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Complications After Treatment of Flexor Tendon Injuries

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Abstract
The goals of flexor tendon repair are to promote intrinsic tendon healing and minimize extrinsic scarring in order to optimize tendon gliding and range of motion. Despite advances in the materials and methods used in surgical repair and postoperative rehabilitation, complications following flexor tendon injuries continue to occur, even in patients treated by experienced surgeons and therapists. The most common complication is adhesion formation, which limits active range of motion. Other complications include joint contracture, tendon rupture, triggering, and pulley failure with tendon bowstringing. Less common problems include quadriga, swan-neck deformity, and lumbrical plus deformity. Meticulous surgical technique and early postoperative tendon mobilization in a well-supervised therapy program can minimize the frequency and severity of these complications. Prompt recognition of problems and treatment with hand therapy, splinting, and/or surgery may help minimize recovery time and improve function. In the future, the use of novel biologic modulators of healing may nearly eliminate complications associated with flexor tendon injuries.

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in specific areas between the joints; these thickened areas are called pulleys. The pulleys enhance efficiency of motion within the digit by preventing tendon bowstringing and maximizing tendon excursion. Most critical to this system are the A2 and A4 pulleys, which are located over the proximal and middle phalanges, respectively. Figure 1. The FDP and FDS tendons are contained within the digital flexor sheath.

**Flexor Tendon Healing**

Tendon healing consists of three phases: inflammatory, proliferative, and remodeling. The inflammatory phase occurs during the first week after injury and involves migration of fibroblasts and macrophages to the injured area, with ensuing phagocytosis of the clot and necrotic tissue. In the proliferative phase, which lasts from weeks 1 through 3, fibroblasts proliferate, and there is immature collagen deposition and neovascularization. Finally, the remodeling phase occurs in weeks 3 through 8. Collagen fibers become organized in a linear manner parallel to the tendon. Adhesion formation between tendon and sheath is most clinically evident during this last phase.

Two mechanisms for healing have been described in the literature: extrinsic and intrinsic. The extrinsic mechanism is predominately mediated by an influx of synovial fibroblasts and inflammatory cells from the tendon sheath. Healing also occurs via the intrinsic mechanism, in which fibroblasts and inflammatory cells from the tendon and epitenon invade the injured site. The extrinsic mechanism is thought to predominate early in tendon healing and in cases of digit immobilization; the intrinsic mechanism becomes increasingly active after 21 days. The greater proliferative and inflammatory response of the synovial sheath, along with the greater cytokine reactivity and capacity for matrix degradation of synovial fibroblasts, favor the extrinsic pathway.

Extrinsic healing produces increased collagen content at the injury site, but in a disorganized fashion. Tendon healing is likely a combination of both mechanisms, but the predominance of extrinsic healing leads to scar formation and adhesions between the tendon and the surrounding sheath.

**Requirements for Tendon Healing**

Requirements for tendon healing include motion and tension at the repair site, adequate tendon nutrition and vascular perfusion, minimal gap formation at the repair site, and a strong repair. Early-motion protocols in animal flexor tendons resulted in a progressively greater ultimate tensile load over time than was the case in tendons managed with immobilization protocols. Early-motion protocols also helped avoid the loss of strength that occurs in early phases of tendon healing. Additionally, both motion and tension are needed to stimulate tenocyte development and increase collagen amount and organization.

Tendon nutrition is provided through vascular perfusion and synovial fluid diffusion. Flexor tendon vascular supply originates from vessels in the proximal synovial fold, segmental branches of digital arteries through the vincular system, and the osseous insertion of the FDS and FDP tendons. Diffusion of nutrients through synovial fluid occurs via imbibition, in which fluid is forced through interstices on the surface of the tendon. This process is facilitated by the pumping mechanism created by flexion and extension of the digit.

Gap formation as a result of cyclic loading before tendon failure is seen routinely after flexor tendon repair. The average gap is 3.2 mm. Gaps have previously been associated with adhesion formation and poor gliding. Gelberman et al, however, demonstrated that gap length has no relationship to adhesion formation, but it does have a negative effect on the acquisition of tendon tensile properties during healing. In their canine study, repair gaps >3 mm did not gain stiffness or strength from 10 to 42 days, but gaps <3 mm had a 320% increase in stiffness and a 90% increase in strength over the same period.

Techniques for maximizing tendon repair strength comprise a large portion of flexor tendon research. A strong repair is one that can withstand early motion with minimal gap formation, thereby allowing successful tendon healing. Well-accepted, established principles of tendon repair include using core sutures of 3-0 or 4-0 nonabsorbable polyfilament material, an increased

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number of sutures crossing the repair, and equal strength across all strands. In addition, certain locking suture techniques (ie, transverse limb of repair passed superficial to the longitudinal component) have been shown to increase repair strength.18–20 A peripheral locking epitendinous suture also should be added to enhance repair strength.21

Complications

Adhesion Formation

Adhesion formation is the most common complication following flexor tendon repair. Prevention of adhesion formation is facilitated by optimizing intrinsic healing. Early research reflected the belief that tendon healing depended on extrinsic cellular ingrowth, which required immobilization. However, the ability of tendons to heal by intrinsic mechanisms alone has since been well documented.22 Methods of adhesion prevention can be divided into mechanical and biologic factors designed to promote intrinsic healing.

Mechanical Factors

Mechanical factors for preventing adhesions include early postoperative motion protocols, preservation of sheath and pulley components, partial FDS resection, and atraumatic handling of the tendon and sheath. Motion, which leads to a predominance of intrinsic over extrinsic healing, is critical to preventing adhesions. Three primary motion protocols are described in the literature: passive, active, and synergistic. In 1977, Lister et al22 published the first results of tendon repair using a controlled passive motion protocol. The Kleinert splint was used to allow active digital extension coupled with passive digital flexion. Good to excellent results were reported in 80% of tendon lacerations in zone II.23 The splint has since been modified by adding a midpalmar bar or pulley, resulting in improved distal tendon gliding and differential tendon excursion.24 The addition of synergistic wrist motion (wrist flexion–finger extension combined with wrist extension–finger flexion) also has been shown to improve overall tendon gliding and excursion.25

Early active motion protocols subsequently have been developed to address concerns about variability in tendon gliding with passive protocols. Bainbridge et al26 reported on a consecutive series comparing controlled active motion with active extension–passive flexion protocols. Patients treated with controlled active motion acquired greater final motion.26 Other series using early active motion have reported good to excellent results ranging from 57% to 92%, with rupture rates from 5% to 46%.27–29 These findings are comparable to rates reported with passive motion regimens. Improved suture materials and techniques seem capable of Withstanding the higher forces associated with active motion protocols.30–32 However, recent research in repaired canine tendon by Boyer et al33 demonstrated no advantage with high-force rehabilitation in the accrual of either stiffness or strength compared with low-force rehabilitation.

The synergistic motion regimen allows high tendon excursion with low force on the repair site.34 This protocol consists of passive digit flexion combined with active wrist extension, followed by active wrist flexion combined with passive digit extension. Zhao et al35 compared synergistic motion with passive motion regimens in the management of canine flexor tendon repairs. They noted fewer adhesions with the synergistic motion group but reported elevated gap formation in the motion group (30%) versus the passive group (6%).35 Currently, agreement is universal that repaired flexor tendons should be subjected to early mobilization; however, no single rehabilitation protocol is accepted by all.

Preservation of sheath components is controversial. When the vascular source of nutrition is compromised because of trauma, the tendon sheath can maintain nutrition through imbibition until the vascular system is reestablished.36 Preservation of flexor tendon sheath integrity may reduce adhesions through its positive effect on intrinsic healing.37 However, sheath repair also may lead to impaired tendon gliding and increased resistance.17 Another study compared sheath repair with excision and found no difference in final motion when early mobilization was done.38

Recently, resection of all or part of the FDS tendon has been suggested as a method of decreasing gliding resistance of the FDP within the sheath.39 Loss of the FDS tendon is not associated with significant functional compromise. However, this technique was initially dismissed because a considerable portion of the FDP blood supply is provided by capillaries emanating from the FDS tendon. In a cadaveric study, FDS resection was found to be a viable option for improving the gliding of a bulky FDP Repair. The authors did not demonstrate any advantage of complete resection versus partial resection.39

The use of meticulous surgical technique as a method for decreasing adhesion formation is well documented. Adhesion formation is known to be proportional to the amount of tissue crushing and to the number of surface injuries incurred by the tendon and sheath during repair.4 Accordingly, stiffness is more common in digits after crush injuries as well as in those with concomitant neurovascular and bone injuries.40

Biologic Factors

Development of novel biologic factors to provide so-called scarless healing is an active area of research.25,41 Advances in this area could lead to less reliance on postoperative motion for adhesion prevention. Methods currently under investigation include mechanical barriers to adhesion formation, as well as
chemical and molecular modulation of scar formation. Many mechanical barrier methods have been studied, including silicone, alumina sheaths, polyethylene, and polytetrafluoroethylene, but none is in widespread clinical use.22 ADCON-T/N (Gliatech, Cleveland, OH), a gelatin and carbohydrate polymer, has shown some potential.41 In a recent double-blind randomized study in which ADCON-T/N was applied to the tendon after repair, the authors found no significant effect on final motion; however, time to achieve final motion was shorter with the use of ADCON-T/N.41

Ibuprofen and corticosteroids have been investigated as possible modulators of adhesion formation.12,42,43 Ibuprofen has been shown to improve tendon excursion in animal models.42 Ketchum43 demonstrated that although corticosteroids decrease the strength and density of adhesions, they are associated with smaller, weaker tendons, diminished wound healing, and decreased resistance to infection. These problems have limited their use in flexor tendon repair.

New Research

Modulation of scar formation on a molecular level is a new area of research in tendon healing. This research has been directed toward understanding the role of cytokines in tendon metabolism and repair.23,44,45 Two cytokines, transforming growth factor-β (TGF-β) and basic fibroblast growth factor (bFGF), have shown the most potential in adhesion prevention.44,45 TGF-β has been implicated in numerous biologic activities related to wound healing, such as fibroblast and macrophage recruitment, angiogenesis, stimulation of collagen production, downregulation of proteinase activity, and increased metalloproteinase inhibitor activity.44

Chang et al45 demonstrated that flexor tendons exposed to transection and repair exhibit increased TGF-β in both tenocytes and inflammatory cells from the tendon sheath. These findings are significant because TGF-β is thought to be involved in the pathogenesis of excessive scar formation. Therefore, perioperative modulation of this cytokine may lead to decreased adhesion formation. Three isoforms have been identified; the TGF-β1 isoform is thought to be primarily responsible for the proinflammatory and scarring activities.22 The TGF-β3 isoform demonstrates anti-scarring properties and acts as an inhibitor of scarring in injury models.52

Similar to TGF-β, bFGF has been implicated in early tendon healing.45 Basic FGF is a potent stimulator of angiogenesis and is able to induce migration and proliferation of endothelial cells in tissue culture. In 1998, Chang et al45 found that bFGF was upregulated in tenocytes, tendon sheath fibroblasts, and inflammatory cells from flexor tendons exposed to a tendon wound environment. With further research, modification of bFGF expression may also be useful in postoperative adhesion reduction.

Research into chemical modulation of cytokines has yielded 5-fluorouracil (5-FU) as a possible candidate.46,47 5-FU is an antimitabolite that decreases scarring by an unknown mechanism. Khan et al46 tested this drug in a rabbit model by treating the injured synovial sheath of partially lacerated tendons with a 5-min application of 5-FU before closure. A significant (P < 0.001) decrease in the proliferative and inflammatory response of synovial fibroblasts was demonstrated. There was also a significant (P < 0.001) decrease in the expression of TGF-β in the treated tissue. Others have reported the ability of 5-FU to reduce postoperative adhesions in a chicken model.47 These findings are still experimental, however, and have not yet been implemented in clinical practice.

When adhesion prevention is unsuccessful, early recognition is critical to ensure a good clinical outcome and prevent further progression of stiffness. Adhesion and tendon rupture present clinically with similar physical findings. Both conditions may demonstrate loss of active flexion, but patients with adhesions have preservation of some residual active motion. Imaging studies, such as magnetic resonance imaging or ultrasound, may be indicated to determine the source of motion loss. Magnetic resonance imaging has been shown to be 100% accurate in distinguishing adhesions from rupture.48

When adhesions are identified, therapy should be directed toward programs that maximize differential motion between the FDS and FDP tendons.25,26 Splinting also may be a useful adjunct. When therapy and splinting fail to produce effective results, tenolysis may be indicated.

Tenolysis

Flexor tenolysis is indicated when active range of motion (ROM) measurements do not improve within several weeks to months, despite strict patient compliance with splinting and ROM exercises.49 Tenolysis should not be considered until the soft tissues have reached a state of equilibrium, with supple skin and subcutaneous tissues. To achieve a good result, the digit must have minimal joint contractures and near-normal passive ROM.17 Most surgeons recommend waiting for 3 to 6 months after tendon repair or grafting before performing tenolysis.49,50

When performing flexor tenolysis, a local anesthetic combined with intravenous sedation is recommended to allow the patient to perform active flexion in the operating room.50 This intraoperative testing is critical to achieve a successful outcome. A midlateral or Bruner zigzag incision is used to expose the length of the tendon. The neurovascular bundles are encountered at the ends of the digital creases, and the surgeon must take care to prevent iatrogenic injury to these structures. The scarred tendon and its sheath are visualized (Figure 2, A),51 the adhe-
Flexor tenolysis is performed by identifying the scarred tendon and sheath (A), followed by release of adhesions and careful preservation of the pulley system (B). Release may be facilitated by passing a small elevator or dental probe through windows in less critical portions of the sheath (eg, proximal to A2, or between A2 and A4 pulleys). (Reprinted from Strickland JW: Flexor tenolysis, in Strickland JW [ed]: Master Techniques in Orthopaedic Surgery: The Hand. Philadelphia, PA: Lippincott-Raven, 1998, pp 525-538. Illustrations copyright © Gary Schnitz and the Indiana Hand Center.)

Flexor tenolysis is performed by identifying the scarred tendon and sheath (A), followed by release of adhesions and careful preservation of the pulley system (B). Release may be facilitated by passing a small elevator or dental probe through windows in less critical portions of the sheath (eg, proximal to A2, or between A2 and A4 pulleys). As much of the pulley system as possible must be preserved (Figure 2, B); when this is not feasible, pulley reconstruction or a staged tendon implant should be considered. If pulley reconstruction requires protected mobilization, however, the end result may be compromised. Additionally, any concomitant procedure, such as tendon shortening, skin grafting, osteotomy, or capsulotomy, may have an adverse effect on the outcome of flexor tenolysis. At the end of the procedure, the patient should be placed in a splint that permits immediate active ROM. Patients for whom active ROM improves in the first few weeks after surgery tend to maintain these gains. Significant pain and little early improvement in motion may be an indication for inserting an indwelling polyethylene catheter containing local anesthetic.

One complication of flexor tenolysis is tendon or pulley rupture, which should be managed with a staged tendon reconstruction. Other complications include postoperative edema and pain as well as inadvertent neurovascular injury that may lead to loss of viability in a digit with marginal preoperative circulation. Flexor tenolysis is a technically demanding procedure, and the postoperative rehabilitation is equally arduous. Not all patients are candidates for tenolysis. The surgeon must evaluate how the loss of active motion will affect the patient's needs and desires as well as the ability to perform activities of daily living and to return to his or her occupation. The surgeon also must consider the sensory and circulatory status of the finger, the condition of the other digits, and the age and general health of the patient. Patients who are noncompliant with therapy after their initial repair typically are poor candidates for tenolysis.

Joint Contracture

Even with adherence to early-motion regimens, the reported rate of proximal interphalangeal (PIP) and distal interphalangeal (DIP) joint contracture is 17%. Contractures may be caused by unrecognized disruption or scarring of the volar plate, tendon bowstringing secondary to pulley incompetence, concomitant fracture or neurovascular injury, prolonged healing in a flexed position, collateral lig-
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Figure 3

Splints used to manage proximal interphalangeal (PIP) flexion contractures. A, Dorsal forearm-based thermoplast splint with a felt block placed dorsally at the level of the PIP joint. B, Joint Jack Finger Splint (Sammons Preston Rolyan, Bolingbrook, IL). C, Safety Pin Splint (Sammons Preston Rolyan).

ament contracture, skin contracture, or flexor tendon adhesions. They also may be secondary to inadequate postoperative motion regimens and dynamic flexion splinting. The latter may be prevented through correct positioning of the wrist, hand, and digits in the postoperative splint and early motion. Most postoperative protocols involve splinting the metacarpophalangeal (MCP) joint in flexion (approximately 60°) with the interphalangeal (IP) joints fully extended.

Nonsurgical management of joint contractures consists of early identification and modification of splinting to allow greater PIP and DIP joint extension. A felt or foam block placed inside a dorsal splint at the level of the proximal phalanx, in addition to increasing MCP joint flexion to relax the intrinsic mechanism, will help resolve PIP joint contracture (Figure 3, A). This method can be used with buddy taping and active-assisted extension exercises. Static nighttime extension splinting and passive extension exercises with Velcro bands applied to the splint to impart an extension force on the digit also may be useful. As the tendon continues to heal and strengthen, finger splints (eg, Joint Jack, Safety Pin) can be used (Figure 3, B and C).

When nonsurgical management of contractures is unsuccessful, surgery should be considered. No absolute guidelines exist regarding the degree of contracture that requires surgical release; rather, the decision for surgery is based on the patient’s functional limitations and goals. Preoperatively, the surgeon should attempt to determine whether the contracture is caused by extrinsic factors (eg, skin contracture, proximal flexor tendon adhesions) or an intrinsic joint contracture. When extrinsic factors are responsible, PIP joint extension will improve with MCP joint flexion. PIP joint release should be performed only after all flexor tendon adhesions and skin contractures have been addressed.

For PIP joint release, exposure is performed through a Bruner or midlateral incision. The radial and ulnar neurovascular bundles are identified and protected. The C1 portion of the flexor sheath is excised between the A2 and A3 pulleys, and the FDP and FDS tendons are exposed (Figure 4, A). Flexor tenolysis is performed initially; the checkrein ligaments are identified by passing a small hemostat or elevator volar to the transverse retinacular vessels as they enter the flexor sheath just proximal to the collateral ligament origin. The checkrein ligaments are volar to the transverse retinacular vessels and can be divided sharply at this level. The transverse retinacular vessels should be preserved whenever possible because they supply the tendon vincular system.

When full passive PIP joint extension cannot be obtained, release of the collateral ligaments is performed at their insertion on the head of the proximal phalanx, beginning with the accessory collateral ligaments (Figure 4, B). Release of the collateral ligaments should be performed sequentially, progressing from palmar to dorsal, until full extension is achieved. When full extension cannot be achieved, release of the volar plate may be necessary.

Tendon Rupture

Rupture of a tendon repair is not an uncommon problem. In one study, a rupture rate of 4% was reported in 728 digital flexor tendon repairs [440 patients]. The authors were unable to identify the inciting factor in these failures. Another series reported a 5.7% rate of rupture in digital flexor tendon repairs. Factors that predispose tendon repairs to rupture include inadequate suture material, poor surgical technique, overly aggressive therapy, or early termination of postoperative splinting. Patient noncompliance, such as removing the splint, lifting heavy objects, or attempting strong grasp, is a frequent cause of rupture.

Tendon repairs are weakest between postoperative days 6 and 18. Although rupture is most common during this period, it may be
seen as late as 6 to 7 weeks after surgery. Timely surgical exploration is indicated once tendon rupture is identified. When repair attenuation is seen without obvious rupture and <1 cm of scar is present, the scar can be resected and the primary repair revised. When the scar is >1 cm, a tendon grafting procedure should be considered because excessive distal advancement of the tendon can lead to contractures and quadriga. With complete tendon rupture, the time from the original repair influences the course of action. If the rupture occurs in the early postoperative period, the tendon may be primarily repaired. When the rupture occurs 4 to 6 weeks after the original repair, tendon grafting or a staged reconstruction is recommended. Staged grafting is preferred when there is significant scarring within the sheath. Pediatric urethral or vascular dilators can be used to expand a constricted but otherwise intact sheath, thereby eliminating the need for a two-stage reconstruction.

**Triggering**

Triggering can occur after tendon repair and is usually the result of the repair site’s catching on a pulley or sheath. Causes of triggering include a bulbous tendon repair or a tightly repaired area of the tendon sheath. The surgeon should intraoperatively assess tendon gliding to identify areas that may cause triggering or restrict gliding. In the acute setting, a partial tendon sheath excision or release may be used. In contrast, sheath repair may reduce triggering of a bulky repair by acting as a funnel. Postoperatively, ultrasound or massage may be helpful. Once the tendon is healed, a corticosteroid injection may be indicated. Reduction tenoplasty may be considered when nonsurgical measures fail; however, this technique carries a risk of tendon rupture.

Recent studies have addressed the feasibility of partial sheath resection to decrease triggering and gliding resistance. This problem is of particular concern when it involves the A2 or A4 pulleys. Tang et al found a decrease in gliding resistance with partial pulley release. However, a cadaveric study by Misationis et al demonstrated that, although excision of up to 25% of both the A2 and A4 pulleys had no significant effect on the efficiency of motion, it did not achieve the goal of decreasing sheath resistance.

**Partial Tendon Injury**

Partial tendon lacerations can be challenging, if not managed properly, they carry the risk of triggering, entrapment, or secondary rupture. Repair has been recommended for lacerations involving >60% of the tendon substance. In other studies, the authors reported that trimming digital flexor tendon lacerations involving >50% of the tendon substance was not associated with trig-

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Figure 4

gering or rupture.\textsuperscript{59} In a study by Erhard et al\textsuperscript{60} that compared trimming with repair of partial lacerations, the lowest gliding resistance was produced with trimming, without a concomitant decrease in tendon strength.

**Pulley Failure and Bowstringing**

The A2 and A4 pulleys are responsible for preserving digital motion and finger strength (grip and pinch power). Loss of the integrity of these pulleys results in bowstringing, with loss of the A4 pulley causing the greatest change in the efficiency of tendon excursion, work, and force.\textsuperscript{61} Avoidance of bowstringing is the best management strategy and may be facilitated by performing tendon repair through cruciate pulley windows, using external pulley rings for compromised pulleys, and reconstructing pulleys in a one- or two-stage procedure when native tissue is unsalvageable\textsuperscript{62} (Figure 5).

Many techniques for pulley reconstruction have been described, such as Bunnell, Kleinert, Lister, and Karev. Nishida et al\textsuperscript{63} found that Lister’s technique of using the extensor retinaculum for pulley reconstruction had the least resistance to tendon gliding.

**Quadriga**

Quadriga is the inability of unjured fingers of the same hand to obtain full flexion. It manifests as a weak grasp on physical examination. This complication is caused by functional shortening of the FDP tendon. Shortening of one FDP tendon affects the function of the FDP tendons of adjacent fingers, causing overadvancement of the FDP tendon, proximal tendon tethering or adhesions, and insertion of a short tendon graft. Anatomically, quadriga occurs because the common FDP muscle belly to the middle, ring, and small fingers permits only as much proximal excursion in each digit as that of the shortest tendon. Proper tendon tensioning during repair prevents this problem. When quadriga occurs, tenolysis of the proximal adhesions or transection of the shortened tendon will release the uninjured profundus.\textsuperscript{7}

**Swan-neck Deformity**

Swan-neck deformity consists of hyperextension at the PIP joint with flexion at the DIP joint. In flexor tendon repair, common causes include isolated FDS rupture and volar plate injury. This complication is infrequent, however; loss of the FDS is usually associated with minimal functional deficit. Careful attention to and correction of volar plate injuries at the time of tendon repair prevents this problem. Surgical management of the hyperextension deformity may be facilitated through tenodesis with one slip of the FDS tendon.

**Lumbrical Plus Deformity**

Lumbrical plus deformity is the paradoxical extension at the IP joints of the injured digit with attempted forceful flexion. Normally, PIP and DIP joint flexion occurs in conjunction with simultaneous relaxation of the lumbrical muscle (Figure 6, A). Paradoxical extension arises when the FDP distal to the lumbrical muscle is functionally too long or is not present. Flexor tendon force is thereby transmitted to the lumbrical and subsequently to the extensor mechanism via the lateral bands before full digital flexion is reached (Figure 6, B). Other causes of lumbrical plus deformity include avulsion of the
FDP tendon or amputation through the proximal phalanx. Management involves lumbrical muscle release or placement of a tendon graft of appropriate length.

Summary

Despite advances in flexor tendon surgery over the past 50 years, complications continue to occur. The most common are adhesion formation and joint contracture. Achieving optimal outcomes occurs through meticulous surgical repair using 3.0 or 4.0 polyfilament core suture with a minimum of four strands reinforced with an epitenonous suture, a well-fitting splint, early controlled mobilization, and vigilant patient monitoring for compliance with the rehabilitation program. Biochemical and molecular advances in the research into scarless healing likely will lead to future advances.

References

Evidence-based Medicine: Level I/II prospective studies include references 16, 26, 27, 29, 30, 40, and 41. The remaining references are case-controlled reports or experimental observations.

Citation numbers printed in bold type indicate references published within the past 5 years.

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