The Anatomy and Pathogenesis of Tendinous Interconnection between Flexor Tendons in the Hands of Musicians

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Introduction

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The Anatomy and Pathogenesis of Tendinous Interconnection between Flexor Tendons in the Hands of Musicians

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Abstract

The tendinous interconnections occurring between the flexor pollicis longus (FPL) and the flexor digitorum profundus (index finger) (FDPi) (called the Linburg - Comstock syndrome) can be classified as acquired or congenital. Constant and repetitive movements in the hand of musicians can lead to inflammatory changes resulting in an increase in the Tendon Cross Sectional Area (TCSA) and, subsequently, lead to acquired tendinous interconnections. The difference in the morphology of the superficial and deep flexor tendons consequently predispose to the synovial membrane getting trapped between the individual tendon strands. In addition, the lack of differentiation of the common mesodermal mass during the foetal life may lead to the congenital tendinous interconnections. This article reviews the relevant embryology, salient anatomy and the essential pathophysiology of tendinous interconnections in the hand.

Key Words

Linburg-Comstock anomaly, tenosynovitis, peritendinuem, epitendinuem, work related musculoskeletal disorders

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Introduction

Tendinous interconnections are estimated to occur in about 20% of the general population[1]. Although string music players present with symptoms associated with tendinous interconnections and, consequently, considered to have a higher incidence, there is insufficient evidence in the literature that conclusively establishes higher incidence in this group. Studies by Miller *et al*[2]. (2003) and Karalezli *et al*[3]. corroborate the above observation.

Constant and repetitive movements involving the flexor tendons of the thumb and the fingers whilst typing, playing sports, playing string and keyboard-based instruments can result in tenosynovitis (inflammation of the outer synovial sheath that covers the tendon - tenosynovium) and tendonitis (inflammation of the tendon). This leads to an increase in the tendon cross sectional area (TCSA), which subsequently contributes to the development of tendinous interconnections[4]. These interconnections, coupled with an anatomically congested carpal tunnel at the wrist, may lead to compression of the median nerve resulting in carpal tunnel syndrome (Slater, 2001). Aside from being acquired, interconnections may also be developmental or inherited[5].

This paper looks at the pathogenesis of the tendinous interconnections in the hands of the musicians. It also analyses the embryological basis of the tendinous interconnections and the factors that predisposes to the formation of these interconnections around carpal tunnel including the morphology of the superficial and deep flexor tendons and the process of muscle differentiation.

Overview of the hand

The human hand, an intricate and prehensile part of the body, is capable of a wide range of movements involving extreme precision and exactitude. The hand is the region of the upper limb distal to the wrist joint. Its skeleton consists of carpal bones, metacarpals and phalanges. The soft tissue covering the skeleton consists of tendon and its coverings, small muscles of the hand and neurovascular structures. These structures cover (envelope) the phalanges to form the digits. The five digits consist of the laterally positioned thumb and medial to the thumb the four fingers the index, middle, ring and little fingers. The hand has a volar (anterior or palmar) and dorsal (posterior) surface.

The ability to use the hands has evolved over time, starting with primitive gestures such as grabbing objects to more precise and highly dexterous activities such as threading a needle that warrants accurate hand-eye coordination. Fine motor skills require controlled use of the small muscles of the hand, fingers and the thumb, in conjunction with forearm muscles and wrist movements. The development of these skills allows humans to undertake complex tasks such as typing, writing, buttoning, sewing, and playing certain musical instruments (such as the guitar, violin and piano).

Histology of the flexor tendons

The normal histology of the flexor tendons consists of densely packed collagen fibrils running parallel to each other. A dense connective tissue called the endotendineum separates collagen fibrils from each other. The blood vessels and nerves run through the endotendineum in a longitudinal fashion. The capillary wall is composed of lining endothelium resting on a basal lamina and sub-endothelial connective tissue. The ground substance (the extracellular space between collagen fibrils) consists of polysaccharides and extra-cellular fluid. Groups of endotendinuem may be reorganised to form larger functional units by thicker connective tissue to form the peritendineum. The group of peritendineum are surrounded by dense irregular connective tissue to form epitendineum. The nucleus of the longitudinal elongated fibroblasts lies in the widest portion of the cell and these are scattered between the collagen fibril bundles. Within these collagen fibres also lie elongated and flattened nuclei of inactive fibrolast (tendinocytes)[6].

Histology of the tenosynovium

To protect the epitendineum layer of the tendon from friction they are surrounded on the outside by two layers of flattened synovial cells of mesenchymal origin. Of these two layers, one of the layers is attached to the tendon while the other one is attached to the neighbouring structures. The space (tendon sheath space- TSS) between the two layers contains a viscous fluid that is composed of water, protein and hyaluronate[7].

Carpal tunnel anatomy:

Structures passing through the Carpal Tunnel (Contents of the Tunnel)

The carpal tunnel contains the median nerve and all the long flexor tendons to the digits and the thumb (FDP, FDS and FPL). The median nerve is the most superficial structure in the carpal tunnel[8]. The motor branch of the median nerve in hand arises from the main trunk under or just distal to flexor retinaculum, and winds around the distal border of retinaculum to reach thenar muscles (including the APB, FPB and OP) and the first two lumbricals. The sensory branches of the median nerve innervate the skin over the lateral three and 1/2 digits including the nail bed and dorsal surface of the distal phalanx (can be up to distal half of the middle phalanx)[9]. A cross section through the carpal tunnel is shown in **Figure 1**.

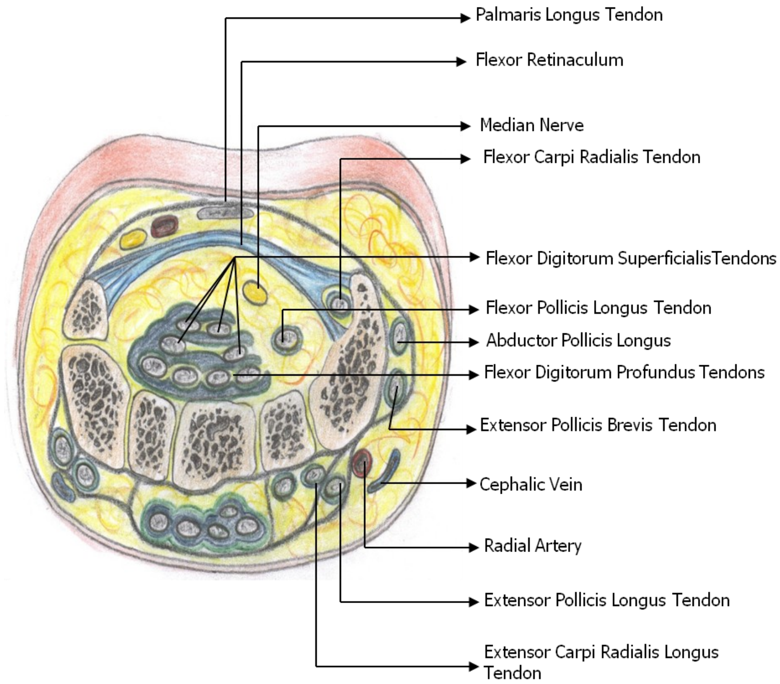

***Figure 1*:** Cross section of the carpal tunnel.

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Within the carpal tunnel, the alignment, shape and relationship of the median nerve to the flexor tendons varies depending on wrist movements. Using US scan, Zeiss and colleagues (1989) noted that during flexion of the wrist, the nerve lies anterior to the FDS (index) tendon and during extension the nerve became interposed between the superficial flexor tendons of the index finger and FPL of the thumb or between the FDS of the middle and ring fingers. It was also noted that the area of the nerve changed with wrist movements. During flexion the nerve flattened antero-posteriorly whilst it became rounded during extension.

There is an inverse relation between the width and the thickness of the median nerve as it passes through the carpal tunnel. As the width increase from an average of 6.1 mm (at the middle portion of the tunnel) to 7.7 mm (at the exit of the tunnel), the thickness of the nerve decreases from an average of 2.1 mm (at the middle portion of the tunnel) to 1.9 mm (at the exit of the tunnel)[10]. Thus the median nerve flattens during its course through the carpal tunnel. This may be to easily pass through the tunnel and to accommodate the accompanying flexor tendons[11]. Similar findings have also been established using US scan by Buchberger and colleagues in 1991. The increase in the area of the nerve is due to increased density of the intraneural connective tissue especially within the epineurial layers[12]. There is also an increase in the thickness of the arteriole wall and the venules due to endo proliferation[13]. This change to the vessel wall is regarded as an adjunct protective feature that opposes increased intra-tunnel pressure during wrist movements and reduces the chances of vascular collapse[12].

Structural changes in the Carpal Tunnel during wrist movements

Movements such as normal flexion and extension of the wrist and fingers affect the width and dynamic pressure within the carpal tunnel. Flexing the wrist causes the flexor retinaculum to move closer to the radius which considerably decreases the cross section of the proximal opening of the tunnel and also the distal end of the capitate moves into the opening. In extreme extension the lunate constricts the passage as it is pressed toward the interior of the tunnel.

During ulnar deviation of the wrist, the triquetrum glides distally across the hamate. This movement causes the triquetrum to move into extension resulting in reduced height of the ulnar aspect of the wrist. In addition, during this movement, the hamate approaches the ulnar styloid and the lunate rotates antero-medially along with it into extension. During radial deviation, the distal row of carpal bones migrate radially whilst the proximal row, mainly the scaphoid and lunate, move towards the ulnar styloid. The capitate along with trapezoid moves more towards the radial styloid in relation to the scaphoid and lunate movements[14].

During forceful flexion of the fingers (such as when making a fist), the lumbrical muscles migrate proximally into the carpal tunnel and increase the pressure within the carpal tunnel from about 2.5 mmHg to 31 mmHg at the most constricted part of the tunnel (at the level of hook of hamate)[15]. The cross sectional area (CSA) of the carpal tunnel is found to increase during flexion of the wrist. This is to accommodate the lumbrical muscles that move into the carpal tunnel during flexion. This adjustment causes overall reduction in the space within the carpal tunnel and may result in compression of the median nerve (carpal tunnel syndrome is discussed later in this chapter). The other adjustments that occur during flexion of the wrist include compression of the fat, flattening and displacement of the median nerve, and pressure on the superficial and deep flexor tendons[16]. During wrist extension, the cross-sectional area of the carpal tunnel increases at the level of the hook of hamate thus decreasing the pressure within the tunnel[17].

Essential embryology

Upper limb

In humans, the upper limb develops by the end of the first month of intrauterine life. At this stage, the limb bud appears as a mesenchymal core (which is a type of undifferentiated loose connective tissue derived mostly from the mesoderm - one of the three primary germ cell layer) covered by a thin layer of epithelium[18]. The hands and the fingers are well developed by the end of second month of intrauterine life. The differentiation of the tendons at the end of muscle belly begins between the seventh and eighth week[19]. Limb fibroblasts and tenoblasts (tendon cells) primarily originate from the somatopleura, which is formed from the outer layer of the lateral plate mesoderm found at the periphery of the embryo[20].

Development of flexor tendons and pulleys

Limb muscles are formed by myogenic precursor cells that migrate into the limb buds and differentiate into myoblasts. The myogenic precursor cells are derived from the dorsolateral muscle-forming region of the somites (which are bilaterally paired segments of mesoderm that are arranged along the anterior-posterior axis of the developing embryo)[21]. In humans, they migrate into the limb buds during the fourth week of development. Following migration of the mesodermal cells into the limbs, the axons of the nerve from the corresponding rami of the spinal cord follow them proximally to distally[22]. These mesodermal cells unite into two common muscle masses which later splits to form the extensor and flexor compartment respectively. The myoblasts hypertrophy and fuse into myotubules; every muscle is recognizable by seven weeks.

Shellswell and Wolpert[23] (in 1977) demonstrated that tendons (which are somatopleuric in origin) develop independent of the muscle bellies. The tendinous and muscle blastemae (the formative, undifferentiated material from which muscles and tendons develop) start to develop separately from one another and join up secondarily. In chick embryo study, it has been found that the tendons start to develop earlier and anterior to the future forearm muscles, despite the absence of these muscles. However, for their maintenance and further development the tendons require connection to at least one muscle belly or the whole muscle group. Further to this observation, experiments by Kieny and Chevallier[20] (1979) demonstrated that if the dorso-ventral axis of the limb were to be inverted the tendons developed normally but joined with the wrong muscles *i.e.,* ventral tendons matched with dorsal muscle group and vice versa. This establishes that the attachment to a muscle is necessary for the further development of the tendon. The pulley system is recognised by week nine as condensing mesenchyme. The pulleys are well-developed by the 12th week of intra-uterine life and are identifiable around the flexor tendon in positions similar to that found in the adult hand[24].

Work Related Musculoskeletal Disorders (WRMD)

Musculoskeletal problems are considered significant health factors for performing artists, especially instrumentalists. Use of the hand in continuous and repeated activities such as typing, playing string- or key-based musical instruments places an increased degree of stress and strain on the soft tissue structures including the tendons. In the long-term, this can lead to reduced functional efficiency of the involved digits and/or the hand, resulting in inability to perform the task that requires a very high degree of dexterity. The symptoms of this condition include pain, weakness of the hand, tingling and stiffness[26], which might be related to the tendonitis, tenosynovitis or carpal tunnel syndrome. Common playing-related musculoskeletal disorders (PRMDs) of musicians include overuse problems, such as tendonitis, and peripheral nerve entrapment syndromes[27].

In musicians, repetitive movements at the wrist such as flexion, extension, radial and ulnar deviation of the wrist may cause tenosynovitis of the long flexor tendons, which in the long-term contribute to the formation of tendinous interconnections mainly within the carpal tunnel[28]. These interconnections can either be fine tendinous linkages or strong adherent sheets of tenosynovium, both of which may be very resistant to stretch. Tenosynovium helps with the smooth gliding of the tendon with the least amount of friction. Interconnections at the level of tenosynovium act as adhesions that prevent movements of the tendon and causes tension during finger movements. Whilst playing a musical instrument, these may become the potential sites for pain and inflammation[29]. The postulated pathophysiology for development of tendinous interconnection is discussed in detail in subsequent sections.

The risk factors that have been identified for PRMD include the type of instrument that the musician frequently plays, their gender, age, duration and intensity of playing, and individual physical characteristics such as the hand size[30]. Abréu-Ramos and colleagues[32] (2007) demonstrated that PRMD is more common in adult musicians (mean age: 22-29 years) than in adolescents as this age group played the musical instrument for longer hours and with fewer breaks in between playing (28.7 hours/week). Similarly, in 1992, Pratt and colleagues identified that the prevalence of PRMD was approximately 39% to 47% in adults compared to 17% in secondary school music students. They also found that PRMD is more common in female string- and keyboard players compared to their male counterparts. Some of the proposed theories concerning the difference are smaller hand size along with decreased arm strength and more flexibility and joint laxity of the hand in females. However, these theories have not been investigated sufficiently to derive concrete conclusions[31]. There is strong evidence to suggest that the musicians who play an instrument for more than four hours a day and more than 60 minutes without a break are more prone to clinical signs and symptoms of PRMD[33].

Prevention of PRMDs includes recognition of both internal (e.g., musicians strength and flexibility of the musician's body) and external factors involved (e.g., anatomical and functional position whilst playing an instrument, instrument size and techniques of holding the instrument involved); that is, the interface between the musicians, their instruments and the playing environment (e.g., rest breaks or hours of practice)[35].

Common types of tendinous interconnections in musicians

In 1979, Richard Linburg and Brian Comstock identified an anomalous interconnection between the FPL and FDP of index finger at the level of carpal tunnel that restricted independent flexion of these digits (as seen in **Figure 2**). The patient presented with pain in the distal forearm. Now popularly known as the ***Linburg-Comstock anomaly,*** it is characterised by simultaneous flexion of the distal IP joint of the index finger with flexion of the IP joint of the thumb and the inability to actively flex the IP joint of the thumb without simultaneously flexing the distal IP joint of the index finger (as seen in **Figure 3**)[36]. Linburg-Comstock syndrome causes functional impediment in musicians[37] by reducing the independent movement of the FDP (index) when the thumb is flexed[38]. The presence of these interconnection and similar anomalies have been subsequently identified and reiterated by a number of studies both amongst musicians and non-musicians[39].

Any movement against interconnection causes pain (usually of tearing in nature) on the palmar aspect of the hand, radial side of the wrist or in the distal part (mainly the distal one-thirds) of the forearm[2]. These interconnections have been identified just proximal to the radiocarpal joint or the distal forearm[37].

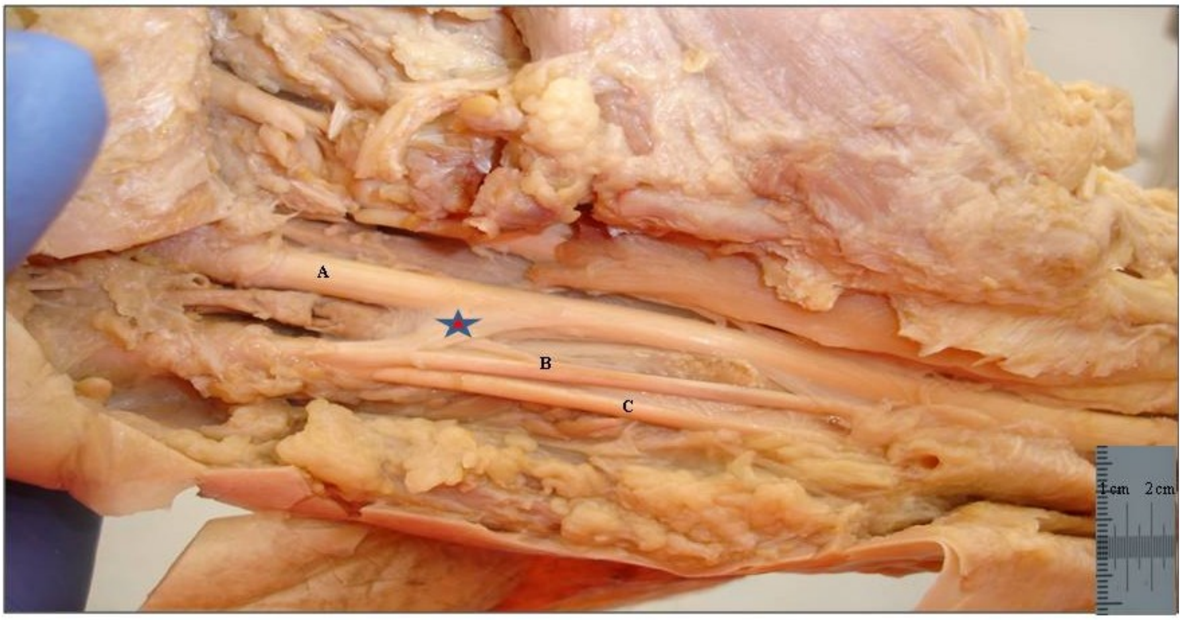

Figure 2 **:** Demonstrating a tenosynovial interconnection (★) between the Flexor Digitorum Superficialis (ring finger) (A) and Flexor Digitorum Superficialis (little finger) (B). Also seen is Flexor Digitorum Profundus (little finger) (C).

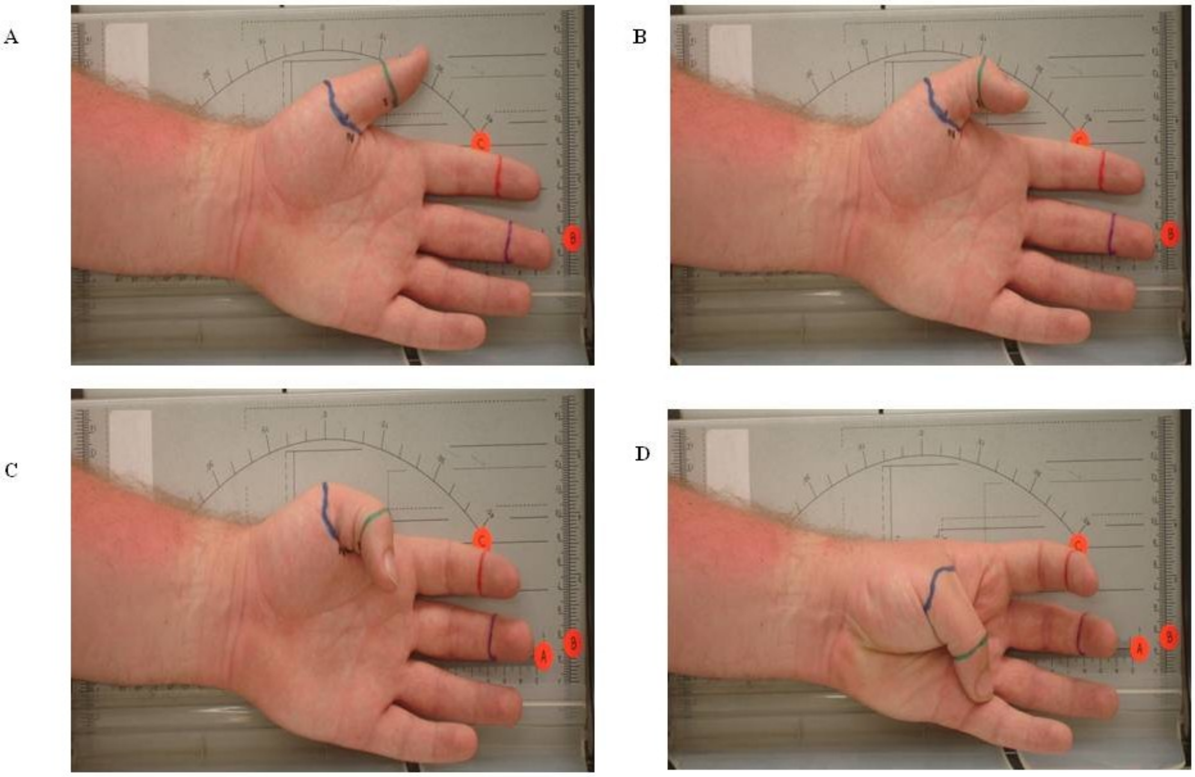

***Figure 3*: Demonstrating the angle of flexion of the thumb and the dependent fingers (A) at rest, (B) initial movement, (C) mid position and (D) fully flexed at mid prone position of volunteer.**

Miller and colleagues[2, 35] (2002 and 2003) have classified the manifestations of FPL and FDP interconnections. This classification is based on the level of the interconnection and the degree of mechanical impediment. These are: (1) synkinesis (involuntary movement of hand or muscles associated with a voluntary movement) (2) synkinesis and positive Linburg-Comstock test results (i.e., pain and discomfort during flexion of the thumb to the base of the little finger while the index finger is held in extension by the examiner), and (3) pain and deformity following continuous use. They also observed that amongst string players, the symptoms were more prevalent in the left hand and this tended to decrease from the radial to the ulnar side of the hand. This is due to the fact that the left hand is involved in free finger and thumb movements when playing bowed or string instruments. The static thumb posture with active finger movements, as in string instrument players, leading to tenosynovitis over time has been suggested to lead to this anomalous interconnection[2].

Discussion

The recognised concept of a synovial sheath (tenosynovium) is that of a two-layered structure surrounding the tendon with the presence of lubricating fluid between these layers. The inner layer holds on to the tendon, and is attached by areolar tissue to the outer layer that adheres to the surroundings. These layers slide relative to each other and the loose connective tissue sandwiched between them is stretched only with larger than physiological tendon displacements. The purpose of the sheaths is to reduce friction of the tendons relative to the environment or other tendons, and they are constructed in a manner that allows nerves and vessels to reach the tendons undamaged. However, within the carpal tunnel, the synovial sheaths comprise many layers of thin membranes (in contrast to the 'classical' two layered tendon sheaths arrangement (e.g., in the digits), the synovial mass in the carpal region will be referred to here as 'synovial membranes'[39]. These membranes enclose all flexor tendons (FDS, FDP and FPL) collectively as well as the individual tendons. Synovial membranes, in general, do not adhere to the superficial tendons, except sometimes in the case of those of the little finger as numerous thin tendon strands[39]. However, within the carpal tunnel they often adhere strongly to the deep flexor tendons. When they are attached to two adjacent tendons, they may form interconnections.

There are differences in the morphology of the superficial and deep flexor tendons. The superficial flexor tendons have tightly packed tendon strands giving it a round and smooth appearance, while the deep flexor tendons (especially, the three on the ulnar side) are a collection of loosely packed tendon strands **(seen in figure 4).** However, distal to the distal border of the flexor retinaculum where the lumbrical muscles originate, the deep flexor tendons assume a round and smooth appearance. In the carpal tunnel where the deep flexor tendons are loosely packed tendon strands, the synovial membrane gets trapped between the individual tendon strands predisposing to the formation of interconnections[39]**.** The interconnection between the tendons of FPL and FDP (index) might be due to the anatomic proximity coupled with the large mean PCSA of the FDP tendons. In addition, although the FPL and FDP tendons are independent of each other, due to the common mesodermal mass from which these tendons are derived, the interconnection could occur as a congenial anomaly. These tendinous interconnections together with adhesive synovial membranes may provide strength to resist the *in vivo* forces that generate the opposite displacements of the connected tendons[39].

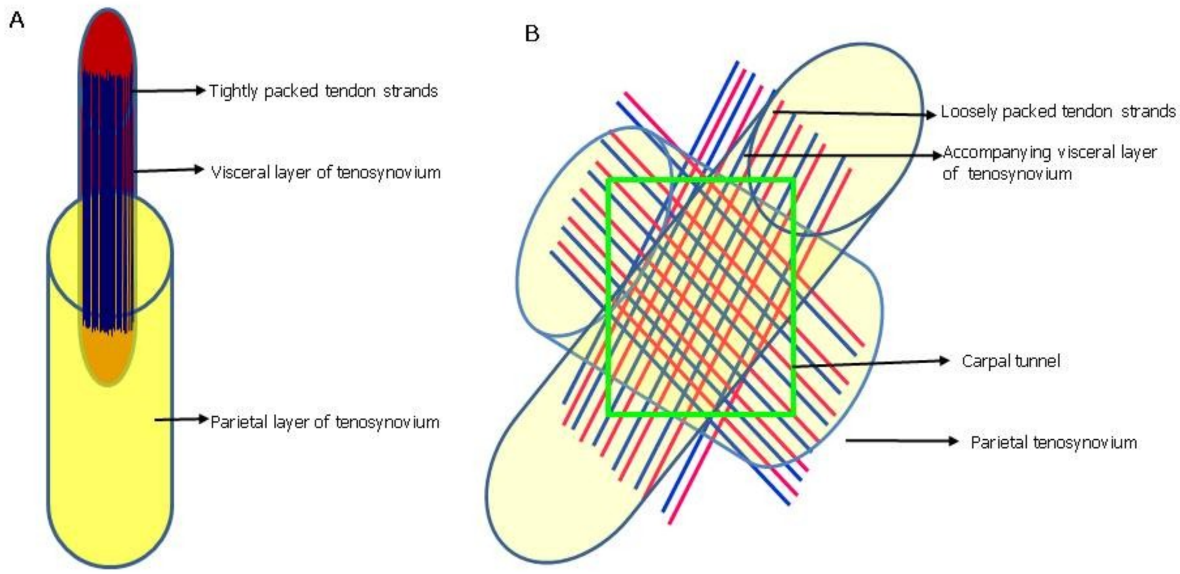

Figure 4: Illustrates the normal arrangement of the tendon strands of FDS and FDP tendons at carpal tunnel. **A:** The FDS tendon strands are tightly packed and better organised than the FDP tendons. **B:** The FDP tendon strands are loosely packed. The visceral layer of the tenosynovium recognises each tendon strand to be a tendon and wrap around them. At the carpal tunnel, there is criss crossing of these tendon strands and their accompanying visceral tenosynovium.

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