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*Letter to Editor*

# The biomechanics of finger flexion: Exploring tendon dynamics and stability

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**Quick Response Code:**

Dear Editor,

Finger flexion is a sophisticated biomechanical process that requires coordinated interactions between extrinsic tendons, intrinsic muscles, and fibrous stabilizers. The primary drivers of finger motion are the flexor digitorum (FD) profundus and FD superficialis (FDS), which act on the distal and middle phalanges, respectively. Extension is regulated mainly by the extensor digitorum (ED), whose force is transmitted through the sagittal bands to prevent ulnar displacement during motion.[1]

The fundamental interaction: Flexor tendons (FD1, FD2) act across phalangeal joints while extensor tendons (ED1, ED2) counterbalance them [Figure 1]. The trajectory and insertion points of each tendon generate directional vectors, which are visually depicted by the red arrows. This simplified anatomical model illustrates how isolated or combined dysfunctions, such as ruptures or adhesions, can disrupt balance, leading to clinical conditions such as claw finger or swan-neck deformity.

The stability of the extensor mechanism also depends on the integrity of passive stabilizers, such as the sagittal bands, triangular ligament, and transverse retinacular ligament. These fibrous elements maintain proper alignment and restrict volar or dorsal displacements during dynamic loading. Although not visible in the selected figures, their anatomical continuity with tendinous structures provides a scaffold for transmitting motion and enhancing energy efficiency.[2-4]

Intrinsic muscles – particularly the interossei and lumbricals – offer essential modulatory roles in both flexion and extension. Their insertion into the extensor hood allows for fine-tuned control of finger posture. As shown in Figure 2, the interossei (labeled int) act by pulling on the lateral bands to achieve simultaneous metacarpophalangeal (MCP) flexion and interphalangeal (IP) extension. This dual action is most effective within a certain MCP flexion range (0–45°), beyond which tendon distalization reduces mechanical efficiency.

The interplay between these systems is sequential and hierarchical. Finger flexion typically begins at the proximal IP joint, triggered by the FDS, followed by the MCP and then the distal IP joint flexion. Such coordination is vital for grasping, precision tasks, and distributing grip force. Disruption of this sequence – either by neurological damage, tendon rupture, or post-surgical stiffness – can severely impair function.

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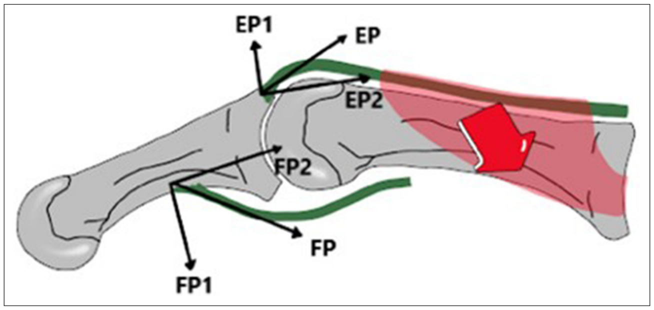


**Figure 1:** Simplified anatomical representation of finger flexion and extension. The flexor digitorum superficialis and profundus (FP1, FP2) are shown in green, initiating motion across the proximal interphalangeal and distal interphalangeal joints. The extensor digitorum (EP1, EP2) in green counterbalances flexion through dorsal stabilization. The red arrow indicates the dominant flexion force vector across the middle phalanx.

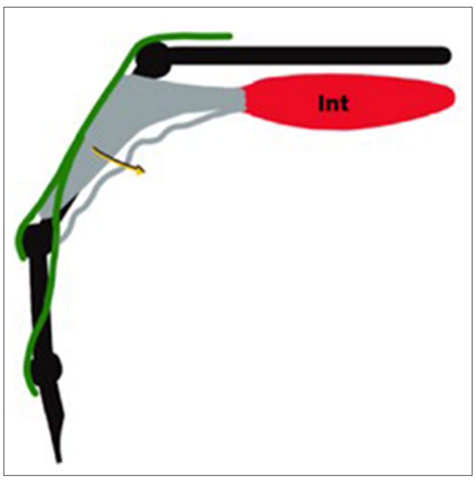


**Figure 2:** Intrinsic muscle function during finger flexion. The interosseous muscle (int = interosseous muscle, red) stabilizes the lateral bands through its attachment to the extensor hood. The grey band represents the sagittal band system. The yellow arrow indicates the resultant vector of the interossei, contributing to metacarpophalangeal flexion and interphalangeal extension simultaneously, a mechanism critical to coordinated hand movement.

Clinically, understanding these dynamics is crucial for planning both conservative and surgical interventions. For example, tendon transfer procedures or splinting protocols must account for the balance between flexors and extensors, as well as the functional range of intrinsic muscle contributions. In addition, early rehabilitation focusing on restoring tendon gliding and intrinsic balance is pivotal in post-operative protocols for flexor tendon injuries.

In summary, finger flexion arises from a finely tuned balance of extrinsic and intrinsic forces modulated through dynamic and passive stabilizers. Educational models, such as the ones shown here, facilitate an understanding of these complex interactions and can inform clinical decision-making in surgical repair, splint design, and targeted rehabilitation strategies.

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