The return of satisfactory digital motion after severance of the flexor tendons has long challenged surgeons dealing with upper extremity trauma. Flexor lacerations in the finger were once found to perform so poorly after primary repair that the digital sheath was referred to as a surgical no-man’s-land. Surgeons were advised not to repair tendon injuries in this zone and to resort to free-tendon grafting at a later date to achieve the best results. While that advice has now been discredited and primary flexor tendon repair has been the accepted procedure for over two decades, the techniques for repair and the postoperative management programs have varied greatly from surgeon to surgeon. Most clinical approaches have been based to a large extent on individual experience with little or no scientific support. Considerable research has been conducted in recent years in an effort to better understand the structure of tendons, their function, the biomechanics of their action at the joints they move, and their biologic response to injury and repair. These investigative efforts have given rise to improved methods of tendon repair, a greater emphasis on flexor sheath preservation and restoration, and an emphasis on the early application of stress as a means of more rapidly increasing the strength and gliding capacity of repaired tendons. This article will review some of the most recent and relevant research and discuss the current clinical approaches to flexor tendon repair that have resulted from these efforts.

**Basic Science of Flexor Tendons**

**Structure and Anatomy**

Tendons are composed of fascicles of long, narrow, spiraling bundles of mature fibroblasts (tenocytes) and type I collagen fibers. The fascicle surface usually has a layer of uniform collagen fibers, and elastin and individual fascicles are capable of sliding past each other with no apparent direct attachments or cellular communications. The surface of the individual bundles of collagen is covered by the endotenon and a fine fibrous and cellular outer layer, the epitenon, which is continuous with the endotenon. In the hand, flexor tendon fascicles are covered by a thin visceral and parietal adventitia called the paratenon, which contains fluid similar to synovial fluid. In the digits, the flexor tendons are enclosed in sheaths lined by visceral and parietal synovial layers. The A2 and A4 annular pulleys arise from the periosteum of the proximal half of the proximal phalanx and the midportion of the middle phalanx, respectively (Fig. 1). The A1, A3, and A5 pulleys are joint pulleys that arise successively from the palmar plates of the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints. The palmar aponeurosis pulley is composed of the transverse and ver-
tical fibers of the palmar fascia and is clinically important when other proximal components of the sheath have been lost. The thin, condensable cruciate sections of the sheath—C1 (between A2 and A3), C2 (between A3 and A4), and C3 (between A4 and A5)—collapse to permit the annular pulleys to approximate each other during digital flexion. The flexor tendons are weakly attached to the sheath by filmy mesenteries composed of vincula.

Flexor tendons are oval. The flexor digitorum superficialis (FDS) tendons usually arise from single muscle bundles and act independently. There is often a common muscle origin for several flexor digitorum profundus (FDP) tendons, with the result that there is simultaneous flexion of multiple digits. The FDS tendons lie on the palmar side of the FDP tendons until they enter the A1 entrance of the digital sheath. Within the proximal sheath, the FDS tendon divides into two slips that wrap around the FDP tendon; rejoin dorsally by means of fibers referred to as the chiasma tendinum, or Camper’s chiasma; and terminate as they insert along the proximal half of the middle phalanx (Fig. 2). The FDP tendons pass through the FDS bifurcation to insert into the proximal aspect of the distal phalanges.

The FDP muscle acts as the primary digital flexor, while the FDS and intrinsic muscles combine for forceful flexion. Digital balance and equilibrium during flexion and extension require a complex integration of extrinsic and intrinsic activity. Forces of 200 N can be achieved during power grip.

**Nutrition**

The vascular perfusion of the flexor tendons includes longitudinal vessels, which enter in the palm and extend down intratendinous channels; vessels that enter at the level of the proximal synovial fold in the palm; segmental branches from the paired digital arteries, which enter in the tendon sheaths by means of the long and short vincula; and vessels that enter the FDS and FDP tendons at their osseous insertions (Fig. 3). Both tendons have relatively avascular segments over the proximal phalanx. The FDP tendon has an additional short avascular zone over the middle phalanx. Fortunately, synovial fluid diffusion provides an effective alternative nutritional and lubricating pathway for flexor tendons. The rapid delivery of nutrients is apparently accomplished by a pumping mechanism known as imbibition, in which fluid is forced into the interstices of the tendon through small ridges, or conduits, in the tendon surface as the digit is flexed and extended.

**Biomechanical Properties**

As much as 9 cm of flexor tendon excursion may be required to produce composite wrist and digital flexion, while only about 2.5 cm of excursion is required for full digital flexion with the wrist stabilized in a neutral position (Fig. 4). The greater the distance a tendon is from the axis of joint rotation, the greater the moment arm and the less motion that a given muscle contraction will generate at that joint. Conversely, a shorter moment arm will result in more joint rotation from the same tendon excursion. The moment arm, excursion, and joint rotation produced by the flexor tendons are governed by the constraint of the pulley system. Loss of portions of the digital pulleys may significantly alter the normal integrated balance between the flexor, intrinsic, and extensor tendons. The A2 and A4 pulleys are the most important to these mechanical functions; the loss of a substantial portion of either may diminish digital motion and power or lead to flexion contractions of the interphalangeal joints (Fig. 5).

Several studies have been carried out to determine the tendon forces generated by various active and passive functions. Despite variation in the methodology used in these investi-
tigations, it appears that during unresisted passive flexion, flexor tendons are subjected to 2 to 4 N of force. Active flexion with mild resistance may result in up to 10 N of force; moderate resisted flexion, up to 17 N; and strong composite grasp, up to 70 N. Firm tip pinch can apparently generate as much as 120 N of tensile load on the index FDP tendon. Forces produced by the FDS tendon have been shown to be considerably less than those produced by the FDP tendon during grasp and pinch.

From a clinical perspective, it should be remembered that these loads are substantially increased by the resistance created by stiffness and swelling of the finger and by the increased drag that a healing tendon may experience within its sheath. Pressure between the pulleys and the flexor tendons may reach as high as 700 mg Hg during active flexion, which perhaps explains the histologic alterations to the fibrocartilagelike tissue in tendons beneath annular digital pulleys.

**Tendon Healing**

After much debate, almost all investigators now believe that tendons have both an intrinsic and an extrinsic capability to heal. The relative contribution of each will depend on factors that relate to the injury and the surgical repair. In the clinical setting, it is impossible to isolate the two types of healing, and the cellular events can be viewed as similar for all flexor tendons. After repair, tendon healing involves an inflammatory phase that lasts 48 to 72 hours, a fibroblast- or collagen-producing phase that lasts from 5 days to 4 weeks, and a remodeling phase that continues for about 112 days. During the inflammatory phase of tendon healing, the strength of the repair is almost entirely reliant on the strength of the suture itself, with a modest contribution from the fibrin and the clot between the tendon ends. Strength increases rapidly during the fibroblast- and collagen-producing phase, when granulation tissue is forming in the defect. When extrinsic healing predominates, adhesions between the tendon and its surrounding tissues are inevitable. Healing that is largely based on intrinsic cellular activity will result in fewer, less dense adhesions.

In a series of definitive laboratory experiments, Gelberman et al. demonstrated that, compared with immobilization, the application of early passive motion stress to...
repaired canine tendons led to a more rapid recovery of tensile strength, fewer adhesions, improved excursion, better nutrition, and minimal repair-site deformation. They concluded that passive mobilization enhances healing by simultaneously stimulating maturation of the tendon wound and remodeling of the tendon scar. From these studies and the work of many others, it appears that the most effective method of restoring strength and excursion to repaired tendons involves the use of a strong, gap-resistant suture technique followed by the application of controlled-motion stress.

Considerable research is being conducted in an effort to understand the influence that soluble polypeptides, including mitogens (growth factors and hormones) and chemotactic and differentiating factors (e.g., fibronectin), exert on the cellular sequence of tendon repair. These factors have been shown to play a role in both normal and pathologic processes. Continuing investigations may, in time, modify current repair methods and postrepair motion protocols for severed flexor tendons.

Adhesion Formation and Control
Factors that influence the formation of excursion-restricting adhesions around repaired flexor tendons include trauma to the tendon and its sheath from the initial injury and the reparative surgery, tendon ischemia, tendon immobilization, gapping at the repair site, and excision of components of the tenon sheath. Quantitative measures have shown that adhesions form in proportion to the amount of tissue crushing and the number of surface injuries to the tendon. Disruption of the vincula also has been associated with a decrease in the recovery of tendon excursion. There continues to be considerable debate about whether primary repair of the digital sheath is favorable for adhesion reduction.

Various biochemical agents have been studied in an attempt to modify adhesion formation around tendon repairs. Anabolic steroids, antihistamines, and nonsteroidal anti-inflammatory drugs have been subjected to recent laboratory investigations, and there has been some evidence that ibuprofen and indomethacin may improve tendon excursion by blocking prostaglandin synthesis through the inhibition of the enzyme cyclo-oxygenase at the cellular level. While hyaluronate appears to reduce adhesions around healing tendons, it was found to have no statistically significant effect on digital motion in one double-blind study.

Flexor Tendon Repair
Indications and Contraindications
Primary repair of flexor tendons severed in the digital sheath has now universally replaced the “no-man’s-land” concept, which favored secondary grafting. The concept that flexor tendon repair should be considered a surgical emergency has also been effectively discredited by several studies that demonstrate that equal or better results can usually be achieved by delayed primary repair.

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James W. Strickland, MD

Fig. 5 Function of the finger flexor tendon pulley system. **Top,** The arrangement of the annular \((A_1, A_2, A_3, A_4, A_5)\) and cruciate \((C_1, C_2, \text{ and } C_3)\) synovial pulleys of the finger flexor tendon sheath within the intact fibro-osseous canal and the normal moment arm (MA) and FDP tendon excursion as the proximal interphalangeal joint is flexed to 90 degrees. **Bottom,** The biomechanical alteration that results from excision of the distal half of the \(A_2\) pulley; the \(C_1, A_3, \text{ and } C_2\) pulleys; and the proximal portion of the \(A_4\) pulley. The distance between the distal edge of the \(A_2\) pulley and the proximal edge of the \(A_4\) pulley is the intra-annular pulley distance (IAPD). The moment arm is increased and a greater FDP tendon excursion is required to produce 90 degrees of flexion because of the bowstringing that results from the loss of pulley support.
Flexor Tendon Injuries: Treatment Principles

flexor tendon suture. It has also been shown that it is better to repair both the FDP and the FDS tendons rather than the FDP tendon alone, which was once the preferred option.

There are a number of important contraindications to primary suture of severed flexor tendons, including severe multiple tissue injuries to the fingers or palm, wound contamination by potentially infecting materials, and significant skin loss over the flexor system. Concomitant fractures and neurovascular injuries are not necessarily contraindications to primary or delayed primary suture. If the fracture can be anatomically reduced and adequately stabilized, it is almost always better to proceed with flexor tendon repair and microscopic nerve and vessel suture, recognizing that the ultimate results after combined tissue injuries are not as good as those following tendon severance with no associated injuries. Rejoining the tendon at its normal length acutely or subcutaneously is usually preferable to delaying the repair for several weeks, because of the inevitable deterioration of the tendon ends and shortening of the extrinsic muscle-tendon system.

Examination and Preparation

The surgeon must carefully examine the patient’s hand to determine the total extent of the injury. Alterations in the normal resting posture of the digits will help identify the loss of continuity of one or both flexor tendons, and well-known functional tests will confirm the loss of FDP and/or FDS action. Lacerations on the palmar aspect of the fingers will almost always injure the FDP tendon before severing the FDS tendon, but the absence of FDP function alone does not rule out the possibility of a near-complete FDS division.

A careful sensory evaluation of the palmar aspect of the distal phalanx will allow the identification of injuries to the digital nerves and will provide important information for planning incisions for their exposure and repair. A deep wound with lacerations of both digital nerves almost surely indicates division of the digital arteries as well. While the digit will probably survive the loss of both vessels, the viability of the skin flaps used for exposure may be in jeopardy. In addition, digital ischemia may impair tendon and nerve healing and result in severe cold intolerance. It is important that one or both digital arteries be repaired in these complex injuries.

Surgical Considerations

The techniques of flexor tendon repair and the protocols for postoperative mobilization of the repaired tendon were, for many years, based on anecdotes and hearsay. In retrospect, publications on this topic often had deficiencies in scientific methodology. In recent years, various hand, orthopaedic, and plastic surgery journals have published a plethora of laboratory and clinical information about methods of flexor tendon repair and postrepair motion protocols. These investigations stem from the consensus that the greater the increments of repair-site stress and tendon excursion, the faster the tendon will achieve normal tensile strength with fewer motion-restricting adhesions. Trying to interpret these reports and compare them with those from other studies is almost impossible given the different laboratory models used (e.g., in vivo versus in vitro), different testing methods, and diverse definitions of failure. A thorough review of this information does, however, permit a few reasonably supportable conclusions on which to base a clinical protocol designed to attain the best possible digital performance after flexor tendon division.

While most in vivo studies of laboratory animal tendon repairs suggest that there is a 10% to 50% loss of the initial repair strength during the first 5 to 21 days following injury, it should be recognized that these studies were carried out on immobilized tendons. Some recent investigations of tendon repairs in which controlled passive motion was used indicate that this drop may be substantially lessened by early stress application. By converting newtons to grams, it is possible to establish some working numbers that allow the determination of the strength of various tendon repairs at the time of surgery and throughout the healing period. These data can then be matched with the stress forces of postrepair motion protocols to determine the relative risk of tendon rupture with each. Conservative working numbers for tensile demands on a normal tendon in an unswollen finger can be estimated as follows: passive motion, 500 g; light grip, 1,500 g; strong grip, 5,000 g; and index finger tip pinch (FDP tendon), 9,000 g. Corresponding values for a finger that has undergone an FDS repair can be calculated as 15% to 30% of these normal values.

Suture Techniques

Core Sutures

Numerous methods of tendon suture (Fig. 6) have been advocated in an effort to satisfy the six characteristics of an ideal repair: (1) easy placement of sutures in the tendon, (2) secure suture knots, (3) smooth juncture of tendon ends, (4) minimal gapping at the repair site, (5) minimal interference with tendon vascularity, and (6) sufficient strength throughout healing to permit the application of early motion stress to the tendon.

A comparison of a number of published investigations of the characteristics and performance of various
flexor tendon repairs leads to the following general conclusions: (1) The strength of a flexor tendon repair is roughly proportional to the number of suture strands that cross the repair site. (2) Locking loops contribute little strength to the repair and may actually collapse and lead to gapping at moderate loads. (3) Repairs usually rupture at the suture knots. (4) Synthetic 3-0 or 4-0 braided sutures are probably the best for flexor tendon repair.

The observation that the number of suture strands crossing the repair will determine the strength of the repair has been best demonstrated by Savage, who found that a complex six-strand repair was three times stronger than a two-strand repair, and by three recently published reports in which four-strand repairs (Fig. 7) were found to have approximately twice the strength of two-strand repairs in vitro. From many repair studies, it is possible to conservatively list the initial strength of two-, four-, and six-strand flexor

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**Fig. 7** Types of four-strand flexor tendon repairs. Top, Robertson and Al-Qattan interlock stitch. Bottom, Lee double-loop locking suture.
tendon repairs and predict their strength at 1 week (−50%), 3 weeks (−33\frac{1}{3}%) and 6 weeks (+20%). This information, based on results in unstressed in vivo studies, can then be plotted against the stress forces of passive-light and moderate-strong active motion to determine the relative safety of each method (Fig. 8, Table 1). From this assessment, it appears that only the six-strand repair can be considered safe from rupture throughout the entire period of unstressed healing. Unfortunately, six-strand repairs, such as those described by Savage,\textsuperscript{12} are technically difficult and may damage the tendon excessively or compromise its nutrition or ability to heal.

Peripheral Epitendinous Sutures
Several studies have indicated that gapping at the repair site becomes the weakest part of the tendon, unfavorably alters tendon mechanics, and may attract adhesions, resulting in decreased tendon excursion. The importance of the use of a peripheral circumferential epitendinous suture at the completion of a tendon repair has been demonstrated by the observation that such sutures may provide a 10% to 50% increase in flexor tendon repair strength accompanied by a significant reduction in gapping between the tendon ends. These benefits have been further confirmed by experiments that apply cyclic loads to the tendon repair. The running-lock stitch and horizontal-mattress epitenon/intrafiber methods have been shown to be the strongest of the peripheral suture techniques (Fig. 9).\textsuperscript{16-18} The gap-retarding quality of these peripheral epitendinous sutures is particularly important in light of the finding that gapping of tagged flexor tendon repairs is associated with poorer clinical results.

On the basis of data from published reports,\textsuperscript{16,17} it appears to be a safe assumption that a horizontal-mattress or running-lock peripheral circumferential epitendinous suture will increase the strength of two-strand repairs by at least 40%, or about 700 g of repair strength to each core suture repair, and that the improved strength will be maintained throughout the healing period. The addition of this 700 g of repair strength to the values already presented for two-, four-, and six-strand repairs performed without epitendinous sutures demonstrates the increased safety that can be obtained (Fig. 10, Table 2). The safety of a four-strand core-stitch repair combined with a running-lock or horizontal-mattress circumferential epitendinous stitch should permit even light composite grip during the entire healing period.

Sheath Repair
In recent years, many surgeons have advocated repair of the flexor tendon sheath after tendon suture. The stated advantages of sheath repair are that it would serve as a barrier to the formation of extrinsic adhesions, should provide a quicker return of synovial nutrition, would act as a mold for the remodeling tendon, and should result in better tendon-sheath biomechanics. Two disadvantages are that sheath repair is often technically difficult and that

<table>
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<th>Table 1</th>
<th>Estimated Repair Strength Without Epitendinous Suture</th>
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<td>Type of Repair</td>
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</tr>
<tr>
<td>Four-strand</td>
<td>3,600 g</td>
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<tr>
<td>Six-strand</td>
<td>5,400 g</td>
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the repaired sheath may narrow, restricting tendon gliding. There have been a number of conflicting laboratory and clinical studies regarding the biologic and biomechanical benefits of sheath repair, and no clear-cut advantage has yet been established. A number of autogenous and synthetic materials have been employed to restore sheath continuity, including tendon, fascia, extensor retinaculum, peritenon, veins, silicone sheeting, and polytetrafluoroethylene, but these methods are rarely required in the acute setting.

**Suture Materials**

Efforts have been made to determine which are the best tendon suture materials. A polyfilament ensheathed by caprolactam was found to be the strongest by one investigator. Absorbable sutures developed for tendon repair seem advantageous because of low long-term foreign-body tissue reaction and reduction of the stress-shielding effects of the host tissue. Unfortunately, the optimal rates of material absorption and strength reduction have yet to be determined. In actual practice, 3-0 and 4-0 braided polyester sutures are the most commonly employed because of their ease of placement, adequate strength, and minimal elasticity.

**Partial Tendon Lacerations**

There has been debate regarding the appropriate management of partial tendon lacerations. Initial investigations created considerable controversy because they recommended that partial flexor tendon lacerations should not be repaired. Recent studies have demonstrated that partial lacerations of 60% or less need not be sutured, but that those greater than 60% should be repaired. The possibility of entrapment, rupture, and triggering of unrepaired partial tendon lacerations has also been reported.

**Rehabilitation**

**Theory of Early Postrepair Motion Stress**

Splints and exercise programs are now routinely used early in the postrepair period in an effort to assist the functional recovery by influencing the biologic process of collagen synthesis and degradation. Favorable remodeling of the scar around a healing tendon is best accomplished by applying stress to the tendon, which in turn transmits stress to the adjacent scar. Small loads result in significant elongation of tendons. As the load increases, the percentage of elongation rapidly decreases until further loading results in tendon breakage. Because the strain resulting from the application of a small force to a tendon is probably the result of changes in the restricting scar, it appears to be biologically effective to impart small but frequent forces in opposite directions in an effort to modify and elongate restrictive tendon adhesions. Although some excellent research is being carried out, there is still inadequate documentation of how much stress is appropriate, the optimum duration and frequency of stress application, and the most advantageous methods for the delivery of that stress to a finger after a tendon injury.

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**Fig. 9** Peripheral epitendinous suture techniques. A, Simple running stitch. B, Running-lock loop (Lin et al). C, Halsted continuous horizontal-mattress suture (Wade). D, Horizontal-mattress intrafiber suture (Mashadi and Amis). E, Running-lock suture (Indiana). The running-lock loop suture, the Halsted continuous horizontal-mattress suture, and the horizontal-mattress intrafiber suture have been shown to be the strongest.
Numerous techniques and modifications of techniques have been advanced in an effort to mechanically alter the normal biologic sequence of tissue healing and to modify the formation of adhesions around a tendon repair. Applying early postrepair motion stress to flexor tendon repairs has been shown to be beneficial for more rapid recovery of tensile strength, fewer adhesions, improved tendon excursion, and minimal repair-site deformation in a canine model. The load at failure of immediately mobilized tendons tested at 3 weeks was twice that of immobilized tendons, while the linear slope was almost three times greater and the differences continued at each interval through 12 weeks. It seems that greater magnitudes, frequencies, and durations of motion stress may have an accelerating effect on tendon healing and that almost all splinting and passive/active protocols now permit greater interdigital motion at more frequent intervals than was previously recommended.

Studies also have been conducted in an effort to determine the normal amount of flexor tendon excursion resulting from increments of digital joint motion and the amount of excursion that may occur with the various splints that are commonly employed after tendon repair. It has been observed that passive metacarpophalangeal joint movement produces no relative motion of the flexor tendons. Distal interphalangeal joint motion produces excursion of the FDP tendon of 1 to 2 mm per 10 degrees of joint flexion, while each 10 degrees of proximal interphalangeal joint flexion results in excursion of both the FDP and the FDS tendons of about 1.5 mm. It has been demonstrated that passive metacarpophalangeal joint movement produces no relative motion of the flexor tendons. Distal interphalangeal joint motion produces excursion of the FDP tendon of 1 to 2 mm per 10 degrees of joint flexion, while each 10 degrees of proximal interphalangeal joint flexion results in excursion of both the FDP and the FDS tendons of about 1.5 mm. Studies measuring the excursion of tagged flexor tendon repairs have demonstrated that there is a substantial decrease in the normal movement of the FDP tendon to an average of 0.3 mm per 10 degrees of distal interphalangeal joint flexion (36%), while proximal interphalangeal joint motion retained about 1.3 mm (90%) of FDS and FDP excursion per 10 degrees of flexion.

The amount of tendon excursion that should occur for uninjured tendons in the original Kleinert splint, modifications of the Kleinert splint with a palmar-bar pulley (Brooke Army Splint), and an experimental “synergistic” dynamic tenodesis splint that permits wrist extension (Mayo Clinic splint) has also been studied. The results demonstrate that improved excursion can be expected from the use of a palmar bar and that even greater excursion can be expected if wrist extension is added (Fig. 11, Table 3). Differential excursion between the two digital flexors was also increased dramatically by use of the synergistic splint. It has been demonstrated that if an active motion protocol is selected, the wrist should be at 45 degrees of extension with the metacarpophalangeal joints flexed to 90 degrees in order to minimize the force required to achieve or hold full active composite digital flexion.

### Practical Early Postrepair Motion Stress Protocols

On the basis of this information, the best postoperative flexor tendon repair protocol probably (1) compensates for

<table>
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<th>Type of Repair</th>
<th>0 Weeks</th>
<th>1 Week</th>
<th>3 Weeks</th>
<th>6 Weeks</th>
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<td>Two-strand</td>
<td>2,500 g</td>
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<td>1,700 g</td>
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<td>Four-strand</td>
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<td>2,150 g</td>
<td>2,800 g</td>
<td>5,200 g</td>
</tr>
<tr>
<td>Six-strand</td>
<td>6,000 g</td>
<td>3,000 g</td>
<td>4,000 g</td>
<td>7,200 g</td>
</tr>
</tbody>
</table>

*Horizontal-mattress or running-lock suture.*
swelling of the finger, (2) keeps the wrist and metacarpophalangeal joints flexed at rest, (3) keeps proximal and distal interphalangeal joints extended at rest, (4) passively flexes all digital joints before wrist extension, (5) permits active maintenance of passively achieved digital flexion with the wrist extended, and (6) utilizes frequent application of motion stress.

Several programs combining a strong four-strand repair or its equivalent with a running-lock loop, horizontal-mattress, or intrafiber circumferential epitendinous repair and employing early protected passive and active motion have now been developed, and the results are clearly better than with previous, more conservative techniques.[27,28]

Summary

Current information supports the use of a four-strand core stitch or its equivalent for flexor tendon repair combined with a strong continuous peripheral epitendinous suture. This suture method should impart sufficient strength to the repair to permit a vigorous postrepair motion protocol, which appears to maximize the excursion of the repaired tendon while minimizing the possibility of rupture. Although the results of these techniques are encouraging, rapid advances continue to occur in many areas of flexor tendon surgery, and even better techniques will lead to improved results in the future.

References

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