Journal of
Clinical & Medical Surgery

www.jclinmedsurgery.com

ISSN 2833-5465
Open Access
Volume 4

Review Article

Entrapment Neuropathies of the Median Nerve: An Updated Review

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Article Information

Received: Dec 27, 2023
Accepted: Feb 07, 2024
Published: Feb 14, 2024
Archived: www.jclinmedsurgery.com

Introduction

In this revision, we will try to address the entrapment neuropathies of the median nerve, from a clinical and practical approach, so as the family physicians or non-specialist practitioners can know more about these pathologies. Despite Carpal Tunnel Syndrome (CTS) is by far the most common entrapment neuropathy, we should know that not all the pains or paresthesias in the volar side of the hand or forearm are due to CTS. We will deliberately avoid too many anatomic data or specific treatment details, of limited utility in general practice. Nevertheless, in the references the reader can get much more information. If this paper can clarify several questions regarding the median nerve compression, signs and symptoms, the authors will feel more than happy, as this is our main objective.

Entrapment neuropathies: Usually, peripheral nerves are prone to entrapment when the pass-through areas of fibrous or fibro osseous tunnels. Other factors can influence nerve entrapment at different locations, like local or generalized oedema (tenosynovitis, arthritis, hormonal changes.), space occupying lesions (tumors, hematoma…), accumulation of substances (amyloidosis, mucopolysaccharidosi). Entrapment Neuropathies (EN) are heterogenous conditions due to different underlying pathophysiology, thus producing very different signs and symptoms, depending upon the nerve, level of compression and other causes. The pathophysiology of the compression will give us the clue for the diagnosis and treatment. The definition of EN is varied. A good definition could be: "disorders of the peripheral nerves that are characterized by pain and/or loss of function (sensory and/or motor) as a result of chronic compression of the nerves" [1]. The main objective of this paper is to draw a schematic map to distinguish among these three different places of compression of the Median Nerve (MN) so as not to mistake them. This knowledge is the only way for an early diagnosis, that is always based upon a clinical suspicion. This is crucial because precocious nerve injuries may be reversible, while prolonged ones may be not. This is applicable not only for family physicians/general practitioners but also for specialists.

The median nerve: The MN is a mixed, sensory and motor nerve, innervating areas of the forearm and hand. It travels down the front of the upper arm, across the front of the elbow into the forearm. There it innervates the flexor and pronator muscles. It also supplies innervation to the thenar muscles and lateral two lumbricals in the hand. This is regarding its motor function. The MN travels through the mid forearm (as its name, "median"), into the hand through the carpal tunnel, giving sensory function to the palm, more precisely to the thumb, finger, middle and radial side of the ring finger.

Carpal tunnel syndrome (compression of the median nerve at the wrist)

CTS was first described by Sir James Paget in 1854 [2], in a man who developed pain and impaired sensation in the hand, from the trauma of a cord drawn tightly around his wrist. It is by far, the most frequent EN, not only of the MN, but of all the rest of EN taken together. It affects about 2 to 3% of the total population [3], and accounts for more than one half of all the EN. Its pathogenesis is the compression of the MN by the transverse carpal ligament at the wrist. The main symptoms are

Citation: Ly-Liu D, Ly-Pen D, O'Riordan E, José-Luis A. Entrapment Neuropathies of the Median Nerve: An Updated Review. J Clin Med Surgery. 2024; 4(1): 1138.

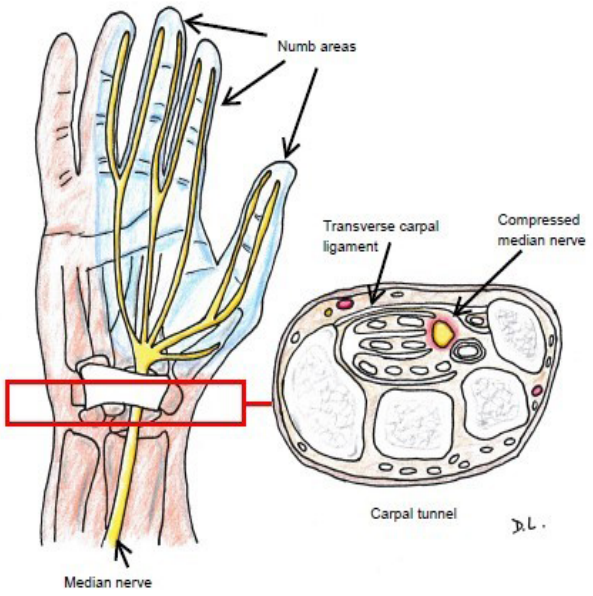

Figure 1: Schematic picture of median nerve compression in the carpal tunnel syndrome. We can also see the area of sensory distribution of the median nerve.

paresthesias and/or pain in the territory of distribution of the MN (in palmar side, thumb, index, middle fingers and the radial side of the ring finger). These symptoms worsen at night and upon awakening (at night, wrist had been in hyperflexion or hyperextension) [4]. Patients try to shake their hands or rub them (Flick´s syndrome) [5]. Nevertheless, the palm sensation is not completely lost, as the palm is innervated by the palmar cutaneous branch, which does not pass through the carpal tunnel. Therefore, if the palmar sensation is lost, the nerve injury is more proximal to the carpal tunnel. On physical examination, percussion on the MN in the carpal tunnel, elicit a sensation of tingling in the distribution of the MN (Tinel´s sign), as we can see in Figure 1. A provocation test that may help in the diagnosis of CTS, consists in increasing the pressure in the carpal tunnel, for example, if the patient hyperflexes the wrist. In the Phalen´s test, the patient holds his wrists in complete and forced flexion (with the dorsal surfaces of both hands together) for up to one minute. If the patient starts feeling the typical CTS symptomatology (paresthesias over MN distribution in the affected hand), then the test is positive (abnormal). Another provocative test over the carpal tunnel is the carpal compression test (Durkan's test). The clinician compresses the MN with his own fingers until his nail bed whitens, for 30 seconds, on the patient´s proximal wrist crease. If the patient feels pain or paresthesia in the MN distribution, then the result is positive. When CTS is long term, patients may have weakness in the hand and thenar eminence atrophy. The diagnosis of CTS can be done with Nerve Conduction Studies (NCS) that may show mainly the functionality of the nerve, if demyelination is present. Image techniques can be also be used, above all Ultrasonography (US), or in rare cases Magnetic Resonance Imaging (MRI). Regarding CTS treatment, the most useful treatments are splints, local injections of corticosteroids and decompressive surgery [6,7]. Splints can be prescribed for every patient, local injections for patients with less severe symptoms, and decompressive surgery when the symptoms are longer or more severe, and definitely when there is a thenar eminence atrophy or severely impaired NCS [8,11].

Pronator teres syndrome (also called pronator syndrome)

The Pronator Teres Syndrome (PTS) was first described by Henrik Seyffarth in 1951 [12]. Behind the CTS, PTS is by far the next more frequent MN entrapment syndrome. PTS is a rare condition, it accounts for <1 per 100,000 annually. Its pathogenesis consists of the MN compression between the humeral and ulnar head of the pronator teres muscle. Usually there is a fibrous band between both heads (Figure 2).

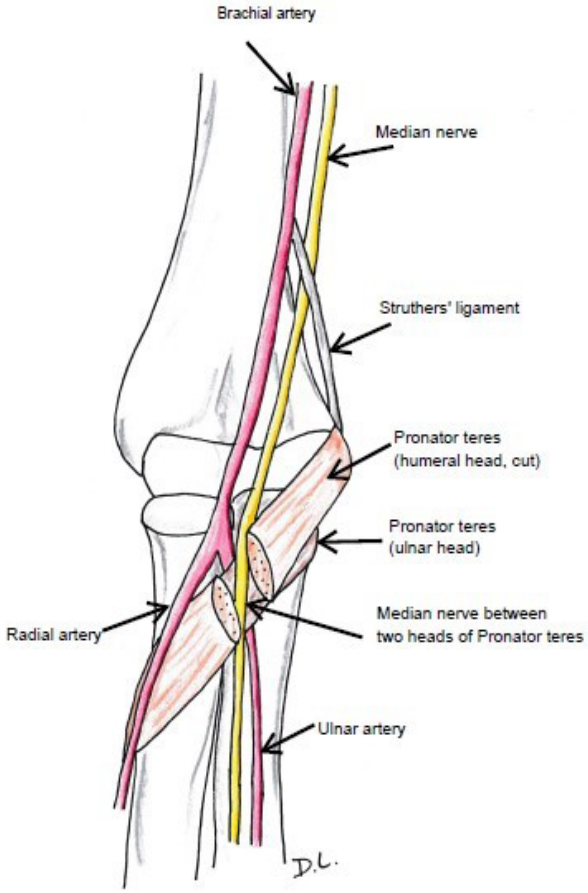

Figure 2: Median nerve entrapment at the pronator teres.

The main symptoms of PTS include paresthesias in the distribution of the MN, similar to CTS, but not preferent nocturnal or when in bed. The pain is more on the proximal volar forearm region, about 5 cm proximal to the carpal tunnel [12]. On physical examination, we can basically practice three maneuvers to evaluate the PTS [13]. The first one is the pronator compression test, probably the most reliable. We apply 30 seconds of pressure proximally and laterally to the proximal edge of the pronator teres muscle belly. If the pain or paresthesia is reproduced, then the test is positive. The second test is the resisted pronation/supination. With the patient´s affected elbow in 90 degrees of flexion, we stabilize with one hand the patient´s elbow, while the other hand grasps the patient´s hand as in handshake position. The patient keeps his forearm in a neutral position, whilst the examiner supinates the patient's forearm, explaining to the patient to actively resist this movement. This will force their pronator muscles (as they try to move into pronation). While holding the resistance against pronation, the examiner extends the patient's elbow. The test is positive if the pain is reproduced. The patient´s elbow should be relaxed, otherwise, if it is kept firmly in flexion, the elbow extension will not be possi-

ble. The last test is the resisted flexion of the proximal interphalangeal joint of the middle finger. This test will reproduce pain and paresthesia when the MN is trapped at the heads of the flexor digitorum superficialis muscle. As in CTS, Tinel sign can be also positive, but it should be in the proximal anterior forearm area, not above the carpal tunnel, as in CTS. Phalen´s test can be positive up to one half of PTS. Nevertheless, muscle atrophy is very uncommon in PTS. All these signs and symptoms may be very confusing because a CTS or an epitrochleitis (golfer´s elbow) can coexist with PTS. The diagnosis of PTS is very challenging, a great dose of clinical suspicious, based upon the clinical history, is needed. Plain x-rays are mandatory, and it should be the first step to diagnosis, to rule out other processes. NCS are also recommended, despite they are not usually diagnostic. They are very useful in excluding coexisting pathology, usually CTS. Image techniques as US and MRI can also be very helpful. They can show direct causes as invasive lesions, cysts, tumors or anatomical variants. The PTS treatment is usually conservative in most of the cases. Local rest, anti-inflammatories and oral corticosteroids can be useful. Splints can be useful as well, to avoid forearm rotation. In refractory cases or in space-occupying lesions, surgical decompression of the area is indicated and it is highly effective. Very likely, due to similar clinical presentation, many authors include in the term PTS, three other anatomical regions where the MN can be compressed in the forearm (besides the pronator teres muscle, sensu stricto): 1) Lacertus fibrosus, 2) Ligament of Struthers, and 3) Fibrous arch of flexor digitorum superficialis [12]. In Table 1: we have them schematically.

Table 1: Most frequent anatomical places of median nerve entrapment in the elbow.

Syndrome	Structure	Location	Extending from
Pronator teres	Muscle	Bellow elbow	The two heads of the Pronator teres muscle
Lacertus fibrosus	Fibrous	Roof over forearm muscles	Biceps tendon
Ligament of Struthers	Ligament	Above elbow	Humerus
Sublimis bridge	Tendinous or muscle	Upper forearm	Inserting in upper forearm, finger flexion
Anterior interosseous nerve	Tendinous or muscle	Upper forearm	The two heads of the Pronator teres, downwards
Carpal tunnel	Fibrous/Ligament	Wrist	Pisiform & hamate bones to scaphoid & trapezium

A compression of the MN at any of the above sites, many authors just refer to them collectively as Pronator Teres Syndrome. 1) MN entrapment by hypertrophied bicipital aponeurosis or lacertus fibrosus, it is known as Lacertus syndrome. Lacertus fibrosus is a ligamentous tissue attaching the biceps to the ulna, over the MN in the proximal forearm (Figure 3). Despite the compression location is not exactly the same in LS than in PTS, the symptoms, physical examination, diagnosis and treatments are very similar [15,16]. The pain in the forearm and paresthesias in the median-innervated digits is exacerbated by resisted flexion of the elbow with the forearm in supination.

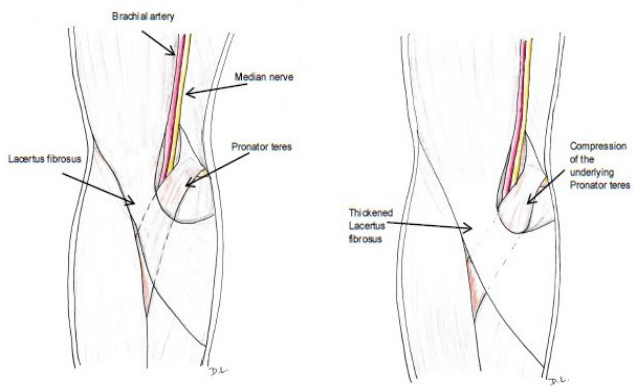

Figure 3: Pathophysiology of the Lacertus syndrome. The aponeurosis from the medial border of the distal biceps brachialis tendon (lacertus fibrosus), compresses the median nerve.

2) MN can also be compressed by the ligament of Struthers, and this is called supracondylar process syndrome, or Struthers syndrome, being extremely rare. This ligament is a fibrous band running from the supracondylar process of the humerus to the junction of the medial humeral epicondyle (Figure 4). The pain in the forearm and paresthesias in the median-innervated digits [17]. The most frequent symptoms are **pain in the forearm** (worsened by extension of the wrist) and paresthesias in the distribution territory of the MN in the hand. On physical exam, the pain in the forearm is exacerbated by supination of the forearm and extension of the elbow (versus pronation in PTS) [17]. Because the brachial artery runs next to the median nerve, occasionally the radial pulse may also be decreased. This syndrome is very difficult to diagnose, and usually, only surgical exploration of the nerve can identify the structure responsible for nerve compression.

3) MN compressed at the fibrous arch of the origin of the flexor digitorum superficialis passing between its humeral and ulnar heads (sublimis bridge or arcade of Fearn and Goodfellow) (Figure 5). The pain in a forearm and paresthesias in the median-innervated digits is exacerbated by resisted flexion of the proximal interphalangeal joint of the middle finger while other fingers are held in extension.

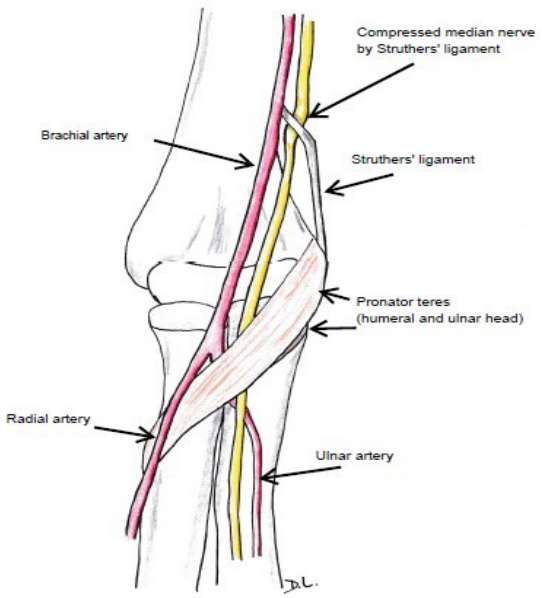

Figure 4: Median nerve entrapment at the ligament of Struthers.

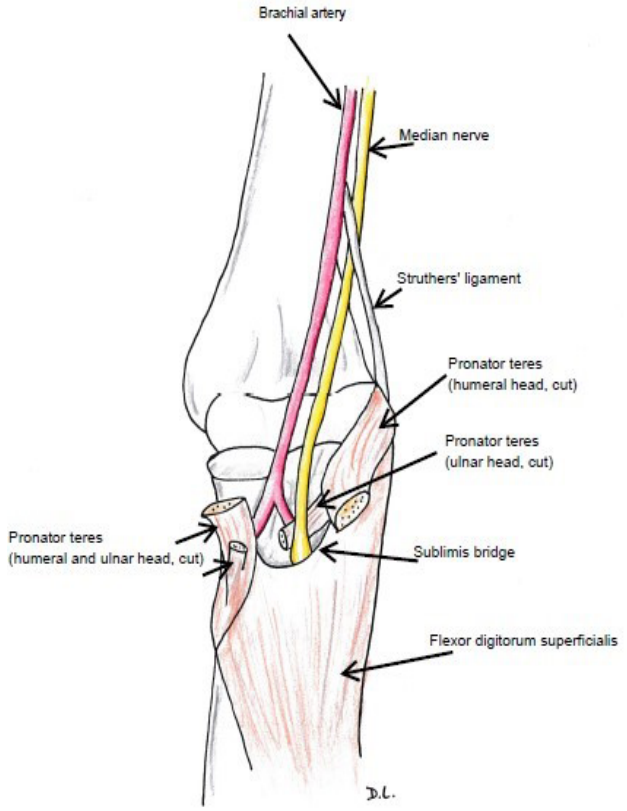

Figure 5: Median nerve entrapment at the "Sublimis bridge", origin of the flexor digitorum superficialis.

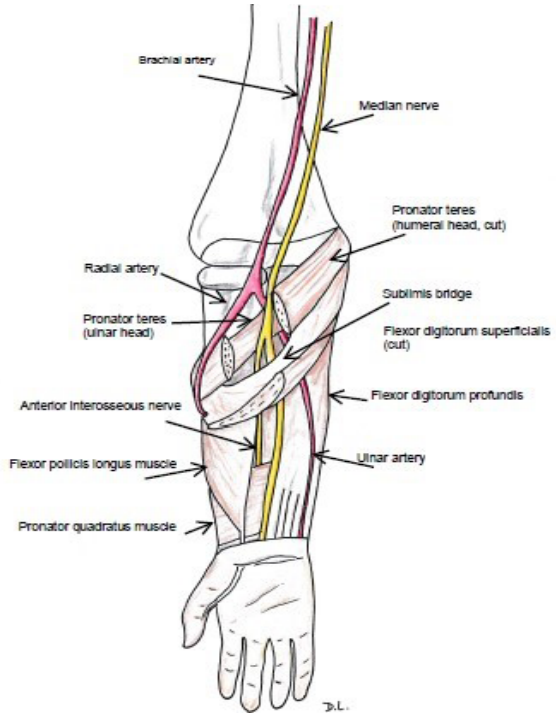

Figure 6: Median nerve entrapment at the flexor digitorum superficialis muscle.

Anterior interosseous nerve syndrome (Kiloh-Nevin Syndrome):

The Anterior Interosseous Nerve Syndrome (AINS) is an extremely rare EN of the MN. It was first described by Parsonage and Turner in 1948 (do not mistake with the Parsonage-Turner syndrome, also known as brachial plexus neuritis or neuralgic amyotrophy). The location of the EN of the AINS are usually the tendinous origin of the flexor digitorum superficialis muscle, at an accessory head of the flexor pollicis longus muscle, and

Table 2: Main characteristics of the most frequent entrapment neuropathies of the median nerve.

	Carpal tunnel syndrome	Pronator teres syndrome	Anterior interosseous nerve syndrome
First description Year	James Paget 1854	Henrik Seyffarth 1951	Parsonage and Turner 1948
Frequency	The most frequent of all. 2.5%	Rare <1/100.000	Very rare
Predisposing factors	Repetitive movements Hypothyroidism Diabetes mellitus	Repetitive movements Hypothyroidism Diabetes mellitus	Traumatic. Compression and brachial plexus neuritis.
Location	Wrist	Elbow	Elbow
Compression by:	Transverse carpal ligament	Humeral and ulnar head of the pronator teres muscle	Usually, it is the tendinous edge of the deep head of the pronator teres muscle.
Symptoms	Paresthesias, pain in wrist and palm	Tenderness over the pronator teres muscle. Pain with resisted pronation of the forearm. Weakness with abduction of the thumb	Only motor deficiency: isolated paralysis of the AIN-supplied muscles: weakness of the index and thumb finger pincer movement.
Compression eliciting (or worsening) numbness	Above the carpal tunnel (Durkan´s test)	Over the pronator region	Negative
Physical examination	Tinel, Phalen, Durkan Thenar eminence atrophy	Pronator compression test. Resisted pronation/supination. Resisted flexion of the proximal interphalangeal joint of the middle finger.	Pinch grip test + Unable to do "OK" sign Difficult fist inability to button their shirts
Diagnosis	Nerve conduction studies Echography, MRI	Nerve conduction studies. Echography, MRI	Nerve conduction studies. MRI
Treatment	Wrist splint Local corticosteroid injection Surgery	Splints Physiotherapy Surgery	Splints Physiotherapy Surgery

at the tendinous origin of the deep head of the pronator teres muscle (Figure 6) [18].

The AIN is the last major branch of the MN, leaving about 5 to 8 cm distal to the lateral epicondyle. AINS is an exclusively motor EN of the MN, affecting three muscles in the forearm (the flexor pollicis longus, flexor digitorum profundus and pronator quadratus).

The main symptoms are motor weakness, with difficulties to pinch the thumb and index finger together. On a physical examination these patients fail to make an "OK" sign with the hand, because of the weakness (palsy) of the flexor pollicis longus muscle and flexor digitorum profundus muscle to the index finger, with a positive Pinch Grip test (Froment's sign). Instead the "OK" sign, the patient will only be able to clap the sheet between the index finger and an extended thumb [19].

When the patient is requested to make a fist, the flexion of index and middle finger will be impaired, showing hand of benediction. Do not mistake this sign with the claw hand from ulnar neuropathy [19].

Clinically, there is no sensory loss in AINS, as it carries only deep sensory fibers to the wrist.

NCS will show no changes in sensory parameters, as there is no sensory innervation in this nerve. Nonetheless, they will show findings in the flexor pollicis longus, the radial portion of the flexor digitorum profundus, and the pronator quadratus. Nevertheless, in a thorough study, Pham et al [20] could not demonstrate these findings. Furthermore, they saw that at least their NCS showed non-localizing nerve damage. They could not discriminate between injury to the AIN itself and a more proximal lesion. They even suggested that because of the predominance of lesions at upper arm level in their patients, it could be supposed that AINS is not an entrapment neuropathy.

MRI can be also very helpful.

Regarding treatment of AINS, oral anti-inflammatories, short term corticosteroids and physiotherapy should be indicated in first term. If no better, then surgical treatment is of choice.

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