Review Article

Tendinopathies of the Hand and Wrist

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Abstract

Tendinopathies involving the hand and wrist are common. Many are diagnosed easily, and in many cases, the management is straightforward, provided the pathology and principles are understood. Common conditions involving the tendons of the hand and wrist include trigger finger, tenosynovitis of the first through sixth dorsal extensor compartments, and flexor carpi radialis tendonitis. Management strategies include nonsurgical treatments, such as splinting, injection, or therapy, and surgical techniques such as tendon release.

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As its name suggests, tendinopathy is an affliction of the tendon, occurring anywhere along its course. Stenosing tenosynovitis, or trigger finger, occurs when a size discrepancy exists between the tendon and the pulley or sheath through which the tendon passes. This condition may occur because the sheath or pulley becomes narrowed or because of the increased size of the tendon secondary to degeneration or tendinosis. This distortion prevents the easy and smooth gliding of the tendon. The pathophysiology can be varied, ranging from overuse to inflammatory pathology from such disorders as rheumatoid arthritis (RA) or crystalline disease (ie, gout) to systemic disorders such as diabetes mellitus (DM) or thyroid disease, which result in tendon adhesions or thickening. Common histologic findings in the sheath include collagen degradation, vascular ingrowth, and fibrocartilage metaplasia, believed to be a response to compression and shear during tendon gliding.

Tendinosis refers to chronic degenerative changes in the tendon. In stenosing tenosynovitis and tendinosis, the conspicuous absence of inflammatory cells typically is noted. It has been proposed that the prolonged repetitive stress of these conditions puts mechanical strain on the tendon, causing microruptures.[1-5] Because of the lack of a robust intrinsic blood supply and uneven strain, the healing and remodeling responses are altered. Changes in gene expression have been noted in the tendons. Pain may be mediated by neurochemical cytokines and potentiated by vascular changes.[1-7]

Stenosing Tenosynovitis

Stenosing tenosynovitis at the A1 pulley is by far the most common tendon pathology seen. Women are affected six times more often than are men, with a higher incidence in patients with DM and RA. The prevalence of trigger digit is 2% to 3%, but in patients with diabetes, the prevalence rises to 10% to 20%.[8,9]

Changes occur in the pulley, including thickening of the A1 pulley, and in the flexor tendons, primarily the flexor digitorum profundus (FDP) but also the flexor digitorum superficialis (FDS) (Figure 1). The etiology

Figure 1

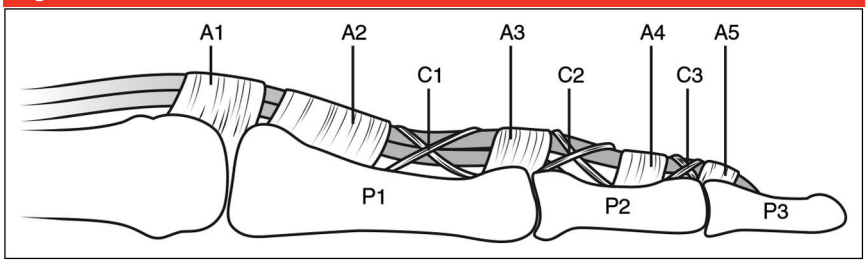


Illustration demonstrating the flexor tendons and the pulley system of the digits. The A1 pulley is released during trigger finger release. A = annular pulleys, C = cruciate pulleys, P = phalanges

is varied, and an exact cause is unknown; it is likely multifactorial. Proposed contributing factors include genetic changes, systemic conditions such as renal insufficiency, thyroid disease, or DM, and occupational issues.[7]

Presenting symptoms often include pain at the level of the A1 pulley, which progresses to triggering or locking. Patients may report a reduced grip, clicking, catching, or locking. In most patients, a tender nodule can be palpated at the site of the A1 pulley. Patients also may have a proximal interphalangeal (PIP) contracture, which often fails to fully reverse even following successful release. A high concordance with concomitant carpal tunnel syndrome is present, with >60% of patients with trigger digits demonstrating clinical or electrodiagnostic evidence of median nerve compression at the wrist.[8,10-13]

Initial management strategies may include a supervised therapy program or instruction in a home exercise program, rest with splints, and NSAIDs. Orthoses are reported to provide relief in 40% to 87% of cases.[14-16] These aids typically immobilize the metacarpophalangeal (MCP) joint but allow PIP joint and distal interphalangeal joint motion, although one splint described by Rodgers et al[17] immobilized only the distal interphalangeal joint and was reported to be successful in 55% of cases. Compliance may be an issue because patients may find the splints to be cumbersome.

Supervised physical therapy programs, often aimed at developing differential gliding between the FDS and FDP tendons, tend to be less effective than other modalities, such as steroid injection, in preventing pain and triggering. One series reported success rates of 69% for physical therapy programs versus 97% resolution for injection at 3 months, but patients who responded to physical therapy experienced no recurrence of pain or triggering at 6 months, indicating that physical therapy was more effective than injection in preventing recurrence.[18]

Corticosteroid injections into the A1 pulley region commonly are used as a first-line treatment of trigger digits. They have shown an excellent response rate of 60% to 90%.[19] Recurrence rates are higher in the setting of concomitant DM, multiple affected digits, and other associated tendinopathies;[20-24] a diminishing likelihood of subsequent successful injection seems to exist following a recurrence of triggering after prior injection.[20] A longer duration of symptoms has been associated with relatively poorer outcomes with injections.[25] Complications following corticosteroid injections are uncommon but can include fat atrophy and tendon rupture.[26-28] A transient elevation of blood glucose levels can occur, particularly in diabetic patients requiring insulin.[20,28,29]

In one study, the type of steroid—soluble triamcinolone versus insoluble dexamethasone—did not play a major role in the final long-term outcome. The insoluble steroid tended to have a more rapid onset of action, but results were less durable than those following injection of the soluble steroid.[24]

If nonsurgical management fails, surgery is considered. Options include open or percutaneous release of the A1 pulley. Open release is the standard and is associated with a high rate of excellent results.[30] Open release can be achieved by a transverse, oblique, or longitudinal incision.

Local anesthetic is preferred because it allows the patient to move the affected finger during surgery to confirm the lack of triggering after release. If triggering persists despite A1 release, additional pathology such as an A0 pulley—a tight band of superficial palmar aponeurosis proximal to the A1 pulley—or structural changes in the FDS or FDP should be sought. In certain patients, such as those with long-standing DM, long-standing trigger digits, or RA, an element of FDP entrapment may be present at the FDS decussation, or enlargement of the FDP may have led to entrapment at the A3 pulley. During the preoperative evaluation in particular, the surgeon should have a heightened level of suspicion about patients with a preoperative PIP joint contracture because these patients may have changes in the tendons that could cause residual triggering after a complete A1 pulley release.[31] Options in this setting include reduction tenoplasty of the FDP tendon or, more commonly, excision of a slip of the FDS.[31]

Recurrence is rare following open release. Complications also are rare but include injury to the neurovascular bundle and stiffness and

bowstringing if the pulley is cut. Will and Lubahn[32] reported a 28% rate of minor complications, including stiffness and wound complications, and a major complication rate of 3% in a study of 78 open trigger releases.

Recent studies document the precise surface anatomy of the A1 pulley, and percutaneous release of the A1 pulley also has been described. Cadaver studies have shown a high rate of division of the A1 pulley and a low complication rate with percutaneous trigger release.[33-38] Clinical studies also have supported the early recovery from and the benefits of the percutaneous procedure.[39,40] Concerns remain, however, about tendon scoring or laceration and neurovascular injury, especially in the border digits and the thumb.[33]

The specific instruments and techniques used for percutaneous release vary but include hypodermic needles, knife blades, or purpose-made devices or blades deployed under tactile feel or with ultrasound guidance. In general, however, some common features exist across all techniques. An injection of local anesthesia over the intended surgical site is used. In general, percutaneous release is easiest in the setting of active triggering because adequate release can be confirmed immediately. To extend or hyperextend the MCP joint, the patient rests the dorsum of the hand on a stack of towels, allowing the digital neurovascular structures to drop more dorsally and away from the surgical site. The patient may be asked to flex and extend the digit, and the region of the A1 pulley is palpated over the volar area of the MCP joint. The blade or needle is introduced at the proximal edge of the A1 pulley and is used to cut distally. If a needle is used, it is typically 18- to 19-gauge, and the bevel is oriented parallel to the tendon. It may be placed into the flexor tendon; the location may be confirmed by movement of the digit; and the needle is withdrawn until it is just out of the tendon. The needle is then advanced longitudinally, producing a tactile grating sensation as the pulley is cut. When the surgeon believes that release is complete, the needle is withdrawn, and an attempt to elicit triggering is made.[41]

Figure 2

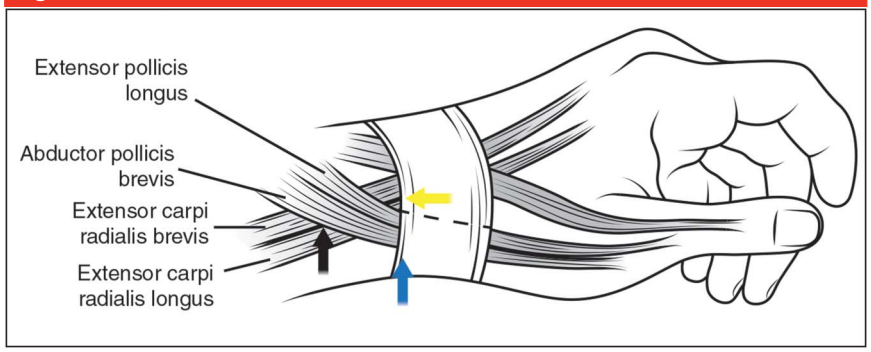


Illustration demonstrating the anatomy of the first (blue arrow) and second (yellow arrow) dorsal extensor compartments, differentiating the locations of de Quervain syndrome (blue arrow) and intersection syndrome (black arrow).

De Quervain Stenosing Tenosynovitis

De Quervain tenosynovitis affects the tendons within the first dorsal compartment, the abductor pollicis longus (APL), and the extensor pollicis brevis (EPB) (Figure 2). A septum may separate the EPB and APL tendons in the first compartment itself in about 40% of the population. Additionally, multiple tendon slips of the APL typically are present.[42-46] De Quervain tenosynovitis is associated with pregnancy, the postpartum period, and lactation, and with activities involving repeated radioulnar deviation, such as hammering, cross country skiing, or lifting a child or pet.

The diagnosis is based on the clinical examination and history. Radiographs of the wrist may be obtained to exclude alternative pathology in the distal radius or carpus; thumb carpometacarpal (CMC) arthritis may coexist and may or may not be symptomatic. Other diagnostic studies rarely are required or useful. On radiographs, soft-tissue calcifications at the first dorsal compartment may be visualized occasionally, but more commonly, radiographs are completely unremarkable.[47]

Patients typically present with pain and swelling over the dorsoradial side of the wrist. These symptoms are aggravated by resisted motion of the thumb. Radial deviation and extension also can worsen the pain. Examination shows tenderness over the first compartment. Often a fullness or swelling is present that represents a thickening of the first dorsal compartment retinaculum, and a small retinacular ganglion cyst may be present in the area. Typically, physical examination and history alone are diagnostic. The assessment commonly described as the Finkelstein test (ie, ulnar deviation of the wrist when the patient grasps the thumb in the palm with the other digits) was in fact described by Eichoff and referenced by Finkelstein[48] in his report. A staged series of testing described by Dawson and Mudgal[49] may be superior because patients may find the Finkelstein test excessively painful. During this testing, the patient places the wrist in neutral pronosupination on a table, with the

ulnar aspect of the forearm resting on the table and the wrist and distal aspect hanging over the edge of the table unsupported. Simple active gravity-assisted ulnar deviation is performed. A painful result indicates first dorsal extensor pathology. If the result is negative, further passive ulnar deviation performed by the examiner may elicit pain to secure the diagnosis. Finally, if de Quervain syndrome is suspected but the previous tests are not suggestive, the examiner may add passive thumb flexion into the palm with concomitant wrist ulnar deviation to assess for pain.[49]

Factors associated with a relatively poorer response to nonsurgical treatment include the presence of separate compartments for the APL and EPB or the triggering of the tendons with thumb motion.[50] Alexander et al[44] described an EPB entrapment test to assess for the presence of a separate compartment for the EPB. The patient is asked to extend the MCP joint of the thumb against resistance and then to palmar abduct the thumb against resistance. If the first maneuver is more painful, the presence of a separate compartment is indicated. The test is highly sensitive (81%) but moderately specific (50%) for a separate EPB compartment.

Treatment options include oral NSAIDs or corticosteroids, splinting, physical therapy, and surgery. Splinting alone is successful in about 14% to 18% of patients.[51,52]

Typically, a forearm-based thumb spica splint is used to enforce a period of rest or prevent motions that exacerbate symptoms. Physical therapy may include education about and modification of activity, modality treatments, and tendon-gliding exercises. Cortisone injection is the most successful nonsurgical treatment modality reported in the literature by far, with a 62% to 100% success rate suggested by prior studies. The addition of physical therapy and/or splinting to steroid injection does not seem to improve the success rate of treatment over steroid injection alone.[51,53,54]

The success rate is poorer if an associated septum is present within the first dorsal compartment[44] or if triggering of the first dorsal extensor compartment tendons occurs.[55] The risks of corticosteroid injection include skin depigmentation and skin and subcutaneous tissue atrophy, in addition to the transient elevation of blood glucose levels.[51,53,54]

Surgery involves the release of the sheath covering the first dorsal compartment. This release can be achieved by a transverse or longitudinal incision. Care should be taken to identify and protect the superficial branch of the radial nerve. After the sheath is released, a separate subcompartment for the two tendons should be sought. If present, this subcompartment also should be released. Missing this step is the most common cause of recurrence or residual symptoms. Most surgeons advocate release of the compartment on the dorsal side, thus leaving a volar portion of the retinaculum to act as a buttress against volar subluxation of the tendons.

Complications are minor and rare. They include volar subluxation of the first compartment tendons over the radial styloid, superficial radial nerve neuritis or neuroma, residual symptoms, or a painful or bothersome scar.

Intersection Syndrome

Irritation of the tendons of the second dorsal compartment, or intersection syndrome, is relatively rare.[56] It is associated with the frequent and repetitive use of the wrist and typically occurs in athletes such as rowers. Pain is located at the intersection of the first and second dorsal extensor compartments and is speculated to be related to rubbing of the EPB/APL against the radial wrist extensors (Figure 2). The wrist discomfort is more proximal than that seen in de Quervain syndrome and is exacerbated by wrist motion, particularly resisted wrist extension. Palpable or audible crepitus may be noted. The diagnosis is based on the history, clinical symptoms, and examination findings. Differential local anesthetic injections may help in supporting the diagnosis or in differentiating between intersection syndrome and de Quervain syndrome.

The initial treatment is nonsurgical and includes a trial of antiinflammatory medications, rest, activity modification, and forearm-based wrist splinting at about 15° of wrist extension. Corticosteroid injection also may be useful. Many patients will respond to these measures, but if nonsurgical management fails, surgical release of the second dorsal extensor compartment is an option. It is important to release any fascia over the tendons about the area of pathology.[56]

Extensor Pollicis Longus Tenosynovitis

Tendinitis or tendinosis of the third dorsal extensor compartment is rare and is even rarer in the patient without RA. Triggering or snapping of the extensor pollicis longus (EPL) resulting from a size discrepancy between the tendon and the compartment has been described and may progress to attenuation and rupture of the tendon. A high rate of rupture is seen; thus, surgery is preferred over nonsurgical treatment.[57-60]

The disorder is believed to be more common in drummers or following trauma to the wrist, particularly a fall on the extended wrist. The proposed mechanism involves impingement of the EPL between the dorsal radius and the third metacarpal.[61]

Impending or completed rupture of the EPL classically is described in the setting of a minimally displaced

distal radius fracture and has an estimated 5% incidence.[62] Although rupture could be related etiologically to rubbing of the tendon over a sharp bony prominence (ie, attritional rupture), it is believed to be related most commonly to a nutritional phenomenon, in which swelling increases the pressure within the compartment and alters the blood supply to the mesotendon in this relative watershed zone, causing ischemic changes in the tendon.[63] This phenomenon likely is related to the unique anatomy at this area, in which the EPL tendon takes a sharp turn just distal to the Lister tubercle and is encased in the tight fibrous sheath of the third compartment, which does not allow for expansion. Thus, swelling increases the pressure in the noncompliant compartment, altering the blood supply and leading to ischemic changes. This theory is supported by findings of tendinopathy—which precedes rupture[57]—on MRI, ultrasonography, or visual inspection during surgical exploration[64] (Figure 3).

Presenting symptoms include pain and swelling at the distal radius in the area of the Lister tubercle; pain is exacerbated by active or passive thumb motion. Occasionally, palpable crepitus is present. Some patients demonstrate snapping or triggering. Radiography may be helpful in excluding any distal radius fracture or possible bony prominence.

Treatment is typically surgical because a high risk of tendon rupture is present. The third dorsal compartment is approached via a dorsal incision centered over the Lister tubercle, and the compartment is released. The EPL is moved to the radial subcutaneous tissues, and the compartment is inspected for any bony prominences. The tendon itself is inspected for attritional changes, and the surgeon should be prepared to graft or perform a tendon transfer if indicated.[57]

Figure 3

Magnetic resonance image demonstrating extensor pollicis longus (EPL) tenosynovitis in the setting of a nondisplaced distal radius fracture, suggestive of impending EPL rupture. The arrow indicates the EPL tendon. (Reproduced with permission from Hasham S, Burke FD: Diagnosis and treatment of swellings in the hand. *Postgrad Med J* 2007;83-296-300.)

Fourth Compartment Tenosynovitis

Extensor tenosynovitis, or extensor digitorum communis tenosynovitis of the entire fourth compartment, can present in the setting of RA or other inflammatory etiologies. It also may begin after trauma or a nondisplaced distal radius fracture. It has been proposed that, because of wrist stiffness, an increase in the moment arm of the extensors occurs as they course toward their target digits and insertion sites. Cooper et al[65] have described extensor digitorum communis tenosynovitis in a nonrheumatoid setting, in which the histopathologic evidence was consistent with traumatic tenosynovitis.

Fourth compartment tenosynovitis presents with pain over the fourth compartment, along with localized tenderness and swelling. Forced extension of the wrist may impinge the tendons and exacerbate the pain. The "tuck sign," in which a mass or synovial thickening tracks with the tendons as the tendons move, commonly is present and bunches or tucks at the edge of the retinaculum during

Figure 4

Clinical photograph demonstrating dorsal tenosynovitis of the fourth extensor compartment, with soft-tissue swelling that tracks with the tendons as they move and bunches or tucks at the edge of the retinaculum, producing the "tuck sign." (Reproduced with permission from Huang HW, Strauch RJ: Extensor pollicis longus tenosynovitis: A case report and review of the literature. *J Hand Surg* 2000;25A:577-579.)

digital extension (Figure 4). Secretan disease can present in a similar way, with dorsal edema. This disorder is self-inflicted for secondary gain or appears as the result of a conversion disorder. Surgery for this condition is avoided.[66]

Initial treatment of fourth compartment tenosynovitis includes rest with splints, ice, and NSAIDs. Steroid injections can be used if the symptoms persist. Failure of nonsurgical management to relieve pain suggests a physical etiology of increased pressure within the compartment, namely an accessory tendon or anomalous muscle, and may lead to surgical release or tenosynovectomy.[67] Cooper et al[65] described 11 patients with proliferative tenosynovitis who presented with a limitation of wrist extension. These patients had no improvement with nonsurgical treatment. They required tenosynovectomy, which improved their wrist extension and grip strength.

Fifth Compartment Tenosynovitis

The fifth compartment, containing the extensor digiti minimi, also may be

Figure 5

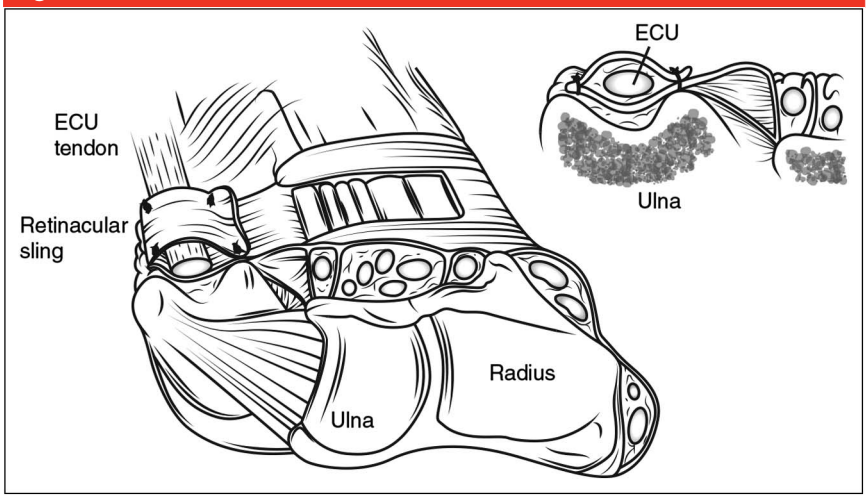


Illustration demonstrating a subluxating extensor carpi ulnaris (ECU) tendon that may be stabilized by creating a flap of extensor retinaculum and securing it about the ECU tendon and to itself. The smaller image is a cross-sectional view.

affected in isolation.[68,69] This occurrence is rare. The pain is localized over the fifth compartment, and the tenderness and swelling are present over the extensor digiti minimi tendon. This condition is investigated and managed similarly to other tendinopathies.

Extensor Carpi Ulnaris Tenosynovitis

Extensor carpi ulnaris (ECU) tenosynovitis is included in the differential diagnosis for ulnar side wrist pain and is a very common source of such pain.[70,71] The diagnosis may be challenging because the ECU is apposed closely to other ulnar side wrist structures, including the triangular fibrocartilaginous complex (TFCC). The ECU is a wrist ulnar deviator when the forearm is in pronation and a wrist extensor when the forearm is supinated. It has been shown to provide a static as well as a dynamic support to the wrist. The volar ECU sheath is adjacent to the TFCC, and ECU injury may coexist with concomitant TFCC injury. Because of the close anatomic location and overlap in pathology between the ECU and the TFCC, it may be difficult to isolate the source of ulnar side wrist pain clinically and to differentiate between TFCC pathology and ECU problems, or both. When examining the ECU, it is important to differentiate between ECU tendinitis and ECU pain secondary to subluxation.

Although ECU subluxation may begin with an injury to the wrist joint with or without any underlying fractures, ECU tendinitis typically occurs following overuse or occurs in an idiopathic fashion. Pain is localized over the ulnar side of the wrist and worsens with gripping and other heavy activities. Unlike ECU subluxation, which may occur in athletes following an injury, tendinitis is seen more often in nonathletes. The patient may be unable to localize the site of pain easily; hence, it is important to differentiate the pathology from other causes of ulnar side wrist pain. In the setting of ECU tendinitis, careful palpation reveals point tenderness over the ECU tendon, with some fullness. Range of motion may not be restricted, but resisted ulnar deviation when the forearm is in pronation or resisted wrist extension when the forearm is supinated increases discomfort. Rolling the tendon under the examiner's finger may elicit crepitus because of an edematous sheath. Altered sensation may be seen over the distribution of the dorsal sensory branch of the ulnar nerve because it travels in close approximation to the sheath. The ECU synergy test is helpful in differentiating between ECU pathology and intra-articular (ie, TFCC) problems.[72] The patient rests the elbow of the supinated forearm on a table, with the digits extended. The examiner grasps the patient's thumb and long finger and asks the patient to deviate the thumb radially against resistance. The examiner's other hand gently palpates the ECU and flexor carpi ulnaris. The presence of pain suggests pathology in the ECU rather than intra-articular pathology. Patients are assessed for subluxation of the ECU tendon. The examiner moves the patient's wrist from a position of supination and extension to one of flexion and ulnar deviation. This maneuver allows the ECU tendon to move out of the groove of the sixth compartment. The test is positive when visible subluxation is present or an audible snap is heard.

Diagnostic studies may include plain radiographs, including PA grip views to evaluate for ulnar positive variance and possible ulnar impaction as an alternative source of ulnar side wrist pain. Occasionally, ultrasonography or MRI may be used to confirm tendinopathy, document subluxation, or exclude other sources of pathology. Differential diagnostic and therapeutic injections may be considered. The diagnostic criteria can be improved by injecting local anesthetic into the ECU sheath. The elimination of symptoms and signs can differentiate this condition from other causes of ulnar side wrist pain.

The initial treatment is nonsurgical and may include rest, activity modification, therapy, ice,

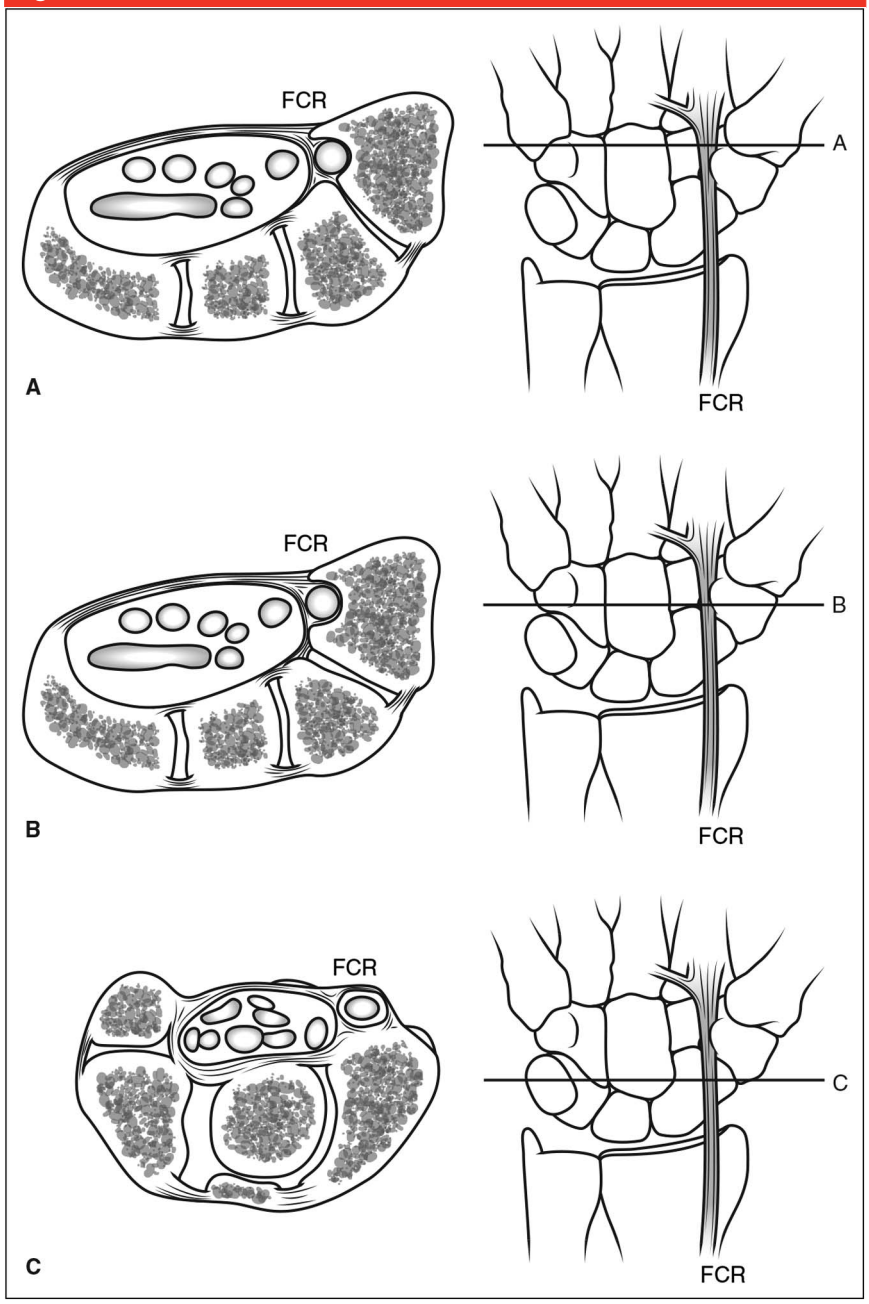
and NSAIDs.[70,73,74] Corticosteroid injection may be considered, but care is taken not to inject into the tendon substance. In addition to the risk of elevated blood glucose levels, steroid atrophy, or skin changes, a risk of tendon rupture also exists following injection.[75]

Surgery is reserved for patients with ECU tendinitis in whom nonsurgical treatment has failed to relieve the pain. The surgical approach is made via a longitudinal incision centered over the sixth extensor compartment. The dorsal ulnar sensory branch should be identified and protected. The compartment is released via a longitudinal incision or by creating a retinacular flap for later repair. Any projecting fibro-osseous structures can be removed. Although concerns exist about subsequent subluxation of the ECU if the retinaculum is not repaired, several series have reported no instability or subluxation, even when the sheath or the retinaculum was not repaired.[71,73] In the setting of surgery for a subluxating ECU, the tendon is stabilized by creating a pulley from the retinaculum after the ECU has been released from its sheath. A variety of techniques have been described, but one method involves creating an ulnarly based flap of the extensor retinaculum elevated from over the fourth extensor compartment. The flap is routed under (volar to) the ECU tendon and then directed dorsally, where it is sutured to the adjacent radial side of the ECU sheath and to itself[76] (Figure 5).

Flexor Carpi Radialis Tendinitis

The flexor carpi radialis (FCR) tendon has a unique anatomic path and insertion. It angles across the trapezial ridge and enters a fibro-osseous tunnel adjacent to the trapezium before inserting into the base of the second metacarpal; in some cases, the trapezium may enclose the tendon[77] (Figure 6). This narrow fibro-osseous tunnel makes the FCR tendon vulnerable to impingement and fraying from osteophytes and degenerative changes in the adjoining joints, including thumb CMC and scaphotrapezial degeneration.[78,79]

Figure 6



A through **C**, Cross-sectional illustrations demonstrating the anatomy of the flexor carpi radialis (FCR) tendon in the fibro-osseous groove. At the level of the distal trapezium (**A**), the FCR occupies approximately 90% of the available space, whereas more proximally at the proximal trapezial level (**B**) and at the scaphoid tuberosity level (**C**), the FCR occupies less of the available space. At the tuberosity level, the FCR occupies approximately 60% of the available space.

Patients with FCR tendinitis may present with pain in the region of the radial volar forearm at the wrist level. The pain worsens with resisted wrist flexion and radial deviation. An associated swelling may be present over the palpable area of tenderness at the FCR. The differential diagnosis for FCR tendinitis includes ganglions and scaphoid injuries. Thumb CMC or scaphoid-trapezium-trapezoid arthritis also are included in the differential diagnosis but may coexist. Plain radiographs are obtained to delineate arthritic changes and osteophytes. MRI may show signal changes or edema at the FCR sheath.

Injection of local anesthetic with or without corticosteroid medication alleviates pain and can differentiate FCR tendinitis from other pathologies. Care must be taken when injecting because the radial artery or its branches lie in close proximity to the tendon. In addition, injection can precipitate tendon rupture, although tendon rupture typically results in resolution of symptoms with little functional deficit. Left untreated, the FCR tendon also may rupture.

Nonsurgical treatment is the first-line approach and begins with symptomatic care, including NSAIDs, splinting for rest, and ice. Occasionally, physical therapy is added. Cortisone injection may be considered.

Intractable symptoms may require surgery. If the radiographs suggest substantial bony impingement of the canal, early release may be considered before the tendon ruptures.

Release is achieved through a volar incision centered over the FCR at the wrist. Blunt dissection should be used to identify any cutaneous branches of the superficial radial nerve or the palmar cutaneous branch of the median nerve, and these nerve branches should be protected to avoid injury and neuroma formation. The FCR tendon and the fibro-osseous tunnel are identified. The fascia over the tendon is decompressed from the volar wrist crease distal to the trapezium. It is common to find fraying of the tendon, which should be débrided, and any bony impingement in the tunnel should be excised. A high rate of symptomatic improvement and a low rate of recurrence are seen following surgical release.[80]

Summary

Common conditions involving the tendons of the hand and wrist include trigger finger, tenosynovitis of the first through sixth dorsal extensor compartments, and FCR tendonitis. Management of tendinopathies of the hand and wrist is typically straightforward. Strategies include nonsurgical treatments, such as splinting, injection, or therapy, and surgical techniques, such as tendon release.

References

References printed in **bold type** are those published within the past 5 years.

1. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M: Histopathology of common tendinopathies: Update and implications for clinical management. *Sports Med* 1999; 27(6):393-408.
2. Xu Y, Murrell GA: The basic science of tendinopathy. *Clin Orthop Relat Res* 2008; 466(7):1528-1538.
3. Sharma P, Maffulli N: Tendon injury and tendinopathy: Healing and repair. *J Bone Joint Surg Am* 2005;87(1):187-202.
4. Fedorczyk JM, Barr AE, Rani S, et al: Exposure-dependent increases in IL-1beta, substance P, CTGF, and tendinosis in flexor digitorum tendons with upper extremity repetitive strain injury. *J Orthop Res* 2010; 28(3):298-307.
5. Benjamin M, Qin S, Ralphs JR: Fibrocartilage associated with human tendons and their pulleys. *J Anat* 1995;187 (pt 3):625-633.
6. Pufe T, Petersen WJ, Mentlein R, Tillmann BN: The role of vasculature and angiogenesis for the pathogenesis of degenerative tendons disease. *Scand J Med Sci Sports* 2005;15(4):211-222.
7. **Lundin AC, Eliasson P, Aspenberg P: Trigger finger and tendinosis. *J Hand Surg Eur Vol* 2012;37(3):233-236.**
8. Koh S, Nakamura S, Hattori T, Hirata H: Trigger digits in diabetes: Their incidence and characteristics. *J Hand Surg Eur Vol* 2010;35(4):302-305.
9. Chammas M, Bousquet P, Renard E, Poirier JL, Jaffiol C, Allieu Y: Dupuytren's disease, carpal tunnel syndrome, trigger finger, and diabetes mellitus. *J Hand Surg Am* 1995;20(1):109-114.
10. **Wessel LE, Fufa DT, Boyer MI, Calfee RP: Epidemiology of carpal tunnel syndrome in patients with single versus multiple trigger digits. *J Hand Surg Am* 2013;38(1):49-55.**
11. **Gancarczyk SM, Strauch RJ: Carpal tunnel syndrome and trigger digit: Common diagnoses that occur "hand in hand". *J Hand Surg Am* 2013;38(8):1635-1637.**
12. Garti A, Velan GJ, Moshe W, Hendel D: Increased median nerve latency at the carpal tunnel of patients with "trigger finger": Comparison of 62 patients and 13 controls. *Acta Orthop Scand* 2001;72(3): 279-281.
13. Rottgers SA, Lewis D, Wollstein RA: Concomitant presentation of carpal tunnel syndrome and trigger finger. *J Brachial Plex Peripher Nerve Inj* 2009;4:13.
14. **Valdes K: A retrospective review to determine the long-term efficacy of orthotic devices for trigger finger. *J Hand Ther* 2012;25(1):89-95, quiz 96.**
15. Evans RB, Hunter JM, Burkhalter WE: Conservative management of trigger finger: A new approach. *J Hand Ther* 1988;1: 59-68.
16. Colbourn J, Heath N, Manary S, Pacifico D: Effectiveness of splinting for the treatment of trigger finger. *J Hand Ther* 2008;21(4):336-343.
17. Rodgers JA, McCarthy JA, Tiedeman JJ: Functional distal interphalangeal joint splinting for trigger finger in laborers: A review and cadaver investigation. *Orthopedics* 1998;21(3):305-309, discussion 309-310.
18. **Salim N, Abdullah S, Sapuan J, Haflah NH: Outcome of corticosteroid injection versus physiotherapy in the treatment of mild trigger fingers. *J Hand Surg Eur Vol* 2012; 37(1):27-34.**
19. **Dala-Ali BM, Nakhdjevani A, Lloyd MA, Schreuder FB: The efficacy of steroid injection in the treatment of trigger finger. *Clin Orthop Surg* 2012;4(4):263-268.**
20. **Sato ES, Gomes Dos Santos JB, Belloti JC, Albertoni WM, Faloppa F: Treatment of trigger finger: Randomized clinical trial comparing the methods of corticosteroid injection, percutaneous release and open**

surgery. *Rheumatology (Oxford)* 2012;51 (1):93-99.

21. Rozental TD, Zurakowski D, Blazar PE: Trigger finger: Prognostic indicators of recurrence following corticosteroid injection. *J Bone Joint Surg Am* 2008;90(8): 1665-1672.

22. Schubert C, Hui-Chou HG, See AP, Deune EG: Corticosteroid injection therapy for trigger finger or thumb: A retrospective review of 577 digits. *Hand (N Y)* 2013;8(4): 439-444.

23. Mol MF, Neuhaus V, Becker SJ, Jupiter JB, Mudgal C, Ring D: Resolution and recurrence rates of idiopathic trigger finger after corticosteroid injection. *Hand (N Y)* 2013;8(2):183-190.

24. Ring D, Lozano-Calderón S, Shin R, Bastian P, Mudgal C, Jupiter J: A prospective randomized controlled trial of injection of dexamethasone versus triamcinolone for idiopathic trigger finger. *J Hand Surg Am* 2008;33(4):516-522, discussion 523-524.

25. Patel MR, Moradia VJ: Percutaneous release of trigger digit with and without cortisone injection. *J Hand Surg Am* 1997; 22(1):150-155.

26. Taras JS, Raphael JS, Pan WT, Movagharnia F, Sotereanos DG: Corticosteroid injections for trigger digits: Is intrasheath injection necessary? *J Hand Surg Am* 1998;23(4):717-722.

27. Fitzgerald BT, Hofmeister EP, Fan RA, Thompson MA: Delayed flexor digitorum superficialis and profundus ruptures in a trigger finger after a steroid injection: A case report. *J Hand Surg Am* 2005;30(3): 479-482.

28. Wang AA, Hutchinson DT: The effect of corticosteroid injection for trigger finger on blood glucose level in diabetic patients. *J Hand Surg Am* 2006;31(6): 979-981.

29. Catalano LW III, Glickel SZ, Barron OA, Harrison R, Marshall A, Purcelli-Lafer M: Effect of local corticosteroid injection of the hand and wrist on blood glucose in patients with diabetes mellitus. *Orthopedics* 2012; 35(12):e1754-e1758.

30. Saldana MJ: Trigger digits: Diagnosis and treatment. *J Am Acad Orthop Surg* 2001;9 (4):246-252.

31. Le Viet D, Tsionos I, Boulouednine M, Hannouche D: Trigger finger treatment by ulnar superficialis slip resection (U.S.S.R.). *J Hand Surg Br* 2004;29(4):368-373.

32. Will R, Lubahn J: Complications of open trigger finger release. *J Hand Surg Am* 2010;35(4):594-596.

33. Habbu R, Putnam MD, Adams JE: Percutaneous release of the A1 pulley: A cadaver study. *J Hand Surg Am* 2012;37 (11):2273-2277.

34. Calleja H, Tanchuling A, Alagar D, Tapia C, Macalalad A: Anatomic outcome of percutaneous release among patients with trigger finger. *J Hand Surg Am* 2010; 35(10):1671-1674.

35. Wilhelmi BJ, Mowlavi A, Neumeister MW, Bueno R, Lee WP: Safe treatment of trigger finger with longitudinal and transverse landmarks: An anatomic study of the border fingers for percutaneous release. *Plast Reconstr Surg* 2003;112(4):993-999.

36. Zyluk A, Jagielski G: Percutaneous A1 pulley release vs steroid injection for trigger digit: The results of a prospective, randomized trial. *J Hand Surg Eur Vol* 2011;36(1):53-56.

37. Pope DF, Wolfe SW: Safety and efficacy of percutaneous trigger finger release. *J Hand Surg Am* 1995;20(2):280-283.

38. Wang J, Zhao JG, Liang CC: Percutaneous release, open surgery, or corticosteroid injection, which is the best treatment method for trigger digits? *Clin Orthop Relat Res* 2013;471(6):1879-1886.

39. Lyu SR: Closed division of the flexor tendon sheath for trigger finger. *J Bone Joint Surg Br* 1992;74(3):418-420.

40. Gilberts EC, Beekman WH, Stevens HJ, Wereldsma JC: Prospective randomized trial of open versus percutaneous surgery for trigger digits. *J Hand Surg Am* 2001;26 (3):497-500.

41. Ryzewicz M, Wolf JM: Trigger digits: Principles, management, and complications. *J Hand Surg Am* 2006;31 (1):135-146.

42. Jackson WT, Viegas SF, Coon TM, Stimpson KD, Frogameni AD, Simpson JM: Anatomical variations in the first extensor compartment of the wrist: A clinical and anatomical study. *J Bone Joint Surg Am* 1986;68(6):923-926.

43. Leslie BM, Carlson G, Ruby LK: Results of extensor carpi ulnaris tenodesis in the rheumatoid wrist undergoing a distal ulnar excision. *J Hand Surg Am* 1990;15(4): 547-551.

44. Alexander RD, Catalano LW, Barron OA, Glickel SZ: The extensor pollicis brevis entrapment test in the treatment of de Quervain's disease. *J Hand Surg Am* 2002; 27(5):813-816.

45. Lane LB, Boretz RS, Stuchin SA: Treatment of de Quervain's disease: Role of conservative management. *J Hand Surg Br* 2001;26(3):258-260.

46. Ta KT, Eidelman D, Thomson JG: Patient satisfaction and outcomes of surgery for de Quervain's tenosynovitis. *J Hand Surg Am* 1999;24(5):1071-1077.

47. Kirkpatrick W: Soft tissue conditions: Trigger fingers and De Quervain's syndrome, in Hunter J, Mackin E, Callahan A, eds: *Rehabilitation of the Hand: Surgery and Therapy*, ed 4. St. Louis, Mosby, 1995, pp 1012-1014.

48. Finkelstein H: Stenosing tendovaginitis at the radial styloid process. *J Bone Joint Surg Am* 1930;12:509-540.

49. Dawson C, Mudgal CS: Staged description of the Finkelstein test. *J Hand Surg Am* 2010;35(9):1513-1515.

50. Alberton GM, High WA, Shin AY, Bishop AT: Extensor triggering in de Quervain's stenosing tenosynovitis. *J Hand Surg Am* 1999;24(6):1311-1314.

51. Richie CA III, Briner WW Jr: Corticosteroid injection for treatment of de Quervain's tenosynovitis: A pooled quantitative literature evaluation. *J Am Board Fam Pract* 2003;16(2):102-106.

52. Weiss AP, Akelman E, Tabatabai M: Treatment of de Quervain's disease. *J Hand Surg Am* 1994;19(4):595-598.

53. Jirarattanaphochai K, Saengnipanthkul S, Vipulakorn K, Jianmongkol S, Chatuparisute P, Jung S: Treatment of de Quervain disease with triamcinolone injection with or without nimesulide: A randomized, double-blind, placebo-controlled trial. *J Bone Joint Surg Am* 2004; 86-A(12):2700-2706.

54. Peters-Veluthamaningal C, van der Windt DA, Winters JC, Meyboom-de Jong B: Corticosteroid injection for de Quervain's tenosynovitis. *Cochrane Database Syst Rev* 2009;3:CD005616.

55. Witczak JW, Masear VR, Meyer RD: Triggering of the thumb with de Quervain's stenosing tendovaginitis. *J Hand Surg Am* 1990;15(2):265-268.

56. Grundberg AB, Reagan DS: Pathologic anatomy of the forearm: Intersection syndrome. *J Hand Surg Am* 1985;10(2): 299-302.

57. Kardashian G, Vara AD, Miller SJ, Miki RA, Jose J: Stenosing synovitis of the extensor pollicis longus tendon. *J Hand Surg Am* 2011;36(6):1035-1038.

58. Mogensen BA, Mattsson HS: Stenosing tendovaginitis of the third compartment of the hand: Case report. *Scand J Plast Reconstr Surg* 1980;14(1):127-128.

59. Huang HW, Strauch RJ: Extensor pollicis longus tenosynovitis: A case report and review of the literature. *J Hand Surg Am* 2000;25(3):577-579.

60. McMahon MS, Posner MA: Triggering of the thumb due to stenosing tenosynovitis of the extensor pollicis longus: A case report. *J Hand Surg Am* 1994;19(4):623-625.

61. Ferreres A, Llusá M, García-Elías M, Lluch A: A possible mechanism of direct injury to the EPL tendon at Lister's tubercle during falls with the wrist fully extended. *J Hand Surg Eur Vol* 2008;33 (2):149-151.

62. **Roth KM, Blazar PE, Earp BE, Han R, Leung A: Incidence of extensor pollicis longus tendon rupture after nondisplaced distal radius fractures. *J Hand Surg Am* 2012;37(5):942-947.**

63. Engkvist O, Lundborg G: Rupture of the extensor pollicis longus tendon after fracture of the lower end of the radius: A clinical and microangiographic study. *Hand* 1979;11(1):76-86.

64. Hirasawa Y, Katsumi Y, Akiyoshi T, Tamai K, Tokioka T: Clinical and microangiographic studies on rupture of the E.P.L. tendon after distal radial fractures. *J Hand Surg Br* 1990;15(1):51-57.

65. Cooper HJ, Shevchuk MM, Li X, Yang SS: Proliferative extensor tenosynovitis of the wrist in the absence of rheumatoid arthritis. *J Hand Surg Am* 2009;34(10):1827-1831.

66. Moretta DN, Cooley RD Jr: Secrétan's disease: A unique case report and literature review. *Am J Orthop (Belle Mead NJ)* 2002;31(9):524-527.

67. Brown FE, Brown ML: Long-term results after tenosynovectomy to treat the rheumatoid hand. *J Hand Surg Am* 1988;13(5):704-708.

68. Drury BJ: Traumatic tendovaginitis of the fifth dorsal compartment of the wrist. *Arch Surg* 1960;80:554-556.

69. Hooper G, McMaster MJ: Stenosing tenovaginitis affecting the tendon of extensor digiti minimi at the wrist. *Hand* 1979;11(3):299-301.

70. Futami T, Itoman M: Extensor carpi ulnaris syndrome: Findings in 43 patients. *Acta Orthop Scand* 1995;66(6):538-539.

71. Nachinolcar UG, Khanolkar KB: Stenosing tenovaginitis of extensor carpi ulnaris: Brief report. *J Bone Joint Surg Br* 1988;70(5):842.

72. Ruland RT, Hogan CJ: The ECU synergy test: An aid to diagnose ECU tendonitis. *J Hand Surg Am* 2008;33(10):1777-1782.

73. Kip PC, Peimer CA: Release of the sixth dorsal compartment. *J Hand Surg Am* 1994;19(4):599-601.

74. Hajj AA, Wood MB: Stenosing tenosynovitis of the extensor carpi ulnaris. *J Hand Surg Am* 1986;11(4):519-520.

75. **Campbell D, Campbell R, O'Connor P, Hawkes R: Sports-related extensor carpi ulnaris pathology: A review of functional anatomy, sports injury and management. *Br J Sports Med* 2013;47(17):1105-1111.**

76. Spinner M, Kaplan EB: Extensor carpi ulnaris: Its relationship to the stability of the distal radio-ulnar joint. *Clin Orthop Relat Res* 1970;68(68):124-129.

77. Bishop AT, Gabel G, Carmichael SW: Flexor carpi radialis tendinitis: Part I. Operative anatomy. *J Bone Joint Surg Am* 1994;76(7):1009-1014.

78. Fitton J, Shea FW, Goldie W: Lesions of the flexor carpi radialis tendon and sheath causing pain at the wrist. *J Bone Joint Surg Br* 1968;50(2):359-363.

79. Parellada AJ, Morrison WB, Reiter SB, et al: Flexor carpi radialis tendinopathy: Spectrum of imaging findings and association with triscaphe arthritis. *Skeletal Radiol* 2006;35(8):572-578.

80. Gabel G, Bishop AT, Wood MB: Flexor carpi radialis tendinitis: Part II. Results of operative treatment. *J Bone Joint Surg Am* 1994;76(7):1015-1018.