What Good Is EMG to the Patient and Practitioner?

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ABSTRACT

Electromyography (EMG) and nerve conduction studies (NCS) are not only tests to be performed in isolation and reported without consideration of the clinical context, but rather form part of what has been referred to as the electrodiagnostic consultation. Using all of the pertinent information available to the electromyographer performing the test, the electrodiagnostic consultation strives toward the goal of helping the patient and the referring physician to establish a correct diagnosis. Although not without limitations, EMG as an extension of the clinical history and physical examination can be a powerful and sensitive diagnostic tool. Like any tool, however, the final result depends on the skill and expertise with which it is wielded.

KEYWORDS: Electromyography (EMG), nerve conduction studies (NCS), electrodiagnosis, neuromuscular disease, diagnostic testing

Electromyography (EMG) is the part of electrodiagnostic medicine consisting of recording the variations of electric potential or voltage detected by a needle electrode inserted into skeletal muscle. This electric activity is displayed on a monitor and played over a loudspeaker for simultaneous visual and auditory analysis. In normal resting muscle little or no electric activity is detected, but during voluntary contraction the action potentials of motor units appear. In disorders of the motor unit, electric activity of various types may appear in resting muscle, and the action potentials of the motor units may have abnormal forms and patterns of activity. Abnormalities of the EMG serve as objective criteria of dysfunction of the motor unit. These abnormalities may characterize the nature of the disease process and its localization in the neuron, neuromuscular junction, or muscle fibers. Critical to understanding the role of electrodiagnostic testing in clinical medicine is a clear realization that EMG is an extension of the neurologic examination.[1] It does not provide a clinical diagnosis of the patient's illness. There are virtually no waveforms that are pathognomonic of specific disease entities. The EMG findings must be integrated with the patient's history, the clinical examination, and the results of other tests in order to arrive at a correct diagnosis.[1]

TERMINOLOGY

Strictly defined, EMG is the recording and study of insertion, spontaneous, and voluntary activity of muscle with a recording needle electrode. Nerve conduction studies (NCS) are an important adjunct to EMG. The 2001 American Association of Electrodiagnostic Medicine Glossary of Terms provides a uniform agreed upon framework for expressing electric phenomena encountered in studying patients with EMG.[2] Although detailed descriptions and illustrations of each phenomenon are beyond the scope of this brief article, the basics of each are listed.

Questions for the Consultant; Editor in Chief, Karen L. Roos, M.D.; Guest Editors, Joseph I. Sirven, M.D., Dean M. Wingerchuk, M.D. *Seminars in Neurology*, Volume 23, Number 3, 2003. Address for correspondence and reprint requests: Benn E. Smith, M.D., Department of Neurology, Mayo Clinic Scottsdale, 13400 East Shea Boulevard, Scottsdale, AZ 85259. [1]Director, EMG Laboratory and Assistant Professor of Neurology, Mayo Clinic Scottsdale, Scottsdale, Arizona. 0271-8235,p;2003,23,03,335,342,ftx,en;sin00263x.

Electromyography Terms

Motor unit: The anatomic element consisting of an anterior horn cell, its axon, the neuromuscular junctions, and all the muscle fibers innervated by the axon.

Insertion activity: Electric activity caused by insertion or movement of a needle electrode within a muscle.

Spontaneous activity: Electric activity recorded from muscle at rest after insertion activity has subsided and when there is not voluntary contraction or an external stimulus.

Fibrillation potential: The action potential of a single muscle fiber occurring spontaneously or after movement of a needle electrode. Usually fires at a constant rate.

Fasciculation potential: The electric activity associated with a fasciculation that has the configuration of a motor unit action potential but occurs spontaneously.

Voluntary activity: In EMG, the electric activity recorded from a muscle with consciously controlled contraction.

Motor unit action potential (MUAP or MUP): The compound action potential of a single motor unit whose muscle fibers lie within the recording range of an electrode. The following measures may be specified after the recording electrode is placed in the muscle: *configuration* (including amplitude, duration, number of phases, polarity of each phase, number of turns, variation of shape with consecutive discharges, presence of satellite potentials, spike duration, and rise time) and *recruitment characteristics* (including threshold of activation, onset frequency, and recruitment frequency—allowing classification into normal, reduced, or rapid recruitment categories).

Activation: The process of motor unit action potential firing, with the force of muscle contraction being determined by the number of motor units firing and their firing rate.

Nerve Conduction Study Terms

Nerve conduction studies: Recording and analysis of electric waveforms of biologic origin elicited in response to electric stimuli.

Action potential: The brief regenerative electric potential that propagates along a single axon or muscle fiber.

Compound muscle action potential (CMAP): The summation of nearly synchronous muscle fiber action potentials recorded from a muscle, commonly produced by stimulation of the nerve supplying the muscle.

Compound sensory nerve action potential (SNAP): A compound nerve action potential from the afferent fibers of a sensory nerve, a sensory branch of a mixed nerve, or in response to stimulation of a sensory nerve or dorsal root.

Nerve conduction velocity (NCV): Speed of propagation of an action potential along a nerve or muscle fiber.

Distal latency (dL): The interval between the delivery of a stimulus to the most distal point of stimulation on a nerve and the onset of a response.

Artifact: A voltage change generated by a biologic or nonbiologic source other than the ones of interest.

QUESTIONS ADDRESSED BY EMG AND NCS

By virtue of the nature of the testing, EMG and NCS are well suited to provide answers to a number of questions of clinical interest. These include investigation of patients with weakness, muscle wasting or fixed sensory loss. Each of these three categories will be looked at in turn.

Weakness

In a patient with weakness, *is there evidence of disease of muscle*? Many myopathies show low-amplitude CMAPs, often with normal conduction velocities. Concentric needle examination often demonstrates small, sometimes polyphasic MUPs in proximal or other muscle territories, on occasion accompanied by abnormalities of insertional and spontaneous activity that have been linked to certain pathologic findings such as inflammation, myonecrosis, fiber splitting, and vacuolar change.[3]

In a patient with weakness, *is there evidence of disease of the neuromuscular junction*? Routine NCS are often normal in postsynaptic defects of neuromuscular transmission, such as autoimmune myasthenia gravis, whereas presynaptic disorders such as the Lambert-Eaton myasthenic syndrome often show low-amplitude CMAPs in a patient with global hyporeflexia. Special techniques such as slow (typically 2 to 3 Hz) repetitive stimulation of distal and proximal muscle nerves often demonstrate a characteristic abnormal pattern of amplitude and area decrement that correlates with defective neuromuscular transmission.[4] Although concentric needle examination is usually normal, single-fiber EMG provides the most sensitive readily available electrodiagnostic method to detect abnormal neuromuscular transmission as evidenced by increased jitter and blocking as well as other findings.[5]

In a patient with weakness, *is there evidence of disease of nerve*? In the most common form of peripheral polyneuropathy, which is distal axonopathy, NCS typically show a length-dependent reduction in sensory and motor response (CMAP and SNAP) amplitudes as well as slowed NCVs and prolonged distal latencies; the conduction slowing is even more pronounced in demy-

elinating neuropathies.[6] Concentric needle examination often demonstrates a similar pattern of distal greater than proximal neurogenic abnormalities with large MUPs and changes in insertional and spontaneous activity.[6] In the special case of focal nerve conduction block, localized mononeuropathies or multifocal disorders such as multifocal motor neuropathy can sometimes be identified.[7]

In a patient with weakness, *is there evidence of disease at the anterior horn cell level*? The patterns of nerve conduction abnormality in patients with anterior horn cell disorders vary but typically show low-amplitude motor responses with relative preservation of sensory waveforms. The EMG often discloses large motor unit potentials and increased insertional activity with both fibrillation potentials and fasciculation potentials in amyotrophic lateral sclerosis.[8] Old and chronic motor neuron diseases may show no abnormalities of insertional or spontaneous activity.

One group of patients that has caused confusion for electromyographers and referring clinicians alike is the cohort with symptoms and often signs of apparent weakness in whom the EMG and NCS are normal. Some of these individuals have poor activation because of pain in the region being tested; a few simple questions and observations of the patient's behavior help the examiner determine whether this is likely to be the case. A second group with poor activation is those who have central nervous system disorders such as stroke, myelopathy, or multiple sclerosis. These individuals invariably have physical findings and other symptoms to corroborate these central disorders that may be interfering with voluntary activation due to upper motor neuron or extrapyramidal pathway dysfunction. Others may not be able to activate fully because of disuse, malnutrition, or prolonged corticosteroid use. A fourth category is those who do not fully activate voluntary muscle for psychological reasons; the absence of pain, lack of central nervous system signs and symptoms, and dearth of other physical factors can lead to their identification. Similarly, the few individuals who are consciously feigning weakness or other deficits for secondary gain usually have no severe pain, spasticity, or other findings that provide a reasonable medical explanation for their lack of voluntary muscle activation.

Muscle Wasting

In a patient with muscle wasting, EMG can assist in determining whether there may be a neuromuscular explanation. Remembering that electrodiagnostic testing extends the reach of the clinical history and examination; the presence, pace of acquisition, and distribution of neurologic deficits can aid the electromyographer in characterizing which elements of the motor unit, if any, may be involved in the wasted patient at hand. As discussed before, patterns of abnormal findings on NCS and EMG suggest either myopathy, neuromuscular junction disease, neuropathy or motor neuronopathy, or, alternatively, that there is no convincing evidence of disease of muscle, nerve, neuromuscular junction, or anterior horn cell. In the latter case, historical and physical examination clues often point the clinician toward either disuse as an explanation of the muscle wasting—which may be physical, psychological, or a combination—or toward weight loss associated with underlying medical disease such as cancer, infection (human immunodeficiency virus being one example), diabetic cachexia, or malnutrition associated with anorexia.

Fixed Sensory Loss

Another category of patient that is effectively interrogated by NCS techniques is that of sensory loss or other persistent sensory symptoms. Sensory NCS can indicate whether or not there is evidence of large-diameter dorsal root ganglion cell or large-caliber sensory axon disease in a particular nerve territory or in a widespread distribution throughout the body.[9] One important observation regarding the individual with clinical sensory loss and normal sensory NCS results is that either the neuropathy affects only small-diameter fibers (so-called small fiber sensory neuropathy), the lesion is central (affecting the dorsal column pathway, for example),[10] or the process is nonorganic.

QUESTIONS NOT LIKELY TO BE ANSWERED BY EMG AND NCS

Although EMG and NCS can often pinpoint and characterize disease of the peripheral nervous system with clarity and quantitative precision, there are times when the electrodiagnostic methods do not provide a specific answer to the question posed by the referring physician. There are a number of situations that fall into this category. The first is that the *referral question is too general*, such as "neurologic disease?," "gait disorder?," "weakness?," "fatigue?," or "total body pain?" Part of the reason that some referring physicians make such general requests of electrodiagnostic medicine is lack of familiarity with the testing procedures. To use an example familiar to the majority of physicians, in electrocardiography (ECG), the testing procedures are very uniform, with standardized electrode placement and recording techniques that are virtually identical for every patient undergoing an ECG test. For NCS, on the other hand, the breadth of techniques as well as nerves and muscles capable of being tested is staggering. More than 30 nerves in the face, neck, thorax, upper limb, and lower limb can be assessed using NCS techniques, some with

multiple different methods of stimulation and recording.[11] Similarly, there are on the order of 100 skeletal muscles from head to foot that can be studied by EMG.[12] Unlike the case of ECG, if all of the available electrodiagnostic techniques were employed to evaluate a single patient, the testing could last more than 10 to 12 hours.

Another reason that EMG may not help the referring provider is that the *symptoms may be too recent*. In many acute neurogenic processes, for example, NCS abnormalities and all but the most subtle EMG changes are not apparent until 10 to 14 days after the inciting event. In this situation, it is usually more useful to wait at least 2 weeks after onset of acute neurologic symptoms before considering EMG.[13] An exception occurs when it is necessary to document whether or not preexisting peripheral abnormalities are present. This can be critically important in medicolegal cases involving traumatic injuries or when an underlying condition is present that is likely to be associated with EMG abnormalities, for example, polyneuropathy. A somewhat related challenge arises when attempting to exclude, identify, or characterize a superimposed peripheral process in the setting of a diffuse disorder, for example, focal mononeuropathy in an individual with known polyneuropathy.[14,15]

Finally, when neuromuscular disease is suspected clinically and electrodiagnostic studies are found to be entirely normal, barring acute neurogenic processes, the clinician must *consider other explanations for the symptoms and signs* posed by the patient.

EXPERIENCING ELECTROMYOGRAPHY AND NERVE CONDUCTION STUDIES

Nerve Conduction Studies

Perhaps the best way to understand the procedures that patients undergo during EMG and NCS is to experience the testing first hand. During motor NCS, metal electrode disks are taped to the skin overlying the motor point of the muscle being examined. Graded electric stimuli are then delivered first to the proximal limb site of the nerve and then, after several seconds, to the distal limb site of the nerve. The responses are recorded at each site and then measured either manually on paper or by computer for amplitude, latency, and other factors. The NCV is calculated dividing the distance between the two stimulation sites by the time required for the response to traverse the path between them (velocity = distance/time). For sensory NCS, both the stimulation and recording sites overlie the sensory nerve trunk being investigated.[9]

Technical factors sometimes limit or prevent successful performance of NCSs.[16] Central venous catheters, pacemaker leads, and implanted defibrillator wires provide a direct current path to the heart. Patients with these devices undergoing a proximal NCS, therefore, may be subjected to the risks of induced cardiac dysrhythmia or of an implanted defibrillator mistakenly being activated to deliver a cardioverting stimulus to the patient and possibly others in contact with the patient. In large or obese patients, the distance between the surface stimulator and the nerve being stimulated is often too great to produce a supramaximal response or, on occasion, any response at all. Needle stimulation of the nerve can be done only if normal values have been collected for the needle stimulation methods being used; most laboratories have not collected normal values of this type. Because percutaneous NCS normal values do not include markedly obese individuals, interpretation of results in such individuals can be difficult. When the patient has a skin lesion in the locations where either stimulating or recording electrodes must be applied, the study cannot be performed. Similarly, when the stimulation and/or recording sites are inaccessible because of a plaster cast or other nonremovable dressing or therapeutic device, the NCS must await removal of the interfering materials.

A special case arises when the electrodiagnostic medicine specialist is asked to evaluate for the possibility of a neuromuscular junction defect a patient who is currently taking an anticholinesterase medication such as pyridostigmine. In this case, testing is best postponed until the subject can suspend the anticholinesterase agent for at least 8 and preferably 12 hours (if the subject can do so without compromising bulbar function, which is usual for individuals being evaluated for possible myasthenia gravis) so as to avoid a false-negative test result.

Electromyography

Needle EMG is typically performed by inserting a fine single-use concentric needle electrode (some practitioners prefer monopolar electrodes) just under the surface of the skin into a skeletal muscle. With the muscle at rest, insertional activity is assessed by making multiple tiny advances of the electrode, each a fraction of a millimeter in distance, through the muscle.[17] Spontaneous electric activity is recorded with both the muscle at rest and the needle stationary within the muscle. During a weak contraction of the muscle volitional activity is recorded, consisting of collecting MUPs and assessing such attributes as duration, amplitude, morphology, and firing patterns.[18]

Factors that limit the performance of EMG include anticoagulant therapy, tremor, spasticity, and morbid obesity. Patients taking warfarin with therapeutic international normalized ratio (INR) values, and particularly those with INR values greater than 2 or 3, pose concern about bleeding complications of needle insertion, particularly into deep muscles and those adjacent to major blood vessels. When a significant involuntary

movement disorder such as tremor is present, it may be quite difficult if not impossible to assess insertional activity, spontaneous activity, and volitional activity by EMG. Similarly, spasticity often renders relaxation an impossibility, creating at least a very prolonged study and, at worst, an inability to assess EMG tracings without contamination by involuntary muscle activation. When an EMG subject is morbidly obese some muscles may be inaccessible, even using a 75- or 90-mm concentric needle electrode. The inability to palpate surface landmarks and detect pulses also makes EMG more hazardous than performing electrodiagnostic studies in individual with normal body mass indices.

What are the unintended consequences of performing EMG and NCS? The only recognized general effects of percutaneous NCS are the transient discomfort and apprehension associated with delivery of brief electric shocks to the skin. These stimuli, which are typically 0.01 to 1 msec in duration and between 0 and 100 mA in current strength, are felt as surprising, make the stimulated limb jerk slightly because of activation of innervated and nearby muscles, and are felt as uncomfortable to slightly painful, especially in proximal sites such as the popliteal fossa, supraclavicular fossa, neck, and mastoid region. Although most patients do not regard NCS as more than a minor discomfort, the average 10-point visual analog scale rating of 300 consecutive patients being 3, a few individuals cannot tolerate the procedure and request that testing be discontinued. With the theoretical exception that proximal upper limb stimulation in patients with indwelling central venous catheters or other artificial current paths to the heart might induce malignant cardiac dysrhythmias or activate an implanted defibrillator, there are no known long-term complications of percutaneous NCS.[19]

Regarding complications of EMG, it is common for the patient to experience transient minor soreness at one or two puncture sites—the typical study including 5 to 20 sites. The needle insertion is often painless but in most patients is felt as mild sharp pain on skin penetration and then a dull ache within the muscle. The average visual analog scale 10-point pain scale rating to the concentric needle examination in 300 consecutive EMG patients was 3. A few cannot tolerate the procedure and request that the study be discontinued. A few individuals sustain a small hematoma, usually at one site, from inadvertent puncture of a nearby blood vessel; this can be minimized by a detailed knowledge of vascular anatomy and by palpating arterial pulses in the region of intended puncture sites in order to avoid large vessels. Rarely, a larger hematoma occurs, sometimes from puncture of the radial artery at the wrist, an artery in the antecubital fossa, or the femoral artery in the inguinal region.[19] There are no prospective data on the rates of occurrence of this complication. The risk of bleeding is greater in patients taking anticoagulants, those who have a coagulopathy of other causes, and those with marked thrombocytopenia. Many laboratories have guidelines concerning what degree of anticoagulation and what platelet count range are acceptable to perform EMG. Another concern in patients with bleeding tendencies is compartment syndromes caused by vessel puncture during EMG. Performing more than 25,000 EMG studies over 19 years, the author's laboratory has never seen either a large hematoma or a compartment syndrome requiring surgical intervention. There is a small risk of pneumothorax when performing EMG of the diaphragm and other thoracic muscles, although the author's laboratory has not had a symptomatic pneumothorax in more than 200 diaphragm,[20] paraspinal, and other chest wall EMG punctures over the last decade. Diaphragm EMG is not performed in patients with flat diaphragms by chest radiography or in those with hyperexpansion from chronic obstructive pulmonary disease and other conditions.

Although infection precautions are the customary practice in modern electrodiagnostic laboratories, there are few if any data regarding the incidence of infection associated with EMG. In most clinical settings disposable electrodes are used for all routine EMG studies. Platinum single-fiber EMG electrodes are sterilized by gas or autoclave employing the same methods used for surgical instruments. In addition, special precautions, including use of disposable NCS electrodes, are taken with patients known to be infected with agents such as hepatitis B virus, hepatitis C virus, Creutzfeldt-Jakob disease, and human immunodeficiency virus.

EMG REPORTING

After having read hundreds of EMG reports written at scores of laboratories throughout North America over the last two decades, it is apparent that many electromyographers have difficulty putting together succinct, clearly written summaries and interpretations. Some of this problem may stem from a desire to report on every finding in order to be complete. Another possible reason may be that some electromyographers are uncertain whether a given result is or is not clinically significant and therefore conclude that if every tidbit of information is cataloged in laundry list fashion, no important observation will be excluded even if several superfluous or unimportant details end up cluttering the report. The EMG report should be terse, to the point, and emphasize clinically relevant findings.[18]

Another shortcoming of many EMG reports is failure to put the findings in a clinical context. Glibly writing noncommittal generalizations and adding a "suggest clinical correlation" at the end in essence says to the referring physician, "I didn't have time to discuss the history with the patient or do a brief examination.

Here are the EMG results. It is up to you to decide whether they make sense or not." A far more useful approach is to summarize the abnormalities concisely, list any pertinent additional history or physical findings that the electromyographer elicits or observes at the time of the EMG, and finally make a determination of whether or not the findings explain the patient's symptoms and signs.

What follow are a few examples of pairs of EMG reports. Each pair consists of a suboptimally crafted summary and interpretation (reports 1A, 2A, and 3A) and then a revised, more useful summary and interpretation of the same patient's EMG visit (reports 1B, 2B, and 3B).

Example 1: The Cluttered Noncommittal Report

Referral indication: paresthesia and pain.

REPORT 1A

Summary The left median antidromic sensory response amplitude was 10 μV (normal greater than 15 μV) with a conduction velocity of 53 m/s (normal greater than 54 m/s) and a distal latency of 4.5 ms (normal less than 3.6 ms). The left ulnar antidromic sensory response was 5 μV in amplitude (normal greater than 10 μV) with a conduction velocity of 51 m/s (normal less than 53 ms) and a distal latency of 3.3 ms (normal less than 3.2 ms). The left median/APB motor amplitude was 4.2 mV (normal greater than 4 mV) with conduction velocity of 49 m/s (normal greater than 48 m/s), a motor distal latency of 5.2 msec, and an F wave latency of 30 ms (normal less than 32 ms). The left ulnar/ADM motor amplitude was 6.1 mV (normal greater than 6 mV) with a conduction velocity of 47 m/s (normal greater than 51 m/s), a motor distal latency of 3.4 ms (normal less than 3.6 ms), and an F wave latency of 29.7 ms (normal less than 33 ms). The left fibular/EDB motor response was 1.0 mV in amplitude (normal greater than 2.0 mV) with a conduction velocity of 38 m/s (normal greater than 41 m/s), a motor distal latency of 5.0 ms (normal less than 6.6 ms), and no elicitable F waves. The left sural sensory response was 2.2 μV in amplitude (normal greater than 6 μV) with a distal latency of 4.6 ms (normal less than 4.5 ms). Concentric needle examination showed large motor unit potentials in the left first dorsal interosseous, abductor pollicis brevis, tibialis anterior, and medial gastrocnemius muscles with fibrillation potentials in the abductor hallucis muscles on both sides and a single train of positive sharp waves in the left low lumbar paraspinal muscles.

Interpretation The EMG findings suggest either median ulnar, fibular, and tibial mononeuropathies (multiple mononeuropathies), polyneuropathy with superimposed carpal tunnel syndrome, polyradiculoneuropathy, or motor neuron disease with an additional sensory neuropathy. Multilevel cervical and lumbosacral radiculopathy or plexopathy cannot be completely excluded. Suggest clinical correlation.

Author Comment Regurgitation of detailed individual data elements with no summary or pattern recognition. Noncommittal interpretation with no attempt at correlating the findings with the patient's symptoms and signs.

REPORT 1B

Summary NCS showed low-amplitude sensory and motor responses with borderline NCVs and disproportionate prolongation of the left median sensory and motor distal latencies. Concentric needle examination demonstrated mild distal MUP enlargement accompanied by low-grade irritability and fibrillation potentials limited to intrinsic foot muscles. The patient reports no hand symptoms whatsoever. Tinel sign absent over the median nerves at the wrists. Mild left thenar atrophy noted of which the patient was unaware.

Interpretation The EMG findings suggest electrophysiologically mild to moderate predominantly axonal sensorimotor peripheral neuropathy with superimposed asymptomatic left median neuropathy at the wrist.

Author Comment Summarized individual data elements with findings presented in a cohesive clinically significant pattern. Interpretation commits to a particular diagnostic formulation (polyneuropathy and subclinical median neuropathy at the wrist) correlating the findings with the patient's symptoms and signs.

Example 2: Failure to Make Pertinent Clinical Observations during EMG Testing

Referral indication: unsteadiness.

REPORT 2A

Summary NCS and concentric needle examination of the lower limbs were normal.

Interpretation Normal EMG. This study provides no electrophysiological explanation for peripheral neuropathy or any other neuromuscular explanation for the patient's unsteadiness.

Author Comment Focus is purely on the electrophysiology, ignoring obvious clinical signs and symptoms at the time of the examination.

REPORT 2B

Summary NCS and concentric needle examination of the lower limbs were normal. Every needle insertion below the knee of either side resulted in either an extensor plantar response in the lower limb or a triple response. Physical examination shower no upper limb deep tendon reflexes, markedly hyperactive lower limb reflexes, sustained bilateral ankle clonus, and bilateral extensor plantar responses. No sensory level to pin prick could be demonstrated on the torso either anteriorly or posteriorly.

Interpretation Normal EMG. Although this study does not suggest a peripheral process, the physical findings described previously are those of a bilateral central nervous system disorder affecting upper motor neuron pathways. Neurologic consultation is recommended. Results discussed by telephone with Dr. Jones.

Author Comment The electrophysiological findings are presented, but pertinent clinical observations of major importance to the neurologic evaluation are also emphasized.

Example 3: Failure to Discount the EMG Findings as Being Clinically Insignificant

Referral indication: Right hip/leg pain.

REPORT 3A

Summary NCS showed a low-amplitude right fibular/EDB compound muscle action potential and absent right fibular/EDB F waves. Concentric needle examination demonstrated large motor unit potentials in the right L5 territory both distally and proximally, unaccompanied by irritability or fibrillation potentials in the leg, hip girdle, or lumbar paraspinal muscles.

Interpretation The EMG findings are those of right L5 radiculopathy.

Author Comment Although the EMG and NCS findings are correctly described and interpreted, the electromyographer does not correctly weave them into the prior clinical history and take into account other possible explanations for the current symptoms.

REPORT 3B

Summary NCS showed a low-amplitude right fibular/EDB compound muscle action potential and absent right fibular/EDB F waves. Concentric needle examination demonstrated large motor unit potentials in the right L5 territory both distally and proximally, unaccompanied by irritability or fibrillation potentials in the leg, hip girdle, or lumbar paraspinal muscles. The patient reported having an episode of severe low back and radiating right lower limb pain 12 years ago associated with a transient right foot drop. The problem resolved spontaneously over 3 to 4 months. Physical examination currently shows normal lower extremity strength and marked pain on flexion abduction and external rotation of the right hip.

Interpretation Although the EMG findings provide evidence of electrophysiologically old inactive right L5 radiculopathy, the current symptoms are perhaps more likely to be due to mechanical disease of the right hip joint.

Author Comment The same NCS and EMG observations are made but this time with the added depth of relevant past medical history and current physical examination abnormalities that lead to the correct diagnosis.

CONCLUSION

The EMG and NCS are not a set of tests to be performed in isolation and reported without consideration of the clinical context but rather form part of what has been referred to as the electrodiagnostic consultation. The electrodiagnostic consultation uses all of the pertinent information available to the electromyographer performing the test with the goal of helping the patient and the referring physician to establish a correct diagnosis. EMG as an extension of the clinical history and physical examination can be a powerful and sensitive diagnostic tool. As with any tool, however, the final result depends on the skill and expertise with which it is wielded.

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