

# Median nerve entrapment by variant anatomical structures

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## SUMMARY

Entrapment neuropathies are common and are frequently encountered by physicians in clinical practice. Median nerve entrapment, being one of the most common neuropathies in the upper extremity, must be studied in detail if the extent of injury it can cause is to be understood fully. Various anatomical variations are discovered frequently and reviewing these will advance medical practice in the search for suitable treatments. A broad understanding of the symptoms of median nerve entrapment, motor as well as sensory, is essential, along with its effects on surrounding structures.

**Key words:** Median nerve – Compression – Anatomical variations – Clinical significance

## INTRODUCTION

Nerves are highly susceptible to injury and often become vulnerable, leading to entrapment, which causes swelling and focal flattening (Jarvik et al., 2000). Any nerve is prone to injury and to entrapment between structures close to it. Entrapment or compression occurs when variant structures pinch or pressure the nerve (Miller and Reinus, 2010). Some peripheral neuropathies, which are typical clinical disorders, can be classified one of two ways: compressive entrapment and non-compressive entrapment (Spinner and Amadio, 2003).

To explain the clinical findings of nerve entrapment, it is essential to understand the broad range of neuropathies usually presenting as sensory abnormalities, including pain, paresthesia, and loss of sensation or numbness. Another possibility is motor weakness in the muscles innervated by that particular nerve (Georgiev and Jeleu, 2009a; Slavchev and Georgiev, 2013).

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Entrapment neuropathies are common and are frequently encountered by physicians in their clinical practice (Hobson-Webb and Juel, 2017). They can be mononeuropathic or polyneuropathic. A classic example of a condition causing neuropathy is diabetes (Rota and Morelli, 2016). While most mononeuropathies are superimposed on a polyneuropathic background, they require knowledge of neuroanatomy and clinical diagnosis (Hobson-Webb and Juel, 2017).

The aim of this study is to summarize for clinicians the different anatomical variations that can provoke median nerve (MN) compression.

## THE MEDIAN NERVE

The MN is one of the most substantial nerves of the upper limb. It contains fibres from all the nerve roots of the brachial plexus (C5 to Th1). It innervates the flexor muscles of the anterior compartment of the forearm (except for the flexor carpi ulnaris and the medial part of the flexor digitorum profundus (FDP), since these two are innervated by the ulnar nerve) (Spinner, 2003). Within the hand, it innervates the thenar muscles and the two lateral lumbricals. The sensory function of the median nerve is quite easy to deduce considering its location. It has a palmar cutaneous branch, which supplies the lateral part of the palm, and a digital cutaneous branch, which supplies the skin over the first two and half fingers and the thumb (Meyer et al., 2018).

Because of its location in the arm, the nerve sits ventral to the brachial artery. As it descends further proximally, it crosses over to become more medial and then enters the elbow at the cubital fossa. This is where it passes into the anterior “superficial” compartment of the forearm. Within the forearm, it travels between the FDP and the flexor digitorum superficialis muscle (FDS), and two major branches emerge. The proximal branch is the anterior interosseous nerve (AIN), which supplies the deep flexor muscles of the anterior compartment of the arm. These muscles include the pronator quadrates, flexor pollicis longus (FPL) and the lateral half of the FDP. The superficial and intermediate layers are directly innervated by the MN. The pronator teres (PT), flexor carpi radialis

(FCR) and palmaris longus (PL) are muscles of the superficial layer supplied by the MN. The flexor carpi ulnaris is innervated by the ulnar nerve.

The distal branch of the MN, called the palmar cutaneous nerve, passes over the flexor retinaculum. It innervates the skin of the lateral palm. It enters the hand and divides into a recurrent and a common palmar digital branch. The recurrent branch supplies the thenar muscles and passes between the flexor pollicis brevis and abductor pollicis brevis and then supplies the opponens pollicis. The palmar digital nerves supply the lateral two lumbricals of the hand. The common palmar digital nerves divide into two to form the proper palmar digital nerves and supply the lateral three and a half digits (Clemente, 1985).

## MEDIAN NERVE ENTRAPMENT

An insight into the location and course of the MN makes it easier to locate the regions where the nerve can become entrapped. Much research on this topic has established that there are several regions in the upper limb where the MN can suffer compression (Andreisek et al., 2006; De Smet, 2002).

### Coracobrachialis and brachialis muscles

Different variations of the coracobrachialis muscle (CB) that pass over the MN can be involved in MN neuropathy: four-headed CB, CB longus and coracoepitrochlearis muscle (El-Naggar and Saggaf, 2004; Georgiev et al., 2017; 2018a, b; Olewnik et al., 2020). Other possible causes of MN compression are accessory slips from the brachialis muscle that cross over the nerve (Bilecenoglu et al., 2005). George and Nayak (2009) presented a case in which the MN and brachial artery appeared normal in the upper part of their course in the arm, but in the lower 1/3rd they both lay deep to the accessory slip of the brachialis instead of passing superficial to brachialis as normal.

A cause of this kind could have greater clinical significance, but as Georgiev et al. (2017) point out, the lack of clinical reports could be explained, on the one hand, by the rarity of these variations and lack of knowledge of them, and, on the other, by the limited skin incision performed during

decompression. The coracobrachialis longus can compress the median nerve at a different level (Olewnik et al., 2020).

### Ligament of Struthers and supracondylar process

The ligament of Struthers and the supracondylar process (Fig. 1a) are found in the distal 1/3 of the medial aspect of the arm. The presence of these in humans is a rare legacy from earlier species. They are reported to interfere with the course of the MN (Pratt, 2005).

### Supracondylar process syndrome

The rarest of all compression neuropathies of the MN is supracondylar process syndrome. Among all compression neuropathy cases, only about 0.5% are attributable to this syndrome (Meyer et al., 2018; Georgiev and Tubbs, 2020).

Struthers described the supracondylar process and its associated ligament in 1848. It is a beak-shaped bone spur arising in the distal anteromedial portion of the humerus. Congenitally, this variation is seen in 0.1% to 2.7% of people and is usually asymptomatic. It is sometimes possible for the CB to attach to the supracondylar process. The process is said to be easily visible on oblique radiographs but is often missed on anteroposterior and lateral images (Opanova et al., 2014).

Following a fracture, the MN can be compressed by the osteofibrous structures lying in this space. Symptoms appear on dynamic examination of the elbow (Wertsch and Melvin, 1982). Compression occurs at the level of the distal humerus as the MN passes under the bony curvature, continuing to the median epicondyle. Muscle hypertrophy or strenuous use can aggravate the irritant effect of this structure (Spinner et al., 1991). A supracon-

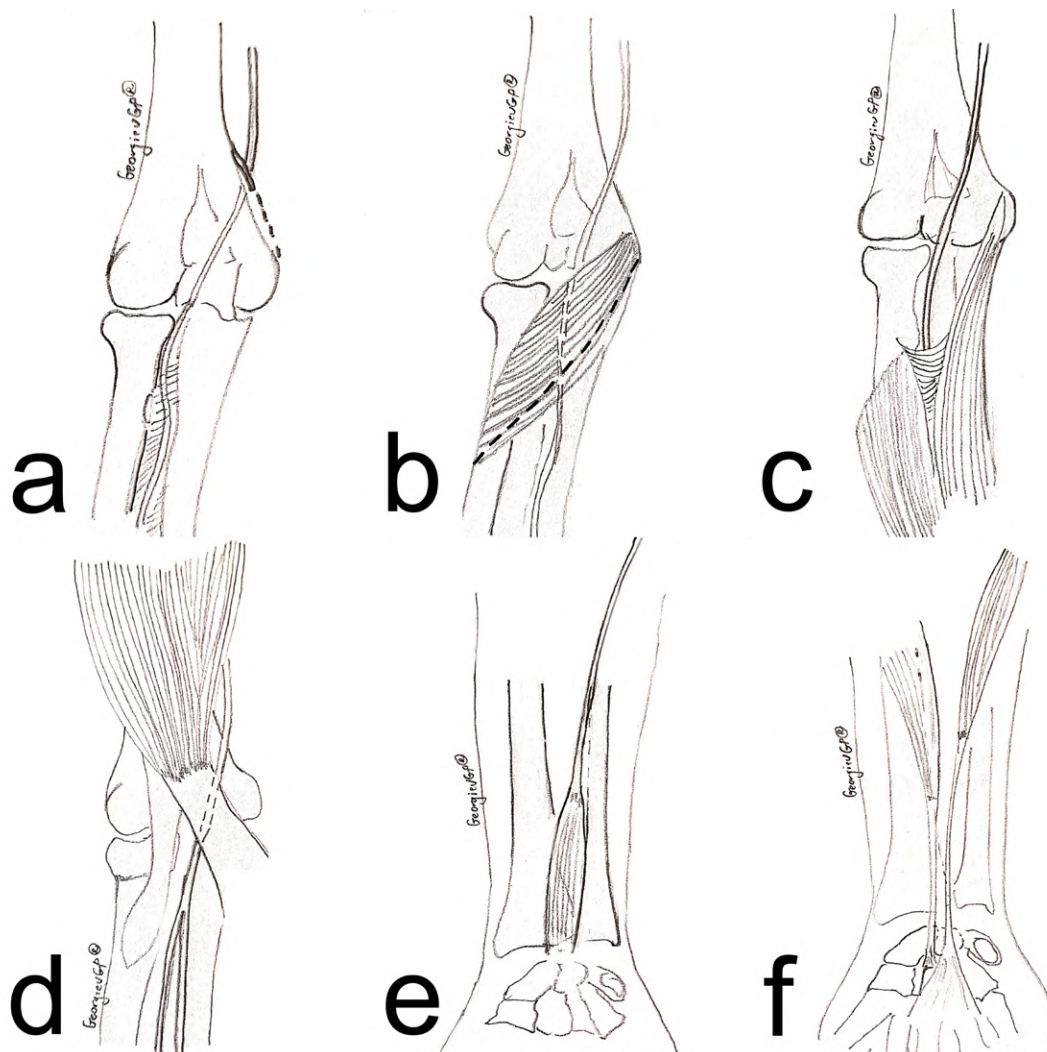


Fig. 1.- Possible sites of compression of median nerve. **a)** Supracondylar process and Ligament of Struthers; **b)** Pronator teres; **c)** Fibrous arcade at the proximal margin of the flexor digitorum superficialis; **d)** Lacertus fibrosus; **e)** Reversed palmaris longus; **f)** Palmaris profundus.

dylar process can be found by X-ray (Shon et al., 2018).

### **Ligament of Struthers**

The MN is rarely entrapped by the Struthers' ligament, which connects supracondylar process to the medial epicondyle. The MN and the brachial artery pass through a small arch. Electromyography is useful in such cases to confirm compression (Sener et al., 1998).

It is usually asymptomatic but is known to produce symptoms post-trauma (Shon et al., 2018). The patient's history often includes avoiding activities that typically involve forearm, wrist and/or finger extension. On examination, discomfort during forearm supination and elbow extension is aggravated. The radial pulse can also be attenuated. Muscle weakness has been noted in the pronator teres and this is the hallmark of pronator syndrome (Shon et al., 2018). Sener et al. (1998) presented a case of a 35-year-old woman with a two-year history of pain and paresthesia involving her right elbow. Symptoms seemed to be worsening during elevation of the hand upwards or active extension of the elbow and pronation of the forearm. Direct radiograms led to a clear diagnosis of supracondylar process without Struthers' ligament on MRI. However, surgical exploration revealed both a supracondylar process and a Struthers' ligament.

### **Bicipital aponeurosis (lacertus fibrosus)**

The bicipital aponeurosis (lacertus fibrosus) (Fig. 1d) is described accurately as a ligamentous sheet just past the elbow joint. It originates from the tendon of the biceps muscle and the flexor-pronator fascia. Along with the MN, the brachial artery passes through the cubital fossa under the lacertus fibrosus (Bilecenoglu et al., 2005).

The most common presenting symptoms are loss of key and tip pinch strength accompanied by loss of fine motor skills and a sense of clumsiness. It causes dropping of objects and, rarely, transient paresthesia in the MN-innervated region of the hand (Lalonde, 2014). There is decreased power in the FPL, FDP and FCR and, rarely, a positive Tinel's sign. In most cases the main complaint is loss of motor function, seldom loss of sensory function.

The initial treatment consists of bivalve cast splinting. If this treatment fails, the patient is indicated for surgery to release the tight lacertus fibrosus that is compressing the MN (Swiggett and Ruby, 1986).

There are rare causes of MN compression due to the lacertus fibrosus, possibly involving hemorrhage in the cubital fossa (Johnson and Melvin, 1967). Caetano et al. (2017) presented a study analyzing anatomical variations of the lacertus fibrosus and their implications for MN compression. They dissected sixty upper limbs of 30 cadavers, 26 male and four female. Fifteen of them had been preserved in formalin and glycerol; 15 were fresh. The results revealed that in 55 limbs, the short and long heads of the biceps brachii muscle contributed to the formation of the lacertus fibrosus. There was a significant contribution from the short head. In 42 limbs there was a thickened lacertus fibrosus, suggesting this could be a potential factor in compression and entrapment.

### **Pronator Syndrome**

This is a comparatively rare syndrome resulting from entrapment of the MN between the humeral and ulnar heads of the PT (Lee, 2014; Eversmann, 1983). Spinner (1991) called it a controversial disorder because the symptoms are vague; discomfort in the forearm with occasional radiation into the arm, often described as fatigue-like pain. Secondary symptoms include numbness in the hand. Women are at a greater risk than men of developing symptoms such as little finger numbness, especially if they are exposed to repetitive industrial movements. The imaging commonly used for diagnosis is electromyography (Spinner, 1991).

Pronator syndrome can be caused by hypertrophy of the PT muscle or by congenital abnormalities (Fig. 1b). A high origin of the PT from the humerus is a rare variation. Commonly, this muscle starts from an existing supracondylar process and from a fibrous band extending between the supracondylar process and the medial epicondyle, the ligament of Struthers. Between this ligament and the distal part of the humerus an arch is formed through which the MN and brachial artery pass distally; in rare variants, the ulnar artery or nerve also passes through it (Jelev and Georgiev, 2009).

Clinical presentations are pain and tingling in the volar aspect of the elbow, forearm and wrist as the prime symptoms, all without muscle weakness. The wrist flexion test, called the Phalen test, shows negative results (Wertsch and Melvin, 1982). Either a Struthers' ligament or lacertus fibrosus level compression can be suspected if the symptoms of pain or weakness are aggravated by flexion of the elbow against resistance, usually between 120 and 135 degrees. Pronator syndrome is suspected only when the symptoms are aggravated by resistance to pronation of the wrist. Another scenario common in this context is irritation due to resisted flexion of the FDS of the middle finger. The arch of the FDS must be examined carefully (Eversmann, 1983; Hagert, 2013).

If pronator syndrome is diagnosed, the MN is thoroughly examined. There are four possible sites of compression of the MN at the pronator teres muscle: (1) around 4 cm above the medial epicondyle of the humerus, a small hook-shaped bony process called the supracondylar process, which acts as accessory origin of the pronator teres. This is the ligament of Struthers; (2) the fascia of the lacertus fibrosus, which courses from the bicipital tendon over the mass of proximal forearm flexor muscles; (3) reflections of muscle fascia forming fibrous bands that form the deep head of the PT, which has a sharp aponeurotic edge, or just simple hypertrophy of the muscle itself; (4) the tendinous aponeurotic arch of the radial attachment of the FDS (Fig. 1c) under which the MN nerve can be compressed (Eversmann, 1983).

The MN can be compressed because of morphological variability of the pronator teres (humeral and ulnar heads). If only the humeral head is present, the nerve passes underneath it. If there are two heads, the nerve passes between them, which predisposes it to compression. Sometimes a PT has two heads and the nerve passes behind them (Olewnik et al., 2018).

### **Anterior interosseous nerve syndrome**

Another syndrome that is common owing to entrapment of a branch of the MN is anterior interosseous nerve syndrome (AINS). It is also referred to as Kiloh-Nevin syndrome. It occurs

when the nerve is compressed in the proximal forearm. There could be direct nerve trauma or compression caused by a hematoma or a mass or tumor. The manifestation of this syndrome is vague pain in the proximal part of the forearm, typically triggered by exercise and subsiding on rest (Eversmann, 1983; Miller and Reinus, 2010). AINS has no characteristic sensory signs and symptoms. There is weakness of the FDP and pronator quadratus, sometimes even paralysis upon clinical examination. Common initial symptoms include a deep, unrelenting pain in the proximal forearm, which then leads to a lack of dexterity or weak pinching ability. This syndrome is often confused with Parsonage-Turner syndrome. EMG reveals fibrillations in the affected muscles and MRI studies are informative (Spinner, 1991).

Much research related to surgical procedures and exploration of the course of the MN indicates that reflecting the superficial head of the pronator teres and even the deep head at the radius enables the distal portion of the MN to be visualized. The FDS can also be reflected. In such cases, the origin of the FDS is separated, enabling us to view the entire MN. It is superficial to the anterior interosseous nerve. The potentially aberrant muscles that have been identified as causing compression neuropathy of the anterior interosseous nerve are: (1) the accessory head of the flexor pollicis longus, also called Gantzer's muscle; (2) the flexor carpi radialis brevis (Eversmann, 1983).

### ***Flexor pollicis longus (FPL)***

Gantzer's muscle originates from the medial epicondyle or the coronoid process, lying on the ulnar side of the FPL. In supination, the anterior interosseous nerve is compressed (Bilecenoglu et al., 2005). Gantzer identified two different variant muscle bellies in 1813. These are parts of the deep flexor region of the forearm and insert into either the FDP or the FPL. They were named after Gantzer himself. Atavism in action; the accessory heads of the FDP and FPL show incomplete division of the deep layers of the muscles (Saxena et al., 2013).

Al Qattan (1996) studied 25 right upper limbs and documented the incidence, origin, insertion, nerve supply and relationships of Gantzer's

muscle. The said muscle was present in 13 of the 25 cadavers. The anterior interosseous nerve supplied it in all of the specimens. It originated from the medial humeral epicondyle in 11 of the cadavers. In the other two, there was a dual origin from medial epicondyle and the coronoid process of the ulna. Insertion was into the FPL in the ulnar part. Although there was no close relationship to Gantzer's muscle, the MN passed between the superficial and deep heads of the pronator teres in 11 of the 13 specimens. In the remaining two, the MN was closely related to Gantzer's muscle because it passed deep to the deep head of the PT in one of the cadavers (Al Qattan, 1996).

### ***Flexor carpi radialis brevis (FCRB)***

There is a growing trend towards treating distal radius fractures with volar plating, so it is useful to have somewhat deeper knowledge of the FCRB. This muscle has been described in the Japanese literature in multiple cadaver studies. Its reported incidence is 2.6% to 7.5%. Insertion of the FCRB is variable; it can insert into any metacarpal base except the first or fifth or the carpal bones on the radial side such as the scaphoid, trapezium, trapezoid and capitate. The FCRB could potentially compress the anterior interosseous nerve, but since the site of compression is very distal to its branches supplying the FPL and FDP, it rarely becomes clinically significant enough to show symptoms (Eversmann, 1983).

### **Carpal Tunnel Syndrome**

The most frequently encountered MN compression syndrome is carpal tunnel syndrome (CTS). Most cases are idiopathic, with a range of etiologies that include trauma, conditions associated with imbalance of hormones, or metabolism. Other physiological causes of CTS are hemodialysis, obesity, lupus erythematosus, scleroderma, thyroid disorders and amyloidosis. The syndrome can possibly be caused by the direct influence of external forces, either vibration or direct pressure (De Smet, 2005; Meyer et al., 2018).

Aberrant muscles can cause syndromes of MN entrapment (Georgiev, 2020; Georgiev, 2021a; Georgiev, 2021b). The usual symptoms include restricted movement of the hand accompanied

by burning pain in the distal part of the forearm. Such anatomical variations can also cause painful compartment syndrome owing to lack of space (Agarwal et al., 2014).

On reviewing the literature, three muscle variations emerge: (1) 1st or 2nd lumbrical; (2) PL and its anatomical variants; (3) superficial flexor of the index finger (Miller, 2010).

#### ***1st or 2nd lumbrical***

Since the MN supplies motor innervation to the 1st and 2nd lumbricals, any variation in these could affect the MN and cause compression or entrapment.

*1st lumbrical:* Different variations of this muscle can be summarized: an accessory slip can arise from the FPL tendon, the FDS tendon, the first metacarpal opponens pollicis or the palmar carpal ligament. Cases have been reported where a fasciculus arose from muscular belly of the superficial or deep flexor and joined the 1st lumbrical. The 1st lumbrical can also be doubled, one being normal while the second arises from the FDS. A slip from the FPL giving a tendon to the 1st lumbrical has also been described. A supplementary head for the 1st lumbrical originating from the 1st palmar interosseous has also been found (Bergman et al., 2021).

*2nd lumbrical:* It is possible for the second lumbrical to arise from the two tendons between which it is present. A doubling of the second lumbrical has been reported, one slip sent to the radial side of the middle finger and one to the ulnar side of the index (Bergman et al., 2021).

Redondo et al. (2011) presented a case of a 52-year-old woman with pain and paresthesia in the left MN several months after a carpal tunnel release. Surgical revision after follow-ups of the echogram revealed an aberrant muscle with proximal origin from the forearm and muscular belly passing under the carpal tunnel inserting into the first lumbrical. Bhandari and Palazzo (2017) also presented a case of an accessory lumbrical muscle found during carpal tunnel decompression. A muscle in the carpal tunnel was discovered superior to the FDS. Pulling this muscle led to flexion at the proximal interphalangeal joint of the index

finger. Sbai et al. (2019) described a 35-year-old left-handed woman who developed numbness, tingling, pain and weakness in the left hand affecting the thumb, index finger and middle finger; she had no prior history of such symptoms, which were aggravated by exercise. Surgical exploration immediately revealed an abnormal lumbrical tendon, which was easily exposed on opening the mid-palmar fascia and flexor retinaculum. The MN looked flattened by the tendon.

### ***Variations of the palmaris longus muscle (PL)***

There are many potential changes or variations in the regular anatomy of the PL. There can be reversed PL (Fig. 1e), bifid, trifid reversed and RPL coexisting with ADM. A digastric PLM or a PLM with intermediate muscle belly are also possible. Absence, duplication, and triplication are all tangible possibilities. There are accessory slips to the hypothenar muscles and PLM profundus (Fig. 1f). The latter is one cause of entrapment and congestion of the MN (Georgiev et al., 2009a, b; Kotov et al., 2017; Georgiev et al., 2017).

Surgical exploration infrequently reveals variant muscles in the carpal tunnel. It can be challenging to identify or classify them and they can cause confusion as they usually obscure the anatomy that is considered normal, posing a dilemma to the surgeon. Acknowledging that they can be present removes the doubts (Bhandari and Palazzo, 2017). A case study by Park (2019) revealed statistics on how many people could develop muscular anatomical variations of the volar aspect of the wrist because of CTS. Among 973 wrists in 644 patients, eight wrists in eight patients presented with variant muscles. They were presumed to be PL tendon variants or accessory ADM muscles. Early recognition of these anatomical variations can help to avoid unnecessary surgery and ensure better recovery among patients (Park, 2019).

Ninković et al. (1995) presented a case of a 28-year-old right-handed male lumberjack admitted to hospital for pain, tingling and numbness of the right thumb, index and middle fingers. Several days earlier, while using a power saw, the patient experienced sudden pain in the right wrist and noticed a concomitant swelling along the right forearm. Treatment provided temporary relief but the

symptoms continued, leading to further exploration of the swelling. A hypertrophic RPL was discovered as the cause of acute MN compression.

Bhandari and Palazzo (2017) presented a case of a palmaris profundus found during surgery on a 24-year-old male for a crush injury to the hand. The tendon to his right ring finger was injured as well as the MN. This variant muscle originated from the distal end of the radius, passing through the carpal tunnel and fusing its tendon with the FDS tendon to the little finger. The patient had no symptoms so the muscle was not resected. Sbai et al. (2019) presented another case of a palmaris profundus tendon in a 25-year-old female who for many years had experienced paresthesia and numbness in the territory of the MN of the left hand. Investigation revealed an aberrant tendon on the anterior surface of the MN. The tendon had a deep insertion into the palmar aponeurosis (Sbai et al., 2019). These authors pointed out that a variant muscle can be supposed to cause compression neuropathy in a patient in the “usual” age group, with symptoms aggravated by physical activity.

### ***Variant muscle belly of flexor digitorum superficialis***

The kind of variant discussed here is not very rare and is usually accompanied by such symptoms as hand tremors. The most common variant muscle belly arises from and inserts into the FDS tendon, and the action is typically on the index finger (Vichare, 1970; Smith, 1971; Das and Brown, 1975, Elias and Schuller-Ellis, 1995).

Another not so common muscle belly variant originates from the transverse carpal ligament and inserts into the tendon of the FDS of the index finger (Wesser et al., 1969; Still and Kleinert, 1973). Tanzer (1959) found that the muscular part of the FDS occasionally extends distally into the carpal tunnel and can only be seen once the tunnel is decompressed (Figueiredo and Hooper, 1980). Baruch and Hass (1977) discovered that the muscle belly passing through the carpal tunnel traveled deep to the other FDS tendons in the forearm and was crossed by the MN.

Kono (2003) presented a case of a transscaphoid perilunate dislocation. The patient complained of

mild numbness in the three radial fingers. He was treated with closed reduction of dislocation and the symptoms cleared. He underwent surgery to fix the scaphoid and luno-triquetral and capitolunate joints. Two hours after surgery he complained of severe paresthesia of the radial three fingers and difficulty in flexing them. Another exploratory surgery showed the MN to be severely compressed between the transverse carpal ligament and the swollen muscle bellies of the FDS of the long and ring fingers within the carpal tunnel. A release was performed and the pain and paresthesia disappeared post-op. Sbai et al. (2019) presented a 65-year-old female suffering from paresthesia and numbness in the MN territory of the right hand, accompanied by a positive Tinel's sign and Phalen test. There were no signs of thenar atrophy. A hypertrophic FDS of the middle finger engaged with the carpal tunnel was identified. The MN was congested and release resolved all the symptoms. Boutasta et al. (2012) presented a case of a 38-year-old housewife with severe pain and paresthesia at the right wrist, worsening after activity. A variant long muscle belly arising from the FDS of the index finger was observed. It extended proximally into the carpal tunnel and the MN was compressed.

There are five types of FDS variants as proposed by Elliot (1999). Type I entails a belly that arises at the carpal level, inserting into the same FDS from which it arose. Type II has a variant muscle arising from the palmar fascia and the distal border of the transverse carpal ligament. In this case, it terminates with the normal tendon and the muscle is entirely in the palm. Type III was described as digastric and appearing in the palm, interrupted and even replaced by a fleshy muscle belly. Type IV originated in the forearm and passed under the flexor retinaculum. It then extended into the carpal tunnel. Type V is less common. It is considered to represent anatomical variations in the superficial muscle layer in the distal part of the forearm (Elliot et al., 1999; Boutasta et al., 2012).

### ***The sublime bridge***

A potential site for compression of the MN is the sublime bridge, described by Tubbs et al. (2010). Despite the scattered incidence, this possibility

must be considered. The sublime bridge is tendinous in most specimens and is intimately related to the MN and the anterior branch of the MN, which arises proximally to it. Usually, the history of a patient with this syndrome involves constant repetitive pronation and supination of the forearm along with pain and paraesthesia over the anterior part of the forearm (Tubbs et al., 2010).

### ***Role of median artery in CTS***

The median artery (MA) is found between the anterior surface of the MN and the deep surface of the FDS. In adults, it is always close to the MN and it usually ends before reaching the wrist. This is called a. *comitans nervi mediani*, meaning it is an MA of the antebrachial type. In other cases, the artery reaches the hand to contribute to the blood supply to the fingers. This is the palmar type of MA (Jelev and Georgiev, 2011). Rarely, an MA originates at the elbow and courses anteriorly to the antebrachial flexor muscles. This is called a 'superficial' MA. Even though its development is not fully understood, the MA is believed to be a transitory vessel that depicts the arterial axis of the forearm during early embryonic life (Jelev and Georgiev, 2011)

Multiple clinical disorders can result from the presence of a well-developed MA; in turn, it can affect closely related structures such as the MN. In the carpal tunnel, if the MA is the palmar type, it could have an external diameter more than 2.0 mm, causing increased pressure on the MN and therefore potentially causing CTS. Even MA injuries such as thrombosis, aneurysm, traumatic rupture or calcification can cause CTS. According to Jelev and Georgiev (2011), confirming the presence of a sufficient anastomotic blood supply is crucial in such cases as the MN can be injured by ischemia.

Lisanti et al. (1995) presented a case where the patient exhibited no systemic pathological evidence, but had a history of neurolysis of the MN at both wrists. Symptoms resolved on the right side, while those on the left persisted. Surgical exploration revealed a persistent MA. Sometimes, a distal thrombosis of the MA also manifests as MN compression. Salter et al. (2011) presented a case of CTS due to thromboses of the MA detected by



high resolution ultrasound and Doppler. The MA was also associated with a bifurcated MN. This thrombus was resolved by anticoagulant treatment. Akgun et al. (2017) presented a similar case in which there was a bifurcated MN with an MA lying between the divisions. Doppler examination was used to confirm the diagnosis.

### ***Transverse carpal muscle (TCM)***

Tuncali et al. (2005) described three cases of TCM found during routine decompression for CTS. A rare variant of the hand that can cause compression of the MN leading to CTS is a unilateral accessory transverse carpal muscle located palmar to the transverse carpal ligament. The TCM, though an aberrant muscle, is important during surgical exploration of the carpal tunnel as it can be accompanied by a recurrent motor branch and there is an evident risk of iatrogenic injury to it. It is very useful to study and interpret the variables, especially during surgery. Nastis et al. (2020) presented a case of a 38-year-old right-handed male with CTS symptoms in the right hand. The MN was compressed according to a nerve conduction study, with sensory and motor delay. Surgical exploration revealed a transverse muscle overlapping the transverse carpal ligament and flexor retinaculum, which was named the TCM. It was suggested that the TCM could result from aberrant migration of epiblastic cells from the muscle pronator quadratus. It could be accompanied by a recurrent motor branch and could potentially be an iatrogenic injury risk. The MN motor branch is at high risk during operations on the transverse carpal ligament, particularly if there is flexor retinaculum division. If there is a wide TCM, it could also cause the MN motor branch to follow an abnormal course.

### ***Role of Imaging Studies***

For any sort of diagnosis suspected clinically, imaging is the main method of confirmation. This is true for MN entrapment. The usual types of imaging used for such cases are MRI and sonographies. Sometimes, imaging leads to an alternate diagnosis that could have been proposed as a differential (Miller and Reinus, 2010). Diagnostic imaging such as cross-sectional

imaging, typically ultrasound or MRI, reveals the anatomical intricacies of nerves and the changes that can occur during compression (Spratt et al, 2002). Even though MRI should be reserved for cases that are inconclusive on clinical and electrodiagnostic findings, or where the symptoms are unusually severe, it is an extremely useful technique for compressive neuropathies (Bordalo-Rodrigues and Rosenberg, 2004).

Axial views are the most useful images for demonstrating CTS changes in MRI features. Even a hyperintense signal of the nerve is seen on T2-weighted or STIR images. Specificity and sensitivity of MR findings for CTS are low but MRI findings are useful for detecting a space-occupying lesion, inflammatory arthritis or anatomical variations as causes of CTS (Dong et al., 2012).

In pronator syndrome, there is a noted pattern of muscle denervation that indicates edema on fluid-sensitive sequences on MRI, unless there is a mass or hematoma as secondary cause (Dong et al., 2012). Usually, because the perifascial fat is minimal, the MN is depicted poorly at the elbow. In axial images, the MN is visible between the pronator teres and brachialis muscles. It can even appear normal at the site of entrapment.

In some cases of nerve injury, thickening or signal abnormalities have been reported. On MRI, the anatomical basis of pronator syndrome is inconspicuous unless a mass or osseous fracture is close to the nerve. The pronator teres or other muscles innervated by the MN distal to the site of the lesion can give an abnormally high signal. This is on T2-weighted fat-suppressed, STIR, or T1-weighted images (Andreisek et al., 2006).

In Kilon-Nevin syndrome or AINS, patients can show signal intensity changes related to muscle denervation involving the FDP, FPL and pronator quadratus (Dong et al., 2012). The AI nerve is usually visible between the FDS and FDP muscles on MRI. In patients who have this syndrome with an acute or subacute onset, STIR images depict increased signals in the FDP, FPL and pronator quadratus. MR signal intensity corresponding to the fourth and fifth fingers is normal as they are not involved in Kilon-Nevin syndrome (Andreisek et al., 2006).

Most of the anatomical constraints causing this syndrome are not visible on MRI. However, if a focal entrapment or compression of the nerve is observed in MRI, it can alert surgeons to avoid a long incision crossing the cubital fossa. If there is an additional development of T1-weighted MRI signal abnormalities, it could indicate worsening of the syndrome; but if there is normalization of T2-weighted muscle signal intensity, there is recovery of nerve function (Andreisek et al., 2006).

The MRI features of patients with supracondylar process syndrome are not well established but we can draw conclusions from conventional radiographs. Apart from the supracondylar process, MRI can show the Struthers' ligament and its relationship to the MN. This can be useful for detecting occult fractures of the supracondylar process radiographically (Andreisek et al., 2006).

The MN appears ovoid in axial cross-sectional views in the proximal part of the carpal tunnel and has a flatter appearance at the pisiform bone level and distally in the carpal tunnel. The findings in patients with CTS are not always directly linked to the nerve but can involve other contents of the carpal tunnel. The best way to evaluate the syndrome is to note the flattening of the MN, comparing the nerve diameter at the levels of the hook of the hamate and the distal radius. MRI can also show increased signal intensity on T2-weighted fat-suppressed or STIR images. It can show bowing of the flexor retinaculum at the level of the hook of the hamate. If the sensitivity and specificity are low for CTS, it has no role in the clinical assessment of this condition. Nor does it have clinical value if the CTS is caused by a neoplasm, arthritis or anatomical variations (Bordalo-Rodrigues et al., 2004; Dong et al., 2012).

### ***Sonography***

A method being used widely for evaluating structures within the carpal tunnel is musculoskeletal sonography. Bifurcated MNs and persistent MAs have been documented and multiple examples of sonographic appearances of the FDS and lumbrical muscle bellies extending into the carpal tunnel have been provided, but dynamic imaging makes it easier to describe and work with such cases as we can recognize specific mus-

cle bellies. Several potential differential diagnoses can be made such as persistent MA, lipoma, masses and cysts, or even inflammation around the tendons of the carpal tunnel. Along with investigations of the structures in the longitudinal plane, isolated finger movements can be used to identify specific muscles (Takata and Roll, 2019). Moreover, ultrasound-guided minimally invasive release of the cubital tunnel and carpal tunnel has also been performed (Gruber et al., 2021; Loizides et al., 2021).

### ***Theoretical causes for muscle anatomical variations***

Anatomical variations can be divided into anatomical variations of nerves, tendons, vessels and muscles. Exact causes of muscle anatomical variations are not yet known, but according to many authors and researchers, they were formerly attributed to the “ontogeny recapitulates phylogeny” conjecture. Even though this conjecture has been discredited, muscles are known to originate by myocytes migrating into the muscle anlage by the tendon primordium. These anatomical variations are likely to be due to aberrations in embryological signaling (Elliot et al., 1999; Laxminarayan and Michelle, 2017).

## **CONCLUSION**

MN entrapment neuropathies can affect people for a wide range of reasons. There are multiple common causes while others are considered rare. Usually, they are discovered upon surgical intervention or exploration or during imaging studies, but awareness of these anatomical variations that can potentially trap the MN help in achieving better outcomes of recovery and patient care. Surgery has countless adverse effects on the body. The first rule of medicine is “Do no harm”, and if treatment can be made suitable to patients, long-term wellbeing can be promoted.

## **AUTHORS' CONTRIBUTIONS**

Manasi Telang — student/assistant — project development, data collection and management, data analysis and manuscript writing.

Boycho Landzhov (MD, PhD) — professor — data analysis and manuscript editing.

Lukasz Olewnik (D.P.T., PhD) — professor — data analysis and manuscript editing.

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Georgi P. Georgiev (MD, PhD) – assistant professor – data collection, data analysis and manuscript editing.

All authors have read and approved the manuscript.

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