The Functional Anatomy of the Extensor Mechanism of the Finger

CRAMPTON HARRIS, JR. and GUY L. RUTLEDGE, JR.


This information is current as of July 24, 2010

Reprints and Permissions

Click here to order reprints or request permission to use material from this article, or locate the article citation on jbjs.org and click on the [Reprints and Permissions] link.

Publisher Information

The Journal of Bone and Joint Surgery
20 Pickering Street, Needham, MA 02492-3157
www.jbjs.org
The Functional Anatomy of the Extensor Mechanism of the Finger

BY CRAMPTON HARRIS, JR., M.D., AND GUY L. RUTLEDGE, JR., M.D.,
MOBILE, ALABAMA

Extension of the human finger is achieved by a complex mechanism. In the past, and at present, there exists considerable difference of opinion as to how extension is achieved, and what the functions of the ligaments and tendons involved are. In reconstruction of the hand we must attempt to understand the normal function of the various anatomical elements for otherwise it will not be possible to formulate logical plans for the treatment of specific injuries. The purpose of this paper is to present new anatomical observations on the subject of extension of the finger, in the hope that they may help clarify matters still in dispute.

In order to determine the true function of the various components of the extensor mechanism, we performed our investigations on fresh material, which had not been preserved, or subjected to any treatment that might alter normal function.

We then tried to eliminate systematically the various components of the extensor mechanism so that the function of the remaining parts could be determined. This was performed serially at each joint of the finger, and the rationale and results were recorded.

The Distal Interphalangeal Joint

For many years, it was considered that the true extensor of the distal interphalangeal joint was the two lateral bands of the extensor mechanism which, after they join together, insert into the distal phalanx. In 1949, Landsmeer called our attention to the oblique retinacular ligament which had been previously noted by Weitbrecht in 1742. In his work at that time, Landsmeer stated "Extension at the proximal interphalangeal joint also implies a corresponding extension of the distal joint. The retinacular ligament, which passes ventral to the axis of the joint, is put under tension and exerts a force on the terminal tendon, resulting in extension of the third phalanx." He postulated that extension of the distal phalanx was a combination of the action of the common extensor through the lateral bands and tenodesis effected through the oblique retinacular ligament. In other words, if the proximal interphalangeal joint is brought up into extension, then the distal interphalangeal joint must also go into extension due to tightening of the oblique retinacular ligament. In 1951, Haines stated, "... the middle and distal joints usually extend together ... in the earlier stages of the movement [of extension], the tendon to the terminal phalanx is absolutely slack owing to the volar shift mechanism, and in the later stages the link mechanism comes into play and the terminal joint extends with the middle." Stack, in 1962, wrote, "On pulling and relaxing the extensor tendon, the middle phalanx is extended and flexed, and at the same time the retinacular ligament transmits the pull to the base of the distal phalanx, and the distal interphalangeal joint is extended and flexed. The two joints move in concert and are always at the same angle." He also constructed a model to show "that the retinacular ligament links the movements of
the two distal joints, transmitting the pull of the median band (central slip) to the distal phalanx". Stack apparently felt that the retinacular ligament was an extensor of the distal phalanx, although not a complete one, for he stated that, "another extending system is required to achieve full extension of the distal joints." Recently, in 1969, Stack stated that the oblique retinacular ligament extends the distal interphalangeal joint from 90 to 45 degrees, and the lateral bands extend it from 45 to 0 degrees.

**Method and Observations**

We have been particularly interested in the function of the oblique retinacular ligament. In a fresh finger, after the skin and subcutaneous tissue were removed, the oblique ligament could be seen as a small ligamentous structure running from the flexor-tendon sheath on the volar side of the proximal phalanx, passing obliquely dorsalward, and inserting into the lateral band as it inserts into the distal phalanx (Fig. 1). It was much thinner than the joined lateral bands at the level of their insertion into the distal phalanx. The oblique ligament is a tiny structure, and must be looked for carefully if it is to be found at all. If size bears any relation to function, the relative size of the long extensor would seem to indicate that it is much the more important.

![Fig. 1](image)

*Fig. 1*

The oblique retinacular ligament is a small fibrous structure which arises from the volar aspect of the proximal phalanx and the flexor-tendon sheath. It passes obliquely dorsally and joins the lateral band as it goes distally to insert into the distal phalanx.

In the fresh hand, we then held the proximal interphalangeal joint in full extension at 0 degrees, and noted that the oblique retinacular ligament was relaxed. While maintaining the proximal interphalangeal joint at 0 degrees we slowly flexed the distal joint until the oblique retinacular ligament became taut. In the fresh hands which we have dissected, this occurred at about 70 degrees of flexion of the distal interphalangeal joint.

It is difficult to see how the oblique retinacular ligament could be considered an extensor of the distal phalanx, since it was relaxed from 0 to 70 degrees of flexion of the distal interphalangeal joint. If the proximal interphalangeal joint was fully flexed, then the distal interphalangeal joint could be flexed to 90 degrees. When the proximal interphalangeal joint was brought up into full extension, the distal joint could be flexed to 70 degrees. Thus the oblique ligament could contribute only 20 degrees to the extension of the distal joint when acting alone, and this only from 90 to 70 degrees.

In order to determine further the function of the oblique retinacular ligament, it was carefully sectioned on both sides of the finger, completely freeing the extensor mechanism from it. Then, alternating pull was made, first on the flexor profundus tendon, and then on the long extensor and intrinsics together. Insofar as we could tell, flexion and extension of the finger were completely normal. In other words, loss of the oblique retinacular ligament caused no perceptible functional loss.

In another freshly dissected finger we pulled proximally on the long extensor and the intrinsics, and complete finger extension resulted. While holding the finger in
EXTENSOR MECHANISM OF THE FINGER

TABLE I

<table>
<thead>
<tr>
<th>Finger Moving from Extension to Flexion</th>
<th>PIP joint was at</th>
<th>MP joint was at</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Degrees)</td>
<td>(Degrees)</td>
<td>(Degrees)</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>10</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>25</td>
<td>35</td>
<td>20</td>
</tr>
<tr>
<td>35</td>
<td>55</td>
<td>25</td>
</tr>
<tr>
<td>45</td>
<td>75</td>
<td>40</td>
</tr>
<tr>
<td>65</td>
<td>85</td>
<td>45</td>
</tr>
<tr>
<td>65</td>
<td>85</td>
<td>60</td>
</tr>
<tr>
<td>85</td>
<td>85</td>
<td>70</td>
</tr>
<tr>
<td>90</td>
<td>90</td>
<td>80</td>
</tr>
</tbody>
</table>

Finger Moving from Flexion to Extension

<table>
<thead>
<tr>
<th>If the DIP joint was at</th>
<th>PIP joint was at</th>
<th>MP joint was at</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Degrees)</td>
<td>(Degrees)</td>
<td>(Degrees)</td>
</tr>
<tr>
<td>90</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>90</td>
<td>90</td>
<td>80</td>
</tr>
<tr>
<td>90</td>
<td>90</td>
<td>70</td>
</tr>
<tr>
<td>85</td>
<td>75</td>
<td>60</td>
</tr>
<tr>
<td>70</td>
<td>70</td>
<td>60</td>
</tr>
<tr>
<td>40</td>
<td>65</td>
<td>50</td>
</tr>
<tr>
<td>40</td>
<td>60</td>
<td>35</td>
</tr>
<tr>
<td>15</td>
<td>20</td>
<td>22</td>
</tr>
<tr>
<td>23</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

complete extension, we then completely sectioned the two lateral bands just distal to the proximal interphalangeal joint, being very careful to spare the oblique retinacular ligaments on both sides of the finger. As soon as the lateral bands were sectioned, the distal phalanx immediately dropped into a flexed position of 70 degrees. The finger was then flexed and extended by alternating pull, first on the profundus, and then on the long and intrinsic extensors together. Extension at the metacarpophalangeal joint was normal. Extension at the proximal interphalangeal joint was normal. There was no functional extension at the distal interphalangeal joint. It remained at a flexed position of 70 degrees. This could be increased to 90 degrees when we pulled tightly on the profundus, fully flexing the finger.

In order to determine the relative flexion of the proximal interphalangeal and distal interphalangeal joints, as the fingers were increasingly flexed, high-speed movies were made of a normal hand flexing and extending. The results obtained in measuring these films are shown in Table I. Next the dissected fingers were carefully examined. These results are shown in Table II.

In unresisted flexion, it can be seen from these results that the greatest difference in angle between the proximal interphalangeal and distal interphalangeal joints

TABLE II

<table>
<thead>
<tr>
<th>If PIP joint was at</th>
<th>to tighten oblique retinacular ligament</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Degrees)</td>
<td>(Degrees)</td>
</tr>
<tr>
<td>0</td>
<td>70</td>
</tr>
<tr>
<td>30</td>
<td>The ligament would not tighten</td>
</tr>
<tr>
<td>60</td>
<td>The ligament would not tighten</td>
</tr>
<tr>
<td>90</td>
<td>The ligament would not tighten</td>
</tr>
</tbody>
</table>
occurred when the metacarpophalangeal joint was at 40 degrees of flexion. This difference is only 30 degrees. The oblique retinacular ligament was not tight when there was only this much angular difference in the position of the proximal interphalangeal and distal interphalangeal joints. Therefore, in unresisted flexion this ligament never becomes tight.

Similarly, in unresisted extension, the greatest angular difference between the distal interphalangeal and proximal interphalangeal joints which occurred was 25 degrees, and this occurred when the metacarpophalangeal joint was at 50 degrees. Again, the oblique retinacular ligament was not tight at this degree of angulation.

It was noted on repeated flexion and extension of the fingers without resistance, that the angles of the proximal interphalangeal and distal interphalangeal joints were not always the same relative to each other. In other words, measurement of the angles may give one set of values on one cycle of flexion and extension, and these may be different on the next cycle. However, they remained within a 10 to 20-degree range of the values shown in Tables I and II. The greatest difference between the angles of the proximal interphalangeal and distal interphalangeal joints never exceeded 35 degrees.

The normal hand was then tested in resisted extension, and it was noted that the relative angles of the proximal interphalangeal and distal interphalangeal joints remained remarkably constant, and the angles remained almost exactly the same at the two joints. The unresisted finger did not maintain this constancy.

Our conclusion was that, in the normal finger, in all positions which the normal hand can actively assume, extension of the distal phalanx is performed entirely by the two lateral bands. The oblique retinacular ligament must be considered a stay or retaining ligament maintaining tendon centralization on the dorsum of the finger, just as does its more proximal namesake, the transverse retinacular ligament. While it can be made to tighten by certain passive tests of the finger, it never tightens in normal finger function.

The Proximal Interphalangeal Joint

The extensor mechanism at the proximal interphalangeal joint should be thought of as a trifurcation of the extensor tendon into the central slip and the two lateral bands. The central slip of the extensor tendon inserts into the base of the middle phalanx, the two lateral bands pass on either side of the proximal interphalangeal joint and fuse distally to insert into the distal phalanx. The entire mechanism is held centered over the joint by the transverse retinacular ligaments, and is joined on either side by tendons of the lumbricals and interossei.

How does the extensor mechanism achieve simultaneous extension of the two finger joints? It does so by a mechanism in which the central slip extends the middle phalanx, and the two lateral bands bypass the proximal interphalangeal joint to join and extend the distal phalanx. The essence of this mechanism is that the three elements must be in balance. The lengths of the central slip and of the two lateral bands must be such that extension of the proximal interphalangeal and distal interphalangeal joints takes place together, so that when the middle phalanx is brought up into alignment with the proximal phalanx, the distal phalanx reaches alignment at the same instant. This precision depends on the lengths of the central slip and the two lateral bands. It has no relation to any retinacular system. It is this very precision of balanced lengths that is so difficult to restore when the mechanism has been deranged due to injury or disease.

Dorsal migration of the lateral bands, as the proximal interphalangeal joint extends, is generally attributed to the elastic quality of the triangular ligament, which is supposedly placed on a stretch in flexion. As the joint is extended, this elastic liga-
In the extended finger, the point of origin of the lateral bands is shown by the arrow. This point is proximal to the proximal interphalangeal joint. In the lower part of the figure, when the finger starts to flex, the pull of the flexors pulls the point of insertion below the long axis of the proximal phalanx. The lateral bands, being free to move, bowstring across the interval. 

In the extended finger, the point of origin of the lateral bands is shown by the arrow. This point is proximal to the proximal interphalangeal joint. In the lower part of the figure, when the finger starts to flex, the pull of the flexors pulls the point of insertion below the long axis of the proximal phalanx. The lateral bands, being free to move, bowstring across the interval.

In the extended finger, the point of origin of the lateral bands is shown by the arrow. This point is proximal to the proximal interphalangeal joint. In the lower part of the figure, when the finger starts to flex, the pull of the flexors pulls the point of insertion below the long axis of the proximal phalanx. The lateral bands, being free to move, bowstring across the interval.

ment is supposed to pull the lateral bands dorsally. Loss of this elastic tissue as a result of injury has been considered one of the main causes of difficulty in reconstruction, because there was no known way to replace it. In a similar manner, a tie-down by the fibers of the transverse retinacular ligament is supposed to pull the lateral bands ventralward when the proximal interphalangeal joint is flexed. We were particularly interested in determining, if possible, the basis for this migration of the lateral bands.

Method and Observations

Using a fresh hand, a very careful dissection was made of the finger. The triangular ligaments were cut. The lateral bands were then freed, both from the transverse retinacular ligaments and the oblique retinacular ligaments, and from their connection to the central slip of the extensor tendon. This dissection was carried proximally to the origin of the lateral bands from the extensor mechanism, one to two centimeters proximal to the proximal interphalangeal joint. In other words, a boutonnière lesion was created, without detaching the central slip.

Flexion and extension of the altered finger were then tested, by pulling first on the profundus, and then together on the long extensor and intrinsics. It was noted that extension and flexion of the finger were completely normal. The lateral bands shifted volarward in flexion, and migrated dorsally in extension, even though they were completely free throughout their length.

The reasons for this were then examined. In the extended finger, when the proximal interphalangeal joint begins to flex, the distal phalanx and the distal interphalangeal joint assume a position volar to the long axis of the proximal phalanx. They thus tend to pull the insertion of the lateral bands to a position volar to the proximal interphalangeal joint. These bands, being free to move, begin to take a more volar position. It is a simple act of bowstringing from the point of origin of the lateral bands in the extensor mechanism to their insertion on the distal phalanx. The
As flexion continues, the insertion is brought more and more volarward and the lateral bands become more volar in relation to the proximal interphalangeal joint.

lower the distal phalanx is brought beneath the long axis of the proximal phalanx by the contracting profundus tendon, the more the lateral bands will bowstring ventral to the proximal interphalangeal joint (Figs. 2 and 3). In bowstringing, they move toward the wider portion of the distal end of the proximal phalanx, and thus separate. As the flexion motion continues, the separation of the two lateral bands is wider.

Extension is just the reverse. Extension is initiated by the central slip which begins to bring the proximal interphalangeal joint into extension. At 10 degrees of extension of the metacarpophalangeal joint, extension of the proximal interphalangeal and distal interphalangeal joints begins. As extension continues, the central slip pulls the proximal interphalangeal joint into extension. The proximal shift of the extensor mechanism causes increased tightening of the lateral bands. The dorsal migration of the lateral bands is brought about by three factors:

1. The dorsal movement of their point of insertion produced by the central slip as it extends the middle phalanx. This brings their point of insertion closer to the line of the proximal phalanx, and thus back toward the line through their point of origin (Fig. 4).

2. The sloping sides of the end of the proximal phalanx. This facilitates the dorsal movement of the bands because they encounter no obstruction (Fig. 5).

3. The tendency of any linear structure to assume a straight line when tightened. The end of the proximal phalanx is narrower on the dorsal than on the palmar side, and the lateral bands, tight in attempted extension, tend to move toward the narrow portion of the phalanx.

They thus move not only dorsally, but they also move toward each other, so that they are closely approximated in extension. The tighter one pulls the finger into extension, the more closely the two lateral bands move toward each other (Fig. 6). This ingenious bypass mechanism is thus able to achieve the simultaneous extension of the distal interphalangeal joint as the central slip extends the proximal interphalangeal joint, but it can only do this if the system is perfectly balanced, and if extension of both joints takes place simultaneously. The dorsal migration of the extensor bands is a function of this mechanism, rather than any elastic quality of the triangular ligament.
Dorsal migration of the lateral bands is simply the reverse of flexion and is the result of extension of the middle phalanx. As the middle phalanx is extended by the central slip and moves from position $a$ to position $b$, the lateral bands are brought up with it and become dorsal to the proximal interphalangeal joint. The point of origin of the lateral bands is a relatively fixed point, moving back and forth only a few millimeters in flexion and extension. It is the distal insertion which moves up and down and causes the dorsal and volar migration of the lateral bands.

The palmar migration of the extensor bands in flexion does not result from any tie-down by the transverse retinacular ligaments. It is a simple mechanical result of flexion of the proximal interphalangeal joint. The shape of the distal end of the proximal phalanx, which is wider in the volar than in the dorsal aspect, causes the bands to separate in flexion.

The proximal interphalangeal joint, flexed to 90 degrees and seen end-on with the central slip inserting on the base of the middle phalanx. As the proximal interphalangeal joint goes into extension, the lateral bands move dorsally. Due to the slope of the distal end of the proximal phalanx they can move toward each other as they move dorsally. In flexion, they separate.

The Metacarpophalangeal Joint

Extension of the metacarpophalangeal joint has been the subject of controversy in recent years. Baumann and Patry, 1943, denied that there was an extensor slip which inserts on the base of the proximal phalanx. Sunderland, in 1945, stated “The anatomical features of this slip have been carefully examined in forty dissected specimens. It is a relatively loosely arranged band of weak fibres in the majority of cases.
The migration of the lateral bands toward each other is a result of this simple mechanism. In the figure on the left the bands are separated; in the center figure, with proximal pull on the extensor, the bands move toward each other; in the figure on the right, the more strongly the finger is extended, the closer the bands move together.

. . . On no occasion was a strong slip observed in this position.” Haines, in 1951, stated, “In most individuals . . . the slip is strong and well-defined . . .”

Methods and Observations

We were particularly interested in determining the answer to this question, “Is there an insertion of the extensor tendon into the base of the proximal phalanx?” In our dissections there definitely was a strong fibrous insertion of the extensor mechanism into the capsule of the metacarpophalangeal joint, and through it into the base of the proximal phalanx. It does not insert, however, by Sharpey’s fibers as does the central slip at the proximal interphalangeal joint. We have also observed that this slip is functionless in all positions of the metacarpophalangeal joint except hyperextension. Just as extension at the proximal interphalangeal joint occurs because of the interaction of several structures, so does extension of the metacarpophalangeal joint. The interesting difference here is that the situation exactly reversed that of the proximal interphalangeal joint. The primary extensor of the metacarpophalangeal joint is not the slip from the extensor tendon but rather the encircling series of fibers which connect the extensor mechanism to the flexor sheath. These may be secondarily assisted by the central slip at the base of the middle phalanx, but this slip is not
The primary extensor of the metacarpophalangeal joint is the encircling series of fibers which connect the extensor mechanism to the flexor sheath. When the finger is in complete extension, the proximal slip is under no tension.

essential because the proximal phalanx still extends normally after section of the central slip inserting on the middle phalanx (Fig. 7).

In the normal range of motion of the finger, that is from 0 to 90 degrees of flexion of the metacarpophalangeal joint, the slip inserting into the base of the proximal phalanx is completely lax and has no function. This central slip only becomes tight in a position of extreme hyperextension of the metacarpophalangeal joint (Fig. 8).

From a theoretical standpoint, this laxity of the proximal slip is essential to normal finger function, for if there were a tight insertion of this slip, thus binding the extensor mechanism to the base of the proximal phalanx, the extensor expansion would be check-reined, and could, therefore, have no action on the interphalangeal joints.

Within the range of normal finger function, this slip never becomes tight, but simply swings to and fro with the movements of the extensor mechanism. Thus, there is no check-rein effect, and the long extensor can exert its full power on the interphalangeal joints. Haines commented on this phenomenon in 1951, when he stated, with regard to this proximal slip, "since it is slack in all positions of the proximal

The primary extensor of the metacarpophalangeal joint is the encircling series of fibers which connect the extensor mechanism to the flexor sheath. When the finger is in complete extension, the proximal slip is under no tension.

The proximal slip of the extensor mechanism is lax even in complete finger extension as shown in the upper figure. When the finger is flexed, it becomes even more lax. Movement of the finger from flexion to extension is not assisted in any way by this slip. This slip must be lax for the finger to function normally. It is only tight in full hyperextension of the metacarpophalangeal joint.
joint except in full hyperextension its presence does not normally interfere with the action of the common extensor on the more distal joints."

In a normal finger, we sectioned the proximal slip inserting into the base of the proximal phalanx. The function of the finger was then examined, and it was found that the finger extended normally, and if no interossei or lumbricals were used, the long extensor would still simply hyperextend the metacarpophalangeal joint. This, of course, is due to the fact that the encircling fibers around the proximal phalanx represent the primary extensor of the proximal phalanx.

When the central slip at the metacarpophalangeal joint is left intact in a normal hand, and the metacarpophalangeal joints hyperextended by very forcible contraction of the long extensor muscles, the interphalangeal joints can then be flexed and extended by the intrinsics only.

So long as the metacarpophalangeal joints remain in a position of extreme hyperextension, the long extensors no longer participate in interphalangeal extension. The proximal excursion of the extensor mechanism has gone to its maximum, and is check-reined by the central slip inserting onto the base of the proximal phalanx.

If the metacarpophalangeal joints are held in this position, and the finger is flexed and extended, flexion is by the profundus tendon and extension of the interphalangeal joints is done entirely by the intrinsics.

**Discussion**

*The Distal Interphalangeal Joint*

Our investigations indicate that in flexion and extension in the normal hand, the oblique retinacular ligament never becomes tight and does not participate in function of the normal finger.

Contracture of the oblique retinacular ligament may occur in several different conditions. One of these is Dupuytren's contracture of the palmar fascia, which is occasionally accompanied by fibrous thickening and contracture of other connective-tissue elements in the hand. It may also be seen in certain instances after burns or traumatic injury of the fingers, which have resulted in skin and deep structure contracture. In such instances, the test for contracture of the oblique retinacular ligament will be positive, that is, flexion of the proximal interphalangeal joint allows full flexion of the distal interphalangeal joint, but when the proximal interphalangeal joint is brought up into passive extension, then the distal interphalangeal joint goes into fixed extension due to the tenodesis effect of the contracted oblique retinacular ligament. In such cases it should be noted that when the proximal interphalangeal joint is at 0 degrees, no distal interphalangeal flexion is possible.

Surgical procedures which artificially produce a tenodesis of the extensor mechanism to the flexor tendon sheath at the level of the proximal phalanx have been described, and used effectively in certain instances. It should be emphasized, however, that these procedures do not duplicate any normal function of the oblique retinacular ligament. Although they can be made to work, they are based on a misconception, namely, that the oblique ligament extends the distal phalanx. In the normal hand it does not do this.

*The Proximal Interphalangeal Joint*

Our investigation has shown that the dorsal and volar migration of the lateral bands has a simple mechanical basis. It is not in any way dependent on the pull either of the triangular ligaments dorsally or the transverse retinacular ligaments ventrally. In any reconstructive procedure aimed at correcting extensor imbalance, it is essential that the lateral bands be left free to move and not subjected to any surgical tie-down either on their dorsal or volar aspects.
Our investigations necessitate a reassessment of our thinking of the boutonnière deformity. It must be thought of as consisting of two components: a disruption of the attachment of the central slip and a buttonhole rupture through the roof. Disruption of the central slip alone will not produce the deformity as long as the roof of the extensor mechanism is intact. (See the Fowler procedure for old mallet-finger deformity.) Similarly, buttonholing of the roof will not produce the deformity if the central slip and lateral bands are intact, as shown by our investigations.

Detachment of the central slip at the proximal interphalangeal joint does not, of itself, produce a boutonnière deformity nor does it cause significant loss of extension of the proximal interphalangeal joint. In treating fresh boutonnière injuries, therefore, we can splint the proximal interphalangeal joint in extension, leaving the distal interphalangeal joint free to move, and if we get healing of the roof of the extensor mechanism, we will get back finger extension and correction of the boutonnière deformity, even though the central slip may not reattach. If the central slip does reattach, so much the better.

These findings indicate that in the surgical repair of old boutonnière deformity, we should not disturb the two lateral bands. These remnants of the extensor system are normal and should not be disturbed. Our aim should be to reattach the central slip to the base of the middle phalanx. If direct suture is possible, according to the technique of Elliott, this should be utilized, being sure that the tension is correct to extend both proximal interphalangeal and distal interphalangeal joints simultaneously.

If direct suture is impossible, it is necessary that we employ a tendon graft. This should be passed through a tunnel made in the base of the middle phalanx, and imbricated into the central slip and central tendon proximally, according to the technique of H. Minor Nichols. The graft should, under no circumstances, be woven into the lateral band mechanism, which must be left free and unfettered to extend the distal phalanx. The tension must be tested and adjusted so extension of the proximal interphalangeal and distal interphalangeal joints takes place simultaneously.

Surgical procedures have been described which weave the lateral bands together over the proximal interphalangeal joint. These are mentioned here only to be condemned because they are unphysiological and, if done, will not permit flexion of the proximal interphalangeal joint. The only indication for disturbing the lateral bands is to free them from a contracted transverse retinacular ligament, or a contracted triangular ligament. In such a case, they should be freed from these ligaments so that the proximal interphalangeal joint can be flexed and extended normally.

It has been noted that when the proximal interphalangeal joint hyperextends, there is some dorsal bowstringing of the lateral bands. When the joint is in hyperextension, the lateral bands can short-cut across the joint. This gives some slack in the distal insertion. Some people, who have normal extensor mechanisms, but whose fingers are lax enough to permit hyperextension at the proximal interphalangeal joint, can lock the proximal interphalangeal joint in hyperextension. The lateral bands then bowstring, which gives a few millimeters of slack at the distal joint and by selective contraction of the profundus, these individuals can produce the swan-neck deformity. This is a simulated imbalance of the extensor mechanism.

In old mallet finger, balance is lost when the distal tendon heals in a lengthened condition. Attempts by the patient to extend the distal phalanx only result in hyperextension of the middle joint. Balance can be restored either by tightening up the distal insertion or by section of the central slip allowing a more proximal migration of the extensor mechanism, thus extending the distal phalanx (Fowler procedure). It could be argued, perhaps, that tightening of the distal insertion is the more physiological approach. From a surgical standpoint, however, it may be technically easier to
rebalance the finger by the Fowler procedure. Mallet finger is thus a true imbalance of the extensor mechanism.

Laxity of the volar plate, and loss of the superficialis tendon produce a similar deformity, but in these cases there is no imbalance of the extensor mechanism; there is loss of the normal flexor forces on the joint. In these cases, the extensor system is normal. Treatment must be directed toward preventing hyperextension of the proximal interphalangeal joint.

Therefore, the swan-neck deformity may come about from different causes, and just as the causes are different, so must the treatment be. The two types of deformity are easily distinguished. In the volar plate and superficialis tendon type of swan-neck deformity, if the proximal interphalangeal joint is prevented from hyperextending, the extensor mechanism can completely extend the distal joint. This is not true in the mallet finger type of deformity.

One further point deserves mention. The insertion of the interossei and lumbricals into the extensor mechanism makes them participants, with the long extensor, in extension of the interphalangeal joints. They insert both into the central slip and into the lateral bands. Many investigators have shown that complete extension of the interphalangeal joints can be done either by the intrinsics alone or the long extensor acting alone, so long as the metacarpophalangeal joint is not allowed to go into hyperextension. The fact that the long extensor, acting alone, can completely extend the interphalangeal joints of the finger in any position of the metacarpophalangeal joint from 0 degrees to 90 degrees, shows that the function of the long extensor is not dependent on any lateral pull of the intrinsics at the proximal interphalangeal joint level. The lateral pull of the intrinsics at the proximal interphalangeal joint, as described by some investigators, is not supported by experimental evidence. If this lateral pull were essential to finger extension, the long extensor, acting alone, could not completely extend all joints of the finger.

In point of fact, the more strongly the finger is extended, the closer the bands move together, rather than laterally.

Extension of the proximal interphalangeal joint is achieved by the central slip of the extensor mechanism. This may be considered the primary extensor at this joint. Experiments have shown, however, that this is not the only extensor. If the transverse retinacular ligament is sectioned and the hood lifted up and the central slip sectioned without buttonholing the roof, which incidentally is Fowler's operation, the middle phalanx still extends. This extension is achieved by a secondary mechanism of encircling fibers which connect the extensor hood to the flexor sheath. These fibers completely encircle the finger, and will extend the middle phalanx with no help from the central slip. It should be noted, however, that with section of the central slip the joint has been deprived of its primary extensor, and a very slight finger imbalance may occur. This may be manifested by a slight drooping of the middle phalanx of 10 to 15 degrees.

One further observation should be emphasized. When the proximal interphalangeal joint is strongly flexed with the superficialis alone, it is noted that the distal phalanx becomes completely free and cannot be extended. This has been attributed by some investigators to the volar displacement of the lateral bands. Our investigations have shown that when the central slip is sectioned in the Fowler procedure, the extensor mechanism can completely extend the distal phalanx in any position of the proximal interphalangeal joint.

It is apparent that the complete loss of the ability to extend the distal phalanx, when the proximal interphalangeal joint is flexed to 90 degrees, is due solely to the fact that the extensor mechanism is held distally by the central slip and thus check-reined. The proximal pull on the extensor mechanism has no action because the cen-
In our opinion these facts have an important application in the treatment of fresh mallet finger. We feel that the proximal interphalangeal joint should be splinted in flexion, because the central slip will thus pull the extensor mechanism distally, and check-rein any tension effects of the tendon at the distal interphalangeal joint level where tendon healing is being sought. Likewise, the distal interphalangeal joint should be splinted in extension. We do not feel that in the treatment of fresh mallet finger the proximal interphalangeal joint should be left free. It should be splinted in flexion.

The Metacarpophalangeal Joint

In cases of rheumatoid arthritis with severe flexion deformity of the metacarpophalangeal joints, it has been suggested that a tight artificial insertion of the extensor mechanism to the base of the proximal phalanx be used to improve extension of the metacarpophalangeal joint. Such a procedure must be approached with a great degree of caution. If the extensor mechanism is anchored to the base of the proximal phalanx with the finger in extension, then finger flexion will no longer be possible. If the extensor is anchored with the finger in flexion, all action of the long extensor on the interphalangeal joints is thus eliminated. Interphalangeal extension must henceforth be done entirely by the intrinsics, which must of necessity be normal in both innervation and insertion. (In other words, they cannot have been altered by injury or disease.)

After such a procedure, if the intrinsics are not normal, interphalangeal extension of the fingers will no longer be possible.

In conclusion, a few general observations should be made on the balance of the extensor mechanism. The concept of balance has been well defined by Fowler, and need not be gone into in any detail here, except to restate that at each joint of the finger there must be an extensor and a flexor. If either of these is lost, either due to injury, disease, or surgery, then a predictable deformity will develop.

If the extensor mechanism exerts an excessive pull at one joint, there will usually be a reciprocal loss of pull at one or more of the other joints (swan-neck deformity). Similarly, loss of pull at one joint may result in excessive pull at another joint (old mallet finger).

Whenever the extended finger is not in a straight line, the finger is shorter, and then becomes a collapsing segmented column which will assume various angular deformities, depending on the level and direction of the imbalance.

Studies of the kinesiology of the human hand reveal that the least number of muscles contract which will do the job in question. Thus, when the hand is alternately opened and closed, only the lumbricals are used with the long extensors to achieve finger extension. This has been shown by the electromyographic studies of Long and Brown. This is simply an expression of the economy of nature, which always attempts to achieve the maximum effect with the least effort. It does not mean in any sense that the interossei are not powerful extensors of the interphalangeal joints. It simply means that they were not needed for the particular movements involved. The interossei contract powerfully in resisted extension.

As has been previously stated, the control of minute and discriminatory movements of the human fingers is the result of a complex interrelationship between the intrinsics and the long flexors and extensors, under the control of the motor cortex and cerebellum. Thus, in the playing of a musical instrument, a particular lateral movement of a finger may require a certain tension on the profundus, an isometric tightening of the superficialis, the introduction of a slight contracture of a lumbrical,
CRAMPTON HARRIS, JR., AND G. L. RUTLEDGE, JR.

a more powerful contraction of a volar interosseus, and also a tightening of the long extensor and one or more dorsal interossei. All this is to achieve simply one particular precise movement. All of these things are done, of course, entirely without our conscious coordination.

In the normal hand, this balance is maintained by the cerebellum and motor cortex. Any needed impulses to a lumbrical, the superficialis, the long extensor, or to an interosseous muscle, are supplied entirely automatically and finger balance is maintained. When this wonderfully integrated system is damaged, as in cerebral palsy or other forms of brain or nerve injury, then all the various deformities and imbalances with which we are familiar can develop.

Summary

1. Our investigations indicate that the oblique retinacular ligament does not extend the distal phalanx in the normal hand. It, therefore, need not be reconstructed in attempting to restore distal interphalangeal extension. Our efforts should, in general, be directed toward a restoration of the lateral band mechanism.

2. Dorsal and volar migration of the lateral bands at the proximal interphalangeal joint level is not dependent on the triangular ligament or the transverse retinacular ligaments. It is based on simple mechanical principles, and will take place even though the lateral bands are completely free throughout their length.

In the surgical repair of old boutonnière deformities, our efforts should be directed toward re-establishment of the central slip. The lateral bands, which are normal, should not be disturbed.

3. The central slip at the base of the proximal phalanx has no extensor function in the normal finger, except when the metacarpophalangeal joint is in extreme hyperextension. We must not, therefore, fix this mechanism rigidly to the base of the proximal phalanx by surgically produced insertions.

References


