Section

I

Short Cases and Viva Voce

1. **Trauma and its Complications:** Cubitus varus, cubitus valgus, malunion, myositis ossificans, nerve injury module—basics of nerve injury, wrist drop (radial and posterior interosseous nerve injury), clawhand (median nerve injury, ulnar nerve injury), footdrop non-union, reflex sympathetic dystrophy, complex regional pain syndrome, stiff joint, Volkmann's ischemic contracture
2. **Congenital Musculoskeletal Conditions:** Congenital talipes equinus (CTEV), developmental dysplasia of the hip (DDH), spina bifida
3. **Bone and Joint Infections:** Chronic osteomyelitis, TB of the knee
4. **Arthritis:** OA knee, rheumatoid arthritis
5. **Painful Hip:** Transient synovitis, tuberculosis of the hip, Perthes' disease
6. **Metabolic Disease:** Rickets, osteomalacia
7. **Bone Tumours:** Ewing's sarcoma, osteosarcoma, solitary exostosis (osteochondroma), giant cell tumor
8. **Low Back Pain:** Intervertebral disc prolapse, spondylolysis/spondylolisthesis, lumbar canal stenosis, ankylosing spondylitis
9. **Pott's Spine**
10. **Painful Shoulder:** Frozen shoulder, rotator cuff tendinitis, rotator cuff tear, glenohumeral arthritis
11. **Spine Injury its Complications and Rehabilitation**
12. **Unstable Knee (ligament injury of the knee):** ACL tear, PCL tear, meniscal injury, patella dislocation
13. **Miscellaneous Conditions:** Carpal tunnel syndrome, tendo Achillis rupture, lateral epicondylitis, medial epicondylitis, De Quervain's tenosynovitis
14. **Case Taking Format**

Chapter

1

Trauma and its Complications

ELBOW DEFORMITY (CUBITUS VARUS, CUBITUS VALGUS)

Common Presentation of Patient with Cubitus Varus of Elbow

Case summary: An 8-year-old boy presented with a deformity of his left elbow since two years. Two years back, there was history of fall on outstretched hand followed by a fracture around the elbow. He was taken to a local hospital where an above elbow cast was applied. Three week later, cast was removed. He underwent regular physiotherapy which led to restoration of movement and function of his left elbow. He regained complete movements only later to observe that his elbow was progressively showing a deformity with forearm moving closer to the body. Local examination revealed cubitus varus deformity of left elbow. There was bony irregularity felt over the distal humerus. Three bony point relationship was intact. Flexion range of motion was 0–160° with no limb length discrepancy. Neurovascular examination, and the examination of shoulder and wrist-hand was normal.

Q. What is the clinical diagnosis?

The clinical diagnosis is malunited supracondylar fracture with cubitus varus deformity of left elbow (Fig. 1.1).

Q. Why do you say so?

It is because of following reasons.

1. History of trauma with **fall on outstretched hand**
2. Presence of cubitus varus deformity
3. Intact three bony point (medial and lateral epicondyle, olecranon) relationship
4. Bony irregularity over the distal humerus (indicates a healed fracture in that region)

Note: Carrying angle is the inner angle formed between the axis of humerus and forearm (Fig. 1.1). Carrying angle/Cubitus varus/Cubitus valgus is assessed with elbow in complete extension. If elbow cannot be completely extended due to flexion deformity, the exact assessment of carrying angle is not possible, and its assessment should be avoided.

Fig. 1.1: Clinical picture of a child with cubitus varus deformity of left elbow. On the right upper limb, line HE represents humerus-elbow longitudinal axis and line EF represents elbow-forearm longitudinal axis. Normal carrying angle is represented by A which is an inner angle between line HE and EF

Q. What is the normal alignment of the elbow?

Normal alignment of the elbow is "cubitus valgus", i.e. the forearm is drifted outwards with respect to the midline (or arm). This is also known as carrying angle (normal carrying angle in males is 7° and females is 15°).

Q. Why carrying angle is greater in females?

It is greater because of broader pelvis in females.

Q. How will you comment upon the cubitus varus, if there is flexion deformity of elbow?

Cubitus varus or valgus **should not be commented** upon, **if there is flexion deformity** of elbow as varus or valgus tends to become less obvious with increasing flexion of the elbow joint.

Q. What is the methodology to palpate three bony points (lateral epicondyle, medial epicondyle and tip of olecranon)?

For lateral and medial epicondyles, first supracondylar ridge is palpated which is situated in the supracondylar region over medial and lateral side of the elbow above the two epicondyles. As the ridge is traced downwards, the most prominent point palpated over these ridges is medial and lateral epicondyle respectively.

For palpation of tip of the olecranon, first posterior ulnar border is palpated and then traced upwards. The most prominent point situated over the olecranon is the tip.

Q. What is the normal relation between these points?

In a normal elbow, these three bony points form a straight line in an extended elbow and form a triangle in 90° flexed elbow. Pronated/supinated forearm does not make

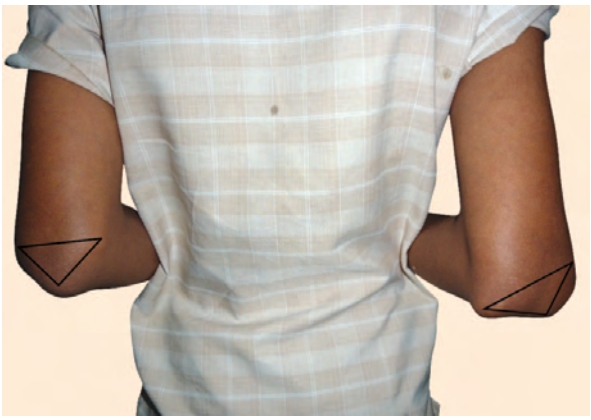


Fig. 1.2: Clinical photograph showing altered three bony point relation over the right elbow as compared to left elbow

any difference. Figure 1.2 shows altered relationship between the two triangles formed on right and left elbow in flexion.

Note: Remember; the type of triangle, i.e. scalene/isosceles is not important. What is important is that is it comparable with normal side or not!

Q. Which conditions around the elbow could result in disturbed three bony point relation?

a. Posterior dislocation of elbow
b. Intercondylar fracture of humerus
c. Lateral or medial condyle fracture of elbow
d. Fractures of medial or lateral epicondyle.

Q. Which condition other than malunited supracondylar fracture humerus could result in cubitus varus deformity of the elbow?

Trochlear avascular necrosis.

Q. Which nerve is more prone for injury in supracondylar fracture?

Anterior interosseous nerve is most commonly affected in extension type of supracondylar fracture followed by radial and ulnar nerve whereas **ulnar nerve** is most commonly **affected in flexion** type of supracondylar fracture (#).

Q. What is the difference between anterior interosseous nerve (AIN) and median nerve?

AIN is a branch of median nerve on volar aspect which supplies deeper muscles of forearm, viz. flexor pollicis longus (FPL), pronator quadratus (PQ), and the radial two tendons of flexor digitorum profundus (FDP).

Commonest fracture around the elbow joint in children is supracondylar fracture

Q. What are the complications of supracondylar fracture?

Acute

1. Compartment syndrome or Volkmann's ischemia
2. Anterior interosseous nerve or radial nerve injury (extension type) and ulnar nerve (flexion type)
3. Brachial artery injury

Chronic

1. Volkmann's ischemic contracture
2. Myositis ossificans
3. Malunion leading to cubitus varus deformity

Q. How will you treat this patient?

I will ask for X-ray of the elbow, AP and lateral view

Q. What will be the X-ray finding?

X-ray will reveal

a. Healed supracondylar fracture

b. Cubitus varus deformity

Q. What is the treatment of the cubitus varus deformity?

Since this deformity is **non-progressive** for last 6 months, corrective osteotomy should be performed.

Q. What if the deformity would have been progressive?

If the deformity is progressive; it is important to wait till the deformity ceases to progress as if osteotomy is performed in progressive stage, deformity can later recur or correction would be lost.

Q. Which osteotomy you will perform?

Modified French osteotomy. It is a lateral closing wedge osteotomy performed in the supracondylar region of elbow (Fig. 1.3).

Q. Is there a possibility that the three bony point relation can get disturbed in a malunited supracondylar fracture too?

Yes, it is possible. Sometimes during fall over elbow, there can be

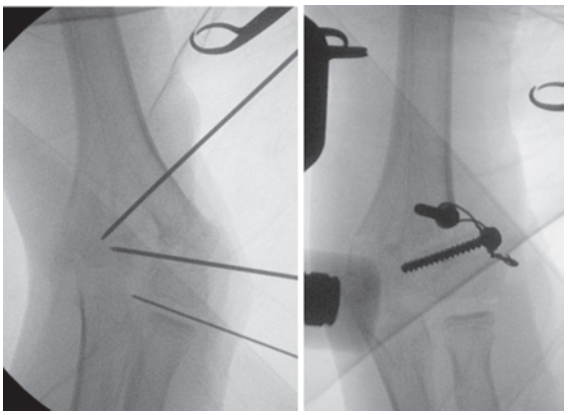


Fig. 1.3: X-ray of a varus elbow (left image) undergoes corrective osteotomy and fixation by screws and tension band wiring (right image)

injury to distal humerus physis/growth plate which could result in disturbed relation between three points.

Q. Mention a common condition in which cubitus valgus deformity is observed?

Cubitus valgus is observed in **non-union of lateral condyle fracture** of humerus.

Q. What is lateral condyle?

Lateral condyle is comprised of:
a. Lateral epicondyle
b. Capitellum
c. Lateral half of trochlea

Q. Why fracture lateral condyle of humerus results in non-union?

The lateral condyle fragment gets displaced and rotated due to pull from common extensor muscles which are attached over it.

Q. Which other complication can happen with non-union of lateral condyle associated with cubitus valgus?

Tardy ulnar nerve palsy.

Q. Why does tardy ulnar nerve palsy happen?

Tardy ulnar palsy is a result of **friction neuritis of ulnar nerve** behind medial epicondyle due to constant friction because of **progressively increasing valgus of elbow.**

Q. What is the treatment of tardy ulnar nerve palsy?

Anterior transposition of ulnar nerve should be done (ulnar nerve is surgically transposed anterior to medial epicondyle. This prevents further damage to the ulnar nerve due to friction and results in gradual recovery of the ulnar nerve due to friction neuritis).

Q. What is the treatment of non-union of lateral condyle?

In children, **non-union should be treated with internal fixation and bone grafting.** However, in adults it can be left alone.

Q. What should be done with cubitus valgus deformity?

If asymptomatic and minimal deformity: It should be left alone.

In adults: It can be left alone without any consequence after ulnar nerve transposition. However, if patient demands, it should be corrected.

In children: It can be corrected by corrective osteotomy along with internal fixation of non-union.

MALUNITED COLLES' FRACTURE

Common presentation of patient with malunion of fracture around wrist:

Case summary: A 55-year-old female patient case of mild pain and deformity in her right wrist for 6 months. 6 months back, she had a fall on outstretched hand and sustained a fracture around the wrist. She underwent closed reduction and below elbow cast was application. The cast was removed after 6 weeks. After cast removal, she noticed a deformity which has been non-progressive. She also complains of mild pain during daily routine activities. There was no history of fever, loss of weight or appetite. General and systemic examination was normal. Local examination revealed manus valgus deformity with bony irregularity over the distal radius. There was tenderness over distal radioulnar joint. The radial styloid process was at the level of ulnar styloid process. Palmar and dorsiflexion were limited but not painful while pronation and supination were limited and painful. Neurovascular examination was normal.

Q. What is the clinical diagnosis?

The clinical diagnosis is malunited distal radius fracture with stiffness of wrist joint.

Q. Why do you say so?

It is because of the following reasons:

1. History of fall on outstretched hand (common mechanism to cause distal radius fracture)
2. History of cast application for 6 weeks (indicates a fracture)
3. Presence of manus valgus deformity (Fig. 1.4) (common after malunited Colles' fracture)
4. Irregularity at lower end of radius (healed fracture site)
5. Radial and ulnar styloid process at same level (indicates malunion of distal radius)
6. Limited movements at the wrist joint (indicates stiffness)

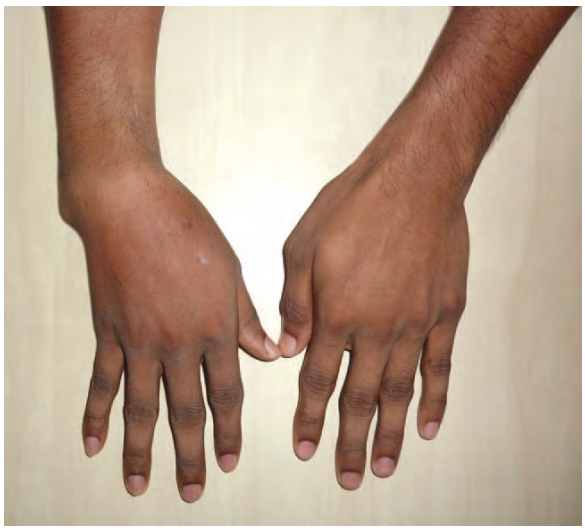


Fig. 1.4: Clinical photograph of both hands showing manus valgus deformity of right wrist

Q. What is the relationship between age and sex with distal radius fracture?

In elderly females, osteoporosis ensues faster especially after the menopause resulting in a weak bone. After fall on the outstretched hand; this is the commonest fracture around the wrist.

Bones which are prone for osteoporotic fractures are mostly cancellous bones, e.g. dorsal and lumbar vertebrae, neck of the femur, distal radius, proximal humerus and pelvis.

Q. What are the common injuries due to fall on the outstretched hand?

1. Colles' fracture
2. Scaphoid fracture
3. Radial head fracture
4. Supracondylar fracture
5. Humeral neck fracture
6. Clavicle fracture

Q. What is principal clinical finding to suggest that it is a malunited Colles' fracture?

1. Manus valgus deformity
2. Radial and ulnar styloid process are at same level

Note: Sometimes, the radial styloid process can be at a higher level than ulnar styloid process. Also, the relationship between the two styloid process should always be compared with the normal side.)

Normal relation between radial and ulnar styloid process
Radial styloid process is 8–14 mm lower than ulnar styloid process

Q. Define malunion.

When fracture does not unite in original anatomical alignment, it is called malunion.

Q. What is the clinical presentation of malunion?

Clinically, malunion presents as deformity and/or functional deficit.

Q. Do you think that his distal radioulnar joint is also involved and why do you say so?

Yes. Because patient has limited and painful pronation and supination indicating involvement of the distal radioulnar joint.

Note: Pronation and supination occurs at radioulnar joint and not at the wrist joint).

Q. Is Colles' fracture is synonymous with distal radius fracture?

No. Colles' fracture is a type of distal radius fracture.

Q. Define Colles' fracture?

Colles' fracture is defined as fracture at the distal end of radius 2 cm proximal to the distal radius articular margin at cortico-cancellous junction characterized by six displacements.

1. Lateral tilt
2. Lateral displacement
3. Dorsal tilt
4. Dorsal displacement
5. Supination, and
6. Impaction.

Q. What is the classical deformity seen in acute Colles' fracture?

Dinner fork deformity.

Q. How will you confirm the diagnosis of malunited Colle's fracture?

X-ray of the wrist (posteroanterior and lateral view).

In the wrist, posteroanterior view is asked for rather than anteroposterior.

Q. Which other investigation would delineate the fracture lines and comminution in better way?

CT scan with 3D reconstruction of distal radius is a great tool to assess the complex distal radius fracture.

Q. What are the common findings in a X-ray of malunited distal radius fracture?

1. Radial and ulnar styloid process relation is disturbed, i.e. the radial styloid process is often at the same level or above the ulnar styloid process.
2. Healed fracture in a malunited position.

Normal distal radius radiological parameters

1. The radial styloid process is 8–18 mm distal to the ulnar styloid process
2. The average radial inclination is 22°
3. The average volar inclination is 11°

It should be compared with normal side.

Q. What is the common method to treat Colles' fracture?

Closed reduction and below elbow cast application.

Q. How long cast is kept on Colles' fracture?

6 weeks.

Q. What are the other methods to treat Colles' fracture?

1. ORIF with plate and screws
2. Closed reduction and external fixator application.

Q. If there is patchy osteoporosis over various bones of wrist and hand, what else must be clinically ruled out?

If there is **patchy osteoporosis,** one must suspect **reflex sympathetic dystrophy** (complex regional pain syndrome/sudeck's osteodystrophy).

Q. How will you treat this patient?

Since patient is still having pain and stiffness, I would do the following measures.

1. *General Measures*
   a. Activity modification (avoiding lifting heavy weight and excess pronation–supination movements)

2. *Medications*
   a. Analgesics sos (as and when required)

3. *Physiotherapy*
   a. Moist heat
   b. Short wave diathermy/local ultrasound therapy
   c. Wrist and finger mobilization exercises
   d. Intrinsic muscle of hand strengthening exercises

Q. Should the deformity of distal radius be corrected at this stage?

Not now. Because

1. First; goal is to achieve "functional, painless range of movement"
2. Then, reassess the function of hand and wrist

If the deformity still hampers in restoration of adequate function (poor movements and strength, residual pain) despite appropriate physiotherapy, deformity correction should be considered. However, most malunited lower end radius fractures especially Colles' fracture may not need corrective osteotomy as after few months, patients do not have any major functional deficit hampering the activities of daily living.

Remember that **not all malunion need surgical correction**. As long as deformity does not cause any major functional deficit, it may not require surgical correction except when it is cosmetically not acceptable like cubitus varus or it may be causing any functional deficit.

Three basic function of hand

1. Pinch
2. Grasp
3. Hook

Q. If required, which surgery is performed for the malunited distal radius fracture?

Corrective osteotomy, internal/external fixation with or without bone graft is performed to correct the deformity.

Corrective osteotomy is the general term used to indicate **surgical correction of bony deformity.**

Q. What are the other common fractures around wrist?

1. Colles' fracture
2. Smith's fracture
3. Chauffeur's fracture (radial styloid fracture)
4. Scaphoid fracture
5. Volar and dorsal Barton #

Q. What are the common complications of Colles' fracture?

1. Malunion
2. Stiffness of wrist and hand
3. Carpal tunnel syndrome
4. Reflex sympathetic dystrophy (also known as Sudeck's dystrophy/complex regional pain syndrome)
5. Rupture of extensor pollicis longus tendon
6. Osteoarthritis of the radiocarpal and distal radioulnar joint

Note: Rupture of extensor pollicis longus tendon is more commonly observed in undisplaced fractures of distal radius. It happens due to fracture callus resulting in attritional rupture of tendon.

Q. What is the clinical presentation of malunited fracture of forearm bones?

1. Deformity of forearm
2. Restricted pronation-supination

Q. Name some fractures which are classically prone for malunion?

Upper Limb

1. Clavicle #
2. Supracondylar humerus fracture in children
3. Colles' fracture

Lower Limb

1. Intertrochanteric fracture
2. Calcaneum #

Colles' # was described by Abraham Colles in 1814 even before X-rays were discovered.

MYOSITIS OSSIFICANS

Common Presentation of Patient with Myositis Ossificans

Case summary: A 15-year-old boy complained of pain and stiffness in his left elbow since one year. The problem started after trauma to his left elbow due to fall following which he sustained olecranon fracture. The fracture was managed by open reduction and plate fixation followed by cast application. Mobilisation of the elbow was started after 3 weeks of surgery. Patient initially gained some movements but later, he gradually started losing movements till the elbow became very stiff. His father gave history of massage around elbow and forcible passive mobilization during the phase of mobilization. There was no history of fever or swelling.

General and systemic examination was normal. There was bony irregularity over the Olecranon. The three bony point relationship was normal. A hard bony mass was palpable in front of the cubital fossa which was non-tender. The range of movement was 40°–90° with normal neurovascular examination.

Q. What is the clinical diagnosis?

The clinical diagnosis is operated olecaranon fracture with post-traumatic myositis ossificans of left elbow.

Q. Why do you say so?

It is because of the following reasons.

1. History of trauma
2. **History of massage and vigorous passive mobilisation** (massage and vigorous passive mobilisation could initiate myositis ossificans)
3. Healed scar over the olecranon along with bony irregularity over the olecranon (indicates operated fracture)
4. Palpable bony mass in front of the elbow (indicates myositis ossificans)
5. Regaining some movement while physiotherapy and then again loss of movement may indicate myositis ossificans.

Q. Which history is relevant for myositis ossificans?

a. Massage around the elbow, and
b. Passive forcible mobilization.

Massage and **forcible passive mobilization** of a stiff joint during the phase of early mobilisation could increase the chance of myositis ossificans. (Both factors can result in bleeding in the muscles adjacent to the joint which in turn leads to myositis ossificans.)

Q. How does myositis ossificans occurs?

Myositis ossificans around elbow occurs due to bleeding in the brachialis muscle. Later, the hematoma gets converted into a bony mass (myositis ossificans) due to osteoblastic proliferation. The reasons behind osteoblastic proliferation remains idiopathic.

Q. Is there any inflammation in the muscle as name suggests (myositis)?

No. There is **no** inflammation in the muscle. It is a **misnomer**. There is an ossified (not calcified) hematoma in the substance of the brachialis muscle.

Q. How will you confirm the diagnosis?

I would like to take X-ray. X-ray shows a myositis mass in front of the humerus and a *in situ* plate over olecranon indicating an operated olecranon # (Fig. 1.5).

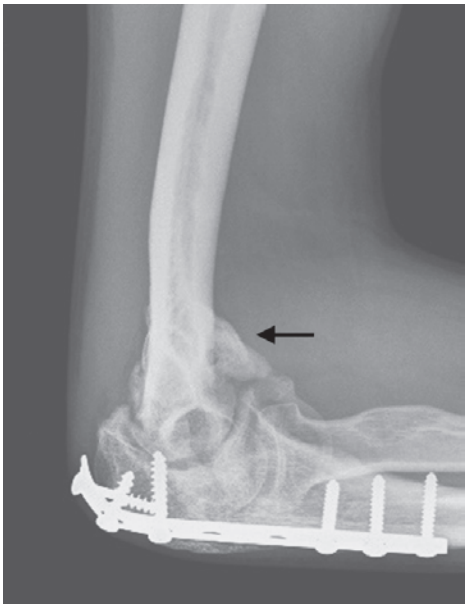


Fig. 1.5: Lateral view of elbow reveals myositis mass (black arrow) in front of the humerus

Q. Is there any clinical-radiological staging of myositis ossificans?

Yes. There are **two clinical-radiological** stages of myositis ossificans

1. ***Early stage:*** It starts after few days of mobilization. There is local rise in temperature and tenderness around the joint. **The movements suddenly become more painful and much less than what patient gained in the period of mobilization.**
   Radiographs of the region reveals a **"cotton wool appearance"** as early myositis.
2. ***Late stage:*** After a few weeks, pain and inflammation subsides and a bony mass is gradually formed due to ossification of hematoma. The bony mass is clinically palpable. The range of movement is restricted but is painless.
   Radiologically, a **"bony mass"** is seen in front of the bone with **usually a clear line of demarcation between the mass and bone.**

Q. Is there any other investigation which can reveal the bone mass in a more accurate way?

CT scan can be done to confirm the bone mass and its extent.

Q. What could be the radiological differential diagnosis of myositis ossificans?

Parosteal osteosarcoma

Features Similar

1. Acute history
2. Young age
3. Pain and swelling at the lower end of humerus (near metaphysis which is site for osteosarcoma)
4. Localized tenderness and swelling
5. Radiologically, cotton-wool appearance and bony mass can confuse the picture with periosteal reaction.

Features Against

1. History of trauma (though, it can be present in case of tumor and infection too!!)
2. Both cotton-wool appearance and bony mass of myositis are slightly away from bone (gap between them) whereas in case of osteosarcoma, the bony mass or periosteal reaction is in continuity with bone.
3. Serum ALP (alkaline phosphatase) is very high in case of osteosarcoma.

Q. How will you manage a case of myositis ossificans?

The management depends upon the **clinical and radiological stage.**

A fact need to be remembered that once the process of myositis ossificans starts, it is inevitable. It will form a mature bony mass in future. So, the treatment is aimed at reducing the further extent of bony mass and not to prevent the formation!

Early Stage

Goal is to reduce pain, inflammation and edema and prevent extent of myositis mass formation

a. **Re-immobilization** in the POP cast for two to three weeks
b. **NSAIDs** (indomethacin is the drug of choice unless there is a contraindication)
c. **Anti-edema measures** (limb elevation)
d. **Cold pack** to the elbow

After two to three weeks, the limb is taken out of POP and re-mobilized. Only **gentle active mobilization is permitted**.

Late Stage

Once mature bony mass is formed, it can be **surgically excised if it hampers the function.**

Q. What is the complication of myositis ossificans excision surgery?

Re-formation of the myositis mass is the commonest complication after the surgery as the post-surgical hematoma in the muscle can re-form the mass and it can lead to re-stiffness of the joint. (This has to be explained to the patient before the surgery.)

Q. How much is the flexion deformity in this patient?

It is 40°.

Q. What is the meaning of flexion deformity?

Flexion deformity means that the joint cannot be fully extended, actively or passively.

Q. Do you think that is it a stiff joint or ankylosed joint?

It is a stiff joint as joint with any degree of loss of movement is known as stiff joint. However, in an ankylosed joint, there is just a jog of movement or no movement at all. So, an ankylosed joint is a stiff joint but not all stiff joints are ankylosed.

This patient has ROM of 40° to 90° which indicates that it is stiff joint but not ankylosed.

Q. What do you mean by ankylosis?

Ankylosis means pathological fusion of a joint which hardly exhibits any movements.

Note: Arthrodesis means surgical fusion of a joint)

Q. What are the types of ankylosis?

There are two types of ankyloses.

a. True ankylosis: Fusion in the joint

b. False ankylosis: Stiffness in the joint due to extra-articular causes.

Q. What are the types of true ankylosis?

There are two types of true ankyloses: Fibrous and bony.

a. *Fibrous Ankylosis Occurs due to*

1. Tuberculous arthritis of peripheral joints
2. Gonococcal arthritis
3. Rheumatoid arthritis

- Fibrous ankylosis occurs due to dense fibrous adhesions running across the joint surfaces, and tight capsule-synovium-ligament complexes.
- Fibrous ankylosis is characterized by **painful, but slight jog of movement.**
- Any attempted movements are painful as highly innervated dense adhesions and contracted capsule-synovium-ligament are stretched during attempted movements.
- Jog of movements are present due to slightly flexible and pliable nature of fibrous adhesions.
- X-ray reveals relatively preserved joint space as there might not be complete damage to the articular cartilage.

b. *Bony Ankylosis Occurs due to*

1. Septic arthritis
2. Occasionally when tuberculous arthritis becomes infected
3. Tuberculosis of the vertebrae ends with bony fusion of the two vertebrae.

- Bony ankylosis occurs when subchondral bone from ***both the articular surfaces*** is denuded of the articular cartilage and subchondral bone is exposed resulting in bony fusion of the two articular surface.
- Bony ankylosis is characterized by **painless joint, with no movements.** It is painless as the two articulating surfaces are fused due to bony trabeculae formation across the joint due to severe or complete destruction of cartilage.
- X-ray of the joint reveals obliteration of joint space with bony trabeculae crossing the joint (Fig. 1.6).

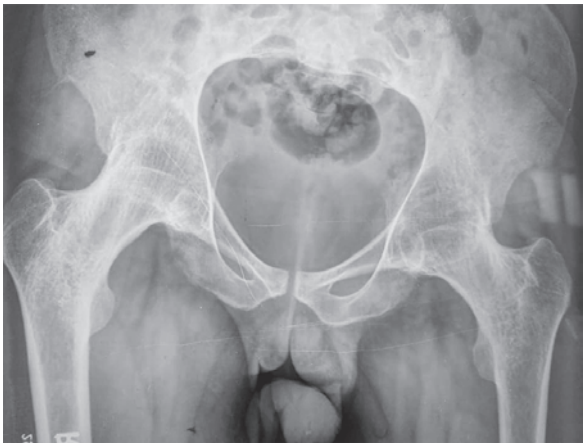


Fig. 1.6: Ankylosed hip with total obliteration of hip joint space copy

Q. What are the causes of false ankylosis?

The causes of false ankylosis vary from skin to the joint.

a. ***Skin contracture:*** Burn scar, surgical or open wound scars
b. ***Underlying soft tissue contracture:*** Subcutaneous tissue/fascia scars, Dupuytren's contracture.
c. ***Muscles tendon units contracture*** which fails to slide while joint is attempting to move.
d. ***Joint capsule contracture*** after prolonged immobilisation, trauma, inflammation or surgical procedure . It fails to relax while joint is attempting to move.
e. ***Intra- and extra-articular ligaments contracture***
f. ***Bone:*** New bone formation (myositis) or excess callous formation after fracture healing or malunited displaced bone fragment.

Q. How will you manage true ankylosis?

a. *Bony Ankylosis*

1. **Arthrodesis in sound position** if bony ankylosis is in unsound position of the joint.
2. **Joint replacement if there is no evidence of infection.** At least few (5–10) years after the episode of septic arthritis and 1–2 years after complete treatment of tuberculosis, if ESR and CRP are normal.

b. *Fibrous Ankylosis*

Initial attempt of physiotherapy can be tried to overcome the tight intra-articular adhesions and other reasons for a stiff joint.

However, if it fails to improve open or arthroscopic adhesiolysis should be performed.

Q. How will you manage false ankylosis?

False ankylosis is managed according to the structure affected.

a. *Skin:* Lengthening of scar by Z or V-Y plasty. Scar excision and reconstruction by flap
b. Tight fascia or subcutaneous tissue-excision
c. *Tight muscle tendon unit:* Lengthening of tendon by Z plasty or V-Y plasty
d. *Tight capsule:* Capsulectomy and/or capsulotomy.
e. *Tight ligaments:* Lengthened if possible otherwise should be left alone lest it would lead to instability.

Q. What should be done after release of ankylosed or stiff joint?

Patient needs to undergo extensive supervised physiotherapy to retain the movements; otherwise joint is prone for stiffness.

Q. What is the complication of surgery done for release of stiff joint?

It can lead to re-stiffness, however may not be same degree. Hence, guarded prognosis about movement should be given.

NERVE INJURY MODULE

To understand the clinical symptoms, signs, management and prognosis of nerve injury; it is important to understand the anatomy of the nerve (Fig. 1.7), causes of nerve injury and pathophysiology (wallerian degeneration and regeneration) of nerve injury (Table 1.1).

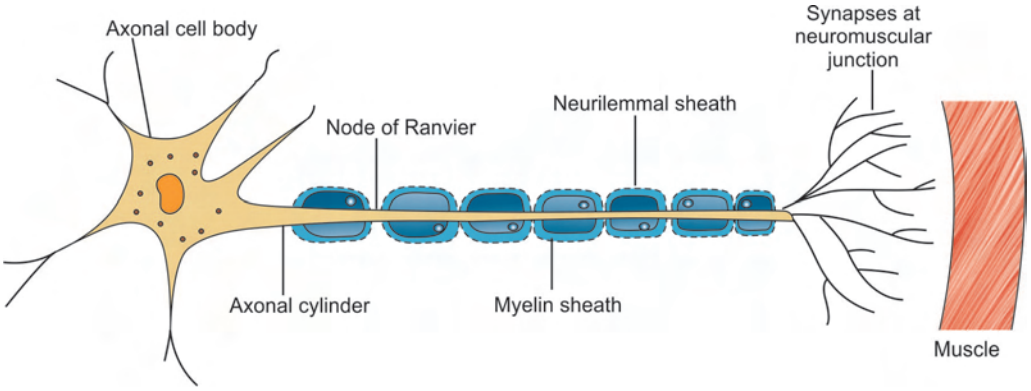


Fig. 1.7: Illustrated diagram of a single nerve axon

Table 1.1: Summary of wallerian degeneration and regeneration

Any structural injury to axonal cylinder and/or neurilemmal sheath leads to **wallerian degeneration (WD)**. WD is characterized by:

1. Complete breakdown of axonal cylinder and myelin sheath in both proximal and distal segments, proximally up to the node of Ranvier just proximal to the level of injury and distally up to the motor end plate.
2. The broken down material of axonal cylinder and myelin is engulfed and, area is further cleared of debris by macrophages. However, **neurilemmal sheath is not broken down**. This phenomenon is wallerian degeneration.

Wallerian regeneration (WR) is characterized by:

1. Growth of new axonal sprouts (50–100) from the end of the proximal axon terminal. Only one of them makes connection with neurilemmal sheath and continues to grow as axonal cylinder at the rate of **1 mm/day**.
2. New axon is further myelinated by Schwann cells. Finally, it meets with motor end plate.

Nerve regeneration (WR) is characterized by two phenomena.

A. **Tinel's sign:** Hence, Tinel's sign is **NOT seen in neuropraxia where there is neither WD and WR**.

B. **Motor march:** Nerve re-innervates in the order of its innervation pattern to the muscles, i.e. proximal to distal which known as motor march. Hence, **motor march too is not seen in neuropraxia** as there is no injury/regeneration.

Q. What are the causes of nerve injury/affection?

Peripheral nerve involvement is seen in:

1. **Infective:**
   a. Poliomyelitis—history polio in childhood
   b. Hansen's disease—history of patchy loss of sensation over body, thickened and tender nerves like CPN, greater auricular, ulnar, superficial radial nerve
2. **Traumatic:** Traumatic injury to spinal cord or peripheral nerves
3. **Acute or chronic pressure over the nerves:** Intervertebral disc prolapse, carpal or tarsal tunnel syndromes
4. **Charcot-Marie-tooth** disease
5. **Neoplastic:** Tumors compressing the nerves; neurofibroma, neurofibromasarcoma
6. **Metabolic:** Vitamin B_1, B_{12} deficiency, diabetes mellitus, chronic alcoholism, porphyria
7. **Chemical:** Lead poisoning

Note: Students must take pertinent history in cases of nerve affection.

Q. Classify nerve injuries?

Seddon's Classification of Nerve Injury

1. *Neuropraxia:* **Focal conduction block** at axonal cylinder but no demonstrable anatomical disruption.
2. *Axonotmesis:* **Injury to the axonal cylinder** but the **neurilemmal sheath is intact.**
3. *Neurotmesis:* Complete injury to the nerve including neurilemmal sheath.

Description of Type of Nerve injury

1. *Neuropraxia:* Since there is only focal conduction block and no anatomical damage to the nerve in neuropraxia, there is no wallerian degeneration of the nerve. As there is no demonstrable structural damage to nerve, nerve function is restored within a few weeks (3–6 weeks). It carries **excellent prognosis**.

Neuropraxia is comparable to a vehicle overturn on a highway and a resultant traffic blockade. However, there is no damage to road. Hence, complete restoration of traffic is expected once blockade is over. Likewise, neuropraxia resolves once axonal blockade resolves.

2. *Axonotmesis:* It is observed after traction or compression injury of the nerve. The axonotmesis is characterized by **damage to the axonal cylinder but intact neurilemmal sheath.** It undergoes WD and WR. Further, recovery period of axontmesis is characterized by presence of **Tinel's sign and motor march phenomena.** Recovery is **usually complete** because neurilemmal sheath is intact

Once in exam, there was a question on "classify nerve injury". A student wrote correct classification. However, he/she fumbled on the name! Rather than Seddon's, it was mentioned as Saddam's classification!!

Choice is yours, Seddon or Saddam!

but may take a few months according to the length of the nerve damaged. It carries **good prognosis**.

3. *Neurotmesis:* The nerve is **completely severed including axonal cylinder and neurilemmal sheath** and is characterized by:
   a. Wallerian degeneration.
   b. Since the **neurilemmal sheath is also severed,** the **two ends of the nerve along with sheath retract.** When the nerve starts regenerating from proximal end; axonal sprout **fails to connect** with the distal end of the nerve since both ends are retracted, and gap is filled with scar tissue.
   c. Also, it may lead to **neuroma formation** at the regenerating end of axon as it fails to makes a connection with sheath.
   d. Unless surgical intervention is done, neurotmesis may not recover and carries **poor prognosis**.
   e. Motor march and Tinel's sign could be observed if nerve starts regenerating after the two severed ends are surgically repaired.

Any of the three types of nerve injury would result in various clinical feature.

1. **Sensory loss:** Anaesthesia/hypoaesthesia/paresthesia
2. **Motor dysfunction:** Paresis/palsy
3. **Loss of reflexes**
4. **Autonomic dysfunction:** Trophic changes in skin and nails

Clinical facts

1. By and large, **all closed fractures and dislocations commonly result in neuropraxia or axonotmesis.** Since neuropraxia/axonotmesis usually recover, the line of treatment is usually expectant.
2. **Open fractures or open injuries** result in **neurotmesis** unless proved otherwise. Hence, nerve should always be explored in open injuries to ascertain its anatomical status. If the nerve is completely severed, it should be approximated to ensure recovery.

Diagnosis of nerve injury is established by

1. *Nerve conduction velocity (NCV) test:* ***Usually performed at the end of three weeks.*** It is useful in ascertaining
   a. Location of the lesion
   b. Type of lesion
   c. Prognostic
2. *Electromyography (EMG):* **Not useful till 3 weeks of acute injury.** After 3 weeks, EMG is performed. It shows fibrillations and positive sharp waves in axonotmesis and neurotmesis.
3. *Specific investigation* as per the etiology, e.g. Hansen's and carpal tunnel.

With this background information of nerve injuries and understanding of pathology, we will proceed with case history and questions.

Points to remember

1. ***Best prognosis:*** Neuropraxia (since no nerve damage)
2. ***Worst prognosis:*** Neurotmesis (since the nerve is completely severed)

3. ***Tinel's sign and motor march never observed in:*** Neuropraxia (as there is no WD)
4. ***Tinel's sign and motor march always observed in:*** Axonotmesis (as WD is followed by WR)
5. ***Tinel's sign and motor march may be observed in:*** Neurotmesis (if surgical repair is done)
6. ***Since neuropraxia always recovers:*** Line of treatment is expectant!
7. ***Since neurotmesis will not recover:*** Surgical intervention is must
8. ***Since axonotmesis mostly recovers:*** Line of treatment is expectant initially and surgical intervention only if incomplete or no recovery.

WRIST DROP

Case summary: A 25-year-old man met with RTA, 2 months back following which he sustained fracture shaft of the humerus. At the time of injury, he had weakness at the wrist and finger with inability to extent wrist, thumb and fingers. He underwent open reduction and internal fixation of fracture shaft humerus with plate. Current clinical examination reveals mild tenderness at the fracture site. Elbow and shoulder examination is normal. Distal neurological examination revealed wrist and finger drop with decreased sensation in 1st web space.

Q. What is the clinical diagnosis?

The clinical diagnosis is healing operated fracture shaft humerus with radial nerve palsy.

Q. What is the impression you get about the wrist and finger drop?

Wrist and finger drop means that there is radial nerve palsy.

Q. Why patient is having wrist and finger drop?

Wrist and finger drop is due to:

a. *Paralysis of wrist extensor:* Extensor carpi radialis longus (ECRL) and brevis (ECRB)
b. *Paralysis of finger extensor:* Extensor digitorum (ED), extensor indicis (EI)
c. *Paralysis of thumb extensor:* Extensor pollicis

Q. Which nerve injury results in only finger drop?

Posterior interosseous nerve injury.

Q. Which condition results in posterior interosseous nerve (PIN) injury?

PIN palsy is observed in Monteggia fracture dislocation.

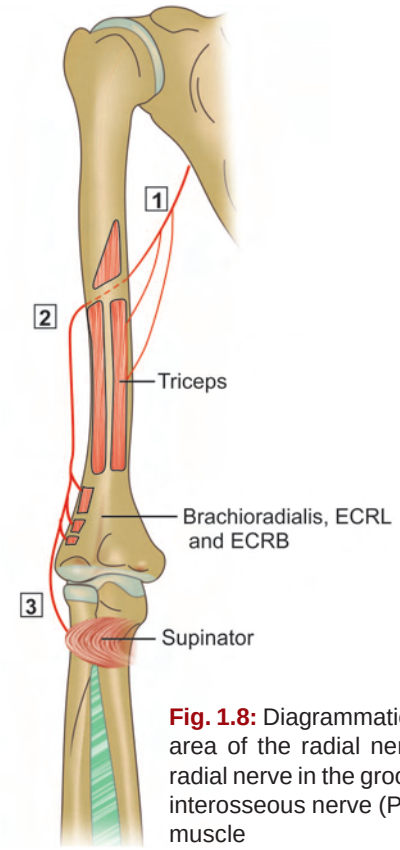
Q. Why there is only finger drop in PIN palsy?

PIN palsy results in thumb and finger drop because of paralysis of finger extensors (EDC, EI) and extensor polllicis whereas wrist extensor ECRL and ECRB escape paralysis as latter are supplied by the radial nerve.

Q. What do you mean by high and low radial nerve palsy?

In **high radial nerve palsy,** the radial nerve is paralyzed in the radial groove affecting the function of brachioradialis, ECRL, ECRB and other common extensors originating from lateral epicondyle. Hence, patient has wrist, thumb and finger drop. Since triceps is supplied before the radial nerve enters the radial groove, hence elbow extension is not affected (Fig. 1.8).

In **low radial nerve palsy,** the nerve is injured after it has supplied brachioradialis, ECRL and ECRB. It is known as PIN after it enters substance of supinator. So, only common extensors are affected causing palsy of finger-thumb (Fig. 1.8).



Radial nerve, after crossing the elbow joint enters the supinator muscle which is around the radial head and neck. From this point, radial nerve is known as posterior interosseous nerve (PIN).

In Monteggia fracture dislocation; there is proximal ulna fracture with dislocation of proximal radioulnar joint. The PIN which winds around neck of radius, sustains traction injury due to dislocated head radius (Fig. 1.8).

Fig. 1.8: Diagrammatic representation of course of radial nerve. Point 1 represents area of the radial nerve before it enters the radial groove. Point 2 represents radial nerve in the groove and point 3 represents radial nerve to be called posterior interosseous nerve (PIN) just after it crosses the elbow joint and enters supinator muscle

Points to examine in nerve injury

a. Wasting of muscle
b. Deformity at joints
c. Scar and tenderness over nerve course
d. Trophic change in hand and nails
e. Joint movement
f. Tinel's sign, motor march
g. Sensori-motor examination, reflexes

Q. What are the clinical factors indicating a recovering nerve?

1. Improving sensory perception and motor power
2. Motor march phenomena
3. Progressive Tinel's sign
4. Return of reflexes

Q. How did you elicit Tinel's sign and what is its relevance?

Tinel's sign is elicited by gently tapping along the nerve its course from **distal to proximal**. In case of "**positive Tinel's sign"**, patient will experience current/shock-like sensation along the course of the nerve.

If the Tinel's sign is progressive (means, it is progressing distally with every follow up of patient), it suggests a recovering nerve. If the Tinel's sign is static (not progressive at every follow up), it indicates a neuroma formation and is a bad sign.

Q. What is motor march?

In recovering phase of nerve injury, there is proximal to distal recovery of muscle power.

Q. How will you confirm the diagnosis of nerve injury?

Diagnosis is confirmed by NCV and EMG.

Q. What is the role of X-ray?

X-ray is done to look for any associated

a. Fracture

b. Foreign body/lesion which can affect nerve.

Q. What type of nerve injury is suspected in this patient?

It is most likely either neuropraxia or axonotmesis.

Q. Why do you think that this is neuropraxia or axonotmesis?

Generally, all closed fractures and dislocation result in neuropraxia or axonotmesis type of nerve injury.

Q. When do you suspect neurotmesis?

In case of open fracture/dislocation/any other open injury with nerve palsy.

Q. Why it is important to differentiate between these type of nerve injuries?

It is important to differentiate between these nerve injuries because neuropraxia is always managed on expectant lines whereas axonotmesis initially is managed on the expectant line of treatment. However, neurotmesis always needs a surgical intervention.

Q. How will you treat radial nerve injury in this patient?

Since the nerve injury in this patient is associated with closed fracture, hence it will be either neuropraxia or axonotmesis type of nerve injury. These type of nerve injury usually recover in due course of time with expectant line of treatment. Hence, one must follow expectant/conservative line of treatment.

Q. What is expectant or conservative treatment of nerve injury?

The meaning of expectant line is:

a. **Passive mobilization of wrist and fingers** several times a day (passive mobilization of affected joint to prevent stiffness of affected joints)

b. **Active mobilization of unaffected joints**

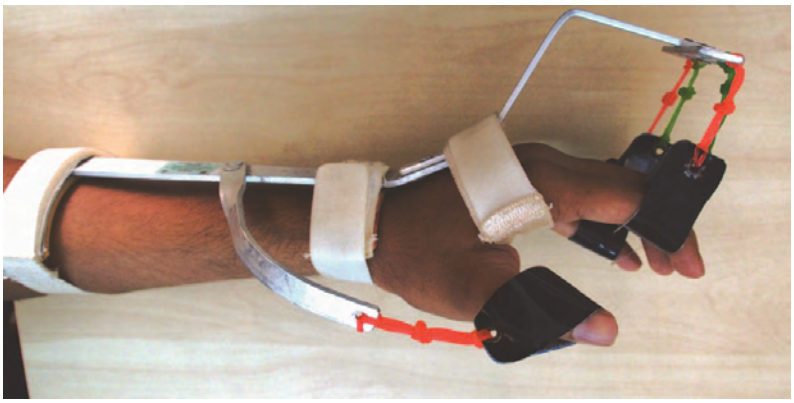


Fig. 1.9: Dynamic cock up splint applied on forearm, wrist and hand. It keeps finger in extended position

c. **Application of dynamic cock-up splint:** Encourages mobilization and keeps wrist and finger in functional position at the time of rest (Fig. 1.9)
d. **Electrical stimulation to the affected muscle:** Prevents disuse atrophy
e. Oral and/injectable vitamins B_1, B_6, B_{12} can be used but are of questionable value.

Q. How will you follow this patient?

Since nerve recovery is a slow process (1 mm/day), patient should be followed every 6–8 weeks. At every follow-up, clinico-electrophysiological examination must be performed which includes:

a. **Sensory examination** and recovery
b. **Motor examination** and its recovery **(motor march)**
c. **Tinel's sign** which must be progressive: It means that the point over skin where Tinel's sign was last elicited, should migrate at least 30–40 mm as nerve grows at the rate of 1 mm/day from the last follow up. So, after 6–8 weeks (42–56 days) a regenerating nerve should move 30–40 mm distally, if it is regenerating at the rate of 1 mm/day.

Note: A **static Tinel's sign** indicates a **neuroma** formation which is a bad sign and indicates that nerve is not recovering)

d. **NCV**
e. **EMG** (shows fibrillation and positive sharp waves)

Q. What is the next step if there is no nerve recovery?

a. **Explore** the nerve
b. **Neurolysis** if nerve is adhered within fibrous tissue or fracture callus
c. **Neurorrhaphy** (nerve repair) with or without nerve grafting **if nerve is found to be completely severed**

Q. What will you do after neurolysis/neurorrhaphy?

I would again follow the expectant line of treatment

Q. What is the next step if nerve still does not recover even after neurolysis/ neurorrhaphy?

Tendon transfer should be performed to restore wrist, thumb, and finger extension.

Remember, before you answer tendon transfer!! Ensure that
- Joint is NOT stiff, i.e. it is mobile
  - Stiff joint need to be mobilized before tendon transfer
  - At least grade 4 powered tendon to be transferred

Q. Which tendon transfer is performed for radial nerve palsy?

Modified Jones transfer:

To restore wrist extension: Transfer pronator teres to ECRL and ECRB

To restore finger extension at MCP joint: Transfer FCU/FDS to EDC

To restore thumb extension and abduction: Transfer palmaris longus to EPL

FCU, flexor carpi ulnaris; FDS, flexor digitorum superficialis; EDC, extensor digitorum communis; EPL, extensor pollicis longus.

Q. What could be done if there are no viable tendons to transfer?

Wrist arthrodesis in functional position

Note: Functional position for wrist and hand is **glass holding position**

Various surgical procedures performed for a paralysed nerve
1. Neurolysis
2. Neurorrhaphy (nerve repair) with or without nerve graft
3. Tendon transfer
4. Arthrodesis

Q. What are the prerequisite for tendon transfer?

1. **Age >5 years:** After tendon transfer, patient need to be re-educated to use the **new muscle tendon unit in new location and new function.** Children lesser than 5 years may not be able to cooperate with re-education and rehabilitation programme.
2. **Mobile joints:** If involved joint is/are stiff, then tendon transfer should not be performed till the joints become passively mobile and free.
3. **Muscle to be transferred should have minimum grade IV power**
4. Preferably muscle tendon unit from same action group also known as **synergistic/ phasic** (i.e. flexor to flexor and extensor to extensor).

Note: Muscle-tendon unit tends to **lose one grade power** after transfer. Hence, muscle with grade IV power post-transfer will retain grade III power which is sufficient to act against the gravity and accomplish reasonable function. However, if we transfer muscle with grade III power, the resultant power post-transfer would be grade II which would be insufficient to act against gravity and perform the optimal function.

Q. How do you grade muscle power?

According to medical research council (MRC), muscle power is graded from 0 to 5.
0 = no power
1 = flicker of contraction
2 = contraction with gravity eliminated
3 = contraction against gravity but no resistance
4 = contraction against mild-moderate resistance
5 = normal power

Note: Refrain using + or – with power, e.g. patient has 3+ power in elbow flexion. There is no standardization of + or – and has not been suggested by MRC.

Q. What are the factors which affect the nerve recovery?

1. **Age: Younger** the person, better is recovery
2. **Distal** lesion: **Better** recovery
3. **Predominantly motor (radial) or sensory** nerve: **Better** recovery
4. Mixed nerves/nerve with fine function (median/ulnar)—poor recovery
5. **Type of nerve injury:** Neuropraxia → axonotmesis → neurotmesis in order of bad prognosis

Q. What you would have done if this nerve injury was associated with open wound in arm?

Then, this should be treated like an open fracture of humerus with radial nerve injury. It involves:
1. **Debridement** of the wound
2. **Fixation** of fracture by external fixator (usually, internal fixation is avoided in open fracture)
3. **Exploration of nerve**
4. If nerve is found to be intact, then expectant line of treatment should be followed
5. If nerve is found to be severed (neurotmesis); primary repair of nerve is performed if the wound is clean. However, if the wound is quite contaminated, then secondary repair is performed after a few weeks once the wound is clean after repeated debridements.

Principle of nerve repair

1. **Repair in clean, vascular field** (that is why no primary repair is attempted if wound is contaminated)
2. **Tensionless repair** (it means that if there is defect in nerve length, then either nerve graft is used or joint is flexed to perform and protect repair from tension at repair site).

Q. What is the reason of persistent pain after the nerve injury in the area of nerve distribution?

It is known as causalgia. It is another type of reflex sympathetic dystrophy associated with nerve injury (also known as Type 2 complex regional pain syndrome).

Q. What is the root value of radial nerve?

The root value of radial nerve is C5, 6, 7, 8 and T1.

Q. What do you mean by autonomous zone?

Autonomous zone is the sensory area which is exclusively supplied by a single nerve without overlap by other nerve.

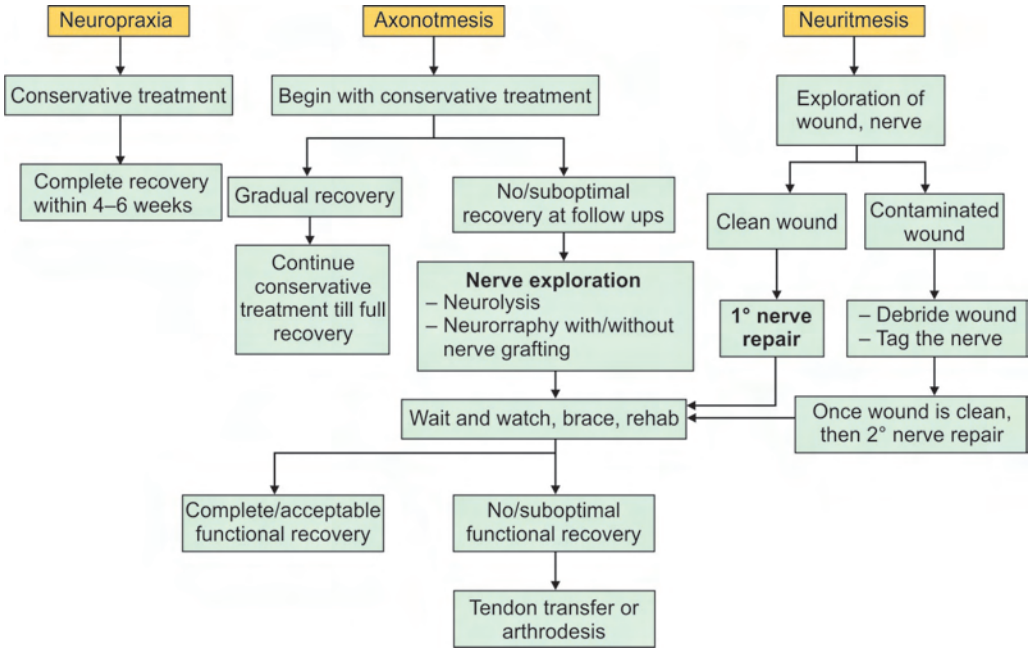
Q. What is the location of the autonomous zones of radial, median, ulnar and axillary nerve in upper limb?

a. *Radial nerve:* Dorsal 1st web space
b. *Median nerve:* Volar aspect of index finger
c. *Ulnar nerve:* Ulnar border of little finger
d. *Axillary nerve:* Upper lateral aspect of arm (known as regimental badge sign)

Q. Outline the treatment plan for all three types of peripheral nerve injury.

A brief outline of management of all three types of nerve injuries has been explained in Flowchart 1.1.

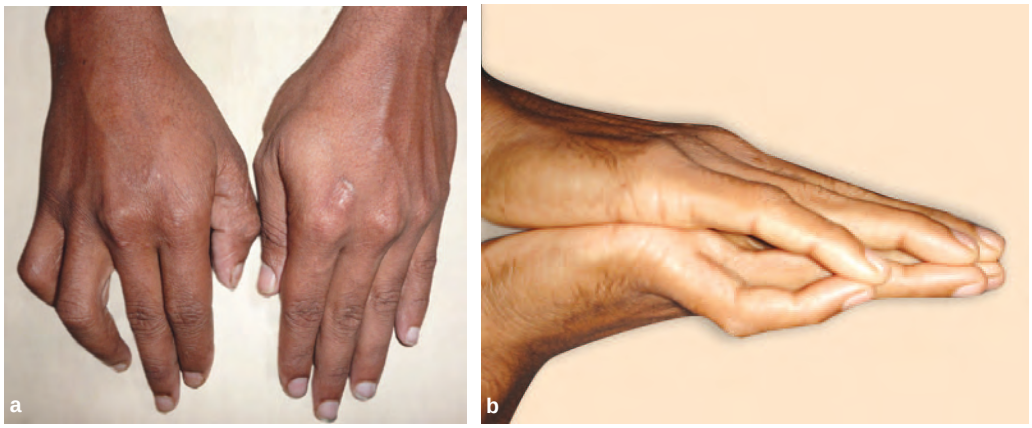
Flowchart 1.1: Management of three types of nerve injuries



CLAWHAND

Q. Define clawhand?

Clawhand is characterized by hyperextension at the metacarpophalangeal (MCP) joint and hyperflexion at the interphalangeal (IP) joint (Figs 1.10a and b).



Figs 1.10a and b: Clinical picture of hand shows a classical clawhand which becomes clear in folded hand or *namaste* position

Q. Which nerve injury causes clawhand?

Clawhand deformity is seen after ulnar nerve injury or combined injury to both ulnar and median nerve.

Q. What is the difference between the clawhand pattern in 'ulnar' and 'ulnar with median' nerve injury?

In ulnar nerve injury: Partial clawhand affecting only little and ring fingers is observed.

In combined injuries of median and ulnar nerve: Complete clawhand deformity is observed affecting all four fingers.

Q. What is the reason behind clawhand after ulnar nerve injury?

Ulnar nerve supplies medial two lumbricals and all Interossei muscles. The combined action of interossei and lumbricals is to flex metacarpophalangeal (MCP) and extend interphalangeal (IP) joints. When these muscles are paralysed due to injury to the ulnar nerve, it results in MCP hyperextension and IP flexion due to unopposed action from finger extensors acting at MCP and flexors acting at the IP joints respectively.

Q. Why clawhand is not seen after isolated median nerve palsy?

Absence of clawhand deformity after the median nerve injury, even though the lateral two lumbricals are paralysed, is based upon a fact that the 'predominant MCP flexor and IP extensor of finger is interossei muscle and not lumbrical'. The lumbricals only assist interossei in above said action.

In case of ulnar nerve injury, both interossei and lumbricals are paralyzed resulting in clawhand.

In case of median nerve injury; even though there is paralysis of lateral two lumbricals but the interossei muscles are not involvement. Hence, there is no significant weakness in MCP flexion and IP extension and thereby, the clawhand is not observed.

Q. What is the nerve supply to the lumbricals and interossei?

Lateral two lumbricals: Median nerve
Medial two lumbricals: Ulnar nerve
Interossei (palmar and dorsal): Ulnar nerve

Q. What is the function of lumbricals and interossei?

1. *Lumbricals:* MCP joint flexion and IP joint extension
2. *Interossei: Palmar*—adduction of fingers; *dorsal*—abduction of fingers
   Furthermore *interossei assist lumbricals in MCP flexion and IP extension.*

Q. Which specific muscles are to be tested for median nerve?

1. Flexor pollicis longus
2. Flexor digitorum superficialis
3. Lateral two tendons of flexor digitorum profundus (FDP) **(Ochsner's clasping index sign)**
4. Abductor pollicis brevis **(pen test)**
5. Opponens pollicis **(test opposition of thumb to rest of fingers)**
6. Lateral two lumbricals
7. Pronators

Q. Which specific muscles are to be tested for ulnar nerve?

1. Flexor carpi ulnaris
2. Medial two tendons of flexor digitorum profundus
3. Adductor pollicis **(book test or Froment's sign)**
4. Palmar interossei **(finger adduction/card test)**
5. Dorsal interossei **(finger abduction)**
6. Abductor digiti minimi

Note: It is important that student must know the test for each muscle

Q. What is 'Hand of benediction' or 'Preacher's sign'?

In median nerve injury or compression at the elbow or forearm level, where it affects long flexors of fingers along with lateral two lumbricals of hand, patient finds difficult to flex his finger at the PIP and DIP joints in index and middle finger while attempting to make a fist.

This gives hand an appearance of 'benediction'.

Q. What is intrinsic plus hand?

It is a condition where extrinsic muscles of hand (long flexor group and extensor digitorum communis) are paralysed whereas intrinsics muscles are normal resulting in overpowered intrinsics causing MCP flexion and IP joint extension (Fig. 1.11).

Q. What do you mean by intrinsic minus hand?

Intrinsic minus means paralysis of intrinsic muscles of hand whereas extrinsic muscles of hand (long flexors and extensors) are normal.

It is observed after paralysis of ulnar nerve or ulnar and median nerve palsy at the level of wrist resulting in clawing of the hand.

Q. Which splint is used to prevent deformity in clawhand?

Knuckle bender splint is used in clawhand. It keeps MCP and IP in functional position (Fig. 1.12).

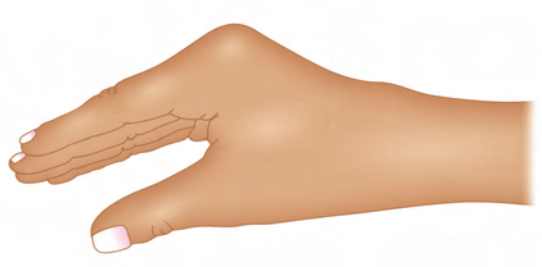


Fig. 1.11: Diagrammatic representation of intrinsic plus hand

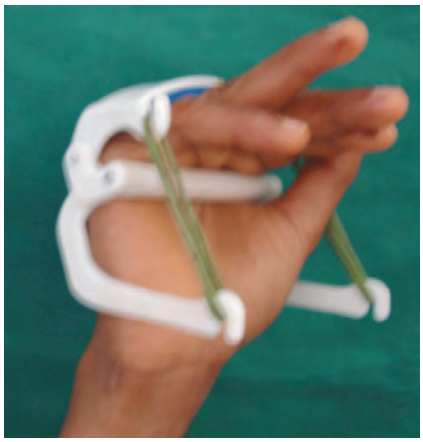


Fig. 1.12: Clinical picture of hand with knuckle bender splint

Q. What are the various procedure(s) for ulnar nerve injury?

1. Exploration of the nerve followed by neurolysis
2. Neurorrhaphy (nerve repair) with or without nerve grafting
3. Tendon transfers

Q. What is ulnar paradox?

The ulnar nerve innervates medial two tendons of FDP proximally, and intrinsic muscles of hand (interossei and medial two lumbricals) distally. When ulnar nerve is paralysed near the elbow; two medial FDP, medial two lumbricals and interossei lose its power and hence, fingers lay flat due to no action from FDP.

However, when ulnar nerve is paralyzed below the elbow or near the wrist, medial two FDP retains its innervation but lumbricals and Interossei remain paralysed. Hence, normal FDP results in unopposed flexion at of PIP and DIP resulting in typical clawing of hand.

Thus distal palsy (lower lesion) results in "more deformed appearance (claw)" of the hand as compared to the proximal palsy (higher lesion) where less deformity is apparent in the hand.

So, this is the paradox! Higher the lesion, lesser is the apparent deformity in hand whereas lower the lesion, severe is the appearance! Normally one would expect more severe deformity with higher lesion and lesser deformity with lower lesion.

Q. What is tardy ulnar nerve palsy?

Tardy means "Late onset". Tardy ulnar nerve palsy is **most commonly observed in gradually progressive cubitus valgus deformity with non-union of lateral condyle fracture of humerus.** As the lateral condyle goes in for non-union, it results in progressive cubitus valgus deformity of the elbow. The progressive valgus deformity results in traction over the ulnar nerve. Further, every flexion-extension movement of the elbow results in frictional neuritis of the ulnar nerve followed by the fibrosis of ulnar nerve. So, this causes a slow onset of paralysis of ulnar nerve (tardy ulnar nerve palsy), usually a few months to years later after valgus deformity sets in.

Q. How do you treat tardy ulnar nerve palsy?

Tardy ulnar nerve palsy is treated by neurolysis and anterior transposition of ulnar nerve (anterior to medial epicondyle) to prevent further friction.

Q. What is the classical deformity after the median nerve injury?

Ape thumb deformity.

Note: The thumb abductor (abductor pollicis brevis) is paralysed with overacting adductor pollicis results in adducted thumb.

Q. Which splint is used after median nerve injury?

Thumb abductor splint.

Note: After abductor palsy, the thumb remains in adduction which could result in adduction contracture. Hence, an abduction splint keeps thumb in abduction avoiding adduction contracture.

Q. What is Klumpke's paralysis?

Klumpke's paralysis commonly occurs at birth during vaginal delivery. Occasionally, it happens due to catching a tree branch while fall from tree. It is due to **involvement of lower trunk of brachial plexus (C8, T1).** Mostly, **ulnar nerve is involved** resulting in paralysis involving intrinsic muscles of hand (interossei, medial two lumbrical, adductor pollicis and other hypothenar muscles) leading to clawhand and weakness of wrist and finger flexors (FCU, medial two FDP). Due to T1 involvement, there can be Horner's syndrome.

Q. What is Erb's paralysis?

Erb's paralysis is caused by injury to the upper trunk of the brachial plexus (C5, 6) in conditions wherein there is excess lateral flexion of the neck.

Most commonly, it happens during vaginal delivery in babies with shoulder dystocia when head and neck is excessively pulled sideways with respect to body axis to aid delivery of the trunk. Another common cause is fall on to the tip of shoulder during RTA.

Both conditions result in excess traction on upper trunk of Brachial plexus causing the palsy of upper trunk. The commonly involved nerves (with muscles) are:

- Suprascapular nerve (supra- and infraspinatus muscle),
- Musculocutaneous nerve (biceps and brachialis), and
- Axillary nerve (deltoid and teres minor)

The paralysis of shoulder abductors (deltoid, supraspinatus), external rotators (infraspinatus, teres minor), elbow flexors and forearm supinators (biceps, brachialis) result in:

- Arm hang by side in adduction
- Internal rotation at shoulder
- Extended elbow
- Pronation at forearm.

The classic deformity is known as **"policeman hand/waiter tip hand"**.

What is Erb's point?

Erb's point is a point at upper trunk of brachial plexus with confluence of six nerves (C5, C6 root, suprascapular nerve, nerve to subclavius, anterior and posterior divisions of C5 and C6).

It is located 2–3 cm above the clavicle and is named after Wilhelm Heinrich Erb.

FOOTDROP

Q. What do you mean by footdrop?

Footdrop means inability to actively dorsiflex the ankle.

Q. Which nerve paralysis can lead to footdrop?

1. Lateral popliteal/common peroneal nerve (CPN) injury
2. Sciatic nerve (SN) injury

Q. Are there any other causes for footdrop other than nerve injury?

Complete severance of extensor tendons especially tibialis anterior (TA) can also lead to footdrop even though the nerve is intact.

Q. How will you differentiate that footdrop is due to CPN or SN injury?

Understand the basic anatomy before you can answer this question. In the beginning of its course in gluteal and upper thigh region, sciatic nerve (SN) first supplies the knee flexors (hamstring). After descending the back of thigh, SN divides into common peroneal nerve (CPN) and the tibial nerve at the upper end of popliteal fossa.

The tibial nerve: It continues straight down to supply muscles on back of calf followed by the muscles in the plantar aspect of the foot. Important ones are plantar flexor at ankle (tendo Achillis), tibialis posterior, other toe long flexors and foot plantar intrinsic muscles.

CPN: It winds around the fibular neck and then divides into superficial and deep peroneal nerve. It supplies the various muscles of anterior and lateral compartment of the leg (Table 1.2).

Table 1.2: Summary of important muscles of anterior, lateral, and posterior compartment of the leg

Muscle	Function
Anterior compartment supplied by deep peroneal nerve	
Tibialis anterior	Dorsiflexion at ankle, inversion at subtalar joint
Extensor hallucis longus	Great toe dorsiflexion
Extensor digitorum longus	Small toe extension
Peroneus tertius	Dorsiflexion at ankle joint, eversion at subtalar joint
Lateral compartment supplied by superficial peroneal nerve	
Peroneus longus	Eversion, plantar flexion
Peroneus brevis	Eversion, plantar flexion
Posterior compartment and foot muscles supplied by tibial nerve	
Tendoachilles	Plantar flexion at ankle
Tibialis posterior	Plantar flexion at ankle, inversion at subtalar joint
Flexor hallucis longus	Flexion of great toe
Flexor digitorum longus	Flexion of small toes

So **in case of SN palsy**, patient will lose

1. Knee flexion
2. All movements at the ankle (dorsiflexion, plantar flexion, eversion and inversion) resulting in flail foot (no strength as all foot intrinsic and extrinsic muscles paralysed)

Note: Knee extension is normal as knee extensors (quadriceps) are supplied by femoral nerve.

In case of CPN palsy, patient will lose ability to:

1. Dorsiflex the ankle
2. Dorsiflex the great toe
3. Dorsiflex the small toes
4. Eversion of subtalar joint

Note: In **case of isolated tibial nerve palsy**, patient will lose his ability to plantarflex and invert whereas he will retain dorsiflexion of ankle, toes and eversion of subtalar joint.

So, by examining the various functions of knee and ankle-foot, we can differentiate between sciatic nerve, tibial nerve and common peroneal nerve palsy.

Q. Which splint is used for footdrop?

Ankle-foot orthosis (AFO) (Fig. 1.13).

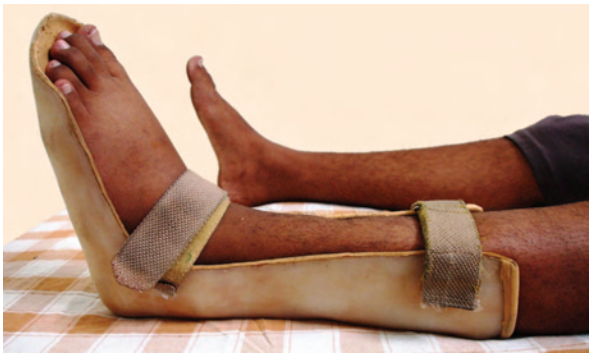


Fig. 1.13: Clinical picture of ankle-foot orthosis (AFO) applied on left foot-ankle

Q. What will happen if AFO is not used?

The foot will remain in plantar flexion position resulting in equinus deformity at the ankle.

Q. What could be done if SN/CPN fail to recover after a period of conservative treatment?

1. Explore the nerve
2. Neurolysis, neurorrhaphy with/without nerve graft
3. Tendon transfer

Q. Which tendon transfer is done for footdrop due to CPN palsy?

Tibialis posterior (TP) is transferred to correct footdrop. It is transferred onto the dorsum of foot. There are two classic tendon transfers for footdrop.

Ober's transfer: Circumtibial re-routing of TP and anchoring onto the 3rd metatarsal base.
Watkin's interosseous transfer: Re-routing through the interosseous membrane onto the 3rd metatarsal base.

Note: Do not forget to check the power of tibialis posterior as it could also be paralyzed or ineffective. If that is the case, then it cannot be transferred. Many at times, students are allotted case of sciatic nerve palsy or Volkmann's ischemic contracture of the leg and foot following a compartment syndrome of the leg where all/most muscles of leg and foot are ineffective. Answering TP transfer in such a case where no muscles are working a mistake!

Q. What is the role of examining ankle and foot movement in a case of foot-ankle paralysis?

If there is stiffness in the ankle-foot complex, the tendon transfer is not suitable till the ROM is restored to functional level.

Q. What should be done if ankle is stiff in equinus position?

1. First, patient should be advised physiotherapy of ankle and foot to restore the ROM of stiff joints.
2. If conservative measures (physiotherapy) fail, then surgical release of stiff joint can be performed.

Note: Commonly, tendo Achillis lengthening and posterior ankle joint capsule release is done to correct ankle equinus. It is important to have a good range of movements at the involved joint before tendon transfer. There is no point supplying electricity to a fan which is jammed and rusty!

Q. What will you do if there is no tendon remaining to be transferred?

Such patients can be managed in two possible ways:
a. AFO for the ankle-foot to keep ankle foot in functional position.
b. Arthrodesis of ankle and subtalar joint in neutral position.

Note: Avoid ankle-foot arthrodesis in patients with sensory loss over the foot especially over the plantar aspect as it may result in non-healing ulcers due to a rigid-foot result from arthrodesis.

Q. What are the other causes of footdrop?

Apart from other general causes of nerve palsy, there are some other causes of footdrop.

1. Infective:
   a. Poliomyelitis—history of polio in childhood

b. Hansen's disease—history of patchy loss of sensation over body, thickened and tender nerves like CPN, greater auricular, ulnar, superficial radial nerve

2. Traumatic

a. CPN, sciatic nerve injury

b. Spinal cord injury

3. **Lumbar intervertebral disc prolapse:** History of lifting heavy weight, low back pain with radiation to the lower limb, Laségue and straight leg raising test positive
4. **Spastic cerebral palsy (since birth)**
5. **Cerebrovascular accidents:** History of CVA
6. **Charcot-Marie-Tooth** disease
7. Rupture of tibialis anterior and other ankle-foot extensor tendons
8. **Neoplastic:** Tumors compressing the nerves

Note: In non-traumatic nerve palsy, student must take history to rule out other causes of nerve injury.

NON-UNION

Common Presentation of Patient with Non-union

Case summary: A 32-year-old male case of inability to bear weight over his right lower limb. He had a RTA 6 months back following which he sustained fracture of tibia and fibula. He was taken to a local hospital where fracture was managed by a POP cast. Cast was changed twice in three months. Currently, patient cannot bear weight over right lower limb. There is no pain over the fracture site. General and systemic examinations are normal. Local examination of right tibia revealed painless abnormal mobility over lower fourth of shaft of the tibia. The right lower limb is short by 3 cm and shortening is in the tibia. Knee movements are normal but ankle joint is stiff having only 10° plantar and dorsiflexion each. Neurovascular examination is normal.

Q. What is the clinical diagnosis?

The clinical diagnosis is non-union of tibia with stiffness of ankle joint and shortening of tibia.

Q. Why do you say so?

It is because of the following reasons.

1. History of RTA
2. Presence of **painless abnormal mobility** over the fracture site (lower fourth of shaft tibia)
3. Stiffness of the ankle joint
4. Shortened tibia

Q. What is the main evidence to say that it is a non-union of the tibia?

Painless, abnormal mobility at the fracture site

Q. How will you confirm the diagnosis?

Plain X-ray of the leg (anteroposterior and lateral view).

Q. What are the common finding in a X-ray of non union?

Following are the common findings in the X-ray of non-union (Fig. 1.14)

1. Gap at the fracture site
2. Closed medullary canal
3. Sclerotic ends of fracture
4. Callus could be absent, minimal or in excess.

Q. What are the principles of treatment of non-union?

The treatment of non-union consists of the following principle:

a. Correct any systemic factor, if present: Stop smoking; correct anemia, control diabetes mellitus
b. Local principles involve
   1. **Open reduction** of the fracture site

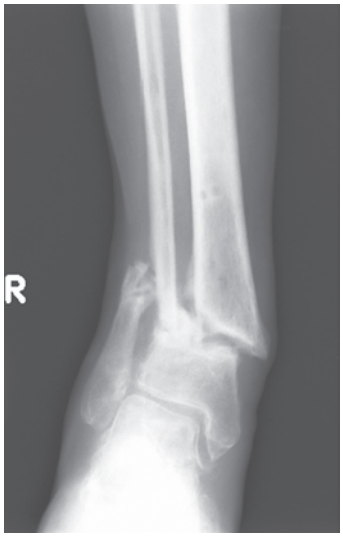


Fig. 1.14: Plain radiograph of tibia and fibula showing non-union at lower fourth shaft with gap at fracture site

2. **Excision of sclerotic ends** with **freshening of bone ends** till fresh bleeding is evident
3. **Open the medullary canal**
4. **Internal or external fixation:** If the patient has history of infection/currently having local infection, external fixation is a safer option. If there was no infection or infection is completely cured, then internal fixation can be safely performed)
5. **Bone grafting:** Essential for atrophic type whereas hypertrophic type may not require grafting.**

**Bone grafting should not be done if there is slightest doubt of infection otherwise the graft will act like a dead piece of bone and will become a sequestrum and infection will flare up.

Q. What is atrophic and hypertrophic type of non-union?

Atrophic and hypertrophic type of non-union is a radiographic presentation/classification of callus in patients with non-union.

Q. What is the radiological classification of non-union?

This radiological classification (atrophic or hypertrophic type) is based upon presence/absence of callus

1. *Atrophic type:* Poor callus
2. *Hypertrophic type:* Excess callus

Q. What is the rationale behind this classification?

1. ***Atrophic type:*** It indicates "poor vascularity or deficient biological activity at the fracture site" resulting in nil or minimal callus.

2. ***Hypertrophic type:*** It indicates "poor mechanical stability at the fracture site" wherein the callus is present in reasonable or in abundance but two ends of callus could not stabilise due to persistent instability (or poor mechanical stability) at the # site.

Q. How can one use this classification in treating the patient?

1. Since atrophic type of non-union reflects poor vascularity or biological activity at the fracture site, ***fresh autologous cancellous bone graft or vascularized bone graft*** is applied at the fracture site along with fracture fixation to stimulate the fracture union after the fixation of fracture.
2. Since hypertrophic type of non-union reflects poor mechanical stability, the ***stability at the fracture site is improved by using stronger/larger/different implant (nail/plate) to stimulate union.***

Q. Define non-union?

Non-union of fracture is defined as 'when there is no clinical or radiological evidence of union of fracture for three consecutive months, and there is no possibility of fracture union without further intervention'.

US FDA definition of non-union

The US Food and Drug Administration (FDA) defines a non-union as a fracture that is at least 9 months old and has not shown any signs of healing for 3 consecutive months.

Q. What are the local causes of non-union?

Common local causes are:

1. Infection
2. Inadequate vascularity
3. Intact fellow bone
4. Interposition of soft tissue
5. Inadequate reduction
6. Inadequate immobilization
   (Note: All causes start with "I")

Important systemic causes are:

1. Smoking
2. Diabetes
3. Osteoporosis
4. Malnutrition and vitamin deficiency

Q. Why lower end tibia is more prone for non-union?

Lower end tibia is prone for non-union because of precarious vascularity. There is minimal soft tissue envelope around the lower end of tibia reducing the vascularity of the lower end of tibia.

Q. Why does infection cause non-union?

Infection result in destruction to the callus, damage to the vascularity of the soft tissue envelope around the bone and fibrosis at the fracture site resulting in non-union.

Q. Why open fracture causes non-union?

Open fracture causes non-union because:

a. Soft tissue damage, periosteal stripping compromise the local vascularity
b. Infection
c. Loss of bone fragment

Q. What are the common sites of non-union?

Upper Limb

1. *Scaphoid waist fracture:* Disrupted vascularity which enters scaphoid from distal to proximal
2. *Lateral condyle # of humerus:* The fragment gets rotated and displaced due to pull from extensor muscles attached over the lateral condyle.

Lower Limb

1. *Fracture neck femur:* Disrupted retinacular blood supply, absent cambium layer in periosteum, no fracture hematoma formation as it gets lysed by synovial fluid
2. *Fracture lower end of tibia #:* Less vascularity at the lower end, poor soft tissue envelope over the lower end of tibia
3. *Fracture talus neck:* Disrupted blood supply

Q. How you would have treated a patient with a non-union and local infection (osteomyelitis) as well?

It can be treated as "two-staged procedure".

First, the local soft tissue and bony infection must be treated as per standard principles to treat chronic osteomyelitis.

Once infection heals completely, one should treat non-union on the standard principles.

Q. So, it means one has to undergo two-stage procedure for infective non-union. Is there a way to treat both simultaneously?

Yes. It can be treated in the same setting by Ilizarov's technique wherein, the infected bony segment is excised completely and the new bone is regenerated using Ilizarov's technique or limb reconstruction system.

Q. What is delayed union?

Delayed union refers to ***a fracture in which union is not complete within the interval expected for that specific fracture***. Nevertheless, the clinical and radiographic evidence of healing is present and if given a proper milieu, has the potential to unite.

Q. What is the difference between delayed and non-union?

In non-union, the biological process of union is **completely halted** and there are no more biological attempts at union of fracture whereas in delayed union, the biological process of union is going on, albeit slow. The non-union will not unite without surgical intervention whereas in delayed union, fracture may unite with the current treatment plan without any intervention.

Q. What is the treatment of delayed union?

1. **Wait and watch:** Regular follow ups and continuing the present method of treatment
2. **Correct any local or systemic factor responsible for delayed union.**

   If above two does not work, then
3. **Bone marrow injections** at the fracture site to accelerate the healing process
4. **Bone grafting** at the fracture site
5. **Ultrasonic stimulation** at the fracture site

Q. How will you manage ankle stiffness?

Ankle stiffness can be managed by:

1. Physiotherapy
2. If physiotherapy fails to achieve any significant improvement in ROM, surgical release of stiff joint should be performed (*refer* to page 47: Stiff joint)

Q. How will you manage shortening of tibia?

Since shortening is only 3 cm, it can be managed by shoe raise or limb lengthening.

Q. What are the various ways in which shortening of a limb can be managed?

General principles of managing shortening of limb

1. *Shortening <4 cm:* Shoe raise
2. *Shortening >4 cm:* Limb lengthening by Ilizarov's technique.
   In paediatric patients with shortening, two more undermentioned methods can be utilized
3. Shortening of other limb
4. Epiphysiodesis

Q. How will you manage shortening of tibia?

Since shortening is only 3 cm, it can be managed by shoe raise or limb lengthening.

REFLEX SYMPATHETIC DYSTROPHY (RSD) OR SUDECK'S DYSTROPHY/CRPS

Common Presentation of Patient with Reflex Sympathetic Dystrophy (RSD)

Case summary: A 35-year-old male gave history of fall on outstretched left hand following which he sustained fracture of lower end radius. He underwent closed reduction and below elbow cast application for 6 weeks. After removal of cast, patient was advised physiotherapy of hand and fingers. However, patient noticed swelling of his fingers and hand. He experienced severe pain while moving the wrist and fingers and altered sensations over his hand. Now, after 12 weeks of injury, the swelling is still present over the hand and finger with quite painful movements of joints of wrist and finger. Examination revealed shiny skin with mottled bluish hue, spindle shape fingers with pulp atrophy with cold and sweaty feel over the skin of wrist-hand. Movements of wrist and finger are grossly restricted and painful. Mild paraesthesia was noted over the hand without any motor deficit. There was bony irregularity at distal end of radius but no abnormal mobility elicited.

Q. What is the clinical diagnosis?

The clinical diagnosis is united fracture lower end radius with reflex sympathetic dystrophy/RSD/complex regional pain syndrome (CRPS).

Q. Why do you say so?

I say so because of the following reason:

History

1. History of trauma with POP applied for fracture lower end radius
2. Swelling of hand and finger (Fig. 1.15)
3. Exaggerated pain while attempted movements
4. Painfully restricted movements of wrist and fingers even after a few weeks of active and passive mobilization/physiotherapy.

Examination

5. Bluish mottled hue of hand and finger.
6. Shiny skin with spindle shaped finger and finger pulp atrophy

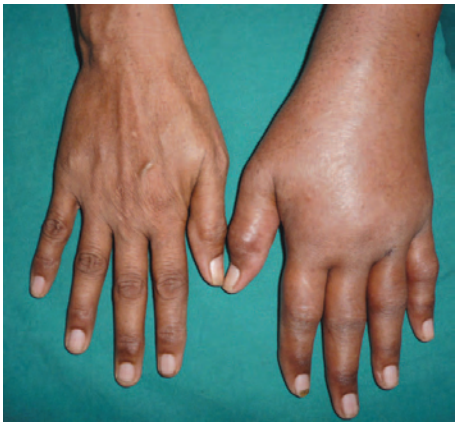


Fig. 1.15: Clinical photograph of left hand with RSD showing swelling and spindle shape fingers

7. Cold, sweaty hand
8. Grossly restricted and painful movement of fingers
9. Altered sensations (paraesthesia)

Q. How will you confirm the diagnosis?

Diagnosis is essentially clinical. However, some investigation may aid in diagnosis.

a. X-ray shows **patchy osteoporosis** in the bones of wrist and hand (Fig. 1.16).
b. **Phase III positive bone scan**

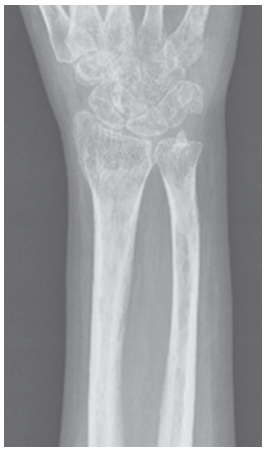


Fig. 1.16: Plain X-ray of left wrist and hand shows united fracture of distal radius with patchy osteoporosis in bones of forearm, wrist and hand

Q. What are the other synonyms for RSD?

1. Sudeck's osteodystrophy
2. Complex regional pain syndrome

Q. Which terminology is more appropriate?

Currently, CRPS is more appropriate and acceptable terminology.

Q. What it CRPS type 1 and type 2?

Type 1 CRPS: CRPS without any underlying cause
Type 2 CRPS: CRPS after a nerve injury

Q. What is causalgia?

Causalgia is a type 2 CRPS or RSD associated with nerve injury.

Q. What is the fundamental pathology in RSD?

The principal pathology in RSD is **idiopathic overactivity of sympathetic nervous system** (or exaggerated response to injury) in the affected limb **due to constant pain stimulus.**

Q. What are the predisposing factors which can lead to RSD?

a. Patient not mobilizing the affected limb effectively
b. Persistent edema
c. Pain, if not controlled well, could result in RSD

Note: By controlling these predisposing factors pre- and postoperatively, RSD could be mostly prevented.
a. Encourage active and passive mobilization of limb
b. Limb elevation and compression bandage to prevent edema
c. Adequate analgesics to control pain

Q. How will you treat patients with RSD/CRPS?

The aim of the treatment is

a. Break the pain-stiffness cycle by appropriate conservative measures such as

1. Appropriate **analgesia** (NSAIDs, opioids, etc.)
2. **Control limb edema** by limb elevation, compression bandage
3. Prolonged supervised physiotherapy to **mobilize the limb** is the key to success
4. Other medical measures:
   a. Calcium channel blockers: Nifedipine
   b. Pregabalin, Gabapentin
   c. Vitamin C

If above conservative measures for few weeks fail to provide relief, one could opt for

b. Blocking the sympathetic nerve supply to the limb

Block the sympathetic ganglion supplying the extremity by locally injecting lignocaine/phenol/alcohol. For example, stellate ganglion for the upper limb which is situated anterior to transverse process of C7 vertebra and coeliac ganglion for lower limb situated next to L1 vertebra.

The sympathetic ganglion block decreases pain in the affected part by blocking sympathetic response enabling the clinician to aggressively mobilize the limb and reduce stiffness.

Q. What you would have done if this patient presented with grossly malunited lower end of radius affecting his hand function with RSD?

First, RSD must be completely treated to reduce pain and swelling along with restore the movements.

Then **reassess the malunion** affecting the functional disability. If needed, then treat malunion.

Note: However, **no elective surgical intervention** should be done in presence of RSD. (Note: Also, all malunited fractures may not need surgical intervention)

Q. Why not to treat malunion in the setting of RSD?

RSD will aggravate if any surgical procedure is performed without treating RSD.

Note: Any surgical procedure around a joint with RSD is usually contraindicated as it can further aggravate the RSD by exacerbating pain stimulus.

STIFF JOINT

Common Presentation of Patient with Stiff Joint

Case summary: A 35-year-old man case of pain and difficulty in bending his right knee for 4 months. Four months back, following a RTA, he sustained distal femur intercondylar fracture. He underwent open reduction and internal fixation (ORIF)of fracture by a plate followed by knee immobilisation in a brace for three weeks. Three weeks later, active knee mobilisation was started. There was no history of infection or open fracture. Currently, he is full weight bearing but unable to squat or sit cross leg. General and systemic examination were normal. Local examination revealed a 16 cm long primarily healed scar over lateral aspect of thigh. The knee flexion range of movement was 0–90°. The limb lengths on both sides were equal and neurovascular examination was normal.

Q. What is the clinical diagnosis?

Healed fracture of lower end of femur with stiffness of the knee.

Q. Why do you say so?

I say so because of the following reasons:

History

1. History of trauma
2. ORIF of distal femur fracture and immobilisation (both could lead to stiffness)
3. Full weight bearing (indicates a healed fracture)

Examination

4. Decreased range of flexion movement (0–90°) (Fig. 1.17)
5. Irregular lower end femur (suggestive of a fracture)

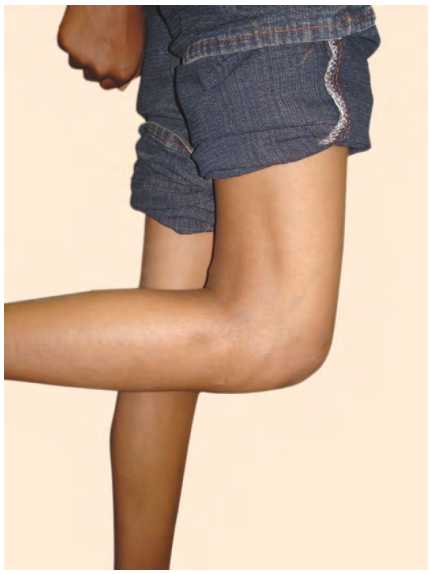


Fig. 1.17: Right knee showing decreased movements

Q. What is role of massage or forcible passive physiotherapy (often done by quacks) in the history of a stiff joint?

Massage and/or forcible passive physiotherapy could lead to myositis ossificans (read Myositis ossificans on page 13).

Q. How will you proceed?

I would like to take an X-ray.

Q. What would you see on X-ray?

X-ray can reveal

- United fracture with implant *in situ*.

Q. What is the treatment of a patient with stiff joint?

1. Physiotherapy
   a. Gentle active and assisted passive mobilization
   b. Moist heat, short wave diathermy (SWD)

Note: Moist heat and SWD help in relaxing the tight fibrous tissue and then active and gentle passive mobilisation of joint helps in regaining movements)

2. If physiotherapy for few weeks to months fails to result in significant improvement in range of movements, then surgical release could be considered to restore a functional or total range of movements.

Q. What surgery can be performed for a stiff joint?

In a stiff joint, one or more following structures can be tight. They can be released surgically.

1. **Skin** if scarred: Scar excision/lengthening (Z or V-Y plasty)
2. **Subcutaneous tissue** and **deep fascia:** Excise/divide them
3. **Muscles** are fibrotic and contracted: Concerned tendon should be lengthened using **Z or V-Y plasty.**
4. **Joint capsule is tight:** Capsulotomy/capsulectomy
5. **Intra-articular adhesions:** Release
6. **Finally a gentle manipulation:** It may also help in getting over some adhesions in and around joint.

Q. How can we prevent stiffness of joint after fracture or any injury?

Stiffness of a joint can be prevented or minimised by appropriate management of fracture or injury and early mobilization and physiotherapy of the joint.

Q. What do you mean by ankylosis?

Ankylosis is an abnormal stiffness of the joint means that the joint which exhibits hardly any movements.

Note: Ankylosis implies pathological fusion of the joint.

Q. What are the types of ankylosis?

There are two types of ankyloses: True and false

True ankylosis

1. Bony ankylosis
2. Fibrous ankylosis

(Read false ankylosis on page 17)

Q. What is bony and fibrous ankylosis? How do you differentiate between them?

In **bony ankylosis**, after the denudation of cartilage due to trauma/infection/inflammation; new bone forms across the two articular surfaces bridging two articular ends of bone resulting in complete fusion of the two articular surfaces. So

there is no movement elicited across the joint and is painless **(painless, no movements)**.

In **fibrous ankylosis,** dense and tough fibrous bands run across the two articular surfaces and in the joint. Since there are fibrous bands running across, some movement can be elicited, and that is painful **(painful, jog of movement)**.

Q. What will be the X-ray finding in two types of ankyloses?

Bony Ankylosis

- Bony trabeculae cross the joint joining the two articular surface, and
- Joint space will be obliterated.

Fibrous Ankylosis

- Joint space is seen
- No bony trabeculae crossing the joint.

Q. What is the treatment of choice of bony ankylosis?

In case of bony ankylosis, physiotherapy is of no use as there is bony bridge between the two ends which would not allow any movements to be regained by physiotherapy, surgery is the preferred treatment option.

1. **If the joint is in sound functional position of bony ankylosis:** It could be left alone as long as it satisfies the functional demands of the patient.
2. **If the joint is in unsound position of bony ankylosis,** it should be converted into fusion (arthrodesis) in sound functional position.
3. However, above two options would not restore the movement at the fused joint. To restore ROM at the joint, total joint replacement can be performed.

Functional position means the position of joint in which several important functions can be performed. Functional position of various joints is mentioned below

Functional position of upper limb joints:
1. Shoulder: 30° forward flexion; 30° abduction and 30° internal rotation
2. Right elbow: 90° flexion; left elbow: 30° flexion
3. Forearm: Mid prone
4. Wrist and hand: Glass holding position

Lower limb joints
1. Hip: Neutral in extension and slight abduction
2. Knee: Full extension
3. Ankle and foot: Neutral

Q. If there is myositis ossificans, what will you do?

Management will depend upon the type of myositis ossificans (discussed in detail in chapter of myositis osssificans).

VOLKMANN'S ISCHEMIC CONTRACTURE (VIC)

Common Presentation of Patient with VIC of Forearm and Hand

Case summary: A 6-year-old boy case of pain and stiffness in his right forearm and hand. He had RTA one year back and fractured his both forearm bones. He was taken to a local hospital where an above elbow cast was applied after the fracture reduction, and was discharged. After reaching home, he developed severe pain in the forearm. He was rushed to the nearest hospital where the cast was removed. Examination revealed painful passive stretch of his fingers. His fracture was further managed on above elbow slab. Later, he developed gradually progressive flexion deformity in the hand and finger. Presently, he has difficulty in using his hand. Examination reveals wasting of forearm and hand muscles. On dorsiflexion of the wrist, the fingers become flexed at MCP and IP joint while palmar flexion of the wrist allows straightening of the fingers at the IP joint. There is sensory loss over forearm and hand over the median and ulnar nerve distribution areas with motor weakness in muscles supplied by the ulnar nerve.

Q. What is the clinical diagnosis?

Post-traumatic Volkmann's ischemic contracture (VIC) of right forearm and wrist.

Q. Why do you say so?

It is because of following reasons.

History

1. History of trauma with fracture forearm bone (susceptible for compartment syndrome)
2. History of above elbow cast application followed by severe pain in forearm (symptom of compartment syndrome)

Examination

3. Wasting of forearm muscles (Fig. 1.18)
4. Volkmann's sign present (this is the most important late clinical sign of VIC)
5. Median and ulnar nerve involvement

Fig. 1.18: Clinical photograph of VIC of right forearm reveals gross wasting of forearm muscles and flexion contracture of fingers

Q. What is Volkmann's sign?

In Volkmann's sign; when the wrist is dorsiflexed, the fingers become palmar flexed and cannot be extended passively or actively. However, when the wrist is brought to neutral or palmar flexion, the fingers can be extended passively or actively.

VIC was first described in 1881 by Richard von Volkmann of Germany.

Q. What is the mechanics of Volkmann's sign?

It is due to **bow stringing effect** of fibrosed flexor muscle tendon unit over a fixed length of bone.

Q. What is Volkmann's ischemic contracture?

VIC is the **contracture and fibrosis of flexor muscles of forearm (deep and superficial)** after an episode of ischemia to the muscles due to the compartment syndrome.

Q. What is Volkmann's ischemia?

Volkmann's ischemia is also known as **compartment syndrome.**

Q. Which areas are prone for Volkmann's ischemia?

Although Volkmann's ischemia or compartment syndrome could occur in any area, it frequently occurs in following locations:

a. *Injury around the elbow:* Fracture supracondylar humerus, elbow dislocation
b. Fracture both bones forearm
c. Fracture both bones leg
d. Fracture of metacarpal or metatarsals
e. *Injuries around the knee:* Knee dislocation, fractures around the knee.

Note: Compartment syndrome usually occurs where there are parallel bones (forearm, leg, hand or foot).

Q. What is compartment syndrome and its pathophysiology?

Normally the pressure in the osseofibrous compartment is less than 10 mm Hg. If the intracompartment pressure increases more than 30 mm of Hg, it indicates an established compartment syndrome.

The gradually increasing intracompartment pressure from 10 mm Hg and above results in decreased venous outflow while arterial inflow is maintained. This causes increased venous capillary pressure which results in increased transudation and exudation in extravascular space further increasing the compartment pressure.

The increasing compartment pressure causes ischemia to the smaller arteries supplying the muscle and the nerves resulting in painful gradual onset global ischemia of the muscles. If remains untreated within the stipulated time, nerves and muscles suffer irreversible damage.

a. *Muscles:* Ischaemic necrosis followed by fibrosis and contracture.
b. *Nerves:* Ischemia of arteria nervosa results in damage to nerves followed by partial or total sensorimotor dysfunction.

c. Complete arterial obstruction of the limb is very rare. Hence, gangrene is quite rare in compartment syndrome.

Q. What is the commonest cause of compartment syndrome?

Tight bandage/cast in the areas prone for compartment syndrome.

Q. What are other causes of compartment syndrome?

Burns, vascular injuries, reperfusion injuries, crush injuries, bleeding disorders, infections, intense athletic activity, and poor positioning during surgery.

Q. How do you clinically diagnose compartment syndrome?

The **most common clinical symptom is "pain out of proportion of the injury".**

Normally after the injury, pain decreases once patient receives medical aid in form of analgesics and splintage/cast application of the affected limb.

But in case of compartment syndrome, pain is quite severe and even after primary medical aid or splint/cast application, it remains very uncomforting or out of proportion of the injury and patient keeps demanding more analgesics.

Q. Why patients with compartment syndrome have pain out of proportion of injury?

It is due to the ischemia to the nerves (crying of the dying nerves!).

Treatment of compartment syndrome is

1. Remove all tight bandages, splint or cast
2. Urgent fasciotomy.

If untreated or treatment is delayed, it could result in VIC.

Q. What is the treatment of VIC of forearm and hand?

a. ***Mild cases:*** Splinting in functional position, stretching of tight structures.

b. ***Moderate cases***

1. Excision of fibronecrotic muscle mass in the compartment
2. Flexor-pronator muscle sliding surgery ***(Maxpage operation)***. The flexor groups of muscles are released (slided) from medial epicondyle.
3. Lengthening (Z plasty) of muscle tendon unit.
4. Neurolysis of the nerves as they are surrounded by thick fibrous tissue
5. Bone shortening, proximal row carpectomy, wrist arthrodesis

c. ***Severe cases***

1. Free muscle transfer
2. Nerve grafting and reconstructions
3. Flap coverage of skin.