Trigger Digits: Principles, Management, and Complications

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Stenosing tenosynovitis, or trigger finger, is an entity seen commonly by hand surgeons. This problem generally is caused by a size mismatch between the flexor tendon and the first annular (A-1) pulley. Conservative management includes splinting, corticosteroid injection, and other adjuvant modalities. Surgical treatment consists of release of the A-1 pulley by open or percutaneous techniques. Complications are rare but include bowstringing, digital nerve injury, and continued triggering. Some patients require more extensive procedures to reduce the size of the flexor tendon. Comorbid conditions affect how trigger finger is treated. Patients with rheumatoid arthritis require tenosynovectomy instead of A-1 pulley release. In children trigger thumb resolves reliably with A-1 pulley release but other digits may require more extensive surgery. In diabetic patients trigger finger often is less responsive to conservative measures. An understanding of the pathomechanics, risk factors, and varied treatments for trigger finger is essential for appropriate care. (J Hand Surg 2006;31A:135–146. Copyright © 2006 by the American Society for Surgery of the Hand.)

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Stenosing tenosynovitis, or trigger finger, is diagnosed when a patient presents with a symptomatic locking or clicking of a finger or the thumb. It is caused by a mismatch between the volume of the flexor tendon sheath and its contents. As the flexor tendon attempts to glide through a relatively stenotic sheath, it catches, producing an inability to flex or extend the digit smoothly. In more severe cases the finger may become locked in flexion, requiring passive manipulation of the finger into extension. The patient may complain initially of a painless clicking with finger movement. This often progresses to painful triggering, which is localized variably to the palm or the metacarpophalangeal (MCP) or proximal interphalangeal (PIP) joints. Reluctance to range the digit fully because of pain or locking can lead to secondary contracture at the PIP joint. Middle-aged women are the age group most often affected. The most commonly involved digit is reported to be the ring finger or thumb—with the index and small fingers being the least symptomatic. It is not unusual for a single patient to have multiple trigger digits. Trigger fingers first were described by Notta[1] in 1850. The diagnosis and treatment of an uncomplicated primary trigger digit is quite familiar to most primary care providers, orthopedic surgeons, and hand surgeons. Certain patient populations, however, including children, diabetic patients, those with rheumatoid arthritis, distal triggering, PIP joint contractures, or conditions that cause systemic deposition of protein require special consideration. Complications of treatment such as bowstringing of the flexor tendon are unusual but occasionally do occur. A number of surgical techniques in addition to the traditional open A-1 pulley release have been described to treat these more complex scenarios.

Primary Trigger Finger

The vast majority of trigger digits are primary idiopathic trigger fingers or thumbs in which the site of obstruction is the first annular (A-1) pulley (Fig. 1). Power grip causes high angular loads at the distal edge of the A-1 pulley. Hueston and Wilson[2] proposed that chronic repetitive friction between the flexor tendon and the enclosing sheath caused a reactive intratendinous nodule. They compared this with the fraying that occurs at the end of a piece of thread after it has been passed numerous times through the eye of a needle.

Histologic analyses of diseased A-1 pulleys and superficialis tendons from patients with trigger digits have shown fibrocartilaginous metaplasia.[3] The cells stain positive for S-100, a protein that is found in

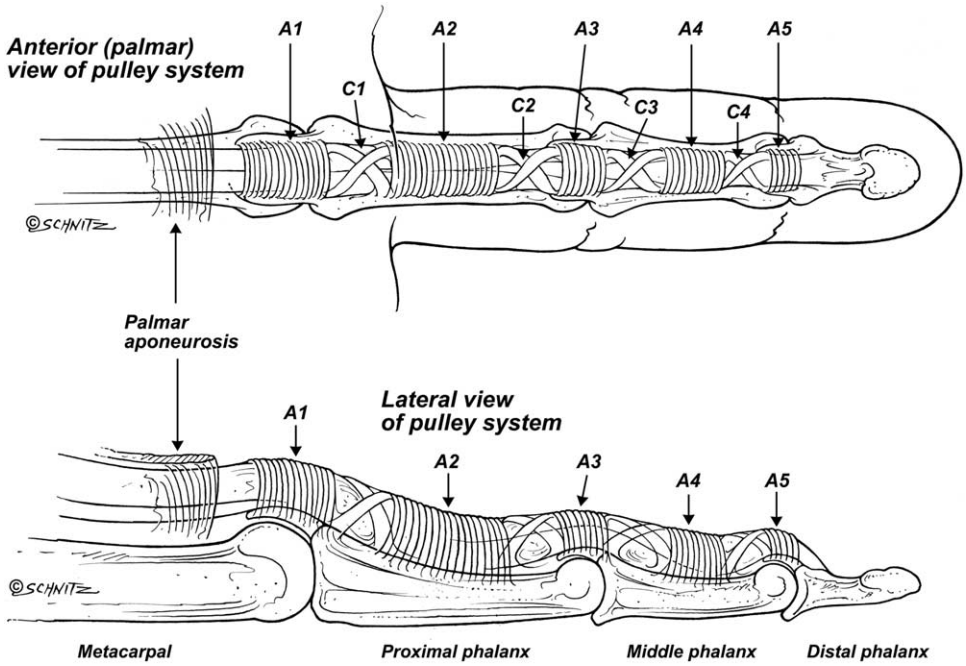

Figure 1. The digital pulley system of the fingers.

cartilage. The A-1 pulley may triple in thickness as the histologic inner gliding layer of the A-1 pulley changes from the spindle-shaped fibroblasts and ovoid cells normally seen to cells with chondrocyte characteristics.

It has been proposed that *tendovaginitis* is a more accurate term to describe the condition than *tenosynovitis*. This is because the pathologic inflammatory changes are found in the retinacular sheath and peritendinous tissue rather than in the tenosynovium. The 2 terms continue to be used interchangeably in the literature.

Conservative Treatment

Activity modification, nonsteroidal anti-inflammatory drugs, splinting, steroid injection, and surgical release all have been used in the management of trigger finger. If the patient's history shows that a specific activity is associated with the onset of triggering then avoidance of that activity may result in spontaneous resolution of the tendovaginitis. For patients who do not have a contraindication such as renal disease or peptic ulcer disease, nonsteroidal anti-inflammatory drugs may be added to an initial treatment regimen.

Splinting is another conservative treatment option. A custom-made splint to hold the MCP joint of the involved finger at 10° to 15° of flexion with the PIP and distal interphalangeal (DIP) joints left free has been applied with some success. The splint is worn continuously for an average of 6 weeks. In patients with marked triggering, symptoms of longer than 6 months duration, and involvement of multiple digits or of the thumb, splinting alone does not eliminate the triggering. Splinting appears to be a reasonable option for patients with mild triggering who do not wish to undergo a steroid injection or as an adjuvant to injection.

Corticosteroid Injection

Long-acting corticosteroid injection is the mainstay in initial management of the symptomatic trigger digit. Injection of the involved flexor tendon sheath provides long-term relief of symptoms in 60% to 92% of affected digits with up to 3 injections.[4] Betamethasone sodium phosphate is the steroid of choice because it is water soluble, does not leave a residue in the tendon sheath, is not known to cause tenosynovitis, and it causes less fat necrosis if the injection is placed in the tissue around the tendon sheath. Other corticosteroids such as triamcinolone and methylprednisolone also have been used successfully.

A diminished response to injection has been associated consistently with an increased duration of symptoms, usually more than 4 to 6 months, and with an increasing number of injections.[5] This may be a result of the inability of corticosteroids to reverse fibrocartilaginous metaplasia of the stenotic A-1 pulley once it has occurred. Benson and Ptaszek[6] reported a 60% success rate for a single injection. Of those treated with a second injection, 36% were asymptomatic at 3 months. Six patients were injected a third time, none of whom had long-term relief.

Various techniques of injection have been used effectively. Both palmar and lateral approaches can

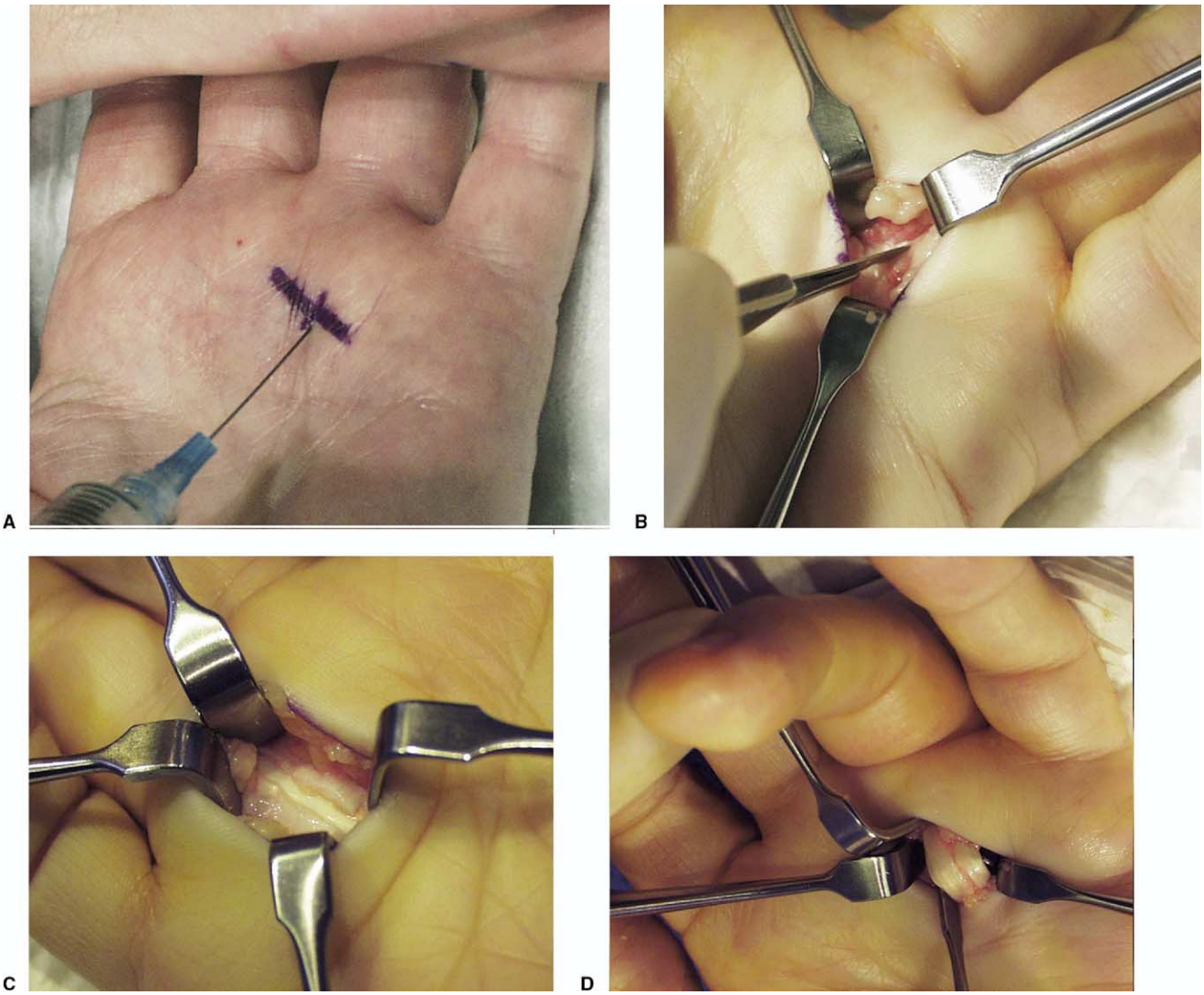

Figure 2. Intraoperative photographs of an open trigger digit release. (A) Injection of local anesthetic. (B) After appropriate exposure of the A-1 pulley a knife is used to incise the pulley longitudinally. (C) Exposure shows complete release of the A-1 pulley (check for any tightness of the palmar pulley of Manske and if so release as well). (D) A traction tenolysis of the FDP and FDS tendons may be performed to check that all triggering is gone.

be used to infiltrate the flexor tendon sheath with corticosteroid and local anesthetic. Patients should be warned that fat necrosis or skin depigmentation are potential complications of subcutaneous injection. Intrasheath injections generally do not result in complications; however, tendon rupture has been reported and is likely the result of inadvertent intratendinous injections leading to collagen necrosis.

Surgical Considerations

Open release of the A-1 pulley has been used to treat trigger digits for more than 100 years. Some surgeons prefer to perform an open A-1 pulley release under local anesthetic so the absence of triggering can be seen intraoperatively before closure of the wound. Others believe that local anesthetic distorts the surgical anatomy and therefore prefer a Bier block. Transverse, longitudinal, or oblique incisions on the volar aspect of the hand overlying the MCP joint and A-1 pulley all have been described. Blunt dissection is continued down to the level of the flexor tendon and the A-1 pulley is visualized (Figs. 2A, 2B), with care taken to protect the neurovascular bundles that are located on the radial and ulnar sides of the tendon sheath. The radial neurovascular bundle to the thumb is most at risk for injury because it takes an oblique ulnar to radial course across the A-1 pulley. This bundle also is subcutaneous, averaging 1.19 mm deep to the dermis at the thumb MCP flexion crease, and may be transected with a deep skin incision. The A-1 pulley should be released completely for symptoms to resolve reliably.

Generally only supportive dressings are needed after surgery. Some patients note palmar tenderness or finger stiffness after surgery. Incisional tender-

ness, however, generally resolves with time and scar massage and few patients require formal occupational therapy.

The results of open A-1 pulley release generally are excellent. Turowski et al,[7] in a group of 59 patients treated by a variety of surgeons, reported 97% complete resolution of triggering with no complications such as infection, bowstringing, or digital nerve injury. The 2 patients who did not have complete resolution of triggering did have notable improvement.

Annular Digital Pulleys

Division of the A-1 pulley usually causes minimal morbidity. Near-normal hand function can be maintained with only the A-2 and A-4 annular pulleys intact.[8] The 10% increased work of flexion that has been shown biomechanically after A-1 pulley excision does not appear to be relevant clinically for most patients. A-2 pulley injuries in rock climbers and reports of patients who have had part of their A-2 pulleys transected surgically show the importance of preserving this pulley in preventing bowstringing of the flexor tendon. The biomechanical studies of pulley excision by Peterson et al[8] showed a 44% increase in work of finger flexion after A-2 pulley excision and a 62% increase after removal of both A-1 and A-2 pulleys.

A distinct separation between the first and second annular pulleys is considered the usual configuration. Anatomic studies, however, have shown a nearly 50% incidence of continuity between the A-1 and A-2 pulleys.[9] The separation between the 2 pulleys generally is 0.4 to 4.1 mm. When the separation is not present, however, several millimeters of pronounced thinness of the retinacular tissue is observed at the usual site of separation.[10] Care should be taken not to extend a surgical release into the substance of the A-2 pulley. Rarely the A-2 pulley may be involved critically in producing a trigger digit. The treatment strategy for this special situation is discussed later.

Complications of Surgical Treatment

Bowstringing

Bowstringing after A-2 pulley injury manifests as a protrusion of the flexor tendon into the palm with finger flexion. It often produces a painful pulling sensation in the palm with associated failure to fully extend or flex the digit actively.

To understand fully the adverse effects of bowstringing it must be remembered that the effect of a tendon at a joint depends on both the tension on the tendon and the moment arm. Because bowstringing increases the perpendicular distance of the tendon from the MCP joint axis of rotation, the moment arm is increased.[11] With an increased moment arm the bowstrung flexor tendon gains a mechanical advantage that cannot be overcome actively by the extensors without manual correction of the flexor tendon.

Furthermore the available excursion of the flexor tendon does not increase with the increase in radius from the center of the MCP joint to the tendon. A rule of geometry states that when the radius of a circle moves through 57.29° (1 radian), any point on that circle moves through a distance equal to the radius. The distance and the required tendon excursion required to move the MCP joint through 57.29° increases with an increasing radius (Fig. 3). As the flexor tendon bowstrings and the radius of the moment arm across the MCP joints increases, a given distance of tendon excursion will move the joint through a smaller arc of motion. This increases the work of finger flexion. Because the amount of available excursion is generally the same as the required excursion for full range of motion, full excursion of the bowstrung flexor tendon moves the finger through a smaller less-than-full arc of motion.[11]

A-2 Pulley Injury

Inadvertent release of the A-2 pulley that results in clinically significant bowstringing is treated with an A-2 pulley reconstruction. The forces generated against the pulley in flexion are considerable. Therefore the reconstructed pulley must be strong, with an ideal length of approximately 10 mm, and should be tested vigorously under direct visualization on the operating table.

Bunnell[12] described pulley reconstruction by encircling a single loop of free tendon around the proximal phalanx deep to the extensor mechanism, which then was overlapped and sutured to itself. Either the palmaris longus or a slip of flexor digitorum superficialis was used as tendon graft. Modifications of this technique include the use of extensor retinaculum or anchoring of the tendon graft through the volar plate. The Weilby technique sutures the graft material to the fibrocartilaginous remnants of the rim of the disrupted pulley, which nearly always are present.[13]

Digital Nerve Injury

Digital nerve injury is an infrequent but serious complication of trigger finger release. Special care in protecting the radial digital nerve to the thumb and the index finger must be exercised because of their particular anatomy. Caution in the use of electrocautery is necessary to prevent potential thermal injury to the nerve. An accurate diagnosis is crucial to implementing the appropriate treatment. A digital

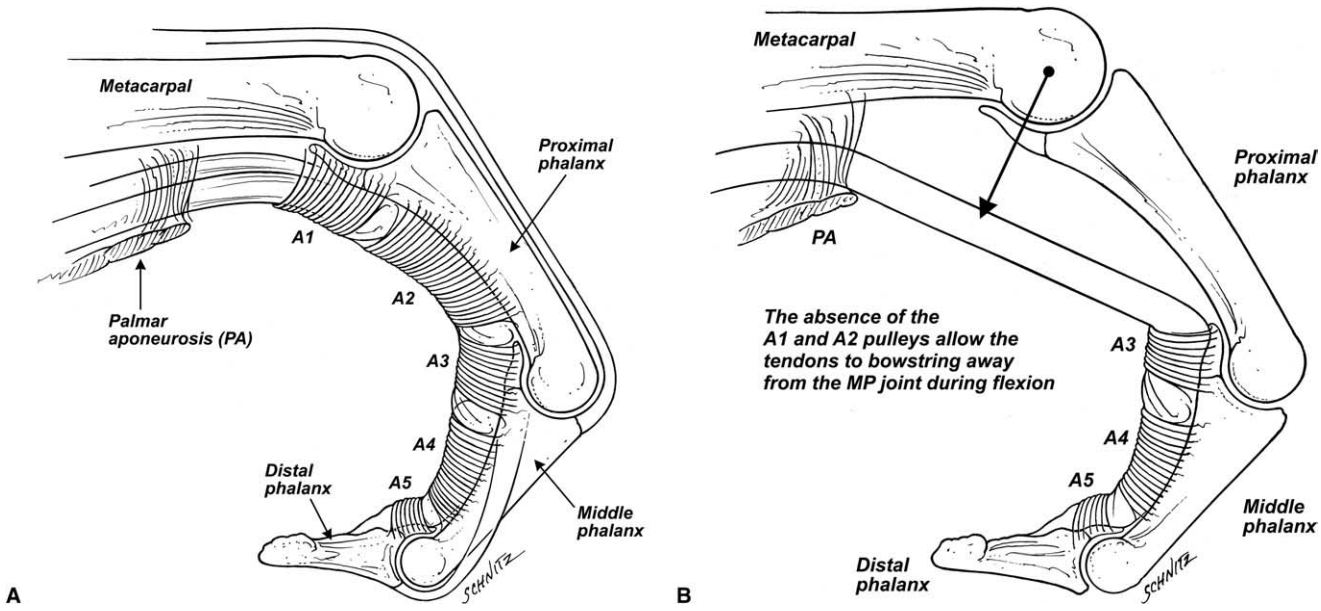

Figure 3. (A) Normal tendon mechanics with intact pulley system. (B) Bowstringing. The flexor tendon bowstrings across the MCP joint when both the A-1 and A-2 pulleys have been removed. This increases the radius from the center of rotation of the MCP joint to the tendon. The amount of tendon excursion necessary to move the joint through 1 radian (57.29°) of motion is equal to the distance of this radius. Therefore more tendon excursion is required to move a joint with a bowstrung tendon through a given arc of motion.

nerve that has been cut or cauterized should be explored and undergo microsurgical repair. If the surgeon is satisfied fully by direct observation before closure that the nerve was not transected or cauterized at the time of surgery then the injury likely is neuropraxic in nature and may resolve with expectant observation. If sensation does not return to the affected side of the digit by 3 months, however, then exploration of the nerve is indicated.

Alternative Procedures

Percutaneous Release

Percutaneous release of the A-1 pulley first was described in 1958 by Lorthioir.[14] The technique has gained popularity recently and a number of studies have evaluated the safety and efficacy of percutaneous release. Several instruments have been advocated for the procedure including a hypodermic needle, a tenotome, or specially designed knives.

The main concern with percutaneous release is digital nerve injury. Lorthioir used a fine tenotome without reported complications in 52 patients. Eastwood et al[15] used a 21-gauge hypodermic needle on 35 trigger digits and relieved symptoms in 94% without complications. Although Eastwood et al[15] released 3 thumbs in their study they noted that the obliquity and volar position of the neurovascular bundles in the thumb required particular caution. Cadaver studies have shown that the digital nerves in the index finger and thumb lie within 2 to 3 mm of the needle puncture site.[16]

Incomplete division of the pulley is another concern regarding percutaneous techniques. Pope and Wolfe[16] performed percutaneous releases in 13 trigger fingers using a 19-gauge needle and then proceeded to open the wound immediately and inspect the result. Although all patients showed clinical improvement, a complete release was found in only 8 patients; with the distal 10% to 15% of the pulley remaining intact in the other patients. They hypothesized that triggering resolves even if the distal edge of the A-1 pulley is not released.

Painful tenosynovitis without triggering often occurs in patients after a percutaneous release. This may be a result of the high rate of flexor tendon scoring. The rate of longitudinal laceration to the superficialis tendon in cadaveric studies has approached 100%.[16] The use of a corticosteroid along with local anesthetic may prevent the post-procedure inflammatory reaction, and the superficial scoring does not appear to have any clinically remarkable consequences.

The literature has shown both open and percutaneous methods of A-1 pulley release to be effective and safe for the treatment of trigger finger. In a prospective randomized study of 100 patients comparing the 2 techniques, Gilberts et al[17] successfully relieved symptoms in 100% of patients percutaneously and in 98% of patients treated with an open surgical procedure, with no complications. The 1 treatment failure was a result of excessive scar formation, causing recurrent triggering that required another procedure. These investigators

favored the percutaneous technique with the benefits of shorter procedure time (7 vs 11 min), shorter duration of postoperative pain (3.1 vs 5.7 days), quicker recovery of full hand function (7 vs 18 days after the procedure), and faster return to work (3.9 vs 7.5 days).[17]

Technique of Percutaneous Release

Percutaneous release can be performed in the clinic setting. Local anesthetic mixed with corticosteroid is administered and the palmar base of the affected finger is prepared sterilely. The patient is asked to flex the affected digit actively. The surgeon then hyperextends the finger. This brings the flexor tendon sheath directly under the skin and allows the neurovascular bundles to displace to either side.

An 18-gauge needle or other device is inserted at the proximal aspect of the A1 pulley. Care should be taken to stay centered over the flexor tendon sheath to avoid neurovascular structures and to enter the skin perpendicularly with the bevel of the needle parallel to the tendon. Alternatively some investigators have advocated inserting the needle slightly more distally in the middle of the pulley and then proceeding with release proximally and distally (Fig. 4).

The proximal edge of the A-1 pulley is located near the distal horizontal palmar crease for the small, ring, and middle fingers. For the index finger it is located at the proximal horizontal palmar crease. Release of the ring and middle fingers is believed to be relatively safe. The oblique course of the flexor tendons and neurovascular structures to the index and small finger, however, pose a greater challenge. Wilhelmi et al[18] described reliable landmarks for the small finger flexor tendon sheath in the area of the A-1 pulley as lying underneath a line connecting the ulnar border of the scaphoid tubercle proximally to the center of the proximal digital crease distally. For the index finger the landmarks were the radial border of the pisiform proximally and the midline of the proximal digital crease distally. By using these landmarks in a cadaver study the A-1 pulley was transected reliably. The distance from the scoring of the flexor tendon to the neurovascular bundles was 5.4 mm radially and 6.7 mm ulnarly in the small finger. In the index finger it was 8.5 mm radially and 6.2 mm ulnarly. None of the digital nerves or arteries were transected.

In the thumb the intersection of the proximal thumb digital crease and a perpendicular line up the central axis of the palmar aspect of the thumb is the preferred insertion site.

The needle may be inserted into the tendon. This is confirmed by needle movement when the patient flexes and extends the distal phalanx. The needle is withdrawn slowly until this motion ceases. The needle tip is now in the A-1 pulley. The A-1 pulley is cut by moving the needle forward and back while advancing it in line with the longitudinal axis of the flexor tendon sheath. A grating sensation indicates the A-1 pulley is being cut. Once the surgeon believes the pulley has been released adequately then the needle is withdrawn and the patient is asked to flex and extend the digit to show relief from triggering.

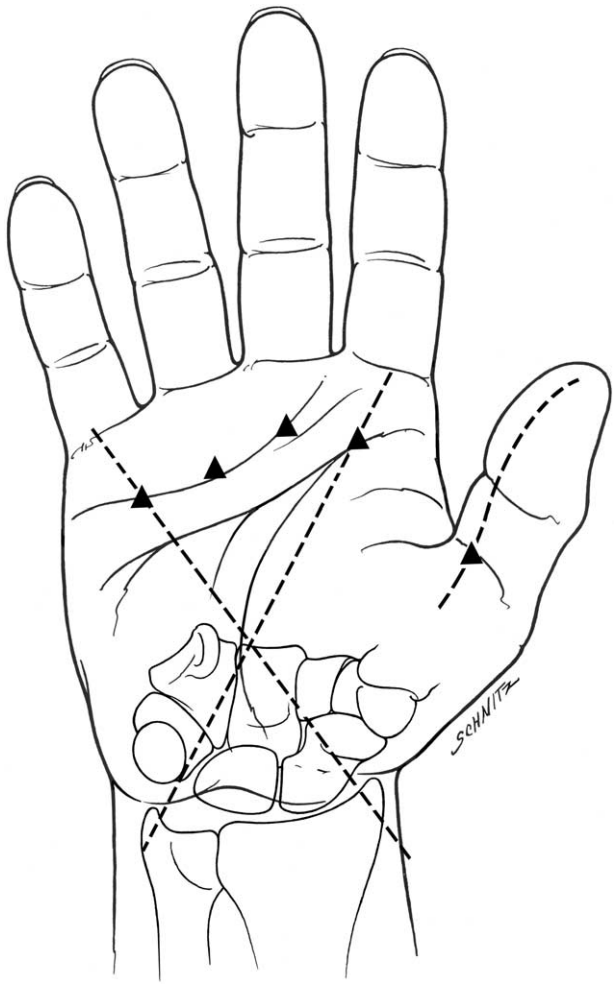

Figure 4. Use of surface landmarks for percutaneous A-1 pulley release. Index finger: at the proximal palmar crease at a line connecting the radial border of the pisiform and the center of the proximal digital crease of the index finger. Middle finger: at the distal palmar crease in the midaxis of the digit. Ring finger: at the distal palmar crease in the midaxis of the digit. Small finger: at the distal palmar crease at a line connecting the ulnar border of the scaphoid tubercle with the center of the proximal digital crease of the small finger. Thumb: at the proximal digital crease in the midaxis of the thumb.

Proximal Interphalangeal Joint Flexion Contracture and Distal Triggering

Patients with long-standing trigger finger rarely may develop flexion contracture at the PIP joint that persists after division of the A-1 pulley. In some patients this is caused by intra-articular pathology. Others have a preoperative flexion contracture that may resolve either

with a simple surgical release of the A-1 pulley or with additional postoperative therapy.

In other cases the involved flexor digitorum superficialis (FDS) tendon has degenerated markedly. The degenerative process may cause the tendon to lose its normal surface smoothness, fray its fibers, form an especially large nodule, and lose its ability to glide smoothly under the A-2 pulley. Treatment of these patients poses a challenge because function is not regained fully after A-1 pulley division and continuing the release into the A-2 pulley is not an option given the likelihood of bowstringing. These patients generally have very long-standing disease and likely already have had fibrocartilaginous metaplasia of their pulley and FDS tendon and may go on to distal triggering, defined as continued locking of the tendon because its excursion is blocked distally.

Ulnar Superficialis Slip Resection

Le Viet et al[19] described 228 such fingers in 172 patients who were treated with a resection of the ulnar slip of the superficialis tendon. His patients had an average of 48 months of preoperative symptoms and 11 had a previous A-1 pulley release that was unsuccessful.

The technique starts with a simple open A-1 pulley release. In a patient with a preoperative fixed PIP joint flexion contracture an attempt then is made by the surgeon to extend the PIP joint passively. If this attempt is successful the procedure is ended and the skin is closed. If the PIP joint is not extendable fully, however, the gliding of the flexor tendon through the A2 pulley is inspected closely. If direct visualization confirms this to be the site of restriction then the ulnar slip of the superficialis tendon is resected.[19]

Le Viet et al[19] used a Bruner palmar-digital incision to expose the tendon sheath to the middle phalanx. The ulnar slip of the FDS tendon is released at the distal aspect of the carpal tunnel and then at the distal edge of the A-3 pulley with care taken to preserve that pulley. The tendon slip then is delivered from the sheath through another incision placed between the A-2 and A-3 pulleys (Fig. 5).

In the series by Le Viet et al[19] all patients with a preoperative fixed flexion deformity of less than 30° were able to achieve full extension after ulnar superficialis slip resection (USSR). Patients with greater than 30° deformity improved their PIP joint extension by an average of 30°, with an average residual 12° of fixed flexion deformity. Two patients had an intraoperative A-2 pulley rupture that occurred when passing the tendon slip distally. Immediate reconstruction was performed and both patients had a good result despite longer rehabilitation. Three patients developed permanently restricted finger motion as a result of reflex sympathetic dystrophy. Conclusions from the study by Le Viet et al[19] were limited by the lack of a control group with preoperative PIP joint contracture treated traditionally. The results for patients who had a previously unsuccessful A-1 pulley release were not analyzed separately.

Reduction Flexor Tenoplasty

Reduction flexor tenoplasty is the removal of a central core from an enlarged tendon. Seradge and Kleinert[20] used the technique to treat patients with nodular triggering distal to the A-1 pulley. One of their patients had a previously unsuccessful A-1 pulley release with subsequent flexion deformity of the PIP joint. The other patient had normal-appearing A-1 pulleys and flexor tendons observed on open A-1 pulley release of multiple digits. This prompted further exploration, showing fusiform enlargement of the flexor digitorum profundus (FDP) tendon at the level of the A-2 pulleys. Reduction flexor tenoplasty resolved triggering in these patients and has been successful in other series of patients with distal triggering.

In principle a reduction flexor tenoplasty could be used in any location that a bulbous hypertrophy of the flexor tendon was an impediment to smooth gliding through the retinacular sheath. Generally, however, it is used only when there is triggering caused by a nodular swelling at the proximal or distal edge of the crucial A-2 pulley.

Initially the tendon nodule is exposed. On the proximal edge of the A-2 pulley this can be performed through an A-1 pulley release. Distally the second cruciform pulley can be resected at the level of the head of the proximal phalanx. A lateral incision, slightly longer than the bulbous swelling, is made through the epitenon and superficial tendon fibers. A central core of tendon then is excised (Fig. 6) until the remaining tendon is smooth and shows no restrictive nodularity. The tenotomy is closed with a running 7-0 suture.

A-3 Pulley Triggering

Triggering at the A-3 pulleys has been described rarely in bowlers as a result of repetitive trauma to the finger flexor apparatus at this site, in conjunction with intratendinous ganglia, or as a consequence of partial flexor tendon lacerations.[21] Physical findings in patients with A-3 triggering have been subtly different than the typical palmar tenderness. Pain and tenderness palmar to the PIP joint and swelling within the flexor tendon adjacent to the PIP joint is suggestive of this unusual variant. These patients characteristically have triggering that occurs when

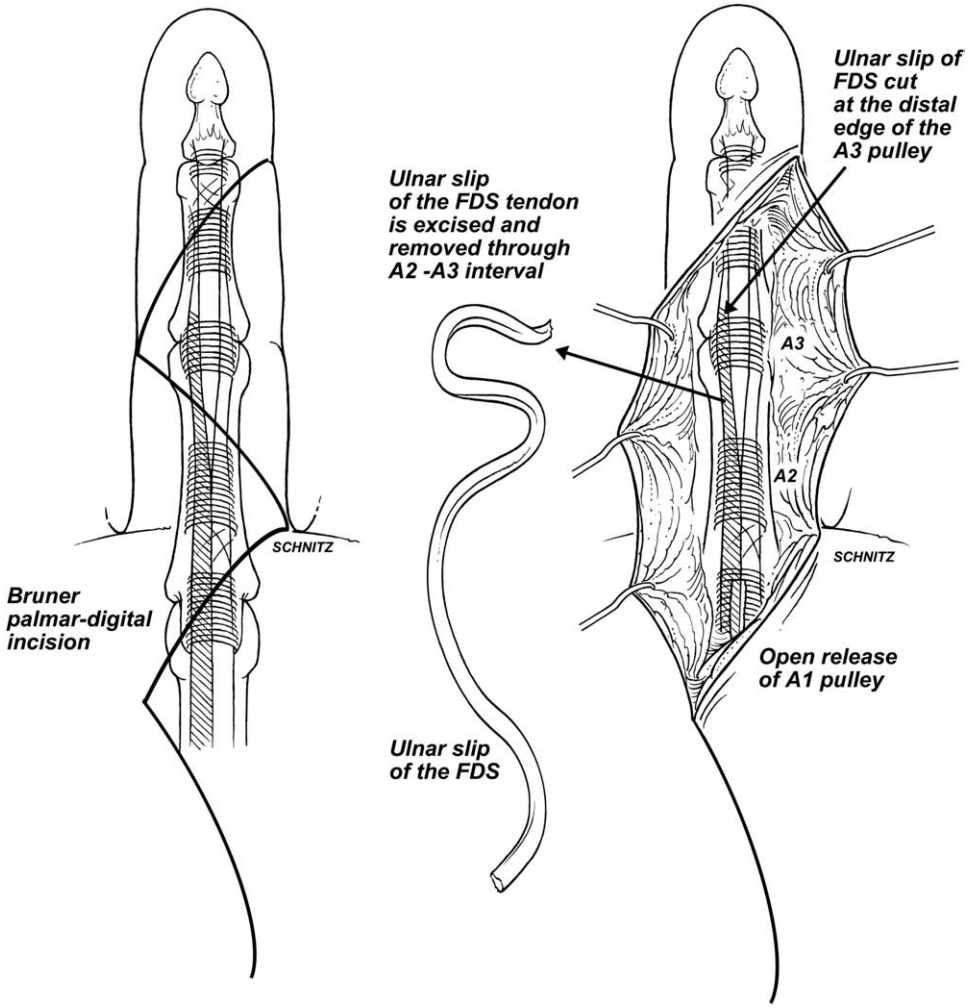

Figure 5. Ulnar superficialis slip resection. The proximal part of the ulnar slip has been released at the level of the carpal tunnel. Distally the ulnar slip has been transected distal to the A-3 pulley. The slip of tendon is delivered in the interval between the A-2 and A-3 pulleys.

the PIP joint is at or beyond 90° of flexion. In contrast to conventional triggering in which the FDS tendon is involved, it is FDP pathology that produces distal triggering; therefore symptoms are found at the DIP joint. When flexion deformity at the PIP joint is corrected passively in these patients it causes flexion at the DIP joint. Pain is reproduced with resisted DIP joint flexion.

A-3 pulley excision has been shown to be successful in these patients. In the case of intratendinous ganglia or fusiform nodules, which also impinge on the A-2 pulley, debulking of the enlarged tendon by reduction flexor tenoplasty has been effective.

Trigger Finger in Association With Other Diseases

Carpal Tunnel Syndrome

Carpal tunnel syndrome often is co-existent with trigger fingers. Patients with endocrine and metabolic diseases are known to be predisposed to both conditions. Increased median nerve latency at the carpal tunnel, however, has been shown in patients with idiopathic trigger finger alone. It is hypothesized that the association between the 2 conditions may be caused by an inflammatory process in the tendons at both the level of the A-1 pulley and the carpal tunnel. Furthermore when a patient avoids finger motion because of painful triggering the resultant hand edema may aggravate median nerve compression under the transverse carpal ligament. Clinically the 2 conditions often co-exist. Patients with trigger fingers should be evaluated thoroughly for clinical symptoms of carpal tunnel syndrome and vice versa.[22]

Systemic Disease

Amyloidosis. Amyloidosis most frequently results from the inability of dialysis membranes to remove β-2 microglobulin protein from filtered plasma. The protein accumulates in bones and soft tissues, causing a number of musculoskeletal complications. The severity of disease is proportional to the duration of

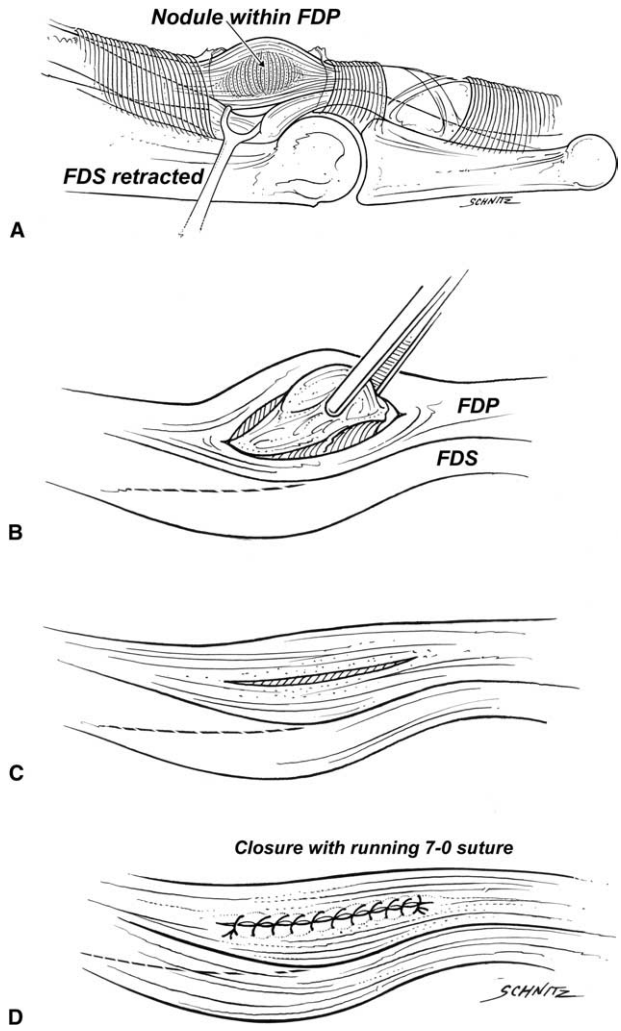

Figure 6. Reduction flexor tenotomy. A central core from the involved area of bulbous swelling on the flexor tendon is removed. (A) Bulbous enlargement with tendon proximal to A-2 pulley. (B) Excision of central core of FDP tendon. (C) Smooth gliding surface of tendon after excision of bulbous portion. (D) Tenotomy closed with running 7-0 suture.

time a patient has been on hemodialysis. The most common manifestations in the hand are cystic lesions in the carpal bones and destructive arthropathy; carpal tunnel syndrome is the most common condition requiring surgery.

Infiltrative amyloid tenosynovitis often extends distally to the palm and digits, which may cause trigger fingers, flexion contracture, or even tendon rupture. A large number of these patients will develop hand dysfunction that affects activities of daily living and self-care.[23]

Surgical treatment should consist of A-1 pulley release and complete tenosynovectomy with preservation of all other annular pulleys. A small number of older patients with idiopathic trigger digits have been found to have amyloid deposits in their excised tendon sheaths. This, however, is a distinctly different type and quantity of amyloid than the abundant β-2 microglobulin found in dialysis patients. Whether this is a part of the mechanism of idiopathic stenosing tenosynovitis or an incidental part of the aging process is unknown.

Mucopolysaccharidosis. Mucopolysaccharidoses are lysosomal storage diseases that result from genetic enzyme deficiencies. Several different clinical variants of mucopolysaccharidosis have been described, each resulting from the deficiency of 1 specific enzyme. The musculoskeletal manifestations result from the accumulation of glycosaminoglycans including dermatan, heparan, keratan, or chondroitin sulfate in cartilage, tendon, and joint capsule. Management of these associated conditions has taken on increased importance since the 1980s when bone marrow transplantation resulted in improved mentation and longer life expectancy for affected patients.

Van Heest et al[24] reported their experience treating hand disorders in 22 children with various mucopolysaccharidosis disorders. In their series 17 patients were treated for carpal tunnel syndrome along with 45 trigger digits in 8 affected children. They used a combination of A-1 and A-3 pulley releases with variable use of USSR. All of their patients had improved active digital flexion and overall improved function after this surgical release.

Diabetes mellitus. Diabetic patients are known to be at risk for developing dysfunction of many different organ systems. Although attention often is focused on the eyes, kidneys, feet, and cardiovascular system, the hand is affected frequently, resulting in both disability and deformity. Carpal tunnel syndrome, neuropathy, Dupuytren's disease, and trigger finger all have been reported with increased incidence in diabetic patients. Not only do patients with diabetes mellitus have a higher incidence of stenosing tenosynovitis, but these patients also are less responsive to treatment. The duration of diabetes has been related strongly to the incidence of hand complications.

Griggs et al[25] treated 54 diabetic patients with 121 trigger digits by corticosteroid injection, and they performed an open A-1 pulley release in those patients who failed conservative measures. Their overall success rate of 50% with corticosteroid injection is significantly less than that reported in most studies of nondiabetic patients. Furthermore insulin-dependent diabetic patients had resolution of symptoms in only 44% of cases with injection. Surgically treated patients also had somewhat compromised results, including residual PIP joint flexion contracture and persistent A-1 pulley tenderness. The reason for diminished results in the treatment of diabetic trigger

digits may be the higher incidence of a diffuse inflammatory stenosis of the tendon sheath rather than a focally nodular process.

Studies have reported poor glucose control in patients for several days after steroid injection for trigger fingers but none have documented the incidence, extent, or management of such phenomena. Patients should be advised of the possibility of increased blood sugar levels after steroid treatment but the presence of diabetes should not be considered a contraindication for flexor tendon sheath injection.

Diabetic hand complications are believed to be primarily fibrosing processes related to the same pathogenic mechanisms that induce other diabetic complications. Hyperglycemia increases collagen cross-linking while conferring a resistance to degradation, therefore causing collagen accumulation. This could explain the predilection toward trigger fingers in diabetic patients.

Rheumatoid arthritis.

In contrast to idiopathic trigger finger, triggering in rheumatoid patients is referred to correctly as *tenosynovitis*. Rheumatoid arthritis is a systemic disorder that affects the synovial tissues. The digital flexor tendon sheath is lined with synovium. Inflammation of the tenosynovium causes a mismatch between the size of the contents of the sheath and the enclosing fibro-osseous canal, producing symptoms that may resemble closely those of idiopathic trigger finger. Such symptoms in a patient with rheumatoid arthritis, however, require an entirely different diagnostic and therapeutic approach than that for idiopathic tendovaginitis.

Flexor tenosynovitis in the rheumatoid patient may cause finger pain, swelling, triggering, limited motion, or rupture of the flexor tendon. The diagnosis is characterized by digital triggering or stiffness with palpable swelling on the volar aspect of the digit. Passive range of motion in the finger that exceeds the active range of motion is helpful for distinguishing flexor tenosynovitis from articular pathology. Fixed joint stiffness may develop in chronic cases, however, making the diagnosis of restricted flexor tendon excursion as a consequence of diffuse tenosynovitis more difficult.

The surgical treatment of rheumatoid flexor tenosynovitis is tenosynovectomy and preservation of the annular pulleys, with selected cases requiring USSR or excision of rheumatoid nodules from the tendon. Although the condition may respond temporarily to corticosteroid injection, early surgical intervention in the form of flexor tenosynovectomy with decompression of the carpal tunnel is recommended by many investigators to prevent flexor tendon rupture and irreversible damage to the median nerve.[26]

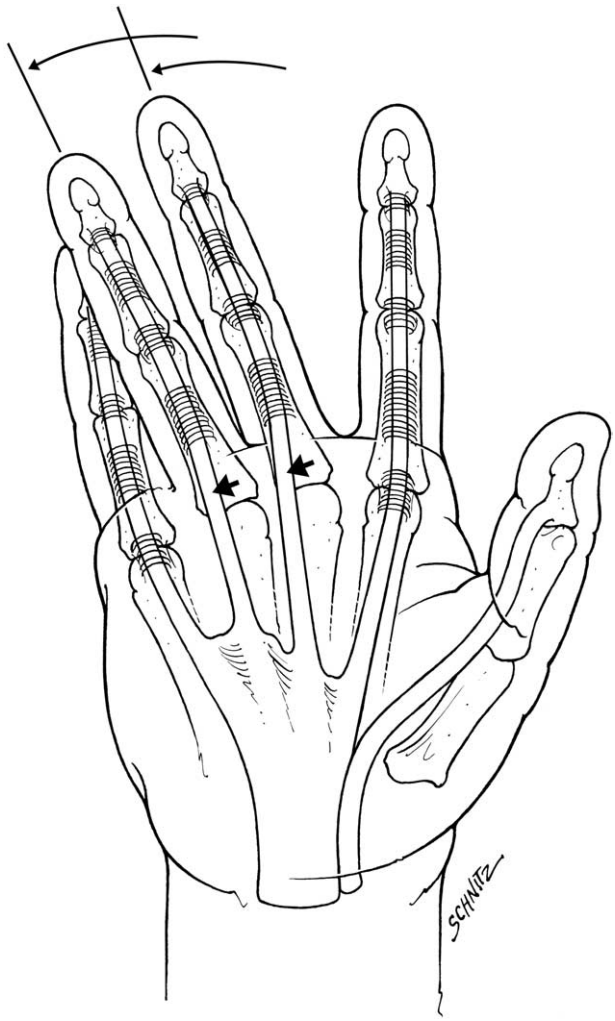

Figure 7. The tendency toward ulnar drift of the flexor tendons after the A-1 pulley of the middle and ring fingers is shown.

An A-1 pulley release in rheumatoid flexor tenosynovitis is not recommended. Despite pulley release, motion still may be limited by rheumatoid nodules or diffuse flexor tenosynovium in the area of one of the more distal pulleys. The division of the A-1 pulley will increase the rheumatoid tendency for digital ulnar drift (Fig. 7), with resultant increase in the ulnar torque across the MCP joint.

Surgical Technique

Standard Bruner incisions are used to approach the digital flexor tendon sheath. The more proximal aspect of the sheath is approached in the palm through transverse incisions in the distal palmar crease. A standard approach to the carpal tunnel is used to expose the flexor tendons at this level if necessary. The tendon sheath is opened proximal to the A-1 pulley and between the A-2 and A-4 pulleys. The A-1, A-2, and A-4 pulleys all are preserved.

Diseased tenosynovium surrounding the tendon is removed. Intratendinous nodules are excised care-

fully. Incomplete excision of nodules may be necessary to prevent late flexor tendon rupture. Tendon excursion then is tested passively. If passive finger flexion is greater than what can be achieved when traction is applied to the proximal aspect of the tendon then further debulking may be required. Ferlic and Clayton[26] recommended excision of the ulnar slip of the superficialis tendon in these patients.

Trigger Fingers in Children

Ninety percent of pediatric trigger digits are trigger thumbs. It is a rare condition affecting less than 0.05% of children. In children trigger thumbs do not trigger but remain locked in a flexed position. Palpation of a Notta's node on the flexor pollicis longus tendon in the area of the thumb A-1 pulley is an important clinical sign differentiating a trigger thumb from other more severe clasped thumb anomalies. Controversy remains concerning the congenital versus acquired nature of pediatric trigger thumbs and the rate of spontaneous resolution. Most patients who present with a trigger thumb are older than 6 months of age.

The reported rate of spontaneous resolution of pediatric trigger thumbs has varied between 0% and 49%. There are multiple conflicting reports in which some documented a marked rate of spontaneous resolution, with other investigators noting that nearly all trigger thumbs persisted. The rate of successful conservative treatment is increased with splinting of the MCP joint in some studies.

Pediatric trigger thumbs respond predictably to a simple A-1 pulley release. A thumb with normal range of motion can be expected immediately after the procedure. McAdams et al[27] investigated the long-term results of pediatric A-1 pulley release at an average of 15 years after surgery. There was no recurrence of triggering. Five of 21 patients had an average of 15° reduced interphalangeal joint motion and 4 of 21 patients showed MCP joint hyperextension; however, no patient complained of functional limitation. The most common concern was scar appearance, which was associated with a longitudinal instead of a transverse incision in the skin crease.

A delay in surgical intervention does not have adverse consequences. Multiple studies have documented good outcomes even in patients for whom surgical release was delayed up to 4 years after the onset of symptoms. Although the existing literature is far from conclusive, a trial of splint therapy is appropriate before performing an A-1 pulley release for pediatric trigger thumb.

Pediatric trigger finger is about one tenth as common as trigger thumb.[28] Some patients present with a fixed flexion deformity. Similar to adult trigger digits, however, snapping and triggering is often the chief complaint. Cardon et al[28] reported a high incidence of flexor tendon abnormalities in these children. In their series of 33 trigger fingers in 18 patients, 8 patients had continued triggering after A-1 pulley release. Documented abnormalities included a more proximal than normal decussation of the FDS, a slip of FDS that inserted into the FDP tendon, nodules in the tendon, and a stenotic A-3 pulley. These patients were treated with USSR, with 2 patients also having an A-3 pulley release. No patient had recurrence of triggering.

Conclusions

A symptomatic trigger digit is a mechanical problem caused by a mismatch between the relative size of the flexor tendon and its sheath. Appropriate treatment is based on understanding the location and nature of the mismatch. Measures to release sites of sheath impingement or to reduce the local volume of the flexor tendon will relieve symptoms. Activity modification, anti-inflammatory medication, splinting, corticosteroid injection, and open and percutaneous A-1 pulley release all have a role in treatment. In select cases an A-3 pulley release, USSR, reduction flexor tenoplasty, and flexor tenosynovectomy are techniques that resolve less common types of trigger finger successfully. A thorough understanding of comorbid conditions and the biomechanics of the finger flexor apparatus will facilitate effective care of the affected patient with prevention of complications.

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