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An in-depth look at zone III and IV anatomy of the finger extensor mechanism and some clinical implications for use of the relative motion flexion orthosis

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A B S T R A C T

Background: For hand therapists and hand surgeons acute and chronic injuries of the extensor mechanism (EM) in zones III-IV are challenging to treat with satisfying results.
Introduction: Early active motion combined with relative motion flexion (RMF) orthoses to manage EM zone III injuries and boutonnière deformity has renewed interest in the complex anatomy and biomechanics of the EM.
Purpose: To provide an in-depth discussion of EM zones III-IV anatomy with emphasis on inter-tendinous structures, often omitted in simplified, model-wise illustrations which focus mostly on the tendinous structures.
Method: In collaboration the authors combined on the one hand extensive clinical experience and knowledge of the EM literature and on the other hand decades of anatomical, biomechanical and kinesiology research of the EM with special interest for the spiral fibers, through gross anatomy and microdissection anatomy laboratory work, MRI and ultrasonography studies.
Results: The inter-tendinous tissues (i.e., spiral fibers) in zone III are of imminent importance for proper functioning of the EM and to prevent boutonnière deformity to develop after EM surgery or injury.
Discussion: Inter-tendinous links between the tendinous structures of the EM are necessary for balanced finger motion. The spiral fibers are described in more detail because of their role in controlling volar migration of the conjoined lateral bands and because their disruption makes development of boutonnière deformity more likely. Understanding the anatomy and biomechanics of the EM may assist in progress toward 'proof of concept' for use of RMF orthoses and controlled early active motion after EM injury or surgery.
Conclusion: Hand surgery and hand therapy practice interventions, including use of RMF orthoses for management of non-surgical and surgical EM injuries may benefit from an in-depth look at the EM zone III and IV anatomy and biomechanics.

The extensor mechanism is the term used by the authors while others have referred to it as the extensor hood or extensor apparatus. All terms refer to the weave of tendons, bands, fibers and ligaments that cover the dorsum of each finger from the metacarpophalangeal joint (MCPJ) proximally to the distal interphalangeal joint (DIPJ) distally.

Throughout this text the terms lateral and medial bundles, bands and slips will be used interchangeably and always refer to the three tendinous structures into which the extensor digitorum (ED) tendon splits.

Volar or palmar glide/migration/slide all refer to the shifting of the conjoined lateral bands at the proximal interphalangeal joint (PIPJ) and in this article will be used interchangeably.

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Introduction

> "*It is misleading to speak of isolated, individual and specific actions of the extensor digitorum, lumbricals and interossei muscles. They function as well integrated and coordinated groups in every movement of the digits" Sunderland 1945*[1]

What is fascinating about the extensor mechanism (EM) is that it is composed of fibers from many muscle and tendon systems woven together into one fabric. Even more fascinating is how this complexly woven fabric coordinates finger extension and flexion. When one starts reading on the anatomy and function of the EM, it becomes clear that many little known details have been overlooked in the hand therapy- and hand surgery literature.

This article will provide an overview of extensor zones III-IV EM anatomy and kinesiology, in particular details of zone III, which we believe have been under- or misreported. We have combined more recent imaging findings with sometimes century old morphological publications to unravel and weave together how we believe the EM in zone III functions both normally and pathologically after proximal interphalangeal joint (PIPJ) injury or surgery. We have applied this basic science to the use of the relative motion flexion (RMF) orthosis to restore normal balance of finger extension after a zone III injury, surgery or any loss of *active* PIPJ extension.

Extensor mechanism zones III-IV anatomy

In zones III-IV, the EM functions as an extensor of the PIPJ and distal interphalangeal joint (DIPJ) through two systems, the extrinsic and intrinsic muscles. The extrinsic finger extensors are the extensor digitorum (ED), extensor indicis (EI), and extensor digiti minimi (EDM). The contributions from the intrinsic muscles are the palmar and dorsal interossei and the lumbricals. Even with the big differences in muscle type and length they work together flawlessly within the extensor mechanism to accomplish interphalangeal joint (IPJ) extension and flexion.[2]

The EM consists of three distinct types of structures (1) tendinous, originating from muscle and inserting into bone (2) intertendinous, connecting two tendons or tendon systems, and (3) retinacular/ligamentous, originating from bone, fibrous and subcutaneous tissues, and inserting into bone or tendon. Landsmeer (personal communication, 1972) moreover identified three key features of the extensor mechanism: (i). the fiber layers, (ii). formation of various fiber bundles, and (iii). spiralization within fibers, fiber bundles, and tendons. The three types of structures and the three key features as indicated by Landsmeer will be discussed in the description of the zone III and IV anatomy in the following sections.

Extrinsic contribution to the extensor mechanism

The proximal edge of the EM starts from the ED tendon at the level of the metacarpophalangeal joint (MCPJ). The ED has two 'insertions' at this proximal edge: (1) through the sagittal bands that run laterally and in a volar direction at the MCPJ inserting into the volar plate forming a loop around the proximal end of the proximal phalanx, which functions to pull the MCPJ into (hyper) extension, and (2) a small slip which may not always be present[3,4] from the volar aspect of the ED at the MCPJ to insert at the dorsal side of the base of the proximal phalanx. Both insertions function to limit proximal and distal glide of the ED and thus the EM.

Over the distal one-third of the proximal phalanx the ED tendon fibers are rearranged into three tendinous bundles: a medial band and two lateral bands. The medial band passes the PIPJ dorsally and inserts on the base of the middle phalanx as the central slip (CS). There is sometimes a sesamoid fibrocartilage located on the volar surface of the medial band where it passes over the PIPJ.[5,6,7]

At the distal one-third of the proximal phalanx the lateral bands merge on each side with the intrinsic tendon and converge into the conjoined lateral bands (CLBs) that run on each side of the distal-dorsal end of the proximal phalanx. These CLBs continue distally past the PIPJ where they converge into one tendon as the terminal tendon (TT), which runs over the dorsum of the middle phalanx and inserts on the base of the distal phalanx. Even though the description of the three distinct fiber bundles proximal to the PIPJ is accurate, this is a simplification of the EM in zones III-IV. Kaplan[8] (1965) provided a more accurate description of the EM in these zones as "a continuous fanlike structure with a more or less condensed central portion and spread-out lateral bands. In fact the extensor tendon system actually spreads out over the entire dorsal capsule of the PIP joint."

If a diagnosis is made indicating an extensor "*tendon*" laceration at the PIPJ this would, more accurately, be a partial laceration of the EM, as there is not one tendon but a continuous fanlike structure according to Kaplan. Therefore an injury in this area should be considered as not only an injury of one or more of the tendon bundles but also of the inter-tendinous fabric of tissues *connecting* the three bundles. Importantly, in this article, we will discuss how injuries to tissues interconnecting tendon bundles and slips are more important contributors in the development of boutonnière deformities than an isolated laceration of a lateral band or the CS.[9]

Intrinsic contribution to the EM

The tendons originating from the intrinsic muscles, i.e., the dorsal and palmar interosseus and the lumbrical (radial side only), merge to form the intrinsic tendons, sometimes called wing tendons, just distal to the transverse metacarpal ligament near the MCPJ. The lumbrical tendon on the radial side runs volar to the transverse metacarpal ligament and the interosseus tendons, on the radial and ulnar sides, run dorsal to the transverse metacarpal ligament and lateral to the sagittal bands. On both sides, after the interosseus (palmar and dorsal) and lumbrical (only the radial side) merge they form the intrinsic tendon that runs distally and laterally along the proximal phalanx to form the borders of the triangular-shaped EM overlying the proximal phalanx[†].

Sometimes the intrinsic tendon is said to split into two tendon slips at the distal half of the proximal phalanx with a lateral slip continuing distally and laterally along the PIPJ and a "medial slip" connecting dorsally with the medial band of the CS. This "medial slip", often seen in illustrations (Fig. 1)[10], may have started because of a simplification (model-wise representation) of the intrinsic contributions to the EM into tendon slips and bands, but this is incorrect. There are in fact fibers originating from the medial interosseus tendon fibers of the intrinsic tendons, however, this occurs along most of the length of the proximal phalanx thus forming not a tendon or tendon slip but a thin lamina of fibers, which are known as the transverse and oblique tendon fibers.[2,11,12] (Fig. 2). Infantolino et al.[13] describe a spiraling of fibers coming from within interosseus muscle bellies, which manifest as a fan of fibers after splitting off the intrinsic tendon. The direction of fanning is from proximal to distal with increasing pitch,[14] beginning transverse in orientation to more oblique distally. The fibers run dorsally and attach firmly to the medial band of the ED. These

[†] As the focus of this article is anatomy of zones III-IV, the authors have chosen not to detail the insertions of the palmar and dorsal interosseus at the base of the proximal phalanx.

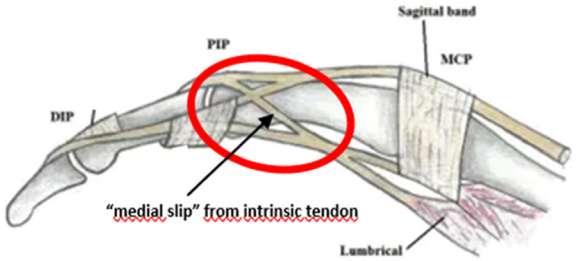

Fig. 1. This is a simplified representation of the extensor mechanism (EM). Notice that in this picture the intrinsic tendon is shown to split into a lateral and medial slip. Anatomically, the intrinsic tendon does continue laterally to meet the lateral band of the extensor digitorum and form the conjoined lateral band, however, medial fibers of the intrinsic tendon do not form a band or slip but are the transverse and oblique fibers that form a tendinous lamina overlying most of the proximal phalanx. (adapted from fig. 2B, page 261 in Skinner & Isaacs[10]).

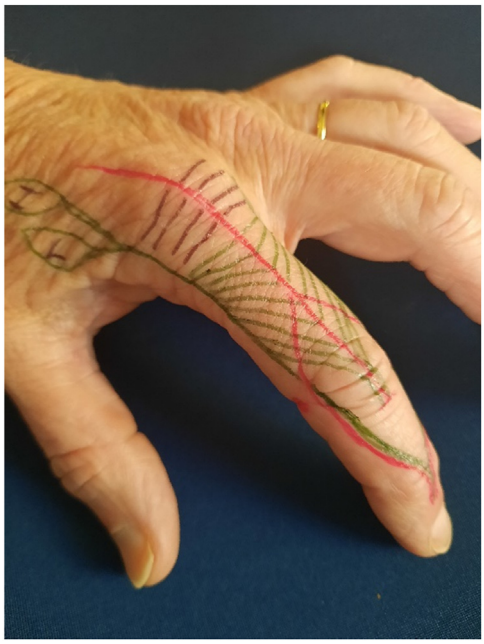

Fig. 2. Schematic depiction, drawn on the index finger, of the extrinsic contribution of the extensor mechanism in red and the intrinsic contribution in green. The sagittal bands are seen in black. (© 2021 anatomy workshop by G van Strien). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).

fibers form a triangular or wing shaped tendinous sheet that covers two-thirds of the proximal phalanx.

A lesser known feature of the EM is its layering in zones III-IV as mentioned by Landsmeer more than a half a century ago (personal communication, 1972). The medial interosseus fibers that form the transverse and oblique fibers over the dorsum of the proximal phalanx consists of two layers. First there is a palmar layer (mip), extending over the proximal one-third of the proximal phalanx, intertwining with the fibers of the ED tendon. Secondly there is a dorsal layer (mid) where the medial interosseus and lumbrical fibers cross over the lateral band of the ED, creating a phenomenon of intercrossing at about the middle of the proximal phalanx.[14-21] After crossing the lateral band, the dorsal (intrinsic) layer joins the (extrinsic) medial band of the ED which continues distally to insert on the proximal end of the middle phalanx as the CS[2], which importantly, results in the CS being a mixture of extrinsic and intrinsic tendon fibers. The layering of the EM has since been described by van Zwieten,[16] in 1980, and van Zwieten

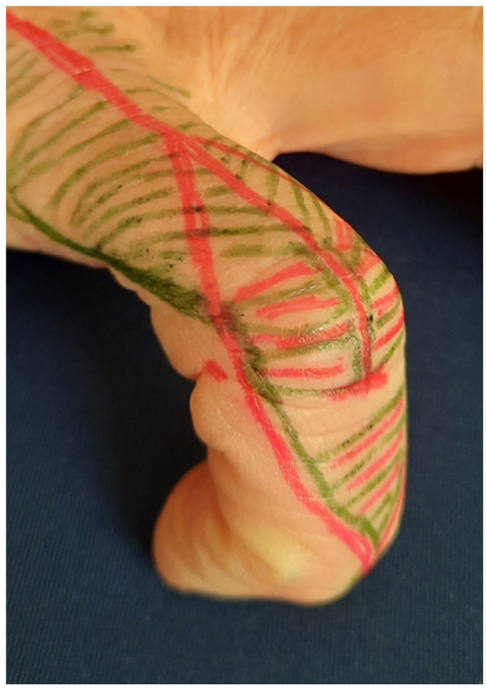

Fig. 3. Schematic depiction of the extensor mechanism "mixed zone", drawn on the index finger positioned in proximal interphalangeal phalanx joint and distal interphalangeal joint flexion. Structures originating from the extrinsic extensor (extrinsic fibers) are drawn in red. Structures originating from the intrinsic muscles (intrinsic fibers) are drawn in green. The conjoined lateral bands (CLBs) are formed by both intrinsic and extrinsic fibers through the merging of the intrinsic tendon and the lateral band of the extensor digitorum and are therefore drawn in both red and green. Both spiral fibers and triangular lamina originate from CLBs and have both extrinsic and intrinsic fibers and are drawn in red and green. (© 2021 anatomy workshop by G van Strien) (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).

and Lauw[17] in 1985 and confirmed by Linscheid[22] in 2002 who describes how the intrinsic 'wing' tendon envelops the ED lateral tendon slips with a dorsal and palmar layer before it merges with the medial bundle to form the central slip, making it here a structure of three layers.

Clearly the *anatomical* literature has described the multiple layers of the EM, however to our knowledge, it has not been described in the hand surgery or therapy literature. The implications clinically for this zone IV EM layering corresponds with the complications of adhesion formation following extensor zone IV repair and proximal phalanx fracture[23] and stresses the importance of implementing early active motion after injury/repair to lessen the complications and to support gliding of this multilayered structure.

The mixed zone: Parts of the EM that have origins from both intrinsic and extrinsic fibers

On the ulnar side of the digit the lateral interosseus fibers of the intrinsic tendon merge with the ED lateral bands to form the ulnar CLB, and on the radial side the lateral interosseus fibers first blend with lumbrical fibers, before merging with the ED lateral band over the distal third of the proximal phalanx to form the radial CLB. The merging of the extrinsic and intrinsic fibers to form the CLBs makes this a mixed zone (Fig. 3). Distal to the PIPJ the radial and ulnar CLBs merge to form the TT which then inserts at the base of the distal phalanx. Both CLBs are transversely connected by a triangular lamina over the middle phalanx, commonly referred to as the triangular ligament, however, since it is an inter-tendinous structure, we believe the term 'lamina' is better suited.

Similar to the triangular lamina, however located just proximal to the triangular lamina and located over the PIPJ, there is a transverse/oblique connection between the CLBs and CS formed by the spiral fibers.[11,24-26] These spiral fibers originate from the lateral margin of the CLB, spiral outwards and back over the CLBs (hence the spiraling name), thereby running over the CLB's dorsal surface in a medial and distal direction. The SFs run oblique on each side and meet in the middle firmly fixed to the CS, as two flat opposing dense tendinous structures, forming a distinct U-shape. The SF layer appears much denser than the intrinsic oblique and transverse tendon fiber layers. And contrary to the intrinsic oblique tendon fibers, the more distal SFs become gradually less oblique, as the CLBs converge to form the terminal tendon, and become even transverse in flexion when the U-shape is pulled around the head of the proximal phalanx and over the dorsum of the base of the middle phalanx in full PIPJ flexion. The SFs form a tendinous sheet that bridges the gap between the CLBs and the CS. The most distal SFs are covered dorsally by a layer of fibers that are a continuation of the fibers of the transverse retinacular ligament. Not only do the SFs spiral around the CLBs, the fibers themselves also possess a spiraling characteristic, which is thought to be acquired from the CLBs. The effect may also be caused by the intrinsic fibers with their spiraling course[13] that merge with the ED lateral band to form the CLBs. Although the SFs are a direct distal continuation (morphologically) of the intrinsic medial interosseus and lumbrical fibers (intrinsic tendon) they also intermingle with extrinsic fibers of the ED lateral bands as they have their origin from the mixed CLBs.[16,17] Hence, the SFs are different than the oblique and transverse fibers, which are of purely intrinsic origin and have no connection with the CLBs.

While less often discussed in clinical publications, for more than a century the SFs have been acknowledged by various authors.[11,24,27-29] Already in 1918 Seifert[30] noticed that "from the aponeurosis at the level of the P.I.P.- joint, narrow tendon tissue runs in proximal direction to the middle of the lateral edges of the [conjoined] lateral bundles." Seifert further concluded that the fibers functioned as a restraining mechanism against lateral sliding of the lateral bundles.[30] Later, Hauck[11] (1923) portrayed the location and direction of "narrow tendon tissues" in a clear line drawing and characterized them as spiraling radiations ("spiral fibers") from the [conjoined] LBs towards the medial band. He too suggested that these spiraling radiations acted as a restraining mechanism against lateral and downward (palmar) gliding of the [conjoined] LBs.[11] Over the years the SFs have been infrequently mentioned, although often depicted in illustrations,[10,31-33] sometimes aberrantly directed[34-36] or even wrongly located.[12] Furthermore, it has not been unusual that SFs are incompletely described[37,38] or absent in the anatomical, surgical, and biomechanical literature. In direct contrast, there are scientists and clinicians, who have acknowledged SFs through their basic and applied research, in the areas of anatomy[29,39], biomechanics[22,40-42], clinical practice[27], and surgery.[27,33,43,44]

The failure to mention the SFs in [some of] the clinical literature does not indicate the SFs do not exist or do not have a function as documented in the aforementioned publications. For years, there has been a general lack of interest in the tissues that span between the tendinous parts of the EM. Illustrations of the EM are often simplified drawings that focus on tendons and tendon slips and bands, sometimes indicating an anatomically non-existing tendon slip in model-wise representations as seen in Figure 1, where a more accurate view would be a tendinous sheet connecting the tendinous parts (i.e., the oblique and transverse fibers). It is the connecting inter-tendinous fibers and tissues which allow the EM to function properly. Notably the SFs, controlling volar migration of the CLBs, may prevent collapse of the system eventually leading to a boutonnière deformity. Therefore we believe the SFs are worthy of more attention as they may be key to obtaining more satisfying results in our management of PIPJ injuries.

Functional anatomy

To understand the functioning of the EM in zone III, it is important to review what is currently understood about normal coordinated movement between the PIPJ and DIPJ. The CS, aside from assisting in PIPJ extension, assists coordinated movement between the IPJs. As the PIPJ flexes, the intact CS via the medial band, pulls the EM distally thereby creating less tension in the LBs of the ED and therefore in the CLBs and subsequently the TT, permitting simultaneous PIPJ and DIPJ flexion.[11,12] Less tension of the LBs in turn allows the CLBs to migrate more lateral/volar at the PIPJ. This lateral/ volar shift of the CLBs during flexion of the PIPJ was described by Hauck[11] in 1923 and confirmed by Bunnell[12] in 1942 who aptly stated, that the "short cut" along the sides of the PIPJ is needed because the limited excursion of the [conjoined] lateral bands would otherwise not allow for simultaneous flexion of both IPJs.[11,12] Volar migration of the CLBs during PIPJ flexion has been demonstrated in normal subjects by van Zwieten et al.[21] using high resolution ultrasonography. These scientists found that even more CLB volar migration was needed for simultaneous PIPJ *and* DIPJ flexion, but never found the CLBs going beyond the axis of PIPJ rotation.[21] While the flat ribbon-like CLBs slide from a relatively dorsal to a more volar position following the contours of the head of the proximal phalanx they turn or tilt inwards. As the lateral edge becomes more volar it will tension the SFs that originate from the lateral edge and spiral around in the opposite direction dorsally towards the medial band.

Given their location the SFs function as retaining structures for the CLBs during finger flexion[21,29] (Fig. 4). The SFs along with the EM are pulled distally by the CS, and as the SFs are pulled distally, they end up as a U-shaped sling around the head of the proximal phalanx.[16] Because the SFs are pulled around the condyles of the proximal phalanx which are palmarly slightly diverging they become taut between the CLBs thus retaining the CLBs in their relatively dorsal position.[16,27] In our experience, this distal glide of EM and tension of the SFs around the condylar head of the phalanx during finger flexion is not typically shown in anatomical drawings, except for example, Tubiana[27] in 1986 and Tubiana et al.[28] in 1996, as most drawings of the finger with overlying EM are in an (almost) extended position.

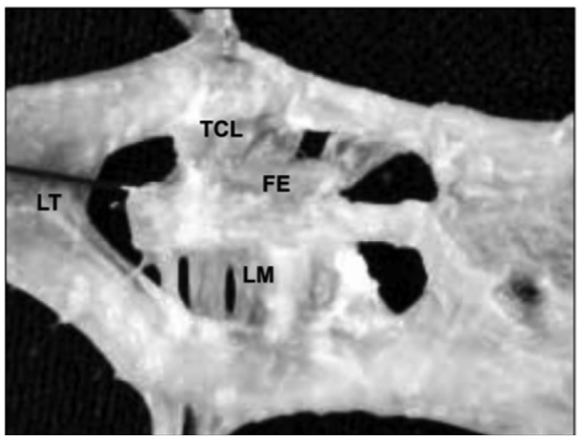

Fig. 4. Photograph of a cadaver dissection of an isolated extensor mechanism, palmar view, showing the spiral fibers (EF) between the central slip (LM) and the conjoined lateral bands (TCL). The triangular lamina is labeled as (LT). (from: Nigro[29]).

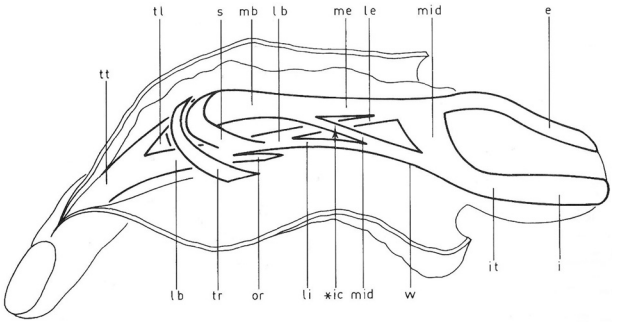

Fig. 5. Schematic representation of the extensor mechanism of the finger.[16] The interosseus muscle (i) continues as the interosseus tendon (it) which continues as the "wing" tendon (w) (known elsewhere in this article as the intrinsic tendon and not to be confused with the interosseus tendon (it) in this study). From the wing tendon (w) medial interosseus tendon fibers (mid) form a lamina that connects the wing tendon with the ED tendon (e) and medial ED tendon (me). The ED tendon (e) splits into 3 fiber bundles - the medial bundle (mb) and 2 lateral bundles (le) that merge with the wing tendons on both sides to form the conjoined lateral bands (lb). The "intercrossing" phenomenon of the intrinsic and extrinsic fibers is at the asterisk (ic).From the conjoined lateral bands (lb) the spiral fibers split off (s) connecting the conjoined lateral bands (lb) with the distal part of the medial bundle (mb) at the CS. The conjoined lateral bands (lb) continue distally to form the terminal tendon (tt) with insertion at the distal phalanx. The conjoined lateral bands (lb) are connected distally over the middle phalanx by the triangula lamina (tl). The transverse retinacular ligament (tr) and the oblique retinacular ligament (or) are also indicated in this view.

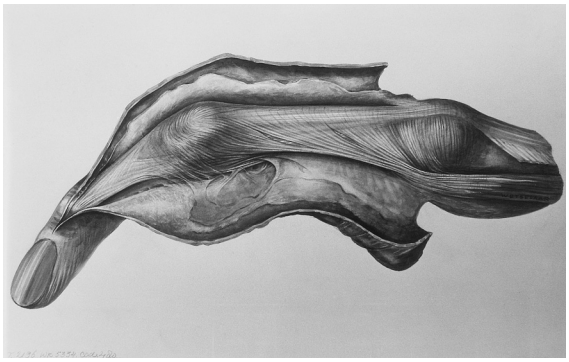

Fig. 6. Ulnar view of the middle finger of the left hand in extension.[16] (© rights retained by van Zwieten).

There are situations in which the retaining ability of the SF-CLB unit is surpassed and the CLBs drop below the axis of PIPJ rotation.[45] With volar migration of the CLBs below the PIPJ axis, extension of the PIPJ generated by the ED and intrinsic muscles is then redirected to flex the PIPJ via the LBs and the intrinsic tendons through the CLBs. Loss of SF-CLB retaining ability ultimately sets the stage for a boutonnière deformity. When trying to rectify this, the challenge for the surgeon reconstructing the EM is to restore PIPJ extension via CLB repositioning by use of the correct amount of tension, to both restore the retaining function of the SFs and at the same time to allow ample room for the lateral shift (widening) needed for full simultaneous flexion of IPJs.[46]

Observations of the balanced functioning of the SFs, LBs and CLBs from extension to flexion

Schoening[47] (1887) more than a century ago described the interdependency of the DIPJ and PIPJ during flexion and extension. In describing this, he stated that "When the middle phalanx is flexed, the lateral bundles of the extensor tendon do not remain on the dorsal surface of the first interphalangeal joint, but rather slide down to the sides. This appears to make them more slack than

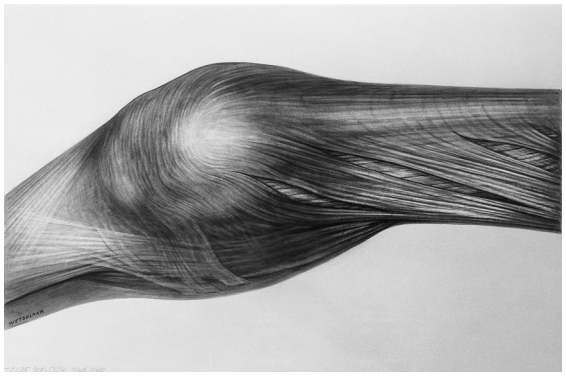

Fig. 7. The extensor tendon zone III area over the PIPJ with the finger in extension.[16] (detail of figure 6) (© rights retained by van Zwieten).

when they traverse the angle formed by flexion of the first interphalangeal joint, and hence they cannot exert more traction on the base of the distal phalanx. The reason for this phenomenon is that when the middle phalanx is flexed, the lateral bundles which serve to extend the end phalanx, relax."[47] Schoening also observed that one "cannot extend the end-phalanx on its own with the middle phalanx flexed; rather, both phalanges are extended to the same extent and at the same time, they are quite dependent on each other." Hauck[11] (1923) in agreement with Schoening described "the lateral sliding of the lateral bundles when the middle phalanx is flexed."[11] Others noted that the volar slide of the CLBs becomes apparent by what is known as, the "release of the third phalanx"[24] or the "floating distal phalanx."[28]

Van Zwieten[16,25,26] reported based on anatomic laboratory studies his observations of the SFs, medial band and the conjoined lateral bands in three positions; IPJ extension (Figs. 5,6 and 7), PIPJ flexion only, and PIPJ flexion first-followed by DIPJ flexion‡.

In Figure 8 the finger is flexed at the PIPJ only. The SFs now lie over the flexed PIPJ. Notice how the fiber directions have adapted to the change in location of the medial band and the change in direction of the CLBs, due to the EM being pulled distally. The SFs no longer run obliquely as with the finger in extension, but rather longitudinally relative to the proximal phalanx. With each spiral fiber ending on the medial bundle the SFs now lie almost in the frontal plane around the head of the proximal phalanx which explains why the SFs are now described as being located more "palmarly". On flexion of the PIPJ the width of the trochlea alters any pair of opposite SFs into a U-shape that also becomes broader than during PIPJ extension.

Then when the PIPJ is flexed first, followed by DIPJ flexion, the SFs and CLBs change position and direction (Fig. 9). The SFs are less longitudinal relative to the proximal phalanx as they bridge a larger distance between the medial band (mb) and the more volarly migrated CLBs than when only the PIPJ is flexed. For each successive SF, the angle to the longitudinal axis of the proximal phalanx is somewhat greater, requiring the distal fibers to run nearly transverse. The most distal SFs are no longer confined to the trochlea of the proximal phalanx, as they have shifted distalward so that the distal fibers are now over the proximal base of the middle phalanx. It is here where the distal pairs of SFs form an inverted (reverse) U-shape and form the proximal border of the triangular lamina.

‡ Note: some components of the EM have been named differently in the publication by van Zwieten[16] (1980) and do not correlate with the nomenclature used elsewhere in this article.

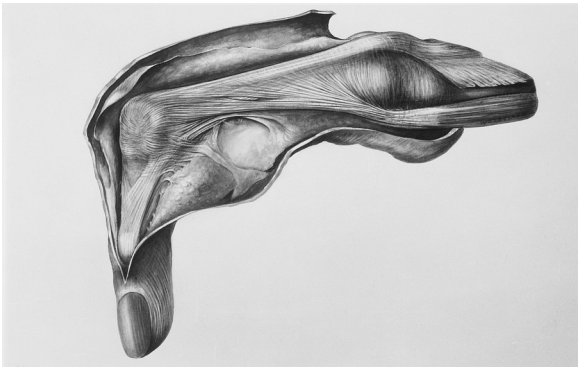

Fig. 8. Ulnar view of the middle finger of the left hand with the proximal interphalangeal joint in flexion.[16] (© rights retained by van Zwieten).

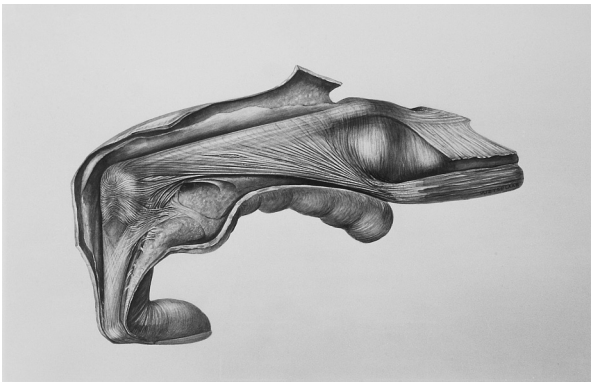

Fig. 9. Ulnar view of the middle finger of the left hand with the proximal interphalangeal and distal interphalangeal joints in flexion.[16] (© rights retained by van Zwieten).

More recent in vivo studies of fingers in normal healthy subjects with high resolution-ultrasound[21] agree with the previous anatomical observations made some 50 years ago by Landsmeer[24] (1976) and others[16,18,25,44] confirming the palmar displacement of the CLBs at the PIPJ with consequently also a widening of the EM at the PIPJ as observed during PIPJ flexion only, and during coordinated PIPJ *and* DIPJ flexion (Fig. 10).

Winslow's tendinous rhombus – Implications of volar glide and widening of the CLBs

As far back in history as the 1700s the EM and the mechanics of PIPJ motion have been studied and published. Winslow (1669-1760) first described the tendinous parts of the extensor mechanism by a diamond shape or rhombus over the dorsum of the proximal and middle phalanges[48] (Fig. 11), Zancolli[43] was the first to make a modelled representation of Winslow's rhombus, while Garcia-Elias et al.[35] suggested a model with a transverse band between the CS and the CLBs ("Distal lateral band: Portion of the lateral band that includes fibers coming from the central-to-lateral bundle"), indicating the SFs. Winslow's tendinous rhombus consists of a V-shape located proximal to the PIPJ formed by the ED LBs as they run towards the intrinsic tendon to form the CLBs and a reverse V-shape distal to PIPJ over the middle phalanx formed by convergence of the CLBs. Together, these two V-shapes form a diamond commonly referred to as Winslow's tendinous rhombus. It is this diamond-shaped structure which resembles a buttonhole (or boutonnière in French) through which the PIPJ (as a button) could dorsally protrude, hence the name buttonhole or boutonnière deformity. Normally the soft tissues between the CLBs and the CS that span over the PIPJ, i.e. the SFs, shown by Garcia-Elias et al.[35] in their version of the rhombus, as a transverse band running across between the long sides of the rhombus, prevent this from happening, however when the retaining SFs attenuate or get injured the PIPJ will push through the "buttonhole."[27,45]

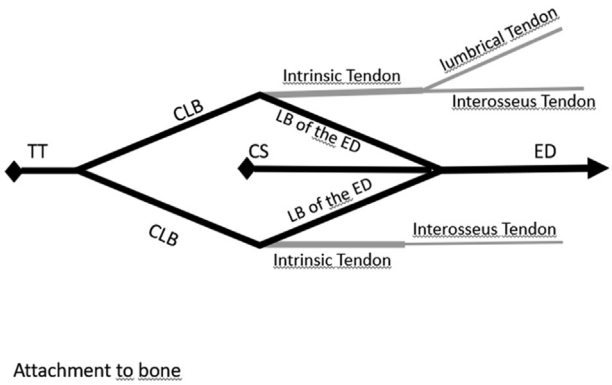

Fig. 11. Modelled representation of Winslow's rhombus adapted after Zancolli.[42] In this figure the tendinous parts of the rhombus are in black, the intrinsic tendons attaching to the side of the rhombus are in grey and the small black diamond-shapes indicate where the rhombus is attached to bone. The extensor digitorum (ED) pulls proximally to extend the finger. Extension forces run through the central slip (CS) to the attachment on the middle phalanx, and through the lateral bands (LB) of the ED; the lateral bands merge with the intrinsic tendon and form the CLB through which the extension force continues distally. The CLBs meet over the middle phalanx and form the terminal tendon (TT) which attaches to the distal phalanx. (© picture by G. van Strien).

Additional stabilizing structures around the PIPJ

As previously mentioned the CLBs under normal circumstances migrate volarly during isolated PIPJ flexion and always remain dorsal to the axis of the PIPJ during composite IPJ flexion because

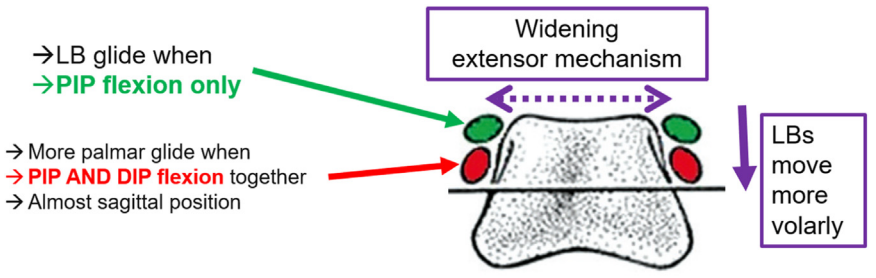

Fig. 10. Volar migration of the conjoined lateral bands (CLBs) and widening of the extensor mechanism at the proximal interphalangeal joint (PIPJ). Volar migration of CLBs in PIPJ flexion only (green); volar migration of CLBs with PIPJ and distal interphalangeal joint (DIPJ) flexed (red). Widening of extensor mechanism (purple arrow). (Based on observations with high resolution MRI and in vivo observations with high resolution sonography, adapted after van Zwieten et al.[21]).

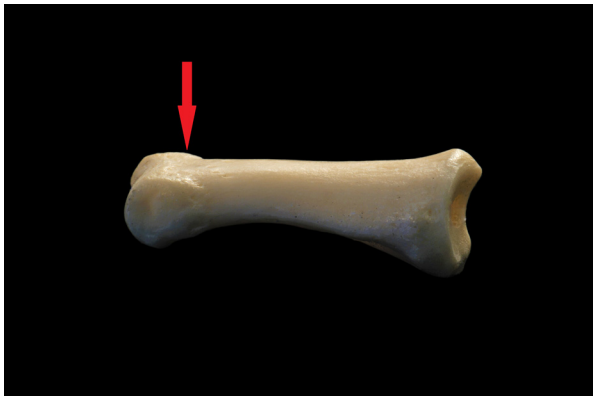

Fig. 12. Lineae asperae seen as bony ridges at the onset of the condyles of the head of the proximal phalanx, index finger, indicated with the red arrow.[21] (internal report).

of the SFs that are located over the PIPJ and between the CLBs.[21] There are however, a few additional anatomical structures that assist in maintaining the position of the CLBs above the PIPJ axis during motion.

The head of the proximal phalanx and the proper collateral ligament (PCL) of the PIPJ

Kanavel[49] (1921, p. 430: figure 144), showed the morphological role of the head of the proximal phalanx, specifically the condyles and dorsal osseous ridges (Lineae asperae) in relationship to the CLBs. These structures, apart from functioning as an attachment for the PIPJ proper collateral ligament (PCL) act as a minute bulge to assist restraint of CLB palmar shift during finger flexion (Fig. 12). Although not often acknowledged, the PIPJ proper collateral ligaments (PCLs) also aid in keeping the CLBs from sliding volarly. In PIPJ extension the PCLs form a bulky mass on which parts of the CLBs rest.[50,51] During PIPJ flexion the PCLs drop volarly removing their support of the CLBs allowing volar migration.[21,52] (Fig. 13A-C).

Inter-tendinous structures

> *"There is considerable controversy concerning the anatomy and mechanics of the extensor mechanism about the PIP joint. The numerous, subtle interconnections among the extensor components in this region create confusion in anatomic descriptions". (Hurlbut and Adams, 1995)*[53]

The triangular lamina (TL) (formed by transverse fibers originating from the CLBs and interconnecting the CLBs, distal to the PIPJ) has been mentioned for decades as the main structure preventing the unwanted volar shift of the CLBs that gives way to boutonnière deformity.[24,32,54] However, more recently Grau et al.[9] and Houston et al. [55] have shown that the role of the TL in retaining the CLBs and preventing volar migration leading to boutonnière deformity may be limited.

When viewing the proximal EM dorsally, the triangular-shape of fibers that cover the proximal two-thirds of the proximal phalanx is formed by the transverse and oblique tendon fibers originating from the intrinsic tendons and attaching to the ED medial band. The oblique and transverse fibers however, do not originate from the CLBs and therefore are not considered as CLB stabilizers. From a palmar view, just proximal to the PIPJ the transverse and oblique fibers form an arch between the intrinsic tendons named by Kaplan[8] (1965, p. 37: fig. 2) as the arciform fibers. Merritt et al.[56] refer to the arciform fibers as the "oblique and horizontal arciform fibers of the extensor hood". They describe the (horizontal) arciform fibers as the ligaments retaining the CLBs from shifting volarly, thus stopping Winslow's diamond from widening. Yet, as these arciform fibers do not originate from the CLBs but from the intrinsic tendons they cannot retain the CLBs from migrating. Granted the oblique interosseus layered lamina which envelops the ED lateral bands may have a limited role in retaining the LBs via their thin connections with the lamina, however we believe their role in preventing boutonnière deformity is questionable and defer to the SFs of the CLBs at the PIPJ as playing the prominent retaining role. Furthermore, as the distal oblique fibers during PIPJ flexion are pulled into a more acute oblique direction, their alignment and the position proximal to the PIPJ provide in our opinion little leverage in restraining the widening of Winslow's diamond during PIPJ flexion, in contrast to the SFs which are pulled distally around the PIPJ where the U-shaped fibers become tight between the CLBs in a frontal plane.[16]

The SFs, located distal to the oblique fibers are located more directly over the PIPJ where the "opening" between the CLBs needs to be "closed". The SFs originate from the CLBs and attach to the ED medial band and the CS, spanning between the CLBs and the CS. During PIPJ flexion the SFs and oblique fibers are both pulled distally however the SFs become more longitudinally oriented than the oblique fibers and are pulled distally around the PIPJ (in front of the PIPJ overlying the middle phalanx)[16,18] (Figs. 8 and 9), becoming more taut between the CLBs at the widest part of the rhombus.[27] It is our opinion that the SFs are anatomically and biomechanically the most effective structure for restraining widening of the tendinous rhombus. Our rationale is that the SFs are better suited mechanically as a consequence of their location that is, at the middle part of the diamond shape. In fact, the mechanical effect appears to get more outspoken during PIPJ flexion, when it really counts, as these SFs get pulled around and even in front of the head of the proximal phalanx getting more taut, putting them in the best position to "fine-tune" the amount of CLB volar shift needed for PIPJ and DIPJ flexion.

Although important for normal-balanced finger function, the oblique -and transverse ligaments (ORL and TRL) play a very small role in stabilizing the CLBs for preventing boutonnière deformity. In fact the ORL to our knowledge has no role in the stabilization of the CLBs at PIPJ level and as such is not further discussed in this article. The transverse retinacular ligament (TRL) originates mainly from the flexor tendon sheath at the first cruciate pulley near the PIPJ with most fibers inserting into the lateral and palmar edges of the CLBs proximal to the triangular lamina. TRL location and insertion show that the primary function is to prevent excessive *dorsal* migration of the CLBs during PIPJ extension thereby helping to prevent PIPJ hyperextension and swanneck deformity.[57] Only a very thin dorsal continuation of the TRL crosses over the most distal SFs and attaches to the medial band and/or CS and therefore the TRL has been mentioned to possibly also play some CLB restraining role by Nigro.[29] Nigro does agree with Milford[58] that the CLBs cannot entirely be dependent on the TRL to restrain volar migration.

When stabilizers fail: Volar shift of CLBs and boutonnière deformity

Boutonnière deformity is thought to emanate from a zone III injury or attenuation of structures that control volar migration of the CLBs. The traditional belief after closed PIPJ injury was that if not protected, overtime the triangular lamina (TL) would attenuate losing capacity to retain the CLBs, eventually resulting in CLBs volar migration and progression to PIPJ flexion contracture or boutonnière deformity. Recently, Grau et al.[9] in 2018 and Houston et al.[55] in 2021 have challenged this traditional scenario. In their independent cadaver studies, Grau et al.[9] demonstrated in 18 digits that

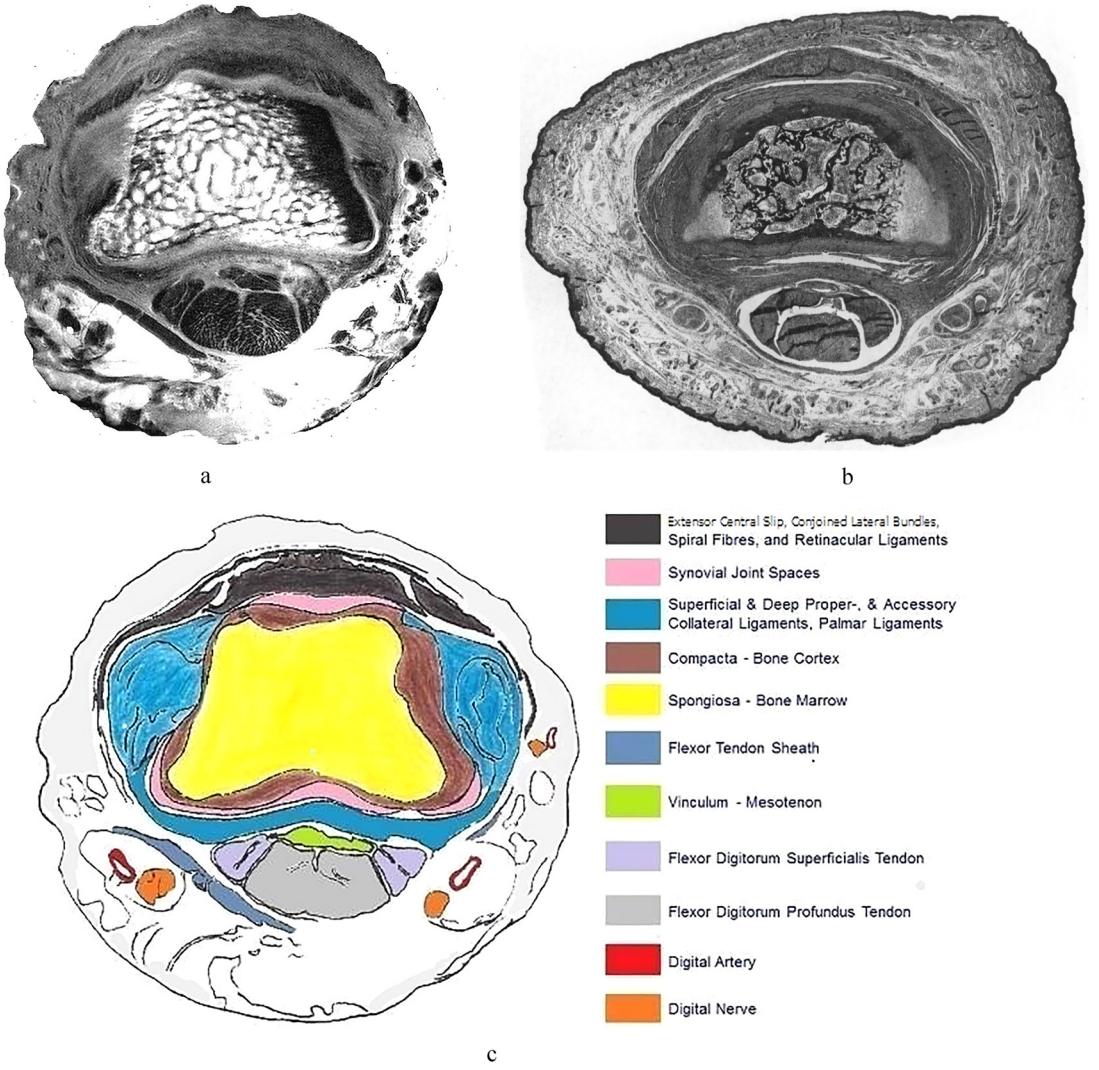

Fig. 13. (A) High resolution magnetic resonance imaging of the proximal interphalangeal joint in full extension, transverse slice.[21] The proper collateral ligaments are shown supporting the conjoined lateral bands (CLBs) (B). Microscopic view of the proximal interphalangeal joint, transverse section (Permission of Wiley).[15] During proximal interphalangeal joint flexion the proper collateral ligaments eventually "drop down" and the conjoined lateral bands migrate volarly.[15,24] (C). Schematic representation of Fig. 13A with different structures colored and legends indicating these structures. (adapted after van Zwieten et al.[21]).

injury to the TL alone does not cause loss of PIPJ extension nor is it the primary retaining structure of the CLBs. Additionally, Houston et al.[55] in a study on 13 digits described the TL as having little effect on preventing CLBs volar migration. In our opinion, comparison between these two studies is difficult as hand positions, forces applied and measurement techniques used differed. Nevertheless both groups concurred in their conclusions about the role of the TL in boutonnière deformity.

As previously stated, the literature and terminology regarding the functional anatomy of extensor zone III is confusing. Even more recent studies do not always mention the SFs, referring instead to the "interosseus fibers" or the oblique and transverse fibers[9] and the tendinous parts of the EM and TL[55] It is our contention based on their descriptions and illustrations in both publications that the structures identified as the oblique and transverse - or interosseus fibers were actually the SFs. To illustrate, in the article by Houston and colleagues[55] they chose to measure widening of the tendinous rhombus after transection of the CS by two points on the CLBs located at the level of the transected CS, at the very distal end of the CS (Fig. 14). It is clear from this illustration that these measuring points were not at the more proximally located ED lateral slips where the oblique and transverse tendon fibers are located, but rather between the CLBs over the PIPJ indicating in fact the location of the SFs at the widest section of Winslow's tendinous rhombus. The incision, slightly proximal to the attachment of the CS where one can find the attachments of the SFs also suggests that these SFs were most likely also (partially) divided causing the *immediate* widening of the EM at the PIPJ.

When stabilizers fail: how to test integrity of the EM at the PIPJ

In the Houston article[55] the efficiency of the Elson test commonly used to assess for *acute* CS injury was challenged. The authors concluded in their simulated acute CS injury that it required 100-200 cycles of finger flexion and extension before a positive Elson's test was observed. It should be mentioned that there was immediate loss of DIPJ resistance indicating a positive Elson's test however this loss was too small to be detected clinically, making the test for an acute injury not efficient for clinical use. Another interesting observation that may need further investigation was that immediate widening of the [conjoined] lateral bands and

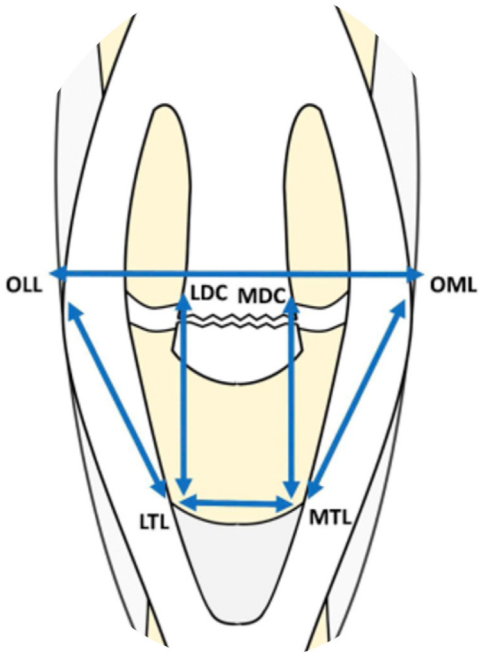

Fig. 14. Widening of the extensor mechanism (EM) was measured between points indicated as "OLL" and "OML" which is directly over the proximal interphalangeal joint (PIPJ) at the widest point of the rhombus where the spiral fibers are located.[55] The authors, however, described the oblique fibers as the retaining fibers in this article. (from: Houston et al.[55]).

volar subluxation correlated to the amount of force that was used during the Elson test.[59]

From a practical perspective it seems questionable to flex the (painful) PIPJ to 90° and then also resist active PIPJ extension putting maximal tension on the LBs and CLBs, especially if there may be a partial CS injury and likely SF injury, that could also be further stressed. As correction of boutonnière deformity is a surgical conundrum,[60] and Elson's provocative test[59] or modified Elson test[61] may in a closed PIPJ injury add insult to injury, why not use the relative motion flexion (RMF) pencil test to assess a zone III injury.[62] If the RMF pencil test restores PIPJ extension, then a RMF orthosis can be applied to protect the acute injury and initiate controlled early active motion.[62]

Extensor force distribution within the EM zones III-IV

Often, we consider anatomy as being static however once movement is introduced, the form and function observed statically, can change. This is definitely true when applied to the anatomy of the finger, as the various joints of the finger change position, the forces of the multi-origin extrinsic and intrinsic muscles change and dispersal of forces shift within the EM.[35,40,53,63,64] Recovery of EM shifting proximally and distally to orchestrate movement of finger is challenged after zone III or IV injury or surgery as the EM's multilayered gliding fibers attenuate or adhere. Understanding force dispersion in the different parts of the EM during normal finger motion is difficult. For example, Dogadov et al.[65] discussed the problem with previous studies on force transmissions in the EM using equations with constant coefficients determined on experiments done with the finger in extension only, not taking into account that these values will change during finger flexion because of EM deformation causing inaccurate results. They proposed an experimentally modeled EM in three positions of finger flexion taking these changing coefficients into account. The research team of Sang Wook Lee et al.[66] studied cadaveric hands to record the effect of finger posture on force distribution within the EM, measuring both longitudinal and lateral strain (the latter of importance in regard to the widening of the EM with PIPJ flexion). Of particular interest was the 30%-50% decrease in CS and TT tension during IPJ extension with the MCPJ in a flexed position, which is simulated by a RMF orthosis.

To understand the force distribution within the EM, Kai Qian et al.[67] obtained tissue samples of 19 finger extensor hood specimens harvested from fresh-frozen human cadaveric hands. The samples were taken from proximal to distal at various sites along the EM. The tissue samples were loaded until failure. Results demonstrated significant differences in ultimate load and strain, thickness, and tangent modulus for the different samples taken from proximal to distal within the EM. Thickness, ultimate load, and ultimate strain were greater over the proximal phalanx and the tangent modulus greater over the middle phalanx and proximal to the insertion of the TT. Of course one should never presume force transmission would be the same in vivo or following surgical reconstruction or repair of EM structures. Nevertheless, this study shows that, apart from many other variables, these differences in tissue characteristics in zones III and IV as compared to zones I and II may also need to be taken into account when planning our therapy management after injury and surgery.

The above-mentioned studies inform us that there is much more to still learn about the dynamic EM, from position change of the finger to force dispersal under normal, post-surgical and pathological conditions. Future study of zones III and IV with and without the RMF orthosis may enlighten us regarding the orthosis' role in the preservation of IPJ extension through the remaining intact fibers and the inhibition of volar migration to avoid paradoxal PIPJ flexion and ultimately boutonnière deformity.

Theoretical mechanism of relative motion flexion (RMF) in PIPJ extension and boutonnière

Merritt,[68] innovator of the relative motion (RM) concept who protected repairs of finger extensor tendons with RM extension (RME) orthoses learned in his conversation with a colleague about her use of a "reverse RME orthosis" with a patient to correct an active PIPJ extension lag (2005 ASHT meeting, Hollywood, CA). Subsequently with the assistance of his colleagues, Maureen Hardy and Sandra Robinson, he demonstrated in a video the use of what now is referred to as a RM flexion (RMF) orthosis, on a cadaveric hand with a fully divided CS.[69] In the video demonstration, after fully dividing the CS of the index finger, the ED tendon (no intrinsic muscles) was manually pulled on with a RMF orthosis in place. The RMF orthosis did not allow the MCPJ to hyperextend and IPJ extension was normal that is, no claw or boutonnière deformity developed. Next the RMF orthosis was taken off and again the ED tendon was manually pulled on. Clawing started immediately (MCPJ hyperextension and PIPJ flexion) as MCPJ hyperextension was not stopped by RMF and almost immediately the CLBs dropped volar to the PIPJ axis of rotation and a boutonnière ensued. Since viewing the video many therapists have been using the RMF orthosis to manage acute open and closed zone III injuries and chronic boutonnière deformities.[60,70]

But how does it work? Howell (AAHS newsletter, 2017) stated that "Fibers of the extensor apparatus work in harmony to produce balanced finger extension. A shift in this balance (extensor zone III injury) will result in loss of PIPJ extension. If this imbalance is unchecked, the forces applied through the remaining fibers of the apparatus progress to a boutonnière deformity." From her words we should infer that leaving the balance unchecked or uncontrolled seems to be the key factor.

The exact mechanism(s) of how a RMF orthosis works to protect a suspected zone III injury or corrects a boutonnière deformity is unknown. Houston et al.[55] observed in a simulated test environment an *immediate* widening of the CLBs indicating that their volar migration starts soon after CS injury. This is however only a small change which may not be noticed by the patient. What we and others[10,46,60] have observed in practice is that there is often a delay of 1-2 weeks following a closed injury of the PIPJ before a PIPJ extension lag shows enough for the patient to seek help. As hypothesized by Merritt and Howell[60] and demonstrated by Grau et al.[9] if after partial injury to a CLB stabilizing structure, these injured structures are not immediately protected they will overtime attenuate secondary to the stress of repeated PIPJ flexion, as with normal use of the hand, causing the CLBs to shift below the axis of the PIPJ and then active PIPJ extension becomes impaired.

One possible mechanism for how a RMF orthosis works to protect zone III closed PIPJ injuries and correct boutonnière deformity is through blocking MCPJ hyperextension. Without an intact CS the EM is able to slide more proximally acting as extensor mostly on the MCPJ ED attachment through the sagittal bands inducing MCPJ hyperextension while losing tendon forces in the distal attachments of the EM. In support of these observations is the work of Sarrafian et al.[63] who measured force tension in human and other primate fingers with the MCPJ in hyperextension. Their results measured no tension in the CS or TT (as the force was diverted through the volarly shifted CLBs with the MCPJ in hyperextension) and return of the force to the CS and TT when MCPJ hyperextension was blocked.[63] In controlling MCPJ hyperextension the extensor forces will act less at the MCPJ and will redirect extension forces on the ED LBs restoring active PIPJ extension. Similarly, the rebalancing of the extrinsic forces is also seen as described after Fowler procedure[71] and by slightly passively flexing the MCPJs of patients with intrinsic muscles weakness or paralysis.[72]

Another possible mechanism for how RMF orthoses work to protect a suspected zone III closed injury or re-establish extension forces to correct a boutonnière deformity is that MCPJ flexion 'pretensions' the ED thus increasing tension in the ED LBs, countering any volar-directed tension from the intrinsic muscles as they contribute to IPJ extension. The relatively flexed position of the injured finger MCPJ is also proposed to impose a quadriga effect on the FDP and its lumbrical making IPJ extension easier against the more relaxed FDP antagonist and possibly also decreasing the volarly directed force from the lumbrical on the CLB.[60] The volar pull on the CLB by the lumbrical muscle is suggested by some as the major deforming force to increase volar migration of the CLBs.[60] However the fibers of the lumbrical merge with the intrinsic tendon only on the radial side therefore they can only pull on the radial CLB, which makes this assumption less likely. Furthermore the force of the lumbrical for MCPJ flexion and IPJ extension is weak compared to the interosseus contribution which has the same function that is, active extension of the PIP and DIP joints, as well as flexion of the MCPJ,[73,74,75,76] albeit that the angle of approach of the lumbrical is from a more volar direction which would make it a more efficient force for volar displacement of the CLBs than the interosseus muscle. However, the lumbrical is seen as a muscle with limited motor function. In a study by Schreuders and Stam[77] the strength of the lumbricals for PIPJ and DIPJ extension was measured with a specially designed dynamometer on 12 patients with ulnar nerve paralysis. With paralyzed interosseus muscles it was possible to measure strength for PIPJ and DIPJ extension of the first and second (median nerve innervated) lumbrical. The data showed a 90% loss of intrinsic muscle strength for PIPJ and DIPJ extension compared to the other hand, indicating the weakness of the lumbricals compared to the interosseus muscles. The high concentration of muscle spindles in the lumbrical muscles indicates a more proprioceptive controlling role in finger flexion and extension.[75]

Another potential positive effect of the RMF orthosis is related to both the above described mechanisms, using the RMF orthosis to re-route the predominant extension force through the ED LBs and then through the CLBs, thus increasing the extension force via the long sides of the rhombus. The long sides of the rhombus (CLBs) will become more parallel as they are pulled "straight", creating a correcting medial-dorsal directed resulting force that pulls the long sides (i.e., the LBs and CLBs) of the rhombus towards each other (Fig. 15). As the EM can slide more proximally when there is not an intact CS it will enhance this ability of the rhombus to become narrow, pulling the diamond shape "shut."

The RMF however does *not* control the amount of PIPJ flexion which would put tension on the CS and the SFs. Van Zwieten et al.[21] (2018) observed that PIPJ and DIPJ flexion caused CLB migration and therefore widening of the CLBs, indicating that PIPJ flexion must cause some tension on the retaining structures of the CLBs. Sarrafian et al.[63] measured tension in the CS after flexing the digit by FDP action. There was no increase of tension in the CS until PIPJ flexion reached 60-70°. In this study the increase of lateral tension (on the SFs between the CLBs) was not recorded and remains an area of interest. It seems that some form of protection of the healing retaining tissues of the CLBs and of the CS might be needed during flexion. Several authors[60,78,79] and author GvS have proposed ways to control PIPJ flexion for example, by combining a short arc motion (SAM) orthosis or dorsal taping with the RMF orthosis, while delivering controlled early active motion (EAM) in the management following zone III injury for suspected boutonnière deformity, acute and chronic boutonnière deformity and CS repair or reconstruction (Figs. 16A-C).

Questions for future discussion

Although use of RMF orthoses for management of extensor zone III (and sometimes zone IV) is being reported in the literature[60,69,80,81] there remains a gap in the biomechanical and kinesiological evidence to support our clinical observations and many unanswered questions.

Is the CS necessary for PIPJ extension? The short answer seems to be that the CS is probably not necessary for PIPJ extension. This answer can be observed in the Merritt video[69] discussed earlier and after careful surgical CS transection in Fowler's procedure, and the findings extrapolated from the cadaveric research of Grau et al.[9] and Houston et al.[55] However after CS injury when uncontrolled active motion of the PIPJ is allowed, attenuation of the CLB retaining structures may progress to boutonnière deformity. The difference between a careful transected CS in Fowler's procedure and a closed PIPJ injury is that in the latter, the extent of tissue injury is unknown. The practical use of the RMF pencil test after a closed injury rather than any version of Elson's test, to assess active PIPJ extension seems clear.

Is surgery the only choice after closed injury rupture of the CS? After reviewing surgical outcomes of patients with closed injuries of the CS, Colzany et al.[82] concluded that in most situations an attempt of conservative treatment should always be made as the conservative approach had better results compared to direct surgical repair of a ruptured CS. Not only does the surgery itself cause adhesions, it is commonly known that a closed injury with a ruptured CS results in frayed tendon ends that are difficult to suture increasing the likelihood of adhesions. As discussed, the RM pencil test can non-invasively provide information on the functioning of the EM after closed injury which can help decide the effectiveness of a conservative approach with RMF. The test can however not in-

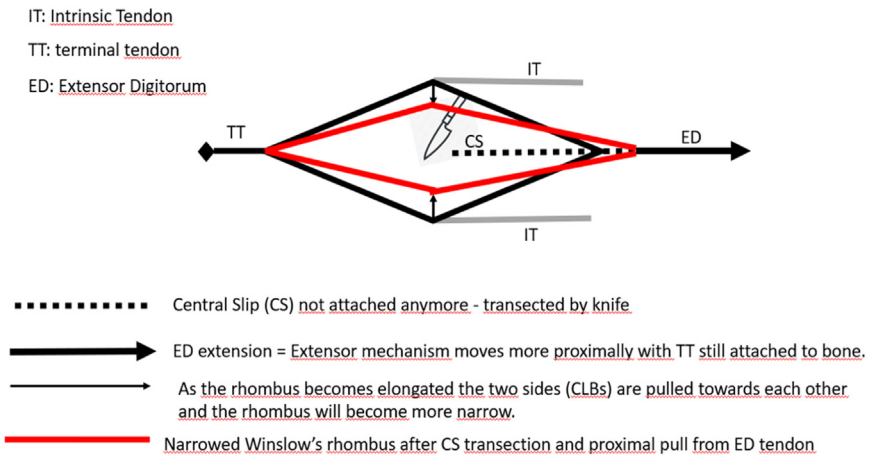

Fig. 15. Narrowing of Winslow's tendinous rhombus during extensor digitorum (ED) active extension shown in a simplified representation of the tendinous rhombus. The transected central slip (CS) allows the ED to pull the extensor mechanism (EM) more proximally which elongates the rhombus and pulls the sides towards each other.[46] (© picture by G van Strien)

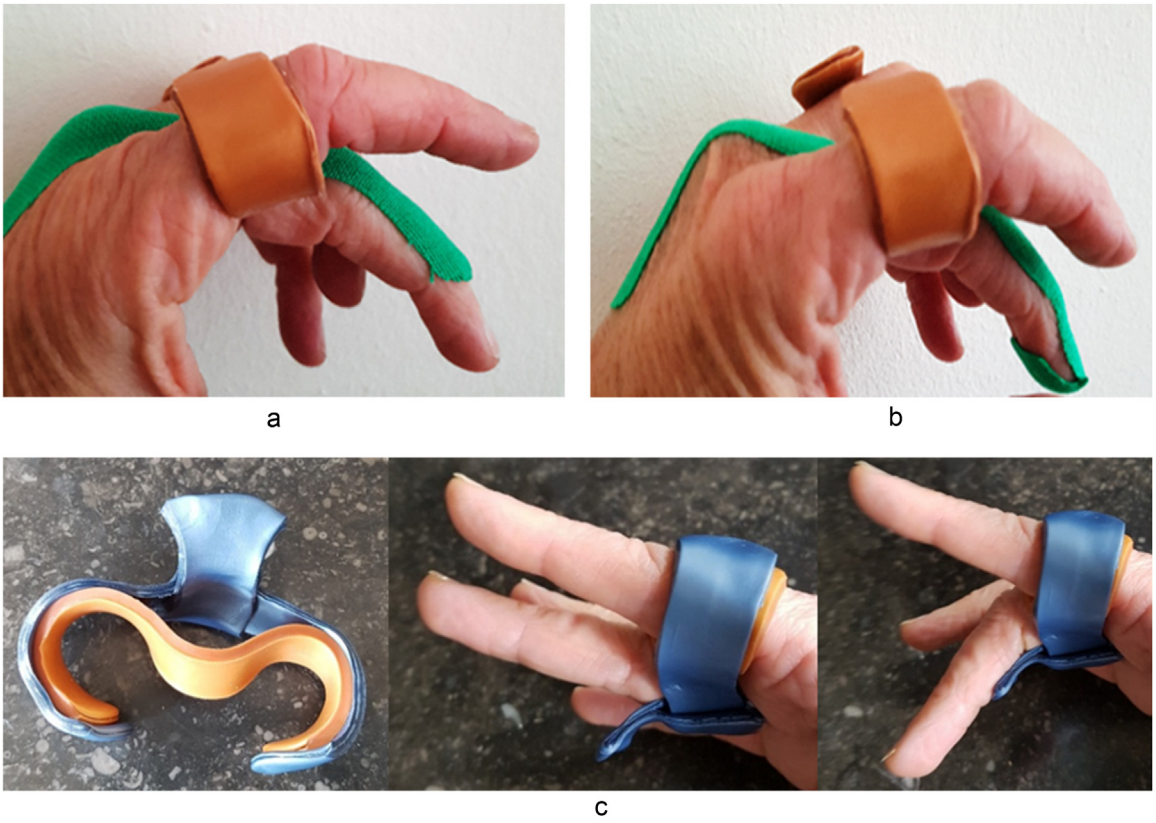

Fig. 16. (A) Relative motion flexion (RMF) orthosis option using elastic tape to limit proximal interphalangeal joint (PIPJ) and sometimes distal interphalangeal joint (DIPJ) flexion to protect the healing- or repaired central slip (CS). The elastic tape limits PIPJ flexion in RMF orthosis when tape is applied proximal to the DIPJ. (© picture by G van Strien). (B). Elastic tape (Kinesiotape) can be applied to limit too much PIPJ *and* DIPJ flexion when tape is applied further distally around the tip of the finger. (© picture by G van Strien) (C). Relative motion flexion (RMF) orthosis idea for limiting proximal interphalangeal joint (PIPJ) and sometimes distal interphalangeal joint (DIPJ) flexion to protect the healing- or repaired central slip (CS). Combining a RMF orthosis (gold) with an overlying PIPJ flexion block orthosis (blue) to allow short arc motion (SAM). The combination of orthoses allows PIPJ and DIPJ extension through the RMF orthosis and also blocks PIPJ flexion as in SAM. Block can be adjusted as healing allows. (© picture by G van Strien).

dicate to what extent the tissues are damaged. For both a surgical or conservative approach, implementation of a RMF orthosis to deliver controlled EAM should be considered to keep the zones III-IV interfaces gliding, all the while protecting the injured or repaired structures.

Does the CS retract after injury? There is always the chance after closed injury of the PIPJ the CS will not heal where it was originally attached. However, consider that the EM is distally connected to the distal phalanx and the proximal EM is attached to the MCPJ, minimizing the opportunity for great EM and thus CS retraction. Houston et al.[55] measured the amount of CS retraction during their study on cadaveric fingers and demonstrated retraction was no more than 5mm. Regardless, a very small increase in tendon length due to CS retraction should not cause problems as

we already discussed that the finger can even function without the CS.

After CS injury/repair, should PIPJ flexion be prevented? The short answer is no. However, keep in mind that two entirely different scenarios have been incorporated into this question. In regard to PIPJ flexion after CS repair, in an ideal world, the surgeon can, through intra-operative observation, provide the therapist with guidelines to how much initial flexion is safe postoperatively. During DIPJ flexion with the PIPJ held in extension, the EM is pulled distally thereby decreasing tension on the CS and Sarrafian et al.[63] showed that no substantial increase in CS tension was noted until PIPJ flexion is beyond 60-70°. Keep in mind however, these findings were based on the non-surgical/non-injured finger, so clinical reasoning, guided by each patient's circumstances, will be required when EAM of the PIPJ post CS injury/repair is used. By simply limiting the arc of PIPJ flexion, following the patient reports on pain or feeling of tightness or stretch at the CS area, one can prevent too much tension on the CS and other EM stabilizing structures during controlled EAM. If a restricted arc of PIPJ flexion is indicated, the RMF orthosis will need to be modified[60,79,83] and as the structures heal, progressively allow more controlled active PIPJ flexion. The CS is not the only EM structure that may be exposed to too much tension during composite IPJ flexion.[10] Recall that van Zwieten et al.[21] noted more lateral shift of the CLBs with composite PIPJ and DIPJ flexion, hence with this in mind, clinicians may want to prescribe exercises that isolate PIPJ from DIPJ flexion and vice versa, and limit composite flexion of the IPJs beyond comfort during the first several weeks after injury/repair.[78]

Summary

The clinical use of RMF orthoses to manage injuries involving extensor zones III-IV has heightened since its introduction nearly twenty years ago and has sparked renewed interest in the anatomy of the extensor mechanism, especially in zones III and IV. We felt it was time for a more detailed overview of the anatomy of the extensor mechanism in these zones including new insights in the biomechanics and kinesiology of this structure in order to enhance our understanding of how the RMF orthosis works. In doing so, we have generated more questions, but hope to have answered and addressed many more. Our intent was to stimulate further investigations and clinical study regarding the RMF orthosis in the management of closed PIPJ injuries, acute and chronic boutonnière deformity.

Declaration of Competing Interest

None.

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