Royal Medical Services

Oral , Short & Long cases summary

Done by :

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النّصر للمتمرّدين. والهزيمة للمتردّدين،

والنّهايات لمن يملك البدايات ....

بين يديكم ثمرة ليال طوال ومجهود شاق ، بذل على طريق النجاح ، لنطوي

به متاهات الاستعداد للامتحان الشفوي ، متمنين لكم السداد والتوفيق

لا تنسونا من صالح دعائكم ....

اخوانكم : أكرم البلاع , ناصر الشرع , زيد ذنيبات

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This is a summary collection for Oral , Short & Long cases collected from FRCS Viva books , Orthobullets & lectures

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1.Basic Sciences :

Orthopedics history taking template

❖ History :

- This history was taken On …./…/..2019 , at ….00:00 , from : …..

I. Biodata / Patient profile : تعرف على المريض

1) Name : Mr, Mrs ,….
2) Age
3) Occupation
4) Marital status
5) Level of education
6) Body mass index : BMI
7) Site of living / where does pt lives
8) Dependency
9) Level of nurserary at living place
10) Smooking
11) Amission type

II. Chief complaint :

1- (Specifies the ***incindince*** *or* ***orthopedic complains*** that disturbed pt.'s life , or the orthopedic complaint that resulted after this incidnice)

2- clarify the **duration** of the chief complaint

(Duration of CC in Post OP pt is from day of onset till day of surgery & you have to mention which day post op at time of taking hx)

Ex : *Wrist Pain* for 2 hrs after FD / after being victim of RTA

* Most common orthopedic complaints are :

1. Pain
2. Deformity
3. Swelling
4. Change in shape
5. Loss of activity (function) : daily or extra acitivity
6. Stiffness
7. Weakness
8. Instability (ex : giving way , dislocation)
9. Change in sensibility

III. History of present illness :

*clarification of chief complaint , to be written as a story :

Pt hx dates back to …… when he/she started to c/o ……

*Hand dominance : if upper limb pathology

*Walking character (alone , assisted , with help …):if lower limb pathology

A. Pain : Analyzed by **SOCRATES**

1. Site

2. Onset

a. sudden or gradual

b. progressive or regressive.

3. Character :

a. Nature of pain :

1. sharp and stabbing (as in #)

i. stiffness' or aching (as in muscle pain)

ii. Shooting' pain (nerve impingement)

b. Coninuity of pain : constant or intermittent

c. Localised vs Generalised (diffuse pain)

4. Radiation :

5. Associations

6. Time course : Diurnal , Nocturnal

7. Exacerbating/relieving factors

8. Severity

B. Other Orthopedic complaints analysed by : **OPQRSTA**

1. Onset
2. Prior occurrences of this problem
3. Progression
4. Quality
5. Radiation
6. Scale
7. Timing
8. Associated symptoms

C. Neoplastic and infectious symptoms :

1. night symptoms
2. fever
3. sweats
4. Anorexia
5. Fatigue
6. Weakness
7. weight loss

If pts problem is **acute** . then HPI ends with the mentioned above analysis

But if it is **chronic** , then after you are done with chief complaint analysis you shift to :

Old orthopedic surgical hx related to this complaints by **saying**:

Pts history dates back to …….. when he started to have …….

(and you continue all related old orthopedic surgery history)

B. Review of Systems

C. Past medical History (presence of chronic diseases)

D. Past Surgical History

E. Hx of Drugs (medication) : cortisone , Anticoagulants ….

F. Hx of Allergies : Egg allergy , Anesthesia medications allergy…

G. Hx of blood transfusion

H. Family Hx : major medical conditions in Parents , siblings

I. Hx of travel abroad

J. Social Hx: (briefly in patient profile , but details have to be discussed here)

1- Regular or extraordinary skill ? (Athlete)

2- Level of education & occupation

3- Marital status

4- House living condition , where does he live , what floor he lives in , the presence of elevator , what type of bathroom is there

5- Smooking

6- Alcohol

7- Type of walking : assisted , alone

❖ Physical Exam :

1. general Apperance :
    a. Pt is seen in bed , lying supine , sitting …..
    b. not in pain or in sever pain
    c. Looks well , or looks tired
    d. Pain score
2. Vital signs : BP ,HR , Temp , RR , O2 Sat

3. Rapid clinical examination :

1- Face exam : looks pale , jaundiced
2- Neck exam : No masses , no LNE , no deformity
3- Abdomine : Soft , lax
Those syetemic general examination has to be done in 2 minutes before shifting to :

4. **Muscolskeletal exam :**

Requirements:

- **Tape measure**
- **Goniometer**
- **Patella hammer**
- **Sharp point and cotton wool**

1- Look
2- Feel
3- Move

1- Look : Start from superficial to deep (from skin , to bone)

1) Skin condition
    1. swelling
    2. ulcer
    3. blister
    4. ecchymosis & discoloration
    5. Infection signs (ex: redness, pus discharge ….)
    6. Surgical scars

2) vascularity of limb (color of limb)
Ex : You want to do foot surgery , and by hx you know he is smoker , or DM , you found his toes cyanosed , so he has lower limb ischemia

3) Signs of neuropathy : (Periphral neuropathy)
1- Ulcers
2- Hair loss
3- Skin dryness

4) Musculature (looks at muscles) : Good muscle bulk / atrophied

5) Bone :
1- Bone deformity : assessed when :
1) Pt is in Supine position
2) Walk

2- Feel : By palpation :
* Start with skin , muscle , bone
1- Dry
2- Hot
3- Sweaty
4- Muscle bulk
5- Tenderness
6- Lump
7- Deformity

3- Move :
1. Start with Passive ROM then go to Active (in order to assess the widest ROM that could be obtained before pt starts to move)
2. Compare between passive & active ROM
3. Joint stability according to pt complaint
4. Special tests : according to joint complaints

❖ Lab tests & Radiology tests

❖ Impression :

❖ Plan :

❖ Summary :
Example : a 37 yr old female single MF , known case of Bil NOF stress # 13 months ago complicated by Lt sided Non union , admitted for possible metal removal & THR

One pack-year is smoking 20 cigarettes a day for one year. If someone has smoked 10 cigarettes a day for 6 years they would have a 3 pack-year history. Someone who has smoked 40 cigarettes (2 packs) daily for 20 years has a 40 pack-year history.

Calculation

Number of pack-years = (packs smoked per day) × (years as a smoker)

or

Number of pack-years = (number of *cigarettes* smoked per day/20) × number of years smoked. (1 pack has 20 cigarettes in some countries)

Note that despite the unit being called a "pack-year," the actual unit is simply a number of packs (as noted above).

$$
\begin{aligned}
1 \text{ pack-year} &= \cdot 1 \text{ year} \\
&= \cdot 365.24 \text{ days} \\
&= 365.24 \text{ packs} \\
&= 365.24 \text{ packs} \cdot \\
&= 7,305 \text{ cigarettes}
\end{aligned}
$$

For example: a person who has smoked 15 cigarettes a day for 40 years has a (15/20) x 40 = 30 pack-year smoking history.

History

General (Mnemonic: AMPLE or SAMPLE)

1. Signs and Symptoms
2. Allergies
3. Medications
4. Past illness
5. Last Meal
6. Events or Environment related to injury

Surgical Safety Checklist

World Health Organization | Patient Safety
A World Alliance for Safer Health Care

Before induction of anaesthesia

(with at least nurse and anaesthetist)

Has the patient confirmed his/her identity, site, procedure, and consent?
- ☐ Yes

Is the site marked?
- ☐ Yes
- ☐ Not applicable

Is the anaesthesia machine and medication check complete?
- ☐ Yes

Is the pulse oximeter on the patient and functioning?
- ☐ Yes

Does the patient have a:

Known allergy?
- ☐ No
- ☐ Yes

Difficult airway or aspiration risk?
- ☐ No
- ☐ Yes, and equipment/assistance available

Risk of >500ml blood loss (7ml/kg in children)?
- ☐ No
- ☐ Yes, and two IVs/central access and fluids planned

Before skin incision

(with nurse, anaesthetist and surgeon)

- ☐ **Confirm all team members have introduced themselves by name and role.**
- ☐ **Confirm the patient's name, procedure, and where the incision will be made.**

Has antibiotic prophylaxis been given within the last 60 minutes?
- ☐ Yes
- ☐ Not applicable

Anticipated Critical Events

To Surgeon:
- ☐ What are the critical or non-routine steps?
- ☐ How long will the case take?
- ☐ What is the anticipated blood loss?

To Anaesthetist:
- ☐ Are there any patient-specific concerns?

To Nursing Team:
- ☐ Has sterility (including indicator results) been confirmed?
- ☐ Are there equipment issues or any concerns?

Is essential imaging displayed?
- ☐ Yes
- ☐ Not applicable

Before patient leaves operating room

(with nurse, anaesthetist and surgeon)

Nurse Verbally Confirms:
- ☐ The name of the procedure
- ☐ Completion of instrument, sponge and needle counts
- ☐ Specimen labelling (read specimen labels aloud, including patient name)
- ☐ Whether there are any equipment problems to be addressed

To Surgeon, Anaesthetist and Nurse:
- ☐ What are the key concerns for recovery and management of this patient?

This checklist is not intended to be comprehensive. Additions and modifications to fit local practice are encouraged.

Revised 1 / 2009

IL 6 :
increase in 1st hr of infection
peak : 12 hrs
normalize in 3 days

CRP :
increase after 6 hrs of infection
Peak : 3 days
normalize in 3 weeks

ESR :
increase after 24 hrs of infection
Peak : 5-7 days
Normalize in 3 months

23 6:27 PM

Laboratory testing:

- **ESR :**

  Reflects the changes in the concentration of fibrinogen synthesized by the liver

  - Sensitive indicator of inflammation
  - Sensitive indicator of infection (90% in OM)
  - Increase after 24-48 h
  - Return to normal in 3 weeks
  - Helpful in diagnosis and follow up
  - Extreme elevation ? Associated septic arthritis

What should I observe and test once the joint fluid has been aspirated?

You should describe the general characteristics of the gross appearance. The aspirated fluid is inspected for viscosity by the string test, looking for clarity, color, and the presence of blood or fat droplets. The aspirated fluid is then placed in different containers and sent for laboratory tests, depending on the differential diagnosis. In general, testing often includes a cell count with differential, an immunologic test for arthritis, and analysis of glucose and protein. A smear for Gram stain and a bacterial culture are ordered when infection is suspected.

Table 2

Synovial Fluid Characteristics and Arthritis-Related Laboratory Findings

Characteristic	Finding: Normal (No Arthritis)	Noninflammatory Arthritis*	Inflammatory Arthritis†	Septic Arthritis (Native Joint)‡	Prosthetic Hip or Knee Infection§
Appearance	Clear	Clear	Opaque or translucent	Opaque Yellow or green	Clear or opaque
WBC/mm^3	< 1,000	< 1,000	5,000–75,000	> 50,000	> 1,100-3,000
Polymorphonuclear cells	< 25%	< 25%	> 50%	> 75%	> 64%-80%
Culture	Negative	Negative	Negative	Positive	Positive

WBC = white blood cell count.
*Associated conditions: Degenerative joint disease, trauma, pigmented villonodular synovitis, neuropathy, systemic lupus erythematosus, acute rheumatic fever.
†Associated conditions: Rheumatoid arthritis, crystal-induced arthritis, seronegative arthropathy, systemic lupus erythematosus, acute rheumatic fever.
‡An immunocompromised patient may not have an elevated synovial WBC. A normal or noninflammatory WBC does not preclude active septic arthritis.
§Data are unavailable for other types of prosthetic joints. Underlying inflammatory arthritis may cause false-positive results.

TETANUS PROPHYLAXIS

- **Tetanus**
  - Caused by tetanospasmin
  - Exotoxin of anaerobic Gram +ve rod (bacillus) – Clostridium Tetani which is found in soil
  - Neuroparalytic disease
  - Toxoid is booster -TTB (inactivated vaccine) & IGg is active immunity

- **DOH guidelines**

• Full course & booster <10 years	Nil if clean	TTB if dirty
• Full course & booster > 10 years/non-immunised	TTB if clean	TTB if dirty

Clean wound is defined as wounds less than 6 hours old, non-penetrating with negligible tissue damage

Immunization History	Clean, Minor Wound (GA I)	GS II and III
Unknown History or <3 doses	• Give vaccine only	• Give vaccine • Give immune globulin
Vaccination complete (3 prior doses)	• No prophylaxis if last dose within 10 years • Give vaccine if >10 years since last dose	• No prophylaxis if last dose within 5 years • Give vaccine if >5 years since last dose

	Clean Minor Wounds		All Other Wounds	
	Give Td	Give TIG	Give Td	Give TIG
Vaccination history unknown or < 3 doses	Yes	No	Yes	Yes
Vaccination history ≥ 3 doses	Only if last dose received ≥10 years ago	No	Only if last dose received ≥ 5 years ago	No

Td = tetanus and diphtheria vaccine; TIG = tetanus immune globulin

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

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	

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

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

PREVENTION OF SURGICAL SITE INFECTIONS

Stop further operating and investigate if recurrent problem
Root-cause analysis if high incidence of SSI

Pre op

- Adequate nutrition
- Optimise medical conditions — HbA1c should be < 7%
  Hold short-acting insulin and oral medications on morning of surgery
  Give half dose of long-acting insulin
- Shower NICE shows no evidence of benefit for preoperative showering or bathing with chlorhexidine over other wash products (e.g soap) or placebo, to reduce SSI
- Treat infection
- Shaving
- Equipment sterilization
- Screening for MSSA and MRSA
- Stop smoking, Serum Cotinine

Intra op

- Ultraclean air
- Minimise traffic and personnel
- Theatre personnel and patient clothing
- Prophylactic Abx
- Hand decontamination
- Skin preparation – alcohol-based solution of chlorhexidine first choice unless contraindicated and surgical site not next to mucous membrane

Meticulous tissue handling, haemostasis
Closure methods

Post op

- Occlusive dressing, allow hypoxic & acidic environment which retards growth of skin pathogens
- Dedicated elective wards

Sterilization	Autoclave	Steam
	Gamma irradiation	
	UV light	for surface sterilization only
	Gas	Ethylene Oxide

Infection control

Two approaches are taken to address this issue:

- Reducing the size of the inoculum
- Enhancing the host defences.

Reducing the inoculum

- Ward hygiene
- Screening/separation of infected cases
- Skin cleanliness (*not* antisepsis – as this encourages resistance)
- Theatre design and practice (see below)
- Limiting dressing changes.

Enhancing host defences

- Good nutrition
- Antibiotic prophylaxis where appropriate
- Tetanus prophylaxis
- Optimize the skin preoperatively (e.g. psoriasis treatment, avoidance of blisters)
- Avoid unnecessary antibiotics (resistance).

Prevention of infection in THA

Prophylactic measures to reduce hip arthroplasty infection are given in Table 14.4.

Preoperative factors

- Same-day admission
- Separation of elective from trauma cases
- All septic lesions should be examined and treated (feet, urinary, dental)
- Shave in the anaesthetic room (not night before).

Perioperative factors

- Antibiotic prophylaxis: systemic antibiotics, antibiotic-loaded cement
- Surgical technique: gentle handling of tissues, careful haemostasis, limitation of haematoma formation, avoid cremation of tissues/necrosis, length of surgery, wound lavage, etc.
- Movement: avoid unnecessary theatre personnel movement during surgery
- Ace masks: BOA guidelines
- Gowns: modern, weaved patterns
- Gloves and hands: two pairs of gloves, changing the outer ones frequently
- Head gear: no hair exposed
- Body exhaust systems
- Sterile drapes: disposable non-woven drapes
- Drainage wound: arguments for and against
- Ventilation system: laminar flow, ultra-clean-air system
- Ultraviolet light: bactericidal.[43]

Postoperative factors

- Antibiotic cover for urethral catheterization
- The risk of infection is increased in rheumatoid arthritis, diabetes, those with immunosuppression and those with a history of previous joint infection.

with metal on the operating table resulting in iatrogenic skin burns
  - 'Isolated system' – the return electrode becomes the only route back to the generator so 'grounding' is no longer a risk
  - Return electrode placement should be over well vascularized muscle mass. Most systems now monitor the impedance at the return electrode to reduce burn risk
  - Care should be taken with flammable prep solutions, which may soak into drapes and then catch fire – alcohol burns without a visible flame
- Bipolar electrosurgery involves active and return point electrodes at the surgical site. The forceps points (electrodes) must be separated for current to pass through tissue. Advantage of bipolar – avoids risk of damage from passage of current through surrounding tissues (particularly arteries in digits, etc.), but still a risk of burns from alcohol-based prep solutions
- Note that, in electrocautery, direct current is used, in contrast to electrosurgery, which involves alternating current.

Infection control

Two approaches are taken to address this issue:

- Reducing the size of the inoculum
- Enhancing the host defences.

Reducing the inoculum

- Ward hygiene
- Screening/separation of infected cases
- Skin cleanliness (*not* antisepsis – as this encourages resistance)
- Theatre design and practice (see below)
- Limiting dressing changes.

Enhancing host defences

- Good nutrition
- Antibiotic prophylaxis where appropriate
- Tetanus prophylaxis
- Optimize the skin preoperatively (e.g. psoriasis treatment, avoidance of blisters)
- Avoid unnecessary antibiotics (resistance).

Bacteria

Gram staining involves staining with crystal violet, fixing with iodine then washing with alcohol: Gram-positive retain dye; Gram-negative dye washes out and then re-stained with safranin O:

- Gram-positive cocci: staphylococci, streptococci
  - Staphyloccoci may be coagulase-positive (*Staph. aureus*), or coagulase-negative (*Staph. epidermidis*)
  - Panton–Valentine leukocidin (PVL) is a toxin produced by *Staph. aureus*, which may cause necrotizing fasciitis
  - Streptococci may be alpha-haemolytic (*Strep. viridans*, *Strep. pneumoniae*), or beta-haemolytic (group A – *Strep. pyogenes*, group D – *Strep. faecalis*)
- Gram-negative cocci: *Neisseria*
- Gram-positive bacilli (rods): *E. coli*, *Proteus*, *Klebsiella*, *Pseudomonas*
- Gram-negative bacilli: *Clostridia* (*tetani*, *difficile*, *perfringens*).

Antibiotic actions

- Bacteriostatic
- Bacteriocidal
- Mixed
- Penicillin/cephalosporins – prevent bacterial cell wall synthesis – cell wall enzyme
- Glycopeptides (vancomycin, teicoplanin) – interfere with cell wall enzyme
- Fucidin and clarithromycin – block ribosomal peptides
- Linezolid – inhibits protein synthesis.

Bacterial resistance

Resistance acquired in one of two ways:

- Genetic – resistance transferred via DNA — plasmids (small circles of double-stranded DNA), integrons and transposons
- Proteomic – altered target site on bacterium or altered enzyme that is the target of drug actions.

Skin flora

- Includes coagulase-negative *Staphylococcus epidermidis*, *Staphylococcus aureus* and Gram-positive diphtheroid bacilli. These are accessed by lipophilic antibiotics secreted in perspiration.

MRSA

Acquired penicillin binding protein PBP2a, encoded by gene *mecA*.

If found on screening swabs:

- 5 days intranasal mupirocin
- 4% chlorhexidine baths
- Re-swab and repeat if necessary.

Treatment:

- Glycopeptides – vancomycin and teicoplanin
- Oxazolidinones – linezolid.

Biofilms

- Bacteria on surface of implants secrete a glycoprotein biofilm
- This biofilm reduces access of antibiotics to bacteria (rifampicin has good penetration of biofilms)

Bacterial Resistance

- Mechanisms
  1) Phagocytosis of the drug (beta lactamase)
  2) Genetic mutation (MEC A gene MRSA)
  3) Altered cell wall permeability
  4) Biofilm Formation
  5) Ribosome Alteration
  6) Active efflux pumps

Bacteriology & infection)

- There are two main types of resistance

Intrinsic resistance	The bacteria has properties that prevent antibiotics to act on it E.g. changes in cell wall permeability, efflux pumps, enzyme production
Extrinsic Resistance	The bacteria develop resistance to an antibiotic to which it was previously sensitive e.g. chance mutations, drug resistance gene (this is mediated via Plasmids)

British Orthopaedic Association Recommendation on Sterile Procedures in Operating Theatres

- All hair to be kept covered
- Face masks to be worn at all times within operating theatre
- Entrance and exit through clearly identified doors to prevent disturbance of air flow in theatres
- Number of people in theatres kept to minimum
- Traffic from dirty areas rigidly controlled
- Drapes and gowns made of impervious material

British Orthopaedic Association (1999). Recommendations on sterile procedures in operating theatres.

Hughes SP, Anderson FM. Infection in the operating room. J Bone Joint Surg Br. 1999;81(5):754-5

<u>Prevention of surgical site infection (SSI)</u>

- Pre-operative
  - Pre-assessment – optimise medical conditions e.g. diabetic control; microbiological swabs
  - Pre-operative showering
  - Hair removal – only if necessary and with electric clippers just prior to procedure
  - Skin preparation – alcohol; chlorhexidine; iodine
  - Antibiotic prophylaxis
  - Pre-operative theatre personnel preparation – surgical caps/hoods; face masks; hand washing; gowns; gloves; footwear
- Peri-operative
  - Ultraclean air/Laminar flow
  - Meticulous tissue handling
  - Minimise operative time
  - Minimise traffic into and out of theatre
  - Minimise theatre personnel
- Post-operative
  - Antibiotic prophylaxis
  - Meticulous wound care

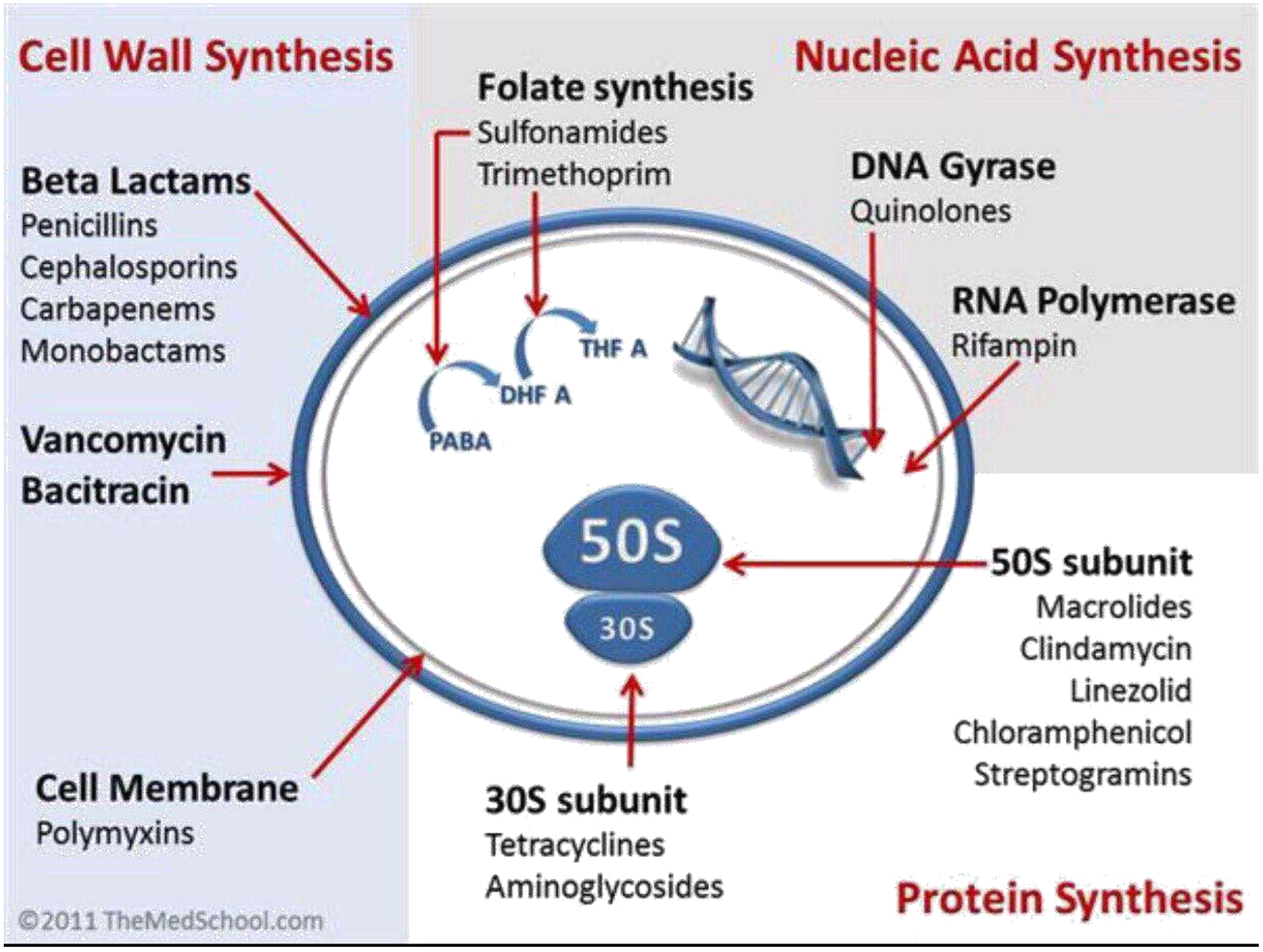
---

14. Tourniquets. Precautions, complications and time limits?How will you determine cuff

Bactericidal vs. Bacteriostatic

- Prefer ‘Bactericidal’ to ‘Bacteriostatic’
  - Bactericidal
    - disrupts the cell wall synthesis-killing the bacteria
    - Less reliance on host resistance
    - Drug works **faster than ‘static’**
    - More flexibility with dosage interval

  - Bacteriostatic
    - inhibits the RNA synthesis/reproduction
    - Inhibit growth and reproduction of bacteria
    - Help the host defenses to take over


Cell Wall Synthesis
Nucleic Acid Synthesis
Folate synthesis
Sulfonamides
Trimethoprim
Beta Lactams
Penicillins
Cephalosporins
Carbapenems
Monobactams
DNA Gyrase
Quinolones
RNA Polymerase
Rifampin
THF A
DHF A
PABA
Vancomycin
Bacitracin
50S
30S
50S subunit
Macrolides
Clindamycin
Linezolid
Chloramphenicol
Streptogramins
Cell Membrane
Polymyxins
30S subunit
Tetracyclines
Aminoglycosides
©2011 TheMedSchool.com
Protein Synthesis

Antibiotic Grouping By Mechanism	
Cell Wall Synthesis	Penicillins
	Cephalosporins
	Vancomycin
	Beta-lactamase Inhibitors
	Carbapenems
	Aztreonam
	Polymycin
	Bacitracin
Protein Synthesis Inhibitors	
	Inhibit 30s Subunit
	Aminoglycosides (gentamicin)
	Tetracyclines
	Inhibit 50s Subunit
	Macrolides
	Chloramphenicol
	Clindamycin
	Linezolid
	Streptogramins
DNA Synthesis Inhibitors	Fluoroquinolones
	Metronidazole
RNA synthesis Inhibitors	Rifampin
Mycolic Acid synthesis inhibitors	Isoniazid
Folic Acid synthesis inhibitors	Sulfonamides
	Trimethoprim

Exopolysaccharide glycocalyx allows bacteria to adhere to orthopaedic implants and elude antimicrobial therapies through the creation of biofilms.

Biofilms are defined as a structured community of bacterial cells enclosed in a self-produced polymeric matrix and adherent to an inert or living surface. Biofilm production usually occurs within 4 weeks, and is extraordinarily hard to eradicate with antibiotic therapy alone. In the setting of a chronic infection of an orthopaedic implant (>4 weeks), explantation of the implant followed by antimicrobial therapy is the most reliable method of curing the infection.

Vancomycin (bactericidal: disrupts peptioglycan cross-linkage)	Vancomycin	MRSA** PCN/Ceph allegies** *S. aureus* *S. epidermidis*	Red man syndrome Nephrotoxicity Ototoxicity

Bone Infections

Indicated for treatment of bone infections due to: susceptible isolates of MRSA and coagulase negative staphylococci, methicillin-susceptible staphylococci in penicillin-allergic patients, or those patients who cannot receive or have failed to respond to other therapies

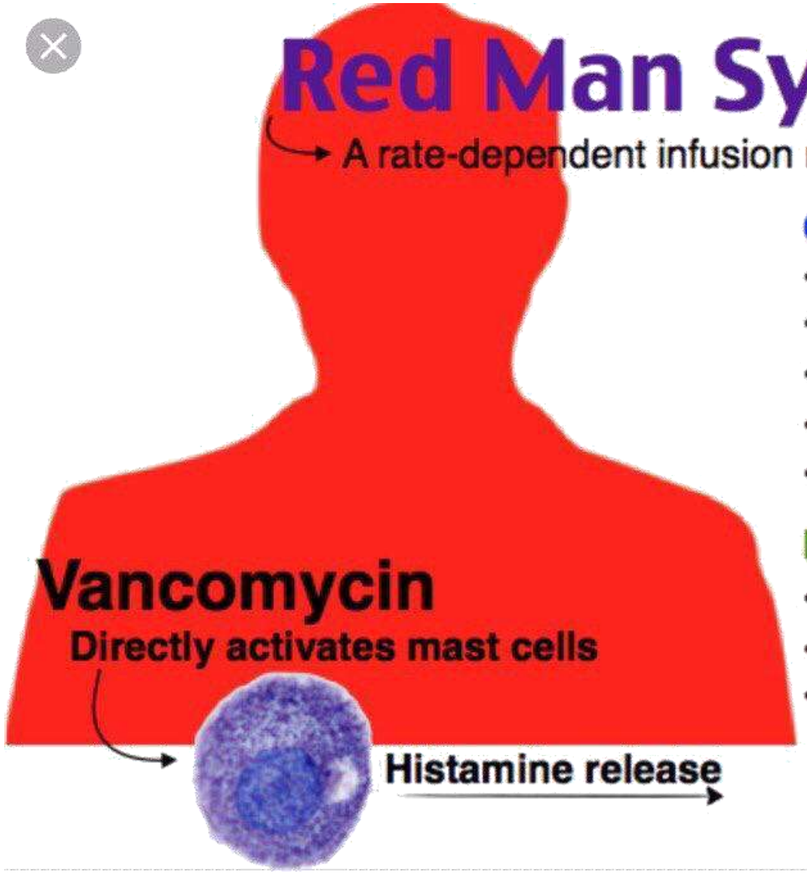
Usual dosage: 2 gdivided either as 500 mg q6hr or 1 gram q12hr

Initial daily dose should be no less than 15 mg/kg

10-15 mg/kg/dose

Red Man Syndrome

A rate-dependent infusion reaction (***not a true allergic reaction***)



Clinical

- Flushing
- Erythema
- Pruritus
- Affecting upper body, neck and face > lower body
- Myalgia, dyspnea, hypotension

Management

- Stop infusion
- Administer antihistamine (diphenhydramine)
- Can restart at slower rate once symptoms resolve

Theatre Zones

- **Outer** — General access - Patient reception, Office corridor, changing room
  Open to foot & trolley access
- **Clean** — theatre complex
  Limited access, between reception and theatre
- **Restricted** — Anaesthetic room, scrub room
  Aseptic operating theatre
  Hat and masks, minimum personnel
- **Ultraclean** — Inside laminar flow area
  Only scrubbed and gowned personnel
- **Dirty** — Disposal, sluice

HOW TO REDUCE INTRA-OPERATIVE BLEEDING

- **Pre op** Erythropoietin erythrocyte–stimulating glycoprotein
  Made by kidneys
  Tranexamic acid - 1g prior to incision
  Iron, Vit B12 and Folate supplemnts
- **Intra op** diathermy
  Tourniquet
  Controlled hypotensive anaesthesia
  Local adrenaline injection
  Cell salvage
  Topical haemostatic agents, fibrin sealant
  TXA for topical usage 3g introduce this solution into the cavity of the joint before deflating tourniquet
  Leave to bath the bony surfaces and peri-articular tissue for five minutes
  Then remove with the sucker before routine irrigation and closure.
- **Post op** reinfusion drains

Strategies for reducing peri-operative blood loss in total knee arthroplasty – **BJJ, 2016**

Tranexamic acid (TXA)

- Overview
  - an antifibrinolytic that promotes and stabilizes clot formation
  - studies have shown that TXA reduce perioperative blood loss and transfusion in THA and TKA
- Mechanism
  - synthetic derivative of the amino acid lysine
  - competitively inhibits the activation of plasminogen by binding to the lysine binding site
  - at high concentrations, is a non-competitive inhibitor of plasmin
  - has roughly 8-10 times the antifibrinolytic activity of ε-aminocaproic acid
- Dosing
  - intravenous
    - 10-20 mg/kg initial bolus dose followed by repeated doses of the initial TXA dose every 3 hours for 1-4 doses
    - 10-20mg initial bolus followed either by an infusion of 1-10 mg/kg/hr for 4-30 hou
  - topical application is as effective as IV
    - sprayed onto open wound at completion of procedure
    - no detectable TXA in the bloodstream after

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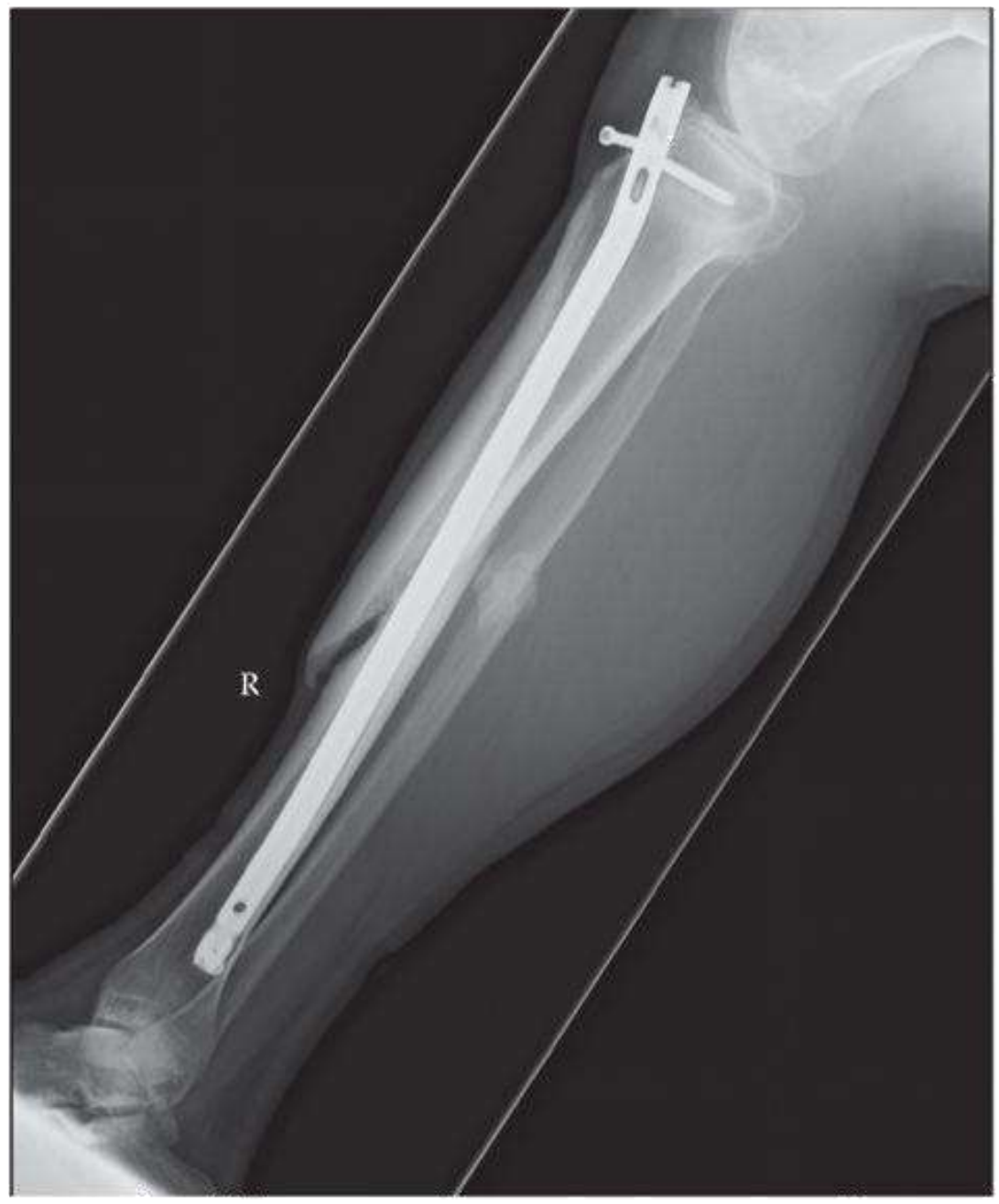
  - topical application is as effective as IV
    - sprayed onto open wound at completion of procedure
    - no detectable TXA in the bloodstream after topical application
- Metabolism
  - <5% of the drug is metabolized
  - biological half-life in joint fluid is 3h, present in tissues for up to 17h
- Risks
  - systematic review shows no increase in thromboembolic events
  - relatively few adverse reactions have been reported in the arthroplasty literature

A nonunion is defined as a fracture that has not healed and has no further capacity to heal without further intervention. Nonunions are typically classified as hypertrophic, oligotrophic, and atrophic. Hypertrophic nonunions show clear evidence of the ability to heal without bridging of fracture gaps. Atrophic nonunions show no evidence of biologic healing and no bridging of fracture gaps. Oligotrophic nonunions tend to fall somewhere in between hypertrophic and atrophic nonunions with some evidence of biologic activity however incomplete healing. Understanding these characteristics allows for proper identification of the nonunion and selection of appropriate intervention with regard to increasing bone biology and fracture stability to achieve healing.

NON-UNION



This patient is 1 year down the line following an open fracture of the tibia treated with an intramedullary nail.

1. **Describe the radiograph and explain the diagnosis.**
   This is a lateral radiograph showing a tibial shaft fracture treated with an intramedullary nail. The nail is backing out and the proximal screw is clearly broken. The fracture shows no evidence of healing 12 months post-surgery and therefore this would be described as a non-union.

   Treatment with a reamed intramedullary nail for closed fractures has a reported rate of non-union of between 1% and 4%. Following grade 1 open fractures, the rate of non-union remains low, at 2%, but it increases up to 36% for Gustilo and Anderson grade IIIB injuries.

2. **What is the definition of a non-union?**
   A non-union is described as a failure of a fracture to heal within the time frame expected for that specific fracture. The U.S. Food and Drug Administration (FDA) defined non-union as a failure for a fracture to unite by 9 months, with no radiographic progression towards union in the previous 3 months.

3. What are the clinical findings in a non-union?

Clinically, patients may have ongoing pain at the fracture site and, in the lower limb, pain on weight bearing is a classical symptom of non-union. On examination, there may be pain on palpation in addition to movement and crepitus at the fracture site.

In an infected non-union, patients may describe wound problems after surgery such as infected or leaking wounds and may have required antibiotics in the post operative phase. There may be ongoing inflammation at the fracture site in the form of erythema and persistent/night pain may be present, in addition to constitutional symptoms of infection such as sweats, fever, rigors, weight loss and loss of appetite.

4. What is the difference between clinical and radiographic union?

Clinical union is defined as the absence of tenderness or motion at the fracture site with no pain on loading. *Radiographic union* is defined as the presence of visible bridging trabeculae on three out of four cortices on orthogonal radiographs.

5. What are the causes of non-union?

Factors causing non-union can be divided into patient factors, fracture factors or surgical factors.

Patient factors:

- Age (paediatric fractures heal quicker than adult fractures)
- Smoking and excess alcohol
- Drugs (NSAIDs, corticosteroids)
- Medical co-morbidities (diabetes, peripheral vascular disease, malnutrition, anaemia, hypothyroidism, hyperparathyroidism)

Fracture factors:

- Bone involved (femoral shaft take 16 –/+ 4 weeks to unite, whereas a distal radius fracture may heal in under half this time)
- Area of bone involved (diaphyseal fractures generally take longer to heal than metaphyseal fractures). Classically, the distal tibia and proximal pole of the scaphoid are at high risk of non-union
- Fracture type (high-energy fractures, open fractures, comminuted fractures, bone loss, and fractures associated with significant soft tissue damage or periosteal stripping will take longer to heal)
- Infection

Surgical factors:

- Extensive soft tissue damage/periosteal stripping
- Inadequate stability
- Rigid fixation with gapping at the fracture site
- Introduction of infection

6. Do you know of any different types of non-union?

Non-unions can be described as being either hypertrophic or atrophic:

Hypertrophic non-unions are a mechanical problem. They occur when there is a good blood supply but excessive strain at the fracture site prevents progression of the callus to form bone.

Atrophic non-unions are a biological problem. Almost all the patient, fracture, and surgeon factors already discussed can lead to an atrophic non-union. A fracture fixed with rigid fixation but with the fracture fragments distracted will also lack stimulation of callus formation.

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First infection must be excluded as this can also cause non-union.

If not infected:

- Open debridement
- Bone graft (iliac crest graft)
- Additional BMPs?
- Fixation—repeat internal or external (Ilizarov)

What factors influence fracture healing?

1. Fracture mechanical environment
2. Local biology
   Blood supply
   Degree of soft tissue injury
   Open or closed injury
   Degree of fragmentation/bone loss
   Site of fracture (metaphyseal versus diaphyseal)
   Soft tissue interposition
   Stability (cf. absolute/relative/dynamization)
   Presence of infection
   Presence of pathological lesion
   Previous irradiation to that area
3. Systemic biology
   Age
   Smoking
   Drugs—non-steroidal anti-inflammatory drugs (NSAIDs), steroids, bisphosphonates
   Medical co-morbidities—diabetes mellitus (DM)
   Nutrition
   Associated head injury

4 of 7

7. What are the principles of non-union surgery?

- Eradicate infection
- Excision of interposing tissues
- Restore blood supply
- Stabilisation of bone ends
- Bone graft any fracture gaps

As a general rule, hypertrophic non-unions require increased mechanical stability, usually by compression of the non-union site.

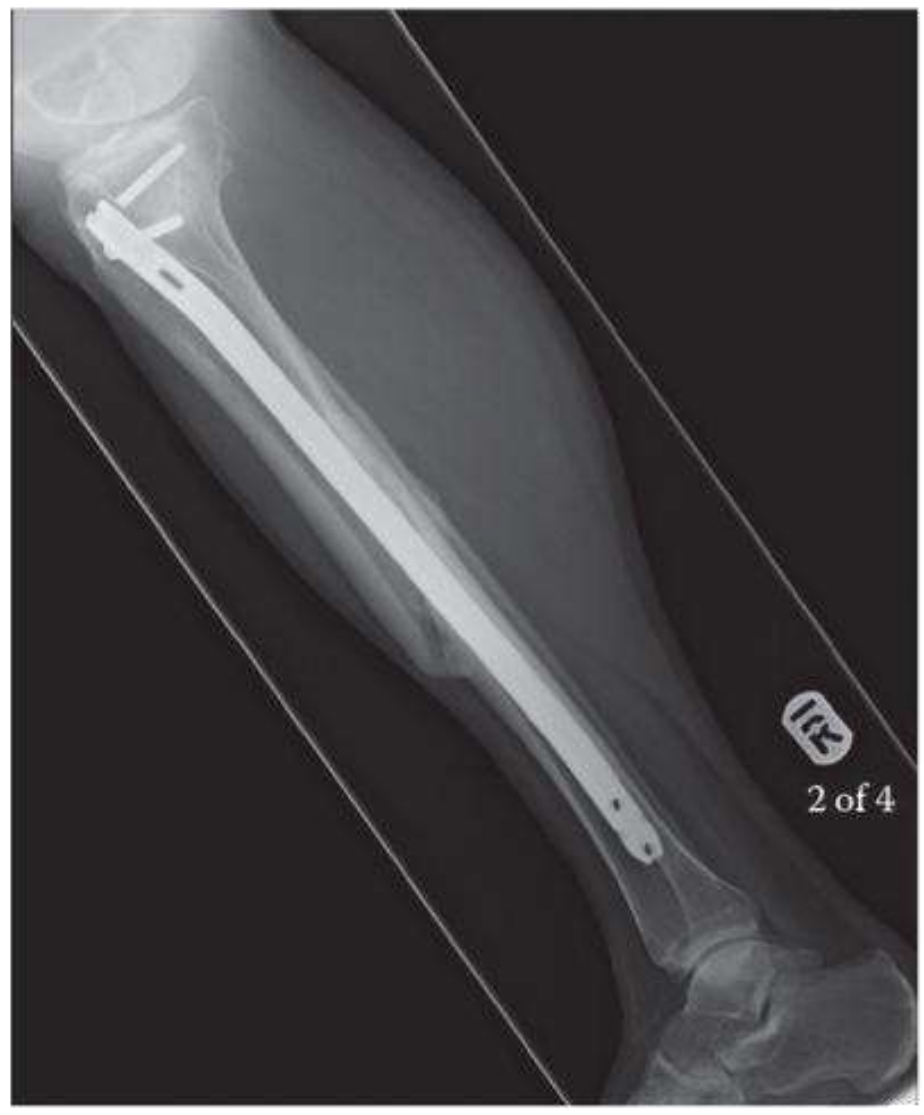
With atrophic non-union, all the principles listed here are required. They need stabilisation and biological enhancement.

8. How would you treat the fracture pictured above?

I would start by investigating for infection with a thorough history (as per Q3) and performing a full set of blood tests including FBC, ESR and CRP. If there was no infection, a non-union of the tibia can be treated very effectively with an exchange nailing with a nail 1–2 millimetres larger in diameter after reaming, and I would send reamings for microscopy, culture and sensitivity to rule out an indolent infection. As above, the increased diameter nail will lead to increased stabilisation of the fracture site and the reaming will deliver autologous bone graft to the fracture site.

The largest cohort of tibial diaphyseal non-unions treated with exchange nailing comes from Edinburgh and, in the aseptic cases, showed a union rate of 75% with a single exchange nailing, rising to 95% with repeat exchange nailing. In the context of infection, exchange nailing had a union rate of 35%, rising only to 61% after a second exchange nailing. Other methods of treatment, such as Ilizarov frames, are therefore indications in infected non-unions.

The radiograph below shows a successful union following exchange nailing.



Principles of management of non-union

Generally, long bone fractures are considered to be non-unions if they have not healed by double the time required for the fracture to heal usually. Non-progression of radiological healing, pain and mobility at the fracture site all point to non-union.

Patient factors

- Comorbidities – diabetes, steroids, smoking, rheumatoid arthritis. Type of host
- Previous surgery – number of surgeries, approaches, infection, implant type, bone graft
- Local soft-tissue status – poor skin, open wounds.

Weber–Cech classification:

- Hypertrophic (intact blood supply and mechanical instability)
- Atrophic (loss of blood supply and mechanical instability).

Host type in infected non-unions:

- Cierny classification:
  - Type A – good immune system
  - Type B – compromised systemically or locally
  - Type C – significant immune compromise so that surgery is not contemplated.

Goals of non-union management:

- Achieve fracture union
- No shortening or malalignment
- Good function of the limb.

Investigations:

- Blood tests – FBC, ESR, CRP
- Imaging – radiographs, CT and/or MRI scans – to assess bony defects, osteomyelitis, arteriography in cases where plastic surgery is anticipated owing to poor soft-tissue status.

Planning management:

- Host type
- Soft-tissue status – coordinate with plastic surgeon
- Bone factors – alignment, shortening, bone loss, joint stiffness.

Options

No previous surgery

- Hypertrophic non-unions in good alignment and no shortening or gap:
  - Functional cast brace and/or electromagnetic stimulation or LIPUS (low intensity pulsed ultrasound)
  - Nail fixation for diaphyseal non-union
  - Plate fixation for metaphyseal/diaphyseal junction non-union

- Atrophic non-union, shortening, gap, malalignment, poor soft tissues:
  - Circular frame fixation for bone transport or lengthening or gradual correction of malalignment.

Previous surgery

- Investigations to rule out infection and other investigations as described above
- Aseptic non-union:
  - Single stage revision fixation (fixation options as above) with or without augmentation with autologous bone graft or demineralized bone graft or recombinant bone morphogenic protein
- Septic non-union – staged procedure:
  - First stage – removal of implants, resection of devitalized bone and soft tissues, local and systemic antibiotics, temporary spanning external fixation. Serial blood markers of infection, blood cultures, repeat debridement if required. Temporary vacuum-assisted dressing
  - Second stage – soft-tissue cover by plastic surgery if required
  - Third stage – definitive procedure with either plate or nail or circular frame as discussed above.

Fracture non-union management is a subspecialty and needs a dedicated multidisciplinary team, including surgical team, physiotherapists, occupational therapists, specialist nurses, microbiologist, pathologist and radiologist.

- Cortical bone become less ductile and more brittle at very high strain rates
- Trabecular bone has lower stiffness and strength than cortical bone
- Trabecular bone is less anisotropic than cortical bone
- Trabecular bone compressive strength is highly dependent on its density
- Trabecular bone is relatively tough (it absorbs a high amount of energy before failing completely)
- Trabecular bone yield point is independent of its mass or the applied load but depends almost entirely on the strain - the strain before yield will, therefore, be the same for osteoporotic and normal bone; the applied load to achieve that strain will, however, differ between the two as the normal bone is stiffer than the osteoporotic bone

Fracture healing

Fracture healing can occur by primary or secondary bone healing.

Primary bone healing

- Requires close anatomical reduction with minimal movement at the fracture site (<2% strain)
- In the initial stages, osteoblasts differentiate from mesenchymal cells and lay down woven bone in any gaps. Lamellar bone may be laid down directly if there are no gaps
- Remodelling then occurs across the fracture site, with cutting cones passing across the fracture site
- Healing is slow Without callus

Secondary healing (by callus)

- Requires some motion at the fracture site (>2% but <10%)[8]
- Hard callus forms under periosteum at periphery
- Endochondral callus – Fibrocartilage forms, becomes calcified and is then replaced with bone
- In secondary healing by callus, the callus undergoes a process of progressive stiffening. In the earlier, less stiff, stages it is more resilient to movement at the fracture site but less good at taking loads or resisting deformation. The strength of the healing fracture does not necessarily correlate with its stiffness

Stages of secondary fracture healing by callus

- Stage 1: First week. Haematoma formation with invasion of macrophages, leukocytes and lymphocytes. Proinflammatory cytokines (including IL-1 and IL-6 and tumour necrosing factor α), and peptide signal molecules (including BMPs, TGFβ and PDGF) are present. Progenitor cells invade. Granulation tissue forms
- Stage 2: 1 week to 1 month. Soft callus forms. In this stage, fibrous tissue, cartilage and woven bone form. Chondroblasts and fibroblasts differentiate and form collagen (mainly type II) and fibrous tissue. Proteoglycans are produced, which suppress mineralisation. The chondrocytes then release calcium into the ECM and also protein-degrading enzymes that break down the proteoglycans, thus, allowing mineralization to take place
- Stage 3: 1–4 months. Hard callus forms. The soft callus is invaded by new blood vessels and chondroclasts break down the calcified callus, which is replaced by osteoid (type I collagen) formed by osteoblasts. The osteoid calcifies to form woven bone. The osteoid callus is stiffer than the soft chondroid callus Enchondral ossification converts soft callus to hard callus (woven bone)
- Stage 4: Remodelling – Several years. The woven bone is remodelled to lamellar bone. The medullary canal reforms as the bone remodels in response to the stresses placed upon it

Bone graft

Function

- Mechanical (structural support)
- Biological (bone healing)

Graft properties

Osteoconductive

- Acts as three-dimensional scaffold or matrix on which new bone forms
- Supports ingrowth of capillaries, perivascular tissues and osteogenic precursor cells
- Example – Coral scaffolds

Osteoinductive

- Provides a biological stimulus that has the capacity to activate and recruit from the surrounding mesenchymal-type cells, which then differentiate into cartilage-forming and bone-forming cells
- Mediated and regulated by graft-derived factors, including TGF, BMPs (bone morphogenetic proteins), IGF-1 and IGF-2 (insulin-like growth factors), interleukins, etc
- Example – Fresh frozen allograft

Osteogenic

- Graft contains living cells that are capable of differentiation into bone
- Graft has inherent biological activity
- Example – Fresh allograft

Genetics

- *Autograft* (same individual) – Including vascularized and free grafts
  - No immunogenicity
  - No risk of disease transmission

1. Bone healing

Bone is :
substance that forms the skeleton of the body. It is composed chiefly of :
1. **organic** component (40% of dry weight)
2. **inorganic** component
(60% of dry weight)

Bone Healing a complex sequential set of events to restore injured bone to pre-fracture condition

Fracture stability (mechanical stability) determines the type of healing that will occur

- when the strain is below 2%, **primary** bone healing will occur
- when the strain is between 2% and 10%, **secondary** bone healing will occur

Modes of bone healing (Modes of bone healing differs from stages of healing)
(Modes = Types)

1. primary bone healing (strain is < 2%)

- The method of bone formation in Primary bone healing is intramembranous healing

- occurs via Haversian remodeling
- occurs with absolute stability constructs

2. secondary bone healing (strain is between 2%-10%)

- The method of bone formation in Secondary bone healing is enchondral healing

- involves responses in the periosteum and external soft tissues.
- occurs with non-rigid fixation, as fracture braces, external fixation, bridge plating, intramedullary nailing

Stages of Fracture Healing	
Inflammation	1. **Hematoma** formation 2. **Growth factors** secretion (Macrophages, neutrophils and platelets release several **cytokines** (PDGF, TNF-Alpha, TGF-Beta, IL-1,6, 10,12) detected 24 H post injury 3. **Fibroblasts** and **mesenchymal cells** migrate to fracture site and **granulation tissue** forms around fracture ends 4.**Osteoblasts** and fibroblasts proliferate

Repair	1.**Primary callus** forms within two weeks. 2.The mechanical environment drives differentiation (Amount of callus is inversely proportional to extent of immobilization)	
	osteoblastic (stable environment , Strain < 2%) primary cortical healing occurs with **rigid immobilization** (ie. compression plating)	**chondryocytic** (unstable environment strain 2-10%) enchondral healing with periosteal bridging occurs with **Non rigid immobilization** Enchondral ossification converts soft callus to hard callus (woven bone). cytokines drive **chondocytic** differentiation.
Remodeling	1.Begins in **middle** of **repair** phase and continues long after clinical union 2. **Woven** bone is gradually **converted** to **lamellar** bone 3.Medullary cavity is reconstituted 4. chondrocytes become apoptotic and VEGF production leads to new vessel invasion 5. **Bone is restructured** (Shaped) in response to stress and strain (**Wolff's** Law & **piezoelectic** charges)	
	In **Primary** bone healing : **Haversian** remodeling (development of **cutting cones**) In **Secondary** bone healing : **Osteoclastic resorption** via **Howship lacunae** (Trabecular bone remodling method) ??	

cement is used to describe a substance that bonds two things together. However, PMMA acts as a space-filler that creates a tight space which holds the implant against the bone and thus acts as a ‘grout

Table 2 – Components of Bone Cement

Powder	Liquid
Polymer Polymethylmethacrylate/copolymer (PMMA)	Monomer Methylmethacrylate (MMA)
Initiator Benzoyl peroxide (BPO)	Accelerator N, N-Dimethyl para-toluidine (DMPT) diMethyl para-toluidine (DMpt)
Radio-opacifier Barium sulphate ($BaSO_4$) Zirconia (ZrO_2)	Stabilizer Hydroquinone
Antibiotics (e.g., gentamicin)	

Generations of Cementing Technique

First generation cementing technique

1)- Hand mixing
2)-Minimal preparation of the femoral canal
2)-Digital application of cement.

Second generation cementing techniques

1)-Preparation ,packing and drying of the femoral canal
2)-Distal cement restrictor
3)-Pulsatile irrigation,
4)-Retrograde insertion of cement with a cement gun.

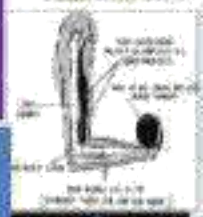
Third generation cementing techniques

1)-Cement is prepared using a vacuum-centrifugation(reduces porosity).
2)-The femoral canal is irrigated with pulsatile lavage and then packed with adrenaline soaked swabs.
3)-Insertion and pressurisation of the cement in a retrograde fashion

Fourth generation cementing techniques

Insertion using distal and proximal centralizers to ensure an even cement mantle (4th generation).




Functions of Bone cement

- Allows secure fixation of implant and bone
- Mechanical interlock and space filling
- Load transferring
- Maintenance/restoration of bone stock
- Release of antibiotics

Contraindications

- Pregnancy
- Active infection
- Hypersensitivity (d/t histamine release n Complement activation C3a/C5a)
- Metabolic disorder

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Phases of cement setting

- **Mixing phase** — 50 sec - 2min for low viscosity
- **Dough phase** — from mix to phase when cement is non-sticky, includes mixing time
  If the cement is inserted too early, blood mixes into it reducing its strength
  Increased humidity lengthens dough phase
- **Working phase** — Implantation phase, from end of dough time to begening of setting
  Up to 7 minutes from start
  Increased temp & increased mixing and handling reduces working phase
- **Hardening phase** — Implant should be kept still, as cement is still notch sensitive
- **Setting time** — From mixing till it reaches maximum heat and becomes hard
  In vivo temperatures are reported to be between 40 and 56
  Increased theatre temperature and humidity reduces setting time

Polymerisation – exothermic reaction

1. Dough time –time from beginning of mixing to when cement is no longer sticking (2-3 min)
2. Setting time – time from beginning of mixing to point where surface temperature is half maximum

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(8-12 min)

3. Working time – Difference between dough time and setting time

IDEAL ANTIBIOTIC PROPERTIES

- Preparation must be thermally stable.
- Must be water soluble for diffusion into tissues.
- Bactericidal.
- Must be released gradually over an appropriate time period.
- Minimal local inflammatory response.
- No resistance.
- Must have action against common pathogens e.g.: aureus, s. epidermidis, coliforms & anaerobes.
- Must not significantly compromise mechanical integrity.

Dose of Antibiotic

The dosage of the antibiotic varies according to the use for which the cement is destined. Many authors argue that in case of **acute infections** high doses of antibiotics should be used: more than 2 g each 40 g of cement, usually from 6 to 8 g each 40 g, for a prolonged and effective release against pathogens Whereas if it is used **for prophylaxis** in first implants, where the first function of the cement is to fix the implant, the antibiotic can be mixed at low doses: less than 2 g each 40 g of antibiotic

Question 83 of 100

When using antibiotic-laden polymethylmethacrylate (PMMA) to treat osteomyelitis, vancomycin and tobramycin are heat stable and have not produced systemic toxicity at various levels. Which dose is closest to the highest recommended concentration for each drug?

1- 2 grams each per 40 mg PMMA
2- 3.6 grams each per 40 mg PMMA
3- 10 grams each per 40 mg PMMA
4- 20 grams each per 40 mg PMMA

PREFERRED RESPONSE: 3- 10 grams each per 40 mg PMMA

DISCUSSION

Vancomycin and tobramycin do not produce systemic toxicity in doses as high as 10.5 grams of vancomycin and 12.5 grams of tobramycin per 40 mg of PMMA.

RECOMMENDED READINGS

BONE CEMENT IMPLANTATION SYNDROME

- Hypotension
- Pulmonary hypertension
- Increased central venous pressure
- Pulmonary edema
- Bronchoconstriction
- Anoxia or hypoxemia
- Decreased partial end tidal carbon dioxide
- Cardiac arrhythmia
- Cardiogenic shock
- Transient decrease in arterial oxygen tension
- Hypothermia
- Thrombocytopenia
- Cardiac arrest
- Sudden death

LOCAL TISSUE EFFECT OF CEMENT

Heat of polymerisation: may exceed 67 degrees.

Occlusion of the nutrient metaphyseal vessels.

Cytotoxic and lipolytic effects.

Nerve injuries

Exothermic reaction

The polymerization process is an **exothermic** reaction, which means it produces heat. With a maximum *in vivo* temperature of 40°C to 47°C, this thermal energy is

dissipated into the circulating blood, the prosthesis, and the surrounding tissue. Once

polymerization ends, the temperature decreases and the cement starts to shrink

Exothermic reaction-cont..

During the exothermic free-radical polymerization process, the cement heats up. This polymerization heat reaches temperatures of around 82–86 °C in the body. The cause of the low polymerization temperature in the body is the relatively thin cement coating, which should not exceed 5 mm, and the temperature dissipation via the Available from

URL:http://en.m.wikipedia.org/wiki/Bone_cement. [Ref list]large prosthesis surface and the flow of blood.

BONE GRAFT

Material that assists and supports bone healing through its mechanical and or its biological properties

Indications
- structural stability
- Bone loss
- Enhance fracture healing
- non-union

Classification according to qualities

- **Osteoconductive**
  - Provides a three-dimensional scaffold for new bone to grow on e.g. cancellous bone graft
- **Osteoinductive**
  - Contains biological factors that stimulate bone growth and increase differentiation of undifferentiated mesenchymal cells into bone forming cells such as:
    - Bone Morphogenetic Protein (BMP)
    - Transforming growth factor beta (TGF-B) e.g. Allomatrix
- **Osteogenic**
  - Contains live mesenchymal cells, osteoblasts, and osteocytes cells that produce bone

Clinical picture of bone graft

Definition – material that assists with bone healing through its osteoconductive, osteoinductive and/or osteogenic properties.

Properties

1. Osteoconduction – process by which graft acts as three dimensional scaffold for ingrowth of capillaries and osteoprogenitor cells from recipient host bed. Examples – Calcium phosphate, Synthetic polymers.

2. Osteoinduction – process supporting differentiation of mesenchymal stem cells into osteoprogenitor cells capable of forming new bone. Examples – Bone Morphogenetic Proteins (BMP), Transforming growth factor-β, Platelet-derived growth factor.

3. Osteogenic – use of naturally occurring materials that induce or support formation of bone. Examples – autologous bone marrow graft, autologous/allogenic bone grafts

Classification

Type

1. Cortical- structural integrity
2. Cancellous- more porous compared to cortical therefore more rapid revascularisation
3. Corticocancellous

Vascularity

1. Vascularised graft – due to vascularity, more rapid healing and lower infection rate. Examples – vascularised fibular strut graft for tibial bone defects, vascularised distal radius graft for scaphoid non-union
2. Non-vascularised graft

Source

1. Autograft - same person
2. Allograft - same species (Isograft – twins)
3. Xenograft – different species
4. Synthetic

Preservation

1. Fresh – unprocessed; immunogenic
2. Fresh frozen – unprocessed stored at -70°C for minimum of 180 days; least impact on strength and preservation of immunogenicity and growth factors

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3. Freeze dried – processed to remove soft tissues and washed to deplete cell and marrow content followed by freeze drying; least immunogenic but lowest likelihood of disease transmission as irradiated

a regional anesthetic is converted to a general anesthetic and ORP may be requested to assist the anesthetist during this transition.

Tourniquet considerations

When a tourniquet is necessary for a surgical procedure, antibiotics should ideally be administered 10 minutes before inflation of the tourniquet cuff to assure a therapeutic blood level at the operative site. Muscle relaxation drugs should also be given before cuff inflation for optimal neuromuscular blockade. When the tourniquet is deflated there is a potential risk of the patient experiencing hypotension from sudden enlargement of the vascular bed as well as systemic acidosis due to the sudden release of anabolic waste products into the venous circulation. ORP should be aware of this and should be prepared to assist the anesthetist.

Potential complications

ORP should know the intraoperative complications specifically

Examination corner

Basic science oral 1

- What cell activity is demonstrated by a three-phase technetium bone scan?
- What malignant conditions might not show up on such a scan and why?

Candidate should be able to describe a three-phase bone scan, the isotope and the carrier. The uptake by osteoblasts during the third phase should be discussed. If there is no osteoblastic activity the scan will be negative in the third phase.

Basic science oral 2

- How can I tell whether an MRI scan is T1- or T2-weighted?
- What is the basic manner in which an MRI scan is obtained?

Tourniquets

Tourniquets can be useful in providing a bloodless field but there are a number of complications that can arise from their use. Proper use of tourniquets is an important aspect of patient care.

Tourniquets may be non-pneumatic or pneumatic:

- Non-pneumatic tourniquets are only used for short operations on the digits
- Pneumatic tourniquets may be non-automatic or automatic. The non-automatic types have a hand-operated pump and a pressure gauge and they cannot compensate automatically for leaks in the system. The automatic type operate from either an air line or an electric pump
- Tourniquets not attached to a fixed air line carry a risk of being inadvertently left in place at the completion of surgery.

Inflation pressure

- There is no absolute value for pressure of inflation; the surgeon should consider:
  - Age of patient
  - Condition of the soft tissues
  - Intercurrent medical conditions (especially vascular pathology)
  - The circumference of the limb
- In the upper limb the inflation pressure should be 50 mmHg higher than the systolic pressure
- In the lower limb the inflation pressure should be double the systolic pressure.

Contraindications

- Severe crushing injuries
- Sickle cell disease
- Peripheral vascular disease (relative).

Exsanguination

- Either by elevation or expression
- Expression should be avoided in the presence of venous thrombosis, malignancy or infection, all of which may be spread by embolism
- In frail patients cardiac arrest may occur from circulatory overload if both lower limbs are exsanguinated at the same time.

Complications

Local

- Compression neurapraxia
- Bone and soft-tissue necrosis
- Direct vascular injury
- Postoperative swelling and stiffness
- Delayed recovery of muscle power
- Wound haematoma
- Wound infection.

Systemic

- Cardiorespiratory decompensation
- Increased CVP
- Deep vein thrombosis
- Cerebral infarction
- Alterations in acid–base balance.

Tourniquet paralysis syndrome

- Caused by cuff pressure rather than ischaemia
- Flaccid motor paralysis with sensory dissociation
- Pain sensation often altered although temperature appreciation is usually preserved
- Colour, skin temperature and peripheral pulses are usually normal
- EMG – nerve conduction block at the level of the tourniquet
- May take up to 3 months to recover
- Nerves in patients with diabetes, alcoholism and rheumatoid arthritis have increased susceptibility.

Post-tourniquet syndrome (tourniquet-induced skeletal muscle ischaemia)

This is a reperfusion injury and is due to ischaemia. After release of the tourniquet the following occur:

- Oedema
- Stiffness
- Pallor
- Weakness
- Subjective numbness.

Myonephropathic metabolic syndrome

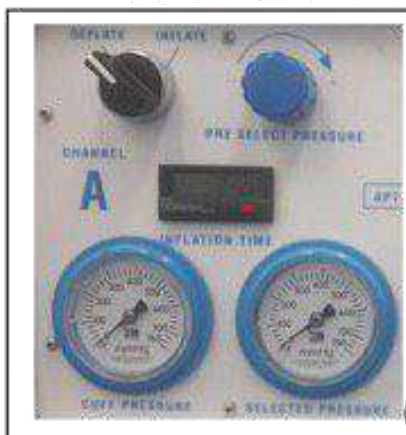
- Metabolic acidosis
- Hyperkalaemia
- Myoglobinuria
- Renal failure.

TOURNIQUETS

Device which is applied proximal to site of surgery to achieve bloodless field or to control hemorrhage

Types

1) Non-pneumatic on digits
2) Pneumatic Automatic timer and audio-visual alarm for abnormal pressure, air leakage and time



(Courtesy of F Arnaout)

Surgical Tourniquets in Orthopaedics

Inflation pressure

Factors taken into account - Age, skin condition, circumference of limb and co-morbidities

Cuff width: At least equal diameter of the limb
Wider cuffs require lower pressures to stop blood flow

Cuff length: 3 to 6 inches of overlap

Cuff pressure: There is no consensus regarding what pressure the tourniquet should be inflated to
Ideally as low pressure as will provide arterial and venous occlusion
Upper limb 50 – 75 mmHg above systolic pressure
Lower limb 100 – 150 mmHg above systolic pressure

LOP (Limb Occlusion Pressure):
Minimum pressure required to occlude arterial blood flow distal to cuff
Dependent on systolic BP, limb circumference and shape
Higher at subcutaneous tissues and mid-point of tourniquet

Tourniquet time The absolute limit of tourniquet time has never been firmly established, **2 hours** is the most widely accepted.

Exsanguination External compression using Reece-Davis exsanguinator or esmarch bandage
Simple elevation of the limb for one minute
Digital pressure over brachial artery in cubital fossa plus elevation
Contraindications: infection, malignancy

Padding: 2-3 layers of soft padding to distribute pressure evenly deep to cuff and to avoid pinching of skin

Contraindications
Infection, can enhance anaerobic infection
Peripheral vascular disease
Sickle cell disease - hypoxia, stasis and acidosis may lead to sickling
Poor skin
Crush injuries

Relative Contraindications
IM nailing to decrease thermal injury
Open fractures

Complications Nerves are most susceptible to mechanical pressure and muscle most susceptible to ischemia

Local	Increased with increasing time (2hr is the maximum)
	Post-tourniquet paralysis
	Bone and muscle necrosis
	Direct vascular injury
	Post-op swelling and stiffness
	Wound haematoma
	Re-perfusion injury **Post tourniquet syndrome**
	Characterized by Oedema, stiffness, pallor, weakness, numbness and pain
Systemic	Cardio-respiratory decompensation
	Increased CVP
	Altered Acid Base

TABLE 1-2

Braithwaite and Klenerman's Modification of Bruner's Ten Rules of Pneumatic Tourniquet Use

APPLICATION	Apply only to a healthy limb or with caution to an unhealthy limb
SIZE OF TOURNIQUET	Arm, 10 cm; leg, 15 cm or wider in large legs
SITE OF APPLICATION	Upper arm; mid/upper thigh ideally
PADDING	At least two layers of orthopaedic felt
SKIN PREPARATION	Occlude to prevent soaking of wool. Use 50-100 mm Hg above systolic for the arm; double systolic for the thigh; or arm 200-250 mm Hg, leg 250-350 mm Hg (large cuffs are recommended for larger limbs instead of increasing pressure)
TIME	Absolute maximum 3 h (recovers in 5-7 days) generally not to exceed 2 h
TEMPERATURE	Avoid heating (e.g., hot lights), cool if feasible, and keep tissues moist
DOCUMENTATION	Duration and pressure at least weekly calibration and against mercury manometer or test maintenance gauge; maintenance every 3 months

Modified from Kutty S, McElwain JP: Padding under tourniquets in tourniquet controlled surgery: Bruner's ten rules revisited, Injury 33:75, 2002.

ELECTROSURGERY

Transform electricity into heat
Cutting and coagulation of body tissue with high frequency current
Main current in UK is 240 Volts and 55 Hertz
Frequency > 100 kHz safe to avoid nerve and muscle stimulation – electrocution does not occur
Called Radio-Frequency because the frequency is similar to the domestic radio band
Active and return electrodes
Generator, foot/hand switch

Pad position can lengthen or shorten the circuit and increae or decrease impedence changing efficiency of operating electrode
10 000 times larger surface area than the electrode to reduce chance of burning
Shave underneath as hair reduce contact area
Apply on large smooth skin with large underlying muscle
Don't apply on a scar or bony prominences or over prosthesis

Types

- **Mono-polar** current established between diathermy electrode and electrical plate (large to minimise heating)
  Larger impedance – higher voltage (1000s)
  Heating inversely proportional to area of contact
  Cut continuous current
  Vaporises cells
  Pure narrow cut that can be used on skin
  Blended associated with coagulation
  Coag intermittent (pulsed) current at higher voltage
  Burns
- **Bi-polar** Current between two electrodes within diathermy forceps – lower impedance
  Lower voltage (100s)
  Safer - Avoids risk of damage from passage of current through surrounding tissues
  Use in - peripheral areas (fingers, toes, and penis) due to effect of channelling, Pacemaker

Use with pacemaker & ICD (Implantable Cardioverter Defibrillator)
Risk of electrical interference can lead to inhibition/increase in pacing
Preoperative check
Self-adhesive return electrode away from cardiac device implant site
Deactivate ICD and monitor with ECG and have external defibrillator in close proximity
Use magnet in emergency

Complications:
- Burn from contact with metal or alcoholic skin preparations
  Spirit-based skin preparation fluid should not pool and should be dry or dried before electrosurgery commences
  Current passes through passage of lower impedance such as metal
- Exposure to smoke products can contain infectious organisms
  Use high filtration face mask
- Explosion from anaesthetic or bowel gases

Electrosurgery

- An electric circuit is made involving the patient, where the patient is the point of current resistance, generating heat
- Frequency chosen is above 100 kHz to avoid nerve and/or muscle stimulation
- Monopolar electrosurgery involves an active electrode (high current density) at the surgical site and a return electrode elsewhere on the patient. The return electrode must be of large surface area to reduce the current density and avoid burns
- Waveforms
  - 'Cut' – involves continuous current to generate heat and vaporize tissue
  - 'Coag' – involves intermittent current (on for <10% of the time) with less heat and this permits a coagulum to form. If this waveform is used to cut, higher voltages are required with more surrounding tissue damage
  - 'Blend' – involves a longer 'on time' than coag
  - 'Fulguration' means the coagulation/charring of tissue over a wider area and employs a coag waveform with the diathermy point held slightly away from the tissue
- Safety:
  - 'Grounded system' – original technology; the risk of a return electrode being formed by patient contact

Traction

- Reduce pain
- Overcome deformity
- Keep fracture aligned and support movement while relieving pain
- **Types**
  - Skin
  - Skeletal
  - Fixed
- **Reduction Principles**
  - Use in the long axis of the limb
  - Align the fragment that can be controlled with the one that cannot be controlled
  - Reverse the mechanism that created the fracture
- **Skin Traction**
  - Applied over a large area of skin - spreads the load
  - Never more than 10lb (4.5 kg) or not more than 10% body weight
  - Contraindicated in vascular impairment, open fractures & marked shortening
- **Skeletal traction**
  - Used in lower limb fractures and cervical spine (Halo traction)
- **Fixed traction**
  - Force applied against a fixed point of the body
    i.e. ischial tuberosity
  - E.g.: Thomas splint and Hamilton Russell traction

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DRAINS

Cochrane – 2007 closed suction surgical wound drainage after orthopaedic surgery
No significant difference in incidence of wound infection, haematoma, dehiscence or re-operations between those allocated to drains and un-drained wounds
Blood transfusion required more frequently in those who received drains
Need for reinforcement of wound dressings and bruising were more common in group without drains

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Skin closure

Meta-analysis, BMJ (2010) After orthopaedic surgery, significantly higher risk of developing wound infection when wound closed with staples rather than sutures

- **Sutures**
  - Absorbable — Monofilament — PDS, Monocryl, Biosyn
    Braided — Vicryl
  - Non-absorbable — Monofilament — Nylon, Prolene
    Braided — Ethibond, Fiberwire

Dyed or undyed

Round bodied (for friable tissues) or cutting needle (for closure of wounds)

- **Donati-Allgöwer suture pattern**
  Mattress suture with one side in subcutaneous tissue
  Does not result in kinking or folding of skin and, thus, does not compromise blood flow

Plaster of Paris :

- Cast uses

1. To support # bones .
2. To stabilize & rest Joints in lig. Injury.
3. To support & immobilize J. & limbs post op until healing has occurred .
4. To correct deformity .
5. To ensure rest of infected tissue.
6. To make a negative mould of apart of body .

- Consist of:

- Roll of muslin stiffened by dextrose or starch & impregnated w̄ the hemihydrate of calcium sulfate .

- 2 (CaSo4 & ½ H2o) 3 H2o 2 (CaSo4 2H2o) + heat.

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- When water added , calcium sulfate takes heat up its water of crystallization .

- Heat : 5- 50 co

- Times

1. Setting time : time taken to change from powder form to crystalline form (3-9 min)

2. Drying time : time takes to change from crystalline form to anhydrous form (24- 72 hr) .

- Factors that ↓ setting time :

1. Hot water
2. Salt
3. Resin
4. Borax

- Factors ↑ setting time :

1. Cold water
2. Sugar

- Forms :

1. Slab
2. Encircle cast
3. Spica
4. brace

- advantages :

1. cost effective
2. non allergic
3. easily moulded

- dis advantages :

1. radio opaque
2. heavy
3. easily breake when contact $\bar{w}$ water

- rules of application :

1. 8 inch forthigh, 6 inch for leg , 4 inch for arm & forearm
2. Padding from distal to proximal, minimum 2 layers
3. 1 J. abore & 1 J. below
4. Mould $\bar{w}$ palm
5. Dip pop vertically in water till air bubble ceases to come

- After care :

1. Keep plaster dry

2. Mobilize all J. which are not incorporated in plaster

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3. Come immediately if you develop - swelling

- pain

- Bluish or whitish discoloration of fingers or toes

4. Notice any cracks in plaster

5. Physiotherapy of m.s $\bar{w}$ in plaster & J. out side plaster to ensure early rehabilitation .

- Complications :

1. Due to tight cast

- Pain

- pressure sores

- compartment syndrome

- peripheral n. injury

2. due to plaster allergy

- allergic dermatitis

3. due to improper application

- J. stiffness

- Plaster blister & sores

- Breakage

4. Burn due to exothermic reaction

↳ - temp elevated can be due to plaster being dipped too briefly & water sequeezed to aggressively out of plaster.

- so if water not enough or squeezed plaster get hotter .

- Lite Cast :

- Fibre glass & poly urethane resin

- Advantage :

1. Water proof
2. Harder
3. Many colors & shaper
4. Lighter
5. Short setting time

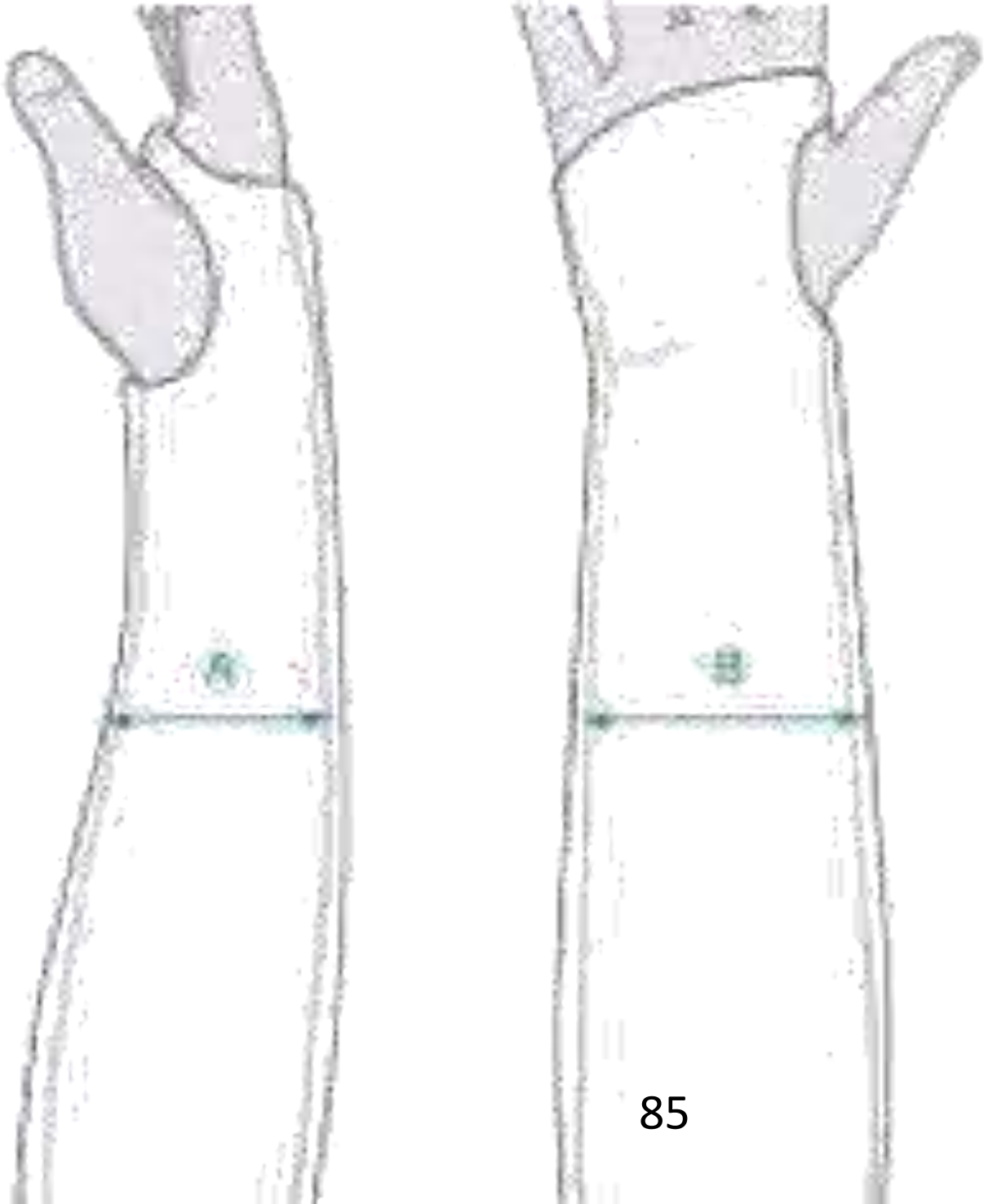
- Dis advantage :

1. Cant mould
2. Tension free
3. Sharp edges

	Description	Cut-off Values*
Cast index	Inner diameter of cast on lateral (at fracture site)/inner diameter of cast on AP (at fracture site)	0.8
Padding index	Dorsal gap on lateral (at fracture site)/ maximum interosseus space on AP	0.3
Canterbury index	Cast index + padding index	1.1
Second metacarpal-radius index	Angle created by bisection of the long axis of the second metacarpal and long axis of the radius on AP radiographs	$> 0^{\circ}$
Gap index	[(Radial fracture-site gap + ulnar fracture-site gap)/inner diameter of cast in AP plane] + [(dorsal fracture-site gap + volar fracture-site gap)/inner diameter of cast in lateral plane]	0.15
Three-point index	[(Distal radial gap + ulnar fracture-site gap + proximal radial gap)/contact between fracture fragments in transverse projection] + [(distal dorsal gap + volar fracture-site gap + proximal dorsal gap)/contact between fracture fragments in sagittal projection]	0.8

*All indices except the second metacarpal radius angles are ratios and therefore do not have units applied to them.

AP indicates anteroposterior.



$$\text{CAST INDEX} = \frac{A}{B}$$

* start with % of Local Anasthetic.

if [A%], then each 1 cc has A x 10 mg concentration.

ex:- In 2% lidocaine, 1 cc has 20mg of lidocaine.

In 1% Lidocaine, 1cc has 10mg of lidocaine.

In 0.5% lidocain, 1cc has 5mg of lidocaine.

* The given does for pt is according to wt.

[1] lidocaine: 3-5 mg/kg if was given without epinephrine.
(30sec)

7 mg/kg if " " with epinephrine.

[2] Marcaine (=Buvocaine.) : 2mg/kg with or without epinephrine.
(2-4 min)

ex: 70 kg pt

How many ~~ml~~ (ml) of lidocaine 2% to be given?

70 X 3 = 210 mg ~~=~~ the required concantration to be given.

For 2% lidocaine, each 1 ml has 20mg lidocaine.

so,

1 ml → 20
X ml → 210 ⇒ $X = \frac{210}{20}$ = [10 mL of 2% lidocaine]

* Onset of Action for:-

lidocaine: 30 seconds.

Marcaine:- 2-4 minutes. } → if given together, Marcaine will delay the onset of action of Lidocaine, so it's pretred not to be given together.

AMIDE

Marcain : 2ml/kg (2 - 4 min)

Concentration: 0.5%

0,5% ⇒ 5mg

ex: 50kg ⇒ 50 x 2 = 100mg

100mg / 5 = 20mL

adrenalin dose:-

1/100.000

1ampul ⇒ 1mg

1 mg ⇒ 1/1000

+ 10cc NS ⇒ 1/10.000

+ 10cc NS ⇒ 1/100.000

Ready

Complications of local anesthesia

Local complications

- Needle breakage
- Persistent anesthesia or paresthesia
- Facial nerve paralysis
- Trismus
- Soft-tissue injury
- Hematoma
- Pain on injection
- Burning on injection
- Infection
- Edema
- Sloughing of tissues
- Post-anesthetic intraoral lesions

Systemic complications

- Vasodepressor syncope
- Over dosage (toxic reaction)
- Allergy
- Psychogenic reactions

- Local anesthetic systemic toxicity (LAST)
  - intravascular bupivicaine
    - effect
      - CNS
        - seizures, coma, respiratory arrest
      - CVS
        - asystole, ventricular fibrillation, cardiac arrest
    - antidote
      - intravenous 20% lipid emulsion
- Bone cement implanation syndrome
  - associated with use of bone cement during joint arthroplasty procedures
  - symptoms
    - hypotension
    - hypoxemia
  - treatment
    - intravenous fluids
    - vasopressors
    - 100% inspired oxygen

Numerous steps have been described to decrease radiation in the operating room.
They include:

1. Decrease time of exposure
2. Decrease dose of exposure
3. Beam collimation
4. The use of mini C arm
5. Inverting the C arm (shortens distance between body part and collection surface)
6. Surgeon's control of the C arm
7. Use of protective equipment (glasses, thyroid shields, aprons)
8. Increase the distance from the X ray source

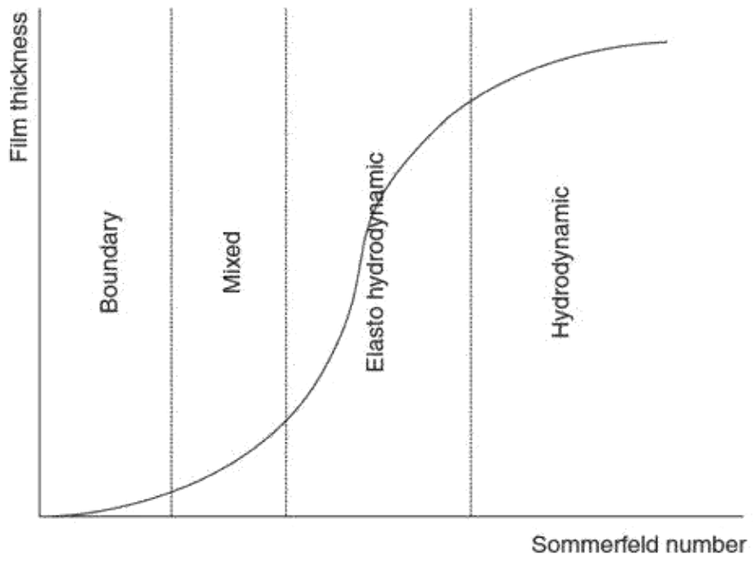


Figure 22.12 Lubrication.

- Has unique fluid properties conferred by the hyaluronic acid (it is a non-Newtonian fluid)
  - When the shear rate is varied, the shear stress does not vary in the same proportion (or even necessarily in the same direction)
  - Exhibits pseudoplasticity; a decrease in viscosity when the shear rate increases
  - Exhibits thixotropy; a time-dependent decrease in viscosity under constant shearing
- In conditions where hyaluronic acid is reduced (such as in rheumatoid arthritis or after joint replacement), the lubricating properties are impaired
- The **Sommerfeld number** is a property of a given lubricant = viscosity × velocity/stress and describes the relationship between the lubricant, the fluid film thickness and the potential types of lubrication.

Wear

Lubrication and wear are closely related topics, and questions on both frequently arise in the basic science oral.

- Wear is the progressive loss of material from the surface of a body owing to relative motion at that surface
- Wear generates further 'third body' wear particles
- The softest material is worn.

Modes of wear in artificial implants

- **Mode 1** wear occurs between the two surfaces that are articulating together in the manner intended by the designer
- **Mode 2** wear occurs between a bearing surface and a non-bearing surface
- **Mode 3** wear occurs as a result of third body particles coming between the bearing surfaces
- **Mode 4** wear occurs between two non-bearing surfaces.

Wear mechanisms

Wear is usually either mechanical or chemical. Mechanical wear mechanisms include abrasive, adhesive, fatigue and third body wear. Chemical mechanisms include corrosion and corrosion fretting.

The surface roughness of the two materials influences the amount of mechanical wear.

Abrasive wear

- Asperities on the harder material come into contact with the softer material
- The harder material asperities plough and cut the softer surface, causing grooves and detached particles
- The detached particles become third bodies, causing further wear.

Adhesive wear

- The softer surface forms a bond with the harder surface
- The intermolecular bonds cause friction and, if the junction is stronger than the cohesive strength of the softer bearing material, fragments of the softer material become adherent to the harder material or become smeared
- Tends to cause steady low rate wear.

Fatigue wear

- Repetitive/cyclical stressing of the asperities causes accumulation of microscopic damage
- Decreased by greater conformity of the bearing surfaces
- Decreased by thicker bearing surfaces (in UHMWPE) because of less concentration of shear stresses near the surface
- Mainly a problem in knee replacements where there are less conforming joint surfaces.

Third body wear

- Particles become trapped between the articulating surfaces
- Very high local stresses produced
- Cause localized abrasive and fatigue wear.

Fretting

- Localized wear from relative motion over a very small range
- Can produce a large amount of debris.

Corrosive wear

- Can be exacerbated by mechanical factors such as abrasion removing the passivating layer or corrosion within the crevasses caused by fretting.

Measurement of wear

Linear wear and volumetric wear are two methods of measuring wear in an implant. The mechanisms of wear are the same in both.

- Low wear rates have been reported with the use of radial clearances that are small enough to lead to a small amount of bedding-in and for efficient fluid film lubrication but large enough to avoid binding. High tolerance manufacture is required
- The procedure preserves bone on the femoral side
- The orientation of the acetabular component may be critical and the operation may therefore be more surgeon-dependent than some other techniques
- There are concerns over the potential effects of metal ions with some implants
- Severe local reactions (ALVAR) have been reported in a proportion of cases.

Corrosion[28]

Corrosion is the reaction of a metal with its environment, resulting in its continuous degradation to oxides, hydroxides or other compounds.

Passivation

- An oxide layer forms on the alloy surface; strongly adherent; acts as a barrier to prevent corrosion. It can be jeopardized by mechanical wear such as fretting or abrasion.

Types of corrosion

- Uniform attack
- Galvanic
- Crevice
- Pitting
- Fretting corrosion
- Intergranular
- Leaching
- Inclusion
- Stress.

Uniform attack

- Most common type of corrosion
- Occurs with metals in electrolyte solution uniformly affecting the entire surface of the implant.

Galvanic corrosion

- Two dissimilar metals are electrically coupled together
- An anode and cathode form, creating in essence a small battery as ions are exchanged.

Crevice corrosion

- Occurs in a crevice or crack where there is low O_2
- The tip of the crack is unable to passivate because of lack of O_2
- Accelerated by high concentration of H^+ and Cl^-.

Pitting

- Localized form of corrosion in which small pits or holes form
- Similar to crevice corrosion but the corrosive attacks are more isolated and insidious
- Dissolution occurs within the pit.

Fretting corrosion

- Synergistic combination of wear and crevice corrosion between two materials in contact
- Relative micromovement between the two materials removes the passivating layer
- Can cause permanent damage to the oxide layer and particles of metal and oxide can be released by the fretting.

Intergranular corrosion

- Metals have a granular structure (see biomaterials section)
- As a metal cools during manufacture, impurities and additional trace metals crystallize differentially in different grains and this allows galvanic currents between the grains
- Intergranular corrosion occurs at grain boundaries.

Leaching corrosion

- Similar to intergranular corrosion but the result of electrochemical differences within the grains themselves.

Inclusion corrosion

- Occurs because of impurities left on the surface of the materials (such as metal fragments from a screwdriver)
- Similar to galvanic corrosion.

Stress corrosion

- Metals that are repeatedly deformed and stressed in a corrosive environment show accelerated corrosion and fatigue damage
- Stainless steel is particularly prone to stress corrosion cracking.

Examination corner

Basic science oral 1

- Candidate is shown a picture of a worn hip replacement
- Question – what are the different mechanisms of wear in an artificial joint?

Basic science oral 2

- Theoretically how might lubrication in an artificial joint differ from lubrication in a synovial joint?

This question allows an exploration of the differences between elastohydrodynamic lubrication, weeping and boosted lubrication as may occur in synovial joints, and hydrodynamic lubrication and squeeze film as may occur in artificial joints.

Classes of levers:

Rigid structure that turns around fixed point (e.g. bones)

Moment arm: Perpendicular distance from pivot point (fulcrum) to line of action of force

Classes of levers:

- 1st class Fulcrum between force and load
  Atlanto-occipital joint between erecter spinae and head
  Scissors
- 2nd class load between force and fulcrum
  Body weight on ankle between calf muscles and toes when standing on tip toes
  Nut cracker
- 3rd class force between fulcrum and load
  Elbow flexion muscles between elbow and hand

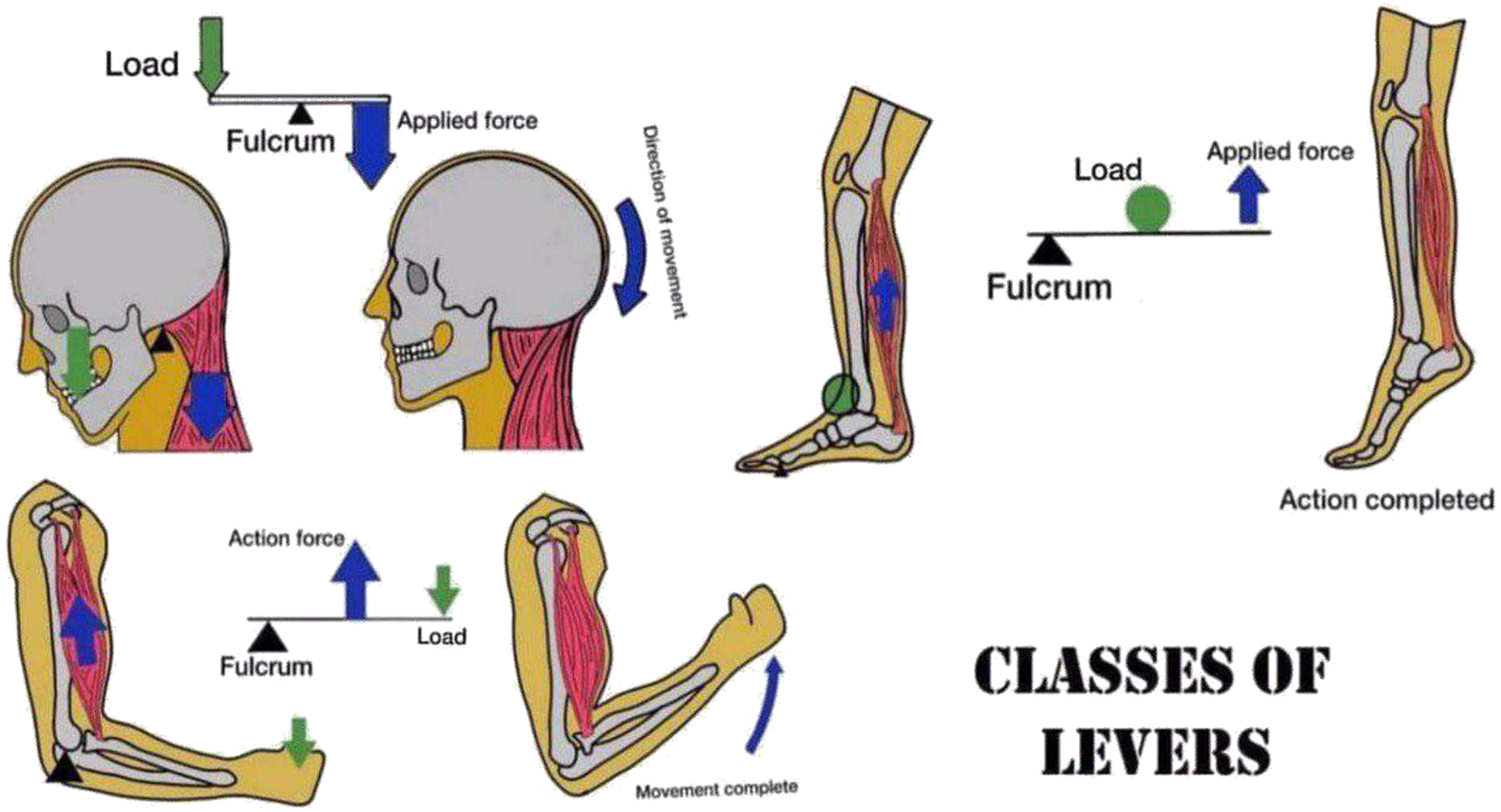


Table. Budapest Criteria for CRPS

All of the following statements must be met:

- The patient has continuing pain that is disproportionate to any inciting event
- The patient has a least 1 sign in 2 or more of the categories below
- The patient reports at least 1 symptom in 3 or more of the categories below.
- No other diagnosis can better explain the signs and symptoms.

No.	Category	Signs/Symptom
1	**Sensory**	Allodynia (pain to light touch and/or temperature sensation and/or deep somatic pressure and/or joint movement) and/or hyperalgesia (to pinprick)
2	**Vasomotor**	Temperature asymmetry and/or skin color changes and/or skin color asymmetry
3	**Sudomotor/edema**	Edema and/or sweating changes and/or sweating asymmetry
4	**Motor/trophic**	Decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair/nail/skin)

Based on reference 3.

synthesis 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

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

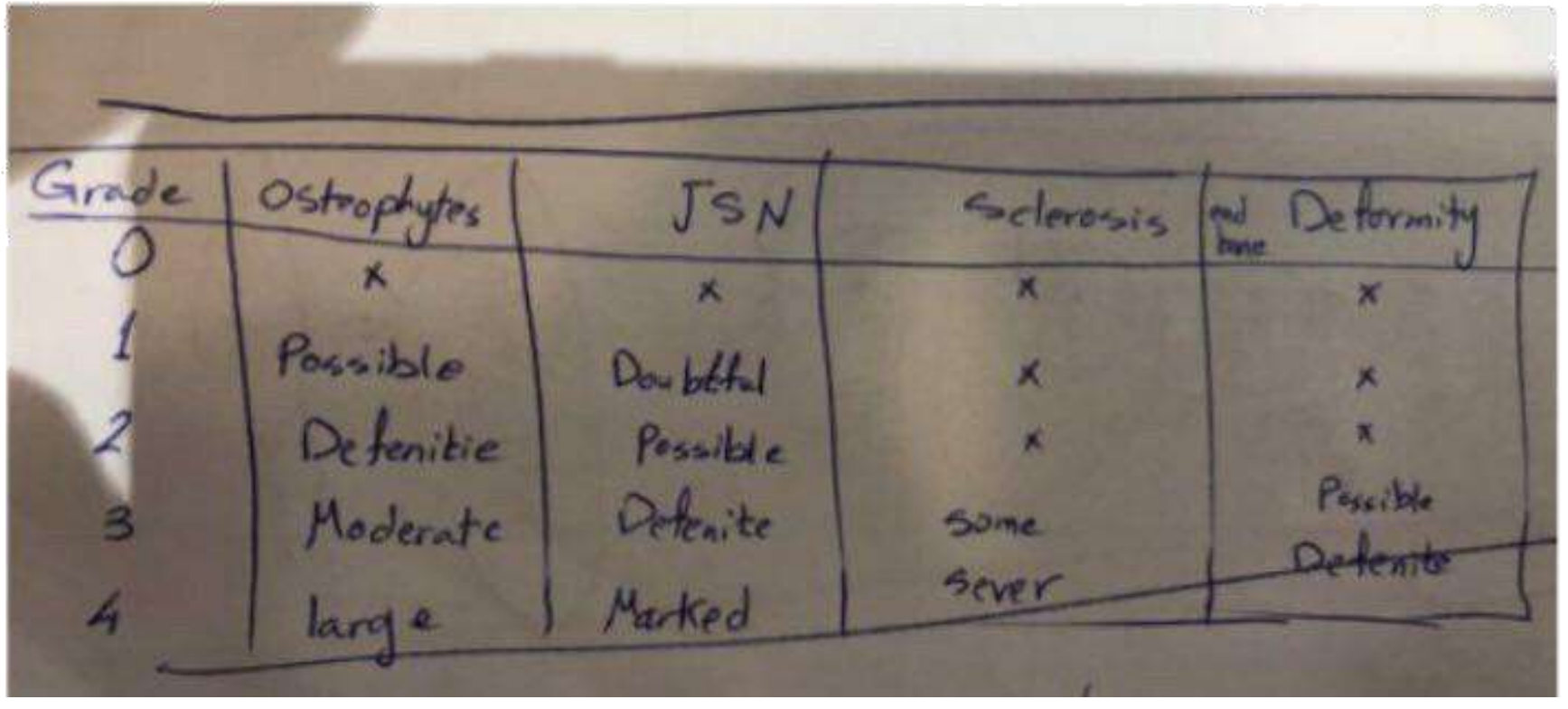
Marginal osteophytes

Subchondral cysts

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

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



Grade	Ostrophytes	JSN	Sclerosis	end bone Deformity
0	x	x	x	x
1	Possible	Doubtful	x	x
2	Definitie	Possible	x	x
3	Moderate	Definite	some	Possible
4	large	Marked	sever	Definite

	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

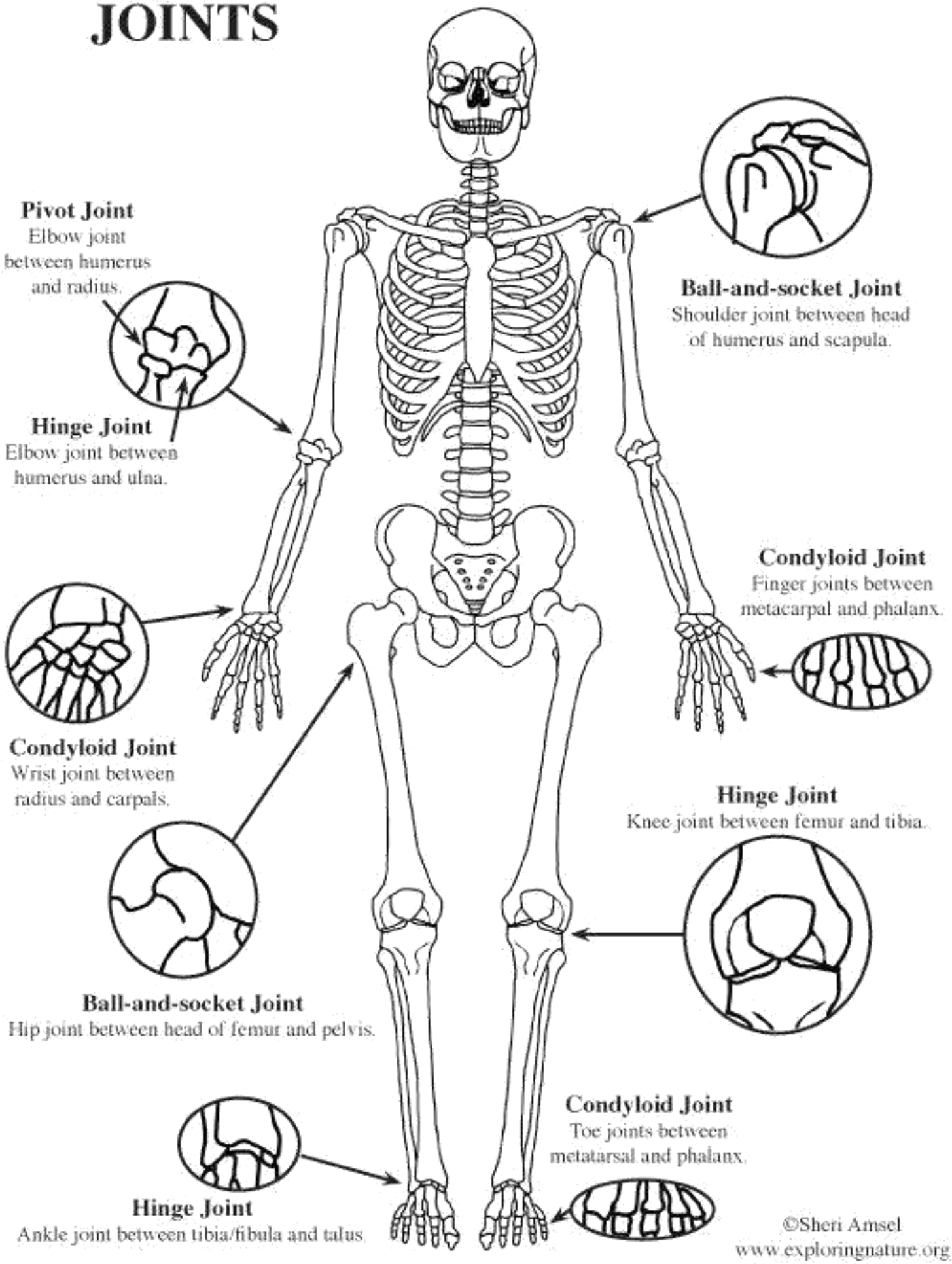
Effect of Aging

↑ 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

JOINTS



Pivot Joint
Elbow joint between humerus and radius.

Hinge Joint
Elbow joint between humerus and ulna.

Ball-and-socket Joint
Shoulder joint between head of humerus and scapula.

Condyloid Joint
Finger joints between metacarpal and phalanx.

Condyloid Joint
Wrist joint between radius and carpals.

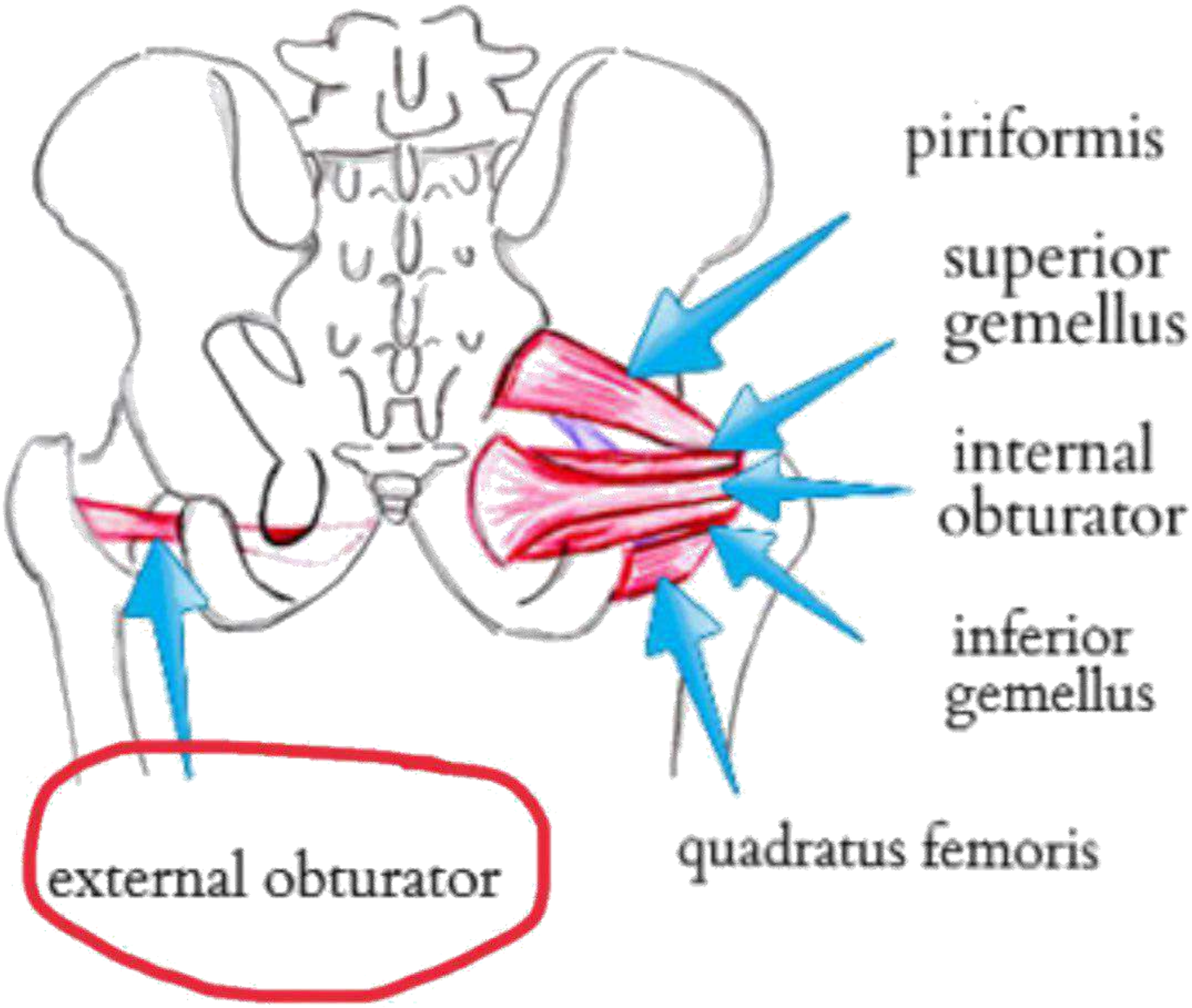
Hinge Joint
Knee joint between femur and tibia.

Ball-and-socket Joint
Hip joint between head of femur and pelvis.

Condyloid Joint
Toe joints between metatarsal and phalanx.

Hinge Joint
Ankle joint between tibia/fibula and talus

External Hip Rotators



- Tensor fascia lata :- Muscle extending from ASIS to ITB

- Fascia lata : Fascia covering the whole thigh composed of deep & superficial layers

- ITB :- Condensation of Tensor fascia lata inserted into Gerdy tubercle

- Three-phase bone scan
  - indications
    - can help to rule out CRPS type I (has high negative predictive value)
  - phases
    - phase I (2 minutes)
      - shows an extremity arteriogram
    - phase II (5-10 minutes)
      - shows cellulitis and synovial inflammation
    - phase III (2-3 hours)
      - shows bone images
    - phase IV (24 hours)
      - can differentiate osteomyelitis from adjacent cellulitis
  - findings
    - increased uptake in all phases
    - phase III is most sensitive

7 dehydrocholesterol

SKIN

cholecalciferol

(1)

"vit D"

LIVER

→ 25OH vitamin D

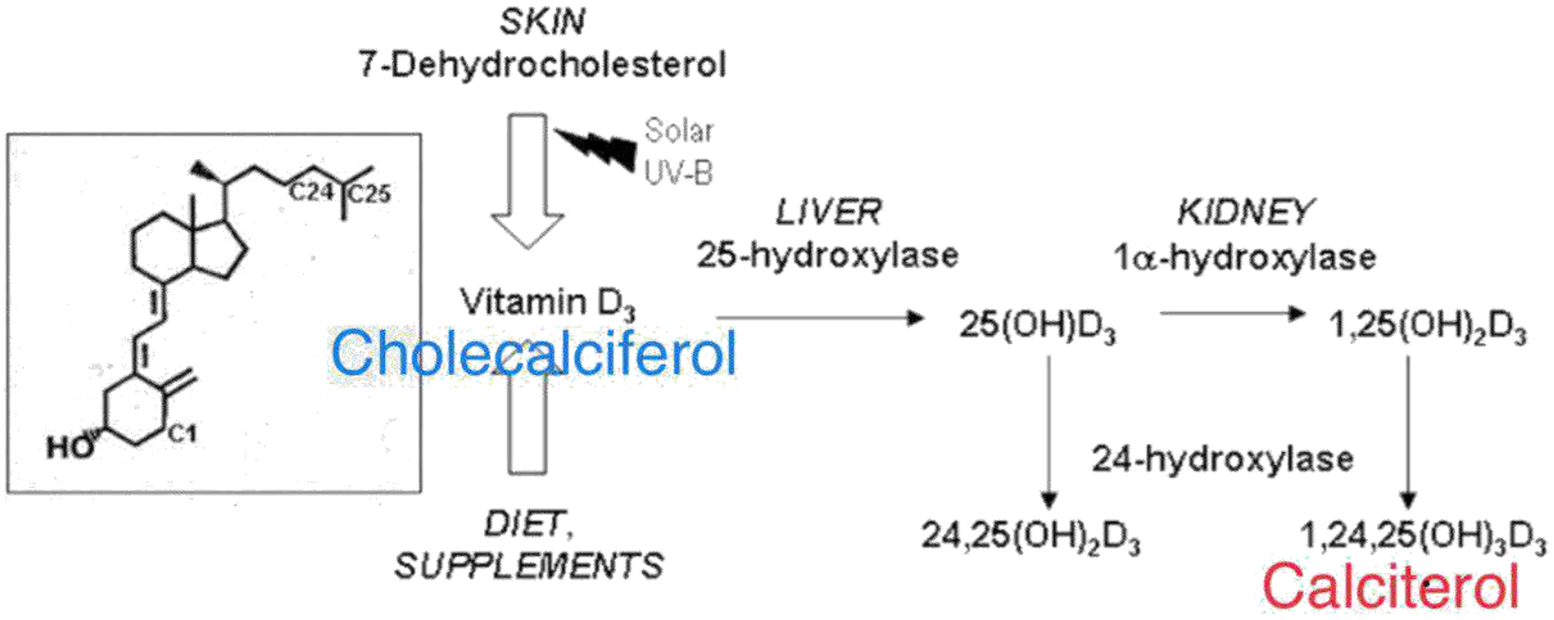
xylase

Mg

KIDNEY

→ 1,25OH Vitamin D

Calcitriol


SKIN
7-Dehydrocholesterol
Solar
UV-B
C24
C25
HO
C1
Vitamin D3
Cholecalciferol
DIET,
SUPPLEMENTS
LIVER
25-hydroxylase
KIDNEY
1α-hydroxylase
25(OH)D3
1,25(OH)2D3
24-hydroxylase
24,25(OH)2D3
1,24,25(OH)3D3
Calciterol

	Diseased (TD)	**Healthy (TH)**	
Test Positive	True positive (T_P)	False Positive (F_P)	$\text{PPV} = \frac{(TP)}{(TP)+(FP)}$
Test Negative	False Negative (F_N)	True negative (T_N)	$\text{NPV} = \frac{(TN)}{(FN)+(TN)}$
	$\text{Sensitivity} = \frac{(TP)}{(TP)+(FN)}$	$\text{Specificity} = \frac{(TN)}{(FP)+(TN)}$	

2.Trauma:

A- Oral

1. **Evaluation, Resuscitation & DCO:**

Golden Hour

- period of time when life threating and limb threatening injuries should be treated in order to decrease mortality

Damage control orthopedics

temporary stabilization of skeletal fractures in patients whom the initial insult of trauma has compromised the ability of them to undergo the physiologic stress of definitive treatment to avoid 2nd Hit

DCO : (delay of definitive treatment until physiology has improved)

Early Total Care (ETC),

fixing long bone fractures as soon as possible because patients were "too sick not to operate" which led to exacerbation of the "second-hit" in patients with hemodynamic instability, head, and/or chest injuries

Optimal time of surgery :

1. acute inflammatory window (period from 2 to 5 days characterized by a surge in inflammatory markers) : patient are at increased risk of ARDS and multisystem failure during

2.window of opportunity : its an immunological window between day 5 and day 10 after trauma used for defnitive stabilization of fractures.

3.The period of immunosuppression lasts for about 2 weeks, so that secondary reconstructive procedures can be planned for the third week after trauma.

Early appropriate care :

identifies major trauma patients and definitively treats the most time-critical orthopaedic injuries while **minimizing the secondary inflammatory response**, guided by laboratory parameters of **adequate resuscitation** (oxygenation)

This means return to normal oxygenation, vital signs, temperature, coagulation, and normal microcirculation resulting in aerobic metabolism, as measured by pH, base defcit, and venous lactate

Adult Respiratory Distress Syndrome

Acute Respiratory Distress Syndrome (ARDS) results from acute lung injury that leads to

- non-cardiogenic pulmonary edema
- respiratory distress
- refractory hypoxemia
- decreased lung compliance

Osteomyelitis

Infection of **bone & bone marrow** characterized by progressive inflammatory destruction and apposition of new bone

Biofilm :

A structured community of bacterial cells **enclosed** in a self produced polymeric matrix entering in a **no-growth, or sessile, phase** and adherent to inert or living surface , made of **85 %** exopolysaccharide glycocalyx & **15 %** bacterial cells which makes them **more resistant to antibiotics** occuring **within 4 weeks**

sequestrum: devitalized bone that serves as a nidus for infection
involucrum**:** formation of new bone around an area of bony necrosis

Septic arthritis

Intra-articular infection which may cause articular surface damage due to:
1.release of proteolytic enzymes (matrix metalloproteinases) from inflammatory and synovial cells, cartilage, and bacteria
2.increased joint pressure may cause osteonecrosis if not relieved within 8 hours

Necrotizing Fasciitis

Necrotizing fasciitis is a life threatening infection that spreads along soft tissue planes

Gas Gangrene : also called clostridial myonecrosis
Clostridium perfringens (most common), Clostridium novyi, Clostridium septicum

triad of death" reflects inadequate resusitation and is characterized by:

- acidosis
- hypothermia
- coagulopathy

Transient Synovitis of Hip

inflammation of the synovium and a common cause of hip pain in pediatric patients which occurs due to non-specific inflammation and hypertrophy of the synovial lining/membrane which is related to Hx of Viral(URTI) / bacterial infection (poststreptococcal toxic synovitis)

Psoas Abscess

Ultrasound diagnostic imaging study of choice

Brachial Plexus Injuries

The brachial plexus is a network (plexus) of nerves (formed by the **anterior ramus** of the **lower four cervical nerves** and **first thoracic nerve** (C5, C6, C7, C8, and T1). This plexus extends from the spinal cord, through the cervicoaxillary canal in the neck, over the first rib, and into the armpit. It supplies afferent and efferent nerve fibers to the chest, shoulder, arm, forearm, and hand

Heterotopic ossification :

The inappropriate formation of mature lamellar bone in nonosseous locations.

Morel-Lavallée:

a closed traumatic soft tissue degloving injury characterized by separation of the dermis from the underlying fascia due to a shearing force

Timing	Resuscitation state/physiological status	Surgical intervention
Day 1	Normal, no fluctuation	Early total care
	Transient response	Damage control
	No response-inotropes	Life-saving surgery
Day 2–3	Hyperinflammation (↑SIRS)	"Second look" only
Day 4–10	Safe window of opportunity	Definitive surgery
Day 11–21	Immunosuppression (↑CARS)	Avoid surgery
Day 22+	Normal physiology	Secondary reconstructive surgery

Variables	Score
Skeletal/soft-tissue injury	
Low energy (stab; simple fracture; pistol gunshot wound)	1
Medium energy (open or multiple fractures, dislocation)	2
High energy (high speed MVA or rifle gunshot wound)	3
Very high energy (high speed trauma + gross contamination)	4
Limb ischemia	
Pulse reduced or absent but perfusion normal	1[a]
Pulseless; paresthesias, diminished capillary refill	2[a]
Cool, paralyzed, insensate, numb	3[a]
Shock	
Systolic BP always >90 mmHg	0
Hypotensive transiently	1
Persistent hypotension	2
Age (years)	
<30	0
30–50	1
>50	2

Note: [a]Score doubled for ischemia >6 hours.

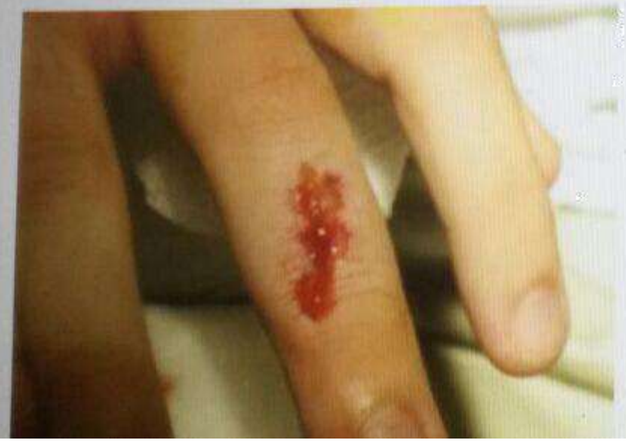
Abbreviations: BP, blood pressure; MVA, motor vehicle accident.

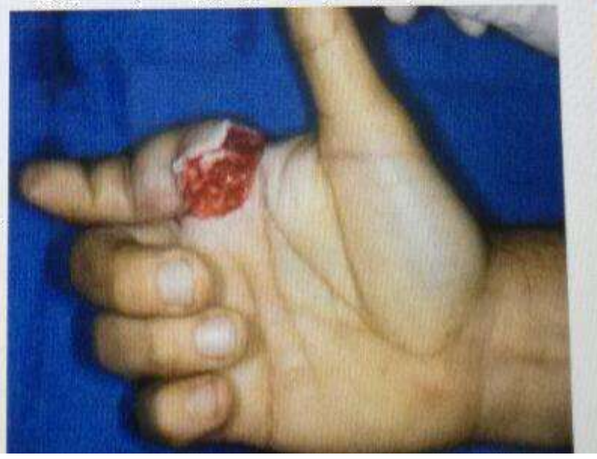
Classification

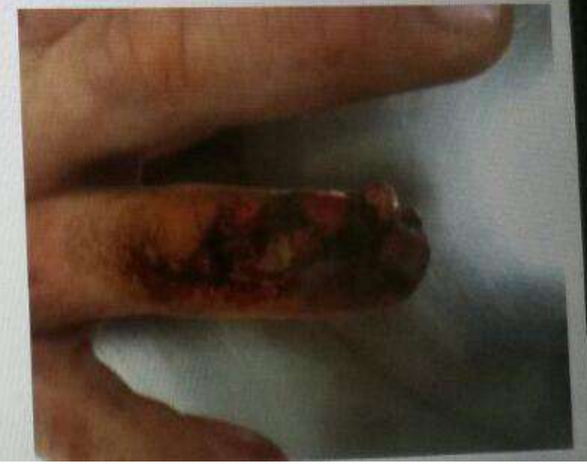
The McLain et al[34] Modified Gustilo Classification for Open Hand Fractures

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".







4. What initial management steps would you take in A & E?
Answer: Advanced trauma life support resuscitation. Analgesia, wound swab, photograph, early broad spectrum intravenous antibiotics (Cephalosporins - first generation combined with aminoglycosides. Farm yard injuries—add Penicillin), tetanus prophylaxis, irrigate to remove gross contamination, macroscopic debris, splint limb and plan for theatre. + Neurovascular examination
+realignment the limb in better position to prevent NV and skin compromise

	Clean Minor Wounds		All Other Wounds	
	Give Td	Give TIG	Give Td	Give TIG
Vaccination history unknown or < 3 doses	Yes	No	Yes	Yes
Vaccination history ≥ 3 doses	Only if last dose received ≥10 years ago	No	Only if last dose received ≥ 5 years ago	No

Td = tetanus and diphtheria vaccine; TIG = tetanus immune globulin

Immunization History	Clean, Minor Wound (GA I)	GS II and III
Unknown History or <3 doses	• Give vaccine only	• Give vaccine • Give immune globulin
Vaccination complete (3 prior doses)	• No prophylaxis if last dose within 10 years • Give vaccine if >10 years since last dose	• No prophylaxis if last dose within 5 years • Give vaccine if >5 years since last dose

TETANUS PROPHYLAXIS

- **Tetanus**
  - Caused by tetanospasmin
  - Exotoxin of anaerobic Gram +ve rod (bacillus) – Clostridium Tetani which is found in soil
  - Neuroparalytic disease
  - Toxoid is booster -TTB (inactivated vaccine) & IGg is active immunity

- **DOH guidelines**

• Full course & booster <10 years	Nil if clean	TTB if dirty
• Full course & booster > 10 years/non-immunised	TTB if clean	TTB if dirty

Clean wound is defined as wounds less than 6 hours old, non-penetrating with negligible tissue damage

Compartment syndrome

This is a very important topic and you *must* be able to explain the mechanism clearly.

Compartment syndrome is defined as increased pressure in an enclosed osteofascial space resulting in decreased capillary perfusion below that necessary for sustained tissue viability.

Normal compartment pressures are of the order of 5 mmHg.

Two possible mechanisms can precipitate compartment syndrome:

1. Increased content within the compartment – haemorrhage, ischaemic swelling, reperfusion injury, etc.
2. Decreased space – tight cast, premature closure of fascia.

The final common pathway involves:

1. Compartment pressure exceeds the venule/venous pressure.
2. The venous outflow from the compartment is impaired and tissue ischaemia follows.
3. Pressure within the compartment increases whilst arterial inflow is not impaired.
4. As compartment pressure approaches systolic pressure, flow into the compartment will cease.

NB. It is important to be aware that at a pressure well below arterial pressure there will be no perfusion of the tissues within the compartment although at such a pressure there will still be a distal pulse.

Clinical presentation

- Pain out of proportion to the injury
- Pain on passive movement (pain on stretching the muscles of the compartment)
- Compartment palpably tight
- Paraesthesia
- Paralysis
- Pallor of the extremity
- Pulseless
- 'Perishing cold'.

Of the above, disproportionate pain, pain on passive movement and a tight compartment on palpation are the most important as all the others are *too late* and tissues may necrose even though a pulse is still present distally

- At 1 hour of ischaemia, a reversible neurapraxia develops
- At 8 hours of ischaemia, axonotmesis occurs.

Measurement

Compartment syndrome is a clinical diagnosis except when pain cannot be assessed (e.g. in impaired consciousness or in the presence of regional anaesthesia), when a pressure monitor can be used.

- A catheter/needle and pressure transducer are used with simultaneous blood pressure measurement

- In trauma the measurement is taken within the zone of injury and should be undertaken in all relevant compartments
- The threshold can be an absolute value of 30 mmHg or pressure within 20–30 mmHg of diastolic blood pressure – Edinburgh group.

Fasciotomies

- A diagnosis of acute compartment syndrome is a surgical emergency
- Leg compartments – two-incision technique (anterolateral and medial) is used to decompress all four compartments
- Forearm – volar decompression to include carpal tunnel, then check the dorsum and decompress if necessary
- Foot – several different methods have been described, depending on the part of the foot affected (i.e. if calcaneal fracture or not) – the usual method is two dorsal incisions medial to the second metatarsal and lateral to the fourth metatarsal, respectively, to decompress the interossei, and the lateral compartment and a medial incision to decompress the medial, central ± calcaneal compartments (the latter through a more posterior medial incision)
- Closure – not before 48 hours; there is a low threshold for skin grafts rather than delayed primary closure
- Late presentation – once muscle necrosis is established there is no indication for fasciotomy, which may lead to rhabdomyolysis and infection. The definition of 'late' is controversial.

Acute compartment syndrome

- Definition – increased intracompartmental pressure within a fascio-osseous compartment that requires surgical release to prevent muscle necrosis or permanent damage to nerves in the compartment
- Causes – long bone fractures (closed or open), crush injury with or without fractures, tight splints or casts, burns, electrocution, infection, snake bite, arterial injury and clotting disorders
- Pathophysiology – tight compartments – increased interstitial pressure – reduced venous outflow – further increasing interstitial pressure (vicious cycle) – critical interstitial pressure is reached – cellular level hypoxia owing to reduced inflow and outflow
- Clinical features – pain out of proportion to injury or increasing pain or increasing requirement of analgesia; stretch pain – stretching the muscles of the affected compartment, causing pain. For example, if the deep posterior compartment of the calf is to be tested then passive extension of interphalangeal joints of all the toes should be performed, not passive extension of the ankle. Any movement of the ankle in a tibia fracture is bound to produce pain and this could be confused with compartment syndrome. The involved compartment is usually tense and tender. Other features tend to occur later in the pathological process of compartment syndrome – pulseless, pallor, paraesthesia and muscle weakness.

Difficulties in clinical assessment of compartment syndrome occur in unconscious patients or in polytraumatized patients or with nerve injuries or blocks.

Under these conditions or when diagnosis is doubtful, measuring compartment pressures with a slit catheter (slit to reduce risk of blockade of catheter) or pressure transducer is helpful. Continuous or serial monitoring gives a better picture of an evolving condition rather than a one-off measurement. One-off measurements of all compartments separately could be performed but, if continuous monitoring is considered, the transducer tip should be as close as possible to the fracture.

Delta pressure is the difference between diastolic pressure and compartment pressure. A delta pressure <30 mmHg (critical pressure) is considered as diagnostic for compartment syndrome in tibial fractures.

Younger patients are at higher risk for compartment syndrome because of higher rates of high-energy injuries and bigger muscles which give little space for expansion. Elderly patients often sustain low-energy fractures and the presence of hypertension could be a protective factor.

Blood tests – WBC, urea, creatinine, potassium, creatinine kinase.

Management

Release all bandages, splints or casts all the way down to skin and keep the limb at heart level. Impending or established

compartment syndrome is treated surgically. It is essential to release all compartments completely (including full length skin incisions), avoiding iatrogenic neurovascular injuries, and thorough debridement of any necrotic tissues is necessary. Appropriate antibiotic cover is instituted to reduce the risk of post-surgical infection. Primary skin closure is avoided. Associated fractures should be stabilized to reduce further tissue swelling. Wounds are debrided again at 48–72 hours and, if clean, closure of the wounds should be considered. Options of closure include the shoelace technique, closure of one wound and skin graft of the other. Plastic surgical procedures may be required when skin loss or difficulties in skin cover are anticipated.

Reperfusion injury should be anticipated. The patient's renal function (including serum potassium and urine output) should be closely monitored and hydration should be maintained. If required, diuretics to 'flush out' the toxins can be instituted. Missed compartment syndromes lead to gangrene or, if partial, Volkmann's contractures. It is one of the commonest reasons for lawsuits in orthopaedics.

Leg compartment syndrome

The leg is the commonest site of compartment syndrome. Critical pressure or delta pressure is 30 mmHg (some consider 40 mmHg). There are four compartments in the leg: anterior (tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius), lateral (peroneus longus and brevis), posterior superficial (gastrosoleus, plantaris) and posterior deep (tibialis posterior, flexor digitorum longus, flexor hallucis longus).

Surgical technique

Double incision – posteromedial (superficial and deep posterior compartments) and anterolateral (anterior compartment and via anterolateral intermuscular septum, the lateral compartment, avoiding injury to the superficial peroneal nerve) incisions, avoiding the perforators that cross the intermuscular septae. It is prudent to use Doppler assessment to identify the locations of the perforators prior to compartment release.

Other compartments do not have a specific critical pressure as a cut-off for compartment syndrome.

Thigh compartment syndrome

There are three compartments: anterior (quadriceps), posterior (hamstrings) and medial (adductors).

Surgical technique

Double incision – lateral (anterior and posterior compartments via the lateral intermuscular septum) and medial (adductor compartment).

Gluteal compartment syndrome

The gluteal compartment is enveloped in a tight fascia, which is continuous with fascia lata. This fascia splits to form three

separate compartments: the gluteus maximus, the gluteus medius and minimus, and tensor fasciae latae.

Surgical technique

An extended Kocher–Langenbeck approach is used to release all three compartments, avoiding injury to the sciatic nerve and the superior and inferior gluteal neurovascular bundles.

Forearm compartment syndrome

There are four compartments: mobile wad of Henry (brachioradialis, extensor carpi radialis longus and brevis), superficial volar (pronator teres, flexor carpi radialis and ulnaris, palmaris longus, flexor digitorum superficialis), deep volar (flexor digitorum profundus, flexor pollicis longus and pronator quadratus) and extensor compartment (all the extensors except the mobile wad of Henry).

Surgical technique

Extended Henry's approach (mobile wad of Henry, superficial and deep flexor compartments and carpal tunnel decompression) and posterior approach (deep posterior muscles).

Arm compartment syndrome

There are two compartments: flexor (biceps, brachialis, coracobrachialis) and extensor (triceps).

Surgical technique

Medial incision (flexor); posterior incision (extensor).

Hand compartment syndrome

There are 10 compartments: dorsal interossei (four compartments), palmar interossei (three compartments), adductor pollicis compartment, thenar compartment and hypothenar compartments.

Surgical technique

Two dorsal incisions (over second and fourth metacarpals) and carpal tunnel release.

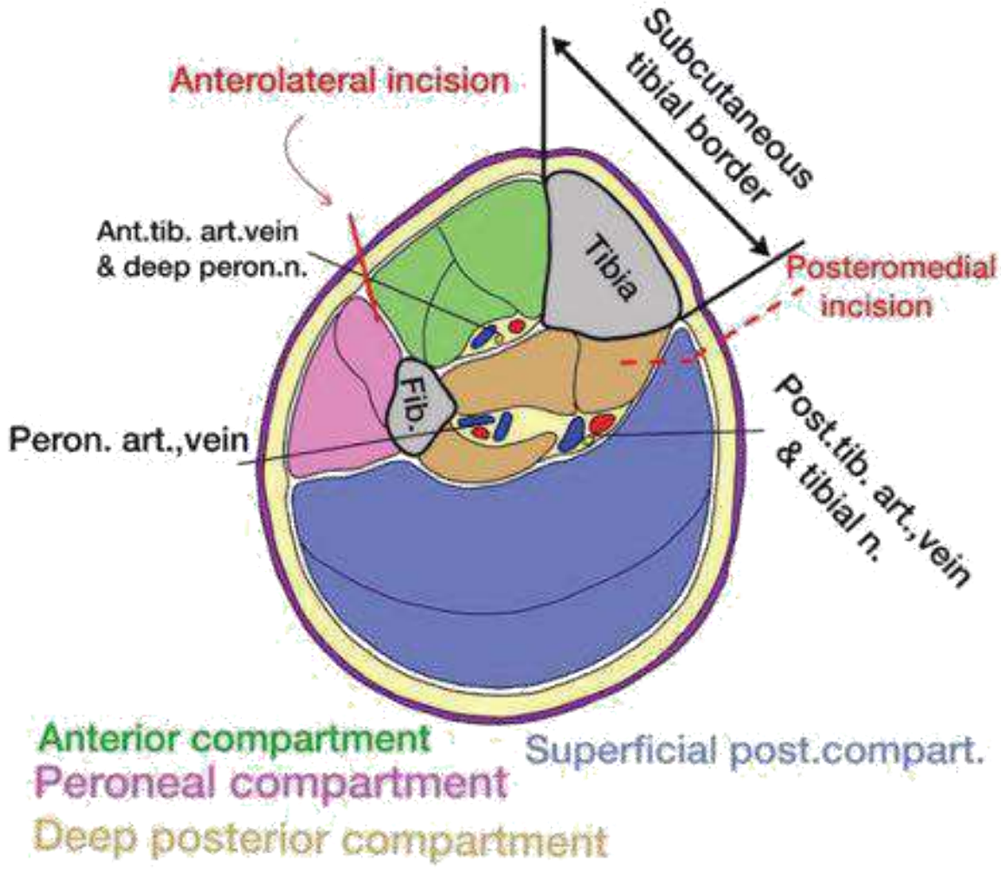
Foot compartment syndrome

The number of compartments in the foot is controversial. However, the compartments identified include: interossei (four compartments), medial (abductor hallucis and flexor hallucis brevis), lateral (abductor digiti minimi, flexor digiti minimi), superficial (flexor digitorum brevis), calcaneal (quadratus plantae) and abductor hallucis.

Surgical technique

Two dorsal incisions (over second and fourth metatarsals) and a medial incision allow release of all the foot compartments.

- Document compartments decompressed, debride necrotic muscle, 2nd look within 48 hours and early plastic surgery involvement
- Late presentation (>12 hours) has higher complication risk. 2 consultant decision to operate and non-operative management is an option
- **Two incisions Fasciotomy Technique** (15-20 cm wounds)
  - **Anterolateral incision**
    - 2 cm anterior to fibular shaft
    - Find and protect superficial peroneal nerve
    - Find anterior intermuscular septum
    - Release anterior compartment longitudinally ½ way between intermuscular septum and tibia
    - Release lateral compartment in line with fibula (danger to superficial peroneal nerve)
  - **Posteromedial incision**
    - 2 cm posterior to posteromedial edge of tibia
    - Incise posterior superficial compartment
    - Release posterior deep compartment
    - Cannot be done without proper elevation of soleus
    - Examine epimysium and incise when tight
    - Prevent retraction of skin edges with tensioned vessel loops weaved through staples (shoelace technique)

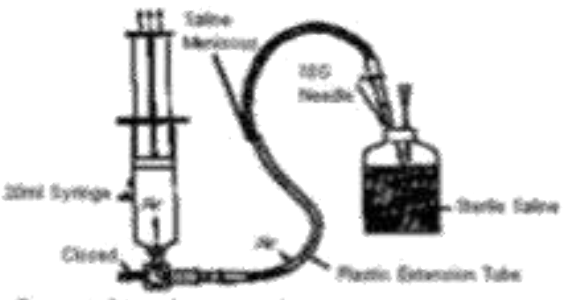


- **Post-operative**
  - Delayed primary closure or VAC or skin graft at 3-7 days

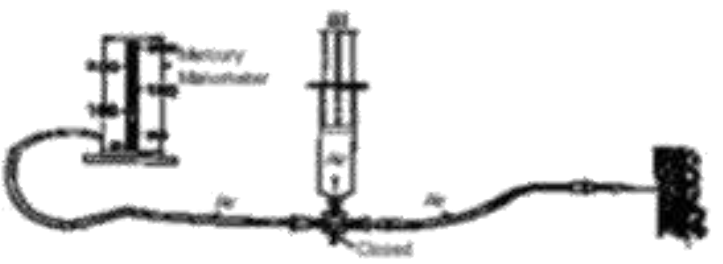
Neglected compartment syndrome

- Observe and do tenolysis later
- Decompression can kill patient from rhabdomyolysis

Whitesides Technique



Three-way Stopcock open to syringe and one extension tube.

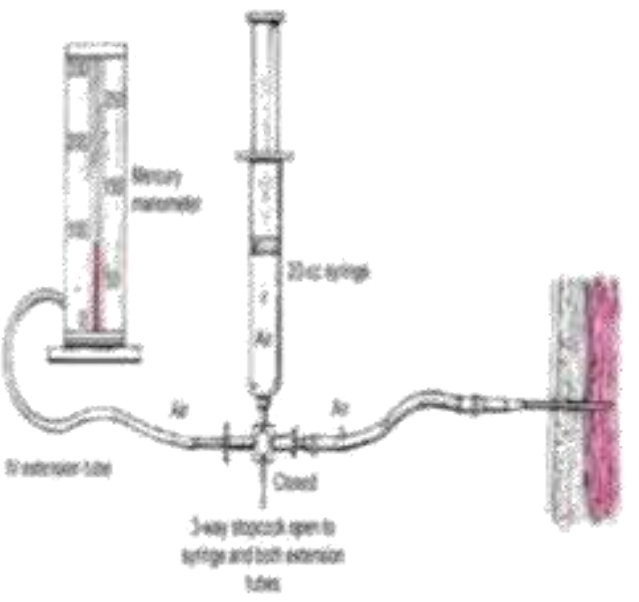


Three way stopcock open to syringe and both extension tubes.

Measurement of ICP

Needle Infusion Technique-Historical

- Needle inserted into muscle, tube with air/saline interval kept at this height, manometer indicates pressure
- Air injected by syringe via 3-way stopcock
- When the pressure of the injected air exceeds the compartment pressure pressure, the saline interval moves in the tube
- AT this point, ***the second person reads the pressure from the manometer***

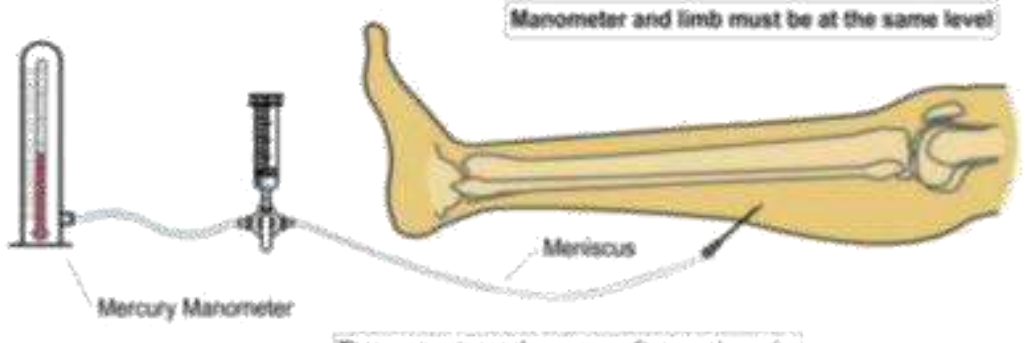


3-way stopcock open to syringe and both extension tubes

MEASUREMENT TECHNIQUES

1. Needle manometer
- Landerer
- 18 gauge needle, 20ml syringe, column of saline and air, mercury manometer

Traditional Compartment Measurement Set-Up



Three-way stopcock - open, saline meniscus in proximal tube, insert needle, increase pressure in syringe until meniscus moves, read pressure

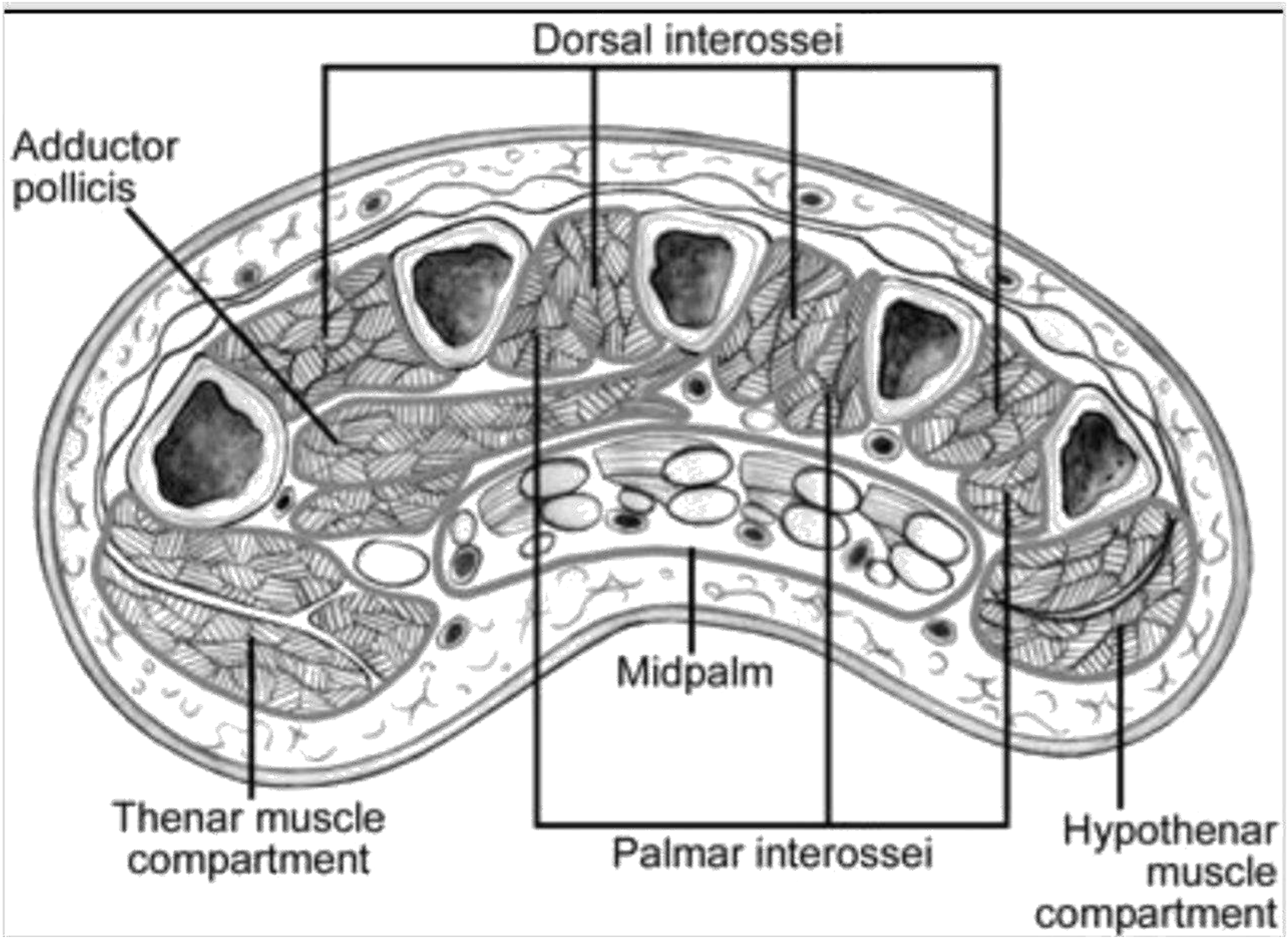
Compartment syndrome

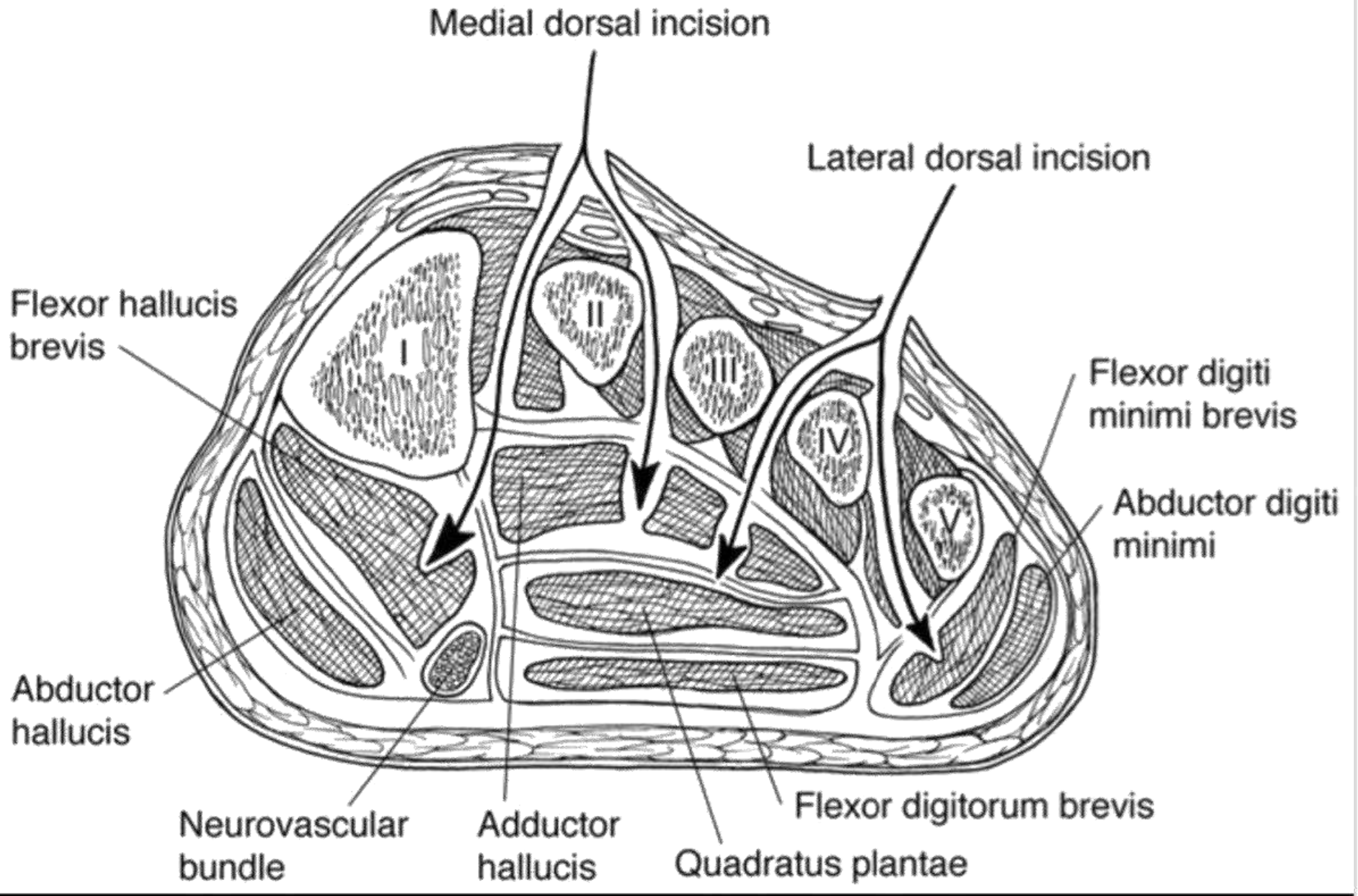
STRYKER METHOD

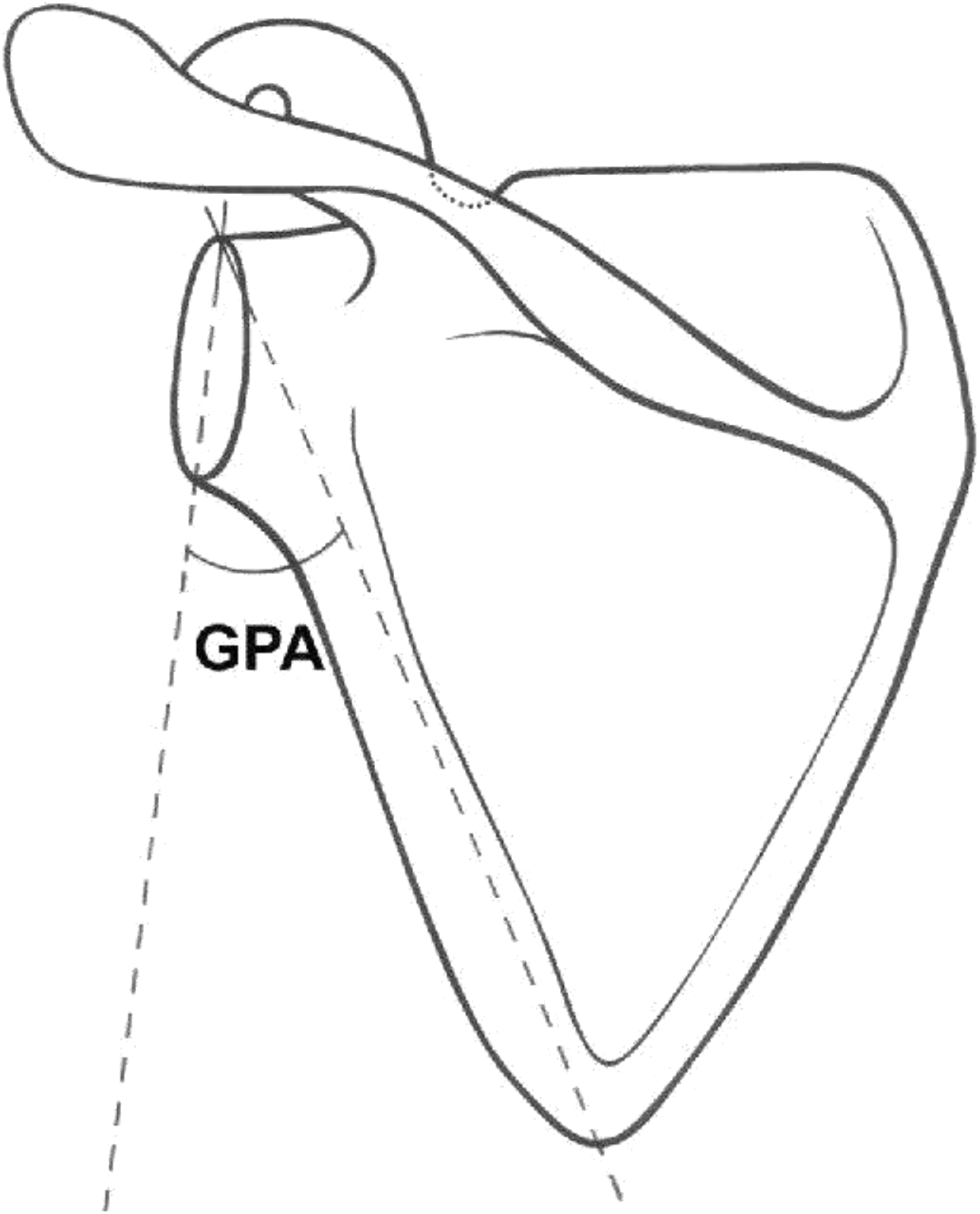
- (1) Fill the Stryker instrument with normal saline
- (2) zero the Stryker instrument
- (3) insert the needle into the area of measurement
- (4) inject 0.3 cc normal saline
- (5) read the pressure measurement

IV PUMP METHOD

- (1) Set the IV pump to manometry mode
- (2) zero the IV pump
- (3) insert the needle into the tissue being measured
- (4) infuse 0.3 cc normal saline at a slow infusion rate
- (5) read the pressure measurement.


Dorsal interossei
Adductor pollicis
Midpalm
Thenar muscle compartment
Palmar interossei
Hypothenar muscle compartment


Medial dorsal incision
Lateral dorsal incision
Flexor hallucis brevis
Flexor digiti minimi brevis
Abductor digiti minimi
Abductor hallucis
Neurovascular bundle
Adductor hallucis
Quadratus plantae
Flexor digitorum brevis
I
II
III
IV
V



1 Definition of the GPA. The GPA is defined as an angle forme

- LaFontaine predictors of instability
  - patients with three or more factors have high chance of loss of reduction
    - dorsal angulation > 20°
    - dorsal comminution > 50%, palmar comminution, intraarticular comminution
    - initial displacement > 1cm
    - initial radial shortening > 5mm
    - associated ulnar fracture
    - severe osteoporosis
  - radial shortening is the most predictive of instability, followed by dorsal comminution

Summary Box 17.1 Technical objective checklist

Concerning screws in the distal fragments (articular segment):

1. Each screw should pass through a plate.

2. Each screw should engage a fragment on the opposite side that is also fixed by a plate.

3. An adequate number of screws should be placed in the distal fragments.

4. Each screw should be as long as possible.

5. Each screw should engage as many articular fragments as possible.

6. The screws should lock together by interdigitation, thereby creating a fixed-angle structure and linking the columns together.

Concerning the plates used for fixation:

7. Plates should be applied such that compression is achieved at the supracondylar level for both columns.

8. Plates used must be strong enough and stiff enough to resist breaking or bending before union occurs at the supracondylar level.

Driscoll principles :

anatomic reconstruction of the articular surface, stable fixation of the fracture fragments to allow early and full ROM.

Failure, when it occurs, typically occurs at the supracondylar level through loss of fixation in the distal fragments.

To prevent such failure and maximize the potential for union and full elbow mobility after a severely fractured distal humerus, 2 principles must be satisfied:

(1) fixation in the distal fragment must be maximized

(2) all fixation in distal fragments should contribute to stability between the distal fragments and the shaft.

There are 8 technical objectives by which these principles are met: (Driscoll Principles)

(1) every screw in the distal fragments should pass through a plate;

(2) engage a fragment on the opposite side that is also fixed to a plate;

(3) as many screws as possible should be placed in the distal fragments;

(4) each screw should be as long as possible;

(5) each screw should engage as many articular fragments as possible;

(6) the screws in the distal fragments should lock together by interdigitation, creating a fixed-angle structure;

(7) plates should be applied such that compression is achieved at the

supracondylar level for both columns;

(8) the plates must be strong enough and stiff enough to resist breaking or bending before union occurs at the supracondylar level.

These can be achieved with parallel plating.

Severe metaphyseal comminution and bone loss are managed by supracondylar shortening.

From the biomechanical data in the literature, 3 conclusions can be reached concerning fixation of distal humeral fractures.

First, the concept that plates need to be placed perpendicular (90 degrees /90 degrees orientation) is unsubstantiated and incorrect.

Second, parallel placement of 2 plates in the sagittal plane is as strong or stronger than the 90 degrees /90 degrees orientation.

Finally, linking the plates together through the bone, thereby creating the architectural equivalent of an arch, offers the greatest biomechanical stability for comminuted distal humeral fractures.

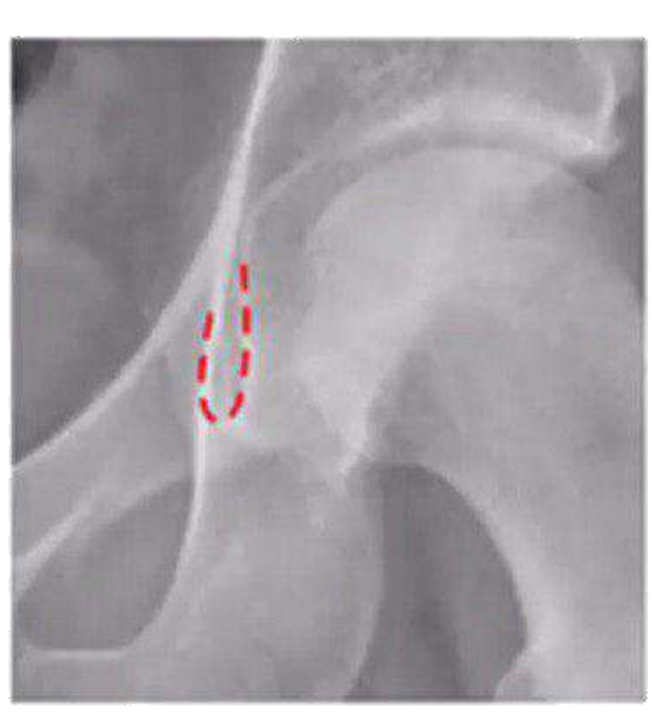
This can be done by interdigitating and locking the screws together as they pass through the distal fragments from the medial and lateral plates placed in the sagittal plane.

- **ORIF** for type II with block 2 mm plates cause minimal re

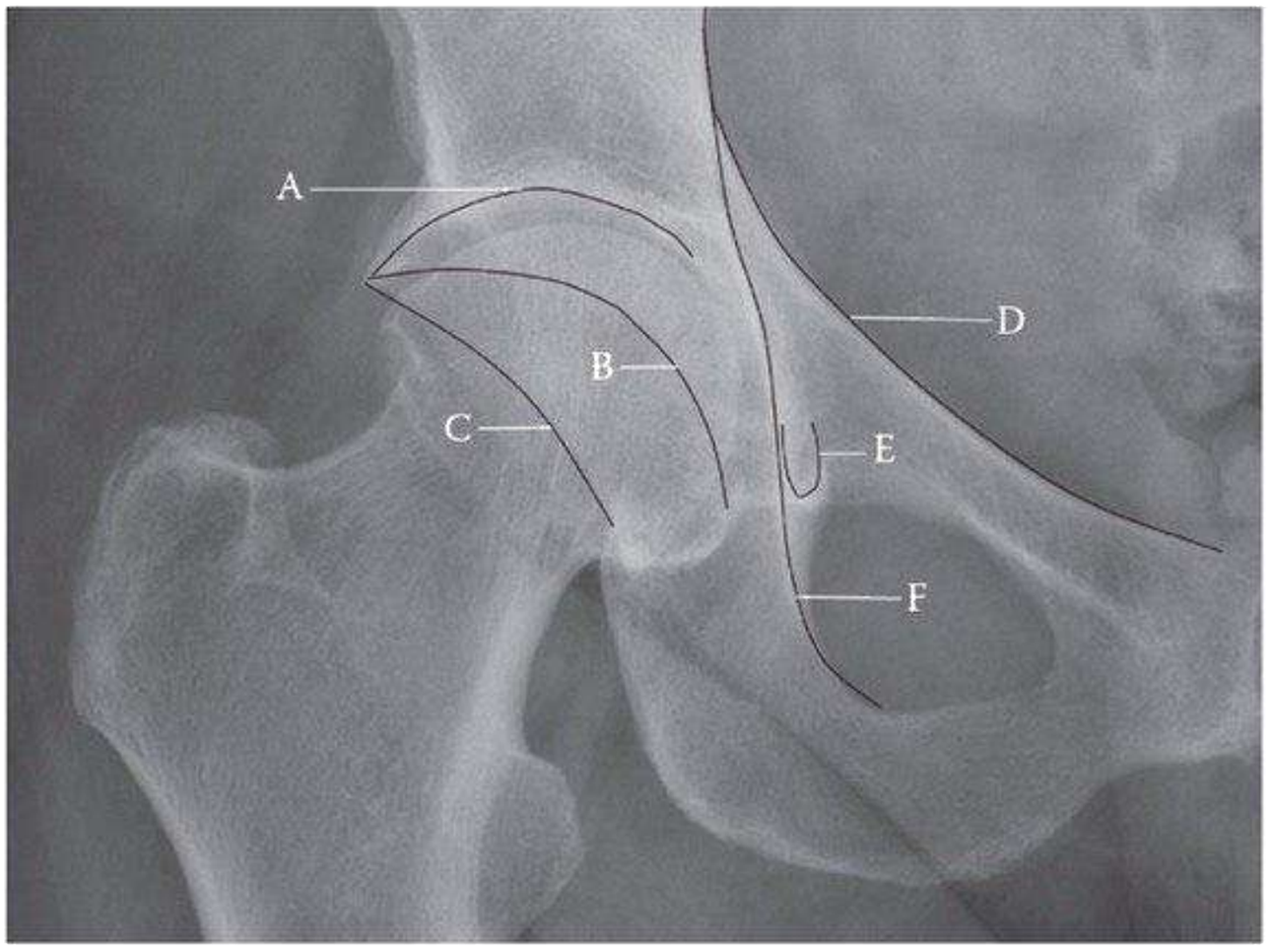
➢ **Safe Zone for implants:**

- Posterolateral portion of cartilage
- Appears yellow and thinner, non-articulating
- 90° arc between radial styloid and Lister's tubercle

Teardrop



medial side of acetabulum
(quadrilateral plate)
anterior wall of
acetabulum
acetabular
fossa
posterior wall
of acetabulum



3. Can you name the labelled areas of the plain AP radiograph of the pelvis?

A – Acetabular dome/roof
B – Anterior wall
C – Posterior wall
D – Iliopectineal line (represents the anterior column)
E – Tear drop
F – Ilioischial line (represents the posterior column)

4. What is the 'tear drop'?

The pelvic tear drop results from the end-on projection of a bony ridge running along the floor of the acetabulum.

It is formed laterally by the confluence of subchondral bone at the floor of the acetabular fossa (also known as the cotyloid fossa) and medially by the anterior flat portion of quadrilateral plate.

It represents the true floor of the acetabulum and is frequently used for preoperative planning in hip arthroplasty.

5. What anatomical structures make up the anterior and posterior columns of the acetabulum?

Anterior column:

- Anterior iliac wing
- Anterior wall and dome
- Iliopectineal eminence
- Superior pubic ramus

Posterior column:

- Quadrilateral plate
- Posterior wall and dome
- Ischial tuberosity
- Greater/lesser sciatic notches

I. Posterior & Anterior rims lines only ?

-----> **Posterior wall**

-----> Anterior wall

II .Iliopectineal & Ilioischial lines; Either Disrupted ?

Iliopectineal # -----> Anterior Column

Ilioischial # -----> Posterior Column or PC with PW

III . 4 lines Disrupted ; Post ,Ant rims , Iliopectineal & Ilioischial lines ?

Intact obturator ring & iliac wing -----> **Transverse / Tr with PW**

IV. Obturator Ring /Ischiopubic ramus and iliac wing?

Obturator ring / Ischiopubic ramus & intact iliac wing -----> **T- Shaped**

Obturator ring / Ischiopubic ramus & iliac wing -----> **Both Column**

What should I observe and test once the joint fluid has been aspirated?

You should describe the general characteristics of the gross appearance. The aspirated fluid is inspected for viscosity by the string test, looking for clarity, color, and the presence of blood or fat droplets. The aspirated fluid is then placed in different containers and sent for laboratory tests, depending on the differential diagnosis. In general, testing often includes a cell count with differential, an immunologic test for arthritis, and analysis of glucose and protein. A smear for Gram stain and a bacterial culture are ordered when infection is suspected.

Table 2

Synovial Fluid Characteristics and Arthritis-Related Laboratory Findings

	Finding				
Characteristic	Normal (No Arthritis)	Noninflammatory Arthritis*	Inflammatory Arthritis†	Septic Arthritis (Native Joint)‡	Prosthetic Hip or Knee Infection§
Appearance	Clear	Clear	Opaque or translucent	Opaque Yellow or green	Clear or opaque
WBC/mm^3	< 1,000	< 1,000	5,000–75,000	> 50,000	> 1,100-3,000
Polymorphonuclear cells	< 25%	< 25%	> 50%	> 75%	> 64%-80%
Culture	Negative	Negative	Negative	Positive	Positive

WBC = white blood cell count.
*Associated conditions: Degenerative joint disease, trauma, pigmented villonodular synovitis, neuropathy, systemic lupus erythematosus, acute rheumatic fever.
†Associated conditions: Rheumatoid arthritis, crystal-induced arthritis, seronegative arthropathy, systemic lupus erythematosus, acute rheumatic fever.
‡An immunocompromised patient may not have an elevated synovial WBC. A normal or noninflammatory WBC does not preclude active septic arthritis.
§Data are unavailable for other types of prosthetic joints. Underlying inflammatory arthritis may cause false-positive results.

IL 6 :
increase in 1st hr of infection
peak : 12 hrs
normalize in 3 days

CRP :
increase after 6 hrs of infection
Peak : 3 days
normalize in 3 weeks

ESR :
increase after 24 hrs of infection
Peak : 5-7 days
Normalize in 3 months

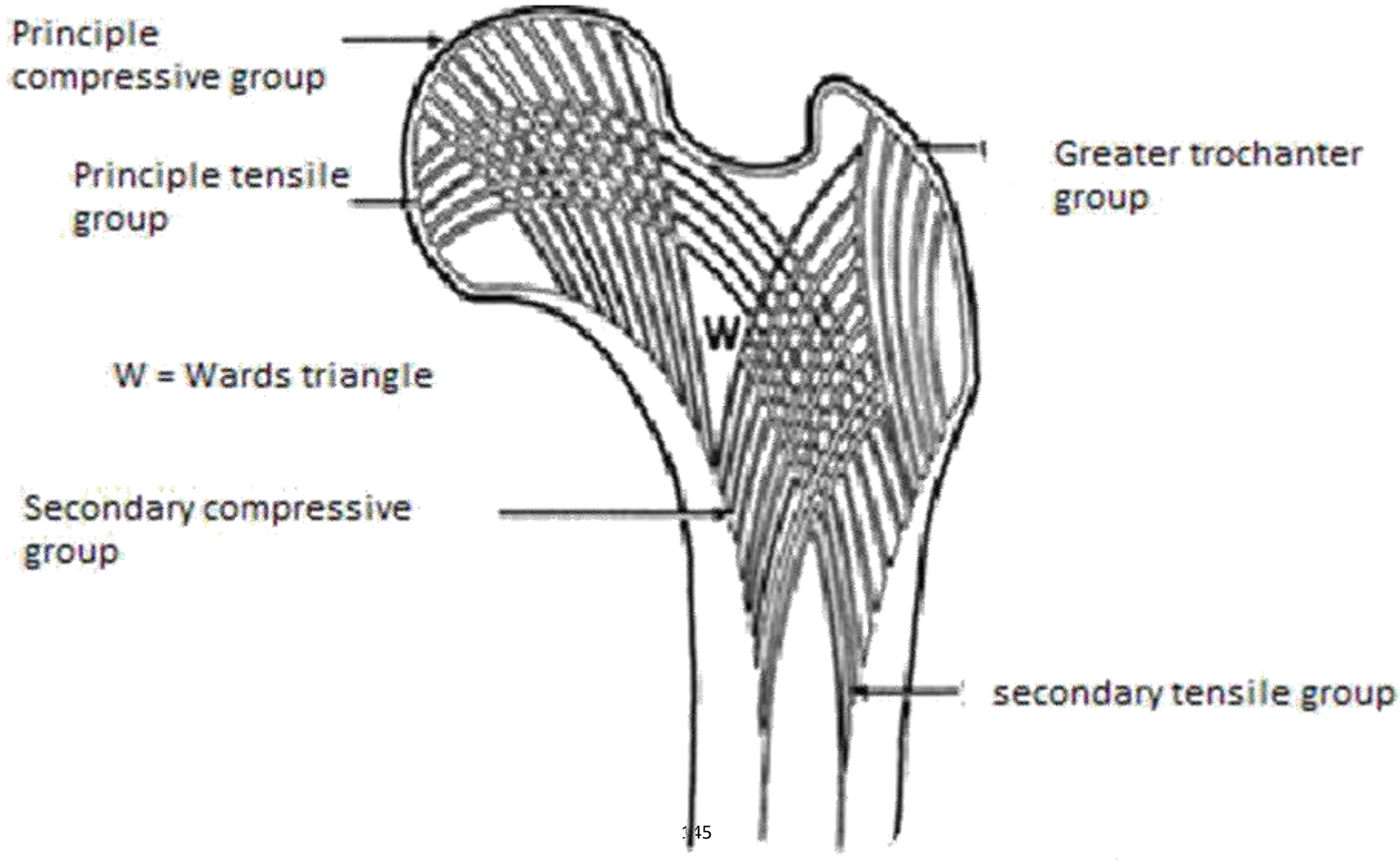
23 6:27 PM

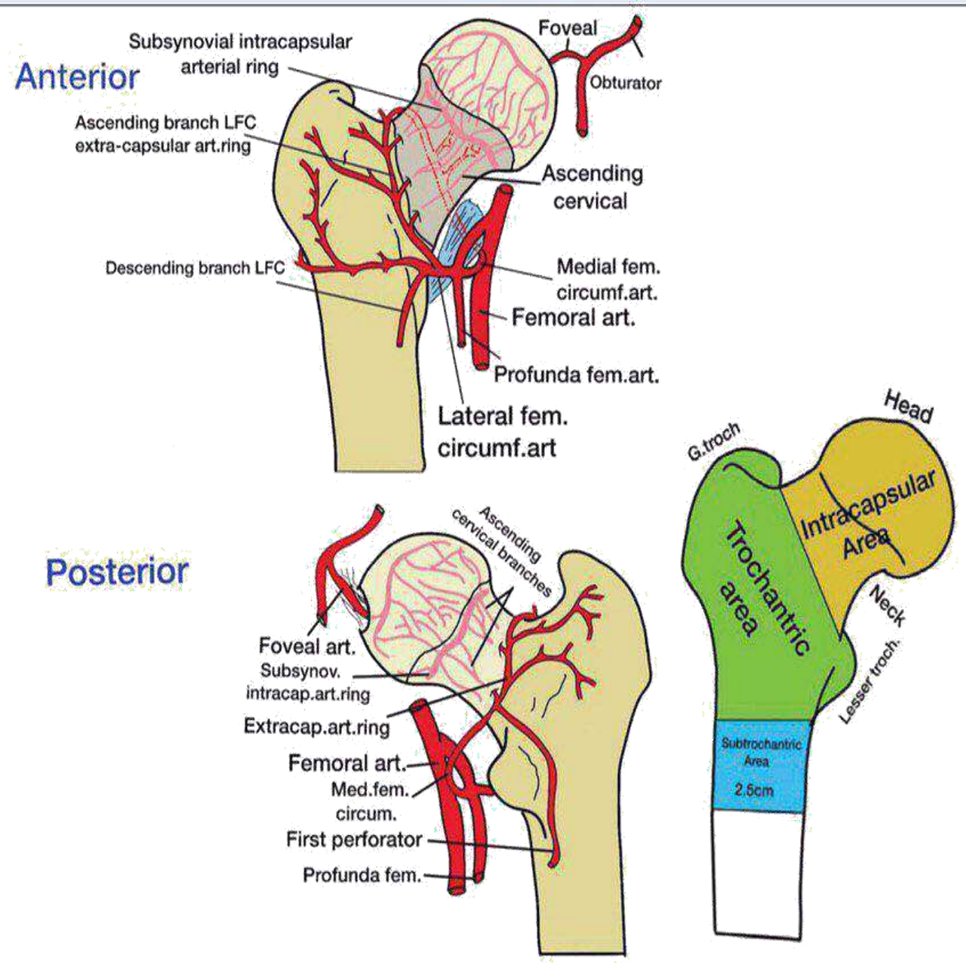
Laboratory testing:

- **CRP:**

  An acute-phase protein synthesized by the liver in response to inflammation

  - Rises within 6h
  - Returns to normal within 6-10 days
  - More sensitive (WBC, ESR) in assessing the ttt effectiveness predicting recovery
  - 2ry rise suggests relapse


Principle compressive group
Principle tensile group
W = Wards triangle
Secondary compressive group
W
Greater trochanter group
secondary tensile group



Blood supply of femoral head

- Medial epiphyseal artery from obturator artery via ligamentum teres
- Medial & lateral circumflex femoral arteries from profunda femoris, medial is the main one ,emerges from under the quadratus femoris and goes posterior to femoral neck
- Form extra-capsular arterial ring around base of trochanters giving off ascending cervical retinacular arteries
- Medial at risk when doing iliopsoas tenotomy, courses between iliopsoas and pectineus
- Intramedullary supply from shaft and metaphysis
- < 4 years All 3 arteries
- 4 – 10

65E. Describe the blood supply to the femoral head.

The blood supply to the femoral head is as follows:

Extracapsular arterial ring at the base of the femoral neck,

- formed posteriorly by large branch of medial femoral circumflex artery
- formed anteriorly by smaller branches of lateral femoral circumflex artery
- superior and inferior gluteal arteries have minor contributions

Ascending cervical branches:

- these give rise to retinacular arteries
  gives rise to subsynovial intra articular ring

Artery of ligamentum teres:

- derived from obturator or medial circumflex femoral artery
- forms the medial epiphyseal vessels
- only small amount of the femoral head is nourished this artery

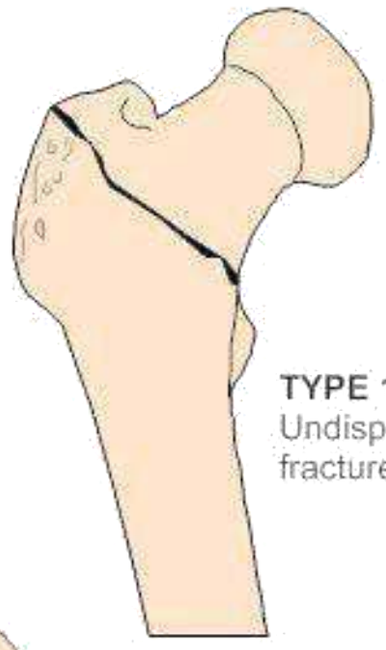
Epiphyseal blood supply:

- arises primarily from lateral epiphyseal vessels that enter head posterosuperiorly
- vessels from medial epiphyseal artery entering through ligamentum teres

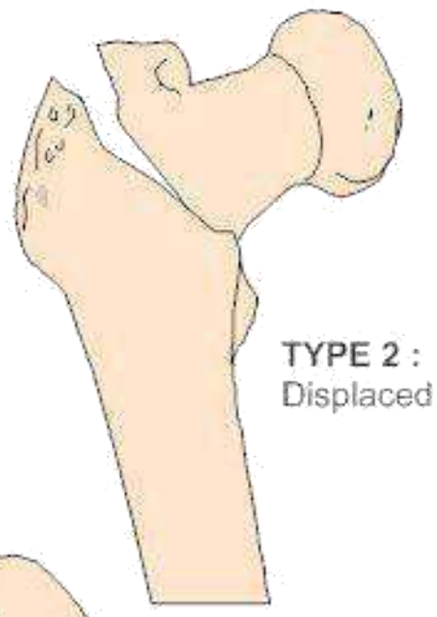
Metaphyseal blood supply:

- arises from extracapsular arterial ring
- arise from branches of ascending cervical arteries and subsynovial intra-articular ring

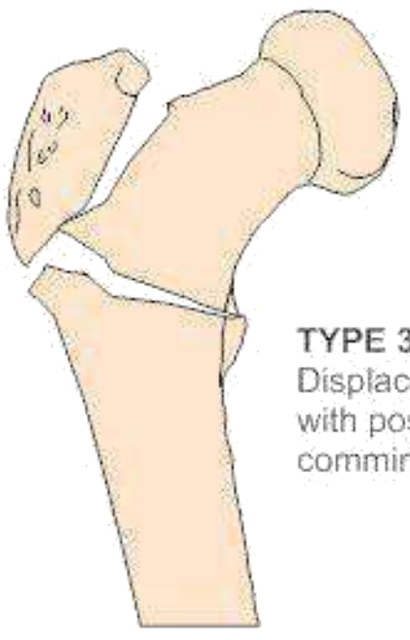
Evan's Classification of Intertrochanteric Fractures



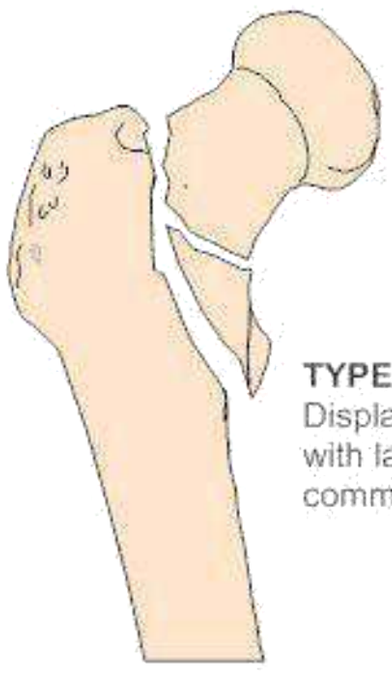
TYPE 1:
Undisplaced 2 part fracture.



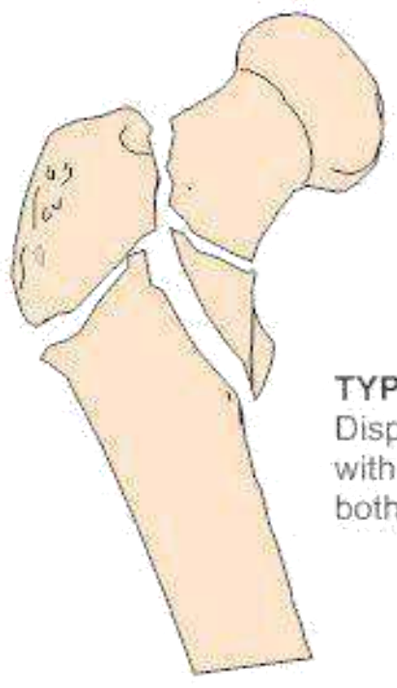
TYPE 2 :
Displaced 2 part fracture.



TYPE 3 :
Displaced 3 part fracture with posterolateral comminution.



TYPE 4 :
Displaced 3 part fracture with large posteromedial comminuted fragment.



TYPE 5 :
Displaced 4 part fracture, with comminution involving both trochanters.

Stress Examination

C-Arm - Ipsilateral

Obturator Oblique

Flexion

Adduction

Internal Rotation

Posterior Stress

Like Comment Share

- C-arm ipsilateral
- Obturator oblique view
- Flexion
- Adduction
- Internal rotation
- Posterior stress

Moed JOT 2009

CLOSED REDUCTION

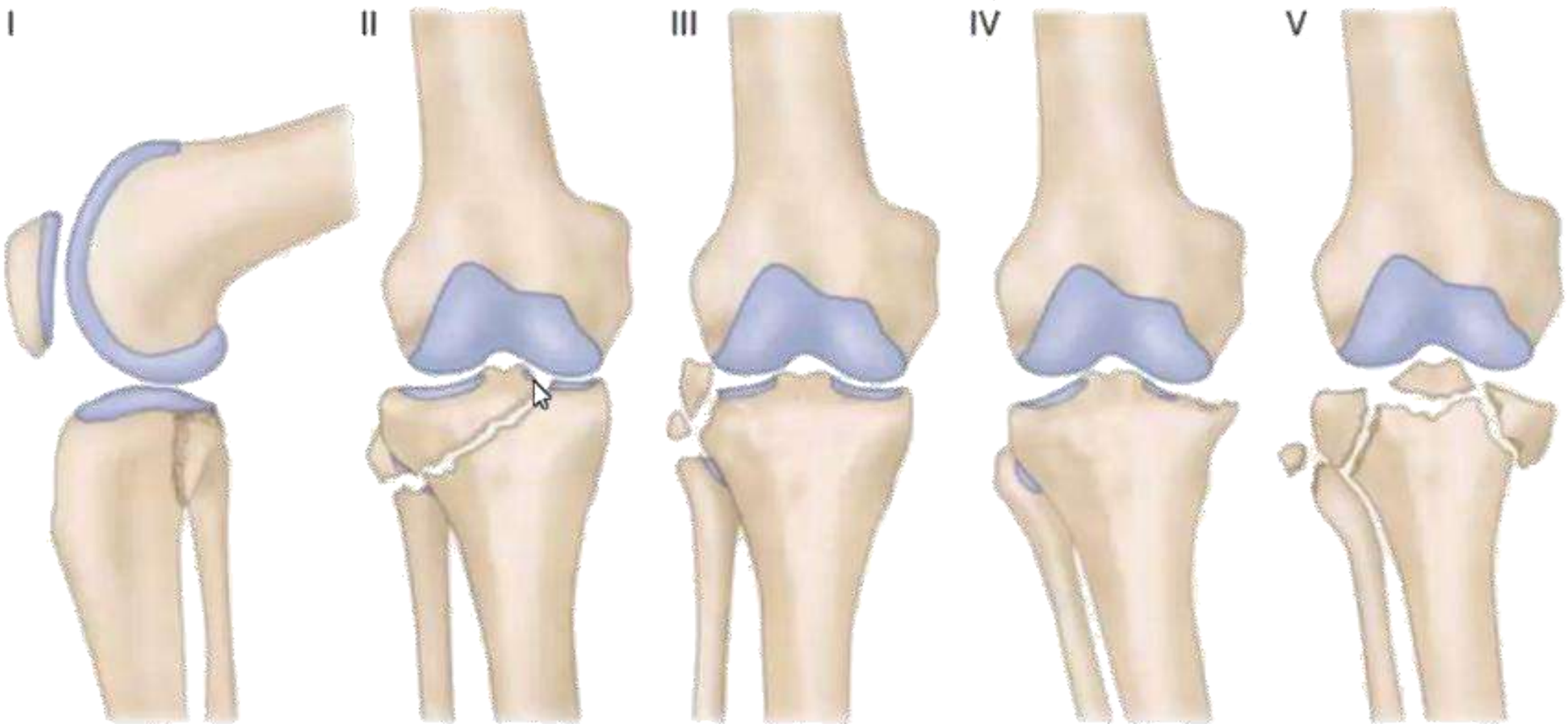
WHITMAN TECHNIQUE

- The fractured extremity is tied to footplate in an externally rotated position
- With the extremity externally rotated, it is abducted approximately 20*
- Traction is applied
- The extremity is internally rotated until the patella is internally rotated 20 - 30*.

LEADBETTER TECHNIQUE

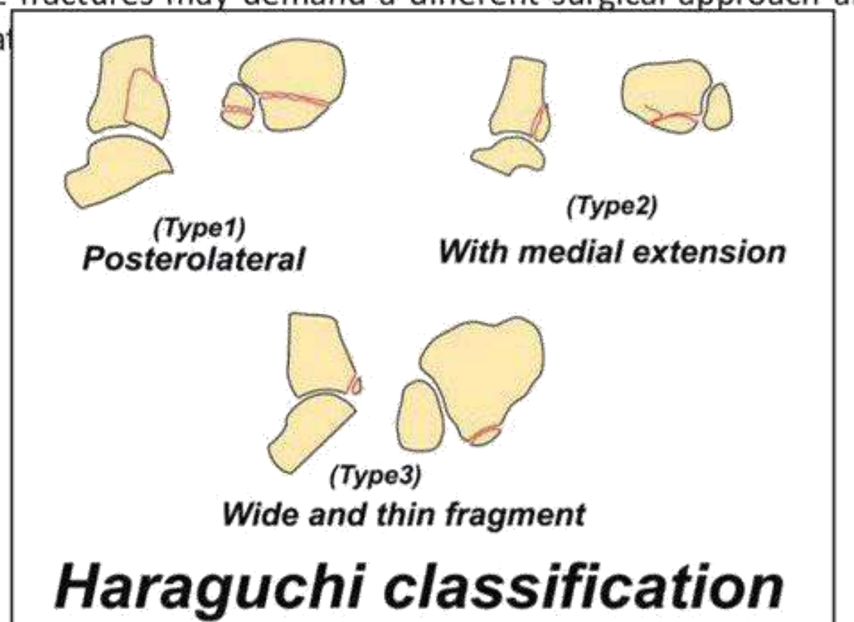
- Hip is flexed to 90*
- Traction along long axis of femur
- Thigh is internally rotated & abducted
- Reduction is evaluated by “heel palm” sign

Hohl & Moore

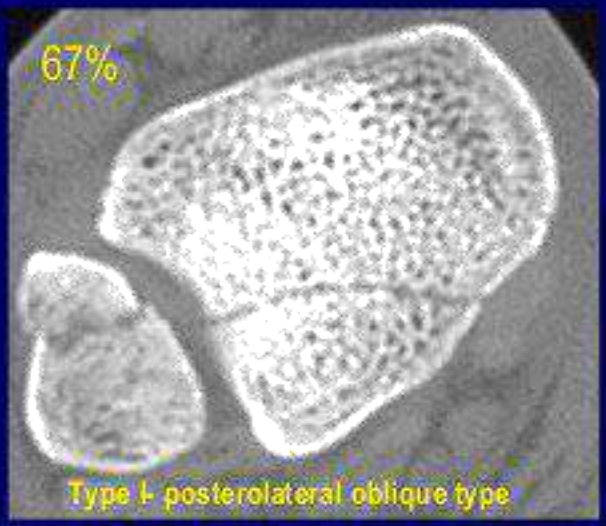

I
II
III
IV
V
Split
Entire condyle
Rim avulsion
Rim compression
Four part

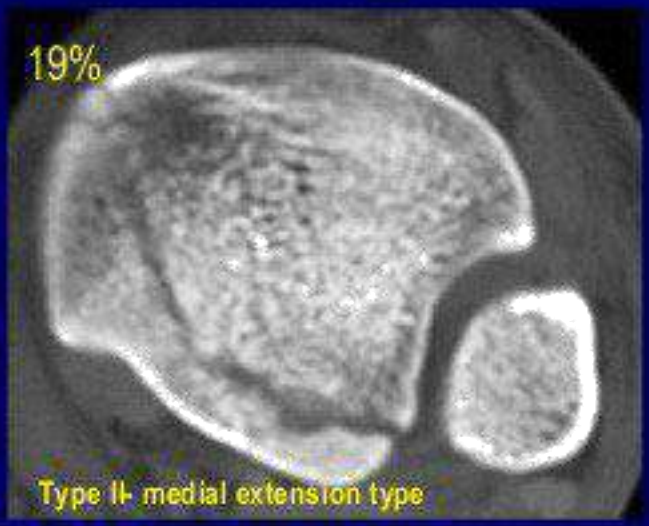
Haraguchi classification

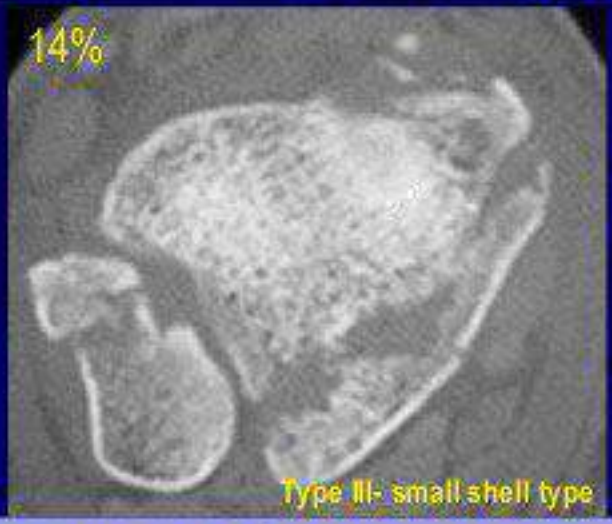
- Three types of fracture in a CT study
  - Type 1 The most common, is a single posterolateral fragment
  - Type 2 Extension to posteromedial side of distal tibia
    There may be more than one fragment.
  - Type 3 Thin shell of bone
- The classification is useful, as type 2 fractures may demand a different surgical approach and type 3 fractures may be too small or thin to allow fixat



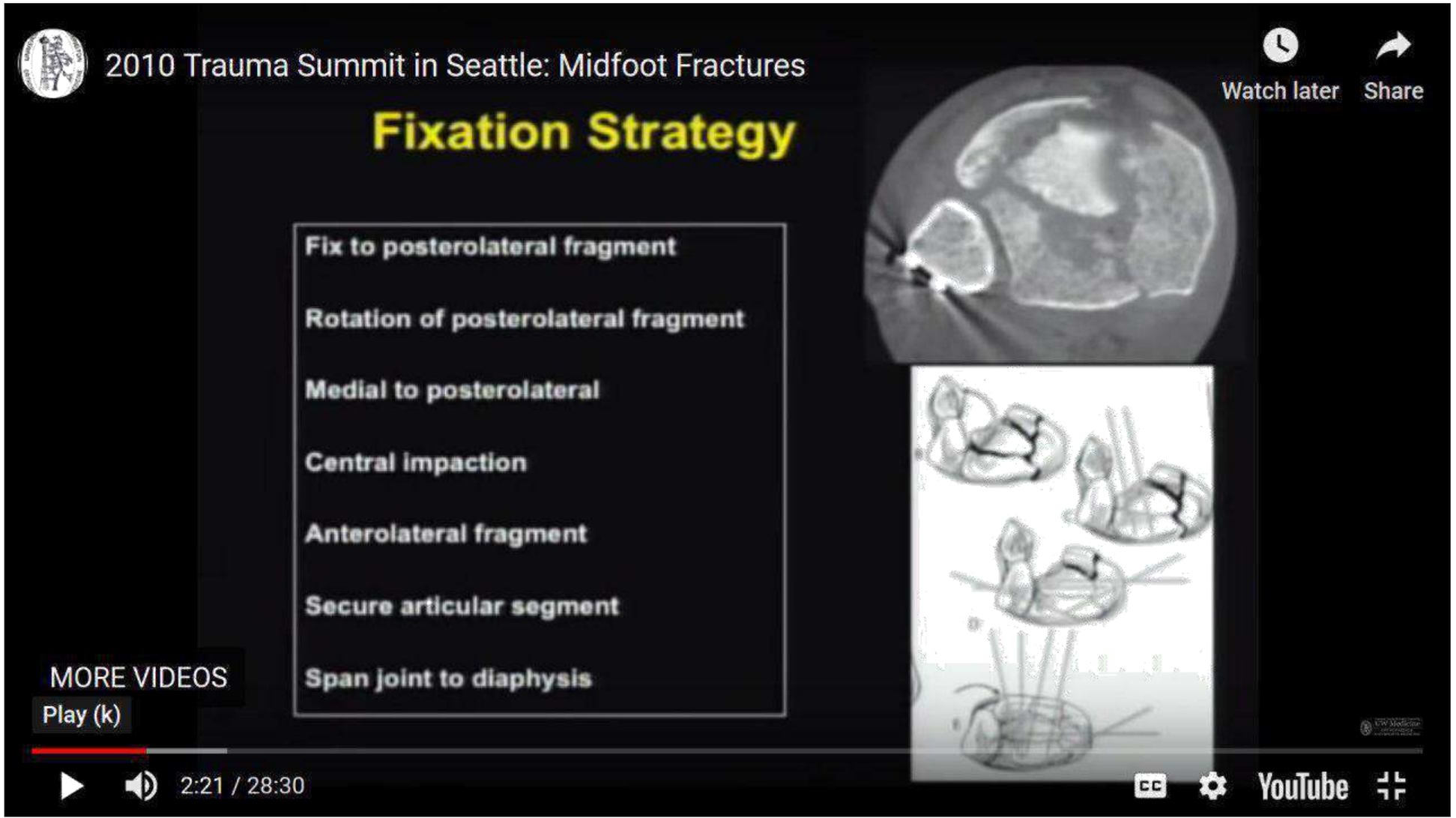
Posterior Malleolus Fracture







3 common PM fracture patterns


2010 Trauma Summit in Seattle: Midfoot Fractures
Watch later
Share
Fixation Strategy
Fix to posterolateral fragment
Rotation of posterolateral fragment
Medial to posterolateral
Central impaction
Anterolateral fragment
Secure articular segment
Span joint to diaphysis
MORE VIDEOS
Play (k)
2:21 / 28:30
YouTube

4. What is the classification of a Lisfranc fracture?
Answer: When assessing Lisfranc injuries the most commonly used classification is that of Quenu and Kuss (1909) as modified by Hardcastle (1979). Myerson (1986) revised the classification:

Total incongruity (type A)
A - In any plane or direction.
Partial incongruity (type B)
B1 - Medial (only the first ray is involved).
B2 - Lateral (one or more of the lateral rays) – commonest group.
Divergent displacement (type C)
C1 - Partial incongruence.
C2 - Total incongruence.

B. Viva : Long & Short

Section 3

Trauma

Chapter

8

Lower limb trauma

Mohammed Al-Maiyah and Ali S. Bajwa

Introduction

As far as the exam is concerned, it is very similar to a chess match. It ought to be treated with respect, but played in a clever fashion. As soon as you try to think of the exam as a wrestling match pitfalls for failing are plentiful. In the orals, just like chess, you have to preempt the move of your opponent, which in this situation is the examiner. It is a time-dependent chess match where every move must be undertaken in a specified time. Keep this analogy as you attempt different clinical scenarios. It is not only knowledge of the subject that is important, but to impart it in an appropriate fashion, which is inherently more important in the orals.

Treat each subject as a chess game that is going to last 5 minutes.

Structured oral examination question 1

A 35-year-old motorcyclist came off his bike yesterday; he has been resuscitated and has an isolated closed injury of the knee. (Figure 8.1.)

Minute 1

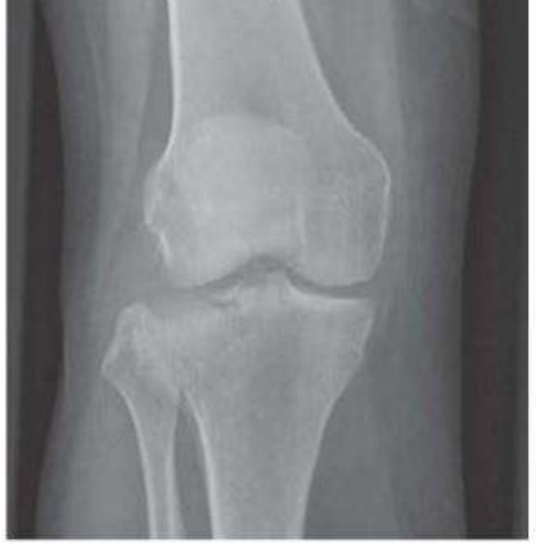
EXAMINER: What are your views?

Here the next 1 minute belongs to the candidate and you can take it whichever way you want to. However, there are essentials to be covered. In the first 30 seconds you are expected to comment on the following:

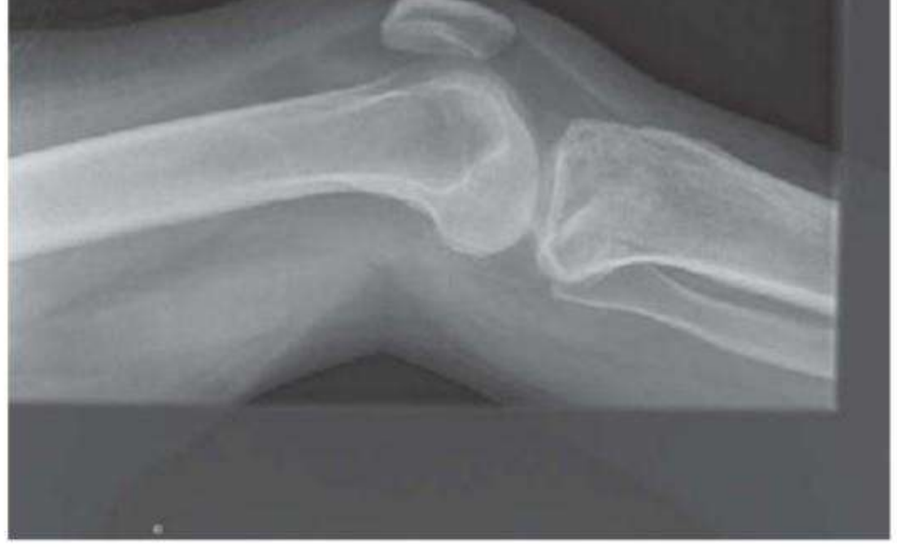
- Name of the patient.
- Site of radiograph.
- Fracture through the tibia with depression of the lateral tibial plateau and always ask for a lateral radiograph.

In the next 30 seconds the candidate is expected to comment on the exact nature of the injury, such as Schatzker III fracture with more than 10 mm depression of the articular surface, comminution, concern about the fracture going through the tibial spines and whether the medial side is involved. A candidate should end the 30 seconds by mentioning they would assess the whole patient for any co-existing injuries,

(a)



(b)



Figures 8.1a and 8.1b Anteroposterior (AP) and lateral radiographs of right knee demonstrating tibial plateau fracture.

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

the whole lower limb for other injuries and then arrange further imaging studies.

Minute 2

EXAMINER: What further imaging studies would you arrange?

CANDIDATE: A CT scan to evaluate the fracture pattern and plan surgery.

The examiner is likely to produce promptly two slices of the CT, in sagittal and coronal.

Note: Do not make a stupid remark 'do you want me to comment on this?'! But it is useful to correlate that it is the same patient's CT. Describe the findings and end up by offering to discuss the findings with the patient (Brownie points). As well as offering in the closing comments about management options. (Let the examiner ask you about the management options rather than carrying on.)

You should be at this stage by 90 seconds.

EXAMINER: What are the management options?

CANDIDATE: [Take the next 30 seconds to describe operative and non-operative options.] Non-operative management is a poor choice because of the patient's young age and amount of articular segment depression. Operative management would require a non-compromised soft tissue envelope and exclusion of lower limb neurovascular injury. Assessment of the injury pattern and of any significant knee ligament damage needs to be done prior to surgical intervention.

Minute 3

At the 2-minute mark you should have committed yourself to operative intervention. Before the examiner asks, offer your management option because it annoys them to keep asking again and again: what will you do? Stick with the principles.

The principles of management are to restore the articular surface, stabilize and hold the fracture in such a fashion as to allow early mobilization. The aim of the treatment is to have a mobile, pain-free and functional joint.

The options of surgical treatment include direct or indirect reduction, percutaneous or open fixation augmented with plate osteosynthesis or external fixation. Before being prompted, suggest your preferred option, which in the authors' opinion is indirect reduction using a cortical window in the proximal tibia, restoration of articular surface with a raft of screws augmented with a buttress plate. Suggest at this stage you will perform an assessment under X-ray control for a ligamentous stability and if needed an arthroscopic assessment.

Minute 4

The examiner can lead the viva along two aspects:

EXAMINER: What is a buttress plate?

CANDIDATE: A plate applied perpendicular to the force that it resists.

EXAMINER: What is the role of knee arthroscopy?

CANDIDATE: It is threefold. Firstly, to assess the reduction of articular surface. Secondly, to check that soft tissues are not trapped in the fracture such as lateral meniscus. Thirdly, to assess intra-articular ligament damage. Pressure pumps are not to be used to avoid iatrogenic compartment syndrome due to extravasation of fluid. I will use a bladder syringe through the arthroscopy cannula to wash out the haemarthrosis before viewing the joint. [This gives the examiner the impression that you have done the procedure before.]

EXAMINER: What surgical approach will you use?

CANDIDATE: Anterolateral approach with the skin incision being longitudinal and if needed muscle retraction with a reverse L-shaped incision inside.

The examiner can also talk about a posterolateral approach and a fibula osteotomy for posterolateral fractures. Whilst discussing these issues you should be at the 4-minute stage.

Minute 5

With 1 minute left and if the examiner is discussing rehabilitation and weightbearing status, you know that you are probably winning. Talk about range of motion and protected weightbearing. Assessment of weightbearing will be based on stability of the fracture, strength of the fixation, stiffness of the construct and the quality of the host bone plus reliability of the patient. Do not, repeat do not, use absolute numbers such as 20 kg for each patient on day 1. Tailor the management for each individual patient based on host quality of bone, fracture pattern and construct stability.

Warning: Be prepared to be shown a radiograph of metalwork failure with screws cut out into the articular surface. Stay calm. Assess the patient clinically, radiologically (including CT), rule out infection,

soft tissue problems, patient compliance and then proceed from the start, take out the metalwork, align the articular surface, stabilize the fracture and mobilize again. It is the same story all over.

Structured oral examination question 2

A 79-year-old woman fell in her garden sustaining this injury. She is generally quite independent, has a history of angina which is controlled and likes meeting her friends at the local social club every Wednesday.

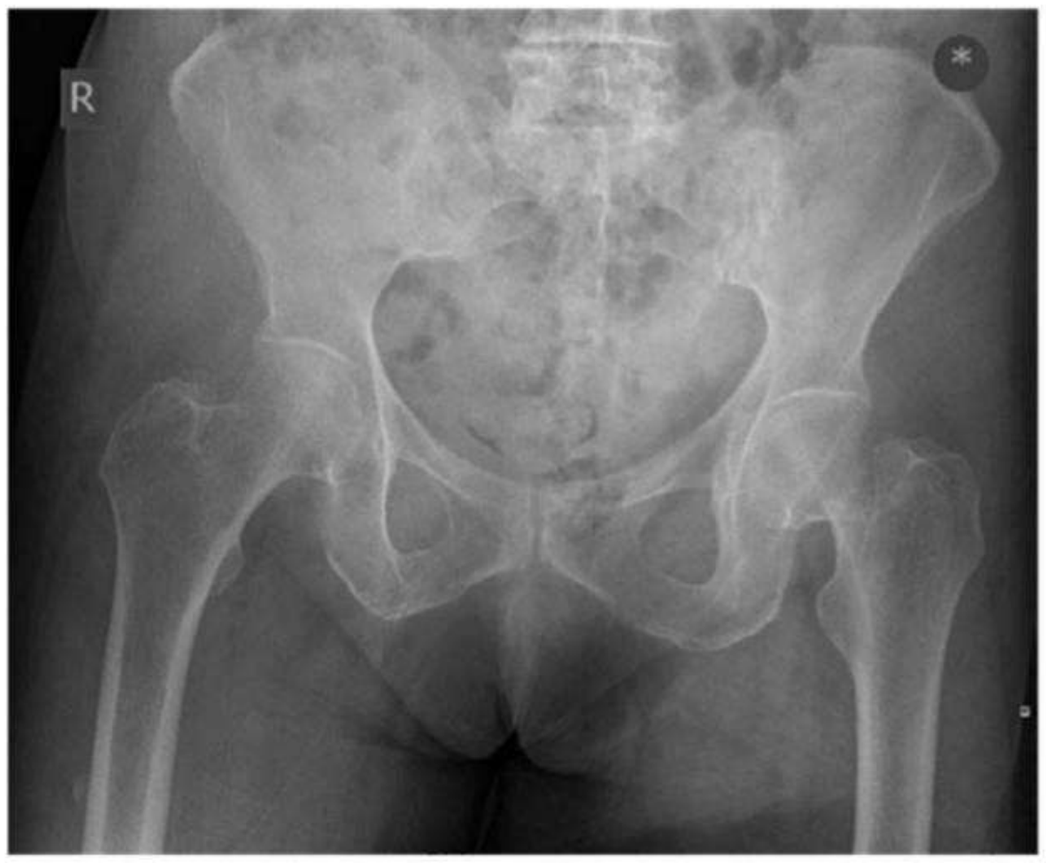


Figure 8.2 Anteroposterior (AP) pelvis radiograph demonstrating intracapsular fractured left neck of femur.

Minute 1

In the first 30 seconds you are expected to comment on the name of the patient, site of radiograph and the exact nature of the injury:

EXAMINER: Please comment on the radiograph. (Figure 8.2.)

CANDIDATE: She has got a left-sided intracapsular neck of femur fracture in the presence of early degenerative changes of the hip joint. [Always ask for the lateral radiograph.]

EXAMINER: How will you manage this patient?

CANDIDATE: I'd like to assess the whole patient. The degree of mobility prior to injury, comorbidities, any systemic illness, red flag signs for any pathological lesions, drug history, cause of fall and appropriate investigations. In addition, I would perform a full clinical examination of the patient including the left lower limb. [Be a safe surgeon but not hesitant.]

Minutes 2 and 3

EXAMINER: She has well-controlled angina and is otherwise independent.

CANDIDATE: In this patient group surgery would be my preferred option so as to avoid the known complications of non-operative management. These include the risks from a period of bed rest (HEAD TO TOE: depression, confusion, chest infections, pnuenomia, constipation, ileus, urinary tract infection, renal calculi, pressure sores, DVT/PE, muscle wasting, osteoporosis, joint contractures) and a probable painful non-union of the fracture. We can perform either an uncemented or cemented hip hemiarthroplasty. I would generally only use an uncemented hemiarhroplasty in very frail high risk patients as the implant can work loose in the femur, rattle around and cause pain. Cement allows a firm fixation of implant to bone, reduces the need for revision surgery and allows better mobility with less thigh pain. My choice [authors' opinion] in this situation would be a total hip arthroplasty rather than a cemented hemiarthroplasty in line with current NICE guidelines.

EXAMINER: Why do you prefer a THA rather than hemiarthroplasty? It is more expensive!

CANDIDATE: [At this stage you should take the presented opportunity to talk and cover the whole subject!] The THA has a better functional outcome than hemiarthroplasty and has better survivorship results. My choice will be a cemented double-tapered polished stem with long-term proven results (such as Exeter) with a cemented highly cross-linked PE and a relatively large head (such as 32 mm). We have data from the Swedish hip registry favouring THA for functional results. Recent NICE guidelines have endorsed such a practice in the selected population, which include mentally alert patient with good pre-injury mobility levels and who are relatively healthy. This patient ticks all the criteria and will benefit from THA. My practice is to use a relatively larger head such as 32 mm, to reduce the risks of dislocation. I would aim for correct orientation of components, good soft tissue balancing and restoration of leg length. The management would continue with aggressive rehabilitation including early mobilization with full weightbearing and repatriation to the place of usual abode. It also includes addressing any underlying metabolic abnormalities such as osteoporosis, risk assessment for falls and nutritional deficiency. Ideally the management will be carried out by a multidisciplinary team.

Minute 4

EXAMINER: She arrives at 18:00 to your ward. When will you undertake the surgery?

CANDIDATE: The surgery should be undertaken as soon as safely possible. It should not be rushed in the middle of the night, however, so if the patient is fit for anaesthesia then aim for surgery on the next morning's trauma list with the necessary theatre staff, kit and consultant cover available. It is important to optimize any correctable causes prior to surgery. This should be undertaken in an objective and efficient manner to avoid 'unnecessary' delay.

Minute 5

The examiner may talk about the potential complications of THA in this patient group, which may include higher risk of dislocation due to a previously mobile hip as opposed to stiff arthritic hip with capsular fibrosis, leg length discrepancy, cement reaction, infection, early loosening etc.

Structured oral examination question 3

Minutes 1 and 2

EXAMINER: A 49-year-old lady fell on the stairs. Her foot is very painful, bruised, swollen and she can't bear weight. The CT1 went to see her in A&E, but he is not sure what the problem is, what do you think? (Figure 8.3.)

CANDIDATE: AP and oblique radiographs of left foot. There is diastasis of >2 mm between the base of the first and second metatarsals, features suggestive of Lisfranc tarsometatarsal fracture dislocation. There is a small avulsed fragment of bone in that interval. This avulsion fracture could be from the insertion of the Lisfranc ligament into the base of the second metatarsal, called a 'fleck sign'. [Always ask for the lateral radiograph.]

EXAMINER: Okay, how will you manage this patient?

CANDIDATE: I would start with assessing the patient as a whole, following ATLS protocol. I would take relevant history: mechanism of injury, patient's general condition, past medical history, allergies, smoking as well as occupation and previous level of activity.

EXAMINATION OF THE INJURED FOOT:

- Soft tissue status, swelling, pain, tenderness and ecchymosis.
- Painful passive abduction/pronation.

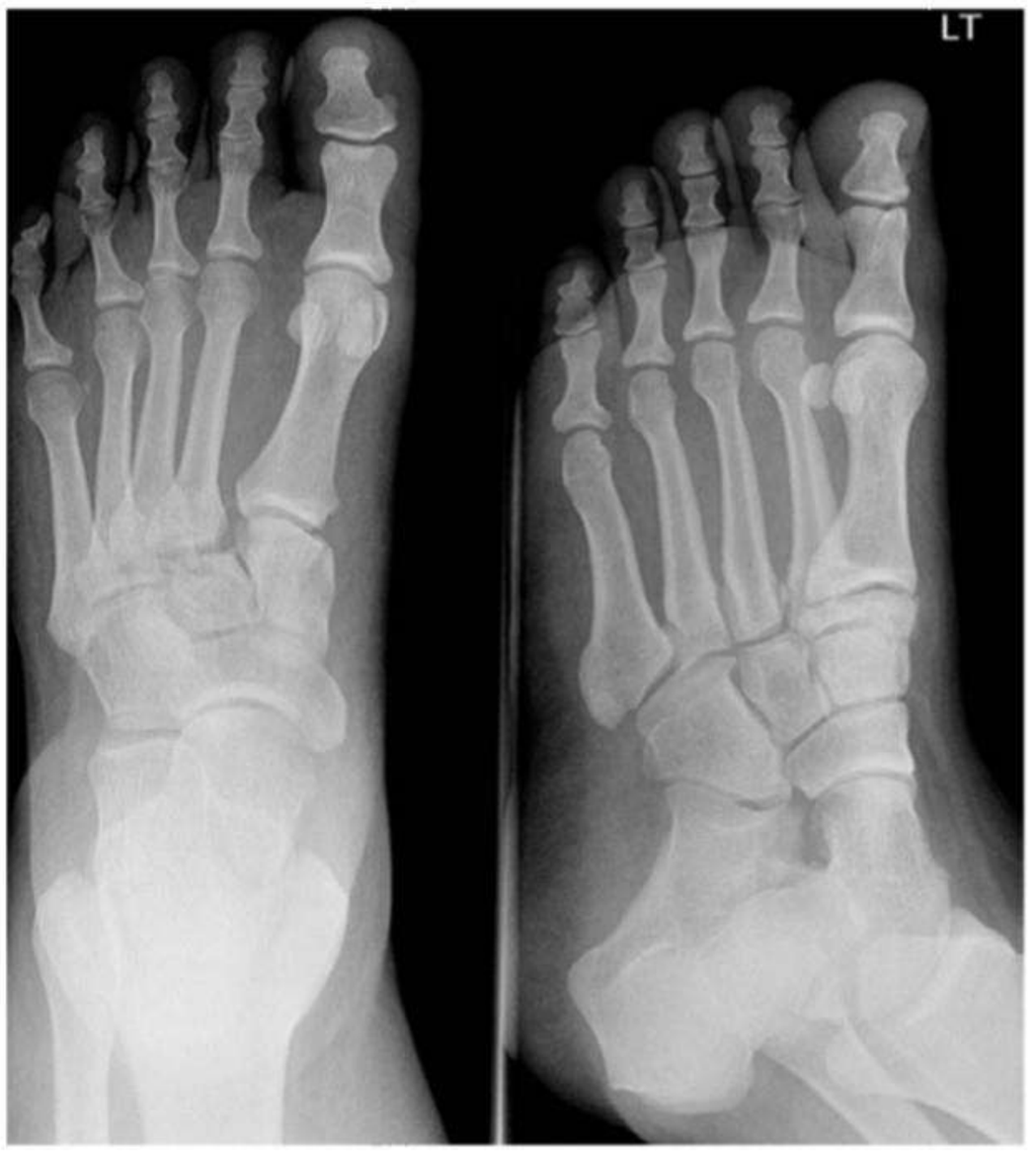


Figure 8.3 Anteroposterior (AP) and oblique radiographs left foot.

- Neurovascular status, dorsalis pedis pulse.
- Compartment syndrome must be excluded.

Following assessment, my initial management includes analgesia, elevation and splinting using below-knee backslab. On admission to hospital I would arrange for regular clinical examinations and monitoring in order not to miss an early developing compartment syndrome.

EXAMINER: What would you do if the radiographs were inconclusive in diagnosing this condition?

CANDIDATE: I would consider further radiographic imaging, oblique and lateral view, stress views and a CT scan or may opt for an MRI scan.

Minute 3

EXAMINER: How do you treat Lisfranc tarsometatarsal fracture dislocation?

CANDIDATE: This depends on severity of injury and degree of displacement of fracture. There is a role for non-operative management of an undisplaced stable injury or sprain which includes a non-weightbearing cast for 6 weeks and regular clinical and radiological review. However, in the presence of subluxation or dislocation, accurate reduction and stable

fixation is essential. In this case, I would consider open reduction and internal fixation with screws and possible plating, as required. With a severely comminuted fracture, primary arthrodesis of tarsometatarsal joints may be required.

Informed consent should be taken. The management options, postoperative rehabilitation, outcome and potential complications should be discussed in detail with the patient and documented in medical records.

Minute 4

EXAMINER: What prognosis will you give this patient?

CANDIDATE: This is a serious injury with potentially a poor outcome. Post-traumatic osteoarthritis may occur in more than 50% of cases despite surgical intervention. Residual pain and stiff foot are not uncommon complications of this injury. What makes the outcome of a serious condition even worse is that up to 20% of tarsometatarsal joint complex injuries are missed on initial examination. Patients must be informed about the length of treatment, recovery period and future implications for work and lifestyle.

Minute 5

EXAMINER: If this patient developed compartment syndrome, then how would you manage it?

CANDIDATE: Once compartment syndrome has been diagnosed clinically, emergency decompression is required. Theatre staff and anaesthetic on call team should be informed, informed consent must be obtained. I will take patient to theatre as soon as it is safe to do that. There is more than one technique described to decompress compartment syndrome of the foot, but I have been trained to decompress the nine compartments of the foot through three incisions, two dorsal over the second and third metatarsals and one on the medial side, just under the medial border of the first metatarsal. The patient will need to go back to theatre to have the wounds closed, once the soft tissue swelling has gone down.

Structured oral examination question 4

A 33-year-old roofer fell from a height of 20-feet, when scaffolding collapsed under him, landing on his feet. (Figure 8.4.)

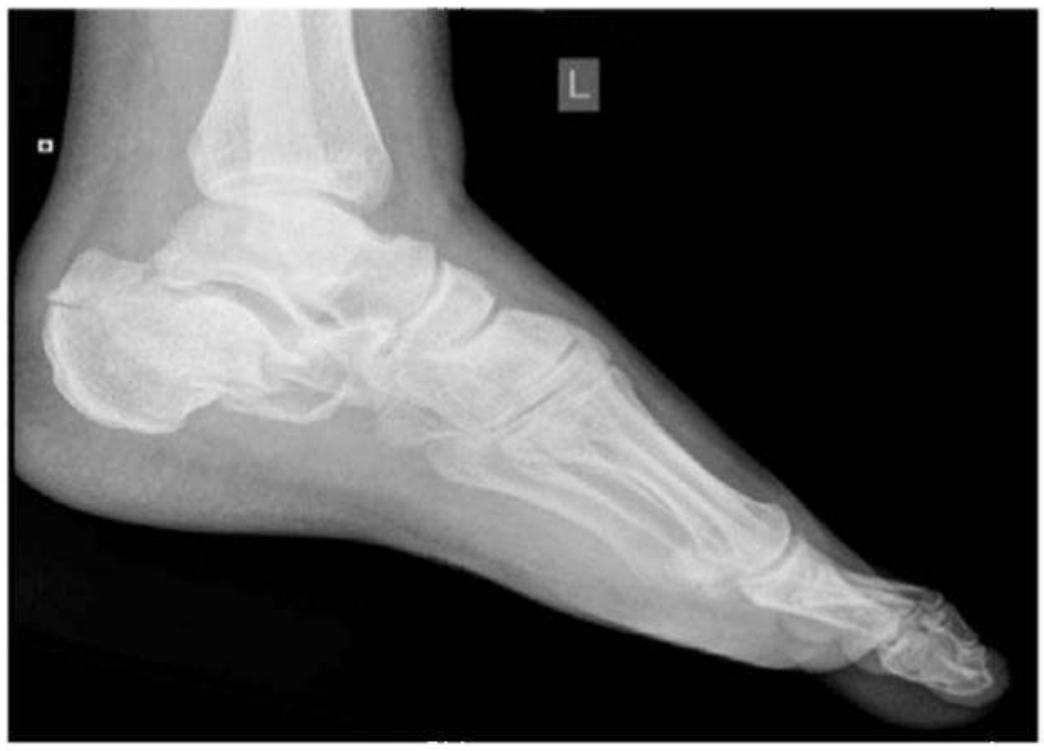


Figure 8.4 Radiograph left lateral foot.

Minute 1

EXAMINER: This is a radiograph of his foot. What are your thoughts?

CANDIDATE: This is a radiograph of left foot, lateral view. It shows a displaced intra-articular fracture of calcaneus with reduced calcaneal height. There is flattening or even reversal of Bohler's angle and a fracture of the calcaneal tuberosity.

A fall from a 20-feet height is a serious injury, so initially I would assess the patient as a whole following ATLS protocol (Airway and protect cervical spine, Breathing, Circulation, Disability, Exposure and environment control).

I will exclude potential associated injuries. These include compression fractures of the spine (10–15% of cases), fracture of proximal femur, fractures around the knee, ankle and other foot injuries, open fractures and neurovascular deficit.

Minute 2

EXAMINER: Assume that there are no other injuries, how would you manage this closed calcaneal fracture?

CANDIDATE: My management would start with initial management, followed by further investigation, planning and then definitive management.

Initial management includes analgesia, splinting, foot elevation and monitoring for compartment syndrome of foot and status of the soft tissue envelope as well as patient reassessment. The key is to manage the soft tissue envelope, which may require cryotherapy and use of foot pumps to reduce the swelling. CT scan would be arranged to plan definitive management. Patient's factors including medical

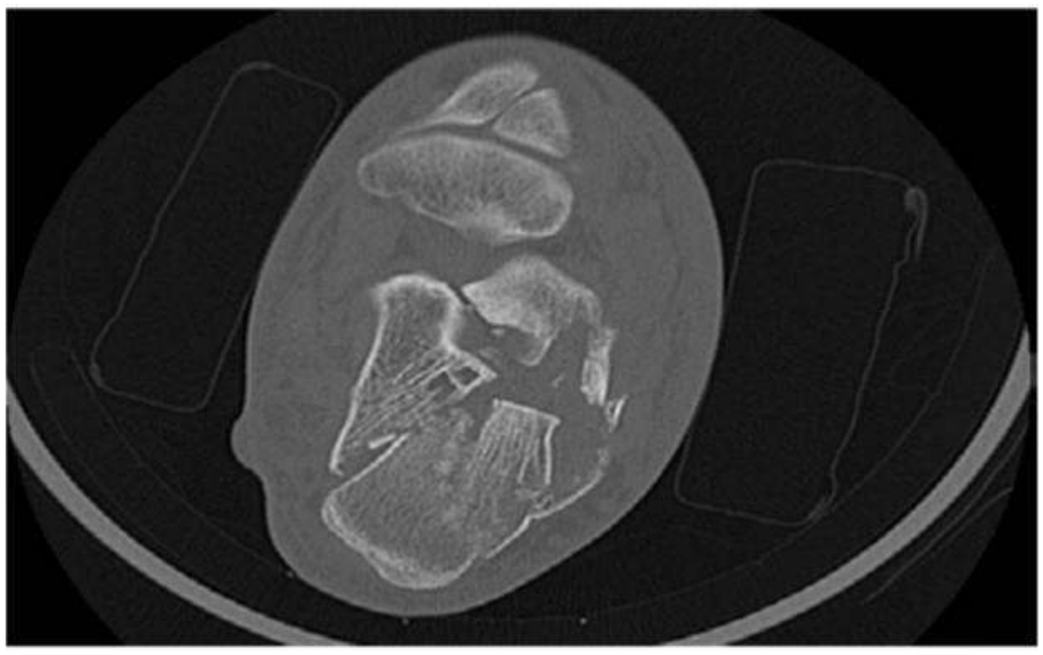


Figure 8.5 CT scan axial view left foot demonstrating calcaneal fracture.

conditions such as diabetes and peripheral vascular disease as well as smoking and occupation.

EXAMINER: Okay, this is the CT scan you requested, what can you see and what would you do next? (Figure 8.5.)

CANDIDATE: This CT scan section in axial view shows shorting of the calcaneus, varus deformity with a comminuted displaced fracture. There is a large sustentacular fragment, depressed middle fragment and blow-out of lateral wall. It also shows considerable heel widening.

I would discuss management options with the patient including open reduction and internal fixation once the soft tissue envelope has settled and swelling gone down. I would base the decision on the fracture pattern, soft tissue status and patient factors. This fracture pattern would benefit from surgical intervention but the decision will depend on factors such as smoking, drinking pattern, occupation, systemic illnesses and expectations of the patient.

Minute 3

EXAMINER: Following discussion with the patient you have decided to proceed with internal fixation. How will you do it?

CANDIDATE: I would take informed consent from the patient. General anaesthesia, prophylactic antibiotics, tourniquet, lateral decubitus position and fluoroscopy control. I would use an L-shaped lateral incision taking care to avoid any damage to the sural nerve. I would keep full thickness flap by taking the incision down to the bone, I would use K-wires bent over themselves to act as retractors, take off the lateral wall, manipulate the fracture fragments to restore length and height of calcaneum as well as correction of varus deformity and reconstruct the articular surface and then reapply the lateral wall. I would use K-wire for temporary fixation and definitively fix using screws and a calcaneal plate. My preference is a low-profile lateral calcaneal plate, the size of which depends on the patient's calcaneus and I would contour the plate prior to application. The key is to capture the sustantacular fragment under fluoroscopy. Postoperatively, the patient should mobilize non-weightbearing for 6 weeks and then a further 6 weeks of partial weightbearing.

EXAMINER: What prognosis will you give for this patient?

CANDIDATE: A calcaneal fracture is a significant injury with a high incidence of long-term pain and disability. Thordarson & Krieger and Buckley *et al.* reported a more favourable outcome associated with open reduction and internal fixation, compared to non-operative treatment.[1,2] Similar findings were also reported by Potter & Nunley.[3] Management of calcaneum fracture is an ongoing controversial issue and attracts a lot of debate.

In other vivas, candidates were asked about associated injuries, Sanders' classification and dealing with complications.

1. Thordarson DB, Krieger LE. Operative vs. nonoperative treatment of intra-articular fractures of the calcaneus: a prospective randomized trial. *Foot Ankle Int* 1996; **17**(1):2–9.
2. Buckley R, Tough S, McCormack R *et al.* Operative compared with nonoperative treatment of displaced intra-articular calcaneal fractures: a prospective, randomized, controlled multicenter trial. *J Bone Joint Surg Am* 2002;**84-A**:1733–1744.
3. Potter MQ, Nunley JA. Long-term functional outcomes after operative treatment for intra-articular fractures of the calcaneus. *J Bone Joint Surg Am* 2009; **91-A**:1854–1860.

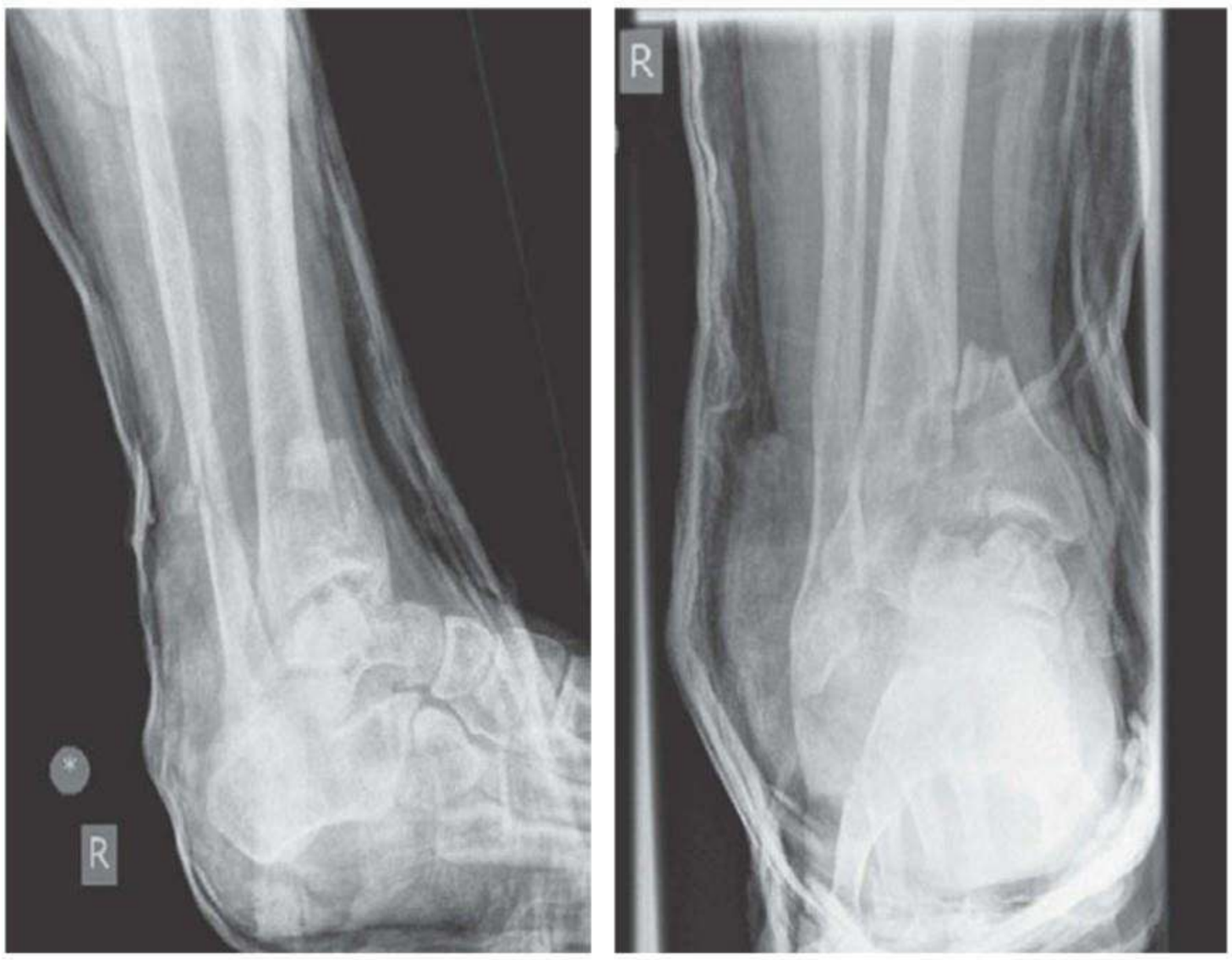
Structured oral examination question 5

A 21-year-old motorcyclist was involved in a road traffic accident. He was brought to A&E fully conscious and alert, and formal assessment following ATLS protocol showed that this was an isolated closed injury. (Figure 8.6.)

Minute 1

EXAMINER : Tell me how would you manage this injury?

CANDIDATE: This is an intra-articular fracture, around the right ankle, as a result of high energy trauma. There is a pilon fracture, fractured fibula, disruption of the syndesmosis and possibly a fractured talus.



Figures 8.6a and 8.6b Anteroposterior (AP) and lateral radiographs right lower leg.

I will take a relevant history and perform a detailed clinical examination, including the skin and soft tissue condition, realign the foot into a better position, relieve pressure on the skin to avoid skin necrosis, perform a neurovascular assessment of the involved limb and exclude compartment syndrome. The limb should be splinted and elevated. Regular monitoring of neurovascular status and detection of a developing compartment syndrome is of paramount importance.

Minute 2

CANDIDATE: My principles of treatment are 'Span–Scan–Plan'.

- Span: to reduce fracture and joint, and immobilize the injured limb using spanning external fixator. This will restore alignment and allow soft tissue resuscitation and monitoring. This will also give us time to plan definitive surgery.
- Scan: CT scan will provide more details of fracture type and pattern, therefore allowing for informed planning of definitive treatment.
- Plan: Definitive treatment in details; approach, how to fix the fragments, what implant to use, timing of surgery, obtaining consent from the patient and to get an indication of what can be achieved and possible outcome.

Minute 3

EXAMINER: When are you going to fix this fracture?

CANDIDATE: There are many factors to be considered, this is a serious injury and it must be treated properly in one operation. The patient needs to be resuscitated; the soft tissue envelope needs to be in a good condition (this may take 2–3 weeks to settle), a CT scan needs to be performed and the required surgical expertise and implant should be available.

EXAMINER: How are you going to fix this fracture?

CANDIDATE: Principles of intra-articular fracture fixation are anatomical reduction, fracture stabilization and early mobilization. My approach would be tailored depending on fracture configuration, which would identified by a CT scan prior to surgery and with fluoroscopy during surgery. Looking at the fractures in the radiographs provided, I would favour an anterolateral approach that will allow me to reduce the pilon fracture, and approach the fibula as well as the talus. I would aim for anatomical reduction of the pilon fracture and stabilization using an anterolateral plate and then go on to address the fibula and talus on their relative merits. Exposure of the talus can be improved by extending the release of the ATFL through a subperiosteal approach. I would anatomically reduce and fix the talus fracture with screws making sure any loose debris is removed from the joint.

Minutes 4 and 5

EXAMINER: What is the functional outcome following a pilon fracture?

CANDIDATE: Well, generally a poor outcome. A patient may end up with a stiff ankle with a poor soft tissue envelope and there is considerable risk of post-traumatic osteoarthritis which may require an ankle arthrodesis or ankle arthroplasty.

EXAMINER: What is Hawkins' sign – is it a good or bad sign?

CANDIDATE: It is the appearance of osteopenia in subchondral bone of the talar dome, 6–8 weeks following a talar neck fracture. It is a good sign and means that the talar dome is well perfused and has a viable blood supply. It indicates that bone healing is likely to occur without the development of avascular necrosis.

Structured oral examination question 6

A 50-year-old woman, front-seat passenger, was involved in a head-on high speed car collision. In A&E she was diagnosed with a right hip dislocation.

Minute 1

EXAMINER: What will be your management?

CANDIDATE: I would assess and resuscitate the patient along ATLS principles, exclude any associated injuries. I would examine the lower right leg looking at alignment, position and document neurovascular status especially of the sciatic nerve. With the mechanism of injury it is likely to be posterior dislocation with often an associated acetabular rim or femoral neck fracture. In a posterior dislocation the leg is usually shortened and internally rotated, whilst with anterior dislocations the leg is externally rotated and abducted.

EXAMINER: It appears to be an isolated injury with paraesthesia in the sole of the foot, however motor function is intact. How will you manage this further?

CANDIDATE: A traumatic hip dislocation is a surgical emergency because of the risks to the vascularity of the femoral head, danger of chondrolysis as well as pressure effects on the surrounding soft tissues especially neurovascular structures. The paraesthesia in the foot is an indication of pressure or traction involving the sciatic nerve. I would arrange urgently for the patient to go to the theatre the same night. I would get an urgent CT scan performed provided it did not delay surgery; inform theatres, the anaesthetic team and the ward. I would arrange for haematological and biochemical investigations including crossmatch. I would take an informed consent for a closed or open reduction under general anaesthesia.

Minute 2

EXAMINER: There is delay in getting the CT scan and you take her to the operating theatre. How will you reduce the hip?

CANDIDATE: I would attempt closed reduction under general anaesthesia. I would position her supine on the table with the table height as low as possible. I would request the anaesthetist to use muscle relaxant to make it easier to reduce the hip. I would stand on the side of the dislocated hip and have the image intensifier (II) come in from the opposite side. My assistant would be on the opposite side towards the head end of the patient to hold down and stabilize her pelvis when I attempt manipulation. I would screen her first before attempting reduction to exclude a femoral neck fracture and assess the acetabulum using Judet views. If it is a posterior dislocation, I would gently apply traction on the hip (in-line) and then gradually flex the hip and the knee maintaining traction. I would check reduction under II and also check once again for any associated fractures.

EXAMINER: You manage to reduce the hip and get this radiographic image. What are your thoughts? (Figure 8.7.)

CANDIDATE: In this II view of the right hip, the femoral head appears to be in the acetabulum but is incongruent. I would like to confirm this on lateral radiograph. The femoral head is inferiorly subluxed and there appears to be a bony fragment in the hip joint superiorly and another one inferiorly. There is

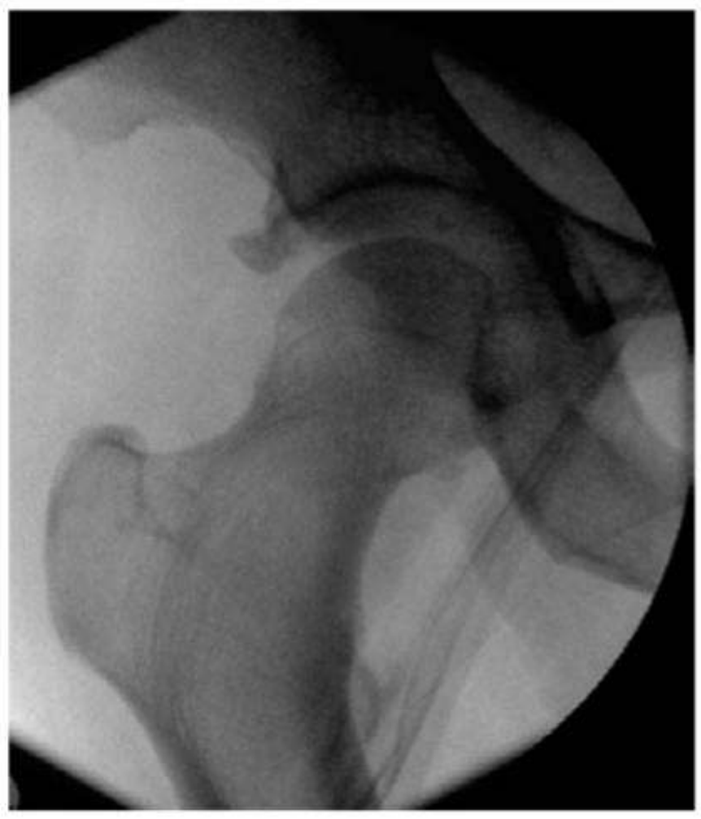


Figure 8.7 Image intensifier (II) image right hip.

one more fragment on the superolateral lip of the acetabulum. The bony fragments are most likely to be from the acetabulum, however femoral head fragments need to be ruled out.

Minute 3

EXAMINER: How will you assess this hip further?

CANDIDATE: Peroperatively I would screen the hip in AP and lateral views as well as obtaining Judet views to assess the anterior and posterior walls and columns. In addition, I would assess the hip for stability. A CT scan will be useful to delineate this further, if it has not already been done.

EXAMINER: You obtain a CT scan in the morning and this is one of the sections. What do you think? (Figure 8.8.)

CANDIDATE: [Note: just comment on what you have rather than ask for more images.] In this axial section of the pelvis at the level of the hip joints, I note that the femoral head on the injured side is at a different height to the opposite hip. There is a bony fragment trapped in the hip joint as well as a bony fragment lying posterior to the hip. It looks like a fracture dislocation with compromise of the acetabular wall postero-superiorly. I would need to study the whole CT sequence to ascertain the extent of damage.

EXAMINER: How will you deal with the bony fragment in the hip joint?

CANDIDATE: This depends on a number of factors including the exact original site of the bony fragment, the size of it, weightbearing dome state and the stability of the hip. The options for a bony fragment trapped in the hip joint are to remove it or to retrieve and fix it. If the fragment is quite small and does not affect hip stability or the weightbearing dome then it can be removed arthroscopically. However, if it compromises the weightbearing area of the hip or stability then I would retrieve it and fix it. It will have to be an open procedure though reports of arthroscopic intervention have been published. [Note: If the candidate does not have sound hip arthroscopy knowledge, then the safe option is open procedure and stay clear of hip arthroscopy.]

Minute 4

EXAMINER: You find that it is the postero-superior lip of the acetabulum. Which approach will you use to fix the fracture?

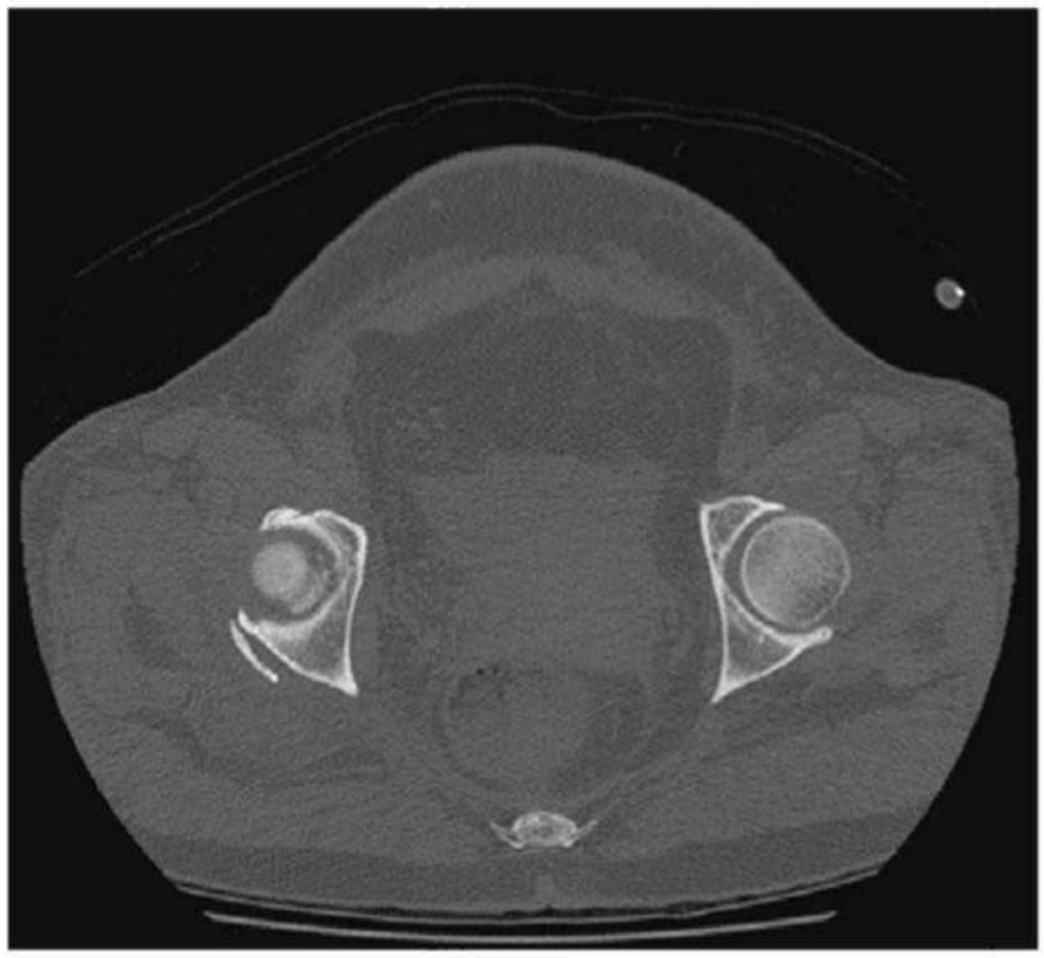


Figure 8.8 CT axial view pelvis.

CANDIDATE: The approach depends on where the bony fragment is arising from. If it is postero-superior or posterior, I would use a posterior approach. I would position the patient on the fracture table in lateral decubitus. To preserve the blood supply to the femoral head, I would remove the fractured loose body arthroscopically [however, see the argument above]. In the posterior approach to the hip one needs to respect the blood vessels supplying the femoral head and therefore not take down the quadratus or the short rotator muscles. I would retract the gluteus medius superiorly, identify the capsule and dissect superiorly to identify the fractured rim of the acetabulum. I would reduce the fragment anatomically under image intensifier screening and secure it with 2–3 partially threaded cannulated screws making sure that the screws do not penetrate the hip joint.

Minute 5

EXAMINER: What are the risks with posterior dislocation of the hip?

CANDIDATE: Immediate complications are neurovascular damage (10% with posterior dislocation), fractures and haemorrhage. Intermediate risks are chondrolysis, post-reduction neurological damage, avascular necrosis, intra-articular loose bodies and hip instability. Late complications are pain and post-traumatic arthritis.

EXAMINER: What other injuries are associated with this injury pattern?

CANDIDATE: This is determined by the direction of forces and may include patella fracture, PCL rupture, femoral fracture, femoral neck and head fractures.

[Note: The candidate is smiling as the examiner has run out of questions on his crib sheet!]

Structured oral examination question 7

A 78-year-old woman fell out of her bed and sustained this injury. She is in reasonably good health and independently mobile, able to care for herself and do her own shopping. (Figure 8.9.)

Minute 1

EXAMINER: What can you see and how you going to manage her?

CANDIDATE: This is an AP radiograph of the right hip showing a reverse-obliquity inter-trochanteric fracture with subtrochanteric extension. The lesser trochanter is proximally displaced with loss of the medial buttress. I would like to see a lateral radiograph, however, based on just the AP view, it is an unstable fracture pattern.

My management for this patient would start with a thorough assessment and optimization of her general medical condition. We need to exclude the possibility of pathological fracture, although the available radiograph shows no evidence of that. I would obtain full-length radiographs of the femur. Provided she is fit and agrees to surgery, I would aim to manage this fracture operatively and I will do so as early as possible, preferably within 36 hours of admission [new NICE guidelines]. I would use a cephalomedullary device as this has shown better results rather than fixed-angle plating devices in this fracture configuration.[1,2]

Minute 2

EXAMINER: This woman's fracture was managed elsewhere and presents during your on-call week with this complication. Can you explain what has happened? (Figures 8.10 and 8.11.)

CANDIDATE: This lady was treated with a fixed-angled locking plate. Two things are perhaps responsible for this failure: biomechanics and biology.

Looking at the postoperative radiograph, there is a gap at the fracture site especially medially. The fixed-angled device has been used with locking screws with five screws on either side of the fracture, which will make it a very rigid implant. This will prevent any micro-motion necessary for callus formation. In addition, there is a fracture gap and lack of compression that will preclude primary bone union. This has resulted in a delayed union/atrophic non-union at the fracture site.

The implant has been under constant biomechanical load, which has led to the fatigue failure of the implant. In this particular design there is a stress riser at the junction of the last proximal locking hole and the tapered part of the plate, which dictates the failure point in the implant. In addition, the plating device is applied on the lateral aspect of the femur increasing the lever arm for the moment of force as compared with a cephalomedullary device, which further puts the fixed-angle

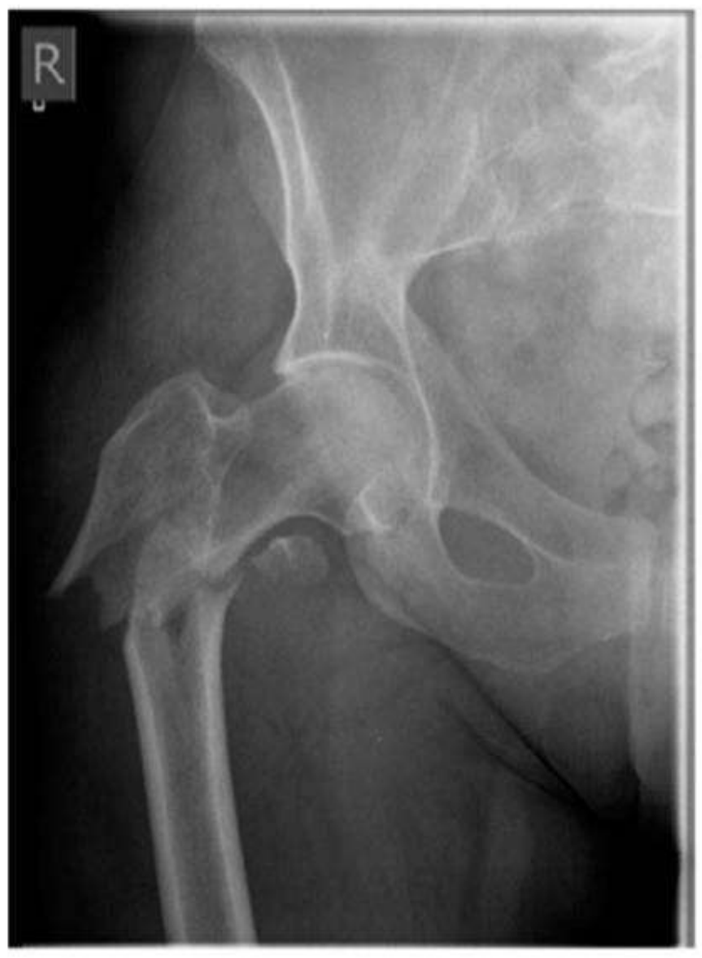


Figure 8.9 Anteroposterior (AP) radiograph right femur demonstrating inter-trochanteric fracture.

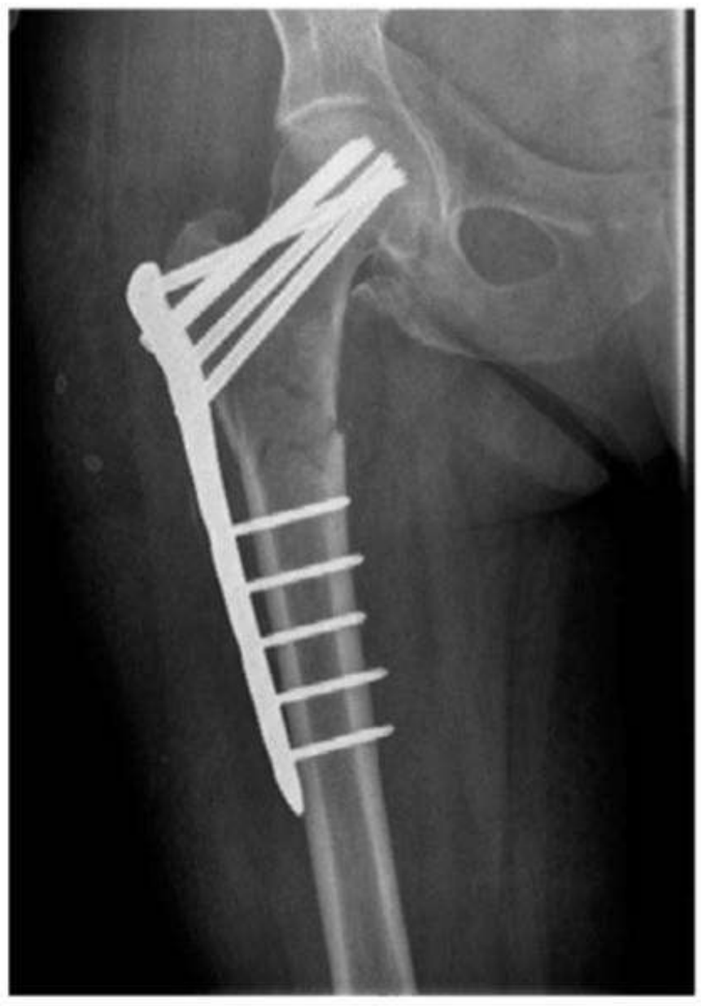


Figure 8.10 Anteroposterior (AP) radiograph right femur with fixed locking plate *in situ*.

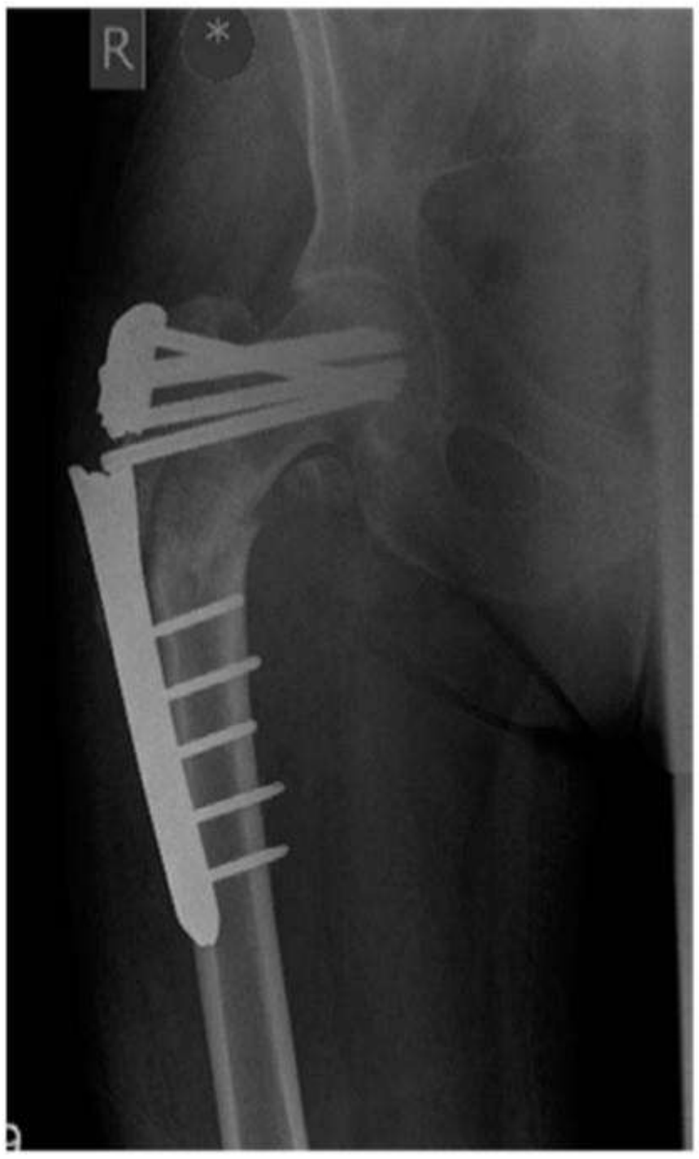


Figure 8.11 Anteroposterior (AP) radiograph right femur demonstrating hardware failure, 4 months postoperative.

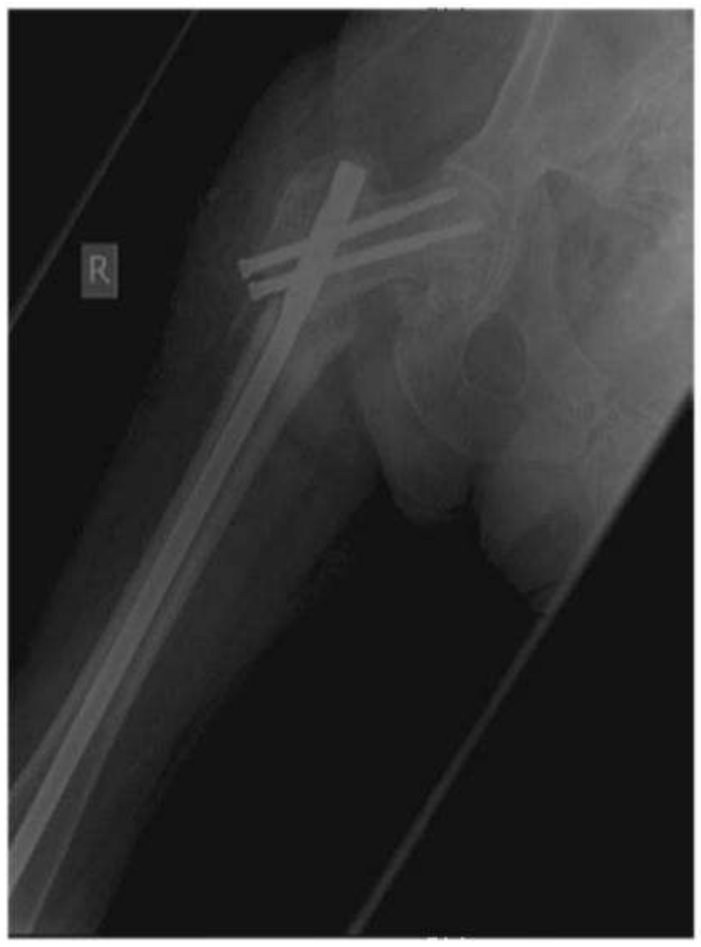


Figure 8.12 Anteroposterior (AP) radiograph demonstrating non-union of the femoral fracture.

plating device in this position at a biomechanical disadvantage. Similar results were reported with the use of compression hip screw and 95° plate.[2] In this type of fracture an intramedullary device has better results and biomechanical stability.

Minute 4

EXAMINER: You fixed it with this recon nail. What do you think about your check X-ray? (Figure 8.12.)

CANDIDATE: As I mentioned earlier the literature reports better results with the use of a cephalomedullary nail. I hope that when the recon nailing was performed bone grafting to the fracture site was performed as well so as to address both biomechanics and biology. The cephalomedullary nail is in slight varus and there is some translation at the fracture site. The screws in the proximal fragment are a bit superior to where I would normally like them. The screws are not absolutely parallel and I would study my lateral radiographs carefully to make sure that the screws have not missed the head.

Minute 5

EXAMINER: How will you follow-up this patient?

CANDIDATE: I would follow-up this patient with clinical reviews and serial radiographs until the fracture heals. I would start her weightbearing as able, stop NSAIDs, counsel against smoking if she does, keep an eye on her inflammatory markers and do serial radiographs 6 weeks apart. If there is no callus formation at 3–4 months, I would consider revising the intramedullary nail with autologous bone grafting.

1. Sadowski C, Lübbeke A, Saudan M *et al.* Treatment of reverse oblique and transverse intertrochanteric fractures with use of an intramedullary nail or a 95 degrees screw-plate: a prospective, randomized study. *J Bone Joint Surg Am* 2002;**84-A**:372–381.
2. Kregor PJ, Obremskey WT, Kreder HJ, Swiontkowski MF. Evidence-Based Orthopaedic Trauma Working Group. Unstable pertrochanteric femoral fractures (Review). *J Orthop Trauma* 2005;**19**:63–66.

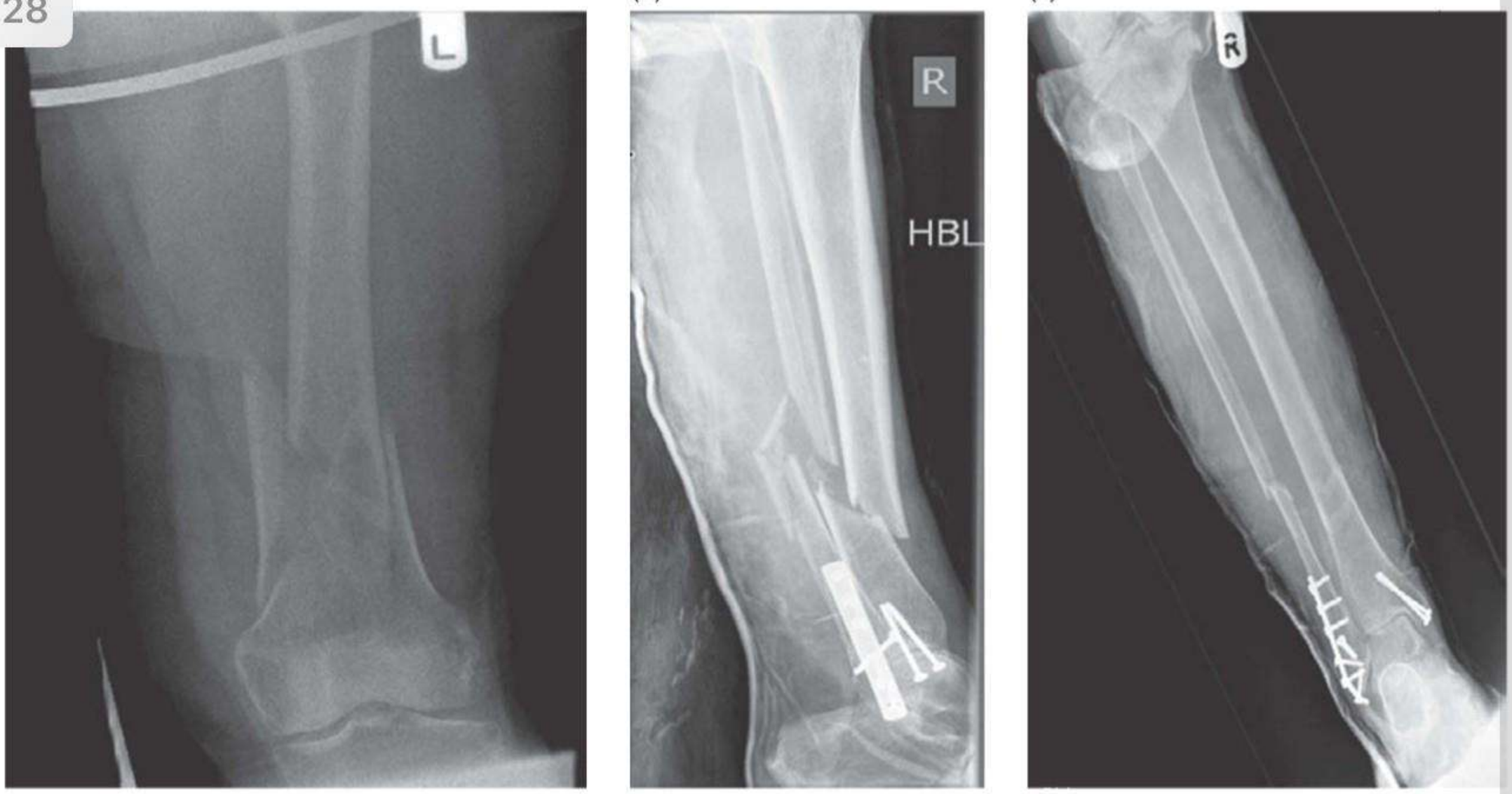
Structured oral examination question 8

A 72-year-old woman, fully independent with good health, was hit by a car when she was walking on a kerb. She was brought to hospital with these injuries. She was assessed following ATLS protocol. She was resuscitated and her injuries were splinted. (Figure 8.13.)

Minute 1

EXAMINER: What your thoughts about this patient's management? Do you have any concerns?

CANDIDATE: This 72-year-old lady has multiple high-energy injuries. This is a serious situation, so although she had good health prior to this accident, I would be concerned about her trauma response and her well-being. Trauma scores show that elderly patients have limited physiological reserves and they tend to do worse than young people. So she needs to be closely



Figures 8.13a, 8.13b and 8.13c Anteroposterior (AP) radiograph left femur demonstrating supracondylar fracture femur and AP and lateral radiographs right lower leg.

observed and kept well hydrated and her condition optimized before undertaking definitive treatment. Fractures of long bone should be stabilized as early as possible for many reasons: for pain relief, to reduce trauma response, to allow for early mobilization and rehabilitation as well as to decrease the complications from bed rest.

Ideally an anaesthetic team and geriatrician should also be involved in planning her treatment, when to take her to theatre.

Minute 2

EXAMINER: What implants are you going to use to fix these fractures?

CANDIDATE: For the left femur fracture, it is a multi-fragment supracondylar unstable fracture, with femur shortening. We need to use a femoral distracter for temporary reduction and then to use either a nail or plate. Personally I would prefer a fixed-angle plate with less invasive technique. Nailing may increase the risk of ARDS and fat embolism.

Regarding the tibia fracture, it is a fracture of the distal third of tibia and there is metalware from a previous ankle fracture fixation. Although an intramedullary nail could be used, we still have the same argument of fat embolism and ARDS. The fracture could be plated but this means soft tissue stripping and is perhaps not ideal for a 72-year-old woman's leg which is already contused and swollen with the high-impact injury. A circular frame would be a valid option and I would prefer to use it.

Minutes 3 and 4

EXAMINER: Okay, have a look at this radiograph and explain to me the technique the surgeon has used and what the principles are of this technique. (Figure 8.14.)

CANDIDATE: The AP radiograph shows a multifragment distal diaphysis/metaphysis fracture that has been stabilized with a fixed-angle plate, using bridging plating the fracture zone has been bridged. Looking at the skin staples used to close the skin, I can infer that a closed indirect reduction and a less invasive technique was used. This technique was introduced to decrease soft tissue disruption and preserve blood supply. Length, alignment and rotation of bone was restored. Baumgaertel *et al.* introduced the concept of biological plating and proved that indirect reduction and bridge plating was superior to direct fragment reduction and anatomical fixation in respect to bone healing.[1]

Chapter 8: Lower limb trauma

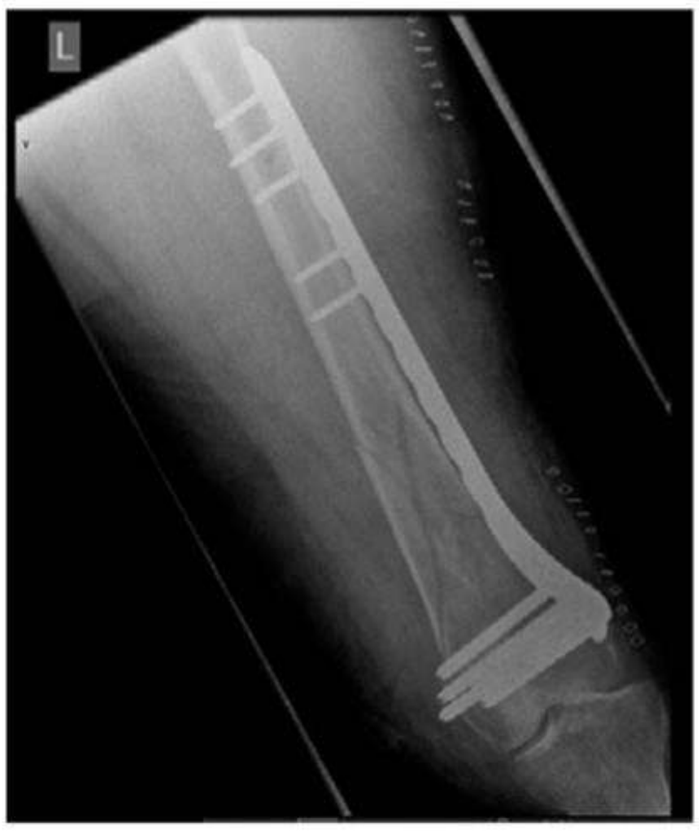


Figure 8.14 Anteroposterior (AP) radiograph left distal femur with locking plate *in situ*.

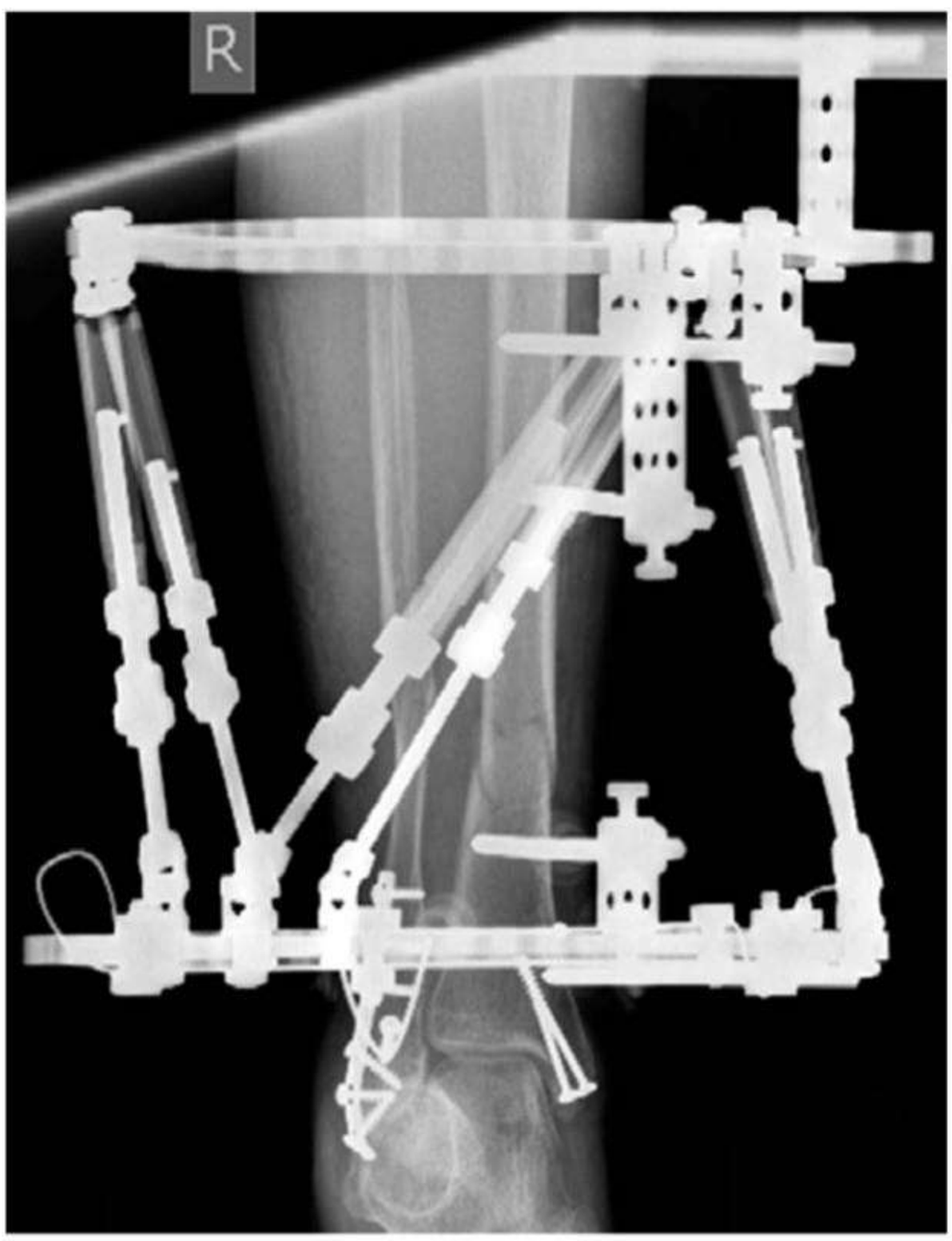


Figure 8.15 Anteroposterior (AP) radiograph right distal tibia with circular frame in situ.

EXAMINER: Can you explain why the surgeon put screws on either ends of the plate and missed the middle?

CANDIDATE: The surgeon intended to increase the working length of the implant (the distance between two points on either side of the fracture where the bone is fixed to plate or nail). This produces an even distribution of forces over a long segment and decreases stress at fracture and implant.

Minute 5

EXAMINER: Can you tell me the principles of using a circular frame? (Figure 8.15.)

CANDIDATE: It uses tensioned wires for bone fixation and these wires are fixed to rings to form segments proximal and distal to fracture site. Segments of frame can be moved in terms of angulation, rotation, translation and length. The frame can be built to fit all bones and can be temporary or definitive fixation. It can be fitted using a less invasive technique and can be adjusted when needed.

1. Baumgaertel F, Buhl M, Rahn BA. Fracture healing in biological plate osteosynthesis. *Injury* 1998;**29** (Suppl. 3):C3–C6.

Structured oral examination question 9

A 29-year-old female horse rider fell off her horse; she has been fully assessed in A&E and has an isolated closed injury of the foot. (Figure 8.16.)

Minute 1

EXAMINER: What are your views?

CANDIDATE: The radiographs of the left foot, AP and oblique show a displaced fracture of the body of navicular bone with comminution. There is overlap of mid-tarsal bones and I can't exclude fractures of other tarsal bones. The alignment of the foot is still maintained and there is no varus or valgus deformity.

This is a serious injury, probably high energy. I need to assess the patient as a whole: relevant history, clinical examination in general and in particular of the foot ruling out compartment syndrome, any neurovascular damage and assessing the soft tissue envelope of the foot. I would then request further imaging, the modality of choice being CT scan.

I would initially treat the injured foot in a backslab, with strict elevation and intermittent cryotherapy, adequate analgesia and close monitoring for evolving compartment syndrome.

Minute 2

EXAMINER: This is the scan you requested. What do you see, and how would you manage it? (Figure 8.17.)

CANDIDATE: These coronal and sagittal sections of the CT scan confirm X-ray findings of a displaced fracture of the body of navicular bone with comminution. It is an unstable displaced

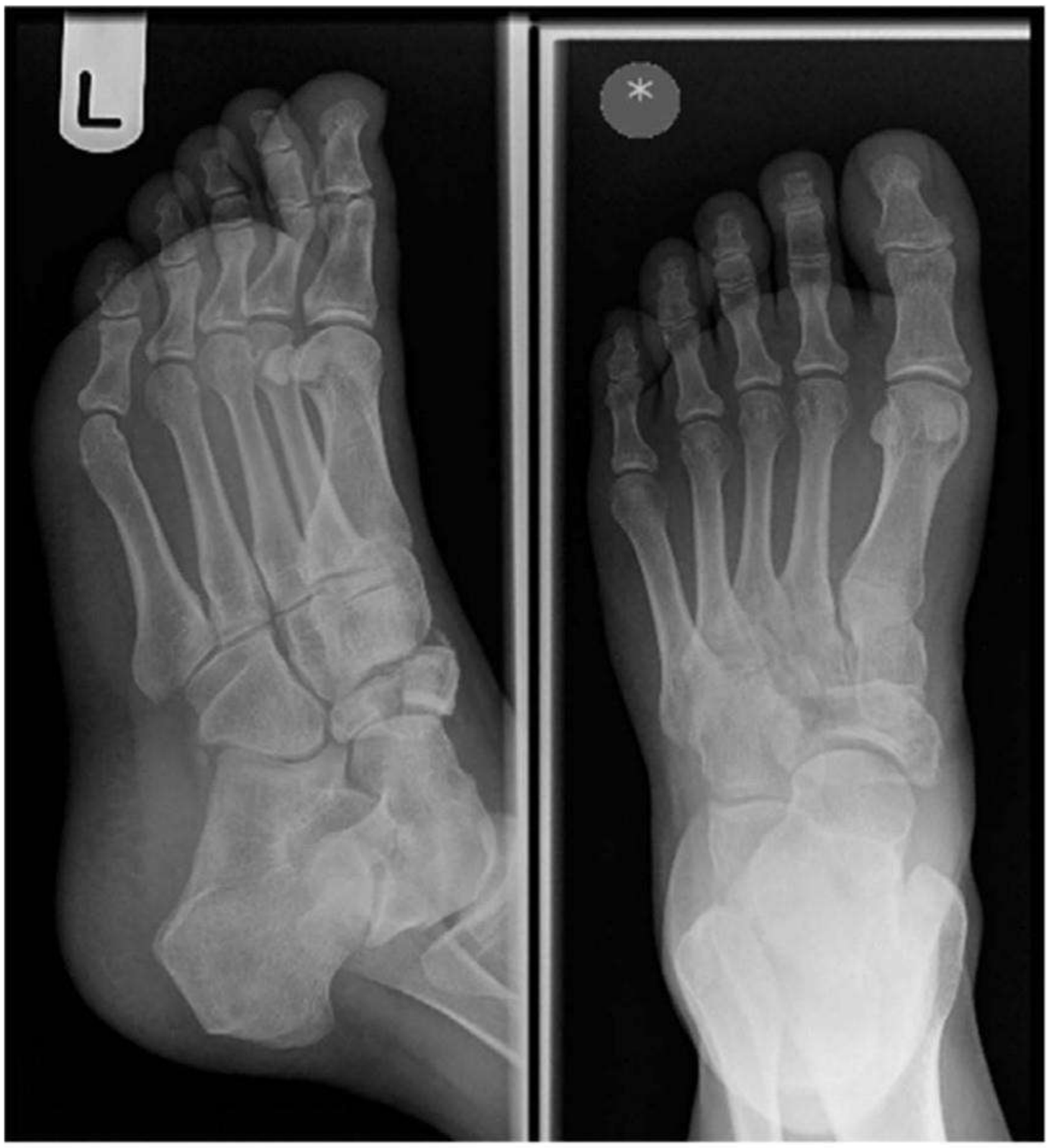
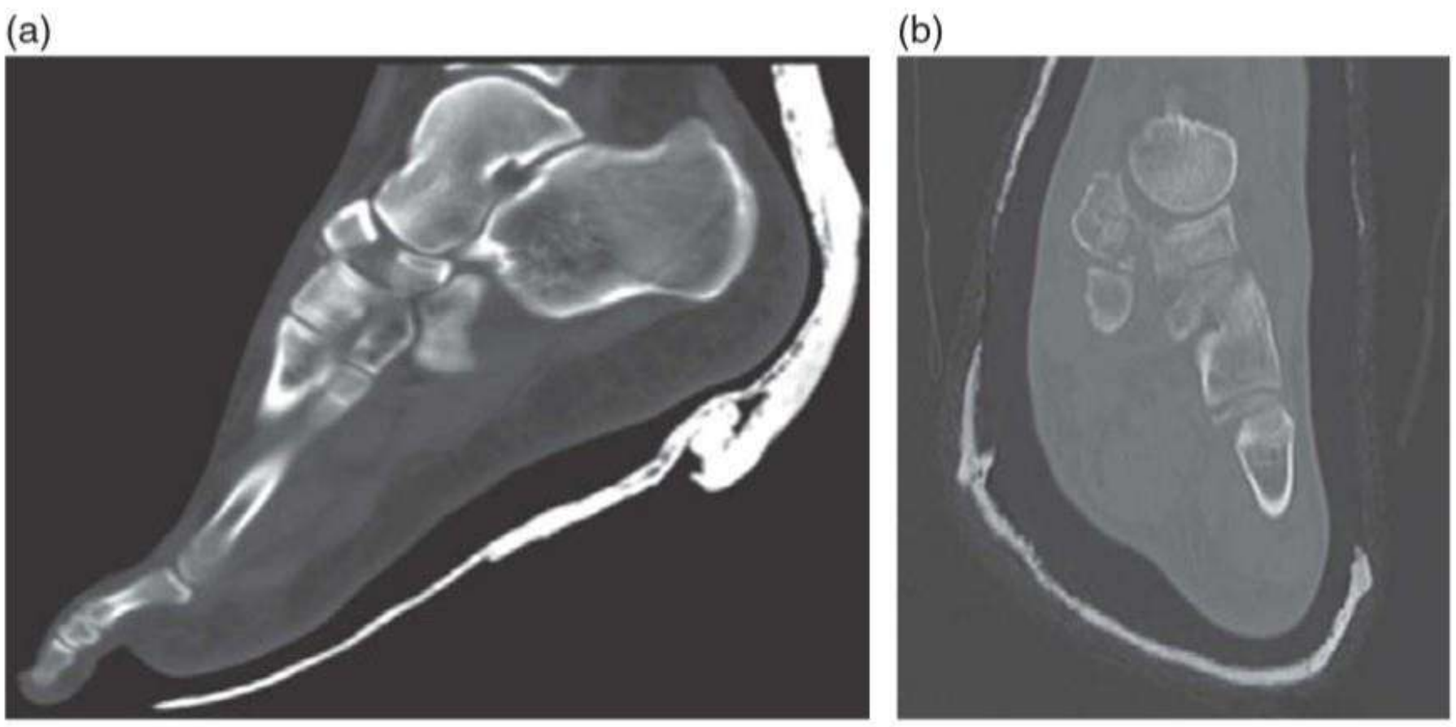


Figure 8.16 Anteroposterior (AP) and lateral radiographs left foot.



Figures 8.17a and 8.17b CT scan coronal and sagittal sections of left foot.

intra-articular fracture and I would favour operative intervention rather than non-operative. The principles of management are to restore the articular surface, stabilize and hold the fracture to allow early mobilization. The aim of the treatment is to have a mobile, pain-free and functional joint. However, sometimes that is not possible due to severe comminution of the articular surface, in which case I may consider primary fusion of the talonavicular joint.

I would discuss findings, management options, aims of the treatment as well as potential complications with the patient and seek informed consent before proceeding.

Minute 3

EXAMINER: Can you tell us about possible complications associated with this case?

CANDIDATE: These are immediate, early and late complications. Immediate complications are in the perioperative period and include iatrogenic injury to structures, compartment syndrome and anaesthetic problems. Early complications include infection, nerve injury (branches of superficial and deep peroneal nerves) and vascular injury (dorsalis pedis). Late complications include non-union and loss of medial longitudinal arch support, painful talonavicular joint, post-traumatic osteoarthritis, as well as avascular necrosis and collapse.

Minute 4

EXAMINER: Why do non-union and avascular necrosis occur in this fracture?

CANDIDATE: The navicular bone, similar to talus, has a large articular surface area and for the blood supply it relies on the radial arcade of vessels arising from the dorsalis pedis and medial planter arteries and this could be injured either at the time of fracture or during surgery, which could lead to AVN, non-union and/or collapse of the bone resulting in a painful mid-foot.

EXAMINER: What surgical approach are you going to use?

CANDIDATE: I would use a medial approach, between the tibialis anterior and tibialis posterior tendons, preserving the remaining blood supply as much as possible, reduce the articular surface and stabilize with cannulated screws from lateral to medial. The eventual configuration of screws will depend on the fracture pattern.

Minute 5

EXAMINER: Take me through your consent process, in general.

CANDIDATE: I follow GMC guidelines: 'Consent: patients and doctors making decisions together'. I work in partnership with the patient to ensure high quality of care.

(a) I listen to patients and respect their views about their health;
(b) discuss with patients what their views about diagnosis, prognosis, treatment and care involve;
(c) share with patients the information they want or need in order to make decisions;
(d) maximize patients' opportunities, and their ability to make decisions for themselves;
(e) respect patients' decisions.

Remember what the golfer Gary Player said: 'The more I practise the luckier I get'. That is exactly what you need to pass FRCS Orth. Exam, practice and luck!

Section 3

Trauma

Chapter 9

Upper limb trauma

Gunasekaran Kumar

Structured oral examination question 1

EXAMINER: A 38-year-old left-hand dominant lady fell onto her right arm when out drinking and attended the casualty department the next day at 16:00 when pain in the right shoulder did not settle down. These are the X-rays of right shoulder. What is your diagnosis? (Figure 9.1.)

CANDIDATE: Fracture dislocation of the right shoulder. It is an anterior dislocation as the humerus is in an abducted position and the greater tuberosity is fractured.

EXAMINER: How will you manage this condition?

CANDIDATE: After assessing the patient for other associated injuries and neurovascular status I will try to reduce the right shoulder dislocation under sedation in casualty.

EXAMINER: What are the risks and complications you anticipate?

CANDIDATE: Since the greater tuberosity is fractured, there is a risk that there may be an undisplaced fracture of the humeral neck not seen on X-ray. Further, during manipulation of the shoulder there is a risk of propagating the fracture through the humeral neck. Other risks are injury to neurovascular structures including axillary nerve and artery or the brachial plexus.

EXAMINER: Attempted closed reduction in casualty failed and it is 19:00 now. What will you do next?

CANDIDATE: I will check with emergency theatres whether there is theatre space available and check with the on-call consultant whether it is okay to get the patient onto the emergency list for closed reduction. In case the humeral neck fractures during manipulation, it will require open reduction and stabilization. I am not experienced with this procedure. Hence, I would prefer the consultant to be around or perform the reduction next day first in the trauma list.

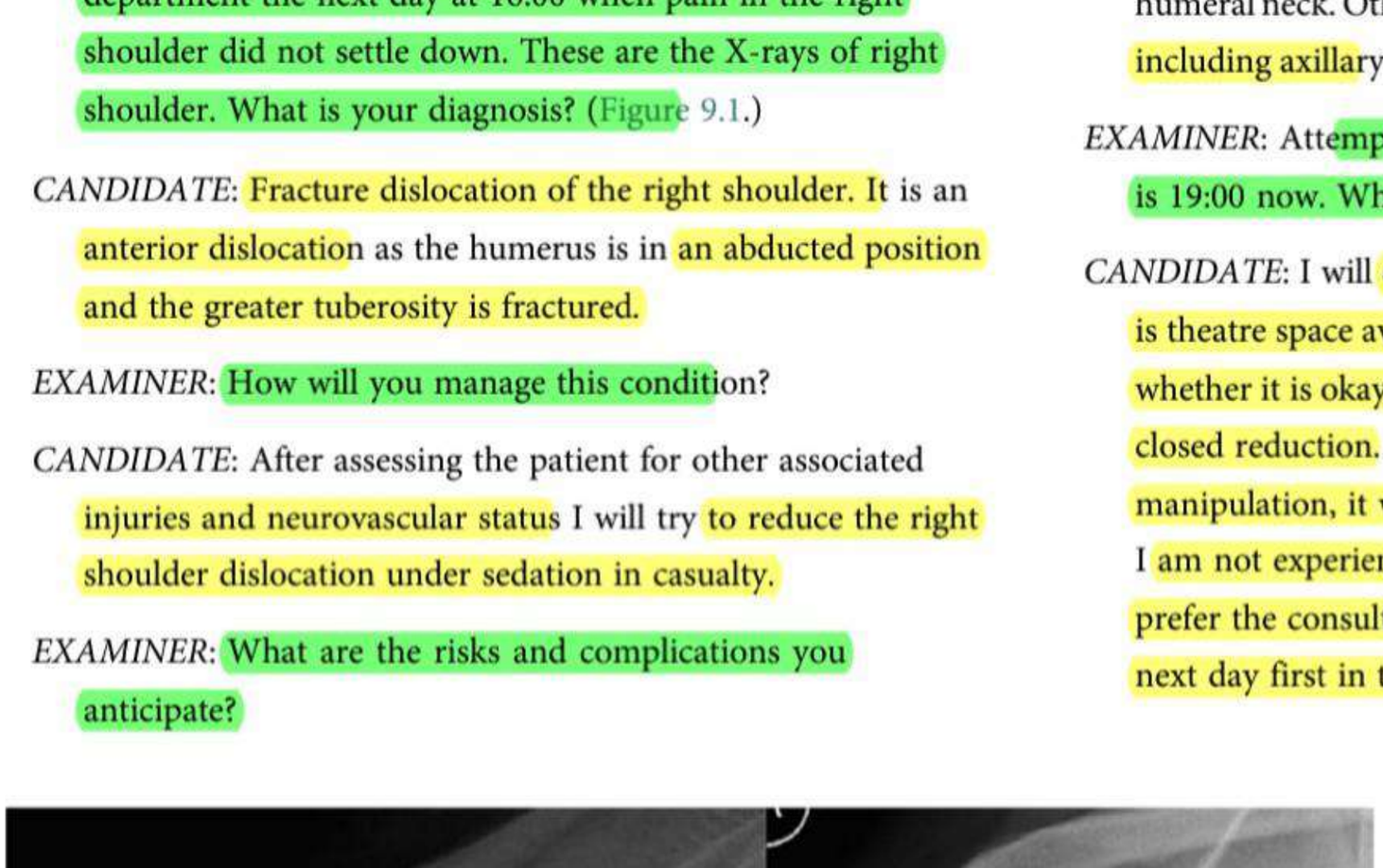


Figure 9.1 Anteroposterior (AP) radiograph right shoulder demonstrating fracture/dislocation.

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

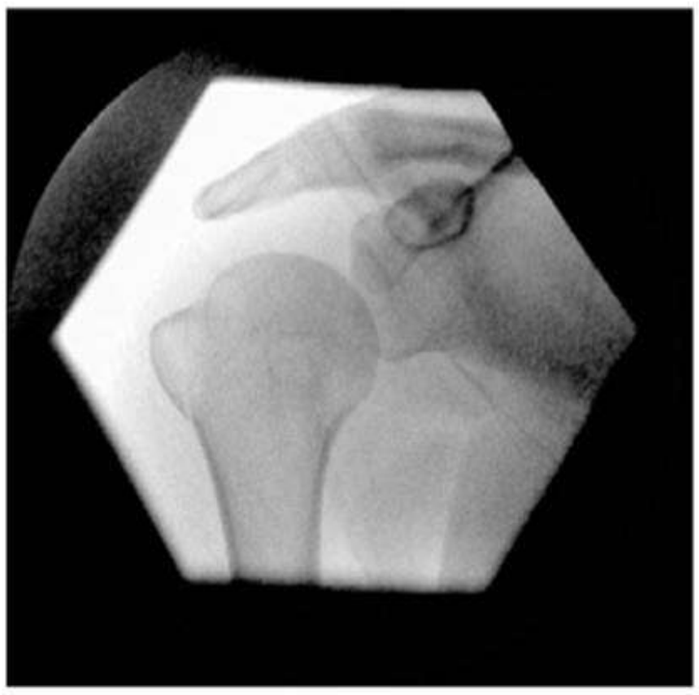


Figure 9.2 II films relocated right shoulder.

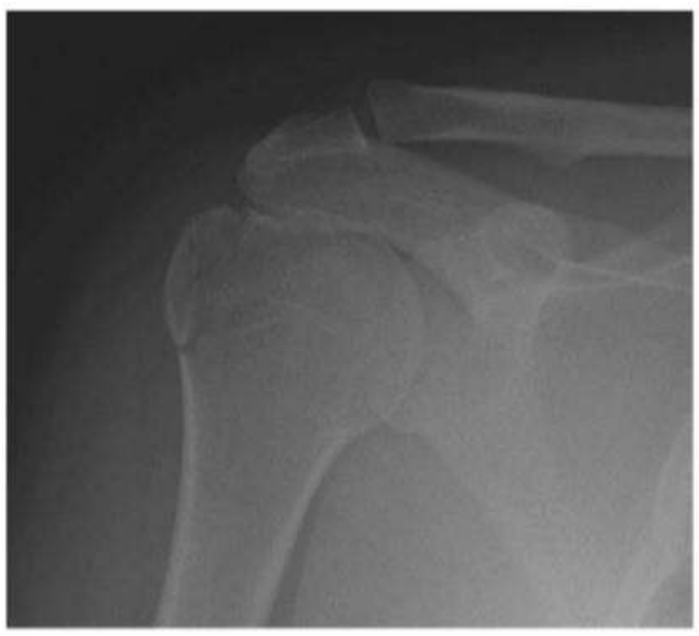


Figure 9.3 Anteroposterior (AP) radiograph right shoulder with greater tuberosity fracture.

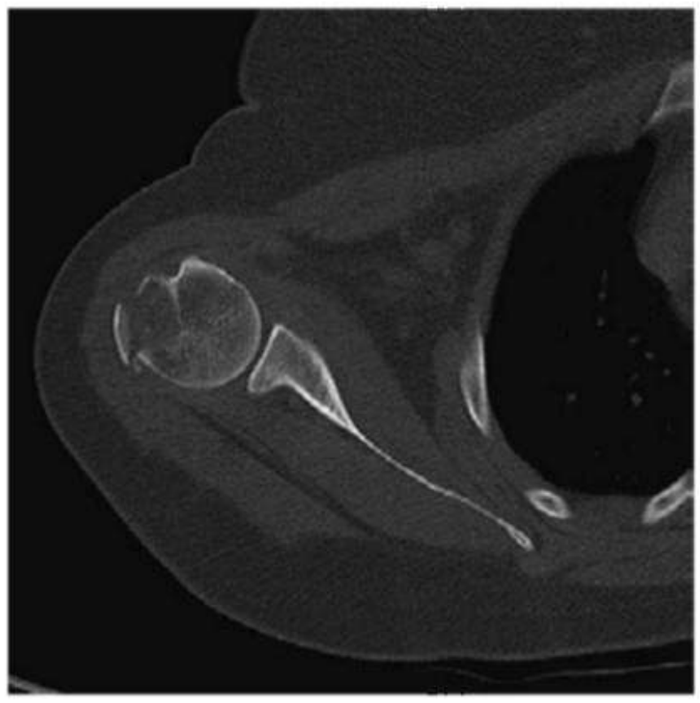


Figure 9.4 CT images right shoulder.

EXAMINER: What manoeuvre would you perform to achieve shoulder reduction?

CANDIDATE: Under complete muscle relaxation, I will manipulate the shoulder with gentle traction, external rotation and adduction with internal rotation.

EXAMINER: Next day in theatre, closed reduction is achieved. What will you do next? (Figure 9.2.)

CANDIDATE: I will assess greater tuberosity fracture reduction. If it is well reduced as the X-ray shows, I will treat it non-operatively with a poly sling for 3 weeks with serial X-rays on a weekly basis and if there is no fracture displacement, I will start shoulder mobilization under physiotherapy care.

EXAMINER: Here is the X-ray of the right shoulder 1 week later. What will you do? (Figure 9.3.)

CANDIDATE: I will reassess the patient clinically including assessing neurovascular status and organize a CT scan of the right shoulder to check for amount of displacement of greater tuberosity fragment and also to check for humeral neck fracture.

EXAMINER: CT scan of the right shoulder shows no humeral neck fracture and axial views of the greater tuberosity are shown in this scan. What will be your management strategy? (Figure 9.4.)

CANDIDATE: The greater tuberosity fragment is displaced. Hence, I would offer her the options of surgical management in the form of open reduction and fixation with screws.

EXAMINER: What are the risks of non-operative management of a displaced greater tuberosity fracture?

CANDIDATE: Non-union, malunion leading to subacromial impingement.

Structured oral examination question 2

EXAMINER: A 24-year-old man fell down the last few steps of a flight of stairs and sustained injury to his left wrist. These are his X-rays. What is this injury? (Figure 9.5.)

CANDIDATE: This is a fracture of the radial styloid that is displaced.

EXAMINER: Is this injury more than just a displaced radial styloid fracture?

CANDIDATE: Yes, it involves dorsal subluxation of the radiocarpal joint also.

EXAMINER: What injuries have occurred other than the radial styloid fracture?

CANDIDATE: Rupture of volar capsular and ligamentous structures.

EXAMINER: How will you manage this injury?

CANDIDATE: I will assess the patient's general condition, look for other injuries, check whether it is a closed or an open fracture

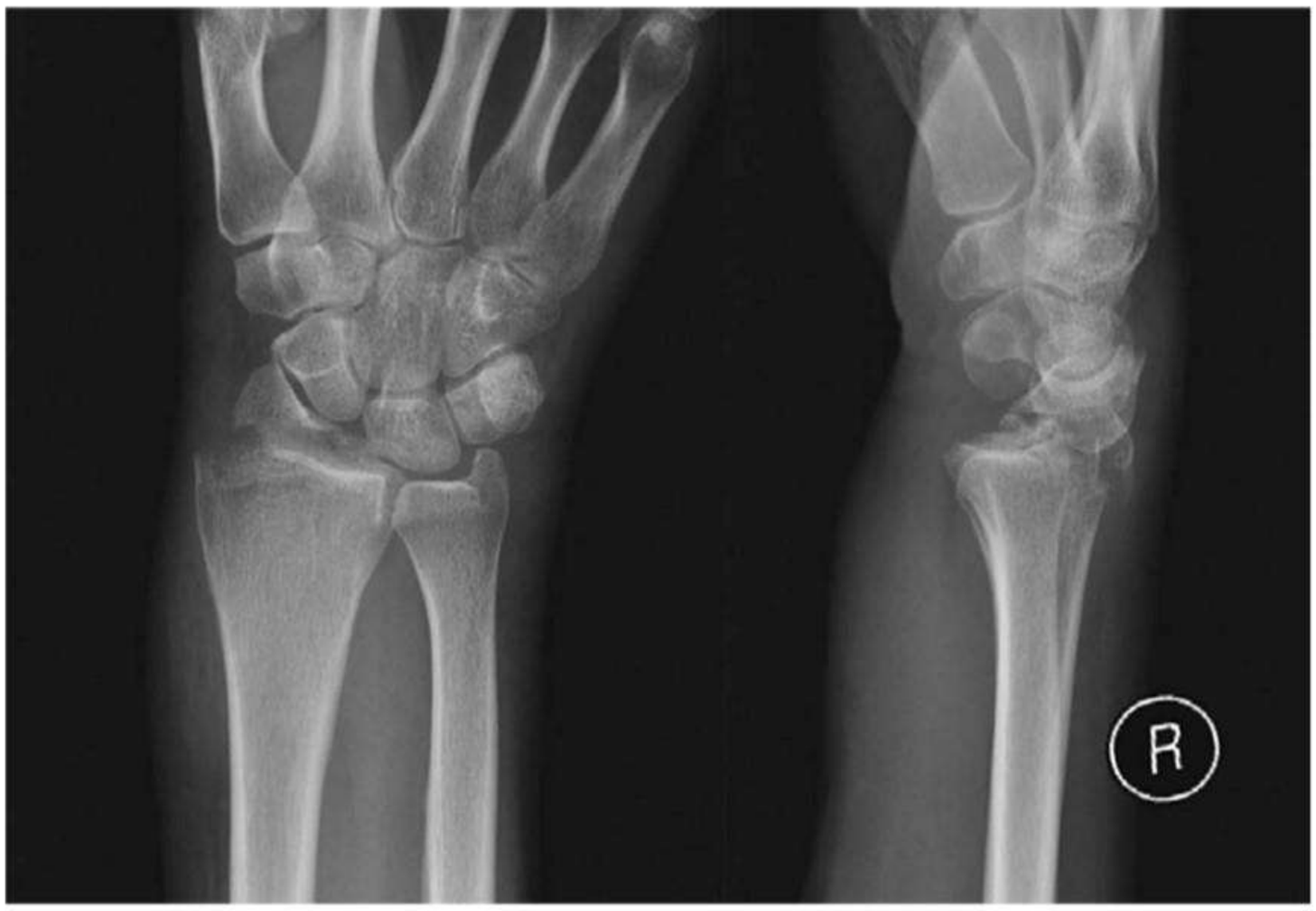


Figure 9.5 Anteroposterior (AP) and lateral radiographs left wrist.

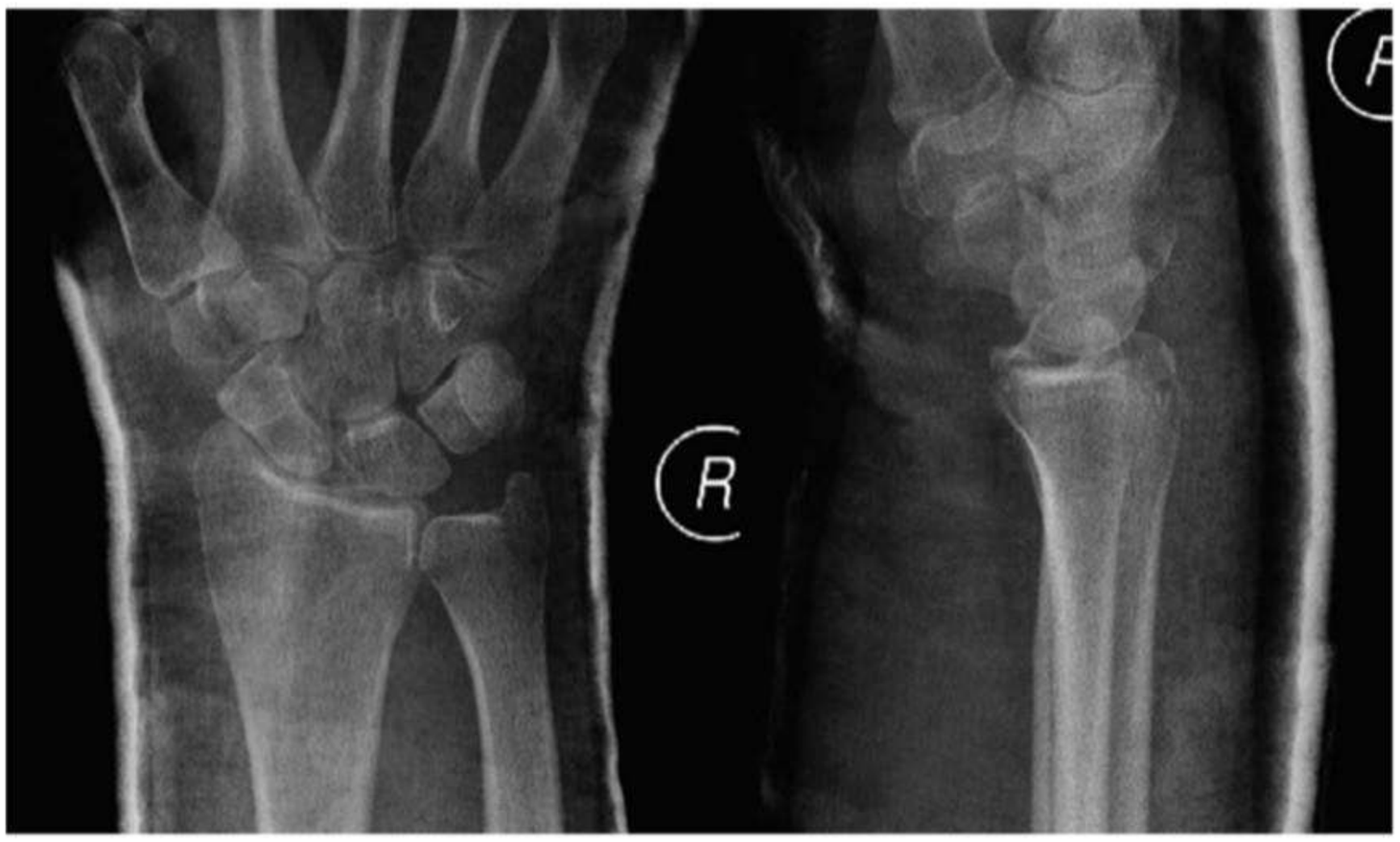


Figure 9.6 Anteroposterior (AP) and lateral post-reduction film left wrist.

and distal neurovascular status. If it is an isolated closed injury, I will attempt closed reduction under sedation in casualty, apply a below-elbow moulded dorsal plaster slab, check distal neurovascular status and get a repeat X-ray of the wrist.

EXAMINER: This is the post-reduction X-ray. How will you manage this injury? (Figure 9.6.)

CANDIDATE: Post-reduction X-rays show that the fracture is well reduced and the radiocarpal alignment is satisfactory. It is a potentially unstable injury due to damage to volar capsular and ligamentous structures. I would offer an examination under anaesthesia of the wrist and if it is unstable I will stabilize the fracture with open reduction and stabilization with screws to achieve compression at the fracture site. I will again examine the wrist for stability; if it is still unstable, I will apply a spanning external fixator with radiocarpal joint well reduced. The external fixator will be removed at 4 weeks post-surgery and I will start mobilization of the wrist with physiotherapy.

Structured oral examination question 3

EXAMINER: A motorcyclist came off his bike at around 80 miles/hour and has sustained an isolated injury to his right elbow. These are his X-rays in casualty. (Figure 9.7.)

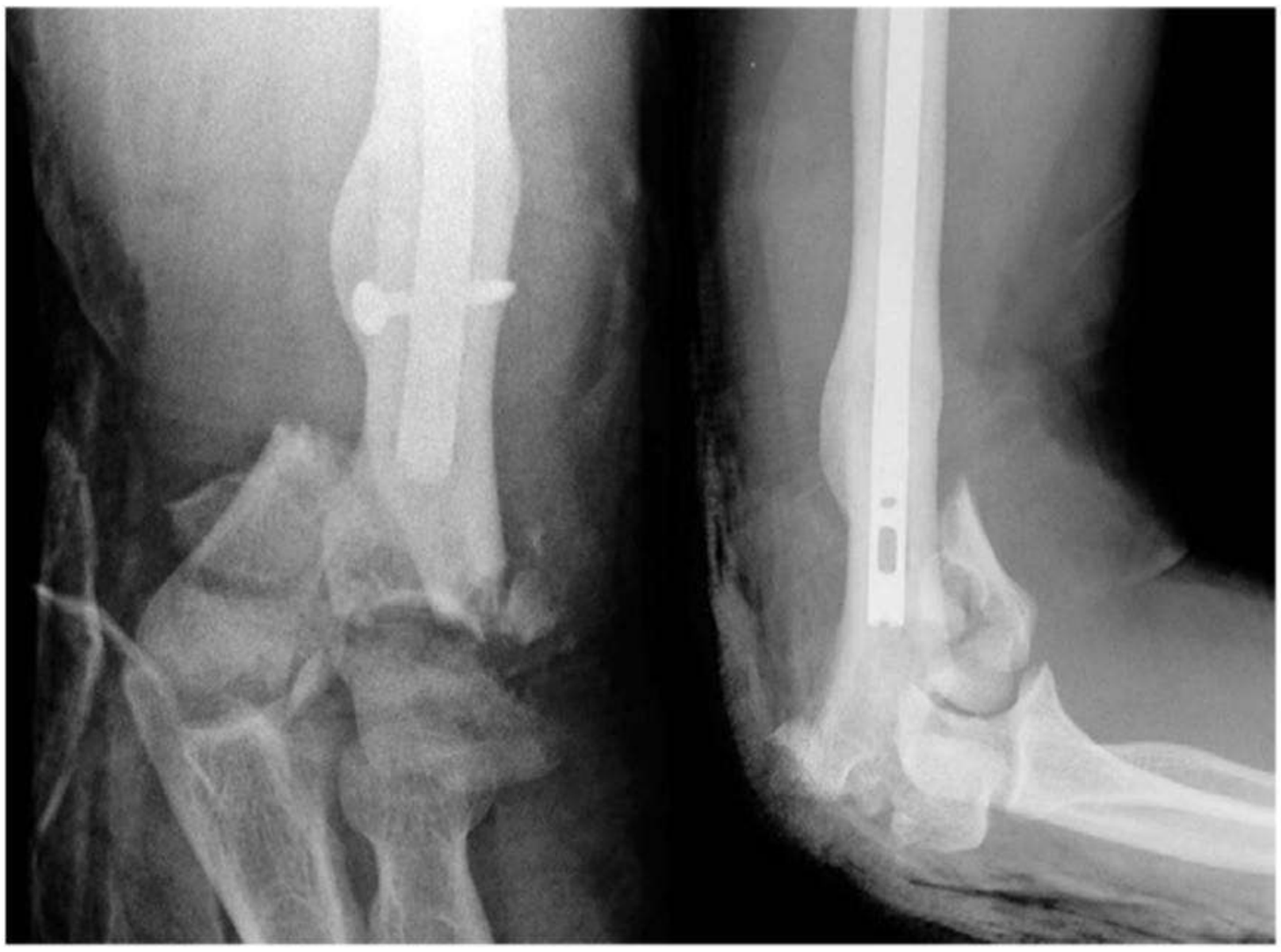


Figure 9.7 Anteroposterior (AP) and lateral radiographs right elbow demonstrating comminuted fracture.

CANDIDATE: These X-rays of the right elbow and distal humerus show a comminuted fracture of the distal humerus. It is difficult to identify the fracture fragments but it is a bicondylar fracture with some intercondylar articular fragments too. There is a retrograde humeral nail in situ. I can see some dressing around the elbow. I will assess the patient according to ATLS protocols and assess to see whether it is an isolated injury with no distal neurovascular deficits. I will also check if it is an open fracture.

EXAMINER: It is an open fracture. How will you deal with the wound in casualty?

CANDIDATE: I will assess the open wound including size, location, any skin loss, active bleeding or any exposed bone. I will remove any gross contamination, clean with normal saline, cover the wound with sterile dressing gauze and OpSite after taking photographs of the wound. I will check the patient's tetanus status and give a booster dose if required and analgesia. I will start the patient on intravenous cefuroxime which will continue until the wound is closed.

EXAMINER: What will be the definitive management and its timing?

CANDIDATE: The timing of debridement is dependent on when a thorough debridement and stabilization of fracture can be performed. It is not essential to perform debridement within 6 hours, as per BOA standards of trauma guidelines.

Principles of management will include a CT scan if possible before surgery to provide a better understanding of the fracture pattern. Wound edges will be excised and extended as appropriate, fracture ends will be debrided including removal of any devitalized soft tissue and bone fragments with no soft tissue attachments. Thorough lavage of the wound with at least 6 litres of gravity-assisted normal saline is then performed. After the debridement is completed, I will assess whether wound and fracture are suitable for definitive stabilization.

EXAMINER: If the wound is satisfactory and definitive stabilization is planned, how will you go about it?

CANDIDATE: I will not remove the retrograde humeral nail but plan stabilization around it. I will perform a posterior approach, incorporating the wound if it is posteriorly. I will perform a chevron olecranon osteotomy after pre-drilling the olecranon. This will allow me to accurately reduce the intra-articular fractures and stabilize them with lag screws. Following this I will stabilize the metaphyseal part of the fracture with medial and lateral pre-contoured plates and try to bypass the nail; if not possible, I will use locking screws in the diaphyseal segment.

EXAMINER: How will you stabilize the olecranon osteotomy?

CANDIDATE: I will use a 6.5 mm partially threaded cannulated screw or a pre-contoured plate fixation, dependent on the bone quality.

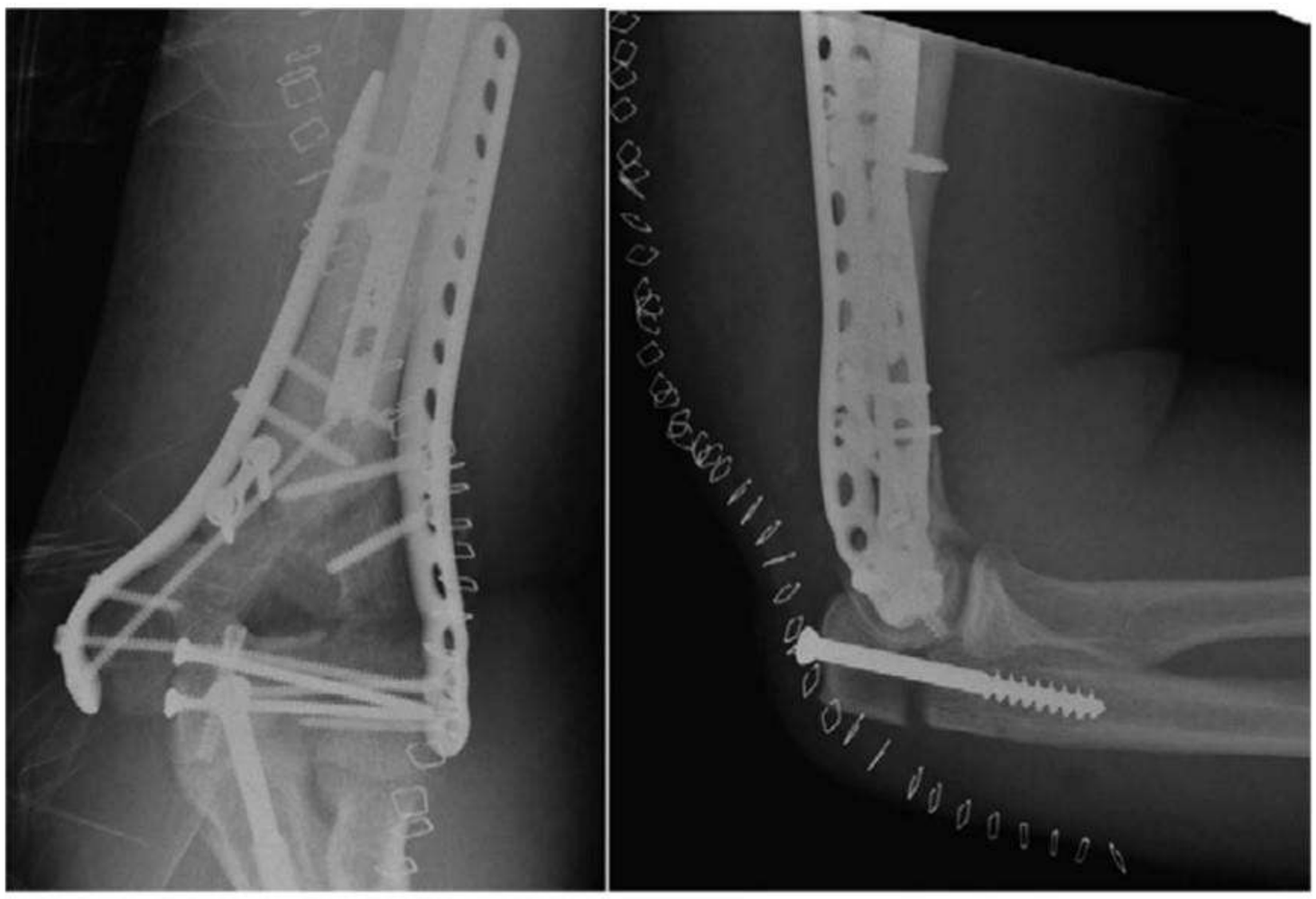


Figure 9.8 Anteroposterior (AP) and lateral radiographs right elbow post fixation.

EXAMINER: This is the postoperative X-ray. What will be your postoperative management? (Figure 9.8.)

CANDIDATE: There appears to be good intra-articular and extra-articular fracture reduction and stabilization. The olecranon osteotomy has been stabilized with a screw but there is a gap in the osteotomy.

I would still proceed with early mobilization of the elbow. If the osteotomy opens up further or does not show signs of healing I will revise the olecranon stabilization.

Structured oral examination question 4

EXAMINER: A cyclist was knocked over by a car and he landed on his elbow. This is an isolated injury. These are his X-rays. (Figure 9.9.)

CANDIDATE: This is a fracture of the proximal ulna associated with an anterior dislocation of the radial head. There is a small fragment seen next to the radial head and it is possibly from the radial head. I will check for distal neurovascular status and for any wounds around the injury.

EXAMINER: Do you know of any eponymous name and classification of this injury?

CANDIDATE: This is a Monteggia fracture dislocation.

A classification based on the direction of radial head dislocation is Bado classification. Types are anterior, posterior, lateral and associated radial fracture. This is a posterior type injury.

EXAMINER: How will you manage this?

CANDIDATE: Once I have fully assessed the patient, I will reduce the fracture under sedation in casualty and apply an above-elbow backslab. After that I will get a check X-ray to make sure the fracture dislocation is reduced.

EXAMINER: Can this fracture be treated non-operatively?

CANDIDATE: It is an unstable fracture configuration with risk of re-displacement. Hence, I will treat this fracture with surgery in the form of open reduction and plate stabilization of the ulna fracture. Most of the time, the radial dislocation will reduce and be stable.

EXAMINER: What is the cause for a radial head still subluxing after ulna fracture stabilization?

CANDIDATE: Malreduction of ulna fracture, incarceration of annular ligament, large radial head fracture.

EXAMINER: This is the postoperative X-ray. What will be your postoperative management? (Figure 9.10.)

CANDIDATE: I will start physiotherapy to mobilize the elbow as tolerated to prevent stiffness and follow up the patient to make sure the wound and fracture have healed along with good functional outcome.

Structured oral examination question 5

EXAMINER: A 23-year-old while on a night out fell on to his left hand and has come to casualty with pain and deformity. This is an X-ray of the left distal forearm (Figure 9.11).

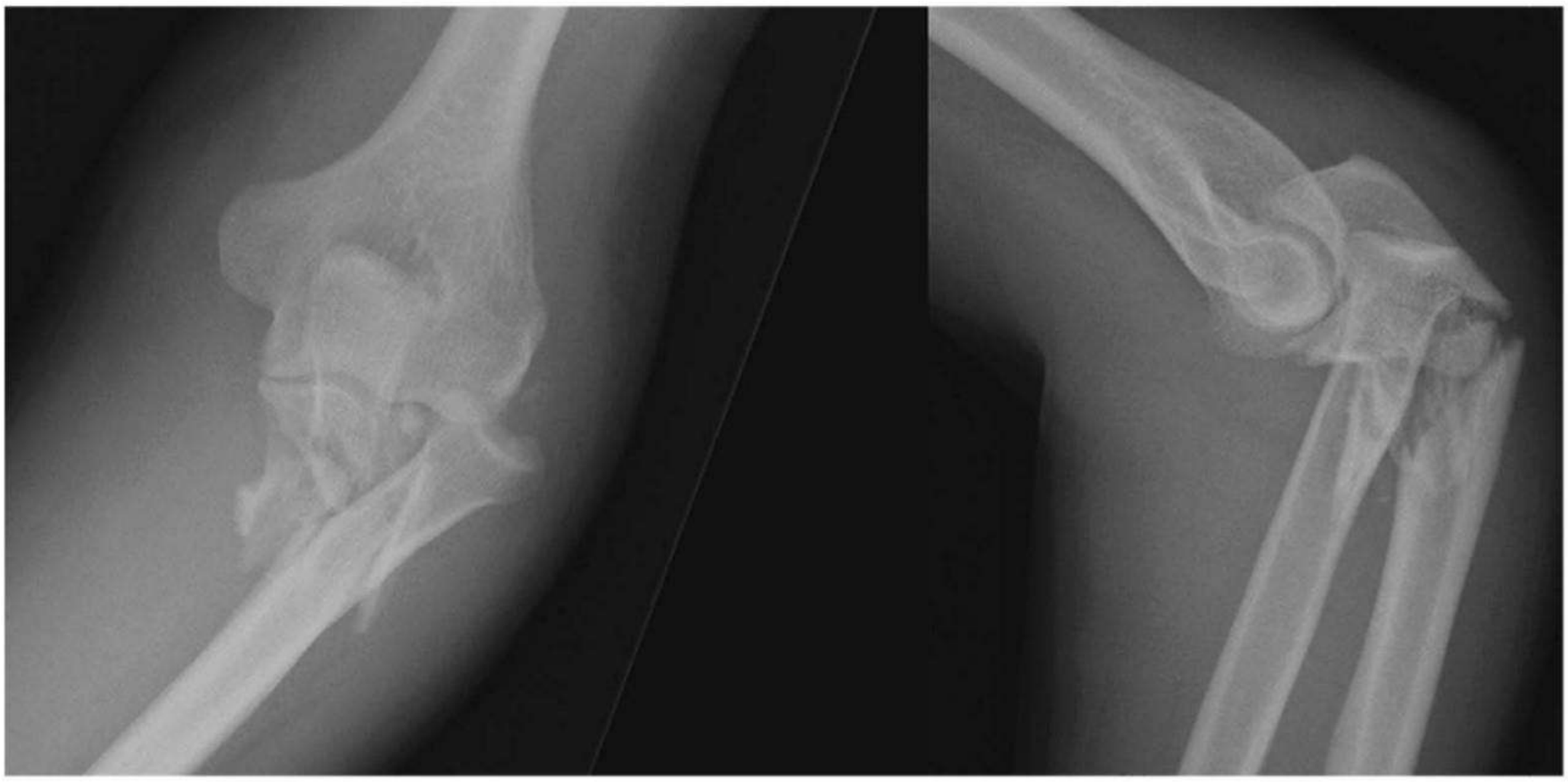


Figure 9.9 Anteroposterior (AP) and lateral radiographs Monteggia fracture/dislocation right elbow.

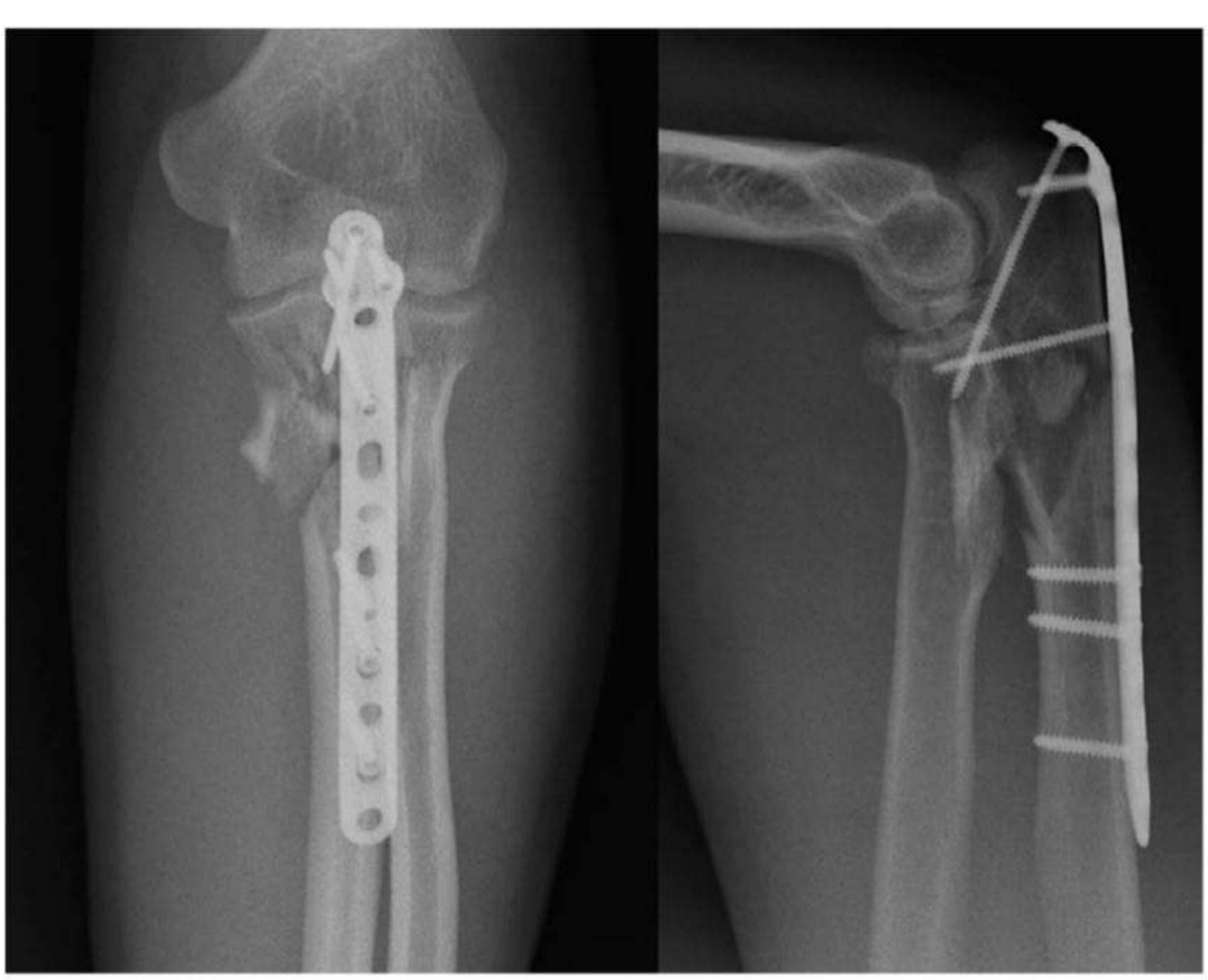


Figure 9.10 Anteroposterior (AP) and lateral radiographs right elbow post-fixation.

CANDIDATE: This is a fracture of radial shaft associated with dislocation of distal radioulnar joint. I will assess the patient with regards to medical conditions, associated injuries, distal neurovascular status and whether it is a closed or open injury.

EXAMINER: This is an isolated closed injury with no distal problems. How will you manage this injury?

CANDIDATE: I will try to reduce the fracture dislocation in casualty under sedation, apply an above-elbow backslab and get an X-ray of the forearm and wrist.

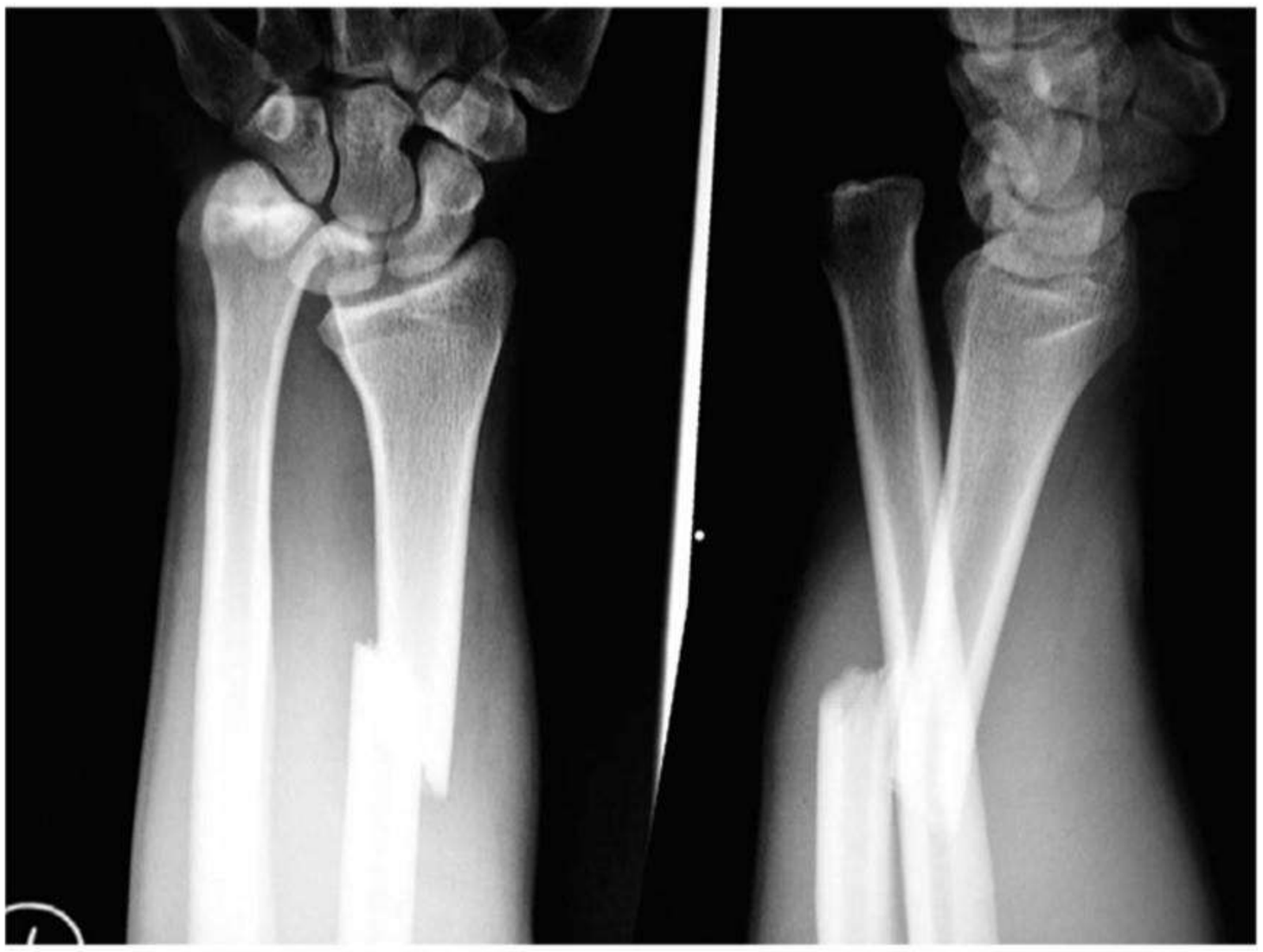


Figure 9.11 Anteroposterior (AP) and lateral radiographs left forearm.

EXAMINER: Check X-ray shows no change in position, the time is now 21:00. What will you do?

CANDIDATE: If there are no signs of any neurovascular deficit, I will prioritize the patient in the next day's trauma list for open reduction and stabilization of radial fracture with stabilization of distal radioulnar joint, if required. Overnight, the arm will be kept elevated and frequent neurovascular assessment will be made.

EXAMINER: During surgery the radius fracture is stabilized but the distal radioulnar joint is still dislocated. What are the causes for this?

CANDIDATE: Malreduction of radius fracture or soft tissue interposition in the distal radioulnar joint or there is significant soft tissue disruption of the joint.

EXAMINER: Radius fracture reduction is satisfactory and there is no interposition but the joint is dislocated. How will you deal with it?

CANDIDATE: I will check if the joint reduces with supination. If the joint reduces, then I will stabilize it with two 2.0 mm K-wires percutaneously.

EXAMINER: What will be your postoperative protocol?

CANDIDATE: I will immobilize the forearm in a below-elbow backslab with wrist in supination. I will convert it to a below-elbow cast with wrist in supination for 4 weeks followed by removal of wires in clinic and start mobilization of wrist under physiotherapy care.

Structured oral examination question 6

EXAMINER: A 58-year-old man sustained an injury to his arm when he fell from standing height. He is right handed, suffers from hypertension and has a sedentary lifestyle. These are his X-rays. (Figure 9.12.)

CANDIDATE: The X-rays show a transverse fracture of the right humerus shaft in the middle third. There is 100% translation and minimal angulation in lateral view and 80% translation in anteroposterior view.

I will check for other injuries, neurovascular status and whether it is a closed or open fracture.

EXAMINER: It is a closed fracture with no associated problems. How will you manage it?

CANDIDATE: In casualty, I will apply a U slab, then check for distal neurovascular status and get a check X-ray.

If there are no further problems and X-ray is satisfactory, then I will bring the patient to the fracture clinic in a few days to convert the U slab to a humeral brace.

EXAMINER: What will you do once the humeral brace is applied?

CANDIDATE: I will get a check X-ray to be sure that the fracture is not displacing. I will also see the patient on a weekly basis for first 3 weeks with X-rays to assess for fracture position.

EXAMINER: This is week 2 X-ray. What will you do? (Figure 9.13.)

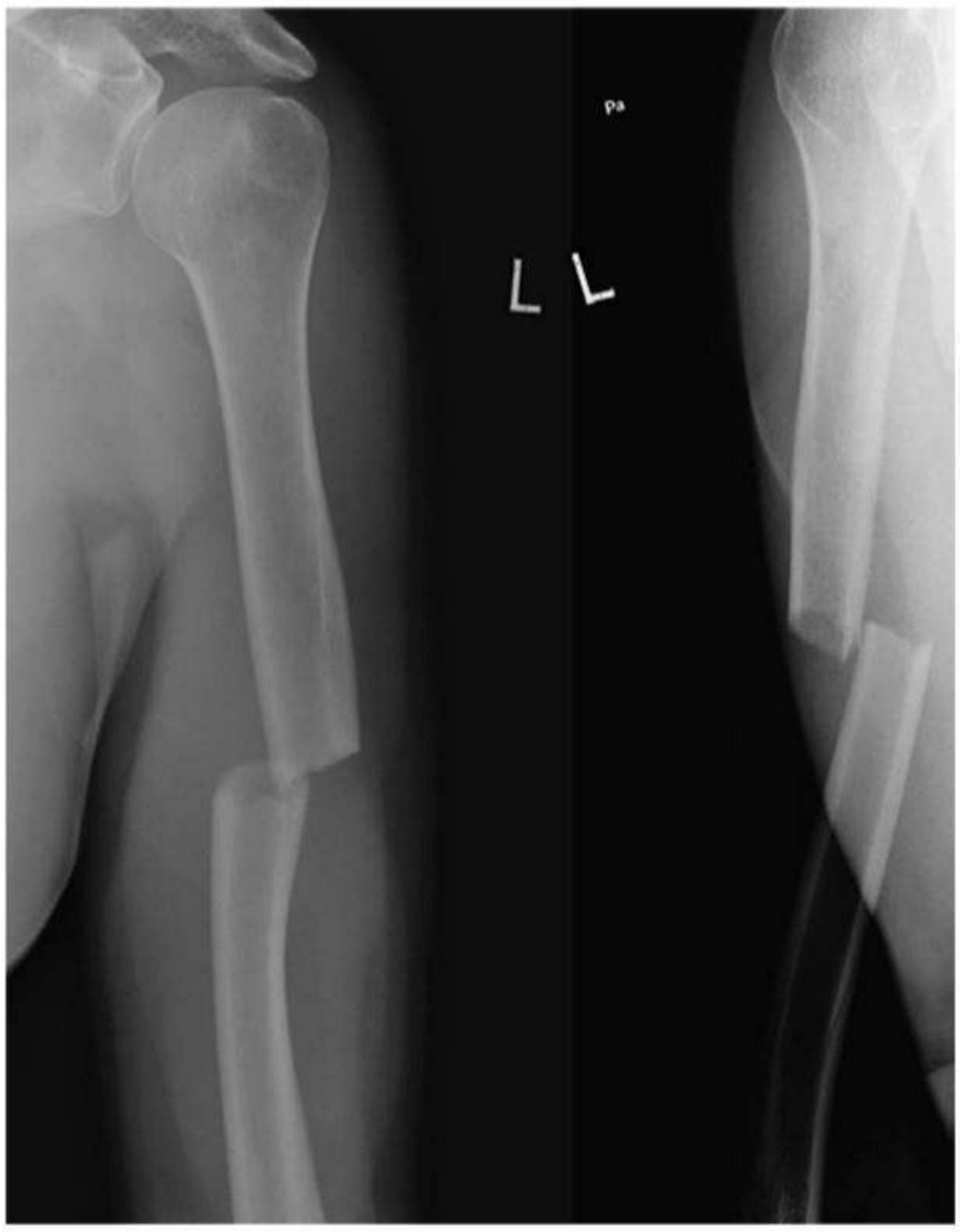


Figure 9.12 Anteroposterior (AP) and lateral radiographs transverse fractured right humerus.

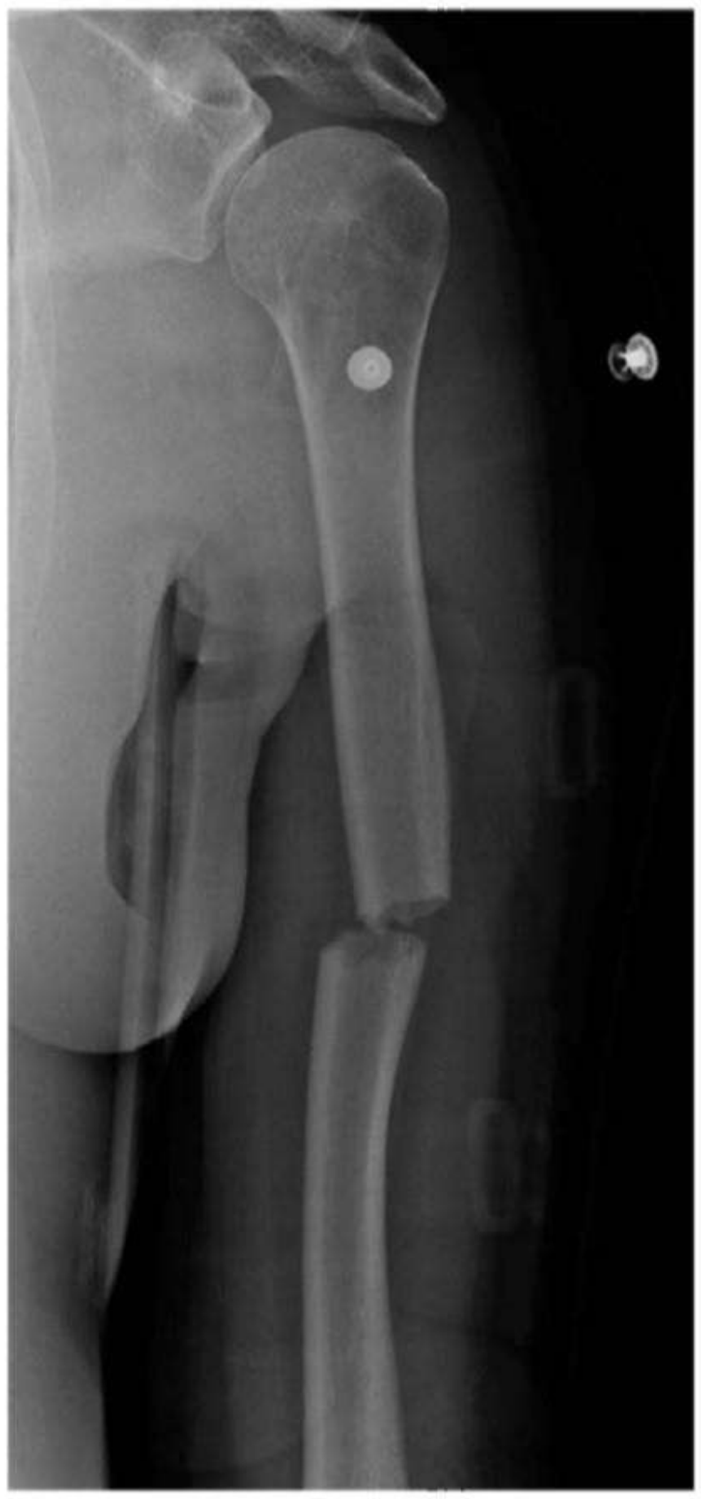


Figure 9.13 Anteroposterior (AP) radiograph distracted right humerus fracture.

CANDIDATE: There is some distraction at the fracture site. This could be an indication of possible non-union in the future. I will discuss this with the patient and continue with non-operative management as planned with adjustment of the humeral brace. However, I will reiterate the possibility of non-union.

EXAMINER: Patient does not want to wait and see. He is keen to have the operation. What will you do?

CANDIDATE: I will discuss with the patient the advantages and risks involved in operative management of humeral fractures, including infection, neurovascular deficit, stiffness, non-union, malunion, implant failure, complex regional pain syndrome.

EXAMINER: What operative intervention will you undertake?

CANDIDATE: The surgical options are intramedullary nail fixation or plate fixation. I prefer plate fixation to avoid the risk of shoulder subacromial symptoms. For this fracture, I will do a plate fixation via an anterior approach.

EXAMINER: This is the X-ray at 3 months. What will you do? (Figure 9.14.)

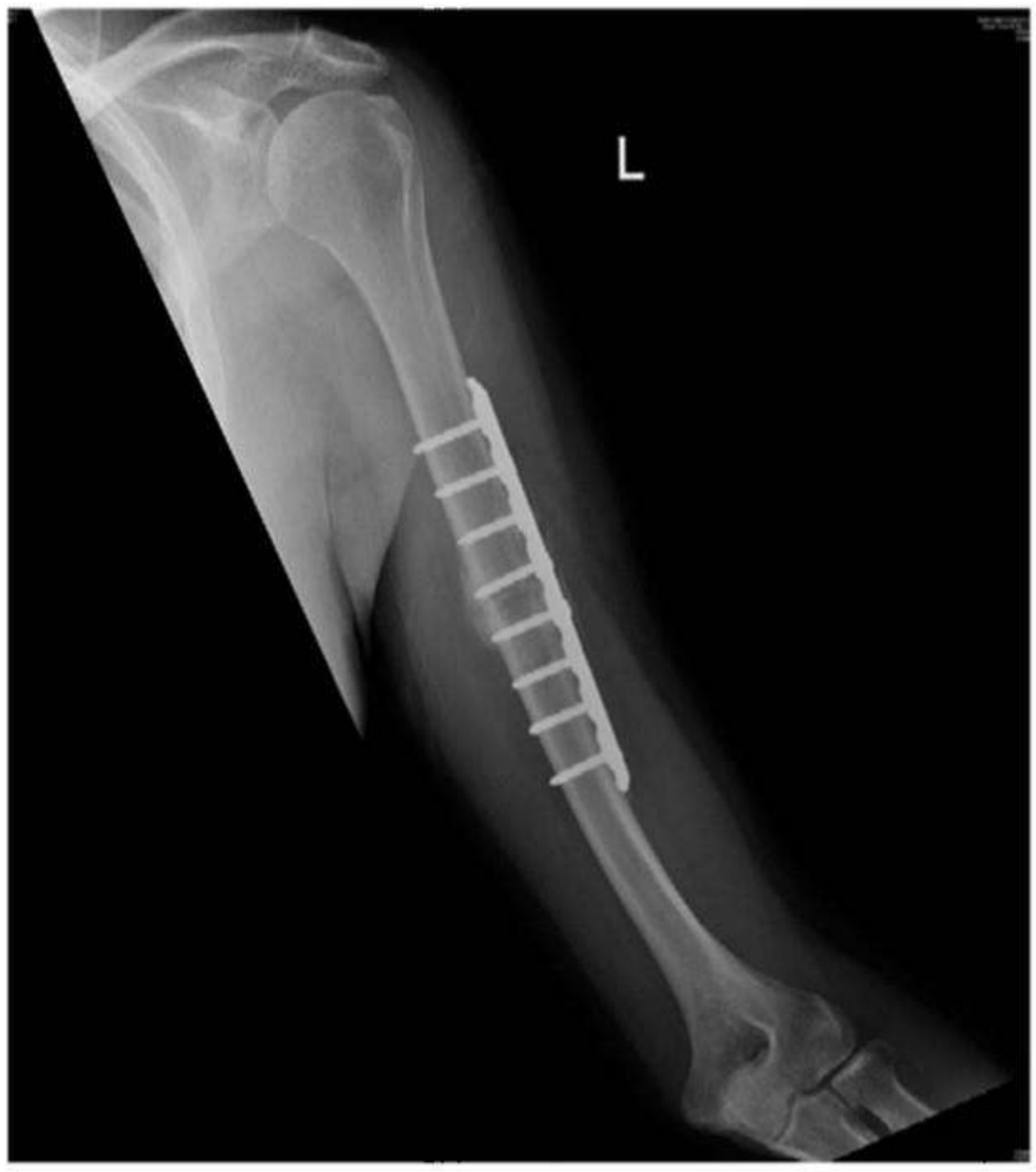


Figure 9.14 Anteroposterior (AP) radiograph non-union right humerus fracture post-plate fixation.

Section 3: Trauma

CANDIDATE: My first aim will be to rule out infection. I will check patient for systemic illnesses, like fever, chills, shivering, loss of appetite/weight. I will also perform blood tests – FBC, C-reactive protein.

EXAMINER: Patient has no symptoms and is happy with progress with physiotherapy. Why do you suspect infection?

CANDIDATE: In a plate fixation, absolute stability is the aim. This means that the fracture will heal by primary intention. In the presence of callus formation, I will suspect infection or aseptic implant loosening.

This last question is about primary healing in rigid/stiff fixation. When there is callus formation in these 'rigid fixations' especially of transverse or oblique fractures then the possibility of either early plate loosening or grumbling infection should be kept in mind. Though external callus can occur in plate fixations, in these circumstances the stiffness of the construct is lower and is flexible enough to allow secondary fracture healing as the working length is longer.

A perfectly plated Swiss fracture does not go through endochondral repair.

If there is a plateau in recovery with MRI-proven cord compression, or if there are signs of instability, surgical decompression and stabilization should be considered.

EXAMINER: Are you aware of any other incomplete cord syndromes?

CANDIDATE: Anterior cord syndrome, typically caused by a flexion/compression injury, affects the anterior two-thirds of the spinal cord via anterior spinal artery lesions. In this case the lower extremity is affected to a greater extent than the upper extremity but it is useful to note that proprioception and vibratory sense (both carried in the dorsal, unaffected, columns) are preserved. This condition has the worst prognosis.

Brown–Séquard syndrome is a hemitransection of the spinal cord, seen with a penetrating trauma. There is ipsilateral loss of motor function, proprioception and vibratory sense; there is contralateral loss of pain and temperature sensation. This has the best prognosis of the incomplete injuries with 99% of patients ambulatory at final follow up.

Finally a rare syndrome, characterized by a loss of proprioception, is the posterior cord syndrome. Motor, light touch and pain and temperature are all preserved.

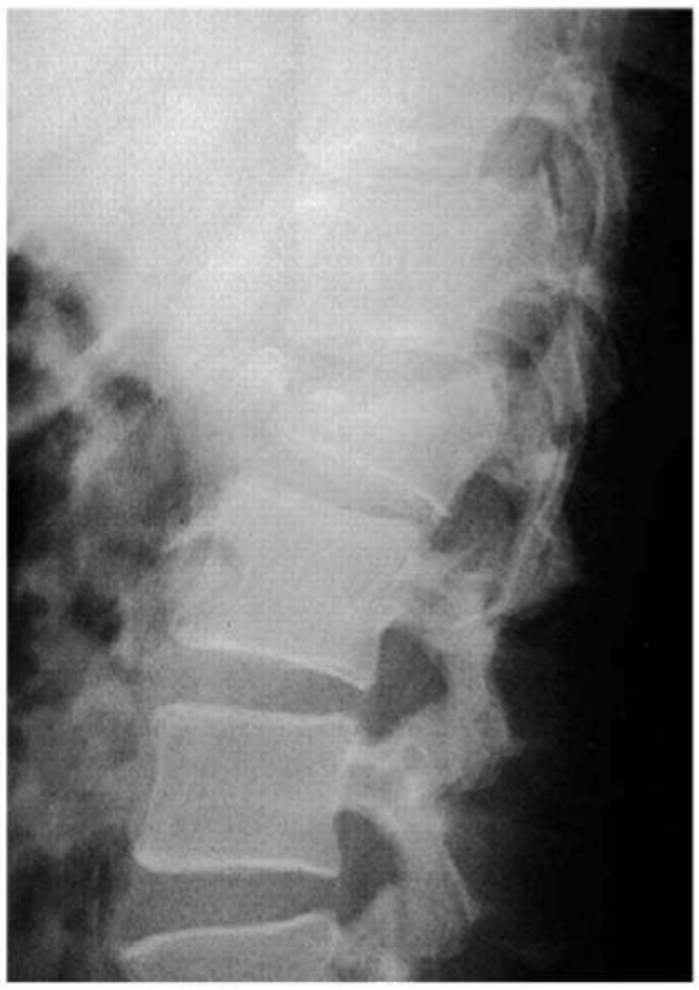


Figure 10.14
Lateral radiograph burst thoracolumbar fracture.

Structured oral examination question 3: Thoracolumbar burst fractures

EXAMINER: What does this X-ray show? (Figure 10.14.)

CANDIDATE: There is a fracture of the T12 vertebral body with greater than 50% loss of the vertebral body height. The posterior margin is poorly defined. On the AP view there is widening of the interpedicular distance.

EXAMINER: Is this a stable or unstable injury?

CANDIDATE: This is an unstable injury. Two of Denis' three columns have failed, and there is greater than 50% loss of vertebral body height.

EXAMINER: What is your management?

CANDIDATE: I would manage the patient according to ATLS guidelines and ensure that the spine is kept in alignment at all times. My aims are to exclude any other spinal trauma, concurrent abdominal trauma, and to prevent secondary injury.

A full neurological examination is undertaken and the presence of sacral sparing documented (which is suggestive of a better prognosis). The neurological examination is repeated frequently in order to ascertain whether or not there is a progressive neurological deficit.

I would assess the patient for signs of spinal shock, which is a temporary loss of spinal cord function and reflex activity below the level of the injury. It is typically characterized by diaphragmatic breathing (if level appropriate), paralysis, absent reflexes, erection, urinary retention and an absent bulbocavernosus reflex.

EXAMINER: What is the importance of spinal shock and how do you know when it's over?

CANDIDATE: The importance of spinal shock is that one cannot evaluate the neurological deficit until the spinal shock phase has resolved. Resolution is determined by the return of the bulbocavernosus reflex – squeezing the glans penis (or clitoris in the female) or gently pulling on an indwelling urinary catheter elicits an anal sphincter contraction.

EXAMINER: What is neurogenic shock?

CANDIDATE: Neurogenic shock can be thought of as a temporary generalized sympathectomy. Typically the patient will be hypotensive but bradycardic. It is important to exclude other causes of hypotension however (10–15% of patients with spinal injuries have visceral injuries) before attributing hypotension to neurogenic shock.

EXAMINER: The patient is no longer in spinal shock, and has some flickers of movement in his left lower limb only. What is your management now?

CANDIDATE: The patient has an incomplete injury. Given the fact that he also has an unstable spine, as determined by the loss of greater than 50% of vertebral body height (and a kyphosis angle greater than 30°), surgical decompression and stabilization is indicated.

EXAMINER: What if they were to have a complete injury? Is there a role for surgery then?

CANDIDATE: The role of surgery in a complete spinal cord injury is to facilitate rehabilitation by providing a stable and pain-free spine.

EXAMINER: What complications do you expect in the next few days?

CANDIDATE: There are many potential complications, all of which may be anticipated. Proper bedding will avoid any pressure sores as will regular turning (once the spine has been stabilized). Prophylaxis for thromboembolic disease must be provided and strict hygiene undertaken to prevent urosepsis. Paralytic ileus may require the patient to be kept nil-by-mouth and they may require a nasogastric tube to decompress. Prophylaxis should also be provided for stress ulceration (Curling's ulcer).

EXAMINER: What about the bowel?

CANDIDATE: With an upper motor neurone lesion (typically T12 and above), reflex activity is maintained and the bowel will contract and empty. A lower motor neurone lesion (typically at L1 or below) will have return of peristalsis but without the support of the spinal reflex. This leads to faecal retention. I would manage these patients with daily glycerine suppositories, and when bowel sounds return I would prescribe senna and lactulose.

British Orthopaedic Association. *The Initial Care and Transfer of Patients with Spinal Cord Injuries*. London: British Orthopaedic Association, 2006.

Structured oral examination question 4: Odontoid peg fractures

EXAMINER: What does this X-ray show? (Figure 10.15.)

CANDIDATE: This is a lateral radiograph of the cervical spine. The most obvious abnormality is a fracture through the base of the odontoid peg of C2.

EXAMINER: Yes, are you aware of any classification systems for this type of injury?

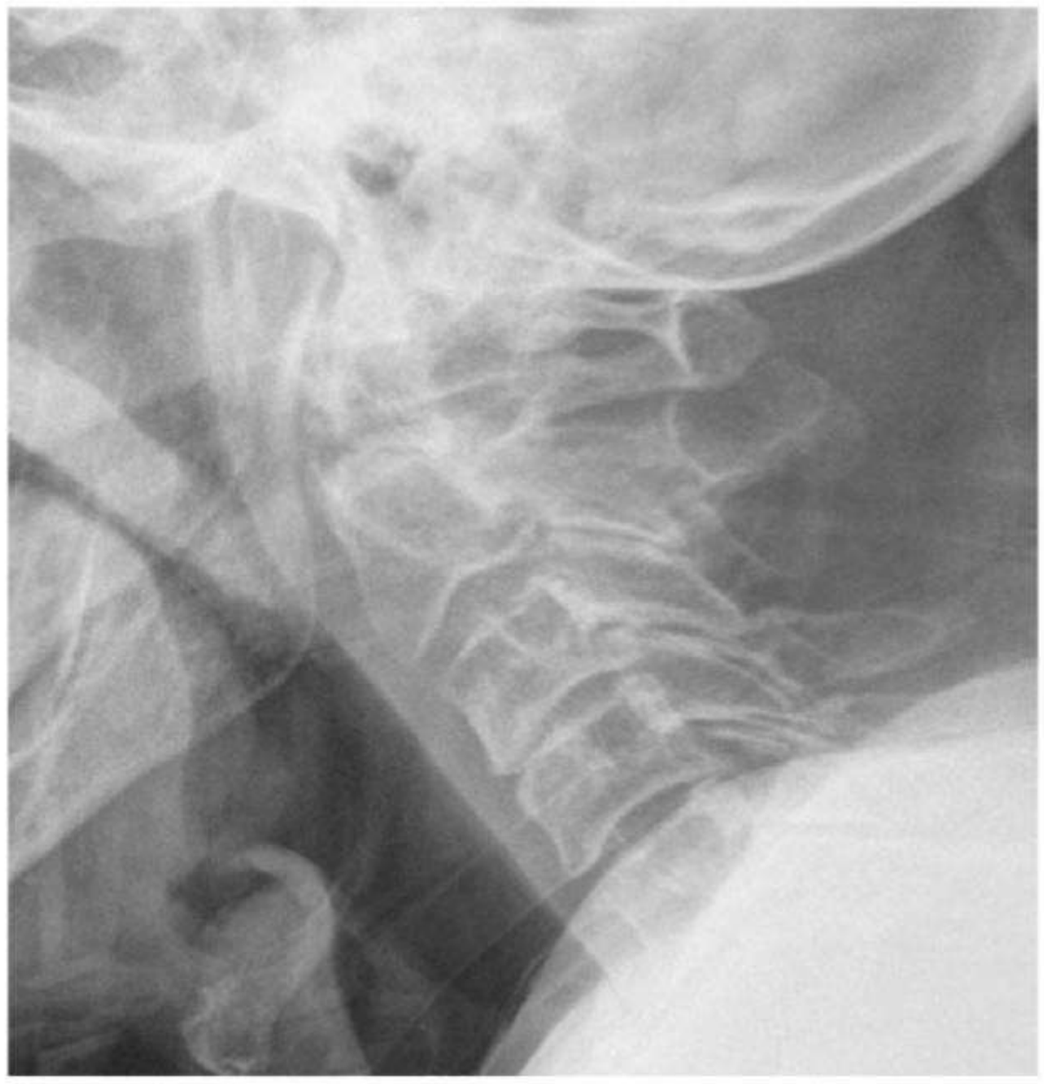


Figure 10.15 Lateral cervical spine radiograph demonstrating an odontoid peg fracture.

CANDIDATE: I am familiar with the Anderson and D'Alonzo classification. This classifies fractures according to their location within the peg. Type I fractures affect the tip of the odontoid and are caused by avulsion of the alar ligaments proximal to the transverse ligament and are usually stable injuries. Type II injuries run through the base of the odontoid peg just below the transverse ligament making them unstable injuries. They have a high ratio of cortical to cancellous bone and so have a higher rate of non-union than other fractures. Type III injuries are seen distal to the base of the peg running through the metaphyseal bone of the vertebral body. As these fractures have a higher proportion of cancellous to cortical bone and a greater surface area they are more likely to heal than Type II injuries.

EXAMINER: So how does this classification guide your management?

CANDIDATE: Type I injuries are stable and so I would treat them in a hard collar such as an Aspen collar. Type III injuries are likely to heal and so I would immobilize them in a halo jacket or possibly in a hard collar in elderly frail individuals due to the risks of halo jackets in the elderly. These injuries would then require follow-up in clinic. Type II injuries are more likely to go on to non-union and so they are the injuries that I would consider fixation as an option as opposed to a halo jacket.

Structured oral examination question 6: Chance fractures

EXAMINER: What does this picture show? (Figure 10.17.)

CANDIDATE: This is a clinical photograph of a man's abdomen. There is gross bruising across the anterior abdominal wall.

EXAMINER: What do you think has happened to him?

CANDIDATE: I would imagine that he has been involved in a road traffic accident restrained by a seat belt.

EXAMINER: Yes, that's right. So how would you assess this man in the emergency department?

CANDIDATE: I would follow ATLS principles and identify and treat any life- or limb-threatening injuries.

EXAMINER: [interrupting] Yes alright, but what injuries would you expect to find?

CANDIDATE: I would expect this man to have intra-abdominal visceral injuries due to the blunt trauma. With this mechanism I would suspect a chance-type injury which has a 50–60% association with intra-abdominal injuries.[1] I also would want to exclude cervical spine injuries with any flexion–distraction type of injury.

EXAMINER: Tell us about chance-type injuries. (Figure 10.18.)

CANDIDATE: These are flexion–distraction injuries affecting the thoracolumbar spine. It involves an injury to both the anterior and posterior columns (B type injury according to the AO classification system), or all three columns (according to the Denis classification system) making it an unstable injury. It can be purely bony, purely ligamentous or mixed. Typically, the anterior column fails in compression whereas the posterior columns fail in tension.

EXAMINER: So how would you investigate this person in the emergency department?

CANDIDATE: I would request a CT scan of the whole spine to look for any bony injuries.

EXAMINER: What would you expect to see?

CANDIDATE: If there was a bony injury then you could see the pattern of the fracture running through the vertebral body and the posterior elements. If it was a ligamentous injury then there may be some translation of the vertebral bodies at that level and there may be a widening of the interspinous space.

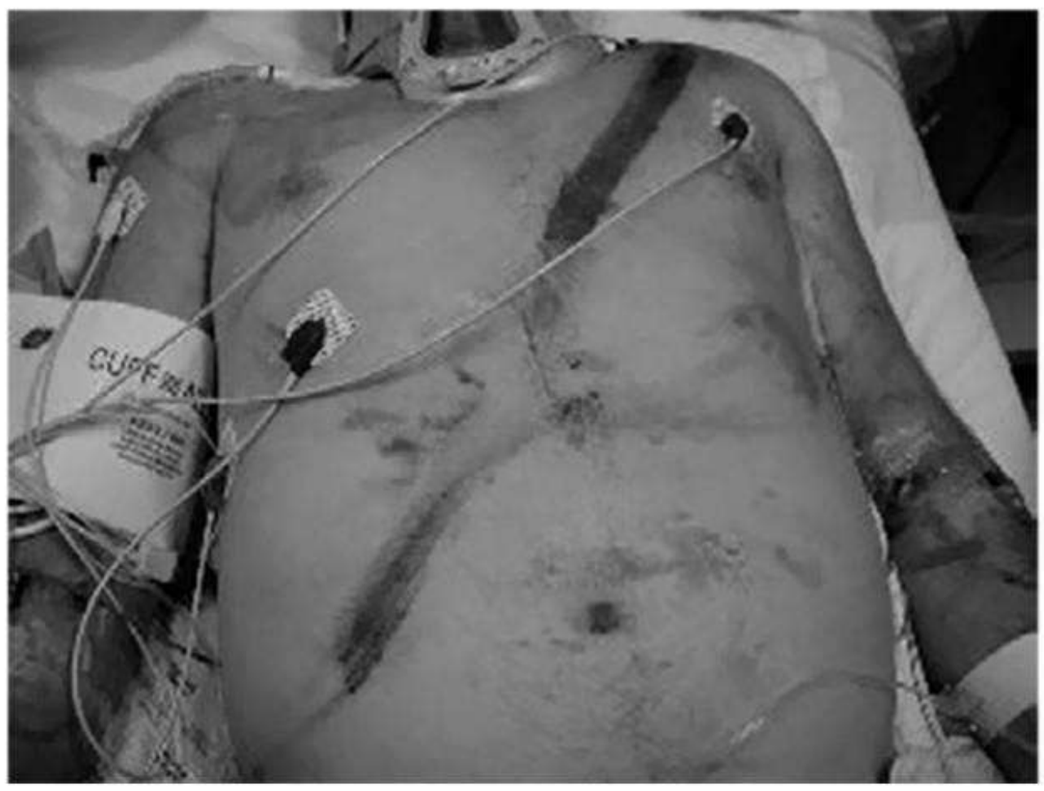


Figure 10.17 Clinical photograph demonstrating gross abdominal bruising. See colour plate section.

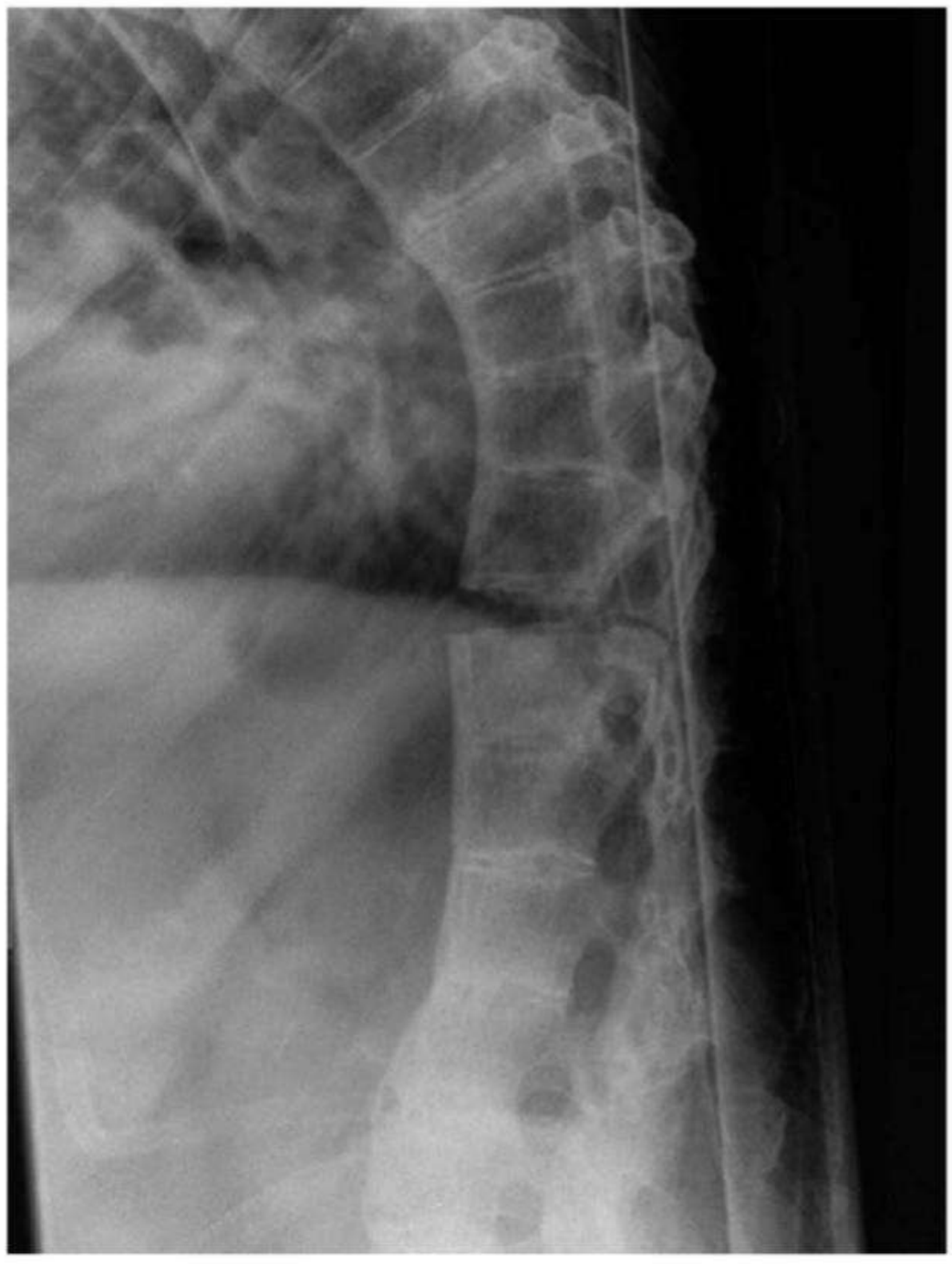


Figure 10.18 Lateral radiograph thoracic spine demonstrating Chance fracture.

EXAMINER: Would you do any further investigations?

CANDIDATE: If there was no bony injury and I suspected a chance-type injury then I would also request an MRI scan to assess the soft tissues.

EXAMINER: How would you manage these injuries?

CANDIDATE: If the patient was neurologically intact and there was minimal displacement of a bony injury it could potentially be managed in a hyperextension brace. This would require very close monitoring to ensure that there is no displacement. If there was any loss of position with this regime then it should be treated operatively. If it is a ligamentous injury then it will require fixation as it will not heal as effectively as a bony injury and will remain unstable. If there is a very displaced fracture that cannot be reduced or held effectively in a brace then they should have surgical stabilization.

EXAMINER: Yes, let's move on.

1. Anderson PA, Rivara FP, Maier RV, Drake C. The epidemiology of seatbelt-associated injuries. *J Trauma* 1991;**31**:60–67.

Structured oral examination question 7: Application of a halo

EXAMINER: How do you apply a halo? (Figure 10.19.)

CANDIDATE: I would first explain to the patient what I am going to do and why.

I would match the skull to the appropriate halo ring prior to application. Typically, three people are required to apply a halo with one person maintaining alignment and the remaining two applying the halo.[1]

Using local anaesthetic and antiseptic, four pins are applied to the adult skull (eight in the paediatric population) and tightened with a torque-limiter (8 inch-pounds; 2–4 inch-pounds in the paediatric skull). The pins are placed equidistant and symmetrically in order to allow for stability of the construct.

CARE SHOULD BE TAKEN TO PREVENT DAMAGE TO IMPORTANT STRUCTURES: the superficial temporal artery and vein, the supraorbital nerves and the sinuses.

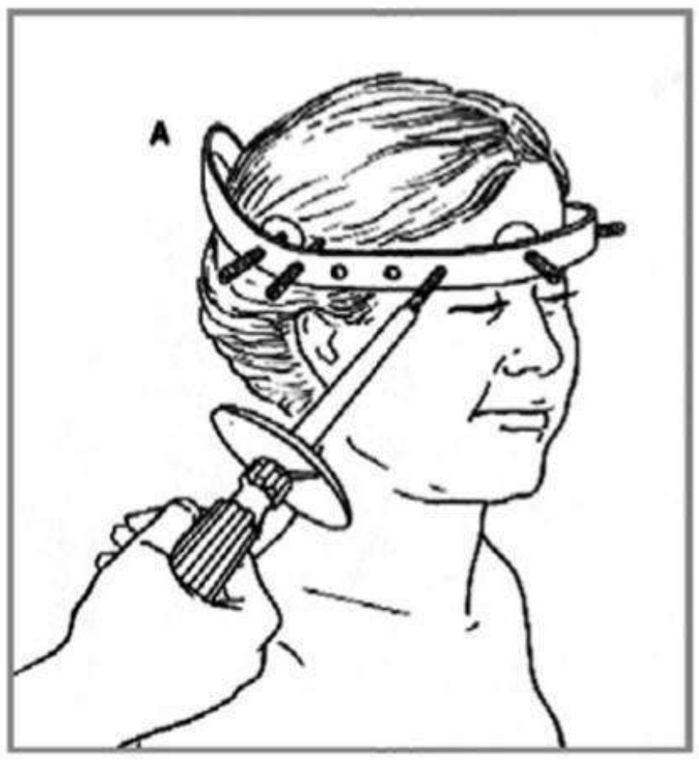


Figure 10.19
Halo traction.

The anterior pins are placed 1 cm above the lateral one-third of the eyebrow *with the eyes tightly closed*. This is lateral to the supraorbital nerve.

The posterior pins are placed behind the earlobe, just above the mastoid.

An appropriately sized jacket is then applied (or traction as may be necessary).

A radiograph is obtained to ensure correct reduction.

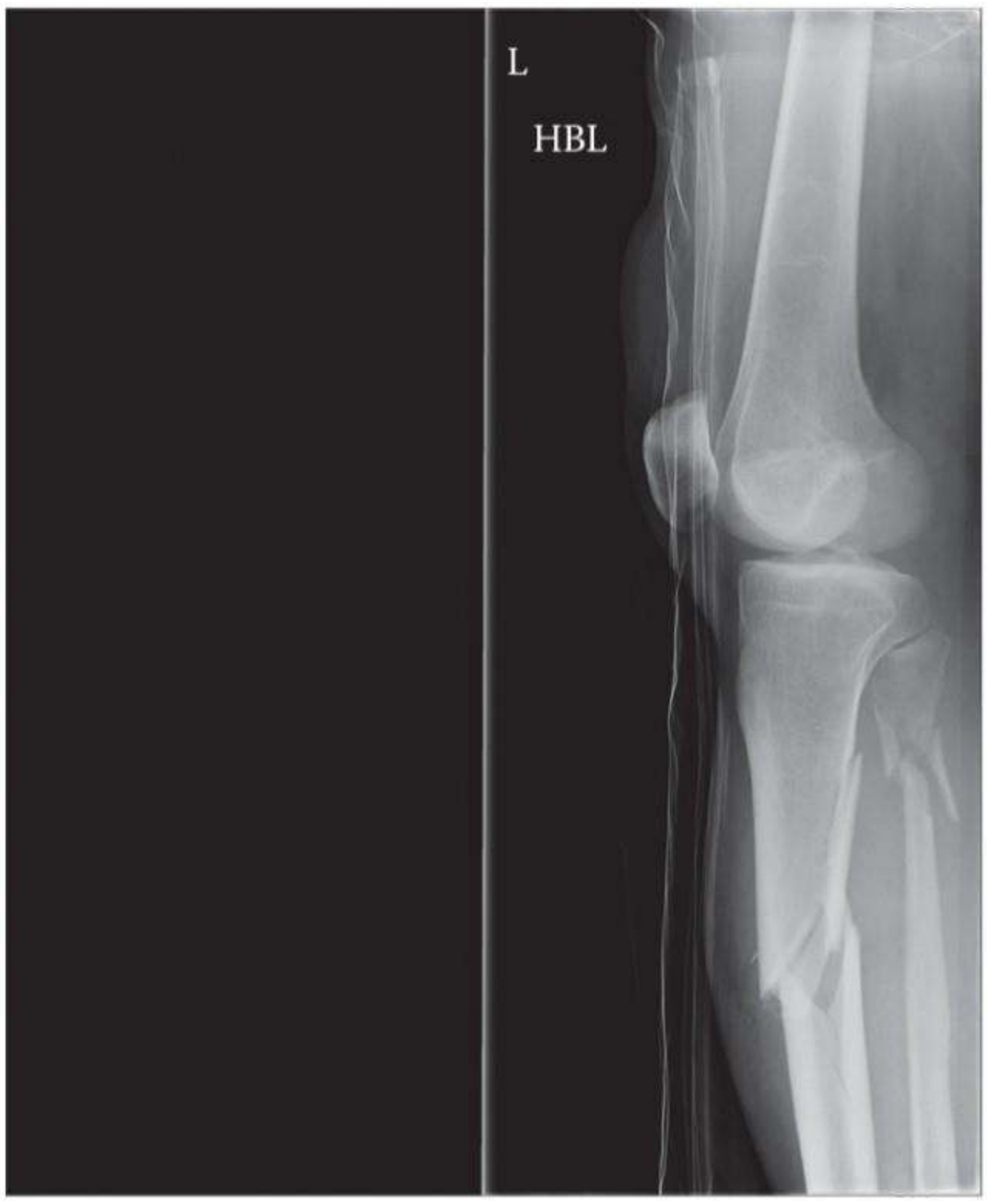
The patient should be instructed to return at 24–48 hours to have the pins retightened, and should be educated on pin hygiene.

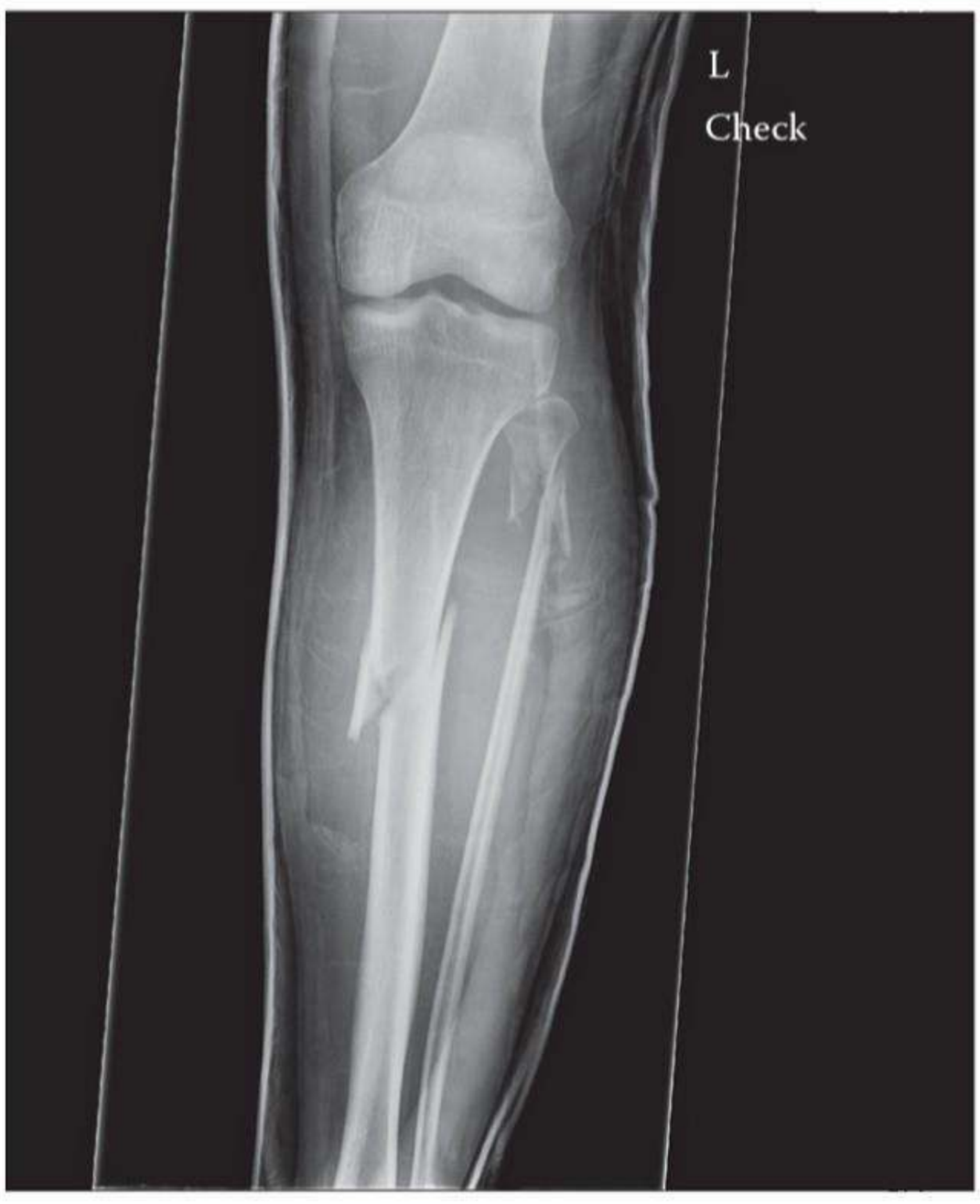
EXAMINER: What are the potential complications?

CANDIDATE: Loss of position or reduction, pin site infection and loosening, pain, nerve or vessel injury. One-fifth of patients also complain of pain which can be managed by loosening. Rarely there is a complication of dural puncture (1%).[2,3]

1. Bono CM. The halo fixator. *J Am Acad Orthop Surg* 2007;**15**:728–737.
2. Botte MJ, Byrne TP, Abrams RA *et al.* Halo skeletal fixation: techniques of application and prevention of complications. *J Am Acad Orthop Surg* 1996;**4**:44–53.
3. Garfin SR, Botte MJ, Waters RL *et al.* Complications in the use of the halo fixation device. *J Bone Joint Surg Am* 1986;**68-A**:320–325.

TIBIAL DIAPHYSEAL FRACTURE (PROXIMAL)

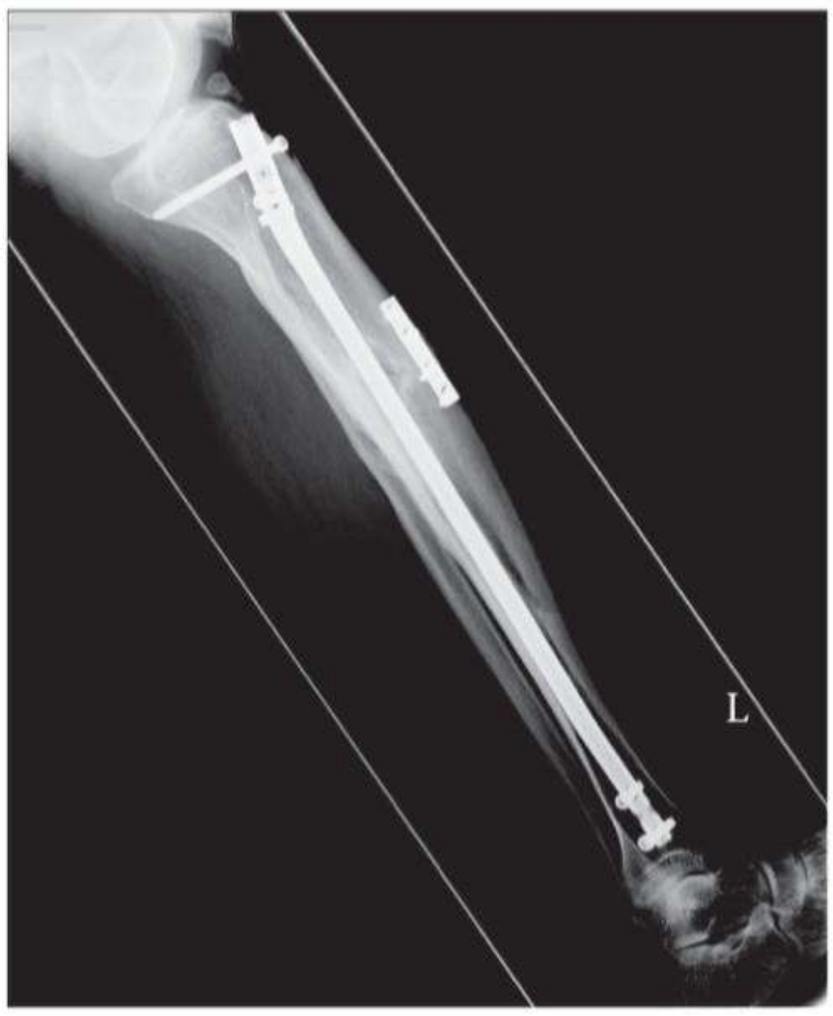




1. **Can you describe the radiographs?**
These are AP and lateral radiographs of the left tibia and fibula showing a proximal tibial diaphyseal fracture. The fracture is in valgus and procurvatum (apex anterior).

2. **What is responsible for the deformities seen here?**
The procurvatum is due to the unopposed pull of the patellar tendon, whereas the valgus deformity is due to the pull of the pes anserinus attached to the proximal fragment, and the bulky anterior compartment musculature preventing the fracture from displacing into a varus configuration.

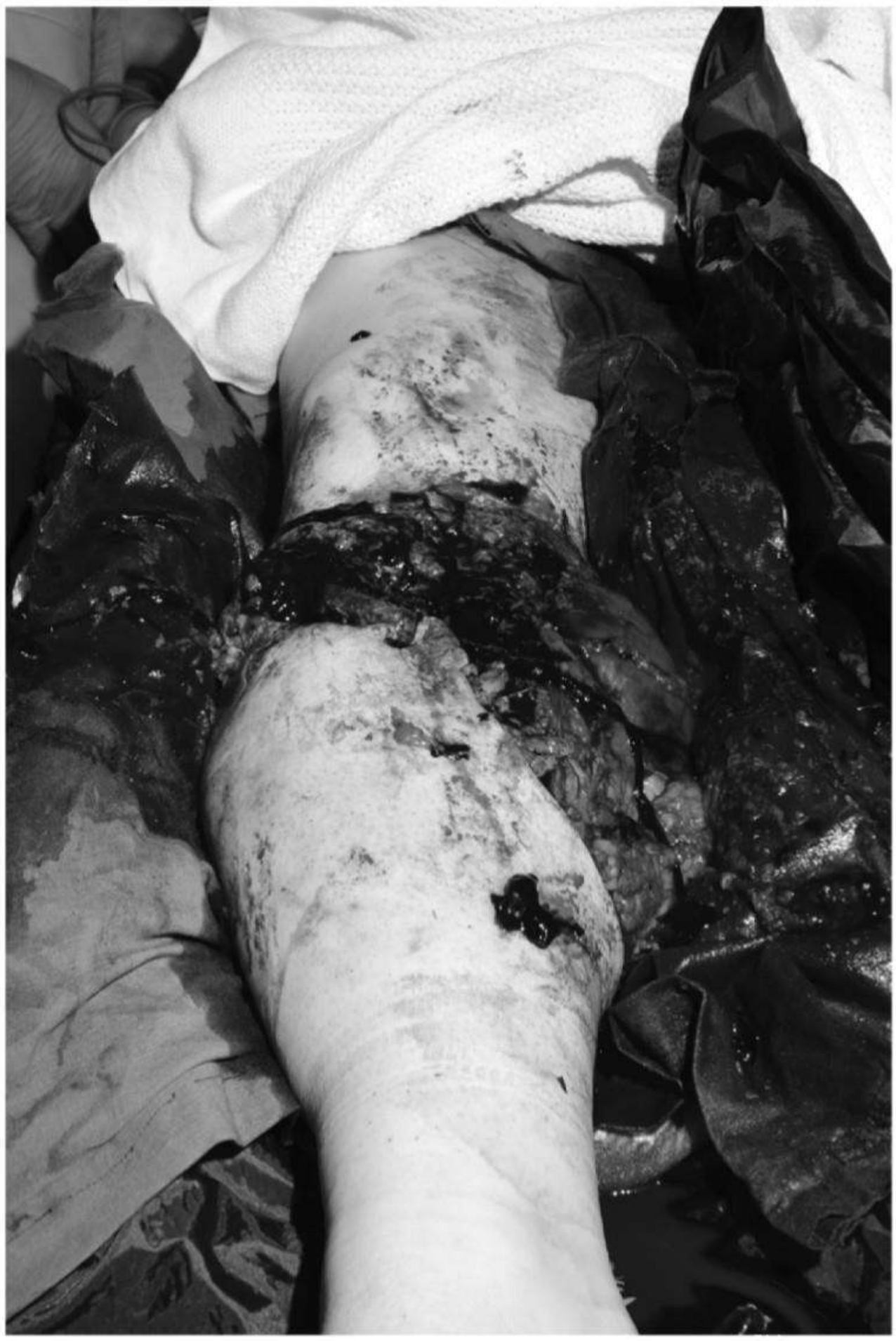
3. **If you were to treat this fracture with an IM nail, what techniques could you use to counteract these deformities?**
There are several techniques one could utilise to prevent this valgus and procurvatum deformity. Since the nail is not in contact with the cortical bone at the level of the fracture, the fragment can displace until the posterior or lateral cortex lies in contact with the nail:
   a. Poller* blocking screws: These are bicortical screws inserted before reaming and nailing. Alternatives include thick K-wires or 3/16-inch smooth Steinmann Pins. The screw/wire/pin blocks the incorrect path of the nail and channels the nail along the correct path preventing a mal-union. In the case of the fracture pictured above, a poller screw placed posteriorly will prevent the procurvatum deformity and a lateral screw will prevent the valgus deformity.
   b. Unicortical plate: A mini-open approach can be utilised to reduce the fracture and maintain fixation using a small fragment dynamic compression plate with unicortical fixation.
   c. Lateral starting point: A lateral parapatellar approach and lateral proximal starting point will allow the nail to abut the lateral cortex of the proximal fragment and prevent a valgus deformity.
   d. Semi-extended nailing: Nailing with the knee in flexion can exaggerate any procurvatum deformity. Most companies can now supply instrumentation to allow for nailing of tibial fractures in a semi-extended position, preventing fracture malalignment.



* *Poller* is German for bollard.

13

MANGLED EXTREMITY



This is a clinical photograph of a patient brought to the emergency department after being hit by a car that crushed and pinned his leg to a wall.

1. **Describe this clinical picture.**
   This is a clinical photograph of a patient with an obviously mangled and deformed leg. The wounds are ragged and the soft tissues appear dusky. There is obvious bone and soft tissue loss. It is not possible to tell from this photograph if this is an isolated injury.

2. **Assuming that this is an isolated injury, how would you manage this patient in the emergency department?**
   This patient would be treated along ATLS principles with concurrent resuscitation to treat life- and limb-threatening injuries and to address any catastrophic

haemorrhage early. I would address active haemorrhage from the wound with limb elevation and direct pressure and the use of a tourniquet if required. I would treat the patient with intravenous fluid and blood resuscitation as required and activate our major haemorrhage protocol if clinically indicated based on the patient's initial state, response to resuscitation and mechanism of injury triggers as per our local major haemorrhage policy. I would administer intravenous antibiotics, tetanus toxoid and analgesia. A clinical photograph should be taken before gross contamination is removed and saline swab dressings and a bandage are applied. Radiographs to confirm the bony injury and to help identify any proximal foreign body contamination should be taken. I would carefully assess and record the neurovascular status and include an assessment of pulses with Doppler ultrasound.

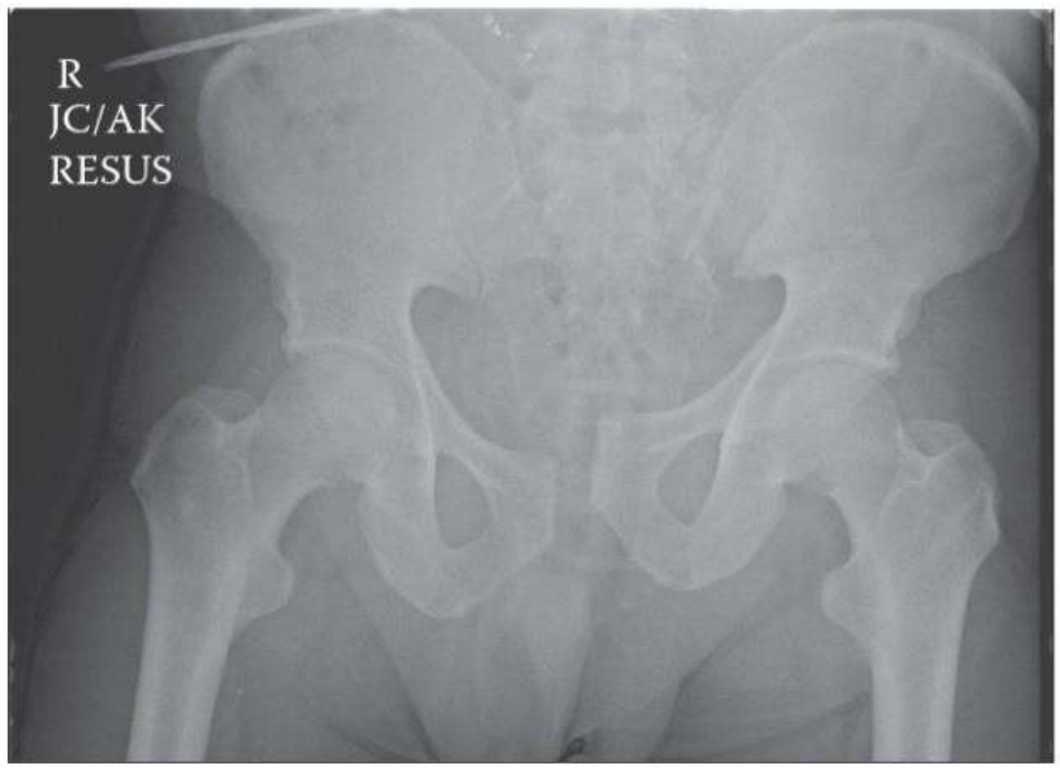
I would look closely for any suggestion of compartment syndrome, and an early senior surgical opinion is required as to whether limb salvage or early amputation for trauma is required. In any case, I would plan to take this patient to the operating theatre to perform a wound debridement. I would plan to provide temporary stability using an external fixator or plaster of Paris, but I would also consent the patient for amputation of the limb.

3. **How would you decide between limb salvage and primary amputation?**
There are a number of scoring systems to guide this decision. The Mangled Extremity Scoring System (MESS) is the most widely known but has largely fallen from favour. Loss of plantar sensation has also been used as a predictor of a poor outcome for limb salvage, as indicative of injury to the posterior tibial nerve, but this is also no longer used since the LEAP study demonstrated that at 2 years almost 50% of patients regained some plantar sensation.

The agreement of two experienced surgeons as to the need for amputation should be sought. In general, absolute indications for amputation are taken to include a contaminated traumatic amputation, a mangled extremity in a shocked and severely injured patient, and a crushed extremity with arterial injury and a warm ischemia time of greater than 6 hours. Relative indications include severe bone or soft-tissue loss, an anatomic transection of the tibial nerve, an open tibial fracture with serious associated polytrauma or a severe ipsilateral foot injury, or a prolonged predicted course to obtain soft-tissue coverage and tibial reconstruction. Despite this, the LEAP study suggests that the outcomes for both limb salvage and primary amputation are poor and that there is a high complication and re-operation rate. Where there is doubt about the state of limb perfusion, a CT angiogram can be helpful, but this should not introduce unnecessary delay in moving to the operating theatre to undertake debridement, restore limb perfusion if required and stabilisation.

4. **At what level would you plan to undertake this amputation and what are the principles that would guide your surgery?**
Current guidance from the BOA and BAPRAS is that open tibial fractures should be operated on within 24 hours with a combined 'orthoplastic' approach. Surgery should be undertaken more urgently if there is gross contamination or an arterial injury requiring repair. The level of amputation should be guided by the soft tissue injury and the level at which adequate soft tissue coverage can be obtained. Preservation of length improves energy expenditure during rehabilitation for the patient but should not compromise the adequacy of the debridement. In a clean wound, it would be usual to perform a definitive procedure at the time, fashioning flaps at the index surgery. For this injury, it may be possible to undertake a below

PELVIC FRACTURE



1. What does this radiograph show, and how would you manage this?

This radiograph shows a vertical shear type injury to the pelvis. This is typically the result of a high-energy injury, such as a road traffic accident or a fall from heights, and the possibility of other life-threatening injuries must be actively searched for using an ATLS approach to systematic diagnosis and management.

2. What would you expect to find on examination?

As part of the ATLS assessment it is possible that embarrassment to any and several body systems might be identified during the concurrent assessment and resuscitation of the patient. If there is an isolated injury to the pelvis, the patient may very well be distressed due to pain and potentially confused or agitated due to haemorrhagic shock. Additionally, there may be tachypnoea, tachycardia and hypotension.

On inspection, there may be an apparent leg length discrepancy in a vertical shear fracture where one hemipelvis is displaced proximally.

With regard to urogenital and rectal examinations, I would inspect for blood at the urethral meatus, as well as scrotal/labial/perineal haematoma, which may suggest significant urological trauma.

I would also inspect the state of the soft tissues in order to rule out an open fracture and to exclude a Morel–Lavallée lesion.

Moving onto palpation, a vaginal examination and rectal examination (in the supine position) to rule out a surreptitious open fracture is mandatory. I would palpate for a high-riding prostate (another sign of significant urethral injury) and anal tone/sensation to assess the sacral nerve roots.

I would also perform a full neurovascular examination of the lower limbs.

I would advise against 'springing' the pelvis as a method of diagnosing a pelvic fracture as this is painful, may disrupt a clot which is preventing torrential haemorrhage and is superfluous in the presence of an AP pelvic radiograph taken as part of a trauma series.

3. **In the absence of a CT, what additional radiographs might give you a better appreciation of the bony injury, and how would you obtain these?**
Two views are utilised to better visualise the pelvic ring. Although rarely used for diagnostic purposes given the ready access to modern CT scanning, these are the views that can be achieved with an image intensifier in theatre.

The first of these is an inlet view: The x-ray beam is angled approximately 45 degrees caudal, and an adequate image is obtained when S1 overlaps S2. This view is ideal for diagnosing widening of the SI joints, sacral ala impaction fractures, subtle pubic symphyseal injuries, as well as internal/external rotation or anterior/posterior translation of a hemipelvis.

The second is the outlet view: To obtain this, the x-ray beam is angled approximately 45 degrees cephalad. An adequate image is obtained when the pubic symphysis overlies the S2 body. Conversely, this is ideal for visualising vertical translation of a hemipelvis, as well as flexion/extension of a hemipelvis. As this is a true AP radiograph of the sacrum, it is ideal for diagnosing sacral fractures and their location in relation to the foramina.

4. **How would you classify pelvic fractures?**
I would classify these using the Young and Burgess classification of pelvic fractures. This define pelvic fractures into three categories, with subtypes of each. The categories are I – anterior posterior compression (APC); II – lateral compression (LC); and III – vertical shear (VS).

- APC I – Pubic symphysis widening <2.5 cm. This can be difficult to assess in the presence of a pelvic binder however. Can be managed conservatively.
- APC II – Pubic symphysis widening >2.5 cm. There has been disruption of the sacrospinous and sacrotuberous ligaments, as well as the anterior SI ligaments. The strong posterior SI ligaments remain intact and subsequently there is no loss of vertical stability.
- APC III – As per APC II but with disruption of the strong posterior SI ligaments. This results in loss of vertical stability and is indistinguishable from a vertical shear injury radiographically.
- LC I – Oblique or transverse pubic rami fractures, in addition to crush/compression fracture of the ipsilateral sacral ala.
- LC II – In addition to the pubic rami fractures, there is a characteristic 'crescent' fracture of the iliac wing.
- LC III – There is an ipsilateral lateral compression injury and a contralateral open book injury, known as a 'windswept pelvis'.
- VS – There is complete discontinuity of the sacral attachment to the lower limb. The posterior sacral ring may fail through the SI joint, the sacrum, or the ilium.

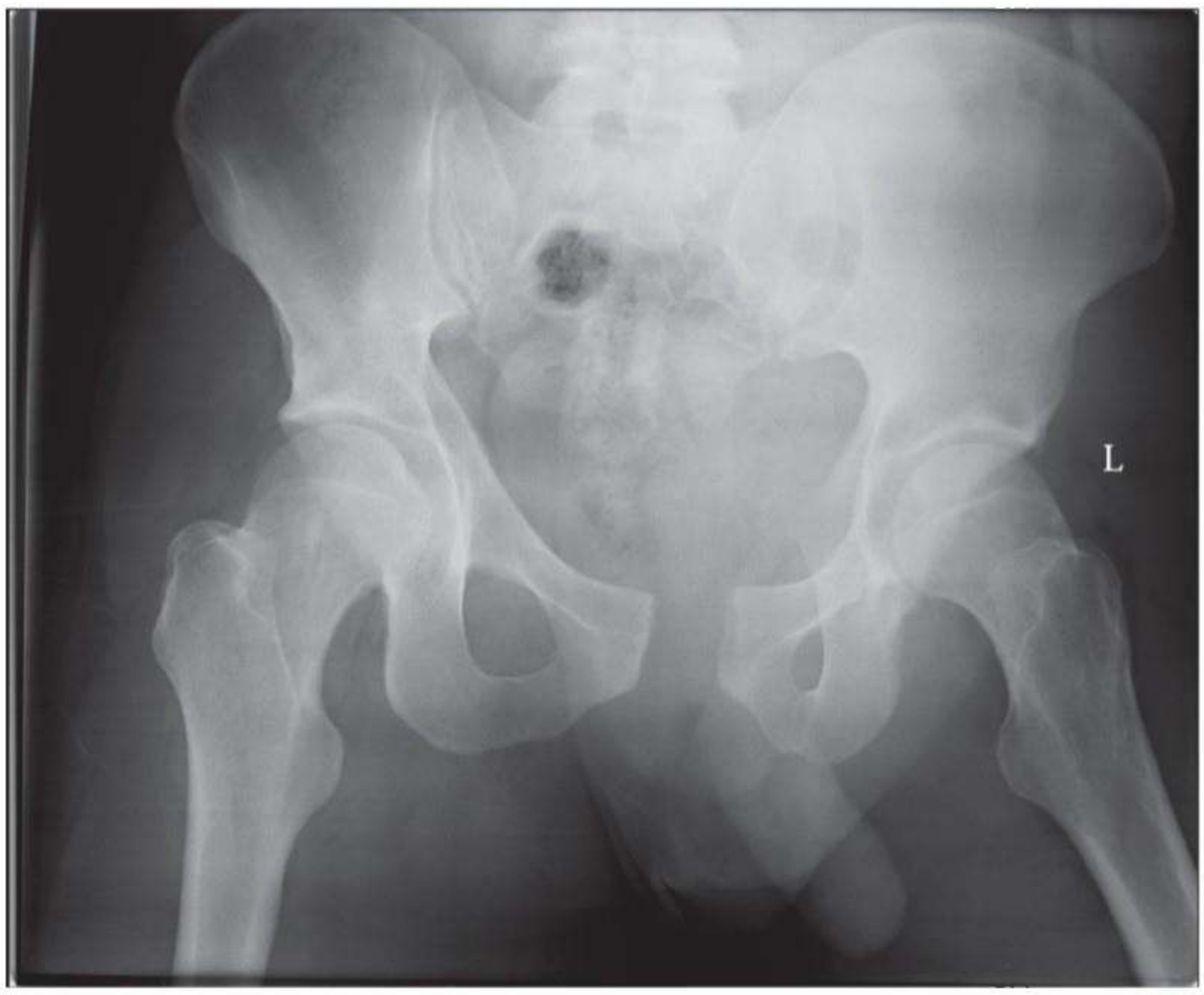
5. **What are the common sources of bleeding in a pelvic fracture?**
The most common source of bleeding (approximately 80%) in pelvic fractures is secondary to a shearing injury to the thin-walled posterior venous plexus. Fractures may result in clinically significant blood loss from cancellous bone surfaces.

Arterial injury is a less common source but the arteries most frequently implicated include the superior gluteal artery (APC pattern), the internal pudendal artery (LC pattern) and the obturator artery (LC pattern).

Although there are several specific sites for bleeding in association with pelvic fractures, one must remain vigilant for other sources associated with the high-energy mechanism of injury (intra-abdominal, intra-thoracic, limbs).

28

PELVIC FRACTURE



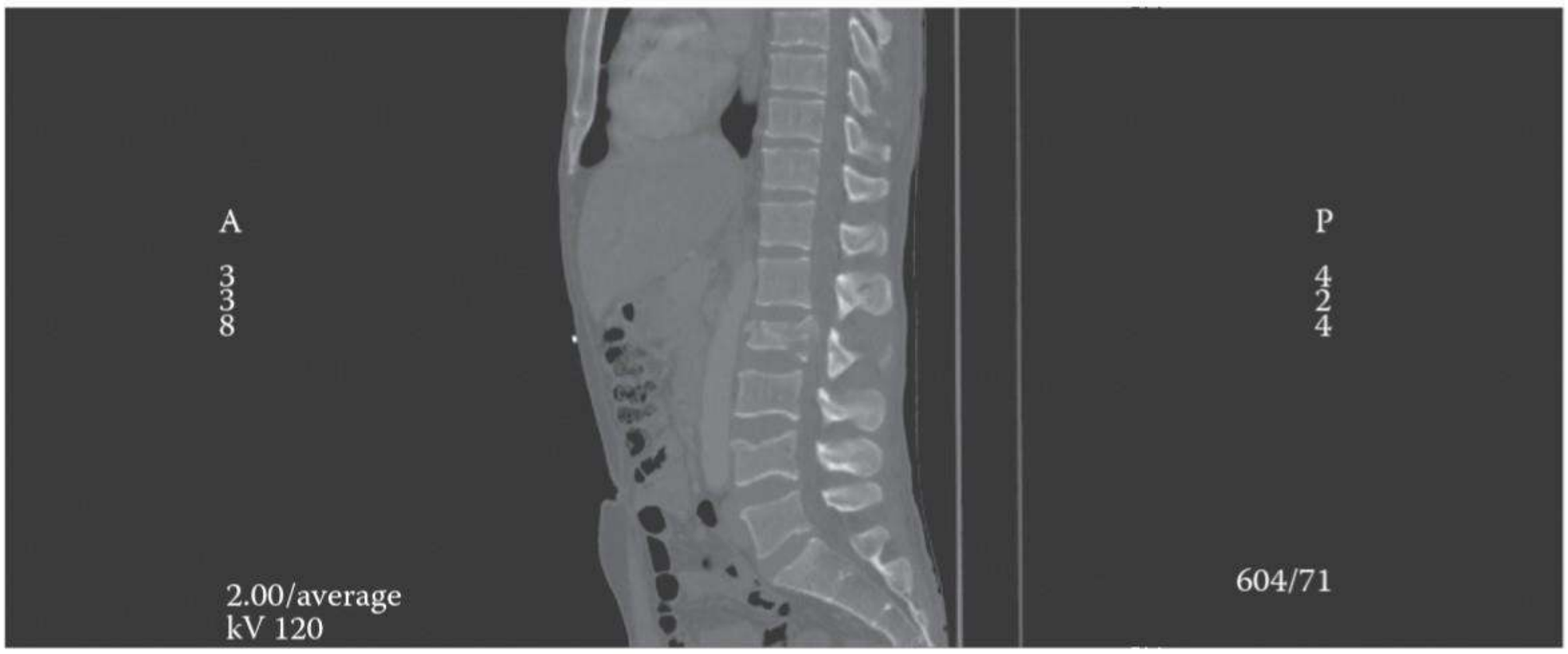
1. **What does this radiograph show and how would you classify this injury?**
This is an AP radiograph of the pelvis. It shoes an APC II fracture as per the Young and Burgess classification of pelvic fractures. The pubic symphysis has been disrupted, as well as the sacrotuberous, sacrospinous and anterior SI joint ligaments. The strong posterior SI ligaments appear intact as the left hemipelvis seems to have maintained vertical stability, distinguishing it from an APC III or vertical shear type injury.

2. **What signs would you look for to rule out a urological injury and how would you manage a suspected urethral injury in conjunction with this pelvic fracture?**
A high-riding prostate, blood at the urethral meatus or scrotal/labial/perineal haematoma may suggest significant urological trauma. This requires caution but does allow for a single, gentle attempt at urethral catheterisation. If the catheter does not pass or drains blood, the balloon must not be inflated. The catheter should be withdrawn and a retrograde urethrogram should be performed. Any concerns regarding urological injury must be discussed with the urology service.

   Any suspected urethral injuries in females and children should be discussed with the urology service urgently.

   If a urethral catheter cannot be passed, a suprapubic catheter is required. However, this can alter the approaches available for fracture fixation due to their

THORACOLUMBAR SPINE INJURY



A 44-year-old man has jumped from the fifth floor of a building and has been brought to the emergency department. He is complaining of back pain and spinal precautions have been taken. This is an image from the CT taken in the emergency department.

1. **How would you investigate and manage this patient in the emergency department?**
   This patient should be treated along ATLS guidelines to ensure that life- and limb-threatening injuries are identified and treated appropriately. With respect to this specific injury, I would maintain spinal precautions and examine the patient carefully looking for any sign of neurological deficit.

   The CT shows a fracture at the level of L2. This is a burst fracture pattern with involvement of the anterior, middle and posterior columns. The CT image shows significant loss of vertebral height and retropulsion of fracture fragments and spinal canal compromise.

2. **What are the indications for surgery in this injury?**
   Fractures with associated neurological injury should be considered for surgery as should fractures where there is 30 degrees or more of kyphosis, compromise of the spinal canal by 50% or more and where there is progressive collapse into kyphosis or risk of this. I would also consider surgery in polytraumatised patients or where bracing is ineffective because of other injuries or body habitus. Loss of 50% of vertebral height or more suggests injury to the posterior ligamentous complex and resulting instability. Most other fractures can be treated in a thoracolumbar spinal orthosis (TLSO) to be worn whenever the patient is upright. If there is any doubt as to the stability of a fracture then I would arrange for an MRI to examine the integrity of the posterior ligamentous complex. I would check that bracing maintains satisfactory position with serial standing radiographs following application of the TLSO.

   These indications for surgery have been consolidated into an injury scoring system, the Thoracolumbar Injury Classification and Severity Score (TLICS), which incorporates injury morphology, posterior ligamentous complex integrity and

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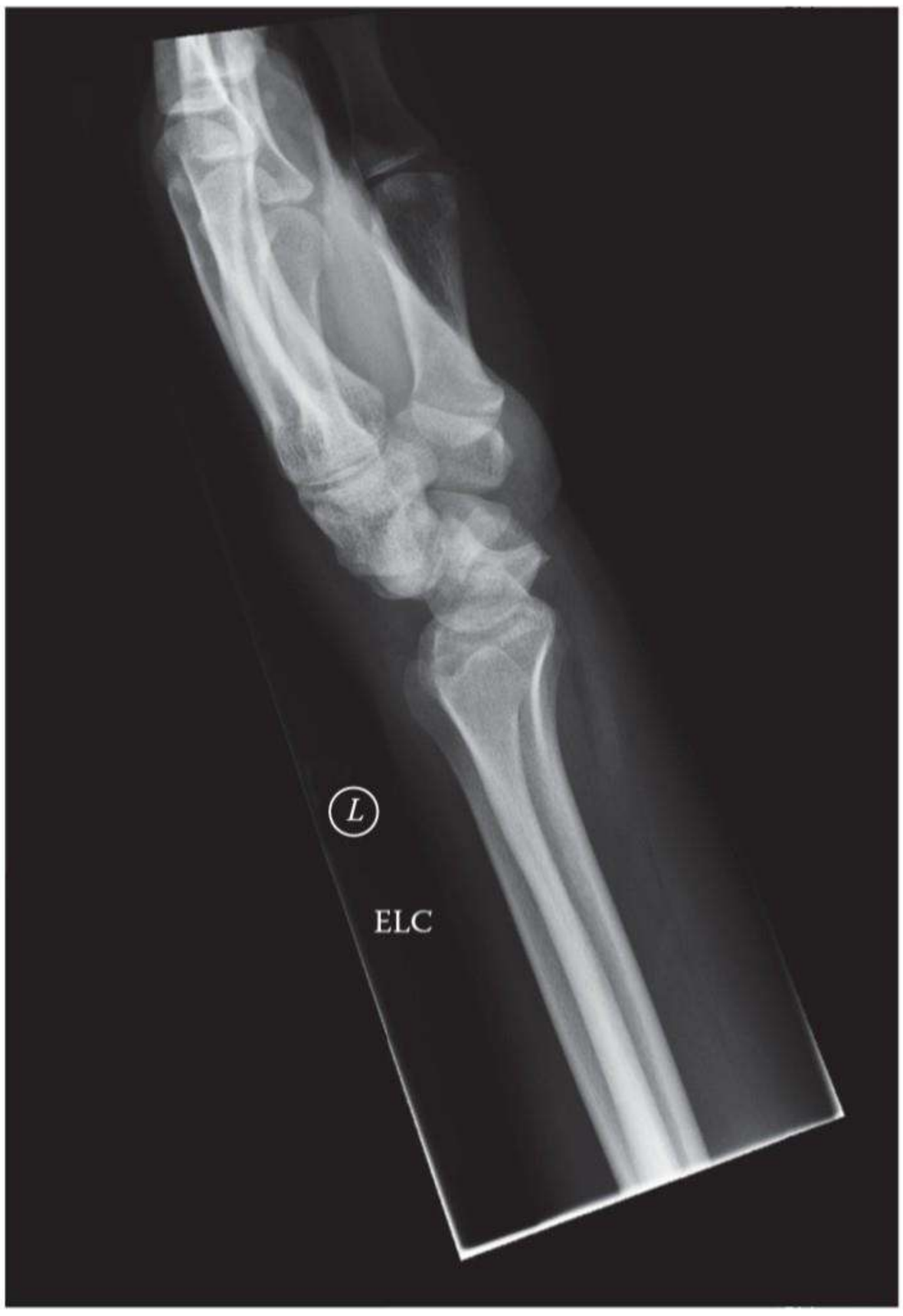
PERILUNATE DISLOCATION



1. Describe this radiograph.

This is a lateral radiograph of the wrist showing a perilunate dislocation. The lateral view shows a dorsal dislocation of the capitate and distal carpal row. The lunate remains in the lunate fossa of the radius owing to its strong ligamentous attachments from the short and the long radiolunate ligaments.

The PA view would show disruption of Gilula's lines and the capitate overlapping the lunate. There are no obvious fractures but I would look closely in order to identify any of the more commonly associated bony injuries, including fractures to the radial styloid, scaphoid, capitate, trapezoid, hamate and ulnar styloid.

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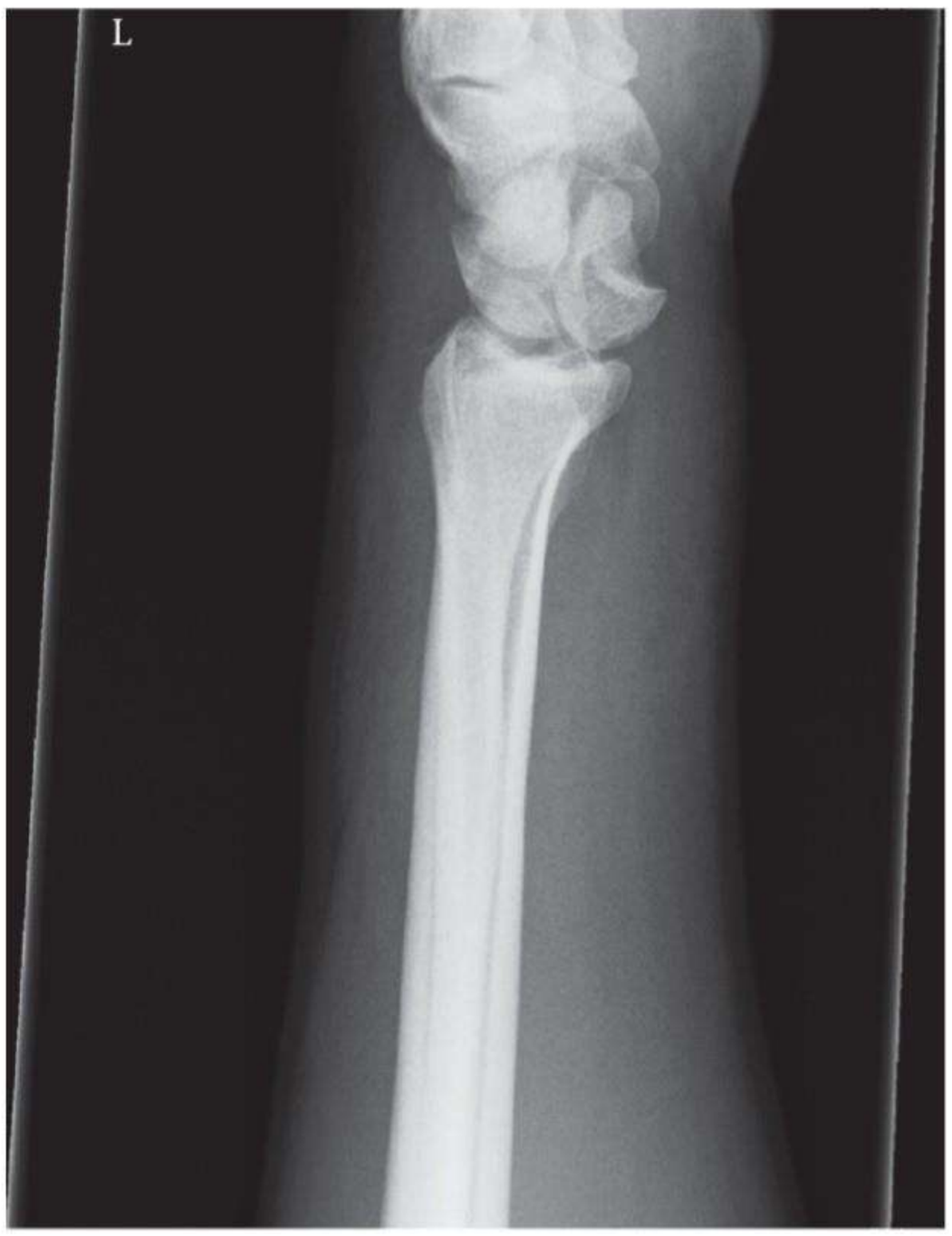
LUNATE DISLOCATION



1. **Describe this radiograph. What additional imaging might you request and what would you expect this to show?**
   This is a lateral radiograph of the left wrist which shows a volar dislocation of the lunate while the capitate is now articulating with the radius.

   I would request a PA view of the wrist and I would scrutinise this to look for malalignment in the carpus and of the radiocarpal axis. I would expect to see interruption of Gilula's lines and the lunate might appear to be 'wedge'- or 'slice of pie'-shaped and overlap the capitate. I would scrutinise the radiographs for any evidence of fractures and or intercarpal widening.

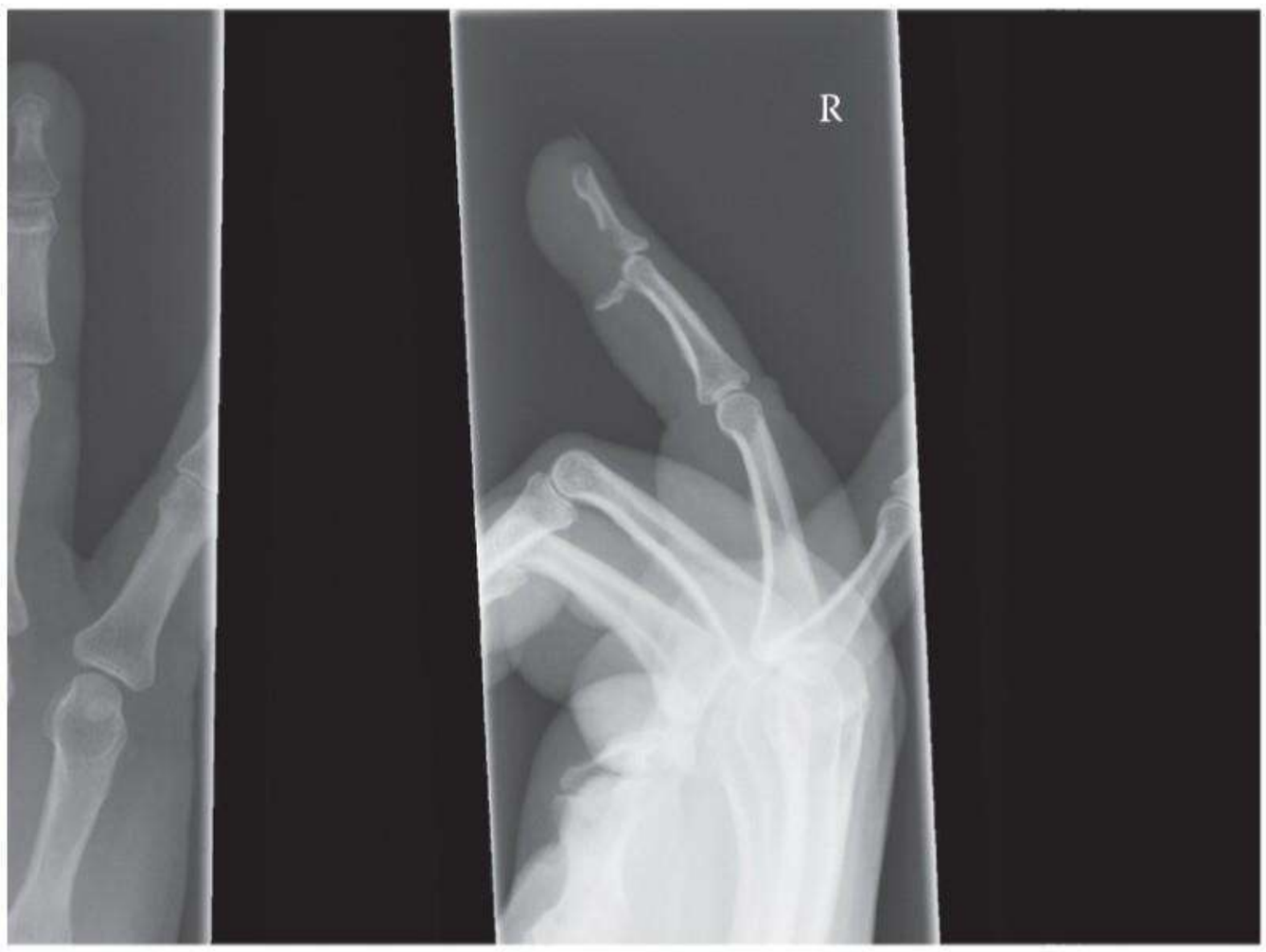
   The PA view would likely show disruption of the Gilula's lines, with the capitate appearing to overlap the lunate. One would also look for common bony injuries including the radial styloid, scaphoid, capitate, trapezoid, hamate and ulnar styloid.

2. **How many Gilula lines are there?**
   There are three in total:

   - The first line is a smooth curve outlining the proximal convex surfaces of the scaphoid, lunate and triquetrum.
   - The second line traces the distal concave surfaces of the same bones.
   - The third line follows the proximal curvatures of the capitate and hamate.

- The second line traces the distal concave surfaces of the same bones.
- The third line follows the proximal curvatures of the capitate and hamate.

JERSEY FINGER



A 22-year-old medical student presents to the emergency department with a painful and swollen ring finger at the distal interphalangeal joint. Forty-eight hours earlier he injured his finger playing for the Edinburgh Medics Rugby Football Club and he thinks the injury occurred whilst tackling an opponent. He managed to finish the game but complains of discomfort and reduced movement in the finger ever since.

1. **What does the picture show?**
   This history would lead me to suspect a 'jersey finger' or 'rugger jersey finger', which is a type of zone 1 flexor tendon injury. These are commonly described in American football players and classically occur when a tackler grabs hold of an opponent's jersey. The distal interphalangeal joint (DIPJ) is forcibly hyperextended while the flexor digitorum profundus (FDP) muscle contracts leading to tendon rupture at its weakest point, the insertion on the distal phalanx.

2. **How would you examine the patient to confirm the diagnosis and what would the radiographs likely show?**
   Inability to flex the DIPJ is suggestive of rupture of the FDP tendon. On inspection, the injured finger may lie in slight extension compared with the other fingers in the resting posture. There may be tenderness on the volar side of the finger and the retracted tendon may be palpable over the proximal phalanx or in the palm.

   I would test FDP function by fixing the middle phalanx in extension preventing flexion at the proximal interphalangeal joint (PIPJ) and asking the patient to attempt to actively flex the finger at the DIPJ. This prevents the action of flexor digitorum superficialis (FDS) and isolates the FDP.

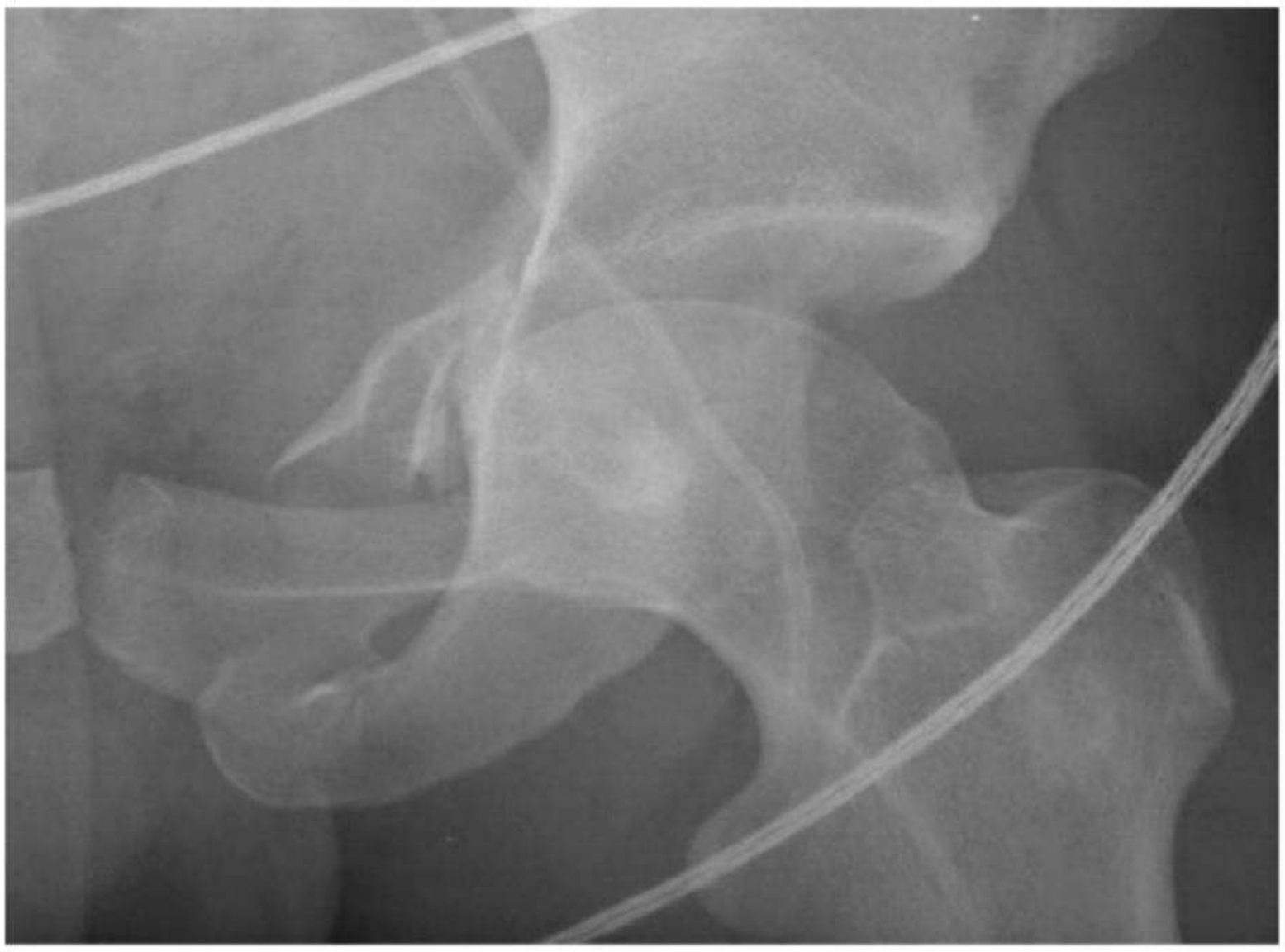
   Radiographs are often normal in a jersey finger injury, although in this case they reveal an avulsion fracture from the base of the distal phalanx.

Trauma Viva questions:

1 of 9

1. ABULAR FRACTURE.

Your registrar has asked you to come to A&E and help him manage this patient who was involved in a RTA. This is an isolated closed injury. Take me through what you are going to do.



What will you do?

This is an AP pelvic radiograph demonstrating an acetabular fracture, with both column involvement and a fracture of the acetabular floor, as demonstrated by the incongruity of both the ilioischial and ilioinguinal line. This is a high energy injury and I would:

1. Assess the patient according to ATLS protocol, and would identify any concurrent life threatening injuries.

2. With regards this specific fracture, I would undertake a thorough assessment of the neurovascular status.

3. I would investigate this fracture with further imaging, such as CT scan, and discuss the management of this patient with my local pelvic unit.

Which acetabular fractures require emergent surgery?

1. Open
2. NV injury-40% chance of sciatic injury with posterior column fracture.
3. Irreducible posterior dislocation

What are the indications for non-operative management?

What is the roof arc angle?-

2 lines- drop vertical from centre and another 45 degrees to it (like Centre edge angle). A fracture in the roof suggests an unstable fracture pattern.

What is the optimal timing of operation?
Ideally, not longer than two weeks.

What are the principles of fixation in acetabular fractures?

1. Anatomical reduction
2. Stable internal fixation
3. Early mobilisation

What approaches do you know for acetabular fracture fixation?

I would base my approach based on the fracture configuration:
1. If posterior dislocation of femoral head – posterior approach or extended Kocher-Langenbach approach.
2. If dislocation with femoral head fracture – anterior approach to fix femoral head fracture.

How will you manage femoral neck fracture with dislocation?

Lateral approach- to provisionally stabilise neck with K - wires. Reduce dislocation-and subsequently undertake definitive fixation of neck.

Do you know of any classification for acetabular fractures?

Letournal and Judet
Simple-Anterior wall/Anterior coloumn/posterior wall/posterior colomn
Associated-T shape/Posterior wall plus posterior coulomn/transverse plus posterior wall/Anterior colomn plus posterior hemitransverse/Both colomn

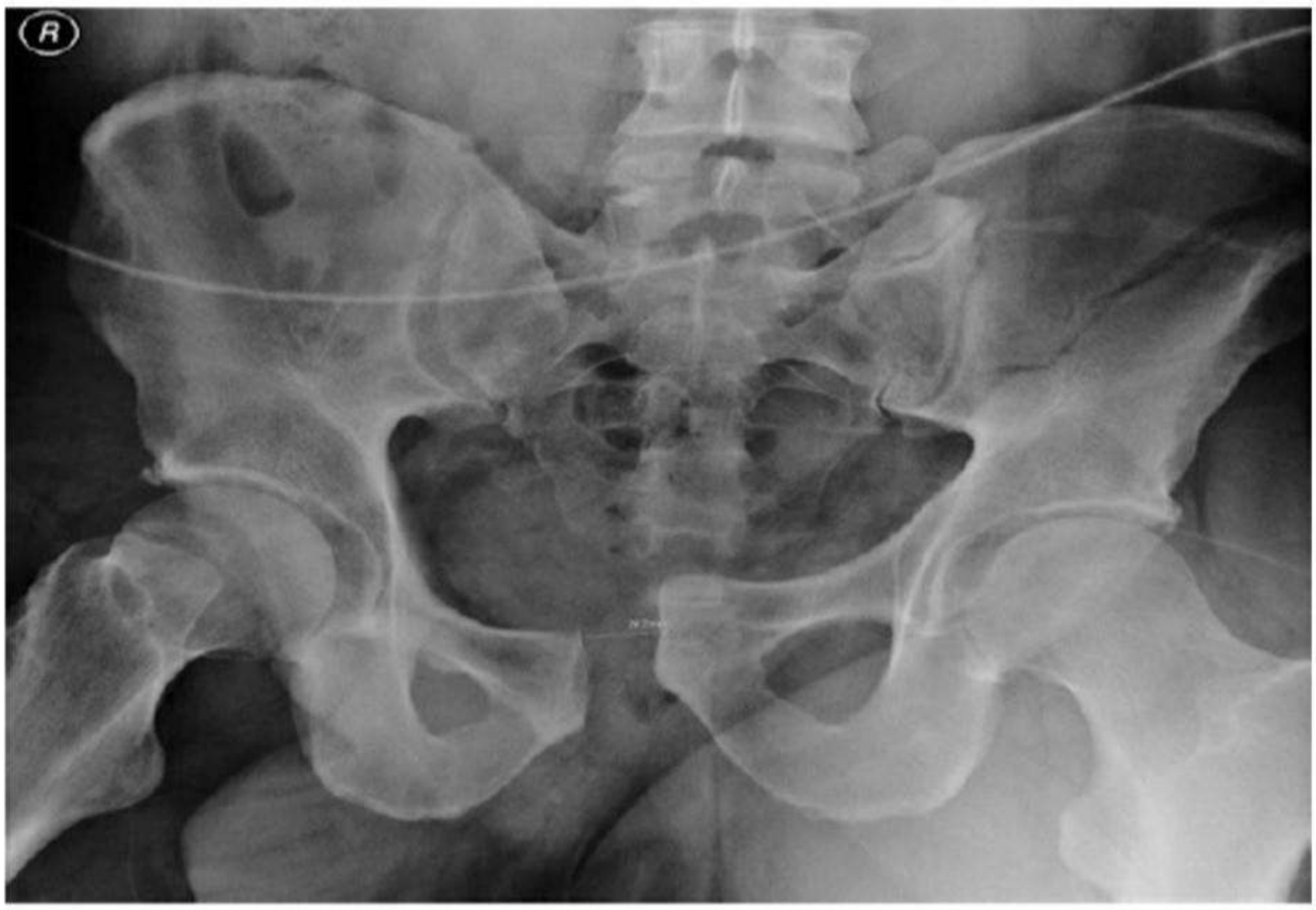
What factors have been shown to influence the outcome in acetabular fractures?

1. Age
2. Osteoporosis
3. Type-Anterior wall fracture/femoral head fracture/articular damage/Posterior wall multifragmentarity.
4. Early surgery in less than two weeks
5. Minimal surgical approach
6. Quality of reduction

2. fracture.

25 year old front seat driver, presents to A&E following RTA.

How will you manage this patient?



This is an AP pelvic radiograph demonstrating an open book pelvic injury. According to the Young and Burgess classification, this is an (anteroposterior compression)APC 3 fracture, with complete disruption of the sacroiliac joint and anterior pelvic diastasis.

I would undertake my initial assessment according to ATLS protocol as this is a high energy trauma. The principles of my initial management – would be to follow along agreed **BOAST** guidelines.

1. Assess haemodynamic status and fracture stability.

2. If haemodynamically unstable (major bleed)
I would consider:
-Early application of pelvic binder/ sheet or external fixator
-Resuscitate –Blood/FFP/platelets.
- Determine the need to initiate a **massive transfusion protocol.**
-Aim to get a primary clot.
-Assess chest/abdomen by surgeons- plain radiograph chest/FAST scan, to rule out other sources of bleeding.

3. If still unstable-- consider angio suite for emergency embolisation OR open pelvic packing after ex-fixator.

4. CT scan pelvis- only after haemodynamic stabilisation.
5. For definitive pelvic reconstruction – within five days after haemodynamic stable and temporary skeletal stabilisation.
6. Assess for other associated injuries –Genito urinary/GIT

You notice blood on PR, and blood at penile tip. What else would you consider doing at this

You notice blood on PR, and blood at penile tip. What else would you consider doing at this stage?

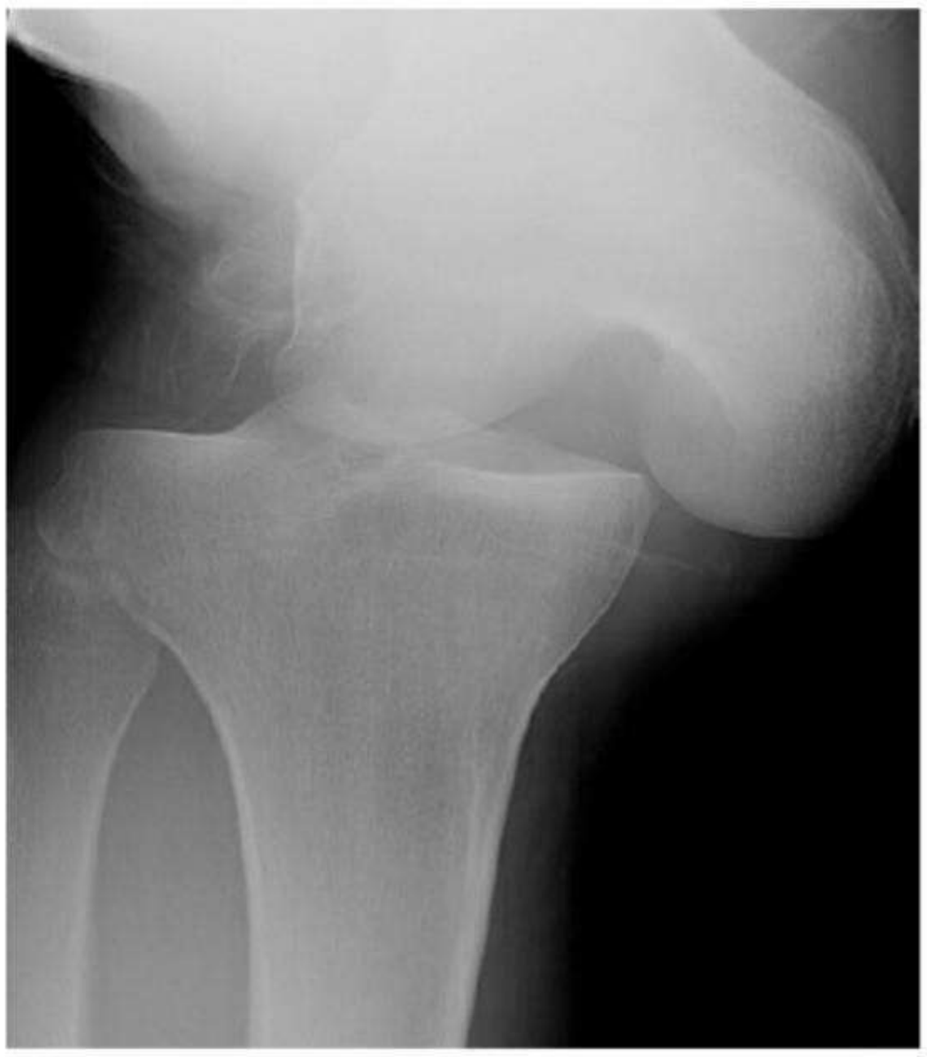
I would be worried about associated genitourinary and gastrointestinal tract injury. I would therefore involve an urologist and a general surgeon early.

- Urologist to consider urgent contrast studies (cysto/urethrogram) to assess for:
- extra peritoneal injury –such as urethral laceration/rupture.
- intra peritoneal injury- open bladder laceration.
- open pelvic injury with wound to the groin/buttockvagina/rectum- need to consider draining bladder with cystostomy.
-posterior urethral injury- supra pubic catheter and repair.
-open pelvis with bowel injury- needs bowel diversion with end colostomy upper quadrant.

When would you consider applying a temporary external fixator on before laparotomy?
This is controversial, but one of the indications would be to stabilise the pelvis during transfers.

4. KNEE DISLOCATION.

This are the plain radiographs of a 25 year old patient brought in by ambulance following a rugby tackle.



How will you assess, this patient?

1. Initial assessment according to ATLS protocol
2. Immediate reduction needed – in A & E,
- splint in 20 degree flexion.
- Postero lateral dislocations may need open reduction, as the medial femoral condyle may have button holed through the capsule.

3. Assess vascular injury -30-40%
-Assess at regular intervals if pulse normal
-Angiography if (ankle brachial pressure index)ABPI less than 0.9
-Maximum ischaemic time -6-8 hours
Popliteal artery tethered
- proximally in Hunters canal under the fibrous arch of the adductor muscles
- distally under fibrous arch of soleus
- in middle by 5 geniculate arteries

4. Assess neurology -10-30%
5. Get radiographs to assess associated fractures
6. Get MRI/MR arthrogram to assess ligament damage

Do you know of any Classification?

Shenck Anatomic Classification 1992
Five types -1/2/3/4ligaments/ Fracture dislocation with or without neurovascular injury.

When would you consider emergency surgery?

Absolute indications for emergency surgery are:
1. Arterial injury
- external fixator and vascular surgery

2. Irreducibility
- open reduction
- apply external fixator if unstable in brace

3. Open dislocation / washout
- do not perform early ligament reconstruction
- apply external fixator if unstable in brace

4. Compartment syndrome

When could you consider options for dealing with this injury?

Non operative

Brace 30 – 40 for six weeks
- for elderly patients with multiple co-morbidities.
- external fixator for obese patient
- Disadvantage: stiffness

Levy et al Arthroscopy 2009 Systematic Review
-showed improved outcomes with operative management.

Early versus Late Surgery?

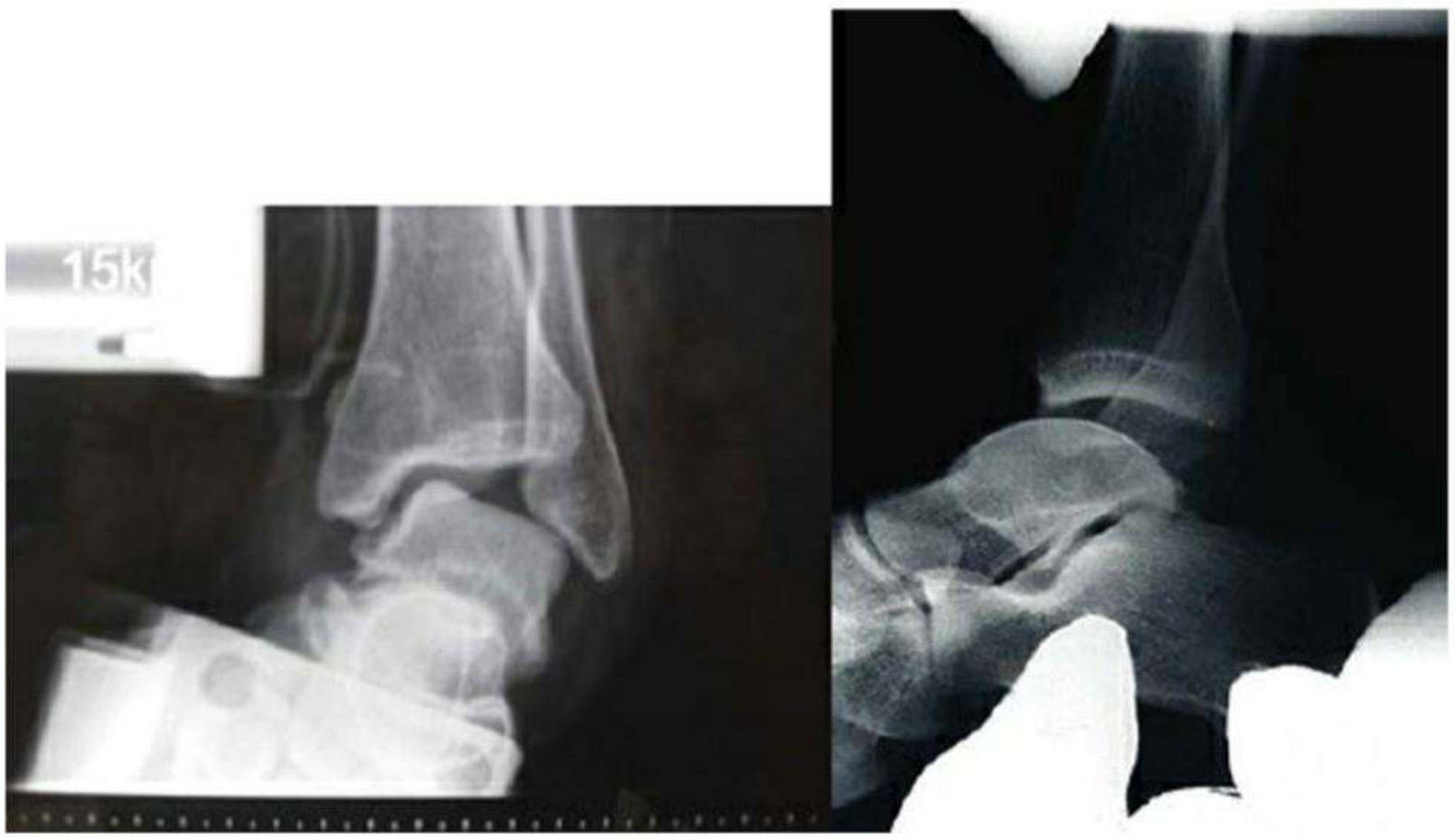
Levy et al Arthroscopy 2009 Systematic Review
- early treatment consistently results in higher outcome scores.
- easier identification of tissues
- some tissue amenable to repair

Timing for definitive surgery

- Allow swelling to settle
Beyond three weeks, collateral and posterolateral structures too scarred for early repair and will need late reconstruction.

Repair versus Reconstruction?
1. Postero-lateral corner (PLC)
- usually treated early with repair of avulsions.
- reconstruction for midsubstance injuries

1. Ankle instability



Shown stress radiographs of ankle. Management, surgical options and long term outcome

How will you proceed?

Need to know basic anatomy of lateral ligament complex
-Three main structures

- ATFL(Anterior talofibular ligament)-primary resistant to anterior shift
- CFL(Calcaneofibular ligament)-primary resistant to talar shift.
- PTFL(Posterior talofibular ligament)-resists posterior shift

Type of injury:
-Acute

- Stable
- Unstable

- Partial tear
- Complete tear

-Chronic injury

- Previous history of inversion injury
- Delayed presentation of ankle instability

Important component of the injury is proprioceptor damage

History.
-Duration of symptoms
-Mechanism- commonly inversion injury

- Forced inversion injury leads to damage to ATFL(Anterior talofibular ligament) first

- Forced inversion injury leads to damage to ATFL(Anterior talofibular ligament) first

- Forced inversion injury leads to damage to ATFL(Anterior talofibular ligament) first
- More severe injury follows damage to CFL(Calcaneofibular ligament)
- Isolated CFL injury is impossible
- PTFL(Posterior talofibular ligament) injuries are rare

-Instability/giving away
-pain
-clicking symptoms
-swelling

Examination

1. Test Anterior talofibular ligament(ATFL) –test in plantar flexion-Invert foot
2. Test Calcaneofibular ligament(CFL) –invert foot in neutral
3. Tender posterior to lateral malleoli
4. Hind foot varus
5. Anterior drawer test- Ankle in 20 degree plantarflexion, foot drawn forwards
6. Test syndesmosis- externally rotate foot with tibia stabilised
7. Test generalised ligamentous laxity- beighton score

Grade –
1 is injury without macroscopic tear, pain, no instability
2-Partial tear with mild to moderate instability
3-complete tear with instability

Investigations

1. Stress radiographs- tilt/ anterior tibial osteophytes/ talar exostosis

-AP stress view

- Normal tilt is 10-15 degree
- More than 15 degree- Anterior talofibular ligament (ATFL) +Calcaneofibular ligament(CFL)
- More than 30 degree-Anterior talofibular ligament (ATFL)+ Calcaneofibular ligament(CFL)+Posterior talofibular ligament(PTFL)
- Higher degree tilt may be due to general laxity
- Comparing with other side is useful

-Lateral stress view

- -Look for forward displacement
- Normal 3mm
- More than 9mm indicates a torn anterior talofibular ligament (ATFL)

2. MRI –look for osteochondral defects

Commonest associated lesions

Anterior talofibular ligament (ATFL)/ Calcaneofibular ligament (CFL)/Osteochondral defect talus

release all compartments.

8. Clinical picture of ruptured tendo Achillis. Clinical assessment and treatment options. Surgical options for chronic rupture.

Features

1. Middle aged men –sudden dorsiflexion mechanism
2. Degenerate tendon–
3. Risk factors- steroid intake/flouroquinolones

Mechanism

-direct trauma
- sudden dorsiflextion (e.g while playing squash or badminton)
- previous injury or degenerative tendon pathology (watershed area)

Examination

Look – feel- Move

-skin
-swelling
-feel gap
-Squeezing/Simmomd's calf muscle test

Management

*it is important to discuss with patient
*following factors should be consider

-age
-activity level
-associated medical conditions

Operative vs non operative

Evidence- Canadian RCT 2010 jbjs- no difference between 2 methods

Comparison

	Operative	Non operative
Re rupture	3%	13%

chapter
18

BASIC SCIENCE OF ORTHOPAEDIC TRAUMA

Viva 1 Questions

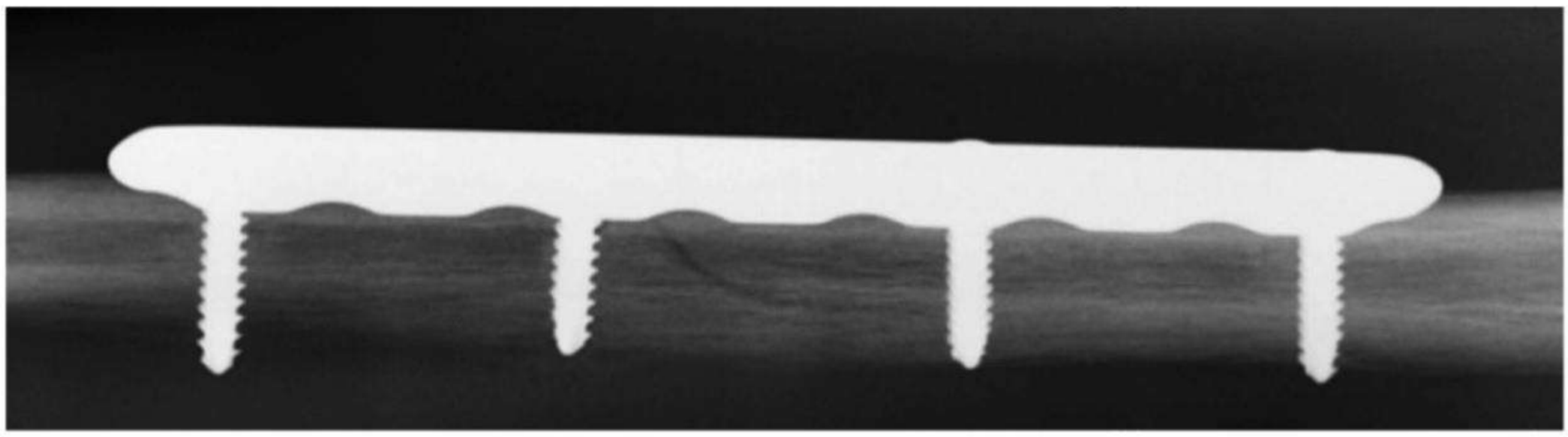


Figure 18.1 Plate fixation of fracture

This image shows a forearm bone fixed with a plate and screws. What type of bone healing would you expect to occur here?

Under what circumstances can the contact healing type of primary/direct bone healing occur?

Describe the Haversian structure of cortical bone.

Can you draw and label a cutting cone?

How can you achieve interfragmentary compression across a fracture in a long bone?

What influences how cortical bone remodels over time?

Viva 1 Answers

This image shows a forearm bone fixed with a plate and screws. What type of bone healing would you expect to occur here?

I would expect primary direct bone healing without callus formation in this situation.

Under what circumstances can the contact healing type of primary/direct bone healing occur?

Primary direct bone healing without callus can only occur with anatomical reduction and rigid internal fixation with compression and absolute stability. Anatomical reduction means a gap of less than 0.01 mm and rigid fixation with interfragmentary compression is characterized by a strain environment of under 2%. These conditions lead to cutting cones forming at the ends of osteons adjacent to the fracture site. They generate longitudinal cavities at a rate of 50–100 μm/day. Subsequently, bone produced by osteoblasts fills these cavities. This results in the simultaneous formation of a bony union and the restoration of Haversian systems formed in an axial direction.

Describe the Haversian structure of cortical bone.

[NB: DRAW A DIAGRAM WHILE YOU TALK.]

Mineralized bone is deposited on concentric lamellae arranged around a central vascular (Haversian) channel containing vessels and nerves. Lacunae containing osteocytes are present between the lamellae and are connected to adjacent lacunae by canaliculi, which facilitate cell-to-cell signalling and nutrient supply. A cement line marks the periphery of an osteon containing the concentric lamellae, lacunae, and Haversian canals. Transverse and oblique Volkmann's canals connect the Haversian canals both to each other and to the endosteal and periosteal vessels. Irregular fragments (interstitial systems) are present between the osteons but are separated from them by cement lines. Inner and outer circumferential lamellae extend around the whole circumference of the cortical bone adjacent to the respective endosteum and periosteum.

Can you draw and label a cutting cone?

[NB: DRAW A DIAGRAM WHILE YOU TALK.]

A cutting cone is characterized by a broad leading front formed of osteoclasts which resorb bone. A narrowing tapered tail—the closing cone, formed of osteoblasts that lay down new bone—follows the cutting cone. The cone is surrounded by lamellar bone, and just behind the lead osteoclasts lies a network of new capillaries.

How can you achieve interfragmentary compression across a fracture in a long bone?

Key to any attempt at gaining compression is the need for bony contact, and in most cases anatomical reduction of the fragments. Assuming this, interfragmentary compression can be achieved by placing a device under tension across the fracture site, which in turn creates compression at the fracture site. There are three main ways in which this can be achieved.

Firstly, it is possible to use a lag screw perpendicular to a fracture plane. In this case, a screw passes through a gliding hole in the near fragment to grip the opposite fragment in a threaded pilot hole, producing interfragmentary compression when it is tightened.

Secondly, one could use a compression plate. The plate should have oval holes through which eccentrically placed screws can be inserted to provide compression across a fracture site. The holes of the plate are shaped like an inclined and transverse cylinder—the screw head slides down the inclined cylinder as it is tightened. This results in the plate (and the previously attached fracture fragment on the opposite side of the fracture) being moved horizontally towards the eccentrically placed screw as it is driven home. For transverse fractures the plate should be slightly pre-bent in order to prevent gapping on the far cortex as tension is applied to the plate. For oblique fractures it is important to apply tension so that the spike of the mobile fragment is pressed into the axilla formed by the plate and the other main fragment.

Finally, you can use an articulated tension–compression device. One side of the fracture is fixed to the plate and on the other side a screw is inserted distal to the end of the plate and through the tension device, which itself is hooked into the last hole of the plate. As the articulated tension–compression device is tightened, it pulls the plate (and the previously attached fracture fragment on the opposite side of the fracture) towards the screw, generating tension in the plate and compression across the fracture site.

What influences how cortical bone remodels over time?

Wolff's law defines the relationship between mechanical stress and bone remodelling. Bone that is subject to compressive loads will be reinforced as osteoblasts are stimulated and additional lamellar bone is deposited. The mechanism for this is piezoelectric charge/polarity; on the compression/concave side of a bone this is electronegative (stimulates osteoblasts) and on the tension/convex side of a bone it is electropositive and stimulates osteoclasts (to resorb bone). There must also be an adequate blood supply and sufficient mechanical stability.

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Viva 2 Questions

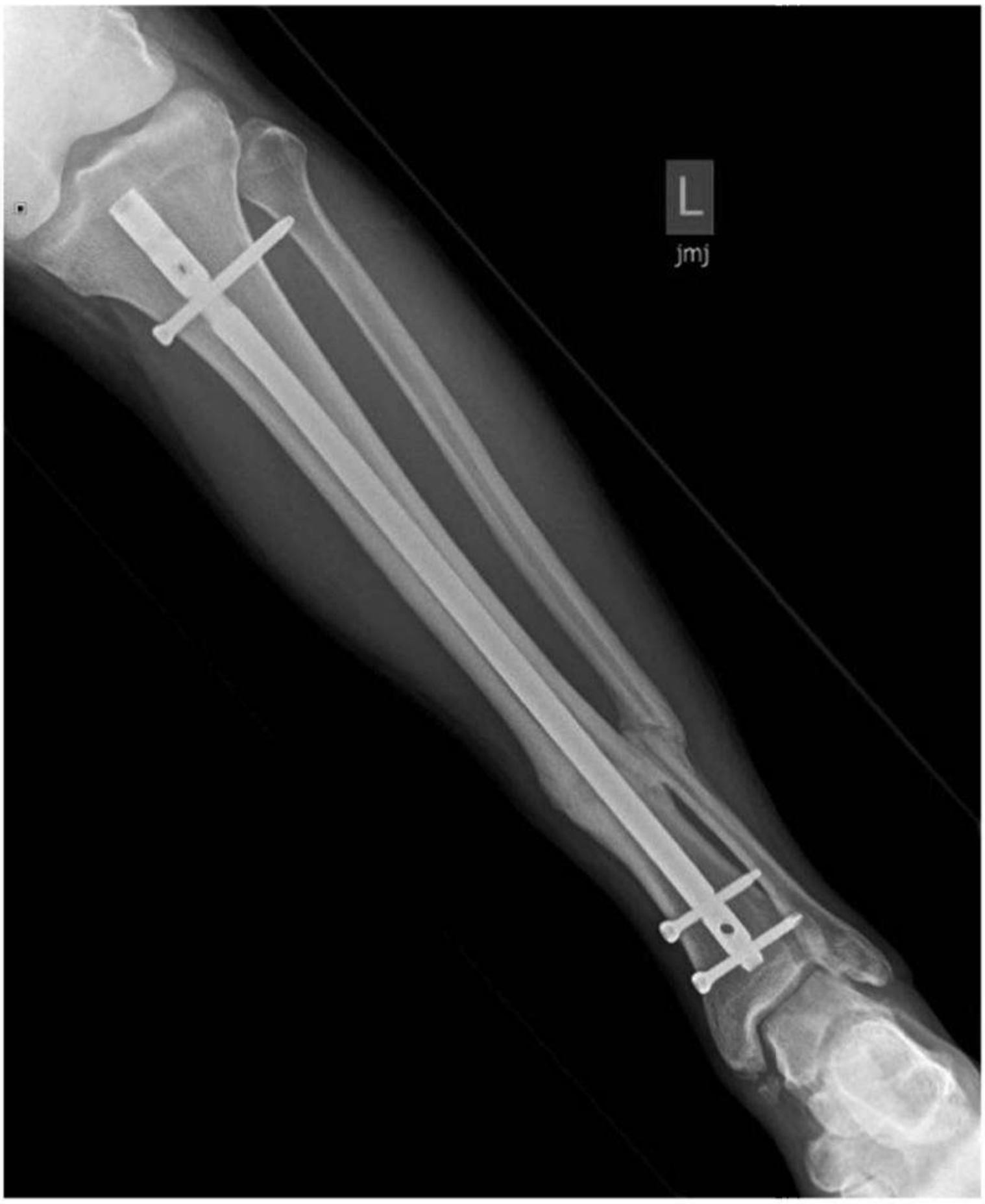

Figure 18.2 Tibial nail

What type of fracture healing has occurred in this case?

Describe the stages of secondary fracture healing.

How can you affect this process as a surgeon?

What impact do patient lifestyle factors have on bone healing?

Viva 2 Answers

What type of fracture healing has occurred in this case?

The intramedullary nail provides relative stability. Assuming an adequate blood supply the fracture has healed through callus formation. In orthopaedics we refer to this mode of healing as secondary or indirect healing. The callus that forms will be a mixture of fibrocartilagenous bridging callus (endochondral ossification) and periosteal bony callus (intramembranous ossification).

Describe the stages of secondary fracture healing.

Fracture healing with callus involves a continuum of stages from inflammation and haematoma all the way to bone remodelling. These stages are not mutually exclusive, and in reality the stages happen in parallel rather than rigidly sequentially. Different areas of the fracture may well be subject to different stages of healing as the local strain environment changes within different parts of the fracture site over the course of progression to union.

In essence the phases of fracture healing start with haematoma formation and activation of a local inflammatory cascade. Platelets, polymorphs, macrophages, and osteoclasts aggregate in response to endothelial disruption and the release of pro-inflammatory factors such as IL-1 and IL-6, TGF-β, TNF-α, and PDGF. Dead bone fragments are phagacytosed and fibrin clots are formed. Local strain here will approach 100%. Following this phase, fibroblasts arrive in response to VEGF, forming granulation tissue. This early part of soft callus formation is accompanied by the process of revascularization and neoangiogenesis. Chondrocytes subsequently arrive and form the cartilaginous matrix of the soft callus in response to a strain environment of 2–10%. As the interfragmentary strain reaches levels below 2%, osteogenic cell lines are recruited and osteoblasts are formed, allowing the production of bone (type I collagen matrix). BMPs have an active role at this point. This leads to the formation of hard callus (woven bone) which is ultimately replaced with lamellar bone in response to Wolff's law in the process of remodelling. The continuum lasts for up to 2 years, with the majority of the initial inflammatory and soft callus phases complete by 8–12 weeks.

How can you affect this process as a surgeon?

As a surgeon, I can influence both the biology and the mechanical stability of the fracture environment. Biological interference in the form of soft tissue dissection and stripping can disrupt the healing cascade, as can the introduction of infection. It is essential that a viable blood supply is maintained to the fragments to allow healing to occur, and this has led to the development of minimally invasive operative techniques which often base incisions away from the zone of injury.

The mechanical environment can be manipulated by the surgeon both intentionally and by accident. Failure to achieve reasonable bony contact and alignment may well lead to unacceptably high-strain environments, whilst excessive use of screws and locked implants may induce exceedingly low-strain environments, in effect slowing or even stopping the healing process. There is no way to measure strain clinically at a fracture site, so basic principles such as near–far fixation constructs should be employed to minimize the chance of having a wildly inappropriate level of strain.

What impact do patient lifestyle factors have on bone healing?

Several patient factors beyond medical comorbidities such as diabetes and malnutrition influence bone healing. Smoking has been shown to increase the incidence of delayed and non-union in tibial fractures—indeed even being a previous smoker increases the likelihood of non-union.

Non-steroidal anti-inflammatory drugs (NSAIDs) have also been shown to increase time to union. Interestingly HIV has not been shown to be a risk factor for infection or wound complications in open tibial fractures.

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Viva 6 Questions

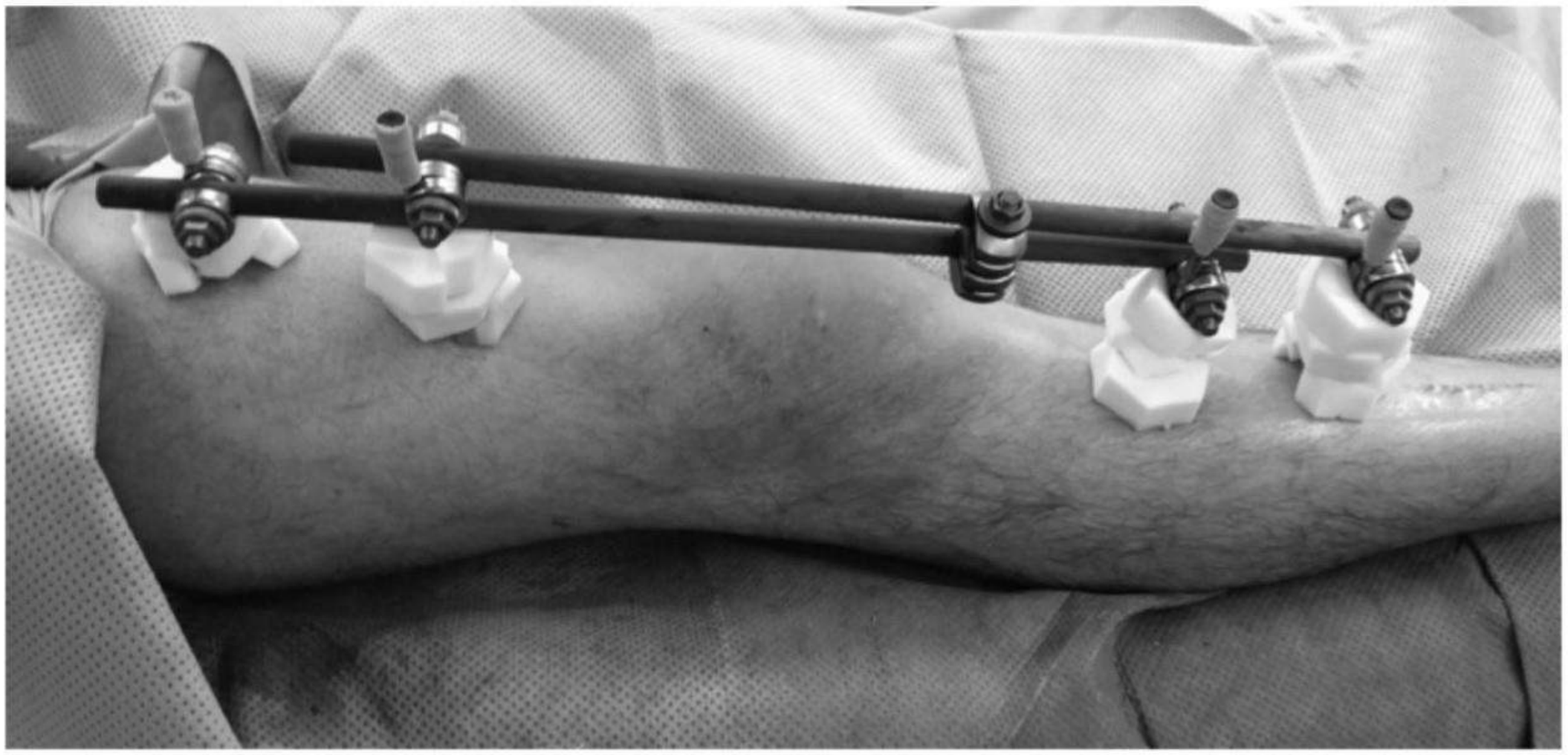

Figure 18.6 External fixator

What does this photograph show?

What are the underlying biomechanical principles that should be followed when applying a basic emergency external fixation device to a mid-diaphyseal fracture?

What methods are available to stiffen a basic external fixation construct?

You mentioned that the effect of adding a second bar in the same plane has a much greater effect on the stiffness in the plane of the fixator than in a plane at right angles to the fixator. What concept explains this?

What does this photograph show?

This is a photograph of an external fixator bridging a knee joint. It is a four-pin and two-bar construct in a single plane. I suspect it is being used to maintain the reduction of a knee dislocation or a fracture about the knee.

What are the underlying principles that should be followed when applying a basic emergency external fixation device to a mid-diaphyseal fracture?

If possible I like to ensure that the fracture is reduced prior to applying the fixator, although this is not always possible. I avoid placing pins inside the zone of injury. I like to ensure that the fixator pins are applied in a manner that avoids bone necrosis—with the use of a pilot drill first in hard cortical bone such as the anterior tibia, and also using cooling saline solution. I also ensure that 'safe corridors' are followed to avoid neurovascular structures.

Whenever I can, I use the near–near far–far principle—the two pins near to the fracture reduce the strain at the fracture site—they make the construct stiffer. The two 'far' pins reduce the stress on the construct—they can be considered as providing two turning couples (each with its corresponding 'near' pin) to resist the rotation of each length of bone that would occur if angulation forces were applied to the fracture.

The bar is placed far enough away from the skin to allow skin care but not so far away as to allow too much flexibility in the pins. I keep adding pins and bars as needed, including in more than one plane, until the construct is stable enough to resist any movement at the fracture in any direction.

What methods are available to stiffen a basic external fixation construct?

The 'near' pins can be placed closer to the fracture—this reduces the working length of the device. The compromise here is the risk of encroaching into the zone of injury. Thicker pins could be used—this increases the stiffness of the pins as a function of the fourth power of their radius. More pins can be added in the same plane as the existing ones—this increases the stiffness proportionally in the same planes as the initial fixator.

The bar of a uniaxial fixator can be moved closer to the bone—this decreases the working length of the pins. A second bar could be added to the existing pins—this gives a much greater proportional increase in stiffness in the plane of the fixator than in a plane perpendicular to the plane of the fixator.

Pins can be added in a different plane with a second bar added to form a V-configuration—this decreases the difference in stiffness between different planes.

In essence, more pins, thicker pins, and using more bars and thicker bars make stiffen the fixator.

You mentioned that the effect of adding a second bar in the same plane has a much greater effect on the stiffness in the plane of the fixator than in a plane at right angles to the fixator. What concept explains this?

The stiffness of a construct can be calculated using the concept of the second moment of area. For a simple beam the stiffness depends on the modulus of elasticity and the second moment of area. For any given material the stiffness of a rectangular beam will increase in proportion to the width and in proportion to the third power of the thickness. The thickness is defined in relation to the direction of bending. The concept can be applied to more complex cross-sections, although the

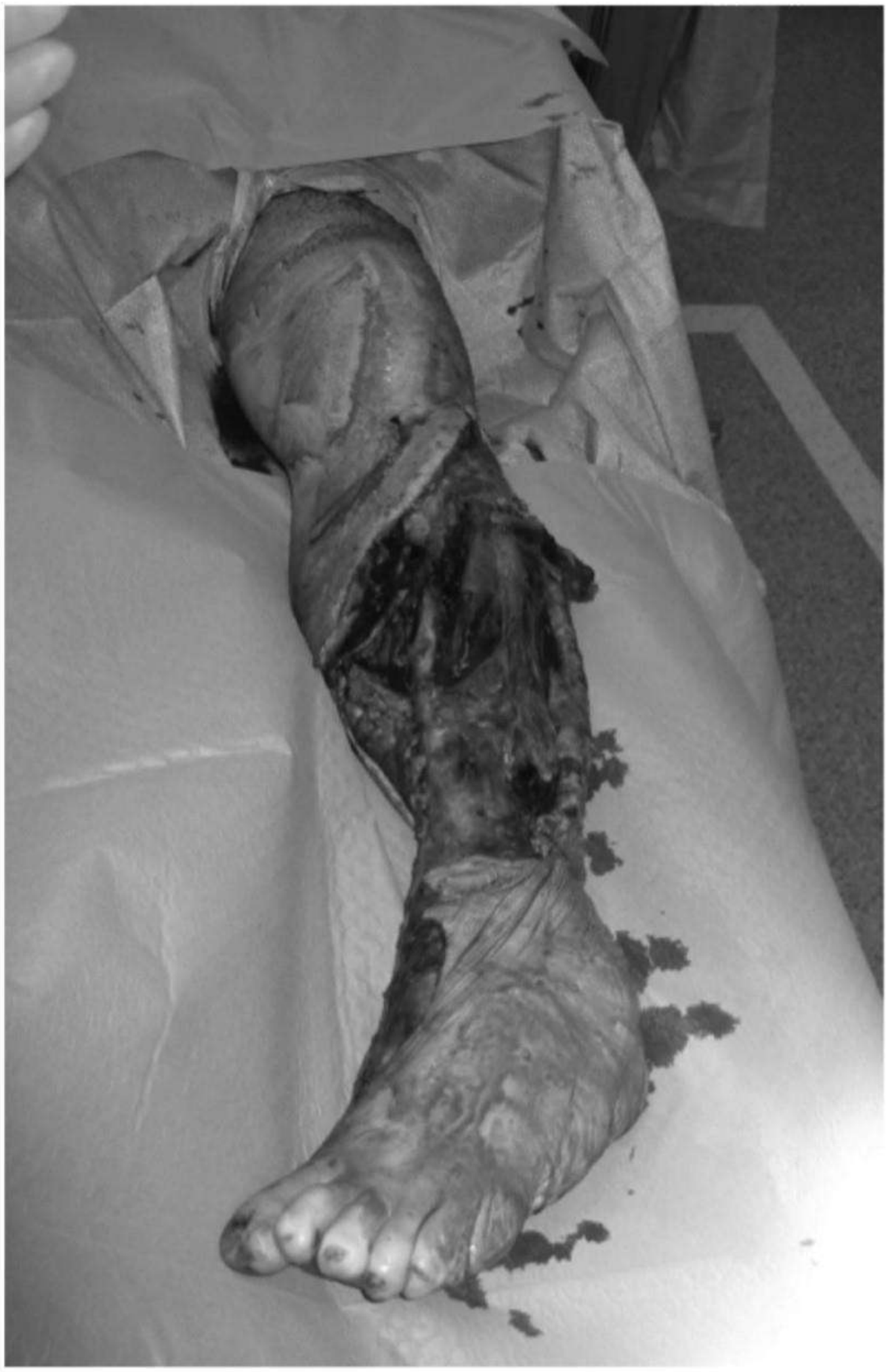

Figure 18.8 Mangled limb

This is a photograph of a mangled extremity. Aside from trauma, what are the other indications for an amputation?

What are the aims of an amputation?

What pre-operative evaluation would you consider?

Describe the surgical technique of a below-knee amputation.

Discuss your aftercare principles for an amputee.

Discuss the complications associated with amputations.

Viva 8 Answers

This is a photograph of a mangled extremity. Aside from trauma, what are the other indications for an amputation?

Other indications would include peripheral vascular disease, infection, tumours, nerve injury (trophic ulceration), and congenital anomalies.

What are the aims of an amputation?

It is essential to retain a maximum level of independent function whilst removing all diseased tissue, and minimizing morbidity and mortality. The amputation should be considered to be the initial stage of a reconstruction procedure aiming to produce a physiological end organ, and as such a multidisciplinary approach is required if I am to be successful in returning the patient to maximal function.

What pre-operative evaluation would you consider?

In the elective setting, after taking a thorough history, I would assess the tissues clinically—feeling pulses and skin temperature. I would use a Doppler ultrasound looking for an ankle–brachial index of more than 0.45, which predicts 90% healing, although this is inaccurate with calcified vessels. A toe systolic blood pressure of 55 mmHg is also predictive of distal healing, as is a minimum transcutaneous PO_2 of 35 mmHg for assured healing. I could use an arteriogram to aid planning. I would like the patient to have a serum albumin of at least 3 g/dl and a white cell count of more than 1500/ml. I would aim for pre-operative control of diabetes, evaluate cardiac, renal, and cerebral circulation, and provide nutritional support for a malnourished patient. As important as clinical evaluation is pre-operative psychological counselling and input from the pain team. In the trauma or acute setting not all of this is always possible, but I would still try to achieve as much of this as I can.

Describe the surgical technique of a below-knee amputation.

I prefer to use a skew flap in order to move the skin incisions away from any areas that bear weight. This relies on skin flaps being half the length of the diameter of the limb, with lateral apices at the level of the bony resection, and 'skewed' to one side of the mid-axis of the limb. A stump of less than 6 cm is not functional so I aim for a minimum of 8 cm, and more if possible. I prefer to use a tourniquet and divide skin, subcutaneous fat, and fascia in the same line as the periosteum of the anteromedial surface of the tibia. I elevate flaps to the level of the amputation and work through each muscle compartment systematically. I identify the superficial peroneal nerve between extensor digitorum longus and peroneus brevis, pull it distally, and divide under tension. Then I divide the anterior tibial vessels and deep peroneal nerve and section anterior muscles 1–2 cm distal of the bony resection. Now the anterior and lateral compartments are prepared, I can section the tibia and bevel the end, and then section the fibula 3 cm proximal to the tibia. In the posterior compartment I must divide the posterior vessels and nerve and fashion a posterior flap which will involve thinning of the muscle bulk and bevelling the muscles. Drill holes in the tibia allow for my gastrocnemius myoplasty. I would then release the tourniquet and obtain haemostasis, and close the wound in layers over a drain.

Discuss your aftercare principles for an amputee.

I prefer a soft dressing for the residuum, and ideally this is taken down within 48 hours. Drains are removed after 24 hours. Any dressings that are used must avoid proximal compression as they risk acting like a venous tourniquet. If the patient has a stiff joint above, or is at risk of a contracture, a

splint may be needed. As soon as the wound is healed, pomade can begin, which is the process of massage to reduce swelling. I would then arrange for early prosthetic fitting. All patients should receive input from psychologists and pain specialists.

Discuss the complications associated with amputations.

Common complications include haematoma, infection, and skin necrosis. Contractures and muscle imbalance can occur if the myoplasty is inadequate. Neuromas can occur if nerves are not cut under tension, or not cut proximally enough. Other neurological complications include phantom sensation and phantom pain. Pain can also be a result of a sharp bony prominence—a failure to bevel the bone ends or leave enough muscle to cover them adequately.

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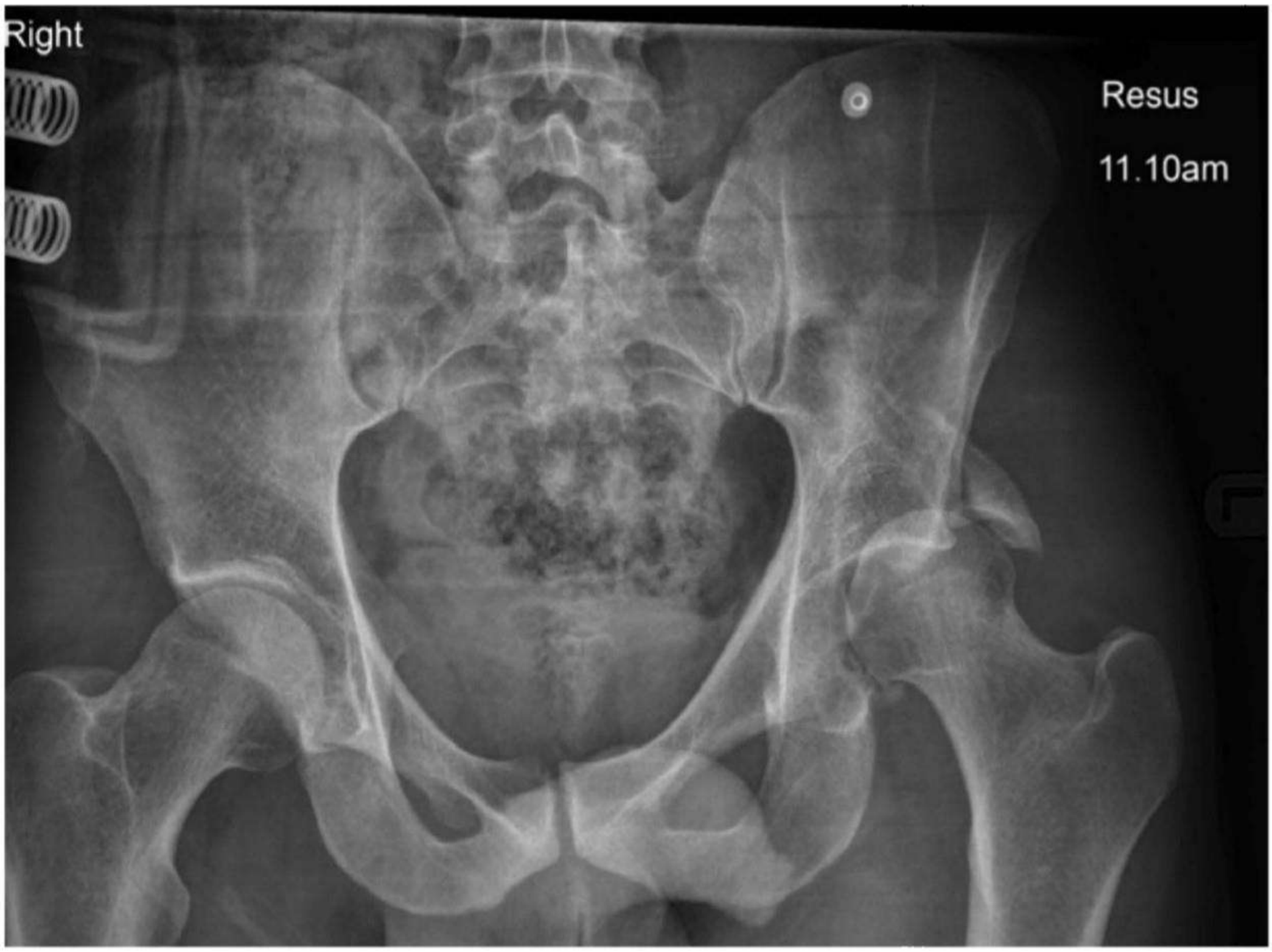


Figure 19.5 Pelvis radiograph

What does this radiograph show?

What would be your initial management of this patient?

Do you know of any guidelines for the management of such injuries?

Having reduced the dislocation, what are the guidelines for subsequent management?

Do you know the evidence upon which the BOAST guidelines are based?

Viva 5 Answers

What does this radiograph show?

This is an anteroposterior image of the pelvis of a skeletally mature patient showing a left hip dislocation with an associated acetabular wall fracture. Ideally I would like to see another view, but this is most likely to be a posterior dislocation and posterior wall fracture.

What would be your initial management of this patient?

This is a high-energy injury with a high probability of associated injuries, and the patient should therefore be assessed and resuscitated following ATLS guidelines. Once life- and limb-threatening injuries have been identified and treated, and the patient is stable, I can move on to the management of this particular injury. Hip dislocations must be reduced urgently, with the neurovascular status before and after reduction assessed and documented. Ideally I would perform the reduction under general anaesthetic, in order to allow an assessment of hip stability following reduction. Having the patient anaesthetized in theatre would also allow me to apply skeletal traction. I would then seek urgent advice from a specialist in acetabular reconstruction and would consider urgent transfer of the patient to my regional pelvic and acetabular centre.

Do you know of any guidelines for the management of such injuries?

Yes, in 2008 the British Orthopaedic Association Trauma Group published a Standard for Trauma (BOAST) for the management pelvic and acetabular fractures.

Having reduced the dislocation, what are the guidelines for subsequent management?

A CT scan must be performed within 24 hours of the injury in order to assess associated fractures and joint congruence, and to exclude entrapment of any bony fragments. However, CT scanning should not be relied upon to predict stability, and those patients whose dislocations have been reduced in A&E should have a formal examination under anaesthesia. The images should be reviewed promptly by an expert in acetabular reconstruction in order to plan definitive treatment, and arrange for urgent transfer if surgery is required. Patients for whom reduction and fixation of the fracture is required should undergo surgery as soon as possible. Ideally this should be within 5 days of the injury, and certainly no later than 10 days post-injury. Chemical thromboprophylaxis should be commenced within 48 hours of the injury, unless there are specific contraindications.

Do you know the evidence upon which the BOAST guidelines are based?

The guidelines are based on a combination of retrospective case series and prospective cohort studies, and are consistent with evolved international consensus.

The British Orthopaedic Association has issued guidance on the management of patients with pelvic and acetabular fractures (BOAST 3).

British Orthopaedic Association (2008). BOAST 3: pelvic and acetabular fracture management. https://www.boa.ac.uk/wp-content/uploads/2014/12/BOAST-3.pdf

Grimshaw CS, Moed BR (2010). Outcomes of posterior wall fractures of the acetabulum treated nonoperatively after diagnostic screening with dynamic stress examination under anesthesia. *J Bone Joint Surg Am*, 92, 2792–2800.

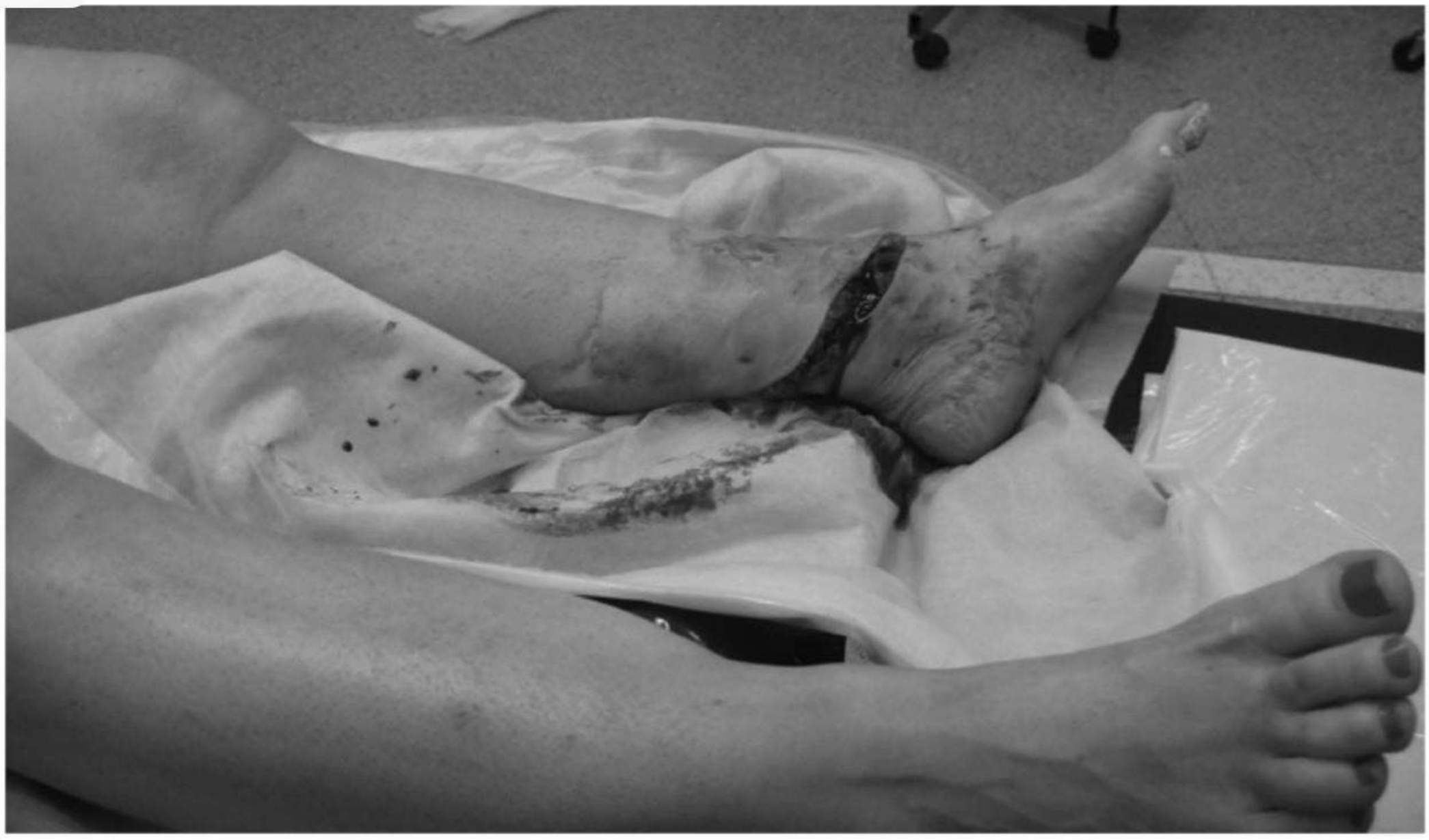


Figure 19.6 Leg wound

What does this photograph show?

How would you define a severe open lower limb injury?

What are the principles of management of open fractures?

What is compartment syndrome?

What are the facial compartments of the lower leg?

Describe how you would perform a fasciotomy of the lower leg.

Viva 6 Answers

What does this photograph show?

This is a clinical photograph showing a significant soft tissue injury to the distal medial left tibia of a female patient. Judging by the nature of the wound and the attitude of the limb, I have a high index of suspicion that this is a severe open distal tibial fracture.

How would you define a severe open lower limb injury?

I would consider both the soft tissue and the bony injury in this situation. From a bony point of view, the fracture pattern in a severe injury would often be either a multifragmentary or segmental tibial fracture. If the fibula is fractured at the same level, this often implies higher energy transfer and greater instability. And of course fractures with bone loss, either from extrusion or after debridement, would fall into the spectrum of severe bony injuries.

Considering the soft tissue injury, any situation where there is swelling or skin loss such that direct, tension-free wound closure is not possible, or a degloving injury, should be treated a severe injury requiring specialist input. If there is a muscle injury that requires excision of devitalized muscle or an injury to one or more major arteries of the leg this would fall into the definition of severe injury, as would any contamination of the wound with marine, agricultural, or sewage material.

What are the principles of management of open fractures?

All open fractures should receive appropriate antibiotics and tetanus prophylaxis as a matter of urgency in the emergency department. Initial limb splintage, clinical photography, and simple saline-soaked barrier dressings are also essential. Once the patient is stabilized, they should be managed in the operating theatre with joint senior orthopaedic and plastic surgery input, with initial soft tissue with/without bone debridement to healthy tissue plus temporizing fixation being performed urgently, but not emergently unless heavily contaminated or associated with significant vascular injury. Definitive soft tissue cover and skeletal fixation should be achieved within 5–7 days.

What is compartment syndrome?

Acute compartment syndrome is defined by a rise in the pressure within an osseofascial compartment above the perfusion pressure of the muscle and nerve within that compartment. Untreated it leads to irreversible muscle ischaemia and death. It is characterized by extreme pain that worsens as pressure within the muscles builds. Neurological dysfunction, pallor, and loss of pulse may be late signs. A high index of suspicion should be had in any high-energy injury or crush injury to the limb.

Chronic compartment syndrome, also known as exertional compartment syndrome, is a different condition, where pressures reached within the compartment are much higher but they only occur transiently on exercise. It is commonly associated with activities such as distance running and is also seen in military recruits.

20 SPINAL TRAUMA

Viva 1 Questions

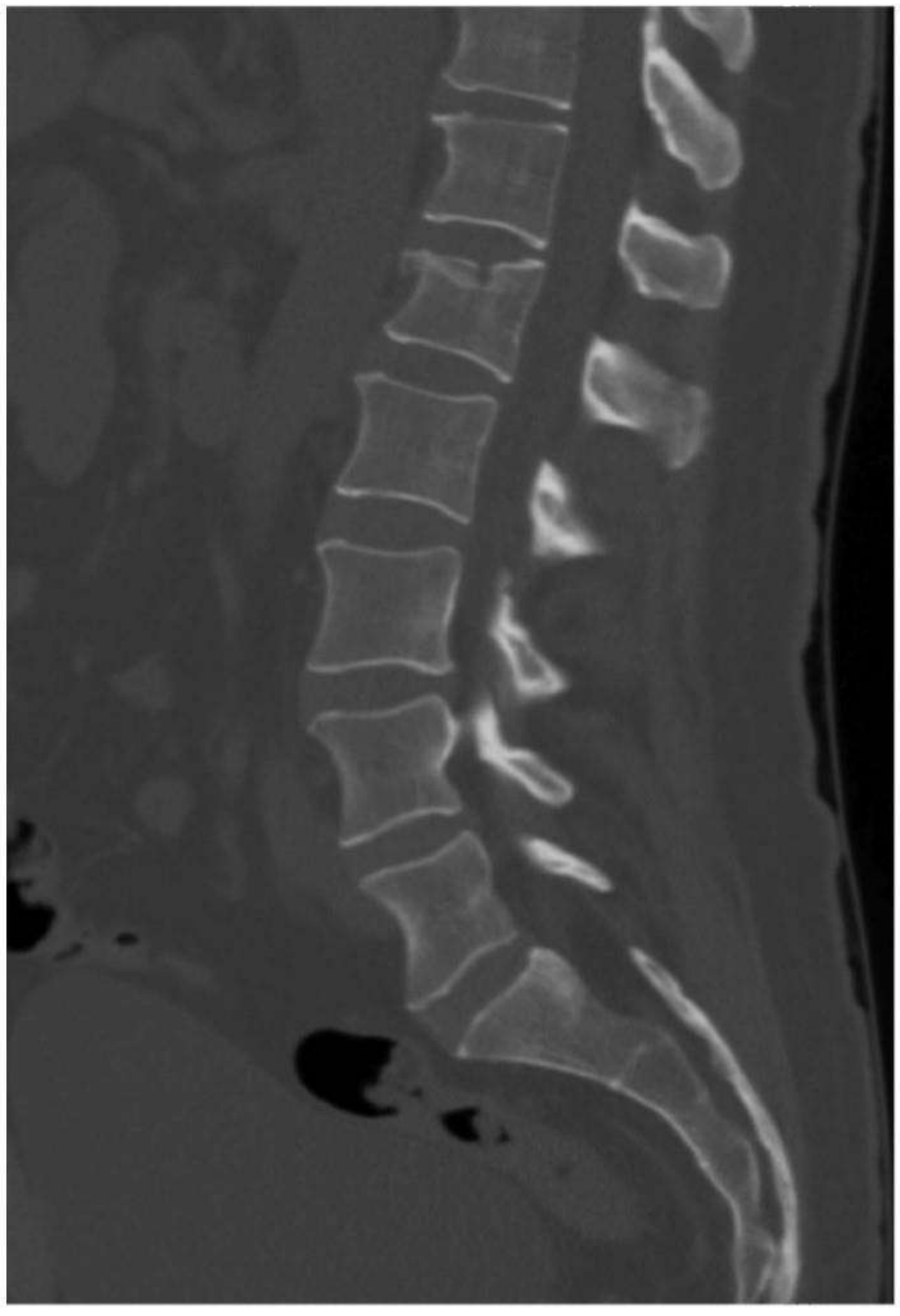


Figure 20.1 Lumbar spine CT

What does this image show?

What further imaging would you like?

MRI shows no injury to the posterior elements. How would you classify this fracture?

How would you manage this patient?

Viva 1 Answers

What does this image show?

This is a sagittal CT slice of a skeletally mature spine. There is an anterior compression fracture of the vertebral body of L1. I would like further imaging in order to fully assess the fracture morphology.

What further imaging would you like?

I would like full CT imaging, including axial and coronal reconstructions, to establish whether there is any further injury within the vertebral body and posterior elements, any scoliosis, lateral displacement, or any other indication that there may be injury to other structures causing instability and in particular the posterior ligamentous complex. In 1990 White and Panjabi defined the concept of instability as 'the loss of the ability of the spine under physiologic loads to maintain its pattern of displacement so that there is no initial or additional neurological deficit, no major deformity, and no incapacitating pain'. Before that, in 1970, Holdsworth had proposed the two-column theory while Denis proposed the three-column theory in 1984. Recent evidence has suggested that the middle column in Denis' theory may be less important than the integrity of the posterior elements as originally described by Holdsworth. The posterior elements, and in particular the posterior ligamentous complex, are vital in preventing post-traumatic kyphosis leading to poor outcomes and neurological deficits. Further imaging including MRI may be needed to exclude injury to the posterior ligamentous complex. MRI has a 100% negative predictive value, and excluding an injury helps to ascertain that the fracture may have stability.

MRI shows no injury to the posterior elements. How would you classify this fracture?

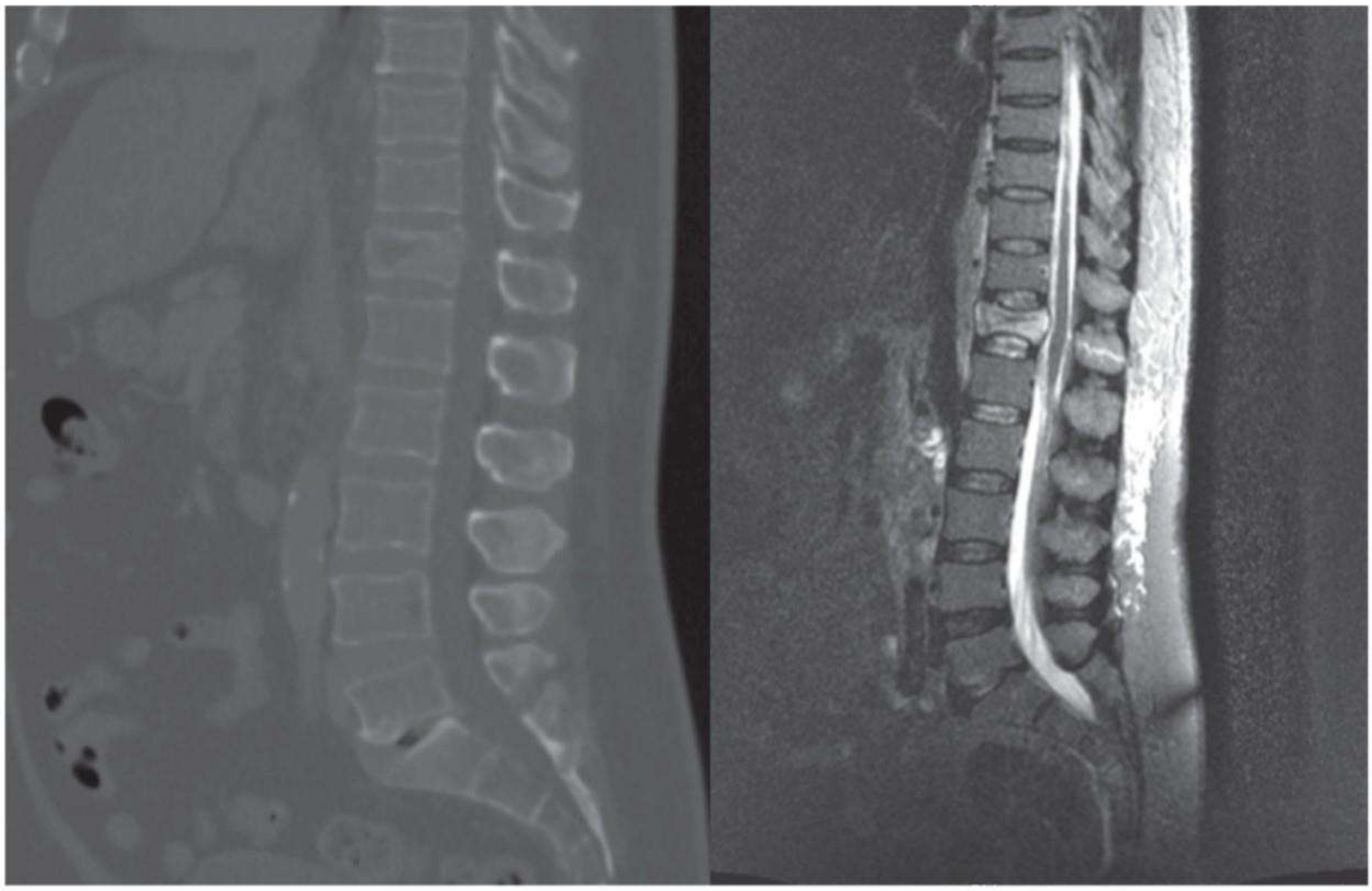
I would classify this fracture according to Denis as a compression fracture. There is less than 20° kyphosis and less than 50% loss of vertebral body height. It is therefore likely to be stable.

How would you manage this patient?

I would brace this patient immediately with an extension orthosis and then perform standing anteroposterior and lateral radiographs to ensure that further kyphosis does not occur under physiological load. If there was no increase in kyphosis I would see the patient weekly for 2 weeks with standing anteroposterior and lateral radiographs, and then at 6 weeks and 3 months when the fracture should have healed. At this point the brace can be removed.

Weninger P, Schultz A, Hertz H (2009). Conservative management of thoracolumbar and lumbar spine compression and burst fractures: functional and radiographic outcomes in 136 cases treated by closed reduction and casting. *Arch Orthop Trauma Surg*, 129, 207–219.

Viva 4 Questions



(a) (b)

Figure 20.4 Lumbar spine CT (a) and MRI (b)

What does the imaging show?

Can you classify this injury?

What would you do next?

What does the MRI show?

What other injuries are common and often missed in such patients?

How would you manage this injury?

Are you aware of any other techniques or approaches?

Viva 4 Answers

What does the radiograph show?

This radiograph shows a T12 flexion–distraction type injury. There is compression anteriorly, but a horizontal distraction fracture through the spinous process posteriorly. There is minimal retropulsion of the posterior vertebral body wall into the spinal canal.

Can you classify this injury?

This is a T12 flexion–distraction injury with compression of the anterior column and distraction of the posterior elements. It is an unstable injury with a risk of developing post-traumatic kyphosis, neurological deficit, and a poor outcome.

What would you do next?

A detailed neurological assessment of the patient is mandatory as spinal cord injury is not uncommon in these patients and may present with mixed upper and lower motor neurone signs if the conus medullaris is injured. I would request a MRI scan to assess the degree of cord compromise and confirm the posterior complex injury.

What does the MRI show?

This sagittal T_2/STIR (short-tau inversion recovery) sequence shows both the anterior flexion fracture and the distraction injury posteriorly causing the horizontal fracture through the spinous process and blood in the posterior structures.

What other injuries are common and often missed in such patients?

Intra-abdominal injuries are present in about 40% of thoracolumbar flexion–distraction fractures. Careful examination and appropriate imaging should be completed in all these patients with frequent re-examination, and a high index of suspicion should be maintained. Multidisciplinary team working with other specialities is of utmost importance.

How would you manage this injury?

Although some clinicians would treat this injury non-operatively there is a risk of post-traumatic kyphosis and neurological compromise. I would treat this injury operatively to enable early mobilization and reduction in the risk of kyphotic collapse. Traditionally, this has been done using a pedicle screw construct two above and two below the level of injury (T10–L2 in this example); however, modern fixed-angle screw constructs have demonstrated successful outcomes whilst preserving motion segments. I would use this method via a posterior midline approach to stabilize T11–L1 maintaining sagittal alignment.

Are you aware of any other techniques or approaches?

Combined anterior and posterior approaches have been described for flexion–distraction injuries.

Minimally invasive techniques have recently been described and used by some surgeons. The perceived advantage of such techniques is to minimize posterior muscle stripping in an area that has already had the posterior elements damaged. Currently there is no evidence that minimally invasive techniques are superior in terms of final outcome measures, but there is evidence that

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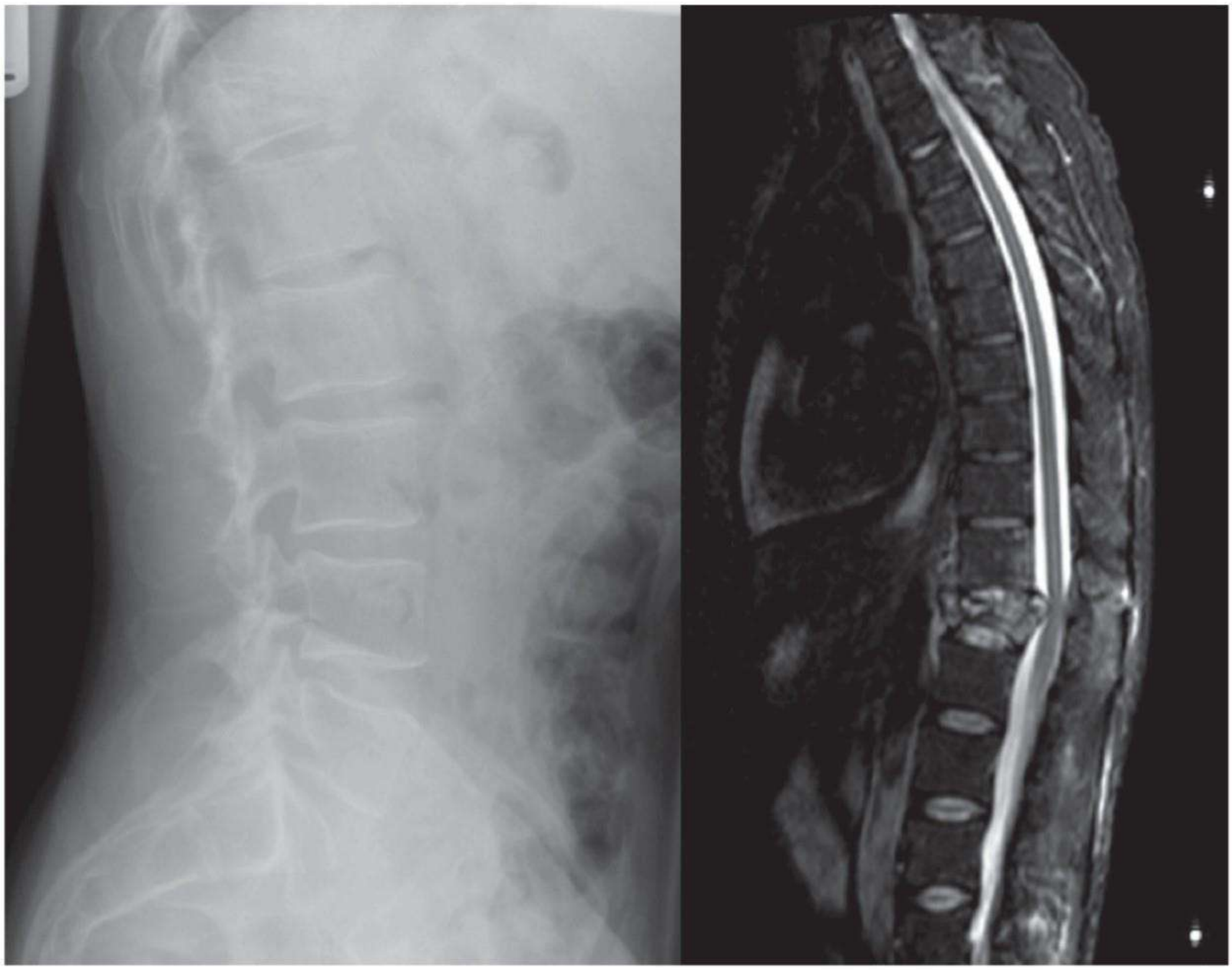

(a) (b)

Figure 20.6 Lumbar spine: radiograph (a) and MRI (b)

What does the radiograph show?

How would you manage this patient?

How would you manage this injury (he has no other injuries)?

He has CT and MRI scans. What does the MRI show?

How should this injury be treated definitively?

Viva 6 Answers

What does the radiograph show?

This lateral plain radiograph shows a T12 fracture with greater than 50% loss of anterior vertebral height. I would be concerned that this is an unstable burst type thoracolumbar injury, which is unstable. It is difficult to interpret, but I would suspect middle and posterior column involvement given the degree of anterior collapse.

How would you manage this patient?

I would use ATLS protocols to assess and manage this patient. Full spinal precautions would be in place, including cervical spine immobilization and strict logrolling until the full extent of all injuries was known.

How would you manage this injury (he has no other injuries)?

CT imaging would be useful as the first modality for assessing fracture morphology and potential instability. However, MRI is also indicated to assess the stability of the posterior ligamentous complex and integrity of the intervertebral disc. This would help determine the most appropriate management.

He has CT and MRI scans. What does the MRI show?

This shows Denis three-column or Holdsworth two-column disruption. There is injury to the anterior and posterior columns with ligamentous incompetence. This injury is unstable and highly likely to collapse into kyphosis with potential neurological sequelae even if the patient is initially neurologically intact. There is significant encroachment into the spinal canal and I suspect the posterior longitudinal ligament is disrupted.

How should this injury be treated definitively?

I would perform posterior instrumented stabilization and decompression. In a fit, healthy patient there may be the option to perform single-level above and below the fracture stabilization. In a patient with osteoporosis/osteopenia two-level above and below stabilization would be the preferred option.

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21 UPPER LIMB—SHOULDER, HUMERUS, AND ELBOW

Viva 1 Questions

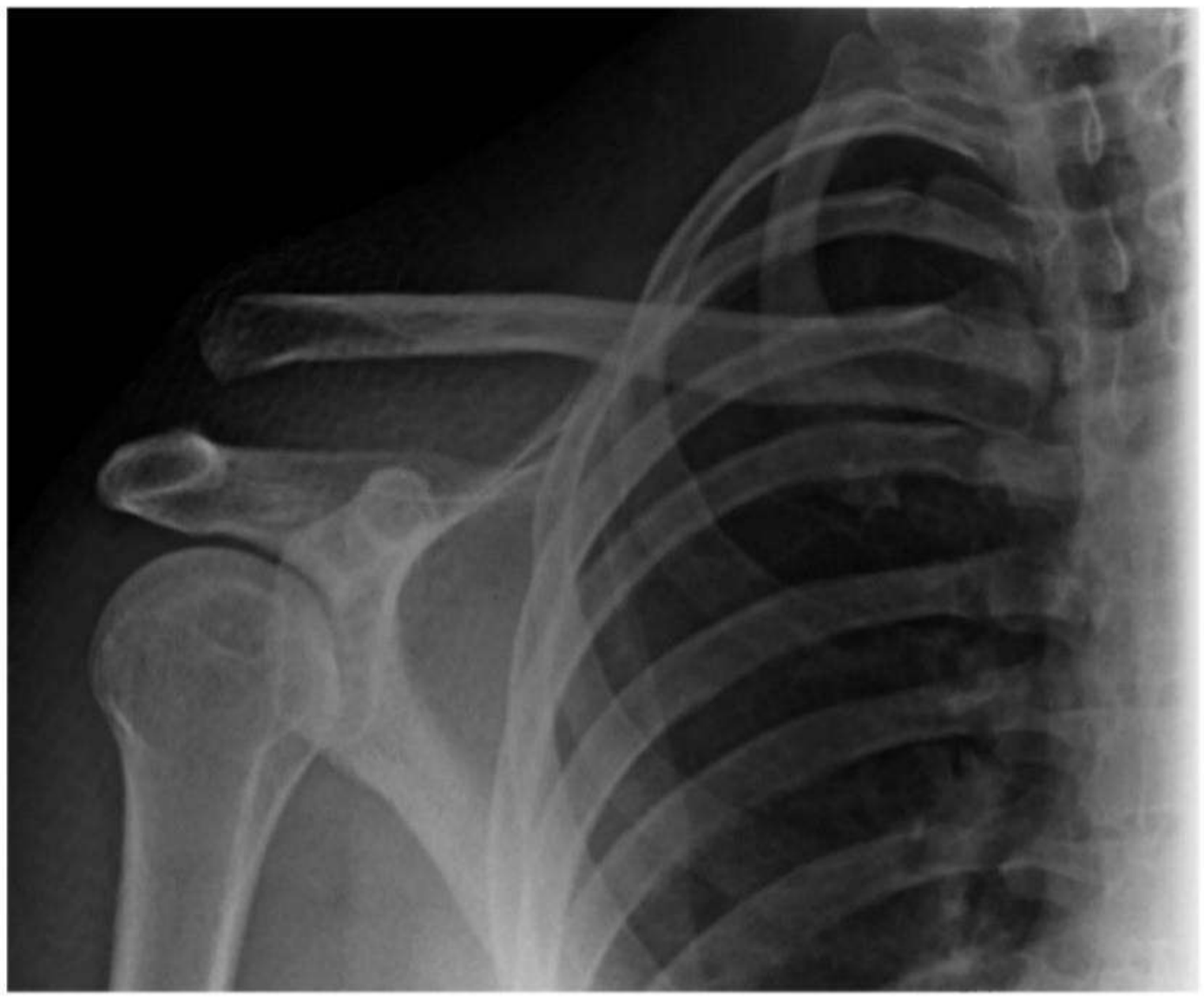

Figure 21.1 Shoulder radiograph

This is a radiograph of a 31-year-old man who fell whilst skiing in France. He has injured his right shoulder. Describe what you see.

It is now 5 days since this injury, and the patient has been treated with an acromioclavicular injection, manual reduction of the dislocation, and taping. How are you going to manage him now?

How would you fix this injury?

Viva 1 Answers

This is a radiograph of a 31-year-old man who fell whilst skiing in France. He has injured his right shoulder. Describe what you see.

This is a weight-loaded radiograph of the right acromioclavicular joint (ACJ) demonstrating a type V right acromioclavicular dislocation.

It is now 5 days since this injury, and the patient has been treated with an acromioclavicular injection, manual reduction of the dislocation, and taping. How are you going to manage him now?

This is type V ACJ dislocation and is best managed with surgical intervention. A type V dislocation involves a complete rupture of both coracoclavicular (CC) ligaments and the acromioclavicular ligament. I would refer him to a colleague with a special interest in shoulder and upper limb surgery. The principles of acute surgical management are to reduce the dislocation and reconstruct the CC ligaments. Various techniques have been described. I am familiar with the following techniques:

- Hook plate. Advantages of the Hook plate are that it is technically simple and does not disrupt the deltoid. Its disadvantages are: the plate has to be removed; osteolysis of the acromion; regeneration/healing of ruptured CC ligaments is required to regain stability; the range of movement must be restricted until the plate is removed at 3–4 months post-operation.
- Reconstruction of CC ligaments with suture/new ligament. The advantages of this technique are that no routine operation is needed to remove metalwork and a full range of movement can be allowed after 6 weeks. Its disadvantages are that it is technically more demanding; there is the possibility of iatrogenic injury to the brachial plexus or coracoid fracture; and the anterior deltoid can be disturbed with an open approach.

How would you fix this injury?

I would fix this through an open approach. In the beach chair position under general anaesthetic I would make a bra strap incision based distally over the coracoid process and proximally 1–2 cm from the ACJ. Raising skin flaps and protecting cutaneous nerves I would open the ACJ and lift a flap of anterior deltoid off the anterior clavicle. I would then expose the coracoid process. [DESCRIBE THE TIGHTROPE TECHNIQUE.] I would protect the patient in a sling for 6 weeks post-operatively.

Andreani L, Bonicoli E, Parchi P, Piolanti N, Michele L (2014). Acromio-clavicular repair using two different techniques. *Eur J Orthop Surg Traumatol*, 24, 237–242.

Grassbaugh JA, Cole C, Wohlrab K, Eichinger J (2013). Surgical technique affects outcomes in acromioclavicular reconstruction. *J Surg Orthop Adv*, 22, 71–76.

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Viva 2 Questions

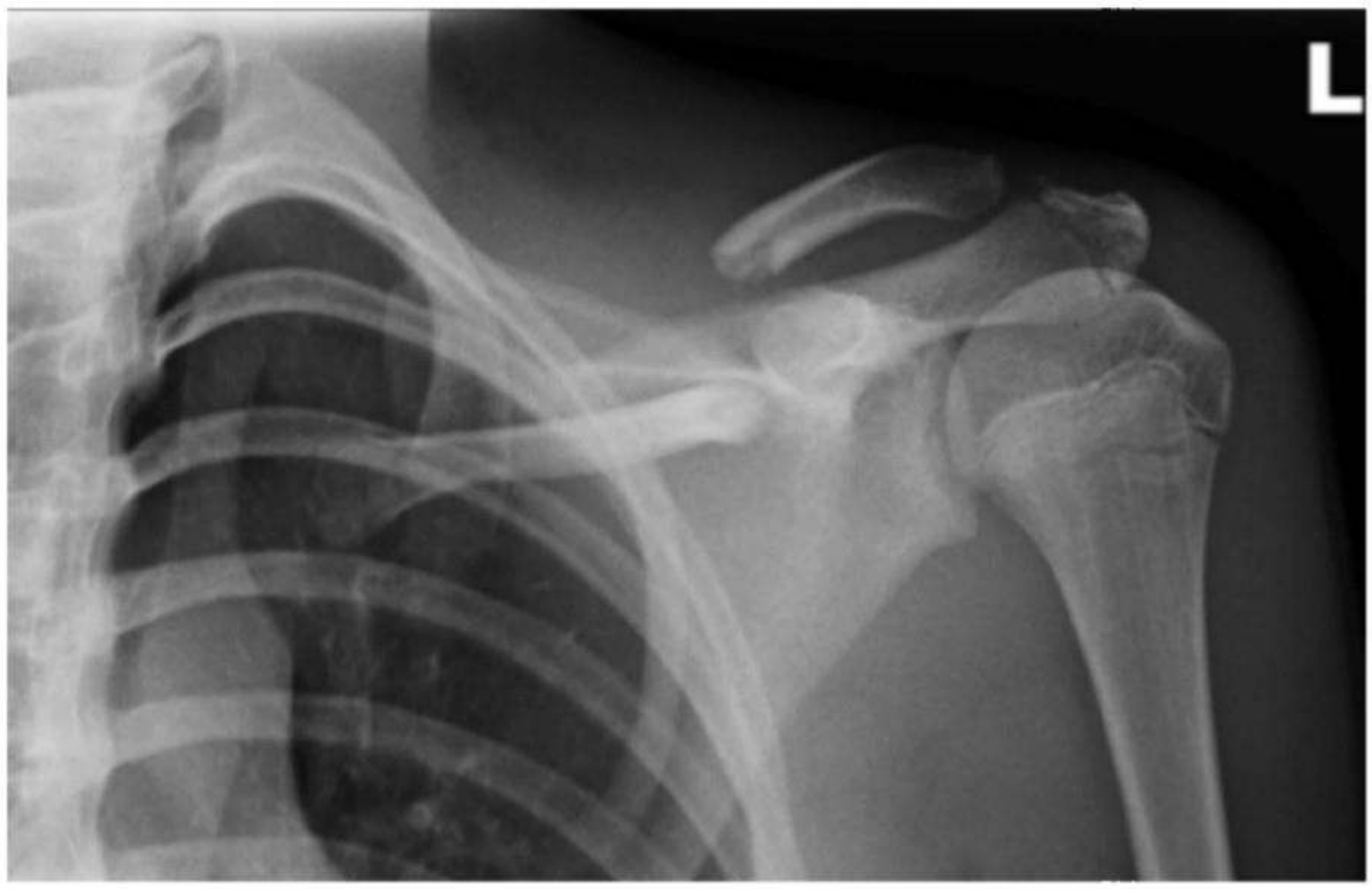

Figure 21.2 Left Shoulder radiograph

What does this radiograph show?

The patient is 15 years old and came off a 3-m jump when mountain biking. He was wearing a helmet and has a GCS score of 15/15. How are you going to manage him?

His neck has been cleared and he is haemodynamically stable. His left hand is warm and well perfused with good pulses. He has some tingling in his hand but the power and sensation are otherwise normal in his hand and elbow. What is your next step?

What are your indications for acute fixation of midshaft clavicle fractures?

What risks would you mention to this patient when obtaining informed consent?

Viva 2 Answers

What does this radiograph show?

This is an anteroposterior radiograph of the left clavicle and proximal humerus of an immature skeleton, demonstrating a fracture of the midshaft of the left clavicle with over 2 cm of displacement. There is no fragmentation at the fracture site. The proximal clavicle appears to be displaced under the coracoid process. I would be concerned about a neurological injury to the brachial plexus in this case.

The patient is 15 years old and came off a 3-m jump when mountain biking. He was wearing a helmet and has a GCS score of 15/15. How are you going to manage him?

I would manage him according to ATLS principles. The mechanism of injury would arouse my suspicions for other pathology such as cervical spine and head injuries. Assuming this is a closed, isolated injury, I would offer analgesia and simple splintage such as a sling, and perform a thorough documented neurological and vascular examination.

His neck has been cleared and he is haemodynamically stable. His left hand is warm and well perfused with good pulses. He has some tingling in his hand but the power and sensation are otherwise normal in his hand and elbow. What is your next step?

Given the position of the fracture this needs to be treated with surgical fixation. The fracture is displaced by over 2 cm and is a result of a high-energy mechanism. It is also displaced under the coracoid. I would like to take him to theatre to reduce the fracture and fix it with internal fixation. This could be done on the next routine trauma list, with no need for emergent out of hours management.

What are your indications for acute fixation of midshaft clavicle fractures?

My indications for fixation of midshaft fractures are open fractures, skin risk over the fracture site, superior displacement/shortening of 2 cm or more, neurovascular compromise, or high-energy injuries with gross fragmentation. I would also consider fixation in multitrauma patients if it allowed early mobilization of other joints/limbs. In 2007 the Canadian Orthopaedic Trauma Society published the results of a randomized controlled study of non-operative treatment compared with plate fixation of midshaft clavicle fractures. It was concluded that those treated with plate fixation had better functional scores and less chance of non-union and malunion at 1-year follow-up compared with those in the non-operative group. There were weaknesses to the study, particularly the large number lost to follow-up in the non-operative group.

What risks would you mention to this patient when obtaining informed consent?

I always consent patients as to the general risks of surgery relating to the anaesthetic, infection, and thromboembolic events. Specific to fixation of clavicle fractures, I would mention the risk of neurovascular damage, numbness below the scar site, prominent hard wear, re-operation to remove metal work, non-union, and hypertrophic scar formation.

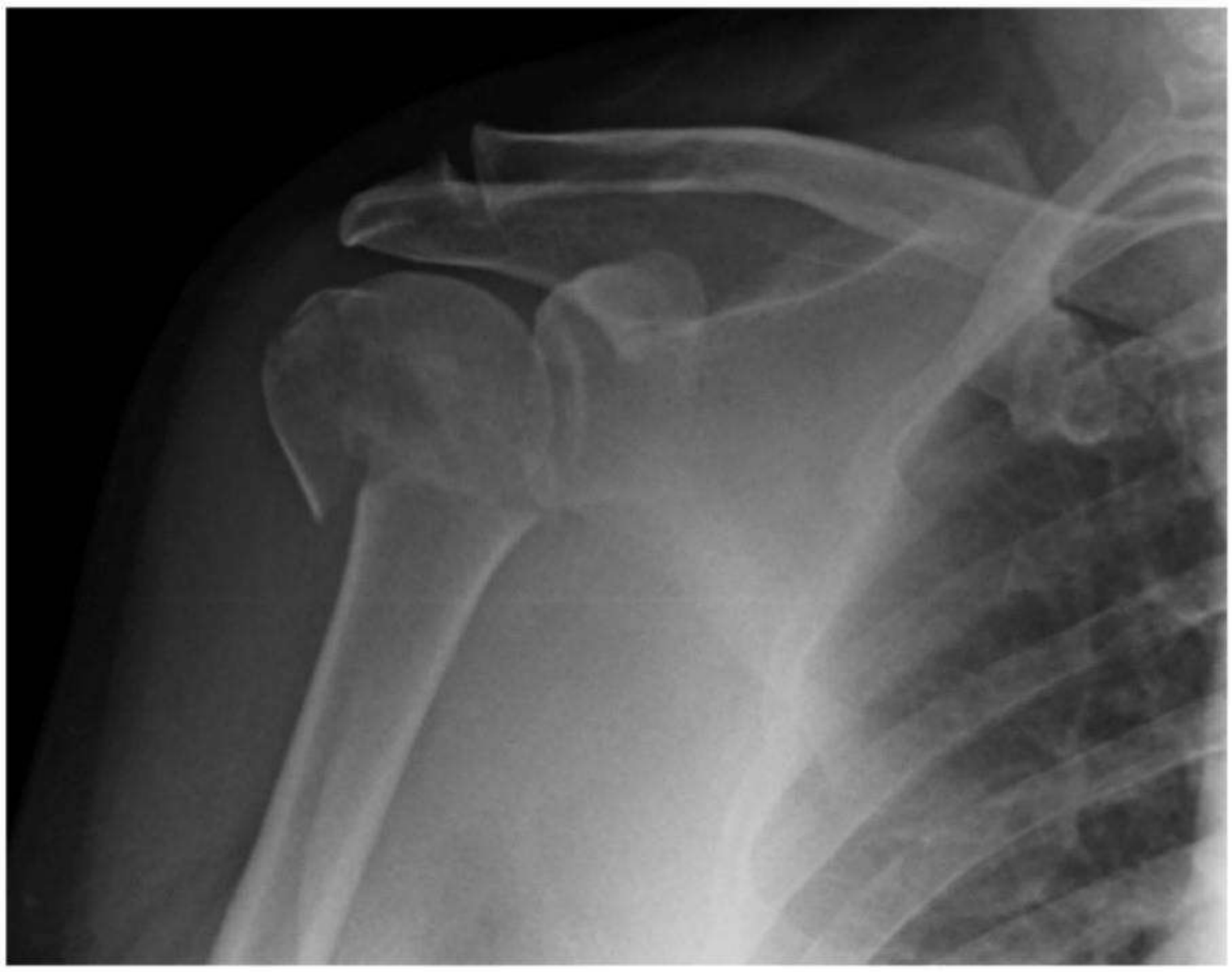

Figure 21.3 Right shoulder radiograph

A64-year-old right-hand dominant man who is normally fit and well falls onto his outstretched right arm. He complains of right shoulder pain, and A&E arrange the radiograph shown here. Describe what you see.

How would you classify this injury?

What are the management options for this patient?

How are you going to proceed?

Describe the deltopectoral approach.

What are the complications of internal fixation of proximal humerus fractures?

Viva 3 Answers

A 64-year-old right-hand dominant man who is normally fit and well falls onto his outstretched right arm. He complains of right shoulder pain and A&E arrange the radiograph shown here. Describe what you see.

This is an anteroposterior view of the right shoulder demonstrating a three- to four-part fracture of the proximal humerus. There is fragmentation medially at the neck of the humerus and the humeral head is in valgus. The humeral head is in joint.

How would you classify this injury?

I would classify this injury according to Neer's scheme. This scheme defines the fracture according to the number of osseous parts (Codman's parts) that are displaced. Neer classified displacement as separation >1 cm or 45° angulation between the fragments. This is at least a three-part fracture with displacement of the greater tuberosity and humeral head from the shaft of the humerus. I cannot comment on the position of the lesser tuberosity on this radiograph.

What are the management options for this patient?

The position of the fracture on these views is unacceptable and I would treat this patient with ORIF using a pre-contoured periarticular locking plate. However, the Level 1 evidence for operative treatment and type of operative treatment is sparse. A Cochrane review published in 2012 states that there is insufficient evidence to suggest that surgery is better for three- or four-part fractures of the proximal humerus, although surgery is associated with a requirement for further surgery. It also stated that there is not enough evidence to suggest a best method of treating fractures of this type. However, there are Level 3 studies demonstrating that locking plates have good function with this type of injury, and from my experience I would treat this with a locking plate.

How are you going to proceed?

Assuming that the patient is adequately prepared for theatre, including being starved, marked, consented, and having had the appropriate anaesthetic investigations, I would place the patient in the beach chair position. I would perform the procedure through the deltopectoral approach under image intensification.

Describe the deltopectoral approach.

The deltopectoral approach is an extensile approach based proximally on the coracoid process and distally along the deltopectoral groove. I would mark out the anterior acromion, distal clavicle, and coracoid process. The incision is 10–15 cm based proximally on the coracoid following the deltopectoral groove. The internervous plane . . . [see Chapter 17].

What are the complications of internal fixation of proximal humerus fractures?

The complications of proximal humeral fractures are varus malunion, AVN, screw perforation, reoperation, and infection. A 2011 review article by Sproul et al. published in *Injury* suggested that the complication rate approaches 49% if all of these are included as complications.

Viva 4 Questions

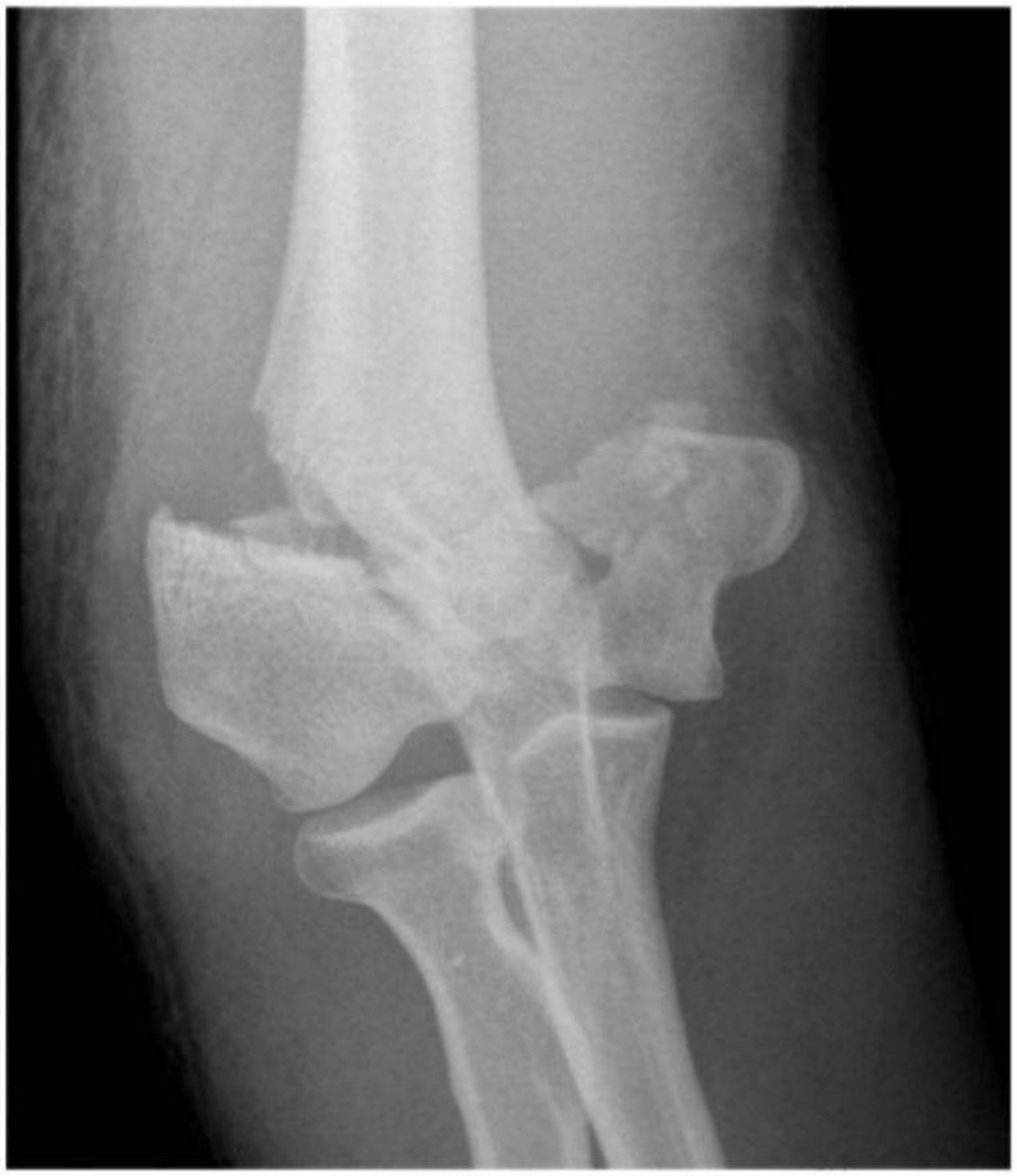

Figure 21.4 Radiograph of elbow

What does this radiograph show?

Can you classify this fracture?

What are the principles of management for this type of injury?

What are the short- and long-term consequences of distal humeral fractures?

What does this radiograph show?

This is an anteroposterior view of the distal humerus of a skeletally mature patient, showing a displaced intra-articular distal humeral fracture. There is some comminution, especially of the medial column and possibly of the articular surface itself, although the joint appears to be in two principal fragments.

Can you classify this fracture?

Both column distal humeral fractures have been classified by Jupiter. Although displaced, this is most likely a type C fracture as it has the characteristic Y-shaped pattern. Other classification systems include the AO-OTA classification which in this case would be a 13-C fracture. Further imaging in the form of lateral radiograph and CT views would be mandatory in this case for planning any surgical procedure.

What are the principles of surgical management for this type of injury?

Although some distal humeral fractures may be treated non-operatively, this would only be the case for undisplaced fractures and injuries in those patients who don't warrant surgery. In my department these injuries may get referred on to the specialist trauma centre, but the principles of management are to achieve anatomical articular reduction and preserve the blood supply while providing rigid stable internal fixation that is strong enough to withstand early functional motion.

Several surgical approaches have been described: triceps sparing is useful for extra-articular or simple articular fractures; triceps splitting is useful for exploiting skin lesions; and triceps reflecting preserves triceps function in the event of need for total elbow replacement. Olecranon osteotomy is still considered to be the approach best suited for articular visualization, but it risks non-union and involves prominent hardware, and should also be avoided if elbow arthroplasty is likely in the future.

Locking plates appear to provide optimal biomechanical fixation, and recent cadaveric studies have shown that parallel and perpendicular plates both provide adequate biomechanical strength. Postoperative immobilization is associated with stiffness and should generally only be used to allow the soft tissues a chance to heal for 7–10 days. Early active and active-assisted exercises should be undertaken. It is very important to be sure that the fixation provided will be sufficient to allow early mobilization.

Total elbow replacement should be considered in patients with osteoporosis if there is any doubt as to whether fixation will be possible. In patients over 65 the results of total elbow replacement are more predictable and better at 2 years, and there is less need for re-operation.

What are the short- and long-term consequences of distal humeral fractures?

Unfortunately there is generally a residual degree deficiency of flexion, extension, and pronosupination, with figures quoted for the flexion contracture of up to 25°. Other complications of distal humeral fractures include failure of fixation, non-union, infection, and heterotopic ossification.

Doornberg JN (2007). Surgical treatment of intra-articular fractures of the distal part of the humerus: functional outcome after 12–30 years. *J Bone Joint Surg Am*, 89, 1524–1532.

Viva 5 Questions

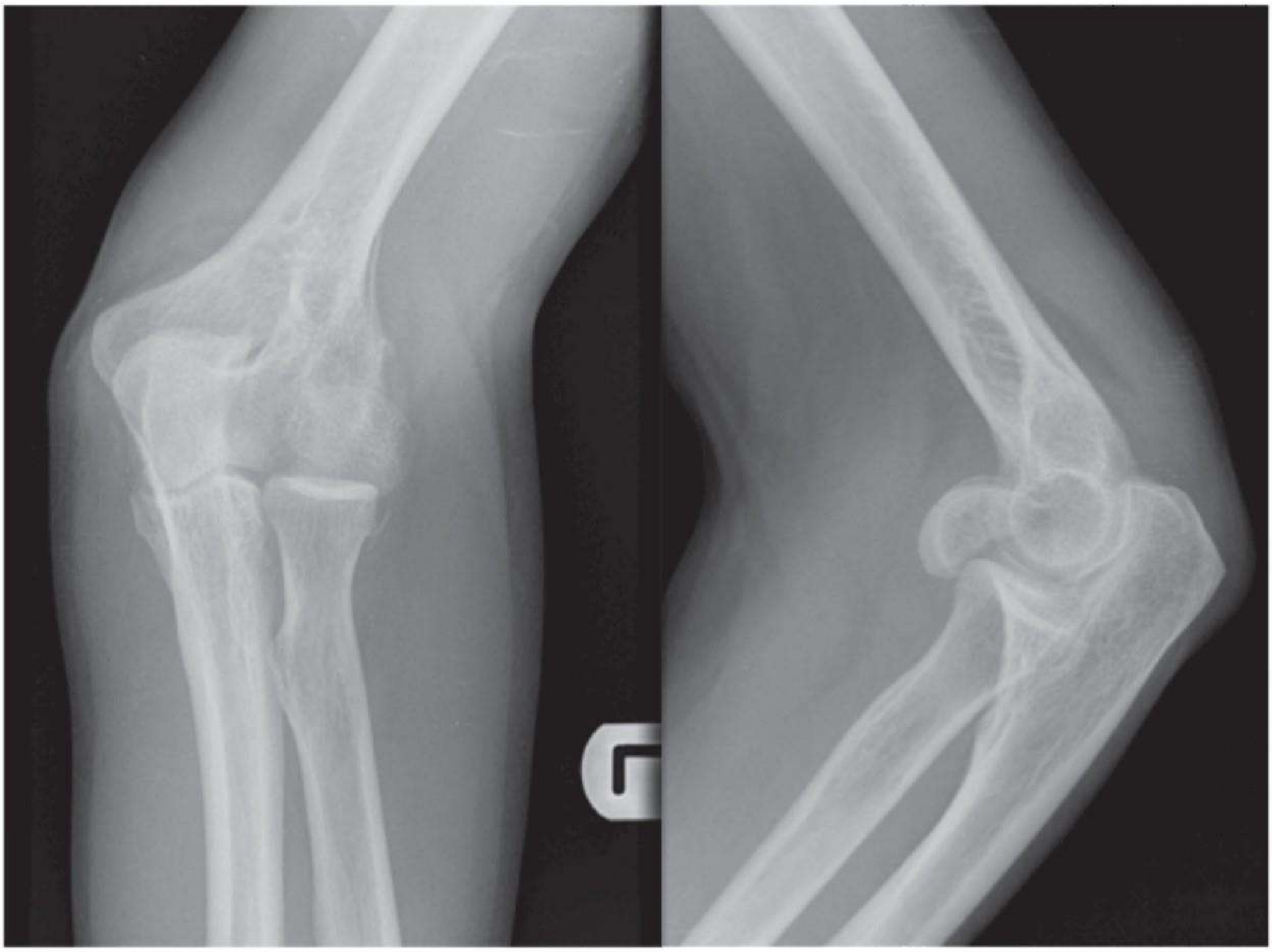

Figure 21.5 AP and lateral elbow radiograph

What do these radiographs show?

Can you classify this fracture?

What is the significance of this injury?

What are the principles of management for this injury?

What are the short- and long-term consequences of capitellar fractures?

Viva 5 Answers

What do these radiographs show?

These are lateral and anteroposterior views of the distal humerus of a skeletally mature patient showing a displaced capitellar fracture.

Can you classify this fracture?

Capitellar fractures were originally subdivided into three groups by Bowner: type I, a complete fracture; type II, a superficial osteochondral fragment also known as the lesion of Kocher Lorenz; and type III, a comminuted capitellar fracture. Types I and II usually have coronal shear patterns. McKee modified the classification to include a type IV, which is where the trochlear is fractured along with the capitellum, also in a coronal shearing pattern.

This is a type I fracture as the capitellum is one whole fragment as per Jupiter's original description.

What is the significance of this injury?

This shows the double shadow or 'double bubble' sign which represents a coronal shear fracture of the distal humerus and involves most of the anterior joint surface. As such it is quite possible in this case that some of trochlear may be involved, making it a type IV fracture. However, it seems the trochlear on the anteroposterior projection is intact. I would like further imaging in the form of a CT as this would be helpful in this case to aid diagnosis and for planning treatment.

What are the principles of management for this injury?

Non-operative treatment can be used for minimally displaced type I fractures and type II fractures where displacement is less than 2 mm. Operative treatment in the form of ORIF is required for displaced fractures, and fixation is with headless screws. Reduction must be anatomical and fixation must be rigid and stable as these are articular fractures.

What are the short- and long-term consequences of capitellar fractures?

Complications of this injury include non-union after ORIF (1–10%), heterotopic ossification, AVN, ulnar nerve neuropathy, and post-traumatic arthrosis.

Dubberley J, Faber K, MacDermid J, Patterson S, King G (2006). Outcome after open reduction and internal fixation of capitellar and trochlear fractures. *J Bone Joint Surg Am*, 88, 46–54.

Viva 6 Questions

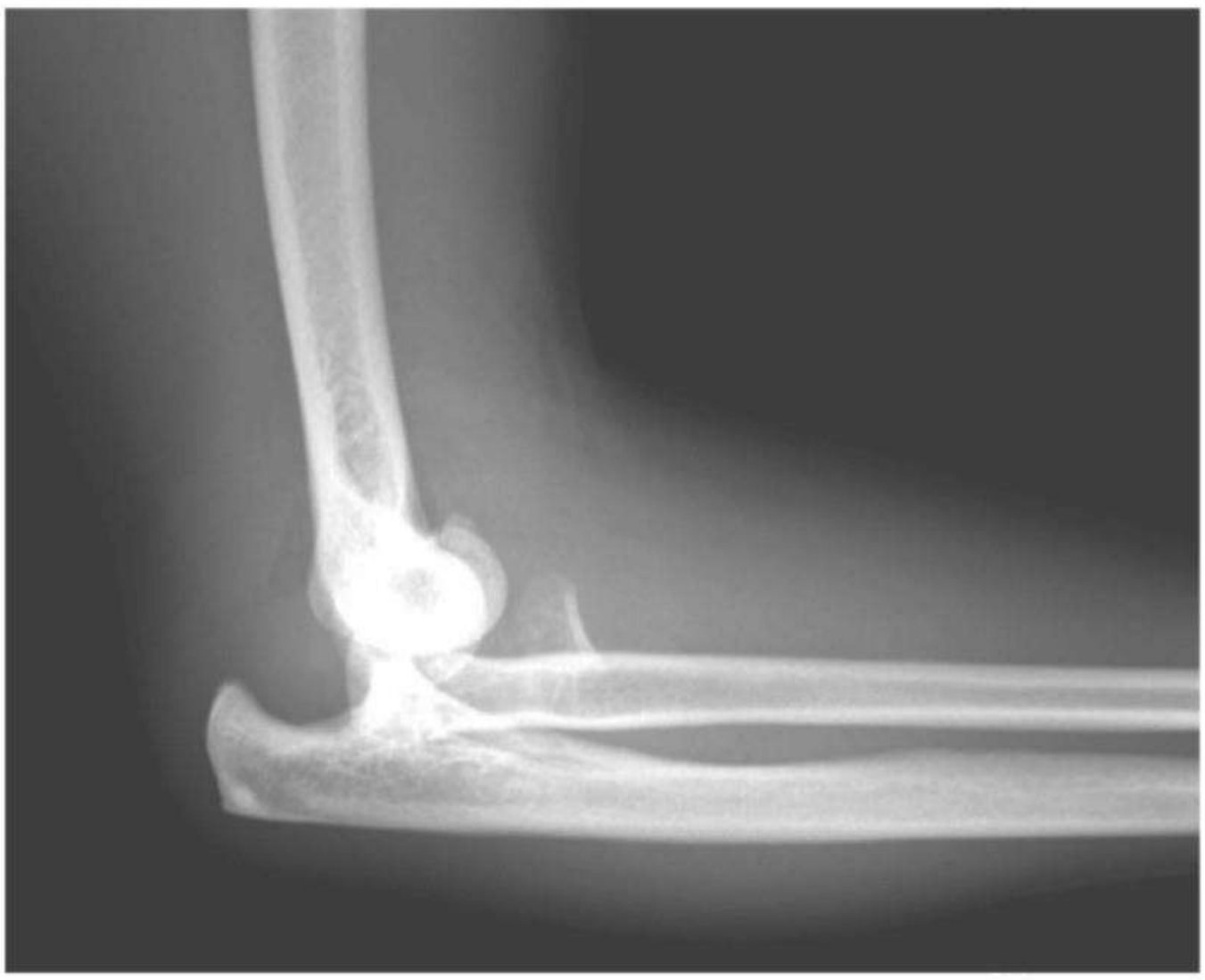

Figure 21.6 Lateral elbow radiograph

What does this radiograph show?

Which structures normally contribute to stability of the elbow?

How do most elbow dislocations arise?

How would you manage this isolated closed injury in the emergency department?

CT confirms the radial head fracture and a coronoid tip fracture. What are the principles of management for this type of injury?

What surgical approach would you use, and how would deal with the radial head?

What would your rehabilitation regime be?

Viva 6 Answers

What does this radiograph show?

This lateral elbow radiograph demonstrates a posterior elbow fracture dislocation. I'd like to see another view, but it looks as though there is a radial head fracture and possibly a fracture of the coronoid tip.

Which structures normally contribute to stability of the elbow?

While we often think of static and dynamic restraints, O'Driscoll of the Mayo Clinic has described a fortress of stability, which considers both static and dynamic restraints as primary or secondary stabilizers. Primary restraints include the ulnar–humeral articulation and the collateral ligament complexes. Secondary restraints include the radiocapitellar articulation and the common flexor and extensor origins. The capsule is also important.

How do most elbow dislocations arise?

Most elbow dislocations occur as a result of axial load, valgus force, and forearm supination. Hori described a circle of injury progressing from lateral to medial. The first structure to fail is the lateral collateral ligament complex. The radial head then dislocates or fractures and the continuing force then tears the capsule (front and back) from lateral to medial as the elbow hinges out of joint. The final structure to fail is the medial collateral ligament.

How would you manage this isolated closed injury in the emergency department?

Having taken a brief history from the patient, I would document whether the distal neurovascular supply is intact. This injury requires urgent reduction and I would do this under sedation in the emergency department. I would then re-examine the patient for neurovascular status, place the elbow in a plaster backslab at 90°Flexion, and obtain further radiographs to confirm reduction. The patient would be admitted. I would like post-reduction films and a CT scan to help me plan my approach and whether or not I expect to fix or replace the radial head. I strongly suspect the coronoid is also fractured here.

CT confirms the radial head fracture and a coronoid tip fracture. What are the principles of management for this type of injury?

This represents a terrible triad injury—an elbow dislocation with a fracture to the coronoid and the radial head. These are unstable and, unlike simple dislocations, they are best treated with early surgery. I am careful to emphasize the gravity of this injury to the patient when gaining consent for surgery as long-term stiffness and reduced function are common.

My goals in surgery are to restore the bony anatomy of the elbow and address the capsule-ligamentous injuries such that early mobilization of the elbow is possible. I would adopt a stepwise approach to restoration of anatomy as described by McKee and colleagues in 2004 from their series of 36 cases. First, the bony anatomy, then the lateral ulnar collateral ligament, then, rarely, the MCL and/or hinged external fixation.

Ideally I would try and fix this fracture, but I would have a radial head replacement available. The coronoid tip is likely to be a small fragment that doesn't compromise the integrity of the ulnar–humeral articulation. While anatomical reduction of this fragment is not required, it may still need

to be re-attached if it carries a portion of the anterior capsule and confers stability on fixation. The ligaments, and sometimes the medial ones, need repairing.

What surgical approach would you use, and how would deal with the radial head?

Having prepared the patient for surgery I would place him lateral with the injured arm over a bolster and fit a high-arm tourniquet. I would confirm I could achieve fluoroscopic imaging before scrubbing. I'd use an extended Kocher's posterolateral approach between the anconeus and the ECU . . . [see Chapter 17].

What would your rehabilitation regime be?

Having optimized stability intraoperatively I'd allow the wound and soft tissues to rest in a cast for 1 week. I would then encourage active elbow flexion in a hinged brace. This will initially be locked at 60° of flexion. After two more weeks I would allow extension to 30°Flexion and at 6 weeks full extension would be allowed. There should be no resistance training for 3 months to allow healing of the ligaments and capsule.

O'Driscoll SW, Jupiter JB, King GJ, Hotchkiss RN, Morrey BF (2001). The unstable elbow. Instr Course Lect, 50, 89–102.

Viva 7 Questions

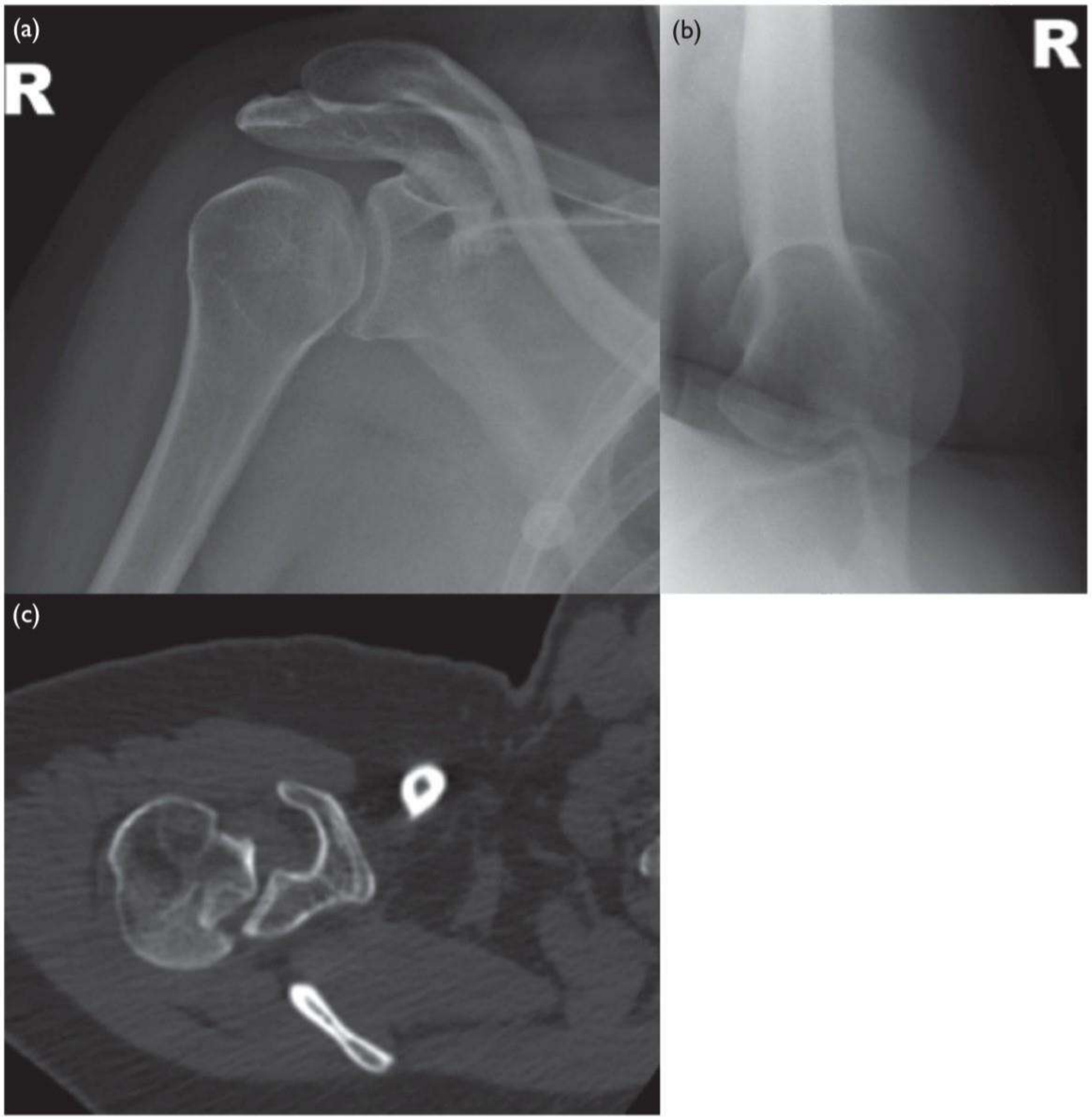

Figure 21.7 Right shoulder (a), (b) plain radiographs; (c) axial CT

Describe these images.

What would you expect to find upon physical examination?

What aspects of the clinical history would raise one's suspicion of such an injury?

How would you definitely treat such an injury?

ribe these images.

These are plain radiographs of a skeletally mature right shoulder and an axial CT through the same shoulder. The anteroposterior (AP) view in the plane of the scapula demonstrates the 'light bulb' appearance of the humeral head consistent with a posterior dislocation of the glenohumeral joint. The axillary lateral view demonstrates a 'locked' posterior glenohumeral dislocation with the humeral head impacted on the glenoid rim creating a depression in the anteromedial aspect of the humeral head, termed a 'reverse Hill–Sachs lesion'. The axial CT image demonstrates a defect of approximately 40% of the articular surface of the head.

What would you expect to find upon physical examination?

On examination the patient would typically hold his or her arm in internal rotation in the adducted position. The arm is locked in internal rotation and neither active nor passive external rotation is possible from this position. Rowe and Zairns described a test in which there is inability to supinate the forearm when the arm is flexed forwards because of the internal rotation deformity of the shoulder. There is increased prominence of the coracoid process anteriorly and of the humeral head posteriorly.

What aspects of the clinical history would raise one's suspicion of such an injury?

This type of injury is usually caused during an epileptic seizure, an electric shock, or by trauma such as a fall on an outstretched arm. In the case of involuntary muscle contraction, the strong internal rotators (latissimus dorsi, pectoralis major, subscapularis, and teres minor) simply overpower the weak external rotators (infraspinatus and teres minor). Clinical suspicion of such an injury is imperative, because although they account for less than 2% of all dislocations of the shoulder most are missed on initial examination. Djurdjevic reported that in a series of 24 patients with posterior dislocation, 21 had not been recognized initially.

How would you definitely treat such an injury?

Appropriate management of a posterior glenohumeral dislocation depends upon the size of the defect, the duration of the dislocation, and the age and activity of the patient. Non-operative treatment must be considered for patients with uncontrolled seizures or any patient unable to comply with a post-operative rehabilitation programme. Gerber recommends 'supervised neglect' for elderly patients with limited demands on the affected shoulder, an acceptable functional range of motion, and a normal contralateral shoulder.

Reduction of acute traumatic posterior dislocation should be carried out under general anaesthetic as soon as possible. Under general anaesthetic and muscle relaxation gentle reduction is attempted by flexion and adduction with axial traction on the arm. If the humeral head is 'locked' upon the glenoid rim, gentle internal rotation may be applied prior to lateral traction to unlock the humeral head from the glenoid rim. Once unlocked the humerus is gently externally rotated. If closed reduction is unsuccessful open reduction is performed under the same general anaesthetic. Such an injury would be deemed chronic when the duration is longer than 3 weeks and closed reduction is usually impossible.

A small impression defect of up to 25% of the articular surface of the head can be treated by closed or open reduction. If the shoulder is unstable, a transfer of the upper one-third of the subscapularis

performed via a deltopectoral approach. A medium defect, between 25% and 50% of the articular surface, can be treated by transfer of the lesser tuberosity. McLaughlin described the transfer of subscapularis for a defect between 20% and 40%. Rotational osteotomy of the surgical neck of the humerus may be considered to ensure the defect remains anterior to the glenoid throughout the entire range of motion. The defect may be filled with an allograft from the femoral head which is contoured to fit the segmental defect and restore sphericity of the head. A large defect of more than 50% of the articular surface can be treated by shoulder arthroplasty. Retroversion of the humeral component should be decreased from approximately 35° to 20°.

Cicak N (2004). Posterior dislocation of the shoulder. *J Bone Joint Surg Br*, 86-B, 324–332.

Djurdjevic D (2003). Unrecognised posterior dislocation of the shoulder. Masters Thesis, Zagreb.

Gerber C. Chronic, locked anterior and posterior dislocation. In: Warner JJP, Ianotti JP, Gerber C (eds) (1997). *Complex and Revision Problems in Shoulder Surgery*, pp.99–113. Lippincott-Raven, Philadelphia, PA.

McLaughlin HL (1952). Posterior dislocation of the shoulder. *J Bone Joint Surg Am*, 34-A, 584–590.

Robinson MC, Aderinto J (2005). Posterior shoulder dislocations and fracture-dislocations. *J Bone Joint Surg Am*, 87-A, 639–650.

Rowe C, Zairns B (1982). Chronic unreduced dislocation of the shoulder. *J Bone Joint Surg Am*, 64-A, 494–505.

Petersen SA (2000). Posterior shoulder instability. *Orthop Clin North Am*, 31, 263–274.

Viva 8 Questions

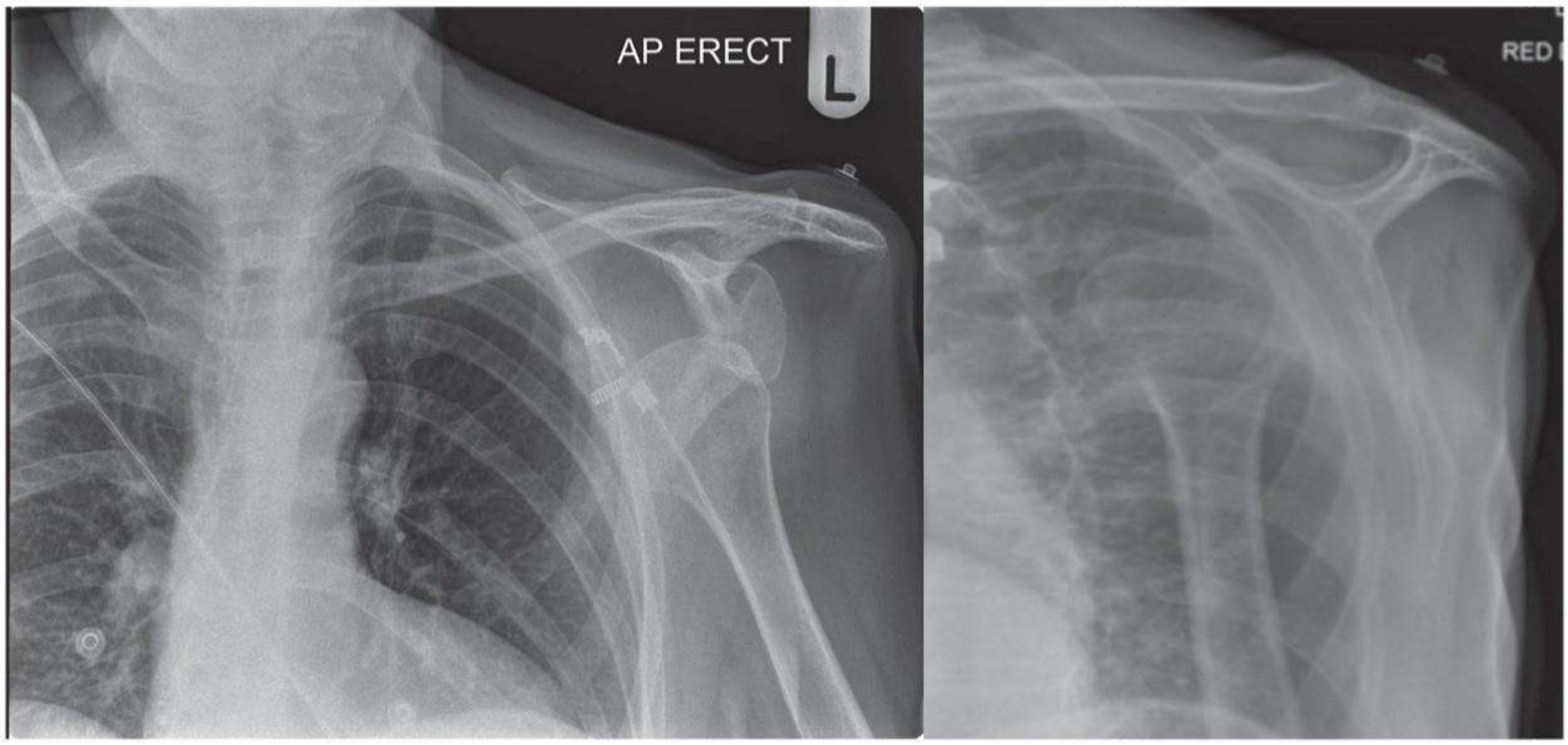

Figure 21.8 Left shoulder radiographs

A 42-year-old man falls off a 1.2-m fence, injuring his left shoulder. He is brought to A&E and complains of pain in his left shoulder. Describe the radiograph.

You are called to assess him in A&E. Outline your management of his shoulder.

Do you know how to reduce a dislocated shoulder?

What are the risks of manipulation?

What structures may be preventing a stable reduction?

Assuming you have achieved a satisfactory reduction, how do you plan to manage this patient?

Viva 8 Answers

A 42-year-old man falls off a 1.2-m fence, injuring his left shoulder. He is brought to A&E and complains of pain in his left shoulder. Describe the radiograph.

This is an anteroposterior view of the left shoulder. There is an anterior dislocation of the left glenohumeral joint. An axial image is useful to confirm the direction of dislocation and my diagnosis, although from this radiograph that is not necessary.

You are called to assess him in A&E. Outline your management of his shoulder.

I would assess this patient using an ATLS approach. As his has fallen off a fence he may have other injuries, some of which could be life threatening. Assuming this is an isolated injury, my primary goal would be to reduce the left glenohumeral joint. I would perform a neurovascular examination of the patient before any reduction was attempted and document the findings in the notes. Specifically, I would check for axillary nerve sensation over the regimental badge area and musculocutaneous nerve sensation over the lateral edge of the forearm. I would also test distal pulses and medial, ulnar, and radial nerve sensation and document all my findings.

Do you know how to reduce a dislocated shoulder?

There are several methods for reducing an anterior dislocation of the glenohumeral joint. The principles involve applying gentle traction or leverage with adequate pain relief and muscle relaxation. The simplest method is Stimson's technique. This method needs the patient to be awake but with plenty of analgesia. The patient is asked to lie face down on the bed with the injured shoulder hanging off the bed towards the floor while holding a 4.5-kg (10-lb) weight in his or her hand. This allows for gentle traction, and often the shoulder will fall back into joint. If this fails I prefer to use a modification of the Hippocratic method or Matsen's method. Gentle traction in placed in the longitudinal direction of the arm with counter traction from one's own heel or a sheet in the axillae. The arm is then adducted and slowly externally rotated until the glenohumeral joint reduces. After manipulation, I would obtain anteroposterior and axial radiographs and repeat a neurovascular examination of the affected limb.

What are the risks of manipulation?

The risks include proximal humeral fracture (including greater tuberosity fracture), rotator cuff injury, nerve injury (particularly the axillary nerve), vascular injury, and inability to reduce the dislocation. Robinson and colleagues performed a prospective study of over 3000 patients presenting with an anterior glenohumeral dislocation. Over 30% had either a rotator cuff injury, a greater tuberosity fracture, or neurological injury.

What structures may be preventing a stable reduction?

The shoulder is maintained in joint by both static and dynamic factors. Static factors include the humeral head and glenoid version, conformity of the joint, the labrum, the glenohumeral ligaments and joint capsule, and negative intra-articular pressure. If there is a significant glenoid fracture or significant capsular labral injury, the humeral head may not remain reduced. Dynamic factors include the rotator cuff muscles, long head of biceps, and the deltoid muscle. A massive rotator cuff tear or large greater tuberosity fracture or axillary nerve injury may prevent the shoulder from remaining

stable. The long head of biceps can get caught posterior to the humeral head and prevent reduction. An iatrogenic fracture of the humeral head during reduction may also prevent reduction of the glenohumeral joint.

Assuming you have achieved a satisfactory reduction, how do you plan to manage this patient?

I would manage this patient in a broad arm sling for comfort and allow a gentle range of movement as pain allows. I would encourage elbow and wrist movements immediately. Historically, patients were advised to remain in a broad arm sling in internal rotation for 3 weeks. However, in 2008, Hovelius and colleagues showed that shoulder immobilization had no effect on the recurrence of dislocation. Use of an external rotation brace for 3 weeks was popularized by a 2007 study by Itoi and colleagues. In that randomized controlled trial patients were placed in a sling in internal or external rotation for 3 weeks following an acute first-time anterior glenohumeral joint dislocation. Patients placed in an external rotation brace had a significantly lower re-dislocation rate in the first 2 years. However, these encouraging results (along with MRI studies suggesting that the damaged anterior labrocapsular structures are reduced better with the arm in external rotation) have not been replicated by other work, and compliance in an external rotation brace is a problem. I would also recommend follow-up at the fracture clinic with an upper limb specialist within the next 1–2 weeks. There is some evidence that it may be better to manage patients with a first-time dislocation of the glenohumeral joint with acute surgical stabilization; therefore patients warrant a discussion regarding this with a shoulder specialist.

Hovelius L, Olofsson A, Sandström B, et al. (2008). Nonoperative treatment of primary anterior shoulder dislocation in patients forty years of age and younger. A prospective twenty-five-year follow-up. *J Bone Joint Surg Am*, 90, 945–952.

Itoi E1, Hatakeyama Y, Sato T, et al. (2007). Immobilization in external rotation after shoulder dislocation reduces the risk of recurrence. A randomized controlled trial. *J Bone Joint Surg Am*, 89, 2124–2131.

Robinson CM, Jenkins PJ, White TO, Ker A, Will E (2008). Primary arthroscopic stabilization for a first-time anterior dislocation of the shoulder. A randomized, double-blind trial. *J Bone Joint Surg Am*, 90, 708–721.

Robinson CM, Shur N, Sharpe T, Ray A, Murray IR (2012). Injuries associated with traumatic anterior glenohumeral joint dislocations. *J Bone Joint Surg Am*, 94, 18–26.

22 UPPER LIMB—FOREARM, WRIST, AND HAND

Viva 1 Questions

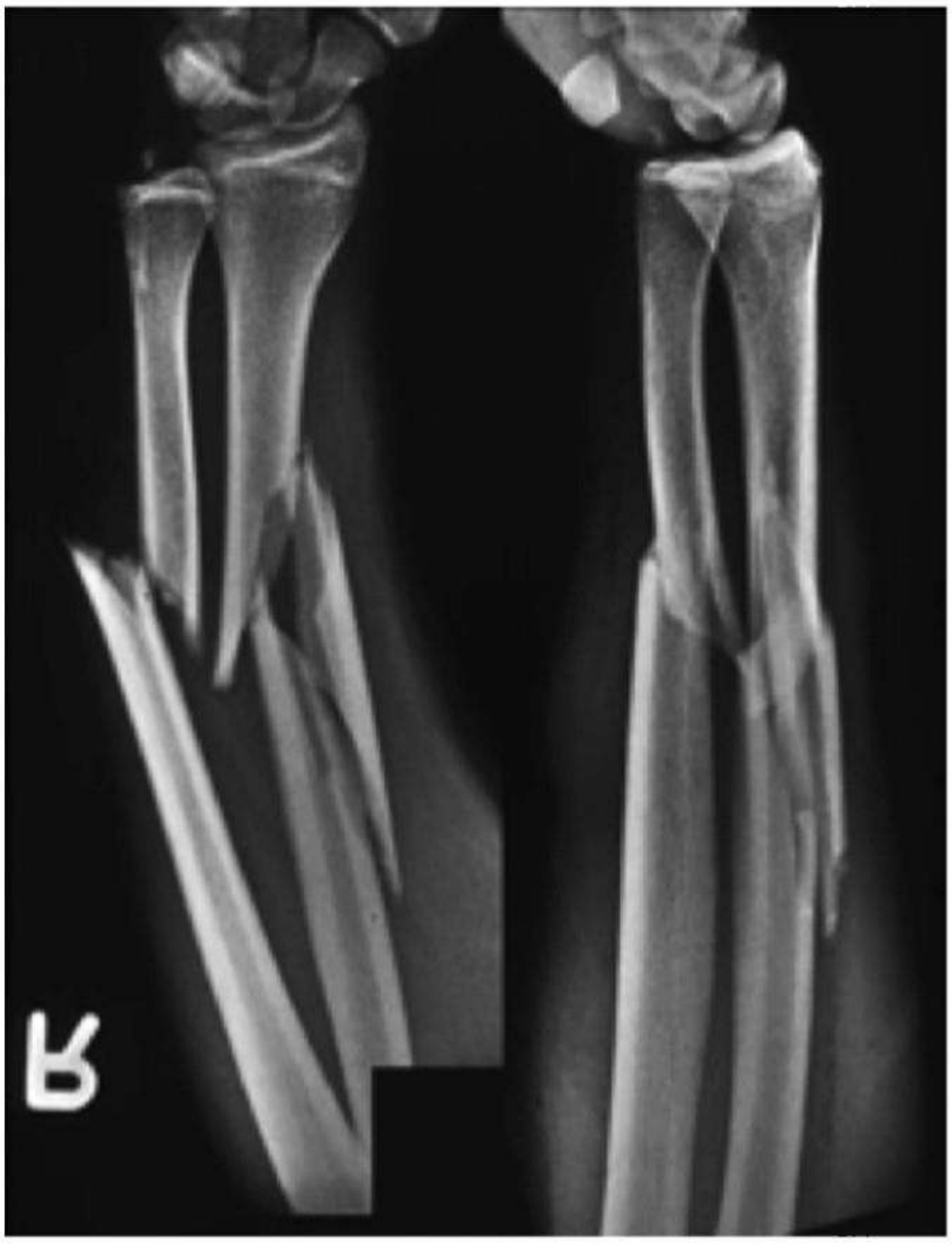

Figure 22.1 Right forearm radiographs

A 32-year-old man has fallen off his motorcycle, sustaining this closed neurovascularly intact injury. Describe what the radiograph shows.

Why is surgical treatment preferred for this type of injury?

What method of fixation would you use for each bone?

Which bone would you fix first, and why?

How could you achieve compression of the ulna fracture?

How would you manage a non-union of the radius?

Viva 1 Answers

A 32-year-old man has fallen off his motorcycle, sustaining this closed neurovascularly intact injury. Describe what the radiograph shows.

These radiographs show middle third diaphyseal fractures of a skeletally mature right radius and ulna. The ulna fracture is a short oblique pattern and the radius fracture is comminuted with a radial-sided butterfly fragment. The fractures are at the same level.

Why is surgical treatment preferred for this type of injury?

The preferred treatment for all adult forearm fractures is surgical, with the aim being to restore length, rotation, and the correct bow of both bones. This maintains forearm rotation and prevents pain, instability, and arthrosis occurring at the distal and proximal radioulnar joints. It is important to remember that the bones of the forearm act together as a unit facilitating rotation and this must be anatomically restored for the return of full function.

What method of fixation would you use for each bone?

I would use open anatomical reduction and rigid internal fixation with small-fragment locking compression plate or dynamic compression plate systems. I would aim for absolute stability techniques where possible—in this case a compression plate for the ulna and, if possible, interfragmentary screws and a neutralization or protection plate for the radius. If there is significant comminution of the radius, it may be that relative stability is the only achievable technique for some or all of the fracture fragments.

Which bone would you fix first, and why?

There is no fixed rule for this. Problems reducing one bone may occur after the first bone has been rigidly fixed. It is hence prudent to stabilize the more difficult fracture first, which tends to be the radius. It may be wise to partially stabilize one bone by leaving some screw holes free before addressing the other bone in case the fixation of the first bone needs to be released to achieve reduction of the second bone. In this case, therefore, it would be wise to address the radius first and the ulna second.

How could you achieve compression of the ulna fracture?

Whilst an interfragmentary screw could be placed either through the plate or separate from the plate, my preferred technique would be a compression plate technique. In this case, after reducing the fracture anatomically, the configuration of screws should allow the fracture to be compressed into the 'axilla' formed by the plate and the bone to prevent sliding with compression.

How would you manage a non-union of the radius?

Non-unions should be initially worked up to rule out infection. Adverse patient factors should be controlled or reversed as much as possible. Surgical principles include removal of non-viable bone, drilling of the canals to encourage bleeding, and management of residual segmental defects with cancellous autograft, which has good results for defects of less than 6 cm. If there is a fracture gap and/or excessive interfragmentary strain, then re-fixation, compression, and reduction of interfragmentary strain is likely to work without grafting. More complex techniques such as bone transport or vascularized grafting may be needed for very large defects.

Viva 2 Questions

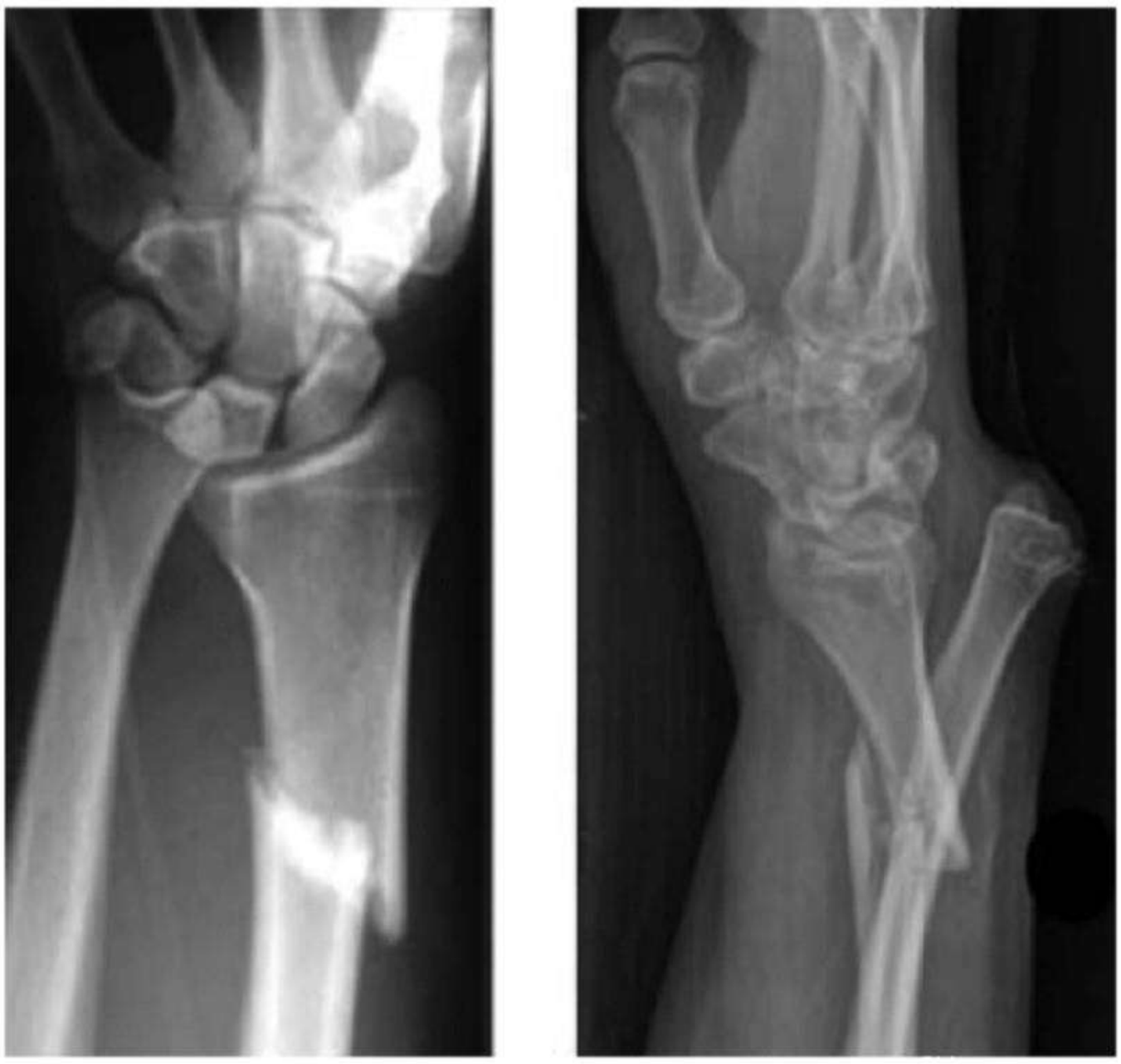

Figure 22.2 AP and lateral wrist radiographs

A 22-year-old man has fallen 2 m from a ladder onto his hand, sustaining this isolated injury. Can you tell me what you see?

What structures might be injured?

How would you manage this patient?

What approach would you use for surgery? Describe it.

How would you assess DRUJ stability?

What options do you have if the DRUJ is unstable after fixation of the radius?

What are the consequences of malreduction or continued instability?

What are your salvage options?

Viva 2 Answers

A 22-year-old man has fallen 2 m from a ladder onto his hand sustaining this isolated injury. Can you tell me what you see?

This radiograph shows a skeletally mature distal forearm and wrist with a short oblique fracture of the distal radius which is shortened and dorsally translated 100%. There is an associated dislocation of the distal ulna dorsally from both its ulnocarpal and radioulnar articulations, which is likely to be open or at least putting the skin in peril. This is a Galeazzi fracture–dislocation of the forearm.

What structures might be injured

Apart from the bony injury to the radius, the structures that stabilize the DRUJ are potentially injured, especially the triangular fibrocartilaginous complex, the most important component of which is now thought to be the ligamentum subcurentum. There may also be an injury to the ulnar nerve, its dorsal cutaneous branch, and the extensor and flexor tendons of the fingers and wrist.

How would you manage this patient?

If this is an open injury, then antibiotics, tetanus prophylaxis, saline gauze, and clinical photography would be mandated. A documented neurological and vascular examination prior to any reduction is also essential. I would attempt to perform a closed reduction and cast immobilization initially to reduce soft tissue and neurological compression. Definitively this patient requires anatomical reduction and stable fixation of the radius followed by assessment of the DRUJ, which is usually, but not always, rendered stable after appropriate fixation of the radius.

What approach would you use for surgery? Describe it.

Either a volar Henry's approach or a dorsal Thompson's approach may be used . . . [see Chapter 17].

How would you assess DRUJ stability?

The DRUJ of the unaffected wrist should be assessed pre-operatively and an X ray should be saved for reference. Once the radius of the affected wrist has been stabilized a radiographic assessment of the congruity of the DRUJ should be performed. The DRUJ is then stressed for anteroposterior stability with the elbow flexed and rested on the table while the forearm is pronated, put in neutral, and supinated. Anteroposterior translation should not be greater than that of the opposite wrist in any position. Finally continuous fluoroscopy should be used to assess if there is dynamic subluxation of the DRUJ during pronosupination.

What options do you have if the DRUJ is unstable after fixation of the radius?

In the acute setting the reduction and length of the radius should first be scrutinized. The fluoroscopy images should then be assessed to see if there is a gap at the DRUJ, which may indicate soft tissue interposition. If this is the case the DRUJ should be explored via a dorsal approach and open reduction performed. If there is no interposition, there are three broad options: explore and repair the soft tissue restraints of the DRUJ; immobilize the forearm in a position where the DRUJ is stable (usually supination); or place two quadracortical K-wires between the radius and ulna to

Viva 2 Answers

A 22-year-old man has fallen 2 m from a ladder onto his hand sustaining this isolated injury. Can you tell me what you see?

This radiograph shows a skeletally mature distal forearm and wrist with a short oblique fracture of the distal radius which is shortened and dorsally translated 100%. There is an associated dislocation of the distal ulna dorsally from both its ulnocarpal and radioulnar articulations, which is likely to be open or at least putting the skin in peril. This is a Galeazzi fracture–dislocation of the forearm.

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How would you manage this patient?

If this is an open injury, then antibiotics, tetanus prophylaxis, saline gauze, and clinical photography would be mandated. A documented neurological and vascular examination prior to any reduction is also essential. I would attempt to perform a closed reduction and cast immobilization initially to reduce soft tissue and neurological compression. Definitively this patient requires anatomical reduction and stable fixation of the radius followed by assessment of the DRUJ, which is usually, but not always, rendered stable after appropriate fixation of the radius.

What approach would you use for surgery? Describe it.

Either a volar Henry's approach or a dorsal Thompson's approach may be used . . . [see Chapter 17].

How would you assess DRUJ stability?

The DRUJ of the unaffected wrist should be assessed pre-operatively and an X ray should be saved for reference. Once the radius of the affected wrist has been stabilized a radiographic assessment of the congruity of the DRUJ should be performed. The DRUJ is then stressed for anteroposterior stability with the elbow flexed and rested on the table while the forearm is pronated, put in neutral, and supinated. Anteroposterior translation should not be greater than that of the opposite wrist in any position. Finally continuous fluoroscopy should be used to assess if there is dynamic subluxation of the DRUJ during pronosupination.

What options do you have if the DRUJ is unstable after fixation of the radius?

In the acute setting the reduction and length of the radius should first be scrutinized. The fluoroscopy images should then be assessed to see if there is a gap at the DRUJ, which may indicate soft tissue interposition. If this is the case the DRUJ should be explored via a dorsal approach and open reduction performed. If there is no interposition, there are three broad options: explore and repair the soft tissue restraints of the DRUJ; immobilize the forearm in a position where the DRUJ is stable (usually supination); or place two quadracortical K-wires between the radius and ulna to

maintain reduction of the DRUJ and immobilize the forearm in a plaster. Which option is used depends on the experience of the surgeon and the pattern of instability encountered. My preferred choice is to . . .

What are the consequences of malreduction or continued instability?

The patient will have decreased grip strength, limited pronation and supination, and on-going pain. He is likely to go on to develop DRUJ arthritis and chronic instability which is likely to affect his function.

What are your salvage options?

Salvage options include reconstruction of the triangular fibrocartilage complex, distal ulna resection, Suave–Kapandji-type procedures, and DRUJ fusion. Arthroplasty is unlikely to work due to soft tissue instability in the acute setting.

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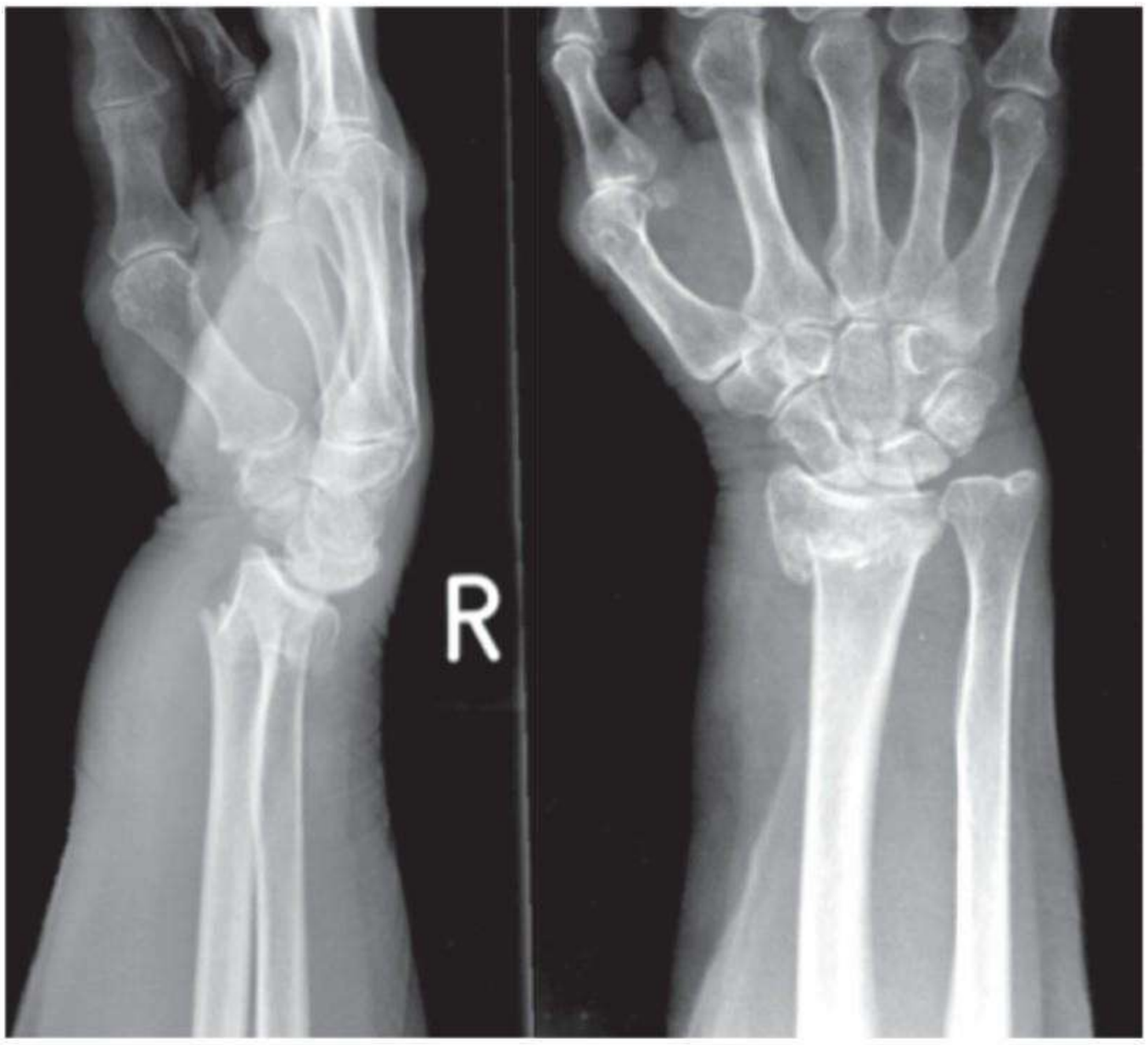

Figure 22.3 AP and lateral wrist radiographs

This independent, right-hand dominant 70-year-old woman presented with an isolated injury to her right wrist after a fall in her kitchen. She is neurovascularly intact. Can you describe the radiographs?

What might help predict instability after a distal radius fracture?

What factors affect functional outcome?

What are the management options for this patient? What would you do?

What are the specific complications of that treatment modality? How would you avoid these?

Viva 3 Answers

This independent, right-hand dominant 70-year-old woman presented with an isolated injury to her right wrist after a fall in her kitchen. She is neurovascularly intact. Can you describe the radiographs?

These radiographs show an extra-articular right distal radial fracture with dorsal angulation and translation. There is shortening, loss of normal radial inclination, and radiocarpal malalignment. There is some dorsal comminution and the bone appears osteopenic.

What might help predict instability after a distal radius fracture?

Patient factors include anything that may cause osteoporotic, weaker bone, such as age, medications, or alcohol and smoking. Higher-energy injuries with greater soft tissue damage may indicate that the fracture is more likely to be unstable. Initial severe displacement is an indicator of instability, as are intra-articular fracture, dorsal comminution, and loss of carpal alignment.

What factors affect functional outcome?

The three primary factors proven to affect functional outcome are radiocarpal malalignment on the lateral view; intra-articular step or gap > 2 mm, and radial shortening > 2 mm. Dorsal tilt > 12° from the normal position has also been shown to affect function, although not as conclusively as the other parameters.

What are the management options for this patient? What would you do?

Evidence for treatment of distal radial fractures in the elderly is controversial. Some studies show satisfactory function in the elderly population despite significant radiographic malunion. Treatment should therefore be tailored to the individual depending on pre-morbid functional level, handedness, and expectations. The aim should be to recognize which fractures may be unstable and therefore fail plaster treatment and those with features of poor functional outcome. In this case the patient had a high pre-morbid functional state and has features of instability and poor functional outcome. Operative treatment is therefore reasonable. Most commonly this would be in the form of a volar periarticular locking plate, because of her osteoporotic bone and greater danger of late collapse with use of K-wires alone. External fixation is not ideal for elderly patients due to pin site infection, pin loosening, metacarpal fracture, its cumbersome nature, and wrist stiffness. Nevertheless, the various surgical treatment modalities appear to have similar long-term functional outcomes.

What are the specific complications of that treatment modality? How would you avoid these?

Despite its exponentially increasing use, volar locking plate fixation is associated with a significant rate of complications, ranging between 4% and 30% in published studies. Specific complications include median nerve injury, CRPS, rupture of the EPL and FPL, intra-articular screw placement, and late collapse and loss of fixation. EPL rupture can be minimized by avoiding breach of the dorsal cortex when drilling, using smooth pegs rather than threaded screws distally, and avoiding overly long screw placement. Taking an axial view radiograph helps to appreciate screw length. FPL rupture is usually caused by incorrect positioning of the plate beyond the watershed line or too radially. This causes prominence of the plate and has been associated with FPL rupture. Correct plate

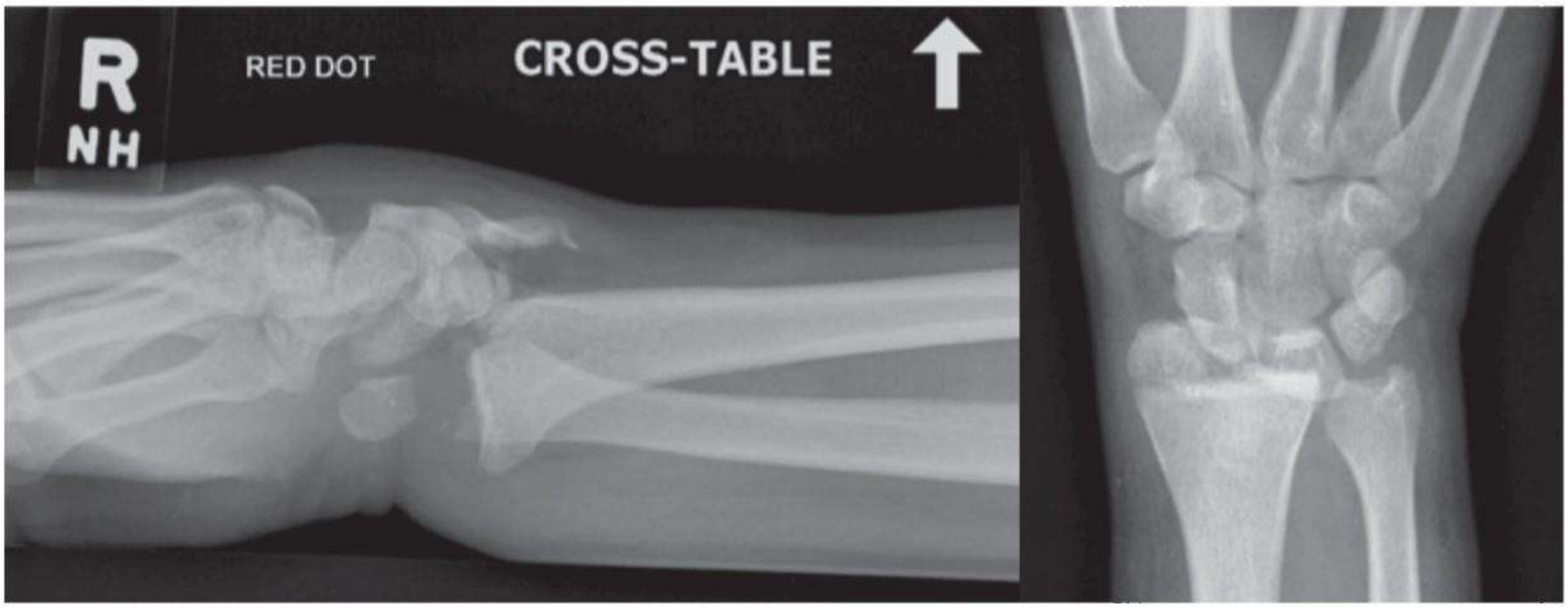

Figure 22.4 AP and lateral wrist radiographs

Describe these radiographs of a 23-year-old who was involved in a high-speed RTA, sustaining an isolated, neurovascularly intact, closed wrist injury. Tell me how you would manage this patient.

The wrist remains unstable despite closed reduction—what are your options?

How would you apply an external fixator and what are the risks?

What measures could you use to increase the rigidity of the external fixator?

Do you know of any evidence to support or refute the use of external fixation for distal radius fractures?

Viva 4 Answers

Describe these radiographs of a 23-year-old who was involved in a high-speed RTA, sustaining an isolated, neurovascularly intact, closed wrist injury. Tell me how you would manage this patient.

These radiographs shows a highly comminuted fracture of the dorsal rim of the right distal radius with dislocation of the radiocarpal joint in a skeletally mature patient. This is a high-energy injury and mandates urgent management as I would expect structures such as the median nerve to be compromised.

The wrist remains unstable despite closed reduction—what are your options?

The priority is to achieve a concentric, stable reduction. If this is not possible by closed means then the options are ORIF, external fixation, or a combination of both. I would use external fixation in the acute setting as this is a complex fracture requiring planning and appropriate expertise if internal fixation is desired.

How would you apply an external fixator and what are the risks?

I would perform a closed reduction and use the principle of ligamentotaxis applied through the external fixator to maintain the reduction. I would place two pins in the radial aspect of the radius proximal to the zone of injury. I would do this with a mini-open approach to avoid damage to the superficial radial nerve. I would bridge the radiocarpal joint and place two pins in the radial aspect of the index metacarpal at approximately 45° radially inclined to avoid the extensor tendon, in particular the extensor hood. The metacarpal is also at risk of fracture if the pins are inappropriately large or placed incorrectly. I would therefore feel the bone with the tip of the pin prior to insertion to ensure I was not aiming eccentrically, which would increase the likelihood of iatrogenic fracture. I would connect the pins to bars, taking care not to overdistract the radiocarpal joint.

What measures could you use to increase the rigidity of the external fixator?

Increasing the number and diameter of the pins and/or the bars and placing the pins in a near–far configuration can help, as does using pins in different planes relative to each other. Stiffness increases as the distance between the bars and the skin is reduced. Placing the pins in a slightly divergent manner increases pre-load in the system and thus stiffness, but risks earlier loosening of the pins. Above all else, however, I must ensure that the fracture is reduced with maximal bony contact as this has the greatest effect on stability.

Do you know of any evidence to support or refute the use of external fixation for distal radius fractures?

Multiple prospective randomized and non-randomized trials have shown improved functional and radiographic outcomes, or only improved radiographic outcomes, with external fixation versus closed reduction and casting. Two prospective randomized control trials have shown improved early functional outcomes (3 months) but equivalent medium-term outcomes (1 year) between volar plate fixation and bridging external fixation. Bridging external fixation is thought to be superior to non-bridging external fixation which was once in vogue but not appropriate in this case.

Viva 5 Questions

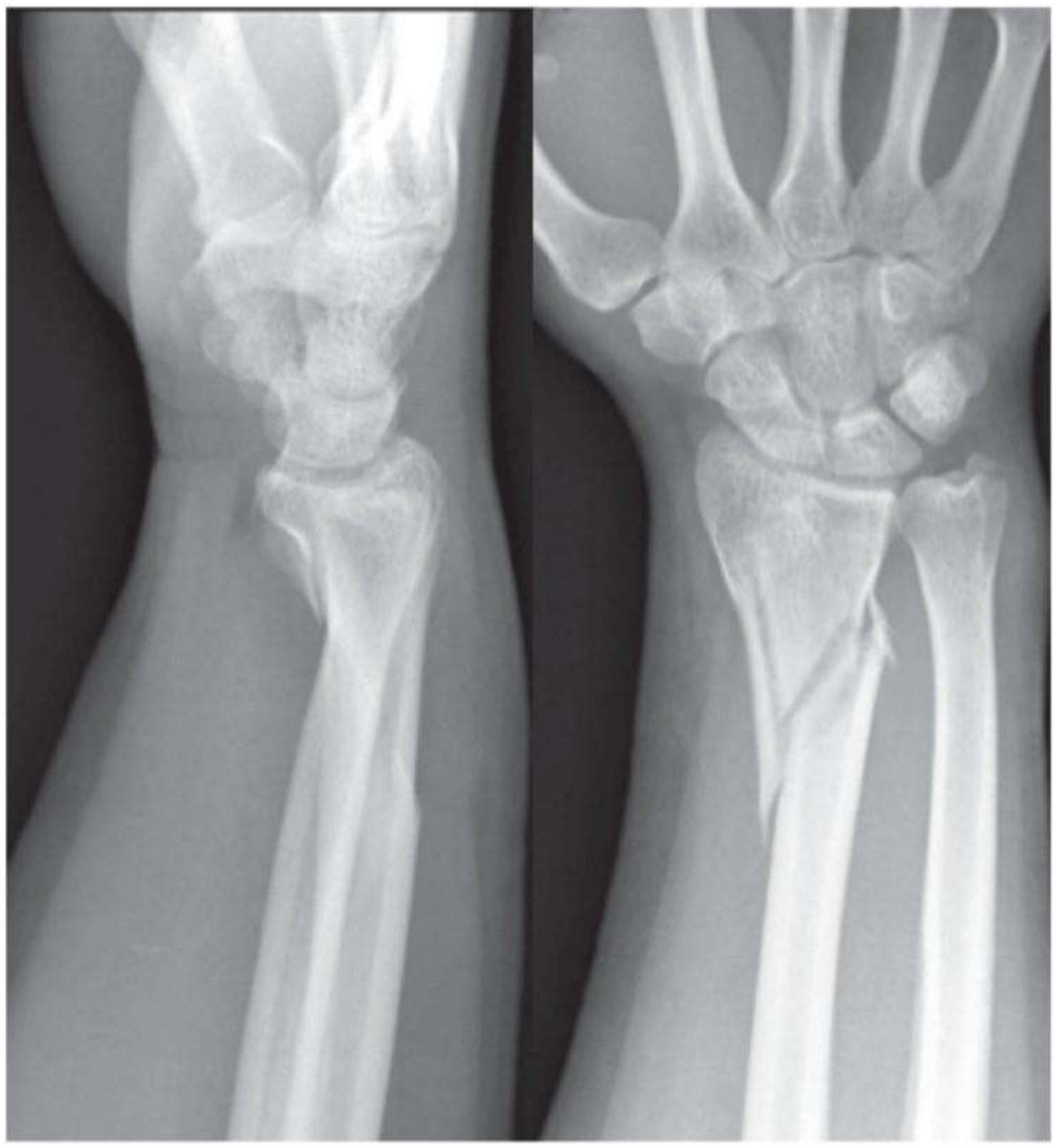

Figure 22.5 AP and lateral wrist and forearm radiographs

A 46-year-old man involved in a RTA presents at 11 p.m. with an isolated right wrist injury. Describe the radiographs.

On examination, he has tingling in the index finger and thumb since the injury—how will you manage him further?

Despite reduction and elevation, he now has complete numbness in his index finger and thumb—how will you manage him further?

What are your indications for urgent surgical intervention?

What surgery would you perform and how would you do it? What approach would you use?

Would you use a single or separate incision for the carpal tunnel release?

A 40-year-old man involved in a RTA presents at 11 p.m. with an isolated right wrist injury. Describe the radiographs.

These are anteroposterior and lateral views of a skeletally mature individual with a comminuted radius fracture and associated ulna styloid tip fracture. There is volar subluxation of the carpus, and I suspect there is a partial articular component to this fracture in the sagittal plane.

On examination, he has tingling in the index finger and thumb since the injury—how will you manage him further?

Given that he has a high-energy injury I would assess him as per the ATLS protocol to rule out systemic or life-threatening injuries. I'd assess his limb for the extent of neurological compromise, specifically in the median nerve distribution, as well as looking for vascular compromise and for signs of compartment syndrome. I would document my findings. I would splint his wrist and elevate his arm and reassess his neurological state—again documenting the result.

Despite reduction and elevation, he now has complete numbness in his index finger and thumb—how will you manage him further?

This patient has clinically evolving median nerve compression. As such I would like to take him urgently to theatre with the intention of stabilizing his fracture and decompressing his carpal tunnel.

What are your indications for urgent surgical intervention?

The main indication for urgent surgery is evolving, progressive median nerve compression despite simple measures such as fracture reduction, splinting, and elevation. Mild neurological symptoms that are unchanged or improve after simple measures do not warrant emergent surgery as they are indicative of a neurapraxia rather than a progressive compression neuropathy that may result in significant neural damage and poor prognosis if surgery is delayed. There are three general scenarios relating to nerve dysfunction after distal radius fracture. (1) There is neurological compromise from the outset, which does not get worse—this indicates a neurapraxia or rarely a laceration sustained at the time of injury. Emergent intervention will not change the natural course of nerve recovery. (2) There are evolving and deteriorating neurological signs/symptoms that are not alleviated by simple measures. This indicates an acute carpal tunnel syndrome at best and impending compartment syndrome at worst. Emergent decompression should be performed. (3) Neurological symptoms develop in the subsequent days or weeks. This is probably due to perineural oedema or inflammation. If symptoms are significant then decompression is warranted but not on an emergent basis.

What surgery would you perform and how would you do it? What approach would you use?

This is an unstable fracture as indicated by the injury mechanism, the comminution, and the soft tissue component, resulting in nerve compression. Surgery should include reduction and stable plate fixation of the fracture as well as median nerve decompression. A distal radial volar periarticular plate with sufficient length to bridge the comminution should be used and reduction should concentrate on restoring radial length, alignment, and any intra-articular step. Most commonly the Henry approach or a modification through the bed of the FCR would be used.

Would you use a single or separate incision for the carpal tunnel release?

It is recommended that concurrent carpal tunnel decompression should be performed via a separate incision. This avoids ulnar zig-zagging of the Henry incision to meet the carpal tunnel incision which places the palmar cutaneous branch of the median nerve at risk. This is because this branch arises from the radial aspect of the median nerve around 5 cm proximal to the wrist crease and lies on the ulna aspect of the FCR before supplying the skin over the thenar eminence.

Hoppenfeld S, DeBoer P, Buckley R (2009). *Surgical Exposures in Orthopaedics: the Anatomic Approach*, 4th edn. Lippincott Williams and Wilkins, Philadelphia, PA.

Niver G, Ilyas M (2012). Carpal tunnel syndrome after distal radius fracture. *Orthop Clin N Am*, 43, 521–527.

Schmidt AH, Anglen J, Nana AD, Varecka TF (2010). Adult trauma: getting through the night. *J Bone Joint Surg Am*, 92, 490–505.

Viva 6 Questions

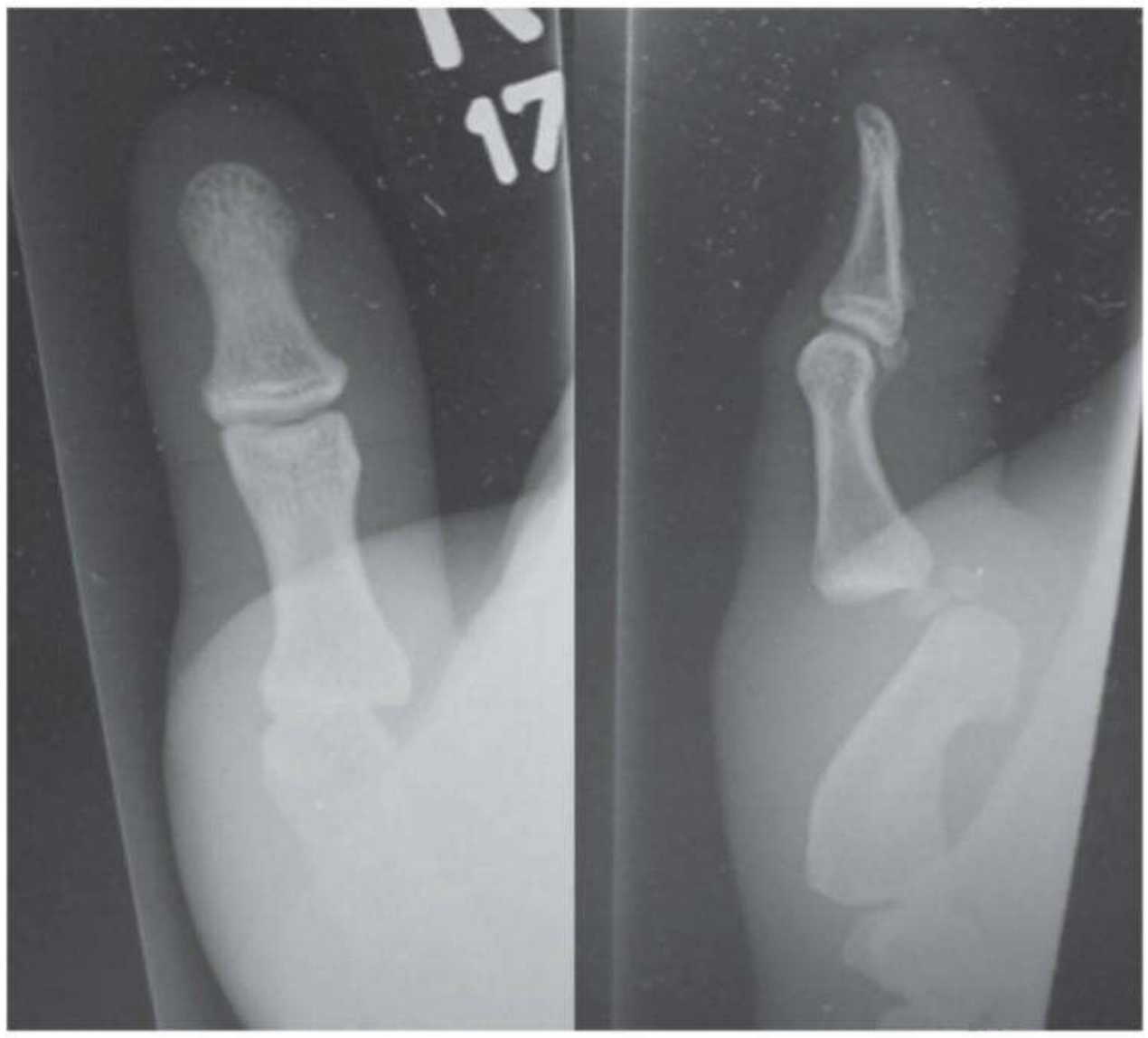

Figure 22.6 Thumb AP and lateral radiograph

Describe these radiographs.

What is unique about this injury?

Explain the 'noose effect'.

What are the complications associated with this injury?

What would your approach be—volar or dorsal? Are you aware of any relevant literature?

Viva 6 Answers

Describe these radiographs.

These are radiographs of the thumb of a skeletally mature patient showing a dorsal dislocation of the thumb metacarpophalangeal joint. I cannot see any associated fractures but the thumb sesamoids are entrapped between the metacarpal and proximal phalanx.

What is unique about this injury?

Most dislocations of the thumb metacarpophalangeal joints are dorsal, although palmar dislocations have also been reported. The mechanism of injury involves hyperextension with associated complete rupture of the volar plate. Rupture of the volar plate mostly occurs proximal to the sesamoids. These injuries are usually reducible but sometimes they can be irreducible if it is a complex dislocation. These radiographs show the intra-articular position of the sesamoid; this is pathognomonic for a complex dislocation with the so-called 'noose effect'.

Explain the 'noose effect'.

In a complex dislocation the FPL tendon remains within the flexor tendon sheath and displaces to the ulnar side of the metacarpal head, creating a 'noose' around the metacarpal neck together with the radially displaced thenar intrinsic musculature. Any traction applied to reduce the dislocation tightens this noose, making reduction difficult.

What are the complications associated with this injury?

In the short term there is a risk of injury to the digital nerve if the volar approach is used, along with the risk of infection. In the long term there is a risk of stiffness, secondary arthritis, osteonecrosis of the metacarpal head, and even premature closure of the physis in the paediatric population.

What would your approach be—volar or dorsal? Are you aware of any relevant literature?

In his original description of the surgical approach for an irreducible dorsal dislocation Farabeuf used a dorsal incision. This involves a lower risk of NVB injury and easier access to the entrapped volar plate than the volar approach. Also, it may be easier to address any associated metacarpal head fractures using a dorsal approach. Kaplan described a volar approach for the treatment of irreducible dorsal dislocations. A volar approach allows access to the volar plate and also to the surrounding ligaments and tendons that can trap the metacarpal head and cannot be addressed through a dorsal incision. Eaton and Dray described a technique of releasing the A1 pulley, which then releases tension on the flexor tendons allowing the proximal phalanx and attached volar plate to reduce into their anatomical positions.

Dinh P, Franklin A, Hutchinson B, Schnall SB, Fassola I (2009). Metacarpophalangeal joint dislocation. *J Am Acad Orthop Surg*, 17, 318–324.

Eaton RG, Dray GJ (1982). Dislocations and ligament injuries in the digits. In: DP Green (ed.) *Green's Operative Hand Surgery*, Vol. 1, pp.647–668. Churchill Livingstone, New York.

Farabeuf LHF (1876). De la luxation du ponce en arrière. *Bull Soc Chir*, 11, 21–62.

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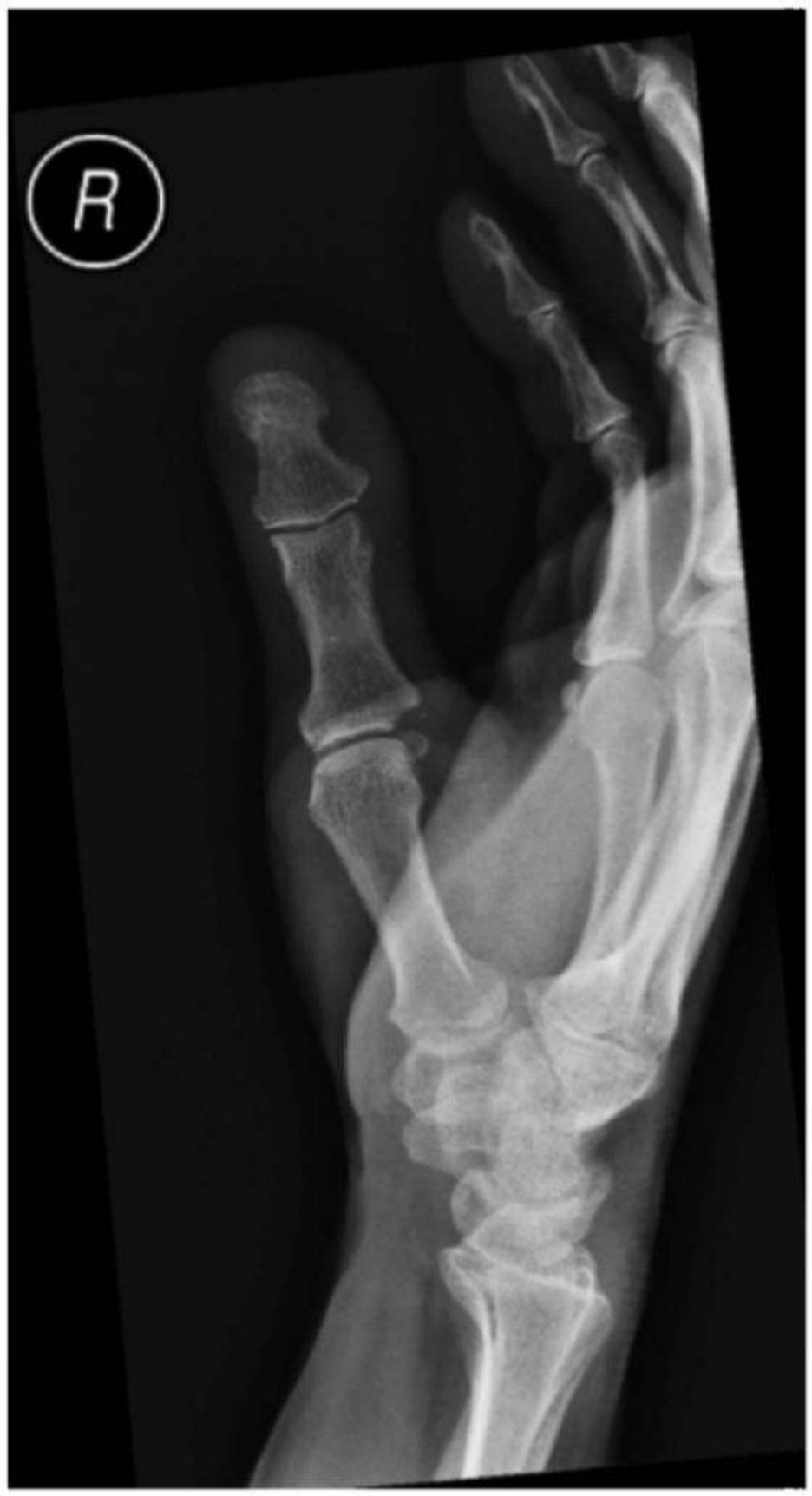

Figure 22.7 Thumb radiograph

Describe the injury shown in this radiograph.

Can you describe the mechanism of injury?

What would you do before you stress this injury?

Describe a Stener lesion.

How would you manage a chronic ulnar collateral ligament injury?

Viva 7 Answers

Describe the injury shown in this radiograph.

This is a plain radiograph of a thumb showing an avulsion fracture of the proximal phalanx suggestive of avulsion of the ulnar collateral ligament. I would like to see another view.

Can you describe the mechanism of injury?

Valgus stress to the thumb can result in ligamentous or bony disruption of the ulnar collateral ligament. The most common mechanism is a fall on the abducted thumb. This can also result in rupture of the dorsal capsule, adductor mechanism, and extensor pollicis brevis tendon which will increase the degree of instability found on examination. When the proper collateral ligament is ruptured, instability will be present when the thumb is tested in flexion. When the accessory collateral ligament is also torn the tear is considered complete, and there is instability in extension as well as flexion.

What would you do before you stress this injury?

When dealing with any patient with a possible injury of the ulnar collateral ligament I would ensure a radiograph of the involved thumb is performed as there is a risk of displacing the avulsed fragment, creating a Stener lesion, which could jeopardize the option of treating this injury non-operatively.

Describe a Stener lesion.

In a Stener lesion there is displacement of the distal end of the completely ruptured ligament and it comes to lie superficial and proximal to the adductor aponeurosis. Due to the interposition of the adductor aponeurosis, these injuries do not heal with non-operative treatment and need operative intervention to restore function.

How would you manage a chronic ulnar collateral ligament injury?

Chronic ulnar collateral ligament injury usually results from the failure of a patient to seek advice following an acute injury. Management is dependent on the underlying condition of the metacarpophalangeal joint. If there are no arthritic changes then a reconstruction can be performed with a graft harvested from the palmaris longus or partial flexor carpi radialis. The graft is woven through the metacarpal neck and the proximal phalanx and secured with suture anchors. In long-standing cases patients can develop arthritic changes and would benefit from fusion of the metacarpophalangeal joint.

Heyman P (1997). Injuries to the ulnar collateral ligament of the thumb metacarpophalangeal joint. *Am Acad Orthop Surg*, 5, 224–229.

Tang P (2011). Collateral ligament injuries of the thumb metacarpophalangeal joint. *J Am Acad Orthop Surg*, 19, 287–296.

Viva 9 Questions

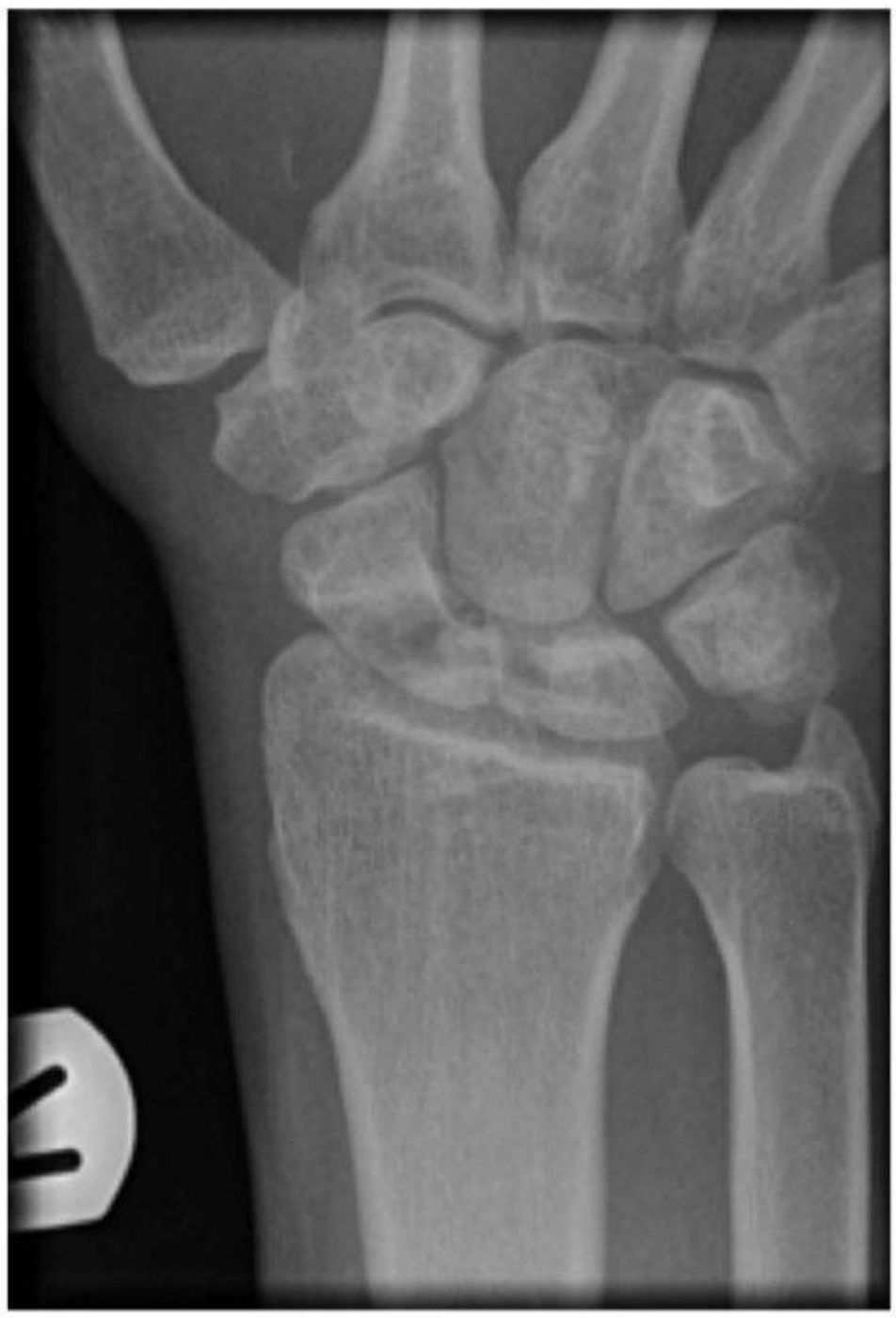

Figure 22.9 Wrist AP radiograph

What does this radiograph show?

What causes non-union of the scaphoid?

What are the principles of management of this type of injury?

If a patient presents late with a non-union and already has osteoarthritic change in the wrist joint, what salvage procedures might you consider?

Viva 9 Answers

What does this radiograph show?

This is an anteroposterior view of the wrist showing an established non-union of the scaphoid. There does not appear to be any osteoarthritis or scaphoid non-union advanced collapse (SNAC). I would like to see further views to look for any humpback deformity of the scaphoid, and also a CT of the wrist to aid surgical planning.

What causes non-union of the scaphoid?

The blood supply to the scaphoid has been considered to be the most important cause of non-union. The blood supply comes from branches of the radial artery that enter the bone through dorsal foramina. As these are mainly located distally, the proximal pole has a poor blood supply, increasing the risk of non-union. The degree of displacement of the fracture or instability of the fracture may be relevant. Proximal pole fractures have high non-union rates and all of these factors may play a role. The length of time from fracture to diagnosis and institution of the correct treatment may influence the union rate.

What are the principles of management of this type of injury?

The objective of treatment is to achieve union, relieve symptoms, and reduce the long-term risk of osteoarthritis in the joint. If the non-union is established then internal fixation with bone grafting is appropriate. A headless compression screw, of which there are several on the market, is considered the gold standard for fixation as it allows the screw to be buried so impingement is not an issue. The graft may be cancellous or corticocancellous depending on the type of bone defect that is found at surgery. Some surgeons use vascularized grafts. Grafts may be taken from the distal radius or the iliac crest.

If a patient presents late with a non-union and already has osteoarthritic change in the wrist joint, what salvage procedures might you consider?

Simple procedures include radial styloidectomy, excision of part of the scaphoid—usually the proximal pole—and denervation of the wrist joint. More formal reconstructive procedures include scaphoid excision and four-corner fusion, arthroplasty, proximal row carpectomy, and total wrist fusion.

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Viva 1 Questions

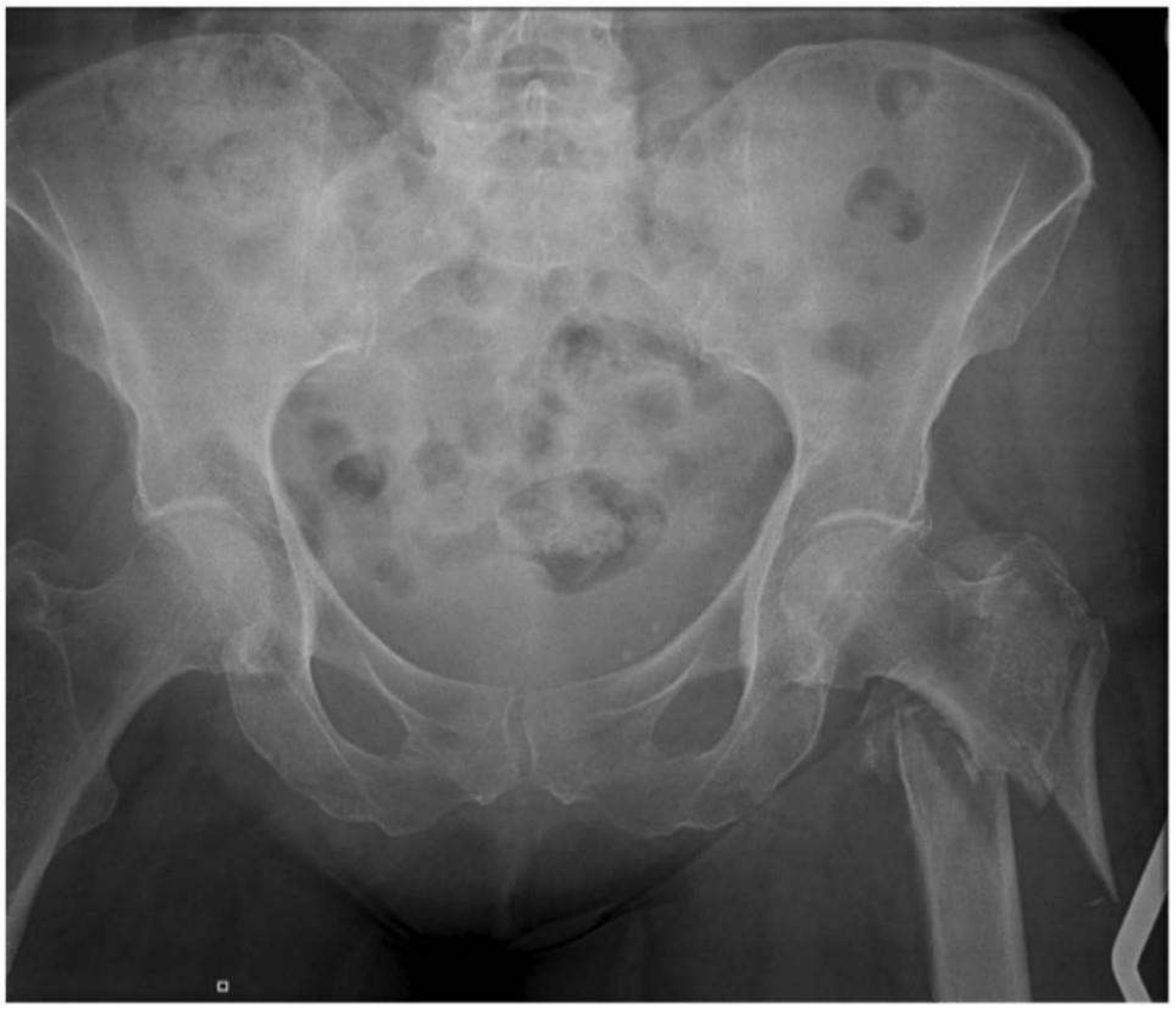

Figure 23.1 Pelvis radiograph

An 80-year-old woman is admitted under your care. How would you manage the fracture shown in the radiograph?

Why have you suggested a nail rather than a dynamic hip screw?

What DVT prophylaxis would you use?

What causes the deformity typical in these fractures, and how will you reduce the fracture?

You mentioned the suspicion of skeletal metastases and pathological fracture. What primary cancers are most likely? How will this affect your management?

Viva 1 Answers

Viva 1 Answers

An 80-year-old woman is admitted under your care. How would you manage the fracture shown in the radiograph?

The radiograph shows a subtrochanteric fracture of the left femur, with reverse obliquity. I would provide prompt analgesia then take a history, paying particular attention to the mechanism of injury, comorbidities, and pre-injury functional level. Specifically I would ask about previous history of cancer, as there is a high incidence of these fractures occurring secondary to pathological lesions of the bone. I would assess the patient for cognitive impairment and examine her. This examination would be focused on the identification of dehydration, neurovascular status and the soft tissue condition of the limb, cardiac and respiratory pathologies, and, in this patient, a screen for common cancers that metastasize to bone because subtrochanteric fractures are often pathological.

I would then arrange simple investigations. These would include full-length femur radiographs, a chest radiograph, ECG, urine dipstick, and blood tests. Blood tests would include full blood count, urea and electrolytes, liver function tests, erythrocyte sedimentation rate, bone profile, and a cross-match for two units of blood. If the imaging showed a lytic lesion I would be concerned about renal deposit and major bleeding and, if supported by further investigation, would consider pre-operative embolization and further cross-matching. I would begin rehydration with intravenous crystalloids and ask the orthogeriatrician to assess the patient pre-operatively. If the patient is frail I'd also ask for an anaesthetic review.

Having done this, I would recommend surgical stabilization of the fracture with the use of a long cephalomedullary nail and consent the patient. I would organize the surgery to be performed on the next routine trauma list (ideally within 24 hours). At my hospital we adopt enhanced recovery principles with our neck of femur patients and I would therefore prescribe the appropriate carbohydrate drinks. The patient would be allowed to eat until 6 hours before surgery and drink water until 2 hours before.

Why have you suggested a nail rather than a dynamic hip screw?

This is a subtrochanteric fracture. With a dynamic hip screw (DHS) dynamization will not compress the fracture. Instead, the reduction may fail as the shaft medializes due to loss of lateral buttressing—if this happens there is a significant risk of screw cut-out and failure. An intramedullary device is on-axis and therefore has a mechanical advantage in resisting varus cantilever forces compared with a DHS in an unstable proximal femoral injury. Further, it resists shaft medialization as the nail is within the diaphyseal bone.

There are now clinical data to support this biomechanical theory. An example is the paper by Matre and colleagues from 2013, based on the Norwegian Hip Fracture Database. The DHS group had 50% higher re-operation rate, worse pain scores, and worse mobility at 1 year.

What DVT prophylaxis would you use?

Patients with hip and femur fractures are at high risk of VTE. I would therefore use both mechanical and chemical prophylaxis. Unless contraindicated, I would prescribe **thromboembolic deterrent** (TED) stockings and use an intermittent calf-compression boot during surgery. After surgery, I would encourage early mobilization and aim to keep the patient well hydrated throughout her stay. In line with NICE guidelines for VTE prophylaxis after hip fracture, I would prescribe LMWH (dalteparin 5000 units) and begin this on admission. It would need omission for 18 hours before

spinal anaesthetic, but would then recommence 6–12 hours post-operatively. I would continue this and the stockings for 35 days.

What causes the deformity typical in these fractures, and how will you reduce the fracture?

The proximal fragment is abducted by the insertions of the gluteus minimis and medius tendons on the anterior and middle facets of the greater trochanter. If the lesser trochanter remains attached to the proximal fragment, it provides a flexion force. The distal fragment is adducted by the insertion of adductors on the diaphysis of the femur. I often find it difficult to achieve adequate reduction via closed means on the traction table. When I have achieved as much as possible by closed means on the traction table I therefore routinely open subtrochanteric fractures (via a small mid-lateral incision) to reduce the fracture. I hold the fracture reduced with a large plate-holding forceps and, if required, pass a circlage cable. Having achieved reduction I then insert my nail, ensuring that my entry point is not too lateral so I don't re-displace the fracture. The small open incision is almost always at the correct site for placing the recon screws, so does not add significantly to the operative insult. I always take time to ensure there is no residual varus deformity as this increases the risk of failure of my construct.

You mentioned the suspicion of skeletal metastases and pathological fracture. What primary cancers are most likely? How will this affect your management?

Lytic bony lesions in men are commonly from lung or renal primaries. In contrast, prostate cancer tends to cause sclerotic deposits. Chest radiograph and urine cytology would therefore help. A contrast CT of chest, abdomen, and pelvis would pick up 85% of unknown primaries.
Renal metastases can bleed heavily at surgery. I would therefore consider angiography and embolization of the lesion before surgery and would ensure I have several units of blood cross-matched and tranexamic acid available. I would warn the anaesthetic team of the potential for haemorrhage and counsel the patient accordingly. In patients with skeletal metastases it is preferable to stabilize the whole bone, so a short nail would not be acceptable.

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Viva 2 Questions

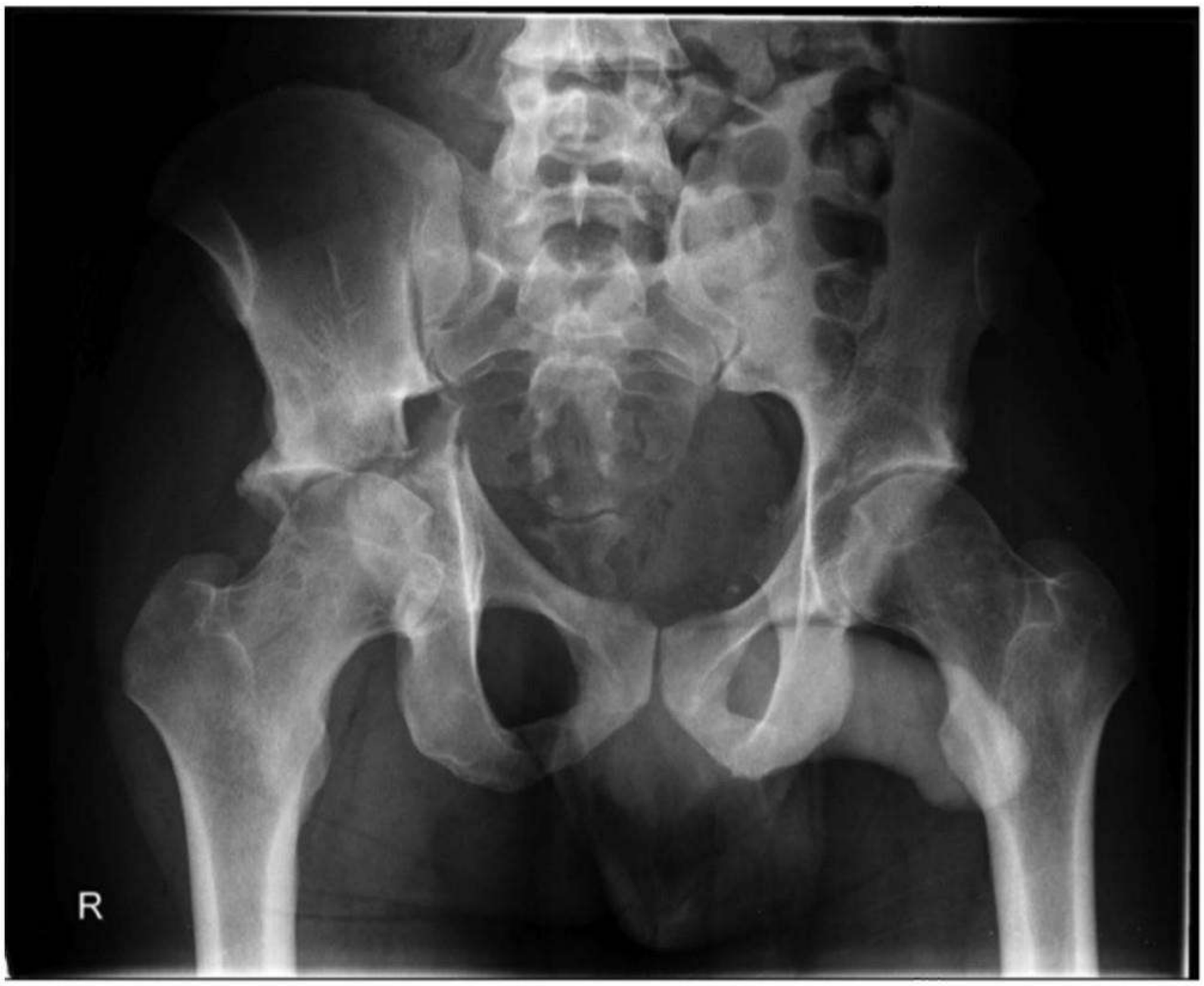

Figure 23.2 Pelvis radiograph

Describe this radiograph.

What are the radiological features on an anteroposterior pelvis radiograph that are evaluated for acetabular fractures?

What are Judet views?

How are these fractures classified?

Are you aware of any guidelines regarding acetabular fractures?

What are the complications of both these fractures and their treatment? Do you know of any factors which predict outcome?

Viva 2 Answers

Describe this radiograph.

This is an anteroposterior view of the pelvis of an adult male showing a displaced right acetabular fracture with medial displacement of the femoral head.

What are the radiological features on an anteroposterior pelvis radiograph that are evaluated for acetabular fractures?

The anterior column is represented by the ilioinguinal line, and the posterior column by the iliois-chial line. The anterior and posterior walls are examined; they can be identified by tracing the inferior border of the superior and inferior pubic rami, respectively, to the edge of the acetabulum. The teardrop is the radiographic representation of the medial wall. The sourcil represents the weight-bearing dome. Further evaluation of the fracture pattern would be helped with Judet views and a CT scan with coronal and sagittal reformats.

What are Judet views?

These are 45° oblique views taken using a wedge, and centred on the affected hip joint. Two views are gained. For the right hip, angling the left hip up by 45° provides an iliac oblique view which visualizes the posterior column and the anterior wall. Angling with the right hip upwards gives an obturator oblique view to visualize the anterior column and the posterior wall.

How are these fractures classified?

These fractures are classified using the Judet and LeTournel classification. This describes five simple fracture patterns: posterior column, posterior wall, anterior column, anterior wall, and transverse. There are also five combined fracture patterns: posterior column with posterior wall, anterior column with posterior hemitransverse, T-fractures, and an associated fracture of both columns. The defining feature of this final fracture pattern is the loss of contact of any of the articular surface with the axial skeleton.

Are you aware of any guidelines regarding acetabular fractures?

The BOAST guidelines lay out standards for the management of these fractures. Fractures requiring reduction and internal fixation should undergo surgery by a specialist trained in acetabular reconstruction, ideally within 5 days of injury, and no later than 10. Hip dislocations must be reduced urgently, with a pre- and post-reduction neurovascular examination, and skeletal traction applied. Following reduction, a CT scan should be performed within 24 hours and referral should be made to a specialist centre. Thromboprophylaxis should start within 48 hours of injury provided there are no contraindications.

What are the complications of both these fractures and their treatment? Do you know of any factors which predict outcome?

Posterior fractures and dislocations have a high risk of nerve damage and carry a high risk of DVT. Operative fixation, especially though posterior approaches, carries a high risk of heterotopic ossification. With intra-articular fractures, the main risk is joint arthrosis as a late complication. There may also be a risk of AVN to the femoral head, especially in the setting of a hip dislocation.

Viva 4 Questions

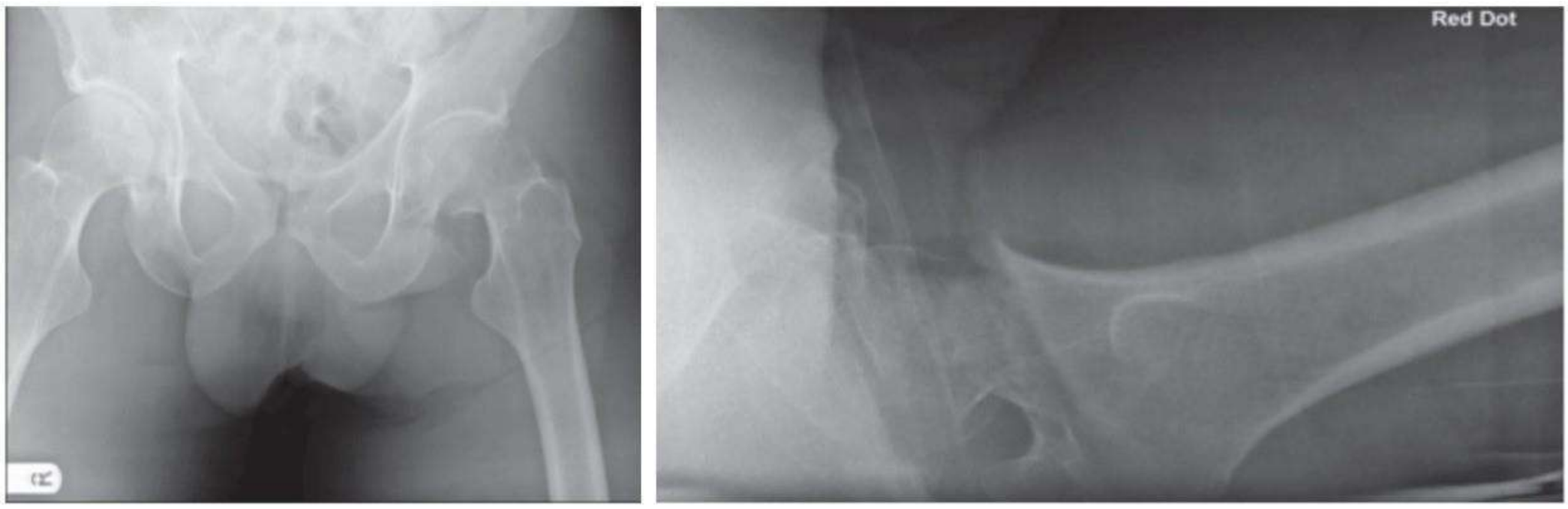

Figure 23.4 Hip AP and Lateral radiograph

Describe what you can see in these radiographs.

How would you manage this patient if he was referred to you by the A&E team?

The patient is a 45-year-old solicitor who sustained this injury whilst mountain biking. He has no other injuries. What would be your management plan for treatment of this fracture?

Could you describe the blood supply to the femoral head?

When consenting this patient for surgery what particular risks would you warn him about?

Can you quote the incidence of these complications and any literature to back this up?

Describe what you can see in these radiographs.

These radiographs show an anteroposterior view of the hips and a lateral view of the left hip. The anteroposterior film shows a displaced intrascapsular fracture of the left neck of femur, this is supported by the appearance on the lateral view confirming the presence of a fracture with displacement and some posterior angulation of the femoral head.

How would you manage this patient if he was referred to you by the A&E team?

I would take a history and examine him, ensuring that the possibility of any other injuries had been ruled out. I would want to know his age and activity level, any comorbidities, and the mechanism of injury. He should be kept nil by mouth and given intravenous fluids and analgesia. Surgery should be performed on the next available trauma operating list, either on the day of admission or the day following admission, as per national guidelines set out by NICE.

The patient is a 45-year-old solicitor who sustained this injury whilst mountain biking. He has no other injuries. What would be your management plan for treatment of this fracture?

The majority of femoral neck fractures occur in the older population and are the result of low-energy injuries. This is not the case in younger patients who are active and have fewer medical problems and better bone quality than their older counterparts. The goals of treatment are to preserve the femoral head and avoid arthroplasty if at all possible. I would therefore treat this patient with closed reduction, with the caveat that only an anatomical reduction is acceptable. I would be prepared to perform an open reduction if necessary and then internal fixation either with cannulated screws or a two-hole DHS with de-rotation screw.

Could you describe the blood supply to the femoral head?

The femoral head blood supply comes from three main sources: the medial femoral circumflex artery, the lateral femoral circumflex artery, and the obturator artery, although in adults the latter may have little to contribute. The lateral femoral circumflex artery gives off an ascending branch, the inferior metaphyseal artery, and supplies most of the inferoanterior aspect of the femoral head. The largest contributor to the blood supply is the lateral epiphyseal artery, which originates from the medial femoral circumflex artery and courses along the posterosuperior aspect of the femoral neck before supplying the femoral head.

When consenting this patient for surgery what particular risks would you warn him about?

Apart from the risks associated with anaesthetic, the main risks specific to this operation that the patient should be informed about prior to surgery are avascular necrosis (AVN) and non-union.

Can you quote the incidence of these complications and any literature to back this up?

The incidence of non-union has been reported to range from 0% to 45% and that of AVN from 0% to 86%: these figures were quoted in a 2005 paper by Damany et al. in *Injury* on a meta-analysis of

Viva 5 Questions

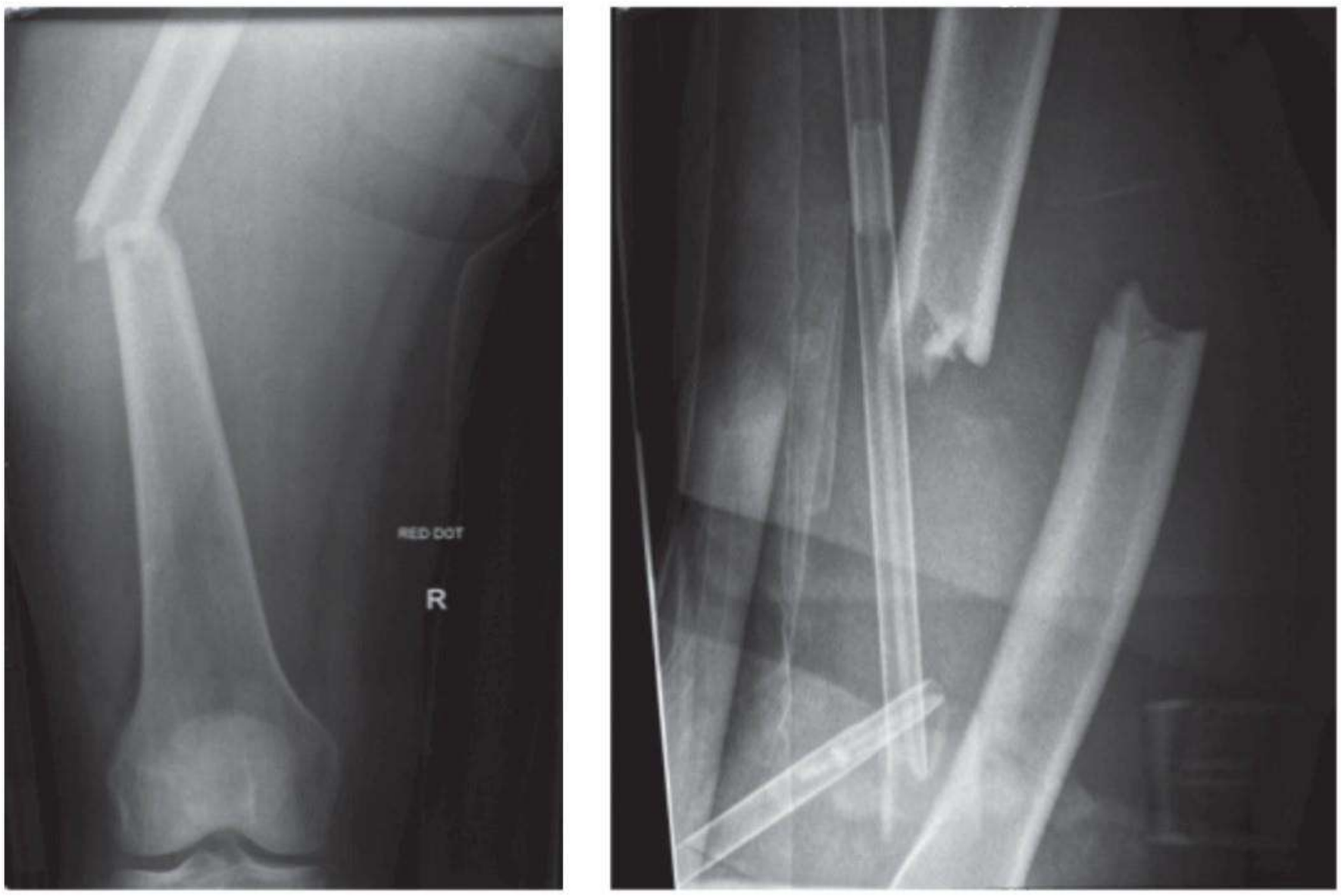

Figure 23.5 Femoral radiographs

A 25-year-old motorcyclist is hit by a car. These are the radiographs taken in the resuscitation bay. How would you manage this patient?

How would you set up the patient? What if any problems would you anticipate?

Are there any situations in which you might consider alternative treatments?

The post-operative films show a fracture of the femoral neck not supported by the interlocking antegrade nail. How has this happened?

A 25 year-old motorcyclist is hit by a car. These are the radiographs taken in the resuscitation bay. How would you manage this patient?

The radiographs show a displaced midshaft fracture in a high-energy injury. The patient should initially be treated according to ATLS protocols to identify life-threatening injuries first. As an isolated injury, the overlying soft tissue envelope and distal neurovascular status should be examined. A complete set of radiographs to include the ipsilateral hip and knee should be obtained, and the remainder of the limb examined for more distal injuries. Then traction should be applied to give temporary stability to the fracture and reduce blood loss and provide analgesia. Definitive treatment should occur promptly, and I would choose to stabilize this fracture with an antegrade reamed intramedullary nail.

How would you set up the patient? What if any problems would you anticipate?

Assuming the patient is adequately resuscitated and prepared for theatre, I would set the patient up on a traction table, with the affected leg raised and the left leg scissored down to allow access by the image intensifier to both the hip and knee. The patient's hips should be brought to the right side of the table, and the shoulders as far left as is safe to provide the best access for entry point and instrumentation. Looking at the initial radiographs, my first operative concern would be reduction of the fracture, as the injury has led to wide displacement and there is the risk of soft tissue interposition. I would apply sufficient traction to overcome any shortening and assess whether it is possible to reduce the fracture closed either with external pressure, a crutch, or by using an instrument such as the 'F' tool. A Schanz pin could also be used to manipulate either fragment and could safely be inserted from the lateral side. If these techniques do not aid fracture reduction, I would open the fracture site and remove any interposed tissues.

Are there any situations in which you might consider alternative treatments?

In certain situations, largely related to polytrauma, it might be wise to consider a damage control approach involving temporary stabilization of the bone with an external fixator, with a plan to convert this to definitive fixation at a later date. This decision depends on both the physiological status of the patient and the presence of concomitant injuries. In such cases the more prolonged surgery and physiological effects associated with instrumentation of the femoral canal for reamed nailing might prove an unwanted extra physiological hit to the patient and worsen their condition. I would consider alternatives to definitive reamed nailing in instances such as multiple long bone fractures, significant chest injury, and significant closed head injuries. Physiological parameters that might give an indication of the potential need for damage control surgery include serum lactate, which can be easily measured from venous samples on blood gas machines. Bilateral femoral fractures have been shown to be a significant risk factor for the development of systemic inflammatory response syndrome (SIRS) and acute respiratory distress syndrome (ARDS). In polytrauma patients with multiple injuries, I would consider a Damage Control Approach also.

The post-operative films show a fracture of the femoral neck not supported by the interlocking antegrade nail. How has this happened?

This could have been a missed injury from the initial presentation or it may have been a complication of the nailing procedure. Femoral neck fractures may occur in up to 10% of high-energy

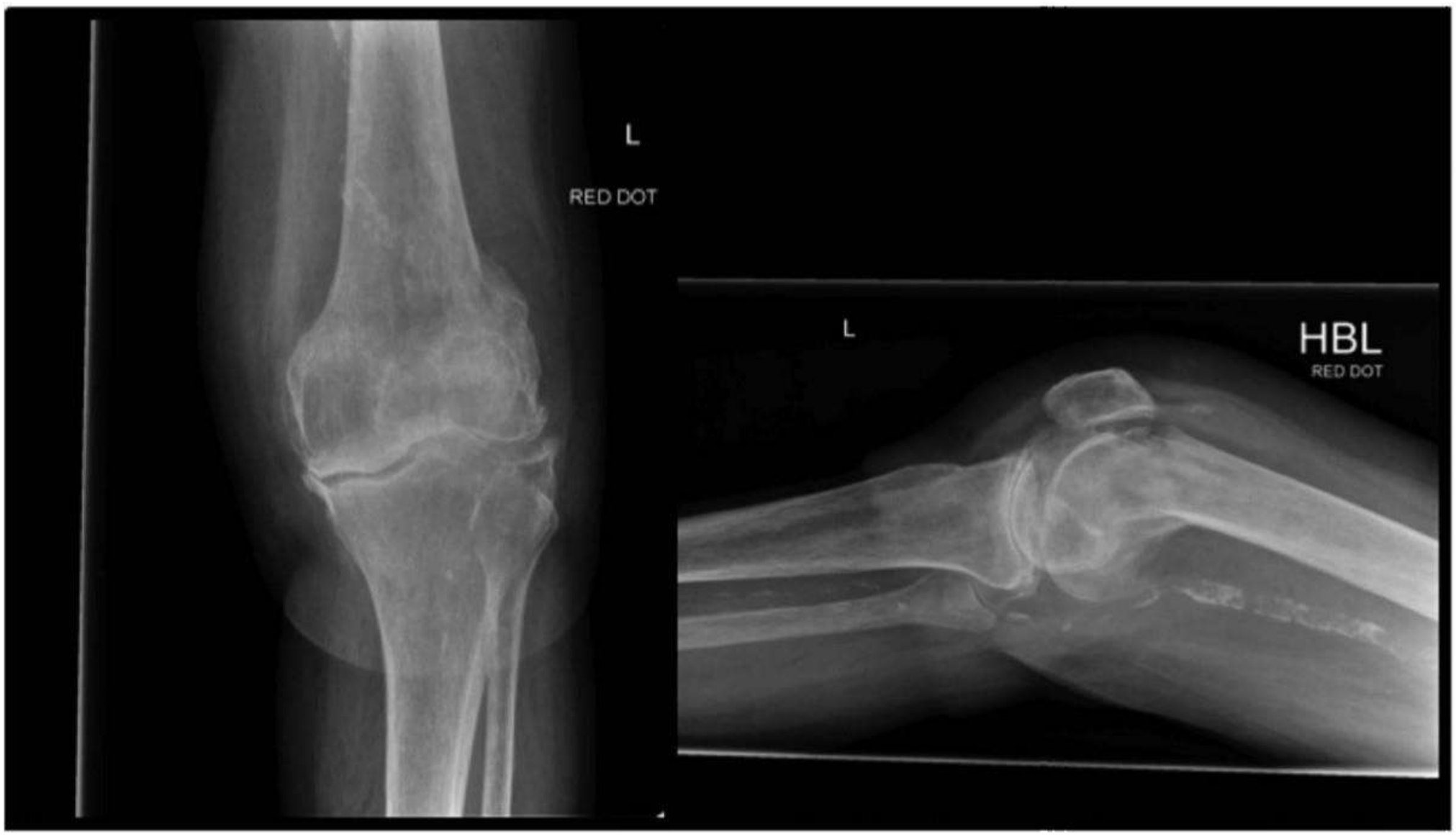

Figure 23.6 AP and Lateral radiograph of knee

Describe these radiographs of a 67-year-old man who fell outside his home on a patch of ice. How are you going to treat him?

What are the challenges and pitfalls of plate fixation?

How would your surgical technique change if you were treating this fracture in a 30-year-old motorcyclist?

Describe these radiographs of a 67-year-old man who fell outside his home patch of ice. How are you going to treat him?

These are anteroposterior and lateral views of a left knee showing a supracondylar fracture of the distal femur. The distal fragment has flexed. In addition there is significant degenerative disease in the knee, and notable calcification in the popliteal vessels.

Provided the patient's general condition does not preclude it, I would elect for surgical intervention. Non-operative treatment would require prolonged immobilization of what is likely an already stiff joint, and in addition to the potential for medical complications there is also a risk that the fracture will continue to displace and potentially threaten the overlying soft tissue envelope. It would also be difficult to control the fracture because the arthrosis in the knee will make the joint stiff and there would be a tendency for any movement to preferentially occur at the fracture site, increasing the risk of both further fracture displacement and non-union.

Broadly speaking, the surgical options here are internal fixation or primary replacement. If this knee had been relatively symptom free prior to the accident I would elect for fixation through a lateral approach with relative stability, using a periarticular locking plate in bridging mode in order to achieve union. Plating would not preclude total knee replacement at a later date if required. However, if the knee had been symptomatic, it may be more appropriate to perform primary arthroplasty. Because of the position of the fracture, this would be likely to involve a distal femoral replacement with a constrained articulation such as a rotating hinge. Thus, this decision should involve a surgeon with the necessary expertise as well as an analysis of the physiological capacity of the patient to withstand this procedure.

What are the challenges and pitfalls of plate fixation?

My preference when plating the femur in fractures in the elderly is to use as long a plate as possible, spanning the whole femur, and to use minimally invasive techniques if appropriate. In this case a small distal incision to allow the plate to be inserted will be needed. It is essential that this allows the iliotibial band to be divided as the plate must sit deep to this—if it is not deep to this layer, the patient will suffer from irritation and prominent metalwork. When plating, it is also essential that the plate is orientated correctly—a 95° wire through the central distal hole of almost all plates should be parallel to the joint on the anteroposterior projection. The plate also sits at an inclined angle on the lateral femoral cortex matching the shape of the native femur; it is important to recognize this otherwise my distal screws will aim anteriorly and penetrate the patellofemoral joint. Proximally it is essential that the plate sits along the mid-axis of the shaft and not too anterior or posterior—this is a common error in percutaneous plating. The number of screws and their configuration are also controversial—distally I prefer to use as many locked screws as possible in my construct, effectively filling the metaphysis with metal. Proximally I prefer a near–far pattern of locked screws, with a total of at least five or six screws. Previous studies suggested a unicortical or non-locked screw at the tip, but in my practice a bicortical screw is preferable.

How would your surgical technique change if you were treating this fracture in a 30-year-old motorcyclist?

In high-energy distal femoral fractures in younger people there is a risk of significant intra-articular involvement, and anatomical restoration of the joint surface is required in addition to fixation to the femoral shaft. Prior to surgery I would obtain a CT scan to allow me to better define the articular

Viva 7 Questions

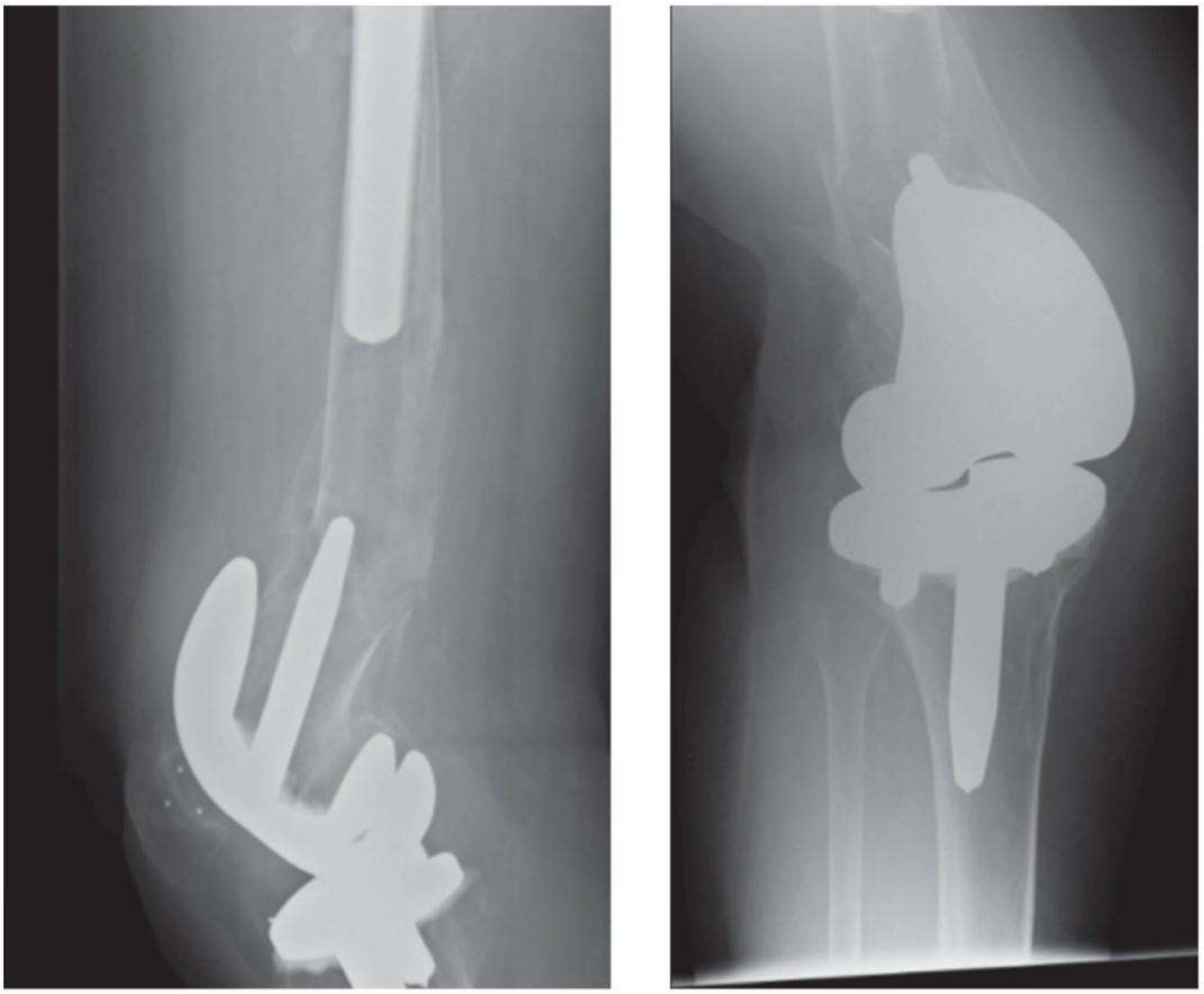

Figure 23.7 AP and Lateral radiograph of knee

What do these radiographs show?

Can you classify this fracture?

Can you describe the Vancouver classification?

How does this classification help the surgeon decide the best intervention for treatment of the fracture?

What are your treatment options for this injury, assuming further imaging shows the femoral component of the total hip replacement to be well fixed?

Are you aware of any literature describing the treatment of periprosthetic fractures?

Viva 7 Answers

What do these radiographs show?

These are anteroposterior and lateral views of the distal femur. There is a long femoral stem visible from a total hip prosthesis but also a short-stemmed total knee prosthesis distally within the femur. The radiographs show that a periprosthetic fracture has occurred within the distal femur from the level of the tip of the stem on the knee prosthesis and extends proximally, it is not possible to tell on these radiographs how far proximally the fracture extends. The bone has an osteopenic appearance.

Can you classify this fracture?

Using the Vancouver classification for periprosthetic fractures around a hip prosthesis this would be a Vancouver B fracture. Periprosthetic fractures around the knee in the supracondylar region of the femur have been classified by Lewis and Rorabeck; this would be a type II fracture using their classification.

Can you describe the Vancouver classification?

This classification system describes fractures around a hip prosthesis. Type A fractures are around the trochanters and are subdivided into groups A_G and A_L for involvement of the greater and lesser trochanters, respectively. Type B fractures are around the stem, with further subdivision depending on solid fixation or not of the implant and whether the bone stock is deficient: B1 is a well-fixed stem, B2 is a loose stem, and B3 is deficient bone stock. Type C fractures are distal to the prosthesis.

How does this classification help the surgeon decide the best intervention for treatment of the fracture?

The Vancouver classification helps the surgeon to focus on the environment in which the fracture has occurred. If the stem is well fixed, as with type B1 injuries, the fracture can generally be treated leaving the prosthesis *in situ* using an onlay cortical strut graft and a suitable plate that enables the use of screws and cables. B2 fractures are around a stem that was not solidly fixed at the time of injury, so the treatment of these fractures would normally require revision of the femoral stem to a prosthesis that bypasses the fracture by two cortical diameters. Type B3 fractures occur in the presence of insufficient bone stock and can be particularly difficult to treat. The literature describes the use of a stem with diaphyseal fit that bypasses deficient proximal bone stock, distally locked stems, or the use of prostheses that replace the proximal femur. Type C fractures can be treated independently of the prosthesis.

What are your treatment options for this injury, assuming further imaging shows the femoral component of the total hip replacement to be well fixed?

This is an extremely difficult fracture to treat. In my centre these injuries are referred to our regional specialist arthroplasty and complex trauma service. If the femoral stem from the total hip replacement is well fixed it should be left in situ, especially as there appears to be poor bone stock around the stem. The knee prosthesis also appears well fixed. The bone quality between the two prostheses is very poor. The presence of a stem on the femoral component of the knee would make any attempt at extramedullary fixation using a plating system difficult, and proximal fixation will have to rely on cables alone. This would be my preferred choice but I would be worried about

Viva 8 Questions

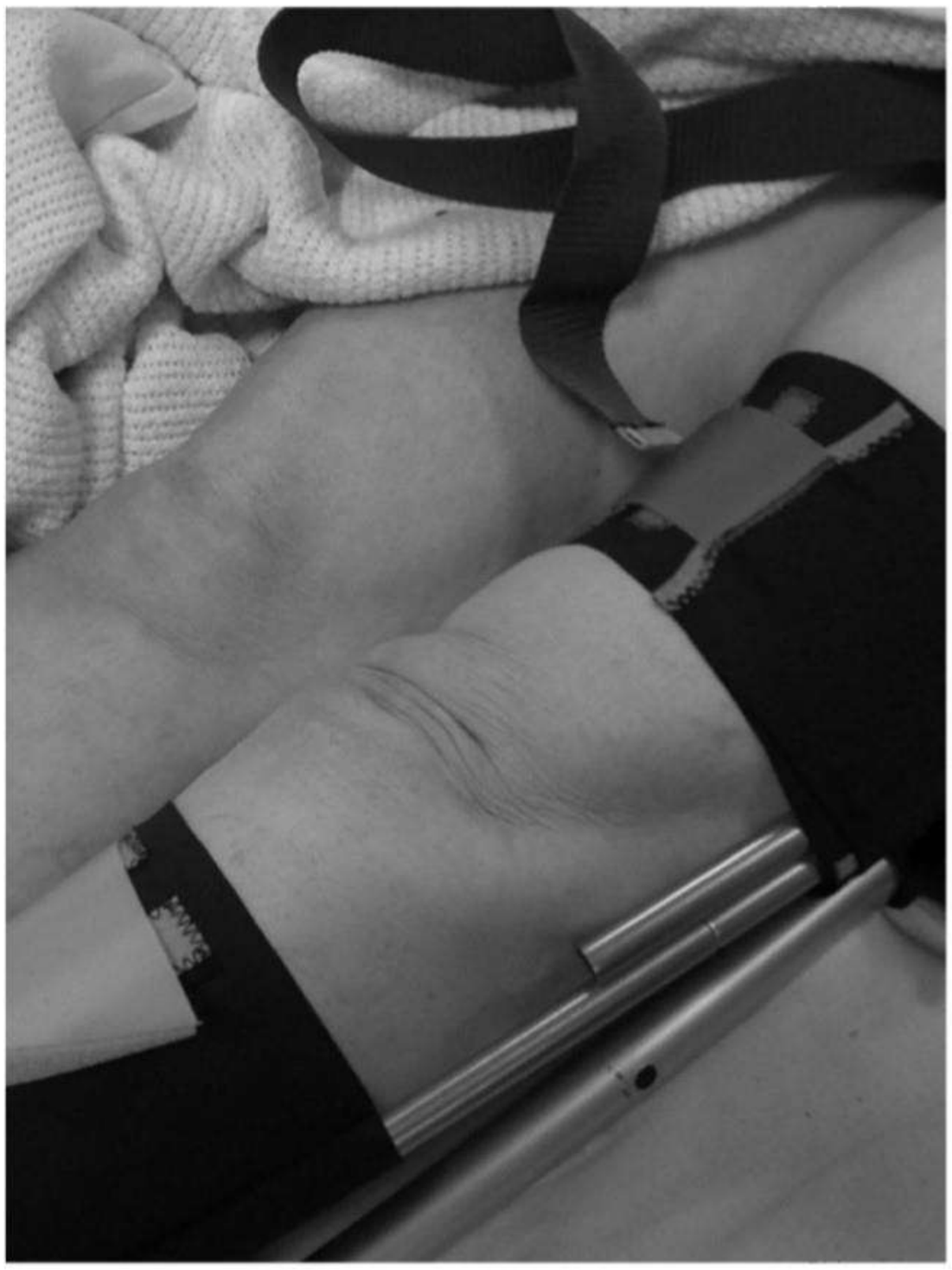

Figure 23.8 Clinical photograph of a left knee injury

This is a photograph of the knee of a 35-year-old woman who has come off her motorcycle on her way to work. What can you see?

How would you manage this injury?

There was a weak pulse prior to reduction, and the foot was pink. You have successfully reduced the tibia; what would you do next?

The pulse is still present. There is no foot drop, but due to head and chest injuries and concerns about the airway the anaesthetists have intubated the patient and she is now fully sedated. The radiograph shows a fibula head avulsion fracture only. What is your major concern and what is your management now?

How would you measure the ABI?

The patient's ABI remains above 0.9 and the foot continues to be well perfused. Three days later the patient is awake. You arrange a MRI scan which shows an almost 'full house' of ligament injuries with only the MCL partially intact. What are your priorities for repair or reconstruction and the timing of these surgeries?

Viva 8 Answers

This is a photograph of the knee of a 35-year-old woman who has come off her motorcycle on her way to work. What can you see?

The left leg is splinted. There is a dislocation of the tibiofemoral joint of the knee with the tibia dislocated anteriorly in relation to the tibia. It appears to be a closed injury.

How would you manage this injury?

This is a high-energy injury and the patient should be appropriately assessed and resuscitated according to the ATLS protocol. While the primary survey is being completed I would assess the neurovascular status of the distal limb, which I would record carefully. If there are no other life-threatening injuries, the patient is stable, and appropriate analgesia is given, I would attempt closed reduction of the knee. I would perform this with gentle in-line traction on the foot and posterior pressure on the anterior part of the proximal tibia.

There was a weak pulse prior to reduction and the foot was pink. You have successfully reduced the tibia; what would you do next?

I would reassess the neurovascular status and document my findings; if I was happy there was distal blood flow I would arrange immobilization of the limb in a backslab or brace and then request radiographs.

The pulse is still present. There is no foot drop, but due to head and chest injuries and concerns about the airway the anaesthetists have intubated the patient and she is now fully sedated. The radiograph shows a fibula head avulsion fracture only. What is your major concern and what is your management now?

Popliteal artery damage is my major concern. There has been a recent shift from routine arteriography to selective arteriography in knee dislocation. Selective arteriography would suggest the need in cases of abnormal distal examination findings. Recent animal and human studies have shown that non-flow-limiting intimal tears rarely progress to occlusive thrombi. In this case it seems that the patient's flow is not occluded as the foot is pink and a weak pulse can be felt, so I would elect for regular measurement of the ankle–brachial index (ABI), which can be done in intensive care.

How would you measure the ABI?

With the patient supine a blood pressure cuff is placed proximal to the ankle of the injured limb. Systolic pressure is determined with a Doppler probe at either the posterior tibial artery or the dorsalis pedis artery. The same measurement is made on the ipsilateral uninjured arm. The ABI is calculated as the systolic pressure of the injured limb divided by the systolic pressure of the uninjured limb. Mills et al. reported 100% specificity; sensitivity, and positive predictive value of a significant arterial injury if this pressure difference is less than 0.9. Other series have suggested a threshold of 0.8.

I would consider arranging an urgent arteriogram and calling a vascular surgeon for advice if the clinical examination changed or the ABI, having been normal, dropped to below 0.9.

Viva 9 Questions

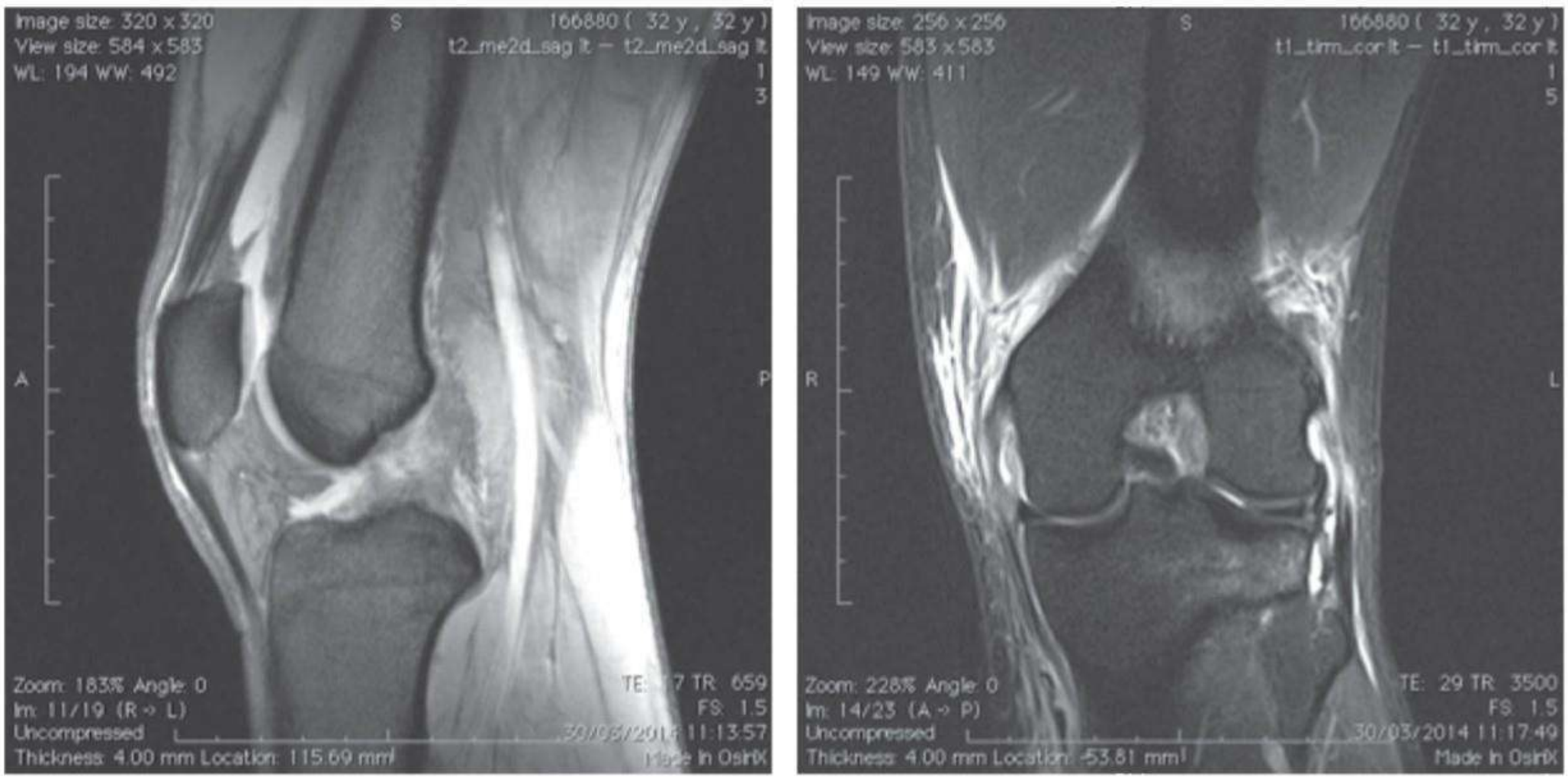

Figure 23.9 Knee MRI

These are MRI scans of the injured knee of an 18-year-old elite junior netball player. She heard a large pop when she tried to change direction. What do the scans show?

How would you treat this injury?

When would you perform reconstruction?

What are the graft choices for ACL reconstruction?

What would be your choice and why?

What are the key steps in performing an ACL reconstruction?

What fixation would you use?

What would your rehabilitation programme be, and when would you allow her to return to playing netball?

Viva 9 Answers

These are MRI scans of the injured knee of an 18-year-old elite junior netball player. She heard a large pop when she tried to change direction. What do the scans show?

These are T_2 MRI scans showing sagittal and coronal views through the knee. I can see a ruptured ACL and a large bone bruise on the posterolateral tibial plateau consistent with the dislocation of the lateral femoral condyle that occurs during this injury. There is some increased signal around the structures of the posterolateral corner and MCL but they look grossly intact.

How would you treat this injury?

I would take a history and examine the patient looking for other ligament injuries, particularly the MCL. If there was no MCL injury I would give the patient crutches but no brace, allow her to bear weight as tolerated, and arrange for a physiotherapist to see her. I would recommend the application of ice twice a day and gentle range-of-movement exercises. I would then discuss reconstruction with her.

When would you perform reconstruction?

Reconstruction should only be performed when the knee is pain free and mobile. It usually takes around 6 weeks for full motion to return.

What are the graft choices for ACL reconstruction?

Autograft in the form of hamstring tendon, bone–patellar tendon–bone, quadriceps tendon. Allograft should preferably be non-irradiated and is usually Achilles tendon, patellar tendon, or tibialis anterior. Artificial ligaments such as the LARS ligament are available.

What would be your choice and why?

I would use a four-strand hamstring tendon. It is a low-morbidity harvest and has excellent 15-year results. Bone–patellar tendon–bone is reliable with good 15-year results too, but some people have concerns over pain in the anterior knee and on kneeling. I would avoid the LARS ligament as some surgeons have serious concerns about the long-term effects on the joint.

What are the key steps to performing an ACL reconstruction?

(1) A low-morbidity harvest, minimizing soft tissue damage. (2) A short arthroscopy time, preserving as best as possible the fat pad and other intra-articular structures. (3) Accurate anatomical tunnel placement including femoral tunnel drilling through the anteromedial portal. (4) Robust fixation devices for the graft.

What fixation would you use?

I would use round cannulated interference (RCI) screws in both femoral and tibial tunnels. They have been shown to be reliable, have low morbidity, and combined with hamstring tendon graft have excellent 15-year results. I would use a suspension device on the femoral side first, however, in order to allow for accurate tensioning of the graft, before additional fixation with the femoral RCI screw.

What would your rehabilitation programme be, and when would you allow her to return to playing netball?

I would not use a brace, and would allow her to fully weight-bear and to begin gentle exercise such as cycling after the first week. Gentle running can begin at 8 weeks, and I would see her at 3 and 6 months with a view to allowing netball training to start at 6 months. She should not return to competition before 9 months, and then only if she had met the rehabilitation goals.

Bourke HE, Gordon DJ, Salmon LJ, Waller A, Linklater J, Pinczewski LA (2012). The outcome at 15 years of endoscopic anterior cruciate ligament reconstruction using hamstring tendon autograft for 'isolated' anterior cruciate ligament rupture. *J Bone Joint Surg Br*, 94, 630–637.

Viva 10 Questions

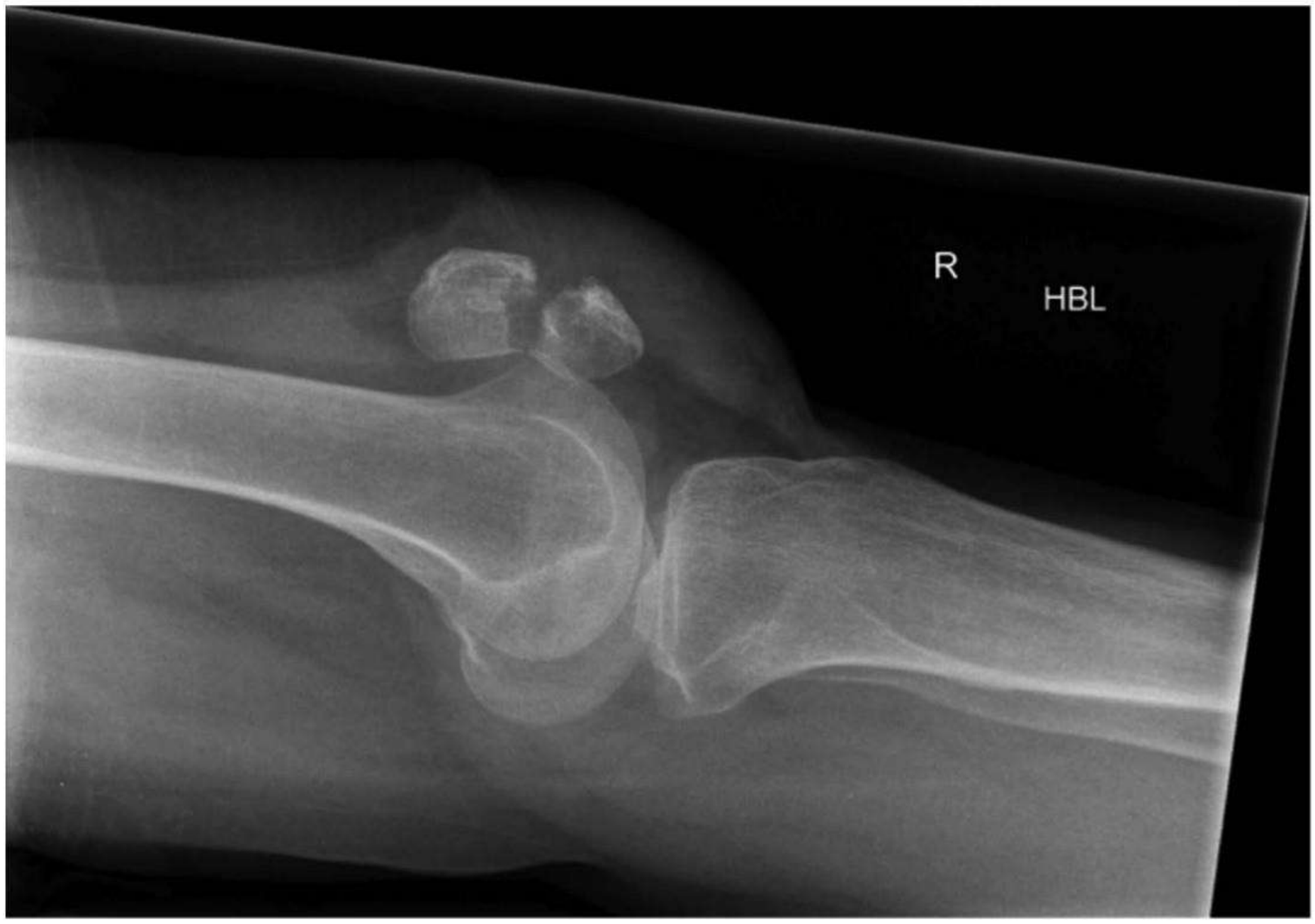

Figure 23.10 Lateral radiograph of knee

What does this radiograph show?

This is a closed isolated injury in a 45-year-old male patient. What is your management?

Talk me through your fixation technique, concentrating on the biomechanical principles.

What is your on-going management following this procedure?

What are the potential complications of this procedure?

Viva 10 Answers

What does this radiograph show?

This radiograph demonstrates a completely displaced transverse patella fracture in a skeletally mature patient.

This is a closed isolated injury in a 45-year-old male patient. What is your management?

This injury requires operative fixation to restore the extensor mechanism of the knee joint. I would immobilize the patient in a cast or brace for comfort and provide adequate analgesia. I would then arrange to admit the patient for surgery on the next available trauma list.

Talk me through your fixation technique, concentrating on the biomechanical principles.

[THE EXAMINER WILL OFTEN VOLUNTEER A PENCIL AND PAPER AT THIS POINT. IF HE OR SHE DOES NOT, THEN REACH FOR THE PENCIL AND PAPER (IF AVAILABLE) FOR THE BEST DESCRIPTION OF TENSION-BAND WIRING.]

I would utilize a tension-band wiring technique. In an appropriately consented and anaesthetized patient, exposure to the patella should be achieved through a midline longitudinal incision. Fracture haematoma should be thoroughly washed to clearly expose the fracture fragments. All interposing soft tissue should be removed from the fracture edges. Two 1.6-mm K-wires with points at both ends should be drilled into the fracture side of the superior bone fragments, evenly spaced and in parallel in an axial direction until the tips just disappear into the bone. With the knee in full extension the patella can then be reduced anatomically and temporarily held with large pointed reduction forceps with their tips on the inferior and superior poles. The K-wires can then be driven through the inferior poles and out of the soft tissue. Figure-of-eight tension-band wiring can then be achieved with 1.25-mm thick steel wire, keeping the wire as close to the bone as possible. This can be achieved with the help of a curved large-bore injection needle to pass the wire through the soft tissue. Tensioning is achieved through a single loop which should be buried in the medial or lateral retinacular tissue as best as possible to minimize later discomfort. I would bend the superior ends of the K-wires over and bury them in the patella and then cut the inferior ends flush with the soft tissue of the patellar tendon.

The biomechanical principles of this technique are that the greatest tension through the fracture occurs on the most anterior aspect while the quadriceps muscle exerts a force on the tendon and the patella within. The tension band lies on the anterior aspect of the patella. Steel wire, which is strong in tension, resists this force and transfers the energy via the fixation from the K-wires at the superior and inferior poles and to the posterior aspect of the patella, where it acts as a compressive force at the articular surface. Compression forces across a fracture stimulate direct bone healing.

It is also essential to augment the tension band with a strong repair of the extensor retinaculum. This is almost always torn in these cases, and repairing this will protect my fixation and allow for improved outcomes.

What is your on-going management following this procedure?

I would immobilize this patient's knee in a hinged knee brace locked from 0–45° and allow full weight-bearing as tolerated. The brace should stay on for a minimum of 6 weeks. Following this I would repeat the radiographs, and subject to satisfactory progress, ask a physiotherapist to begin

range-of-movement exercises to avoid stiffness. I would perform a second procedure to remove the wires at 6 months, as symptomatic hardware is among the most common complications of this procedure.

What are the potential complications of this procedure?

Early complications include infection, wound breakdown, rapid failure of fixation, and VTE. Intermediate complications include symptomatic hardware, quadriceps weakness, knee stiffness, and non-union (rare). Late issues would be conditions such as extensor lag and late-onset osteoarthritis of the patellofemoral joint from inadequate reduction.

AO Foundation. *AO Surgery Reference*. Patella 34–C1.3. Open reduction; tension band wiring. https://www2.aofoundation.org/wps/portal/surgery?showPage=redfix&bone=Knee&segment=Patella&classification=34-C1.3&treatment=&method=ORIF%20-%20Open%20reduction%20internal%20fixation&implantstype=Tension%20band%20wiring&approach=&redfix_url=1285238823429&Language=en

Melvin JS, Mehta S (2011). Patellar fractures in adults. *J Am Acad Orthop Surg*, 19, 198–207.

Viva 11 Questions

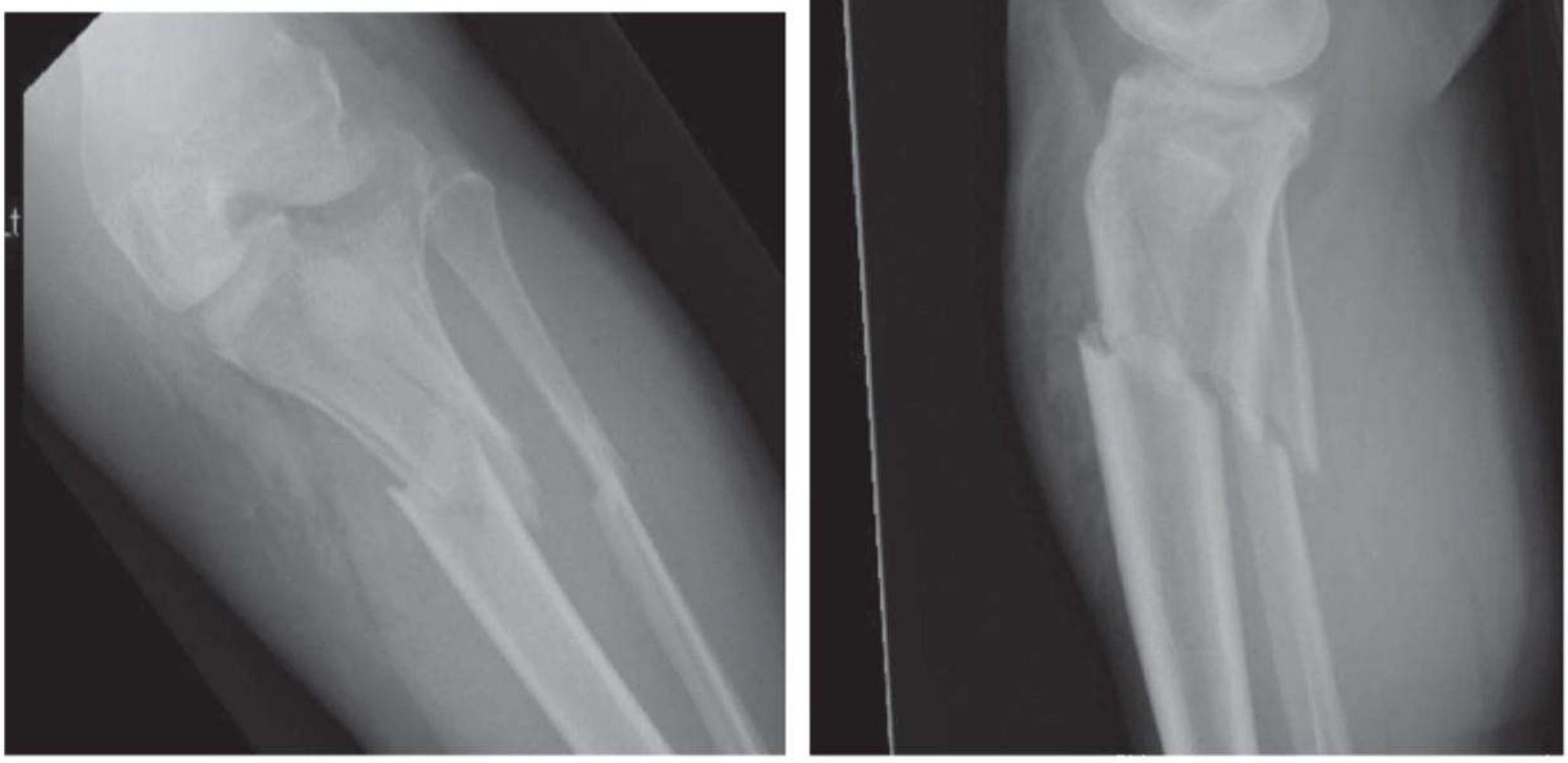

Figure 23.11 AP and lateral radiograph of proximal tibia

These radiographs are of the knee of a man who fell from the seventh rung of a ladder, landing awkwardly on his left knee. A&E has assessed him, and his injury is isolated and closed. Tell me what you can see.

How would you manage this patient's isolated injury?

The knee is grossly swollen and the depression of the joint surfaces has not improved with simple splintage. You decide to perform external fixation. Describe how you would do this.

The CT scan confirms a significant depression laterally, minimal joint involvement medially, and large amounts of comminution in the metaphysis. What is your plan for definitive management?

Describe your operative plan.

What will you tell your patient regarding the likely outcome and need for knee replacement in the future?

These radiographs are of the knee of a man who fell from the seventh rung of a ladder, landing awkwardly on his left knee. A&E has assessed him, and his injury is isolated and closed. Tell me what you can see.

These are anteroposterior and lateral views showing a displaced bicondylar tibial plateau fracture. There is no intact communication between the articular surface and the tibial diaphysis, so this is a Schatzker VI pattern injury or a type C3 fracture in the AO terminology, indicating both metaphyseal and articular comminution. There is significant depression of the lateral articular surface. The fibula is fractured at the same level as the diaphyseal–metaphyseal dissociation of the tibia.

How would you manage this patient's isolated injury?

This is a high-energy injury. Although it is a closed injury I would have concerns regarding the soft tissue envelope and would assess the patient's limb for distal neurovascular deficit or signs of compartment syndrome. Initial management would include appropriate analgesia and immobilization of the limb in an above-knee backslab. The patient requires admission and, depending on the degree of soft tissue injury and the position of the knee on a radiograph, he might also benefit from bridging external fixation.

The knee is grossly swollen and the depression of the joint surfaces has not improved with simple splintage. You decide to perform external fixation. Describe how you would do this.

I would prepare the patient for surgery by gaining informed consent, ensuring he is starved, and by arranging the theatre and anaesthetic support. Once supine and prepared for operation, with no tourniquet, I would place two anteroposterior Schanz pins in the distal femoral diaphysis. This can be done percutaneously. I tend to start the pin on power and then complete it manually to optimize feedback on the far cortex. In the tibia I would place my pins distal to the zone of injury, aiming anterior-to-posterior with my entry at the lateral aspect of the palpable tibial crest. This ensures the pins will not impede the later placement of plates if internal fixation is proposed. The frame is built off these pins and the fracture reduced manually under fluoroscopic control before the construct is tightened with the knee in slight flexion. I would add bars and pins as needed to ensure a stable construct, and ideally use a system that offers 11- or 12-mm bars for optimal stability. Postoperatively I would again check the neurovascular status of the limb, begin DVT prophylaxis, and monitor for compartment syndrome. I would arrange a fine-slice CT scan with reconstructions to plan definitive surgery. It may take several days for the soft tissues to settle, so I would anticipate performing definitive fixation a week or more after injury.

The CT scan confirms a significant depression laterally, minimal joint involvement medially, and large amounts of comminution in the metaphysis. What is you plan for definitive management?

The best option for fixation in these high-grade injuries remains controversial. Outcomes appear to be comparable between circular frame fixation and internal fixation. The key determinant for success is restoration of the mechanical axis. The key feature to aid decision-making about what implant to use is the state of the soft tissues. In this case, in my practice, the pattern would be amenable to limited internal fixation of the articular surface with screws, and circular frame fixation for the meta-diaphyseal component. This allows wires to be kept away from the joint, but avoids

Viva 1 Questions

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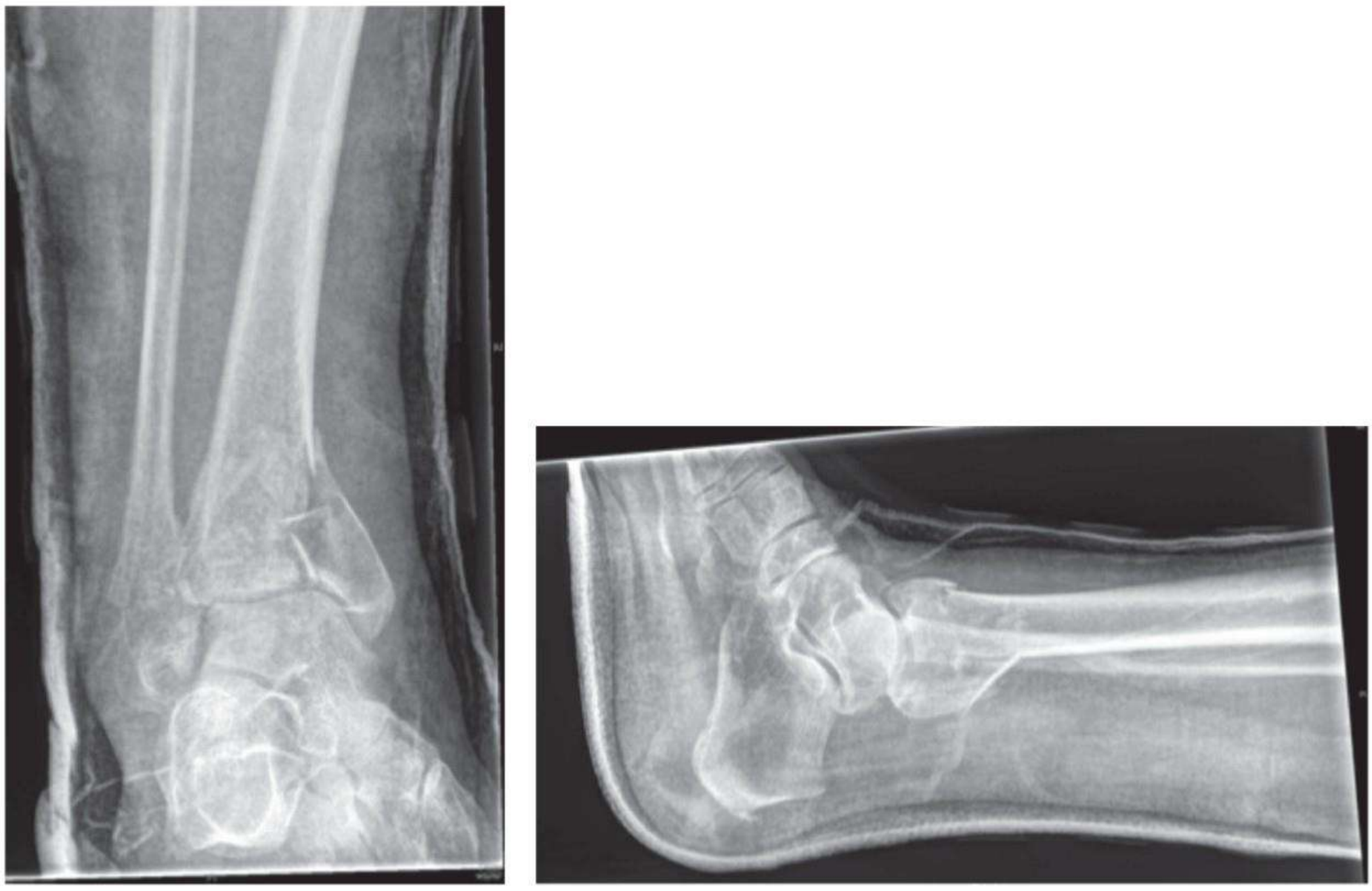


Figure 24.1 AP and Lateral ankle radiograph

Describe what these radiographs show.

How would you proceed?

This represents the main injury in a stable patient. How would you manage this fracture?

This is a closed injury with no gross neurovascular concerns but significant soft tissue injury. How would you proceed?

Is CT scanning necessary given the pre-operative films and fluoroscopic examination?

What external fixator configuration would you use? Are there alternatives?

What are the principles of definitive fixation?

What are the outcomes of pilon fractures?

Viva 1 Answers

Viva 1 Answers

Describe what these radiographs show.

These are anteroposterior and lateral views of the distal tibia and ankle joint. There is a complex, intra-articular, multifragmentary fracture extending proximally into the metaphysis. There is significant proximal migration of the talus. There is no obvious air in the soft tissues, indicating that this is an open injury, although the soft tissue component and zone of injury will be extensive. This is a pilon fracture.

How would you proceed?

This is a high-energy injury. I would proceed following ATLS protocols to identify immediately life-threatening injuries and deal with these in a logical hierarchal sequence using the ABCDE (airway, breathing, circulation, disability, exposure) approach. I would initiate a trauma call to summon help.

This represents the main injury in a stable patient. How would you manage this fracture?

I would take full history and examination, concentrating on important risk factors, for example smoking, diabetes, and peripheral vascular disease. Examination would assess soft tissue integrity and neurovascular status with full documentation.

This is a closed injury with no gross neurovascular concerns but significant soft tissue injury. How would you proceed?

This injury requires reduction and skeletal stabilization to allow resolution of soft tissue before definitive fixation. If the patient remains stable and there are no concerns about anaesthesia I would take the patient to theatre to apply an external fixation, focusing on correction of length, alignment, and rotation. I would then obtain a CT scan in order delineate the fracture further. Once soft tissues have recovered I would consider definitive fixation depending upon the fracture configuration. This is the staged management protocol described by Sirkin, and represents the gold standard management algorithm for complex intra-articular fractures of the tibial plafond.

Is CT scanning necessary given the pre-operative films and fluoroscopic examination?

Yes, CT scanning is necessary. The scan improves recognition of fracture fragments as described by Topiliss, and helps in planning incisions to minimize additional trauma to soft tissues and maintain their viability. CT scans change management in approximately two-thirds of cases and reduce operating time in three-quarters of cases.

What external fixator configuration would you use? Are there alternatives?

I would use a simple 'A' frame, two pins in the tibia away from the zone of injury, and a pin through the calcaneus. There are many alternatives, including fixation in calcaneus and talus, supplemental first metatarsal fixation to avoid equinus problems, quadrilateral frame, hybrid frames, and fine wire frames.

What are the principles of definitive fixation?

The principles are anatomical reduction and rigid fixation of the articular components, and restoration of length, alignment, and rotation of the meta-diaphyseal components, in order to restore anatomy and function. Careful technique is essential to preserve blood supply to fracture fragments.

What are the outcomes of pilon fractures?

After the initial success of Rüedi and Allgöwer and their principles of fixation the complication rate rose significantly in the hands of non-experts. Multiple studies reported high complication rates (30% non-union, malunion, infection). However, the evidence is beginning to suggest that in appropriate centres with experienced surgeons and appropriate techniques (staged, early intervention, limited reduction) the complication rate is improving, with excellent clinical outcomes (2.7% complication in closed fractures).

Boraiah S, Kemp TJ, Erwteman A, et al. (2010). Outcome following open reduction and internal fixation of open pilon fractures. *J Bone Joint Surg Am*, 92, 346–352.

Liporace FA, Yoon RS (2012). Decisions and staging leading to definitive open management of pilon fractures: where have we come from and where are we now? *J Orthop Trauma*, 26, 488–498.

Tornetta P III, Gorup J (1996). Axial computed tomography of pilon fractures. *Clin Orthop Relat Res*, Feb(323), 273–276.

White TO, Guy P, Cooke CJ, et al. (2010). The results of early primary open reduction and internal fixation for treatment of OTA 43. C-type tibial pilon fractures: a cohort study. *J Orthop Trauma*, 24, 757–763.

Viva 2 Questions

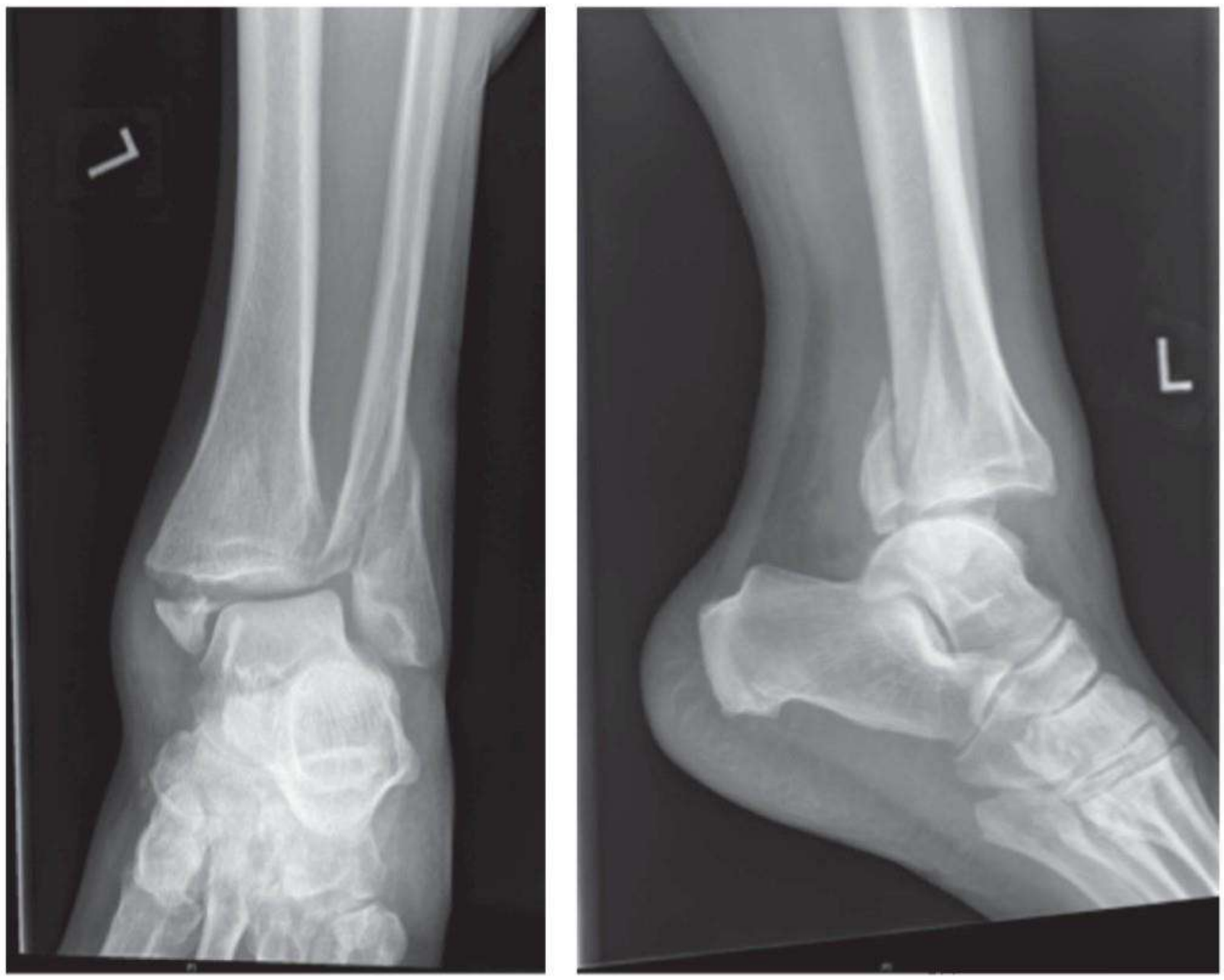


Figure 24.2 AP and Lateral ankle radiograph

Describe these radiographs.

This is an isolated injury that is closed and neurovascularly intact. How would you manage it?

What is the mechanism of the injury? Can you classify the injury?

What is the difference between SER II and IV injuries, and how can you diagnose this?

What is your definitive management for this patient and why?

Do you know the current literature on the gravity stress view?

Viva 2 Answers

Describe these radiographs.

These are anteroposterior and lateral views of a skeletally mature left ankle. There is a trimalleolar fracture with talar shift approaching 50%. The fibular is a spiral fracture starting at the level of, and extending below the level of, the syndesmosis and the medial side is a transverse fracture. There is a posterior malleolar fragment—this only carries a small part of the articular surface but probably confers significant instability in this instance due to the attachment of the posterior inferior tibiofibular ligament (PITFL).

This is an isolated injury that is closed and neurovascularly intact. How would you manage it?

This injury needs immediate reduction in the emergency department under appropriate analgesia or sedation to prevent compromise of the medial side. I would stabilize the reduction in a below-knee plaster of Paris cast, obtain radiographs to confirm adequate reduction, and arrange admission for definitive fixation.

What is the mechanism of the injury? Can you classify the injury?

There are several classifications for ankle factures based on either the mechanism (Lauge-Hansen) or anatomical description (Weber) of the injury. The Weber classification relates to the fibular with type A below the syndesmosis, type B at the level of the syndesmosis, and type C above. A more detailed system is that proposed by Lauge-Hansen based upon cadaveric work. The first term represents the position of the foot and the second to the force placed upon the foot and talus in the ankle mortise. There are four main types: supination–adduction, supination–external rotation (SER), pronation–abduction and pronation–external rotation (PER). Dorsiflexion patterns are also described and relate more to Pilon fractures than ankle injuries.

In this case, the fibula has a spiral pattern, indicating a rotational force, so this could be PER or SER. However, the fibula fracture runs from the anterior inferior to posterior superior direction, which would be more in keeping with a SER pattern. In a PER pattern the spiral starts anterior superior to the posterior inferior position. The supination–adduction pattern tends to have a vertical orientation to the medial malleolus and pronation–abduction tends to have a fibular fracture above the syndesmosis and a transverse or short oblique fibular fracture due to the bending force. Supination external rotation injuries begin anteriorly, with rupture of the anterior inferior tibiofibular ligament (AITFL), then the fibula fractures followed by either a posterior malleolus fracture or PITFL rupture. The last structure to fail is the deep deltoid or medial malleolus. Therefore this fracture is a SER IV injury.

What is the difference between SER II and IV injuries, and how can you diagnose this?

The difference is essentially whether the medial structures, i.e. the deep deltoid and the medial malleolus, are intact; these structures play a significant part in the stability of the ankle joint. The absence of pain and tenderness on the medial side is a poor indicator of the integrity of these structures. The gravity stress view has been developed to help confirm the integrity of the deltoid, with a medial clear space of more than 5 mm being suggestive of medial injury. Furthermore, the presence of a posterior injury (SER III) means this has to be more than an SER II.

What is your definitive management for this patient and why?

Given no contraindications to surgery, my definitive management would be operative. I would perform ORIF, either before swelling has occurred or allow swelling to reduce, as long as the fracture is in a satisfactory position. Using a tourniquet, and with antibiotic prophylaxis, I would fix the posterior malleolus and the lateral side though a posterolateral approach. The posterior fixation would be a buttress plate and additional screw, with an anti-glide plate plus supplementary interfragmentary screws for the fibula. I would address the medial side ideally using two short (40 mm) partially threaded cancellous screws. I would protect the soft tissues in plaster of Paris for 2 weeks and then begin protected weight-bearing for a further 4 weeks. My indications for operative intervention are that this fracture would be very difficult to maintain in position in plaster of Paris due to instability, and for a shift of more than 1 mm in contact area the forces in the ankle joint change significantly, predisposing to post-traumatic osteoarthritis.

Do you know the current literature on the gravity stress view?

Currently, the role of the deep deltoid is being investigated further. Stress views may over-diagnose injuries and partial tears of the deep deltoid may result in positive stress views, but could be successfully treated non-operatively. Weight-bearing mortise views or MRI scans may be more sensitive predictors of instability than stress views.

van den Bekerom MP, Mutsaerts EL, van Dijk CN (2009). Evaluation of the integrity of the deltoid ligament in supination external rotation ankle fractures: a systematic review of the literature. *Arch Orthop Trauma Surg*, 129, 227–235.

Koval KJ, Egol KA, Cheung Y, Goodwin DW, Spratt KF (2007). Does a positive ankle stress test indicate the need for operative treatment after lateral malleolus fracture? A preliminary report. *J Orthop Trauma*, 21, 449–455.

Lloyd J, Elsayed S, Hariharan K, Tanaka H (2006). Revisiting the concept of talar shift in ankle fractures. *Foot Ankle Int*, 27, 793–796.

Ramsey PL, Hamilton W (1976). Changes in tibiotalar area of contact caused by lateral talar shift. *J Bone Joint Surg Am*, 58, 356–357.

Viva 3 Questions

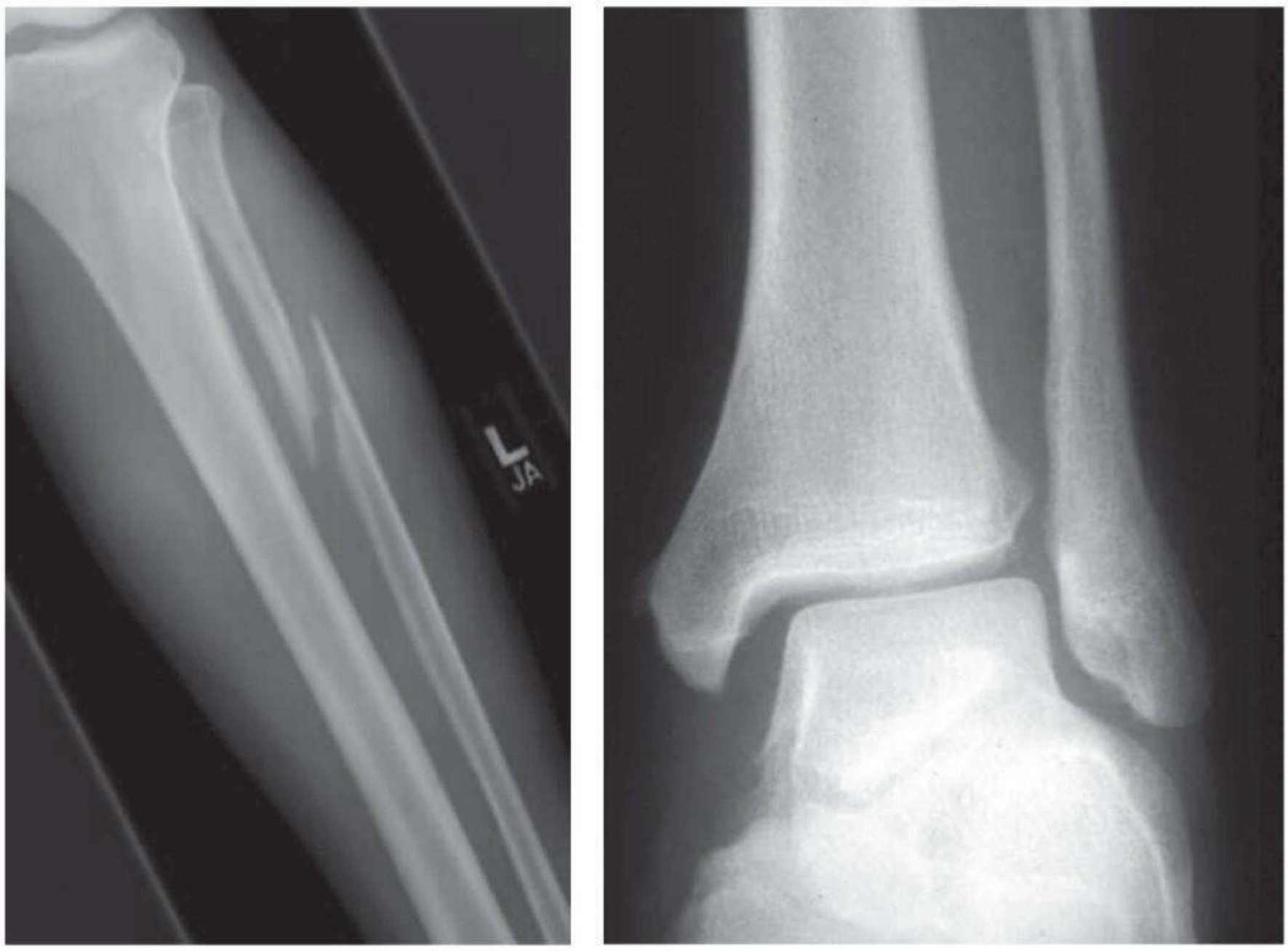


Figure 24.3 AP radiographs of lower limb

A 28-year-old female aerobics instructor twisted her ankle in a nightclub, sustaining an isolated injury to the right lower limb. What do her radiographs show?

How would you classify this injury?

How would you treat this injury initially?

What is your preferred method for syndesmosis fixation and why?

Viva 3 Answers

A 28-year-old female aerobics instructor twisted her ankle in a nightclub, sustaining an isolated injury to the right lower limb. What do her radiographs show?

These anteroposterior views of the left lower limb demonstrate a proximal third spiral fibula fracture, and an ankle mortise with widening of the medial and superior clear spaces leading to significant talar shift.

How would you classify this injury?

This injury is a Weber type C fibular injury with associated deltoid and syndesmotic ligament ruptures. It is also called a 'Maisonneuve' fracture as per the original description. It can also be classified according to the Lauge-Hansen system as a PER injury, which implies an external rotational force applied to a pronated foot. This leads to a medial injury first—in this instance the disruption of the deltoid ligament—followed by anterior syndesmosis, interosseous membrane to the level of a fracture of the fibula, or to the disruption of the proximal tibiofibular joint followed by the posterior syndesmosis in type IV injury. This fracture would represent a type III or IV PER injury.

How would you treat this injury initially?

The injury pattern is unstable, and therefore in all but extremely unfit patients surgical fixation would be my preferred management. Initial treatment would include analgesia and splintage, a thorough history, examination and documentation of neurological and vascular status, and pre-operative investigations such as blood tests and ECGs as needed.

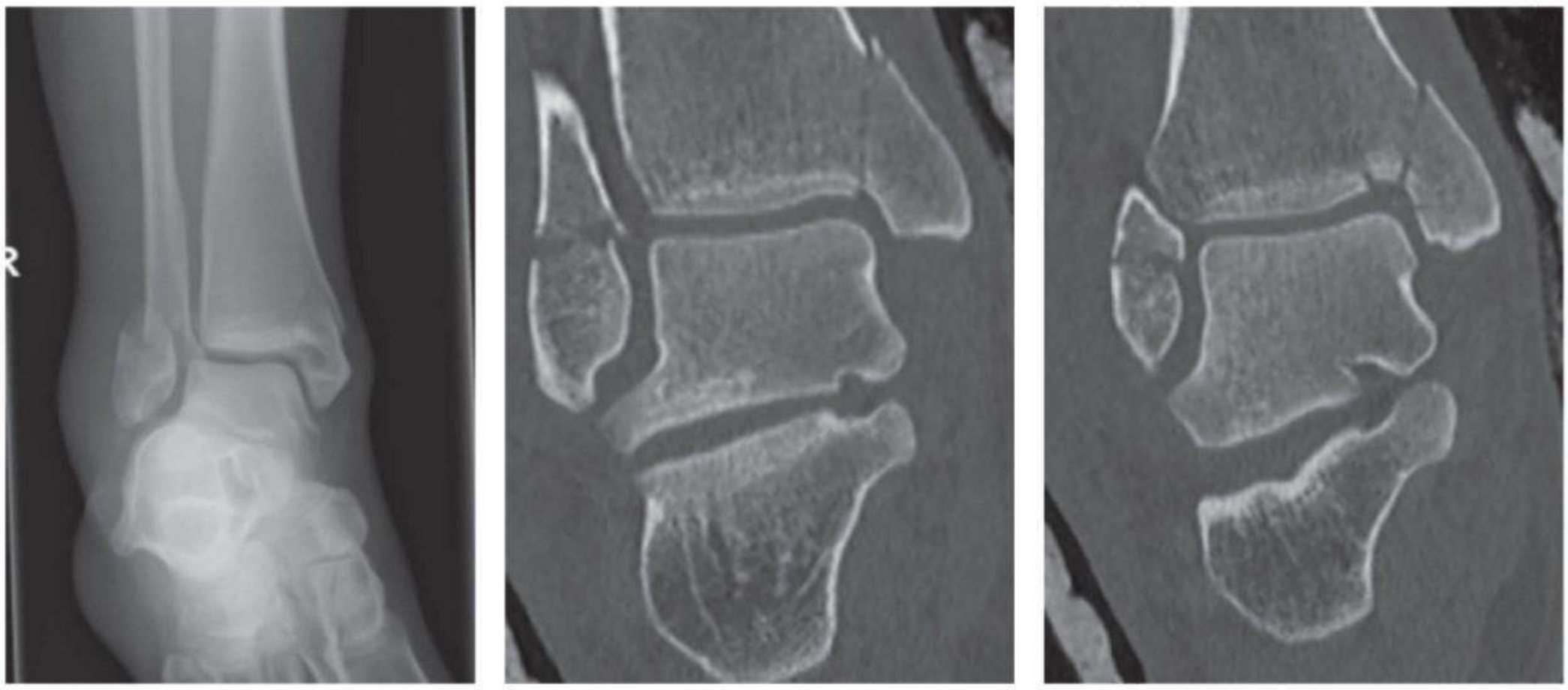
This fracture configuration typically requires stabilization of the syndesmosis. In this instance, under tourniquet and image control, and with a sandbag under the ipsilateral buttock, I would attempt closed reduction of the syndesmosis using a large pelvic reduction clamp between the malleoli. Although the position of the foot was once thought to be important it has since been shown that there is no need to dorsiflex the foot with this manoeuvre. However, I would place a bolster, such as a kidney dish, under the Achilles tendon to avoid resting the heel on the bed and driving the talus forward. As the fibula fracture is very proximal it is not suitable for plate fixation which can make it difficult to achieve correct length and rotation of the fibula. In order to be sure of an accurate reduction I would check the level of the fibular tubercle on the mortise view to ensure the fibular length and rotation were accurate.

If the syndesmosis did not reduce it would require further open reduction to clear any fragments that could block the reduction. There is no need to repair the deltoid in most cases provided the fibula and syndesmosis have been accurately reduced. However, to allow for adequate deltoid healing the patient should be put in a cast for 6–8 weeks.

What is your preferred method for syndesmosis fixation and why?

Controversy exists around the choice of implant and technique for syndesmotic stabilization in the ankle. While there are many options, I prefer to use two small-fragment 3.5 cortical screws, over four cortices. I prefer the small-fragment screws as the heads are less prominent than the large-fragment screws, and they have been shown to be sufficiently strong. I prefer four cortices—although there may be a slightly increased incidence of screw breakage, they are easier to remove from the medial side if this is the case. I am aware that tightrope fixations are an option, but have no experience of these. After fixation, I splint the limb but allow early range of motion from about

Viva 4 Questions



(a) (b) (c)

Figure 24.4 Right ankle (a) radiograph; (b), (c) coronal CT

You are called to the review a young woman who has slipped down a few steps. This is an isolated injury in a healthy young adult. Please describe what the imaging shows.

What is the likely mechanism and what are you definitive options?

What are your principles of fixation and how would you go about it?

What is the prognosis for ankle fractures?

Viva 4 Answers

You are called to the review a young woman who has slipped down a few steps. This is an isolated injury in a healthy young adult. Please describe what the imaging shows.

The images are an anteroposterior radiograph and coronal CT slices of a skeletally mature right ankle. The radiograph shows a low fibula fracture and a medial malleolar fracture. The CT scans confirm this and show the orientation of the fibula fracture to be predominantly transverse and the medial side injury to be vertical—a shearing fracture. There is also a small segment of depressed articular surface in the medial corner of the tibial plafond.

What is the likely mechanism and what are you definitive options?

The mechanism here is supination–adduction force. In this mechanism the injury starts on the lateral side with failure in tension of the fibula or lateral ligament complex—hence the low transverse fibula fracture. As the talus then undergoes an adduction force this classically pushes off the medial malleolus, resulting in a shear fracture line that is oblique or vertical in nature. Depression of the medial corner of the tibial plafond has occurred as the talus has continued to be adducted after the malleolus has sheared off. I prefer to use the Lauge-Hansen classification here as the Weber system would simply classify this as an 'A' fracture and underestimate the severity and instability of the injury.

What are your principles of fixation and how would you go about it?

The main principle is that this is an intra-articular fracture and it therefore needs anatomical reduction with absolute stability to allow early range of motion and restoration of function. The medial side represents a shear force with comminution of the medial malleolus. Therefore I would tend to favour a plate to withstand the shear forces after reduction of the intra-articular fragment. This is tricky and requires planning as the plate is applied through a direct medial approach which does not allow visualization of the joint or the depressed fragment. This fragment may be able to be reduced percutaneously through a mini anterior incision in the plane of the fracture line to allow in a narrow instrument to push the fragment down. The fibula requires an anti-glide plate posteriorly. I would utilize a posterolateral approach or a standard lateral approach, although this needs to be longer than the plate to allow for safe retraction of the skin edge and access.

What is the prognosis for ankle fractures?

The prognosis for ankle fractures is variable. The majority have good or excellent outcomes at 2 years, but with some limitations. Simple fracture patterns have superior outcomes. Removal of metalwork can improve lateral pain in 50% of cases, but is not routinely recommended.

Fifty per cent of bimalleolar ankle fractures will have good or better outcomes at 10 years, with 24% having a poor outcome with evidence of post-traumatic degenerative changes. Posterior malleolus fracture is associated with deteriorating outcomes, particularly if joint congruity is not restored.

Bhandari M, Sprague S, Hanson B, et al. (2004). Health-related quality of life following operative treatment of unstable ankle fractures: a prospective observational study. *J Orthop Trauma*, 18, 338–345.

Lash N, Horne G, Fielden J, Devane P (2002). Ankle fractures: functional and lifestyle outcomes at 2 years. *Aust NZ J Surg*, 72, 724–730.

Lauge-Hansen N (1954). Fractures of the ankle III. Genetic roentgenologic diagnosis of fractures of the ankle. *Am J Roentgenol Radium Ther Nucl Med*, 71, 456–471.

Viva 5 Questions

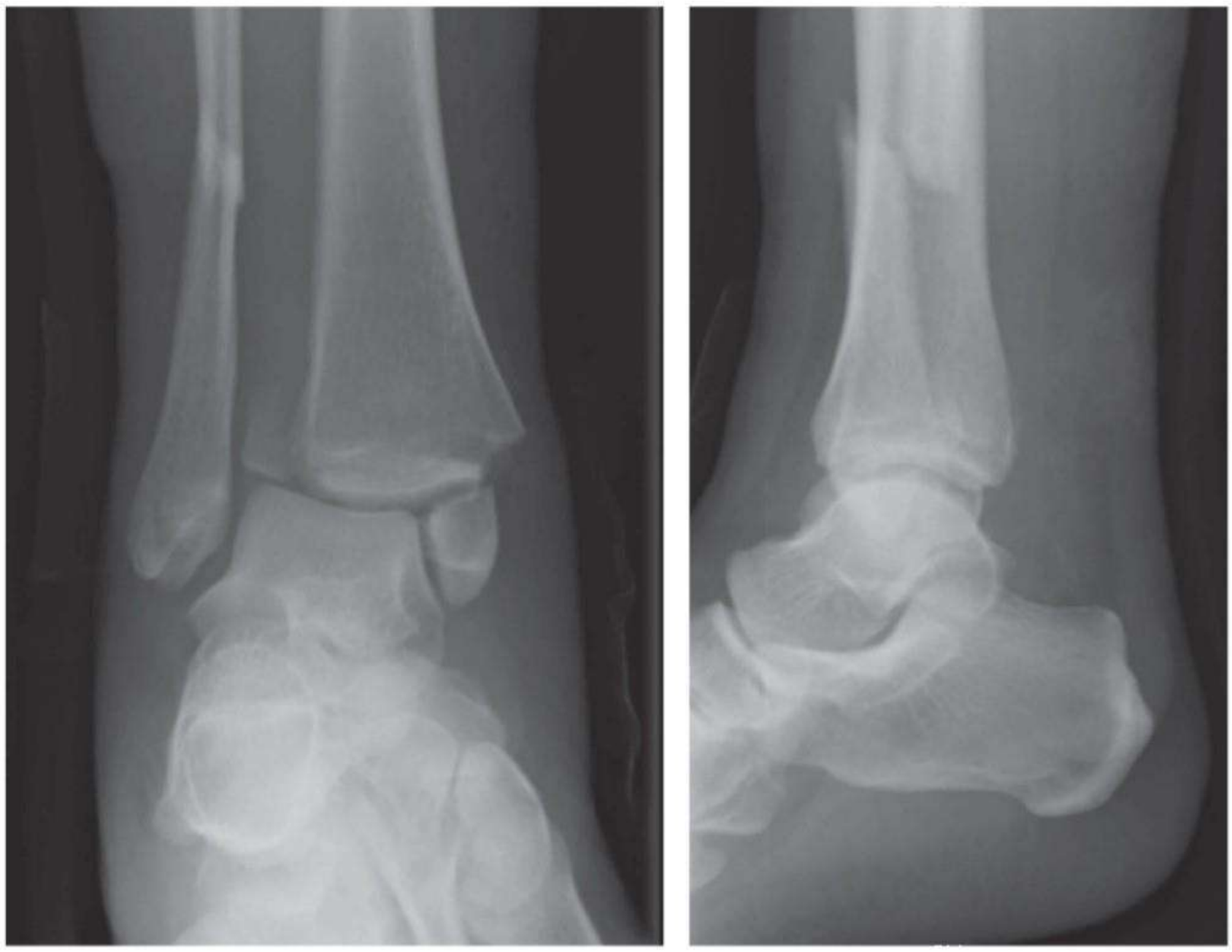


Figure 24.5 AP and Lateral ankle radiograph

What do these radiographs demonstrate?

How would you classify this injury?

What would you definitive management be?

What techniques can you use in comminuted fibula fractures to ensure superior outcomes?

What do these radiographs demonstrate?

These are anteroposterior and lateral views of a skeletally mature patient. They show a Weber type C transverse/short oblique fibula fracture, a 'pull-off' of the medial malleolus, and a small posterolateral fragment, with significant talar shift.

How would you classify this injury?

The simplest system by Weber would classify this as type C. Lauge-Hansen's more comprehensive system would classify this as a pronation–abduction injury. The foot is pronated at the time of injury (i.e. everted) with an abduction force applied to the talus. This leads to the characteristic pull-off injury on the medial side, with a bending pattern injury to the fibula above the syndesmosis. The syndesmosis is disrupted, in this instance with a pull-off of the tibial side of the PITFL. This injury is sometimes described as a Dupuytren's fracture dislocation.

What would you definitive management be?

Assuming no contraindications I would recommend ORIF. I would plan for fixation on the next available list as long as there is no significant soft tissue swelling; if there were I would delay until this subsided.

My preference is to fix the posterior malleolus, even if small, when there is such obvious instability in the ankle; however, in this instance, the fibula is fractured reasonably proximally so it may not be possible to fix this through a combined posterolateral approach. I would therefore fix the fibula through a lateral approach, being aware that the superficial peroneal nerve is likely to cross my incision. I would aim for anatomical reduction and rigid internal fixation of the fibula—a compression plate in this case, as it is a simple fracture pattern. This would require me to use a small-fragment locking compression plate because a third tubular plate would not be strong enough. After this I would certainly screen the ankle for external rotation instability and be prepared to use syndesmosis screws. I would prefer to put them through the plate, using two 3.5-mm screws over four cortices. The plate would need to be long enough to accommodate these. I would then address the medial side with ORIF. This looks to be a sizeable fragment and I should be able to get two small-fragment 40-mm partially threaded screws across the fracture. If the fragment was too small or comminuted I would consider a tension band wire technique or even just a stout 2-mm K-wire or two.

What techniques can you use in comminuted fibula fractures to ensure superior outcomes?

With the fibula the key is to accurately restore the length and the rotation; this becomes difficult in comminuted fractures. One technique is to stabilize the medial side first, which tensions the lateral ligaments helping to ensure the correct length and rotation. Fixation of the fibula with a lag screw and a one-third tubular plate is often impossible with comminution, therefore I would tend to use a thicker plate to withstand bending forces, either a reconstruction plate or a pre-contoured locking plate. However, one must use locking plates with caution because they are associated with an increased rate of wound complications. Radiographic parameters include the talocrural angle formed by two lines, one from the tip of the fibular to the tip of the medial malleolus and a second along the tibial plafond which is around 83°. Another feature is Shenton's line running from the lateral process on the talus to the tip of fibular; a disruption in this represents shortening and rotation malreduction.

Schepers T, Van Lieshout EM, De Vries MR, Van der Elst M (2011). Increased rates of wound complications with locking plates in distal fibular fractures. *Injury*, 42, 1125–1129.

Viva 6 Questions

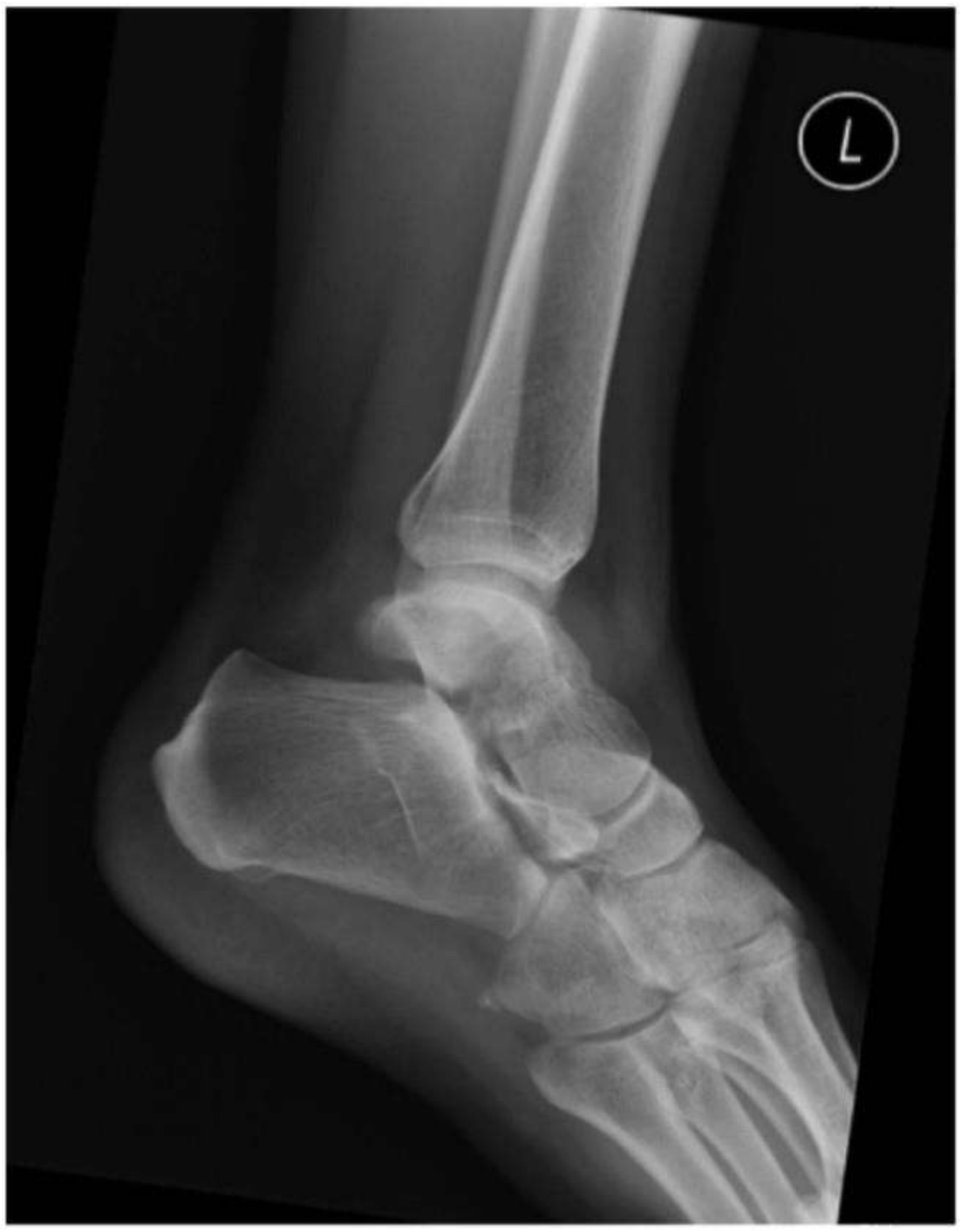


Figure 24.6 Lateral ankle radiograph

This patient fell from their horse. Can you describe the radiograph?

This patient has been managed according to ATLS principles. Their injury appears to be an isolated closed injury. How would you manage the patient in A&E?

How would you classify this injury?

How would you treat this patient definitively?

How would you manage this patient post-operatively?

How would you counsel the patient regarding their prognosis?

Viva 6 Answers

This patient suffered a high energy injury. Can you describe the radiograph?

This is a high-energy injury. The lateral view shows a significantly displaced fracture through the neck of the talus with an associated dislocation of the subtalar joint. I suspect there is a degree of comminution as I cannot delineate the fracture line very easily. There is also a fracture of the anterior process of the calcaneus—up to 89% of these types of injuries have an associated fracture elsewhere in the foot. I would like to see further views.

This patient has been managed according to ATLS principles. Their injury appears to be an isolated closed injury. How would you manage the patient in A&E?

I would take a thorough history—asking the patient about the mechanism of injury, significant medical history, drugs and allergies, smoking status, and employment. My examination would focus on a documented assessment of distal neurological and vascular status and an assessment of the soft tissues. I would splint the limb in a backslab and arrange for further imaging in the form of a CT scan to detail the anatomy of the fracture, especially any comminution, to allow for planning of surgery. While waiting for the scan I would elevate the limb and ask the nursing team to alert me should the patient's pain not be controlled by simple analgesia, because this patient is at risk of foot compartment syndrome. In the absence of neurovascular compromise there is no urgency in this situation to fix the fracture out of hours, and getting the CT would be vital. Recent studies have dismissed the fact that delay to ORIF is associated with an increased risk of AVN.

How would you classify this injury?

This is a type II fracture according to the Hawkins classification.

Hawkins classified these fractures in 1970, and gave specific focus to the risk of developing AVN. Type I fractures are non-displaced. They are rare and have a very low chance (<13%) of proceeding to AVN. The classification then looks at associated joint dislocations. Type II fractures have an associated subtalar dislocation, as in this case. The risk of AVN here is 20–50%. Type III have a dislocation of the entire body of the talus (subtalar and tibiotalar joint dislocation), while type IV also have a dislocation of the talar neck at the talonavicular articulation. Type III and IV injuries have reported AVN rates of up to 100%.

How would you treat the patient definitively?

All displaced articular fractures require anatomical reduction and rigid internal fixation. In this case, assuming the patient is adequately prepared for theatre and the soft tissue envelope is in good condition, I would offer ORIF as this allows for direct visualization of the reduction. Generally I perform this using a two-approach technique, with an anteromedial incision between the anterior and posterior tibialis tendons, avoiding any dissection of the deep deltoid which carries the blood supply to the body. My anterolateral approach is in line with the fourth ray, so the superficial peroneal nerve may need to be mobilized. I would elevate the belly of the extensor digitorum brevis and clear the fat pad from the sinus tarsi to expose the talus. It can be very difficult to reduce the dislocated talar body, and if required I would use an external fixator to provide distraction. Depending on the amount of comminution, I would be prepared to fix the talus with screws, plates, or both. The medial side is often comminuted and a plate may be best here to avoid late varus collapse.

Viva 7 Questions

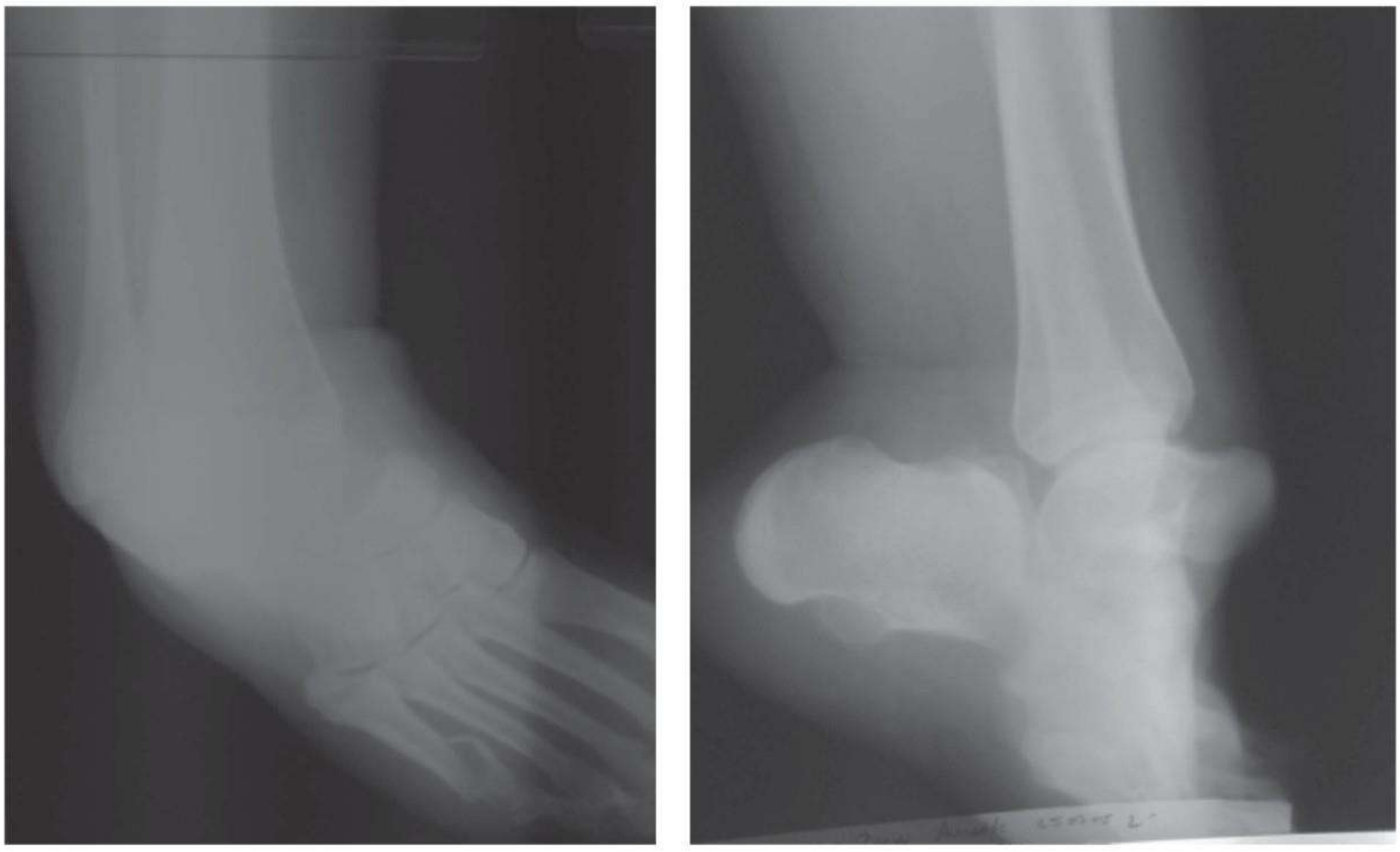


Figure 24.7 AP and lateral ankle radiographs

A young man who had been playing football is brought into the emergency department These are his radiographs. What can you see?

This is an isolated injury in a young healthy man with no relevant medical history. How would you manage him initially and what is this type of injury?

The emergency department has tried a closed reduction but failed. What are your concerns? How would you proceed?

Once the dislocation is reduced how would you manage the patient and what would his prognosis be?

Viva 7 Answers

A young man who had been playing football is brought into the emergency department These are his radiographs. What can you see?

This is an anteroposterior view centred on the left ankle. I can see a normal relationship between the talus and the tibia. The calcaneus is translated medially and posteriorly in relation to the talus, and the talonavicular articulation is also dislocated. This is a subtalar dislocation. For all intents and purposes, the whole foot is no longer articulating with the talus.

This is an isolated injury in a young healthy man with no relevant medical history. How would you manage him initially and what is this type of injury?

I would take a full history and examine the patient, concentrating in particular on whether this is an open injury and the neurovascular status. This needs urgent reduction, either under sedation or general anaesthesia. This is a medial subtalar dislocation, the commonest type (80%), followed by lateral dislocation (15%), with anterior or posterior dislocations occurring rarely. Medial dislocation is also described as acquired clubfoot and lateral dislocation as acquired flat foot. The calcaneonavicular ligaments remain intact, with the force rupturing the talonavicular ligaments and the talocalcaneal ligaments. Medial dislocations tend to be associated with low-energy injuries (e.g. basketball), whilst lateral dislocations tend to be higher-energy injuries. This needs urgent reduction to prevent tissue necrosis or neurovascular compromise.

The emergency department has tried a closed reduction but failed. What are your concerns? How would you proceed?

Assuming a good standard of technique was employed, my concern now would be that something was blocking reduction, such as an osteochondral fragment, an unrecognized fracture, or soft tissue interposition (extensor tendons or retinaculum, dorsalis pedis, deep peroneal nerve). I would arrange theatre urgently for closed reduction/open reduction. In the interim I would perform a CT scan to identify missed fractures or osteochondral lesions that could be blocking reduction. Closed reduction would entail relaxing the Achilles tendon (by flexing the knee), then exaggerating the deformity, and then reducing the deformity. If this failed I would open anteriorly, allowing exposure to both sides of the talar head to facilitate reduction. If the CT scan demonstrated associated fractures I would be prepared to supplement the reduction with fixation of these.

Once the dislocation is reduced how would you manage the patient and what would his prognosis be?

The management of these injuries remains controversial. In the absence of associated injuries (e.g. open fractures, neurological injury), the joint is usually stable post-reduction. I would allow a short period of immobilization so soft tissues can recover before commencing range-of-motion exercises to prevent stiffness, as that is the main problem post-injury. With more complex injuries the rehabilitation would be delayed. In the long term up to 80% of patients with these injuries will have stiffness in the subtalar joint and 50% will have evidence of degenerative changes; however, osteonecrosis is seen much less often. The majority of patients will still have good function in the long term. High-energy injuries are associated with increased stiffness, degenerative changes, and a worse outcome.

Bibbo C, Anderson RB, Davis WH (2003). Injury characteristics and the clinical outcome of subtalar dislocations: a clinical and radiographic analysis of 25 cases. *Foot Ankle Int*, 24, 158–163.

Viva 8 Questions

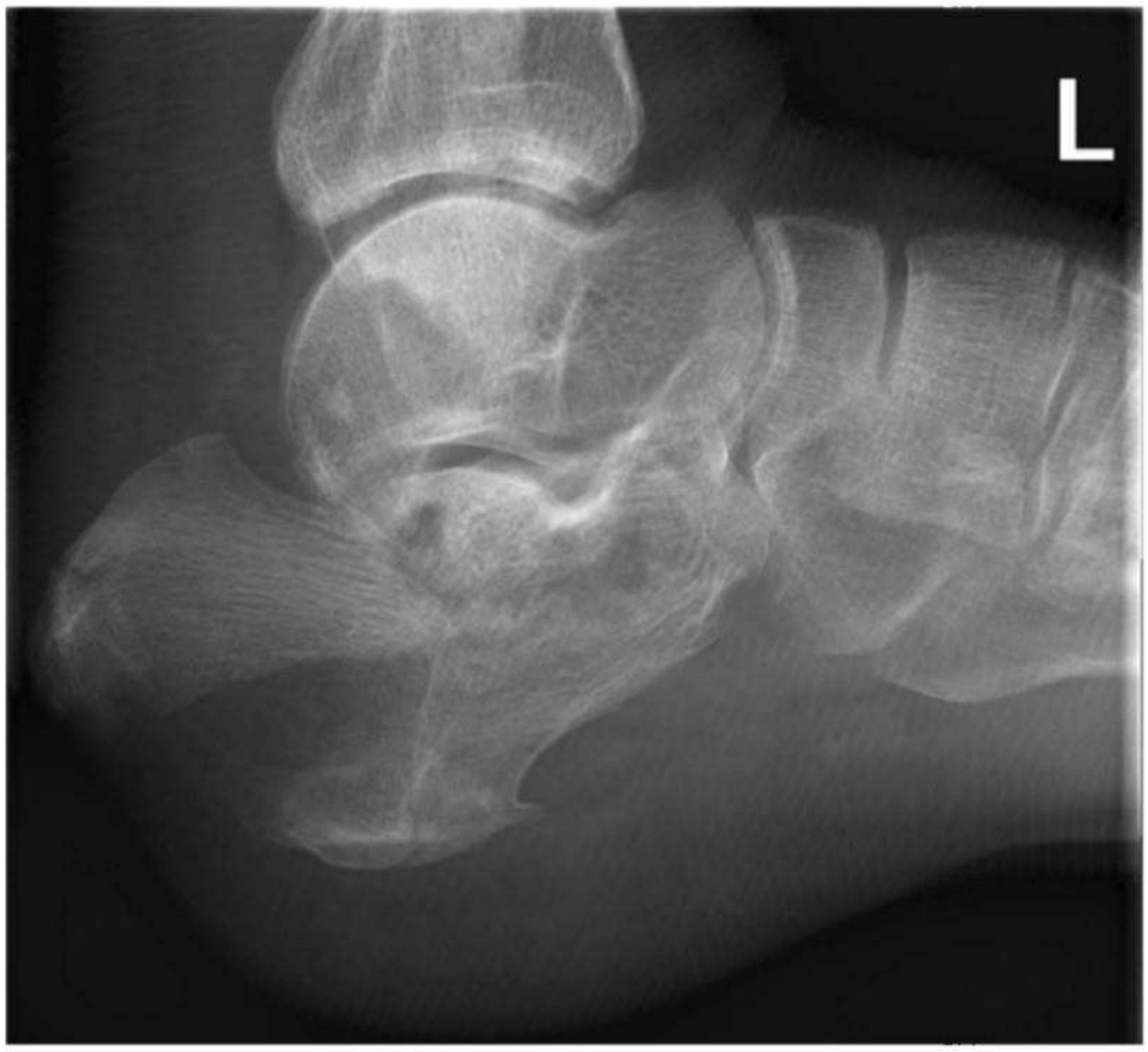


Figure 24.8 Lateral ankle radiograph

What does this radiograph show?

Can you classify this fracture?

What are the principles of management for this type of injury?

What are the short- and long-term consequences of calcaneal fractures?

Are you aware of any literature showing a difference between non-operative and operative management of calcaneal fractures?

What does this radiograph show?

This is a lateral view of the ankle and hindfoot of a skeletally mature patient with a significantly displaced calcaneal fracture. The majority of the posterior tuberosity is split and displaced and I suspect the fracture extends into the subtalar joint.

Can you classify this fracture?

This is a severe tongue-type fracture (also known as a beak fracture).
Calcaneal fractures can be broadly classified as extra-articular or intra-articular. Extra-articular fractures (25%) include tuberosity fractures, anterior process sustentacular fractures, and fractures of the body not involving the joint. Intra-articular fractures (75%) can be classified by the Essex–Lopresti system. This distinguishes those intra-articular fractures that do not involve the subtalar joint from those that do. Fractures involving the subtalar joint can broadly be classified as tongue type or joint depression type according to whether the fracture line on the lateral radiograph exits posterior (tongue) or anterior (joint depression) to the proximal insertion of the Achilles tendon at the posterior tuberosity. A further classification by Sanders, based on coronal CT slices through the subtalar joint, allows for operative planning and prognosis of intra-articular fractures involving this joint.

What are the principles of management for this type of injury?

Some calcaneal fractures can be treated non-operatively, but almost all tongue-type fractures necessitate operative intervention due to their threat to the skin on the posterior heel. In my department these injuries may get referred on to the specialist trauma centre, but the principles of management in this case revolve about two issues: soft tissue management and fracture management. The soft tissues posteriorly may be in peril. I would assess the tissues for signs of breakdown or necrosis, and if there is any concern I would ask a plastic surgeon to review the skin at the time of the index operation. Tongue-type fractures often need reducing to allow the soft tissues to settle, unlike joint depression types, which can afford to wait. If skin coverage was not achievable after fixation I would use a vacuum-assisted closure device before getting the plastic surgery team to provide definitive cover. After a CT scan to better visualize the fracture lines and comminution, my operative plan in this case would be to reduce the fracture under image control with a large clamp and stabilize the fracture with two screws from posterosuperior to anteroinferior through a minimal-access incision adjacent to the Achilles tendon on the lateral side. The reduction of the tongue fragment should reduce the majority of the subtalar joint; however, if I am unsure of the reduction I could assess it through an open visualization via a sinus tarsi approach. I am aware that the tension band principle has been described but it has high complication and metalwork removal rates.

What are the short- and long-term consequences of calcaneal fractures?

Calcaneal fractures are often the result of axial compression. There is an association with tibial pilon and plateau fractures, as well as spinal injuries. These can be high-energy injuries, so immediate issues include haemodynamic compromise from other fractures or visceral injury.

Early complications include swelling—one often needs to wait 10 days or more before fixation in joint-depression-type fractures. Neurovascular compromise is rare. Early post-operative wound breakdown is an early complication seen in up to 10% of patients who have an extended lateral incision. Meticulous tissue handling should reduce this. Later issues include subtalar arthrosis, either from initial comminution or malreduction. The need for potential fusion should be discussed early

Viva 9 Questions

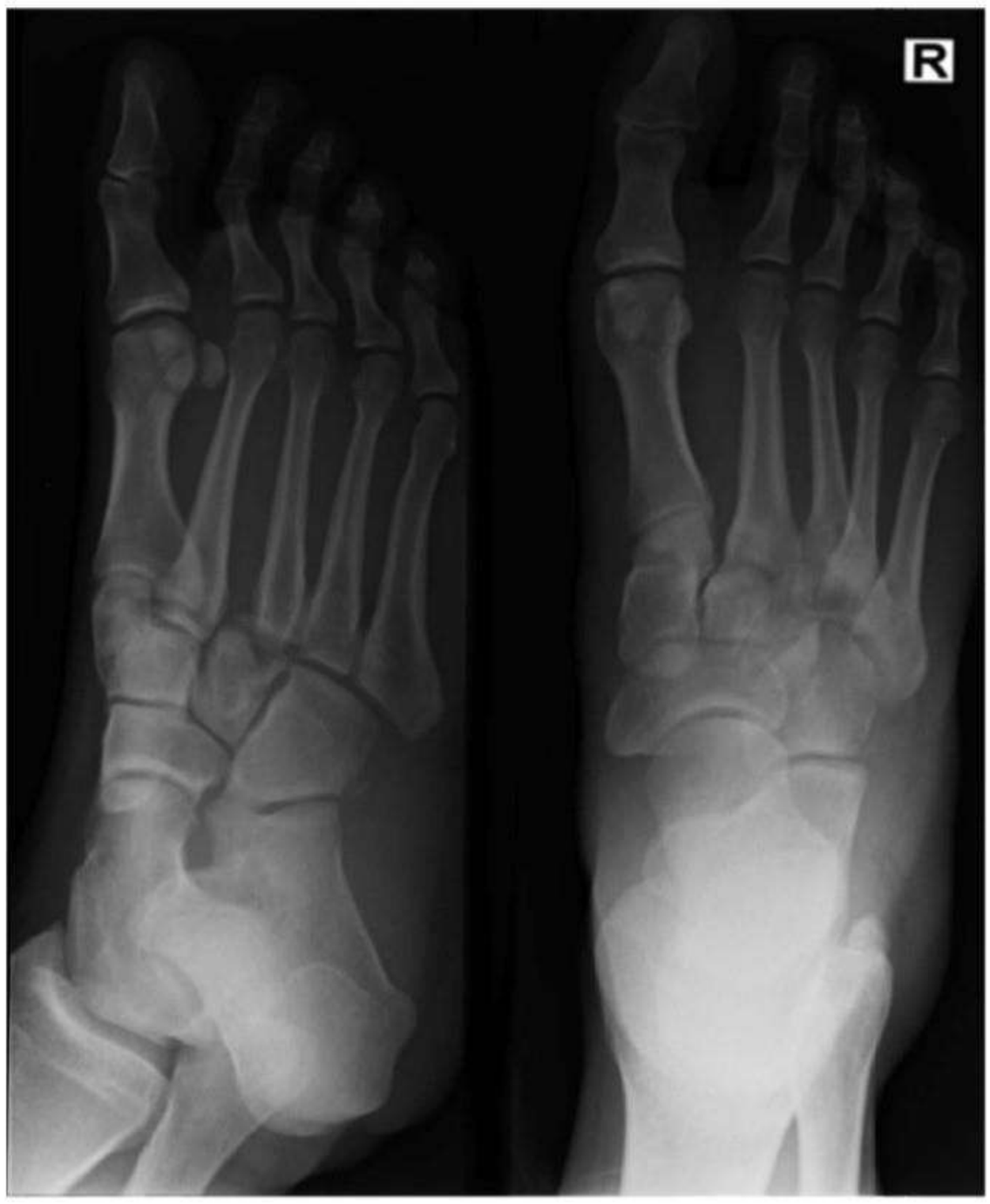


Figure 24.9 AP and oblique foot radiographs

What does this radiograph show?

What is the usual mechanism of injury?

What are the clinical and radiographic features of this injury?

Do you know any classifications for this type of injury?

Can this injury happen without a fracture, i.e. ligamentous injury?

How would you treat this injury?

What are the short- and long-term complications?

Viva 9 Answers

What does this radiograph show?

These anteroposterior and oblique views of the right foot show a Lisfranc fracture dislocation This predominantly involves the second and third tarsometatarsal (TMT) joints, although the fourth joint appears to have mild incongruity. On the anteroposterior view the second metatarsal is laterally subluxed and its medial border does not line up with the medial border of the middle cuneiform. On the oblique view the third metatarsal is not in line with the lateral cuneiform. There is also a flake of bone at the calcaneocuboid joint, although this may be old. I would like to see a lateral view to assess for any dorsal subluxation of the metatarsals.

What is the usual mechanism of injury?

Lisfranc was a French surgeon who originally described this injury in Napoleonic soldiers who had fallen from a horse with a foot trapped in the stirrup. Two distinct mechanisms of injury are now described. (1) A direct injury in which the foot is often crushed, leading to a midfoot 'break' or loss of arch, with a disruption of the tarsometatarsal joints. (2) An indirect injury, in which the foot is subject to a torsional force, such as being retained in the stirrup as the rider falls from a horse, or an axial and rotational load to a plantarflexed foot.

What are the clinical and radiographic features of this injury?

Any patient with suspected 'midfoot sprain' should be carefully assessed for Lisfranc injury. The mechanism of injury should be elicited by thorough history-taking, which often leads to the diagnosis. Swelling of the midfoot disproportionate to the mechanism of injury, plantar ecchymosis, and pain should raise the suspicion. Radiological signs include the 'fleck sign' at the TMT joint, dorsal subluxation of metatarsal base in lateral view, misalignment of the second TMT joint in anteroposterior view, and sometimes misaligned third and fourth TMT joints in oblique view. A high index of clinical suspicion, weight-bearing radiographs of both feet, CT or MRI scan, and at times examination under anaesthetic may be needed to diagnose this injury, especially in subtle cases.

Do you know any classifications for this type of injury?

Quenu and Kuss's original classification, as modified first by Hardcastle and then by Myerson, is the most widely used. It consists of three patterns of injury. Type A (total incongruity, homolateral, or complete) injuries involve all five TMT articulations displacing dorsolaterally as a unit. Type B (partial incongruity, homolateral, or incomplete) injuries involve one or more, but not all, of the metatarsals. The pattern of injury usually involves medial displacement of the first metatarsal or dorsolateral displacement of one or more of the lateral metatarsals. Myerson's modification divided type B injuries into those affecting the medial articulation alone (type B1) and those involving one or more lateral metatarsals (type B2). Type C (divergent, total, or partial displacement) injuries occur when the lateral and medial metatarsals are displaced in opposite directions and in different planes. It is difficult to establish a more useful classification because significant variation exists in potential fracture patterns. Furthermore, the pattern of injury and degree of displacement are less significant for poor outcome than the quality of the reduction or the extent of soft tissue injury to the foot. Therefore the simple distinction between direct and indirect injuries is more useful for prognosis.

Viva 10 Questions

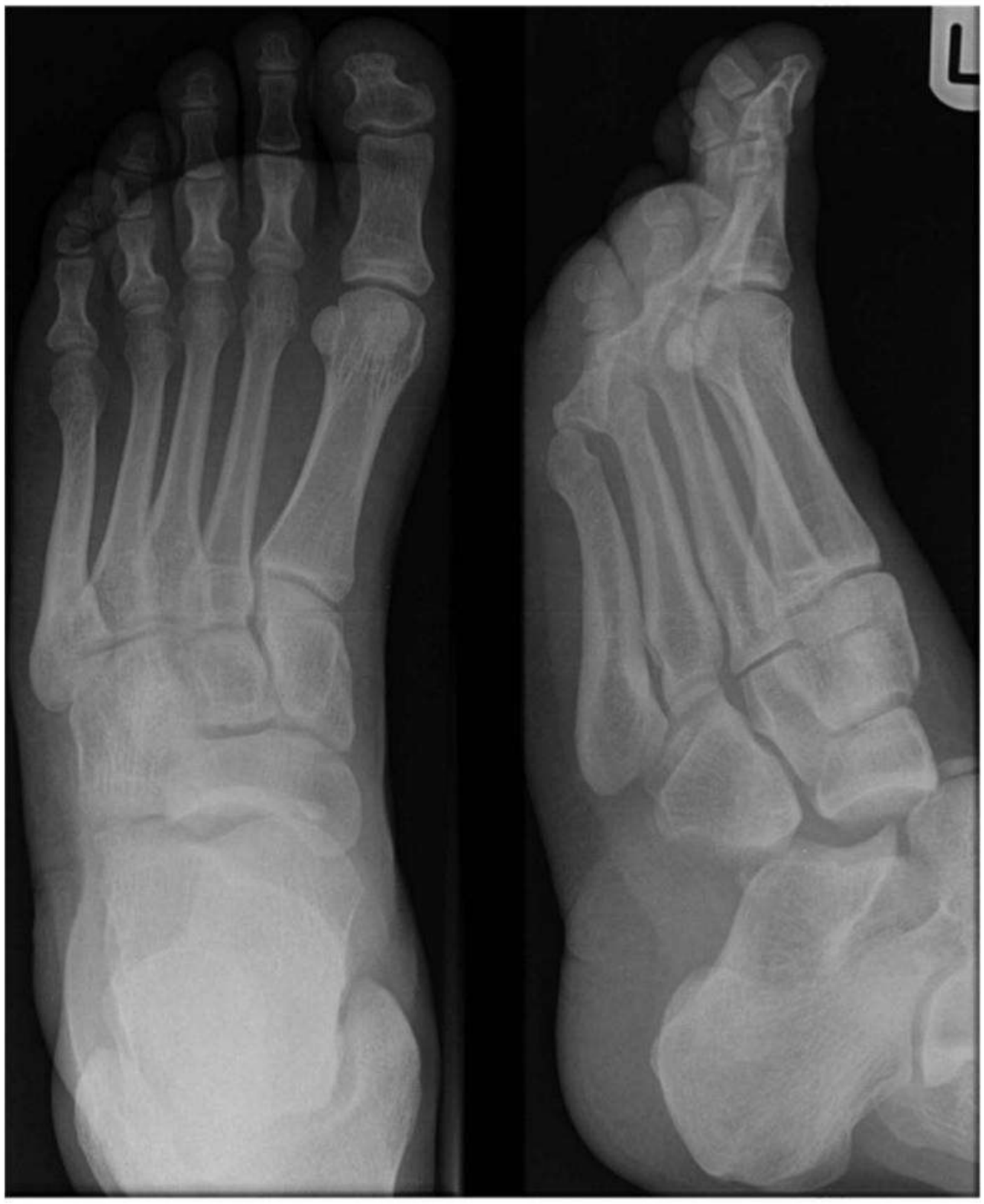


Figure 24.10 AP and oblique foot radiographs

What do these radiographs show?

How would you manage the patient?

This is an isolated injury—the foot was run over. How would you proceed?

During admission, but prior to fixation, you are informed that this patient's pain is resistant to opioids and increasing in severity. How would you proceed?

How will you manage this?

How do you decompress compartments in the foot?

What do these radiographs show?

These are anteroposterior and oblique views of a skeletally mature left foot showing a dislocation through the Chopart joint. The talonavicular and calcaneocuboid joints are dislocated, with plantar displacement of the distal foot. There is a small fragment of bone at the level of the talonavicular joint. I cannot see any other fractures but would like further imaging to confirm this.

How would you manage the patient?

This is clearly a high-energy trauma. I would therefore manage this patient following ATLS protocols, which deal with life-threatening injuries in a logical hierarchal sequence. For the foot, a thorough and documented neurological and vascular examination, with an assessment of the soft tissues, is mandatory. Analgesia and splintage are required. I would plan to reduce the foot in theatre under anaesthetic on the first available trauma list. I would only do this overnight if I was concerned about the viability of the foot or if there was obvious vascular compromise.

This is an isolated injury—the foot was run over. How would you proceed?

I would still take a full history and examination of the patient. Concentrating on the foot I would assess the degree of soft tissue injury, swelling, and neurovascular status. The patient would need to be admitted for definitive management following a CT scan to delineate fracture patterns and to plan surgical reduction and fixation.

During admission, but prior to fixation, you are informed that this patient's pain is resistant to opioids and increasing in severity. How would you proceed?

The concern here is that compartment syndrome of the foot may be developing. This requires immediate assessment and management. Pain that increases despite opioids and pain on passive stretch of the muscle compartments is characteristic, with evolving neurological and vascular compromise being late signs.

How will you manage this?

If the diagnosis was clinically confirmed I would proceed to surgical decompression. If there was doubt I would try and measure compartment pressure, with a value of greater then 30 mmHg being highly suggestive of compartment syndrome.

How do you decompress compartments in the foot?

Compartment syndrome of the foot has been described in the last 20–30 years but still generates some debate. Up to nine compartments have been described, but these may not all be functional because some have been demonstrated to communicate with other compartments at low pressure. The nine compartments are five in the forefoot (four interosseous and the adductor hallucis) and four in the hindfoot (medial, lateral, superficial central, and calcaneal compartments). Others have disagreed, stating there may be up to six compartments with only four compartments being functionally important. Current opinion in the UK is that the morbidity associated with decompressions (nerve injury, the need for skin grafts, infection) may be greater than the sequalae of an untreated compartment syndrome, and it may be best left alone, the only real indication for decompression being intractable pain.

Viva 11 Questions

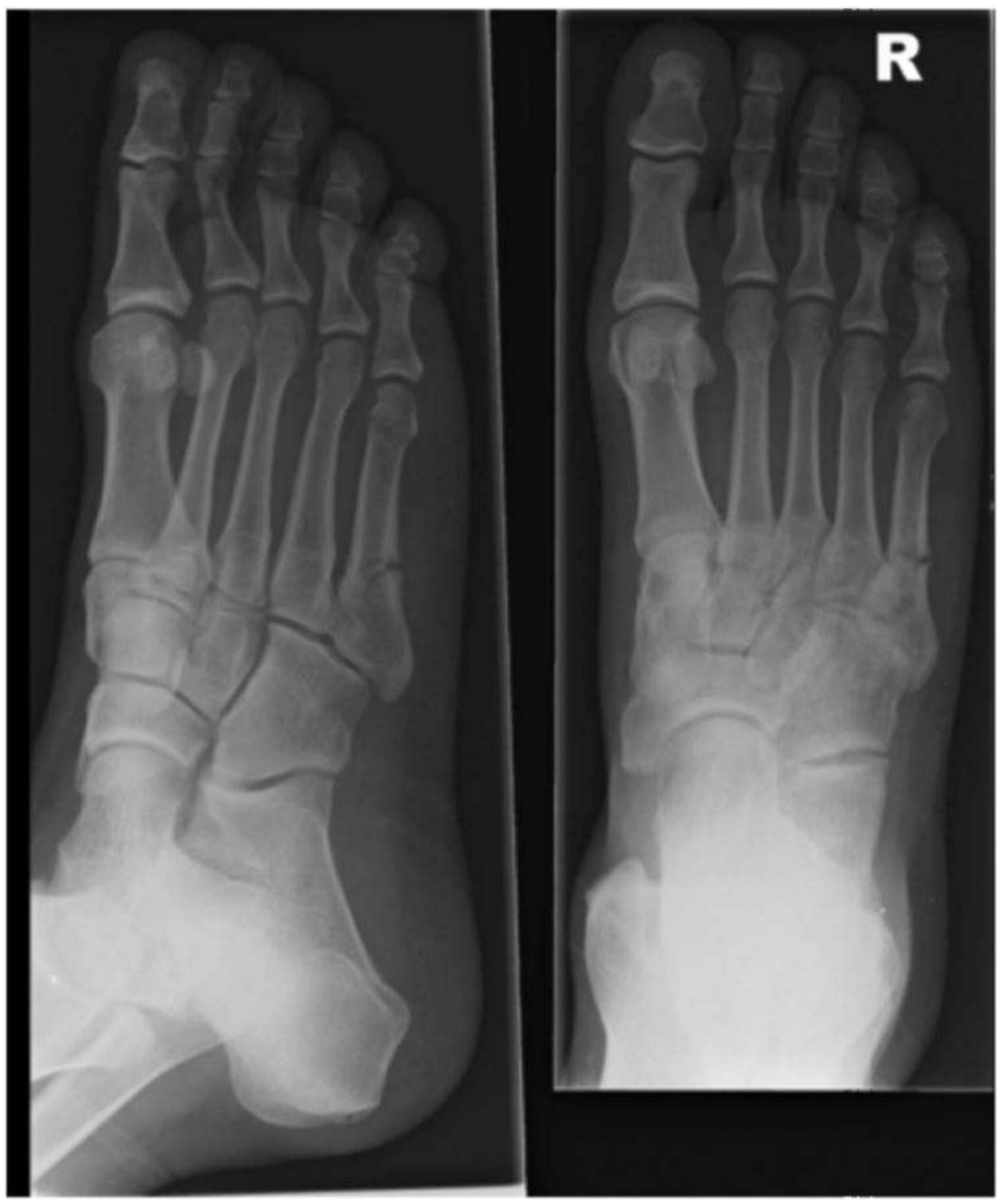


Figure 24.11 AP and oblique foot radiographs

Describe the injury shown in this radiograph.

Can you classify fifth metatarsal fractures?

What is the mechanism of injury?

What are the management options?

How would you treat a Jones fracture surgically?

What are the complications?

Viva 11 Answers

Describe the injury shown in this radiograph.

These are anteroposterior and oblique views of a skeletally mature right foot. There is a minimally displaced transverse fracture of the fifth metatarsal at the proximal diaphyseal–metaphyseal junction.

Can you classify fifth metatarsal fractures?

Injuries of fifth metatarsal can be grouped into neck, shaft, and proximal fractures. Fractures of the neck of the fifth metatarsal are uncommon and often associated with injuries of multiple metatarsals. Diaphyseal fractures can be oblique/spiral with significant displacement. Fractures of the proximal fifth metatarsal are common and are classified by zones: tuberosity avulsion fracture, metaphyseal–diaphyseal junction (Jones fracture), and diaphyseal stress fractures.

What is the mechanism of injury?

It is not clear, but is thought to be either inversion with a fixed forefoot or adduction of the forefoot with rapid application of forces to the proximal metatarsal.

What are the management options?

I prefer to manage the majority of these fractures non-operatively. The options vary between a simple Tubigrip (compression) bandage, boot and plaster cast, or functional brace. Most heal in 3–4 weeks although diaphyseal fractures can take much longer. The proximal metaphyseal–diaphyseal (Jones) fracture is known for an increased risk of delayed/non-union, possibly due to poor blood supply. It is recommended that a non-weight-bearing plaster cast should be tried for 6 weeks first, unless in athletes or patients who wish to have it fixed surgically. This is often performed using a screw placed percutaneously. Some fractures of the fifth metatarsal occur in diaphysis, often as a result of repetitive stress in runners and athletes. These range from an undisplaced fracture to established fracture with sclerosis at the fracture site and in the cortex. The treatment often is surgical with intramedullary screw fixation with or without bone grafting as there is a high rate of non-union in this group.

How would you treat a Jones fracture surgically?

I would like to discuss this with a foot and ankle specialist in our department, although I am confident of dealing with these fractures. The general preference is to fix these fractures using a partially threaded screw, either cannulated or simple, under radiological control. It is important to use a tap to reduce the risk of fracture of the shaft. The screw should be long enough to pass the fracture line with good purchase in the bone—a 4.5-mm screw or larger is often required. The surgical risks are minimal, rehabilitation is enhanced, and healing is often achieved with internal fixation. Recovery takes about 4–6 weeks. There are reports of using tension band wiring and small plates with or without bone graft.

What are the complications?

The common risk is of delayed or non-union, but infection, intra-operative fracture, sural nerve injury, and complications related to metal work can also occur.

chapter 25

PAEDIATRIC TRAUMA

Viva 1 Questions

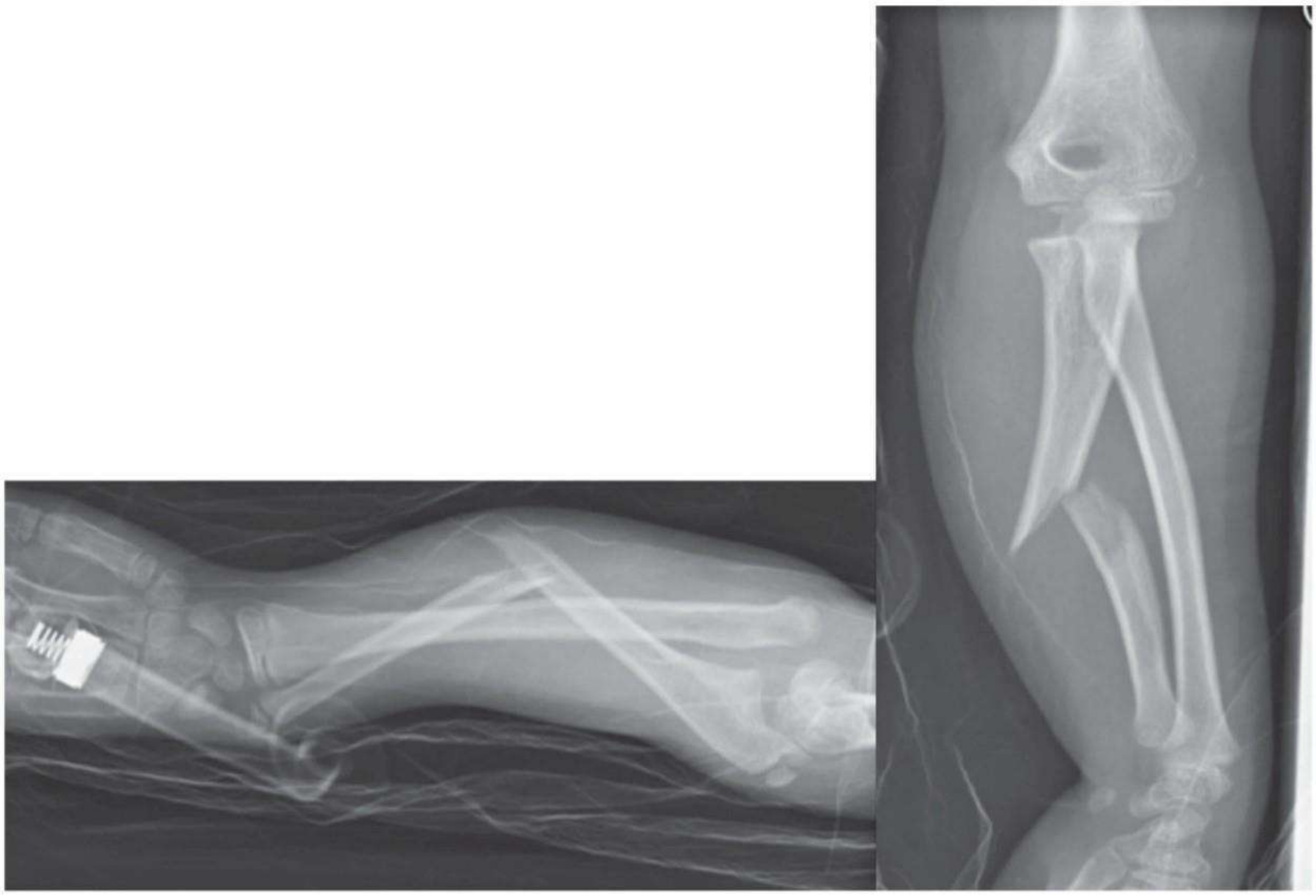


Figure 25.1 Forearm radiographs

An 8-year-old girl has fallen from a trampoline, injuring her forearm. There is a bleeding puncture wound dorsally and she is neurovascularly intact. Describe her radiographs.

How would you classify this injury?

How would you manage this patient?

What would you do if, following this, the radial head was not fully reduced?

How would you reduce the radial head?

How would you manage the patient post-operatively?

Viva 1 Answers

An 8-year-old girl has fallen from a trampoline, injuring her forearm. There is a bleeding puncture wound dorsally and she is neurovascularly intact. Describe her radiographs.

There is a dorsally and radially severely angulated midshaft ulna fracture with an associated anterior dislocation of the radiocapitellar joint. This is a Monteggia fracture dislocation.

How would you classify this injury?

An open fracture may be classified according to the Gustilo–Anderson system after debridement. The Monteggia fracture is classified by the Bado system. This is a Bado type I injury where there is a fracture of the ulna with an anterior dislocation of the radial head.

How would you manage this patient?

The goal of treatment is to achieve anatomical restoration of the length, alignment, and rotation of the ulna. On doing this the radial head invariably reduces. I would therefore debride and extend the skin wound to allow delivery of the fracture ends followed by meticulous debridement of any unviable tissue. I would reduce the fracture anatomically and, because this is a simple fracture pattern, aim for absolute stability which I would achieve by using a lag screw and 3.5 mm dynamic compression plate, with the screw being placed either through or separate from the plate.

What would you do if, following this, the radial head was not fully reduced?

I would first re-check my ulna fixation, as that is the most common reason why this might occur. If this was OK I would be worried about soft tissue interposition, most likely the annular ligament. I would radiograph the opposite elbow to be sure the patient did not have a congenital radial head dislocation (this can occur) before proceeding to open reduction of the radial head.

How would you reduce the radial head?

I would perform a Kocher's approach to the lateral elbow between the anconeus and extensor carpi ulnaris. The annular ligament is typically intact and flipped into the joint. It may be possible to flip it out but it may need to be divided then repaired after reduction has been achieved.

How would you manage the patient post-operatively?

I would discontinue antibiotics after a maximum of 72 hours and follow the patient up every week for at least the first 3 weeks to ensure there is no late subluxation of the radial head. I would use an above-elbow plaster for 6 weeks as this is a child and stiffness will not be a major issue. Contact sports are not allowed for at least 3 months.

Edwards SG, Weber JP, Baecher NB (2013). Proximal forearm fractures. *Orthop Clin North Am*, 44, 67–80.

Ring D (2013). Monteggia fractures. *Orthop Clin North Am*, 44, 59–66.

3.Spine

A- Oral

Cervical Myelopathy :

The patient will continue to deteriorate in a step-wise manner.

Myeoplpathy : **Stepwise deterioration**

common degenerative condition caused by compression on the spinal cord that is characterized by **clumsiness in hands** and **gait imbalance.**

Cervical Radiculopathy

clinical condition characterized by **unilateral** arm pain, numbness and tingling in a **dermatomal** distribution in the hand, and weakness in specific musle groups caused by cervical **nerve root compression**

Radiculopathy (Radiated pain) : A greater than 75% chance of complete resolution of symptoms

Myeoplpathy : **Stepwise deterioration**

Neurogenic shock

characterized by hypotension & relative bradycardia in patient with an acute spinal cord injury

potentially fatal

mechanism

circulatory collapse from loss of sympathetic tone

Spinal shock

defined as temporary loss of spinal cord function and reflex activity below the level of a spinal cord injury.
characterized by
Flaccid areflexic paralysis
bradycardia & hypotension (due to loss of sympathetic tone)
absent bulbocavernosus reflex

Scheuermann's Kyphosis

A rigid thoracic hyperkyphosis defined by > 45 degrees
caused by anterior wedging of >5 degrees across three consecutive vertebrae, narrowed disc spaces

- **Congenital Muscular Torticollis**

A congenital "packaging deformity" typically caused by contracture of the sternocleidomastoid (SCM) muscle

Klippel-Feil Syndrome

multiple abnormal segments of cervical spine characterized by Congenital fusion of 2 or more cervical vertebrae due to failure of normal segmentation or formation

Diastematomyelia

A fibrous, cartilagenous, or osseous bar creating a longitudinal cleft in the spinal cord

Syrinx & Syringomyelia

Syringomyelia a syrinx (fluid filled cavity) within the spinal cord that progressively expands and leads to neurologic deficits
Syringobulbia a syrinx within the brain stem that leads to neurologic symptoms

Spondylolisthesis : forward translation of one vertebral segment over the one beneath it

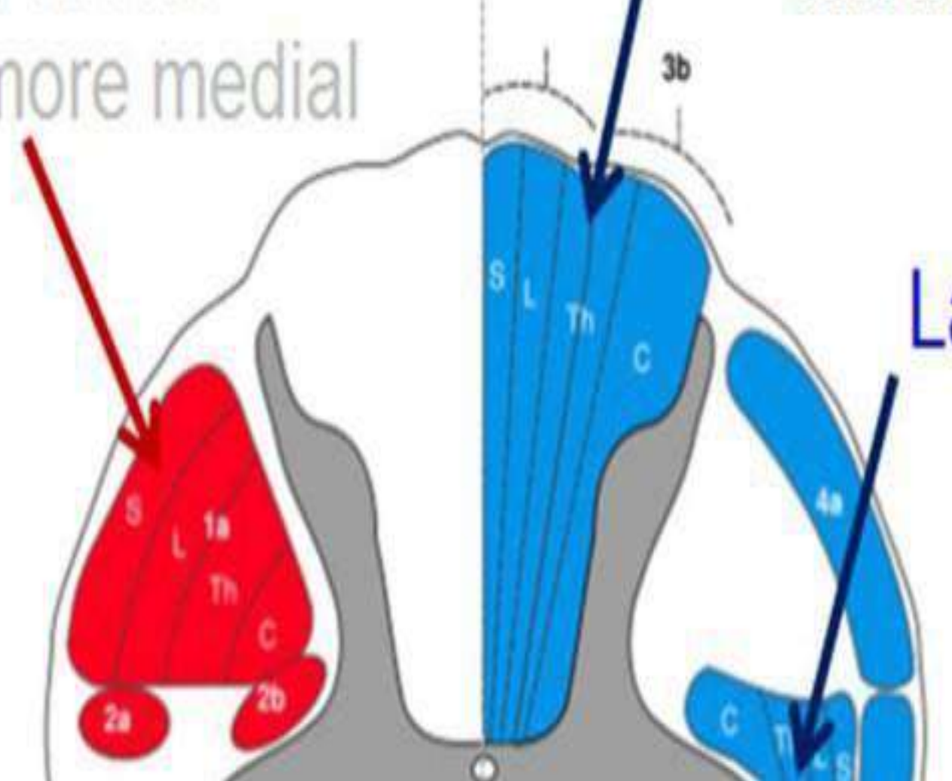
S**pondylolysis** : Defect (radiolucent gap) in pars interarticularis with adjacent bone sclerosis

Descending Tracts (Motor)

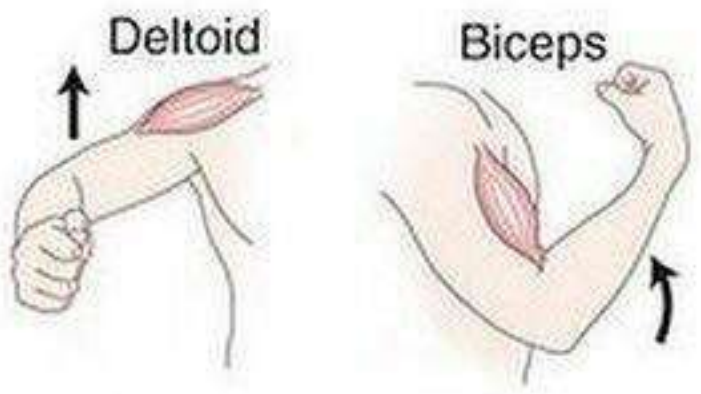
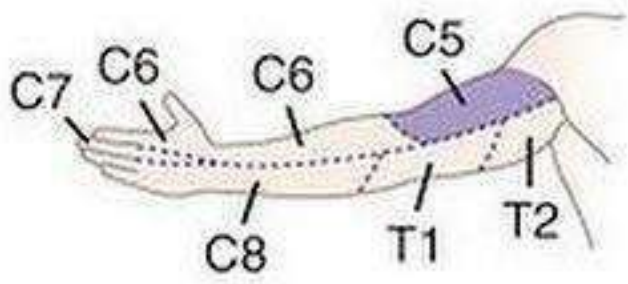
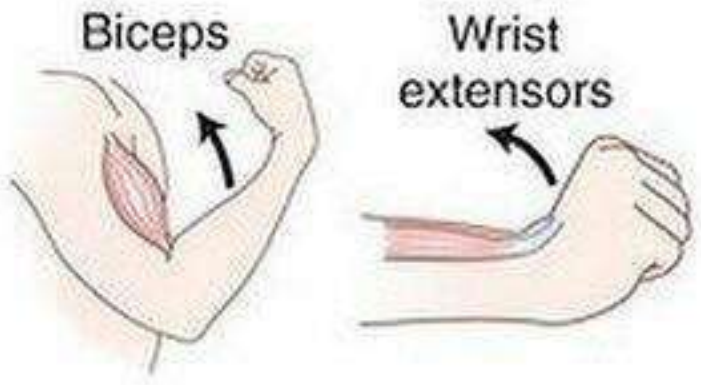
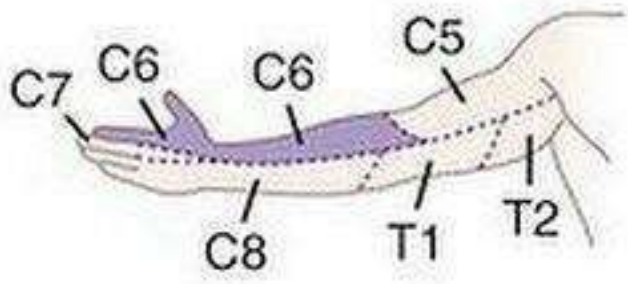
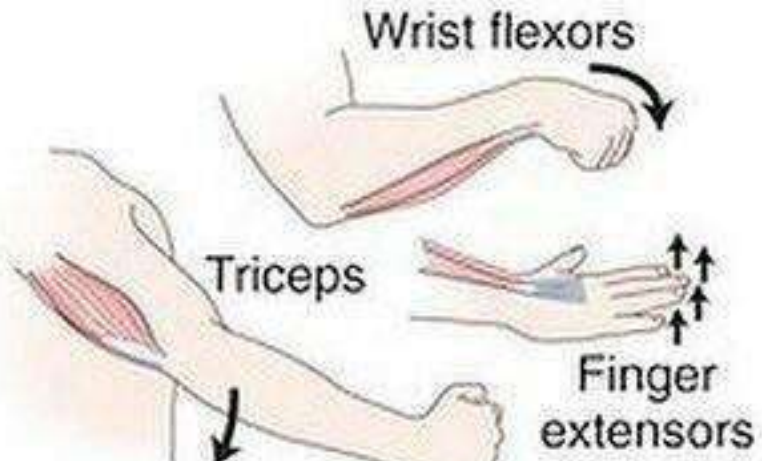
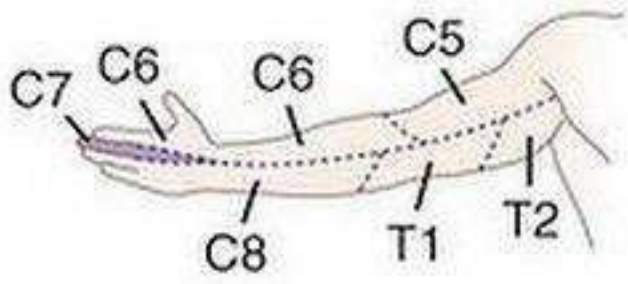
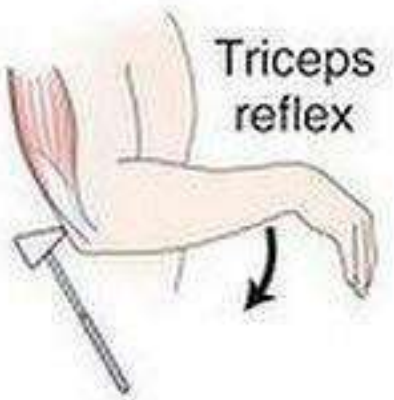
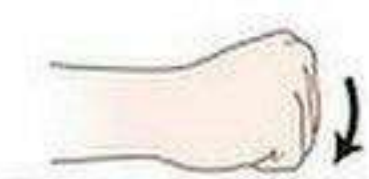
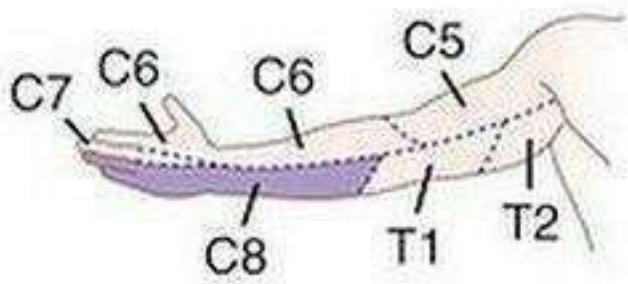
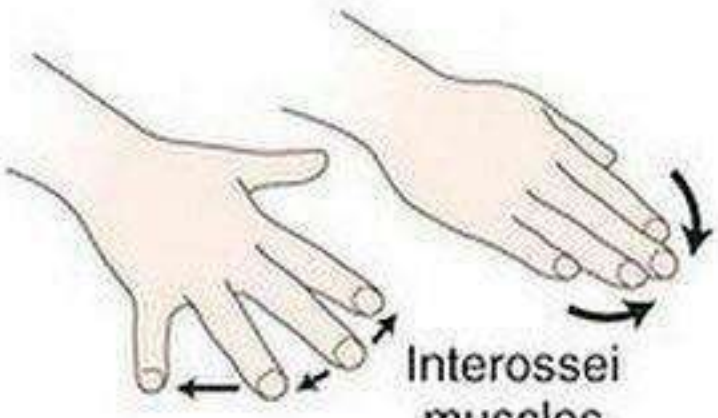
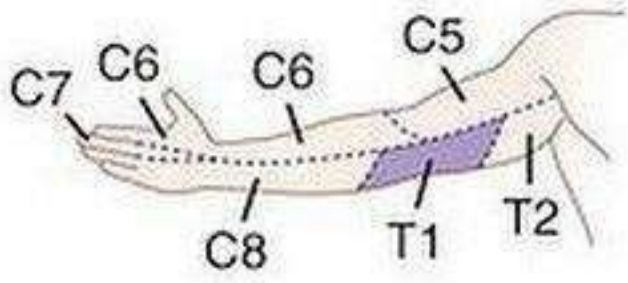
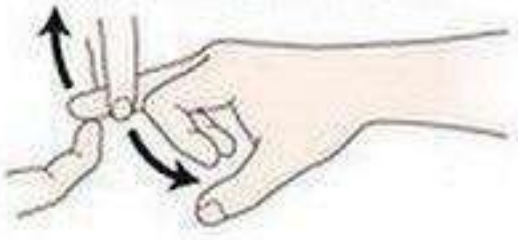
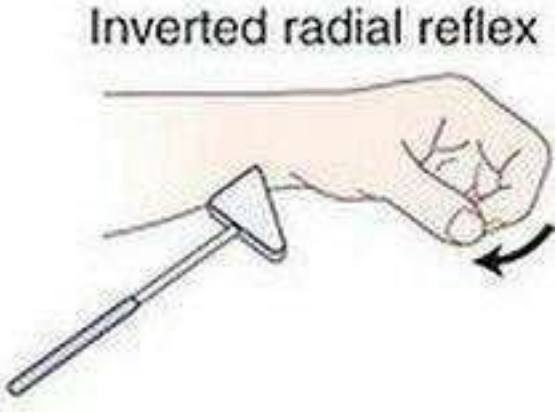
Lateral Corticospinal Tract
main voluntary motor
upper extremity motor
pathways are more medial
(central)

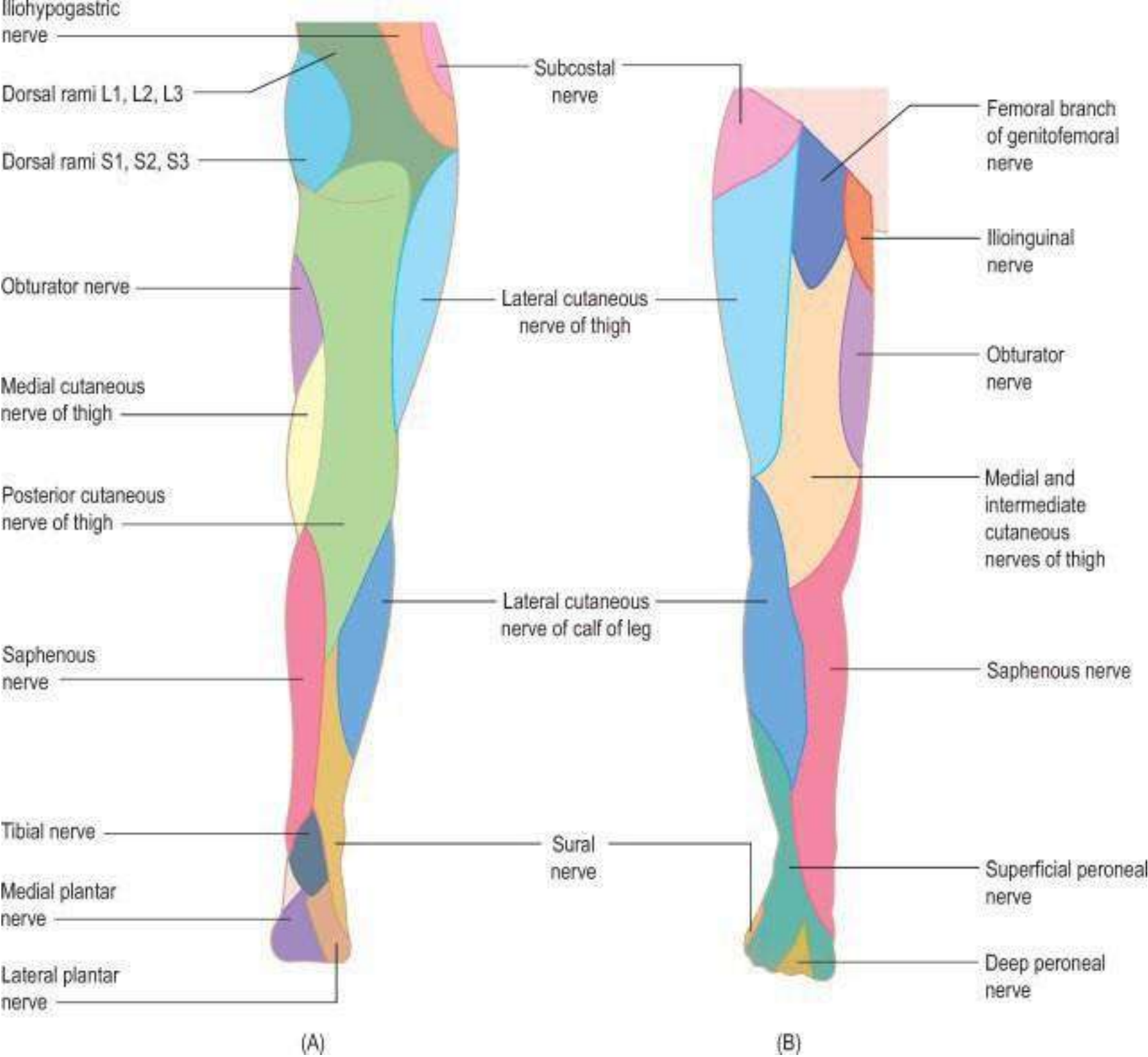
Ascending Tracts (Sensory)

Dorsal Columns (posterior funiculi)
deep touch, proprioception,
vibratory



Lateral spinothalamic tract
pain and temperature

	Motor evaluation	Sensory evaluation	Reflexes
C5			
C6			
C7			
C8			No reflex
T1			No reflex
Abnormal reflexes in cervical myelopathy			


Iliohypogastric nerve
Dorsal rami L1, L2, L3
Dorsal rami S1, S2, S3
Obturator nerve
Medial cutaneous nerve of thigh
Posterior cutaneous nerve of thigh
Saphenous nerve
Tibial nerve
Medial plantar nerve
Lateral plantar nerve
Subcostal nerve
Lateral cutaneous nerve of thigh
Lateral cutaneous nerve of calf of leg
Sural nerve
(A)
Femoral branch of genitofemoral nerve
Ilioinguinal nerve
Obturator nerve
Medial and intermediate cutaneous nerves of thigh
Saphenous nerve
Superficial peroneal nerve
Deep peroneal nerve
(B)

T1	Abduction and/or adduction of hand intrinscis	Flexor digitorum
	Myotomes of the Lower Limb	
L1/L2	Hip flexion	Psoas, iliacus, s
L3	Knee extension	Quadraceps, ad
L4	Ankle dorsiflexion	Tibialis anterior,
L5	Toe extension	Extensor hallicis tertius, popliteus
S1	Ankle plantar flexion and eversion, hip extension, knee flexion	Gastrocnemius, extensor digitoru
S2	Knee flexion	Biceps femoris,
S3	Rectal sphincter tone	Intrinsic foot mus

Deep Tendon Reflex	Spinal Level	Action / Normal Response
Biceps Tendon	C5-C6	Contraction of the biceps m
Brachioradialis Tendon	C5-C6	Elbow flexion and/or forear
Triceps Tendon	C7-C8	Elbow extension or contrac
Patellar Tendon	L3-L4	Knee extension

Causes of low back pain

Mechanical: muscle strains, degenerative disc diseases, disc prolapse, spinal stenosis, spondylolysis and other congenital disorders.
Inflammatory: RA, seronegative spondyloarthropathies.
Infectious: OM, epidural abscess, spondylodiskitis.
Neoplastic: mostly metastatic (Lung in 31%).
Metabolic: osteoporotic fractures, Paget's disease.
Psychosomatic or referred pain: ovarian cyst, chronic prostatitis, pelvic infections.

Risk factors:

- Heavy physical work: frequent bending, twisting, lifting, pushing and pulling, static postures and vibrations.
- Cigarette smoking.
- Obesity.
- Pregnancy.
- Psychosocial: job dissatisfaction, stress, anxiety, depression.

BACK PAIN RED FLAGS

AGE	<18 >50	Congenital anomaly Malignancy Infection AAA
IMMUNOCOMPROMISED	Chronic kidney disease Chronic liver disease	Infection (osteomyelitis, discitis, spinal epidural abscess)
INTRAVENOUS DRUG USE	Any IVDU	Infection (osteomyelitis, discitis, spinal epidural abscess)
HISTORY OF CANCER	Any cancer	Tumor Pathologic Fracture
SYSTEMIC SYMPTOMS	Fever/rigors Weight loss	Infection Malignancy
ANTICOAGULATION	warfarin, target specific anticoagulants	Epidural hematoma
TRAUMA	Major in young patients, minor trauma in elderly or those with rheumatologic disease	Fracture
SYMPTOMS OF CORD COMPRESSION	Saddle anesthesia Urinary or bowel incontinence or retention Perineal sensory loss Anal sphincter laxity	Compression via tumor, disc, absess etc
SEVERE or PROGRESSIVE NEUROLOGIC DEFICIT		Compression via tumor, disc, absess etc

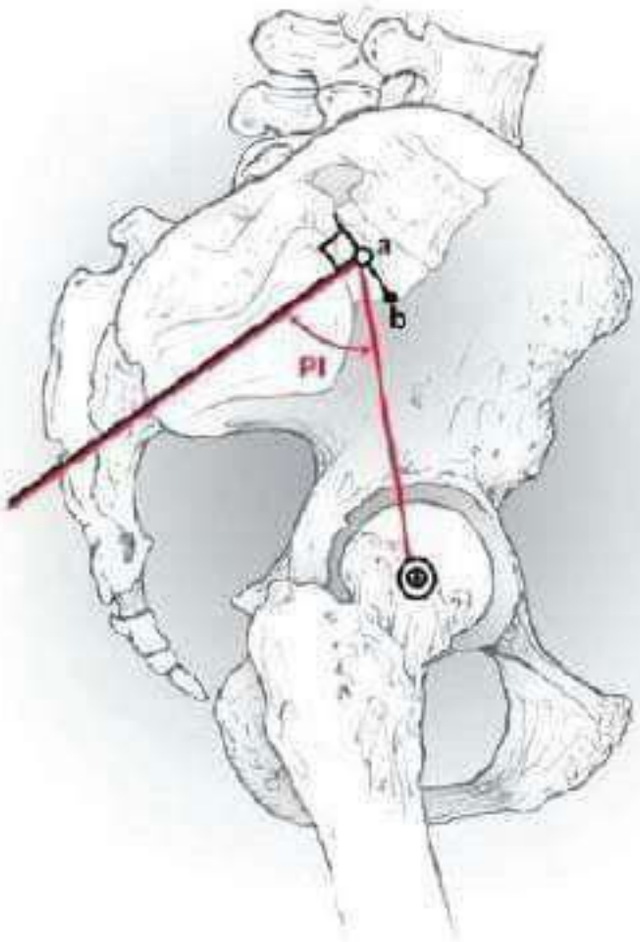
@FOAMpodcast

Referred pain

- pelvic infection
- ovarian cyst
- Dysmenorrhoea
- Cancer(ovary or uterus)
- Endometriosis
- Cronic prostatitis
- Pyelonephritis, hydronephrosis
- Ureter obstruction
- Renal stone

- Inflammatory bowel disorders
- Diverticulitis
- Colonic neoplasm
- Retro peritoneal hge
- Abdominal aortic
- Pancreatitis
- Cholecystitis

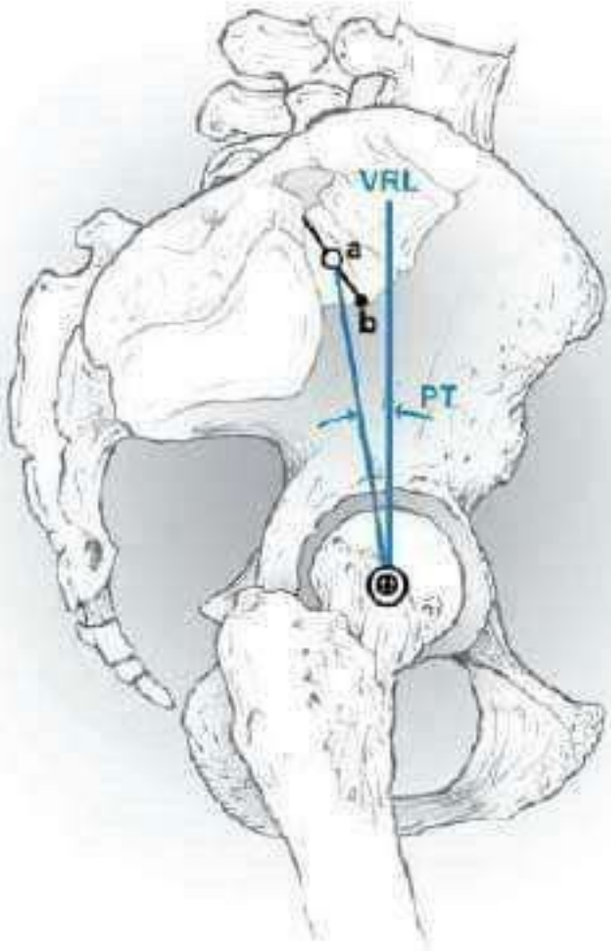
Assessment of Pelvic Parameters



Pelvic incidence is defined as the angle subtended by a line drawn between the center of the femoral head and the sacral endplate and a line drawn perpendicular to the center of the sacral endplate.

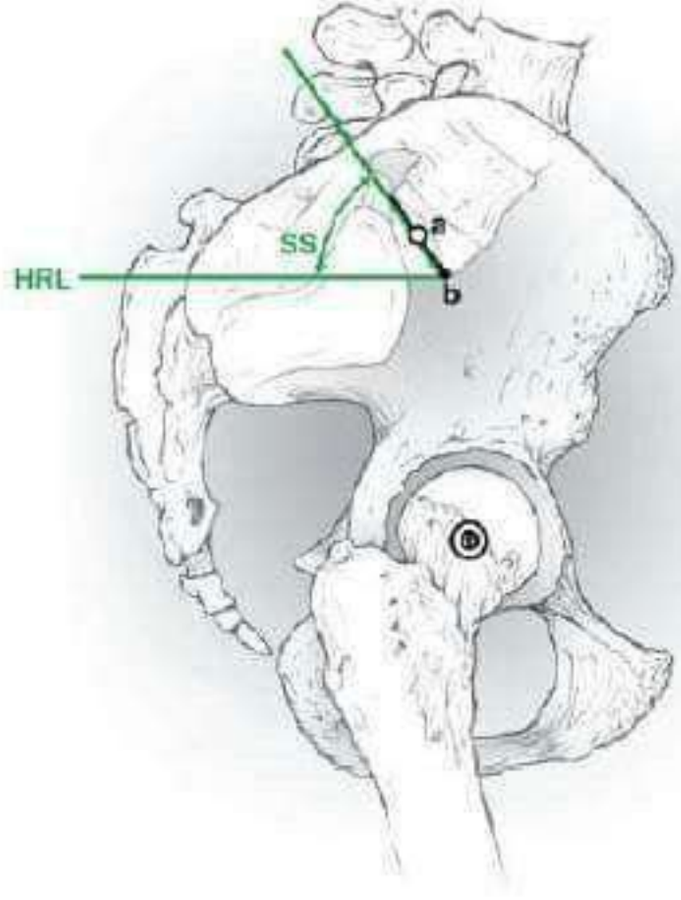
Following puberty, PI is generally considered to be a fixed morphological parameter, reflecting the relationship of the sacrum to the pelvis.

Assessment of Pelvic Parameters



Pelvic tilt is defined as the angle subtended by a line drawn from the midpoint of the sacral endplate to the center of the bicoxofemoral axis And a vertical plumb line extended from the bicoxofemoral axis.

Assessment of Pelvic Parameters



Sacral slope is defined as the angle subtended by a line drawn along the endplate of the sacrum and a horizontal reference line extended from the posterior superior corner of S-1.

Scoliosis:

Lateral curvature of spine >10’ associatited with vertebral rotation (3D deformity)

2 <u>type of classification :</u>

A- Cause related scoliosis:

1- Congenital
2- Idiopathic
3- Syndromatic (Marfan syndrome, Ehlers Danlos, Neurofibromatosis)
4- Neoplastic (osteoid osteoma)
5- Neuromuscular
6- Paralytic (CP)
7- Degenerative

B- Age related scoliosis:

Old classification:

1- Congenital
2- Infantile (=or < 3yr)
3- Juvenile (4-10 yr)
4- Adolescent (10-18 yr)
5- Adult (>18 yr)

Recent classification:

1- Early oncet. (befor 5 or 7 years)
2- Late oncet (after 5 or 7 years)

According to FRCS THE AGE OF ONCET 7 YEARS IS USED BY THE AO GROUP
OTHERS HAVE SUGGESTED AGE OF 5 YEARS

Chapter 6

The spine

Ashley A. Cole and Lee M. Breakwell

Spinal deformity

Spinal deformities can be divided into scoliosis and kyphosis, which affect either the whole spine or a region thereof, or spondylolisthesis, which usually affects a localized section of the spinal column.

Scoliosis

Scoliosis is defined as a frontal or coronal plane curvature with a Cobb angle of greater than 10°. The Cobb angle is defined as the maximal angle subtended by the endplates of the vertebrae within the curve.

Classification

1. Congenital,
2. Idiopathic,
3. Syndromic,
4. Neuromuscular,
5. Degenerative,
6. Paralytic.

Congenital

Congenital scoliosis is due to a developmental defect in the formation of the mesenchymal anlage. The resulting abnormal vertebra conveys uneven growth, creating angulation of the endplates and leading to unbalanced growth in adjacent vertebrae (Figure 6.1).

The defects are due to:

- Failure of formation: the commonest abnormality in this group is a hemivertebra, which may be fully, partially or unsegmented.
- Failure of segmentation: this results in block vertebrae, and unsegmented bars.
- A combination of these.

Associations

Once a diagnosis of congenital scoliosis has been made, the following associated anomalies must be sought and excluded:

- Spinal abnormalities (21–37%, MRI):
  - Hairy patch,
  - Dysraphism,
  - Myelomeningocele,
  - Diastematomyelia.
- Cardiac anomalies (12–26%, echo),
- Renal anomalies (20%, renal ultrasound),
- VACTERL association.

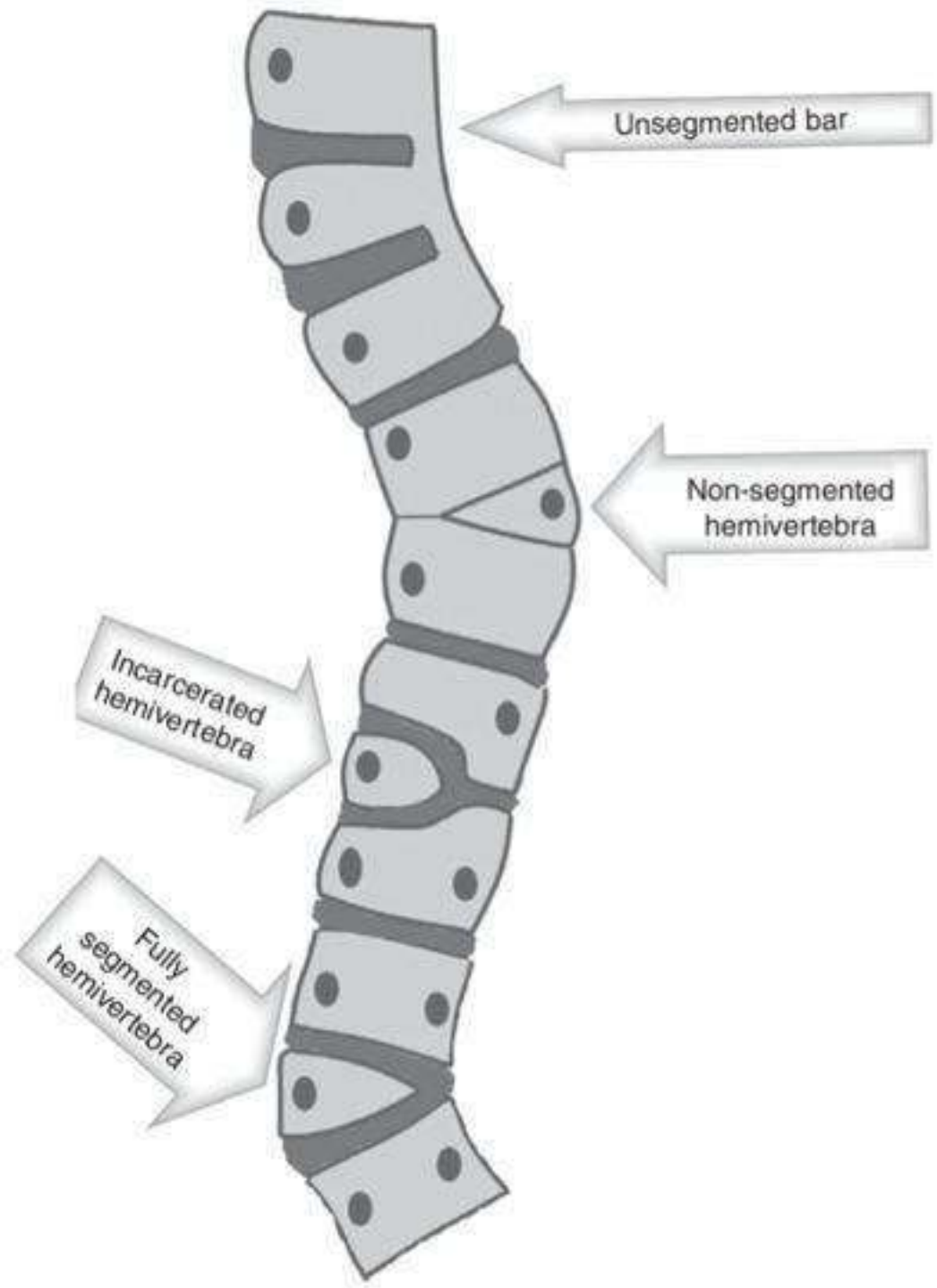


Figure 6.1 Congenital scoliosis.

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

Chapter 6: The spine

Risk of progression

Several factors will help to indicate the risk of progression and hence the indications for treatment:

1. Age of patient (remaining growth),
2. Site of the anomaly (worse at junctional regions, such as thoracolumbar and lumbosacral),
3. Type of anomaly (from worst to best):
   i. Unilateral unsegmented bar with contralateral fully segmented hemivertebra,
   ii. Unilateral unsegmented bar,
   iii. Fully segmented hemivertebra,
   iv. Partially segmented hemivertebra,
   v. Incarcerated hemivertebra,
   vi. Non-segmented hemivertebra.
4. Size of curve at presentation.

Idiopathic

Idiopathic scoliosis is by far the commonest form of scoliosis and affects approximately 3% of girls. Although the aetiology is unknown, there is growing evidence of a genetic causation.

Right thoracic curves are the commonest, followed by double major (right thoracic and left lumbar) then left lumbar.

Idiopathic scoliosis is subdivided into three forms based on the age of onset:

1. Infantile (0–3 years),
2. Juvenile (4–10 years),
3. Adolescent (>10 years).

Infantile

This represents less than 1% of all idiopathic curves, is commonly seen in boys and is usually a left thoracic curve.

It is the only true scoliosis that can resolve spontaneously: this can be predicted by measuring the rib–vertebra angle difference (RVAD) of Mehta on an AP radiograph.

This is derived by taking the angle of the concave and convex ribs to the apical vertebra bisector, and subtracting the concave from the convex angles (Figure 6.2).

An angle difference of greater or less than 20° implies a significant chance of progression or resolution respectively; 83% of the curves that resolved had an initial RVAD measuring less than 20°, whereas 84% of the curves that progressively worsened had an RVAD exceeding 20° [1].

Juvenile

Juvenile curves have a relatively high risk of progression due to the remaining growth potential. Approximately 70% will progress and many will require treatment. As many as 1 in 10 juvenile scoliosis patients are revealed to have a neural axis abnormality by MRI.

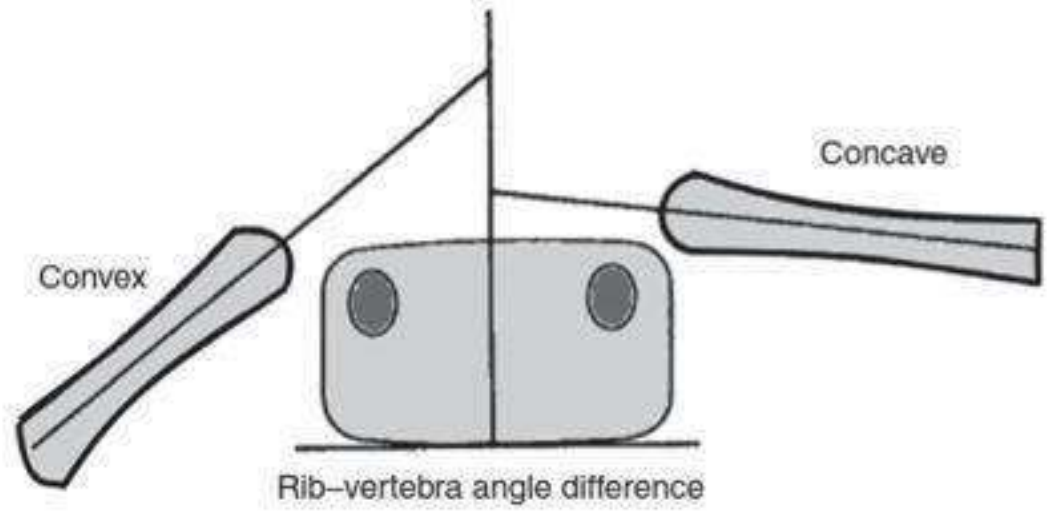


Figure 6.2 Congenital scoliosis, showing rib–vertebra angle difference.

Adolescent

This is the most common form of idiopathic scoliosis; it is commonly seen in girls.

Factors that aid in identifying risk of progression:

1. Curve size (>20°),
2. Remaining growth (curves worsen with growth). This is usually assessed:
   i. Clinically (menarche and peak height velocity (PHV)),
   ii. Radiological triradiate cartilage closure and Risser's stages (Figure 6.3).
3. Curve type (double curve and thoracic curve, thoracolumbar then lumbar).

In clinical practice, the PHV (i.e. growth spurt) is documented by serial measurement of the patient's height over time. The average age of the PHV is approximately 11.5 years in girls. Triradiate cartilage closure, a radiographic index of maturity, occurs after PHV and before Risser grade 1 and menarche.

Factors of no predictive value for curve progression before skeletal maturity include:

1. A family history of scoliosis,
2. Patient height-to-weight ratio,
3. Lumbosacral transitional anomalies,
4. Thoracic kyphosis,
5. Lumbar lordosis,
6. Spinal balance.

Syndromic

Scoliosis is a common feature of many well-known syndromes. In addition, it is present in many rare syndromes that are regularly seen in spinal clinics. Common syndromes with scoliosis include:

- Neurofibromatosis,
- Marfan syndrome,
- Ehlers–Danlos syndrome.

Rarer syndromes with scoliosis include:

- Rett,
- Sotos,
- Prader–Willi.

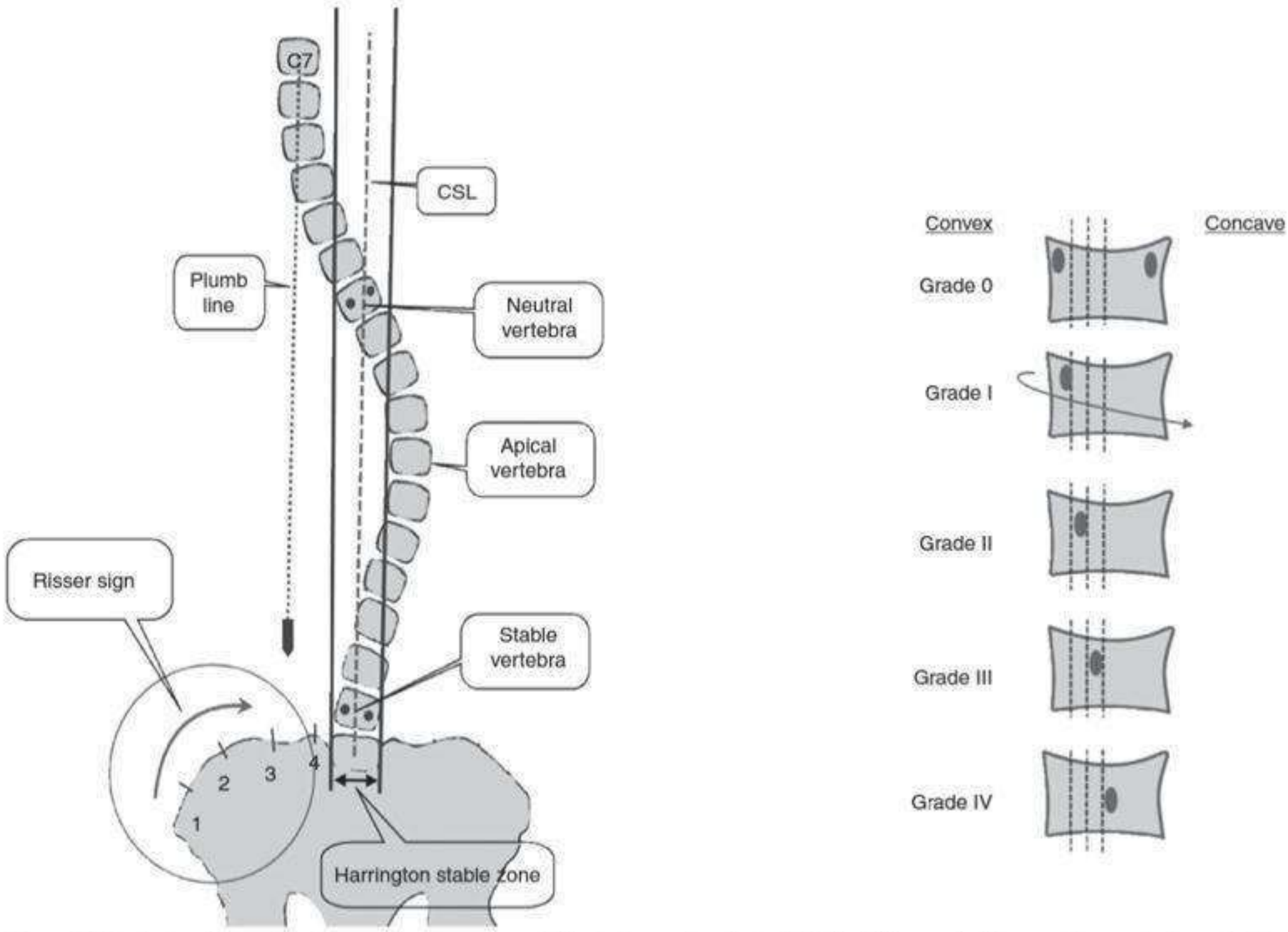


Figure 6.3 Scoliosis radiology. Left: Diagram showing various radiological terms of scoliosis. Right: Nash–Moe method for assessing vertebral rotation. Grade 0: Both pedicles are symmetric, Grade I: the convex pedicle has moved away from the side of the vertebral body. Grade III: the convex pedicle is in the centre of the vertebral body. Grade II: the rotation is between Grades I and III and Grade IV when the convex pedicle has moved past the midline. The curved arrow denotes the direction of the rotation. CSL, central sacral line.

The majority of these patients already have a diagnosis prior to identification of the scoliosis; however, in some patients, the spinal deformity is the index problem.

Neurofibromatosis scoliosis is the most common skeletal manifestation of neurofibromatosis.

The cause is unknown but various theories have been proposed, such as primary mesodermal dysplasia, erosion or infiltration of the bone by localized neurofibromatosis tumours, and endocrine disturbances.

Neurofibromatosis scoliosis can be either:

- Non-dystrophic,
- Dystrophic.

Differentiation between the two types is important because the prognosis and management differ significantly.

Dystrophic scoliosis is more common (Figure 6.4), usually located in the thoracic region, and has a short (4–6 vertebrae), sharply angled curve. It has a greater tendency to progress, and is at risk of developing neurologic deficits. Non-dystrophic scoliosis more closely resembles idiopathic scoliosis in both curve patterns and behaviour.

Neuromuscular

These curves are usually long, less likely to have compensatory curves and may progress after maturity. Pulmonary problems, such as decreased lung function, are observed.

This form of scoliosis is divided into two subgroups:

- Cerebral palsy,
- True neuromuscular diseases, in which there is primary nerve or muscle disorder, e.g. Duchenne muscular dystrophy.

Cerebral palsy

The incidence of spinal deformity increases with the severity of cerebral palsy. It is around 20%; however, in the quadriplegic group, the incidence is in excess of 70%.

Chapter 6: The spine

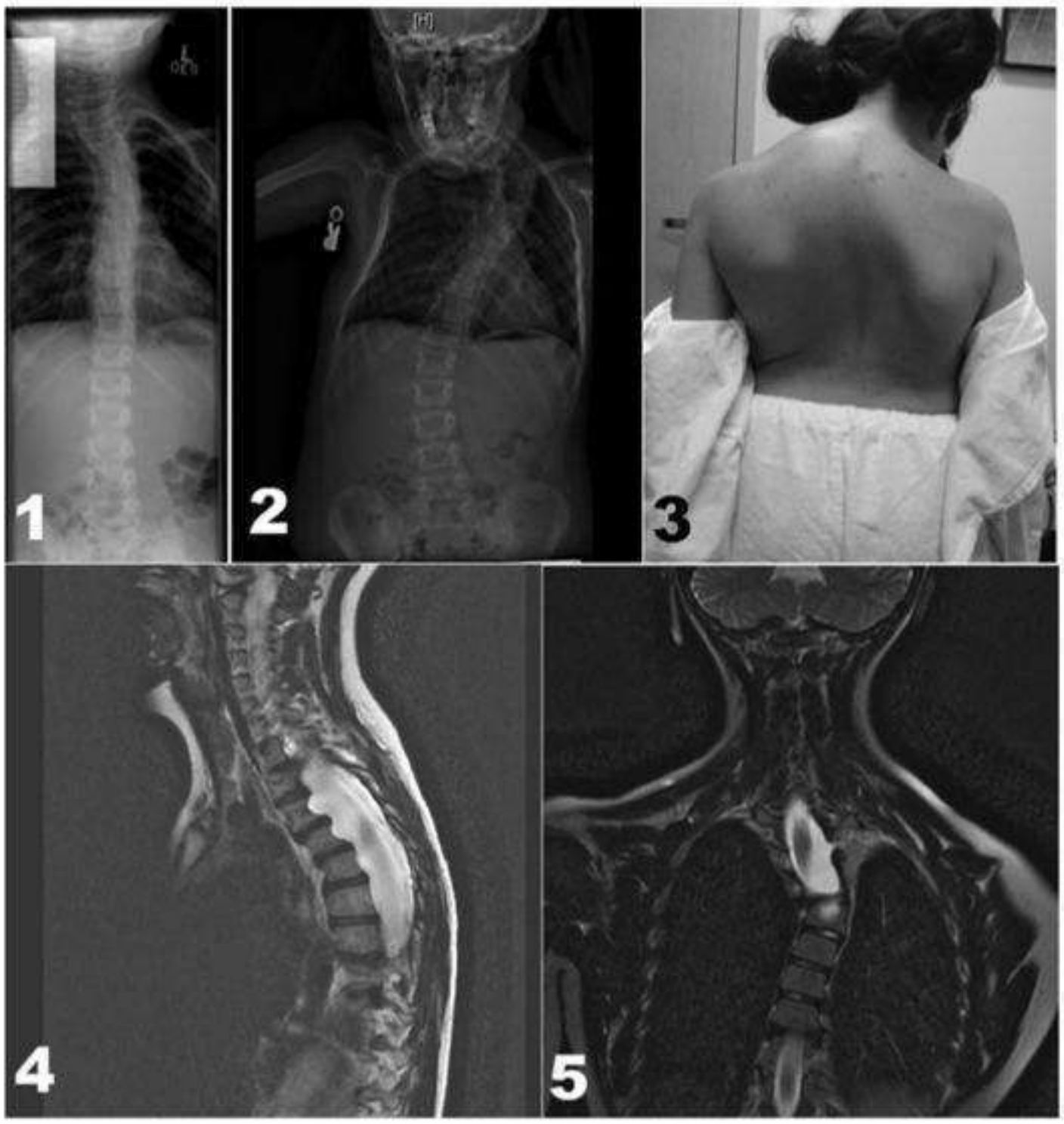


Figure 6.4. Dystrophic scoliosis in a child with neurofibromatosis scoliosis. 1, 2: There was a rapid progression in the severity over 2 years. 3: Café au lait spots and a sharply angled kyphoscoliosis. The kyphosis is more pronounced than the scoliosis. 4, 5: MRI scan shows large dural ectasia and scalloping of the posterior vertebrae. 5: There is a large plexiform neurofibroma in continuity with a nerve root.

The ability to walk is relatively protective, and therefore wheelchair dependence is a risk factor for development and progression of a spinal curvature.

Duchenne muscular dystrophy

This X-linked recessive condition, affecting the production of dystrophin, almost exclusively affects boys, with their mothers as the carrier. Historically, wheelchair dependence happened at about 10 to 11 years of age, following which the patient developed a scoliosis. Curves beyond 20° were seen to progress inexorably and surgery was advised at an early stage, to prevent further respiratory embarrassment.

Since the advent of steroid treatment, the progression of weakness has significantly slowed, and many subjects continue with some form of ambulation into their teens. This, coupled with the fact that treated curves do not always progress, means that the indication for surgery has changed, and not all patients require surgery.

The decision to undertake corrective surgery can now often be left until the curve has progressed to 40° or more, as the child is often older, and the lung function better than the historical cohort.

Spinal muscular atrophy

This autosomal recessive muscular wasting disease commonly causes scoliosis, which is often progressive. A defect in the *SMN1* gene leads to loss of the SMN protein, which is vital for muscle function. There are three types of spinal muscular atrophy in children, with function and life expectancy increasing from Type I through to Type III. Respiratory function can be severely restricted; hence, scoliosis is a major concern. The median survival in Type I spinal muscular atrophy is 7 months, with a mortality rate of 95% by the age of 18 months. In Type II spinal muscular atrophy, the age of onset is between 6 and 18 months and the age of death varies. The decision to undertake surgery for spinal deformity must be made in close collaboration with respiratory physicians, ideally considering life expectancy and function, as well as risk of curve progression.

Degenerative

This form of curve typically develops in the fifth or later decade of life, often in a previously normal spine; hence, it is beyond the scope of this book.

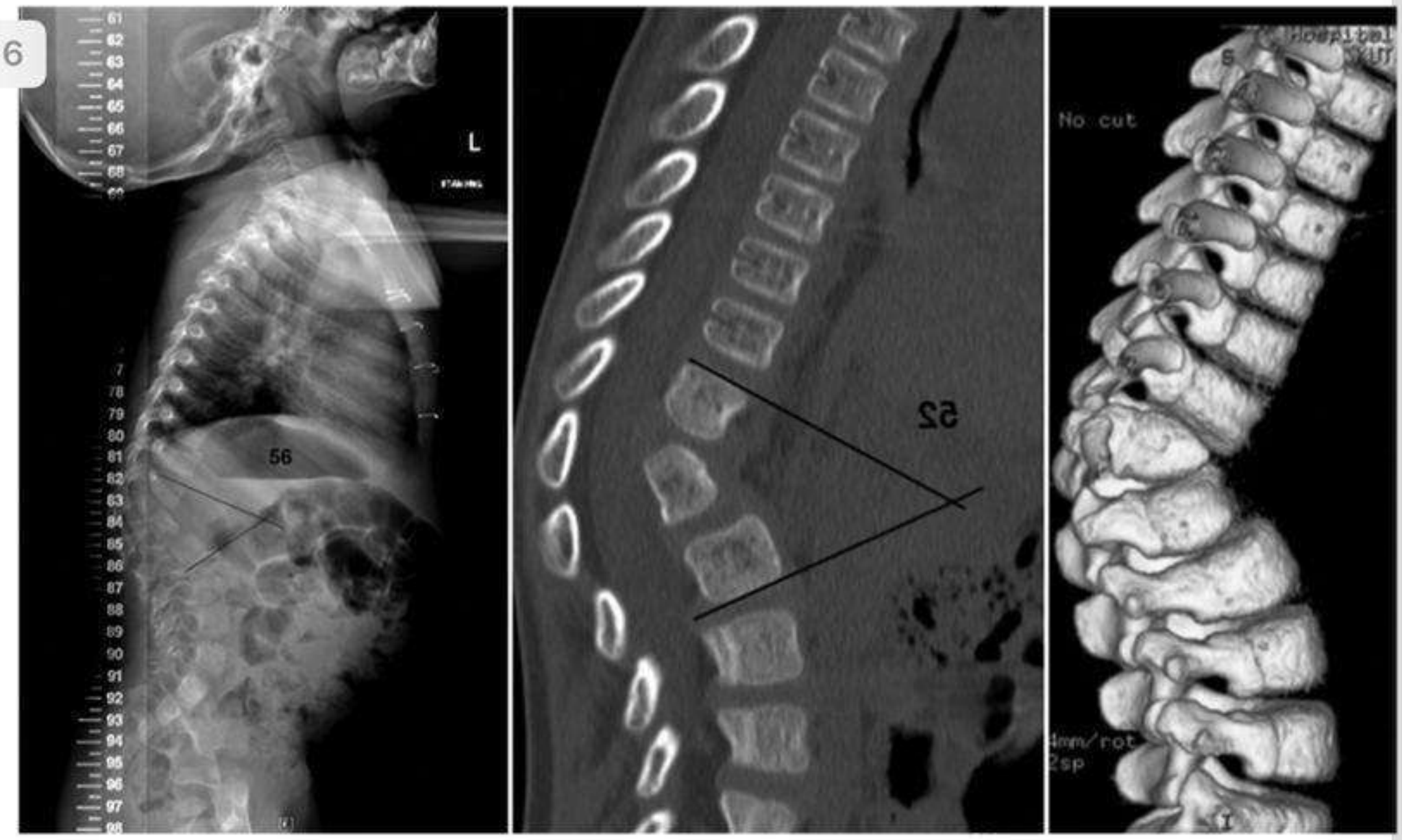


Figure 6.5 Congenital kyphosis.

Paralytic

Spinal cord injury with resultant paralysis before the onset of the adolescent growth spurt leads to the development of scoliosis in 97% of patients [2].

Kyphosis

Kyphosis is a forward curvature of the spine in the sagittal plane. A certain degree of thoracic kyphosis (20°–50°) is normal and desirable for spinal balance. Thoracic kyphosis does not strictly have a normal value, as it exhibits a range throughout different body shapes.

Classification

1. Congenital,
2. Idiopathic,
3. Neuromuscular,
4. Syndromic,
5. Traumatic,
6. Degenerative.

Congenital

As in scoliosis, this deformity develops due to an underlying structural disorder. The same basic types exist as in the coronal plane scenario.

A hemivertebra positioned posteriorly (Figure 6.5) will gradually deform the spine in a kyphotic direction. This causes a localized angular, deformity called a gibbus.

As the angulation progresses with growth, the centre of gravity of the body moves forward, increasing the load on the anterior aspect of the vertebral ring apophysis. This impedes anterior growth, in compliance with the Hueter–Volkmann law, unbalancing in favour of posterior height increase and leading to worsening of the kyphosis. By this process, kyphosis progresses throughout growth, and, if beyond 90°, continues into adulthood.

Whereas scoliotic deformity causes neurological deficit extremely rarely, congenital kyphosis has a relatively high risk of curve progression and neurological deficit when the angle is localized and beyond 90°.

Idiopathic

Scheuermann's kyphosis is seen in children older than 10 years and is more common in boys. The incidence ranges from 1–8%.

The accepted pathoaetiology is that slight kyphosis in the growing spine causes an anterior shift in the body weight centre, unevenly loading the anterior apophysis. This then leads to fragmentation and poor growth, as seen in the anterior vertebral body. It is characterized by vertebral wedging, disc space narrowing, endplate irregularities, including Schmorl's nodes, and kyphosis (Figure 6.6).

Age of onset – Idiopathic scoliosis has been classified by age into infantile (age 0–3 years), juvenile (3–10 years) and adolescent (10–maturity) idiopathic scoliosis. An alternative classification divides scoliosis into early-onset scoliosis (associated with a high risk of cardiorespiratory compromise as the developing heart and lungs may be affected) which has its onset before the age of 7 and late-onset scoliosis which has its onset after the age of 7. (The age of 7 is used by the AO group – others have suggested 5.)[1,2]

Chapter 6: The spine

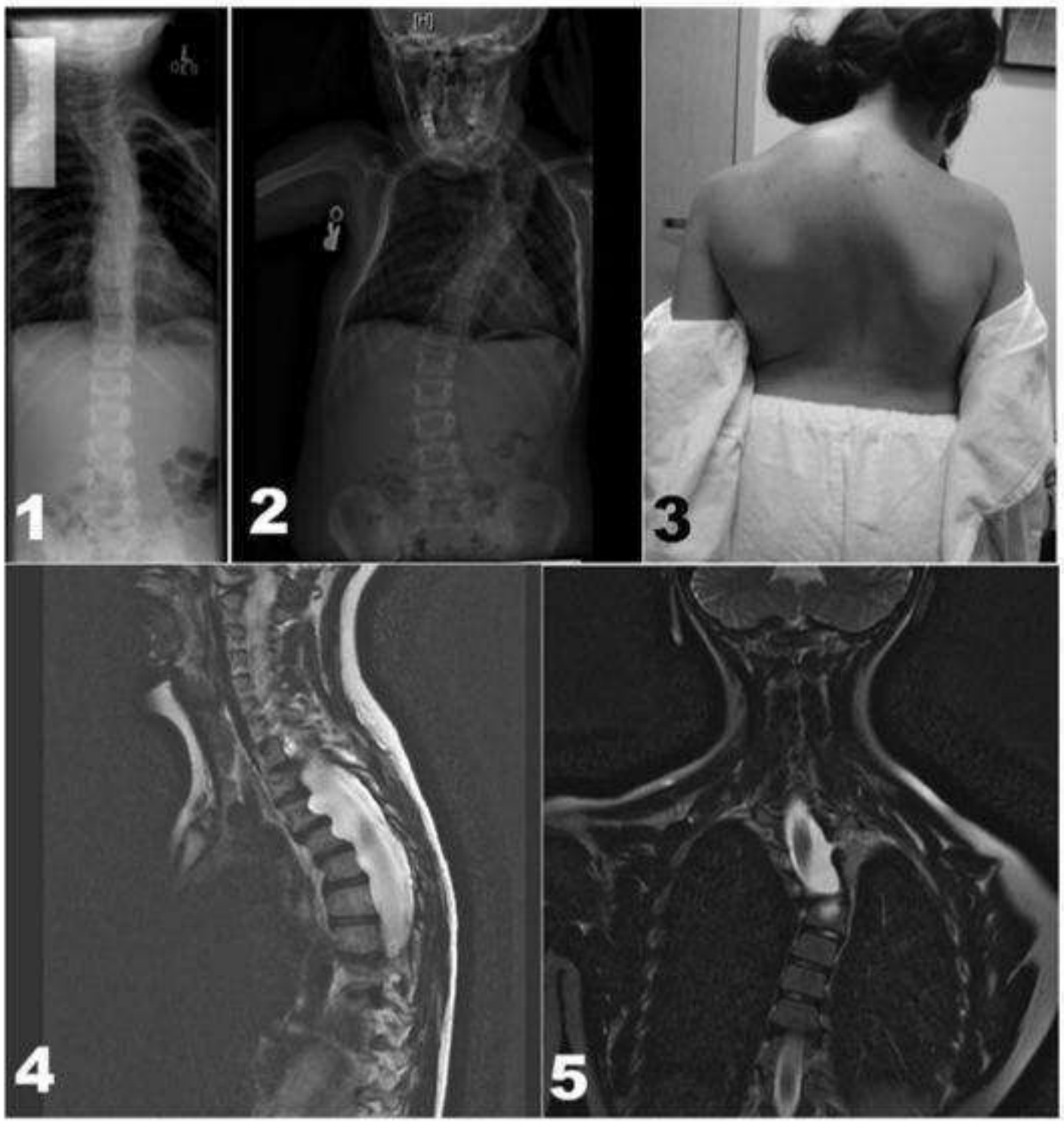


Figure 6.4. Dystrophic scoliosis in a child with neurofibromatosis scoliosis. 1, 2: There was a rapid progression in the severity over 2 years. 3: Café au lait spots and a sharply angled kyphoscoliosis. The kyphosis is more pronounced than the scoliosis. 4, 5: MRI scan shows large dural ectasia and scalloping of the posterior vertebrae. 5: There is a large plexiform neurofibroma in continuity with a nerve root.

The ability to walk is relatively protective, and therefore wheelchair dependence is a risk factor for development and progression of a spinal curvature.

Duchenne muscular dystrophy

This X-linked recessive condition, affecting the production of dystrophin, almost exclusively affects boys, with their mothers as the carrier. Historically, wheelchair dependence happened at about 10 to 11 years of age, following which the patient developed a scoliosis. Curves beyond 20° were seen to progress inexorably and surgery was advised at an early stage, to prevent further respiratory embarrassment.

Since the advent of steroid treatment, the progression of weakness has significantly slowed, and many subjects continue with some form of ambulation into their teens. This, coupled with the fact that treated curves do not always progress, means that the indication for surgery has changed, and not all patients require surgery.

The decision to undertake corrective surgery can now often be left until the curve has progressed to 40° or more, as the child is often older, and the lung function better than the historical cohort.

Spinal muscular atrophy

This autosomal recessive muscular wasting disease commonly causes scoliosis, which is often progressive. A defect in the *SMN1* gene leads to loss of the SMN protein, which is vital for muscle function. There are three types of spinal muscular atrophy in children, with function and life expectancy increasing from Type I through to Type III. Respiratory function can be severely restricted; hence, scoliosis is a major concern. The median survival in Type I spinal muscular atrophy is 7 months, with a mortality rate of 95% by the age of 18 months. In Type II spinal muscular atrophy, the age of onset is between 6 and 18 months and the age of death varies. The decision to undertake surgery for spinal deformity must be made in close collaboration with respiratory physicians, ideally considering life expectancy and function, as well as risk of curve progression.

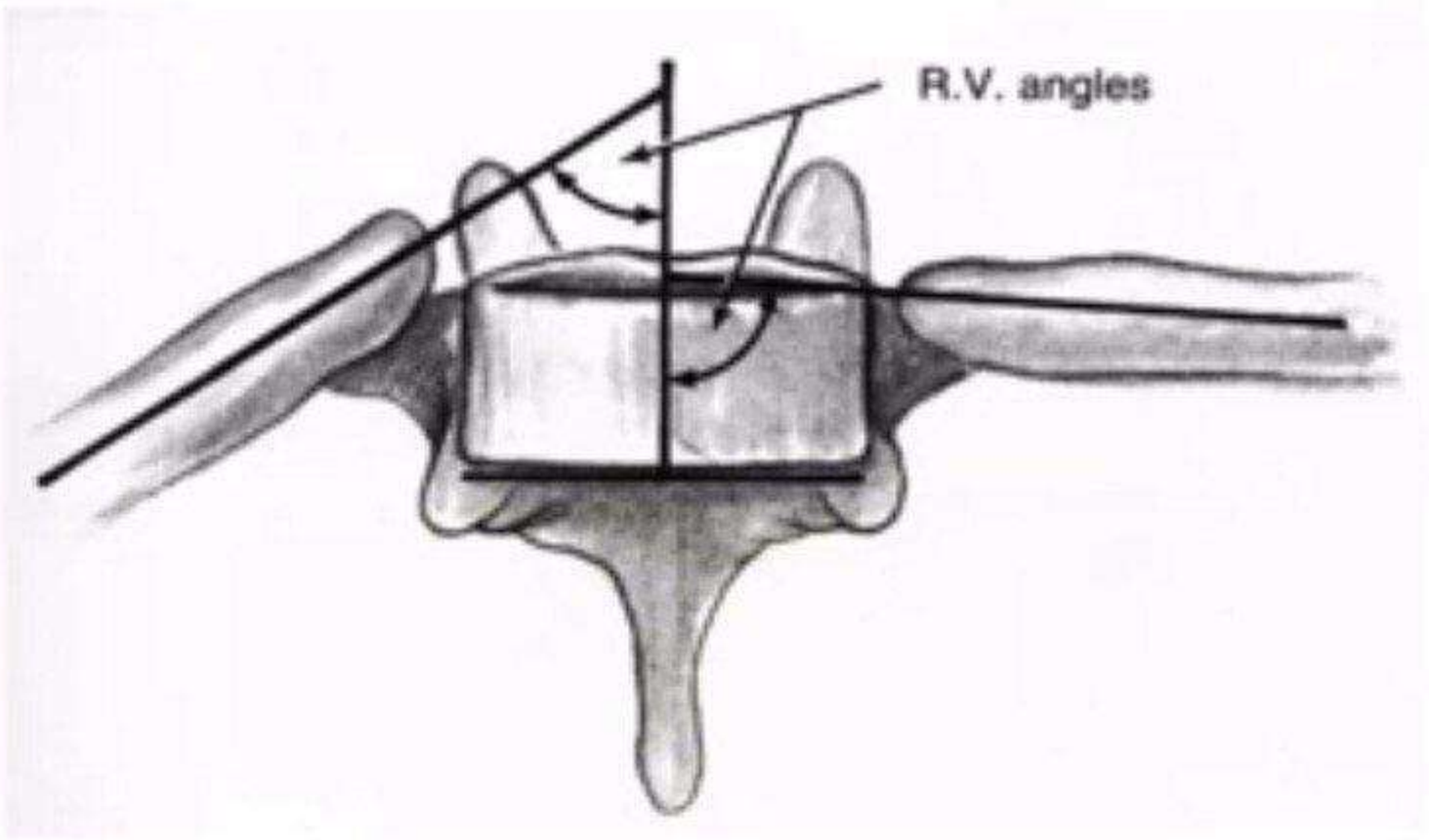
Degenerative

This form of curve typically develops in the fifth or later decade of life, often in a previously normal spine; hence, it is beyond the scope of this book.

Risk factors for progression

Age	The younger the age, the greater potential for growth and progression of curve with a rapid rise in progression at the onset of adolescent growth spurt
Sex	Progression is more common in girls
Menarche	Progression is least common after menarche
Risser sign	The incidence of progression has been shown to decrease as the Risser sign increases
Curve pattern	Double curves progress more frequently than single curves
Curve magnitude	The incidence of progression increases with curve magnitude

Rib Vertebral Angle Difference



Critical RVAD value of

20 degrees

Phase of rib head

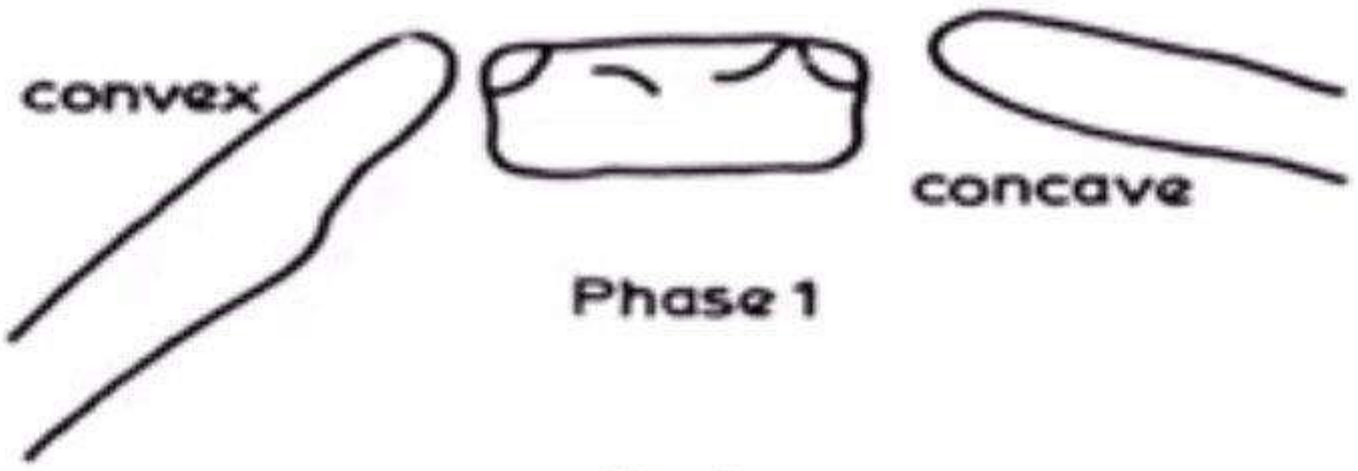
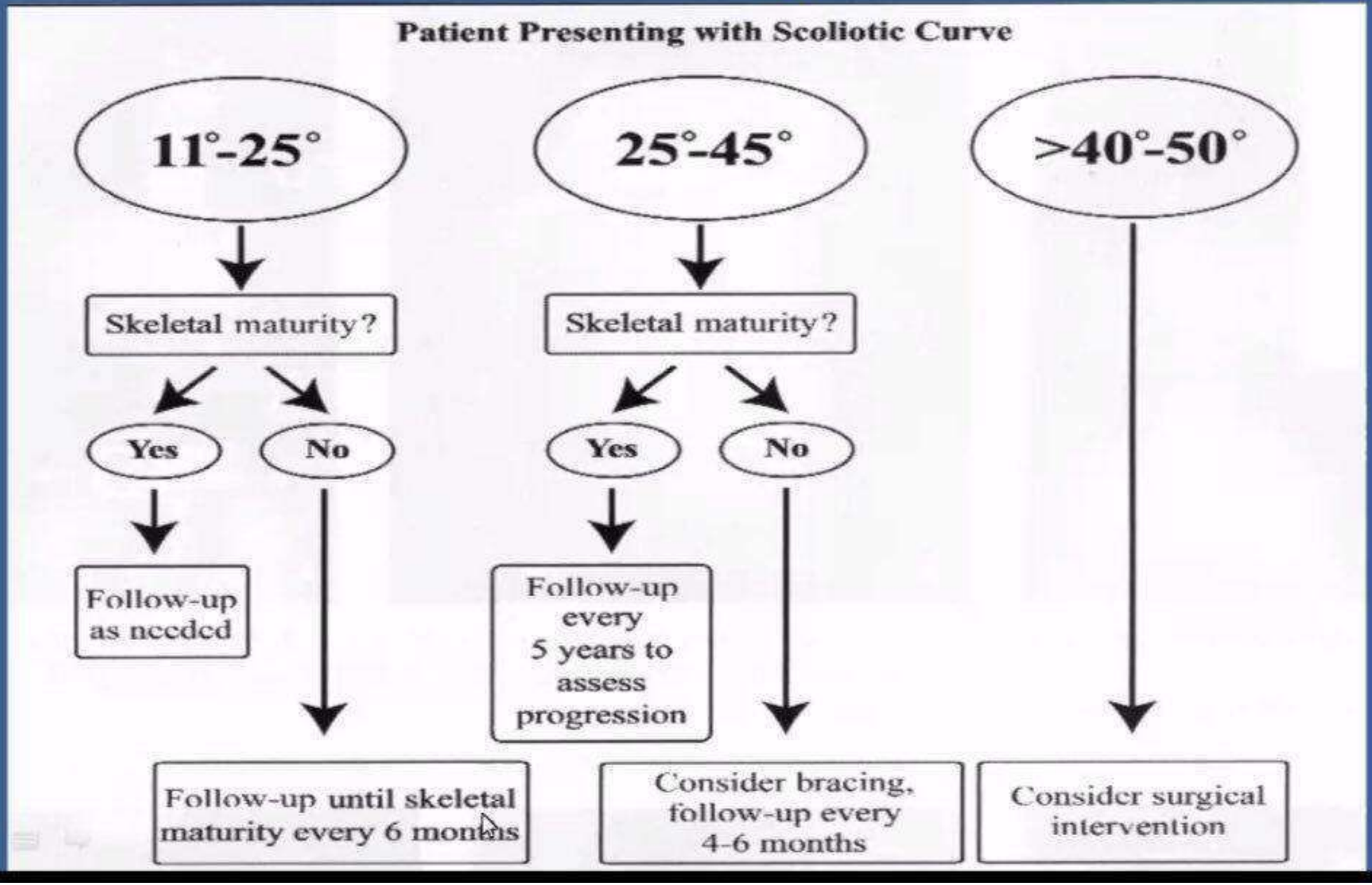


FIG. 1
The outlines of the apical vertebra and ribs at an early stage of scoliosis.

FIG. 2
The outlines at a later stage, showing overlap of rib and vertebra.

Progressive curve – a shift from Phase I to Phase II

Treatment Algorithm



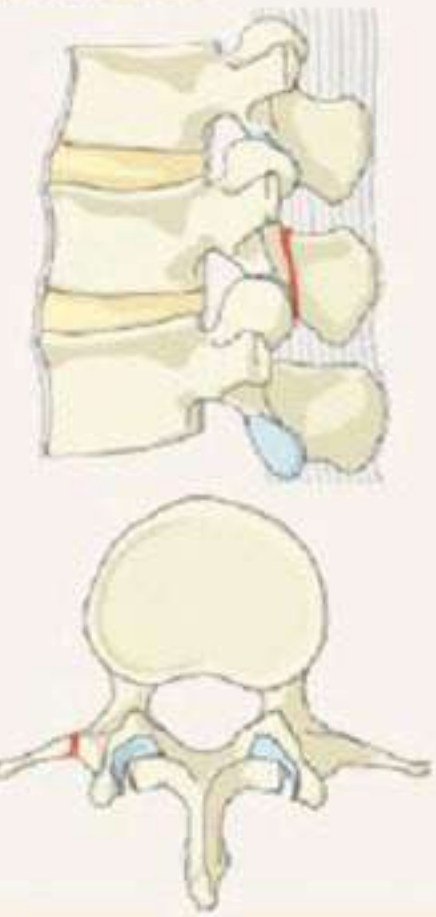
- **Neurologic Monitoring**
  - monitoring with somatosensory-evoked potentials (SSEPs) and/or motor-evoked potentials (MEPs) is now the standard of care
    - motor-evoked potentials can provide an intraoperative warning of impending spinal cord dysfunction
  - neurologic event defined as drop in amplitude of > 50%
  - if neurologic injury occurs intraoperatively consider
    - check for technical problems
    - check blood pressure and elevate if low
    - check hemoglobin and transfuse as necessary
    - lessen/reverse correction
    - administer Stagnaras wake up test
    - remove instrumentation if the spine is stable

	Type of claudication	
Symptom	**Neurogenic**	**Vascular**
Pain	Worse on standing	Relieved by standing
Numbness	Present	Absent
Site of pain	Buttock/thigh	Calf (rarely anterior)
Relieving factors	Bending forward	Standing
Walking distance	Reduced and variable	Reduced and fixed
Worse going	Downstairs	Upstairs

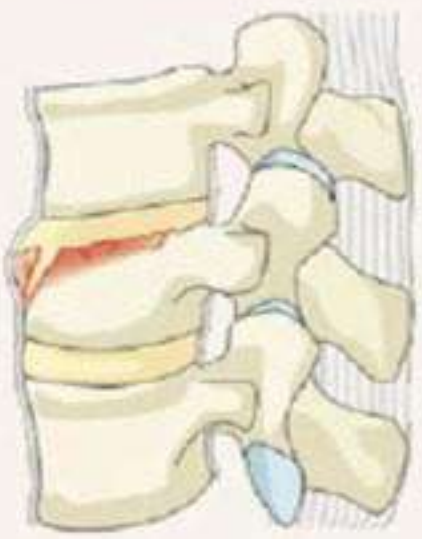
TLICS	3 independent predictors		
1 **Morphology** immediate stability	- Compression - Burst - Translation/rotation - Distraction	1 2 3 4	- Radiographs - CT
2 **Integrity of PLC** longterm stability	- Intact - Suspected - Injured	0 2 3	- MRI
3 **Neurological status**	- Intact - Nerve root - Complete cord - Incomplete cord - Cauda equina	0 2 2 3 3	- Physical examination
Predicts	- **Need for surgery**	0 – 3 4 > 4	- nonsurgical - surgeon's choice - surgical

Type A. Compression Injuries

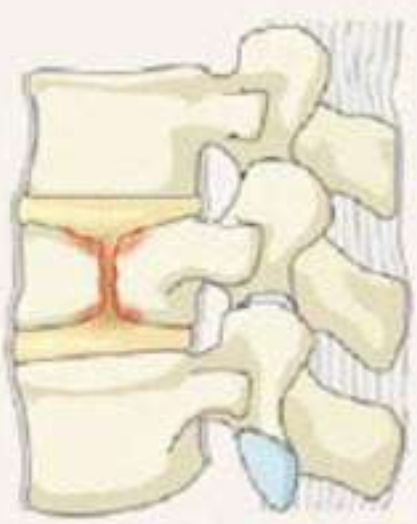
A0. Minor, nonstructural fractures



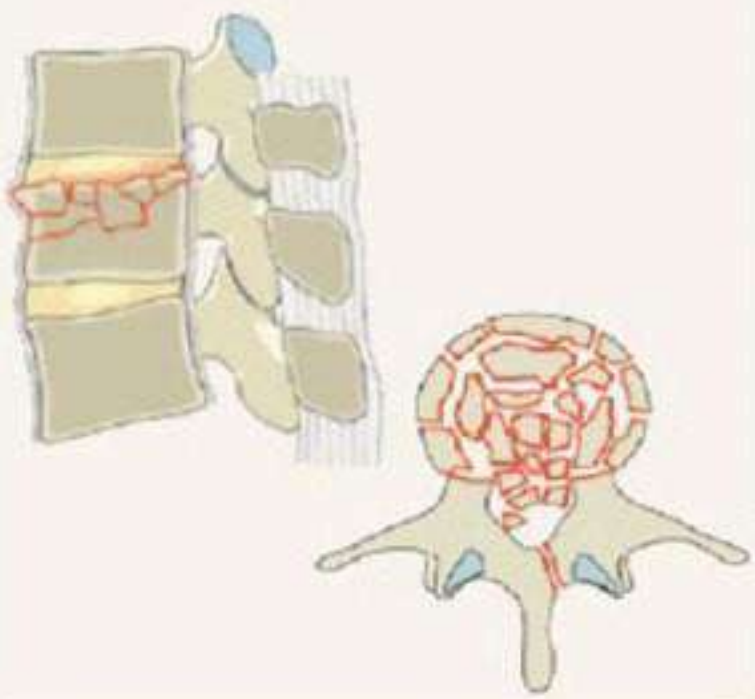
A1. Wedge-compression



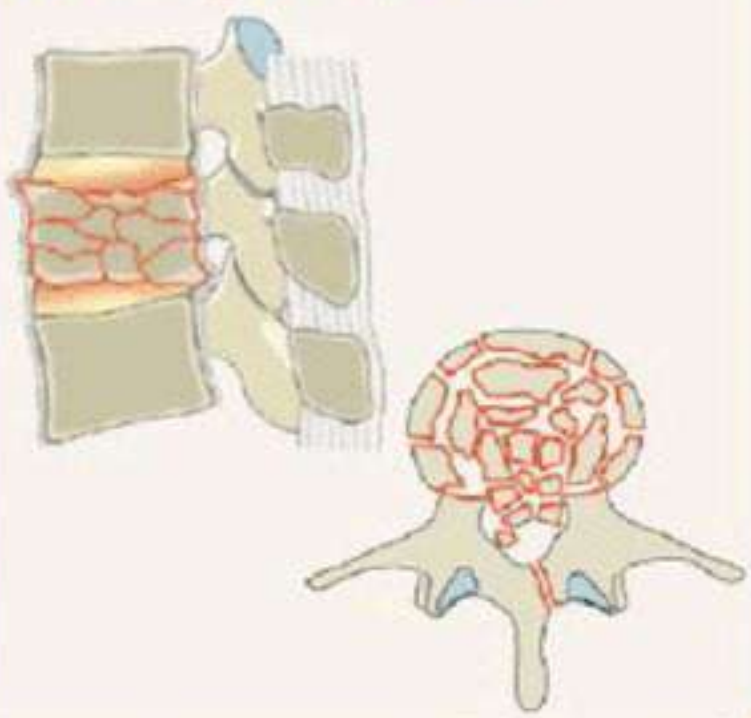
A2. Split



A3. Incomplete burst

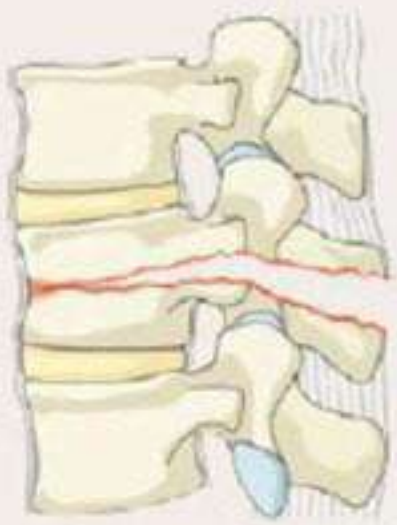


A4. Complete burst

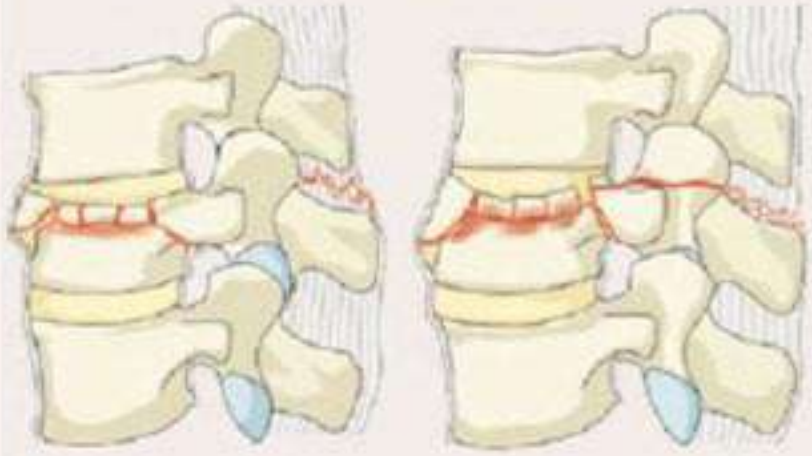


Type B. Distraction Injuries

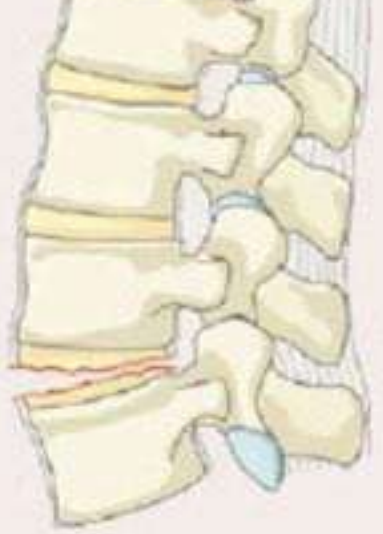
B1. Transosseous tension band disruption / Chance fracture



B2. Posterior tension band disruption

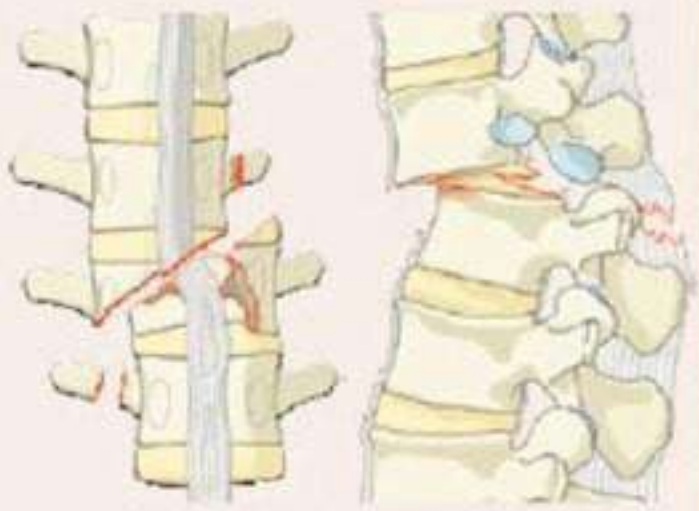


B3. Hyperextension



Type C. Translation Injuries

C. Displacement / Dislocation



Characteristic	Score
General condition	
Poor (PS 10% to 40%)	0
Moderate (PS 50% to 70%)	1
Good (PS 80% to 100%)	2
Number of extraspinal metastatic foci	
≥3	0
1-2	1
0	2
Number of metastases in vertebral body	
≥3	0
2	1
1	2
Metastases to other internal organs	
Unresectable	0
Resectable	1
Absent	2
Primary site of malignancy	
Lung, osteosarcoma, stomach, bladder, esophagus, or pancreas	0
Liver, gallbladder, unidentified	1
Others	3
Kidney, uterus	4
Thyroid, breast, prostate, carcinoid	5
Palsy	
Complete (Frankel A, B)	0
Incomplete (Frankel C, D)	1
Non (Frankel E)	2

Total Score	Months
0-8	>6
9-11	≥6
12-15	≥12

TOKOHASHI SCORE

B. Viva : Long & Short

To predict the LLD at a desired age, we need to draw a line that best fits the previous skeletal ages of measurement. From the intersection of this line with the desired age line, another line is drawn parallel to the side of the chart; its intersections with the long leg and short leg lines represent the length of these legs, respectively. The difference is the LLD at that desired age. In this example, the desired age is skeletal maturity; 5.5 cm.

Examiner: What about that small square called 'reference slope'?
Candidate: This helps predict the correct timing of the epiphysiodesis to treat LLD, as per Figure 19.7.

Topic 3: Paediatric spine

Viva practice 3

Examiner: This is a 15-year-old girl with adolescent idiopathic scoliosis. How would you describe her radiographs?
Candidate: These radiographs are a scoliosis series for this patient with a standing PA and lateral views on the top and right and left bending films (the right and left parts, respectively) at the bottom. From the standing PA and lateral views, I can see that she has a thoracic curve, which is convex to the left side, and a thoracolumbar curve, which is convex to the right side. Both curves appear to be structural as they do not correct completely in the bending films. (However, I would measure the Cobb angles and see if they are correcting to less than 25° or not, according to Lenke [5].) The lateral X-radiographs show some increased thoracolumbar kyphosis; however, I should again measure the Cobb angle to know exactly how much. Triradiate cartilages are closed on both sides; regarding iliac apophysis, it is Risser grade 3 to 4.

Examiner: What are the main steps in your clinical assessment of such cases?
Candidate:

- History:
  - Main concern (pain or cosmesis),
  - Family history,
  - Growth and developmental history,
  - Onset of menses,
  - Review of systems and other work-ups.

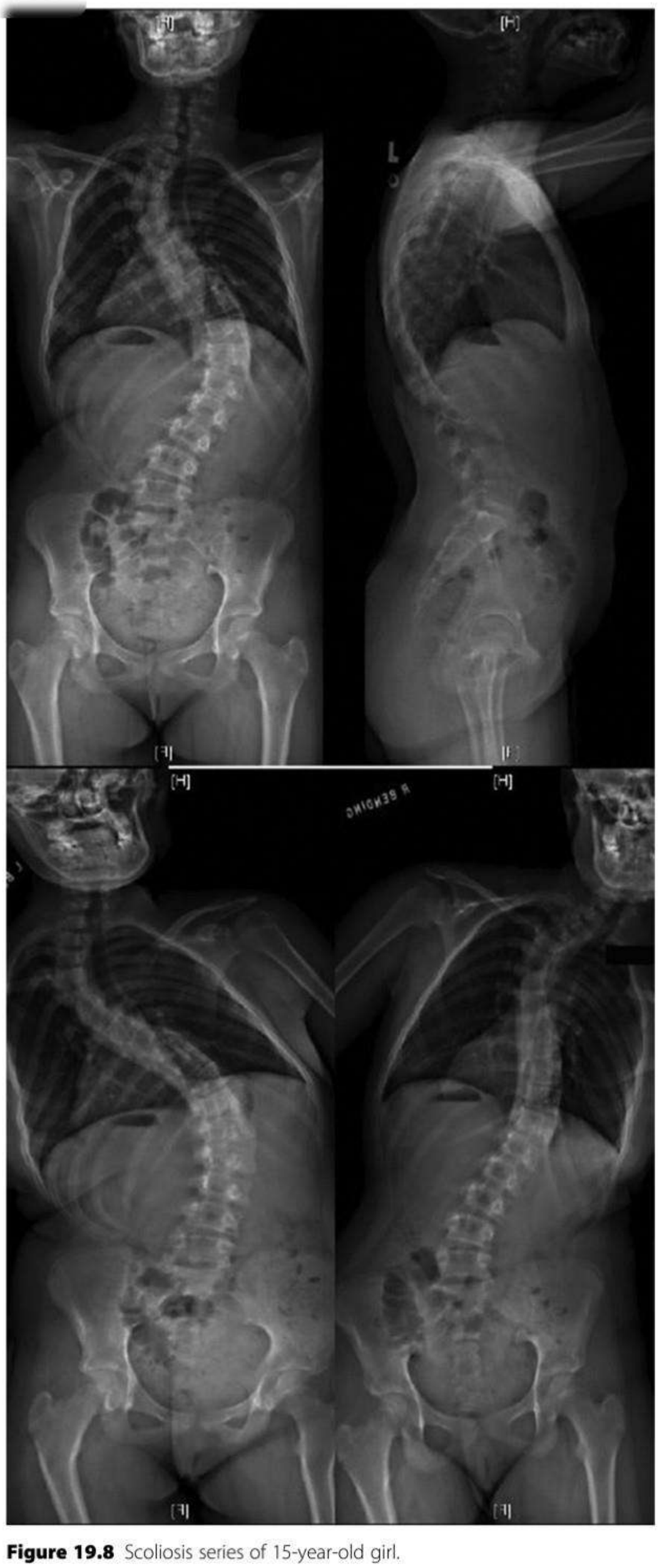


Figure 19.8 Scoliosis series of 15-year-old girl.

- Physical examination:
  - Height and weight growth chart,
  - Arm span to height ratio,
  - Limb length discrepancy,
  - Shoulder heights,
  - Rib prominence, waist asymmetry and lumbar prominence,
  - Angle of trunk rotation as measured by scoliometer on Adam's forward bending test both in the thoracic and lumbar areas (Figure 6.6),
  - Flexibility or laxity – Beighton scale,
  - Skin examination and presence of café au lait spots,
  - Complete neurological examination, including abdominal reflexes.

Examiner: How can you define different curve types?
Candidate: Each curve can be defined mainly by its apical vertebra or disc.
The apical vertebra or disc is the vertebra or disc with the greatest rotation and the farthest lateral deviation from the centre of the vertebral column and the horizontal (the least tilted in the curve).
The end vertebrae on both sides of a curve are those with the maximum tilt toward the concavity of the curve.
Neutral vertebrae are those that show no evidence of rotation on standing PA radiographs.
Curve types are mainly described according to the location of the apex of the curve (Scoliosis Research Society definitions) and then according to the direction of the convexity, either convex to the left or to the right.

Examiner: How do you measure the Cobb angle?
Candidate: The Cobb angle is the angle formed by the intersection of two lines, one parallel to the superior endplate of the superior end vertebra and the other parallel to the inferior endplate of the inferior end vertebra. If the endplates are obscured, the pedicles can be used instead. (See Chapter 6 for more detail.)

Examiner: What are the main limitations in measuring the Cobb angle?
Candidate: The main limitations are [6]:

- A diurnal variation of 5° has been observed in Cobb angle measurements of the same curve over the course of a single day, with an angular increase occurring in the afternoon.
- Because of the vertebral rotation associated with scoliosis, it may be difficult to position the patient so as to obtain an accurate frontal view, and the actual Cobb angle might be 20% greater than that plotted on radiographs.
- The Cobb angle varies with supine, prone (including during surgery), sitting and standing views.
- A total error of 2°–7° in Cobb angle assessment has been reported to result from variations in radiographic acquisitions and measurement error. Because the measurement error is smaller when end vertebrae are consistently defined, the same endpoints should be used at follow-up as at the initial curve assessment.
- An intra-observer variation of 5°–10° in Cobb angle measurement has been reported, and the inter-observer variation may be even greater.

Table 19.3 Lenke classification system

Curve type	Description	Proximal thoracic	Main thoracic	Thoracolumbar, lumbar
1	Main thoracic (MT)	Non-structural	Structural	Non-structural
2	Double thoracic (DT)	Structural	Structural (major)	Non-structural
3	Double major (DM)	Non-structural	Structural (major)	Structural
4	Triple major (TM)	Structural	Structural (major)	Structural
5	Thoracolumbar/lumbar (TL/L-M)	Non-structural	Non-structural	Structural
6	Thoracolumbar/lumbar – main thoracic (TL/L-MT)	Non-structural	Structural	Structural (major)

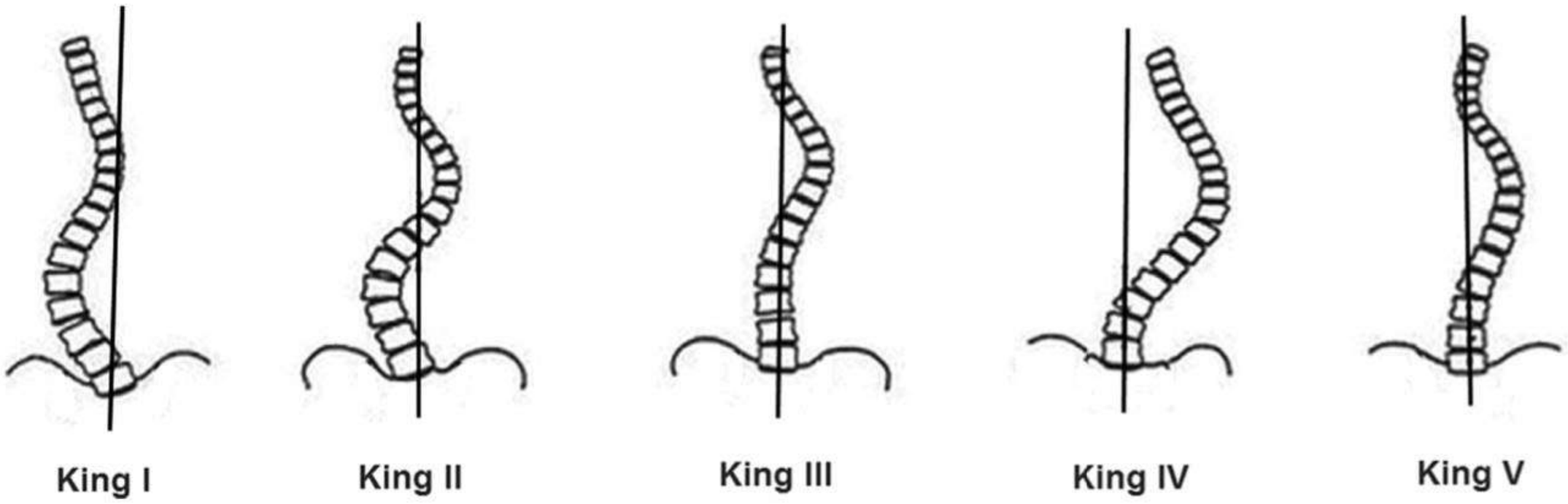


Figure 19.9 King's classification.

Examiner: How can you assess the vertebral alignment and balance on radiographs?

Candidate: The plumb line is a vertical line drawn downward from the centre of the C7 vertebral body, parallel to the lateral edges of the radiograph.

It is used to evaluate coronal balance on standing frontal radiographs and sagittal balance on standing lateral radiographs. Coronal balance is evaluated by measuring the distance between the CSL (central sacral vertical line) and the plumb line, and sagittal balance is evaluated by measuring the distance between the posterosuperior aspect of the S1 vertebral body and the plumb line. For both coronal and sagittal measurements, balance is considered abnormal if the distance is greater than 2 cm.

Examiner: Which classification system would you use for adolescent idiopathic scoliosis?

Candidate: There are two common classification systems. King and Moe described Types I to V, depending on the shape of the curve.

Type I: S-shaped double curve, where the lumbar curve is larger or less flexible,

Type II: S-shaped double curve, where the thoracic curve is larger or less flexible,

Type III: Single thoracic curve,

Type IV: Long thoracic curves, where L4 is tilted into the curve,

Type V: Double thoracic curve, where T1 is tilted into the thoracic curve.

The other widely used classification system for adolescent idiopathic scoliosis is the Lenke classification system. It is more sophisticated and complicated. According to Lenke, there are four types of curve pattern:

1. Proximal thoracic (apical vertebra located at T3–5),
2. Main thoracic (apical vertebra located at T6–11),
3. Thoracolumbar (apical vertebra located at T12–L1),
4. Lumbar (apical vertebra located at L2–4).

Each curve is further classified into a major curve (the curve segment with the larger Cobb angle) and a minor curve (the curve segment with the smaller Cobb angle). Major curves are always considered structural, while minor curves can be structural if they meet the following criteria:

- Minimal residual coronal curve on bending film of at least 25°,
- Kyphosis of at least 20°.

There are six curve types, corresponding to different combinations of structural and non-structural curve patterns (Table 19.3).
Lenke further proposed two modifiers.

A lumbar modifier to emphasize the importance of deformities in the lumbar region, as this affects spinal balance as well as proximal curves. The lumbar modifier could be A, B or C:

- **A:** Central sacral vertical line (CSL) passes between pedicles up to the stable vertebra,
- **B:** CSL touches the apical body up to the stable vertebra,
- **C:** CSL passes medial to apical body up to the stable vertebra.

A thoracic spine sagittal modifier to introduce a three-dimensional analysis to the classification system. The thoracic spine sagittal modifier is denoted by −, N or +, based on the sagittal Cobb angle (between T5 and T12):

- **−:** Hypo <10°,
- **N:** Normal 10–40°,
- **+:** Hyper >40°.

Examiner: So what type of curve does this patient have?
Candidate: To define this curve accurately, I need to measure the Cobb angle of the curves in the frontal and sagittal as well as bending films.

Examiner: The thoracolumbar curve Cobb angle is 65°, the thoracic curve is 54°, the thoracic T5–T12 kyphosis is 37° and the thoracolumbar kyphosis (T10–L2) is 24°. In side bending films, the thoracic curve corrects to 46° and the thoracolumbar curve to 29°. How would you classify this deformity?
Candidate:

1. We have here two curves, a thoracic curve and a thoracolumbar curve. Both of them are structural and the thoracolumbar curve is the major curve, therefore it is Type 6 (thoracolumbar/lumbar – main thoracic).
2. If we draw the CSL, it would run outside the pedicles of the lumbar vertebrae, therefore the lumbar spine modifier is C.
3. Then, the thoracic spine sagittal modifier will be N because T5–T12 kyphosis is within the normal range (10°–40°).
4. The final classification of this curve would be Lenke type 6CN.

Examiner: How would you manage this patient's spinal deformity?
Candidate: The main lines of treatment of scoliosis deformity are observation, bracing and surgical treatment. Usually, the threshold for surgical treatment would be a thoracic curve >50° or a lumbar curve >40°. So this patient is already within the surgical indication and in such cases, we would do an instrumented posterior spinal fusion for the structural curves, which are the thoracic and thoracolumbar curves. Regarding the exact fusion levels, it is always a controversial subject but many surgeons would include both the upper and lower end vertebrae of the structural curves.

Section 2 Core structured topics

Chapter 6

The spine

Ashley A. Cole and Lee M. Breakwell

Spinal deformity

Spinal deformities can be divided into scoliosis and kyphosis, which affect either the whole spine or a region thereof, or spondylolisthesis, which usually affects a localized section of the spinal column.

Scoliosis

Scoliosis is defined as a frontal or coronal plane curvature with a Cobb angle of greater than 10°. The Cobb angle is defined as the maximal angle subtended by the endplates of the vertebrae within the curve.

Classification

1. Congenital,
2. Idiopathic,
3. Syndromic,
4. Neuromuscular,
5. Degenerative,
6. Paralytic.

Congenital

Congenital scoliosis is due to a developmental defect in the formation of the mesenchymal anlage. The resulting abnormal vertebra conveys uneven growth, creating angulation of the endplates and leading to unbalanced growth in adjacent vertebrae (Figure 6.1).

The defects are due to:

- Failure of formation: the commonest abnormality in this group is a hemivertebra, which may be fully, partially or unsegmented.
- Failure of segmentation: this results in block vertebrae, and unsegmented bars.
- A combination of these.

Associations

Once a diagnosis of congenital scoliosis has been made, the following associated anomalies must be sought and excluded:

- Spinal abnormalities (21–37%, MRI):
  - Hairy patch,
  - Dysraphism,
  - Myelomeningocele,
  - Diastematomyelia.
- Cardiac anomalies (12–26%, echo),
- Renal anomalies (20%, renal ultrasound),
- VACTERL association.

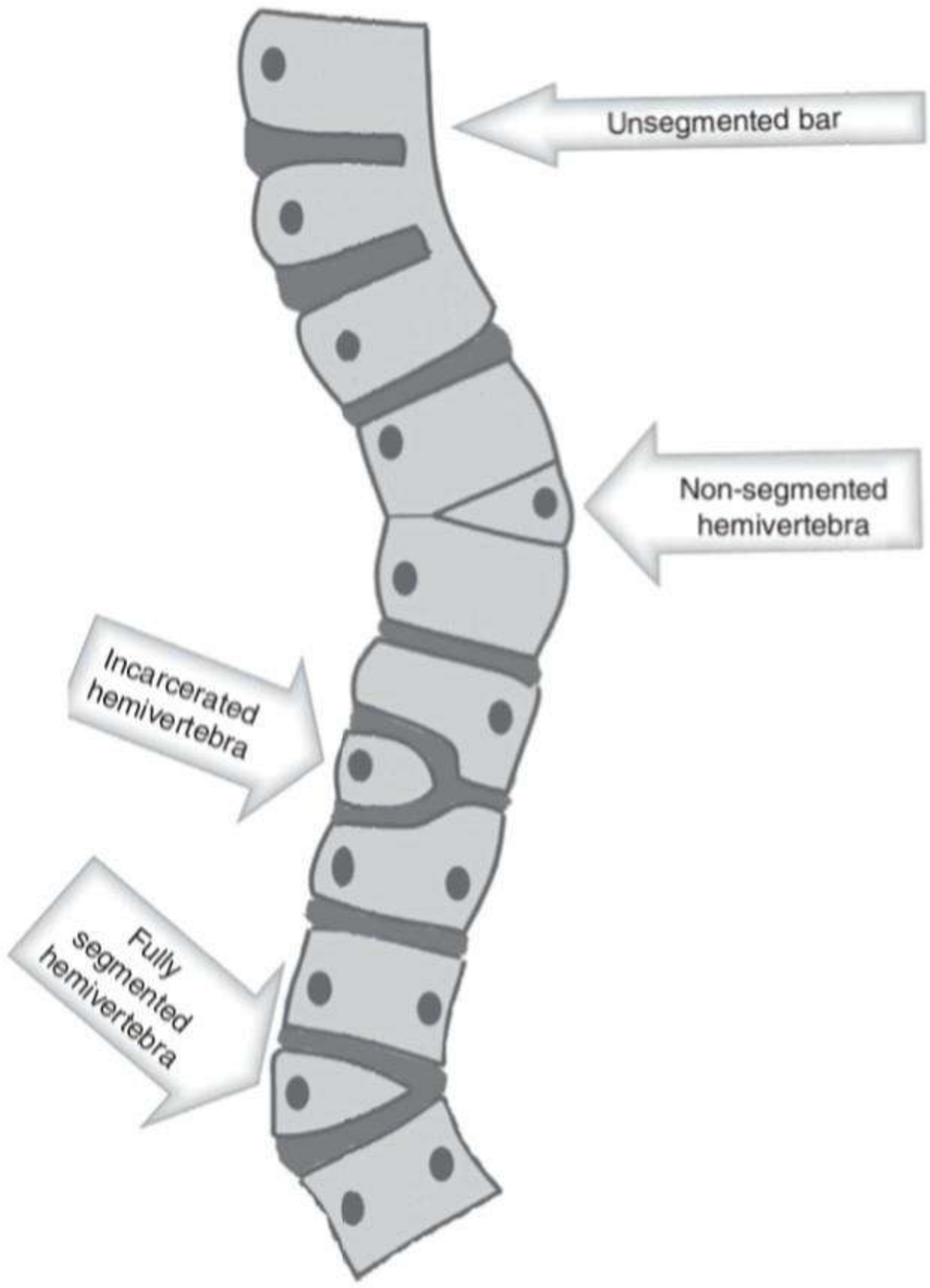


Figure 6.1 Congenital scoliosis.

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

Chapter 6: The spine

Risk of progression

Several factors will help to indicate the risk of progression and hence the indications for treatment:

1. Age of patient (remaining growth),
2. Site of the anomaly (worse at junctional regions, such as thoracolumbar and lumbosacral),
3. Type of anomaly (from worst to best):
   i. Unilateral unsegmented bar with contralateral fully segmented hemivertebra,
   ii. Unilateral unsegmented bar,
   iii. Fully segmented hemivertebra,
   iv. Partially segmented hemivertebra,
   v. Incarcerated hemivertebra,
   vi. Non-segmented hemivertebra.
4. Size of curve at presentation.

Idiopathic

Idiopathic scoliosis is by far the commonest form of scoliosis and affects approximately 3% of girls. Although the aetiology is unknown, there is growing evidence of a genetic causation.

Right thoracic curves are the commonest, followed by double major (right thoracic and left lumbar) then left lumbar.

Idiopathic scoliosis is subdivided into three forms based on the age of onset:

1. Infantile (0–3 years),
2. Juvenile (4–10 years),
3. Adolescent (>10 years).

Infantile

This represents less than 1% of all idiopathic curves, is commonly seen in boys and is usually a left thoracic curve.

It is the only true scoliosis that can resolve spontaneously: this can be predicted by measuring the rib–vertebra angle difference (RVAD) of Mehta on an AP radiograph.

This is derived by taking the angle of the concave and convex ribs to the apical vertebra bisector, and subtracting the concave from the convex angles (Figure 6.2).

An angle difference of greater or less than 20° implies a significant chance of progression or resolution respectively; 83% of the curves that resolved had an initial RVAD measuring less than 20°, whereas 84% of the curves that progressively worsened had an RVAD exceeding 20° [1].

Juvenile

Juvenile curves have a relatively high risk of progression due to the remaining growth potential. Approximately 70% will progress and many will require treatment. As many as 1 in 10 juvenile scoliosis patients are revealed to have a neural axis abnormality by MRI.

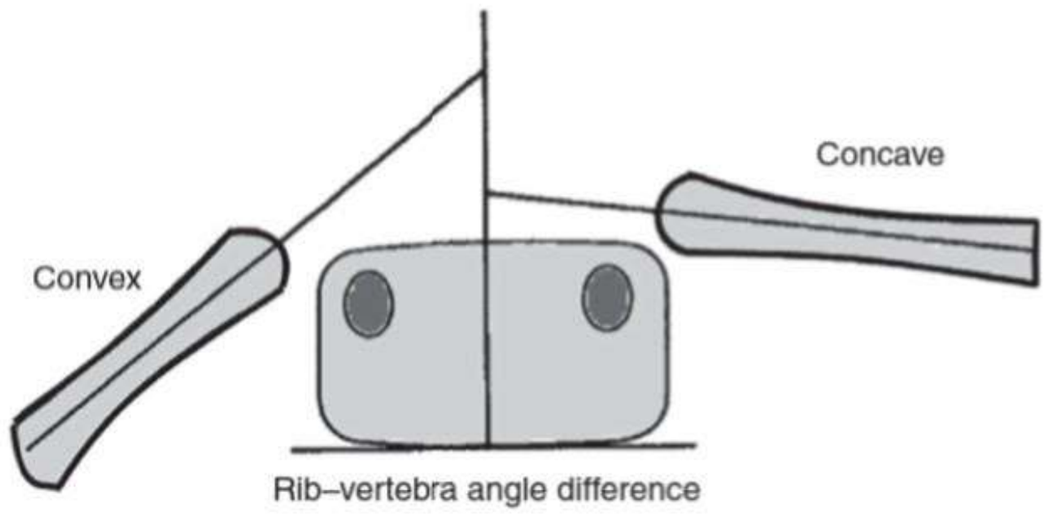


Figure 6.2 Congenital scoliosis, showing rib–vertebra angle difference.

Adolescent

This is the most common form of idiopathic scoliosis; it is commonly seen in girls.

Factors that aid in identifying risk of progression:

1. Curve size (>20°),
2. Remaining growth (curves worsen with growth). This is usually assessed:
   i. Clinically (menarche and peak height velocity (PHV)),
   ii. Radiological triradiate cartilage closure and Risser's stages (Figure 6.3).
3. Curve type (double curve and thoracic curve, thoracolumbar then lumbar).

In clinical practice, the PHV (i.e. growth spurt) is documented by serial measurement of the patient's height over time. The average age of the PHV is approximately 11.5 years in girls. Triradiate cartilage closure, a radiographic index of maturity, occurs after PHV and before Risser grade 1 and menarche.

Factors of no predictive value for curve progression before skeletal maturity include:

1. A family history of scoliosis,
2. Patient height-to-weight ratio,
3. Lumbosacral transitional anomalies,
4. Thoracic kyphosis,
5. Lumbar lordosis,
6. Spinal balance.

Syndromic

Scoliosis is a common feature of many well-known syndromes. In addition, it is present in many rare syndromes that are regularly seen in spinal clinics. Common syndromes with scoliosis include:

- Neurofibromatosis,
- Marfan syndrome,
- Ehlers–Danlos syndrome.

Rarer syndromes with scoliosis include:

- Rett,
- Sotos,
- Prader–Willi.

Section 2: Core structured topics

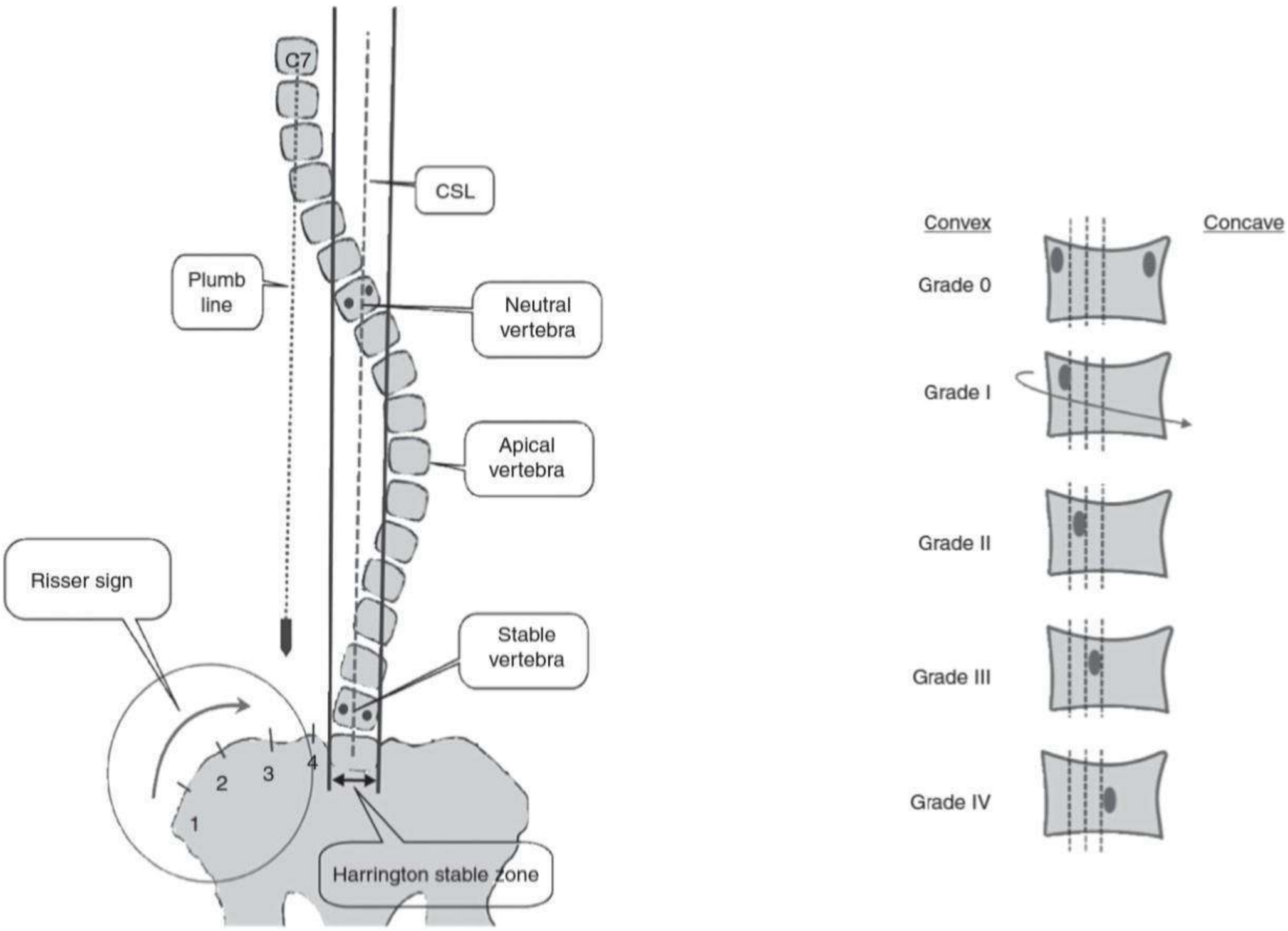


Figure 6.3 Scoliosis radiology. Left: Diagram showing various radiological terms of scoliosis. Right: Nash–Moe method for assessing vertebral rotation. Grade 0: Both pedicles are symmetric, Grade I: the convex pedicle has moved away from the side of the vertebral body. Grade III: the convex pedicle is in the centre of the vertebral body. Grade II: the rotation is between Grades I and III and Grade IV when the convex pedicle has moved past the midline. The curved arrow denotes the direction of the rotation. CSL, central sacral line.

The majority of these patients already have a diagnosis prior to identification of the scoliosis; however, in some patients, the spinal deformity is the index problem.

Neurofibromatosis scoliosis is the most common skeletal manifestation of neurofibromatosis.

The cause is unknown but various theories have been proposed, such as primary mesodermal dysplasia, erosion or infiltration of the bone by localized neurofibromatosis tumours, and endocrine disturbances.

Neurofibromatosis scoliosis can be either:

- Non-dystrophic,
- Dystrophic.

Differentiation between the two types is important because the prognosis and management differ significantly.

Dystrophic scoliosis is more common (Figure 6.4), usually located in the thoracic region, and has a short (4–6 vertebrae), sharply angled curve. It has a greater tendency to progress, and is at risk of developing neurologic deficits. Non-dystrophic scoliosis more closely resembles idiopathic scoliosis in both curve patterns and behaviour.

Neuromuscular

These curves are usually long, less likely to have compensatory curves and may progress after maturity. Pulmonary problems, such as decreased lung function, are observed.

This form of scoliosis is divided into two subgroups:

- Cerebral palsy,
- True neuromuscular diseases, in which there is primary nerve or muscle disorder, e.g. Duchenne muscular dystrophy.

Cerebral palsy

The incidence of spinal deformity increases with the severity of cerebral palsy. It is around 20%; however, in the quadriplegic group, the incidence is in excess of 70%.

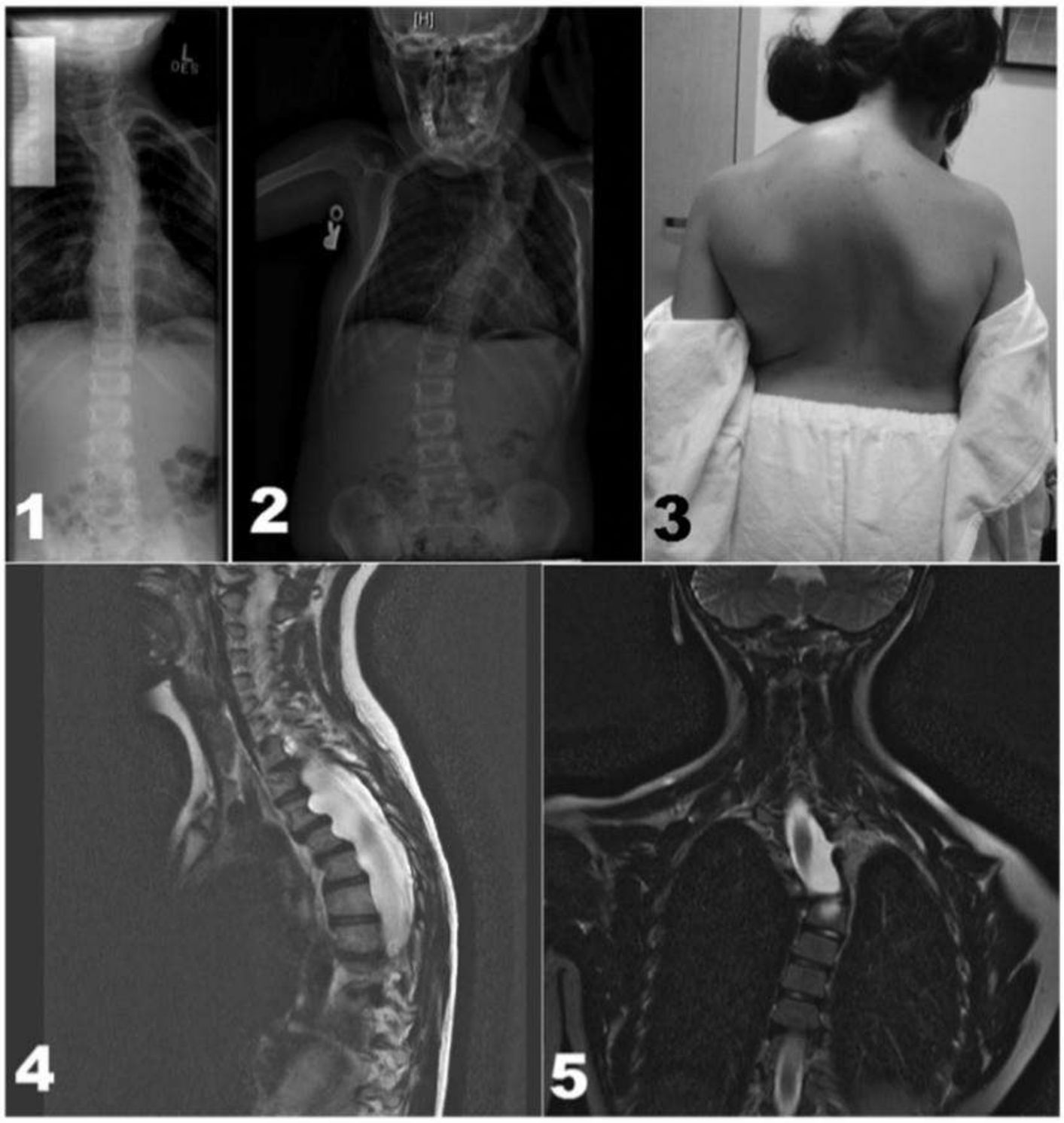


Figure 6.4. Dystrophic scoliosis in a child with neurofibromatosis scoliosis. 1, 2: There was a rapid progression in the severity over 2 years. 3: Café au lait spots and a sharply angled kyphoscoliosis. The kyphosis is more pronounced than the scoliosis. 4, 5: MRI scan shows large dural ectasia and scalloping of the posterior vertebrae. 5: There is a large plexiform neurofibroma in continuity with a nerve root.

The ability to walk is relatively protective, and therefore wheelchair dependence is a risk factor for development and progression of a spinal curvature.

Duchenne muscular dystrophy

This X-linked recessive condition, affecting the production of dystrophin, almost exclusively affects boys, with their mothers as the carrier. Historically, wheelchair dependence happened at about 10 to 11 years of age, following which the patient developed a scoliosis. Curves beyond 20° were seen to progress inexorably and surgery was advised at an early stage, to prevent further respiratory embarrassment.

Since the advent of steroid treatment, the progression of weakness has significantly slowed, and many subjects continue with some form of ambulation into their teens. This, coupled with the fact that treated curves do not always progress, means that the indication for surgery has changed, and not all patients require surgery.

The decision to undertake corrective surgery can now often be left until the curve has progressed to 40° or more, as the child is often older, and the lung function better than the historical cohort.

Spinal muscular atrophy

This autosomal recessive muscular wasting disease commonly causes scoliosis, which is often progressive. A defect in the *SMN1* gene leads to loss of the SMN protein, which is vital for muscle function. There are three types of spinal muscular atrophy in children, with function and life expectancy increasing from Type I through to Type III. Respiratory function can be severely restricted; hence, scoliosis is a major concern. The median survival in Type I spinal muscular atrophy is 7 months, with a mortality rate of 95% by the age of 18 months. In Type II spinal muscular atrophy, the age of onset is between 6 and 18 months and the age of death varies. The decision to undertake surgery for spinal deformity must be made in close collaboration with respiratory physicians, ideally considering life expectancy and function, as well as risk of curve progression.

Degenerative

This form of curve typically develops in the fifth or later decade of life, often in a previously normal spine; hence, it is beyond the scope of this book.

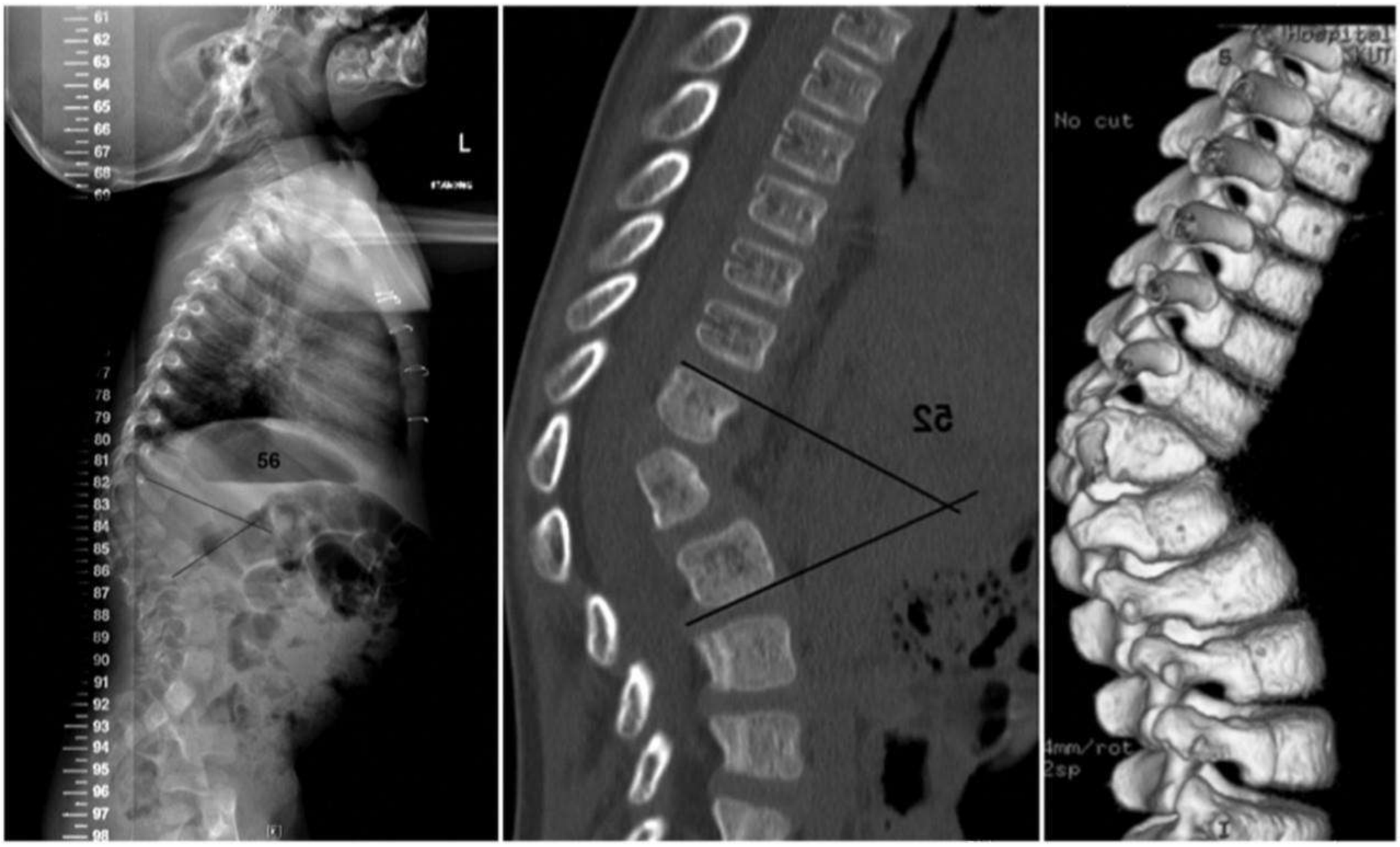


Figure 6.5 Congenital kyphosis.

Paralytic

Spinal cord injury with resultant paralysis before the onset of the adolescent growth spurt leads to the development of scoliosis in 97% of patients [2].

Kyphosis

Kyphosis is a forward curvature of the spine in the sagittal plane. A certain degree of thoracic kyphosis (20°–50°) is normal and desirable for spinal balance. Thoracic kyphosis does not strictly have a normal value, as it exhibits a range throughout different body shapes.

Classification

1. Congenital,
2. Idiopathic,
3. Neuromuscular,
4. Syndromic,
5. Traumatic,
6. Degenerative.

Congenital

As in scoliosis, this deformity develops due to an underlying structural disorder. The same basic types exist as in the coronal plane scenario.

A hemivertebra positioned posteriorly (Figure 6.5) will gradually deform the spine in a kyphotic direction. This causes a localized angular, deformity called a gibbus.

As the angulation progresses with growth, the centre of gravity of the body moves forward, increasing the load on the anterior aspect of the vertebral ring apophysis. This impedes anterior growth, in compliance with the Hueter–Volkmann law, unbalancing in favour of posterior height increase and leading to worsening of the kyphosis. By this process, kyphosis progresses throughout growth, and, if beyond 90°, continues into adulthood.

Whereas scoliotic deformity causes neurological deficit extremely rarely, congenital kyphosis has a relatively high risk of curve progression and neurological deficit when the angle is localized and beyond 90°.

Idiopathic

Scheuermann's kyphosis is seen in children older than 10 years and is more common in boys. The incidence ranges from 1–8%.

The accepted pathoaetiology is that slight kyphosis in the growing spine causes an anterior shift in the body weight centre, unevenly loading the anterior apophysis. This then leads to fragmentation and poor growth, as seen in the anterior vertebral body. It is characterized by vertebral wedging, disc space narrowing, endplate irregularities, including Schmorl's nodes, and kyphosis (Figure 6.6).

Figure 19.10 Child with bilateral foot pain.

Chapter 5

Spine structured oral questions

Alexander D. L. Baker

Introduction

What could be more central to orthopaedics than the 'orthos' (correct or straight) and 'paideion' (child) of paediatric spinal deformity surgery? Despite this, the spine viva is often an area where candidates for the FRCS (Tr & Orth) exam feel less well prepared. The subject area of orthopaedic spine surgery is broad and rapidly evolving. This makes it a fascinating area to study, but it also presents candidates for the FRCS exam with a daunting task if an exhaustive knowledge is sought. Viva questions tend to be one of two types. Either they are sufficiently 'core' that any consultant orthopaedic surgeon should know about the condition, or they are general orthopaedic questions that are being applied to the spine. In order to cover the breadth of material required this chapter will be succinct, covering core spine topics in sufficient depth to provide the candidate with a framework with which to tackle spine questions. Areas that will be covered include:

1. Tumours.
2. Infection.
3. The prolapsed intervertebral disc.
4. Scoliosis.
5. Spinal stenosis.
6. Spondylolisthesis.
7. Trauma.
8. Notes on various other viva scenarios.

Structured oral examination question 1: Spinal tumour

CANDIDATE: The images show a destructive lesion in the vertebrae which given the age (> 50) is most likely to be metastatic tumour. Breast, lung, prostate, renal, thyroid and GI malignancies are the most common sources of primary disease. (Figure 5.1.)

EXAMINER: How would you go about investigating this?

CANDIDATE: Staging and grading. Initial assessment would include a detailed history and examination, paying particular attention to any history of malignancy and asking about symptoms of altered bowel habit, respiratory problems, any prostatic symptoms or breast lumps. Examination should include breast, thyroid, respiratory, abdominal and rectal examinations (with faecal occult blood tests).

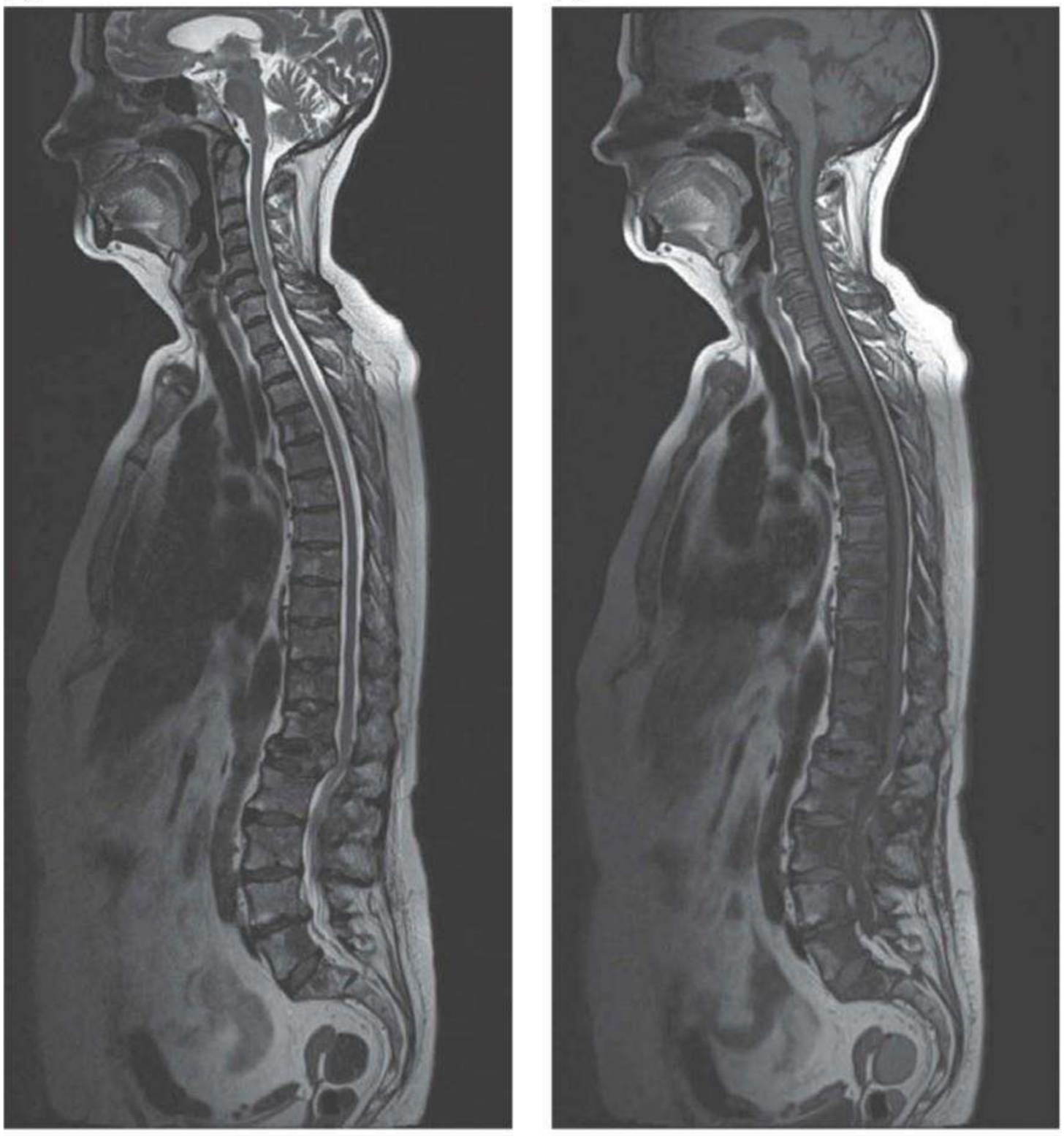
Investigations should include *local* and *distant* imaging. Local imaging should include plain radiographs and a whole spine MRI (looking at neural compression and the extent of spinal involvement). A CT may be required for detailed bony anatomy if resection is being considered. The distant imaging selected depends on the likely pathology. It might include a bone scan (looking for evidence of other skeletal metastases), a chest X-ray, or a CT chest, abdomen and pelvis to search for a primary tumour or visceral metastasis. Inflammatory markers should also be sent as well as tumour markers such as serum plasma electrophoresis or PSA.

Histological grading requires a biopsy. Following the general principles applicable to all musculoskeletal tumours this biopsy should be done within the unit that will treat the tumour and also samples sent for culture. 'Biopsy all infections and culture all tumours.'

EXAMINER: How would you decide about subsequent treatment?

CANDIDATE: The scoring system proposed by Tokuhashi is useful in establishing indications for treatment and subsequent surgical goal.[1] A poorer prognosis is correlated with a lower score. Six parameters are given a score from 0 to 2. A score of less than 5 indicates a life expectancy under 1 year and a palliative approach is suggested. A score of over 9 indicates a longer life expectancy and suggests resection/excision should be considered.

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



Figures 5.1a and 5.1b Sagittal T1(b) and T2(a) weighted MRI images.

General condition (Poor 0; Moderate 1; Good 2)
Number of extra-spinal metastases (3 or more scores 0; 1 or 2 scores 1; 0 scores 2).
Number of spinal bony metastases (3 or more scores 0; 2 scores 1; 1 scores 2).
Number of metastases to major internal organs (not removable 0; removable 1; no metastases 2).
Tissue of origin (lung, stomach 0; kidney, liver, uterus 1; other, breast, thyroid, prostate, rectum 2).
Spinal cord palsy (complete 0; incomplete 1; none 2).

Tumour background knowledge

Overall, metastatic disease is the most common cause of spinal involvement and primary tumours of the spine are rare. Curative resection is possible in a few cases, but palliative intervention is more common. Pain from bony destruction and resultant mechanical instability may respond well to surgical stabilization.

Decompressive surgery may prevent (or prevent progression of) neurological impairment.

Epidemiology

Vertebral body lesions are more likely to be malignant and posterior lesions benign. Under the age of 21 most spinal tumours are benign, over 21 most are malignant. Under the age of 3 metastatic malignant tumours become more common again. Breast, lung, prostate, renal, thyroid and GI malignancies are the most common sources of primary disease.

Surgical treatment

Surgery is increasingly being performed. Following surgery, patients can often expect functional improvement, pain relief, and in a few cases cure. NICE has issued guidelines on the treatment of metastatic cord compression.[2] Decompression of compressed neural structures may lead to functional improvement even with prolonged paraplegia.

Simple laminectomy to 'decompress' the tumour is rarely indicated as the presence of the tumour (most frequently found in the vertebral body) is likely to lead to mechanical instability and thus kyphosis. Instrumented stabilization is frequently undertaken.

Surgical resection of tumour is aimed at improving survival. Resection may be undertaken anteriorly, or posteriorly, or both, and depending on the size and location of the lesion. In general terms, if a curative resection is hoped for, or survival is likely to extend beyond 6 months, intervertebral bony fusion should be undertaken to avoid instrumentation failure. If life expectancy is short and a palliative procedure is being considered, fusion may not be required and posterior surgery is more commonly undertaken.

Radiotherapy

- Mainly used to reduce tumour bulk.
- Many GI and renal tumours are resistant but most breast tumours are sensitive.
- Prostate and lymphoreticular tumours respond best.
- There is an increased risk of wound problems with adjuvant radiotherapy (separate radiotherapy and surgery by a period of 6 weeks).

Minimally invasive surgery and cement vertebral body augmentation

These techniques are novel and their role is yet to be firmly established.

Some patients are too unwell or are unwilling to consider major surgery.

When pain caused by instability does not require decompression, vertebral body augmentation with high viscosity cement (PMMA) may be considered.

Minimally invasive surgery may allow the surgeon to stabilize the spine whilst minimizing soft tissue trauma facilitating a faster postoperative recovery in patients with limited life expectancy.

Specific tumours

Benign

Haemangioma – Slow growing and often asymptomatic. Often detected as an incidental finding on imaging.

Osteoid osteoma/osteoblastoma are usually found in the posterior neural arch. Most present with pain (NSAID sensitive). Excision is curative but NSAID may be all that is required.

Osteochondroma are most commonly found on the spinous process (related to the apophysis). Excision is for symptomatic treatment. Sarcomatous change has been described and excision is indicated if a large (> 10 mm) cartilage cap is seen on MRI.

Aneurysmal bone cysts typically affecting the posterior elements and giant cell tumours (affecting the vertebral body) are also seen.

Malignant

Myeloma/solitary plasmacytoma typically presents with pain and can be treated with radiotherapy (highly sensitive), or cement augmentation.

Chordoma is locally aggressive and may present with compression of pelvic contents.

Lymphoma most commonly occurs in the elderly (mean age 85) and more frequently in men than women.

Chondrosarcoma typically presents with pain and X-rays may show typical matrix calcification.

Osteosarcoma presents in the young (< 20). It is rare and survival is poor (median survival 6–10 months).

Intradural tumours

In contrast to extradural tumours most intradural tumours are not metastatic.

Extramedullary tumours occur inside the dura but outside the spinal cord. They are usually benign. They cause symptoms by compressing neural structures which can lead to pain or loss of motor function. Examples include neurofibromas, schwannoma (of dorsal sensory roots) and meningioma.

Intramedullary tumours occur within the spinal cord. Most are malignant. Examples include astrocytomas (affecting children), ependymomas (affecting adults), and rarely haemangiomas.

1. Tokuhashi Y, Matsuzaki H, Toriyama S, Kawano H, Ohsaka S. Scoring system for the preoperative evaluation of metastatic spine tumor prognosis. *Spine* 1990;**15**(11):1110–1113.
2. NICE Clinical Guideline 75. *Metastatic Spinal Cord Compression*. November 2008.

Structured oral examination question 2:

Infection (epidural abscess)

EXAMINER: A 68-year-old man with a past history of a lung tumour 10 years ago presents following a fall with a 4-week history of worsening thoracic back pain. Back pain is a

common presenting complaint to general practitioners and orthopaedic departments. What red flags are there to indicate possible underlying pathology?

CANDIDATE: In this individual, age, the past history of tumour, the thoracic location of his pain, and the history of trauma are all 'red flags'. Other possibilities include: fever, weight loss, night sweats, night pain, non-mechanical pain, severe intractable pain, thoracic pain, age over 55 or below 20, a history of carcinoma, steroid use, IV drug abuse, saddle anaesthesia, urinary or bowel symptoms, deformity.

EXAMINER: Here is his MRI scan. What can you see? (Figure 5.2.)

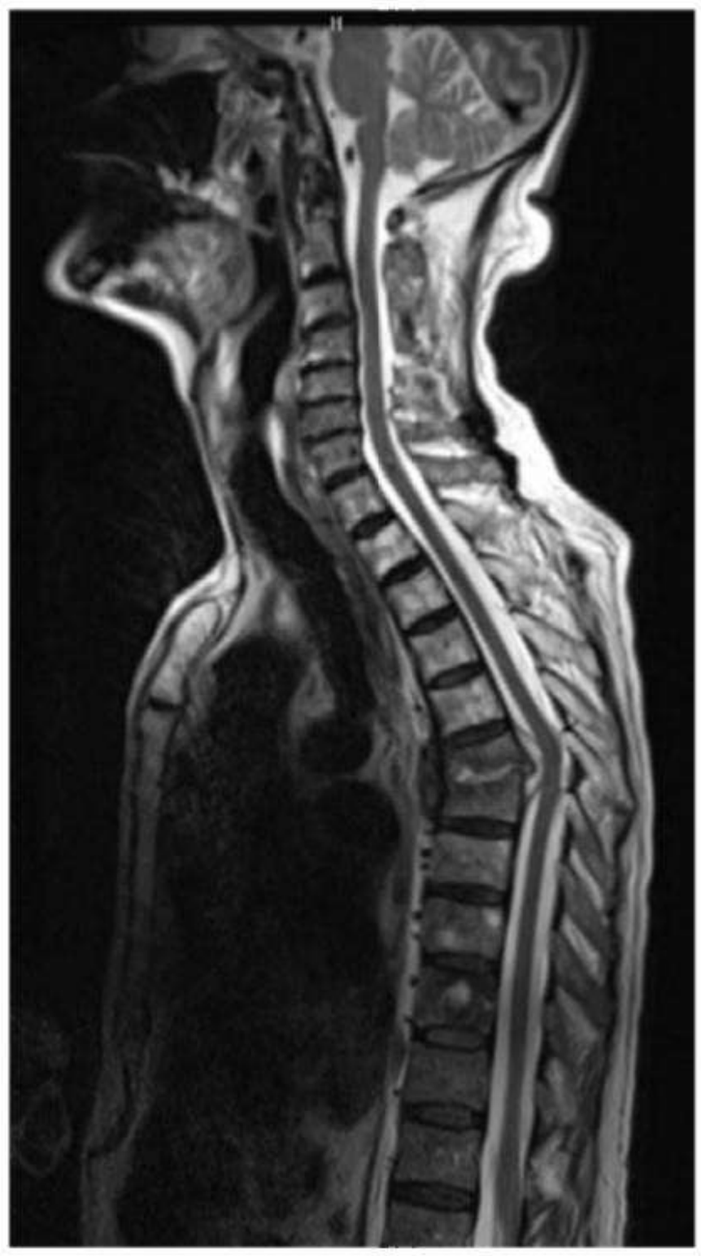


Figure 5.2 T2-weighted sagittal MRI image epidural abscess.

CANDIDATE: This is a sequence of MRI scans, both T1- and T2-weighted MRI scans. There is a lesion in the thoracic spine, which appears to be compressing the spinal cord. The fact that the lesion is bright on the T2 scan implies that this is likely to be fluid filled and suggests an infective aetiology.

EXAMINER: How would you proceed?

CANDIDATE: We are aware of the history of a fall and should establish this man's neurological status. I would start by obtaining a history and detailed neurological examination. His temperature, routine blood tests (WCC) and inflammatory markers (CRP, ESR) will help confirm the diagnosis of infection.

The most likely diagnosis is an epidural abscess with signs of neurological compression. I would therefore proceed to urgent surgical decompression of the abscess. I would not start antibiotics before obtaining a sample for microbiology and I would also send tissue to pathology (history of tumour).

Infection background knowledge

Spinal infection still remain a serious, potentially life-threatening problem.

Diagnosis is often delayed.

MRI is the imaging modality of choice.

The vertebral body (osteomyelitis), the intervertebral disc (discitis), or the epidural space (epidural abscess) may be affected.

In the absence of a localized collection and no neurology, initial treatment is conservative and should be treated in a similar way to osteomyelitis. (High-dose intravenous antibiotics for 6 weeks or until CRP normalizes and then oral antibiotics until there are no signs of infection.) Consider radical debridement in persistent infections.

Discitis is more common in younger children and vertebral osteomyelitis more common in adults. The intervertebral disc is vascular in younger children.

In the neonate intraosseous, vertebral arteries anastomose with the adjoining disc through the vertebral end plate. With increasing age the disc loses its vascularity.

Risk factors for infection include intravenous drug use, diabetes, steroid use, chronic infection and other immunocompromised states. Most infections are caused by *Staphylococcus aureus* or *Streptococcus*.

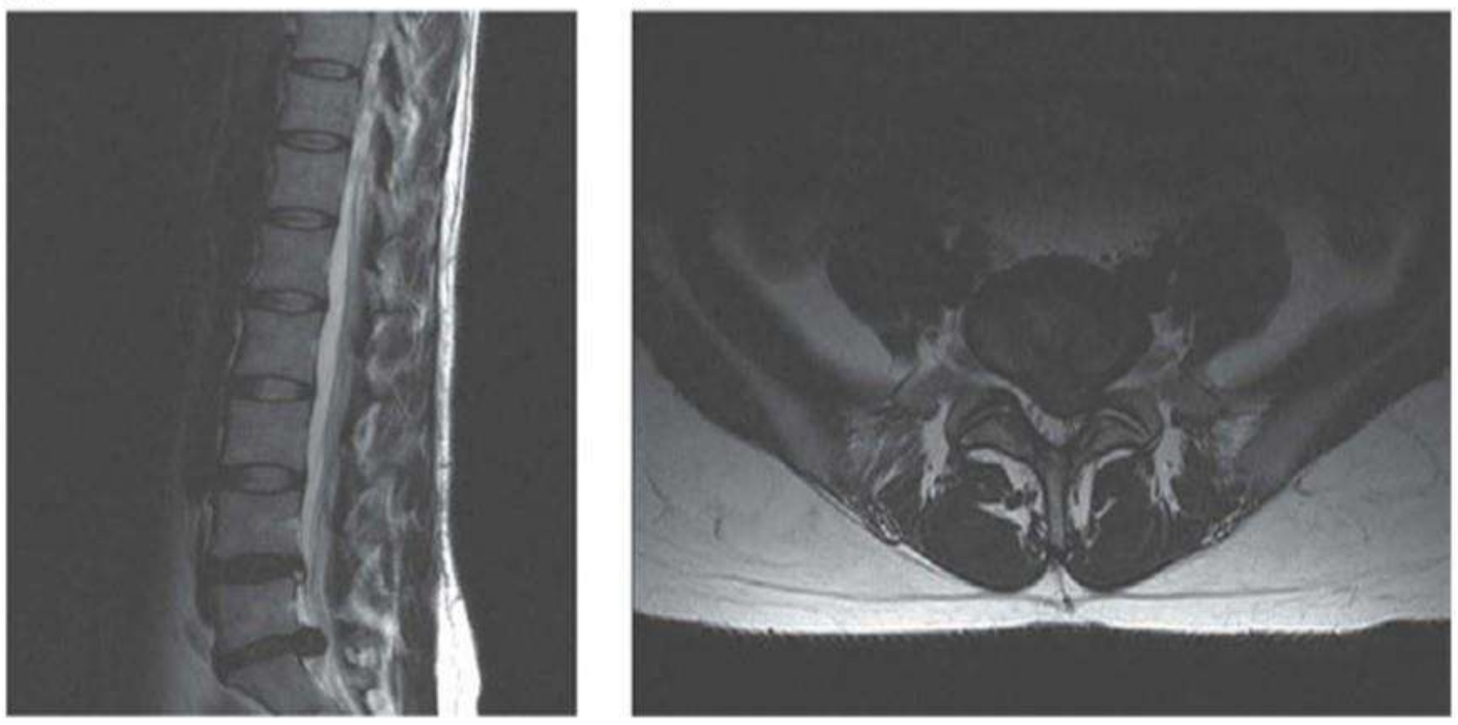
Consider decompressing an abscess in the presence of neurology and/or a localized collection.

Consider radiologically guided decompression.

Structured oral examination question 3: The prolapsed intervertebral disc

EXAMINER: A 37-year-old man has been referred to your clinic with back and left lower limb pain. The general practitioner suspects a 'slipped disc'. What features in the history and on examination will you be looking for?

CANDIDATE: Dermatomal limb pain that predominates over back pain, described as burning in nature, associated with paraesthesia and numbness. Examination should reveal



Figures 5.3a and 5.3b T2-weighted axial and sagittal MRI images showing paracentral disc L5/S1 prolapse.

positive nerve root tension signs, altered sensation in the affected dermatome and a decreased ankle jerk reflex on that side. Also, I would like to rule out serious spinal pathology or signs of a cauda equina syndrome.

EXAMINER: You request an MRI scan, here it is, what can you see? (Figure 5.3.)

CANDIDATE: This is a T2-weighted MRI scan showing the lumbar spine in coronal and sagittal section. There is a paracentral disc prolapse at the L5/S1 level.

EXAMINER: What would you expect to find in this patient?

CANDIDATE: I would expect the pain, paraesthesia and numbness to be in an S1 distribution (posterior calf, heel and lateral border of the foot) on the left. There may be an associated subjective decreased sensation in the same distribution, a decreased ankle jerk on that side, decreased straight leg raise and positive cross-over sign.

EXAMINER: How would you treat this patient?

CANDIDATE: Initially conservatively as the natural history of most lumbar disc prolapses is that they resolve with time. If it has not resolved after 6–12 weeks of conservative management I would offer the patient microdiscectomy.

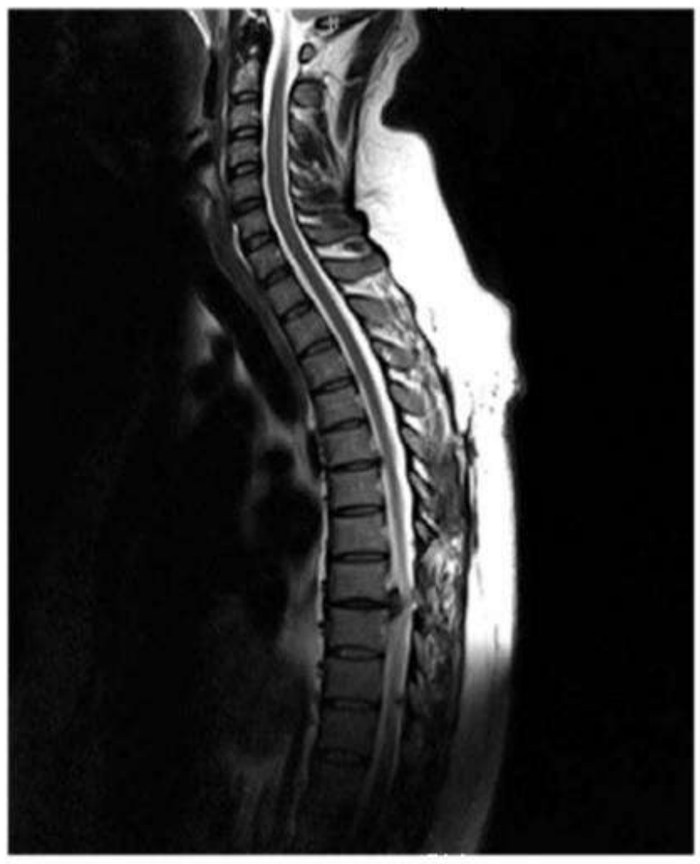


Figure 5.4 T2-weighted MRI showing a thoracic disc prolapse.

A thoracic disc prolapse (rare) will typically present with symptoms and signs of spinal cord compression associated with thoracic back pain (Figure 5.4). The discs are usually calcified and require decompression from the front. Treatment therefore is via a thoracotomy and partial vertebrectomy.

A cervical disc prolapse may present with symptoms and signs of a cervical radiculopathy or cervical myelopathy.

Disc prolapse background knowledge

The clinical features and treatment options for disc prolapse vary depending on age and the location of the prolapsed disc.

In children the symptoms and signs of disc prolapse are less well defined and back pain is a more prominent feature. Nerve root tension signs are also less likely to be positive and spontaneous resolution is less likely.

Cauda equina syndrome

Cauda equina syndrome caused by compression of the cauda equina (usually by a large acute disc prolapse) is characterized by some or all of the following:

Urinary retention.
Faecal incontinence.
Saddle area numbness and loss of anal tone.
Widespread neurological signs.

The importance of detecting cauda equina syndrome early is that early intervention (< 24 hours) has been shown to improve outcome. More recently the extent of the compression has also been linked to outcome and the importance of timing questioned.[1]

Exiting nerve roots in the cervical and lumbar spine

The knowledge that the L4 nerve root exits the spinal canal below the L4 pedicle may (incorrectly) lead the candidate to expect the L4 nerve root to be compressed when a disc prolapse occurs below the L4 vertebra in the L4/5 interspace. It is best to think of this nerve root as 'already having left the canal' and therefore it is the L5 'traversing' nerve root that is most commonly compressed by the common 'paracentral' disc prolapse. (It is true to say that a 'far lateral' disc prolapse may compress the exiting nerve root in the exit foramen but this is rare.) Thus an L4/5 disc prolapse commonly affects the L5 nerve root.

In the cervical spine, a prolapsed disc typically affects the exiting nerve root at that level (there is no traversing nerve root because the roots leave the spinal cord and exit the canal almost horizontally). But there is a nomenclature change in the cervical spine. Because the C6 nerve root exits above (not below) the C6 vertebra this double change means a prolapsed cervical disc at the C5/C6 level most commonly affects the C6 nerve root.

Nomenclature

A herniated disc is a localized displacement of nucleus pulposus beyond the normal limits of the disc. This can be broad-based (involves between 20% and 50% of the disc circumference), focal (involves < 25%) or symmetrical (involves 50–100% of the circumference of the disc).

A focal disc herniation may be described as a protrusion or extrusion. An extruded disc has a narrow 'neck' at its base. Extruded disc material is sequestrated if it is no longer in continuity with the disc.[2]

1. Sell P, Qureshi A. Cauda equina syndrome treated by surgical decompression: the influence of timing on surgical outcome. *Eur Spine J* 2007;**16**:2143–2151.
2. Fardon D, Milette P. Nomeclature and classification of lumbar disc pathology. Recommendations of the Combined Task Forces of the North American Spine Society, American Society of Radiology, and American Society of Neurology. *Spine* 2001;**26**(5):E930E113.

Structured oral examination question 4: Scoliosis

EXAMINER: What can you see? (Figure 5.5.)

CANDIDATE: This is an AP radiograph showing the spine, ribs and iliac crests. There is a left-sided, lumbar scoliosis.

EXAMINER: What different types of scoliosis do you know? What type of scoliosis is this?

CANDIDATE: The radiograph shows a congenital scoliosis. There is a hemivertebra within the lumbar spine producing the scoliosis.

Scoliosis occurs in different groups of patients and can be classified according to aetiology. Scoliosis may be idiopathic, congenital, neuromuscular or associated with other conditions such as Marfan's syndrome or neurofibromatosis.

EXAMINER: Why might you treat a scoliosis?

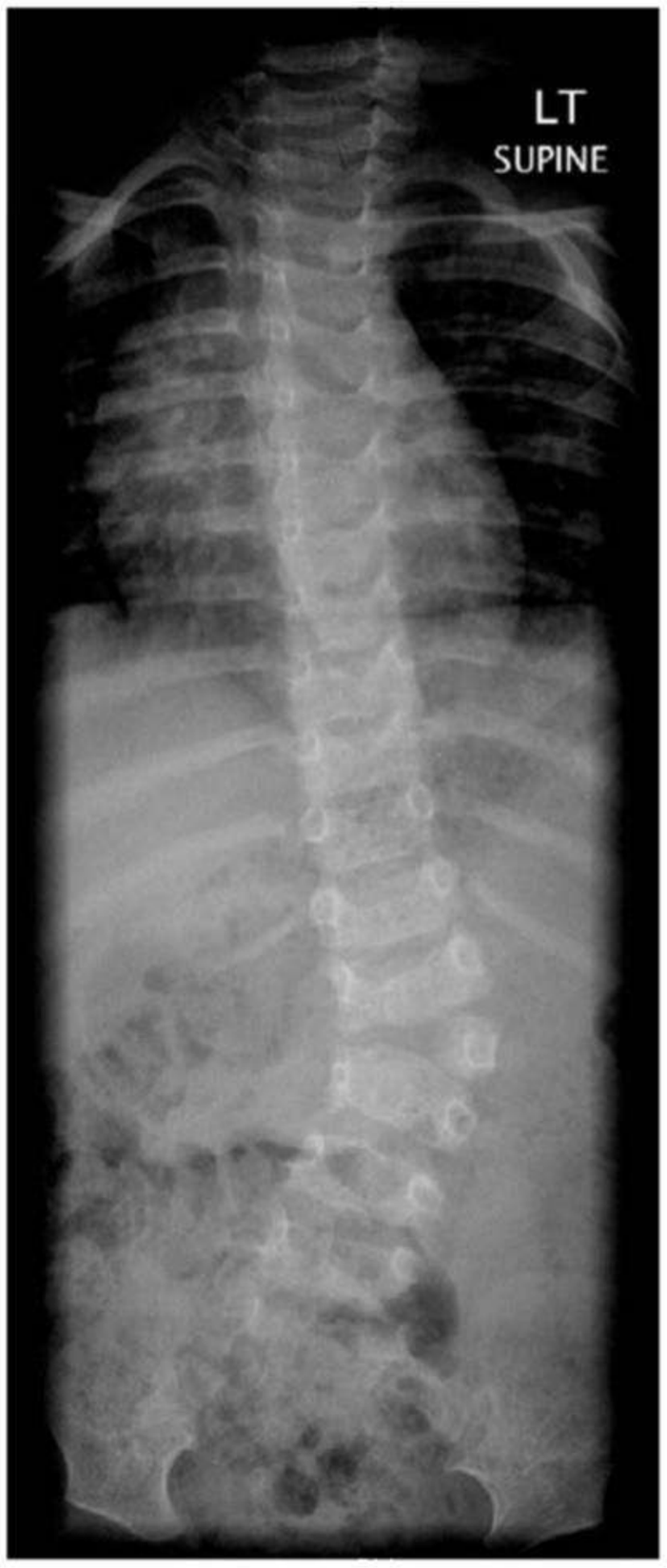


Figure 5.5 Anteroposterior (AP) radiograph of the thoracic and lumbar spine demonstrating a left-sided lumbar scoliosis.

CANDIDATE: The primary indication for treating a scoliosis is progressive deformity. Additionally, patients with 'early-onset' scoliosis and some types of neuromuscular scoliosis are at risk of progressive cardiorespiratory compromise as the curve deteriorates.

EXAMINER: How might you treat this scoliosis?

CANDIDATE: I would refer this patient to a specialist centre for treatment. I suspect that, if after monitoring the curve, it shows signs of progression they might consider excising the hemivertebra.

EXAMINER: The vertebra could be excised using an anterior, posterior or combined approach. Can you describe the thoraco-abdominal (Hodgson's) approach that might be used to approach this vertebra?

CANDIDATE: The patient is positioned in the lateral position with the limbs and trunk supported. The table is 'broken' with apex at the thoracolumbar junction. A skin incision is made over the 10th rib and curved distally to run longitudinally along the lateral border of rectus abdominus. Skin and fat are incised, as are serratus anterior, external oblique and latissimus dorsi. The rib is removed subperiosteally. The parietal pleura is incised exposing the lung and diaphragm.

A key step in this procedure is splitting the costal cartilage to enter the retroperitoneum. Retroperitoneal fascia is swept away with swabs. The diaphragm is divided 2 cm from its origin down to the vertebrae using marking stitches. Segmental vessels are ligated and the discs above and below the vertebrae excised.

Scoliosis background knowledge

Scoliosis is defined as a lateral curvature of the spine in the coronal plane that measures more than 10° using the Cobb method. When present it usually forms part of a three-dimensional spinal deformity. It is sometimes described as a four-dimensional deformity (the fourth dimension being time, emphasizing the progressive nature of the condition).

The key to treating scoliosis is knowledge of the natural history of the condition in order to predict curves that are likely to deteriorate rapidly so that they can be detected and treated at an early stage. Scoliosis has been classified according to severity, location, aetiology and age of onset.

Severity – The Cobb angle also defines the magnitude of the curve with minor (small) curves measuring between 10° and 25°, moderate curves between 25° and 50° and severe (large) curves measuring over 50°.

Location – The 'side' of a scoliosis is the side of the patient to which the spine deviates away from the midline, it is the side of the convexity of the curve. A scoliosis is also described by the region of the spine that it affects. The Scoliosis Research Society have defined a 'thoracic' scoliosis as having its apex between T2 and the T11–T12 disc, a 'thoracolumbar' curve as having its apex between the T12 and L1 vertebrae and a lumbar scoliosis as having its apex between the L1–2 disc space and L4. The apex of the curve is located by the most laterally deviated vertebra. Curves can be single, double or triple 'major' curves depending on whether the curves above and below the main curve are flexible or structural (flexible curves reduce to less than 25° on lateral bending).

Aetiology – Scoliosis is classified according to its aetiology and pathogenesis.

- **Idiopathic** (the largest group – 70%). In this group the scoliosis is produced by an imbalance in the growth of the spine with the convexity of the curve growing at a faster rate than the concavity.
- **Congenital scoliosis**. In this group abnormalities of one or more vertebrae are present at birth. The subsequent growth of these abnormal vertebrae cause the scoliosis. (The name might be confusing as it is the vertebral abnormalities that are present at birth and the scoliosis develops later with growth.)
- **Neuromuscular** (cerebral palsy, Duchenne muscular dystrophy, spinal muscular atrophy). In this group the scoliosis is produced by a lack of support to the spine causing the spine to collapse to one side.
- **Miscellaneous conditions associated with scoliosis** (5% – e.g. Marfan syndrome, neurofibromatosis).

Age of onset – Idiopathic scoliosis has been classified by age into infantile (age 0–3 years), juvenile (3–10 years) and adolescent (10–maturity) idiopathic scoliosis. An alternative classification divides scoliosis into early-onset scoliosis (associated with a high risk of cardiorespiratory compromise as the developing heart and lungs may be affected) which has its onset before the age of 7 and late-onset scoliosis which has its onset after the age of 7. (The age of 7 is used by the AO group – others have suggested 5.)[1,2]

Congenital scoliosis

- Scoliosis develops as a result of the growth of vertebral anomalies present at birth.
- The vertebral anomalies may be part of the VATER or VACTERL associations.
- Multiple vertebral anomalies are often hereditary.
- Isolated anomalies are mostly sporadic.
- No single genetic or environmental cause has been identified.

Classification: Anomalies present can be failures of formation or segmentation. More common congenital vertebral anomalies include the unilateral unsegmented bar, the hemivertebra (either fully segmented, semi-segmented or incarcerated), wedge vertebra and block vertebra. A fully segmented hemivertebra is one that has growth plates cranial and caudal to it.

Progression of congenital curves depends on growth potential and whether that growth is balanced. Thus a fully segmented hemivertebra in connection with a contralateral unsegmented bar has the least balanced growth and the worst prognosis. A block vertebra on the other hand has benign prognosis rarely leading to a curve beyond 20°.

1. Aebi M, Arlet V, Webb J. *AO Spine Manual.* New York: Theime Publishing, 2007.
2. Dickson RA. Early-onset scoliosis. In Weinstein SL (Ed.), *The Paediatric Spine: Principles and Practice*. New York: Raven Press, 1994.
3. Tsirikos AI, Chang WN, Dabney KW *et al.* Comparison of parents' and caregivers' satisfaction after spinal fusion in children with cerebral palsy. *J Ped Orthop* 2004; **24**(1):54–58.

Structured oral examination question 5: Lumbar spinal stenosis and cervical myelopathy

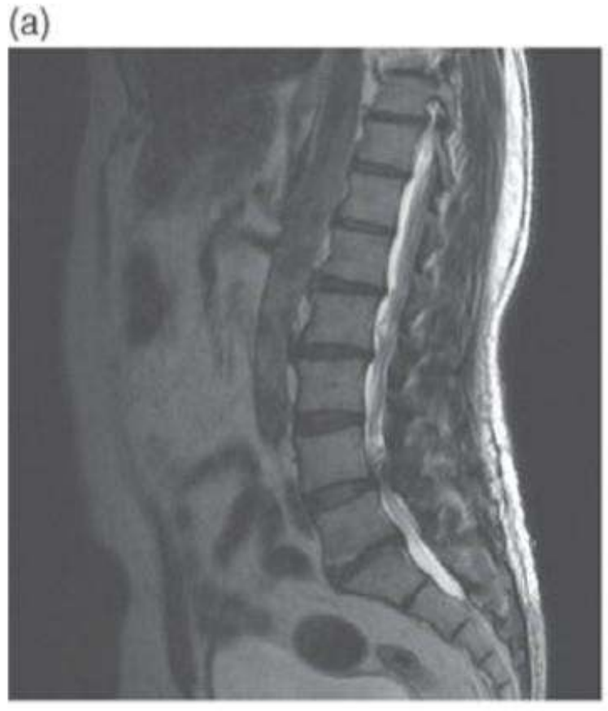
EXAMINER: A 70-year-old lady has been referred to your clinic having been seen by one of your arthroplasty colleagues. Her walking distance had reduced significantly but no abnormalities of her hips had been found and this MRI scan had been requested. Can you see anything that might cause this lady's symptoms? (Figure 5.6.)

CANDIDATE: Yes. The images are T2-weighted MRI scans showing the lumbar spine in axial and sagittal section. Both sagittal and axial scans show significant narrowing of the spinal canal, judging from the sagittal scan this appears to be at the L4/5 level.

EXAMINER: Yes, there is a very significant spinal stenosis at that level with obvious compression of the thecal sac surrounding the cauda equina and significant reduction of the CSF signal on the axial scan. How does this kind of stenosis arise and what neurological abnormalities are you like to find on examination?

CANDIDATE: Neurological examination of patients with lumbar spinal stenosis is often remarkably normal. The stenosis arises as a consequence of dehydration of the intervertebral disc leading to bulging of the disc, overload and hypertrophy of the facet joints, segmental instability and hypertrophy of the ligamentum flavum and osteophyte formation.

EXAMINER: Okay, so how do these patients typically present, and what will you be looking for on examination?



Figures 5.6a and 5.6b T2-weighted MRI axial and sagittal images of lumbar stenosis.

CANDIDATE: Patients with symptomatic lumbar spinal stenosis typically present with neurogenic claudication. Neurogenic claudication is a reduction in walking distance as a result of bilateral aching leg pain, a feeling of heaviness, fatigue, numbness and unsteadiness in the lower limbs. Symptoms are frequently reduced by rest and bending forward. Bending forward flexes the lumbar spine, reducing the lumbar lordosis, and increases the space available for the cauda equina within the spinal canal. Activities that involve flexion of the lumbar spine (e.g. walking uphill, upstairs, pushing a shopping trolley and cycling) are frequently found to be easier than less arduous tasks that extend the lumbar spine (increasing the lordosis).

The most common differential diagnosis is vascular claudication. Clinical examination with palpation of peripheral pulses as well as ankle–brachial pressure measurement is required. Standing relieves vascular claudication whereas neurogenic claudication may be made worse.

EXAMINER: Here is an MRI scan showing severe narrowing of the cervical spinal canal. Is this likely to present in the same way? (Figure 5.7.)

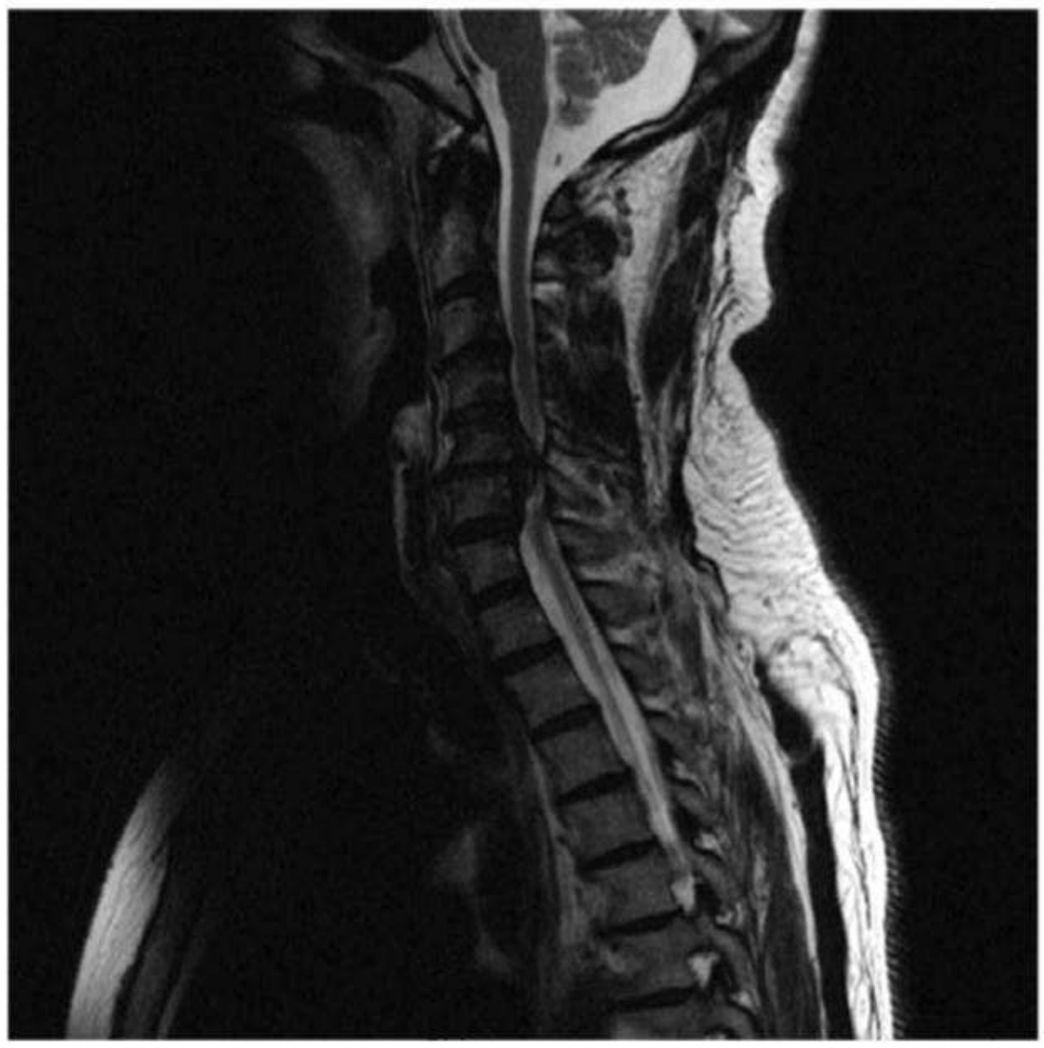


Figure 5.7 T2-weighted sagittal MRI image demonstrating cervical stenosis.

CANDIDATE: No, in this case we are at the level of the spinal cord rather than the cauda equina. There is a bulging cervical disc at the (most common) C5/6 level and the patient will present with symptoms of cervical myelopathy.

EXAMINER: What are the typical features of cervical spondylotic myelopathy and what would you expect to find on examination?

CANDIDATE: Cervical myelopathy presents with upper motor neurone signs and symptoms in both upper and lower limbs. Symptoms include decreased coordination, loss of fine dexterity (e.g. buttoning a shirt, handwriting, manipulating small objects), balance and gait problems, and problems with bowel and bladder function. Typically symptoms follow a slow, progressive course deteriorating in a stepwise manner with stable periods and periods of rapid deterioration. Balance and walking problems may lead to patients complaining of frequent trips, falls or bumping into things.

Associated (upper motor neurone) signs include: a wide based unsteady gait, upper and lower limb weakness, hyper-reflexia, intrinsic muscle waiting in the hand, positive Babinski and Hoffman signs and an inverted radial reflex.

Stenosis background knowledge

Lumbar spinal stenosis can occur within the spinal canal, the lateral recesses or the intervertebral (neural exit) foramen. Central stenosis may be asymptomatic or it may give rise to the symptoms of neurogenic claudication. Lateral recess stenosis or foraminal stenosis may lead to unilateral or dermatomal symptoms. Stenosis is frequently associated with a degenerative spondylolisthesis (which will be discussed in more detail in the next section).

Distinguishing between neurogenic claudication and vascular claudication:

	Type of claudication	
Symptom	**Neurogenic**	**Vascular**
Pain	Worse on standing	Relieved by standing
Numbness	Present	Absent
Site of pain	Buttock/thigh	Calf (rarely anterior)
Relieving factors	Bending forward	Standing
Walking distance	Reduced and variable	Reduced and fixed
Worse going	Downstairs	Upstairs

Hoffman's sign – Flicking the distal phalanx of the middle finger produces reflex contraction of thumb and index finger.

Babinski's sign – Extension of the toes on scraping/firmly stroking the sole of the foot.

Anterior approach to the cervical spine

Many right-handed surgeons prefer the right-sided approach. The left-sided approach has been reported as having a lower rate of recurrent laryngeal nerve injuries. Consider using a foot rest and tapes over the shoulders (acromion) to allow as much of the cervical spine to be exposed as possible for lateral imaging. The head is positioned on a horseshoe ring and neck is in slight extension (towel roll between shoulders). The skin crease incision is made in line with the following landmarks.

C3/4 – Hyoid bone
C4/5 – Laryngeal prominence
C5 – Thyroid cartilage
C6 – Cricoid cartilage

Platysma is incised in line with the skin incision and the fascia dissected to expose the medial border of sternocleidomastoid. The plane between the larynx and oesophagus medially and the carotid sheath laterally is dissected using blunt dissection. The omohyoid muscle is retracted or divided. Pre-cervical fascia is divided medial to the neurovascular bundle and further blunt dissection exposes the longus colli muscles. These are elevated and a retractor placed. The intended spinal procedure can then be undertaken.

Anterior cervical decompression and fusion provides excellent results and has a low complication rate. The anterior approach allows access to the cervical disc that can be removed along with osteophytes at the posterior aspect of the vertebral body. It allows removal of most lesions causing myelopathy or radiculopathy. Placement of anterior bone graft between the vertebral bodies in the excised disc space helps to decompress the exit foramen indirectly and facilitates fusion.

Complications include pseudarthrosis (increased in smokers), hoarse voice and swallowing problems caused by retraction or injury to the recurrent laryngeal nerve (2–5%). This may also be caused by placement of the ET tube (more common). Graft complications also include the graft loosening and migration. Fusion alters the mechanics of the cervical spine, increasing the lever arms of forces acting at adjacent levels, and there is a significant rate of adjacent level degeneration.

One contributing cause of cervical stenosis may be ossification of the posterior longitudinal ligament, particularly in Japanese individuals.

Cervical disc replacement

Cervical disc replacement is a newer technique which treats similar pathologies through the same anterior approach but attempts to preserve motion in the cervical spine by replacing the cervical disk with materials similar to those used in large joint arthroplasty. Initial results are encouraging.[1]

1. Murrey D, Janssen M, Delamarter R *et al*. Results of the prospective, randomized, controlled multicenter Food and Drug Administration investigational device exemption study of the ProDisc-C total disc replacement versus anterior discectomy and fusion for the treatment of 1-level symptomatic cervical disc disease. *The Spine J* 2009;**9**:275–286.

Structured oral examination question 6: Spondylolisthesis

EXAMINER: What is this? (Figure 5.8.)

CANDIDATE: I can see T2-weighted sagittal and coronal MRI images showing the lumbar spine and there is a spondylolisthesis at L5/S1.

EXAMINER: What grade is it and what types of spondylolisthesis do you know?

CANDIDATE: Spondylolisthesis is graded according to Meyerding's grading system which is graded I–IV according to how far from posterior to anterior the more cranial vertebral

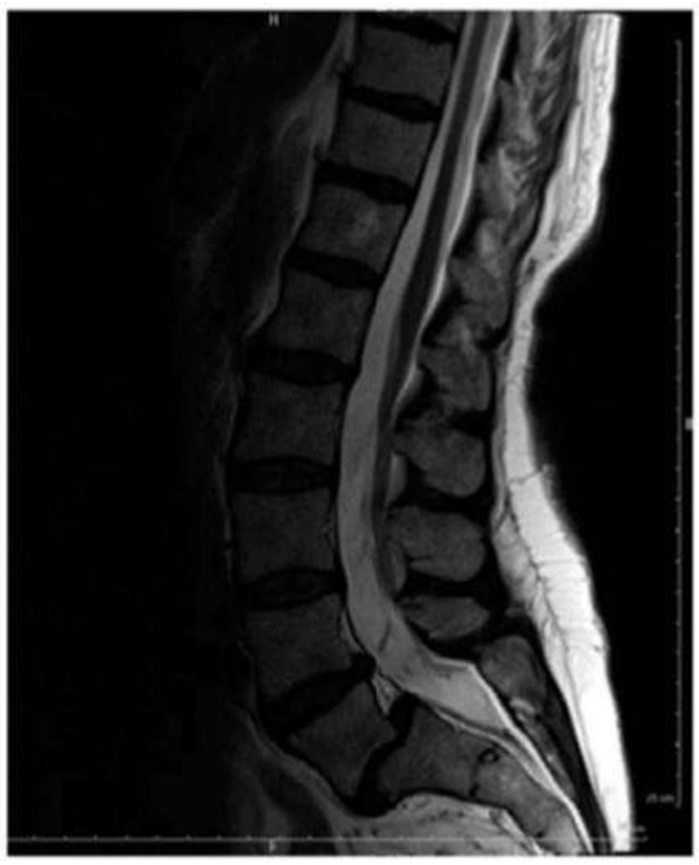


Figure 5.8 T2-weighted MRI sagittal demonstrating L5/S1 spondylolisthesis.

body has slipped forward. Grade I is less than a ¼ (25%), grade II is ¼ – ½ (25–50%), grade III is ½ – ¾ (50–75%) and grade IV is > ¾ (>75%). A spondyloptysis is a slip greater than 100% where the more cranial vertebral body lies anterior to the more caudal one (grade V).

Five different types of spondylolisthesis were described by Wiltze

I. Dysplastic – Congenital abnormalities of the sacrum or L5 allow the slip to occur.
II. Isthmic – Here the defect is in the pars and it is subdivided into a lytic failure, an acute fracture, or an elongated but intact pars.
III. Degenerative – This is due to degenerative change that produces intersegmental instability (due to changes in disc, joint capsules and facet joints).
IV. Traumatic – Due to a fracture (but not of the pars, e.g. pedicle).
IV. Pathological – Caused by local bone disease (disease may not be localized).

Spondylolisthesis background knowledge

Note: When considering an isthmic (spondylolytic) spondylolysis the 'step' in the spinous processes posteriorly the step in the posterior elements will occur one level above that of the pars defect. The posterior element step is at L4/5 in an L5 spondylolysis (the spondylolisthesis being at L5/S1).

In children spondylolytic (isthmic) spondylolisthesis at the L5/S1 junction is more common. Approximately 50% of spondylolyses have a spondylolysis without the associated slip. It is twice as common in men as in women. Typically it first occurs during or just before adolescence and may progress until skeletal maturity. There is a genetic component with between one-third and two-thirds having a family member affected. It may also be associated with spina bidifa (up to 40%). The main symptoms are usually a dull aching pain in the low back and buttocks exacerbated by activity; this may be associated with an L5 radiculopathy. Many are asymptomatic. Of symptomatic children the majority (90%) also become symptomatic again in adult life. Hamstring shortening is a common finding on examination. In high grade slips adolescents may present with a 'spondylolytic crisis' in which pain, neurological compromise and the Phalen–Dickson sign of flexed hips and knees and a waddling gait when walking may all be present.

Conservative management (activity modification, +/− bracing) may allow healing of the pars defect. Core stability exercises, hamstring stretching and bracing all have a role. Surgical stabilization may be considered if conservative management fails or a progressive slip is identified. Patients should be followed up until skeletal maturity after which it is unlikely that the slip will progress.

Degenerative spondylolisthesis most commonly occurs at the L4/L5 level and is frequently associated with stenosis at that level. There is an intact neural arch and the slip is caused by instability of the motion segment, in turn caused by dehydration of the disc and loss of disc height as well as facet joint degeneration. This type is five times more common in women than in men. Symptoms may be of back pain radiating into the thighs, radicular symptoms (50%), or symptoms of neurogenic claudication.

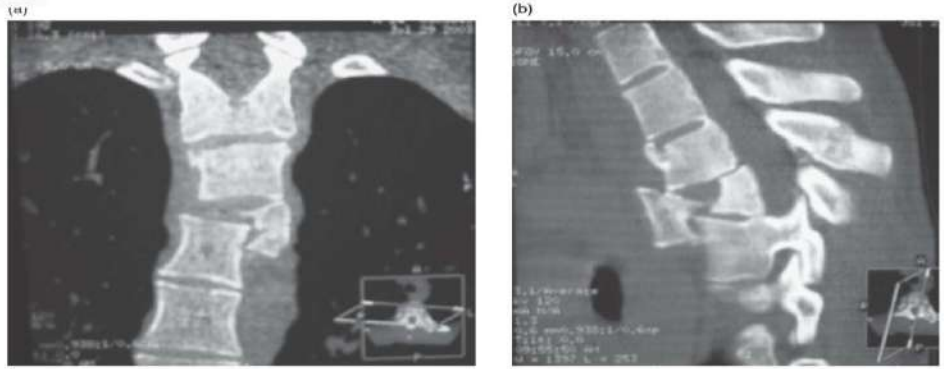
Structured oral examination question 7: Spinal trauma

EXAMINER: A 26-year-old man crashes his motor-bike and sustains the fracture shown. How would you go about assessing a patient with a suspected spinal injury? (Figure 5.9.)

CANDIDATE: The assessment of seriously injured patients begins with the Airway (with cervical spine control), Breathing and Circulation. Circulation assessment includes assessment for neurogenic shock. Then comes neurological disability assessment using the Glasgow Coma Scale followed by a log roll (looking for steps, swelling or bruising indicating posterior injury), rectal examination and neurological examination (using an ASIA chart). Initial imaging will include the trauma series (chest, c-spine and pelvis) X-rays. If a fracture is identified, imaging of the whole spine is required as there is a significant chance of a second fracture (10%). Once a fracture has been identified further imaging with a CT scan is indicated. MRI may also be required to assess disc and spinal cord injuries. Spine fractures are often associated with other injuries. Cervical spine fractures may be associated with vascular injuries, thoracolumbar fractures with visceral injuries and lumbar fractures with lower limb (calcaneal) fractures.

EXAMINER: What is neurogenic shock?

CANDIDATE: Neurogenic shock should be distinguished from hypovolaemic shock. Relative bradycardia and warm peripheries indicate the cause of shock is loss of sympathetic tone secondary to spinal cord injury (SCI).



Figures 5.9a and 5.9b CT scan images of a thoracic spinal fracture.

Spinal shock is transient neurological dysfunction that is caused by a contusion or oedema of the spinal cord that usually resolves over 24–72 hours. The bulbocavernosus reflex (usually tested by pulling on an indwelling catheter) is the first reflex to return.

EXAMINER: Would you give this person steroids?

CANDIDATE: No. The current (2006) BOA guidelines on the initial care of patients with spinal cord injuries states 'the use of high dose steroid in the management of acute spinal cord injury could not be recommended or supported on the current evidence'.[1]

EXAMINER: How do you classify thoracolumbar fractures?

CANDIDATE: I would use the AO classification. This divides thoracolumbar fractures into three types: A, B and C based on the mechanism of injury. Type A fractures are compression type injuries and are subdivided into three subtypes (type 1 – wedge, type 2 – pincer and type 3 – burst fractures). Type B fractures are distraction injuries associated with a fracture or ligamentous injury to the posterior column (flexion–distraction or hyper-extension injuries). They are subdivided into three types: type 1 – posterior ligamentous injury with anterior injury, type 2 (chance type) fractures of both anterior and posterior elements with distraction posteriorly, type 3 are associated with anterior distraction. Type C injuries are injuries that occur with rotation. The subtype C1 are A type fractures with rotation, C2 are B type fractures with rotation and C3 injuries are injuries with rotation and shear. Fractures become more unstable as type progresses from A to C and subtype from 1 to 3.

EXAMINER: What role does spinal surgery have and what factors do you know that indicate prognosis?

CANDIDATE: Incomplete spinal cord injuries are more likely to recover than complete injuries. Sacral sparing implies an incomplete lesion and improved prognosis. Spinal surgery attempts to decompress the injured spinal segment and stabilize the injury. Decompression aims to remove compression (either direct or indirect) but currently there is limited evidence showing improved neurological outcome. Stabilization of the spine reduces pain, facilitates patient handling and allows earlier mobilization helping prevent the complications associated with recumbency. Surgical stabilization prevents further displacement in unstable fractures preventing further injury and late deformity with better posture and balance. (A stable fracture is one that will not displace under normal physiological loads.)

1. *The Initial Care and Transfer of Patients with Spinal Cord Injuries*. London: British Orthopaedic Association, 2006.

Spinal trauma background knowledge

Vaccaro and colleagues have devised the TLICS system (thoracolumbar injury classification and severity score) for decision making in spinal trauma,[1]

4. Shoulder & Elbow :

A. Oral :

Glenoid Labrum : A fibrocartilagenous tissue structure that surrounds glenoid which helps create cavity-compression and creates 50% of the glenoid socket depth & considered a static GH Joint stabilizer

Buford complex (absent anterosuperior labrum + cordlike MGHL)

cordlike MGHL with attachment to base of biceps anchor and complete absence of the anterosuperior labrum

Rotator Interval : Space that bridge the gap between the supraspinatus and the subscapularis consists of capsule, SGHL, CHL LHBT

boundaries

medially by lateral coracoid base
superiorly by anterior edge of supraspinatus
inferiorly by superior border of subscapularis
lateral apex formed by transverse humeral ligament

Subacromial Impingement

is the first stage of rotator cuff disease which results due to either extrinsic compression of the rotator cuff between the humeral head and

1. anterior acromion
2. coracoacromial ligaments
3. acromioclavicular joint

or intrinsic degeneration of supraspinatus which leads to inability to balance the humeral head on the glenoid causing superior migration and narrowing of the subacromial space

Anterosuperior escape :

Proximal humeral migration due to acromioplasty and CA ligament release (coracoacromial arch disruption) which occurs in patients with massive, **irreparable rotator cuff tears**

Subcoracoid Impingement : impingement of the subscapularis between the coracoid and lesser tuberosity which is maximally experienced when arm is adduction,flexion, and internal rotation

Calcific tendonitis : Calcification and tendon degeneration near the rotator cuff insertion (most commonly involving supraspinatus) associated with subacromial impingement & Highly associated with endocrine disorders

Biceps Subluxation : **LHBT** *sublux*, or partially dislocate **out of its groove** due to disruption of the biceps sling comprised of

1- fibers of the subscapularis
2- supraspinatus
3- CHL
4- SGHL

& most commonly associated with subscapularis tears

Rotator Cuff Arthropathy :

degenerative joint disease that results from ***rotator cuff tears*** lead to loss of joint congruence due to Loss of dynamic concavity-compression which results in abnormal glenohumeral wear

Rocking-horse phenomenon :

A phenomenon characterized by high rate of glenoid wear & loosening when using TSA in RCA (RC deficient shoulder) due to HH displacement superiorly with motion due to loss of the compressive and inferiorly-directed force of the intact rotator cuff leading to eccentric loading of the glenoid component & leading to wear & loosening of component

Bankart lesion is an avulsion of the anterior labrum and AB of the IGHL from the anterior inferior glenoid.

(HAGL) : Humeral avulsion of the glenohumeral ligament
Glenoid labral articular defect (GLAD) : is a sheared off portion of articular cartilage along with the labrum
(ALPSA) : Anterior labral periosteal sleeve avulsion
Bony Bankart lesion is a fracture of the anterior inferior glenoid
Hill Sachs defect is a chondral impaction injury in the posterosuperior humeral head secondary to contact with the glenoid rim.

Drive-through sign : is considered the ability to pass an arthroscope easily between the humeral head and the glenoid at the level of the AB of IGHL

SLAP Lesion

Superior Labrum from Anterior to Posterior tears that may or may not involve the biceps attachment occurs due to **tightness** of the posterior-IGHL which shifts the GH contact point posterosuperiorly and increases the shear force on thesuperior labrum & increasing strain on the
anterior band - IGHL compromising stability of shoulder(by predisposing to Bankart through increasing strain over AB-IGHL

Internal Impingement :
repetitive impingement of the posterior undersurface of rotator cuff
(supraspinatus tendon) between greater tuberosity (Humeral head) & Glenoid
occurs during maximum arm abduction and external rotation during late cocking and early acceleration phases of throwing

Glenohumeral Internal Rotation Deficit (GIRD)
a condition resulting in the loss of internal rotation of the glenohumeral joint tightening of posteroinferior capsule which leads to translation of humeral head (capsular constraint mechanism) is in the OPPOSITE direction from area of capsular tightening

Little Leaguer's Shoulder
An overuse injury resulting in epiphysiolysis of the proximal humerus
Considered Salter Harris type I physeal injury occurs due to repetitive torsional and distractive stresses at the physis during late cocking phase (Max ER through growth plate) & deceleration phase (opposing forces of forward arm motion and rotator cuff results in excessive eccentric physeal stress)

Posterior Labral Tear :

referred to as a reverse Bankart lesion, or attenuation of the posterior capsulolabral complex occurs due to repetitive microtrauma to the posterior capsulolabral complex Or posteriorly directed force with the arm in a flexed, internally rotated and adducted position

Suprascapular Neuropathy

Can be caused by
suprascapular notch entrapment
weakness of both supraspinatus and infraspinatus
spinoglenoid notch entrapment
weakness of infraspinatus only

Brachial Neuritis (Parsonage-Turner Syndrome)

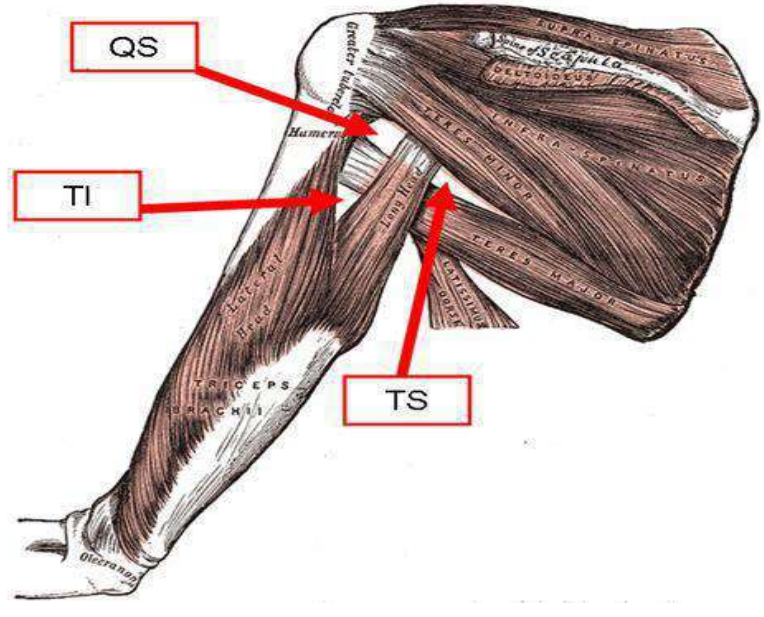
severe shoulder pain followed by patchy muscle paralysis and sensory loss . due to either 1. Autoimmune 2. Biomechanical (may disrupt the blood-nerve barrier that normally prevents immune factors from contacting the peripheral nerve system)

Thoracic Outlet Syndrome

Neurovascular disorder resulting from compression of the brachial plexus and/or subclavian vessels in the interval between the neck and axilla

Quadrilateral Space Syndrome

Axillary nerve and posterior humeral circumflex artery compression in the quadrilateral (quadrangular) space results in weakness of ER in Abduction



Scapulothoracic Dyskinesis : abnormal scapula motion leading to shoulder impingement and dysfunction characterized by scapulothoracic power imbalance leads to protraction of scapula resulting in excessive stress placed on anterior capsule of shoulder and posterosuperior labrum

Adhesive Capsulitis (Frozen Shoulder) :

condition of the shoulder characterized by functional loss of passive and active shoulder motion with no clear underlying cause (idiopathic) , or due to immobilization post trauma or surgery of RCT

Avascular Necrosis of the Shoulder

bone cell death caused by interruption of blood supply to humeral head leads to subchondral bone collapse and morphological/arthritic change

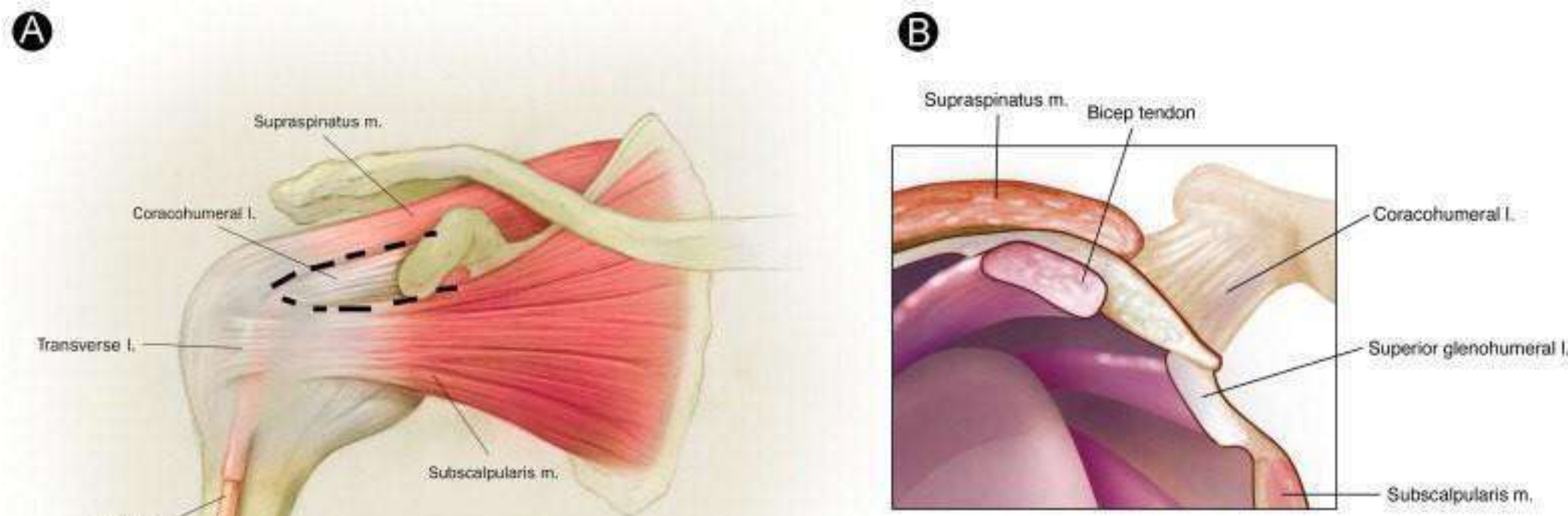
Reverse Shoulder Arthroplasty

Use of a convex glenoid and concave humerus to reconstruct the glenohumeral joint in order to establish center of rotation (COR) inferiorly and medialized
which allows the deltoid muscle to act on a longer fulcrum
to have more mechanical advantage

Cubital fossa

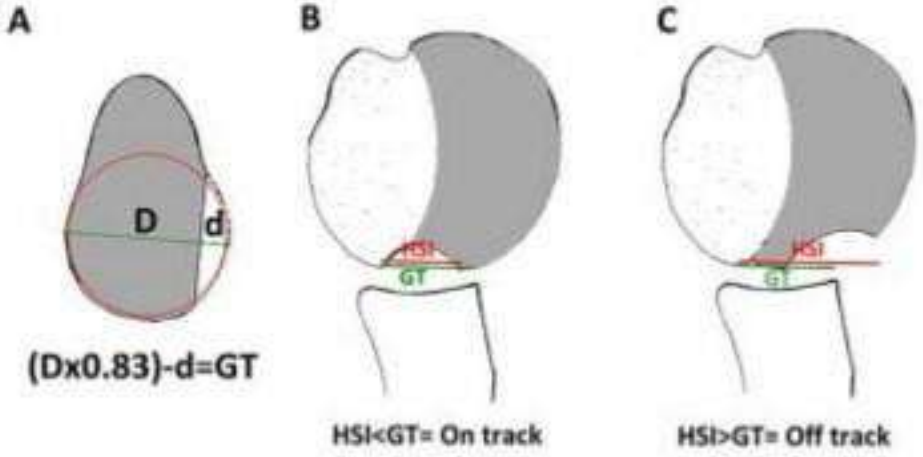
contents-- (medial to lateral): Median nerve (most medial structure), brachial artery, biceps tendon, radial nerve (most lateral structure)

lateral border--brachoradialis
medial border--pronator teres
proximal border --distal humerus

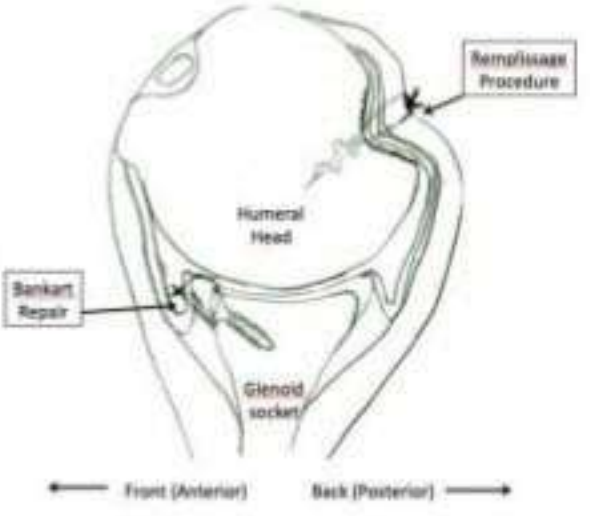


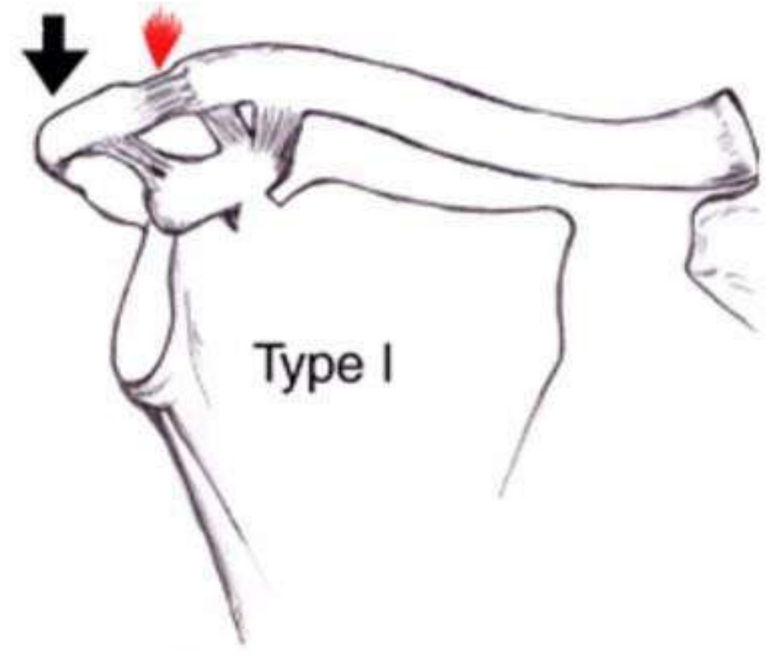
Gap between SupraS & SubS
Capsule+CHL+Long H of B

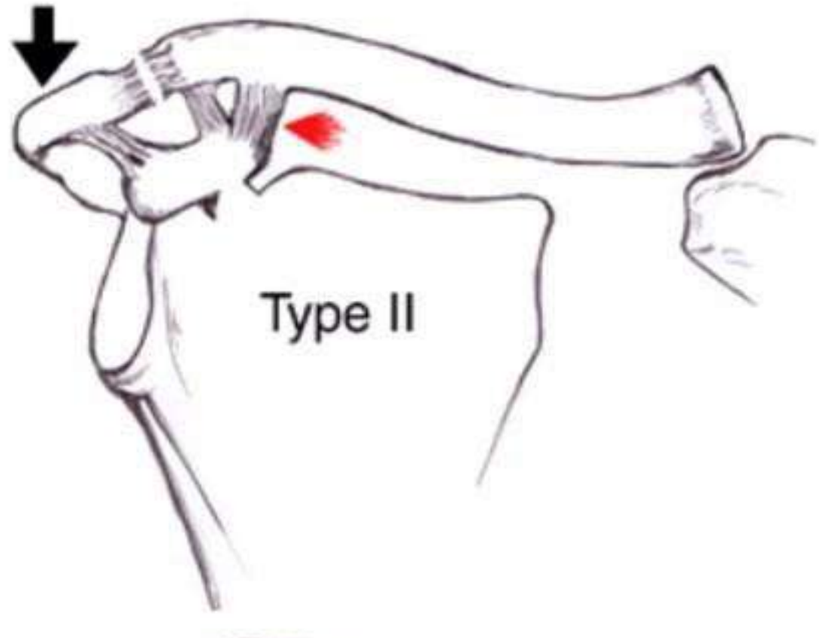
A Hill-Sachs lesion has a risk of engagement and dislocation if it extends medially over the medial margin of the glenoid track.



Remplissage procedure




Type I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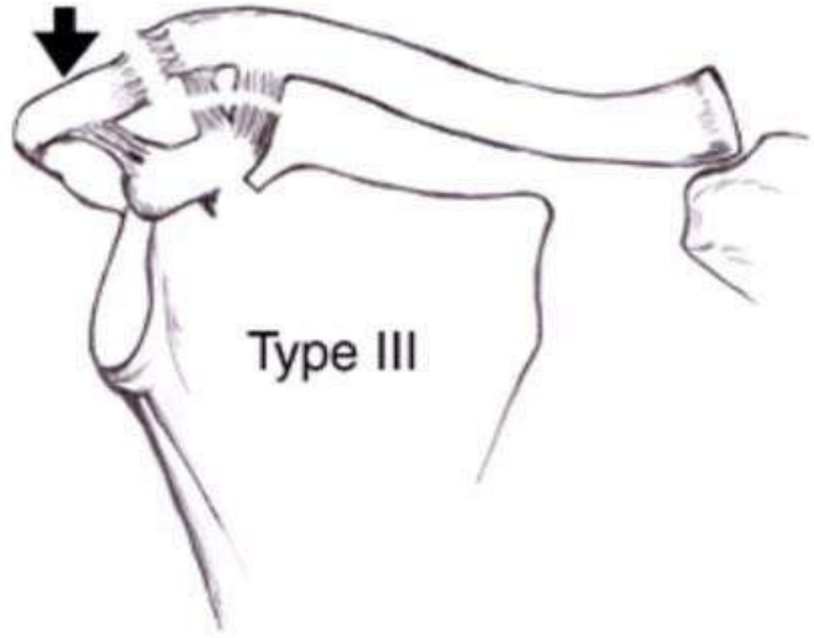
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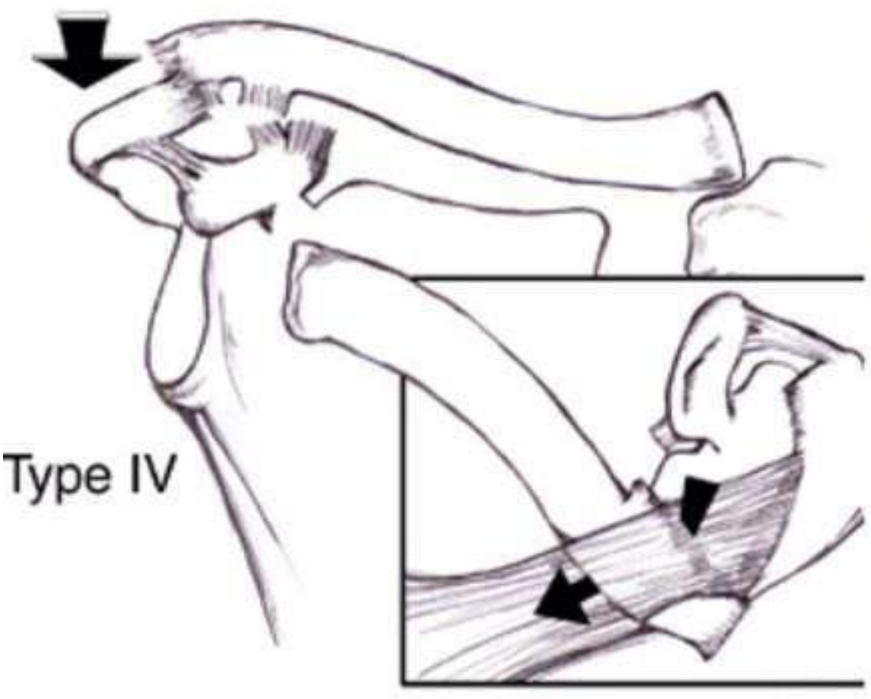
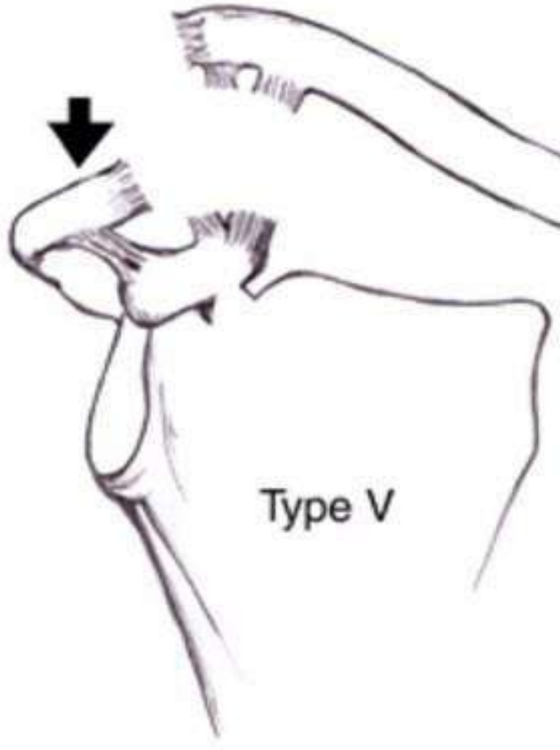
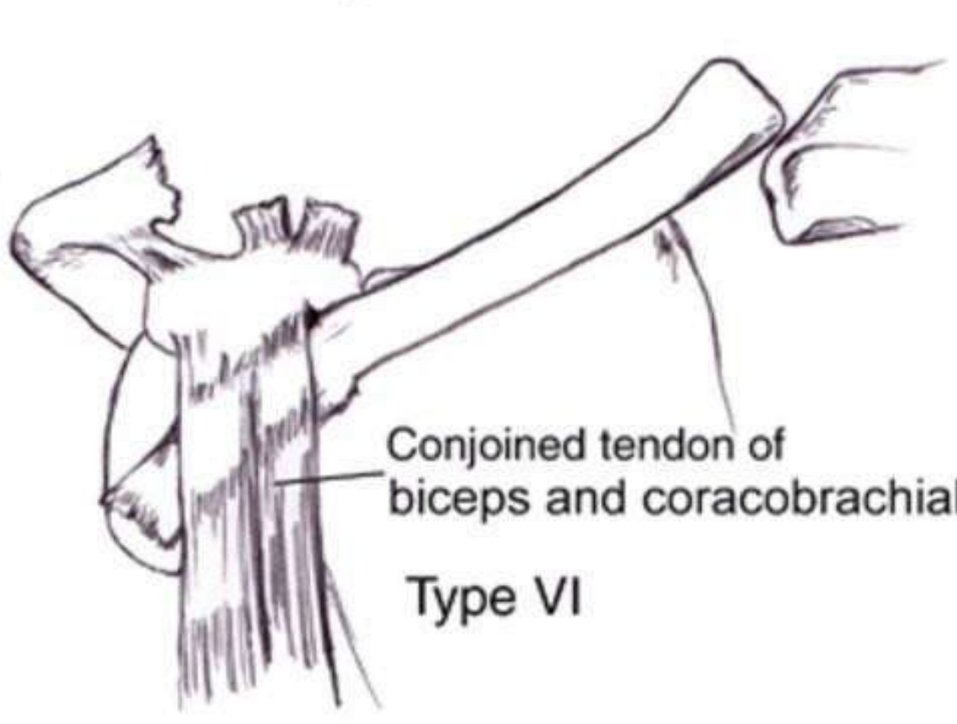
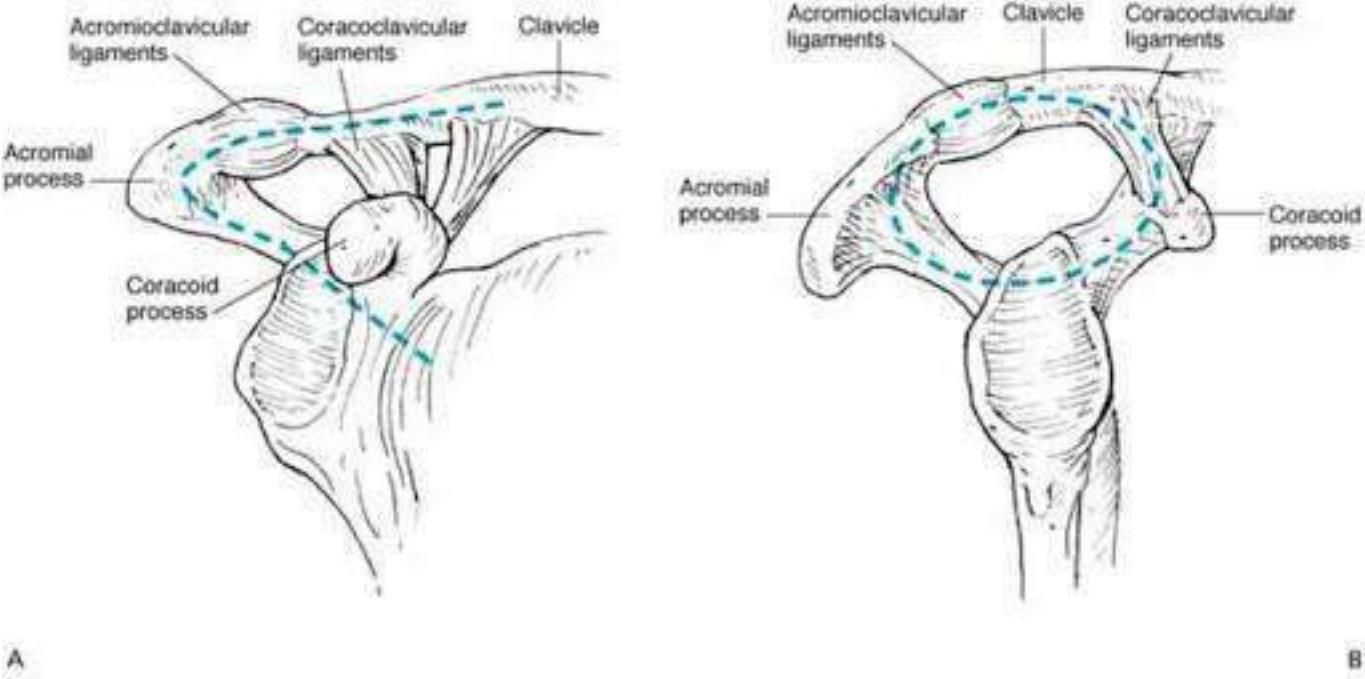
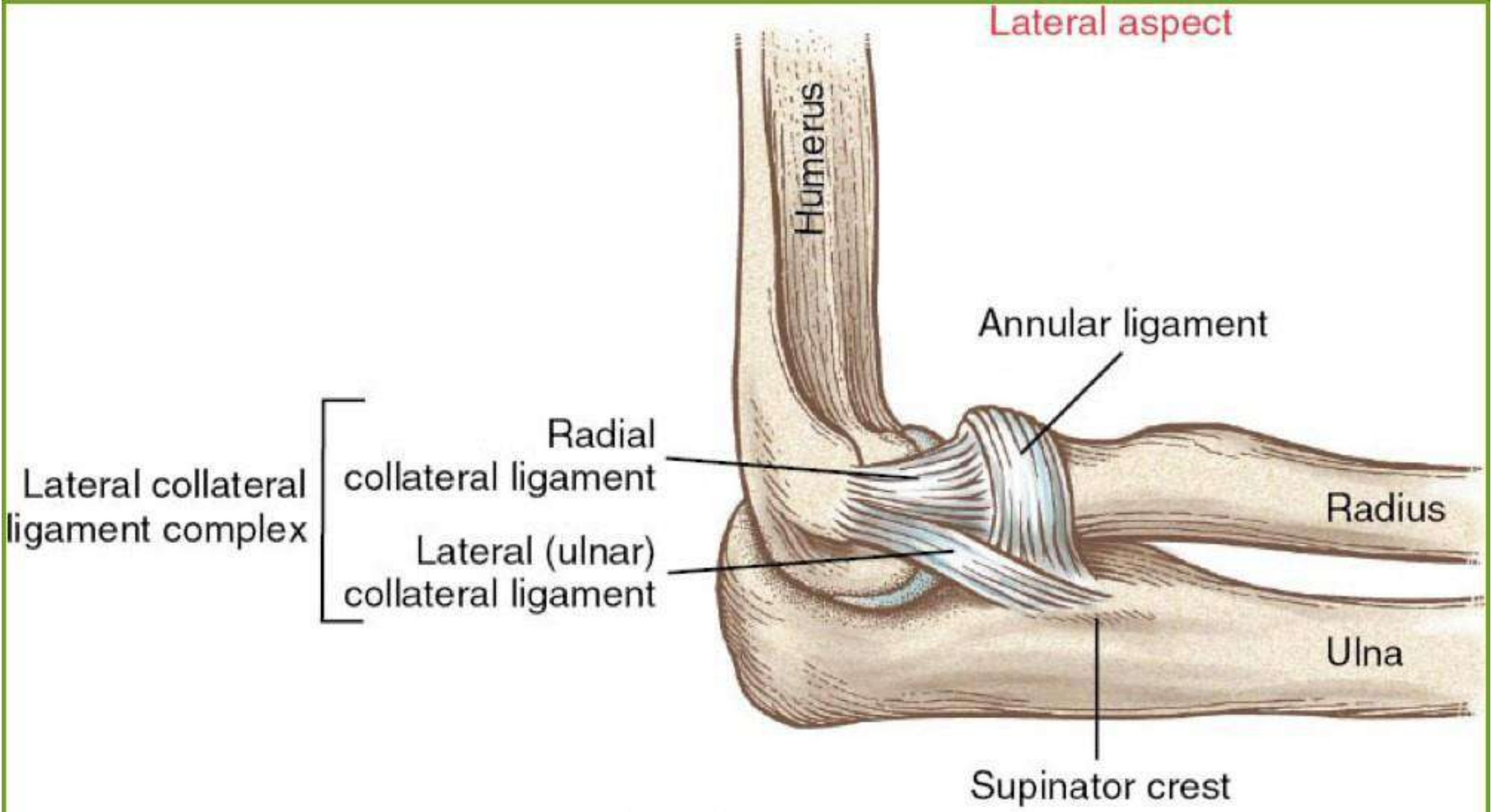

Type IV


Type V

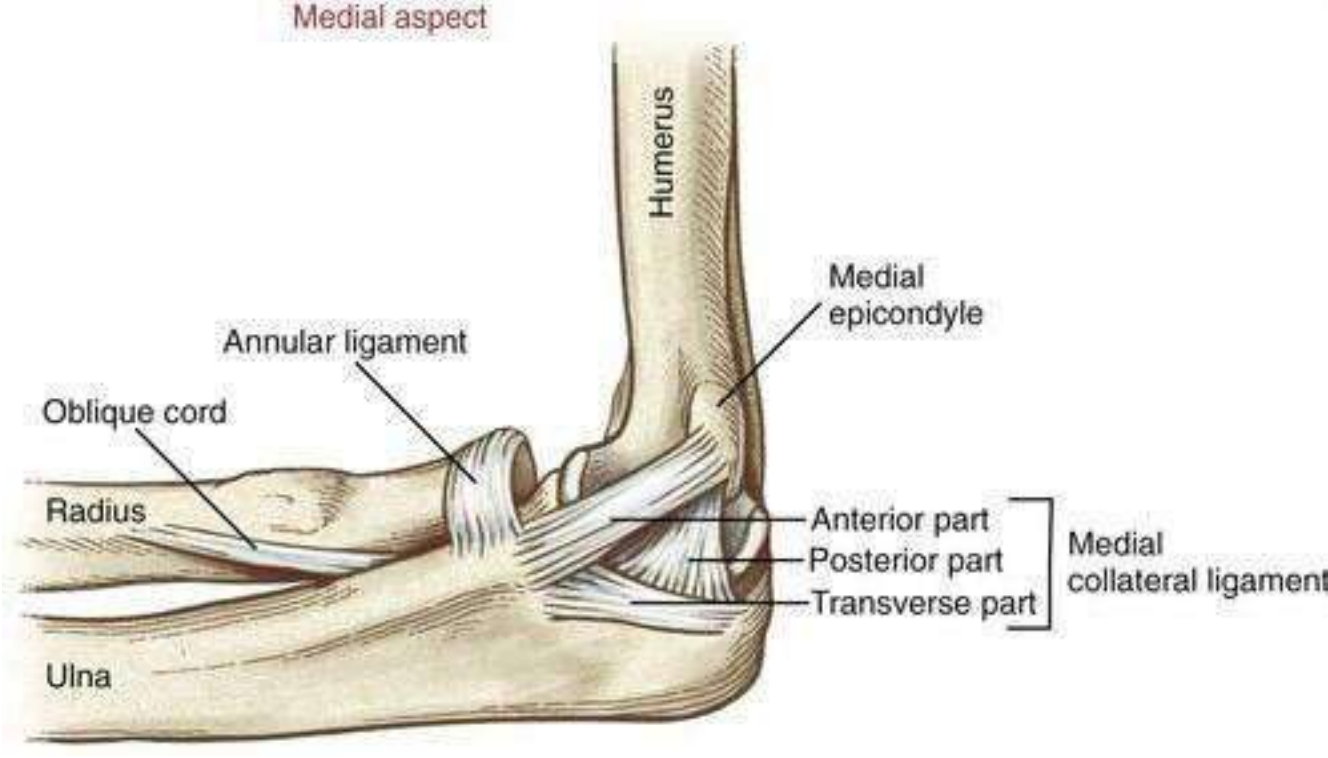

Conjoined tendon of
biceps and coracobrachialis
Type VI

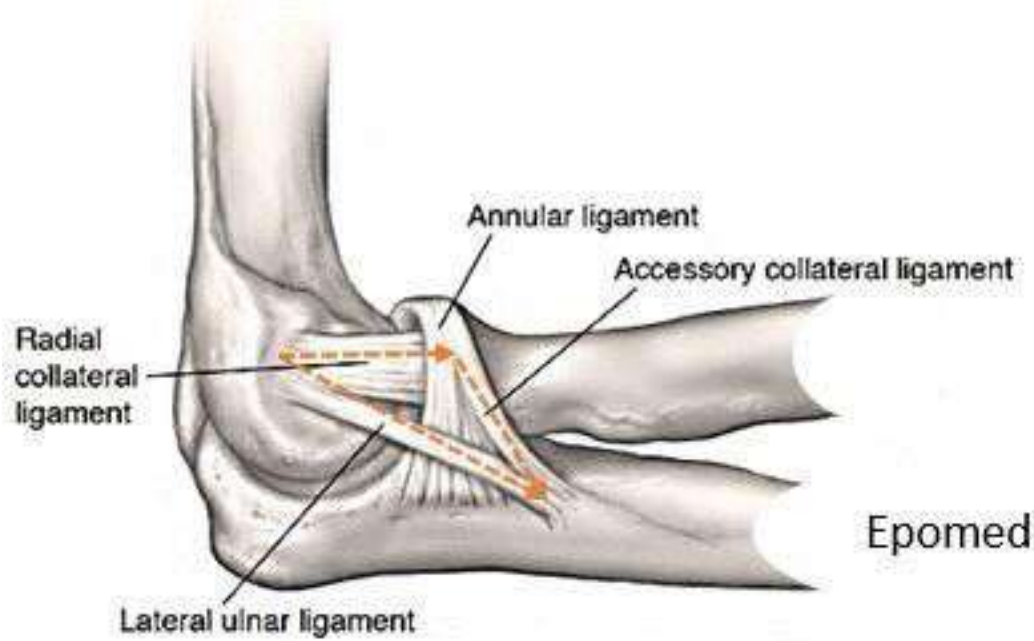

Acromioclavicular ligaments
Coracoclavicular ligaments
Clavicle
Acromial process
Coracoid process
A
Acromioclavicular ligaments
Clavicle
Coracoclavicular ligaments
Acromial process
Coracoid process
B

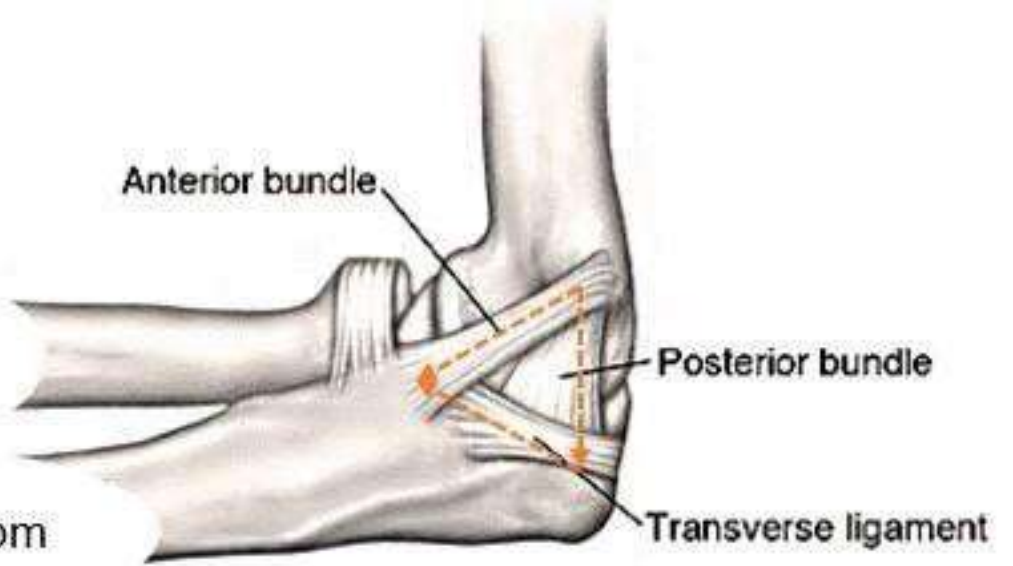
- Static and dynamic stabilizers confer stability to the elbow
  - static stabilizers (primary)
    - ulnohumeral joint
    - anterior bundle of the MCL
    - LCL complex (includes the LUCL)
  - static stabilizers (secondary)
    - radiocapitellar joint
    - joint capsule
    - origins of the common flexor and extensor tendons
  - dynamic stabilizers
    - muscles that cross the elbow joint, which apply compressive (stabilizing) force
      - anconeus
      - brachialis
      - triceps



(From Neumann: Kinesiology of the Musculoskeletal System, 2nd edition.)


Medial aspect
Humerus
Medial epicondyle
Annular ligament
Oblique cord
Radius
Anterior part
Posterior part
Transverse part
Medial collateral ligament
Ulna





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Lateral collateral ligament complex

Medial collateral ligament complex

B.Viva : Long & Short

Chapter 6

Shoulder and elbow structured oral questions

Asir Aster

Introduction

Viva is like playing a game. The candidate should know the subject well, have a game plan and should know the opponent (who has been bored by the previous unchallenging candidates). A candidate who asks clever questions and answers appropriately will gain control (over the examiner) making it a rewarding 5 minutes (for both examiner and candidate) and more importantly will score highly in the viva. An examiner relishes a candidate who takes control and makes his life easy.

Avoid guess work. Avoid talking generally about the shoulder conditions to fill the time if your aim is to score well. A targeted question or answer will take you far.

Again I must stress the importance of time management in viva, as you have got only 5 minutes to score either 8 or 4.

The main aim of this chapter is to express the importance of the viva techniques and therefore it is not written as a textbook. Analyse the good as well as the poor techniques illustrated in the scenarios and follow the ones you find useful.

Shoulder

In a shoulder structured oral question try to analyse the question according to its presentation. Broadly, the shoulder pathology could be classified as painful, weak, stiff or unstable conditions. Shoulder pathology varies with different age groups and therefore you should have a list of age-related diagnoses clear in your mind, which will be helpful in the viva. There can be overlaps of these conditions, for example a painful stiff shoulder may represent frozen shoulder or acute calcific tendonitis. Therefore candidates should have a list of conditions and one or two classic questions to differentiate one from another, to lead into the scenario comfortably right from the start.

Structured oral examination question 1

EXAMINER: This is a radiograph of the left shoulder of an 84-year-old lady. Describe the radiograph please. (Figure 6.1.)

CANDIDATE: Well . . . Good morning.

This is the plain radiograph of an 84-year-old lady's left shoulder . . . anteroposterior (AP) view. There is evidence of joint destruction with loss of articular anatomy.

EXAMINER: What do you think is wrong with this shoulder?

CANDIDATE: Well, to be certain I need to ask a few questions and examine the patient.

EXAMINER: Go on then and ask some questions.

CANDIDATE: Is she right handed or left handed?

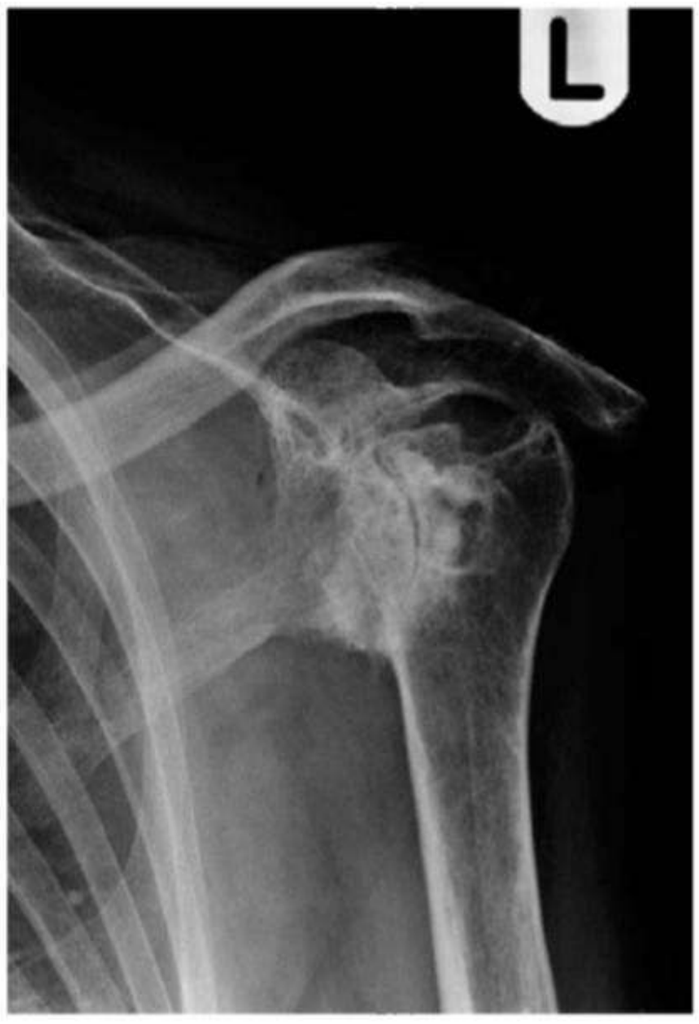


Figure 6.1 Anteroposterior (AP) radiograph left shoulder.

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

EXAMINER: Right handed.

CANDIDATE: How long has she had a problem with this shoulder?

EXAMINER: 70 years.

CANDIDATE: How did the problem start?

EXAMINER: It started as a painless lump when she was 14 and a few months later she began to have a discharging sinus that required several joint washouts and medication.

CANDIDATE: Does she have an active sinus now?

EXAMINER: No, the sinus healed after she underwent the washouts and started the medication and has never recurred.

CANDIDATE: That is good. What are her current problems?

EXAMINER: Well, she has some restriction of movements and therefore visited her GP who performed this X-ray and sent her to you for your opinion.

CANDIDATE: Then I would examine the patient.

EXAMINER: She has 60° of abduction and forward elevation and has very restricted rotations.

CANDIDATE: I would like to know the power of her cuff muscles.

EXAMINER: It is not possible to assess the power as she has very restricted range of movements.

CANDIDATE: Now . . .

TRING. . . . . .

EXAMINER: Thank you.

Did this candidate do well? Was there a diagnosis? Was there a discussion about the management? Only a 4 or 5 score would be given as the candidate did not even arrive at a diagnosis and missed all the clues.

A different candidate with the same scenario:

EXAMINER: This is a radiograph of left shoulder of 84-year-old lady. Describe the X-ray please.

CANDIDATE: This radiograph of shoulder anteroposterior (AP) view shows evidence of joint destruction and loss of articular cartilage.

EXAMINER: What do you think is wrong with this shoulder?

CANDIDATE: This appearance suggests several possible causes such as previous joint infection, trauma or a neurogenic cause. May I know how the problem started?

EXAMINER: Problems started as a painless lump when she was 14 and a few months later she began to have a discharging sinus for which she had several joint washouts and medication.

CANDIDATE: The presentation sounds like she had a low-grade joint infection. Was there any microbiological investigation performed at the time of the joint washouts?

EXAMINER: Yes, it was diagnosed as acid-fast bacillus and now what will be your management?

CANDIDATE: Well, I would like to know if she had any reactivation of infection in the last 70 years.

EXAMINER: No.

CANDIDATE: In that case what is the expectation of the patient?

EXAMINER: The patient does not want any surgical treatment. She wants to know if she can have some injections into her shoulder which can prevent the pain at the extremes of movements.

CANDIDATE: I will be cautious about the intra-articular injections as it can trigger the dormant bacillus and rekindle the infection.

EXAMINER: The patient does not want to take this risk and wants to be left alone.

Thank you.

Although the viva questions started in the same manner, this candidate with his or her knowledge took the viva to a good level of demonstration of his or her clinical judgement by asking specific questions and had control over the situation. Certainly this candidate deserves a good score.

Structured oral examination question 2

EXAMINER: Good afternoon. Can you tell me what is going on in this radiograph of the right shoulder (Fig. 6.2.)? This patient had anterior dislocation 2 years ago and has on-going problems.

CANDIDATE: Well this shoulder is reduced congruently. I cannot see any interposition of bony fragments. And I would like to investigate this shoulder with MR arthrogram.

EXAMINER: !! What do you want to rule out?

CANDIDATE: Well the risk of re-dislocation of the shoulder is much higher with anterior dislocation due to labral detachment in younger patients and it could be treated successfully if identified with MR arthrogram.

EXAMINER: This gentleman is claustrophobic!

CANDIDATE: I would talk to the radiologist and anaesthetist to find out if it could be done under sedation.

Section 2: Adult Elective Orthopaedics and Spine

EXAMINER: The anaesthetist is not happy! And your radiologist suggests an ultrasound examination of the shoulder.

CANDIDATE: Ultrasound examination is not the gold standard examination for labral pathology.

EXAMINER: Well, the patient had only ultrasound examination and it shows a subscapularis tear!

CANDIDATE: There is then a high risk of having damaged the anterior labrum also . . . I think I have to speak to the anaesthetist again . . .

Another candidate follows this miserable viva of negotiations between anaesthetist and radiologist in the FRCS ortho exam (by the candidate's own fault).

EXAMINER: Good afternoon. Can you tell me what is going on in this radiograph of the right shoulder? This patient had anterior dislocation 2 years ago and has on-going problems.

CANDIDATE: Thanks. May I know the age of the patient and the nature of the ongoing problem please?

EXAMINER: This 76-year-old gentleman dislocated his shoulder 2 years ago. Now he has got difficulties in overhead activities and we found out that he is claustrophobic!

CANDIDATE: I suspect rotator cuff tear in this age group following dislocation and also there is a risk of infra-clavicular plexus injury following the dislocations, therefore I would like to assess his cuff muscles clinically.

EXAMINER: He has got weakness on internal rotation and the rest of the cuff power is good. Neurologically he is intact.

CANDIDATE: I suspect subscapularis tendon tear from this clinical assessment and I would investigate this shoulder with an ultrasound examination.

EXAMINER: The ultrasound examination shows subscapularis tear, with proximal migration of the tendon by 4 mm.

CANDIDATE: I would like to know, what has been done so far? And what are his expectations?

EXAMINER: Nothing has been done so far. He wants to play golf, which he has not been able to in the last 2 years.

CANDIDATE: Well, I would assess his shoulder arthroscopically and repair his cuff.

EXAMINER: Would you call this a cuff arthropathy as it is going on for 2 years?

CANDIDATE: No. The radiograph does not show any evidence of proximal migration of the humeral head. And the ultrasound scan shows intact supra- and infraspinatus tendons. To develop cuff arthropathy at least two of the three supports should have been lost.

TRING . . .

This candidate knew the importance of age-related pathophysiology and succeeded well in the viva.

Structured oral examination question 3

EXAMINER: This is a radiograph of right shoulder of a lady who has got severe pain in her shoulder. Anything you find interesting? (Figure 6.2.)

CANDIDATE: Well . . . No not really . . . I cannot see any abnormal or disease process in this radiograph.

EXAMINER: She is in your clinic referred by her GP. What would you like to do for her?

CANDIDATE: I want to get history . . . then to examine the patient . . . to decide on the management plan.

EXAMINER: Go ahead.

CANDIDATE: In the history I will first find out her age, job and dominant side . . . and how and when the problem started.

EXAMINER: She is 45, right-hand dominant and does clerical work. The pain started 8 months ago when she was reaching out for the seat belt in her car.

CANDIDATE: The age and history suggest probable frozen shoulder . . . I will proceed with the examination.

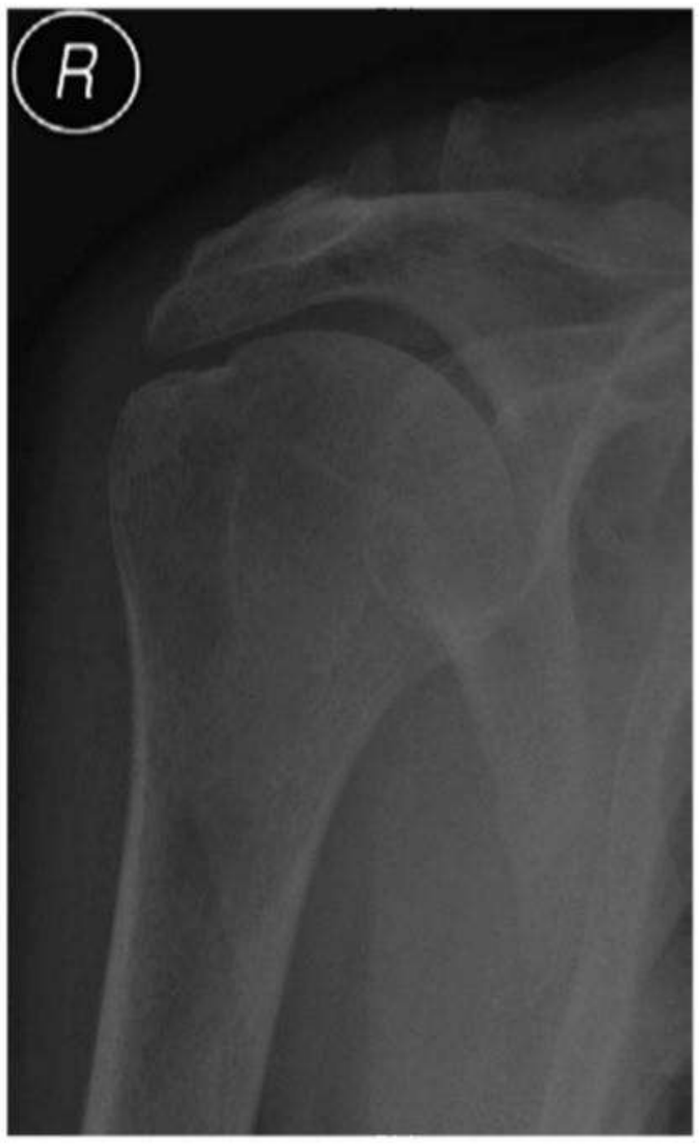


Figure 6.2 Anteroposterior (AP) radiograph right shoulder.

EXAMINER: She has got global restriction of her movements.

CANDIDATE: That confirms frozen shoulder. So . . .

EXAMINER: What do you want to do?

CANDIDATE: I would offer intra-articular steroid injection for her shoulder and also advice on stretching exercises by physiotherapists.

EXAMINER: She has already had three intra-articular steroid injections and regular physiotherapy from her GP practice.

CANDIDATE: Well in that case I would advise her to have manipulation under anaesthesia or arthroscopic arthrolysis.

EXAMINER: What will you specifically offer the patient?

CANDIDATE: mmm . . . MUA.

EXAMINER: The patient wants to know the risks associated with MUA.

CANDIDATE: Well apart from the anaesthetic risks, there is a risk of fracturing the humerus as it can be osteopenic from disuse . . . also the risk of recurrence.

EXAMINER: If the bone fractures, what will be the management?

CANDIDATE: It is like any fracture. Can be treated in a cast or operated.

EXAMINER: The patient decides now to leave it alone.

CANDIDATE: I will then convince her to have an injection today and review her situation in 12 weeks.

Do you think this candidate impressed the (patient) or the examiner, with this simple shoulder scenario? Before we look at the next candidate, think how you would approach this differently!

EXAMINER: This is a radiograph of right shoulder of a lady who has got severe pain in her shoulder. Anything do you find interesting?

CANDIDATE: Yes, this radiograph is essentially normal. May I know the age of this patient and does she suffer from diabetes or thyroid-related problems?

EXAMINER: Well she is 45 and she has hypothyroidism. Is there anything else would you like to examine other than her shoulders?

CANDIDATE: Yes, I would like to look at her hand to see if she has any evidence of Dupuytren's contracture as it has some association with frozen shoulder.

EXAMINER: She is in your clinic referred by her GP. What would you like to do for her?

CANDIDATE: I want to know the history and examination findings.

EXAMINER: She is right-hand dominant and does clerical work. The pain started 8 months ago when she was reaching out for the seat belt in her car. She has got global restriction of her movements.

CANDIDATE: Does this pain affect her sleep? What is the range of her external rotation?

EXAMINER: Yes, she struggles to sleep at night and her ER is only to neutral position. What would you like to do for her?

CANDIDATE: I want to know what has been done to her so far and what is her expectation?

EXAMINER: She has had three intra-articular injections and physiotherapy from her GP practice. She wants to be able to wash and dress herself independently.

CANDIDATE: Well, I would like to offer her either manipulation under anaesthesia or arthroscopic capsular release, explaining the advantages and disadvantages of both procedures and the importance of immediate post-intervention physiotherapy, and make her understand the disease process of frozen shoulder so that the patient could have a realistic expectation of the treatment process.

EXAMINER: The patient understands your explanation very well and wants to have the key-hole surgery. What will you do in arthroscopic capsular release?

CANDIDATE: The anterior capsule release especially at the rotator interval, followed by middle glenohumeral ligament release and the release of coracohumeral ligament. Inferior capsule will be stretched by manipulation . . . this is my preference as the arthroscopic release of inferior capsule carries a small risk of damaging axillary nerve.

EXAMINER: Thank you.

When the examiner sensed an ability, a small extra challenge was given – anywhere else you want to examine? And the candidate was able to demonstrate his or her knowledge – association with Dupuytren's contracture – the candidate would have been given an extra point for these smart moves.

Structured oral examination question 4

EXAMINER: This is a radiograph of a 63-year-old gentleman's right shoulder. Proceed.

CANDIDATE: This plain AP radiograph shows normal glenohumeral joint and acromioclavicular joint, well-maintained subacromial space but the undersurface of the acromion is sclerotic suggesting possibility of him suffering from subacromial impingement. Can I see an axillary view please?

EXAMINER: Yes.

CANDIDATE: There are deposits of calcium in the supraspinatus tendon . . .

EXAMINER: What is your opinion about his pain in the shoulder?

CANDIDATE: Well, he could be struggling with calcific tendonitis.

EXAMINER: What do you want to do?

CANDIDATE: I would like to know the patient's symptoms, examination findings, the treatments he had so far and his expectations.

EXAMINER: He is a keen golfer and gradually over the last 2 years he has developed the pain on over-head activities. He has not had any interventions so far. He wants to continue playing golf without pain. He has got positive impingement signs.

CANDIDATE: Well, I would inject his subacromial space with steroid today to relieve the bursitis secondary to the calcific tendonitis, which is causing impingement symptoms and review him in 8 weeks in clinic with repeat X-rays to assess the calcium deposits.

EXAMINER: Incidentally there is also another X-ray of his right shoulder which was performed 2 years ago when he started to have the pain, which shows the same calcium deposits. Does it change your plan?

CANDIDATE: . . . Well, I would then book him now for arthroscopic excision of the calcium deposits.

EXAMINER: Will you perform any other procedures during the surgery?

CANDIDATE: I will consent him for arthroscopy and proceed . . . so that I can assess the shoulder and perform the necessary at the time of the surgery.

Did he not start well? Did this candidate proceed well – with diagnosis and management plan? Did he pick up the clues by the examiners and correct himself? What will be your scoring for this candidate? Will you diagnose and manage this problem differently like the next candidate?

EXAMINER: This is a radiograph of a 63-year-old gentleman's right shoulder. Proceed.

CANDIDATE: This plain AP radiograph shows normal glenohumeral joint and acromioclavicular joint, well-maintained subacromial space but the undersurface of the acromion is sclerotic suggesting possibility of him suffering from subacromial impingement. Can I see an axillary view please?

EXAMINER: Yes.

CANDIDATE: There are deposits of calcium in the supraspinatus tendon . . .

EXAMINER: What is your opinion about his pain in the shoulder?

CANDIDATE: Looking at the radiographs, duration of his problem . . .

(*EXAMINER*: 2 years) and his age I feel he has got degenerative calcification in his cuff and subacromial impingement.

EXAMINER: What do you want to do?

CANDIDATE: I would like to know the patient's symptoms, examination findings, the treatments he had so far and his expectations.

EXAMINER: He is a keen golfer and gradually over the last 2 years he has developed the pain on over-head activities. He has not had any interventions so far. He wants to continue playing golf without pain. He has got positive impingement signs.

CANDIDATE: I want to know if he had any X-rays in the past and would like to assess the status of his cuff with an ultrasound scan.

EXAMINER: This is the X-ray taken 2 years ago – showing the same calcification. The ultrasound scan shows intact cuff.

CANDIDATE: Well I would inject the subacromial bursa today with steroid and review the patient in 8 weeks to see if the injection has helped his pain as a diagnostic test for impingement.

EXAMINER: He comes back in 8 weeks saying the pain was well controlled for 3 weeks and now the pain is back. What will you do?

CANDIDATE: This proves the pathology of subacromial impingement and I am going to talk to the patient about the subacromial decompression.

EXAMINER: Will you perform excision of the calcium deposits?

CANDIDATE: No, not necessarily. This degenerative calcification is a chronic one. It is not acute calcific tendonitis. Therefore I will perform only the subacromial decompression.

EXAMINER: Thank you.

This second candidate was much clearer about the pathology and management plan, which will be rewarded by a better score. He did not have to be prompted by the examiners regarding the calcium deposit which was there 2 years ago suggesting the degenerative calcification. The previous candidate failed to understand these prompting clues.

Structured oral examination question 5

EXAMINER: Good afternoon. Can you tell me the findings from this radiograph of the left shoulder of a 76-year-old left-handed fit gentleman? (Figure 6.3.)

CANDIDATE: This anteroposterior view of left shoulder shows no evidence of glenohumeral joint or acromioclavicular joint arthritis. The subacromial space is narrowed with sclerosis of the undersurface of the acromion.

EXAMINER: Would you like any other investigations . . . prior to committing yourself with a diagnosis?

CANDIDATE: I would like to have ultrasound of his shoulder . . . and may I know his symptoms please?

EXAMINER: The ultrasound, which was requested by his GP, shows torn subscapularis and supraspinatus with massive retraction of the tendons. He has difficulties with overhead activities. Can you tell me what is wrong with this shoulder?

CANDIDATE: From the X-ray . . . which shows evidence of impingement by narrowing of the subacromial space, from the ultrasound scan . . . which shows evidence of torn subscapularis and supraspinatus tendons and clinically he has got difficulties in overhead activities . . .

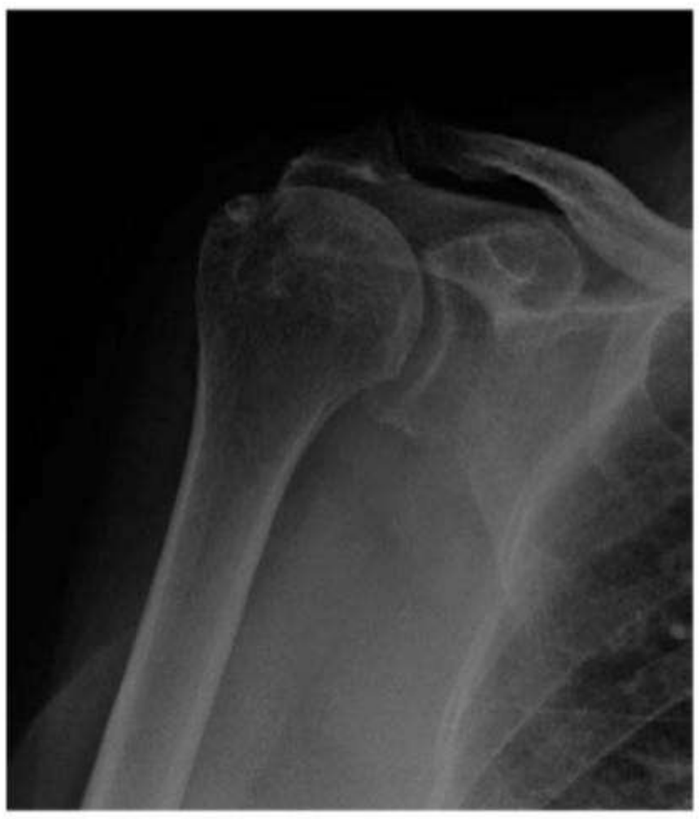


Figure 6.3 Anteroposterior (AP) radiograph left shoulder.

EXAMINER: Yes, it is a nice summary of the situation (wasting time)

CANDIDATE: I think he has severe subacromial impingement and secondary cuff tear.

EXAMINER: What would you do for this gentleman?

CANDIDATE: Well, first I would perform a steroid injection into his subacromial space.

EXAMINER: Can you tell me the landmarks and how will you perform the injection?

CANDIDATE: Yes, 2 cm inferior and medial to the posterolateral corner of the acromion, I will direct the needle towards the anterolateral corner of the acromion to be specific into the bursa.

EXAMINER: Is it necessary to be specific in this patient . . . he has got a massive cuff tear?

CANDIDATE: ??

EXAMINER: Well, he comes back to clinic in 8 weeks with no difference to his symptoms. Do you have any management plans?

CANDIDATE: I will then perform an arthroscopic debridement of the cuff and bursa and a subacromial decompression.

EXAMINER: !! Thank you.

Do you recognize the candidate's mistakes? What will you do differently? Did he treat the patient or the investigations? Did he interpret the investigations appropriately? Now the last candidate of the day arrives for the same scenario.

EXAMINER: Good afternoon. Can you tell me the findings from this radiograph of the left shoulder of a 76-year-old left-handed fit gentleman?

CANDIDATE: This anteroposterior view of the left shoulder shows proximal migration of humeral head with narrowing of the subacromial space and there is no evidence of glenohumeral joint or acromioclavicular joint arthritis.

EXAMINER: Would you like any other investigations . . . prior to committing yourself with a diagnosis?

CANDIDATE: I would like to have an axillary view of his shoulder.

EXAMINER: Yes, we have axillary view. What are you looking for?

CANDIDATE: I am looking for anteroposterior subluxation of the humeral head in the axillary view . . . yes, there is anterior subluxation, suggesting torn anteriorly placed subscapularis and from the AP view, the proximal migration of the humeral head suggesting supraspinatus tear . . . this gentleman has got established cuff arthropathy.

EXAMINER: What would you do for him?

CANDIDATE: I need to know the patient's symptoms, what has been done to the patient so far and what are his expectations?

EXAMINER: He has got difficulties in overhead activities. He has had three injections by his GP which has made no difference and being an artist he would like to have reasonable ability to abduct his shoulder to reach for the top of the canvas during painting.

CANDIDATE: Could you please tell me if he has any pain associated with his shoulder abduction?

EXAMINER: No . . . not at all

CANDIDATE: I would then offer a reverse-polarity shoulder replacement if he is otherwise healthy and fit for surgery.

EXAMINER: He is very fit. Why do you prefer reverse shoulder to a total shoulder replacement?

CANDIDATE: The reverse shoulder although non-anatomical brings the centre of rotation of the glenohumeral joint medially and thereby increases the moment arm of the deltoid, allowing good abduction of the shoulder.

EXAMINER: Would you not try to repair the cuff prior to this major surgery?

CANDIDATE: No. The radiographs show an established cuff arthropathy and in this situation a rotator cuff repair is not possible.

EXAMINER: Well, we will move on to the next scenario.

Whom do you think played the game well in this scenario? Analyse the candidate's ability to show their knowledge to the examiner. Learn how not to waste time and not to lower the expectations of the examiner. When the examiner's expectations go down, the questions may become simpler and the score becomes lower. Show the knowledge appropriately to please the examiner. Make the game interesting for the examiners and you walk away with a good score. Treat each scenario as a separate exam to reach a good overall score. Remember the examiners do not know your previous performance – either good or bad. Therefore forget the previous performance – either good or bad – and move on.

Elbow

Make a list of conditions causing painful, locking, stiff, flail and unstable elbow. Painful elbow pathology could be best remembered by its anatomical position – anterior, medial, posterior and lateral. Do not forget the nerves around the elbow while making your list.

Structured oral examination question 1

EXAMINER: A 36-year-old right-hand dominant manual worker, referred by GP with painful right elbow. His elbow radiograph is essentially normal. What would you like to do?

CANDIDATE: Well, I need to assess the patient's elbow . . . after I had asked the history of his pain.

EXAMINER: Pain is on the lateral side, started gradually 3 months ago . . . no history of injury, aggravated by using hammer and was initially relieved by rest. Now it is constant. He has normal range of movements. The point of tenderness is just around the lateral epicondyle.

CANDIDATE: From history and examination I think he has got tennis elbow . . .

EXAMINER: What do you do to confirm the diagnosis?

CANDIDATE: I will test if the pain is reproduced by resisted wrist extension.

EXAMINER: Well, he has more pain on resisted finger extension than wrist extension. Does it make you think more specifically?

CANDIDATE: . . .

EXAMINER: Which tendons are involved in tennis elbow?

CANDIDATE: ECRB . . .

EXAMINER: Can EDC also be affected?

CANDIDATE: . . .

EXAMINER: Well, tell me the pathophysiology of tennis elbow.

CANDIDATE: It is termed angiofibroblastic hyperplasia, which is . . . hyperplasia of the angiofibroblasts . . .

EXAMINER: Do you know any other similar pathology around the elbow?

CANDIDATE: Golfer's elbow, which is tendonitis of the common flexor origin.

EXAMINER: Why do you say tendonitis? What is the difference between tendonitis and tendonosis?

CANDIDATE: . . .

EXAMINER: Going back to the provocation test, if he had tenderness over the lateral proximal forearm on resisted finger extension, what does it tell you?

CANDIDATE: Maybe the disease process is extensive into the common extensor muscle belly.

EXAMINER: We'll move onto the next scenario.

How easy it is to mess up a simple scenario? Is the candidate a classic example for tennis elbow misdiagnosis? Does the candidate deserve anything above a score of 4? Will you approach this subject differently? Think and analyse before looking into the performance of the next candidate.

EXAMINER: A 36-year-old right-hand dominant manual worker, referred by GP with painful right elbow. His elbow radiograph is essentially normal. What would you like to do?

CANDIDATE: I want to know the history of his right elbow pain please.

EXAMINER: It is on the lateral side, started gradually 3 months ago . . . no history of injury, aggravated by using hammer and was initially relieved by rest. Now it is constant.

CANDIDATE: I will proceed with his examination . . . posture of elbow, range of movements especially looking for the lack of full extension and rotation . . . proceed to examine the specific site of tenderness on the lateral aspect.

EXAMINER: He has normal range of movements. The point of tenderness is just around the lateral epicondyle.

CANDIDATE: I would like to know if he has tenderness anterior or posterior to the lateral epicondyle and also any tenderness just distal to the lateral epicondyle.

EXAMINER: What does it tell you?

CANDIDATE: Anterior and distal to lateral epicondyle – ECRB tendonosis. Posterior and distal to lateral epicondyle – EDC tendonosis.

EXAMINER: It is anterior and distal to lateral epicondyle. Tell me the provocation test for ECRB tendonosis.

CANDIDATE: Pain on elbow extension/forearm pronation/fingers flexion/wrist in extension against resistance.

EXAMINER: What is the test for EDC?

CANDIDATE: EDC tendonosis should have pain on elbow extension/forearm pronation/wrist neutral/fingers extension/long finger extension against resistance.

EXAMINER: Does the EDC provocation test tell you anything else?

CANDIDATE: Yes. If EDC provocation test produces pain over EDC origin, it suggests EDC tendonosis. Pain over radial tunnel – radial tunnel syndrome.

EXAMINER: What do you understand by tennis elbow?

CANDIDATE: It is the tendonosis and not tendonitis of ECRB/EDC tendons.

EXAMINER: Tell me the histological appearance of tendonosis.

CANDIDATE: Histologically, there are no acute inflammatory cells. There is granulation-like tissue consisting of immature fibroblasts and disorganized non-functional vascular elements called angiofibroblastic hyperplasia. It is theorized to result from an aborted healing response to repetitive micro-trauma. Pain arises possibly from tissue ischaemia. Electron microscopy has shown that these vascular elements do not have lumina. Essentially the repetitive tensile overload, which exceeds tissue stress tolerance, causes tissue damage. If the tissue damage occurs at a rate which exceeds tissue's ability to heal, this causes tissue degeneration.

EXAMINER: Lastly, do you know any other tendonosis around the elbow other than golfer's elbow?

CANDIDATE: Yes, the posterior tennis elbow, which is triceps tendonosis.

If you were the examiner, what score would you give this candidate?

Structured oral examination question 2

EXAMINER: Look at these radiographs of of the right elbow of a 33-year-old patient and tell me the findings.

CANDIDATE: This plain radiograph of a right elbow shows one loose body in the anterior aspect of the joint.

EXAMINER: What would you like to know if you are allowed to ask only one question?

CANDIDATE: I want to know his presenting symptoms.

EXAMINER: He gets intermittent painful locking symptoms. What is the diagnosis here?

CANDIDATE: Well, he has a loose body in the elbow . . .

EXAMINER: Tell me the conditions which produce loose bodies in a joint.

CANDIDATE: Could be post-traumatic, secondary to osteoarthritis, osteochondritis dissecans (OCD) or synovial chondromatosis.

EXAMINER: Now again . . . What would you like to know if you are allowed one more question?

CANDIDATE: Did he have any injury in the past?

EXAMINER: No, never . . . What is your diagnosis here, keeping in mind that there is only one loose body in the elbow?

CANDIDATE: It could be either secondary to osteoarthritis or OCD and I could rule out post-traumatic cause as he had no injury.

EXAMINER: Can you look at the radiographs again and be more specific? (Showing the X-ray again to the candidate.)

CANDIDATE: I can see only one loose body. There is no calcification in the muscle or capsule.

EXAMINER: What does it tell you?

CANDIDATE: It helps me to rule out myositis ossification and synovial sarcoma.

EXAMINER: I want you to concentrate in the intra-articular pathology and try to narrow down your diagnosis between OCD and osteoarthritis.

CANDIDATE: I would like to know the history of his symptoms and have more investigations to be more specific.

EXAMINER: Well he had unexplained painful elbow which lasted for about 18 months when he was 17 years of age. What do you think is going on with this elbow?

CANDIDATE: It sounds like it may not be osteoarthritis . . . it could be OCD.

EXAMINER: If you had been consulting him at the time of initial presentation 16 years ago, what will be your concern?

CANDIDATE: I would . . .

TRING. . .

Was the candidate a happy customer at the end of this viva? Did he lack the knowledge of this subject of loose bodies? Did he use his knowledge appropriately?

EXAMINER: Look at these radiographs of the right elbow of a 33-year-old patient and tell me the findings.

CANDIDATE: This plain radiograph of a right elbow shows well-maintained joint space with evidence of one loose body in the anterior aspect of the joint.

EXAMINER: What would you like to know if you are allowed to ask only one question?

CANDIDATE: I want to know if this patient had any problem with this elbow in the past.

EXAMINER: Yes, this patient had unexplained painful elbow which lasted for about 18 months when he was 17 years of age. What do you think is going on with this elbow?

CANDIDATE: Well, he could have had osteochondritis dissecans when he was 17, which explains the unexplained pain he had for 18 months and the OCD segment must have separated to form the loose body.

EXAMINER: Do you know a name for OCD of elbow?

CANDIDATE: Yes, Panner's disease.

EXAMINER: If you had consulted him at the time of initial presentation of OCD, what would you have done and why?

CANDIDATE: I would have performed an MRI scan.

EXAMINER: MRI was not widely available then.

CANDIDATE: Well, I would have performed an elbow arthrogram with contrast to assess if the segment had separated from the base. Also the age at which he presented was not in the favourable range . . . that is after the closure of the physis . . . therefore I would have followed him clinically more closely.

EXAMINER: This patient unfortunately had only one X-ray at the start of the presentation and as it did not show any obvious pathology, he was discharged from follow-up. What would you like to do now?

CANDIDATE: I would like to know his presenting symptoms. Has he had any treatment so far and what are his expectations?

EXAMINER: He has had no treatment so far. And can you tell me what would be his presenting symptom?

CANDIDATE: I would expect him to have intermittent painful locking of the elbow.

EXAMINER: Yes, that is his symptom. He wants to have something done to prevent these unexpected painful locking episodes.

CANDIDATE: I would perform an arthroscopic removal of the loose body.

EXAMINER: Can you tell me another cause for one or two loose bodies in a joint?

CANDIDATE: In osteoarthritis the osteophytes can break and present similarly. But the radiograph will show evidence of osteoarthritis.

EXAMINER: If you see multiple loose bodies, what is the diagnosis?

CANDIDATE: Synovial chondromatosis.

This is a good example of using your knowledge appropriately. Compare these two candidates. Candidate 2 has made life easier by being specific and appropriate on every opportunity available.

Structured oral examination question 3

EXAMINER: What do you see in this radiograph of a 67-year-old lady's right elbow? (Figure 6.4.)

CANDIDATE: This radiograph shows extensive erosion of the articular cartilage which has involved both ulnohumeral and radiocapitellar joints. The radial head is dislocated and the elbow articulation is aligned only with ulna and humerus. There is peri-articular osteopenia. There is no subchondral sclerosis or osteophytes.

EXAMINER: What could be the cause?

CANDIDATE: It is characteristic of inflammatory arthropathy and I suspect rheumatoid arthritis. It is a flail elbow.

EXAMINER: Indeed this lady has had RA for the last 34 years. What would you like to do for her?

CANDIDATE: I want to know her presenting symptoms from this elbow. What has changed now to think about doing something about this elbow now? What has been done to this elbow so far? What are her expectations?

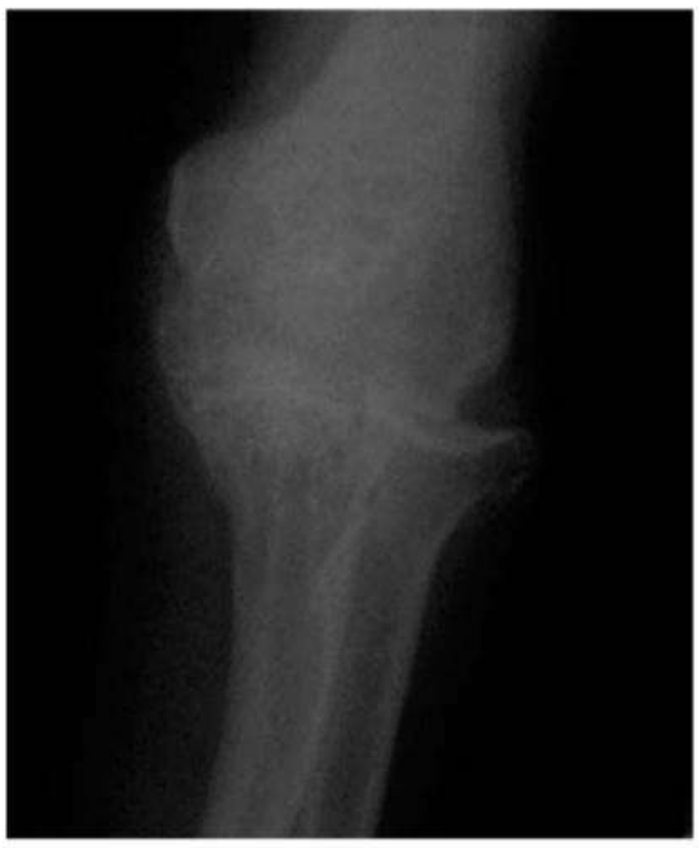


Figure 6.4 Anteroposterior (AP) radiograph right elbow.

EXAMINER: As an RA patient she has many joint problems and recently she is finding lack of strength in her right upper limb to do day-to-day activities. She has had no specific elbow treatments. She wants to do her normal household activities.

CANDIDATE: I would specifically assess her elbow stability and range of movements. And more importantly check her hand function with regards to any tendon ruptures and posterior interosseous nerve function.

EXAMINER: She has no valgus and varus stability but good range of active and passive movements. Hand function is also good. Now how will you differentiate between PIN palsy and extensor tendon rupture?

CANDIDATE: Well if there is no active extension of the fingers at MCP joint and tenodesis test is showing no passive extension of finger at MCP joint on passive flexion of wrist, then the diagnosis is extensor tendon rupture. If the tenodesis test produced passive extension at MCP joint then the diagnosis is PIN palsy. But I will cautiously assess the other tendons supplied by PIN prior to making final diagnosis as in RA patients both can exist together.

EXAMINER: What will be your management plan?

CANDIDATE: It is a multidisciplinary approach with re-consultation with rheumatologists and assessment by occupational therapists. I would initially offer her an elbow brace.

EXAMINER: She comes back after 3 months and says the brace has improved her life quality to some extent but finds it difficult as it gets wet in the kitchen and she still has difficulties in the shower as she could not wear it in the shower.

CANDIDATE: If she is fit for a general anaesthetic, I will do a cemented linked/semi-constrained total elbow replacement for her as this elbow is unstable. I would perform a c-spine X-ray to assess the atlanto-axial joint and obtain an anaesthetic opinion.

EXAMINER: Finally, what happens to juvenile rheumatoid joints?

CANDIDATE: Contrasting to adult RA, juvenile RA produces stiff joints.

Who had the control in this viva? Did this candidate get the questions he played for? Was his technique good? Did he not manage to get a bonus question? Would you be happy if you were the candidate of this scenario? Would you have played it any better? Now the next candidate approaches this table.

EXAMINER: What do you see in this radiograph of a 67-year-old lady's right elbow?

CANDIDATE: This radiograph shows extensive erosion of the articular cartilage which has involved both ulnohumeral and radiocapitellar joints. The radial head is dislocated and the elbow articulation is aligned only with ulna and humerus.

EXAMINER: What could be the cause?

CANDIDATE: It is characteristic of inflammatory arthropathy and I suspect rheumatoid arthritis. It is a flail elbow.

EXAMINER: Indeed this lady has had RA for the last 34 years. What features in the radiograph made you rule out osteoarthritis?

CANDIDATE: In osteoarthritis there will be joint space narrowing, subchondral sclerosis, subchondral cysts and osteophytes. This radiograph does not show these features.

EXAMINER: What is the bone quality here?

CANDIDATE: . . . The bone appears to be osteopenic . . . could be disuse from pain or the disease process itself.

EXAMINER: Now, what would you do for her?

CANDIDATE: I need to know the history of presenting complaints and I would examine the elbow.

EXAMINER: She recently finds her right upper limb weak affecting her day-to-day activities. In the examination there is valgus/varus instability.

CANDIDATE: It is an unstable elbow from advanced RA. Therefore I would do a total elbow replacement for her.

EXAMINER: Is there anything you would consider prior to surgery?

CANDIDATE: Well, I can try a splint if she is willing to try . . .

EXAMINER: She comes back after 3 months and says the brace has improved her life quality to some extent but finds it difficult as it gets wet in the kitchen and she still has difficulties in the shower as she could not wear it in the shower.

CANDIDATE: Then I will proceed with the total elbow replacement.

EXAMINER: Which nerve specifically would you like to assess in the RA elbow especially prior to total elbow replacement?

CANDIDATE: Posterior interosseous nerve as it can be affected by the synovial swelling/dislocation of the radiocapitellar joint.

EXAMINER: What would be the findings if she has PIN palsy?

CANDIDATE: There will be no active extension of the fingers at the level of MCP joints.

EXAMINER: Do you know any other cause for the inability to extend MCP joints?

CANDIDATE: Yes, progressive rupture of extensor tendons called Vaughn–Jackson syndrome.

EXAMINER: Is there any concern regarding this RA patient undergoing general anaesthesia?

CANDIDATE: These patients can have lung fibrosis . . . apart from this, yes . . . of course I will perform a c-spine X-ray to see the stability of atlanto-axial joint.

EXAMINER: Thank you.

Did he not answer all the questions? Did he not possess the knowledge of the subject? But, did he gain the control of this viva? Did he ever lead the examiner to the next question? Or did the examiner have to guide him with leading questions? Would he ever get a score of 8?

Structured oral examination question 4

EXAMINER: Good morning. Here are the radiographs of a right-hand dominant 43-year-old man's right elbow. Tell me the findings. (Figure 6.5.)

CANDIDATE: Good morning. These radiographs show narrowing of joint space on both ulnohumeral and radiocapitellar joints with subchondral sclerosis and cysts and medial, anterior and posterior osteophytes suggesting osteoarthritis. Has he had any previous injury to this elbow?

EXAMINER: Well he had a dislocation of this elbow 8 years ago which was reduced in A&E and as he improved to full function in 8 weeks he was discharged from the fracture clinic. Now over the last 3 years he has got problems with this elbow. What would you advise for this patient?

CANDIDATE: I want to know his present symptoms. How much does it affect his job? What are the treatments he has had so far? And what is his expectation?

EXAMINER: This elbow is affecting his job as he has got restricted movements – flexion extension from 50° to 110° and supination is only to 40°. He had a few intra-articular injections by his GP. He wants to have more movement in the elbow.

CANDIDATE: He has got post-dislocation osteoarthritis with stiffness. He is not presenting with pain as a main symptom. Therefore I would like to perform an arthroscopic debridement/arthrolysis of his elbow.

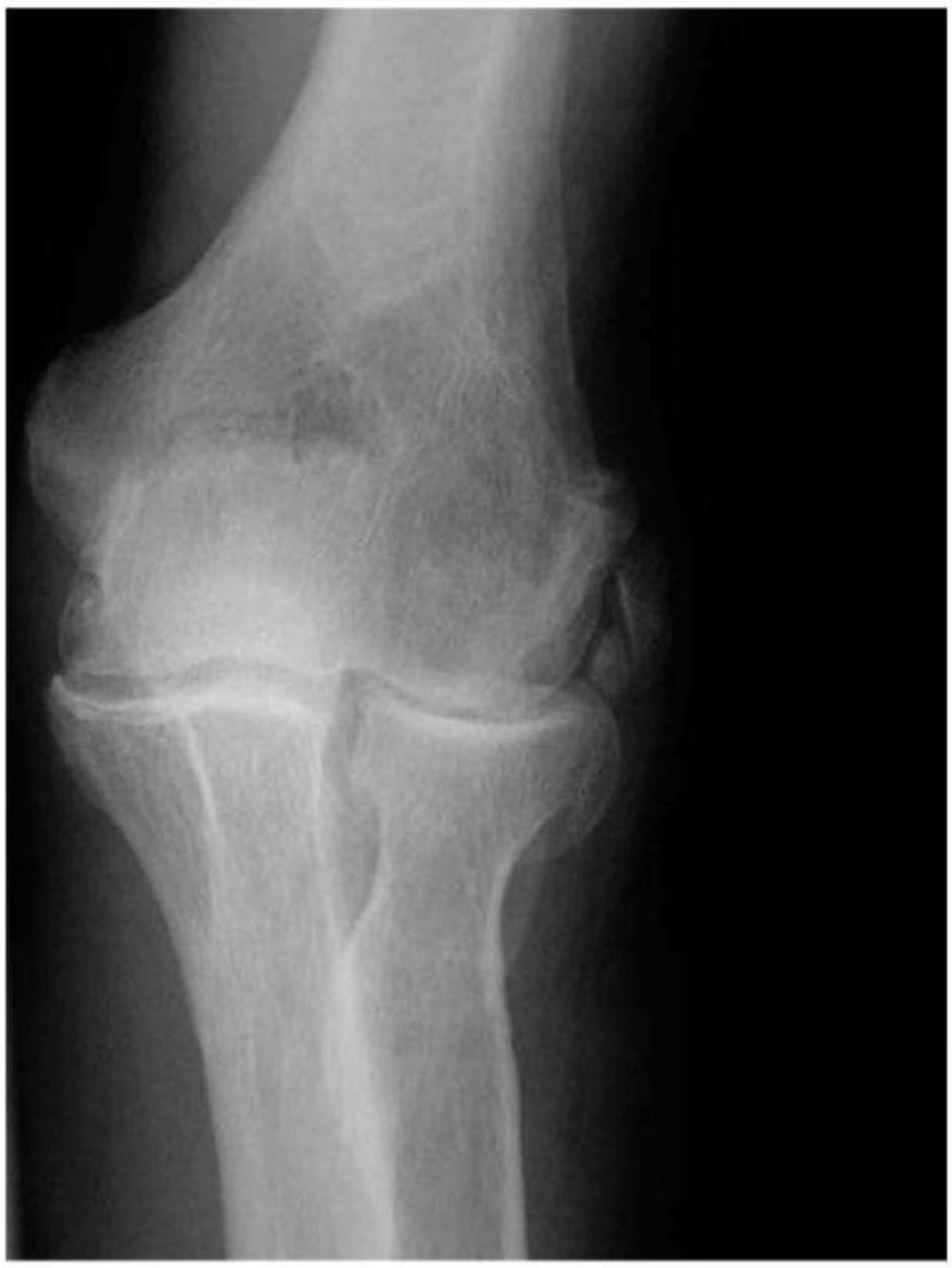


Figure 6.5 Anteroposterior (AP) radiograph right elbow.

EXAMINER: Can you show me the arthroscopic portals in this elbow picture?

CANDIDATE: (Marking and talking to the examiner.)

Direct lateral portal:	At the centre of a triangle defined by the lateral epicondyle, the radial head and the olecranon. This is frequently used as the initial entry portal to inflate the joint with saline.
Anterolateral portal:	1 cm distal and 1 cm anterior to the lateral epicondyle, between the radial head and the capitellum. This gives good access to the anterior aspect of the joint.
Anteromedial portal:	2 cm distal and 2 cm anterior to the medial epicondyle. This is often created using an 'inside out' technique by cutting down onto the tip of the arthroscope inserted using the anterolateral portal.
Proximal medial portal:	2 cm proximal to the medial epicondyle along the anterior surface of the humerus towards the radial head.
Direct posterior portal:	1.5 cm proximal to the tip of the olecranon. Access to olecranon fossa.
Posterolateral portal:	Access to radiocapitellar joint.

EXAMINER: Is the benefit of the debridement permanent?

CANDIDATE: No, it is not . . . and varies between individuals.

EXAMINER: Patient wants to know if there is any procedure which can provide long-lasting benefit.

CANDIDATE: The longer-lasting result can be achieved by a total elbow replacement . . . But as this patient is only 43 and he is a manual worker and his dominant elbow is affected with osteoarthritis, I would not advise a total elbow replacement at this moment as the TERs do not have long life expectancy in young osteoarthritic patients.

TRING . . .

Would you handle this scenario differently? How much will you score this candidate? Was his knowledge sufficient and well presented? Now a confident-looking candidate approaches the table.

EXAMINER: Good morning. Here are the radiographs of a right-hand dominant 43-year-old man's right elbow. Tell me the findings.

CANDIDATE: The radiographs show advanced osteoarthritis of his dominant elbow.

EXAMINER: Correct. What would be your advice to this patient?

CANDIDATE: It depends on if he has pain, stiffness, difficulty with his job and also depends on his expectations.

EXAMINER: Pain is not a main issue here. This elbow is affecting his job as he has got restricted movements – flexion extension from 50° to 110° and supination is only to 40°. He wants to have more movement in the elbow.

CANDIDATE: I will initially inject his elbow with steroids and send him for stretching physiotherapy.

EXAMINER: Patient has had a few injections already and also physiotherapy from his GP and therefore he prefers to have a more definitive procedure.

CANDIDATE: Well, if the injections have been tried without any success, I would advise a total elbow replacement.

EXAMINER: Is there anything you could offer prior to TER?

CANDIDATE: (Suddenly losing confidence.) Probably an attempt at manipulation under anaesthesia . . .

EXAMINER: Is MUA and passive stretching of a stiff elbow good advice?

CANDIDATE: . . . perhaps not . . . as there is a small risk of myositis ossification.

EXAMINER: In the last 30 years . . . the number of implanted TERs is in decline. Why?

CANDIDATE: . . .

EXAMINER: Well, 20 to 30 years ago the TER was commonly used for which group of patients?

CANDIDATE: Rheumatoid patients, and it has declined as rheumatoid patients are better treated now and we do not see advanced joint pathology in this group of patients.

EXAMINER: What is the clinical finding in an advanced RA elbow?

CANDIDATE: Arthritis affects the entire joint, the ligament stability is also lost as RA is primarily a soft tissue problem and the radial head dislocates and the elbow becomes flail.

EXAMINER: Have you seen flail RA elbow recently?

CANDIDATE: No, I haven't seen any which have progressed to radial head dislocations . . . instead the appearance we see now is more like osteoarthritis.

EXAMINER: Does this modification of disease pathology have anything to do with declining number of implanted TER?

CANDIDATE: Yes, the TER failed earlier in this group.

EXAMINER: This is because we are treating stable disease-modified osteoarthritic RA elbows, with the implant designed to treat flail elbows.

CANDIDATE: . . .

EXAMINER: Would you like to offer anything else prior to TER for this young manual worker?

CANDIDATE: An arthroscopic washout?

EXAMINER: Is there any . . .

TRING . . .

Did the confident start last long? Was the knowledge adequate to handle this scenario? Would you like to be this candidate on the day of exam?

Structured oral examination question 5

EXAMINER: I have a problem with my left elbow. Proceed.

CANDIDATE: Well, I want to know your age, hand dominance, your occupation and the nature of your problem please.

EXAMINER: I am 47, a right-hand dominant mechanic and in certain positions my elbow pops which is painful.

CANDIDATE: Is the popping sensation on the inner side or outer side of your elbow?

EXAMINER: The outer side . . . yes, my thumb side.

CANDIDATE: Did you ever have any problem in your elbow as a child?

EXAMINER: I had problems as a child in my right elbow, but now my right side is fine. My left side, although I did not have any problem as a child, 3 years ago I had a simple dislocation.

CANDIDATE: What problem did you have on the right side?

EXAMINER: My older sister pulled me by my right hand and my elbow became painful and the doctor had manipulated my elbow and told my parents not to let anyone pull me by my hand. And he said it was a pulled elbow . . . where the radial head pops out.

CANDIDATE: I want to check if you have general joint laxity.

EXAMINER: No I am rather stiff. What do you think is wrong with my left elbow?

CANDIDATE: I think radial head dislocations . . . probably secondary to annular ligament insufficiency secondary to the dislocation. In what position do you get this popping sensation?

EXAMINER: Whenever I push myself off the chair with my arm.

CANDIDATE: I would like to perform an X-ray of your elbow to assess the radial head.

EXAMINER: The X-ray is normal. Can you tell me about the ligaments around the elbow?

CANDIDATE: Sure. There are two main groups of ligaments, medial and lateral collateral ligaments. MCL has three bundles: anterior, posterior and transverse bands. LCL has lateral ulnar collateral ligament (LUCL), annular ligament, radial collateral ligament and accessory collateral ligament.

EXAMINER: Have you heard of postero-lateral rotatory instability of the elbow?

CANDIDATE: . . .

Did the candidate reach the diagnosis? Did he understand the clues given by the examiner? The next candidate arrives.

EXAMINER: I have a problem with my left elbow. Proceed.

CANDIDATE: Well, I want to know your age, hand dominance, your occupation and the nature of your problem please.

EXAMINER: I am 47, a right-hand dominant mechanic and in certain positions my elbow pops with pain.

CANDIDATE: Have you ever injured your left elbow in the past? And in what position are you feeling the popping sensation in the elbow?

EXAMINER: Well, I had a simple dislocation of my left elbow 3 years ago which was reduced in A&E. Now whenever I push myself off a chair using my arm I get this sensation.

CANDIDATE: I would like to have a quick assessment of your elbow.

EXAMINER: What would you like to test?

CANDIDATE: I want to perform the pivot-shift test to assess the lateral ulnar collateral ligament.

EXAMINER: If the pivot-shift test is positive, what is your diagnosis?

CANDIDATE: Postero-lateral rotatory instability of the left elbow.

EXAMINER: I had been told that I had 'pulled elbow' on the other side as a child. Could this be the same?

CANDIDATE: No, usually the pulled elbow settles as the child grows and you had a definite injury to the left elbow.

EXAMINER: What could you do to me to prevent these unpleasant episodes?

CANDIDATE: I need to perform an MRI scan to confirm injury to LUCL and to see if the injury to the ligament is intra-substance or from the origin to decide on the treatment. And did you have any recent X-rays?

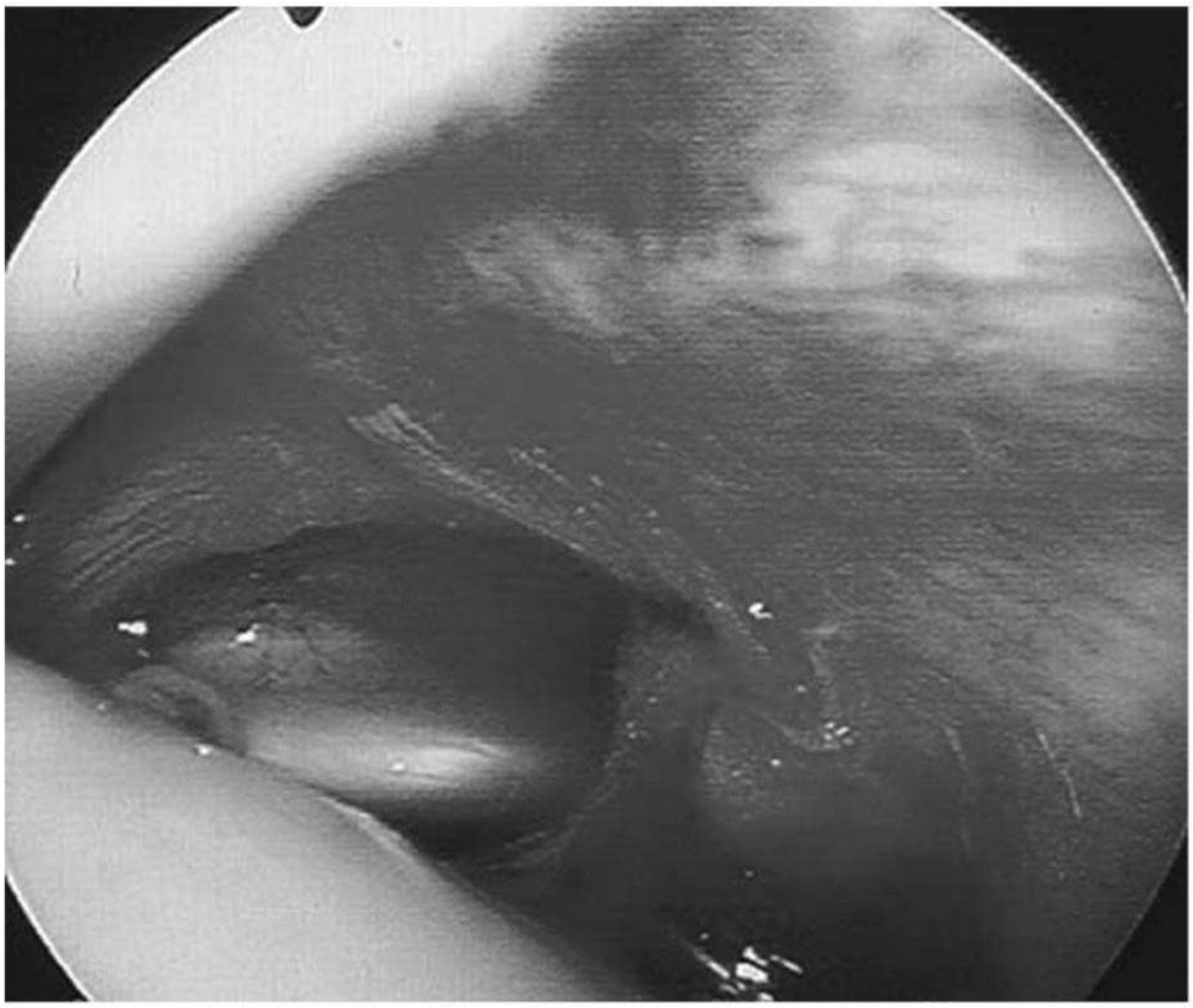
EXAMINER: My X-rays were normal. If the MRI scan shows injury to the LUCL, how will you manage this problem?

CANDIDATE: If the LUCL is avulsed from the origin or insertion and the ligament itself is healthy, it could be re-attached to the bone using bone anchors. It may not be possible in your case as the injury was 3 years ago. My main inclination is to reconstruct the LUCL using palmaris longus tendon or triceps fascia.

Did this candidate manage to please the examiner? Which candidate you would prefer to treat your elbow?

The examiner's aim is all about finding out, can I let this candidate be my consultant. As you would like to win the patient's confidence while consulting in the clinics, it is vital to win the examiner's confidence in each and every scenario by showing adequate knowledge expressed with correct technique.

Viva 75



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What do you understand by the term 'frozen shoulder'?

What are the classical stages described?

What are the factors associated with this condition?

How would you manage this condition?

Are you aware of any operative procedures for this condition?

What are the typical findings during arthroscopy?

What do you understand by the term 'frozen shoulder'?

Frozen shoulder is the term used to describe the condition in which there is gradual onset of pain in the shoulder followed by stiffness.

What are the classical stages described?

The condition is typically characterized by three stages:

- Stage 1 is the painful phase which usually lasts 2–9 months. Patients usually complain of pain at night
- Stage 2 is the phase of stiffness and usually lasts 4–12 months. All movements are usually affected
- Stage 3 is the stage of thawing which also usually lasts 4–12 months. The stages overlap each other and are not discrete

What are the factors associated with this condition?

Factors associated with frozen shoulder are diabetes mellitus, trauma, chest disease, rotator cuff tear, hyperlipidaemia, and thyroid and autoimmune disease.

How would you manage this condition?

I would explain the diagnosis and natural history of frozen shoulder. I would offer an intra-articular steroid injection and analgesia, particularly in the painful phase. I would also refer the patient for physiotherapy. If symptoms fail to resolve, I would consider manipulation under anaesthesia.

Are you aware of any operative procedures for this condition?

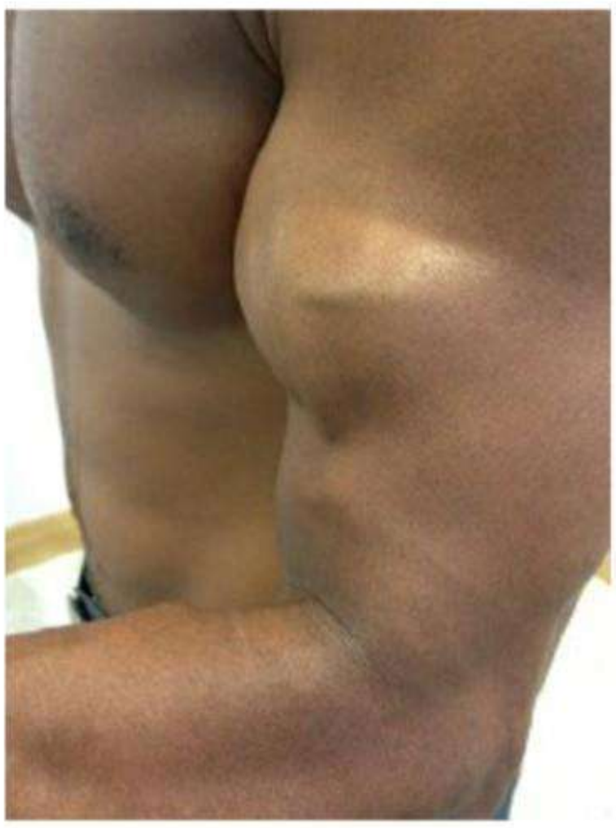
Manipulation under anaesthesia by an experienced shoulder surgeon or arthroscopic capsular release is sometimes necessary for resistant cases.

What are the typical findings during arthroscopy?

The joint feels tight and the rotator interval is narrowed. Marked synovial injection is seen in the rotator interval.

Shoulder viva

1. Clinical picture of tear of the distal biceps tendon. Please outline its causes and management? What is the prognosis of non operative and operative management? Please describe the principles of non operative and operative management and the relevant scientific evidence



Causes

Two theories

1. Reduced blood supply at zone two of tendon
2. Mechanical impingement in pronation
- extension force in flexed elbow causes rupture

Clinical features

More common in men than women
Middle age: during lifting or rotation of forearm

Acutely, bruising antecubital fossa and tenderness

1. Reduced function – weakness particularly supination, flexion less marked
2. Loss of contour
3. Biceps squeeze test – less supination

Management
Imaging:Radiographs usually normal, ultrasound scan (operator dependent) and MRI

Non operative

-Acceptable outcome in less active patients
- Cosmetic deformity is not an indication for surgery
- cramping pain with prolonged use is rare
-reduced flexion strength of 15 percentage and supination strength of 40 percentage

Surgical

A. Anatomic versus non anatomic

Anatomic- re attach to radial tuberosity
Non anatomic – Attach to brachialis (salvage procedure with chronic rupture, no increase in supination strength, can help with pain.
Late reconstruction with hamstrings and palmaris longus have been described

B. Two incision vs one incision

Historically a single incision with drill holes and sutures was used, this carried a high risk to the Posterior Interosseus Nerve. Two incision approach of Boyd and Anderson was used giving less chance of nerve injury, but increased incidence of heterotrophic ossification and radio-ulnar synostosis

The use of suture anchors has allowed a return to a single incision with reduced nerve risk with similar or superior outcomes reported.
Ref: McKee et al JSES 2005

2. Radiograph of rotator cuff arthropathy. How will you manage? What is your method of rotator cuff repair, and what is the scientific evidence behind it?

Describe radiograph- superior migration/glenoid erosion/Osteoarthritis

Several options to classify this:
Most straightforward FAVARD classification but more common HAMADA classification

FAVARD: 1 to 3

1) Superior migration of the humeral head and changes visible on the inferior surface of the acromion
2) Central gleno-humeral space narrowing
3) Lysis of the humeral head or the acromion

HAMADA

Grade 1 to 5

Management
-Arthroplasty (hemi/ reverse?)

Pre operative factors

1. Cuff deficiency
2. Bone stock (assess with pre-operative CT scan)
3. Wear pattern
4. Soft tissue status

Crucial concept compensation for tear:
About 50% of patients with arthritis and massive cuff tear can elevate limb: acceptable outcome with lower risk with hemi or re-surfacing.

50% of patients are unable to elevate the limb with a cuff tear and arthritis. In these patients, consider reverse shoulder arthroplasty

Principles of reverse shoulder arthroplasty

-Medialises gleno humeral joint center of rotation- lengthen glenoid lever arm- increases power
-Humeral head lowered-increases tension on deltoid- increases power

Disadvantages with reverse
Complication rates are high (up to 50% Werner et al)
Limited survival and difficult to revise (5 -30% at 5 years)
In Europe, reverse shoulder arthroplasty is used more commonly that in North America

Problems associated with reverse shoulder arthroplasty
1. Instability- dislocation

2. Polyethylene wear

2. Polyethylene wear
3. Lack of external rotation
4. Scapular notching
5. Are there long term results? Plenty! Good gains reported in pain and function reflected in Constant score and Oxford shoulder score

Caution
1) Using reverse in acute trauma

2) Do not implant a total shoulder arthroplasty if the rotator cuff is torn, unless the tear can be repaired

Rotator cuff tears

Common pathology increase with age: approximately 30% has Partial thickness tear or full thickness tear at 70 years

Genetic basis: siblings of patients with symptomatic tears more than twice as likely to have a tear and four times to have symptoms than controls

Not all torn cuffs need to be repaired

Investigations:
Radiographs: in the axial view, beware of os acromiale
Ultrasound scan good for tear identification, role in partial tears not proven
MRI Tear size, retraction, repairability and fatty substitution
(Goutallier 0-4, first described in CT, validated for MRI)
0 = normal,
1 = fatty streaks,
2 = more muscle than fat,
3= same muscle and fat,
4= more fat than muscle
Higher grades associated with worse surgical outcome

Management

Conservative treatment: injection and physiotherapy

Indications for cuff repair

Symptomatic tears failing to respond to conservative treatment in appropriate patients. Significant intervention and rehabilitation prolonged (six weeks sling, six months to recover). Not without risk.

1. Age – caution after 70
2. Proven tear – Ultrasound scan/MRI

3. Cubitus varus. Shown radiograph. Causes. Complications. Methods of ulnar nerve transposition and evidence

3. Cubitus varus. Shown radiograph. Causes. Complications. Methods of ulnar nerve transposition and evidence

Causes

Uncommon but well recognised complication of fractures, particularly paediatric distal humeral fractures.

Late presentation with ulnar nerve symptoms of variable severity.

Associated with varus angulation

Treatment options

Transposition ulnar nerve
Distal humeral osteotomy

Choice of transposition: submuscular or subcutaneaus
Surgeon preference rather than scientific evidence

5. Clinical picture of winging of scapula. What are the causes? What are the principles of management?

Winging scapula may result in severe shoulder dysfunction

Causes
1-Trapezius palsy
2-Serratus anterior palsy
3-Sprengel deformity (not truly winging)
4-Fascio-scapular-humeral dystrophy (FSHD)

1. Trapezius Palsy
Commonly a result of injury to the spinal accessory nerve (iatrogenic, can be intentional with neck clearance)
Difficulty in abducting arm without pain
Treatment: physiotherapy and expectant attitude
If symptoms persist, consider EMG and neurolysis
In chronic cases, consider muscle transfer (Eden –Lange procedure)

2. Serratus Anterior Palsy
The long thoracic nerve (C5, C6, C7) is involved
Causes: viral illness, recumbency, Pregnency, idiopathic
Recovery after closed injury: usually 1 year, but it can take up to 3 years
Treatment: physiotherapy
Braces often poorly tolerated
Surgery: transfer of sternal head of pectoralis major to the inferior pole scapula with fascia lata.

3. Sprengel's deformity, the scapula is high and hypoplastic. It produces variable disability, and surgery is not usually needed
It can be associated with other congenital abnormalities (eg. Klippel–Feil syndrome)

4. Facio-scapular-humeral dystrophy (FSHD)
Autosomal dominant
Unilateral presentation in teens, progressing to bilateral involvement
Surgery: scapulothoracic fusion

5. Sport :

A. Oral :

Lateral Structures of Knee	
Layer I	Iliotibial tract, biceps femoris
	Common peroneal nerve lies between layer I and II
Layer 2	Patellar retinaculum
Layer 3	Superficial: LCL, fabellofibular ligament, ALL
	Lateral geniculate artery runs between deep and superficial layer
	Deep: Arcuate ligament, coronary ligament, popliteus tendon, popliteofibular ligament, capsule

Medial Structures of Knee	
Layer I	Sartorius and fascia (patellar retinaculum)
gracilis, semitendinosis, and saphenous nerve run between layer 1 and 2	
Layer 2	Semimembranosus, superficial MCL, MPFL, posterior oblique ligament ?
Layer 3	Deep MCL, capsule, coronary ligament

Outerbridge Arthroscopic Grading System	
Grade 0	Normal cartilage
Grade I	Softening and swelling
Grade II	Superficial fissures
Grade III	Deep fissures, without exposed bone
Grade IV	Exposed subchondral bone

ICRS (International Cartilage Repair Society) Grading System	
Grade 0	Normal cartilage
Grade 1	Nearly normal (superficial lesions)
Grade 2	Abnormal (lesions extend < 50% of cartilage depth)
Grade 3	Severely abnormal (>50% of cartilage depth)
Grade 4	Severely abnormal (through the subchondral bone)

Classification of articular cartilage lesions

3 Categories of surgical options

1. Palliative (arthroscopic débridement and lavage).
2. Reparative (marrow stimulation techniques).
3. Restorative (osteochondral grafting and autologous chondrocyte implantation).

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

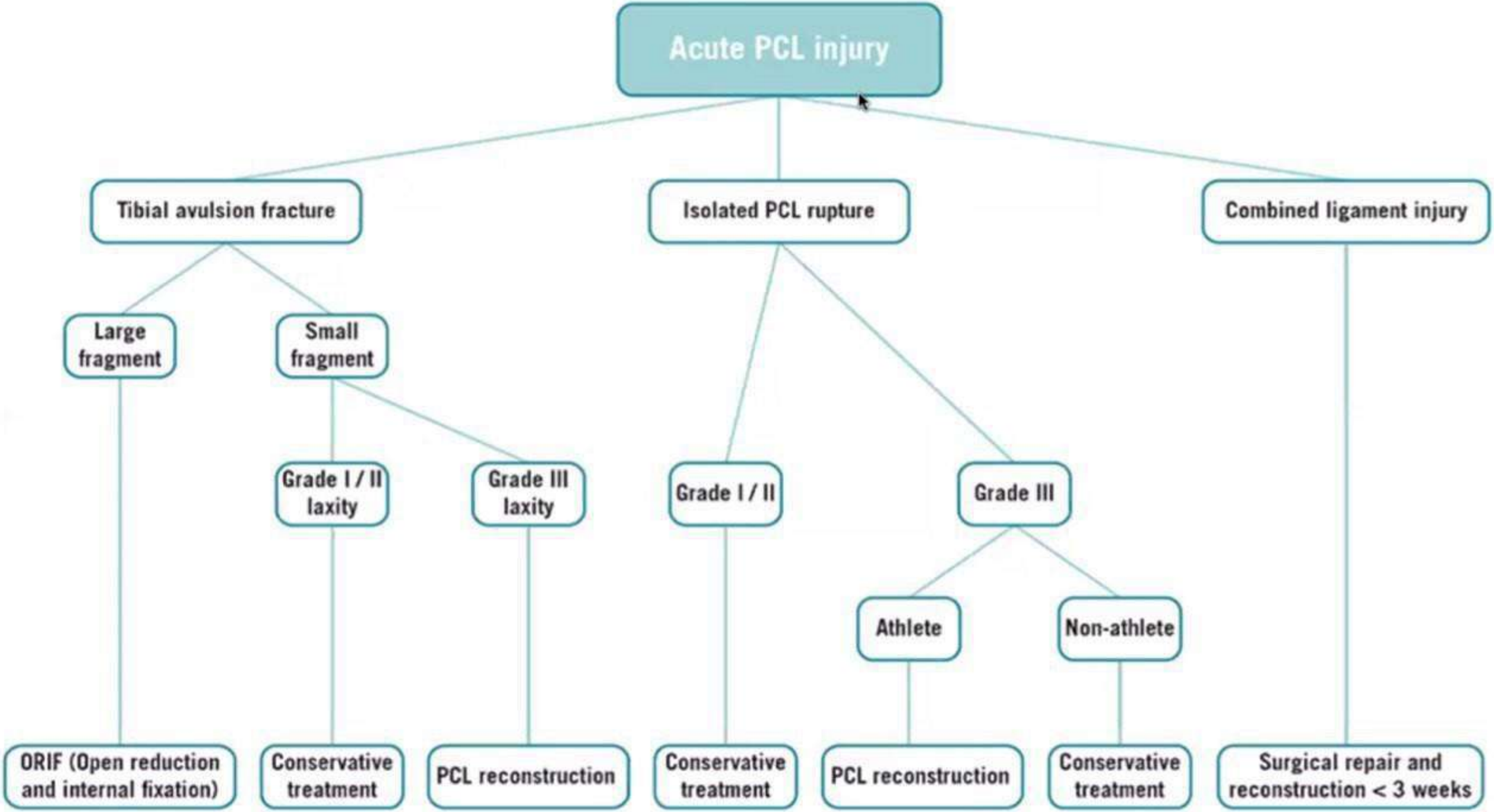
   Walking with valgus thrust.

5. Genetic factors such as connective tissue disorder syndromes.

Risk factors include:

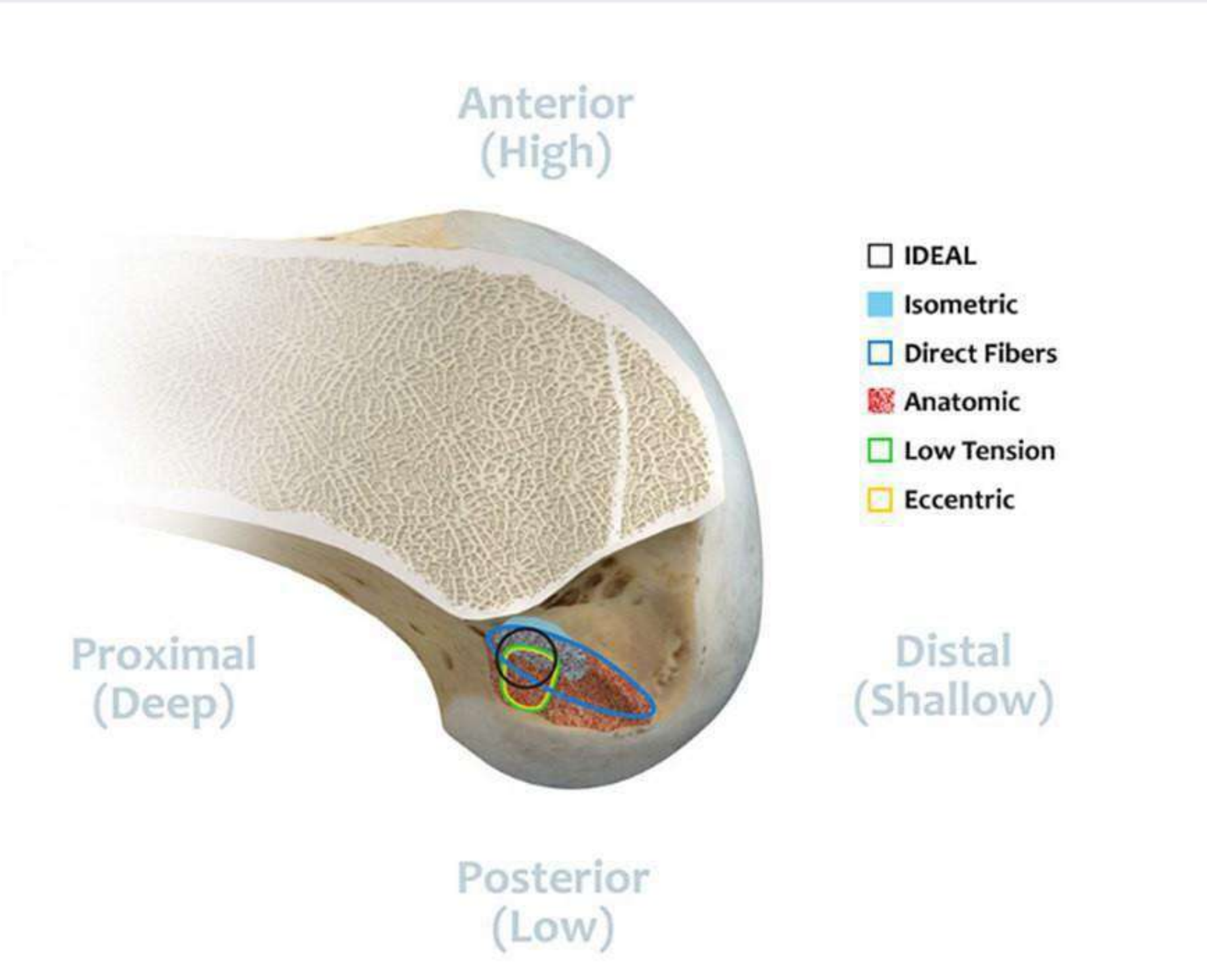
1. Female,
2. Q angle $> 20°$ (normal is 10° in boys and 15° in girls),
3. Genu valgus,
4. Rotational abnormalities, such as increased femoral anteversion or external tibial torsion,
5. Patella alta (Figure 4.17),
6. A shallow patella-femoral sulcus angle (ABC $> 144°$ is abnormal) (Figure 4.17),
7. 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.)

Berndt and Harty Radiographic Classification	
Stage 1	• Small area of subchondral compression
Stage 2	• Partial fragment detachment.
Stage 3	• Complete fragment detachment but not displaced.
Stage 4	• Displaced fragment.


Acute PCL injury
Tibial avulsion fracture
Isolated PCL rupture
Combined ligament injury
Large fragment
Small fragment
Grade I / II laxity
Grade III laxity
Grade I / II
Grade III
Athlete
Non-athlete
ORIF (Open reduction and internal fixation)
Conservative treatment
PCL reconstruction
Conservative treatment
PCL reconstruction
Conservative treatment
Surgical repair and reconstruction < 3 weeks

Anatomic	• BMI • Impingement on intercondylar notch, smaller notch • Smaller ACL • Hypermobility/joint laxity • Previous ACL injury
Biomechanical	• Increased knee valgus and extension during landing • Decreased knee and hip flexion • Fatigue resistance
Neuromuscular	• Lower hamstring:quad ratio (more quads dominant) • Lower hamstring recruitment • Weaker core stability
Hormonal	• Preovulatory phase of menses (hormones affect coordination) • Females on OCP not as affected during this phase
Genetic	• Collagen production (COL5A1 gene linked to decreased risk of injury in women)

This position is Isometric, in the Direct Fibers, Equidistant, Eccentric, Anatomic, and Low Tension. You have selected the I.D.E.A.L. femoral tunnel location.



IDEAL Position :

I : **ISOMETRIC**: Graft will have a length tension relationship that is similar to the native ACL

D : **DIRECT FIBERS**: Fibers that have direct insertion on the footprint and fibers that are closest to the ridge.

E : **ECCENTRIC**: Anterior or higher in the footprint and in the anterior-medial portion of the ACL

A : **ANATOMIC**: Fibers within the bounds of the native ACL graft, which is anterior (higher) than the indirect fibers.

L :

LOW TENSION: Tension of the native ACL should match the low tension-flexion pattern of the native ACL