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Improving Results of Flexor Tendon Repair and Rehabilitation

Jason K. F. Wong, M.B.Ch.B., Ph.D.
Fiona Peck, M.S.C.P.

Manchester, United Kingdom

Background: The global time and effort attributed to improving outcomes in the management of flexor tendon injury are large, but the degree of advancement made over the past 50 years is relatively small. This review examines the current perceived wisdom in this field and aims to explore the limitations to the authors' understanding of the tendon healing process, examining how this may be a factor that has contributed to the authors' modest progress in the field.

Methods: The authors critically evaluate the sum of laboratory and clinical literature on the topic of zone II flexor tendon management that has guided their practice and provide evidence to support their methods.

Results: The review highlights some of the key developments over the years and assesses their influence on changing current practice. It also highlights recent innovations, which have the potential to influence flexor tendon outcomes by altering the surgical approach, techniques, and rehabilitation regimens. Future innovations in the field will also be discussed to examine their potential in expanding the development in the management of flexor tendon injury.

Conclusions: A better understanding of flexor tendon biology will allow progress in developing new therapies for flexor tendon injuries; however, there are as yet few real breakthroughs that will dramatically change current practice. (*Plast. Reconstr. Surg.* 134: 913e, 2014.)

The management of flexor tendon injuries remains one of the most published topics in hand surgery, with the numbers of publications on this subject seeing a year-on-year increase (Fig. 1). The perfect repair and outcome continue to evade us,[1] despite the flexor tendon repair being one of the earliest skills acquired as a hand surgeon in either plastic or orthopedic surgery training.[2] New tendon repairs and hand therapy regimens are reported regularly. Occasionally, there is an announcement of a new treatment modality that promises hope for this clinical conundrum, but this rarely becomes part of standard practice. Over the past 50 years, there have been many innovations, but overall outcomes have not changed dramatically. For example, the best series published in the 1970s showed that a two-strand repair with simple circumferential suture and a Kleinert type rehabilitation regimen had a 5 percent rupture rate, with 75 percent of patients achieving good to excellent functional outcomes in 28 zone II injuries.[3] This compares favorably with more recent studies showing that a four-strand repair and early active mobilization regimen had a 5 percent rupture rate, with 71 percent achieving good to excellent outcomes in 73 cases.[4]

Real paradigm shifts in this area require us to rethink the whole process of flexor tendon biology

From the University Hospital South Manchester, Wythenshawe Hospital; Plastic Surgery Research, Institute of Inflammation and Repair, University of Manchester.
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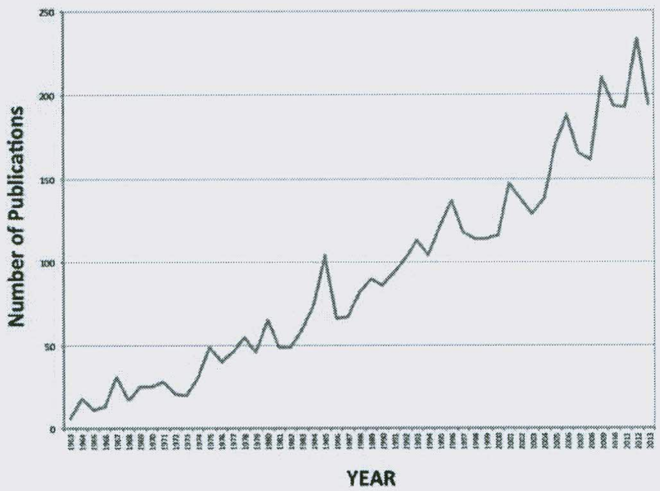


Fig. 1. PubMed statistics on flexor tendon publications over the past 50 years.

and how this relates to the clinical picture. This review aims to present the current best evidence of how to manage flexor tendon injury, but also challenges these surgical and rehabilitation management strategies with alternative approaches that may potentially see further improvement.

SCALE OF THE PROBLEM

Flexor tendon injuries are increasing in frequency on an annual basis in the United Kingdom, with there being an estimated 18,000 primary repairs performed per year, equating to an incidence of 41 per 100,000. Approximately 2000 per year require secondary tenolysis or two-stage flexor tendon grafting, suggesting an 11 percent reoperation rate.[5] Through meta-analysis of systematic reviews,[6] complications have been reported as 4 percent rupture rates, 6 percent reoperation rates, and 4 percent adhesion rates, which suggest that the problem is underreported. Of these secondary operations, 58 percent had tenolysis, 38 percent had simple rerepair, and 4 percent had tenolysis and rerepair. Ninety-one percent had one reoperation, whereas 8 percent had two further operations and 1 percent had three.

FLEXOR TENDON HEALING PARADIGM

Despite better scientific understanding of how tendon heals, the clinical community still relies on concepts that were introduced 50 years ago. The concepts of intrinsic and extrinsic healing were largely borne of observations relating to the experimental design and the modality being used to investigate the biology at the time. For example, Potenza's original experiments[7] involved using a tube to interrupt adhesion growth into the tendon in canine forepaws, which led to tendon necrosis caused by lack of an extrinsic blood supply. This led to the notion of an "extrinsic" healing process, whereby adhesions were necessary to revascularize the tendon. A critical explanation would suggest that insertion of silicone foreign material led to a significant inflammatory response, increasing sheath and tissue pressure within the digit and thereby contributing to tendon necrosis. Studies over the years have shown that using other barrier methods to inhibit adhesions actually has little bearing on how the tendon heals, other than to reduce adhesions.[8] Matthews and Richards[9] found that retracting the tendon out with the sheath and wounding the tendon led to healing in the absence of adhesion formation, indicating that repair could be initiated by cell populations "intrinsic" to the tendon. Studies have supported these findings by showing injured intrasynovial tendon healed independently of adhesions in a synovial environment,[10] in culture[11,12] and when transplanted into subcutaneous diffusion chambers.[13] Cells provided with the appropriate nutrition in culture will behave in this fashion, which is not unique phenomenology to tendon. Like most cells, tendon fibroblasts will proliferate and spread, smoothing over any rough surface of tissue in the process although, uniquely, when given two points of tension, tendon fibroblasts will self-assemble into three-dimensional structures.[14,15]

The reality of tendon healing is far more complex, involving a careful interplay of cells, matrix, gene expression, and growth factors between the

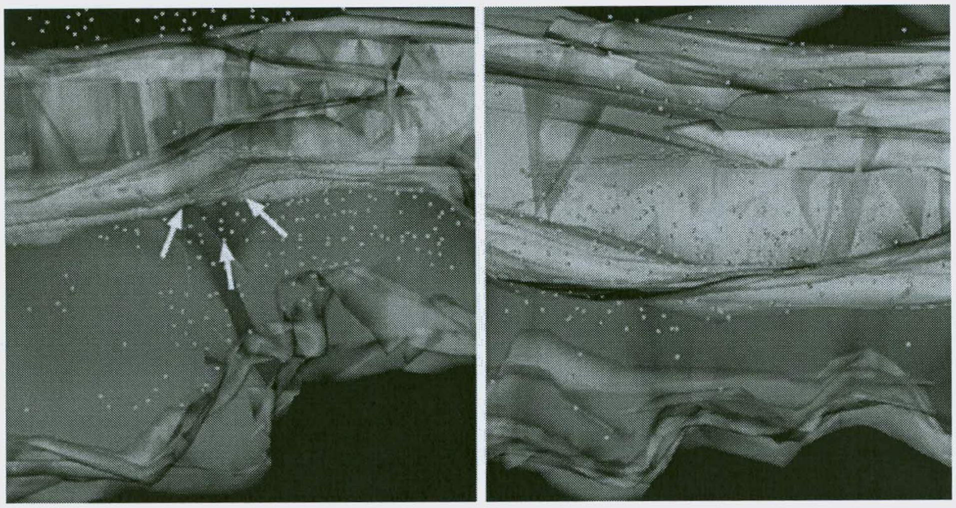
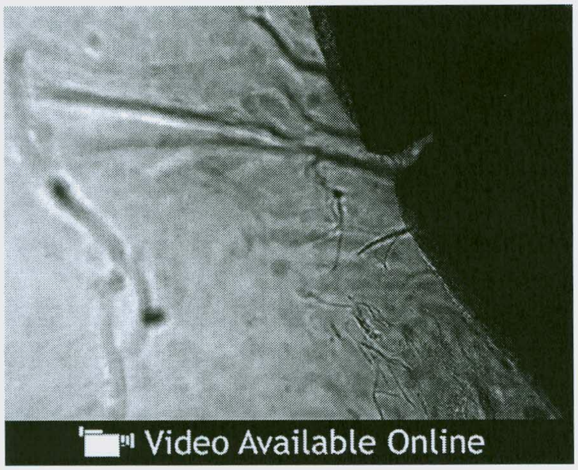


Fig. 2. Three-dimensional cell mapping of adhesion formation in zone II of the mouse flexor tendon. (*Left*) At 7 days after injury, early formation of tendon adhesion (outlined in *dotted red*) at the interface between tendon and surrounding tissue. Collagen synthesizing cells are shown in *green*. (*Right*) At 21 days after injury, there is established adhesion formation with lots of collagen being deposited at the interface between tendon and surrounding tissue.

tendon and the surrounding structures.[16,17] The tendon healing paradigm is similar to that of wound healing but must be taken in context with the dynamic gliding anatomy of the tissues and digits.[18]

Immobilizing flexor tendon injuries allows for healing to occur between the two injured tissues, which overlap during the healing process. Our observations on a simple murine flexor tendon injury model[16] showed some resemblance to early experiments by Lindsay and McDougall,[19] who found that the cells from both subcutaneous tissues, sheath and tendon, were active in cell migration, collagen synthesis, and cellular proliferation. It was also apparent that the inherent swelling caused by injury allows the tendon and sheath to come into contact while the healing process begins (Fig. 2). [**See Video, Supplemental Digital Content 1,** which displays a zone II flexor tendon adhesion formation in a mouse (using three-dimensional cell mapping). (*A - Partial Laceration*) After the initial partial laceration of tendon and tissues. (*B - Inflammatory Phase*) The inflammatory phase is initiated and a big influx of inflammatory cells occur (*red*). The tissues begin to swell, which brings the tendon and surrounding tissues into contact and the initiation of collagen synthesis (*green*) begins in the subcutaneous tissues. Macrophages are recruited into the adhesion (*yellow*). (*C - Early Proliferative Phase*) Gradually, the adhesion-forming process increases with maximum synthetic activity and cellular infiltration at approximately 3 weeks. (*D - Established Synthetic Phase*) Gradually the tissue swelling and cell number decrease through apoptosis and the adhesion remodels leaving a

Video 1. Supplemental Digital Content 1 displays a zone II flexor tendon adhesion formation in a mouse (using three-dimensional cell mapping). (*A - Partial Laceration)* After the initial partial laceration of tendon and tissues. (*B - Inflammatory Phase*) The inflammatory phase is initiated and a big influx of inflammatory cells occur (*red*). The tissues begin to swell, which brings the tendon and surrounding tissues into contact and the initiation of collagen synthesis (*green*) begins in the subcutaneous tissues. Macrophages are recruited into the adhesion (*yellow*). (*C - Early Proliferative Phase*) Gradually, the adhesion-forming process increases with maximum synthetic activity and cellular infiltration at approximately 3 weeks. (*D - Established Synthetic Phase*) Gradually the tissue swelling and cell number decrease through apoptosis and the adhesion remodels leaving a thinner adhesion (*E- Remodelling and Resolution Phase*). This video is available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B157***.



Video 2. Supplemental Digital Content 2 displays a partial tendon laceration in vitro. A mouse flexor tendon is partially divided and time lapsed over the course of 24 hours, ***http://links.lww.com/PRS/B158***.

thinner adhesion (*E- Remodelling and Resolution Phase*). This video is available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B157***.] The tendon and sheath are covered by an epithelium that acts as a barrier to the migration of cells which, when breached, allows for cells to move freely along a growth factor gradient.[20] (**See Video, Supplemental Digital Content 2,** which displays a partial tendon laceration in vitro. A mouse flexor tendon is partially divided and time lapsed over the course of 24 hours, ***http://links.lww.com/PRS/B158***.)

Mechanism of injury is important in predicting the degree of damage signaling in the tissues. For example, a sharp clean cut will heal well if the repair is performed carefully; however, the trauma from a tearing, crush, avulsion, aggressive coagulation, infection, or overzealous handling will lead to a greater injury response, which in turn dictates adhesion formation (Table 1).[21–24] In the clinical setting where the zone II flexor tendon had tearing and saw injuries, significantly poorer functional outcomes were observed compared with those with sharp clean injuries to their flexor tendons (range of movement, 86 ± 14 degrees versus 114 ± 7 degrees, respectively; $p = 0.05$).[25] In vivo studies in canines have also shown that suture techniques that exhibit more suture on the tendon surface, such as the Massachusetts General Hospital Repair, generate more friction, presumably through adhesions, than suture techniques such as the modified Kessler repair that exhibit less suture on the tendon surface[26]; thus, a balance must be made between gaining adequate strength without overtraumatizing the tendon[27] or producing too much glide resistance.[28]

TENDON REPAIR

Hand therapy, with early active mobilization, has proved to be the main effective modality in preventing adhesions[29]; thus, surgically, the aim has been to make our tendon repairs strong. Any repair should take into account the forces acting on the finger during rehabilitation.[30] Strength can be gained by suture material, gauge of suture, number of core strands, suture purchase, suture configuration, and addition of an epitendinous suture (Fig. 3).

The ideal suture is deemed to be strong, minimally reactive, easy to handle and knot,[38] and, some would argue, biodegradable.[39,40] Most surgeons opt for the strongest material to which they have access. Comparative studies have shown that with all factors controlled for, using a standardized 4-0 locked cruciate repair, stainless steel was strongest (87.4 N), followed closely by FiberWire (Arthrex, Inc., Naples, Fla.) (80.5 N), then Ethibond (Ethicon, Inc., Somerville, N.J.) (65.6 N), then Prolene (Ethicon) (63.4 N), and finally Nylon (Ethicon) (46.7 N).[31] The size of suture is proportional to the strength of the repair, but a

Table 1. Mechanism of Injury Chart

Etiologic Factor	Model	Evidence	References
Trauma	Chicken flexor tendons	Crush injury, suture repair, sheath excision, and sublimis excision and immobilization cause violent adhesion formation	Lindsay and Thomson, 1960[21]
	Rabbit flexor tendons	Combination of sheath and tendon injury with tendon suturing and immobilization	Matthews and Richards, 1976[22]
Thermal energy	Chicken flexor tendons	Diathermy and carbon dioxide laser exposure to peritendinous tissue was directly proportional to adhesion formation	Hatano et al., 2000[23]
Infection	Horse flexor tendons	Tenoscopic evidence of dense fibrous adhesions following tendon sheath infections	Bertone, 1995[24]

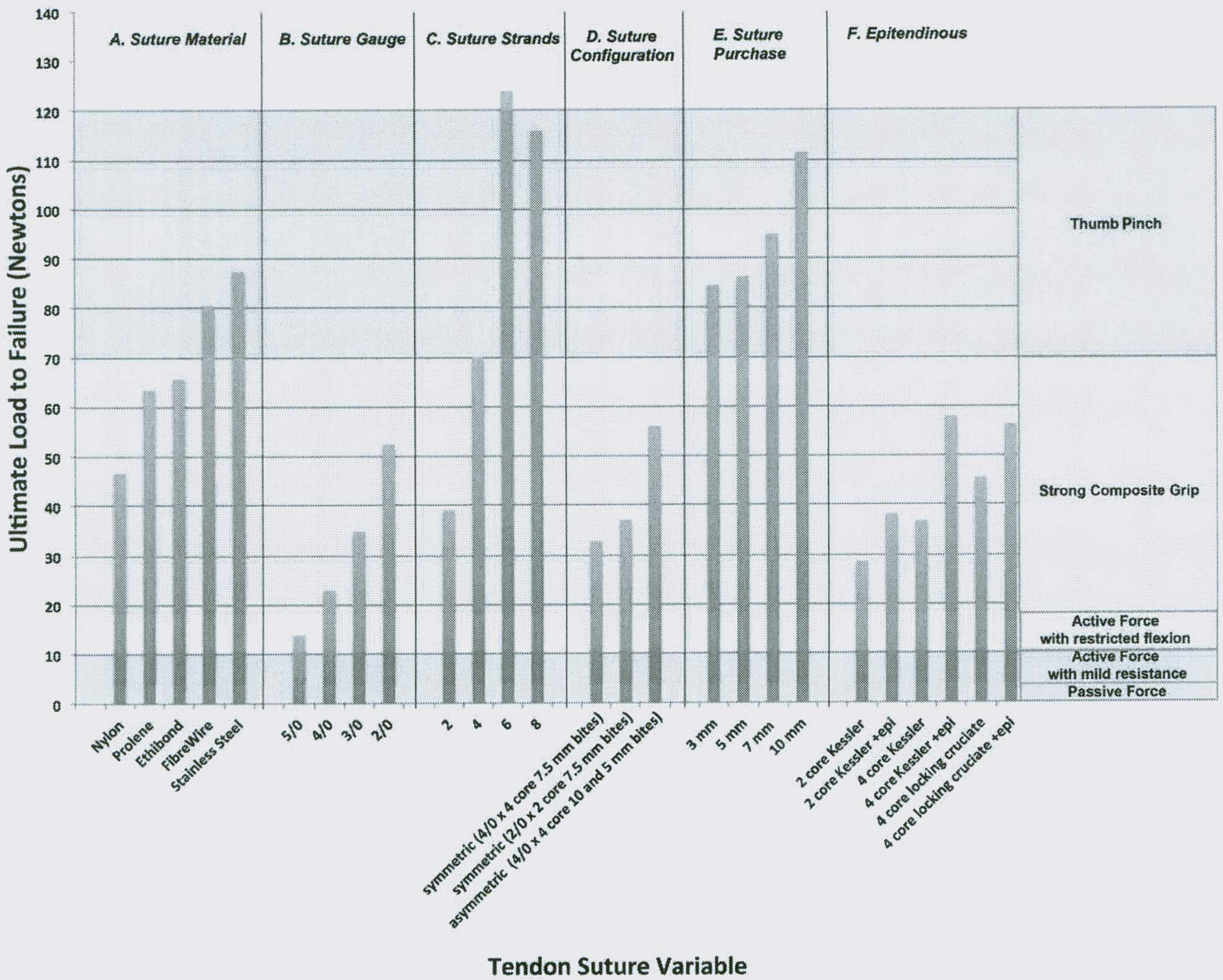


Fig. 3. Factors that influence tendon suture repair strength. Variables were deduced from ex vivo cadaver studies into (*left*) suture material with standardized 4-0 four-strand locked cruciate,[31] (*second column*) suture gauge with Ethibond as a standardized two-strand modified Kessler.[32] (*Third column*) Number of strands comparing Kessler (two-strand), cruciate (four-strand), Savage (six-strand),[33] and eight-strand Kessler[34] using standardized 4-0 Ethibond and 6-0 nylon epitendinous locking. (*Fourth column*) Suture configuration using standardized 4-0 FiberWire.[35] (*Fifth column*) Suture purchase using standardized 3-0 FiberWire locked cruciate and 6-0 interlocking horizontal mattress epitendinous suture.[36] (*Right*) Epitendinous suture using standardized 3-0 nylon and 5-0 nylon running epitendinous.[37]

reasonable size should be selected for the tendon size, to avoid overly damaging the tendon with needle size.[32] Multistrand repairs have gained popularity over double-strand repairs over the years, supported by a number of ex vivo and clinical studies (see Wu and Tang[41]). In average hands, the multistrand repair provides an extra 20 to 30 N of strength safeguard over a double-strand repair. The Savage six-strand repair[42] can give 81 percent excellent or good results in experienced hands but has failed to gain universal acceptance because of its complex nature.[43] The development and evolution of six-strand looped suture repairs by Wu and Tang has shown equivalent results with simpler M and U configurations that benefit from fewer suture passes and balanced load across the cross-section of the tendon.[41]

Repair geometry is critically important to disperse load and offer a balanced repair. Performing an epitendinous suture in modified Kessler repairs has this stabilizing effect,[37] or the load dispersion effect can be produced by having the sutures placed asymmetrically.[44] For a given suture caliber, altering the configuration of a repair by asymmetrically staggering the strands of a four-strand Kessler repair offers a greater repair strength than a symmetrical four-strand Kessler repair.[35]

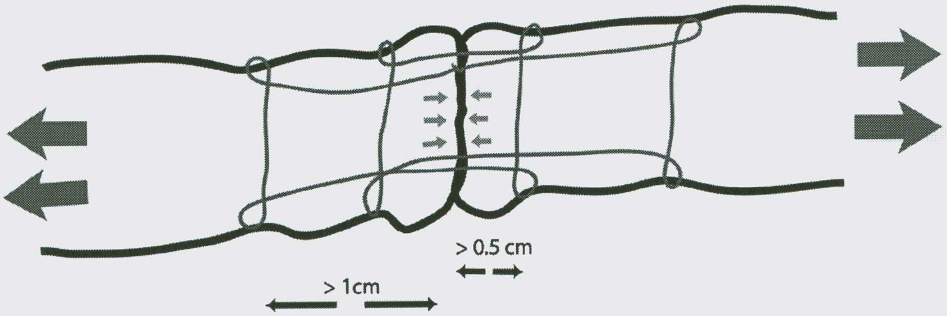
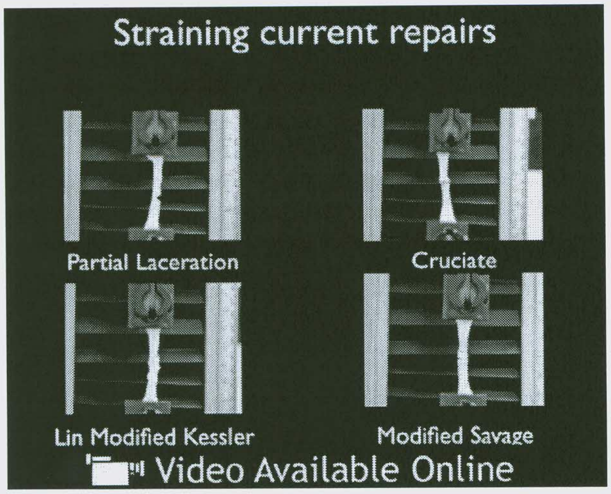


Fig. 4. Lin modified four-strand Kessler repair, one of the few dynamic repairs that offers compression on distraction through its continuous and asymmetric design. (See Lin GT. New suture techniques for flexor tendon repair. In: Saffar P, Amadio P, Foucher G, eds. *Current Practice in Hand Surgery*. London: Martin Dunitz Ltd; 1997:17–20.)

In our center, a four-strand repair favored and familiar to the individual operating surgeon is used, which is key to the repair being performed well and minimizing trauma from overzealous suture insertion.[27] We advocate as large a bite as possible of the tendon ends based on the findings of Kim et al.[45] My personal favored option is the Lin modified Kessler, which is a continuous asymmetric four-strand Kessler with a single continuous 3-0 Prolene suture that allows for distraction of the repair to result in compression of the repair site[46] (Fig. 4). This dynamic repair will adjust its configuration according to the pull across the repair site; a characteristic that other repairs do not provide. (**See Video, Supplemental Digital Content 3,** which displays a straining of popular repairs. The repairs include a standardized four-strand cruciate repair, four-strand Savage repair, and four-strand Lin modified Kessler repair. All are pulled apart at the same rate and force using 4-0 Prolene. Partially divided tendon, 50 percent, is used as a comparison and repairs are pulled to failure. Note the gapping that occurs with cruciate and Savage compared with the Lin modified Kessler repair. Ultimately, partial tendon is stronger than the other displayed repair types, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B159***.) In addition, I perform a simple 5-0 or 6-0 running epitendinous to stabilize the repair. The biomechanics of looped suture techniques[36] and barbed suture techniques[48] are also a current vogue but are not readily available to all hand centers.



Video 3. Supplemental Digital Content 3 displays a straining of popular repairs. The repairs include a standardized four-strand cruciate repair, four-strand Savage repair, and four-strand Lin modified Kessler repair. All are pulled apart at the same rate and force using 4-0 Prolene. Partially divided tendon, 50 percent, is used as a comparison and repairs are pulled to failure. Note the gapping that occurs with cruciate and Savage compared with the Lin modified Kessler repair. Ultimately, partial tendon is stronger than the other displayed repair types, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B159***.

It is worth highlighting that some studies and systematic reviews indicate the best functional outcomes have been achieved using two-strand repairs and early active mobilization or Kleinert type rehabilitation, with acceptably low rupture rates.[49–52] For progress to be made in tendon repair design, there has to be a move away from thinking about the number of cores, locking and grasping, to calculating the precise architecture of an optimal repair based on the tendon morphology, biology, and unique characteristics of each tendon injury.

TENDON EXPOSURE AND RETRIEVAL

Exposure for a tendon repair is usually down to operator preference. Some prefer Bruner's zig-zag

Video 4. Supplemental Digital Content 4 displays a flexor tendon retrieval using minimal access technique, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B160***.

Video 5. Supplemental Digital Content 5 displays a wide-awake flexor tendon operation, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B161***.

approach,[53] and others prefer midlateral.[54] These exposures aim to minimize the possibility of scar across the joint crease. The midlateral approach minimizes scarring on the volar aspect of the digit and, when performed with distal extension, gives excellent exposure to the retracted distal ends of tendon. However, in neonates, its use should be guarded, as there have been reports that the lateral scar migrates volarly with growth and may cause flexion contractures.[55] To retrieve the proximal tendon end, if it is not readily visible, the natural creases can serve as vents to localize the divided tendon, and with the aid of a feeding catheter can be retracted back into the injury zone for repair.[56] (**See Video, Supplemental Digital Content 4,** which displays a flexor tendon retrieval using minimal access technique, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B160***.) This has the benefit of minimizing surgical wounds and thus reduces the inflammatory response, adhesions, and scarring over the zone II region of the hand.[16]

WIDE-AWAKE FLEXOR TENDON SURGERY

The convention of operating on tendons in a bloodless field is a privilege that most hand surgeons appreciate and requires an anesthetized patient or at the very least an upper arm block so that the patient can tolerate the tourniquet for the required duration of surgery. The benefits of this are a clean, bloodless operating field in which structures can be easily identified and wounds can be extended. Therefore, operating on a patient without tourniquet is counterintuitive and goes against Bunnell's philosophy.[57] To challenge dogma, Lalonde and others have published numerous series on one of the major paradigm shifts in hand surgery using wide-awake anesthesia.[58–60] More hand surgeons are turning to this option for simple hand surgery. The benefits are not immediately apparent, as the first few cases require one to become comfortable with minor amounts of blood in the operating field; however, with experience, the benefits and ease of this technique in the context of flexor tendon repairs are quite dramatic. (**See Video, Supplemental Digital Content 5,** which displays a wide-awake flexor tendon operation, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B161***.) The ability to assess the repair intraoperatively for gapping, triggering, bowstringing, and smoothness of glide gives both the surgeon and patient confidence to proceed to an early active mobilization regimen.[59]

PULLEY MANAGEMENT

The debate on whether to vent or not vent the pulleys depends largely on what degree of impingement can be seen by putting the digit through a full range of motion on the operating table. Despite the early studies by Doyle suggesting that the A2 and A4 pulley were sacrosanct,[61] Franko et al. showed that venting the A4 pulley, in the context of a repair close to the pulley, allowed an increase in excursion by 5 percent and reduced the work of flexion compared with the nonvented A4 pulley.[62] The more detailed analysis by Tang and Xie has shown that 75 percent of the A2 and complete division of the A4 pulley can be performed without much detriment to the digital arc of motion.[63] In addition, clinical studies indicate that as many as 56 percent of zone II injuries would benefit from venting either the A2 or A4 pulley to allow for sufficient glide.[64]

EVOLVING PRACTICE

The heterogeneity in the timing of the repairs, the demographics of the patient populations, mechanisms of injury, surgical approach, and surgical method make it difficult to define the factors that give the best outcomes. Incrementally evolving one's own practice, as Sirotakova and Elliot have with flexor pollicis longus tendons, is one way of controlling for these confounding influences. The evolution of their flexor pollicis longus injury management has seen a stepwise improvement from using two-strand Kessler with simple peripheral sutures[65] to two-strand Kessler and Silfverskiöld peripheral sutures, to a double-stranded Kessler and Silfverskiöld peripheral suture,[66] to triple Tsuge repair with no peripheral suture,[67] and saw improvements from 70 to 75 to 75 percent good, excellent functional results and 17 to 8 to 0 percent and 0 percent rupture rates, respectively. Our own experience with repeated audit of our clinical activities in flexor tendons has seen incremental improvements from 1997 to 2013, with rupture rates dropping from 30 percent with two-strand Kessler repairs, epitendinous repair, and controlled active mobilization regimens, through to 17 percent with the introduction of hand therapist–led clinics, to rupture rates of 4 percent with the introduction of four-strand repairs.[68] Through the unit policy introduction of four-strand core repairs, circumferential epitendinous suture, along with consultant-led trauma lists, practitioner-led clinics, and dedicated tendon repair training days, our unit rupture rate was reduced to 0 percent in 2006. We also found that delaying the time of surgery beyond 7 days had a significant negative impact on patients' range of motion which, once ameliorated, led to better functional results. As such, there is great merit in performing in-house analysis and continual health care improvement in centers that manage flexor tendon injuries because of the heterogeneous patient demographics, surgical skill sets, and therapy provisions from unit to unit. Arguably, this is more relevant to individual centers than reported systematic reviews that pool results and give confounding outcomes.[48-]

REHABILITATION

The evidence for the best rehabilitation regimen has been heavily debated over the years. Biologically, there is sound evidence that correlates well with clinical observations that tensile motion facilitates a reduction in inflammation,[69] up-regulation of collagen synthesis,[70] deposition of large-diameter fibrils,[71] and alignment of collagen fibrils.[72] As such, the argument for early active mobilization is scientifically quite compelling. Many reviews have now compared early active mobilization with passive mobilization and combined therapies, and the rupture rates are comparable but overall range of active movement is certainly better with the active regimens as indicated by level IIa evidence.[73] A review of electronic databases between 1970 and 2009 of 15 articles that reviewed rupture rates, range of motion, and quality of life identified that rupture rates were lowest in combined therapy regimens (controlled passive motion and passive flexion, active extension Kleinert and Duran) (2.3 percent) and highest in Kleinert-only (passive flexion and active extension) protocols (7.1 percent); however, finding the best functional results were obtained by early active mobilization or combined Kleinert and Duran protocols.[74] Studies into quality-of-life measures are still lacking.

There are a number of elements that in our experience appear to affect outcomes, including mechanism of injury, zone of injury, timing of surgery, caliber of repair, and age and characteristics of the patient, although this is not reflected in systematic review meta-analysis of complications following flexor tendon repair.[6] In each case, it is important that there is a degree of customization of therapy regimens to the needs of the patient, with close effective communication of the operative findings and surgery performed between the surgeon and therapists.

In Manchester, we have redesigned our rehabilitation regimens in line with increased repair strengths. Regimens must adhere to the principles of safety and promote maximum tendon glide. Active mobilization regimens for noncomplex repairs begin on the third to fifth days, in line with current thinking on the subsidence of postoperative edema.[28] To minimize the effects of edema on joint motion and to prevent tightening

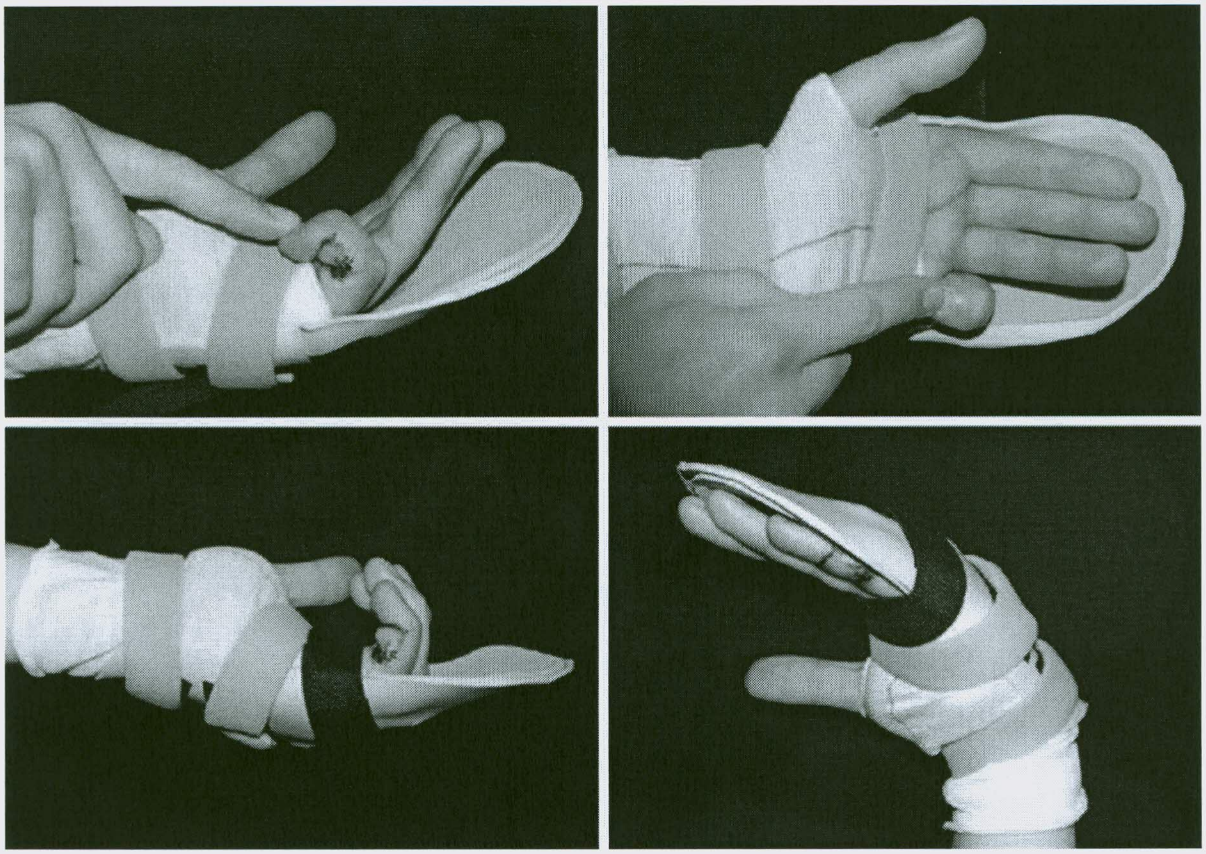


Fig. 5. Manchester short splint. (*Above*) Patients are taught to implement full passive range of flexion of the digit after 5 days, followed by (*below, left*) active flexion, performed with the wrist in 45 degrees' extension initiated at the distal interphalangeal joint in full fist. (*Below, right*) Full active extension of the interphalangeal joints is performed with the wrist in flexion to reduce force across the flexors.

of dorsal structures, the exercise sequence prioritizes full passive flexion stretching of the interphalangeal joints, maximizing passive digital flexion before initiating active motion. We have also radically evolved our splinting design for greater patient freedom. The Manchester short splint extends from the fingertips to the proximal wrist crease with a block to 30 degrees of metacarpophalangeal flexion. This position facilitates active motion initiated from the distal interphalangeal joint with the wrist in 45-degree extension to promote differential glide and minimize the work of flexion (Figs. 5 and 6). This splint also permits maximal wrist flexion, facilitating maximal active interphalangeal joint extension to reduce the risk of extension loss common in this type of injury. (**See Video, Supplemental Digital Content 6,** which displays a Manchester short splint and rehabilitation, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B162.***)

Early results are encouraging, with improvements in the arc of flexion and reduced interphalangeal joint extension loss.[75] Patients are required to adhere to a strict rehabilitation regimen, and compliance is an important factor in achieving good outcomes. Compliance differs according to age, understanding, mental health issues, and socioeconomic factors. Previous studies report high ruptures in those patients who use the hand for inappropriate functional activity.[76] Exclusion of the affected hand from function for 6 weeks is perhaps unrealistic in today's socioeconomic conditions, and we now instruct the patient in safe use of the hand with exclusion of the affected digit (Fig. 6, *left*).

A protective guard or boxing glove dressing may be used to add extra protection to the unpredictable activity of a child. For very young children, immobilization for 4 weeks in dressings does not appear to give rise to the same adhesion problems as for adults.[77]

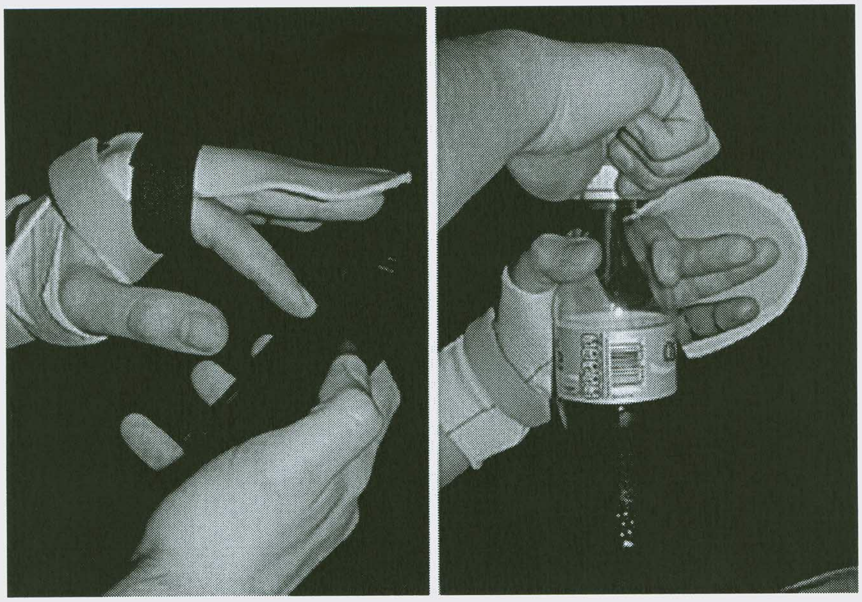


Fig. 6. Patients are taught to exclude the injured digit but can use their hand for day-to-day tasks. This improves patient compliance with the splint.

FUTURE DEVELOPMENTS

There are a number of technologies in development that may impact on the management of tendon injuries in the future. Biomolecules, gene therapies, and cell-based therapies have emerged from the biotechnology field and have been applied to tendon repairs. Transforming growth factor-β and manipulation of these pathways have undergone preclinical study[78] but ultimately have not delivered in the clinical setting, possibly because of the heterogeneity of human populations posing too much of a challenge to single molecular therapies or single pathway manipulation approaches. Even multiple growth factor manipulation in solutions such as platelet-rich plasma have shown mixed results in augmenting flexor tendon repair. Some studies have shown enhancement at 2 weeks[79] and others have shown no significant improvement,[80] and the "silver bullet" approach to flexor tendon injuries is unlikely to be successful. As such, gene therapy and stem cell therapies are not likely to have a great impact on addressing issues arising from the standard simple divided tendon and repair. Adjuncts to improve glide may have a role, and carbodiimide-derived hyaluronic acid with or without lubricin[81] or using shear aggregated fibronectin tubes[82] certainly shows promise in animal studies, but cost-to-benefit consideration to clinical translation is likely to be a major hurdle. For therapies to replace damaged tendon, reseeding acellular tendon with stem cells[83] and using biomaterials as scaffolds[84] are popular research areas at present. In the future, use of individualized custom splints scanned to fit using three-dimensional printing may be commonplace in all hand therapy departments and has already been touted as a massive area of growth in the health care industry.[85]

Video 6. Supplemental Digital Content 6 displays a Manchester short splint and rehabilitation, available in the "Related Videos" section of the full-text article on PRSJournal.com or available at ***http://links.lww.com/PRS/B162***.

CONCLUSIONS

Minor gains in flexor tendon outcomes can be expected with the evolution of operating equipment, surgical devices, techniques, and rehabilitation regimens, but until a major biological or technological disruptive technology arises that affects all forms of wound healing and repair processes, progress in this field will be modest. Until then, the pursuit of improving flexor tendon outcomes will continue to monopolize the research interests of many academic hand surgeons.

At present, using a combination of early surgical intervention, multistrand repairs, minimal access tendon surgery under wide-awake anesthesia, with the Manchester short splint regimen for rehabilitation, along with appropriate patient education is what we believe will improve the functional results in our patient population. This will have to be examined prospectively to provide better levels of supportive evidence; however, history dictates that evolution and innovation in this field are inevitable.

Jason K. F. Wong, M.B.Ch.B., Ph.D.
Plastic and Reconstructive Surgery Research
University of Manchester
Room 3.60 Stopford Building, Oxford Road
Manchester M13 9PT, United Kingdom
jason.k.wong@manchester.ac.uk

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PATIENT CONSENT

The patient provided written consent for use of the patient's image.

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