Original Research Article

SITE-SPECIFIC FASCIA TUNING PEGS AND PLACES OF PERILOUS PASSAGE MYOFASCIAL CONSIDERATIONS IN UPPER EXTREMITY ENTRAPMENT NEUROPATHIES: A CLINICAL ANATOMISTS VIEW

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ABSTRACT

The objective of this study was to identify common anatomical locations of densified fascia associated with axillary, musculocutaneous, median, ulnar and radial nerve entrapment. Additionally, a proposal concerning a tensegrity based expansive decompressive protective role of muscles and ligaments as ‘site-specific fascia tuning pegs’ is offered for consideration. This observational report provides a means to stimulate research into the dynamics of force transfer via tensegral mechanotransductive pathways possibly decompressing neurovascular structures. Morphological changes to fascia profunda, septal tissues, epineurium, perineurium and endomysial tissue in continuity with neural structures were noted. Entrapment neuropathies involving the upper extremity are a growing and widespread phenomenon within modern society. Upper extremity neuropathies affect dentists, athletes (professional and recreational), pianists, grocery store employees, office workers, cab drivers and a host of other professional and non-professional individuals. Neurovascular insults can develop at multiple sites referred to by anatomists as the three P’s [i.e. Places of Perilous Passage]. The complexity of the inter-communicating nerve network, known as the brachial plexus, is well described as are the referred pain patterns of the contributing terminal branches. Sensory innervation to the upper extremity includes most of the axilla while excluding a specific region of the medial upper extremity and axilla which is supplied by the intercostobrachial nerve [i.e. T2]. This observational study identified specific anatomical locations where increased fascial densification lead to reduced gliding of the various facial laminae due to densified, fibrotic or adhered fascial tissues. A new hypothesis emerged concerning “site-specific fascia tuning pegs” described as biological instruments [i.e. muscle fibers and ligaments] that modify the length and width of the various specialist neural and vascular tubes [i.e. epineurium, tunica adventitia]. This author hopes that providing this information will assist in improving diagnosis, treatment and prognosis of upper extremity neurovascular insults that result in pain or unpleasant changes in sensation.

KEY WORDS: Neuropathy, Fascia, Entrapment, Brachial Plexus, Tensegrity, Densification, Fibrotic, Site-Specific Fascia Tuning Pegs.

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metabolic systems necessary to provoke cellular homeostasis [2]. Muscle fibers undoubtedly create forces for ambulation and heat production, however, muscle fibers in continuity with fascia are hypothesised to be playing a role, to date not recognised, as ‘site-specific fascia tuning pegs’ [3]. The role of muscles, acting in additional fashion to movement generators, can clearly be appreciated when examining the fascial continuity of Rectus Capitis Posterior Minor [Fig 1]. Fascia of the Rectus Capitis Posterior Minor [RCPmi] has been demonstrated to be in continuity with the atlantooccipital myodural bridge attaching to the dura mater [DM]. This fascial continuity passes through the atlantooccipital interspace and becomes integrated as the outer dura mater of the spinal cord [6, 7].

Musculofascial structures provides the primary hydraulic source required for effective transportation of cerebrospinal fluid [7]. Furthermore, anatomical investigations of the Rectus Capitis Posterior Major [RCPma] have confirmed a similar fascial continuity with the Dural sleeve [9]. A comparable mechanism is proposed to be at play concerning site-specific fascia tuning pegs supporting normal neurovascular activity in the upper extremity. It is proposed that failure of these fascia tuning pegs to provide the necessary balance of tension and compression expresses dysfunction locally or more globally in anatomical locations known as Places of Perilous Passage [i.e. the three P’s].

Supporting the model of site-specific fascia tuning peg’s, it has been shown that muscle Rectus Capitis Posterior Minor, its fascia and associated ligamentous elements [e.g. To Be Named Ligament-TBNL], provide essential expansive tensioning to the Dural sleeve. Such fine tuning creates a shape change in the Dural sleeve [i.e. Thecal sac] conducive to reducing dural infolding and spinal cord [SC], impingement [8]. In addition, it has been proposed that contraction of the suboccipital muscle fibers

Upper extremity entrapment neuropathies:
The brachial plexus [i.e. C5, C6, C7, C8, andT1]. is a significant somatic nerve network of plexus formed by intercommunications amid the primary ventral rami of the lower 4 cervical nerves and the first thoracic nerve [8]. Comprised of roots, trunks, divisions, cords, and terminal branches the plexus is formed proximal to distal [9]. The lateral and medial cords of the brachial plexus merge to form the medium nerve traveling on the medial aspect of arm accompanied by the brachial artery and basilic vein [Fig 2]. Based on tensegrity focused dissections [10], bicipital aponeurosis and Lacertus fibrosis [Fig 3]. are hypothesised to play a site-specific fascia tuning peg role decompressing neurovascular structures passing on the medial triangular interval at the antecubital fossa. In this hypothesis the teres major muscle also provides expansive
forces to the neurovascular structures proximally [including the radial nerve] in partnership with coracobrachialis [whose muscle gastor is proximal to the elbow joint] as its contractile expansive forces act to decompress the mid to upper humeral fascial compartments.

Axillary nerve arises from the posterior cord of brachial plexus at C5, C6 ventral rami dividing into anterior and posterior divisions in the quadrangular space [11]. Axillary nerve extends to the inferior edge of subscapularis along the inferior aspect of the glenohumeral joint capsule. Posterior to humerus, axillary nerve courses around the surgical neck of humerus accompanied by posterior circumflex artery encased within the fascia profunda of deltoid [11]. Omnidirectional forces produced by deltoid may act to expand fascia on the surgical neck ensuring undue compression of axillary nerve and associated vascular structures. Muscles of the rotator cuff [12], commonly known as the SITS muscles, include Supraspinatus, Infraspinatus, Teres minor and Subscapularis. The fascia laminae of the SITS, and associated axillary muscles, including latissimus dorsi and teres major, blend in an omnidirectional arrangement similar to the hub of a bicycle wheel. Dissection identified multiple vectors with contributions from the fasciculi arrangement of the SITS muscles, pectoralis minor, coracobrachialis, deltoideus, trapezius, biceps brachii, triceps brachii, latissimus dorsi, teres major and pectoralis major [10].

It is proposed that these myofascial laminae coordinate forces as site-specific fascia tuning pegs reducing compressive or tensional insult at places of perilous passage (i.e. coracoid process). Fascial fine tuning ensures the natural gliding, expansion and decompression of the glenohumeral joint and axilla in three-dimensional fashion. To that end, it is proposed that fascial structures, including muscle fibers and ligaments, may operate as local or synergistically global fascia tuning pegs. To underline and better appreciate this assertion, dissecting the brachial plexus including the path of ulnar nerve was completed in the dissection laboratory [10]. Ulnar nerve arises from the medial cord of the brachial plexus at the spinal levels C8, T1. Traveling the length of the humerus on its medial border, ulnar nerve enters an anatomical region slightly posterior and superficial to the medial intermuscular septum. This densified retrocondylar retinaculum tissue, recognised as the Arcade of Struthers, contains thickened fibrous dense fascial tissue found at variable distances proximal to the medial epicondyle [10].

![Fig. 3a & 3b: Lacertus fibrosis, superficial and deep aponeurotic expansions are hypothesised to play a vital site-specific fascia tuning peg role in decompressing the deeper neurovascular structures (e.g. median nerve) passing on the cubital fossa and the distal portion of the Arcade of Struthers and Osbourne’s ligament. Lacertus fibrosis spans in omnidirectional fashion in continuity with the Arcade of Struthers, and Osbourne’s ligament, blending into the antebrachial fascia of the volar forearm. Supporting this expansive synergy is the intermuscular septa of muscle pronator teres flanked by common flexor tendon, and humeral head of the flexor carpi ulnaris [13]. It is proposed that failure of the Lacertus fibrosis to provide appropriate tension could lead to densification of the sublime bridge located deeper in the proximal forearm representing a place of perilous passage specific to ulnar nerve. Median nerve compression also known as ‘eye of the hand’ neuropathy has also been implicated as a result of sublime bridge compression [14]. Median nerve is of vital importance to overall wrist and hand function. Loss of nerve function results in inability to control thumb abduction, wrist flexion, flexion of the digits as well as sensory depreciation in digits one to three, radial half of digit four and palmar cutaneous sensation [14]. A mononeuropathy entrapment of median nerve results from excessive compression of the median nerve within the carpal tunnel [15]. Dissection of the
palmaris longus highlights the criss-cross inter-fascial relationship its tendon shares with the median nerve along its route. In all cadavers dissected for the purpose of this investigation the tendon of palmaris longus terminated in the palmar aponeurosis.

The proximal tendon of this superficial fusiform spindle shaped flexor is associated with the medial epicondyle of humerus continuous with the antebrachial fascia of forearm.

The median nerve announces its arrival at the palm, between the lateral and medial styloid processes. Undercover of the palmar aponeurosis median nerve runs posterior to the tendon of palmaris longus, located outside the carpal tunnel. Lightly placing fingertips above the palmar aponeurosis while gently providing traction to the gastor of palmaris longus resulted in a sensed lift, and expansion, relieving structures deep to palmar aponeurosis overlying the carpus. This observation supports the proposal of palmaris longus as a site-specific fascia tuning peg reducing decompressive forces on the carpal tunnel, and hence, the medium nerve.

Ulnar nerve originates from brachial plexus ventral rami C8,T1, with contributions occasionally from C7, and terminating in the distal phalanges of the fifth and half of the fourth digit, while innervating forearm, wrist and hand along its anatomical path [16]. Ulnar nerve runs superficially in the medial arm [Fig 5]. Traveling in close approximation to the medial tendon of Triceps brachii passing through the cubital tunnel, a bony trough, composed of the olecranon process and medial epicondyle. Places of perilous passage include the medial intermuscular septum as Ulnar nerve travels posteriorly to cubital tunnel, the arcade of Struthers and onwards to Guyon’s canal prior to hook of hamate [17]. On entering the forearm, ulnar nerve traverses flexor carpi ulnaris running superficially upon the oblique ulnar collateral ligament [18].

Fig. 4: Placing traction on the distal tendon of Palmaris Longus highlighted the continuity of fascia and the transfer of forces by means of mechanotransduction via the epimysium. (Image Sharkey, J 2012).

Fig. 5: Ulnar nerve runs superficially in the medial arm. Traveling in close approximation to the medial tendon of Triceps brachii passing through the cubital tunnel, a bony trough, composed of the olecranon process and medial epicondyle.

The humeral attachment of pronator teres, traveling within the cubital fossa, in conjunction with Lacertus fibrosis is considered a site-
specific fascia tuning peg providing a tenserigal expansion at the medial supracondylar ridge of humerus. Should this expansive fascial facilitation fail to occur, ulnar, median and interosseus nerve, with associated vasculature, may become irritated resulting in pain, changes in sensations and loss of function [10]. Changes due to hypertonicity or inhibition may lead to inappropriate tensional and compressional forces with subsequent morphological changes, possible densification of ligamentous structures and dynamic ischemia [18]. Fibers of the biceps brachii and brachialis muscle contribute forces to the deep antebrachial fascia diving to the depths of the floor of the cubital fossa (i.e. elbow pit). The cubital fossa contains the brachial artery and median nerve, with the ulnar and radial nerve within the specific anatomical vicinity but not directly within the cubital fossa. Pulling on the humeral tendon of brachialis muscle allowed for observation of the mechanical effect, felt with fingertips, downstream within the antebrachial fascia. It was observed that the distal attachment of brachialis associates with the tuberosity of ulna, specifically the ventral surface of coronoid process, by means of a splayed or pyramid shaped aponeurosis. Distribution of forces through this thick fan-shaped fascial structure is hypothesised to assist decompression at the internervous plane.

As a major nerve of the upper limb musculocutaneous nerve is an extension of the lateral cord of the brachial plexus containing fibers from the C5, C6, and C7 spinal nerve roots. Musculocutaneous nerve innervates biceps brachii, brachialis and supplies sensory branches to integument over the lateral cubital and forearm regions via the lateral antebrachial cutaneous nerve [19]. It is proposed that the specific location of the proximal portion of the coracobrachialis muscle supplies contractile forces resulting in fascia decompression of the neurovascular sheaths associated with the coracoid process. The coracoid process, viewed as an osseofascial hub, provides the interface for mechanotransductive integration required for the facilitation of omnidirectional expansive forces. Such forces are proposed to act to protect the integrity of the neurovascular structures in the proximity of the axilla in an omni-directional fashion. It is further proposed that inhibition or spastic activity of the coracobrachialis would change the tensional-compressional relationship required for optimum neurophysiological function within the axilla.

CONCLUSION

The author hopes the introduction to site-specific fascia tuning pegs will motivate a discussion concerning the treatment of spastic versus inhibited myofascial tissues in upper entrapment neuropathies. Appreciating the tenserigal balance of forces leading to appropriate or inappropriate tension and compression informs a range of professionals regarding the possible range of therapeutic interventions. It is widely accepted that hypertonic tissues inhibit muscles, and other contractile tissues, which are acting in synergy. Before surgical intervention is considered less invasive manual and movement therapy approaches should be investigated predicated on the hypothesis of site-specific fascia tuning pegs.

Conflicts of Interests: None

REFERENCES


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