Introduction

Diagnosis is a challenge. Complex anatomy combined with difficult radiographic interpretation requires a high index of suspicion, thorough history including mechanism of injury, precise physical examination, and specific radiographic imaging.

The incidence of carpal fractures reported in the literature varies. In general, the order of frequency is as follows: scaphoid, triquetrum, trapezium, hamate, lunate, pisiform, capitate, and trapezoid.

Mechanism of Injury

- Fall on an outstretched hand with a hyperextension moment and varying degrees of radial or ulnar deviation
- Direct impact or crush
- Indirect ligamentous avulsion

Triquetrum

- Most common carpal fracture second to scaphoid.
- Fracture patterns include dorsal rim chip fractures and triquetral body fractures (Table 9–1).
- Chip fractures may represent an avulsion of the dorsal radiotriquetral ligament. Other theories include compression against the ulnar styloid.

- Body fractures can be divided into medial tuberosity, sagittal, transverse proximal pole, transverse body, palmar radial, and comminuted.
- Medial tuberosity fractures are associated with direct blows to the ulnar border of the wrist.
- Sagittal fractures are associated with axial dislocation and severe crush injury.
- Proximal pole fractures are associated with perilunate/greater arc injury.
- Transverse body fractures are associated with scaphoid injury.

History/Examination

- Dorsal hand and wrist edema usually are present. Wrist flexion tends to be more painful than extension.
- Tenderness just distal to ulnar styloid with hand in radial deviation is noted.

Imaging

- Oblique and lateral radiographic views (Figure 9–1).
- Computed tomography (CT) or bone scan may be necessary to make the diagnosis.

Treatment

- Cast immobilization for 4 to 6 weeks. Tender nonunited fragments may require excision. Body fractures with displacement of greater arc injuries typically are treated with open reduction internal fixation (ORIF).
### Table 9–1: Carpal Fracture Patterns

<table>
<thead>
<tr>
<th>Bone (Normal Right Posteroanterior and Lateral)</th>
<th>Fracture Types</th>
<th>Most Common Treatment</th>
<th>Common Associated Injuries</th>
<th>Treatment Pearls</th>
</tr>
</thead>
</table>
| **Lunate**                                   | 1. Palmar pole  
2. Osteochondral (chip)  
3. Dorsal pole  
4. Sagittal oblique  
5. Coronal split | 1. Closed treatment and casting for 4–6 weeks if minimally displaced or small fragments.  
2. ORIF for intraarticular incongruity or associated instability. | 1. Lunotriquetral or radiolunate ligament tears.  
2. Consider MRI for evaluation of vascularity.  
3. Injury may suggest carpal instability pattern. |
| **Triquetrum**                               | 1. Dorsal rim chip fractures  
2. Body fractures  
a. Medial tuberosity  
b. Sagittal  
c. Transverse proximal pole  
d. Transverse body  
e. Palmar radial  
f. Comminuted | 1. Closed treatment with casting for 4–6 weeks if small chip (Type 1) or minimally displaced.  
2. If large Type 1 or significantly displaced body type may require ORIF. | 1. Dorsal avulsion may represent avulsion from DRC and DIC Ligament.  
2. Triquetrum and lunate may secondarily flex if DIC Ligament torn.  
3. Ulnar impaction/TFCC injury may accompany body fracture. | 1. Stabilization of DRC and DIC ligament may be required if large dorsal avulsion.  
2. Arthroscopy may be necessary to evaluate ulnar/TFCC injury after healing of body fracture. |
| **Trapezium**                                | 1. Vertical transticular  
2. Horizontal  
3. Dorsoradial tuberosity  
4. Anteroomedial ridge  
2. Spanning ex-fix if comminuted.  
3. ORIF vs. K-wires for displaced intraarticular.  
5. Trapezium excision or CMC fusion for late arthrosis. | 1. First MC fractures common.  
2. Ridge fractures may secondarily cause CTS.  
3. Late first CMC arthritis may develop after intraarticular (IA) injury.  
4. FCM/PPL rupture possible if medial irregularity. | 1. Anatomic reduction for intraarticular fractures.  
2. May consider primary fusion for combined trapezium and proximal first MC intraarticular fractures. |
| **Trapezoid**                                | 1. Dorsal rim  
2. May require closed reduction of fracture or second MC and pinning for stabilization.  
3. ORIF rarely necessary. | 1. Unusual as an isolated injury.  
2. Usually associated with second MC dorsal dislocation. | 1. Often requires CT or MRI to diagnose.  
2. Recurrence of posterior subluxation of second MC must be carefully followed.  
3. Fusion of trapezoid-second MC may be necessary for late arthrosis and pain. |

*Continued*
### Table 9–1:

<table>
<thead>
<tr>
<th>BONE (NORMAL RIGHT POSTERIOANTERIOR AND LATERAL)</th>
<th>FRACTURE TYPES</th>
<th>MOST COMMON TREATMENT</th>
<th>COMMON ASSOCIATED INJURIES</th>
<th>TREATMENT PEARLS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Capitate</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>3. Coronal oblique</td>
<td>3. ORIF for irreducible displaced, intraarticular, or proximal pole fractures.</td>
<td>3. Beware associated (but not apparent) scaphoid fracture, lunotriquetral ligament injury, or other perilunate injury.</td>
<td>3. Beware associated (but not apparent) scaphoid fracture, lunotriquetral ligament injury, or other perilunate injury.</td>
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<tr>
<td>4. Parasagittal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hamate</strong></td>
<td></td>
<td></td>
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<tr>
<td>1. Hook</td>
<td>1. Cast immobility for 4–6 weeks for minimally displaced fractures.</td>
<td>1. Irritation and eventual rupture of ulnar finger flexors may occur with displaced hook fracture.</td>
<td>1. Cast immobilization in slight radial deviation will minimize the deforming force of the ulnar finger flexors.</td>
<td></td>
</tr>
<tr>
<td>a. Avulsion (tip)</td>
<td>2. Hamate hook excision if continued pain after period of immobilization.</td>
<td>2. May be associated with fourth or fifth MC dislocation.</td>
<td>2. Hamate hook provides mechanical advantage of ulnar finger flexors.</td>
<td></td>
</tr>
<tr>
<td>b. Waist</td>
<td>3. Rest, equipment adaptation, and immobilization for stress or repetitive injury fracture.</td>
<td>3. May occur with avulsion of FCU.</td>
<td>3. Hook has watershed blood supply at wrist with feeding vessels through tip and base.</td>
<td></td>
</tr>
<tr>
<td>c. Base</td>
<td>4. ORIF of displaced body or intraarticular fractures.</td>
<td>4. Consider hamate hook lateral or carpal tunnel view radiograph for visualization.</td>
<td>4. Consider hamate hook lateral or carpal tunnel view radiograph for visualization.</td>
<td></td>
</tr>
<tr>
<td>2. Body</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>b. Medial tuberosity</td>
<td>2. Hamate hook excision if continued pain after period of immobilization.</td>
<td>2. May be associated with fourth or fifth MC dislocation.</td>
<td>2. Hamate hook provides mechanical advantage of ulnar finger flexors.</td>
<td></td>
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<tr>
<td>c. Sagittal oblique</td>
<td>3. Rest, equipment adaptation, and immobilization for stress or repetitive injury fracture.</td>
<td>3. May occur with avulsion of FCU.</td>
<td>3. Hook has watershed blood supply at wrist with feeding vessels through tip and base.</td>
<td></td>
</tr>
<tr>
<td>d. Dorsal coronal fractures</td>
<td>4. ORIF of displaced body or intraarticular fractures.</td>
<td>4. Consider hamate hook lateral or carpal tunnel view radiograph for visualization.</td>
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<td></td>
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<tr>
<td><strong>Pisiform</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Transverse (common)</td>
<td>1. Immobilization for 2–4 weeks for minimally displaced or comminuted fractures.</td>
<td>1. FCU disruption (partial or complete).</td>
<td>1. Best visualized on lateral radiograph.</td>
<td></td>
</tr>
<tr>
<td>2. Parasagittal</td>
<td>2. Consider ORIF or excision and tendon reconstruction if FCU disrupted.</td>
<td>2. Triquetral or hamate impaction injury related to mechanism.</td>
<td>2. Fibrous union may be well tolerated if FCU in continuity.</td>
<td></td>
</tr>
<tr>
<td>3. Comminuted</td>
<td>3. Excision and tendon reconstruction arthrodesis related to healed (or unhealed) fracture.</td>
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<td></td>
</tr>
<tr>
<td>4. Pisotriquetral impaction</td>
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<td></td>
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</tr>
</tbody>
</table>


CMC, carpometacarpal; CT, computed tomography; CTS, carpal tunnel syndrome; DIC, dorsal intercarpal ligament; DRC, dorsal radiocarpal ligament; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; FPL, flexor pollicis longus; IA, intraarticular, MC, metacarpal; MRI, magnetic resonance imaging; ORIF, open reduction internal fixation; TFC, triangular fibrocartilage complex.
Isolated trapezium fractures are uncommon. Usually occur in association with first metacarpal or distal radius fracture (Figure 9–2).

Five patterns: vertical transarticular, dorsoradial tuberosity, horizontal, anteromedial trapezial ridge, and comminuted2 (see Table 9–1).

Dorsoradial fractures occur as a result from compression between the first metacarpal and radius. Compression of the first web space (i.e., handle bar injury) may create this force.

Trapezial ridge fractures may occur as a result of dorsopalmar crush and flattening of the transverse carpal ligament and resultant avulsion. Look for associated hook of hamate fracture in this mechanism.

History/Examination

- Palpate distal to snuffbox. Thumb flexion and extension may produce pain. Resisted wrist flexion produces pain. Weak or painful pinch. Median nerve compressive symptoms occur occasionally.1

Imaging

- Standard anteroposterior (AP)/lateral radiographs.
- Betts view: Semipronated hand with ulnar palm resting on plate and x-ray beam centered on scaphotrapezoid (STT) joint.
- Carpal tunnel views needed to see trapezial ridge fracture.7
- CT scan may be useful.
Treatment

- Thumb spica immobilization for 4 to 6 weeks. Excision of fragment if painful.
- Carpal tunnel release for median nerve symptoms.

Hamate

- Patterns include hook of hamate and body fractures (see Table 9–1).
- Hook of hamate fractures classically occur with stick handling sports. Direct and forceful impact with the end of a golf club, hockey stick, or repetitive trauma with the handle of a tennis racket.
- Ulnar nerve and artery pass ulnar to hamate; therefore, ulnar nerve symptoms may occur.
- Vascularity of the hamate includes a palmar and dorsal pedicle with intraosseous communication. The watershed occurs at the waist, making waist fractures more susceptible to nonunion and osteonecrosis.
- Body fractures can be divided into proximal pole, medial tuberosity, sagittal oblique, and dorsal coronal. Proximal pole fracture can end up as loose intraarticular bodies. Medial tuberosity fractures can result from a direct blow to the ulnar wrist. Sagittal oblique fractures result from dorsopalmar crush. Coronal fractures result from axial compression, as in a fist fight (in association with fifth metacarpal base subluxation/dislocation).

History/Examination

- Considering all hamate fractures, the hook of hamate fracture is the most common and is discussed in detail here.
- Typical presentation for hook of hamate fractures is a weak or painful grasp and hypothenar tenderness. Pain with resisted little finger flexion. Pain with axial loading of fourth and fifth metacarpal. Ulnar and median nerve symptoms may or may not be present.

Imaging

- Carpal tunnel radiographic views. Oblique views may be helpful.
- CT scan may be appropriate (Figure 9–3).
- Bone scan may be appropriate.

Lunate

- Controversy exists as to whether Kienböck’s disease (osteonecrosis of the lunate) should be included in the total incidence of lunate fractures. Regardless, acute trauma can result in isolated lunate fractures.
- Five patterns: frontal palmar pole, proximal osteochondral fractures, frontal dorsal pole, sagittal oblique, and coronal split (see Table 9–1).
- Dorsal chip fractures of the lunate may exist but often are confused with dorsal chip fractures of triquetrum by plain radiographs.
- Transverse fractures are associated with Kienböck’s disease.

History/Examination

- Tenderness just distal/ulnar to Lister’s tubercle

Imaging

- AP/lateral views

Treatment

- For nondisplaced hook of hamate fractures, we prefer using an ulnar gutter cast extending beyond the metacarpophalangeal (MP) joints to the ring and little fingers for 3 weeks. At 3 weeks, the MP joints are mobilized, and the wrist is immobilized for an additional 2 to 3 weeks in a splint or cast. Displaced fractures or associated instability patterns may warrant operative reduction and stabilization procedures.

- Excision may be indicated for a painful hook fragment but is not without pitfalls. Flexor power of the little finger may be decreased because the hook acts as a moment arm, and an excision may result in shortening of the moment arm and decreased power.

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History/Examination

- Tenderness just distal/ulnar to Lister’s tubercle

Imaging

- AP/lateral views

Treatment

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Pisiform

- Functions as a sesamoid bone in the flexor carpi ulnaris tendon. Pisiform fractures may disrupt the continuity of this tendon.
- Forms the medial border of Guyon canal.
- Four patterns: transverse, parasagittal, comminuted, and impaction (see Table 9–1).
- Transverse fractures are the most common, resulting from a direct blow or sudden flexor carpi ulnaris contraction against a fixed pisiform, as in a fall on the palm of the hand.

History/Examination

- Tenderness to palpation at the hypothenar eminence. Painful little finger flexion.

Imaging

- AP/lateral views (Figure 9–4).
- Oblique and carpal tunnel views commonly required.
- CT or bone scan may be necessary.

Treatment

- Cast immobilization for 4 to 6 weeks. Psotriquetral incongruity is a complication that can lead to degenerative arthritis and may warrant pisiform excision.

A painful pisiform fragment may warrant surgical excision. Excision usually does not compromise the strength of wrist flexion.1

Capitate

- Isolated fractures are rare. Capitate fractures usually occur in the setting of carpal instability. They may occur with scaphoid fractures in what is known as scaphocapitate fracture syndrome, a variant of a perilunar dislocation.
- The proposed mechanism for this syndrome is fall on a dorsiflexed, radially deviated wrist. The scaphoid is fractured, with concomitant transverse capitate fracture.
- Four patterns: transverse proximal pole, transverse body or waist fractures, coronal oblique (verticofrontal), and parasagittal (see Table 9–1).
- Transverse is the most common and may end up 180 degrees rotated, with the fracture plane pointed proximally leading to nonunion and osteonecrosis.
- Cyclists are prone to fractures of the dorsal body.

History/Examination

- Tenderness proximal to base of third metacarpal
- Decreased grip strength

Imaging

- AP/lateral views.
- Transverse CT may be useful.

Trapezoid

- Lies in a very guarded position. Isolated fractures are extremely rare. Look for associated second metacarpal/carpometacarpal joint injury. Fracture patterns include dorsal rim and body fractures (see Table 9–1).

Treatment

- Early diagnosis is key. The general goal is to maintain normal anatomic alignment and prevent arthrosis.
- Early immobilization with splint/cast is standard care, usually for 4 to 6 weeks. Reduction rarely is necessary.
- Be aware of fracture patterns that require operative reduction and fixation to prevent nonunion or osteonecrosis.2–4,9

References

A general reference on carpal fractures excluding the scaphoid.

A general review on several carpal fractures and their presentation and treatment.

Figure 9–4: Anteroposterior radiograph of pisiform fracture.
   A general reference on carpal fractures, excluding the scaphoid, from a textbook devoted exclusively to the wrist. Excellent detail and illustrations for select fractures.

   Fractures of the triquetrum are discussed, emphasizing their common misdiagnosis as lunate injuries and their frequently subtle radiographic nature.

   A general reference on carpal fractures excluding the scaphoid.

   A large series of a relatively common carpal bone fracture. A mechanism of injury is proposed, and management is discussed.

   A small series is discussed. The fracture is difficult to demonstrate on standard radiographs. Oblique radiographs and the required technique are presented.

   A relatively unusual fracture, a particular mechanism, and treatment are discussed.

   Fractures of the hook, body, and dorsal hamate are discussed and treatment options outlined.

    Discusses treatment with anatomic reduction by closed or open means. A high union rate is noted despite relative avascularity (as opposed to the scaphoid).
Introduction

- Understanding of the wrist and its disorders often is a complex and lifelong pursuit secondary to its complex anatomic and biomechanical intricacies, its abundant pathology, and the widely varied approaches to treatment of its disorders.
- The seminal event in the history of carpal instability occurred in 1972, when Linscheid et al. reported their experience with carpal instability and introduced the terms dorsal intercalated segmental instability (DISI) and volar intercalated segmental instability (VISI).
- Between 1972 and 1994, further refinement of the definitions of carpal instability and of a variety of types of instability were reported and defined, including midcarpal instability, nondissociative instability, and axial carpal instability.
- Despite the explosion of interest in carpal instability and the research efforts into increasing our understanding of these instabilities, many significant questions remain unanswered.

Anatomy

Osseous Anatomy of the Wrist

- The wrist is the link between the forearm and the hand.
- Fifteen bones, excluding sesamoid and supranumerary bones
  - Distal radius and ulna
  - Two carpal rows
    - Proximal carpal row: scaphoid, lunate, triquetrum, pisiform
    - Pisiform considered by many a sesamoid bone but provides an important lever arm for the flexor carpi ulnaris tendon
    - Distal carpal row: trapezium, trapezoid, capitate, hamate
  - Base of the five metacarpals
- Joints
  - Radiocarpal joint: two parts
    - Distal articular surface of the radius and the triangular fibrocartilage complex
    - Distal articular surface of the radius has two separate articular facets: scaphoid and lunate facet
  - Proximal carpal row: convex articular facet of the proximal carpal row
- Midcarpal joint
  - Combination of three types of articulations
    - Lateral: scaphoid trapezium trapezoid (STT) articular
    - Central: scapholunate (SL) and capitate
• Medial: hamate-triquetrum articulation, helicoid shaped
  □ Carpometacarpal joint
  □ Index, middle carpometacarpal joint highly interlocked with little motion
  □ Ring and small carpometacarpal less restrained, greater degree of motion
  □ Thumb carpometacarpal, saddle joint, most unconstrained, greatest degree of motion

Ligamentous
• All ligaments of the wrist are intracapsular, except for three ligaments30–34: transverse carpal (flexor retinaculum), pisohamate, and pisometacarpal.
• All ligaments are contained within capsular sheaths of loose connective tissue and fat.30–34
• Difficult to visualize individual ligaments when approaching the carpal joints surgically
• Within the joint themselves, the ligaments can be viewed as distinct entities (i.e., during arthroscopy of the wrist or when visualizing the volar ligaments via a dorsal approach between the carpal bones)
• Two general categories of ligaments:
  □ Intrinsic: have their origin and insertions within the carpus proper (Figure 10–1).
    □ Large area of insertion onto cartilage rather than bone and much less elastic fibers compared to extrinsic ligaments
    □ Tend to avulse from insertion or origin rather than rupture
  □ Proximal carpal row intrinsic ligaments: scapholunate interosseous ligament (SLIL) and lunotriquetral interosseous ligament (LTIL)
• Distal carpal row ligaments: trapezium trapezoid, trapezoid capitate, and capitolunate
• Dorsal intercarpal ligament: scaphotriquetral ligament
• Palmar intercarpal ligaments: STT and triquetrohamate-capitate
• Extrinsic: typically connect forearm bones to the carpal bones (see Figures 1-5 and 1-6).
  □ Stiffer with lower ultimate yield compared to intrinsic ligaments
  □ Tend to rupture midsubstance rather than avulse from origin or insertion
  □ Volar extrinsic ligaments: mirror configuration of two V-shaped ligamentous bands and include radioscaphocapitate, long radiolunate, short radiolunate, ulnolunate, ulnolunocapitate, and ulnotriquetralcapitate
  □ Dorsal extrinsic ligaments: dorsal radiotriquetral

Biomechanics of the Wrist

History
• Most authors agree that the SL ligament is the most important factor in the spatial coherence of the carpus.35–38

Mechanics
• The distal carpal row has very little interosseous motion and can be thought of biomechanically as one unit in conjunction with the second and third metacarpals.33,36
• The proximal carpal row has no direct tendinous attachments and is described as an intercalated segment.

Figure 10–1:
Intrinsic ligaments of the wrist are ligaments that both originate and insert among the carpal bones. A, Dorsal view of the wrist demonstrates the scapholunate (SL), lunotriquetral (LT), trapezium trapezoid (TT), capitotrapezoid (CT), and capitolunate (CH) intrinsic ligaments. B, Volar view of the wrist illustrates the volar aspect of the same ligaments and the scaphoid trapezium trapezoid (STT), scaphocapitate (SC), triquetrocapitate (TC), and triquetrohamate (TH) ligaments. (Copyright Mayo Clinic. Reproduced with permission of the Mayo Foundation.)
Its motion is dictated by the push and pull of the flexor and extensor tendons inserting onto the metacarpals.

During wrist flexion the distal carpal row flexes and deviates ulnarily. With ulnar deviation the distal carpal row flexes, moves ulnarily, and pronates.

With extension the distal carpal row extends and moves radially. With radial deviation the distal carpal row extends deviates radially and supinates.39

During flexion the proximal carpal row flexes with the distal carpal row. The proximal row extends with the distal carpal row during wrist extension.

Unlike the distal carpal row, the proximal carpal row does not function as a single unit during wrist motion. Motion between the scaphoid, lunate, and triquetrum differs significantly.36–38,40–45

In radial and ulnar deviation the motion of the proximal carpal row is the inverse of the distal carpal row (Figure 10–2).

In radial deviation the distal carpal row moves radially, extends, and supinates, while the proximal carpal row flexes and translates ulnarily. This action results because the scaphoid must flex to make room for the distal carpal row.

In ulnar deviation the distal carpal moves ulnarily, flexes, and pronates while the proximal carpal row extends and translates radially.43,46,47

Proximal Row Mechanics

The scaphoid has a tendency to flex because of the push through the trapezium and capitate. The triquetrum tends to extend because of its helicoid articulation with the hamate.1

Because of the lunate's ligamentous attachments to the scaphoid and triquetrum through the SLIL and the LTIL, the lunate tends to remain balanced and in a neutral position on lateral imaging.

During radial deviation, as the scaphoid is pushed into flexion by the distal carpal row, the scaphoid pulls the lunate and triquetrum into flexion through the linkage created through the SLIL and LTIL.

In contrast, during ulnar deviation the hamate is pushed into the articular surface of the triquetrum, creating an extension force on the triquetrum that pulls the rest of the proximal carpal row into extension.48

Several theories on the mechanics of the carpus have arisen. One theory is the role of the scaphoid in linking the proximal and distal rows. This theory maintains that the scaphoid provides a cross link across the rows preventing carpal collapse.49 Another theory, called the oval ring theory, describes a link between the two rows at the radial and ulnar aspects of the wrist (Figure 10–3).

Axial Force Distribution

At the radiocarpal level, approximately 80% of the axial joint compressive force is directed through the scaphoid and lunate into the distal radius; the remaining 20% is directed through the ulnocarpal joint.50

When forces are measured directly in the radiocarpal joint, 60% of axial force is directed through the scaphoid fossa and 40% is directed through the lunate fossa.51

Biomechanical Properties of the Wrist Ligaments

Radial Wrist and Scaphoid Stabilizers

SLIL is as the primary stabilizer of the SL joint.
• SLIL has a failure force of 300 N.52
• SLIL is composed of three distinct portions53 (Figure 10–4):
  ❍ Proximal or membranous portion
  ❍ Dorsal portion: the strongest portion that prevents translation
  ❍ Palmar portion: acts as a rotational constraint
• Distal scaphoid stabilizers include the scaphotrapezial interosseous ligaments (STIL).54
• Radioscapholunate ligament (ligament of T estut) is a neurovascular structure and provides little mechanical stability.
• Palmer stabilizers: radioscaphocapitate ligament (RSC), long radiolunate ligament, and short radiolunate ligament (SRL) have properties similar to other ligaments throughout the body, with a failure force of 100 to 200 N.52,55 These ligaments all are thought to be a secondary stabilizer of scaphoid.

Carpal Instability

Definition of Carpal Instability

• Instability initially was considered analogous to malalignment, which often referred to a static alteration in the normal alignment of the wrist as seen on radiographs. However, the concept of the asymptomatic wrist that is grossly malaligned (as in congenitally
hyperlax wrists) or symptomatic wrists that had normal alignment) confused the definition of “instability.”

- Based on the collaboration of many wrist surgeons and investigators, the definition of instability of the wrist consists of not only abnormal transfers of load across the wrist (dyskinetics) but also abnormal motion (dyskinematic) within the wrist.

Classification of Carpal Instability

- Carpal instability is extremely difficult to classify and many classification schemes exist, each with advantages and disadvantages.
- Classification scheme should be a tool to assist the surgeon in understanding the pathoanatomy and guiding treatment.
- One such guide is that developed by Larsen et al. that takes into account six key factors in guiding treatment.56,57

Box 10–1  Classification of Carpal Instability

- **Chronicity**
  - Acute: within a week of injury
  - Subacute: 1 to 6 weeks after injury
  - Chronic: more than 6 weeks after injury
- **Constancy**
  - Predynamic: no malalignment, only sporadic symptomatic dysfunction
  - Dynamic: malalignment under stress
  - Static: permanent alteration of alignment
- **Etiology**
  - Traumatic: especially if diagnosed acutely and treated, outcome better than if performed late
  - Nontraumatic: chronic conditions, such as rheumatoid arthritis, that result in carpal instabilities had significantly different treatment than traumatic conditions
- **Location**
  - Proximal carpal row
  - Distal carpal row
  - Multilevel
- **Direction**
  - Dorsal intercalated segmental instability (DISI)
  - Volar intercalated segmental instability (VISI)
  - Ulnar translocation
  - Dorsal translocation
- **Pattern**
  - Carpal instability dissociative (CID)
    - Dissociation within row
    - Scapholunate, lunotriquetral
  - Carpal instability nondissociative (CIND)
    - Instability between carpal rows
    - Midcarpal instability, capitohamate instability, ulnar translocation
  - Carpal instability complex (CIC)
    - Feature of CID and CIND
    - Perilunar injuries, axial carpal dislocations
  - Carpal instability adaptive (CIA)
    - Extrinsic to wrist
    - Dorsal malunion of the distal radius that causes CIND

- **Chronicity**
  - Acute: within a week of injury
  - Subacute: 1 to 6 weeks after injury
  - Chronic: more than 6 weeks after injury
- **Constancy**: described by Watson et al.58 to grade the severity of instability
  - Predynamic instabilities: no malalignment, only sporadic symptomatic dysfunction
  - Dynamic instabilities: malalignment demonstrated under stress
  - Static instabilities: permanent alteration in carpal alignment
- **Etiology**
  - Traumatic: especially if diagnosed acutely and treated, outcome better than if performed late
  - Nontraumatic: chronic conditions, such as rheumatoid arthritis, that result in carpal instabilities had significantly different treatment than traumatic conditions
- **Location**
  - Proximal carpal row
  - Distal carpal row
  - Multilevel
- **Direction**
  - Dorsal intercalated segmental instability (DISI)
  - Volar intercalated segmental instability (VISI)
  - Ulnar translocation
  - Dorsal translocation
- **Pattern**
  - Carpal instability dissociative (CID)
    - Dissociation within row
    - Scapholunate, lunotriquetral
  - Carpal instability nondissociative (CIND)
    - Instability between carpal rows
    - Midcarpal instability, capitohamate instability, ulnar translocation
  - Carpal instability complex (CIC)
    - Feature of CID and CIND
    - Perilunar injuries, axial carpal dislocations
  - Carpal instability adaptive (CIA)
    - Extrinsic to wrist
    - Dorsal malunion of the distal radius that causes CIND
Pathomechanics

Direct Mechanisms of Injury

- Force directly applied to wrist causing malalignment and derangement of the wrist.
- Common examples of a direct mechanism:
  - Wringer or crush injury to the wrist that results in an axial carpal dislocation
  - Blast injuries
  - Forceful impact, for example, from a hammer, ball, or other object to the wrist

Indirect Mechanisms of Injury

- Deforming force initially applied at a distance from the wrist resulting in injury to the wrist secondary to the tensile forces applied to the ligaments and compressive forces applied to the articular surfaces.
- Majority of wrist injuries occur secondary to indirect mechanisms and a multitude of combinations of positions of the wrist.
- To understand the injury patterns, Mayfield, Johnson, and Kilcoyne performed cadaveric studies to ascertain the sequence of wrist ligament injuries and progression of ligament injury about the wrist (Figure 10–11). In their model, they studied wrists that sustained a hyperextension/volar radial wrist load injury and
Carpal instability dissociative results when there is dissociation between elements of a single carpal row. Within the proximal carpal row, there can be a scapholunate or lunotriquetral ligament dissociation. Additionally, fractures can result in a dissociation within a row, that is, scaphoid fracture (A). Within the distal carpal row, any of the interosseous ligaments can be dissociated, resulting in CID of the distal carpal row (B).

Figure 10–8:  
Carpal instability nondissociative results when there is abnormal motion between rows of the carpus or the proximal carpal row and the distal radius. The elements within each row are normal, but the articulation between rows and/or the distal radius is abnormal.

Figure 10–9:  
When there is a combination of both dissociative (dotted lines) and nondissociative (solid lines) elements, the instability pattern is called carpal instability complex. This pattern is best illustrated by perilunate dislocations or axial carpal dislocations of the wrist.
described the classic four stages of progressive perilunar instability of the wrist.
• Stage I: SL dissociation or scaphoid fracture
• Stage II: capitolutunate dislocation
• Stage III: LT dissociation or triquetral fracture
• Stage IV: lunate dislocation
• Reverse perilunar instability also has been described and suggested by several authors as a mechanism of isolated LT ligament injury.26,63,64
• Three stages of reverse perilunar instability (Figure 10–12)63
  ❍ Stage I: LT dissociation
  ❍ Stage II: capitolutunate dislocation
  ❍ Stage III: SL dissociation

Diagnosis

History

• Patients presenting with wrist instability have a wide range of complaints, symptoms, and disabilities. It is essential to differentiate the mechanisms of injury, chronicity, location of pain, work-related issues, and quality of pain. As such, history is an important part of the diagnosis of wrist instability and should not be overlooked.

Examination of the Wrist

• Examine the normal wrist first to serve as a baseline of normality for the patient.
• Assess overall ligamentous laxity (thumb laxity, elbow laxity, shoulder laxity).

Figure 10–10:
Lateral radiograph of a malunited distal radius fracture with carpal instability adaptive. The patient reported pain with motion at the radiocarpal joint secondary to the carpal instability that developed at the distal radius and proximal carpal row.

Figure 10–11:
Progressive perilunar instability of the wrist was defined and presented by Mayfield, Johnson, and Kilcoyne.62 A, Stages of progressive perilunar instability of the wrist. The description of each stage is elaborated in text. B, When injury occurs in this manner, a pure ligamentous injury can occur (lesser arc injury) or injury that results in a fracture-dislocation pattern (greater arc injury). (Copyright Mayo Clinic. Reproduced with permission of the Mayo Foundation.)
Note any crepitation, clicks, clunks, or abnormal motion and determine if pain can be elicited similar to the pain causing the complaint.

Strength testing.

Pearl: Grip strength and pinch strength can elicit dynamic instabilities and serve as a baseline for diagnostic anesthetic injections.

Diagnostic injections into the radiocarpal, midcarpal or distal radioulnar spaces of the wrist that are associated with relief of pain and improvement of grip strength correlate highly with potential carpal pathology.

Specific examination findings.

A multitude of special tests elicit specific ligament injuries. These tests are detailed in their respective ligament injury section.

Plain Radiographs and Fluoroscopy

Posteroanterior (PA), lateral, and oblique films are mandatory for all suspected wrist injuries. Initial evaluation includes assessment of all carpal bones for fractures. The following measurements can be used to assess ligamentous stability:

- Gilula’s lines
  - Gilula described three parallel arcs observed on PA radiographs: one arc corresponds to the proximal articular surface of the proximal row, the second arc corresponds to the distal articular surface of the proximal row, and the third arc represents the proximal articular surface of the distal carpal row (Figure 10–13). Disruption of one of these arcs suggests a carpal fracture or ligamentous injury.
  - Carpal height ratio
    - This ratio is calculated by dividing the carpal height by the length of the third metacarpal (Figure 10–14). The normal ratio is $0.54 \pm 0.03$.
    - In disease processes such as SL dissociation, scapholunate advanced collapse (SLAC) wrist, and Kienböck’s disease, collapse of the midcarpal joint decreases this ratio.
  - Intercarpal angles
    - Significant deviation from normal values can indicate a disruption of the SLIL or LTIL (Figures 10–15 and 10–16).
    - SL angle
      - Mean 45 degrees.
      - Less than 30 degrees or greater than 60 degrees is considered abnormal (see Figure 10–15).
    - Radiolunate angle
      - Mean zero degrees.
      - Greater than 15 degrees dorsal or palmar is abnormal.
      - Greater than 15 degrees palmar suggests VISI deformity.
● Greater than 15 degrees dorsal suggests DISI deformity (see Figure 10–15).
  ▶ Capitolunate angle
  ▷ Mean zero.
  ▷ Range 30 degrees dorsal to 30 degrees palmar.
● Intercarpal distance
  ▶ Increased distance or diastasis between the scaphoid and lunate or lunate and triquetrum may indicate SLIL or LTIL injury.
  ▷ Greater than 2 mm between scaphoid and lunate is considered abnormal (see Figure 10–5A).

- *Pearl:* Early ligamentous injuries may produce no abnormalities on plain radiographs. If the mechanism and physical examination suggest ligamentous injury, proceed with further studies.

**Computed Tomography**
- Computed tomography (CT) provides information about the bony anatomy and may aid in identifying small avulsion fractures.

**Bone Scintigraphy**
- Technetium Tc 99m bone scans identify varying degrees of bone remodeling and accesses blood flow within the carpus.
- These studies are most helpful in identifying occult fractures, although they also identify areas of increased blood flow, as in cases of arthritis.
- Studies are sensitive but not specific.

**Arthrography**
- Historically the gold standard for diagnosis of ligamentous injuries.
Contrast medium can be injected into the midcarpal, radiocarpal, and radioulnar joints. Dye flows between any of the compartments indicates a tear.

- Unless real-time fluoroscopy is used to visualize the origin of the dye leak, arthrography lacks specificity.
  - Attritional changes seen with advancing age may lead to spurious findings.
  - Arthrography mostly has been replaced by diagnostic arthroscopy.

### Magnetic Resonance Imaging

- Magnetic resonance imaging (MRI) can provide diagnostic information about the intrinsic and extrinsic ligaments and carpal bones.
- Sensitivity for identifying interosseous ligament injuries has not been clearly established.

### Carpal Instability Dissociative

- When carpal instability is caused by dissociation between bones of the same carpal row, the instability is termed dissociative or carpal instability dissociative (CID). Distal carpal row CID is very rare and has been reported as case reports. Proximal CIDs are much more common and are the focus of this section.

### Scapholunate Ligament Injuries

#### Introduction

- SL instability is the most common form of carpal instability.
- Disruption in the SL relationship can lead to
  - Unopposed extension forces on the lunate imparted by the triquetrum
    - Leading to a DISI deformity.
  - Abnormal scaphoid motion and dorsal subluxation of the scaphoid from the radial fossa during wrist flexion
    - Leading to eventual wrist arthritis.
  - Migration of the capitate proximally between the scaphoid and capitate
    - Leading to stage III SLAC arthritis.

#### Diagnosis

**Physical Examination**

- Patients may have pain dorsally over the SLIL.
- The SLIL can be palpated by flexing the wrist and pressing 1 cm distal to Lister tubercle.
- Positive scaphoid shift test or Watson maneuver (Figures 10–17 and 10–18).
- The wrist is moved from ulnar to radial deviation, with the examiner's thumb pressing against the scaphoid tubercle. Patients with partial tears have increased pain dorsally over SL articulation. In complete tears an audible clunk may be heard as the scaphoid is actively subluxed with dorsal pressure and spontaneously reduces into the radial fossa when the thumb is removed.
- The scaphoid shift test may give a false-positive result in up to one third of individuals because of ligamentous laxity without injury. Therefore, remember to always check both sides.

#### Imaging Studies

**Radiographs**

- PA and lateral views may show
  - Increased SL angle
    - 45 degrees is normal; greater than 60 degrees is considered abnormal (see Figure 10–5B).
  - Diastasis between the scaphoid and lunate
    - Greater than 2 mm is abnormal (see Figure 10–5A).
  - “Ring sign”
    - As the scaphoid flexes, its distal pole appears as a ring in PA radiographs (see Figure 10–5A).
  - Increased radiolunate angle
    - Greater than 15 degrees dorsal indicates a DISI deformity.
  - Disruption of Gilula lines
    - With advanced carpal instability the capitate migrates into the proximal carpal row, causing disruption of Gilula lines and a change in the carpal height ratio.
● "Clenched fist" views
  ◆ May show early SLIL changes (dynamic instability) with widening of the SL interval or increased SL angle as the capitate is driven down into the SL interspace.
  ◆ Remember that partial and even complete division of the SLIL does not always produce an abnormality on plain films because of the substantial numbers of secondary stabilizers of the scaphoid in addition to the SLIL.73

Arthrography
● May show communication between the midcarpal and radiocarpal joint, with a dye leak seen at the SLIL indicating a tear.

Computed Tomography
● Not very accurate for assessing ligamentous injuries.

Magnetic Resonance Imaging
● Can often identify SLIL injuries but is not 100% sensitive.

Arthroscopy
● Now the gold standard for diagnosis of “dynamic” instability patterns. Allows for direct inspection of SLIL and evaluation of supporting extrinsic ligaments (Figure 10–19).
  ● Arthroscopic instability is graded by the Geissler classification74:
    ◆ Grade I: attenuation or hemorrhage of the interosseous ligament as seen from the radiocarpal space. No incongruity of carpal alignment in the midcarpal space.
    ◆ Grade II: attenuation or hemorrhage of interosseous ligament as seen from the radiocarpal space. Incongruity or step-off of carpal space. There may be slight gap (less than width of probe) between carpal bones.
    ◆ Grade III: incongruency or step-off of carpal alignment as seen from both the radiocarpal and midcarpal spaces. Probe can be passed through gap between carpal bones.
    ◆ Grade IV: incongruency or step-off of carpal alignment as seen from both radiocarpal and midcarpal spaces. There is gross instability with manipulation. A 2.7-mm arthroscope can be passed through gap between carpal bones.
    ◆ Midcarpal arthroscopy is the key to assessing the stability of the SL joint. From the midcarpal perspective, the normal SL joint is smooth without a step-off or diastasis.

Stages of Scapholunate Instability
● It is important to distinguish between dynamic and static SLIL instability. SLIL is a spectrum of disease beginning with partial volar tears to total disruption of the SLIL with adaptive changes throughout the carpus.72
  ● Occult or predynamic instability58,75

[Images and figures are referenced as follows: Figure 10–18: In the normal wrist, the intact scapholunate interosseous ligament (SLIL) maintains the scaphoid in alignment with the lunate despite the dorsal pressure. When the SLIL ligament has been disrupted, the scaphoid is no longer stabilized to the lunate and the proximal poles displace dorsally to abut the dorsal rim of the radius, producing an audible clunk. (From Fractures and dislocation of the carpus. In Trumble TE, editor: Principles of hand surgery and therapy. Philadelphia, 2000, WB Saunders.)

Figure 10–19: Midcarpal arthroscopy shows evidence of a Geissler type III pattern, indicating scapholunate instability. The probe can be inserted between the scaphoid and lunate and rotated freely.
Partial tear or attenuation of SLIL
- Radiographs normal
- Possible abnormalities with fluoroscopy
- Dynamic
  - Partial or complete tear of SLIL
  - Stress radiographs abnormal
  - Arthroscopy abnormal (Geissler II or III)
- Static
  - Complete SLIL tear with attenuation or attrition of supporting wrist ligaments
  - Radiographs positive for scaphoid changes.
    - SL gap greater than 3 mm, SL angle greater than 60 degrees
    - Arthroscopic findings reveal a Geissler type IV pattern
  - With time the lunate extends because of unopposed extension force of the intact LTIL. The lunate becomes fixed in dorsiflexion, visualized as a DISI deformity on lateral x-ray films, (radiolunate angle >15 degrees).
- SLAC
  - With longstanding abnormal positioning of the carpal bones, arthritic changes occur. Arthritic changes are first seen at the scaphoid and move to the midcarpal joint in a standard progression (Figure 10–20).
    - Stage 1: arthritis noted at radial styloid
    - Stage 2: arthritis noted at radiocarpal joint
    - Stage 3: arthritis noted at capitolunate interface

**Treatment**

**Acute Injuries**
- Cast immobilization
  - Few long term studies to justify effectiveness in cases of confirmed SLIL rupture.
- Open repair.
  - Technique: The carpus is approached through a dorsal midline incision. Retinacular flaps are elevated from the third dorsal compartment to the second compartment radially and from the third dorsal compartment to the fifth ulnarily. A ligament sparing capsulotomy is made as described by Berger, Bishop, and Bettinger (see Figure 10–39B). This capsulotomy is lined with the fibers of the dorsal intercarpal and dorsal radiotriquetral ligaments. The carpus then is evaluated. The SLIL usually is torn off the scaphoid. The SL relationship is reestablished by placing joysticks into the lunate and scaphoid. The SLIL then is repaired with the aid of drill holes through the scaphoid or with bone anchors. The sutures are passed but not tied until internal fixation has stabilized and reduced the scaphoid to the lunate. The SL interface is reduced and the bones are held with two to three Kirschner (K)-wires or a lag screw. The midcarpal joint also can be pinned for greater stabilization. K-wires are left for 8 to 10 weeks, and patients are protected from full loading for an additional 4 to 6 weeks.

**Chronic Injuries (Dynamic or Static)**
- Open repair
  - Open repair of the SLIL can be considered in cases of chronic injury if
    - There is satisfactory ligament remaining for repair.
    - The scaphoid and lunate remain easily reducible.
    - There is no degenerative changes within the carpus.
- Soft tissue procedures (Best for dynamic instability)
  - Dorsal capsulodesis: Procedures that utilize a portion of the dorsal wrist capsule to tether the scaphoid and prevent it from subluxing from the radial fossa.
    - Blatt: A 1-cm broad flap of wrist capsule is elevated off the ulnar side of the carpal incision. The flap is released from its distal insertion. After derotation of the scaphoid and reduction of the lunate, the capsular flap is attached to the dorsal distal surface of the scaphoid through a drill hole and pullout stitch or with the use of a suture anchor.

Figure 10–20:
Stages of scapholunate advanced collapse. With advancing carpal collapse, the capitate may migrate proximally, resulting in midcarpal arthritis and disruption of the Gilula lines. (Copyright Mayo Clinic. Reproduced with permission of the Mayo Foundation.)
cast. Full loading of the wrist is delayed for up to 6 months (Figure 10–21).

- Mayo: The Mayo capsulodesis begins with a ligament-sparing capsulotomy. The proximal strip of the dorsal intercarpal ligament is rotated and attached to the lunate. This capsulodesis avoids crossing the radiocarpal joint and reestablishes the SL relationship. SL repair is performed using previously described techniques (Figure 10–22).
- Szabo: This technique advances the scaphotrapezial origin of the dorsal intercarpal ligament onto the radial aspect of the scaphoid in order to control.

Figure 10–21:
Blatt capsulodesis uses a proximally based flap of wrist capsule that is inserted into the distal pole of the scaphoid. This procedure restrains abnormal scaphoid flexion.

Figure 10–22:
Mayo capsulodesis uses the proximal portion of the dorsal intercarpal ligament to tether scaphoid and limit palmar rotation. (Copyright Mayo Clinic. Reproduced with permission of the Mayo Foundation.)

Figure 10–23:
Advancing the dorsal intercarpal ligament (DIC) along the radial surface of the scaphoid (S) controls palmar rotation of the scaphoid and scapholunate gapping. DRC, dorsal radiocarpal ligament; R, radius; Tz, trapezium. (From Fractures and dislocation of the carpus. In Trumble TE, editor: Principles of hand surgery and therapy. Philadelphia, 2000, WB Saunders.)

Figure 10–24:
Brunelli tendon reconstruction. A portion of the flexor carpi radialis (FCR) is brought through the distal portion of the scaphoid and then anchored to the dorsal scapholunate ligament and the distal radius.
scaphoid rotation and prevent scaphoid gapping (Figure 10–23).83,84
- Tenodesis: These techniques attempt to reestablish the SLIL and SL relationship through the use of varying tendon weaves.
  - Brunelli procedure: Uses a strip of the flexor carpi radialis (FCR) brought palmarly through a bone tunnel in the distal scaphoid. The tendon is brought dorsally and proximally and attached to the distal radius in an attempt to limit scaphoid flexion and stabilize the SLIL and STIL. Modification involves attaching the FCR to the lunate (Figure 10–24).85
  - Technique: The wrist is approach through standard longitudinal or ligament sparing capsulotomy. The SL ligament is visualized. The scaphoid and lunate are reduced with K-wires. A separate palmar Russe-type incision is made over the distal portion of the FCR. Half of the FCR is harvested, leaving it attached distally to the second metacarpal. This portion of FCR is passed through a bone tunnel in the distal scaphoid. The tunnel is made parallel to the distal articular surface of the scaphoid. The bone tunnel can be made with a cannulated drill passed over a K-wire. The tendon is passed through the tunnel and brought dorsal and proximal to the radiocarpal joint. It is sewn to the dorsum of the SLIL and to the dorsal distal lip of the radius. K-wires are removed at 6 weeks, and range of motion therapy is initiated at 8 weeks.
- Ligament reconstruction
  - Attempts have been made to reconstruct the SLIL with bone ligament–bone constructs from the carpus, foot, and extensor retinaculum (Figure 10–25).86,87
- Arthrodesis
  - Scaphotrapezial: Watson recommended partial wrist arthrodesis with a fusion of the scaphoid, trapezium, and trapezoid (STT or triscaphe fusion) (Figure 10–26).88,89 This fusion decreases the flexion extension by 30% and decreases radial to ulnar deviation by 40%. Load distribution is altered at the radial fossa.
  - Technique: A transverse or longitudinal incision can be used over the STT joint, allowing visualization of the second and third extensor compartments.
  - The articular cartilage is removed down to bleeding cancellous bone. Three K-wires are driven from the trapezium and trapezoid into the STT joint to confirm position. Bone graft is packed into the defect. Compression is achieved by forcing the scaphoid tubercle dorsally and placing the wrist in some radial deviation. Ideally the scaphoid is placed into mild flexion, to aid in radial deviation and wrist flexion postoperatively. The patient is placed into a long arm cast for 4 weeks, followed by a short arm cast until fusion is achieved.
  - STT fusion has a long track record; however, some complications, including nonunion, have
been reported. Meticulous technique is essential for excellent results.88–93

SL fusion
- Attempts to fuse these two bones have resulted in high failure rates.94

Scaphocapitate fusion
- Scaphocapitate arthrodesis is biomechanically very similar to STT arthrodesis.95

Chronic injuries with arthritis (SLAC changes)

Stage I
- Early arthritic changes only present at radial styloid.
  - STT fusion: Combine procedure with radial styloidectomy for pain relief.

Stage II (Figure 10–27)
- At this point, arthritis already is present in the radial fossa, and attempts at preserving the scaphoid often are futile.
  - Four corner fusion: The scaphoid is excised and the midcarpal joint is fused to create one bone block that articulates on the lunate fossa.
  - Technique: A longitudinal dorsal exposure to the wrist is used. The scaphoid is excised piecemeal. The opposing surfaces of the lunate, capitate, triquetrum, and hamate are prepared for arthrodesis. A palmar cortical shell of bone is left to maintain proper spacing between the bones. At this point, any DISI deformity is corrected by flexing the lunate back to a neutral position, often using a K-wire in the lunte as a ‘joystick.’ The bones are held with three to four K-wires and cancellous bone graft is packed into the intercarpal spaces. Final fixation can be obtained using K-wires, screws, plates and screws, or staples (Figure 10–28). Patients are immobilized in a short arm cast until union (6–8 weeks).
  - Pearl: The lunate must be placed into a neutral position prior to pin or screw placement to maximize postoperative motion.
  - Advantage
    - Preserves natural lunate radius articulation
  - Disadvantages
    - Requires meticulous technique to prevent complications
    - Requires 4 to 6 weeks of immobilization

Proximal row carpectomy
- Removal of the entire proximal carpal row allows the capitate to articulate with the radius in lunate fossa. The lunate fossa of the radius usually is always preserved in SLAC arthritis. This procedure can be
performed openly or arthroscopically (Figure 10–29).

- **Technique:** Either a transverse or longitudinal dorsal wrist incision can be used. The extensor retinaculum is incised over the extensor pollicis longus, and flaps are created. We routinely excise the terminal portion of the posterior interosseous nerve just proximal to the wrist because entrapment of this purely sensory portion of the nerve (to the wrist capsule) has been implicated as a possible cause of postoperative pain. The nerve can be found at the most radial margin of the fourth dorsal compartment, just deep to the extensor digitorum communis tendons and alongside the 3–4 intercompartmental septum. A 2- to 3-cm segment is removed just proximal to the articular surface of the radius. The wrist capsule is incised longitudinally, along the third metacarpal axis. Alternatively, a ligament-splitting approach can be used. The lunate and triquetrum are removed, usually in this order. The scaphoid can be excised in its entirety, although we prefer to leave its distal portion attached to the trapezium and trapezoid for additional support of the thumb ray. Carpal impingement on the radial styloid in radial deviation can be managed by limited stylopectomy, but it is important to not destabilize the wrist by removing the radioscapohamate ligament in continuity with the resected styloid. The proximal pole of the capitate is seated into the lunate concavity of the radius. If stability is questionable, bone anchors can be used to tension the dorsal flap of the capsule or a temporary K–wire can be used for 3 to 4 weeks to maintain this position. The pisiform is not excised. Closure is performed in layers and includes the extensor retinaculum. The wrist is immobilized in a bulky conforming plaster dressing for 4 to 6 days and then converted to a short arm cast for 3 weeks. Gradual increased motion is permitted using a removable splint for 2 to 4 weeks or until the patient can freely use the wrist.

- **Advantages**
  - Easy technical procedure
  - Requires only 3 weeks of immobilization

- **Disadvantages**
  - Degenerative changes can be expected at the capitate–radius interface; however, arthritic changes at the capitate–radius interface do not always correlate with clinical symptoms.

- **Ongoing debate exists as to the benefits of four corner fusion over proximal row carpectomy and vice versa; however, no studies to date clearly show superiority of one procedure over the other.

- **Stage III (Figure 10–30)**
  - At this stage the capitate may be too arthritic to allow for proximal row carpectomy. Options may be limited to
  - Four corner fusion
  - Total wrist fusion
  - Total wrist arthroplasty

### Lunotriquetral Ligament Injuries

#### Introduction

- Although prior reports of LT injury have been reported, it was not until 1984 that Reagan, Linscheid, and Dobyns recognized and reported on the role of the LT ligament in the development of proximal carpal row VISI deformity.
- Although LT ligament dissociation with attenuation of secondary ligamentous restraints resulted in a VISI deformity, not all VISI deformities result from LT ligament injury. VISI deformities appear to have a final common pathway that occurs by multiple mechanisms, all of which depend on LT ligament attenuation.
- In describing injuries of the LT ligament, distinguishing between dynamic and static instability is imperative.
LT ligament injuries with normal conventional radiographs and dynamic instability (present only under load or in certain positions) are classified as LT tears.

Fixed carpal collapse (VISI) on conventional radiographs represents static instability and is classified as LT ligament dissociation.

Anatomy of the Lunotriquetral Ligament

- Like the SL ligament, the LTILs are C-shaped ligaments that span the dorsal, proximal, and palmar edges of the joint surfaces (Figure 10–31).
- Microscopically, there are true ligaments in the dorsal and palmar subregions and a proximal fibrocartilaginous subregion, known as the membranous portion.
- In a study of the ligament properties of the LT ligament, Ritt et al. demonstrated that the palmar region of the LT was the thickest and strongest region. These findings support the “balanced lunate” concept, which proposes that the lunate is torque suspended between the scaphoid, exerting a flexion moment through the SL ligament and the triquetrum, exerting an extension moment through the LT ligament.
- The dorsal LT ligament region was most important in rotational constraint, whereas the palmar region of the LT ligament was the strongest and transmitted the extension moment of the triquetrum as it engaged the hamate. The membranous proximal portion of the LT ligament complex was of little significance with respect to constraining rotation, translation, or distraction.

Pathomechanics

- In an uninjured wrist, the scaphoid imparts a flexion moment to the proximal carpal row, while the triquetrum imparts an extension moment, secondary to the midcarpal contact forces. These opposing moments are balanced by their ligamentous attachment to the lunate.
- With loss of the integrity of the SL ligament, the scaphoid tends to flex, while the lunate and triquetrum tend to extend, imparting a DISI stance.
- With loss of integrity of the LT ligament, the triquetrum tends to extend, while the scaphoid and lunate rotate in flexion.
- A complete LT ligament dissociation is not sufficient to cause a static carpal collapse into a VISI stance.
- Sectioning of volar and dorsal subregions of the LT ligament results in a slight divergence of the triquetrum and lunate at extremes of wrist flexion and radial deviation but no collapse, unless considerable compressive forces are applied.
- Additional tear or attenuation of secondary restraints is necessary to create static carpal instability. Both palmar and dorsal carpal ligaments may play roles as secondary restraints. Two anatomic studies have implicated palmar ligament injury in the development of VISI in LT dissociation. Trumble et al. created dynamic carpal collapse with division of the LTIL and ulnar arcuate ligament (lunocapitate) and static with division of the dorsal radiotriquetral, whereas Horii et al.
demonstrated that sectioning of the dorsal radiotriquetral (dorsal radiocarpal) and dorsal scaphotriquetral (dorsal intercarpal) ligament also produced static VISI following LT ligament injury.

- Loss of dorsal ligament integrity allows the lunate to flex more easily, in part by shifting the point of capitate contact palmar to the lunate axis of rotation (Figure 10–32).

**Physical Examination**

- Ulnar deviation with pronation and axial compression elicits dynamic instability with a painful snap if a nondissociative midcarpal joint or LT ligament injury is present.

- Palpation always demonstrates point tenderness at the LT joint. A palpable wrist click occasionally is significant, particularly if it occurs with pain and radioulnar deviation. Provocative tests that demonstrate LT laxity, crepitus, and pain are helpful for accurately localizing the site of pathology. Three useful tests include LT ballottement, shear (Kleinman) test, and compression test.

- Ballottement of the triquetrum, described by Reagan, Linscheid, and Dobyns, is performed by grasping the pisotriquetral unit between the thumb and index finger of one hand and the lunate between the thumb and index of the other. If positive, pain and increased anteroposterior laxity are noted during manipulation of the joint (Figure 10–33).

- The shear test, described by Kleinman, is performed with the forearm in neutral rotation and the elbow on the examination table. The examiner’s contralateral fingers are placed over the dorsum of the lunate. With the lunate supported, the examiner’s ipsilateral thumb loads the pisotriquetral joint from the palmar aspect, creating a shear force at the LT joint (Figure 10–34).

- Pressure on the triquetrum in the “ulnar snuffbox” creates a radially directed compressive force against the triquetrum. Pain elicited with this maneuver may be of LT origin but also may arise from the triquetrotaminate joint or triangular fibrocartilage complex pathology. These tests are considered positive when pain, crepitance, and abnormal mobility of the LT joint are demonstrable (Figure 10–35).

- A nondissociative instability pattern secondary to midcarpal laxity at the triquetrotaminate joint should be ruled out because the symptoms may be similar. The possibility of injury at both levels should be considered.

- Selective midcarpal injection of local anesthetic is useful as a diagnostic tool. Resolution of pain with increased grip strength following injection in patients with LT injuries has been a reliable predictor of satisfactory outcome in our experience. A poor
response to injection implies an extraarticular cause of the patient’s symptoms.

**Radiographic Evaluation**

- Radiographs with LT ligament tears often are normal.
- LT dissociation results in a disruption of the smooth arcs formed by the proximal and distal joint surfaces of the proximal carpal row (Gilula arcs 1 and 2) and the proximal joint surfaces of the distal carpal row (Gilula arc 3) (Figure 10–36).
- LT dissociation results in proximal translation of the triquetrum and/or LT overlap. Unlike SL injuries, no LT gap occurs (see Figure 10–36).
- Motion studies, including deviation and clenched fist anteroposterior views, are helpful.
- In LT dissociation, the normal reciprocal motion of scaphoid, lunate, and distal row is accentuated in deviation while triquetral motion is diminished.
- The increased palmar flexion of the scaphoid and lunate in radial deviation without change of the triquetrum is a manifestation of the loss of proximal row integrity in the normal wrist.
- Careful evaluation of the lunate and triquetrum on lateral radiographs may reveal a malalignment in the absence of frank carpal collapse. The perimeters of the triquetrum and lunate can be traced and their relationship assessed.
- The longitudinal axis of the triquetrum, defined as a line passing through the distal triquetral angle and bisecting the proximal articular surface, forms a 14 degree angle (range +31 degrees to −3 degrees) with the lunate longitudinal axis, defined as a line passing perpendicular to a line drawn from the distal dorsal and volar edges of the lunate. LT dissociation results in a negative angle (mean value −16 degrees) (Figure 10–37).
- If a VISI deformity is present with LT dissociation, the SL and capitolunate angles are altered. The SL angle may be diminished from its normal 47 to 40 degrees or less but often is normal. The lunate and capitate, which normally are colinear, collapse in a zigzag fashion, resulting in an angle greater than 10 degrees.
- Arthrography is valuable, demonstrating leakage or pooling of dye at the LT interspace. However, age-related
LT membrane perforations, other communications between the radiocarpal and midcarpal joints, and asymptomatic LT tears on arthrography of normal wrists have been reported. Therefore, the results of arthrography must be correlated with clinical examination findings.

- A videotaped arthrogram with motion sequences in flexion-extension and radioulnar deviation can further confirm the presence of an LT injury by demonstrating abnormal pooling of the dye column and abnormal proximal row kinematics as previously described.
- Videofluoroscopy is useful for demonstrating the site of a “clunk” that occurs with deviation. In LT sprains, this occurs with a sudden “catch-up” of the triquetrum into extension as the wrist moves into maximal ulnar deviation.

- Technetium Tc 99m-methylene diphosphonate (MDP) bone scans can help identify the site of acute injury but are less specific than arthrography. They may prove helpful in cases where standard films and motion studies are negative.
- MRI technology is not yet reliable for LT ligament imaging but continues to improve and soon may supplant arthrography and bone scans.

Treatment

- Wrist arthroscopy is both diagnostic and therapeutic. Wrist arthroscopy provides a means to directly inspect the integrity of the LT ligaments and allows for identification and treatment of any associated pathology (Figure 10–38).
• In our experience, arthroscopy has provided the most accurate means of diagnosis of LT pathology and may replace all other diagnostic studies.
• Carpal instability can be assessed by direct visualization and probing of the carpal joint articulations. Additionally, provocative maneuvers can be performed while directly visualizing the carpal joint articulations.
• Arthroscopic instability is graded by the Geissler classification.74
• Midcarpal arthroscopy is the key to assessing the stability of the LT joint. From the midcarpal perspective, the normal LT joint is smooth without a step-off or diastasis.
• Surgical reconstruction
  • In acute and chronic dissociations that demonstrate a VISI collapse and chronic tears unresponsive to conservative management, operative treatment is indicated.
  • The goal of surgical intervention is realignment of the lunocapitate axis and reestablishment of the rotational integrity of the proximal carpal row.
  • Performed via a dorsal ligament-sparing capsulotomy (described in SL dissociation section).
  • A variety of procedures have been described, including LT arthrodesis, ligament repair, and ligament reconstruction (Figure 10–39). If concomitant ulnar negative or positive variance or midcarpal or radiocarpal arthrosis is present, additional procedures, such as ulnar lengthening or shortening, midcarpal arthrodesis, or proximal row carpectomy, may be indicated. Total wrist arthrodesis may be indicated when degenerative changes make other salvage procedures impossible.
• LT ligament repair
  • LTIL is reattached to the site of its avulsion, generally from the triquetrum.
  • The technique is demanding, requiring use of multiple sutures passed through drill holes or suture anchors placed in the site of the avulsion.
  • Because the strong volar ligament also is disrupted, a combined dorsal and volar approach and augmentation of the repair by plication of the dorsal radiotriquetral and dorsal scaphotriquetral ligaments may be of some value.
  • Protracted immobilization is necessary (10–12 weeks).
  • Patients with strenuous pursuits, chronic instability, or poor-quality LT ligament may be best managed by ligament reconstruction.
• LT ligament reconstruction
  • Ligament reconstruction with a distally based strip of extensor carpi ulnaris tendon graft is the authors' recommended surgery for LT dissociation.
  • Unlike SL ligament reconstruction, this technique, although demanding, yielded uniformly good results in two studies.64,99–101
  • Reconstruction preserves LT motion and provides the optimal chance for restoration of normal carpal interactions, unlike LT arthrodesis.
Figure 10–39:
Treatment of lunotriquetral (LT) ligament instability includes repair, reconstruction, or arthrodesis. A curvilinear or longitudinal dorsal incision is made, and the third extensor compartment is released and a ulnar-based retinacular flap is created by dividing the septations between the 3-4 and 4-5 extensor compartments (A). The extensor tendons are retracted, and a dorsal ligament sparing capsulotomy is made along the lines of the dorsal intercarpal (dorsal scaphotriquetral [DST]) and dorsal radiocarpal ligaments (dorsal radiotriquetral [DRT]), extending over the radioscaphoid interval (B). The ulnar-based flap is created, carefully elevating it from the lunotriquetral ligament, which is intimate to the dorsal radiocarpal ligament (C). The LT joint integrity can be evaluated from the midcarpal joint. If the dorsal ligament is attenuated or avulsed, a ligament repair can be performed. The site of the avulsion is prepared with either drill holes or with suture anchors (authors' preference) (D). The LT joint is reduced and pinned with two Kirschner wires (D). The ligament is repaired, and the ligament-sparing capsulotomy is closed down using the same suture anchors, followed by repair of the remainder of the capsulotomy (E). If the LT joint is grossly lax, demonstrating complete instability, LT reconstruction is recommended. A distally based strip of extensor carpi ulnaris (ECU) tendon is harvested using a few transverse incisions over the ECU tendon (F). A 2-0 surgical steel wire is used to hold half of the ECU tendon approximately 8 cm proximal to the wrist. It is passed distally in the ECU sheath and brought out at the level of the dorsal wrist exposure. The wire is pulled, creating a distally based strip of ECU tendon.
Figure 10–39: cont’d
(G). The tendon strip is passed into the carpus (H). K-wires are placed in the lunate and triquetrum so that they meet at the volar aspect of the joint (I). Bone tunnels are prepared by hand drilling over the K-wires (J). The ECU tendon strip is passed into the triquetrum, volarly through the lunate, and tightened dorsally (K). Two K-wires are used to secure the reduced lunate and triquetrum, and the tendon graft is tied to itself (L, M). If arthrodesis is chosen, the articular cartilage is removed, leaving a rim of articular cartilage intact to maintain the spacing between the lunate and triquetrum (N).
● LT arthrodesis
  ❍ Technically less demanding than ligament reconstruction or repair and has become the technique of choice of many authors. However, the method is not without substantial problems.64
  ❍ Reported nonunion rate varies from 0% to 57%.64,110–115
  ❍ Use of K-wires has resulted in an unacceptably high nonunion rate of 47%.113 Use of compression screws may improve results, but nonunion remains a significant problem. A 9% nonunion rate was reported with the Herbert screw, and conventional cortical screws may exhibit nonunion rates as high as 57%.113–115
  ❍ Ulnocarpal impingement required additional surgery in 14% of LT arthrodesis patients.110
  ❍ A variety of techniques exist. The authors prefer denuding the articular cartilage while maintaining a rim of articular cartilage to preserve the normal LT width. Cancellous bone graft (iliac crest or distal radius bone graft) is impacted into the defect, and a cannulated compression screw is placed from the ulnar to radial direction.

● Outcomes and comparative studies
  ❍ Comparison of results and outcomes following arthrodesis, ligament repair, and reconstruction has demonstrated superior results with LT ligament repair or reconstruction.104
  ❍ Fifty-seven patients with 57 isolated LT injuries treated by arthrodesis, direct ligament repair, or ligament reconstruction were compared.
  ❍ Average follow-up was 9.5 years (range 2–22).

● The probability of remaining free of complications at 5 years was 68.6% for reconstruction, 13.5% for repair, and less than 1% for arthrodesis.
● Among the LT arthrodeses, 40.9% developed nonunion and 22.7% developed ulnocarpal impaction.
● The probability of remaining free of subsequent surgery at 5 years was 68.6% for reconstruction, 23.3% for repair, and 21.8% for arthrodesis.
● Objective improvements in strength and motion and subjective measures of pain relief and satisfaction were significantly higher in the LT repair and reconstruction groups than in the arthrodesis group.

● Attritional LT instability secondary to ulnar positive variance
  ❍ Refers to LT instability secondary to a long ulna that chronically impacts the triquetrum, resulting in an LT tear with instability. Often associated with a degenerative nonrepairable triangular fibrocartilage tear.
  ❍ Ulnar shortening is an attractive alternative in these cases (Figure 10–40).

### Carpal Instability Nondissociative

When a normal relationship exists between the components of the distal and proximal rows (i.e., no dissociative instability findings) and abnormal motion or dysfunction occurs between the radius and proximal carpal row or between the proximal and distal rows, the wrist is considered to have a nondissociative carpal instability.

### Radiocarpal

- Nondissociative instabilities of the radiocarpal group included patients with incompetent or insufficient radiocarpal ligaments.
  - Chronic
    - Rheumatoid arthritis secondary to the laxity of the radiocarpal ligaments
    - Developmental deformities
    - Madelung deformity
  - Acute

### Radiocarpal Dislocations

- Ulnar translocation
  - First described by Dobyns et al.116 in 1975
  - Further redefined by Taleisnik117,118 into two types (Figure 10–41):
    - Type I: entire carpus, including the scaphoid, is displaced, and the distance between the radial styloid and the scaphoid is widened.
    - Represents a pure CIND
Type II: radius, scaphoid, and distal carpal row relationships are normal; however, the SL space is widened and the lunate and triquetrum are ulnarly translocated.

Combination of CIND and CID (SL dissociation), making it a CIC

Treatment

Only a single series of eight patients with these injuries was reported from the Mayo Clinic.\textsuperscript{119}

Difficult problem to treat effectively

Disappointing results

Recommended radiolunate arthrodesis

Pure radiocarpal dislocations

Exceedingly rare, with 11 cases reported in 1995.\textsuperscript{120}

Dumontier et al.\textsuperscript{121} reported on 27 radiocarpal dislocations (most associated with radiostyloid fractures)

seen over a 23-year period. Seven radiocarpal dislocations were considered “pure.”

Recommended open reattachment of volar carpal ligaments in pure dislocation through a volar approach to obtain best outcome

Midcarpal

Historically, \textit{midcarpal instability} was the term used to describe any instability that occurred in the wrist without a dissociative component. Technically, the dysfunction occurred at either the radiocarpal or midcarpal joint but predominated at the midcarpal joint.

First description by Lichtman et al.\textsuperscript{12} in 1981, when they described ulnar midcarpal instability.

Described characteristic VISI pattern

Symptoms included pain and spontaneous wrist chunk/click with ulnar deviation

Attributed to failure of the ulnar limb of the volar ulnar arcuate ligament

Taleisnik and Watson\textsuperscript{23} in 1984 described the concept of extrinsic midcarpal instabilities

Patients who have a painful snapping/chunking of their wrist with ulnar to radial deviation

Attribute the lesion to extraarticular injury, in this series a distal radius malunion

Pathomechanics

In radial and ulnar deviation of the normal wrist, a smooth transition occurs with a synchronous reciprocal motion between the proximal and distal carpal rows.

In the pathologic state, the smooth synchronous reciprocal motion is disrupted.

The stout triquetrohamate and triquetrocapitate (ulnar arcuate) ligaments are necessary to prevent midcarpal collapse during radial and ulnar deviation but also allow for the smooth progressive transition of the proximal row from flexion to extension as the wrist ulnarly deviates.

The helicoids articulation between the hamate and triquetrum assist in the smooth transition.

Failure or laxity in the triquetrohamate and triquetrocapitate ligaments results in inadequate prevention of midcarpal collapse (development of VISI deformity), and extension of the proximal row is hampered. This results in the sudden and painful clunk when the wrist is ulnarly deviated, and the triquetrum suddenly follows the hamate surface (Figure 10–42).

Classification

Proposed by Lichtman et al.\textsuperscript{14} in 1993 based on their experiences and previous reports.

Palmar Midcarpal Instability

Acute or chronic VISI malalignment secondary to attenuation or rupture of palmar midcarpal ligaments
Medial sided: triquetral hamate capitate ligaments
Lateal sided: scaphoid capitate trapezoid trapezium ligaments

Dorsal Midcarpal Instability
- Young patients with bilateral hypermobile wrists
- Secondary to excessive attenuation from trauma or chronic injury
- Symptoms secondary to disruption or attenuation of radiocapitate ligament
- CLIP: dorsal subluxation of the capitate compared to the lunate^{11,12}

Combined Dorsal and Palmar Midcarpal Instability
- Secondary to laxity of both midcarpal and radiocarpal ligaments
- Includes the proximal carpal row instabilities described by Wright et al.^{29}

Extrinsic Midcarpal Instability
- In patients with chronic dorsal angulated malunited distal radius fractures secondary to stretching of the volar carpal ligaments
- Included in the CIA category

Diagnosis
- Patients often demonstrate painful clunk maneuver, which often involves ulnar deviation and pronation of the wrist (Figure 10–42).
- Wrist may present with a sag in the midcarpal joint, with limited range of motion and impaired grip strength.
- Circumduction test (axial load and ulnar–dorsal rotation of the wrist) may reproduce the clunk and pain, as does axial loading of the wrist with radioulnar deviation.
- Real-time fluoroscopy examination often is the most useful tool for diagnosis.
- Normal synchronous motion in radial and ulnar deviation is lost.
- While ulnarly deviating the wrist from maximal radial deviation, the proximal carpal row suddenly snaps into an extended position, with a dramatic, sometimes audible, and painful clunk.
- Dorsal and volar stress on the carpus may demonstrate the CLIP.

Treatment
- All patients are initially treated with nonoperative measures, which include immobilization, nonsteroidal antiinflammatory medications, activity modifications, and corticosteroid intraarticular injections.^{29}
- Operative options include

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Figure 10–41: Ulnar translocation has been classified into two types. A, Type I is a complete ulnar translocation of the entire carpus (C). B, Type II represents the ulnar translocation of the carpus, leaving the scaphoid (S) in its normal location. L, lunate. (Copyright Mayo Clinic, reproduced with permission of the Mayo Foundation.)
Soft tissue reconstructions\textsuperscript{11,14,29,123}

- Volarly the space of Poirier is obliterated.\textsuperscript{11}
- A volar extended carpal tunnel release is performed, and sutures are placed between the radioscaphocapitate and the long radiolunate ligament radially and between the ulnotriquetrocipitate and ulnolunate ligament ulnarily. The sutures are tightened, thus obliterating the space of Poirier.

Dorsally the dorsal intercarpal and radiocarpal ligaments are imbricated.

Limited intercarpal arthrodesis
- Triquetrohamate arthrodesis\textsuperscript{14}
- Four corner arthrodesis\textsuperscript{14,124}
- Radiolunate arthrodesis\textsuperscript{125}

Carpal Instability Adaptive

- Concept introduced in 1982 by Allieu, Brahin, and Ascencio.\textsuperscript{47}
- Taleisnik and Watson\textsuperscript{23} in 1984 reported on 13 patients with dorsally malunited distal radius fractures with secondary midcarpal malalignment.
- DISI deformity secondary to fracture malunion (see Figure 10–10).
- Progressive pain, tenderness in midcarpal joint, occasional painful clunk.
- Corrective osteotomy consistently resulted in excellent resolution of pain and symptoms.

Complex Instability of the Carpus

- Combination of CID and CIND in the same wrist is called carpus instability complex. All carpal dislocations except for pure radiocarpal dislocations belong in this category. Five groups of dislocations fall in this category:
  - Dorsal perilunate dislocations
  - Dorsal perilunate fracture-dislocations
  - Palmar perilunate fracture-dislocations
  - Axial carpal dislocations
  - Isolated carpal dislocations

Perilunar

- Rare injury patterns that usually are associated with significant trauma (e.g., fall from a height).
- Diagnosis can be delayed because some radiographic findings may be subtle to the untrained eye. Herzberg et al.\textsuperscript{126} reported that 25\% of these injuries are missed during initial presentation.
- Lunate often remains bound to the distal radius by stout radiolunate ligaments but carpus dislocates around it, hence the name perilunar injury. Capitate may move dorsally, causing dorsal perilunate dislocation, or palmarly, causing palmar perilunate dislocation (Figure 10–43).
- Lunate dislocation occurs when the lunate dislocates from radial fossa palmarly (palmar lunate dislocation) (Figure 10–44) or dorsally (dorsal lunate dislocation).
- Fractures may pass through any bone found within the greater arc of the wrist and include the distal radius, scaphoid, trapezium, capitate, hamate, and triquetrum.
- Lesser arc injuries pass only through ligamentous structures with no corresponding fractures (Figure 10–45).

Diagnosis

Physical Examination
- Physical examination may reveal significant swelling, ecchymosis, and decreased range of motion.
- Up to 25% chance of acute carpal tunnel syndrome.126

Imaging

Plain Films
- PA films show disruption of Gilula lines and gapping between carpal bones.
- Lateral films show dislocation of capitate or lunate.

Computed Tomography, Bone Scan, and Magnetic Resonance Imaging
- CT, bone scan, and MRI usually are not required to make the diagnosis.

Treatment

Acute Presentation
- Closed reduction may be performed initially for pain relief, but surgery is the definitive treatment.

Technique for Open Repair
- The carpus is opened dorsally with a longitudinal incision over the third and fourth compartments. The retinaculum is released over the third compartment, and retinacular flaps are elevated radially to the second compartment and ulnarily to the fifth compartment. A longitudinal or ligament-sparing capsulotomy is created, and the carpus is exposed. The lunate is initially reduced and held in place with a longitudinal K-wire. This becomes the foundation for carpal reduction. The triquetrum and scaphoid are reduced and held in place with two to three K-wires. SLIL and LTIL repairs are performed with suture anchors or bone tunnels.
Intraoperative fluoroscopy is essential to verify adequate reduction of the proximal carpal row. We reduce the midcarpal joint with two K-wires through the scaphoid into the capitate and an additional two wires from the triquetrum into the hamate and capitate. Once the carpus is reduced, the initial lunate wire may be removed to prevent immobilization across the radiocarpal joint. The wrist is turned palmarly, and an extended carpal tunnel incision is made to view the palmar ligaments. A large rent may be seen in cases of palmar lunate dislocations, or the palmar ligaments may be avulsed from the bone. Ligament repair is performed with heavy suture, wire or screw, and anchor fixation in cases of frank avulsion. Patients are immobilized for 8 to 10 weeks. Radiocarpal motion is allowed at 8 to 10 weeks. K-wires remain for 10 to 12 weeks (Figure 10–46).

Lunate Dislocations
- These dislocations usually require an extended carpal tunnel approach initially for lunate reduction if the lunate cannot be reduced by closed means (Figure 10–47).

Delayed Presentation
- Outcomes worse than dislocations repaired acutely.

Treatment
- Open reduction internal fixation
- Proximal row carpectomy
- Total wrist fusion

Figure 10–45:
Perilunate fracture-dislocations can occur with disruption of the scapholunate interosseous ligament, fracture of the scaphoid, or fracture of the scaphoid and capitate, depending on the path of injury. (From Fractures and dislocation of the carpus. In Trumble TE, editor: Principles of hand surgery and therapy. Philadelphia, 2000, WB Saunders.)

Figure 10–46:
Repair of perilunate dislocation using bone anchors and K-wires. Scapholunate and lunotriquetral ligaments were repaired using suture anchors via a dorsal approach.
Two studies examining patients treated a minimum of 6 weeks after injury both concluded that open reduction internal fixation provided the most reliable improvement in function and pain.127,128

Axial Carpal

Axial carpal dissociation of the carpus consists of a traumatic longitudinal disruption, in which the carpus is longitudinally split and displaced.61,129–131

These injuries typically result from severe trauma, such as blast or crush injuries, resulting in fracture-dislocations with loss of the normal architecture of the distal transverse (metacarpal) and proximal (carpal) arch.

The normal convex relationship between the metacarpal heads is lost, the palm is flattened, rotational deformities of the fingers occur, and the carpometacarpal area is widened.132,133

The spectrum of axial carpal instability ranges from acute, gross traumatic fracture-dislocations with severe soft tissue trauma to chronic dynamic instability between the axial components of the carpus.70,134 Although a majority of axial carpal instability has been reported as gross traumatic injuries, the concept of axial carpal sprains, with longitudinal dynamic derangement, was described in a case report.70,134

Classification

After reviewing 40 cases reported in the literature and adding 16 other cases from their retrospective review, García-Elias et al.61 classified axial dislocations of the carpus into three groups according to the direction of instability (Figure 10–48):

Axial-ulnar disruption: carpus splits into two columns, in which the radial column is stable with respect to the radius, and the ulnar column (with the metacarpals) displaces ulnarly and proximally

Axial-radial disruption: disruption in which the ulnar column is stable with respect to the radius, and the radial column (including the metacarpals) displaces proximally and radially

Combined axial-radial-ulnar disruptions: combination of ulnar and radial displacement of the columns

Demographics of Injury

The estimated incidence of axial carpal disruptions is between 1.4% and 2.08% of patients with carpal fracture-dislocations or subluxations.61,132,135

In a retrospective review of patients treated for axial carpal disruptions at the Mayo Clinic over a 15-year

Figure 10–47:
Extended carpal tunnel release incision provides exposure to reduce the volarly displaced lunate. A small elevator is used to reduce the lunate, and the rent in the palmar capsule is repaired with sutures. (From Fractures and dislocation of the carpus. In Trumble TE, editor: Principles of hand surgery and therapy. Philadelphia, 2000, WB Saunders.)
period, Garcia-Elias et al. identified 16 patients of 1,140 patients with carpal injuries (1.4%).

- With the increasing awareness of axial carpal disruptions and the increasing frequency of industrial accidents, the reported frequency of axial disruptions of the carpus has increased.

**Mechanism of Injuries**

- The typical mechanism of injury of axial carpal disruptions is a crush (molding press, roller press, wringer machine), twisting, or blast injury.
- As such, a majority of axial dislocation of the carpus have been industrial injuries.61,132,138
- The most common mechanism of injury has resulted from a dorsopalmar compression or crush of the wrist.61,132,136,137
- With sufficient dorsopalmar force applied to the entire wrist, the bones involved in axial carpal disruption either dislocate or sustain sagittal fractures, depending on the obliquity of the force applied and the plane of the intercarpal joints. The more parallel the intercarpal joint to the direction of force, the increased likelihood for dislocation. With increasing obliquity of the intercarpal joint to the direction of force, the likelihood of fracture in the sagittal plane increases.

**Diagnosis**

- A majority of axial carpal dislocations result from high-energy injuries and often present with significant associated soft tissue damage. The severity of associated injuries is directly related to the mechanism of injury and degree of energy imparted.
The spectrum of soft tissue injury can range from swelling with tenderness to total denudation of the hand.

Typically, axial disruptions of the carpus have a dramatic appearance, with massive swelling and tenderness over the entire hand and wrist.

With increasing severity of injury, thenar and intrinsic muscles often are severely damaged, flexor and extensor tendons are disrupted, and neurovascular injuries often are present.

Vascular injuries, with disruption of the radial, ulnar, or both arteries, are not frequent.

Nerve injuries are common and can range from transient neuropraxia to axonotmesis. Acute carpal tunnel syndrome is infrequent and most likely is secondary to the traumatic decompression of the carpal canal that occurs with discontinuity of the flexor retinaculum.

Associated fractures of the metacarpals, carpometacarpal joint, carpals, and distal radius and ulna frequently are associated with axial dislocation.

Axial carpal sprains are injuries to the intercarpal ligaments along the longitudinal axis of the carpus and often present as chronic wrist pain. The hallmark of axial carpal dynamic instability of the capitohamate articulation is the arthroscopic finding of diastasis between the capitate and hamate. Normally this articulation does not allow placement of an arthroscopic probe between the capitate and hamate.

**Treatment**

- Surgical treatment of axial carpal dislocations often is directed toward treatment of the soft tissues that result from the high-energy crush or blast (Figure 10–49).
- Initial evaluation of these injuries must include a thorough assessment of the neurovascular and musculotendinous status of the extremity.
- Early and accurate diagnosis of the soft tissue and bony injuries are essential, because delayed treatment because of inaccurate diagnosis is much less successful compared to early treatment.
- Fasciotomies of the intrinsic and/or forearm compartments should be performed if compartment syndrome is suspected or present.
- Closed reduction with percutaneous fixation of the dislocated and/or fractured carpal bones may be successful; however, interposed soft tissue or bone fragments often preclude an anatomic reduction. In such cases, open reduction is indicated.
- Surgical treatment should be directed at debridement of devitalized tissue, open reduction and percutaneous fixation with K-wires, primary repair or grafting of damaged tendons and/or neurovascular structures, and...
immediate skin coverage with the local tissues or with local or free tissue flaps.

- A dorsal longitudinal approach is used for direct carpal reduction, and a palmar extended approach is used to evaluate the soft tissues.
- Carpal alignment should be obtained via the dorsal approach, and K-wires are used to stabilize the dislocation.
- Intercarpal ligament repair is seldom possible secondary to the severe damage but occasionally can be performed. In these cases, use of suture anchors is preferred rather than drill holes to minimize trauma to the carpus.
- The volar approach allows for evaluation of the neurovascular and musculotendinous structures and of the carpal canal. If not already decompressed traumatically, a prophylactic carpal tunnel release can be performed. Primary repair or grafting of all damaged structures is recommended, followed by immediate wound coverage with loosely sutured skin or with local or distant flaps.
- The ultimate outcome and functional results of treatment depend more on the associated injuries than on the carpal disruption. Lower-energy injuries with fewer associated injuries had better functional outcome than did the high-energy injuries with significant soft tissue damage.
- In the Mayo Clinic series, more severe soft tissue injuries were present, and only four of 13 patients had a good result, five had a fair result, and four had a poor result. Nerve injury was the most predictable factor in determining outcome, as was an axial-ulnar type of injury.

**Isolated Carpal Bone Dislocations**

- Isolated carpal bone dislocations are relatively rare and typically are caused by localized direct or indirect concentrated force over a single area of the wrist. Reports for each carpal bone exist, and detailing each one is beyond the scope of this chapter. With the exception of the lunate and scaphoid, removal of the dislocated bone does not cause significant carpal dysfunction and is well tolerated.

**References**


21. Talesnik J, Malerich M, Prietto M: Palmar carpal instability secondary to dislocation of scaphoid and lunate: report of
This article provides a detailed analysis of the SL ligament. The ligament can be divided into three parts; the thickest portion is dorsal.


A review of the anatomic basis, performance, interpretation, and utility of the “scaphoid shift” in assessing scaphoid pathology.


Reports 13 of 19 patients were completely satisfied at 3.6-year follow-up after SL ligament reconstruction using an autogenous bone-retinaculum-bone graft.


The probability of remaining free from complications at 5 years was 68.6% for reconstruction, 13.5% for repair, and less than 1% for arthrodesis. Of the lunotriquetral arthrodeses, 40.9% developed nonunion and 22.7% developed unnocarpal impaction. Results were significantly better for lunotriquetral repair and reconstruction compared to arthrodesis.

102. Ritt MJ, Bishop AT, Berger RA et al: Lunotriquetral liga-


115. Sennwald GR, Fischer M, Mondi P: Lunotriquetral arthrode-

116. Dobyns JH, Linscheid RL, Cooney WP et al: Lunotriquetral liga-


Open injuries and a delay in treatment resulted in poorer outcomes. The incidence of posttraumatic arthritis was 56%. The best radiographic results were observed after open reduction internal fixation.


Introduction

- Osteonecrosis, or avascular necrosis (AVN), of the carpal bones without a known cause (idiopathic) is rare.
- Lunate AVN (Kienböck’s disease) is the most common type of idiopathic carpal AVN.
- Scaphoid AVN not related to fracture (Preiser’s disease) is much less common. Idiopathic AVN of the remaining carpal bones is even more rare. Principles described here for the lunate are generally applicable to AVN of other carpal bones.
- The etiology of carpal AVN often is unclear. Trauma may be involved but usually is not obvious.
- Disease progression involves fragmentation and collapse of the affected carpal bones, loss of carpal height, and progression of arthritis eventually involving the entire wrist. Treatment is based on the patient’s symptoms and the stage of the disease and attempts (in early stages) to revascularize the bone or (in later stages) to arrest progression of carpal collapse.
- Isolated or repetitive trauma to a lunate predisposed to injury because of any of several factors (bony geometry, vascularity) may lead to a fracture or vascular compromise. Bone necrosis resulting from diminished blood supply results in trabecular fractures, sclerosis, fragmentation, and collapse of the lunate. Carpal height decreases, the capitate migrates proximally, and the scaphoid hyperflexes, leading to degenerative changes in the radiocarpal joint and throughout the wrist.
- The etiology of Kienböck’s disease probably is multifactorial. Abnormalities in blood supply may be a primary factor. The majority of cadaveric specimens receive multiple contributions from both dorsal and palmar branches. However, the lunate was supplied by only a single palmar artery in 7% of wrists in one study and 26% in another study. In addition, intrasosseous branching patterns vary; 31% of specimens show a single path through the bone without significant arborization (Figure 11–1).
- A lunate with a single vessel and minimal branching may be at greater risk for AVN following hyperflexion or hyperextension injuries or perhaps following a minimally displaced fracture.
- Severe injuries such as lunate dislocation result in only a transient appearance of AVN, probably because the lunate usually dislocates palmarly, with a flap of vascularized palmar tissue still attached.
- Venous stasis may be a factor. In one study, in vitro intrasosseous pressure measurements within normal and...
necrotic lunate showed markedly increased pressure in the necrotic bones.4

● The geometry of the lunate itself and of surrounding bones may play a role in AVN. Some, but not all, studies have shown negative ulnar variance predisposes to lunate AVN. Negative ulnar variance then may predispose certain patients to development of lunate AVN but likely is not the sole factor.

● Other studies have suggested that a flatter than normal radial inclination predisposes to Kienböck’s disease.5,6 One investigator also noted a tendency toward a smaller lunate in patients with Kienböck’s disease.5

● Acute trauma is another possible cause of Kienböck disease. An isolated event, such as a fall on the outstretched hand, can compress the lunate between the capitate and the distal radius. Concomitant soft tissue injury may include disruption of the vascular inflow or outflow of the bone.

● Repetitive lesser trauma or multiple cycles of loading may act cumulatively to cause AVN.

Presentation

● Patients occasionally relate a remote history of trauma, either isolated or repetitive. Symptoms vary depending upon the stage of the disease at presentation and may range from mild discomfort to constant, debilitating pain and a dramatically reduced range of motion.

● Symptoms in stage I resemble intermittent wrist sprains and early nonspecific synovitis, with pain associated with motion, and mild swelling.

● Clinical findings in stage II can include more frequent and severe swelling and discomfort with motion.

● Clinical findings in stage III include progressive stiffness and loss of motion. In stage IIIA, these findings are combined with painful radial instability resulting from flexion malposition of the scaphoid.

● Symptoms in stage IV are similar to those of degenerative arthritis of the wrist, with constant pain either with motion or at rest, chronic swelling, significantly diminished range of motion, and reduced grip strength.

Physical Examination

● Inspection of the wrist with Kienböck’s disease may reveal obvious swelling dorsally.

● Range of motion tends to decrease with increasing stage of Kienböck’s disease, first because of swelling and pain and later because of lunate collapse, scaphoid rotation, and arthritic changes.

● As noted in the discussion on presentation, grip strength may be markedly reduced. Swelling over the carpus is common and may occur palmarly and dorsally. Tenderness over the dorsum of the lunate is a frequent finding.

● Pain specifically associated with the scaphoid shift maneuver may be present, beginning with stage IIIa disease, as the scaphoid rotates palmarly.

● Kienböck’s disease in its early stages can be difficult to diagnose. Findings of otherwise unexplained mid-dorsal wrist pain, particularly in the younger patient, should suggest further evaluation with magnetic resonance imaging (MRI).

Radiographic Evaluation and Staging

● Plain x-ray films are obtained if physical examination suggests Kienböck’s disease.

● The Lichtman classification uses radiographic criteria to describe disease stage (Box 11–1).

● In stage I Kienböck’s disease, plain radiographs either are normal or demonstrate a linear compression fracture without sclerosis or collapse of the lunate (Figure 11–2).

● MRI in stage I disease is suggestive of AVN when uniformly decreased signal intensity (reduced vascularity of the lunate) is noted on T1-weighted images in comparison with the surrounding normal bones (Figure 11–3).

● Partial T1 signal loss also may be seen with ulnar abutment, fractures, enchondromas, and osteoid osteoma. Ulnar
abutment should be distinguished from Kienböck’s disease by the focal proximal and ulnar changes in the lunate resulting from mechanical forces applied to the lunate by the relatively long ulna.

**Box 11–1**  
**Stages of Kienböck’s Disease**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>Normal radiographs or linear fracture, abnormal but nonspecific bone scan, diagnostic magnetic resonance appearance (lunate shows low signal intensity on T1-weighted images; lunate may show high or low signal intensity on T2-weighted images, depending on extent of disease process)</td>
</tr>
<tr>
<td>Stage II</td>
<td>Lunate sclerosis, one or more fracture lines with possible early collapse of lunate on radial border</td>
</tr>
<tr>
<td>Stage III</td>
<td>Lunate collapse</td>
</tr>
<tr>
<td>IIIA</td>
<td>Normal carpal alignment and height</td>
</tr>
<tr>
<td>IIIB</td>
<td>Fixed scaphoid rotation (ring sign), carpal height decreased, capitate migrates proximally</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Severe lunate collapse with intraarticular degenerative changes at midcarpal joint, radiocarpal joint, or both</td>
</tr>
</tbody>
</table>


- T2-weighted images typically show low signal intensity in Kienböck’s disease but show increased signal in the presence of revascularization. If radiographs are not diagnostic, particularly in stage I, MRI is obtained.
- Stage II Kienböck’s disease shows increased radiodensity of the lunate, often with one or more fracture lines (Figure 11–4).
- Lunate height is preserved in stage II disease. Density changes in the lunate often are best appreciated on the lateral plain radiograph (Figure 11–5).
- Stage III is divided into two subcategories. In stage IIIA, lunate collapse has occurred but carpal height is relatively unchanged (Figure 11–6). In stage IIIB, in addition to collapse of the lunate, carpal collapse is apparent, with proximal migration of the capitate and/or fixed hyperflexion of the scaphoid (Figure 11–7). Carpal height is decreased (Figure 11–8).
- Lateral radiographs in stage III disease demonstrate a widened anteroposterior dimension of the lunate and an increased scapholunate angle (Figures 11–9 and 11–10).
- Once the lunate has collapsed, computed tomography (CT) can outline the geometry of the bone for surgical planning.
- Stage IV Kienböck’s disease has the findings of stage IIIB disease and degenerative arthritic changes throughout the radiocarpal and/or midcarpal joints.

**Treatment** (Box 11–2)

**Stage I**

- A trial of immobilization (cast or external fixator) for up to 3 months is appropriate as the first treatment
option in stage I Kienböck’s disease. This treatment should allow restoration of vascularity in cases of transient AVN of the lunate. Often, however, the disease progresses.

- In an early series of 22 cases of Kienböck’s disease of various stages treated conservatively,2 17 showed progression and five showed no improvement.

Stage II or IIIA with Positive Ulnar Variance

- In stage II, lunate avascularity has developed, but the bone has not collapsed, as in stage IIIA. Carpal height is normal. Revascularization procedures (restoring blood supply to the lunate) are most successful in stage II.
- Revascularization can be either direct (via insertion of a vascularized pedicle bone graft into the lunate) or indirect (via unloading of forces across the lunate, through shortening of the capitate or the radius). Direct and indirect methods often are combined.
- Vascularized bone grafts useful in operative procedures to treat lunate AVN can be taken from either the dorsal distal radius or the base of the second or third metacarpal.

Surgical Technique for Vascularized Bone Graft from the Base of the Second or Third Metacarpal

- This technique takes advantage of the distal vascular arcade or rete over the carpus and allows harvest of a
vascularized bone graft and procedures on the carpus (e.g., capitate shortening) through a single midline incision (Figure 11–11).

- Releasing the distal portion of the fourth dorsal compartment exposes the distal vascular arcade. The pedicle can be mobilized to either the ulnar or the radial direction to achieve sufficient length to reach the lunate. The bone graft can be harvested from the base of either the second or third metacarpal, depending on where the artery has the greatest area of contact. The periosteum is incised and included with the bone. A segment of bone several millimeters on a side is harvested from the metacarpal using an osteotome and inserted into a cavity prepared in the lunate using a high-speed burr to perforate the nonarticular surface of the lunate and remove the necrotic bone. The procedure can easily be combined with an unloading procedure such as the capitate shortening procedure.

Surgical Technique for Vascularized Bone Graft from the Radius
- The incision curves from the dorsal wrist centered over the lunate to the radial border of the distal forearm. The
branch of the radial artery between the first and second dorsal compartments, referred to as the 1,2 intercompartmental supraretinacular artery, is identified and traced distally to the radial artery by releasing the distal portion of the first compartment. The 1,2 intercompartmental supraretinacular artery joins the distal radius after traveling beneath the tendons of the first dorsal compartment. Once the artery has been traced out, a vessel loop is used to measure the length of the pedicle needed for the bone graft to reach the lunate dorsally. Bipolar cautery is used to cauterize the vessel proximal to the planned site of bone graft harvest. The retinaculum between the first and second dorsal compartments is incised and harvested with the bone graft to protect the perforating vessels entering the distal radius. The bone graft is elevated with an osteotome. The vascularized bone graft is placed into the lunate cavity with the pedicle draped distally to prevent impingement against the dorsal lip of the radius.

- Approximately 50% to 75% of patients treated with direct revascularization procedures show evidence of revascularization of the lunate. Carpal collapse continues to occur in approximately 20% of cases.
- Additional or alternative treatment options in stage II or IIIA with positive ulnar variance include radial closing wedge osteotomy or capitate shortening.

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**Box 11–2 Options for Treatment of Kienböck’s Disease**

<table>
<thead>
<tr>
<th>Stage of Disease</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I-III with negative or neutral ulnar variance</td>
<td>Immobilization (3 months)</td>
</tr>
<tr>
<td>II and III with positive ulnar variance</td>
<td>Direct revascularization and external fixation or temporary scaphotrapeziotrapezoid fusion (stage II only), radial wedge or dome osteotomy, capitate shortening with or without capitohamate fusion, combination of joint leveling and direct revascularization procedures</td>
</tr>
<tr>
<td>IIIB</td>
<td>Scaphotrapeziotrapezoid or scaphocapitate fusion with or without lunate excision with palmaris longus autograft, radius-shortening osteotomy, proximal row carpectomy</td>
</tr>
<tr>
<td>IV</td>
<td>Proximal row carpectomy, wrist arthrodesis, wrist denervation</td>
</tr>
</tbody>
</table>

Surgical Technique for Capitate Shortening

- The capitate is approached using a dorsal incision. The tendons of the fourth compartment are retracted to the ulnar side, and the capsule overlying the capitate is incised longitudinally. The osteotomy site is planned to pass through the waist of the capitate and is confirmed with fluoroscopy. With a fine oscillating saw, a 2- to 3-mm wafer of bone is resected from the capitate (Figure 11–12). The saw cuts are completed with small osteotomes to prevent injury to the flexor tendons on the palmar surface of the capitate. The bone surfaces of the capitate are compressed manually and stabilized using crossed Kirschner wires or countersunk screws. Capitohamate arthrodesis can be performed using a small burr to denude the cartilage between the carpal bones, and the bone from the capitate osteotomy is packed in the space. Transverse Kirschner wires or cannulated screws are inserted percutaneously from the ulnar border of the hand. There usually is little motion between these bones, and we often omit this portion of the procedure. If Kirschner wires are used, the ends are cut off and buried beneath the skin.

- A biomechanical study of capitate shortening with capitohamate fusion showed significantly decreased resultant load across the radiolunate articulation. This procedure is technically easier than procedures involving the radius and is my preference.

Stage II or IIIA with Negative or Neutral Ulnar Variance

- In the patient with stage II or IIIA Kienböck’s disease and significant negative ulnar variance, shortening osteotomy of the radius may be performed to reduce load on the lunate (Figure 11–13).

Radial Shortening

- This technique is used in symptomatic patients with stage I, II, or IIIA Kienböck’s disease and negative ulnar variance. The procedure aims to achieve joint leveling, leaving an ulnar neutral to 1-mm ulnar positive variance. This technique can be combined with a revascularization procedure as described in the section on stage II or IIA with positive ulnar variance.

- The distal radius can be approached either dorsally or palmarly. The dorsal approach is more straightforward, but meticulous attention must be paid to the volar structures when performing the osteotomy. A straight dorsal incision is made just ulnar to Lister’s tubercle, from the level of the radiocarpal joint to a point 10 cm proximal. The extensor pollicis longus (EPL) tendon is identified and released from its compartment. The extensor digitorum communis (EDC) tendons and muscle bellies are dissected ulnarward, and the dorsal distal radius is exposed between the EPL and EDC. A T plate or seven-hole dynamic compression plate is applied to the bone. Lister’s tubercle usually is removed with an osteotome or rongeur to allow plate application. The T plate allows for a more distally based osteotomy in metaphyseal bone to facilitate healing at the osteosynthesis site. When a dynamic compression plate is used, the most distal two screw holes are drilled, measured, and tapped, and the screws are placed. The second most distal screw then is removed and the plate rotated out of the way on the last screw. The osteotomy is planned at the level of the fourth, or middle, screw hole in the plate. Preoperative planning using radiographs allows estimation of the amount of bone to be removed with the osteotomy. Removal of a wedge of bone sufficient to leave ulnar neutral or 1-mm ulnar positive variance is planned, which generally amounts to 2 to 4 mm of radius. The osteotomy site is marked with two transverse light saw cuts separated by the planned width of bone resection. The distal cut is made partway through, protecting volar structures with retractors. The proximal cut is begun using a free blade fitted into the distal cut as a guide to ensure the cuts are parallel. The proximal cut is completed, and then the distal cut is made. The plate is rotated back into position and the screw replaced in the next-to-last hole. The proximal screws are applied in compression mode. The wound is irrigated and closed in layers, and a sugar tong splint is applied. Removal of the plate at a later date may be advisable if extensor tendon irritation develops.

- Sufficient bone should be removed to result in neutral to 1-mm positive ulnar variance. Positive ulnar variance greater than 1 mm risks abutment of the ulna upon the lunate or triquetrum, with persistent or worsening ulnar-sided symptoms after surgery.

Figure 11–12:
A report on radial shortening demonstrated diminished pain in 93% of cases, with radiographic signs of lunate revascularization in one third of patients.\(^\text{11}\) Range of motion was improved in 52% but worsened in 19%. Grip strength improved in 74%.

**Stage IIIB**

- Stage IIIB Kienböck’s disease includes radiographic signs of lunate collapse and loss of carpal height because of hyperflexion of the scaphoid.
- Correcting the scaphoid to its normal posture of 45 degrees of flexion followed by fusion to either the trapezium and trapezoid (scaphoid trapezium trapezoid [STT] fusion) or to the capitate (scaphocapitate [SC] fusion) decreases load across the radiolunate joint and prevents further carpal collapse, although wrist flexion and extension are decreased.\(^\text{2,12}\)

**Surgical Technique for Scaphoid Trapezium Trapezoid Arthrodesis**

- An incision is made along the dorsoradial aspect of the wrist beginning approximately 1 cm proximal to the base of the thumb metacarpal (Figure 11–14). The EPL tendon is identified and its sheath partially released with that of the extensor carpi radialis longus and brevis tendons so that they can be retracted radially while the EDC tendons are retracted ulnarly. A small fat pad overlying the STT joint must be bluntly dissected. Care should be taken to avoid injuring the branch of the sensory radial nerve. The capsule of the STT joint is incised transversely, and there typically is an overhanging prominence of the trapezoid blocking the surgeon’s view. This prominence can be removed with a small osteotome. The surfaces are denuded with a rongeur, and two or three 0.0625-inch Kirschner
wires are driven in a dorsal to palmar direction. The dorsal distal to palmar proximal direction ensures the Kirschner wires will cross the STT joint to stabilize the scaphoid. Bone graft can be harvested from the distal radius and is packed into the space produced by removal of the STT joint cartilage, particularly in the palmar depths of the wound where substantial contact occurs. Placing pressure against the scaphoid tubercle and bringing the wrist into slight radial deviation causes a tight coaptation of the scaphoid and trapezoid surfaces. The Kirschner wires are driven into the scaphoid. A styloidectomy of the radius should be performed at the same time as the STT joint arthrodesis.

- Cancellous bone graft is obtained either from a cortical window in the dorsum of the distal radius just proximal to Lister tubercle or via a radial styloidectomy. Alternatively, the iliac crest can be used as the donor site. The scaphoid is manually reduced from its hyperflexed position into approximately 45 degrees of flexion, and two 1.6-mm Kirschner wires are driven from the radial side of the scaphoid into and through both cortices of the capitate (Figure 11–15). These wires should be introduced at an angle, rather than parallel, to one another to reduce motion between the bones. After confirming their position on plain radiographs, the pins are cut short beneath the skin. The cancellous bone graft is packed into the cavity between the scaphoid and the capitate. The capsule is closed, followed by wound closure in layers and application of a sugar tong thumb spica splint. This splint is converted to a short arm thumb spica cast, which is worn until radiographic signs of union are apparent, usually at 8 to 10 weeks.

- In a three-dimensional theoretical wrist model, Iwasaki et al. demonstrated reduced force across the radiolunate joint after STT or SC fusion.

- If significant synovitis is present in stage IIIB disease, excision of the lunate may be performed in addition to the fusion or other procedure. Silicone prostheses are no longer used due to high rates of particulate synovitis.

Surgical Technique for Scaphocapitate Arthrodesis

- This technique can be used as an alternative to STT fusion in stage IIIB Kienböck’s disease, and the objectives are the same. The incision and approach are as described for STT fusion, with retraction of the second and third dorsal compartment tendons radially and the fourth compartment ulnarly. Alternatively a straight dorsal incision as described for the vascular pedicle harvest from the second or third metacarpal base can be used. The wrist capsule is opened longitudinally and the scaphoid, capitate, and lunate identified. The articular surfaces at the scaphoid-capitate interface are denuded of bone with a rongeur or a burr. The very most palmar cartilage and subchondral bone can be left in place to maintain appropriate spacing of the bones.

- Postoperatively, the patient is immobilized in a cast with the index and middle fingers flexed 90 degrees at the metacarpophalangeal joint and the thumb included in the cast. The cast is maintained for 6 weeks, and then a short arm thumb spica is used for another 2 weeks. The Kirschner wires are removed at this time. Radiographs should be obtained to confirm a solid arthrodesis.

Figure 11–14: Scaphoid trapezium trapezoid arthrodesis. (From Allan CH, Trumble TE: Kienböck’s disease. In Trumble TE, editor: Principles of hand surgery and therapy. Philadelphia, 2000, WB Saunders.)

Stage IV

- In stage IV Kienböck’s disease, generalized degenerative changes are seen throughout the midcarpal joint, radiocarpal joint, or both. Lunate collapse and fixed scaphoid rotation with loss of carpal height are still present.
- Attempts to revascularize or decompress the lunate that do not address the arthritic process fail.
- Treatment options include proximal row carpectomy (PRC) and wrist fusion.
- PRC preserves most of the already limited motion of a wrist with stage IV disease, is simple to perform, and leaves open the possibility for wrist fusion at a later date if required.

Technique for Proximal Row Carpectomy

- This procedure removes the scaphoid, lunate, and triquetrum, allowing the capitate to settle into the lunate fossa. Motion then occurs through this joint. Preoperative evaluation must include careful assessment of articular wear in the radiolunate joint, which risks early failure of this procedure. Mild wear encountered at surgery can be addressed by interposing dorsal capsule between the capitate head and the lunate fossa. More extensive wear may leave total wrist arthrodesis as the procedure of choice.
- A straight midline incision is made just ulnar to Lister tubercle and centered over the carpus from distal radial articular surface to the level of the metacarpal bases. The EDC tendons are retracted ulnarly and the wrist extensors radially. The wrist capsule is incised longitudinally (if interposition of a flap in the lunate fossa is planned, this is constructed now) and the carpus exposed. Often the collapsed lunate is difficult to identify, and the capitate head is found first. Careful exposure of the entire proximal row is performed. Small Hohmann retractors can be helpful in keeping the capsule edges from obscuring the surgeon’s view. The scaphoid, lunate, and triquetrum are removed. Volar flexion of the scaphoid can make this bone particularly challenging to excise completely but is recommended to prevent impingement of retained distal pole fragments on the radial styloid. Using a large K-wire or small Steinmann pin as a joystick can simplify this procedure. Small, sharp osteotomes and curettes can be useful in addition to knife dissection. It is important to identify and protect the radioscaphocapitate ligament, running from proximal and radial across the volar waist of the scaphoid toward the center of the capitate. Injury to this structure may allow excessive ulnar translation of the capitus. Once the proximal row is removed, the capitate is approximated within the lunate fossa (interposing a flap of dorsal capsule, if mild arthritic wear is present) and the wound closed in layers. A final check is performed to ensure the capitate is located; if it is not, temporary Kirschner wires are used to maintain the capitate in its reduced position within the lunate fossa. A sugar tong splint is applied, and 2 weeks postoperatively the sutures are removed, a removable splint provided, and gentle active range of motion started.
- A 1-cm segment of the posterior interosseous nerve is excised within the fourth dorsal compartment when performing PRC to minimize postoperative pain.

Kienböck’s Disease in Children

- AVN of the lunate in the pediatric population is rare.
- Treatment has ranged from observation alone to temporary pinning of the scaphotrapezial joint or formal radial shortening.13,14
- A review article collected data from reports on 32 patients, most treated operatively, and noted significant potential for revascularization of the lunate in children.15

Preiser’s Disease

Etiology

- The factors leading to development of Preiser’s disease, or AVN of the scaphoid, are not known with certainty but probably include some form of trauma in the majority of cases. One report found that all of Preiser’s original cases and three of seven subsequent reported cases occurred after fracture or other injury.1 Other suggested factors have included alcoholism, corticosteroids, chemotherapy, systemic lupus erythematosus, and progressive systemic sclerosis.16
- Ulnar positive variance has been present in some cases,1 but a large series found no such association.16

Presentation

- In a multicenter retrospective series of 19 patients diagnosed with Preiser’s disease, the average patient age at presentation was approximately 45 years. Women outnumbered men by approximately 3 to 1. The dominant wrist was affected in approximately two thirds of patients.

Physical Examination

- Pain was present in the dorsoradial aspect of the involved wrist in all cases, whereas swelling was present in only five of the patients. Of interest, nine patients were diagnosed with concomitant carpal tunnel syndrome at some point during the disease course.
Radiographic Evaluation

- In this large series, two patterns of scaphoid involvement were identified in Preiser’s disease. Cases where the entire scaphoid bone showed MRI signal changes of necrosis and/or ischemia were classified as type I, and those displaying MRI signal changes involving 50% or less of the bone were classified as type 2 (Figure 11–16).

Staging

- A four-stage classification system for scaphoid necrosis originally was described by Herbert and Lanzetta1 and modified by Kalainov et al.16 In stage 1, plain radiographs are normal but MRI reveals signal changes of necrosis. In stage 2 scaphoid sclerosis, lucencies and fissuring are found on plain radiographs. Stage 3 is defined by radiographic fragmentation of the scaphoid. In stage 4, fragmentation and collapse of the scaphoid and periscaphoid arthritis are present.

Treatment

- As with Kienböck’s disease, the optimal treatment of Preiser’s disease is not known. A similar array of therapies have been used, ranging from cast immobilization or external fixation to curettage, vascularized bone grafting, scaphoid excision with or without intercarpal fusion, PRC, and total wrist arthrodesis.

References


The author reviews the technique and indications for capitate shortening for Kienböck’s disease.

This biomechanical cadaver model suggests that capitate shortening with capitate-hamate fusion increases radioscaphoid mean pressure and decreases radiolunate mean pressure.

The authors report good results in 68 patients who underwent radial recession osteotomy for avascular necrosis of the lunate.

A three-dimensional mathematical model was used to simulate and compare the biomechanical effects of three different intercarpal fusions for Kienböck’s disease.

Temporary scapho-trapezoidal joint fixation with Kirschner wires was performed for stage IIIB Kienböck’s disease in a 12-year-old girl, allowing clinical and radiographic healing.

The authors report the case of an 11-year-old girl with Kienböck’s disease treated successfully with radial shortening.

A 13-year-old boy with symptomatic stage III Kienböck’s disease was treated successfully with radial shortening.

A large series of patients with Preiser’s disease was reviewed and two different categories of this disorder were identified: complete versus partial vascular impairment of the scaphoid bone as determined by MRI.
History

- In 1922, Sterling Bunnell, the founding father of American hand surgery, wrote, “If flexor tendons are severed in the finger, the usual place opposite the proximal phalanx, one cannot join them together by sutures with success, as the junction will become adherent in the narrow fixed channel and will not slip. It is better to remove the tendons entirely from the finger and graft in new tendons smooth throughout its length.” Dr. Bunnell’s statement has offered a challenge to surgeons and scientists interested in tendon injury and repair.
- Before 1966, flexor tendon lacerations in the area of the digit were treated with delayed methods of staged tendon reconstruction.
- Today, despite the challenges imparted by flexor tendon and digital anatomy, advances in the basic science of tendon repair, surgical methods of repair, and rehabilitation, reasonable outcomes can be obtained in most of patients with zone II and other flexor tendon injuries.

Introduction

- Flexor tendon injuries are common. They involve lacerations and/or ruptures and occur predominately in males between 15 and 30 years old.
- Complete tendon injuries require repair/reattachment for return of function.
- Zones of tendon injury (Figure 12–1) influence the type of repair and postoperative regimen.

Differential Diagnosis

- Nerve injury → muscle paralysis
- Paralysis secondary to pain (self-splinting)
- Underlying medical condition, such as polio, leprosy, Charcot-Marie-Tooth, spinal muscular atrophy

Flexor Tendon Anatomy

Overview

Carpal Tunnel

- In the carpal tunnel, the middle and ring finger superficialis tendons lie volar to the small and index finger superficialis tendons (see Figure 1–14).
- A good way to remember the relationship is that 34 is greater than 25.

Digital Sheath

- Each of the fingers has a deep flexor digitorum profundus (FDP) and a superficial flexor digitorum superficialis (FDS) tendon.
- Note: Approximately 20% of patients are missing an FDS tendon in the little finger.
- The fibroosseous sheath begins at the level of the metacarpal neck.
- The flexor tendon sheath is composed of five annular pulleys (A1–A5) and three cruciate pulleys (C1–C3) (see Figure 1–18).
● A2 and A4 are the most important pulleys to preserve in order to prevent bowstringing of the tendon(s).
● Without pulleys, the tendons can no longer glide juxtaposed to the phalanges. Greater amplitude of muscle contraction is required to obtain the same amount of flexion of the finger.
● Digital artery branches, or vincula, assist with tendon nutrition (Figure 12–2).
● Once in the sheath, the FDS forms Camper’s chiasm by splitting into two slips that attach on the palmar sides of the middle phalanx.
● The FDP tendon passes through the FDS chiasm and continues on to attach to the volar aspect of the distal phalanx.

Thumb Sheath (Figure 12–3)
● Only one tendon, the flexor pollicis longus (FPL), provides flexion of the interphalangeal joint.
● The sheath of the FPL is composed of:
  ● Two annular pulleys
  ● Only one oblique pulley, which is the main structure that prevents bowstringing of the tendon
● The FPL has one continuous synovial sheath that begins just proximal to the carpal tunnel and continues the length of the tendon.
● Note: The index, middle, and ring fingers also have a synovial sheath that begins proximal to the carpal tunnel, but the sheath stops at the level of the transverse intermetacarpal ligaments and resumes within the fibroosseous tunnel. The synovial sheath for the small finger continues on from the palmar bursa to the end of the finger (Figure 12–4).

Tendon Zones
● Five very important zones exist (see Figure 12–1).

Zone I: Zone of Flexor Digitorum Profundus Avulsion Injuries (“Jersey Finger”)
● Region between the middle aspects of the middle phalanx to the fingertip.
● Contains only one tendon, the FDP.
● Tendon laceration usually is very close to its insertion.
● Tendon to bone repair usually is required instead of tendon to tendon repair.

Zone II: “No Man’s Land”
● Region from the metacarpal head to the middle of the middle phalanx.
● Bunnell* referred to this area as “no man’s land” because the initial results were so poor that he believed no one should attempt primary tendon repairs in this zone.
● Two flexor tendons: FDS and FDP within the one flexor tendon sheath.
Adhesion formation risk is amplified at the point where the profundus travels through the FDS at the narrow and tight Camper’s chiasm (see Figure 12–2).

- Camper’s chiasm can be up to 2 cm long.
- During digit flexion, the two slips of the FDS move toward the midline and compress the profundus tendon.

**Zone III: Distal Palmar Crease**

- Region between the transverse carpal ligament and the proximal margin of the tendon sheath formation.
- The lumbrical muscle origins in this zone prevent the profundus tendons from overretracting. Delayed end to end repairs have been successful even several weeks after the initial injury.

**Zone IV: Transverse Carpal Ligament**

- This is the region deep to the transverse carpal ligament.
- Tendon injuries in this zone are rare because of the protection provided by the stout transverse carpal ligament.

**Zone V: Proximal**

- Region proximal to the transverse carpal ligament.
- Distal portion of zone V tendons consists of discrete structures.
• Proximal portion of zone V meets the musculotendinous junction.
  ○ The junction is a poor site for repair because the tendons become thinner and fan out into fibers that merge with the muscle belly.

**Examination**

• Observe the patient from a distance before manipulating the patient who has sustained a painful injury.
• There is a normal arcade to the hand.
  ○ The index finger demonstrates the least flexion.
  ○ The little finger displays the greatest flexion.
• If the injured digit demonstrates more extension than the other digits, there is a high likelihood of a tendon laceration that should be evaluated surgically.
• Avoid using a local anesthetic until a careful sensory test has been performed. Determination of a concurrent digital nerve injury is imperative.

**Provocative Testing**

**Testing the Flexor Digitorum Profundus**

• Hold the proximal interphalangeal (PIP) joint in extension (blocking the PIP joint) and ask the patient to flex each of the injured digits at the distal interphalangeal (DIP) joint (see Figure 1–16).
• The FDP muscles all fire as a group, except for the index FDP, which can flex independently.

**Testing the Flexor Digitorum Superficialis**

• When the patient holds the other digits in extension, the FDP is blocked from flexing any of the digits except for the index finger (see Figure 1–15).
• The FDS, with independent innervation, can be tested separately.

**Testing the Thumb**

• The FPL can be tested by stabilizing the metacarpophalangeal (MP) joint and asking the patient to flex the interphalangeal (IP) joint (see Figure 1–17).

**Imaging Studies**

• The injured hand should be radiographed to exclude any underlying fractures.

**Tendon Structure and Biomechanics**

• The overall excursion of the FDS and FDP tendons (wrist and finger motion) is 88 mm and 86 mm, respectively.
• Tendon composition:
  ○ 78% of type I collagen
  ○ 19% of type III collagen
  ○ Remaining 3% includes a wide variety of other collagen types and noncollagen proteins
• Tendons demonstrate a nonlinear viscoelastic property.
  ○ These properties derive from the tendon’s collagen structural deformity, which is time rate dependent. For example, at the bone-tendon interface, a slow loading rate results in an avulsion fracture of bone, whereas a fast loading rate causes tendon failure.

**Tendon Nutrition and Blood Supply**

• The nutrition derives from two sources: vascular perfusion (intrinsic) and synovium (extrinsic).
  1. Vascular perfusion (intrinsic):
    ○ Longitudinal vessels enter the palm and extend down the intratendinous channels.
    ○ Vessels enter at the level of the proximal synovial fold in the palm.
    ○ Vincula harbor segmental branches from the digital arteries.
    ○ Osseous insertions are sources of vascular perfusion.
  2. Direct diffusion (extrinsic) of nutrients from the synovial fluid:
    ○ Enhanced by the capillary pumping mechanism known as imbibition.
    ○ Imbibition: Finger flexion provides a pumping mechanism as the tendon glides into the fibroosseous pulleys, which helps draw fluid into the interstices of the tendon through small ridges or conduits.

**Cellular and Biochemical Factors in Tendon Healing**

**Physiology of Tendon Injury and Repair** (Table 12–1)

• Tendon injuries stimulate a potent chemotactic response.
• Stimulated cells migrate into the zone of injury from either the epitenon or the synovial sheath.
• Cells histologically resembling myofibroblasts synthesize collagen.
• The epitenon is clearly the most active segment of the tendon both for collagen synthesis, with initiation of α-procollagen, and for phagocytosis of collagen debris resulting from the injury.
Table 12–1: Stages of Intrinsic Repair for Intrasynovial Flexor Tendons

<table>
<thead>
<tr>
<th>INFLAMMATORY PHASE (0 TO 14 DAYS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrin clot forms at the repair site</td>
</tr>
<tr>
<td>Macrophage migration and leukocyte migration to the repair site</td>
</tr>
<tr>
<td>Phagocytosis of the repair site</td>
</tr>
<tr>
<td>Fibronectin production peaks (chemostasis)</td>
</tr>
<tr>
<td>bFGF production peaks</td>
</tr>
<tr>
<td>Upregulation of integrins</td>
</tr>
<tr>
<td>Cells from the epitenon proliferate and migrate to the repair site</td>
</tr>
<tr>
<td>Gliding surface is restored</td>
</tr>
<tr>
<td>Fibrin strands identified in fibroblasts surrounding the repair site</td>
</tr>
<tr>
<td>Immediately after repair, the strength of the repair is related to the strength of the suture and the suture method</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reparative Phase (2 to 6 Weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intense collagen production, mostly type I</td>
</tr>
<tr>
<td>Fibers of collagen are laid down randomly and gradually orient themselves along the axis of tensile forces</td>
</tr>
<tr>
<td>Cellular ingrowth from the epitenon fills the repair site gap</td>
</tr>
<tr>
<td>Neovascularization of the repair site occurs</td>
</tr>
<tr>
<td>TGF production peaks</td>
</tr>
<tr>
<td>Fibrous strands of collagen bridge the repair site</td>
</tr>
<tr>
<td>DNA content is increased</td>
</tr>
<tr>
<td>At 2 weeks after repair, the repair site strength may temporarily decrease, but the overall strength increases during this period as collagen deposition occurs at the repair site</td>
</tr>
<tr>
<td>Repair site strength is still principally related to the strength of the suture and the suture material</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Remodeling Phase (&gt;6 Weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collagen fibers are smooth and uniform in the repair site</td>
</tr>
<tr>
<td>Collagen fibers are remodeling to be oriented parallel to the longitudinal axis of the tendon</td>
</tr>
<tr>
<td>The surface of the tendon is smooth and nonadherent</td>
</tr>
<tr>
<td>DNA content remains increased</td>
</tr>
<tr>
<td>Decreased rates of cell division</td>
</tr>
<tr>
<td>Increase in repair site strength</td>
</tr>
</tbody>
</table>

Cytokines and Growth Factors

- Platelet-derived growth factor and epidermal growth factor were identified in healing flexor tendons from 3 to 17 days after repair.²
- Transforming growth factor-beta was noted in healing tendons and in the tendon sheath.
- Fibroblastic growth factor was absent from the repair process.
- Hyaluronic acid, a normal component of tendon synovial fluid, did not increase collagen synthesis and actually depressed cellular proliferation.
- Ascorbic acid at a concentration greater than 50 μg/ml is essential for tendon healing.
- Metalloproteinases assist in regulating collagen synthesis.
- Proteoglycan concentrations are elevated in regions of the tendon and tendon sheath that are subjected to tendon load.

Table 12–2: Ideal Core Suture

<table>
<thead>
<tr>
<th>Material suture properties of sufficient strength to allow early mobilization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy material handling when placed into soft tissue</td>
</tr>
<tr>
<td>Material durable through the process of tendon repair</td>
</tr>
<tr>
<td>Surgical method that is easy to perform and accurately coaptsthe tendon ends</td>
</tr>
<tr>
<td>Surgical method should not interfere with tendon repair</td>
</tr>
</tbody>
</table>

Tendon Repair Characteristics

- **Repair tendon injuries early.**
- Most repairs should be performed within the first 2 weeks after injury because the tendon ends and tendon sheaths become scarred, and the musculotendinous units retract.
- Repairs after 2 weeks may decrease the ultimate mobility of the fingers.
- The strength of the flexor repair and the ability to resist gapping appear to be directly proportional to the number of sutures that cross the repair site.
- Tendon repairs demonstrate a 20% to 40% decrease in tendon strength during the first 3 to 7 days after tendon repair. If repair strength dips below the threshold of the loads produced by active or passive motion, then tendon rupture occurs.
- Tensile stress on normally repaired flexor tendons is as follows:
  - Passive motion: 500 to 750 g
  - Light grip: 1500 to 2250 g
  - Strong grip: 5000 to 7500 g
  - Tip pinch, index FDP: 9000 to 13,500 g
- Tendon gapping is the hallmark of tendon failure (gapping of 2 mm or more)

Six characteristics of an ideal repair:
1. Easy placement of sutures in the tendon (Table 12–2)
2. Secure suture knots
3. Smooth juncture of tendon ends
4. Minimal gapping at the repair site
5. Minimal interference with tendon vascularity
6. Sufficient strength throughout healing to permit application of early motion stress to the tendon

Note: A practical and strong repair allows early active motion without significantly increasing friction with tendon gliding.³⁴

- Numerous methods of tendon suture (Figure 12–5) have been advocated in an effort to satisfy the characteristics of an ideal repair.
- Techniques to minimize gapping
  - Place core sutures dorsally.
  - During digit flexion, the dorsal aspect of the tendon is under the greatest tension. Dorsal placement of core sutures is the best mechanical location for tendon repair.⁵
Dorsal core sutures result in less work of flexion.\(^6\)

- **Note:** Prior conventional wisdom specified that the sutures should be placed in the palmar aspect of the tendon because the blood supply to part of the tendons is via the vincula that are attached to the dorsal surface of the tendon. (Theoretically, sutures placed in the dorsal aspect of the tendon obstruct the blood supply and cause tendon ischemia.) However, later studies of tendon nutrition indicated that imbibition of synovial fluid may be the major pathway for tendon nutrition following tendon injury.

Epitenon sutures improve the strength and quality of tendon repairs.

- Improves the contour of the repair.
- Minimizes gap formation and adds to the ultimate tensile strength of the repair.\(^7\)

- **No need for tendon sheath repair.**

Although clinical reports have indicated that repairs of the tendon sheath appear to be safe, a clinical comparison showed no significant improvement in patients treated with sheath repair compared with patients treated without sheath repair.\(^8\)

- **Note for partial tendon laceration repair:** Lacerations of less than 60% of the cross-sectional area of the tendon should be treated without tenorrhaphy and with early mobilization.\(^9\)

- **Note:** It is advantageous to trim frayed or partial lacerations to prevent triggering of the frayed edges.

**Practical Approach: Acute Tendon Repair**

- **Exposure:** The skin lacerations often are transverse, making it difficult to extend the incision with a Brunner-type zigzag incision. Longitudinal incisions on opposite sides proximally and distally can provide enough exposure without compromising the skin margins (Figure 12–6).
The level of tendon laceration is defined with the digit fully extended. The distal end of the tendon will be distal to the skin and tendon sheath (Figure 12–7) when the tendon is cut with the finger in flexion, as is usually the case. If the finger was cut in extension, the tendon laceration is at the same level as the skin laceration.

Zone I

- As with most tendon lacerations in the digits, the wound must be extended distally and proximally for precise visualization.
- With the proximal tendon retrieved, core sutures are placed in the tendon for subsequent passage with Keith needles. Care is taken to minimize disruption of the important A4 pulley.
- Tendon fixation: Keith needles are used to pass the sutures around the distal phalanx exiting through the nail plate dorsally (Figure 12–8). The pullout suture is left in place for 6 to 7 weeks over the nail plate or suture button.
- The repair can be augmented by suturing the remaining distal end of the tendon to the reattached proximal portion of the tendon in a belt and suspenders manner, taking care not to injure (cut) the core sutures with the suture needle.
- Note: Some surgeons prefer to drill the Keith needles through the distal phalanx with a volar to dorsal angle (similar to Figure 12–8) rather than on either side of the bone. Alternatively, mini-suture anchors can be used, although my preference is to use a dorsal suture button.

FDP avulsion injuries (Jersey finger) (Box 12–1)
- There are three types of FDP avulsions.10
- In type I avulsions, the tendon has retracted into the palm. The tendon should be repaired within 7 days. The blood supply is compromised, and the tendon contracts/shortens.
- Type II avulsions have retracted to the level of the PIP joint. Blood supply remains. These injuries can be repaired even after a delay of up to several weeks, although an earlier repair is favorable.
- Type III avulsions usually are attached to a large fracture fragment that prevents the tendon from retracting past the DIP joint. These injuries can be repaired in a delayed fashion similar to type II injuries.
Note: Urgent surgery is recommended in acute injuries because determination of the type of avulsion from radiographs and clinical examination is inexact.11

Ultrasonography or magnetic resonance imaging can help differentiate the type of injury if necessary.

Zone II (No Man’s Land)
Repair Technique

- Repair both tendon lacerations in zone II.
- In one retrospective study, 74% of digits with both tendons repaired had good or excellent results compared with 47% of digits with only the profundus tendon repaired.12
- Four-strand repair with epitendon repair (see Figure 12–5).
- Strickland modification of the Kessler repair is performed using two sets of core sutures for the FDP tendon.

- An epitendon suture is placed routinely regardless of vincula attachments or lack thereof.
  ❖ Note: By placing the dorsal sutures of the epitendon repair (leaving the 6–0 Prolene suture needle attached loosely to the side to enable completion of this suture) before the core sutures are tied, proper circumferential placement of the epitendon sutures are facilitated, thus ensuring the tendon ends come together evenly without bunching.
  - Once the dorsal epitendon sutures are placed, the core sutures are placed. The outer core sutures are tied and then the inner core suture is tied. The remainder of the epitendon repair is completed volarly.
- The slips of the FDS tendon are repaired with simple Kessler sutures or mattress sutures.
- If enlargement of a pulley is desirable, an L-shaped incision can be made in the tendon sheath distally. The sheath will have a funnel shape, which helps accommodate the tendon end.
- If the proximal ends of the tendons have retracted into the tendon sheath and cannot be retrieved, a counter incision is made at the level of the distal palmar crease. In most cases, the tendon can be gently pushed distally with a forceps so that the ends can be retrieved from the incision in the finger.
- If necessary, a no. 5 pediatric feeding tube or silastic tube can be passed from proximal to distal and sutured to the side of the tendon to pass the tendon distally.3
- Occasionally, severe retraction of the tendon prevents distal passage of the tendon until a core suture is placed into the tendon as a means of passing the tendon distally.

Zone III Injuries

- The tendons are repaired using the same suture techniques as described earlier. The exposure of the tendons is much easier and the results are much better because of the absence of the fibroosseous sheath at this level.
Zone IV Injuries

- The tendons are repaired as described earlier, along with any injuries to the median nerve that is superficial to the tendons.

Zone V Injuries

- Injuries near the musculotendinous junction can be difficult to repair because the muscle tissue will not hold a suture. Often multiple mattress sutures are necessary if the musculotendinous junction will not hold a core suture.

Flexor Pollicis Longus Injuries

- These injuries are treated with repairs similar to the finger tendons.
- All efforts are made to preserve the oblique pulley and the A2 pulley.
- To retrieve the FPL tendon if it has retracted proximally, an incision at the level of the wrist (through the flexor carpi radialis sheath) is recommended rather than in the thenar region, where the tendon dives between the thenar muscles. Take care to avoid injuring the thumb’s digital nerves, the median, and palmar cutaneous nerves.

Rehabilitation of Flexor Tendon Injuries

- The key to successful flexor tendon repair is close adherence to a regimented hand therapy rehabilitation program.
- Various protocols following flexor tendon repair are available. Each protocol must consider the stress placed on flexor tendons before and after the repair.
- Active assisted range of motion.
- Passive range of motion (Kleinert et al. vs. Duran) (Box 12–2).
- Immobilization is recommended for children younger than 10 years and for patients unable or unwilling to follow a controlled-motion protocol. Immobilize with the wrist in 10 degrees of flexion, MP joints blocked in 70 degrees flexion, and IP joints neutral for 4 to 6 weeks based on the patient’s maturity level and the strength of the initial repair. If the patient is immobilized for only 4 weeks, a removable splint in similar position is used for an additional 2 weeks.

○ Early active motion protocol
  - Early active motion protocols are indicated for intelligent, compliant patients with a solid four-strand repair who can comply with hand therapy on a regular basis for zone I or II injuries.
  - In theory, active motion allows for better overall motion because greater tendon gliding prevents adhesion formation.
  - Concern exists regarding higher rates of tendon rupture with early active motion in noncompliant patients.

○ Passive motion protocol (see Box 12–2)
  - Passive motion protocols are better suited for patients with a two-strand repair and for less compliant patients.
  - Zone III, IV, and V injuries are treated with the passive tendon protocol.

Passive Flexor Tendon Protocol

0–3 Weeks Postoperative Regimen

- No active finger flexion.
- Do not remove the splint except under the supervision of the therapist.
- Do not pick up objects with the injured hand.

Splinting

- Fabricate a dorsal extension block splint with the wrist in 10 degrees flexion, MP joints blocked in 70 degrees flexion, and IP joints neutral (Figure 12–9).

Box 12–2 Passive ROM Protocols

<table>
<thead>
<tr>
<th>Kleinert Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevents patients from moving their digits against resistance.</td>
</tr>
<tr>
<td>Maintains the digits in a protective position.</td>
</tr>
<tr>
<td>Requires that the patient’s injured digits be maintained in flexion by using an elastic band that is attached to the level of the wrist.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Duran Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses a protective splint but no elastic bands (Duran and Houser, 1975).</td>
</tr>
<tr>
<td>Passive flexion achieved by the therapist or with use of the uninjured hand.</td>
</tr>
<tr>
<td>Decreases incidence of flexion contractures at the PIP joint.</td>
</tr>
</tbody>
</table>

Figure 12–9: Dorsal block splint used for protection after flexor tendon repair. (From Trumble TE, Sailer SM: Flexor tendon injuries. In Trumble TE, editor: Principles of hand surgery and therapy. Philadelphia, 2000, WB Saunders.)
Treatment

- Passive flexion using the uninvolved hand, active extension exercises while wearing the splint.
- Passive extension exercises of individual joints performed in guarded positions (all joints flexed except the joint being extended), that is, flex the DIP and MP joints when working on PIP extension.
- Edema management with elevation and compressive wrap (Coban), if needed.

3–6 Weeks Postoperative Regimen

Splinting Changes

- Bring the wrist position in the splint to neutral at 3 weeks.
- Can strap the PIP joints into progressively greater extension within the splint if flexion contracture occurs.

Treatment

- Continue passive flexion/active extension exercises in the splint.
- Start “place and hold” exercises by passively placing the injured digit in flexion, then asking the patient to voluntarily contract the muscle in an effort to hold the finger in flexion while releasing the pressure from the hand applying passive flexion.
- Begin gentle soft tissue mobilization for scarred areas.

6–9 Weeks Postoperative Regimen

Splinting Changes

- Wean from the dorsal extension block splint as patient reliability permits.

Treatment

- Begin active blocking exercises for DIP and PIP flexion to facilitate differential tendon glide.
- Begin light functional activities (e.g., picking up handfuls of beans or rice, stacking small blocks, picking up lightweight objects of varying sizes requiring different prehension patterns).

9–12 Weeks Postoperative Regimen

Treatment

- If stiff, begin static-progressive splinting to correct joint contracture.
- Continue active exercises (full active flexion and extension).
- Continue blocking exercises for DIP and PIP flexion.
- Begin resistive exercises and progress as follows:
  - Light functional activities at 6 weeks (picking up handfuls of beans/rice).
  - Light resistance at 8 to 10 weeks (squeezing soft foam or soft putty).

12–16 Weeks Postoperative Regimen

Precautions

- No heavy lifting.

Treatment

- Continue active exercises and blocking exercises, as needed.
- Progress to full resistive activities.
- Work conditioning/hardening, if needed.

16 Weeks Postoperative Regimen

Precautions

- None.

Treatment

- Continue for residual deficits until resolved or patient plateaus.

Early Active Motion Protocol

- Note: The following is based on the Indianapolis protocol by Strickland.3

Initial 24–48 Hours Postoperative Regimen

- Postoperative dressing is removed.
- A custom dorsal protective splint is fabricated in the standard position (wrist 10 degrees flexion, MPs 60–70 degrees flexion, IPs neutral) (see Figure 12–9).
- Passive PIP, DIP, and composite joint flexion and extension are started within the dorsal block splint.
- Active extension of the PIP and DIP joints to the limits of the dorsal blocking splint is allowed.
- Compressive wraps or sleeves are used to help decrease edema.

24–72 Hours Postoperative to 4 Weeks Postoperative Regimen

- A hinged tenodesis splint is fabricated, permitting wrist position to be varied (wrist motion limited to 30 degrees extension with unrestricted flexion, MPs in 60–70 degrees flexion, IPs neutral).
- Instruction on active “place and hold” exercises in the tenodesis splint:
  - Within the tenodesis splint, the wrist is passively brought into 30 degrees of extension while the fingers are placed into full composite flexion.
  - The patient gently contracts the finger flexors and attempts to hold the flexed position for 5 seconds.
- After each session, the patient returns to the dorsal block splint.
4 Weeks Postoperative Regimen

- The same place and hold exercises that were performed in the tenodesis splint are now performed without the guidance of the splint.
- The fingers are passively flexed with the wrist extended. Flexion of the MP joints with the wrist extended helps decrease the force on finger flexor tendons.
- Active movement from a full fist, to a hook fist, to a straight fist, to full finger extension is used to facilitate maximum tendon gliding.

6 Weeks Postoperative Regimen

- Dorsal blocking splint is discontinued.
- Active finger flexion exercises with joint blocking at both the PIP and DIP joints are added to facilitate tendon gliding.
- Buddy taping can be used to facilitate full flexion.
- Note: Blocking exercises to the small finger are not recommended.

8–9 Weeks Postoperative Regimen

- Light strengthening exercises (e.g., squeezing soft foam ball) are initiated.
- Soft putty can be used for strengthening.

10–14 Weeks Postoperative Regimen

- Progressive resistive strengthening program is initiated.
- Work simulation and reconditioning may be necessary in cases where severe deconditioning precludes return to work in some occupations.
- Return to full unrestricted activity is allowed at 14 weeks.

Conclusion

- Successful results require precise surgical technique and strict adherence to a rehabilitation program.

References

   Steenb Bunnell further elaborates on his classic article from 1918, discussing atraumatic technique, the need to preserve the pulley system, and the need for postoperative therapy.

   Growth factors are present in normal canine intrasynovial flexor tendons.

   The author provides an excellent overview of tendon healing and methods for tendon repair emphasizing a protocol for four-strand repair and early active motion.

   Following cyclic loading, two-strand repairs had significantly greater gap formation after cyclic loading than either four-strand or six-strand repairs. The tensile strength of the six-strand repair was significantly greater than either the four-strand or two-strand repair.

   Dorsal placement of the core suture in tendon repair is stronger than palmar suture placement.

   Volar location of suture material significantly increased the work of flexion following tendon repair, compared to a dorsal location.

   A method for evaluating flexor tendon repair techniques with use of cyclic testing is presented.

   There was no significant difference between the results of sheath repair and leaving it open following flexor tendon repair in zone II.

   The relative effects of immobilization, early protected mobilization, tenorrhaphy, and no repair of partial flexor tendon lacerations were evaluated in a non–weight-bearing canine model.

    A discussion of avulsions of the flexor profundus tendon insertion.

    Although the classification of Leddy and Packer is very helpful in determining the prognosis for avulsion fractures of the flexor digitorum profundus, the tendon may pull off the bone and retract farther than suggested by the fracture pattern. Therefore, all flexor digitorum profundus tendon avulsions should be surgically repaired as soon as possible.

“No man’s land” of the flexor tendon system is divided into four subdivisions based on an anatomic study. Comparison of treatment results in each subzone reveals the IIc subzone is the most difficult area for satisfactory functional recovery.

The authors present their results of early passive rehabilitation following zone II flexor tendon repairs. The authors use elastic bands to passively flex the injured digits.

Active and passive muscle tension is discussed in relation to finger flexor and extensor tendons.
CHAPTER 13

Extensor Tendon Repair
and Reconstruction

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Introduction and Anatomy

- The extensor mechanism of the hand and digits is a balance between intrinsic and extrinsic forces and is easily disrupted.
- Two thirds of all acute extensor tendon lacerations are associated with concomitant bone, skin, or joint injuries. Wound debridement, rigid internal fixation, bony healing, repair of neurovascular structures, and skin coverage all take precedence over extensor tendon repair.
- Chronic disruption of the extensor mechanism frequently can be addressed by attempting to rebalance forces that contribute to the extensor mechanism.

Extrinsic Extensor Anatomy

- All of the extrinsic extensor muscles for wrist, thumb, and finger extension are innervated by the radial nerve. The brachioradialis, extensor carpi radialis longus, and extensor carpi radialis brevis are innervated directly by the radial nerve proper. The rest of the extensors are innervated by a branch of the radial nerve, the posterior interosseous nerve.
- There are six dorsal compartments or fibroosseous tunnels at the wrist created by the extensor retinaculum to prevent bowstringing during wrist extension (see Chapter 1).
- There are two tendons to the index and small fingers. The extensor indicis proprius tendon is usually found ulnar to the EDC tendon in the index finger but variations can occur in this relationship. After loss of extensor indicus proprius, independent index finger function usually is preserved via the extensor digitorum communis (EDC) tendon. However, the extensor digitorum communis tendon to the small finger is present as a complete structure only 20% of the time. Therefore, loss of extensor digiti minimi (EDM) commonly results in loss of full extension of the small finger.
- Juncturae tendineae interconnect the extensor digitorum tendons and the EDM tendon. These juncturae are important for force redistribution, tendon spacing, and coordination of extension. A laceration to the juncturae can result in subluxation of the extensor tendon over the metacarpophalangeal joint (MCPJ) into the radial or ulnar gutter. Lacerations to EDC tendons can be masked by intact juncturae bridging between tendons. The adjacent intact extensor tendon can extend the finger with a lacerated EDC if the juncturae to the lacerated tendon is intact distal to the laceration (Figure 13-1). Over time the juncturae weaken and an extensor lag can present several weeks after the initial laceration.

Extensor Mechanism at the Level of the Digits

- The intrinsic muscles of the hand are intricately connected to the extensor mechanism. The sagittal bands at the level of the MCPJ centralize the extensor tendon and attach to the volar plate and periosteum of
interphalangeal joint (DIPJ). The lumbrical and interosseous tendons pass volar as flexors at the MCPJ and dorsal as extensors at the proximal interphalangeal joint (PIPJ) (Figure 13–3).

- The oblique retinacular ligament ([ORL] Landmeer ligament) crosses volar to the PIPJ and dorsal to the DIPJ. When the PIPJ is extended, the ORL tightens, which assists DIPJ extension, that is, coupling PIPJ extension to DIPJ extension (see Figure 13–3).

### Zones of Injury to the Extensor Tendon and Mechanism

- The extensor mechanism is divided into nine zones to facilitate discussion of injury and treatment of acute injuries. Eight zones are commonly used and are discussed here. The odd numbered zones are over joints and the even numbered zones are over bones (Figure 13–4, Table 13–1).

- In zones I through VI, tendon nutrition is via perfusion through the paratenon. In zone VII, a tenosynovium provides tendon nutrition. Zones VIII and IX are fed by small arterial branches from surrounding fascia.

### Evaluation and Diagnosis

- Bony assessment and determination of neurovascular status and flexor tendon function are essential. Thorough wound inspection may reveal joint capsule injury, foreign bodies, and partial tendon lacerations not identified on clinical examination.

- When examining the hand for extensor tendon injury, the wrist should be in neutral position. Examine each finger individually with the adjacent fingers flexed at the MCPJs. This position eliminates the pull of juncturae.
from the adjacent extensor tendons, which could mask an isolated extensor tendon laceration. Other signs of extensor tendon laceration are loss of hyperextension at the MCPJ, extensor lag of the finger, and relative weakness and pain in one finger compared with the other fingers (Box 13–1).

Suture Technique

● Extensor tendons have less excursion than flexors, so minimal shortening can result in significant loss of motion. Six millimeters of shortening results in a loss of 18 degrees of motion at the MCPJ and PIPJ. Modified Kessler and modified Bunnell techniques provide the best repair strength, minimal gapping, and least loss of motion (Figure 13–5). Nonabsorbable 4-0 or 5-0 sutures are acceptable for zones I through VI. Lacerations through the extensor tendon in zone I can be repaired with a nonabsorbable suture passed through tendon and skin (tenodermodesis). The DIPJ also must follow the mallet finger protocol. Lateral bands should be repaired separately with nonabsorbable 5-0 or 6-0 suture. Zone VI and VII are repaired with 5-0 or 6-0 suture, and zone VIII is repaired with 3-0 suture.

Zone I Injury: Mallet Finger

● Classic mallet finger involves disruption of the extensor mechanism at the DIPJ. The mechanism is forced flexion of an extended DIPJ. This action can result in tendon rupture, avulsion from its insertion, or bony avulsion (bony mallet). Laceration to the extensor tendon is less common.
● Four types of acute mallet finger deformities have been described (Table 13–2). Treatment depends on patient age, mechanism of injury, associated fractures or osteoarthritis, and chronicity of the injury.
● Type I injuries are treated with splinting for 6 to 8 weeks (Table 13–3).
● Type II injuries should be primarily repaired at the time of laceration with a figure-of-eight suture of nonabsorbable material. Sutures can incorporate the overlying skin for dermatotenodesis.
● Type III injuries require soft tissue coverage of the injury, often a reverse cross-finger graft or other local graft.
● Types IVa and IVb should be reduced and splinted until the fracture heals. Appropriate reduction implies restoration of joint congruency and realignment of articular surface and contour.
● Type IVc with subluxation of the distal phalanx requires open reduction via a dorsal H-shaped incision and internal fixation with a Kirschner wire or 1.0-mm screw. Sometimes a pullout suture or wire provides the best fixation.

<table>
<thead>
<tr>
<th>Table 13–1: Extensor Zones of Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zone I</td>
</tr>
<tr>
<td>Zone II</td>
</tr>
<tr>
<td>Zone III</td>
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<tr>
<td>Zone IV</td>
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<tr>
<td>Zone V</td>
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<tr>
<td>Zone VI</td>
</tr>
<tr>
<td>Zone VII</td>
</tr>
<tr>
<td>Zone VIII</td>
</tr>
</tbody>
</table>
Therapeutic Regimen

- Type I injuries should be immobilized for 6 to 8 weeks in extension, followed by 6 to 8 weeks of nocturnal splint use and slow daytime weaning from the splint (Figure 13–6). During this time, full active and passive motion of the PIPJ is achieved. If this regimen is interrupted or if deformity recurs, the protocol should be restarted from the beginning. The patient must be reminded that a brief loss of full extension at the DIPJ disrupts whatever healing has begun and restarts the clock for splinting. Attention to the skin on the dorsum of the finger during this splinting period is important because the skin can become macerated and irritated. The best form of splinting is a padded Alumafoam splint contoured to hold the DIPJ in hyperextension. This splint is taped to the dorsum of the finger with cloth tape. For the first few weeks, the patient can return to the clinic for splint changes. The patient should be reminded of the importance of keeping the finger extended 100% of the time and taught how to change the splint and watch for skin breakdown. Stack splints have been used successfully because they hold the finger in hyperextension nicely. Dorsal skin maceration is more problematic with these splints because of direct contact of the plastic on the skin. Another option for maintaining the finger in extension is to place a transarticular K–wire for the first 6 to 8 weeks if compliance or skin irritation is a problem.

Table 13–2: Mallet Finger Classification

<table>
<thead>
<tr>
<th>TYPE I:</th>
<th>MALLET FINGER CLASSIFICATION</th>
<th>TYPE II:</th>
<th>TYPE III:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Closed soft tissue disrupting due to hyperflexion injury</td>
<td>Laceration at dorsum of distal interphalangeal joint</td>
<td>Open hyperflexion injury with deep skin abrasion or tissue loss</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TYPE IVA:</th>
<th>TYPE IVB:</th>
<th>TYPE IVC:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transphyseal injury in skeletally immature digit</td>
<td>Hyperflexion injury involving 20–50% of articular surface</td>
<td>Hyperextension injury involving &gt;50% of articular surface</td>
</tr>
</tbody>
</table>

*Results in early or late volar subluxation of distal phalanx
● Chronic mallet fingers up to 3 months old can be treated following the splinting protocol (see Table 13–3). If the deformity persists, tendon advancement is indicated for flexible deformities, and arthrodesis is recommended for rigid deformities.

● Swan-neck deformity develops in chronic mallet fingers because the lateral bands migrate proximally and dorsally, causing hyperextension at the PIPJ (Figure 13–7). The spiral ORL can be used to restore balance or a superficialis tenodesis can be used to prevent hyperextension. Many swan-neck deformities are treated with long-term splinting.

### Zone II Injuries

● Most injuries to the extensor mechanism in zone II result from lacerations and crush injuries. Lacerations distal to the insertion of the central slip result in a mallet deformity. Partial lacerations less than half the width of the tendon can be treated with wound closure and splinting in extension for 7 to 10 days, followed by active range of motion. Lacerations greater than 50% of the tendon width should be repaired with nonabsorbable suture in running fashion to minimize tendon shortening.

### Therapeutic Considerations

● Zone II injuries can follow the same splinting protocol as zone I injuries (see Table 13–3). Gradual DIPJ flexion exercises begin with 20 to 25 degrees in the first week and progress 10 degrees each week. If the distal joint is tight in extension, the ORL can be stretched by flexing the DIPJ with the PIPJ in extension.

### Zone III Injuries: Boutonnière Deformity

● Disruption of the central slip allows the lateral bands to subluxate volarly and flex the PIPJ while extending the DIPJ (Figure 13–8).

● In the acute setting, the inability to extend the PIPJ against resistance with the joint flexed at 90 degrees

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**Table 13–3: Mallet Finger Extension Splinting Protocol**

<table>
<thead>
<tr>
<th>MALLET FINGER</th>
<th>EXTENSION SPLINTING PROTOCOL (TYPES I, II, III, AND IVB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 6–8 weeks</td>
<td>Immobilize DIPJ in full extension/hyperextension, and leave the proximal interphalangeal joint free for active motion. Take care not to move the DIPJ when changing the splint.</td>
</tr>
<tr>
<td>Second 6–8 weeks</td>
<td>Begin weaning from the splint during the daytime. Continue immobilization at night.</td>
</tr>
<tr>
<td>After 6–8 weeks</td>
<td>Accept residual deformity up to 15 degrees flexion at the DIPJ. Repeat protocol from beginning if &gt;15 degrees.</td>
</tr>
<tr>
<td>Splint care and changing splint</td>
<td>Instruct patient to monitor dorsal skin where splint rests for irritation and maceration. When changing splint, the DIPJ must be kept in extension.</td>
</tr>
<tr>
<td>Other treatments</td>
<td>Type IVa (pediatric) may require transarticular pinning for compliance. Type IVc requires reduction internal fixation.</td>
</tr>
</tbody>
</table>

DIPJ, Distal interphalangeal joint.

---

**Figure 13–6:**
Extension splinting for mallet finger leaves the proximal interphalangeal joint free to flex but keeps the distal interphalangeal joint in full extension. (From Trumble TE: Principles of hand surgery and therapy. Philadelphia, 2000, W.B. Saunders.)

**Figure 13–7:**
Swan-neck deformity from proximal migration of the lateral band complex after disruption of the distal extensor mechanism. The lateral bands move proximally into a more dorsal position and contract, which pulls the proximal interphalangeal joint into extension. (From Trumble TE: Principles of hand surgery and therapy. Philadelphia, 2000, W.B. Saunders.)
suggests central slip avulsion or rupture. Active extension of the DIPJ while in the same position also suggests acute central slip disruption. This test was described by Elson and, if carefully performed, is a reliable way of evaluating an acute boutonnière deformity (Figure 13–9, Box 13–2). Pain may limit patient cooperation, and a digital block improves the success of the test.8

- Chronic boutonnière deformities usually involve subluxation of the lateral bands with scarring and adhesions to the underlying capsule. The finger is less supple and the deformity more difficult to passively reduce. Boyes described a test to identify fixed subluxation and adhesions of the lateral bands. The finger is held in extension at the PIPJ, and the patient is asked to flex the DIPJ. When the lateral bands have been in a relatively shortened, volarly subluxated position and are scarred down, PIPJ extension increases the tension in the lateral bands. Therefore, the DIPJ is held tightly in extension, and the patient cannot actively flex this joint (Figure 13–10, Box 13–3).8

### Treatment of Acute Boutonnière Deformity9

- Closed acute central slip injuries are treated with continuous splinting of the PIPJ in extension for 6 weeks. The DIPJ is left free, and active and passive DIPJ flexion are encouraged to keep the lateral bands and ORL supple.
- Surgical indications for an acute boutonnière deformity are a displaced avulsion fracture of the central slip at the base of the middle phalanx, instability of the PIPJ associated with loss of active or passive extension of the finger, or failed nonoperative treatment. Open central slip injuries require thorough debridement and evaluation of the underlying joint. Both primary closure and healing secondary intention have been described because the central slip tendons do not retract and the scar tissue can bridge the defect if constant splinting is maintained for four to six weeks. Active DIPJ and MCPJ motion is essential while holding the PIPJ in extension during healing. DIPJ motion stretches the lateral bands and keeps them dorsal to the PIPJ. Tension on the lateral bands also reduces the tension on the central slip and facilitates healing.
- Primary suture repair of the central slip to the middle phalanx may be possible; if not, portions of the lateral bands can be sutured together dorsally to recreate a central slip. If the lateral bands cannot be moved dorsally, the Matev procedure, which consists of transecting the lateral bands and suturing one to the remnant central slip tissue and cross suturing the remaining lateral band, can be used (Figure 13–11).

### Treatment of Chronic Boutonnière Deformity9

- Treatment is predicated on first restoring passive motion at the DIPJ and PIPJ with therapy and dynamic splinting. Surgical excision of redundant central slip and
reconstruction of the slip with appropriate extensor tension then can be achieved. Transarticular K-wires are helpful in maintaining extension at the PIPJ while moving the DIPJ and MCPJ.

- In a supple PIPJ with a Boutonnière deformity, transcription of the central portion of the extensor tendon at the middle phalanx and partially transecting the lateral bands has been described. This procedure allows the extensor mechanism to migrate proximally, moving the lateral bands dorsally and rebalancing the PIPJ. The ORL must be protected during this release to maintain extension at the DIPJ (Figure 13–12).

- When motion cannot be achieved by therapy and splinting, a two-stage procedure can be performed. First, the volar structures are released to allow motion. Then a second procedure is performed to reconstruct the central slip. Tenotomy or partial transection of the distal tendon may allow motion at the DIPJ while leaving contracture at the PIPJ alone. The lateral bands can be partially divided, mobilized dorsally, and attached to the dorsum of the middle phalanx to provide PIPJ extension.

**Therapeutic Considerations**

- Treatment of closed acute Boutonnière deformities consists of extension splinting of the PIPJ for 6 weeks and repeat splinting if the deformity recurs. The surrounding joints are left free for active full motion during splinting (Figure 13–13).

- After open repair of central slip injuries, the PIPJ is immobilized in extension for 3 to 6 weeks before gradual motion is started. If the lateral bands were not repaired, the DIPJ can be left free to flex and stretch the ORLs.

**Zone IV Injuries**

- Partial lacerations are treated with splinting the MCPJ, PIPJ, and DIPJ in extension and the wrist in a neutral position for 3 to 4 weeks. Complete lacerations should be repaired, and associated phalangeal fractures should be internally fixed to allow early active range of motion. Restoration of length and appropriate rotation is important. If proximal phalangeal fractures are allowed to shorten, the extensor mechanism is effectively elongated at the proximal phalanx, leading to an extensor lag at the PIPJ. Dissection should be limited and care taken in handling soft tissues to minimize scarring.

**Therapeutic Considerations**

- After repair of zone IV lacerations, tendon-bone adhesions frequently prevent tendon gliding. Traditionally, the MCPJ, PIPJ, and DIPJ are splinted in extension with the wrist in a neutral position for 4 to 6 weeks. To prevent dorsal hood adhesions, an early motion protocol has been suggested. The PIPJ and DIPJ are splinted in extension, except during exercises. The exercises involve flexing the wrist to 30 degrees and the MCPJ is kept at 0 degrees. The PIPJ is slowly actively flexed to 30 degrees and the DIPJ to 20 to 25 degrees for 20 repetitions. This
Figure 13–11: Matev procedure for treatment of boutonnière deformity. One lateral band is divided and sutured to the remnant central slip stump on the middle phalanx. The other lateral band is divided, crossed, and sutured to the opposite lateral band.

Figure 13–12: Fowler procedure for chronic boutonnière deformity. The central tendon and lateral bands are divided just proximal to the distal interphalangeal joint, leaving the oblique retinacular ligament intact. This procedure allows the extensor mechanism to migrate proximally, release tension from the lateral bands, and facilitate flexion at the PIPJ.

Figure 13–13: Splinting for boutonnière deformity holds the proximal interphalangeal joint in extension but leaves the distal interphalangeal joint free to flex. The Bunnell rigid extension splint (B) is used for the first 3 to 6 weeks. The patient then can be placed in a Capener dynamic extension splint (C), which allows active proximal interphalangeal joint flexion and passive extension. The patient is weaned to nighttime splint use at 8 to 11 weeks, and then the splint is discontinued.
process may prevent adhesions that ultimately result in poor tendon gliding in zone IV.

**Zone V Injuries**

- The “fight bite” is an open wound overlying the MCPJ that occurred with the finger in flexion. The wound can involve the underlying extensor mechanism and even can extend into the joint. Appropriate debridement and assessment of the depth of the wound are imperative to prevent infections such as septic arthritis and osteomyelitis. The wound should be examined with the fingers in the same position when the wound occurred. This action facilitates visualization of the underlying extensor mechanism, joint capsule, and even the joint if the wound extends to that depth. Thorough irrigation of the maximal depth of the wound is required, including intraarticular irrigation if the joint is involved. Wound cultures should be sent, broad-spectrum antibiotics started, and the hand splinted for a few days before starting motion again. If pain persists, operative debridement of the wound and joint may be required.
- Rupture of the sagittal bands presents as a painful snapping at the MCPJ as the extensor tendon subluxates either radially or ulnarly as the finger is brought from full extension into flexion. Splinting in extension with the wrist centralized for 6 weeks usually is sufficient. If the problem presents late or persists after splinting, surgical repair of the sagittal band with a flap of extensor tendon used to reinforce the repair allows early active motion.

**Therapeutic Considerations**

- Protocols exist for complete immobilization, early passive motion, and immediate active motion. Clinical studies show that wrist tenodesis exercises with active extensor motion result in greater range of motion without significant rupture rates. With the wrist flexed to approximately 20 degrees, the patient can actively hold the fingers in extension and then actively move the MCPJ from 30 degrees flexion to 0 degrees. Wrist tenodesis exercises are used to allow passive tendon gliding. Ideally, this protocol is started within 24 to 36 hours after surgery. Dynamic splinting with the MCPJ in extension can also be very useful.

**Zone VI Injuries**

- Juncturae injuries frequently are missed. Exploration of wounds is the best method for diagnosing and treating these lacerations. Proximal tendon lacerations frequently retract, and repair should be done using four-strand core suture technique (Figure 13–5). Splinting of all of the fingers from DIPJ to wrist in extension protects EDC repairs. Splinting the index finger alone in extension is acceptable after repair of the extensor indicis proprius (EIP) tendon. Dynamic splinting as in zone V injuries can be very helpful.
- If multiple extensor tendons are injured and there is inadequate length and tendon quality for primary repair, then the more damaged tendons can be repaired end to side to the less injured adjacent tendons. If there is complete loss of tendon and no adjacent tissue is adequate, tendon rods can be placed while the soft tissue bed heals. Then flexor to extensor transfers are used to restore extensor function.

**Therapeutic Considerations**

- The controlled active motion protocol for zone V also can be used for zone VI injuries. Immobilized tendons lose strength over time, whereas controlled motion improves the tensile strength of the tendon, improves gliding properties, increases repair-site DNA, and accelerates changes in the surrounding vascularity. Controlled early active tension and motion promotes gliding motion without placing stress at the repair site. Dynamic splints are designed to allow active flexion and thus gliding of the extensor mechanism. Many patients inadvertently actively extend through the slings, which promotes tendon healing.

**Zone VII Injuries**

- Laceration to extensor tendons at the level of the retinaculum presents a complex problem. The tendons retract significantly, scar under the retinaculum after repair, and limit finger flexion because of loss of extensor excursion. Partial retinaculum release is required, but complete release results in bowstringing.
- Injuries to the sensory branches of the radial nerve at this level should be addressed with either epineurial repair or resection of the proximal nerve end and burial in surrounding tissue to prevent neuroma formation.

**Therapeutic Considerations**

- Immobilization of tendon repair at the wrist leads to scarring and adhesions and should be limited to a short period. The wrist should be immobilized in 10 to 20 degrees extension and early tenodesis exercises started. Active wrist motion from 0 degrees to full extension can begin by 3 to 4 weeks. By 5 to 6 weeks, gradual wrist flexion is started, progressing slowly over the next 2 to 3 weeks to full flexion. When the extensor retinaculum has been disrupted, the extensor tendons bowstrings and increases the work load across the repair site. Slower advances in the protocol may be necessary to protect the repair.

**Zone VIII Injuries**

- Wrist and thumb extension should be priorities when sorting out multiple extensor lacerations in zone VIII.
Appropriate incisions and exploration are necessary to identify both proximal and distal tendon ends. Muscle bellies can be repaired with multiple figure-of-eight sutures. Laceration to the posterior interosseous nerve should be repaired because the distance to regenerate to the neuromuscular junction at this level usually is short.

**Therapeutic Considerations**

- Splint the wrist in extension and the MCPs in 15 to 20 degrees flexion for 4 to 6 weeks. After immobilization, mobilization is started with both active extension and flexion.

**Extensor Injuries to the Thumb**

- Zone T1 injuries are treated similar to mallet injuries in the other fingers by splinting, primary repair in lacerations, and bony fixation with K-wires or screws where more than 50% of the joint is involved.
- Injury to the extensor pollicis brevis is uncommon but is associated with avulsion of the dorsal capsule and radial collateral ligament complex. Cast immobilization is appropriate early, but persistent laxity should be treated surgically.
- Rupture of the extensor pollicis longus (EPL) tendon following a distal radius fracture is well described. Proposed mechanisms of rupture are attritional rupture secondary to fracture fragments, ischemia, or hemorrhages within tendon sheath. Delayed diagnosis is common, with the patient presenting 6 to 8 weeks after fracture with the inability to extend the thumb and resting in an adducted position. Free tendon grafting often is required using an intercalated graft or by transferring EIP to EPL.

**Therapeutic Considerations**

- For zone T1 injuries, follow the mallet finger protocol (see Table 13–3). Zones TII to IV, early active tensioning, and motion exercises are recommended. Zone V at the wrist involves the extensor retinaculum and a synovial sheath. Adhesions form if the tendon is immobilized, and early dynamic gliding exercises or controlled active motion is important.

**Complications of Extensor Tendons**

- Complex injuries usually include soft tissue damage, tendon injury, nerve injury, and even fractures or other bony injuries. Soft tissue management and fracture care are priorities. Tendon function often is impaired by scarring from associated soft tissue injuries. Early postoperative motion helps decrease adhesions from scarring and improve tendon gliding and function. Tenolysis may be required in more complex injuries to facilitate better range of motion and function.

**References**

**Anatomy**

   The extensor anatomy, including anomalous tendons and multiplicity, is described in detail.
   Of 72 cadaver hands, 19% showed anatomic variation from the classic single slip of the EIP lying ulnar to the EDC.

**Suture Technique**

   Biomechanical testing on 16 fresh-frozen cadaver hands found modified Bunnell and modified Kessler techniques superior to the mattress and figure-of-eight techniques used for extensor tendon repairs.

**Mallet Finger**

   The authors tested 32 cadaveric fingers with open mallet finger lesions and found that joint motion proximal to the DIPJ did not cause a tendon gap.
   After conservative treatment of 31 patients with mallet finger deformities, patient satisfaction was high, and there was little evidence of functional impairment despite high rates of arthritis and extensor lag.
   This retrospective review found results of conservative treatment of mallet fingers were independent of time from injury to treatment, even up to 8 months.

**Boutonnière Deformity**

A treatment algorithm for all types of boutonnière deformities is presented in this in-depth review.

**Rehabilitation**

   The authors report good results with an immediate motion and tendon mobilization program with dynamic splinting for extensor tendon repairs.

   This chapter provides a review of therapeutic regimens and a comparison of outcomes for each regimen. Considerations of specific anatomy, associated injuries and patient factors are included to help guide a complete therapeutic plan.

   This randomized, prospective study comparing early active motion with dynamic splinting for extensor tendon repairs in zones IV to VIII found no significant differences between the two groups at final follow-up.

**Zone VII Injury**

   Discussion of bowstringing at the wrist after division of the extensor retinaculum.

**Review Articles**

   This review article covers the basic anatomy, initial evaluation, treatment, surgical technique and rehabilitation of extensor tendon injuries.

   This comprehensive review article describes the anatomy of the extensor tendons, the acute and chronic pathologic conditions affecting the extensor mechanism, the physiology and repair techniques of traumatic injuries, and the reconstructive options for chronic disorders.
Introduction

- The term tendonitis is often used in clinical practice to describe the inflammatory process involving one or more of the numerous tendons that cross the wrist and the hand. Although this term might be accurate in the early stages, with more chronic lesions the synovial sheath of intrasynovial tendons almost invariably is involved, and the term tenosynovitis or tenovaginitis is more appropriate.
- The synovial sheath is a membranous structure with an inner visceral layer and an outer parietal layer that plays an important role in intrasynovial tendon gliding and nutrition by diffusion. This membranous structure is reinforced locally by outer retinacular components (as are the digital pulleys or the extensor retinaculum) to enhance function. When the membranous sheath becomes inflamed and edematous, the retinacular components prohibit its expansion, and thus the tendons are locally constricted. With chronicity, local ischemia leads to secondary changes to the sheath and tendon. The sheath becomes fibrotic and even can undergo cartilaginous metaplasia, whereas the tendon is locally thinned and fibrotic. These pathologic changes are encountered in the majority of the disorders discussed in this chapter.
- The primary etiology of most of these disorders is still unclear. Repetitive trauma can induce a local inflammatory process, but in most cases an anatomic or intrinsic predisposing factor must exist. Age and gender also are important factors because these conditions are seen more frequently in women in their 50s. Pregnancy is a predisposing factor, and the dominant extremity is more often affected.
- These conditions should be differentiated from more generalized inflammatory processes, as in rheumatoid arthritis. Less commonly, deposits as calcifications, crystals, and amyloid are the primary cause of tendosynovitis. The need to rule out septic tendosynovitis (although its presentation is markedly different than most of these disorders) cannot be overemphasized.

Trigger Finger

Anatomy and Pathology

- The digital flexor tendon sheaths are bilayer synovial linings supported and enclosed by a series of five annular
and three cruciate pulleys, the so-called retinacular sheath
(Figure 14–1). The thumb flexor sheath is enclosed by
two annular pulleys and one oblique pulley
(Figure 14–2).

- The proximal edge of the A1 pulley is near the distal
domal crease for the ring and small fingers, near the

Figure 14–1:
The digital flexor tendon sheath is enclosed and supported by
a series of five annular and three cruciate pulleys. The A2 and
A4 pulleys should be preserved to prevent tendon bowstringing.
The A1 pulley is most commonly involved in trigger
finger.

proximal palmar crease for the index finger, and midway
between the proximal and distal palmar creases for the
middle finger. The proximal edge of the first annular
pulley of the thumb lies near the metacarpophalangeal
(MP) joint flexion crease. The A1 pulleys and the first
annular pulley of the thumb arise at the level of the MP
joints. The A2 pulleys of the fingers are at the level of the
proximal portion of the proximal phalanges and the
oblique pulley of the thumb is at the middle region of
the proximal phalanx.

- The digital arteries are volar to the digital nerves in the
palm but become dorsal to the digital nerves in the
fingers. Both structures lie in close proximity to the
flexor sheath, paralleling the sheath on both its radial and
ulnar borders. The neurovascular bundles are at risk for
injury during surgery. The radial neurovascular bundle to
the thumb is the most at risk as it passes obliquely across
the thenar eminence from ulnar to radial and lies just
deep to the dermis at the MP joint flexion crease (see
Figure 14–2). It has been reported to be approximately
1 mm anterior to the radial sesamoid and 1 mm deep to
the dermis at the thumb MP flexion crease.

- Trigger digits occur because of a disproportion between
the digital retinacular sheath and its contents, the flexor
tendons and synovial sheath. The first annular (A1) pulley
is the usual site of obstruction in trigger fingers. The first
annular pulley is the usual source of obstruction in
trigger thumbs. The disproportion may be caused by the
angular course required by the flexor tendons as they
enter the retinacular sheath under the A1 pulley. Authors
have proposed that this angular course results in
“bunching” of the tendon fibers, leading to an
intratendinous nodule. This nodule occurs just distal to
the A1 pulley. In diffuse stenosing tenosynovitis resulting
from a systemic condition such as rheumatoid arthritis or
amyloidosis, the size discrepancy between the retinacular
sheath and its contents may extend well distal to the A1
pulley.

- Histologically, both the A1 pulley and the flexor tendon in
the region of the A1 pulley undergo fibrocartilaginous
metaplasia. The normal A1 pulley is composed of an
outer vascular layer and an inner gliding layer. In trigger
finger, the inner gliding layer hypertrophies, and the cells
increase in number, taking on the histologic appearance
of chondrocytes. The hypertrophy may progress until the
A1 pulley is two to three times its normal thickness. The
tendon undergoes fraying and degeneration on its volar,
avascular surface in the region where it passes under the
A1 pulley. Positive histologic staining for S-100 protein, a
protein associated with chondrocytes, indicates the
tendon undergoes fibrocartilaginous metaplasia, similar to
the A1 pulley.

- The pathologic process in infants usually consists of a
nodule (Notta node) within the flexor pollicis longus
tendon without hypertrophy of the first annular pulley.
Diagnosis

- Trigger digits, or stenosing tenosynovitis (tenovaginitis) of the digits, are one of the most common causes of hand pain and dysfunction. Primary (idiopathic) trigger finger occurs two to six times more frequently in women than in men and has a peak incidence between the ages of 40 and 60 years as well as being higher in patients with diabetes. Secondary trigger finger is associated with predisposing conditions such as rheumatoid arthritis, diabetes mellitus, gout, amyloidosis, and mucopolysaccharidoses. Multiple digit involvement is not uncommon, nor is bilateral involvement. The ring finger is most commonly involved, followed by the thumb and long finger, the index finger, and then the small finger.
- Patients often notice a painless click in the finger that eventually becomes painful. The pain, when present, frequently localizes to the MP joint and may radiate proximally. The digit may become locked in flexion, with passive extension required to unlock the digit, or locked in extension, with the patient unable to fully flex the finger. Chronic cases of locked trigger digits may result in fixed joint contractures, presenting a diagnostic challenge. Patients frequently attribute the problem to the proximal interphalangeal (PIP) joint.
- Physical examination often reveals a noticeable catching of the digit during active extension from a flexed position. Manipulation by the examiner may be required to extend a locked digit. The flexor sheath should be palpated for a discrete nodule or diffuse tenosynovitis, because this finding may have prognostic and treatment implications. The nodule usually is painful to palpation (Box 14–1).
- Most proposed classification systems divide trigger digits to one of five grades, based on the findings of the physical examination (Box 14–2).
- Trigger digit in children is a separate entity from adult trigger digit. Any digit may be affected, but involvement of digits other than the thumb is rare. When digits other than the thumb are involved, spontaneous resolution is more likely. The term congenital trigger thumb reflects the belief that the condition is present at birth, but it frequently is not recognized initially. Others suggest the condition is acquired based on the findings of two large prospective studies in neonates that failed to identify congenital cases of trigger digits.1,2 Trigger thumb in children is rare, affecting less than 0.05% of children. Bilateral involvement may be present in up to one third of patients overall, and it is even more common in patients with trigger thumb diagnosed at birth. Parents notice that the thumb interphalangeal (IP) joint remains partially flexed. Inability to flex the extended thumb also can be the first manifestation. On physical examination, there usually is a thumb IP joint flexion contracture of 10 to 20 degrees and a palpable nodule near the volar aspect of the MP joint. Trigger thumb in a child must be distinguished from congenital clasped thumb, spasticity, or arthrogryposis. Congenital clasped thumb consists of flexion contractures at both the IP and MP joints, whereas the MP joint is uninvolved in trigger thumb.

Treatment

- Many primary trigger digits in adults can be successfully treated nonoperatively.
- Nonoperative treatment includes activity modification, splinting, ice, massage, nonsteroidal antiinflammatory drugs (NSAIDs), and injections. Many authors recommend corticosteroid injections without splinting as the initial treatment for symptomatic primary trigger digits. Patients who refuse an injection can be treated with immobilization alone.
- Several factors help predict which patients will respond favorably to injections. In one series, 93% of patients with nodular trigger digits treated with one injection remained symptom free at 3 months. Only 48% of patients with diffuse stenosing tenosynovitis were successfully treated with an injection.4 Patients with multiple digit involvement, symptoms lasting longer than 6 months, or insulin-dependent diabetes mellitus respond less favorably to corticosteroid injections.4
- Flexor tendon sheath injections can be performed through either a volar or lateral approach. Through either approach, the tip of a small-gauge needle can be initially placed into the tendon and placement confirmed by asking the patient to flex and extend the digit. The needle is withdrawn slightly such that the flexor sheath can be insufflated with the steroid mixture. Caution should be exercised to avoid intratendinous injections.
Lidocaine injections alone are less efficacious than corticosteroid injections with or without a local anesthetic. Counsel the patient regarding the possibilities of transient elevation of serum glucose (in diabetic patients), skin depigmentation, local subcutaneous fat atrophy or necrosis, and tendon rupture. The patient is instructed to move the finger freely after the injection.

- Many authors recommend a series of up to two corticosteroid injections over the course of 3 weeks for patients with trigger digits resulting from a discrete nodule, a short duration of symptoms (<6 months), and single digit involvement.
- Diffuse stenosing tenosynovitis of short duration can be treated initially with a corticosteroid injection, but repeat injections may not be warranted given the poor response of this form of trigger digits to nonoperative treatment.
- **Open release of the A1 pulley** is the standard surgical treatment for trigger digits. Surgical treatment may be indicated for nodular trigger digits that are unresponsive to a series of two corticosteroid injections or for trigger digits that upon initial presentation are locked, involve multiple digits, result from diffuse stenosing tenosynovitis, or are of long duration (>6 months).
- Transverse incisions at the proximal edge of the A1 pulley, oblique incisions, Chevron (Bunnell-type) incisions, and longitudinal incisions have been described. Longitudinal incisions that traverse perpendicular to flexor creases may result in flexion contractures.
- The A1 pulley in adults measures 1.0 to 1.5 cm in length and should be released in its entirety. Preservation of the second annular (A2) pulleys in the fingers, and the oblique pulley in the thumb, is important for prevent bowstringing of the flexor tendons that results in decreased tendon excursion and ultimately to decreased active IP joint flexion. However, the A2 pulley may be continuous with the A1 pulley in approximately 50% of individuals.
- Care should be taken to protect the neurovascular bundles, acknowledging the radial neurovascular bundle to the thumb takes an oblique ulnar to radial course across the MP joint crease.
- The procedure is performed with local anesthesia such that the patient can actively flex and extend the digit following A1 pulley release, confirming successful resolution of the triggering.
- A1 pulley release is not recommended in patients with rheumatoid arthritis and diffused tenosynovitis because of the risk of exacerbating ulnar drift of the digits. Instead, flexor tenosynovectomy should be performed to eliminate the cause of triggering. Some patients with rheumatoid arthritis who have no involvement of flexor tendons respond well to Standard A1 pulley release.

- Partial excision of the FDS with the removal of one slip may be required in some patients with dramatic enlargement of the FDP tendon.
- **Percutaneous A1 pulley release** has been proposed as an alternative technique. Under local anesthesia, the beveled tip of an 18- to 20-gauge needle is inserted into the middle of the A1 pulley. The bevel is oriented parallel to the flexor tendons. A sweeping motion proximally and distally produces a grating sound and results in A1 pulley release. The needle is withdrawn, and the patient flexes and extends the digit to confirm an adequate release. Repeat needle placements may be needed to adequately eliminate the triggering. Percutaneous A1 pulley release usually is performed in conjunction with corticosteroid injection to prevent postoperative painful tenosynovitis without triggering. Some authors recommend not performing percutaneous release in the thumb and possibly the index finger, especially if a PIP joint flexion contracture is present, because the neurovascular bundle may be at increased risk.
- Good results can be expected following A1 pulley release. Triggering is eliminated with no complications in approximately 90% of patients undergoing open A1 pulley release. Percutaneous A1 pulley release has been reported to be successful in approximately 93% of patients.
- Complications include scar tenderness, mild PIP flexion contractures, neurovascular bundle injuries, ulnar drift of the digit (especially the index finger or any finger in a patient with rheumatoid arthritis), tendon bowstringing, infection, algodystrophy, and recurrence. Complications generally occur in less than 5% of patients, although higher complication rates have been reported. With percutaneous release, concerns remain regarding scoring of the flexor digitorum superficialis tendon, which occurs in many cases; injury to the neurovascular bundles, which lie within 2 to 3 mm of needle tip placement for the index finger and thumb; and potential recurrence from incomplete release.

### Management of Trigger Thumb in Children

- Treatment options include observation, splinting, and open surgical release of the first annular pulley.
- Most authors recommend at least a 6-month period of observation prior to surgical intervention. One third of patients younger than 6 months may have spontaneous resolution of the trigger thumb. Spontaneous resolution after age 6 months is much less common in most series.
- Nonetheless, surgery is required for a significant number of patients. Surgery before age 3 years is generally recommended with open release of the thumb’s first annular pulley. The outcome of surgical release in children is generally good. Removal or debulking of the flexor pollicis longus nodule is not recommended.
de Quervain Syndrome

- de Quervain syndrome is stenosing tenovaginitis of the first dorsal compartment of the wrist, which contains the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) tendons.

Anatomy

- The anatomy of the first dorsal compartment of the wrist is highly variable. Failure to recognize these anatomic variations can lead to treatment failures in de Quervain syndrome.
- The EPB tendon is rounder and smaller than the APL and is absent in 5 to 7 percent of individuals. Muscle fibers, if seen within the first dorsal compartment, usually help identify the EPB tendon as the EPB muscle belly extends further distally. Its distal insertion is at the base of the proximal phalanx. The APL usually has two or more tendon slips that may insert onto the trapezius, volar carpal ligament, opponens pollicis, or abductor pollicis brevis and the consistent and functionally important insertion onto the base of the first metacarpal. The most common anatomic variation is one EPB tendon and two APL tendon slips.
- In up to one third of the general population, the first dorsal compartment is subdivided by a septum into two separate fibroosseous tunnels (Figure 14–3). The ulnar-sided tunnel contains the EPB tendon, and the radial-sided tunnel contains the multiple APL tendon slips. A third deep tunnel containing an anomalous tendon has been reported but is uncommon. A higher incidence of septation of first dorsal compartments is reported in patients with de Quervain syndrome, suggesting that separate fibroosseous tunnels predispose to development of de Quervain syndrome.
- The deep branch of the radial artery passes through the anatomic snuffbox distal to the radial styloid process and just deep to the first and second dorsal compartments. The artery’s location should be recognized, but the artery need not be exposed during first dorsal compartment release.
- Several branches of the superficial radial nerve lie within the subcutaneous fat overlying the first dorsal compartment and should be preserved during surgical approaches (Figure 14–4, A).

Diagnosis

- The typical patient diagnosed with de Quervain syndrome is a woman (occurs up to six times more frequently than in men) in her 40s or 50s. Association with pregnancy is common but even higher are mothers with infant.
- Patients present with radial-sided wrist pain exacerbated by thumb movements, particularly thumb abduction and/or extension. Pain may radiate distally or proximally along the course of the APL and EPB tendons. By the time of presentation, symptoms usually have been present for weeks to months.
- Physical examination often reveals localized swelling and tenderness over the first dorsal compartment, extending 1 to 2 cm proximal to the radial styloid process. The Finkelstein test involves asking the patient to clasp the thumb into the palm and then applying an ulnar deviation force to the wrist. Rarely, pseudotriggering of the thumb is present and may be related to interference of smooth EPB tendon excursion in its separate fibroosseous tunnel within the first dorsal compartment.
- Intersection syndrome, which involves the junction of the tendons contained in the first and second dorsal compartments, typically presents with symptoms more proximal (4 cm) to the wrist (see Figure 14–4, B). Thumb carpometacarpal joint, radiocarpal, and intercarpal arthritis frequently can be distinguished from de Quervain syndrome based on radiographs and a positive grind test, although the conditions may coexist. Finally, superficial radial nerve neuroma is unlikely without a prior history of local trauma or

Figure 14–3:
The first dorsal compartment often contains a septum that divides the spaces containing the abductor pollicis longus tendon and the extensor pollicis brevis tendon. The most common combination of tendon slips is two abductor pollicis longus tendon slips and one extensor pollicis brevis tendon slip.

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surgery and usually can be carefully distinguished using Finkelstein test and seeking a Tinel sign (Box 14–3).

**Treatment**

- **Nonoperative treatment** options for de Quervain syndrome include splinting, corticosteroid injections, and various techniques of surgical release of the first dorsal compartment.
  - Splinting alone may be beneficial for acute symptomatic relief but has resulted in a 80% failure rate.9
  - Corticosteroid injections into the first dorsal compartment are a moderately effective nonoperative treatment option. A single corticosteroid injection into the first dorsal compartment sheath is successful in alleviating symptoms in approximately 70% of patients. Two injections are successful in approximately 80% of patients.10 Long-term relief is achieved in approximately 60% of patients.11 Several studies have documented the high failure rate of injections in patients with a separate EPB fibroosseous tunnel within the first dorsal compartment. Corticosteroid injections in patients with diabetes mellitus may be less successful.
  - **Surgical treatment** for de Quervain syndrome must adhere to two principles. First, care must be taken to protect and avoid excessive dissection of the superficial radial nerve and its branches. The second principle is to ensure complete release of the first dorsal compartment, especially the separate EPB fibroosseous tunnel.
  - Longitudinal incisions generally lead to fewer superficial nerve incisions than do transverse incisions. Symptoms secondary to superficial radial nerve injury can be far more severe than those resulting from stenosing tenovaginitis of the first dorsal compartment. Transversal incisions have minimized scarring to the tendons and have resulted in a better appearing scar.
  - Release the first dorsal compartment on its dorsal margin, thereby leaving a palmarly based flap of retinaculum to prevent tendon subluxation. It is important to identify both the EPB and all the slips of the APL by placing traction on the tendons. Most authors agree that sheath excision is unnecessary and predisposes the patient to symptomatic volar tendon subluxation. Volar subluxation of the APL and EPB tendons can be addressed with a retinacular sling or a slip of brachioradialis tendon, if symptomatic.
  - Good results have been reported with release of only the EPB fibroosseous tunnel in septated first dorsal compartments, suggesting isolated stenosing tenovaginitis of the EPB sheath is responsible for de Quervain syndrome.12 Despite this report, most authors

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**Box 14–3** Differential Diagnosis of de Quervain Syndrome

- Intersection syndrome
- Thumb carpometacarpal joint arthritis
- Radiocarpal or intercarpal joint arthritis
- Scaphoid fracture
- Superficial radial nerve neuroma
Flexor Carpi Radialis Tendonitis

- Flexor carpi radialis (FCR) tendonitis is a cause of radial pain at the volar side of the wrist. It is rare in comparison with other disorders in the region.

Anatomy

- The proximity of the FCR tunnel to the radius, trapezium scaphoid, and palmar arch explains the predisposition of the tendon to tenosynovitis with or without trauma.
- The FCR tendon originates from the bipennate flexor carpi radialis muscle approximately 15 cm proximal to the radiocarpal joint. The last 8 cm is completely tendinous. Four to five centimeters proximal to the radiocarpal joint, the tendon is circumferentially covered by the transversely oriented fibers of the antebrachial fascia, which gradually thicken to an average thickness of 3 mm at the level of the trapezial crest.
- Passing the trapezial crest the tendon enters a 17-mm-long fibroosseous tunnel bordered radially by the body of the trapezium, palmarly by the trapezial crest and the transverse carpal ligament, ulnarly by a retinaculum septum that separates the tendon from the carpal tunnel, and dorsally by the insertion of this septum onto the trapezial body. Just proximal to the tunnel, the tendon occupies 60% of the cross-sectional area of the fibrous sheath. Within the tunnel, the tendon occupies 90% of the fibroosseous canal.
- Distal to the tunnel the tendon divides into two slips that attach into the base of the second and third metacarpals (approximately two thirds insert to the second metacarpal and one third to the third metacarpal). The direction of the tendon is collinear with the forearm proximally and deviates approximately 45 degrees within the tunnel and distally.
- Less than 1 mm of soft tissue separates the tendon from the scaphoid tubercle, the scaphoid trapezium trapezoid joint, and the trapezium. Less than 3 mm separates the tendon from the first carpometacarpal joint.

Pathology

- Primary stenosing tenosynovitis may develop because of overuse or inflammation at the site of the fibroosseous tunnel because of the narrow confines and the deviation of the tendon by 45 degrees from the neutral forearm axis. Tendonitis at the site of insertion also may develop.
- Secondary tendonitis may develop because of pathology from the adjacent structures, including scaphoid fractures, scaphoid cysts, arthritis of the scaphoid trapezium trapezoid joint, or arthritis of the carpometacarpal joint of the thumb.

Diagnosis

- In primary stenosing tenosynovitis, the patient complains of radiovolar pain at the wrist, often localized to the proximal aspect of the trapezium. Pain occurs with resisted active wrist flexion and radial deviation and with passive extension and ulnar deviation. Pain relief with lidocaine injection into the sheath confirms the diagnosis.
- Secondary tendonitis can result from previous injuries or arthritis of the adjacent structures, making a detailed history of previous injury at the radiovolar aspect of the wrist important.
- Radiographs are essential to recognize pathology from adjacent osseous structures. Magnetic resonance imaging (MRI) may help rule out cysts or ganglia if suspected (Box 14–4).
- Linberg syndrome is an anomalous intertendinous connection between the tendon of the flexor pollicis longus and that of the flexor digitorum profundus.

Treatment

- Nonoperative treatment includes splinting preventing wrist flexion, physical therapy, and corticosteroid injections. Nonoperative treatment usually is effective for primary tendonitis.
- Splinting in combination with administration of antiinflammatory medications have yielded good results in acute FCR stenosing tenosynovitis.
- One or two corticosteroid injections are the next step for stenosing tenosynovitis of the FCR tendon. The injection is preferably administered from the volar side over the site of tenderness. Caution should be exercised to avoid intratendinous injections, which increase the rate of spontaneous rupture of the tendon.
- Ultrasound courses have shown good results as an adjacent measure to the previous nonoperative treatments.
- Operative treatment is essential when conservative treatment is unsuccessful or in cases of secondary tendonitis resulting from local pathologic changes.
Operative treatment involves treatment of local lesions (removal of ganglions and osteophytes, bone grafting of cysts, fusion of arthritic joints) and decompression of the tunnel.

Operative decompression is performed through a volar incision starting proximal to the wrist crease and extending over the proximal thenar eminence.

Caution should be exercised to avoid the palmar cutaneous branch of the median nerve, the lateral antebrachial cutaneous nerve, and the superficial radial sensory nerve.

The thenar muscle is elevated from the transverse ligament and retracted radially exposing the sheath of the FCR tendon.

The tenosynovial sheath and the tunnel are released longitudinally, taking care not to injure the tendon.

The tendon is mobilized from the trapezial groove, and the trapezial insertion is released.

An alternative approach for decompression includes release from within the carpal tunnel if a concomitant carpal tunnel release is performed.

Good results with operative treatment of FCR tendinitis with a success rate of 90% have been reported in the literature. A history of overuse, cases associated with workers’ compensation, long duration of symptoms before treatment, and failure of local anesthetic to ease the symptoms are associated with poor results.

**Extensor Carpi Ulnaris Tendonitis**

**Anatomy**

- The extensor carpi ulnaris (ECU) is one of the most important stabilizers of the distal radioulnar joint.
- It is the most ulnar extensor of the wrist and originates from the extensor carpi ulnaris muscle approximately 6 to 7 cm proximal to the wrist.
- Although it passes through the extensor retinaculum, a separate deep fibroosseous sheath is formed around it. This fibroosseous deep sheath maintains the tendon in its normal position. The tendon occupies approximately 90% of the space of the fibroosseous sheath. Over the distal ulna it curves ulnarily passing into an ulnar groove on the dorsal surface of the ulna before inserting onto the base of the fifth metacarpal. The narrow space in the fibroosseous sheath and the angulation of the tendon, particularly after voluntary contraction of the muscle, predisposes to tenosynovitis. The ECU subsheath is part of the triangular fibrocartilage complex of the wrist.
- A few centimeters before insertion, the fibroosseous sheath becomes thinner. Maximum ulnar translocation stress is noted on the tendon and its sheath during ECU muscle contraction with the forearm in supination and the wrist ulnarily deviated. Rupture of the fibroosseous sheath leading to subluxation or dislocation of the tendon may occur with hypersupination of the forearm and ulnar deviation and flexion of the wrist, with active voluntary contraction of the extensor carpi ulnaris muscle. Division or attenuation of the fibroosseous sheath allows dislocation of the tendon, even with the overlying extensor retinaculum intact.

**Diagnosis**

- Usually the patient presents with pain on the ulnar side of the wrist after a single injury or with progressively increased pain.
- Physical examination reveals pain on the ulnar side of the wrist, pain with palpation over the fibroosseous tunnel and/or the insertion of the tendon to the base of the fifth metacarpal, pain with passive flexion and radial deviation of the wrist, pain with resisted extension and ulnar deviation of the wrist with the forearm in hypersupination, and pain while applying maximum grip strength with resisted ulnar deviation of the wrist. Ulnar nerve dorsal (sensory) branch dysesthesias may coexist.
- If the fibroosseous sheath is ruptured, the patient may complain of a painful soft snap. Swelling on the dorsal side of the wrist is noted, as is dislocation of the tendon with voluntary contraction of the ECU muscle.
- Radiographs may be helpful to exclude pathology from the adjacent structures. Diagnosis of ECU tendonitis based on MRI findings alone is not reliable, but MRI is useful in the differential diagnosis of ulnar-sided wrist pain (Box 14–5). Dynamic ultrasound can quickly and accurately make the diagnosis.

**Treatment**

- Tendonitis or tenosynovitis of the ECU tendon can be initially treated with nonoperative measures such as rest and splinting, antinflammatory medications, corticosteroid injections, and physical therapy, although the efficacy of a conservative approach has been questioned. Other causes of ulnar-sided wrist pain should be ruled out.
- Splinting of the wrist in slight extension for 3 weeks and administration of antinflammatory medications is the first step of the therapy.

**Box 14–5  Differential Diagnosis of Extensor Carpi Ulnaris Tendonitis**

- Injury of the triangular fibrocartilage complex
- Distal radioulnar joint arthritis
- Fracture, nonunion, or malunion of the ulnar styloid
- Ulnocarpal impaction syndrome
- Calcification of the ECU tendon insertion
- “Snapping” of the ECU tendon
Operative Treatment

- If pain persists, local injection of corticosteroids is advocated. Caution should be exercised to avoid intratendinous injections.

Conclusions

- In chronic cases or those unresponsive to conservative treatment, surgical decompression is an excellent option. Common causes of failure in these procedures are incomplete release and nerve injury. Another common pitfall is failure to recognize underlying conditions, such as arthritic osteophytes, ganglion cysts, or coexisting pathology contributing to the patient’s symptoms. Although most of these procedures are technically simple, they should not be undertaken lightly.

References

3. Freiberg A, Mulholland RS, Levine R: Nonoperative treatment of trigger fingers and thumbs. J Hand Surg 14A:553-558, 1989. The authors make the clinical distinction of trigger digits into nodular and “diffuse” types, with a higher success rate following injection in the nodular type (93%) compared to the “diffuse” type (48%).
4. Patel MR, Bassini LB: Trigger fingers and thumb: when to splint, inject, or operate. J Hand Surg 17A:110-113, 1992. In this comparative study of splinting (for an average of 6 weeks) versus injection (with cortisone and lidocaine) in 100 patients, injections were more successful (84% success rate) than splinting (66%).
Cortisone and lidocaine injections were successful in 62% when the need for operative release was used as an outcome result for treatment failure in this prospective study of 87 wrists.


Sixteen patients underwent surgical release of only the EPB subcompartment for de Quervain disease, and all had relief of symptoms.


This retrospective study of 43 wrists treated surgically for de Quervain disease reported a complication rate of 9%.


This is an excellent review of the pertinent anatomy to flexor carpi radialis tendons based on the cadaveric dissection of 25 specimens.


This is a classic article on the anatomy and biomechanics of ECU tendon and its fibroosseous sheath.


In an MRI study of 26 normal wrists, high signal intensity of the ECU tendon simulating tendonitis was a very common finding.


In this report, 12 patients were treated with decompression of the sixth dorsal compartment for ECU tendonitis without an attempt at repair of the ECU sheath, with no resulting distal radioulnar joint or ECU instability.


This article provides a practical algorithm for treating recurrent dislocation of the ECU tendon by dividing the pathology into three types.


In this report of 72 patients with ECU tendonitis, local steroids provided long-standing pain relief in only nine patients. The 63 patients treated surgically had uniformly good results.

This study describes high complication (28%) and failure (40%) rates following open trigger digit release and attributes problems to surgeon inexperience.


In this study, all 53 pediatric trigger thumbs eventually required surgical release, although waiting up to 3 years did not adversely affect the outcome.


This study reported a 50% rate of spontaneous recovery of trigger thumb in children.


Conservative treatment for trigger thumb in children, including splinting and regular exercises, yielded an overall rate of success of 66%.

**de Quervain Disease**


A study of 300 cadaveric wrists and a prospective study of 40 patients who underwent de Quervain tenosynovitis release revealed a significantly higher occurrence of septation of the first dorsal compartment in the population with de Quervain syndrome (67%) than in the cadavers (33%).

**Flexor Carpi Radialis Tenosynovitis**


In a study of 10 patients who had undergone flexor carpi radialis tunnel release, seven needed additional procedures.

**Extensor Carpi Ulnaris Tenosynovitis**


This is a classic article on the anatomy and biomechanics of ECU tendon and its fibroosseous sheath.


This early report discusses surgical technique for release of the ECU compartment.


This was the first report of surgical treatment for acute traumatic subluxation of the ECU tendon.


This article provides a practical algorithm for treating recurrent dislocation of the ECU tendon by dividing the pathology into three types.

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**Suggested Readings**

**Trigger Finger**

Nerve Physiology and Repair

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Introduction

- Although significant advances have been made in the microsurgical repair technology and in our understanding of nerve physiology at the molecular level, restoration of function after a peripheral injury remains a great challenge.
- This chapter aims to elucidate the current understanding of the peripheral nerve’s physiology in its normal, injured, and regenerative phases and the basic principles in the treatment of nerve injury.

Nerve Physiology

Microanatomy of the Peripheral Nerve

- The neuron is the basic unit of the peripheral nerve. Each neuron consists of a cell body (soma) with cytoplasmic extensions (dendrites and an axon), covered with various synaptic terminals called boutons (Figure 15–1).
- The axon is a special cylindrical extension arising from the neuron at the axonal hillock. The function of the axon is twofold: bidirectional axonal transport and electrical impulse conduction.
- For an unmyelinated nerve, each Schwann cell surrounds several small axons. For a myelinated nerve, Schwann cells surround a single axon.
- The myelinated larger axons are wrapped along their entire length by contiguous Schwann cells that are the glial cells of the peripheral nervous system. The myelinated axon is covered by myelin sheath except for the nodes of Ranvier, which are specialized myelin-free areas that allow ionic exchange and facilitate propagation of electric conduction.
- The myelin sheath is a proteophospholipid, multilayered, compacted cell membrane that maximizes the conduction efficiency and the velocity of action potentials.
- Myelinated axons have four distinctive regions: node of Ranvier, paranode, juxtaparanode, and internode. Each zone is characterized by a specific set of axonal proteins. Voltage-gated sodium channels are clustered at the node of Ranvier, whereas potassium channels are concentrated at juxtaparanodal regions (Box 15–1).

Connective Tissue Components of the Peripheral Nerve

- The peripheral nerves contain an abundant amount of collagen that makes them strong and resistant to trauma, in contrast to the cranial nerves, which are rich in fiber but have minimal collagen content.
- The cross section of a peripheral nerve contains variable amount of connective tissue (up to 85%) (Figure 15–2).
- The epineurium, which is the elongation of the dural sleeve of the spinal nerve roots, can be divided into two layers: external and internal. The external epineurium is the outermost layer surrounding the peripheral nerve and anchors blood vessels entering from the surrounding...
The internal epineurium fills between fascicles and cushions them from external force.

- The perineurium encircles each fascicle and provides the diffusion barrier. It is composed of many layers of flattened cells alternating with collagen fibers. This layer is an extension of the blood-brain barrier and maintains positive pressure within the fascicles. This pressure difference is evident when the cut end of a nerve shows round herniating fascicular ends. This pressure difference is essential for axoplasmic transport and nerve conduction. The perineurium is the strongest component of the nerve trunk and protects nerve fibers from stretch injury.

- The endoneurium is the innermost collagen layer surrounding individual axons within the perineurial layer. Larger myelinated axons are wrapped with two layers of endoneurium: the outer longitudinally oriented collagen layer and the inner randomly oriented layer. Smaller myelinated axons have only the outer layer.

**Box 15–1  Neuron Anatomy and Function**

<table>
<thead>
<tr>
<th>Cell body</th>
<th>Axon</th>
<th>Myelin</th>
<th>Node of Ranvier</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein synthesis</td>
<td>Bidirectional axonal transport and nerve conduction</td>
<td>Maximizes conduction efficiency</td>
<td>Myelin free area for ionic exchange</td>
</tr>
</tbody>
</table>
Longitudinal intrinsic blood vessels in the epineurium, perineurium, and endoneurium are interconnected with each other and with extrinsic blood vessels, the arteriae nervora, entering the nerve from surrounding tissue. This rich, longitudinal plexus of blood vessels allows a nerve to be mobilized for a significant distance without ischemic compromise.

Loose areolar tissue surrounding the peripheral nerve allows longitudinal excursion of the nerve. This is an important feature to prevent traction injury to the nerve crossing a limb joint. Wilgis et al. demonstrated that the median nerve crossing the wrist glides as much as 15.5 mm and the ulnar nerve glides as much as 14.8 mm (Box 15–2).

The peripheral nerves contain motor, sensory, and autonomic fibers. The motor fibers originate from the motor neurons in the anterior horn of the spinal cord. The sensory fibers originate from the sensory neurons in the dorsal root ganglia. The autonomic fibers are either preganglionic, arising from the neurons in the brainstem/spinal cord, or postganglionic, arising from the neurons in paravertebral ganglia.

Axoplasmic Transport

- Bidirectional axoplasmic transport can be fast or slow type. Membrane and secretory proteins synthesized in the soma are transported orthograde to the terminal by either a fast or a slow transport system.
- Fast axoplasmic transport moves subcellular organelles, such as synaptic vesicles, down to the nerve ending. This transport requires energy; therefore, it is sensitive to hypoxemia. The reported rates of fast transport are variable, reaching up to 400 mm/day. The fast retrograde transport moves degraded axoplasmic materials, neurotransmitters, and neurotrophic substances ingested at the nerve terminal back to the cell body. Nerve growth factors (NGFs) manufactured by the motor and sensory target organs are transported back to the cell body and are crucial for neuron survival. Retrograde transport also is responsible for carrying pathogens such as herpes simplex virus.
- Slow transport is orthograde only and moves 1 to 4 mm/day, moving components of cytoskeleton such as neurotubules and neurofilaments to the nerve terminal. This speed of transport limits the rate of peripheral nerve regeneration.

Nerve Conduction

- The neuron has the capability for propagating electric currents. This ability is achieved by the resting potential across the axonal membrane. This negative potential of $-60$ to $-70$ mV is maintained by actively partitioning the extracellular sodium and intracellular potassium ions against the concentration gradient.
Sodium ion gates open when stimulated by an electric impulse and allow sodium ions to rush into the cell following their concentration gradient. This process causes a decrease in the resting membrane potential and represents the rise in the action potential. The sodium channels then close and the voltage-gated potassium channels open, causing potassium ions to diffuse out of the cell, following its concentration gradient. The localized potential thus produced is propagated down the axon, creating the action potential, which is an all-or-none phenomenon. The action potential, once initiated, travels the entire length of the axon without decrement (Figure 15–3).

In unmyelinated fibers, the longitudinal spread of the electric current occurs with progressive excitation of the adjacent inactive areas. In myelinated fibers, the electric impulse jumps from one node of Ranvier to next the node, in a process called salutatory conduction. Therefore, conduction in unmyelinated fibers is slower than in myelinated fibers.

After each depolarization, a period of recovery is needed to restore the resting potential. The adenosine triphosphatase (ATPase) pump restores the membrane resting potential by actively moving sodium ions out, allowing potassium ions to passively flow back in.

Figure 15–3:
Conduction velocity represents how fast the action potential is propagated to the target organ. Conduction velocity can be increased in two ways: by increasing the diameter of the axon and by decreasing capacitance to the flow. Conduction velocity is proportional to the square of the diameter of the axon. A nerve must be of gigantic size to conduct fast. Schwann cells allow the axon to overcome this problem by surrounding the axon with the high-resistance, low capacitance-insulator myelin. However, the entire nerve cannot be surrounded by a myelin sheath because sodium and potassium ion movements across the axonal membrane would be prevented. Thus, the node of Ranvier, which is a myelin-free segment, plays a crucial role in facilitating ionic exchange and salutary conduction. Voltage-dependent sodium channels are uniformly distributed along unmyelinated axons but are highly concentrated in the nodes of Ranvier to facilitate salutary conduction in myelinated axons.

### Neuromuscular Transmission

- Motor axons terminate into several branches, which end in synaptic boutons. The bouton membrane contains voltage-gated calcium channels, which allow influx of calcium ions when stimulated with an impulse. This action triggers the release of synaptic vesicle content: acetylcholine (ACh).
- ACh neurotransmitters cross the synaptic cleft and bind to the receptors in the muscle basement membrane. These receptors are transmitter-gated ion channels that allow passage of sodium ions across the membrane. Sodium influx depolarizes the muscle membrane and results in muscle contraction.
- The smallest unit of this neuromuscular function is the motor unit, which represents one motor neuron and the muscle fibers innervated by it. The innervation ratio of the muscle fibers to the motor neuron is 80 to 100 in small muscles, such as the intrinsic muscles in the hand, to 1000 to 2000 in large muscles of the leg.
- Each muscle contains a mixture of the various fiber types: slow fatigable (type I), fast oxidative (type IIA), fast fatigable (type IIB), and fast intermediate (type IIC). Specific nerve fibers innervate different types of muscle fibers: fast fatigable muscle fibers are innervated by large rapidly conducting axons and slow fatigable fibers by smaller, slower axons.
- Botulinum toxins, exotoxins of *Clostridium botulinum*, act at the motor endplates by blocking the release of ACh neurotransmitters. Once feared as the most toxic substance known to man, Botulinum toxin enjoys wide popularity in treatments of dystonia and spasticity.

### Sensory Receptors

- The three common types of mechanoreceptors are the Merkel cell complex, Meissner corpuscles, and Pacinian corpuscles (Box 15–3).

#### Box 15–3 Sensory Mechanoreceptors

<table>
<thead>
<tr>
<th>Sensory Mechanoreceptors</th>
<th>Location</th>
<th>Receptive Field</th>
<th>Adaptation</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Merkel cell complex</td>
<td>Clustered around the sweat duct</td>
<td>2- to 4-mm receptive field</td>
<td>Slowly adapting</td>
<td>Static two-point discrimination</td>
</tr>
<tr>
<td>Meissner corpuscle</td>
<td>Located at the sides of the intermediate ridge</td>
<td>Responds to flutter vibration</td>
<td>Rapidly adapting</td>
<td>Moving two-point discrimination</td>
</tr>
<tr>
<td>Pacinian corpuscle</td>
<td>Located in the subcutaneous tissue</td>
<td>Looks like a grain of rice</td>
<td>Rapidly adapting</td>
<td>Several-centimeter receptive field vibration at 250 cps</td>
</tr>
</tbody>
</table>

### Nerve Injury and Repair

#### Classification of Injury

- In 1948, Sir Herbert Seddon devised a classification system of nerve injury based on disrupted internal structures of the peripheral nerve. His system of *neuropaxia, axonotmesis, and neurotmesis* is simple and widely used today, because it correlates the degree of nerve injury with the prognosis of functional recovery.¹
- In 1968, Sir Sydney Sunderland expanded on Seddon’s classification system by putting more emphasis on the fascicular layers of the nerve.
- Sunderland’s first-degree injury is Seddon’s neuropraxia, which is a demyelinating injury with a temporary conduction block that resolves completely in 1 to 2 days.
- The second-degree injury is axonotmesis, where distal degeneration of the injured axon occurs. Regeneration of the axon almost always is complete, because the endoneurial layer is intact and may take up to several weeks, depending on the location of the injury.
- Sunderland’s third-degree injury is the less severe of Seddon’s neurotmesis category, where the perineurial layer is intact. Regeneration occurs but is not complete because of endoneurial scarring and loss of end-organ specificity within the fascicle.
- Sunderland’s fourth-degree injury is more severe, where the axon, endoneurium, and perineurium are disrupted, causing more extensive scarring that blocks axonal regeneration. Spontaneous nerve regeneration often is unsatisfactory in the fourth-degree injury, resulting in neuroma in continuity.
- Sunderland’s fifth-degree injury corresponds to severed nerve trunk, and spontaneous regeneration is not possible without surgical coaptation. Sunderland emphasized that any given nerve injury may contain mixed degrees of injury and rarely is pure in classification (Table 15–1).
Degeneration and Regeneration

Degeneration

- The axon distal to the injury site undergoes a well-described degenerative process known as Wallerian degeneration, which involves breaking down the old axon and clearing the myelin debris of the axoplasm (see Figure 15–3). This degenerative process takes at least 1 to 2 weeks; therefore, the electrodiagnostic test performed early may not indicate abnormalities during this period.

- The known key players for Wallerian degeneration are macrophages, Schwann cells, various neurotrophic factors, the injured neuron, and the end organs. They influence each other in an intricate interplay, which is understood only rudimentarily at present.

- Macrophages, in addition to performing phagocytosis, express interleukin (IL)-1, which is a potent stimulator for Schwann cells to produce neurotrophic factors and express neural adhesion molecules.

- The roles of Schwann cells are multiple and crucial. They begin by breaking down myelin, which is followed by proliferation and formation of the band of Bünger within the basal lamina of the original Schwann cell tube. Schwann cells secrete various neurotrophic factors and express neural adhesion molecules. Last, they circumscribe regenerating axons and myelinate them.

- If an axon fails to regenerate immediately, the distal endoneurial tube shrinks irreversibly, making later regeneration of the axon even more difficult. This process appears to be the main reason for the failure to successfully reinnervate muscles following a delayed surgical nerve repair, rather than degeneration of muscle fibers and motor endplates as previously thought.

Regeneration

- Within hours of injury, the proximal axon begins to regenerate with sprout formation. Collateral sprouts grow from the most distal node of Ranvier proximal to the site of injury. These regeneration sprouts may be fairly close to the injury site in a sharp transection or far in avulsion or crush-type of injury.

- During regeneration, Schwann cells and neurons have an interdependent relationship mediated by neurotrophic factors and neurite outgrowth promoting factors. The neurotrophic factors facilitate bidirectional communication between neurons and Schwann cells. Neurotrophic factors not only act on neurons as previously well described in the literature, but also they exert a vital effect on Schwann cells. Neurite outgrowth promoting factors facilitate attachment of growing axons to other axons and/or Schwann cells, guiding nerve regeneration.

Neurotrophic Factors

- Neurotrophic factors are a family of peptides essential in neuronal survival after nerve injury. Three major groups of neurotrophic factors are identified: neurotrophins, neurokines, and transforming growth factor (TGF)-β family, including glial-derived neurotrophic factor (GDNF).

Neurotrophins

- The neurotrophins are small, basic polypeptides, which include NGF, brain-derived neurotrophic factor (BDNF), neurotrophin-3 (NT-3), and neurotrophin-4/5 (NT-4/5).

- NGF is the best known and studied neurotrophic factor since it was first isolated from mouse sarcoma tissue by Levi-Montalcini and Hamburger in 1951. NGF produced by Schwann cells binds to NGF-specific receptors on the axonal membrane. NGF-receptor complex is internalized and transported retrograde to the cell body, which responds with increased synthesis of tubulin, neurofilament, and neurotransmitters. NGF increases survival of sympathetic and sensory neurons but has no significant influence on survival or regeneration of motor neurons. NGF has been proposed for clinical uses, including treatment of Parkinson disease and Alzheimer disease.

- BDNF, NT-3, and NT-4/5 appear to support survival of motor neurons, promote regeneration of the motor neurons, and regulate neuromuscular synapses.
Neurokines

- The neurokines play a significant role in nerve regeneration. Almost all neurokines of the IL-6 family, except ciliary neurotrophic factor, are up-regulated after nerve injury. Other neurokines, such as leukocyte inhibitory factor and IL-6, are up-regulated in Schwann cells after nerve injury and appear to support motor neurons in vitro.

Glial-Derived Neurotrophic Factor

- GDNF, which belongs to the TGF-β family, is up-regulated in nerve injury and promotes motor neuron survival. It has a strong trophic effect on Schwann cells, thus implicating its role in nerve regeneration.

Neurite Outgrowth Promoting Factors

- In addition to producing many neurotrophic factors and their receptors, Schwann cells up-regulate production of neurite outgrowth promoting factors: cell adhesion molecules and extracellular matrix proteins in basement membrane.

- Cell adhesion molecules (CAMs), such as Ng-CAM/L1, N-CAM, and N-cadherin, as well as L2/HNK-1 expressed on the surface of Schwann cells, bind with L1, N-CAM, and with other cell adhesion molecules and integrins on the axonal surface. Interestingly, L2/HNK-1 is expressed only in Schwann cells that line the motor pathways and may play a role in motor neurotropic guidance.

- Extracellular matrix proteins in basement membrane, such as laminin, fibronectin, heparan sulfate proteoglycans, and tenascin, bind to integrins on the surface of growing axons and guide them distally.

Growth Cone

- The leading edge of each regenerating fiber is called the growth cone and contains numerous filopodia that probe into surrounding areas in ameboid fashion. When a suitable surface such as Schwann cell membrane or basement membrane is reached, the filopodium adheres to the surface, the growth cone is pulled forward, and the axon grows in that direction. The growth cone is also active in endocytosis of extrinsic molecules such as neurotrophic factors produced by Schwann cells. These molecules are transported retrograde to the cell body to up-regulate structural protein synthesis and promote further growth.

Myelination

- When the growing axon reaches the end organ, maturation of the axon progresses proximal to distal. Increase in axonal diameter occurs first, followed by myelination. The final diameter of the mature regenerated axon usually is smaller with a thinner myelin sheath compared to the preinjury status.

- Neurotrophins are key mediators of the peripheral nerve myelination process, both as positive and negative modulators. The myelination program involves numerous intercellular signals, including neuregulins, adenosine triphosphate, and the neurotrophins.

Reinnervation of the Motor Unit

- When a regenerating motor axon reaches a denervated muscle, reinnervation occurs at the old motor endplates. In normal muscles, there is a random distribution of motor units. In reinnervated muscles, there are clusters of smaller motor units. Even if only a few fibers reach the denervated muscle, axons sprout to reinnervate many surrounding endplates, forming giant motor units. Schwann cells appear to play an important role in forming motor endplates. Terminal Schwann cells induce and guide regenerating nerve ends into the muscle (Figure 15–4).

Reinnervation of Sensory Receptors

- When sensory nerve fibers are injured, sensory receptors degenerate much like muscles, the other type of end organ. The Merkel cell-neurite complex degenerates first, followed in a few months by Meissner corpuscles. Pacinian corpuscles appear stable and intact over 1 year.

- Among different sensory modalities, the return of perception, pain, and temperature precedes the return of touch. The touch submodalities recover in an orderly sequence: first the perception of 30 cycles per second (cps) frequency, followed by moving touch, static touch, and finally 256 cps stimulus.

Clinical Application of Peripheral Nerve Physiology

Clinical Assessment

- When evaluating a patient with a nerve injury, the hand surgeon must learn the mechanism of injury (stretch, crush, avulsion, sharp, or blunt injury), how much time has elapsed since the injury, the patient’s hand dominance, the patient’s occupation, and other relevant medical history. Especially crucial is the patient’s subjective complaint. Is pain or cold intolerance most debilitating for the patient instead of an obvious dysfunction noted on physical examination?

- The clinical assessment of peripheral nerve function uses tests of both sensibility and motor function. Threshold testing, density testing, and empiric testing are examples of sensory clinical tests (Box 15–4).

- Assessment of motor function can be achieved using subjective muscle grading and quantitative strength testing. As devised by the British Medical Research Council.
Figure 15–4: Wallerian degeneration and regeneration. A, Breaking down of axoplasm and cytoskeletons into granular, ovoid debrides. B, Macrophages within the nerve and from circulation are attracted to the injured area in large numbers. By 2 to 3 days, they start to phagocytize these debrides and clear them away. C, Schwann cells start to proliferate with mitosis and form a line known as Bungar band. D, Schwann cells wrap around newly regenerating axons and myelinate. (From Snell RS: Clinical neuroanatomy, ed 5. Philadelphia, 2001, Williams & Wilkins.)
subjective staged levels of motor recovery have gained wide clinical acceptance (Box 15–5).

- Quantitative strength testing that relies on measurable data enables the surgeon to compare alternative methods of nerve repair. In quantitative testing, the force of an isolated contracting muscle or muscle group after reinnervation is measured using a load cell, and the result is expressed as a percentage of the contralateral uninjured side. This technique provides more accurate data of the patient’s clinical recovery to allow for comparison of results of different methods of nerve repair.

- Based on the evaluation, the hand surgeon determines whether a more proximal or systemic pathology such as cervical radiculopathy or degenerative neuropathy is causing the neurologic deficit, whether a partial or complete nerve injury is present, whether to offer a nonsurgical or surgical treatment option, and the timing of surgical intervention if any is needed. Other laboratory or ancillary tests may help the surgeon in answering some of these questions.

Ancillary Tests

- Nerve conduction velocity (NCV) studies, electromyography (EMG), somatosensory evoked potentials (SSEP), and magnetic resonance imaging (MRI) are some of the ancillary tests useful for evaluating peripheral nerve function.

| Threshold test | Semmes-Weinstein monofilament pressure Vibration | 30–256 cps | Nerve compression syndrome |
| Density test | Static two-point | Tests functioning sensory end organs | Reinnervation and cortical reeducation status |
| Empiric test | British Medical Research Council | Subjective | Qualitative |

Nerve Conduction Velocity / Electromyography

- The most sensitive test for detecting nerve compression syndromes with demyelination is the NCV test. EMG, in contrast, is most helpful after a nerve injury that results in denervation with loss of axons.

- To measure NCV, a nerve is stimulated proximally and the response is recorded distally over a fixed distance. The distance between the stimulus and the distal electrode is divided by the latency to calculate the NCV. Latency is the measured time interval between the proximal nerve stimulation and distal recording. Electrodes placed in or over muscle detect motor conduction, whereas leads on the digits detect sensory conduction.

- Injuries that cause focal demyelination produce delays in NCV. A decrease in nerve conduction across a localized region indicates compression neuropathy (Box 15–6).

- Various factors such as the nerve being studied, the equipment used and its calibration, the room temperature, and the patient’s age influence the results of nerve conduction studies.

- For median nerve motor latency, normal values generally are less than 4.0 ms, whereas sensory latencies are less than 3.6 ms across the wrist.

- For ulnar nerve latency, conduction velocities across the elbow average 60 m/s, with the lower limit of normal 50 m/s. In cubital tunnel syndrome, the conduction velocity across the elbow is slowed by at least 10 m/s compared to the forearm segment.

- Normal muscle has no measurable electrical activity in the absence of voluntary contraction, except for a brief burst of insertional activity when the EMG needle is introduced into the muscle. With voluntary muscle contraction, the action potentials form and increase in frequency with increasing intensity of muscle contraction. An injury that disrupts nerve axons produces electromyographic changes such as fibrillations and positive sharp waves in the denervated muscle within 2 to 3 weeks. As reinnervation occurs, the target skeletal muscle begins to demonstrate polyphasic potentials and small voluntary action potentials. Fibrillation patterns can also be detected with severe nerve compression syndromes that produce axonal disruption.
Somatosensory Evoked Potentials

- A peripheral sensory stimulus produces electric signals detectable by electrodes placed on the contralateral scalp. These signals are called somatosensory evoked potentials. Any disruption along the sensory pathway results in the failure to detect these signals. The disruption may be either preganglionic or postganglionic. Preganglionic injuries demonstrate abnormal SSEPs but normal conduction velocities because the postganglionic fibers are intact. When ordering the NCV and EMG studies, the surgeon must carefully identify the clinical questions to be answered by these studies.

Magnetic Resonance Imaging

- MRI technology has advanced significantly in the past 10 years and has been useful in diagnosing nerve lesions early. The MRI axis can be oriented along the course of a peripheral nerve. Experienced neurologists can provide reliable information regarding neurona formation or spinal nerve root avulsion. Damage to the myelin sheath in neuropaxia may be discerned by the loss of T2-weighted signals.

Treatment of Nerve Injuries

- For a nerve injury requiring surgical exploration and repair, the surgeon determines whether a primary repair or secondary repair is indicated. The general rule is that the primary coaptation of the severed nerve ends is preferable to a secondary repair using a nerve conduit. It is extremely important for the surgeon to realize that this rule has a limited application. Overcoming a nerve gap by stretching the injured nerve, joint positioning in flexion, or repairing under tension using thick sutures guarantees a suboptimal outcome. Stretching causes a secondary traction injury to the nerve, compromising intraneural blood supply. Joint positioning limits the rehabilitation process and may disrupt the nerve repair site when the patient starts moving the extremity.
- The ideal nerve repair should ensure accurate alignment of each and all fascicles with minimal use of suture material or foreign body. The current state of nerve repair is still evolving, and we have yet to determine the perfect repair technique. In the present era, all nerve repairs should be performed using the operating microscope or at least adequate loupe magnification and microsurgical techniques. Nerve repair sites should be tension free.

Primary Nerve Repair

- Primary nerve repair includes epineural, group fascicular, and interfascicular repairs. Epineural repair is ideal when the proper alignment of the nerve is apparent in partial nerve injuries, sharp lacerations, or distal nerve injuries (e.g., at the digital nerve level). When a nerve injury includes a crushing component or when the repair is delayed, group fascicular repair is preferred if the group fascicular pattern is distinguishable. Interfascicular repair usually is not performed because of the excessive sutures required at the repair site (such that the suture material potentially prevents regeneration) and the difficulty in aligning the individual fascicles.

Secondary Nerve Repair

- Secondary repair is used to overcome nerve gaps resulting from segmental nerve loss with crush-type injuries or with delayed repairs. Nerve grafting usually is required for secondary nerve repair. The sural nerve graft is the gold standard for nerve autografts because of the high ratio of axons to connective tissue and the minimal sensory deficit on the lateral aspect of the foot that is generally well tolerated. Digital nerves can be grafted using the lateral or medial antebrachial cutaneous nerves. However, loss of these nerves can lead to significant paresthesias and may not be tolerated well. The small articular branch of the posterior interosseus nerve is a good donor nerve for grafting digital nerves without producing a sensory deficit. Fibrin glue techniques are helpful when using multiple cable grafts to glue them together and for use at the nerve repair site.
- No great substitute presently exists for autogenous nerve grafts; however, research studies are ongoing to find an ideal nerve conduit that will obviate donor site morbidity from nerve graft harvest.
- Sporadic reports on the use of a segment of vessel to bridge a nerve gap have been reported since Bungner's report in a canine model in 1891. Chiu et al. presented histologic and electrophysiologic evidence of nerve regeneration across a venous nerve conduit in rodents. Subsequently, a prospective clinical study comparing sensory recovery in distal peripheral nerves between autogenous vein graft, nerve graft, and direct repair showed comparable results for short nerve gaps (<3 cm) (Figure 15–5).3
- Various investigators researched the use of nerve allografts to reduce donor morbidity resulting from autogenous nerve graft harvest.6 The problems of nerve allografts include the possible transmission of viruses and the need for at least temporary host immunosuppression. Current research studies also focus on using allogenic nerve grafts in conjunction with immunosuppression with minimal side effects or rendering them less antigenic with cryopreservation.
- Other various nonneural conduits have been studied in animals and humans, including polyglycolic acid collagen tubes, silicone tubes, muscle basement membrane, amniotic membrane, and fibronectin mats. When seeded with Schwann cells, these conduits appear more effective in bridging nerve gaps than without Schwann cells in animal studies. Some of these conduits are commercially available, and a few studies show favorable results when
the conduits are used as an entubulation device in primary neurorrhaphy.

- Nerve transposition is a treatment for injuries of the ulnar nerve near the elbow because of the relative lack of nerve branches near this area. Bone shortening is generally limited to situations in the upper extremity where there is a concurrent fracture.

### Treatment of Neuromas

- Failure of the regenerating nerve fibers to reach distal targets results in either a terminal neuroma or a neuroma in continuity. By definition, every nerve laceration or avulsion that is not repaired results in the formation of a terminal neuroma, and even repaired nerves form a neuroma in continuity.
- Repair of the severed peripheral nerve is the only way to prevent or minimize a terminal neuroma formation. A neuroma located in an area with little soft tissue coverage, such as in the dorsum of the hand or the foot, can be extremely painful. For example, a neuroma involving the sensory branch of the radial nerve may render the patient intolerant of even wearing a long sleeve shirt or a wrist watch. The treatment methods for repairing neuromas include excising the neuroma, burying the neuroma, and capping the neuroma. Excising the neuroma and performing a primary repair or nerve grafting can be an effective treatment if the repair site can be protected under a muscle or soft tissue. When a repair of the severed ends of the nerve is not possible or desirable, resecting the neuroma and burying the proximal end in muscle or even bone can be an effective treatment.
- Treatment of a neuroma in continuity provides another set of challenges because some fibers remain intact and therefore must be protected. The intact fibers may be adherent or traverse within the neuroma. In a mixed nerve with sensory and motor fibers, the surgeon must determine whether the intact fibers are mostly sensory or motor, whether they are worth saving, and, if so, whether a direct separation of the fibers within the neuroma can be avoided. Dissection of the nerve proximal and distal to the neuroma may allow bypassing the neuroma with a nerve graft to the injured group of fascicles, therefore avoiding direct dissection within the neuroma, which may cause further scarring and disruption of the intact nerve fibers. Intraoperative electrical stimulation can be used to help identify the intact motor fibers and protect them during the neuroma resection. Neuromas formed by disrupted motor fibers with intact sensory axons are less symptomatic and, in the upper extremity, can be treated with tendon transfers without the need to explore the neuroma.

### End to Side Neurorrhaphy

- Viterbo et al.\(^7\) demonstrated collateral nerve growth with lateroterminal neurorrhaphy without and with an epineural window in rats, confirming the result with electrophysiologic studies. The clinical application of lateroterminal neurorrhaphy remains experimental because of variable reported results.\(^8\) Donor muscles may become acutely denervated when its innervation is compromised with an end to side neurorrhaphy; however, no long-term deficit has been noted in experimental studies.\(^9\) Subsequent studies of end to side repair have demonstrated that the transected nerve branch sends axons down the outer epineurium of the intact nerve branch receiving the end to side coaptation. Therefore the axons that enter the end to side neurorrhaphy end up traveling through the outer epineurium of the intact nerve rather than coming from spontaneous budding of axons from an intact nerve.

### Neurotropism

- The concept of purposeful nerve regeneration toward the correct distal pathway and the correct endorgan is called neurotropism. Some evidence indicates that tissue specificity exists for regenerating axons by their growing toward nervous tissue rather than other types of tissue. The hypothesis is that axons grow toward a gradient of diffusible substance produced by an appropriate target. Except in the rat sciatic nerve Y-tube model, nerve trunk specificity has not been proven.\(^11\)
- However, increasing evidence suggests that sensory-motor specificity exists. Motor neurons do not preferentially reinnervate motor nerve or muscle rather than sensory...
neurons. Random reinnervation occurs but motor axons that regenerate into a sensory pathway later become pruned when connected to wrong target organs. After Wallerian degeneration, the motor pathway is different from the sensory later pathway in that carbohydrate epitope L2/HNK-1 is present in the motor pathway.\(^\text{12}\)

- No evidence for topographic or end-organ specificity is evident at present, contributing to the great difficulty of mixed nerve reconstructions.

### Conclusions

- Various factors contribute to the outcome following a nerve injury, including the nature of the lesion, presence of axonotmesis or neurotmesis, degree of intraneural scarring, and distance to the target organ. Despite significant advances in neurorrhaphy techniques, the outcomes of nerve repair remain far from ideal.
- Distal nerve pathway deterioration appears to be a primary problem and not muscle atrophy as previously thought. Prolonged denervation causes deterioration in the ability of Schwann cells to guide and promote nerve regeneration. With delayed regeneration, Schwann cells lose β1 subunits of the integrins, receptors for laminin, cell adhesion molecules, and p75 low-affinity neurotrophin receptors, all of which are important players in nerve regeneration.
- The last 2 decades have been marked by heightened interest in nerve regeneration. The successes of microscopic neurorrhaphy and autogenous nerve grafting, including interfascicular and cable grafting, have brought enthusiasm. However, the functional result is still far from perfect. Current areas of evolving interests include bridging the nerve gap with nonneural conduits, lateroterminal neurorrhaphy, nerve transfer, immunologic modulation, and deciphering the complexity and interplay at the molecular level of nerve regeneration.

### References

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Introduction

- Upper extremity chronic nerve compression is a common source of patient sensory and motor complaints. Symptoms present in the hand, forearm, arm, shoulder, or many combinations of these regions.
- Compression neuropathy implies a chronic condition, in contrast to acute compression of a peripheral nerve, as might be seen in trauma.
- Motor symptoms related to chronic compression range from weakness to paralysis.
- Sensory symptoms related to chronic nerve compression include numbness, tingling, and buzzing but do not include pain.
- Pain related to the peripheral nerve represents acute nerve compression (with axonal loss), a vascular infarct of a nerve, or a systemic neuropathy.
- With chronic nerve compression, the perceptions of temperature and pain are the last to be lost, whereas perceptions related to touch, such as movement, vibration, and pressure, are the first to become impaired (Box 16–1).
- Clinical presentations and syndromes related to the upper extremity peripheral nerves are related to the particular sensory and motor territories supplied by the given nerve and the location along that nerve’s anatomic pathway at which one or more compression sites occur.

Box 16–1  Pain Related to the Peripheral Nerve

- Pain most often implies direct nerve trauma.
- Pain can be caused by a vascular infarct of a nerve.
- Pain can be caused by acute nerve compression, e.g., crush.
- Pain usually is not related to chronic compression.
- Pain in bilateral extremities may represent neuropathy.
- Pain, bilaterally, with normal two-point discrimination is small-fiber neuropathy.
- Pain and temperature sensation, which are small-fiber perceptions, are the last perceptions to become abnormal in chronic nerve compression. Touch sensations, such as movement and pressure, which are large-fiber perceptions, are the first to become abnormal.
- Absence of pain perception with normal two-point discrimination is a syrinx.
- Absence of pain perception with abnormal two-point discrimination may be leprosy.

Pathophysiology of Chronic Nerve Compression

- Understanding the pathophysiology of chronic nerve compression permits an appreciation for how the results of physical examination change over time and how patient care must change over time.1–3
Intraneural microvessels have decreased flow with just 20 mm Hg external compression.

Decreased blood flow causes neural ischemia, the source of paresthesias.

After approximately 2 months of mild compression, the blood-nerve barrier changes, causing intraneural edema, which increases the pressure upon the microvessels.

After approximately 6 months of compression, in the rat and subhuman primate models, large nerve fiber demyelination begins (fibers related to perception of touch and motor function).

After approximately 6 months of compression, perineurial thickening and interfascicular fibrosis begins.

After approximately 12 months of compression, axonal loss begins in these large fibers.

With continued external pressure upon a peripheral nerve, progressive structural changes occur that produce predictable clinical symptoms and physical findings.

Early in chronic nerve compression, symptoms (e.g., paresthesia) related to the sensory nerve occur and change in coordination with the motor nerve, but no physical manifestations are found on physical examination. In contrast, with acute compression, which implies a large increase in pressure upon the nerve over a short time frame, sudden loss of nerve function occurs and is associated with pain and abnormal sensory and motor findings within hours of the onset.

In the intermediate phase of chronic nerve compression, a combination of ischemic block to axonal transmission and structural changes to the nerve results in weakness of the motor nerve and increased threshold of the sensory nerve.

In the later phases of chronic nerve compression, there is axonal loss that for the motor system produces muscle wasting and ultimately paralysis and for the sensory nerve produces abnormal two-point discrimination and ultimately anesthesia.

Based upon the known pathophysiology of chronic nerve compression, a numerical grading system can be constructed to permit staging of nerve compression and to provide an evidence base for clinical decision making (Tables 16–1 to 16–3).

**History Taking**

- Listening to the patients’ complaints often gives sufficient information to make the correct diagnosis.
- Critical phrases related to upper extremity nerve compressions are given in Box 16–2.
- If the onset of symptoms was sudden or associated with a “cold” or pain episode, consider the cause to be Parsonage-Turner syndrome, a usually self-resolving inflammatory brachial plexopathy, not a nerve compression syndrome.4
- If the onset of symptoms was related to a work injury or other form of trauma, obtain the exact description of the injury.

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**Table 16–1: Pathophysiologic Basis for Peripheral Nerve Grading Scale**

<table>
<thead>
<tr>
<th>DEGREE OF SEVERITY</th>
<th>PATHOPHYSIOLOGY</th>
<th>CLINICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Blood-nerve barrier breakdown</td>
<td>Symptoms, no signs on PEX</td>
</tr>
<tr>
<td>Moderate</td>
<td>Demyelination abnormal threshold</td>
<td>Symptoms, signs of elevated cutaneous pressure or vibratory thresholds, and/or of weakness</td>
</tr>
<tr>
<td>Severe</td>
<td>Axonal loss decreased innervation density</td>
<td>Symptoms, signs of decreased innervation density (abnormal two point discrimination), and/or muscle wasting</td>
</tr>
</tbody>
</table>

**Table 16–2: Numerical Grading Scale: Median Nerve at the Wrist Level**

<table>
<thead>
<tr>
<th>NUMERICAL SCORE</th>
<th>SENSORY</th>
<th>MOTOR</th>
<th>DESCRIPTION OF IMPAIRMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td>Paresthesia, intermittent</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>3</td>
<td>Abnormal pressure threshold (Pressure-Specified Sensory Device)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;45 years old</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≤3 mm, at 1.0–20 gm/mm²</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>≥45 years old</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≤4 mm, at 2.2–20 gm/mm²</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Weakness, thenar muscles</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>7</td>
<td>Abnormal pressure threshold (Pressure-Specified Sensory Device)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;45 years old</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≤3 mm, at &gt;20.0 gm/mm²</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>≥45 years old</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≤4 mm, at &gt;20.0 gm/mm²</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td>10</td>
<td>Paresthesias, persistent</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td></td>
<td>Abnormal innervation density (Pressure-Specified Sensory Device)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;45 years old</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≥4 mm and &lt;8 mm, at any gm/mm²</td>
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<td></td>
<td></td>
<td></td>
<td>≥45 years old</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≥5 mm and &lt;9 mm, at any gm/mm²</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Muscle wasting (1–2/4)</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>10</td>
<td>Abnormal innervation density (Pressure-Specified Sensory Device)</td>
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<td></td>
<td></td>
<td></td>
<td>&lt;45 years old</td>
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<td></td>
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<td></td>
<td>≥8 mm, at any gm/mm²</td>
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<td></td>
<td>≥45 years old</td>
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<tr>
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<td></td>
<td>≥9 mm, at any gm/mm²</td>
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<tr>
<td>9</td>
<td></td>
<td></td>
<td>Anesthesia</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td></td>
<td>Muscle wasting (3–4/4)</td>
</tr>
</tbody>
</table>
incident and time course of symptoms since the injury or trauma.

- If the symptoms have been present for more than 6 months and are slowly progressing, nerve compression is likely.
- If both feet are involved, the upper extremity complaints must be viewed in the context of a systemic neuropathy, such as diabetic neuropathy.
- The most common causes of nerve compression related to neuropathy in the United States are given in Box 16–3.

### Observation of the Hand with Regard to Nerve Compression

- Observe the appearance of the hand, looking particularly for evidence that the patient has been using the hand at work (e.g., calluses or dirt beneath the fingernails) in order to obtain some idea of hand function.
Observe for unusual or protective clothing over the hand (e.g., a glove).
Observe for any scars.
Observe for any skin or joint conditions that may be related to a systemic disease (see Box 16–3).

Physical Examination Related to Upper Extremity Peripheral Nerves

Motor
- Examine muscle strength related to a muscle(s) that is(are) unique for a given peripheral nerve (Box 16–4).
- Examine the muscle by manual muscle testing, grading it on a strength scale such as that given in Table 16–4.
- Determine whether the patient is cooperating by giving maximal effort during the examination.
- If possible, specific strength measurements should be made with an instrument, noting dominant and nondominant sides.
- Pinch strength can be measured as either lateral key pinch, which is the preferred method, or between the thumb and one or more fingertips. There is no preferred device, although computer-linked devices that permit data entry directly to a printed report are helpful in reporting results.

Table 16–4: Staging Nerve Compression: The British System

<table>
<thead>
<tr>
<th>Sensory Recovery Within the Autonomous Zone of the Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>S0</td>
</tr>
<tr>
<td>S1</td>
</tr>
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<td>S1+</td>
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<td>S2</td>
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<td>S3</td>
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<td>S3+</td>
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<td>S4</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Motor Recovery of Muscles Innervated by the Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>M0</td>
</tr>
<tr>
<td>M1</td>
</tr>
<tr>
<td>M2</td>
</tr>
<tr>
<td>M3</td>
</tr>
<tr>
<td>M4</td>
</tr>
<tr>
<td>M5</td>
</tr>
</tbody>
</table>

Grip strength should be measured with the patient seated, the elbow at the side and at 90 degrees, and forearm in a neutral position.

Hydraulic dynamometers, of the Jamar™ type, have interinstrument variability because of fluid loss over time and need to be recalibrated. Their measured force is calculated from the center of their curved handle. A systematic error occurs if there is an absent index finger (strength shifts ulnarly) or an ulnar nerve compression (strength shifts radially). The Digit-Grip™ has a straight handle for grasping and records force uniformly across the handle because it has a force transducer at each end of the handle, thus eliminating error. Computer-linked devices are available that print directly for report documentation.

Sensory
- Sensibility implies the neuroanatomy (nerve ending and nerve fiber). Sensation implies the cortical interpretation of the transmitted nerve impulse.
- By definition, all testing of sensibility is subjective because the person being tested must give an interpretation of the test stimulus.
- Measurements of cutaneous thresholds for temperature, vibration, and pressure have been termed in neurology

Box 16–4 Nerve-Specific Muscle Testing

<table>
<thead>
<tr>
<th>Muscle</th>
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</thead>
<tbody>
<tr>
<td>Median nerve at wrist</td>
</tr>
<tr>
<td>Abductor pollicis brevis, opponens pollicis brevis (with other median-innervated muscles being normal)</td>
</tr>
<tr>
<td>Median nerve in forearm</td>
</tr>
<tr>
<td>Flexor pollicis longus, flexor profundus to index finger, pronator teres, flexor superficialis</td>
</tr>
<tr>
<td>Ulnar nerve at wrist</td>
</tr>
<tr>
<td>Abductor digiti minimi, first dorsal interosseous (with flexor profundus to little finger being normal)</td>
</tr>
<tr>
<td>Ulnar nerve at elbow</td>
</tr>
<tr>
<td>Flexor profundus to little finger, flexor carpi ulnaris (with weakness in ulnar innervated intrinsics)</td>
</tr>
<tr>
<td>Radial nerve at elbow</td>
</tr>
<tr>
<td>Extensors of wrist, thumb, and all fingers</td>
</tr>
<tr>
<td>Radial nerve proximal to elbow</td>
</tr>
<tr>
<td>Brachioradialis, triceps</td>
</tr>
<tr>
<td>Musculocutaneous nerve</td>
</tr>
<tr>
<td>Biceps</td>
</tr>
<tr>
<td>Axillary nerve</td>
</tr>
<tr>
<td>Deltoid</td>
</tr>
<tr>
<td>Suprascapular nerve</td>
</tr>
<tr>
<td>Supraspinatus, infraspinatus</td>
</tr>
<tr>
<td>Spinal accessory nerve</td>
</tr>
<tr>
<td>Trapezius</td>
</tr>
<tr>
<td>Long thoracic nerve</td>
</tr>
<tr>
<td>Serratus anterior (scapular winging)</td>
</tr>
</tbody>
</table>
quantitative sensory testing. They have been demonstrated to be reliable and valid because they are reproducible and correlate with patient symptoms. The measurements are also termed neurosensory testing.

- The skin territory to be tested should be selected in relation to the unique area innervated by the peripheral nerve being evaluated (Box 16–5).
- The only time testing for pain perception with a pin is necessary is if you believe the patient is malingering and states he or she has no feeling in a finger, or if you suspect a syrinx, with involvement of the anterolateral spinothalamic tract (carries pain and temperature perception), in which case there will be diminished pain perception with normal two-point discrimination and intrinsic muscle wasting. Small-fiber function, like pain perception, is the last to be lost with compression neuropathy.
- The only time testing for temperature perception is necessary is if you believe a small-fiber neuropathy is the source of the pain. Pain symptoms predominate, and large-fiber functions related to touch are normal. Thermal threshold testing is indicated. Small-fiber function, like temperature perception, is the last to be lost with compression neuropathy.
- Perception of touch, a large-fiber function, is the first to become involved with compression neuropathy and includes perception of moving touch, constant touch, and vibration.
- Vibratory testing with a tuning fork is indicated in the emergency room setting to evaluate an injured nerve or in the office setting for a quick evaluation or screening of large-fiber function. For example, if the index finger pulp is perceived as “loud or strong” but the little finger pulp is perceived as “soft or weak,” then compression of the ulnar nerve is suspected.
- The high-frequency tuning fork (256 Hz) evaluates Pacinian corpuscle function. The low-frequency tuning fork (30 Hz) evaluates Meissner corpuscle function. At a sufficient stimulus intensity, any tuning fork evaluates the entire population of quickly adapting large fibers.
- Perception of movement is mediated by the quickly adapting fibers.
- The disadvantage of using a tuning fork is that the stimulus causes, such as vibrating hand-held instruments.
- The vibrometer changes the qualitative information of the tuning fork into quantitative information, measuring in terms of microns of motion or volts. Vibrometers can be single frequency or multiple frequency but still have the disadvantage that their stimulus input is a wave. Vibratory threshold measurements are valuable for research comparing populations of patients and evaluation of neuropathy related to certain industrial causes, such as vibrating hand-held instruments.
- Perception of constant touch or static touch is mediated by the slowly adapting fibers. These fibers are also large myelinated fibers whose receptors are the Merkel-Cell neurite complex located along the intermediate ridge of the dermal papillae.
- Pressure perception is mediated by the slowly adapting fibers because they vary their frequency of impulse transmission in response to varying stimulus intensity.
- Measurement of cutaneous pressure threshold is the most critical for evaluation of compression neuropathy because the stimulus is perceived only through the unique piece of skin tested and because large fiber function is the first to change with compression of a peripheral nerve.
- The nylon monofilaments, described by Semmes and Weinstein in 1960, give an estimate of a range for the cutaneous pressure threshold. The number on the monofilament handle is a logarithm of the force in tenths of milligrams. This force must be divided by the cross section of the filament to give pressure. The filaments vary by 10% in their calibration at the time they are purchased and lose up to 10% of their reliability after 100 uses. The filaments are a set, a discontinuous set, and therefore they give an estimate of a range, and not a true measurement. The true measurement is somewhere between two filaments.
- Two-point discrimination requires a high innervation density, that is, a large number of innervated sensory
The pattern of sensory abnormality varies during progressive compression neuropathy because a large number of nerve fibers must have either an ischemic conduction block or axonal degeneration. Measurement is achieved with a paper clip and ruler, an engineering gauge with rounded tips, or a Disk-Criminator™.

- Two-point discrimination changes late in the course of compression neuropathy. Measurement from the Pressure-Specified Sensory Device™ is as sensitive and specific as electrodiagnostic testing for carpal tunnel syndrome.11
- In a blinded, prospective study, neurosensory testing with the Pressure-Specified Sensory Device was as sensitive and specific as electrodiagnostic testing for carpal tunnel syndrome.11
- The pattern of sensory abnormality varies during progressive compression of the peripheral nerve and neural regeneration after nerve decompression (Box 16–6).

### Other Physical Examination Techniques

- **Provocative maneuvers** increase pressure upon the nerve, eliciting symptoms during the office visit, such as flexion (Phalen sign) in carpal tunnel syndrome, elbow flexion for ulnar nerve compression at the elbow, digital pressure upon the median nerve in the forearm for pronator syndrome, and pressure upon the brachial plexus for thoracic outlet syndrome.
- **Tinel sign** originally was defined as distally radiating paresthesias elicited by tapping over a regenerating nerve. Distal progression of the point of maximal response is indicative of nerve regeneration. Today, it is taken as a sign of demyelination or injury to the nerve related to chronic compression at known sites of anatomic narrowing.
- In the **tourniquet test**, application of a tourniquet to the upper arm causes ischemia distally. Ischemia affects the large nerve fibers first because of the longer distance required for oxygen to diffuse into them. If there is a median nerve compression, the first fingers to become symptomatic are the thumb and index finger. If there is an ulnar nerve compression, the first fingers to become numb is the little finger.
- Each of these tests has important problems with sensitivity and specificity and must be interpreted in the overall clinical context. Nerve compression can be present in the absence of a positive Tinel sign, and a positive Tinel sign can be present in an individual who is asymptomatic.

### Staging Degree of Nerve Compression

- Clinical decision making regarding treatment must be based upon the degree to which the nerve is compressed.5,12
- Traditional descriptive staging is given in Table 16–4.
- Nonoperative treatments are indicated early in nerve compression.
- As pathophysiology progresses and structural changes occur within the nerve and between the nerve and its anatomic relations, the measurements of nerve function change, indicating surgical intervention is necessary and suggesting the time course and potential for clinical improvement (for correlations see Table 16–1).
- Staging of degree of nerve compression can be either descriptive category (e.g., minimal, moderate, severe) or numerical (e.g., on a scale from 0–10).
- Staging of nerve compression should be used for reporting of clinical results.
- Numerical grading scales for the median nerve at the wrist and the ulnar nerve at the elbow are given in Tables 16–2 and 16–3.
Electrodiagnostic Testing

- Electrodiagnostic testing consists of the electrical stimulation of the skin, muscle, or peripheral nerve and the recording of the transmitted neural impulse or muscle contraction in response to that electrical stimulation.
- Nerve conduction velocity (NCV) and electromyography (EMG) are the traditional tests used for measuring sensory and motor peripheral nerve function.
- When the site being tested is sufficiently distal such that two separate stimulation points cannot be obtained to obtain a velocity (distance/time) measurement, then just the time required to get the response (distal latency) is recorded.
- In compressive neuropathy, demyelination slows nerve conduction and increases distal latency. With time, axonal loss causes a decrease in the amplitude of the recorded sensory or motor potential.
- Electrodiagnostic testing is objective because no patient interpretation is needed to obtain the measurement.
- EMG testing is essential for diagnosis of cervical radiculopathy because individual muscle patterns of involvement can be correlated with cervical nerve roots.
- EMG is essential for diagnosis of primary myopathy in patients with diffuse weakness or with diseases of central nervous system origin, such as myasthenia gravis, amyotrophic lateral sclerosis, or spastic hemiplegia.
- Electrodiagnostic testing has been available since the 1950s and is widely accepted for peripheral nerve testing. However, it has significant problems for upper extremity peripheral nerve surgery.\textsuperscript{13,14}
- To localize a site of compression, the NCV must demonstrate a site of slowing, which implies that more than one site must be stimulated. This is not possible for the brachial plexus or for the median nerve at the wrist. This is compensated by the “inching technique” of Kimura in the palm, but not for the brachial plexus.
- Even for median nerve compression at the wrist, where NCV/EMG is the most specific and most sensitive, a recent meta-analysis demonstrated a 33% false negative rate.\textsuperscript{15}
- In the presence of a peripheral neuropathy, like diabetes, NCV/EMG is sufficiently unreliable to demonstrate superimposed nerve compression that reliance upon the physical examination (positive Tinel sign) is recommended.\textsuperscript{16}
- NCV/EMG in the postoperative period after nerve decompression is often unreliable because remyelination of the peripheral nerve is usually incomplete.
- Studies of patients who underwent nerve decompression show no difference in outcome regardless of whether the patients had a positive or negative electrodiagnostic study prior to surgery.\textsuperscript{17,18}
- If surgery is performed in a patient with a “normal” NCV/EMG and a postoperative complication occurs, a difficult medical legal situation may arise. Abnormal sensory and motor function can be documented with neurosensory and manual motor testing sufficient to indicate the need for surgery.

Peripheral Nerve Surgery

Technical Guidelines

- A series of textbooks related to peripheral nerve surgery is available.
- James Learmonth, MD, who perhaps did the first median nerve decompression at the wrist and the first submuscular transposition of the ulnar nerve at the elbow, can be considered the first peripheral nerve surgeon.\textsuperscript{19}
- The American Society for Peripheral Nerve was established in 1990.
- Use of the tourniquet in upper extremity surgery to create a bloodless field is essential for appropriate visualization.
- Use of the bipolar coagulator is critical for gentle microsurgical technique.
- Using loupe magnification is necessary for safety in peripheral nerve surgery.
- Simply removing the external compressive upon a peripheral nerve and separating the peripheral nerve from its adjacent structures is considered a decompression or external neurolysis.
- Continuing to operate within the peripheral nerve itself, through the epineurium with or without interfascicular neurolysis, called an internal neurolysis, remains controversial.
- A meta-analysis of internal neurolysis in median nerve decompression at the wrist demonstrated no significant benefit of internal neurolysis.\textsuperscript{20}
- To date, all studies on the benefit of internal neurolysis have used preoperative analysis of staging or randomization; they have not included patients with recurrent nerve compression.
- These studies do not give adequate advice to the surgeon who, during the course of a nerve decompression, identifies a firm, thickened peripheral nerve that has no fascicular pattern. Intraoperative identification of pathology may be sufficient justification for additional intraneural dissection.

Nerve Compression Syndromes

CARPAL TUNNEL SYNDROME

- Carpal tunnel syndrome is the most common compression neuropathy in the hand.
The median nerve is compressed in the carpal tunnel, which has the transverse carpal ligament as the roof. The tunnel contains nine flexor tendons, their synovium, and the median nerve.

Numbness or paresthesias most commonly occur in the index and middle finger, followed by the thumb. Complaints about the ring finger are uncommon.

Nighttime awakening is almost universal, as the stronger flexor muscle mass pulls the wrist into flexion during sleep.

Thenar muscle wasting is a classic sign but is rarely seen today because the medical community and the lay public are so aware of carpal tunnel syndrome symptoms.

Preserved short flexor function results from ulnar nerve innervation of this muscle.

Dropping objects and weakness are rare unless median compression is advanced, so these symptoms often reflect a coexisting cubital tunnel syndrome.

Incidence of carpal tunnel syndrome is 2% in general population, 14% in diabetics, and 30% in diabetics with neuropathy.\(^{16}\)

Nonoperative treatment is splinting with the wrist in neutral position. Note that most shelf splints have the wrist at 20 degrees dorsiflexion and should be bent into the wrist neutral position to reduce pressure on the median nerve.

Nonoperative treatment should consist of changes in the activities of daily living and nonsteroidal antiinflammatory medication for 3 months in addition to splinting.

Cortisone injection into the synovial tissues of the carpal tunnel is appropriate and provides at least temporary relief in up to 80% of patients.\(^{21}\) Cortisone injection can be done from the proximal to the distal wrist crease and slightly ulna to it (Figure 16–1).

Nonoperative treatment is indicated in each patient. However, if staging the degree of compression demonstrates the patient is in an advanced category, then the chance of successful nonoperative treatment is small.

In patients with an advanced degree of compression, surgical decompression of the median nerve is indicated.

Surgical decompression of the median nerve at the wrist has a high percentage of good to excellent results, regardless of whether an open or endoscopic decompression technique is used (Figures 16–2 and 16–3).\(^{22}\)

In the open technique, the incision must be made along the radial border of the ring finger to avoid injury to the palmar cutaneous branch of the median nerve. Injury to this nerve causes a painful postoperative scar without radiation to the fingers. It is important to protect the recurrent motor branch of the median nerve. This can be identified using the landmarks described in Figure 16–3. The median nerve can be located by determining the intersection of Kaplan's line and a line bisecting the index-middle finger web space.

Decompression routinely includes division of the distal antebrachial fascia as it thickens to form the transverse carpal ligament, because this process can compress the median nerve against the volar surface of the radius.

Postoperative immobilization of the wrist in neutral position for up to 1 week is indicated to prevent bowstringing of the flexor tendons out of the carpal canal.

Early movement of the fingers to create gliding of the median nerve across the wrist operative site is indicated to minimize adhesions during the healing process.

Improvement following carpal tunnel decompression is so typical that failure to improve suggests additional problems may be present.

Steroid cream massaged into the scar from the sixth week to the third month is helpful in minimizing the scar.
Differential diagnosis of the patient who does not improve after carpal tunnel decompression is given in Box 16–7.

The double crush concept applies to carpal tunnel syndrome, in that compression anywhere or at multiple sites along the axis of C6 can summate to cause symptoms, even though compression at one site alone would not be sufficient to cause symptoms. Carpal tunnel syndrome patients often have symptoms that go into their neck and shoulder. If these symptoms persist after carpal tunnel decompression, a more proximal source of compression exists.

**Figure 16–2:**
Kaplan’s line is drawn along the ulna border of the abducted thumb. The motor branch is identified as the intersection of Kaplan’s line in a line drawn longitudinally in the web space of the index and middle fingers.

- Differential diagnosis of the patient who does not improve after carpal tunnel decompression is given in Box 16–7.
- The double crush concept applies to carpal tunnel syndrome, in that compression anywhere or at multiple sites along the

**Figure 16–3:**
A carpal tunnel incision made along a line drawn proximally from the radial border of the ring finger avoids injury to the recurrent motor branch and the palmar cutaneous motor branch of the median nerve.

**Figure 16–4:**
The hamate sound helps dilate the carpal canal to prepare for insertion of the endoscopic device.

**Figure 16–5:**
The important anatomic structures to consider when using the single portal endoscopic device include the transverse carpal ligament, the superficial palmar arch, and the ulna border of the median nerve, particularly the common digital nerve to the third web space.

**Box 16–7**
Differential Diagnosis of Failed Carpal Tunnel Surgery

- Recurrent or persistent carpal tunnel syndrome
- Proximal median nerve compression in the forearm
- Radial sensory nerve entrapment in the forearm
- Upper trunk brachial plexus compression
- Cervical radiculopathy (C5/C6)
**MEDIAN NERVE COMPRESSION IN THE FOREARM**

- The median nerve at the elbow level contains all the fibers that transverse the carpal tunnel, the motor fibers to the radial wrist flexors, finger flexors (except the flexor profundus to the little and ring fingers), and forearm pronators, and sensory fibers to the palm and volar wrist joints.
- Median nerve compression in the forearm has been subdivided historically into the *anterior interosseous nerve syndrome* and the *pronator syndrome*.
- Proximal median nerve compression is commonly seen in patients whose job requires repetitive flexion and pronation of the forearm.
- Proximal median nerve compression is often seen in association with other compression neuropathies, such as carpal and cubital tunnel syndrome. In the setting of a worker using both hands, proximal median nerve compression may appear as a bilateral problem or a systemic neuropathy.
- Classically, the pronator syndrome has little motor deficit and primarily results in symptoms of numbness in the hand with the median-innervated fingers.\(^{23}\)
- Classically, the anterior interosseous nerve syndrome results in weakness and/or paralysis of the flexor pollicis longus and the flexor profundus to the index finger and no sensory symptoms.
- The double crush concept applies to the median nerve in the region of the proximal forearm and elbow in that, depending upon the topographic organization of fascicles in the median nerve and the anatomic variables of the muscle origins, many patients have so-called incomplete or mixed syndromes.
- Although electromyographic studies can identify denervation of the muscles innervated by the median nerve, often there is a combination of forearm flexors and pronator teres abnormalities, or the test is interpreted as normal.
- Neurosensory testing of the thenar eminence with the Pressure-Specified Sensory Device can identify involvement of the palmar cutaneous branch of the median nerve, documenting compression of the proximal median nerve.\(^{24}\)
- The anatomic structures that can be responsible for compression of the median nerve proximally, and therefore the structures that must be evaluated intraoperatively and divided, are given in Box 16–8.
- The anterior interosseous nerve is at risk for compression if the deep head of the pronator teres is fibrous and if the nerve fascicle originates on the radial side instead of the deep (dorsal) side of the median nerve.\(^{25}\)
- The terminal branches of the anterior interosseous nerve innervate the volar wrist joint. Therefore, complaints of volar wrist pain may accompany compression of the median nerve in the forearm.
- Pain referred to the proximal forearm with resisted middle or ring finger proximal interphalangeal joint flexion indicates the presence of an arch between the superficialis muscles crossing the median nerve. This often is a separate arch or site than is the deep head of the pronator teres.
- The ligament of Struthers is exceedingly rare, whereas the lacertus fibrosis is constant (Figure 16–6).
- Nonoperative management includes changing activities of daily living and, in the work environment, may require an ergonomic evaluation and job rotation.
- Splinting and cortisone injections usually are not helpful.
- Surgical approach should permit exposure from the medial elbow to the midforearm but does not have to incorporate a long zigzag incision, except where crossing the antecubital crease is necessary (Figure 16–7).
- With the surgical approach, care should be taken not to injure either the medial or the lateral antebrachial cutaneous nerve, which can be a source of a painful scar.

**Box 16–8: Anatomic Structures Causally Related to Median Nerve Compression in the Forearm**

- Ligament of Struthers (humerus to medial humeral epicondyle)
- Lacertus fibrosis
- Deep head of the pronator teres
- Fibrous arch between heads of the superficialis muscles

![Figure 16–6:](image)

The ligament of Struthers bridges the supracondylar process of the humerus to the medial epicondyle or the origin of the humeral head of the pronator teres.
Figure 16–7:
Decompression of the proximal median nerve involves release of the lacertus fibrosis, the humeral head of the pronator teres, the vascular leash proximal to the flexor digitorium sublimis (FDS), the FDS and its fascia. FDP, Flexor digitorium profundus.
• Immediate postoperative use of the arm in terms of flexion, pronation, and supination is essential to prevent scarring of the median nerve.
• In the muscular forearm, postoperative muscle bulk contributes to hypertrophic scarring. A steroid cream massaged into the scar for 6 weeks beginning at postoperative week 3 can be effective in minimizing the scar.
• If the surgery is to include an ulnar nerve transposition, then exposure of the lacertus fibrosis and sometimes the deep head of the pronator teres can be accomplished through the medial elbow incision, minimizing the length required for the volar incision.

Ulnar Nerve Compression at the Wrist

• Ulnar nerve compression at the wrist is termed the ulnar tunnel syndrome.
• Ulnar nerve compression at the wrist can accompany carpal tunnel syndrome, as demonstrated by abnormal distal ulnar nerve findings when electrodiagnostic studies are performed for carpal tunnel syndrome.
• The ulnar nerve and ulnar artery plus a collection of fat occupy a space described by Guyon. There is a thin but firm roof bridging from the palmar fascia, the palmaris brevis muscle (present in 20%), and the hamate toward the pisiform. The deep surface of the ulnar tunnel is the pisohamate ligament.26

• The shape of the ulnar tunnel in cross section changes from triangular to round and increases in area when the transverse carpal ligament is divided. Therefore, the ulnar tunnel should not be opened routinely during carpal tunnel surgery.
• Symptoms related to ulnar nerve compression at the wrist always include numbness of the little finger pulp and sometimes the ring pulp, but not the dorsum of the hand, and variable degrees of intrinsic muscle weakness or loss of coordination of the fingers.
• The double crush concept suggests that if a patient has cubital tunnel syndrome or a systemic neuropathy, the ulnar nerve may be sufficiently compressed in the ulnar tunnel so as to contribute to ulnar-sided hand symptoms.
• Traditionally, ulnar nerve compression at the wrist occurs distal to the hook of the hamate, causing compression of just the motor branch. Clawing and first dorsal interosseous muscle wasting may occur in the presence of a normal hypothenar muscle mass and function and normal sensibility in the little finger.
• The most common cause of compression of the ulnar nerve at the wrist level is a lipoma or a ganglion. Symptoms of ulnar tunnel syndrome vary, depending on the site of the compression (Figure 16–8).

Figure 16–8:
The ulna tunnel can be divided into three sections: Lesions in zone I often produce a combined sensory and motor deficit. Lesions in zone II, cause a pure motor deficit. Lesions in zone III cause pure sensory deficits. (This is from Figure 18–10 on page 330 in the textbook.)
Twenty-five percent of patients have a fibrous origin of the hypothenar muscles that can cause compression of the deep motor branch.

Inflammation of the pisotriquetral joint may cause ulnar nerve symptoms, as may occlusion of the ulnar artery or an ulnar artery aneurysm.

Work conditions (e.g., using a jackhammer) or sports activities (e.g., bicycling) can cause ulnar nerve compression at the wrist.

Two classic sports injuries—hitting the ground with a golf club or hitting a baseball—can cause fractures of the hook of the hamate with an associated ulnar nerve compression. Special imaging may be required to demonstrate this fracture.

Surgical decompression of the ulnar nerve at the wrist can be performed through the typical incision for carpal tunnel decompression. Care should be taken to trace out the course of the motor branch to make sure that the decompression is complete.

Cubital Tunnel Syndrome

Ulnar nerve compression in the cubital tunnel is the second most common nerve entrapment in the upper extremity.

The ulnar nerve lies within the postcondylar groove, where it is covered by a relatively fibrous roof going from the medial humeral epicondyle to the olecranon.

Prior to about 1959, this syndrome almost always was related to fracture or dislocation about the elbow, and the syndrome was called posttraumatic ulnar palsy. With improvement in the care of this trauma, so-called idiopathic ulnar neuritis continued and was identified by Osborne as the ulnar nerve being compressed beneath the fibrous arch joining the two heads of the flexor carpi ulnaris. He called this syndrome tardy ulnar palsy. The name cubital tunnel syndrome was given to the symptom complex by Stafford and Feindel in 1973.

With flexion of the elbow greater than 30 degrees, the shape of the tunnel narrows distally, at Osborne’s band, causing pressure upon the ulnar nerve. This pressure increases through 120 degrees of elbow flexion and then increases further with shoulder abduction, as the medial cord origins of the ulnar nerve are stretched.

The double crush concept applies to the ulnar nerve in that compression of the lower trunk of the brachial plexus in the thoracic outlet syndrome often is present in patients with cubital tunnel syndrome. The C8 potential contribution to these multiple sites of compression along the same nerve is less common than compression at the most distal site, Guyon’s canal, at the wrist.

Sensory symptoms resulting from cubital tunnel syndrome usually include numbness in the little and ring finger that worsens with use of the hand requiring elbow flexion but does not occur at night unless the person sleeps with the arm beneath the pillow.

Motor symptoms include weakness, clumsiness, and dropping objects.

For musicians, the symptoms are related to difficulty in using the fingers either as fast, or for as long, as they used to, especially for violin, guitar, and piano players. These symptoms in musicians may be mistaken for “spasticity” or “dystrophy.” They usually are helped by relaxation techniques and stretching, such as Pilates, and changing the practice schedule. Surgical decompression is rarely required.

When determining treatment for cubital tunnel syndrome, staging the degree of compression is critical (see Table 16–1).

Nonoperative treatment consists almost entirely of avoiding elbow flexion beyond 30 degrees for up to 3 months. Such treatment has been demonstrated to result in improvement in 80% of patients with a mild degree of compression but in less than 30% of those with a severe degree of compression. The treatment must be done every night using a towel wrapped around the arm to prevent extreme elbow flexion; however, during the day, critical activities that require elbow flexion are allowed.

Electrodiagnostic studies that show conduction velocity slowing across the elbow less than 50 m/s or a decrease of more than 10 m/s from the conduction velocity below the elbow or the contralateral side is evidence of cubital tunnel syndrome. However, the false-negative rate is approximately 50%.

If patients have symptoms of cubital tunnel syndrome after more than 3 months of nonoperative treatment and they have documented neurosensory and motor evidence of abnormal ulnar nerve function and a positive Tinel sign in the postcondylar groove, then surgical decompression is indicated.

No single operation for ulnar nerve decompression at the elbow is universally accepted.

Box 16–9 lists the five operations commonly used for cubital tunnel syndrome.

A study measuring intraneural ulnar nerve pressures in fresh cadavers demonstrated that the only technique that decreased the pressure on the ulnar nerve proximal to,

**Box 16–9** Operations for Cubital Tunnel Syndrome

- In situ (simple) decompression
- Open or endoscopic
- Medial humeral epicondylectomy
- Anterior subcutaneous transposition
- Anterior intramuscular transposition
- Anterior submuscular transposition
- Learmonth technique (reattachment of muscle origin in situ)
- Dellon musculofascial lengthening (Z-lengthening)
across, and distal to the elbow, and for all ranges of motion of the elbow, was the anterior submuscular transposition using the Z-lengthening or musculofascial lengthening technique described by Dellon et al.29

- No randomized prospective study has identified which procedure has the best outcome in preoperatively staged patients (see Table 16–1).
- Two meta-analyses, one decade apart, arrive at the same conclusion: there is a high percentage of recurrent or failed ulnar nerve decompressions at the elbow; approaching 25% to 33% for the different techniques.30,31 The best consistent results are achieved for anterior submuscular transposition by the musculofascial lengthening technique.31

- The most common anatomic structures responsible for failure of ulnar nerve surgery at the elbow are listed in Table 16–5. Failure to address each of these structures creates a situation in which a new site of increased pressure upon the ulnar nerve can occur postoperatively. Surgical options include:
  - In situ decompression: In this technique, the roof of the cubital tunnel and Osborne’s fascia is released from just posterior to the medial epicondyle distally through a small curved incision. The goal is to leave the roof of the cubital tunnel proximal to the medial epicondyle intact in order to prevent anterior subluxation during flexion. If the nerve subluxes following this in situ release, a formal anterior transposition should be performed.
  - Medial epicondylectomy: In this approach, the goal is to remove the medial epicondyle, which forms the anterior wall of the cubital tunnel. Care must be taken to expose the nerve, release the ligament, and identify the medial collateral ligament inserting on the medial epicondyle. Muscle origins of the FCU in pronator teres have to be elevated from the medial epicondyle in order to provide an adequate release and then these are reattached to the adjacent periosteum (Figure 16–9).

- Anterior subcutaneous transposition: In this technique, the roof of the cubital tunnel and Osborne’s ligament are released through a curvilinear incision. Care must be taken to expose and excise the medial intermuscular septum for approximately 10 cm proximal to the medial epicondyle, in order to avoid a secondary site of compression. A neurolysis is performed, and the ulnar nerve is transposed anteriorly onto the fascia of the flexor-pronator mass. In order to prevent the nerve from relocating back into the cubital tunnel, a fascial flap is elevated from the anterior surface of the flexor-pronator mass as demonstrated in Figure 10. This flap is then sutured to the superficial fascia, providing a barrier against relocation (Figures 16–10 and 16–11). The anterior submuscular transposition is performed using a curvilinear incision made just posterior to the cubital tunnel to protect the medial brachial cutaneous nerve. After release of the cubital tunnel ligament, 10 cm intramuscular septum is removed and Osborne’s fascia between the two heads of the FCU is incised. A blunt curved clamp is placed under the origin of the FCU to protect the median nerve. The clamp should be placed proximal to the median nerve’s first branch, the motor branch to the pronator teres. The FCU origin is divided in a step-cut fashion, leaving a cuff of tendon for reattachment of the muscle origins. All septae under the FCU need to be divided to allow anterior transposition of the ulna nerve parallel to the median nerve. Frequently, the posterior motor branch to the FCU has to be mobilized from the ulna nerve for several centimeters by internal neurolysis to complete the transposition.
- The incision for ulnar nerve decompression almost always crosses a branch of the medial brachial cutaneous nerve.

![Figure 16–9:](image)

The medial epicondylectomy is performed with an osteotome. Intra-operative radiographs are required to ensure adequate resection of the medial epicondyle. FCU, flexor carpi ulnaris.

<table>
<thead>
<tr>
<th>Table 16–5: Critical Anatomic Structures that Must be Treated to Prevent Recurrent Ulnar Nerve Compression at the Elbow</th>
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<tbody>
<tr>
<td><strong>ANATOMIC STRUCTURE</strong></td>
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<tr>
<td>Roof of cubital tunnel</td>
</tr>
<tr>
<td>Fascia of flexor carpi ulnaris</td>
</tr>
<tr>
<td>Medial intermuscular septum</td>
</tr>
<tr>
<td>Fascia from medial head of triceps</td>
</tr>
<tr>
<td>Anomalous origins of triceps</td>
</tr>
<tr>
<td>Epitrochlearis anconeus</td>
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<tr>
<td>Arcade of Struthers</td>
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</table>
Direct injury to this nerve, stretching, or adherence to the medial humeral epicondyle after surgery causes a painful scar. Pain posterior to the medial humeral epicondyle may be a neuroma of the medial brachial cutaneous nerve.32

- The painful postoperative scar is treated with topical steroid massage into the scar, ultrasound, and, if this fails, resection of the neuroma and implantation of the nerve proximally into the triceps muscle.
- When evaluating the postoperative patient who complains of pain in the elbow region, remember that the ulnar nerve does not innervate the elbow. This symptom indicates a neuroma of the medial antebrachial or medial brachial cutaneous nerve. You must find a Tinel sign that radiates to the little finger to diagnose recurrent or failed ulnar nerve compression at the elbow. A painful trigger point or Tinel sign that only hurts at the elbow represents a neuroma in that elbow location.

Figure 16–10:
The anterior subcutaneous transfer of the ulna nerve relies on a fascial sling created from the fascia of the flexor pronator origin to prevent the nerve from returning to the cubital tunnel. FCU, flexor carpi ulnaris.

Radial Sensory Nerve Compression
- The original article by Wartenberg, translated from the 1932 report in German, described what was believed to be an inflammation of the radial sensory nerve, not a compression neuropathy. He described the provocative maneuver of forearm pronation that elicited symptoms and pain in the distribution of this nerve with sudden ulnar deviation of the wrist.
- This syndrome is found in the setting of trauma, with crush injury, or with use of an external fixation device in the forearm.
- The radial sensory nerve is compressed in pronation by the movement of the extensor carpi radialis longus toward the brachioradialis tendon, at the site where the nerve transits from deep to superficial. Sometimes there is a small sheath binding the radial sensory nerve to the deep surface of the brachioradialis. Sometimes the nerve exits through the brachioradialis tendon.

Figure 16–11:
Medial view of the elbow. The anterior submuscular transposition requires the release of the intermuscular septum, the cubital tunnel, and a division of the flexor pronator origin to transpose the nerve anteriorly. FCU, flexor carpi ulnaris.
Phenomenon 1: Symptoms are just sensory and consist of a burning, paresthesia, or numbness along the dorsoradial aspect of the hand.

Phenomenon 2: These symptoms can be described simply as numbness in the thumb and index finger, leading to a misdiagnosis of carpal tunnel syndrome.

Phenomenon 3: Radial sensory nerve crosses the wrist joint while being tethered proximally. Ulnar wrist deviation, especially if the thumb is grasped, causes a shooting dorsoradial pain. This is similar to the positive Finkelstein sign with de Quervain tenosynovitis and must be distinguished from tendonitis by demonstrating the resisted thumb extension is not painful.

Phenomenon 4: The physical examination should identify the Tinel sign just posterior (lateral) to the insertion of the brachioradialis into the radius.

Phenomenon 5: The compression site is where the radial sensory nerve exits from deep to the fascia that joins the extensor carpi radialis longus to the brachioradialis.

Phenomenon 6: The surgical technique consists of releasing the fascia from the exit point of the radial sensory nerve as far proximal as necessary so that the nerve is no longer compressed with forearm pronation.

Phenomenon 7: During the dissection, care should be taken to not injure the overlying lateral antebrachial cutaneous nerve.

Phenomenon 8: Postoperative care requires immediate mobilization. Allow pronation/supination.

Radial Nerve Compression at the Elbow

Phenomenon 9: Traditionally, compression neuropathy of the radial nerve at the elbow is considered two different syndromes: the posterior interosseous nerve compression and the radial tunnel syndrome.

Phenomenon 10: The concept of the double crush is important for the posterior interosseous nerve compression neuropathy. The critical difference on physical examination is that with epicondylitis the tender site is at the lateral humeral epicondyle, at the extensor muscle origin, but with compressive neuropathy of the radial nerve the nerve itself is tender approximately 1.5 cm anterior and distal to the epicondyle.

Phenomenon 11: Symptoms with radial tunnel syndrome may give a distribution throughout the length of the radial nerve distally, including the forearm and dorsal wrist. A coexisting radial sensory nerve compression must be evaluated.

Phenomenon 12: Physical examination for radial tunnel syndrome should include resisted middle finger extension. This action forces the extensor carpi radialis brevis to contract to maintain the third metacarpal position, causing pressure on the radial nerve. Pain is referred to the region of the radial tunnel.

Phenomenon 13: The symptoms of posterior interosseous nerve “palsy” all are motor, with the exception of aching in the dorsal wrist, because the terminal branches of this nerve innervate the dorsal wrist capsule.

Phenomenon 14: The motor symptoms of posterior interosseous nerve palsy range from weakness of grasp to paralysis of individual or all muscles that extend the wrist and fingers.

Phenomenon 15: Surgical exposure for decompression of the radial nerve can either be anterior or posterior. Our preference is anterior (Figure 16–12).

Phenomenon 16: For this approach, a curvilinear incision is performed crossing the antecubital fossa. The interval between the brachialis and the brachioradialis is identified and bluntly dissected. The radial nerve is identified and traced distally to the Arcade of Frohse, which is the thickened anterior fascial edge of the supinator muscle. This fascia is split, as is the fascial edge of the extensor carpi radialis brevis, and any constricting vascular leash (see Figure 16–12).

Phenomenon 17: The posterior approach uses a curved incision along the posterior border of the mobile wad. The interval between the ECRB and EDC is identified and split longitudinally. The Arcade of Frohse, the thickened fascial edge of the supinator muscle, and the fascia over the proximal supinator muscle is identified and released in order to decompress the nerve. Through this approach, it is easier to decompress the nerve from distal to proximal, as branches of the posterior interosseous nerve can be identified exiting distal to the supinator muscle and traced proximally by splitting the muscle (Figure 16–13).

Phenomenon 18: Postoperatively, immediate mobilization is critical.

Brachial Plexus Compression (Thoracic Outlet Syndrome)

Phenomenon 19: Compression of the brachial plexus in the thoracic inlet traditionally was called scalenus anticus syndrome but more lately and incorrectly has been called thoracic outlet syndrome.
The symptoms of brachial plexus compression can be so universal in the neck, shoulder, and entire hand and arm that many neurologists continue to doubt the existence of neurologic thoracic outlet syndrome. The “belief” in the existence of this syndrome is worsened by the inability of electrodiagnostic tests to demonstrate the compression of the plexus, with the exception of the rare “true neurogenic” type in which the lower trunk of the plexus is involved.35,36

Routine chest x-ray films and cervical oblique x-ray films should be obtained to determine a pulmonary cause of the symptoms, such as a Pancoast tumor, or the presence of a cervical rib.

Symptoms can extend to facial pain and temporomandibular joint pain, presumably because of secondary effects on the cervical plexus.

Figure 16–12:
The anterior, or Henry’s approach for decompression of the distal portion of the radial nerve and the posterior interosseous nerve first identifies the radial nerve in the interval between the brachialis and the brachioradialis. ECRL, extensor carpi radialis longus.

Figure 16–13:
The posterior approach, or Thompson’s approach, uses the interval between the extensor digitorium communis (EDC) and extensor carpi radialis brevis (ECRB). ECRL, extensor carpi radialis longus; APL, abductor pollicis longus.
The transaxillary first rib resection is performed through 

- Headaches can be a common feature because of tightness in the scalene muscles.
- There usually is a history of trauma, such as whiplash or direct neck/shoulder blunt trauma. Diagnostic studies should include MRI of the cervical spine and the shoulder if these symptoms are present.
- An occupational component may contribute if work involves extensive overhead use of the hands or even work at shoulder level.
- Symptoms of hand swelling reflect compression of the subclavian vein, whereas symptoms of coldness may reflect either lower trunk compression with involvement of the sympathetic inflow or subclavian artery compression. Diagnostic studies should include imaging of these vessels for these symptoms.
- Congenital anomalies related to muscles and fibrous bands are common in the thoracic inlet (Table 16–6) and in the presence of trauma may limit excursion of the brachial plexus sufficiently to cause stretch/traction-type problems.
- Physical examination must demonstrate tenderness of the brachial plexus beneath the anterior scalene muscle and a positive provocative sign (of Roos), which is onset of symptoms and heaviness in the hands and arms within 1 minute of holding the hands above the head.37
- No surgery should be contemplated until the patient has undergone at least 6 months of special exercises designed to stretch the scalene muscles and strengthen other shoulder muscles.38
- Neurosensory testing can be performed with the hands at rest and after provoking the brachial plexus by elevating the hands.
- Thoracic surgeons began treating this problem in 1966 by transaxillary first rib resection. Gaining exposure of the origin of the first rib injures the intercostobrachial nerve in 66% of patients and is associated with risk for pneumothorax and injury to the subclavian artery and the C8 and T1 nerve roots.37
- The transaxillary first rib resection is performed through a curved incision. The surgery is performed with the patient in the lateral decubitus position. The curved skin incision is made over the third interspace. The supreme thoracic artery is ligated and the anterior scalene muscle is divided with scissors after being separated from the subclavian vein. The middle scalene muscle is released subperiosteally, in order to avoid injury to the long thoracic nerve. The periosteum is stripped from the first rib during periods when the lung is deflated in order to avoid injury to the pleura. An angled rib cutter is used to divide the anterior and posterior margins of the rib. It is important to check the pleura for leaks, and a chest tube is placed if necessary (16–14). Supraclavicular approach is performed with the patient in a beach chair position. The incision is made 1 cm above and parallel to the clavicle. For any surgery near the great vessels, the patient’s blood should be cross-typed and transfusion blood should be readily available in the operating room. The platysma muscle is divided and the external jugular vein is also ligated and divided. Using a superiosteal dissection, a third of the trapezius a sternocleidomastoid insertions are released from the clavicle. The omohyoid muscle, transverse cervical artery, and suprascapular artery run across the posterior triangle, and they are divided. The phrenic nerve runs along the anterior surface of the anterior scalene muscle, and it should be protected. Any cervical ribs that are present are removed if they impinge on the brachial plexus. The first thoracic rib is identified and the periosteum overlying it is incised. The anterior and middle scalene attachments are elevated subperiosteally. Kerrison rongeurs are used to divide the first rib at the anterior and posterior margins of the wound without injuring the pleura below. Anomalous bands between the scalene muscles and the first thoracic rib are often present and should be released. Once the rib is removed, the wound is filled with saline while the lungs are inflated to identify any pleural leaks that would require a chest tube. The wound is closed in layers.
- Supraclavicular approach offers the surgeon better exposure and therefore the opportunity to minimize operative complications. Transient phrenic palsy

Table 16–6: Anatomic Anomalies in the Thoracic Inlet

<table>
<thead>
<tr>
<th>Cervical rib</th>
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<tbody>
<tr>
<td>Fibrous bands from C7 transverse process</td>
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<tr>
<td>Extra origins for scalene muscles</td>
</tr>
<tr>
<td>Prefixed or postfixed brachial plexus</td>
</tr>
<tr>
<td>Intraperiosteal anomalous connections</td>
</tr>
<tr>
<td>Elevated position of subclavian artery</td>
</tr>
<tr>
<td>Muscle of Albinus (scalenus minimus)</td>
</tr>
<tr>
<td>Fibrous edges of scalene muscles</td>
</tr>
<tr>
<td>Anomalous vessels crossing plexus</td>
</tr>
<tr>
<td>Sibson fascia crossing T1 nerve root</td>
</tr>
<tr>
<td>Proximal junction of T1 to C8</td>
</tr>
</tbody>
</table>

Figure 16–14: The transaxillary first rib resection offers a cosmetically appealing approach, but the limited exposure and the potential injury to the intercostobrachial nerve are significant concerns.
reportedly is the most common complication, so avoiding traction on this nerve during plexus neurolysis and scalenectomy must be emphasized. Anterior scalenectomy with or without first rib resection is the preferred approach. The same success can be achieved leaving the first rib (Figure 16–15). Postoperatively the patient should be encouraged to turn the head and shrug the shoulders to encourage gliding of the plexus and prevent recurrence because of scar.

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The spectrum of radial tunnel and posterior interosseous nerve compression and treatment algorithms and surgical approaches are described.


True, neurogenic “thoracic outlet syndrome” is a rare compression of the lower trunk of the brachial plexus. It can be identified by an EMG positive for median and ulnar innervated intrinsic muscle denervation, decreased ulnar sensory amplitude, and normal amplitude for median sensory action potentials.


Emphasis on the inability of electrodiagnostic testing to identify brachial plexus compression in the thoracic inlet.


One of the earliest reports of the transaxillary approach. Discusses techniques and potential complications.


An exercise program that stretches the anterior scalenes and strengthens the upper trapezius, rhomboids, and serratus anterior is outlined and demonstrated to provide relief in up to 90% of patients with symptoms of “thoracic outlet syndrome.”


Following surgery for thoracic outlet syndrome, good or excellent responses were achieved in 86%.


Neurolysis of the brachial plexus for compression at the thoracic inlet, preserving the first rib, can give excellent results, with few complications.


Long-term outcome was similar for treatment of “thoracic outlet syndrome” by supraclavicular plexus neurolysis with preservation or resection of the first rib.