Extensor Tendon Injuries in the Hand

Mary Lynn Newport, MD

Abstract

Until recently, extensor tendon injuries were often discounted as an important hand problem. However, studies have shown that not all extensor lacerations fare well and that loss of flexion can be problematic. Newer postoperative protocols emphasizing tendon gliding have improved results, and better repair techniques and postoperative rehabilitation regimens are under investigation. This article reviews the evaluation of acute open and closed extensor tendon injuries, their conservative and surgical treatment, and postoperative rehabilitation options.

J Am Acad Orthop Surg 1997;5:59-66

The extensor mechanism of the hand and digits has received considerable attention in the reconstruction of chronic deformities, but acute injuries have received far less attention. Although the extensor mechanism is relatively superficial and therefore easy to expose, operative repair can be technically challenging because the extensor tendons are thin and flat, are weaker than flexor tendons, have less gliding amplitude, and are difficult to suture well.

There has been a resurgence of interest concerning acute extensor tendon repairs because outcomes after traditional static splinting have been shown to be less than acceptable by contemporary standards.[1-5] Investigations have shown that dynamic postoperative protocols can improve the outcome.[6-10] Stronger suture repair techniques and postoperative protocols with active motion are currently being developed.[11,12] This article will review the diagnosis and treatment of acute open and closed extensor injuries in zones I through VIII, as well as recent advances in treatment and rehabilitation.

Anatomy

The extensor mechanism of the hand can be divided into eight zones (Fig. 1) to aid in the evaluation and treatment of acute injuries.[13] The even-numbered zones are over bones, and the odd-numbered zones are over joints. The different numbering for the extensor mechanism of the thumb reflects its smaller number of phalanges.

Zone VIII, the most proximal zone, contains the musculotendinous junction. In zone VII, the tendons lie within an enveloping tenosynovium and the sheath of the extensor retinaculum over the wrist joint. Under normal circumstances, diffusion is responsible for most of the tendon nutrition in this region.[14] The vascular supply is derived from the mesotendon, which runs the entire length of the retinaculum. In the remaining zones, the extensor tendons are covered by paratenon, and nutrition is supplied primarily by perfusion. The intratendinous vascular architecture outside the retinaculum is similar throughout the tendon, with small branches to the tendon from the surrounding fascia.[15]

In zone VI, the tendons of the long, ring, and small fingers are connected by juncturae tendinum that course distally and obliquely. These interconnections must be considered when evaluating extensor tendon injuries. The extensor indicis proprius and extensor digiti quinti, which run ulnar to their respective extensor digitorum communis (EDC) tendons and allow independent extension of the index and small fingers, must also be considered in an evaluation.

In zone V, the extensor tendons are centrally located over the metacarpal head, held in place by sagittal bands that run on each side

Dr. Newport is Associate Professor, Department of Orthopaedic Surgery, University of Connecticut Health Center, Farmington.

Reprint requests: Dr. Newport, Department of Orthopaedic Surgery, University of Connecticut Health Center, 10 Talcott Notch Road, Farmington, CT 06034-4037.

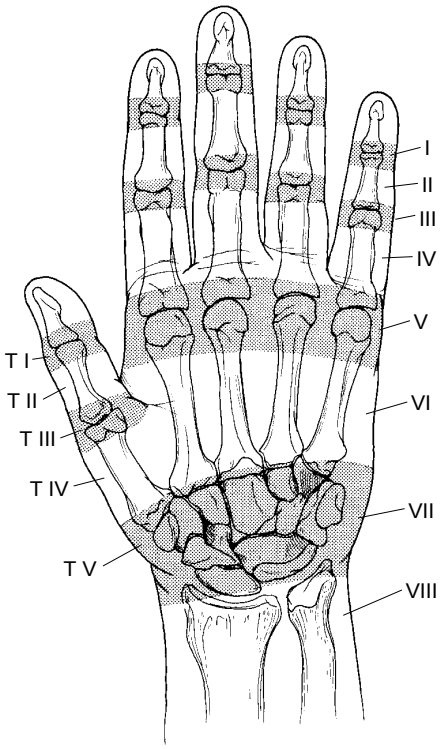


Fig. 1 The eight extensor tendon zones. T = thumb.

of the tendon and attach palmarly to the volar plate of the metacarpophalangeal (MCP) joint. The extrinsic extensor insertion into the proximal phalanx itself is quite weak.

The extensor mechanism becomes more complicated over the digit as the EDC becomes linked to the intrinsic mechanism of the lumbricals and the dorsal and volar interossei. The EDC tendons extend the MCP joints by pulling up on the sling formed by the sagittal bands. The EDC tendons are also capable of extending the interphalangeal joints if hyperextension of the MCP joints is prevented. Resistance to MCP hyperextension is provided primarily by the intrinsic musculature, as the volar plates of the MCP joints do not prevent hyperextension. Because the intrinsic tendons are directed volar to the axis of the MCP joint, they flex this joint. The intrinsic tendons continue distally as lateral bands, with contributions inserting into the dorsum of the middle phalanx along with the central slip of the EDC. The last continuations of the lateral bands meet dorsally at the midportion of the middle phalanx, forming the terminal extensor tendon, which inserts on the distal phalanx. Because the intrinsics course dorsal to the axis of the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints, they extend these joints.

The intrinsic and extrinsic extensor systems are intricately coordinated during joint flexion and extension. Injury or adhesion formation between the extensor mechanism and adjacent tissues will disturb this delicate balance and potentially limit tendon excursion and joint motion.

History

Despite the work of Dargan[1] and others[2,3] relating good and excellent results in only two thirds of cases, many review articles and handbooks indicate that extensor tendon injuries do uniformly well. In a large series of extensor injuries treated by static splinting, Newport et al[5] reported good or excellent results (using a rating system developed by Miller[16] [Table 1]) in only 52% of cases. They showed that good or excellent results occurred more frequently in the four proximal extensor zones than in more distal zones (65% vs 40%).[5] These results are consistent with the anatomy, as the extensor mechanism in the proximal zones is less complex and less apposed to bone. The extensor mechanism in the distal zones, as previously mentioned, is significantly more complex and more closely apposed to bone.

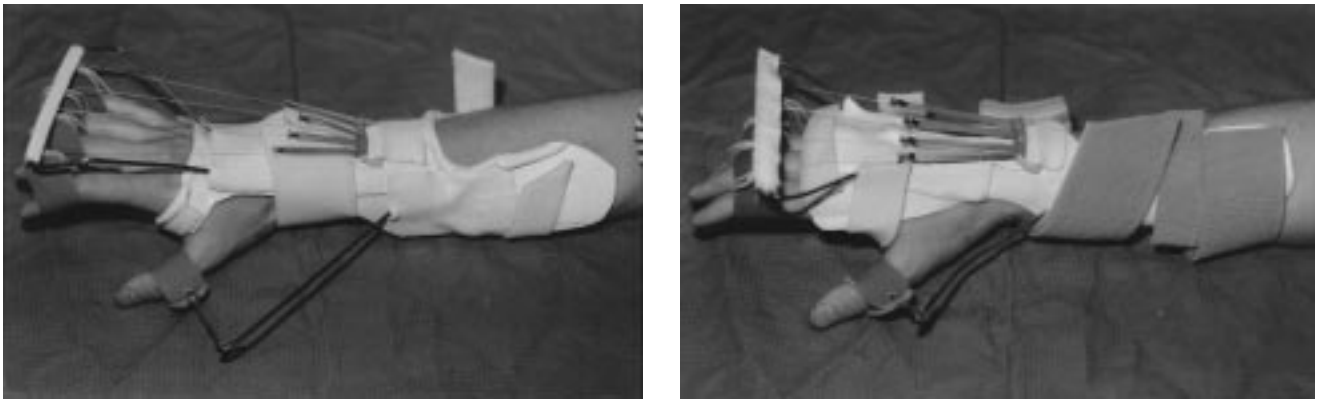
Hung et al[9] and others[17] have also noted poorer results in injuries over the digit. An unexpected finding in the series by Newport et al[5] was that injuries within the retinaculum of zone VII did not have outcomes different from those in zones VI and VIII; all three areas had approximately 66% excellent or good results. Others had predicted a poorer result in zone VII, hypothesizing that the surrounding tenosynovium and the enclosing retinaculum would be a greater source of adhesion formation.[8,17]

Two thirds of all extensor tendon lacerations are associated with concomitant injury to bone, skin, or joint. In three large series,[3-5] injuries associated with fracture had a poorer outcome (50% good or excellent results); joint capsule laceration did not adversely affect outcome (64% good or excellent results). Overall loss of flexion exceeded overall loss of extension.[5]

Dynamic splinting for extensor injuries has been shown to markedly improve results compared with static splinting, with 98% to 100% good or excellent results.[6,7,11] Dynamic splinting typically involves a rubber-band outrigger apparatus (Fig. 2, A). It is based on

Table 1
Miller's Classification of Result After Extensor Tendon Repair

Result	Total Extensor Lag	Total Flexor Loss
Excellent	0°	0°
Good	≤10°	≤20°
Fair	11°–45°	21°–45°
Poor	>45°	>45°



A B

Fig. 2 **A,** Traditional dynamic extension splint. **B,** Dynamic extension splint modified with dorsal hood to minimize extensor muscle activity.

principles developed for flexor tendon rehabilitation whereby protected gliding of tendons decreases adhesion formation without causing undue stress at the repair site. Although Duran estimated that 3 to 5 mm of excursion is required to prevent adhesion formation after flexor tendon repair, no such criterion exists for extensor tendons. Indeed, there is disagreement about how much extensor tendon excursion occurs in the noninjured hand.[8,18-21] Dynamic extensor splinting has also been used in more distal zones, including zones III and IV, with improved results compared with injuries treated by static splinting.[9,10]

The relatively poor results with static splinting and the enthusiasm directed toward dynamic splinting led to electrophysiologic investigation of the dynamic splinting technique. Unexpected and inappropriate extensor musculature activity occurred during both active flexion and passive extension and at rest.[22] The addition of a dorsal block hood, which holds the MCP joints in 15 degrees of flexion (Fig. 2, B), halted this inappropriate activity. While inappropriate muscular activity has not been shown to cause extensor repair rupture with standard dynamic splinting, it does point to a peril peculiar to extensor tendon repair.[6-8] Many assumptions based on flexor tendon repair, but without substantiation for extensor tendon repair, have been made in the past. These are now being consistently challenged with laboratory and outcomes analysis.

The clinical success of the new rehabilitation protocols has stimulated studies on repair techniques.[11,18,23,24] Common suture techniques (Fig. 3) used for repair in zones IV and VI have been evaluated for their strength, tendency to shorten the tendon, and potential effect on digital range of motion.[23,24] It has been shown that there may be an important iatrogenic component to fair and poor results, at least in zone VI, where common suture techniques typically shorten the tendon approximately 6 mm. In a cadaveric model, this amount of shortening produces an 18-degree loss of motion at both the MCP and PIP joints.

These biomechanical studies have shown that the modified Bunnell technique produces the best repair in zone VI, providing the optimal combination of tendon shortening, resultant MCP and PIP range of motion, and repair strength.[23] The modified Bunnell and modified Kessler techniques are equally effective in zone IV.[24] However, a newly modified Bunnell technique and a whipstitch technique have recently been shown to be stronger and more advantageous in a cadaveric model.[12] These techniques are simple to use and add approximately one third greater strength, as judged by 2-mm gap formation and load to failure, than the usual modified Bunnell technique. Clinical testing currently under way shows promising early results.

Other studies have investigated the effect of wrist position on extensor tendon excursion; however, there continues to be disagreement about the amount of extensor excursion that occurs, varying from 2 mm to as much as 8 mm.[8,18-20] Additional research has analyzed mathematically the forces across the extensor system that occur with short-arc active motion (0 to 30 degrees), as is seen during postoperative rehabilitation.[11]

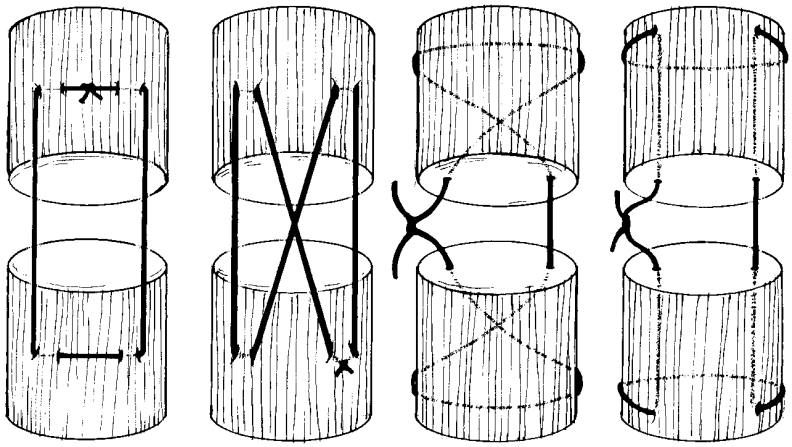


Fig. 3 Four commonly used repair techniques for extensor tendon lacerations.

Tendon Lacerations

Evaluation

A careful evaluation of the injured hand, including a thorough neurovascular examination, is essential in the treatment of extensor tendon lacerations. Flexor tendon function should be assessed, and appropriate radiographs obtained. The wound should be thoroughly inspected, with adjunctive use of local or regional block anesthesia as necessary. Injuries near a joint must be carefully inspected for violation of the capsule; sterile saline or methylene blue should be injected into the joint for verification if any doubt exists.

For lacerations in zones V through VIII, careful testing of MCP extension should be performed with the wrist held in neutral and the interphalangeal joints extended. Extension of the interphalangeal joints, produced by the intrinsics, should not be interpreted as representing extrinsic extensor integrity. The patient should be asked to fully extend each finger at the MCP joint against gentle resistance. A complete laceration of the extensor tendon will prevent full MCP extension.

A partial extensor laceration may be painful or may demonstrate incomplete MCP extension. Partial lacerations should be directly visualized to determine whether repair is necessary. Although no studies have been performed to determine the amount of partial laceration that requires repair, I believe that a tendon lacerated over 50% of its width should be repaired to ensure adequate balance and to prevent further disruption.

Injuries proximal to the juncturae tendinum in zone VI require special attention. A finger may fully extend by way of a junctura even though its extensor tendon is completely lacerated. This can be evaluated by asking the patient to extend each finger individually while holding all others flexed at the MCP joints, thereby blocking the pull of the juncturae. If the extensor tendon is intact, the patient will be capable of at least some extension, which should be comparable to that of the contralateral finger of the noninjured hand.

Treatment

Severely contaminated wounds, open fractures, and joint capsule lacerations require emergent and thorough irrigation and debridement. Fractures and skin loss should be treated in the initial procedure when feasible. Fractures should be fixed rigidly enough to allow early dynamic splinting or active motion.

For lacerations without associated injury, the extensor tendon can be repaired emergently or in a delayed primary fashion after irrigation, debridement, and loose closure of the wound. If the repair is delayed, it should be performed within 7 days, before the tendon ends retract or soften.

All repairs are best performed with adequate anesthesia, lighting, and exposure. Repair should be done with 3-0 or 4-0 nonabsorbable suture material. For repairs in zones V through VIII, the modified Kessler or Bunnell technique with 4-0 nonabsorbable suture is effective.

Injuries to the extensor retinaculum in zone VII should be repaired with suture, taking care to avoid impingement of the repair on the retinaculum. If impingement would occur, a portion of the retinaculum can be resected to allow unhindered tendon excursion.

For injuries in zones III and IV, emergent care should proceed as necessary for open joints, fractures, or contaminated wounds. A modified Kessler or Bunnell technique with 4-0 nonabsorbable suture is effective if sufficient tendon thickness is present. These techniques are significantly better ($P<0.05$) than a mattress or figure-of-eight repair (Fig. 3).[24] The lateral bands, if injured, should be repaired separately with 5-0 or 6-0 suture. Extensor lacerations in these zones occur with a high rate of associated injuries (80%) and have the poorest

outcome.[11] Some advocate the use of percutaneous pinning of the PIP joint in full extension to keep tension off the repair. I perform this only when mandated by the bone injury or when the patient is noncompliant.

The surgical techniques used for repair in zones I and II are less well defined than for the more proximal zones. Most often used are a running or mattress technique or the tenodermodesis technique of McFarlane and Hampole,[25] in which the skin and tendon are sutured as one layer. Because the DIP joint is extremely difficult to maintain in full extension by splints, pinning the joint in extension best protects the repair and allows monitoring of the skin. The PIP joint should remain free for full activity after the skin and nail bed have healed.

Rehabilitation

In recent years, research has been directed toward postoperative rehabilitation for extensor injuries in an effort to improve results. Static splinting has a long history, and the results are well documented.

For injuries in zones V through VIII, a static splint can be made of plaster or molded plastic that is fitted to the volar aspect of the hand and wrist. The fingers next to the injured digit are also included to protect against the pull of adjacent tendons through the juncturae. The wrist is placed in approximately 30 degrees of extension, the MCP joints in approximately 15 degrees of flexion, and the interphalangeal joints in full extension. Splinting is maintained for 4 to 6 weeks, after which active range of motion is begun. Passive range-of-motion exercises are begun approximately 2 weeks later.

The inherent disadvantages of immobilizing a lacerated tendon are obvious. The tendon becomes adherent to underlying periosteum and overlying skin, producing a potential loss of flexion. Evans and Thompson[11] have shown that the force of efforts to overcome adhesions can attenuate the repair and result in increasing extensor lag during the course of rehabilitation. Immobilized tendons also lose strength over time.[11,26] Controlled stress has been shown to combat this by improving tensile strength, improving gliding properties, increasing repair-site DNA, and accelerating changes in peritendinous vessel density and configuration.[26]

Dynamic extension splinting offers an alternative for minimizing adhesion problems by allowing several millimeters of extensor tendon gliding without placing undue stress across the repair site. Although the optimal amount of extensor excursion that is required to limit adhesion is not known, dynamic splinting has been useful in improving outcomes in proximal zones. The splint is applied dorsally 3 to 5 days after injury and holds the wrist in 30 degrees of extension and the MCP joints in 10 to 15 degrees of flexion (Fig. 2). The interphalangeal joints are held in 0 degrees of extension by rubber bands attached to slings. While most reports have dealt with simple injuries, this postoperative protocol can also be beneficial in the treatment of injuries that involve well-stabilized fractures or other soft-tissue damage.

Postoperative treatment is still evolving for injuries in zones III and IV. Static splinting of only the affected finger can be used. Although some would advocate splinting of the wrist and MCP joints for distal injuries, Dagum and Mahoney[19] have shown that little or no stress occurs at a zone III or IV repair site if the PIP joint and wrist are splinted and the MCP joint is left free. Evans and Thompson[11] recommend that only the interphalangeal joints be included. If a static rehabilitation protocol is chosen, an aluminum-and-foam or molded-plastic splint is positioned on the dorsal or volar aspect of the digit, depending on the soft-tissue injury and the surgeon's preference. Both the DIP and PIP joints are held in full extension. If the wrist is included, it should be splinted in 30 degrees of extension. Static splinting should be maintained for 4 to 6 weeks, after which gentle active motion is begun. Passive motion is delayed another 2 to 4 weeks.

Others have advocated dynamic extension splinting for these distal injuries.[9,10] The splint is similar to that used for more proximal injuries, but does not include the wrist and need include only the affected digit. The MCP joint is held in slight flexion, and the outrigger mechanism and sling hold the interphalangeal joints in full extension. Hung et al[9] modify this apparatus slightly to place the MCP joints in approximately 70 degrees of flexion. In theory, this position rotates the sagittal bands distally, thus decreasing tension within the distal segments of the extensor mechanism and limiting the pull of the EDC tendon on the repair site. These techniques produced improved outcomes compared with static splinting.

Another rehabilitation option for injuries in zones III and IV is early controlled active motion as advocated by Evans and Thompson,[11] who demonstrated excellent clinical results. With this method, the finger is splinted in full extension between exercise sessions. The MCP joint is not included in the splint. During exercise, performed four to six times a day, the static splint is replaced by a splint

that allows 30 degrees of flexion. Active flexion to the block and active extension to neutral are performed several times during each session. After 2 weeks, the splint is modified to allow approximately 40 degrees of flexion. Splinting is discontinued after 6 weeks, and full active range-of-motion exercises are begun. The authors have calculated that this short arc of motion produces approximately 290 g of force across the extensor repair and that limiting flexion to 30 degrees provides a safety factor. Corroboration of these calculated forces in zone IV was shown in a laboratory study in which the force across the repaired tendon produced by full flexion was approximately 400 g.[24] This compares favorably with the initial repair strength with the modified Bunnell and Kessler techniques, which was approximately 2,150 g in a cadaver study.[24]

Static splinting of the DIP joint alone after repair of lacerations in zones I and II is generally recommended for 4 to 6 weeks, after which active range-of-motion exercises can be begun. A pin transfixing the DIP joint is particularly helpful in dealing with any soft-tissue component of these injuries.

Gliding is my preferred method of postoperative rehabilitation whenever the clinical situation allows. For extensor tendon lacerations in zones V through VIII, I have used a dynamic splint with a dorsal block hood in the past. With the use of stronger suture techniques, active motion exercises have been incorporated into the rehabilitation program with promising early results. For injuries in zones III and IV, I recommend short-arc active-motion exercises. In zones I and II, I typically keep the DIP joint pinned for 6 weeks after tendon repair and allow only active motion for another 6 weeks after the pin is removed.

Closed Injuries

Closed injuries to the extensor mechanism in zones III through VIII are relatively rare, especially when compared with open injuries and mallet injuries. Closed injuries in zones VI through VIII are almost nonexistent except when associated with a systemic disorder, such as rheumatoid arthritis.

While the central tendon is seldom injured in closed injuries to the extensor mechanism in zone V, the sagittal bands that hold the EDC tendon centrally over the metacarpal head can rupture. This can occur from a direct blow to the dorsal aspect of the metacarpal head or from forced extension or flexion of the MCP joint. Swelling and ecchymosis are minimal. The patient usually notes modest pain and is unable to initiate MCP extension from a flexed position, but will be able to maintain extension after the finger has been passively extended.

The differential diagnosis should include extensor tendon rupture, tendon laceration, and radial nerve dysfunction. The long finger is most often affected, because its radial sagittal fibers are weaker and its joint is the most exposed to blunt trauma; however, any finger may be affected. The extensor tendon usually dislocates ulnarly into the intermetacarpal valley because of the ulnar pull of the flexor and extensor tendons, but ulnar sagittal fiber rupture with radial dislocation has also been described.[27]

If the injury is detected immediately, a static splint or short-arm cast can be applied with the MCP joints of the affected finger and each adjacent finger held in 0 degrees of flexion. The PIP joints are left free, and range of motion is encouraged to promote gliding of the lateral bands. Incomplete injuries can be treated with static splinting or with buddy-taping. Rayan and Murray[28] recommend conservative splinting after sagittal band injury for up to 6 weeks after injury, even with complete dislocation of the extensor tendon. However, because the balance of the extensor mechanism over the MCP joint is delicate, open repair of complete injuries is generally recommended to ensure that the ruptured sagittal fibers are appropriately reapproximated and the tendon is well centered over the MCP joint. Repair can usually be accomplished by simple reapproximation of the sagittal fibers with 4-0 or 5-0 absorbable suture in a mattress fashion. If some delay has occurred or the extensor tendon has a tendency to subluxate after reapproximation of the sagittal fibers, the opposite (usually ulnar) sagittal fibers should be partially released to allow accurate centralization of the tendon.

Closed ruptures of the central slip overlying the PIP joint can occur with volar dislocation of the PIP joint, with forced flexion of the PIP joint, or with a severe contusion to the dorsum of the PIP. The joint dislocation itself is generally recognized from a careful history and radiographs. A central slip rupture may not be recognized in the immediate postinjury period because of the pain and swelling associated with the dislocation.[17] Deformity may take 2 to 3 weeks to occur as the untethered lateral bands slide volarly, producing a typical boutonniere posture. Because patients frequently present with a presumed dislocation that they themselves have reduced, a high index of suspicion should be maintained to identify a concomi-

tant injury to the central slip when there is dorsal tenderness and swelling about the PIP joint. Early simple treatment can prevent late disabling deformity.

Detection of a central slip rupture is relatively straightforward. After the PIP joint has been relocated, extension of the joint is carefully evaluated. Extension lag, pain with extension, or pain with resisted extension should raise the suspicion of a central slip rupture.[17] Full extension may still be possible if the lateral bands have not yet moved volarly, but it will be painful and weak. If the diagnosis remains unclear, a dilute solution of arthrographic dye can be injected into the joint. Extrusion of dye dorsally into the soft tissues indicates a central slip rupture along with rupture of the dorsal capsule.

Central slip rupture is treated with immobilization of the PIP joint in 0 degrees of extension with a dorsal or volar splint for 6 weeks; this encourages DIP joint motion to keep the lateral bands mobile and gliding. These injuries can be treated successfully up to 6 weeks after rupture, although serial casting or dynamic splinting may be necessary to regain full passive extension before continuous splinting in full extension. If the PIP joint cannot be passively brought into 0 degrees of extension initially or after serial splinting, capsular and volar plate release may be necessary before performing a difficult extensor tendon reconstruction.

Closed rupture of the extensor mechanism is quite common in zone I. The well-known mallet finger is usually evident immediately after injury, with an obvious droop of the finger at the DIP joint and a lack of active extension. The mechanism of injury is usually forced flexion of the fingertip, often from the impact of a thrown ball. Pain, swelling, ecchymosis, and finger droop are the usual presenting signs and symptoms. Radiographs are used to assess the presence of a fracture and the degree of joint subluxation.

Doyle[29] has described four types of mallet injury. Type I is a typical extensor tendon avulsion from the distal phalanx. Type II is a laceration of the tendon. Type III is a deep avulsion that injures tendon and skin. Type IV is a fracture of the distal phalanx; this type is further divided into three categories. Type IVA is a transepiphyseal fracture in a child. Type IVB involves less than half the articular surface of the joint without joint subluxation; the mechanism of injury is usually hyperflexion. Type IVC involves more than one half the articular surface of the joint and can involve volar subluxation of the joint; this injury may be caused by hyperextension of the joint.

Type I mallet finger is treated by splinting the DIP joint, holding it in 0 degrees of extension with a dorsal or volar splint. Care must be taken not to hyperextend the joint as this can compromise the blood supply to the dorsal skin. I prefer to use a dorsally applied aluminum-and-foam splint, as this leaves the patient's touch pad free. Half the thickness of the foam is removed so that the digit is not pulled into hyperextension. Splinting is continuous for 6 weeks. If there is active extension and little or no droop at reevaluation, the splint is applied at bedtime only for another 6 weeks.

Type II injuries should be repaired as discussed previously. Type III injuries may require skin grafting and other reconstruction. Type IVA fractures should be reduced if necessary and splinted for 6 weeks. Type IVB fractures should also be reduced if necessary and splinted.

Treatment of type IVC injuries is still controversial. Many are reducible and can be treated conservatively. Wehbé and Schneider[30] have shown that these injuries, even if associated with volar subluxation, can be treated with splinting alone, with results at least comparable to those for injuries treated with open reduction and internal fixation. The difficulty of approaching this area surgically is evident, as the skin has a tenuous blood supply, and the fracture fragment is quite small. Anatomic restoration is often difficult, and complications are frequent.[30]

For type IVC injuries in which the volar subluxation of the joint is unacceptable after attempted reduction and appropriate splinting, a more conservative option is to reduce the joint and pin it percutaneously without direct exposure of the fracture. A Kirschner wire in the distal fragment can serve as a joystick to maneuver the fragment into position. The wire is then advanced through the proximal fragment (if it is large enough) and across the DIP joint. This allows near-anatomic restoration of joint and fracture and avoids the potential complications of internal fixation. Nevertheless, complications, such as articular incongruence, wire breakage, and infection, can still occur.[31]

Summary

While extensor injuries often result in loss of extension, this is seldom disabling. However, these injuries frequently result in loss of flexion, which is greater in severity and frequency than loss of extension. As a consequence, extensor tendon injuries deserve careful attention in diagnosis, treatment, and rehabilitation to offset potential loss of function.

References

1. Dargan EL: Management of extensor tendon injuries of the hand. *Surg Gynecol Obstet* 1969;128:1269-1273.
2. Flynn JE: Problems with trauma to the hand. *J Bone Joint Surg Am* 1953;35: 132-140.
3. Hauge MF: The results of tendon suture of the hand: A review of 500 patients. *Acta Orthop Scand* 1954;24: 258-270.
4. Kelly AP Jr: Primary tendon repairs: A study of 789 consecutive tendon severances. *J Bone Joint Surg Am* 1959; 41:581-598.
5. Newport ML, Blair WF, Steyers CM Jr: Long-term results of extensor tendon repair. *J Hand Surg [Am]* 1990;15:961-966.
6. Browne EZ Jr, Ribik CA: Early dynamic splinting for extensor tendon injuries. *J Hand Surg [Am]* 1989;14:72-76.
7. Chow JA, Dovelle S, Thomes LJ, et al: A comparison of results of extensor tendon repair followed by early controlled mobilisation versus static immobilisation. *J Hand Surg [Br]* 1989; 14:18-20.
8. Evans RB, Burkhalter WE: A study of the dynamic anatomy of extensor tendons and implications for treatment. *J Hand Surg [Am]* 1986;11:774-779.
9. Hung LK, Chan A, Chang J, et al: Early controlled active mobilization with dynamic splintage for treatment of extensor tendon injuries. *J Hand Surg [Am]* 1990;15:251-257.
10. Saldana MJ, Choban S, Westerbeck P, et al: Results of acute zone III extensor tendon injuries treated with dynamic extension splinting. *J Hand Surg [Am]* 1991;16:1145-1150.
11. Evans RB, Thompson DE: An analysis of factors that support early active short arc motion of the repaired central slip. *J Hand Ther* 1992;5:187-201.
12. Newport ML, Waters SN: A new suture technique for extensor tendon repair. Presented at the 50th Annual Meeting of the American Society for Surgery of the Hand, San Francisco, 1995.
13. Kleinert HE, Verdan C: Report of the Committee on Tendon Injuries. *J Hand Surg [Am]* 1983;8(5 pt 2):794-798.
14. Manske PR, Lesker PA: Nutrient pathways to extensor tendons within the extensor retinacular compartments: An experimental study in dogs. *Clin Orthop* 1983;181:234-237.
15. Smith JW, Bellinger CG: The blood supply of tendons, in Tubiana R (ed): *The Hand.* Philadelphia: WB Saunders, 1991, vol 1, pp 353-358.
16. Miller H: Repair of severed tendons of the hand and wrist: Statistical analysis of 300 cases. *Surg Gynecol Obstet* 1942; 75:693-698.
17. Lovett WL, McCalla MA: Management and rehabilitation of extensor tendon injuries. *Orthop Clin North Am* 1983;14:811-826.
18. Minamikawa Y, Peimer CA, Yamaguchi T, et al: Wrist position and extensor tendon amplitude following repair. *J Hand Surg [Am]* 1992;17:268-271.
19. Dagum AB, Mahoney JL: Effect of wrist position on extensor mechanism after disruption separation. *J Hand Surg [Am]* 1994;19:584-589.
20. DeVoll JR, Saldana MJ: Excursion of finger extensor tendon elements in zone III. *Orthop Trans* 1989;13:220.
21. Duran RJ, Houser RG, Coleman CR, et al: Management of flexor tendon lacerations in zone 2 using controlled passive motion postoperatively, in Hunter JM, Schneider LH, Mackin EJ, et al (eds): *Rehabilitation of the Hand,* 2nd ed. St Louis: CV Mosby, 1984, pp 273-276.
22. Newport ML, Shukla A: Electrophysiologic basis of dynamic extensor splinting. *J Hand Surg [Am]* 1992;17: 272-277.
23. Newport ML, Williams CD: Biomechanical characteristics of extensor tendon suture techniques. *J Hand Surg [Am]* 1992;17:1117-1123.
24. Newport ML, Pollack GR, Williams CD: Biomechanical characteristics of suture techniques in extensor zone IV. *J Hand Surg [Am]* 1995;20:650-656.
25. McFarlane RM, Hampole MK: Treatment of extensor tendon injuries of the hand. *Can J Surg* 1973;16:366-375.
26. Feehan LM, Beauchene JG: Early tensile properties of healing chicken flexor tendons: Early controlled passive motion versus postoperative immobilization. *J Hand Surg [Am]* 1990;15: 63-68.
27. Kettelkamp DB, Flatt AE, Moulds R: Traumatic dislocation of the long-finger extensor tendon: A clinical, anatomical, and biomechanical study. *J Bone Joint Surg Am* 1971;53:229-240.
28. Rayan GM, Murray D: Classification and treatment of closed sagittal band injuries. *J Hand Surg [Am]* 1994;19:590-594.
29. Doyle JR: Extensor tendons: Acute injuries, in Greene DP, Hotchkiss RN (eds): *Operative Hand Surgery,* 3rd ed. New York: Churchill Livingston, 1993, vol 2, pp 1925-1954.
30. Wehbé MA, Schneider LH: Mallet fractures. *J Bone Joint Surg Am* 1984;6: 658-669.