The evaluation of numbness and paresthesias in geriatric patients can present a particular challenge to the primary care physician. Careful sensory examination, in combination with recognition of motor and reflex involvement, will suggest a pattern that aids in neuroanatomic localization. This article reviews the common patterns seen in polyneuropathies, focal neuropathies, plexopathies and radiculopathies. Central nervous system etiologies also are mentioned. The differential diagnosis and further evaluation of sensory disturbances in the elderly population are discussed.

Key words: paresthesias, numbness, neuropathy, radiculopathy, plexopathy.

The evaluation of sensory complaints in patients of any age can present a diagnostic challenge to the primary care physician. Numbness (loss of sensation), paresthesias (abnormal spontaneous sensations) and dysesthesias (unpleasant sensations to stimulation) may be difficult for patients to describe and even more difficult for the clinician to localize and characterize. In older adults, the situation can be complicated by comorbid conditions, memory and cognitive difficulties, and a wider range of what is considered “normal” on the neurologic exam.1 Nonetheless, recognition of the basic patterns of common disorders can aid in neuroanatomic localization and can direct further evaluation.

Polyneuropathies
In polyneuropathies, sensory loss and paresthesias are generally symmetric with greater distal than proximal involvement, described as the “stocking-glove” pattern. The feet are usually involved first, and there may be no upper extremity involvement at the time of presentation. In fact, lower extremity involvement usually progresses to approximately knee level by the time the hands become involved.2 The most important historic factor to glean from the patient is the time course of progression, as the differential diagnoses for acute versus chronic polyneuropathies are distinct. The examination should focus not only on localization of sensory loss, but also on which modalities are most compromised. Some neuropathies preferentially affect sensation of vibration and proprioception (large fibre), while others affect pain and temperature (small fibre). Motor involvement may be limited to subtle weakness of the feet or intrinsic hand muscles, or more prominent with distal muscle wasting, foot drop or even proximal leg weakness. Symmetric loss of deep tendon reflexes, distally then proximally, is also an expected finding in large fibre polyneuropathy. It must be kept in mind that modestly reduced vibratory sensation and ankle areflexia can be seen in the healthy elderly population.1,3

The nerve conduction study/electromyography (NCS/EMG) can be helpful in narrowing the differential diagnosis of a patient’s polyneuropathy. It may quantify the neuropathy, distinguish acuity from chronicity, and find evidence of demyelination. It is important to interpret this study in the context of the patient’s age. Reduction in distal leg sensory responses and even mild demyelination are considered normal in patients older than 60 years.1,4

The list of polyneuropathies that evolve acutely—over days to weeks—is short. Guillain-Barre Syndrome (acute inflammatory demyelinating polyneuropathy), the most common etiology in the ambulatory elderly population, should be identified early as it is treatable.5 Other etiologies include vasculitis, porphyria, toxins and medications.

The list of chronic polyneuropathies is much longer and, in addition to a careful history to elicit risk factors such as alcoholism and certain medications, at least some blood work is usually required to identify a cause. The most common identifiable etiology is diabetic polyneuropathy which usually occurs after diabetes of greater than five years’ duration.6 Polyneuropathies related to malignancy are also important in this population.5 A relapsing-remitting course, early arm involvement or proximal weakness should raise suspicion of chronic inflammatory demyelinating polyneuropathy (CIDP) or other autoimmune-mediated polyneuropathies.

In addition to screening for diabetes, the initial serologic evaluation of chronic polyneuropathies generally should include a complete blood count, serum electrolytes, liver function studies, thyroid function studies, B12 level, sedimentation rate, chest X-ray and screening for paraproteins with serum protein electrophoresis and immunofixation.7 Further investigations, including autoimmune and infectious serologies, heavy metal screens, paraneoplastic antibodies, anti-nerve antibodies, lumbar puncture and nerve biopsy, should be determined by the specific clinical pattern of the neuropathy.5 Even after comprehensive evaluation, more than 20% of chronic polyneuropathies will be of undetermined cause and labeled “idiopathic” or “cryptogenic”.4

Focal Neuropathies
Focal neuropathies, usually from compression or entrapment, often present with pain or paresthesias before weakness becomes apparent. Careful delineation of the exact anatomic distribution of the sensory involvement should reveal the focal nature of the neuropathy (Figure 1). NCS/EMG is the most helpful
investigation to confirm the focal neuropathy and to estimate the severity.

Median neuropathy at the wrist, or “carpal tunnel syndrome”, is the most common and typically produces pain and paresthesias of the thumb and adjacent two or three fingers, often nocturnally, upon awakening or while holding the hands in certain postures during activities. However, many patients will report involvement of the entire hand or isolated involvement of one or two fingers. Wrist pain is a variable complaint, and there can be radiation of paresthesias or pain to the forearm or more proximally.9

Ulnar neuropathy at the elbow, the second most common entrapment neuropathy, typically produces numbness and paresthesias of the fourth and fifth digits and may produce tenderness at the elbow and weakness or atrophy of intrinsic hand muscles. Neuropathy of the radial nerve usually presents with motor manifestations, such as wrist drop. Depending on the location of injury, numbness, pain or paresthesias of the dorsum of the wrist, thumb, index and middle fingers may be present.10

In the legs, peroneal neuropathy is the most common compressive neuropathy, usually occurring as the nerve crosses the fibular neck below the knee. External compression, such as during operative positioning, bed rest or leg crossing, is often a factor. Recent weight loss appears to increase the risk. Foot drop or weakness is the chief complaint, but patients commonly have sensory loss over the lower calf and dorsum of the foot.9 Tarsal tunnel syndrome, although frequently discussed, is quite uncommon. It occurs from compression of the tibial nerve posterior to the medial malleolus and presents with foot pain. Sensory loss and paresthesias can involve the entire sole of the foot or be restricted to the medial or lateral plantar aspect.10

Lateral femoral cutaneous neuropathy, historically coined “meralgia paresthetica”, is a fairly common, purely sensory mononeuropathy of the leg. Numbness, paresthesias or dysesthesias over the anterior-lateral thigh are caused by entrapment of the nerve as it passes under or through the inguinal ligament. Tight belts, obesity and pregnancy have traditionally been invoked as inciting factors.10

**Plexopathies**

When patterns of sensory and motor involvement of a limb do not fit a peripheral nerve or root distribution, the possibility of a plexus lesion should be entertained and may be suggested by NCS/EMG. Trauma is the major cause of brachial plexus lesions, and symptoms usually are dominated by pain with sensory loss or paresthesias of the arm and hand. Etiologies include motor vehicle accidents, operative positioning and dislocation of the shoulder.11 Upper trunk injury causes pain across the trapezius ridge and down the medial scapular border with shoulder girdle weakness. Lateral cord involvement affects sensation of the lateral forearm, and thumb, index and radial third fingers. Medial cord involvement results in paresthesias and numbness of the medial upper arm, forearm, and the fourth, fifth and medial third fingers.12 Lower trunk injury affects sensation of the medial arm, forearm and hand with intrinsic hand muscle weakness, and may be accompanied by an ipsilateral Horner’s syndrome (ptosis, miosis and anhydrosis).13

Two non-traumatic sources of brachial plexopathies should be considered in the aging population and require MRI imaging. Metastatic infiltration of the plexus from breast or lung carcinoma presents with pain in the supraclavicular or axillary region and is accompanied by sensory and motor deficits in a lower trunk pattern.13 Post-radiation plexopathy occurs months to years after treatment for breast cancer, lung cancer or lymphoma and results in progressive paresthesias of median-innervated fingers and weakness of intrinsic hand muscles with variable pain.

Lumbosacral plexopathies occur less frequently than brachial plexopathies, and trauma is not generally a factor. Compression of the lumbosacral plexus from a pelvic tumour or retroperitoneal hemorrhage causes low back, hip or groin pain with leg weakness that is more prominent than numbness or paresthesias. Conversely, radiation plexopathy presents with weakness and paresthesias of the limb and less prominent pain.13

**Radiculopathies**

In the geriatric population, degenerative changes of intervertebral discs and joints
Paresthesias

make cervical and lumbosacral radiculopathies very common causes of sensory disturbances (Figure 2). Neck or lower back pain with radiation to the limb is the dominant complaint. Both NCS/EMG and spinal MRI are helpful in evaluation. In the neck, the C7 root is frequently affected, presenting with numbness and paresthesias of the middle and index fingers, weakness of elbow and wrist extension, and diminution of the triceps reflex. The C6 root, also commonly affected, causes sensory disturbances in the lateral forearm, thumb and index finger (sometimes mimicking carpal tunnel syndrome) and diminution of the biceps reflex. C8 involvement can produce sensory loss or paresthesias of the medial forearm and fourth and fifth digits and weakness of hand muscles (mimicking ulnar neuropathy).14

In the lower back, L5 involvement results in numbness and paresthesias of the lateral leg and dorsum of the foot and great toe with weakness of ankle dorsiflexion. S1 involvement produces sensory disturbances of the sole and lateral foot with gastrocnemius, hamstring and gluteal weakness and loss of the ankle jerk. When the L4 root is affected, the medial leg will show sensory involvement and there may be quadriceps weakness and loss of the knee jerk.15 Degenerative spine disease in the elderly often affects two or more root levels. In fact, chronic, lumbosacral polyradiculopat-
Paresthesias

Polyneuropathy - symmetric “stocking-glove” numbness
  - distal weakness
  - distal hyporeflexia

large fibre
small fibre
- loss of vibration and proprioception sensation
- loss of temperature and pain sensation

Focal Neuropathy
- numbness and weakness in the distribution of a peripheral nerve
  - usually asymmetric
  - pain or paresthesias elicited by palpation or percussion of nerve (e.g., “Tinel sign”)

Plexopathy
- numbness and weakness of a limb outside of peripheral nerve or root distribution
  - limb girdle pain often prominent

Radiculopathy
- numbness in a dermatomal distribution
  - weakness in a myotomal distribution
  - loss of deep tendon reflex served by nerve root

Myelopathy
- classically produces sensory loss below level of spinal cord disease
  - may produce glove or stocking numbness
  - hyperreflexia, spasticity, Babinski response

lopathies from spinal stenosis can present with distal symmetric sensory loss. This may be very difficult to distinguish from polyneuropathy.

Another common radiculopathy in older adults results from herpes zoster. “Shingles” most frequently produces pain in a thoracic dermatome that preceeds the vesicular eruption, followed by post-herpetic neuralgia in up to 50% of adults older than 60 years.16

One potential neurosurgical emergency that can present with sensory loss is the cauda equina syndrome. This occurs with a lower lumbosacral central disc herniation that compresses the sacral nerve roots and results in pain and paresthesias in the perineal region and “saddle anesthesia”. The accompanying bowel and bladder dysfunction and leg weakness may be irreversible if this is not recognised and treated immediately.17

Central Nervous System

In addition to producing the radiculopathies already mentioned, degenerative changes in the cervical spine can cause central cervical stenosis, resulting in a myelopathy. This can present with distal sensory loss in a glove or stocking distribution mimicking polyneuropathy or even carpal tunnel syndrome. Hyperreflexia and other upper motor neuron signs (spasticity, Babinski response) with leg weakness or fatigability should raise the suspicion of cervical stenosis and prompt cervical spine imaging.18

Complaints of sensory disturbance over one-half of the face and body localise to the brain. Thalamic lesions, such as strokes and tumours, may lead to impaired sensation of all modalities on the contralateral side of the body with accompanying spontaneous pain and dysesthesias. Cortical or subcortical lesions, usually from strokes, generally produce numbness on the contralateral face and body, rather than paresthesias or pain. Often, even patients with a pure motor deficit from a stroke will complain of subjective numbness when no objective sensory loss can be found. Transient and recurrent numbness raises the possibility of transient ischemic attacks (TIAs), whereas recurrent paresthesias may rarely be caused by focal seizures.

Conclusion

The evaluation of sensory disturbances in the elderly begins with careful delineation of the area of numbness or paresthesias. When combined with motor and reflex involvement, patterns will emerge that enable the clinician to localise the dysfunction in the peripheral or central nervous system. An approach can then be tailored for the further evaluation of these common and often diagnostically challenging complaints.

References


