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Diagnostic Utility of EMG and Nerve Conduction Studies in Differentiating Radiculopathy from Peripheral Nerve Disorders

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Abstract

Radiculopathy and peripheral nerve disorders share overlapping symptomatology, ranging from pain, paresthesia, and weakness. This makes clinical differentiation difficult. Physical examination has limited sensitivity, and structural imaging frequently shows poor symptom correlation, particularly in patients with coexisting conditions such as diabetic neuropathy or double crush syndrome. We critically reviewed the diagnostic principles and clinical utility of electromyography (EMG) and nerve conduction studies (NCS) in differentiating radiculopathy from mononeuropathy, plexopathy, and polyneuropathy. Needle EMG is the primary tool for radiculopathy detection (sensitivity 42–77%, specificity 71–85%), identifying myotomal denervation across muscles supplied by different peripheral nerves. Paraspinal EMG, the earliest EMG finding in radiculopathy, reduces the false-negative rate from 33% to 5% when combined with limb EMG. Preserved sensory nerve action potentials in NCS are the key localizing feature of radiculopathy, reflecting its preganglionic nature. Advanced H-reflex techniques achieve sensitivity up to 98% for S1 radiculopathy. NCS are often independently diagnostic in entrapment neuropathies (sensitivity 56–91%), while reduced SNAPs with normal paraspinal EMG reliably identify plexopathy. EMG may alter diagnosis or management in 24–43% of polyneuropathy evaluations. EMG and NCS are complementary modalities that, when used together, significantly improve diagnostic accuracy over either test alone. Limitations include the inability to detect pure sensory radiculopathies and small fiber neuropathies. Emerging adjuncts such as MR neurography and dermatomal somatosensory evoked potentials show promise but require further validation.

Keywords: Electrodiagnostic testing; Electromyography; H-reflex; Hoffman reflex; Mononeuropathy; Nerve conduction; Peripheral neuropathy; Plexopathy; Radiculopathy; Sensory nerve action potential.

Introduction

Peripheral nerve diseases and radiculopathy are two major neurological disorders that represent a growing public health burden due to their prevalence, significant disability, complexity of diagnosis, substantial healthcare costs, and limited treatment efficacy. Peripheral neuropathy, the most common type of peripheral nerve disease, affects about 1% of all adults worldwide, and that number rises to 6-10% of adults who are over the age of 60 years [1]. Diabetic Neuropathy has been identified as the fifth most common neurological cause of disability in the Global Burden of Disease 2021 study, as it has been reported in approximately 206 million people worldwide. In 2021, diabetic

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neuropathy generated over 2.2 million disability adjusted life years in the United States, and is the leading cause of non-fatal neurological disability [2]. Radiculopathy is similarly common, with the incidence of lumbar radiculopathy being estimated at 486 per 100,000 person-years, and cervical radiculopathy at 83 per 100,000 person-years [3]. The one-year prevalence of lumbosacral pain ranges from 3% to 14%, and more than half the patients continue to experience pain after the one-year period [4].

In the most common form of peripheral neuropathy, length-dependent polyneuropathy, the classical presentation includes deficits in a symmetric stocking-glove distribution that is not referrable to a single nerve root or peripheral nerve [1,5]. Pain in peripheral neuropathy typically begins distally in the toes or feet as a burning or tingling sensation, and progresses proximally, with the hands involved later as the disease progresses [1,6]. Sensory loss in peripheral neuropathy is symmetric and length-dependent, often affecting multiple modalities, and is accompanied by reduced or absent ankle reflexes and possible autonomic features depending on the underlying etiology [6,7]. The onset of peripheral neuropathy is generally gradual and progressive and is typically not associated with back or neck pain [8].

Peripheral nerve disorders may also present as mononeuropathies, involving a single named nerve, and represent a critical category that can closely mimic radiculopathy and contribute significantly to diagnostic uncertainty. Mononeuropathies produce symptoms confined to the distribution of a single peripheral nerve, whereas radiculopathy produces symptoms that follow dermatomal sensory and myotomal motor patterns [9]. Classically, in mononeuropathies, abnormal sensory findings are restricted to a single peripheral nerve territory, and motor changes are limited to muscles innervated by that nerve. The pain in mononeuropathies is typically more localized along a peripheral nerve distribution. Additionally, the muscle reflexes are typically preserved in patients on a clinical exam [9].

In contrast to peripheral nerve diseases, radiculopathy, which results from compression or irritation of a spinal nerve root, will produce unilateral symptoms that follow dermatomal and myotomal distributions [8,10]. Pain in radiculopathy radiates from the spine into the extremity, often extending below the knee in lumbar cases or into the arm in cervical cases, and is frequently associated with back or neck pain [11,12]. Motor and reflex changes in radiculopathy are root-specific, with myotomal weakness and asymmetric hyporeflexia, and symptoms are sometimes reproduced with provocative maneuvers such as the straight leg raise or Spurling test [13,14]. Sensory findings in radiculopathy follow a dermatomal pattern but may be subtle due to overlapping innervation [15].

Distinguishing peripheral nerve diseases from radiculopathy at the bedside is often difficult, however, due to the extensive overlap of the symptoms, as both conditions can produce numbness, tingling, and pain, the neuropathic symptom triad, as well as weakness. Patients with either condition can complain of burning, electric shock sensations, and paresthesias, and while peripheral and poly neuropathies classically follow a stocking glove-like distribution, mononeuropathies classically follow a single nerve distribution, and radiculopathy classically radiates from the spine distally in a dermatomal and myotomal pattern, these patterns are not always clearly delineated, particularly in early or mild disease [5].

In clinical practice, physical exams have proven to be limited in diagnostic efficacy, as provocative maneuvers (i.e Spurling test and Straight Leg Raise) have modest sensitivity for radiculopathy and can be negative in chronic and mild cases [10]. Musculoskeletal conditions (i.e hip and knee osteoarthritis, lumbar facet syndrome, myofascial pain syndrome, greater trochanteric pain syndrome, and plantar fasciitis) also can mimic radiculopathy with radiating pain and sensorimotor disruption, which makes diagnosing the source cause of the pain with a clinical exam extremely challenging [16]. Additionally, the peripheral nerve diseases and radiculopathies can often coexist, especially in patients with diabetes and other systemic conditions, which makes the clinical picture more blurred [17].

For instance, according to the “double crush” concept, the proximal nerve root compression and distal nerve entrapment neuropathies can work synergistically to worsen patient symptoms. In a hypothesis presented by Upton and McComas in 1973, they stated that compression of a peripheral nerve at a proximal site (i.e at the nerve roots which lead to radiiculopathy) will render the nerve more susceptible to injury at the distal sites (peripheral neuropathy presentation; i.e. carpal tunnel and cubital tunnel), and that the two conditions can work synergistically to worsen the symptoms to a greater level than each individual condition alone [18]. However, contrary to this hypothesis, electrodiagnostic data suggest that anatomically appropriate double crush is actually rare (about 0.5-0.8% of entrapment neuropathies), pointing to the fact that when both conditions exist, they often have independent processes co-occurring, which makes localizing the more significant cause of the issue difficult [19]. This diagnostic uncertainty highlights the need for objective diagnostic tools that can provide more precise localization beyond what can be achieved with clinical evaluation and imaging alone.

This critical review describes the foundational principles and clinical applications of electromyographic (EMG) and nerve conduction studies (NCS) in assessing radiculopathy, emphasizing their ability to differentiate between nerve root

and peripheral nerve disorders. The review will also discuss limitations of the studies, analyze the use of adjunct studies, and analyze future diagnostic approaches that could improve clinical decision-making.

Diagnostic Limitations and Role of Electrodiagnostic Testing

While imaging can help identify structural abnormalities and potential sources of nerve compression, it often lacks the specificity needed to accurately localize the underlying source of the patient's symptoms, particularly when multiple processes may be present. MRI findings frequently do not align well with the clinical picture, with guidelines noting poor correlation between imaging abnormalities and clinical findings [20]. Studies have also demonstrated low sensitivity for detecting nerve root compromise, further limiting the reliability of imaging for localization [21]. In addition, degenerative changes are highly prevalent in asymptomatic individuals, suggesting that many imaging findings may reflect age-related or incidental changes rather than the true symptomatic lesion [22-24].

This limitation has important clinical implications, as excessive reliance on imaging may lead to overdiagnosis of incidental asymptomatic spinal changes. These findings may not reflect the true pain generator and can contribute to unnecessary surgical intervention, particularly in patients with complex and overlapping pain presentations [25]. Imaging has also not consistently been shown to improve outcomes in these settings and may contribute to increased rates of intervention without clear clinical benefit [11]. In patients with suspected radiculopathy, inaccurate localization can also result in targeting the wrong level during epidural steroid injections, despite evidence emphasizing the importance of precise targeting for optimal outcomes [26].

The picture becomes even more complicated when pathology overlaps, as peripheral nerve diseases, radiculopathies, and plexopathies may coexist, and conditions such as double crush syndrome can make it difficult to identify the main driver of symptoms, leading to incomplete treatment or persistent symptoms if not recognized [18,27]. Electrodiagnostic studies further support this by demonstrating distinct patterns in patients with combined proximal and distal nerve involvement, with proximal EMG findings raising suspicion for coexisting radiculopathy in patients presenting with peripheral nerve symptoms [28].

Delays in identifying the correct diagnosis can also affect outcomes, as prolonged time to appropriate referral and intervention in peripheral nerve entrapment syndromes has been associated with worse recovery [29]. Timely evaluation and referral are therefore critical for optimizing recovery and functional outcomes [30].

In this setting, electrodiagnostic testing with electromyography and nerve conduction studies offers an important complement to imaging by providing functional information rather than just structural detail. Needle EMG is particularly useful for identifying radiculopathy by demonstrating myotomal patterns of denervation, including involvement of paraspinal muscles [31]. These findings help distinguish root level pathology from peripheral nerve and plexus disorders [12,32].

Nerve conduction studies further support the evaluation by identifying focal slowing or conduction block in entrapment neuropathies and by helping exclude other peripheral nerve and neuromuscular conditions with similar presentations [31,33]. Beyond improving diagnostic accuracy, electrodiagnostic findings can also help guide management, as EMG-confirmed radiculopathy has been associated with better response to conservative treatment, more accurate targeting of interventional procedures, and improved surgical outcomes [34].

Electrodiagnostic testing also provides prognostic information and helps guide appropriate patient selection for interventions such as peripheral nerve stimulation and spinal cord stimulation [35, 36]. Taken together, these limitations of imaging and the consequences of misclassification highlight the need for additional tools to support clinical decision making, and EMG and nerve conduction studies play a key role in clarifying the clinical picture and enabling more accurate diagnosis of peripheral nerve diseases, including mononeuropathies, polyneuropathies, and plexopathies, as well as radiculopathies, ultimately helping guide more targeted and effective treatment.

Needle Electromyography: Principles and Distinguishing Features

Overview

Needle electromyography (EMG) remains the single most important electrodiagnostic tool in the evaluation of radiculopathy, offering modest sensitivity but high specificity for the diagnosis [31,34]. A study that used electromyography experts to identify radiculopathy found sensitivity of 77% and specificity of 71% [37]. Its primary clinical value lies in anatomic localization, specifically, in determining whether a pattern of denervation follows a myotomal (root-level) distribution or peripheral nerve distribution. It also helps to characterize both the acuity (acute versus chronic) and severity (degree of axonal loss) of the lesion [38,39].

Spontaneous Activity (Fibrillation Potentials and Positive Short Waves)

Fibrillation potentials and positive sharp waves (PSWs) represent the hallmark spontaneous activity markers of active denervation resulting from axonal interruption and

are critical for confirming ongoing nerve injury [12,38]. The timing of spontaneous activity onset following nerve injury is an important consideration in electrodiagnostic evaluation. The widely cited guideline that fibrillation potentials appear within one to four weeks of injury derives primarily from mid-20th century animal studies. Human data on this timeline remain limited [40]. In cervical radiculopathy, abnormal insertional activity, including PSWs and fibrillation potentials, typically appears in limb muscles of the involved myotome within three weeks of nerve compression onset [32]. Paraspinal muscles, owing to their shorter axonal distance from the lesion site, may demonstrate changes earlier, as soon as ten days after injury [32,38]. Conversely, distal muscles show changes later than proximal muscles, reflecting the time required for Wallerian degeneration to reach the motor endplate along a proximal-to-distal gradient [12]. These temporal considerations carry direct clinical implications: EMG performed too early, within two to three weeks of symptom onset, may yield false-negative results. Optimal timing for electrodiagnostic evaluation is generally three to four weeks after symptom onset.

Although PSWs and fibrillation potentials are often grouped together in clinical practice, they are electrophysiologically distinct waveforms [41]. Notably, PSWs may appear earlier than fibrillation potentials following peripheral nerve injury. This may suggest differences in the underlying mechanisms of membrane instability that generate each discharge type [41]. Furthermore, PSWs alone, in the absence of fibrillation potentials, can occur in non-pathologic contexts. Common sites this can occur at include distal muscles of normal subjects, where there has been local muscle trauma from needle insertion, and in certain demyelinating polyneuropathies [41]. These observations indicate that isolated PSWs identified in a single muscle should be interpreted with caution. Moreover, fibrillation potentials carry greater diagnostic weight as markers of true denervation.

The distinction between myotomal and peripheral-nerve distributions of denervation is the critical localizing principle in needle EMG. In radiculopathy, spontaneous activity is expected in two or more muscles that share the same myotomal innervation but are supplied by different peripheral nerves [9,42]. For example, an L5 radiculopathy would produce denervation in both the tibialis anterior (innervated by the deep peroneal nerve) and the tibialis posterior (innervated by the tibial nerve), as both muscles share L5 root innervation despite receiving their peripheral supply from distinct nerves. In contrast, a peripheral nerve lesion produces spontaneous activity confined to muscles innervated by a single peripheral nerve, regardless of their root-level origin [9, 43]. Peroneal neuropathy, for instance, would produce denervation in the tibialis anterior and peroneus longus, both supplied by the peroneal nerve. However, it would spare the tibialis posterior, which shares L5 myotomal innervation but is supplied by the tibial nerve. This distribution pattern highlights the fundamental needle EMG principle for distinguishing root-level from peripheral nerve pathology.

The severity of spontaneous activity is quantified using the grading scale recommended by the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM), which ranges from 0 to 4+ [44, 45]. A grade of 0 indicates no spontaneous activity. Grade 1+ denotes the presence of persistent but infrequent fibrillation potentials or positive sharp waves identified within the muscle on needle examination. Grade 2+ is assigned when occasional spontaneous potentials are present in three or more areas of the muscle. Grade 3+ indicates moderate numbers of fibrillation potentials and PSWs firing in all areas of the muscle. Grade 4+ represents profuse, widespread spontaneous activity with fibrillation potentials and PSWs filling the baseline in all areas examined [44,46].

The grade of spontaneous activity serves as a semiquantitative index of the severity of axonal loss. Higher grades reflect a greater proportion of denervated muscle fibers within the sampled muscle, indicating more extensive axonal injury [45,46]. In the context of radiculopathy, the presence of denervation on needle EMG, manifested by fibrillation potentials and positive sharp waves, signifies axonal interruption and has important clinical implications. As emphasized by Eisen, evidence of denervation is a key determinant of the degree of nerve injury and may influence the course and extent of recovery [38]. Longitudinal studies further support this relationship; for example, Forero et al. demonstrated that patients with lumbar radiculopathy and initially abnormal EMG findings exhibited both electromyographic and clinical improvement over time, with greater clinical improvement observed in those with positive baseline electrodiagnostic studies [47]. Additionally, EMG-confirmed radiculopathy has been associated with better clinical outcomes following aggressive conservative management, epidural corticosteroid injections, and spine surgery, suggesting that the electrodiagnostic confirmation of the lesion, and by extension, the characterization of its severity, enhances prognostic accuracy and guides treatment selection [34].

Motor Unit Morphology and Recruitment

Motor unit action potential (MUAP) analysis provides complementary information to spontaneous activity findings, offering insight into lesion chronicity and the degree of reinnervation [39,48].

Large-amplitude, long-duration, polyphasic MUAPs indicate collateral reinnervation, surviving motor neurons sprouting to adopt denervated muscle fibers, and typically reflect a chronic or subacute process of three to six months or longer [48]. As with spontaneous activity, the distribution

of these changes carries localizing value: a myotomal pattern indicates radiculopathy, whereas confinement to a single peripheral nerve territory suggests a peripheral nerve lesion. Recent EMG filling analysis has demonstrated that radiculopathy patients exhibit distinct motor unit remodeling patterns compared to healthy controls, with linear rather than exponential filling curves and reduced amplitude increases during ramped contractions [48].

Reduced recruitment is described as fewer motor units firing at abnormally rapid rates for a given force level. This is the hallmark of neurogenic loss. In the earliest period after nerve injury, before denervation potentials appear, recruitment abnormalities may be the only EMG finding. This is most useful for distinguishing neurogenic weakness from pain inhibition or poor effort [38]. Reduced recruitment is notably not specific to neurogenic processes and can also result from poor effort, pain, or upper motor neuron lesions.

Incorporating MUAP changes into diagnostic criteria improves sensitivity. Tong et al. showed that adding a threshold of thirty percent or greater MUAP changes to fibrillation/PSW criteria increased lumbar radiculopathy detection from 27.1% to 45.8% (P = 0.002). while maintaining specificity [42]. This suggests that reliance on spontaneous activity alone underestimates radiculopathy prevalence, particularly in chronic or partially reinnervated cases.

Paraspinal Muscle Examination

Paraspinal EMG provides electrophysiologic proof of a lesion at or proximal to the spinal nerve root, as the paraspinal muscles are innervated by the posterior primary rami, which branch off before the plexus [32,38]. Abnormal paraspinal EMG therefore differentiates radiculopathy from plexopathy and peripheral nerve lesions, and paraspinal denervation is the earliest EMG finding in radiculopathy, appearing as early as ten days after injury. This precedes limb muscle changes due to the shorter axonal distance from the lesion site [32,38]. An abnormal paraspinal EMG combined with a myotomal pattern of limb denervation provides the strongest electrodiagnostic evidence for radiculopathy.

The Mini-Paraspinal Mapping (MiniPM) technique is a quantified, standardized needle EMG method for assessing paraspinal denervation [49,50]. Adding MiniPM scoring to the diagnostic protocol increased radiculopathy detection to 50%, compared with 27.1% using fibrillation potentials and PSWs alone (P = 0.001) [42]. The technique also correlates with imaging findings: the radiologist's overall impression of spinal pathology correlates with MiniPM scores, with the "smallest two levels" measurement showing the strongest relationship (r = 0.400, P = 0.007) [49].

Paraspinal EMG increases the overall sensitivity of the needle examination for radiculopathy [32]. However, paraspinal findings alone carry a false-negative rate of approximately 33%. This decreases to 5% when combined with lower extremity EMG [50]. The S1 radiculopathy is a notable exception: in one series, all seven subjects with isolated S1 radiculopathy had normal paraspinal mapping scores, suggesting that S1 may not reliably innervate the paraspinal muscles [50]. Specificity is also a consideration, as 14.5% of asymptomatic subjects demonstrate PSWs or fibrillation potentials in lumbosacral paraspinal muscles, with prevalence increasing significantly with age [51].

Several limitations of paraspinal EMG warrant consideration when interpreting findings. Prior spine surgery produces persistent paraspinal EMG abnormalities that may confound interpretation: in one study, 17 of 20 post-laminectomy patients demonstrated abnormalities for up to 41 months postoperatively, with 15 abnormal at three or more vertebral levels, even in the absence of recurrent radiculopathy [52]. Age-related changes also affect interpretation, as paraspinal denervation increases with age in both symptomatic and asymptomatic populations. MiniPM scores are significantly higher in subjects aged 55 to 79 compared with those aged 18 to 54 (mean difference 1.7, P = 0.008), with the 95th percentile upper limit reaching 10 in older adults [53]. Furthermore, diffuse paraspinal abnormalities in the setting of multilevel spondylosis may reflect degenerative disease rather than a discrete single-level radiculopathy, thereby reducing the localizing value of the examination. Paraspinal findings should therefore always be interpreted in the context of age, surgical history, and limb EMG findings; isolated paraspinal abnormalities in older or post-surgical patients should not be overinterpreted.

Sampling Strategy

A systematic muscle sampling strategy is essential to maximize diagnostic sensitivity while minimizing patient discomfort and procedure time [33, 54]. The fundamental localizing requirement is to examine muscles from at least two different peripheral nerves that share the same myotomal innervation, thereby confirming a root-level lesion [9,42]. For suspected L5 radiculopathy, for example, the examiner should sample the tibialis anterior (deep peroneal nerve), tibialis posterior (tibial nerve), and peroneus longus (superficial peroneal nerve); abnormalities across muscles supplied by different nerves but the same root confirm radiculopathy over mononeuropathy. Muscles from adjacent myotomes should also be sampled to define the boundaries of the lesion and to exclude plexopathy or polyneuropathy.

Regarding the minimum number of muscles required, Dillingham et al. [33] demonstrated that a streamlined examination of six muscles, one of which is the paraspinal, provides a high diagnostic yield while minimizing patient discomfort. Ropper and Zafonte (12) similarly recommend sampling four to five relevant muscles including the paraspinal regions as adequate for most evaluations. Yaar [54] used

computational analysis to identify minimum subsets of six, seven, and nine muscles that ensure each root and its adjacent roots are represented by at least two muscles innervated via different peripheral nerves. The choice of subset size depends on clinical suspicion: a six-muscle screen suffices for most cases, but more extensive sampling may be warranted when the clinical picture is ambiguous or multilevel disease is suspected.

Two general approaches to muscle selection exist. A screen approach employs a standardized set of muscles covering all lumbosacral or cervical roots, ensuring no root level is missed, but it requires more needle insertions; this is most useful when clinical localization is uncertain. A targeted approach selects muscles guided by the clinical hypothesis, for example, focusing on L5-innervated muscles from different peripheral nerves plus adjacent L4 and S1 muscles. This offers greater efficiency but risks missing unexpected pathology. A recommended hybrid strategy begins with a focused set based on clinical suspicion and expands if initial findings are inconclusive or suggest a different localization [33]. At least one control muscle from an uninvolved myotome should always be included to rule out diffuse abnormalities, which would suggest polyneuropathy or motor neuron disease.

Several key considerations should be noted. For lumbosacral radiculopathy, muscles innervated by both the peroneal and tibial divisions should be included to distinguish radiculopathy from sciatic neuropathy [43]. For cervical radiculopathy, muscles innervated by different trunks or cords of the brachial plexus help distinguish root-level pathology from plexopathy. Sampling the contralateral homologous muscle can further help differentiate unilateral radiculopathy from bilateral or systemic processes.

Nerve Conduction Studies - Principles and Distinguishing Features

Overview

Nerve conduction studies (NCS) are an important clinical test used by providers to analyze electrical function in clinical nerves. The tests can localize peripheral nerve damage, assess neuropathy severity, and even distinguish between axonal and demyelinating neuropathy. The test can also be used to determine if the nerve damage is generalized, focal, or multifocal. These are key clinical indicators to further identify the cause of the neuropathy.

The test is performed by placing the patient in a comfortable position and appropriately cleaning the testing surface to ensure proper electrode contact. It is essential to check limb temperature, as colder temperatures can lead to slower conduction velocities or increased amplitudes, thus causing false abnormalities. There are a few main electrodes: the recording electrode, which is placed over the nerve or muscle of interest; the reference electrode, which is placed nearby the recording electrode; and the ground electrode, which is placed between the recording and reference electrodes to reduce interference. Electrical impulses are delivered until a maximal response is achieved, and the response is subsequently recorded. The electrical activity is then measured and analyzed to assess sensory nerve action potentials (SNAPs) and compound muscle action potentials (CMAPs), which are used to assess the presence and severity of neuropathy [55].

Sensory Nerve Action Potentials

Sensory Nerve Action Potentials (SNAPs) are an important marker assessed in NCS. SNAPs assess for electrical activity in sensory nerve fibers. SNAPs can be the earliest and most sensitive marker for peripheral nerve disease [1]. Two key values are assessed when analyzing SNAPs: amplitude and conduction velocity. Amplitude analyzes the number of functioning large myelinated sensory axons. Conduction velocity reflects the integrity of the myelin sheath [56].

Amplitude is an essential component of measuring SNAPs. It is measured either from baseline to peak in microvolts. The value is directly proportional to the number of large myelinated sensory axons conducting impulses. It is valued as one of the most important SNAP markers, as it reflects the severity of neuropathy. Decreased amplitude indicates axonal loss, as seen in diabetic polyneuropathy (56). There may be mild drops in conduction velocity, defined as up to 80% of the lower limit of normal, which is primarily due to the fact that the fastest-conducting large fibers are preferentially lost. This causes the slower fibers to be the last remaining fibers to assess for velocity [57].

Conduction velocity reflects the speed at which impulses propagate along myelinated sensory fibers. It is measured as an equation: Conduction velocity = Distance (mm)/ Onset latency (ms). The distance is the value between the stimulating and recording electrodes. The onset latency is the time difference between the stimulus and the initial deflection of the SNAP. A decreased conduction velocity could indicate a demyelinating process, as seen in acute inflammatory demyelinating polyneuropathy (AIDP) [56].

It is important to note that the absence of SNAPs may indicate severe axonal neuropathy or severe demyelination with secondary axonal degeneration. Therefore, the absence could be an important clinical marker to analyze the extent of the disease. If both amplitude and conduction velocity are decreased, this suggests a mixed process involving axonal degeneration and demyelination [58]. Other tests will be needed to determine the cause of the patient's symptomatology.

SNAPs can be a highly diagnostic marker when assessing for peripheral neuropathy. Sensitivity varies with the nerve being tested and the type of neuropathy; however,

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abnormal results are clinically significant. In the setting of clinically-suspected polyneuropathy, Kural et al. [59] found that near nerve testing of the sural nerve had a sensitivity of 77%, demonstrating the value in sural nerve testing in neuropathy. The same study also reports similar sensitivities for near-nerve testing of the dorsal sural (72%) and medial plantar (75%) nerves, indicating that distal nerves can also identify peripheral neuropathies. With all tests performed, the specificity of nerve conduction studies for peripheral neuropathy ranged from 85% to 95% [59].

Abnormal Median - Sural Sparing Pattern

A clinically important diagnostic clue when analyzing SNAPs is the abnormal median - normal sural pattern. The clue suggests absent or reduced SNAPs in the median nerve, with preserved SNAPs in the sural nerve. This is the opposite of what is expected in a length-dependent neuropathy, where the long sural nerve is typically affected early in the course of the disease.

The pattern is strongly associated with Acute Inflammatory Demyelinating Polyneuropathy (AIDP)/Guillain-Barré syndrome (GBS)/Chronic Inflammatory Demyelinating Polyneuropathy (CIDP). Sensitivity of this sign among patients with AIDP is 39%. It occurs almost exclusively in AIDP and CIDP [3]. When this sign is included in diagnosing GBS, which typically includes motor-only criteria, the diagnostic sensitivity of GBS increases from about 50% to 79 to 90% [60].

Compound Muscle Action Potentials

Compound Muscle Action Potentials (CMAPs) are the electrical response of a muscle after stimulating a motor nerve. Similar to SNAPs, the key values measured are conduction velocity and amplitude, albeit with some slight differences. Important to note: CMAPs are measured in millivolts, whereas SNAPs are measured in microvolts, which reduces technical artifacts during testing.

Amplitude is measured from baseline to peak, versus peak to peak in SNAPs. A decrease in amplitude reflects axonal degeneration of motor neurons. This form of degeneration could point towards peripheral neuropathies, such as advanced diabetic neuropathy. It can also point to amyotrophic lateral sclerosis, particularly in the later stages. In axonal neuropathies, there is no correlation between CMAP amplitude and duration; the amplitude will drop without widening the waveform [61].

The conduction velocity is now measured with the equation Conduction Velocity = Distance / (Proximal Latency - Distal Latency). The subtraction in the denominator is necessary to account for the delay in the neuromuscular junction. A decreased conduction velocity points towards demyelination, seen in AIDP. In demyelinating neuropathies, CMAP amplitude is lower, and duration is increased in contrast to axonal neuropathies. The decreased amplitude is due to temporal dispersion and phase cancellation, rather than to true axonal loss [61].

CMAPs have lower diagnostic value in the context of radiculopathy. CMAPs are usually normal in radiculopathy, as multiple nerve roots will contribute to the innervation of a muscle. Their primary value results in their ability to rule out other pathologies, including polyneuropathy and entrapment neuropathy [62]. Abnormal CMAP amplitudes could potentially be useful in patients with electromyographically confirmed radiculopathies, as they could be a noninvasive way to assess for active denervation. A study tested ulnar and fibular motor CMAP amplitudes in those with electromyographically confirmed C8 radiculopathies and L5 radiculopathies, respectively. The findings showed diminished CMAP amplitudes compared to those without C8 or L5 radiculopathies, highlighting active denervation in those with severe radiculopathy [63].

F-Wave

F-Waves are low-amplitude, late motor responses that are generated when a supramaximal stimulus drives back up the neuron (antidromically) to the anterior horn cell. They are a diagnostically useful tool for assessing lesions in more proximal nerve roots. There are a few key features that distinguish F-waves. Firstly, they require supramaximal stimulation, unlike H-waves, which require submaximal stimulation. Secondly, they vary in latency and amplitude because different motor neurons will antidromically fire with each stimulus. Third, they can be obtained from any motor nerve [64].

A few important parameters can be derived from F-waves. The most commonly used parameter is minimum latency, which reflects conduction from the fastest motor axons over the entire nerve length. A prolonged F-wave minimum latency is a strongly sensitive indicator of polyneuropathy. A study from Jerath et al. [65] found that a longer F-wave minimum latency was found in 96.9% of 484 patients with polyneuropathy. An alternative to minimum latency is mean latency, which is the average latency across all F-waves. In contrast, maximum latency is also measured to assess the slowest conducting motor neurons [66].

Chronodispersion is another measured parameter, defined as the difference between the maximum and minimum latencies. Increased chronodispersion is a prominent indicator of differential slowing along motor axons, which could indicate radiculopathy or AIDP [66]. Chronodispersion is the most commonly abnormal finding in lumbosacral radiculopathies, making it a good measure to assess for when performing an NCS [66].

Repeater F-waves are another important measure. It is a parameter that assesses for F-waves with similar latency and

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morphology, indicating that the same motor neuron is firing repeatedly. A higher percentage of repeaters could indicate fewer motor neurons firing, which is seen in radiculopathy and motor neuron disease [67].

H-reflex

The H-reflex (Hoffman reflex) tests a monosynaptic reflex arc utilizing nerve conduction studies. An example of the pathway of the monosynaptic reflex arc involves the Ia sensory afferent neuron → S1 spinal cord synapse → alpha-motor neuron → muscle (soleus). The test is performed by stimulating the tibial nerve with a submaximal stimulus in the popliteal fossa as shown in Figure 1 [68]. At a low stimulus, the H-wave will appear, and as the stimulus increases, the M-wave will grow while the H-wave fades. The produced waves will have constant stimulus and latency. The pathway is significant as it tests both sensory and motor limbs, giving important diagnostic information. The primary value measured is H-reflex latency, the time it takes for the reflex to appear in the study relative to the stimulus onset [68].

A key distinction of H-waves from F-waves is that the H-reflex will test the whole sensory-motor reflex arc, thus assessing for lesions in the sensory fibers, dorsal root, synapse, or ventral root. F-waves assess lesions only in motor pathways.

The primary utility of the H-reflex involves assessing for S1 radiculopathy. The H-reflex is particularly effective at diagnosing S1 radiculopathy. Eliciting a standard H-reflex in the NCS yields sensitivity values of 50-72% in regards to diagnosing S1 radiculopathy. With advanced techniques, this value can be considerably improved. One variation of the H-reflex involves stimulating the S1 foramen; in one study by Jin et al, it demonstrated a sensitivity of 98% (69). The same study demonstrated a sensitivity of 71% for the tibial reflex. Another technique involving biceps femoris long-head radiculopathy demonstrated a sensitivity of 93% for S1 radiculopathy [69].

There are a few important limitations of the H-reflex that must be considered when performing the test. It is particularly important to note that the conventional H-reflex is specific to the S1 root and cannot be used to assess for dysfunction in the L5 nerve root (https://pubmed.ncbi.nlm.nih.gov/2156485/). It is also important to note that the tibial H-reflex test has reduced diagnostic accuracy in patients with concurrent polyneuropathy (e.g., diabetes) because neuropathy can mask unilateral root pathology [70]. It is also important to note that absent ankle reflexes are present in 23% of individuals over the age of 60 years, making the H-test ineffective for diagnosing pathological conditions in this population [71]. Age can be a confounding factor when assessing H-reflex parameters, as there is a general loss of sensory Ia afferent neurons in this population [72].

The H-reflex can be one of the earliest abnormalities in polyneuropathy. A study assessing the H-reflex versus the Achilles reflex for identifying polyneuropathy revealed sensitivities of 70.3% for the H-reflex and 71.8% for the

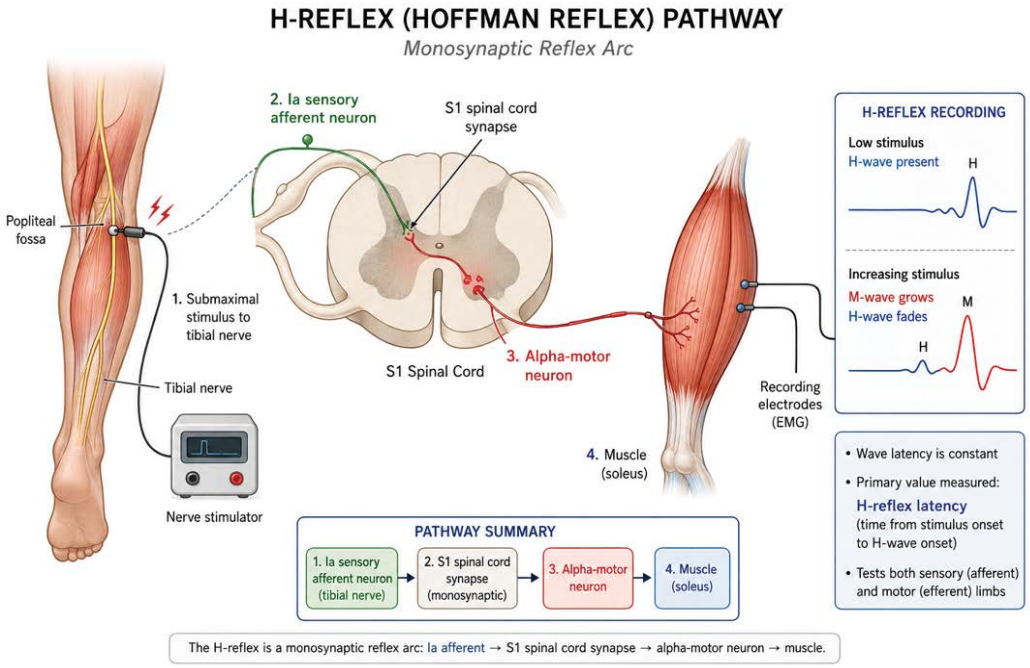


Figure 1: H-Reflex (Hoffman Reflex) Pathway: The H reflex is a monosynaptic reflex arc involving a Ia sensory afferent, S1 spinal cord synapse, and alpha motor neuron to the muscle. It is elicited by submaximal stimulation of the tibial nerve in the popliteal fossa, as shown above, producing an H wave at low stimulus, with the M wave increasing and H wave diminishing as stimulus intensity rises. H reflex latency reflects conduction through both the sensory and motor limbs of the pathway. The clinical information is gathered from Knikou [68].

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Achilles reflex. However, the specificity of the H-reflex was 85.2%, and that of the Achilles reflex was 70.5%, a statistically significant difference. The study shows that the H-reflex could be a useful indicator when polyneuropathy is clinically suspected [71].

In Guillain-Barré syndrome, bilaterally absent H-reflexes is one of the earliest electrodiagnostic findings when performing NCS [73]. A study found that the H-reflex was 100% abnormal in patients with Guillain-Barré syndrome and was the only abnormality in 16% of patients [74]. The study shows that H-testing could be useful when Guillain-Barré syndrome is clinically suspected.

Combined Use of EMG and NCS for Diagnostic Localization

Radiculopathy

Needle electromyography has been shown to be the most sensitive electrodiagnostic component for detecting radiculopathy, as it identifies motor axon loss through fibrillation potentials, positive sharp waves, and motor unit remodeling in a myotomal distribution, as demonstrated by prior electrodiagnostic studies [31]. However, EMG alone has important limitations, as it cannot assess sensory nerve integrity, detect focal demyelination at entrapment sites, or fully characterize coexisting peripheral nerve pathology.

In contrast, nerve conduction studies rarely detect radiculopathy directly because the lesion is preganglionic, resulting in preservation of sensory nerve action potentials and often normal motor responses unless axonal loss is severe enough to reduce compound muscle action potential amplitudes, as shown in electrodiagnostic investigations [62]. SNAPs remain preserved because the sensory cell body in the dorsal root ganglion and its distal axon remain intact despite proximal root compression [12].

Preserved sensory nerve action potentials represent a key localizing feature that is only obtainable through nerve conduction studies. Mondelli et al found that sensory nerve action potential amplitudes are rarely reduced in lumbosacral radiculopathy due to disc herniation, with reductions observed in only 7% of cases, typically in the setting of foraminal involvement affecting the dorsal root ganglion [75]. Additionally, Simmons et al emphasized that radiculopathy represents a preganglionic lesion, resulting in preserved sensory nerve conduction studies even in the presence of clinical sensory deficits, whereas plexopathies and peripheral nerve disorders produce abnormal sensory responses due to pathology distal to the dorsal root ganglion [76].

While sensory nerve action potentials are typically preserved in nerve root disease due to the proximal location of the lesion relative to the dorsal root ganglion, important exceptions exist that can complicate diagnosis. Severe foraminal and extraforaminal disc herniations may directly compress the dorsal root ganglion, leading to Wallerian degeneration and reduced SNAP amplitudes. In the Mondelli et al. study [75], 7% of patients without preserved SNAPs in the setting of nerve root compression had foraminal herniations that compressed the dorsal root ganglion. This inevitably led to Wallerian degeneration of the dorsal root ganglion, thus displaying abnormal SNAPs on nerve conduction studies [75]. Ando et al. [77] used a method to measure the SNAP amplitude ratio (affected/unaffected) to accurately diagnose L5/S1 foraminal stenosis. The study found that the median SNAP amplitude ratio was 0.42 in patients with foraminal and extra-foraminal lesions. Patients who had intraspinal canal lesions would present with a SNAP ratio of 0.85. Using a SNAP amplitude ratio cutoff of 0.5, they accurately assessed foraminal lesions, with a sensitivity of 91.3% and a specificity of 85.7%. The study shows that the SNAP amplitude ratio could be a valuable tool in diagnosing L5 radiculopathy. It can be used to further localize a patient's symptoms and guide future management [77].

Late response studies further extend the utility of nerve conduction studies in evaluating proximal nerve involvement. Jin et al showed that the conventional tibial H reflex identified abnormalities in 39 of 55 patients with S1 radiculopathy, while S1 foramen stimulation identified abnormalities in 54 of 55 patients, demonstrating improved sensitivity for detecting S1 root lesions [69]. Zheng et al. [70] similarly found that S1 foramen H reflex testing had higher sensitivity than conventional tibial H reflex testing in diabetic patients, with sensitivities of 91.0 percent and 63.6 percent, respectively. These findings support the role of late responses in evaluating proximal nerve root pathology.

Needle EMG demonstrates fibrillations and positive sharp waves in a myotomal distribution, affecting muscles innervated by the same root but different peripheral nerves [12]. Paraspinal EMG is also abnormal. This is a key differentiator, as paraspinal muscles are innervated by the posterior primary rami directly from the nerve root, proximal to the plexus [32].

Motor nerve conduction parameters may also provide independent diagnostic value. McNeish et al. [63] demonstrated that reduced compound muscle action potential amplitudes can predict electromyography confirmed radiculopathy, with patients with C8 and L5 radiculopathy showing decreased ulnar and fibular motor amplitudes, respectively, compared to those without radiculopathy. They further identified optimal cut off values of 10.2 mV for ulnar motor amplitudes in C8 radiculopathy and 3.6 mV for fibular motor amplitudes in L5 radiculopathy, with area under the curve values of 0.85 and 0.82, respectively, demonstrating good diagnostic accuracy [63]. These findings suggest that motor amplitude reductions may provide additional diagnostic information beyond needle electromyography alone and may be particularly useful in patients who are unable to tolerate needle electromyography.

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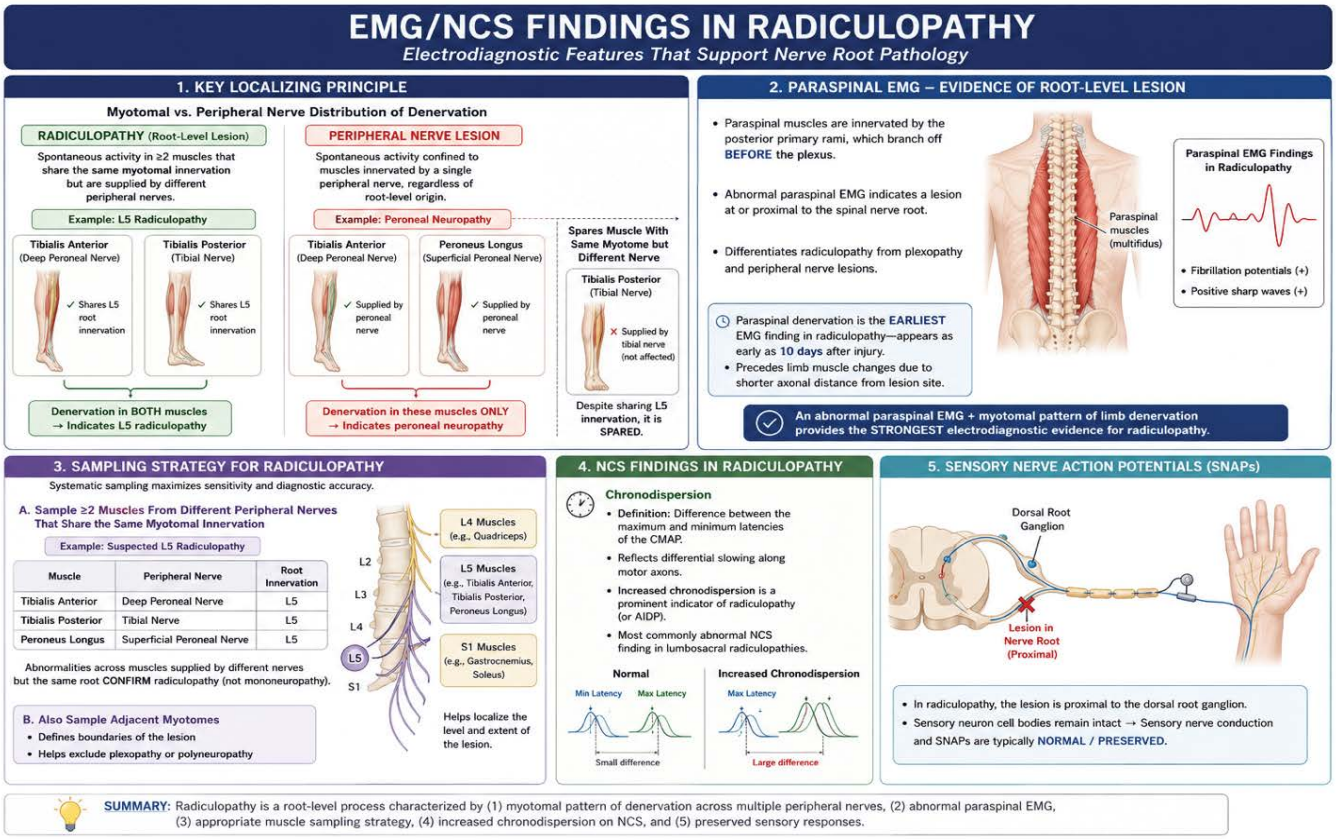


Figure 2: Electrodiagnostic Features that Support the Diagnosis of Radiculopathy. This figure provides an overview of the key electrodiagnostic features that support the diagnosis of radiculopathy. (1) The myotomal pattern of denervation across the muscles supplied by the different peripheral nerves distinguishes radiculopathy from a peripheral nerve disease. (2) Paraspinal EMG results can indicate a lesion that is at or proximal to the nerve root, before the plexus. (3) The systemic muscle sampling across multiple nerves improves localization. (4) Nerve conduction studies can show motor nerve abnormalities such as chronodispersion, defined as an increased speed between the fastest and slowest conducting motor fibers, which typically reflect nonuniform conduction and root-level involvement. (5) The sensory nerve action potentials (SNAPs) are typically preserved, reflecting a preganglionic lesion that does not invade the dorsal root ganglion.

Peripheral Mononeuropathy

Peripheral mononeuropathies are characterized by focal abnormalities localized to a single peripheral nerve, and nerve conduction studies are often diagnostic on their own in these conditions [78,79]. SNAPs and CMAPs show focal abnormalities across the lesion site, including conduction block, focal slowing, or amplitude drop with stimulation proximal compared to distal to the compression point [56].

Padua et al. [80] reported that electrophysiological testing is both sensitive and specific for the diagnosis of carpal tunnel syndrome and plays a central role in confirming entrapment neuropathies. Fowler et al. [81] similarly found that nerve conduction studies demonstrated a sensitivity of 91 percent and specificity of 83 percent for carpal tunnel syndrome, highlighting their diagnostic reliability in identifying median neuropathy at the wrist [81]. These findings are clinically important, as without NCS, a patient with median neuropathy at the wrist may be misclassified as having C6 to C7 radiculopathy based on overlapping EMG findings in shared muscle groups [80-82].

Needle EMG demonstrates denervation confined to muscles innervated by a single peripheral nerve distal to the lesion [43]. In contrast to radiculopathy, paraspinal EMG remains normal [32].

NCS are often diagnostic on their own for common entrapment neuropathies. This includes prolonged median distal sensory latency across the carpal tunnel, conduction block of the ulnar nerve at the elbow, and conduction block of the peroneal nerve at the fibular head [80].

A practical example is the evaluation of foot drop, where the diagnosis is often less straightforward and NCS alone may not be sufficient [83]. Fidancı et al. [43] found that superficial peroneal SNAP abnormalities were present in 21 percent of L5 radiculopathy, 26 percent of peroneal neuropathy at the fibular head, and 76 percent of sciatic neuropathy, showing that sensory abnormalities are much more prominent in more proximal peripheral nerve lesions [43]. Among patients with abnormal SNAPs, peroneal CMAP abnormalities were frequently seen in both L5 radiculopathy and sciatic neuropathy, but were less consistent in peroneal neuropathy,

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highlighting differences in motor involvement [43]. On needle EMG, peroneus longus was abnormal in 92 percent of L5 radiculopathy compared to 64 percent of peroneal neuropathy, reflecting differences in muscle involvement based on lesion location [43]. Together, these findings show that no single parameter is sufficient, and that combining NCS and EMG is necessary to accurately distinguish peroneal neuropathy from radiculopathy.

Plexopathy

Plexopathies represent postganglionic lesions and are distinguished from radiculopathy primarily through nerve conduction study findings. SNAPs are reduced or absent in the distribution of the affected trunk or cord because the lesion is distal to the dorsal root ganglion [76]. CMAPs are reduced in the distribution of the affected plexus element [84].

Needle EMG demonstrates denervation that follows a plexus trunk or cord distribution rather than a single root or peripheral nerve pattern [76]. Paraspinal EMG remains normal. This is the critical distinction from radiculopathy, since the posterior primary rami branch off proximal to the plexus [32]. The combination of reduced SNAPs and normal paraspinal EMG strongly favors plexopathy over radiculopathy [76].

Peripheral Neuropathy

Peripheral neuropathies are characterized by diffuse, length-dependent involvement of multiple nerves. Nerve conduction studies demonstrate symmetric reductions in SNAP and CMAP amplitudes, typically affecting distal segments first.

Prior studies have demonstrated that polyneuropathy is present in up to 50 percent of diabetic patients referred for electrodiagnostic evaluation [1]. Kothari et al. [85] showed that EMG may change the diagnosis or management in 24 percent to 43 percent of patients undergoing evaluation for peripheral neuropathy, including those with typical clinical presentations.

Needle EMG may demonstrate distal greater than proximal denervation but is less sensitive than NCS for identifying the overall pattern of involvement. In this setting, NCS can define the presence and distribution of neuropathy, while EMG helps assess severity and identify superimposed conditions such as radiculopathy or focal entrapment neuropathy.

Table 1: The Diagnostic Utility of NCS and EMG alone and in Combination for Neuromuscular Localization. The table provides a summary of the electrodiagnostic findings in radiculopathy, mononeuropathy, plexopathy, and peripheral polyneuropathy, demonstrating how EMG and NCS combined in tandem can improve diagnostic accuracy and guide localization.

Condition	NCS Alone	EMG Alone	Combined NCS + EMG
Radiculopathy	Very low direct sensitivity; SNAPs characteristically normal (localizing value) [62,74]; late responses (H-reflex 56–98% sensitivity for S1) may detect root pathology [69,70]; CMAP reductions can predict radiculopathy (AUC 0.82–0.85) [63].	Primary diagnostic tool; sensitivity 42–77%, specificity 71–85% [31,62] detects myotomal denervation pattern [12]; paraspinal EMG differentiates from plexopathy [32].	Gold standard combination: EMG detects radiculopathy while NCS exclude mimics [31, 33] and confirm preganglionic localization via preserved SNAPs [75]; false-negative rate drops from 33% to 5% with combined approach [50].
Mononeuropathy	Often diagnostic alone; identifies focal conduction block, slowing, or amplitude drop across entrapment site; sensitivity 56–85% and specificity 94–99% for CTS [80,82]; comparative/segmental tests reach 80–90% sensitivity and >95% specificity [80].	Confirms axonal loss distal to lesion; determines severity (fibrillations = axonal loss) [82]; distribution confined to single nerve territory; e.g., peroneal neuropathy findings help distinguish L5 radiculopathy from peripheral nerve lesions [43].	NCS localize the lesion; EMG determines severity and chronicity; combined testing excludes superimposed radiculopathy or polyneuropathy [31, 33]; essential for surgical decision-making [80].
Plexopathy	Critical role: reduced SNAPs confirm postganglionic lesion (distinguishes from radiculopathy) [76]; CMAP reductions follow trunk/cord distribution [84]; can identify pre- vs. postganglionic components in trauma	Identifies denervation in plexus trunk/cord distribution across multiple peripheral nerves [76, 84]; normal paraspinals distinguish from radiculopathy [32, 77]	Essential combination: NCS provide the SNAP data that localizes lesion as postganglionic [76]; EMG maps the distribution and severity [84]; together they distinguish from radiculopathy (preserved SNAPs + abnormal paraspinals) and mononeuropathy (single nerve distribution) [76].
Peripheral polyneuropathy	Most informative component for polyneuropathy diagnosis [7]; identifies axonal vs. demyelinating pathophysiology [1]; symmetric length-dependent SNAP/CMAP reductions [1]; conduction block/temporal dispersion suggest CIDP; NCS are the basis for CIDP diagnostic criteria [7].	Confirms denervation in distal muscles [20]; neurogenic MUAP changes support axonal loss [1]; can identify superimposed focal lesions (radiculopathy, entrapment); may change diagnosis/management in 24–43% of patients [20].	NCS define the neuropathy type and pathophysiology [7]; EMG adds severity assessment and identifies superimposed conditions [1]; combined testing is recommended for atypical presentations (asymmetric, motor-predominant, rapidly progressive) [1,5].

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Discussion

Differentiating radiculopathy from peripheral nerve disorders remains a persistent challenge in clinical practice. As outlined throughout this review, the overlap in symptoms, including pain, paresthesias, and weakness, often makes localization difficult using history and physical exam alone. Imaging further complicates the picture, as structural abnormalities are frequently present in asymptomatic individuals and may not correlate with the true source of symptoms.

Electrodiagnostic testing plays a central role in addressing this gap by providing functional information that cannot be obtained through imaging. Needle electromyography remains the most sensitive modality for detecting radiculopathy, particularly by identifying myotomal patterns of denervation and paraspinal involvement. However, the findings of this review make it clear that EMG alone is not sufficient for accurate localization.

Nerve conduction studies provide critical complementary information, particularly through evaluation of sensory nerve action potentials. One of the most important and consistent findings is that SNAPs are preserved in radiculopathy, reflecting the lesion's preganglionic nature. Mondelli et al. [62] demonstrated that SNAPs are reduced in only 7% of cases, typically in the setting of foraminal involvement affecting the dorsal root ganglion. This reinforces the concept that preserved SNAPs remain among the most reliable features for distinguishing radiculopathy from plexopathy and peripheral nerve disorders.

At the same time, this principle is not absolute. In cases of foraminal or extraforaminal compression, direct involvement of the dorsal root ganglion can lead to reduced SNAP amplitudes, which may mimic postganglionic lesions. Additional work, such as the SNAP amplitude ratio described by Ando et al. [77], demonstrated a sensitivity of 91.3 percent and specificity of 85.7 percent for identifying foraminal lesions, suggesting that nerve conduction studies can provide more nuanced localization when interpreted carefully.

Late response studies further extend the diagnostic role of nerve conduction studies. Standard tibial H-reflex testing shows modest sensitivity for S1 radiculopathy, whereas more advanced techniques significantly improve detection. Jin et al demonstrated abnormalities in 54 of 55 patients using S1 foramen stimulation compared to 39 of 55 with conventional testing, while Zheng et al. [70] showed improved sensitivity of 91.0 percent compared to 63.6 percent in diabetic patients. These findings emphasize that nerve conduction studies are not limited to excluding alternative diagnoses, but can directly contribute to identifying proximal pathology when appropriate techniques are used.

The combined use of electromyography and nerve conduction studies becomes even more important in conditions such as mononeuropathy and plexopathy. In focal entrapment neuropathies, nerve conduction studies are often diagnostic on their own, with high sensitivity and specificity, as in carpal tunnel syndrome. However, in more complex presentations, such as foot drop, no single parameter is sufficient. Fidancı et al demonstrated that superficial peroneal SNAP abnormalities were present in 21% of L5 radiculopathy, 26 percent of peroneal neuropathy, and 76% of sciatic neuropathy, highlighting the degree of overlap [43]. At the same time, EMG findings differed significantly, with peroneus longus involvement seen in 92% of radiculopathy cases compared to 64% in peroneal neuropathy. These findings reinforce that accurate localization depends on integrating both modalities rather than relying on any single test.

In diffuse conditions such as polyneuropathy, nerve conduction studies play an even more dominant role. They identify length-dependent patterns of involvement, with symmetric reductions in SNAP and CMAP amplitudes. Given that polyneuropathy may be present in up to 50% of diabetic patients undergoing evaluation, this distinction is clinically important. Kothari et al. [85] further demonstrated that electrodiagnostic testing can change the diagnosis or management in 24% to 43% of patients, highlighting its practical impact.

Despite these strengths, electrodiagnostic testing has important limitations that must be considered when interpreting results. EMG cannot diagnose pure sensory radiculopathies, as it only measures motor unit activity. This is due to the location of the lesion proximal to the dorsal root ganglion, where the sensory neuron cell body remains intact. As a result, the distal sensory fibers are preserved and will appear normal on nerve conduction studies, even in the presence of clinical symptoms. This represents an important limitation of current electrodiagnostic tools that cannot be addressed with existing techniques [86].

Similarly, nerve conduction studies have limitations in detecting small fiber neuropathies. NCS primarily evaluates large-diameter, heavily myelinated fibers, while small fibers are not adequately assessed. Larger fibers have lower activation thresholds and produce larger electrical signals, which makes it difficult for the study to capture dysfunction in smaller nerve fibers. This limitation is clinically relevant, as patients with small fiber neuropathy may present with significant symptoms despite normal electrodiagnostic findings [87].

Taken together, these findings reinforce a central conclusion of this review. Electromyography and nerve conduction studies should not be viewed as independent tests, but as complementary tools that provide different pieces of the same diagnostic picture. When used together, they improve

diagnostic accuracy, guide management decisions, and help avoid misclassification in complex clinical presentations.

Future Directions

Although electrodiagnostic imaging remains the primary diagnostic tool for radiculopathy, it has several limitations, including difficulty detecting pure sensory radiculopathies, poor correlation between imaging abnormalities and symptoms, and variability in electrodiagnostic testing depending on disease timing and severity. The aforementioned limitations have resulted in investigations into newer studies that could help further confirm the diagnosis of radiculopathy.

New modalities are being actively explored to confirm the diagnosis of radiculopathy, including imaging and new electrodiagnostic testing. One emerging modality is MR neurography (MRN), a scan that analyzes peripheral nerves and the surrounding tissues in significantly greater detail than a standard MRI. MRN is currently the most actively explored imaging option. The test assesses for nerve inflammation, compression, entrapment, and signal abnormalities to narrow down the diagnosis of radiculopathy. Sequences used include Short Tau Inversion Recovery (STIR) and T2 fat-suppressed imaging. One study found that 283 of 356 patients (79.8%) with lumbosacral radiculopathy had "definitely contributory" findings on MR neurography, which helped further confirm the diagnosis. The findings of the MRN led to a change in management in 219 of the 356 patients (61.5%) [88]. A subsequent study further modified MRN by combining T2 mapping with MRN, as compressed nerve roots exhibit prolonged T2 relaxation times. Using the ratio of T2 relaxation times from the affected and unaffected sides, respectively, researchers found that larger ratios correlated with increased leg pain severity [89]. This could guide management and determine whether conservative or interventional procedures are more indicated for a patient's lumbosacral radiculopathy. There are still limitations to both studies, including cost, unclear sensitivity and specificity relative to EMG, and interobserver variability in interpreting imaging. Despite this, both studies reveal a potentially useful modality for diagnosing radiculopathy. More studies are needed to further confirm their efficacy.

Other imaging modalities should be considered in diagnosing radiculopathy. Diffusion kurtosis imaging visualizes alterations in cervical nerve root fiber thickness, which could help in better analyzing nerve root compression [20]. Another imaging technique is photon-counting CT, which provides sharper images of the spine. Dual-energy CT enables better evaluation of disc spaces and reduces artifacts from metal objects, making it potentially useful for those who have had spine surgery [20]. These imaging studies share similar limitations as mentioned earlier and warrant further research to determine their efficacy in diagnosing radiculopathy.

A newer electrodiagnostic study is electrical impedance myography, a noninvasive procedure that measures muscle electrical impedance and correlates it with radiculopathy. The study finds that the electrical impedance of muscles was consistently lower in the affected muscles than on the contralateral side [90]. The study reveals a promising tool in further confirming the diagnosis of radiculopathy.

Somatosensory evoked potentials (SSEPs) are another modality that may help improve the diagnostic accuracy, particularly when the EMG is nondiagnostic. SSEPs can assess the integrity of the somatosensory pathway from the peripheral nerve to the cortex and detect abnormalities in patients with radiculopathy that otherwise go undetected with EMG, as the EMG alone can only evaluate motor axon loss. Further Dermatomal SSEPs (DSEPs) can stimulate specific dermatomes and have a high sensitivity for radiculopathy symptoms with the ability to detect sensory radiculopathies that often can go missed [91]. One study by Walk et al. [92] found that SSEPs were abnormal in 84% of patients with myelographic evidence of root compression, compared to only 29% that were detected with an EMG alone. Another study by Vallat et al. [93] showed that SSEP may also help assess the proximal sensory nerve involvement in conditions such as CIDP, where conventional NCS may not adequately evaluate the nerve root [93]. However, an important limitation of SSEP findings is that it can not inherently localize the pathology to a peripheral versus central source, which must be interpreted in the broader clinical context [87].

Quantitative sensory testing (QST) offers another approach to evaluating thermal and mechanical detection and pain thresholds. In the context of radiculopathy versus peripheral nerve diseases, QST can reveal distinct somatosensory profiles. For example a patient with cervical radiculopathy can demonstrate localized loss of function, such as thermal, mechanical, vibrational detection, within the affected dermatome [94]. In lumbar radiculopathy, combinations of pressure pain threshold, mechanical detection threshold, numbness, and prickling have shown good predictive value in distinguishing radicular from non radicular pain [95]. QST is also particularly useful in identifying small fiber neuropathy, which cannot be detected using standard nerve conduction studies or electromyography. However, similar to somatosensory evoked potentials, abnormal QST findings do not independently localize pathology to a peripheral or central source and must be interpreted within the broader clinical context [87, 96].

The new modalities show promise, however, none can definitively provide a clinician with a confirmatory diagnosis [97]. A combination of imaging, electrodiagnostic studies, and clinical suspicion helps narrow the diagnosis to radiculopathy. Future research could potentially focus on integrating advanced imaging techniques or adjunct

electrodiagnostic studies to improve diagnostic accuracy. Development of multimodal algorithms could potentially also assist in standardizing findings that could lead to the diagnosis of radiculopathy and pain management [97]. It is worth mentioning that machine learning and artificial intelligence could further reduce interobserver variability and help to identify nerve compression with greater accuracy. Ultimately, large-scale prospective studies are needed to determine the sensitivity, specificity, and clinical utility of these emerging modalities before adoption into standard of care.

Key Points

The optimal timing for electrodiagnostic evaluation of radiculopathy is 3–4 weeks after symptom onset; EMG performed earlier may yield false-negative results, as fibrillation potentials and positive sharp waves require time to develop via Wallerian degeneration, with paraspinal muscles showing changes as early as 10 days and distal limb muscles changing last.

The key localizing principle of needle EMG in radiculopathy is demonstrating spontaneous activity in two or more muscles sharing the same myotomal innervation but supplied by different peripheral nerves. For example, L5 radiculopathy produces denervation in both the tibialis anterior (deep peroneal nerve) and tibialis posterior (tibial nerve), whereas peroneal neuropathy spares the tibialis posterior entirely.

Paraspinal EMG alone carries a false-negative rate of approximately 33%, and S1 radiculopathy is a notable exception where paraspinal mapping may be entirely normal; combining paraspinal with lower extremity EMG reduces the false-negative rate to 5% and provides the strongest electrodiagnostic evidence for root-level pathology.

Incorporating motor unit action potential (MUAP) changes alongside fibrillation potentials and PSWs substantially improves radiculopathy detection: adding a threshold of ≥30% MUAP changes increased lumbar radiculopathy detection from 27.1% to 45.8% (P = .002) while maintaining specificity, highlighting the risk of underdiagnosis when relying on spontaneous activity alone.

Preserved sensory nerve action potentials (SNAPs) are the most reliable NCS feature distinguishing radiculopathy from plexopathy and peripheral nerve disorders. SNAPs remain normal because the sensory cell body in the dorsal root ganglion and its distal axon are intact despite proximal root compression. Clinically apparent sensory deficits can therefore coexist with entirely normal sensory NCS in radiculopathy.

Severe foraminal and extraforaminal disc herniations can directly compress the dorsal root ganglion, producing reduced SNAP amplitudes that mimic a postganglionic lesion. The SNAP amplitude ratio (affected/unaffected side) can identify these foraminal lesions with a sensitivity of 91.3% and specificity of 85.7% using a cutoff of 0.5, helping distinguish foraminal from intraspinal canal pathology.

- F-wave chronodispersion is the difference between maximum and minimum F-wave latencies, reflecting nonuniform conduction along motor axons, and is the most commonly abnormal NCS finding in lumbosacral radiculopathy. Repeater F-waves, indicating that fewer motor neurons are firing, are also seen in radiculopathy and can provide additional diagnostic information beyond standard latency measurements.
- Advanced H-reflex techniques significantly outperform conventional tibial H-reflex testing for S1 radiculopathy: S1 foramen stimulation achieves sensitivity of 98% compared to 50–72% with standard testing, and retains superior sensitivity (91% vs. 63.6%) even in diabetic patients where concurrent polyneuropathy may mask unilateral root pathology.
- In the evaluation of foot drop, no single electrodiagnostic parameter reliably distinguishes L5 radiculopathy from peroneal neuropathy or sciatic neuropathy. Superficial peroneal SNAP abnormalities are present in 21% of L5 radiculopathy, 26% of peroneal neuropathy, and 76% of sciatic neuropathy; peroneus longus denervation on EMG is seen in 92% of L5 radiculopathy versus 64% of peroneal neuropathy, underscoring the need to integrate both NCS and EMG findings.
- EMG-confirmed radiculopathy carries direct prognostic and therapeutic value: patients with positive baseline electrodiagnostic studies show greater clinical improvement over time, and EMG confirmation has been associated with better outcomes following conservative management, epidural corticosteroid injections, and spine surgery, making electrodiagnostic testing relevant not only for diagnosis but for guiding treatment selection.

Authors' Contribution:

A.A. conceptualized the project; A.A., S.A. performed literature search and selected articles; A.A., S.A., and A.Y. data collection and analysis; A.A., S.A., and A.Y. wrote the initial draft of the manuscript and prepared all figures and tables; A.N. and D.K.A. critically reviewed and edited the manuscript; D.K.A. provided research funds and resources. All authors reviewed and approved the manuscript for submission to the Journal of Neurology.

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Data Availability:

No new studies were performed and all findings were gathered from published articles and critically reviewed. All collected findings and their analyses are deposited in the Cloud provided by WesternU and are also available with the corresponding author upon request through the proper channel.

Competing interests:

All authors have read the manuscript and declare no conflict of interest. No writing assistance was utilized in the production of this manuscript.

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