

Closed Soft Tissue Extensor Mechanism Injuries (Mallet, Boutonniere, and Sagittal Band)

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Learning Objectives

- Clarify the anatomy of the extensor mechanism.
- Describe the function of various components of the extensor tendon.
- Appraise the clinical presentations of the terminal tendon, central tendon, and sagittal band injuries.
- Offer treatment strategies for extensor tendon injuries in zones I, III, and V.
- Discuss the surgical techniques for injuries to various components of the extensor mechanism.

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The extensor mechanism of the hand is complex and can be disrupted at several different points, from hand trauma. This article summarizes current concepts relating to the presentation and treatment of closed soft tissue injuries of the extensor mechanism in zones I, III, and V, commonly known as mallet finger and boutonniere deformities and sagittal band injuries. (*J Hand Surg Am.* 2014;39(5):1005–1011. Copyright © 2014 by the American Society for Surgery of the Hand. All rights reserved.)

Key words Extensor tendon injury, central slip, sagittal band, terminal tendon, mallet finger.

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CLOSED INJURIES OF THE EXTENSOR mechanism in zones I, III, and V are known as mallet finger (terminal tendon disruption), boutonniere injury (central slip disruption), and sagittal band injury (usually radial sagittal band of the middle finger), respectively. Of these, the mallet finger is the most common, with an estimated incidence of 9.9 of 100,000 per year, which makes it one of the most common musculoskeletal soft tissue injuries.¹ This article focused on traumatic closed soft tissue injuries; the pathophysiology and management of extensor injuries resulting from rheumatoid, other arthritic etiologies, or fracture will not be discussed.

ANATOMY

The anatomy of the extensor mechanism of the hand is composed of muscles originating proximal to the wrist and terminating in extensor tendons inserting distally in the phalanges. Extensor tendon anatomy of the hand and finger is complex, with multiple interconnections, including the juncturae tendineae at the level of the metacarpals, the sagittal bands at the metacarpophalangeal (MCP) joint, the central slip insertion at the middle phalanx, and the terminal tendon insertion at the distal phalanx (Fig. 1).

At the level of the MCP joint, the sagittal bands connect the extensor mechanism to the volar plate and surrounding soft tissues to maintain the central position of the extensor tendon. Because the extensor tendons do not insert directly on the MCP joint or proximal phalanx,² they extend the MCP joint through a lasso effect through the sagittal bands. Injury to the sagittal bands at this level can cause instability of the extensor tendon over the MCP joint with flexion and extension.

Over the dorsum of the proximal phalanx, the extensor digitorum communis (EDC) trifurcates into the central slip and 2 lateral components that join the terminal tendons of the lumbricales and interossei to form the lateral bands. There are no extensor tendon insertions over the proximal phalanx.²

Over the dorsum of the middle phalanx, the central slip inserts at the phalangeal base, and the lateral bands converge dorsally, stabilized centrally by the triangular ligament. The triangular ligament is essential to maintain the lateral bands in correct dorsal alignment and prevents excessive volar subluxation of the lateral bands. Injury to both the central slip and triangular ligament is required to produce the boutonniere deformity. The transverse retinacular ligament arises from the volar sheath, attaches to the extensor mechanism at the level of the proximal

interphalangeal (PIP), joint and prevents excessive dorsal subluxation of the lateral bands.

The lateral bands conjoin proximal to the distal interphalangeal (DIP) joint to insert on the distal phalanx base as the terminal tendon. Elongation of the terminal tendon by 1 mm can result in an extensor lag at the DIP joint by as much as 25°, producing the mallet finger deformity.³

Another important but visually elusive anatomic structure is the oblique retinacular ligament (ORL). The ORL arises from the flexor sheath at the proximal phalangeal level and courses obliquely and distally to insert on the terminal tendon. Traditionally, the ORL was presumed to cause concomitant DIP extension with active PIP extension. Although the existence and function of the ORL have been controversial, the surgical creation of a spiral oblique retinacular ligament has been employed in the treatment of mallet finger.⁴ A recent anatomic and biomechanical study found that the ORL was present bilaterally in 38 of 40 fingers and contributed up to a maximum of 31% of passive resistance to DIP flexion at 30° PIP flexion.⁵

CLINICAL PRESENTATION

Mallet finger (zone I injury)

Closed mallet finger presents with an acute extensor lag at the DIP joint, usually painless, and is commonly noted after trivial trauma, such as catching the fingertip on clothing or through ball impact injuries. The injury is more common on the ulnar digits, with the little, middle, and ring finger most often involved.^{6,7} The mechanism of injury is sudden flexion of an actively extended or extending DIP joint. On examination, the patient presents with an extensor lag at the DIP joint (Fig. 2), sometimes associated with hyperextension of the PIP joint, the swan neck deformity. The presence of a painful dorsal bump at the level of the DIP joint typically signifies a concomitant fracture at the insertion of the terminal tendon, known as a mallet fracture. Radiographs should be obtained to assess for fractures (Fig. 3).

Boutonniere injury (zone III injury)

Closed central slip injuries usually present with dorsal PIP swelling and pain. The patient may have full motion immediately if the triangular ligament remains intact and the lateral bands maintain their normal dorsal position. Alternatively, there may be a full-blown boutonniere deformity acutely if the central slip and triangular ligament both tear and the lateral bands subluxate volar to the PIP joint. The typical mechanism of injury is forced PIP flexion, a

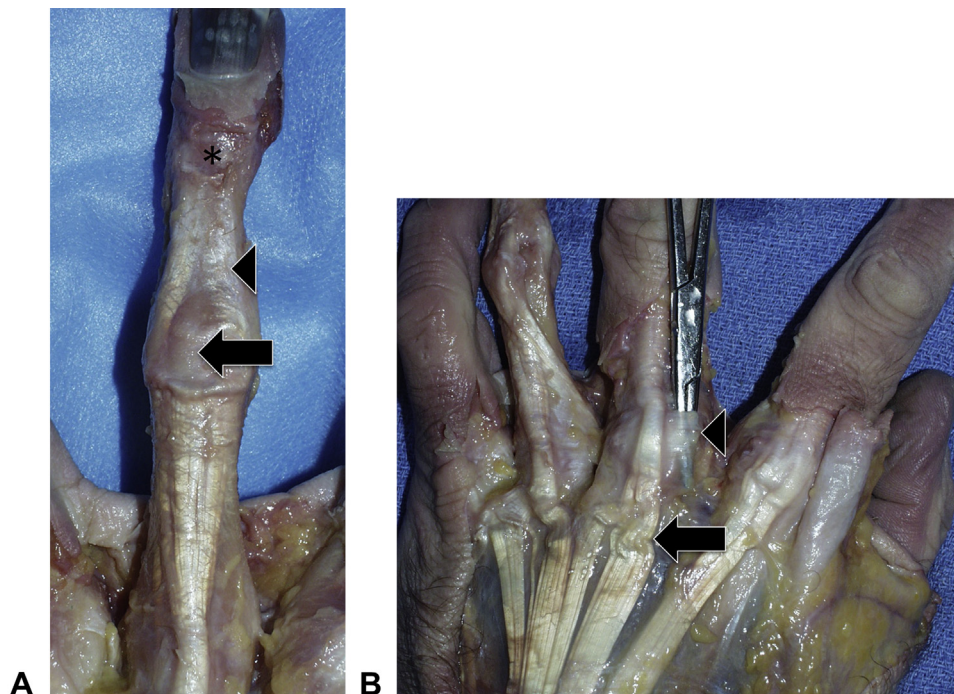


FIGURE 1: **A** Anatomic specimen of the extensor mechanism. Arrow, central slip; arrowhead, lateral band; asterisk, terminal tendon. **B** Extensor mechanism anatomy. Arrowhead, radial sagittal band of the middle finger; arrow, extensor digitorum communis.

direct blow to the dorsum of the PIP joint, or volar dislocation of the PIP joint. Because this deformity may not be obvious initially, a high index of suspicion is necessary; dorsal PIP joint pain and swelling in the emergency department should prompt the Elson test (Fig. 4). Elson⁸ originally described flexing the PIP over the edge of a table to assess for DIP extension. With an intact central slip, flexion of the PIP advances the extensor mechanism, resulting in distal slack in the lateral bands and an inability to extend the DIP. In the presence of complete central slip disruption, there will be increased extensor tone present in the DIP joint (compared with the contralateral digit in a similar position), usually resulting in DIP hyperextension. In practice, the Elson test does not need to be performed over a tabletop and can be performed with the PIP joint in maximum passive flexion to assess extensor tone in the DIP joint compared with the opposite same finger in a similar position.⁹ A digital block should be performed because the examination may be limited by pain. X-rays should be obtained to screen for fracture or dislocation before any manipulation of the finger.

A pseudo-boutonniere deformity can be confused with a boutonniere deformity. The pseudo-boutonniere deformity involves a PIP flexion contracture without increased extensor tone at the level of the DIP joint, which indicates that the extensor mechanism is intact.



FIGURE 2: Mallet finger.

Sagittal band (zone V injury)

Closed sagittal band injuries typically present with focal swelling or tenderness over the involved dorsal MCP joint with or without acute tendon instability.¹⁰ Typically the middle finger is most frequently affected.¹¹ The mechanism of injury is direct trauma, forced flexion, or resisted extension of the MCP joint. Cases of dislocation can also occur with patients reporting a history of flicking or crumpling paper.¹² Atraumatic EDC subluxation can occur in elderly women¹³ (Fig. 5). In these patients, over time, the MCP joint can become stuck in flexion and secondary intrinsic tightness can develop. Atraumatic EDC



FIGURE 3: Mallet fracture. The patient presented with extensor lag and painful dorsal prominence.

subluxation can also occur concomitantly in multiple digits in patients with rheumatoid arthritis.

On examination, the patient will often be able to maintain the MCP joint in extension, but with flexion the extensor mechanism will subluxate into the ulnar valley (most commonly). Once flexed, active extension of the MCP joint will cause a snapping relocation of the extensor mechanism or the joint will remain stuck in flexion, held in place by the volarly and ulnarly displaced extensor tendon. Passive extension of the MCP joint will then allow active extension to proceed as the tendon is displaced from the ulnar gutter. In chronic sagittal band injury, fixed flexion and ulnar deviation of the finger at the MCP joint may occur. The snapping seen with this problem is occasionally mistaken for a trigger finger. Rayan and Murray¹⁰ classified sagittal band injuries into 3 types: type I injuries with no tendon instability, type II injuries with tendon subluxation or snapping, and type III injuries with frank tendon dislocation.

Boxer's knuckle is a related condition involving a tear of the MCP joint capsule, typically resulting from punching, and is often associated with concomitant sagittal band rupture.



FIGURE 4: Positive Elson test with DIP hyperextension indicating central slip injury.

TREATMENT (ACUTE VERSUS CHRONIC)

Mallet

Acute closed mallet finger injuries are treated non-surgically with the DIP joint splinted full time in neutral to full hyperextension for 6 to 8 weeks.¹⁴ This is followed by a weaning off of 4 to 6 weeks of night splinting. The PIP does not need to be included in the splint because PIP joint motion does not affect tendon gapping.¹⁵ Biomechanically, PIP flexion decreases tone in the extensor mechanism over the middle phalanx (with an intact central slip), although this phenomenon is not clinically relevant in the treatment of mallet finger, and prolonged PIP flexion splinting would risk development of PIP flexion contracture. As long as the DIP is immobilized in extension, the exact method of splinting may not matter, because various authors have shown good results using aluminum foam splints, custom thermoplastic splints, or premade plastic splints.

Complications involved in closed treatment of mallet fingers are mostly skin related. Patients should also be informed of the possibility of persistent residual extensor lag.

Crawford⁶ reported on 151 mallet injuries treated using a premade thermoplastic splint for 8 weeks plus 2 weeks at night, with an average of 17 months' follow-up. Of the 35 patients with acute closed tendinous injuries, 5 had extensor lag greater than 10°



FIGURE 5: Sagittal band injury. Ulnar subluxation of the middle finger EDC during MCP flexion in an elderly woman with no history of trauma.

at final follow-up. Okafor et al¹⁶ reviewed 31 patients with acute mallet injuries treated with a dorsal plastic splint at a mean of 5 years, with an average 8.3° residual extensor lag. A recent randomized, controlled trial comparing volar aluminum foam splints, dorsal aluminum foam splints, and custom thermoplastic splints showed no difference in extensor lag at 12 weeks among the 3 groups.¹⁷

Treatment options for chronic mallet finger, loosely defined as injuries presenting weeks to months after injury, include splinting, direct repair, Fowler tenotomy, spiral oblique retinacular ligament reconstruction, and tenodermodesis, among others.

Suh and Wolfe¹⁸ recently reviewed the outcomes of various treatment options for chronic mallet injuries. Good results with less than 10° extensor lag for most patients were demonstrated in small studies involving splinting, tenodermodesis, palmaris longus grafting, central slip tenotomy, and ORL reconstruction.

Kanaya et al¹⁹ reviewed the outcomes of spiral oblique retinacular ligament reconstruction in 7 patients with chronic mallet finger (average, 15 mo) and flexible swan neck deformities. At an average of 19 months' follow-up, 6 of 7 patients had no residual extensor lag.

Our current preferred treatment for acute mallet injury is a dorsally applied aluminum foam splint for

8 weeks, followed by 4 weeks of night splinting. Our preferred treatment for chronic mallet injuries is an 8-week trial of full-time splinting. For patients who fail to respond to splinting, we will generally perform central slip tenotomy for extensor lags up to 35° to 40°, although usually we prefer to wait at least 6 months postinjury for scar tissue in the area to mature, by which time most patients are not bothered by the extensor lag.

Boutonniere

Acute closed central slip injuries as diagnosed by a positive Elson test or after a documented volar PIP dislocation are treated with full-time PIP extension splinting for 4 to 6 weeks to allow the central slip to heal, followed by night splinting for an additional 4 to 6 weeks.²⁰ Active DIP flexion exercises are performed hourly to help coax the lateral bands into the correct dorsal alignment.

Chronic boutonniere injury can be a challenging deformity to treat. Every attempt should be made to obtain full passive motion before consideration of surgery. If the patient fails to correct a PIP flexion contracture with a trial of nonsurgical treatment (PIP extension splinting for 4–6 wk or serial casting into PIP extension), operative PIP joint release followed by therapy would be required to obtain maximum passive motion. Once passive motion of the PIP joint is obtained, several treatment options are available to rebalance the extensor mechanism: terminal tendon tenotomy, tendon grafts,²¹ direct central slip repair, and lateral band mobilization. Techniques that mobilize the contracted lateral bands and then suture them dorsally may limit PIP flexion because this restricts the natural dorsal-volar movement of the lateral bands with PIP flexion and extension.

Our current preferred treatment for acute central slip injuries is PIP extension splinting for 6 weeks followed by night splinting for 4 to 6 weeks. Active DIP flexion exercises are performed to help draw the lateral bands into the correct dorsal alignment. For chronic, flexible boutonniere deformities, we favor the staged approach proposed by Curtis et al.²² Active PIP extension is assessed at each successive stage of the procedure, and if extension of the PIP is achieved, the operation is stopped. In stage 1, an extensor tenolysis is performed. Stage 2 consists of a transverse retinacular ligament release. Stage 3 involves a terminal tendon tenotomy. Stage 4 involves advancement of the central slip. Curtis et al demonstrated that 17 patients who had an average of 41° extensor lag were treated with this technique and improved to 10° extensor lag.



FIGURE 6: Custom MCP flexion block splint.²³

Sagittal band

Acute closed sagittal band injuries with no instability (ie, type I) can be treated with simple buddy taping. Acute injuries with subluxation diagnosed within 3 to 4 weeks can be treated with a custom-made sagittal band MCP flexion block splint for 8 weeks (Fig. 6).

Catalano et al²³ reviewed 11 acute sagittal band injuries treated with a custom MCP joint flexion block splint for 8 weeks. At an average of 14 months, 8 of 11 injuries had no pain and either minimal or no subluxation.

Chronic sagittal band injuries or acute injuries failing nonsurgical treatment are treated operatively. Many procedures have been described involving either primary repair of the sagittal band²⁴ or tendon stabilization using various grafts such as junctura tendinum,²⁵ local proximal or distal based partial EDC slips,^{26,27} free tendon grafts, or accessory tendons.²⁸ Although no large studies have directly compared the various techniques, most small case series have shown resolution of subluxation and dislocation with surgical repair and stabilization.

Three new techniques have recently been described. In 2010, Kang and Carlson²⁹ described creating a horizontal metacarpal tunnel and tendon graft to create a new pulley stabilizing the extensor tendon. A palmaris longus graft or strip of juncturae tendinum was used. All 6 patients had resolution of painful subluxation at 3 months' follow-up. In 2012, Beck et al²⁸ described 2 cases of type III sagittal band injuries of the middle finger treated with an accessory extensor tendon to the middle finger (either the extensor indicis et medii communis or the extensor medii proprius). The accessory tendon was looped around the EDC to form a restraint to dislocation. Both patients had resolution of subluxation at 2-year follow-up. ElMaraghy and Pennings³⁰ described reconstruction of a middle finger radial sagittal band using an ulnar-sided local junctura tendinum graft. The junctura graft was extended with a contiguous, distally

based, half-thickness EDC graft, and the graft was passed through the EDC in the radial direction and sutured to the intermetacarpal ligament. They reported results of 1 patient at 5-month follow-up, with no residual subluxation.

Our current preferred treatment for acute (< 3 wk from injury) sagittal band injuries is a custom-molded MCP flexion block splint for 8 weeks. For chronic injuries or those failing nonsurgical treatment, we prefer the technique of Carroll et al,²⁶ of a proximally based partial EDC slip looped around the radial collateral ligament.

In conclusion, the extensor mechanism of the hand is complex and can be disrupted at several different points through hand trauma. Closed mallet finger, boutonniere, and sagittal band injuries are commonly seen by the hand surgeon, and can result in considerable deformity or disability if left untreated. When diagnosed early, closed treatment with splinting generally produces excellent results. However, patients should be counseled about the possibility of treatment failure and residual extensor lag or permanent deformity.

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JOURNAL CME QUESTIONS

Closed Soft Tissue Extensor Mechanism Injuries (Mallet, Boutonniere, and Sagittal Band)

How much elongation of the terminal tendon will result in 25° of extension lag and mallet deformity at the distal interphalangeal joint?

- a. 1 mm
- b. 3 mm
- c. 5 mm
- d. 7 mm
- e. 9 mm

Which of the following should be the initial treatment for closed acute type III (tendon dislocation) sagittal band injury?

- a. No treatment should be offered
- b. Steroid injection and therapy
- c. Metacarpophalangeal flexion block splint
- d. Surgical repair of the severed sagittal band
- e. Surgical reconstruction with local tendon graft

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