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Physiology of Flexor Tendon Healing and Rationale for Treatment Protocols

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Abstract

The hand functions as both a vital tactile organ and a grasping mechanism that is tempered by finely controlled accuracy. Essential to these functions are the delicate movements of the extrinsic flexor tendons. Repair of the injured flexor tendon in the hand to achieve normal function remains a difficult task, and controversy exists as to what postoperative rehabilitation protocols should be utilised. This chapter will focus on the pathophysiology of repair when flexor tendons are ruptured, the unique anatomy of flexor tendons, the latest molecular updates, repair principles, initial management procedures and the rationale for the various postoperative rehabilitation protocols that are used.

Keywords: flexor tendon injury, rehabilitation, tendon healing, molecular updates, complications

1. Introduction

An accurate understanding of the physiology of flexor tendon healing is essential to maximising patient outcomes and justifying the current treatment regimens of surgical repair and postoperative rehabilitation protocols. Our current understanding of flexor tendon healing is a continually evolving area. Therefore, this chapter aims to instruct the reader of the current understanding of flexor tendon basic science, the latest molecular updates, justifications for various surgical and rehabilitation regimens and future research trends. The reader is strongly encouraged to seek alternative resources for more detail regarding flexor tendon anatomy as this will be covered briefly in this chapter. Secondary flexor tendon reconstruction will not be discussed.

2. Macroscopic flexor tendon anatomy

This section will focus only on the flexor sheath and vascular supply. The reader is strongly encouraged to seek the vast array of anatomy texts to familiarise themselves with flexor tendon anatomy, paying attention to the:

- Flexor digitorum superficialis (FDS)

- Flexor digitorum profundus (FDP)

- Flexor pollicis longus (FPL)

- Flexor sheath and pulley system

- Vascular supply

2.1 Flexor sheath and pulleys

The extrinsic flexor tendons of the hand possess true fibro-osseous tunnels in the digits, called the "flexor sheath". Their purpose is to provide very efficient lubrication in an area subject to a change of direction and increase in friction [1]. Proximal to the metacarpophalangeal (MCP) joints, the flexor tendons enter the flexor sheath. This tunnel functions to hold the tendons in close proximity to the phalanges to prevent "bowstringing" and to increase the efficiency of tendon glide [2].

Condensations in the sheath are called pulleys—these almost encircle the flexor tendons to form a fibro-osseous channel that keeps the tendons adjacent to the phalanges [3]. In effect, the pulleys enable the transfer of a translational force generated from the muscle tendon unit into a rotational moment on the phalanges [3]. Pulleys are classified on the basis of their shape—annular or cruciate. There are five annular pulleys (named A1–A5 from proximal to distal) and three cruciate pulleys (named C1–C3 from proximal to distal). The A2 and A4 pulleys insert directly onto the bone over the proximal and middle phalanges, respectively [3]. Traditionally, A2 and A4 are considered to be the pulleys that prevent bowstringing. However, it has now been shown that partial distal excisions of 25% of the A2 pulley, up to 75% of the A4 pulley and 25% of combined A2 and A4 have no significant effect on digit range of motion or work of flexion [4, 5]. The A1, A3 and A5 pulleys are located over the MCP, proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints, respectively [3]. Proximal to the A1 pulley is the palmar aponeurosis (PA) pulley which has been implicated in the aetiology of trigger finger [6].

The cruciate pulleys lie between the A2–A3, the A3–A4 and the A4–A5 pulleys, respectively [3]. These pulleys function like accordions, allowing the sheath to expand and compress with flexion and extension.

Traditionally, it was once thought that the thumb had three pulleys—A1, oblique and A2 [3]. The A1 pulley lies over the MCP joint, the oblique pulley runs from proximal ulnar to distal radial over the proximal phalanx and the A2 pulley is located over the interphalangeal (IP) joint [3]. A fourth pulley, the variable annular pulley, was first reported in 2012 where it was found to be present in 93% of cadaver specimens [7]. It can have three orientations—transverse, oblique or continuous [7].

2.2 Vascular supply of the extrinsic hand flexors

Both FDP and FDS tendons in the digits receive dual nutritional supply from vascular perfusion and synovial diffusion [8]. There is some variation in the vascular system, but, generally speaking, the flexor tendons receive their blood supply via two vincula each—a short and long vinculum [9]. Vincula are folds of mesotenon carrying blood to the tendons [9]:

- The vinculum longus superficialis (VLS) arises from the radial or ulnar side of the base of the proximal phalanx. It receives its blood supply from the

transverse communicating branch of the digital arteries at the base of the proximal phalanx.

- The vinculum brevis superficialis (VBS) arises from the volar plate of the PIP joint and attached to the decussation of the FDS. The source vessels are from the proximal transverse digital artery.

- The vinculum brevis profundus (VBP) arises along the distal two thirds of the middle phalanx (whose source arteries are from both the interphalangeal and distal transverse digital arteries) to insert dorsally on the FDP.

- The vinculum longus profundus generally originates at the level of insertion of the FDS tendon (through the decussation of the FDS) and attaches to the FDP directly, and its source is the proximal transverse digital artery.

3. Microscopic aspects of flexor tendon anatomy

3.1 Collagen

Tendons consist of mostly Type I collagen and elastin embedded in a proteoglycan-water matrix [1]. Collagen contributes 65–85% of the dry mass of the tendon [1]. The collagen, elastin and proteoglycan-water matrix are formed by tenoblasts and tenocytes (refer to Section 3.4). These cells are elongated fibroblasts and fibrocytes which lie between the collagen fibres and are organised in a complex hierarchal scheme to form the tendon proper [10]. Soluble tropocollagen molecules form cross-links to create insoluble collagen molecules, which then aggregate progressively into microfibrils and then into visible units under the electron microscope called collagen fibrils [1].

3.2 Collagen fibres, fibre bundles and fascicles

The collagen fibrils in turn aggregate together to form the basic tendon unit—the collagen fibre. The collagen fibre is defined as the smallest tendon unit visible using light microscopy [1]. Aggregates of collagen fibres form a primary fibre bundle called a subfascicle, and a group of primary fibre bundles form a secondary fibre bundle called a fascicle. A group of secondary fascicles in turn form a tertiary bundle; it is the tertiary bundles that contribute to the full tendon and are surrounded by epitenon (refer to Section 3.3).

Both the fascicles and tertiary tendon bundles show a spiral formation along the course of the tendon [1]. In the resting state, the collagen fibres and fibrils show a wavy configuration that appears as regular bands across the fibre surface [11]. This configuration disappears when the tendon is stretched—here the collagen fibres straighten. When the stretching forces are removed, the tendon resumes its normal wavy appearance. If an acute stress causes an elongation of 8% or more, the tendon is likely to rupture [1].

Fibres along the tendon are not only parallel. Jozsa et al. [12] demonstrated that there are five types of fibre crossings—parallel running fibres, simply crossing fibres, crossing of two fibres with one straight running fibre, a plait formation with three fibres and an up-tying of parallel running fibres with one fibre. The ratio of longitudinal to transverse running fibres ranges between 10:1 and 26:1 [13]. Within one collagen fibre, the fibrils are oriented longitudinally and transversely.

The longitudinal fibres not only run parallel but also cross each other to form spirals [13].

The complex microstructure of the tendons correlates with their function to transmit the force created by the muscle to the bone and to make joint movement possible. During phases of various movements, the tendons are exposed to a number of forces—longitudinal, transversal and rotational as well as withstanding an array of pressures. Therefore, the internal structure of the tendon described serves as a buffer against forces of various directions, thus preventing damage and disconnection of the fibres [13].

3.3 Epitenon and endotenon

An entire flexor tendon is surrounded by a fine connective tissue sheath called the epitenon. Histologically, the epitenon consists of relatively dense network of collagen with strands of 8–10 nm in thickness [1]. It contains longitudinal, oblique and transverse fibrils. The outer surface of the epitenon is contiguous with the flexor sheath and inner surface with the endotenon. The endotenon resides inside the tendon; it invests each tendon fibre and also binds individual fibres as well as larger fibre bundles. In contrast to the epitenon, the endotenon consists of a thin reticular network of connective tissue inside the tendon with a crisscross pattern of collagen fibrils [1, 11]. The functions of the endotenon are to [10, 11]:

- Bind tendinous collagen fibres.

- Allow fibre groups to glide on each other.

- Carry blood vessels, nerves and lymphatics to the deeper portion of the tendon.

3.4 Tendon cells

Tendon cells are either tenoblasts or tenocytes which comprise 90–95% of the cells of the tendon [1]. The other 5–10% are chondrocytes (at the pressure and insertion sites), synovial cells of the tendon sheath (on the tendon surface) and vascular cells (capillary endothelial cells and smooth muscle cells of the arterioles). In pathological conditions, other cells can be observed in the tendon tissue such as inflammatory cells, macrophages and myofibroblasts [13].

Tenoblasts and tenocytes represent differing maturations of the tendon cell. Newborn tendons are called tenoblasts and have different shapes and sizes. In young individuals, the tenoblasts begin to resemble each other being spindle shaped. In adults, the cells are called tenocytes and are very elongated [13]. Tenoblasts and tenocytes are metabolically active cells and synthesise collagen and other matrix components [13, 14]. The metabolic pathways utilised for energy production change from aerobic to anaerobic with increasing age [10, 13]. The low metabolic rate of the tendon tissue, in addition to well-developed anaerobic energy production, is essential for the function of the tendon to carry loads and remain in tension for periods of time without the risk of ischaemia or necrosis [1]. A likely drawback of this low metabolic rate is the slow rate of recovery and healing after injury [15].

4. Flexor tendon healing: cellular concepts

In the 1960s, flexor tendon healing was thought to rely on the invasion of peripheral cells and blood vessels which lead to the formation of restrictive

adhesions [16, 17]. This concept was contested over the next two decades when bodies of biologic and molecular evidence confirmed that tenocytes actively participate in tissue repair and that tendons are capable of healing from injury [18]. Tendon healing undergoes overlapping inflammation, proliferation and remodelling [19] via two mechanisms—extrinsic and intrinsic [8]. The proliferation of tenocytes and production of their extracellular matrix are the hallmark of the intrinsic process [20, 21]. Extrinsic healing on the other hand involves the invasion of fibroblasts and inflammatory cells into the site of injury from the surrounding synovium, paratenon and tendon sheath [8, 22].

4.1 Intrinsic healing

Intrinsic healing involves only the tenocytes (fibroblasts) within the tendon itself and depends on the migration and proliferation of cells from the epitenon and endotenon [8, 22]. Epitenon tenocytes produce collagen earlier than those of the endotenon. Tenocytes of the endotenon produce large and more mature collagen than epitenon cells. In any event, both endotenon and epitenon tenocytes establish an extracellular matrix and internal neovascular network. Intrinsic healing results in improved biomechanics within the sheath, including tendon gliding. Movement of the tendon within the sheath improves synovial circulation and therefore the delivery of nutrients.

4.2 Extrinsic healing

Extrinsic healing involves the invasion of fibroblasts and inflammatory cells into the site of injury from the surrounding synovium, paratenon and tendon sheath [8, 22]. This produces scarring and peritendinous adhesions which may impair tendon movement, gliding and nutrition (refer to Section 4.5). It is thought that extrinsic healing predominates in the earlier stages of tendon healing. Extrinsic healing also predominates when tendons are immobilised after injury or repair. The extrinsic mechanism is activated earlier and is responsible for initial adhesions, the highly cellular collagen matrix and the high-water content of the injury site [8, 22]. The intrinsic mechanism then causes tenocytes from within the tendon to invade the defect and produce collagen which reorganises and aligns longitudinally to maintain fibrillar continuity and produce a healed tendon [23].

4.3 Early healing stage

After tendon injury, two intricately related and balanced processes take place—tenocyte apoptosis and tenocyte proliferation [18, 24]. Wu et al. [25] specifically examined apoptosis and proliferation of a repaired digital flexor tendon in a chicken model. In uninjured tendons, only $3 \pm 2\%$ of the tenocytes showed signs of apoptosis, and $1 \pm 1\%$ showed signs of active proliferation. The percentage of apoptotic cells went up to more than 40% at days 3–7 after tendon injury; on day 3, the number of inflammatory cells in the wound site also peaked. The number of mainly inflammatory cells as well as tenocytes peaked during the very early days in the healing process (at days 3 and 7) in the chicken model. In addition, the number of proliferating cell nuclear antigen cells (PCNA) and Bcl-2 (an antiapoptotic protein) —markers of proliferation—did not significantly increase until day 7 and peaked during days 7–21. Thus, it was established that tenocyte apoptosis is accelerated within several days after injury, followed by increase in proliferation of tenocytes in 2–4 weeks with activation of molecular events to inhibit apoptosis.

4.4 Middle and late healing stages

Wu et al. then further quantified cell apoptosis and proliferation during the middle and late stages of healing [26]. The percentage of apoptotic tenocytes was generally higher on the surface of the tendon than that in the core, indicating a greater need for cellular clearance and surface remodelling in the surface region in the middle-to-late periods. Their findings also indicated that active tendon remodelling persists through the very late tendon healing period, especially on the surface because:

- The total cell population did not start to decline until after day 56 (2 months). The percentage of apoptotic tenocytes ranged from 30 to 40% in the total cell population.

- Cell apoptosis persisted at a relatively high level on the tendon surface at 3 months.

- Cell apoptosis in the core region declined after 2 months.

In sharp contrast to apoptosis, proliferation of tenocytes in the middle and late healing stages [26]:

- Declined drastically after week 4 (less than 5% of the PCNA-positive cells were found in the tendon).

- PCNA-positive cells were at normal levels at weeks 8–12.

The above two points indicated that tenocyte apoptosis is the dominant event in the middle and late tendon healing period.

In areas distant from the junction site, apoptosis is more prominent in the tendon surface than in the tendon core—this is thought to be associated with the clearance of excess cells, which serves to promote formation of smooth gliding surfaces by remodelling adhesions [26].

4.5 Adhesions

The primitive processes by which tissues repair after injury are indiscriminate to tissue types and lead to fibrotic scarring [27]. Flexor tendon tissue is not exempted from this. Injury to flexor tendons through trauma or surgery can result in problematic tendon adhesion formation. Adhesions affect the normal tendon gliding that occurs within a narrow flexor tendon sheath. Fibreoptic studies of surgical patients have demonstrated that the tendons, sheath, soft tissues and skin glide across each other in vascularised interconnecting tissue planes during finger flexion, with scarring of these planes affecting the fingers' ability to flex [28]. When two dynamic gliding planes are affected by injury, such as the tendon and sheath, the result is adhesions [28]. A landmark 1960 study by Lindsay and Thomson [29] had shown that immobilisation was key to adhesion formation after systematic wounding of the tendon, sheath, skin, soft tissue and vinculum complex. Further studies have shown that damage to the skin, sheath, soft tissues and vinculum alone is insufficient to form adhesions [28]. Additionally, keeping the damaged tendon and damaged soft tissue in relatively close approximation appears to be required for adhesions to form [29]. For these reasons, early active mobilisation is encouraged following tendon surgical repair [30].

Using a murine model, Wong et al. [28] demonstrated that the scarring between two damaged surfaces—i.e. the extrinsic healing process—was responsible for adhesions due to the increased inflammatory activity in the tissue surrounding the flexor tendons. They found that [28]:

- Adhesion formation was propagated by immobilisation of the digit.

- Inflammatory cells predominated in the surrounding tissues in the early phases of healing but appeared in the tendon proper during the remodelling phase.

- Proliferative activity occurred in both the surrounding tissues and tendon but was greater in the surrounding tissues.

- Collagen synthesis in the tendon and subcutaneous tissue is *temporally* different.

- Pericyte and myofibroblast activity predominates in the subcutaneous tissue and not tendon.

- In adhesion forming tendon wounds after 21 days, there were two distinct cell phenotypes observed. The first was a large cell with multiple cytoplasmic protrusions, which were seen to enclose large and small diameter fibrils or even multiple fibrils. The second phenotype was similar to those seen in the developing tendon, with small cytoplasmic protrusions and small fibrils being deposited by "fibripositors" (small fibrils in embryonic tendon fibroblasts which are enclosed in cytoplasmic processes). The number of fibripositors was greater than those seen in development.

What is clear is that the interactions between the damaged tissues and the processes that lead to adhesions are complex. The varying multicellular temporal and spatial expression involved in flexor tendon healing is far more intricate than that proposed by the intrinsic and extrinsic concepts of healing alone.

4.6 Microdynamics of adhesions at different stages of tendon healing

The mechanical characteristics of adhesion tissues determine tendon gliding, but this relationship is difficult to ascertain. Wu et al. [18] performed an in vivo study to determine the microdynamic features of adhesions in the middle and late healing periods (postoperative weeks 4–8). They found that the ability of adhesion tissues to resist tension decreased over time, whereas their flexibility increased; they hypothesised that this phenomenon determined the sliding amplitude of the tendon.

It was also found that, in a chicken toe flexor tendon that was surgically repaired and immobilised for 3 weeks, the percentage of apoptotic cell increased from the tendon core, to the tendon surface, to the adhesion-tendon interface and to the adhesion core [26]. Furthermore, tendons with more severe adhesions, i.e. those with less excursion, see greater apoptosis in their adhesions and adhesion-tendon interfaces.

In summary, it appears that the microdynamics of adhesions and tenocyte apoptosis are associated—*as apoptosis of the cells in the adhesions continues, the adhesions are more easily broken up after the adhesions are loaded* [18]. This would help, in part, explain why early active mobilisation is beneficial after tendon repair. Wu et al. [18] hypothesise that the external force applied to move the tendon during digital motion transfers to shear force over the adhesions and adhesion-tendon gliding

interface. This continuously stimulates cellular apoptosis, in turn reducing the density and strength of the adhesion fibres, resulting in an increasingly greater elasticity and breakup of the adhesion fibres in the late healing stage.

4.7 Research trends

Recent research has examined methods to augment intrinsic biological healing of the flexor tendon whilst minimising adhesions.

- **Transforming growth factor β (TGF-β)**: Small amounts of TGF-β are found in the uninjured tendon [31]. The isoform TGF-β1 increases significantly after tendon injury [32, 33]. TGF-β1 has the highest association with adhesion formation and is therefore a major treatment target [31]. A neutralising antibody to TGF-β was shown to control scarring in rat dermal wounds [34, 35]. Furthermore, the same antibodies increased the total range of motion after flexor tendon repair in a rabbit model [36]. Additionally, the TGF-β1 receptor inhibitor SD208 can prevent progression and can improve tendon mechanical strength and decrease rupture rates [31]. However, suppression of TGF-β has been shown to decrease strength of tendon repair [37, 38]. This seems to be supported by gene therapy studies. Decreased TGF-β was examined by deleting the TGF-β inducible early gene (*Tieg1*)—this resulted in decreased collagen I deposition in an in vitro model of tendon healing [39].

- **Vascular endothelial growth factor (VEGF)**: It is known that tenocytes secrete VEGF and are present in synovial fibroblasts [8]. The VEGF family consists of several isoforms (VEGF-A, VEGF-B, VEGF-C, VEGF-D and VEGF-E and placenta growth factor), and these isoforms exert their effects through 3 tyrosine kinase receptors [31]. VEGF has been implicated in wound healing through epithelialization, collagen deposition and angiogenesis [40]. During flexor tendon repair, Boyer et al. showed that VEGF mRNA is increased during flexor tendon repair. It is postulated that the increased VEGF expression is associated with neovascularization [31]. VEGF genes delivered by adeno-associated virus (AAV) vectors in a chicken model demonstrated that healing strength was improved without increased adhesion formation [41].

- **Basic fibroblast growth factor (bFGF)**: bFGF, found within the tendon and tendon sheath, has been shown to influence wound healing due to its role in fibroblast chemotaxis, proliferation and angiogenesis [42]. However, its role in tendon healing remains unclear. Delivery of bFGF to injured tendons via adenoviral vector demonstrated improved tendon healing and increased strength with reduced adhesions [42].

- **Tissue engineering**: In their study, using a devitalised acellular allograft tendon containing recombinant AAV expressing growth and differentiation factor-5, Basile et al. [43] were able to repopulate the graft, decrease scar tissue and enhance the gliding property relative to the control graft. Tissue-engineered synovial membranes [44] have also been shown to decrease peritendinous adhesions.

5. Flexor tendon repair principles

This section will focus on repair principles. The reader is encouraged to seek alternative resources regarding specific repair techniques.

All suture tendon repair methods have been shown to significantly increase the gliding resistance compared to the intact tendon [45]. Gliding resistance is affected by [18, 46, 47]:

- The number of exposed suture loops and knots outside on the tendon surface

- The suture calibre

- The suture material

- Tendon bulkiness (from both oedema and surgical repair)

- Smoothness of tendon gliding surface

- The presence of intact annular pulleys

- Oedema

- Adhesions

- Joint stiffness

- Repaired flexor digitorum superficialis

Therefore, the ideal method of flexor tendon repair should allow a healing response precisely at the tendon ends but not between the tendon and its surroundings, create a repair site with minimal bulk and low friction and place enough force across the repair to promote motion and remodelling [22].

Strickland described the characteristics of an ideal tendon repair [48], and these were supported by further studies [22]. These are:

- Core sutures easily placed in tendon

- Secure knots

- Smooth junctions

- Minimal gapping

- Minimal interference with tendon vascularity

- Sufficient strength to permit application of early motion stress during the healing process

- Motion at the repair site to increase the amount of collagen deposited at the site of injury

- Equal tension across all suture strands

5.1 Repair strength

Initially, the strength of tendon repair depends solely on the repair technique [45]. It is postulated that postoperative tenomalacia may develop at the suture

tendon junction, therefore decreasing initial repair strength [49]. The initial strength of the repair depends on the material properties and knot security of the sutures as well as on the holding capacity of the suture grips of the tendon [45]. Immobilisation significantly decreases the strength of repair within the first 3 weeks of healing [50], whereas early passive and early active motion have been shown to prevent the initial weakening, leading to progressively increased repair strength, starting from the time of repair [50–52]. The initial strength of the repair depends on the material of the suture itself, knot security of the suture and the holding capacity of the suture grips on the tendon [45]. Therefore, the biomechanical properties of the suture can be improved by:

- Increasing the number of strands crossing the repair site [53]

- Increasing the suture calibre [54]

- The number, size and configuration of the grips [45, 53]

5.2 Suture terminology

The flexor tendon repair is a composite of the *core* and *peripheral* sutures [55]. The core suture is the suture placed within the substance of the tendon proper and consists of at least two of three components—longitudinal, transverse and link. All core suture techniques have a longitudinal and link component.

- The link component is that part of the suture at the junction between longitudinal and the transverse components or between two longitudinal components. The link component lies outside the tendon.

- The longitudinal and transverse components are usually placed within the tendon substance, i.e. they are intratendinous.

- The transverse and/or link components convert the longitudinal pull of the suture to a transverse compressive force and prevent the longitudinal component from pulling out.

- The longitudinal component in turn allows placement of the transverse and/or link components away from the divided end of the tendon.

Pennington [56] first described the relationship of the transverse and longitudinal components when he outlined his locking-loop technique. *Locking* suture configurations tighten around bundles of tendon fibres with tension [56]; it can only do this when the transverse component crosses just superficial to the longitudinal part of the suture. The result is a loop of suture locking around a small bundle of tendon fibres so that when more tension is applied to the repair site, the tighter the grip of the suture loop on these fibre bundles [56]. *Grasping* loops on the other hand have the transverse component passing deep to the longitudinal constituent so that the suture does not pass around or lock a bundle of tendon fibres [57]. Locking loops improve the ultimate force and gap resistance compared to grasping loops in flexor tendon repair [45]. Several studies have demonstrated that locking loops improve the ultimate force and gap resistance compared to grasping loops in flexor tendon repair [45, 58]. However, the biomechanical advantage of the locking loops is obtained only with 3–0 or larger suture [45]. This is because with 4–0 suture, the material strength is inferior to the holding capacity of the suture grips of the tendon

leading to failure by suture rupture before the true biomechanical properties of the locking loops are obtained [45]. Additionally, the size of the locking loop influences the biomechanical properties of the repair technique [59–61]. In the modified Pennington technique, increasing the cross-sectional area of each loop from 5 to 15% improved the ultimate force, whilst further increase did not improve strength, and the tendency for gap formation increased [60]. In the four-strand cruciate repair, the locking loops of 25% reached the highest gap force, ultimate force and stiffness [59].

Variations in the construction of the link component—arc, loop or knot—result in a *sliding* or an *anchored* suture on each half of the divided tendon [55].

- A sliding suture allows the suture to slide within the tendon substance when tension is applied to one of the longitudinal components. An arc link component results in sliding suture. When sliding sutures are used, tension is equally distributed among the different longitudinal strands.

- An anchored suture does not allow the suture to move independent of the tendon. A knot link component results in an anchored suture. When anchored sutures are used, the longitudinal strands are fixed. However, any slack in the suture will result in uneven distribution of tension and gapping at the tendon ends.

5.3 Suture principles

The length of the core suture purchase in the tendon logically determines how much of the segment of the tendon is incorporated into the repair. *The optimal range of core suture purchase has been determined as 1.0 cm with increased gap force, ultimate force and stiffness* [62, 63]. The purchase of 0.4 cm results in very weak repairs, whilst any increase over 1 cm does not improve the biomechanical properties [63].

Increasing the suture calibre has been shown to increase the ultimate force in static testing and fatigue strength in dynamic testing; however, it has not been shown to improve the yield force or gap resistance of the repairs [45]. The strength of the 4–0 suture has been reported to be less than the holding capacity of several locking and grasping repair techniques with failure occurring mostly by suture rupture [54, 64]. A 3–0 suture failure due to suture rupture and pullout has been reported [54, 64]. Therefore, the use of 3–0 suture is generally recommended to offer safety over the 4–0 suture by increasing the material strength [45, 54, 64].

The *ideal suture material* for flexor tendon repair should be strong enough; prevent gapping; be easy to use and knot; be absorbable but maintain its tensile properties until tendon repair has achieved adequate strength; and have minimal tissue response [65]. Non-absorbable, synthetic sutures, (especially coated braided polyester), monofilament nylon and monofilament polypropylene are used in flexor tendon repair [45]. Coated braided polyester suture is the most common core suture material, though nylon is also used, especially in repairs performed with looped suture. Monofilament polypropylene is mainly used in the peripheral sutures. Coated braided polyester suture demonstrates significantly higher tensile strength and stiffness than monofilament nylon and polypropylene sutures and maintains its tensile properties in the body temperature, whilst the stiffness of both polypropylene and nylon suture has been shown to decrease significantly [66, 67]. A braided polyblend polyethylene suture (Fiberwire®) has been introduced for flexor tendon repair. It has significantly higher ultimate force and stiffness than coated braided polyester, monofilament nylon and polypropylene sutures and a similar ultimate force but higher stiffness than braided stainless steel [66]. Bioabsorbable suture

materials are not widely used in flexor tendon repair due to the lack of sufficient tensile strength half-life and potential increased tissue reaction and adhesion formation [45].

The original *peripheral or epitendinous* suture was thought of a "tidying up" suture to improve tendon gliding within the flexor sheath [68]. It has now been shown that the peripheral suture improves the gap resistance and strength of repair [45, 58]. The simple running peripheral suture is the most investigated and used technique in flexor tendon repair because of its simplicity [45]. The strength and stiffness of the running peripheral suture can be increased by:

- Taking deeper suture grasps [69]

- Increasing suture purchase from 1 to 2 or 3 mm [70]

- Increasing the number of suture passes [71]

The location and number of *knots* influence the strength of the tendon repair [72]. Ex vivo studies show that decreasing the number of knots and placing them outside the repair on the tendon surface increase the strength of the repair compared to knots placed between the tendon ends [45]. However, in in vivo studies, the knots placed inside the repair sites were stronger than those outsiders after 6 weeks [72].

6. Postoperative rehabilitation following flexor tendon repair

An understanding of the postoperative rehabilitation regimen after flexor tendon repair is of equal importance to the repair itself. Noncompliance with rehabilitation may lead to poor outcomes including repair rupture, decreased range of motion and joint stiffness. Current postoperative protocols for patients with flexor tendon injuries are immobilisation, early passive mobilisation and early active mobilisation [73, 74].

6.1 Immobilisation

The benefits of early mobilisation on the repair strength, tenocyte healing and formation of adhesions are widely known [22, 75–79]. Immobilisation, however, has its role in certain situations, particularly in patients who are noncompliant with early mobilisation protocols, paediatric patients, patients with cognitive deficits and patients with concurrent injuries that may be worsened with early active mobilisation (fractures, nerves and vessels) [73, 74].

It is difficult to encourage early mobilisation in children under the age of 6 [80]. O'Connell et al. showed outcomes were equal among children who were immobilised and those who underwent early mobilisation for 4 weeks [81]. However, immobilisation for more than 4 weeks resulted in functional deterioration of the repaired tendon [81].

The protocol of Cifaldi, Collins and Schwarze may be used for the noncompliant adult [73, 82]. This protocol involves 3–4 weeks of immobilisation in a forearm-based dorsal splint or cast (20° wrist flexion, MP joints in 50° flexion and the IP joints in full extension) followed by a weaning programme (it may also be used in children) [74, 82]. "Weaning" refers to modifying the splint in such a way that the wrist is in neutral and then instructing the patient to remove the splint every hour to passively flex and extend the injured digit for 10 repetitions. Splint wear is then

discontinued at 6 weeks. From here, differential FDS and FDP gliding exercises are performed every hour for 10 repetitions [74]:

- To isolate FDP gliding, both the MP and PIP joints are held in extension, and the patient flexes the distal interphalangeal (DIP) joint. This prevents FDS glide.

- The FDS tendon glide exercise is achieved by isolating all fingers in extension, whilst the patient actively flexes the PIP joint of the affected finger. Holding the fingers in extension ensures that the common muscle belly of the FDP is held to its full length, preventing it from assisting in flexion.

- At postoperative week 8, sustained grip activities are added to the programme with resistance increasing over the next 4 weeks. Heavy resistive exercises are avoided before 12 weeks due to the risk of tendon rupture.

6.2 Early passive mobilisation

The inhibition of adhesion formation, promotion of intrinsic healing and production of a stronger repair can be encouraged with early passive mobilisation [77–79, 82–84]. The best known early passive mobilisation protocols are the Duran and Houser and Kleinert regimens [73, 74].
In the Duran and Houser protocol:

- A postoperative dorsal blocking splint holds the MP joints at 50° of flexion and the wrist at 20° of flexion. The following regimen is followed twice daily to ensure that 3–5 mm of tendon excursion occurs to prevent firm tendon adhesions [73].

- The patient uses the opposite hand to bring the PIP and the DIP joints from full flexion to full extension. This is done for eight repetitions for each joint.

- Then, the patient performs eight repetitions of composite MP, PIP and DIP flexion. The protocol continues through the fourth postoperative week.

- At 5 weeks, the patients begin active extension exercises with the use of a wristband. A rubber band is attached from the tip of the finger to the wristband, providing passive flexion and active extension. During this time, the patient also performs blocking and FDS gliding exercises.

- The late stage begins 8 weeks postoperatively. Progressive strength building is encouraged.

In the Kleinert protocol:

- A dorsal plaster splint is applied immediately at surgery. This splint blocks the wrist and MP joint in flexion. The wrist is placed at approximately 45° of flexion, the MP joints rest at approximately 20° of flexion and the IP joints are in neutral.

- One week following surgery, the plaster is replaced with a thermoplastic splint that maintains the same flexion angles as above.

- The new splint allows for passive flexion of the digits and active extension of the digits against dynamic traction using rubber bands to facilitate the traction mechanism. These bands are placed on the volar aspect of the splint and directed towards the distal nail plate from just proximal to the wrist.

- Early passive ROM exercises are started within the dorsal splint.

- At 1 month, the splint is removed, and active flexion and extension exercises begin. However, the dorsal splint must be worn when these exercises are not being performed.

- At 6 weeks, the dorsal splint is discontinued, and blocking exercises commence.

- Two months following the repair, resistive exercises are incorporated into the regimen.

- Resumption of normal activities occurs approximately 3 months following the surgical repair.

A major issue with the Kleinert protocol is the development of flexion contractures of the PIP joint [85]. These can be treated with continued intermittent splinting of the IP joints in neutral [86]. In recent years, rubber band traction has been almost completely abandoned, largely because of the problems arising from the flexed resting position of the PIP joint [87].

Continuous passive motion (CPM) uses devices that allow for joints to move through a predetermined arc of motion [73]. The goal is to increase the duration and repetition of exercises. A randomised control comparing traditional early passive motion to CPM exercises [88] showed that, at 6 months, the CPM group had significantly greater range of motion. However, further research in evaluating the CPM following flexor tendon repair is lacking.

6.3 Early active mobilisation

An early active mobilisation (EAM) protocol refers to active contraction of the repaired muscles [89, 90]. EAM has been shown to promote the formation of large diameter fibrils, and it demonstrates the greatest cellular response to injury [83]. There are many different EAM regimens in the literature [91, 92]. Gratton [93] combined the Belfast and Sheffield practices [89] to form a widely used regimen:

- A thermoplastic dorsal blocking splint is applied at postoperative day 2–5 with the wrist positioned in 20° of flexion, the MCP joints in 80° of flexion and the IP joints in full extension. Active ROM exercises are delayed until day 5 if there is significant oedema which should be treated with compression and elevation.

- In the absence of significant oedema, exercises begin with passive flexion of the digits and active extension to the constraints of the dorsal splint.

- At the completion of the above exercises, active flexion exercises begin. Here, a finger of the opposite hand is placed in the palm of the affected hand, and the patient flexes the affected fingers against the contralateral fingers aiming to progress one finger width per week.

- By the end of week 1, the patient is expected to have full passive flexion, full active extension and PIP active flexion to 30°.

- Discontinuation of the splint occurs between weeks 4 and 6—week 4 for patients with poor tendon gliding and 6 for those who have excellent ROM (defined as full active fist at week 2). At this time, exercises consist of passive ROM and active ROM.

- From week 6, blocking exercises of the individual joint is commenced. If flexion contractures are evident, these will need to be corrected with a splint.

- Progressive strengthening exercises begin 3 weeks after the dorsal block splint is discontinued. Resistance should increase so as to allow the patient to have full hand function by week 12.

It should be noted that EAM protocols should be *individualised* to the patient [73, 94] because advancement to the next phase of a protocol may be hindered or augmented based on the level of oedema, passive versus active flexion lags and adhesion formation [94].

Irrespective of whether or not a passive or active protocol is used, it has been shown that initiating a mobilisation therapy by postoperative day 5 decreases the rate of secondary procedures and decreases the costs of treatment [95].

7. Conclusion

The fine, tailored movements of the flexor tendon are essential to hand function. It is clear that the consequences of extrinsic healing of flexor tendons must be overcome to achieve optimal outcomes in patients who have injured their flexor tendons. Until the intrinsic healing process can be biologically augmented, surgical repair and rehabilitation of the injured flexor tendon will remain the mainstays of treatment. It is therefore essential that the surgeon bear in mind the basic tenets of tendon healing and the foundational principles of surgical repair.

Acknowledgements

The author wishes to acknowledge the immense contribution to flexor tendon pathology by the researchers, scientists and clinicians cited in this chapter.

Conflict of interest

None to declare.

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