

## ORTHOPAEDIA: HAND

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THE CODMAN GROUP

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## PREFACE

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In World War I, it was generally known that traumatic hand injuries had poor outcomes since, in part, there was no systematic way of managing the injury. In World War II, the US Army Surgeon General, Major General Norman T. Kirk, knew of these poor outcomes and decided to ask Dr. Sterling Bunnell, who had experience and expertise in hand reconstruction surgery, to educate and train military surgeons on methods of hand surgery. Although originally a military decision to improve hand surgery outcomes, knowledge of hand injuries and conditions is still vital to limit functional disability.

Traditionally, musculoskeletal medicine has not received the attention that it requires in medical schools. In an age when there is a growing number of elderly patients requiring musculoskeletal care, more emphasis is needed in educating medical students about these conditions. Although hand surgery may not receive much attention in the medical school curriculum, it is just as important as any debilitating spine, hip, and knee condition. The hand is perhaps most susceptible to traumatic injuries since we use it ceaselessly. Intricate, delicate, and strong, but prone to severely disabling injuries and conditions, hand injuries can significantly lower the patient's quality of life. Therefore, it is important that medical students and doctors understand the basics of hand pathophysiology.

Our roles as surgeons, researchers, and educators, are to continue the task started by Dr. Bunnell. This book attempts to explain hand injuries and conditions to the younger generation in a way that is both engaging and educational. It is the result of a collaboration of some of today's leading hand surgeons in the US. Effort was made to emphasize key concepts of a condition. The diseases described are those that are most commonly encountered in medical practice. It is our hope that students use this book as a learning tool and also as a reference during their studies at medical school and residency, and throughout their medical careers, regardless of specialty.

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## A WORD ON PEER REVIEW

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There is a great profusion of medical information available for free on the Internet, and a lot of it is good. Yet even good information may not be completely useful to the reader who may not know if it is trustworthy. By contrast, there is also a lot of information of medical information available for sale that is produced by well known authors and organizations, though not always for free.

***Orthopaedia*** aims to be both free and authoritative.

To ensure medical accuracy, all of the material was reviewed by the section editors, who are of course content experts of great renown. In addition, each chapter was reviewed by an expert who was not involved in the creation of the material. These reviewers were asked to read the chapter with one overriding goal in mind: to detect errors. The reviewers were then asked to “certify” the chapter as a reasonable presentation of the topic without any glaring mistakes in content. We are grateful to our reviewers, listed below:

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## CHAPTER 1.

### ARTHRITIS OF THE HAND

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Arthritis of the hand is a common and often disabling condition. There are several distinct types of arthritis affecting the hand; of these, degenerative arthritis (osteoarthritis) is the most common. Also seen are inflammatory arthritis (including rheumatoid arthritis), crystalline arthritis (gout, pseudogout) and infectious arthritis. The features common to all forms of hand arthritis are pain, stiffness, and swelling of the joints, and, in turn, impairment of function.

#### STRUCTURE AND FUNCTION

##### *Osteoarthritis (OA)*

The cause of hand osteoarthritis, while multifactorial, is often attributed to simple wear and tear caused by repetitive use and aging. In point of fact, however, abnormalities of the cartilage surfaces, kinematics and soft tissue forces, as well as inflammation and hormonal influences can all cause or promote the disease. For example, ligamentous laxity may lead to abnormal joint loading. In turn, this causes accelerated wear of the cartilage surface. (The process is similar to what may be seen with an automobile tire that is not secured tightly to the vehicle: it will wobble and erode prematurely.)

The classic radiographic hallmarks of osteoarthritis (subchondral sclerosis, joint space narrowing, synovial cysts, and osteophytes) are usually apparent in arthritis of the hand, however, the extent of these radiographic signs does not always correlate well with patients' symptoms.

The characteristic features of a joint afflicted with osteoarthritis are hyaline cartilage degeneration; sclerosis of the subchondral bone; formation of osteophytes at the periphery of the joint; and cyst formation within the bone. In the hand, so-called mucinous cysts can form outside of the joint as well.

Although osteoarthritis is considered a disease of wear and tear, clearly genetic factors are involved. Twin studies have demonstrated a 59% concordance for hand osteoarthritis, and familial predispositions to osteoarthritis have long been recognized.

Interestingly, elevated body mass index is a risk factor for osteoarthritis of the hand. Given that the hand is not (typically) a weight-bearing structure, this risk suggests a possible systemic effect of an elevated BMI.

##### *Rheumatoid Arthritis (RA)*

The mechanism of joint destruction in rheumatoid arthritis is incompletely understood. The etiology appears to comprise both genetic and environmental components. Rheumatoid arthritis may generally (and incompletely) be described as an immune attack originating in the synovial cells against adjacent cartilage, tendon, bone, and soft tissues.

Rheumatoid arthritis begins in the synovial membrane with activation of the innate immune system, leading to loading of antigen presenting cells (APCs) with auto-antigens. APCs then migrate out of the joint and present these antigens to T-cells, which can then activate B cells or act directly on the synovium. There is likely no specific "rheumatoid arthritis antigen," and the immune response seems to be directed at multiple targets, which vary across individuals. The wide variety of targets may explain the wide variety of treatment responses.

## PATIENT PRESENTATION

The hallmark of hand arthritis, regardless of cause, is pain, stiffness, and swelling. To be sure, hand arthritis is not the only explanation for this presentation, and the examiner must be mindful of gout/pseudogout and infection (discussed below).

Osteoarthritis of the hand is typically seen in patients older than 50 years of age; the onset is often insidious. The classical symptoms of osteoarthritis are pain, tenderness, and stiffness worsened by activity and relieved by rest. Osteoarthritis most commonly involves the distal interphalangeal joints (DIP). The thumb carpometacarpal joint, (CMC) and proximal interphalangeal joints (PIP) are the next most commonly involved joints, but OA can be present in any joint of the hand.

Although osteoarthritis may occur in one joint in isolation, it is often symmetric and involves a “row” of joints.

Osteoarthritis of the hand often produces deformity in its more advanced stages, though these are less dramatic than the deformities induced by rheumatoid arthritis. The most commonly seen osteoarthritis-related deformity is that produced by prominent exostoses (bone spurs) in the fingers. When these are present at the distal interphalangeal joint they are termed Heberden’s nodes; Bouchard’s nodes are similar exostoses seen at the proximal interphalangeal joint (as seen in Figure 1).



Figure 1: Clinical photo of hand osteoarthritis. (Credit: wikipedia.  
<http://bit.ly/1QAtJcp>)

Hypertrophy of the soft tissues (coupled with osteophyte formation) can give a bulbous appearance to joints affected by osteoarthritis.

Basal thumb (CMC) arthritis may present with decreased pinch strength, and in advanced stages an adducted CMC joint with a compensatory MCP joint hyperextension deformity.

Additionally, mucous (mucinous) cysts, similar to ganglion cysts, may form in severe osteoarthritis, occasionally rupturing at the skin surface.

Rheumatoid arthritis in the hand most commonly involves the MCP and PIP joints. An insidious onset of pain, swelling and stiffness of multiple joints of the hand that is not related to activity are the distinctive features of the disease. Morning stiffness, particularly such stiffness lasting greater than one hour, is classic for rheumatoid arthritis. Affected joints will have an effusion (fluid within the joint capsule) and soft tissue swelling. Some additional features characteristic of this disease are inflammatory nodules, tenosynovitis, and tendon rupture.

Chronic rheumatoid arthritis classically results in ulnar deviation of the MCP joints in addition to swan neck and boutonniere deformities of the interphalangeal joints (due to weakening of the ligaments and capsule, as well as intrinsic muscle imbalance).

Tendon ruptures, particularly of the extensor tendon to the thumb (EPL) or to the small finger (EDQ) are also common.

Clinically, three phases of rheumatoid arthritis are typically observed. The initial proliferative phase includes the onset of pain and effusion corresponding to synovial proliferation (described below). The second is the destructive phase in which the synovial pannus invades cartilage and soft tissue. Finally, there is the late or “burnt out” phase in which inflammation is replaced by fibrosis, scarring, and fixed deformities (Figure 2).



Figure 2: Clinical photo of advanced rheumatoid arthritis (Credit: Wikipedia. <http://bit.ly/23BOoE6>)

## OBJECTIVE EVIDENCE

The radiographic findings of osteoarthritis include joint space narrowing, subchondral sclerosis and cysts, osteophyte formation as seen in Figure 3. Advanced disease may demonstrate deformity including subluxation and adjacent joint involvement.



Figure 3: X-ray of osteoarthritis of both the thumb CMC joint as well as the wrist. (Credit: Wikipedia. <http://bit.ly/20AqV3E>)

There are no laboratory tests to confirm osteoarthritis; inflammatory markers are expected to be normal. Also, the synovial fluid should have a leukocyte count less than 2000 WBC/uL and without any crystals.

Any abnormal laboratory test results in the presence of presumed osteoarthritis should prompt the exploration for other diagnoses.

On plain x-ray, early rheumatoid arthritis will demonstrate soft tissue swelling. This will progress to joint space narrowing, subchondral cysts, peri-articular erosions and diffuse osteopenia. Erosions occur first in the “bare areas” of bone that are intra-articular but not covered in articular cartilage. In its advanced stages, the principle findings are gross deformity and auto-fusion of the joints.





Figure 4: X-ray of rheumatoid arthritis (Credit: Radiopaedia.org  
<http://bit.ly/1Su0SbN>)

MRI may additionally demonstrate effusion, synovitis, tenosynovitis, or tendon rupture.

Laboratory values in rheumatoid arthritis typically demonstrate a positive Rh factor and anti-cyclic citrullinated peptide (CCP) antibodies, though these can be negative in 20% of cases. The erythrocyte sedimentation rate (ESR, also known simply as the “sed rate”) and C-reactive protein (CRP) levels are typically elevated, though such elevation is a non-specific finding. Fluid analysis will typically demonstrate 3,500-50,000 WBC/uL.

## EPIDEMIOLOGY

Primary osteoarthritis of the hand is common, with radiographic evidence of OA found in approximately 75% of females and 65% of males older than 55 years of age. Not all of these people have symptoms, however, symptomatic OA of the hand has a prevalence of about 25% among women and 13% among men (about 33% and 20%, respectively, of those with radiographic changes) Indeed, as you see, it is common for radiographic evidence of OA to not be symptomatic.

Radiographic OA is most commonly seen in the DIP joints (more than 50% of cases). The thumb CMC joint is the next most common (20%), followed by the PIP (8%).

RA is the most common inflammatory arthritis affecting 0.5-1% of the population worldwide. 70% of patients with RA report hand and wrist dysfunction. Women are 2-3 times more commonly affected. The age of onset is typically about 60.

## DIFFERENTIAL DIAGNOSIS

Hand pain and swelling can be caused by OA, RA, other inflammatory conditions (such as psoriatic arthritis and gout/pseudogout), infection and, rarely, a neoplastic process.

The location of the pain and its time course, the degree of inflammation, the number and symmetry of joints involved, the presence of systemic disease, and the associated signs of infection or trauma can aid in determining the diagnosis.

Carpal tunnel syndrome is a common cause of hand pain, but it is (by definition) limited to the median nerve distribution (the *palmar side* of the thumb, index and long fingers, primarily). In addition to pain, carpal tunnel syndrome can be detected by the presence of sensory changes and muscle atrophy (neither features of arthritis). Note that carpal tunnel syndrome can co-exist with arthritis and other diagnoses.

Pain at the base of the thumb may be from arthritis or tendinitis of the extensor pollicis brevis and abductor pollicis longus (de Quervain's disease). Finkelstein's test may help differentiate the two. In this test, the thumb is flexed and grasped by the patient's four other fingers. Then, with the thumb stabilized, the examiner ulnar-deviates the hand, thereby stretching the extensor pollicis brevis and abductor pollicis longus tendons attached to the thumb. A positive test is characterized by reproduction of pain at the distal radius.

Hand infection may be a cause of acute swelling and pain. (The presence of a puncture wound, insect bite, or abrasion, of course increases the likelihood of this diagnosis.)

Gout and pseudogout are also causes of swelling and pain. In brief, gout and pseudogout are so-called "deposition diseases" in which crystals (monosodium urate and calcium pyrophosphate, respectively) are deposited in the joint and irritate it. Gout tends to affect the fingers, whereas pseudogout more commonly involves the wrist. The deposition of crystals can make the joint red, hot, tender and swollen – mimicking infection. A medical history, lab tests and x-rays can help differentiate these conditions from arthritis. The best means to make the diagnosis definitely is to examine fluid from the joint for uric acid or calcium pyrophosphate crystals.

*Table 1: Differentiating features of hand arthritis*

Feature	Osteoarthritis	Rheumatoid Arthritis
<b>Patient demographics</b>	Older; genders roughly equally affected	Younger; female
<b>Time of maximal discomfort</b>	End of day, pain with use	Morning stiffness, pain upon awakening
<b>Joint(s) involved</b>	interphalangeal joints; base of thumb	MCP joints
<b>Tendon involvement</b>	No	Yes
<b>Visible deformity</b>	Nodules at IP joint	Ulnar deviation of fingers; swan neck and boutonniere deformities of the interphalangeal joints
<b>Radiographic features</b>	osteophyte formation, sclerosis common Asymmetric joint space narrowing	osteopenia; symmetric joint space narrowing

## RED FLAGS

Patients who present with acute swelling, warmth and erythema must be examined closely to rule out infection.

Patients with RA must be monitored for incipient tendon or ligament failure. Tendon rupture is an absolute indication for surgery, not only to restore lost tendon function, but to eliminate bone deformity or synovitis. This latter step will help prevent subsequent ruptures of neighboring tendons.

For patients with RA, systemic manifestations of disease must be considered as well. Children with juvenile arthritis must be monitored for uveitis (inflammation of the eye, including the iris, ciliary body and choroid).

## TREATMENT OPTIONS AND OUTCOMES

### *Non-operative Treatment*

The mainstay of treatment for mild to moderate OA of the hand involves non-surgical measures aimed at reducing symptoms. These include hand therapy, NSAIDs, activity modifications, and local corticosteroid injections. Hand therapy includes splinting, strengthening exercises, and modalities such as hot soaks, and paraffin baths.

Several of the non-surgical strategies used in OA of the hand are employed in the treatment of RA of the hand as well. In addition, proper medical management of the disease, particularly using Disease Modifying Anti-Rheumatic Drugs (DMARDs), has significantly reduced the disease burden in patients with RA and reduced the need for surgical intervention in these patients.

The results of hand therapy in treating mild OA are good, with the majority of patients experiencing improvements in both pain and function. Those individuals requiring surgical intervention typically report high rates of improvement in pain and reduced deformity.

### *Operative Treatment*

In more advanced disease that has not responded to initial treatment measures, surgery may help. Surgical options generally include tendon releases and transfers, cheilectomy (removal of excess bone), arthrodesis (joint fusion) and arthroplasty (joint replacement). While the specific details of the surgical management of hand arthritis is beyond the scope of this chapter, some common approaches and procedures warrant specific mention.

#### *Synovectomy*

Synovectomy is routinely performed in the surgical management of RA. Synovectomy alone, however, is rarely indicated (as there is 30-50% recurrence rate of synovitis after this procedure alone), but it is often applied in combination with tendon transfers and arthroplasty.

#### *Arthrodesis*

Surgical fusion of the joint (arthrodesis) is generally a very effective procedure in relieving pain. The resulting joint fusion provides a stable and pain free appendage at the expense of motion. Fusion is tolerated quite well in joints such as the DIPs and is particularly useful in joints that present with instability. Arthrodesis of an unstable index finger PIP joint can allow for significantly improved pinch strength and function. Arthrodesis of the MCP joint is poorly tolerated and is generally a last resort.

Arthrodesis of interphalangeal joints is extremely effective in improving pain and function.

## *Reconstructive Surgery*

The thumb CMC joint is the most frequent site of reconstructive surgery in the upper extremity. Common approaches include volar ligament reconstruction, trapeziectomy with or without interpositional tendon graft, or arthroplasty.

The most common reconstructive procedure performed for RA of the hand is MCP joint replacement (arthroplasty), typically with a silicone implant. With the use of DMARDs, however, the need for this procedure has decreased dramatically in recent years.

The gold standard for treatment of advanced thumb CMC arthritis is trapeziectomy with or without interpositional graft and or ligament reconstruction. This method reliably relieves pain and improves function. Synthetic implants have a relatively high complication rate compared to excisional arthroplasty.

## *Arthroplasty*

MCP arthroplasty in RA has demonstrated improved short-term functional and cosmetic outcomes in addition to reliable pain relief. Long-term studies have shown a high failure rate of the silicone implant, however, in such cases the implant can often be retained and retain adequate function. Recurrent ulnar drift is common, but usually less severe than the original deformity.

## **RISK FACTORS AND PREVENTION**

Risk factors for OA include advanced age, female gender, obesity, repetitive use, and family history. Prevention is based on avoiding risk factors such as repetitive activities. Often, such activities are associated with the patient's work, and therefore, difficult to avoid.

Risk factors for RA include gender (female > male, as noted), family history, and smoking. Some studies have shown oral contraceptive use to be protective, as well as breastfeeding and childbirth. There is currently no preventive strategy, but early diagnosis and treatment can prevent many of the complications of the disease.

## **MISCELLANY**

The use of chondroitin sulfate and glucosamine may provide benefit to some patients with osteoarthritis and carries a low risk profile if purchased from a reputable source. Studies have failed, however, to establish a proven benefit to these supplements.

## **KEY TERMS**

Osteoarthritis, rheumatoid arthritis, carpometacarpal (CMC) arthritis, Heberden's nodes; Bouchard's nodes

## **SKILLS**

Recognize and describe deformities of the hands caused by arthritis. Recognize the signs and symptoms of infection. Examine the hand to determine if there is full and painless range of motion, normal strength and intact motor and sensory function.

## CHAPTER 2.

### CARPAL FRACTURES

---

There are eight carpal bones at the wrist, situated between the radius and ulna in the forearm and the metacarpals in the hand. The most common (and important) carpal fracture is that of the scaphoid (discussed in its own Chapter). Among the other carpal bones, only the triquetrum, hamate and pisiform are likely to be fractured in isolation; other carpal fractures are seen more commonly in conjunction with other injuries. Most isolated carpal fractures are caused by direct trauma.

### STRUCTURE AND FUNCTION

There are eight carpal bones, each with its own unique shape and size. The carpal bones articulate with the radius and ulna proximally and the 5 metacarpal joints distally.

There are eight carpal bones arranged in two rows: the proximal row consists of the scaphoid, lunate, triquetrum and pisiform. The distal row consists of the trapezium, trapezoid, capitate and hamate. The proximal row articulates with the distal radius and distal row connects to the metacarpal bones of the hand.

#### *Proximal Row*

The scaphoid (Figure 1) is located on the radial side of the proximal carpal row.



Figure 1: Scaphoid

The scaphoid articulates with 4 other carpal bones and is involved with most carpal motions, especially flexion. The vascular supply of this bone travels from the distal region of the scaphoid back proximally (a so-called “retrograde flow”). Ossification in children also occurs in the distal-to-proximal direction.

The lunate (Figure 2) is half-moon shaped and is positioned between the scaphoid and the triquetrum.



*Figure 2: Lunate*

The lunate is important in flexion/extension and radial/ulnar deviation at the radiocarpal and midcarpal joints.

The triquetrum (Figure 3) is located on the ulnar side of the proximal carpal row. It is pyramid-shaped with an oval shaped facet on its volar side.



*Figure 3: Triquetrum*

The triquetrum articulates with the pisiform on the volar side, the lunate laterally and the triangular fibrocartilage complex proximally.

The pisiform (Figure 4) is a pea shaped sesamoid bone that articulates with the triquetrum.



*Figure 4: Pisiform*

The pisiform is enclosed in the sheath of the flexor carpi ulnaris tendon and is in close proximity to the ulnar nerve.

### ***Distal row***

The hamate (Figure 5) is a wedge-shaped bone on the ulnar side of the distal carpal row.



*Figure 5: Hamate*

On the distal volar surface, a hook protrudes out from the ulnar side of the bone. The hamate serves as an attachment site for many ligaments, including the transverse carpal ligament atop the carpal tunnel. The hook of the hamate acts as a pulley for the 4th and 5th finger flexor tendons.

The capitate (Figure 6) is the largest carpal bone and is positioned in the central column of the wrist.



*Figure 6: Capitate*

The capitate articulates with the scaphoid, lunate, hamate and trapezoid, as well as the 2nd, 3rd and 4th metacarpals.

The trapezium (Figure 7) is a quadrangular shaped bone located on the radial side of the distal carpal row.



*Figure 7: Trapezium*

The trapezium has a double-saddle facet that articulates with the 1st metacarpal allowing both flexion/extension, abduction/adduction, circumduction and opposition at the joint.

The trapezoid (Figure 8) is positioned between the capitate and trapezium.



Figure 8: Trapezoid

## PATIENT PRESENTATION

Patients with acute carpal fractures will typically present with wrist pain and swelling and there will be point tenderness near the fracture. Due to the close proximity of some carpal bones to nearby nerves, some carpal fractures will be associated with neurological injury as well.

Triquetral fractures are usually a result of a direct blow to the dorsum of the hand or a fall, forcing the wrist into extreme extension and ulnar deviation. This position can cause the proximal hamate and distal radius to impinge on the triquetrum and shear it. Patients typically present with pain and swelling in the wrist with increased tenderness on the ulnar side. The triquetrum lies deep to the pisiform bone, making it difficult to palpate on exam. To expose the bone on exam, the hand must be placed into a position of radial deviation.

Hamate fractures are associated with racket sports or sports involving clubs. The injury is often to the hook of the hamate, sparing the body. Patients will usually have history of a trauma, for example, the accidental strike of the ground with a golf swing. They will have ulnar sided wrist pain, pain in the hypothenar region and decreased grip strength. Because this injury is associated with compression of the ulnar nerve as it crosses Guyon's canal in the wrist, patients may have paresthesias in their ring and small fingers, with weakness of the intrinsic muscles supplied by the ulnar nerve. Patients may also have pain with resisted flexion of the 4th and 5th finger. Hamate body fractures are typically seen in injuries where an axial force was applied to a closed fist. Patients will similarly complain of ulnar sided wrist pain and present with swelling.

Pisiform fractures are usually caused by a fall on the outstretched hand. The chief complaint is ulnar sided wrist pain and the physical exam is notable for point tenderness over the pisiform.

Trapezoid and trapezium fractures are usually not caused by direct trauma, but by a so-called "nutcracker effect," namely, pressure from the second metacarpal when the finger and wrist are hyper-extended. An index finger injury with tenderness more proximally, towards the wrist, should increase the suspicion of this (rare) fracture.

"Fractures" of the lunate seen on x-ray, especially without a history of specific injury, are more likely to represent fragmentation of the bone from osteonecrosis (Kienböck's disease).

Capitate fractures in isolation are rare. A ligament injury should be suspected and ruled out when this diagnosis is made (or considered).

Note that many carpal fractures may present as sub-acute or chronic diffuse pain, due to missed diagnosis of a prior injury. In those settings, the presentation is similar to carpometacarpal or radiocarpal arthritis or tendinitis.



## OBJECTIVE EVIDENCE

The standard wrist radiographs ordered for assessment of the carpal bones include AP, lateral and oblique views. Disruption or widening of the spaces between the bones may indicate carpal instability or damage to the ligaments.

CT scan is the preferred study for diagnosis of hook of hamate fractures (as shown in Figure 9).

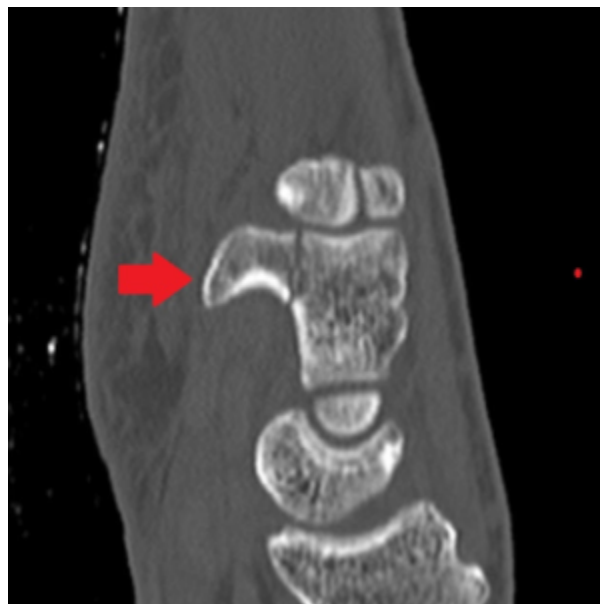


Figure 9: Hamate fracture as seen on CT (credit Radiopaedia.org  
<http://bit.ly/23BP8sM>)

MRI can be a useful modality in not only detecting occult fractures, but also detecting soft tissue injuries such as ligaments or tendon damage.

Interpretation of carpal radiographs is admittedly difficult for the novice, and there is no shame in asking an expert for help. Two things must be kept in mind: 1) dislocations require urgent attention and 2) the wrist should be immobilized until a definitive diagnosis is obtained.

## EPIDEMIOLOGY

Carpal fractures make up nearly one-quarter of hand fractures and about 5% of all fractures. This estimate likely under-represents the true incidence of carpal fractures, as many are presumably not formally diagnosed (and dismissed by the unsuspecting patient as a contusion). As noted, scaphoid fractures account for a majority of carpal bone fractures. The second most common carpal fracture is of the triquetrum, which accounts for 14% of all wrist injuries.

## DIFFERENTIAL DIAGNOSIS

All wrist pain due to falls on an outstretched hand should be carefully assessed for the possibility of a carpal fracture. In particular, scaphoid fracture should be kept in mind, due to the high risk of developing a non-union and, ultimately, osteonecrosis and post traumatic arthritis if left untreated.

Injuries to the radioulnar joint should also be ruled out when considering carpal fractures.

Carpal dislocations can occur commonly in conjunction with carpal fractures.

Fractures of some carpal bones usually occur in conjunction with other carpal fractures, making it important to rule out fractures to adjacent carpal bones.

## **RED FLAGS**

Marked tenderness over the scaphoid tubercle or the anatomic snuffbox in the setting of normal x-rays suggests a non-displaced scaphoid fracture (which must be immobilized).

Finger injuries with wrist tenderness suggest an injury to the carpal bones.

## **TREATMENT OPTIONS AND OUTCOMES**

The treatment of carpal fractures aims primarily to restore pain-free motion of the wrist. An important secondary aim is to reduce the patient's risk of developing osteoarthritis of the carpal bones.

Most stable, non-displaced fractures of carpal bones are treated with casting for 6-8 weeks. Non-displaced scaphoid fractures should be immobilized in a thumb spica cast. In cases of displacement or instability, open reduction with internal fixation is considered.

Most isolated carpal fractures (except the scaphoid, which has a more tenuous blood supply) heal uneventfully. Because there may be an associated ligament injury with subtle instability, osteoarthritis may develop even if the fractures do heal nicely.

## **RISK FACTORS AND PREVENTION**

The use of wrist guards when rollerblading (or when participating in any other activity with a high rate of falls on the outstretched hand) may be helpful.

## **MISCELLANY**

The mnemonic for recalling the names of the carpal bones in medial-to-lateral order, "Some Lovers Try Positions That They Can't Handle," is somewhat self-referential as weight-bearing on the palm is painful for people with injured or arthritic carpal bones. (ed: The vivacity of the mental image is worth the risk of offending the Bluenose readers).

## **KEY TERMS**

anatomic snuffbox; carpal fracture; osteonecrosis; non-union; Scaphoid; Lunate; Triquetrum; Pisiform; Trapezium; Trapezoid; Capitate; Hamate

## **SKILLS**

Recognize and name a carpal fracture; perform a physical exam to detect and define carpal injury.

## CARPAL INSTABILITY

---

The eight carpal bones of the wrist (“carpus”) are held together by ligaments. When there is an injury to a ligament (ranging from an isolated sprain to a catastrophic dislocation) the wrist may be rendered unstable.

Carpal ligament injuries can lead to post-traumatic arthritis because the bones of the carpus are normally “intercalated,” that is, tightly tethered to each other. When a carpal ligament is injured, the bones may not move in their normal concerted fashion. This abnormal motion leads to abnormal positioning of the bones and, in turn, overloading of the joint surfaces.

### STRUCTURE AND FUNCTION

There are eight carpal bones arranged in two rows: the proximal row consists of scaphoid, lunate, triquetrum and pisiform (Figure 1). The distal row consists of trapezium, trapezoid, capitate and hamate. (Please see the chapter, Carpal Fractures, for a more detailed description.) The proximal row articulates with the distal radius and distal row connects to the metacarpal bones of the hand.



*Figure 1: The carpal bones. The scaphoid is shown in blue; the lunate in orange; trapezium in pink; trapezoid in white; the capitate is green; the hamate is purple; the triquetrum is yellow and the pisiform is red.*

The bones have complex geometry to allow them to remain in contact when the hand is moved in three dimensions. Yet this complex geometry offers little bony congruity and in turn less inherent stability of the joints (contrasted to what is seen, for example, in the hip). As such, a network of ligaments is needed to secure the bone-to-bone connections.

The ligaments of the carpus are categorized as “intrinsic” (for those connecting carpal bone to carpal bone) or “extrinsic” (if they connect either the radius or the ulna to an individual carpal bone). The nomenclature is simple: a compound word naming the two connecting bones is used, e.g., “scapho-lunate” ligament.

When a carpal ligament is injured, the bones will tend to separate and the normal contact points are disrupted. This can lead to focal load bearing and articular cartilage breakdown. In turn, arthritis may develop, leading to painful and limited motion and radiographic changes.

The carpal bones are said to be “intercalated.” That term technically means “layered,” but the key concept is that the bones are linked to each other via ligaments, and as such, when a ligament is injured the bones do not move in concert, as they should. Two patterns of disruption are common enough to warrant their own name:

- “dorsal intercalated segmental instability” (DISI), in which the scapholunate ligament is injured, and
- “volar intercalated segment instability” (VISI) caused by rupture of the lunotriquetral ligament and dorsal radiocarpal ligament.

(The names refer to the abnormal position assumed by the carpal bones because they are not tethered appropriately.)

A third pattern of ligamentous injury, perilunate dislocation, is one of the most severe forms of carpal instability. This is seen when multiple intrinsic and extrinsic ligaments are torn, and the lunate completely dissociates from rest of the carpal bones. Because of the high energy associated with this injury, vascularity and neurologic function may be compromised as well.

## PATIENT PRESENTATION

The most common injury mechanism is from a fall on an outstretched hand with wrist extended. Violent trauma such as a motorcycle accident or a contact sports injury is also a frequent cause of carpal instability. Accordingly, the chief complaint of patients with a carpal ligament injury is pain, and their primary findings are swelling and tenderness.

In severe cases of carpal instability, the lunate can be completely displaced (Figure 2) and therefore might be palpable in the palm.

Often, the patient’s pain and tenderness will be too great to allow stress testing of the affected structures and therefore radiographic diagnosis is key.

In many cases, carpal ligament injuries are associated with fractures of the adjacent bones. For instance, distal radius fractures are commonly seen with scapholunate ligament injury. Therefore, it is important to survey the entire overall spatial relationship of the carpal bones, in addition to assessing the more obvious fracture(s).

Unfortunately, there are no pathognomonic clinical features that are diagnostic of carpal instability. The symptoms are often subtle and unimpressive. Generalized pain especially with wrist motion and axial compression without any obvious deformity or radiographic findings can steer the clinicians to suspect carpal instability.

Astute examiners can localize tender spots over the scapholunate and lunotriquetral ligaments based on anatomic landmarks.

Provocative maneuvers such as the Watson test and scaphoid ballottement test are helpful. The Watson test (also known as the scaphoid shift test) is perhaps the most important exam maneuver when there is suspicion for carpal instability. The Watson test is used to identify scapholunate ligament tears.

In order to perform this test, the clinician's thumb presses on the scaphoid tuberosity. As the examiner passively deviates the wrist from ulnar to radial direction, pain and clunking signifies a positive test. This "clunk" results from subluxation of the proximal scaphoid over the dorsal lip of the radius. Although the Watson test is sensitive for the detection of scapholunate ligament tears, its specificity is low.



Figure 2: In this lateral view of a CT of the wrist, a volar dislocation of the lunate is demonstrated (credit: Radiopaedia. <http://bit.ly/1nwVadv>)

## OBJECTIVE EVIDENCE

After a thorough history and physical examination, posteroanterior, oblique and lateral radiographs of the wrist (the so-called "three-views") should be obtained.

Full thickness tears or chronic injuries to the scapholunate and lunotriquetral ligaments are often detected due to widened intervals of the proximal carpal bones.

Carpal instability can be demonstrated on imaging studies, either by detecting the carpal bones out of their normal place (Figures 3 and 4), suggesting laxity, or by demonstrating arthritic damage to the articular surfaces.

When assessing for carpal instability, the bone worthy of particular attention on imaging studies is the lunate. The lunate is tethered to scaphoid and triquetrum.

The key point to recall is that if the carpal bones were not connected, the scaphoid would flex (move in a volar direction) and the triquetrum would extend (move in a dorsal direction). The lunate, connected to both the scaphoid and the triquetrum, stabilizes these bones accordingly. Critically, the position of the lunate can therefore indicate the status of the ligaments:

- when scapholunate ligament is injured, the uninjured lunotriquetral ligament drags the lunate into extension; and
- when the lunotriquetral ligament is injured, the intact scapholunate ligament pulls the lunate into flexion.

With a complete tear of the scapholunate ligament (Figure 5), abnormal extension of the lunate is visible on the lateral view of the wrist. This, as noted above, is known as *dorsal intercalated segment instability* or DISI.

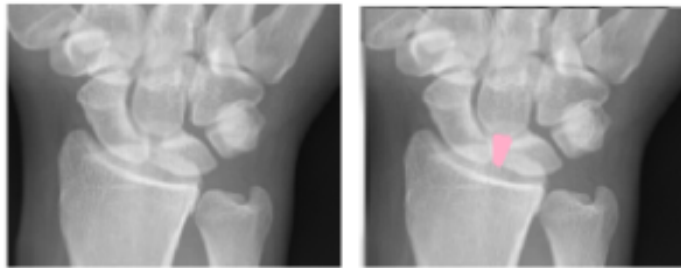
A complete tear of the lunotriquetral ligament will allow the scaphoid to drag the lunate into flexion, indicating *volar intercalated segment instability* (VISI)

Partial tears of the scapholunate and lunotriquetral ligaments are more challenging to identify on static radiographs, which tend to appear normal.

Stress views (for example the “clenched fist” view) can reveal carpal instability.

In cases where multiple views including stress radiographs do not show any abnormality, MRI may prove to be helpful. However, the sensitivity of MRI for scapholunate ligament tear is only approximately 60-70%.

The current gold standard for diagnosing carpal instability is wrist arthroscopy. The direct visualization of the carpal ligaments with the use of a camera and blunt probing instruments, can definitively diagnose or rule out subtle or radiographically negative ligament tears.



*Figure 3: Normal scapholunate interval (modified from wikipedia).*



*Figure 4: The scapholunate interval is wide (modified from wikiradiography).*

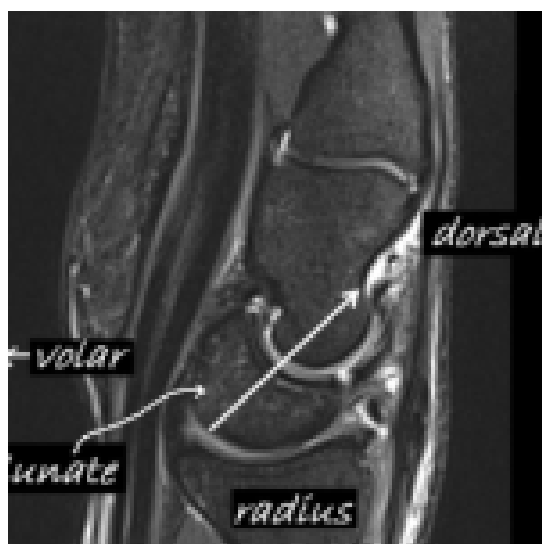


Figure 5: Lateral view of the wrist on MRI, showing the lunate extended, i.e. tilted towards the dorsal surface. (Some have suggested that this looks like a tea cup that has been tipped over.) This "tilted tea cup" is indicative of complete disruption of the scapholunate ligament.

## EPIDEMIOLOGY

Carpal instability is an injury of young and middle-aged populations. The exact incidence of carpal instability is unknown, as the diagnosis is not often apparent.

Instability can be subtle: severe symptoms can be found in the setting of fairly benign-appearing radiographs. It has been shown that carpal instability can be detected (if sought) in approximately 30% of patients with displaced intra-articular distal radius or scaphoid fractures.

## DIFFERENTIAL DIAGNOSIS

Major carpal instabilities and frank dislocations are readily visualized on radiographs. Clinical findings of partial tears or attenuation of scapholunate and lunotriquetral ligaments are subtle and symptoms are nonspecific. More common clinical entities should be entertained prior to diagnosing carpal instability.

Conditions that may present with complaints similar to those of mild carpal instability include:

- DeQuervain's tenosynovitis
- Extensor tendonitis
- Carpal tunnel syndrome
- Distal radioulnar joint instability
- Triangular fibrocartilage complex injury
- Wartenberg syndrome (radial sensory neuritis)
- Intersection syndrome (inflammation at the point where the abductor pollicis longus and extensor pollicis brevis "intersect" the extensor carpi radialis longus and the extensor carpi radialis brevis in the forearm)
- Nondisplaced scaphoid fractures

## RED FLAGS

- A history of high energy trauma. Due to complex geometry of the bones and their overlap on lateral views, the radiographs of carpal bones are very difficult to evaluate. As such the best means to detecting ligament injuries and carpal instability is to maintain vigilance, especially with higher-energy trauma. Loss of the normal symmetrical spaces normal seen radiographs can be a clue that ligament injury is present.
- Presence of a displaced, intra-articular distal radius fracture. Scapholunate ligament tears are commonly associated with distal radius fractures. Persistent wrist pain despite fracture fixation suggests a diagnosis of a ligament injury.
- Sudden onset of neurologic symptoms such as numbness, tingling, and radiating pain along the thumb, index and middle finger distribution suggest median nerve compression (an acute carpal tunnel syndrome) and may require urgent decompression.

## TREATMENT OPTIONS AND OUTCOMES

Partial tears of scapholunate and lunotriquetral ligaments without positive radiographic findings are initially treated nonoperatively with a short course of immobilization, followed by therapy. In persistently symptomatic cases, wrist arthroscopy is the next phase of treatment. Partial tears can be confirmed, and the ligament remnant may be debrided. Arthroscopically-assisted and percutaneous pinning of the injured interval can provide further stabilization to promote healing.

Complete tears of scapholunate and lunotriquetral ligaments (and with that, loss of ligament integrity) allow the carpal intervals to widen. The goal of treatment for this condition is to restore and maintain carpal alignment. Many operative techniques have been described; no single technique has proved superior to the others. An open approach (contrasted to arthroscopy) allows for better visualization of the wrist bones at the cost of more surgical trauma.

Regardless of the operative approach, screws and pins are ultimately used to hold the proximal carpal in an anatomic position as seen in Figure 6.

Primary repair is feasible in certain cases, but more frequently wrist capsule and other ligaments are “borrowed” to augment the injured interval.

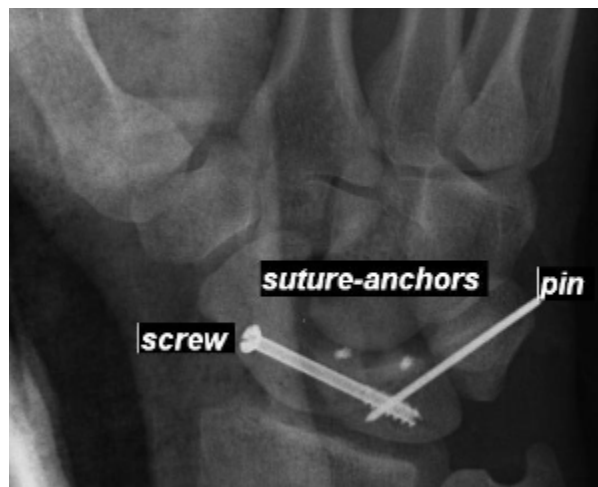


Figure 6: Fixation can be attained with screws, pins or suture anchors- or all three!

In cases of perilunate dislocation, closed reduction with sedation can be attempted in the emergency department, but it is often unsuccessful as a long-term solution. These injuries are treated surgically with nerve decompression (i.e. carpal tunnel release) and open reduction of the lunate.



High level medical evidence (ie prospective trials) regarding treatment outcome does not exist, and case reports may be misleading because of bias. Still, in general it can be observed that an injury severe enough to be easily detected is severe enough to lead to a less than desirable outcome, regardless of how it was treated. Similarly, mild ligament injuries treated with the immobilization often do well though some loss of motion may be expected. In severe or chronic cases, post-traumatic arthritis is expected.

## **RISK FACTORS AND PREVENTION**

Any activity that increases the risk of falls or high energy trauma theoretically increases the risk of carpal instability. In general, there are no specific preventative steps to avoid this condition.

## **MISCELLANY**

One of the most common injuries in orthopaedics is a distal radius fracture. When describing a distal radius fracture, make it a habit to mentally assess the scapholunate interval to detect concomitant injuries there.

## **KEY TERMS**

carpal instability, scapholunate ligament, lunotriquetral ligament, perilunate dislocation, dorsal intercalated segment instability (DISI), volar intercalated segment instability (VISI)

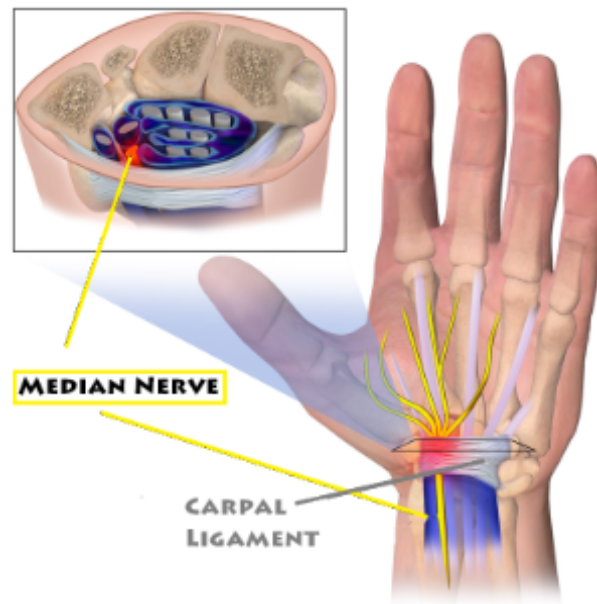
## **SKILLS**

Identify the surface anatomy distal edge of radius, scaphoid, lunate and triquetrum on an uninjured wrist. Perform a Watson test during clinical exam. Name and identify the proximal carpal bones, and recognize, if present, any widened intervals between them on AP radiographs and any lunate (“tea cup”) tilt on lateral radiograph.

## CARPAL TUNNEL SYNDROME

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Carpal Tunnel Syndrome is a compression neuropathy of the median nerve as it passes through the carpal tunnel at the wrist. Patients with Carpal Tunnel Syndrome typically present with numbness and tingling in the thumb, index finger and long fingers, as well as nocturnal wrist or hand pain. Prolonged compression of the median nerve (Figure 1) may also lead to thenar muscle weakness and fine motor difficulty. Carpal Tunnel Syndrome is common and costly: its economic costs in the USA alone are estimated to exceed \$2 billion per year, where approximately 500,000 carpal tunnel releases are performed per year.



*Figure 1: Carpal Tunnel Anatomy: the median nerve accompanies the flexor tendons as they pass through the carpal tunnel at the wrist.*  
(Credit Wikipedia. <http://bit.ly/1Q7JcxZ>)

### STRUCTURE AND FUNCTION

The median nerve originates from the lateral and medial cords of the brachial plexus with contributions from the C6, C7, C8, T1, and sometimes C5 nerve roots.

The median nerve enters the forearm between the heads of the pronator teres muscle and within the forearm, it innervates the pronator teres, flexor carpi radialis, palmaris longus, and flexor digitorum superficialis muscles.

The anterior interosseous nerve branches off the median nerve and innervates the flexor digitorum profundus muscle to the index and long fingers, flexor pollicis longus, and pronator quadratus.

The palmar cutaneous sensory branch of the median nerve originates proximal to the carpal tunnel and provides sensation to the thenar eminence.

The recurrent motor branch of the median nerve, which innervates the lumbricals to the index and long fingers and the opponens pollicis, abductor pollicis brevis, and flexor pollicis brevis muscles, originates as

the median nerve exits the carpal tunnel. However anatomic variants exist, and the nerve may branch more proximally within the tunnel or may course through the transverse carpal ligament as it travels toward the thenar musculature.

The median nerve supplies sensory innervation to the palmar aspect of the thumb, index finger, long finger, and the radial one-half of the ring finger as well as the radial aspect of the palm.

The floor of the carpal tunnel consists of the carpal bones. The transverse carpal ligament forms the roof of the carpal tunnel and extends from the hamate and pisiform ulnarly to the trapezium and scaphoid radially.

The following 10 structures pass through the carpal tunnel: median nerve, flexor digitorum superficialis tendons (4 total), flexor digitorum profundus tendons (4 total), and flexor pollicis longus tendon.

Normal pressure measurements within the carpal tunnel average around 2.5 mmHg. A decrease in epineural blood flow may be seen at pressures greater than 20 mmHg. Hand position can also affect the pressure: wrist extension, wrist flexion, and digit flexion also lead to elevated carpal tunnel pressure.

Acute Carpal Tunnel Syndrome is the result of sudden, sustained compression of the median nerve secondary to local trauma, fracture, or hematoma.

Carpal Tunnel Syndrome more commonly occurs as the result of chronic compression of the median nerve. Most patients present with no identifiable cause and are considered idiopathic cases. Histologic evaluation reveals proliferation of edematous and fibrous tissue within the carpal tunnel.

Anatomic causes of Carpal Tunnel Syndrome include osteophytes, ganglion cysts, tumor, proliferative tenosynovitis, hematoma, infection, and a persistent median artery.

Multiple systemic illnesses are associated with the presence of Carpal Tunnel Syndrome. The most common of these include obesity, diabetes mellitus, and hypothyroidism.

Rheumatoid arthritis may cause chronic compression of the median nerve at the wrist as a result of local pannus formation (synovial proliferation).

Carpal Tunnel Syndrome may occur during pregnancy, usually during the third trimester (perhaps from fluid retention); these cases typically resolve following delivery.

Carpal tunnel syndrome has not been shown to be caused by office work such as typing or using a computer mouse. The NIH website stated explicitly, "There is little clinical data to prove whether repetitive and forceful movements of the hand and wrist during work or leisure activities can cause carpal tunnel syndrome." Nonetheless, it is of course possible that patients with carpal tunnel syndrome may *experience* symptoms of their disease when engaged in such activities. (By analogy, a patient with coronary artery disease may experience angina when running up the stairs. But just as stair climbing does not cause coronary artery disease, typing does not cause Carpal Tunnel Syndrome. Rather, both activities may simply provoke symptoms.)

## **PATIENT PRESENTATION**

Carpal Tunnel Syndrome presents clinically as numbness, tingling, and paresthesias of the thumb and radial digits.

Patients may also experience distal forearm, wrist or hand pain. Pain at night is a common complaint, and many patients report relief with "shaking out" their hands.

In addition to sensory disturbances, chronic median nerve compression may lead to hand weakness, loss of dexterity and fatigability as a result of decreased motor nerve conduction and muscle atrophy.

A thorough upper extremity evaluation including strength and neurologic testing may reveal any of the following:

1. Atrophy or flattening of the thenar eminence.
2. Reported decreased sensation to light touch and pin prick in the median nerve distribution (and a related finding: two-point discrimination >5mm).
3. Weakness with palmar abduction of the thumb as a result of the near muscle weakness.

Tinel's test is performed by percussing the course of the median nerve as it crosses the carpal tunnel into the palm. A positive test elicits shock-like sensations in one or more of the radial digits.

Phalen's test involves having the patient flex the wrist for a period of 60 seconds to see if that provokes numbness and tingling in the median nerve distribution.

The examiner performs Durkan's compression test by applying direct pressure to the wrist in the region of the median nerve at the carpal tunnel for 30 seconds. Like Phalen's test, Durkan's test is considered positive if numbness and tingling in the median nerve distribution is elicited.

## **OBJECTIVE EVIDENCE**

Baseline wrist radiographs should be obtained to rule out fracture, bone spurs, dislocation, or other anatomic sources of median nerve compression. Radiographs, however, are generally of low yield. MRI or ultrasound have been described as adjuncts in the evaluation of tumors or cysts suspected of causing Carpal Tunnel Syndrome, however, these are not commonly performed, and more sensitive and specific tests exist to confirm the diagnosis.

While specific blood tests may be useful in diagnosing systemic conditions predisposed to developing Carpal Tunnel Syndrome, such as hypothyroidism or diabetes mellitus, there are no known serologic studies or markers for compression neuropathies.

Nerve conduction tests and electromyography are commonly performed for evaluation of patients with suspected Carpal Tunnel Syndrome. Nerve conduction tests determine the velocity with which impulses are conducted across specific nerves. Electromyography (EMG) directly measures muscle response to electrical stimulation. The decision to order nerve conduction tests/EMG is a so-called "clinical judgment," meaning that this threshold is not defined precisely.

When a nerve is compressed the velocity with which impulses are conducted will be decreased. Hence, the finding of decreased median nerve conduction velocity as the nerve passes in the carpal tunnel (with normal velocity elsewhere) can be considered as objective evidence of compression. Some patients, however, have Carpal Tunnel Syndrome despite borderline normal nerve conduction testing. (Pre-operative nerve conduction testing is particularly useful in case there is recurrence or failure to improve with treatment.)

Sensory testing is based on the reports of patients' subjective responses and therefore should not be considered objective evidence.

## **EPIDEMIOLOGY**

The incidence of Carpal Tunnel Syndrome is 1 to 3 per 1000 persons per year. The prevalence is 50 cases per 1000 persons. It is more common among women than men and more common with older age. Bilateral involvement is also common.

## DIFFERENTIAL DIAGNOSIS

Carpal Tunnel Syndrome is a compression neuropathy of the median nerve at the wrist, and therefore, by definition, affects only structures in the median nerve distribution distal to the wrist (with referred pain in the forearm, occasionally).

Patients with cervical radiculopathy may present with complaints suggestive of Carpal Tunnel Syndrome.

Local pathology within the hand (arthritis, tendinitis), too, may mimic some of the symptoms associated with Carpal Tunnel Syndrome.

Last, systemic neurological conditions can produce disturbances observed in Carpal Tunnel Syndrome (e.g. pernicious anemia and folate deficiency).

## RED FLAGS

Acute Carpal Tunnel Syndrome as a result of trauma (e.g. radius fracture) requires urgent surgical decompression.

## TREATMENT OPTIONS AND OUTCOMES

### *Non-operative*

Treatment for Carpal Tunnel Syndrome should begin with non-operative treatment. The most commonly prescribed conservative intervention is wrist splint immobilization (especially for night-time symptoms); such splinting minimizes pressure in the canal.

Non-steroidal anti-inflammatory medications may provide symptomatic relief, however the benefit has not been clearly demonstrated in the literature.

For patients with potential occupational risk factors, ergonomic considerations such as workspace modifications may provide some relief; such an approach is justified empirically.

Corticosteroid injection into the carpal tunnel serves not only a therapeutic role but also aids in confirming the diagnosis of Carpal Tunnel Syndrome: symptomatic relief after injection helps confirm the diagnosis. Approximately 1 in 5 individuals remain symptom free at 1 year following injection. Patients with mild disease and those with symptoms for less than 1 year are more likely to experience longer-term relief.

### *Operative*

Surgical decompression of the median nerve at the carpal tunnel involves release of the transverse carpal ligament. A number of surgical techniques (including open (Figure 2), mini-open, and endoscopic) have been described. In each, care is taken to avoid injury to the recurrent motor branch.

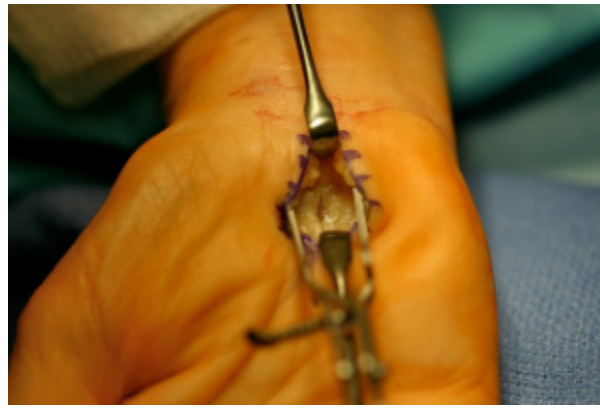


Figure 2: Open carpal tunnel involves release (Credit: flickr.com  
<http://bit.ly/1RVTzuJ>)

Surgical decompression is the most reliable intervention available to treat Carpal Tunnel Syndrome. Recurrence following surgical release ranges from 5% to 20%. Sensory and motor recovery is variable and difficult to predict, however, the pain associated with chronic nerve compression typically resolves. Nerves that have been chronically compressed and thus subjected to longer durations of epineural ischemia are less likely to demonstrate full recovery. This highlights the importance of early diagnosis and therapeutic intervention when Carpal Tunnel Syndrome is present.

## MISCELLANY

A systematic review of conservative treatment of carpal tunnel syndrome (PMID: 17613571) found that vitamin B6 and exercise therapy are ineffective. There is limited or conflicting evidence whether yoga, laser and ultrasound are effective treatments.

A positive Tinel's test has been referred to as "Tinel's sign," however this is a misnomer: the perceived and reported response is a *subjective symptom*, not an objective sign. It has been proposed that specific subjective responses to focused tests be labeled as "wigns," spelled like "sign" but pronounced "whine," to emphasize the blending of the subjective and objective realms.

## KEY TERMS

Carpal Tunnel Syndrome; median nerve; compression neuropathy, anterior interosseous nerve

## SKILLS

Identify and effectively evaluate persons presenting with symptoms of Carpal Tunnel Syndrome. Recognize clinical signs consistent with median nerve compressive neuropathy. Perform provocative examination maneuvers specific to Carpal Tunnel Syndrome as part of the physical exam. Recognize adjunctive tests that aid in the evaluation and diagnosis of Carpal Tunnel Syndrome. Understand potential interventions to treat the disease.

## COMPRESSION NEUROPATHIES OF THE ARM

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Pressure on peripheral nerves can cause numbness, weakness or pain — compression neuropathy — in the areas the nerves supply. Pressure on the median nerve at the wrist (i.e., carpal tunnel syndrome) is the most common compression neuropathy of the upper extremity but compression of the ulnar, radial, posterior interosseous and anterior interosseous nerves is also seen. Among these, compression of the ulnar nerve in the cubital tunnel at the elbow is the more common. The diagnosis of compression neuropathy is made by clinical correlation of the patient's complaints with the anatomic distribution of the nerve, and may be confirmed by electro-diagnostic studies.

### STRUCTURE AND FUNCTION

Within a peripheral nerve, axons are surrounded by endoneurium (shown in black in Figure 1). A group of such axons are then surrounded by perineurium (shown in blue); these groups are called a fascicle. A collection of fascicles may be in turn bundled together with blood vessels (red) within yet another sheath known as the epineurium (shown in green). The blood vessels then penetrate into the perineurium.



*Figure 1: The anatomy of a nerve, showing endoneurium (black) around the axons composing the fascicle, in turn surrounded by perineurium (shown in blue) and then epineurium (shown in green). (Modified from wikipedia. <http://bit.ly/1Sydsqv>)*

Mild nerve compression produces a reduction in epineural blood flow. Prolonged compression can then reduce endoneural blood flow and produce ischemia. Ischemia leads to breakdown of the blood–nerve barrier. Antigens released within the perineurial space then can incite an inflammatory response.

Eventually, scar tissue may form, and in turn irreversible damage to the nerve may result.

When patients with compression neuropathy undergo surgical decompression, the compressed nerve often is indented at the point of compression. Synovitis, sclerosis, fibrous hypertrophy, and edema of the synovium are also seen.

The individual nerves each have their own so-called “sensory domain”, that is, area of skin whose sensation is provided (exclusively) by that peripheral nerve (Figures 2-4). As such, a sensory examination (or related complaints) can help hone the diagnosis.

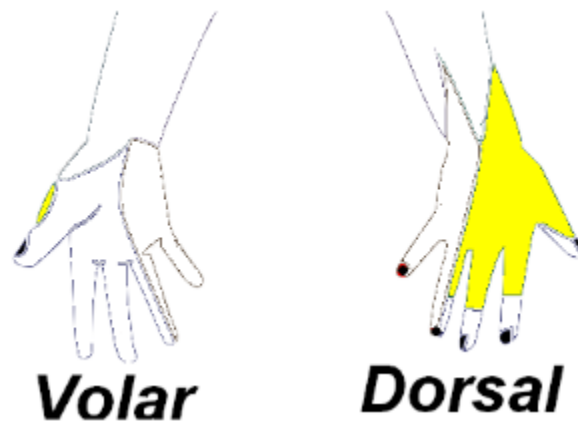


Figure 2: The sensory domain of the radial nerve shown in yellow.  
Modified from wikipedia

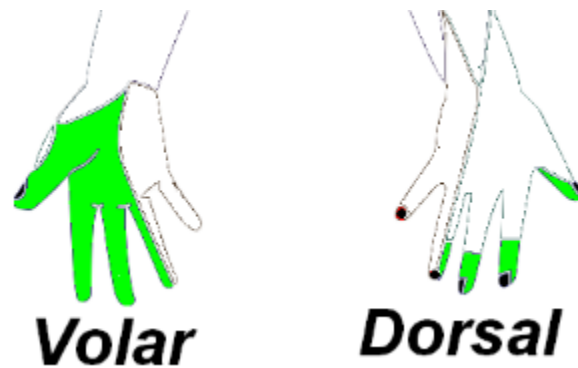


Figure 3: The sensory domain of the median nerve shown in green.  
Modified from wikipedia

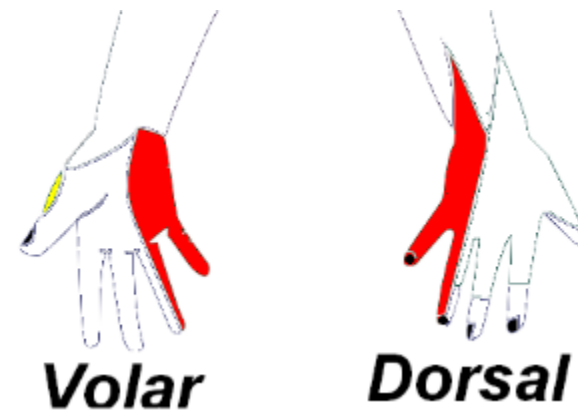


Figure 4: The sensory domains of the ulnar nerve shown in red.  
Modified from wikipedia

The nerves to the hand also have unique motor function. For example, the radial nerve supplies the muscles that extend the wrist; thus, radial nerve dysfunction leads to a wrist drop. (Other examples are detailed below.)

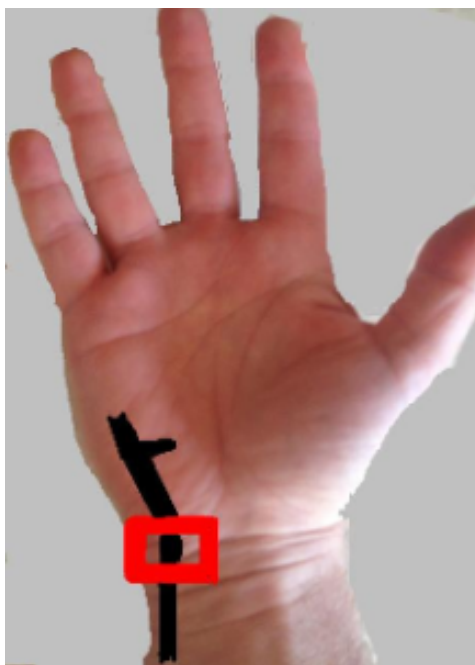
Cubital tunnel syndrome (ulnar nerve compression near the elbow) may be caused by the presence of an accessory muscle, the anconeus epitrochlearis. This muscle, present in about 1/4 of the population, can become edematous and press on the nerve.

Cubital tunnel syndrome may be aggravated by or associated with prolonged periods of flexing the elbow, such as during sleep or holding a phone while working at a desk.



Radial nerve compression may arise from prolonged pressure on the nerve from the surrounding muscles in the proximal forearm area, or from direct pressure to the posterior aspect of the arm. This direct pressure mechanism is seen in patients who fall sleep on their arms; when it is another person's body that exerted the pressure, the condition is known as "honeymoon palsy."

Compression of the ulnar nerve in Guyon's canal (Figure 5) can be found with a fracture of the hamate bone or aneurysmal swelling of the ulnar artery. Compression of the ulnar nerve at the wrist is also seen in avid bicyclists, who may rest their wrists against their handlebars. The most common cause of ulnar nerve compression at Guyon's canal is a ganglion.



*Figure 5: The approximate surface landmarks of Guyon's canal are shown in red, through which the ulnar nerve (black) passes.*

## **PATIENT PRESENTATION**

Compression neuropathy presents as numbness, weakness, pain, or a mixture thereof. The symptoms produced depend on which nerve is involved, where along the nerve it is being compressed, and the severity of the condition.

### ***Ulnar nerve***

Common symptoms of cubital tunnel syndrome, a compression neuropathy of the ulnar nerve at the elbow, are numbness of the small finger, and ulnar half of the ring finger. There may be associated complaints of hand weakness or muscle wasting (causing visible deformity of the hand).

When compression of the ulnar nerve occurs more distally around the wrist and hand (i.e. within Guyon's canal), patients report altered sensation on the palmar surfaces of the small and ring fingers but have normal sensation on the dorsum of the hand. There may also be weakness of the intrinsic muscles of the hand. Ulnar nerve dysfunction may produce a so called Wartenberg's sign (Figure 6); more subtle weakness will be seen only with provocative testing (Figure 7).



Figure 6: Wartenberg's sign is found when a patient's small finger remains Abducted despite an instruction "bring all of your fingers together." The finger may even rest in this position because the extensor tendons pass slightly ulnar to the midpoint of the finger and thus exert a slight abduction force. If the ulnar nerve is dysfunctional, the extensors (which are innervated by the radial nerve) will overpower the intrinsic adductor muscle innervated by the ulnar nerve and the finger will "escape" as shown.

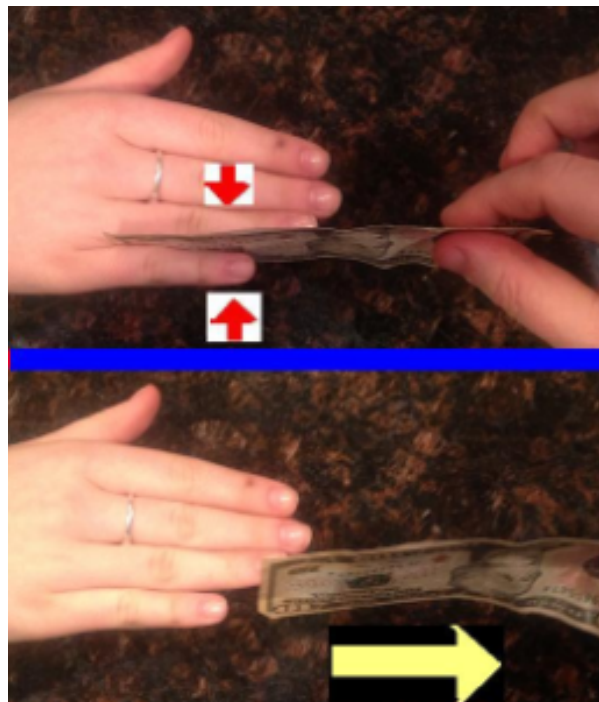


Figure 7: Ulnar nerve dysfunction leading to subtle weakness. More subtle weakness of adduction can be demonstrated by asking the patient to hold a piece of paper between the ring and small fingers (by adducting them, as shown by the red arrows in the top photo). If the ulnar nerve is dysfunctional, the force of adduction, though sufficient to bring the fingers closer together than what is seen with frank paralysis, will not be able to hold on to the paper.

### **Radial nerve**

The radial nerve runs along the humerus, and compression in the posterior aspect of the upper arm may produce weakness in wrist extension (causing a so-called 'wrist drop' (Figure 8)), weakness in finger extension (including the thumb), and decreased sensation along the dorsum of the hand. If the pressure on the nerve is

more distal in the proximal forearm, there may be soreness in the mobile wad area of the proximal forearm and the posterior interosseous nerve alone is affected. The main weakness is of finger and thumb extension; wrist extension will be relatively spared and there are no sensory changes.



Figure 8: Radial nerve dysfunction can produce a "wrist drop."

#### **Anterior interosseous nerve**

When compression involves the anterior interosseous nerve (running in the forearm opposite the posterior interosseous nerve), there is paralysis or weakness of the flexor pollicis longus, the flexor digitorum profundus to the index and long fingers and the pronator quadratus. Compression of the anterior interosseous nerve produces no sensory loss. An affected patient attempting to make an "OK" sign would instead form a triangle, as shown in Figures 9 and 10.



Figure 9: This figure shows intact anterior interosseous nerve function. When the patient is asked to make an "OK sign", there is flexion present at the thumb IP joint and index finger DIP joint.



*Figure 10: Anterior interosseous nerve dysfunction. When the anterior interosseous nerve does not work normally, there is weakness of flexor pollicis longus and flexor digitorum profundus to the index and long fingers producing an absence of flexion at the thumb IP joint and index finger DIP joint when trying to make an "OK sign."*

## OBJECTIVE EVIDENCE

Plain x-rays may be used to detect underlying anatomic abnormalities such as bony spurs or fracture fragments that may be a source of compression.

Magnetic resonance imaging may identify tumors or cysts that can compress nearby nerves. MRI is frequently indicated to evaluate for a mass in ulnar nerve compression at Guyon's canal.

Electrodiagnostic tests — nerve conduction studies and electromyography — can test the function of specific nerves. Because there is a fairly high prevalence of abnormal results on nerve conduction studies and electromyography (both false positives and false negatives), these tests are best considered as tools as an adjunct to physical examination, but not for screening purposes.

## EPIDEMIOLOGY

The incidence of cubital tunnel has been measured in one study to about one in 5,000 (PMID: 15993135). The incidence for the other peripheral neuropathies is known to be lower, but albeit imprecisely so. Note that mild forms of compression neuropathy will not be reported, as such patients will not present for care.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of compression neuropathy can be categorized into three main areas:

1. Diseases with dysfunction of the peripheral nerve, but not because of compression. Amyotrophic lateral sclerosis, multiple sclerosis, leprosy (Hansen's disease is the eponym for infection of the nerves by mycobacterium leprae), Parsonage-Turner syndrome (viral infectious neuritis), and brachial neuritis — all fairly rare conditions — can cause peripheral nerve dysfunction.
2. Cervical radiculopathy. Note that single level compression of one root often produces no gross weakness, and the symptoms would respect the dermatomal border of that nerve root, and not that of the peripheral nerve.
3. Local musculoskeletal pathology. For instance, arthritis of the base of the thumb can cause localized

pain on the radial side of the hand, or bony spurs arising from the edge of joints may cause mechanical rupture of adjacent tendons. (Note: local musculoskeletal conditions that mimic neuropathies should have no associated sensory deficit.)

Distinguishing a cervical radiculopathy from a compression neuropathy may be difficult. As shown, the differences between sensory areas are subtle (Figure 11). The main distinction is that the sensory symptoms of a compression neuropathy typically are located more distally, in the hand only. Also, the weakness tends to be less severe in cases of cervical radiculopathy, as the affected muscle has input from other cervical levels. (This of course gets confusing when multiple cervical levels are involved, as may be the case.) Focal neck complaints may be the main distinguishing feature of cervical rather than extremity disease.



Figure 11: The close resemblance between the sensory distribution of the C8 nerve root and that of the ulnar nerve. The “textbook” distinction, namely, that the ulnar nerve supplies only the ulnar side of the ring finger, is not reliable enough for clinical use.

## RED FLAGS

Generalized complaints, involvement of multiple extremities and central or cranial nerve disturbances are all worrisome and suggest a diagnosis beyond a compression neuropathy. Tongue fasciculations, visual changes, altered gait and a sense of imbalance in particular suggest neurologic disease.

## TREATMENT OPTIONS AND OUTCOMES

Logically, the treatment of a compression neuropathy should be the decompression of the nerve. Nonetheless, a non-operative approach centering on modification of activity may be sufficient, as the compression may have indeed been produced from overuse. Also, some patients may improve spontaneously or learn to tolerate their symptoms (if mild).

At times, splinting the wrist or elbow can minimize the pressure on the nerve. Better still, offending activities should be stopped. For example, avoiding riding a bicycle or hyperflexing the elbow may completely relieve compression of the ulnar nerve at the wrist and elbow respectively.

When the symptoms of compression do not abate with non-operative treatment, surgical decompression may be indicated. Surgical decompression is effective when there is ischemia caused by pressure (i.e. when the diagnosis is correct) and the nerve has not been irreversibly damaged (and the diagnosis is timely).

## RISK FACTORS AND PREVENTION

Mechanical risk factors for compression of the ulnar nerve at the elbow include a supracondylar (distal humerus) fracture in childhood, causing a so called “tardy” (or late-appearing) ulnar nerve palsy and chronic valgus stress, as from pitching in baseball.

## MISCELLANY

Because many patients with a compression neuropathy are apt to get better spontaneously, there are probably many patients who, having tried a non-traditional approach to treatment, will credit this treatment for their (truly spontaneous) recovery. Even “traditional treatments” may benefit from this line of thinking: e.g., the beneficial effect of local injections may be on the basis of a placebo effect. In general, any treatment that “amuses the patient while nature takes its course,” as Voltaire put it, may be reasonable as long as it does not put the patient at risk. In that regard, patients with nerve ischemia may progress from a state of reversible neuropathy to one of permanent damage; and thus, not always is a “watch-and-wait” approach risk-free.

The use of poor-fitted crutches can lead to a compression neuropathy of the axillary nerve: crutches that are too long can press into the axilla.

As noted, the term “honeymoon palsy” refers to a compression neuropathy (typically of the radial nerve) that results from excessive pressure on the arm from one’s bed mate. Cheiralgia paresthetica is the name for sensory abnormalities in the radial nerve distribution from tight handcuffs or other causes.

## KEY TERMS

Ulnar nerve, Electromyography (EMG), Cubital tunnel syndrome; wrist drop; Guyon’s canal

## SKILLS

Recognize the findings on history that are particular to lesions of the radial, median and ulnar nerves versus generalized conditions that may be neurologic or rheumatologic in nature but may mimic upper extremity compression syndromes. Perform physical examination maneuvers that assess the radial, median and ulnar nerves.

## DE QUERVAIN'S DISEASE

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De Quervain's disease is a common cause of pain at the base of the thumb and the radial side of the wrist. This condition is characterized by irritation of the extensor pollicis brevis and abductor pollicis longus, as they pass through their sheaths in the so-called "first compartment" on the dorsum of the wrist. The cause of de Quervain's disease is often unknown, but repetitive grasping tasks and rheumatoid arthritis are associated with increased incidence. The use of a splint usually leads to relief, but injection with steroids, or failing that, surgical release of the tendon sheath may be needed.

### STRUCTURE AND FUNCTION

There are six compartments on the dorsal side of the wrist (as shown in figure 1).

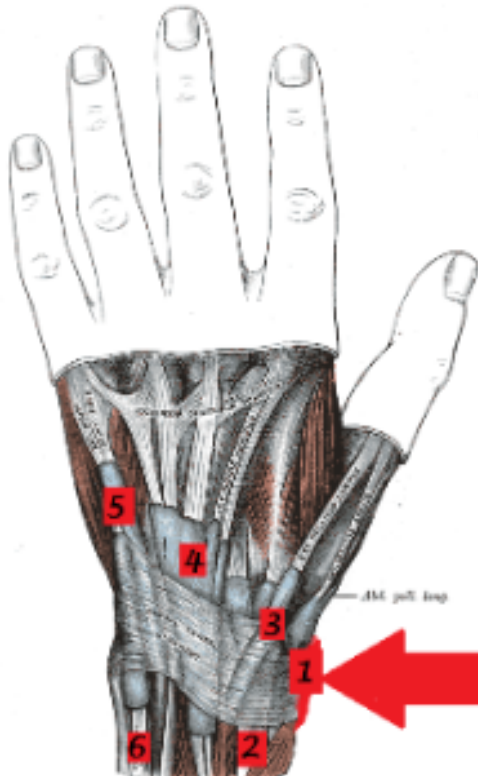


Figure 1: The six compartments on the dorsal side of the wrist (modified from wikipedia. <http://bit.ly/1PQbUcw>)

- The first compartment (and the one involved in de Quervain's disease) houses the extensor pollicis brevis and abductor pollicis longus. This compartment lies over the radial styloid

The other compartments are as follows:

- The second compartment holds the extensor carpi radialis longus and extensor carpi radialis brevis
- The third compartment holds the extensor pollicis longus
- The extensor indicis proprius and the extensor digitorum communis (to 4 fingers) are in the fourth compartment
- The fifth compartment has the extensor to the fifth finger, the extensor digiti minimi
- The extensor carpi ulnaris is in the sixth compartment.

All of these extensor tendons emanate from the forearm and cross the wrist. They are secured by the extensor retinaculum above and by the bones below. All have synovial tendon sheaths to keep them in place and to reduce friction.

Because these tendons are in an enclosed space, any process that makes the tendon larger or the surrounding tissue more confining will impede normal motion of the affected tendon.

De Quervain's disease is frequently associated with attrition and degeneration of the affected tendons. Reactive swelling may be seen as well. In turn, there is resistance to gliding and pain.

The histopathology of de Quervain's disease was studied by Clarke et al (PMID: 9888670). They reported that "the condition was not characterized by inflammation, but by thickening of the tendon sheath and most notably by the accumulation of mucopolysaccharide, an indicator of myxoid degeneration. These changes are pathognomonic of the condition and are not seen in control tendon sheaths." They further stated "de Quervain's disease is a result of intrinsic, degenerative mechanisms rather than extrinsic, inflammatory ones."

Overuse, a direct blow to radial side of the wrist and repetitive grasping are all thought to cause de Quervain's disease, but clearly not always does de Quervain's disease follow these "inciting events." In addition, there are cases with de Quervain's disease with no known cause. In sum, the question of causality has not been answered definitively.

Pregnancy and rheumatoid arthritis are associated with increased incidence of clinically significant de Quervain's disease.

It is possible that anatomic abnormalities, causing the tendon to take a less direct course or perhaps to be caught by a septum within the compartment, may be causal in a minority of cases.

## PATIENT PRESENTATION

The chief complaint of de Quervain's disease is pain on the thumb side of the wrist, worsened with grasping.

Swelling may be seen; if so, it is likely caused by an associated retinacular (ganglion) cyst.

Some patients notice a catching sensation as the tendons to the thumb are momentarily caught within their sheath.

De Quervain's disease can be confirmed clinically with Finkelstein's test: in this test, the thumb is brought toward the ulnar side of the hand by the examiner, as shown below in Figure 2. A positive test is characterized by reproduction of pain at the distal radius.

Another variation is for the patient to place the thumb under the other 4 (flexed) fingers. Then, with the thumb so stabilized, the examiner ulnar-deviates the hand, thereby stretching the extensor pollicis brevis and abductor pollicis longus tendons attached to the thumb.

(The extensor pollicis brevis and abductor pollicis longus are muscles that cause radial abduction of the thumb away from the other fingers, in the plane of the palm. The provocative movement in Finkelstein's test can be thought of as forced ulnar adduction with the tendons under tension.)





Figure 2: Finkelstein's test (from Wikipedia. <http://bit.ly/23Emwzo>)

## OBJECTIVE EVIDENCE

Plain radiographs are generally not needed in diagnosing de Quervain's disease, but can help rule out other processes including fractures (of the radial styloid or carpus) and arthritis.

Lab studies are likewise not part of the diagnostic work-up.

## EPIDEMIOLOGY

Like most conditions that are diagnosed clinically, are self-limited and may have mild forms, de Quervain's disease resists precise epidemiological measurement.

De Quervain's disease is more common among women.

De Quervain's disease is seen in pregnant women, and even more so in the immediate post-partum period.

## DIFFERENTIAL DIAGNOSIS

Thumb pain can be caused by osteoarthritis of the basal joints. (Note: arthritis and de Quervain's disease frequently co-exist.)

Carpal tunnel could cause pain in the thumb, but would not be provoked by Finkelstein's test and would typically involve other fingers.

So-called "intersection syndrome" i.e. pain at the intersection of the abductor pollicis longus and extensor pollicis brevis with the extensor carpi radialis longus and brevis, can cause similar symptoms, though more proximal in the forearm.

Compression of the superficial branch of the radial nerve may cause symptoms in the area characteristic of de Quervain's disease, but would not be affected by Finkelstein's test.

## RED FLAGS

De Quervain's disease has no red flags per se, but treatment with injections can lead to infection which must be detected promptly.

## TREATMENT OPTIONS AND OUTCOMES

The first line treatment includes rest, thumb splinting and NSAIDs. Splinting should keep the wrist neutral and the thumb slightly abducted.

If splinting does not work (adequately), then injection into the first compartment with steroid may help (Figure 3 and 4). One mL of dexamethasone and 1 mL of lidocaine is a reasonable combination. The addition of the local anesthetic (lidocaine) can allow both the physician and patient to get an immediate sense if the medicine was placed correctly (anatomic variance may make it hard to be sure that an injection hit its target) and whether the treatment is apt to work.



Figure 3: The first compartment is identified by palpation, and “marked” by gentle pressure of the examiner’s thumb nails (courtesy of Stuart Myers, MD)



Figure 4: After outlining the compartment (the indentation made by the examiner’s thumb nails is shown in green), the site is injected (courtesy of Stuart Myers, MD)

Surgical release is reserved for those who fail all other modes of treatment. The goals of surgery include complete decompression (including multiple subcompartments within the “first compartment”) and avoidance of the radial sensory nerve.

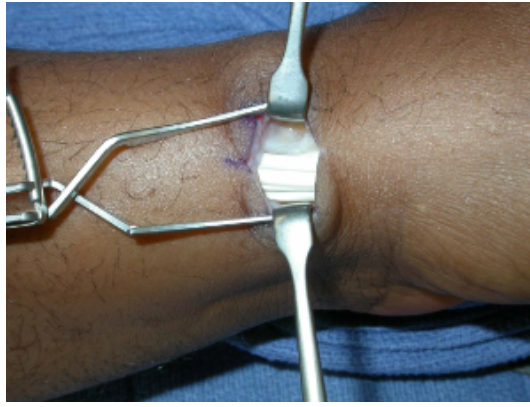


Figure 5: De Quervain's disease surgical release (courtesy of Gene Deune, MD)

Richie and Briner (PMID: 12665175) report “an 83% cure rate with injection alone.” Patients were eligible for inclusion if they had pain at the radial wrist, tenderness at the first dorsal wrist extensor compartment, and a positive Finkelstein test. Patients were considered cured if all three signs were obliterated.

Richie and Briner further report that the cure rate with injection alone “was much higher than any other therapeutic modality” including injection plus splinting (a manifestation of the healthful effects of stress, the authors posit). Interestingly, they found a 0% cure rate for rest or nonsteroidal anti-inflammatory drugs.

Aside: this latter finding – which does not match common clinical experience — shows how difficult it is to assess a case series reporting on conditions that are diagnosed clinically. First, it may well be that rest will eliminate pain and tenderness, but not obliterate the Finkelstein test response. That “two out of three” outcome is probably not a clinical failure, though it is by these authors’ standard. Also, complete obliteration of signs and symptoms may not be needed for clinical success either. (The inventor of a non-invasive method to reduce back pain by 80% without complications will have a large fortune in no time.) Last, patients who were not given any affirmative treatment, but rather rest alone, will not benefit from any placebo effect either.

## RISK FACTORS AND PREVENTION

De Quervain's disease seems to be caused by processes that cannot be avoided. As such, there is no meaningful program for prevention. That said, if a particular inciting event seems to increase symptoms, common sense would dictate trying to minimize that activity.

## MISCELLANY

This condition is named after a Fritz Quervain, a Swiss physician perhaps more famous for his description of Giant Cell Thyroiditis. His name is pronounced something like “Care Van.”

## KEY TERMS

de Quervain's, Finkelstein's test, tendinitis

## SKILLS

Able to perform Finkelstein's test.

## DISORDERS OF THE NAIL

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The fingernail serves multiple functions. In addition to protecting the fingertip, it provides tactile sensation, aids in thermoregulation, and assists in picking up small objects; it also has dense lymphatics in the hyponychium that help resist infection. The fingernail is the most frequently injured portion of the finger secondary to its prominent location. The nail is also susceptible to infection and may be the site of tumors, both benign and malignant. Last, the nail may display signs of underlying systemic disease.

### STRUCTURE AND FUNCTION

The nail is a plate of keratin that covers the dorsal aspect of the distal phalanges of the fingers and toes (Figure 1).

The nail plate (which, in layman's terms, is the "nail" itself) is a hard sheet of translucent keratin in which lie several layers of dead, compacted cells.

The nail bed is the tissue that lies beneath the nail plate. The nail bed contains nerves, lymphatics and capillaries. (The function of these blood vessels can be assessed by compressing the nail to momentarily occlude them, and then releasing the pressure. So-called "capillary refill" indicates good flow.)

Within the nail bed is the germinal matrix, the source of cells that become the nail plate. As these cells are produced, older cells are pushed forward and compressed, giving rise to the typical growing pattern of the nail.

The eponychium is a small band of epithelium that covers the proximal aspect of the nail; the paronychia is a similar border tissue around the medial and lateral borders.

The cuticle is a layer of epidermis that folds back over the surface of the nail plate at its base.

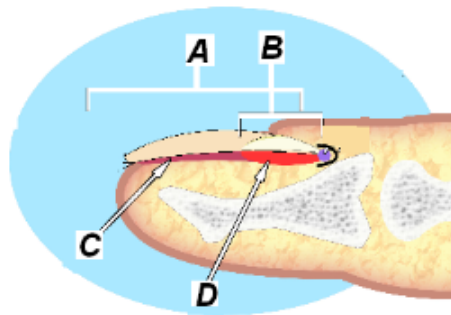


Figure 1: Nail anatomy. A is the nail plate; B is the lunula; C is the nail bed; and D is the germinal matrix. (modified from Wikipedia. <http://bit.ly/1nZphdS>)

## TRAUMA

Trauma to the nails is common, with the middle finger being most commonly affected due to its length and greater exposure compared to adjacent shorter digits. There are often associated fractures of the distal phalanx.

Trauma may result in a painful subungual hematoma (Figure 2), a collection of blood compressed between the nail plate and the nail bed on the distal phalanx.



*Figure 2: Subungual hematoma. Treatment involves placing a hole in the nail plate to relieve the pressure.*

If the subungual hematoma involves more than 50% of the nail, the nail plate is generally removed and the nail bed repaired with fine absorbable sutures (Figure 3).



*Figure 3: Nail bed repair*

### ***Nailbed lacerations***

Nailbed lacerations are often the result of crush injuries, producing a stellate laceration pattern. These injuries require repair. Without adequate repair (and sometimes despite best efforts) the nailbed will not heal smoothly.

Because nailbed lacerations are typically crush injuries, x-rays should always be obtained to exclude a fracture.

In children, it may be possible to repair the nailbed with a cyanoacrylate adhesive (e.g., “Dermabond”). The cosmetic outcome with this approach is comparable to that of suture repair. This option is advantageous in the pediatric population specifically because it avoids keeping the child motionless and is relatively pain-free.

### **Hook nail deformity**

A hook nail deformity is a condition that can appear after a traumatic amputation of the distal aspect of the fingertip (that is, soft tissues and the tuft of the distal phalanx). When the nail bed loses its distal support, the nail can grow downward around the tip of the finger.

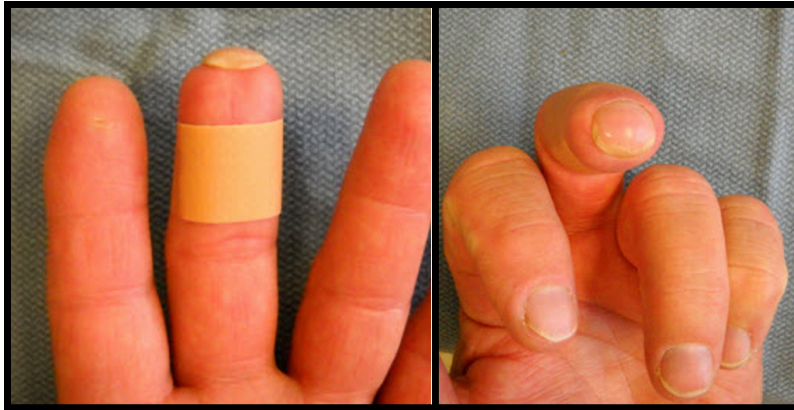


Figure 4: Hook nail. The nail is shown growing around the tip of the finger

## **INFECTION**

The nail is a site for both acute and chronic infections. Acute infections often result from a minor event, such as a superficial laceration or pulling a hangnail, with bacteria — typically *Staphylococcus* and *Streptococcus* — being the most common etiology. Chronic infections are typically fungal and may result from multiple acute infections treated with antibiotics or chronic moisture, with *Candida* being the most common etiology. Less commonly, atypical organisms, such as *Mycobacterium*, are the source of infection.

### **Paronychia**

Paronychia (a collection of pus between the nail plate and the nail fold) and felons (a purulent collection forms on the palmar surface of the distal phalanx) are discussed in detail in the chapter on hand infections.

### **Herpetic whitlow**

Herpetic whitlow is a viral lesion caused by the Herpes simplex virus and involves the paronychium (Figure 5). Prior to use of gloves by dental and medical professionals, this was seen in workers around oral secretions. It is currently more commonly seen in children who suck their thumb or fingers or in adults following contact with infected genitals. Initially, this has the appearance of an acute bacterial infection, but clear vesicles will eventually develop, helping differentiate this from an acute infection. The process is self-limiting, and incision and drainage should be avoided as a subsequent bacterial infection may occur, prolonging the time for resolution and often requiring additional treatment.





Figure 5: Herpetic whitlow (From Wikipedia. <http://bit.ly/1Pam5TB>)

### **Onychomycosis**

Onychomycosis (i.e., fungal infection of the nail- Figure 6) occurs more in toenails than in fingernails, but can be found in the latter. Diabetes, vascular problems or immunocompromised states are risk factors. Nail fungus can cause three problems (in order of frequency): cosmetic deformity, pain and systemic spread. Onychomycosis is difficult to treat; recurrences are common. Oral medications such as terbinafine do not uniformly clear the infection, and significant side effects (e.g. liver damage) may result. Antifungal lacquers can help clear up some nail fungal infections but require diligent application, frequent debridement and use for up to one year. (That is to say, the efficacy of antifungal lacquers may be limited by issues of compliance.) Ablation of the nail may also be chosen.



Figure 6: Onychomycosis (courtesy of Gene Deune, MD)

## **BENIGN TUMORS**

Tumors of the nail bed are relatively rare, but the physician must keep them in mind when a patient presents with pain or a change in the appearance of the nail. The most common benign tumors associated with the nail complex are mucous cysts (ganglion cysts), pyogenic granuloma, verucca, glomus tumors, and benign melonychia.

### **Ganglion cysts**

Ganglion cysts, more commonly referred to as mucous cysts in this location, are the most common tumor affecting the nail bed (Figure 7). These cysts result from an arthritic spur (osteophyte) of the distal

interphalangeal joint (DIP) of the fingers (or interphalangeal (IP) joint of the thumbs). Depending on the location of the cyst, the nail nearby may be deformed. The cyst may involve the eponychial fold. Treatment should be directed at removal of the stalk of the cyst and the underlying spurs of the joint. Nail deformities often improve following removal of the cyst. Because the cyst originates in the joint (and communicates with it) rupture of the cyst through the skin may introduce bacteria into the joint space.

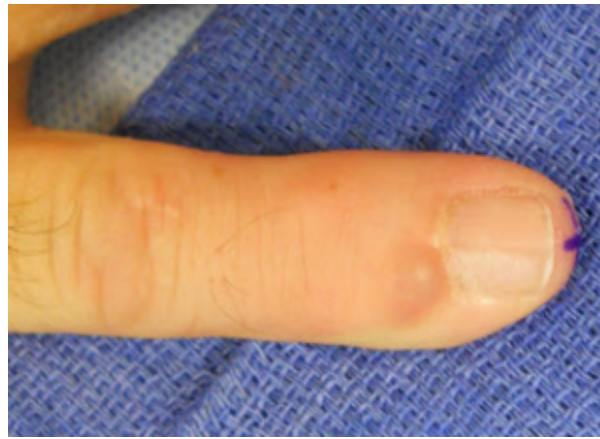


Figure 7: Ganglion (mucous) cyst at the base of the nail

#### ***Pyogenic granuloma***

Pyogenic granuloma is a raised lesion that is a proliferation of granulation tissue; it is frequently traumatized and bleeds (Figure 8). These lesions often develop when a wound has failed to heal and persistent bleeding prompts presentation to the physician. Like the Holy Roman Empire (which was said to be not Holy, Roman or imperial) pyogenic granuloma may be completely misnamed: “pyogenic” suggests infection, incorrectly; and there is no granuloma here either. Accordingly, the term Eruptive Hemangioma has been suggested. Most pyogenic granulomata resolve spontaneously. Those that do not can be removed with surgical excision or thermal ablation (e.g. electrocautery or freezing).



Figure 8: Pyogenic granuloma (From wikipedia.<http://bit.ly/1KSCeet>)

#### ***Verruca vulgaris***

Verruca vulgaris, or the common wart, may present around the perionychium. Multiple treatment methods are available, without any proving to be superior. When the eponychial fold is involved, permanent scarring may result.

#### ***Glomus tumor***

Glomus tumor is a neoplasm of the smooth muscle cells of the glomus body, which regulate blood flow and temperature in the finger (Figure 9). Half of these tumors occur in the subungual area (and give rise to nail



deformities). As might be expected with any mass, glomus tumors can present with severe pain and point tenderness; the more specific finding is focal cold sensitivity. MRI with gadolinium is helpful for diagnosis; treatment involves excision of the mass.



Figure 9: Glomus tumor. (image courtesy of Peter Murray, MD)

## MALIGNANT TUMORS

Squamous cell carcinoma and melanoma are the most common malignancies of the nail complex. Radiographs of the finger should be obtained to evaluate for osseous changes of the distal phalanx.

### *Squamous cell carcinoma*

Squamous cell carcinoma is responsible for approximately 20% of all cutaneous malignancies but is less common than melanoma in the subungual region. It may present with early skin changes or more advanced lesions (Figure 10). Treatment typically involves amputation at the DIP joint of the finger (IP joint of the thumb).



Figure 10: Squamous cell carcinoma of the thumb

### *Malignant melanoma*

Malignant melanoma (Figure 11) accounts for 4% of cutaneous malignancies but is responsible for 80% of the deaths from cutaneous malignancies. Approximately 2% occur in the hand, and half of these are subungual. They may present as a pigmented lesion (Figure 1: Nail anatomy) in the nail bed or in the surrounding eponychium (Figure 2: Subungual Hematoma). A full-thickness biopsy is necessary. Prognosis depends on the depth of invasion and the status of sentinel lymph nodes. Lesions greater than 1 mm in thickness or involving

the entire depth of the nail bed should have sentinel lymph node biopsy to evaluate for metastatic disease. Surgical treatment of the finger typically involves amputation proximal to the adjacent joint. Although Moh's micrographic surgery has been performed in some cases, reconstruction is challenging and often results in a worse functional outcome than amputation.



Figure 11: Malignant melanoma of the finger

#### **Metastatic disease**

Metastatic disease can present in the fingertip, with lung cancers being the most common primary source. In approximately 30% of cases, fingertip metastases are the first presentation of the underlying primary tumor, and may be mistaken at first for an infection.

### **NAIL MANIFESTATIONS OF SYSTEMIC DISEASE**

Clubbing is an increased curvature of the nail and nail fold in a proximal to distal direction, often with the appearance of a bulbous fingertip. The nail plate often has a shiny appearance. This is frequently a manifestation of systemic disease, with cardiac, pulmonary, and gastrointestinal conditions being the most common, although it can occur without underlying etiology. The mechanism by which this occurs has not been clearly elucidated.



Figure 12: Clubbing of the fingers (From Wikipedia. <http://bit.ly/1m8sQNu>)

## RED FLAGS

### *Trauma*

- crush injuries to the nailbed are associated with distal phalanx fractures
- subungual hematomas greater than 50% of the nail should be evacuated to relieve pressure

### *Infection*

- fingernail infection with fungus may be a sign of diabetes, circulatory disorders, a weakened immune system dysfunction
- ruptured mucous cysts communicate with the joint space

### *Tumor*

- Beware of melanoma!

## MISCELLANY

Patients of most vocations and avocations can perform functionally well following injury or amputation of a fingertip. In fact, an article entitled Less than ten – surgeons with amputated fingers in the *Journal of Hand Surgery* describes multiple physicians and surgeons who continue to practice following amputations of a digit.

Fingernails grow approximately 2 inches per year.

Fingernails do NOT grow after death; rather, the surrounding skin retracts, giving that illusion.

Illicit drugs can be identified in nail clippings up to 8 months after use (and unlike hair test, which can be subverted by head-shaving, nail testing is much less likely to be undermined by completely removing the nail).

The first syllable in the word “hangnail” does not refer to the nail “hanging” from the finger; rather, it’s root is *ang*, meaning pain (similar to the word “anguish”).

## KEY TERMS

Fingernail, fingertip, perionychium, nail bed, subungual, mucous cyst, glomus tumor

## SKILLS

Recognize both traumatic and acquired conditions affecting the nail and perionychium and have an understanding of treatment principles.

## DISTAL RADIUS (COLLES) FRACTURES

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Fractures of the distal radius are common. Fractures of the distal radius are often sustained after a fall on the out-stretched hand and are often associated with osteoporosis. Distal radius fractures are frequently accompanied by fractures of the ulnar styloid (with possible injury to the cartilage attached to it) or with injuries to the wrist ligaments. Associated injuries to the nearby cartilage and ligaments are also frequently seen. The eponym “Colles’ fracture” has been used to describe all distal radius fractures, though that name refers specifically to fractures that are angulated dorsally.



Figure 1: Colles fracture (From radiopaedia.org <http://bit.ly/1POPjGJ>)

## STRUCTURE AND FUNCTION

The distal radius forms the proximal side of the wrist joint. There, the radius articulates with the proximal row of carpal bones (allowing flexion and extension); it also articulates with the distal ulna (creating a joint for pronation and supination).

The distal ulna attaches to a meniscus-like structure, the triangular fibrocartilage discus, which can be torn with wrist fractures.

On the lateral side of the radius is a styloid process, onto which the brachioradialis inserts and from which the radial collateral ligament of the wrist originates.

At the distal metaphysis of the radius, the cortex of the bone is thinner than the bone proximal and distal, and the relative amount of cancellous bone increases. The distal metaphysis of the radius is therefore a relative weak point. This makes fractures more likely, especially in patients with decreased bone mineral density.

## PATIENT PRESENTATION

A patient with a fracture of the distal metaphysis of the radius typically describes falling on an outstretched hand or sustaining a direct blow to the wrist. He or she will complain of wrist pain and swelling.

The deformity that results from the Colles' fracture is described as a "dinner fork" deformity because of depression at the fracture site, dorsal angulation, and dorsal displacement of the distal radius.

History-taking should focus on the mechanism of injury and amount of energy involved. This information can alert the clinician to the presence of associated injuries (in the case of high energy mechanisms, such as motor vehicle accidents) or the presence of osteoporosis (in the case of low energy mechanisms, such as a fall from a standing height).

Physical examination (and imaging) should assess for the following:

- An open wound
- Acute carpal tunnel syndrome (characterized by weakness or loss of thumb or index finger flexion, tingling ["paresthesia"] or alterations of sensation in the median distribution)
- Vascular injury (which is rare)
- Injury to the elbow
- Injury to the ulnar side of the wrist
- Injury to the carpal bones

(The latter three should remind you of the rule *"Always assess the joints above and below the known injury."*)

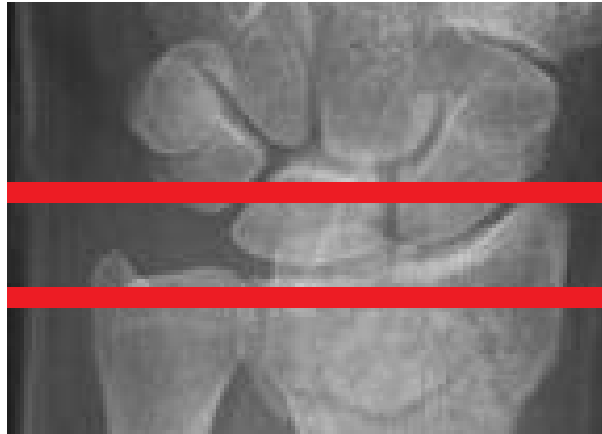
## OBJECTIVE EVIDENCE

PA, lateral, and oblique radiographs of the distal radius that include the carpal bones should be obtained. All three radiographs should be examined for a loss of normal anatomy, disruption of the articular surface, involvement of the distal radio-ulnar and radiocarpal joints, and evidence of comminution.

PA radiographs allow for evaluation of the integrity of the distal radio-ulnar joint, the radiocarpal joint, the proximal carpal row, and the radial and ulnar styloid processes.

Three measurements on PA films allow for characterization of the injury: radial height, radial inclination, and palmar (volar) tilt

Radial height is the distance between two parallel lines perpendicular to the long axis of the radius. The first line is drawn tangential to the distal tip of the radial styloid and the second tangential to the distal ulna, (Figure 2). Normal radial height is approximately 12 mm and is decreased in distal radius fractures.



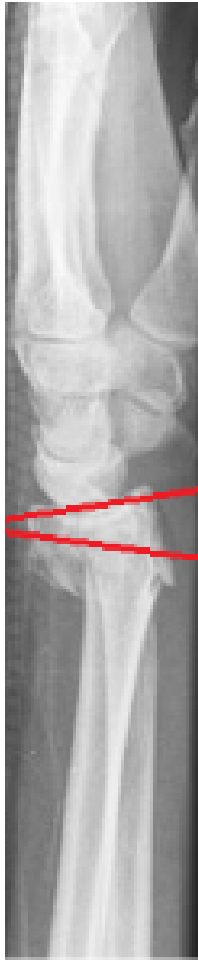
*Figure 2: Assessment of radial height (In this x-ray, the articular surfaces of the distal ulna and radius lie on the same line, so-called "neutral" ulnar variance. When ulna projects more distally to the radius, ulnar variance is said to be "positive".)*

Radial inclination is the angle between one line drawn perpendicular to the long axis of the radius and a second drawn from the distal tip of the radial styloid to the ulna, (Figure 3). Radial inclination ranges from approximately 20 to 25 degrees and is decreased in distal radius fractures.



*Figure 3: Assessment of radial inclination*

Palmar tilt is measured from lateral radiographs. Palmar tilt is the angle formed between a first line drawn perpendicular to the long axis of the radius and a second line connecting the palmar and dorsal edges of radius, (Figure 4). In the fracture shown above, the tilt is actually in the dorsal direction.



*Figure 4: Loss of palmar tilt*

## EPIDEMIOLOGY

Distal radius fractures are one of the most common fractures of the upper extremity, and therefore, represent a large proportion of fractures seen by Primary Care physicians and Emergency Medicine physicians. Distal radius fractures have a bimodal distribution: one peak in the 18-25 year old age group and another in the 65 and older age group. In younger people, high energy injuries are more common, such as those from motor vehicle collisions, motorcycle collisions, sports, or falls from an extended height. In older people, distal radius fractures are typically lower energy injuries, i.e. from a simple fall on outstretched hand from standing height or lower. These are categorized as “fragility fractures” meaning that weakness of the bone contributes to the cause.

## DIFFERENTIAL DIAGNOSIS

Distal radius fractures are detected on imaging. The classification of these fractures should consider whether they are intra- vs extra-articular; displaced vs non-displaced; comminuted or not; open or closed (i.e. skin broken or not); and whether there are associated injuries.

Concomitant injuries must be excluded, including shoulder, elbow and neurovascular injuries. Scaphoid fractures, scapholunate ligament injury, and fractures of the ulnar styloid process are injuries associated with distal radius fractures, given their anatomic proximity to the distal radius. Tenderness in the anatomical snuffbox is suggestive of scaphoid fracture.

## RED FLAGS

An isolated and non-complicated low energy fracture is itself a red flag: it may reflect previously undetected osteoporosis or other metabolic bone disease. Treatment of the fracture without consideration of the source of fragility is, to say the least, a missed opportunity.

Pain proximal to the wrist suggests the presence of an injury proximal to the wrist. Regardless, radiographs must include the elbow joint, even without proximal pain.

Acute carpal tunnel syndrome may develop following a fracture of the distal radius. Clinical findings specific to an acute carpal tunnel syndrome are weakness or loss of thumb or index finger flexion. A sensory finding in median nerve distribution is not specific to acute carpal tunnel syndrome as it may be caused by a median nerve contusion at the time of injury or reduction. Discerning between the two is a difficult one (theoretically, a contusion should have immediate sensory findings which do not get worse over time, whereas acute carpal tunnel syndrome is more likely to have progressive weakness).

## TREATMENT OPTIONS AND OUTCOMES

The goal of treatment is to create the biological and mechanical environment that promotes bone healing and restores maximal function. The goal of completely restoring the wrist to its pre-injury state is often beyond reach.

### *Non-Operative*

Many fractures of the distal radius can be treated without surgery. Nonsurgical treatment is usually indicated for non-displaced or minimally displaced fractures.

Acutely, non-displaced or minimally displaced fractures can be treated with a sugar tong splint. Upon examination 5 to 7 days after initial treatment, the splint can be exchanged for a short arm cast. Immobilization for 6-8 weeks usually suffices; though radiographic healing and clinical exam dictate the length of treatment. A new cast may be required mid-way through treatment if atrophy of the forearm renders the initial cast too loose.

Displaced extra-articular fractures of the distal radius should be reduced (that is, manually placed in a position of normal anatomy). If proper alignment can be attained and held, these originally-displaced fractures can usually be treated as if they were non-displaced from the start, though patients may need to remain in the splint for a longer duration as the swelling subsides.

### *Operative*

Surgical treatment of fractures of the distal radius is indicated when these fractures are largely displaced, reduced but unstable, irreducible, comminuted, or involving the distal radio-ulnar joint or radiocarpal joint.

Options for surgical treatment of distal radius fractures include percutaneous pinning, external fixation, or plating (Figure 5). The choice of surgical treatment varies considerably among patients, surgeons, institutions, and nations; no single surgical method has been uniformly proven to be superior to others.

If a displaced fracture of the distal radius is left untreated, the resulting deformity will leave the person with dorsal angulation, limited supination, and a weak grip. Malunion of the fracture site is associated with wrist pain and rupture of the extensor pollicis longus tendon.

Rupture of the extensor pollicis longus tendon can result from the tendon repeatedly moving across the fracture site (or surgical hardware).

Even well-treated injuries may resolve with objective deficits including loss of motion and radiographic evidence of arthrosis; these may be a consequence of the original injury and damage to the articular surface.



Elderly patients with low demands can typically return to baseline activities of daily living even with less than full motion or arthrosis.



Figure 5: Fixation of a Colles' fracture with a plate

## RISK FACTORS AND PREVENTION

In younger patients with high energy mechanisms of injury, distal radius fractures typically occur from motor vehicle accidents or sports trauma. Wrist guards and elbow pads can prevent upper extremity injuries in many sports, including inline skating and snowboarding.

Decreased bone mineral density (typically from osteoporosis) is a risk factor for distal radius fractures. Therefore, prevention of distal radius fractures in older people should focus on prevention of osteoporosis and risk factor reduction. Clinicians should encourage adequate calcium and vitamin D intake, initiation of weight-bearing exercise, cessation of smoking, and moderation of alcohol intake.

Evaluation for osteoporosis is part of the treatment in an older patient with a distal radius fracture and low energy mechanism of injury. The following lab tests should be obtained to evaluate for secondary causes of osteoporosis: complete blood count, complete metabolic panel (including calcium and phosphorus, parathyroid hormone levels, and testosterone for males), thyroid stimulating hormone level, 25-hydroxyvitamin D level, and alkaline phosphatase. Serum calcium, phosphate, and alkaline phosphatase are normal in a patient with osteoporosis, but alkaline phosphatase may be elevated for months after a fracture.

Additionally, a dual energy x-ray absorptiometry (DEXA) scan of the vertebral bodies and femur can be ordered to evaluate the patient's bone mineral density and screen for osteoporosis. Initiation of treatment may prevent a subsequent fragility fracture.

## MISCELLANY

Eponyms about fracture of the distal radius.

- A Colles' fracture, though at times used imprecisely to refer to all distal radius fractures, is the term for a transverse, dorsally displaced and angulated distal radius fracture about one inch proximal to the wrist. It is named for Abraham Colles, an Irish surgeon.
- A Smith's fracture is a fracture of the distal radius with volar (palmar) displacement of the hand and wrist distal to the fracture. Based on this configuration, it can be thought of as a "reverse Colles' fracture." It is named after Robert William Smith, who followed Abraham Colles' as professor of surgery at Trinity College in Dublin.
- A Barton's fracture is an intra-articular fracture of the distal radius with dislocation of the radiocarpal joint, either in the volar or dorsal direction. It was described by John Rhea Barton, a surgeon at Pennsylvania Hospital in Philadelphia.
- The Chauffeur's fracture is a fracture of the radial styloid process caused by a blow to the hand which compresses the carpus into the styloid process. This is a rare eponym that is not capitalized, as it refers to an occupation, not a person: years ago, when cars were started by a crank, back-firing could cause the starter crank to hit a chauffeur's palm and in turn break the styloid

## KEY TERMS

distal radius fracture, Colles' fracture, radial styloid, osteoporosis, carpal tunnel syndrome, fragility fracture, ulnar styloid.

## SKILLS

Assess radiographs and describe the pattern of injury. Recognize fractures associated with osteoporosis and initiate a workup for it. Be able to counsel a patient on lifestyle modifications that prevent osteoporosis. Be able to apply a sugar tong splint or short arm cast to patient with a fracture of the distal radius.

## DUPUYTREN'S DISEASE

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Dupuytren's Disease (or simply "Dupuytren's") is a progressive disorder characterized by fibrosis of the palmar fascia. Dupuytren's is associated with the formation of palpable nodules and cords in the palm and fingers. In its later stages Dupuytren's causes fixed digital flexion contractures. Although the disease process spares the tendons deep to the fascia because the contractures place the finger in flexion (as shown in Figure X) the tendons cannot function normally. Dupuytren's Disease usually involves the 4th finger but may include others (the 5th finger is next-most likely to be involved; the thumb is least likely). Dupuytren's is rarely a painful condition.

### STRUCTURE AND FUNCTION

Dupuytren's disease is due to abnormal thickening and contracture of the palmar fascia (also known as the palmar aponeurosis (Figure 1)).

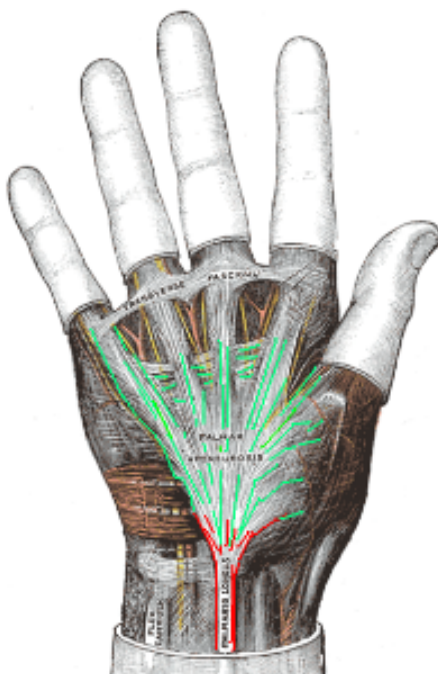


Figure 1: Anatomy of palmar fascia. (Modified from [wikimedia.org](http://bit.ly/1NMrgal) <http://bit.ly/1NMrgal>)

The palmar fascia is a fibrous structure that lies below the dermis and above the flexor tendons and muscles of the hand. It anchors the palmar skin and protects deeper neurovascular structures. The palmar fascia extends from the wrist across the palm (where it is continuous with the transverse carpal ligament); the tendon of the palmaris longus, if present, attaches to it. Distally, it courses into the fingers to become the digital fascia. The fibers of the fascia are architecturally complex, with transverse, longitudinal, or vertical orientations.

In Dupuytren's disease, these fibers contract to form nodules and then cords. The fascia becomes focally adherent to the overlying skin, causing pitting, and the cords contract, causing fixed flexion deformities of the digits.

The abnormal proliferation of myofibroblasts (specialized fibroblast-like cells that are vital for wound healing) is critical to the pathogenesis of Dupuytren's disease.

Myofibroblasts, unlike regular fibroblasts, are capable of contracting, and they are responsible for the fascial contraction that is seen in Dupuytren's Disease. The fascia in Dupuytren's is characterized by increased numbers of myofibroblasts and inflammatory cells and disorderly collagen deposition. There is increased expression of growth factors within the fascia and an increase in the ratio of type III to type I collagen.

The increased numbers of myofibroblasts, disorderly collagen deposition and increased expression of growth factors seen in Dupuytren's disease are found in penile fibromatosis (Peyronie's disease), suggesting a common underlying condition.

The underlying cause of Dupuytren's remains obscure, but ischemic, autoimmune, and inflammatory mechanisms have all been suggested. Certain environmental factors are thought to play a role, including smoking, alcohol use, and diabetes. It has been suggested but not proven that the use of vibrating machinery may lead to Dupuytren's.

In addition to environmental factors, there seems to be a genetic basis to at least some cases of Dupuytren's. This is supported by the large number of patients with a family history of the disorder and by the high prevalence of Dupuytren's in patients of Northern European descent. An autosomal dominant mode of inheritance with incomplete penetrance has been postulated, but a single gene responsible for the disease has yet to be identified.

## PATIENT PRESENTATION

The initial manifestations of Dupuytren's Disease include thickening of the skin of the hand, and loss of normal skin mobility.

The skin changes are followed by the development of palpable nodules and cords in the palm and fingers. The nodules are usually located near the distal palmar crease, near the base of the finger, or adjacent to the PIP joint.

As the disease progresses, often over a period of several years, flexion contractures of the MCP and PIP joints can develop. These contractures most commonly affect the ring and small fingers (Figure 2).



*Figure 2: A flexion contracture of the MCP of the 4th finger, with an obvious cord.*

The "tabletop test" can determine if the patient has a flexion contracture. As shown in Figure 3, the patient here is attempting to lay the hand palm side down, flat on a surface. This test is more sensitive than specific; that is, patients who *can* lay their hands down do *not* have a contracture, whereas an inability to do so may have other causes.



Figure 3: The “tabletop test”

In most cases, both hands are involved, although one is often more advanced than the other. Patients may also experience ectopic manifestations, such as fibrosis on the dorsum of the PIP joints (Garrod’s knuckle pads), plantar fibromatosis (Ledderhose disease), penile fibromatosis (Peyronie’s disease), and frozen shoulder.

## OBJECTIVE EVIDENCE

In general, Dupuytren’s disease is a clinical diagnosis. Hand x-rays will demonstrate flexion contractures and can be used to rule out other causes of deformity. Nodules and cords will not be seen, as they are radiolucent. While hand x-rays should be obtained at the time of initial evaluation, there is no role for routinely obtaining any other imaging or laboratory studies.

## EPIDEMIOLOGY

Typical patients are of Northern European descent, with fair skin and blonde hair. Dark skinned people are not usually affected. It has been suggested that the disease is of Viking origin, although this has not been substantiated. Onset is usually in middle to late life. Dupuytren’s disease is much more common in men than women; and when it is found in women, the onset is later and the disease is less severe.

The prevalence of Dupuytren’s disease varies dramatically by region. It is highest in northern Scotland, Ireland, Norway, and Australia, where as many as 40% of elderly men may be affected. In the United States the prevalence is less than 5%.

## DIFFERENTIAL DIAGNOSIS

While Dupuytren’s disease causes flexion contractures of the digits, a number of other disorders can present in a similar fashion, including stenosing tenosynovitis (or “trigger finger”), ulnar nerve palsy, and camptodactyly.

The thickened skin, nodules, and cords may be confused with scars, calluses, tophi, ganglion cysts, prolapsed flexor tendons (as can be seen in rheumatoid arthritis), or soft tissue tumors such as giant cell tumors or even (rarely) epithelioid sarcomas.

Plantar fibromatosis (Ledderhose disease) and penile fibromatosis (Peyronie’s disease) are sometimes seen in conjunction with Dupuytren’s disease, making thorough history-taking important.

## RED FLAGS

Although the signs of early Dupuytren's can be subtle, pitting of the palmar skin can be one of the first findings and is a relatively specific marker of the disease.

Males with early onset, a family history of Dupuytren's, ectopic involvement, and bilateral disease have the worst prognosis.

Intervention should be considered when there is a MCP joint contracture of thirty degrees or more or for any degree of PIP joint contracture.

## TREATMENT OPTIONS AND OUTCOMES

Patients with minimal impairment can be educated regarding the diagnosis and observed over time. When symptoms become more severe, intervention is warranted.

Indications for intervention and possible surgical treatment include metacarpophalangeal flexion contractures of more than thirty degrees or any proximal interphalangeal flexion contracture. Once this degree of contracture is present, treatment involves removing or breaking apart the cords that pull the fingers into flexion.

One method for removing or breaking apart the cords that pull the fingers into flexion is to cut them with a scalpel or percutaneously with a needle. This latter technique is called "needle aponeurotomy" or "needling." The scalpel or needle disrupts or "breaks" the cord of tissue that is contracting the finger. The advantages of this technique are that there is a small or no incision, it can be performed on multiple fingers at the same setting, and usually requires little therapy after the procedure. The primary risk is accidental injury to nerves or tendons.

Another treatment option for breaking apart the cords is the injection of collagenase clostridium histolyticum, an enzyme that weakens the Dupuytren's cord (Figure 4). This injection is performed in the office. On the day following the injection, the physician manipulates the contracted finger in an attempt to break the cord and straighten the finger. Currently, the collagenase injection can be used on only one joint (in one finger) at a time and additional injections must be spaced at least one month apart. Otherwise, the risks and benefits are similar to those for needling.



Figure 4: Injection for Dupuytren's Disease (Source: Flickr <http://bit.ly/20jDYd5>)

Limited fasciectomy (fascial excision) may be performed for more advanced cases. The principle advantage to fasciectomy is a more complete and reliable joint release than that with the needle or enzyme techniques. Disadvantages include the need for hand therapy postoperatively and longer recovery time.



Figure 5: Post-operative appearance of Dupuytren's release. (source: Flickr <http://bit.ly/1UDrKoO>)

For the most severe and for recurrent cases, the overlying skin must be excised as well and the area skin grafted (a so-called dermo-fasciectomy). Patients are often especially stiff after dermo-fasciectomy. To assist in regaining range of motion, patients are prescribed hand therapy and often thermoplast splints. For more severe stiffness, temporary placement of skeletal traction devices, surgical release of joint contractures, or even joint fusions can be utilized.

Unfortunately, recurrence rates after surgery are high, ranging from 2% to 60% (average 33%). Recurrence may be due to the formation of new nodules at the surgical site or extension of those outside that area.

Potential complications of surgery include skin necrosis, infection, nerve or vascular injury, reflex sympathetic dystrophy, loss of the involved digit, and stiffness.

The outcomes of collagenase injections have been less well-studied. They seem to be effective in the short term and appear relatively safe, although some cases of iatrogenic tendon ruptures have been observed and long-term follow-up is not yet available.

## RISK FACTORS AND PREVENTION

Risk factors include a family history, male sex, smoking, alcohol intake, and diabetes. Epilepsy, anticonvulsant use, hand trauma, and the use of vibrating machinery may also put patients at higher risk, although this is controversial.

## MISCELLANY

Among bagpipe players, Dupuytren's Disease is known as the "Curse of the MacCrimmons," after a family of preeminent bagpipe players in 16th century Scotland. When affected by the Curse, a player's small finger becomes bent, making bagpipe playing impossible.

Famous sufferers of Dupuytren's disease include Ronald Reagan, Bob Dole, and Samuel Beckett.

According to Stigler's (tongue in cheek) Law of Eponymy, "No scientific discovery is named after its original discoverer" (a phenomenon first described by Merton, Stigler points out). Dupuytren's Disease is no exception: Baron Guillaume Dupuytren's 1831 lecture associated his name with a condition Henry Cline characterized in 1808.

## KEY TERMS

Dupuytren's disease, palmar fascia, Dupuytren's diathesis

## **SKILLS**

Recognize the presenting features of Dupuytren's disease; Understand the underlying pathophysiology; Identify risk factors for the development of Dupuytren's disease; Describe the various treatment options and prognosis.



## FINGER AND HAND INFECTIONS

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The hand is susceptible to infection by virtue of its intimate contact with the outside world, its great surface area and its propensity for injury. That is, the hand is exposed frequently to infectious organisms, and these organisms are frequently given a point of entry.

The specialized anatomy of the hand, particularly the tendon sheaths and deep fascial spaces, create distinct pathways for infection to spread. In addition, even fully cleared infections of the hand can result in significant morbidity, including stiffness and weakness. For these reasons, early and aggressive treatment of hand infections is imperative.

In this section, specific hand infections will be considered:

- **paronychia:** infection of the folds of skin surrounding a fingernail
- **felon:** a purulent collection on the palmar surface of the distal phalanx
- **flexor tenosynovitis:** purulent material resides within the flexor tendon sheath.
- **septic arthritis:** infection in the joint space, often related to bite wounds

### *Paronychia*

The paronychium is a small band of epithelium that covers the medial and lateral borders of the nail. The eponychium is a small band of epithelium that covers the proximal aspect of the nail.

A paronychia (Figure 1) is an infection of the paronychium or eponychium. It is caused by minor trauma such as nail biting, aggressive manicuring, hangnail picking or applying artificial nails. Immunodeficiency, poor glycemic control, and occupations involving repeated hand exposure to water (e.g. dishwasher) are risk factors for the development of paronychia.

Tenderness and erythema of the nail fold at the site of infection will become evident within a few days of the inciting trauma. Progression to abscess formation is common.

Turkman et al described the “digital pressure test for paronychia”: A paronychia will appear as a blanched area when light pressure is applied to the volar aspect of the affected digit.



Figure 1: Paronychia (From Wikipedia. <http://bit.ly/203fe3w>)

If left untreated, the paronychia can spread along the nail fold from one side of the finger to the other, or to beneath the nail plate.

Treatment for early cases includes warm water soaks and antibiotics. However, once a purulent collection has formed, treatment requires opening the junction of the paronychial fold and the nail plate (Figure 2). This is normally done with the bevel of an 18 gauge needle.



Figure 2: Drainage of Paronychia. The bevel of an 18 gauge needle is passed between the nail plate below and the nail fold above to allow for drainage of the pus.

Acute paronychia are usually caused by *Staphylococcus aureus* and are treated with a first-generation cephalosporin or anti-staphylococcal penicillin. Broader coverage is indicated if other pathogens are suspected. Chronic paronychia may be caused by *Candida albicans* or by exposure to irritants and allergens.

Paronychia may be prevented by avoiding behaviors such as nail biting, finger sucking, and cuticle trimming. Patients with chronic paronychia should be advised to keep their nails short and to use gloves when exposed to known irritants.

## **Felon**

A felon is an abscess on the palmar surface of the fingertip. Bacteria are normally introduced via minimal penetrating trauma, such as a splinter.

The palmar aspect of the fingertip contains many osteocutaneous ligaments that connect the palmar skin of the fingertip to the distal phalanx. These ligaments prevent excessive mobility of the skin during pinch; they also maintain position of the cutaneous sensory endings and receptors to allow for identification of objects during grasp. The organization of these osteocutaneous ligaments form a relatively non-compliant compartment in the distal phalanx; thus, rather than expanding when pus is introduced, the compartment will simply increase in pressure.

Elevated compartment pressure results in significant pain relative to the (small) amount of pus. In addition, the gradient between capillary pressure and tissue pressure is decreased; the resulting decrease in perfusion can lead to tissue necrosis. Furthermore, because the osteocutaneous ligaments attach to the distal phalanx itself, osteomyelitis (infection of the bone) can occur.

Treatment involves surgical drainage and antibiotics. Incision and drainage is performed at the most fluctuant point. The incision should not cross the distal interphalangeal joint flexion crease (to prevent formation of a flexion contracture from scar formation) or penetrate too deeply (to prevent spread of infection from violating the flexor tendon sheath). Potential complications of excessive dissection to drain a felon include an anesthetic fingertip or unstable finger pad.

Antibiotic treatment should cover staphylococcal and streptococcal organisms. X-rays may be helpful to ensure that there is no retained foreign body.

## **Flexor tenosynovitis**

In the fingers, a series of pulleys hold the tendons in close apposition to the bone, preventing bowstringing during flexion. There are a total of 8 pulleys overlying the finger flexor tendons and 3 pulleys overlying the thumb flexor tendon; these pulleys together are called the flexor tendon sheath.

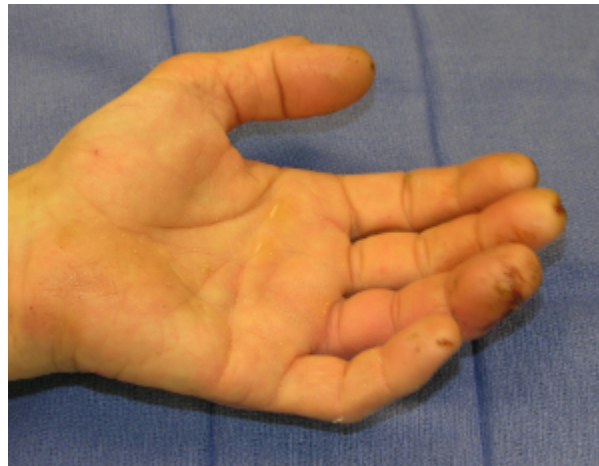
In flexor tenosynovitis (Figure 3), the infection is within the flexor tendon sheath. This infection is particularly harmful because bacterial exotoxins can destroy the paratenon (fatty tissue within the tendon sheath) and in turn damage the gliding surface of the tendon. In addition, inflammation can lead to adhesions and scarring, and infection can lead to overt necrosis of the tendon or the sheath.

Although patients may not recall a specific history of trauma, flexor tenosynovitis is usually the product of penetrating trauma. Flexor tenosynovitis may be caused by inoculation and introduction of native skin flora (e.g., *Staphylococcus* and *Streptococcus*) or by more unusual organisms (e.g., *Pasteurella* and *Eikenella*) when there is a bite wound.

Flexor tenosynovitis can also have noninfectious causes such as chronic inflammation from diabetes mellitus, rheumatoid arthritis or other rheumatic conditions (e.g., psoriatic arthritis, systemic lupus erythematosus, and sarcoidosis).

Kanavel described four classic signs of flexor tenosynovitis, as follows:

1. Flexed posture of the digit.
2. Fusiform (sausage-shaped, or tapering) swelling.
3. Tenderness to palpation over the flexor tendon sheath.
4. Pain over the flexor tendon sheath with passive extension of the finger



*Figure 3: Flexor tenosynovitis. This patient's fourth digit exhibits erythema, fusiform swelling, and mild flexion compared to the adjacent digits.*

Anatomic relationships of flexor sheaths to deep fascial spaces should be kept in mind. Contiguous spread can result in a “horseshoe abscess”: from small finger flexor sheath to the thumb flexor sheath via connection between the radial and ulnar bursae.

If the diagnosis of flexor tenosynovitis is not clear, the patient may be admitted to the hospital for antibiotics, elevation of the affected hand, and serial examination. Non-operative treatment should be reserved for normal hosts. In patients with diabetes or any disease that may compromise the immune system, early surgical drainage is indicated even for suspected cases.

If the diagnosis of flexor tenosynovitis is established definitively, or if a suspected case in a normal host does not respond to antibiotics, surgical drainage is indicated (Figure 4). During this surgery, it is important to open the flexor sheath proximally and distally to adequately flush out the infection with saline irrigation. The distal incision is made very close to the digital nerve and artery as well as the underlying distal interphalangeal joint; it is important to avoid damage to these structures during surgery. Some surgeons will leave a small indwelling catheter in the flexor sheath to allow for continuous irrigation after surgery, but there is no conclusive evidence that this ultimately improves results.



*Figure 4: Drainage of flexor tenosynovitis. Proximal and distal incisions have been made, allowing adequate drainage of the flexor tendon sheath.*

Post-operative active and passive ROM exercises are recommended. Intravenous antibiotics should continue for an additional two or three days. (The duration of IV antibiotic administration as well as the need for oral antibiotics thereafter is determined by the intraoperative cultures and clinical response.)

Post-operative adhesions damage gliding surfaces and decrease active range of motion, and thus require tenolysis. Soft tissue necrosis and flexor tendon rupture are other relatively common complications.

### Joint infection

The metacarpophalangeal and interphalangeal joints are closed, relatively avascular spaces. Infection can reach the joint space via direct penetration or hematogenous spread.

Small (and ring) finger metacarpophalangeal joint infections in particular may result from a “fight bite,” where the patient strikes and an opponent in the mouth with a closed fist and the opponent’s tooth penetrates the joint and seeds it with oral flora. As with flexor tenosynovitis, a major risk of joint space infection is destruction of the gliding surface by bacterial exotoxins, which can compromise recovery of motion after the infection resolves.

A fight bite is at particularly high risk for complications, for the following reasons:

1. the puncher may underestimate the severity of the wound
2. the puncher may have been intoxicated (and sufficiently “medicated” to not feel pain)
3. the puncher may attribute initial symptoms to bone pain from punch and not present for care until cellulitis is rampant
4. the initial examiner may underestimate the severity of the wound, as it is usually small (the size of an incisor tooth or smaller, e.g. 3mm) with clean edges
5. the human mouth has a high concentration of nearly 200 species of bacteria, many “unusual” anaerobes
6. motion of the MCP joint to “shake off the pain” may drive saliva deeper into the tissue
7. the extensor tendon and joint capsule are fairly superficial and may be violated with seemingly shallow wounds
8. the extensor tendon and joint capsule are fairly avascular and thus unable to fight infection

Fight bites (Figure 5) should be meticulously irrigated, preferably with a formal debridement by a hand surgeon in the operating room. The laceration must *not* be closed in the ED.



*Figure 5: Fight bite. A punch to the tooth may inadvertently lacerate the skin over the MCP joint and introduce oral flora into the joint.*

Diagnosis of an established joint infection is often made by clinical examination. Patients will have swelling and erythema centered on the affected joint. Motion or axial loading of the joint will increase pain. Assessment of joint fluid for cell count, gram stain, and crystals (acute crystalline arthropathy such as gout can mimic a joint infection) can aid in the diagnosis, but it is often quite difficult to pass a needle into the narrow joint space and obtain an adequate sample. Serum markers of inflammation (such as white blood cell count, erythrocyte sedimentation rate, and C – reactive protein) are not typically elevated with an infection of a small joint of the hand. X-rays should be obtained to ensure that there is no fracture or retained tooth fragment.

Treatment consists of incision and drainage of the joint space. For the metacarpophalangeal joints of the fingers, the approach is normally dorsal through the long extensor tendon. In “fight bite” situations, there may be an indentation of the head of the metacarpal where it struck the tooth. For the interphalangeal joint, the approach is normally dorsolateral between the extensor mechanism dorsally and the collateral ligament laterally. Arthroscopic approaches have been described for the wrist and even the metacarpophalangeal joint, but an open approach is more commonly used.

## GENERAL PRINCIPLES

- Open wounds must be irrigated to remove debris
- Closed abscesses must be incised and drained
- Devitalized tissue should be debrided
- Penetrating wounds require consideration of tetanus status
- Splinting the hand may enhance healing
- Patients with diabetes mellitus have more gram-negative infections and require broader antibiotic coverage
- Patients in an immunocompromised state may develop a hand infection from hematogenous spread from another site
- Unusual exposures lead to unusual bacteria: e.g. tropical fish aquarium workers, butchers, farmers.

## IMAGING

Patients suspected of having a hand infection will often undergo plain x-rays. The bony structures will typically appear normal except in very advanced infections involving the bone. Ultrasound can show loculated fluid collections, but is heavily dependent on the skill of the person performing the study. Magnetic resonance imaging, with or without gadolinium contrast, may show occult deep space infections if the clinical picture is not clear. Use of MRI is limited by cost as well as availability depending on when and where the patient is being evaluated.

For most cases, the diagnosis of infection is made by history and physical exam. X-rays are a rapid and cost effective way to identify bony changes and radiopaque foreign bodies. More complex imaging studies should be reserved for situations where the diagnosis remains unclear despite adequate examination and initial treatment, or if the patient does not respond to appropriate management.

## FINGER SPRAINS AND DISLOCATIONS

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Injuries to the soft tissues surrounding and supporting the metacarpophalangeal (MCP) and interphalangeal (IP) joints of the fingers and thumb (Figure 1) are common. Injuries range from minor sprains that resolve with minimal treatment (such as a partial ligamentous tear treated with buddy taping or splinting) to more severe injuries that require surgical intervention, such as complete tearing of the ligaments and dislocation of the joint. (Soft tissue injury can also involve the tendons and nail, both discussed elsewhere.)



*Figure 1: Examination under anesthesia of a thumb ulnar collateral ligament rupture (courtesy of Gene Deune, MD)*

### STRUCTURE AND FUNCTION

The second through fifth digits of the hand each have proximal, middle, and distal phalanges and therefore have three joints: the distal interphalangeal (DIP), the proximal interphalangeal (PIP), and the metacarpophalangeal (MCP).

Unlike the other digits, the thumb lacks a middle phalanx, and consequently has only two joints: the interphalangeal (IP) and the metacarpophalangeal (MCP).

The proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints are hinge, or “ginglymus,” joints that only allow flexion and extension; a normal arc of motion for each joint is approximately 100 degrees. In the IP joints, the head of the more proximal phalanx articulates with the base of the more distal phalanx (Figure 2).

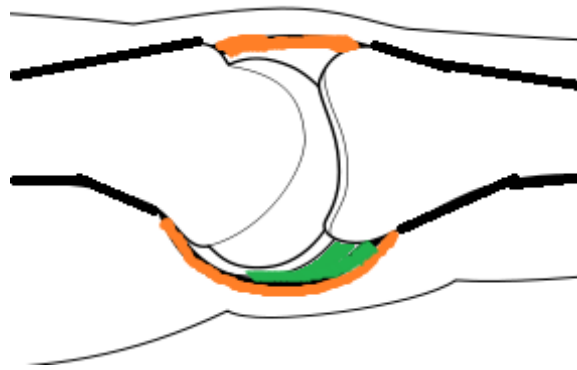


Figure 2: Schematic profile of the anatomy of the IP joint. The volar plate is in green; the capsule is orange. (modified from Wikipedia. <http://bit.ly/1STK5QG>)

The PIP joint is composed of the distal aspect of the proximal phalanx, which consists of two condyles, and the proximal aspect of the middle phalanx, which has two concave spaces that articulate with the condyles of the proximal phalanx. Along each side of the joint are the radial and ulnar collateral ligaments, which are primary restraints to radial or ulnar joint deviation.

On the palmar side, there is a thick fibrocartilagenous structure called the volar plate, that spans the joint and resists joint hyperextension.

The anatomy of the DIP joint is similar to that of the PIP joint, with the exception of one feature: since the flexor and extensor tendons insert on the distal phalanx, the DIP joint is more stable and dislocation of the DIP joint is a less common injury than dislocation of the PIP joint.

In the metacarpophalangeal joint, the metacarpal head articulates with the concave base of the proximal phalanx. Similar to the PIP and DIP joints, the MCP joint is supported by its collateral ligaments and volar plate, as well as the flexor and extensor tendons. Dorsally, the MCP joint is further supported by the sagittal bands, which fan out from the volar plate to attach on the extensor tendon, helping to stabilize the joint and extensor mechanism. The MCP joints permit both flexion-extension and adduction-abduction movements.

## PATIENT PRESENTATION

Patients with finger sprains and dislocations typically report a history of “jamming” the finger, with pain and swelling in the involved digit.

Deformity may be visible if a dislocation has occurred (though a lack of deformity does not exclude that diagnosis).

Inspection of the digit may also reveal ecchymosis, abrasions, or lacerations.

Tenderness to palpation should be assessed: radial or ulnar joint line tenderness implies collateral ligament injury or fracture, whereas volar tenderness implicates injury to the volar plate. Range of motion testing can be difficult given patient pain and swelling but it is important to assess in order to determine joint stability. The joint should be stressed in radial and ulnar deviation to test collateral ligament integrity. The joint should be stressed in full extension to test bony stability, as well as 30° of flexion to test collateral ligament stability. (That is, when the finger is held at full extension, a ligament injury may not be apparent on stress testing, as bony congruity may produce a sense of stability.)

A digital nerve block to numb the finger may be required to assess active range of motion. (It is commonly recommended that this block should not contain epinephrine as the drug may cause constriction of the blood vessels and finger ischemia. Nonetheless, a recent review of more than 1000 cases (PMID: 20697319) found no complications associated with the use of epinephrine in digital blocks.)

Near full arc of motion without visible hinging indicates a stable joint.



In dislocation injuries, range of motion may be blocked by interposed soft tissue; the examiner should avoid further injury and not force motion during the examination.

To rule out tendon injuries, the integrity of the flexor and extensor tendons should be tested as well. (Please see the chapters pertaining to those injuries for detail).

The neurovascular status of the finger's digital nerves and arteries should be assessed, as dislocations can damage the nerves or vessels. Stretching of the nerves can lead to neuropraxia, that is, transient impaired sensory function. Two-point discrimination is more sensitive than light touch sensation when assessing digital nerve function. Discrimination greater than 6mm is indicative of nerve injury. If arterial injury is suspected, a Doppler test may be performed on both the ulnar and radial sides of the digit to assess arterial integrity.

#### ***Collateral ligament injury***

Radial and ulnar collateral ligament injuries occur due to forced lateral deviation of the finger. Injuries are graded by the typical 3-point system, in which a grade 3 sprain is a complete tear, characterized by the absence of a firm end point on exam; a grade 2 sprain has laxity but a good end point; and a grade 1 shows no laxity, only pain, with the application of stress. (The quality of the "end point" is a subjective assessment, but one can think of the restraint produced by a chain link as a "firm end point" –it snaps to attention when its limit is reached.)

Ulnar collateral ligament injuries of the thumb (Figure 3) are particularly common and are known as skier's or gamekeeper's thumb. These injuries occur due to abrupt forced radial deviation of the thumb. Complete tears can include a bony fragment of the base of the proximal phalanx and this bony fragment can become trapped above the thumb adductor tendon (aponeurosis), a condition known as a Stener lesion. (see Miscellany, below.)



Figure 3: MRI of thumb ulnar collateral ligament injury (Modified from Radiopaedia.org <http://bit.ly/1nIDiWl>)

#### ***Volar plate injury***

Volar plate sprain (Figure 4) or partial tear occurs after a hyperextension injury of the finger. These injuries can lead to joint subluxation without frank dislocation or true dislocation of the joint. Patients with injuries to the

volar plate will be tender to palpation along the anterior aspect of the injured joint and may hold the finger in a hyperextended or flexed position at the level of the injured joint. Range of motion should be tested with particular attention paid to any joint subluxation during the arc of motion.



*Figure 4: Volar plate avulsion fracture of the middle phalanx  
(From Radiopaedia  
<http://bit.ly/1nA9GB7>)*

#### ***Interphalangeal joint dislocation***

Dislocations (Figure 5) are characterized by the position of the distal skeletal component relative to its proximal counterpart. Therefore, the three types of dislocation are dorsal, palmar and lateral.



*Figure 5: Fifth proximal phalangeal joint dislocation  
(modified from Radiopaedia.  
<http://bit.ly/20jFJqS>)*

Lateral dislocation involves the rupture of one collateral ligament and the palmar plate. In this type of dislocation, rotation of the middle phalanx may be observed. (When only one collateral ligament is intact, the phalanx is free to pivot.)

The majority of thumb and phalangeal dislocations are dorsal, and the finger or thumb will appear hyperextended at the affected joint. Puckering of skin around the joint suggests that soft tissue may be interposed within the joint. A dorsal dislocation of the PIP joint can tear the volar plate or cause an avulsion fracture of the middle phalanx.

The affected finger should be taken through both an active and (gentle) passive arc of motion, keeping in mind that motion may be significantly limited due to pain and swelling or due to a bony block.

If a dislocation is strongly suspected based upon the clinical presentation, the joint should first be reduced and radiographs documenting a reduced joint should be obtained prior to assessing range of motion. Once the joint is reduced, a smooth, near full arc of motion will indicate a stable joint. A repeat neurovascular examination should always be performed after successful joint reduction.

Volar dislocations are rare injuries; they usually cannot be reduced in a closed fashion due to interposition of soft tissue structures including the collateral ligaments or extensor mechanism. The proximal phalanx can also be “buttonholed” through the space between the central slip and the lateral band blocking closed reduction. These cases often necessitate open reduction, which also allows assessment and potential repair of a disrupted extensor mechanism.

## **OBJECTIVE EVIDENCE**

Radiographs, including posteroanterior (PA), perfect lateral and oblique views of the hand should be taken for all suspected finger injuries. True lateral and anteroposterior radiographs are mandatory for initial diagnosis of the injury, and oblique radiographs can be used optionally for further clarification.

Note that injuries to soft tissue around the digits often occur in the sagittal plane and are not seen on the posteroanterior view.

Even in cases of suspected isolated soft tissue injuries, radiographs should be obtained and evaluated to ensure that joint congruity is present and that there are no accompanying avulsion fractures. Radiographs also provide additional data regarding joint stability: dislocations accompanied by large fracture fragments are very unlikely to be stable even after reduction maneuvers are performed.

## **EPIDEMIOLOGY**

Ligamentous injuries to the hand most commonly involve the PIP joint. The mechanism of injury is typically hyperextension of the fingers, often caused by a fall on an outstretched hand or jamming injuries during contact sports. Injuries can range in severity from partial to complete tears, with associated dislocation of the involved joint.

## **DIFFERENTIAL DIAGNOSIS**

Finger injuries, even minor sprains, can cause significant pain and/or swelling, both of which restrict motion on physical examination and make it difficult to obtain a clinical diagnosis.

The differential diagnosis of a painful or swollen finger includes:

- Contusion
- Extensor tendon injury
- Flexor tendon injury
- Fracture or fracture/dislocation of the phalanx (detected on films)

- Volar plate injury (especially with hyper-extension)
- Interphalangeal joint dislocation
- Infection
- Tumor (rare but possible)

## RED FLAGS

Volar dislocations are less common injuries, but are usually more difficult to reduce. If gentle closed reduction does not work, open reduction in the operating room (by a hand specialist) may be necessary, as soft tissue structures may be interposed into the joint.

Rotational deformity, especially after reduction, suggests soft-tissue interposition, with open reduction in the operating room necessary here too.

Inadequate radiographs, e.g. only having images in a single plane, may lead to a missed diagnosis.

Changes in sensation on the ulnar and radial sides of the finger suggest injuries to the digital nerves.

## TREATMENT OPTIONS AND OUTCOMES

### *Collateral ligament injury*

- Grade 1 sprains should be buddy-taped full time for 1-2 weeks, and for an additional 2-4 weeks during physical activity.
- Grade 2 sprains should be splinted in 30 degrees of flexion for 1-2 weeks followed by buddy taping during physical activities for 4-6 weeks. Prolonged splinting in flexion should be avoided, as this can cause contracture of the volar plate.
- Grade 3 sprains should be treated similarly to grade 2 injuries, but surgery may be considered depending on the severity of the sprain. If the injury is recognized acutely, primary repair of the ligament may be attempted. Prolonged immobilization should be avoided, as the collateral ligaments will scar down after injury, causing joint stiffness and pain.
- Grade 1 or 2 thumb collateral ligament injuries should be treated in a thumb spica cast or splint for 4-6 weeks prior to beginning gradual range of motion exercises. Grade 3 sprains should be referred to an orthopaedic or hand surgeon immediately for surgical treatment.

### *Phalangeal Dislocations*

If the joint is stable after reduction of a dorsal dislocation, active range of motion can be initiated immediately to avoid joint stiffness, and buddy-taping or splinting should be instituted for physical activities for the next 3-4 weeks. A dorsal blocking splint should be considered the first 1-2 weeks to prevent PIP hyperextension and subsequent swan neck deformity.

Fingers should never be immobilized for greater than 30 days, as this may lead to a joint contracture and permanent loss of motion.

If the joint is unstable after reduction of a dorsal dislocation or cannot be reduced, the patient should be referred to an orthopaedic or hand surgeon immediately for surgical evaluation.

The surgical treatment chosen depends on several factors, including the joint affected, the presence of a concomitant fracture, and the amount of time since the injury occurred.

Volar dislocations, though less common, require prolonged immobilization or surgical intervention because concomitant injury to the extensor mechanism is common. Following closed reduction, these injuries require 6 weeks of splinting to permit adequate healing

Most finger sprains and dislocations do well, as long as a stable, congruent joint is maintained.

One of the most common complications is joint stiffness and flexion contracture, which can be avoided by encouraging early range of motion and guided physical/occupational therapy if necessary. Residual swelling can continue for up to 6 months after the injury, and some patients will note a permanent increase in finger size, potentially necessitating them to have their rings resized.

## **RISK FACTORS AND PREVENTION**

Many finger sprains and dislocations are a result of accidental trauma. However, athletes, particularly those playing ball-handling sports, such as basketball and football, may be at a higher risk for injury and should always wear appropriate sport-specific padding. Some athletes (e.g. football linemen) tape their fingers prophylactically, in anticipation of injury.

## **MISCELLANY**

Chronic instability of the thumb MCP is called a “gamekeeper’s thumb.” The condition derives its name from Scottish gamekeeper’s method of killing rabbits by breaking the animals’ necks barehanded; this method not only puts a lot of stress on the rabbit’s neck, but also on the gamekeeper’s thumb. Repetitive neck wringing causes wear-and-tear damage to, and ultimately failure of, the thumb MCP ulnar collateral ligament. This ligament failure produces instability.

The term “skier’s thumb” has been adopted to represent a more acute injury to the thumb ulnar collateral ligament, which occurs when skiers fall while holding their poles leading to hyperabduction of the thumb.

Consideration of the issue of cosmesis after finger sprains may be helpful. At the least, patients should be forewarned about the potential for cosmetic deformity after finger dislocation. (An iron worker may be proud of his work-deformed hands, but not all people think like that.)

## **KEY TERMS**

Collateral ligament injury, Finger dislocation, Finger Sprain, Finger splinting, PIP dislocation

## **SKILLS**

Perform a thorough physical examination of the hand, including evaluation of active and passive joint range of motion and joint stability, and a sensory and vascular exam of the digits.

## FLEXOR TENDON INJURY

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Injuries to the flexor tendons of the hand can be particularly challenging. Without finger flexion, patients will have difficulties with many tasks of daily living. The challenge to the surgeon is to re-attach the muscle to the bone in a way that glides smoothly within the pulley system of the hand. Flexor tendon injuries to the index and long finger tend to impede tasks that require fine motor skills; injuries to the flexor tendons of the ring or small fingers usually have a greater impact on grip strength.

### STRUCTURE AND FUNCTION

Finger flexion occurs at three joints: the distal interphalangeal joint (DIP); the proximal interphalangeal joint (PIP); and the metacarpophalangeal joint (MCP).

Flexion at each of these joints is powered by a distinct muscle group:

- the flexor digitorum profundus flexes the distal interphalangeal joint;
- the flexor digitorum superficialis flexes the proximal interphalangeal joint; and
- the intrinsics (lumbricals and interossei) flex the metacarpophalangeal joints

The flexor digitorum profundus and the flexor digitorum superficialis attach directly to the distal and middle phalanx respectively (Figure 1). These tendons are held close to the bone by a system of annular (ring-like) and cruciate (cross-forming) ligaments or pulleys.

The lumbrical muscles do not attach to the proximal phalanx directly, but rather connect the tendons of flexor digitorum profundus (below, i.e. volar) to the extensor tendons (above, i.e. dorsal) and produce flexion by pulling on the extensor tendons as they pass over the proximal phalanx.

The flexor digitorum superficialis [FDS] and flexor digitorum profundus [FDP] originate proximally in the forearm, at the medial epicondyle of the elbow. They, along with flexor pollicis longus (FPL) and the median nerve, travel through the carpal tunnel at the wrist and enter the palmar surface of the hand. From there, FDS and FDP send individual tendons to the index, long, ring, and small fingers.

Each pair of FDS and FDP tendons runs together through a tendon sheath into the fibro-osseous canal of each digit. This canal is formed by the metacarpals/phalanges, and by the pulley system and tendon sheath. The pulley system keeps the flexor tendons snug to the bone, preventing “bowstringing” during active flexion and converting a linear force into rotation and torque at the finger joints. The tendon sheath provides both lubrication as well as an important source of nutrition to the largely avascular tendons.

The superficialis lies, as its name implies, superficial to profundus (i.e. “deep”) throughout its course until it reaches the first annular pulley (“A1”) in the vicinity of the metacarpal head. At that point, the profundus emerges through a split in the superficialis (known as Camper’s chiasm); this allows the profundus to continue to its attachment on the volar surface of the distal phalanx where it flexes the distal interphalangeal (DIP) joint. The superficialis attaches onto the base of the middle phalanx, and thus flexes the proximal interphalangeal (PIP) joint.

Note that while the profundus distinctly flexes the distal interphalangeal (DIP) joint, it also indirectly flexes the proximal interphalangeal (PIP) joint as well. (This has important implications for physical examination, as the mere ability to flex the proximal interphalangeal (PIP) joint does *not* prove that the superficialis is intact; rather, such motion may reflect the indirect action of the profundus.)



Figure 1: Flexor tendons inserting on the middle and distal phalanges. The FDS is in red, the FDP in yellow.

Innervation to FDS and the radial-sided two FDP tendons (e.g., those to the index and long fingers) is provided by the median nerve, while innervation of the ulnar two FDP tendons (ring and small finger) is provided by the ulnar nerve.

Each digit also has a neurovascular bundle running along both its radial and ulnar borders. While these are not involved in finger flexion, per se, because of their proximity to the flexor tendons, they may also be injured when the flexor tendons are lacerated.

Injuries to the flexor tendons can be classified by the general location of the injury. Five zones have thus been identified. The first zone is distal to the FDS insertion. This is typically an avulsion of the profundus, known as a “Jersey finger,” as it may occur when a rugby player grabs the jersey of an opponent and pulls too hard. Zone II spans the PIP joint to the distal palmar crease near the metacarpal head. In Zone II the FDP and FDS travel in the same tendon sheath and are typically injured together. Zone III is within the palm itself and is often associated with neurovascular injury. Zone IV is the carpal tunnel itself and Zone V is any area proximal to the wrist.

## PATIENT PRESENTATION

Patients will most frequently present with a laceration to the palmar aspect of the hand and an inability to flex one or more digits. It is important to ascertain the mechanism of injury, as it may impact treatment. For example, animal or human bites should prompt antibiotics, and any penetrating injury to the hand should prompt an inquiry of whether the patient’s tetanus is up to date.

Identification of which tendons are injured requires an accurate and complete physical exam, documenting the presence or absence of FDS and FDP function in each digit.

To evaluate the superficialis (Figure 2), begin by having the patient extend all digits. Then, immobilize all other fingers (besides the one being tested) in this position of full extension. (This holds the profundus out to length via the “quadrigia phenomenon” (see Miscellany, below) and thus prevents the profundus from indirectly flexing the proximal interphalangeal (PIP) joint.) Instruct the patient to attempt to flex the finger. If the FDS is intact, the patient will flex at the PIP joint.

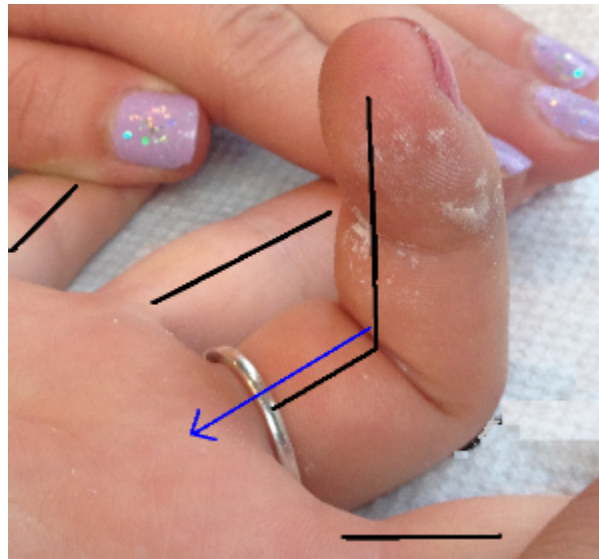


Figure 2: Testing the flexor digitorum superficialis [FDS]. The FDS to the middle finger is tested by holding the other 3 fingers in full extension, thereby immobilizing the profundus [FDP] (shown in black), and asking the patient to "bend the finger." Note that the FDP flexes the DIP specifically, but will also flex the PIP indirectly as well. By holding the other 3 fingers in extension, the FDP cannot move, and thus active PIP flexion in this position will demonstrate the integrity of the FDS (action shown in blue).

To evaluate the profundus, have the patient extend the digit. Grasp the finger to immobilize the PIP and MCP joints. Instruct the patient to attempt to flex the finger. If the profundus is intact, the patient will flex at the DIP joint.

Additionally, ensure the integrity of the digital neurovascular bundles (that course near the flexor tendons and may be injured concomitantly) by testing two-point discrimination. Doppler exam of the digital arteries may also be helpful.

## OBJECTIVE EVIDENCE

While flexor tendon injuries cannot be diagnosed via x-ray, both AP and lateral views of the hand should be obtained to rule out fracture or foreign body. CT and MRI have limited use in this diagnosis, although MRI would be the better of the two to evaluate soft tissue injuries and relative location in the hand or forearm of the tendon stumps. Depending on the experience of the operator, ultrasound may also be used to evaluate tendon integrity within the sheath.

## EPIDEMIOLOGY

Hand injuries make up one of the most common complaints treated in the emergency department (14-30%). Of these, tendon injuries account for approximately 30% of presentations, second in frequency only to patients presenting with fracture (40%).

## DIFFERENTIAL DIAGNOSIS

A direct flexor tendon injury must be suspected in all patients who present with an inability to flex one or more digits. However, depending on the mechanism and extent of injury, other injuries may also be present. Important considerations include fractures of the metacarpals or phalanges that may entrap the flexor tendon,



limiting its motion. Other considerations include injuries to the flexor sheath or pulley system, neurovascular injuries that impair or completely eliminate neurologic or vascular supply to the tendon or associated muscle body, or, as stated in the “Red flags” section, infection of the tendon sheath that limits movement of the tendon.

If multiple digits are affected without sign of infection or laceration to the hand, the cause likely lies proximal, that is up the arm towards the neck. Even without a direct injury to the flexor tendons, injury to the median or ulnar nerves proximal to their innervations of the FDS and FDP muscles in the forearm may result in decreased finger flexion (this could range from peripheral compression or injury in the arm or forearm, pathology of the brachial plexus, to cervical stenosis at the level of the spine).

## RED FLAGS

An inability to flex the fingers after a trauma is itself a “red flag.” Flexor tendon injuries require expeditious treatment from a surgeon who specializes in disorders of the hand.

Impaired flexion of a digit from swelling suggests infection.

Impaired passive motion of a digit because of guarding (that is, tensing the muscles to limit passive motion and pain) may suggest a forearm compartment syndrome.

## TREATMENT OPTIONS AND OUTCOMES

All tendon injuries should be examined to determine the specific anatomy that has been disrupted. At the time of initial evaluation, any open injuries should be thoroughly irrigated and cleaned. Depending on the extent of injury, the affected digit may rest in a more extended position than its neighbors. In this case, a dorsal blocking splint that keeps the wrist in neutral and the metacarpal phalangeal joints and PIP joints in partial flexion is advised.

All flexor tendon injuries should be referred to a surgeon who specializes in disorders of the hand. Flexor tendon injuries should be treated surgically expeditiously (within about 7 days of initial injury) to ensure the best results and a functional outcome. With the passage of time, the proximal edge of the lacerated tendon retracts further proximally; also, adhesions begin to form between the tendon and nearby structures.

The surgical treatment chosen for a particular injury is dependent on several factors, including location (zone) of injury, time since injury, condition of the tendon stumps and surrounding tissues, and experience level of the surgeon. It is generally agreed upon that partial lacerations involving <60% of tendon cross-sectional area should be debrided without primary repair.

Operative treatments of complete lacerations and those involving more than 60% of the tendon cross-sectional area include primary repair and staged reconstruction procedures with either donor tendon or silicon tendon implants.

Successful flexor tendon repair relies not only on successful reattachment at the time of surgery but successful rehabilitation. Consistent and reliable follow-up cannot be over-emphasized. Range of motion of the digit is begun almost immediately with both passive and active flexion and extension. This minimizes adhesions and ensures the patient retains the degree of motion attained in the OR.

(The use of regional anesthesia during surgery allows the patients to confirm with their own eyes that the tendon has been re-attached. Some surgeons believe that this provides the patient incentive and motivation to continue through the difficult and painful rehabilitation process.)

The key to good outcomes following a successful surgery is early mobilization and consistent therapy. Tendon tensile strength improves as the repair site is stressed, adhesions are minimized, and excursion is improved with early and frequent range of motion. A dorsal-blocking splint used between therapy sessions protects the digits and maintains the repair in a slightly shortened position (as compared to full extension), alleviating some of the stress placed across the site of reconstruction when the digit is held in full extension. With appropriate therapy

and protection of the repair site, good functional outcome can be seen in over 75% of patients. Most commonly encountered complications include adhesion formation, stiffness of finger joints, and even tendon rupture (risk of rupture is greatest at days 7-10 following repair).

Understanding of and compliance with therapy and restrictions is critical after surgical repair. Therefore, children and adults with limited mental capacity should be immobilized in a cast for one month following surgical repair.

## RISK FACTORS AND PREVENTION

Most tendon injuries are a result of accidental trauma. However, a history of prior or current infection of the tendons, as well as recent surgical procedures to the tendons or hand may predispose to tendon rupture by compromise of integrity due to local inflammation or due to suture failure, respectively. Prior surgery may compromise tendon integrity and tendon excursion via formation of scar tissue and adhesions of the tendon sheath. Also, some metabolic/inflammatory disorders can weaken tendon integrity and place them at risk of rupture (e.g. infection, rheumatoid arthritis).

## MISCELLANY

The ability to test FDS strength and excursion in isolation of FDP function is made possible by the quadriga phenomenon. A “quadriga” is a Roman chariot pulled by four horses. This phenomenon is named because the four FDP tendons are connected in the forearm and were thought to resemble the appearance of the reins of a quadriga.



*Figure 3: A Quadriga. A Roman coin showing the Roman emperor Tiberius riding on a quadriga (from Wikipedia)*

## KEY TERMS

Flexor tendon, Tendon injury, Tendon repair

## SKILLS

How to perform a thorough physical exam of the upper extremity, including how to evaluate individual nerves, both sensory and motor branches, how to assess distal digital pulses with Doppler exam, how to perform Allen's test, how to differentiate between FDS and FDP function, and how to evaluate two-point discrimination.

## GANGLION CYSTS OF THE HAND AND WRIST

---

Ganglion cysts are benign, fluid filled, soft-tissue masses found near the joints of the wrist and fingers (Figure 1). Ganglions usually grow out of the tissues surrounding a joint or a tendon sheath. The most common location for a hand ganglion — accounting for more than 75% of all cases — is on the dorsum of the wrist, emanating from the scapholunate joint. Cysts found at the DIP joint or IP joint of the thumb are denoted as “mucous cysts” and are associated with osteoarthritis. The ganglion cyst of the flexor tendon sheath is known as a “retinacular cyst.”

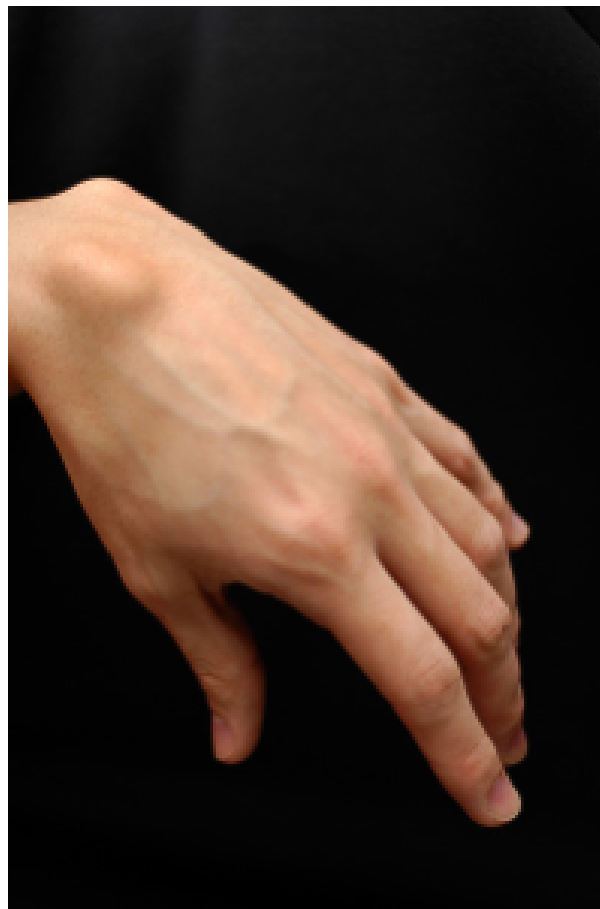


Figure 1: Ganglion cyst of the wrist. (From Wikipedia. <http://bit.ly/1miD7H5>)

### STRUCTURE AND FUNCTION

The exact etiology of ganglion formation is unclear, although the prevailing theory is that the cyst forms when there is a herniation of a weakened portion of the joint capsule or tendon sheath. Accordingly, it is thought that repeated mechanical stress, which would weaken the joint capsule, may be to blame.

So-called “myxoid” (meaning “resembling mucus”) degeneration of periarticular fibrous tissues apparently stimulates the production of hyaluronic acid by fibroblasts and proliferation of mesenchymal cells.

Mucous cysts are typically associated with arthritis in the finger joint.

The cysts themselves are thin-walled sacs. The wall is composed of collagen. Although cysts usually emanate from the joint (and remain connected to it via a “stalk” or pedicle) they lack a synovial lining and therefore are not, technically speaking, “synovial cysts.” (Along those lines, note that the fluid in the cyst is not true synovial fluid either: in cysts, the concentrations of hyaluronic acid, glucosamine, albumin, among others, is higher.)

Microscopic examination may show evidence of a mild, chronic inflammatory cellular reaction in the myxoid wall.

## PATIENT PRESENTATION

Ganglion cysts are usually oval and vary in both firmness and size. They usually form a visible bump, although some can be “occult.” Occult cysts are neither apparent to the naked eye nor to palpation and are visible only with imaging studies.

The average diameter of a ganglion cyst is about 2.0 cm, but cysts more than double this size have been observed. The size of the cyst may vary over time and may increase after activity and decrease with rest.

Although most ganglions are asymptomatic, if pressure is placed on the nerves that pass by the joint, patients may present with tingling and muscle weakness.

Pain is a feature of ganglion cysts, particularly retinacular cysts which are located in the tendon sheath. This pain is typically provoked with gripping motions.

The most common location for a ganglion is on the dorsum of the wrist, emanating from the scapholunate joint. This joint accounts for more than 75% of all cases. Small occult dorsal wrist ganglia may present as a dull ache in the wrist, with tenderness over the scapholunate ligament and pain with hyperextension of the wrist.

Mucous (also known as “mucinous”) cysts (Figure 2) are ganglion cysts specifically located in the dorsal region of the IP joints of the fingers. Mucous cysts are often associated with underlying arthritis and bone spurs (osteophytes).



*Figure 2: Mucous cyst of the thumb IP joint.*

Mucous cysts may ulcerate or may cause deformity of the nail.

Some patients with painless cysts may present to the physician simply to make sure “It’s not cancer.” For these patients, it may be worthwhile to assess for trans-illumination (that is, the passage of light through the cysts). To do this, the examiner dims the room lights and places a small flashlight directly on the mass. The absence of trans-illumination is not necessarily worrisome (for one thing, the cyst may be too small to manifest this phenomenon) but a positive test may be all the patient needs to see.

## OBJECTIVE EVIDENCE

Plain radiographs are generally not useful in diagnosing ganglion cysts, but can help rule out other pathologic processes.

Lab studies are generally not part of the diagnostic work-up for ganglions, as all values are expected to be within normal limits.

Advanced imaging studies may be useful in the evaluation of a mass when clinical diagnosis of a ganglion cyst is uncertain. Ultrasound of the wrist can also be used to evaluate ganglia. However, because ultrasound may not be uniformly available, is operator-dependent, and does not provide maximal anatomic information (e.g. underlying bone quality), MRI may be a better choice. Alternatively, some may recommend ultrasound over MRI because it is less expensive. Ultrasound, moreover, is less likely to find “incidentalomas,” that is, findings that are truly there but also truly irrelevant.

MRI findings of ganglia (Figure 3) include defined margins, a multi-loculated appearance, and wall enhancement. Ganglia are bright on T2 and dull on T1. After contrast administration and fat suppression, diffuse contrast enhancement is seen within the lesion. MRI may be particularly useful for the differentiation of a cyst versus a tumor. MRI is particularly helpful for volar (palmar) wrist ganglia, especially if surgery is planned, as the palmar side has more anatomic structures with which the cyst could interact.

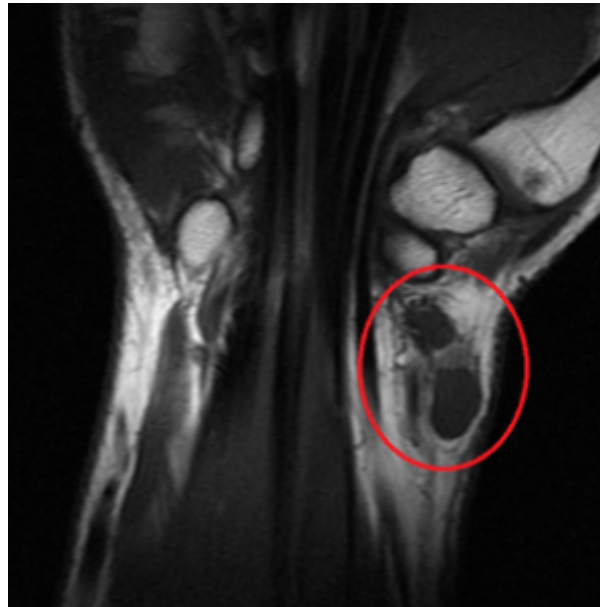


Figure 3: MRI of volar wrist ganglion

## EPIDEMIOLOGY

The true incidence of ganglion cysts is unknown; however, a recent study found that MRI scans of healthy asymptomatic volunteers identified wrist ganglia in about half of the study participants.

Ganglia are three times more prevalent in females than in males.

May occur in children and the elderly, but occur most commonly in the second to fourth decades of life.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of ganglions, including retinacular or mucoid cysts, may include a true synovial cyst, extensor tenosynovitis, synovial sarcoma, extra-skeletal chondrosarcoma, venous aneurysm, or xanthoma. A cyst that looks like ganglion cyst probably is a ganglion cyst; those that are atypical can be studied further with MRI or ultrasound, or sent to a hand specialist.

## RED FLAGS

Pain associated with fever, chills, and erythema about the mass may be due to underlying infection requiring urgent diagnosis and treatment.

Multiple joint pain and swelling may suggest a systemic inflammatory arthritis rather than a localized process such as a ganglion cyst.

## TREATMENT OPTIONS AND OUTCOMES

Many ganglion cysts resolve spontaneously. Consequently, some advocate that the best treatment is no treatment.

Ganglion cysts may shrink in size and later recur. In this situation patients may require nothing more than reassurance (as waxing and waning is not a worrisome sign).

For those patients in whom repetitive motion is a suspected cause (or for those whose personalities insist that some affirmative steps be taken) splints or braces are reasonable treatment options.

Aspiration of the cyst in the office (or variations, including attempts to tear the cyst wall with a needle or puncture the cyst in multiple locations) may be used to remove the fluid and thereby decompress the cyst. Because the stalk remains intact, recurrence of the cyst is not uncommon.

Augmenting the aspiration with injection of methylprednisolone does not seem to decrease the recurrence rate and does introduce the risk (albeit small) of complications. As such, steroids are not recommended. Indications for more aggressive treatment include pain, interference with activity, nerve compression, and imminent ulceration (as may be seen in mucous cysts).

Surgery involves removing the cyst along with a portion of the joint capsule or tendon sheath. In the case of wrist ganglion cysts, both traditional open and arthroscopic techniques are good options. Surgical treatment is generally successful as long as the “stalk” of the cyst is excised.

Osteophyte excision with capsulectomy is the mainstay of treatment for mucous cyst with underlying arthritis of the distal interphalangeal joint or interphalangeal joint of the thumb. Surgeons can excise the osteophyte alone with capsulectomy or the osteophyte as well as the cyst.

Cyst excision alone is not recommended because without capsulectomy there is a high rate of recurrence. Osteophyte excision can sometimes lead to DIP joint instability.

IP joint arthrodesis prevents recurrence of mucous cysts and is a good option if the pain is thought to be secondary to underlying arthritis.

The recurrence rate after cyst puncture and aspiration is greater than 50% for cysts in most locations, but is less than 30% for cysts in the flexor tendon sheath. The recurrence rate after aspiration is higher than the rate after excision of the cyst. The reported incidence of recurrence following cyst excision has varied from ~1% to 50%, presumably due to variations in cyst size, configuration, location, surgical technique, and postoperative immobilization. Stalk resection has been advocated as an important measure to decrease potential recurrence.

In a large prospective trial comparing reassurance alone to aspiration or surgical excision, the resolution of symptoms (wrist pain, weakness, and stiffness) was similar between the 3 groups. However, 58% of patients in

both the reassurance and aspiration groups had persistence of the lesion, whereas 39% of the surgical excision group had recurrence. Up to 80% of those who had aspiration or excision were satisfied, compared with only 53% of patients who were solely reassured—though this may represent a psychological action bias.

In a study of palmar wrist ganglions, there was no difference in symptoms regardless of whether they were treated by excision, aspiration, or observation.

Aspiration of palmar wrist ganglions is not recommended due to the proximity to the radial artery.

Retinacular ganglions often resolve spontaneously, with estimates greater than 50% resolution. In a study of children less than 10 years old with untreated retinacular ganglions, 100% of them resolved. However, many patients with retinacular cysts are symptomatic if they do not resolve.

Mucous cyst excision with osteophyte debridement does not always provide complete pain relief in the context of an underlying arthritic joint because the pain is often caused by the underlying arthritis and not the cyst (or osteophyte).

Cyst excision alone is not recommended, as without osteophyte excision, there is a recurrence rate of 10-27%. Cyst excision with DIPJ osteophyte excision results in a 0% to 3% recurrence rate, with many but not all patients having resolution of nail ridging as well. Surgical complication rate is roughly 8% and may include wrist stiffness, pain, keloid formation, weakness, scar tenderness, infection, decreased motion, damage to the scapholunate interosseous ligament, tendon injury, and neurovascular injury.

## **RISK FACTORS AND PREVENTION**

Ganglion cysts can develop in anyone but are more commonly observed in women. Joints or tendons that have been injured in the past are more likely to develop ganglion cysts in the future. Arthritis is a predisposing factor for mucous cysts.

There are no commonly accepted preventive measures.

## **MISCELLANY**

The term “Bible cyst” is sometimes used to describe ganglion cysts of the hand. This name is derived from the practice of striking (or should we say “smiting”?) cysts with a Bible to rupture them and drain into the surrounding tissues. This is not a currently recommended treatment.

Although physicians may be dismissive of ganglion cysts, given the benign natural history of this condition, patients can have substantial concerns. Indeed, in one report, a majority of patients seeking medical attention for their ganglion cysts came because of concerns beyond symptoms, including alarm about the cyst’s appearance or malignant potential.

## **KEY TERMS**

wrist, ganglion, cyst, mucus cyst, retinacular cyst, tendon sheath ganglion

## **SKILLS**

Recognize the clinical appearance of hand and wrist ganglion cysts. Know how to perform trans-illumination. Recognize ultrasound and MRI images of hand and wrist ganglion cysts. Know how to indulge your patients’ “action bias” without ruining the placebo effect all the while engaging in a fair and accurate informed consent discussion (good luck!). Describe the rationale for various treatment options to patients with this disorder including most especially not treating.

## KIENBOCK'S DISEASE

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Kienböck's disease is the eponym for avascular necrosis of the lunate (Figure 1), the bone between the scaphoid and triquetrum in the center of the proximal row of the carpus. Left untreated, this disorder results in fragmentation with progressive collapse of the lunate and subsequent wrist arthritis.



*Figure 1: The lunate (shown in red)*

### STRUCTURE AND FUNCTION

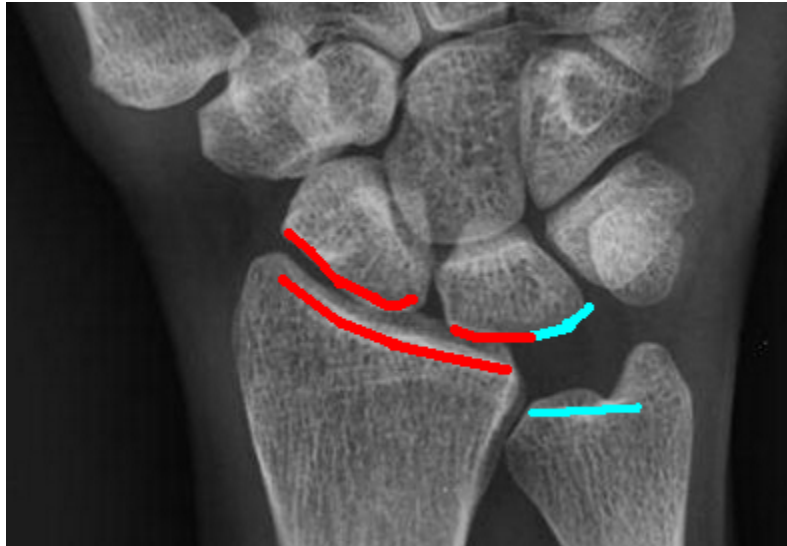
Histopathologic studies of involved bone demonstrate reactive new bone and granulation tissue surrounding necrotic areas with empty lacunae, fatty necrosis, and absent osteoid. The osteonecrosis is thought to occur as a result of disrupted lunate vascularity. The lunate blood supply is tenuous as most of the lunate is covered with articular cartilage. Hyperextension or hyperflexion of the wrist may compress these vessels against the rim of the distal radius, damaging them and leading to decreased blood supply to the lunate resulting in avascular necrosis or death of the bone tissue. The small veins may also be injured and occluded leading to increased venous congestion and pressure in the bone, which may contribute to the development of nonviable bone.



Kienböck's disease results from an infarction in the bone. Owing to ischemia, the bone dies – a process known as avascular necrosis or osteonecrosis. Dead bone cannot remodel (i.e., the process by which osteoclasts remove damaged bone and osteoblasts synthesize new bone). Accordingly, microscopic damage accumulates, and over time, the structural properties deteriorate and the bone begins to collapse, fracture, and fragment. In the lunate, osteonecrosis results in proximal migration of the capitate and loss of carpal height, altering wrist mechanics and increasing forces on the adjacent joints predisposing them to degenerative arthritic changes.

The exact etiology of Kienböck's disease is unknown. Interruption of the blood supply to the lunate may occur at the level of the extraosseous vessels or small intraosseous branching vessels. Additionally, venous congestion or obstructed outflow has been shown to increase the pressure within the lunate and may contribute to the process.

Mechanical factors have also been postulated to be involved in the development of Kienböck's disease. Negative ulnar variance (Figure 2), in which the ulna is short relative to the radius, is believed to be a risk factor.



*Figure 2: Negative ulnar variance. This patient has negative ulnar variance, that is, the ulna does not reach as far distally as the radius. Here, the entire articulation is given by the red lines. The blue lines represent the articular area not used because the ulna is too proximal. In this anatomic configuration the carpus articulates with the radius alone, thereby creating a smaller contact area for the lunate, with resultant greater pressure on it. (Pressure, is of course, force/area; and a smaller area for a given force leads to increased pressure.) In turn, the bone is more apt to fail mechanically.*

Repetitive microtrauma may also play a role in the disease given the clinical observation that young male laborers are most commonly affected.

A combination of both intrinsic and extrinsic factors related to the patient's anatomy as well as the forces the wrist is exposed to likely play a role in the disease.

## PATIENT PRESENTATION

Kienböck's disease presents as wrist swelling and pain with resultant weakness of grip and limited wrist range of motion. Kienböck's disease typically affects the dominant hand of male laborers aged 20 to 40 years. Progressive pain, without a clear history of acute trauma, is the primary presenting complaint. Patients usually localize the pain centrally over the dorsum of the wrist at the radiocarpal joint, which may be aggravated by gripping activities and wrist extension. Pain is initially just activity related, but as the disease progresses it may persist even at rest. Characteristic pain may be reproduced with passive extension of the long finger.

## OBJECTIVE EVIDENCE

Plain radiographs are the initial diagnostic test of choice (Figure 3) but may be normal at the onset of the disease. A subtle linear fracture without sclerosis or collapse may be seen on radiographs early in the disease.

As the disease progresses, the lunate becomes increasingly sclerotic, often with one or multiple fracture lines. The lunate then begins to fragment and collapse leading to proximal migration of the capitate and hyperflexion of the scaphoid.



*Figure 3: X-ray of wrist with Kienbock's (From radiopaedia.org)*

The end stage radiographic changes include joint space narrowing and arthritis of the radiocarpal and midcarpal joints.

Advanced imaging studies are also useful in the evaluation and diagnosis of Kienböck's disease. Magnetic resonance imaging (Figure 4) is the most sensitive test for detecting early Kienböck's. Uniformly decreased T1 signal throughout the lunate is highly suggestive of Kienböck's disease.

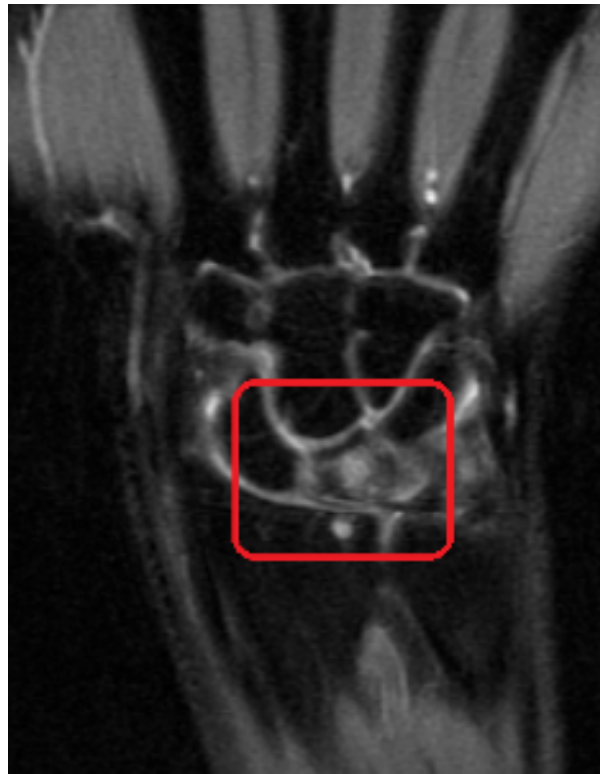


Figure 4: MRI of wrist with Kienbock's (from Radiopaedia.org  
<http://bit.ly/1nAaNkq>)

Computed tomography (CT) best demonstrates the degree of bony destruction and structural collapse once there is radiographic evidence of sclerosis, fracture, or fragmentation of the lunate.

Laboratory studies are not generally necessary or useful in evaluating or diagnosing Kienböck's disease. Inflammatory and rheumatologic markers are not elevated in contrast to infectious or inflammatory conditions.

## EPIDEMIOLOGY

The true incidence of Kienböck's disease is not known or reported, as patients in the early stages may not present for medical evaluation or may elude diagnosis. This disorder most commonly affects males aged 20 to 40 years old. Affected women tend to be older, with less predilection for the dominant hand being affected. There is a higher incidence of Kienböck's disease in patients with negative ulnar variance, thought to be related to increased forces transmitted across the radiolunate joint.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of wrist pain is of course extensive. Soft tissue injuries such as tears of the scapholunate ligament, lunotriquetral ligament, or triangular fibrocartilage complex can lead to chronic pain and/or instability of the wrist. Occult or missed carpal fractures, most commonly of the scaphoid, resulting in a nonunion, can also lead to persistent wrist pain and weakness. These soft tissue and bony injuries can be differentiated from Kienböck's disease based on the location of tenderness on physical exam and, ultimately, on the radiographic findings.

Additionally, numerous conditions not associated with trauma can present similarly. Inflammatory or degenerative wrist arthritis results in chronic wrist pain but typically affects older patients or involves other joints in addition to the wrist. Dorsal ganglions can produce wrist pain with an insidious onset, but are associated with a palpable soft tissue mass that is not present in Kienböck's. Ulnar abutment may also present

with wrist pain and radiographic changes in the lunate, but it is differentiated with magnetic resonance imaging (MRI). Finally, avascular necrosis of the scaphoid (Preiser's disease) or proximal capitate may present similarly to Kienböck's but can be differentiated from it based on radiographic and advanced imaging findings.

## RED FLAGS

Wrist pain associated with fever, chills, and erythema about the wrist may be due to underlying infection requiring urgent diagnosis and treatment. Night pain awakening the patient from sleep may be due to occult malignancy requiring accurate diagnosis to guide treatment. Pain with ulnar deviation of the wrist may be due to ulnar impaction, a diagnosis frequently confused with Kienböck's disease. Finally, multiple joint pain and swelling may suggest a systemic inflammatory arthritis rather than a localized process like Kienböck's disease.

## TREATMENT OPTIONS AND OUTCOMES

Numerous treatment options have been described with variable results. The earliest stage of the disease is initially treated conservatively with splint or cast immobilization. If symptoms persist despite conservative treatment, surgery is considered. Surgical intervention, in early stages, includes revascularization procedures or joint unloading treatments to decrease the load on the lunate.

In patients with neutral or ulnar positive variance and preservation of lunate height, revascularization procedures can be considered. Implantation of a vascular pedicle as well as several different pedicled vascularized grafts have been described including the fourth extracompartmental artery, second dorsal metacarpal artery, a pedicled vascular pisiform, the pronator quadratus bone flap, dorsal distal radius pedicled grafts, and nonvascularized bone grafting. Each of these procedures requires an intact cartilage shell with no degenerative changes.

In patients with ulnar minus variance with no degenerative changes, joint leveling procedures such as radial shortening or, less commonly, ulnar lengthening, to produce 1-mm ulnar positive variance can be considered to offload pressure on the radiolunate joint. Capitate shortening to decrease the forces across the lunate has also been described (for ulnar neutral or positive variant patients).

If carpal collapse or degenerative changes are present, then proximal row carpectomy or limited fusion of the scaphoid, trapezium, and trapezoid or the scaphoid and capitate is preferable to correct and maintain carpal alignment. For patients with advanced pancarpal arthritis, total wrist fusion is the most reliable means of achieving durable pain relief.

Given the relative rarity of the disorder, current literature reporting treatment outcomes in Kienböck's disease is limited to small retrospective studies. Non-operative treatment has demonstrated limited success, with most patients worsening or experiencing no significant change in their symptoms.

Vascularized bone grafting procedures have demonstrated successful outcomes. Several studies have demonstrated subjective pain improvement in greater than 90% of patients, improvement in grip strength to nearly 90% of the contralateral hand, and arrest of lunate collapse or evidence of revascularization in approximately 60-70% of patients. Joint leveling procedures such as radial shortening osteotomy have yielded similar results with well over 90% of patients noting improvement in their pain; however, fewer patients demonstrate evidence of lunate revascularization. While patients treated with revascularization or joint leveling procedures may have radiographic evidence of progression of arthritic changes, these do not generally correlate with subjective symptoms.

## **RISK FACTORS AND PREVENTION**

Several anatomical variations may predispose an individual to developing Kienböck's disease. Negative ulnar variance is thought to make Kienböck's disease more likely, as noted above. Likewise, radial inclination flattening, decreased extraosseous supply to the lunate, and diminished intraosseous arterial branching may increase the risk of the disorder. Additionally, several systemic conditions — including sickle cell anemia, gout, systemic corticosteroid use, and cerebral palsy — have been reported to have an association with Kienböck's, although a clear increased risk warranting screening studies in these patients has not been shown.

Repetitive microtrauma may also increase the risk of Kienböck's, as it has been noted that the condition commonly affects the dominant hand of laborers. This is likely the only modifiable risk factor; however, the exact offending activity has not been elucidated. As with many other musculoskeletal complaints, activity modification and avoidance of aggravating activities is a reasonable first step in preventing further injury.

## **MISCELLANY**

Kienböck's disease is named for the Austrian radiologist, Dr. Robert Kienböck who described the disorder in 1910.

## **KEY TERMS**

lunate, avascular necrosis, Kienböck's disease, lunatomalacia, vascularized bone grafts

## **SKILLS**

Recognize radiographic and MRI images of lunate avascular necrosis. Relate surface anatomy of the wrist to underlying carpal anatomy to facilitate accurate palpation and localization of the point of maximum tenderness. Describe the rationale for various treatment options to patients with the disorder.

## LATERAL AND MEDIAL EPICONDYLITIS OF THE ELBOW

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Epicondylitis of the elbow is a misnomer because it is neither primarily a disease of the epicondyle, nor is it exclusively inflammatory (as the suffix “itis” would suggest).

Instead, epicondylitis is a condition of degenerative tendinopathy within the wrist extensor tendon (as in lateral epicondylitis) or flexor tendon (as in medial epicondylitis), tendons which originate at the elbow.

The common term “tennis elbow” refers to lateral epicondylitis which affects the origin of the wrist extensor muscles, while the term “golfer’s elbow” refers to medial epicondylitis which affects the origin of the flexor/pronator muscles.

Epicondylitis is an overuse injury in which the rate of tendon damage exceeds the rate of tendon repair. This disequilibrium causes pain at the elbow and, ultimately, leads to impaired function – primarily in tasks that involve a power grip and require a stable wrist joint.



*Figure 1: Surface landmark of the lateral epicondyle and the point of origin of the wrist extensor muscles.*

### STRUCTURE AND FUNCTION

Epicondylitis is an overuse injury of the wrist extensor and flexor tendons including the extensor carpi radialis brevis, flexor carpi radialis, and pronator teres.

These muscles originate from the humerus (Figure 1) and cross the elbow joint, but their main action is at the wrist. They act at the wrist via concentric contraction (that is, shortening and changing the position of the joint), but they are also important in stabilizing the hand, wrist, and fingers, either isometrically (not moving at all) or via an eccentric contraction (actually lengthening while offering a resisting force). It is these eccentric contractions, defined as muscle contraction in one direction while another force elongates the muscle in the opposite direction, that are likely to cause microscopic tears within the tendon itself and its origin.

Lateral epicondylitis is commonly called “tennis elbow” because it is a common injury sustained by in tennis players hitting an off-center single arm backhand. When hitting the ball, the player’s wrist extensors are active, to hold the wrist in position. When the tennis ball hits the racket, it applies a force on the wrist in the direction of flexion. This force pulls on the contracting extensor muscles and can create microscopic tears in the fibers that originate from the lateral epicondyle.

On the lateral side the primary focus of damage is within the extensor carpi radialis brevis; on the medial side it is within the flexor carpi radialis and pronator teres origin.

The histology of epicondylitis has been described as “angiofibroblastic hyperplasia”: namely, the presence of fibroblasts and vascular tissue, along with degenerative and torn tendon fibers. (Note that pathological specimens are obtained at the time of surgery, and surgery is, of course, reserved for only severe cases. Thus, as far as we know, “angiofibroblastic hyperplasia” may be present only in end-stage disease.)

## PATIENT PRESENTATION

Patients with epicondylitis present with a chief complaint of pain in the elbow on the affected side, especially with grasping.

Patients with epicondylitis often volunteer a history of repetitive forearm use, including sports involving rackets or bats, but also occupational activities such as working with tools.

In early phases of the condition, patients experience a minor ache after extensive activity. As the disease progresses, the signs and symptoms of epicondylitis are not only more severe, but may also be present without recent activity.

The presence of focal epicondylar pain and tenderness that is worse with provocation (stress on the wrist) is key to the physical diagnosis of epicondylitis.

Patients with epicondylitis complain of pain localized to the elbow; however, the best physical examination maneuver to provoke pain involves manipulation of the wrist, as the affected muscles are primarily used to stabilize the wrist. This is because while the lateral structures (especially the extensor carpi radialis brevis) are active wrist extensors, they also stabilize the wrist by resisting wrist flexion (with so-called eccentric or isometric contraction).

The importance of a stabilizing force against wrist flexion is seen dramatically in the mechanics of a tennis backhand stroke. This action requires that the wrist be held, fixed, in a neutral position despite the impulse of the ball, which, unopposed, would flex the wrist. A similar stabilizing force is employed on a routine basis in everyday grasping. Recall that the muscles of finger flexion originate on the volar surface of the forearm and cross the wrist on their journey to the phalanges. Unless these muscles are opposed by a countervailing wrist extension force, their firing will flex the wrist as well as the fingers. Put simply, in order to flex the fingers without flexing the wrist, the wrist extensor muscles must act. Repetitive grasping with the fingers therefore requires repetitive use of the wrist extensors, even if no wrist movement is produced.

Lateral epicondylitis can be caused by routine grasping tasks because they require the use of wrist extensors.

Symptoms of epicondylitis can be provoked on physical exam by asking the patient to hold his or her hand in maximal wrist extension, with the examiner acting against the patient to flex the wrist (Figure 2). Pain at the origin of the extensor muscles in the vicinity of the epicondyle is diagnostic. Note that the point of maximal tenderness is not at the epicondyle but is, rather, slightly distal, within the tendon itself.



*Figure 2: Provocative examination of lateral epicondylitis. The patient is asked to actively extend the wrist against resistance; pain and tenderness near the origin of the wrist extensor muscles is a positive response.*

## OBJECTIVE EVIDENCE

Epicondylitis is, for the most part, a clinical diagnosis: tenderness, especially provocative tenderness, in a patient with an apt history, makes the diagnosis.

An MRI (Figure 3) may show abnormal signal within the tendon at its origin, typically signifying chronic granulation tissue at the injured site. This test may also be used to exclude other conditions.



*Figure 3: Elbow MRI with lateral epicondylitis (from radiopaedia.org <http://bit.ly/1KSCmut>)*

Similarly, an EMG may be helpful to exclude compression neuropathy. This is not 100% reliable, however. Plain radiographs may help exclude arthritis or loose bodies.



## EPIDEMIOLOGY

The exact incidence of epicondylitis is not known, as there are mild forms of the condition that do not present for medical attention.

In general, it is safe to assume that epicondylitis is very common and should be high on the differential diagnosis of elbow pain.

Clinical experience suggests that medial epicondylitis is far less common than lateral epicondylitis. Moreover, the medial side of the elbow has other structures that may be the source of pain, e.g. the medial collateral ligament and the ulnar nerve. Medial epicondylitis therefore perhaps deserves a less prominent place on the “default” list of causes of medial elbow pain.

The incidence of epicondylitis is highest in the fourth and fifth decades of life. It can be present in both older and younger patients, but like most tendinopathies, epicondylitis is mostly prevalent in middle age.

## DIFFERENTIAL DIAGNOSIS

Cubital tunnel syndrome, i.e., a compression neuropathy of the ulnar nerve at the elbow, is commonly seen in association with medial epicondylitis, but also may be seen on its own.

Entrapment of the radial nerve in the arcade of Frohse may mimic lateral epicondylitis (this can be diagnosed by EMG, clinical examination for radial tunnel syndrome, or diagnostic injection).

Cervical radiculopathy can also present as elbow pain: C5 or C6 nerve roots for lateral elbow pain; C8 or T1 nerve roots for medial elbow pain.

In addition, primary pathology within the joints, including arthritis, loose bodies and synovitis, may mimic the symptoms.

## RED FLAGS

None

## TREATMENT OPTIONS AND OUTCOMES

As epicondylitis is an overuse injury, it is best treated initially by a period of relative rest. Avoiding provocative activities, assuming they can be identified and omitted, may be helpful.

So-called “counterforce bracing” may also be helpful. This bracing involves a strap worn distal to the elbow that helps redirect force away from the origin (please see “Miscellany”, below).

Splinting the wrist may also be helpful for this elbow pain (Figure 4). Note that the finger flexor tendons of course cross the wrist as well. As such, active finger flexion will flex the wrist unless there is a so-called isometric contraction of the wrist extensors. That is, wrist extension is needed to oppose the flexion force. By stabilizing the wrist with a brace, the wrist extensor muscles can rest even while grasping.



*Figure 4: A wrist splint that may ameliorate elbow pain. By holding the wrist in a stable, fixed position, the brace allows the extensor muscles to rest during grasping. (From Flickr <http://bit.ly/1PaqK82>)*

Physical therapy, especially modalities such as ultrasound and tendon glides, may be helpful.

Cortisone injections are also an accepted mode of treatment. Cortisone's precise mechanism of action is not known with certainty, however, it is relatively effective at minimizing pain in the short term.

Surgical treatment of epicondylitis is reserved for severe cases. Lateral epicondylitis can be treated by excision of the chronic granulation tissue of the extensor carpi radialis brevis and subsequent repair with appropriate immobilization while the tendon heals.

In general, patients with epicondylitis do well – especially if they interpret the pain as a signal to stop doing what is causing the pain. Pain, to the extent that it enforces a period of rest, promotes recuperation.

Controlled studies documenting the natural history of treated and untreated epicondylitis, however, have not been performed.

## **RISK FACTORS**

Epicondylitis is an overuse injury that often occurs in the setting of aging. There is not much that can be done to prevent aging, of course. Patients who are entering the fourth and fifth decade of life, however, may need to modulate their activities accordingly.

It is also worth noting that poor form and technique – and the use of poorly maintained or adjusted equipment – may increase the risk of epicondylitis. For example, playing tennis with a racket poorly matched to one's skill and size may help inflict damage.

## MISCELLANY

The concept of counterforce bracing (Figures 5 and 6) is similar to the capo used in a guitar, which effectively changes the “origin point” of the guitar string.



Figure 5: Counterforce bracing for epicondylitis



Figure 6: The guitar capo shown in the photo (Source: Flickr <http://bit.ly/1nZsni5>) effectively shortens the guitar strings, just as a strap on the forearm shortens the extensor muscles. This shortening creates a new virtual point of origin (shown by the green arrow in Figure 5) and shields the proximal origin of the extensor tendons (purple arrow) and thereby allows it to rest and recover.

Just as a dog can have lice and fleas, a patient can have two simultaneous conditions, e.g. lateral and medial epicondylitis. This combination of tennis and golfer’s elbow has been described by Nirschl as “country club elbow.”

## KEY TERMS

epicondylitis, angiofibroblastic hyperplasia

## SKILLS

Perform physical examination to diagnose epicondylitis.

## MALLET FINGER AND OTHER FINGER EXTENSOR INJURIES

---

A mallet finger (Figure 1) results from a separation of the extensor digitorum from its insertion on the distal phalanx. This separation by tearing away (technically, an “avulsion”) results from hyperflexion of the distal interphalangeal (DIP) joint. A commonly seen mechanism of injury is a blow against the tip of the finger by a ball being caught. The injury can either cause the avulsion of the tendon off the bone or the avulsion of the tendon with a piece of the bone attached. When there is bony involvement of the distal phalanx, the injury is referred to as a “bony mallet.” An injury to the extensor digitorum more proximally, at the level of the middle phalanx, damages the so-called “central slip” and can lead to a boutonnière (meaning “button hole” in French) deformity. In a boutonnière deformity (Figure 2), the head of the proximal phalanx pops through the central slip (like a button through a button hole). This causes the characteristic deformity of flexion at the proximal interphalangeal (PIP) joint and hyperextension at the DIP joint.



Figure 1: Mallet finger. A patient with a mallet finger cannot extend the DIP joint.



Figure 2: An x-ray of the finger showing a boutonnière deformity: flexion at the proximal interphalangeal joint (red) and hyperextension at the distal interphalangeal joint (yellow) <http://radiopaedia.org/cases/boutonniere-deformity-grade-4-1>.

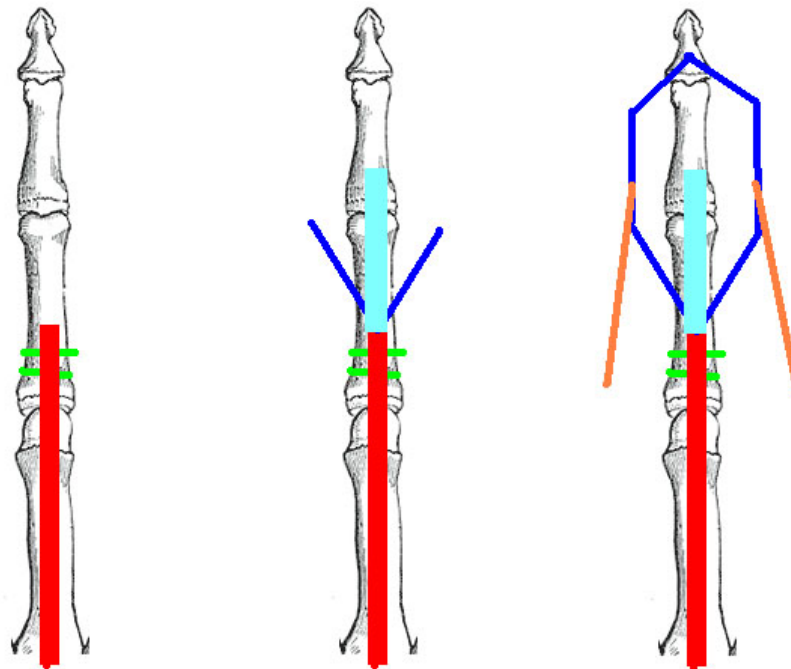
## STRUCTURE AND FUNCTION

The extensor digitorum communis extends the four lesser digits of the hand (excluding the thumb) via four tendons emanating from a single muscle (Figure 3). At the level of the metacarpophalangeal (MCP) joint, there is a common extensor tendon, which is maintained in its central position by “sagittal bands” on both the radial and ulnar sides. These sagittal bands attach to the proximal phalanx and allow the common extensor tendon to extend the metacarpophalangeal (MCP) joint.

Just distal to the MCP joint, the extensor tendon trifurcates: a “central slip” and two “lateral bands.”

The central slip crosses the PIP joint and attaches to the base of the middle phalanx. The central slip is therefore responsible for extension of the PIP joint.

The lateral bands project in the radial and ulnar directions, where they are joined by the tendons of the interosseous and lumbrical muscles. The lateral bands then move more centrally and cross the dorsal surface of the DIP joint where they then insert commonly on the distal phalanx and therefore extend the DIP joint.



*Figure 3: Extensor mechanism of the finger. In the drawing on the left the common extensor tendon is shown in red. It attaches to the proximal phalanx via bands and extends the MCP joint. In the center figure, the trifurcation is shown. the central slip, shown in sky blue, inserts on the middle phalanx and extends the PIP joint. The lateral bands are shown in blue. In the right-most drawing, the lateral bands are shown joined by the tendons of the interosseous and lumbrical muscles and inserting on the distal phalanx, thereby extending the DIP.*

An injured sagittal band, it follows, will fail to keep the extensor tendons centralized. Such an injury can cause subluxation of the extensor tendons over the MCP joint when a fist is made, causing a snapping sensation in both flexion and extension at the joint.

An injury to the central slip (Figure 4) can lead to an inability to extend the PIP joint. Also, such an injury may allow the lateral bands to fall in the volar direction (that is, toward the palm). If the lateral bands end up on the volar side of the PIP joint, they will in turn flex that joint, which accentuates the boutonniere deformity.

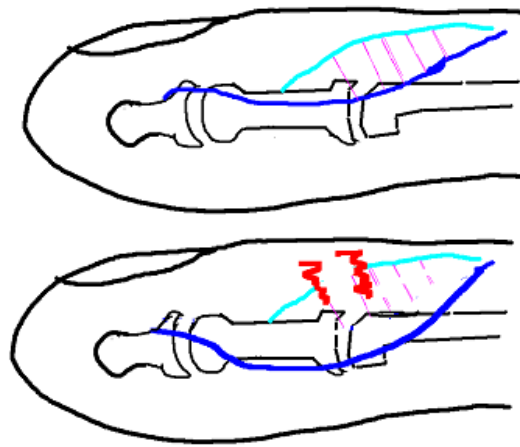


Figure 4: Central slip injury. An injury to the central slip (sky blue) can cause the lateral bands (dark blue) to fall toward the volar side of the finger and make them flexors of the PIP joint.

## PATIENT PRESENTATION

Patients with mallet finger will present after a traumatic injury to the tip of the finger. The dorsal DIP may be ecchymotic and usually painless. There is an obvious droop of the tip of the finger and the patient will be unable to fully actively extend the finger at the DIP joint.

A chronic mallet finger can present as a so-called “swan neck deformity” characterized by DIP hyperflexion with PIP hyperextension. (This the “opposite” of a boutonnière deformity.) The failure of the extensor tendon at the distal phalanx coupled with unopposed action of the flexor digitorum superficialis (FDS) leads to the DIP hyperflexion. Concentration of the extensor on the middle phalanx alone causes the PIP hyperextension.

Patients with central slip injuries may present with pain and swelling along the dorsal PIP joint. The patient may initially have mild weakness in extending the finger at the PIP joint. If the patient presents weeks to months after the injury a progressive boutonnière deformity may develop.

On physical examination a central slip injury can be diagnosed with the Elson test. In order to perform the Elson test, the PIP joint is held fully flexed. If the central slip is injured the patient will be able to extend the DIP joint despite maintaining the PIP in flexion.

Patients with sagittal band injuries will present with a traumatic injury over their knuckle. Sagittal band injuries are commonly called “boxer’s knuckles.” The extensor tendon may slip back and forth over the MCP joint. Patients may report a snapping sensation while flexing the digit with a visible volar subluxation or dislocation of the extensor tendon when making a fist. With progression of the injury, patients may develop difficulty extending the finger at the MCP joint.

## OBJECTIVE EVIDENCE

The best objective evidence for an extensor tendon injury is the physical exam. All finger injuries should have an x-ray on initial evaluation. An x-ray of the finger (A/P, lateral, and oblique) should be ordered. A dedicated finger x-ray should be reviewed, as a hand x-ray will often have overlap of the digits leading to difficulty in diagnosing fractures and dislocations. A finger x-ray may show a bony avulsion at the proximal aspect of the distal phalanx (bony mallet). An x-ray can also assess the presence or absence of a dislocation.

The PIP joint should be assessed for an avulsion fracture of the dorsal base of the middle phalanx indicating a possible central slip injury. A central slip disruption is diagnosed when a volar dislocation of the PIP joint is noted on lateral x-ray.

Patients with a sagittal band injury will usually have normal x-rays. Radiographs are obtained to rule out an associated fracture or subluxation of the MCP joint.

If the diagnosis is still in question, an MRI or ultrasound should be considered to further assess the integrity of the extensor mechanism.

## **EPIDEMIOLOGY**

Mallet finger is most commonly seen in the small, ring, and middle fingers in the dominant hand. Mallet finger more commonly affects men, usually during work or sports related activities. With appropriate splinting most patients can return to work.

Injuries to the sagittal bands or the central slip are less common. Central slip dysfunction may be related to direct trauma or to a volar dislocation of the PIP joint. The sagittal bands may also be damaged from a direct injury to the MCP joint, as occurs in boxing. The radial sagittal band is most commonly injured leading to ulnar subluxation of the extensor mechanism. In addition, patients with rheumatoid arthritis can develop attritional ruptures of the sagittal band.

## **DIFFERENTIAL DIAGNOSIS**

Patients who present with trauma to the distal phalanx and DIP joint may have other associated injuries including nail bed injuries, tuft fractures, nail plate avulsion, and distal phalanx fracture dislocations without tendon injury to name a few.

Patients with a flexion contracture of the PIP joint often appear as if they have a boutonnière deformity. Patients with this pseudo-boutonnière will have full flexion of the DIP joint whereas a true boutonnière deformity leads to hyperextension and limited flexion of the DIP joint.

Sagittal band injuries are commonly misdiagnosed as a trigger finger due to the snapping sensation with finger flexion and extension.

## **RED FLAGS**

Mallet finger leads to the inability to actively extend the DIP joint. It is important to obtain an x-ray to rule out a fracture dislocation of the DIP joint which can present in a similar fashion.

An injury to the central slip may initially lead to mild tenderness about the dorsal PIP. One must have a high index of suspicion for this injury and splint early to prevent a boutonnière deformity. If left untreated, a central slip injury may lead to a stiff deformed finger as the ligaments and volar plate contract. It is very important to refer these patients to a hand specialist. Beware of the volar PIP joint dislocation since the central slip is torn during this injury and treatment is different from the more common dorsal PIP joint dislocation.

It is critical to observe the location of the extensor mechanism as the patient makes a fist to best assess for subluxation.

## TREATMENT OPTIONS AND OUTCOMES

In the acute setting, a mallet finger is treated with splinting (Figure 5) of the DIP joint in full extension for six weeks and occasionally up to three months. Even in the chronic setting extension splinting can be tried. If non-operative treatment is unsuccessful, a primary repair or reconstruction may need to be performed by a hand specialist. If there is subluxation of the DIP joint due to a large bony articular fragment avulsion, primary repair may be the first line of treatment.



Figure 5: DIP splint. Splint holding the DIP in extension  
(From Flickr <http://bit.ly/1POQUfC>)

Central slip injuries are initially treated by extension splinting of the PIP joint. DIP joint flexion exercises are started to prevent stiffness and tendon migration. If non-operative treatment with splinting fails, occasionally operative repair or reconstruction is considered.

Sagittal band injuries are initially treated with extension splinting of the MCP joint for four to six weeks. Sagittal band injuries are one of the rare instances where the MCP joints are immobilized in extension. The decision whether repair or reconstruction of the sagittal band is needed is one best made in consultation with a hand specialist.

The goal in treating a patient with an extensor mechanism injury is to regain full motion of the digit without pain. Despite non-operative or operative treatment of a mallet finger, patients often develop a mild extensor lag (droop). The most important aspect of treating a patient with an extensor tendon injury is coordinating care with a skilled hand occupational therapist; this allows the patient to regain motion while preventing contracture.

A central slip injury treated acutely often leads to full range of motion. Once a boutonnière deformity develops, operative or non-operative treatment typically results in a persistent mild deformity.

Sagittal band injuries treated with extension splinting or by operative repair or reconstruction can be expected to have good results with near full range of motion.

## RISK FACTORS AND PREVENTION

In order to prevent injuries to extensor tendons of the hand it is important to wear appropriate protective devices during high risk activities.

Unfortunately, injuries to the hand occur despite use of these devices, and it is important to seek treatment early to prevent further deformity and disability.



## **MISCELLANY**

The word “boutonnière” comes from the French word for “buttonhole.” In boutonnière deformities, the head of the proximal phalanx “buttonholes” through the defect in the extensor mechanism from the central slip injury leading to the deformity.

## **KEY TERMS**

mallet finger, swan neck deformity, boutonnière deformity, extensor tendon, central slip, extensor digitorum communis, lateral bands, sagittal bands, boxer’s knuckles.

## **SKILLS**

Examination of extensor mechanism of the finger.

## METACARPAL FRACTURES

---

The metacarpals are essential for hand function. Fractures to these bones may affect hand strength and motion, inhibiting the ability to grip and hold objects. Fractures are often the result of high-energy impact, likely seen in athletics, trauma and work injuries. These fractures often can be treated with immobilization and have a good prognosis.

### STRUCTURE AND FUNCTION

The metacarpals consist of five tubular bones that articulate proximally with the carpus and distally with the phalanges (Figure 1). From radial to ulnar, the bones are numbered one (thumb) through five (small finger) and consist of a shaft, base, and head.



*Figure 1: The 5 metacarpal bones are shown in red.*

The metacarpal base is cuboidal in shape, and ligaments provide articulations between the metacarpal bones and the carpus at the carpometacarpal ("CMC") joint.

The body (or shaft) of the metacarpal is concave with respect to the palmar surface. There is a medial, lateral, and dorsal surface on the metacarpal shaft. The medial and lateral surfaces provide attachment for the interossei muscles. This attachment, along with the extensor and flexor muscles crossing (but not attaching to the metacarpal), produces the main deforming force that may displace shaft fractures. The extensor tendons lie atop the flat dorsum of the metacarpal shaft.

The metacarpal neck lies just proximal to the head, distal to the shaft.

The metacarpal head articulates with the proximal phalanx of each finger, with tubercles on each side providing attachment for the collateral ligaments. The dorsum of the head accommodates the extensor tendons while the palmar surface has a ridge for the flexor tendons. The head of the metacarpal receives its own blood supply from the collateral ligaments; this arrangement predisposes the head to possible avascular necrosis with a ligament injury, as the ligament injury may disrupt perfusion.

The first metacarpal is shorter and wider than the other metacarpals and has a more extreme angulation with the carpus. The second and third metacarpals are more rigidly fixed upon the corresponding carpal bones than the fourth and fifth metacarpals. The looser attachments of the fourth and fifth metacarpals allow them to oppose the thumb.

## PATIENT PRESENTATION

Metacarpal fractures usually occur after a fistfight, car accident, or fall. At times, a crush mechanism is responsible. Findings include pain (most intense over fracture site), edema, a shortened finger or finger deformity (such as depressed or missing knuckle), and bruising.

Physical examination of the hand may reveal deformities, decreased grip strength, and possible finger misalignment. In a normal flexed hand, the fingertips should align and point towards the scaphoid tubercle on the radial volar aspect of the wrist. Range of motion tests should be conducted, and the hand should be assessed for sensory changes to determine possible nerve damage.

Fractures within each of the four regions of the metacarpal bone – base (Figure 2), shaft (Figure 3), neck, and head – are considered distinctly.

Metacarpal base fractures usually occur as a result of significant axial load to a flexed hand as seen in clenched fist injuries.



*Figure 2: Fracture of the base of the 5th metacarpal. Soft tissue swelling, denoted by the arrow, is perhaps a more obvious finding than the fracture itself. To the right, a close-up view of the base of the metacarpal shows the fracture line, outlined in red in the image below. (From radiopaedia.org <http://bit.ly/1Sykv2o>)*

Shaft fractures are most frequently the result of direct trauma, axial loading, or twisting injury. These present respectively as transverse fractures (with comminution at times) and spiral fractures. The pull of the interossei muscles and flexor tendons can deform shaft fractures leading to metacarpal shortening or angulation.



Figure 3: Fracture of the shaft of the 5th metacarpal. (From Radiopaedia.org  
<http://bit.ly/1UDuqCY>)

Metacarpal neck fractures are the most common type of metacarpal fracture. Such a fracture seen in the 5th (or rarely, the 4th) metacarpal neck is called a “boxer’s fracture” (Figure 4). Metacarpal head fractures are rare and usually require surgical intervention.



Figure 4: Boxer’s fracture, i.e., a fracture of the neck of the 5th metacarpal. (From radiopaedia.org <http://bit.ly/1PMHFwG>). First metacarpal (thumb) fractures are uncommon. However, these fractures deserve special attention.

A notable thumb injury is the Bennett's fracture (Figure 5), a partial articular fracture of the first metacarpal base. This fracture results in shortening and deformation of the thumb, due to lack of support from the bone and shear pull from the abductor pollicis longus.

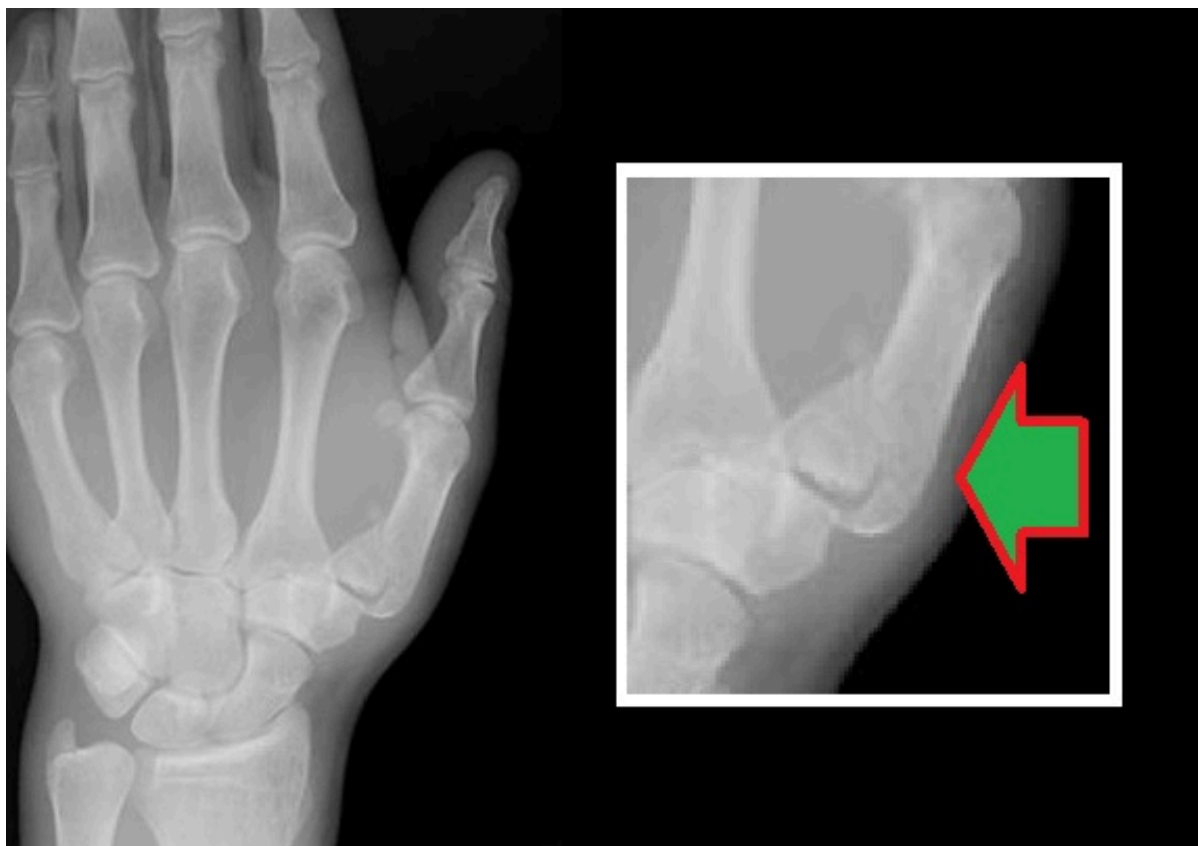


Figure 5: Bennett's fracture. (modified from radiopaedia.org <http://bit.ly/1nIFmVG>)

Another notable thumb injury is the Rolando's fracture (Figure 6), one with T or Y shaped intra-articular fracture line at the base of the thumb metacarpal. This fracture is similar to the Bennett's fracture, but with more comminution and shortening; it is, therefore, a more severe injury.



Figure 6: Rolando's fracture. (From wikiradiography.com  
<http://bit.ly/1nZtcaM>)

Thumb metacarpal shaft fractures are treated similarly to the other four metacarpals.

## OBJECTIVE EVIDENCE

The following three X-ray views should be obtained in order to confirm a metacarpal fracture: posteroanterior (PA), oblique, and lateral. If a metacarpal head fracture is suspected but not seen well, special views (e.g., Brewerton view, in which the patient's hand is flexed at the metacarpophalangeal joint to 65 degrees) or CT scanning may be helpful.

## EPIDEMIOLOGY

Metacarpal fractures represent 10% of all fractures, and there is a lifetime incidence rate of 2.5%. The fifth metacarpal is fractured most frequently and accounts for one-fourth of all metacarpal fractures. Most of these fractures occur in the young, active, or working population and consequently result in a significant loss of revenue and work time each year.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of metacarpal fractures can be thought of as “bad,” “not so bad,” and “very bad.” A non-displaced shaft fracture may be “not so bad;” an angulated fracture sustained by punching a fellow in the face, with a resultant open wound could easily be “very bad.” To sort that out, the following features of the injury must be delineated:

1. The location of the injury (which bone, which region and articular involvement).
2. The pattern of the fracture (e.g. simple vs comminuted; transverse vs spiral).
3. Degree of displacement and rotational misalignment.
4. Status of the soft tissue, paying special attention to possible fight bite wounds.

## RED FLAGS

Malrotation of the fingers. Assessments of the patient's hand rotation can be made by asking the patient to fold their hand half way into a fist. The nails should align in parallel, facing the ceiling; fingertips should point to the scaphoid. The key is that rotation cannot be determined by examining an x-ray. It must be noted during the physical examination.

A punch against the face is at risk for a fight bite – a small wound that is replete with mouth flora (and subsequent infection). The bite mark may not be located near the metacarpophalangeal joint, but the injury may still have damaged the joint capsule. Look closely for this, as it requires prompt surgical intervention.

“Trivial” chip fractures may represent avulsions of the ligaments and a significant soft tissue injury. The size of the bone fragment does not necessarily correlate with the severity of the injury.

Profound hand swelling from a crush injury may produce a compartment syndrome.



## TREATMENT OPTIONS AND OUTCOMES

The three aims of treatment are preserving alignment, mobility and proper finger length.

The following factors should be accounted for to determine whether surgical (open reduction) or non-surgical (closed reduction) intervention is best:

- degree of angulation
- amount of displacement
- presence of excessive rotation

Other factors that might be considered are the patient's age and occupational/recreational demands.

The guidelines regarding how much angulation is tolerable has more to do with eminence-based medicine than evidence-based medicine. The medical eminences have said that up to 10 degrees of angulation can be accepted in the 2nd metacarpal with an additional 10 degrees for every finger moving in the ulnar direction, i.e., 3rd = 20 degrees; 4th = 30 degrees; and 5th = 40 degrees.

And speaking of "eminences": after a fracture, the prominence of the 5th metacarpal-phalangeal joint is apt to be lost – a "cosmetic," not functional loss — with closed treatment. Patients should be so warned.

If surgery is not needed, the patient's fracture should remain immobilized and they should be seen for follow-up in one to two weeks. X-rays should once again be taken to determine if there is proper healing and ensure no further displacement or angulation.

Immobilization of a metacarpal fracture must include at least the proximal phalanx, but there is no particular need to immobilize the inter-phalangeal joints and indeed that may lead to unnecessary stiffness. The metacarpal-phalangeal joint should be held in full flexion as long as the position doesn't further displace the fracture. Such a position will make the collateral ligaments maximally taut (because of the cam shape of the metacarpal head). This can help reduce the distal fracture fragment. This position can also prevent contractures of the joint. (If the ligament were not immobilized under maximal tension, it might set in this shorter position and lose its ability to stretch; subsequently, a joint contracture might form.) Within the cast, the affected finger should be buddy-taped to its neighbor. A vast majority of metacarpal neck fractures affect either the 4th or the 5th metacarpal. In those cases, the 2nd and 3rd fingers can be left free, to fully extend and flex.

When surgery is needed, options include pins, screws, metal wires, and plates (Figure 7). Open reduction of metacarpal head injuries demand an especially delicate touch, to avoid disruption of the blood supply and prevent avascular necrosis.



*Figure 7: Surgical treatment of metacarpal fractures. Options include pins, screws, and plates (though rarely all in the same hand).*

If only casting is needed, recovery should take about six to eight weeks. After three or four weeks of immobilization, the cast may gradually be removed and replaced with a removable splint. Radiographic reassessment should occur one week following the incident and at three week intervals after that. Limited range of motion exercises may be started after three weeks.

If surgical intervention is needed, recovery should take a few months, depending on the stability of the fracture and the care the patient takes to protect the hand from re-injury. The implants will remain in the hand unless they become bothersome to the patient.

In general, with proper treatment and rehabilitation, the prognosis for a metacarpal fracture is excellent. Bone healing begins around six weeks, but it may take years to return to full strength.

The duration of closed treatment has to be “just right.” Left on too long, a cast can cause joint contractures, yet casting for an inadequate period increases the risk for displacement and malunion.

## **RISK FACTORS AND PREVENTION**

Athletes who participate in sports, such as martial arts and football, have an increased risk of metacarpal fracture. Boxers frequently make closed fist contact with solid objects, while football players' hands are frequently exposed and unprotected, which may lead to high energy impact and crush injuries.

Males are three times more likely than females to experience a metacarpal fracture.

Furthermore, adolescents, those in the work force, and the physically active populations have a greater risk of metacarpal injuries.

## **MISCELLANEOUS**

A shortened fourth metacarpal may signify congenital XO abnormality (Turner's syndrome).

Boxer's fractures in non-boxers may be a sign of anger management issues and possible domestic violence (especially when the recited history is "I fell").

A boxer's fracture may suggest not only trouble controlling one's temper but poor boxing technique: ideally, the force of a punch should be transmitted via the more rigid 2nd and 3rd metacarpals. But don't tease the patient – he may slug you with the good hand.

## **KEY TERMS**

metacarpals, Bennett's fracture, boxer's fracture

## **SKILLS**

Be able to communicate orally the nature of the injury. Recognize "fight bite" fractures. Splint the fracture. Recognize which fractures might need surgical stabilization.

## OLECRANON BURSITIS

---

Olecranon bursitis (Figure 1) is a condition in which the soft tissue overlying the olecranon at the elbow is inflamed. Inflammation of the olecranon bursa can be the result of repetitive use, direct trauma or inflammatory conditions such as gout or rheumatoid arthritis. Occasionally, fluid in the olecranon bursae can become infected – a condition that requires surgical drainage. Often, however, olecranon bursitis runs an innocuous course.



*Figure 1: Olecranon bursitis.*

### STRUCTURE AND FUNCTION

The body places bursae (the plural of “bursa”) in those areas where the normal structures must glide (e.g., the skin overlying the olecranon) to facilitate that gliding by reducing friction.

The word “bursa” literally means “sack,” however, a healthy bursa has only a small amount of lubricating fluid within it. When bursitis is present, the inner surfaces of the bursa produce fluid, filling the bursa to become more sack-like.

## PATIENT PRESENTATION

The hallmark of olecranon bursitis is a mass at the elbow over the olecranon.

Tenderness, redness and warmth are classic signs, but may be absent. At times the bursa is not at all painful, unless pressure is applied.

Olecranon bursitis can be caused by conditions such as rheumatoid arthritis or gout, and therefore the presentation may be colored by co-existing symptoms of those conditions (for example, arthrosis within the elbow itself).

Patients on dialysis seem to be at particular risk for olecranon bursitis (for reasons that are not entirely clear).

At times, there is a history of direct trauma to the elbow or of repetitive use. At other times there is no antecedent history at all.

Even in cases where the bursa is not painful, pain can be produced by hyperflexing the elbow as this motion tends to increase the pressure within the bursa. Pain induced by flexion may impede full range of motion on examination.

## OBJECTIVE EVIDENCE

Fluid from olecranon bursitis can be drained and sent for culture. Culture is the definitive test, although the Gram stain may be negative in many cases that ultimately turn out to be infectious.

X-rays may show an olecranon spur. There may be calcification within the bursa itself. An MRI (Figure 2) can demonstrate the fluid collection but is rarely indicated.

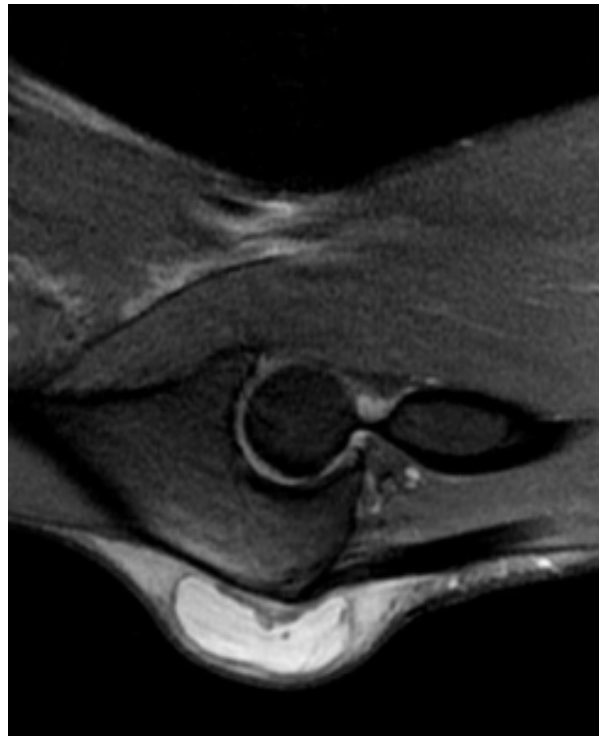


Figure 2: MRI of olecranon bursitis. (From radiopaedia.org  
<http://bit.ly/1NMuGtE>)

## **EPIDEMIOLOGY**

The precise prevalence of olecranon bursitis is not known. It is a common condition, and therefore high (if not first) on the differential diagnosis list of painless masses around the elbow. Approximately 20-25% of cases of bursitis turn out to be infectious. Approximately 5-10% of patients on dialysis may have this condition.

## **DIFFERENTIAL DIAGNOSIS**

There are two levels of diagnostic thinking required: First, “Is this mass bursitis?” Second, “If this is bursitis, what caused it?” (Also, the question, “Is this bursa infected or at risk for infection?” should be kept to the forefront.)

A mass overlying the elbow in the setting of normal radiographs (or radiographs showing only calcification within the bursa) can be considered bursitis until proven otherwise.

The causes of bursitis can usually be deduced from the history. As noted, increased redness, warmth and tenderness (especially in an immunocompromised patient) should prompt an infection work-up.

## **RED FLAGS**

Bursal fluid can serve as a culture medium and easily become infected. Approximately 25% of cases of olecranon bursitis will turn out to be infectious. Septic bursitis is heralded by more redness and warmth and is much more likely to be painful with even gentle, short-arc motion (also known as “micro-motion”) of the elbow. Patients on dialysis should be suspected of having septic bursitis.

Septic bursitis can be diagnosed definitively with an aspiration and culture. A cell count of the aspirate can be a useful clinical guide for initiating empiric treatment.

## **TREATMENT OPTIONS AND OUTCOMES**

The first line treatment of non-septic bursitis includes rest, compression (with ACETM wrap), ice, and NSAIDs. If infection is considered likely, aspiration should be performed and antibiotics started. If the infection does not clear after the first aspiration and appropriate antibiotics, surgical drainage may be needed.

Also, ensuring that any underlying process that may be responsible (e.g., gout) is well controlled is a wise step.

Minimizing the gliding across the bursa may help control the bursitis, but in general the elbow is apt to get stiff when immobilized; as such, prolonged immobilization should be avoided. Typically, immobilization with ace wrap will provide enough compression but will not completely immobilize the elbow.

If the first line treatment is not successful, needle aspiration (Figure 3), with or without a steroid injection, can be helpful. Aspiration can be diagnostic as well as therapeutic.



*Figure 3: Aspiration of olecranon bursitis. Note that the needle is inserted tangential to the joint to avoid penetration of the intra-articular space.*

Treatment of bursitis tends to be effective, yet recurrence is common. That is often the case because the inciting cause remains present, e.g., a worker continues to put pressure on the elbow, or the gout flares up again. In general, however, long-term impairment is rare.

## **RISK FACTORS AND PREVENTION**

People whose occupations require resting on the elbows – for example, carpet installers and gardeners – are best served with elbow pads.

Dialysis, and immune compromise in general, are risk factors for infectious bursitis. Vigilance to ensure early detection of infection in these patients is advised.

## **MISCELLANY**

The “bible” remedy used for ganglion cysts (smashing the mass with a heavy book) is a poor choice for olecranon bursitis. If nothing else, a heavy blow may create an open wound and increase the chance of infection.

The word “bursa” is similar to the word “bursar,” the official at a university who collects the tuition – and carries it away in money sacks. The simple English word “purse” has a similar root.

## **KEY TERMS**

bursa, bursitis, aspiration

## **SKILLS**

Recognize bursitis and associated “red flags.” Aspirate bursa under sterile conditions.

## PHALANGEAL (HAND) FRACTURE

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Phalangeal fractures of the finger are typically due to direct blows to the hand. Most phalangeal fractures are treated with a splint, but unstable fractures may require surgical treatment to prevent complications such as stiffness and malunion. Phalangeal fractures may be seen with other more serious injuries such as laceration of the nail bed or disruption of the flexor tendon. In those instances, treatment is directed more to the soft tissue injury.

### STRUCTURE AND FUNCTION

The phalanges form the fingers and thumb of the hand. The thumb has 2 phalanges (proximal and distal) and the other digits have 3 (proximal, middle and distal) for a total of 14.

Each phalanx is comprised of a base, proximally, and a head, distally, with the shaft between them. The articulation of the proximal and middle phalanges forms the proximal interphalangeal joint (PIP); and the articulation of the middle and distal phalanges forms the distal interphalangeal joint (DIP). These are hinge joints, capable of flexion and extension. The ulnar and radial collateral ligaments along with the joint capsule are the primary stabilizers. The volar plate, a band of fibrocartilaginous tissue, prevents hyperextension. The flexor and extensor tendons attach to bone at their insertion sites, and bone injuries there (typically avulsions) and lead to functional failure of the affected flexor or extensor tendon (Figure 1).

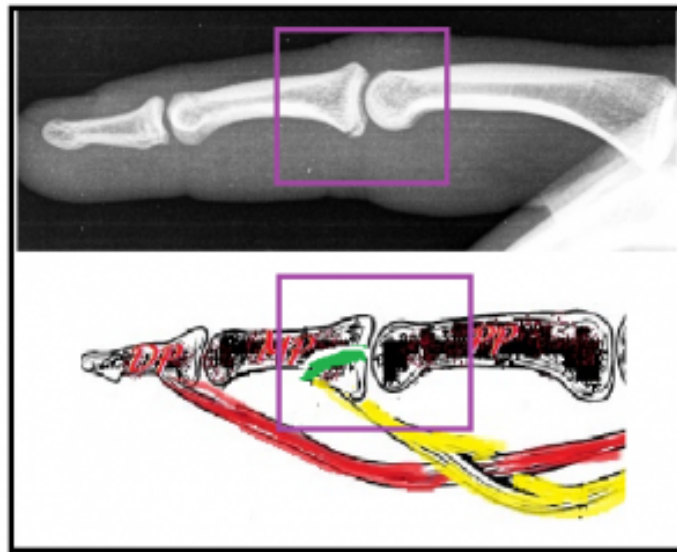


Figure 1: The superficial flexor tendon (shown in yellow in the drawing) can displace an intra-articular fragment, as shown. [The size of the fragment is exaggerated in the drawing for emphasis.] (From radiopaedia <http://bit.ly/1nA9GB7>)



## PATIENT PRESENTATION

Patients typically present with pain and swelling with a history of a blow or crush to the injured digit. Ecchymoses may be present (Figure 2).

Distal phalanx fractures are usually caused by a direct blow or crush, which can also cause nail bed injuries (seen as a hematoma beneath the nail).



Figure 2: Photo of a finger injury suggesting a possible fracture. (from flickr <http://bit.ly/1m8wpmE>)

Tuft fractures are fractures of the cancellous bone at the distal tip. The integrity of the flexor and extensor tendons should be assessed with any injury to the distal phalanx. Nailbed injuries (that require repair) commonly accompany tuft fractures, as do subungual hematomas (that may need to be drained).

The middle phalanx is more susceptible to a direct blow to the dorsal side. The middle phalanx is injured with an axial force, parallel to the shaft.

A direct blow commonly causes proximal phalanx fractures but rotational forces and hyperextension can also cause injury. Proximal phalanx fractures are often angulated at the time of presentation (independent of mechanism) as muscle forces deform the unstable shaft. The collateral ligaments and volar plate at the metacarpophalangeal (MCP) joint stabilize the proximal portion and the extensor tendon pulls the distal fragment into extension.

## OBJECTIVE EVIDENCE

Anteroposterior, lateral, and oblique images of each injured digit are needed. The lateral view should be taken so the other fingers do not obscure the injured digit. The oblique view can help diagnose fractures of the heads. Radiographs (Figure 3) should be examined for angulation, rotational alignment, and shortening; the presence of intra-articular extension should also be noted. Rotational deformities are usually diagnosed via physical examination rather than radiographic finding.

If an intra-articular fracture is suspected but not seen on plain radiograph, a CT scan can be useful.



Figure 3: Radiograph of an extra-articular fracture of the proximal phalanx of the 4th finger. (from radiopaedia.org <http://bit.ly/1KIVFkU>)

Once the fracture is reduced and splinted, post-splinting radiographs are taken. There should be no finger rotation, less than 2 mm of displacement or shortening, and less than 10 degrees of angulation.

## EPIDEMIOLOGY

Phalangeal fractures are common. They are seen in athletic and work-related injuries. The precise incidence is not known, as many patients do not seek medical care.

## DIFFERENTIAL DIAGNOSIS

Collateral ligament tears are seen when there is forced ulnar/radial deviation, especially in the PIP joint, (i.e., “jamming” of the finger). Applying gentle varus and valgus force to the joint can test the ligaments.

Soft tissue trauma can cause swelling within the (confined) fibrous septae found in the pulp of the distal phalanx; blood can thereby accumulate.

Fractures are also seen with complex skin lacerations and nailbed injuries, such as subungual hematomas.

Flexor or extensor tendon injury may be seen with middle and distal phalangeal fractures. Mallet finger is an extensor tendon injury from a flexion blow to an extended DIP joint. It is seen as an inability to actively extend the DIP, leading to a flexion deformity of the DIP joint. Jersey finger is a flexor tendon injury (flexor digitorum profundus) from forceful extension of the flexed DIP joint. Injury typically occurs when the finger is caught on a jersey, usually while tackling, and most commonly affects the fourth digit.

## RED FLAGS

Loss of rotational anatomic alignment (Figure 4). Comminuted oblique, and spiral fractures are more likely to have loss of rotational alignment.

Soft tissue wounds with fracture suggest possible open fractures with soft tissue injury.

Dysfunction of flexion or extension of each phalanx. This can signal a middle phalanx base fracture, as the base of the middle phalanx is the insertion site of the flexor digitorum superficialis (FDS) tendon.

Loss of two-point discrimination (normal is 4-5 mm wide) or capillary refill (normal is less than 2 seconds).



*Figure 4: Loss of rotational anatomic alignment due to proximal phalanx fracture.*

## TREATMENT OPTIONS AND OUTCOMES

The goal of treatment is to restore congruence of joint surfaces as well as anatomic alignment.

A majority of non-displaced fractures of the distal phalanx can be splinted with the DIP joint in extension for 3-4 weeks.

Most non-displaced proximal phalangeal fractures can be managed with a splint or even “buddy taping” (Figure 5).



*Figure 5: “Buddy taping” to treat a non-displaced proximal phalangeal fracture. (from wikiradiography <http://bit.ly/1NMv5fF>)*

Active range of motion should be started after 3 weeks, with clinical healing (no pain with palpation or movement) taking 6-8 weeks to occur. Ice, elevation, and limitations of use can aid healing.

If the nail is separated from the nail root, it should be gently irrigated and then placed beneath the eponychium (cuticle) in order to assist in future nail growth.

Referral to a hand surgeon is needed with middle phalanx base fractures (as this is the insertion site of the flexor digitorum superficialis tendon), comminuted, rotational, intra-articular, oblique, and spiral fractures. Displaced or angulated fractures unable to be reduced adequately need prompt referral.

Surgical options include extension block splinting with Kirschner wires (Figure 6), closed reduction and internal fixation (CRIF) and open reduction and internal fixation (ORIF).



Figure 6: Surgical fixation using a Kirschner wire. (from wikiradiography <http://bit.ly/1NMv5fF>)

The outcome of phalanx fractures depends on many factors, including the forces causing the injury, patient compliance, type of treatment, length of immobilization, and medical expertise.

Stiffness is one of the most common complications following phalangeal fracture, correlating with the amount of soft tissue injury and age of the patient. Some degree of stiffness occurs in greater than 50% of proximal phalangeal fractures.

Nonunion (failure of the fracture to heal) is rare, and in the rare cases where this is seen, the lack of full healing is typically not clinically significant.

Malunion (healing with deformity) is common, especially with nonoperative treatment of fractures that should have been treated surgically. Osteotomy is performed to correct the deformity.

Post-traumatic arthritis is a potential complication of intra-articular fractures.

## **RISK FACTORS AND PREVENTION**

Individuals playing ball sports (basketball, football, baseball) are at a high risk for fractures as well as those with bone/joint disease (osteoporosis) and poor nutrition. Elderly patients are at a higher risk due to their increased likelihood of falls.

## **MISCELLANY**

Wearing proper gloves and protective wear can decrease the chance of work-related phalangeal fractures.

The plural of phalanx is “phalanges” and the adjective is “phalangeal.” Etymology: The ancient Greek word “phalangos” means “line of battle,” referring to a tight array of 800 soldiers. The fingers, closely apposed, may be thought to resemble an infantry row – at least to the people who thought that the scaphoid looked like a boat, or that the coracoid process of the scapula resembled a raven’s beak.

## **KEY TERMS**

phalanges, phalangeal, fracture, distal, middle, proximal, tuft, nailbed, jersey, mallet, finger, proximal interphalangeal (PIP) joint, distal interphalangeal (DIP) joint, flexor digitorum profundus (FDP), flexor digitorum superficialis (FDS)

## **SKILLS**

Accurately diagnose fractures on radiographs. Understand and demonstrate testing of FDS and FDP. Apply a radial or ulnar gutter splint. Describe and demonstrate testing for radial and ulnar digital neuropathies. Be able to describe a phalangeal fracture with correct terminology.

## RADIAL HEAD FRACTURES

---

Radial head fractures are the most common type of elbow fractures in adults. Perhaps counter-intuitively, fractures of the radial head (which is part of the elbow) typically occur after a fall on an outstretched hand. The impact from the fall drives the radius proximally into the humerus, causing an injury at the elbow.

### STRUCTURE AND FUNCTION

The radial head articulates with both the capitellum of the humerus and the ulna. The radial head is concave, matching the convex surface of the capitellum of the humerus (Figure 2). The radial head translates on the capitellum during elbow flexion-extension, and pivots on the capitellum during supination-pronation. Just distal to the radial head lies the annular ligament, which holds the radius to the ulna. This ligament holds the radius as it rotates, but also allows for some translation as well. (The central axis of the radius is not perfectly cylindrical, and thus during pronation, the radius must be able to translate a bit as well). The radial head also ultimately connects to the carpus, as it is the base on which the radius itself is supported. In particular, loss of the radial head may cause *wrist* symptoms, as such loss may cause proximal migration of the radius with additional load now placed on the proximal ulna.

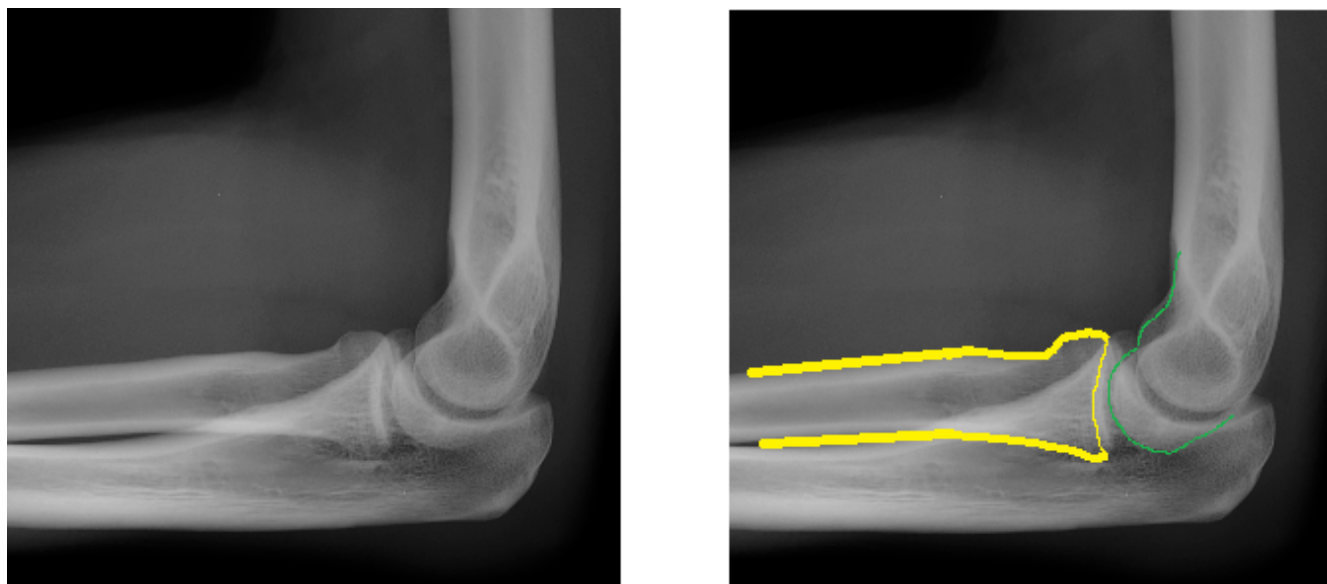


Figure 1: Radiograph of a normal elbow, left, with annotation on right showing the radius (yellow) articulating with the capitellum of the humerus (green). (Modified from Wikipedia <http://bit.ly/203iaNm>)

### PATIENT PRESENTATION

Patients with an injury to the radial head typically present with a history of a fall on an outstretched hand, or, following higher energy trauma and elbow dislocation. Pain, effusion over the elbow, and limited range of motion at the elbow and forearm are common symptoms. There is typically localized tenderness over the radial head on palpation; passive rotation of the forearm is also painful.

Intra-articular bleeding from the fracture may produce a palpable effusion. Aspiration of the effusion may assist with diagnosis and provide pain relief, thereby allowing faster and more effective rehabilitation.

## OBJECTIVE EVIDENCE

Radiographs must be obtained in the case of suspected elbow fracture; standard anteroposterior (AP) and lateral films of the elbow and of the wrist usually suffice. The distal radio-ulnar joint should be assessed on the lateral film for dislocation. A fracture of the radial head with concomitant dislocation of the distal radio-ulnar joint is called an “Essex-Lopresti fracture.”

Oblique views with the forearm in neutral rotation, so-called Greenspan views, show the radiocapitellar articulation and may be useful in the case of a suspected fracture that is not visible on AP or lateral films. Additionally, a positive fat pad sign on a lateral view indicates fluid in the joint, which in the acute setting is usually blood suggestive of a fracture (Figure 2).

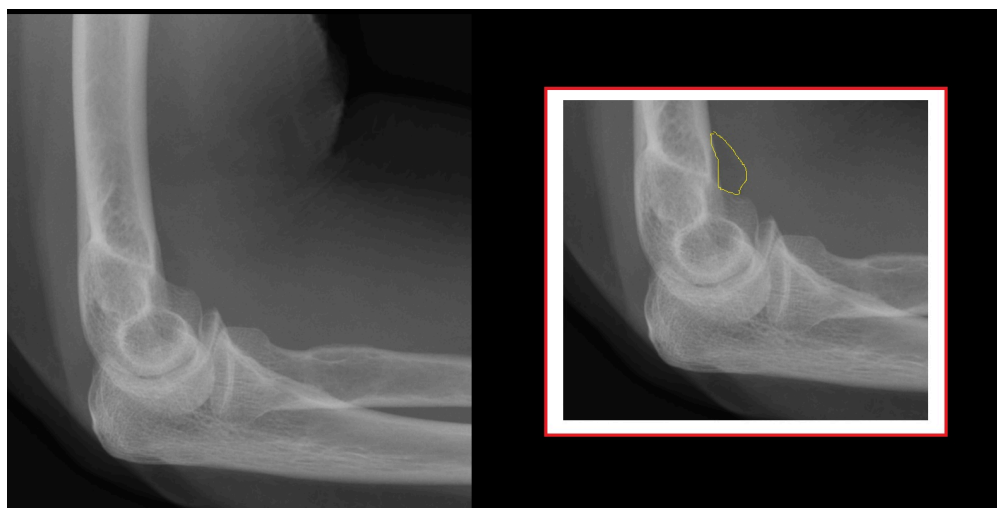


Figure 2: Anterior fat pad, annotated in yellow on right.

CT scanning may be used for preoperative planning, especially in the case of fragment displacement or comminution.

MRI may be used to assess for possible osteochondral injuries of either the radial head itself or, more commonly, of the capitellum. (This is usually reserved for patients with persistent pain after a period of immobilization.)

Although radial head fractures are not typically associated with osteoporosis, it may be prudent to assess bone density in middle-aged women who present with radial head fracture.

## EPIDEMIOLOGY

One-third of all elbow fractures involve the radial head. The male-female ratio roughly 1:1. Men who sustain radial head fractures tend to be younger than women with the same fracture. That may be because men usually have a high-energy mechanism of injury such as a fall from height or sports injury, whereas women, who tend to have lower-energy injuries, sustain the fracture due to inherent bone fragility.

## DIFFERENTIAL DIAGNOSIS

Associated injuries, which should be ruled out when a radial head fracture is suspected or confirmed, include fractures of the capitellum, fractures of the distal radius, dislocation of the distal radio-ulnar joint (the so-called

Essex-Lopresti fracture), rupture of the medial collateral ligament (MCL) causing valgus instability, rupture of the triceps tendon, and elbow dislocation. Radial head fractures are known to present in combination with MCL ruptures and coronoid process fractures, a constellation known as the “terrible triad.”

## RED FLAGS

Limited range of motion of the elbow may be due to a hemarthrosis (which should be aspirated to allow evaluation of passive range of motion as well as for pain relief) but this limitation could also reflect a displaced fragment.

As noted above, the same mechanism that causes a radial head fracture (a fall on the outstretched hand, typically) can also cause other injuries at the elbow, including humeral fractures, medial collateral ligament sprains, or even triceps ruptures (akin to a quad tendon rupture). As such, a radial head fracture seen on radiograph is itself a “red flag” for an associated injury at the elbow and wrist. Therefore, the clinician must be sure that radiographs visualize both elbow and wrist joints.

A fracture from what appears to be a low-energy mechanism in an elderly woman may suggest osteoporosis.

## TREATMENT OPTIONS AND OUTCOMES

The goals of treatment are to correct any inhibition of forearm rotation, to restore stability of the elbow and forearm, and to allow early motion.

The Mason classification of radial head fractures is useful for determining treatment options.

A Mason Type 1 fracture is a nondisplaced fracture with no mechanical blockage to forearm rotation (Figure 3). These fractures can be treated with a sling and early motion after 24-72 hours (indeed, as soon after injury as discomfort subsides). Aspiration of hemarthrosis in the radiocapitellar joint with local injection of anesthetic can relieve pain and aid in early motion. Sometimes a long arm splint for a few days is helpful for pain relief.

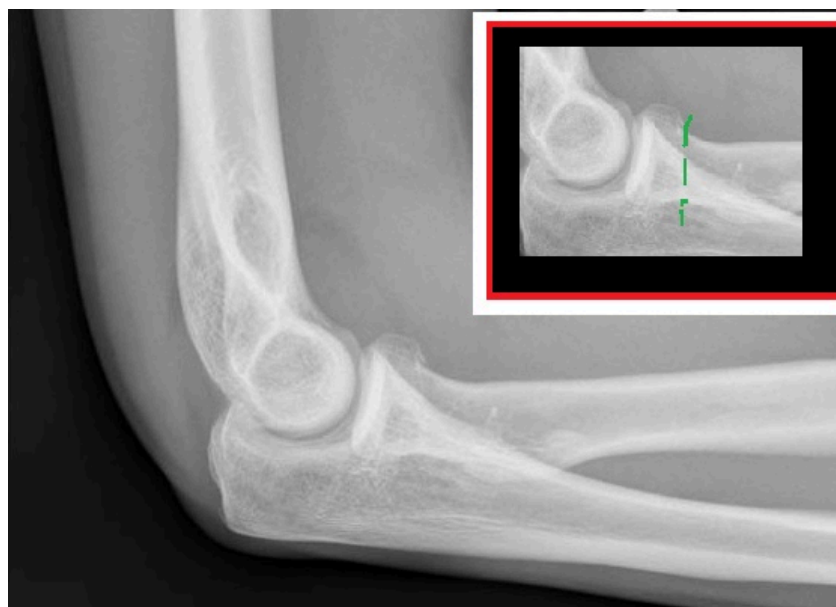


Figure 3: Radiograph of Mason Type 1 radial head fracture (fracture line shown in green). (from radiopaedia <http://bit.ly/1Q7URwP>)

If there is mild displacement, angulation, impaction, or depression of the fracture (but still no mechanical block to forearm rotation), the fracture is a Type 2. These fractures typically do best if fixed surgically. The type of internal fixation will vary depending on fracture pattern and extent. Screws may be sufficient for a



partial articular fracture, whereas a plate may be required for fractures that extend into the radial neck. Surgical fixation works best if the fracture has 3 or fewer articular fragments; if there are 4 or more, even though the fracture is still “type 2,” excision and not fixation may be needed.

Fractures with significant displacement, angulation, impaction, or depression, or with mechanical blockage are classified as Type 3. For most of these, radial head excision with prosthetic replacement is recommended.

Radial head excision alone, without placement of a prosthetic head, obviates the need to wait for bone healing, but may lead to symptomatic proximal migration of the radius. The interosseous ligament between the radius and ulna may prevent some migration, but the absent “base” to the radius may make such migration inevitable.

A Mason Type 4 is a radial head fracture with an elbow dislocation and care is directed first at restoring the joint: patients with this injury should be sent to the Emergency Ward for urgent care.

Stiffness or contracture may occur secondary to prolonged immobilization of the elbow; therefore, it is essential to start active motion as early as possible. Pain, swelling, and inflammation may be hindering motion and should be investigated further for unrecognized injury. A supervised therapy program may maximize outcomes.

Chronic wrist pain may be the result of an unrecognized injury to the distal radio-ulnar joint (DRUJ), interosseous ligament, or triangular fibrocartilage complex (TFCC).

The posterior interosseous nerve (PIN) is vulnerable to injury during operative treatment. PIN neuropathy is a motor syndrome that results in wrist and finger drop.

Proximal radial migration may occur after radial head excision in the case of unrecognized unstable fracture-dislocations such as an Essex-Lopresti injury. (This migration, it should be noted, will occur only if there is a lesion of the interosseous membrane as well.)

Other possible complications include malunion, non-union, avascular necrosis, heterotopic bone formation, complex regional pain syndrome, and posttraumatic radio-capitellar osteoarthritis.

## **RISK FACTORS AND PREVENTION**

The link between osteoporosis and radial head fractures is still being investigated. The fact that women over 50 years old tend to sustain radial head fractures in low-velocity falls is suggestive of a correlation with osteoporosis. If a strong correlation does exist, women over 50 should be offered screening for osteoporosis to prevent osteoporotic fractures.

## **MISCELLANY**

In one study by Duckworth, *et al.*, a trend was observed toward increased incidence of radial head fractures in patients with a lower socioeconomic status.

Note that the radial head and neck are located in the proximal forearm, at a point a lay person might identify as the “foot” of the bone.

## **KEY TERMS**

radial head fracture, elbow, fracture-dislocation, stability, early range of motion

## **SKILLS**

Recognition and description of radial head fractures on radiographs.

## SCAPHOID FRACTURES

---

The scaphoid (also known as the carpal navicular) is perhaps the most important of the eight carpal bones. The scaphoid links the carpal bones closest to the radius and ulna, the so-called the “proximal row,” to those articulating with the metacarpals, the “distal row.” Owing to its tenuous blood supply, the scaphoid is particularly susceptible to osteonecrosis (and ensuing post-traumatic arthrosis) after fracture. Unfortunately, the scaphoid is also the most commonly fractured of the eight carpal bones (Figure 1). Fracture is usually caused by a fall on an outstretched hand (a mechanism also associated with distal radius fractures and radial head fractures).

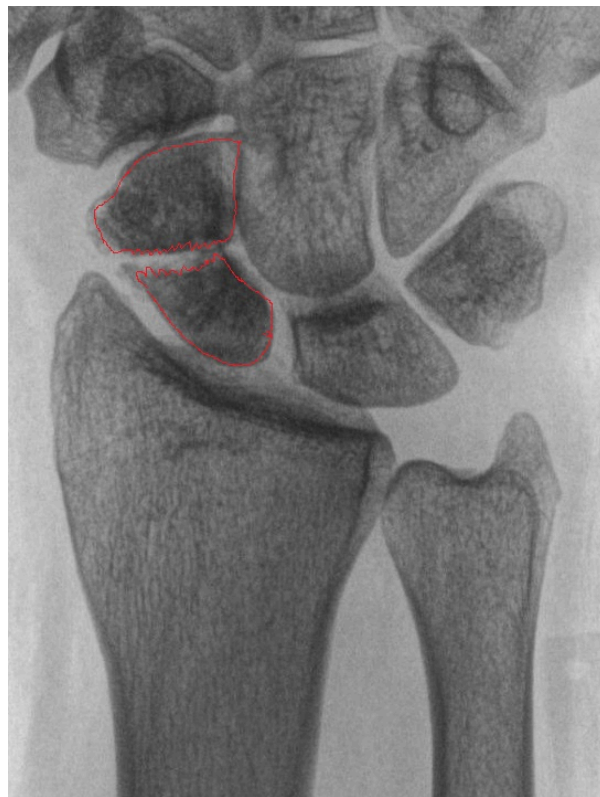


Figure 1: Scaphoid fracture. (modified from [wikimedia.org](http://bit.ly/1Q7URwP)  
<http://bit.ly/1Q7URwP>)

## STRUCTURE AND FUNCTION

The scaphoid can be morphologically divided into three anatomic regions: proximal third (proximal pole), central third (waist), and distal third (distal pole). A tuberosity on the distal palmar aspect serves as an attachment site for the transverse carpal ligament. Nearly 2/3 of all scaphoid fractures occur at the waist, with 15% occurring at the proximal pole, 10% at the distal pole, and 8% at the tuberosity.



Figure 2: The scaphoid outlined in red.

The scaphoid (Figure 2) articulates with the radius and four carpal bones (lunate, trapezium, trapezoid, and capitate). To accommodate so many articulations, 80% of the scaphoid is covered with cartilage.

The scaphoid is supported by the radioscapholunate ligament which stabilizes the scapholunate articulation and acts as a neurovascular conduit. The scaphoid does not have any tendinous attachments.

The scaphoid receives its primary blood supply from the dorsal scaphoid branches of the radial artery, which enter the scaphoid at a point near the distal pole. As such, the scaphoid is perfused in the distal to proximal direction (“retrograde blood supply”).

Because of this retrograde blood supply, an injury to the scaphoid waist (i.e. the middle third of the bone) can interrupt blood supply to the proximal pole. (This is analogous to femoral neck fractures and resulting femoral head ischemia: just as the vessels to the femoral head must traverse the femoral neck, the blood supply to the proximal pole of the scaphoid must traverse the waist).

## PATIENT PRESENTATION

Patients with scaphoid fractures will often present with radial sided wrist pain (worsened with gripping or squeezing), variable swelling and limited range of motion. Because scaphoid fractures may occur without bruising or visible deformity, patients may assume a less serious injury and delay seeking evaluation.

During examination, it is important to always compare the injured to the uninjured wrist.

Check if patients have tenderness to palpation of the “anatomic snuffbox” and over the scaphoid tubercle (Figure 3).

The anatomic snuffbox is located in a triangle formed by the extensor pollicis longus tendon, abductor pollicis longus tendon, and the radial styloid. It can be found more easily when the thumb is actively extended. Anatomic snuffbox pain has a sensitivity of 90% and specificity of 40% for detection of scaphoid fractures.

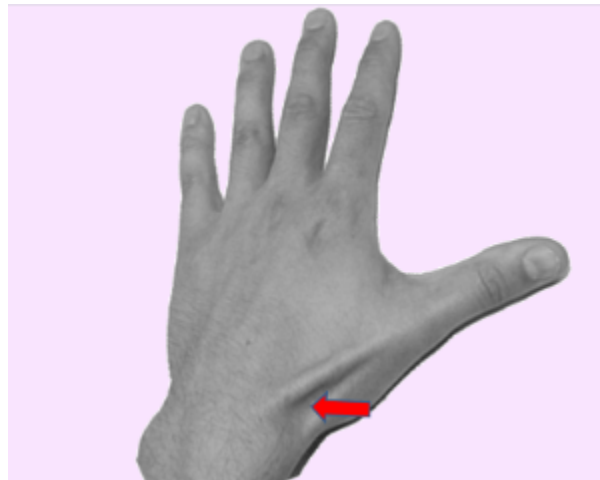


Figure 3: The surface anatomy of anatomic snuff box.

The scaphoid tubercle can be localized on the volar aspect of the wrist (Figure 4). Pain to palpation is a slightly less sensitive but more specific finding for detection of scaphoid fractures.

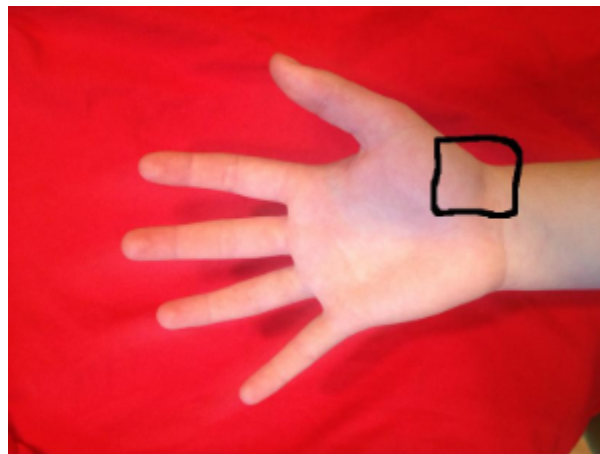


Figure 4: The surface landmarks of the scaphoid tubercle.

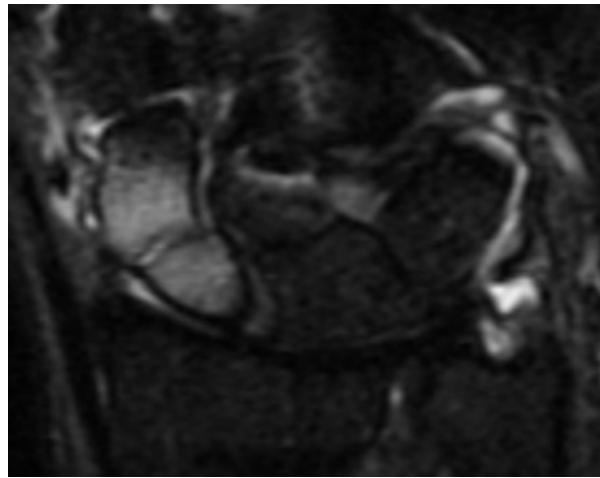
## OBJECTIVE EVIDENCE

Plain radiographs are essential for all patients with suspected scaphoid fractures. Standard radiographs include anteroposterior (AP), lateral, oblique and scaphoid views. The scaphoid view is a posteroanterior (PA) view of the wrist, with the wrist positioned in 45 degrees of ulnar deviation and pronated obliquely at 45 degrees.

Because initial radiographs of a scaphoid fracture may appear normal, patients with pain in the snuff box and a fall on the outstretched hand should be immobilized and sent for additional imaging studies.

One approach would be to obtain repeat radiographs in 10 to 14 days. At that point, the absence of fracture line or healing response on x-ray, coupled with an absence of tenderness on exam reasonably excludes the diagnosis of fracture.

Another approach would be to obtain an MRI. This test can detect an occult scaphoid fracture immediately, as well as any other associated injury.



*Figure 5: MRI showing a radiographically occult scaphoid fracture.  
(from radiopaedia <http://bit.ly/1Q7URwP>)*

Although bone scans (scintigraphy) can also be used to detect occult scaphoid fractures, MRI has two advantages (Figure 5). First, an MRI can be used immediately. (Bone scans, which assess a biological reaction that takes days to mount, can be falsely negative in the first few days.) Second, as noted, MRI can detect associated injuries as well. (The great sensitivity of MRI can impose costs too, as it may detect a clinically insignificant abnormality as well, prompting unnecessary worry to say nothing of unnecessary treatment.)

Note that MRI is still an expensive imaging modality in many locations. In the United States, at least, the use of MRI is advocated nonetheless, as the true price of MRI is not that much more than plain radiographs. Also, placing a patient in a cast for 10 days while awaiting the second round of radiographs represents a significant delay in treatment if a fracture does indeed exist.

## **EPIDEMIOLOGY**

The scaphoid is the most commonly fractured carpal bone. Fractures of the scaphoid often occur in physically active individuals, and have two to four times greater incidence in men than woman. The most common mechanism of injury is a fall onto an outstretched hand.

The true incidence of scaphoid fractures has been difficult to calculate particularly because many of these injuries are undiagnosed. One estimate is that scaphoid fractures represent 1 out of 100,000 people per year.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of suspected scaphoid fracture includes:

- Radial styloid fracture. Here, the tenderness is more proximal; radiographs should make the diagnosis.
- Distal radius fracture. Tenderness localizes to distal radius and deformity may be present; radiographs should make the diagnosis.
- Scapholunate instability. In this condition, the tenderness is more toward the ulnar side of the hand (over the scapholunate ligament) and radiographs should show a gap between scaphoid and lunate (scapholunate dissociation: the Terry Thomas sign).
- Flexor carpi radialis tendon injury. This can be diagnosed by noting pain elicited with resisted wrist extension.
- De Quervain's disease. A positive Finkelstein's test may help make the diagnosis.
- Carpometacarpal joint arthritis. Here, radiographs should make the diagnosis, but recall that a patient with arthritis can suffer a superimposed injury.

As noted, fracture of the scaphoid is usually caused by a fall on an outstretched hand, the same mechanism associated with distal radius fractures and radial head fractures. The astute clinician must be mindful of these potentially associated injuries.

## RED FLAGS

A fall on the outstretched wrist is itself a "red flag" and should raise suspicion for a scaphoid fracture. When there is tenderness in the anatomic snuff box, even if radiographs appear negative, a scaphoid fracture is even more likely.

## TREATMENT OPTIONS AND OUTCOMES

The patient with tenderness in the anatomic snuff box and negative x-rays should be immobilized in a thumb spica splint (Figure 6) and additional imaging should be obtained. The additional imaging can take the form of either repeat radiographs about 2 weeks after the injury, bone scintigraphy or MRI. This initial immobilization will minimize the chance that a non-displaced occult fracture (i.e., one not seen on x-ray) becomes a displaced fracture, and in turn, minimizes the risk of disrupting the blood supply.



Figure 6: A thumb spica splint.

Non-displaced fractures are defined as fractures with less than 2mm of displacement and no fracture site angulation. These types of fractures are treated by immobilization in a long or short arm thumb spica cast for 6 weeks. (The inclusion of the thumb is advised; whether a long arm (vs short arm) cast is needed is perhaps a bit more controversial. Including the elbow will minimize pronation/supination.)

A small (n=25) prospective, randomized study comparing cast immobilization with percutaneous cannulated screw fixation of nondisplaced scaphoid fractures found that screw fixation resulted in faster radiographic union (7 weeks vs 12) and return to military duty (8 weeks vs 15).

Comminuted, displaced or unstable scaphoid fractures benefit from surgical fixation. The precise indications for surgery (that is, how much linear or angular displacement is enough?) are not known.

The favored technique is open reduction internal fixation with screws (Figure 7). The choice of fixation depends on a multitude of factors, such as the location of the fracture, configuration of the fracture, functional concerns, and patient factors.



*Figure 7: X-ray of scaphoid after open reduction and internal fixation with a Herbert screw. (from radiopaedia <http://bit.ly/1Q7URwP>)*

Most scaphoid fractures treated non-surgically heal without difficulty, with the occurrence of nonunion reported between 5% and 15%.

Scaphoid fractures (especially if not recognized and therefore not treated) can result in post-traumatic arthritis. Once arthritis is evident at the radiocarpal joint there are salvage surgical procedures that can be performed. These include a proximal row carpectomy, scaphoid excision and midcarpal arthrodesis, or a total wrist arthrodesis.

## **RISK FACTORS AND PREVENTION**

Scaphoid fractures typically occur in active adolescents and young adults with an incidence two to four times higher in men than in women. The fracture commonly occurs as a result of extreme dorsiflexion of the wrist from a fall.

Short of telling all young, active males to not fall (and getting them to comply!), it is difficult to prevent these fractures. The key point in treating this injury is to prevent non-displaced fractures from displacing, as displacement makes osteonecrosis much more likely.

## MISCELLANY

Scaphoid derives from the Greek word *skaphos*, which means “a boat.” The English word “skiff” has a similar origin. The scaphoid is also referred to as the carpal navicular (coming from *navis*, the Latin word for “boat,” from which we get the English word “navy,” for example). Given that there is also a boat-like bone in the foot, it is probably best to refer to the bone in the hand as the scaphoid and the one in the foot as the navicular.

Regardless of length of immobilization after a scaphoid fracture, appropriate physical and occupational therapy is needed after removal of the cast or splint to optimize range of motion and function.

Smoking is strongly associated with nonunion after surgical fixation of the scaphoid.

## KEY TERMS

scaphoid, fracture, non-union, avascular necrosis, anatomic snuffbox, scaphoid compression test

## SKILLS

Perform physical examination to recognize occult scaphoid fractures. Be able to explain to patients why immobilization is needed despite “normal” x-rays. Apply a thumb spica splint to temporarily immobilize the scaphoid while awaiting definitive treatment.



## TRIANGULAR FIBROCARILAGE COMPLEX (TFCC) INJURY

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The triangular fibrocartilage complex (TFCC) is a soft tissue structure covering the distal ulna at the wrist, which serves to help stabilize the wrist and transmit load across the wrist joint. Because of its anatomic complexity and the forces that it experiences, the TFCC is at risk for both direct injury and degenerative damage. As such, TFCC pathology is a common cause of ulnar sided wrist pain.

### STRUCTURE AND FUNCTION

The TFCC is located at the ulnar wrist, articulating with the head of the ulna proximally and the lunate and triquetrum distally.

The TFCC can be divided into three components (Figure 1):

- the triangular fibrocartilage disc itself;
- the radio-ulnar ligaments; and
- the ulnocarpal ligament complex.

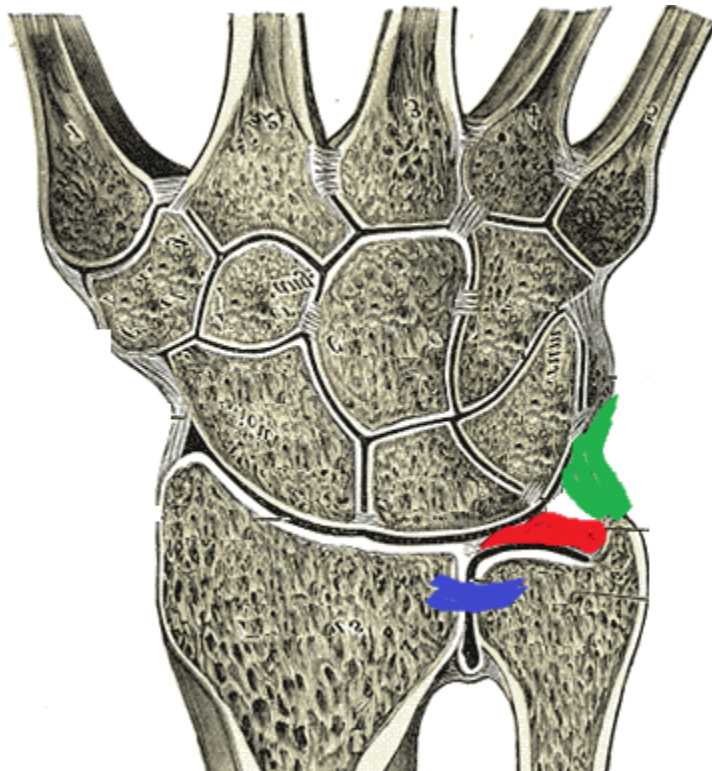


Figure 1: Drawing from Gray's Anatomy, showing the normal TFCC in situ: the triangular fibrocartilage disc is shown in red; the ulnocarpal ligament complex is in green and the radio-ulnar ligament in blue. (modified from wikipedia <http://bit.ly/1Q7URwP>)

The triangular fibrocartilage disc receives and distributes loads to the ulna from the carpus, much like the way the meniscus of the knee receives and distributes force from the femur. Without a TFCC, the ulna is only able to absorb half the force it usually receives from the hand. The loss of the TFCC results in a greater load placed on the radius.

The radio-ulnar ligaments are the primary stabilizers of the distal radioulnar joint.

The ulnocarpal ligament complex prevents dorsal migration of the distal ulna relative to the lunate and triquetrum, to which it attaches.

The vascular supply of the TFCC is limited to the peripheral 10-40% of the volar, ulnar, and dorsal TFCC only. The relative hypovascular nature of the central portion of the TFCC precludes healing of injury in this region, and may necessitate surgical excision.

## **PATIENT PRESENTATION**

Acute injuries of the TFCC result from axial and torsional forces applied to the extended wrist: in short, most often from falling on an outstretched hand. (Falling On an OutStretched Hand is a common mechanism for a variety of injuries and goes by the acronym FOOSH.)

Failure of the TFCC may be found in association with a distal radius fracture (which also comes about from force applied to the extended wrist).

Patients with TFCC injuries may present with chronic complaints of ulnar-sided wrist pain. Often, symptoms are aggravated with axial loading, particularly with the wrist in extension, or torsional activities against resistance such as wringing out a washcloth or turning a screwdriver. With progression, even simple activities such as pouring water from a pitcher may cause pain.

Pain or tenderness is localized to the ulnocarpal joint and may be provoked with ulnar impaction testing, or loading through an extended wrist such as with pushing oneself up from a chair. Patients can also complain of pain in the ulnar wrist in supinated and/or pronated positions.

While the majority of TFCC injuries do not involve instability of the wrist, failure of the distal radioulnar ligaments may cause instability of the distal radioulnar joint (DRUJ). This instability may cause pain and mechanical symptoms of “popping” or subtle signs of “giving way.” There may be visible signs of instability with prominence of the ulnar head relative to the distal radius – often, it is useful to compare the appearance of both the injured and non-injured wrists for evidence of asymmetry.

Gross instability of the DRUJ may be recognized with stress testing of the DRUJ in positions of maximal pronation and supination.

## **OBJECTIVE EVIDENCE**

For patients with a suspected TFCC injury, advanced imaging studies may be useful in assessment of the ulnar wrist.

Plain radiographs can show the congruity of the DRUJ and allow measurement of the ulnar variance (Figure 2). (See “risk factors,” below.)



Figure 2: X-ray showing positive ulnar variance. (from radiopaedia <http://bit.ly/1Q7URwP>)

Arthrography, or, better still, an MRI (or MR-arthrogram) may help detect ulnar wrist pathology. The sensitivity and specificity for detecting a full-thickness TFCC tear are approximately 85% and 95%, respectively, for MR-arthrography.

The gold standard for assessing the integrity of the TFCC is arthroscopy. This is an invasive test requiring anesthesia.

Injuries of the TFCC were broadly classified by Palmer as traumatic and degenerative, with traumatic lesions further sub-classified by their location and degenerative lesions sub-classified by the degree of associated conditions (for example, ulnar chondromalacia or ulnocarpal arthritis).

## EPIDEMIOLOGY

Attritional tears or degenerative pathology involving the TFCC are common, with a reported incidence of tears in greater than 50% of those over the age of 60 years. (Such chronic alterations in TFCC may become symptomatic after minor wrist trauma. On the other hand, the high prevalence of asymptomatic morphological change within the TFCC makes it unwise to necessarily attribute ulnar-sided wrist pain to a structural abnormality seen on an imaging study.)

It is estimated that TFCC injury occurs in up to 80% of displaced distal radius fractures.

## DIFFERENTIAL DIAGNOSIS

It is important to determine whether the symptoms are intrinsic to the wrist, or whether they might be referred to the wrist from other structures.

Intrinsic causes of ulnar wrist pain which might mimic symptoms associated with a TFCC include:

- Arthritis
  1. DRUJ
  2. ulnocarpal joint
  3. pisotriquetral joint
- Chondral injury (e.g., acute, ulnar impaction)
- Fracture:
  1. hook of hamate
  2. pisiform
  3. distal radius
  4. distal ulna
  5. triquetrum
- Ganglion cyst
- Kienböck's disease (avascular necrosis of the lunate)
- Inter-carpal ligament injury
- Extensor carpi ulnaris tendonitis/pathology

Extrinsic causes include cervical radiculopathy, ulnar neuropathy at the elbow, tendinopathy of either the extensor or flexor carpi ulnaris. Rarely, a nerve tumor (schwannoma) or ulnar artery thrombosis is the cause.

The correct inference from the extensive lists above is that TFCC pathology overlaps with many other conditions and therefore may be blamed when other conditions are responsible.

## RED FLAGS

Acute traumatic injury to the wrist requires a full evaluation, including the elbow. Although chronic ulnar-sided wrist pain does not have any “red flags” *per se*, the failure to examine and image “one joint above and one joint below” the focal area of complaints can lead to a misdiagnosis.

## TREATMENT OPTIONS AND OUTCOMES

Management of TFCC pathology should be guided primarily by the severity of symptoms. Other important considerations include the acuity and the location of the injury, and the response to initial treatment.

Most injuries of the TFCC may be treated non-operatively: activity modification, immobilization, and analgesics are the first lines of treatment. Immobilization is typically done above the elbow with limitation of pronation/supination of the wrist, i.e., long arm removable splint or Munster cast.

For persistent symptoms, corticosteroid injection of the ulnocarpal joint or therapeutic modalities may be considered.

The potential and indications for TFCC repair are influenced by the vascular anatomy of the TFCC. Acute or sub-acute injuries to the peripheral TFCC may be amenable to arthroscopic debridement and repair (arthroscopic +/- limited open repair) whereas central TFCC injuries may be treated with arthroscopic debridement when conservative management has failed to relieve symptoms.

Occasionally, when arthroscopic debridement does not provide adequate resolution of symptoms, an ulnar shortening osteotomy (to create negative ulnar variance and in turn decrease ulnar load) may be needed.

In cases with continued pain and disability despite appropriate treatments, various salvage procedures may be considered, such as joint stabilization, joint unloading or decompression, distal ulna resection, arthrodesis, and arthroplasty.

In a case series report examining open repair to treat triangular fibrocartilage injury, excellent or good results were seen in 26 of 33 patients.

In another report, among 28 patients with TFCC tears who were debrided (and not repaired), excellent results in 13, good in 8, fair in 2, and poor in 5 were reported in one study. The authors there concluded that arthroscopic debridement of TFCC tears is “warranted.”

## RISK FACTORS AND PREVENTION

Ulnar variance is determining by comparing the lengths of the distal radius and ulna on a standard PA x-ray (Figure 3). Neutral variance is found when the distal ulnar and radial articular surfaces are at the same level. In positive variance, the ulna projects more distally and in negative variance, the ulna projects more proximally.



Figure 3: Schematic of ulnar variance: neutral (left); positive (center) and negative (right).

The significance of positive ulnar variance is that the distal ulna and TFCC are subjected to greater loading. Hence, positive ulnar variance is a risk factor for TFCC injury. (By contrast, negative variance loads the radio-carpal joint more, and in turn is a risk factor for avascular necrosis of the lunate (Kienböck's disease).

## MISCELLANY

The term “conservative treatment” should not be used synonymously with “non-operative treatment.” There is nothing conservative about deferring surgery for a repairable TFCC tear, especially when such a delay facilitates the transformation of that repairable tear into an irreparable tear.

Some people similarly refer to the “TFCC complex.” This is an instance of the “RAS Syndrome,” a (self-referential) acronym for the Redundant Acronym Syndrome syndrome in which the word denoted by the last letter of an acronym is repeated unnecessarily. Other examples include “ATM machine” or “HIV virus.”

## KEY TERMS

TFCC, triangular fibrocartilage complex, ulnocarpal joint, ulnar wrist

## **SKILLS**

Create a comprehensive differential diagnosis of ulnar sided wrist pain and perform physical examinations to diagnose TFCC.

## TRIGGER FINGER

Stenosing tenosynovitis, also known as “trigger finger,” is a condition in which the flexor tendons of the fingers may get caught within their sheaths thereby limiting movement and causing pain. When the tendons are caught, the finger can get stuck temporarily in a flexed position. When the finger is then forced into full extension, a painful snap or click is felt, hence the name trigger finger. This condition is sometimes known as “stenosing tenosynovitis,” as the common lesion seen in all forms of triggering is a relative narrowing (stenosis) of the space for the tendon relative to the size of the tendon itself.

### STRUCTURE AND FUNCTION

Finger flexion is powered by the flexor digitorum profundus (for the distal interphalangeal, or DIP, joint) and the flexor digitorum superficialis for the proximal interphalangeal (PIP) joint. These tendons travel together in the palm and ascend the fingers through a fibro-osseous canal along each digit (Figure 1). This canal is formed by the metacarpals/phalanges, and by a pulley system and tendon sheath. The pulley system keeps the flexor tendons snug to the bone, preventing “bowstringing” during active flexion and converting a linear force into rotation and torque at the finger joints.

When this canal is abnormally tight (that is, “stenotic”) the tendons may get stuck. Stenosis may result from either thickening of the tendon (with or without nodule formation) or by thickening of the tendon sheath, thereby reducing the relative volume of the canal.



Figure 1: Drawing of the flexor tendons in profile. The tendons are held close to the bone by a system of pulleys (only one of which is shown here in green). If there is a nodule (shown here in red) within the flexor tendon, it might be unable to pass through the pulley. Thus, the finger can get stuck in flexion.

Flexion of the proximal phalanx, particularly with power grip, results in high loads at the distal edge of the most proximal (A1) pulley. The pulleys respond to this load with growth and become hypertrophic. A nodule may develop on the flexor tendon, likely in response to abrasion with the tendon sheath. Hypertrophy or module formation may cause a mismatch between the size of the tendon and the tendon sheath through which the tendon must slide. The mechanical impingement of the flexor tendons as they pass through a narrowed A1 pulley results in the phenomenon of trigger finger. The flexor mechanism in the hand is usually strong enough to overcome the narrowed tendon sheath, but the extensor mechanism generally is not as strong. Hence, the affected finger may become locked in flexion rather than extension.

The term “stenosing tenosynovitis,” in particular the *-itis* suffix, may not accurately reflect the pathophysiology of the condition. There is stenosis, but stenosing tenosynovitis is not an inflammatory condition per se. Inflammation, when seen, is part of the body’s correct (but perhaps too exuberant) response to a stimulus.

The most common etiology of trigger finger is idiopathic – that is, for no known reason. Secondary trigger finger can be seen in patients with collagen vascular disorders and inflammatory disorders including rheumatoid arthritis, amyloidosis, gout, diabetes, hypothyroidism, and sarcoidosis. Secondary trigger finger is associated with a worse prognosis.

## **PATIENT PRESENTATION**

Trigger finger can present as an acquired condition in adulthood or as a congenital condition in children.

In children (<2 years of age), triggering most commonly occurs in the thumb and is then known as “trigger thumb.” Congenital trigger thumb typically presents with the thumb interphalangeal joint locked in some flexion and a palpable nodule at the metacarpophalangeal (MCP) flexion crease.

In adults, mild cases of trigger finger can present with a sense of finger stiffness, particularly in the morning. With more advanced cases, patients will commonly present with an initial complaint of painless clicking with finger movement. This can progress to painful locking of the finger in flexion and may require passive manipulation of the digit into extension. Often a painful nodule can be palpated in the palmar MCP area.

## **OBJECTIVE EVIDENCE**

Patients with stenosing flexor tenosynovitis without a history of injury or inflammatory arthritis do not need routine radiographs.

Similarly, no laboratory tests are needed to make the diagnosis, but such tests may be helpful for detecting an underlying cause. Test for diabetes, thyroid disorders, and rheumatoid arthritis may be clinically indicated.

## **EPIDEMIOLOGY**

This condition occurs most commonly in the fifth and sixth decades of life and has been reported to be up to six times more common in women than in men. Lifetime risk of trigger finger has been shown to be 2-3% but increases up to 10% in diabetics. The ring finger and thumb are the most commonly affected digits followed by the long, index, and then small. The dominant hand is most commonly affected.

## **DIFFERENTIAL DIAGNOSIS**

The classic presentation of a painful click with forced extension of the fingers, with a palpable nodule probably does not have a true “differential diagnosis.” That is, when a tender nodule impeding passive extension is palpated by the examiner, there is no mistaking this condition for something else.

If the presentation is not classic, other conditions may be considered. Dupuytren’s disease can present with fixed contracture of the finger, as can a contracture of the joint capsule itself. A loose body in the MCP joint or an articular irregularity may cause a mechanical blockage. Failure of the extensor mechanism will also cause the finger to be held in flexion (as the flexors are unopposed). Extensor failure is distinguished from trigger finger, as in that case gentle passive correction is unimpeded. Irritation of the sesamoid bones (e.g., sesamoiditis) can mimic the painful sensation of trigger thumb.

It is also possible that reported triggering is a manifestation of psychiatric disease.



## RED FLAGS

Secondary trigger finger can be seen in patients with collagen vascular disorders and inflammatory disorders including rheumatoid arthritis, amyloidosis, gout, diabetes, hypothyroidism, and sarcoidosis and is associated with a worse prognosis after treatment.

## TREATMENT OPTIONS AND OUTCOMES

Non-operative treatment modalities include activity modification, NSAIDs, PIP joint immobilization and corticosteroid injections. NSAIDs and corticosteroid injections both work to reduce inflammation.



Figure 2: Trigger finger injection.

Corticosteroid injections (Figure 2) are placed into the tendon sheath at the level of the A1 pulley. If an injection does not cure the problem, a second or even third injection may be tried before deeming this approach a failure. Possible side effects include fat necrosis, skin hypopigmentation and, rarely, tendon rupture.

Most patients do well with non-operative measures. For those who do not, surgery can be performed to release the A1 pulley. An alternative, and less invasive, method is percutaneous release of the pulley with the use of a needle. This needle method, though, has a higher risk of digital nerve injury in the thumb and is thought to have a higher risk of incomplete release of the pulley.

The success rates of open surgical release has been shown to be greater than 90%. The complication rates are low (3% in some studies). Possible complications include nerve injury, bowstringing, scar formation, and continued pain.

In children less than 6 months old, 30% of trigger thumbs will resolve spontaneously. After 6 months of age, spontaneous resolution is decreased to nearly 10% and surgical intervention with release of the A1 pulley may be indicated.

## RISK FACTORS AND PREVENTION

There is a higher risk of development of trigger finger in patients with diabetes, hypothyroidism, gout, sarcoidosis, rheumatoid arthritis, and amyloidosis. There are no data on prevention methods in general, or those steps that could prevent a nascent case from worsening. (The latter, especially, would be particularly helpful.)

## MISCELLANY

A cross-sectional study of 665 workers at a meat-packing plant found that the person-year incidence rate of trigger finger was more than five-fold higher among those workers with repetitive tool use compared to non-tool use workers. This suggests that trigger finger may indeed be a work-related condition.

In guitar players, standard release of the A1 pulley for trigger finger may interfere with the fine control of the fingertip when the finger is held in extreme flexion.

## **KEY TERMS**

stenosing tenosynovitis, trigger finger, trigger thumb

## **SKILLS**

Recognize the clinical features of trigger finger and the treatment options.

## ULNAR AND RADIAL SHAFT FRACTURES

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In adults, simultaneous fractures of the shaft of the ulna and radius (the so-called “both bone fractures”) are most often the consequence of a direct blow to the forearm or other high energy mechanisms. In the adult, these injuries usually require surgical fixation. By contrast, these injuries in the pediatric population result from simple falls and other lower energy mechanisms. In children, ulnar and radial shaft fractures are amenable to casting. Imperfect healing can lead to loss of pronation and supination of the hand.

### STRUCTURE AND FUNCTION

The human forearm is comprised of two bones: the ulna, medially, and the radius, laterally. Bridging the ulna and radius is an interosseous membrane that transmits forces from ulna to radius and vice versa. The forearm flexes and extends at the elbow, with the articulation of the ulna with humerus at the trochlear notch. The head of the radius also articulates with the humerus at the capitellum, allowing the forearm to pronate and supinate. Pronation and supination also require an intact distal radial ulnar joint.

The median, ulnar, and radial nerves course along the forearm, along with the radial and ulnar arteries. The ulnar and radial nerves are located most medially and laterally, respectively, thus they are most susceptible to damage with fracture of the shaft of their adjacent bones.

The extrinsic muscles of the hand originate in the forearm (and elbow) and therefore forearm fractures, if not treated properly, can also lead to hand dysfunction.

The course of the muscles, likewise, may create deforming forces on the injured bones: for example, the flexor muscles of the fingers and wrist tend to produce dorsal bowing of the radius and ulna, by flexing distal fragments.

### PATIENT PRESENTATION

Patients with fractures of the shaft of the ulna and radius present following trauma with pain in the forearm, at time with gross deformity.

Radiographs allow full characterization of the injury. Care must be taken to fully visualize the wrist and elbow joints.

### ***Greenstick Fractures***

Pediatric fractures can be complete (involving a through-and-through break of the bone) or so-called “greenstick fractures,” a break on one surface of the bone that does not extend to the other side. This configuration occurs in children because, compared to adult bone, pediatric bone tends to be softer and more prone to incomplete breakage (much like the branches of a living tree, hence the name “greenstick”).



Figure 1: X-ray of a greenstick fracture. (from [wikimedia.org](http://bit.ly/1nTfTsC)  
<http://bit.ly/1nTfTsC>)

### ***Galeazzi Fracture***

A radial shaft fracture with distal radial ulnar joint (DRUJ) instability is known by its eponym, the “Galeazzi fracture.” A patient with a Galeazzi fracture will present not only with pain in the forearm where the radius is broken, but also swelling, tenderness, and pain at the wrist where there is a dislocation of the distal radioulnar joint.

Ulnar shaft fractures are most often caused by a direct blow to the border of the ulna on the medial forearm. It can occur when the ulnar border is exposed, such as upon raising the arm in defense (the so-called “night stick fracture”). The patient will usually experience pain and show signs of point tenderness in the ulnar region of the forearm. Swelling will likely be present in the forearm.



Figure 2: X-rays of a Galeazzi fracture. (From wikipedia <http://bit.ly/1Q7URwP>)

### **Monteggia Fractures**

“Monteggia fractures” are injuries to the proximal third of the ulna associated with a dislocation of the head of the radius. It is rare for a fracture to the proximal one-third of the ulna to occur alone. As a result, it is always imperative to do a full work-up of both the ulna and radius to assess whether the radius, especially the radial head, is injured.

Damage to the posterior interosseous nerve may occur with a Monteggia fracture; it is therefore important to assess this nerve by checking the patient’s ability to extend the hand at the wrist.



Figure 3: X-ray of a Monteggia fracture. (from wikipedia <http://bit.ly/1L4s4ro>)

## OBJECTIVE EVIDENCE

Patients with forearm fractures should have the following information collected and documented:

- status of the skin and soft tissues
- range of motion of the wrist and elbow
- gross alignment of the limb
- congruity of the elbow and wrist joints on x-ray
- status of nerves: radial (including posterior interosseous), median (including anterior interosseous), and ulnar (Figure 4)
- perfusion status of hand



*Figure 4: Demonstration of normal anterior interosseous nerve (AIN) and abnormal AIN function (right). In the figure at the right, the IP joint of the thumb and the DIP joint of the index finger will not flex normally when the AIN is injured. When the AIN is injured the flexor pollicis longus and flexor digitorum profundus do not work and thus the patient cannot make an "OK" sign.*

## EPIDEMIOLOGY

Fractures of the ulnar and radial shaft can occur across all age groups but are most common in children. Indeed, mid-shaft forearm fractures are among the most common type of fracture in children. This type of injury is most often the consequence of direct trauma, frequently from a fall on an outstretched hand from a large height.

Other populations at risk are adults or children involved in events such as traffic accidents and sports injuries, commonly from mountain biking and skating. Osteoporosis, mostly occurring in postmenopausal women, is commonly associated with forearm fractures, although fractures in this population are most often present in the distal forearm near the wrist.

## DIFFERENTIAL DIAGNOSIS

A forearm shaft fracture should be readily diagnosed from radiography. As such, there is no “differential” diagnosis list, per se; the question is either about what other injuries may be present, or whether the fracture was caused by some underlying abnormality such as osteoporosis.

Additional injuries that may be seen with Monteggia fractures, for example, include possible olecranon fracture-dislocation; radial head or coronoid fractures, or lateral collateral ligament injury.

Secondary injuries include not only damage to the elbow or wrist, but otherwise more distal unrelated injuries simply sustained at the same time.

Osteoporosis should be considered if the mechanism of injury suggests particularly low energy.

Fractures through a pre-existing lesion (a so-called “pathological fracture,” for example, one occurring in a bone with a tumor or Paget’s disease) are rare but possible.

## RED FLAGS

Radiographs that are too narrowly focused, i.e., do not visualize the elbow and wrist, may lead to missed diagnoses involving those excluded regions.

Both bone fractures can be complicated by acute compartment syndrome of the forearm; this is rare but possible. Signs suggesting compartment syndrome are pain on extension of digits and marked edema. The phrase “pain out of proportion to the injury” is difficult to apply when there is a broken bone, but in general, immobilizing a fracture should reduce the patient’s distress; if it does not, compartment syndrome should be explicitly ruled out by measuring compartment pressures.

Pediatric both-bone fractures, though commonly seen after accidental trauma, may also be the product of child abuse. Careful attention should be paid to the history given by the parents, (obtained from each separately, if abuse is suspected).

## TREATMENT OPTIONS AND OUTCOMES

Displaced and unstable ulnar fractures in adults should be treated operatively. Even non-displaced radial fractures are usually treated with surgery. (Pediatric fractures at times can be treated with closed reduction and casting.)

Monteggia fractures require open reduction and internal fixation of the ulnar shaft fracture. If this procedure fails to reduce the radial head, then that too must be opened and reduced.

Most fractures heal if adequately reduced.

Even subtle malalignment can impede pronation and supination.

Most cases of neuropathy (in the absence of compartment syndrome) tend to resolve with rest.

## RISK FACTORS AND PREVENTION

Beyond giving trite advice (“Don’t drink and drive. Stay out of fights. Don’t fall.”) there is not much physicians can do to prevent these injuries. The key “prevention” step is to prevent complications by not missing associated injuries at the time of initial presentation.



## **MISCELLANY**

Although two fractures of the forearm are named after Italian surgeons from Milan, their contribution did not reflect a golden age of orthopaedic surgery in that city. In fact, they were not contemporaries: Giovanni Battista Monteggia died in 1815, more than a half-century before Ricardo Galeazzi was born in 1866.

## **KEY TERMS**

greenstick fracture, Galeazzi fracture, Monteggia fracture

## **SKILLS**

Recognize and describe fracture patterns on plain radiographs. Assess the neurological function with a patient following fracture. Predict the deforming forces that muscles exert on fractures.