35 Hand and Wrist Anatomy and Examination

Steven C. Haase

I. Before you begin

A. Remember the ABCs of trauma for all injured patients—do not assume that there is an isolated hand/wrist injury.

B. Take a focused history.

1. Age, handedness, vocation and avocations, smoking status.
2. Time of last meal and drink, allergies, current medications, medical problems.
3. Exact mechanism of injury (crush/avulsion, sharp/dull, dirty/clean).

C. Order appropriate x-rays.

D. Examine the joint(s) proximal and distal to the injury to detect associated injuries in the limb.

E. Administer tetanus booster and antibiotics if needed (open injuries).

II. Common abbreviations

A. Digits

1. IF: Index finger
2. MF: Middle finger
3. RF: Ring finger
4. LF: Little finger (or SF, small finger)

B. Joints

1. IPJ: Interphalangeal joint
2. DIPJ: Distal interphalangeal joint
3. PIPJ: Proximal interphalangeal joint
4. MPJ: Metacarpophalangeal joint

C. Muscles

1. FCR/FCU: Flexor carpi radialis/ulnaris
2. FPL/FPB: Flexor pollicis longus/brevis
3. FDS: Flexor digitorum superficialis
4. FDP: Flexor digitorum profundus
5. ECRL/ECRB: Extensor carpi radialis longus/brevis
6. ECU: Extensor carpi ulnaris
7. EDC: Extensor digitorum communis
8. EIP: Extensor indicis proprius
9. EDM: Extensor digiti minimi (or EDQ, extensor digit quinti)
10. EPL/EPB: Extensor pollicis longus/brevis
11. PL: Palmaris longus
12. PT: Pronator teres
13. PQ: Pronator quadratus
14. AdP: Adductor pollicis
15. APL/APB: Abductor pollicis longus/brevis

III. Vasculature

A. Arterial supply

1. The radial artery supplies the deep palmar arch.
2. The ulnar artery supplies the superficial palmar arch.
3. The radial artery is usually dominant, but ulnar dominance and codominance can exist.

B. Venous drainage

1. Dorsal (subcutaneous) venous network and palmar venous arch.

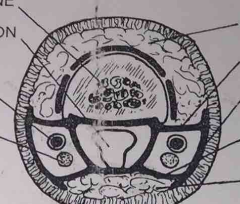

Fig. 35-1. Cross-section of the digit. (From Sherman R and Isenberg S Principles of soft-tissue reconstruction of the lower extremity. In *Plastic, Maxillofacial, and Reconstructive Surgery*, 3rd edition. Georgiade G, Riefkohl R, Levin S. Philadelphia, Williams & Wilkins, 1997. With permission.)

2. Drain into the cephalic vein (originates near the "anatomic snuffbox") and the basilic vein (originates on the dorsoulnar aspect on hand).

C. Allen's test: Compress the radial and ulnar arteries at the wrist, and have the patient make a tight fist (to exsanguinate) and then relax; release one or the other artery to check blood supply to the hand and patency of the palmar arch via that artery.

D. A digital Allen's test can also be performed.

E. Arterial location relative to accompanying nerves.

1. In the palm: Arteries are volar to the nerves.
2. In the digits: Arteries are dorsal to the nerves (Fig. 35-1).

F. Capillary refill should be 2 to 3 seconds; best place to check is the nail bed or eponychial fold.

IV. Nerves

A. Radial nerve anatomy

1. Passes between heads of the supinator and enters the forearm between the brachioradialis and the ECRL/ECRB.
2. Branches
   a. Posterior interosseous nerve: Innervates the EDC, EDM, APL, EPB, EPL, EIP, and ECU; sensory branch to the wrist capsule (deep within fourth dorsal compartment of the wrist); makes a good donor for digital nerve grafts.
   b. Superficial radial nerve: Sensory to the dorsum of the thumb, first web space, IF, MF, and radial RF up to the level of the PIP joints (Fig. 35-2).

B. Median nerve anatomy

1. Enters the forearm with the brachial artery, emerging between the heads of the pronator teres; innervates the pronator teres, FCR, palmaris longus, and FDS.
2. Branches.
   a. Anterior interosseous nerve innervates FPL, radial two FDP (IF and MF), and pronator quadratus; sensory to wrist capsule.
   b. Remainder of median nerve enters the carpal tunnel.
      (1) Motor branch: Innervates the two lumbricals, **o**pponens pollicis (OP), **A**PB, and **F**PB (LOAF).
      (2) Sensory: Palmar cutaneous branch, digital nerves (thumb, IF, MF, and radial RF) (Fig. 35-2).

C. Ulnar nerve anatomy

1. Enters forearm behind the medial epicondyle; innervates the FCU and ulnar two FDP (RF and LF).
2. Travels under the FCU to the wrist, forming the following branches.
   a. Deep palmar branch: Innervates abductor digiti minimi, flexor digiti minimi brevis, opponens digiti minimi, all interosseous muscles, medial lumbricals (two) and AdP.

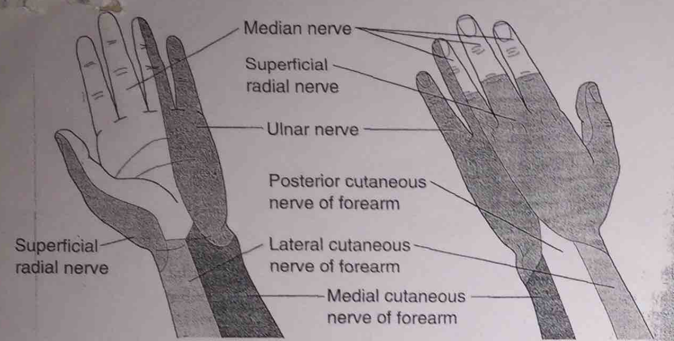

Fig. 35-2. Cutaneous sensory innervation of the hand, volar and dorsal surfaces.

- **b.** Superficial branch: Innervates palmaris brevis; sensory palmar digital nerves to LF and ulnar RF (Fig. 35-2).
- **c.** Dorsal branch: Sensory to the dorsum of the LF and ulnar RF (Fig. 35-2).

3. Martin Gruber anastomosis: An anatomic variant in which the median nerve crosses over to contribute to the ulnar nerve. The two most common variations are as follows.
- **a.** Median nerve in proximal forearm contributes to the ulnar nerve in the distal forearm.
- **b.** Anterior interosseus nerve contributes to ulnar nerve.

4. Riche-Cannieu anastomosis: A more distal variant in which ulnar fibers contribute to median nerve branches in the palm.

D. "Quick and simple" nerve exam

1. Median nerve: Sensation at tip of IF (light touch and two-point discrimination); ability to make "OK" sign (demonstrates FPL, FDP, and OP).
2. Ulnar nerve: Sensation at tip of LF; ability to abduct/fingers "wave hello" and cross fingers (interossei working).
3. Radial nerve: Sensation at dorsal first web space; ability to give "thumbs up" (EPL working).

V. Tendons and muscles

A. Flexors

1. Flexor tendon zones (Fig. 35-3)
- **a.** Zone 1: Middle of middle phalanx to fingertip (distal to insertion of FDS, includes only FDP).
- **b.** Zone 2: Distal palmar crease to middle of middle phalanx (both FDS and FDP tendons, with FDP volar, proceeding distally).
- **c.** Zone 3: Transverse carpal ligament to distal palmar crease.
- **d.** Zone 4: Under transverse carpal ligament (in carpal tunnel).
- **e.** Zone 5: Proximal border of transverse carpal ligament to musculotendinous junction.

2. Flexor muscle mass originates largely at the medial epicondyle.

3. The carpal tunnel contains the median nerve and nine tendons: FDP (four), FDS (four), and FPL (Fig. 35-4).
- **a.** FDS is superficial to the FDP in the carpal tunnel.
- **b.** FDS (MF and RF) is superficial to FDS (IF and LF)—the "hard way" to cross your fingers.

4. Flexor digitorum superficialis
- **a.** Splits into radial and ulnar slips prior to inserting into the proximal aspect of the respective middle phalanges.

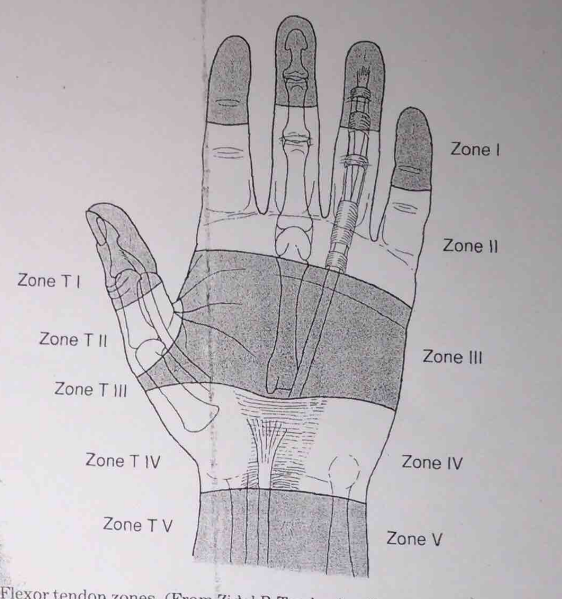

Fig. 35-3. Flexor tendon zones. (From Zidel P. Tendon healing and flexor tendon surgery. In *Grabb and Smith's Plastic Surgery*, 5th ed. Aston SJ, Beasley RW, Thorne CH, eds. Philadelphia, Lippincott-Raven, 1997. With permission.)

- **b.** FDP passes between the two slips of FDS, through a space called "Camper's chiasm."
- **c.** To test: Lay hand flat on exam table, palm up. With other fingers held in extension (blocks FDP), test active flexion (and to resistance) at each PIPJ.

5. Flexor digitorum profundus
- **a.** Inserts into proximal volar aspect of respective distal phalanges.

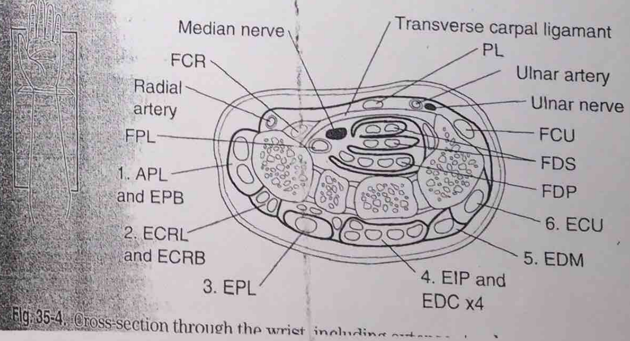

Fig. 35-4. Cross-section through the wrist

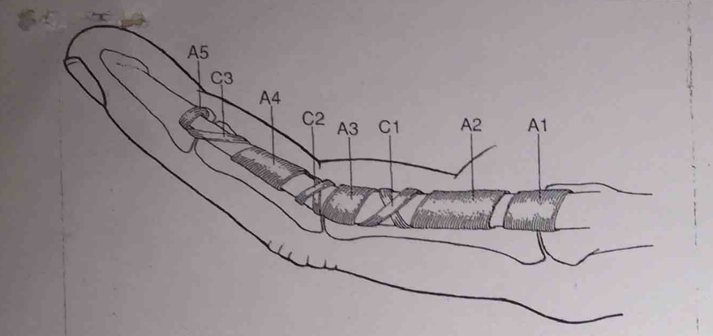

Fig. 35-5. Flexor sheath pulley system. A1–A5: annular pulleys; C1–C3: cruciate pulleys. A2 and A4 should be preserved to prevent bowstringing. (From Hoffman L. Mackenzie D., and Schwartz M. Common inflammatory disorders of the upper limb. In *Grabb and Smith's Plastic Surgery*, 5th ed. Aston SJ, Beasley RW, Thorne CH, eds. Philadelphia, Lippincott-Raven Publishers, 1997. With permission.)

b. To test: With PIPJ held in extension (examiner's pressure on middle phalanx), test active DIPJ flexion (and to resistance).

6. Flexor pollicis longus

a. Inserts into distal phalanx of thumb.

b. To test: Look for active flexion of IPJ ("OK" sign).

7. Pulleys: Prevent bowstringing and increase the mechanical effectiveness of pull across the joints (Fig. 35-5).

a. Five annular pulleys (A1–A5).

(1) A2 and A4 are the most critical to finger function; located on the proximal and middle phalanx, respectively.

(2) A1, A3, and A5 are at the MPJ, PIPJ, and DIPJ, respectively.

b. Three cruciate pulleys (C1–C3) interdigitate between A2, A3, A4, and A5.

c. The thumb has three pulleys: A1, A2, and oblique, which are located over the MPJ, IPJ, and proximal phalanx, respectively. The oblique pulley is the most mechanically important.

B. Extensors

1. Zones: The extensor tendons have nine zones. The odd-numbered zones (1–7) lie over joints, with the even-numbered zones in between (see Chapter 37, "Tendon Injuries and Tendonitis").

2. The extensor muscle mass (also known as the "mobile wad"): Originates largely at the lateral epicondyle.

3. Junctura tendinae: Interconnect tendons on the back of the hand and may mask an extensor rupture, as pull from a proximal tendon from another finger can be transmitted via a junctura to the distal extensor tendon of the injured finger.

4. Wrist compartments (radial to ulnar) (Fig. 35-4)

a. Compartment 1: APL, EPB

b. Compartment 2: ECRL, ECRB

c. Compartment 3: EPL

d. Compartment 4: EDC, EIP

e. Compartment 5: EDM

f. Compartment 6: ECU

5. EIP, EDM

a. These tendons lie *ulnar* to their accompanying EDC tendons.

b. To test: Independent extension of the IF and LF with other fingers flexed.

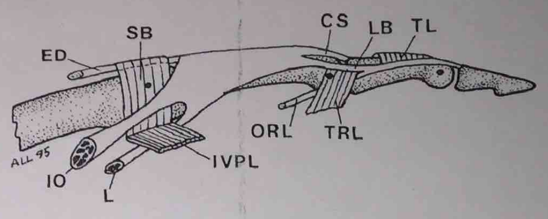

Fig. 35-6. Extensor tendon anatomy. (From Lluch A. Repair of the extensor tendon system. In *Grabb and Smith's Plastic Surgery*, 5th ed. Aston SJ, Beasley RW, Thorne CH, eds. Philadelphia, Lippincott-Raven Publishers, 1997. With permission.)

6. EPL: To test, check ability to raise thumb off table with hand prone.

7. Extensor mechanism (Fig. 35-6).

a. Extensor digitorum communis (plus EIP in the index finger and EDM in the small finger) expands over the MPJ to form the extensor hood.

b. The lumbrical and interosseus tendons join the hood laterally, providing MPJ flexion.

c. The extensor hood divides into three components over the proximal phalanx. The central slip of the extensor hood inserts on the base of the middle phalanx, while the two lateral bands continue distally, then fuse prior to inserting on the base of the distal phalanx (the terminal tendon).

C. Intrinsic muscles

1. ADP: Test for key pinch (thumb to side of IF) by having patient hold a piece of paper here and resist the paper being pulled away; if FPL takes over (DIPJ flexes), this is a positive "Froment sign" and implies that AdP function is absent or diminished (compare simultaneously to the opposite side).

2. Interosseus muscles: Test for abduction/adduction of fingers (including to resistance).

3. Intrinsic tightness test.

a. With the wrist in neutral position, hyperextend the MPJ to relax the extrinsics.

b. Measure passive flexion of PIPJ ("intrinsic tightness").

(1) Mild: 60 to 80 degrees.

(2) Moderate: 20 to 59 degrees.

(3) Severe: Less than 20 degrees.

4. Extrinsic tightness test

a. With the wrist in neutral position, flex the MPJ, to relax the intrinsics.

b. Check for resistance to PIPJ flexion, which will indicate extrinsic tightness.

VI. Bones and joints

A. Wrist

1. Radius and ulna articulate with each other and with the carpus.

2. Conceptually, the radius rotates around the fixed ulna at the wrist.

3. Normal alignment is reviewed in Chapter 36, "Fractures and Dislocations of the Hand and Wrist."

B. Carpus

1. Eight bones (scaphoid, lunate, triquetrum, pisiform, trapezium, trapezoid, capitate, hamate). Mnemonic: "**S**cared **L**overs **T**ry **P**ositions **T**hat **T**hey **C**annot **H**andle."

2. The pisiform is actually a sesamoid bone, imbedded in the FCU tendon.

3. Normal alignment: The capitate is collinear with the lunate and the third metacarpal.

4. Gilula's lines consist of arcs formed by the outlines of the carpal bones seen on anteroposterior radiographs of the wrist. The proximal row (scaphoid, lunate, and triquetrum) forms the proximal arcs, and the proximal edge of the distal row (trapezoid, trapezium, capitate, and hamate) forms the distal arc. The arcs should be smooth and continuous; otherwise, a fracture or dislocation may be present.

C. Fingers

1. Five metacarpals, 14 phalanges (three per finger, two per thumb)
2. Joint mechanics (see Chapter 36, "Fractures and Dislocations of the Hand and Wrist")

D. Normal ranges of motion (approximate)

1. Finger MPJ: 0 to 45 degrees hyperextension, 90 degrees flexion
2. Finger PIPJ: 0 degrees extension, 100 degrees flexion
3. Finger DIPJ: 0 degrees extension, 70 to 80 degrees flexion
4. Thumb MPJ: 10 degrees hyperextension, 55 degrees flexion
5. Thumb IPJ: 15 degrees hyperextension, 80 degrees flexion
6. Wrist: 70 degrees extension, 75 degrees flexion, 20 degrees radial deviation, 35 degrees ulnar deviation
7. Forearm: 70 degrees pronation, 85 degrees supination

VII. Tourniquet use

A. Never put on a finger tourniquet without establishing a prominent, obvious reminder to remove it (e.g., hemostat, sterile glove).

B. Blood pressure cuffs make good arm tourniquets in the emergency room.

C. Tourniquet pressure should be about 125 to 150 mm Hg above systolic pressure (usually set around 230–250 mm Hg).

D. Limit tourniquet time to 2 hours to prevent permanent damage.

1. Nerves are the most vulnerable structures to pressure and hypoxia.
2. Blood flow should be returned to the ischemic part for 5 minutes for every 30 minutes of ischemia (i.e., deflate tourniquet for 20 minutes every 2 hours).

VIII. Nerve blocks (see Chapter 8, "Local Anesthetics")

A. Median nerve

1. Place needle at the distal wrist crease between the PL and FCR; enter at 45-degree angle to the forearm. Usually a "pop" is felt as the deep fascia is penetrated.
2. If there is no "pop," insert until paresthesias or bone is encountered, at which time the needle is withdrawn slightly and 3 to 5 cc of local anesthetic is injected.

B. Ulnar nerve

1. Place needle just proximal to the ulnar styloid; enter behind the FCU tendon with the needle directed from ulnar to radial in the coronal plane.
2. Once the needle is just past the FCU, inject the anesthetic (3–5 cc).

C. Radial nerve

1. Must block a wide area: Start proximal to the radial styloid and inject the subcutaneous tissue.
2. Extend the field block in both directions (volar and dorsal) around the distal forearm.

D. Digital block

1. **Dorsal approach** (some think is less painful): Subcutaneous wheal over the extensor tendon to block the dorsal nerves. Two injections just proximal to the web, one on either side of the digit—advance the needle until the tip approaches the palmar skin surface and then withdraw while injecting slowly.
2. **Volar approach:** A subcutaneous wheal is placed directly over the flexor tendon and then laterally near the digital neurovascular bundles.
3. **Sheath approach:** Insert the needle volarly, just distal to the distal palmar crease, down into the flexor tendons. With slight pressure on the syringe plunger, withdraw the needle slowly until there is a loss of resistance (indicating injection into the potential space of the flexor sheath). Inject a couple of cc's of local. Sometimes, a fluid wave can be felt distally in the finger over the sheath. This technique reliably results in digital anesthesia with one injection, but is not very effective in cases of sheath violation, such as distal amputation.

IX. Incisions on the hand

A. When planning incisions, avoid crossing flexion creases at a right angle.

1. Midaxial incisions: With the finger fully flexed, mark the radial and ulnar extent of the flexion crease at each finger joint.
2. Bruner incisions: Flexor surface incisions that zigzag from midaxial line to midaxial line.

B. On the extensor surface, longitudinal, curvilinear, "gentle-S," and transverse incisions are all usually acceptable.

Pearls

1. Do not forget that hand trauma patients are also *trauma* patients.
2. When unsure of anatomy, physiology, or pathology, compare the injured hand with the contralateral (uninjured) one—most people are symmetric.
3. Always document a neurovascular examination *before* injecting local anesthetic.
4. Always test a tendon or muscle against *resistance*—you may detect a partial injury that is otherwise compensated.
5. Always incorporate a "reminder" with finger tourniquets, such as a hemostat.

36

Fractures and Dislocations of the Hand and Wrist

John C. Austin

General Principles

I. **Classification of fractures:** Universal descriptive system
   A. **Open versus closed**
   B. **Orientation** (transverse, oblique, spiral, or comminuted)
   C. **Location** (base, shaft, neck, head, or condyle)
   D. **Displaced versus nondisplaced**
   E. **Level of comminution**
   F. **Degree of rotation, angulation, or shortening**
II. **Salter-Harris classification of pediatric fractures**
   A. **Type I:** Fracture through physis only
   B. **Type II:** Involves physis and metaphysis
   C. **Type III:** Involves physis and epiphysis
   D. **Type IV:** Fracture extends from metaphysis through physis, into epiphysis
   E. **Type V:** Crush injury to physis
III. **Stability:** Key to the management of fractures and dislocations
   A. **Stable fractures:** Once reduced, are able to resist deforming forces
   B. **Unstable fractures:** Significant tendency to redisplace once reduced
IV. **Treatment:** Best results are achieved by the following.
   A. **Anatomic reduction**
   B. **Appropriate immobilization**
      1. Length of time needed is determined by fracture location and severity.
      2. General guidelines
         a. 2 to 4 weeks for phalangeal and metacarpal fractures
         b. 4 to 6 weeks for distal radius fractures
         c. 8 to 12 weeks or more for scaphoid fractures
   C. **Early mobilization with occupational therapy**
      1. Fingers are generally mobilized early, since they can develop contractures faster.
      2. The wrist generally recovers mobility better than the fingers.
V. **Assessment of healing**
   A. **Serial x-rays** (fracture callus, new bone formation)
   B. **Physical examination** (loss of tenderness to direct palpation at the fracture site)

Fractures

I. ***Phalangeal fractures***
   A. Phalangeal and metacarpal fractures are common (about 10% of all fractures).
   B. ***Evaluation***
      1. Swelling, pain, decreased range of motion (ROM), deformity, and associated injuries.

      2. **Distal phalanx fractures.**
         a. Evaluate for nail bed injury.
         b. Evaluate for mallet finger (flexion deformity of distal interphalangeal joint due to disruption of the extensor mechanism).
      3. **Deformity is based on key deforming forces.**
         a. Middle phalanx fractures.
            (1) Proximal to flexor digitorum superficialis (FDS) insertion: Apex dorsal tendency.
            (2) Distal to FDS insertion: Apex volar tendency.
         b. Proximal phalanx fractures: Apex volar tendency, due to the following.
            (1) Flexion of proximal fragment (interosseous muscles).
            (2) Extension of distal fragment (extensor mechanism via central slip).
      4. **Radiographs:** Anteroposterior (AP), lateral, and oblique x-rays in varying amounts of flexion to isolate affected digit(s).
   C. **Treatment:** Guided by reducibility and stability
      1. **Reducible and stable:** Usually treated closed with buddy taping, splinting, or casting.
      2. **Reducible and unstable:** Require percutaneous pinning or open reduction and internal fixation (ORIF) to maintain reduction.
      3. **Irreducible:** Require ORIF.
      4. **Fixation choices**
         a. K-wires (percutaneous or buried).
         b. Plates and screws (e.g., modular hand system).
         c. Lag screws.
         d. Interosseous wires.
   D. **Complications**
      1. **Tendon adhesions** (to fracture site).
         a. Early ROM minimizes scarring.
         b. Tenolysis has variable success.
      2. **Malunion.**
         a. Rotational deformity can be treated with derotational osteotomy.
         b. Angular deformity can be treated with closing wedge osteotomy.
      3. **Symptomatic hardware.**
         a. Prominent plates or screws can lead to pain and tenosynovitis.
         b. Treatment: Hardware removal after fracture has healed.
      4. **Pin-tract infections.**
II. **Metacarpal fractures**
   A. **Evaluation:** Similar to that for phalangeal fractures
   B. **Metacarpal head fractures**
      1. Undisplaced fractures: Treated with immobilization alone.
      2. Displaced oblique fractures: ORIF with K-wires or small screws.
   C. **Metacarpal neck fractures** (e.g., Boxer's fracture, little finger metacarpal)
      1. Result of a direct blow to metacarpal head; common in little and ring fingers.
      2. Metacarpal head is usually depressed (apex dorsal); closed reduction should be attempted. Acceptable residual deformity postreduction:
         a. Index finger (IF) or middle finger (MF): Less than 10 degrees.
         b. Ring finger (RF): Less than 30 degrees.
         c. Little finger (LF): Less than 40 degrees.
         d. More angular deformity is allowed at little and ring metacarpals due to increased carpometacarpal joint motion relative to index and middle fingers.
         e. The allowable residual deformity should be individualized for each patient. Untreated residual angulation can result in the patient feeling the metacarpal head in the palm with gripping, and result in a loss of strength with power grip.
      3. No rotational deformity is acceptable for any of the metacarpals.
   D. **Metacarpal shaft fractures**
      1. **Transverse fractures:** Can often be treated with closed reduction and splint or cast.

2. **Spiral and long oblique fractures.**
   a. More unstable; prone to shortening and malrotation.
   b. ORIF indicated if:
      (1) Concern regarding shortening/rotation and desire to get moving earlier to prevent stiffness associated with splinting.
      (2) Greater than 3 mm shortening.
      (3) Angulation greater than 10 degrees (index/middle metacarpals).
      (4) Angulation greater than 20 degrees (ring/little metacarpals).
      (5) Multiple fractures.
3. **Fractures with excessive comminution,** open fractures with severe soft tissue injury, and fractures with segmental bone loss may require external fixation.

E. Metacarpal base fractures

1. When central digits are involved (IF, MF, RF), usually immobilization alone is sufficient.
2. Base of thumb metacarpal is less protected.
   a. **Bennett's fracture-dislocation.**
      (1) Oblique intraarticular fracture of the base of the thumb metacarpal.
      (2) Small volar ulnar fragment remains attached to anterior oblique ligament ("volar beak ligament").
      (3) Larger (distal) fragment is displaced proximally and abducted by pull of abductor pollicis longus (APL).
      (4) Usually repaired with closed reduction and percutaneous pinning.
   b. **Rolando's fracture**
      (1) Comminuted intraarticular fracture of the base of the thumb metacarpal.
      (2) Comminution results in T- or Y-shaped fracture lines.
      (3) Usually requires ORIF (i.e., plate and screws).
3. **Base of little finger metacarpal.**
   a. "Baby Bennett's" or reverse Bennett's fracture.
   b. Analogous to Bennett's fracture, but deforming force on main metacarpal fragment is the extensor carpi ulnaris (ECU).

III. Scaphoid fractures

A. Introduction.

1. Most common carpal bone fracture.
2. Common in young men.
3. Scaphoid waist fractures are most frequent.

B. Anatomy

1. Principal blood supply enters the distal pole of the scaphoid.
2. Proximal pole receives blood supply via intraosseous branches from distal pole, leading to slower healing and higher rates of nonunion in proximal pole fractures.

C. Physical examination suggestive of scaphoid pathology

1. **"Anatomic snuffbox" tenderness** (between extensor pollicis longus [EPL] and extensor pollicis brevis [EPB] tendons).
2. **Watson's scaphoid shift test** (palmar pressure at proximal pole while radially deviating the wrist; the scaphoid flexes volarly with radial deviation of wrist) is positive (scaphoid flexion is able to be counteracted with examiner's pressure) in cases of ligamentous injury and/or fracture.

D. Radiographs

1. Standard wrist x-ray series with scaphoid view (posteroanterior wrist in ulnar deviation).
2. Undisplaced scaphoid fractures may take up to 2 weeks to become visible on radiographs (fracture resorption improves visualization).

E. Treatment

1. Splint or cast all suspected cases (mechanism, tenderness) of scaphoid fractures for 1 to 2 weeks, with follow-up exam and x-rays, because many scaphoid fractures are unrecognized on initial x-rays.

2. **Undisplaced waist and distal pole fractures:** Usually stable; treat with short-arm thumb spica cast for 6 to 12 weeks.
3. **Undisplaced proximal pole fractures:** Heal more slowly (12–24 weeks); treat with long-arm thumb spica cast for first 6 weeks, followed by short-arm thumb spica cast. Consider ORIF.
4. **Displaced fractures** (>1 mm displacement or any angulation): Unstable and require ORIF via dorsal or volar approach. Herbert or Acutrak screws (headless, variable, or multipitch screws) are used to compress the fracture; immediate ROM is possible with rigid fixation.

F. Complications

1. **Nonunion**
   a. Defined as failure to heal after 6 months.
   b. Incidence increases for proximal pole fractures and displaced fractures (up to 90%, versus 5%–10% for waist fractures).
   c. Risk factors: Delay in diagnosis, inadequate immobilization and associated ligamentous instability. If left untreated, post-traumatic arthritis and carpal collapse often follow.
   d. Evaluation: Thin-cut computed tomographic (CT) scan to determine exact anatomy.
   e. Treatment: ORIF with bone grafting if articular surfaces are intact and reduction is possible; salvage procedures (proximal row carpectomy, intercarpal arthrodesis) are indicated in advanced cases (carpal collapse, arthritis, etc.).
2. **Malunion**
   a. Occurs when angulated fracture heals without anatomic reduction.
   b. Usually heals with apex dorsal angulation (humpback deformity); this leads to dorsal intercalated segmental instability (DISI), as evidenced by an increased scapholunate angle.
   c. End result: Post-traumatic arthritis, loss of motion, and decreased grip strength.
3. **Post-traumatic arthritis**
   a. Associated with scaphoid nonunion and malunion.
   b. Scapholunate advanced collapse (SLAC), a pattern of post-traumatic arthritis, can result from scaphoid nonunion and malunion.
4. **Avascular necrosis**
   a. Risk is 90% in proximal pole fractures; risk is 30% to 50% in waist fractures.
   b. Evaluation: X-rays reveal sclerosis in the proximal fragment; magnetic resonance imaging (MRI) is the most sensitive and specific test and is indicated when x-rays are equivocal.
   c. Treatment: ORIF with bone grafting in select cases; vascularized bone graft from distal radius has been successful; salvage procedures include proximal pole excision, proximal row carpectomy, and various subtotal and total wrist fusions.

IV. Distal radius fractures

A. General information

1. Most common fracture of the upper extremity.
2. Very heterogeneous group of injuries; wide variety of patients.
3. Management remains controversial and challenging; consistently good clinical outcomes are difficult to obtain.
4. Residual articular incongruity results in post-traumatic arthritis in most patients.
5. Maximum functional recovery may take 6 to 12 months or longer.

B. Anatomy

1. Distal radius has three concave articular surfaces: scaphoid fossa, lunate fossa, and sigmoid notch.
2. Distal radioulnar joint (DRUJ): Sigmoid notch articulates with ulnar head.
3. Triangular fibrocartilage complex (TFCC): Soft tissue structure between the ulna and the carpus; may be injured in distal radius

4. Radius normally carries about 80% of axial load across the wrist; increased radial shortening or dorsal tilt with fractures can lead to greater loads shifted to the ulna.

C. **History**

1. Mechanism.
   a. Most common: Compressive loading on dorsiflexed wrist ("fell on an outstretched hand")
   b. Others: Excessive force placed on flexed wrist, shearing mechanism, or direct blow
2. Osteoporosis is a significant predisposing factor.
3. Degree of comminution is proportional to the energy transferred to the bone.

D. **Physical examination**

1. Wrist tenderness, swelling, deformity.
2. More severe swelling with high-energy injuries.
3. "Silver fork deformity": Dorsal displacement of distal radius (classic Colles' pattern).
4. Skin integrity (abrasions from fall versus open fractures).
5. Neurovascular status.
   a. Median nerve compromise is relatively common.
   b. Reassess after fracture reduction.

E. **Radiographs and imaging**

1. Posteroanterior (PA) and lateral x-rays: Oblique views are often helpful.
2. Scaphoid series (PA wrist with wrist in ulnar deviation and fist clenched): Helps evaluate associated scaphoid fracture and/or scapholunate ligament injury.
3. Radiographic measurements: Assess severity of fracture and evaluate reduction (Fig. 36-1).
   a. Radial inclination: Average is 23 degrees (PA view).
   b. Radial length: Average is 12 mm (PA view).
   c. Volar tilt: Average is 11 degrees (lateral view).
   d. Also assess Gilula's arcs/lines, scapholunate interval, associated ulna fractures (especially ulnar styloid), and DRUJ.
4. Subtle or complex fractures may require other imaging modalities.
   a. CT: Best for evaluating complex or comminuted fractures (e.g., articular depression fractures).
   b. MRI: Best for evaluating soft tissue injuries.

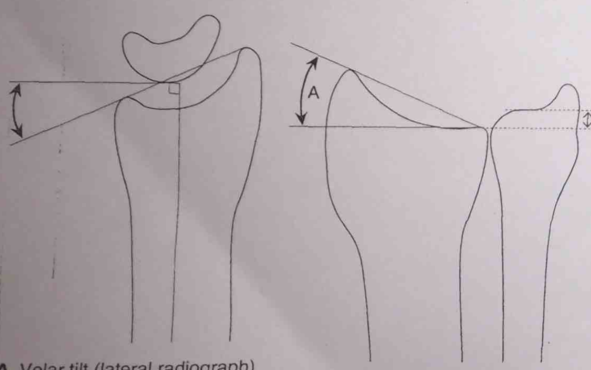

Fig. 36-1. Distal radius, normal angles of inclination. **A:** Lateral view and **B:** PA view.

F. **Fracture description or classification**

1. Numerous classification systems exist (Frykman, Melone, AO/ASIF, Rayhack).
2. None is universally accepted; each has its shortcomings.
3. The descriptive system is most practical (see "General Principles").

G. **Eponyms and named fractures**

1. Historically used, but many fracture patterns do not fit into one specific pattern.
2. **Colles' fracture:** Extraarticular fracture with dorsal comminution, dorsal displacement of distal fragment, and radial shortening.
3. **Smith's (reverse Colles') fracture:** Volar displacement of distal fragment.
4. **Barton's fracture:** Intraarticular fracture (wrist fracture-dislocation); can be volar or dorsal, usually unstable.
5. **Chauffeur's fracture:** Intraarticular fracture of radial styloid.
6. **Die-punch fracture:** Intraarticular depression fracture of lunate fossa.

H. **Associated injuries**

1. **Median nerve compression or neurapraxia.**
   a. Usually improves after reduction.
   b. If not improved after 48 hours, exploration and carpal tunnel release are indicated.
2. **Compartment syndrome:** Fewer than 1% of distal radius fractures (see Chapter 45, "Hand Infections, Compartment Syndrome, and High-Energy Injuries").
3. **TFCC injury:** Associated with fractures at the base of the ulnar styloid.
4. **Ligament injury:** Carpal ligament injury is common (e.g., scapholunate ligament).

I. **Treatment principles**

1. **Goal: Anatomic reduction**
   a. Maximizes function and minimizes the risk of post-traumatic arthritis.
   b. The younger and more active the patient, the more critical it is to achieve anatomic reduction.
2. **Stability**
   a. Stable, reduced fractures: Usually treat nonoperatively.
   b. Unstable and/or irreducible fractures: Usually treat operatively.
      (1) Articular depression greater than 2 mm.
      (2) Radial shortening greater than 5 mm.
      (3) Dorsal tilt greater than 20 degrees.
      (4) Excessive comminution.
3. **Timing**
   a. In most cases, initial reduction and splinting can be performed in the emergency room.
   b. Operative intervention, if required, can be scheduled within 7 to 10 days.
4. **Immobilization**
   a. Initial splint is usually sugar-tong type, to allow for some swelling and to prevent pronation/supination.
   b. May change to a removable splint or short- or long-arm cast, depending on individual patient and fracture pattern.
   c. If uninjured, fingers should be free and patient should work on finger ROM as early as 1 to 2 weeks after injury.
   d. Do not immobilize the shoulder, because of the risk of rotator cuff tendonitis and adhesive capsulitis.
   e. Undisplaced fractures: Usually immobilize for 4 weeks.
   f. Displaced fractures (reduced): Usually immobilize for 6 weeks.
      (1) Less in children.
      (2) Less when stable ORIF performed.
      (3) More for complex fractures.

J. **Treatment algorithm**

1. **Open fractures**
   a. Washout in the operating room is generally required.

b. Thoroughly debride all contamination and nonviable tissues.
c. Controversy exists regarding immediate versus delayed fracture fixation.
d. Consider external fixation.

2. **Closed fractures**
a. Undisplaced fractures: Immobilization alone is usually sufficient.
b. Displaced fractures: Attempt closed reduction (see below).
(1) Adequate reduction.
(a) Stable.
(i) Immobilization may be sufficient.
(ii) Weekly x-rays to check reduction.
(b) Unstable: Operative fixation required.
(2) Inadequate reduction: Operative fixation required.

K. Closed reduction
1. Relies on ligamentotaxis to restore anatomic alignment
2. Procedure
a. Hang hand from finger traps with shoulder abducted 90 degrees and elbow flexed 90 degrees.
b. Anesthesia: Conscious sedation and/or hematoma block (plain lidocaine injected into fracture hematoma site).
c. Longitudinal traction is provided by weights (usually 10–20 pounds) hung from stockinette just proximal to elbow.
d. Fracture reduction maneuvers.
(1) Re-creation of injury pattern (e.g., wrist hyperextension for Colles' fractures) under continuous longitudinal traction.
(2) Followed by reduction move (e.g., palmar translating force for Colles' fractures) distal to fracture site.
e. C-arm guidance may be used to assess adequacy of reduction.
f. Apply sugar-tong splint and obtain postreduction x-rays.
3. Guidelines for adequate closed reduction
a. Articular incongruity less than 2 mm.
b. Radial shortening less than 5 mm.
c. Dorsal tilt less than 10 degrees.

L. Operative reduction and fixation
1. **Closed reduction with percutaneous pinning (CRPP)**
a. Generally for unstable and/or irreducible extraarticular fractures.
b. Kapandji technique (intrafocal pinning).
(1) Heavy K-wire is directed through the fracture line dorsally or radially.
(2) Use K-wire as lever arm to reduce fracture.
(3) Drive K-wire into opposite cortex for stabilization.
2. **Open reduction and internal fixation**
a. Many types of implants are available: K-wires, screws, intraosseous wiring, plates and screws, and manufacturer-specific fracture fixation systems.
b. Limited approaches are preferred for simple, intraarticular fracture patterns such as radial styloid fractures and articular margin fractures.
c. Complex articular fractures require more complete exposure and/or multiple approaches.
d. Surgical approaches
(1) Volar: Between flexor carpi radialis (FCR) and radial artery; indicated for volarly displaced distal radius fractures; allows for concomitant carpal tunnel release.
(2) Dorsal: Through third dorsal extensor compartment; indicated for complex intraarticular fractures.
(3) Dorsal radial: Used for displaced radial styloid fractures.
3. **External fixation**
a. Indicated for unstable fractures with excessive comminution.
b. Maintains length of the distal radius while healing occurs.

c. Avoid overdistraction: Can lead to finger stiffness and fracture nonunion or delayed union.
d. Avoid leaving wrist in flexion: Can lead to median nerve compression.
e. Place pins with open technique to avoid eccentric pin placement and injury to radial sensory nerve.
4. **Arthroscopy**
a. Can be used to confirm reduction (i.e., check articular surface).
b. Can be used to diagnose and treat ligamentous tears, TFCC tears.
5. **Bone grafting**
a. Indicated when articular depression, bone loss, and extensive comminution are present.
b. Example: Die-punch fracture—the articular fragment is elevated, and bone graft is placed proximally in the created metaphyseal defect.
c. Bone graft source is often allograft or bone substitute materials (nonunion is rare in distal radius fractures).
6. **Combinations of** the previously listed options: Complex fracture patterns frequently require combinations of the above (such as ORIF, external fixation, and bone grafting).

M. Complications
1. **Malunion:** Relatively common (e.g., dorsal tilt and loss of radial length can lead to ulnocarpal impaction, DRUJ incongruity, pain, weakness, and decreased ROM).
2. **Nonunion:** Rare except in cases of overdistraction during external fixation.
3. **Tendon rupture:** Usually EPL (a late complication).
4. **Post-traumatic arthritis.**
5. **Complex regional pain syndromes** (CRPS).

Dislocations and Ligamentous Injuries

I. Phalangeal dislocations
A. Distal interphalangeal (DIP) joint dislocations
1. **Anatomy and mechanism**
a. Usually dorsal dislocation; due to hyperextension injury of the DIP joint.
b. About 60% are associated with a volar laceration.
c. Often involve disruption of both collateral ligaments and the volar plate.
2. **Treatment**
a. **Simple (reducible)**
(1) Closed reduction under metacarpal block: Extend distal phalanx, then push dorsally over condyles of middle phalanx.
(2) Check x-rays to confirm joint congruity and rule out associated fracture; splint for 2 weeks.
b. **Complex (irreducible)**
(1) Open reduction is required.
(2) Possible causes include interposition of the profundus tendon (implies disruption of at least one collateral ligament), volar plate, or displaced articular fracture fragment.
3. **Complications:** Recurrent instability, post-traumatic arthritis

B. Proximal interphalangeal (PIP) joint dislocations
1. **Anatomy and mechanism**
a. PIP joint is a hinged joint with articular congruity between the two condyles of the proximal phalanx and two concave surfaces of the middle phalanx.
b. Strong volar plate and collateral ligaments provide additional stability.
2. **Dorsal dislocation**
a. More common than volar dislocation.
b. Obvious deformity with dorsal location of middle phalanx.

c. Radiographs may reveal an avulsion fracture from the volar surface of the middle phalanx (distal volar plate insertion site).
d. Treatment.
(1) Closed reduction under metacarpal block with longitudinal traction.
(2) Stable reductions are treated with extension block splinting or buddy taping for 3 weeks and early ROM.
(3) Unstable reductions are treated with extension block splinting in 20 degrees of flexion for about 3 weeks.
e. Rotational deformity of the finger may suggest displacement of the middle phalanx between the central slip and lateral band.

3. Volar dislocation
a. Lack of active extension of middle phalanx against gentle resistance indicates central slip rupture.
b. Radiographs may reveal avulsion fracture from dorsum of middle phalanx (central slip insertion).
c. Treatment
(1) Closed reduction with longitudinal traction and flexion at PIP joint.
(2) After reduction, must assess PIP joint extension.
(a) If central slip intact: Immobilization with buddy taping and early ROM.
(b) If central slip disrupted: Treat as per central slip rupture (see Chapter 37, "Tendon Injuries and Tendonitis").
d. Complications: Progressive boutonnière deformity, PIP joint stiffness.

4. PIP joint fracture-dislocations
a. Can be quite disabling.
b. Radiographs are essential. Injuries range from a small articular fragment to pieces involving more than 50% of joint surface.
c. Treatment.
(1) Stable PIP joint with less than 30% articular surface involved: Dorsal extension block splinting.
(2) Unstable PIP joint and/or more than 30% of articular surface involved: ORIF.
(3) Comminuted fractures: Volar plate arthroplasty (volar fragment excision and advancement of volar plate distally on middle phalanx).
d. Complications: Persistent instability; joint stiffness.

C. Metacarpophalangeal (MP) joint dislocations
1. **Less common** than PIP joint dislocations
2. Dorsal dislocations are more common; usually involve the index or little fingers.
3. **Evaluation.**
a. **Simple (reducible):** Notable deformity with marked MP joint hyperextension.
b. **Complex (irreducible).**
(1) Deformity not as obvious; MP joint only slightly hyperextended.
(2) Cannot be reduced, due to interposition of the volar plate ("buttonhole effect"; look for puckering of volar skin and/or presence of a sesamoid bone in widened MP joint) or trapping of metacarpal head between lumbrical (radially) and flexor tendon (ulnarly).
4. **Treatment.**
a. **Simple dislocations.**
(1) Closed reduction via gentle hyperextension at MP joint followed by relocating proximal phalanx onto metacarpal head.
(2) After reduction, splinting with MP joints in 50 degrees of flexion for 7 to 10 days followed by buddy taping. ORIF is recommended if associated joint subluxation and/or intraarticular fracture involving more than 20% of joint surface is present.

b. **Complex dislocations:** Usually require open reduction (via dorsal or volar approach).

II. Thumb dislocations

A. Thumb MP joint dislocation
1. Multiaxial diarthrodial joint: Allows for flexion, extension, limited adduction, abduction, and circumduction.
2. Stability is critical for grip and pinch.
3. **Dorsal dislocation.**
a. Simple: Closed reduction and thumb spica splint immobilization.
b. Complex: Irreducible; usually due to volar plate or FPL interposition; requires operative reduction and soft tissue repair followed by splinting.
4. **Volar dislocation:** Rare.

B. Thumb ulnar collateral ligament (UCL) injuries
1. Injury produced by forceful radial deviation of thumb.
a. Acute rupture: "Skier's thumb".
b. Chronic attenuation: "Gamekeeper's thumb".
2. Physical examination and treatment.
a. Tenderness over ulnar collateral ligament; ligamentous laxity should always be compared with the contralateral (healthy) thumb.
b. Partial tear: Opens less than 45 degrees with radial stress; requires thumb spica splinting with thumb MP joint held in slight flexion for 4 to 6 weeks.
c. Complete tear: Opens more than 45 degrees with radial stress.
(1) Can be associated with a (palpable) Stener lesion (interposition of adductor pollicis aponeurosis between torn end of UCL and its insertion at the base of proximal phalanx).
(2) Requires operative repair with suture anchor or pullout button.

C. Thumb radial collateral ligament (RCL) injuries
1. Rare and less debilitating than ulnar collateral ligament injuries.
2. Treated nonoperatively with splint or cast.

III. Carpal dislocations and ligamentous injuries

A. Anatomy
1. **There are seven carpal bones** (excluding pisiform, a sesamoid bone within FCU tendon).
2. **Row theory** (traditional wrist model) divides carpal bones into two rows.
a. Proximal row: Scaphoid, lunate, triquetrum.
b. Distal row: Trapezium, trapezoid, capitate, hamate.
3. **Wrist ligaments.**
a. Intrinsic ligaments.
(1) Connect carpal bones within a carpal row.
(2) Most important are scapholunate (SL) and lunotriquetral (LT) ligaments.
b. Extrinsic ligaments
(1) Connect bones between carpal rows (spans midcarpal joint).
(2) Volar extrinsic ligaments are stronger than dorsal extrinsic ligaments.
4. **Kinematics.**
a. Wrist motion is complex and occurs primarily at radiocarpal and midcarpal interface.
b. Proximal carpal row flexes with radial deviation of wrist and extends with ulnar deviation. This function is impaired with SL and LT ligament disruptions.

B. Injury types
1. No single classification system effectively describes all of the various patterns of injury.
2. **Perilunate injury.**
a. Occurs in stages as ligaments sequentially fail around the lunate.
(1) Stage I: Scapholunate ligament tear.
(2) Stage II: Capitolunate ligament tear.

(3) Stage III: Lunotriquetral ligament tear (dorsal perilunate dislocation).
(4) Stage IV: Dorsal radiolunate ligament tear (volar lunate dislocation).

b. Note that **dorsal** perilunate dislocation (stage III) is caused by the same injury pattern as **volar** lunate dislocation (stage IV).

3. Greater and lesser arc injuries.

a. Perilunate carpal disruptions can be purely ligamentous or can have associated carpal fractures.

b. Greater arc injury (involves carpal fracture).
(1) Combines carpal bone fracture with perilunate dislocation.
(2) Fractures occur because injury pattern involves an arc of greater radius around lunate that passes through surrounding osseous structures.
(3) Transscaphoid perilunate fracture-dislocation
(a) Combines scaphoid fracture with perilunate dislocation.
(b) Most common type of greater arc injury.
(c) Immediate treatment: Closed reduction and splinting to minimize damage to neurovascular structures.
(d) Definitive treatment.
(i) Volar approach: Allows for scaphoid fracture ORIF with screw or K-wires; repairs volar ligament injury.
(ii) Dorsal approach: Assists with anatomic reduction of carpal bones (restores lunate in its fossa in distal radius and capitate in fossa in distal lunate articulation).
(4) Transradial styloid perilunate fracture-dislocation.
(a) Combined radial styloid fracture with perilunate dislocation.
(b) Immediate treatment: Closed reduction and splinting as in above injuries, with adequate reduction of radial styloid fragment.
(c) Definitive treatment.
(i) ORIF of radial styloid fracture.
(ii) Reduction, soft tissue repair, and K-wire fixation of perilunate injury are then performed as in isolated perilunate dislocations.

c. Lesser arc injury (purely ligamentous injury).
(1) No fractures because pattern of injury is via an arc of smaller radius through ligaments immediately adjacent to lunate.
(2) Most common injury patterns are dorsal perilunate (lunate remains in its fossa in distal radius with dorsal dislocation of capitate and rest of carpus) and volar lunate (lunate displaced from fossa of distal radius) dislocations.
(3) Perilunate dislocation (dorsal perilunate and volar lunate dislocations).
(a) Immediate treatment.
(i) Closed reduction and splinting to minimize damage to neurovascular structures.
(ii) Open reduction is often required in volar lunate dislocations.
(b) Definitive treatment.
(i) ORIF via combined dorsal (between third and fourth compartments) and volar (via carpal canal) approaches.
(ii) Repair volar ligament injury.
(iii) K-wires secure scaphoid, lunate, and capitate in their respective anatomic positions.
(iv) Pins remain in place for 8 to 12 weeks.

4. Axial (longitudinal) carpal instability.

a. Rare, high-energy injuries associated with significant soft tissue disruption.

b. Direction of disruption is in longitudinal plane that is perpendicular to plane of perilunate injury.

c. Radial axial injuries: Involve separation of first and second metacarpals and trapezium/trapezoid from remaining ulnar part of hand and carpus.

d. Ulnar axial injuries: Involve separation between third and fourth metacarpals and capitate/hamate.

e. Treatment: ORIF via dorsal approach.

C. Instability patterns

1. **Carpal instability dissociative (CID):** Includes intrinsic ligament disruptions that occur within a carpal row.

a. **Dorsal intercalated segmental instability** (DISI; rotatory subluxation of the scaphoid).
(1) Scaphoid and lunate dissociated due to scapholunate ligament disruption or scaphoid fracture.
(2) Lunate rotated dorsally via the LT ligament as the counterbalancing force of the SL ligament is lost.
(3) Physical examination: Watson test.
(a) Identifies SL ligament disruption.
(b) Palpable clunk present with thumb pressure applied dorsally over volar scaphoid tubercle during radial and ulnar wrist deviation.
(4) Radiographs.
(a) Lateral wrist x-ray.
(i) Increased dorsal tilt of lunate (radiolunate angle >15 degrees).
(ii) Increased scapholunate angle (>60 degrees).
(b) PA x-ray.
(i) Widened scapholunate interval (>3 mm) or scaphoid fracture.
(ii) Cortical ring sign ("double density" appearance of shortened scaphoid) occurs due to abnormal scaphoid rotation.
(5) Early diagnosis and treatment of SL ligament rupture can prevent DISI deformity and associated carpal arthritis (SLAC wrist).
(6) Treatment of acute SL injuries.
(a) ORIF with direct SL ligament repair.
(b) Dorsal approach between third and fourth dorsal compartments.
(c) K-wires passed from scaphoid to lunate and capitate.
(d) Suture anchors: Allow for anatomic repair of ligament to bone when possible.
(e) Blatt capsulodesis: Repair augmented with dorsal capsule if SL ligament attenuated.
(f) Pins removed after 8 to 12 weeks.
(7) Treatment of chronic SL injuries (>6–8 weeks old).
(a) No associated arthritis: Treat as acute injury with Blatt capsulodesis.
(b) Associated arthritis present or irreducible dislocation: Treat with limited arthrodesis (scaphotrapezial-trapezoid fusion; scapholunate fusion; scaphocapitate fusion).
(8) Scapholunate advanced collapse (SLAC wrist).
(a) Systematic progression of intercarpal degenerative arthritis following chronic SL ligament rupture or scaphoid fracture nonunion with collapse.
(b) Four stages.
(i) Stage I: Stylocarpal arthritis.
(ii) Stage II: Scaphoid fossa involvement.
(iii) Stage III: Capitolunate arthritis.
(iv) Stage IV: Diffuse carpal arthritis.
(c) Treatment.
(i) Scaphoid excision with four-corner fusion.

(ii) Other options: Proximal row carpectomy, complete wrist fusion, and wrist arthroplasty.

b. Volar intercalated segmental instability (VISI).

(1) Much less common and not understood as well as DISI.

(2) Thought to be due to disruption of both LT ligament and dorsal radiocarpal ligaments.

(3) Lunate is rotated volarly via forces applied through SL ligament alone (because the LT ligament is disrupted).

(4) Physical examination: Shuck test.

(a) Identifies LT ligament disruption.

(b) Triquetrum and lunate shifted volarly and dorsally to elicit instability or discomfort.

(5) Radiographs: Lateral wrist x-ray reveals decreased SL angle (<30 degrees) and increased radiolunate angle (>15 degrees).

(6) Treatment.

(a) Isolated LT ligament tears: Nonoperative management with nonsteroidal anti-inflammatory agents and splinting.

(b) LT tears associated with VISI deformity.

(i) Management controversial

(ii) Options include soft tissue reconstruction with dorsal capsule or tendon autograft or arthrodesis (such as four-corner fusion).

2. Carpal instability nondissociative (CIND).

a. Extrinsic ligament disruption between carpal rows.

b. Midcarpal instability.

(1) Frequently insidious onset.

(2) Common in patients with increased ligamentous laxity.

(3) Sudden painful clunk often present when shift occurs between the two carpal rows.

(4) Diagnosis.

(a) Physical examination (palpable clunk).

(b) Stress radiographs.

(c) Fluoroscopic examination under anesthesia.

(5) Treatment.

(a) Soft tissue reconstruction (if no associated arthritis).

(b) Midcarpal arthrodesis (in chronic cases with associated arthritis).

3. Carpal instability complex (CIC).

a. Combines CID and CIND (both intrinsic and extrinsic ligament disruption patterns are present).

b. Perilunate dislocations.

4. Static versus dynamic instability.

a. Static instability patterns.

(1) Fixed.

(2) Clearly seen on routine radiographs.

b. Dynamic instability patterns.

(1) Transient.

(2) Not present on routine radiographs.

(3) Require stress x-rays or fluoroscopy for diagnosis.

(4) Example: Clenched-fist PA view to identify dynamic SL ligament instability.

D. Acute versus chronic injury.

1. Acute injury.

a. Readily identifiable injury with associated pain and swelling.

b. Complete neurovascular evaluation important.

2. Chronic injury.

a. Injury patterns more subtle than acute injuries both clinically and radiographically.

b. Additional studies often needed.

(1) Wrist arthrography, fluoroscopy, and MRI have been used historically.

(2) Wrist arthroscopy.

(a) Becoming more popular as the most complete and direct means of carpal ligament evaluation.

(b) Also provides for examination of carpal bones (osteochondral defects, arthritis assessment) and synovium (hypertrophy, inflammation).

Pearls

1. Use the universal descriptive system for all fractures.
2. The choice of fixation must maximize rigidity while minimizing exposure (periosteal stripping).
3. Sometimes a more invasive approach to fixation allows earlier mobilization and a better result overall.
4. In the adult patient, interphalangeal joint stiffness should be avoided at all costs—get the fingers moving as soon as possible!
5. Recovery from a wrist fracture can take 6 to 12 months or longer; prepare patients for the long haul.

37 Tendon Injuries and Tendonitis

Michelle S. Caird

Tendon Blood Supply and Healing

I. **Anatomy and general considerations**
   A. **Definition:** Strong, dense connective tissues that attach muscle to bone.
   B. **Tendon makeup:** Type 1 collagen → longitudinal bundles → fibrils → fascicles → tendons.
   C. **Paratenon-covered tendons and synovial tendons** have different blood supply and methods of healing.
   D. **Three phases of healing**
      1. **Inflammatory (first week):** Cell proliferation and cleanup.
      2. **Proliferative (weeks 2–4):** Fibroblasts and capillary buds migrate in and produce random collagen.
      3. **Remodeling (months 2–6):** Longitudinal organization of collagen fibers in line with stress.

II. **Tendons with paratenon**
   A. **In general, tendons that move in straight lines** (no sharp turns) have paratenon (e.g., extensor carpi radialis longus [ECRL] and extensor carpi radialis brevis [ECRB]).
   B. **Paratenon** is composed of loose connective tissue, continuous with the tendon surface.
   C. **Blood supply emanates** from vessels in the perimysium, at the bony insertion, and at **many points** along the course of the tendon, forming a longitudinal anastomotic capillary system.
   D. **Healing from paratenon:** Fibroblasts and capillary buds migrate into the injured area.

III. **Tendons with synovial sheath**
   A. **In general, tendons that take sharp turns around joints (e.g., flexor tendons)** have synovial sheaths.
   B. **The tendon sheath** surrounds the tendon and produces synovial fluid for low-friction gliding.
   C. **Blood supply emanates** from vessels in the perimysium, at the bony insertion, and through mesotendon conduits (vincula) at discrete points along the tendon.
   D. **Areas of relative avascularity** exist along the tendon, which receive nutrition by synovial diffusion.
   E. **Healing (controversial)**
      1. **Extrinsic:** Fibroblasts migrate from the sheath into the injured site (also form adhesions).
      2. **Intrinsic:** Tendon cells can migrate across closely approximated ends and heal with nutrients from synovial fluid.

Timing of Repair

I. **Acute and subacute repair**

   A. **If the condition of the soft tissues allows,** primary tendon repair should be performed on an urgent basis (within several days).
   B. **After 1 week:** Increased risk of adhesions with primary repair.
   C. **After 3 weeks:** Muscle contraction interferes with primary repair.

II. **Delayed reconstruction**
   A. **Flexor tendon injuries**
      1. **One-stage reconstruction with a tendon graft** is used *only* if the flexor sheath is intact and full passive motion is present.
      2. **Two-stage reconstruction is preferred** if the above conditions are not met and after soft tissues are quiet (3–4 weeks after injury).
         a. First stage: Temporary silicone implant (Hunter rod) to create a bed for the graft.
         b. Second stage: Wait at least 2 to 3 months, then exchange the rod for a tendon graft (palmaris longus, extensor digitorum longus, or plantaris).
   B. **Extensor tendon injuries**
      1. **Frequently need staged reconstruction** after degloving injuries to the hand.
      2. **Two-stage reconstruction**
         a. First stage: Obtain adequate skin coverage with or without placement of silicone implants.
         b. Second stage: Wait about 3 months, and then perform tendon graft.

III. **Tenolysis**
   A. **Indicated if discrepancy exists between active and passive range of motion (ROM).**
   B. **Timing**
      1. **After maximized therapy** for full passive motion of all joints.
      2. **After soft tissues achieve equilibrium** and tendon is healed.
   C. **In flexors,** preserve A2 and A4 pulleys to prevent bowstringing.

Tendon Suture Techniques

I. **General considerations**
   A. **Atraumatic handling of tendon ends,** synthetic braided nonabsorbable suture, and end-to-end repair.
   B. **Sheath repair is controversial.** The theory is that closure improves synovial nutrition for healing, but this has not been proven.
   C. **Tensile strength–time relationship**
      1. **Weakest 7 to 10 days after repair.**
         a. Postoperative day 10 is the most common time for rupture after primary repair.
         b. Treat with prompt exploration and repair.
      2. **Most strength has returned 4 to 6 weeks after repair.**
      3. **Maximum strength 6 months after repair.**

II. **Core suture** (Fig. 37-1)
   A. **Strength of repair** is proportional to the number of core strands crossing the repair.
   B. **There are a number of ways to suture a tendon,** but all have common features.
      1. **Small grasping stitches avoid pullout.**
      2. **Knots buried in the repair site assist in smooth gliding.**
   C. **Most common:** Modified Kessler stitch.

III. **Epitendinous suture**
   A. **Consists of a running stitch** along the edges of the tendon repair after the core sutures are placed.
   B. **Leads to decreased gap formation,** which is the first step toward failure.
   C. **Smoothes edge for gliding.**
   D. **Decreases adhesion formation**

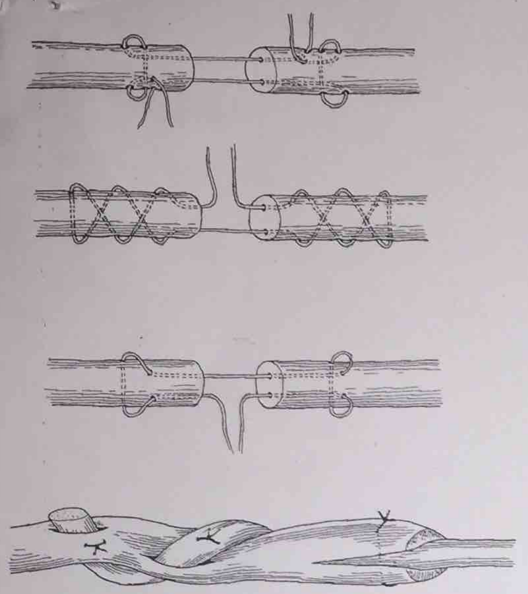

Fig. 37-1. Flexor tendon suturing techniques. **A:** Kessler suture. **B:** Bunnell suture. **C:** Modified Kessler suture. **D:** Pulvertaft weave suture results in a very strong juncture, but is not suitable for use within the flexor tendon sheath due to its bulk. (From Zidel P. Tendon healing and flexor tendon surgery. In *Grabb and Smith's Plastic Surgery*, 5th ed. Aston SJ, Beasley RW, Thorne CH, (eds). Philadelphia, Lippincott-Raven, 1997. With permission.)

Therapy Protocols

I. General considerations

- **A. Early controlled motion** prevents adhesions and improves healing and strength of tendon repairs.
- **B. Requires a cooperative patient** and close interaction with physician and hand therapist.

II. Splinting

- **A. Flexor tendons:** In general, wrist and metacarpophalangeal (MP) joints flexed; interphalangeal (IP) joints extended. Allows passive flexion of IP joints, if wrist flexed.
- **B. Extensor tendons:** Extension splinting of involved joints.
- **C. Small children or noncompliant patients** may need above elbow splint of cast to prevent removal.

III. Therapy

- **A. Tendon therapy** is a whole practice unto itself.
- **B. Flexor tendon therapy.**
  1. **Combine dynamic splints** with particular protocols.

  2. **Active extension** with rubber band flexion (Kleinert and modifications), controlled passive motion (Durand and similar), and controlled active motion protocols all have their advocates.
- **C. Extensor tendon therapy:** A variety of early controlled motion protocols are available, depending on the injury.

Extensor Tendon Injuries

I. Management by zones

- **A. General considerations.**
  1. **The dorsum of the hand and forearm** are divided into nine zones, which guide management and predict outcome.
  2. **Odd zones are over each joint;** even zones are in between.
  3. **Multiple attachments limit retraction** of extensors and allow emergency room (ER) repair of many injuries.
- **B. Zone I:** Over the distal interphalangeal (DIP) joint; commonly called mallet finger and classified into four types.
  1. **Type I:** Rupture at insertion. Treat with extension splinting of DIP joint for 6 weeks (Stack splint).
  2. **Type II:** Laceration. Treat with repair and extension splinting. Repair can be performed as a single layer with the skin, using nonabsorbable (nylon) sutures.
  3. **Type III:** Abrasion with tendon loss and overlying soft tissue injury. Treat with coverage and graft or joint fusion.
  4. **Type IV:** Avulsion fracture (bony mallet). Treat with extension splinting if fragment reduces or with internal fixation if joint is subluxed.
- **C. Zone II:** Over the middle phalanx. Assess active and passive extension.
  1. **If active extension is intact** and tendon ends do not move, use simple splinting.
  2. **If full, active extension is not intact** or ends separate, repair.
- **D. Zone III:** Over the proximal interphalangeal (PIP) joint: central slip injury.
  1. **Laceration:** Treat with repair and extension splinting (of PIP only; leave DIP joint free to flex, as this action draws lateral bands and central slip distally, taking pressure off the central slip repair).
  2. **Avulsion:** Commonly from jammed finger. Treat with extension splinting (of PIP only).
  3. **If injury is missed, lateral bands** can sublux volarly and result in a boutonnière deformity.
- **E. Zone IV (and zone II of the thumb):** Over the proximal phalanx. Treat like a zone II injury.
- **F. Zone V (and zone III of the thumb):** Over the MP joint, at extensor tendon or sagittal bands.
  1. **Simple laceration:** Explore and repair tendon or sagittal band.
  2. **Beware of the "fight bite" or human bite wound** with injury in this location (suspect even if not the history given).
     - a. Acute: Explore, repair tendon, give antibiotics, and leave skin open.
     - b. Old/chronic: May require delayed treatment of tendon, until infection is controlled and soft tissue equilibrium is reached.
     - c. The lacerations of the skin, tendon, and joint may not line up directly in the position in which the wound is explored (compared with the position in which the injury occurred): Be wary of deeper penetration.
     - d. Suspect and rule out joint involvement.
- **G. Zone VI (and zone IV of the thumb):** Over the metacarpals. If tendon ends are easily retrievable, repair in ER (separate layer to skin repair, using nonabsorbable braided suture—horizontal mattress is acceptable); otherwise, repair in operating room.

H. **Zone VII:** Beneath the extensor retinaculum. Repair tendon with or without partial retinacular excision to prevent adhesions.
I. **Zone VIII:** Over the distal forearm. Treat as zone VI. May require tendon transfers if function lost.
J. **Zone IX:** Muscles of proximal forearm. Difficult to repair: Use large bites of large braided nonabsorbable sutures. May require transfers if function lost.

II. **Outcomes and complications**
A. **With early treatment, most do fairly well.**
B. **Worse outcome is associated with the following.**
1. Distal injuries (zones I–IV).
2. Infection.
3. Associated bony and soft tissue injuries.
C. **Adhesions and extrinsic tightness:** Treat with therapy, followed by tenolysis versus tendon release.
D. **Missed injuries or late presenting injuries**
1. **Fight bite:** Septic arthritis and/or cellulites. Needs aggressive irrigation and debridement. Can result in amputation.
2. **Central slip injuries:** Can result in boutonnière deformity.
3. **Sagittal band injury:** Subluxation of extensor tendon ulnarly (most common) or radially (rare).

Flexor Tendon Injuries

I. **Pulley system**
A. **General considerations**
1. **Pulleys are thickenings along flexor sheaths,** lined with synovium.
2. **They improve biomechanics of flexor tendons** by preventing bowstringing of tendons during flexion.
B. **Finger: Annular (five) and cruciate (three) pulleys** (see Fig. 35-5)
1. **Annular pulleys**
a. A1 at MP joint, A2 over proximal phalanx, A3 at PIP joint, A4 over middle phalanx, A5 at DIP joint.
b. A2 and A4 are the most important biomechanically to prevent bowstringing.
2. **Cruciate pulleys** are between the annular pulleys; they are thinner and less biomechanically important than annular pulleys.
C. **Thumb:** Annular (two) and oblique (one) pulleys
1. **Annular pulleys:** A1 at MP joint and A2 at IP joint.
2. **Oblique pulley:** Extension of adductor pollicis attachment. Lies between A1 and A2. Most important pulley to prevent bowstringing in thumb.

II. **Management by zones** (see Fig. 35-3)
A. **General considerations**
1. **Hand and wrist** are divided into five zones, which guide management and help predict outcome.
2. **The small number of attachments allows for proximal retraction** of lacerated tendons and therefore usually requires exploration and repair in the OR.
3. **Associated neurovascular injury is common,** so a detailed examination is paramount prior to local anesthetic infiltration.
B. **Zone I:** Middle of middle phalanx to fingertip (distal to insertion of flexor digitorum superficialis [FDS]; includes only flexor digitorum profundus [FDP]).
1. **Laceration**
a. Treat with direct repair.
b. Avoid advancement, which can result in the **quadrigia effect:** incomplete flexion of FDPs of uninjured fingers when the shortened FDP reaches maximum flexion too early.

2. **Profundus avulsion (jersey finger):** Classification and treatment are based on the level of retraction of the tendon end.
a. Type I: Into palm. Vincula are disrupted. Requires early treatment.
b. Type II: Retracts to level of PIP joint because the long vinculum remains intact. Treatment can be performed up to a few months out.
c. Type III: Retracts to A4 pulley (middle of middle phalanx) because a bony avulsion fragment gets stuck on the pulley. Avulsion fragment should be fixed back into position.
C. **Zone II:** Distal palmar crease to middle of middle phalanx (both FDS and FDP tendons, with FDP volar, proceeding distally).
1. **Both tendons should be repaired.**
2. **Tendon lacerations** may not match level of skin laceration or each other, depending on the joint positions at the time of injury.
3. **Formerly known as "no man's land"** due to high risk of adhesions and poor rehabilitation outcomes.
D. **Zone III:** Transverse carpal ligament to distal palmar crease.
1. **No sheath here;** outcome is generally better.
2. **Associated injury to the common digital nerves** and superficial palmar arch is common.
E. **Zone IV:** Under transverse carpal ligament (in carpal tunnel).
1. **Treat with operative repair** with or without lengthening of transverse carpal ligament.
2. **Examine median nerve closely for injury.**
F. **Zone V:** Proximal border of transverse carpal ligament to musculotendinous junction. Generally has the best outcomes.

III. **Partial injuries**
A. **Full ROM is present,** but patient has weakness or pain with resisted flexion.
B. **Should be explored.**
1. **Repair if laceration is greater than 60%,** due to risk of late rupture.
2. **If less than 60%,** laceration should be left alone because the repair may impede blood supply or create adhesions, or both.
C. **If partial injuries are missed, or fail to present,** late rupture, adhesions, or triggering can result.

Trigger Finger and Thumb

I. **Definition and epidemiology**
A. **Stenosing tenosynovitis of flexor tendons.**
B. **A nodule forms in the flexor tendon,** creating a size mismatch between the tendon and the annular pulley.
C. **A1 pulley**
1. **The most common site.**
2. **Proximal edge** is located at the distal palmar crease in the little and ring fingers, between the proximal and distal creases in the middle finger, and at the proximal crease in the index finger and thumb.
D. **Middle-aged women** are the most commonly affected group.
E. **Increased incidence is seen with diabetes, rheumatoid arthritis, gout, and amyloidosis.**

II. **Complaints and examination findings**
A. **Catching, sticking, or occasional locking of fingers in flexed position.**
B. **Pain in distal palm,** or commonly referred to as the PIP joint.
C. **Pain and sticking are often worse on awakening.**
D. **More common in the thumb, long, and ring fingers.**
E. **Slight tenderness volarly over the tendon at the A1 pulley.** Nodule is palpable on tendon with movement. Popping or clicking on flexion/extension, as nodule moves through pulley.

III. **Treatment**

A. Injection
 1. **Most successful for recently acquired cases of triggering.**
 2. **Inject steroid plus local anesthetic** (50% Kenalog-40, 50% plain 1% lidocaine) into tendon sheath at the level of the A1 pulley (locate as above).
 3. **Use a small needle.** Insert down to bone; back out slowly, with pressure on plunger, stopping when solution flows easily into tendon sheath. A fluid wave can be felt down the finger in most cases.
 4. **Remind diabetics** that steroids will affect their blood sugar levels.

B. Splinting
 1. **Low success rate.**
 2. **Extension splint of affected digits** (especially at nighttime if there are morning symptoms).
 3. **Alone or with steroid injections.**

C. Surgery
 1. **Indications.**
  a. Long-standing triggering
  b. Persistent triggering that fails injection and splinting
 2. **Procedure:** Small transverse or Bruner-type incision at proximal edge of pulley (see above). Division of A1 pulley under direct vision.
 3. **Watch and protect the digital neurovascular bundles,** especially at the thumb where the radial bundle courses directly over the A1 pulley.
 4. **Release the entire pulley and have (awake) patient actively test in OR.**

Tenosynovitis

I. De Quervain's tenosynovitis

A. **Definition: Stenosing tenosynovitis** of the tendons in the first dorsal compartment of the wrist (abductor pollicis longus [APL] and extensor pollicis brevis [EPB]).

B. **Most common in middle-aged women.**

C. **Symptoms and signs.**
 1. **Radial-sided wrist pain** with thumb use.
 2. **Positive Finkelstein's test:** Pain with thumb tuck and ulnar deviation of wrist.
 3. **Tenderness over first dorsal compartment.**
 4. **No pain with axial grind test,** which is positive in first carpometacarpal joint arthritis.

D. Treatment
 1. **Injection with steroid** plus lidocaine into first dorsal compartment.
  a. Inject at distal end of compartment.
  b. Often fails due to multiple slips of APL, each in their own subcompartment.
 2. **Forearm-based thumb spica splinting.**
 3. **Surgery:** Release the first dorsal compartment and perform tenosynovectomy.
  a. Release of all subcompartments must be confirmed: As above, APL can have multiple slips, and fool the surgeon into thinking both APL and EPB have been released.
  b. Superficial radial nerve should be avoided during dissection to prevent paresthesia or painful neuroma formation.

II. Intersection syndrome

A. **Definition: Inflammation and pain at the site where the first dorsal compartment tendons** (APL and EPB) cross the second dorsal compartment tendons (ECRL and ECRB).

B. **Associated with repetitive wrist motion.**

C. **Pain and crepitation with wrist motion at the intersection site.**

D. **Treatment.**
 1. **Forearm-based wrist splinting.**
 2. **Activity modification and rest.**
 3. **Injection with steroid and lidocaine.**
 4. **Surgery uncommon.**

III. Other common forms of tendonitis

A. **Flexor carpi radialis/ulnaris (FCR/FCU) tendonitis.**
 1. **Associated with forceful repeated wrist flexion (overuse).**
 2. **Inflammation and tenderness along FCR/FCU** and pain with resisted wrist flexion and radial/ulnar deviation.
 3. **Treatment:** Nonsteroidal antiinflammatory drugs (NSAIDs), splinting, activity modification, occasionally injection; surgery is uncommon.

B. **Extensor carpi ulnaris (ECU), extensor pollicis longus (EPL), and extensor indicis proprius (EIP) tendonitis also occur.**
 1. **Associated with overuse.**
 2. **Treatment:** NSAIDs, splinting, activity modification, occasionally injection.
 3. **Surgery for EPL tendonitis to prevent rupture.**

C. **Rheumatoid arthritis–related** tendon attrition and rupture: See Chapter 40, "Rheumatoid Arthritis, Osteoarthritis, and Dupuytren's Contracture."

38 Amputation, Replantation, and Fingertip and Nail Bed Injuries

Keith G. Wolter

Amputation

I. **Emergency department assessment**
   A. What is the level of the injury?
   B. What is the mechanism and type of injury (sharp, dull, compression, avulsion, crush)?
   C. What are the patient's occupation, mental health, and level of cooperativeness?
   D. Which is the dominant hand?
   E. Is the patient a smoker? How much?
   F. What are the patient's perceptions of injury as it relates to body image? (May have a cultural component.)
   G. What are the patient's expectations? (May be unrealistic.)
   H. What is the patient's age? (Children heal more quickly and adapt more easily to changes in form and function.)
   I. What other injuries exist?
   J. Is replantation possible? Is it a worthwhile option? (See "Replantation.")
II. **Amputation goals**
   A. Preserve function
   B. Create a durable amputation site
   C. Preserve sensation; prevent neuromas
   D. Minimize disfigurement of adjacent joints
   E. Allow early return to work and activity
III. **Distal finger amputations**
   A. **Assess injury**
      1. Dorsal versus volar
      2. Angle of injury
      3. Involvement of nail and/or nail bed (see "Fingertip and Nail Bed Injuries")
      4. Exposure of bone
   B. **If no exposed bone**
      1. **Secondary intention healing.**
         a. Gives the best results in most cases.
         b. Treat with dressing changes and antibiotic ointment to keep moist and clean.
         c. Cold intolerance is common, but no worse than with other treatment options.
      2. **Primary closure** is an option only if tissue loss is minimal; otherwise, tight closures can limit function and cause pain.
      3. **Skin grafts**
         a. Recovery of sensation is not as good as with secondary intention healing.
         b. If used, the best alternative is a full-thickness skin graft. The best donor site options include the following:
            (1) Original skin (if salvageable). This skin should be aggressively trimmed of all fat and even some dermis.

            (2) Skin from ulnar/hypothenar aspect of hand.
            (3) Volar wrist skin.
            (4) Antecubital skin.
         c. Split-thickness skin grafts should only be used on noncritical areas (i.e., ulnar side of index, middle, and ring fingers).
   C. **If bone is exposed**
      1. **Completion amputation:** Bone shortening and primary closure.
         a. Allows quick return to work.
         b. Best option for a patient unlikely or unwilling to do dressing changes.
      2. **Bone shortening and healing by secondary intention.**
         a. Patients are often skeptical about outcome initially.
         b. If patient is able to tolerate dressing changes, then this is a good option.
      3. **Fingertip flaps** (see "Fingertip and Nail Bed Injuries").
         a. Many surgical options have been described; however, these procedures will not necessarily result in better outcomes or quicker recovery.
         b. Individual patient presentation and the surgeon's preference and expertise play large roles in determining the treatment method to use.
IV. **Proximal finger amputations**
   A. **Amputations through joints** (distal interphalangeal or proximal interphalangeal joints)
      1. The bone end must be debrided. Use a rongeur to smoothly contour the distal end by removing the condylar prominences and irregular bone spikes.
      2. Digital nerves must be transected proximally to prevent painful neuroma formation.
      3. The extensor and flexor tendons should be debrided, but take care not to suture the ends of them together, as this will limit the excursion of both.
         a. If the flexor digitorum profundus (FDP) tendon is shortened and tethered, then the **quadrigia effect** can occur. The FDP tendons share a common muscle belly. If one tendon is shortened, than the others cannot be fully contracted, leading to the inability to make a fist.
         b. Another potential problem in amputations at the distal interphalangeal (DIP) level is the **lumbrical-plus deformity**. If the FDP is severed from its insertion and migrates proximally, it pulls on the lumbrical; attempts to flex the digit cause proximal interphalangeal (PIP) extension (from FDP pull on lumbrical tendon, pulling on extensor mechanism). Sectioning of the lumbrical tendon is the treatment.
   B. **Middle and proximal phalanx amputations**
      1. A fish-mouth closure of the skin is used, with the incision oriented transversely across the end of the stump.
      2. Tendons, when preserved, are secured to their insertion on the phalanx.
      3. If an amputation occurs too proximally along the middle phalanx to allow resecuring of the tendon to the bone, or if the tendon is missing, then use of the next joint will be limited. Length preservation remains preferable, even if the joint is nonfunctional. In this case, the joint should be fused.
      4. Amputations near the metacarpophalangeal (MP) joint often result in problems with small object manipulation, especially in ring and middle finger amputations.
         a. Consideration should be given to eventually converting to a ray amputation to maximize function.
         b. Alternatively, prosthetic finger replacement is possible.
V. **Metacarpal and carpal amputations**
   A. **Ray amputations**
      1. **Injuries at or near the level of the MP joint** usually benefit from removal of most of the bone and closure of the space between remaining digits.
         a. Leaving a "gap" in the fingers can allow small objects to slip through.
         b. An amputation of the index finger at the MP level leaves a stump that can interfere with thumb use and creates a bulky web space.

c. The overall appearance of the hand is better if the stump is removed and any gap closed, although the palm is made narrower in the process.

2. **Ray amputations** are carried out electively at a later time, after the wound has healed.
3. **Central ray (middle or ring finger) amputations** leave defects that must be closed between the remaining metacarpals.

B. **Carpal amputations**
1. Initially, the treatment is tissue preservation.
2. Functional recovery is poor. Some patients may opt for more proximal amputation, followed by fitting with a hand prosthesis.
3. Alternatively, the tissue at the hand base can be preserved and used to anchor a nonfunctional cosmetic appliance.

VI. **Amputations at and proximal to the wrist**

A. **Wrist disarticulation.**
1. Once felt to be inferior to a long below-elbow amputation; now performed increasingly, in part because of improvement in prostheses.
2. Preserving the radioulnar joint allows for a full range of pronation and supination.
3. A fish-mouth skin closure, with a longer skin flap on the palmar side, is used.

B. **Below-elbow amputation:** The goal is length preservation. More length preservation of the radius and ulna means greater pronation and supination; ideally, 65% to 80% of length should be preserved for maximal function.

C. **Elbow and above-elbow amputations.**
1. Humeral condyle preservation, when possible, allows for translation of rotation to the eventual prosthesis. Therefore, an elbow disarticulation is a very adequate level of amputation.
2. In above-elbow amputations, length preservation is key. Amputations proximally, at or above the axillary fold, have no real advantage versus shoulder disarticulations.

Replantation

I. ***Evaluation for replantation***

A. **"Life before limb":** Patients may have other serious injuries, which must be addressed prior to any attempt at replantation.

B. **Assess injury,** as outlined earlier in "Amputation" section.

C. **Additional history is** needed before considering replantation.
1. Overall patient health and comorbidities.
2. Previous injuries to this extremity.
3. Willingness of patient to comply with rehabilitation and to tolerate lengthy time off work (average time until return to work is 7 months), as well as future operations.

D. **Obtain x-rays of** both the hand and the amputated part. Give the patient a tetanus update and/or antibiotics if indicated, check the hematocrit, and perform fluid resuscitation.

E. ***Assess the amputated part and stump site.***
1. **Sharp amputations** do better than avulsion or crush amputations.
2. **Length of ischemia time of** the amputated part is critical.
   a. Digits can tolerate up to 12 hours of warm ischemia time or 24 hours (or more) of cold ischemia time.
   b. More proximal amputations, which include muscle, tolerate less ischemia: 6 hours of warm ischemia or 12 hours of cold ischemia is considered the limit of viability for wrist or more proximal replantations.

II. ***Indications for replantation***

A. ***Indications to attempt replantation.***
1. Thumb amputations.
2. Multiple-finger amputations.

3. Amputation in child.
4. Amputation at the palm, wrist, or forearm level.
5. Single-digit injury distal to flexor digitorum superficialis (*FDS*) insertion (does well functionally).

B. **Absolute contraindications to replantation.**
1. Life-threatening injuries.
2. Prolonged ischemia time of part.
3. Part in multiple pieces (i.e., transected at more than one level).

C. **Relative contraindications.**
1. Severe crush/avulsion amputations.
2. Injuries at multiple levels.
3. Severe preexisting illness.
   a. Diabetes mellitus.
   b. Heart disease and/or atherosclerosis.
   c. Recent stroke and/or myocardial infarction.
   d. Psychiatric disorders.
4. Gross contamination of site.
5. Prior surgery or trauma to amputated part.
6. Smoking history.

D. **A number of controversies persist.**
1. Amputation proximal to the elbow: Technically possible, but functional outcomes are quite poor. The success is greater in children.
2. Amputation of single finger proximal to the FDS insertion: Some surgeons feel that poor functional outcome negatively affects the use of the remaining digits and leads to a less functional situation than amputation.

III. **Preoperative care**

A. **Care of amputated parts:** The part should be gently cleaned, wrapped in saline-moistened gauze, and placed in a sealed plastic bag. The bag is stored at 4°C for transport.
1. Saline-ice bath will maintain the proper temperature.
2. Do *not* place the finger directly on ice; freezing the part is worse than warm ischemia.

B. **Consent.**
1. Obtaining surgical consent for replant *attempts* is not trivial.
   a. The patient and/or family will likely have unrealistic expectations prior to counseling.
   b. The extensive recovery time, the need for rehabilitation, and the likely amount of optimal function should be explained in detail prior to replantation attempts.
   c. The possibility of long hospitalizations, multiple operations, heparinization, and blood transfusions must be recognized.
   d. The significant chance of failure must also be addressed.
2. Consent must provide for a number of contingencies.
   a. Vein graft.
   b. Nerve graft.
   c. Skin graft.
   d. Flap for coverage.
   e. Revision amputation.

IV. **Operative techniques**

A. **Amputated part**
1. Begin work preparing the amputated part on the "back table" prior to patient arrival to the OR.
2. Carefully expose the vessels and nerves, and tag their ends with fine Prolene suture.
   a. For fingers, use midaxial longitudinal incisions.
   b. A corkscrew appearance to the arteries suggests traumatic stretch from an avulsion. These will need debridement.
   c. Bruising of the neurovascular bundle also suggests avulsion or traction injury.

3. Preservation of "spare parts" may optimize outcome.
   a. Heterotopic replants transfer tissue from one site to another (e.g., thumb restoration with another amputated digit when the thumb is unsalvageable).
   b. Use of components from one amputated part for another part's replant is economical (e.g., digital nerves from another amputated, but unreplantable, finger).
4. When the amputation is more proximal, the part will contain muscle, which will swell after reperfusion. Therefore, any fascial compartments in the part must be released.

B. Operative overview

1. **The recommended replant sequence is as follows.**
   a. Prepare the stump; debride the wound.
   b. Identify arteries, veins, nerves, and tendons; place tagging sutures.
   c. Stabilize the bone(s).
   d. Repair extensor tendons and muscles, then flexor tendons and muscles.
   e. Coapt nerves.*
   f. Anastomose arteries.*
   g. Anastomose veins.*
   h. Cover wound with soft tissue and/or skin.
2. The sequence for the repairs marked by asterisks is altered depending on circumstances and surgeon preference.
   a. In patients with short ischemia times, some authors feel nerve repair can and should precede vascular repair.
   b. In amputations involving a part with significant muscle, the risk of prolonged ischemia and subsequent reperfusion injury is high. Therefore, arteries should be repaired first, then nerves, and finally veins. This sequence provides inflow to the amputated part and the "flushing out" of toxic metabolites while the nerves are being fixed, before the veins are anastomosed.

C. Stump preparation: Avulsion injuries involving tendons sometimes require fasciotomies or carpal tunnel release, or both, because swelling proximal to the level of the amputation will occur.

D. Bone fixation

1. Bone should be debrided. Shortening is beneficial in that it decreases tension on anastomoses and skin repairs.
2. Fixation can be achieved via various methods.
   a. Kirschner wires: Simple; very useful for fingers. Placed retrograde in amputated part first.
   b. Interosseous wires: Used to augment K-wire repairs.
   c. Plate fixation: Not typically necessary for phalanges; useful for amputation at or proximal to the metacarpals.
   d. External fixation: May be useful for forearm replants.

E. Tendon and muscle repair

1. Clean the tendon edges, but do not shorten excessively.
2. Extensor tendons are repaired first, with two or three horizontal mattress sutures using 4–0 braided polyester.
3. Next, repair flexor tendons with a core suture technique, such as a modified Kessler or Tajima repair (see Chapter 37, "Tendon Injuries and Tendonitis").

F. Vessel anastomoses

1. **Arterial repair**
   a. The artery must be trimmed back to healthy intima.
   b. Vein grafts of the appropriate size may be found in the volar forearm, in the dorsal foot, or in "spare" amputated parts.
   c. Papaverine and/or lidocaine is used to minimize vasospasm.
   d. Repair of two arteries to a digit yields a higher successful replantation rate than repair of a single artery, but one good anastomosis is adequate.

2. **Venous repair**
   a. Two vein repairs per artery are preferred.
   b. Tension on venous repair must be minimal to prevent congestion.

G. Nerve coaptation

1. Trim the nerves back to undamaged areas.
2. Realign fascicles when possible to maximize the return of sensibility.

H. Coverage

1. Skin is closed loosely over repaired vessels. A tight closure will restrict venous outflow.
2. Split-thickness skin grafts are used as needed.
3. For more proximal replants, local or free muscle flaps are used to cover the operative site and protect the anastomoses.
4. A well-padded splint, with absolutely no circumferential pressure, should be made to protect the replant. It should extend above the elbow to prevent any rotational movement. A poorly made dressing can foil the whole case.

V. Postoperative care

A. Acute care

1. **Aggressive hydration** to keep vessels patent (usually a total fluid intake of one and a half maintenance).
2. **Avoid *any* vasoconstrictors** for the first several days after the operation, including caffeine and nicotine.
3. **Analgesia** is important to minimize catecholamine release. The patient should be resting comfortably.
4. **Medical therapies** used to diminish complication rates.
   a. Systemic heparinization should be used in cases of wide vessel damage, such as in crush amputations. Initiate therapy intraoperatively for the best result.
   b. Dextran 40 infusion is used by many replant surgeons for its plasma expansion and antiplatelet effects. A dosage of 500 cc is administered per day in adults (or 25 cc/hr). A test dose of 5 cc is usually administered in the operating room. Although statistical proof of efficacy is lacking, side effects (anaphylaxis, acute renal failure, pulmonary edema) are rare.
   c. Aspirin 325 mg a day is given for 3 weeks to retard platelet aggregation.
   d. Other agents are advocated by some authors, including chlorpromazine (Thorazine), dipyridamole (Persantine), and calcium channel blockers.
5. Objective monitoring of the replant can be done in a number of ways.
   a. Temperature probes: Probably the most reliable.
   b. Laser Doppler flowmetry.
   c. Pulse oximetry.
6. Frequent evaluation by the surgeon and staff for color and capillary refill (subjective monitoring) is essential and represents the best monitoring method.

B. Failing replant

1. **In the acute setting,** the problem is usually vascular, either inflow or outflow.
   a. Arterial insufficiency: Cool, pale replant; no capillary refill; pin prick produces little or no bleeding.
   b. Venous insufficiency: Congested replant; increased tissue turgor; pin prick yields copious bleeding with dark blood.
2. **Initial treatment options** include nonoperative measures.
   a. Elevate hand and arm.
   b. Loosen dressing; if needed, relax or release sutures.
   c. Add medical therapies (heparin, Thorazine, etc.).
   d. Use better pain control (e.g., axillary block).
   e. Use medicinal leeches.

(1) Can reduce venous congestion.
(2) Secrete hirudin, a potent anticoagulant that remains localized.
(3) Treatment lasts for up to 6 days.
(4) Patients should be placed on a third-generation cephalosporin to protect from *Aeromonas hydrophila* infection.

f. Use of thrombolytics in replants is controversial.

3. **Reexploration** is the definitive treatment for vascular problems.
   a. Functional outcomes are poorer in patients requiring reoperation.
   b. Outcomes are best when reoperation is performed within 6 hours of loss of perfusion.

VI. Outcomes

A. **With good patient selection,** replant failure rate is low, on the order of 20%. However, that number may be deceiving, because *viable* replants are not always *valuable* replants.

B. **Late complications diminish the value of a replantation.**
1. **Decreased range of motion** in joints.
   a. Due to tendon adhesions and joint contracture.
   b. Many patients undergo one or more secondary procedures to address these problems and improve replant function, especially in more proximal amputations.
2. **Decreased sensation** is a function of injury mechanism, repair technique, and level of injury.
3. **Loss of motor function is** a problem in more proximal amputations, where the slow axonal regeneration limits muscle reinnervation.
4. **Chronic pain,** including chronic regional pain syndrome (CRPS).
5. **Cold intolerance** is a very common postreplant complaint. It typically improves over 2 years, but some intolerance is often permanent.

C. **Functional outcome** depends on multiple factors.
1. Sharp amputations always have better recovery of sensation and function than crush or avulsion amputations.
2. Children have better outcomes.
3. Thumb replants do best. Even if mobility is poor, the replant has value as a sensate post.
4. Zone I finger replants regain an average of 82 degrees of motion at the PIP joint.
5. Zone II finger replants regain an average of 35 degrees of motion at the PIP joint.
6. The average two-point discrimination in a finger replant is 11 mm.

Fingertip and Nail Bed Injuries

I. Overview

A. Everything distal to the DIP crease is considered the fingertip (Fig. 38-1).
B. The fingertip is the most sensitive part of the hand.
C. The glabrous skin on the fingertip is specialized for **pinch** and **grasp** functions.
D. The nail protects the distal phalanx and provides counterforce to the tip pulp.
E. Fingertips are essential for normal hand appearance.
F. Fingertips are commonly injured.
1. Fingertip and nail injuries account for 45% of all hand injuries seen in the emergency room.
2. The middle fingertip is most commonly injured, followed by the ring fingertip.
3. Thumb tip injury is least common.

G. Fingertip injuries can have a great impact.

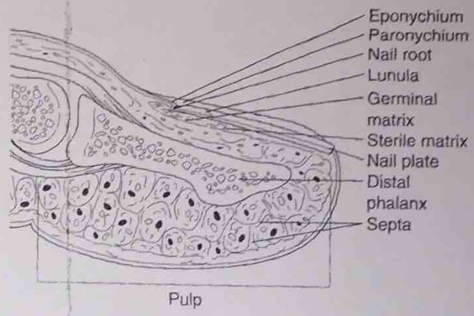

Fig. 38-1. Anatomy of the fingertip.

1. May appear minor, but can have serious implications because of the effect on so many activities.
2. Typically result in lost work (sometimes the end of a career).

H. Immediate repair is preferable for the best outcome. Repair can often be performed in the emergency room.

II. Preparation and examination

A. A digital nerve block is usually sufficient.
B. In sterile fashion, prep the hand to the forearm, and secure on an arm board or table.
C. Place a tourniquet on the finger, using a Penrose drain and hemostat or similar method to ensure tourniquet removal. Do *not* roll a cut glove finger onto the digit, because this band may be forgotten and can result in digit loss. A simple sterile glove secured by a hemostat works very well.
D. Surgical loupes are helpful.
E. Irrigate the fingertip first, then debride any clearly nonviable tissue.
F. Have a low threshold for moving to the operating room when necessary.

III. Fingernail injuries

A. **Nail bed hematomas**
1. If less than 25% of surface of the nail in size, drain by lancing the nail with cautery or a heated paperclip end.
2. If greater than 25%, remove the nail to repair the nail bed.

B. **Nail bed lacerations**
1. Use a Freer elevator or tenotomy scissors to separate the nail from the eponychium and underlying matrix.
2. Set the nail aside in sterile saline.
3. Repair the nailbed with 6–0 or 7–0 absorbable suture material. Use precise, interrupted stitches under loupe magnification.
4. Maintain the eponychial fold with a stent. One can use the trimmed nail if available, or other material such as the foil from a suture pack. This is usually considered helpful to prevent the formation of painful split nails.

C. **Avulsed nail bed**
1. If attached to nail, replace as an onlay graft.
2. If the nail bed is missing, use a split-thickness nail bed graft from another nail to fill the defect (usually electively).

D. **Amputations through the lunula/germinal matrix:** If unable to repair or replant, remove the remaining germinal matrix before closure. Otherwise, the remaining matrix will form an irritating, painful nail remnant.

IV. Flaps for fingertip repair

A. As discussed earlier, healing by secondary intention often gives as good or better outcomes than flap repairs. A variety of techniques can be used to repair fingertip injuries. The angle of injury or amputation, as well as individual surgeon experience, determines when and where to use a given technique.

B. **Advancement flaps**

1. **Lateral V-Y advancement flaps (Kutler flaps)**
   a. Most useful for transverse amputations.
   b. Bilateral triangles are advanced and sutured to distal nail bed.
   c. Can advance up to 5 mm if skin alone, and up to 14 mm if a neurovascular flap is elevated down to the level of the periosteum.
   d. Disadvantages.
      (1) Vascular supply is sometimes unreliable.
      (2) Scar is at the tip; may be painful or insensate.
2. **Volar V-Y advancement flap (Atasoy-Kleinert flap)**
   a. Most useful for dorsal oblique amputations.
   b. Triangular flap, with base no wider than nail bed.
   c. Skin incisions are through the dermis; deep aspect is dissected off of phalanx.
   d. Advancement of up to 10 mm.
   e. Good survival; disadvantages include possible hypersensitivity or hook nail.
3. **Volar neurovascular advancement flap (Moberg flap)**
   a. Best sensation preservation.
   b. Longitudinal incisions are made on both sides, dorsal to the neurovascular structures. Nerves and arteries are contained in the flap.
   c. Advanced to cover tip defect.
   d. Requires some joint flexion during healing; therefore, there is a high risk of flexion contracture.
   e. Used for the thumb tip, when padding and sensation are critical and some flexion contracture can be tolerated. The Moberg flap should generally not be used for fingers.

C. **Regional flaps**

1. **Cross-finger flap**
   a. Dorsal skin from one digit is transferred to the injured area of an adjacent digit; can be used for volar or dorsal amputations.
   b. Pedicled flap with delayed division, usually in 2 to 3 weeks.
   c. Donor site requires a skin graft.
2. **Thenar flap**
   a. The injured digit is flexed and tucked into the thenar area, and the palmar skin is used to cover the tip.
   b. Advantage: No defect on fingers adjacent to injury.
   c. Disadvantage: PIP flexion contracture of recipient finger. Therefore mostly used in children.
3. **Neurovascular island transfer flap (Littler flap)**
   a. Used for insensate fingers following trauma to recreate sensibility in the tip.
   b. Usually reserved for thumb, index finger, or ulnar little finger.
   c. Must balance recipient sensation restoration with donor site loss.
   d. Flap pedicle is composed of digital vessels and nerve.
   e. Typically raised from the ulnar aspect of the ring or middle finger; raised at the level of the flexor sheath.
   f. Donor site is closed either with graft or primarily.

39 Nerve Injuries, Compression Syndromes, and Tendon Transfers

Keith G. Wolter

Nerve Injury

I. Peripheral nerve anatomy

A. **Neurons:** The basic unit of nervous tissue.

1. **Motor neurons**
   a. Large, multipolar cell body, with multiple dendrites and a long axon.
   b. The cell body is located in the ventral horn of the spinal cord; the axon leaves the spinal cord via the ventral root.
   c. Terminate on the motor end plate of the innervated muscle.
2. **Sensory neurons**
   a. The cell body is found in the dorsal root ganglion.
   b. Unipolar, with a single axonal process running from the periphery (either an encapsulated receptor in the skin or a terminal branch in tissue), through the dorsal root ganglion, and into the spinal cord.
   c. Terminate in the dorsal horn of the spinal cord, or may ascend to the brainstem.
3. **Sympathetic neurons**
   a. Control vasomotor and pilomotor function.
   b. Axons are unmyelinated.

B. **Glial cells:** Produce myelin to insulate axons and increase conduction speed.

1. **Schwann cells** are the primary glial cell of the peripheral nervous system.
   a. Schwann cells produce a sheath of myelin that wraps multiple times concentrically around the axon.
   b. Each Schwann cell covers a segment of axon 1 mm in length.
   c. Spaces between Schwann cells are the nodes of Ranvier.
2. Peripheral nerve fibers can be myelinated or unmyelinated.

C. **Connective tissue:** Makes up 25% to 75% of the cross-section of nerves; it has three layers.

1. **Endoneurium**
   a. Within the fascicle, individual axons are surrounded by endoneurium.
   b. Unmyelinated nerve fibers have a diameter ranging from 0.2 to 3.0 μm.
   c. Myelinated fibers have diameters ranging from 2 to 25 μm.
2. **Perineurium**
   a. Surrounds individual fascicles within nerves.
   b. Each fascicle represents a group of nerve fibers.
   c. Fascicular diameter ranges from 0.04 to 3.0 mm.
3. **Epineurium**
   a. **External epineurium** surrounds the whole nerve as an outer sheath.
   b. **Internal epineurium** is composed of loose connective tissue and serves to cushion the fascicles from trauma.
   c. Contains a longitudinal plexus of blood vessels.
   d. Usually the site for suturing during nerve reconstruction.

D. **Nerve topography**

1. Peripheral nerves can be:

a. **Monofascicular** (e.g., terminal skin branch): One fascicle, either pure motor or sensory.
b. **Oligofascicular** (e.g., common digital nerve): Either purely motor or sensory, or mixed function.
c. **Polyfascicular** (e.g., radial nerve): Major nerves contain fibers for a variety of functions.

2. The organization of the fascicles in the major (polyfascicular) upper extremity nerves has been mapped, with detailed study of the pattern of motor and sensory axons along the length of the nerve. These maps are critical during fascicular nerve repair procedures.

II. Chronology of nerve injury: Degeneration followed by regeneration.

A. Nerve degeneration

1. Cell bodies of injured axons swell, and alter their metabolism to produce proteins needed to rebuild the axons.
2. **Wallerian degeneration** occurs along the axon at and distal to the level of injury. The distal axon degrades, and Schwann cells withdraw. Macrophages clear debris from the axonal tract. Wallerian degeneration can also extend up to 2 cm proximal to the injury site.
3. Synapses distal to the nerve injury degrade.

B. Nerve regeneration

1. Injured peripheral nerves can regenerate, provided continuity with the distal portion of the nerve tract is maintained or is reestablished surgically.
2. **Nerve growth factors,** secreted by target cells (postsynaptic neurons or muscle cells) and by Schwann cells, are required for axonal regrowth.
3. **Macrophages** secrete interleukins that induce Schwann cell proliferation.
4. **Schwann cells** along the distal axonal tract express laminins and adhesion molecules, which help guide the regenerating axon.
5. **Axonal sprouts** from the proximal cut end must enter the distal tract to regrow. If disruption of the nerve is severe and/or scarring is great, the budding axons cannot cross the gap, and regeneration does not occur.
6. Prior to any regeneration, degradation and clearing of debris takes approximately 15 to 30 days.
7. Once growth is initiated, axons extend by approximately 1 mm a day.

III. Nerve injury classification

A. Seddon nerve injury classification system: Described by Sir Herbert Seddon.

1. **Neuropraxia:** Local transient block of conduction along a nerve. The anatomy of the nerve is preserved, and no Wallerian degeneration occurs. Recovery is usually rapid, but may take several months. Selective demyelination of fibers may occur.
2. **Axonotmesis:** Axonal damage within the nerve. Anatomic continuity is preserved. Wallerian degeneration occurs. Recovery rate is 1 mm per day along the nerve, once healing begins. Fibrillations are present on electromotor testing. Recovery is typically complete eventually (without surgery).
3. **Neurotmesis:** Nerve is transected. Continuity of the nerve is lost, and Wallerian degeneration occurs. Some recovery may occur, but it is never complete; surgical repair is needed for the best outcome.

B. Sunderland nerve injury classification system (with Mackinnon modification)

1. **First-degree injury:** Nerve is demyelinated, resulting in a local conduction block. Treatment is nonoperative, and recovery is complete within approximately 12 weeks.
2. **Second-degree injury:** Some nerve fibers are disrupted, but the endoneurial sheaths remain intact. Wallerian degeneration occurs with second-degree and higher injuries. Tinel's sign indicates an advancing growth cone. Treatment is nonoperative. Complete recovery is expected.

3. **Third-degree injury:** Some endoneurial sheaths are disrupted with scarring, while the perineurium remains intact. Incomplete recovery; some nerve fibers do not reinnervate their target. Treatment is usually nonoperative.
4. **Fourth-degree injury:** Loss of continuity of the perineurium. Scar blocks all fiber growth; little or no nerve recovery. Treatment is operative.
5. **Fifth-degree injury:** The nerve is completely transected. The epineurium is disrupted. No recovery is expected without operative management.
6. **Sixth-degree injury:** Added to Sunderland's classification scheme by Susan Mackinnon.

Nerve Compression Syndromes

I. General principles

A. Pathophysiology

1. **Ischemia:** Pressure on the nerve leads to loss of blood flow (Table 39-1).
2. **Fibrosis:** Ischemia leads to nerve fibrosis, which worsens in progressive stages (Table 39-2).
3. **Traction:** Entrapment can limit nerve excursion. With limb motion, this can lead to additional traction-induced conduction block.
4. **Double-crush phenomenon:** A given locus of compression impairs axonal transport along the nerve, lowering the threshold for a second locus to become symptomatic (e.g., thoracic outlet plus carpal tunnel syndrome).
5. **Systemic conditions:** Can depress peripheral nerve function, which in turn lowers the threshold for symptoms (diabetes, alcoholism, hypothyroidism, and exposure to industrial solvents).

B. Diagnostic testing

1. **Sensory testing:** Use threshold tests to evaluate compression neuropathies.
   a. **Semmes-Weinstein test:** Monofilament test for slow-adapting touch fibers.
   b. **Vibration test:** Best test for quick-adapting touch receptors.
2. **Tinel's sign:** The skin is percussed over the course of the nerve. Tingling over the nerve distribution indicates a partial lesion of the nerve. After nerve repair, this may be a positive finding, indicating nerve regeneration.
3. **Phalen's test:** The wrist is palmar flexed to 90 degrees (can be accomplished by having the patient press the backs of his or her hands together) and held in this position for 60 seconds. Patients with carpal tunnel syndrome will exhibit hypesthesia/paresthesia in the median nerve distribution of the affected hand(s).
4. **Electrodiagnostic testing:** The gold standard; provides objective data to support or disprove a compression neuropathy.

Table 39-1. Effects of increasing pressure on peripheral nerves

Pressure (mm Hg)	Effect on Nerve
20–30	Reduced epineurial blood flow
30	Impaired axonal transport
30–40	Paresthesias
50 (for 2 hr)	Epineurial edema; axonal transport block
>60	Complete intraneural ischemia

Table 39-2. Management and outcome of fibrosis of peripheral nerves

Stage	Treatment	Expected Outcome
Early	Splinting, steroids	Full recovery
Intermediate	Operative release	Full recovery
Advanced	Operative release	Permanent nerve loss

a. Electrodiagnostics are limited by operator experience and reliability.
b. Typically, nerve conduction studies and electromyography (EMG) are performed at the same time to assess both nerves and muscles.
c. In compression mononeuropathies, there is a drop in amplitude of nerve action potentials, decreased nerve conduction velocity, and prolonged latency.
d. Results are more useful if compared with old tests on the same individual and/or on contralateral nerves; population norms may be used, but individual patient values can vary widely.
e. Nerve conduction and EMG can help establish a diagnosis, but in isolation do not establish one. Patients must have symptoms first to have a diagnosis made.

5. **Radiologic studies:** Plain films and/or magnetic resonance imaging (MRI) as needed, to rule out the following.
   a. Fractures and post-traumatic deformities (e.g., hamate fracture).
   b. Congenital deformities (e.g., cervical rib).
   c. Neoplasms and masses (e.g., Pancoast's tumor).

II. Median nerve

A. Carpal tunnel syndrome (CTS) is the most common mononeuropathy.

1. **Epidemiology**
   a. Mean age of diagnosis is 50 years.
   b. Intrinsic risk factors: Female gender, pregnancy, diabetes, and rheumatoid arthritis.
   c. Controversial risk factors: Repetitive or forceful tasks, mechanical stress; occupational posture; vibration and/or temperature.
   d. *Not* a risk factor: Anatomically small carpal tunnel.
2. **Anatomy**
   a. Carpal tunnel boundaries.
      (1) Dorsal and lateral: The carpal bones.
      (2) Volar: The flexor retinaculum, consisting of the deep forearm fascia, transverse carpal ligament (TCL), and the distal fascia between the thenar and hypothenar muscles.
   b. The carpal tunnel contains the median nerve and nine tendons: the flexor pollicis longus (FPL), flexor digitorum superficialis (FDS, four), and flexor digitorum profundus (FDP, four).
   c. Median nerve branches of the hand.
      (1) The palmar cutaneous branch: Arises 5 cm proximal to the wrist crease.
      (2) The recurrent motor branch: Usually arises at or just beyond the distal edge of the TCL from the radiopalmar aspect of the nerve (many variants are described). **Kaplan's cardinal line** runs from the apex of the thumb/index web toward the hook of the hamate, parallel with the proximal palmar crease; the intersection of this line with the long finger localizes the recurrent motor branch.
      (3) Two common and three proper digital nerve branches: Arise distal to the TCL.
3. ***Pathophysiology***
   a. Vascular compromise: Leads to localized anoxia. Edema follows, resulting in further reduction of blood flow.

   b. Inflammation: A function of both ischemia and external repetitive stress; can cause synovial hypertrophy of the flexor tendons.
   c. Fibroblast proliferation: Interferes with nerve oxygenation and nutrition.
   d. Scarring and compression: Can tether the median nerve in the carpal canal, leading to traction injury.
   e. In general, the pathology is **progressive.** Stopping the compression earlier leads to quicker and more complete recovery.
4. **Diagnosis**
   a. **History and physical examination**
      (1) Pain and paresthesias of the palmar radial hand. Worse at night and with repetitive use.
      (2) Phalen's test and Tinel's signs may be positive; these support a CTS diagnosis but are not specific.
      (3) Advanced cases may demonstrate wasting of the thenar muscles.
   b. **EMG findings**
      (1) Distal motor latencies greater than 4.5 msec (or >1 msec difference between the hands).
      (2) Distal sensory latencies greater than 3.5 msec (or >0.5 msec difference between hands).
      (3) Conduction velocities less than 50 m/sec.
5. **Nonoperative treatment**
   a. Splint the wrist in neutral position. Have the patient wear the splint all the time or just at night, depending on the severity of symptoms.
   b. Antiinflammatory agents such as NSAIDs, to decrease inflammation.
   c. Control of systemic diseases (e.g., diabetes, rheumatoid arthritis).
   d. Steroid injections
      (1) Inject 1 cm proximal to the distal wrist crease between the palmaris longus (PL) and flexor carpi radialis (FCR) tendons.
      (2) Transient relief for 80% of patients, but few remain symptom free after 1 year.
      (3) Better results in milder cases of CTS, with symptoms present for less than 1 year.
      (4) Can be beneficial for debilitating CTS in pregnancy.
      (5) Response to steroids is also diagnostic, predictive of a good response to surgery.
6. **Operative treatment: Carpal tunnel release (CTR)**
   a. Objective: Release of the superficial (palmar) and deep (TCL) layers from the antebrachial fascia to the distal palmar crease.
   b. **The standard open technique** provides better exposure.
      (1) Incision is ulnar and parallel to the thenar crease, 2.0 to 2.5 cm long.
      (2) Do not need to cross the wrist crease with the incision.
      (3) Cut the TCL with care to avoid injury to the underlying nerve.
   c. **Minimal-incision techniques** require more blind dissection.
      (1) Incision is halfway between the distal wrist crease and a transverse line intersecting the base of first web space made in line with the radial border of the fourth ray, just ulnar to the thenar crease; 0.7 to 1.0 cm in length.
      (2) Use a scalpel to open the tensor fascia lata (TFL), then tenotomy scissors to divide the TCL first distally, then proximally.
      (3) The antebrachial fascia is divided blindly.
   d. **Endoscopic techniques:** Leave even smaller scars, but no superiority has been demonstrated over open methods.
   e. **Synovectomy** is only indicated in cases of proliferative or invasive tenosynovitis.
   f. **Co-release of Guyon's canal** is not indicated in isolated CTS.
   g. Postoperative splinting is optional; use for 1 week or less to minimize debilitation.

B. Pronator syndrome and anterior interosseous syndrome
1. **Sites of compression** (from proximal to distal)
   a. Supracondylar process of the humerus
   b. Ligament of Struthers (accessory origin of the pronator teres)
   c. Accessory bicipital aponeurosis
   d. Pronator arch: Between the humeral and ulnar heads of the pronator teres muscle (most common cause of these syndromes)
   e. Flexor digitorum superficialis arch
   f. Fibromuscular bands in the distal forearm
   g. Accessory/variant muscles; for example, Gantzer's muscle (accessory head of FPL)
   h. Post-traumatic (radius or ulna fractures)
2. **Diagnosis**
   a. **Pronator syndrome**
      (1) Forearm pain and paresthesias; hypesthesia in the cutaneous distribution of the median nerve, including the palmar cutaneous branch, which helps differentiate it from CTS. There can be a positive Tinel's sign in the proximal forearm and a negative Phalen's test.
      (2) Symptoms with isolated, resisted contraction of the biceps, pronator teres (PT), and/or FDS may indicate the site of compression.
      (3) Fewer than 50% of EMGs are diagnostic; EMG findings poorly predict the response to surgery.
      (4) Use three-view elbow films to look at the supracondylar process.
   b. **Anterior interosseous syndrome**
      (1) Weakness or loss of function of the FPL, index finger FDP, long finger FDP, and pronator quadratus (PQ).
      (2) No sensory symptoms.
      (3) Classic "pinch deformity": flexion of the interphalangeal (IP) joint of the thumb with key pinch (Froment's sign).
3. **Treatment**
   a. **Pronator syndrome**
      (1) Nonoperative (splinting and rest): 50% of patients will respond.
      (2) Operative: Only for failure of conservative therapy. Must explore above and below the elbow to remove all possible sites of compression (see previous list). Start movement after 5 days.
   b. **Anterior interosseous syndrome**
      (1) Obtain a baseline EMG after 3 weeks of weakness, then observe for 2 to 3 months.
      (2) If there is no improvement in the exam or the EMG, then surgery is indicated.
      (3) Operative intervention is the same as above. Neurolysis of the anterior interosseous nerve to above the elbow should be performed to eliminate fascicular constrictions.

III. Ulnar nerve

A. Cubital tunnel syndrome
1. **Sites of compression** (proximal to distal)
   a. Arcade of Struthers (8 cm above the elbow, between the triceps and the medial intermuscular septum)
   b. Medial intermuscular septum (from the medial epicondyle to the coracobrachialis muscle)
   c. Medial epicondyle
   d. Cubital tunnel retinaculum (also known as arcuate ligament of Osborne)
   e. Deep flexor-pronator aponeurosis (2–3 cm distal to cubital tunnel, between FDP and FDS muscles)
2. **Pathophysiology:** Work-related activities may aggravate the syndrome, but there are no data to support work as a risk factor.
3. **Diagnosis**
   a. **History and physical examination**

Table 39-3. Treatment of cubital tunnel syndrome

Grade	Compression	Findings	Treatment
1	Minimal	No motor weakness	Nonoperative trial
2	Intermediate	Muscle atrophy	Nerve transposition
3	Severe	Paralysis/clawing	Nerve transposition

      (1) Numbness of the small and ulnar half of ring fingers and the ulnar dorsal hand.
      (2) Weakness of grip and intrinsic wasting.
      (3) Positive Tinel's sign or pain at the medial elbow.
      (4) Ulnar nerve subluxation with elbow flexion.
      (5) Ulnar nerve paresthesias/numbness within 1 minute of full elbow flexion (Phalen's analogue).
   b. **EMG findings**
      (1) Ulnar nerve conduction across the elbow is less than 49 m/sec.
      (2) The first dorsal interosseous muscle is the most commonly affected, with evidence of denervation.
      (3) Abductor pollicis brevis (APB) should be normal (this excludes a C8/T1 nerve root or plexus lesion).
   c. **Elbow x-rays:** Especially useful if range of motion is abnormal or there is a history of trauma.
4. **The treatment algorithm** was described by McGowan (Table 39-3).
5. **Operative treatment options**
   a. ***In situ* decompression:** Only for mild and/or intermittent symptoms. Use in patients with a nonsubluxating ulnar nerve, normal bony anatomy, and absence of pain around the medial epicondyle.
   b. **Subcutaneous anterior transposition:** Indicated in most cases.
   c. **Submuscular transposition:** Preferred for very thin patients, reoperative cases, and cases of severe compression.
   d. **Intramuscular transposition:** Least popular surgical option.
   e. **Medial epicondylectomy**
      (1) Useful for post-traumatic cases with bony deformity.
      (2) Carries the risk of damaging the ulnar collateral ligament, resulting in elbow instability and pain.

B. Ulnar tunnel syndrome
1. **Anatomy**
   a. Ulnar nerve analogue of the carpal tunnel. First described by Guyon in 1861; commonly referred to as **Guyon's canal.**
   b. Canal is 4.0 to 4.5 cm in length, extending from the proximal edge of the palmar carpal ligament to the fibrous arch of the origin of the hypothenar muscles.
2. **Pathophysiology** (various compressive etiologies)
   a. Ganglia and soft tissue masses: Most common cause (33%–50% of all cases).
   b. Muscle anomalies: 10% to 15%.
   c. Thrombosis or pseudoaneurysm of the ulnar artery.
   d. Fracture of the hook of the hamate.
   e. Edema/scarring from burns.
   f. Inflammatory arthritis.
3. **Diagnosis**
   a. **History and physical examination**
      (1) Pain in the wrist, with paresthesias radiating into the small and ring fingers; local tenderness.
      (2) Symptoms are exacerbated with sustained hyperextension or hyperflexion of the wrist.

(3) Intrinsic muscle weakness (a late finding).
(4) The ulnar palm is not affected, since compression is distal to the palmar (and dorsal) branches.
(5) A bruit may be present. Check Doppler studies, Allen's test, Phalen's test, and Tinel's sign.

b. **EMG:** Usually diagnostic.

c. **Computed tomographic scan:** Helpful in detecting hook of the hamate fractures.

4. **Nonoperative treatment**
   a. Indicated if no identifiable lesion is present.
   b. Activity modifications.
   c. Splint the wrist in neutral position.
   d. NSAIDs.

5. **Operative treatment**
   a. Indications: An identifiable lesion or failure of nonoperative treatment.
   b. Operative technique.
      (1) Divide the volar carpal ligament and pisohamate ligament.
      (2) Divide the fibrous arch of the hypothenar muscle's origin.
      (3) Examine the floor of the canal for masses and fractures; examine the ulnar artery with the tourniquet up and then down.
   c. Postoperative care: Splint the wrist in slight extension for 3 weeks.

IV. Radial nerve

A. Posterior interosseous nerve (PIN) syndrome and radial tunnel syndrome

1. **Anatomy**
   a. PIN innervates the extensor carpi radialis brevis (ECRB), supinator, extensor carpi ulnaris (ECU), extensor digitorum communis (EDC), extensor indicis proprius (EIP), extensor digiti quinti (EDQ), abductor pollicis longus (APL), extensor pollicis longus (EPL), and extensor pollicis brevis (EPB; no sensory component).
   b. Radial tunnel: 5 cm long. It is bounded by the biceps tendon, ECRL, ECRB, brachioradialis, and radiocapitellar joint capsule. It ends at the proximal part of the supinator.

2. **Sites of compression**
   a. Thickened fascial tissue superficial to the radiocapitellar joint.
   b. "Leash" of vessels from the radial recurrent artery **(leash of Henry).**
   c. Fibrous edge of the ECRB muscle.
   d. Proximal edge of the supinator **(arcade of Frohse).**
   e. Distal edge of the supinator, or fibrous bands within the supinator.
   f. Entrapment or traction injury from proximal radius surgery.

3. **Diagnosis**
   a. **PIN syndrome**
      (1) Gradual weakness of finger and wrist extensors.
      (2) Acute onset following trauma.
      (3) Rheumatoid disease at the elbow can mimic symptoms, including radial head dislocation placing traction on the PIN.
      (4) Incomplete syndrome may be confused with tendon rupture (check tenodesis).
      (5) EMG is usually diagnostic.
      (6) Elbow x-rays to rule out radial head dislocation or fracture.
      (7) MRI or ultrasound to evaluate elbow masses (usually lipoma or ganglia).
   b. **Radial tunnel syndrome**
      (1) Chiefly a pain syndrome (weakness is secondary); sometimes night pain is significant.
      (2) Pain at the lateral elbow; exacerbated by resisted supination with the elbow extended.
      (3) Often seen in a work-related setting with a history of repetitive forceful elbow extension or forearm rotation.

      (4) Must differentiate from lateral epicondylitis: Tenderness is more distal with radial tunnel syndrome.
      (5) "Middle finger test": With elbow and fingers extended, press on the dorsum of the proximal phalanx of the middle finger. The test is positive if this produces pain at the edge of the ECRB in the proximal forearm.
      (6) EMG may not be useful.
      (7) Injection of local anesthetic into the radial tunnel that relieves pain and produces PIN palsy is diagnostic.

4. **Nonoperative treatment**
   a. **PIN syndrome**
      (1) Selected patients may be observed for 2 to 3 months, provided there are no signs of progression. However, excessive delay can lead to permanent muscle weakness.
      (2) Rheumatoid patients may benefit from steroid injection.
      (3) Surgery is indicated if there is no improvement.
   b. **Radial tunnel syndrome**
      (1) Nonoperative care should always be tried first (rest, splinting, NSAIDs).
      (2) No progression to muscle palsy has been documented.

5. **Operative treatment**
   a. **PIN syndrome**
      (1) Indications: Failure of nonoperative treatment, known space-occupying lesion, post-traumatic or associated with open reduction and internal fixation (ORIF) of proximal radius.
      (2) Good or excellent results are obtained in 85% of patients, but may take up to 18 months.
   b. **Radial tunnel syndrome:** Surgery is indicated for failure of conservative treatment.

B. Superficial radial nerve compression (Wartenberg) syndrome

1. **Anatomy and pathophysiology**
   a. Described by Wartenberg in 1932. Sites of compression are as follows.
      (1) External compression (watch bands, handcuffs).
      (2) Overuse/repetitive activity (using a screwdriver, typing, writing) or wrist contusion.
      (3) Scissoring of ECRL and the brachioradialis tendons. May be worsened by nerve tethering in distal scar tissue.
   b. The superficial branch of the radial nerve becomes subcutaneous about 9 cm proximal to the radial styloid. It may pierce the tendon of the brachioradialis.

2. **Diagnosis**
   a. **History and physical examination**
      (1) Pain, numbness, and tingling over the dorsal radial hand, exacerbated by wrist movement, index-thumb pinch, or forceful pronation of the forearm (symptoms within 30–60 seconds).
      (2) May have a false-positive Finkelstein's sign (pain with ulnar deviation of the hand with the thumb grasped in palm).
      (3) Diagnosis can be confirmed by tracing Tinel's sign over the nerve or a diagnostic nerve block.
   b. **Electrodiagnostic studies** are useful.

3. **Nonoperative treatment:** NSAIDs, splinting, local steroid injections, and activity alteration should be tried before operative intervention.

V. Thoracic outlet compression syndrome (TOCS)

A. Epidemiology and risk factors

1. Female gender: Four times more risk than men.
2. Age: Average age is 37.
3. Occupation involving awkward or static arm positioning at or above the shoulder level (e.g., painters, nurses, typists, etc.).
4. Insurance status: Private insurance more than Medicaid.

B. Anatomy and pathophysiology
1. Sites of potential compression
a. Interscalene triangle: Between the anterior and middle scalene and the first rib.
b. Costoclavicular triangle: Between the clavicle and the first rib.
c. Subcoracoid or pectoralis minor space.
d. Cervical ribs: Present in 0.5% of the general population; 50% to 80% are bilateral.

2. Wilbourn's classification: Based on the etiology of symptoms.
a. **Arterial** (1%–2% of cases)
(1) Major subtype is associated with obvious osseous anomalies.
(2) Minor subtype is intermittent compression, usually at the pectoralis minor insertion.
(3) 50% of arterial TOCS will have a fully developed cervical rib.
b. **Venous** (2%–3% of cases)
(1) Paget-Schroetter syndrome: Sudden, effort-induced thrombosis.
(2) May also occur after prolonged malpositioning.
(3) Less acute cases lead to large collateral development.
c. **"True" neurogenic** (rare; only 1 in 1,000,000 patients)
(1) Objective signs of chronic nerve compression: atrophy, weakness (usually C8-T1).
(2) A bony anomaly is always present.
d. **"Disputed" neurogenic** (97% of cases): Wide variety of complaints; no objective findings

C. Diagnosis
1. History and physical examination
a. Chronic pain of insidious onset (shoulder, upper back, neck) with upper extremity paresthesias.
b. Easy fatigability and nighttime pain are common.

2. Provocative tests: Looking for symptom reproduction and/or loss of the radial pulse.
a. **Adson's test (scalene test):** With arm at the side, hyperextend the neck and turn the face toward the affected side and breathe deeply.
b. **Halstead maneuver (costoclavicular test; military brace test):** With the arms at the sides, move the shoulder down and back with the chest out.
c. **Wright's hyperabduction maneuver:** With the arm externally rotated and abducted 180 degrees, inhale deeply (look for symptoms within 1 minute).
d. **Roos' test (stick-up test):** Hold arms abducted and externally rotated; pump hands open and closed quickly. Look for symptoms of rapid fatigue of the involved arm.
e. **Cervical rotation lateral flexion test:** Rotate the head away, then flex toward the affected side. Look for bone blocking lateral flexion or bony asymmetry between the two sides.

3. Other tests
a. Doppler probe: Evaluate flow, bruits, etc.
b. Diagnostic injection of the scalene(s) with lidocaine.
c. Chest and cervical spine x-rays are part of the initial evaluation.
d. Angiography and venography are the gold standards for vascular TOCS.
e. MRI is generally not helpful: 10% false-positive rate and it is not very specific.
f. EMG is recommended for routine workup of "disputed" cases, but is rarely helpful.
g. Psychiatric workup or treatment may be helpful. Some insurers may require one.

D. Nonoperative treatment
1. Should be the first course of therapy for all "disputed" neurogenic TOCS (97% of cases).

2. May be inpatient or outpatient.
3. Four stages
a. Stage I: Identify and treat trigger points, spasm, tendonitis, and bursitis using medication and other therapies.
b. Stage II: Restore normal mobility and posture with stretching, relaxation, and education (typically done concurrently with stage I).
c. Stage III: Strengthen muscle and restore presymptomatic level of function.
d. Stage IV: Establish a home program and return to work.
4. Success rates are 50% to 100% for "disputed" forms of TOCS.

E. Operative treatment
1. Principles of current treatment.
a. Excision or release of any or all anomalous anatomy.
b. Resection of the first rib.
c. Release or excision of the anterior and middle scalene muscles.
d. Neurolysis of the brachial plexus as indicated.

2. Indications
a. Failure of conservative therapy.
b. Intractable pain.
c. Significant neurologic deficit ("true" neurogenic group).
d. Impending or acute vascular catastrophe (vascular TOCS).
e. For "disputed" cases, indications continue to be controversial.

3. Surgical techniques
a. **Transaxillary approach:** Safest for routine decompression, but it is hard to see the posterior first rib, and access to the plexus is incomplete.
b. **Supraclavicular approach:** Better for vascular TOCS operations, but retraction injury to the phrenic and long thoracic nerves is more common.
c. **Combined:** Use both approaches.

4. **Complications** include brachial plexus injury (probably underreported), hemothorax, pneumothorax (up to 62% in reoperative cases), chylothorax, causalgia, and cutaneous nerve dysesthesias (usually resolve within 6 months, occasionally permanent).
5. **Operative management** is successful in 80% of cases (range 25%–100%).

Tendon Transfers

I. Principles
A. **Definition:** The relocation of a tendon from a functioning muscle to replace an injured or nonfunctional muscle-tendon unit.
B. **Concept of a "muscle balance operation":** Tendon transfer (TT) is a redistribution of power units from areas of lesser functional need to areas of greater functional need.
C. **Loss of a single major nerve** (i.e., ulnar/median/radial) is more amenable to TT; if two or three nerves are damaged, severe extremity impairment is inevitable.
D. **Other important points**
1. Joints affected need to have a good passive range of motion; joint contractures need to be either prevented or released.
2. Sensibility need not be perfect.
3. In general, simpler procedures have better results. E.g., do not introduce more than one change of direction in a tendon.

II. Indications
A. **Nerve injury:** The most common indication. May be at the level of the spinal cord or in a major nerve trunk. Injuries to smaller branches are more likely to recover without the need for TTs.
B. **Muscle/tendon destruction:** May be from trauma or disease processes such as rheumatoid arthritis (however, most traumatic injuries do *not* require a TT).

C. **Spastic disorders:** A less common indication, in part because TTs are less beneficial to these patients.

III. **Preoperative planning**

A. **Evaluation**

1. Establish patient goals and needs.
2. Rank priority of the functions desired.
3. Assess expectations, making sure they are realistic.
4. Verify motivation and ability to follow through with rehabilitation.

B. **Timing**

1. **Factors in the recovery of nerve function**
   a. Children do better than adults, who do better than the elderly in recovering nerve function.
   b. Clean, sharp cuts recover better than large and/or contaminated wounds.
   c. Injuries close to the muscle have better functional recovery than those far from the target muscle.
2. **Immediate tendon transfer:** Only done if there is no chance of neurologic recovery (i.e., the muscle is destroyed or a large section of the nerve is missing and repair/regrowth is not feasible).
3. **Delayed tendon transfer:** Usually performed 9 to 12 months following injury to allow for potential regrowth of the nerve. The higher the nerve injury, the longer regrowth will take.

C. **Wound site factors** that must be addressed before a TT

1. The bony skeleton must be stabilized.
2. The wound must be closed.
3. Scars must be soft, or must be excised.
4. Adequate soft tissue must be present to protect the TT.
5. Joint mobility must be maximized for the best result.

D. **Donor tendon/muscle evaluation**

1. **Donor muscle assessment:** Inventory all muscles and rate their power on a scale of 0 to 5. Only donor muscles with power grades of 4 or 5 should be used for tendon transfer.
   a. 0: No active movement.
   b. 1: Can resist gravity.
   c. 2: Can overcome gravity, but too weak for tasks.
   d. 3: Weak but useful power.
   e. 4: Weaker than normal strength.
   f. 5: Normal strength.
2. **Control:** Tendons to be transferred should have independent power (e.g., the FDP tendon slips do not have independent function and therefore are poor donor choices).
3. **Excursion (amplitude)**
   a. Specific excursions.
      (1) Digits: 70 mm.
      (2) Wrist: 30 mm.
   b. Tenodesis can increase excursion by approximately 25 mm.
   c. Need to match the donor being transferred with the amount of excursion needed (e.g., a wrist flexor will not work well as a digit extensor).
4. **Need to match strength** (e.g., the FCU is too strong for use as a motor for the APB). Therapy can improve muscle strength, but not excursion.
5. **Location:** Reroute the donor tendon in as direct a line as possible; do not change direction of tendon more than once.
6. **Synergism:** If possible, use muscles that naturally work together (e.g., wrist extensors and finger flexors); this makes postoperative rehabilitation easier.
7. **Expendability:** Is tendon function worth giving up for the benefit gained at its new location?

E. **Smith and Hastings' planning algorithm**

1. What muscles are functional?

2. What muscles are available for transfer?
3. What transfers/functions are needed?
4. Match what is available with functional requirements.
5. What else needs to be done (arthrodeses, tenodeses, etc.)?
6. Protect transfers postoperatively with splinting.
7. Rehabilitate to protect transfers while maximizing function.

IV. **Tendon transfers for specific nerve palsies**

A. **Radial nerve palsy**

1. **Anatomy:** The radial nerve innervates the triceps, brachioradialis, and ECRL before branching into superficial and deep branches in the forearm. The superficial radial nerve is purely sensory, whereas the deep radial nerve innervates the ECRB, supinator, APL, and all finger extensors. It continues as the posterior interosseous nerve to innervate the dorsal wrist.
2. **Functional deficits**
   a. The radial nerve is mostly motor, and the return of function is often more complete than in median or ulnar nerve injuries.
      (1) Common radial nerve injury patterns include "Saturday night palsy" and humerus fracture palsies.
      (2) Such injuries generally recover without surgery on the nerve.
   b. Uncomplicated nerve repairs in the distal third of the arm should recover some function within 4 to 6 months (1 mm per day). Patients without recovery after 6 months should be considered for TT.
   c. **High radial nerve paralysis:** Wrist, thumb, and digit extension are lost, producing a wrist drop deformity. Loss of wrist extension weakens power grip strength. Over time, patients develop an adaptive functional pattern.
      (1) Patients use wrist flexion to assist with finger extension (i.e., the "tenodesis effect").
      (2) Wrist flexion may be difficult to overcome later with TTs.
      (3) Splints should be worn to force wrist extension and assist with finger extension; splints are cumbersome and often tolerated only at night, but are critical to the eventual outcome.
      (4) Alternatively, some surgeons advocate doing an end-to-side tendon transfer of the PT to the ECRB to facilitate grip strength during nerve recovery (termed an "internal splint" procedure).
   d. **Low radial nerve injury:** Greater radial wrist deviation than higher-level injury due to unopposed ECRL function with loss of other extensors.
3. **Common tendon transfers for radial nerve paralysis** (Table 39-4). Radial nerve palsy TTs require one tendon each for wrist, digit, and thumb extension.

B. **Median nerve palsy**

1. **Nerve function and anatomy**
   a. The median nerve enters the forearm through the pronator teres, and innervates the PT, FCR, PL, and FDS.
   b. The anterior interosseous nerve then branches off and innervates the FPL, PQ, and radial head of FDP.
   c. The main branch of the median nerve continues through the carpal tunnel and gives off sensory and motor branches.
   d. The thenar motor branch innervates the APB, opponens pollicis (OP), and (variably) flexor pollicis brevis (FPB) muscles.
   e. The common digital branches innervate the lumbricals to the long and index fingers.
2. **Functional deficits**
   a. **Low median nerve palsy:** Loss of nerve function at level of the wrist.
      (1) Loss of **thumb opposition** from paralysis of APB, OP, and superficial head of FPB.
      (2) Loss of the lumbricals to the index and middle fingers.

Table 39-4. Tendon transfers for radial nerve paralysis

Desired Function	Tendon Transfer	Comments
Wrist extension (high nerve palsies)	PT to ECRB	PT is the optimal donor for wrist extension. It may not be needed if the injury is below the level of ECRB innervation.
Wrist lateral deviation (low palsies)	ECRL to ECRB or ECU	Minimizes radial wrist deviation.
Digit extension (low and high palsies)	FCR to EDC; FCU to EDC	FCR is favored, because ulnar deviation is preserved. FCU has twice the force of FCR but less excursion. No independent digit extension is allowed with these TTs.
	FDS (MF, RF only) to EDC	For independent motion (Boyes' transfer).
Thumb extension (low and high palsies)	PL to EPL	PL is present in 80% of patients.
	FDS (MF) to EPL	FDS can be used if PL is not present (Boyes' transfer)
	BR to APL	APL tenodesis at the BR insertion at the radial styloid prevents flexion-adduction of the thumb metacarpal and compensatory MCP hyperextension and IP flexion.

APL, abductor pollicis longus; BR, brachioradialis; ECRB/ECRL, extensor carpi radialis brevis/longus; EDC, extensor digitorum communis; EPL, extensor pollicis longus; FCU, flexor carpi ulnaris; FDS, flexor digitorum superficialis; IP, interphalangeal joint; MCP, metacarpophalangeal joint; MF, middle finger; RF, ring finger; PL, palmaris longus; PT, pronator teres; TT, tendon transfer.

b. High median nerve palsy

(1) In addition to the muscle losses listed, the anterior interosseous nerve is affected. Patients lose FPL, PQ, and FDP to index and middle fingers, resulting in loss of thumb and index flexion.

(2) Higher-level injuries will damage FCR and PT function, but these losses do not require tendon transfers.

3. Common tendon transfers for median nerve paralysis (Table 39-5).

C. Ulnar nerve palsy

1. Nerve function and anatomy

a. Enters the forearm between the two heads of the FCU, and innervates the FCU and the ulnar portion of the FDP (ring and little fingers).

b. Continues into the hand via Guyon's canal, and innervates the abductor digiti minimi (ADM), flexor digiti minimi (FDM), and opponens digiti minimi (ODM) muscles (i.e., the hypothenar muscles) as well as the seven interosseous muscles, the adductor pollicis, and the ring and little finger lumbricals. The ulnar nerve may innervate part or all of the FPB.

2. Functional deficits

a. Low ulnar nerve palsy

(1) Paralysis of the AP, the deep head of FPB, all interossei, the hypothenar muscles, and the lumbricals to ring and little fingers.

Table 39-5. Transfers for median nerve paralysis

Desired Function	Tendon Transfer	Comments
Thumb opposition (low and high palsies)	EIP to APB	EIP is usually preferred to avoid using a tendon from the potentially scarred volar area.
	ADM to APB	The Huber procedure. Also used for thumb hypoplasia, combined nerve palsies, and trauma.
Thumb opposition (low palsy)	FDS (RF) to APB	FDS (RF) is routed through a loop of FCU at the wrist to approach the APB at the proper angle.
	PL to APB	The Camitz procedure. Used after long-standing CTS and can be performed at the time of CTR.
Thumb flexion (high palsy)	BR to FPL	Allows thumb IP flexion.
Index flexion (high palsy)	FDP (LF, RF) to FDP (MF, IF)	Tenorrhaphy (not transfer) allows DIP flexion of MF and IF.
	ECRL to FDP (IF)	Provides strength to index flexion.

ADM, abductor digiti minimi; APB/APL, abductor pollicis brevis/longus; BR, brachioradialis; CTR, carpal tunnel release; CTS, carpal tunnel syndrome; DIP, distal interphalangeal joint; ECRL, extensor carpi radialis longus; EIP, extensor indicis proprius; FCU, flexor carpi ulnaris; FDP/FDS, flexor digitorum profundus/superficialis; IF, index finger; IP, interphalangeal joint; LF, little finger; MCP, metacarpophalangeal joint; MF, middle finger; RF, ring finger; PL, palmaris longus.

(2) Clawing of the hand: Specifically, metacarpophalangeal (MP) joint hyperextension and IP flexion in the ring and little fingers due to loss of intrinsic musculature in the setting of intact extrinsic function.

(3) Weak key pinch: Due to denervation of the first dorsal interosseous and AP muscles. To compensate, patients use FPL flexion to stabilize the thumb and EPL to adduct the thumb. Exaggerated thumb IP flexion during key pinch is termed **Froment's sign.**

(4) Little finger ulnar deviation occurs due to unbalanced extensors (EDC, EDM) to that digit **(Wartenberg's sign).**

b. High ulnar nerve palsy

(1) Less clawing than with low ulnar palsies, because the paralysis of FCU and FDP to ring and little fingers decreases the deforming force.

(2) Reconstruction can improve the function of the hand, but normal function usually cannot be restored.

3. Common tendon transfers for ulnar nerve paralysis (Table 39-6).

4. Additionally, static block procedures can be done to prevent MP hyperextension, including MP arthrodesis, MP joint capsulodesis, or bone blocks on the dorsum of the MP head. These procedures can be used alone or in concert with TTs.

V. Tendon transfers for specific diseases

A. Rheumatoid arthritis

1. Rheumatoid patients often rupture extensor tendons due to synovial invasion of the tendon or to attrition from a subluxed ulnar head.

Table 39-6. Transfers for ulnar nerve paralysis

Desired Function	Tendon Transfer	Comments
Thumb, key pinch (low and high palsies)	ECRB to abductor tubercle of the thumb metacarpal	Abductorplasty through the second intermetacarpal space to attach to the abductor tubercle. A PL tendon graft is usually required.
	FDS (RF) to abductor tubercle of the first metacarpal	Abductorplasty. Use only the radial half of the RF FDS tendon. Palmar fascia acts as pulley. No tendon graft is needed.
Clawing (low palsy)	FDS (MF or RF) to LF, RF	Tendons are split, then sutured to lateral bands or to the proximal phalanx.
	FDS "lasso" to LF, RF A2 pulley	FDS (RF or MF) is divided and looped through the A2 pulley, and then sutured to itself, with half going to the LF and half going to the RF.
	EDC or BR to LF, RF LBs	Attached to the lateral bands. Stabilizes finger, but does not aid in power flexion.
	EIP or EDC (LF) to LF, RF LBs	Attached to the lateral bands through the intermetacarpal space.
Clawing (high palsy)	FDP (MF) to FDP (LF, RF)	Side-to-side tenorrhaphy.
Index abduction for key pinch (low and high palsies)	Slip of APL to first dorsal interosseous	Main part of the APL remains attached to thumb; may use tendon graft to augment.
	EIP or EPB to first dorsal interosseous	Alternatives used to restore IF abduction. Many variations, but all attach to the first interosseous.
Power digit flexion (low and high palsy)	ECRL to digits	Tendon grafts are used to extend the ECRL in two to four tails, which go under the TCL to attach to the digital lateral bands or to the A2 pulleys.
Little finger adduction (low palsy)	EDM to LF	Ulnar half of EDM is passed through the metacarpal space to attach to either bone or the A2 pulley.
Wrist flexion (high palsy)	FCR to FCU	Restores FCU function. Some authors feel this is not necessary.

APL, abductor pollicis longus; BR, brachioradialis; ECRB/ECRL, extensor carpi radialis brevis/longus; EDC, extensor digitorum communis; EDM, extensor digiti minimi; EIP, extensor indicis proprius; EPB, extensor pollicis brevis; FCR/FCU, flexor carpi radialis/ulnaris; FDP/FDS, flexor digitorum profundus/superficialis; IF, index finger; LB, lateral band; LF, little finger; MF, middle finger; RF, ring finger; PL, palmaris longus; TCL, tibial collateral ligament.

2. EDM and little finger EDC are often the first affected; this manifests as difficulty extending the little finger at the MP joint.
3. To repair, the distal EDC tendon from the little finger can be attached to the ring EDC. Alternatively, the EIP can be transferred to either the EDM or the little finger EDC.
4. If both the ring and little finger EDC tendons have eroded, then either the EIP or the FDS from the ring or middle fingers can be used.
5. An extensor tenosynovectomy and a Darrach procedure should be done to prevent additional extensor tendon loss.

B. Leprosy

1. The most common deformity is clawing due to ulnar or combined ulnar/median nerve palsies.
2. TTs are used to restore thumb opposition and key pinch activity.
3. ECRL to intrinsic muscle tendon transfers can also be performed, using plantaris tendon graft(s).

C. Poliomyelitis

1. The muscles weakened early in the disease course will often regain function.
2. Wait until strength is stable (no progression of disease *and* no new recovery of function) for 6 months to a year before doing any tendon transfers.

49 Necrotizing Soft Tissue Infections

Andrew P. Trussler

Introduction

The keys to successful management of necrotizing soft tissue infections are early recognition and prompt surgical debridement. These lesions may present with few external manifestations. Diffuse internal inflammation may progress rapidly, with significant underlying deep tissue destruction. A 24-hour delay in diagnosis and treatment may result in a mortality rate of 50%.

General Information

I. History

A. 1883: Fournier described a necrotizing infection of the scrotum (Fournier's gangrene).

B. 1924: Meleney reported streptococcal gangrene associated with bacterial synergism (Meleney's synergistic gangrene).

C. 1952: Wilson described necrotizing fasciitis.

D. 1983: Greenberg reported necrotizing fasciitis with group A streptococci and toxic shock syndrome (TSS).

II. Etiology

A. Monomicrobial infections: Caused by three broad classes of organisms.

1. Bacteria

a. *Streptococcus pyogenes.*

b. *Clostridium perfringens.*

c. Rarely, Gram-negative aerobes such as *Pseudomonas aeruginosa* or Fungi *vibrio vulnificus* may be involved.

2. Fungus (e.g., *Mucor*): Can invade deeply, bypassing fascial planes.

3. Protozoa: Rarely cause necrotizing infections (e.g., *Entamoeba histolytica, Trichinella, Toxocara* spp.). Meleney's synergistic gangrene is thought to be caused by *Entamoeba* infection.

B. Polymicrobial infections: Much more common than monomicrobial necrotizing infections.

1. Bacterial synergism: Allows one organism to potentiate the growth of another.

2. Gram-positive aerobes (*S. pyogenes, Staphylococcus aureus,* or *Enterococcus faecalis*) **plus Gram-negative aerobes** (*Escherichia coli, Pseudomonas* spp., *Clostridium* spp., *Bacteroides* spp., or *Peptostreptococcus*).

III. Risk factors

A. Impaired host defense mechanisms: Extremes of age, immunosuppression (transplant patient or infection with human immunodeficiency virus), extremity lymphedema, or chronic systemic illnesses (cancer, chronic renal failure, alcoholism, diabetes mellitus, and peripheral vascular disease).

B. History of trauma, burns, wound contamination, or foreign body.

IV. Pathogenesis

A. A microaerobic wound environment promotes the growth of bacteria, leading to a local decrease in oxygen, producing a permissive environment for anaerobic bacteria.

B. The presence of proteolytic enzymes enhances the rate and extent of spread of infection.

C. Thrombosis of nutrient blood vessels to the skin and subcutaneous tissues produces more ischemic tissue, creating a vicious cycle.

V. Presentation

A. Necrotizing soft tissue infections are characterized by sudden presentation and rapid progression.

B. The extent of infection is often diffuse, with deeper tissues more affected than superficial ones.

C. Early signs

1. Presence of edema beyond the extent of erythema
2. Crepitus
3. Skin vesicles or bullae
4. Fever and early sepsis
5. Grayish watery drainage ("dishwater pus")
6. Coppery hue of the skin

D. Late signs

1. Cutaneous anesthesia
2. Focal skin gangrene
3. Shock, coagulopathy, and multiorgan failure

Classification

I. Clostridial necrotizing infections

A. Caused by multiple species of *Clostridia*, most commonly *C. perfringens* (80%), *C. novyi* (20%), and *C. septicum*, which are common contaminants of traumatic wounds.

B. A decrease in local oxygen tension results in spore activation.

C. Production of multiple exotoxins: Most common is alpha toxin (lecithinase), which causes cell membrane breakdown.

D. The diagnosis of gas gangrene is made clinically. Necrotizing cellulitis has early local signs with moderate pain and involvement of superficial tissue. Myonecrosis presents with severe pain and involvement of deep tissues.

E. A Gram stain of wound fluid reveals gram-positive rods without inflammatory cells.

II. Nonclostridial necrotizing infections

A. Streptococcal gangrene

1. Caused by hemolytic streptococci.
2. Presents with rapid development of erythema over 24 hours, with progression to blue discolored bullae and then superficial gangrene in 4 to 5 days.

B. Necrotizing fasciitis

1. Typically has a slower onset of symptoms compared with streptococcal gangrene.
2. Bacterial synergy between aerobic and anaerobic bacteria occurs when lytic toxins enhance the spread of anaerobic bacteria.
3. **Two main bacteriologic types**
   - **a.** Type I: Enterobacteriaceae/non-group A β-hemolytic streptococci and anaerobic cocci/*Bacteroides;* more common than type II.
   - **b.** Type II: Group A streptococci and staphylococci.
4. **Meleney's synergistic gangrene:** Occurs postoperatively in surgical wounds, typically either thoracic or abdominal.
5. **Variant causes**
   - **a.** *Vibrio vulnificus:* Puncture wounds exposed to seawater; rapid onset.
   - **b.** Phycomycosis: More insidious; differentiated only by histology.

C. **Necrotizing fasciitis with streptococcal toxic shock syndrome**
1. "Flesh-eating bacteria": Caused by *Streptococcus pyogenes*.
2. Systemic pathogenesis is related to superantigens, M proteins, and induction of monokines (tumor necrosis factor, interleukins 1 and 6).
3. Rapid presentation of pain, edema, fever, shock, and organ failure.
4. Clindamycin impairs M-protein synthesis and exotoxin production.

D. **Idiopathic scrotal gangrene**
1. Fournier's gangrene: Perineal gram-negative synergistic necrotizing cellulitis.
2. Causative organisms: Anaerobic streptococci.
3. Presentation: Sudden onset of fever and rapid development of scrotal gangrene and skin sloughing (24–30 hours).

Management

I. **Early diagnosis**
A. **Primarily based on clinical suspicion**, but may be assisted with laboratory studies that include a complete blood count, serum electrolyte levels, and lactate levels.
B. **Soft tissue x-rays** (soft tissue gas), computed tomographic scan, or magnetic resonance imaging with gadolinium.
C. **Early tissue biopsy** may facilitate early recognition of phycomycoses.

II. **Radical surgical debridement**
A. **Debridement of all necrotic tissues should be performed, with intraoperative quantitative culture** and collection of biopsy specimens. Gram staining of wound fluid should be performed.
B. **Debridement should extend to viable tissue,** with possible extremity amputation in clostridial gangrene, debridement of abdominal wall in Meleney's synergistic postoperative gangrene, and creation of a testicular thigh pouch in Fournier's gangrene.
C. **Postoperative intensive care** is usually required, with invasive monitoring, aggressive resuscitation, immobilization and elevation of involved extremities, and initiation of dressing changes (topical antimicrobial versus moist gauze).
D. **Repeat exploration in 24 to 48 hours** is performed, and remaining infected tissue is excised.

III. **Antibiotic coverage**
A. **Broad coverage** should be used until microbiologic analysis of the wound is available. Penicillin, ampicillin, and beta-lactams are effective for *Clostridia*, enterococci, and *Peptostreptococcus*. Clindamycin is excellent for anaerobes, and gentamicin is effective against most *Enterobacter* and gram-negative species.
B. **Single-agent**, broad-spectrum drug therapy may be initiated.
C. **Amphotericin B** should be started for demonstrated phycomycoses.
D. **Third-generation cephalosporins and doxycycline or fluoroquinolones** should be used for *Vibrio vulnificus* infection.
E. **Human immunoglobulin** should be given to patients with streptococcal TSS.
F. **Antibiotic treatment alone is not enough**. Surgical debridement of all devitalized tissues is required.

IV. **Hyperbaric oxygen therapy**
A. Can be used for clostridial necrotizing infections in conjunction with the previously listed treatments.
B. There is no proven efficacy in nonclostridial infections.

Brown Recluse Spider Bites

I. **Entomology**
A. ***Loxosceles reclusa*** is identified by a violin-shaped mark on the dorsal cephalothorax.

B. The spider measures 1 to 3 cm in size, and is often found indoors or outdoors in debris piles.

II. **Clinical presentation**
A. The bite presents with superficial erythema with surrounding purplish discoloration (6–24 hours).
B. **Progression to full-thickness skin necrosis** often ensues (over >48 hours).
C. **Systemic symptoms** may include fever, myalgia, malaise, and/or gastrointestinal upset (beginning at 12–24 hours).

III. **Pathophysiology**
A. **The spider's venom is cytotoxic,** with protease, hyaluronidase, esterase, and sphingomyelinase components.
B. There is potentiation of the local neutrophil-mediated immune response, with development of dermatonecrosis and systemic lymphokine response.
C. Histologic polymorphonuclear neutrophil perivasculitis with local hemorrhage also occurs.

IV. **Treatment**
A. Correct identification of the lesion can be difficult, and is often delayed.
B. Evaluate for other causes and monitor for systemic symptoms.
C. Initial irrigation, local cold therapy, tetanus prophylaxis, and elevation of the affected extremity are helpful.
D. Closely observe for 72 hours.
E. **Dapsone (a leukocyte inhibitor)** should be initiated orally if a brown recluse spider bite is suspected. Dapsone is continued until the skin lesion resolves.
F. **Surgical debridement with skin grafting** is indicated if medical therapy fails and the lesion is well demarcated.
G. Failure of grafting is high—around 15%.

Pearls

1. Necrotizing fasciitis is a surgical disease.
2. Biopsies of tissue for culture are mandatory, as is a "second look" operation in 24 hours.
3. Hyperbaric oxygen therapy may be helpful in the treatment of clostridial infections.
4. Dapsone can reduce the need for surgical treatment of wounds from brown recluse spider bites.
5. Skin grafting of brown recluse spider bites is associated with high failure rates.