Invited Personal Review

INDICATIONS, METHODS, POSTOPERATIVE MOTION AND OUTCOME EVALUATION OF PRIMARY FLEXOR TENDON REPAIRS IN ZONE 2

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Abstract

In recent years, our unit has put into practice of flexor tendon repairs a number of novel concepts, which we hope address some critical difficulties in primary flexor tendon repairs in Zone 2, thus pointing the way towards predictable surgical outcomes. In this article, I present my practical views on indications, techniques, post-surgical treatment and outcome measures, and describe our methods of sheath-pulley release, tendon repair, postoperative motion and outcome evaluation.
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The advent of primary flexor tendon repairs within the synovial sheath region should be credited to those pioneers, such as Verdan (1960) and Kleinert et al. (1967), nearly half a century ago. Prior to that, over a long period of the previous half century, primary tendon repair was not advocated and surgeons followed Bunnell's advice to remove the tendons entirely and graft in new tendon (Bunnell, 1918, 1922). The reports of Verdan and Kleinert and his colleagues on primary flexor tendon repairs established that the lacerated digital flexor tendon can be treated by direct end-to-end repairs when wound conditions are favorable. However, most surgeons have noted that the outcomes of primary repairs remain hard to predict, particularly in respect of restrictive adhesion formation and rupture of repairs (Cullen et al., 1989; Elliot et al., 1994; Small et al., 1989; Strickland and Glogovac, 1980; Tang et al., 1994). Over the last two decades, surgeons have tried to identify flexor tendon repairs which yield optimal outcomes consistently. Considerable research and clinical effort has been expended and the number of reports on this subject probably surpasses those on any other single topic in Surgery of the Hand during this period. While the overwhelming number of investigations reflects the elaborate nature of the basic science and clinical practice regarding digital flexor tendon repairs, the volume of work also indicates that a path leading to a satisfactory and predictable treatment outcome has not yet been identified.

In recent years, our unit has put into practice a number of novel concepts which we believe may ensure more predictable surgical outcomes and help to address some critical difficulties in primary flexor tendon repair. We hope that they eventually point the way towards optimal flexor tendon repairs. In this article, I present our practical views on indications, techniques, post-operative mobilisation and outcome measures.

INDICATIONS

"Clean-cut" wounds, the simplest clinical situation associated with digital tendon lacerations, are a prime indication for primary flexor tendon repair. I consider a wound to be such when cut cleanly and tidily, usually as a single transverse, or oblique, wound in the fingers or distal palm, and produced by a knife or a piece of glass. The cut is also "clean" in terms of minimal potential for contamination and infection. Anatomically, the tendon(s) is only "severed", and without tissue defect. The cut tissues may even align well. This is the best indication for primary repair, with the greatest likelihood of relatively uncomplicated repair, rehabilitation and satisfactory outcome. Such wounds are very often accompanied by divisions of the digital neurovascular structures, which does not contraindicate primary repair of the tendons.

Crush injuries to a very limited segment of the fingers, or palm, produce untidy skin and subcutaneous injuries and tendon wounds. It is accepted that such wounds are also good candidates for primary repairs, because the soft tissue wounds and tendons can be made "similar" to those associated with a clean-cut wound through debridement of nonviable tissues and direct wound closure. However, these injuries have a greater potential for contamination. Primary tendon surgery is possible, although more difficult than with a truly "clean-cut" wound. Phalangeal fractures are rarely associated with a clean-cut flexor tendon laceration, but can become part of a crush injury. A simple and stable fracture in the phalangeal shaft can be securely fixed internally and, so, presents no contraindication to primary tendon repair.

The borderline indications for primary repairs have been less thoroughly addressed and I have seen no clinical investigations devoted solely to this topic. Nevertheless, it is in such cases that we must explore

the limits of the indications for primary repair. Below, I outline five clinical situations representing borderline indications for primary tendon repairs, along with some considerations used in the decision making process as to whether to undertake primary surgery.

(1) *Localised soft tissue injuries*: Crush, or compression, injuries on the palmar aspect of the fingers sometimes lead to localised soft tissue defects. The underlying flexor tendons may present with a short traumatic defect, or such a defect arises after debridement of nonviable, ragged tendon tissue. I deem this situation to include no contraindications to primary repair if the soft tissue defect is less than $\frac{1}{3}$ the length of the fingers and the tendon loss is less than 1.5 to 2 cm. The soft tissue can be repaired easily with a local or free flap transfer and the tendon is repaired by direct end-to-end suture. However, a tendon with a defect length close to 2 cm is hard to pull together, sometimes. In this case, intramuscular tendon lengthening through a forearm incision may release the tension (Le Viet, 1986). Direct end-to-end suture of the tendon should be accompanied by a procedure to reduce the tension on the tendon when surgeons or therapists are less experienced with dealing with tendons with a defect, otherwise the repair may be ruptured easily upon starting active digital mobilisation. Flap transfer provides fresh and vascularised tissue coverage, not very different from the original digital subcutaneous tissue, and early mobilisation of the tendon is still possible under the flap.

(2) *Injuries including a simple and stable fracture*: As mentioned above, a simple, stable fracture in the phalanx is by no means a contraindication to primary tendon surgery. What are seen more frequently, however, are tendon injuries associated with fractures involving joints in more than one phalanx, with crush, or abrasion, of the overlying soft tissues. These skeletal injuries are contraindications to primary tendon repair, because fractures involving joints tend to be unstable, the soft tissue wounds are always contaminated and early postoperative tendon mobilisation is difficult, or not feasible. Fractures in the shafts of more than one metacarpal bone may sometimes accompany a cut digital flexor tendon. These injuries do not preclude primary tendon surgery, providing the fractures are simple, limited to the shaft and do not involve the joints. Internal fixations in the palm with mini-plates, screws, or K-wires usually ensure a stable reduction, but early postoperative exercise may have to be less aggressive.

(3) *Rupture of tendon repairs*: Ruptures of primarily repaired tendons have been noted in almost all case series incorporating early active finger mobilisation. However, the first report exclusively considering repair of ruptured tendon has only just been published (Dowd et al., 2006). I approach the ruptured tendon repair as I would a primary tendon repair. Tendons need to be trimmed. About half, or more than half, (if not the entire) segment encompassed by the original sutures should be trimmed off, because the ends are softened and ragged and this decreases the holding power of the subsequent re-repair. The length of tendon segments that I trim off is about 0.8 to 1.0 cm (0.5 cm or less on either end). This amount of shortening is of no biomechanical consequence to the flexor digitorum profundus (FDP) tendon, even if the tendon had been trimmed by a similar amount previously at the initial surgery. In my experience, the shortening that the FDP tolerates can be up to 1.5 to 2 cm. The ruptured flexor digitorum superficialis (FDS) tendon should be removed. I find re-repair of both tendons impractical, and shortening of the FDS, particularly within Zone 2, is mechanically disadvantageous, because the structures and gliding direction of the FDS tendon varies greatly and the two parts of the FDS tendon are hard to match after loss of a tendon segment. The digital sheath system, both the parts mainly consisting of synovial sheath and those which are dense annular pulleys, is usually less elastic, narrow and inclined to collapse after the primary repair ruptures. Rupture of a repair seen within one month after the initial repair is always worth an attempt at re-repair. However, after one month from primary repair, re-repair is rarely indicated as ruptured tendons one month after primary surgery are likely to be surrounded by adhesions and their healing potential is limited, particularly if the tendons are repaired under increased tension.

(4) *Delayed repairs*: I have found no clinical investigation which actually validates the textbook concept of "the best time" for primary repairs. All estimates of the "best time" to carry out primary flexor tendon repair suggested so far have been empirical. I do not have a rigid "best" time frame in mind, as previous suggestions regarding the timing of primary repair are not consistent and may not be imperative. The ideal situation is that a patient with digital flexor tendon lacerations is brought into the clinic soon after injury, surgery begins within a few hours and an experienced surgeon is readily available. The tendon should not be repaired primarily by an inexperienced surgeon. Rather, the tendon repair can be delayed until an experienced surgeon is available. My preferred period of deliberate delay is 4 to 7 days, when the risk of infection can be properly addressed and oedema has reduced substantially. My clinical impression is that treatment outcomes after delay for such a short period are almost identical to those associated with primary repair promptly after the trauma. Upon re-opening of the wound, the cut tendon ends still appear fresh and no collapse or fibrosis of the sheath is seen. The tendons can be treated as if they were freshly cut. However, when the surgery is postponed further beyond that period, the tendon ends may be rounded, with varying degrees of adhesions present, and the elasticity of the sheath is likely to be reduced, making repair more difficult. Although it is generally considered that a delay of over one month would rule out direct end-to-end repair surgery, surgeons may need to pay attention to a largely forgotten, but potentially

important, report of McFarlane et al. (1968), in which direct end-to-end repair after delay for one to several months had been possible without undue tension, and consider the possibility of a direct repair. In addition, I deem it an option for these late cases that the surgeon lengthens the tendon within the muscles in the forearm to ease the tension on the proximal tendon end (Le Viet, 1986). Of particular note is one situation in which repair delayed over a month is still feasible, viz. a wound around the proximal interphalangeal (PIP) joint level in which the FDP tendon has been cut but the long vincula connecting to the proximal cut tendon has not been severed. In this instance, retraction of the FDP tendon is limited. When the wound is opened, the retracted proximal end is found locally within the sheath and the tendon can be repaired with relative ease. I have encountered a few such cases and believe that a tendon wound at this particular site without proximal tendon retraction is worth an attempt at primary repair, even if a lengthy period has elapsed since the injury.

(5) *Massive soft tissue damage*: Generally, this is a contraindication to primary flexor tendon surgery and I discourage primary repairs under this circumstance. However, this situation can be broken down further into: (a) extensive soft tissue damage without apparent loss of tendon substance and (b) soft tissue damage with loss of a significant length of tendon. The former injury may still leave room to allow primary tendon repair, providing the surgeon is prepared to carry out secondary tenolysis later, if necessary. Not surprisingly, this is controversial and has not been well defined. Although the decision is difficult, we must balance the merits of primary repair and early mobilisation, followed by tenolysis if necessary, against those of secondary tendon grafting. It may be acceptable, although somewhat aggressive, to repair these injured tendons primarily and prepare the patient for the possibility of tenolysis, on the strength of soft tissue integrity having been restored and no bony or articular injuries being present, and, of importance, wound infection having been eliminated, or prevented effectively, with antibiotics, instead of delaying to do a secondary tendon graft, which sacrifices a donor and, occasionally, still require tenolysis.

Absolute contraindications for primary flexor tendon repair are severe contamination, signs of infection, bony injuries involving joint components and lengthy defects of the flexor tendons.

REPAIR TECHNIQUES

Incisions and pulley release

I prefer a zig-zag (Bruner) incision to expose the wound. In a clean-cut wound, a laceration through skin and sheath is transverse or oblique and, in the majority of the cases, does not overlie the site of tendon transection. When the level of tendon laceration is judged to be in the vicinity of the PIP joint, I make a "window" to open the sheath. When the tendon cut is at the level of the A3 pulley, I open this window between the A3 and A2 pulleys. When the cut is between the PIP joint and A4 pulley, I frequently have to include the A3 pulley in the sheath incision, but preserve the sheath proximal to this window. In many other cases, the tendons are cut where one or more of the strong annular pulleys is present, viz. a little distal to the A2 pulley, through the A2 and/or A1 pulleys and in the vicinity of the A4 pulley. In contradistinction to methods described in many texts and to traditional advice, I, purposefully, cut the entire A4 pulley or a major part of the A2 pulley around tendon repair sites, while leaving the synovial part of the sheath and other pulley structures. When the tendons are cut a little distal to the A2 pulley, that is within Zone 2B, according to my subdivisions of Zone 2 (Tang, 1994), I cut open the sheath longitudinally for one cm distal to the A2 pulley and also open the distal half of the A2 pulley (Fig 1). When repairing the tendon at the distal edge, or in the distal part, of the A2 pulley, I cut open a half cm of the sheath distal to the A2 pulley together with the distal two-thirds of the A2 pulley. When repairing a tendon under the middle or proximal part of the A2 pulley, I cut open the proximal two-thirds of the A2 pulley. Because the excursion of the FDP tendon within this part of Zone 2 is usually about 2 cm, the above lengths of release of the A2 pulley and the adjacent sheath are, in most cases, sufficient to free the tendon from restriction by the pulley, or catching on the rim of the pulleys, during movement of the finger joints through a full range. The merit of releasing the pulleys and sheath while restricting the length of these releases is that it allows tendon to glide freely while avoiding clinically significant bowstringing. The natural design of the flexor sheath and pulley system embodies allowance for such limited releases, which provide for adequate motion of the flexor tendons without impairing tendon mechanics. When repairing both tendons proximal to the A2 pulley, I also, frequently, cut open part of the sheath, including the A1 pulley. When the FDP tendon has been lacerated in the proximity of the A4 pulley and the tendon repair has difficulty passing beneath this pulley during surgery, I completely cut the A4 pulley.

The idea of purposefully incising a part of the most critical pulley (A2) came to me when I performed an anatomical and biomechanical study devoted exclusively to the area of the A2 pulley in 1994, as an alternative approach to local excision of the FDS tendon in treatment of the cases of injuries of both FDP and FDS tendons in Zone 2C (Tang, 1995). This concept of purposeful release of the A2 pulley is in agreement with the results of an earlier study of Savage (1990), who reported no substantial effects of divisions of a combination of, or individual, pulleys of digital fibrous sheath. Subsequently, Tomaino and his colleagues (Mitsionis et al., 1999; Tomaino et al., 1998) extended

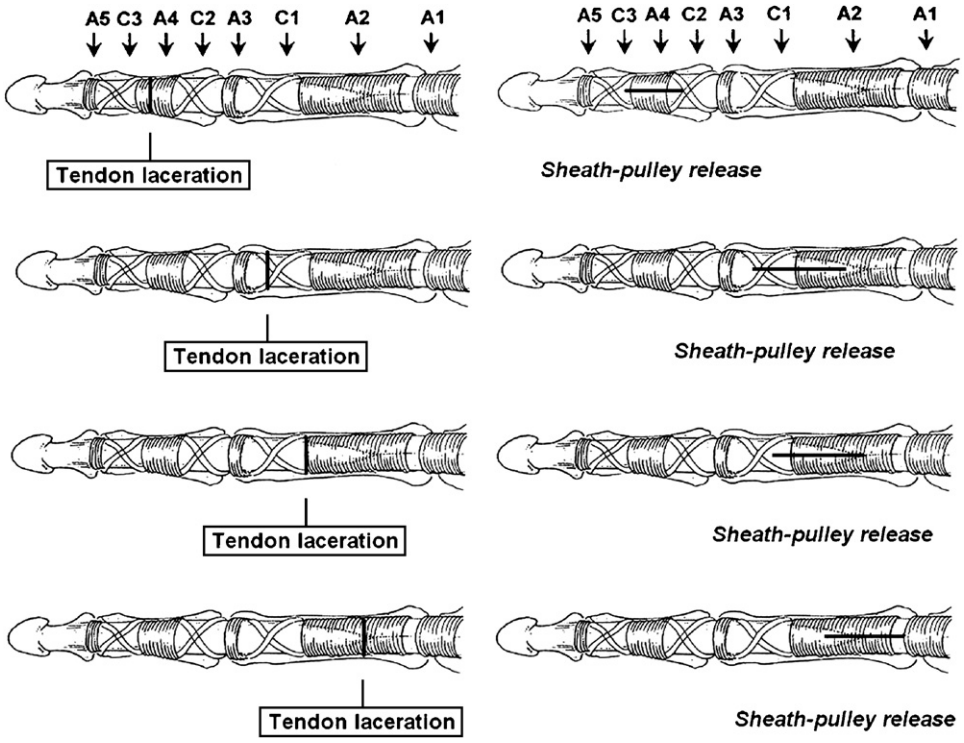

Fig 1 Four clinical situations requiring release of the critical pulleys: (1) release of the entire A4 pulley when the FDP tendon has been cut around the A4 pulley and the tendon cannot pass easily beneath this pulley during surgery; (2) release of the sheath for a half one cm distal to the A2 pulley and release of the distal half of the A2 pulley, when the tendons are cut a little distal to the A2 pulley; (3) release of the sheath for a half cm distal to the A2 pulley and release of the distal two-thirds of the A2 pulley when repairing tendons cut at the edge of, or in the distal part, of the A2 pulley; and (4) release of the proximal two-thirds of the A2 pulley when repairing a cut in the middle, or proximal part of, the A2 pulley.

these biomechanical investigations by more precise divisions of the A2 pulley and incision of the A4 pulley. I had not considered the need for active release of the A4 pulley until the report of Kwai Ben and Elliot (1998) about their experience of ''venting'' the A2 and the A4 pulley, but started to incise the A4 entirely in needed cases from that time. I have purposefully incised (or excised) a part of the A2 pulley for the past 12 years and the entire A4 pulley for the past 8 years without encountering clinically perceivable adverse effects from this procedure, and my view on the critical role of this procedure has not changed since I first wrote on this subject in 1995.

Before I begin a discussion of the repair of lacerated flexor tendons, I must highlight the importance of familiarity with the anatomy of the digital sheath and tendons in Zone 2. Most surgeons are familiar with the number of structures, their names and their functions, but not all have acquired the detailed knowledge necessary to perform fine, reparative surgery comfortably. Surgeons who lack precise understanding of the structures in this area can operate, but cannot operate predictably. I would suggest that not only the nomenclature and approximate locations of the structures, but also the following critical detail, should be borne in mind, or reviewed before starting the surgery: the A2 pulley is a central landmark in Zone 2. It is about 2 cm long and is located over the proximal two-thirds of the proximal phalanx. The middle and distal parts of the A2 pulley are very narrow. The FDS tendon bifurcates in the middle part of the A2 pulley. The length of the A1 pulley is about 0.5 to 1 cm and its diameter is larger than that of the A2 pulley. Of particular note is that the A1 pulley can occasionally unite with the A2 pulley, making an extremely long (3.0 cm) constrictive pulley-band over half the length of Zone 2 (Fig 2). The A4 pulley is about 0.5 to 0.8 cm long and is located at the midpoint of the middle phalanx. The FDS tendon ends proximal to, or under, the A4 pulley, so that only the FDP tendon glides under the A4 pulley. The A4 pulley is narrow even for a single FDP tendon, especially when the tendon is oedematous. Surgeons should know this anatomy in this degree of detail, including the approximate lengths and relative diameters of the sheath and pulleys in Zone 2 and around the A4 region (The lengths given above are those of index or middle fingers of average sized adults. The pulleys in the little finger and, of course, in children are narrower and have much shorter spans.)

How to repair the tendon surgically and enhance its strength has been a central issue over the last two

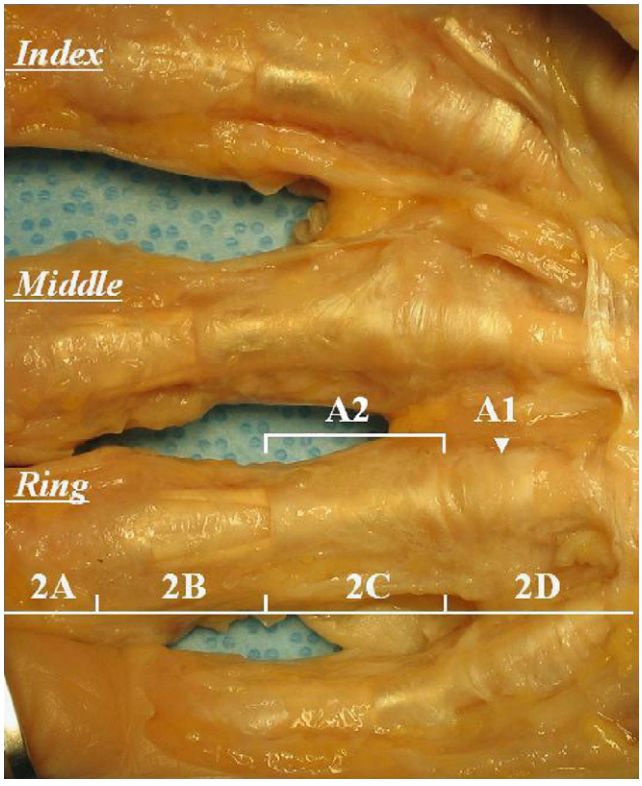

Fig 2 A cadaver dissection illustrating the series of dense annular pulley bands in the proximal part of Zone 2, which constrict motion of the tendons located not only under these pulleys (Zones 2C and 2D) but also distal to these pulleys (Zone 2B). The treatment outcomes of tendon lacerations in Zones 2B to D are all affected by the constriction of the A2 and A1 pulleys. In particular, the outcomes in Zones 2B and 2C tendon injuries are affected by the presence of the large, and tightest, A2 pulley as tendon repairs in Zone 2B have to glide into the A2 pulley area to flex the fingers adequately. The constitution of the A2 and A1 pulley complex is variable. In this ring finger, the A2 and A1 pulleys are separately identifiable. In the middle finger, they pretty well unite into a single long pulley. In other instances, the A2 and A1 pulleys are a series of separated pulley bands. In almost all cases, the distal and middle parts of the A2 pulley are continuous, very dense, and the narrowest in diameter. It is these parts of the A2 pulley that are most restrictive to tendon gliding and should be released completely when treating tendon injuries in Zone 2B and the distal half of the Zone 2C. These parts of the A2 pulley may be a major cause of rupture of primarily repaired tendons and of development of dense constrictive adhesions if not released sufficiently.

decades, but I have increasing belief that treatment of the major pulleys is equally important, or even more so, than enhancement of the strength of the repair. It is the presence of segmental and constrictive annular pulleys that make Zones 1 and 2 structurally distinctive from the other regions. Diminishing the adverse effects of these few critical pulleys on the repaired tendons sufficiently would eliminate the factors unique to Zones 1 and 2 which are probably the main cause of the unpredictability in outcome of treatment of tendon divisions in these zones. In other words, proper pulley treatment may eventually make outcomes predictable. Increasing the number of strands of core sutures and modifying their configurations certainly increases the safety margin for early postoperative exercise and this increase can be to double or triple the strength of a conventional two-strand repair. However, if a repaired tendon glides over the rim of a pulley, or catches against an intact pulley during finger flexion, particularly during extremes of active finger flexion, when the repair strength may decline to half or an even smaller fraction of the strength of a tendon being pulled linearly (Tang et al., 2003), a two-strand repair is likely to rupture and even a tendon with multi-strand repairs may do so. It is, thus, obvious to me that even a multi-strand tendon repair should be accompanied by proper and sufficient release of the critical part or the entire pulley in the vicinity of the repair, to eliminate the danger of overloading the tendon as a result of tendon gliding against the pulley rim or constriction by the narrowest pulley parts. Of the critical points of surgery, releasing the pulleys appropriately is probably the most important, followed by use of stronger repairs to increase the margin of safety. Closure of the synovial sheath is of least importance and repair of the strong annular pulleys over, or proximal to, oedematous tendon repairs would seem positively harmful. In dealing with the sheath, including the main synovial parts of the sheath and the annular pulleys, we should consider primarily whether they compromise tendon gliding and might constitute a cause of rupture of the repaired tendon. Their role in providing, or maintaining, nutrition is insignificant and their role in preventing adhesions is unimportant provided the tendons move in the period immediately after surgery.

In the past, the details of treatment of the annular pulleys during primary repairs were largely obscure. The concept of maintaining the integrity of the A2 and A4 pulleys, embraced by many surgeons, was "borrowed" from the operations for secondary tendon grafting or tenolysis, in which other parts of the sheath could be opened, or excised, to facilitate grafting or to free the tendon from adhesions but the A2 and A4 pulleys were sacrosanct. Previous treatment of these pulleys, largely by preservation at all costs, may help account for the unpredictability of the outcome of primary flexor tendon surgery. The results were likely to have been good when the pulleys happened to be partially destroyed or had been unintentionally opened for the purpose of exposure of the tendons. However, when the repairs were sited just distal to, or under a strong, yet well-preserved and lengthy, annular pulley, such as the A2, tendon gliding might have been restricted and the repair would have ruptured more easily. Both situations would have lead to a poor outcome.

Tendon repair techniques

One of the basic elements of core suture is the length of its suture purchase, but this has been rarely addressed in articles, or texts, dealing with primary tendon surgery. I consider the length of purchase of the core suture to be an important point. It is imperative to ensure that the core suture is anchored reliably in sufficient of the tendon ends. I recommend that a minimal length of purchase greater than 0.7 cm be used, with the length of suture purchase in each tendon stump being in the range 0.7 to 1.0 cm but never less than 0.7 cm. Otherwise, the core suture has insufficient tendon substance to grip, particularly as the tendon ends soften after repair. I have noted that the length of core suture purchase is very much smaller than the diameter of the tendons in drawings illustrating repair techniques in some book chapters. (The diameter of the FDP tendon in the middle finger is about 0.6 cm in an average-sized adult hand). I am not sure whether these diagrams are intended to represent the reality of this situation, or whether they are meant to only show suture configurations, without particular attention to the degree of suture purchase. A shorter core suture purchase than suggested above would be weak, as shown in our recent investigation (Tang et al., 2005). Multi-strand repairs with a short core suture purchase do not generate maximal strength.

Tightness of the core suture is another consideration. I usually create a 10% shortening of the tendon segment encompassed by the core suture by adding a little tension to the suture. Although tension-free repair is an important principle of nerve repair, the appropriate amount of tension added in the suturing for tendons has not been discussed specifically. Observing other surgeons has given me the impression that some repair the tendon in a ''tension-free'' manner. Perhaps they wanted to avoid bulkiness of the tendons at the repair site. If slightly tensioned, the tendon repair may look a little bulky when proximal pull by the muscles is eased by temporary fixation of the proximal tendon during surgery. This is never the case after release of the temporary fixation. My belief is that adding a slight baseline tension to the repair sites results in appropriate tension after surgery as this counteracts the tension of the muscles proximally during active motion. If tension has not been added to the repair site, the repair may overstretch and gap after surgery, particularly during early digital mobilisation. From my unpublished experiments and clinical observations, I feel that adding a little tension is as important as adding peripheral sutures. With appropriate pre-tension of the repair site, the chance of gapping, or the size of the gap, should be decreased and, therefore, the risk of catching of the repair sites on any edge of the sheath lessened. Peripheral sutures over a tension-free tendon repair site may smooth the approximated tendon ends during surgery, but gapping is more likely to develop when the repair is under muscle tension and the tendon moving. Tendon gaps, even when small, may disrupt the repair with relative ease if they catch on the rim of a pulley.

The methods I have used, and currently use, in repairing tendons have been explained in previous articles (Tang et al., 1994, 2001; Tang, 2005). The looped sutures introduced by Tsuge were used in these repairs. Fig 3 details the latest modification of this six-strand repair. Because this method is both a modification of the original 6-strand looped repair which I described previously and employs an ''M'' configuration of the suture within the tendon, it is referred to by my colleagues as the ''M-Tang'' method (Wang et al., 2003) (Fig 3). Recently, a four-strand modification has also been introduced by one of my colleagues (Cao and Tang, 2005).

Treatment of the FDS tendon in Zone 2 is not as straightforward as that of the FDP. The least problematic part of the FDS tendon is proximal to the bifurcation. Here, the injured FDS tendon can be treated in almost the same way as the FDP tendon, except that the FDS tendon is flatter and does not accommodate more than four strands of suture material. I am comfortable with a four-strand method, such as the double looped suture, when repairing FDS tendons in this part of the hand and would consider a cruciate repair to be another acceptable option. In my experience, the bifurcating part of the FDS tendon (the segment within Zone 2C) is hard to repair by any other method than two separate looped sutures. Treatment of the FDS tendon within Zone 2B is most frustrating and has been discussed specifically in a few papers (Boulas and Strickland, 1993; Britto et al., 2001). I use a variety of techniques, including repair with a tendon-to-bone junction (as for re-attachment of the FDP tendon to the distal phalanx) if the residual distal stump is very short and repair with a two-strand core suture for each slip if the distal stump is long enough. When one slip is completely cut but the other is uninjured, repair may not be necessary. Not infrequently, one or both slips of the bifurcated FDS tendon are partially severed, a situation in which it is hard to decide whether, or how, to carry out any repair. In this situation, we have to make a judgement according to the length of the distal stump and the extent of the tendon division. No single method can be recommended, but, in most cases, they need no repair.

Another challenging issue is how many tendons to repair when repair is delayed by one or more weeks after injury. When the cut is at, or just distal to, the A2 pulley (Zones 2C and 2B), it is almost impossible to repair both tendons with this delay in presentation. The FDS tendon retracts far proximally and it is hard to pass both tendons under the A2 pulley, or even a residual part of the pulley. Previously, I suggested that the FDS tendon is better left unrepaired in Zone 2C (Tang, 1994). With the advent of pulley releasing, in a clean-cut wound and when repair is not delayed too much, I would suggest repairing both tendons in Zone 2C and dividing two-thirds of the length of the A2 pulley.

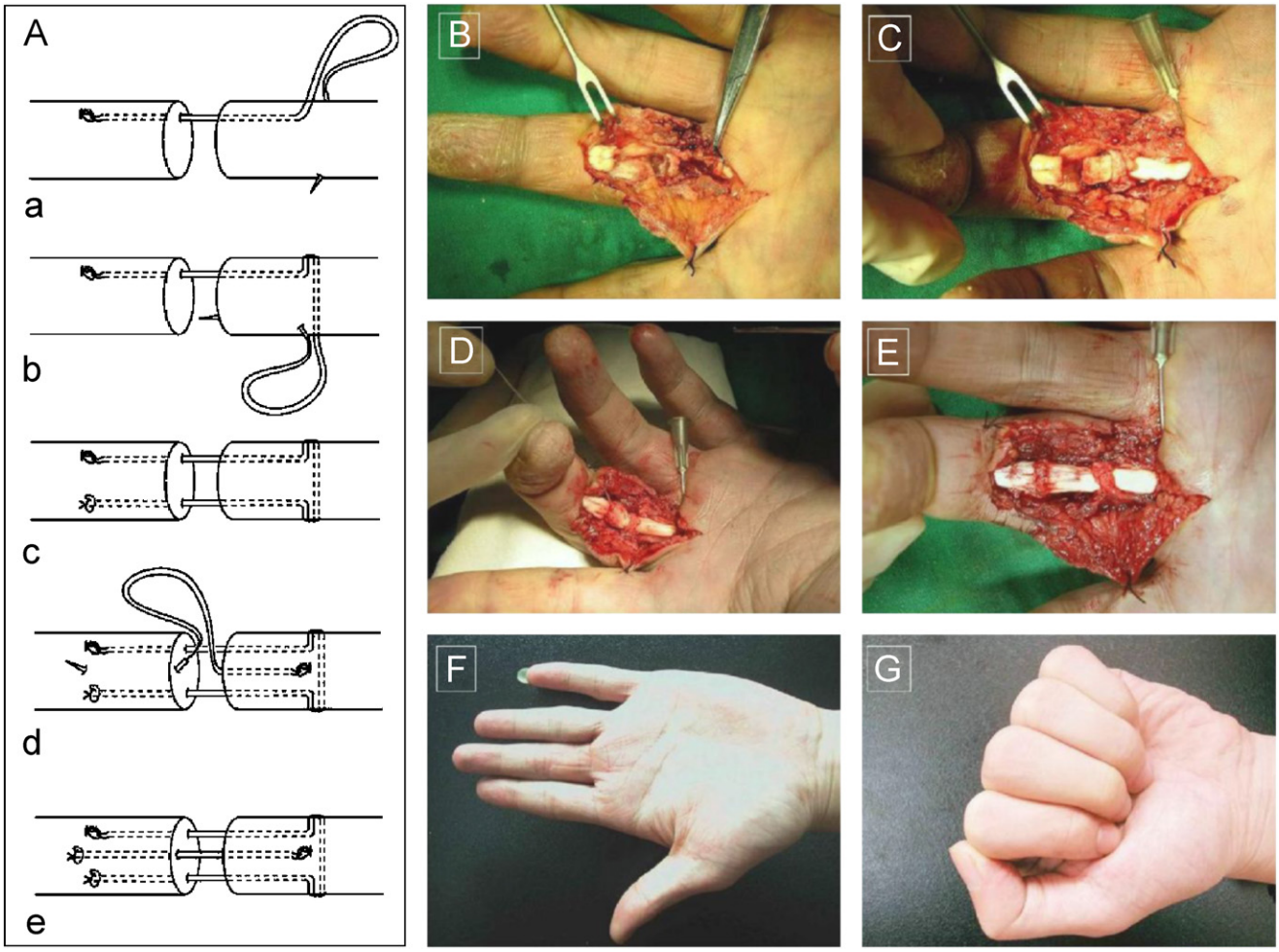

Fig 3 Our method of making a six-strand repair (the so called "M-Tang repair"): two separated looped nylons are used to make a M-shaped repair configuration within the tendon (shown in A); cross-sectionally these suture strands are evenly placed and form the points of a triangle. Use of this suture and the outcome are illustrated in B to G in a case of ruptured primary flexor tendon repair which was referred to the author. We performed direct repair of the ruptured flexor tendon 3 weeks after the first tendon repairs (B). The ragged tendon stumps were trimmed and the A2 pulley was adequately released, although preserving a part of the A2 pulley (C). The FDP tendon was repaired with the M-Tang technique and the FDS tendon was removed (D) and (E). (F) and (G) show the range of finger motion six months after this surgery.

However, in cases in which primary repair is considerably delayed, when the FDS tendon cannot be passed easily through the residual part of the A2 pulley and when the peritendinous injuries are more severe, the FDS tendon is still better left unrepaired, even if the major part of the A2 pulley has been vented. Repair of one slip of the FDS tendon, or demi-FDS tendon repair, is an alternative option should a surgeon be enthusiastic to restore FDS function and if it is hard to pass the complete FDS tendon under the A2 pulley.

POSTOPERATIVE MOBILISATION

The central dogma that primarily repaired flexor tendons should be mobilised soon after surgery is well known to most surgeons dealing with digital flexor tendons. However, one finds a perplexingly variety of rehabilitation protocols in the literature (Pettengill, 2005). A number of the currently used controlled active motion protocols are efficient. Past reports on the outcomes of the surgery appear to indicate that variants based on certain essential design principles eventually lead to comparable clinical outcomes. This illustrates the fact that slight alterations in the angles to which the joints should be flexed, or the number of repetitions of exercise in each episode of mobilisation, are relatively insignificant. On the other hand, we may need to consider modification of certain fundamental concepts in protocol design to reach the ultimate goal of restoration of a close-to-normal range of active motion without repair rupture. Currently, early controlled

active finger flexion is becoming the mainstay of motion exercise (Amadio et al., 2005; Baktir et al., 1996; Elliot, 2002; Pettengill, 2005), and early passive flexion by rubber band traction may be on its way to being abandoned. Our own clinic has shifted from passive flexion to early controlled active finger flexion over the last two decades. Our protocol in recent years has noticeable differences from documented protocols.

The following protocol was designed to actively flex the finger in a controlled manner but incorporates a number of manoeuvres based on conclusions derived from mechanical studies. Essentially, the hand is protected in a dorsal thermoplastic splint, with the wrist in slight flexion (20°–30°), the metacarpophalangeal (MCP) joints in slight flexion and the interphalangeal joints in extension (or minimal flexion), for the first 2.5 weeks (Fig 4). We do not encourage patients to move the finger during the first few postoperative days, because, at this time, the hand is painful, oedema is more prominent and, more importantly, adhesions do not form. Decreasing the days of motion decreases the chance of repair rupture. Exercise starts at 3 to 5 days (at 4 or 5 days in most cases) after surgery. Before each episode of active digital flexion, the fingers is passively flexed 10, or more, times to lessen the overall resistance of the finger joints and soft tissues – a ''warming up'' process—after which active flexion should encounter lower resistance. The patient is then instructed to flex the fingers actively with gentle force 20 to 30 times during each morning, noon, evening and before sleep, up to the range with which the patients feels comfortable. The motion range is usually from full extension to one-third, or half, of the full flexion range, although this may even increase to two-thirds of the full range if this can be achieved with ease. Active flexion over the full range is not encouraged, unless it can be achieved very easily. Patients may increase the number of motion episodes up to 5 or 6 per day, but we do not, necessarily, require patients to move hourly. In this 2.5-week period, full active extension is particularly encouraged and the fingers are passively stretched against the splint if full extension is not achieved. Prevention of extension

Fig 4 Illustration of the positions of splinting of the hands and postoperative exercise. In the first 2.5 weeks, the wrist is splinted in about 20° to 30° of flexion and the MCP joints are maintained in slight flexion. Complete extension of the finger joints is emphasised during this period. The active flexion may be only up to the mid-range and no forceful active flexion is encouraged. However, the fingers should be flexed passively over the full range. In the second 2.5 weeks, the wrist is splinted in extension and the MCP joints are maintained in the functional position. Active and passive finger flexion are emphasised during this period. Full passive flexion is ensured and active finger flexion is encouraged, but not forced to the final flexion range. The thumb is included in the splint to prevent unintended pinch or other uses of the hand, so protecting against rupture of the repairs. This is not an absolute requirement for cooperative patients who follow the guidelines of therapies.

deficits rather than full active flexion is emphasised during this period.

At 2.5 weeks, a new thermoplastic splint is made, with the wrist splinted in 30° of extension (Fig 4). Finger flexion, both passively and actively, is emphasised during the period from 2.5 to 5 weeks. The patients are instructed to actively flex after a passive warm-up, as earlier. Active flexion up to the mid-range is required as a minimum and is encouraged further, up to two-thirds, or the full range of flexion, depending on the patient's ability to perform resistance-free motion. Digital flexion from the mid-range to the full range, in particular over the final one-third of the flexion range, is usually carried out passively if the fingers encounter resistance. Our studies show that finger flexion over the final one-third of the full range of motion range encounters resistance 5 to 10 times that in the previous two-thirds of the range of motion, so ruptures are much more likely in this final part of the flexion range, even if the repair has survived the previous (and greater) part of the range of motion. In addition, the strength of repaired tendons moving over the final one-third of the flexion range can be much lower, because the ultimate strength and gap resistance decreases as the curvature of the gliding arc increases (Tang et al., 2003). This adds to the risk of repair rupture. Ensuring full passive flexion, to prevent dorsal ligament tightening and extensor tethering, and encouraging finger flexion actively, while avoiding flexing the finger forcefully over the final flexion range, are guidelines during this second 2.5 week period. Differential FDS and FDP motion exercise is encouraged through the first 5 weeks when two flexor tendons are repaired at the same level. The method of achieving this is, basically, by separated active flexion of the two interphalangeal joints. After 5 weeks, full active finger flexion is encouraged. This can be started earlier if flexion in the final part of the flexion range is judged to have less resistance. After 5 to 6 weeks, the splint is discarded or used only at night. The patients can return to normal use of the finger from 8 weeks.

This type of exercise regimen, incorporating passive and active elements within each exercise episode and each cycle of finger motion, is based on understanding of the mechanics of movement of repaired tendons during finger flexion and shifting the tension on the flexors according to the wrist position. By changing the wrist position at 2.5 weeks, emphasis can be shifted from achieving full extension to achieving full flexion, enabling the full range of intended motion to be achieved with relative ease while diminishing the risk of joint contractures of both the wrist and the finger joints and also diminishing the risk of rupture of tendon repairs. The mechanical basis behind the protocol design is synergy between wrist and finger actions: with the wrist flexed, full finger extension is achieved with less tension on the flexor tendons, while full finger flexion can be achieved with less tension on the repairs with the wrist extended. Savage (1988) first pointed out that wrist extension is not harmful during motion of the interphalangeal joints and an experiment by Amadio and his colleagues highlighted the merits of synergistic wrist extension in reducing the tension of the finger flexors during active finger flexion (Tanaka et al., 2005). Active finger flexion to full flexion encounters much less resistance when the wrist is extended than when the wrist is flexed. This effectively avoids overload of the repaired tendons. However, we do not encourage maximal active flexion of the finger when the exercise meets remarkable resistance, but, instead, incorporate active finger flexion up to the mid-range and passive motion from mid- to maximal flexion into individual motion cycles. We believe that repair rupture can be minimised using the above regimen of exercising, aimed at avoiding high levels of tension on the repairs, while achieving sufficient active motion, together with the above described releases of the sheath and pulleys and an increase in surgical suture strength.

EVALUATION OF OUTCOMES

The most commonly used evaluation system used in the last two decades has been the Strickland criteria (Strickland and Glogovac, 1980). The TAM and Buck-Gramcko (1976) methods are also used extensively (Kleinert and Verdan, 1983). It is somewhat hard to believe that the TAM method was not as popular as the Strickland criteria, even among hand surgeons in America, The modified Strickland (1985) criteria, have not enjoyed popularity because they are too lenient. Previously, I have used the original Strickland criteria, the TAM method and the White criteria, but I favoured the original Strickland criteria. However, range of motion as the only measure of functional status is insufficient. I would suggest that range of motion, grip strength, finger motion arc and activities performed by the finger flexors should be combined into one, yet simple, formula defining the functional status of the fingers after flexor tendon injury. Clinically, I use the method shown below, which includes three items, to record the outcome of finger flexor tendon repairs, viz.: (1) active range of motion, (2) grip strength and (3) quality of motion.

In evaluating active range of motion, I adopt a percentile distribution of range of active motion, which is not exactly as Strickland suggested. I use the ranges of active motion from the contralateral hand as the normal values, if the contralateral hand is normal. I think the range of active motion for a finger categorised as "excellent" should be more stringent than in Strickland's original criteria, to allow cases with truly excellent recovery to be distinguished from "very good" cases. In my experience, the inclusion of cases of active range of motion not exceeding 90% of the normal in the "excellent" category appears lenient. I have also created a category of "failure", to identify, specifically, the cases

with rupture of the repairs or development of severe adhesions and/or joint contracture preventing tendon motion and definitely requiring secondary surgery. The fingers categorised as ''failures'' are not able to perform essential functions, being worse than ''poor'', and need further surgery. Thus, they can be reasonably separated from ''poor'' cases.

Grip strength greater than the contralateral hand, if the latter is the non-dominant hand, or over 70% of that of the contralateral hand, if the latter is the dominant hand, is considered normal and recorded as (+). Otherwise, grip strength is graded as abnormal and marked as (−).

Quality of motion is judged from essential features of active digital movement. Currently, flexion arc, digital coordination and speed of movement are included in judging the quality of motion. Descriptors of the quality of motion may be modified upon accumulation of experience. Smoothness and perfection of motion arc can be hampered when one finger joint moves satisfactorily while another does not, which is very frequently seen after digital flexor tendon injuries. Imperfect digital coordination is seen when multiple fingers are injured. The speed of finger flexion determines how swiftly the finger can accomplish active flexion. Currently, instruments have not yet been developed for measuring the speed of motion or digital coordination. The speed of motion or digital coordination are judged in clinic by asking the patient to move all the fingers of the hand at varying speeds, when we can observe the speed of the motion of the previously repaired fingers directly and in contrast to the normal fingers of the hand. I note that a considerable percentage of fingers having undergone flexor tendon repairs do not move as swiftly as normal fingers do when patient attempts quick flexion of all fingers, although the finger, finally, achieves a good range of active motion. In the experience of Elliot and Harris (2003), at 6 to 12 months after tendon repair, patients will sometimes say the finger is much better than when last seen in clinic. On measurement, the range of motion is unchanged and what these patients are noticing as better is increased speed of movement and better integration with the rest of the hand. This phenomenon of decreases in motion speed may relate to increases in the friction of the repaired tendon against tissues such as the sheath, the presence of adhesions which slow down tendon movement, or some residual extensor tethering or joint stiffness of the hand. All the above characteristics of finger motion affect finger function within an achievable motion range, but none are mirrored in existing criteria for judgment of the functional status of the hand. I record quality of motion as ''excellent'' when all three – motion arc, coordination and speed – appear normal; as ''good'' when any two are normal; as ''poor'' when only one, or none, is normal.

The functional status can be grouped into the five (or seven) categories shown in Table 1. By inclusion of different sets of joints into the evaluation of the active range of motion, this system may be extended as a framework for primary flexor tendon repairs in different parts of the hand. I use the sum of the ranges of motion of the PIP and DIP joints when determining outcomes of Zone 2 tendon repairs. As the levels of tendon injuries move distal or proximal to Zone 2, the number of the joints to be included into the evaluation can be decreased or increased appropriately. In addition, a system including assessment of quality of motion may also be desirable when assessing results of secondary surgery such as tendon grafting. The donor tendons such as the palmaris longus tendon are thinner than the FDP tendon and the frequent disturbance of the digital motion arc as a result can be factored into the assessment.

In recent years, we have obtained good to excellent treatment outcomes fairly consistently and avoided postoperative ruptures. It may not be sufficient to draw any final conclusions, but it appears that the three issues described in some details above, are critical to predictable outcomes viz. sufficient and proper release of the major annular pulleys, stronger surgical repair strength and appropriate, and mechanically sound, postoperative mobilisation. By dealing appropriately with these critical aspects of flexor tendon repairs, we seem to be seeing the dawn of days of treating Zone 2 flexor tendon injuries with predictable results. Of these issues, release of the pulleys and post-operative mobilisation protocols seem to be more important than

Table 1—Criteria of assessment of functional outcomes of flexor tendon repairs

Motion range (%)	*Grip strength*[1]	*Quality of motion*[2]	*Function grade*[3]
90–100	+	Excellent or good	Excellent +
	−	Poor	Excellent −
70–89	+	Excellent or good	Good +
	−	Poor	Good −
50–69			Fair
30–49			Poor
0–30			Failure

[1]Grip strength is recorded as (+) when it is greater than that of the contralateral hand (the non-dominant hand), or over 70% of that of the contralateral hand (dominant hand). Otherwise, grip strength is considered abnormal and recorded as (−).

[2]Quality of motion is rated on a basis of direct observation of finger motion by surgeons. It is recorded as ''excellent'' when all three aspects, viz. motion arc, coordination and speed, appear normal; as ''good'' when any two are normal; as ''poor'' when only one, or none, is normal.

[3]The overall function is graded as excellent (+) when grip strength is (+) and quality of motion is excellent or good; the function is graded as excellent (−) when either the grip strength is (−) or quality of motion is graded ''poor'', on the basis of return of 90% to 100% normal motion range.

Table 2—Three critical aspects of flexor tendon repairs in Zone 2

Aspect	*Treatment*	*Significance*
Release of critical pulleys	Partial (2/3) release of the A2 pulley, or complete release of the A4 pulley, with extension to the adjacent synovial sheath, as necessary, to ensure an ample length of release for free tendon gliding.	Eliminate an essential feature of Zone 2 to reduce restriction of tendon motion.
Stronger surgical repairs	Employ a four- or six-strand repair for the FDP tendon, with pre-tension, and a four-strand repair for FDS tendon.	Increase baseline strength, thus ensuring a greater safety margin during motion.
Mechanically sound motion protocols	Active motion under least tension, synergistic with wrist position; combining passive motion into an active motion regimen; avoiding active motion over the vulnerable range; emphasise full extension and flexion in separate parts of the recovery period.	Reduce tension during active finger motion; Achieve functional or full active motion reliably.

surgical suture strength. Release of the annular pulleys alters the anatomical features of Zone 2, eliminates a structural feature which restricts tendon gliding to produce an extremely high local load on the tendons and, fundamentally, converts flexor tendon repairs in Zone 2 into something similar to repairs outside this region. An adequate motion protocol avoids tension to the tendon above the safety limits of even a multi-strand surgical repair. A multi-strand repair would still rupture if the repair site was entrapped, or compressed, by a strong pulley, or if an extremely high load to the tendon is not avoided during active finger flexion. This may explain why multi-strand repairs alone have not completely eliminated rupture of repairs. Table 2 summarises a few of these considerations which we consider pertinent to a reliable outcome of primary Zone 2 flexor tendon repair. It is highly possible that refinements, or modifications, can be made to our currently used methods and that there may be more than one path to the eventual goal of predictable outcomes.

My final consideration regards the significance of improving intrinsic healing capacity and, so, influencing the abovementioned critical issues of this surgery. Through a series of in vitro and in vivo studies over the past several years, my colleagues and I have been able to achieve repaired tendon strengths of 150% to 170% of that of non-treatment controls within 2 to 5 weeks of surgery using gene therapy in a complete chicken tendon laceration model. This shows the potential of molecular treatment, which tackles the problems of weakness in healing in the early stages and reverses the status of "non-gain" in the tendon strength over the initial weeks of the healing process to a steady increase of the repair strength. However, in biomechanical studies, we have noted a decrease in repair strength by approximately half and an increase in the resistance to tendon motion by as much as 5- to 10-fold as the flexing finger approaches full flexion. A molecularly enhanced tendon is probably still unable to tolerate this extreme mechanical disadvantage. Therefore, even with the advent of molecular therapies, the need for surgery and rehabilitation to avoid mechanical disadvantages to the tendons is likely to continue to be necessary within the foreseeable future. Molecular therapies, if they eventually come into clinical use, will increase healing strength, allowing surgeons and therapists to be more comfortable about postoperative motion, and should provide particular benefit to tendons that are severely traumatised and have lost their healing capacity.

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