*Review Articles*

# Entrapment Neuropathies

*Philip-Ephraim[1] E E & Agan TU[2]*

**Abstract**

**Background**: Entrapment neuropathies are common medical disorders frequently encountered in all fields of medicine. They are focal peripheral nerve injuries due to mechanical compression of a nerve through a fibro-osseous tunnel. Magnetic resonance imaging techniques provide useful information in dealing with these conditions.

**Objectives**: This was a review of current information on entrapment neuropathies.

**Methodology**: A literature search of current information on entrapment neuropathies was done. Articles on print and electronic publications were read and current information extracted.

**Results:** The causes were unknown in about 50% of cases. Nerve injuries were neuropraxia, axonotmesis or neurotmesis. Entrapment neuropathies usually existed close to joints where reduction in space and nerve pressure compromised neural blood supply resulting in anoxia. This made repetitive joint movement a major risk factor. Carpal tunnel syndrome (CTS) was commoner in women while cubital tunnel syndrome was commoner in men. The median nerve could be compressed in the carpal tunnel and lacertus fibrosus, ulnar nerve in the cubital tunnel, and the common peroneal at the fibular neck. Fibrous bands and arches compress the posterior interosseous nerve, deep motor branch of the radial nerve at the arcade of Frohse. Myofascial bands compress the sciatic nerve between the adductor magnus and biceps femoris. Nerve entrapment might follow soft tissue swellings in rheumatoid arthritis, or ganglion. Pregnancy, hypothyroidism, amyloidosis, diabetes, rheumatoid disease, myeloma and trauma to nerve increase risk of carpal tunnel syndrome. The idiopathic CTS were associated with females 40 -60 years old, hypercholesterolemia, stroke, and anticoagulation therapy. Pain, numbness, tingling sensation and loss of function were common complaints.

Electrodiagnostic testing involving electromyography, nerve conduction studies, computed tomography and magnetic resonance imaging were used for diagnosis. Standard magnetic resonance pulse sequence helped in visualizing various anatomical features of peripheral nerves and the nearby tissues. Management included both medical and surgical treatments. Education on vocation especially sports offered non-drug therapy. Use of non-steroidal anti-inflammatory drugs and steroidal drugs gave variable results. Local injections with lidocaine were also successful. Refractory cases had surgical decompression.

**Conclusion**: Entrapment neuropathies were common conditions prevented by early identification and treatment.

***Keywords***: *Nerve entrapment, neuropathies*

[1] *Department of Internal Medicine, College of Medical Sciences, University of Calabar, Nigeria.* [2]*Department of Medicine & Obstetrics/ Gynaecology, College of Medical Sciences, University of Calabar, Nigeria.*

**Correspondence:** *Dr. Ekanem E. Philip-Ephraim, Department of Internal Medicine, College of Medical Sciences, University of Calabar, Nigeria. E-mail:nneninge@yahoo.com*

## Introduction

Entrapment neuropathies are a common medical disorders encountered in all fields of medicine irrespective of age, sex or nationality. They may be acute or chronic. They create a state of misery for even the obstetric client. The majority of the patients present to the physician with diverse symptoms.

Entrapment neuropathy is a term that applies to focal peripheral nerve injuries due to mechanical compression of a nerve through a fibro-osseous tunnel. Fibrous bands have also been observed to constrict a peripheral nerve in a closed space leading to entrapment of such a nerve[1,2]. Entrapment neuropathy runs a gradual continuing chronic course with a typical pathological process [3]. There are many risk and predisposing factors which have been identified although in some the cause is unknown. Some entrapment neuropathies are commoner in some gender than others. For example, carpal tunnel syndrome is commoner in females 4while cubital tunnel syndrome is commoner in males 5. Entrapment neuropathy of the upper extremities is commoner than the lower extremities. The clinical features are often very classical and follow the distribution of the affected nerve. In some, the diagnosis of such entrapment has been very challenging to some inexperienced doctors leading to misdiagnosis, mismanagement, waste of time and resources before referral. Investigations include electrodiagnostic testing and imaging studies. Treatment is usually conservative and surgical decompression in refractory cases.

The knowledge of the anatomy of the peripheral nerves is paramount in making a correct diagnosis. Studies identifying the various anatomical sites of chronic compression have been carried out to aid the surgeons[6].

## Types of nerve injuries:

Nerve injuries are classified into three main types namely, neurotmesis, axonotmesis and neuropraxia. In neuropraxia, conduction block occurs only at the site of injury with normal conduction at the other sites of the nerve. The integrity of the nerve is still maintained with no injury to the axon. Areas of segmental demyelination have been found histologically. Compression is the usual cause of this type of injury and patients recover full function in days to weeks. Axonotmesis involves disruption of the axons with Wallerian degeneration occurring distally to the injury site. The endoneurium and perineurium are unaffected and therefore promote nerve regeneration. Schwann cells covering the injured axons are also not affected. In neurotmesis there is total transection of the peripheral nerves. This type of injury is seen in open traumatic wounds. Recovery here has a mixed picture[7]. Integrity of the axon is maintained till late but ultimately the number and function of the axons reduce. Chronic nerve compression usually leads to Schwann cell apoptosis and changes in myelination. There is multiplication of Schwann cells of the affected nerve segments culminating in striking increase in the number of Schwann cells. These signify that chronic nerve compression generate a sustained cycle of Schwann cell production and demyelination with subsequent remyelination which may be a consequence of an immediate reaction to mechanical stimuli [8].

Histologic changes do occur at sites of compression of a nerve. Only few cases have been reported since permanent nerve damage can result from nerve biopsy in humans. This has led to paucity of histologic studies in humans. The changes that may occur histologically in a resected nerve segment include thickening of the walls of the micro vessels in the endoneurium and perineurium , oedema in the epineurium and perineurium, fibrosis and thickening at area of

injury, myelin thinning with nerve fibre degeneration and regeneration[9].

**Pattern of entrapment**

The peripheral nerves of the upper extremities originate from the brachial plexus and those of the lower extremities from the lumbosacral plexus. Some of the nerves involved in upper extremity entrapment are the suprascapular nerve, the axillary nerve, musculocutaneous nerve, radial nerve, median nerve, ulnar nerve and those involved in the lower extremity entrapment neuropathies include the iliohypogastric, ilioinguinal nerves, genitofemoral nerves, lateral cutaneous nerve of the thigh, femoral nerve, obturator nerve, sciatic nerve, tibial nerve, the peroneal nerves.

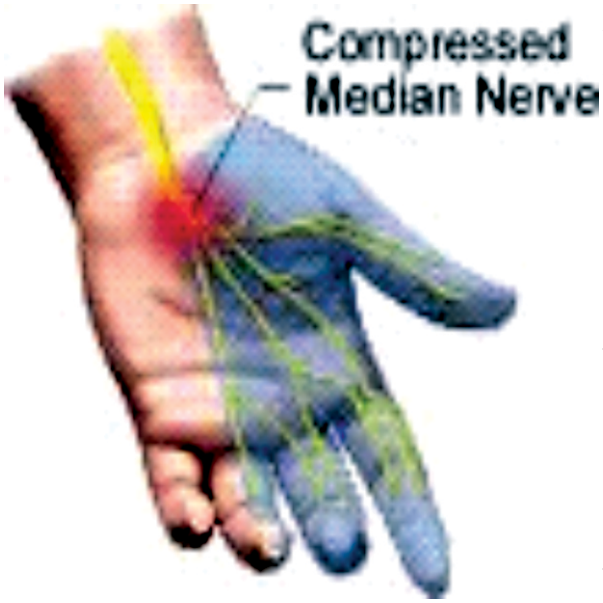
Entrapment neuropathies usually exist close to joints as the nerve traverses a fibro osseous tunnel making its way from one body segment to the other. Examples include the carpal tunnel syndrome (CTS) and cubital tunnel syndrome (CUTS) affecting the median and ulnar nerves respectively; the tarsal tunnel where the posterior tibial nerve gets entrapped10 and at the fibular neck as the common peroneal nerve spins round it to pass through the fibular canal [11,12]. Fibrous bands and arches have also compressed nerves leading to entrapment neuropathies. The posterior interosseous nerve, the deep motor branch of the radial nerve, has been observed to be vulnerable to entrapment at the arcade of Frohse [13, 14]. The sciatic nerve has been found to be compressed by myofascial band between the adductor magnus and the biceps femoris [15] while the superficial branch of the radial nerve was compressed at its exit by fascial bands[16]. Entrapment can also result from soft tissue swelling e.g. rheumatoid arthritis, or by anomalies in muscles e.g. pronator teres syndrome, deformities of bone or by a mass e.g. ganglion.

**Carpal tunnel syndrome**

This is the commonest entrapment neuropathies of the upper extremities[17,18] and the most frequently seen by hand surgeons[19]. It is due to entrapment of the median nerve in the carpal tunnel at the wrist. The tunnel contains the median nerve and nine tendons passing to the palm from forearm. After traversing the tunnel, the median nerve divides into both the sensory and motor branches. Compression of the median nerve at the tunnel leads to carpal tunnel syndrome (CTS) [19, 20]. It is commoner in females than males4. Predisposing factors include pregnancy, hypothyroidism, amyloidosis, diabetes, rheumatoid disease, myeloma, trauma causing raised pressure on the nerve. Nevertheless, the aetiology is unknown in about 50% of cases and this idiopathic group exceeds the other causes[19,] occurring in females between the age of 40-60 years where symptoms tend to be bilateral21. One of the risk factors for idiopathic CTS was postulated to be hypercholesterolemia. Carpal tunnel syndrome was also reported in stroke patients and this was attributed to excessive use of the non- paretic hand [22,23]. Though unusual, CTS has resulted from anti-coagulation therapy[24]. Patients experience numbness, burning and tingling sensations over the lateral three and a half fingers and wrist. Symptoms worsen at night and are aggravated by frequent wrist flexion and extension. Phalen, Flick and Tinel's tests are usually positive. Atrophy of thenar eminence is present in only serious and chronic cases [18].

The median nerve can also be entrapped between the two heads of the pronator teres muscle at the lacertus fibrosus. The anterior interosseous nerve, the pure motor branch of the median nerve could be entrapped deep to the pronator teres heads by fibrous bands. Pain and numbness are experienced at the volar surface of the forearm and elbow. Sensation is reduced in the thumb, second and third and half of the ring fingers[25]. Patients find it difficult pinching thumb

***Figure 1***: Carpal tunnel showing compressed median nerve




and index finger[26].

**Other neuropathies**

The posterior interosseous nerve, a pure motor branch of the radial nerve is commonly entrapped at the arcade of Frohse causing difficulty in finger extension and thumb at the metacarpophalangeal joint (MCP) joint[11]. The sensory branch of the radial nerve can be compressed by brachioradialis and extensor carpi radialis tendons leading to pain, numbness and tingling sensation on the dorsolateral part of the hand[16]. The ulnar nerve is frequently entrapped at the elbow when passing through the cubital tunnel producing pain on the medial part of the elbow or forearm and numbness or tingling sensation on the little and ring fingers. The intrinsic muscles of the hand may be weak or even atrophy in severe cases[27]. Entrapment of ulnar nerve at the wrist as it traverses the Guyon's canal is not common. Symptoms depend on which branch is affected so patients can present with sensory, sensorimotor or purely motor symptoms[28]. The femoral nerve can be entrapped by the iliopectineal arch producing weakness of knee extension, loss of knee jerk and loss of sensation on the inner calf area[29]. The posterior tibial nerve is usually entrapped at the tarsal tunnel behind the flexor retinaculum. Examination reveals weakness of plantar flexion and foot inversion with reduced sensation over sole of foot[11]. The sciatic nerve entrapment at the sciatic notch, the piriformis syndrome, is well known. The sciatic nerve can also be bound down by myofascial bands. Patients present with weakness of knee flexion. Muscles of the leg and foot are also weak with reduced sensation in the foot and the lateral part of the leg[30]. The peroneal nerve can be entrapped at three sites which are: at the level of the fibular head, the anterior tarsal tunnel and at the ankle. Symptoms include weakness of both foot evertors and dorsiflexion.The lateral femoral cutaneous nerve, a purely sensory nerve, is usually entrapped at the inguinal ligament. Lesions result in numbness, burning and tingling sensation on the anterolateral part of the thigh[11].

**Mechanisms of entrapment**

Since carpal tunnel syndrome is the prototype of entrapment neuropathy and indeed the commonest, followed by cubital tunnel syndrome, the mechanisms of entrapment would be discussed in relation to them.

There are various mechanisms underlying entrapment neuropathies. Some of the mechanisms include compression by pressure, 'stretch', friction and angulations31. But the basic event is usually a reduction in the space needed by a peripheral nerve to function normally. This reduction leads to increase in tissue pressure which jeopardizes the blood supply to a nerve segment. The ensuing tissue anoxia leads to oedema formation which further compresses the peripheral nerve. The changes that occur pathologically can be easily reversible if the compression is mild and short –term. Intense and chronic compression result in alteration in axonal

transport and malfunction of the cell body. The duration of such chronic compression on a nerve has differing effects including increase in fibroblast formation and nerve scarring. [19,27]. In a study carried out on a Sprague Dawley rat, where the sciatic nerve of the adult male rat was banded with a silastic tube for differing periods of time, no histologic abnormality was demonstrated at three months. At five months, abnormalities of the nerve noted were perineural thickening and segmental demyelination at the periphery of the fascicles but with no abnormality found in the central fibres. At eight months, there were additional epineurial and perineurial thickening with considerable thinning of the myelin and Wallerian degeneration[32].

Movements at joints, such as flexion and extension, have been implicated as one of the mechanisms. The carpal tunnel volume is not static but changes with wrist position. It is inversely related to the interstitial pressure when the wrist is at neutral position[17]. Repetitive exercises and changes in position of wrist cause periodic increase in the interstitial pressure and intermittent chronic compression in predisposed patients[19,20]. Epidemiologic studies have linked CTS with abnormal wrist postures and repetitive movements. The angle of the metacarpophalangeal (MCP) joint has been observed to have considerable effect during various motion tasks involving wrist flexion-extension and radioulnar manouvres. Finger posture was postulated to increase the carpal tunnel pressure. Pressures were markedly increased with straight finger than during flexion of the MCP joint to 450. The difference was highest when the wrist was extended33. Entrapment of the posterior interosseous nerve was observed in those engaged in activities requiring repetitive pronation and supination such as is found in athletes, orchestra conductors, and tennis players [34, 35]. Cubital tunnel syndrome has been reported in athletes especially in baseball throwers where the vulnerability is more, due to extreme flexion at elbow during such events[36]. In cubital tunnel syndrome, the distance between the medial epicondyle and the olecranon widens by 5mm for every 450 flexion of the elbow. Flexion of the elbow puts tension on the medial collateral ligament and the retinaculum. This causes the cubital canal to lose height of about 2.5 mm thereby distorting its shape from a circular to an oval tunnel. This loss in height causes a 55% reduction within the canal leading to increased intraneural pressure from 7mmHg to 14mmHg. Marked increase in cubital tunnel pressure (CUTP) with a six times rise in ulnar intraneural pressure results from the additive effect of shoulder abduction, flexion of the elbow and extension of the wrist and this makes the nerve susceptible to entrapment [37]. Flexion of the elbow leads to excursion and traction of the nerve due to the location of the ulnar nerve behind the axis of rotation of the elbow, Valgus deformity can lead to increased traction forces on the ulnar nerve and this predisposes to compression[38]. Inflammation of the nerve results from the friction produced with frequent subluxation thereby making the nerve vulnerable to an accidental trauma predisposing to entrapment[37].

Flexion of the elbow with raised CUTP was reported in 10 patients with confirmed diagnosis of CUT syndrome. In that study, the cubital tunnel pressure was noted to be 9mmHg with elbow extension but with elbow flexion the pressure rose to 63mmHg. The pressures during extension increased further with contraction of the flexor carpi ulnaris to 92mmHg and with flexion of the elbow to 209mmHg. All the pressures were measured intra-operatively[20].

The carpal tunnel pressure (CTP) has been found to be a crucial factor in the mechanism of CTS. An association between CTP and nerve conduction was established by Luchetti *et al* where he found nerve conduction velocity action

potential amplitude to be diminished at the distal part of the tunnel where the pressure was markedly increased[39].

Narrowing of the tunnel has been attributed to certain congenital anomalies. Incursion of lumbricals into the carpal tunnel during flexion is considered a normal phenomenon [40], but the space within the tunnel can be compromised if the lumbricals are longer or hypertrophied. Anomalous tendinous flaps from long flexors and muscle bellies have been found in the tunnel[41].

Hypercholesterolemia has been known to cause entrapment neuropathy. Fibrogenesis causes proliferation of the connective tissue of the median nerve in the carpal tunnel and this has been linked with increased levels of low density lipoprotein in the serum which influences nerve enlargement with eventual increase both in volume and pressure of the tunnel contents[42].

Vibration has been found to cause peripheral neuropathy. Patients using vibrating tools have been found to be vulnerable to entrapment neuropathy. In animal studies, such vibration has caused intraneural oedema and in humans, demyelination of the nerve, axonal loss and fibrosis have been demonstrated. These changes are prone to occur in the median nerve in the carpal tunnel of such exposed workers[9].

Systemic diseases like chronic renal failure and diabetes mellitus have been found to cause entrapment neuropathies. Undoubtedly, carpal tunnel syndrome is more common in diabetics than in normal healthy individuals. The mechanism whereby diabetes mellitus causes CTS has been attributed to frequent unnoticed trauma, metabolic changes and oedema formation in the narrow space of the carpal tunnel. Different forms of entrapment neuropathies have also been observed in patients on chronic dialysis. Amyloidosis related to the dialysis is postulated to be the possible aetiology. There are changes in phosphate and calcium metabolisms leading to soft tissue calcification with resultant peripheral neuropathies. Factors contributing to the amyloidosis include the age of the patient, dialysis membrane biocompatibility, dialysate fluid, the length and type of dialysis. Thus long-term dialysis has led to carpal tunnel, cubital tunnel and Guyon's tunnel syndromes and rarely tarsal tunnel syndrome [43].

**Clinical features**

Entrapment neuropathies cause focal nerve dysfunctions leading to sensory loss, motor affectation or sensorimotor abnormalities in the distribution of the areas innervated by these nerves depending on whether the nerve affected is a pure motor, pure sensory or mixed nerve . The clinical features therefore include tingling sensations, numbness, burning pain, and atrophy of the affected muscles, difficulties in performing movements such as extension and flexion and positive Tinels sign. [11,17,27,44].

**Investigations**

These include electrodiagnostic testing involving electromyography, nerve conduction studies and somatosensory evoked potentials recording. Other investigations found useful include imaging techniques like computed tomography (CT) scan and magnetic resonance imaging (MRI) in diagnosing avulsions of spinal nerve roots, tumours and cysts. Standard Magnetic Resonance Pulse sequences have also been employed in visualizing various anatomical features of peripheral nerves and the nearby tissues[31,45].

**Management**

The management of patients includes both medical and surgical treatments. The medical treatment has two approaches, the non-pharmacological and the pharmacological therapies. The non-pharmacological therapy

involves the education of the patient with adjustment of vocational and recreational functions with reference to minimizing or preventing repetitive motions. Splinting of the affected site has also been employed. Treatment with non-steroidal anti-inflammatory drugs (NSAIDS) and steroids has given conflicting results. Whereas both drugs were found to be of no beneficial effect in the management of CUTS27, the use of steroids in combination with lidocaine injected locally were found to be quite successful in the treatment of entrapment neuropathy of the lateral femoral cutaneous nerve of the thigh[46]. Some patients have also been observed to recover spontaneously without any intervention. Surgical decompression by various methods has been used in the management of entrapment neuropathies in refractory cases where medical treatment has failed to reverse the process. This method was employed by Solheim *et al*[47] in reversing the compression of the sciatic nerve that was bound down by adhesions.

**Conclusion**

Entrapment neuropathies are common. Carpal tunnel and cubital tunnel syndromes are well recognized and are commoner than other entrapment neuropathies which are uncommon. A high index of suspicion in predisposed patients is needed. The knowledge of the anatomy and functions of the peripheral nerves with the possible sites of entrapment are invaluable in making the correct diagnosis. Early identification of the problem can prevent permanent nerve damage resulting in favourable outcomes.

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