

Paraesthesia and peripheral neuropathy



Roy Beran



Background

Paraesthesia reflects an abnormality affecting the sensory pathways anywhere between the peripheral sensory nervous system and the sensory cortex. As with all neurology, the fundamental diagnostic tool is a concise history, devoid of potentially ambiguous jargon, which properly reflects the true nature of what the patient is experiencing, provocateurs, precipitating and relieving factors, concomitant illnesses, such as diabetes, and any treatments that could evoke neuropathies.

Objective

Some localised neuropathies, such as carpal tunnel syndrome (CTS) or ulnar neuropathy, produce classical features, such as weakness of the 'LOAF' (lateral two lumbricals, opponens pollicis, abductor pollicis brevis and flexor pollicis brevis) median innervated muscles, thereby obviating need for further neurophysiology. Nerve conduction studies may be necessary to diagnose peripheral neuropathy, but they may also be normal with small fibre neuropathy. Even with a diagnosis of peripheral neuropathy, definition of the underlying cause may remain elusive in a significant proportion of cases, despite involvement of consultants.

Discussion

Treatment is based on the relevant diagnosis and mechanism to address the cause. This includes better glycaemic control for diabetes, night splint for CTS or elbow padding for ulnar neuropathy, modifying lifestyle with reduced alcohol consumption or replacing dietary deficiencies or changing medications where appropriate and practical. Should such intervention fail to relieve symptoms, consideration of intervention to relieve symptoms of neuropathic pain may be required.

Keywords

peripheral nervous system diseases

Peripheral neuropathy is such a broad subject that it is impossible to do it justice in a brief overview such as this. The topic, for the preparation of this review, was to explore 'paraesthesia and peripheral neuropathy' with a focus on the needs of the general practitioner (GP). It follows that this paper will, of necessity, be somewhat superficial, the goal being to assist GPs in their approach to patients with paraesthesia. Some of the investigations and management will be touched on but further consideration is reserved for the consultant. To offer maximal value and focus, the style adopted will be pragmatic, trying to offer some pearls, rather than offer a comprehensive treatise as is available in any standard text.

History

As is the case with all neurology, the most important diagnostic tool is a detailed comprehensive history that reflects what the patient is trying to convey. Patients will often volunteer vague terms, such as 'numb', and need help to tease out what they are describing. The term 'numb' may mean abnormal sensation, including loss of feeling, tingling, pins and needles, electric shock-like sensations, a feeling of heaviness or loss of function, a loss of dexterity, or a host of other possibilities, depending on the individual patient. Patients do not present with the complaint of 'paraesthesia' but will often complain of 'numbness' and it is imperative to determine if this means paraesthesia, before embarking on a diagnostic paradigm.¹

Paraesthesia is a technical term used to reflect a perception of abnormal sensation, including feelings of pins and needles, tingling, pricking or a feeling as if ants are crawling over/under the skin and patients should be encouraged to fully describe what they are feeling. Patients should be encouraged to use their own language, rather than trying to inject jargon, which may be ambiguous and fail to really convey what the patient is trying to describe. Paresthesia suggests abnormality affecting the sensory nervous system and may arise anywhere from the peripheral nerve to the sensory cortex. It is one of the terms

attached to peripheral neuropathy for which there is a lexicon (*Table 1*). Patients should describe what they are feeling and the distribution of the abnormal sensation, what provokes it, when it started, what, if anything, they can do to relieve it and any associated features.¹

When taking a history from the patient, it is important to explore other features, such as concomitant illnesses, medications and/or drugs used, other treatments provided, past medical history and family history, system review and exposure to chemicals. Perhaps the most common diagnosis associated with peripheral neuropathy is diabetes² and paraesthesia is often accepted as the hallmark symptom for neuropathic pain.¹ It is important to differentiate the discomfort of paraesthesia from other sources of pain, such as musculoskeletal pain, as may occur with osteoarthritis.

Focus on aetiology

Clinicians will often overlook the potential for iatrogenically induced peripheral neuropathy, as may occur with frequently used medications, including amiodarone, statins, antiretrovirals, tacrolimus or even agents not often considered to be associated with peripheral neuropathy, such as levodopa, which is commonly used to treat Parkinson’s disease.³ It is widely accepted that various chemotherapies for malignancy can cause peripheral neuropathy, including taxanes, platinum compounds, vinca alkaloids, proteasome inhibitors and antiangiogenic/immunomodulatory agents.⁴ Deficiencies of vitamins, such as B6 or B12, may evoke peripheral neuropathies and may be associated with therapies,

including levodopa.³ This may necessitate special consideration, especially in vegans.

Other dietary deficiencies may be associated with peripheral neuropathy. For example, toxic levels of homocysteine are associated with vitamin B6 and B12 deficiencies.² Medications such as metformin cause B12 deficiency⁵ as do other medications, such as phenytoin, which reduces folate needed for B12.⁶ There has been a push to offer dietary supplements, including vitamins B6, B12, D and E, and magnesium to address problems with peripheral neuropathy.^{2,7} Patients who are alcohol-dependent often have a variety of causes for peripheral neuropathy, which include both direct toxic effects of excess alcohol as well as poor diet, particularly deficient in thiamine (vitamin B1).⁸ It follows that the history obtained should include the usual causes of nerve damage being explored but doctors often ignore taking an adequate dietary history and nutritional neuropathies may be overlooked.⁹

Diagnosis

If a detailed history is taken, the diagnosis of peripheral neuropathy should be straightforward. The suspicion should have been established and features such as a ‘glove and stocking’ abnormal sensation should be a giveaway to suggest peripheral neuropathy.

What has not been addressed, thus far, are some of the very localised peripheral neuropathies that may present with very localised paraesthesia. These include carpal tunnel syndrome¹⁰ or ulnar neuropathy,¹¹ which relate to direct pressure or vascular supply to the nerve.¹¹

Table 1. Glossary of pain terms

Term	Meaning
Allodynia	Pain perceived following non-noxious, innocuous stimulus (eg light touch causes burning pain)
Antalgia (antalgic)	Pain perception (noun), pain provoked action (adjective) (eg antalgic gait – altered gait due to the influence of pain)
Dysaesthesia	An altered perception of sensation with abnormal (often unpleasant) feeling associated with stimulation, such as touching over the affected area causes ‘strange feeling’
Hypaesthesia/hypoaesthesia	Reduced perception of stimulus (both words are interchangeable) Decreased sensation
Hyperalgesia	Increased perception of pain
Hyperaesthesia	Increased perception of stimulus (need not be pain)
Hyperpathia	Decreased sensation to one or more modalities while concurrently having increased perception of pain (hyperalgesia) or pain with innocuous stimulation (allodynia)
Hypoalgesia	Reduced perception of pain
Paraesthesia	Abnormal sensations, such as ‘pins and needles’, tingling, prickling, reduced or even loss of sensation. It implies abnormality anywhere along the sensory pathway from peripheral nerve to sensory cortex – the epitome of ‘neuropathic pain’

Reproduced with permission from Beran R. Neurology for General Practitioners. Sydney: Elsevier Australia, 2012

Focal neuropathies

A proper history and examination may be all that is required to diagnose carpal tunnel neuropathy or ulnar neuropathy. The median nerve supplies only four muscles in the hand, represented by the mnemonic 'LOAF': lateral two lumbricals; opponens pollicis, abductor pollicis brevis and flexor pollicis brevis.¹ Patients will complain of paraesthesia in the hand but the history may be very vague, although it usually includes being woken from sleep because of the dysaesthesia.¹ Weakness, restricted to these four muscles of the hands, is pathognomonic of median nerve impairment, as is found with carpal tunnel syndrome. Similarly, sparing of the LOAF muscles of the hand through involvement of the abductor digiti minimi and medial two lumbricals is indicative of ulnar neuropathy, usually traumatised by turning over in bed and focusing the full body weight on the elbow, where the ulnar nerve traverses the medial humeral epicondyle.¹

If the above has been found then the diagnosis has been made, thereby negating a need for nerve conduction studies unless the patient fails to respond to conservative intervention with either a night splint for carpal tunnel or padding of the medial epicondyle of the elbow for ulnar neuropathy.¹ In addition to diagnosing these focal neuropathies and instituting local conservative intervention, it is imperative to exclude contributory diagnoses, such as vitamin deficiencies, thyroid disease, vasculitides, diabetes and various medications, such as those already described, together with other agents, such as amiodarone, nitrofurantoin or statins.¹ It follows that other investigations are warranted, particularly blood tests to look for these associated diagnoses.

Proper physical examination should have defined the distribution of the dysaesthesia reflective of the paraesthesia, which was the presenting symptom. Impairment of deep tendon reflexes, especially at the periphery (such as ankle jerks) provides additional confirmatory evidence of a possible sensory–motor neuropathy. When testing sensation, movement from impaired sensation to retained sensation is favoured, thus moving from the periphery, passing distal-to-proximal (foot, ankle, calf to knee), is preferable as it is easier for a patient to appreciate increased perception, especially pin prick and light touch, than is the reverse. This will help demarcate the 'glove and stocking' impaired sensation that accompanies peripheral neuropathy.

Investigations

Neurophysiology, using nerve conduction studies and electromyography, may be required, especially for the patient who proves to be a very poor historian or in whom confirmation of a diagnosis may dictate altered therapy. This may be the case in a poorly compliant patient whose diabetes is uncontrolled and in whom such confirmation may encourage better compliance, as well as the possible addition of insulin to

the regimen. It must be acknowledged that nerve conduction studies may not be abnormal in small fibre neuropathy yet the patient may still present with neuropathic symptoms. A perfect example of this is the painful diabetic neuropathy, which requires insulin and improved glycaemic control.

For more complex cases of peripheral neuropathy, there may be a need for more detailed investigation including evoked studies, imaging (possibly with magnetic resonance imaging or ultrasonography) and nerve biopsy. In these cases it seems appropriate to involve a consultant neurologist, which implies further consideration that is beyond the scope of this paper. Even with the involvement of consultant colleagues and comprehensive evaluation, the underlying mechanism of a significant proportion of neuropathies remains indeterminate.

Treatment

Treatment is determined by the relevant diagnosis. By far the most common cause of peripheral neuropathy, heralded by paraesthesia, is diabetes mellitus. Once diagnosed and confirmed, patients should be more closely monitored, encouraged to be more compliant with the prescribed treatment and, in many cases, insulin should be added to the treatment regimen,¹ together with nutritional approaches.²

Nutritional neuropathies are addressed by correcting the nutritional deficiencies that have been identified.^{7–9} Those neuropathies caused by exposure to medications are addressed by reconsidering treatment options, when possible, and close monitoring of the consequences complemented by conservative supportive symptomatic relief.

Carpal tunnel syndrome may be provoked by direct trauma to the median nerve, as may occur with exposure to vibrating machinery or direct impact from repeated use of tools, such as a screwdriver pushing into the palm of the hand, but by far the most common cause is sleeping with the wrist hyperflexed, thereby impeding vascular supply to the median nerve. Treatment with a night splint, specifically prepared for the individual patient (not a generic splint) designed to be in the 'natural' position (mildly dorsiflexed rather than the flat, 'neutral' position) is usually all that is needed. Padding of the elbow to protect the ulnar nerve as it passes over the humeral epicondyle when turning over in bed at night is often sufficient to relieve ulnar neuropraxia. The response to therapy is predicated by the severity of the problem at the time of presentation and hence early detection and intervention are invaluable.

Addressing the underlying cause of the peripheral neuropathy, which presents with paraesthesia, is the initial approach to intervention. There may be a need to consider lifestyle issues and the involvement of other health practitioners, such as occupational therapists, physiotherapists or podiatrists, and review of fundamental factors such as diet, alcohol consumption and medications. Should the paraesthesia

persist, even after treating the cause of neuropathy, the question of neuropathic pain assumes greater significance.

Treatment of neuropathic pain is beyond the scope of this paper, which has focused specifically on paraesthesia and peripheral neuropathy. There is a host of agents available to treat neuropathic pain, including antidepressants (tricyclic antidepressants and serotonin and noradrenaline reuptake inhibitors), antiepileptic medications (such as carbamazepine or pregabalin), antispasmodics (such as baclofen) and a variety of other treatments (including topical analgesics).

Conclusion

As with all neurological conditions, the most important diagnostic tool is a good history, complemented by physical examination – usually to confirm the diagnosis, already considered, based on the history obtained. Paraesthesia is the usual presentation for a sensory neuropathy, which may affect the sensory pathway from peripheral nerve to sensory cortex. Having made the diagnosis, many of the conditions, but particularly carpal tunnel syndrome and ulnar neuropathy, can be effectively managed by the GP without requiring consultant involvement. Addressing the underlying problem is often all that is required but, in complex cases, referral to a specialist may be necessary.

Author

Roy Beran MBBS, MD, FRCP, FRACGP, FACLM, B LegS, Consultant Neurologist, Conjoint Associate Professor of Medicine, Department of Medicine, University of New South Wales, Sydney, NSW; Professor, School of Medicine, Griffith University, Gold Coast, QLD. roy.beran@unsw.edu.au

Competing interests: None.

Provenance and peer review: Commissioned, externally peer reviewed.

References

- Beran RG. *Neurology for General Practitioners*. Sydney: Elsevier, 2012.
- Curtis L. Nutritional approaches to treat diabetic neuropathy: a systematic review. *Int J Diab Research* 2013;2:56–60.
- Muller T, van Laar T, Comblath DR, et al. Peripheral neuropathy in Parkinson's disease: levodopa exposure and implications for duodenal delivery. *Parkinsonism Relat Disord* 2013;19:501–07.
- Argyriou AA, Kyritsis AP, Makatsoris T, Kalofonos HP. Chemotherapy-induced peripheral neuropathy in adults: A comprehensive update of the literature. *Cancer Manag Res* 2014;6:135–47.
- Singh AK, Kumar A, Karmakar D, Jha RK. Association of B12 deficiency and clinical neuropathy with metformin use in type 2 diabetes patients. *J Postgrad Med* 2013;59:253–57.
- Shorvon SD, Reynolds EH. Anticonvulsant peripheral neuropathy: A clinical and electrophysiological study of patients on single drug treatment with phenytoin, carbamazepine or barbiturates. *J Neurol Neurosurg Psychiatry* 1982;45:620–26.
- Reynolds E. Vitamin B12, folic acid and the nervous system. *Lancet Neurol* 2006;5:949–60.
- Koike H, Sobue G. Alcoholic Neuropathy. *Curr Opin Neurol* 2006;19:481–86.
- Kumar N. Nutritional Neuropathies. *Neurol Clin* 2007;25:209–55.
- Bland JD. Carpal Tunnel Syndrome. *BMJ* 2007;335:343–46.
- Werner CO, Ohlin P, Elmqvist D. Pressures recorded in ulnar neuropathy. *Acta Orthopod Scand* 1985;56:404–06.