Extensor Tendon: Anatomy, Injury, and Reconstruction

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Learning Objectives: After studying this article, the participant should be able to: 1. Describe the anatomy of the extensor tendons at the wrist, hand, and fingers. 2. Describe acute and chronic pathologic conditions affecting the extensor mechanism. 3. Understand physiology and techniques for repair of traumatic injuries. 4. Understand reconstructive options for chronic disorders.

Although seemingly simple in its anatomy and function, the extensor mechanism of the hand is actually a complex set of interlinked muscles, tendons, and ligaments. A thorough understanding of the extensor anatomy is required to understand the consequences of injury at various levels. Reconstructive options must restore normal function. Whereas primary repair of anatomic structures is frequently possible in acute injury, it is rarely possible in chronic situations. Technically exacting procedures may be necessary to restore function. (Plast. Reconstr. Surg. 106: 1592, 2000.)

The anatomy and function of the extensor mechanism of the hand are more intricate and complex than those of the flexor system. The extensor apparatus is a linkage system created by the radial nerve innervated extrinsic system and the ulnar nerve and median nerve innervated intrinsic system.1 These interconnecting components can compensate for certain deficits in function.

The muscle bellies of the extrinsic extensors arise in the forearm and enter the hand through six compartments formed by the extensor retinaculum, a fibrous band that prevents bowstringing of the tendons (Fig. 1). At the wrist, tendons are covered by a synovial sheath, but not over the dorsal hand or fingers. The extensor pollicis brevis, abductor pollicis longus, extensor pollicis longus, extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi have independent origins and functions.2 The extensor indicis proprius and extensor digiti minimi are usually ulnar and deep to the extensor digitorum communis at the metacarpophalangeal joints. The extensor digitorum communis to the little finger is present less than 50 percent of the time.3 When absent, it is almost always replaced by a junctura tendinum from the ring finger to the extensor apparatus of the little finger.4 The juncturae tendinum join the extensor digitorum communis tendons proximal to the metacarpophalangeal joint. Lacerations proximal to the juncturae may still allow extension of the involved digit by pull from an adjacent finger passing through the juncturae. The four extensor digitorum communis tendons originate from a common muscle belly and have limited independent action. The extensor indicis proprius and extensor digiti minimi have independent muscle bellies and are common donor tendons for transfer.

At the wrist, the extensor tendons are more round and have sufficient bulk to hold a suture. Over the hand, they are thin and flat with longitudinal fibers that do not hold suture well.5 The extrinsic extensor tendons have four insertions—the metacarpophalangeal joint palmar plate through the sagittal bands, a tenacious insertion on the proximal phalanx, and stout insertions on the middle and distal phalanges.6 At the metacarpophalangeal joint level, the extensor tendons are held in place by the intrinsic tendons and the sagittal band that
arises from the palmar plate and the deep intermetacarpal ligament. The extrinsic extensor tendons extend the metacarpophalangeal joint primarily and the interphalangeal joints secondarily.6

The intrinsic tendons are composed of four dorsal interossei (abductors), three palmar interossei (adductors), and four lumbrical muscles. The interossei originate from the lateral sides of the metacarpals and run distally on both sides of the fingers except the ulnar side of the little finger. The interossei tendons enter the finger dorsal to the intermetacarpal ligament. The lumbricals arise from the radial side of the flexor digitorum profundus tendon and pass palmar to the intermetacarpal ligament. The tendons of these intrinsic muscles join to form the lateral bands, all passing palmar to the axis of the metacarpophalangeal joint (Fig. 2). The lateral bands join the extrinsic extensor mechanism proximal to the midportion of the proximal phalanx and continue to the distal finger dorsal to the axis of the proximal interphalangeal and distal interphalangeal joints.7 The intrinsic muscles flex the metacarpophalangeal joint and extend the proximal and distal interphalangeal joints.

At the midportion of the proximal phalanx, the central slip of the extensor mechanism trifurcates.8 Distal to this level, there is an exchange of fibers from the central slip to the lateral bands and from the lateral bands to the central slip. The central slip primarily attaches to the base of the middle phalanx, but both components are capable of proximal and distal interphalangeal extension.

At the proximal interphalangeal joint, the transverse retinacular ligament maintains the position of the extensor mechanism and creates limits on its dorsal-palmar excursion.9 The oblique retinacular ligament arises proximally from the middle third of the proximal phalanx and the A2 pulley and inserts into the lateral portion of the extensor tendon along the middle phalanx. When coursing palmar to the proximal interphalangeal joint, it helps stabilize the lateral bands.8 Its previously described role of mechanically linking simultaneous proximal and distal interphalangeal extension10 has been discounted.8,11 The triangular ligament connects the lateral bands over the dorsum of the middle phalanx, maintaining them in close proximity.

Excursion of the extensor tendons over the finger is less when compared with the flexor tendons. At the proximal interphalangeal joint, excursion may vary from 2.12,13 to 8 mm.14 This small excursion contributes to a system with a delicate balance among its various components. Preservation of relative tendon length between the central slip and lateral bands is important. Once the balance is disturbed, the deformities are progressive, and restoration of normal balance may be very difficult.

Overlapping linkage systems also contribute to this balance.6 The components of the linkage system pass palmar to one joint and dorsal to the next. The intrinsic tendons create the linkage at the metacarpophalangeal and proximal interphalangeal joints, and the oblique retinacular ligaments function at the proximal and distal interphalangeal joints. Because of this interrelationship of joints, a deformity at one joint may cause a reciprocal deformity at an adjacent joint. The boutonnière and swan-neck deformities are examples. When evaluating a deformity at a particular joint, be mindful of the tendon pathways and their relationship to the flexion-extension axis of that joint and adjacent joints.

The extensor mechanism of the thumb is different from that of the fingers in that each joint has an independent tendon for extension. The extensor pollicis longus extends the interphalangeal joint, the extensor pollicis brevis extends the metacarpophalangeal joint, and the abductor pollicis longus extends the carpometacarpal joint. The abductor pollicis longus almost always has multiple tendon slips, whereas the extensor pollicis brevis usually has one.15 The intrinsic muscles of the thumb primarily provide rotational control, but also contribute to metacarpophalangeal flexion and interphalangeal extension. On the radial side, the abductor pollicis brevis tendon continues to insert on the extensor pollicis longus. On the ulnar side, fibers of the adductor pollicis also insert on the extensor pollicis longus. These two muscles can extend the interphalangeal joint to neutral, significantly masking an extensor pollicis longus laceration.

ACUTE INJURY

Extensor tendon injuries are encountered much more frequently than flexor tendon injuries because of their less protected anatomic location. However, because of the misconception that they are comparatively simple to treat, they are often treated in the emergency room by uninitiated physicians who underestimate
The management of extensor injuries demands the same degree of skill and knowledge required for the care of flexor tendon injuries. Recent clinical reports advocate the importance of initial treatment and postoperative rehabilitation of extensor tendon injuries, because good outcomes are not always as easily obtained as once assumed. Because excursion of the extensor tendon over the finger is less than with the flexors, preservation of length is far more critical to restore normal tendon balance.

The relationship between the location of injury and outcome is an important concept. The type of injury, deformity, and surgical outcome are varied because structural and functional systems are different from fingertip to forearm. This led to the categorization of tendon injuries into anatomical zones. Verdan’s zone system is the most widely accepted and allows for a more logical discussion of treatment plans and outcomes associated with each area. Verdan defined eight zones—four odd-numbered zones overlying each of the joints and four

![Fig. 1. The extensor tendons enter the hand through six fibrous tunnels that are made by the extensor retinaculum at the wrist. A small sheath is present around each individual tendon at the wrist, but not in the hand or fingers. The extensor indicis proprius (EIP) and the extensor digiti minimi (EDM) are typically ulnar and deep to the communis tendons at the metacarpophalangeal joint. Juncturae tendinum provide interconnections between the extensor digitorum communis (EDC) tendons in the distal portion of the dorsal palm. ECU, extensor carpi ulnaris; EPL, extensor pollicis longus; EPB, extensor pollicis brevis; APL, abductor pollicis longus; ECRL, extensor carpi radialis longus; ECRB, extensor carpi radialis brevis.](image1)

![Fig. 2. The extrinsic extensor tendons contribute the central slip to the extensor mechanism in the finger. The intrinsic system contributes the lateral bands that pass palmar to the axis of the metacarpophalangeal (MP) joint and dorsal to the axis at the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints. The sagittal band centralizes the extensor tendon over the metacarpal head at the metacarpophalangeal joint. The intermetacarpal ligament separates the lumbrical tendon that is palmar from the interossei tendons that are dorsal. The transverse retinacular ligament stabilizes the extensor tendon at the proximal interphalangeal joint. The oblique retinacular ligament is a component of the linkage system, passing palmar to the rotational axis of the proximal interphalangeal joint and dorsal to the joint axis at the distal interphalangeal joint. The triangular ligament helps maintain close proximity of the lateral bands over the middle phalanx. The extensor tendon has four insertions, with one being on the metacarpophalangeal palmar plate through the sagittal band. The tendon then inserts on the base of the proximal, middle, and distal phalanges. Over the distal portion of the proximal phalanx, the central slip trifurcates as the central slip and lateral bands share fibers. The central slip inserts on the base of the middle phalanx, whereas the lateral band component continues to insert on the base of the distal phalanx.](image2)
even-numbered zones overlying the intervening tendon segments and increasing in number from distal to proximal.

Zone 1 Injury (Mallet Finger)

Disruption of continuity of the extensor tendon over the distal interphalangeal joint produces the characteristic flexion deformity of the distal interphalangeal joint. A mallet finger may be open, but is more often closed. The mechanism for the closed injury is most commonly a sudden, forceful flexion of the distal interphalangeal joint in an extended digit. This results in rupture of the extensor tendon or avulsion of the tendon often, with a bony fragment from its insertion in the distal phalanx. When left untreated for a prolonged time, hyperextension of the proximal interphalangeal joint (swan-neck deformity) may develop because of proximal retraction of the central band.

Mallet finger injuries are classified into four types:

- Type I: Closed, with or without avulsion fracture.
- Type II: Laceration at or proximal to the distal interphalangeal joint with loss of tendon continuity.
- Type III: Deep abrasion with loss of skin, subcutaneous cover, and tendon substance.
- Type IV: (A) Transepiphyseal plate fracture in children; (B) hyperflexion injury with fracture of the articular surface of 20 to 50 percent; and (C) hyperextension injury with fracture of the articular surface usually greater than 50 percent and with early or late palmar subluxation of the distal phalanx.

The management of mallet finger is still a topic for debate. In the vast majority of cases, splinting alone is sufficient. For type I injuries, the recommended treatment is continuous splinting of the distal interphalangeal in extension for 6 weeks, followed by 2 weeks of night splinting. An Alumafom splint (Millpledge Healthcare, Nottinghamshire, U.K.) is used to keep the distal interphalangeal joint at 0 degrees. The patient must understand that splinting must be continuous (e.g., using the thumb to apply extension force to the distal phalanx of the injured finger when showering). The physician should be aware that there are complications with splinting, including dorsal skin necrosis, especially with acute swelling. Alternatively, and only in rare circumstances, Kirschner wire fixation of the distal interphalangeal joint in extension, with the wire cut off beneath the skin to allow the patient to continue working, will achieve the same results. Kirschner wire should be left in place for 6 weeks, followed by 2 weeks of night splinting. Type II injuries may be repaired with a simple figure-of-eight suture through the tendon alone or a roll-type suture (dermatotenodesis) incorporating the tendon and skin in the same suture. The distal interphalangeal joint is splinted in extension for 6 weeks, followed by 2 weeks of night splinting. Type III injuries with loss of tendon substance require immediate soft-tissue coverage and primary grafting or late reconstruction using a free tendon graft. Type IV-A is best managed by closed reduction, followed by splinting for 3 to 4 weeks. Mallet finger deformity in a child is usually a transepiphyseal fracture of the phalanx. The extensor mechanism is attached to the basal epiphysis, so closed reduction results in correction of the deformity. In type IV-B, where there is no palmar subluxation, splinting for 6 weeks with 2 weeks of night splinting yields good results. Excellent remodeling of the articular surface occurs in most mallet fractures. Type IV-C with palmar subluxation of the distal phalanx is usually best managed operatively with open reduction and internal fixation using a Kirschner wire and possibly a pull-out wire or suture (Fig. 3). This should also be protected with a splint for 6 weeks, after which the Kirschner wire is removed and motion started. The fracture fragment size is a less important consideration than the fragment’s location. A proximally displaced fragment not in continuity with the distal phalanx may also require open reduction and internal fixation.

Zone 2 Injury (Middle Phalanx)

In contrast to zone 1 injuries, zone 2 injuries are usually secondary to a laceration or crush injury rather than an avulsion. If less than 50 percent of the tendon width is cut, the treatment involves routine wound care and splinting for 7 to 10 days, followed by active motion. Injuries involving more than 50 percent of the tendon should be repaired primarily, followed by 6 weeks of splinting.
Zone 3 Injury (Boutonnière Deformity)

The boutonnière deformity is caused by disruption of the central slip at the proximal interphalangeal joint. This results in the classic deformity with loss of extension at the proximal interphalangeal joint and hyperextension at the distal interphalangeal joint. The injury can be closed or open, and the central slip may avulse with or without a bony fragment.

The early injury may be associated with localized swelling, but no deformity. The boutonnière deformity then develops gradually, especially in closed trauma. It usually appears 10 to 14 days after the initial injury.16 Diagnosis is best made after splinting the finger straight for a few days and reexamining it after swelling subsides. Absent or weak active extension of the proximal interphalangeal joint is a positive finding.22 Initial treatment for closed injury should be splinting of the proximal interphalangeal joint in extension. The distal interphalangeal, metacarpophalangeal, and wrist joints are left free. Length of time for splinting ranges from 4 to 6 weeks, with reapplication of the splint if the deformity recurs.

Surgical indications for closed boutonnière deformity are: (1) displaced avulsion fracture at the base of the middle phalanx13; (2) axial and lateral instability of the proximal interphalangeal joint associated with loss of active or passive extension of the joint25; and (3) failed nonoperative treatment. The usual method of repair is to pass a suture through the central tendon and secure it to the middle phalanx with or without the bony fragment. Kirschner wire fixation of the proximal interphalangeal joint is maintained for 10 to 14 days, followed by an extension splint until there is radiographic evidence of bony union. If primary repair of the central slip is not possible, portions of the lateral bands can be sutured together in the dorsal midline of the finger to reconstruct the central slip (Fig. 4). A flap may be raised from the proximal portion of the central slip to restore active extension (Fig. 5).

For open injuries, surgical repair might be avoided by splinting, because the tendon ends do not retract in this area. However, in a true boutonnière deformity, both central slip and lateral band injuries should be expected. In the elderly, the period of absolute immobilization can be reduced to 2 weeks to help them regain full flexion. Results are still fairly good.24

Fig. 3. A closed mallet injury without a fracture or with a nondisplaced fracture can be treated with closed splintage. A mallet fracture with joint subluxation requires reduction and internal fixation. Through a dorsal approach over the distal interphalangeal joint, the fragments are isolated. A Kirschner wire is passed antegrade through the bulk of the distal phalanx. Reduction is then completed and the Kirschner wire is passed retrograde through the fragment (if possible) and into the middle phalanx. Intraoperative radiographs confirm appropriate reduction. The Kirschner wire is removed in 6 weeks.

Fig. 4. Acute disruption of the extensor tendon over the proximal interphalangeal joint may not be amenable to primary repair of the central slip. The lateral bands can be divided longitudinally over a distance of 2 cm, centered over the proximal interphalangeal joint. The middle segments are sutured in the midline to reconstruct the function of the central slip. This technique will help prevent formation of a boutonnière deformity.
Zone 4 Injury (Proximal Phalanx)

Zone 4 injuries usually involve the broad extensor mechanism, are usually partial, and usually spare the lateral bands. Diagnosis can only be made by direct inspection. However, when there is no loss of extension in the interphalangeal joints, repair is rarely required. Splinting the proximal interphalangeal joint in extension for 3 to 4 weeks without repair is equivalent to repair of the tendon with 5-0 nonabsorbable suture. For partial injuries, early motion should be considered. For complete lacerations, primary repair should be performed, followed by 6 weeks of splinting in extension.

Zone 5 Injury (Metacarpophalangeal Joint)

Injuries over the metacarpophalangeal joint are almost always open and should be treated as a human bite until proven otherwise. The injury more often occurs with the joint in flexion, so the tendon injury will actually be proximal to the dermal injury. Primary tendon repair is indicated after thorough irrigation. All involved structures should be repaired separately, including partial injuries. The sagittal bands should be repaired to prevent lateral migration of the extensor digitorum communis tendon and subsequent metacarpophalangeal extension loss. Immobilization of the wrist in 30 to 45 degrees of extension and the metacarpophalangeal joint in 20 to 30 degrees of flexion is performed with the proximal interphalangeal joint free. Early dynamic splinting has also improved outcome.

When associated with a human bite, the incidence of complications is directly related to the time from injury to treatment. The wound is extended for thorough inspection and debrided, irrigated, and left open. Arthrotomy of the metacarpophalangeal joint should be considered in cases of bite injury or suspected pyarthrosis. Wound cultures should be taken before irrigation and the patient started on broad-spectrum antibiotics. The hand is splinted with the wrist in 45 degrees of extension and the metacarpophalangeal joint in 15 to 20 degrees of flexion. The wound usually heals within 5 to 10 days, and secondary repair is rarely needed.

Zone 6 Injuries (Dorsal Hand)

Single or partial tendon lacerations in zone 6 may not result in loss of extension at the metacarpophalangeal joint because extensor forces are still transmitted from adjacent extensor tendons through the juncturae tendinum. Diagnosis, therefore, is best made by direct inspection. Because the tendons are thicker and more oval, repair should be performed with stronger, core-type sutures. Conventional splinting places the wrist and fingers in extension for 4 to 6 weeks. If the extensor digitorum communis is involved, all fingers should be splinted. If just a proprius tendon is involved, only the affected finger need be splinted with the wrist. Again, use of early dynamic splinting has been shown to improve results.

Zone 7 Injuries (Wrist)

Controversy exists whether excision of part of the retinaculum over the injury site is necessary to prevent postoperative adhesions. If early dynamic splinting is used, adhesions are less likely. In any case, partial release of the retinaculum is required in most cases to gain exposure to the lacerated tendons, which retract significantly in this area. However, at least some portion of the retinaculum should be preserved to prevent extensor bowstringing.
A four-strand nonabsorbable core suture is used.

**Zone 8 Injuries (Dorsal Forearm)**

Multiple tendons may be injured in this area, making it difficult to identify individual tendons. In this situation, restoration of independent wrist and thumb extension should be given priority. Difficulty also may be encountered with injuries at the musculotendinous junction because the fibrous septa retract into the substance of the muscles. When repairing the muscle bellies, multiple figure-eight sutures are used with a slowly absorbing material. Static immobilization of the wrist in 45 degrees of extension and metacarpophalangeal joints in 15 to 20 degrees of flexion is maintained for 4 to 5 weeks. Also, because the extensors originate from the lateral epicondyle, splinting the elbow in flexion may also be beneficial. Dynamic motion of the metacarpophalangeal joints may be started at 2 weeks and has improved results.

**Thumb Injuries**

Although anatomically the thumb interphalangeal and finger distal interphalangeal joints are similar, the terminal extensor tendon is much thicker on the thumb. Therefore, mallet thumbs are rare, especially as closed injuries. There is little data comparing operative and nonoperative treatments. For open injuries, most would recommend primary repair, followed by splinting for 6 weeks. For closed injuries, splinting for 6 weeks without surgical repair is appropriate, although some would still opt for surgical repair.

Because of the broad extensor expansion at the metacarpophalangeal joint of the thumb, traumatic division of all components of thumb extension is rare. Isolated laceration of the extensor pollicis brevis at this level is also rare and its repair is optional but recommended, because extension of the metacarpophalangeal joint is possible with an intact extensor pollicis longus. There is an extension lag in both metacarpophalangeal and interphalangeal joints with extensor pollicis longus injury, and it should be repaired. All tendons and the capsule should be repaired separately using core-type sutures. Splinting should be for 3 to 4 weeks, with the wrist in 40 degrees of extension and slight radial deviation and the thumb metacarpophalangeal joint in full extension.

In zones 6 and 7, the abductor pollicis longus retracts significantly when divided and usually requires that the first compartment be released for successful repair. Splinting is for 4 to 5 weeks, with the wrist in radial deviation and the thumb in maximal abduction. Treatment of the thumb extensors at the forearm level is identical to that of the fingers.

**Dynamic Splinting for Extensor Injuries**

Since its introduction by Kleinert et al., early controlled motion has been used after flexor repair to overcome the adhesions associated with repair in “no man’s land.” Early controlled motion with a dynamic extensor splint has been found to decrease adhesions and subsequent contractures. It has been most effective when used for injuries in zones 5 to 8. Its usefulness in the more distal zones is less apparent, although several studies are promising. The technique should only be used in the cooperative patient, and is most advantageous when a skilled hand therapist is available to fabricate the splint and monitor the patient. The splint allows incomplete active flexion and passive extension. After 4 weeks, full active extension is initiated.

**Chronic Injury**

To understand better extensor tendon reconstruction, several key points should be reemphasized. The extensor mechanism has much less tolerance for changes in tendon length than the flexor mechanism. Finger extension is synergistic with wrist flexion, and controlled wrist function is essential to normal finger extensor mechanics. Therefore, wrist flexors are excellent tendon transfer donors for finger extension. The delicate balance of the extensor complex is partially based on a series of overlapping linkage systems, lying palmar to the axis of rotation at a proximal joint and dorsal at the next distal joint. The precise tension in this system changes with joint flexion and contraction. Thus, late extensor disorders are reciprocal at adjacent joints (e.g., proximal interphalangeal flexion is associated with distal interphalangeal extension in the boutonnière deformity). Evaluation of established extensor disorders must include all three joints as they are interrelated, and treatment is usually directed at the most proximal involved joint.

Preoperatively, ensuring patient education is paramount, because bad outcomes are likely for patients who cannot understand or partic-
ipate in postoperative therapy and those who have unrealistic goals. Joint contractures are contraindications to reconstruction of extensors. The hand surgeon must remember that many established imbalances will respond to nonoperative treatment.

Inability to extend the finger metacarpophalangeal joint or radially abduct the thumb despite intact radial nerve function indicates a problem proximal to the metacarpophalangeal joint.33 Unless held to length by the juncturae, the motor unit undergoes myostatic contraction, precluding secondary tendon repair.

The extensor pollicis longus tendon is a commonly ruptured tendon, usually associated with distal radius fractures and rheumatoid arthritis, but the other finger extensors also have a significant propensity to rupture.34 Tendon transfers are the most reliable technique for reconstruction of chronic extensor tendon deficits at this location. The extensor indicis proprius or palmaris longus is frequently used for extensor pollicis longus functional transfer, whereas the extensor indicis proprius, flexor carpi ulnaris, or side-to-side extensor digitorum communis transfers are used for extensor digitorum communis ruptures. For wrist extensors, the pronator teres is the common choice.

Technical points in this procedure include passing the transfer subcutaneously rather than beneath the retinaculum because wrist flexion is synergistic with finger extension,36 and around the wrist rather than through the interosseous membrane to lessen scarring. Setting tension properly with the wrist maximally extended and the fingers flexed is essential. The procedure concludes with the wrist splinted in moderate flexion and the metacarpophalangeal joints splinted at 70 degrees flexion. This should be continued for 3 to 4 weeks, followed by active range of motion for 1 to 2 weeks and later passive ranging, although dynamic splinting may be used in motivated, reliable patients. Complications after repair of lesions proximal to the metacarpophalangeal joint include adhesions of the tendons (most common), rupture or attenuation, donor deficits, and joint stiffness.

The sagittal bands wrap around the metacarpophalangeal joint, stabilizing the extrinsic extensors centrally with attachment to the palmar plate.1,6 With rupture or attenuation of these bands, usually on the radial side, the extensor slips off the metacarpal head fulcrum and loses its mechanical advantage for extension. This situation occurs most commonly with not only rheumatoid arthritis, but also with traumatic, congenital, epileptic, and degenerative states.37 This can be an acute or chronic finding. Physical findings include “catching,” extensor lag, ulnar deviation of the digit, and pain and swelling. The patient cannot extend from flexion; but, if passively extended, the tendon reduces, and the patient can hold this position.

When diagnosed during the first 2 weeks, this subluxation can be effectively treated with metacarpophalangeal extension splinting and encouraged proximal interphalangeal motion.38 After 2 weeks, this condition generally should be surgically corrected by delayed primary repair that is technically difficult, dorsal tenodesis, or a sling procedure.6,35,37,39 The latter two procedures take a sling of extensor digitorum communis to recentralize the tendon. The metacarpophalangeal joints must remain in extension for several weeks while the other joints are ranged.

If the extrinsic extensor becomes adherent or foreshortened, flexion at the metacarpophalangeal causes an extension force at the proximal interphalangeal by dorsal tenodesis restraint. These patients with extrinsic extensor tendon tightness are not able to fully flex the involved fingers.1,6 Clinically, the patient can flex the proximal interphalangeal only with the metacarpophalangeal extended. Most patients respond well to exercises that emphasize extensor excursion and splinting, especially if this is begun early.

Operative treatment of this condition includes tenolysis or extrinsic extensor release. However, these procedures should not be considered until a 6-month trial of hand therapy has been completed. Tenolysis is best for fingers with scarring but adequate tendon length. Although the sagittal bands must be preserved, the extensor retinaculum is expendable in this procedure. Littler’s technique of extrinsic extensor tendon release is indicated for short tendons or long lengths of scarring.1,6 This procedure separates the extrinsic and intrinsic extensors. The central portion of the extensor assembly is excised over the proximal phalanx, preserving the sagittal bands and the central slip distally at its insertion on the base of the middle phalanx. It is recommended that both of these procedures be performed while allowing the patient to move the fingers actively on
the table. Range of motion is begun almost immediately.

In contrast, intrinsic tendon tightness (Fig. 6) is diagnosed by the inability to flex the proximal interphalangeal joint when the metacarpophalangeal joint is brought into full extension, putting maximum tension on the intrinsic. This condition usually responds well to exercise and splinting. For patients in whom nonoperative treatment fails, surgical treatment includes eliminating the dual control of proximal interphalangeal joint extension by excision of the oblique fibers and lateral band of the extensor hood over the middle of the proximal phalanx. Be aware that this does not address the palmar plate laxity in the swan-neck deformity.

Swan-neck deformity describes a finger with proximal interphalangeal joint hyperextension and distal interphalangeal flexion. Although it is a dynamic imbalance initially, it may progress to a fixed deformity. This posture can result from various causes, including palmar plate laxity at the proximal interphalangeal joint, spastic conditions such as cerebral palsy and stroke, rheumatoid arthritis, malunion of middle phalanx fractures, and mallet deformity with coexistent palmar plate laxity. Pathomechanically, the incompetent palmar plate allows hyperextension at the proximal interphalangeal joint, causing the lateral bands to migrate dorsally. This creates laxity in the distal tendon because of the fixed attachment of the central tendon at the proximal interphalangeal joint. The unopposed flexor digitorum profundus deforms the distal phalanx into flexion.

Conservative splinting and exercises are not helpful in the treatment of swan-neck deformity, fixed or dynamic. Surgical correction must recognize the underlying cause: only the mallet deformity need be corrected if this is the cause. Correction of the malunited middle phalanx is the appropriate treatment of this cause. When compliance with postoperative splinting and exercises is difficult, as with spastic patients, arthrodesis of the proximal interphalangeal joint is appropriate. In rheumatoid patients, one must correct the metacarpophalangeal joint deformity first. When intrinsic tightness is present, intrinsic release must also be performed.

For those cases where rebalancing of the extensor mechanism with correction of the proximal interphalangeal palmar plate incompetence is necessary, two general techniques are commonly used: oblique retinacular ligament reconstruction or superficialis tenodesis. Oblique retinacular ligament reconstruction involves creating a tenodesis that passively tightens and extends the distal interphalangeal joint as the proximal interphalangeal joint actively extends, while remaining palmar to the proximal interphalangeal joint axis and dorsal to the distal interphalangeal joint axis. The original method described by Littler uses the lateral band either ipsilaterally or spiraling palmarly attached at the base of the proximal phalanx for oblique retinacular ligament reconstruction. A modification of this, spiral oblique retinacular ligament technique, uses a free tendon graft to spiral palmarly to reconstruct the oblique retinacular ligament (Fig. 7) with Kirschner wire fixation of the proximal interphalangeal joint in 20 degrees of flexion. The superficialis tenodesis technique uses one slip of the flexor digitorum superficialis attached distally and affixed proximally to the proximal phalanx. This method does not rebalance the extensor mechanism at the distal joint.

Postoperatively, the Kirschner wire is removed at 4 weeks and replaced by a dorsal blocking splint. A realistic goal is 5 to 10 degrees flexion at the proximal interphalangeal joint rather than complete extension. Complications include recurrence of the deformity caused by attenuation, excessive proximal interphalangeal joint flexion deformity from too
tight of a tenodesis, and flexor adhesions, especially after superficialis tenodesis.

Boutonnière deformity refers to a finger posture with the proximal interphalangeal joint flexed and the distal interphalangeal joint hyperextended (Fig. 8). Like swan-neck deformity, it begins as a dynamic process that can lead to a fixed deformity. It is primarily caused by disruption of the central slip and transverse retinacular fibers at the proximal interphalangeal joint, allowing the lateral bands to migrate palmar to the axis of the proximal interphalangeal joints, and become flexors of this joint. The oblique retinacular ligament shortens along with the lateral bands. If the condition is left untreated, the distal interphalangeal hyperextends. Three stages have been described, including dynamic imbalance, established tendon contracture with supple joints, and fixed-joint changes.

Similar to many other extensor imbalances, boutonnière deformity is best treated with splinting and exercises, especially before the deformity occurs. This cannot be stressed enough because surgical repair is fraught with complications, and results with conservative therapy may be as good or better than with surgery. The exercises include active-assisted proximal interphalangeal joint extension that will cause worsening of the distal interphalangeal extension. Secondly, active-forced flexion of the distal interphalangeal joint with the proximal interphalangeal in extension will cause stretching of the oblique retinacular ligament. Splinting supports the proximal interphalangeal joint and the two proximal phalanges only.

Perhaps a systematic approach to the management of chronic boutonnière deformity is best. Curtis et al. described a four-stage approach progressing from tenolysis to transverse retinacular ligament sectioning to shortening of the lateral bands over the middle phalanx to repair of the central slip. One technique to repair late boutonnière deformity uses the central slip primary repair with reattachment of the lateral bands dorsal to the proximal interphalangeal joint axis, with or without division of the terminal extensor insertion on the distal phalanx to restore distal interphalangeal function. However, probably the most reliable procedure is that advocated by Littler and Eaton. This involves shortening of the lateral band by incomplete transection of the extensor mechanism over the middle of the middle phalanx, but leaving the oblique retinacular ligament intact. Littler originally described this technique with suturing of the lateral bands dorsally. For any reconstruction, a supple proximal interphalangeal joint with satisfactory passive motion is a prerequisite.

Occasionally, the central tendon deficit is so large that it requires a tendon graft passed through a bone tunnel at the dorsal base of the middle phalanx and criss-crossed to be sutured.
to the lateral bands.\textsuperscript{51} Lateral band tendon transfers have also been described by Littler\textsuperscript{52} and Matev.\textsuperscript{53} The hyperextension deformity at the distal interphalangeal joint may cause greater difficulty for the patient than the lack of proximal interphalangeal joint extension. A tenotomy of the terminal extensor mechanism alone may be sufficient.\textsuperscript{54}

The complexity of the extensor mechanism is created by a delicately balanced musculotendinous system controlled by two linkage systems. Anatomical and functional knowledge is a prerequisite to provide care for extensor deficits, both acute and chronic.

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by W. Bradford Rockwell, M.D., Peter N. Butler, M.D., and Bruce A. Byrne, M.D.

1. WHAT PERCENTAGE OF PEOPLE HAVE AN EXTENSOR DIGITORUM COMMUNIS TENDON TO THE LITTLE FINGER?
   A) 100 percent
   B) 98 percent
   C) 90 percent
   D) 80 percent
   E) Less than 50 percent

2. INDICATIONS FOR OPERATIVE MANAGEMENT FOR CLOSED BOUTONNIÈRE DEFORMITY INCLUDE ALL OF THE FOLLOWING EXCEPT:
   A.) Failure of conservative, nonoperative management.
   B) Palmar migration of the lateral bands 7 to 10 days after injury.
   C) Displaced avulsion fracture of the base of the middle phalanx.
   D) Axial and lateral instability of the proximal interphalangeal joint associated with loss of active or passive proximal interphalangeal joint extension.

3. ZONE 6 (DORSAL HAND) INJURIES ARE BEST DIAGNOSED BY:
   A) Loss of active extension of distal interphalangeal joint.
   B) Loss of active extension of proximal interphalangeal joint.
   C) Loss of active extension of metacarpophalangeal joint.
   D) Direct inspection.
   E) Intrinsic tightness plus posture of finger.

4. THE RECOMMENDED TREATMENT FOR CLOSED MALLET FINGER IS:
   A) Surgical repair of tendon.
   B) Percutaneous Kirschner wire fixation of distal interphalangeal joint in extension.
   C) Splinting of only distal interphalangeal joint in extension.
   D) Splinting of proximal interphalangeal and distal interphalangeal joint in extension.
   E) Controlled early motion of distal interphalangeal joint.

5. WHICH OF THE FOLLOWING STATEMENTS IS TRUE?
   A) Intrinsic tightness refers to resistance of proximal interphalangeal joint flexion while the metacarpophalangeal joint is flexed.
   B) Joint contractures are contraindications to extensor reconstruction.
   C) Swan-neck deformity refers to a finger posture of proximal interphalangeal joint flexion and distal interphalangeal joint hyperextension.
   D) When the extrinsic extensor subluxates off the metacarpal head, it usually falls to the radial side.
   E) Extrinsic tightness causes a posture of metacarpophalangeal flexion, proximal interphalangeal extension and distal interphalangeal extension.

6. ALL OF THE FOLLOWING CONDITIONS USUALLY RESPOND WELL TO SPLINTING AND EXERCISE PROGRAMS EXCEPT:
   A) Extrinsic extensor tightness.
   B) Intrinsic extensor tightness.
   C) Swan-neck deformity.
   D) Boutonnière deformity.
   E) Mallet finger.

To complete the examination for CME credit, turn to page 1673 for instructions and the response form.