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Abstract
The effects of peripheral nerve compression have only been appreciated for a century. Panas published the first description of tardy ulnar palsy in 1878(17), exactly a hundred years ago, although Morton had preceded him by two years in his classical paper(14) about the neuroma which may form in the forefoot after presumed pressure, repeated trauma or degeneration. Since that time many other peripheral entrapment neuropathies have been encountered and discussed (2) (10) and the importance of early and effective treatment appreciated.

This paper will present a synopsis of the pathology and sites of such entrapment, excluding the lesions of nerve that may occur from proximal pressure secondary to vertebral disc, spinal or thoracic outlet pathology. The effects of ulnar nerve compression at the elbow will be discussed in some detail using data obtained from a recent retrospective review of 100 patients with this complaint.

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PERIPHERAL ENTRAPMENT NEUROPATHIES

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The effects of peripheral nerve compression have only been appreciated for a century. Panas published the first description of tardy ulnar palsy in 1878[17], exactly a hundred years ago, although Morton had preceded him by two years in his classical paper[14] about the neuroma which may form in the forefoot after presumed pressure, repeated trauma or degeneration. Since that time many other peripheral entrapment neuropathies have been encountered and discussed[2] [10] and the importance of early and effective treatment appreciated.

This paper will present a synopsis of the pathology and sites of such entrapment, excluding the lesions of nerve that may occur from proximal pressure secondary to vertebral disc, spinal or thoracic outlet pathology. The effects of ulnar nerve compression at the elbow will be discussed in some detail using data obtained from a recent retrospective review of 100 patients with this complaint.

Pathology

Mild but chronic nerve compression has been shown experimentally to cause partial obstruction of the vasa nervorum. This results in hyperaemia and oedema of the nerve trunk and gradual narrowing of the axons due to increased interstitial fluid pressure. The axons themselves may also increase in size, but this is again due to distention by oedema fluid. The axonal substance is displaced by this process[4] and the nerve swells both proximal and distal to the site of compression [6] [25]. Eventually the oedema fluid is invaded by fibroblasts and an irreversible fibrosis occurs which causes permanent loss of nerve conduction[24].

Although compression is probably the most common primary cause of these changes, traction at sites such as the ulnar nerve in the post-condylar groove will also affect conduction. Paine [16] quotes 19 possible mechanisms of chronic nerve injury at the elbow although most derive from nerve compression of one kind or another. The actual pressure to which the ulnar nerve is subjected was studied by Pechan and Julis[18]. Using fresh autopsy material they were able to show that the normal pressure within the "cubital tunnel" was 7mm of mercury when the arm is extended. This cubital tunnel is formed by the aponeurotic arch of the flexor carpi ulnaris muscle origin overlying the ulnar nerve just distal to the elbow joint and bridging the medial aspect of the proximal ulna to the common flexor origin. When the elbow is flexed to a right angle pressure around the nerve increases to approximately 20 mm of mercury, and increases further if the wrist is extended and the shoulder abducted. Pressures well over 30 mm of mercury have been recorded, similar to those observed in the carpal tunnel by Brain[1] and Tanzer [19]. The pressure required to impede flow in the vasa nervorum is not known, although in other tissues the capillary pressure is approximately 20 mm of mercury (Fronek[9]).

Diagnostic Features

The onset of symptoms secondary to compression of a peripheral nerve may be either gradual, intermittent or acute. Much depends on the underlying pathology and the site of the lesion. Pain is often a feature, although it is more accurate to describe this as paraesthesiae since pain in the hand is not, for instance, a constant feature of ulnar neuritis. Symptoms are present during both night and day, although they may be exacerbated by occupation or certain positions in which the limb may be held; thus the symptoms of ulnar neuritis may be worsened

by lying with the arm acutely flexed at the elbow, or the symptoms of meralgia paraesthetica (from compression of the lateral cutaneous nerve of thigh) may be worsened by adducting the thigh, for example when crossing the legs. It is said that symptoms from the carpal tunnel syndrome are more acute and continuous since the compression of the nerve is not intermittent, unlike ulnar neuritis where symptoms may be relieved by extending the arm. After symptoms have been present for some time there is usually blunting of sensation in the distribution of the nerve, although hyperaesthesia may be noted in the early stages. In mixed nerves, motor function may be preserved for some time, with no apparent loss of power. However muscle wasting often presents quite early and may be associated with a feeling of clumsiness in the hand or of subjective weakness in the leg.

Local tenderness at the site of compression may be acute, and it is often possible to exacerbate the distal symptoms by compressing the tissues at this site. A positive Tinel sign may also be elicited, indicating definite nerve injury. Infiltration with local anaesthetic at the site of tenderness may produce temporary relief and help towards the diagnosis. Steroid infiltration is also helpful and may give a permanent relief of symptoms in a proportion of cases, although it is never entirely successful. There are often associated syndromes affecting the limb, such as de Quervain's tenovaginitis and lateral humeral epicondylitis ('tennis elbow"). This led to the suggestion of a "summation theory" which assumes that the nerve is subjected to mild and subclinical compression at a number of sites, such as the vertebral canal, in relation to areas of external pressure (the radial nerve in the spiral groove) and at the various osseofibrous tunnels through which the nerve must pass. Although individually the nerve compressions are insufficient to produce symptoms, the net effect is cumulative and symptomatic. Thus cervical spondylosis may co-exist with more peripheral compression lesions, and the resultant symptomatology may be confusing. Furthermore, release of an apparently convincing compression at a site such as the elbow or the wrist may not relieve symptoms entirely since the root compression proximally remains.

Sites of Compression

1. Upper Limb

(a) *Ulnar Nerve.* The nerve is rarely compressed proximal to the elbow, although it is occasionally trapped in the elbow joint after a dislocation at that level. Entering the forearm by passing round the medial epicondylar groove, the nerve is normally free to glide at this level, thus preventing any stretching during flexion of the arm and abduction of the shoulder. If the mechanics of the elbow are distorted, particularly in cases of cubitus valgus resulting from a supracondylar humeral fracture, then the nerve will be liable to injury. This is particularly the case if adhesions form round the nerve trunk, binding it to the post condylar groove. Symptoms may present many years after the fracture, and this syndrome constitutes tardy ulnar palsy. Osteoarthritis of the elbow joint may also affect the gliding mechanism of the nerve, and osteophytes from a degenerating joint may compress the nerve just distal to the post condylar groove.

In 1957 Osborne described constriction of the nerve at the level of the flexor carpi ulnaris aponeurosis[15]. He noted a band of tissue stretching across the nerve in 13 cases, and observed that it was relatively fixed at the medial epicondyle but mobile at its olecranon attachment. Simple division of this band gave results which he considered to be comparable to those after the more formal operation of transposition of the nerve. A year later Feindel and Stratford described much the same lesion [7], but stressed that a second constricting band was often present more distal to the flexor carpi ulnaris aponeurosis. This "Osborne lesion" or cubital tunnel compression syndrome accounted for approximately half the cases of ulnar neuritis followed up in a recent review by the author (Table 1). Other pathology which may produce compression includes constriction by the anconeus epitrochlearis muscle or by triceps hypertrophy, adhesions in the post condylar groove, a ganglion or a bony prominence such as a spicule or a loose body. The nerve may also be subjected to pressure if it dislocates anteriorly out of the post condylar groove. Childress[5] found that the ulnar nerve could be palpated in this dislocated position in 16 per cent of the normal population, and that this was often a bilateral feature. The nerve may be at particular risk in these instances when there has been prolonged recumbency such as Woltman described in the chronically bedridden[22], or as may occur post-operatively when the patient uses the point of the elbow to move about in bed. In spite of certain of these clear cut causes of ulnar nerve compression, no cause is apparent in one quarter of the cases of ulnar neuritis that present

clinically.

TABLE 1

	No. of cases	Percent.
' Osborne lesion"	50	46
Negative	25	23
"Adhesions"*	20	18
Anconeus Epitrochlearis	10	9
Triceps hypertrophy	9	8
Dislocated nerve	8	7
Ganglion	4	4
"Stretched"*	2	2
Bony spicule	1	1
Loose body	1	1
Total =	130	

Operative Findings in 110 Explorations

(*With or without "narrowing" or "thickening" of nerve)

Treatment of nerve compression at the elbow is best managed by anterior transposition deep to the flexor muscle mass[13] although a number of surgeons still consider that a more superficial transplantation is sufficient. Simple decompression of the cubital tunnel will only suffice if the lesion is relatively acute and where there is clear cut evidence of compression of the nerve at the aponeurotic arch with a proximal neuroma but no adhesions in the post condylar groove. The longer the symptoms have been present the less likely is complete recovery of the nerve. Fortunately the majority of patients obtain reasonable symptomatic relief, with cessation of paraesthesiae in the hand and usually some gain in power of the fingers. A poor end result occurs in those cases where no pathology is evident, or where adhesions in the groove are present, and where only a release of the aponeurotic band is carried out. The nerve in these instances may still be trapped by some other pathology, and will not glide around the condylar groove on account of the adhesions, stretching presumably continues postoperatively and the symptoms are only slightly relieved, if at all. Anterior transposition is not free of unsatisfactory results either, but a direct comparison between this technique and simple release is difficult since the underlying pathology is often different, and the more chronic the lesion, the more likely that transposition is chosen. If the results of either transposition or release are reviewed at least one year post-operatively it is found that poor recovery occurs if the symptoms have been present for more than one year, and that the best results are in those patients whose elbows are explored within three months of the onset of ulnar neuritis.

The ulnar nerve may also be compressed at the wrist [21] in Guyon's canal and also at its deep branch in relation to the hook of the hamate. The latter lesion may occur after repeated compression of the nerve against the carpus, such as in cyclists or gymnasts.

(b) *Median Nerve.* The most common peripheral entrapment neuropathy of all involves the median nerve at the wrist. The nerve travels beneath the carpal ligament in the so-called carpal tunnel and is subject to compression. The size of the tunnel may be reduced following fractures of the carpal bones or a Colles fracture, in advanced hypertrophic osteoarthritis of the wrist, or if the flexor retinaculum is thickened. The volume of the other contents of the canal may increase, and this has been described with ganglia [3], fatty infiltration[23], tendon bursitis, acromegaly, myxoedema and various other infiltrative conditions. Symptoms brought on by pregnancy or the menopause may also be due to water retention and increase in the size of the contents of the canal. In many cases, however, the onset of the carpal tunnel syndrome cannot be attributed to any obvious cause. It is most common in middleaged women, and like all compression syndromes may be bilateral. Some cases of carpal tunnel syndrome subside, while others may be minimally symptomatic for over 20 years. Diurnal symptomatology is common and symptoms may be exacerbated by dorsiflexing the wrists.

Release of the carpal ligament is almost always accompanied by complete relief of symptoms, and usually by recovery of any thenar motor loss which may have been present. Simple splinting of the wrist in a plaster for a few weeks may relieve symptoms in a proportion of cases, and injections of lignocaine and hydrocortisone are also advocated, but may only provide temporary relief.

The median nerve may be compressed proximally by fractures of both the humerus and the radius, and occasionally radiological evidence of such entrapment is present. The nerve may also be compressed by the ligament of Struthers which is present when a supracondylar bony spur is evident radiologically projecting from the distal humerus.

This spur is present in one to two per cent of the population and is connected by the ligament of Struthers to the medial humeral epicondyle. The pronator teres and coracobrachialis muscles may be attached to the ligament, which has also been noted to compress the ulnar nerve [8].

The anterior interosseous branch of the median nerve may be compressed between the two heads of the pronator teres muscle, resulting in weakness of the long finger flexors. Tenderness is often present over the nerve at this level, but as in any other case where uncertainty exists about the level of the nerve lesion, a nerve conduction study may be of use. Digital nerve compression has also been described [21] but is relatively uncommon and reasonably simple to diagnose. If symptoms become chronic, the digital nerve should be explored.

(c) **Radial Nerve.** The radial nerve is open to trauma as it winds round the spiral groove of the humerus. If a fracture occurs at the junction of middle and distal thirds of the humerus damage to the nerve is very likely since it is held against the bone at this site by the lateral intermuscular septum. However, non-traumatic palsies of the radial nerve may also occur in the groove, either from external pressure against the nerve or due to constriction from the fibrous arch of the lateral head of the triceps which bridges over the nerve. A supinator tunnel syndrome has also been described, in which the posterior interosseous nerve is compressed by the ligament of Frohse, resulting in weakness of the finger extensors and the ulnar extensor of the wrist. The extensor carpi radialis longus and brevis muscle branches come off the nerve before it enters this tunnel and therefore these muscles are not involved. Some cases of "tennis elbow" (lateral humeral epicondylitis) may be due to nerve entrapment, including the supinator tunnel syndrome, although this has not been established unequivocally. A compression of the radial nerve just prior to its division at the elbow has also been described but is rate. The cutaneous, terminal branches of the radial nerve may also produce painful paraesthesiae if trapped in scar tissue over the radial aspect of the wrist and this can prove difficult to manage surgically.

Other nerves may be compressed in the region of the shoulder, such as the supraclavicular nerve if it passes through the clavicle, the musculo-cutaneous nerve which may be entrapped as it passes through the coracobrachialis muscle, and the suprascapular nerve which can be compressed as it passes through the suprascapular notch[20].

2. Lower Limb

Meralgia paraesthetica. The lateral cutaneous nerve of thigh passes from the pelvis into the thigh beneath the inguinal ligament at its attachment to the anterior superior iliac spine. It may become wedged between this ligament, the iliac bone and the origin of the sartorius muscle, resulting in symptoms over the antero-lateral aspect of the thigh. A burning pain is described by the patient and this may be intermittent and positional. The condition appears to be less commonly diagnosed now and surgical release of the nerve does not always result in relief of symptoms. This may be due to the fact that the lateral end of the inguinal ligament is excised but the nerve may remain tented over the adjacent anterior iliac spine. Compression is therefore unrelieved.

The obturator nerve may be compressed by a hernia, tumour or abscess secondary to diverticular disease or appendicitis. Symptoms are often felt over the medial aspect of the knee but a proximal lesion must always be considered. The femoral nerve may be compressed by a haematoma of the posterior abdominal wall or by the lower edge of a plaster jacket at the groin. These proximal leg lesions are now less common than more distal lesions, the most clearly described of which are the tarsal tunnel syndrome, the lateral popliteal nerve compression, and Morton's metatarsalgia.

A **tarsal tunnel syndrome** may be produced by compression of the posterior tibial nerve as it runs under the laciniate ligament at the ankle[10] [12]. Symptoms may be produced by fatty infiltration, vascular engorgement, fibrosis, tenosynovitis, a valgus foot deformity, fractures of the ankle or calcaneum, or a neurilemmoma. There is pain over the instep and often an area of numbness over the distribution of the medial or lateral plantar nerves. Any weakness of the foot usually passes unnoticed. Eventually, however, the neuropathy manifests with weakness of metacarpophalangeal joint flexion and interphalangeal joint extension of the toes, resulting in clawing of the toes and a possible pes cavus deformity [11].

Treatment of the tarsal tunnel syndrome is once again surgical, with careful dissection and release of any fibrous compression involving the posterior tibial nerve or its medial plantar, lateral plantar and calcaneal branches. Conservative management using a medial sole flare or a foot brace to correct pes valgus may be tried prior to surgery[11] but neither this nor surgical release guarantee the belief of symptoms.

Lateral popliteal nerve compression is not a true entrapment neuropathy since it is usually an external pressure or a traction lesion which produces damage. The nerve is prone to injury as it commonly consists of one large funiculus and there is little in the way of connective tissue, this arrangement being less resilient than the more resistant architecture of multiple small funiculi ensheathed in a high proportion of connective tissue. It is for this same reason that the ulnar nerve is always so susceptible to injury at the elbow. However Kopell & Thompson [11] have drawn attention to a fibrous edge that de-limits the opening in the peroneus longus muscle through which the nerve passes. Compression may follow repeated inversion or combined inversion and plantar flexion of the foot, and thus they advocate the use of a lateral shoe wedge to keep the foot everted. If this conservative method fails they advise surgical exploration if prior lumbar myelography rules out sciatica from more proximal compression. More distal neuropathies have been encountered involving the superficial peroneal nerve if it is entrapped in the distal third of the leg where it penetrates the deep fascia and the deep peroneal nerve over the dorsum of the foot where it passes under the extensor hallucis brevis tendon and pierces the deep fascia.

Morton's neuroma is included in the list of peripheral entrapment neuropathies, although its aetiology is uncertain. The symptoms of pain under the forefoot, particularly in the web space between the third and fourth metatarsal necks, and of numbness in the associated cleft between the toes, are fairly characteristic. The patient often removes the shoe to massage the foot, and finds walking on hard ground extremely painful. Pain may also occur spontaneously at night. Trauma has been implicated in the onset of the symptoms, and at exploration a neuroma is found between the third and fourth metatarsal necks. There are those who advocate injection with lignocaine and hydrocortisone to control symptoms, but this is usually only of temporary benefit and the use of a local anaesthetic is of principal value diagnostically. The thickening found in the nerve at surgery is histologically a neuroma, with considerable fibrosis around it. Some of these changes may be due to the anatomical arrangement of the nerve at this site, or may even be due to a congenital or degenerative cause.

Entrapment neuropathies have also been described at the subsartorial canal involving the saphenous nerve and in relation to the lateral malleolus involving the sural nerve. Digital nerve compression has also been reported, and thus virtually every nerve in both the lower and upper limbs, whether motor, mixed or solely sensory, can be compressed pathologically.

Summary

Simple release of the carpal tunnel usually relieves completely the symptoms of median nerve compression. It is only rarely, such as in cases of uncertain diagnosis or with associated cervical spondylosis, that the operation fails. Motor and sensory recovery is seldom incomplete. Regrettably such success cannot be guaranteed with nerve release at other site. The ulnar nerve, for example, may remain symptomatic after cubital tunnel decompression in approximately one-third of cases. Even after anterior transposition the more chronic ulnar nerve lesion will rarely recover completely. In the lower limb entrapment neuropathies are far less common but can also be dealt with surgically. Nerve conduction studies may be helpful in difficult cases, but the essence of diagnosis is accurate history taking, a knowledge of anatomy, and experience in dealing with these interesting neuropathies.

From a dissertation read before the society

"On Diabetes"
Frederick Wright 1860
Writing of sugar in the urine;
'It resembles grape more nearly than cane sugar and is usually called *glucose*. It forms with common salt a compound which crystallises with facility. Glucose may be obtained from its solution by evaporating rapidly in a water bath, when a thick syrup is produced, resembling treacle. This should be placed in a shallow vessel for a fortnight, when the crystallisation is complete."

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