A Day in the EMG Laboratory: Case Studies of 10 Patients with Different Clinical Problems

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KEYWORDS

• Electromyography • Nerve conduction studies • Algorithm
• Neuromuscular disorders

Other articles in this issue have reviewed basic concepts of nerve conduction studies (NCSs) and needle electromyography (EMG), and have detailed the electrodiagnostic features and approaches that are used to evaluate different types of neuromuscular disorders. This article discusses 10 representative case vignettes that may be encountered during a day in the EMG laboratory, which demonstrate the approaches used in our EMG laboratory to evaluate patients presenting with specific symptoms and a variety of suspected neuromuscular conditions. Each case presents a brief description of the patient's symptoms and clinical findings, suggests the suspected localization or diagnosis that was considered based on the clinical features before the performance of the electrodiagnostic study, and then presents the NCS and needle EMG data that were actually gathered from that patient. Comments and instructive electrodiagnostic considerations as they relate to each case are discussed at the end of the case. Although it would be uncommon to encounter all of these patients in a single day in the EMG laboratory, it would surely be an interesting and educational workday.

CASE 1. A HOSPITAL VOLUNTEER WITH HAND NUMBNESS

Clinical History

A 79-year-old woman, who worked as a volunteer at the information desk of our hospital, complained of numbness and paresthesias in her right thumb and index finger. Her symptoms were constant, but worse when she would drive to work in the morning and during the night when she was trying to sleep. She noted that rubbing

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or shaking her hand improved the symptoms slightly. She denied weakness; however, she reported dropping objects that she was holding in her hand on occasion. She also reported some achiness of her shoulder and entire arm, and occasional mild neck stiffness. She did not experience any similar symptoms in her left hand.

Physical Examination

The pertinent neurologic examination findings were decreased sensation to pinprick on the flexor surface of the thumb, index, and middle fingers. There was no weakness or atrophy noted in the right thenar or other arm muscles. Reflexes were normal and symmetric in her upper extremities.

Differential Diagnosis

The clinical features were most suspicious for a right median neuropathy at the wrist (carpal tunnel syndrome [CTS]). However, other localizations that may present with similar features and should be considered include a C6-C7 radiculopathy (especially given some neck and arm discomfort), a proximal median neuropathy, or, less likely, a brachial plexopathy.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 1** and **2**. The NCSs demonstrated prolonged right median motor and sensory distal latencies and a low median sensory amplitude with a mildly slowed conduction velocity. Needle EMG was normal. The findings indicate a moderately severe right median neuropathy at the wrist (CTS).

Case Comment

This case demonstrates typical features of a median neuropathy at the wrist, such as occurs in CTS, with conduction slowing identified in motor and sensory fibers in the distal median nerve across the wrist. In this case, motor NCSs were performed first and because the median motor fibers demonstrated a prolonged distal latency, the antidromic sensory techniques were selected for the sensory studies. Had the median motor NCSs been completely normal, the orthodromic (palmar) sensory studies or other comparison studies (median–radial to thumb or median–ulnar to ring finger) would have been performed to increase the sensitivity of identifying a very mild distal median neuropathy. The needle examination consisted of evaluation of muscles supplied by the median nerve as well as those innervated by the C5 through T1 roots to exclude a superimposed cervical radiculopathy (although a cervical radiculopathy at any level would not affect the median sensory NCSs). Given the normal median

Table 1
Case 1: nerve conduction studies

	Amplitude (mV or μV)			Velocity (m/s)			Distal Latency (ms)			F-Wave Latency (ms)		
Stimulate (Record)	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**Est**
Ulnar, m (hypothenar)	12.7		>6	55		>51	3.0		<3.6	27		24.8
Ulnar, s anti (fifth)	33		>10	60		>54	3.0		<3.1			
Median, m (thenar)	7.7	8	>4	51	58	>48	5.1	3.4	<4.5	27.3	28.0	28.6
Median, s anti (index)	10	20	>15	51	66	>56	4.6	3.0	<3.6			

Abbreviations: Est, estimate; NL, normal values.

Table 2
Case 1: needle examination of the right upper extremity

Muscle	Insertional Activity	Fibrillation Potentials	MUP
Deltoid	NL	0	NL
Biceps	NL	0	NL
Triceps	NL	0	NL
Pronator teres	NL	0	NL
Abductor pollicis brevis	NL	0	NL
Flexor pollicis longus	NL	0	NL
First dorsal interosseous	NL	0	NL

Abbreviations: MUP, motor unit potential; NL, normal.

compound muscle action potential (CMAP) amplitude, the yield of identifying significant abnormalities in the abductor pollicis brevis was relatively low; however, because the median motor NCS was not entirely normal, the muscle was examined in this case.[1]

The study was interpreted as showing a moderately severe median neuropathy. Different grading scales are used to grade the severity of median neuropathies at the wrist: mild cases typically demonstrate only sensory NCS abnormalities, whereas moderately severe cases are those in which there is slowing of conduction in median motor fibers across the wrist without a loss of amplitude, and severe cases have reduction in the motor amplitude. Documenting an electrophysiologic grade of the degree of the median neuropathy at the wrist is useful in the EMG report, because it may guide the referring physician's decision on treatment. However, the degree of electrodiagnostic abnormalities and the clinical symptoms may not always correlate well. Although this patient did not have any symptoms in her left hand, sensory NCSs were performed to assess for subclinical involvement of her left hand because CTS is frequently bilateral. This patient did not have a median neuropathy on the left.

CASE 2. A COMPUTER PROGRAMMER WITH A NUMB PINKY AND WEAK HAND

Clinical History

A 31-year-old man who works as a computer programmer presented with numbness of his left fifth (pinky) digit. He noted that he frequently would rest his elbow on a pad at his desk in an effort to keep his wrists in line while typing, to avoid developing CTS. By the end of his workday, he noticed a tingling radiating down the medial aspect of his forearm into his left ring and pinky finger. This progressed to persistent numbness involving his left fifth digit specifically. He also complained of occasional elbow discomfort. He denied any weakness in his hand or arm.

Physical Examination

The pertinent findings included decreased sensation to pinprick over the medial aspect of the fourth and fifth digits of the left hand. There was equivocal weakness but no atrophy in his hypothenar and interosseous muscles. Reflexes were normal and symmetric.

Differential Diagnosis

The clinical history suggests a left ulnar neuropathy, probably localized at the elbow given his history of resting his elbow on a pad while at work. Additional considerations

include a distal ulnar neuropathy at the wrist, a C8 or T1 radiculopathy, or a brachial plexus lesion involving the lower trunk or medial cord.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 3** and **4**. NCSs demonstrated low left ulnar sensory (antidromic) nerve amplitude. With elbow stimulation, no reliable response was recorded and, therefore, conduction velocity could not be determined. Short segmental incremental stimulation (inching) along the left ulnar nerve showed a partial motor conduction block of 28% in amplitude and 30% in the area localized adjacent to the medial epicondyle (**Fig. 1**). Needle examination demonstrated only reduced recruitment in the first dorsal interosseous and abductor digiti minimi. No fibrillation potentials or other motor unit potential (MUP) morphologic changes were seen. There is electrodiagnostic evidence of a mild left ulnar neuropathy in the region of the elbow that is localized to the medial epicondyle, characterized primarily by focal demyelination.

Case Comment

This case demonstrates typical features of an ulnar neuropathy at the elbow. In this patient the sensory NCSs demonstrated low ulnar sensory amplitudes, which supports axonal degeneration of the ulnar sensory fibers. However, in isolation this finding could be seen with an ulnar neuropathy localized proximal to the wrist, but could also be seen with a lower trunk or medial cord plexopathy. In this case, palmar studies were also performed to assess for slowing across the wrist, as may be seen in a distal ulnar neuropathy at the Guyon canal. If there was a high index of suspicion for a lesion at the wrist, the dorsal ulnar cutaneous sensory study would have been useful because it would be normal in an ulnar nerve lesion at the wrist and abnormal (low amplitude) in a lesion at the elbow.

The standard ulnar motor NCS, with stimulation at the wrist and above the elbow, was normal, including the conduction velocity across the elbow, making it difficult to precisely localize the process to the ulnar nerve at the elbow. However, this case demonstrates the value of inching along the ulnar nerve in 2-cm segments across the elbow to assist in more precise localization of a mild ulnar neuropathy characterized by very focal demyelination. The inching study demonstrated a segment

Table 3
Case 2: nerve conduction studies of the left upper extremity

Stimulate (Record)	Amplitude (mV or μV) (Normal)	Velocity (m/s) (Normal)	Distal Latency (ms) (Normal)	F-Wave Latency (ms) (Estimate)
Ulnar, m (hypothenar)	8.8 (>6)	53 (>51)	2.5 (<3.6)	30 (30)
Ulnar, s anti (fifth)	7 (>10)	NR (>54)	2.7 (<3.1)	
Ulnar, s palmar (fifth)	13 (>15)	NR (>54)	1.9 (<2.3)	
Median, m (thenar)	7.7 (>4)	58 (>48)	3.4 (<4.5)	28 (28.6)
Median, s palmar (wrist)	197 (50)	66 (>55)	1.9 (<2.3)	

Abbreviation: NR, no response obtained.

Table 4
Case 2: needle examination of the left upper extremity

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Recruitment (Reduced)
Deltoid	NL	0	NL	
Biceps	NL	0	NL	
Triceps	NL	0	NL	
Pronator teres	NL	0	NL	
Extensor indicis	NL	0	NL	
Flexor digitorum profundus III and IV	NL	0	NL	
First dorsal interosseous	NL	0	NL Duration	1+
Flexor carpi ulnaris	NL	0	NL Duration	
Abductor digiti minimi	NL	0	NL Duration	1+

Abbreviation: NL, normal findings.

where a definite conduction block (>10% over a 2-cm segment) and a focal shift in latency (1.4 milliseconds in this case) were identified between two sites of stimulation (see **Fig. 1**). This finding, when present, provides localizing value in identifying the precise site of compression or injury to the ulnar nerve.

In this case, the needle examination only demonstrated subtle abnormalities (reduced recruitment) in ulnar innervated hand muscles. As the only finding, the reduced recruitment would be compatible with underlying pathophysiology of

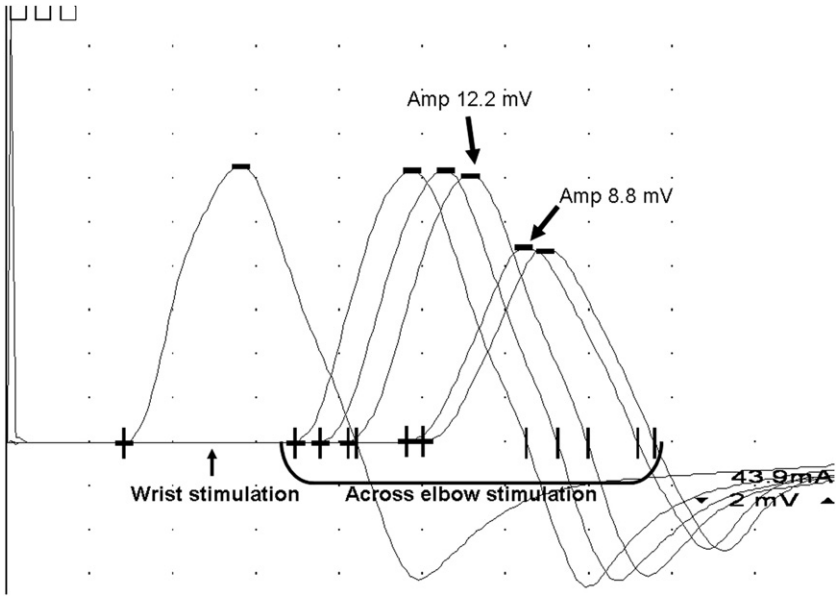

Fig. 1. Ulnar motor inching study in case 2, demonstrating a partial focal conduction block at the medial epicondyle. The responses at each stimulation site are superimposed. The first response on the left is stimulation at the wrist. The remaining responses occur from stimulation around the elbow in 2-cm increments.

focal demyelination and partial conduction block without evidence of axonal loss (in which fibrillation potentials or long-duration MUPs would be expected). Other, nonulnar C8-T1 muscles, such as the abductor pollicis brevis and extensor indicis proprius, were normal and helped to exclude those localizations. In this case, and in some ulnar neuropathies at the elbow, the proximal ulnar muscles (flexor carpi ulnaris and flexor digitorum profundus) were normal. These muscles may have been spared because the branches to those muscles often exit the ulnar nerve proximal to the elbow (and thus proximal to the lesion) or because the nerve fascicles to those muscles may have been preferentially spared. In cases such as this where the pathologic changes suggest focal demyelination, the prognosis is generally favorable if the offending cause of the ulnar neuropathy is eliminated.

CASE 3. A CANCER PATIENT WITH TINGLING TOES

Clinical History

An 18-year-old woman with a history of cancer presented with decreased sensation in her feet. Her symptoms began shortly after the completion of a course of chemotherapy, which included vincristine and cyclophosphamide. She reported decreased sensation in both feet and described a feeling of "walking on staples." She felt generally weaker in her arms and legs. She denied any pain in her extremities.

Physical Examination

The pertinent neurologic examination abnormalities included decreased sensation to pinprick, temperature, vibration, and joint position in both feet up to the level of the ankles bilaterally. Reflexes were diminished in her upper limbs and absent in her quadriceps and ankles bilaterally. Strength testing revealed bilateral weakness of foot dorsiflexion, eversion, inversion, and plantar flexion (Medical Research Council [MRC] grade 4/5). Her Romberg sign was present and her gait examination was notable for a sensory ataxia.

Differential Diagnosis

Given the temporal relationship between the patient's chemotherapy and development of symptoms, the most likely diagnosis is a chemotherapy-associated, length-dependent, peripheral neuropathy involving sensory and motor fibers. Another possibility is a polyradiculoneuropathy or sensory ganglionopathy (which would be less likely with true weakness). In addition, other unlikely considerations are bilateral lumbosacral radiculopathies or lumbosacral plexopathies.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 5** and **6**. NCSs demonstrated absent sensory responses diffusely, an absent peroneal motor response, low tibial motor amplitude with a slowed conduction velocity, and a slowed median motor conduction velocity with a prolonged distal latency. Needle examination demonstrated mild long-duration MUPs in the anterior tibialis. The findings are those of a severe, length-dependent, large-fiber, peripheral neuropathy involving mainly sensory fibers, with milder involvement of motor fibers.

Case Comment

In this case a peripheral neuropathy was suspected, based on the clinical history of having received chemotherapeutic agents known to be associated with a peripheral neuropathy, and the typical clinical examination findings of distal sensory loss, hyporeflexia, mild distal weakness, and sensory ataxia. NCSs and EMG were performed to

Table 5
Case 3: nerve conduction studies

Stimulate (Record)	Amplitude (mV or μV) (Normal)	Velocity (m/s) (Normal)	Distal Latency (ms) (Normal)	F-Wave Latency (ms) (Estimate)
Ulnar, m (hypothenar)	7.2 (>6)	53 (>51)	2.8 (<3.6)	31.4 (23.8)
Ulnar, s anti (fifth)	NR	NR	NR	
Radial, s (wrist)	NR		NR	
Median, m (thenar)	8.7 (>4)	48 (>48)	5.2 (<4.5)	28 (28.6)
Median, s anti (index)	NR	NR	NR	
Peroneal, m (extensor digitorum brevis)	NR	NR	NR	
Sup. Peroneal, s (ankle)	NR		NR	
Tibial, m (abductor hallucis)	3.1 (>4)	32 (>40)	4.7 (<6.1)	66.8 (73.2)
Plantar, medial, s (ankle)	NR		NR	

Abbreviation: NR, no response.

characterize the distribution of involvement, differentiate between sensory and motor components, differentiate between axonal degeneration or demyelination as the primary pathologic changes, exclude multiple radiculopathies or a plexopathy, and assess severity.

The most prominent finding on NCSs was the diffusely absent sensory responses. This finding can be seen in a diffuse severe sensory neuropathy or sensory ganglionopathy, which is difficult to distinguish on an electrophysiologic basis. The performance of blink reflexes may help, although absent blink reflex responses can be seen in either process. Some have advocated using the masseter (jaw-jerk) response in situations in which all sensory responses, including blink reflexes, are absent (see the article on Cranial Neuropathies by Lacomis elsewhere in this issue). Because the jaw-jerk reflex is a monosynaptic reflex with the mesencephalic ganglion located within the brainstem rather than extramedullary, the response should be preserved in ganglionopathies but be abnormal in sensory neuropathies. However, this reflex is technically difficult to elicit in some normal people, so interpretation should be made with caution.

Table 6
Case 3: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Duration (Long)	Amplitude (High)
First dorsal interosseous	NL	0	NL		
Tensor fascia lata	NL	0	NL		
Vastus medialis	NL	0		±	±
Gluteus maximus	NL	0	NL		
Tibialis anterior	NL	0		±	±
Medial gastrocnemius	NL	0	NL		

Abbreviation: NL, normal.

In this case the motor conduction studies were mildly abnormal, indicating that the neuropathy involved motor fibers to some degree in addition to the more severely affected sensory fibers. The tibial and median motor conduction velocities were mildly slowed but not in the range of definite demyelination. To be confident that the neuropathy is primarily due to demyelination in the case of a low tibial motor amplitude, the conduction velocity should be less than 50% of the lower limit of normal, which in this case was not. In addition, the median motor distal latency was prolonged with a normal amplitude, suggesting that there may be a possible superimposed median neuropathy at the wrist.

The NCS findings could also be seen in a diffuse, patchy polyradiculoneuropathy. The needle examination demonstrated only mild abnormalities in the distal leg muscles, and the proximal leg muscles were normal. Examination of proximal muscles is important to exclude a polyradiculopathy, in which distal and proximal muscles supplied by the same roots would be expected to be abnormal. In this case, the discrepancy between the moderately severe lower extremity motor NCS abnormalities and the very mild needle examination abnormalities in the distal leg muscles may reflect the possibility that only the very distal motor fibers to the distal foot muscles were predominantly affected. Needle examination was not performed in foot muscles, such as the abductor hallucis or first dorsal interosseous pedis in this case; had those muscles been examined, they may have shown more significant abnormalities than the leg muscles. The features of this case are typical for chemotherapy-induced peripheral neuropathy.

CASE 4. A WOMAN WITH PAINLESS, PROXIMAL WEAKNESS

Clinical History

A 48-year-old woman presented with a 1-year history of progressive, painless, proximal muscle weakness. She described difficulty walking around her block, which she had done regularly for years. She noted weakness with climbing stairs, and difficulty raising her arms over her head while blow-drying her hair. She reported no speech or swallowing impairment and did not experience double vision or drooping of her eyelids. She denied muscle tenderness or pain and had no systemic symptoms, such as skin abnormalities or pulmonary complaints. Bowel and bladder function was also normal.

Physical Examination

The findings were notable for symmetric, moderately severe weakness of proximal shoulder muscles, hip flexors and abductors, and foot dorsiflexors bilaterally. Sensory examination was normal. Reflexes were normal diffusely.

Differential Diagnosis

The clinical possibilities in this case include a myopathy, a disorder of neuromuscular junction transmission, such as myasthenia gravis or Lambert-Eaton myasthenic syndrome (LEMS), or polyradiculopathy (such as chronic inflammatory demyelinating polyradiculopathy).

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 7** and **8**. NCSs of the left upper and lower extremities were normal. Needle examination demonstrated fibrillation potentials and short-duration MUPs with rapid recruitment in several proximal muscles. There was decreased insertional activity and markedly

Table 7
Case 4: nerve conduction studies of the left side

Stimulate (Record)	Amplitude (mV or μV) (Normal)	Velocity (m/s) (Normal)	Distal Latency (ms) (Normal)	F-Wave Latency (ms) (Estimate)
Ulnar, m (hypothenar)[a]	8 (>6)	77 (>51)	2.6 (<3.6)	26.2 (20.2)
Median, s anti (index)	16 (>15)	63 (>56)	3 (<3.6)	
Peroneal, m (extensor digitorum brevis)	3.6 (>2)	46 (>41)	4 (<6.6)	51.6 (49.6)
Tibial, m (abductor hallucis)	5.2 (>4)	51 (>40)	4.3 (<6.1)	55.6 (45.4)
Sural, s (malleolus)	4 (>0)	47 (>40)	3.9 (<4.5)	

[a] Repetitive stimulation at 2 Hz (no decrement).

reduced recruitment of short-duration MUPs in the anterior tibialis. The findings were consistent with a patchy myopathy, mainly involving lower extremity muscles.

Case Comment

The electrodiagnostic approach to this patient began with standard motor and sensory conduction studies in the legs, which were most clinically affected, as well as screening conduction studies in an arm. The normal findings on the NCSs would make a polyradiculopathy less likely, but could be seen in myopathies and neuromuscular junction disorders. Repetitive stimulation studies were performed on the ulnar nerve, using 2-Hz stimulation at rest, to screen for a neuromuscular junction disorder. Had the index of suspicion for a neuromuscular junction disorder been high or had the routine needle examination demonstrated marked MUP variation, repetitive stimulation of additional proximal nerves, such as the spinal accessory nerve, would have been performed.

Her needle examination consisted of examination of moderately weak muscles and demonstrated changes typical for myopathy, including short-duration, low-amplitude, polyphasic MUPs with rapid recruitment in proximal muscles. The findings of reduced recruitment (along with short-duration MUPs) in the anterior tibialis were consistent with end-stage myopathic changes. The presence of fibrillation potentials suggests underlying pathologic changes of muscle fiber necrosis or splitting, or vacuolar

Table 8
Case 4: needle examination of the left side

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Recruitment	Duration	Amplitude	Phases
Deltoid	Increased	1+		NL	1+ short	1+ low	25%
Biceps	NL	0	NL		±		
First dorsal interosseous	NL	0	NL				
Vastus medialis	Increased	2+		Rapid 1+	2+ short	2+ low	25%
Tensor fascia lata	Increased	1+		Rapid 1+	1+ short	1+ low	50%
Tibialis anterior	Decreased	0	NL	Reduced 2+	3+ short	3+ low	
T10 paraspinal	Increased	1+	0	Rapid 1+	1+ short	1+ low	

Abbreviation: NL, normal.

damage to muscle fibers. These findings would raise the possibility of an inflammatory myopathy, such as polymyositis, although many other types of myopathies can produce similar findings on EMG (see the article on Myopathies elsewhere in this issue). Although neuromuscular junction disorders can occasionally be associated with fibrillation potentials and mild short-duration MUPs in some muscles, the findings in this patient are much more pronounced than what would be expected in myasthenia gravis or LEMS, and the rapid recruitment is more compatible with a myopathy.

This patient underwent a muscle biopsy of her deltoid, the findings of which were consistent with polymyositis.

CASE 5. A GOLFER WITH NECK PAIN

Clinical History

A 74-year-old man presented with a 6-week history of neck pain and intermittent sharp pain that radiated down his left arm into the middle finger. His symptoms began approximately 6 weeks previously, after playing in a golf tournament. He had a previous history of occasional neck pain that would last for several weeks at a time but never radiated down his arm. He reported a vague sense of heaviness in his arm and described some numbness in his hand.

Physical Examination

The pertinent findings included mild weakness of elbow and wrist extension, and a depressed left triceps muscle deep tendon reflex. Neck movement to the left caused pain to radiate from the neck, down the arm, and into the middle finger (positive Spurling sign). Placing the left arm over the head relieved his symptoms temporarily. Sensory examination was normal.

Differential Diagnosis

In this case the distribution of the patient's subjective symptoms and findings on his neurologic examination, as well as the reproducibility of his symptoms with neck movement, suggested that the most likely diagnosis was a left C7 radiculopathy. Other less likely possibilities included a posterior cord or middle trunk brachial plexus lesion or a radial neuropathy.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 9** and **10**. The NCSs of the left upper extremity were normal. Needle examination demonstrated a mild degree of increased insertional activity and fibrillations in left C7 innervated muscles. There was increased polyphasia and mild long-duration,

Table 9
Case 5: nerve conduction studies

Stimulate (Record)	Amplitude (mV or μV) (Normal)	Velocity (m/s) (Normal)	Distal Latency (ms) (Normal)	F-Wave Latency (ms) (Estimate)
Ulnar, m (hypothenar)	7.6 (>6)	55 (>51)	3.2 (<3.6)	31.9 (27.7)
Ulnar, s anti (fifth)	13 (>10)	56 (>54)	2.8 (<3.1)	
Median, m (thenar)	10.0 (>4)	50 (>48)	3.6 (<4.5)	28.1 (30.2)
Median, s anti (index)	33 (>15)	60 (>56)	3 (<3.6)	

Table 10
Case 5: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Recruitment (Reduced)	Duration (Long)	Amplitude (High)	Phases
Deltoid	NL	0	NL				
Biceps	NL	0	NL				
Triceps	Increased	1+		1+	±	±	75%
Extensor indicis proprius	NL	0	NL				
Pronator teres	Increased	±		1+	±	±	50%
Extensor carpi radialis	Increased	±		1+	±	±	50%
First dorsal interosseous	NL	0	NL				
C7 paraspinal	Increased	1+		1+			75%

Abbreviation: NL, normal.

high-amplitude MUPs in the C7 distribution. The findings were consistent with a subacute, active, left C7 radiculopathy.

Case Comment

In this case, the clinical features were very typical of a cervical radiculopathy. Because the most frequently affected root in a cervical radiculopathy is the C7 root and also because the clinical findings fit within this root distribution, it might be argued that the performance of electrodiagnostic testing provides little additional information and that an EMG could be bypassed in place of imaging studies. Although in certain cases this approach would not be incorrect, EMG does have utility and may provide complementary information to imaging studies regarding the problem. The goal of electrodiagnostic studies in the evaluation of a suspected cervical radiculopathy is not only to confirm that the process is at the root level and to localize which root(s) is (are) involved, but also to assess severity and activity (ie, whether there is denervation of the muscles innervated by the root).

In this case routine motor NCSs (median and ulnar) were performed and were normal, which would be expected in all cervical radiculopathies outside of the C8 or T1 distribution. In patients with suspected C7 (or C6) radiculopathies, other more proximal motor NCSs, such as the radial (extensor digitorum communis recording) in C7 radiculopathies or musculocutaneous (biceps recording) in C6 radiculopathies, could be performed. However, the more proximal NCSs are more technically challenging and would typically still be normal unless there was significant axonal loss. Therefore, these conduction studies are not routinely performed and the C5 to C7 roots are mostly assessed by needle EMG. The F-wave latencies may occasionally be prolonged in root disorders (or plexus lesions), although the median and ulnar F waves again only assess conduction through the C8 and T1 roots.

The sensory NCSs may perhaps be more useful than motor NCSs when evaluating patients with suspected cervical radiculopathies. The sensory responses should always be normal in cervical radiculopathies, because the roots are typically injured proximal to the dorsal root ganglia, thereby sparing the distal axons in the arm. In this case, the median sensory (antidromic) study assessed the sensory pathway through the median nerve, lateral cord and upper/middle trunk of the plexus, and

through the C6/7 root, whereas the ulnar antidromic sensory study assessed the pathway through the ulnar nerve, lower trunk, medial cord, and C8 root. Had either of these been abnormal, a lesion involving the distal nerve or brachial plexus would have been considered. In this case, no radial NCSs were performed. Although radial neuropathy was in the differential diagnosis given the distribution of weakness, it was not strongly suspected because the patient had significant neck pain. Had the index of suspicion for a radial neuropathy been high, radial motor and sensory NCSs would have been performed.

Needle EMG is typically the most sensitive and useful component of the evaluation in cervical radiculopathies. In this case the focus was on the muscles supplied by the C7 root (triceps, pronator teres, extensor carpi radialis, and C7 cervical paraspinals), but muscles innervated by other roots were also examined to help define the localization. The C7 innervated muscles demonstrated fibrillation potentials, which indicated denervated muscle fibers, and often implies an active radiculopathy (in which there is either denervation from an ongoing process or a process that is resolving but in which reinnervation has not yet occurred). The MUP abnormalities were mainly an increased percentage of polyphasic MUPs with only minimally increased duration. This pattern of MUP changes occurs early in reinnervation, usually after about a month, and is helpful in defining the temporal profile of the process, which was subacute in this case. Although the findings in the limb muscles could also have been seen with a middle-trunk brachial plexopathy, the presence of abnormalities in the cervical paraspinals and the absence of an abnormal median sensory NCS confirm the process at the root level.

CASE 6. A TRAVELING BUSINESSMAN WITH LEG PAIN

Clinical History

A 48-year-old business executive who traveled overseas each month presented with a 4-month history of lower back and leg pain. He did not recall any precipitating single event but would experience more pain the week after his trips. The pain was described as a deep ache in his low lumbar region with a deep, achy pain that radiated into his right hip and occasionally down his buttock and posterior thigh to the knee. He denied experiencing any weakness but had noted that he nearly tripped a few times while walking quickly through the airport. The symptoms would improve but did not resolve completely after a week of minimal activity.

Physical Examination

His neurologic examination demonstrated mild weakness of the right foot dorsiflexion and foot eversion, and equivocal weakness of right hip abduction. Sensory testing reveals decreased pinprick sensation of the right lateral leg and dorsum of the foot. Reflexes were normal and symmetric in his lower extremities.

Differential Diagnosis

The clinical history and physical examination findings were most concerning for a right lumbosacral radiculopathy, localized to the L5 or S1 nerve roots. However, a lumbosacral plexopathy, sciatic neuropathy, or peroneal neuropathy was also a consideration.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 11** and **12**. The NCSs demonstrated a low right peroneal CMAP amplitude with a mildly slowed conduction velocity. The peroneal F wave was absent. The superficial peroneal sensory response was present but the amplitude was approximately 50% of

Table 11
Case 6: nerve conduction studies

	Amplitude (mV or μV)			Velocity (m/s)			Distal Latency (ms)			F-Wave Latency (ms)		
Stimulate (Record)	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**Est**
Peroneal, m (extensor digitorum brevis)	1.7	4.2	>2	39	42	>41	5.5	5.3	<6.6	NR		
Superficial peroneal, s (ankle)	5	13	>0				3.8	3.7	<4.1			
Tibial, m (abductor hallucis)	5.9		>4	43		>40	4.5		<6.1			
Sural, s (malleolus)	11		>6	45		>40	3.9		<4.5			

Abbreviations: Est, F-wave estimate; NL, normal values.

the amplitude on the left. Needle EMG demonstrated fibrillation potentials and long-duration MUPs in right L5 innervated muscles. These findings were interpreted as consistent with a subacute to chronic, active right L5 radiculopathy.

Case Comment

This case brings up several important points related to the electrodiagnostic features of a lumbosacral (particularly L5) radiculopathy. The lower extremity motor NCSs demonstrated a low peroneal CMAP amplitude, which was consistent with, but not specific for, an L5 radiculopathy. A low peroneal CMAP can also be seen in a sacral plexus, sciatic nerve, or a peroneal nerve lesion. Given this finding and because the patient had sensory loss on the dorsum of his foot, the superficial peroneal sensory nerve was performed in addition to the sural sensory NCSs. In this case, the superficial sensory response was present but the amplitude was possibly low (slightly less than 50% of the unaffected side). Although a low sensory nerve action potential (SNAP) amplitude typically indicates a postganglionic process, such as a lumbosacral plexopathy, sciatic neuropathy, or peroneal neuropathy, in some individuals the dorsal root ganglion at the L5 level is situated more proximal in the spinal canal, and a lateral disk may directly compress the ganglion rather than the proximal rootlet.[2] Therefore, a low superficial peroneal SNAP can occur with an L5 radiculopathy, complicating the interpretation and localization of the process based on NCSs alone in this case. The fact that the tibial motor and sural responses were normal (although were not

Table 12
Case 6: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Recruitment (Reduced)	Duration (Long)	Amplitude (High)
Vastus medialis	NL	0	NL			
Tensor fascia lata	Increased	1+			1+	1+
Medial gastrocnemius	NL	0	NL			
Tibialis anterior	Increased	±			±	±
Tibialis posterior	Increased	2+		1+	2+	2+
Peroneus longus	Increased	2+		1+	2+	2+
Gluteus maximus	Increased	0	NL			
L5 paraspinal	Increased	1+			± Long	

Abbreviation: NL, normal.

compared with the other side, so could potentially be of low amplitude) would make a sciatic neuropathy or sacral plexopathy less likely. In this patient, the peroneal F waves were absent. Absent peroneal F waves may occur in normal individuals and therefore do not necessarily indicate a proximal nerve or root lesion.

The needle examination included assessment of muscles in the L5, as well as the L4 and S1, distribution. Because the NCS findings raised the possibility of a peroneal neuropathy versus an L5 root, the needle EMG incorporated L5 muscles that were not supplied through the peroneal nerve (such as the posterior tibialis and tensor fascia lata). The needle examination demonstrated fibrillation potentials and long-duration, polyphasic MUPs in L5 innervated muscles. However, each muscle with L5 innervation was not affected to a similar degree. In particular, the anterior tibialis, which is a very commonly examined muscle when screening for an L5 or lumbosacral radiculopathy, was much less abnormal than the posterior tibialis or peroneus longus because the latter muscles often have more L5 innervation than the anterior tibialis, which usually has more L4 innervation. Therefore, in patients in whom there is a strong suspicion of an L5 radiculopathy, distal and proximal muscles that are predominantly supplied by the L5 root (posterior tibialis or peroneus longus and tensor fascia lata or gluteus medius) should be examined.

The pattern of findings on the needle examination, with long-duration MUPs and fibrillation potentials, indicated a relatively long-standing process, which has likely been present for or recurrent for months (because there is evidence of substantial reinnervation). The presence of fibrillation potentials in distal and proximal L5 muscles suggests an active process with ongoing denervation. Had fibrillation potentials only been present in the distal L5 muscles, without proximal fibrillations, it would have suggested that the process may be old or resolving, with adequate reinnervation proximally.

CASE 7. A WOMAN WITH WRIST DROP

Clinical History

A 54-year-old woman underwent an uneventful minor surgical procedure on her right wrist. About a week after the surgery, she awoke one morning and noted that she was unable to extend her fingers or wrist on the right. She had some possibly mild numbness in her hand, but denied experiencing any pain in her arm or neck. She had no symptoms in the left hand.

Physical Examination

The abnormal findings included severe weakness in the finger extensors, wrist extensors, and supinator on the right. The remaining muscles, including elbow extensors, were normal. She was able to feel pinprick throughout her right hand but thought that the sensation was slightly diminished on the dorsum of her hand compared with the left. Reflexes were normal.

Differential Diagnosis

The clinical history and examination findings suggested a radial neuropathy as the cause of the patient's symptoms. The distribution of deficits fits mostly into the radial nerve distribution, although the triceps seemed to be spared. However, other less likely possibilities included a posterior cord brachial plexopathy or C7 to C8 radiculopathy.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 13** and **14**. The electrodiagnostic study was performed about 3 weeks after the onset of

Table 13
Case 7: nerve conduction studies

	Amplitude (mV or μV)			Velocity (m/s)			Distal Latency (ms)			F-Wave Latency (ms)		
Stimulate (Record)	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**Est**
Median, m (APB)	8.4		>4	51		>48	4.2		<4.5	26.7		27.7
Ulnar, m (ADM)	14.1		>6	53		>51	3.1		<3.6	26.1		23.2
Median, s (index)	34		>15	58		>56	3.4		<3.6			
Ulnar, s (5th)	21		>10	57		>54	2.8		<3.1			
Radial, m (EDC)	1.2	6.2		55			2.0	2.1				
Superficial radial, s	26	28	>20				1.7	2.0	<2.9			

Abbreviations: ADM, abductor digiti minimi; APB, abductor pollicis brevis; EDC, extensor digitorum communis; NL, normal values.

the patient's symptoms. NCSs demonstrated normal median and ulnar motor and sensory responses. The right radial motor amplitude was low compared with the left. Inching along the radial nerve around the spiral groove demonstrated an 80% drop in amplitude over a 2-cm segment at the proximal spiral groove (**Fig. 2**). The radial sensory amplitude was normal and similar to that of the left. Needle examination demonstrated fibrillation potentials and no voluntary MUP activation in radial innervated muscles, apart from the triceps and anconeus. The findings were consistent with a right radial neuropathy located at the proximal spiral groove, characterized by focal demyelination with some degree of axonal loss.

Case Comment

This case demonstrated findings of an uncommon upper extremity mononeuropathy: a radial neuropathy at the spiral groove. The goal of the electrodiagnostic study in this patient was to confirm localization to the radial nerve and exclude other possibilities, such as a posterior cord plexopathy or a C7 to C8 radiculopathy. In addition, the electrodiagnostic studies were helpful in determining the precise site of the nerve lesion. In

Table 14
Case 7: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Recruitment
First dorsal interosseous	NL	0	NL	
Pronator teres	NL	0	NL	
Triceps	NL	0	NL	
Anconeus	NL	0	NL	
Brachioradialis	Increased	2+		No activation
Supinator	Increased	2+		No activation
Extensor digitorum communis	Increased	2+		No activation
Extensor indicis proprius	Increased	2+		No activation
Biceps	NL	0	NL	
Deltoid	NL	0	NL	

Abbreviation: NL, normal findings.

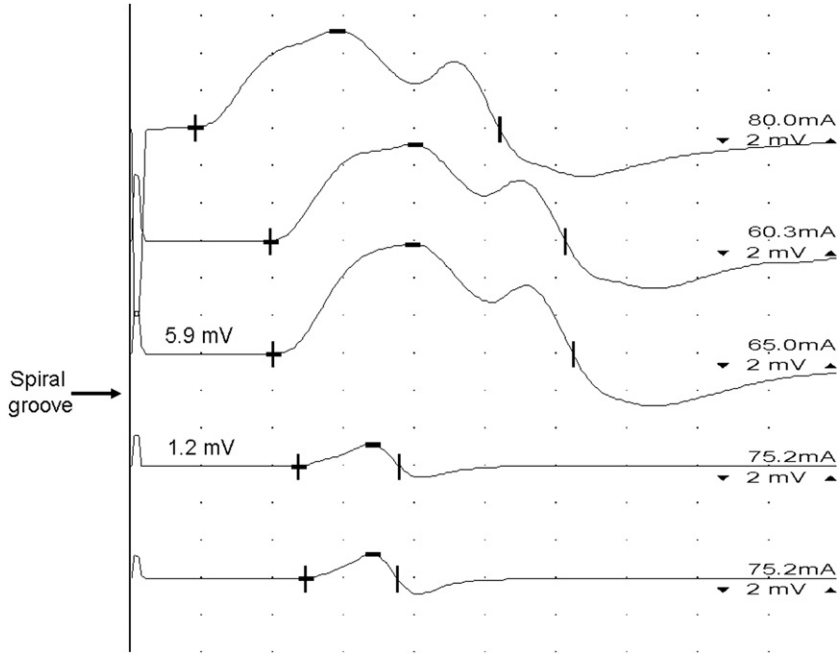

Fig. 2. Radial motor (inching) nerve conduction study in case 7, demonstrating a focal conduction block at the spiral groove.

this case standard, nonradial motor and sensory conduction studies were performed to ensure that there was no evidence of a more diffuse process or localization outside of the radial nerve distribution. The radial motor NCSs demonstrated a normal amplitude at the distal stimulation site (at the elbow), and even demonstrated a normal response when stimulation was performed at the distal portion of the spiral groove. Had stimulation not been performed at the proximal spiral groove, as occurred during the inching study, the severe focal conduction block would not have been identified on NCSs. This finding demonstrates the instructive point that if a patient has significant weakness believed to be caused by a peripheral nerve process and the motor conduction study to the weak muscle is normal, stimulation more proximally along the nerve (or even at the plexus or root) to assess for proximal conduction block should be considered.

The needle examination demonstrated abnormalities in the radial muscles distal to the anconeus. The triceps and the anconeus are the only two radial innervated muscles whose nerve branches emanate from the radial nerve proximal to the spiral groove. The presence of fibrillation potentials indicates some degree of axonal loss (in addition to the focal demyelination noted by the conduction block). This temporal progression from conduction block to axonal loss is common with many nerve compression lesions.

It is also notable that the radial sensory amplitude was spared, likely because the major pathophysiologic change was conduction block and because the typical radial sensory study is performed with stimulation distal to the site of block (at the wrist). Identifying proximal conduction block in sensory fibers is very difficult because of the normal dispersion of the SNAPs over long distances. This patient was thought to have a “Saturday night palsy.”

CASE 8. A MAN WITH ARM PAIN AND WEAKNESS AFTER YARD WORK

Clinical History

A 54-year-old man presented with right upper extremity weakness and pain. The morning after a day of yard work, he experienced severe pain in his right shoulder that worsened with movement of his arm. The pain continued over the next week, during which he noted weakness of his right arm. Over the next 2 weeks his pain reduced and was present only intermittently; however, he continued to note weakness of his arm. He described weakness in raising his right arm and some weakness on gripping objects. He had some mild numbness in his right shoulder. He did not complain of any symptoms in his left arm.

Physical Examination

The only abnormal findings were seen in the patient's right arm. Muscle strength testing in the right arm was as follows (graded on the MRC scale): deltoid 4, biceps 4, infraspinatus 2, supraspinatus 2, triceps 5, pronation 4, supination 4, wrist extension 5, wrist flexion 5, flexor digitorum profundus (index and middle digits) 2, flexor digitorum profundus (fourth and fifth digits) 5, flexor pollicis longus 2, extensor digitorum communis 5, interossei 5, and abductor pollicis brevis 5. There was prominent atrophy of his right supraspinatus and infraspinatus. Reflexes were normal. There was mild decreased pinprick sensation over his right shoulder.

Differential Diagnosis

In this patient, the onset of pain in the arm after a day of yard work would first raise the concern for a cervical radiculopathy. The pattern of weakness on his neurologic examination is complicated and difficult to localize into a specific root distribution. One possibility was that of a C5 to C6 root lesion, although the weakness in some of his more distal arm muscles would suggest a possible C8 to T1 lesion. Other possibilities would include multiple mononeuropathies (axillary, musculocutaneous, and partial median), a brachial plexopathy, or a central cord lesion (although he did not have features of a myelopathy).

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 15** and **16**. NCSs demonstrated a reduced right suprascapular CMAP amplitude relative to the left (**Fig. 3**). The right lateral antebrachial and radial sensory amplitudes were

Table 15
Case 8: nerve conduction studies

	Amplitude (mV or μV)			Velocity (m/s)			Distal Latency (ms)			F-Wave Latency (ms)		
Stimulate (Record)	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**NL**	**R**	**L**	**Est**
Median, m (APB)	6.8		>4	52		>48	4.4		<4.5	29		30
Ulnar, m (ADM)	12.2		>6	60		>51	3.5		<3.6	29		25
Median, s (index)	44		>15	58		>56	3.3		<3.6			
Ulnar, s (5th)	39		>10	61		>54	3.2		<3.1			
Lateral antebrachial, s	19	39					2.0	2.0				
Suprascapular, m	5.9	9.8					2.0	2.2				
Superficial radial, s	20	42	>20				2.4	2.2	<2.9			

Table 16
Case 8: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	MUP	Recruitment (Reduced)	Duration (Long)	Amplitude (High)	Phases
Rhomboid major	NL	0	NL				
Infraspinatus	Inc	2+		2+	2+	1+	100%
Deltoid	Inc	1+			1+		
Biceps brachii	Inc	1+			±		
Supraspinatus	Inc	±		2+	2+	2+	100%
Triceps brachii	NL	0	NL				
Extensor digitorum communis	NL	0	NL				
Pronator teres	NL	0	NL				
Flexor pollicis longus	Inc	2+		2+	1+	1+	
Pronator quadratus	Inc	2+		2+	1+		
Abductor pollicis brevis	NL	0	NL				
First dorsal interosseous	NL	0	NL				
C6 paraspinal	NL	0	NL				

Abbreviations: Inc, increased; NL, normal.

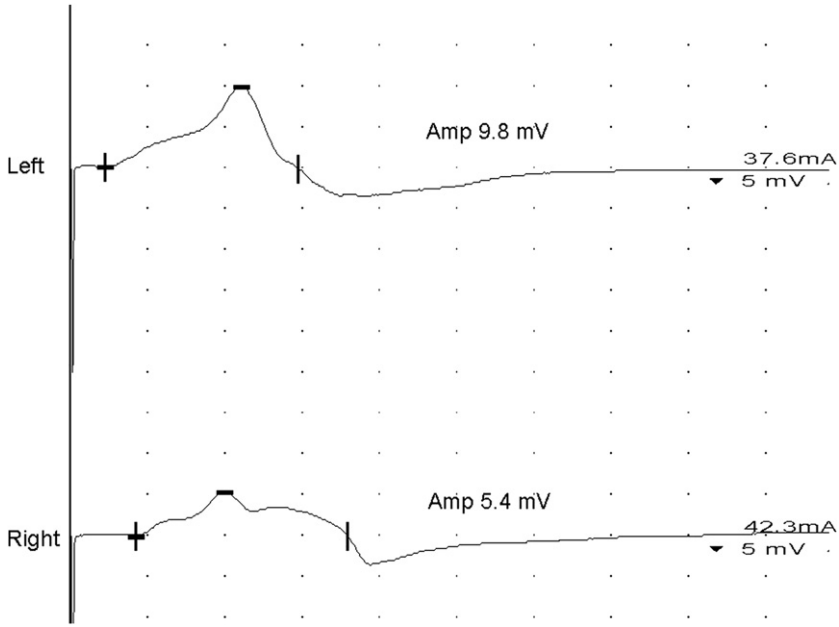

Fig. 3. Suprascapular nerve conduction studies in case 8, demonstrating an approximately 50% amplitude reduction in the affected (right) side compared with the unaffected side.

mildly low relative to the left. Concentric needle EMG demonstrated fibrillation potentials and long-duration, polyphasic MUPs with markedly reduced recruitment in the right supraspinatus, infraspinatus, flexor pollicis, and pronator quadratus, and similar, but much milder, findings in the deltoid and biceps. The remaining muscles were normal. The study was interpreted as a complex study with evidence of a patchy process, most likely involving the upper trunk of the brachial plexus (and mainly the fibers to the suprascapular nerve) and the anterior interosseous branch of the median nerve. The pattern of findings, primarily involving 2 individual nerves, would be compatible with a patchy inflammatory process involving the nerve, such as seen in neuralgic amyotrophy.

Case Comments

This is a complicated case, in which the pattern of abnormalities is difficult to precisely localize into a single site within the peripheral nervous system. The initial concern based on the history and clinical examination was for multiple cervical radiculopathies, a brachial plexopathy, or multiple mononeuropathies. The study began with standard motor and sensory NCSs (median and ulnar). Despite the patient's weakness in some median-innervated muscles, the normal responses obtained on median motor (abductor pollicis brevis) and sensory (recorded from the index finger) argued against a process involving the proximal median nerve. However, routine median NCSs do not reliably assess the anterior interosseous branch of the median nerve (AION) (in which less commonly performed NCSs, such as recording from the pronator quadratus, would need to be performed). In addition, because most of his weakness outside of the AION was in the shoulder girdle muscles (especially the spinati), the routine NCSs do not thoroughly assess for a C5 to C6 root lesion or an upper trunk plexopathy. Because these localizations were strongly considered, additional sensory NCSs of fibers that course through the upper trunk (lateral antebrachial cutaneous and superficial radial) were performed. The reduction in amplitudes in both of these indicated a lesion that was distal to the dorsal root ganglion and supported a brachial plexopathy (or multiple mononeuropathies). The suprascapular motor NCS was also performed to provide an objective measure of the degree of nerve dysfunction, although assessment of this nerve could have also been made solely on the needle examination of the supraspinatus and infraspinatus.

The needle examination demonstrated findings of a neurogenic process that again was difficult to precisely localize into a single lesion. The most severe abnormalities were present in muscles supplied by the suprascapular and AION, but there were also mild abnormalities in other muscles supplied by the upper trunk of the brachial plexus.

The pattern of electrodiagnostic findings, in the context of the patient's history, was typical of neuralgic amyotrophy (Parsonage-Turner syndrome). This entity is categorized as an inflammatory or immune-mediated brachial plexopathy; however, preferential involvement of a single or multiple individual nerves in addition to other portions of the brachial plexus is very common.[3] This entity may have a predilection to certain nerves, such as the long thoracic, suprascapular, and AION. As a result of the patchy nature of involvement, neuralgic amyotrophy may be one of the most difficult entities to study from an electrodiagnostic standpoint. Maintaining a high index of suspicion, performing a very thorough clinical neuromuscular examination before performing any electrodiagnostic studies, and liberalizing the number of NCSs (often with side-to-side comparisons) and muscles examined with needle EMG are important steps in an appropriate assessment of these patients.

CASE 9. A MAN WITH POSTOPERATIVE DIPLOPIA, DYSARTHRIA, AND FATIGUE

Clinical History

A 67-year-old man underwent an uncomplicated arthroscopic surgical procedure on his left shoulder for shoulder pain. In the next 1 to 2 weeks he began to experience fluctuating dysarthria, ptosis, and intermittent diplopia. He noted blurring of his vision when reading or driving, with occasional double vision. He also noted that either or both of his eyelids would droop when he was tired. He had some difficulty swallowing liquids. The symptoms were present throughout the day, but tended to fluctuate in nature and were typically worse toward the afternoon and evening. He also reported feeling generalized fatigue and some weakness in his arms and legs. He had no sensory complaints or pain.

Physical Examination

The patient's neurologic examination demonstrated mild bilateral ptosis that seemed to worsen slightly with sustained upgaze. There were no definite extraocular movement abnormalities. Strength was normal except for mild weakness in his orbicularis oculi and oris muscles, and in the proximal shoulder muscles bilaterally. Deep tendon reflexes, gait, sensory, and coordination were normal.

Differential Diagnosis

This patient seemed to have a generalized process, primarily causing weakness in a cranial-cervical distribution. In this patient with fatigable weakness, double vision, and bulbar symptoms, the primary concern was a neuromuscular junction disorder such as myasthenia gravis. Other generalized neuromuscular conditions, such as a myopathy, polyradiculopathy, or motor neuron disorder, were also considered, and can be associated with fatigue or an increased sense of weakness after activity or later in the day. In this case, it is unclear whether or how the patient's antecedent surgery contributed to his symptoms, but the stress of the surgery could have been a trigger for unmasking certain conditions, including myasthenia gravis, an inflammatory polyradiculopathy, or a subclinical myopathy.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 17** and **18**. The median sensory and ulnar motor conduction studies were normal. Repetitive stimulation studies were performed at 2 Hz before and after 1 minute of exercise in the left spinal accessory and facial nerves. No abnormal decrement in the CMAP amplitude or area was seen in the spinal accessory nerve (maximum decrement

Table 17
Case 9: nerve conduction studies

Stimulate (Record)	Amplitude (mV or μV) (Normal)	Velocity (m/s) (Normal)	Distal Latency (ms) (Normal)	F-Wave Latency (ms) (Estimate)
Median (index), s	23 (>15)	57 (>56)	3.2 (<3.6)	
Ulnar (hypothenar), m	7.2 (>6.0)	52 (>51)	3.4 (<3.6)	28 (27)
Facial (nasalis), m[a]	1.5 (>1.8)		2.0 (<4.1)	
Spinal accessory, m (trapezius)[a]	4.5		2.5	

[a] 2-Hz repetitive stimulation performed.

Table 18
Case 9: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	MUP
Deltoid	NL	0	NL
First dorsal interosseous	NL	0	NL
Frontalis	NL	0	Varying
Orbicularis oculi	NL	0	Varying

Abbreviation: NL, normal findings.

between the first and fourth stimulus was 8%), but a mild degree of abnormal decrement (maximum of 12%) was seen in the facial nerve. After 1 minute of exercise, the maximum degree of decrement was 9% in the spinal accessory nerve and 20% in the facial nerve (**Fig. 4**). The needle examination demonstrated varying (unstable) MUPs in the orbicularis oculi and frontalis, but not in limb muscles. The mild abnormalities on repetitive stimulation studies and needle examination of cranial muscles were suggestive of a mild defect of neuromuscular transmission, consistent with a neuromuscular junction disorder such as myasthenia gravis.

Case Comment

This type of case, a patient with mild weakness in cranial and proximal muscles, can be challenging from an electrodiagnostic standpoint. In these patients, the main differential diagnosis is a neuromuscular junction disorder, unusual distribution myopathy (eg, mitochondrial, facioscapulohumeral muscular dystrophy, and so forth), motor neuron disorder, or polyradiculopathy. The study began with one routine motor and sensory NCS in the arm, both of which were normal as was expected. Even though the patient had no sensory symptoms and no distal weakness, the performance of at least a few routine studies can help to identify or exclude a process such as a polyradiculoneuropathy (in which there may be abnormal sensory responses, or conduction velocity slowing or increased temporal dispersion on the motor studies) as well as a motor neuron disorder (in which the CMAP amplitudes may be low). In addition, low CMAP amplitudes may also increase the suspicion for the less common types of

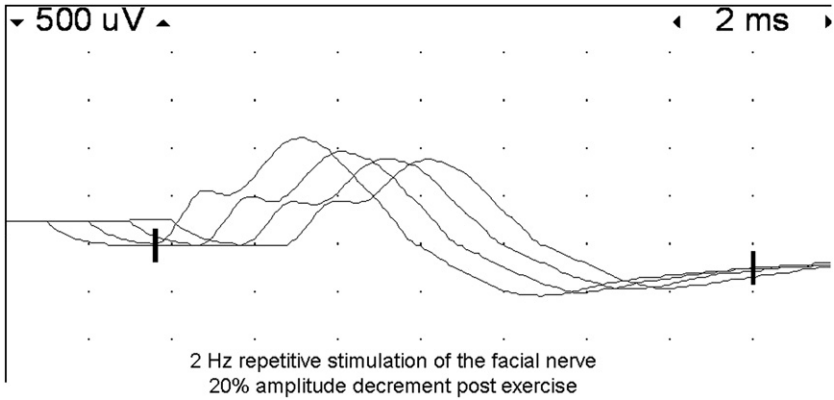

Fig. 4. Facial nerve repetitive stimulation at 2 Hz at 3 minutes after exercise in case 9. There is a 20% decrement in amplitude between the first and the fourth stimulus.

neuromuscular junction disorders, such as LEMS. When this disorder is suspected and low CMAP amplitudes are seen, assessment for facilitation by supramaximally stimulating the motor nerve immediately after 10 seconds of exercise should be performed.

In addition to routine motor and sensory NCSs, repetitive stimulation is important and necessary to evaluate for a neuromuscular junction disorder, such as myasthenia gravis. Choosing the nerve on which to perform repetitive stimulation studies depends on the distribution and extent of clinical involvement. In patients with generalized symptoms, distal nerve-muscle combinations, such as the ulnar or peroneal, are technically the easiest and most reliable nerves to test. However, this patient had only bulbar and proximal weakness and, therefore, repetitive stimulation was performed on the spinal accessory and facial nerves. The repetitive nerve stimulation studies were essentially normal in the spinal accessory, because many consider an abnormal decrement as greater than 10% reduction in amplitude and area between the first and fourth or fifth stimulus. However, any degree of true decrement is technically abnormal; so in this patient the 8% decrement that was consistently present in three trials at rest may have been a clue to a defect in neuromuscular transmission. Caution should always be used when interpreting abnormal decrement to ensure that the study is technically reliable and that the pattern of decrement (largest drop between the first and second stimulus with a tapering pattern) is physiologic. This patient demonstrated more significant (>10%) decrement in the most affected muscles, with repetitive stimulation of the facial nerve. Because the decrement was only mild or borderline at rest, repetitive stimulation studies were performed also after 1 minute of exercise, which may increase the degree of decrement in some patients.

The needle examination focused on not only muscles that were clinically weak but also sampled muscles (such as the first dorsal interosseous) that were clinically spared, to assess for more widespread subclinical involvement. The absence of fibrillation potentials or markedly short-duration or long-duration MUPs essentially excluded a severe myopathy or motor neuron disorder. However, some mild myopathies may not demonstrate prominent needle examination abnormalities, but these conditions would also not be expected to produce decrement on repetitive stimulation either. The only finding on the needle examination in the clinically weak muscles was abnormal MUP variation (unstable or varying MUPs), which is the typical finding in neuromuscular junction disorders. Unstable MUPs can also be seen in myopathies and neurogenic disorders, but those conditions would also be associated with other configurational changes in the MUPs. The unstable MUPs in conjunction with the abnormal repetitive stimulation were consistent with a neuromuscular junction disorder.

In this patient, had the routine needle examination been normal, single-fiber EMG would have been performed as the most sensitive test for a defect of neuromuscular transmission. Single-fiber EMG should be performed if there is a high clinical index of suspicion or if ptosis and/or diplopia are the only clinical symptoms, and the routine studies are normal.

As a final note, patients who are being studied for a neuromuscular junction disorder should not take pyridostigmine (Mestinon) for at least 4 to 8 hours before electrodiagnostic testing, because the medication may improve and mask abnormal decrement, producing a false-negative study. This patient was being treated for presumed myasthenia gravis with pyridostigmine before confirmation with the EMG study. When he arrived at the laboratory on the morning of the test, he had indicated that he had taken his pyridostigmine 1 hour before. The test was delayed until later in the afternoon to avoid false-negative results.

CASE 10. A VETERINARIAN WITH PROGRESSIVE WEAKNESS

Clinical History

A 35-year-old left-handed veterinarian presented with a 3-year history of slowly progressive weakness. The weakness initially began in her right hand and gradually worsened to involve both upper and lower extremities. She required the use of a walker to assist with ambulation. She reported mild dysphagia, but no ptosis, diplopia, dysarthria, or respiratory symptoms. She described no pain or sensory complaints.

Physical Examination

Her neurologic examination demonstrated generalized weakness of moderate severity in her arms and legs, right side worse than left, with increased tone in the right lower extremity, brisk deep tendon reflexes, and bilateral Babinski signs. She had atrophy of her intrinsic hand muscles bilaterally and fasciculations in many muscles. Her speech was notable for a subtle spastic dysarthria, and tongue strength was mildly weak. Sensory examination was normal.

Differential Diagnosis

The primary concern in this case, given the combination of upper and lower motor neuron examination findings, is progressive motor neuron disease such as amyotrophic lateral sclerosis (ALS). Other possibilities include multiple cervical, thoracic, and lumbosacral radiculopathies, a polyradiculopathy, or a severe neuromuscular junction disorder or myopathy. However, the upper motor neuron signs would not be typical of these other possibilities, unless she had two processes.

Electrodiagnostic Summary and Interpretation

The electrodiagnostic studies (NCSs and needle examination) are shown in **Tables 19** and **20**. The NCSs of the right upper and lower limbs revealed an absent median motor response and low ulnar and tibial motor CMAP amplitudes. No conduction velocity slowing, conduction blocks, or abnormal temporal dispersion were seen. Needle examination demonstrated fibrillation potentials in most muscles studied with fasciculation potentials in many muscles. In addition, there was reduced recruitment of long-duration, high-amplitude, and frequently polyphasic and varying MUPs in most muscles. The findings were those of a diffuse neurogenic disorder affecting anterior

Table 19
Case 10: nerve conduction studies

Stimulate (Record)	Amplitude (mV or μV) (Normal)	Velocity (m/s) (Normal)	Distal Latency (ms) (Normal)	F-Wave Latency (ms) (Estimate)
Ulnar, m (hypothenar)	3.8 (>6)	65 (>51)	3.0 (<3.6)	24 (28)
Ulnar, s anti (fifth)	66 (>10)	64 (54)	2.8 (<3.1)	
Median, m (thenar)	NR	NR	NR	
Median, s anti (index)	64 (>15)	57 (>56)	2.7 (<3.6)	
Peroneal, m (extensor digitorum brevis)	4.8 (>2)	48 (>41)	5.4 (6.6)	45 (43)
Superficial peroneal, s (ankle)	17 (>0)		3.7 (<6.1)	
Tibial, m (abductor hallucis)	1.9 (4)	41 (>40)	5.2 (<6.1)	52 (49)

Abbreviation: NR, no response.

Table 20
Case 10: needle examination

Muscle	Insertional Activity	Fibrillation Potentials	Fasciculation Potentials	MUP	Recruitment (Reduced)	Duration (Long)	Amplitude (High)	Phases
Biceps[a]	Inc	2+	1+		2+	2+	2+	
Triceps[a]	Inc	3+	0		2+	2+	2+	25%
First dorsal interosseous	Inc	2+	0		2+	2+	2+	
Vastus lateralis[a]	Inc	2+	1+		1+	2+	2+	
Tibialis anterior[a]	Inc	1+	0		2+	2+	2+	
Lateral gastrocnemius	Inc	1+	1+		1+	2+	1+	
T7 paraspinal	Inc	1+	0		1+	±	±	25%

Abbreviation: Inc, increased.
[a] Varying MUP.

horn cells or their axons affecting the cervical, thoracic, and lumbosacral segments, consistent with a progressive motor neuron disease such as ALS.

Case Comment

This case demonstrates electrodiagnostic features that are seen with progressive motor neuron disease (eg, ALS). The motor NCSs in the upper and lower extremities demonstrated low CMAP amplitudes, indicating axonal loss. Careful observation of the waveforms was important to assess for abnormal temporal dispersion or conduction block (which were not seen in this case), which would be seen in some inflammatory/demyelinating polyradiculopathies, such as chronic inflammatory demyelinating polyradiculopathy or multifocal motor neuropathy with conduction block, both of which can have some clinical similarities to ALS. The sensory NCSs were normal, which would be expected in motor neuron disease unless there was a concomitant peripheral neuropathy or mononeuropathy.

The needle examination approach was based on clinical findings. The needle examination included examination of 2 to 3 muscles in the cervical, thoracic, and lumbar region that were not innervated by the same root or nerve, to assess for a diffuse process. The findings of fibrillation potentials and long-duration MUPs with reduced recruitment in a widespread distribution were consistent with a severe, diffuse neurogenic process. Varying or unstable MUPs are common in progressive neurogenic disorders, such as ALS, but are often overlooked when the examiner is focusing more on the MUP size and recruitment pattern. Muscles selected were those likely to be abnormal. Thoracic paraspinal muscles were examined to demonstrate involvement of the disease process in this segment, and the presence of abnormalities in these muscles helps to exclude multiple chronic cervical and lumbosacral radiculopathies. Of note, fasciculation potentials were also present in several muscles sampled, a finding which is nonspecific but consistent with motor neuron disease.

The combination of findings fit with progressive motor neuron disease. However, taken in isolation the EMG findings could also be seen with a severe axonal polyradiculopathy and therefore must be interpreted in the context of the clinical features. In severe polyradiculopathies, deep tendon reflexes are typically reduced or absent rather than hyperactive, as in this case of ALS. In addition, there is often some degree

of subjective or objective sensory loss in polyradiculopathies, despite the normal sensory NCSs.

SUMMARY

These 10 cases demonstrate the approaches taken in the EMG laboratory to certain common and uncommon clinical problems. Although the approach to any one patient may vary to some extent by different laboratories or electromyographers and needs to be individualized toward the clinical problem and findings on the examination, the general guidelines as have been described in these cases and elsewhere can assist in identifying the appropriate localization and diagnosis of each type of clinical problem.[4]

REFERENCES

1. Vennix MJ, Hirsh DD, Chiou-Tan FY, et al. Predicting acute denervation in carpal tunnel syndrome. Arch Phys Med Rehabil 1998;79:306–12.
2. Levin KH. L5 radiculopathy with reduced superficial peroneal sensory responses: intraspinal and extraspinal causes. Muscle Nerve 1998;21:3–7.
3. Rubin DI. Neuralgic amyotrophy: clinical manifestations and evaluation. Neurologist 2001;7:350–6.
4. Rubin DI, Daube JR. Application of clinical neurophysiology: assessing peripheral neuromuscular symptom complexes. In: Daube JR, Rubin DI, editors. Clinical neurophysiology. 3rd edition. New York: Oxford University Press; 2009. p. 801–37.