

Extensor Tendon Injuries and Common Tendinopathies

Extensor tendon injuries have long been regarded as simple problems solved by the reapproximation of defects. Recent studies, however, have shown that extensor tendon injuries may pose difficult problems for the physician and significant functional impairment for the patient. (1,2) The delicate, dynamic balance of the extensor mechanism demands that an exacting knowledge of its form and function be gained before it can be successfully treated.

This chapter provides an overview of the anatomy and pathomechanics of common acute and chronic extensor tendon injuries along with indications, techniques, pitfalls, and rehabilitation principles involved in treating these syndromes. Common extensor tendinopathies of the wrist and hand are also discussed.

RELEVANT ANATOMY

The extensor mechanism of the digits is known for its complexity, subtlety, and interdependence of structures. The mechanism is powered by two components: extrinsic, radially innervated musculature arising from the forearm or elbow and intrinsic, ulnar-median-innervated musculature arising within the hand.

The extrinsic muscles and tendons arise proximal to the hand and pass under the extensor retinaculum, which divides into six compartments dorsally (Fig. 10.1). The extensor pollicis brevis (EPB) and abductor pollicis longus (APL) pass through the first dorsal compartment, which is subdivided by one or more septa. The second compartment contains the extensor carpi radialis longus (ECRL) and extensor carpi radialis brevis (ECRB). The extensor pollicis longus (EPL) passes through the third compartment and around Lister's tubercle to the distal phalanx of the thumb. The fourth compartment contains the extensor digitorum communis (EDC) tendons to the four digit and the extensor indicis proprius (EIP), which lies ulnar to the

EDC on the index finger. The fifth compartment includes the extensor digiti minimi (EDM). The sixth compartment has a subsheath, below the extensor retinaculum, that holds the extensor carpi ulnaris (ECU) firmly against the ulna (Fig. 10.2).

The extensor retinaculum is a pulley that prevents bowstringing of the extensor tendons. The extensor tendons (fourth compartment) at the wrist are covered with tenosynovium and pass over the metacarpals. These tendons are connected by the juncturae tendinum. The juncturae are responsible for simultaneous and uniform finger extension (Fig. 10.3) and prevent tendon retraction after a laceration occurs over the dorsum of the hand.

The tendons to the digits cross the metacarpal-phalangeal (MP) joints and are held in place by the sagittal bands, which pass from the tendon to the volar plate of the proximal phalanx and prevent subluxation (Fig. 10.4). The intrinsic muscles lie volar to the axis of rotation and flex the MP joint. The extrinsic muscles and tendons remain dorsal and extend the MP joint. The extensor tendon trifurcates over the proximal phalanx to form the central slip and two lateral slips (Fig. 10.5). The lumbricals arise from the tendons of the flexor digitorum profundus (FDP) on the radial side of each digit. Distally, the lumbrical tendon remains volar to the deep transverse metacarpal ligament, passes radial to the MP joint, and divides to contribute to the central and lateral slips of the extensor mechanism (Fig. 10.6).

The three palmar interossei arise from the metacarpal shaft, pass dorsal to the deep transverse metacarpal ligament, and send one slip to the base of the proximal phalanx; the other slip mimics the insertion of the lumbrical. The four dorsal interossei arise from the two metacarpal shafts that border them. The dorsal interossei provide the same insertion pattern as the palmar interossei but pass above the MP axis. There is a considerable variation in the

Figure 10.1. Extensor retinaculum with the six dorsal compartments. *EPB*, extensor pollicis brevis; *APL*, abductor pollicis longus; *ECRL*, extensor carpi radialis longus; *ECRB*, extensor carpi radialis brevis; *EPL*, extensor pollicis longus; *ECU*, extensor carpi ulnaris; *EDM*, extensor digiti minimi; *EDC*, extensor digitorum communis; *EIP*, extensor indicis proprius.

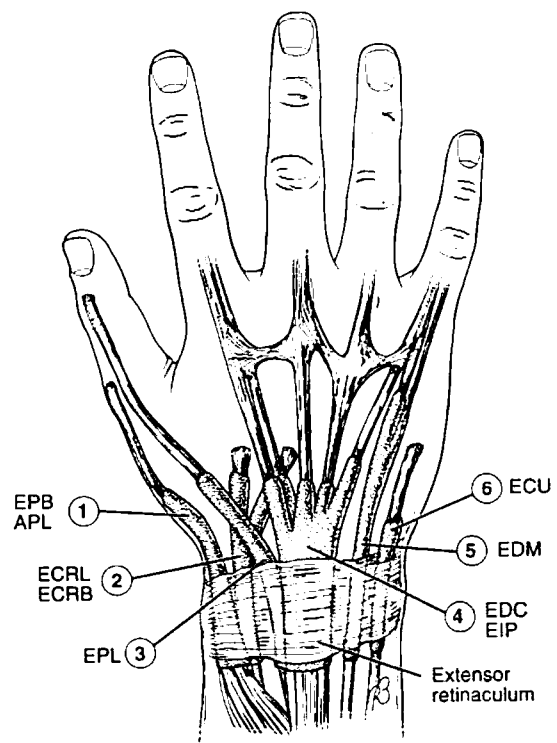


Figure 10.2. ECU and subsheath in a cadaveric dissection.



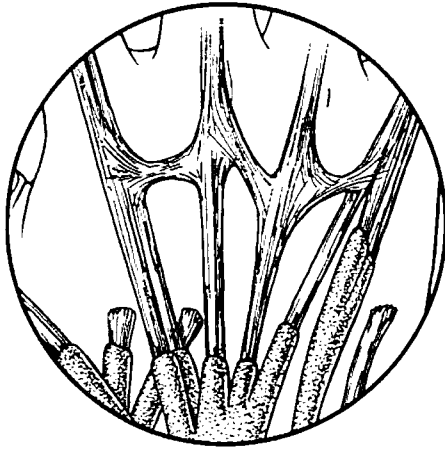


Figure 10.3. Juncturae of the wrist.

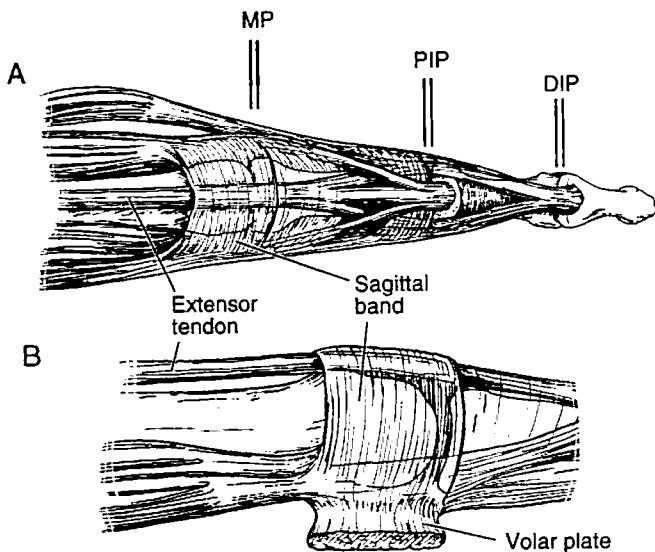


Figure 10.4. A. The sagittal band holds the extrinsic tendons in place at the MP joint. B. Lateral view of the sagittal band at the MP joint. PIP, proximal interphalangeal joint; DIP, distal interphalangeal joint.

amount of insertion into the proximal phalanx or extensor mechanism between the digits (3) (Fig. 10.7). The functions of the dorsal and volar interossei are abduction and adduction, respectively.

On the dorsum of the proximal phalanx, the central slip is formed by the EDC and small contributions from the lumbrical and both interossei. These structures insert on to the base of the middle phalanx. The intrinsic tendons (lumbrical and interossei) form the lateral bands, which receive a small contribution of cross-over fibers from the central slip, e.g., the EDC. These lateral bands join on the dorsum of the middle phalanx to form the terminal tendon, which inserts onto the base of the distal phalanx (Fig. 10.5).

The extensor mechanism is stabilized by several accessory structures. The transverse retinacular ligament acts at the level of the proximal interphalangeal (PIP) joint to re-

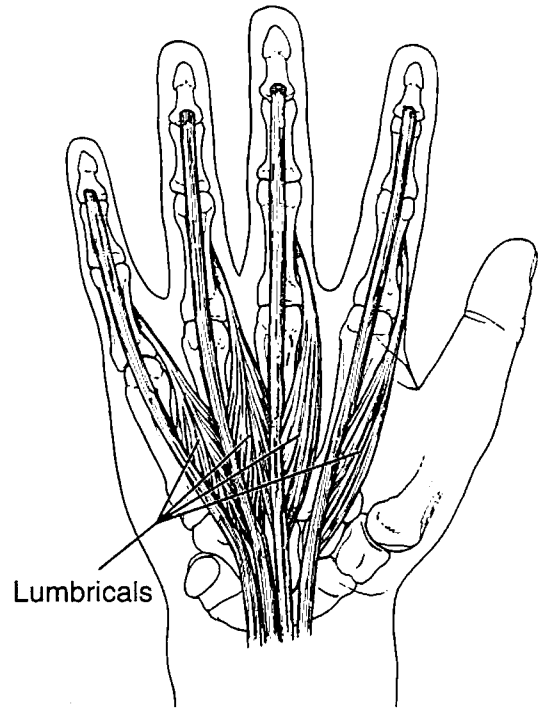


Figure 10.5. Lumbricals of the hand.

strain dorsovolar translation of the lateral bands. More distally, the triangular ligament consists of transverse fibers holding the conjoined lateral bands together dorsally. It is these conjoined lateral bands that form the terminal extensor tendon. The oblique retinacular ligament runs from the flexor sheath, volar to the PIP joint to join the terminal tendon, inserting on the dorsal base of the distal phalanx. This ligament augments distal interphalangeal (DIP) extension with active extension of the PIP joint (4).

The extensor system of the thumb includes one muscle that inserts on each joint: the EPL inserts on the distal phalanx; EPB, on the proximal phalanx; and APL, on the base of the metacarpal. The extensor function of the thumb does not depend on the dynamic interrelationship of its components, though the dorsal hood does bear some resemblance to that of the finger.

The extensor mechanism has been divided into eight zones by Kleinert and Verdan (5). Disruption of the tendon at each level causes a different type of injury. The next section reviews injuries to zones I through VIII (Fig. 10.8).

ACUTE EXTENSOR TENDON INJURY

Zone I: Distal Interphalangeal Joint (Mallet Injuries)

Zone I extends from the most distal insertion of the extensor mechanism to the attachment of the central slip on the proximal end of the middle phalanx. Disruption of the terminal tendon or conjoined lateral bands at the DIP joint causes an extensor lag of the distal phalanx. This has been referred to as a drop, baseball, cricket, or (most com-

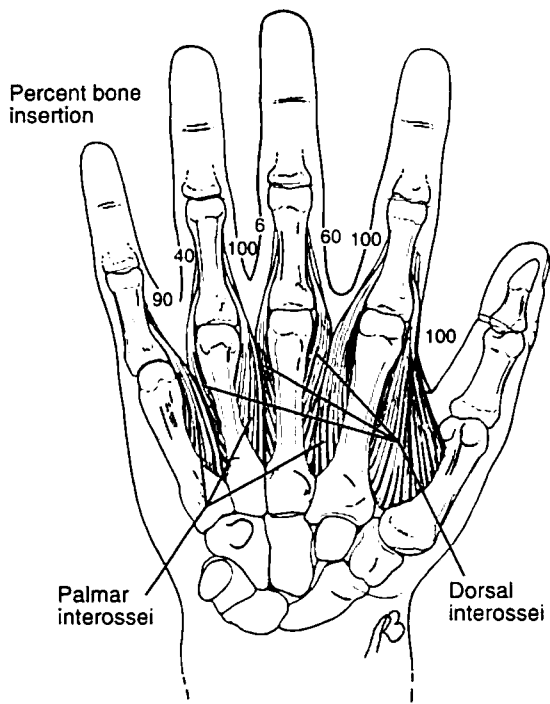


Figure 10.6. Dorsal and palmar interossei and the amount of insertion into the proximal phalanx or extensor mechanism.

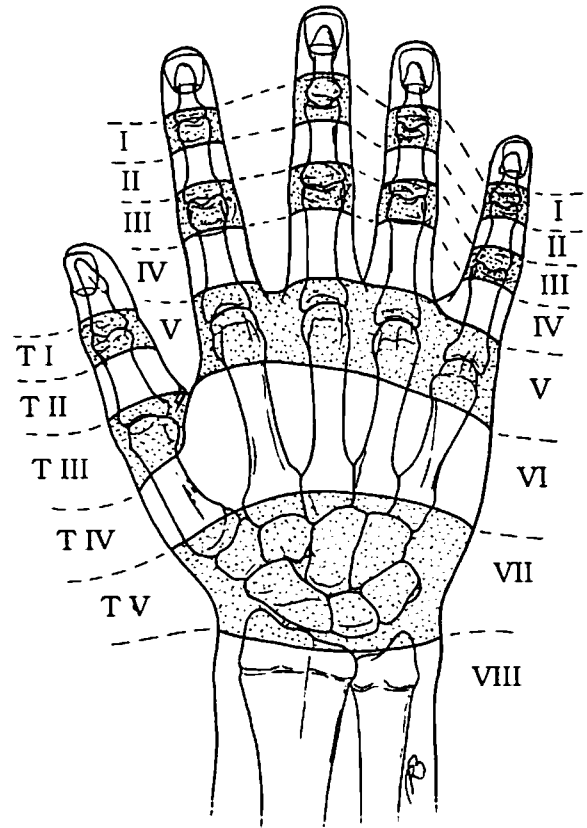


Figure 10.8. Zones of extensor tendon injuries.

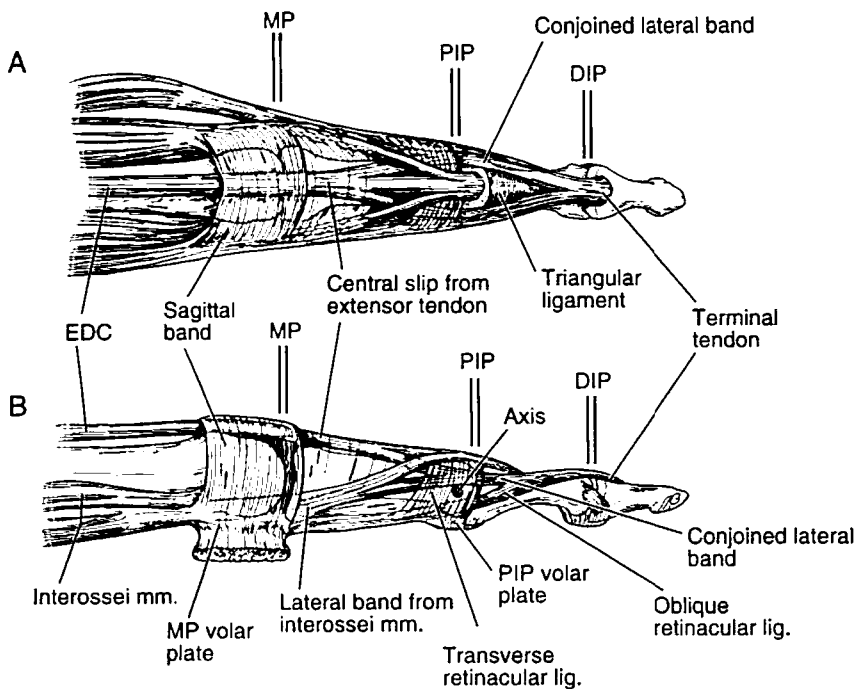


Figure 10.7. AP (A) and lateral (B) views of the extensor mechanism of the digit. *PIP*, proximal interphalangeal joint; *DIP*, distal interphalangeal joint.

monly) mallet finger. This injury is seen in factory workers and as sports-related injuries. A mallet finger may occur spontaneously in a patient with rheumatoid arthritis or with minimal trauma to the digit (6). The mechanism of in-

jury involves a sudden flexion force on an extended digit. Less commonly, this injury may be produced by a hyperextension force, resulting in a dorsal fracture and subsequent mallet finger (Fig. 10.9).



Figure 10.9. Dorsal mallet fracture.

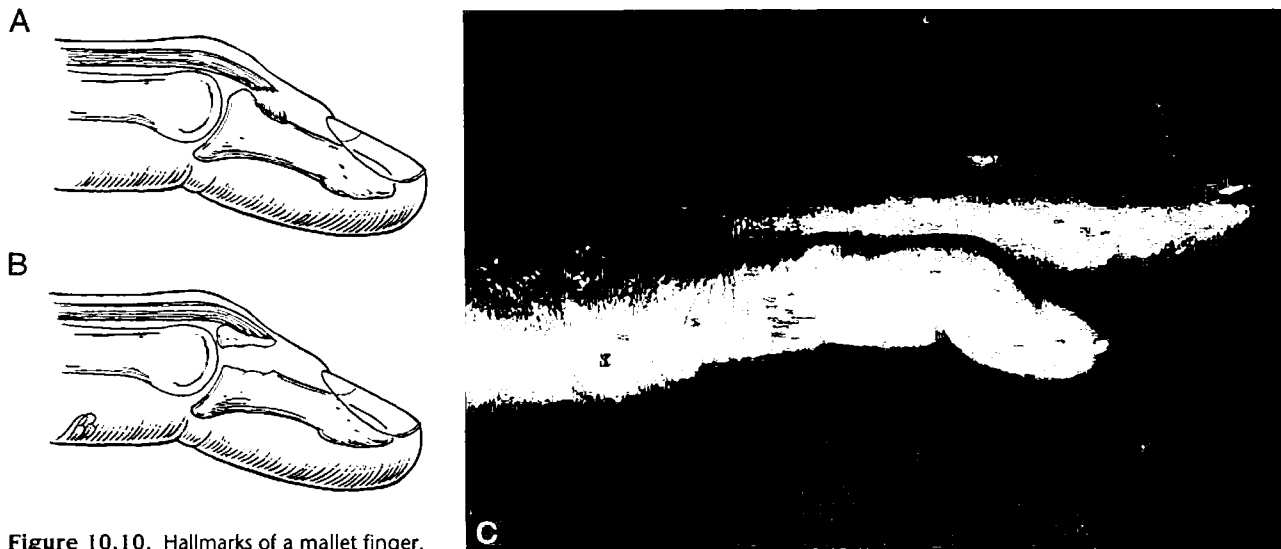


Figure 10.10. Hallmarks of a mallet finger.

Warren et al. (7) localized an avascular critical zone at 11 to 16 mm from the insertion of the lateral bands, corresponding to an area of compression over the head of the middle phalanx when in flexion. These findings suggest that vascular compromise may also play a role in the cause of a mallet finger.

The hallmark finding of a mallet finger is the flexed posture of the DIP joint and loss of active terminal extension (Fig. 10.10). The degree of deformity can vary, and some authors (2,7) have noted that the appearance of the flexion deformity after trauma to the DIP joint may be delayed for several days. The chronic mallet finger has a flexion deformity at the DIP joint and hyperextension of the PIP joint. This is known as a secondary swan-neck deformity.

We recommend that radiographs be obtained with all acute and chronic mallet fingers. The lateral view can reveal a fracture fragment and joint subluxation. The AP view may show an associated collateral ligament injury (Fig. 10.11).

Doyle (2) classified mallet fingers into four types. A type I injury is caused by hyperflexion secondary to closed or blunt trauma. This injury involves disruption or stretching of the extensor tendon just proximal to its insertion and may or may not include a small avulsion fracture. A type II injury is a laceration that divides the terminal ten-

don. A type III mallet finger is characterized by a deep abrasion involving loss of the overlying soft tissue. Type IV injuries can be subdivided into three groups: (a) a transepiphyseal plate fracture in children, (b) a fracture involving 20 to 50% of the articular surface produced by a hyperflexion injury, and (c) a fracture produced by hyperextension that involves more than 50% of the articular surface and demonstrates volar subluxation of the distal phalanx (Fig. 10.12).

Closed acute mallet injuries are treated nonoperatively. The digit is immobilized with the DIP joint in extension by using a prefabricated plastic splint finger splint (8), a foam-padded aluminum splint, or a custom-made fingertip protector (Fig. 10.13). The splint should permit unrestricted motion of the PIP joint. Kaplan's (9) anatomic studies demonstrate relative relaxation of the extensor mechanism over all three joints when only the DIP joint was held in extension. More extensive splinting or Smillie's (10) serial casting method may be required for the active or uncooperative patient.

Treatment requires 6 to 8 weeks of continuous splinting, followed by 2 weeks of night splinting as the patient begins to resume flexion. The splint is applied during the weaning period if any recurrence or deformity is noted. We recommend that the splint be applied as soon as possible



Figure 10.11. Stress radiograph showing injury to the collateral ligaments of the DIP joint.

following the initial injury, although satisfactory results have been recorded with delays of 4 to 6 weeks. McFarlane and Hampole (11) found acceptable results up to 3 months postinjury, and Garberman et al. (12) showed good results in patients treated early and on a delayed basis.

The unusual transepiphyseal mallet fracture in children is caused by the attachment of the extensor tendon to the epiphysis of the distal phalanx. Treatment includes a closed reduction that extends the digit. The finger is placed in a splint for 3 to 4 weeks.

COMPLICATIONS

Complications with the use of a splint include ulceration and maceration of the skin. Extreme hyperextension and pressure over the dorsum of the joint must be avoided to prevent these complications. Patients are encouraged to keep the skin clean and the splint dry; but they must be warned to keep the DIP joint in extension when removing the splint.

OPERATIVE

Open repair of the closed injury is rarely necessary and has not been found to be efficacious, given the thin and tenuous nature of the extensor tendon at this level. Kirschner (K) wire fixation crossing the DIP obviates the need for an external splint and may be useful in patients

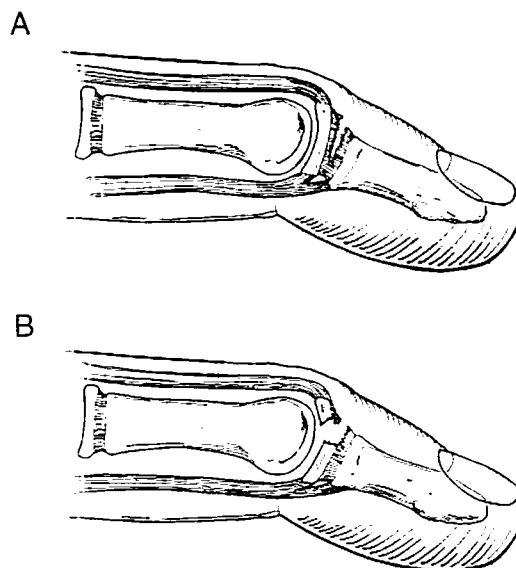


Figure 10.12. Transepiphyseal fracture in children, resulting in a mallet finger.

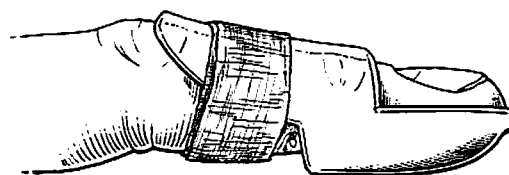


Figure 10.13. Custom-made fingertip protector for a mallet finger.

such as dentists, surgeons, and others whose daily activities do not allow wearing a splint (Clinical Table). The K wire is kept in for 6 weeks, after which motion is begun. Potential complications, such as a nail bed injury and osteomyelitis, must be explained to the patient before the K wire is used.

Open mallet finger injuries are treated by the reapproximation of the tissues, using the figure eight or roll suture technique. The DIP is splinted in extension postoperatively. Sutures are removed at 10 to 14 days, and splinting is continued as in a closed injury (Fig. 10.14). More significant open injuries with loss of skin and tendon require reconstructive procedures that will not be discussed here.

OUTCOME

The mallet finger with a small avulsion fragment can be treated in a closed fashion. Several authors have shown that the presence of this small piece of bone does not significantly affect the outcome of treatment (6,13,14). Wehbe and Schneider (14) recommend extensive splinting of all types of mallet fractures. They believe that joint congruity is not essential, since remodeling has been observed to lead to a functional and painless joint. Crawford (15) noted that splinting in hyperextension should be avoided in the presence of a significant fracture, as this may accentuate

Clinical Table: Extensor Tendon Injuries and Common Tendinopathies

Procedure	Indications	Technique	Anatomy	Pitfalls
Stack splint mallet repair	<ul style="list-style-type: none"> Mallet finger (flexed DIP) 	<ul style="list-style-type: none"> Occupational therapy Figure eight or roll suture or Kirschner wire repair 	<ul style="list-style-type: none"> Terminal tendon laceration 	<ul style="list-style-type: none"> Ulceration and maceration
Boutonniere repair	<ul style="list-style-type: none"> Boutonniere PIP joint flexion with DIP joint hyperextension 	<ul style="list-style-type: none"> K-wire immobilization and primary repair central slip 	<ul style="list-style-type: none"> Discontinuity of the central slip vs. crush injury 	<ul style="list-style-type: none"> Stiff chronic boutonniere with missed diagnosis
Dislocated extrinsic extensor tendon repair	<ul style="list-style-type: none"> Extensor tendon subluxation 	<ul style="list-style-type: none"> Splinting in extension vs. acute surgical repair 	<ul style="list-style-type: none"> Radial sagittal band 	<ul style="list-style-type: none"> Missed diagnosis
Extensor tendon laceration zone VI repair	<ul style="list-style-type: none"> Extensor tendon laceration 	<ul style="list-style-type: none"> Tendon repair with non-absorbable sutures 	<ul style="list-style-type: none"> Extensor tendon and juncturae 	<ul style="list-style-type: none"> Failure of splinting to juncturae
Chronic mallet finger repair	<ul style="list-style-type: none"> Flexed DIP Fixed DIP contracture 	<ul style="list-style-type: none"> Tenodesis K-wire immobilized arthrodesis PIP 	<ul style="list-style-type: none"> Repair of shortened extensor mechanism Degenerative DIP 	<ul style="list-style-type: none"> Wound breakdown Infection
Littler reconstruction (24)	<ul style="list-style-type: none"> Swan-neck deformity Flexed DIP Hyperextended PIP 	<ul style="list-style-type: none"> Reconstructing oblique retinacular ligament 	<ul style="list-style-type: none"> Fixed contracture and intrinsic tightness in the PIP 	<ul style="list-style-type: none"> Flexor tendon scarring and recurrence of deformity
Extensor tenolysis	<ul style="list-style-type: none"> Fixed tendons with limited motion 	<ul style="list-style-type: none"> Release adhesions to skin, tendons, bones, and joints (26) 	<ul style="list-style-type: none"> Extensor tendons 	<ul style="list-style-type: none"> Rupture tendons or inadequate release
First dorsal compartment release	<ul style="list-style-type: none"> De Quervain's tenosynovitis 	<ul style="list-style-type: none"> Surgical decompression of the first compartment 	<ul style="list-style-type: none"> APL EPB 	<ul style="list-style-type: none"> Cut superficial radial nerve Missed release Independent septae

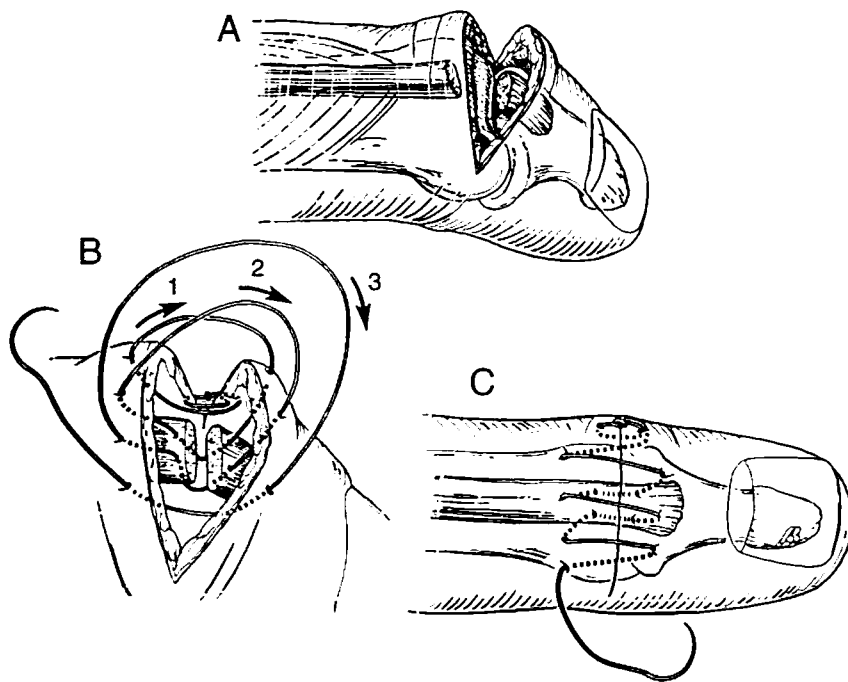


Figure 10.14. Roll suture technique for open mallet repair.

the subluxation. Some authors have recommended operative treatment for any fracture involving more than one-third of the articular surface. Most authors currently recommend open treatment to restore joint congruity only when joint subluxation has occurred (2,15,16).

If volar subluxation is present and the fracture is greater than 50%, the joint is exposed dorsally and a single K wire is placed longitudinally through the distal phalanx to reduce and hold the DIP joint. If the fracture fragment is not well apposed, a pullout suture or wire may be used and brought out over a button volarly (2,15,16). The DIP joint is then splinted for 6 weeks before the K wire is removed and rehabilitation begun.

Operative repair of mallet deformities can be challenging and often unnecessary. The surgeon must guard against comminution of the fracture fragment, resulting in an inability to reattach the extensor mechanism, skin necrosis, and a decrease in the range of motion at the DIP joint. Clement and Wray (17) reported a decrease in flexion of the PIP joint after DIP surgery. They also noted an incidence of cold intolerance and persistent pain.

Mallet thumb injuries are much less common than are mallet finger injuries, but the principles of treatment are similar. Though some surgeons recommend operative repair, Doyle (2) prefers splinting in extension for the closed injury. A laceration at the level of the interphalangeal joint lends itself to repair of the EPL tendon. A fracture through the joint with a pulloff of the EPL insertion requires operative reduction and internal fixation.

Zone II: Middle Phalanx

Zone II involves the middle phalanx. Lacerations at this level are seldom complete, owing to the width and curvature of the extensor mechanism. Zone II lacerations are treated similarly to zone I injuries, with figure eight or roll sutures and splinting of the DIP joint for 6 weeks. Lacerations of less than 50% do not require repair, and active motion may begin following wound healing with a frayed tendon program.

Zone III: PIP Joint (Boutonniere Injury)

PIP joint flexion with DIP joint hyperextension is the hallmark of the boutonniere deformity (Fig. 10.15). This results from discontinuity in the central slip of the extensor mechanism at the level of the PIP joint, with volar migration of the lateral bands. The injury is caused by laceration, forced flexion at the PIP joint, crush injuries, volar PIP dislocations, and synovitis from inflammatory arthritis.

Diagnosis in the acute setting may be difficult, and a high degree of suspicion is required. Suggestive findings include swelling and tenderness at the base of the dorsal middle phalanx with weak PIP joint extension against resistance. Volar dislocations of the PIP joint should alert the physician to the possibility of a late boutonniere injury. X-rays rarely show fracture fragments involving the dorsum of the middle phalanx. Acutely, the patient is often able to

extend the PIP joint through the lateral bands. With continued use, the triangular ligament stretches and the transverse retinacular ligaments progressively contract. The lateral bands migrate volar to the axis of the PIP joint and begin to act as PIP flexors. This new alignment concentrates the force of the extensor mechanism at the DIP joint, which, along with the MP joint, begins to hyperextend. In a chronic boutonniere, the deformity becomes fixed secondary to contractures of the transverse retinacular, oblique retinacular, collateral ligaments, and volar plate. Fixed deformities are discussed later in this chapter.

The acute closed boutonniere deformity is treated with immobilization of the PIP joint in extension. This may be accomplished with a splint or a transarticular K wire. The PIP joint is immobilized and active flexion of the DIP joint is encouraged to prevent lateral band adhesions and contractures of the oblique retinacular ligament (18). The splint is worn for 6 weeks; active PIP flexion is begun, and the joint is protected in extension for 2 weeks more between exercises. Initially, some digits may not allow full extension, and serial splinting or casting may be required (2). The goal of treatment is to prevent a stiff, chronic boutonniere deformity.

Surgical treatment of an acute closed boutonniere deformity has been advocated for avulsion fracture, subacute boutonniere deformity in a young person (19), volar dislocation of the PIP joint, and lacerations across the PIP joint (20). Bony reattachment or excision of the fragment and central slip reattachment is the treatment of choice for an avulsion fracture (19). If no fracture is present, the central slip is explored and repaired, as are other structures requiring repair (20). In both circumstances a K wire is used to place the PIP joint in extension for 3 weeks.

Lacerations in the region of the PIP joint may be intra-articular and may require irrigation, débridement, and primary repair of the central slip and/or lateral bands with pullout sutures; K wire immobilization of the PIP joint in extension may also be necessary (Fig. 10.16). Several reconstructive procedures, using local tissues, are available to avoid a chronic deformity if there is tissue loss involving the extensor mechanism (2).

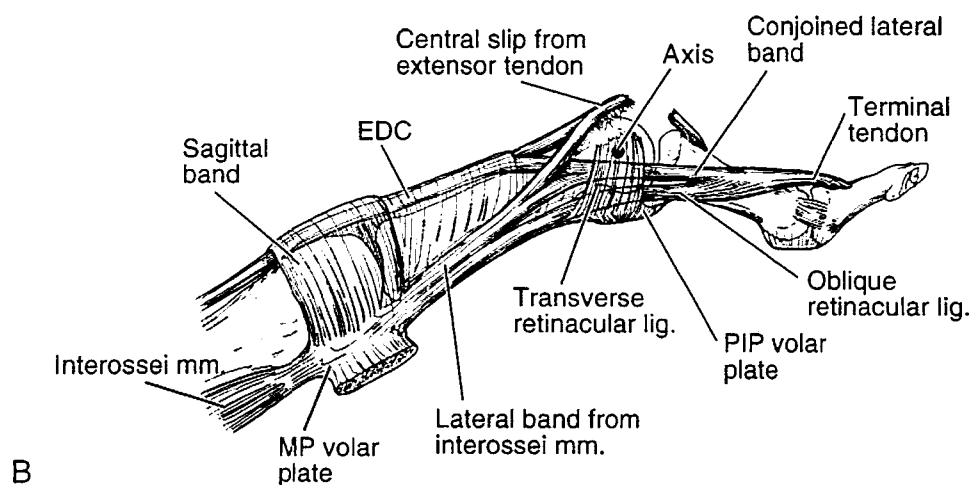
Zone IV: Proximal Phalanx

Lacerations in zone IV seldom result in complete transection, because of the broad, circumferential orientation of the extensor mechanism at this level. Partial lacerations are repaired primarily, and the patient is started on early range of motion protocols. Repairs involving the central slip are maintained in splints, with the PIP joint extended for 6 weeks to avoid lengthening of the central slip and developing a boutonniere-type deformity.

Zone V: MP Joint

The MP joint is a common location for abrasions and lacerations in the hand because of its prominence. A pen-

Figure 10.15. A. Hallmark of the boutonniere deformity. B. The lateral bands are subluxed in a boutonniere deformity.



etrating injury at this level may affect the MP joint. All wounds must be inspected, cleaned, and débrided. Any small puncture wound in this location must be considered a human bite until proven otherwise. A human bite is a contaminated wound, usually polymicrobial involving gram-positive bacteria (streptococci, staphylococci) or gram-negative *Eikenella*. The wound should be extended, débrided, and irrigated. If the wound is identified within 6 h of injury, it may be closed; otherwise it is left open, and secondary tendon repair, if necessary, is planned for 4 to 7 days later. In simple, clean lacerations, the tendon is repaired primarily. Repair of the sagittal bands is performed to prevent subluxation of the extensor tendon. Dynamic splinting in an intrinsic-minus position and early motion are recommended after repair of the sagittal band.

Subluxation or dislocation of the extrinsic extensor tendon occurs at this level secondary to injury of the sagittal bands from a laceration, direct blow, or forceful flexion or extension stress. Most commonly seen in the middle finger, this involves a tear in the radial side of the sagittal

band and ulnar subluxation of the tendon. On examination, there is tenderness, swelling, and an inability to actively extend the MP joint. Ulnar deviation of the involved digit may be noted in flexion. If the extensor tendon is passively extended, the tendon will reduce, and the patient will then be able to actively maintain its position.

Initial treatment of a closed extensor tendon subluxation at the MP joint should consist of splinting in extension. Acute surgical repair of the radial sagittal band is preferred. Postsurgery, the MP joint is splinted in maximum extension for 4 weeks. Active motion of the PIP and DIP joints is begun at 2 weeks, with the MP extended.

Zone VI: Metacarpals

The extensor tendons over the dorsum of the hand are superficial and thus commonly injured. Prognosis after operative repair is good, but tendon lacerations associated with more severe injuries have an increased risk of adhe-

sions. Tendons in this region may be repaired with nonabsorbable sutures and then splinted with the MP joint in neutral and the wrist slightly extended. Because of the juncturae, all the fingers should be splinted in EDC injuries. Dynamic splinting is started early, and protection is continued for 6 weeks.

Zone VII: Wrist

Treatment of extensor tendon injuries at the wrist is complicated by the presence of the extensor retinaculum. Tendon lacerations are routinely repaired using standard methods. Depending on the location of the repair, some portion of the extensor retinaculum must be excised to avoid impingement. Repair of the retinaculum proximal or distal to the tendon repair must remain intact to prevent bowstringing of the tendons. Dorsal lacerations may involve branches of the superficial radial or ulna nerve. These nerves should be repaired acutely at the time of tendon repair. Tendons often retract proximally after laceration at this level and may require longitudinal extension of the wound to identify the two cut ends.

After surgery, the surgeon must immobilize the wrist in extension and the MP joints in neutral. Multiple tendon injuries respond better to early motion, which promote excursion of the tendons beneath the retinaculum. Dynamic extension splinting is begun at 10 days. Protected range of motion exercises are begun between 4 and 6 weeks.

Zone VIII: Distal Forearm

Injuries to the distal forearm are caused by penetrating trauma. Deep penetration should caution the physician to perform a complete neurovascular examination. Diagnosis is confirmed with the loss of the tenodesis effect, causing digital extension of wrist flexion. Longitudinal extension of the wound and meticulous exploration are required to achieve satisfactory repair at this level. Injuries at the musculotendinous junction are repaired by approximating the tendon to the fibrous septa in the muscle belly. Intramuscular lacerations are reapposed using absorbable sutures. Postsurgery, the wrist is immobilized in extension, with the MP joints in slight flexion. The elbow is immobilized if the tendons involved have their origin proximal to the elbow. Protected range of motion is started at 4 weeks. The splint may be removed at 6 weeks.

EXTENSOR TENDON RECONSTRUCTION

Chronic Mallet Finger

An extensor lag at the DIP joint is treated with a splint up to 12 weeks postinjury, although the true limits of this time frame have yet to be established. Dysfunction with chronic mallet finger may be minimal and tolerable. Patients who have pain, deformity, and functional loss may desire surgical correction.

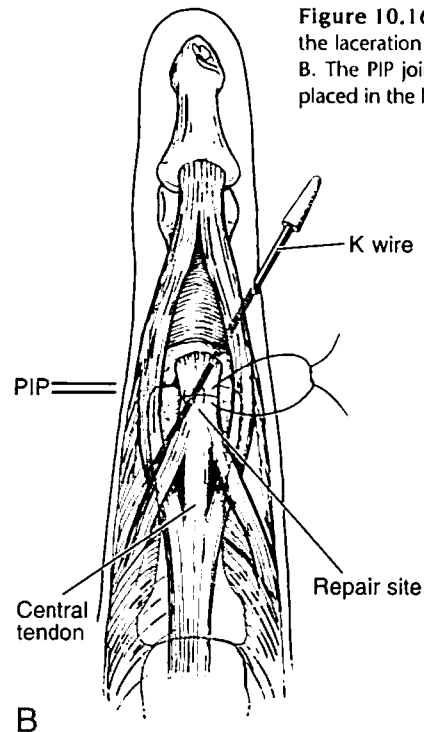
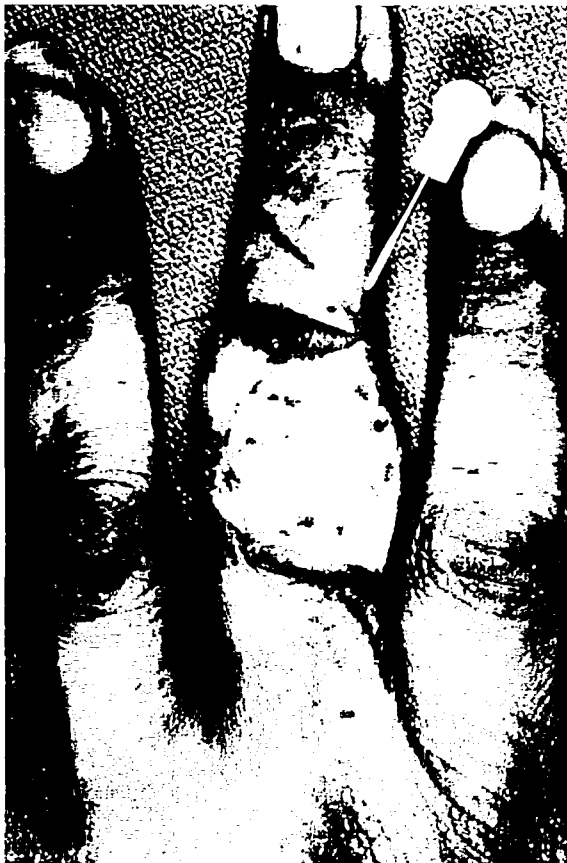


Figure 10.16. A. The K wire is in place, and the laceration of the central slip is visualized. B. The PIP joint is pinned, and the suture is placed in the lacerated extensor mechanism.

Correction of a chronic mallet finger requires a repair that shortens the extensor mechanism. Through a dorsal approach to the DIP joint, this may be accomplished by three methods: (a) a segment of scar tissue may be resected, followed by end-to-end repair of the tendon; (b) the tendon may be imbricated on itself; and (c) the insertion of the scarred tendon may be divided, advanced, and reattached to bone (21). An alternative procedure is a tenodesis. The skin and extensor mechanism are excised in an elliptical fashion, and the edges are reapproximated as described by Iselin et al. (22). The joint is stabilized in extension with a longitudinal K wire and protected with a splint for 8 weeks.

Another technique used to treat chronic mallet fingers is the release of the central slip (23). Through a midlateral incision at the level of the PIP joint, the lateral band is identified; this structure and the entire extensor mechanism are elevated. The insertion of the central slip is released from the proximal middle phalanx. Postsurgery, the DIP joint is splinted in extension and the PIP joint is held at 30° flexion for 3 weeks, after which PIP motion is begun, with the DIP joint being held in extension for an additional 4 weeks. This procedure has the effect of moving the extensor mechanism proximal and tightening the lateral bands. It will also reduce tone at the PIP joint, avoiding hyperextension.

The procedures described all require a congruent and supple DIP joint. Chronic repair of the extensor mechanism, especially following mallet fractures, can be difficult and frustrating. The bony alignment may be restored; but contractures of the joint capsule, collateral ligaments, and volar plate may limit function. Fixed deformities, degenerative changes, and joint malalignment are all indications for arthrodesis of the DIP joint. We perform an arthrodesis with either K wires or a Herbert screw. An incision is made dorsally; and using a rongeur, the articular surface is removed. K wires are placed retrograde, whereas the Herbert screw may be placed antegrade under fluoroscopic guidance.

Swan-Neck Deformity

A swan-neck deformity is a flexed DIP joint with hyperextension at the PIP joint (Fig. 10.17). This deformity is caused by a dynamic imbalance that may be an interruption of the distal extensor mechanism, which focuses its force at the middle phalanx, or an incompetence of the volar plate at the PIP joint. The result is hyperextension of the PIP joint, leading to dorsal displacement of the lateral bands, which in effect lengthens the extensor mechanism. Factors leading to a swan-neck deformity include chronic mallet finger, fracture malunion, volar plate injury to the PIP joint, spasticity, rheumatoid arthritis, and ligamentous laxity.

Splinting and exercise may be used to relieve fixed contractures and intrinsic tightness, but they cannot correct a swan-neck deformity (21). Correction must limit hy-

perextension at the PIP joint and augment DIP extension. This is accomplished by constructing an oblique retinacular ligament analog and securing it in a volar position to prevent PIP hyperextension and create passive extension of the DIP joint as the PIP is actively extended.

The original technique described by Littler (24) made use of the lateral band. Thompson et al.'s (4) modification employs a free tendon graft. A dorsolateral approach is used to expose the lateral band. A hole is created in the anteroposterior direction at the base of the distal phalanx and transversely at the base of the proximal phalanx. An appropriate tendon graft (usually palmaris or plantaris) is secured through the distal phalanx using pullout sutures over a button or a mini-Mitek. The graft follows the course of the lateral band volarly and proximally across the middle phalanx. The graft passes deep to the neurovascular bundle to cross anteriorly to the opposite side of the proximal phalanx, where it is passed through the transverse hole and secured over a second button. Before securing the graft proximally, the surgeon sets the appropriate tension by pulling the graft until the DIP joint is in full extension and the PIP joint is held in approximately 20° of flexion. A K wire may be employed to hold the PIP joint in this position. K wires are removed at 4 weeks, and the finger is placed in a dorsal splint in the same position. Active flexion is also started. The splint is discontinued at 8 weeks, and active extension is begun. The patient is told not to attempt to stretch the PIP joint into full extension.

The major concern postoperatively is avoiding flexor tendon scarring and the recurrence of deformity (21). A well-supervised rehabilitation program is imperative. Preoperative patient selection is important. If the PIP joint is severely damaged, is deformed, or has a fixed contracture, the surgeon may wish to consider arthroplasty or an arthrodesis instead.

Chronic Boutonniere Deformity

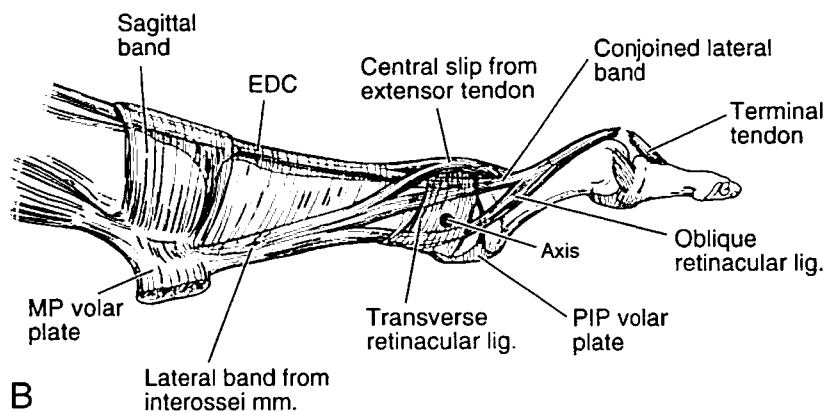
Boutonniere deformities may lengthen the central slip, leading to increased volar migration and rigid fixation of the lateral bands. The deformity becomes fixed with a contracture of the transverse retinacular and oblique retinacular ligaments, the volar plate, and the collateral ligaments of the PIP joint. The treatment plan is determined by the PIP joint deformity and whether it is supple or fixed.

Many flexible boutonniere deformities will respond to splinting and therapeutic intervention (21). Active-assisted extension of the PIP will stretch volar structures and increase the tenodesis effect on the DIP joint. Active flexion of the DIP joint with the PIP joint held in extension will stretch the lateral bands and oblique retinacular ligaments.

The wide variety of procedures used to treat this deformity is a testament to the difficulty of successful treatment. Most of the procedures fall into one of four categories: anatomic repair of the central slip, reconstruction using the lateral bands or other local tissues, tendon graft,



Figure 10.17. Hallmarks of the swan-neck deformity.



or redistribution of joint forces. Burton (21) describes a procedure to realign joint forces. Using a dorsal bayonet incision centered over the PIP joint, the extensor tendon is divided over the proximal portion of the middle phalanx. This effectively lengthens the lateral bands, allowing proximal migration of the extensor mechanism, increasing tone in the central slip. The lateral bands maintain their dorsal positioning. Postsurgery, the PIP joint is splinted in extension, but active-assisted range of motion exercises continue. We perform the Elliott (25) repair for a flexible deformity (Fig. 10.18).

In a fixed deformity, the first priority is the reestablishment of passive motion. Dynamic or serial splinting and exercise may help. Operative release of the joint may become necessary, followed by staged reconstruction of the extensor mechanism. If there is evidence of degenerative changes or extensive fibrous ankylosis, we consider arthroplasty or arthrodesis.

Extensor Tenolysis

When extensor tendons become fixed at points other than the anatomic insertions (tenodesis), motion is lim-

ited. The simple release of extensor tendon adhesions (tenolysis) is the preferred procedure; but this may be inadequate if other anatomic structures are involved. Joint stiffness and contractures may necessitate capsule and collateral ligament release.

A technique of extensor tenolysis has been well described (26). We use a dorsal approach to the digit for maximal exposure. Adhesions between the skin and extensor tendon are lysed as the exposure is developed. Beginning with the proximal phalanx, the central and lateral slips are elevated, and the interval between the two is released at the level of the PIP joint. This provides access to the extensor mechanism and the dorsal capsule. The terminal extensor tendon is freed along its radial and ulnar borders to improve access to the dorsal capsule of the DIP joint (Fig. 10.19). If capsulotomies do not restore range of motion, collateral ligament release may be necessary. Partial collateral ligament release is preferred to preserve joint stability. Passive and active motion are tested before the completion of the soft tissue procedures.

Another approach to overcoming adhesions of the extensor mechanism is a tendon release. Extrinsic extensor tendon release is an attempt to separate the extrinsic and

intrinsic systems. Excising the central portion of the extensor mechanism at the level of the proximal phalanx isolates the extrinsics as MP extensors and the intrinsics as interphalangeal extensors (21).

Full range of motion is started immediately postsurgery. Narcotic analgesia, indwelling catheters, and transcutaneous electrical nerve stimulation (TENS) are employed as required to reduce the pain of motion (27). Continuous passive motion (CPM) and dynamic splinting may assist with range of motion, but active use of the extensor tendons is essential. A well-supervised therapeutic regimen and a motivated patient are necessities.

Traumatic Tendon Subluxation at the MP Joint

Chronic extensor tendon subluxation causes impairment owing to the loss of active MP joint extension, ulnar drift of the involved digit, and secondary hyperextension of the PIP joint. Multiple operative techniques have been described to recentralize the extensor tendon (28). Most involve the creation of some type of check rein on the radial side of the tendon to resist dislocation in flexion. This is combined with limited ulnar-sided releases as needed. Postsurgery, the MP joint is splinted in extension for 4 weeks. PIP joint motion is allowed after wound healing to help with tendon excursion.

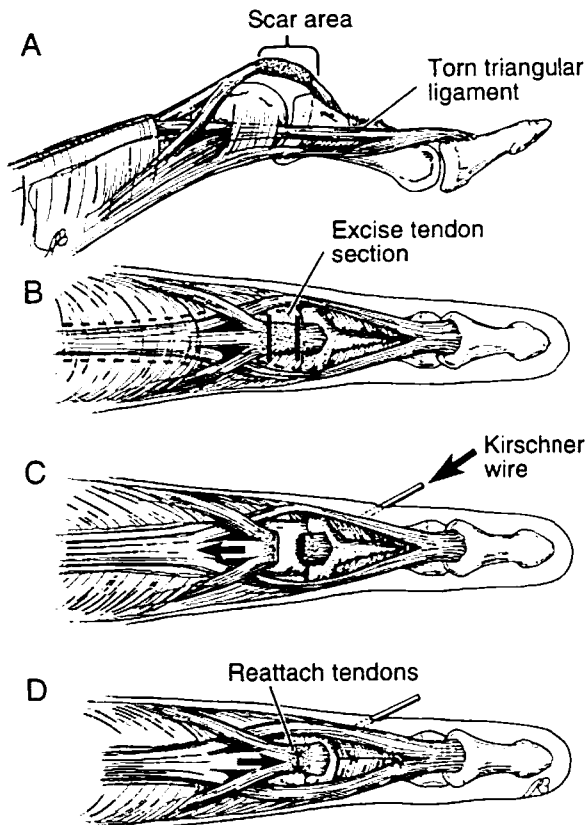


Figure 10.18. The Elliott repair of a chronic boutonniere deformity.

Proximal Reconstruction

Patients with a loss of MP joint or wrist extension display significant disability. Reconstructive procedures may be attempted to obtain a functional wrist. Direct delayed repair will be hampered by the presence of myotatic contractures. The options for reconstruction are tendon transfers and interpositional grafts. Transfers are generally preferred if a suitable donor exists. The patient's wrist is immobilized for 4 to 6 weeks before motion is started.

COMMON EXTENSOR TENDINOPATHIES

De Quervain's Tenosynovitis

De Quervain's tenosynovitis is a stenosing tenosynovitis of the first dorsal compartment. APL and EPB pass through a fibro-osseous tunnel formed by a groove in the radial styloid and the overlying retinaculum. Symptoms include pain over the radial styloid worsened with thumb or wrist motion, swelling, and occasional crepitus or triggering. A positive Finkelstein test (reproduction of pain with ulnar deviation of the wrist while the thumb is adducted) is typical but not pathognomonic (29,30) (Fig. 10.20).

Initial management includes thumb spica splinting, anti-inflammatory medication, and a corticosteroid injection into the first dorsal compartment (Fig. 10.21). If conservative care fails, surgical decompression of the first dor-

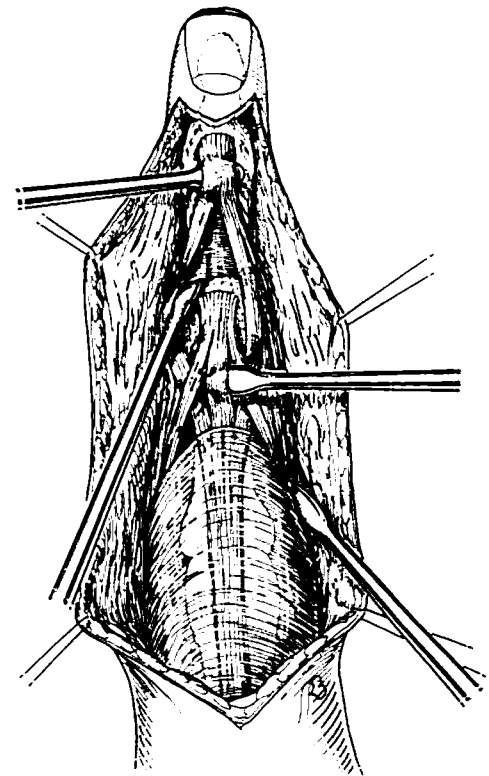


Figure 10.19. Surgical technique for an extensor tenolysis.

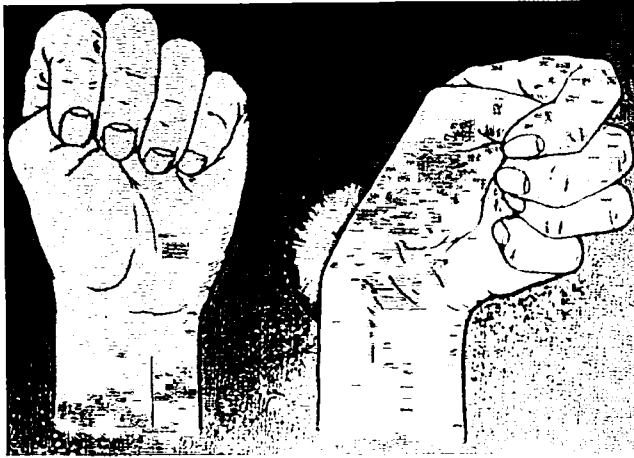


Figure 10.20. The Finkelstein test is used to diagnose de Quervain's tenosynovitis. Courtesy of Gary Schnitz, The Indiana Hand Center.

sal compartment is performed. Care is taken to divide any septa in the compartment, as anatomic studies have shown these are present in 20 to 30% of cases (29) (Fig. 10.22). Recognized complications include tendinous adhesions, volar tendon subluxation, superficial radial nerve injury, and persistence of symptoms.

Intersection Syndrome

Intersection syndrome is a tenosynovitis of the second dorsal compartment (31), commonly seen in athletes. The patient complains of swelling, tenderness, and crepitus 4 to 6 cm proximal to Lister's tubercle (Fig. 10.23). Conservative treatment is often successful (95%) and includes rest, a volar splint, anti-inflammatory medication, and a corticosteroid injection. If necessary, surgery will release the second dorsal compartment, with exploration of the zone of intersection between the radial wrist extensors

and APL and EPB. Any inflammatory or bursal tissue is removed, and the fascial sheaths of APL and EPB are released.

EPL Tendinitis

Originally described as "drummer boy palsy," EPL tendinitis is most commonly seen in patients with rheumatoid arthritis and those with a previous distal radius fracture. Patients complain of swelling, tenderness, and crepitus over the third dorsal compartment. Pain is aggravated by thumb motion. Initial treatment includes a thumb spica splint, rest, and anti-inflammatory medication. Corticosteroid injections are not recommended, because they increase local tissue pressure and may increase the risk of rupture (32). Surgical treatment involves the release of the third dorsal compartment, transposition of the EPL tendon to the radial side of Lister's tubercle, and closure of the retinaculum to prevent relocation (29) (Fig. 10.24).

EIP Syndrome

Irritation of EIP tendon may be precipitated by muscular hypertrophy or synovitis from overuse. The patient describes pain and swelling over the fourth dorsal compartment. Resisted extension of the index finger with the wrist extended is a reliable provocative test. Conservative treatment involves splinting of the wrist and metacarpal phalangeal joints, anti-inflammatory medication, and corticosteroid injections. If conservative therapy fails, the extensor retinaculum over the fourth dorsal compartment may be divided and a synovectomy performed.

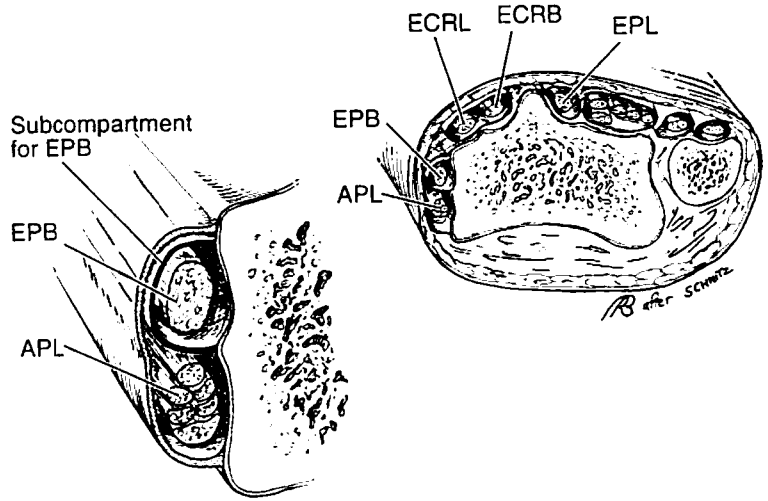
EDM Tendinitis

Tenosynovitis of the fifth dorsal compartment is rare; it is associated with trauma, overuse, and anatomic anomaly.

Figure 10.21. Corticosteroid injection into the first dorsal compartment.



Figure 10.22. Anatomy of the first dorsal compartment and the septa sometimes seen between APL and EPB.



lies (30). Pain and swelling is detected just distal to the ulnar head. Standard conservative therapy includes an ulnar gutter splint. Surgical division of the retinaculum over the fifth dorsal compartment is required in resistant cases.

ECU Tendinitis

ECU tendinitis is the second most common stenosing tenosynovitis of the hand. Seen frequently as a sports injury, ECU tendinitis may be present from posttraumatic ECU subluxation. The ECU is unique because it passes through its own fibro-osseous tunnel, separate from the overlying extensor retinaculum (Fig. 10.2) (20). The fibrous subsheath overlying the ECU may rupture, even in the presence of an intact extensor retinaculum.

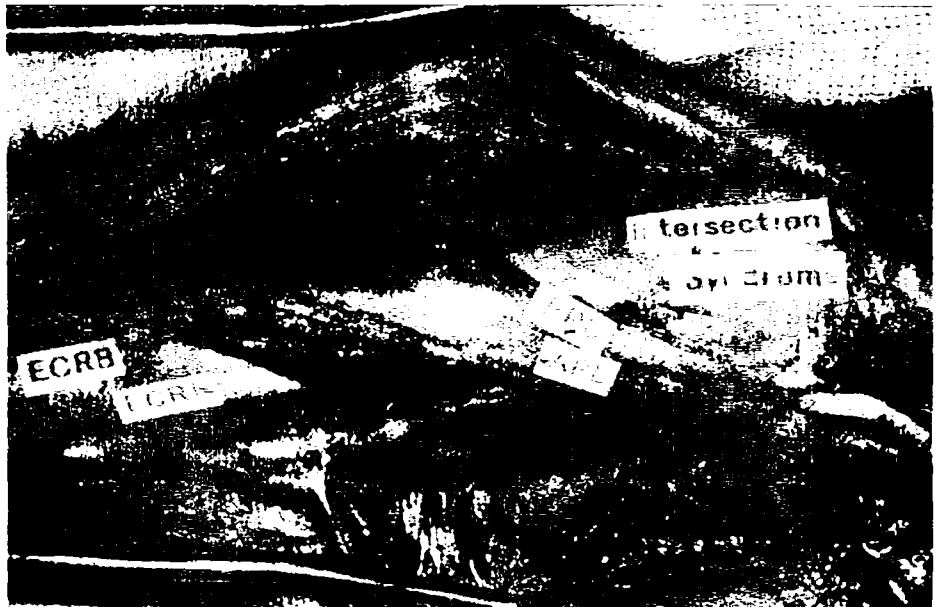
Patients with ECU tendinitis have pain and swelling distal to the ulnar head, exacerbated by resisted wrist exten-

sion and ulnar deviation. ECU subluxation may be elicited with supination and ulnar deviation, resulting in a painful snap of the dorsal wrist. If conservative therapy fails, Hajj and Wood (33) recommend a radial release of the fibro-osseous tunnel followed by repair of the extensor retinaculum. ECU subluxation may require a retinacular graft or sling to stabilize the tendon. After surgery the wrist is immobilized in pronation and slight dorsiflexion in a long arm cast.

SUMMARY

The complexity and interdependence of the various parts of the extensor mechanism make treatment of extensor tendon injuries and tendinopathies challenging. A thorough understanding of anatomy, pathomechanics, surgical technique, and rehabilitation is required to maximize

Figure 10.23. Anatomy of the intersection syndrome.



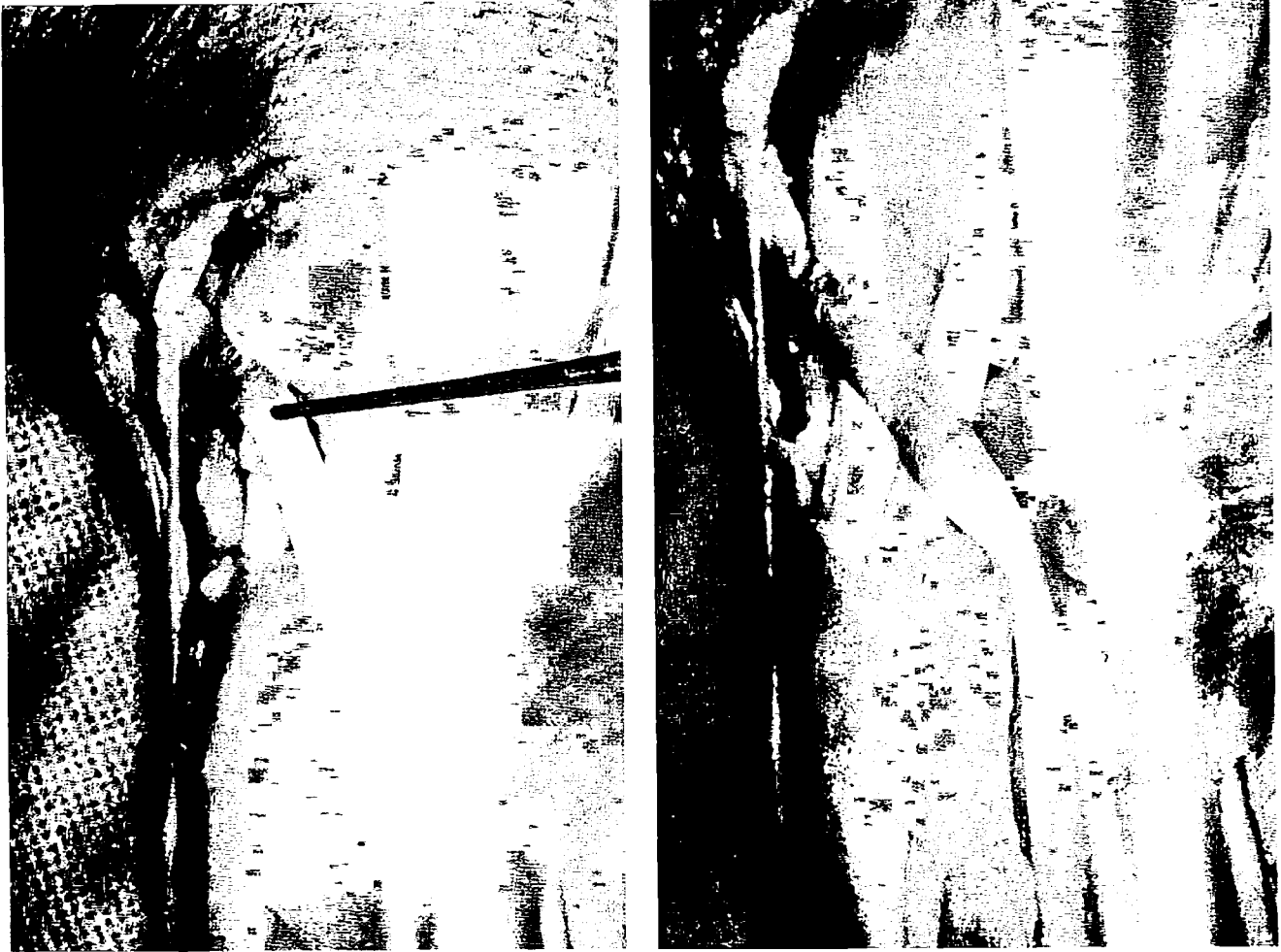


Figure 10.24. Release of the third dorsal compartment.

therapeutic outcome and avoid the many pitfalls inherent in extensor tendon surgery.

REFERENCES

- Newport ML, Blair WF, Steyers CM Jr. Long term results of extensor tendon repair. *J Hand Surg* 1990;15A:961–966.
- Doyle JR. Extensor tendons—acute injuries. In: Green DP, ed. *Operative hand surgery*. New York: Churchill Livingstone, 1992:1925–1954.
- Eyler DL, Markee JE. *J Bone Joint Surg* 1954;36A:1.
- Thompson JS, Littler JW, Upton J. The spiral oblique retinacular ligament (SORL). *J Hand Surg* 1978;3:482–487.
- Kleinert HE, Verdan C. Report of the committee on tendon injuries. *J Hand Surg* 1983;8:794–798.
- Abouna JM, Brown H. The treatment of mallet finger; the results in a series of 148 consecutive cases and a review of the literature. *Br J Surg* 1968;55:653–667.
- Warren RA, Kay NRM, Norris SH. The microvascular anatomy of the distal digital extensor tendon. *J Hand Surg* 1988;13B:161–163.
- Stack HG. A modified splint for mallet finger. *J Hand Surg* 1986;11B:263.
- Kaplan EB. Anatomy, injuries, and treatment of the extensor apparatus of the hand and fingers. *Clin Orthop* 1959;13:24–41.
- Smillie IS. Mallet finger. *Br J Surg* 1937;24:439–445.
- McFarlane RM, Hampole MK. Treatment of extensor tendon injuries of the hand. *Can J Surg* 1973;16:366–375.
- Garberman SF, Diao E, Peimer CA. Mallet finger: results of early versus delayed closed treatment. *J Hand Surg* 1994;19A:850–852.
- Stark HH, Boyes JH, Wilson JN. Mallet finger. *J Bone Joint Surg* 1962;44A:1061–1068.
- Wehbe MA, Schneider LH. Mallet fractures. *J Bone Joint Surg* 1984;66A:658–669.
- Crawford GP. The molded polyethylene splint for mallet finger deformities. *J Hand Surg* 1984;9A:231–237.
- Lange RH, Engber WD. Hyperextension mallet finger. *Orthopedics* 1983;6:1426–1431.
- Clement R, Wray RC Jr. Operative and nonoperative management of mallet finger. *Ann Plast Surg* 1986;16:136–141.
- Coons MS, Green SM. Boutonniere deformity. *Hand Clin* 1995;11:387–402.
- Boyes JH. *Bunnell's surgery of the hand*. Philadelphia: Lippincott, 1970:439–442.
- Spinner M, Choi BY. Anterior dislocation of the proximal interphalangeal joint, a cause of rupture of the central slip of the extensor mechanism. *J Bone Joint Surg* 1970;52A:1329–1336.
- Burton RI. Extensor tendons—late reconstruction. In: Green DP, ed. *Operative hand surgery*. New York: Churchill Livingstone, 1992:1955–1988.
- Iselin F, Levane J, Godoy J. A simplified technique for treating mallet fingers: tenodesis. *J Hand Surg* 1977;5:214.
- Bowers WH, Hurst LC. Chronic mallet finger: the use of Fowler's central slip release. *J Hand Surg* 1978;3:373–376.
- Littler JW. Restoration of the oblique retinacular ligament for cor-

- rection of hyperextension deformity of the proximal interphalangeal joint. In: Fournice A, ed. *La main theumatismale*. Paris: Expansion Scientifique Francoise, 1966.
25. Elliott RA. Boutonniere deformity. Cramer LM, Chase RA, eds. *Symposium on the hand*. Vol. 3. St. Louis: Mosby, 1971:42-56.
 26. Creighton JJ Jr, Steichen JB. Complications in phalangeal and metacarpal fracture, management result of extensor tenolysis. *Hand Clin* 1994;10:1.
 27. Uhl RL. Salvage of extensor tendon function with tenolysis and joint release. *Hand Clin* 1995;3:461-470.
 28. Vaccaro AR, Kupcha P, Schneider LH. The operative repair of chronic nontraumatic tensor tendon subluxations in the hand. *Hand Clin* 1995;11:431-440.
 29. Froimson A. Tenosynovitis and tennis elbow. In: Green DP, ed. *Operative hand surgery*. New York: Churchill Livingstone, 1992:1989-2006.
 30. Plancher KD, Peterson RK, Steichen JD. Compressive neuropathies and tendinopathies in the athletic elbow and wrist. *Clin Sports Med* 1996;15:331-370.
 31. Grundberg AB, Reagan DS. Pathologic anatomy of the forearm intersection syndrome. *J Hand Surg* 1985;10:299-302.
 32. Stern PJ. Tendinitis, overuse syndromes, and tendon injuries. *Hand Clin* 1990;6:467-476.
 33. Hajj AA, Wood MB. Stenosing tenosynovitis of the extensor carpi ulnaris. *J Hand Surg* 1986;11:519-520.