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REVIEW

Common peripheral nerve entrapments in the upper limb

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Abstract

Entrapment of peripheral nerves can occur as they travel through restrictive spaces. This nerve compression can result in a constellation of signs and symptoms, which are often called syndromes. Patients initially report pain, paraesthesia and numbness, followed by weakness and clumsiness and, ultimately, muscle wasting. The specific region of paraesthesia and pain and the specific muscle weakness is determined by the peripheral nerve involved and the location of the entrapment. Diagnosis is mainly based on history and examination. Further investigations are available for atypical presentations. Each syndrome has its own set of risk factors, but repetitive action and muscle overuse are commonly associated with most syndromes. The treatment is activity modification followed by steroid injection and finally surgical decompression for ongoing persistent symptoms or severe initial presentation. This article outlines the history, examination, possible investigations and management for common peripheral nerve entrapments of the median, ulnar and radial nerves.

Key words: Carpal tunnel syndrome; Cubital tunnel syndrome; Double crush syndrome; Median; Peripheral nerve entrapment; Ulnar

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Introduction

Peripheral nerve entrapments commonly occur when a peripheral nerve is compressed as it travels through narrow, restricted spaces between the spinal cord and the end organ. This manifests itself as pain and/or loss of nerve function; this may be sensory and/or motor. These signs and symptoms are commonly grouped together as entrapment syndromes. While usually straightforward, diagnosis of this common condition can be difficult if patients present with atypical signs and symptoms. This article outlines the diagnosis and management of syndromes caused by peripheral entrapment of upper limb nerves.

Clinical history and examination

While sensory, motor or mixed manifestations of nerve entrapments can exist at the time of presentation, sensory loss is the first facet of mixed nerve function to be affected. Further prolonged compression can lead to paraesthesia, loss of coordination, muscle weakness and, eventually, muscle wasting (Jobe and Martinez, 2013). Night symptoms are common because patients tend to remain static for long periods in a position that aggravates nerve entrapment, such as elbows held in flexion. Furthermore, when asleep, peripheral vasodilation results in a lowering of blood pressure and therefore reduced perfusion of the vasa nervorum overnight.

Predisposing conditions and risk factors for nerve compression syndromes are shown in Table 1 and should be fully explored.

Examination entails look, feel, move and special tests. Inspection (look) can reveal muscle wasting, deformity, asymmetry, scars and swelling. Sensory examination (feel) and muscle strength examination (move) should be guided by the peripheral nerve of concern and the location of suspected entrapment. It is important to note that motor weakness is usually a late sign, and not necessarily present in the early stages. There are eponymous names given to some special tests, depending on the nerve being examined. In general, these tests aim to either stimulate an already inflamed neural sheath, or to recreate compression of the nerve.

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Table 1. Conditions associated with nerve compression

Category of association	Associated condition
Systemic	Diabetes
	Alcoholism
	Renal failure
	Raynaud's phenomenon
Inflammatory	Rheumatoid arthritis
	Infection
	Gout
	Tenosynovitis
Fluid status	Pregnancy
	Obesity
Anatomical variance	Malunion secondary to trauma
	Synovial fibrosis
	Anomalous tendon
	Persistent median artery
Space-occupying lesions	Ganglion
	Lipoma
	Haematoma

Median nerve

The median nerve is vulnerable to entrapment at the following anatomical sites (Figure 1):

1. Ligament of Struthers
2. Lacertus fibrosus – bicipital aponeurosis
3. Between two heads of the pronator teres
4. Fibrous arch at the proximal flexor digitorum superficialis
5. Carpal tunnel.

The median nerve entrapment syndromes can be broadly divided into three syndromes based on the location of entrapment:

1. Carpal tunnel syndrome
2. Pronator syndrome
3. Kiloh–Nevin syndrome.

Carpal tunnel syndrome

Carpal tunnel syndrome is the most common peripheral neuropathy. A UK GP research database showed that 88 men and 193 women present as new cases per 100 000 population per year (Latinovic et al, 2006). The annual surgical decompression rate in the UK is around 43–74 per 100 000 population per year (Burke, 2000). Carpal tunnel syndrome is caused by compression of the median nerve as it passes through the narrow carpal tunnel in the wrist, thought to be caused by a combination of oedema, tendon inflammation, trauma, overuse and hormonal changes (Padua et al, 2016). The prevalence of carpal tunnel syndrome is higher in women than men, especially during pregnancy. Although the causes of carpal tunnel syndrome in pregnant women may be multi-factorial, the main driver is likely to be increased fluid retention during pregnancy, resulting in increased pressure in the carpal tunnel. A systematic review of the natural history of pregnancy-related carpal tunnel syndrome demonstrated that the majority of cases spontaneously resolved 1 year after birth (Padua et al, 2010).

Although there is wide variation in presentation, patients initially report nocturnal paraesthesia and pain in the radial three and a half digits, which increases in frequency to

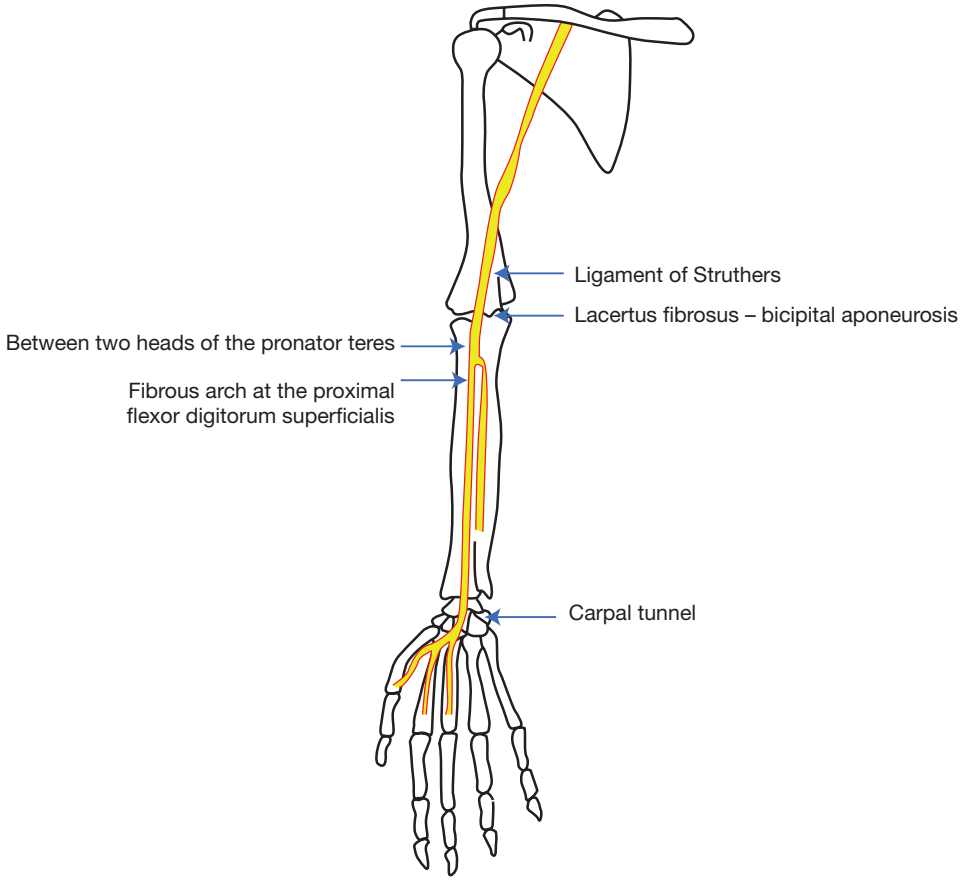

Figure 1. Common sites of median nerve entrapment.

occur in the daytime (Padua et al, 1999). With ongoing nerve compression, weakness and atrophy of the thenar muscles can develop and this is sometimes described by patients as clumsiness, particularly when gripping items. The median nerve also carries the majority of autonomic fibres to the hand and carpal tunnel syndrome can result in autonomous dysfunction such as reduced or loss of sweating (Kuwabara et al, 2008).

Classically, the sensory deficit described is of the radial three and a half digits with sparing of the ulnar side innervated by the unaffected ulnar nerve and sparing of the radial palmar surface innervated by the palmar cutaneous branch of the median nerve, which travels outside the carpal tunnel. However, it is not uncommon for patients to report numbness of the whole hand (Uluc et al, 2015). Around 55–65% of cases are bilateral at first presentation and most patients report symptoms initially in the dominant hand (Bland, 2003). Although digit and hand symptoms are the predominant feature, in more severe cases, patients report pain spreading more proximally to the forearm, upper arm and shoulder (Bland, 2007), with paraesthesia generally confined to the radial three and a half digits.

Diagnosis can be aided by provocation clinical examinations: Phalen's test (maximum wrist flexion for 60 seconds), Tinel's sign (finger percussion over the carpal tunnel), Durkan's test (manual compression of the carpal tunnel for 30 seconds) and the Phdurkan test (manual compression of the carpal tunnel with the wrist held in maximum flexion). Tests are considered positive if they result in paraesthesia in the median nerve distribution of the hand. There is a wide range of sensitivity and specificity reported for these tests (Brüske et al, 2002). As the examination manoeuvre becomes more provocative (from Tinel sign to Phalen's test, Durkan's test and Phdurkan test), the test becomes more sensitive but less specific (Zhang et al, 2020).

Investigation usually involves nerve conduction studies, which have a high sensitivity of 80–90% and high specificity of >95% (Jablecki et al, 2002). For the majority of patients diagnosed with carpal tunnel syndrome based on history and clinical examination, nerve conduction studies do not significantly change the probability of diagnosis (Graham,

2008). However, they are invaluable in cases of atypical presentations and are also used by clinicians to provide evidence of nerve compression before surgery (Sears et al, 2016).

Once diagnosed, first-line management should be optimisation of any underlying conditions such as hypothyroidism and rheumatoid arthritis, and reduction of any changeable risk factors, which may include awaiting resolution of pregnancy by birth. If elements of overuse are identified, change in behaviour to reduce repetitive strain on the volar wrist and reduction of heavy work activities should be recommended. The next line of treatment would be local corticosteroid injections, which improve symptoms (Chesterton et al, 2018) and nerve conduction outcomes (Ertem et al, 2019), and reduce the rate of surgery (Atroshi et al, 2013). If non-surgical treatments fail, the patient must be referred to an appropriate provider for consideration of carpal tunnel release. This can be done as an open or endoscopic procedure and both can be done under general or local anaesthesia. Compared to being performed under sedation, carpal tunnel release under local anaesthesia is equally effective and significantly cheaper (Alter et al, 2018), and there is less postoperative pain (Kang et al, 2019). As a result, this is now the more common method of carpal tunnel release. A 125% increased risk of subsequent nerve repair had been found with the endoscopic procedure compared with the open procedure (Trehan et al, 2019) but as the procedure has improved, more recently Williamson et al (2021) showed no statistically significant difference in complications. Although endoscopic carpal tunnel release has shown some benefit over open carpal tunnel release in the intensity of postoperative pain in the 24 hours after surgery (Orak et al, 2016), the significantly higher cost has meant that endoscopic procedures are still the minority of those performed for carpal tunnel syndrome.

Pronator syndrome

Pronator syndrome is a constellation of symptoms caused by peripheral entrapment of the median nerve at or around the elbow. The most common cause is entrapment between the two heads of the pronator teres and is often a result of overuse of this muscle (Lee and LaStayo, 2004).

The presentation can be similar to that of carpal tunnel syndrome but the more proximal entrapment means that there is also paraesthesia of the palm, volar elbow and forearm that are not present in carpal tunnel syndrome. Similar to carpal tunnel syndrome, the symptoms may be reproduced by tapping over the suspected entrapment location or by resisted forearm pronation. As it is an uncommon presentation, nerve conduction studies are very useful in reaching the correct diagnosis. Management is primarily conservative with change in lifestyle to avoid repeated pronation of the concerned limb.

Kiloh–Nevin syndrome

The two heads of the pronator teres or the fibrous arch of the proximal flexor digitorum superficialis can also compress the anterior interosseous nerve. Patients will have a weakness of the flexor pollicis longus and the radial half of the flexor digitorum profundus. The anterior interosseous nerve may be affected in conjunction with pronator syndrome or present as an isolated syndrome, also called Kiloh–Nevin syndrome (Kiloh and Nevin, 1952). Unlike pronator syndrome, patients usually present with a painless weakness. Again, as it is an uncommon presentation, nerve conduction studies are very useful in ruling out alternative diagnoses.

Ulnar nerve

The most common locations for entrapment of the ulnar nerve from proximal to distal are shown in Figure 2.

1. Medial intermuscular septum
2. Arcade of Struthers
3. Medial epicondyle
4. Cubital tunnel
5. Fibrous arch of the flexor carpi ulnaris
6. Guyon's canal.

The collection of signs and symptoms caused by ulnar nerve entrapment can be broadly divided into cubital tunnel syndrome and Guyon canal syndrome.

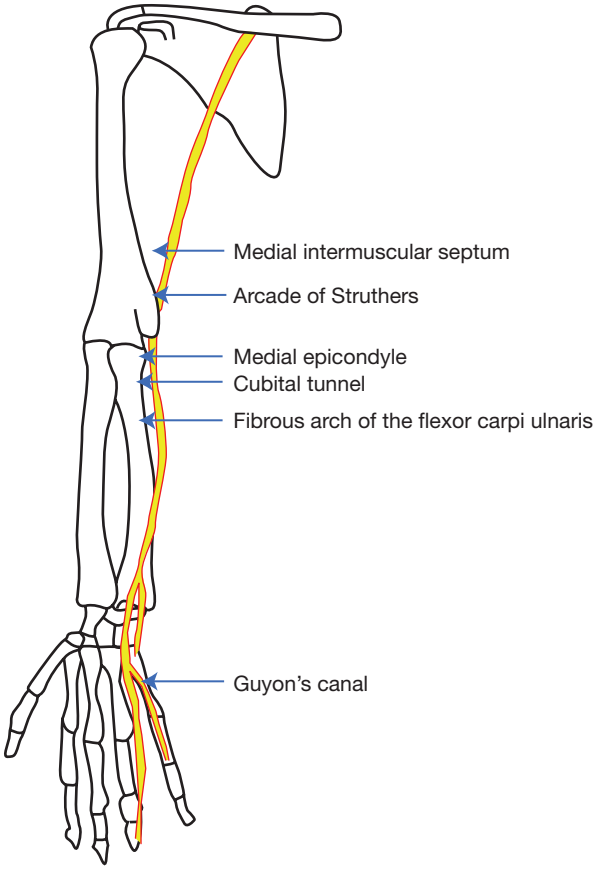

Figure 2. Common sites of ulnar nerve entrapment.

Cubital tunnel syndrome

This is the second most common syndrome of peripheral nerve neuropathy after carpal tunnel syndrome, with 25 men and 19 women presenting as new cases per 100 000 population per year (Latinovic et al, 2006). Entrapment at the medial intermuscular septum, arcade of Struthers, medial epicondyle, cubital tunnel or the fibrous arch of the flexor carpi ulnaris can cause cubital tunnel syndrome, but the most common location is at the cubital tunnel. There are several risk factors associated with the syndrome, such as obesity and diabetes, but repeated physical labour involving a flexed elbow seems to be the most important (van Rijn et al, 2008). Athletes such as baseball pitchers, who repeatedly throw objects, are also susceptible to cubital tunnel syndrome (Cummins and Schneider, 2009). While not strictly a peripheral entrapment, people with recurrent anterior subluxation of the ulnar nerve are also at risk of dynamic ulnar neuropathy, resulting in cubital tunnel syndrome. This recurrent translation can be felt as a snapping sensation during flexion (Spinner and Goldner, 1998), but has also been reported in up to 20% of asymptomatic people (Okamoto et al, 2000). Tardy ulnar nerve palsy is another form of cubital tunnel syndrome, which can be caused by elbow deformity secondary to childhood fractures such as lateral condyle or supracondylar fractures, but only presents in adulthood.

Patients with cubital tunnel syndrome present with an ill-defined pain or ache around the elbow and forearm, with accompanying paraesthesia of the dorsal and volar aspect of the ulnar one and a half digits. With progression of pathology, patients can report clumsiness and weakness of the hand. As people tend to sleep with flexed elbows, night-time exacerbation is common.

Examination in the early stages can reveal numbness as described above. Provocative tests can be used; tapping over the cubital tunnel or keeping the elbow flexed for 60 seconds can both elicit or exacerbate the symptoms. As the condition progresses, objective motor weakness, such as a weakened pinch grip, may be seen. In late stages, atrophy of the intrinsic muscles of the hand, and even clawing of the fingers, can be observed.

Diagnosis is made primarily on good history taking and examination. Nerve conduction studies and electromyography can be helpful in establishing the diagnosis and prognosis in cases of atypical or mixed presentation with carpal tunnel syndrome.

The first line of treatment is activity modification to avoid elbow flexion and the use of elbow flexion splints but the compliance with these tend to be lower than wrist splints for carpal tunnel syndrome. Patients that continue to have symptoms despite conservative management should be referred to a specialist for consideration of further investigation and decompression surgery.

Guyon canal syndrome (ulnar tunnel syndrome)

Guyon's canal is a 4 cm fibro-osseous canal in the medial aspect of the palm, through which the ulnar nerve travels. If it becomes entrapped, this causes Guyon canal syndrome, also known as ulnar tunnel syndrome. This is a rare presentation and compression at this level may be caused by pathology intrinsic or extrinsic to Guyon's canal. Intrinsic compression is caused by space-occupying lesions, such as pseudoaneurysms or ulnar artery thrombosis, but most commonly ganglion cysts. Extrinsic compression is usually a result of sustained physical activity, such as racquet sports and prolonged cycling with the palm leaning on the handlebars (Patterson et al, 2003). Trauma to Guyon's canal such as fracture or non-union of the hook of hamate or pisiform can also result in Guyon canal syndrome.

Radial nerve

Radial nerve entrapment (Figure 3) is very rare and alternative diagnoses such as radiculopathy and neuropraxia need to be ruled out. Such uncommon presentations warrant further investigations.

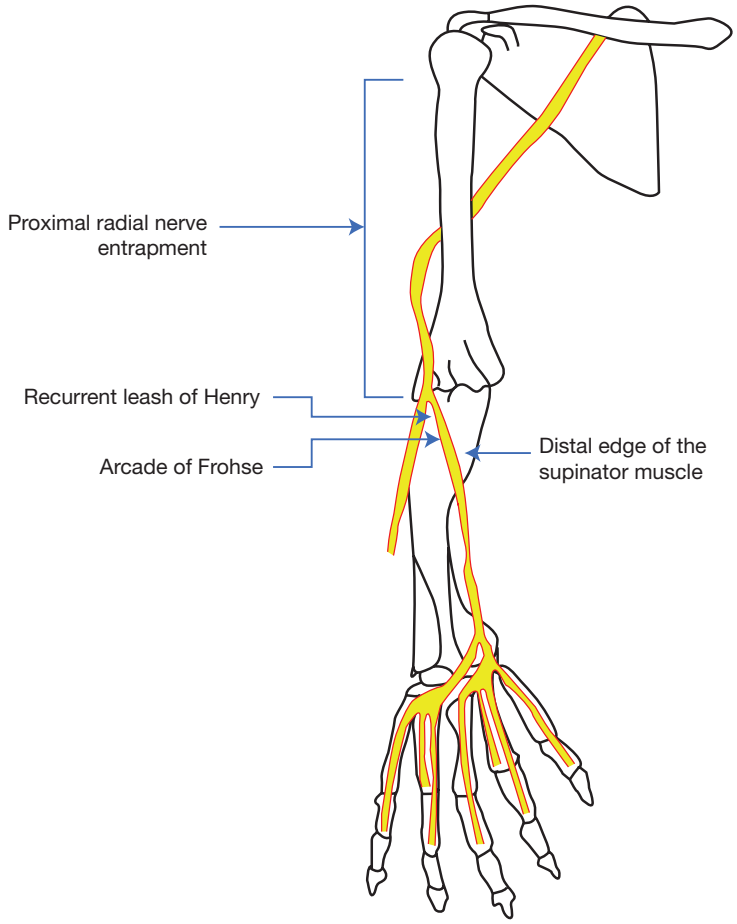

Figure 3. Common sites of radial nerve entrapment.

Posterior interosseous nerve syndrome

Posterior interosseous nerve syndrome can be caused by entrapment of the posterior interosseous nerve, a branch of the radial nerve, at the following sites:

1. Recurrent leash of Henry – recurrent radial artery branches that fan out across the posterior interosseous nerve
2. Arcade of Frohse
3. Distal edge of the supinator muscle.

The arcade of Frohse is a thickened proximal edge of the supinator and the most common cause of posterior interosseous nerve syndrome. This is likely a result of repetitive pronation and supination movement, putting manual labourers at risk (Dang and Rodner, 2009). Diagnosis of posterior interosseous nerve syndrome is made clinically with thumb and wrist extensor weakness in the absence of sensory deficits (Suematsu and Hirayama, 1998). Patients may also experience a burning sensation along the lateral aspect of the forearm mimicking lateral epicondylitis. Treatment is primarily conservative with lifestyle modification; surgical decompression is reserved for failed conservative management.

Double crush syndrome

Double crush syndrome is a compression of a peripheral nerve at two or more locations along its course that can coexist and synergistically increase the symptom intensity. The exact mechanism of this phenomenon is uncertain (Schmid and Coppieters, 2011). Clinical examination combined with electrodiagnostic studies can help with diagnosis of double crush syndrome. Furthermore, a magnetic resonance imaging scan can identify more proximal pathologies, such as degenerative discs or neuroforaminal narrowing, which may coexist with a more distal nerve compression.

In patients with a strong suspicion of double crush syndrome and failure of conservative measures, surgical intervention in the form of simple decompression of the peripheral site is usually addressed first and the patient is counselled about the possibility of incomplete relief.

Further investigation

Diagnosis of most peripheral nerve entrapments is based primarily on careful history taking and clinical examination. However, in atypical or mixed presentations and recurrences after surgical treatment, various investigations may be of value in diagnosis and management.

Imaging

Plain X-rays are of limited value but can be used to identify fractures, acute or old, as the cause of nerve entrapment.

Ultrasound can be used to assess possible soft tissue causes of entrapment and can demonstrate an increased cross-sectional area of a nerve just before the site of compression (Yoon et al, 2008), but the process is highly operator dependent (Martinoli et al, 2007).

Secondary signs of nerve entrapment, such as focal nerve flattening, local oedema and denervation changes in the muscle, can be seen on magnetic resonance imaging. However, a normal magnetic resonance imaging finding does not rule out nerve injury (Neal and Fields, 2010) and, given the cost, magnetic resonance imaging has limited use in primary diagnosis in typical presentations. However, it is very useful in identifying alternative diagnoses, space-occupying lesions and to identify incomplete soft tissue resection in cases of recurrence after surgical intervention.

Electrodiagnostic testing

Nerve conduction studies are the investigation of choice for peripheral nerve entrapments. They assess the integrity of sensory and motor nerves with areas of injury and demyelination appearing as slowing of conduction velocity. Electromyography records the electrical activity of a muscle from a needle placed into the muscle to look for signs of denervation. For most patients with a typical presentation of a common peripheral nerve entrapment, electrodiagnostic testing does not change the diagnosis (Graham, 2008) or management

(Finsen and Russwurm, 2001). However, in rare, atypical or mixed presentations, nerve conduction studies are recommended to confirm the diagnosis.

Nerve conduction studies can be used to help differentiate carpal tunnel syndrome from pathologies that mimic it such as diabetic polyneuropathy and radiculopathies (Ito et al, 2014). The British Orthopaedic Association and Royal College of Surgeons of England (2017) advocate the use of nerve conduction studies in cases of equivocal clinical examination and history, persistent or recurrent carpal tunnel syndrome and an unclear diagnosis suggesting peripheral neuropathy.

Treatment

The natural history of each peripheral nerve entrapment varies greatly and so does the need for non-surgical and surgical treatment. First, driving factors contributing to entrapment must be reduced with education and lifestyle modification. These may be repetitive actions or flexed joint positions held for long durations. This can be supplemented with splinting, with good evidence for the use of night-time neutral wrist splints for carpal tunnel syndrome (Shi and MacDermid, 2011) and some evidence for elbow flexion splints to treat mild to moderate cubital tunnel syndrome (Kooner et al, 2019). If non-surgical interventions do not result in improvement, patients should be referred to a specialist for further investigation and consideration of ultrasound-guided injection or surgical intervention. Patients who initially present with severe symptoms may be considered for referral as they are likely to benefit from early surgical intervention (Chandra et al, 2013). For carpal tunnel syndrome and cubital tunnel syndrome, the British Orthopaedic Association and Royal College of Surgeons of England (2017) recommend referral to a secondary care provider for persistent symptoms and disability not responding to up to 6 weeks of evidence-based non-surgical treatments or sudden and severe symptoms.

Although 1 in 4 patients with carpal tunnel syndrome is expected to have symptom resolution without any intervention within 1 year (Knott, 2017), the presence of several factors increases the likelihood of unsuccessful management with non-surgical intervention: age over 50 years, duration over 10 months, constant paraesthesia, stenosing flexor tenosynovitis, and a Phalen's test that is positive in less than 30 seconds (Kaplan et al, 1990). The presence of severe symptoms or a number of these factors should encourage clinicians to refer to secondary care for consideration of surgery at an early stage.

Conclusions

The history and clinical examination findings in peripheral nerve entrapment depend on the peripheral nerve and its location of entrapment but often entails an element of muscle

Key points

- Diagnosis of peripheral nerve entrapment is based primarily on history taking and clinical examination. For most patients with a typical presentation, further investigations do not change the diagnosis or outcome.
- Examination is dependent on the specific nerve and location of entrapment but tends to involve either stimulating an already inflamed neural sheath, or recreating compression of the nerve. This is done by tapping the location of entrapment or positioning a limb to recreate the compression.
- Nerve conduction studies is not recommended for typical presentations of common peripheral nerve entrapments but provide valuable information in uncommon or atypical presentations as well as post-surgical recurrences.
- Mild to moderate presentations of common peripheral nerve entrapments should be managed with activity modification and splints. In cases of severe symptoms or failure of initial conservative management, patients must be referred to a specialist for further investigation and consideration of surgical decompression.

overuse or repeated actions. Diagnosis is based primarily on good history taking and examination, with further investigations reserved for atypical presentations and recurrences. For typical mild to moderate cases, a trial of evidence-based non-surgical intervention such as activity modification and splints should be given. In cases of atypical presentations, severe symptoms or a failure of initial conservative management, patients must be referred early to specialists for further investigation and consideration for corticosteroid injection or surgical decompression.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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