NORMAL STRUCTURE AND FUNCTION

The versatility and power of the human hand are largely dependent on the smoothly balanced function of the intrinsic muscles of the fingers and the long flexors and extensors. Phylogenetically the intrinsic muscles were the earliest of the digital motors to appear, and their precursors are found in the pectoral fins of fish. In the evolution of the upper limb many of the layers of intrinsic muscles merged with the long flexors until both the function and the structure of the intrinsic and extrinsic muscles became integrated in the human hand.

The four dorsal interossei are the abductors of the index, middle, and ring fingers. The anatomic axis of the hand coincides with the axis of the third metacarpal. Thus the first dorsal interosseous lies to the radial side of the index finger, the second to the radial side of the middle finger, the third to the ulnar side of the middle finger, and the fourth to the ulnar side of the ring finger. The middle finger, which at rest lies within the axis of the third metacarpal, therefore has two abductors.

Each of the dorsal interossei with the exception of the third has two separate muscle bellies. The more superficial belly, which arises from adjacent surfaces of the midshafts of contiguous metacarpals, inserts through the medial tendon onto the lateral tubercle at the base of the proximal phalanx. This muscle belly functions primarily as an abductor of the proximal phalanx and is also a weak flexor of the proximal phalanx. The superficial belly has no direct effect on interphalangeal joint extension. The third dorsal interosseus has no superficial belly, does not insert into the proximal phalanx, and therefore has no medial tendon.

Each of the deep bellies of the dorsal interosseus continues as the lateral tendon to form the lateral band of the dorsal aponeurosis ("dorsal apparatus") of the radial three fingers. Through the dorsal aponeurosis the lateral band flexes and abducts the proximal phalanges and extends the distal two phalanges of these fingers. The first dorsal interosseus inserts almost exclusively onto the lateral tubercle and therefore contributes little to the radial lateral band of the index finger. Thus the dorsal interosseus of the index finger has a small and inconstant deep belly. The third dorsal interosseous, which has no insertion onto the proximal phalanx, is almost entirely a deep belly and all its fibers are inserted onto the lateral band.

The dorsal interossei are usually all supplied by the ulnar nerve. Each of these muscles and their deep and superficial bellies are separately innervated by distinct neural fasciculi. It is therefore possible to contract the deep belly of a dorsal interosseus without contracting the superficial belly or vice versa. Infrequently the median nerve may supply the first dorsal interosseus. This variation may be associated with a median-to-ulnar nerve crossover in the forearm (Martin-Gruber anastomosis) or in the palm (Riche-Cannieu anastomosis). Rarely the dorsal interosseus may be innervated by the radial nerve or, more infrequently, there may be intercommunication between the musculocutaneous and median nerves.

The three volar interossei are more uniform in
structure and in function than are the dorsal interossei. Arising from adjacent sides of the metacarpal shafts, they insert onto the lateral band at the adductor side of the index, ring, and little fingers: the first volar interosseous to the ulnar side of the index finger, the second to the radial side of the ring finger, and the third to the radial side of the little finger. These muscles have no distinct deep and superficial bellies and none are inserted onto the proximal phalanx. Each volar interosseous can adduct and flex the proximal phalanx and can extend the distal two phalanges of the finger. Usually the volar interossei are innervated by the ulnar nerve.

The adductor digitii quinti arises from the pisiform bone, the pisohamate ligament, and the volar carpal ligament. It inserts onto the lateral tubercle at the base of the proximal phalanx and into the ulnar lateral band of the little finger. The flexor digiti quinti proprius lies deep to the abductor digitii quinti. It inserts volar to the abductor tendon onto the anterior part of the ulnar lateral tubercle at the base of the proximal phalanx of the little finger.65 The opponens digiti quinti is a small and deep muscle belly arising from the pisohamate ligament and the hook of the hamate. It inserts along the ulnar shaft of the fifth metacarpal, which it flexes and supinates.

The lumbricals arise from the tendons of the flexor digitorum profundus, pass volar to the deep transverse metacarpal ligament (the intercapitular ligament that interconnects the volar plates), and insert onto the radial lateral band of each finger, approaching the finger at an angle of about 40 degrees. The first and second lumbricals originate from the flexor profundus tendon of the index and middle fingers and are supplied by the median nerve. They are the only intrinsic muscles of the fingers with median nerve supply. The third lumbral arises from the profundus tendon of the middle and ring fingers and the fourth from the profundus tendon of the ring and little fingers. Both are supplied by the ulnar nerve.

The lumbral muscle is the "workhorse of the extensor apparatus."64,66 Electromyography of the lumbricals reveals high levels of activity whenever there is active extension of the interphalangeal joints. Strong electrical stimulation of the lumbrical produces interphalangeal joint extension followed by metacarpophalangeal joint flexion. Low levels of electrical stimulation produce only interphalangeal joint extension. There is no radial deviation of the finger when the lumbral contracts.65

Because the lumbral arises from the flexor digitorum profundus tendon, it is the only muscle that is able to relax the tendon of its own antagonist. When considering lumbral action it is best not to consider its origin and insertion but rather its two attachments—to the profundus tendon and to the lateral band. Thus, if the profundus contracts and the lumbral relaxes, the interphalangeal joints of the fingers will flex. If the profundus is relaxed, contraction of the lumbral will pull the lateral band proximally and the profundus tendon distally (Fig. 12-1). The viscoelastic force provided by the profundus tendon within the finger will be lessened and the interphalangeal joints will fully extend.58 When both the profundus and the lumbral contract the interphalangeal joints and metacarpophalangeal joint will flex simultaneously.24,35,44

Flexion of the proximal phalanx is achieved chiefly by the interossei. Electromyography has indicated that under normal circumstances the lumbral contributes little to metacarpophalangeal joint flexion.44 When the interossei are paralyzed, however, the lumbral can initiate flexion at this joint. Flexion of the proximal phalanx may also be achieved through contraction of the flexor digitorum superficialis and the flexor digitorum pro-
Fig. 12-2. Diagrammatic representation of dorsal apparatus of finger. A, Radial side of left middle finger. B, Dorsum of left middle finger. A, Extensor digitorum communis tendon; B, sagittal bands; C, transverse fibers of intrinsic muscle apparatus; D, oblique fibers of intrinsic apparatus; E, conjoined lateral band; F, terminal tendon; G, flexor digitorum profundus tendon; H, second dorsal interosseous muscle; I, lumbrical muscle; J, flexor digitorum superficialis tendon; K, medial tendon of superficial belly of interosseus; L, lateral tendon of deep belly of interosseus; M, flexor pulley mechanism; N, oblique retinacular ligament; O, transverse retinacular ligament; P, medial band of oblique fibers of intrinsic expansion; Q, central slip; R, lateral slips; S, triangular ligament. (From Smith, R. J.: Clin. Orthop. 104:95, 1974.)
fundus. When these muscles contract, they first flex the interphalangeal joints. After full interphalangeal joint flexion is achieved, the long flexors will flex the metacarpophalangeal joint until the finger is completely closed. If finger flexion were solely performed by the flexor digitorum profundus and flexor digitorum superficialis, metacarpophalangeal joint flexion would occur only after interphalangeal joint flexion was complete.\(^3\)\(^,\)\(^5\)\(^,\)\(^13\)

The *dorsal aponeurosis* (dorsal apparatus) of the finger consists of an intimate weave of the tendons and the long extensors with the aponueroitic expansion of the intrinsic muscles (Fig. 12-2). The long extensors of the fingers include the extensor digitorum communis, the extensor indicis proprius, and the extensor digiti quinti proprius. The extensor indicis proprius and extensor digiti quinti proprius each lie to the ulnar side of their companion common extensors. The extensors of the ulnar three fingers are interconnected by the *juncturae tendinum* just proximal to the metacarpophalangeal joints. The extensors of the index finger rarely have juncturae and the finger is therefore the most independent; it can extend fully even when the adjacent fingers are flexed.

At the dorsum of the metacarpophalangeal joint, short, deep articular fibers often insert onto the dorsal joint capsule or occasionally, onto the base of the proximal phalanx.\(^34\) There is some controversy about the importance of these fibers.\(^24\)\(^,\)\(^34\) They may help to hold the extensor tendons to the dorsal midline of the joint. They may reinforce the normally thin dorsal capsule of the joint. Under normal circumstances, however, these fibers do *not* act to extend the proximal phalanx.

The proximal phalanx is lifted into extension by the *sagittal bands*. The sagittal bands represent a continuation of the dorsal intertendinous fascia that becomes more firm at the level of the metacarpal head. The bands envelop the long extensor tendons for about 1.5 to 2.0 cm. and then dive volarily. They pass on either side of the metacarpophalangeal joint capsule to insert at the lateral edges of the volar plate and onto the volar periosteum of the proximal phalanx (Fig. 12-3). With contraction of the extensor muscle, the extensor tendon lifts the proximal phalanx through the sagittal band sling. If the metacarpophalangeal joint hyperextends, the direction of the sagittal bands becomes more parallel to the metacarpal shaft and the extensor tendon becomes “blocked.” The obliquity of the sagittal bands then tethers the long extensors to the volar plate and limits its effect on more distal joints (Fig. 12-4).

Distal to the metacarpophalangeal joint the long

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**Fig. 12-3.** Diagrammatic representation of cross section through head of third right metacarpal. 1, Extensor digitorum communis tendon; 2, sagittal band; 3, collateral ligament; 4, tendons of interosseous muscles (medial tendon deep to sagittal band, lateral tendon superficial to sagittal band); 5, accessory collateral ligament; 6, volar plate; 7, deep transverse metacarpal ligament; 8, metacarpal head; 9, articular fibers of extensor tendon. Lumbrical tendon does not appear on this diagram; it would appear volar to deep transverse metacarpal ligament.

**Fig. 12-4.** When metacarpophalangeal joint is hyperextended, sagittal bands run progressively more transversely, finally “blocking” extensor tendon. When blocked at metacarpophalangeal joint, extensor tendon has less control on interphalangeal joints, which then tend to fall into flexion.
extensor tendon divides into three parts: one central slip and two lateral slips. The central slip continues distally to insert onto the central tubercle at the base of the middle phalanx. Through this slip the long extensor tendons extend the proximal interphalangeal joint (and indirectly the metacarpophalangeal joint when the proximal interphalangeal joint is held in a flexed position). The two lateral slips diverge to join the lateral bands of the intrinsic muscles at the distal third of the proximal phalanx, where they form the conjoined lateral band, which will be described later.

The superficial belly of the first, second, and fourth dorsal interosseous insert onto the lateral tubercle of the base of the proximal phalanx through the medial tendon. All the interosseous (with the occasional exception of the first dorsal) are inserted onto the lateral tendon, which passes distally and dorsally. The sagittal band of the extensor tendon runs between the medial tendon, which is deep to it, and the lateral tendon, which is more superficial. At the level of the proximal flexion crease of the finger the radial lateral band is joined by the tendon of the lumbrical. In this region the radial and ulnar lateral tendons become the lateral bands as they join the dorsal apparatus.

Transverse fibers arch dorsally from the lateral bands to surround the trifurcating extensor tendons at the middle third of the proximal phalanx. The transverse fibers are distal to the sagittal bands. Although they pass in a similar direction to the sagittal bands, the transverse fibers flex the metacarpophalangeal joints when the interossei (and lumbrical?) contract. They are part of the intrinsic muscle apparatus of the finger. The sagittal bands extend the metacarpophalangeal joints when the extensor muscles contract. They are part of the extrinsic muscle apparatus of the fingers (Fig. 12-5).

At the distal third of the proximal phalanx, the dorsal aponeurosis of the lateral band arches more obliquely to form the oblique fibers, which insert onto the dorsolateral tubercles of the middle phalanx through the medial bands. The oblique fibers of the intrinsic mechanism assist the central slip to extend the middle phalanx.

At the dorsolateral side of the distal condyle of the middle phalanx the lateral band of the intrinsics is joined by the lateral slip of the long extensor tendon to form the conjoined lateral band. The conjoined lateral bands merge at the distal third of the middle phalanx and form the terminal tendon. The terminal tendon inserts at the base of the distal phalanx and extends it.

Several features of this dorsal apparatus deserve particular note.

1. The woven fibers of any part of the apparatus are intricately bound to one another and have lost the specificity of their parent muscle fiber. For example, the fibers of the conjoined lateral band that arise from the common extensor tendon cannot be functionally or anatomically distinguished from the interosseous muscle or the lumbrical.

2. With the exception of the deep articular fibers from the long extensor tendons to the metacarpophalangeal joint, no tendon fibers have two bony insertions. A separate and distinct fascicle of tendon fibers will proceed to a more distal attachment wherever a portion of the dorsal apparatus has a bony insertion.

3. A portion of the dorsal apparatus is primarily
flexor (the transverse fibers of the lateral bands). To call it the extensor apparatus is thus a misnomer.

The oblique fibers of the intrinsic tendons and the central slip of the long extensor tendons extend the middle phalanx. The conjoined lateral band of the intrinsics and the long extensors join to extend the distal phalanx. Thus the motion of the two joints is interrelated. Often we may wish to flex one joint without fully flexing the other, as in threading a needle, writing, and activities requiring pulp-topulp pinch. Some independence of tendon excurs-

ion at the interphalangeal joints is therefore essential.

Independence is achieved by a volar shift of the conjoined lateral band at the dorsolateral sides of

the base of the middle phalanx. When both inter-
phalangeal joints flex, the conjoined lateral band shifts several millimeters volarly. When the proximal interphalangeal joint and the distal inter-
phalangeal joint extend, the conjoined bands shift dor-
sally. Since the diameter of the distal interphalangeal joint is smaller than that of the proximal interphalangeal joint, this shift is essential for normal finger function. If the excursion of the dorsal aponeurosis at the distal interphalangeal joint were not regulated by the volar-dorsal tendon shift at the base of the middle phalanx, the arc of distal interphalangeal joint extension would exceed proximal interphalangeal joint extension by the ratio of their joint diameters (Fig. 12-6). The distal phalanx would therefore hyperextend when the proximal interphalangeal joint extended fully.

Three sets of ligaments contribute to control of the position of the dorsal aponeurosis about the finger. The oblique retinacular ligament (or link ligament) is a fibrous cord that extends to the terminal tendon from the flexor pulley adjacent to the proximal phalanx. It lies volar to the axis of motion of the proximal interphalangeal joint and thus links proximal interphalangeal joint extension with distal interphalangeal joint extension. When the proximal interphalangeal joint extends, the oblique retinacular ligament tightens and pulls the terminal tendon proximally; the distal phalanx then extends and its passive flexion is limited.

The transverse retinacular ligament passes from the lateral border of the conjoined lateral band to the fibrous flexor sheath at the level of the proximal interphalangeal joint. This ligament prevents dorsal dislocation and "bowstringing" of the con-

joined lateral bands when the proximal interphalangeal joints are extended.

The triangular ligament is a fascial layer at the dorsum of the middle phalanx. The base of the triangle is the central slip attachment to the middle phalanx. Its sides are the conjoined lateral bands. At its apex is the proximal edge of the terminal tendon. This fascial ligament or layer prevents excessive volar subluxation of the conjoined lateral bands with flexion of the middle phalanx.

All the intrinsic muscles, particularly the lum-
bricals, are extremely rich in proprioceptive nerve fibers. They thus serve not only to provide power and to control the versatility of small joint motion, but they are capable of governing and integrating the function of the long flexors and extensors of the fingers. If these muscles become either con-

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**Fig. 12-6.** A, \( r_1 \) and \( r_2 \) represent radii of two pulleys with unequal diameters. B, If lifting mechanism were to be applied about both of these pulleys so that distance \( (d) \) through which the rope moved was equally shared by both pulleys, the smaller pulley would traverse a greater arc of motion \( (d_2) \). Radius of proximal interphalangeal joint is greater than that of distal interphalangeal joint. If dorsal apparatus about proximal interphalangeal joint were not allowed to slacken by subluxating volarly, distal joint motion would always exceed proximal interphalangeal joint motion when obtained through motion of extensor apparatus.
tracted or paralyzed, the function of the hand is significantly disturbed.

**INTRINSIC CONTRACTURES**

In the normal finger there is a fine balance of forces at each joint. This balance of flexors, extensors, and intrinsic is easily upset (Fig. 12-7). Intrinsic contracture of the fingers is due to an abnormally tight lumbrical, interosseus, or lateral band. If passive mobility is retained at the metacarpophalangeal and proximal interphalangeal joints, intrinsic contracture is demonstrated by the **intrinsic tightness test**: there is greater passive proximal interphalangeal flexion when the metacarpophalangeal joint is flexed and less passive proximal interphalangeal flexion when the metacarpophalangeal joint is extended (Fig. 12-8). If there is ankylosis of either proximal interphalangeal or metacarpophalangeal joints, the intrinsic tightness test cannot be performed. This test was described by Finochietto\(^ {25} \) in 1920 and has been traced by countless eponyms ever since. The site, severity, and nature of the deformity and disability resulting from intrinsic contracture depends on its cause and its duration. The treatment of the contractures varies accordingly.

**Lateral band tightness**

Occasionally the lateral band may become displaced or stretched over an osteophyte, osteochondroma, benign tumor, or cyst at the dorsal or lateral side of the proximal phalanx.\(^ {56} \) Flexion of the middle phalanx will be limited with metacarpophalangeal extension and the intrinsic tightness test will be positive. There will be no deformity of the metacarpophalangeal joint.

**Treatment.** At surgery, if the intrinsic tightness test remains positive after the tumor is resected, it is probably due to lateral band adhesions. Under such circumstances the lateral band and its oblique fibers should be excised from the middle of the proximal phalanx to the region of the proximal interphalangeal joint.

**Interosseous contracture following trauma**

Frequently there is persistent limitation of proximal interphalangeal joint flexion many months after fractures about the hand and wrists have healed.\(^ * \) Some joint stiffness may be expected to result from the swelling and immobilization that follow trauma. If the intrinsic tightness test is positive, interosseous contracture may be contributing to the stiffness. Mild contractures of the interossei following injury are usually due to the loss of elasticity of hemorrhagic muscles that have become swollen with fluid and have undergone interstitial fibrosis.\(^ {33} \)

Although contracture of the interosseous muscles affects both the metacarpophalangeal and interphalangeal joints, stiffness and deformity following mild posttraumatic interosseous contracture are usually most severe at the proximal interphalangeal joints. The collateral ligaments of the metacarpophalangeal and interphalangeal joints are lax and shortened in joint extension and stretched and lengthened in joint flexion. The

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\(^ * \)See references 14, 17, 25, 51, and 65.

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Fig. 12-7. Normally, forces at metacarpophalangeal and interphalangeal joints are balanced. At metacarpophalangeal joint, extensor tendon is balanced by intrinsics; at proximal interphalangeal joint, combined extensor (E) and intrinsic (I) force is balanced mainly by flexor digitorum superficialis (FDS); at distal interphalangeal joint, combined extensor and intrinsic force is balanced by flexor digitorum profundus (FDP).
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Fig. 12-8. Intrinsic tightness test described by Finocchietto in 1920. There is less passive proximal interphalangeal flexion when metacarpophalangeal joint is extended, A, than when metacarpophalangeal joint is flexed, B.

Collateral ligaments thus resist metacarpophalangeal joint flexion by the interossei, whereas they allow the proximal interphalangeal joint to be pulled into hyperextension. Severe contracture of the interossei overcomes the resistance of the metacarpophalangeal collateral ligaments. The metacarpophalangeal joint is pulled into a severe flexion contracture and the proximal interphalangeal joint hyperextends (Fig. 12-9). The intrinsic tightness test may be difficult to perform if passive motion at the metacarpophalangeal joint is lost.

**Treatment.** When there is mild contracture of the interossei the intrinsic tightness test is positive and stiffness is noted only at the proximal interphalangeal joint. In such hands the dorsal apparatus should be exposed through a dorsal longitudinal incision at the distal half of the proximal phalanx. Each lateral band and its oblique fibers are transected. Care is taken to preserve the transverse fibers of the intrinsic apparatus as well as the central and lateral slips of the extensor tendon (Fig. 12-10). This *distal intrinsic release* may be performed with the patient under local anesthesia. Flexion exercises are begun 2 or 3 days postoperatively.

If severe contractures cause metacarpophalangeal joint flexion deformity, a more proximal *intrinsic release* is required. If clinical examination and electromyography reveal no evidence of interosseous function, the interosseous tendons should be severed proximal to the metacarpophalangeal joints. A dorsal scalloped incision over the metacarpophalangeal joints may be used. Secondary capsular contractures may require division of the collateral ligaments of the proximal interphalangeal joints and release of the volar plates and accessory collateral ligaments of the metacarpophalangeal joints. One need not fear that proximal interosseous tendon resection in such hands will produce a claw hand deformity; the problem of

Fig. 12-9. In severe intrinsic contracture, balance at all three joints of finger is disturbed. Metacarpophalangeal joint is pulled into flexion, proximal interphalangeal joint is pulled into hyperextension, and distal joint falls into flexion due to laxity of conjoined lateral band at dorsum of middle phalanx. *E*, Extrinsic extensor digitorum communis force; *I*, intrinsic interosseous and lumbrical force; *I*^+^, abnormally increased intrinsic muscle force.

Fig. 12-10. In mild intrinsic contracture affecting motion of proximal interphalangeal joint, full proximal interphalangeal joint flexion may frequently be achieved by "distal intrinsic release," wherein lateral band and oblique fibers are excised about distal third of proximal phalanx.
overcorrection is rarely encountered after release of these contractures. More frequently full correction cannot be achieved because of secondary joint changes. Persistence of a mild flexion contracture of the metacarpophalangeal joints may well be beneficial; it will stabilize the joint and allow the common extensor tendon to fully extend the proximal interphalangeal joint after intrinsic release.

In cases of severe ischemic contracture an intersosseous muscle slide may be indicated if the intersossei are found to be functional. Through a dorsal transverse incision at the level of the midshafts of the metacarpals, the finger extensors are isolated and retracted radially, then ulnarily. Subperiosteal dissection will free the intersossei from their metacarpal origins. Capsulectomy is frequently required. The fingers are then splinted in the clawed position and the intersosseous muscles are permitted to reattach more distally. Early active interphalangeal flexion exercises are encouraged.

The intrinsic slide operation has met with some disfavor and has been criticized as being extensive, traumatic, and bloody. These criticisms appear unwarranted, as the procedure can be performed cleanly, neatly, and rapidly with little loss of blood. The intrinsic muscle slide operation effectively loosens tight but functional intersossei while preserving their function. Intrinsic contracture may be avoided in some cases of severe injury to the hand by early intersosseous fascial release.

Swan-neck deformity

Swan-neck deformity is a deformity of the finger characterized by hyperextension at the proximal interphalangeal joint and flexion at the distal interphalangeal joint (Fig. 12-11). This deformity may be caused by any imbalance of forces about the proximal interphalangeal joint that results in a relative increase in the power of extension or decrease in the power of flexion (Fig. 12-12). For the deformity to occur, the volar plate must be stretched or ruptured; an intact and strong volar ligament will prevent hyperextension of the middle phalanx. (Many otherwise normal people are capable of voluntarily hyperextending their middle phalanges because of a congenital laxity of the volar plate.)

There are many causes of the swan-neck deformity. Volar subluxation at the metacarpophalangeal joint in rheumatoid disease may increase the pull on the central slip through the tethered common extensor and produce the swan-neck deformity. Conversely, disruption of the terminal tendon attachment to the distal phalanx (a "mallet finger") may cause the power of the long extensors and intrinsics that were formerly directed to two joints to become concentrated on the middle phalanx, which is pulled into hyperextension. Laceration or transfer of the flexor digitorum superficialis will decrease the flexor force of the proximal interphalangeal joint and result in a swan-neck deformity unless care is taken to reinforce the volar side of the joint.

The swan-neck deformity is frequently caused by intrinsic contracture. The severity and duration of the contracture are among the principal factors that determine whether the finger merely becomes "tight" or the deformity develops. Regardless of whether the intrinsic contracture is due to fibrosis, ischemic necrosis, spasticity, or rheumatoid disease, a continuous increase in the extension force on the middle phalanx will gradually stretch the volar plate; the collateral ligaments will shorten as the
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Fig. 12-13. A, In long-standing swan-neck deformity there are at least four abnormalities: 1, intrinsic tightness; 2, dropped distal phalanx; 3, laxity of volar plate; and 4 tightened collateral ligaments. B, Retinacular ligament reconstruction transfers lateral band volar to Cleland's ligament. Collateral ligament is transected. Lateral and volar sides of proximal interphalangeal joint are reinforced by transposed lateral band, intrinsic contracture is released, and distal interphalangeal joint will be pulled into extension by a tenodesis effect when proximal interphalangeal joint extends.

joint hyperextends, and the conjoined lateral band will “bowstring” dorsally. The transverse retinacular ligaments, which extend volarily from the conjoined lateral bands, will tether these bands to the middle phalanx and so will restrict their excursion and power on the distal phalanx. The distal joint will then fall into flexion. When the deformity is long-standing the collateral ligaments of the proximal interphalangeal joints become contracted and the intrinsic tightness test cannot be performed (Fig. 12-13). There are four principal abnormalities that must be corrected in a finger with a swan-neck deformity due to intrinsic contracture: (1) tight intrinsics, (2) a dropped terminal phalanx, (3) a lax volar plate, and (4) contracted collateral ligaments at the proximal interphalangeal joint.

Treatment. For many patients the intrinsic tenodesis or retinacular ligament reconstruction opera-
tion corrects all of these abnormalities and provides a flexible and stable finger. Through a dorsal incision (a dorsal C incision may be used to avoid a skin wound over the proximal interphalangeal joint), one of the lateral bands is identified and is transected just distal to its transverse fibers, at the level of the proximal flexion crease of the finger. The lateral band and the more distal conjoined lateral band are sharply separated from the central slip and triangular ligament up to the terminal tendon. They are not detached from the terminal tendon.

The distal third of the opposite lateral band and its oblique fibers are resected. If necessary to permit passive flexion, the collateral ligaments are excised from both sides of the proximal interphalangeal joint. Blunt intra-articular dissection frees a volar pouch between the membranous portion of the volar plate and the head of the proximal phalanx. Capsulectomy and intra-articular dissection proceed until the joint can easily be flexed to 90 degrees without the snap that is characteristic of collateral ligament tightness. The transected end of the freed lateral band is then passed from distal to proximal under Cleland's ligament and volar to the axis of proximal interphalangeal joint motion. The joint is flexed 30 to 45 degrees and the transposed tendon is sutured to a fibrous pulley of the proximal phalanx. It will thus parallel the direction of the oblique retinacular ligament. Rerouting the lateral band volarily releases the intrinsic tightness, prevents proximal interphalangeal joint hyperextension, and lends lateral stability to the capsulectomized joint. When the proximal interphalangeal joint is extended through the common extensor tendon, the transposed lateral band will tighten, pulling on the terminal tendon and extending the distal phalanx. Thus the function of the transposed tendon is similar to that of the oblique retinacular ligament, which links proximal interphalangeal and distal interphalangeal joint extension.

The decision as to which of the lateral bands is to be tenodesed depends on the cause of the swan-neck deformity. In rheumatoid patients the ulnar lateral band is tenodesed. It is usually tighter than the radial lateral band and its more proximal fibers often require release in order to correct ulnar deviation of the fingers. In these patients the proximal third of the ulnar lateral band (the lateral tendon of the interosseus) may be transferred to the radial side of the adjacent finger, midway between the volar and dorsal cortex of the base of the proximal
phalanx, as a step in balancing the fingers that have undergone an ulnar drift. This is known as the crossed intrinsic transfer. In nonrheumatoid hands it may be preferable to tenodese the radial lateral band to afford better lateral pinch stability to the capsulectomized proximal interphalangeal joint.

Postoperative proximal interphalangeal joint flexion exercises may be begun at once. A dorsal splint is worn for 4 weeks to prevent extension of more than 45 degrees. Occasionally the correction obtained by the intrinsic tenodesis may be gradually lost. This may be due to stretching of Cleland's ligaments, which lose their effect as pulleys. In such cases, tenodesis of one slip of the flexor digitorum superficialis to the neck of the proximal phalanx will effectively stabilize the joint. If the intrinsic tenodesis has overcorrected the deformity and a boutonnière deformity occurs, the lateral band may be lengthened.

In fingers with severe intrinsic-plus and swan-neck deformities caused by cerebral palsy, tenodesis of the flexor superficialis may be combined with an intrinsic muscle slide to offer a safe combination for permanent correction of the deformity. Intrinsic tenodesis alone is often of insufficient strength to withstand the abnormal forces to which the joint may be subjected.

**Rheumatoid intrinsic contracture**

The causes of interosseous muscle contracture in rheumatoid disease of the hand is uncertain. One rarely sees the histologic changes of increased collagen deposition and fibrosis that were reported some years ago in these patients. Perivascular inflammation and increased numbers of round cells are frequently noted within these muscles but their relationship to intrinsic contracture is unclear. Some have ascribed the contracture to spasm and others to nodules on the interosseous tendons, irritation from adjacent metacarpophalangeal joint synovitis, or simply secondary to postural or positional deformity. Nonetheless, at least 40% of all rheumatoid patients appear to have interosseous muscle contracture.

At first the hands of these patients will rest in the intrinsic-plus position. Later the intrinsic tightness test will be positive and the proximal interphalangeal joint will feel stiff. Thereafter the deformity will depend on whether the integrity of the metacarpophalangeal joint remains intact. If there is little or no synovitis of the metacarpophalangeal joints, if the collateral ligaments remain strong, and if the joints do not subluxate or deviate, continued intrinsic contracture will cause a swan-neck deformity, the treatment of which has been described previously. More frequently, however, synovitis of the metacarpophalangeal joints will cause weakening of the collateral ligaments through stretching and invasion of their sites by bony attachment. The ligaments will be unable to withstand the volarly directed force of the interossei on the proximal phalanges and the joint will subluxate volarly (Fig. 12-14). Following volar subluxation the interossei will relax, and often full proximal interphalangeal joint flexion is preserved (Fig. 12-15). If the proximal phalanx can be relocated (this is often clinically impossible), proximal

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**Fig. 12-14.** Collateral ligament of metacarpophalangeal joint resists downward force of tightened intrinsic muscles under normal conditions. In rheumatoid arthritis, attachments of collateral ligament may be weakened or collateral ligament itself may be stretched. Under these circumstances, proximal phalanx readily subluxates volarly in response to increased interosseous muscle contraction.

**Fig. 12-15.** In rheumatoid hand, if proximal phalanx subluxates volarly, tightened intrinsic muscle will relax, usually allowing proximal interphalangeal joint to flex fully.
interphalangeal joint flexion may be lost. These contracted interossei are relaxed surgically by resection and shortening the metacarpal with arthroplasty.

In the rheumatoid hand, volar subluxation of the proximal phalanx is often associated with ulnar drift. In rheumatoid patients the ulnar intrinsics are tighter than the radial intrinsics and it is tempting to blame the deviation on the unequal intrinsic contracture. It appears more likely, however, that once ulnar deviation of the subluxated fingers has occurred (through a combination of other factors), the radial intrinsics will stretch and the ulnar intrinsics will contract further. Asymmetric intrinsic contracture associated with rheumatoid ulnar drift is probably the result rather than the cause of the ulnar deviation.

**Treatment.** Intrinsic release is only one component of the surgery to rebalance and reconstruct metacarpophalangeal joints with volar subluxation and ulnar deviation. Bony and tendinous insertions of all ulnar intrinsics (including the abductor digiti quinti and flexor digiti quinti proprius) should be released. The crossed intrinsic transfer is frequently of value. The transverse as well as the oblique fibers of the dorsal aponeurosis may be resected. If radial intrinsics are relatively lax (secondary to ulnar deviation), they are often best left unresected, as these muscles may be the only effective extensors of the middle phalanx remaining.

Examination of rheumatoid hands in early stages before subluxation or swan-neck deformity has occurred will often reveal a positive intrinsic tightness test and evidence of metacarpophalangeal joint synovitis. In these hands, resection of the ulnar intrinsic mechanism (medial tendon, lateral band, and transverse and oblique fibers) and the oblique fibers of the radial lateral band may restore full motion to the proximal interphalangeal joint and lessen the chance and severity of ultimate deformity of the metacarpophalangeal joint.

**Mixed and complex muscle contractures**

**Volkmann's contracture with ischemic contracture of the interossei.** The pathologic similarity of Volkmann's contracture and interosseous contracture has been noted. The deformities they cause, however, are totally different. Whereas Volkmann's contracture causes clawing of the fingers, interosseous contracture results in the intrinsic-plus position. Not infrequently, however, the two are associated. Under these circumstances the nature of the deformity will be determined by the more powerful muscles, the contracted long flexors. We thus have the paradoxical situation of an intrinsic-plus hand with a claw deformity. In these patients, however, the intrinsic contracture is usually not appreciated until after the long flexors have been released by muscle slide, tendon lengthening, or tenotomy and transfer. Only then will the intrinsic tightness test be positive and we will note (often with surprise) that full passive proximal interphalangeal joint flexion may be difficult. Volkmann's contracture should be released first. Frequently it may be unwise to completely release the intrinsic contracture; mild metacarpophalangeal joint flexion will prevent recurrence of the claw deformity.

**Cerebral palsy, stroke, and other neurological diseases.** Many muscle groups are affected by central nervous system disease. In cerebral palsy, extrinsic and intrinsic muscles may alternatively contract (become spastic) and relax. Analysis of the pathokinetcs of these hands is beyond the scope of this chapter. It should be noted, however, that many patients with central nervous system disease may be helped by appropriate intrinsic muscle release. Interosseous muscle slide has frequently proved of benefit.

**Lumbral-plus finger.** The delicate balance between the tendon of the flexor digitorum profundus and the lumbrical muscle has been emphasized earlier. If the tendon of the profundus is lacerated within the finger, the normal tone of the profundus muscle belly will pull its cut end and the attachment of the lumbral proximally. The lumbrical muscle fiber length averages 50 mm. It is thus unable to stretch the entire distance through which the profundus is usually retracted. The retracted lumbral will cause increased tension on the radial lateral band and the proximal interphalangeal joint will extend or hyperextend. This is known as the lumbral-plus finger. Amputation of the distal phalanx will also sever the profundus tendon and produce comparable consequences.

If a tendon graft is inserted too loosely, a lumbral-plus finger will result. When the patient tries to flex the interphalangeal joints, the contracting profundus will pull first on the lumbral rather than on the loose tendon graft. Paradoxical extension of the proximal interphalangeal joint will result. In effect, the profundus belly will serve as a functional continuation of the lumbral. Often a
lumbral belly is wrapped about the site of the tendon juncture in a tendon graft. If the muscle fibers become ischemic or tight, a lumbral-plus finger may be the result.

Treatment. To lessen the risk of paradoxical extension after tendon grafts, we have routinely divided the lumbral tendon in the palm or transected the radial lateral band at the distal third of the proximal phalanx. No appreciable permanent defect in proximal interphalangeal joint extension has been observed to result. Diagnosis of a lumbral-plus finger can be made if the intrinsic tightness test is positive after a tendon graft, amputation, or flexor digitorum profundus laceration and if the patient demonstrates paradoxical proximal interphalangeal joint extension. The lumbral-plus finger can best be differentiated from the finger with an associated interosseous contracture at the time of surgery. The lumbral tendon is transected through a dorsal incision. If the intrinsic tightness test remains positive, the distal third of the radial lateral band and its oblique fibers are resected. If the intrinsic tightness test is still positive, interosseous muscle contracture is probably also present, and the distal third of the ulnar lateral band and oblique fibers are resected. The intrinsic tightness test may be performed at the time of surgery, as it is not influenced by anesthesia and does not require active flexion by the patient.

The causes, effects, and treatment of intrinsic contractures are variable. The term “intrinsic release” is as specific as the term “tendon transfer” and may refer to any one of a number of procedures. The use of conservative measures such as intrinsic stretching exercises to release intrinsic contractures is rarely of much benefit except in the mildest of cases following trauma to the hand.

INTRINSIC MUSCLE WEAKNESS

Weakness or paralysis of the intrinsic muscles of the hand may follow peripheral nerve injury, peripheral neuritis, cervical cord injury, or peripheral myopathy.

Low ulnar and median nerve palsy

The term “low ulnar and median nerve palsy” refers to paralysis due to interference with ulnar and median nerve conduction distal to their innervation of the forearm muscles. In low ulnar and median nerve palsy the flexor digitorum profundus and superficialis function normally, but all the interossei, the lumbricals, and the hypothenar muscles are paralyzed. (The thenar muscles and thumb adductor are paralyzed as well.) Loss of intrinsic muscle control of the fingers disturbs (1) strength, (2) balance, (3) integration of metacarpophalangeal and interphalangeal motion, and (4) active lateral mobility of the fingers.

Strength. The interossei and flexor digiti quinti proprius are the prime flexors of the proximal phalanges. As noted, the lumbricals may also contribute to metacarpophalangeal joint flexion. It might be thought that these small muscles can add little to the strength of grip if the flexor digitorum profundus and superficialis are intact. Surprisingly, however, the grip strength of the hand following low ulnar and median nerve palsy is decreased as much as 80%.9,45

Balance. Loss of intrinsic muscle function results in an imbalance of forces at all three joints of each finger. At the metacarpophalangeal joint the long extensors have no effective antagonist through their sagittal bands and readily lift the proximal phalanx into full extension when they contract. As the volar plate stretches, the joint hyperextends. The proximal interphalangeal joint, having lost its active extension through the lateral bands, must depend on the central slip of the long extensor tendon to extend. Further contraction of the long extensors only serves to hyperextend the proximal phalanx further. The sagittal bands come to lie progressively more horizontally and the excursion of the central slip over the proximal phalanx is blocked.48 As the central slip becomes more lax, the power and excursion of the extensor tendon is expended on the metacarpophalangeal joint. The middle and distal phalanges are drawn into flexion by the intact flexor digitorum profundus and superficialis. The finger will become clawed (Fig. 12-16).

![Fig. 12-16. Clawhand typical of distal ulnar and median nerve palsy. With attempt at complete extension of fingers, metacarpophalangeal joints will hyperextend and interphalangeal joints will flex.](image-url)
Any unconscious attempt to further tighten the long extensors by palmar flexion of the wrist, only serves to increase the deformity. If interphalangeal joint extension is to be achieved, metacarpophalangeal joint hyperextension must be corrected or the paralyzed intrinsic muscles must be replaced. (Another theoretical alternative, certainly therapeutically unthinkable, would be to divide the antagonistic flexor digitorum profundus and superficialis.)

Integration of metacarpophalangeal and interphalangeal motion. The size, shape, weight, texture, and use to which an object is to be put determine the type of grip employed. Thus different patterns of grasp are required to hold a small pen, a filled coffee cup, a suitcase handle, and an orange. Pulp-to-pulp pinch (holding a needle) and hook grip (holding a suitcase handle) are relatively undisturbed by loss of intrinsic muscle function. However, any grasp that requires simultaneous metacarpophalangeal flexion and interphalangeal extension (threading a needle or using a fine paint brush) or simultaneous metacarpophalangeal and interphalangeal flexion (grasping a large orange) will be awkward (Fig. 12-17). The metacarpophalangeal joint does flex in intrinsic paralysis, but only after interphalangeal flexion is complete. The fingers curl as they are flexed—the interphalangeal joints first, the metacarpophalangeal joints last. A large object is pushed out of the palm by the curling

Fig. 12-17. In intrinsic muscle palsy, flexion of metacarpophalangeal joints only occurs after interphalangeal joints are fully flexed. Fingers thus curl into hand, pushing away any large object they wish to grasp. This results in marked awkwardness of many types of grip.
fingertips as the long flexors contract.\textsuperscript{3,4} Thus, for many types of grasp, simultaneous metacarpophalangeal joint flexion and interphalangeal joint flexion are essential.

Active lateral mobility. The fingers converge when flexed and diverge when extended. This facilitates the grasp of large objects, which can be surrounded by the spread fingers. The automatic divergence and convergence of the fingers in extension and flexion are due to the anatomic alignment of the metacarpophalangeal joints and are not dependent on active lateral mobility. The interosseus muscles and the hypothenars can abduct and adduct the fingers only in metacarpophalangeal joint extension. In metacarpophalangeal flexion, not only are the proximal phalanges drawn firmly toward each other, but the tightened collateral ligaments prevent lateral deviation of the proximal phalanges.

In addition to actively deviating the fingers, the interosseous also support the lateral sides of the metacarpophalangeal joints. Such support is unnecessary if the collateral ligaments are intact. However, if too radical a capsulectomy is performed for stiffened metacarpophalangeal joints in the presence of intrinsic palsy and if the collateral and accessory collateral ligaments are totally excised, there is a risk of converting a clawhand into a hand with severe ulnar drift due to loss of lateral joint support.

Low ulnar nerve palsy

The deformity and disability produced by intrinsic paralysis following peripheral nerve trauma depend on the site of injury and whether or not the median nerve is injured along with the ulnar nerve. Usually the median nerve supplies the radial two lumbricals. In low ulnar nerve palsy without median nerve involvement these lumbricals may prevent clawing of the index and middle fingers (Fig. 12-18). They also integrate metacarpophalangeal joint flexion at the same time that the long flexors flex the interphalangeal joints. As the "workhorse of the extensor apparatus," the lumbricals also strongly and fully extend the interphalangeal joints, even in the presence of interosseous palsy. However, lateral deviation of the fingers and the normal strength of metacarpophalangeal joint flexion are lost. Thus in low ulnar nerve palsy there is weakness of grip, loss of lateral deviation of all the fingers, clawing of the ring and little fingers, and loss of integrated metacarpophalangeal and interphalangeal flexion of the ring and little fingers.

![Fig. 12-18. In low ulnar nerve palsy, when median nerve is intact, lumbrical muscles to index and middle fingers prevent these digits from clawing. Resultant posture at rest is likened to "position of benediction."](image)

High ulnar nerve palsy

In high ulnar nerve palsy the flexor carpi ulnaris and the flexor digitorum profundus of the ulnar two fingers are lost. The effect on the fingers is similar to that of low ulnar nerve palsy except that clawing is less severe because of profundus loss. Loss of the flexor digitorum profundus also further weakens the grip.

High ulnar and median nerve palsy

In high ulnar and median nerve palsy there is a loss of action of the flexor digitorum profundus and superficialis in addition to total intrinsic paralysis. Thus the common extensor tendons have no antagonist. When they contract, the interphalangeal joints extend completely despite metacarpophalangeal hyperextension. The fingers do not claw. All flexion at all joints is lost and the hand is essentially without function. If the ulnar and median nerves are successfully repaired, the long flexors are reinnervated before the intrinsics. As the flexor digitorum profundus and superficialis regain their tone, the extensor digitorum communis regains its antagonist and the fingers will gradually assume the clawed position typical of low ulnar and median nerve palsy. This is a good prognostic sign. Similarly, if the flexor digitorum profundus is replaced by tendon transfers, clawing of the fingers may be anticipated.

Nerve damage plus muscle contracture

In Volkmann's contracture, ischemic necrosis of the forearm musculature is usually (but not always)
associated with severe injury to the median and ulnar nerves. Anatomically there is a high ulnar and median nerve palsy. The severe contracture of the muscle bellies of the flexor digitorum profundus and superficialis in the presence of nerve palsy causes the most severe claw deformities noted clinically. There is total imbalance about the fingers, with the extrinsic flexors overpulling and the intrinsic muscles paralyzed. To compound this imbalance, ischemic contracture of the wrist flexors draws the wrist into extreme palmar flexion, exerting abnormal tension on the extensor digitorum communis. The metacarpophalangeal joints of the fingers further hyperextend and the flexion contracture of the interphalangeal joints is accentuated. Here, indeed, the term “clawhand” is justified. If the hand is not properly splinted and exercised, contractures of the skin, tendons, muscles, and joint capsules may occur. The dorsal aponeurosis stretches, tendons of the dorsal interossei may come to lie dorsal to the axis of the metacarpophalangeal joints, and the volar capsule may invaginate and adhere to the metacarpal head.

**Treatment**

The treatment of intrinsic palsy depends in large measure on its course and the patient’s complaints. Certainly the progressive muscular weakness associated with Charcot-Marie-Tooth disease, the severe and diffuse paralysis associated with brachial plexus injury, the combined motor and sensory loss in leprosy, and the combined upper and lower motor neuron lesion following cervical fracture-dislocation each require a different approach in the treatment of the intrinsic palsy. The surgical procedures to be outlined deal specifically with measures to correct the deformity and disability of fingers with intrinsic muscle palsy. Surgical measures to correct other abnormalities of the palsied hand are not considered here.

Procedures to correct deformities of fingers with intrinsic palsy can be classified into two main groups: those that prevent or correct metacarpophalangeal joint hyperextension and those that actively flex the metacarpophalangeal joints or both flex the metacarpophalangeal and extend the interphalangeal joints (Fig. 12-19).

Surgical procedures to correct clawing of the hand, particularly tendon transfers, should be postponed until all joints are supple. Splints, serial casts, physical therapy, or capsulectomy may be employed to loosen tight ligaments. One should not perform reconstructive surgery until the passive joint motion achieved is considered satisfactory for good function.

**Procedures to prevent metacarpophalangeal hyperextension.** Normally, full proximal interphalangeal joint extension can be achieved in intrinsic paralysis if metacarpophalangeal joint hyperextension is prevented. In long-standing claw deformity, however, the central slip may become so attenuated and lengthened over the dorsum of the proximal interphalangeal joint that the common extensor can no longer extend the middle phalanx. The exam-

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![Fig. 12-19. There are many means of correcting ulnar clawhand deformity. A, Volar capsule may be tightened. B, Tendon transfer may be passed through palm to lateral band (volar to deep transverse metacarpal ligament). C, Tendon transfer may be passed dorsally between metacarpals, passed volar to deep transverse metacarpal ligament, and inserted onto lateral band.](image-url)
iner can readily test the competence of the extensor digitorum communis and the central slip by simply holding the metacarpophalangeal joint in flexion and requesting the patient to actively extend the proximal interphalangeal joint. If full extension is possible, one can expect that preventing metacarpophalangeal hyperextension will correct the claw deformity. It will not increase the power of flexion; it will not provide coordinated metacarpophalangeal and interphalangeal flexion; it will not provide independent interphalangeal joint extension; and it will not provide active lateral mobility.

Splints. The dorsal “knuckle bender” is an “active” splint with rubber bands that provide mild volar pressure at the dorsum of the proximal phalanx. Full metacarpophalangeal joint extension can be obtained if the rubber bands are applied loosely. The splint is somewhat bulky and cumbersome but corrects the claw deformity. A “lumbrical bar” is a small static dorsal splint that prevents hyperextension of the metacarpophalangeal joints. It is usually placed to hold these joints at an angle of about 25 degrees. (The term “lumbrical bar” is somewhat of a misnomer, as metacarpophalangeal flexion is not the prime responsibility of the lumbricals.) The splint is light, more aesthetic, and less awkward than the “knuckle bender.” It does not, however, allow the metacarpophalangeal joints to fully extend in grasping large objects.

Surgical procedures. The insertion of a dorsal bone block into the head of the metacarpal has been recommended to prevent hyperextension of the proximal phalanx. Because of complications due to resorption of the bone block and the necessity to osteotomize the articular cartilage of the metacarpal head, the procedure is rarely used today.

Volar capsulorrhaphy was advised by Zancolli and Bunnell for the treatment of claw deformity. The proximal pulley is opened through a volar incision and the flexor tendons are retracted. The volar plate is incised beneath the metacarpal head, two incisions are made laterally, and the volar capsule is advanced proximally. The joint is flexed about 20 degrees and the volar plate resutured into its new position. Thus a mild flexion contracture of the metacarpophalangeal joints is created to correct the claw deformity.

Several modifications of this procedure have been suggested to prevent the gradual stretching of the resutured capsule and recurrence of the deformity. Some have recommended the excision of 1.5 cm. wide ellipse of palmar skin and the subcutaneous tissues from the midpalm to prevent stretching of the resutured capsule. Others have advised inserting the advanced volar plate into the metacarpal neck with a pullout wire and holding it in this position with a dorsal splint for a minimum of 6 weeks. During this time the interphalangeal joints are actively exercised. The procedure may be combined with a pulley advancement.

The metacarpophalangeal joint may be held in flexion by tenodesis. Parkes uses a tendon graft attached proximally to the transverse volar ligament and distally to the lateral band of each finger. Riordan has suggested a tenodesis if there is weakness of the extensor digitorum communis and the flexor digitorum superficialis. He divides the extensor carpi radialis longus and extensor carpi ulnaris longitudinally to the juncture of the middle and distal thirds of the forearm. One half of each of the divided tendons is further split longitudinally. The four tendon strands, still attached to their insertions, are passed between the metacarpals and then volar to the deep transverse metacarpal ligament, finally to be attached to the lateral bands.

Each of these procedures, if successful, corrects the claw deformity. Each permits full interphalangeal joint extension through the extensor digitorum communis if the central slip is not stretched and the joint is mobile. None of these procedures increases the power nor alters the pathokinetics of finger motion.

Procedures to actively flex the metacarpophalangeal joint and extend the interphalangeal joint. Many procedures to flex the metacarpophalangeal joint and extend the interphalangeal joint through active control have been developed. In each of the tendon transfers and active tenodesis the tendon passes volar to the deep transverse metacarpal ligament. With few exceptions the tendon is attached distally to the lateral bands. Some of these procedures are summarized here.

Bunnell pulley advancement

METHOD: Each side of the proximal pulley is split 1.5 to 2.5 cm. to the middle of the proximal phalanx “until the pull on the flexor tendon flexes the metacarpophalangeal joint well.” The flexor digitorum profundus and superficialis will “bowstring” somewhat, increasing the moment arm on the proximal phalanx and therefore increasing the power of metacarpophalangeal flexion. The procedure is not effective if there is a slack central slip. The procedure is frequently performed along with volar capsulorrhaphy.
Intrinsic muscles of the fingers: function, dysfunction, and surgical reconstruction

Stiles tendon transfer

**Motor:** Flexor digitorum superficialis.

**Tendon:** Flexor digitorum superficialis.

**Route:** Volar to dorsal—one half of split flexor digitorum superficialis to either side of proximal phalanx.

**Attached to:** Extensor digitorum communis.

**Comments:** This procedure is mainly of historical interest. Stiles reported only two cases, neither of which was followed up. Bunnell tried the procedure with unsuccessful results and then modified it.

Stiles-Bunnell tendon transfer

**Tendon:** Flexor digitorum superficialis.

**Route:** Volar—split superficialis of index finger to radial sides of index and middle fingers; split superficialis of middle finger to ulnar sides of index, middle, and ring fingers; split superficialis of ring finger to radial sides of ring and little fingers; split superficialis of little finger to ulnar side of little finger. The ends are attached to the dorsal surface of the transverse fibers and lateral band of the intrinsic mechanism.

**Comments:** Bunnell had hoped to provide lateral deviation to each of the fingers as well as to correct the clawing. It required use of all four superficialis tendons with two transfers to each finger. The surgery was time-consuming and required multiple incisions on each finger. Many tendon strands crossed in the palm, risking adhesions.

Modified Stiles-Bunnell tendon transfer

**Motor:** Flexor digitorum superficialis.

**Tendon:** Flexor digitorum superficialis.

**Route:** Volar—one or two superficialis tendons are used and split into several strands, one to each finger.

**Attached to:** Lateral bands.

**Comments:** This procedure is much more popular than the original Stiles-Bunnell transfer. It does not add strength to the fingers and may even decrease their strength. Occasionally, particularly in leprosy patients, secondary swan-neck patients deformity may develop after several years. The transferred tendon is out of phase for interphalangeal extension.

Riordan tendon transfer

**Motor:** Flexor carpi radialis.

**Tendon:** Brachioradialis tendon for graft.

**Route:** Dorsal—through intermetacarpal spaces to lumbrical canal.

**Attached to:** Lateral bands.

**Comments:** The transferred tendon is in phase for interphalangeal extension; no reeducation is necessary; and wrist palmar flexion, which is often overactive, is decreased.

Fowler tenodesis

**Motor:** Wrist flexion (through normal wrist flexors).

**Tendon:** Two extensor tendon grafts.

**Route:** Dorsal—tendons are attached through a hole in the dorsal carpal ligament and then through the intermetacarpal spaces and the lumbrical canal.

**Attached to:** Lateral bands.

**Comments:** The tenodesis works actively since the tendons are sutured proximal to the wrist joint. Thus in wrist flexion the tenodesis is effective; in wrist extension it is relaxed.

Extensor proprius tendon transfer, Bunnell proprius transfer, or Fowler tendon transfer

**Motor:** Extensor indicis proprius and extensor digiti quinti.

**Tendon:** Extensor indicis proprius split and extensor digiti quinti split.

**Route:** Dorsal—through intermetacarpal spaces and lumbrical canals.

**Attached to:** Lateral bands; extensor indicis proprius to index and middle fingers, extensor digiti quinti to ring and little fingers.

**Comments:** Fowler originally attached the extensor digiti quinti to the ulnar sides of the ring and little fingers; Riordan recommends attachment to the radial side of all four digits. Transfer decreases the metacarpophalangeal hyperextension of the index and little fingers. The extensor digiti quinti is often very thin when split. The extensor digitorum communis to the little finger is often vestigial.

Brand dorsal route intrinsic transfer

**Motor:** Extensor carpi radialis brevis or longus.

**Tendon:** Plantaris, split in four.

**Route:** Dorsal—through intermetacarpal spaces, then lumbrical canals of middle, ring, and little fingers and ulnar side of index finger.

**Attached to:** Radial lateral bands of middle, ring, and little fingers and ulnar lateral band of index.

**Comments:** The strong motor increases the power of flexion of the fingers.

Brand volar route intrinsic transfer

**Motor:** Extensor carpi radialis longus or brachioradialis.

**Tendon:** Plantaris, split in four.

**Route:** Volar—through carpal canal.

**Attached to:** Radial lateral band of middle, ring, and little fingers and ulnar lateral band of index finger.

**Comments:** The procedure increases the mass in the carpal tunnel and thus risks a possible carpal tunnel syndrome.

Burkhalter transfer

**Motor:** Extensor carpi radialis longus or brachioradialis.

**Tendon:** Palmaris longus or plantaris graft.

**Route:** Volar—through intermetacarpal spaces, then lumbrical canal.

**Attached to:** Hole in proximal phalanx.

**Comments:** Acts directly on metacarpophalangeal flex-
ion. Interphalangeal extension must occur through the extensor digitorum communis and central slip. The power of grip is significantly increased, particularly in high ulnar and median nerve palsy.

Brooks-Jones transfer

Motor: Flexor carpi radialis.
Tendon: Toe extensors.
Route: Through carpal canal.
Attach to: Proximal pulley.
Comments: Acts directly on metacarpophalangeal flexion as Burkhalter transfer. The procedure is easier technically than bone insertion but there is a risk of loosening of the pulley.

Zancolli “lasso” operation

Motor: Flexor digitorum superficialis.
Tendon: Flexor digitorum superficialis.
Attach to: Looped around proximal pulley.
Comments: This is a modified Stiles-Bunnell procedure but the superficialis is attached to the pulley instead of the lateral band.

Fritschi transfer

Comments: Fritschi used the Brand procedure but found that fascia lata serves as excellent graft material in lepers.

Lennox transfer

Motor: Palmaris longus.
Tendon: Plantaris, split in four.
Route: Carpal canal.
Attach to: Lateral bands.
Comments: Used only in highly mobile hands.

It should be unnecessary to emphasize that these procedures for total intrinsic paralysis must be modified in hands having only ulnar nerve palsy. In the case of progressive disease such as leprosy it has been suggested that intrinsic transfers be performed to all four fingers, including those not (yet) paralyzed. If not, these digits may feel weak and awkward and may later develop paralysis and deformity.

With so many procedures from which to choose, several considerations must of course influence our choice.

1. The volar route transfers require immobilization of the wrist in palmar flexion postoperatively and thus can be performed simultaneously with median and ulnar nerve repair, opponens plasty, or repair of flexor tendons. The transfer is more direct than the dorsal route transfers and does not risk development of adhesions in the intermetacarpal spaces.

2. In dorsal route transfers, wrist palmar flexion will potentiate interphalangeal joint extension. These motions are usually synergistic and thus readily learned. The dorsal route transfer also diminishes the risk of causing carpal tunnel syndrome. The problem of possible adhesions of the transfer between the metacarpals, however, should be considered.

3. The use of a wrist flexor or extensor or the brachioradialis as the motor for the tendon transfer may increase the power of metacarpophalangeal joint flexion as well as correct the deformity and restore the normal kinetics of finger motion. A static tenodesis or the use of a flexor digitorum superficialis or extensor indicis proprius tendon will not appreciably increase the power of grip.

4. In high median and ulnar nerve palsy, neither wrist flexors nor the flexor digitorum superficialis can be used for transfer.

5. Interphalangeal joint extension with metacarpophalangeal joint flexion can best be achieved in the supple hand by transfers to the lateral bands. Transfers to the proximal phalanx or to the proximal pulley will not provide independent proximal interphalangeal extension but will prevent the risk of overcorrection and swan-neck deformity.

6. If the middle and distal phalanges cannot be extended when the metacarpophalangeal joint is stabilized in mild flexion, any tendon transfer should be attached to the dorsal apex of the proximal interphalangeal joint or to the conjoined lateral bands of the middle phalanx. The little finger is sutured last and is placed under somewhat greater tension than the radial digits. It has been recommended that sutting of the transfer in the supple hand be performed with the wrist straight, the metacarpophalangeal joints flexed at 90 degrees, and the interphalangeal joints straight.

It has been questioned whether intrinsic tendon transfers work actively or merely by their tenodesis effect. There is little question that many of these tendons work in proper phase and effectively correct the deformity, integrate metacarpophalangeal flexion and interphalangeal extension, and increase the power of finger flexion. No doubt, some do become adherent to local tissues; in such cases they may serve principally to prevent metacarpophalangeal hyperextension.
Procedures to restore lateral deviation of the metacarpophalangeal joints. In ulnar nerve palsy the little finger deviates ulnarly during metacarpophalangeal extension through the action of the extensor digitii quinti proprii. Tendons transferred to restore metacarpophalangeal joint flexion are usually sutured to the radial sides of the middle, ring, and little fingers and to the ulnar side of the index finger. Thus the fingers are drawn together after intrinsic tendon transfer. For strong pinch, radial deviation of the index finger must be restored (as well as thumb adduction). Several tendon transfers have been recommended, all of which include suturing the transferred tendon either to the lateral band of the proximal third of the proximal phalanx or into the lateral side of the proximal phalanx itself. The motors suggested for first dorsal interosseous transfer have included the extensor indicis proprius, extensor carpi radialis longus (with a tendon graft), extensor pollicis brevis, extensor digiti quinti proprius, and flexor digitorum superficialis. In performing this transfer, one must be cautious not to provide too much power of radial deviation since ulnar deviation through intrinsic tendon transfer may be insufficient to restore the finger to its neutral position. Although function is usually significantly improved by transfer to first dorsal interosseus, the strength of pinch is often not materially improved.

CONCLUSIONS

The intrinsic muscles “complete with the long extensors and long flexors the muscle balance in the hand. Normal position, normal motion, and even strength of grip of the hand are dependent on this nice balance of these three sets of muscles.” In planning the reconstruction of the palsied or injured hand, one must carefully consider the condition of the intrinsic muscles. The delicate anatomic structures and balance of the normal finger demand our study and understanding. Only then can we intelligently diagnose, analyze, and correct the abnormalities that may impede the intricate systems that normally afford the hand such extraordinary versatility.

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