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A Reappraisal of The Utility of Needle Electromyography In Low Back Pain: An Observational Retrospective Study

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Abstract

Objective: Needle electromyography (EMG) assesses the function of the motor unit components of the peripheral nervous system. While EMG is effective in evaluating muscle and motor neuron disorders, its value in the assessment of pain per se and of sensory nerve components requires reappraisal. This observational retrospective study, performed by a practicing neurologist and an orthopedic surgeon, examined its utility in the evaluation of low back pain (LBP) with and without neurological symptoms and deficits (NSDs).

Methods: We reviewed the EMG findings concerning 150 patients (100 males, 50 females; age range, 25-65 years) who had been referred by various health care providers for evaluation of post-traumatic LBP. All patients underwent plain radiographs and magnetic resonance imaging (MRI) of the lumbosacral spine, which showed various degrees of intervertebral disc displacements.

EMG examinations were performed from two to six months following the onset of LBP. None of the patients had prior LBP or injury. The patients were categorized into three groups: 1) LBP confined to the lower back or extending to the buttocks or hips (60 patients); 2) LBP associated with unilateral or bilateral sensory symptoms in the thighs or legs, and sometimes the feet, without NSDs (50 patients), and; 3) LBP associated with NSDs in the lower limb, unilaterally or bilaterally (40 patients).

Results: The EMG findings in Group 1 were normal. All but 10 patients in Group 2 showed normal findings. In Group 3, all EMG findings were abnormal, including one patient with cauda equina syndrome secondary to a large herniated disc.

Conclusion: EMG in LBP is predictably abnormal in patients with clear and unequivocal NSDs. EMG for such patients—with or without MRI findings—is debatable, especially if the main disabling symptom is LBP. This study underscores the importance of a good history and a meticulous physical examination to maximize the value of EMG, and to bring awareness to some health care providers that EMG cannot assess low back pain directly, and it only reflects the abnormalities in the motor unit components of the spinal nerve roots.

Keywords: Back pain, Musculoskeletal pain, Vertebral pain generators, Peripheral neuropathy, Electromyography, Herniated disc, Radiculopathy, Sinuvertebral nerves

Introduction

Low back pain (LBP) is a very common symptom that brings patients to various health-care providers. It often leads to protracted pain management and long-term disability [1,2]. Healthcare costs can be considerable[3], not to mention the cost of legal proceedings related to personal and work- and vehicular accident–related injuries. Despite the advent of diagnostic imaging and electrophysiological test procedures, localization of the causes and treatment of LBP are not straightforward. At times, the lack of correlation between the clinical presentation and the results of the diagnostic test procedures, particularly electromyography (EMG), can lead to diagnostic confusion and unfavorable outcomes for certain treatment modalities, including surgery. However, when associated with neurological symptoms and deficits (NSDs) such as paresthesia and dermatomal sensory loss, focal muscle weakness affecting the specific myotomes, and loss of the muscle stretch reflex—combined with abnormal EMG findings, a diagnosis of concurrent radiculopathy or nerve root irritation becomes unequivocal.

It is typically assumed that when NSDs or EMG abnormalities are absent, the spinal nerve root is not compromised because of a spinal lesion, particularly due to a herniated disc (HD). However, regardless of the absence of such abnormalities and laboratory findings, LBP can sometimes be overwhelming and disabling. It can be disconcerting to patients and some health care providers unfamiliar with the intricate details of the test procedure to receive a normal EMG report in the setting of severe LBP with or without imaging abnormalities, which may sometimes lead to further medical and legal complications. Moreover, a normal EMG may lead some health care providers and insurance companies to attach little importance to the significance of LBP. We evaluated the utility of EMG in LBP, with or without clear radicular deficits, through conducting an observational study of 150 EMG examinations performed over the preceding past five years.

Our study aimed to help develop awareness of the relationship between LBP with or without NSDs and EMG abnormalities, and to draw attention to the significance of that relationship in relation to anatomical changes revealed using imaging studies. Therefore, this study is likely to have important therapeutic, prognostic and economic implications.

Methods

The EMG findings of 150 patients (100 males, 50 females) aged 25-65 years who had been referred for evaluation of post-traumatic

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LBP were reviewed. These patients were selected from a group of 500 patients examined by the authors over the preceding five years, from 2015 to 2019. Patients were referred to us by various healthcare providers, including family physicians, internists, physician assistants, family nurse practitioners, podiatrists, chiropractors, and surgical specialists with expertise in spine management (neurosurgeons and orthopedic surgeons), and were examined prior to and during the procedure. Using the Cadwell (Kennewick, Washington, USA) Sierra II Wedge NCV System, studies were performed by RPL, a board-certified neurologist and electromyographer, from two to six months following the onset of LBP. Musculoskeletal examinations, including evaluations of patients who sought a second opinion, were performed by TSE, a board-certified orthopedic surgeon. Unilateral or bilateral EMG sampling using monopolar needle electrodes was performed on a minimum of five muscles for each limb, including the paraspinal muscles. One motor and one sensory nerve conduction study (NCS) and an H-reflex examination were performed in some patients using standard techniques for transcutaneous nerve stimulations. EMG abnormalities were defined as the presence of muscle membrane irritability (sharp positive waves evoked through needle electrode insertion) and spontaneous activities (fibrillation potentials). These spontaneous activities were frequently associated with the presence of complex long-duration and high-amplitude motor units that were, at times, associated in turn with time-locked satellite potentials. These abnormalities were always accompanied with a reduction in the number of motor units activated during contraction, along with the presence of rapid firing units. The presence of normal resting activities, rare complex and normal-duration motor units with normal amplitude, and full activation of motor units in all muscles, was considered normal examination. F-wave examinations involving long-latency action potentials elicited through supramaximal stimulation of the peripheral nerve were not routinely performed, as these action potentials are generated through motor nerve fibers, not sensory fibers, and would, therefore, not have provided direct information on the origin of the sensory symptoms, particularly LBP. Moreover, receiving a large amount of electric current to elicit a response can be very uncomfortable for patients already suffering from pain.

Previously healthy patients and those without a history of diabetic neuropathy, cerebrovascular accident, concurrent acute and chronic musculoskeletal injuries affecting the lower extremities, active malignancy, cervical and thoracic myelopathies, spinal tumor, syringomyelia, previous lumbar spine surgery, Lyme disease, hereditary or acquired neuromuscular disorders, and connective tissue or collagen vascular disorders, were included in the study. The patients with those aforementioned conditions were excluded to ascertain whether the origin of NSDs and EMG abnormalities was related to low back injury rather than to concurrent medical disorders. One female patient who developed cauda equina syndrome secondary to a large herniated disc from a work-related injury and who was diagnosed with hereditary polyneuropathy during the EMG and NCS procedures, was included in the study.

The included patients were categorized into three groups (Table 1) 1) LBP either localized or extending to the buttocks or hips (60 patients); 2) LBP associated with paresthesia, with or without nonlocalized radiating pain, and a vague and diffuse numb-like, sometimes cool, sensation in the lower extremities unilaterally or bilaterally with no NSDs (50 patients); and 3) LBP associated with NSDs consisting of dermatomal sensory loss, myotomal muscle weakness and loss of muscle tone, and loss of muscle stretch reflex (40 patients). Focal muscle atrophies in Group 3 were evident in some cases examined months after the injury. None of the patients had prior low back pain. Work-related injuries were identified in 48 patients, injuries due to vehicular accidents in 32 patients, and personal and household injuries in 43 patients. No clear proximate causes of injury were identified in 27 patients. The magnetic resonance imaging (MRI) findings (Table 1) included bulging, protruding, and herniated discs, present at either one or several levels with or without associated disc desiccation and osteophyte formation. No patients had mass lesions, fractured vertebrae, significant spondylolisthesis, or congenital abnormalities. In Group 1, bulging and protruding discs were found in 50 patients, while 10 showed HDs at two levels with root impingement at one level in three patients. In Group 2, 10 patients had HDs, two patients at two levels and eight patients at one level, with root impingement at two levels in two patients, and at one level in three patients. Bulging and protruding discs at two or three levels, without root impingement, were observed in 40 patients. In Group 3, all patients had HDs: 30 patients at one level and 10 patients at two levels. Root impingements at one level (28 patients) and at two levels (12 patients) were found in this group, including the patient with cauda equina syndrome.

We did not specifically address the correlation between the outcome of surgery and the presence or absence of EMG abnormalities and HDs, as this subject matter is a separate issue beyond the scope of this study. Likewise, we elected to exclude EMG examinations of neck pain because the generation of pain by the sinuvertebral nerves (SVNs) and by the small nerve fiber receptors in the facet joints, including the myofascial tissues surrounding the vertebral and shoulder girdle muscles, may be theoretically influenced by the cervical spinal cord, regardless of whether it has been grossly traumatized or not.

Upon completion of our clinical research, we submitted the rough draft of the manuscript to ADVARRA IRB in Columbia, Maryland for advisory review. It was determined that based on the information provided, the research would have met the criteria for exemption from IRB review under 45 CFR 46.104(d)(4). It was deemed unnecessary to obtain written informed consent from patients described in this study, due to its retrospective design.

Results

All patients in Group 1 had normal EMG findings. In Group 2, abnormalities were found in only 10 of the 50 patients. Abnormalities were found in all patients in Group 3. No patients had clinical signs of peripheral neuropathy, and all NCSs were normal. The female patient who developed cauda equina syndrome secondary to a large herniated disc was diagnosed with concurrent but asymptomatic hereditary sensory and motor polyneuropathy while undergoing EMG and NCS evaluations. Her anal sphincter EMG showed active denervation, while the H-reflex was absent on both sides. The EMG abnormalities in Group 3 could be linked to the affected nerve root but no correlation was found with the level of vertebral injury. We also found that the presence of an HD or root impingement, as revealed using MRI, did not necessarily lead to NSDs and abnormal EMG findings, as shown in the results from all the patients in Group 1 and 40 patients in Group 2. Likewise, the presence of multilevel disc displacements, particularly HDs, did not result in polyradiculopathy, as all patients with EMG abnormalities showed monoradiculopathy only.

	Group 1 (n = 60)	Group 2 (n = 50)	Group 3 (n = 40)
NSDs	Absent	Absent	Present
EMG abnormalities	Absent	Absent in 40 patients Present in 10 patients L4 in 2 patients L5 in 5 patients S1 in 3 patients	Present in all patients L3 in 5 patients L4 in 4 patients L5 in 21 patients S1 in 10 patients (absent H-reflex, unilateral in 9 patients, bilateral in 1 patient)
MRI abnormalities	Present Disc bulge and protrusion at 3 levels in 50 patients, HD at 2 levels in 10 patients, and with root impingement at 1 level in 3 patients	Present Disc bulges and protrusions in 40 patients, HD at 2 levels in 2 patients and at 1 level in 8 patients, with root impingement at 2 levels in 2 patients, and one level in 3 patients	Present HD in 40 patients At 1 level in 30 patients 2 levels in 10 patients, with root impingement at 1 level in 28 patients and at 2 levels in 12 patients

Table1: A summary of clinical, EMG, and MRI findings.

EMG: electromyography; HD: herniated disc; MRI: magnetic resonance imaging; NSDs: neurological symptoms and deficits

Discussion

The hallmark of spinal nerve root impingement due to a herniated disc is a constellation of NSDs affecting a corresponding dermatome and myotomes in association with EMG abnormalities. This has been axiomatic and has been taught to generations of medical students and residents. Although these NSDs frequently occur alongside LBP, in some cases, either one may dominate the overall clinical symptomatology. It is noteworthy that numerous cases of LBP are associated with symptoms in the lower extremities that are primarily sensory, with no clear dermatomal distribution or gross or demonstrable focal muscle weakness and loss of muscle stretch reflexes, and without EMG abnormalities, as was observed in the Group 1 and 2 patients. It is intriguing that the disc displacements, involving bulging or protruding and herniated discs, as shown through MRI in these groups, had no clear clinical or EMG correlations. One common denominator among these cases, however, is LBP, a condition distinct from radiculopathy. An analogy to neck pain can be drawn, which should not be confused with cervical radiculopathy; the former (neck or low back pain) is perceived in the spine, while the latter is perceived in the affected extremity [4].

Physiologically, EMG is a recording of motor unit activity[5]. Although its usefulness in the diagnosis of muscle and motor neuron disorders is well established, it cannot assess the function of small- and large-diameter sensory nerve fibers directly. Therefore, it will likely yield negative results when performed in patients with musculotendinous-ligamentous pain and arthralgias, unless these conditions are associated with peripheral nerve lesions [6]. Similarly, diffuse LBP generated primarily through the SVNs, which provide the sensory innervation to the annulus fibrosus of the intervertebral discs, the ventral surface of the dura mater, the periosteum of the spinal canal, facets, and the dorsal and ventral longitudinal ligaments [7-11], will also yield a negative EMG examination result, unless concurrent spinal nerve root compromise is present.

In Group 3, the presence of NSDs was always associated with EMG abnormalities. Except for depressed knee and ankle muscle stretch reflexes due to an involvement of the mid-lumbar and S1 nerve roots, respectively, impingement of the L5 nerve root is diagnosed through the presence of drooping of the big toe [12], due to weakness of the extensor hallucis longus muscle, a segment-pointer muscle for the L5 nerve root, together with dermatomal sensory deficits. Although foot drop resembling peroneal mononeuropathy can also occur, this is easily distinguished through the clinical history and EMG, in conjunction with a nerve conduction examination. All patients in Group 3 exhibited these clinical abnormalities, which raises a question concerning whether performing an EMG in such cases is necessary.

It is worth emphasizing that LBP and radiculopathy are usually mutually inclusive in most cases of spine trauma. This is understandable, as the mechanical stress exerted on the spine through trauma will likely irritate the SVN and the sensory nerve endings in the myofascial compartment, resulting in LBP. When the intervertebral disc is displaced, the spinal nerve is liable to be compressed, resulting in NSDs. All patients in Group 3 experienced this combination of LBP and NSDs from disc displacements or degenerative changes. However, LBP and NSDs can also be mutually exclusive during spontaneous recovery or following conservative therapy or surgical intervention. We have encountered several patients whose LBP recovery lagged behind that from an NSD and vice versa.

Most patients are referred to neurologists or physiatrists for a needle EMG examination, a procedure that is understandably uncomfortable and painful, and much more so for patients already suffering from LBP. Although iatrogenic complications such as bleeding and hematoma formation can be problematic, EMG is generally considered a safe procedure [13-15]. It is debatable whether some patients should be subjected to multiple needle EMG punctures despite the absence of NSDs or imaging abnormalities, in seeking to confirm radiculopathy. Similarly, examination of the paraspinal muscles, which are routinely sampled despite tenderness or spasms, can be discomforting for some patients. However, to determine the significance of an HD and its effect on the nerve root, paraspinal EMG provides incontrovertible evidence for the presence of radiculopathy [16], unless the patient has a history of polymyositis, an inflammatory muscle disease with the most significant spontaneous potentials in the paraspinal muscles [17]. However, as we observed in our Group 3 patients, the presence of NSDs in an appropriate clinical context and abnormal MRI is always associated with abnormal EMG findings in the anterior myotomes and paraspinal muscles. For this reason, it is debatable whether it is necessary to perform EMG in such cases.

Ten patients in Group 2 had no clear NSDs but HDs or various disc desiccations were observed that showed EMG abnormalities. Such occurrences may perhaps be explained as due to anatomical variability, the degree of neural impingement, or an inaccurate description of the symptoms on the part of the patient. The possibility of inadequate muscle sampling on the part of the electromyographer cannot be ruled out either. Differences in experience levels and variability in the interpretation of abnormalities, regardless of the place of practice (whether in an academic setting or community based) are also worthy of consideration. Nevertheless, the need to perform EMG in these patients is warranted, depending on the clinician's skill and acumen. Why such sensory symptoms would occur without true focal muscle weakness in the presence of vertebral abnormalities in imaging

examinations, as in our Group 2 patients, requires elucidation. Some of our patients with this type of clinical presentation in the cervical and lumbar spine had been involved in motor vehicle or work-related accidents. Some patients reported an intermittent vague numb-like and cool sensation and sometimes red discoloration or pallor in the affected extremity without allodynia (personal observation by the authors). Such symptoms are likely generated through the sympathetic nerve components of the SVN, which originate from the rami communicants' and provide the afferent pathways of discogenic low back pain. In such cases, thermal imaging (also known as thermography), a non-invasive but non-localizing procedure, albeit controversial, can be useful in demonstrating the effect of sympathetic nerve dysfunction in the upper and lower extremities [18]. This procedure was not performed in our study.

Our study had limitations. It was a retrospective study, and the number of patients included was small; hence, our findings should be considered with caution. Moreover, the patients in Group 1 and 2 patients with HD's, if followed for an extended period, might have developed clinical and EMG signs of nerve root impingement as the stress of activities of daily living can have a cumulative effect on an already compromised spine. Long-term follow-up evaluations, which would have been useful, were not performed in this study.

Conclusion

We concluded that EMG abnormalities only reflect the pathology in the nerve root, and only if the motor fibers are affected. EMG cannot assess the function, or localize and identify the various pain generators, of the lumbar spine. These pain generators are mediated through the small nerve fibers that contain substance P in the facet joints and free nerve endings in the annulus fibrosus of the discs and longitudinal ligaments and, [19-22] in many patients, LBP can persist even after resolution of NSDs, resulting in long-term disability. The value of EMG is more effectively realized when combined with a good clinical history and clinical assessment, or with an NCS, if there are concurrent clinical symptoms of peripheral neuropathy. EMG is an extension of the physical and neurological examination process. In modern-day health care, cost containment and paying close attention to patient comfort are of considerable practical relevance and the results of this study offer guidance in that regard.

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