

Towards a Splint-free Repair for Flexor Tendon Injuries

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Abstract

Flexor tendon injury has long been recognised as a difficult problem to tackle in hand surgery. Loss of active motion in the digits results in significant morbidity. The increased understanding in tendon healing, role of the tendon sheath, and the development of mobilisation and repair techniques have drastically improved results of flexor tendon injuries over the last 50 years. The treatment of flexor tendon injuries is based on evidence-based medicine, where clinical problems and new concepts were first evaluated in the biomechanical and animal laboratories and clinical practice is dictated by long-term outcome results. However, current surgical repairs are not strong enough to withstand active grip during rehabilitation and patients needed a prolonged period of protected rehabilitation until tendon healing is completed. The need for a more robust repair to allow a splint-free unprotected use of the hand should be the next step forward in the approach to flexor tendon injuries.

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Introduction

“It is easy to bring the two ends of a severed tendon together to allow healing but difficult to maintain a smooth gliding interface with an intact tendon sheath.”

The most problematic area in flexor tendon injuries is zone two. This zone is prone to injury as it is in the grasping part of the hand. In addition to the need to ensure that tendons heal well in continuity, the presence of the tight fibrous tendon sheath dictates that the repair not be bulky. It must preserve vascularity of a relatively avascular portion of the tendon and maintain the biomechanical integrity of the pulley system. These constraints led Bunnell¹ to declare the portion of flexor tendon in the digital sheath (zone 2) as “no man’s land” in the 1920s. This stemmed from his observation of adhesions and loss of function following repair in this area. He postulated that the firm flexor tunnel did not allow for swelling after the repair and resulted in necrosis of the tendon and fibrosis with surrounding tissues. He advocated excision of tendon ends and primary flexor tendon grafting as the procedure of choice for zone 2

injuries. Verdan² advocated excision of flexor tendon sheath to allow for extrinsic healing. In Singapore, Chacha³ advocated utilising free autologous composite tendon for zone 2 flexor tendon repair, given the poor results of primary repair at that time.

Key Concepts

Several key concepts have emerged since the 1960s based on continuous understanding of flexor tendon healing. This revolutionised the practice of flexor tendon repair, where primary repair took precedence over tendon grafting.

Tendon Healing

Potenza⁴ concluded that tendons heal because of fibroblastic response of surrounding injured tissues (extrinsic healing). This is the concept of the common wound where adjacent injured tissues heal with a common scar. Hence, surgical trauma must be kept to a minimum to reduce scarring. Hunter⁵ established that the vincular system provided tendons with intrinsic blood supply. Hence, tendons could heal with minimal scarring to surrounding

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tissues based on its intrinsic blood supply. Cadaveric injection studies by Lundborg and Rydevik⁶ revealed an avascular zone of both flexor tendons in “no-man’s land”. This could account for the propensity for scarring in zone 2 injuries.

This intrinsic system was, however, shown to be of less importance than the diffusion of nutrients from the synovial fluid in the tendon sheath in tracer studies in dogs.⁷ Therefore, it is important to preserve the integrity of the tendon sheath. This was, however, not borne out by clinical studies.

Lundborg and Rank⁸ further demonstrated the ability of flexor tendons for intrinsic healing in its sheath by obtaining nutrition from synovial diffusion alone. In their experimental model, repaired rabbit flexor tendons isolated in the knee joint and left free in synovial fluid were studied. The tendons were nourished only by diffusion from synovial fluid. There were no adhesions and a proliferation of tendon cells with tendon healing was observed. It could be concluded that tendon cells possess an intrinsic potential of repair. Hence, in a largely intact synovial sheath system, adhesions with surrounding tissues could be minimised.

Surgical Repairs

Verdan² first advocated primary repair of flexor tendons, with results comparable to tendon grafting. Poor quality suture materials and repair techniques contributed to the early failures. Patients were unable to undergo adequate postoperative mobilisation as the repairs were not sufficiently strong.

Kleinert et al⁹ published their landmark series of 10-year experience with primary zone 2 flexor tendon repair. A strong repair coupled with an aggressive postoperative rehabilitation programme using protected active mobilisation produced favourable results previously unobtainable. Our first local result of primary flexor tendon repair was published by Pho et al in 1978.¹⁰ The results in the 23 digits reported were encouraging, with 87% achieving good to excellent results. It was concluded that primary tendon repair offered the patients a shorter period of disablement with functional results as good or even better than those after a free tendon graft.

Primary Flexor Tendon Repair

With refinement of suture materials and repair techniques in the 1960s, there were major advances in the strength and gap resistance of tendon repairs. Based on a large body of biomechanical data with regards to the various repair methods, basic principles regarding repairs were formulated.

The ideal tendon repair should be strong enough to withstand the forces of active mobilisation, as early active motion resulted in a stronger repair with fewer adhesions

and improves overall outcome. The strength of a repair is proportional to the number of suture strands crossing the repair site.¹¹ Hence, the development of various repair techniques over the years utilising 2 to 8 core sutures. However, with the increased number of core sutures, the repair bulk increased. This impeded movement and further compromised the intrinsic vascularity of the tendons.

Trail et al^{12,13} demonstrated that repair ruptures often occurred at the suture knots and equal tension across all suture strands was needed to prevent differential loading and weakening of the repair. Gapping at the repair site became the weakest part of the tendon and attracted adhesions resulting in decreased motion. This was well demonstrated by Seradge¹⁴ using radiographic markers. It helped us understand the importance of gapping and the need for active mobilisation.¹⁵ Gelberman et al¹⁶ have shown in a canine model that close coaptation of tendon ends encouraged intrinsic endotenon repair that revascularised by 7 days, while repairs with gaps of 3 mm heal with extrinsic epitenon scarring, which only revascularised in 14 days. Epitendinous suture has been shown to improve repair strength by 10% to 50% and produced a significant reduction in gapping between tendon ends.^{17,18}

The need for a technically less demanding and stronger repair has led us to the use of loop sutures which was first described by Tsuge et al¹⁹ as a single loop repair. This technique, although technically simple, has only two strands and the pull-out strength was 22 newtons. The surgical knots were also extra-tendinous. The employment of 2 loop sutures allows us to perform a 6-strand core suture repair with intra-tendinous knotting.²⁰ This pull-out strength was 49 newtons.²¹ This improvement allowed us to include an active component in the mobilisation regime. The preliminary results of the 6-strand repair showed that all cases have good to excellent results with no ruptures (Figs. 1a to 1d).^{22,23}

Postoperative Management

Gelberman et al¹⁶ showed convincingly that, compared to postoperative immobilisation, early post-repair motion stress to repaired flexor tendons provided a more rapid recovery of tensile strength, less adhesions, improved tendon excursion and less repair site deformation.^{16,24-27} There was a trend towards the use of early active motion following a strong flexor tendon repair which was first reported by Becker et al.²⁸

Postoperative pain during mobilisation has always been a difficult problem to address, and many patients ended up with suboptimal results after flexor tendon surgery because of immobility. Blockade of the brachial plexus through the interscalene space via a disposable pump was effective in



Fig. 1a. Zone II injuries involving the middle, ring and little fingers.



Fig. 1b. Repair done with a double loop sutures six strand technique with tendon sheath repaired.

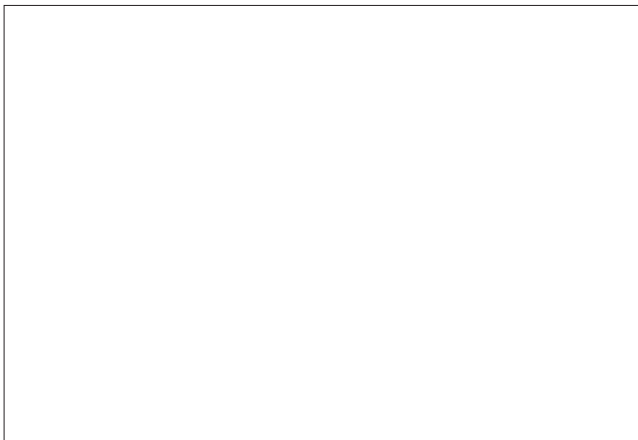


Fig. 1c. Three months after surgery—hand in full extension.

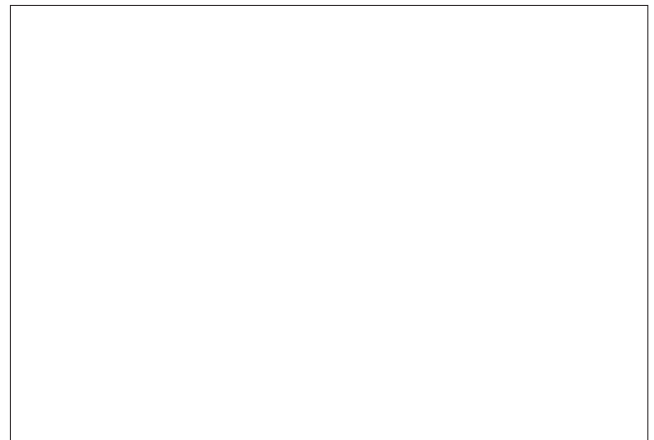


Fig. 1d. Three months after surgery—hand in full flexion.

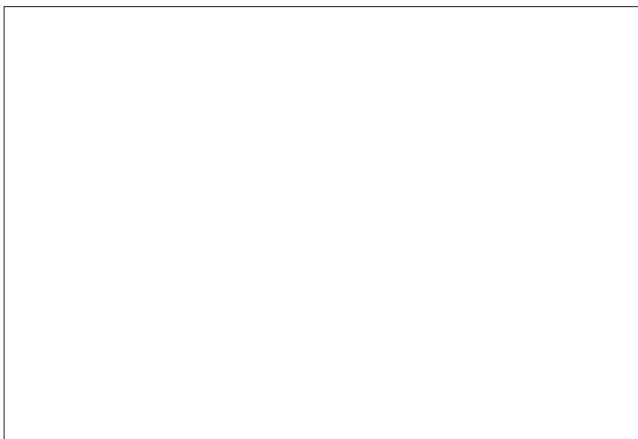


Fig. 2a. Exposure of the wrist over the volar aspect. Flexor tendons were divided and insertion of the intra-tendinous anchoring device. This is done through insertion of the device into the cut ends of the tendon.

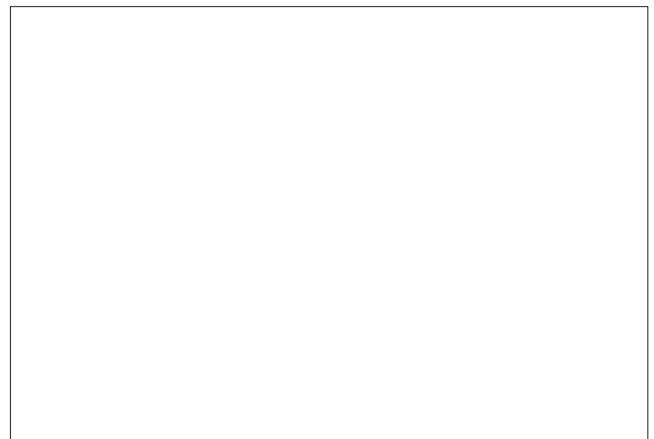


Fig. 2b. Intra-tendinous placement of the anchoring device in place. The flexor tendons were harvested as show prior to biomechanical testing.

controlling pain for patients undergoing hand surgery.²⁹ We have also pioneered the clinical use of long-acting local anaesthetics, delivered by small epidural catheters sited in close vicinity to the distal median and/or ulnar nerves at the wrist, to provide pain relief during mobilisation.³⁰

Towards a Splint-free Repair

An ultra-strong repair that allows active unprotected mobilisation is the dream of many hand surgeons. *In vivo* studies performed by Schiund et al³¹ demonstrated that passive flexion subjected the tendons to 5 newtons of force, active flexion up to 20 newtons, strong composite grip

requires 50 newtons of force and tip pinch up to 90 newtons. These values are likely to be substantially higher in the early postoperative period due to oedema and the additional “drag” forces of the tendon repair as it passes through the pulley system.

A conventional 2-strand Kessler repair has a break strength of 25 newtons, a 4-strand repair has a break strength of 45 newtons and a 6-strand repair has a break strength of 60 newtons. The repair strength of the tendons suffers a 50% decrease during the second week because of softening of the tendon due to inflammation.³² The repair will only recover its original strength at 4 to 6 weeks. Hence, the present day repair techniques will only allow light active flexion during therapy, especially during the first 4 weeks. This has led us to look at the use of intra-tendinous anchoring devices to improve the repair strength. Various experimental models were used and the maximal pull out strength achieved ranged from 40 to 80 newtons.^{33,34} Biomechanical work on one such stainless steel internal tendon anchor³³ has revealed a 99% to 270% increase in mean tensile strength over other repairs. Applied clinically, this would allow the patient to perform composite grip during therapy without the protection of a cumbersome splint. However, these devices could slip out of the tendons and cause severe attrition of the surrounding soft tissues (Figs. 2a & 2b). Accurate placement of these intratendinous devices is technically demanding and the search continues for an ultra-strong repair that allows splint-free postoperative mobilisation.

The Future

Despite the vast amounts of knowledge amassed in the last 30 years, flexor tendon repairs could still not achieve uniformly excellent results. Rupture rates average 5%³⁵ depending on the mobilisation technique and strength of repair. Postoperatively, the patient still need to be rehabilitated under the watchful eye of an occupational therapist and undergo a difficult and frustrating period of gradual and painful improvement. For the present moment, the postoperative regime should take into consideration the tendon repair technique and strength. Only the motivated can result in good recovery of function. A novel solution to this problem may require a different perspective in tendon repair technique to allow splint-free aggressive active mobilisation. This preserves the muscle bulk of the forearm flexors and minimises the possibility of stiffness and contractures, allowing an early return to functional activities. The elimination of scarring and adhesions may lie in the realms of molecular manipulation of wound healing. A lot of work is still needed before we can achieve excellent recovery of function with low rupture rates in all flexor tendon injuries.

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