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Managing the Injured Tendon: Current Concepts

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ABSTRACT: Despite advances in understanding of the mechanical aspects of tendon management with improved suture technique and early stress application with postoperative therapy, clinical results remain inconsistent after repair, especially within the synovial regions. Complementary research to enhance the intrinsic pathway of healing, suppress the extrinsic pathway of healing, and manipulate frictional resistance to tendon gliding is now the focus of current basic science research on tendons. In the future, application of these new biologic therapies may increase the "safety zone" (or tolerance for load and excursion without dysfunctional gapping) as therapists apply stress to healing tendons and may alter future rehabilitation protocols by allowing greater angles of motion (and thus tendon excursion), increased external load, and decreased time in protective orthoses (splints). However, at this time, the stronger repair techniques and the application of controlled stress remain the best and most well-supported intervention after tendon injury and repair in the recovery of functional tendon excursion and joint range of motion. The hand therapist's role in this process remains a critical component contributing to satisfactory outcomes.

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The purpose of this article was to review current concepts that influence clinical decision making for the management of tendon injury. For additional detailed reviews, the interested reader is referred to chapters on flexor[1–5] and extensor[6,7] tendons, which define anatomy, considerations for treatment, and specific protocols for all zones in both systems.

Most functional problems with injured tendon systems are related to the tendon's response to injury and repair,[8] and despite decades of research on the subject, the problem of restrictive scar formation remains one of the most unpredictable factors contributing to postoperative morbidity.[9] Complications of gap formation and repair-site elongation or rupture continue to be an issue especially with intrasynovial tendon repair.[10–14]

The goals of tendon management after repair are to promote intrinsic tendon healing and minimize extrinsic scarring to optimize tendon gliding and functional range of motion (ROM).[15] Over the past four decades, contributions from both clinicians and basic science researchers have improved our knowledge of tendon structure, kinesiology, biomechanics, the biologic response to tendon injury and repair, mechanical characteristics of the various tendon constructs, and the effect of post-repair motion and load on healing and functional ROM.[16–18] The evolution of tendon management is well reviewed in the literature.[5–7,10,13,19,20]

CURRENT REHABILITATION CONCEPTS

Flexor System

Techniques for the *mechanical application* of stress in the flexor system with regard to orthotic (splint) geometry and patterns of motion have not changed much in the past decade. However, over the past 20 years, postoperative orthotic geometry has been gradually altered to relax the wrist and the metacarpophalangeal (MCP) joints to a more neutral position with a decrease in the popularity of dynamic traction (thought to contribute to proximal interphalangeal [PIP] joint flexion contracture)[21] and a change in traction vectors (if used) from the forearm to the distal palmar crease.[22,23] Contemporary clinical practice includes the following: 1) an increase in the use of early

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active tension, including increased angles of allowable flexion[10] and active tension applied with the wrist positioned in mild extension to reduce internal tendon forces;[24] 2) a change in the technique of wrist tenodesis by positioning the MCP joints in more extension with this maneuver to effect increased tendon excursion;[25] 3) a greater appreciation for the importance of some isolated motion of the distal interphalangeal (DIP) joint;[1,26–30] and 4) the timing of the application of stress within three to five postoperative days when wound inflammation begins to subside.[25,31–35]

The contribution of postoperative therapy is well recognized. A recent systematic review of surgical and adjuvant (*biologic*) therapies to prevent adhesions in the flexor system after surgery determined that the only intervention that demonstrated clinical significance in adhesion prevention was early postoperative mobilization, but that to be the best method of mobilization remains controversial.[12]

Extensor System

In the extensor system, we have learned that 1) extensor tendons in all zones (except zone I, isolated wrist extensor tendons, and the musculotendinous juncture) tolerate controlled *active* motion; 2) gapping and rupture are rarely an issue in carefully applied postoperative regimens even with repairs of low tensile strength; 3) zones III and IV do not require continuous immobilization for four to six weeks and will tolerate short arc motion (SAM) of 30° in the initial weeks of healing;[36–38] 4) we probably can allow more digital joint motion with injury in zones V through VII than had previously been thought possible;[39,40] 5) wrist position is critical in decreasing resistive forces from the antagonistic flexor system[24] and is a factor in true tendon excursion gained with digital motion;[41] 6) we have most likely been moving these tendons *actively* all along within the confines of their dynamic orthoses;[42] and 7) early referral to therapy with attention to orthotic geometry and applied stress is a critical variable to outcomes, especially to prevent joint contracture and intertendinous adhesion at the retinacular level.[7,43,44]

Only one systematic review was found comparing the effectiveness of different rehabilitation protocols for repaired extensor tendons. The results found that short-term outcomes after immobilization were inferior to early-motion programs, and that there was some limited support for early passive controlled mobilization over early active mobilization at four weeks. No conclusive evidence was found, however, regarding the long-term (weeks) effectiveness of the different rehabilitation protocols.[45] Therefore, in both flexor and extensor systems, evidence supports early motion at a repair site but does not exist for any specific protocol.

Early Mobilization

Studies demonstrate a rapid and significant decrease in tendon and ligament material properties following immobilization of these structures.[46] Buckwalter and Grodzinsky[47] point out that one of the most important concepts in orthopedics in the past century is the understanding that loading accelerates healing of bone, fibrous tissue, and skeletal muscle, and thus, we can surmise that early controlled motion to the healing tendon, as just noted, remains our best current treatment after tendon repair.

The postoperative challenge for the therapist remains unchanged. The application of stress to a tendon repair site is dependent on the ability to plot the tensile strength of any given repair against the internal tendon forces determined by joint position, external load, and estimated resistance to tendon glide.[10,48]

WHAT IS NEW WITH TENDONS?

Recent advances in tendon management include surgical technique,[10,14,49–56] manipulation of the biologic and biochemical environments to improve intrinsic repair response[14,17,33,57–61] and diminish extrinsic healing,[62–66] and efforts to reduce frictional resistance to tendon excursion.[25,34,35,67–74] These advances may increase the "safety zone" (or tolerance for load and excursion without dysfunctional gapping) for therapists as they apply stress to the healing tendon. Rehabilitation protocols in the future may be altered to allow greater angles of motion (and thus tendon excursion), increased external load, and decreased time in protective orthotics, as these new biologic therapies are applied to clinical situations. The flexor system is the model for most basic science research on tendon.

Mechanical Increase of Repair Strength

When initiating an active motion program for tendon repair, therapists must know the type of core suture, the addition and technique of an epitenon suture, and the status of pulleys at the level of repair. The tensile strength of a freshly sutured tendon depends on the strength of the suture material, the suture method, the balance between the strands and knot, the number of strands, the size of the tendon, and the addition of a circumferential suture to a core suture.[10,14]

The load at which a tendon repair gaps is the number that we must recognize with controlled active-motion programs. These numbers (of load to gap), defined as either newtons (N) or grams (g), can be found in the numerous studies on repair technique. *These reported numbers are the basis of the parameters for motion and load with our early-motion programs.*

Gap formation has been associated with increased adhesion formation, poor gliding function, and poor clinical results.[75,76] Although most surgeons believe that gapping of more than 1–3 mm is incompatible with a good result, an *in vivo* study has demonstrated that gaps of up to 10 mm in repaired flexor digitorum profundus (FDP) tendons are compatible with the functional ROM.[77] Gelberman et al.[78] have demonstrated that gapping at a repair site of more than 3 mm does not increase the prevalence of adhesions or impair ROM but does prevent the accrual of strength and stiffness that normally occurs with time. Gap resistance of less than 2 mm is the goal for most tendon repair techniques designed for early-motion protocols,[10,15,16,53,56,79,80] and is supported by previous studies on the effects of gap formation.[75,76,78]

Flexor System

Modifications of surgical techniques in the flexor system have led to increases in strength, stiffness, and resistance to gap formation at a tendon repair site. The benefits of multi-strand surgical repairs are minimization or prevention of gap formation and increase in the margin of safety with stress application in early-motion programs.[10] Strickland[10] has developed "rough" estimates and working numbers for two-, four-, and six-strand repairs plotted against the forces of passive motion, light active flexion, and strong composite grip adjusting for the resistance of friction, edema, and stress. He concluded that "light" composite digital flexion during the entire healing period is safe with any four-strand core stitch repair with a running lock or horizontal mattress circumferential epitendinous stitch.[10,16]

The literature is replete with studies on flexor tendon repair technique with no definitive answer on which technique is best. A recent *review* of experimental studies on repair technique recommends these modifications: "(1) 8-core suture strands with a high caliber suture material, (2) a purchase length of approximately 1.2 cm, (3) a locking loop configuration with (4) the knot placed outside of the repair site, and (5) a peripheral suture placed deep into the tendon and far from the cut tendon end."[14] Using the combination of these five technical modifications, Kim et al[14] compared the results in an experimental study of 30 canine tendons with those of a previous canine study from the same institution.[79] The modified repairs greatly outperformed the repairs in the earlier study[79] in terms of tensile strength, gap resistance, and rupture rate.[14] Conversely, Peltz et al.[53] have recently recommended a four-strand cross-locked cruciate technique (Adelaide technique) for repairing tendons in zone II as a "favorable method in terms of strength and simplicity." In their experimental study, comparing the four-strand cross-lock with a 2-mm cross-lock and a 4-mm cross-lock, the 4-mm cross-lock demonstrated a higher resistance to 2-mm gap formation at 55–62.2 N.[53] They make the point that a six- or even an eight-strand repair has *practical limitations* dependent on the surgeon's technical ability, increase in surgical time, injury to tendon vascularity, excessive tissue handling, and bulk of the repair (which would increase the work of flexion [WOF]).[53] The four-stand core suture is most commonly used by hand surgeons at this time, with some using a six-stand core suture and a few using an eight-strand core (J.B. Tang, personal communication, 10.20.2011).

The use of other newer repair techniques (tendon splints, grafts, and metallic implants) are not widely accepted but have the same goals of tolerating active tension to gain excursion in the first three weeks of tendon healing.[4,14,49,50,52,55,81] The use of a recently developed 4-0 Fiber Wire (Anthrex, Naples, FL) is gaining popularity.[4,50] McDonald et al.[56] have found that, with a secure attachment to the tendon, the multifilament stainless steel's lower elongation and better knot-holding ability may result in a greater force to produce a 2-mm gap and a greater ultimate tensile strength in a tendon repair.

The WOF, or resistance to tendon excursion, remains an issue with the increased bulk of multi-strand repairs, especially in the intrasynovial regions,[82] and presents its own challenges in clinical practice. Amadio[74] makes the point that some of these repair techniques designed to withstand the forces of active motion, especially those with "multiple loops or knots on the anterior surface, also generate high-frictional forces with movement and may abrade the pulley surface over time."

Some surgeons still use a two-strand core and epitenon suture or four-strand modified Kessler suture with epitenon. One survey of practice patterns in flexor tendon repair found that despite the proposed advantages of the newer, stronger repair techniques, the two-strand Kessler core with a simple peripheral suture remains the most popular flexor tendon repair technique and that most respondents favored sheathe closure.[83] *Although this study was published four years ago and although many hand surgeons have moved to multi-strand repairs, it is my experience in a practice with a general orthopedic referral base that the two-strand core is still used requiring the hand therapist to adjust the rehabilitation program accordingly. With each case, the therapist will need to know specifics regarding the type of repair and tensile strength to gapping and status of the pulleys that will define the postoperative protocol.*

Extensor System

Extensor tendon repair technique is dependent on the dimensions and fiber direction of the tendon and

the status of the peritendinous tissue.[6] Relatively few studies have evaluated extensor tendon repair technique. Clinically, we observe that these repairs (excluding zone I repair, isolated wrist extensor tendon repair, and repairs at the musculotendinous juncture) tolerate immediate controlled *SAM* with few complications as long as the tendon is rested/splinted in its shortened position between exercise regimens.[7,36,37,84]

Studies defining the tensile strength of the various repair techniques[51,80,85,86] with respect to load to gapping guide the therapist in treatment planning for the application of postoperative stress. Newport and Williams[85] reported the biomechanical characteristics of extensor tendon suture at 2-mm gapping and at failure. The mattress suture gapped 2 mm at 488 g and failed at 840 g; figure-of-eight suture gapped at 587 g and failed at 696 g; Kessler suture gapped at 1,353 g and failed at 1,830 g; and Bunnell suture gapped at 1,425 g and failed at 1,985 g. Woo et al.[80] compared four extensor tendon repair techniques specifically designed for zone IV injuries and demonstrated that the strength for 2-mm gapping of the modified Becker suture technique (56.0 ± 9.2 N) and the modified Kessler technique (48.6 ± 12.6 N) were significantly higher ($p < 0.05$) than those of the six-strand double-loop and figure-of-eight techniques. More recently, Lee et al.[51] compared the augmented Becker, modified Bunnell, and running-interlocking horizontal mattress repair methods in 24 extensor tendons in eight cadaveric hands with regard to tendon shortening, stiffness, strength, and time needed to perform the repair. They found that the running-interlocking horizontal mattress repair was stiffer, resulted in significantly less shortening (1.7 mm) than the augmented Becker (6.2 mm) and modified Bunnell (6.3 mm) methods, and took significantly less time to perform without a significant difference in the ultimate load to failure (running-interlocking horizontal mattress repair, 51 N; augmented Becker method, 53 N; modified Bunnell method, 48 N).[51] *The decrease in loss of tendon shortening has a positive clinical significance in the recovery of composite digital flexion.*

Extensor tendons in the finger are flat and intimate with the periosteum. Mattress sutures and Kessler repairs without epitendinous stitching are often used for zones II, III, and IV.[54] As noted in the Newport study, the conventional mattress suture gaps 2 mm at 488 g and fails at 840 g,[85] and thus has a very low tensile strength. Henderson et al.[54] have proposed that the extensor tendons at this level work with a "tension" band effect and that they are suited for repair by a dorsal-only epitendinous suture technique. Their study, comparing this technique with the conventional mattress and Kessler techniques, has demonstrated that using the dorsal epitendinous suture technique is more resistant to gapping, rupture, and deformation.[54]

Any gapping at the repair site can result in elongation of the tendon callous, which is especially critical in extensor zones I–IV, where the tendon moment arms and thus excursions are small.[36,87] However, research and clinical experience have demonstrated that 4–5 mm of excursion are tolerated at levels III and IV even with repairs of low tensile strength.[7,36–38]

Evans and Thompson[36] have demonstrated, in a mathematical model (which eliminated drag), that with controlled active SAM programs, internal tendon forces at both the MCP and PIP levels with active extension from 30° of flexion to 0° of extension (at either joint) are approximately 300 g if the wrist is positioned at 20° of flexion. Therefore, any of the earlier defined suture techniques can withstand the forces of controlled active SAM if the wrist is positioned in mild flexion.[36] *Tensile strengths of extensor repairs are much less of an issue than those with the antagonistic flexor system.*

Biologic Modifications of Tendon Healing

In addition to advances in the mechanical aspects of tendon management with improved suture technique and early stress application, complementary research to enhance the intrinsic pathway of healing, suppress the extrinsic pathway of healing, and manipulate the frictional resistance to tendon gliding hold great promise for the future.[17,57] These biologic advances for tendon healing are being made at the cellular, molecular, and genetic levels in experimental models, but do not yet have clinical application.[14,17,71,74] Growth factors directly affect cellular mitogenesis and chemotaxis and are able to influence the healing cascade in a complex manner.[63] Certain growth factors and their delivery increase fibroblast proliferation, leading to increased collagen production and improved healing response.[63] Although much remains to be discovered, it has become clear that growth factors play a critical role in the complex biochemical pathways that take place after a tendon is injured.[63]

Growth factors are delivered to the injured tendon by the direct application of the growth factor itself or through the use of gene transfer techniques.[63] Direct application uses local injection, suture, or scaffold for delivery to a specific area. Localized gene therapy includes direct localized delivery (*in vivo*) and transplantation of genetically modified tendon fibroblasts or mesenchymal stem cells (MSCs) (*ex vivo*).[17]

"Gene transfer involves delivering the gene that encodes the growth factor, not the growth factor itself to the site of tendon injury."[63] The gene facilitates the cellular production of the growth factor, producing a more prolonged exposure to the growth factor than with direct application of the growth factor.

However, excessive healing may produce negative effects. A recent study of the effect of sustained delivery of basic fibroblast growth factor in the canine model found that, despite a substantial biologic response, application of the growth factor failed to improve either mechanical or functional properties of the repair, but instead, the increased cellular activity resulted in peritendinous scar formation and decreased ROM.[72]

Cell-based strategies utilize mesenchymal (adult stem) cells (MSCs) and tissue engineering to construct new tendon tissues. MSCs have the ability to differentiate into tendon fibroblasts and also have the capacity to produce extracellular matrix and secrete growth factors that are important for initiating the cascade of cellular events needed for tendon healing.[17]

Tissue engineering refers to the ability to construct *new tissues* through the use of a tissue scaffold onto which cells are placed[17] and may, in the future, lead to functional tissue replacement to repair defects.[59–61] These scaffolds, typically made of biodegradable polymers, provide a matrix that allows cell adhesion and growth in a three-dimensional configuration to simulate normal cell architecture.[17] These cell scaffolds may have a significant clinical application in the future in the delivery of molecular and gene therapy products to the zone of injury.[17,60]

The biologic control of peritendinous adhesion or the suppression of the extrinsic pathway of healing is also the focus of many studies.[66] Molecular approaches target the modulation of cytokine expression to reduce adhesion formation. Cytokines are proteins that function to mediate interactions among cells directly and regulate processes in the extracellular environment, and modulate functional activities of the individual cells and tissues.[17] Growth factors also have a role in controlling the peritendinous inflammatory response and its accompanying adhesions. It has been demonstrated that the transforming growth factor beta-1 (TGFß-1) is upregulated in healing flexor tendons[62] and, thus, contributes to the formation of adhesion around healing flexor tendons. Chang et al.[62] demonstrated, in an experimental *in vivo* study of rabbit tendon laceration and repair, that the tendons which received a neutralizing antibody to TGFß-1 had a significant increase in digital ROM when compared with the control group. The cellular chemistry is complex but is important to clinicians as there is evidence that peritendinous tendon adhesion formation can be reduced by the perioperative modulation of cytokines such as TGFß-1.[66]

Manipulation of Frictional Resistance to Tendon Glide

The friction between tendon and sheathe is an important factor causing the risk of tendon rupture.[25] Amadio[25] cautions that "an increase in surface friction will increase the load placed on the repair site during tendon motion, and may also increase adhesion formation." Current efforts to reduce the effects of friction on tendon repair are focused on venting the A2 pulley for the zone II flexor repair;[68,88] using low-friction repair construct and low-friction suture material; and modifying the tendon surface with exogenous application of surface lubricants, such as hyaluronic acid, lubricin, and phospholipids.[14,25,67,69,73,74] Amadio[74] and colleagues at Mayo Clinic are advancing our knowledge and clinical application of these surface lubricants for both restoring tendon surface and reducing the development of adhesion to minimize force requirements.

In the flexor system, it has been demonstrated that the WOF is increased when the repair site has to glide through the A2 pulley in the early wound-healing stages. In an experimental study comparing the rupture rate and resistance to tendon gliding in the chicken model, Tang et al.[68] demonstrated that tendon gliding resistance was significantly increased with preservation of the A2 pulley on postsurgical days 3, 5, 7, and 14, when compared with pulley release. They advocate partial release of the A2 pulley to reduce the WOF and to increase the chances of improved functional excursion.[68] Decreasing frictional resistance at this level with pulley plasty and resection of one slip of the flexor digitorum superficialis (FDS)[89,90] have also been recommended. The biomechanical function of the A2 pulley is well recognized both in the normal and the injured digits. Walbeehm and McGrouther[91] have described the compressional mechanism of the FDS on the FDP at Camper's chiasm with tendon load and motion at the distal edge of the A2 pulley. This mechanism may be important for tendon nutrition and synovial fluid circulation in the uninjured tendon but presents increased frictional resistance with an increase of volume from suture material and edema. The "gripping mechanism" of the FDP by the FDS may be a factor causing tendon rupture at this level.[91]

PRINCIPLES OF APPLYING STRESS TO A FLEXOR TENDON REPAIR SITE

The goals of tendon rehabilitation in any zone are to optimize functional outcomes by providing precise increments of controlled stress to promote enough differential tendon glide to control early collagen deposition; to facilitate the biochemical events that strengthen the repair site; and to avoid the complications associated with adhesion formation, gapping, or rupture at the repair site.

These goals are best met with early referral to therapy and the application of controlled stress through orthotic geometry and precise patterns of

motion. Therapists need a working knowledge of anatomy, biomechanics, and physiology of flexor or extensor tendon gliding. Therapists must also understand the tensile strengths of various repairs, forces imposed on these repairs with active and passive motion, and variables that determine how much internal force the tendon(s) can tolerate before gapping to 2–3 mm. Communication with the surgeon should include *specific* information regarding the type of core suture and epitendinous repair; the status of the pulleys; and associated injury to vessel, nerve, ligament, or bone. Other variables include health status, patient personality, and level of patient intelligence. The experienced therapist will also adjust the program based on postoperative tissue reaction and his or her past experience regarding the skill of the surgeon. Clinical problems for the therapist include late referral, noncompliance,[92] or "stupidity"[93] of the patient; limitations imposed by managed care and insurance; suboptimal repair by the physician; incomplete or inaccurate information regarding the strength of repair; and the *therapist's ability to calculate subphysiologic versus excessive stress when applying active tension. It has been demonstrated that the outcomes are improved by experienced hand therapists.*[94]

Variations in clinical protocols from zone to zone should be altered based on changes in torque and excursion at each joint. Although tension remains constant throughout the length of a tendon in relation to external force, torque and excursion are altered in relation to variations of tendon moment arms at each joint.[48,95]

The Effect of Active Tension

The effects of immobilization and controlled stress have been studied primarily in the animal model in both synovial and extrasynovial tendons. The biochemical effects of immobilization during the inflammatory and fibroblastic stages on tendon healing are loss of glycosaminoglycan concentration, loss of water, decreased fibronectin (FN) concentration, and decreased endotenon healing.[96–102] Many elegant studies have demonstrated the positive influence of stress on healing tendon, with documented improvement of tensile strength, improved gliding properties, increased repair-site DNA, and accelerated changes in peritendinous vessel density and configuration.[103,104] Motion may enhance the diffusion of synovial fluid, increase FN concentration,[101] and facilitate fibroblast chemotaxis[105] at a tendon repair site. Stress-induced electrical potentials may increase tendon tissue healing potential.[106] Peritendinous adhesion is modulated by stress. Mechanical shear stress produces an overall antifibrotic expression pattern and downregulation of TGFß and other growth factors.[107,108] *The positive biomechanical effects of mechanical stimulation on a repair site continue to be supported by basic science research.*[109–112]

It has been demonstrated that motion and tension improve healing better than no motion, no tension, or combination of both.[113] The applied internal tendon tension or load must be sufficient to overcome the forces of friction to create controlled tendon excursion. The actual tendon excursion will be equal to the predicted excursion of earlier studies (3–5 mm)[114] only when more than 300 g of tension is applied to the repair site.[115] A component of active motion may be necessary to create true proximal migration of a flexor tendon repair site, especially at the A3 and A4 pulleys.[116] Significant improvements have been demonstrated with regard to tendon excursion at both the middle (P^1) and proximal phalanx (P^2) levels with active than passive regimens.[29] Recent experimental studies indicate that the addition of load to motion may have little effect on the final result in terms of strength and motion; thus, it is motion and not load that may be the critical factor.[74,79,117]

The Effect of Timing

Early experimental studies indicated that timing in relation to stress during the early inflammatory stage of wound healing is critical. Biomechanically, the immobilized tendon loses tensile strength in the first two weeks after repair and loses gliding function by the first 10 days after repair.[97,100,102,118–120] An experimental study on chicken flexor tendons has demonstrated that tendons treated with controlled passive motion had significantly improved tensile strength by five days after repair compared with digits treated with immobilization.[98] The magnitude of difference in strengths between the two groups increased with time. The authors of that study concluded that immediate constrained digital motion after repair allows progressive tendon healing without the intervening phase of tendon softening or weakening described in the classic study by Mason and Allen in 1941.[121]

In another study of early tensile properties of healing chicken flexor tendons, early controlled passive motion was found to improve healing efficiency.[99] The results of this study indicated that tendons exposed to controlled passive motion had significantly greater values for rupture load, stress, and energy absorbed when compared with immobilized tendons.[99]

FN, which appears to be an important component of the early tendon repair process, has been localized in a clinically relevant tendon repair model. Fibroblast chemotaxis and adherence to the substrate in the days after injury and repair appear to be directly related to FN concentration.[105] Early passive motion has been correlated with an increased FN concentration in the tendon repair model of a previous study.[101]

The importance of timing is supported in another study by Iwuagwu and McGrouther,[120] who determined that load in the first five postoperative days results in better orientation and fewer fibroblasts in repaired tendons. However, Silva et al.,[122] in an experimental study of the effects of increased *in vivo* excursion on digital ROM and strength, found that the repair-site strength changes only slightly during the first 21 days and that substantial improvements in strength are not noted until 42 postoperative days.

There is a risk in applying controlled load to a tendon in an edematous or stiff digit immediately postoperation. Halikis et al.,[31] in an experimental study comparing the effects of immobilization, immediate mobilization, and delayed mobilization on the energy required to fully flex the digit after surgical trauma, concluded that the viscoelastic forces of edema during the initial postoperative days increase the WOF. Delayed mobilization (of at least three to five days) was recommended to allow a decrease in edema and thus the WOF on internal tendon tension with controlled motion programs. Using a canine model, Zhao et al.[32,33] demonstrated that starting controlled motion at day 5 after tendon repair is advantageous over day 1, as gliding resistance associated with postoperative surgical edema and other factors is diminished.

Current research continues to support this concept of delayed mobilization (three to five days) as inflammation and WOF are diminished.[31,34,35] This time frame provides the best combination for tendon stretch and low peak force.[34] Beginning mobilization at day 7 or beyond is the least favorable; this work is supported by the classic experimental studies by Gelberman et al.[119] and others,[32] and in the clinical work of Tottenham et al.[123] and Evans.[1]

I prefer to see patients by 24 hours postoperation to apply the proper orthotic geometry, to prevent PIP flexion contracture with flexor repair and joint problems at all levels with extensor repair, for edema control and for patient education. Motion is delayed by at least three days depending on the degree of edema and inflammation. Passive SAM is applied to reduce the drag and minimize internal tendon forces before the application of active tension, which is initiated somewhere between day 3 and 5 postoperation for both flexor and extensor systems.

The Effect of Duration of Exercise

There is modest support that increased frequency improves interphalangeal (IP) joint motion in the zone II flexor system,[124,125] and none was found to delineate the effect of duration of exercise in other flexor or extensor zones. *In general, patients are noncompliant[9] with details regarding orthotic protection and home exercise programs, and clinical experience teaches us that each therapy visit should include a review and questioning regarding the same.*

The Effect of Tendon Excursion

Flexor System

Physiologic excursion after repair is undetermined. Some researchers believe that increased tendon excursion will correlate with improved functional outcomes.[10,77] In the flexor system, 3 mm[114,126] to 10 mm[77] has been found to be consistent with good functional results. Based on experimental studies, Gelberman et al.[126] found that 3–4 mm of excursion at a *flexor tendon repair* site was necessary to stimulate the intrinsic repair site without creating significant repair-site deformation. More recently, it has been demonstrated in the canine model that 1.7–2 mm excursion at the repair site is sufficient to prevent adhesion formation and that additional excursion provides little added benefit,[122] *but most early-motion programs are designed to produce more.*

Excursion is increased by the addition of wrist tenodesis. Synergistic wrist and MCP motion provide more effective excursion than wrist fixation.[127] New research supports a tenodesis pattern of combined MCP extension and wrist extension with the IP joints in full flexion to provide effective excursion to the FDP.[25] MCP joint motion alone promotes minimal differential excursion to a flexor tendon repair, and motion at this level does not offer much benefit in postoperative early-motion protocols.[10] Isolated DIP motion of at least 35° may be necessary to effect 3–4 mm of differential glide of the FDP on the FDS,[126] and hook fist position also is critical in promoting differential glide within the digital sheathe.[128] Active tension of at least 300 g may be necessary to effect 3 mm of true excursion in zone II.[116] A component of active motion has been recommended for true tendon excursion at the A3 and A4 pulley levels because of poor excursions in these regions with passive motion.[117] Excursion of the FDP in zone I has been calculated mathematically in radians to be approximately 5 mm if the distal joint is moved 57.29° (1 radian) based on a tendon moment arm at this level of 5 mm.[28] Passive motion to obtain physiologic excursion for the repaired flexor pollicis longus (FPL) is improved by 70% with the MCP splinted at 0° for IP passive flexion.[129] *Orthotic geometry and patterns of motion in the various zones are predicated in part on these studies of physiologic excursion.*

Extensor System

Early active and passive motion allowing an estimated 5 mm of excursion has proven to be successful

with extensor tendon repairs in zones V, VI, VII, T-IV, and T-V,[84] and approximately 4 mm of active excursion with extensor repair in the digital zones III and IV.[36] The reported extensor tendon excursions are variable but within a consistent range, and a number of anatomical studies are reviewed in detail elsewhere.[7,84]

Extensor tendon excursion can be calculated geometrically in radians.[130] Based on a mathematical equation (with radians), it was determined that MCP joint motion of approximately 30° for the index and long fingers and 40° for the ring and small fingers (or 0.5 radian) would effect 5 mm of extensor digitorum communis (EDC) glide at an extensor tendon repair site in zones V, VI, and VII,[84] and that PIP motion of 28.64° would effect 3.75 mm of excursion in zones III and IV.[36] Evans and Burkhalter[84] measured extensor tendon excursion intraoperatively and found by gross measurement that 30° of MCP motion effected 5 mm of extensor glide in zones V, VI, and VII, supporting the mathematical calculations for these zones. This limited excursion has worked well in my 35 years of clinical experience with early motion of extensor tendons, but others[131] believe that full digital flexion should be considered safe within the confines of the orthoses that hold the wrist in extension and fingers controlled in dynamic extension traction.

Wrist position influences EDC excursion. Extensor tendon excursions were investigated in eight fresh cadaveric limbs. The authors found that if the wrist is extended more than 21°, the extensor tendon glides with little or no tension in zones V and VI throughout a full simulated grip to full passive extension. On the basis of this cadaveric study, the authors recommend that up to 6.4 mm of tendon can be safely debrided in these zones and that full grip can be permitted postoperatively if the wrist is splinted in more than 45° of extension.[41] Their study emphasizes the importance of wrist position in tendon excursion, but their conclusions based on cadaveric study should be applied to the clinical situation with caution.

Excursions for the extensor pollicis longus (EPL) tendon vary in the literature from 25 to 60 mm and are reviewed elsewhere.[7,84] The simple angular arrangement of the flexion/extension axis at the MCP level of the fingers does not exist for the EPL in zones T-IV and T-V. Calculating excursion mathematically is complicated by the oblique course that the tendon takes at Lister's tubercle, by the moments of adduction and external rotation at the carpometacarpal (CMC) level and by the fact that alterations in thumb position alter the moment arms at each joint. Evans and Burkhalter[84] measured EPL excursion intraoperatively to determine the amount of joint motion necessary to create 5 mm of glide for the early-motion pilot study and found that, with the wrist neutral and the thumb MCP joint extended, 60° of IP joint motion effected 5 mm of tendon excursion at Lister's tubercle.

The Effect of Applied Load

When applying force to a tendon repair site, the tensile strength of the repair must be plotted against the internal tendon force. The therapist should consider the published values of load to gapping of the different combined core and peripheral sutures and then adjust the applied load with early-motion protocols in accordance with the specific repair (as reported by the surgeon) on which they are working. Internal tendon forces are increased by the resistance of the suture material,[82] pulleys,[132] periarticular soft tissues, edema and inflammation associated with wound healing,[31,119] the antagonistic muscle–tendon system, joint angle,[24,48,116] external load,[48] and the speed of exercise.[95,119,133] These elements define the WOF.

The tensile strength of each tendon must be considered. In the flexor system, these numbers must be considered for both the FDP and FDS (as the tendons do not load share)[48] as well as for the FPL. These numbers are then adjusted to account for the possible decrease in strength associated with early tendon healing (decreasing 50% at the end of first week and 33% at the end of second week and increasing by the third to fourth week).[10,16,121,134] For example, a two-strand core with epitendinous suture can be estimated to have 20–30 N (2,500 g) of tensile strength load at the time of repair, which would decrease to 1,200 g by week 1. A four-strand core with epitendinous suture can be estimated to have 43 N (4,300 g) at repair; cruciate with epitendinous, 35–46 N; Tenofix, 54 N; alloy suture, 51 N; Massachusetts General Hospital (MGH), less than 75 N, and all of them were considered to be about 50% weaker by the end of weeks 1 and 2.

Internal tendon forces, as they relate to various joint angles and applied external loads, are defined for the flexor and extensor tendons in two studies on early active motion for both tendon systems.[36,48] The results of these biomechanical analyses are presented in a series of mathematical models that negate resistance from the antagonistic muscle–tendon group and any other drag and apply a known external force. These models provide estimates of internal tendon forces imposed during controlled early-motion programs for both systems and are the basis of clinical protocols proposed by this author.[1,7,30,37] *As the WOF is factored into the equation, these numbers are, at the very least, doubled.*

Flexor System

Internal tendon forces with active and passive motion have been measured in two *in vivo* studies.[134,135] Both studies measured tendon forces on intact tendon at the level of the carpal tunnel while the patients were under local anesthesia, and although the information that they provide is of value, it is important to note that the numbers of internal force provide us with

limited information because, in both studies, external forces were undefined (the end arc of flexion and applied external load) and because the intact tendons were not subject to the increase in forces from suture and wound reaction. The work of Urbaniak et al.[134] showed that passive digital flexion–extension produces a range of 200–300 g; that flexion against "mild resistance" did not impose more than 900 g of internal tension; and that flexion against moderate resistance imposed approximately 1,500 g of internal tension. Schuind et al.[135] measured flexor tendon forces up to 900 g with passive motion and 3,500 g with "active unrestricted motion." Their measurements of "active unrestricted flexion" (otherwise undefined) ranged from 400 to 3,500 g for the FPL, 100–2,900 g for the FDP, and 300–1,300 g for the FDS, even under testing conditions.[135] The wide variation in these numbers, in a group of patients who were given the same instruction for motion, *probably is representative of what happens in the clinical situation when patients are asked to "gently move" a joint.*

Internal tendon forces have also been calculated mathematically as they relate to joint position and external load with *drag eliminated.*[48] *Modified composite flexion* (wrist, 45°; MCP joint, 80°; PIP joint, 70°; and DIP joint, 40° extension) with 50 g of external pressure has been measured at 605 g for the FDS and 41 g for the FDP. However, forces rise dramatically with full composite flexion (MCP joint, 85°; PIP joint, 95°; and DIP joint, 75°) to 1,650 g for the FDS and 2,050 g for the FDP. Therefore, a four-strand repair with 4,300 g at time of repair (2,150 g by days 5–7) is at risk of rupture with acute flexion but would have a greater margin of safety with less acute angles of digital flexion. Based on these mathematical calculations[48] and clinical experience,[1,30,48] it is possible to apply active tension to a two-strand core with epitendinous suture if a *place-and-hold* technique is used with the *modified to relaxed position* of flexion. In general, internal forces will be the least in the midranges of wrist and MCP flexion.[48,95,119,133,136,137] Full flexion at the MCP joint for these active and passive programs yields little to no excursion for the FDP and increases internal tendon tension with the added resistance from MCP periarticular stiffness in this position. Tendon forces for the FDS are decreased with the wrist at 0° as compared with those with wrist flexion.[138] Even with attention to joint angle and speed of exercise,[95,119,133,136] as noted previously, the estimated numbers of internal forces must be at the very least doubled to account for increased resistance of postoperative edema, tight joint, pulley systems, and suture.[48]

Recent experimental research indicates that high-force rehabilitation does not stimulate accelerated healing after intrasynovial tendon repair, and that less than 5 N (or 510 g) of internal tendon force may be sufficient.[79] We have no studies of extrasynovial tendon, but as tendon tension remains the same along its length, this information provides us useful guidelines for the other zones.

At this time, we have no reliable and valid *in vivo* measurements for true tensile strength and internal tendon forces altered by the variables of suture material, wound healing, and pulley in the human hand. However, in review, we can surmise from the aforementioned studies that repaired tendons are best moved by postoperative days 3–5 with a load of less than 5 N[79] and excursion of at least 2 mm.[122] Internal tendon forces are increased with more acute angles of flexion and should be reserved for stronger suture technique. Clinical experience teaches that active hold is safer with the wrist in neutral to slight extension, finger flexion is safer with a modified rather than full fist, PIP flexion at 75° is sufficient, and PIP extension to 0° is critical for differential glide. Wrist tenodesis with the MCP joints in extension and some isolated DIP motion will insure improved FDP excursion as will some active tension. A slow rate of exercise will decrease internal tendon tension,[95,119,133,136] and lateral joint support for isolated DIP flexion will prevent resistance to tendon glide that volar joint blocking would impose. Freely gliding tendons are an indication of minimal peritendinous healing and need to be protected longer than those with restricting adhesion.

Extensor System

A force analysis of the EDC with the wrist in an extended position and no external load has been calculated mathematically so that conservative estimates of tendon forces can be made.[48] With the digital joints in a neutral position, tension on the EDC is 0, but as the wrist and digits are extended, the forces rise to 1,200 g. These forces drop dramatically if the wrist is placed in 20° of flexion (the position recommended for early active controlled motion) because resistance from the flexor tendons is reduced by the wrist position.[24,48] The force applied to the extensor tendon at both MCP and PIP joint levels with active extension from 30° of flexion to 0° of extension (at either joint) when calculated mathematically is approximately 300 g if the wrist is positioned at 20° of flexion.[36,48] These internal tendon forces are compatible with extensor tendon repair techniques, even those with low tensile strength.

CLINICAL TIPS BY ZONE

As noted in the previous section, rehabilitation protocols, although applying in general the same principles, continue to be a practice of individual preference by surgeons and therapists with no proof of "which technique is best" for either flexor[12] or extensor tendons.[45] The following abbreviated guidelines are my preference and are based on published clinical experience and current understanding of

tendon forces and repair strengths. These protocols are defined in detail elsewhere.[1,7,30,36,37,48,84]

Flexor System

Zone 1

The zone I tendon is a one-tendon system with a small moment arm and limited excursion. The traditional traction technique may allow too much excursion at the repair site, contributing to gap formation. I prefer a technique for limited distal joint extension and active flexion (LEAF), which addresses problems associated with the zone I repair: repair-site gapping, unsatisfactory distal joint flexion, PIP joint contracture, and incomplete FDS glide.[28,30] Gap formation at this level may be especially significant because the zone I tendon has limited excursion and less tensile strength due to smaller diameter of the repaired tendon and the "linked extension" that occurs with simultaneous PIP and DIP extension.[87,139] The LEAF technique prevents the extension of the distal joint beyond 35–45° the first four weeks postoperation and imposes patterns of motion that promote complete isolated FDS glide, passive flexion of the distal joint between 35° and 75°, wrist tenodesis (including MCP extension simultaneous with wrist extension), and active hold of the repaired FDP in the previously described *modified composite fist* position.[48]

Orthotic Geometry

The dorsal hood orthosis is positioned with a relaxed wrist position of 20–30° of flexion to reduce the viscoelastic resistance of the antagonistic extensors. The MCP joints are positioned in 30° of flexion to relax the force of the lumbrical muscle on the profundus tendon; to allow the digits to be placed passively into a modified hook fist position, which increases differential excursion of the FDP and FDS tendons;[128] and to allow safe positioning for the *active hold* component. A digital dorsal P^1 and P^2 orthosis that holds the DIP joint flexed at 45° the first 28 days is applied to prevent gap formation and to provide the controlled range for passive flexion from 45° to 75°. This rests the repair site proximal to normal position and prevents gapping. The distal joint extension block also neutralizes the action of the oblique retinacular ligaments, which act to extend the DIP joint with a dynamic tenodesis action when the PIP joint is extended by virtue of their attachment to the terminal extensor tendon. Flexion contracture at the DIP joint will not be a problem unless the A4 pulley was resected.

Patterns of Motion

The exercise regimen includes passive digital flexion; PIP extension to 0° with the MCP joint in full flexion (with the dorsal P^1, P^2 orthosis in place to prevent DIP extension); active hold for the FDS with the wrist in flexion; and wrist tenodesis. The FDS is exposed to increased tension when the wrist is positioned in flexion.[137] Composite active hold in the *modified position* applies some isolated tension to the repair site. This technique applies an excursion of approximately 3 mm to the zone I tendon in a limited arc (45–75°).[28] The modified position of the active flexion applies low loads of force (<500 g), even with the drag considered.[48] This technique is supported by previous mathematical studies of excursion and internal tendon forces, and clinical experience. The average distal joint flexion in my series with a general orthopedic referral base of more than 50 cases is as follows: PIP joint, 95° and DIP joint, 49°.[30]

Zone II

Rehabilitation of the repaired zone II flexor tendon is initiated within 24–48 hours postoperatively with wound care, edema management with Coban (3M Health Care, St. Paul, MN, USA) wraps and elevation techniques, the application of the postoperative orthosis, and patient education. Active tension is not applied until inflammation is decreased somewhere between the third and fifth postoperative days.

Orthotic Geometry

The dorsal protective orthosis is positioned with the wrist in 25–30° of flexion, the MCP joints in 30–40° of flexion, and the dorsal hood extending to the level of the finger tips to allow complete PIP and DIP extension. Four-finger traction is used only in selected cases where there is minimal edema or inflammation around the PIP joint to avoid PIP flexion contracture. If used, the traction is set up with a palmar pulley to rest both PIP and DIP joints in composite flexion.

Patterns of Motion

Under therapist supervision, the orthosis is removed for wound care and light dressing application, followed by passive composite flexion exercise to the digits with the wrist in neutral position. The passive forces should be applied with a gentle technique, slow repetition, and until the cutaneous wound has gained some tensile strength, with less than full flexion to avoid wound site tension on the corner flaps. With the MCP joints held in maximum flexion and the wrist in moderate flexion, the PIP joints are passively extended to 0°, followed by active extension in this same position by the patient. By days 3–5, as edema and inflammation are reduced, active tension is initiated at 50–70% composite flexion dependent on the drag of tight joint and edema.

Internal tendon forces for the active hold component (MCP joint, 75°; PIP joint, 70°; DIP joint, 40°) with finger tip forces less than 50 g and with drag eliminated are calculated to be 41 g for the FDP (82 g with drag considered) and 605 g for the FDS (1,210 g with drag considered).[48] External force can be measured with the Haldex™ pinch meter (JIL Tools; J.D. Mard Industries, Tuckahoe, NY) for the purpose of patient instruction.[48] These light active hold positions have been demonstrated to be compatible with a standard modified Kessler with an epitendinal repair.[48] The newer multi-strand repairs will tolerate increased angles of flexion for the active component. It is important to ensure that some active tension is transmitted to the profundus tendon. The DIP joint is not blocked but is rather positioned in a manner so that it holds a position of flexion of at least 30–35° (with lateral support). Wrist tenodesis is performed with the MCP joints in extension as described by Amadio[25] or built into the orthotic design as described by Strickland.[10] Therapy proceeds with tendon gliding exercises[128] and protocols that are well defined elsewhere.[1,5,48]

Zones III–V

Injuries in zones III–V are commonly caused by lacerations and are less commonly associated with crush. Injuries at these levels are rarely isolated, often involving nerve and vessel. *Orthotic geometry and patterns of motion* will be similar to those of zone II with small variations that address tendon anatomy, torque, and excursions at the different levels. As noted before, tension will be the same throughout the length of the tendon.[95]

Zone III

The WOF, or resistance to tendon glide, will be less a factor at this level because the tendons are extrasynovial and subjected only to linear tension reducing the risk for rupture;[140] therefore, active tension can be applied in a *modified composite fist* position (MCP joint, 40–50°; PIP joint, 60°; DIP joint, 35°) by 24–48 hours postoperation with repair strength at least equal to that of a four-strand core and epitendinal suture.

Orthotic Geometry

The dorsal hood should position the wrist at 20–30° flexion, MCP joints at 30–40° flexion, IP joints in neutral position, with or without traction.

Patterns of Motion

Active tension is applied as with zone II with respect to nerve or vessel repairs with *"place-and-hold"* active tension in the *modified position* noted earlier; tenodesis (if allowed by status of nerve and vessel), and some active hold for the FDP at DIP level.

Zone IV

The zone IV tendons pass through the carpal tunnel and are surrounded by synovial sheathes that provide lubrication and nutrition. At this level, the tendons are at risk of developing intertendinous adhesion, adhesion to the synovial sheathes, and adhesion to other structures within the carpal tunnel. *Orthotic geometry* is the same as for zones II and III. Wrist position, which should be as neutral as possible to keep pressure off median nerve and to decrease drag for active tension, can be adjusted to address tension on the median or ulnar nerve, vessel, or wrist flexor tendons as needed. *Patterns of motion* are similar as for zone III with special attention to isolated FDS glide, especially for the long and ring digits; *"place-and-hold"* active flexion in the modified fist position and tenodesis with MCP extension will promote differential excursion for the long tendons and intrinsics.

Zone V

The zone V level includes the area proximal to the carpal tunnel to the musculotendinous junction in the distal third of the forearm. This level is susceptible to combined injuries and is often termed "spaghetti wrist or full house" and may involve as many as nine digital flexors, three wrist flexors, two major nerves, and two major arteries. Clinical problems at this level include reestablishing differential tendon glide, particularly of the FDS, and problems related to injury and repair of nerve and vessel, which would alter exercise patterns in the early stages. *Orthotic geometry and patterns of motion* are altered depending on nerve and wrist flexor tendon repair but should emphasize independent digital passive extension to minimize intertendinous adhesion. Although repairs at this level can be expected to recover much of the composite grip, independent glide, especially with the FDS, may not be reestablished; ulnar intrinsic function and ulnar nerve sensibility will produce the most disappointing results.[141,142]

Flexor Pollicis Longus

Rehabilitation of the FPL in zones T1 and T2 is more difficult than that at the same level in the digits. This repair is often under more tension because proximal retraction is greater for FPL than for digital tendons, making apposition of the tendon ends difficult with primary repair and causing a high risk of rupture. Results will be poorer with retraction or injury to the neurovascular bundles. Rupture rates in zone TI have been found to be similar to those in the digits, whereas in zone TII, rupture rates have been

reported at 3%, 5%, and 8% with techniques of early passive motion and at 17%, 15%, and 8% with early active mobilization.[143]

Orthotic Geometry

Improved results have been reported with a decrease in rupture by including all digits in the dorsal hood orthosis to prevent inadvertent opposition and by positioning the wrist in 10° of flexion and 10° of ulnar deviation.[144] I prefer to position the wrist at 10–20° of flexion, the CMC and MCP joints in neutral position, and the IP joint dorsally blocked at 20–25° with dynamic traction from the base of the small finger. I include only index finger with dorsal orthotic protection to prevent inadvertent pinch and to protect FPL from forces associated with an anomalous tendon slip to the index FDP.[145] It has been demonstrated that the FPL excursion is increased by 70% if the MCP is neutral with IP flexion.[129]

Patterns of Motion

I prefer to use wrist tenodesis only under therapist supervision; gentle active tendon gliding most specifically for the index FDP and FDS; and after passive exercise, a *place-and-hold* active tension with the wrist extended to 10° *only* if the repair has the strength of at least a four-strand core and epitendinal suture. The more proximal zones would be treated in the same manner, with active tension.

In general, splinting can be altered for *all zones* at four weeks postoperatively to place the wrist at 0–20° extension except with associated injury to nerve or wrist flexor tendons; dynamic extension can be added by weeks 5–6 with the wrist in neutral position for cases with dense adhesion. Increased resistance can be applied as described in a recent study that defines a pyramid of eight levels of exercise that can be applied based on calculations of tendon healing and tensile strength.[146] Freely gliding tendons should be protected two to three weeks longer than the norm, and adherent tendons can be subjected to increased external forces. Internal forces can be reduced by preceding active tension with passive motion, positioning the wrist in neutral position for active hold, especially if the FDS is repaired, and with a *slow rate of flexion*. Joint blocking in the more distal zones should be done with lateral support for P^1 and P^2 as volar support increases resistance to tendon glide. Strict attention to complete PIP joint extension is a critical rehabilitation component from the first postoperative week. Patients should not return to normal activity until 12 weeks, and I would not test grip strength on any tendon until after that time frame for fear of rupture.

Extensor System

Zones I and II

In general, the zone I and II injury or mallet finger requires at least six weeks of continuous immobilization with the DIP joint splinted at absolute 0° of extension or slight hyperextension of no more than 15°. I often immobilize these injuries up to 10 weeks if the terminal tendon is weak or if any lag exists. The volar P^2, P^3 orthosis can be interlocked with a dorsal P^1, P^2, P^3 orthosis that holds the PIP in about 40° of flexion to relax the lateral bands during the first four to six weeks to prevent a swan posture and to reduce tension on the terminal tendon. PIP joint flexion exercises are unrestrained. The key components of treating this injury include precision splinting, management of skin integrity and hypersensitivity, and carefully controlled progressive SAM for distal joint flexion when healing occurs. Insufficient evidence exists to support customized or "off the shelf" splinting or to determine when surgery is indicated.[147]

Zones III and IV

Immediate active SAM for the repaired central slip or zone III or IV extensor injury is defined in a study of excursion, internal tendon forces, and anatomical considerations,[36] and is supported by clinical results.[37] Untreated, the injured central slip will progress to a fixed boutonnière deformity with palmar migration of the lateral bands, hyperextension of the DIP joint, and flexion deformity of the PIP joint. Complications of traditional treatment of these injuries, which typically have been treated with uninterrupted immobilization for four to six weeks before motion was initiated, include extensor lag, insufficient extensor tendon excursion with loss of flexion, and joint stiffness. These factors negatively influence the final outcome of the acutely repaired and immobilized central slip injury: 1) the broad tendon–bone interface in zone IV; 2) resting the tendon at less than absolute 0° of extension during immobilization; and 3) the effects of stress deprivation on the connective tissues (tendon, ligament, and cartilage) of the PIP joint. With late mobilization schedules, the zone IV tendon, devoid of the benefits of early motion, is often adherent to the proximal phalanx. As PIP flexion is initiated, the more proximal portion of the tendon is nongliding, elevating tension at the repair site. The repaired zone III portion of the tendon is exposed to excessive stress and begins to elongate or gap leading to a progressive loss of PIP joint extension and the development of a boutonnière deformity. The SAM program addresses these problems. Immediate SAM of 30° at the PIP joint imposes approximately 300 g of internal tension to the central slip and 3.75 mm of excursion at this level and

prevents these complications. Anatomic influences are analyzed in detail in the article supporting SAM,[36] describing the effect of sagittal band migration with MCP motion and contributions of the interossei and lumbricals on central slip tension.

Orthotic Geometry

Anatomical and clinical work support orthosis immobilization only for the digit with the PIP and DIP joints at absolute 0° of extension.

Patterns of Motion

The PIP joint is actively moved through a range of 30° from flexion to extension with the wrist in 20° of flexion and the MCP joint at 0° of extension in the first three postoperative weeks. With lateral band repair, the DIP joint can be moved to 30° if the PIP joint is held in full extension; with no repair, the DIP joint may be flexed to end range. Volar template orthoses are used during exercise to insure precision with SAM exercise. Parameters of motion are gradually increased in weeks 3–6 if no extensor lag develops; by week 6, the PIP should be moving approximately 0–80°. In my initial study, when compared with three to six weeks of continuous immobilization, SAM produced statistically improved results with regard to extensor lag, total active motion, and treatment time.[37] This specific protocol for early motion of the repaired central slip is recently supported by other investigators.[38]

Zones V and VII

I prefer to treat the repaired EDC in zones V and VI with dynamic splinting, which allows approximately 30–35° of motion for the index and long digits and 40–45° of motion for the ring and small digits,[7,84] effecting approximately 5 mm of excursion.[84] This motion is supplemented in therapy with a modified wrist and digital tenodesis, and "place-and-hold" active extension to insure true proximal tendon migration. Newport and Shukla[42] have demonstrated that these tendons are moving actively within the confines of dynamic traction; hence, "passive" protocols with traction are probably a moot point. The digital joints must rest in full extension within the orthosis to prevent extensor lag. Wrist position should be neutral. It has been demonstrated that if the wrist is extended more than 21°, the extensors glide with little or no tension in zones V and VI throughout full simulated grip to full passive extension.[41] Howell et al.[39] have used an immediate controlled active motion program for these zones since the 1980s with excellent clinical results. Their orthotic design positions the involved digit (digits) in "relative hyperextension" of 15–20° to the uninjured digits to relieve stress at the repair site. The design has two components: a simple yoke orthosis to control the MCP joints and a second wrist control orthosis. Their technique is supported by intraoperative measurement, clinical experience, and a recently published biomechanical study.[40] Their results are impressive.

Zone VII

Orthotic geometry and *patterns of motion* in zone VII are similar to those in zones V and VI, and early motion is especially critical as the tendons are synovial at this level. Wrist position should be in at least 40–45° of extension if the wrist extensor tendons are involved. Attention to differential gliding for the EDC and modest wrist flexion to about 20° with the digits held in active extension will minimize tendon-to-tendon adherence in the fourth dorsal compartment if the wrist tendons are uninvolved. Chinchalkar and Yong[43] have described a technique for encouraging relative excursion of repaired tendons in zones VI through VIII, which they term "double reverse Kleinert" technique. This clever dynamic orthosis promotes differential tendon gliding to control intertendinous adhesions as they are formed in the early postoperative phase. This design encourages both independent and combined wrist and digital motion.

Extensor Pollicis Longus

Zones TI–V

Zone I EPL repairs should be treated as mallet injuries for the digits, with P^1 and P^2 continuous splinting in slight hyperextension; zone TII with a hand-based orthosis and SAM of the IP to about 15° in the early phase; and zone TIII–IV as a boutonnière, again with a hand-based orthosis with all thumb joints in extension. The zone TV repair, which is at the retinacular level, will be subject to the development of dense adhesion. I now prefer a simple long opponens static orthosis with careful attention to the MCP joint position to prevent a position of hyperextension. In therapy, the wrist is moved passively to full extension with manual support of the thumb kinetic chain; the IP is moved to 60° with the wrist and CMC and MCP joints in extension to effect 5 mm of glide at Lister's tubercle;[84] and then, with the wrist in 20° of flexion, the EPL is activated with an active "place and hold" in extension. Dynamic traction can be set up with the wrist in mild extension, CMC and MCP joints at 0° of extension, and an allowed 60° of flexion at the IP. I have found, however, that this orthosis is somewhat cumbersome and that my results are equal with long opponens static splinting.

The principles of postoperative extensor tendon treatment are simple. Initiate treatment by postoperative day

3, orthosis the repair site proximal to its normal resting position, and control tendon excursion with an orthotic design that allows at least 5 mm (or more) of motion at the repair site for zones III–VI, TIV, and V.

SUMMARY

The search for a solution for reestablishing functional motion in a repaired tendon is ongoing and "although biologic and biochemical modifications have shown great promise in animal models, they have not yet been tested clinically."[14] In the future, repair-site modifications may include the local delivery of growth factors to stimulate extracellular matrix formation and prevent adhesion formation, and biochemical tendon surface treatments to reduce frictional resistance. At this time, however, the stronger repair techniques and the application of controlled stress remain the best and most well-supported interventions after tendon injury and repair. The hand therapist's role in this process remains a critical component contributing to satisfactory outcome.

REFERENCES

1. Evans RB. Early active motion after flexor tendon repair. In: Berger RA, Weiss A-P (eds). Hand Surgery, vol. 1. Philadelphia, PA: Lippincott Williams and Wilkins, 2004. pp. 709–35.
2. Goodman HJ, Choueka J. Biomechanics of flexor tendons. Hand Clin. 2005;21:129–49.
3. Allan CH. Flexor tendons: anatomy and surgical approaches. Hand Clin. 2005;21:151–7.
4. Taras JS, Martyak GG, Steelman PJ. Primary care of flexor tendon injuries. In: Skirven TM, Osterman AL, Fedorczyk JM, Amadio PC (eds). Rehabilitation of the Hand and Upper Extremity. 6th ed. Philadelphia, PA: Elsevier, 2011. pp. 445–56.
5. Pettengill K, Van Strien G. Postoperative management of flexor tendon injuries. In: Skirven TM, Osterman AL Fedorczyk JM, Amadio PC (eds). Rehabilitation of the Hand and Upper Extremity. 6th ed. Philadelphia, PA: Elsevier, 2011. pp. 457–78.
6. Rosenthal EA, Elhassan BT. The extensor tendons: evaluation and surgical management. In: Skirven TM, Osterman AL, Fedorczyk JM, Amadio PC (eds). Rehabilitation of the Hand and Upper Extremity. 6th ed. Philadelphia, PA: Elsevier, 2011. pp. 487–520.
7. Evans RB. Clinical management of extensor tendon injuries: the therapist's perspective. In: Skirven TM, Osterman AL, Fedorczyk JM, Amadio PC (eds). Rehabilitation of the Hand and Upper Extremity. 6th ed. Philadelphia, PA: Elsevier, 2011. pp. 521–54.
8. Beredjiklian PK. Biologic aspects of flexor tendon laceration and repair. J Bone Joint Surg Am. 2003;85-A:539–50.
9. Khan U, Occleston NL, Kwaw PT, et al. Differences in proliferative rate and collagen lattice contraction between endotenon and synovial fibroblasts. J Hand Surg Am. 1998;23A: 266–73.
10. Strickland JW. The scientific basis for advances in flexor tendon surgery. J Hand Ther. 2005;18:94–110.
11. Tang JB. Tendon injuries across the world: treatment. Injury. 2006;37:1036–42.
12. Khanna A, Friel M, Gougoulias N, Long UG, Mafulli N. Prevention of adhesions in surgery of the flexor tendons of the hand: what is the evidence? Br Med Bull. 2009;90:85–109.
13. Khanna A, Gougoulias N, Maffulli N. Modalities in prevention of flexor tendon adhesion in the hand: what have we achieved so far? Acta Orthop Belg. 2009;75:433–44.
14. Kim HM, Nelson G, Thomopoulos S, Silva MJ, Das R, Gelberman RH. Technical and biological modifications for enhanced flexor tendon Repair. J Hand Surg. 2010;35A:1031–7.
15. Elliot D, Barbieri CH, Evans RB, Mass D, Tang JB. IFSSH Flexor Tendon Committee Report 2007. J Hand Surg Eur Vol. 2007;32:346–56.
16. Strickland JW. Development of flexor tendon surgery: twenty-five years of progress. J Hand Surg Am. 2000;25: 214–35.
17. Luo J, Mass DP, Phillips CS, He TC. The future of flexor tendon surgery. Hand Clin. 2005;21:267–73.
18. Boyer MI, Goldfarb CA, Gelberman RH. Recent progress in flexor tendon healing. The modulation of tendon healing with rehabilitation variables. J Hand Ther. 2005;18:80–5.
19. Manske PR. History of flexor tendon repair. Hand Clin. 2005; 21:123–7.
20. Pettengill KM. The evolution of early mobilization of the repaired flexor tendon. J Hand Ther. 2005;18:157–68.
21. Burge PD, Brown NM. Elastic band mobilization after tendon repair: splint design and risk of flexion contracture. J Hand Surg Br. 1990;15:443–8.
22. Slattery RG, McGrouther DA. The modified Kleinert controlled mobilization splint following flexor tendon repair. Hand Surg Br. 1984;9:217–8.
23. Werntz JR, Chesher SP, Breiderbach WC, et al. A new dynamic splint for post operative treatment of flexor tendon injury. J Hand Surg Am. 1989;14:559–66.
24. Savage R. The influence of wrist position on the minimum force required for active movement of the interphalangeal joints. J Hand Surg. 1988;13B:262–8.
25. Amadio PC. Friction of the gliding surface: implications for tendon surgery and rehabilitation. J Hand Ther. 2005;18: 112–9.
26. McGrouther DA, Ahmed MR. Flexor tendon excursion in "no man's land". Hand. 1981;13:129–41.
27. Horibe S, Woo SL-Y, Spiegelman JJ, et al. Excursion of the flexor digitorum profundus tendon: a kinematic study of the human and canine digits. J Orthop Res. 1990;8:167–74.
28. Evans RB. A study of the zone I flexor tendon injury and implications for treatment. J Hand Ther. 1990;3:133–48.
29. Silfverskiöld KL, May EJ, Törnvall AH. Tendon excursions after flexor tendon repair in zone. II: Results with a new controlled-motion program. J Hand Surg Am. 1993;18:403–10.
30. Evans RB. Zone I flexor tendon rehabilitation with limited extension and active flexion. J Hand Ther. 2005;18:128–40.
31. Halikis MN, Mankse PR, Kubota H, Aoki M. Effect of immobilization, immediate mobilization, and delayed mobilization on the resistance to digital flexion using a tendon injury model. J Hand Surg Am. 1997;22:464–72.
32. Zhao C, Amadio PC, Paillard P, et al. Digital resistance and tendon strength during the first week after flexor digitorum profundus tendon repair in a canine model in vivo. J Bone Joint Surg Am. 2004;86:320–7.
33. Zhao C, Amadio PC, Tanaka T. Short-term assessment of optimal timing for postoperative rehabilitation after flexor digitorum profundus tendon repair in a canine model. J Hand Ther. 2005;18:322–9.
34. Xie RG, Cao Y, Xu XF, Zhu B. The gliding force and work of flexion in the early days after primary repair of lacerated flexor tendons: an experimental study. J Hand Surg Eur Vol. 2008;33:192–6.
35. Cao Y, Chen CH, Wu YF, Xu XF, Xie RG, Tang JB. Digital oedema, adhesion formation and resistance to digital motion after primary flexor tendon repair. J Hand Surg Eur Vol. 2008; 33:745–52.
36. 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.
37. Evans RB. Early active short arc motion for the repaired central slip. J Hand Surg. 1994;19A:991–7.

38. Kalb K, Prommersbersger KJ. Experiences with "short arc motion" protocol for rehabilitation of extensor tendon injuries in zones E and F according to Wilhelm. Handchir Mikrochir Plast Chir. 2008;40:165–8.
39. Howell JW, Merritt WH, Robinson SJ. Immediate controlled active motion following zone 4-7 extensor tendon repair. J Hand Ther. 2005;18:182–90.
40. Sharma JV, Liang NJ, Owen JR, Wayne JS, Isaacs JE. Analysis of relative motion splint in the treatment of zone VI extensor tendon injuries. J Hand Surg Am. 2006;31:1118–22.
41. Minamikawa Y, Peimer CA, Yamaguchi T, et al. Wrist position and extensor tendon amplitude following repair. J Hand Surg. 1992;17A:268.
42. Newport ML, Shukla A. Electrophysiologic basis of dynamic extensor splinting. J Hand Surg. 1992;17A:272.
43. Chinchalkar S, Yong SA. A double reverse Kleinert extension splint for extensor tendon repairs in zones VI to VIII. J Hand Ther. 2004;17:424–6.
44. Eissens MH, Schut SM, van der Sluis CK. Early active wrist mobilization in extensor tendon injuries in zones 5, 6, or 7. J Hand Ther. 2007;20:89–91.
45. Talsma E, de Haart M, Beelen A, et al. The effect of mobilization on repaired extensor tendon injuries of the hand: a systematic review. Arch Phys Med Rehabil. 2008;89:2366–72.
46. Leigh DR, Abreu EL, Derwin KA. Changes in gene expression of individual matrix metalloproteinases differ in response to mechanical unloading of tendon fascicles in explants culture. J Orthop Res. 2008;26:1306–12.
47. Buckwalter JA, Grodzinsky AJ. Loading of healing bone, fibrous tissue and muscle: implications for orthopaedic practice. J Am Acad Orthop Surg. 1999;7:291–9.
48. Evans RB, Thompson DE. The application of stress to the healing tendon. J Hand Ther. 1993;6:262.
49. Su BW, Protopsaltis TS, Koff MF, et al. The biomechanical analysis of a tendon fixation device for flexor tendon repair. J Hand Surg Am. 2005;30:237–45.
50. Miller B, Dodds SD, deMars A, Zagoreas N, Waitayawinyu T, Trumble TE. Flexor tendon repairs: the impact of fiberwire on grasping and locking core sutures. J Hand Surg Am. 2007;32: 591–6.
51. Lee SK, Dubey A, Kim BH, Zingman A, Landa J, Paksima N. A biomechanical study of extensor tendon repair methods: introduction to the running-interlocking horizontal mattress extensor tendon repair technique. J Hand Surg Am. 2010;35: 19–23.
52. Hirpara KM, Sullivan PJ, O'Sullivan ME. A new barbed device for repair of flexor tendons. J Bone Joint Surg Br. 2010; 92:1165–70.
53. Peltz TS, Haddad R, Scougall PJ, Nicklin S, Walsh WR. Influence of locking stitch size in a four-strand cross-locked cruciate flexor tendon repair. J Hand Surg. 2011;36A:450–5.
54. Henderson J, Sutcliffe M, Gillespie P. The tension band principle and angular testing of extensor tendon repairs. J Hand Surg Eur Vol. 2011;36:297–302.
55. Wu YF, Cao Y, Zhou YL, Tang JB. Biomechanical comparisons of 4-strand tendon repairs with double-stranded sutures: effects of different locks and suture geometry. J Hand Surg (Eur). 2011;36:34–9.
56. McDonald E, Gordon JA, Buckley JM, Gordon L. Comparison of a new multifilament stainless steel suture with frequently used sutures for flexor tendon repair. J Hand Surg Am. 2011;36:1028–34.
57. Huang D, Balian G, Chhabra AB. Tendon tissue engineering and gene transfer: the future of surgical treatment. J Hand Surg Am. 2006;31:693–704.
58. Thorfinn J, Saber S, Angelididis IK, et al. Flexor tendon tissue engineering; temporal distribution of donor tenocytes versus recipient cells. Plast Reconstr Surg. 2009;124:2019–26.
59. Sun H, Liu W, Zhou G, Zhang W, Cui L, Cao Y. Tissue engineering of cartilage, tendon and bone. Front Med. 2011;5:61–9.
60. Shearn JT, Kinneberg KR, Dyment NA, et al. Tendon tissue engineering: progress, challenges and translation to the clinic. J Musculoskelet Neuronal Interact. 2011;11:163–73.
61. Gott M, Ast M, Lane LB, et al. Tendon phenotype should dictate tissue engineering modality in tendon repair: a review. Discov Med. 2011;12:75–84.
62. Chang J, Thunder R, Most D, Longaker MT, Leneaweaver WC. Studies in flexor tendon wound healing: neutralizing antibody to TGF-beta 1 increases postoperative range of motion. Plast Reconstr Surg. 2000;105:148–55.
63. Mehta V, Mass D. The use of growth factors on tendon injuries. J Hand Ther. 2005;18:87–92.
64. Klass BR, Rolfe KJ, Grobbelaar AO. In vitro flexor tendon cell response to TGF-ß1: a gene expression study. J Hand Surg. 2009;34A:495–503.
65. Wong JK, Lui YH, Kapacee Z, Kadler KE, Ferguson MW, Mcgrouther DA. The cellular biology of flexor tendon adhesion formation: an old problem in a new paradigm. Am J Pathol. 2009;175:1938–51.
66. Chen CH, Zhou YL, Wu YF, Cao Y, Gao JS, Tang J. Effectiveness of microRNA in down-regulation of TGF-beta gene expression in digital flexor tendons of chickens: in vitro and in vivo study. J Hand Surg Am. 2009;34:177–84.
67. Sun Y, Chen MY, Zhao C, An KN, Amadio PC. The effect of hyaluronidase, phospholipase, lipid solvent and trypsin on the lubrication of canine flexor digitorum profundus tendon. J Orthop Res. 2008;2:1225–9.
68. Tang JB, Cao Y, Wu YF, Wang YF, Wang GH. Effect of A2 pulley release on repaired tendon gliding resistance and rupture in a chicken model. J Hand Surg Am. 2009;34:1080–7.
69. Taguchi M, Zhao C, Sun YL, Jay GD, An KN, Amadio PC. The effect of surface treatment using hyaluronic acid and lubricin on the gliding resistance of human extrasynovial tendons in vitro. J Hand Surg Am. 2009;34A:1276–81.
70. Hwang MD, Pettrone S, Trumble TE. Work of flexion related to different suture materials after flexor digitorum profundus and flexor digitorum superficialis tendon repair in zone II: a biomechanical study. J Hand Surg Am. 2009;34:700–4.
71. Xia C, Ding C, Yang X, Sun K, Tian S. Effects of antisense transforming growth factor-beta1 gene transfer on the biological activities of tendon sheath fibroblasts. Orthopedics. 2010;33:33–8.
72. Thomopoulos S, Kim HM, Das R, et al. The effects of exogenous basic fibroblast growth factor on intrasynovial flexor tendon healing in a canine model. J Bone Joint Surg Am. 2010;92:2285–93.
73. Ikeda J, Sun Y-L, An K-N, Amadio PC, Zhao C. Application of carbodiimide derivatized synovial fluid to enhance extrasynovial tendon gliding ability. J Hand Surg Am. 2011;36A: 456–63.
74. Amadio PC. Advances in understanding of tendon healing and repairs and effect on postoperative management. In: Skirven TM, Osterman AL, Fedorczyk JM, Amadio PC (eds). Rehabilitation of the Hand and Upper Extremity. 6th ed. Philadelphia, PA: Elsevier, 2011. pp. 439–44.
75. Lindsay WK, Thompson HG, Walker FG. Digital flexor tendons: an experimental study. Part II. The significance of a gap occurring at the line of suture. Br J Plast Surg. 1960;3:1–9.
76. Seradge H. Elongation of the repair configuration following flexor tendon repair. J Hand Surg Am. 1983;8:182–5.
77. Silfverskiold K, May EJ, Tornvall AH. Gap formation during controlled motion after flexor tendon repair in zone II: a prospective clinical study. J Hand Surg Am. 1992;17:593–9.
78. Gelberman RH, Boyer MI, Brodt MD, et al. The effect of gap formation at the repair site on the strength and excursion of intrasynovial flexor tendons. An experimental study on the early stages of tendon-healing in dogs. J Bone Joint Surg. 1999;81:975–8.
79. Boyer MI, Gelberman RH, Burns ME, Dinopoulos H, Hofem R, Silva MJ. Intrasynovial flexor tendon repair: an experimental study comparing low and high levels of in vivo force during rehabilitation in canines. J Bone Joint Surg. 2001;83A: 891–9.
80. Woo SH, Tsai TM, Kleinert HE, et al. A biomechanical comparison of four extensor tendon repair techniques in zone IV. Plast Reconstr Surg. 2005;115:1674–81.

81. Wolfe SW, Willis AA, Campbell D, Clabeaux J, Wright TM. Biomechanical comparison of the Teno Fix tendon repair device with the cruciate and modified Kessler techniques. J Hand Surg Am. 2007;32:356–66.
82. Aoki M, Manske PR, Pruitt DL, et al. Work of flexion after tendon repair with various suture methods. J Hand Surg Br. 1995;20:310–3.
83. Healy C, Mulhall KJ, Bouchier-Hayes DJ, Kneafsey B. Practice patterns in flexor tendon repair. Ir J Med Sci. 2007;176:41–4.
84. Evans RB, Burkhalter WE. A study of the dynamic anatomy of extensor tendons and implications for treatment. J Hand Surg Am. 1986;11A:774.
85. Newport ML, Williams D. Biomechanical characteristics of extensor tendon suture techniques. J Hand Surg. 1992;17A: 1117–23.
86. Zubović A, Egan C, O'Sullivan M. Augumented (Massachusetts General Hospital) Becker technique combined with static splinting in extensor tendons repairs zones III to VI: functional outcomes at three months. Tech Hand Up Extrem Surg. 2008;12:7–11.
87. Brand PW, Thompson DE, Micks JE. The biomechanics of the interphalangeal joints. In: Bowers WH (ed). The Interphalangeal Joints. New York: Churchill Livingstone, 1987. pp. 21–54.
88. Kwai Ben I, Elliot D. "Venting" or partial lateral release of the A2 and A4 pulleys after repair of zone 2 flexor tendon injuries. J Hand Surg Br. 1998;23:649–54.
89. Paillard PJ, Amadio PC, Zhao C, Zobitz ME, An KN. Pulley plasty versus resection of one slip of the flexor digitorum superficialis after repair of both flexor tendons in zone II: a biomechanical study. J Bone Joint Surg Am. 2002;84-A:2039–45.
90. Dona E, Walsh WR. Flexor tendon pulley V-Y plasty: an alternative to pulley venting or resection. J Hand Surg Br. 2006;31: 133–7.
91. Walbeehm ET, McGrouther DA. An anatomic study of the mechanical interactions of flexor digitorum superficialis and profundus and the flexor sheath in zone 2. J Hand Surg Br. 1995;20:269–80.
92. Sandford F, Barlow N, Lewis J. A study to examine patient adherence to wearing 24-hour forearm thermoplastic splints after tendon repair. J Hand Ther. 2008;21:44–52.
93. Harris SB, Harris D, Foster AJ, Elliot D. The aetiology of acute rupture of flexor tendon repairs in zones 1 and 2 of the fingers during early mobilization. J Hand Surg Br. 1999;24:275–80.
94. Peck FH, Kennedy SM, Watson JS, Lees VC. An evaluation of the influence of practitioner-led hand clinics on rupture rates following primary tendon repair in the hand. Br J Plast Surg. 2004;57:45–9.
95. Brand PW, Thompson DE. Mechanical resistance. In: Brand PW, Hollister A (eds). Clinical Mechanics of the Hand. 2nd ed. St. Louis, MO: Mosby, 1993. pp. 92–1280.
96. Woo SL-Y, Gelberman RH, Cobb NG, et al. The importance of controlled passive mobilization on flexor tendon healing: a biomechanical study. Acta Orthop Scand. 1981;52:615.
97. Gelberman RH, Van deBerg JS, Manske PR, Akeson WH. The early stages of flexor tendon healing: a morphologic study of the first fourteen days. J Hand Surg Am. 1985;10A:776–84.
98. Hitchcock TF, Light TR, Bunch WH, et al. The effect of immediate constrained digital motion on the strength of flexor tendon repairs in chickens. J Hand Surg Am. 1987;12A:590–5.
99. Freehan LM, Beauchene JG. Early tensile properties of healing chicken flexor tendons: early controlled passive motion versus postoperative immobilization. J Hand Surg Am. 1990;15A:63.
100. Rothkopf DM, Webb S, Szabo RM, et al. An experimental model for the study of canine flexor tendon adhesions. J Hand Surg. 1991;16A:694.
101. Amiel D, Gelberman RH, Harwood F, Siegel D. Fibronectin in healing flexor tendons subjected to immobilization or early controlled passive motion. Matrix. 1991;2:184.
102. Amiel D, Woo SL, Harwood FL, et al. The effect of immobilization on collagen turnover in connective tissue: a biochemical-biomechanical correlation. Acta Orthop Scand. 1982;53:325.
103. Buckwalter JA. Effects of early motion on healing of musculoskeletal tissues. Hand Clin. 1996;12:13.
104. Aoki M, Kubota H, Pruitt D, et al. Biomechanical and histological characteristics of canine flexor tendon repair using early postoperative mobilization. J Hand Surg Am. 1997;22:107–14.
105. Gelberman RH, Steinberg D, Amiel D, et al. Fibroblast chemotaxis after repair. J Hand Surg. 1991;16A:686.
106. Becker H, Diegelman RF. The influence of tension on intrinsic tendon fibroplasia. Orthop Rev. 1984;13:65–71.
107. Fong KD, Trindade MC, Wang Z, et al. Microarray analysis of mechanical shear effects on flexor tendon cells. Plast Reconstr Surg. 2005;116:1393–404.
108. Maeda T, Sakabe T, Sunaga A, et al. Conversion of mechanical force into TGF-β-mediated biochemical signals. Curr Biol. 2011;21:933–41.
109. Shearn JT, Juncosa-Melvin N, Boivin GP, et al. Mechanical stimulation of tendon tissue engineered constructs: effects on construct stiffness, repair biomechanics, and their correlation. J Biomech Eng. 2007;129:848–54.
110. Abousleiman RI, Reyes Y, McFetridge P, Sikavitas V. Tendon tissue engineering using cell-seeded umbilical veins cultured in a mechanical stimulator. Tissue Eng Part A. 2009;15:787–95.
111. Joshi SD, Webb K. Variation of cyclic strain parameters regulates development of elastic modulus in fibroblast/substrate constructs. J Orthop Res. 2008;26:1105–13.
112. Asundi KR, Rempel DM. MMP-1, IL-1beta, and COX-2 mRNA expression is modulated by static load in rabbit flexor tendons. Ann Biomed Eng. 2008;36:237–43.
113. Kubota H, Manske PR, Aoki M, et al. Effect of motion and tension on injured flexor tendons in chickens. J Hand Surg Am. 1996;21A:456–63.
114. Duran RJ, Houser RG. Controlled passive motion following flexor tendon repair in zones II and III. In: Hunter JM, Schneider LH (eds). Symposium on Tendon Surgery in the Hand. St. Louis, MO: C.V. Mosby, 1975. pp. 105–14.
115. Horii E, Lin GT, Cooney WP, et al. Comparative flexor tendon excursion after passive mobilization: an in vitro study. J Hand Surg Am. 1992;17:559–66.
116. Hagberg L, Selvik G. Tendon excursion and dehiscence during early controlled mobilization after flexor tendon repair in zone II: an x-ray stereophotogrammetric analysis. J Hand Surg Am. 1991;16:669–80.
117. Nessler JP, Amadio PC, Bergland LJ, et al. Healing of canine tendon in zones subjected to different mechanical forces. J Hand Surg Br. 1992;17:561–8.
118. Lane JM, Black J, Bora FW. Gliding function following flexor tendon injury: a biomechanical study of rat tendon function. J Bone Joint Surg. 1976;58A:985.
119. Gelberman RH, VandeBerg JS, Lundborg GN, et al. Flexor tendon healing and restoration of the gliding surface: an ultrastructural study in dogs. J Bone Joint Surg. 1983;65A:70.
120. Iwuagwu FC, McGrouther DA. Early cellular response in tendon injury: the effect of loading. Plast Reconstr Surg. 1998;102: 2064.
121. Mason ML, Allen HS. The rate of healing tendons: an experimental study of tensile strength. Ann Surg. 1941;113:424.
122. Silva MJ, Brodt MD, Boyer MI, et al. Effects of increased in vivo excursion on digital range of motion and tendon strength following flexor tendon repair. J Orthop Res. 1999; 17:777–83.
123. Tottenham VM, Wilton-Bennett K, Jeffrey J. Effects of delayed therapeutic intervention following zone II flexor tendon repair. J Hand Ther. 1995;8:23–6.
124. Gelberman RH, Nunley JA, Osterman AL, et al. Influences of the protected passive mobilization interval on flexor tendon healing: a prospective randomized clinical study. Clin Orthop. 1991;264:189–96.
125. Takai S, Woo SL, Horibe S, et al. The effects of frequency and duration of controlled passive mobilization on tendon healing. J Orthop Res. 1991;9:705–13.
126. Gelberman RH, Botte MJ, Spiegelman JJ, et al. The excursion and deformation of repaired flexor tendons treated with protected early motion. J Hand Surg Am. 1986;11:106–10.
127. Cooney WP, Lin GT, An KN. Improved tendon excursion following flexor tendon repair. J Hand Ther. 1989;2:102–6.

128. Wehbe MA, Hunter JM. Flexor tendon gliding in the hand. II. Differential gliding. J Hand Surg Am. 1985;10:575–9.
129. Brown CP, McGrouther DA. The excursion of the tendon of flexor pollicis longus and its relation to dynamic splintage. J Hand Surg Am. 1984;9:787–91.
130. Brand PW, Hollister A. Clinical mechanics of the hand. 2nd ed. St Louis, MO: Mosby, 1993.
131. Browne EZ, Ribik CA. Early dynamic splinting for extensor tendon injuries. J Hand Surg Am. 1989;14A:72.
132. Williams RJN, Amis AA. A new type of flexor tendon repair: biomechanical evaluation by cyclic loading, ultimate strength and assessment of pulley friction in vitro. J Hand Surg Br. 1995;20:578–83.
133. Wainright A, Briggs WD, Currey JD, et al. Mechanical Design in Organism. Princeton, NJ: Princeton University Press, 1976. 23–29.
134. Urbaniak JR, Cahill JD, Mortenson RA. Tendon suturing methods: analysis of tensile strengths. In: Hunter JM, Schneider LH (eds). Symposium on Tendon Surgery in the Hand. St. Louis, MO: Mosby, 1975. pp. 70–80.
135. Schuind F, Garcia Elias M, Cooney WP, et al. Flexor tendon forces: in vivo measurements. J Hand Surg Am. 1992;17:291–8.
136. Llorens WL. An experimental Analysis of Finger Joint Stiffness. Baton Rouge, LA: Louisiana State University, 1986. (MSME thesis).
137. Arbuckle JD, McGrouther DA. Measurement of the arc of digital flexion and joint movement ranges. J Hand Surg. 1995; 20B:836–40.
138. Long C, Conrad PW, Hall EA, Furler SL. Intrinsic-extrinsic muscle control of the hand in power grip and precision handling. An electromyographic study. J Bone Joint Surg Am. 1970;52:853–67.
139. Smith RJ. Balance and kinetics of the fingers under normal and pathologic conditions. Clin Orthop. 1974;92:104–8.
140. Tang JB, Xu Y, Wang B. Repair strength of tendons of varying gliding curvature: a study in a curvilinear model. J Hand surg. 2003;28A:243–9.
141. Widgerow AD. Full-house/spaghetti wrist injuries: analysis of results. S Afr J Surg. 1990;28:6–10.
142. Hudson DA, de Jager LT. The spaghetti wrist. Simultaneous laceration of the median and ulnar nerves with flexor tendons at the wrist. J Hand Surg Br. 1993;18:171–3.
143. Elliot D, Moiemen NS, Fleming AFS, et al. The rupture rate of acute flexor tendon repairs mobilized by the controlled active regimen. J Hand Surg Br. 1994;19:607–12.
144. Elliot D, Southgate CM. New concepts in managing the long tendons of the thumb after primary repair. J Hand Ther. 2005; 18:141–56.
145. Karalezli N, Karakose S, Haykir R, et al. Linburg-Comstock anomaly in musicians. J Plast Reconstr Aesthet Surg. 2006; 59:768–71.
146. Groth GN. Pyramid of progressive force exercises to the injured flexor tendon. J Hand Ther. 2004;17:31–42.
147. Handoll HH, Vaghela MV. Interventions for treating mallet finger injuries. Cochrane Database Syst Rev. 2004;3: CDOO4574.

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#1. The author believes that the future will see
   a. lower rupture rates
   b. higher rupture rates
   c. the end of splinting during early rehabilitation
   d. greater allowed ranges of motion with greater loads during early rehabilitation

#2. Current approaches to rehabilitation are primarily designed to
   a. decrease the risk of PIP flexion contracture
   b. break adhesions
   c. promote intrinsic healing
   d. balance the contributions of the FDP and FDS

#3. Following flexor tendon repair the rehab community
   a. has not agreed on "the best system of stress application"
   b. has agreed on "the best system of stress application"
   c. has agreed on "the best system of passive digital traction"
   d. is abandoning passive digital traction

#4. The concept of a "Safety Zone" following tendon repair refers to
   a. the tension on elastic traction built into the protective splint
   b. the tolerance a repaired tendon has for load and excursion without excessive gapping
   c. Zone II
   d. Zones II and IV

#5. Recent improvements in surgical technique and early mobilization in therapy have resulted in consistent results in outcomes following primary repair
   a. true
   b. false

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