An Analysis of Factors That Support Early Active Short Arc Motion of the Repaired Central Slip

Roslyn B. Evans, OTR/L, CHT, BS
Director,
Indian River Hand Rehabilitation, Inc.,
Vero Beach, Florida

David E. Thompson, PhD
Professor, Mechanical Engineering,
Louisiana State University,
Baton Rouge, Louisiana

ABSTRACT: This study defines precise parameters for tendon excursion, force application, and exercise position for an early active short arc motion protocol for the repaired central slip. Recommended active excursion for the extensor digitorum communis (ED) in zone III is 3.75 mm during the early healing phase. Based on the radian concept, the proximal interphalangeal joint (PIP) is actively flexed and extended 28.65° (approximately 30°) or one-half radian to effect this tendon excursion. Resistance applied to the central slip with active extension from 30° to 0° is calculated mathematically at approximately 290 g of force. Tensile strengths for various repairs are reviewed to establish the safety margin between tensile strength of the repair site and force application. Force application for the active extension protocol is considered in terms of anatomic position. The position of wrist flexion at 30° reduces ED work requirement 1) by reducing viscoelastic flexor forces and 2) through a contribution from the interossei. The position of MP at 0° 1) transmits extensor forces to the central slip and 2) reduces ED work requirement through lumbrical and interossei action. The distal interphalangeal joint (DIP) is unrestrained during PIP flexion to allow volar slide of the lateral bands. Isolated DIP exercises with the PIP held at 0° creates a distal glide of the ED in zone IV while reducing tensions in zone III through the action of the lateral bands. The short arc motion protocol as defined in this paper and supported by a companion clinical study is safe and physiologically desirable as determined by this study.

The wound healing process of tendon has been the focus of intense basic science research for several decades.[1-15] Predictably, our knowledge of this process will expand in the next decade as we study the role of growth factors, interleukin I, and other biochemical signals responsible for the intrinsic mechanism of tendon repair.[16-20] The experimental research that compares the cellular and molecular activity of the repaired and immobilized tendon with that of the mobilized tendon has defined clinical application for hand surgeons and hand therapists.[21-28]

Many elegant studies have demonstrated the positive influence of stress on healing tendon, with documented improvement in tensile strength, gliding properties, increased repair-site DNA, and accelerated changes in peritendinous vessel density and configuration.[5,29-41] Recent studies have demonstrated that early passive motion in a clinically relevant tendon repair model increases fibronectin concentration[42] and fibroblast chemotaxis[43] at the repair site. Fibronectin concentration in mobilized tendon is directly related to fibroblast chemotaxis and was found to be twice that of immobilized tendon by seven days post repair.[42,43] These new findings support the work of Hitchcock et al.[35] and that of Freehan and Beauchene,[29] which demonstrate the relationship of timing and motion to tendon wound activity and suggest that immediate or very early motion may actually eliminate the decrease in tensile strength noted postoperatively in the classic study by Mason and Allen.[44]

Gelberman and associates have demonstrated in a randomized clinical study that the duration of the daily controlled motion interval is a significant factor in the final outcome of zone II flexor tendon repair.[45] Their conclusion that "more is better" may apply to all tendons treated with early motion.

This report is a companion study (part I) to a clinical study (part II). Part I was presented at the Scientific Session of the American Society of Hand Therapists annual meeting in Phoenix, Arizona, November 5–9, 1992. Part II was presented at the Scientific Session of the American Society for Surgery of the Hand annual meeting in Phoenix, Arizona, November 14, 1992.

Correspondence and reprint requests to Roslyn B. Evans, OTR/L, CHT, BS, Director, Indian River Hand Rehabilitation, Inc., 777 37th Street, Suite D101, Vero Beach, FL 32960.

The authors have received no grant money for this study, nor any financial benefit from the publication or presentation of this work.

The evolution of rehabilitation from immobilization to early passive motion and then to early active motion is a continuum, with each new study on tendon healing, nutrition, excursion, repair, and rehabilitation technique having as its goal the re-establishment of functional tendon glide.

In the clinical spectrum of tendon management, much of this basic science research has been applied to the flexor system, especially synovial tendon in zone II, where tendon glide is most difficult to re-establish (Fig. 1).[21–27,45–50]

For some time, but with less clinical support and attention, the techniques of early motion have been applied to the extensor tendons in zones V, VI, and VII, with improved results (Fig. 2).[50–61]

The concept of early motion for the repaired central slip in extensor zone III or its more proximal counterpart in zone IV, less well accepted and with less consistent success, has been described in the literature[52,60,62] and through professional presentations[63–68] for some time as well, but for the most part, tendon injuries in zones III and IV continue to be treated by most clinicians with four to six weeks of immobilization.[69–78]

The vast bodies of research on the benefits of early motion and tendon healing have by and large not been applied to one of our most vexing and complex tendon management problems, the dorsal digital extensor system.

Recently, early motion for extensor injuries at this level has received more attention. However, protocols for splinting, exercise, and timing vary from study to study, as do methods of patient selection and results. Some clinicians report excellent results,[63–65,67] others report inconsistent results with early passive motion,[52,60,62,68] but none have provided a clear definition of the force application or the excursion limits necessary to scientifically stress the repaired central slip.

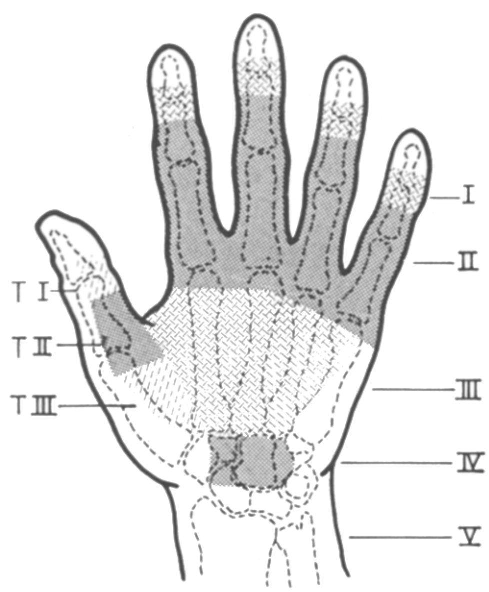

FIGURE 1. Flexor tendon zones as defined by the Committee on Tendon Injuries for the International Federation of the Society for Surgery of the Hand. Reprinted with permission from: Kleinert HE, Schepel S, Gill T: Flexor tendon injuries. Surg Clin North Am 61:267–286, 1981.

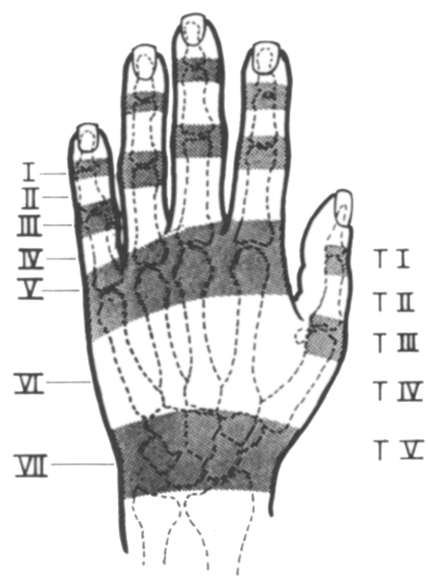

FIGURE 2. Extensor tendon zones as defined by the Committee on Tendon Injuries for the International Federation of the Society for Surgery of the Hand. Reprinted with permission from: Kleinert HE, Schepel S, Gill T: Flexor tendon injuries. Surg Clin North Am 61:267–286, 1981.

The purpose of this study is to provide a detailed analysis of the pertinent dynamic anatomy and the biomechanics and biochemistry of the proximal interphalangeal (PIP) joint in support of immediate active short arc motion following repair of the zone III extensor injury. This report defines safe parameters for tendon excursion and force application to assist the hand surgeon and hand therapist in the application of physiologic stress to the healing extensor tendon in zone III.

This work is a companion study (part I) to a clinical study (part II) that reports statistically superior results in terms of extensor lag, total active motion, and treatment time for repaired central slip injuries treated with immediate active short arc motion as compared with those treated with four to six weeks of immobilization (Table 1).[79]

DEFINING THE PROBLEM

The natural progression of the zone III extensor injury is well defined in the literature.[70–72,76,77,80–90] Untreated, the lacerated middle band will retract, allowing the lateral bands to carry the full force of the extrinsic extensor tendon. The lateral bands migrate palmarwards, act as flexors of the PIP joint, and with an increase in effort to extend the proximal joint, actually hyperextend the distal interphalangeal (DIP) joint. The tendon and retinacular tissues tighten and accommodate to change in joint posture over time; both tendon and retinacular ligaments shorten and begin to resist even passive correction of the deformity.[91]

The acutely repaired central slip injury treated traditionally with four to six weeks of immobilization

TABLE 1. **Clinical Results of Open and Repaired Central Slip Injuries***

	GROUP I† *(Immobilization)*	*GROUP II†* *(SAM)*	*Statistical Significance* *(t Test)*	**Statistical Significance (Chi Square)**
Number of digits	38	26		
Age—mean	39.9 years	42.2 years	>0.5 NS	
Gender—male	86.8%	80.8%		>0.5 NS
Complex injury‡	76.3%	76.9%		>0.5 NS
Day motion initiated—mean	32.9	4.59	<0.001 S	
Day injury to D/C§—mean	76.07	51.38	<0.001 S	
Proximal interphalangeal extension lag on first motion day	13°	3°	<0.01 S	
Proximal interphalangeal extension lag on D/C day	8.13°	2.96°	<0.01 S	
Proximal interphalangeal motion at 6 weeks	44°	88°	<0.001 S	
Proximal interphalangeal motion at D/C	72°	88°	<0.01 S	
Total active motion proximal and distal interphalangeal at D/C	110.7°	131.5°	<0.01 S	
Distal interphalangeal motion at D/C	37.63°	45°	<0.01 S	

*As analyzed by the Department of Orthopedics Medical Statistician, University of Miami. From: Evans RB: Early active short arc motion for the repaired central slip. Part II. Phoenix, AZ, American Society for Surgery of the Hand Scientific Session, November 14, 1992.

†Group I was treated with 3–6 weeks of immobilization, group II with immediate active short arc motion (SAM) of 30°.

‡Simple injuries are defined as tendon or tendon with associated lateral band injuries; complex injuries as those central slip injuries with associated injury to bone, cartilage, or distal joint.

§D/C = discharge day.

is often compromised by problems of extensor tendon lag, insufficient extensor tendon excursion, joint stiffness, and loss of flexion. Newport et al., in a recent report of long-term results of extensor tendon repair, found that extensor tendon injuries within the digit treated with immobilization had high percentages of fair and poor results as compared with those of more proximal injuries, and that injuries in zones III and IV had higher percentages of resultant extensor lag (35%) and loss of flexion (71%).[92] They note that there is little margin for adhesion formation or shortening of the extensor tendon on the dorsum of the digit if a reasonable result is to be obtained.[92] Verdan observed that extensor injury over the proximal phalanx produced the worst results[77]; Lovett and McCalla found the highest percentages of extensor lag in zones III and IV.[74]

The solution to the problem of the dorsal tendon injury is found in the definition of the problem. Clinical observation and literature review indicate that the following factors 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 functional length during immobilization; and 3) the effects of stress deprivation on the connective tissue (tendon, cartilage, ligament) of the PIP joint.

The Broad Tendon–Bone Interface in Zone IV

Brand et al. have observed that there is perhaps no other area in the human body with a ratio of tendon to bone as unfavorable as it is in the extensor zone IV. Brand credits this adverse ratio as a primary cause of surgical failures following attempts to free the dorsal expansion.[91] This ratio of tendon–bone interface, combined with the intimacy of the periosteum and the extensor tendon and the complex requirements of the extensor system on the dorsal aspect of the finger,[76] yields functional problems associated with adhesions. The problem of dorsal tendon-to-bone adherence cannot be overstated and, although recognized as a problem by some clinicians,[76,78,91,93] is probably not appreciated enough by hand therapists.

Rothkopf and associates recently studied mechanical trauma and immobilization in the canine flexor tendon model to study adhesion formation associated with complex tendon injury.[94] They define complex tendon injury as one associated with crush, concomitant nerve injury, or tendon injury treated with immobilization.[94] Their experimental model demonstrated significant decreases in tendon excursions and an increase in work requirement to effect tendon excursion. While this experimental study was performed on an animal-model flexor tendon, it has implication for the extensor injury. We have all observed that the complex injury treated with immobilization can be expected to produce complications associated with increased fibroblastic response. Many authors have endorsed the use of early motion with the complex injury.[55,57,58,95–97]

The companion study (part II) has demonstrated that a large percentage of central slip injuries (79.6% of 64 digits) are complex and that many have associated injury to adjacent soft tissue, the PIP joint, or the distal joint.[79] Other clinicians have confirmed this finding.[76,78,92,93,98]

Considering these factors we may hypothesize that a major problem in mobilization of zone III extensor tendon injury is tendon-to-bone adherence in zone IV. The immobilized repair in zone III devoid of the benefits of greater intrinsic healing and strengthening associated with early motion may attenuate or gap when motion is initiated at four to

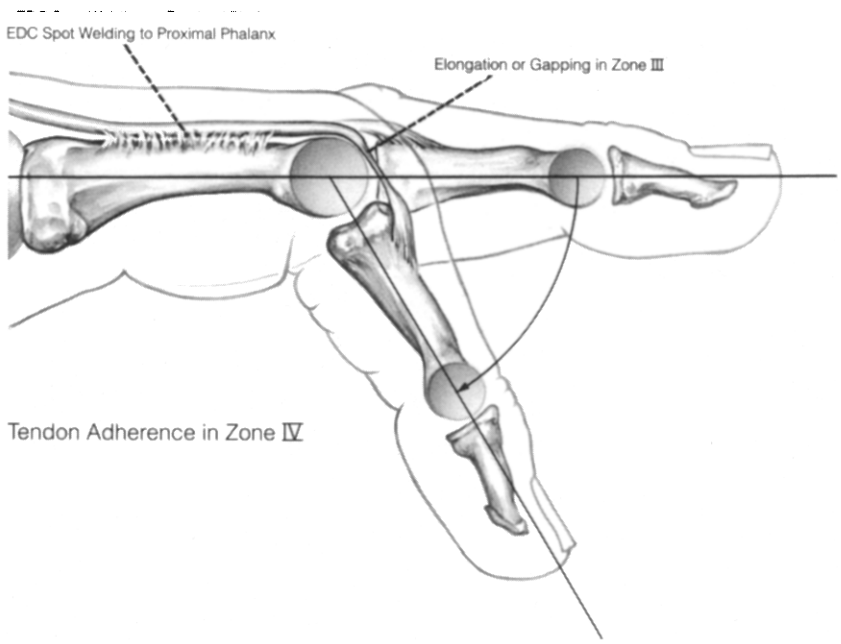

FIGURE 3. Schematic drawing illustrating the problem of tendon-to-bone adhesions following injury to the dorsal digital extensor mechanism. The broad tendon–bone interface in zone IV and the intimacy of periosteum and extensor tendon yield functional gliding problems in the zone III injury. The zone III portion of the tendon (the repaired central slip) may gap or attenuate in late mobilization programs because its more proximal segment is adherent and nongliding. The increased resistance in zone IV increases force application in zone III and may exceed the tensile strength of the repair. EDC = extensor digitorum communis.

six weeks because its proximal segment in zone IV is adherent and nongliding (Fig. 3). This increased resistance or drag in zone IV elevates the extensor tension in zone III, which may exceed the tensile strength of the repair. We observe this clinically in the immobilized central slip that begins to lag in extension as flexion is gained with late mobilization programs.

Resting the Tendon at Functional Length during Immobilization

The anatomy of the PIP joint favors flexion.[99] The normal resting position of the PIP joint is between 30° and 40° flexion.[91] In this position the central slip, with a larger moment arm, and the lateral bands, with a small moment arm, are at equal tensions.[94] An edematous joint associated with complex injury will posture in 30° to 40° as the joint will more comfortably accommodate the increased volume of edema in this position. Brand's schematic drawing of the effects of edema on the dorsal PIP joint and overlying skin (Fig. 4) helps us to visualize the effect of effusion or edema under the central slip, which could likely increase its moment arm and tension on the repair.[81,91,93]

It is not uncommon for the immobilized central slip injury not followed in therapy until the late mobilization phase to have been splinted incorrectly in some flexion with a resultant extensor lag.[79] Alumafoam splints with adhesive tape proximal and distal to the PIP joint encourage swelling and allow the PIP joint to rest in flexion; finger casts that are not checked frequently allow the PIP joint to rest flexed as edema decreases, and in general we believe that unmonitored splinting is often ineffective. These clinical problems emphasize the importance of edema control and resting the PIP joint in the optimum position during periods of immobilization to avoid extensor lag.

We can conclude that the splint position necessary to prevent extensor lag is absolute 0° extension. This position brings the central slip proximal to its normal resting position and reduces tension at the repair site.

Stress Deprivation

Biochemical and biomechanical changes in immobilized connective tissue (tendon, ligament, and cartilage) have been studied primarily in the animal model in various joints.[100] The information gained from these experimental studies must be interpreted with caution as we attempt to alter clinical treatment based on basic science studies in a nonhuman model.

Tendon. The negative effects of immobilization on tendon biochemistry are a loss of glycosaminoglycan concentration, loss of water, decreased fibronectin concentration, and decreased endotenon healing.[29,35,38,39,42,43,100] Biomechanically, the immobilized tendon loses gliding function and tensile strength.[5,21–23,26–28,31,35–37,40,41,45,94,100]

Ligament. Stress deprivation for ligament results in alterations in collagen cross-linking synthesis and degradation as well as in loss of water and proteoglycan content.[101–108]

Nonligamentous injuries treated with immobilization can produce some ligament-length problems.[109] There is some evidence that ligament structures can shorten and limit joint motion. Dahners and Wilson have demonstrated that ligaments that no longer have tension applied to them actually shorten and are associated with the contractile protein actin.[110,111]

Andriacchi et al., in a review of ligament injury and repair, postulate that immobilizing a joint alters the nutritional state of the tissues with loss of pumping action and may also decrease the normal stress-

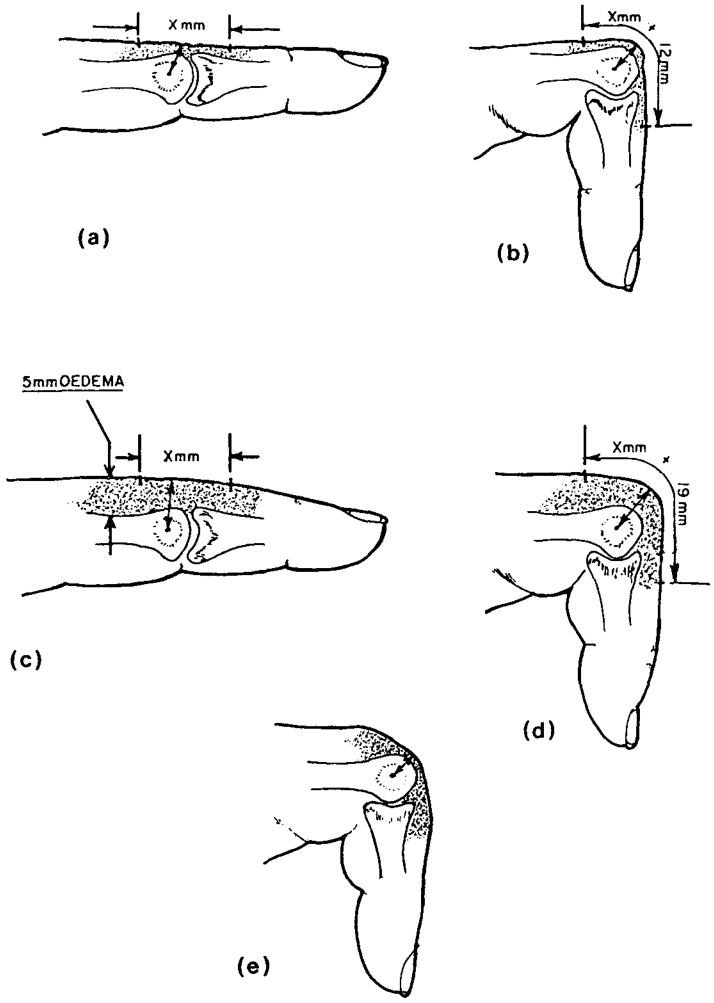

FIGURE 4. Schematic drawing by Brand illustrating the effects of a 5-mm increase in the diameter of a finger from edema and its effect on the dorsal skin. **A** *and* **B** *dorsal skin requires 12 mm of lengthening for 90° of flexion.* **C** *and* **D**, *with 5-mm thickness of edema, skin requires 19 mm of lengthening for 90° of flexion.* **E**, *with continuing torque, slowly applied, the edema fluid moves around, permitting the skin to cross closer to the joint axis and require less stretch. This illustration will help the reader to visualize the effects of protein and cellular edema that may collect under the central slip, which would increase the moment arm of the tendon and possibly contribute to attenuation of the repair or a resting posture of slight flexion. Reprinted with permission from: Brand PW: Clinical Mechanics of the Hand. St. Louis, C. V. Mosby, 1985, p. 79.*

generated electrical potentials in the dense connective tissue of ligament. They postulate that this could be misinterpreted by the fibroblast as a signal to degrade the older collagen molecules and synthesize newer, shorter collagen molecules, which will shorten the ligament structure.[109]

Cartilage. Joint motion is important to maintaining articular cartilage homeostasis. The substances required by the chondrocytes for normal metabolism are derived from synovial fluid.[112] The transport of these nutrients through cartilage occurs by convection, diffusion, or both. The combination of motion and joint loading is essential to effective convection and thus nutrient transport.[112] Applied load and motion are important to water movement in the cartilage, joint lubrication, mechanical behavior, and cartilage surface deformation.[112–116] Thus, joint immobilization decreases nutrient transport for cartilage.

Prolonged immobilization will result in decreased mechanical properties, disorganized ultrastructure, and the biochemical alterations in glycosaminoglycans and water content noted in ligament.[103,105]

Buckwalter et al. described the complex process of cartilage repair and noted that a program of controlled motion is instrumental in this process.[117] Joint and cartilage are violated in a significant number of central slip injuries and do not tolerate total immobilization well.[79]

DePalma and associates described the beneficial effects of early motion and weight bearing on the repair of full-thickness cartilage defects.[118] Salter and associates have studied the benefits of continuous passive motion (CPM) on healing rabbit cartilage. They found that in cases where defects 1 mm in diameter penetrated subchondral bone, the tissues treated with CPM produced a tissue that histochemically and morphologically resembled hyaline cartilage.[119–120] The relationship of motion and cartilage metabolism cannot be ignored.

Guidelines for tendon excursion and force application are proposed based on this assessment of the problems associated with immobilization of the repaired central slip and consideration of the biochemical and biomechanical benefits of immediate controlled stress.

THE CALCULATION OF EXCURSION AND FORCE APPLICATION

The question as we apply stress to a healing tendon in any zone is the same. What is the physiologic tension required by the healing tendon to stimulate increased cellular activity, maintain functional gliding, yet prevent gapping, elongation, or rupture at the repair site?

Excursion

The excursion necessary to maintain functional glide and stimulate cellular activity may be in the range of 3 to 5 mm. Duran and Houser recommended this excursion range for the flexor tendons in the digital sheath and felt that 3 to 5 mm is sufficient to prevent dense adhesions.[21] Gelberman et al. suggest that 3 to 4 mm of excursion is necessary to stimulate the intrinsic repair process without creating significant repair-site deformation with flexor tendons.[33] Early motion allowing 5 mm of excursion has proven to be successful with extensor tendon repairs in zones V, VI, and VII.[55,57,58] Excursion measurements of the central extensor tendon in the literature are variable and dependent on the method of study. Bunnell calculated the extensor digitorum communis excursions at the PIP joint in a cadaver hand with the wrist neutral to be 2 mm for the index finger, 3 mm middle, 3 mm ring, and 2 mm small.[80] In other cadaveric studies, Tubiana[89] cites 8 mm excursion, Valentine[121] 7–8 mm, and Zancolli[90] 6 mm; DeVoll and Saldana have reported excursions of the central slip of up to 5 mm.[122]

An et al. calculated tendon excursion and the moment arm of cadaver index finger joints during rotation and found that excursion and joint displacement were not always linear.[123] Excursion of the extensor digitorum at the PIP level with a mean motion of 89.5° was 5.58 mm.[123] Micks and Reswick determined that the extensor moment arm at the PIP is not constant and increases with the position of flexion.[124]

Elliot and McGrouther investigated the mathematical relationship between extensor tendon excursion and joint motion in seven cadaver hands and found this relationship to be linear for all joints in all five rays of the hand.[125] They found that the excursion of the middle slip over the proximal phalanx is in effect the excursion of the extensor digitorum that accompanies PIP joint movement.[125] They calculate excursion per 10° for each joint with all other joints immobile and all surrounding structures released. The mean motion per 10° was 0.8 mm for the index, middle, and ring PIP joints and 0.6 mm for the small PIP joint. This would translate to 2.4 mm of excursion for the index, middle, and ring fingers per 30° of PIP motion and 1.8 mm of excursion for the small finger. Their findings are contrary to those of An et al.[123] and Micks and Reswick.[124]

For the purposes of this study we examine excursion of the extensor digitorum at the PIP joint mathematically utilizing the radian concept as described by Brand[81] (Fig. 5). Biomechanically the excursion of the extensor tendon at the level of the PIP joint is proportional to angular changes of the joint.[80] Brand found the moment arm of the extensor tendon to be fairly constant at this level, unchanging with

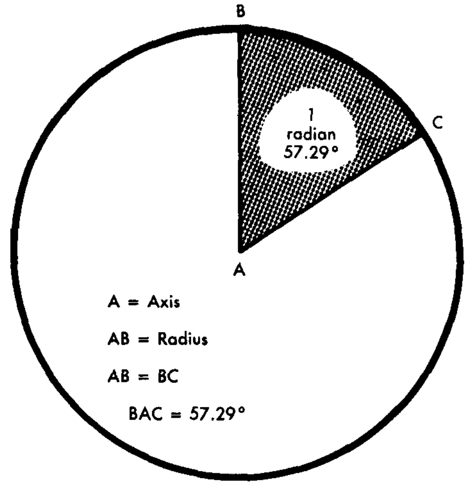

FIGURE 5. A radian is a unit of angular motion that can define joint motion and tendon excursion in relation to the moment arm of the joint in question. It is the angle formed when the radius of a circle (AB) is laid along the circumference of a circle (BC) and the two ends (B and C) are joined to the center (A) of the circle. This angle (∢ BAC) equals 57.29°, or one radian.

motion, and developed an equation to describe the relationship between joint motion and tendon excursion.[81]

Brand has calculated the mean moment arm for the extensor tendon of the middle finger PIP joint to be 7.5 mm.[81] Utilizing the radian concept, if the PIP joint is moved through an angular rotation of one radian or 57.29°, the central slip excursion is equal to the moment arm of the tendon, or 7.5 mm. To obtain the physiologic excursion necessary for the early motion program (based on recommended excursions between 3 and 5 mm as noted by Duran and Houser[21] and Gelberman et al.[33]), the joint is moved through one-half radian, or 28.65°, to obtain an excursion of 3.75 mm (Fig. 6).

As joint size varies so will the excursion to some degree, but it is the constant relationship of moment arm to tendon excursion that makes this equation workable. Clinically, it has been demonstrated that 30° of active motion the first two postoperative weeks progressing to 40° the third postoperative week will prevent restrictive adhesions in zones III and IV without creating repair-site elongation.[79]

It is important to remember that cadaveric studies and mathematical equations are finite and do not factor biology. There is no study that describes extensor digitorum communis excursion in zones III and IV following repair in vivo to give us accurate excursion measurements.

Force Application

Force application must be calculated as the sum of muscle contraction and viscoelastic drag of the tissues. Viscoelastic drag is the sum of the antagonistic muscle tension, resistance from the periarticular support systems, edema, and adhesion. Resistance from Coban wraps or bandaging also must be considered as an increased force application in early active motion programs. A safety margin must be

Central Slip Excursion Calculated By Radians

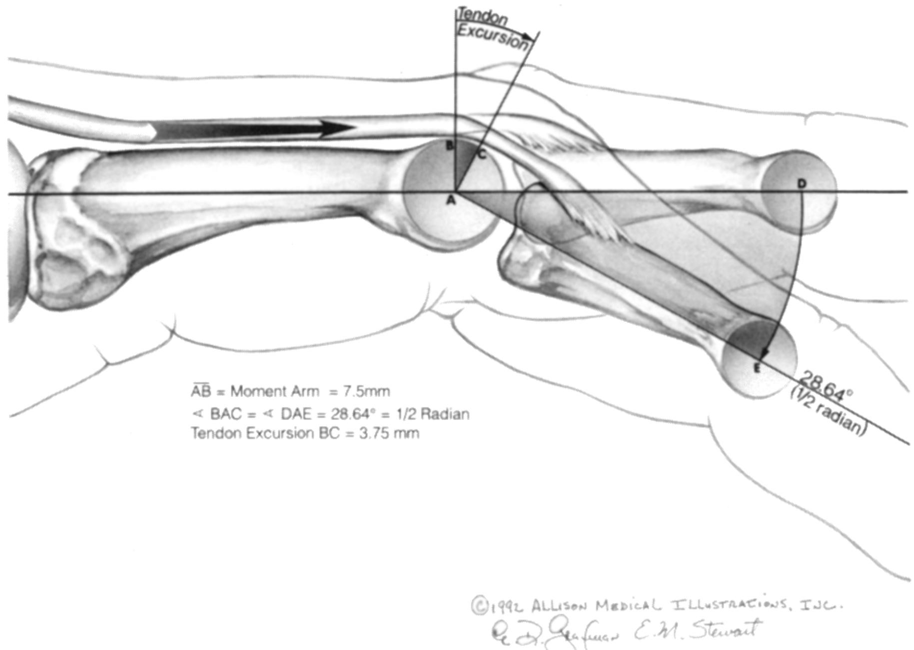

FIGURE 6. *Excursion of the central slip (zone III) as calculated by radians. AB = the moment arm of the central slip. Angle BAC = ½ radian, or 28.64°. If the proximal interphalangeal joint is moved through ½ radian, the central slip excursion will equal ½ the moment arm, or 3.75 mm. The average moment arm of the middle finger central slip = 7.5 mm as measured by Brand.*

established in which the force application (or the stress applied to the tendon) is less than the tensile strength of the tendon with all early motion programs.

Tensile Strength of the Repair. The tensile strength of the freshly sutured tendon is dependent on the strength of the suture material, the grasping power of the suture method or suture technique, and the balance between the strands and the knot.[126–130] A number of authors have investigated the mechanical strengths of tendon repair techniques and suture materials,[126–141] and suture techniques that are designed to allow active motion have been described.[130,142–148] Strickland has stated that the general consensus among hand surgeons is that to date we have no suture technique that will allow early active motion.[27] The problem here lies in the definition of active motion.

The results of these studies on tensile strength were reviewed as a part of this investigation to determine the amount of force application that extensor tendon repairs in the digits would tolerate. These reports calculate tensile strength both in newtons and grams (1 kg = 9.8 N, or 1 g = 0.01 N).

Urbaniak et al. studied eight different tendon anastomoses in digital flexor tendons of adult dogs.[129] They divided these repairs into three groups: 1) suture that applied a shearing force to tendon ends parallel to collagen bundles, 2) suture that applied an oblique or transverse compression force on the tendon, and 3) suture that was perpendicular to both the collagen bundles of the tendon and the stress applied to the repair. They compared tensile strengths initially and during healing in the dog model and then measured tensile forces on intact human tendons in vivo.[129]

Group I, with the shearing force parallel to the collagen bundles, experienced very weak repairs. The interrupted circumferential sutures demonstrated a tensile strength of 1,683 g, the Nicoladoni repair 2,683 g, and the side-to-side anastomosis 3,230 g. The degree of weakening was evaluated and the Nicoladoni repair was the lowest at all times, falling to 560 g at five days postoperative.

In group II the Bunnell measured 3,930 g initially, dropping to 630 g five days postoperative; the Kessler repair 3,970 g, dropping to 1,830 g; and the Mason-Allen 4,030 g.

In group III the fishmouth (4,055 g) and the endweave (6,430 g) were the strongest, but these repairs are usually reserved for tendon transfers where bulk of the repair is not an issue.

Measurement of the forces in intact human digital flexor tendons revealed that the stress of passive flexion–extension ranged from 200 to 300 g, flexion against mild resistance 900 g, and flexion against moderate resistance 1,500 g.[129]

Haddad et al. measured the force required to create 1 mm gap at 3.0 N (300 g) for modified Kessler, 5.3 N (530 g) for the Bunnell suture, and 4 N (400 g) for his Looped suture repair. The mean breaking force with braided nylon for the modified Kessler was 14.4 N (1,440 g), Bunnell 28.07 N (2,807 g), and Looped suture 24.5 N (2,450 g).[126]

Robertson and Al-Qattan, in an in-vitro study on fresh porcine tendon repaired with 3/0 monofilament polypropylene, measured the gap-producing force and mean breaking force on three repair techniques: the modified Kessler, Strickland, and their proposed new interlock suture technique. The gap-producing force was 22.4 N (2,240 g) for modified Kessler, 16.7 N (1,670 g) for Strickland, and 46.2 N (4,620 g) for the interlock technique. Gap size is not clearly defined in this study. Breaking force was 34.9 N (3,490 g) modified Kessler, 30.4 N (3,040 g) Strickland, and 51.6 N (5,160 g) interlock technique.[138]

Newport et al. recently reported the biomechanical characteristics of extensor tendon suture techniques with measurements at 2 mm gapping and at failure. The mattress suture gapped 2 mm at 488 g, failed at 840 g; figure of eight gapped at 587 g, failed at 696 g; Kessler gapped at 1,353 g, failed at 1,830 g; and Bunnell gapped at 1,425 g, failed at 1,985 g.[149]

Analyzing Resistance Applied to the Central Slip with Active Motion of 30° to Zero. An analysis of joint angle versus tendon force for the PIP joint was calculated mathematically. This simple model assumes that the flexors are inhibited and exert no active torque on the PIP joint. In this model, the only torques on the joint are due to the weight of the finger, the tension in the central extensor tendon, and an elastic torque arising from compression/tension of the surrounding tissues. A constant moment arm is assumed based on the work of Brand, and the finger is modeled as a simple weight acting downward at the center of mass (Fig. 7). From these assumptions, an equation for the tendon force, F, as a function of the flexion angle, θ, is expressed by the formula:

$$F = \frac{\Pi p R^2 H^2}{2r} \cos\theta + \frac{|k(\theta - \theta_o)|}{r} \quad (1)$$

Where:
p = tissue density = 1.0 g/cm³
r = finger radius = 1.0 cm
h = finger length = 3.0 cm
k = elastic modulus = 15 g-cm/degree
r = moment arm = 0.75 cm or 7.5 mm
θ_o = resting angle = 30°

The elastic torque modulus, k, was measured by Llorens[150] using a computer-controlled joint-torque-measuring device. His results showed an average elastic modulus of k = 15 g-cm/degrees over a large number of normal subjects.

The resulting tendon force variation with flexion angle of the PIP joint is shown in Figure 8. Note that

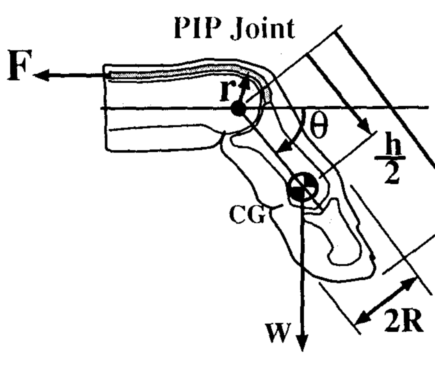

FIGURE 7. A schematic drawing of the finger model. The finger is modeled as a simple weight acting downward at the center of mass. PIP = proximal interphalangeal; F = tendon force; r = finger radius; h = finger length; W = finger weight.

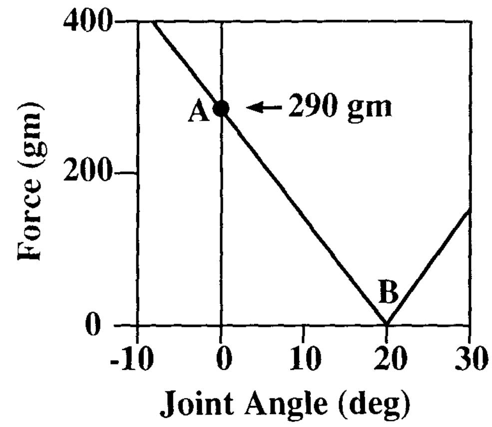

FIGURE 8. Proximal interphalangeal (PIP) tendon force versus gravity for moment arm, r = 0.75 cm. A = full extension (zero flexion angle) of the PIP joint; B = rest position of the PIP joint.

the position B is the rest position of the PIP joint and the torque at this position is zero. The force analysis, with and without gravity, effects predicted tendon forces that are 291 g and 286 g of force, respectively. Because the effects of gravity are minimal, the force at zero flexion angle (point A) is shown as 290 g. A half-radian of angulation at the PIP joint would produce a tension excursion of r/2 = 3.75 mm.

The largest of the caveats associated with this analysis are enumerated as follows: 1) Edema and inflammatory swelling will undoubtedly increase the elastic modulus k, producing a greater tendon force. It would not be unexpected to see a doubling of this force. 2) If the rest position of the finger is dramatically altered by surgery through alteration of either extensor or flexor length, the analytic prediction of the maximum force in equation 1 would be modified by the new resting angle θ_o. Generally, this equation predicts that a decrease in resting flexion angle would diminish the maximal force needed to achieve a fully extended PIP joint (zero flexion angle).

Therefore, from this study we conclude that active extension of the central slip with flexor forces diminished from the 30° resting position to 0° extension would result in a force application in the neighborhood of 286 g to 291 g to the healing tendon.

ANATOMIC CONSIDERATIONS FOR THE EARLY ACTIVE SHORT ARC MOTION PROTOCOL

Anatomic description of the entire digital extensor mechanism is beyond the scope of this paper and is available through literature review.[69–71,73,76, 78,82,84–86,88–90,121,151–156]

A review of the anatomy and biomechanics of the PIP joint and its ligamentous support will provide a clear understanding of the delicate gliding requirements of the accessory collateral ligaments, proper collateral ligaments, volar plate, superficialis tendon, lateral bands, and oblique ligaments; all of which are affected by immobilization protocols.[157–162]

The action of the middle or central extensor tendon, which inserts on the base of the middle phalanx, functions in some regard to extend all three phalanges. It extends the middle phalanx on which it inserts except when the metacarpophalangeal (MP) joint is in hyperextension; it contributes to extension of the proximal phalanx when the PIP joint is flexed; and it contributes to distal joint extension through the coordinating action of the oblique retinacular ligament.[121,163]

Tubiana makes the point that the coordination of motions in the hand is dependent on active and passive factors of the more proximal joints. The passive factors include the restraining action of the ligaments and muscular viscoelasticity, and the active factors include the dynamic balance between the antagonistic muscles.[163]

Littler and Thompson emphasize how exquisitely sensitive interphalangeal (IP) joint movement is to alterations in the gliding and elastic characteristics of the surrounding soft-tissue components.[88]

It is the intent of this section of our report to consider the effect of joint positional changes and the dynamic anatomy of the surrounding tissues to extensor tendon gliding and work requirement. This section establishes the rationale for joint position with the short arc motion protocol.

The recommended protocol for early active short arc motion is simple. The involved digit is exercised within 24 hours postoperative with controlled active exercise that allows the PIP joint to flex to 30° and actively extend to 0° extension. The wrist is positioned in 30° flexion and the MP joint in 0° extension or 5° to 10° flexion during PIP exercise. The distal joint is unrestrained during PIP joint flexion exercise.

If the lateral bands were not repaired, the PIP joint is then manually held at 0° extension and the distal joint is flexed independently. Exercises are performed frequently, hourly if possible. Between controlled exercise sessions the PIP and DIP joints are immobilized at absolute 0° and a volar static digital thermoplastic splint is taped down directly over the PIP joint. Wrist and MP joint immobilization is not recommended.[79]

The Significance of Wrist Position

The position of the wrist during splinting and the concept of wrist tenodesis exercises as they affect excursions for flexor tendon repairs[164] and extensor tendon repairs[165] have recently been studied. The extrinsic tendons have the greatest moment arm, thus the greatest excursion at the wrist joint.[81] Wrist position influences tension in the extrinsic tendons of the digits due to the viscoelasticity of the antagonist muscle–tendon unit.[81,163,166,167] Passive tension is minimal when the muscle is short and increases as the muscle lengthens.[167]

Wrist Flexion. The movements of wrist flexion and finger extension are synergistic; finger extension is effectively increased as the wrist flexes.[70] The position of wrist flexion reduces the force of the digital extrinsic flexors.[166] While this position may increase passive tension in the extensor system as the muscle–tendon unit lengthens,[167] the actual force required of the extensor communis to extend the digital joints is reduced by the reduction of viscoelastic flexor forces.[167]

The action of the interossei muscles with the wrist flexed may further reduce work requirements of the digital extensor mechanism in active extension of the IP joints. Close and Kidd have concluded that the interossei do contract in extension even when no resistance is applied if: 1) all the fingers are extended simultaneously or 2) the finger is extended with the wrist flexed.[168]

Wrist Extension. Active wrist extension is synergistic with finger flexion. With the digital splint taped in place between exercise sessions, unrestricted motion takes place in the wrist and MP joint. As the wrist extends, the MP joint will flex due to the viscoelasticity of the flexors. The sagittal bands glide distally with MP flexion, actually reducing tension on the central slip.[90] Tubiana states that the movements of the phalanges can be independent of wrist position through the action of the interossei.[163]

Unrestricted motion for the wrist and MP joint with the PIP and DIP joints splinted in extension has not been a problem clinically.[79] The recommended position for the wrist with the short arc motion protocol is 30° flexion during PIP exercise. This position reduces flexor resistance, facilitates interossei function to extend the PIP, and thus reduces work requirement of the extensor digitorum (ED) with active extension of the PIP joint.

The Significance of the Metacarpophalangeal Joint

Metacarpophalangeal joint positional changes from complete extension to complete flexion glide the sagittal bands and interosseous hood proximal and distal by 16 mm[121] to 20 mm.[90]

Metacarpophalangeal Joint Flexion. As the sagittal bands glide distally with MP joint flexion the ED is able to transmit virtually no force distal to the MP joint because of its insertion on the dorsal hood/sagittal band complex.[169] Tension on the central tendon is decreased with MP flexion because of sagittal band distal migration.[90]

Rouzaud et al. recently reported the same observation in a study of central slip tension measured intraoperatively.[170]

Long has demonstrated with electromyography (EMG) that the interossei are electrically active with MP flexion contributing to IP joint extension, and that the lumbrical is electrically silent with MP flexion.[171,172] Valentine determined by anatomic dissection that the lumbrical does work with the MP joint in flexion, contributing to IP joint extension.[173]

Therefore, with the MP joint flexed, PIP joint extension would be affected primarily through the interossei,[171,172] possibly with some contribution from the lumbrical,[173] but with little contribution from the ED.[169]

The central slip is also not stressed with MP joint flexion if the IP joints are splinted. No clinical problem has been observed from leaving the wrist and MP joint free. The position of MP flexion is not recommended during PIP exercise as extensor communis force transmission is minimal in this position.[79]

Metacarpophalangeal Joint Extension. The sagittal bands glide proximally with MP joint extension.[90] The attachment of the dorsal hood with the MP extended is slack and allows distal transmission of power to the ED.[169] Long has determined by EMG that the lumbrical is electrically active with the MP extended, thus contributing to IP joint extension.[171,172] Indirectly, the lumbrical contributes to IP extension by reducing the viscoelastic resistance of the flexor digitorum profundus. Lumbrical contraction pulls the profundus tendon distal, reducing the work requirement of the antagonistic extensor.[171,172] Thus the lumbricals neutralize the viscoelastic tension of the profundi during digital extension.[169]

Although Long has determined by EMG that the interossei are silent with the MP extended,[171,172] Valentine has determined by anatomic dissection that if the MP is extended, contraction of the interossei is transmitted directly to the lateral bands, which in turn extend the two distal joints.[173] As mentioned previously, Close and Kidd have demonstrated that the interossei will contract if all the MP joints are extended simultaneously or if the wrist is in flexion.[168]

Thus, the position of MP joint extension facilitates ED function,[169] yet minimizes work requirement because in this position the lumbricals assist IP extension both directly through the action on the PIP joint and indirectly by neutralizing the resistance of the profundus.[171,172] The interossei assist in PIP extension by transmitting force through the lateral bands.[173] The position of MP joint extension or very slight flexion is biomechanically superior for the PIP-controlled exercise portion of this protocol.

The Significance of Distal Interphalangeal Joint Position

The DIP joint is unrestrained during the active PIP exercise of 30°. With simultaneous flexion of the PIP and DIP, gliding of the terminal tendon is not transferred to the extensor communis but is taken up by the volar slide of the lateral bands.[121] The terminal tendon slackens through the action of the lateral band migration when the PIP is flexed, thus DIP flexion is facilitated.[121]

Zancolli observed in two cadaveric dissections that when the flexor digitorum profundus acts both IP joints flex (linked flexion); the middle phalanx flexes before the distal phalanx; and during the course of flexion, the angle of flexion of the middle joint is greater than that of the distal joint.[90]

Distal joint extension is facilitated by the combined action of the extensor digitorum, action of the lateral bands, and tenodesis of the oblique retinacular ligament.[88,121] Active PIP joint extension, which is initiated by the long extensor, creates tension in the oblique retinacular ligament, which assists DIP extension. Distal interphalangeal joint extension is then completed as the lateral bands rise dorsally and finally reach the same tension as the central extensor tendon.[88]

Thus, the moderate PIP joint flexion of 30° with the DIP joint unrestrained may create no more than an estimated 1 to 2 mm excursion of both lateral bands and terminal tendon, based on lateral band and terminal tendon excursion cited by Zancolli[90] and Littler and Thompson.[88]

If the lateral bands are unrepaired, the DIP joint is then flexed independently while the PIP joint is manually stabilized at 0° extension. Flexion of the DIP joint with the PIP joint restrained imparts 3 to 4 mm of distal glide to the ED in zone IV through the action of the lateral bands in their attachment to the ED proximal to zone III. This exercise reduces tendon tension in zone III while creating distal migration of the ED in zone IV.

RESULTS

Safe parameters for tendon excursion and force application are defined as they apply to the application of controlled stress to the healing tendon in zone III.

To obtain the physiologic excursion necessary for the early motion program, it is recommended that the PIP joint be actively moved through one-half radian (28.65°) to obtain a central slip excursion of 3.75 mm within 24 hours postoperative.

Resistance to the central slip is calculated by mathematical formula (equation 1, Figs. 7 and 8) to be from 286 g to 291 g, which leaves an adequate safety margin between repaired tensile strength and force application.

The position of application of stress based on a study of dynamic anatomy of the PIP joint is 30° wrist flexion, MP joint 0° extension or slight flexion, and distal joint unrestrained.

DISCUSSION

This study was designed to provide scientific rationale for immediate active motion of the repaired central slip and to provide precise parameters for force application and excursion in support of a clinical study that compares immobilized central slip injuries with those treated with immediate active motion.[79]

Basic science studies that identify the adverse effect of immobilization of connective tissue (tendon, ligament, and cartilage) and the positive influence of immediate or early controlled motion provide the biochemical and biomechanical rationale for early motion.[5,21–43,45,94,100–120]

The protocol recommends that motion be initiated immediately based on basic science research that suggests wound activity and tensile strength are enhanced by very early motion.[29,35,42,43]

Frequent exercise is recommended based on the work of Gelberman et al., who determined in a clinical study that "more is better,"[45] and Salter et al., who determined, in a comparison of cartilages treated with immobilization, mobilization, and continuous passive motion, that continuous passive motion produced significantly better healing in violated cartilage.[119,120]

Active motion versus passive motion is recommended to ensure tendon glide in the zone IV region where the tendon–bone interface is great[91] and often compromised by restricting adhesions, a problem associated with zone III injury.[76,78,91,93]

Tendon excursion in a proximal direction may require some muscle contraction[166]; thus, the active extension portion of this protocol is an important component. The importance of active motion in tendon rehabilitation has been studied by a number of authors,[130,142–148] and this concept is becoming more popular clinically. The concept of "minimal active tension" as described by Savage[167] or "active muscle–tendon tension" as described by Evans,[174] in which the force applied to the healing tendon is just enough to exceed the passive tension of the antagonist muscle group, is proposed. The application of force to the healing tendon must be less than the tensile strength of the tendon.

The portion of this report that summarized the tensile strengths of the various repairs is critical to this formula.[126–149] Attention should be focused on the lowest reported tensile strength with forces that create tendon gapping. The force that produces a gap should be considered as much of an issue as the forces that cause repair-site failure. While Gelberman et al. found that a repair-site gap of 3–4 mm in flexor tendons was compatible with stimulation of the intrinsic repair process without creating repair-site deformation in the flexor system,[33] gap formation or tendon elongation in the digital extensor mechanism may be less tolerable and more of a problem. Synchronous extensions of the PIP and DIP joints depend on balanced lengths of the central slip and the lateral bands.[86,87,154]

There is only one report in this study dedicated to the biomechanical strength of the extensor tendon suture.[149] Newport et al. reported 2-mm gaps for the mattress suture when force application reached 488 g. The mattress suture failed at 840 g of force. The figure of eight gapped 2 mm at 587 g and failed at 696 g; the Kessler gapped 2 mm at 1,353 g and failed at 1,830 g.[149]

Urbaniak et al. found the weakest anastomosis in their group I repairs, where the shearing force was parallel to the collagen bundles. They measured the tensile strength of the interrupted circumferential sutures at 1,683 g, the Nicoladoni at 2,683 g, and the side-to-side anastomosis at 3,230 g. Five days postoperative, the Nicoladoni repair had the lowest tensile strength at 560 g.[129]

These studies in the animal model[121] and in cadaver[149] are helpful, but are limited in that they do not measure the tensile strength in vivo of a repaired tendon moved immediately. Tensile strength may be increased by immediate controlled motion.[29,35,42,43]

Force application to the central slip is measured in this study at 286 g to 291 g with PIP joint extension from 30° flexion to 0° (equation 1, Figs. 7 and 8). However, it may be that the work requirement of the ED is further reduced by action of the interossei and lumbrical muscles in the suggested exercise position. We can consider that this motion, which applies an estimated 291 g of force to the central slip, will be tolerated by the lowest measured tensile strength, which would gap 2 mm with 488 g of force.[149] This safety margin of 200 g is small and is at best a scientific estimate. It does, however, offer some basis for force application, and clinically this force application has caused no problem.[79]

There is disagreement in the literature regarding the extensor moment arm at the PIP joint. Micks and Reswick[124] determined that the extensor moment arm at the PIP joint is not constant and increases with flexion. Brand found the moment arm of the extensor to be fairly constant at this level, unchanging with motion.[81] An et al. found that excursion and joint displacement were not always linear at the PIP joint,[123] but Elliot and McGrouther found that relationship to be linear.[125] It is important to remember that this protocol calls for only 30° motion and thus the changes in the extensor moment arm from 0° to 30° are small and unlikely to be significant enough to alter the calculation of excursion. For this reason, the simple equation calculated by radians (PIP motion of 28.65° or one-half radian = extensor tendon excursion of 3.75 mm) is workable for the early short arc motion program.

CONCLUSION

This report has defined precise parameters for force application, tendon excursion, and exercise position to allow the hand therapist to scientifically apply immediate active controlled stress to the open and repaired zone III extensor tendon. Tendon excursion of 3.75 mm, force application of 291 g, and exercise position of wrist flexion 30°, MP extension 0°, and DIP unrestrained are defined as safe treatment guidelines in this study and proven to be effective in the companion study (part II).[79] The method of splinting and exercise and concepts of timing, duration of exercise, and joint position have been described to address the problems as defined for the zone III extensor injury, adhesion formation in zone IV, incorrect splint immobilization, and connective-tissue stress deprivation as imposed by total immobilization protocols.

REFERENCES

1. Banes AJ, Donlon K, Link GW, et al: Cell populations of tendon: A simplified method for isolation of synovial cells and internal fibroblasts: Confirmation of origin and biologic properties. J Orthop Res 6(1):83–94, 1988.
2. Barfred T: Experimental rupture of the Achilles tendon: Comparison of experimental sutures in rats of different ages and

living under different conditions. Acta Orthop Scand 42:406–428, 1971.
3. Fujita M, Hukuda S, Doida Y: The effect of constant direct electrical current on intrinsic healing in the flexor tendon in vitro. J Hand Surg [Br] 17B:94–98, 1992.
4. Gauss-Muller V, Kleinman HK, Martin GR, et al: Role of attachment factors and attractants in fibroblast chemotaxis. J Lab Clin Med 96:1071–1080, 1980.
5. Gelberman RH, VandeBerg JS, Manske PR, Akeson WH: The early stages of flexor tendon healing: A morphologic study of the first fourteen days. J Hand Surg 10A:776–784, 1985.
6. Manske PR, Gelberman RH, VandeBerg JS, Lesker P: Intrinsic flexor tendon repair. J Bone Joint Surg 66A:385–396, 1984.
7. Manske PR, Lesker P: Histologic evidence of intrinsic flexor tendon repair in various experimental animals: An in vitro study. Clin Orthop 182:297–304, 1984.
8. Mass DP, Tuel RJ: Intrinsic healing of the laceration site in human superficialis flexor tendons in vitro. J Hand Surg 16A:24–30, 1991.
9. Mass DP, Tuel R: Human flexor tendon participation in the in vitro repair process. J Hand Surg 14A:64–71, 1989.
10. Matthews P, Richards H: Factors in the adherence of flexor tendon after repair. An experimental study on the rabbit. J Bone Joint Surg 58B:230–236, 1976.
11. Nessler JP, Mass DP: Direct current electrical stimulation of tendon healing in vitro. Clin Orthop 217:303–312, 1987.
12. Okuda Y, Gorski JP, Ank N, et al: Biochemical, histological, biomechanical anlayses of canine tendon. J Orthop Res 5:60–68, 1987.
13. Riederer-Henderson MA, Gauger A, Olson L, Robertson C, Greenlee TK: Attachment and extracellular matrix differences between tendon and synovial fibroblastic cells. In Vitro 19(2):127–133, 1983.
14. Stevenson JH, Cho Y, Pang WK, et al: Functional, mechanical and biochemical assessment of ultrasound therapy on tendon healing in the chicken toe. Plast Reconstr Surg 77:965–967, 1986.
15. Lundborg G, Rank F: Experimental studies on cellular mechanisms involved in healing of animal and human flexor tendon in synovial environment. Hand 12:3–11, 1980.
16. Clark RAF, Folkvord JM, Hart CE, et al: Platelet isoforms of platelet-derived growth factor stimulate fibroblasts to contract collagen lattices. J Clin Invest 84:1036–1040, 1989.
17. Falcone PA, Caldwell MP: Wound metabolism. Clin Plast Surg 17:443, 1990.
18. Morris J, Markham H, Higgs P, et al: Platelet derived growth factor enhances healing in a rabbit deep flexor tendon model. Unpublished research, 1992.
19. Pierce CF, Mustoe TA, Altrock BW, et al: Role of platelet-derived growth factor in wound healing. J Cell Biochem 45:319–326, 1991.
20. Schmidt JA, Mizel SB, Cohen D, et al: Interleukin I, a potential regulator of fibroblast proliferation. J Immunol 128:2177, 1982.
21. Duran RJ, Houser RG: Controlled passive motion following flexor tendon repair in zones II and III. *In* The American Academy of Orthopaedic Surgeons: Symposium on tendon surgery in the hand. St. Louis, C. V. Mosby, 1975, p. 105.
22. Gelberman RH, Manske PR, Akeson HH, Woo SL-Y, Lundborg G, Amiel D: Flexor tendon repair. J Orthop Res 4:119–128, 1986.
23. Gelberman RH, Woo SL-Y: The physiological basis for application of controlled stress in the rehabilitation of flexor tendon injuries. J Hand Ther 2:66–70, 1989.
24. Kleinert HE, Kutz JE, Cohen MJ: Primary repair of zone II flexor tendon lacerations. *In* AAOS Symposium on Tendon Surgery in the Hand. St. Louis, C. V. Mosby, 1975, pp. 91–103.
25. Lister GD, Kleinert HE, Kutz JE, Atasoy E: Primary flexor tendon repair followed by immediate controlled mobilization. J Hand Surg 2:441–451, 1977.
26. Strickland JW, Glogovac SV: Digital function following flexor tendon repair in zone II: A comparison of immobilization and controlled passive motion techniques. J Hand Surg 537–543, 1980.
27. Strickland JW: Biologic rationale, clinical application and results of early motion following flexor tendon repair. J Hand Ther 2:71–83, 1989.
28. Woo S, Gelberman RH, Cobb NG, Amiel D, Lothringer K, Akeson WH: The importance of controlled passive mobilization on flexor tendon healing: A biomechanical study. Acta Orthop Scand 52:615–622, 1981.
29. Freehan LM, Beauchene JG: Early tensile properties of healing chicken flexor tendons: Early controlled passive motion versus postoperative immobilization. J Hand Surg 15A:63–68, 1990.
30. Gelberman RH, Menon J, Gonsalves M, Akeson WH: The effects of mobilization on the vascularization of healing flexor tendons in dogs. Clin Orthop 153:283–289, 1980.
31. Gelberman RH, Woo S, Lothringer K, Akeson WH, Amiel D: Effects of early intermittent passive mobilization on healing canine flexor tendons. J Hand Surg 7:170–175, 1982.
32. Gelberman RH, VandeBerg JS, Lundborg GN, Akeson WH: Flexor tendon healing and restoration of the gliding surface: An ultrastructural study in dogs. J Bone Joint Surg 65A:70–80, 1983.
33. Gelberman RH, Botte MJ, Spiegelman JJ, et al: The excursion and deformation of repaired flexor tendons treated with protected early motion. J Hand Surg 11A:106–110, 1986.
34. Gelberman RH, Woo S, Amiel D, Horibe S, Lee D: Influences of flexor sheath continuity and early motion on tendon healing in dogs. J Hand Surg 15A:69–77, 1990.
35. 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 12A:590–595, 1987.
36. Seyfer AE, Bolger WE: Effects of unrestricted motion on healing: A study of post traumatic adhesions in primate tendons. Plast Reconstr Surg 83:122–128, 1989.
37. Viidik A: Tensile strength properties of Achilles tendon systems in trained and untrained rabbits. Acta Orthop Scand 40:261–277, 1969.
38. Woo SL-Y, Ritter MA, Amiel D, et al: The biomechanical and biochemical properties of swine tendons: Long term effects of exercise on the digital extensors. Connect Tissue Res 7:177–183, 1980.
39. Woo SL-Y, Gomez MA, Amiel D, et al: The effects of exercise on the biomechanical and biochemical properties of swine digital flexor tendons. J Biomech Eng 103:51–56, 1981.
40. Woo SL-Y: Mechanical properties of tendons and ligaments: I. Quasi-static and nonlinear viscoelastic properties. Biorheology 19:385–396, 1982.
41. Woo SL-Y, Gomez MA, Woo Y-K, et al: Mechanical properties of tendons and ligaments: II The relationships of immobilization and exercise on tissue remodeling. Biorheology 19:397–408, 1982.
42. Amiel D, Gelberman R, Harwood F, Siegel D: Fibronectin in healing flexor tendons subjected to immobilization or early controlled passive motion. Matrix II:184–189, 1991.
43. Gelberman RH, Steinberg D, Amiel D, Ing D, Akeson W: Fibroblast chemotaxis after repair. J Hand Surg 16A:686–693, 1991.
44. Mason ML, Allen HS: The rate of healing tendons. An experimental study of tensile strength. Ann Surg 113:424–459, 1941.
45. Gelberman RH, Nunley JA, Osterman AL, Breen TF, Dimick MP, Woo SL-Y: Influences of the protected passive mobilization interval on flexor tendon healing: A prospective randomized clinical study. Clin Orthop 264:189–196, 1991.
46. Chow JA, Thomes LJ, Dovelle S, et al: A combined regimen of controlled motion following flexor tendon repair in "No Man's Land." Plast Reconstr Surg 79:447–453, 1987.
47. Langlais F: Early mobilization after primary flexor tendon repair in 152 fingers (excluding zone II) and in 60 thumbs. Int Orthop 13:269–274, 1989.
48. Saldana MJ, Chow JA, Gerbino P: Further experience in rehabilitation of zone II flexor tendon repair with dynamic traction splinting. Plast Reconstr Surg 87:543–546, 1991.
49. Saldana MJ, Ho PK, Lichtman DM, et al: Flexor tendon repair and rehabilitation in zone II open sheath technique versus closed sheath technique. J Hand Surg 12A:1110–1114, 1987.
50. Kleinert HE, Verdan C: Report of the committee on tendon injuries. J Hand Surg 8:794, 1983.
51. Allieu Y, Ascencio G, Gomis R, et al: Controlled mobilization of extensor tendon lacerations. Study of 120 cases. Rev Chir Orthop 70:68–73, 1984.
52. Allieu Y, Ascencio G, Rouzaud JC: Protected passive mobi-

lization after suturing the extensor tendons of the hand: A survey of 120 cases. *In* Hunter JM, Schneider LH, Mackin EJ: Tendon Surgery in the Hand. St. Louis, C. V. Mosby, 1987, pp. 344–348.
53. Browne EZ, Ribik CA: Early dynamic splinting for extensor tendon injuries. J Hand Surg 14A:72–76, 1989.
54. Chow JA, Dovelle S, Thomes LJ, et al: Postoperative management of repair of extensor tendons of the hand—Dynamic splinting versus static splinting. Orhtop Trans 11(2):258–259, 1987.
55. Evans RB, Burkhalter WE: A study of the dynamic anatomy of extensor tendons and implications for treatment. J Hand Surg 11A:774–779, 1986.
56. Evans RB: Therapeutic management of extensor tendon injuries. Hand Clin 2:157–169, 1986.
57. Evans RB: Clinical application of controlled stress to the healing extensor tendon: A review of 112 cases. Phys Ther 68(12):1041–1049, 1989.
58. Evans RB: Therapeutic management of extensor tendons. *In* Hunter JM, Schneider LH, Mackin EJ, Callahan AD (eds): Rehabilitation of the Hand, 3rd ed. St. Louis, C. V. Mosby, 1990, pp. 492–514.
59. Frere G: Inteŕêt d'une mobilisation relative par appareil: de Levame en postopératoire immédiat des lésions traumatiques récentes de l'appareil extenseur des doigts longs. Réunion du GEM (Société Française de Chirurgie de la Main), Lyon, France, May 21–22, 1982.
60. Hung LK, Chan A, Chang J, et al: Early controlled active mobilization with dynamic splinting for treatment of extensor tendon injuries. J Hand Surg 15A:251–257, 1990.
61. Laboureau JP, Renevey A: Utilisation d'un appareil personnel de contention et de reéducation segmentaire élastique de la main type "crabes." Ann Chir 25:165, 1980.
62. O'Dwyer FG, Quinton DN: Early mobilization of acute middle slip injuries. J Hand Surg 15B:404–406, 1990.
63. Evans RB: Physiology and management of connective tissue injury. Hand Therapy Certification Commission Lecture Series, 1989–1991.
64. Evans RB: Early active controlled motion for connective tissue injury at the dorsal proximal interphalangeal level. Key Biscayne, FL, Florida Hand Society, April 26, 1991.
65. Rolph-Roeming K: Early mobilization of extensor tendon laceration in zones III and IV. Orlando, FL, American Society of Hand Therapists Scientific Session, September 1991.
66. Silverman P (discussant for review for Evans RB and Burkhalter WE during Scientific Session for presentation of): A study of the dynamic anatomy of extensor tendons and implications for treatment. New Orleans, LA, American Society of Hand Therapists, February 1986.
67. Thomes L: Controversies in the management of tendon injuries. Phoenix, AZ, American Society for Surgery of the Hand, November 9, 1990.
68. Walsh MT, Rinehimer WE, Muntzer E, Patel J: Early controlled motion vs static splinting for zone III and IV extensor tendon laceration. Orlando, FL, American Society of Hand Therapists Scientific Session, September 1991.
69. Boyes JH: Boutonnière deformity [discussion]. *In* Cramer LM, Chase RA (eds): Symposium on the Hand, Vol. 3. St. Louis, C. V. Mosby, 1971, p. 56.
70. Burton RI: Disorders of the extensor mechanism. *In* Evarts CM (ed): Surgery of the Musculoskeletal System. New York, Churchill Livingstone, 1983.
71. Doyle JR: Extensor tendons—Acute injuries. *In* Green DP (ed): Operative Hand Surgery. New York, Churchill Livingstone, 1988, pp. 2045–2072.
72. Froehlich JA, Akelman E, Herndon JH: Extensor tendon injuries at the proximal interphalangeal joint. *In* Burton RI (ed): Hand Clinics. Philadelphia, W. B. Saunders, (1):25–37, 1988.
73. Kilgore ES, Graham WP: The Hand: Surgical and Non-surgical management. Philadelphia, Lea & Febiger, 1977.
74. Lovett WL, McCalla MA: Management and rehabilitation of extensor tendon injuries. Orthop Clin North Am 44(4):811–826, 1983.
75. McFarlane RM, Hampole MK: Treatment of extensor injuries of the hand. Can J Surg 16:366–375, 1973.
76. Rosenthal EA: Extensor surface injuries at the proximal interphalangeal joint. *In* Bowers WH (ed): The Interphalangeal Joints. New York, Churchill Livingstone, 1987, pp. 94–110.
77. Verdan CE: Primary and secondary repair of flexor and extensor tendon injuries. *In* Flynn JE (ed): Hand Surgery. Baltimore, Williams and Wilkins, 1966, pp. 220–275.
78. Wilson RL: Management of acute extensor tendon injuries. *In* Hunter JM, Schneider LH, Mackin EJ (eds): Tendon Surgery in the Hand. St. Louis, C. V. Mosby, 1987, pp. 336–343.
79. Evans RB: Early active short arc motion for the repaired central slip. Phoenix, AZ, American Society for Surgery of the Hand Scientific Session, November 14, 1992.
80. Boyes JH: Bunnell's Surgery of the Hand. Philadelphia, J. B. Lippincott, 1964, pp. 13–20.
81. Brand PW: Clinical Mechanics of the Hand. St. Louis, C. V. Mosby, 1985.
82. Eaton RG: The extensor mechanism of the fingers. Bull Hosp Joint Dis 30:39, 1969.
83. Landsmeer JMF: The anatomy of the dorsal aponeurosis of the human finger and its functional significance. Anat Rec 104:31–44, 1949.
84. Littler JW: Principles of reconstructive surgery of the hand. *In* Converse JM (ed): Reconstructive Plastic Surgery. Philadelphia, W. B. Saunders, 1964, pp. 1612–1632.
85. Littler JW: The digital extensor–flexor system. *In* Converse JM (ed): Reconstructive Plastic Surgery. Philadelphia, W. B. Saunders, 1977, pp. 3166–3175.
86. Littler JW: The finger extensor mechanism. Surg Clin North Am 47:415, 1967.
87. Littler JW, Eaton RG: Redistribution of forces in the correction of the boutonnière deformity. J Bone Joint Surg 49A:1267, 1967.
88. Littler JW, Thompson JS: Surgical and functional anatomy. *In* Bowers WH: The Interphalangeal Joints. New York, Churchill Livingstone, 1987, pp. 14–20.
89. Tubiana R: Extensor apparatus of the fingers. *In* Hunter JM, Schneider LH, Mackin EJ (eds): Tendon Surgery in the Hand. St. Louis, C. V. Mosby, 1987, pp. 319–324.
90. Zancolli EA: Structural and dynamic bases of hand surgery, 2nd ed. Philadelphia, J. B. Lippincott, 1979, pp. 38–55.
91. Brand PW, Thompson DE, Micks JE: The biomechanics of the interphalangeal joints. *In* Bowers WH (ed): The Interphalangeal Joints. New York, Churchill Livingstone, 1987.
92. Newport ML, Blair WF, Steyers CM: Long-term results of extensor tendon repair. J Hand Surg 15-A:961–966, 1990.
93. Burkhalter WE: Personal communication, February 1992.
94. Rothkopf DM, Webb S, Szabo RM, Gelberman RH, May JW: An experimental model for the study of canine flexor tendon adhesions. J Hand Surg 16A:694–700, 1991.
95. Beasley RW: Management of the mutilated hand. *In* Hunter JM, Schneider LH, Mackin EJ, Callahan AD (eds): Rehabilitation of the Hand. St. Louis, C. V. Mosby, 1978.
96. Burkhalter WE: Wound classification and management. *In* Hunter JM, Schneider LH, Mackin EJ, Callahan AD (eds): Rehabilitation of the Hand, 3rd ed. St. Louis, C. V. Mosby, 1990, pp. 167–171.
97. Wilson RL, Carter MS: Management of hand fractures. *In* Hunter JM, Schneider LH, Mackin EJ, Callahan AD (eds): Rehabilitation of the Hand, 3rd ed. St. Louis, C. V. Mosby, 1990, pp. 284–294.
98. McAuliffe J: Personal communication, February 1992.
99. Bowers WH: The anatomy of the interphalangeal joints. *In* Bowers WH (ed): The Interphalangeal Joints. New York, Churchill Livingstone, 1987.
100. Woo SL-Y, Buckwalter, JA (eds): Injury and repair of the musculoskeletal soft tissues. Park Ridge, IL, American Academy of Orthopaedic Surgeons, 1988.
101. Akeson WH: An experimental study on joint stiffness. J Bone Joint Surg 43A:1022–1034, 1961.
102. Akeson WH, Amiel D, LaViolette D: The connective-tissue response to immobility: A study of the condroitin-4 and 6 sulfate and dermatan sulfate changes in periarticular connective tissue of control and immobilized knees of dogs. Clini Orthop 51:183–197, 1967.
103. Akeson WH, Amiel D, Mechanic GL, et al: Collagen cross-linking alterations in joint contractures: Changes in the reducible cross-links in periarticular connective tissue collagen after nine weeks of immobilization. Connect Tissue Res, 5:15–19, 1977.
104. Akeson WH, Amiel D, LaViolette D, et al: The connective

tissue response to immobility: An accelerated aging response? Exp Gerontol 3:289–301, 1968.
105. Amiel D, Woo SL-Y, Harwood FL, Akeson WH: The effect of immobilization on collagen turnover in connective tissue. A biochemical–biomechanical correlation. Acta Orthop Scand 53:325–332, 1982.
106. Gamble JG, Edwards CC, Max SR: Enzymatic adaptation in ligaments during immobilization. Am J Sports Med 11:379–389, 1983.
107. Grood ES, Butler DL, Noyes FR: Models of ligament repairs and grafts. *In* Finerman G (ed), American Academy of Orthopaedic Surgeons. Symposium on Sports Medicine: The Knee. St. Louis, C. V. Mosby, 1985, pp. 169–181.
108. Woo SL-Y, Matthews JV, Akeson WH, et al: Connective tissue response to immobility: Correlative study of biomechanical and biochemical measurements of normal and immobilized rabbit knees. Arthritis Rheum 18:257–264, 1975.
109. Andriacchi T, Sabiston P, DeHaven K, et al: Ligament, injury and repair. *In* Woo SL-Y, Buckwalter JA (eds): Injury and Repair of the Musculoskeletal Soft Tissues. Park Ridge, IL, American Academy of Orthopaedic Surgeons, 1988, pp. 103–128.
110. Wilson CJ, Dahners LE: An examination of the mechanism of ligament contracture. Clin Orthop 227:286–291, 1988.
111. Dahners LE: Ligament contraction—A correlation with cellularity and actin staining. Trans Orthop Res Soc 11:56, 1986.
112. Mow V, Rosenwasser M: Articular cartilage: Biomechanics. *In* Woo SL-Y, Buckwalter JA: Injury and Repair of the Musculoskeletal Soft Tissues. Park Ridge, IL, American Academy of Orthopaedic Surgeons, 1988, pp. 427–463.
113. Mak AF: The apparent viscoelastic behavior of articular cartilage, the contributions from the intrinsic matrix viscoelasticity and interstitial fluid flows. J Biomech Eng 108:123–130, 1986.
114. Myers ER, Armstrong CG, Mow VC: Swelling pressure and collagen tension. *In* Hukins DWL (ed): Connective Tissue Matrix. London, MacMillan Press, 1984, pp. 161–186.
115. Hall B, Newman S: Cartilage, molecular aspects. Boca Raton, FL, CRC Press, 1991, pp. 240–241.
116. Woo SL-Y, Mow VC, Lai WM: Biomechanical properties of articular cartilage. *In* Skalak R, Chien S (eds): Handbook of Bioengineering. New York, McGraw-Hill, 1987, pp. 4.1–4.44.
117. Buckwalter J, Rosenberg L, Coutts R, et al: Articular cartilage, injury and repair. *In* Woo SL-Y, Buckwalter JA: Injury and Repair of the Musculoskeletal Soft Tissues. Park Ridge, IL, American Academy of Orthopaedic Surgeons, 1988, pp. 465–482.
118. DePalma AF, McKeever CDE, Subin DK: Process of repair of articular cartilage demonstrated by histology and autoradiography with tritiated thymidine. Clin Orthop 48:229–242, 1966.
119. Salter RB, Simmonds DF, Malcolm DW, et al: The biological effect of continuous passive motion on healing of full-thickness defects in articular cartilage: An experimental study in the rabbit. J Bone Joint Surg 61A:1232–1251, 19980.
120. Salter RB, Minster RR, Bell RS, et al: Continuous passive motion and the repair of full-thickness articular cartilage defects: A one-year followup. Trans Orthop Res Soc 7:167, 1982.
121. Valentine D: Physiology of extension of the fingers. *In* Tubiana R: The Hand, Vol 1. Philadelphia, W. B. Saunders, 1981, pp. 389–398.
122. DeVoll JR, Saldana MJ: Excursion of finger extensor elements in zone III. Presented at the annual meeting of the American Association for Hand Surgery, Toronto, Ontario, Canada, 1988.
123. An KN, Ueba Y, Chao EY, et al: Tendon excursion and moment arm of index finger muscles. J Biomech 16(6):419–425, 1983.
124. Micks J, Reswick J: Confirmation of differential loading of lateral and central fibers of the extensor tendon. J Hand Surg 6:462–467, 1981.
125. Elliot D, McGrouther DA: The excursions of the long extensor tendons of the hand. J Hand Surg 11B:77–80, 1986.
126. Haddad RJ, Kester MA, McCluskey GM, Brunet ME, Cook SD: Comparative mechanical analysis of a looped suture tendon repair. J Hand Surg 13A:709–713, 1988.
127. Lin GT, An KN, Amadio PC, Cooney WP: Biomechanical studies of running suture for flexor tendon repair in dogs. J Hand Surg 13A:553–558, 1988.
128. Trail IA, Powell ES, Noble J: The mechanical strength of various suture techniques. J Hand Surg [Br] 17B:89–91, 1992.
129. Urbaniak JR, Cahill JP, Mortenson RA: Tendon suturing methods; Analysis of tensile strength. *In* American Academy of Orthopaedic Surgeons, Symposium on Tendon Surgery in the Hand. St. Louis, C. V. Mosby, 1975, pp. 70–80.
130. Wade PJF, Wetherell RG, Amis AA: Flexor tendon repair, significant gain in strength from the Halsted peripheral suture technique. J Hand Surg 14B:232–235, 1989.
131. Becker H, Davidoff M: Eliminating the gap in flexor tendon surgery, a new method of suture. The Hand 9(3):306–311, 1977.
132. Greulich M, Gubisch W, Reichert H: The Tsuge suture, experimental and clinical experiences. Handchir Mikrochir Plast Chir 22:59–63, 1990.
133. Ketchum LD: Suture materials and suture techniques used in tendon repair. Hand Clin 1:43–53, 1985.
134. Ketchum LD, Martia NL, Kappel DA: Experimental evaluation of factors affecting the strength of tendon repairs. Plast Reconstr Surg 59:709–719, 1977.
135. Kim SK: Further evolution of the grasping technique for tendon repair. Ann Plast Surg 7:113–119, 1981.
136. Mashadi ZB, Amis AA: The effect of locking loops on the strength of tendon repair. J Hand Surg [Br] 16B:35–39, 1991.
137. Mashadi ZB, Amis AA: Strength of the suture in the epitenon and within the tendon fibres. Development of stronger peripheral suture technique. J Hand Surg [Br] 17B:171–175, 1992.
138. Robertson GA, AL-Qattan MM: A biomechanical analysis of a new interlock suture technique for flexor tendon repair. J Hand Surg [Br] 17B:92–93, 1992.
139. Sántha VE, Répasý G: Modified and new methods of flexor tendon sutures and tensile strength testing, a comparative in vitro study. Handchir Mikrochir Plast Chir 22:240–246, 1990.
140. Von Fraunhofer JA, Storey RS, Stone IK, Masterson BJ: Tensile strength of suture materials. J Biomed Mater Res 19:595–600, 1985.
141. Wade PJF, Muir IFK, Hutcheon LL: Primary flexor tendon repair, the mechanical limitations of the modified Kessler technique. J Hand Surg 11B:71–76, 1986.
142. Becker H, Orak F, Duponselle E: Early active motion following a beveled technique of flexor tendon repair. Report on fifty cases. J Hand Surg 4:454–460, 1979.
143. Braun F, Foucher G, Buch JN, et al: Flexor digitorum profundus and flexor pollicis longus repair by the "Rope Down" technique, results of a series of 77 cases. Ann Chir Main 10:13–21, 1991.
144. Lee H: Double loop locking suture: A technique of tendon repair for early active mobilization, part 1. J Hand Surg 15A:945–952, 1990.
145. Lee H: Double loop locking suture, a technique of tendon repair for early active mobilization, part II, clinical experience. J Hand Surg 15A:953–958, 1990.
146. MacMillan M, Sheppard JE, Dell PC: An experimental flexor tendon repair in zone II that allows immediate post operative mobilization. J Hand Surg 12A:582–589, 1987.
147. Messina A: The double armed suture, tendon repair with immediate mobilization of the fingers. J Hand Surg 17A:137–142, 1992.
148. Savage R: In vitro studies of a new method of flexor tendon repair. J Hand Surg 10B:135–141, 1985.
149. Newport ML, Sims JB, Williams MS: Biomechanical characteristics of extensor tendon suture techniques PEO61. Orlando, FL, Scientific poster exhibit, American Society for Surgery of the Hand annual meeting, September 1991.
150. Llorens WL: An experimental analysis of finger joint stiffness. MSME thesis, Baton Rouge, Louisiana State University, 1986.
151. Eyler DL, Markee JE: The anatomy and function of the intrinsic musculature of the fingers. J Bone Joint Surg 36A:1–9, 1954.
152. Garcia Elias M, An KN, Berglud BS, et al: Extensor mechanism of the fingers. I. A quantitative geometric study. J Hand Surg 16A:1130–1136, 1991.
153. Haines RA: The extensor apparatus of the finger. J Anat 85:251–259, 1951.

154. Harris C, Rutledge GL Jr: The functional anatomy of the extensor mechanism of the finger. J Bone Joint Surg 54A:713, 1972.
155. Schultz RJ, Furlong J II, Storace A: Detailed anatomy of the extensor mechanism at the proximal aspect of the finger. J Hand Surg 6:493–498, 1981.
156. Snow JW, Switzer H: Method of studying the relationship between the finger joints and the flexor and extensor mechanisms. Plast Reconstr Surg 55:242, 1975.
157. Bowers WH, Wolf JW, Nehil JL, Bittinger S: The proximal interphalangeal joint volar plate I. An anatomical and biomechanical study. J Hand Surg 5:79–88, 1980.
158. Eaton RG: Joint injuries of the hand. Springfield, IL, Charles C Thomas, 1971, pp. 15–32.
159. Kuczynski K: The proximal interphalangeal joint. J Bone Joint Surg 50B:656–663, 1968.
160. Kuczynski K: Lesser known aspects of the proximal interphalangeal joints of the human hand. The Hand 7:31–34, 1975.
161. Landsmeer JMF: A report of the coordination of the interphalangeal joints of the human finger and its disturbances. Acta Morphol Neer/Scand 2:59, 1953.
162. Landsmeer JMF: Anatomical and functional investigation of the articulation of the human fingers. Acta Anat (suppl 24) 25:1, 1955.
163. Tubiana R: Architecture and functions of the hand. *In* Tubiana R (ed): The Hand, Vol 1. Philadelphia, W. B. Saunders, 1981, pp. 19–93.
164. Cooney WP, Lin GT, An K-N: Improved tendon excursion following flexor tendon repair. J Hand Ther 2:102–106, 1989.
165. MinamiKawa Y, Peimer CA, Yamaguchi T: Wrist position and extensor tendon amplitude following repair. J Hand Surg 17A:268–271, 1992.
166. Brand PW: Personal communication, June 1992.
167. Savage R: The influence of wrist position on the minimum force required for active movement of the interphalangeal joints. J Hand Surg [Br] 13B:262–268, 1988.
168. Close JR, Kidd CC: The functions of the muscles of the thumb, the index and the longer finger. J Bone Joint Surg 51A:1601–1620, 1969.
169. Simmons BP, De La Caffinière JY: Physiology of flexion of the fingers. *In* Tubiana R: The Hand, Vol I. Philadelphia, W. B. Saunders, 1981, pp. 377–388.
170. Rouzaud JC, Chammas M, Micaleff JP, et al: Mesure de la tension différentielle du système extenseur au niveau de la PIP. Paris, France, 2nd International Congress, International Federation of Societies of Hand Therapists, May 19, 1992.
171. Long CH: Intrinsic–extrinsic muscle control of the finger electromyographic studies. J Bone Joint Surg 52A:853–867, 1970.
172. Long C: Electromyographic studies of hand function. *In* Tubiana R: The Hand, Vol I. Philadelphia, W. B. Saunders, 1981, pp. 427–440.
173. Valentine P: The interossei and the lumbricals. *In* Tubiana R: The Hand, Vol I. Philadelphia, W. B. Saunders, 1981, pp. 244–254.
174. Evans RB: Application of active controlled muscle–tendon tension to the healing tendon. Paris, France, 2nd International Congress, International Federation of Societies of Hand Therapists, May 29, 1992.