# Tendon Healing, Edema, and Resistance to Flexor Tendon Gliding: Clinical Implications

Ya Fang Wu, MD, Jin Bo Tang, MD*

**KEYWORDS**

• Tendon healing • Tenocyte proliferation • Apoptosis • Adhesions • Resistance to tendon gliding • Edema • Annular pulleys • Early mobilization

**KEY POINTS**

- Early flexor tendon healing is characterized by peak cellular apoptosis of cells in the tendons in the first week, followed by progressively greater tenocyte proliferation in the second and third weeks.
- Apoptosis is a predominant event of the tenocytes in the middle and late healing periods, contributing to remodeling and restoration of tendon gliding surface. Tenocyte proliferation is minimal in these periods.
- Edema in subcutaneous tissue and the tendon is an inevitable biologic process, adding substantially to the resistance to tendon gliding. Major annular pulleys may greatly resist gliding of a repaired and swelling tendon.
- Experimentally, edema of the subcutaneous tissues contributed 20% to 25%, the intact A2 pulley 30%, and extensor tendons 15% to 20% of total resistance to the gliding of the flexor tendon. Tendon bulkiness (swelling and surgical repairs) also greatly contributes to the resistance.
- The contribution made by each of the factors changes dynamically with timing elapsed since surgery and the position of finger flexion. The overall resistance to tendon motion is progressively increased when the digit is extremely flexed.

The difficulties in functional recovery after digital flexor tendon injury stem from the weak healing capability of the tendon itself. Although the tendon has healing capacity through proliferation and collagen synthesis by tenocytes, the healing process is slow and usually weak in the first few weeks. This process causes 2 major problems—repair rupture and adhesion formation. During early tendon mobilization after surgery, repair rupture is the result of weak tendon healing as well as increases in resistance to tendon motion. Additionally, adhesions form because of insufficient tendon gliding. During the early tendon healing period, another factor also exerts a great influence on resistance to tendon gliding—edema formation, an inevitable biologic process after surgery to subcutaneous tissues and the tendon itself. Edema peaks a few days after surgery and persists as long as biologic healing processes are active.

Tendon healing, edema, resistance to tendon gliding, and formation of adhesions around the tendon are all innately associated. Their intricate relationship has become clearer over the past decade, and elucidation of their relationship has brought about modifications in surgical and postsurgical treatment of the digital flexor tendons.

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Department of Hand Surgery, The Hand Surgery Research Center, Affiliated Hospital of Nantong University, 20 West Temple Road, Nantong, Jiangsu 226001, China
* Corresponding author.
*E-mail address:* jinbotang@yahoo.com

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## TENDON HEALING

Tenocytes were once believed incapable of proliferating and repairing the lacerated tendon, and tendon healing was thought to rely on the invasion of peripheral cells and blood vessels, which frequently lead to formation of restrictive adhesions.[1,2] However, this concept was challenged in the 1970s and 80s. A large body of biologic and molecular evidence confirmed that tenocytes actively participate in tissue repair and that tendons are capable of healing from injury and revitalizing themselves. This process is dominated by tenocyte proliferation and production of their surrounding extracellular matrix early after surgical repair and by tendon surface remodeling and collagen realignment in the late healing stage.[3,4] How the tenocyte proliferation is balanced with other cellular events during the early tendon healing period and how tenocytes are cleared after tendon healing is complete have been partly elucidated by recent studies.[5–11]

### *Early Healing Stage: Peak Apoptosis Followed by Gradual Proliferation of Tenocytes*

After tissue injury, cellular apoptosis and proliferation are closely associated and elegantly balanced through the entire healing period.[5–8] In a rat central 1/3 patellar tendon injury model,[8] apoptosis of the healing tendon was found to increase on day 14 and reached a maximum on day 28 after injury. However, proliferative cells reached a maximum population at day 4 and remained high up to day 28. Afterward, the number of both apoptotic and proliferative cells reduced and gradually returned to normal levels. We studied cellular apoptosis and proliferation of the repaired digital flexor tendon in a chicken model.[9] The tendons were stained with an in situ terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) assay to detect apoptotic cells over a 12-week period after surgical repair. We also stained the proliferating cell nuclear antigen (PCNA) and Bcl-2 (an antiapoptotic protein) to assess responses suppressive to cell apoptosis.

In uninjured tendons, only 3% ± 2% of the tenocytes showed signs of apoptosis and 1% ± 1% showed signs of active proliferation. The percentage of apoptotic cells went up to more than 40% at days 3 to 7 after tendon injury; on day 3, the number of inflammatory cells in the wound site also peaked. The number of TUNEL-positive cells, presumably composed mainly of inflammatory cells as well as tenocytes, peaked during the very early days in the healing process (days 3 and 7 in our chicken model).

In contrast, the number of PCNA-positive cells did not significantly increase until day 7 and peaked during days 7 to 21. At day 28, the number of PCNA-positive cells dropped; Bcl-2–positive cells showed parallel changes. We found that tenocyte apoptosis is typically accelerated within several days after injury, followed by increases in proliferation of tenocytes in 2 to 4 weeks with activation of molecular events to inhibit apoptosis (**Fig. 1**).

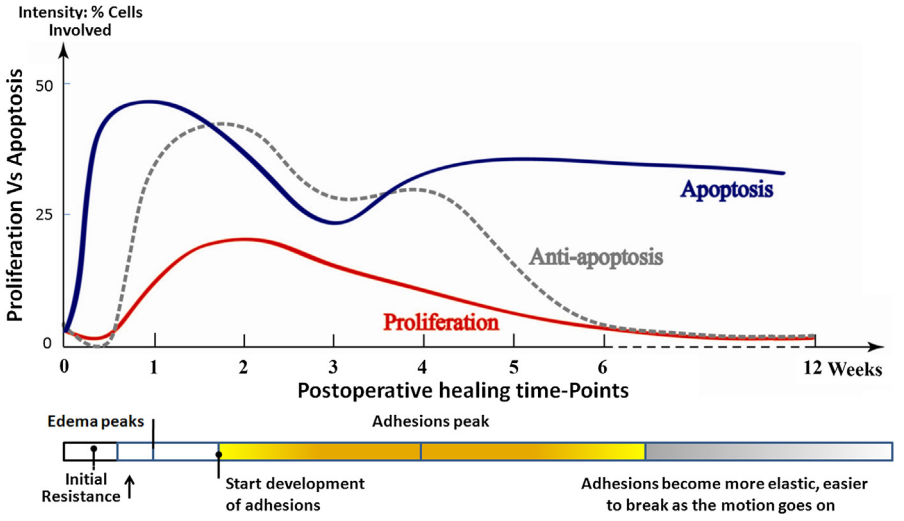


**Fig. 1.** Changes in proliferation of tenocytes, apoptosis of tenocytes, and their dynamic balance at different tendon healing periods. In the initial days after trauma and surgery, apoptosis peaks and proliferation declines, but from week 2 to 3, proliferation peaks and apoptosis declines. After week 4, apoptosis dominates while proliferation is minimal. Associated changes in edema and formation of adhesions are shown as well.

### *Middle and Late Healing Stages: Persistent Apoptosis with Greatly Declined Proliferation of Tenocytes*

In the next step of our in vivo studies, we quantified cell apoptosis and proliferation during the middle and late healing stages.[10] We recorded cell apoptosis separately at the junction site of the repair and away from the junction site (ie, the extended regions), as well as regions of surface and core. The percentage of apoptotic tenocytes was generally higher on the surface of the tendon compared with that in the core, indicating a greater need for cellular clearance and surface remodeling in the surface region in the middle to late periods.

We recorded that the total cell population did not start to decline until after day 56 (2 months). Cell apoptosis persisted at a relatively high level on the tendon surface even at 3 months. Cell apoptosis in the core region declined after 2 months. These findings indicate that active tendon remodeling persists through the very late tendon healing period, especially on the tendon surface. In sharp contrast to tenocyte apoptosis, proliferation of tenocytes declined drastically after week 4, with less than 5% of the PCNA-positive cells in the tendon. At weeks 8 to 12, PCNA-positive cells returned to nearly normal levels. This finding points to the fact that in the middle and late tendon healing periods tenocyte apoptosis is a dominant event; it may even be a major biologic event in the remodeling and restoration of the gliding surface.

The percentage of apoptotic tenocytes ranged between 30% and 40% in the total cell population in both surface and core regions in the middle and late healing periods, except at the junction regions at week 8 (around 45%). Tenocyte apoptosis was slightly greater in the junction sites of the surgical repair, but generally apoptotic tenocytes did not exceed 40%; this indicates that apoptosis is at a relatively high level in the middle and late periods, but is still lower than in the first few days after surgery.

In areas distant from the junction site, apoptosis is more prominent in the tendon surface than in the tendon core. The increase in cellular apoptosis in the surface region is likely associated with the clearance of excess cells, which serves to promote formation of smooth gliding surfaces by remodeling adhesions. The junction region encompasses the greatest cellularity and the most robust healing reactions. Clearance of cells and reestablishment of collagenous connection can be a major event in the junction site in the late healing period.

### *Cellular Apoptosis in Adhesions*

We then extended our investigation to adhesions forming around the repaired flexor tendons after a complete cut and surgical repair of the digital flexor tendon and 3 weeks of immobilization of the chicken toes. We correlated tendon gliding excursions recorded with a tensile testing machine as well as adhesion severity scores with degrees of cellular apoptosis. The percentage of apoptotic cells was noted to increase from the tendon core, to the tendon surface and to the adhesion-tendon interface, with that in the adhesion core the highest. The percentages of apoptotic cells in adhesions and at the adhesion-tendon gliding interface were generally 50% to 65%. The percentage of apoptotic cells was as high as 69% in the adhesion over the junction site of the tendon ends. Analysis of apoptotic index (ie, the percentage of apoptotic cells) against tendon excursions and severity of adhesions indicates that the tendons with more severe adhesions, or a lower excursion, see greater apoptosis in their adhesions and adhesion-tendon interfaces (**Fig. 2**).

Cellular apoptosis in the adhesions and at the adhesion-tendon interface may contribute to the fate of adhesions and the restoration of tendon gliding surface. This suggestion is consistent with the results of Wong and colleagues,[11] who reported that more apoptotic reactions were generally observed in the subcutaneous tissue and the immediate vicinity of the tendon toward the end of tendon healing in a mouse tendon injury model. We believe that the increases in cellular apoptosis at the adhesion and the tendon-adhesion interface are associated with the effect of shear deformation induced by mobilization. More apoptosis of cells in the tendon-adhesion interface would accelerate the recovery of the smooth gliding surface.

### *Microdynamics of Adhesions at Different Stages of Tendon Healing*

The mechanical characteristics of adhesion tissues determine tendon gliding, but this relationship has rarely been investigated. We performed an in vivo study to determine the microdynamic features of adhesions in the middle and late healing period (postoperative weeks 4–8). The tendon with surrounding adhesions was harvested en bloc. A 1-cm tendon segment centered on the cut site was excised dorsally, but the adhesions medial, lateral and palmar to the tendon were kept intact, which bridged the 2 tendon stumps (**Fig. 3**), leaving the tendon ends connected solely by adhesions. The samples were then subjected to cyclic loading tests at 2, 5, 10, and 15 N for 10 cycles each.

The adhesions harvested at week 4 were the strongest, which survived at loads of 2 and 5 N and disrupted at 10 or 15 N. In contrast, adhesions at week 6 disrupted at loads of 5 or 10 N, although

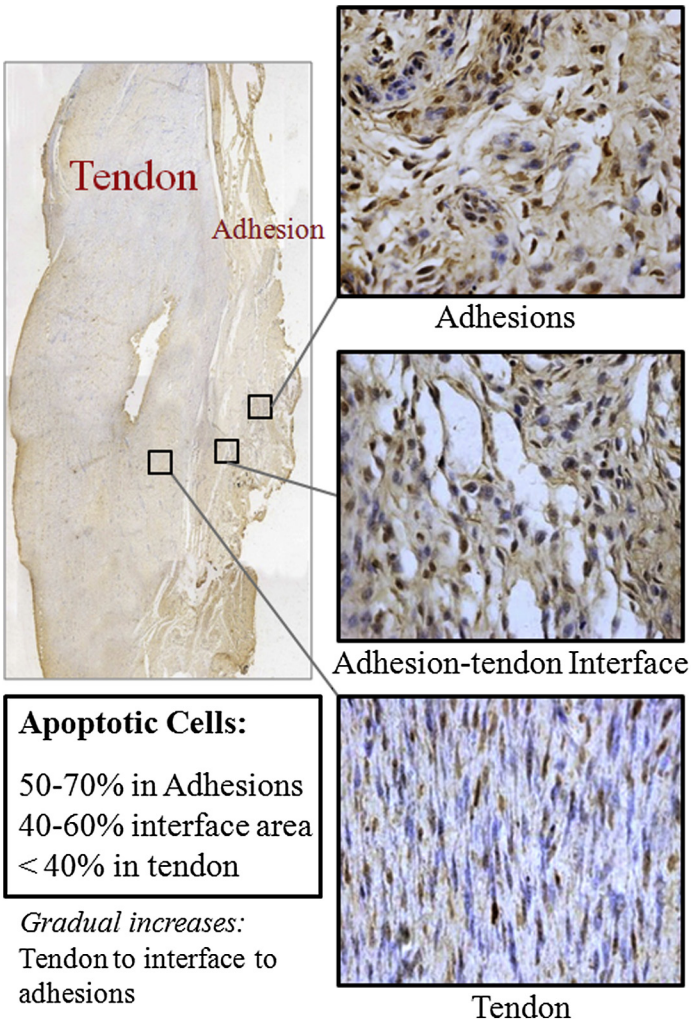


**Fig. 2.** Fifty percent to seventy percent of the total cell population in adhesions are apoptotic. The next highest proportion of apoptotic cells is seen in the tendon-adhesion gliding interface (40%–60%), and the fewest apoptotic cells are seen in the tendon core (<40%). Apoptosis is a predominant finding in the late healing stage.

all withstood a 2-N load. The failure load of the adhesions at week 8 was the lowest, from 2 to 5 N. We recorded minimal plastic deformation of the adhesion at week 4 (an average of 0.5 mm/N) and moderately large deformation at week 6 (1.3 mm/N) after cyclic loading at 2 N for 10 cycles (see **Fig. 3**). At week 8, the plastic deformity was the greatest. This biomechanical test revealed that the plasticity and strength of adhesions vary substantially in the course of tendon healing—the ability of adhesion tissues to resist tension decreases and tissue flexibility increases from middle to late healing stages. The microdynamic features of adhesion tissues determine the sliding amplitude of the tendon and how the tendon responds to postoperative motion.

Microdynamic features of adhesions and tenocytes apoptosis are associated—as apoptosis of cells in the adhesions goes on, the adhesions are more easily broken up after the adhesions are loaded. Digital motion would certainly reduce the strength and increase the elasticity of the adhesion fibers. We assume that the external force applied to move the tendon during digital motion, transferred to shear force over the adhesions and adhesion-tendon gliding interface, acts to continuously stimulate cellular apoptosis, in turn reducing the density and strength of the adhesion fibers, resulting in an increasingly greater elasticity and breakup of the adhesion fibers in the late healing stage (see **Fig. 3**).

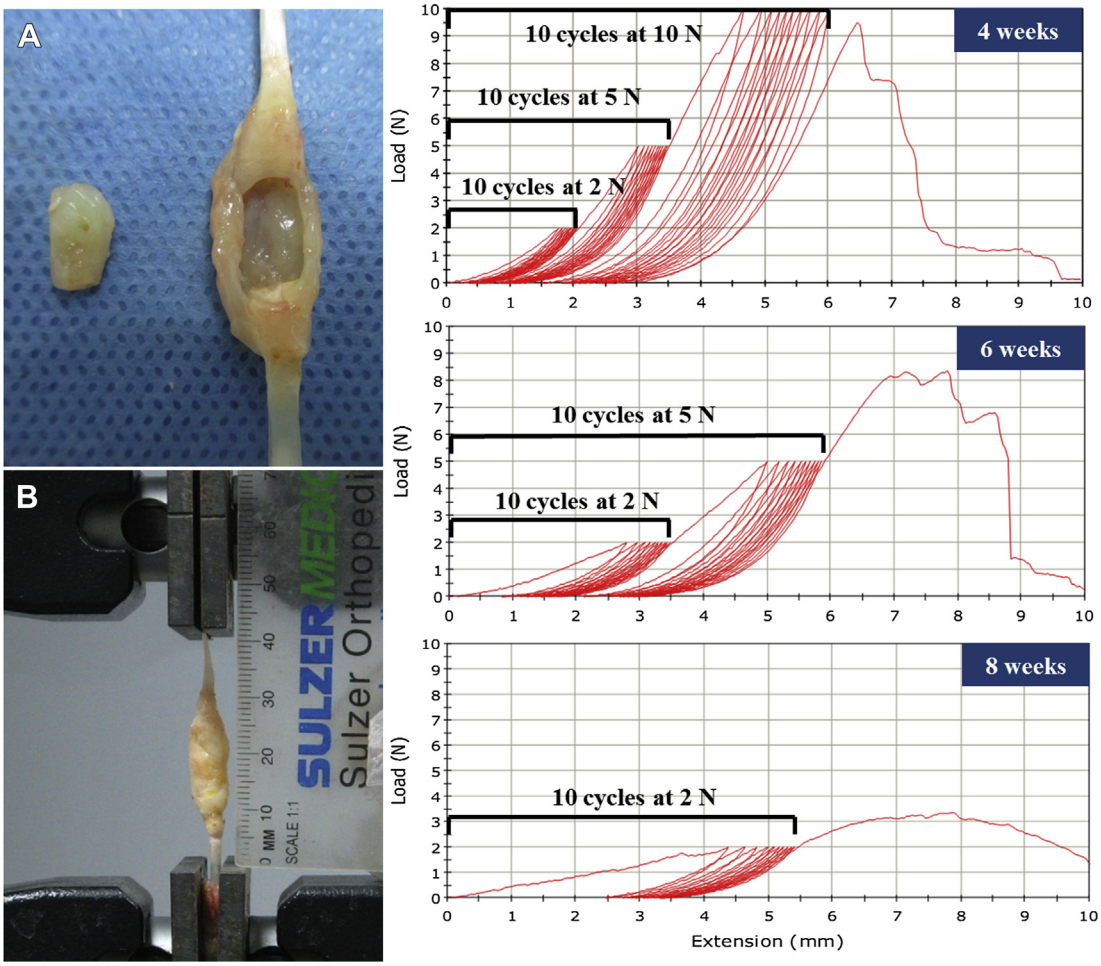


**Fig. 3.** The test method of microdynamic features of the adhesions. (*A*) Removal of a segment of tendon centered by the repair site and dorsal adhesions from the dorsal aspect of the tendon. (*B*) Test of breaking strengths and elasticity of the adhesions connecting the tendon. The right 3 graphs show how the adhesions harvested at 4, 6, and 8 weeks survived under different loads. The microdynamic features relate to how the adhesions resist tendon gliding and how easily these adhesions can be broken because of tensile load during active tendon gliding. The adhesions are more resistant to force in the midperiod of the healing than in the late period, indicating that a persistent mobilization of the digits up to the late healing stage is very important to fully restore tendon function.

### *Molecular Therapies to Reduce Adhesion Formation*

Previous studies showed that expression of transforming growth factor (TGF)-β1 is upregulated in the early tendon healing period,[12–14] which is considered to be responsible for the formation of scars such as adhesions around healing digital flexor tendons. We also found in the lacerated digital flexor tendons of the chicken substantial upregulation of the TGF-β1, in contrast to little (or no) increase in platelet-derived growth factor and vascular endothelial growth factor and downregulation of the basic fibroblast growth factor gene expression.[14] Use of antibodies to neutralize TGF-β1 has been shown to increase range of digital motion.[15] Gene therapy approaches to deliver synthesized small interfering RNA or microRNA (miRNA) to regulate the level of gene expression offers novel therapeutic potentials for a variety of pathologic conditions.[16] We designed the engineered miRNA and delivered the engineered miRNA to silence expression of the TGF-β1gene.[17]

Engineered miRNA was shown to downregulate TGF-β expression in vitro and in vivo.[17] The functional evaluation of this gene modulation approach is under way in our group. We also intended to use nanoparticles loaded with miRNAs to inhibit TGF-β1 gene expression to reduce the adhesions. This study is also ongoing in our lab.

Improving tendon healing strength through molecular modulation has been attempted and proved difficult, with varying results in animal models.[18–21] Currently, a few growth factors have been shown to enhance tendon healing strength, but their effectiveness appears to depend greatly on the persistence of expression of exogenous growth factors and the methods of delivery to the tendons. We expect to see more exciting results in future years in the molecular regulation of tendon healing.

## EDEMA FORMATION AFTER TENDON REPAIR

### *Subcutaneous Tissue Edema: in vitro and in vivo Studies*

Edema of both peritendinous tissues and tendons inevitably persists in the early period of tendon healing. Our group tried to characterize how the extent of peritendinous edema affects the amount of energy required for digital flexion and force of tendon gliding during simulated active digital motion.[22] Edema in subcutaneous tissue was reproduced in vitro in 3 different severities (mild, moderate, and severe) and 3 lengths (1 cm, 2 cm, and 3 cm) along the chicken toes.

Subcutaneous tissue edema was found to increase energy and force required to move the tendons. Increases in the severity of edema produced 2-fold or 3-fold greater resistance to tendon gliding, but changes in the length of the edematous tissue increased the resistance by only 10% to 30% in the presence of mild, moderate or severe edema (**Fig. 4**).[22] In other words, the resistance to tendon gliding was affected more by the severity of tissue edema than by the extension of edema in the digits, which suggests that edema severity is a concern in determining the timing of commencement and methods of tendon motion exercise.

Our continuing in vivo studies validated the in vitro findings. Through measuring edema in the chicken toes over a 2-week period after surgery involving tendon cut and repair,[23] we noted that the resistance to motion of the repaired tendon increased progressively for the first 4 days and remained consistent from days 4 to 7. More severe edema corresponded to greater tendon gliding force and work of digital flexion on each of the initial 5 post surgical days. We therefore suggested that the opportune time to start digital motion is from the fourth to seventh day after surgery to avoid overlapping the period of increased resistance. We also noted that repetitive motion of the digit over a number of cycles (6 in this study) greatly reduced the force and energy of the digital flexion. We suggested passive motion, intended to eliminate finger stiffness, as a "warm-up" measure before more aggressive active digital flexion.[23]

Xie and colleagues,[24] Halikis and colleagues,[25] and Zhao and colleagues[26,27] have also reported the force and energy required to move the tendon in the initial days after surgery in animal models. Most findings support a later start (3–5 days delay) of postoperative motion to avoid the period of increasing resistance presumably from postoperative edema.

### *Edema Inevitably Presents in the Repaired Tendons*

Edema of the tendon is a common finding during delayed primary repair and is especially detrimental to tendon gliding in the flexor sheath region, as the swelling takes up room inside the narrow tendon gliding tunnel. Edema of the tendon increases resistance to tendon gliding, and many surgeons have found that repair of both superficialis and profundus tendons is particularly difficult in the delayed primary repair. Clinically, the senior

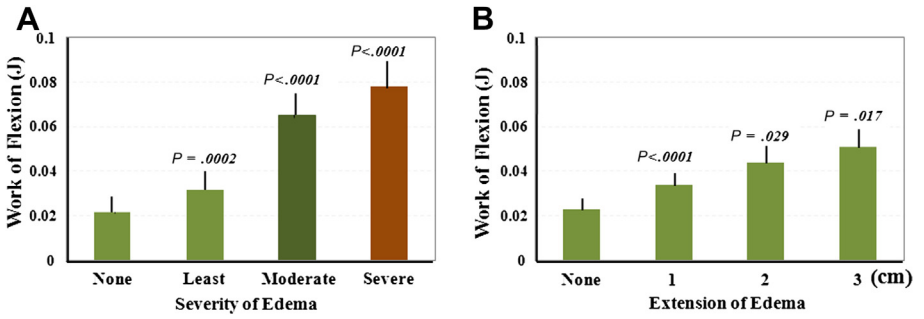


**Fig. 4.** Increases in severity of subcutaneous edema (*A*) produce far greater resistance than increasing extension of edema (*B*). Data and *P* values are obtained from 22 chicken toes after creation of different severity or extension of edema, respectively. These findings indicate that severity of edema, clinically judged by increases in diameter of the digits, loss of skin folds, etc., are more influential to the resistance; whether the edema is localized or extended in the hand is less influential.

author (JBT) often finds it hard to repair the superficialis tendon if the repair is delayed for 1 to 2 weeks. Tight closure of the sheath is harmful to the gliding of the edematous tendons.

Earlier experimental studies by our group support an opening (or nonrepair) of the damaged digital sheath, and partial venting of the annular pulleys if necessary, to accommodate the edematous tendons.[28,29] This policy is particularly relevant in delayed primary repair. In primary flexor tendon repair, the tendons do not swell at the time of surgery but do become edematous a few days later. This swelling contributes to the resistance to tendon gliding during early postoperative motion. From a few days to a few weeks after surgery, the repaired tendon at the repair site always has a greater diameter than a normal tendon, which is an important consideration in planning both surgery and rehabilitation.

Self-adhesive tapes could produce energy of digital flexion 4 times the baseline values.[30] Removal of dressing decreases the resistance to tendon motion.

### *Edema Peaks in the First Week and Persists in the Later Weeks; Adhesions Start to Form in the Second Week*

Mobilization regimes initiated days after tendon repair are intended to prevent adhesion formation and joint stiffness. In the chicken tendon cut-and-repair model, we found no adhesion formation within the first 7 days after surgery.[31] Granulation tissue or filmy adhesions were seen at day 9, but no well-formed adhesions around the tendons were detected. At day 14, loose adhesions were found around the repaired tendons, which to some degree were restrictive to tendon gliding. The gliding force and energy required to move the tendon increased progressively from day 0 to day 3, but did not increase between days 3 and 9.[31]

We found that the early postoperative changes have 3 stages: *initial* (days 0–3, increasing resistance with development of edema), *delayed* (days 4–7, higher but consistent resistance with continuing edema), and *late* (after day 7–9, hardening of subcutaneous tissue with development of adhesions) (**Box 1**, see **Fig. 1**).[31] Based on these findings and the stage classification, we suggest that mobilization be delayed to avoid most of the period of increased resistance to mobilization of the injured tendons, possibly not starting until 4 or 5 days after repair. Digital motion may be started as late as day 7 or 9 for patients who are unable to start earlier. Adhesions do not form before then, and motion in the initial few days may not affect recovery, but would only increase the pain of patients and workload of therapists.

**Box 1**
**Edema within 2 weeks post-repair and severity scores**

*Major changes of edema and resistance to the tendon*[31]

*Days 0–3*: increasing resistance with development of edema

*Days 4–7, 9*: consistent resistance with continuing edema

*Days 7, 9–14*: hardening of edematous tissues; adhesions start

*Cao and Tang edema severity score*[23] *for recording the degree of edema:*

0 (none): swelling is absent

1 (slight): swelling is minimal or slight

2 (moderate): swelling is prominent with increases in digital diameter

3 (severe): swelling with opening in skin incision

## RESISTANCE TO FLEXOR TENDON GLIDING

Resistance to tendon gliding is an important consideration, as ideally postoperative tendon motion should counteract all forces resisting digital motion without rupturing the repaired tendon. The following 10 factors are a list of factors in resistance to tendon gliding: (1) surgical repairs, (2) tendon bulkiness (tendon edema and bulkiness produced by surgical repair), (3) smoothness of tendon gliding surface, (4) healing responses of the tendons, (5) presence of intact constructive annular pulleys, (6) edema formation, (7) adhesion formation, (8) joint stiffness, (9) extensor tendon tethering, and (10) splints and bandages, and speed, frequency, and methods of postoperative motion. All these factors should be considered in designing a motion protocol and in instructing patients to follow a protocol. The protocol should be fine-tuned to counterbalance the increases in resistance to tendon motion when used in individual patients.

How edema and adhesions contribute to the resistance to tendon gliding is discussed ealier. Therefore, here the authors discuss how the presence of the flexor digitorum superficialis (FDS) tendon, major annular pulleys, the extensors, and joint stiffness contribute to the resistance to tendon gliding. Each type of surgical repair may contribute to gliding resistance of the tendon

differently; many previous papers[32–35] have discussed this subject, so will not be discussed here.

### *Resistance Caused by Repaired Flexor Superficialis Tendons*

Removal of one slip of the FDS tendon decreases the gliding resistance after flexor digitorum profundus (FDP) repair.[36] In cadaveric fingers,[37] we found that after incision of the FDS tendon proximal to, under, and distal to the A2 pulley, work of digital flexion of the intact FDP tendon decreased by 6%, 18%, and 20%, respectively, when compared with that with the FDS intact. Removal of the FDS under the A2 pulley affected the gliding of the FDP most manifestly. Removal of the FDS proximal to the A2 pulley had a less notable effect, and removal of the FDS distally did not alter the biomechanics of the FDP tendon substantially.

In a chicken tendon cut-and-repair model, we found that excision of the FDS tendon decreased resistance to FDP tendon gliding.[38] At the end of the eighth week, the excursions and work of digital flexion were better with the FDS excised than those with both tendon repairs when the tendons were cut within the pulley, and adhesions were more severe when both tendons were repaired. Those findings suggest that repairs of both FDS and FDP tendons in the area of the A2 pulley carry the risk of worsening the FDP tendon gliding. The repaired FDS, FDP, and intact A2 pulley may get stuck in some cases, allowing little tendon motion. We thus consider that if tendon injury is unclean, or tendon repair is delayed, repair of the FDS tendon together with FDP tendon may not be a wise decision. Either the FDS can be left unrepaired (or excised locally), the A2 pulley can be sufficiently vented, or both.

### *Resistance Caused by an Intact A2 Pulley*

Segmentally located annular pulleys are one of the defining features of the digital flexor tendon system. These pulleys play important roles in keeping the tendon close to the joint rotation axis during finger flexion, preventing tendon bowstringing. However, such a biomechanical advantage comes at the cost of a strict limitation of gliding space within the sheath tunnel, in particular at the sites covered by the pulleys. Movement of a normal tendon is smooth; flexor pulleys and the tendons are an elegantly coordinated press-fit system. Nevertheless, the swelling and biologic healing responses of lacerated tendons disturb this system, hindering the gliding of swollen tendons. Resistance increases when the tendon repair site is located underneath the pulleys. The A2 pulley is the longest pulley in the digits, presenting a major hindrance to tendon motion in such cases.

Over the past decade, an increasing number of surgeons have sought to judiciously vent a part of the A2 pulley to “free” the repaired tendon, and this has been a major paradigm shift in treatment of the major pulley during flexor tendon repair (**Fig. 5**).[39–43] Pulley venting during repair is intended to facilitate tendon exposure, permit smoother tendon gliding without catching on the rim of the pulley edge, make room for postoperative tendon edema, and prevent postoperative tendon entrapment by adhesions.

Previous mechanical results support venting the A2 pulley up to 50% in cadaver studies.[44–46] In an in vivo model, we found that incision of the pulley improved excursion of the FDP tendon and decreased the work of digital flexion.[47] We also noted that rupture rates of the repaired tendons were greater in the toes with an intact A2 pulley compared with those with the pulley venting at postoperative weeks 2 and 4.[48] Venting of the pulley alters the force distribution in the tendon under the rigid pulley. In fact, in a subsequent study repair strength in lacerated FDP tendons was found to increase if the A2 pulley was vented.[49]

The relative contributions of tissue edema, tendon edema, and the intact A2 pulley to resistance to tendon gliding have been studied as well.[50] At postoperative weeks 1 and 2, work of digital flexion decreased by 19% to 25% with removal of the volar 2 cm soft tissues and further

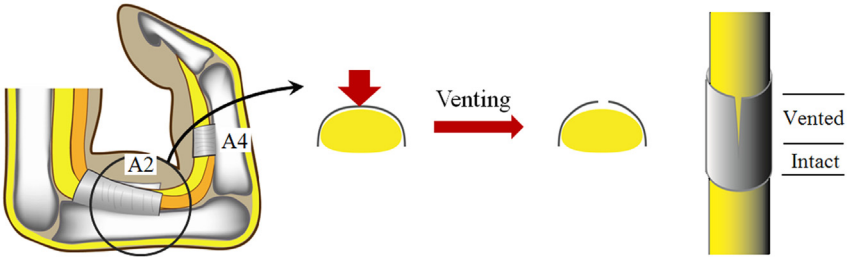


**Fig. 5.** Venting major pulleys reduces constriction to the tendon and reduces the chances of impingement of the repair site to the pulley rims. The A2 pulley can be vented to 1/2 to 2/3 of its length; a portion of this pulley is kept.

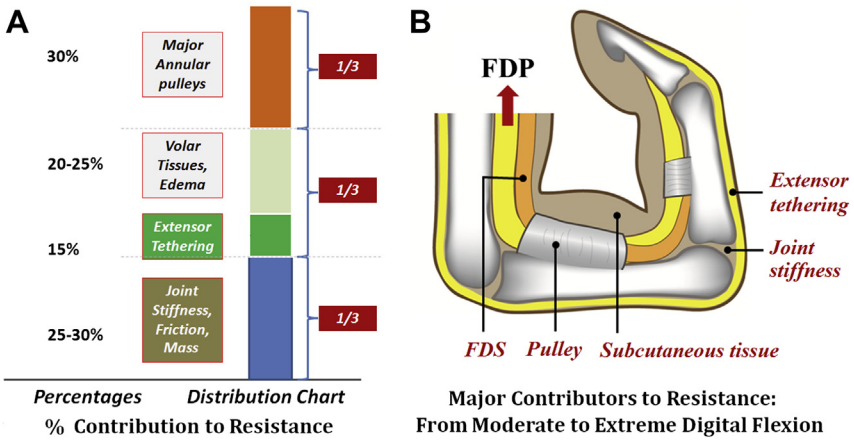


**Fig. 6.** (*A*) Percentage contribution of different factors to resistance to tendon motion. (*B*) Structures causing resistance to tendon gliding. Note that some factors can be eliminated *during surgery*: restrictive pulleys constitute about 30% of the total resistance, so venting of the constrictive portions of pulleys would decrease the resistance. Influences from other factors, such as extensor tethering or joint stiffness (contributing 30%–35% of total resistance), can be reduced by *repeated passive digital motion* before active tendon motion.

decreased by 30% to 34% with division of the A2 pulley. In other words, presence of an intact A2 pulley adds greater resistance than volar edematous subcutaneous tissue.

### *Effect of Joint Stiffness and Extensor Tethering*

Little has been published regarding the effect of joint stiffness and extensor tethering on tendon gliding after surgery. Although these 2 factors have no direct bearing on tendon surgery, they do contribute to tendon gliding resistance. In a chicken model, we sequentially removed the volar subcutaneous tissue, extensor tendons, and flexor pulleys to try to understand the relative contribution of each of these surrounding tissues to tendon gliding resistance. Constrictive pulleys accounted for about 30% of all the resistance to digital flexion when the FDP tendon was injured; the subcutaneous tissue 10% to 25% (about 10% in normal toe and 20%–25% in injured FDP tendon); the FDS tendon about 10%; and the remaining portion (35%) should come from the combined effects of extensor tethering, joint stiffness, and mass of the digit (**Fig. 6**).

Finally, we must emphasize that finger joints are inherently small, and development of stiffness is frequent. At any phase along the flexion arc, if stiffness of these joints presents an obstacle to active tendon movement, the entire scale of contributing factors would be changed. Joint stiffness would become the overwhelmingly major resistance to tendon gliding. Each factor's contribution to the resistance of tendon gliding is a dynamic process, which varies depending on joint position, relative position of the tendon repair site to the pulleys, and preconditioning of the tendon and adhesion tissues.

The overall resistance to tendon motion is progressively increased when the digit is extremely flexed (**Fig. 7**). Extreme active flexion of the fingers would (1) cause the impingement of repair sites to the sheath or pulley rims, (2) increase the bulkiness

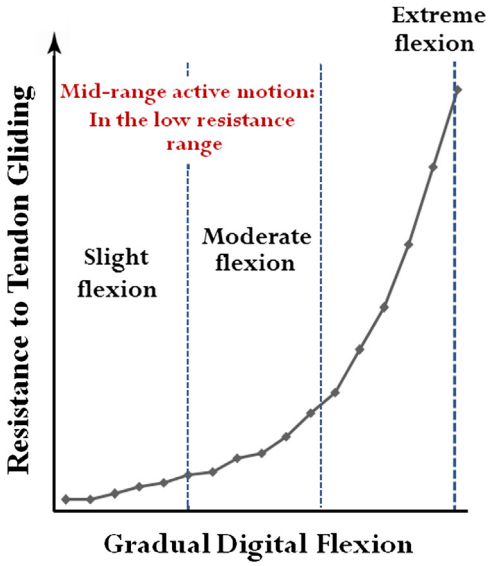


**Fig. 7.** The active digital motion creates a dynamic loading situation in which tendons are consistently subjected to changing forces from digital extension to flexion. Extreme flexion of the fingers greatly increases resistance to flexor tendon gliding. Therefore, in the early postoperative period, midrange active motion is in the low-resistance range and is preferable.

of tendon, (3) narrow the tendon gliding tunnel, (4) increase the tethering of the extensor mechanism, (5) tighten the capsule of the digital joints, and (6) increase the compression of edematous subcutaneous tissues. In the early postoperative period, midrange active motion is in the low-resistance range and is preferable in active motion of the fingers.

Therefore, to maximize functional recovery it is important to take all these factors into consideration when judging the dynamic relationship in designing individualized motion protocols.

## SUMMARY

Early flexor tendon healing is characterized by peak cellular apoptosis of both inflammatory and tendon cells in the first week, followed by progressively greater tenocyte proliferation in the second and third weeks. Tenocyte apoptosis is a predominant event in the middle and late healing periods, contributing to tendon remodeling especially restoration of gliding surface. Proliferation of tenocytes is minimal in these stages. Apoptosis of cells is very prominent in adhesions and at the tendon-adhesion gliding interface. Edema in subcutaneous tissue and the tendon is an inevitable biologic process, contributing substantially to the resistance to tendon gliding. Major annular pulleys may greatly resist gliding of a repaired and swelling tendon. Experimentally, edema of the subcutaneous tissues contributed 20% to 25%, the intact A2 pulley 30%, extensor tendons 15%, and joint stiffness etc of 25% to 30% of total resistance to the gliding of the flexor tendon. Tendon bulkiness (swelling and surgical repairs) also greatly contributes to resistance. The contribution made by each of the earlier mentioned factors changes dynamically with time elapsed since surgery and the position of finger flexion. The overall resistance to tendon motion is progressively increased when the digit is markedly flexed. Careful consideration of the contributing factors and their dynamics provides insight into strategies to reduce repair rupture and maximize tendon gliding through surgery and postoperative motion protocols.

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