Surgical release of the A1 pulley for treatment of trigger finger normally produces excellent results. However, in patients with long-standing disease, there may be a persistent fixed flexion deformity of the proximal interphalangeal joint. This is sometimes due to a degenerative thickening of the flexor tendons and may be treated by resection of the ulnar slip of flexor digitorum superficialis tendon. One hundred seventy-two patients (228 fingers) who had undergone this procedure were reviewed at a mean follow-up of 66 months. Mean pre-operative fixed flexion deformity of the proximal interphalangeal joint was 33°. All but eight fingers were improved by surgery and there was an average gain of 26° in passive extension (7° residual fixed flexion deformity) of the proximal interphalangeal joint. Full extension was attained in 141 of the 228 fingers, and in all 101 fingers with a pre-operative loss of passive extension of 30° or less. This technique is indicated for patients with loss of passive extension in the proximal interphalangeal joint and a long history of triggering.

**Keywords:** trigger finger, extension deficit, flexor digitorum superficialis, ulnar slip, hemi-resection

**INTRODUCTION**

The majority of trigger fingers resolve after release of the A1 pulley. However, there is a subset of patients who present with a long history of trigger finger and locking of the proximal interphalangeal joint in flexion. These patients may develop a flexion contracture at proximal interphalangeal joint level which persists after release of the A1 pulley (Wolfe, 1999). This fixed flexion deformity may be due to degenerative enlargement of the flexor tendons within the fibrous digital canal, caused by chronic repetitive friction. This predominantly affects the flexor digitorum superficialis (FDS) tendon and restricts FDS and flexor digitorum profundus (FDP) excursion through the A1 and A2 pulleys. Thus, simple release of the A1 pulley may not restore full proximal interphalangeal joint extension.

**PATIENTS AND METHODS**

**Material**

This retrospective study reviews the long-term results of resection of the ulnar slip of the FDS tendon for the treatment of long-standing trigger fingers with proximal interphalangeal joint contractures. Most of the patients with trigger finger in our practice had no interphalangeal joint contracture and were treated by simple release of the A1 pulley and were not included in this study. The following categories of patients with trigger finger(s) and the proximal interphalangeal joint contracture were also excluded: patients in whom this loss of extension disappeared per-operatively, after sectioning of the A1 pulley or in whom this was due to intra-articular pathology (confirmed by standard antero-posterior and lateral radiographs); patients suffering from rheumatoid arthritis or receiving haemodialysis treatment.

Two hundred and fourteen patients with this pathology were recruited over a 17-year period (1985–2001) from a total of 883 patients seen for trigger finger. Of these, 42 were lost to follow-up or were in the first 6 postoperative months at the time of this study. Thus, 172 patients were included in the study. There were 38 men (22%) and 134 women (78%), with a mean age of 67 (range, 24–87) years. Twelve patients had this type of operation on both hands (184 hands treated). All the patients had a long history of triggering or snapping with gradual worsening of symptoms over time. The mean duration of symptoms before surgery was 48 (range, 6–120) months and 19 (11%) had previously undergone an isolated release of the A1 pulley with a poor result, due to a residual fixed flexion deformity of the proximal interphalangeal joint. Additionally, 21 patients (12%) had a pre-operative loss of active flexion in the proximal interphalangeal joint.

Surgery was performed on 228 fingers, usually the middle or ring finger (87%). In 80% of the hands only one finger was involved, but in 17% two fingers were involved, usually the middle and ring fingers. When three fingers were involved in the same hand (3%), they were always neighbouring digits.

Passive and active range of motion at the proximal interphalangeal joint was recorded before surgery and at the final follow-up visit. Pre-operatively there was a mean loss of passive extension (fixed flexion deformity) at the proximal interphalangeal joint of 33° (range, 20°–60°). The mean follow-up period was 66 (range, 12–154) months.
Operative technique

All surgical interventions were performed by the senior author (DLV) under regional block anaesthesia. The skin incision varied with the number of involved digits. If there was only one affected finger, a Bruner-type palmar-digital incision was used (Fig 1), which extended from the proximal palmar crease to the proximal half of the middle phalanx. If two or more fingers were involved, transverse incisions were made over their metacarpophalangeal joints, just distal to the distal palmar crease. Additional Bruner-type digital incisions were then made over the proximal phalanx and the proximal half of the middle phalanx of the involved fingers.

The A1 pulley was divided, except in the 19 patients in whom it had already been released. Active finger flexion was simulated by pulling on FDS and FDP tendons in the palm. Additionally, the test described by Brunelli et al. (2001) (application of pressure on the forearm flexor muscle bellies at their musculotendinous junction) was performed to assess whether there were adhesions around the flexor tendons proximal to the palmar incision. Passive proximal interphalangeal joint extension was then tested, with special attention to the quality of the gliding of the flexor tendons into the digital flexor sheath. If there was full extension of the proximal interphalangeal joint, the procedure ended and the skin was closed. If, however, extension was restricted (as was the case in all the patients in this series), then the FDS tendon was closely examined for enlargement between the levels of the divided A1 and A2 pulleys. Frequently the part of the tendon meant to glide under A1 and A2 pulleys during finger movements was enlarged and very degenerate, and had lost its normal surface smoothness and brightness, and there was longitudinal splitting of its fibres (Fig 2). Similar changes occurred less frequently and to a lesser degree in the FDP tendon.

![Fig 1](image1.png)  
(a) For involvement of a single finger, a Bruner palmar-digital incision is used. (b) For two-finger involvement a transverse incision over the metacarpophalangeal joints (just distal to the distal palmar crease) with Bruner digital incisions is used.

![Fig 2](image2.png)  
Per-operative view of the middle finger flexor tendon sheath at the level of the metacarpophalangeal joint. The black asterisk is at the most proximal level of flexor tendon sheath and the A1 pulley has been released. The white asterisk is on the A2 pulley. Degeneration (thickening and enlargement) of the FDS tendon (white arrow) is pronounced under the A1 and A2 pulleys. The FDP tendon (black arrow) is much less affected.
This prevented complete unrestricted extension of the proximal interphalangeal joint despite sectioning of the A1 pulley, because the enlarged tendon(s) could not slide under the A2 pulley.

Next the ulnar slip of the FDS tendon was resected from the distal margin of the carpal tunnel to the distal edge of the A3 pulley. The finger involved was placed in full flexion and the radial and ulnar slips of the FDS tendon were identified distally and split longitudinally in a proximal direction by exerting gentle lateral force with two tendon hooks (Fig 3). The wrist was then flexed, and the ulnar slip of the FDS tendon was sectioned as far proximally as possible (Fig 4), at about the distal margin of the carpal tunnel. The flexor tendon sheath was then opened between the A2 and A3 pulleys, the FDP tendon was retracted radially and the ulnar slip of the FDS tendon was extracted from under the A2 pulley (Fig 5). This was performed by carefully rolling the tendon slip around a haemostat as if opening a tin of anchovies. The ulnar slip was then detached from the chiasma of the FDS tendon (Fig 6) and sectioned distally, below the distal edge of the A3 pulley, with the middle phalanx fully flexed (Fig 7). Care was taken to preserve the A3 pulley and, if located, the vinculum brevis was protected.
Extraction of the ulnar slip of the FDS tendon distal to the A2 pulley was quite simple if the fixed flexion deformity of the proximal interphalangeal joint was less than 30°. However, it was more difficult if there was significant enlargement of the flexor tendons or adhesions between them. If the tendon slip was too enlarged to be pulled through the A2 pulley from proximal to distal, it was sectioned distally and then pulled proximally, in order to avoid a rupture of the A2 pulley. If adhesions between the contents of the fibrous flexor sheath prevented extraction of the slip, a loop of 3-0 monofilament suture was passed around the ulnar slip of the FDS tendon proximal to the A2 pulley. The ends of the loop were then passed under the A2 pulley and pulled longitudinally to release the adhesions around the ulnar slip (Le Viet, 1997).

After resection of the ulnar slip of the FDS tendon, the range of motion of the proximal interphalangeal joint was assessed and the excursion of both the FDS and FDP was checked by pulling on these tendons in the palm. If there was any persistent fixed flexion deformity of the proximal interphalangeal joint, then its palmar plate was exposed and its check-rein ligaments were divided at their insertions onto the proximal phalanx.

**Postoperative care**

Postoperative care consisted of self-rehabilitation, started on the second postoperative day. If there was a persistent fixed flexion deformity of the proximal interphalangeal joint, then a dynamic extension splint was prescribed.

**RESULTS**

**Proximal interphalangeal joint function**

Postoperative evaluation showed that the residual fixed flexion deformity at the proximal interphalangeal joint averaged 7° (range, 0–60°). There was an average gain of 26° (range, 0–50°) in passive extension of the proximal interphalangeal joint at the final follow-up visit. The difference between pre- and postoperative extension deficits was statistically significant (\( P < 0.0001 \), paired \( t \)-test).

In 141 (62%) fingers a full range of motion was achieved at final follow-up. (Fig 8). A deficit in passive extension (fixed flexion deformity) of the proximal interphalangeal joint of between 5° and 15° was seen in 51 (22%) fingers and one of between 15° and 30° was seen in 27 (12%) fingers. A fixed flexion deformity of greater than 30° was found in nine (4%) fingers, all of which also had restricted active proximal interphalangeal joint flexion.

Full extension of the proximal interphalangeal joint was restored in all 101 fingers with a pre-operative fixed flexion deformity of 30° or less. For the 127 fingers with a pre-operative loss of passive extension of more than 30°, an average 30° (range, 0–50°) gain in extension was achieved, leaving, on average, a 12° (range, 0–60°) fixed flexion deformity. Forty of these 127 fingers achieved a full range of motion.

It is to be noted that in all cases in which full correction was not achieved, the surgery substantially
reduced, but did not eliminate, the fixed flexion deformity of the proximal interphalangeal joint. In some of these cases this worsened during post-operative follow-up, but in only eight fingers did it return to the pre-operative level.

Complications

Rupture of the A2 pulley occurred in two fingers when trying to pull the thickened ulnar slip of the FDS tendon under it. This intra-operative complication was treated by pulley reconstruction with the palmaris longus tendon. This lengthened the convalescence period but resulted in satisfactory outcomes.

Nine patients developed reflex sympathetic dystrophy which resulted in permanently restricted finger movement in three patients.

There were no infections, but 16 patients experienced mild hypersensitivity of their operation scars which lasted for some months. Another 11 reported that their scars felt hard, but none of these 27 patients considered that these problems interfered with function.

DISCUSSION

Surgery for trigger finger is indicated when conservative treatment fails to relieve the patient’s symptoms.

In some patients, finger triggering and limitation of motion are due to tendon enlargement distal to A1 pulley. Seradge and Kleinert (1981) reported two such cases with involvement of FDP tendon and triggering under the A2 pulley. A reduction flexor tenoplasty was proposed for this type of triggering in order to avoid division of the A2 pulley. Triggering of the FDP tendon under the A3 pulley has also been reported (Rayan, 1990), and release of this pulley has been proposed as treatment.

In all our patients, the persistent limitation of the proximal interphalangeal joint motion after release of A1 pulley was due to degenerate enlargement of the FDS tendon. These patients were usually old and had long-standing triggering: thus chronic irritation beneath the A1 pulley may have led to the degeneration and enlargement of the FDS tendon, and to a lesser degree the FDP tendon, which prevented free gliding of the flexor tendons beneath the A2 pulley. Complete extension of the proximal interphalangeal joint might have been achieved by section of not only the A1, but also the A2, pulley. However, this may cause bowstringing, with subcutaneous prominence of the flexor tendons resulting in a major disturbance of finger motion biomechanics (Heithoff et al., 1988).

Enlargement of the A1 pulley, reported by Kapandji (1983), is an alternative option for treatment of trigger finger but is technically difficult and does not correct the tightness of the digital sheath at A2-pulley level, which was responsible for the loss of passive extension in our series. Even if the A2-pulley was also enlarged, this would have the major disadvantage of precluding immediate unprotected rehabilitation and might result in adhesions between the healing pulley and the degenerate FDS tendon.

We have used our technique since 1985 in carefully selected patients with a long history of trigger finger(s) and a flexion contracture of the proximal interphalangeal joint. Initially the procedure was performed for triggering in patients on haemodialysis for renal insufficiency who had lost extension and/or active flexion of the finger(s) (Le Viet and Gandon, 1992). Excision of the whole FDS tendon was first proposed by Wissinger (1971) to treat rheumatoid fingers with tendon entrapment in the digital sheath. Resection of only the ulnar slip of the FDS tendon was subsequently described by Ferlic and Clayton (1978) for the treatment of stenosing flexor tenosynovitis in rheumatoid patients. This seems to significantly reduce the recurrence of tenosynovitis and the re-operation rate (Wheen et al., 1995), and conservation of the radial slip of FDS tendon seems to prevent ulnar dislocation of the flexor apparatus and the development of a “swan-neck” deformity (Ferlic and Clayton, 1978).

To our knowledge, the technique of resection of the ulnar slip of the FDS tendon has never been used before to treat long-standing trigger fingers in non-rheumatoid patients. In our series this technique restored full extension of the proximal interphalangeal joint if the pre-operative fixed flexion deformity was 30° or less. Proximal interphalangeal joint function was improved in those patients with a pre-operative fixed flexion deformity of more than 30°. Section of the proximal attachments of the proximal interphalangeal joint palmar plate (check-rein ligaments) did not seem to substantially improve the final range of motion, as any gain in extension due to this was lost during convalescence.
Resection of the ulnar slip of the FDS tendon requires a postoperative rehabilitation program in order to maintain the range of motion of the proximal interphalangeal joint attained at surgery, and a dynamic extension splint may be needed if the pre-operative loss of passive extension exceeded 40°. Thus, the postoperative follow-up is generally longer than for standard treatment of trigger finger.

We believe this technique does not interfere with the vascularity of the flexor tendons, as long as care is taken to preserve the vinculum brevis at the level of the neck of the proximal phalanx. However, histologic studies of some resected FDS tendon slips have shown that these enlarged, ulcerated slips are already quite devascularized.

References


