Redistribution of Forces in the Correction of the Boutonnière Deformity

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Detachment of either interphalangeal extensor-tendon insertion disrupts joint equilibrium. Two common deformities illustrate this imbalance:

1. Hyperextension of the proximal interphalangeal joint secondary to disruption

Redistribution of Forces in the Correction of the Boutonnière Deformity*  

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Fig. 2,a: Disruption of the extensor insertion at the proximal interphalangeal joint results in a characteristic static deformity with flexion of the middle and extension of the terminal phalanges. Volar displacement of the lateral bands accentuates the deformity and can lead to irreversible disabilities secondary to a fixed joint contracture.

Fig. 2,b: A simple dorsal incision gives full exposure of the extensor mechanism.

Fig. 2,c: The extrinsic and interosseous extensor mechanism is separated from the lumbrical tendon and oblique retinacular ligaments (O.R.L.).

Fig. 2,d and e: The major extrinsic and intrinsic extensor force can now be concentrated solely on the middle phalanx by detaching and releasing the volarly displaced lateral bands, rotating them dorsally on to the remnant of the central slip which is left intact and, while the proximal joint is held in full extension, suturing them to each other in the mid-line and to the central slip.
Fig. 2,f: The proximal interphalangeal joint is transfixed in extension with a Kirschner wire which is removed after two weeks.

Fig. 2,g: With extrinsic and interosseous power concentrated wholly on the middle phalanx, active extension of the proximal interphalangeal joint tenses the retinacular ligament which aided by the lumbrical extends the terminal phalanx.

**Fig. 3-A**

Figs. 3-A through 3-F: A housewife, forty years old, lacerated her index-finger extensor tendon over the proximal interphalangeal joint with a paring knife three months before surgery. Initial treatment had been skin closure. When she was examined, three months after injury, there was a typical boutonnière deformity.

Fig. 3-A: Preoperative maximum extension.

of the terminal phalangeal extensor insertion (mallet deformity). (Here detachment of the insertion on the terminal phalanx releases the oblique retinacular ligament and diverts all active extensor power to the middle phalanx. The hyperextension deformity develops as the normal restraint of the volar plate is attenuated.)

2. Flexion of the proximal interphalangeal joint secondary to disruption of the extensor insertion on the middle phalanx (boutonnière deformity).

Our success in correcting the hyperextension deformity at the proximal interphalangeal level by restitution of the oblique retinacular ligament led naturally to a further analysis of this structure's role in correcting the reciprocal flexion deformity.

The basic anatomy of the finger extensor mechanism has been known for several centuries. Recently, however, stimulated by the studies of Houck, Landsmeer, and Benninghof, the subtleties of the checks and balances inherent in this complicated system have been recognized and applied in reconstructive surgery. An exact knowledge of the normal and abnormal anatomy of the extensor mechanism is imperative for surgical correction of finger deformities.

The traditional management of disruption of a middle phalangeal extensor tendon insertion has been external immobilization or a primary surgical reattachment. Mason stated, in 1930, "the condition responds well to operative treatment, though full, free movement can scarcely be promised." Some active extension may be maintained by these methods, but some loss of flexion may occur due to extensor tendon adherence proximal to the joint. Secondary procedures have in general led to an unpredictable result. The many proposed methods of reconstruction using the ulnar lateral band, thin tendon grafts, advancement of the central band and relocation of the lateral bands, and tenotomy, all attest to the uncertain results of any one approach. It appears that even the most careful reconstructive surgery is often negated by the effects of the highly reactive and proliferative extensor paratenon.
Full passive finger flexion is restored after release of the extensor restraint on the distal phalanx.

**Anatomy**

*Normal*

Active extension of the interphalangeal joints is accomplished by the synergistic yet separate and essentially independent extrinsic and intrinsic systems. The extrinsic tendons which trifurcate over the proximal phalanx into central and lateral bands are reinforced by the intrinsic system, the interosseous and lumbrical tendons. The central band is undifferentiated over the dorsal capsule of the proximal interphalangeal joint, and its attachment to the lateral bands has an unique, elastic property permitting their volar displacement as the finger flexes. Each system by itself is capable of extending the interphalangeal joints, provided metacarpophalangeal joint extension is prevented.

The oblique retinacular ligament acts as a dynamic tenodesis serving principally to aid extension of the distal phalanx when tightened by extension of the
Tenodesis effect of the retinacular ligament is demonstrated by extension of the proximal joint which produces full extension of the terminal joint.

With the proximal joint in full extension, the lateral bands are folded dorsally and sutured to each other and to the central slip to reinforce the attenuated proximal insertion. Note that the retinacular ligaments remain in the plane of the axis of joint rotation of the proximal joint.

Active flexion and extension five months after operation. Although mild deformity persists, function of the index finger has been greatly improved.
proximal interphalangeal joint. Normally, the ligament passes just volar to the axis of the proximal interphalangeal joint and is tensed only by extreme flexion of the distal interphalangeal joint or extreme extension of the proximal interphalangeal joint. In states of imbalance such as the boutonnière deformity or in Dupuytren’s malady, this ligament, especially in the little finger, hypertrophies, shortens, and leads to fixed, exaggerated extension of the terminal phalanx (Fig. 1).

Abnormal

The boutonnière deformity occurs when extensor equilibrium has been disrupted by laceration, rupture, or gradual dehiscence of the critical assemblage of tendons on the dorsal aspect of the interphalangeal joint, resulting in flexion of the middle and extension of the terminal phalanges. Loss of tendinous fixation on the extensor aspect of the middle phalanx permits varying degrees of volar migration of the lateral bands and a paradox develops at this level: the extrinsic and intrinsic systems, normally extensors, now become prime flexors of the proximal interphalangeal joint. In simple or early cases, this joint can be passively extended. However, in long-standing cases, irreversible changes occur in the collateral ligaments and volar plate. The displaced lateral bands become fixed volar to the axis of joint rotation and maintain the flexed position, permitting shortening of the oblique retinacular ligaments. Thus the normal distal tenodesis mechanism is disrupted and joint equilibrium is decompensated.

Treatment

A simple operation has been developed whereby all active extensor force is diverted to the middle phalanx and the extensor restraint on the terminal phalanx is released. By carefully preserving the pathologically shortened oblique retinacular ligament and the lumbrical contribution to the radial lateral band, extension of the distal joint will be preserved (Fig. 2).

Technique

Through a dorsal incision (Fig. 2, b), the lateral bands are identified. The lumbrical tendon and radial retinacular ligament are separated from the main por-

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**Fig. 4-A**

Figs. 4-A and 4-B: A printer, forty-three years old, had a crushing injury of all fingers of his left hand fifteen months earlier. The index and long fingers had to be amputated. A boutonnière deformity of the little finger which did not have an open wound developed and severely compromised the remaining function in his hand.

Fig. 4-A: Maximum passive correction of chronic boutonnière deformity before surgery. Note the mild volar subluxation of the middle phalanx.
Correction of Boutonnière Deformity

Nine months after operation there is essentially full extension and flexion of both interphalangeal joints.

tion of the subluxated radial lateral band by sharp dissection. Similarly, on the ulnar side, the lateral band is separated from the oblique retinacular ligament which is preserved intact. The extrinsic and interosseous intrinsic tendons are thus completely separated from the lumbrical and oblique retinacular fibers (Figs. 2,c and 3-B). To prevent volar reattachment, the liberated lateral bands are folded dorsally, bringing their volar margins to the mid-line on the dorsum of the finger where they are sutured with the proximal joint held in extension, thereby reinforcing the attenuated central band (Figs. 2,d and 3-E). Occasionally when excessive redundancy of the attenuated central band is present, the relocated lateral bands should be sutured into drill holes in the dorsal cortex of the base of the middle phalanx. All active extrinsic extensor and interosseous muscle power is now concentrated on the middle phalanx through the cicatrized and reinforced central insertion and, as active extension of the proximal interphalangeal joint occurs, the lumbrical-reinforced retinacular ligament, through its tenodesis effect, extends the distal phalanx (Figs. 2,g and 3-D). After operation the proximal joint is maintained in full extension by a transarticular Kirschner wire for two weeks (Fig. 2,f). Gentle active motion is begun three weeks after operation.

This procedure has been carried out in eight patients with boutonnière deformity, the result of a variety of causes exclusive of rheumatoid arthritis. All deformities have been improved. In those patients with displacement of only one lateral band, care must be taken not to disturb the less displaced band. It is the volar displacement of the lateral band which creates the flexion deformity. It is therefore most important that nothing be done to a band that is only minimally displaced. Under these circumstances the procedure described is performed only on the side where the lateral band is displaced. In the acute phase, this procedure is con-
traindicated since the presence of tendon-scar continuity is a prerequisite for the dorsal relocation of the lateral bands.

Discussion

In the normal finger, the extrinsic extensor and intrinsic muscle systems act on the interphalangeal joints, maintaining an exquisite balance of forces. When this equilibrium is disrupted at either the proximal or distal joint level, both joints are impaired. A simple release-type operation is proposed which separates the extrinsic extensor and the interosseous components from the lumbrical and retinacular systems, diverting all active power to extension of the proximal interphalangeal joint. This separation creates two interdependent systems which provide full extension of both proximal and distal joints, the latter through the dynamic tenodesis effect of the oblique retinacular ligaments.

Dolphin, using Fowler's tenotomy technique, to relieve the disability of the hyperextended terminal phalanx of a boutonnière deformity, observed improvement following section of the extensor tendon distal to the dorsal transverse retinacular fibers. Since an immediate mallet-finger deformity did not develop, it is apparent that the retinacular ligament's distal insertion was spared, thereby retaining tenodesis extension of the terminal phalanx. In such a release, it is essential to recognize and preserve this ligament; otherwise, a significant number of drop-finger deformities may result.

Summary

Secondary reconstructive procedures to correct chronic boutonnière deformity involving reparative or additive techniques frequently fail to restore the necessary full excursion of the extensor aponeurosis. A simple release procedure based on precise anatomy, which redistributes the forces acting on the interphalangeal joints, has been successful in restoring full interphalangeal extension and flexion in a limited number of patients.

References

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