LETTERS TO THE EDITOR

THE PREVALENCE OF LUMBAR PARASPINAL SPONTANEOUS ACTIVITY IN ASYMPTOMATIC SUBJECTS

Much damage may be done by the inappropriate conclusions drawn by Date et al.[1] in their study of the paraspinal muscles in persons without low back pain.

I accept that the current study demonstrates an increased prevalence of abnormal spontaneous activity with age in their population, and that it has confirmed our work demonstrating that some reproducible spontaneous activity does occur in persons without back pain.

The authors claim that their finding of a 19.5–50% chance of reproducible spontaneous activity suggests that electromyography (EMG), like magnetic resonance imaging and computed tomography, suffers from a high percentage of "false positives," and thus cannot reliably differentiate between persons with radiculopathy and normals. We have already addressed that problem in our own study of persons without back pain.[2] When the abnormalities are quantified, there is a statistically significant and clinically obvious difference between normals and persons with demonstrated spinal pathology. Our subsequent study of over 110 patients will confirm in more detail the ability to differentiate normal from symptomatic (submitted, AAEM, 1996).

A second problem is the use of a nonvalidated method of assessing the paraspinal muscles. Despite the presence of a review article[4] and at least four different protocols which both quantify the extent of abnormalities and have been tested in persons with pathology,[5,7–9] the authors struck out on their own, using unreferenced guides from a minimonograph and a textbook. Because our technique, Paraspinal Mapping, has been the subject of a considerable amount of work, including blinded anatomical validation,[3] radiologic verification,[6] prospective assessment in a population of patients with mixed pathology,[5] and blinded testing in normals with interobserver correlations,[2] we wish it had been chosen by the authors, instead. But the authors state that they were unaware of any study which has evaluated the back muscles of normals. This seems improbable, given the rather high profile of our own work which has received three national awards in the years prior to publication of the author's work.

In conclusion, we recommend that readers accept the article as a confirmation of previous work, demonstrating the presence of abnormal spontaneous activity in "normal" backs. It is no longer valid to judge a few fibrillation potentials in the back as evidence for radiculopathy. But there are better documented techniques for quantifying paraspinal EMG which accurately differentiate between normal and abnormal—and maintain EMG's unique niche in the era of modern imaging.

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THE PREVALENCE OF LUMBAR PARASPINAL SPONTANEOUS ACTIVITY IN ASYMPTOMATIC SUBJECTS (A REPLY)

I respectfully disagree with Dr. Haig's comment that "much damage may be done" by our article. The results from our article do not diminish the utility of the electromyographic study in the workup of low back pain, but do indicate that the paraspinal needle study can be abnormal in the older individual without low back pain. This correlates well with findings of degenerative disease in the lumbosacral spine as a natural part of aging. Therefore, the article simply cautions against drawing substantial conclusions when there are positive sharp waves only in the paraspinal muscles in patients over 40 years.

The article which Dr. Haig feels was overlooked[1] in the discussion of our article is not entirely relevant in my opinion. The article was published at the time that our article was submitted. The 35 normals which Dr. Haig and colleagues used in their study had a mean age of 32.56 years, with zero patients over the age of 58 years. Certainly, this is not comparable in terms of age ranges to our study.

I also feel that the use of the techniques to examine paraspinals which are documented in the works of internationally recognized electromyographers, Dr. Jun Kimura[3] and Dr. Asa Wilbourn and Dr. Michael Aminoff[4] are not appropriately described by Dr. Haig as "unreferenced guides from a minimonograph and a textbook." The techniques used in our study are practical and used by many clinicians universally; therefore I feel that our article has wide applicability. Although the technique of paraspinal mapping has been published previously by Haig et al,[1,2] its clinical utility has not been prospectively demonstrated in any of his cited publications. In the earlier article,[2] paraspinal EMG findings in low back pain patients were compared to pain drawings, various spinal imaging tests, and leg EMG findings. Fibrillations potentials were observed in the legs of 61% of 45 subjects, all of which had paraspinal abnormalities. There was no evaluation of specificity or sensitivity of the paraspinal EMG technique compared to surgical findings, standardized magnetic resonance imaging diagnostic criteria, clinical signs, or electrodiagnostic criteria for a single level radiculopathy. In the later study in 35 normal subjects,[1] paraspinal fibrillations scores are compared to 17 selected patients from a previous study with "spinal pathology" (no average age given of the spinal pathology patients). There is a large overlap of scores between spinal pathology patients[2] and the normal subjects,[1] with approximately 50% of spinal pathology subjects falling into the retrospectively established normal range, and nearly 100% falling into the prospectively established normal range.

Dr. Haig and associates incorporate the concept of unisegmental innervation of the medial multifidus paraspinal muscles into their work; investigators at our Center have since performed electrophysiologic studies in normals, paraplegics, and a high lumbar radiculopathy patient which have demonstrated polysegmental innervation of this musculature. This article has been submitted to *Muscle and Nerve* for review.

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FREQUENCY OF MEDIAN MONONEUROPATHY IN PATIENTS WITH MILD DIABETIC NEUROPATHY IN THE EARLY DIABETES INTERVENTION TRIAL (EDIT)

Dr. Albers et al. report on the "Frequency of median mononeuropathy in patients with mild diabetic neuropathy in the early diabetes intervention trial (EDIT)".[1] They eventually face the paradox that patients with diabetic neuropathy have so many electrophysiologic abnormalities that it is difficult to distinguish which diabetic patient does or does not have carpal tunnel syndrome based on electrophysiologic study.

Capobianco and I tried to figure out the significance of slowing of orthodromic motor conduction velocity across the elbow in diabetics. By measuring slowing across the elbow relative to velocity in the upper arm or brachium, we were able to devise criteria for ulnar neuropathy in otherwise healthy patients with ulnar neuropathy due to elbow lesions only; and patients with various causes of diffuse neuropathy (but not diabetic) with and without ulnar neuropathy (Fig. 1 in Ref. 2). However, when we tried to devise criteria for patients with diffuse *diabetic* neuropathy, we ran into a brick wall. Patients with diabetic neuropathy but no superimposed clinical ulnar neuropathy had nearly as much relative slowing across the elbow as patients with

diabetic neuropathy and superimposed clinical ulnar neuropathy.

Ernest Johnson[3] has suggested relative criteria for diagnosis of carpal tunnel syndrome in patients with underlying diabetic neuropathy, including comparisons of distal latency with proximal conduction, median with radial or ulnar nerve conduction, carpal tunnel segment with more distal segment, and the duration of the negative spike of the compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) stimulating proximal and distal to the carpal tunnel. These criteria are rational, though each requires validation by formal controlled study comparing patients with diabetic neuropathy with and without clinical carpal tunnel syndrome.

Given the high frequency of electrophysiologic abnormalities in patients with diabetic neuropathy, particularly at common sites of entrapment or pressure damage, we might not be able to use electrophysiologic criteria as the gold standard of localizing nerve damage or diagnosing or even confirming syndromes such as the carpal tunnel syndrome. Our electrophysiologic criteria may be only *permissive,* in that, if present, they may *permit* the clinical diagnosis to be made. In Aristotelian terms, focal electrophysiologic abnormalities may be necessary, but not sufficient, to diagnose a syndrome such as the carpal tunnel syndrome. Unfortunately for the patient, if the electrophysiologic abnormalities do not represent the sole and sufficient cause of the focal nerve damage, treating the focal nerve damage will not help the patient.

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FREQUENCY OF MEDIAN MONONEUROPATHY IN PATIENTS WITH MILD DIABETIC NEUROPATHY IN THE EARLY DIABETES INTERVENTION TRIAL (EDIT) (A REPLY)

We enjoyed reading of Dr. Hawley's efforts to define criteria for conduction slowing across the elbow in ulnar neuropathy, and the difficulty he encountered in applying the resultant criteria to patients with diabetes mellitus. The high frequency of abnormal ulnar conduction studies he found in diabetic patients asymptomatic for ulnar neuropathy is analogous to our demonstration of frequent median conduction abnormalities suggestive of median neuropathy at the wrist in diabetic patients asymptomatic for carpal tunnel syndrome. We would add that we did evaluate the use of relative criteria (e.g., median minus ulnar sensory latencies) in establishing the presence of focal lesions. While these comparison are rational and of demonstrated diagnostic importance, the performance of the relative criteria was disappointing because of the continued high frequency of abnormal responses in diabetic patients. The suggestion that "normal values" for focal electrodiagnostic measures are needed for diabetic subjects is a logical extension of these observations, and such values are required to establish the sensitivity and specificity of electrodiagnostic studies. We particularly like and agree with Dr. Hawley's conclusion that appropriate nerve conduction abnormalities are best thought of as required to establish a diagnosis of carpal tunnel syndrome, but are permissive only and insufficient to establish the diagnosis. This promotes rational interpretation of electrophysiologic abnormalities in the context of the overall evaluation, viewing nerve conduction measures as continuous variables useful in defining the magnitude of abnormality, as opposed to viewing electrodiagnostic results as discrete "normal vs. abnormal" values.

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DERMATOMAL/SEGMENTAL SOMATOSENSORY EVOKED POTENTIAL EVALUATION OF L5/S1 RADICULOPATHIES

In their recent article,[3] Drs. Dumitru and Dreyfuss show further evidence supporting the low sensitivity of both segmental and dermatomal somatosensory evoked potentials (SEPs) in the evaluation of L5/S1 radiculopathies (LSR). The data are convincing and are consistent with other similar studies that used strict clinical and electrophysiologic criteria.[1,2,4,5] It is true that there is no clearly established "gold standard" for the diagnosis of LSR, and this complicates testing of the diagnostic utility of various electrophysiologic techniques.

Ideally, the diagnosis should be confirmed by a definitive test or procedure (i.e., surgical or radiologic evidence of root compression), and then a new or unproven tech-

nique may be compared to one or more alternative, usually more established, techniques. By requiring a combination of strict clinical, electrophysiologic, and radiologic *criteria*, the authors have confirmed the diagnosis of LSR at the expense of biasing the population toward severe disease. It is possible that the SEP techniques studied have greater utility in less severe disease, although the low sensitivity shown for severe disease in their study makes this unlikely. More importantly, their study design precludes a comparison of segmental and dermatomal SEPs with the established technique of needle electromyography or tibial H-reflex latency, since abnormalities in the latter tests were part of the inclusion criteria. Such a comparison is important because the diagnostic utility of a test is evident not only when its sensitivity and specificity are high but also when the test being studied shows true positive results in instances where the comparative test is false negative. One might expect that intraoperative findings at diskectomy best approach a "gold standard," though this is not clearly established and such an approach would also skew the study sample toward severe disease.

Similar, rigorous studies have used a combination of clinical and radiologic features to define "clinically definite" LSR.[2,6] This approach is preferable because it does not skew the patient population toward severe disease and it allows for a comparison of two or more electrophysiologic tests. Additionally, the combination of characteristic clinical features with radiologic evidence of root compression at the appropriate level is highly suggestive of LSR, despite the potential shortcomings indicated by the authors.

I also wanted to raise an unrelated issue. Was the study prospective as suggested by patient informed consent and institutional review board approval, or were records reviewed?

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DERMATOMAL/SEGMENTAL SOMATOSENSORY EVOKED POTENTIAL EVALUATION OF L5/S1 RADICULOPATHIES (A REPLY)

We would like to thank Dr. Scelsa for his comments regarding our investigation[3] and welcome this opportunity to respond. Dr. Scelsa raises two issues: (1) the existence of multiple other somatosensory evoked potential (SEP) studies providing "strict" criteria clearly establishing that a select group of individuals have a lumbosacral radiculopathy;[1,2,4,5] and (2) that a procedure or test such as surgical visualization or radiologic imaging provides "definitive" evidence of root disease.

It is our supposition that the investigation we performed provided unquestionable evidence of an L5 or S1 radiculopathy present at the time of the study, which other studies[1,2,4,5] attempted but only in part succeeded to do. Additionally, we assessed both dermatomal and segmental somatosensory evoked potentials, which no other study has examined in the same patient population. Neither surgery nor imaging studies (structural assessments) used by prior studies[1,2,4,5] provide "definitive" physiologic evidence of conduction block, axonal loss, or demyelination/remyelination. Neural irritability, conduction block, and axonal loss can best be determined by an unquestionable correlation between objective neurologic examination findings, imaging studies showing nerve root deflection, and electrophysiolgic data demonstrating axonal loss as utilized in our study.[3] The utilization of clinical, radiographic diagnostic tools (e.g., magnetic resonance imaging, computerized tomography imaging), and even nerve root block to establish "clinically definite" disease is debatable and not beyond question as Dr. Scelsa would have us believe. We chose to add the criterion of denervation in our study to clearly establish that clinical symptoms and radiologic studies defined an axonal loss nerve root lesion. Hence, if dermatomal and segmental studies could not approach both a high sensitivity and high specificity in these patients, they certainly are of questionable value in persons with less well-defined nerve root disease.

A major area of contention is Dr. Scelsa's implication that we "biased" our investigation toward more severe disease and that, "it is possible that the SEP techniques studied have greater utility in less severe disease, although the low sensitivity shown for severe disease makes this unlikely." A number of persons have made this rather counterintuitive argument to us, i.e., that the dermatomal or segmental SEP is somehow more able to diagnose mild disease or a pure "sensory radiculopathy," but is unable to detect moderate or severe disease producing axonal loss. Our assertion is simply, if the dermatomal and segmental SEPs do relatively poorly at diagnosing unquestionable unilateral/unilevel L5 and S1 nerve root compromise, then it is unlikely SEPs will have a higher diagnostic yield in patients with mild or questionable nerve root disease.

Dr. Scelsa is quite correct in concluding that our study cannot assess the sensitivities and specificities for H reflexes and needle electromyography with dermatomal and

segmental SEPs. Our investigation was purposefully not designed to examine the comparable diagnostic abilities of the above-described electrophysiologic techniques.

Finally, our investigation was conceived and performed in a prospective manner and did not depend on a retrospective chart review.

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