Low Ulnar Nerve Palsy

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Low ulnar nerve palsy causes a complex disability that can be difficult to treat. Over the past 100 years an improved understanding of this disorder has led to the development of effective treatment options. Successful treatment depends on a thorough understanding of normal anatomy, the patient-specific functional deficit, and the individual needs of the patient. A surgeon must tailor the selected tendon transfer(s) to the individual patient and should not apply the same transfers to every patient. A variety of different treatment options are reviewed with a discussion of the strengths and weaknesses of the author's preferred surgical procedures.

Low ulnar nerve palsy causes a complex and multifaceted disability. Treatment depends on a thorough understanding of normal anatomy, the patient-specific functional deficit, and the individual needs of the patient. In the developed world, most ulnar nerve palsies are the result of trauma. Other causes include neuromuscular dysfunction (hereditary sensory-motor neuropathy or poliomyelitis), infection (leprosy), and chronic ulnar nerve compression.

The classic deficit seen in low ulnar nerve palsy is based on the most common innervation pattern of the ulnar nerve. Typically, the palmaris brevis, the hypothenar muscles, the 3rd and 4th lumbricals, all of the interossei, the adductor pollicis, and the deep head of the flexor pollicis brevis are ulnar nerve innervated. The ulnar nerve also provides sensibility to the small finger and ulnar half of the ring finger.

Although an understanding of the classic deficit present with low ulnar nerve palsy is important (and will serve as the basis for the following discussion), variations on the classic deficit are common. A forearm median-ulnar nerve connection (Martin-Gruber) is present in 17% of individuals[1] and the more common hand ulnar-median nerve connection (Riche-Cannieu) has been reported in up to 77% of patients.[2] These named connections and other less well described variations alter the standard muscle innervation patterns, make the deficit in low ulnar nerve palsy heterogeneous, and necessitate a detailed examination to determine the exact deficit for each patient.

Anatomy/Pathologic Anatomy

To better understand the deficiency associated with low ulnar nerve palsy, 5 areas of pathology are explored: pinch, digital motion, grip, clawing, and digital abduction/adduction. A multitude of tests and observations on the physical findings in low ulnar nerve palsy have been described. A compilation is provided in Table 1 with eponyms when appropriate.

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TABLE 1

Signs and Tests for Ulnar Nerve Palsy

Eponym	Description
Froment's sign[7]	Hyperflexed thumb IP with attempted pinch
Jeanne's sign[8]	Thumb MP hyperextension with attempted pinch
Duchenne's sign[12]	MP hyperextension and IP flexion of ring and small fingers
Masse's sign[36]	Flattening of metacarpal arch (inability of the hand to cup water)
Wartenberg's sign[15]	Abducted small finger
Egawa sign[5]	Inability to abduct/adduct fingers with MP flexion
Andre-Thomas sign[37]	Flexion of wrist with attempted IP extension (patients attempt to use tenodesis to increase IP extension)
Pollock sign[5]	Inability to flex distal interphalangeal joint of small finger (typically absent with low ulnar nerve palsy)
Bouvier maneuver[13]	Ability to extend to IP joint of the fingers with the MPs flexed; lost with MPs hyperextended
Earle-Vlasou sign[38]	Inability to cross middle finger over index finger
Mumenthaler's sign[39]	Loss of dimple along ulnar border of hand with abduction of the small finger (related to absent function of palmaris brevis)
Bunnell's sign[11]	Thumb no longer pinches against index to make an "O"
Other	
Confrontational testing[40]	Abductor digiti minimi strength test; patient holds palms in front of face, places small finger ulnar tips together, and pushes; asymmetry is positive test
Index finger abduction	Flex index finger 30° and abduct; palpate 1st dorsal interosseous muscle

Pinch

An elaborate interaction of multiple intrinsic muscles is required for effective pinch. The ulnar nerve–innervated muscles most important to pinch include the adductor pollicis, the deep head of the flexor pollicis brevis (FPB), and the first dorsal interosseus. The transverse fibers of the adductor pollicis lie perpendicular to the thumb and insert onto the base of the proximal phalanx, the metacarpophalangeal joint (MP) capsule, and the ulnar sesamoid. This insertion highlights the functions of the adductor pollicis: thumb adduction, MP flexion, and interphalangeal (IP) extension. The insertion of the FPB into the MP capsule and ulnar sesamoid allows it to reinforce the functions of the adductor pollicis. In contrast to the other 3 dorsal interossei, which partially insert into the dorsal apparatus, the first dorsal interosseus inserts entirely onto the lateral tubercle at the base of the index proximal phalanx. This allows a maximum concentration of abduction force to the index finger.[3] Although it is classically viewed as solely an index finger abductor, the first dorsal interosseus also can adduct the thumb if the index ray is stabilized (based on its origin from both the first and second metacarpals). Together, the adductor pollicis and the first dorsal interosseus provide 75% of thumb adduction power.[4] The extensor pollicis longus (EPL) and the flexor pollicis longus (FPL) are weak thumb adductors[5] that become essential in low ulnar nerve palsy.

An ulnar nerve palsy disrupts the muscle balance necessary for effective pinch, leading to a significant deficit in both pinch strength and precision. Mannerfelt,[5] in clinical studies of normal patients with anesthetized ulnar nerves, found pinch strength to be 17% to 20% of normal. Brown[6] estimated pinch strength to be 10% to 20% of normal. Furthermore, the normal fluid pinch is replaced by an awkward and inefficient pinch maneuver.

In normal pulp-to-pulp pinch (tip pinch), the thumb MP is flexed slightly and the IP joint is extended or only slightly flexed. In ulnar nerve palsy, the joint position is inverted with MP hyperextension and significant IP joint flexion. This alteration can be attributed to a denervation of the adductor pollicis and FPB (and the loss of their MP stabilizing effects), with an increased adductor contribution from the EPL and FPL. The EPL is the more efficient adductor but the thumb IP hyperextension caused by EPL contraction is detrimental to pulp-to-pulp pinch. The FPL contributes to thumb adduction and, more importantly, overpowers the hyperextending force of the EPL. The force of the FPL leads to significant IP flexion with attempted pinch (Froment's sign[7]). Over time, the loss of MP dynamic stabilizers leads to MP

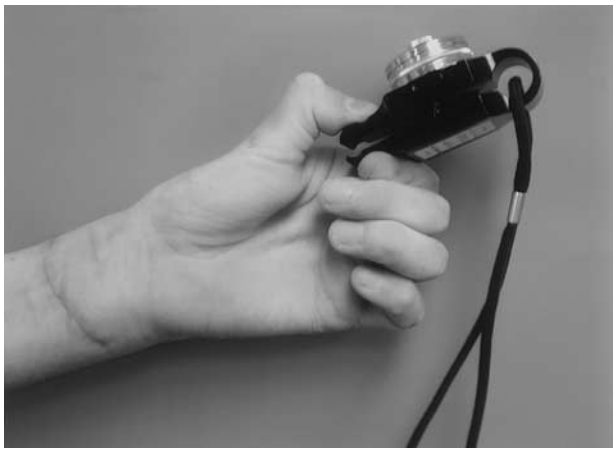

FIGURE 1. Patient with low ulnar nerve palsy showing key pinch. Pathology includes Froment's sign (IP hyperflexion), Jeanne's sign (MP hyperextension), and a lack of stability of the index finger.

volar plate laxity and MP hyperextension with attempted pinch (Jeanne's sign[8]). Although Froment's sign is manifest immediately after the development of ulnar nerve palsy, Jeanne's sign may not develop for an extended period if the volar plate and MP capsule are sufficiently strong to prevent MP hyperextension (Fig 1).

A stable index ray is important for efficient pinch. Normally, the first dorsal interosseus serves to stabilize or abduct the index ray toward the thumb. Without its stabilizing influence and despite the weak abduction force provided by the extensor digitorum communis of the index finger, the index finger gains most of its stability to serve as a post for pinch by bracing against the other digits (Fig 1).[5] The extrinsic flexors of the index finger also will fire in an attempt to increase pinch strength. Typically, the MP and distal interphalangeal joint remain extended and only the proximal interphalangeal (PIP) flexes.[6] Interestingly, this pseudoparalysis of the flexor digitorum profundus tendon (FDP) may further weaken pinch strength. Patients often compensate by further flexing the thumb IP inside the flexion arc of the index to allow index FDP flexion.[9]

Digital Motion

The interossei are the primary flexors of the MP joint. In normal digital flexion, the MP joint will flex first and then the extrinsic digital flexors contribute to near-simultaneous flexion of the MP and interphalangeal joints. The synchrony of flexion allows both large object grasp and small object precision grasp.[4] Independent MP joint flexion allows assumption of the intrinsic plus position (MP joint flexion and IP joint extension), which is necessary for precision pinch activities including buttoning and picking up coins.

With interossei paralysis, the primary MP joint flexor is removed. Patients substitute the extrinsic finger flexors as MP flexors. However, the FDP and flexor digitorum superficialis (FDS) will only flex the MP joint after complete flexion of the IP joints. This causes asynchronous flexion and a significant functional disability.[10] When IP flexion occurs before MP flexion, large object grasp is awkward because large objects are pushed out of the hand by the flexing fingertips. Furthermore, patients are unable to assume the intrinsic plus position and therefore have difficulty with precision pinch. The extended posture of the MP joints and flexed posture of the IP joints is called an *intrinsic minus deformity*.

In low ulnar nerve palsy, the index and long fingers maintain a more normal flexion pattern. The median nerve innervated lumbricals will initiate MP joint flexion and thus maintain synchronous flexion and allow the assumption of the intrinsic plus position. However, even with a relatively normal appearing arc of motion, patients will complain of a functional alteration of these digits.

Grip Strength

Although almost all patients note asynchronous finger motion, the loss of grip strength is often the greater functional deficit and has been estimated to be between 50% and 75%,[4,11] of baseline strength. The exact strength deficit is multifactorial and depends on a variety of patient factors including the specific ulnar nerve innervation pattern. Furthermore, the deficit is related to the nature of the initial traumatic injury. Patients with a wrist laceration and a flexor carpi ulnaris or extensor carpi ulnaris injury will manifest a more profound grip strength deficit.

Clawing

Clawing of the ring and small fingers is the most notable physical finding in low ulnar nerve palsy. The pathologic position of clawing is a flexed posture of the IP joints and a hyperextended posture of the MP joint (Duchenne's sign[12]) (Fig 2A). There are 4 prerequisites for clawing: (1) MP joint hyperextension;

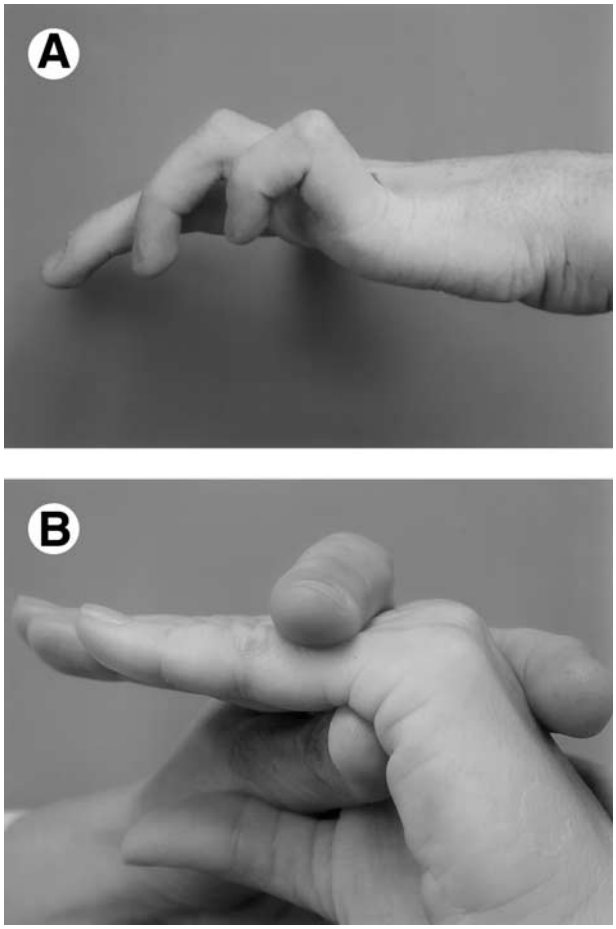

FIGURE 2. (A) Duchenne's sign. Hyperextension of the MP joints and flexion of the IP joints of the ring and small finger. (B) Bouvier maneuver. Prevention of MP hyperextension allows the extrinsic extensors to extend the IP joints.

(2) intrinsic (lumbrical and interosseus) paralysis; (3) intact extrinsic flexor tendons; (4) intact extrinsic extensor tendons.

The loss of MP joint flexion (primarily a function of the interossei) and IP extension (primarily a function of the lumbricals) allows antagonist muscles to overpower the usual dynamic balance and thus cause clawing. Clawing is caused as patients attempt to compensate for the intrinsic deficit in 2 ways. First, patients attempt to use the extrinsic finger flexors to flex the MP joint. Activation of the extrinsic finger flexors, together with the loss of the lumbrical-supplied extensor force to the IP joints, leads to the flexed posture of the IP joints. Second, patients activate the extrinsic extensors in an effort to extend the IP joints. In the normal state the extrinsic extensors work with the lumbricals to extend the IP joints. However, isolated IP joint extension by the extrinsic extensors can only occur if the MP is in a neutral or flexed position. The importance of preventing MP joint hyperextension is shown with Bouvier's maneuver (Fig 2B).[13] This test involves the differential position of the MP joint. If the MP joint is flexed passively, the extrinsic extensors can extend the IP joints. If the MP joint is hyperextended, the extrinsic extensors are rendered ineffective and patients are unable to fully extend their IP joints.

The rigidity of the soft-tissue support will affect the onset of clawing. Patients with laxity of the MP joint volar plate are affected with clawing more rapidly than patients with more rigid soft tissues. All patients will, given sufficient time, develop some degree of clawing. The small finger MP joint often is more ligamentously lax than the ring finger MP and therefore has the greatest tendency to claw. The radial 2 fingers usually are unaffected because the lumbrical muscles to these digits are median nerve innervated. The lumbricals prevent clawing because of their MP flexion and IP extension action. However, strength of MP flexion is lost and, with time and force application, capsular laxity at the MP may lead to a claw deformity in these digits as well.[9,10,14]

Progression of untreated clawing proceeds in a predictable fashion, although the time course depends on a variety of patient-dependent factors including ligamentous laxity. Once a claw deformity develops, the deformity may progress from a passively correctable condition to a fixed deformity. With time, the central slip stretches, the lateral bands translate palmarly, and the volar plate contracts, which results in a fixed deformity. Tendon transfers in the setting of rigid claw hand are contraindicated.

Digital Abduction/Adduction

The dorsal interossei provide digital abduction and the volar interossei allow digital adduction. Although not crucial for general hand function, the loss of the interossei can be both a functional deficit and a source of patient frustration. First, the loss of abduction of the index ray has been mentioned as an important cause of the difficulty with pinch. Second, precision pinch, as noted earlier, requires adducted fingertips and this posture is impossible with ulnar nerve palsy. And third, the loss of the third volar interosseus leads to abduction of the small finger secondary to the unopposed pull of the extensor digiti minimi. The

abducted small finger (Wartenberg's sign[15]) may be a nuisance because it may be caught on objects or make it awkward to place the hand in a pocket.

Loss of sensibility in the small finger and the ulnar half of the ring finger also occurs. This deficit is problematic in that patients are unable to feel with that portion of the hand that commonly rests on flat surfaces. If the nerve injury is more than 7 cm proximal to the wrist crease, the dorsal sensory branch of the ulnar nerve also may be affected.

Patient Evaluation/Initial Intervention

In the developed world, the majority of patients with low ulnar nerve palsy present after a trauma. The mechanism of injury (sharp *v* crush), age of the patient, status of the ulnar artery, and associated bony and soft-tissue injuries all will affect the outcome of the surgically repaired ulnar nerve. A young patient with an isolated, sharp transection of the ulnar nerve in the distal forearm has the best prognosis for recovery.

After initial recovery from the surgical repair of the injured nerve and other structures, a detailed physical examination is mandatory to allow an understanding of the exact functional deficit. Grip and pinch strength, including tip, key, and chuck strength, are assessed in both extremities. Static, single performance evaluations are useful but should be accompanied by an assessment of fatigability. Ligamentous laxity should be assessed from a global standpoint (hyperextensible elbows, and so forth) and an injury-specific standpoint (the volar plate of the MP joint and PIP joint). The presence of MP joint laxity portends a rapid onset of clawing. Active and passive range of motion for all joints is recorded. Finally, finger flexion synchrony and sensibility should be assessed.

Patients who present in a delayed fashion warrant an even more detailed examination of joint mobility and central slip status. With a long-standing untreated ulnar nerve deficit, both the PIP joint and, to a lesser extent, the MP joint may become stiff. Tendon transfers are precluded unless passive mobility can be restored by using hand therapy and dynamic splinting. To prevent this situation, hand therapy is begun immediately after neurorrhaphy. The most important aspect of therapy is the maintenance of a full passive range of motion to all joints. Claw prevention also becomes a central task of the therapist, and, with patient compliance, is accomplished readily. Static and dynamic splinting are both effective in the prevention of MP hyperextension. We most commonly use the lumbrical bar (Fig 3), a hand-based static splint that will prevent MP joint hyperextension and usually is well tolerated by the patient. A dynamic splint, most commonly the Wynn-Parry splint (Fig 4), can prevent MP joint hyperextension and also assist in initiating flexion at the MP joint.

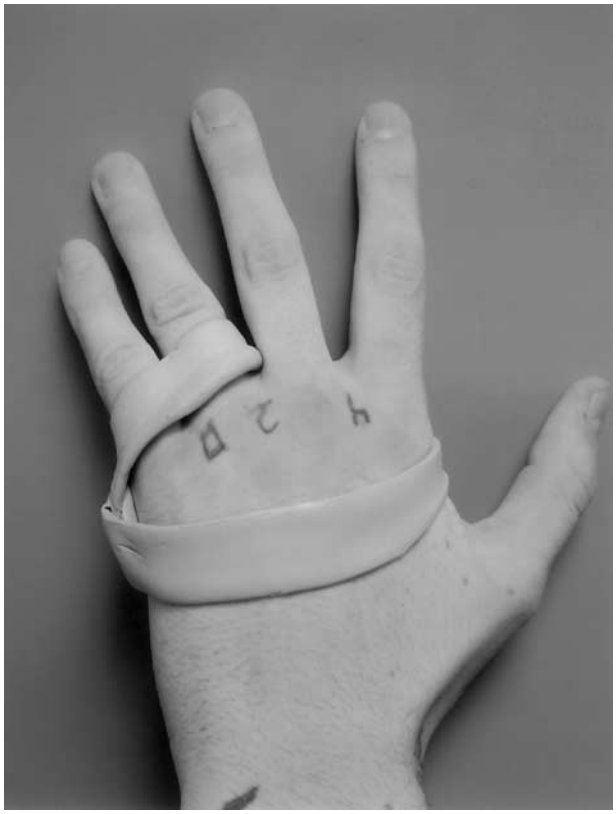

FIGURE 3. Lumbrical bar to prevent MP hyperextension and allow IP extension. This splint is well tolerated by patients and helps to prevent contracture formation.

Although general treatment strategies are appropriate, each patient must be evaluated for what he/she considers to be the most significant deficit. Patient occupation, hobbies, and the aesthetic needs of the patient also should be considered. Interestingly, many patients accommodate well and have few complaints despite obvious functional deficits on physical examination.

Reconstruction

Untreated low ulnar nerve palsy will lead to both deformity and disability.[14] Full correction of the deformity, specifically clawing, can be accomplished

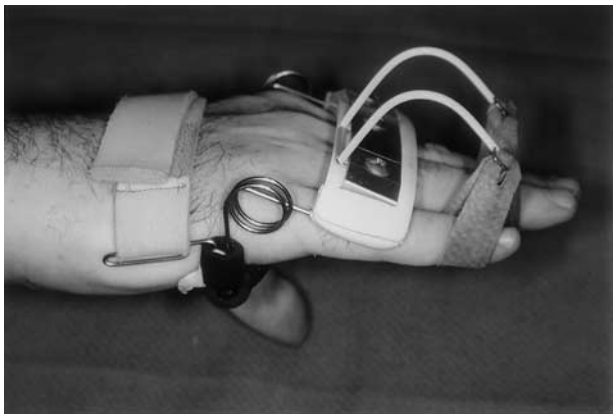

FIGURE 4. Wynn-Parry splint.

by a variety of either static or dynamic procedures outlined later. Complete elimination of clawing is a reasonable goal. The disability in low ulnar nerve palsy, including weak pinch, weak grip, and asynchronous digital flexion, requires dynamic tendon transfer(s). However, complete correction of the disability (as opposed to the deformity) is not possible and patients must be educated as to the likely outcome.

Reconstruction choices are outlined in Table 2. Although many procedures have been described, the exact goals and expectations of each procedure must be considered when choosing the correct surgical intervention for an individual patient. Low ulnar nerve palsy affects all 4 fingers, but the small and ring fingers are most affected because these fingers lose the function of both the interossei and lumbrical muscles. Reconstructive procedures can provide tendon transfers to all 4 fingers or to only the ring and small fingers. Some surgeons feel strongly that all 4 fingers are abnormal and should be addressed whereas others only surgically treat the ring and small fingers. In our experience, it usually is not necessary to treat the index and long fingers because patient function in these digits is satisfactory. The procedures we most commonly perform, with the strengths and weaknesses of each, are outlined later.

Claw Deformity Correction

Procedures to correct clawing of the small and ring fingers fall into 2 main groups: static and dynamic. Static procedures prevent MP joint hyperextension, thereby allowing the extrinsic extensors to extend the IP joints. The feasibility of these procedures should always be tested preoperatively with a Bouvier maneuver. These procedures do not improve the synchrony of finger flexion and do not improve grip strength. MP joint capsulodesis is our static procedure of choice and, in the presence of severe clawing, it can be performed in conjunction with a dynamic procedure. Capsulodesis also is indicated for the treatment of combined nerve injuries when there are no suitable motors available for transfer. We rarely use it as an isolated procedure for low ulnar nerve palsy.

The Zancolli metacarpophalangeal capsulodesis prevents MP joint hyperextension by tightening the volar plate.[16] It can be performed in isolation or combined with other procedures such as the lasso procedure (see later) or pulley advancement. The capsulodesis is performed through a transverse incision in the distal palmar crease. The A1 pulley is divided and the flexor tendons are retracted to expose the volar plate. The volar plate is elevated off of its metacarpal origin and split longitudinally. The volar plate is advanced proximally and is secured with suture anchors, a pullout button over the dorsum of the hand,

TABLE 2

Procedures for Ulnar Nerve Palsy

- Static procedures to correct clawing
  - Capsuloplasty[16,17]
  - Dorsal bone block[41,42]
  - Capsulodesis with pulley advancement[43]
  - Tenodesis[44]
- Dynamic procedures to correct clawing, provide MP flexion, increase grip strength (from weakest to strongest)[45]
  - EIP to lateral bands[46]
  - Zancolli lasso[16]
  - Modified Stiles-Bunnell[47]
  - ECRB to lateral bands[28]
  - Flexor carpi radialis[24]
- Procedures to restore power pinch
  - Replacement of transverse head of adductor pollicis
    - ECRB adductorplasty[30]
    - Extensor indicis proprius (EIP) adductorplasty[6,27]
    - Extensor digitorum communis adductorplasty[47]
    - Brachioradialis[48]
    - Extensor carpi radialis longus[48]
    - FDS adductorplasty[32,33,49]
  - Split transfers to adductor and 1st dorsal interosseous
    - Split EIP[50]
    - Split extensor digiti quinti[51]
  - Pure Index Abductors
    - APL[34]
    - EIP[52]
  - Other
    - MP arthrodesis[53]
    - IP arthrodesis

or suture repair through a bone tunnel. The volar plate is tightened so that at least a 20° to 30° flexion contracture results. This technique of bony capsulodesis is more resistant to recurrence than the original soft-tissue capsulodesis (or volar plate placation),[17] but it does not provide any correction of flexor strength, dexterity, or synchrony.[16]

Brown[18] evaluated the results of capsuloplasty in 44 hands with moderate claw deformity. The surgical technique differed from that of Zancolli[16] in that the volar plate was repaired with the MP in 60° of flexion and, in some patients, a dermodesis was added. Twenty-one patients had improvement or correction of the clawing. These patients had more efficient grasp and often an increase in grip strength by objective (30% to 50% in 11 hands) or subjective evaluation. These mixed results have led to our hesitancy to use this procedure for isolated ulnar nerve palsy.

Claw Deformity and Intrinsic Minus Deformity Correction

Multiple dynamic procedures have been described for low ulnar nerve palsy and most can be expected to correct clawing successfully and improve the pattern of digital flexion. Dynamic tendon transfers for low ulnar nerve palsy may be divided into 2 groups: those that provide MP flexion alone and those that provide MP flexion and IP extension. The function of the tendon transfer is determined largely by its route of passage and its insertion site. In our practice, 2 tendon transfers (1 of each variety) are used most commonly: the Zancolli lasso procedure and the modified Stiles-Bunnell transfer.

We prefer the modified Stiles-Bunnell transfer or the Zancolli lasso procedure for several reasons. Both procedures are volar transfers, are technically straightforward, are not overly time consuming, and do not require tendon grafting. Both are time-tested procedures that have been modified over time to increase their success and decrease their complication rates.

The Zancolli lasso procedure was first described in 1974. A transverse incision is made in the distal palmar crease over the ring and small fingers. The A1 pulley is exposed and carefully protected. Subcutaneous dissection exposes the proximal portion of the A2 pulley. The FDS tendon of the affected finger is divided approximately 2 cm distal to its separation into 2 tendons. After division, the tendon is passed anteriorly between the A1 and A2 pulleys, directed proximally (anterior to the A1 pulley), and sewn to itself (forming a loop) (Fig 5). To adjust tension, maximal pull is placed on the FDS with the wrist and fingers in neutral position. If tension is appropriate, the MP will assume a partially flexed position and IP joints will extend secondary to the pull of the extrinsic extensors.

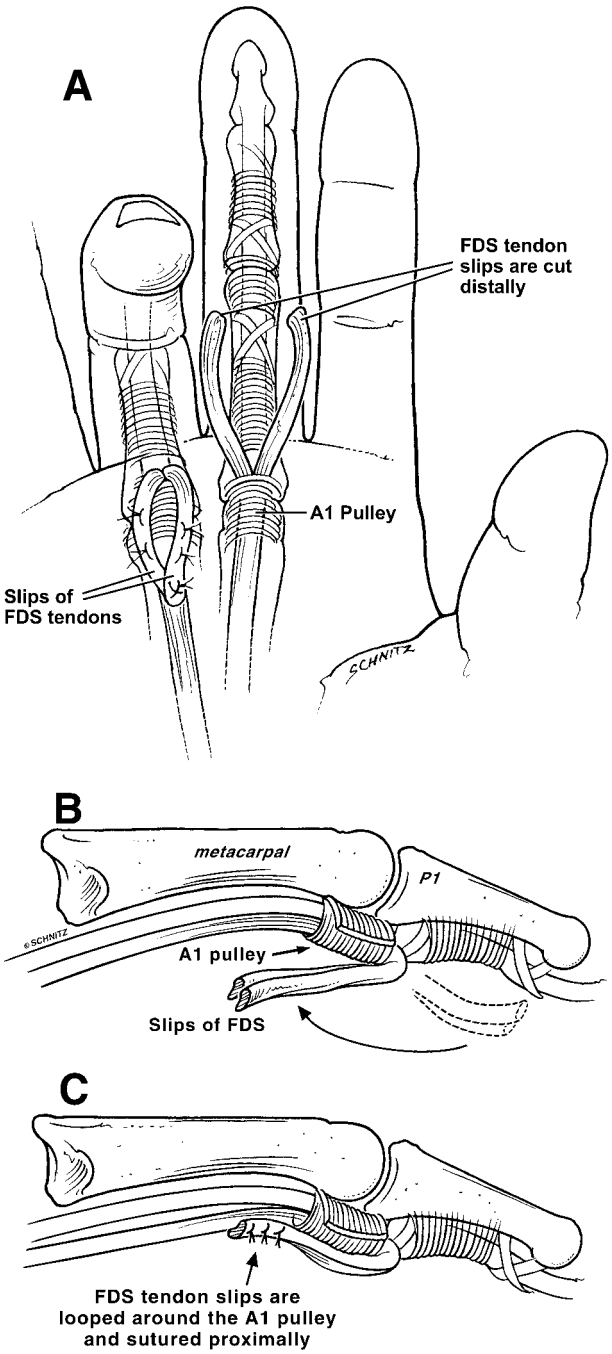

FIGURE 5. Zancolli lasso procedure.

If the tension is not sufficient, the lasso can be completed with the MP in 20° to 30° of flexion. The lasso procedure corrects the claw deformity, allows simultaneous flexion of all joints of the fingers, and allows the fingers to assume the intrinsic plus position for precision pinch.[16] The drawbacks of this procedure are that it does not significantly increase grip strength and, by removing the FDS support of the PIP, it can lead to a swan-neck deformity.

Hastings and McCollam[19] examined the results of the FDS lasso procedure in 23 digits at an average of 5 years after surgery. In 19 of 23 digits the preoperative clawing was corrected. Two of the 4 failures had PIP flexion contractures before surgery. Grip strength at 40-month follow-up evaluation increased 13%, but the investigators felt this most likely was related to muscle conditioning. Patients typically were happy with the procedure and noted a decreased feeling of clumsiness. Four patients had a postoperative swan-neck deformity.

FDS transfer has undergone several modifications since originally described by Stiles and Forrester-Brown[20] in 1922, and the modified Stiles-Bunnell transfer[21] is performed commonly today. We usually use the FDS of the middle finger as a motor when clawing is mild to moderate. Rarely, if a severe deformity is present, we will use the FDS from the finger that is clawing. The flexor digitorum longus tendon of the long finger is divided approximately 2 cm distal to the split in the superficialis tendon. The 2 slips are then divided longitudinally for a distance of approximately 5 cm. Each slip provides motor power to either the ring or small fingers. Each slip is redirected through the radial lumbrical canal volar to the deep transverse intermetacarpal ligament. It is then passed dorsally and inserted into the transverse fibers of the radial lateral band of the ring and small fingers. The transfer is tensioned with the wrist in neutral position and the ring and small fingers in the intrinsic plus position (90° of MP flexion). The surgeon should always err on the side of overtensioning this transfer for the best outcome. If appropriate tension has been applied, passive wrist flexion will extend the MP joints fully.

The procedure is simple to perform, leaves minimal scarring, improves MP flexion and IP extension, and improves digital coordination. There are 3 disadvantages:

1. PIP hyperextension: PIP joint hyperextension (swan-neck deformity) can occur with any procedure that harvests the FDS tendon. (a) Donor digit harvesting the FDS tendon too far distally (ie, at Camper's chiasm): by harvesting the tendon more proximally, enough of a tail is left to allow scarring and a tenodesis effect to prevent PIP joint hyperextension. (b) Recipient digit: any tendon transfer sutured too tightly into the lateral band can lead to a PIP joint hyperextension deformity. Volar plate laxity increases the chances of this complication and warrants consideration of alternative procedures. Correction of PIP joint hyperextension requires the division of the oblique fibers of the dorsal apparatus distal to the insertion of the transfer into the lateral band.
2. PIP flexion contracture: this complication is difficult to prevent but it usually is tolerated better than PIP joint hyperextension. It most likely is related to scarring from the FDS harvest.
3. Grip strength: FDS transfer generally does not significantly improve grip strength and may even lead to decreased grip strength. We prefer the long finger FDS donor to prevent further weakening of the ring and small fingers (inevitable if the FDS is harvested in these fingers).

A change in the insertion site of the transfer will alter its function (Fig 6). Certain insertion points are designed to only flex the MP joint. Burkhalter and Strait[23] recommended a bony insertion into the proximal phalanx through a drill hole. A bony attachment will decrease the swan-neck deformity by avoiding the additional extensile force at the PIP. This insertion is more time consuming because it requires greater surgical dissection, but allows easier tensioning and can be overtensioned effectively without fears of complications at the PIP. If FDS transfer is attached directly to the A2 pulley, it will also serve as an MP flexor.[24] In cases in which there has been an attenuation of the central slip with a long-standing deformity, the transfer may be sutured to the remnant central slip. This theoretically maximizes the extensor power at the PIP,[22] but it also carries the greatest risk for PIP hyperextension.

Grip Strength Correction

Although some proponents of the lasso procedure and the modified Stiles-Bunnell transfer suggest that

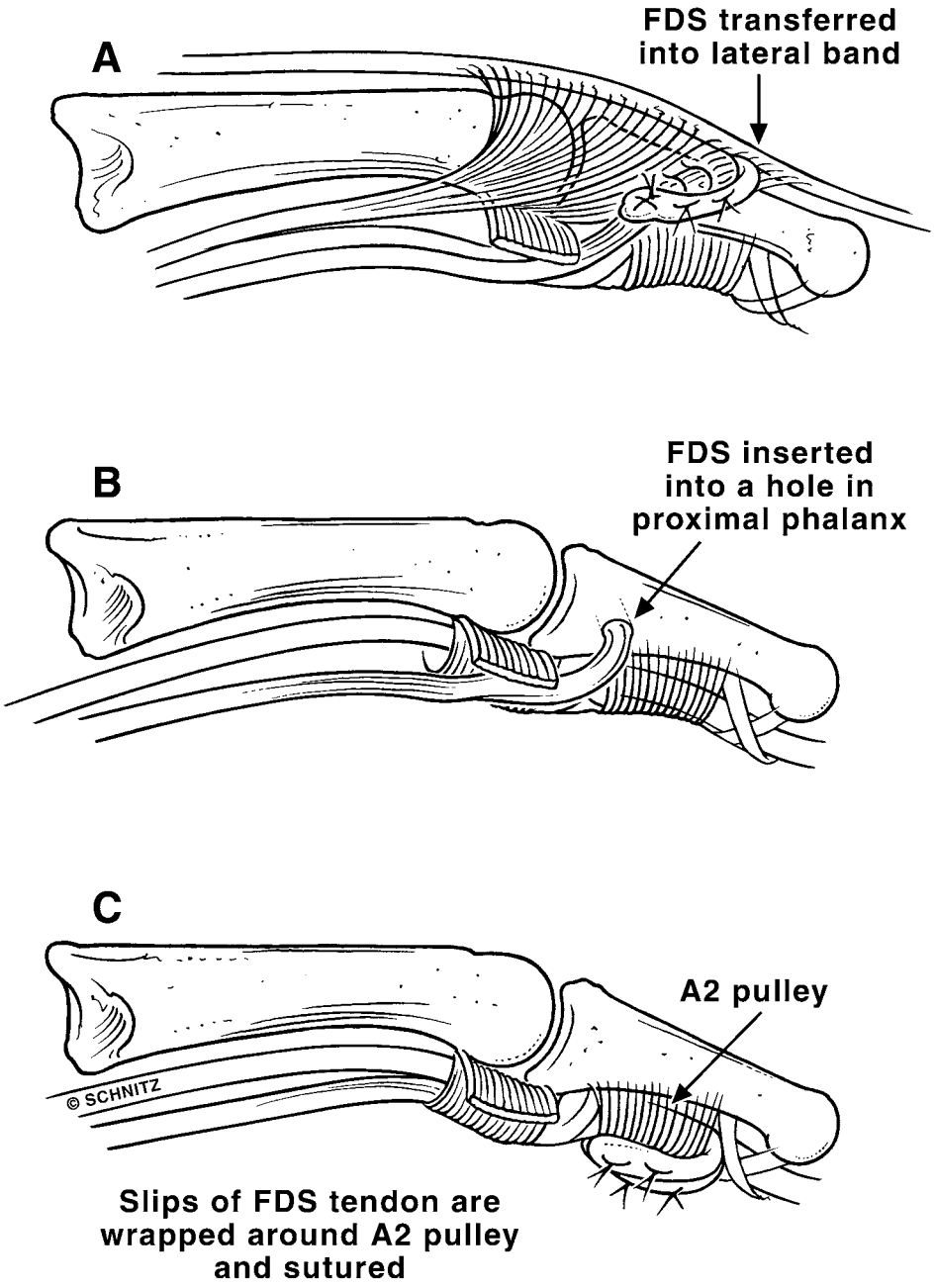

FIGURE 6. Modified Stiles-Bunnell tendon transfer insertion options. The lateral band insertion and the bone tunnel insertion are shown.

these procedures increase grip strength, most studies suggest that grip strength is affected minimally and may even be decreased.[25] Burkhalter[26] advocated bony insertion in the proximal phalanx of the FDS transfer to maximize MP flexion power with an increased muscle tension. Although conceptually sound, only one third of his patients with low ulnar nerve palsy noted any increase in grip strength.

To increase grip strength, most investigators advocate the use of wrist extensors or flexors to provide additional muscle power. Furthermore, in patients in whom grip strength is a major concern, the transfer may be used for all 4 digits[22,27] because all are affected even if not all show clawing. However, these procedures have disadvantages. The motor for these transfers is dorsal and requires a dorsal to volar route of transfer. This may lead to scarring in the intermetacarpal space and the possibility of the transfer acting only as a tenodesis. Each of these transfers requires prolongation with a tendon graft, further increasing

the risk for scarring. To our knowledge, there is no objective data in the literature confirming the increase in grip strength with these transfers. The choice of muscle for transfer can be difficult. The extensor carpi radialis brevis tendon (ECRB) is ideal, but it is our first choice for restoration of power pinch and, therefore, may be unavailable. Some investigators recommend the flexor carpi radialis tendon for transfer.

The Brand transfer uses the ECRB to augment grip strength through a 4-tailed graft.[28] An oblique incision is made over the radial wrist to harvest the ECRB. A transverse incision is made between the second and third metacarpals and fourth and fifth metacarpals. The plantaris tendon(s), palmaris longus tendon, or toe extensor tendons are harvested as extension grafts and divided into 4 tails. Two tails are passed between the second and third metacarpals, 1 between the third and fourth metacarpal, and 1 between the fourth and fifth metacarpal. After passing volar to the deep transverse intermetacarpal ligament, the grafts are inserted into the radial lateral bands of the middle, ring, and small fingers, and the ulnar lateral band of the index finger. Attachment to the radial side of the index is avoided because it can lead to a bothersome, abducted posture of the index finger. Once an unimpeded passage is confirmed, the grafts are sutured into the ECRB with the wrist maximally dorsiflexed and the fingers in the intrinsic plus position. Wrist flexion should tenodese the MPs into extension.

Abducted Small Finger Correction

Persistent abduction of the small finger (Wartenberg's sign) may be one of the more bothersome features of low ulnar nerve palsy. The therapist may correct this deformity with buddy taping and most of the other procedures mentioned also will decrease the abduction tendency of the small finger. Occasionally, this deformity is treated in isolation. The simplest technique transfers the ulnar tendon of the extensor digiti minimi to the radial side of the small finger into either the radial collateral ligament or the A2 pulley.[29] Both insertion sites help to decrease small finger abduction and the A2 pulley insertion also will decrease clawing.

Pinch Correction

Although patients with low ulnar nerve palsy often complain of pinch weakness, many do not note a significant disability. For those who are disabled by the loss of pinch strength, multiple procedures with a variety of different vectors and pulleys have been described to restore thumb adduction. These procedures usually are combined with a tendon transfer to the first dorsal interosseus. Our preferred treatment is the ECRB adductorplasty as described by Smith (Fig 7).[30] This transfer is effective in increasing pinch strength because it follows the course of the transverse head of the adductor pollicis. However, this transfer provides no additional support to the MP joint and therefore no additional MP joint stability is provided.

A dorsal, transverse incision is made over the proximal aspect of the third metacarpal to expose the insertion of the ECRB. The ECRB is detached from its third metacarpal insertion and tunneled proximally to a transverse incision just proximal to the extensor retinaculum. A third dorsal transverse incision is made between the second and third metacarpals. This incision allows the creation of a passageway in the intermetacarpal fascia for the transfer. A curvilinear incision is made along the ulnar aspect of the thumb MP joint to expose the adductor pollicis insertion. The palmaris longus is harvested and attached to the adductor tendon. A curved tendon passer is placed from the second/third metacarpal interspace deep to the adductor pollicis to route the palmaris longus graft dorsally. By using a tendon weave, the palmaris graft is sutured to the ECRB proximal to the extensor retinaculum with the wrist in neutral and the thumb slightly volar to the index finger. Wrist extension should cause thumb abduction and wrist flexion should cause the thumb to adduct firmly against the palm. A doubling of the pinch strength is anticipated.[25,30]

Several investigators recommend thumb MP fusion for stability in combination with adductorplasty.[22,25] Omer[31] recommends ECRB transfer for restoration of key pinch, but abductor pollicis longus tendon (APL) to first dorsal interosseus transfer with thumb MP fusion for restoration of tip pinch. In our hands, ECRB adductorplasty alone has provided excellent results.

If the ECRB is used to maximize grip strength with the Brand transfer, the FDS of the long finger can be used as an adductorplasty. The long or ring finger FDS is divided between the A1 and A2 pulleys and is retracted into the palm. It is passed volar to the

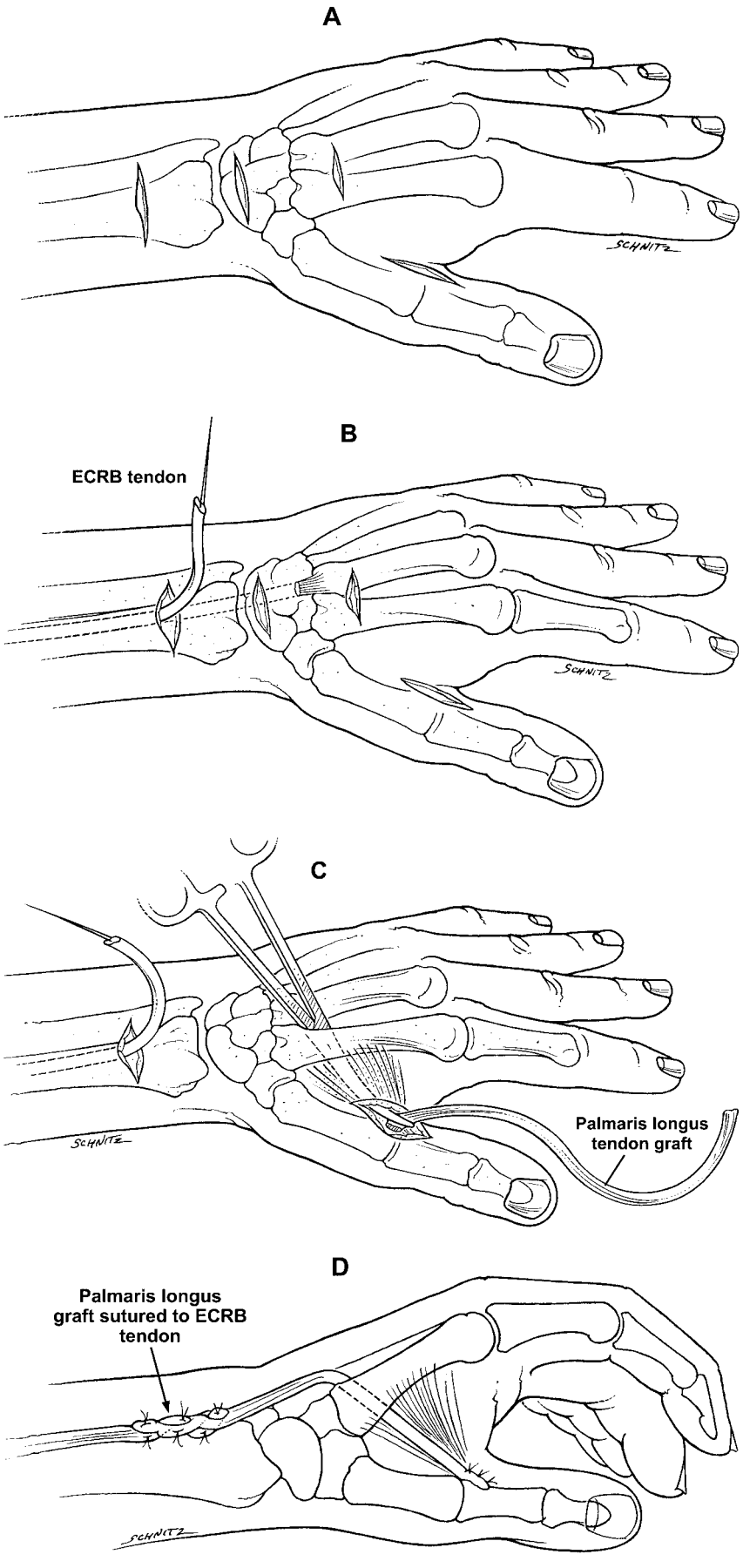

FIGURE 7. ECRB adductorplasty technique.

adductor pollicis muscle and dorsal to the neurovascular structures and the other tendons of the hand to insert on the tendon of the adductor pollicis. This transfer uses the distal edge of the vertical septum of the palmar fascia as a pulley[32] and has been reported to increase pinch from 30% of normal to more than 70% of normal.[33] The disadvantages of this transfer include the harvest of the FDS with its associated complications and the questionable ability of this transfer to reproduce the natural pull of the adductor pollicis. The pulley to best redirect this tendon for the most effective line of pull is a source of controversy.

The second component necessary to increase pinch strength is the supplementation of the first dorsal interosseus muscle. Our preferred method is the APL transfer.[34] In this simple technique, the APL tendons are identified through a transverse incision at the radial styloid. There is more than one insertion of the APL in 86% of patients[35] and the most important insertion, to the thumb metacarpal, is preserved. The tendon of the first dorsal interosseus muscle is exposed through a separate longitudinal incision on the radial aspect of the index MP. A tendon graft is used to prolong the APL and the tension is adjusted so that the wrist and index finger are in neutral position. Although this transfer may increase abduction power of the index finger, its more important function is to stabilize the index ray to increase pinch strength.

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