Acute Mallet Finger Injuries—A Review

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- The anatomy of the terminal tendon and the pathophysiology of closed and open mallet injuries.
- The diagnosis and classification of mallet injuries.
- The recommended treatment and reported outcomes for mallet injuries.

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Acute disruptions of the terminal extensor tendon are common and can result in significant dysfunction if not recognized and treated appropriately. This article provides a topical review of the contemporary literature concerning acute mallet finger injuries. It also proposes a modification to the Doyle classification to make it more encompassing and less prone to

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Key words Extensor, mallet finger, terminal tendon.

THE TERM "MALLET FINGER" has come to mean a disruption to the terminal extensor mechanism, with a lack of full or active extension through the distal interphalangeal (DIP) joint. It is a misnomer, for rarely do these digits actually resemble a mallet. Other descriptions have been based on etiology, such as "baseball finger" or "cricket finger"; however, perhaps the most apt title is "drop finger."

These injuries are common, and although a precise incidence is difficult to ascertain because of the wide variety of etiologies and presentations seen, it is estimated that they comprise almost 10% of all tendinous and ligamentous injuries in the body.[1] If not recognized and treated appropriately, these injuries can result in significant dysfunction. This article provides a topical review of the contemporary literature concerning acute mallet finger injuries.

ANATOMY AND BIOMECHANICS

The extensor apparatus is a complex, linked system between the radial nerve innervated extrinsic musculature and the median and ulnar nerve innervated intrinsic musculature. Mallet finger injuries result from a disruption of the extensor apparatus, primarily in the region corresponding to zone I of the Kleinert and Verdan topographic classification, between the convergence of the lateral bands proximally and its insertion onto the distal phalanx distally.[2] This segment of the extensor apparatus can be termed the terminal tendon (TT; Fig. 1).[2]

Schweitzer and Rayan[2] dissected more than 50 cadaveric digits with no underlying pathology to investigate the anatomy of the TT. They found that the TT is a flat and thin structure with dense, longitudinally oriented fibers. Although the TT was located primarily in Kleinert and Verdan zone I, the proximal margin extended into zone II, spanning from the distal third of the middle phalanx to the proximal aspect of the distal phalanx. On average, the TT was 10.1 mm (±2.6; 5.1−15.9 mm) in length from the convergence of the lateral bands to the insertion; 1.1 mm (±0.2; 0.8−1.4 mm) thick and 5.6 mm (±0.9; 4.0−8.2 mm) wide at the level of the DIP joint; and inserted on the dorsal articular margin of the distal phalanx, extending distally 1.2 mm (±0.4; 0.8−1.7 mm).[2] The volar aspects of the TT and the dorsal capsule of the DIP joint were found to be intimately associated.

It is important to be aware of the relationship between the TT and the germinal matrix when performing surgery in this region. The rate of nail plate deformity after surgical intervention in mallet finger injuries may be as high as 18%.[3] Schweitzer and Rayan[2] found that the TT insertion was located a mean of 1.4 mm (±0.6; 0.9−2.0 mm) proximal to the edge of the germinal matrix and nail plate (Figs. 2, 3).[3] Similarly, Shum et al[3] reported an average distance of 1.2 mm (0.9−1.8 mm) between the insertion of the TT and the proximal margin of the germinal matrix.

Schweitzer and Rayan[4] also examined the kinematics of the intact and disrupted TT. An excursion of between 1 to 2 mm was noted in the intact TT during full DIP joint range of motion, with no apparent change in length or fiber orientation throughout. No substantial change in excursion was noted with alterations to the positions of the more proximal joints. The TT was then sectioned via Z-cuts and repaired at altered lengths to assess variations in DIP joint posture. The resting DIP joint flexion (mallet) posture was found to increase with incremental lengthening of the TT, with the middle finger generally revealing the greatest amount of increased flexion at each lengthening (Table 1).[4]

Husain et al[5] created osteotomies through the articular surface of cadaveric distal phalanges without evidence of osteoarthritis to replicate bony avulsions of the extensor apparatus. They then loaded the extrinsic flexor and extensor musculature and subjected the digits to 1,200 cycles of full range of motion to approximate full flexion and extension of a finger 5 times an hour for 16 hours per day over 2 weeks. They found that there was no subluxation of the DIP joint if the avulsed fragment measured <43% of the joint surface, and consistent subluxation when >52% of the articular surface was involved.[5]

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Although common, the combined incidence of bony and soft tissue TT injuries is difficult to quantify. An

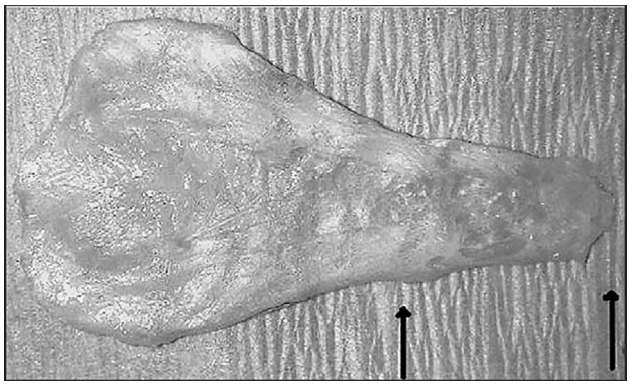

FIGURE 1: The distal extensor mechanism showing the TT between the convergences of the lateral bands to its insertion [between arrows]. The thin triangular ligament between the lateral bands is also noted. Reproduced with permission.[2]

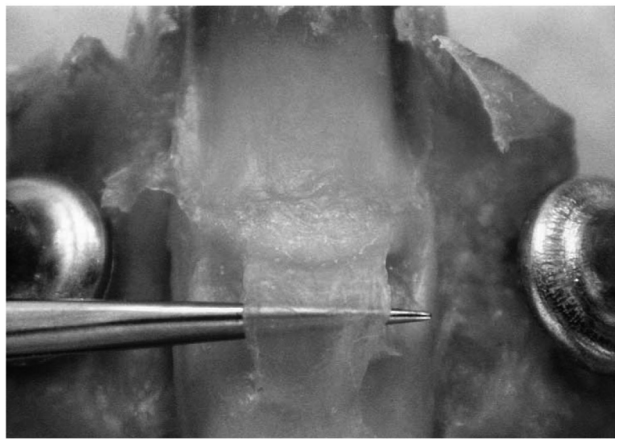

FIGURE 3: The TT *in situ*, showing proximity to the germinal matrix. Reproduced with permission.[3]

FIGURE 2: The TT *in situ*, showing proximity to the germinal matrix (shown by arrow). Reproduced with permission.[2]

TABLE 1. Influence of TT Lengthening for All Digits. Reproduced with permission.[4]

TT Lengthening	DIP Joint Resting Posture (° of Flexion)
TT Intact	22 (16–28)
At 1 mm	25 (20–32)
At 2 mm	36 (30–46)
At 3 mm	49 (38–60)
At 4 mm	63 (50–90)

epidemiologic study of soft tissue musculoskeletal injuries found that mallet fingers were the fifth most common in the body, accounting for approximately 10% of all injuries.[6] A male preponderance was noted in the first 3 decades of life, likely because of sporting or social activities. The incidence was much lower for women in the second and third decades but rose steadily to match the incidence in men with a peak in the sixth decade (Fig. 4).[6] Jones and Peterson[7] investigated 20 closed mallet deformities in 7 members of a 24-member family, the majority of which occurred spontaneously or with minimal trauma, suggesting an underlying genetic predisposition. The 3 ulnar digits are most commonly involved in closed injuries, with the middle finger at particular risk because of its relative length, as well as the biomechanical peculiarities discussed previously.[4] A retrospective cohort study of penetrating trauma to the hand and wrist also reported a male predominance, and found that zone I injuries of the extensor tendons to the thumb and lesser digits comprised 8.2% of all injuries.[8]

Closed injuries to the TT occur via a 2-step process, with an initial axial load applied to an extended digit, followed by either a hyperflexion moment

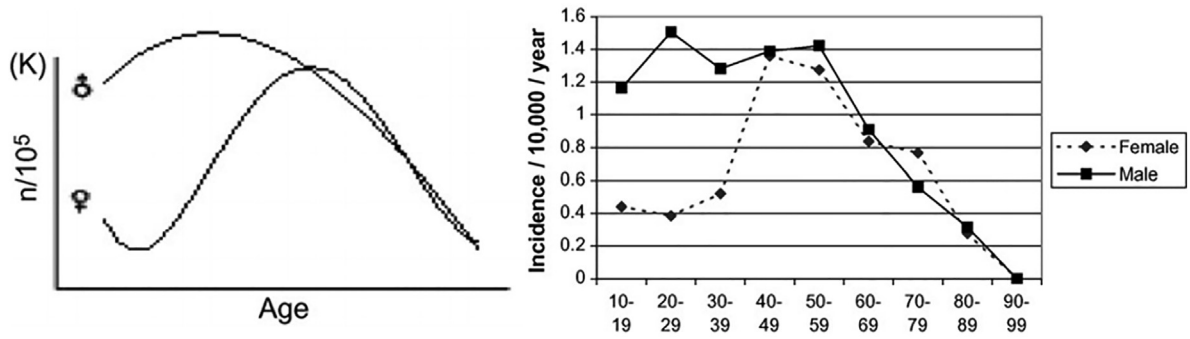

FIGURE 4: Injury distribution curves and age-specific incidence of mallet fingers. Reproduced with permission.[6]

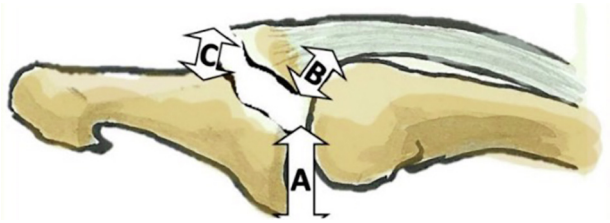

FIGURE 5: The ratio of displacement of the avulsed fragment compared with the articular surface: D = C/[A + B]. Reproduced with permission.[1]

against resistance (resulting in a soft tissue disruption or avulsion fracture) or hyperextension moment (causing a dorsal lip fracture as the distal phalanx abuts the head of the middle phalanx).[1,9] The energy required to cause these injuries is dependent on patient age and tissue quality. In younger patients, a higher degree of energy is often imparted, whereas in an older cohort, these injuries may occur during activities of daily living.[1]

DIAGNOSIS AND CLASSIFICATION

Patients may present experiencing pain, swelling, deformity, and dysfunction at the DIP joint after trauma; alternatively, the deformity may be an incidental finding. In the chronic setting, patients may also present with a swan-neck deformity (in keeping with the intercalary collapse theory of Landsmeer) because of proximal migration of the extensor mechanism with increased tone across the proximal interphalangeal (PIP) joint.[9]

Passive correctability of the extension deficit is important to quantify because a fixed flexion deformity in the setting of chronic TT injury is a contraindication to reconstruction. Although no consensus exists on the optimal method of measuring extensor lag, the use of a goniometer on the dorsal aspect of the DIP joint is likely to be reproducible.[1]

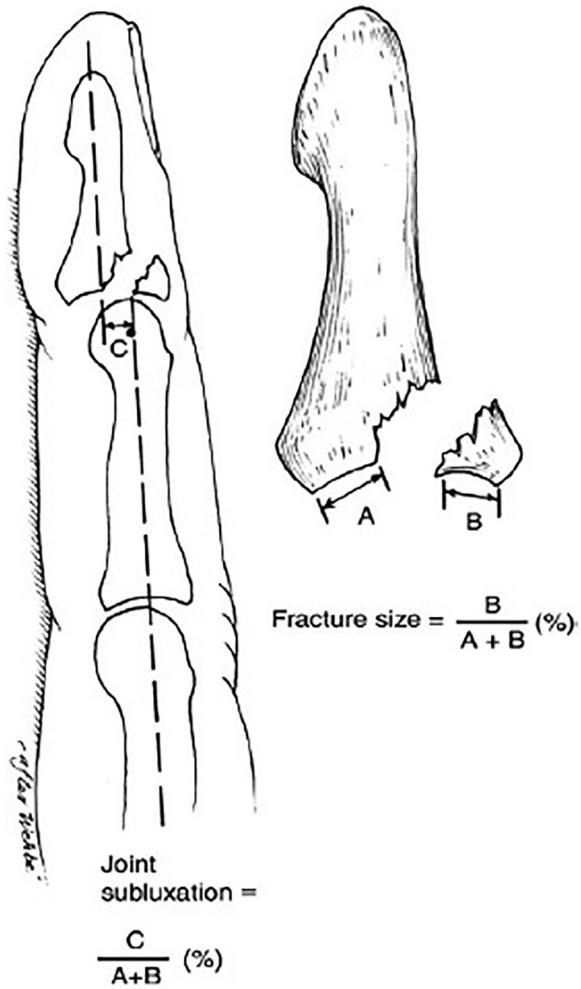

FIGURE 6: Alternative methods to quantify fracture size and joint subluxation. Reproduced with permission.[5]

Suspected injuries to the TT should be investigated via anteroposterior and lateral radiographs of the injured digit to assess for bony involvement, subluxation of the DIP joint, or injury to a physis. Various methods have been proposed to quantify the proportion of bone avulsed, the degree of

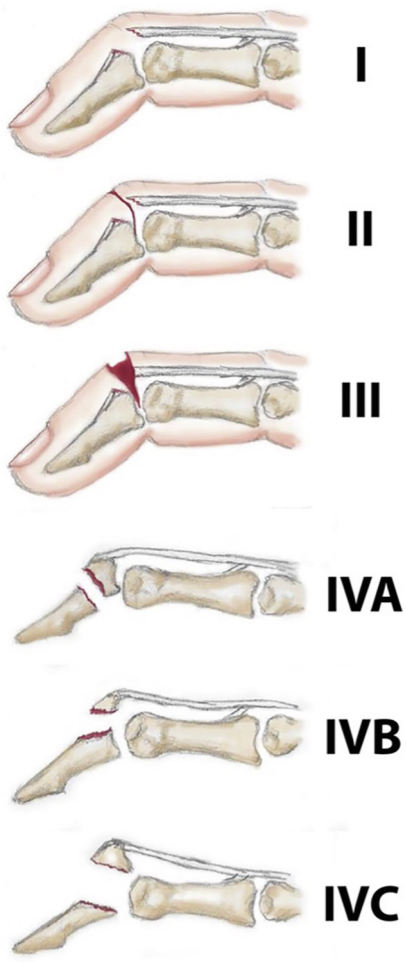

FIGURE 7: The Doyle classification of TT injuries. Type I—closed injury; type II—open tendinous injury; type III—open injury with the loss of skin and substance of the extensor tendon; type IVA—physeal injury; type IVB—fragment between 20% and 50% of the articular surface; type IVC—fragment >50% of the articular surface. Reproduced with permission.[13]

displacement of the avulsed fragment, and the amount of joint subluxation (Figs. 5, 6).[1,5] In practice however, radiographic assessment is usually performed via the naked eye using clinician experience.[1,5] Radiographs will also allow the exclusion of other diagnoses, such as dystelephalangy (Kirner's deformity) or the sequelae of prior distal phalangeal physeal injury.

Terminal tendon injuries can be categorized by soft tissue envelope violation as open or closed, and by the time to presentation, with Gerberman et al[10] defining those presenting >4 weeks after injury as chronic. A number of formal classification systems have also been proposed, although deficiencies in scope have limited clinical adoption. Wehbe and Tubiana[11] broadly categorized closed injuries based on the presence and size of an avulsed fragment, volar subluxation, and physeal injury.[12]

Perhaps the most comprehensive classification system is that suggested by Doyle, which categorizes closed tendinous disruptions as type I and open tendinous injuries as type II. Those with segmental loss requiring reconstruction are type III (Fig. 7).[13,14] Type IV injuries are bony, and can be physeal (type IVA), involve less than half of the articular surface (type IVB), or comprise more than half of the articular surface (type IVC). This classification is limited in that it does not include open bony injuries; further, the division of bony injuries into less than or greater than half of the articular surface is clinically unhelpful, prone to interobserver error, and does not address the important issue of joint subluxation. Thus, the authors suggest a modification to the type IVB and IVC subsets to indicate a bony injury without or with DIP joint subluxation, respectively, regardless of the proportion of the articular surface involved. We also suggest the inclusion of open bony injuries within a type V category, with further subdivisions as per type IV (Fig. 8).[15]

NATURAL HISTORY AND TREATMENT

Both soft tissue and bony TT disruptions heal in a stepwise fashion. As with all tendon injuries, a tendinous disruption of the terminal extensor initially undergoes an inflammatory phase, with the recruitment of neutrophils within the first 24 hours, followed by monocytes (in particular macrophages).[16] These cells serve to phagocytose necrotic tissue, as well as secrete vasoactive and chemotactic factors that increase angiogenesis and initiate tenocyte proliferation and synthesis of collagen type III.[1,17] The recruitment and proliferation of intrinsic tenocytes (located in the endotenon and epitenon) and fibroblasts (from the paratenon and synovial sheath) peak in the proliferative stage, which commences approximately 2 days after injury.[16] The remodeling phase begins approximately 1 month after injury, with an alteration in collagen content (type III to type I) and a decrease in cellularity and glycosaminoglycan content.[16,18] The maturation of the fibrous tissue to a scar-like tendon starts at 10 weeks and continues for up to a year. It is important to note that the disrupted tendon never completely regains its preinjury biomechanical, biochemical, or ultrastructural properties.[1,17]

Bony injuries similarly undergo an initial inflammatory phase. The formation of a fracture hematoma leads to platelet activation and cytokine release,

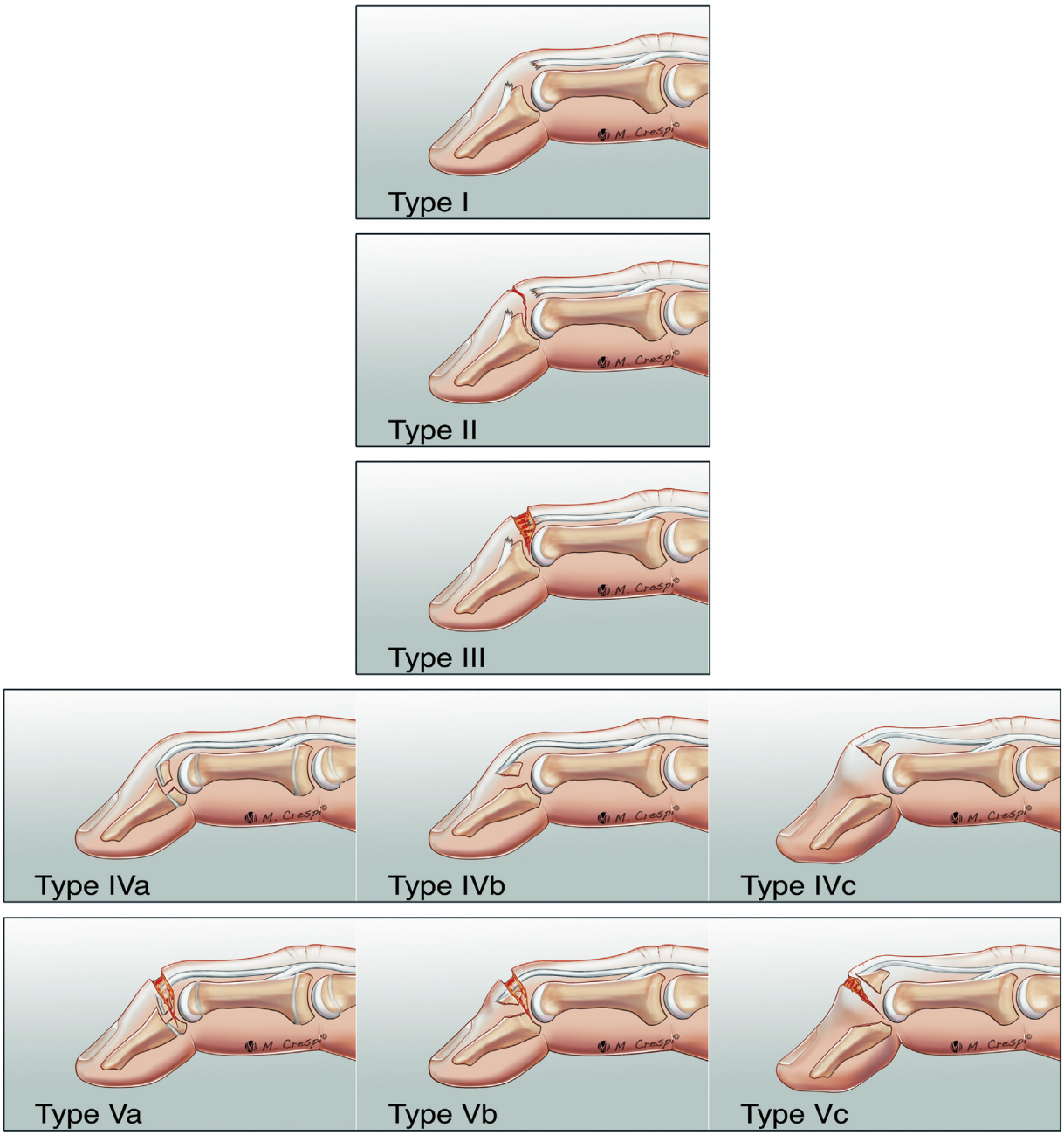

FIGURE 8: The suggested modification of the Doyle classification. Type IVB and IVC indicate bony injuries without or with joint subluxation. Type V encompasses open bony injuries.

resulting in the recruitment of inflammatory and reparative cells.[1] In the proliferative phase during the third and fourth weeks, a bridging cartilaginous scaffold is formed. This is then replaced by immature bone in the maturation stage, which commences in the fifth week. Remodeling to mature lamellar bone, because of linked osteoclastic and osteoblastic action, occurs from the 12th week after injury onward.[1]

The goal of management, therefore, is to maintain the tendinous or bony disruption in a reduced position for an appropriate duration, allowing the aforementioned processes to occur and restoring active DIP joint extension. Closed tendinous injuries can be managed nonsurgically via splinting, as can closed osseous injuries where an acceptable alignment of the fracture can be achieved without subluxation of the joint. These 2 subgroups comprise the vast majority of TT disruptions. Early splinting protocols immobilized the PIP and DIP joints to prevent distal slippage of the cast, and because of concerns

that PIP joint motion would transmit tension through the intrinsic musculature resulting in gapping.[19] From the latter half of the 20th century, splinting of just the DIP joint has been deemed adequate, with Katzman et al[19] proving on cadaveric sectioned terminal extensor tendons, that movement of the PIP joint does not cause tendon gapping, but flexion of the distal phalanx does.

Orthoses can be broadly divided into volar bearing, dorsal bearing, or combined. The major concern with volar-bearing orthoses is insufficient DIP joint hyperextension. In contrast, dorsal or combined orthoses may impart excessive pressure on the inflamed injured region, resulting in skin necrosis or nail dystrophy.[1] Equivalent results have been found regarding the range of motion, complications, or patient-reported outcomes when comparing volar, dorsal, and custom thermoplastic orthoses.[20] Full-time immobilization of bony injuries is recommended for a duration of 6 weeks, whereas tendinous disruptions are usually splinted for at least 8 weeks to account for the longer duration required for tendon healing. Satisfactory results have been reported with splinting of closed soft tissue injuries with delayed presentations, with no difference in residual lag and range of motion noted between those who presented before and after 4 weeks post-injury, and should thus be considered first-line therapy for this subgroup.[10] Anecdotally, splinting can be trialed at any point after injury while there is evidence of residual inflammation at the DIP joint.

The authors recommend a position of approximately 10° hyperextension when immobilizing closed soft tissue injuries to allow apposition of the disrupted tendon ends and a neutral position for bony injuries to prevent DIP joint subluxation. A period of orthosis weaning (including nocturnal splinting) has traditionally been used after the cessation of full-time immobilization. However, recent evidence has questioned the use of this measure, with no difference in extensor lag noted between those who underwent nocturnal splinting and those who did not.[21] Ultimately, patient compliance and good hand therapy services are key to the success of nonsurgical treatment.

Although no consensus exists, indications for surgical intervention in the acute or subacute setting that have some unanimity include open injuries and bony disruptions with DIP joint subluxation despite splinting. Some authors have advocated for the fixation of bony injuries based on the proportion of the articular surface involved, with thresholds ranging from greater than a third to more than two-thirds.[1] Although contentious, surgical intervention may also be contemplated in patients who do not wish to undergo splinting for professional or other considerations. A recent retrospective analysis comparing splinting with pinning for acute closed tendinous mallet injuries reported a significantly improved extension lag in those who underwent surgery (2.1° vs 13.8°). This finding should be considered when managing these patients and is an area for further investigation.[22]

Open soft tissue disruptions can be repaired via the surgeon's suture configuration of choice if there is enough residual tendinous tissue available, and via suture anchors or transosseous tunnels if not. The primary author's preference is to use a running-interlocking horizontal mattress suture with a dissolvable monofilament suture if the tissue suffices because of the advantages of stiffness, lack of tendon shortening, and the decreased time to perform the repair compared to other methods, as demonstrated in prior cadaveric models.[23] A Kirschner wire is often used to transfix the joint and protect the soft tissue repair. Significant or segmental tendon loss may require grafting, as even 1 mm of shortening can result in severely restricted DIP joint flexion.[4,14]

A number of fixation methods have been reported for bony injuries and can be broadly divided into open or percutaneous methods, as well as via the use of sutures, wires, screws, or plates as the primary mode of fixation. No superiority has been demonstrated between fixation methods in the literature. Ishiguro et al[15] noted that hyperflexion of the DIP joint reduced an avulsed bony fragment into the trochlea of the middle phalanx and devised a technique of extension block wiring to hold this dorsal fragment in a reduced position prior to the reduction of the distal fragment to achieve a congruent articular surface and longitudinal pinning (Fig. 9). They advocated this technique for noncomminuted fractures within 5 weeks of injury. They also suggested the use of a hypodermic needle to clear the fracture site percutaneously if the injury was older than 3 weeks. Kirschner wires may also be utilized to transfix the DIP joint alone, joystick fragments into position, bent into an 'umbrella handle' which is then pulled to effect reduction, or be incorporated as part of a tension band construct in the setting of comminution.[9] Open reduction of bony mallet injuries can be secured via sutures or wires passed through or around the bony substance of the distal phalanx

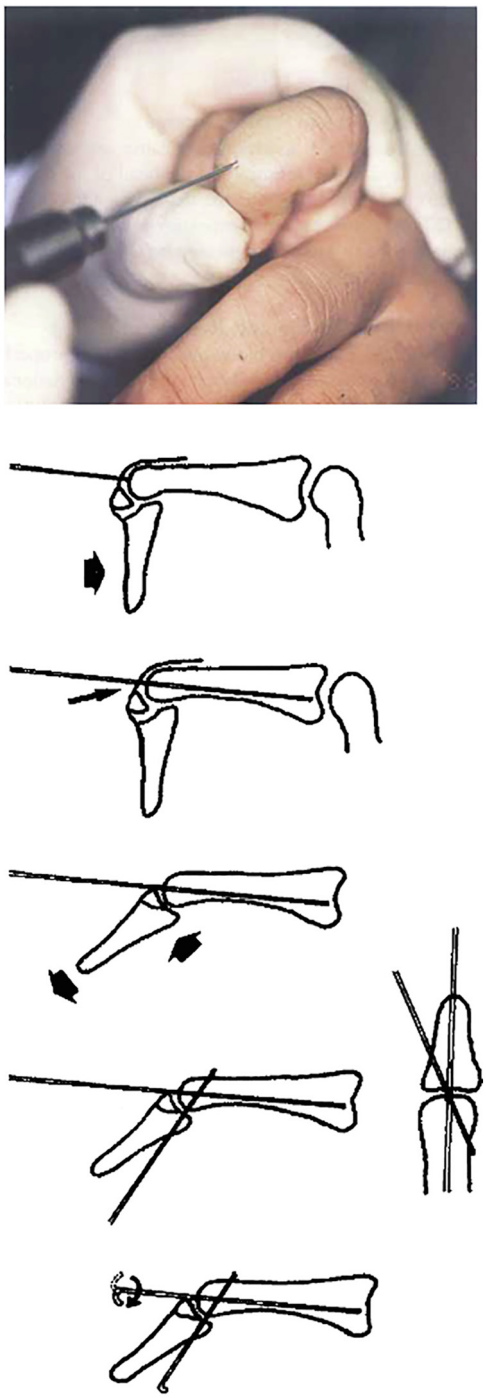

FIGURE 9: The method of extension block pinning, showing the PIP and DIP joints held in maximal flexion to effect reduction; the extension block pin is introduced into the middle phalanx 1−2 mm dorsal to the fragment; the remainder of the distal phalanx is reduced to a congruent articular surface; the DIP joint is transfixed in the reduced position. Reproduced with permission.[15]

and secured around the volar septa, or on the pulp for later removal, screw fixation (often augmented with a transfixing Kirschner wire), or the application of a plate.

TABLE 2. Crawford Evaluation Criteria

Grade	Description
Excellent	Full DIP joint extension, full flexion, no pain
Good	0°−10° extension deficit, full flexion, no pain
Fair	10°−25° extension deficit, any flexion loss, no pain
Poor	>25° extension deficit or persistent pain

OUTCOMES

The classification described by Crawford is most commonly used to assess outcomes after disruption of the TT. It stratifies patients based on residual lag, range of flexion, and the presence of pain (Table 2).[9] A systematic review in 2017 of all available literature identified 44 studies for inclusion, with 22 studies evaluating surgical treatment, 17 studies reporting nonsurgical measures, and 5 providing a direct comparison.[13] Overall, surgery offered a slightly decreased (but likely clinically insignificant) reduction in extensor lag when compared with nonsurgical management, with interstudy calculations of 5.7° and 7.6°, respectively. Similar rates of complications were also noted for surgical (14.5%) and nonsurgical (12.8%) management. However, surgery was more likely to result in serious complications, such as secondary fracture displacement, tendon rupture, and skin necrosis.[13] The majority of complications seen with nonsurgical treatment were transient skin issues, such as maceration or irritation; only 1 serious complication of full-thickness skin ulceration was found in the literature. No stratification by bony or tendinous injury was possible, due to the majority of included studies not providing adequate subanalyses. The review concluded that the treatment options are equivalent, and that treatment should be individualized to the patient.

In conclusion, acute disruptions of the terminal extensor tendon are common and can largely be managed nonsurgically with satisfactory outcomes. We propose a modification to the Doyle classification to make it more encompassing and less prone to interobserver error. Further studies comparing surgical with nonsurgical measures, and elucidating indications for surgery, will be beneficial.

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JOURNAL CME QUESTIONS

Acute Mallet Finger Injuries—A Review

1. The terminal tendon insertion is located approximately how far proximal to the germinal matrix?

a. 1.4 mm
b. 2.5 mm
c. 3.5 mm
d. 4.5 mm

2. Closed injuries to the terminal tendon occur as a result of which of the following sequential combination of forces?

a. Hyperextension followed by flexion.
b. Hyperflexion followed by axial loading.
c. Axial loading followed by hyperflexion.
d. Axial loading following by radial deviation.

3. Tendon healing following disruption occurs in which of the following sequences?

a. Proliferation, inflammation, remodeling, and maturation
b. Inflammation, proliferation, remodeling, and maturation
c. Inflammation, proliferation, maturation, and remodeling
d. Proliferation, inflammation, maturation, and remodeling

4. Which of the following is an absolute operative indication following a terminal tendon extensor injury?

a. Closed soft tissue injury
b. Bony disruptions with distal interphalangeal joint subluxation despite splinting
c. Patients who are noncompliant with splinting
d. Patients who want a guarantee against the development of an extensor lag

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