Neurological Exam and Neurophysiologic Evaluation for the Pain Patient

9

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Key Points

- The neurophysiological tests of nerve conduction studies (NCS) and electromyogram (EMG) are considered an extension of the neurological exam.
- NCS measure thickly myelinated Aβ fibers of the somatic motor and sensory nerves but not the nociceptive Aδ and C fibers. Thus, NCS is expected to be normal in primarily painful small fiber neuropathy.
- Axonal injury NCS features include significantly reduced amplitude with no more than mild slowing of conduction velocity.
- Demyelinating lesions NCS features include prolonged distal latency, significant slowing of conduction velocity, and conduction block.
- A single-isolated radiculopathy generally does not result in severe or dense numbness due to overlapping dermatomes.
- Sensory NCS are typically normal in radiculopathy because the lesion is proximal to the dorsal root ganglion.
- Motor NCS and EMG are abnormal and demonstrate neuropathic pattern of injury in radiculopathy.
- EMG findings of "acute denervation," including positive sharp waves and fibrillation potentials, can take several weeks to develop on account of time necessary for Wallerian degeneration to occur.
- NCS and needle EMG cannot diagnose discogenic pain and facetogenic pain and are usually unhelpful in the diagnosis of spinal stenosis.

Case Presentation

A 62-year-old man presents with 6 weeks of lower back pain. He reports the pain radiates from his lower back down his left buttock, to the left lateral thigh and calf, and into the dorsum of his left foot. He shares that he has trouble walking and has fallen a few times due to ankle weakness. The neurological exam demonstrates mild sensory impairment in the left L5 dermatome with light touch, temperature, and vibration. He has clinical weakness with ankle dorsiflexion and eversion. A nerve conduction study/electromyography (NCS/EMG) study demonstrates normal compound muscle action potentials (CMAPs) and sensory nerve action potentials (SNAPs). EMG shows positive sharp waves and fibrillation potentials. The motor unit action potential (MUAP) morphology is normal, but reduced recruitment is recorded. Clinically and neurophysiologically, he presents with subacute L5 radiculopathy with active denervation.

How can this precise neurological assessment, both by examination and neurodiagnostic studies, inform the pain physician? First, the tests can help establish a diagnosis, providing valuable information to the patient and practitioner alike. Second, the test can help guide treatment and interventional procedures. Third, the results can help with prognosis and monitoring clinical progression, potentially helping inform decisions on when patients may be most likely to benefit from surgery. This chapter will help the reader become familiar with the performance and interpretation of neurological evaluation of the pain physician using clinical examination and neurodiagnostic testing.

The basic approach of the neurological examination is first to identify the pathology and second to determine the etiology. Neurophysiologic tests complement the physical examination by providing more objective, quantitative functional data and depending much less on patient effort. Primarily, the most utilized electrophysiologic tests are nerve conduction studies (NCS) and needle electromyography (EMG). NCS/EMG interrogate the peripheral nervous system and can be useful in distinguishing radiculopathy, plexopathy,

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mononeuropathies, and polyneuropathies. Painful small fiber neuropathies can be investigated with quantitative sensory testing (QST) and quantitative sudomotor axon reflex testing (QSART) [1–3]. Additional neurophysiologic tests include somatosensory evoked potentials (SSEP) [4] and magnetoencephalography (MEG) [5], which record cortical responses to peripheral stimulation. This chapter will focus on how the neurological examination and NCS/EMG together serve to localize and determine the pathology.

Neurological Exam

How does a practitioner efficiently but comprehensively evaluate the broad spatial localizations and etiologies of neurological dysfunction and pain? One answer is that a thorough history and screening exam will help identify other symptoms and signs that suggest a particular anatomical location. For example, in the case of a left foot drop, one would pay particular close attention to relevant cortical signs that would implicate the right (non-dominant in the majority of people) cortical hemisphere. One would focus on difficulty recognizing numbers drawn on the hand (agraphesthesia), impaired two-point discrimination, or problems identifying objects by feel (astereognosia). On the cranial nerve exam, one would emphasize the assessment of the left visual field and left facial movements. On the motor exam, in addition to upper motor neuron signs, one would focus on signs of subtle weakness in the left upper extremity (pronator drift and rapid finger movements). Any such identified abnormalities would strongly suggest a cortical or subcortical location of pathology, as opposed to the frequently seen and even more frequently presumed radiculopathy. Because always anticipating in advance what particular locations one should focus on can be difficult, particularly in the real-time environment of a clinical visit and examination, one should instead perform a "screening exam." Such an exam should be sufficiently broad so as to identify abnormalities that would help both identify and localize particular pathologies. At the same time, the exam must be brief enough that it can be completed within the appropriate timeframe of a typical outpatient encounter.

Furthermore, a thorough screening exam will help identify a group of symptoms that when identified initially are not specific with regard to etiology but when identified as a group can point strongly toward a particular diagnosis. For example, both ataxia and neuropathy can be due to a wide range of causes. However, the combination of the two may suggest a specific etiology, such as in this case, Friedreich's ataxia.

While a reliable screening exam is an essential tool, often further meticulous detail is required. Clinicians should use the chief complaint and history to generate a hypothesis that can be tested during the examination. As appropriate for evaluating the hypothesis, the examiner may delve into certain components of the examination in more detail. For example, in evaluating a patient with wrist drop (which when due to peripheral injury often localizes to C6 or the radial nerve), one should investigate a C6 muscle that is not innervated by the radial nerve, such as the pronator teres (C6 but innervated by the median nerve), even though routine assessment of that muscle is not included in most screening examinations. Indeed, this expansive assessment of myotomes, components of the brachial and lumbar plexi, and individual nerve roots embodies the strategy used by neuromuscular physicians during the NCS/EMG evaluation.

One should pay particular emphasis to the identification of "upper motor neuron signs" versus "lower motor neuron signs." Distinguishing the two can be of critical importance in the practice of pain medicine. Upper motor neuron injury can masquerade as many of the common lumbar pain syndromes. As a general rule, spine injury thought to occur at a particular level, for example, pain involving the front of the thighs (L2 distribution), can be due to injury at any more cephalad level. One should pay particular attention to cervical spine injury mimicking pain thought to be due to a more caudal injury.

Mental Status Exam

Undoubtedly the most complex component of the neurological examination is the mental status examination, and a screening exam checks only the most superficial components. The basic components of the mental status exam include the following assessments: level of arousal, attention, orientation, language, memory, integrative sensory function, and integrative motor function.

A number of terms are used, often ambiguously, to define level of arousal. What one means by "sleepy, somnolent, obtunded" is often unclear and varies from physician to physician. Instead, it can be more helpful to simply describe what one sees with regard to whether the eyes are closed or open and what stimulus is necessary for a patient to open the eyes. For example, an ICU level exam might include "Eyes closed. Patient does not open eyes to verbal stimulus but opens eyes to gentle movement of the arms." Attention refers to maintained arousal. It can easily be assessed by asking a patient to count backward from 20 to 1 or in a patient with a high level of education by subtracting serial 7s from 100. As delirium is a common feature of pain patients, particular in the setting of over-medication, testing attention is an important component of the exam. Delirious patients can seem surprisingly normal, in that they can speak fluently and interact appropriately, but simple tests of attention can expose the

deficit. Appearance, mood, and affect can be important to consider in assessing how a patient's psychological state may modulate pain perception.

Language is most commonly assessed based on categorization of aphasias such as those due to receptive and expressive language areas in the brain. Such classification depends on assessing comprehension, naming, fluency, and repetition. Typically, reading and writing are checked as well. Immediate registration, short-term memory, and long-term memory can be evaluated by individual questions. However, often simple questions, such as "what did you eat for dinner last night" or "have we met before," can provide similar information.

Appearance/mood/affect and thought content, both components of the psychiatric mental status examination, are important to include in the assessment of pain patients. Identifying features of depression can alert a provider to potential affective enhancement of pain.

Integrative sensory functions focus on non-dominant parietal signs such as neglect of a particular visual or sensory region, graphesthesia (recognizing a number drawn on the hand), astereognosia (identifying an object by feel alone), two-point discrimination, somatognosia (inability to recognize that a body part is self), and anosognosia (lack of awareness of disease or disability). The most common integrative motor functions include apraxias, which refer to sequences of movements for which all the components are themselves performable but not the entire ensemble. Examples of apraxias include demonstrating how to comb hair or salute.

Cranial Nerve Exam

Although the cranial nerve exam, like the mental status assessment, is often not the focus of a pain practitioner's neurological exam, it is nonetheless critically important. The first cranial nerve, the olfactory nerve, is not routinely assessed. The second cranial nerve, the optic nerve, is evaluated in several ways. First, through fundoscopy, the practitioner visualizes the optic nerve head and can identify pathological processes such as atrophy or papilledema. Second, the pupillary light response assesses afferent fibers in the optic nerve (as well as connections from the pretectal nucleus to the Edinger-Westphal nucleus, which contains the parasympathetic efferent fibers for the reflex). Third, one tests visual fields via confrontational testing as well as visual acuity.

The extraocular muscles are innervated by the oculomotor (III), trochlear (IV), and abducens (VI) nuclei. One should assess the lids and pupils, looking for components of the Horner's syndrome, namely, ptosis, miosis, and anhidrosis. In addition to a Pancoast tumor that affects the apices of the lungs, one should remember that Horner's syndrome can be caused by carotid dissection (which causes neck pain that radiates to the jaw) or a mass lesion involving the cavernous sinus.

The trigeminal nerve (V) provides sensory innervation from the face as well as motor innervation to the muscles of mastication. The facial nerve (VII) innervates the muscles of facial expression. The motor neurons innervating muscles above the forehead generally receive bilateral innervation from the primary motor cortex, so that a peripheral VIIth nerve lesion will affect all the unilateral muscles of facial expression but a central lesion will spare the muscles above the eye. The facial nerve also conveys taste sensation from the anterior tongue, explaining why taste is affected in a Bell's palsy. Finally, the facial nerve mediates secretion from the salivary glands (except the parotid) and the lacrimal gland.

One can coarsely test the vestibulocochlear nerve (VIII) using finger rub. The glossopharyngeal nerve (IV) and vagus nerve (X) serve a number of functions including taste and general sensory inputs from the posterior tongue, pharynx, swallowing, afferent visceral inputs, and parasympathetic innervation to the body. The spinal accessory nerve (XI) provides partial innervation to the sternocleidomastoid and trapezius muscles. The hypoglossal nerve (XII) is responsible for motor innervation to most of the tongue muscles. It too is important for swallowing.

Motor Exam

The motor exam is of primary importance in the assessment of pain patients. The first component of the motor exam is an assessment of the bulk and tone. It is important to observe the patient, often in different positions. For example, scapular winging can be brought out by leaning against a wall with shoulders abducted and the elbows flexed.

In observing the muscles, one also is in a position to identify abnormal movements such as fasciculations, myoclonus (sudden, short contractions), asterixis (sudden, short relaxations), and tremor.

Movements should be assessed as much as by isolating particular joints for investigation. For example, when checking a patient's wrist extensors, one should anchor the wrist with one hand and assess the power of the wrist extensors with the second hand. By stabilizing the wrist, one ensures that only the wrist extensors are used in generating the power and not, for example, more powerful proximal muscles such as the brachioradialis or biceps. Additionally, one must test individual muscles against one's own muscles of comparable strength. For example, one cannot identify subtle weakness in a patient's tibialis anterior when using one's finger flexors.

Power is graded from 0 (no movement) to 5 (full strength). 1/5 represents trace movements, 2/5 movement in a sup-

ported plane, 3/5 movement against gravity, 4−/5 movement against minimal resistant, 4/5 movement against resistance, and 4+/5 trace weakness. One should realize that 4/5 represents an enormous range, from very weak to very strong. Precise and accurate grading of power is important for ensuring agreement among different practitioners and for assessing changes over multiple assessments. Such information is often critical, for example, the urgency for a decompressive surgery is much higher when weakness is progressive as opposed to static.

When evaluating individual muscles, one should keep the innervation of the muscles in mind. On a screening exam, one should not only evaluate muscles innervated by most limb-supplying spinal levels but also compare muscles innervated by the same spinal level but supplied by different nerves. For example, identifying weakness in the abductor pollicis brevis (APB) (a C8/T1 muscle supplied by the median nerve) but preserved strength in the abductor digiti minimi (a C8/T1 muscle supplied by the ulnar nerve) might suggest carpal tunnel syndrome as an explanation.

When assessing power, one should keep in mind that rapid alterations in level of power are usually due either to voluntary changes in effort or to pain. Such "give-way" weakness often results in physicians' "over-calling" weakness. That is, a patient with pain radiating down the leg has pain on straightening the leg. One should focus on the maximum power generated, even if for a very brief period of time (a second or less). Often, encouraging a patient to produce maximum power can be helpful or necessary. However, equally often pain limits full assessment, and one must document this, for example, "pain in the left hip flexor at least 4/5 but full assessment limited by pain."

As mentioned previously, distinguishing upper and lower motor neuron signs is of principal importance in making a correct assessment and pursuing the appropriate diagnostic and treatment strategies. Signs of upper motor neuron injury include preserved bulk with increased tone and hyperreflexia. Additionally, certain patterns of weakness are characteristic of upper motor neuron injury. In the upper extremities, upper motor neuron lesions cause weakness in the extensor muscles out of proportion to weakness in the flexor muscles; in the lower extremities, flexor muscles (including foot dorsiflexion) are more strongly affected. In contrast, lower motor neuron signs include decreased muscle bulk with decreased tone and hyporeflexia. Additionally, fasciculations may be seen in the muscles.

See Table 9.1 for a list of frequently assessed muscles.

Table 9.1 Frequently assessed muscles

Upper extremities
Shoulder abduction: axillary nerve to deltoid C5 (C6), suprascapular nerve to supraspinatus C5 (C6) mediates first 15° of abduction
Elbow flexion: musculocutaneous nerve to bicep C5 (C6)
Wrist extension: radial nerve to extensor carpi radialis longus C6 (C5)
Elbow extension: radial nerve to triceps C7 (C6, C8)
Forearm pronation: median nerve to pronator teres C6–C7
Extension at metacarpophalangeal joint: radial nerve (posterior interosseous branch) to extensor digitorum C7 (C8)
Finger flexion: median nerve to flexor digitorum superficialis C8 (C7, T1) flexes at PIP joints, digitorum profundus (digits 2–3) C8 (C7, T1) (anterior interosseous branch of median nerve) flexes at DIP joints Ulnar nerve to flexor digitorum profundus (digits 4–5) C8 (C7,T1)
5th digit abduction: ulnar nerve to abductor digiti minimi C8, T1
2nd digit abduction: ulnar nerve to first dorsal interosseous T1 (C8)
Thumb abduction: median nerve to abductor pollicis brevis T1 (C8)
Lower extremities
Hip flexion: spinal nerves and femoral nerve to illiopsoas L1–L2 (L3)
Knee extension: femoral nerve to quadriceps L3–L4 (L2)
Dorsiflexion: deep peroneal nerve to tibialis anterior L4 (L5), deep peroneal nerve to extensor hallucis longus L5 (S1)
Plantarflexion: tibial nerve to gastrocnemius S1–S2
Knee flexion: sciatic nerve to hamstrings S1 (L5, S2)
Foot inversion: tibial nerve to tibialis posterior L4–L5
Foot eversion: superficial peroneal nerve to peroneus longus and brevis L5, S1
Hip adductors: obturator nerve L2–L3 (4)
Hip abduction: superior gluteal nerve to gluteus medius and minimus and tensor fasciae latae L4–L5 (S1)
Hip extension: inferior gluteal nerve to gluteus maximus L5, S1 (S2)

Reflexes

Reflexes are a critical and often overlooked component of the neurological exam. Increased reflexes can help identify a "central" etiology, whereas loss of reflexes can help localize to a particular root level or lower motor neuron injury. One should always consider metabolic effects on reflexes such as in diseases that affect levels of calcium or thyroid hormones.

There is less consistency in the grading of reflexes than in the grading of power. One system often used by neuromuscular neurologists is the following:

- 0/4: absent.
- 1/4: obtainable with distraction (such as the Jendrassik maneuver, when the patient is asked to interlace his finger tips and then pull from both sides simultaneously).
- 2−/4: less than normal but obtainable without distraction.
- 2/4: normal.
- 2+/4: brisker than normal but without spreading.
- 3/4: hyperreflexic in that there is spreading from one reflex to a nearby one. For example, stimulation at the bicep causes finger flexion. Alternatively, the presence of crossed adductor reflexes would warrant 3/4.
- 4/4: clonus.

One should keep in mind that reflexes tend to be much more brisk in younger patients than in older ones. While not

strictly considered in the grading, such information must be considered in the interpretation of the findings.

Babinski sign (dorsiflexion of the great toe upon stroking of the plantar lateral foot) typically indicates injury to the corticospinal tract. Hoffman's sign (flexion of the thumb and index finger upon flicking of the distal third digit) indicates hyperreflexia due to a process at the level of the cervical spinal cord or more cephalad.

Sensory

When assessing the sensory system, one should consider what sensory fibers are being assessed. Pinprick, heat, or cold assess nociceptors, the unmyelinated C fibers and thinly myelinated Aδ fibers. Vibration sense and proprioception assess the thickly myelinated Aβ fibers. One should keep in mind the anatomy: the first group of fibers (for nociception) synapse in the ipsilateral dorsal horn, and the second order neurons cross and then ascend contralaterally in the lateral spinothalamic tract; in contrast, the second group of fibers (for vibration and proprioception) ascend ipsilaterally in the dorsal columns and then synapse and cross in the lower brainstem.

One should remember that common peripheral sensory processes can involve particular fiber types. Furthermore, one should look for common patterns of involvement, including the "stocking-glove" pattern of distal symmetric neuropathy, single nerve or root involvement, or mononeuropathy multiplex patterns. As a general rule, because of overlap of sensory dermatomes, complete sensory loss is not expected after a single-level radiculopathy. In contrast, injury to a peripheral nerve would cause complete or near complete sensory loss. For example, injury to the deep peroneal nerve produces more profound sensory loss in the web between the first and second toes than an L5 radiculopathy.

Common signs associated with neuropathic pain should be documented. These include the following: allodynia, pain in response to a stimulus that normally is innocuous; hyperalgesia, increased pain response to a noxious stimulus; summation, increased pain response to repeated innocuous or mildly painful stimulus; paresthesias, abnormal positive sensory phenomena, such as a pins and needles sensation; and dysesthesias, painful paresthesias.

Coordination

Coordination is assessed on finger-nose and heel-shin testing as well as assessment of the truncal posture. Cerebellar injury commonly produces a coarse horizontal wavering present on the entire motion arc between the finger and the nose (dysmetria). In contrast, essential tremor causes higher frequency deviation predominantly at the beginning and end of the trajectory.

Stance and Gait

High-level functions such as stance and gait are particularly important and should be assessed during every visit. The basic characterization of stance should be whether the feet are normally spaced or broadly spaced. Gait can be described as fluid or rigid, with attention to the arm swing and the smoothness of turns. Posture should be noted as normal, or stooped. Walking on the toes and heels assesses the foot dorsiflexors and plantarflexors in a functional manner (and is more sensitive for identifying subtle weakness than confrontational testing). One should assess cerebellar functioning by asking the patient to walk heel to toe "on a tightrope."

A number of terms are used in describing the gate abnormalities due to particular etiologies. For example, a wide-based or "drunken" gait can be associated with cerebellar pathology. A "shuffling" gait is typically due to parkinsonism, whereas a "magnetic" gate – in which the feet do not leave the floor – is associated with normal pressure hydrocephalus. An "antalgic" gait refers to abnormalities due to pain.

One should always assess for Romberg's sign, which primarily reflects interference with proprioceptive signaling. To test for Romberg's sign, ask the patient to stand with the feet together and the eyes open, focused on a distant target. Romberg's sign refers to impairment in balance when visual inputs are removed (by closing the eyes), in a patient who can stand with the feet together when the eyes are opened. Importantly, if a patient cannot stand with the feet together while the eyes are open, this should not be referred to as a positive Romberg sign; rather, it reflects a problem in, for example, the vestibular or cerebellar functioning.

Putting Together an Assessment

In the assessment, one should review key elements of the history and clinical findings. One often depends on combinations of the different components of the exam to help establish as precise as possible the localization of disease, such as in the motor and reflex components of lower motor neuron signs. Based on the localization, one should be familiar with pathological processes that occur at particular locations. For example, with the presenting symptom of foot drop, the lesion could localize to the CNS at the contralateral cortical gray matter, subcortical white matter, brainstem, or ipsilateral spinal cord. However, it could also be an initial presentation of motor neuron disease affecting the anterior horn cells; the foot drop could result from an L4 or L5 radiculopathy, lumbar plexopathy, peroneal neuropathy, disease of the neuromuscular junction (NMJ), or a myopathy. The practitioner can then determine what laboratory, imaging, or neurophysiologic studies might help further distinguish among the proposed possible diagnoses.

NCS/EMG

Nerve conduction study (NCS) and electromyography (EMG) are neurophysiologic studies that evaluate the peripheral nervous system. NCS/EMG acts as an extension of the neurological exam by providing electrophysiologic data of the nerves and muscles. NCS is performed by electrically stimulating a targeted nerve (sensory or motor) and recording the resultant action potential response. These action potentials are conducted through thickly myelinated Aβ fibers of the somatic motor and sensory nerves. Patients will feel a sharp electrical sensation, but the nociceptive Aδ and C fibers are not recorded. EMG is performed by placing a recording needle into the targeted muscle belly and recording the electrical activity. Although any distal peripheral nerve and muscle can theoretically be tested, the approach to NCS/EMG is reliant on the patient's history and clinical exam. By evaluating individual nerves and muscles via NCS/EMG, the clinician can localize the pathology to the level of the nerve root, plexus, peripheral nerve, neuromuscular junction, or muscle. Furthermore, specific electrophysiologic patterns can also distinguish the degree of injury as well as the underlying pathology (e.g., axonal loss vs demyelinating disorders). This is especially helpful in cases where the clinical exam is limited by pain or volition. We will review the terminology required for interpretation of NCS/EMG studies.

Motor Nerve Conduction Study

Motor NCS is performed by stimulating a motor nerve and then recording with an electrode placed over the motor nerve innervated muscle belly. The resultant potential is called the compound muscle action potential (CMAP). The CMAP represents the combined potentials of all underlying muscle fiber action. Important CMAP components include amplitude, duration, latency, and conduction velocity (Fig. 9.1).

CMAP amplitude is directly correlated with the number of muscle fibers that are activated. CMAP duration represents synchrony of individual muscle fibers firing. Latency describes the time between stimulation of targeted nerve and onset of the fastest muscle action potentials. This includes time from stimulus to neuromuscular junction (NMJ), NMJ activation, and depolarization time across muscle. Conduction velocities are defined as the speed of the fastest action potentials [6].

Sensory Nerve Conduction Study

Sensory nerve action potentials (SNAP) are generated by stimulating a sensory nerve and recording the cutaneous response with two recording electrodes. SNAP characteristics include onset latency, peak latency, amplitude, duration, and conduction velocity (Fig. 9.2).

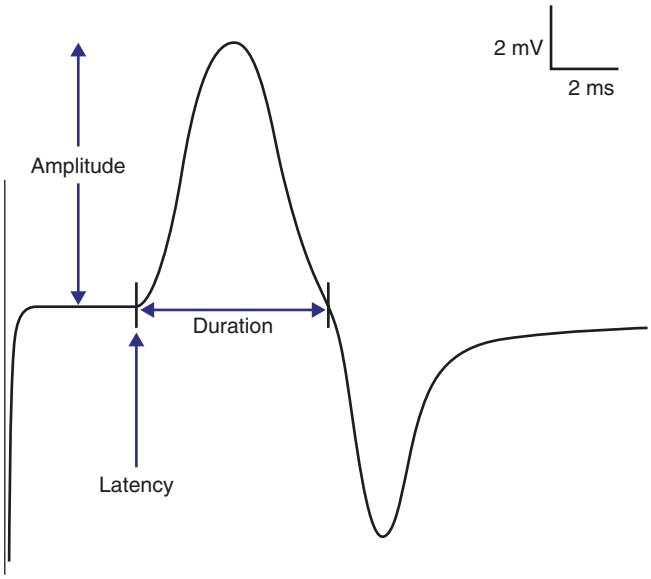

Fig. 9.1 CMAP waveform. Amplitude represents the summation of muscle fibers activated and is calculated from baseline to peak of waveform in millivolts. Latency is the combined time from nerve stimulation, conduction of motor nerve, depolarization across NMJ, and depolarization across muscle. Latency is measured from time of stimulation to initial deflection from baseline. Duration of the waveform represents synchrony of individual muscle fibers firing and is measured from initial deflection to return to baseline. (Reprinted with permission from Preston and Shapiro [6])

Amplitude represents the total depolarization of all individual sensory nerve fibers. Onset latency represents time from stimulus to initial deflection from baseline; this typically represents nerve conduction time from the largest heavily myelinated cutaneous sensory fibers as these are the fastest fibers [7]. Peak latency is the time to peak amplitude on the SNAP waveform. Conduction velocity is the speed of the fastest action potential between the stimulator and recording electrode. Again, an important point related to the use of neurodiagnostic tests in pain medicine is that SNAPs do not reflect the activity of nociceptors, the unmyelinated C fibers and thinly myelinated Aδ fibers. The action potentials from these fibers are too small and too temporally dispersed to contribute to the SNAP amplitude. Thus, SNAPs are typically affected by a "large fiber" neuropathy but not by a "small fiber" neuropathy.

NCS F Response and H Reflex

NCS F response and H reflex are specialized NCS tests for evaluation of the proximal nerve segments including the nerve roots and plexus. Late responses are obtained by stim-

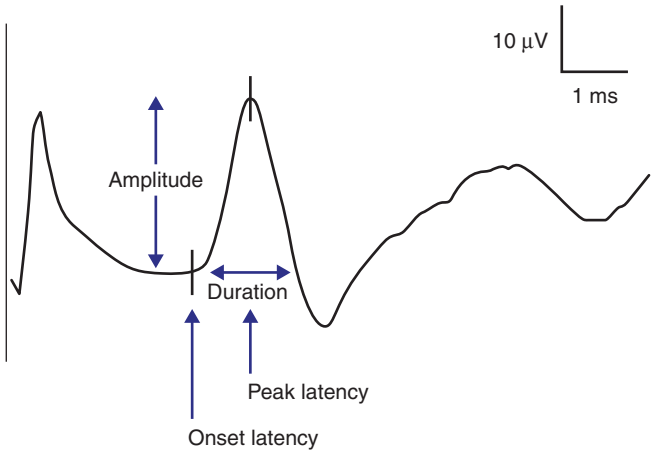

Fig. 9.2 SNAP waveform. Amplitude represents the total depolarization of all individual sensory nerve fibers which is measured in microvolts. Onset latency represents time from stimulus to initial deflection from baseline; this typically represents nerve conduction time of the largest heavily myelinated cutaneous sensory fibers as these are the fastest fibers. Peak latency is the time to peak amplitude on the SNAP waveform. Duration represents the synchrony of action potentials and is measured from initial deflection to return to baseline. (Reprinted with permission from Preston and Shapiro [6])

ulation of the nerve and allowing for antidromic conduction (afferent) toward the spinal cord and recording the following orthodromic conduction (efferent) response toward the axon terminals.

The F response is a late motor CMAP which is obtained by supramaximal depolarization of a motor nerve, allowing for antidromic nerve conduction up to the anterior horn cell followed by orthodromic response down to the recorded muscle. The F response is usually small in amplitude, representing 1–5% of muscle fibers. Of note, the F responses are purely motor and provide no information regarding lesions that only affect sensory nerve fibers. If distal nerve conduction studies are abnormal, a prolonged F response could then be suggestive of proximal neuropathy, plexopathy, or radiculopathy. Unfortunately, utility of diagnostic F response is limited by targetable nerves. In the upper extremities, supramaximal stimulation of the median and ulnar nerve can evaluate F responses in C8–T1. In the lower extremities, supramaximal stimulation of the peroneal and tibial nerves can evaluate F responses in L5–S1.

The H reflex is a late response like the F response. The H reflex differs in that it is a true reflex involving stimulation of sensory afferent fibers, a synapse at the anterior horn cells, followed by efferent motor fibers. The H reflex can only be reliably obtained by stimulating the tibial nerve in the popliteal fossa with an expected response in the gastrocnemius-soleus muscle. It is an NCS correlate to the physical exam's ankle reflex. Therefore, the H reflex will be prolonged in S1 radiculopathy, lumbosacral plexopathy, tibial and sciatic neuropathy, and polyneuropathy. The H reflex is a sensitive early electrodiagnostic test for Guillain-Barre syndrome (Fig. 9.3) [8].

Needle Electromyography (EMG)

The needle EMG study is performed by inserting a recording needle into the target muscle to measure electric potentials of the muscle at rest and during activation. The electrical potentials of the muscle at rest are described as spontaneous activity, and the electrical potentials of voluntary muscle activation are called motor unit action potentials (MUAP). Almost all skeletal muscles can be interrogated; however a priori muscle testing is recommended due to limited tolerance for this invasive test. For example, in suspected L5 radiculopathy with clinical history of back pain and radiating paresthesias to the posterior lateral calf extending to dorsum of foot with concomitant weakness in ankle dorsiflexion, the EMG operator will evaluate myotomes above, below, and at the level of the expected lesion – specifically targeting lumbar paraspinal, proximal muscles (gluteus medius, vastus medius) and distal muscles (tibialis anterior, tibialis posterior, medial gastrocnemius).

Spontaneous Activity

Normal healthy muscle will not generate spontaneous activity at rest. Abnormal spontaneous activity can inform the underlying pathology. Fibrillation potentials and sharp waves represent single muscle fiber depolarization and are electrophysiologic markers of active denervation suggestive of a recent lesion. Complex repetitive discharges represent a group of denervated adjacent muscle fibers that are more typically seen in chronic myopathies than neuropathies. Myokymic discharges are rhythmic, grouped, spontaneous repetitive discharges of the same motor unit. Myokymic dis-

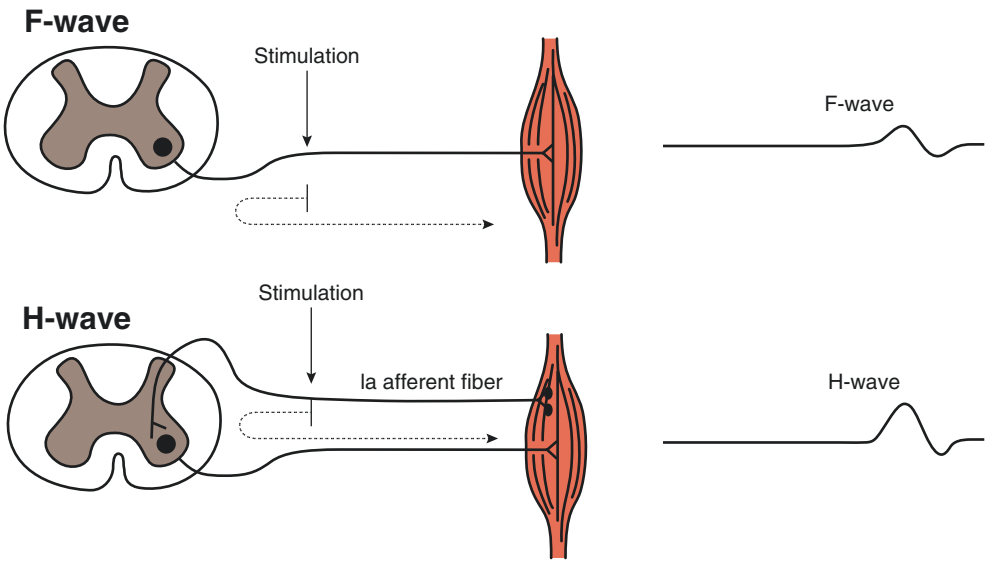

Fig. 9.3 NCS late response. Diagram of the NCS F response test and H reflex test with their resultant waveforms. F waves are produced with afferent conduction of a motor nerve to the anterior horn cell followed by efferent conduction of the same motor nerve to the recorded muscle. H waves represent afferent conduction of a sensory nerve synapsing onto the anterior horn motor neuron followed by efferent conduction of a motor nerve to the recorded muscle. (Adapted from Kai and Nakabayashi [26]. This is an open access peer-reviewed chapter InTech Open, licensed under a Creative Commons Attribution 3.0 Unported License)

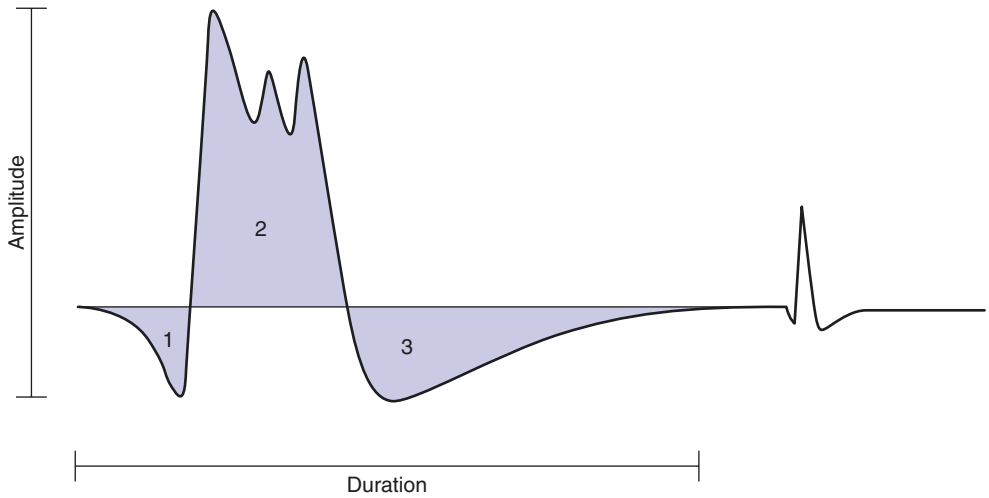

Fig. 9.4 Motor unit action potential (MUAP) measurements. Motor unit action potential amplitude is measured from peak to peak. The duration is measured from the time of initial deflection to when it returns to baseline. Polyphasia is evaluated by the number of phases above and below the baseline (triphasic in this sample) MUAP. (Reprinted with permission from Preston and Shapiro [6])

charges are features strongly suggestive of radiation-induced plexopathy or neuropathy and are rarely seen in spinal cord lesion, radiculopathy, and entrapment neuropathy.

MUAP

Motor unit action potentials (MUAP) are obtained with target muscle activation. A motor unit consists of a motor neuron and its innervated muscle fibers. Muscle strength is a function of the number of motor units and the fire rate of each individual motor unit. MUAP can be defined by two major characteristics: morphology and firing pattern. *Morphology* of MUAP varies by duration, amplitude, and phase. Duration is the length of time from the initial deflection to return to baseline. Duration reflects the number of muscle fibers within a motor unit. Amplitude is measured from lowest peak to highest peak and reflects the overall strength of the motor unit. Phase reflects the number of times the MUAP crosses the baseline, normally two to four, and reflects the synchrony of the muscle fibers firing within the motor unit (Fig. 9.4).

MUAP firing patterns function to increase muscle force through *activation* (the ability to increase the firing rate) and *recruitment* (the ability to add additional motor units). Impaired activation is typically suggestive of a central ner-

vous system lesion but can also be seen in pain limited or noncooperative exams. Reduced recruitment is suggestive of a peripheral neuropathic lesion: because additional motor units are not available, the intact units must fire at high rates to generate increased force.

Electrophysiologic Patterns of Disease

Neuropathies are disorders of the peripheral nerves of which the localization of the pathology could be attributed to the cell body (neuronopathy), axon (axonopathy), or myelin (demyelinating disorders). Clinically, neuropathies can also be defined by their time course, acute, subacute, and chronic, as well as by their primary symptoms: motor predominant, sensory predominant, or mixed. Etiologies are all-encompassing ranging from hereditary, idiopathic, autoimmune, toxic-metabolic, infectious, inflammatory, infiltrative, neoplastic, structural, to postradiation. NCS/EMG can play a pivotal role in diagnosis as pathologies produce specific electrophysiologic patterns of disease.

Axonal Injury

The electrophysiologic pattern of axonal injury is significantly reduced NCS amplitude with only mild reduction in conduction velocity. The major reduction in amplitude with relative preservation of conduction velocity is reflective of the functions of the axon vs myelin in nerve conduction. In hyperacute axonal injury, NCS can sometimes be normal if the lesion is proximal to the nerve that is being evaluated. The abnormal NCS findings are expected to develop after Wallerian degeneration (atrophy of the distal disconnected nerve), which can take days to weeks. EMG in acute axonal injury demonstrates normal spontaneous activity and reduced recruitment of MUAP, which is reflective of the loss of axons and motor units. Denervation occurs in the following weeks, and fibrillation potentials and sharp waves manifest in the subacute period of injury. Nerves have the potential for axonal repair which occurs at a rate of approximately 1 mm/day. If reinnervation is successful in chronic axonal injury, the remaining intact axons will have sprouted and connected with the denervated muscle. The former denervation potentials can resolve, and larger amplitude and polyphasic MUAPs will be recorded. However, if reinnervation is not successful, denervation potentials will persist. Of importance to the pain physician, complex regional pain syndrome type II (causalgia) – and potentially complex regional pain syndrome type I (reflex sympathetic dystrophy) – is thought to be a complication of incomplete or incorrect reinnervation. Please refer to Table 9.2 for common axonal neuropathies.

Table 9.2 Axonal loss neuropathy

Diabetes mellitus
Cryoglobulinemia
Ischemic monomelic neuropathy
Sarcoidosis
Amyloidosis
Lymphoma
Acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN)
Toxins: taxanes, colchicine, lead, alcohol

Table 9.3 Demyelinating polyneuropathy

Charcot-Marie-Tooth
Hereditary neuropathy with liability to pressure palsies (HNPP)
Krabbe disease
Metachromatic leukodystrophy
Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)
Multifocal motor neuropathy
Multifocal acquired demyelinating sensory and motor neuropathy
Distal acquired demyelinating symmetric neuropathy (DADS)
Toxins (diphtheria, buckthorn, amiodarone, n-hexane, arsenic)
Human immunodeficiency virus (HIV)

Demyelination

The hallmark of demyelinating disorders in NCS is slowing of conduction velocity and delayed distal latency. Injury to myelin will impair saltatory nerve conduction producing these results. NCS amplitude is normal in mild to moderate demyelinating disease. However, amplitude can be reduced in cases where demyelination is significant enough to produce conduction block or phase dispersion and loss of signal. EMG is generally normal in demyelinating disorders with the exception of conduction block where MUAP recruitment is reduced. Of note, prolonged and severe demyelination can result in secondary axonal injury and a mixed pattern NCS/EMG. Please refer to Table 9.3 for common demyelinating disorders.

NCS/EMG in Spine Conditions and Mimics

Neck and back pain is one of the most common clinical complaints. It is estimated to be a leading cause of years lived with disability in both developing and developed countries [9].

Spondylosis of the cervical and lumbar spine refers to degenerative structural changes in the spine that can result in compression of the nerve root (radiculopathy) and compression of the spinal cord (myelopathy). Nonspondylotic causes are many including infectious, autoimmune, infiltrating/tumors, ischemic, and toxic-metabolic. Presentation can be variable and involve pain, sensory changes, and motor weakness. The

neurological exam in conjunction with electrophysiologic testing can localize these lesions and provide information in regard to pathophysiology, severity, and chronicity.

Radiculopathy

The clinical presentation of radiculopathy is of pain, paresthesias, and muscle weakness of the associated nerve root. Cutaneous sensory innervation of the nerve root is defined as a dermatome. Anatomically, this is innervated by the dorsal root ganglion at each root level. Muscle innervation of the nerve root is known as a myotome. Anatomically, each myotome is innervated by lower motor neurons in the anterior horn of the spinal cord. The clinical and electrophysiologic diagnosis of radiculopathy relies heavily on the examiner's knowledge of root level dermatome and myotome innervation. Notably, a single root radiculopathy rarely presents as dense numbness or severe weakness as there is overlap in sensory innervation and most skeletal muscles are innervated by more than one nerve root. After a comprehensive neurological exam is performed, the suspected root levels can then be interrogated with NCS/EMG (Fig. 9.5).

In a case of cervical spondylosis resulting in C6 radiculopathy, a patient classically can present with the following clinical syndrome: pain and neck tightness from paraspinal muscle spasm, pain and numbness radiating down the lateral aspect of his arm in the C6 dermatome, and weakness with shoulder abduction, elbow flexion, and elbow pronation in addition to a wrist drop. Physical exam maneuvers such as ipsilateral neck rotation, extension, and downward pressure on the head (Spurling test) may exacerbate his symptoms. Reflexes in radiculopathy are expected to be abnormal and reduced. The brachioradialis and potentially biceps reflexes are likely diminished in isolated C6 radiculopathy. Triceps reflex should be preserved.

NCS/EMG in Radiculopathy

Sensory NCS is normal in radiculopathy because the lesion is proximal to the dorsal root ganglion. Therefore, the nerve cell

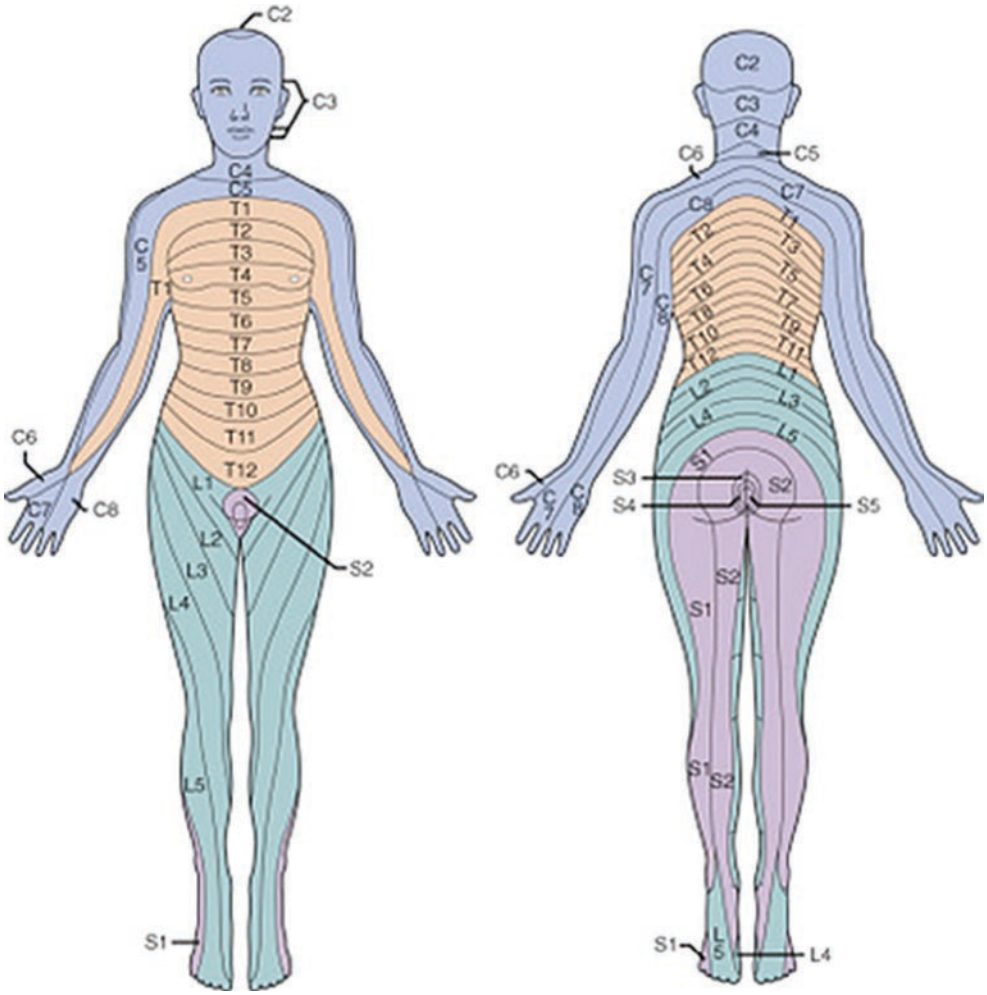

Fig. 9.5 Dermatome map. Dermatome map with anterior and posterior view. (Reproduced with permission from Strakowski et al. [27])

bodies in the dorsal root ganglion are unaffected, and the axons extending to the distal peripheral nerves are likewise normal. If SNAPs were to be abnormal, then this would generate suspicion of a lesion distal to the dorsal root ganglion such as a plexopathy, mononeuropathy, or polyneuropathy (Fig. 9.6). Motor NCS can be abnormal in radiculopathy as the lesion is distal to the anterior horn motor neurons in the spinal cord (Fig. 9.7).

Of note, motor NCS changes are directly related to the time course of injury. To elucidate, for days to weeks after a radiculopathy injury, the interrogated peripheral nerves may have normal CMAP, because the distal portion of the nerves is not yet injured. After several weeks, the motor nerve has undergone Wallerian degeneration thereby resulting in CMAPs with reduced amplitude, increased distal latency, and reduced conduction velocity. Nonetheless, because only a select few motor nerves are assessed by motor NCS, needle EMG is generally better suited for radiculopathy assessment.

Needle EMG findings in radiculopathy are consistent with neuropathic injury. We will describe the time course of radiculopathy needle EMG findings in regard to spontaneous activity, MUAP firing pattern, and MUAP morphology. In acute injury, the clinically weak muscles of the affected myotome will demonstrate reduced recruitment of MUAPs. Reduced MUAP recruitment reflects the loss of axons and motor units. However, the healthy remaining axon and motor units will continue to function with normal MUAP morphology. In the subacute phase of injury, Wallerian degeneration progresses in a proximal to distal fashion where denervation is first noted in the paraspinals (10–14 days), followed by the proximal muscles (2–3 weeks) and later in the distal muscles (5–6 weeks). During this period, abnormal spontaneous activity in the form of fibrillation potentials and sharp waves is observed. MUAP morphology continues to be normal with reduced recruitment. In chronic injury, typically after 2 months, denervated motor units will connect with surviving axons to produce larger motor units which are described as reinnervation. Reinnervation produces large amplitude, extended duration, and polyphasic MUAPs. After successful reinnervation, spontaneous activity normalizes, and fibrillation potentials and sharp waves are no longer detected. MUAP recruitment continues to remain reduced

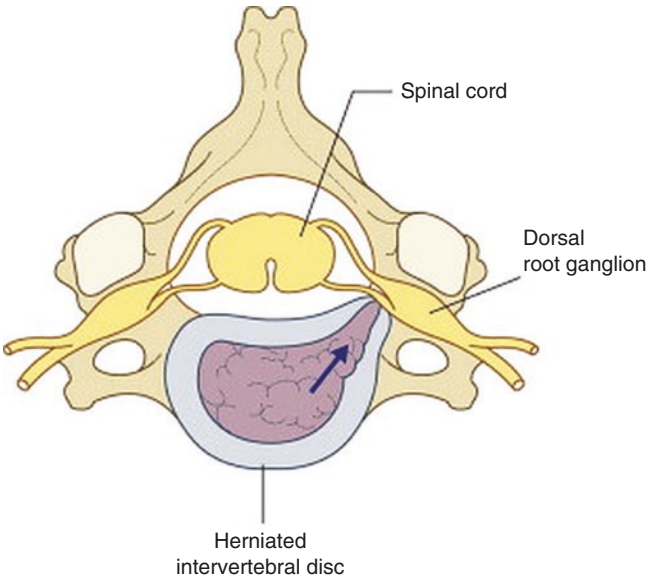

Fig. 9.6 Radiculopathy and sparing of the dorsal root ganglion. Herniated intervertebral discs can cause cervical and lumbosacral radiculopathy. Lateral posterior herniations compress the exiting spinal nerves but spare the dorsal root ganglion. Consequently, sensory NCS of peripheral nerves distal to the dorsal root ganglion are normal in radiculopathy. (Reprinted with permission from Wilbourn [28])

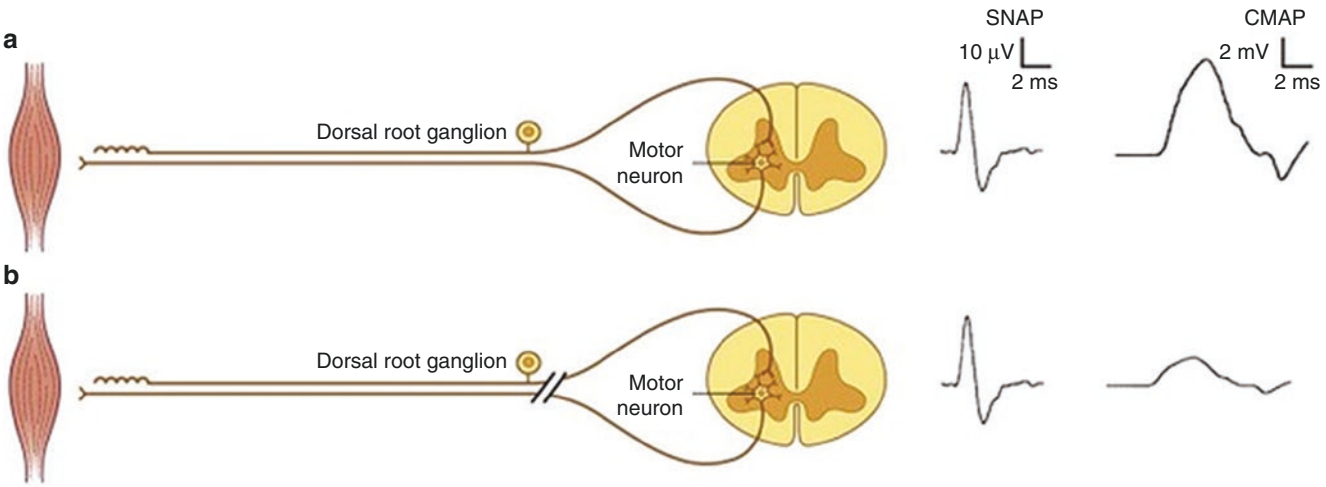

Fig. 9.7 NCS changes in radiculopathy. (**a**) Normal. (**b**) Radiculopathy. The lesion is proximal to the dorsal root ganglion. The sensory nerves distal to the dorsal root ganglion are spared. SNAPs are normal. The motor nerves will be affected. CMAPs are abnormal. (Reprinted with permission from Preston and Shapiro [6])

in chronic radiculopathy. The time course of neuropathic changes witnessed in radiculopathy allows NCS/EMG to describe the chronicity of disease [10].

Cervical Myelopathy

The clinical manifestation of myelopathy is dependent on the extent of cord involvement as well as the level of the lesion. Cervical myelopathy may present with local axial pain, cervicogenic headache, gait impairment, bladder changes, and sensory changes and motor weakness in upper and lower extremities. The neurological exam may demonstrate upper motor signs including positive Babinksi and Hoffman sign, increased muscle tone, and pathologically brisk reflexes. Lhermitte's sign, a radiating electrical shock sensation down the spine and into the extremities, can be generated by flexion of the neck. In chronic disease, there may also be lower motor neuron findings of fasciculations, atrophic muscles, reduced muscle tone, and suppressed reflexes in the setting of damaged anterior horn cells and Wallerian degeneration. "Myelopathic hands" are characterized by muscle wasting, weakness, and spastic dysfunction [11].

NCS/EMG in Myelopathy

Sensory NCS SNAPs are normal in myelopathy because the disease is proximal to the dorsal root ganglion. In chronic myelopathy affecting the lower motor neuron anterior horn cells, there will be reduced CMAP amplitude and slowed conduction velocity and distal latency. Late responses will be abnormal or absent if the affected cord levels are involved. Affected myotomes will present in the same pattern of axonal loss injury as described in radiculopathy (above). NCS/EMG cannot independently diagnose myelopathy; the diagnosis heavily relies on clinical history, physical exam, and neuroimaging. However, NCS/EMG is helpful in evaluating for peripheral nerve pathology which can mimic cervical myelopathy [12, 13].

Lumbar Spinal Stenosis

Lumbar spinal stenosis clinically presents with pain in the buttocks and groin with radiation posteriorly down the leg into the feet. Associated symptoms include lower back pain, weakness, and paresthesias. A distinguishing feature of lumbar spinal stenosis is that lumbar extension worsens symptoms whereas lumbar flexion improves symptoms. Patients may complain of pain and paresthesias with lying flat or standing with improvement of symptoms when sitting or curling up on their side with hips flexed [14]. The underlying pathophysiology is attributed to positional mechanical compression of nerve roots.

NCS/EMG in Lumbar Spinal Stenosis

In early disease, NCS/EMG can be normal due to intermittent neuroclaudication. In advanced disease, NCS/EMG findings are similar to chronic multilevel lumbosacral radiculopathy. NCS /EMG demonstrates axonal pattern of injury with abnormal paraspinal and limb muscle fibrillation potentials that corresponds to the root level of injury. It is important to note, that SNAPs are unaffected, as the lesion is proximal to the dorsal root ganglion. Late responses such as the H-reflex are typically abnormal or absent if the spinal stenosis is present at the S1 level [15–17].

Axial Pathology Mimics

Cervical Spine Disease Mimics

Brachial plexopathy and upper extremity neuropathy can mimic cervical spine disease by presenting with prominent pain, sensory loss, or motor weakness in the upper arm, forearm, and hand. We will review the upper extremity neuroanatomy and describe clinical syndromes with NCS/EMG findings.

The brachial plexus is comprised of the roots, trunks, divisions, cords, and terminal nerves. There are five roots beginning at C5 and continuing to T1. The dorsal scapular nerve arises from the C5 nerve root. The long thoracic nerve arises from the C5, C6, and C7 nerve roots. There are three trunks. The upper trunk is formed by C5 and C6 roots and gives rise to the suprascapular nerve. The middle trunk is formed by the C7 trunk. The lower trunk is formed by the C8 and T1 nerve roots. Each trunk has an anterior and posterior division. The lateral cord is formed by the anterior divisions of upper and middle trunk. The posterior cord is formed by the posterior division of all three (upper, middle, and lower) trunks. The medial cord is formed by the anterior divisions of the lower trunk. The lateral cord gives rise to the lateral pectoral and musculocutaneous nerves. The lateral cord also innervates the median nerve with contribution from the medial cord. The medial cord itself gives rise to the medial pectoral, medial brachial cutaneous, medial antebrachial cutaneous, as well as the ulnar nerve. The posterior cord branches off into the axillary nerve, radial nerve, subscapular nerve, and thoracodorsal nerve (Fig. 9.8). Cutaneous innervation by the brachial plexus is described in Fig. 9.9. Distinguishing dermatomal distribution of symptoms versus a peripheral nerve distribution can be instrumental in diagnosis.

Presentation of brachial plexopathy and upper extremity neuropathy varies from acute to insidious. Etiologies are many including traumatic traction, shearing and compression injuries, neoplastic infiltration, mass lesions, ischemic, brachial plexitis (Parsonage-Turner syndrome), and thoracic outlet syndrome. Iatrogenic causes include delayed postradiation injury and perioperative stretch injuries which typically occur

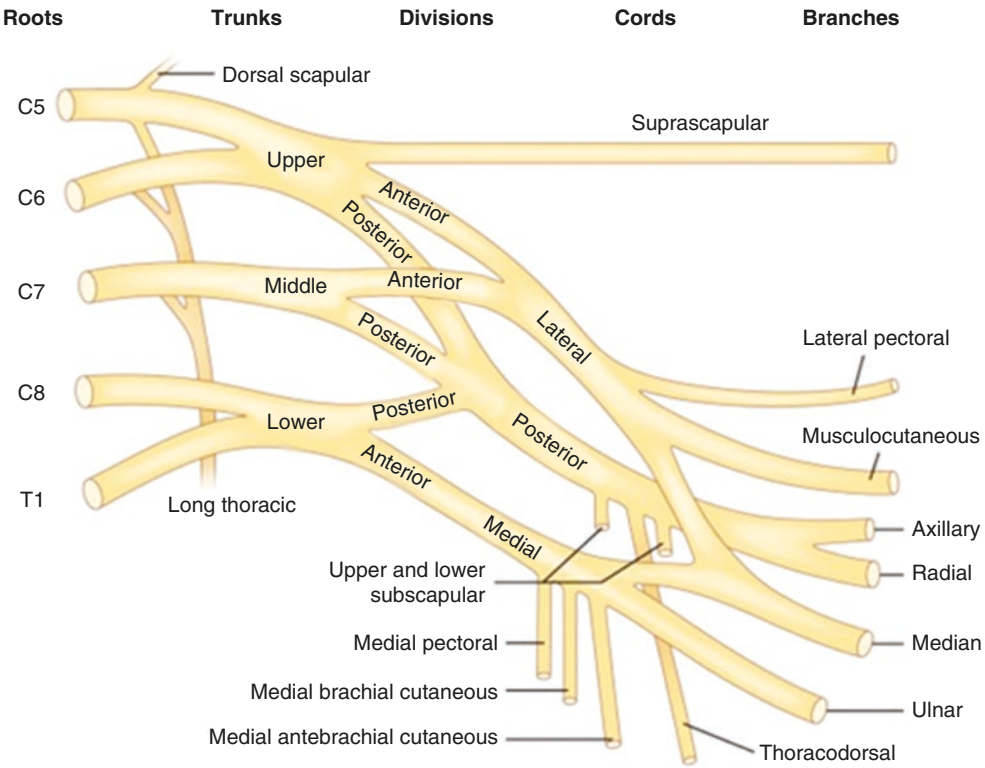

Fig. 9.8 Brachial plexus. Representation of the brachial plexus with depiction of roots, trunks, divisions, cords, and branches. (Reprinted with permission from Bednar and Wurapa [29])

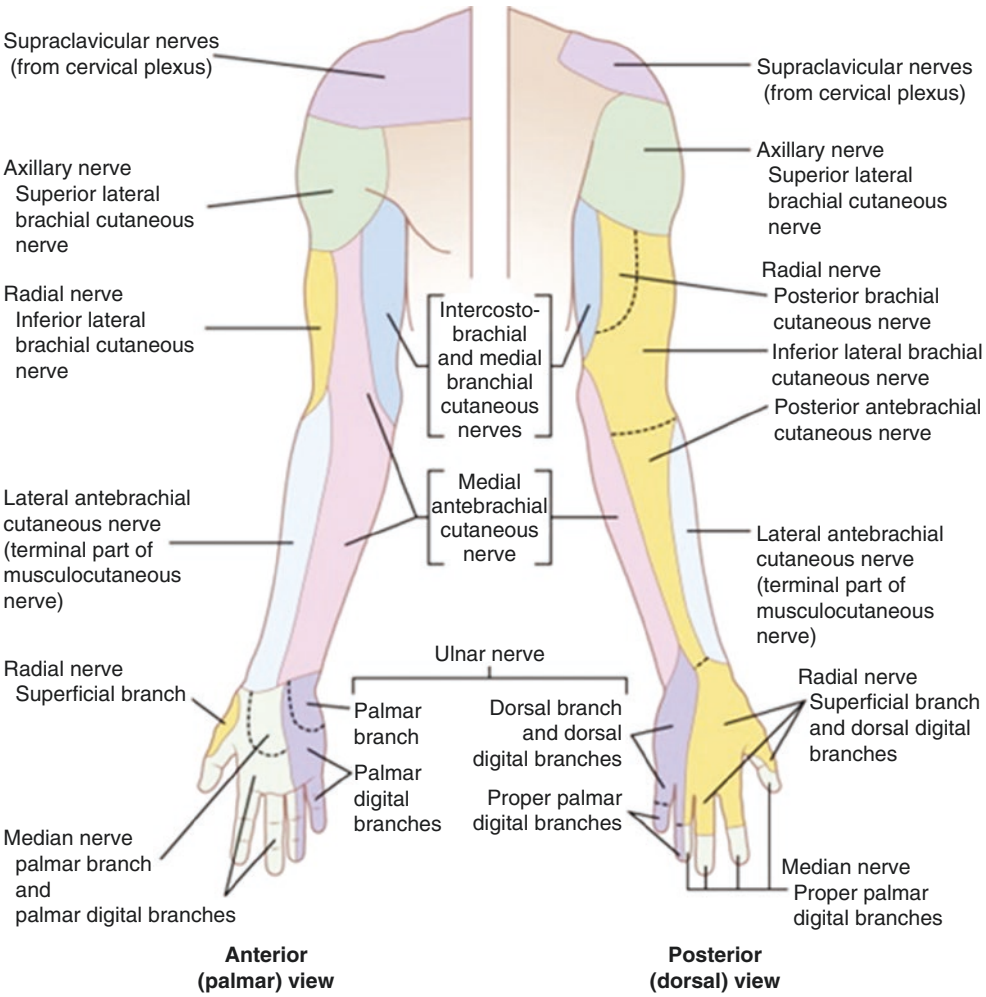

Fig. 9.9 Cutaneous innervation of the upper limb. Anterior and posterior view displayed. (Reprinted with permission from Hooks [30]. © 2012)

with surgeries requiring chest-wall retraction. Brachial plexitis (Parsonage-Turner syndrome), bilateral carpal tunnel, and neurogenic thoracic outlet syndrome are commonly on the differential and will be discussed further [18].

Idiopathic brachial plexitis, known as Parsonage-Turner syndrome, clinically presents with acute onset of severe pain involving the neck, shoulder, and periscapular area followed by weakness and numbness 2–3 weeks later. Clinical prognosis is variable and dependent on severity of injury with functional recovery estimated to occur between months and up to 3 years. The underlying pathophysiology is not well understood but is attributed to an immune-mediated mechanism. Known risk factors include recent infection or vaccination. NCS/EMG of brachial plexitis demonstrates a patchy distribution with neuropathic pattern of injury and can show proximal conduction block [19].

Median nerve entrapment at the wrist (carpal tunnel syndrome) presents as wrist and arm pain with associated hand paresthesia involving the first, second, third, and splitting the fourth digit (Fig. 9.10). Symptoms are aggravated with prolonged wrist flexion or extension. Nocturnal paresthesias are common. Functional hand weakness is typically a delayed finding and is associated with wasting of the thenar eminence. The differential for hand pain, paresthesias, and numbness can also include cervical (C6–C7) radiculopathy especially if the presentation is bilateral. NCS/EMG can distinguish these etiologies. In carpal tunnel syndrome, NCS should demonstrate distal focal slowing or conduction block of the median nerve across the carpal tunnel. EMG may show denervation in the median nerve innervated abductor pollicis brevis (APB) [6]. A cervical radiculopathy would

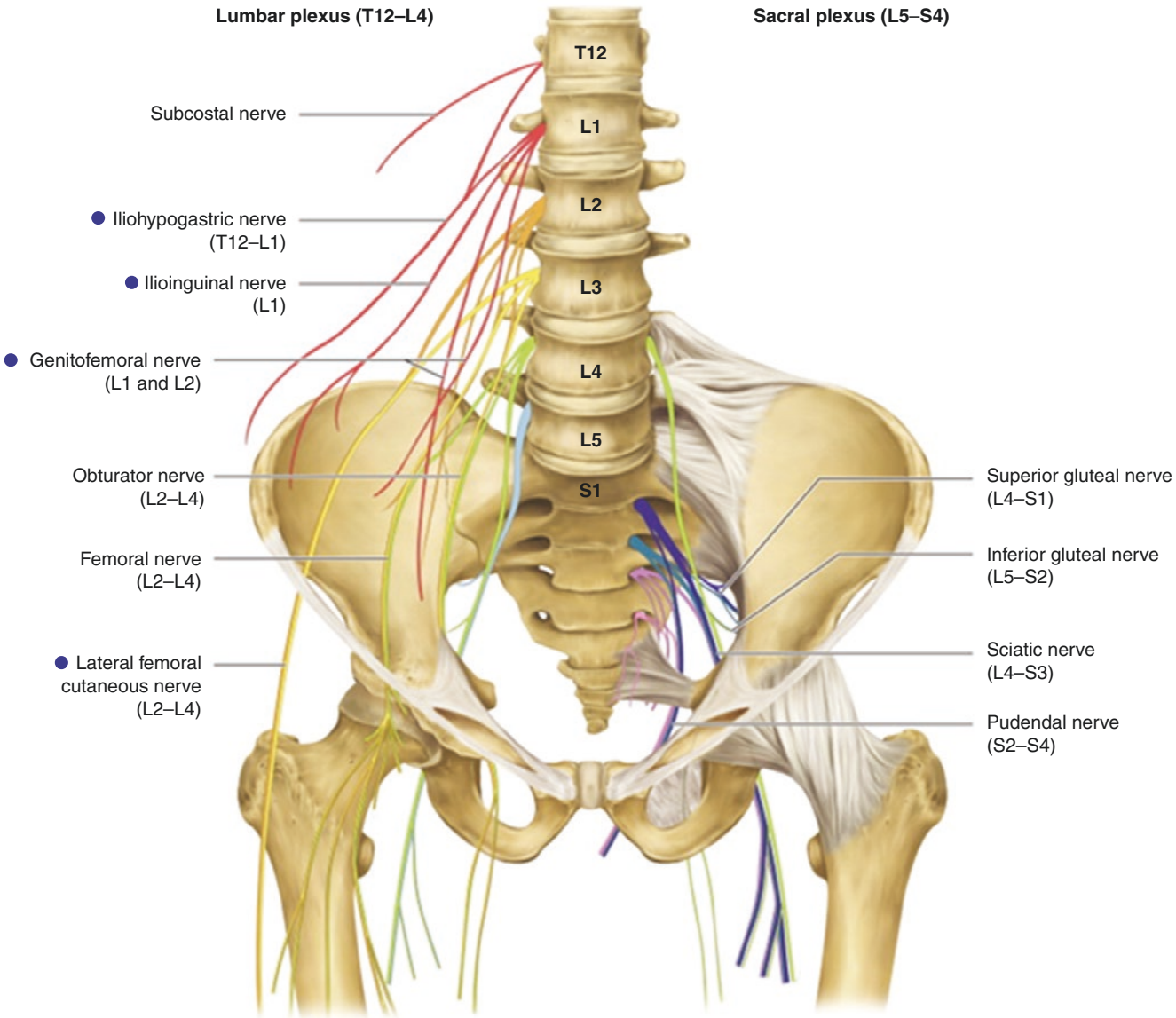

Fig. 9.10 Lumbosacral plexus. (Reprinted with permission from Kim et al. [31])

have normal SNAP (lesion proximal to the dorsal root ganglion) but abnormal CMAP and needle EMG findings in the distribution of the affected myotome and including areas proximal to the wrist [7, 13].

Neurogenic thoracic outlet syndrome is rare condition that can present as neck and shoulder pain with associated limb paresthesias and weakness [20]. Symptoms are worsened or provoked with sustained overhead activity. Neurogenic thoracic outlet syndrome is caused by the entrapment of the lower trunk of the brachial plexus by a fibrous band from a cervical rib. NCS/EMG findings are typically most consistent with a lower trunk plexopathy. Accordingly, sensory NCS studies will have abnormal SNAPs in ulnar and medial antebrachial cutaneous nerves. Motor NCS studies will be abnormal in the median and ulnar innervated muscles. Neuropathic pattern of injury demonstrating denervation, MUAP abnormalities, and reduced recruitment is expected in the median and ulnar innervated muscles.

Lumbar Spine Disease Mimics

Lumbosacral plexopathy and lower extremity neuropathy can mimic lumbar spine disease by presenting with pain, sensory changes, and motor weakness in the lower back and leg. Diagnosing radiculopathy vs plexopathy vs mononeuropathy can be challenging without neurophysiologic testing. We will review the lower extremity neuroanatomy, clinical syndromes, and relevant NCS/EMG findings.

The lumbosacral plexus is divided into the upper lumbar plexus (L1–L4) and lower lumbosacral plexus (L5–S3). The upper lumbar plexus gives rise to the iliohypogastric nerve, ilioinguinal nerve, lateral femoral cutaneous nerve of the thigh, genitofemoral nerve, femoral nerve, and obturator nerve. The lower lumbosacral plexus gives rise to the superior gluteal nerve, inferior gluteal nerve, pudendal nerve, sciatic nerve, and posterior cutaneous nerve of the thigh. The sciatic nerve at the popliteal fossa divides into the tibial nerve and common peroneal nerve, which itself divides into the superficial and deep peroneal nerve (see Fig. 9.10). Cutaneous innervation of the lower limb with branches of the lumbosacral plexus and distal peripheral nerves is described in Fig. 9.11.

Common etiologies in lumbosacral plexopathy include hip or pelvis trauma/surgery, postradiation injury, diabetic amyotrophy (also described as radiculoplexus neuropathy), postpartum plexopathy, and mass lesions including neoplasm, retroperitoneal hematoma, and psoas abscesses [21]. Lumbosacral radiculopathy and lumbosacral plexus lesions can be difficult to distinguish clinically given similarity of symptoms: low back pain, pelvic pain, and lower extremity numbness and weakness. NCS/EMG is helpful in distinguishing these disorders. EMG of the paraspinal muscles is expected to be abnormal in radiculopathy and normal in plexopathy. Sensory NCS is expected to normal in radiculopathy (because the lesion is proximal to the dorsal root ganglion) and abnormal in lumbosacral plexopathy and distal peripheral nerve disorders.

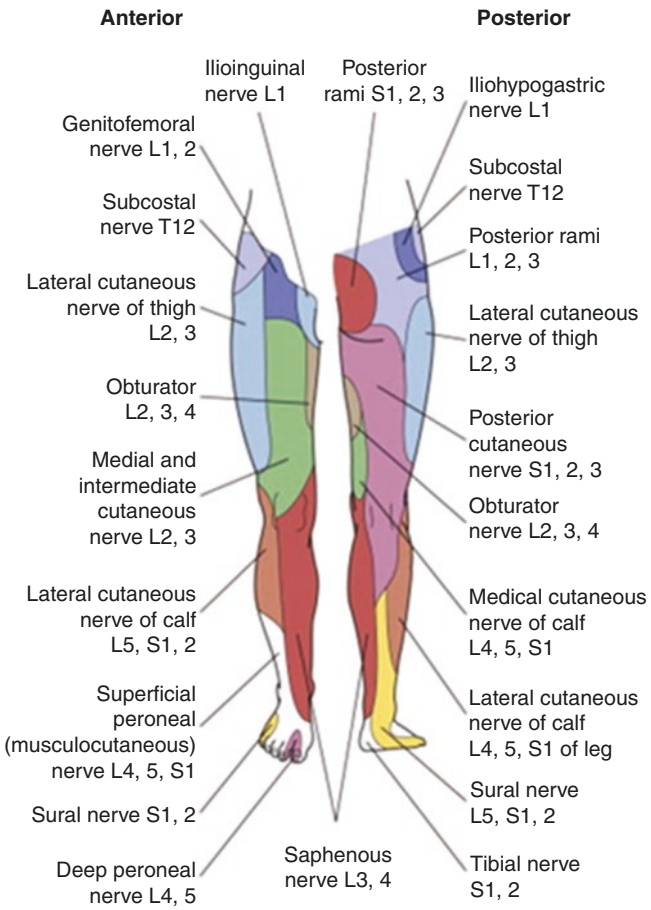

Fig. 9.11 Cutaneous innervation of the lower limb. Peripheral nerve cutaneous innervation of the lower limb. (Reprinted with permission from Harmon et al. [32])

Diabetic amyotrophy presents with unilateral deep pain in the pelvis and hip for 4–6 weeks followed by proximal leg weakness, weight loss, and autonomic dysfunction [22]. Diabetic amyotrophy primarily affects the upper lumbosacral plexus but can also involve the nerve roots and thus is described as a radiculoplexopathy. Clinical weakness primarily involves the obturator and femoral nerve manifesting in hip flexion weakness, hip adduction weakness, and knee extension weakness. The patellar reflex is typically diminished or absent. The pathophysiology underlying diabetic amyotrophy is thought to be chronic microvascular ischemic injury. NCS/EMG in diabetic amyotrophy is consistent with neuropathic pattern of injury predominantly in the L2–L4 myotome which reflects the clinical weakness [6].

Meralgia paresthetica or isolated entrapment of the lateral femoral cutaneous nerve is another clinically important syn-

drome to recognize. The lateral femoral cutaneous nerve arises from L2 to L3 nerve roots and is part of the upper lumbar plexus. Isolated lateral femoral cutaneous nerve entrapment presents with pain, burning, and numbness over the anterior and lateral thigh without focal weakness. Etiology is likely compressive with risk factors of obesity, diabetes, and tight clothing – namely, belts and pants. As discussed above, the lateral femoral cutaneous nerve can be assessed by sensory NCS. Due to technical difficulty, reduced or absent SNAPs should be cautiously interpreted with bilateral comparisons and correlated to clinical history [23].

Limitations of NCS/EMG

NCS/EMG is an important diagnostic tool for radiculopathy, plexopathy, mononeuropathies, and polyneuropathies. It has a limited utility in spinal pathology without associated peripheral nerve or myotome to interrogate. For instance, discogenic pain can clinically present similarly to radiculopathy with radiating pain in the affected dermatomal level with or without associated weakness or paresthesias. The intervertebral disc is innervated by branches of the sinuvertebral nerve and branches of the paravertebral sympathetic trunk which cannot be examined by NCS/EMG [24]. Likewise, facetogenic pain is a source of axial neck and back pain exacerbated with facet loading action such as extension and rotation [25]. Facet joints are innervated by the medial branch nerves dorsal rami at the level of and level above the lesion, and these nerves are not interrogatable by NCS/EMG (Fig. 9.12).

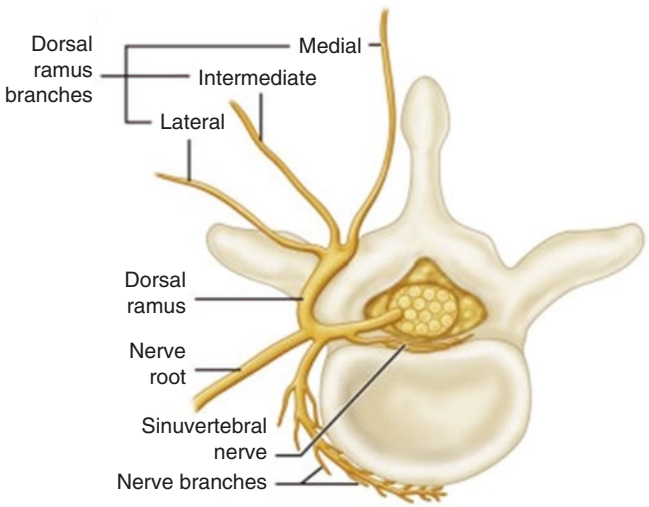

Fig. 9.12 Sensory innervation of the spine. Cross-sectional view of the spine at endplate and disc. Sinuvertebral nerve innervates the dorsal surface of the disc. The medial branch nerves of the dorsal rami innervate the facet joints. (Reprinted with permission from Gardocki and Park [33])

Summary

An understanding of the neurological examination and in continuation the neuromuscular diagnostics tests, EMG and NCS are important tools for the pain physician. Often, pain practitioners play a primary diagnostic role, in addition to treating pain. In such cases, an intimate understanding of neurological assessment is crucial. Performing a screening examination is the best way to make sure that one does not miss a diagnosis or mistake one condition for another. As an unfortunate example, many patients are treated with interventional procedures, including spine surgeries, when the ultimate diagnosis is a progressive neuropathy or even amyotrophic lateral sclerosis. The critical ability to identify upper motor neuron signs can implicate cervical spine stenosis and consequent myelopathy masquerading as lower extremity pain.

Beyond the neurological examination, NCS and EMG are essential for confirming the involvement of specific nerves and muscles or identifying a key pathological process at play in a patient's condition. Important considerations include the timing of injury and whether to expect signs of acute denervation on EMG, namely, fibrillation potentials and positive sharp ways, or whether the main features will reflect chronic denervation and reinnervation, as evidenced by changes in motor unit morphology. For neuropathy, the main features include patterns of axonal loss versus demyelination, as the latter include a smaller group of conditions for which some have specific treatments.

The use of NCS/EMG for pain physicians includes a range of indications, such as diagnosis and prognosis, monitoring disease progression, and evaluating patients for interventions including surgery. While indications for NCS/EMG are at times uncertain, the tests are particularly useful in cases where examination is limited or conclusions uncertain.

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