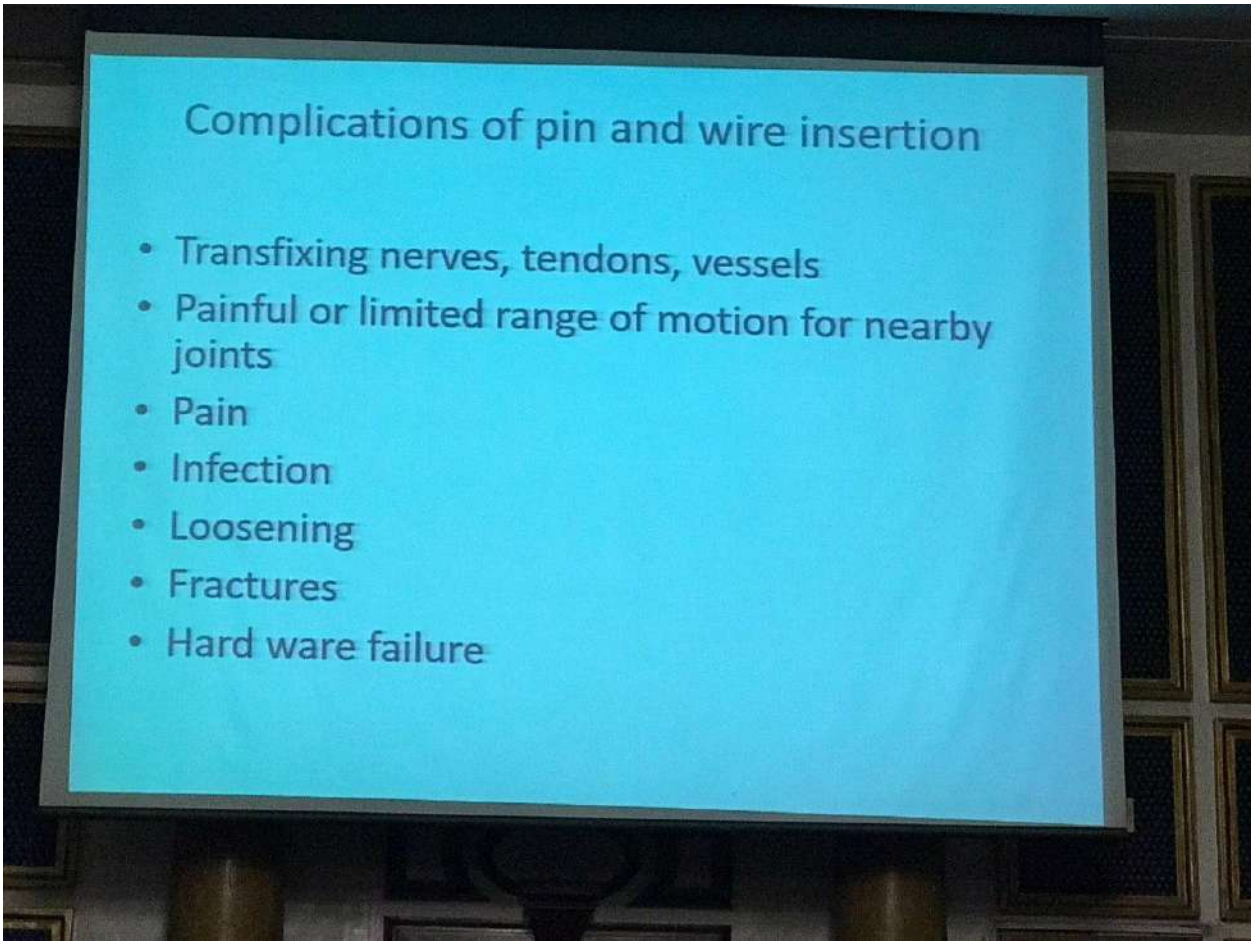

Complications of pin and wire insertion
• Transfixing nerves, tendons, vessels
• Painful or limited range of motion for nearby joints
• Pain
• Infection
• Loosening
• Fractures
• Hard ware failure

Table 2

Pin-tract Infection Classification and Treatment[40]

Grade	Appearance	Treatment
1	Slight erythema, little discharge	Improved pin care
2	Erythema, discharge, and pain in soft tissue	Topical and/or oral antibiotics
3	Grade 2 but no improvement with antibiotics	Remove pin and change antibiotic regimen
4	Soft-tissue infection involving several pins	Remove any loose pins
5	Grade 4 and radiographic evidence of bone involvement	Remove entire fixator construct and curettage pin tract
6	Infection after fixator removal (clinical and radiographic)	Débridement, irrigation, and systemic antibiotics

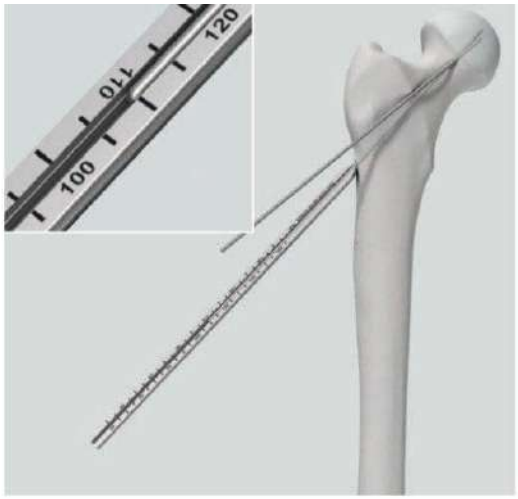
Determine length of DHS Screw / DHS Blade

Instrument	
338.050	DHS/DCS Direct Measuring Device

Read the length of the DHS Screw or Blade directly off the guide wire with the measuring device.

If the guide wire is inserted into the subchondral bone remove 10 mm from the measurement.

Example: If you read 110 mm on the direct measuring device, the measured length of the implant is 100 mm.



8

Ream for insertion of DHS Screw / DHS Blade

A Instruments for DHS Screw	
338.130	DHS Triple Reamer, complete
Consisting of:	
338.100	Drill Bit ∅ 8.0 mm
338.110	DHS Reamer
338.120	Nut, knurled

Alternative instrument for short barrel plates (for DHS screw/blade ≤ 75 mm)

338.440	DHS Reamer

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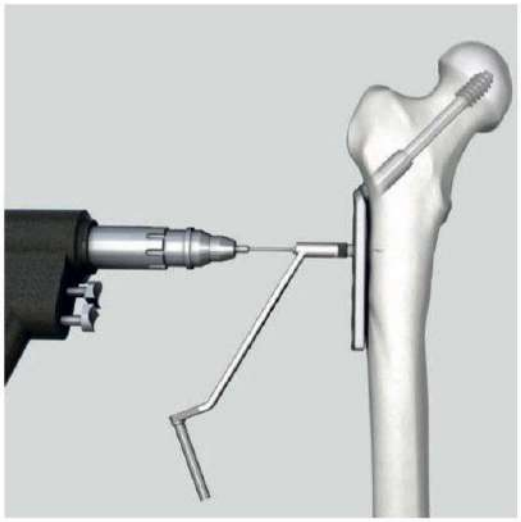
Fix the DHS plate onto the shaft

Remove all the insertion instruments and the guide wire. Discard the guide wire. Then fix the plate to the femoral shaft.

A Cortex screws for the conventional DHS plate

Instruments	
323.460	Universal Drill Guide 4.5/3.2
310.310	Drill Bit ∅ 3.2 mm
319.010	Depth Gauge
314.150	Screwdriver Shaft, hexagonal

Use the drill guide and the drill bit to drill holes in a neutral position through the plate holes. Insert self-tapping 4.5 mm cortex screws of appropriate length.



Stainless Steel	Titanium	Length
251.50	251.50	50mm
251.55	251.55	55mm
251.60	251.60	60mm
251.65	251.65	65mm
251.70	251.70	70mm
251.75	251.75	75mm
251.80	251.80	80mm
251.85	251.85	85mm
251.90	251.90	90mm
251.95	251.95	95mm
251.100	251.100	100mm
251.105	251.105	105mm
251.110	251.110	110mm
251.115	251.115	115mm
251.120	251.120	120mm

Thread Diameter 12.5mm
Thread Length 22.0mm
Shaft Diameter 8.0mm

Length : 50-145 mm

252 DHS Plate (DC Hole) 38mm Barrel

With DC hole dynamic compression holes and slot for tension device. Fixation with 4.5mm cortex screw. For Pertrochanteric and Intertrochanteric fractures. (Barrel length 38mm(Long Barrel) use with DHS Triple Reamer)
Use with DHS/DCS Lag Screw Code No.251.

Nos. Of Hole	Length In MM	125°Code No.252A	130°Code No.252B	135°Code No.252C	140°Code No.252D
2hole	46mm	-----	252B.02	252C.02	-----
3hole	62mm	-----	252B.03	252C.03	-----
4hole	78mm	252A.04	252B.04	252C.04	252D.04
5hole	94mm	252A.05	252B.05	252C.05	252D.05
6hole	110mm	252A.06	252B.06	252C.06	252D.06
7hole	126mm	252A.07	252B.07	252C.07	252D.07
8hole	142mm	252A.08	252B.08	252C.08	252D.08
9hole	158mm	-----	252B.09	252C.09	-----
10hole	174mm	252A.10	252B.10	252C.10	-----
11hole	190mm	-----	252B.11	252C.11	-----
12hole	206mm	-----	252B.12	252C.12	-----
13hole	222mm	-----	-----	252C.13	-----
14hole	238mm	-----	252B.14	252C.14	-----
16hole	270mm	-----	252B.16	252C.16	-----
18hole	302mm	-----	-----	252C.18	-----

Plate Thickness	5.8mm
Width	19.0mm
Hole Spacing	16.0mm
Barrel Diameter	12.6mm
Barrel Length	38.0mm

Angle : 130-150

Short barrel= 25mm

Compression Screw (For DHS/DCS Lag Screw)

Use with DHS/DCS Lag Screw Code No.251

Thread Diameter	4.0mm
Total Length	36.0mm
Hexagonal Socket	3.5mm

255 95° DCS Plate (DC Hole)

With DC hole dynamic compression holes and slot for tension device. Fixation with 4.5mm cortex screw. For Condylar f
avoids further damage to the bone(Barrel length 25mm use with DCS Triple Reamer)
Use with DHS/DCS Lag Screw Code No.251

Stainless Steel	Total Holes	Length
255.04	4hole	82mm
255.05	5hole	98mm
255.06	6hole	114mm
255.07	7hole	130mm
255.08	8hole	146mm
255.09	9hole	162mm
255.10	10hole	178mm
255.11	11hole	194mm
255.12	12hole	210mm
255.13	13hole	226mm
244.14	14hole	242mm
255.16	16hole	274mm
255.18	18hole	300mm
255.20	20hole	338mm

Plate Thickness	5.8mm
Width	19.0mm
Hole Spacing	16.0mm
Barrel Diameter	12.6mm
Barrel Lengtth	25.0mm

1. BEARING SURFACES

Shown ceramic cup and asked to talk.what are the different bearing surfaces. Your choice in different age groups in THR

Metal on Polyethylene

Advantages

1. Tried and tested articulation with widely published evidence-base
2. Cheap
3. Easy to manufacture

Disadvantages

1. Polyethylene induced osteolysis (manufacturing process encouraging cross-linking to combat this problem with polyethylene)

Ceramic on Ceramic

Advantages

1. High wear resistance – High Young's modulus of elasticity (Alumina 380GPa); Hard; Wettability
2. Biocompatibility; Biostability

Disadvantages

1. Brittle – concerns regarding fracture but with improved manufacturing process (increased purity, density, improvement in size and distribution of grains, accurate Morse taper) fracture rate in region of 0.02%
2. Squeaking (high pitched audible sound during hip movements) – often asymptomatic; multifactorial including component malpositioning, edge loading, impingement, third-body particles

3. Striped wear – impingement of the partially distracted head on rim of socket during gait
*(2) & (3) also reported in Metal on Metal articulations

Metal on Metal

Advantages

1. High wear resistance – High Young's modulus of elasticity (Cobalt Chromium 210GPa); Fracture toughness; Hard
2. Self-polish – surface scratches may be substantially polished out during subsequent activity

Disadvantages

1. Metal ions – uncertainty regarding long-term biologic effects therefore caution regarding teratogenicity
2. Metallosis, Aseptic Lymphocytic Vasculitis Associated Lesions (ALVAR), Pseudotumours – believed to be due to wear debris (smaller and higher in numbers relative to Metal on Polyethylene articulation) and hypersensitivity.
3. Run-in wear – initial transitional elevation of wear which stabilises

Campbell P, Shen FW, McKellop H. Biologic and tribologic considerations of alternative bearing surfaces. Clin Orthop Relat Res. 2004;418:98-111

Walter WL, Yeung E, Esposito C. A review of squeaking hips. J Am Acad Orthop Surg. 2010; 18(6): 319-26

Haddad FS, Thakrar RR, Hart AJ, Skinner JA, Nargol AV, Nolan JF, Gill HS, Murray DW, Blom AW, Case CP. Metal-on-metal bearings: the evidence so far. J Bone Joint Surg Br. 2011;93(5):572-9

2. BONE CEMENT