

Neurophysiologists: Quirky Boffins?

It goes without saying that I am a fan of neurophysiological investigations. However I accept that the speciality of clinical neurophysiology could do with the help of a good spin doctor. Neurophysiologists appear to have acquired the reputation of quirky boffins who emerge blinking into the sunlight only occasionally for a grilling by real clinicians at the weekly neurosciences academic meeting. In this article I will attempt to review the utility of nerve conduction studies and electromyography in the management of nerve and muscle disorders. While the evidence police insist that assessment of the utility of diagnostic tests should be based on measurement of sensitivity, specificity, likelihood ratios and the like, I will take a more simplistic approach. After an EMG clinic I often ask myself the following questions:

- In which cases has the patient benefited from having electrodiagnostic tests?
- What did we discover that was not already known to the clinicians?
- Could this case have been managed without sending the patient to the EMG clinic?

This article is based on the answers to these questions with a little bit of literature thrown in to add some weight to my arguments.

Compression Mononeuropathy

For those of us who care there is an argument knocking around about carpal tunnel syndrome that goes something like this. Carpal tunnel syndrome is easily diagnosed by taking a good history and a proper clinical examination, and so sending patients for nerve conduction studies introduces additional delay and adds nothing new. This argument would have some validity if nerve conduction studies for carpal tunnel syndrome were an expensive, dangerous or invasive procedure but they are none of these. Indeed many departments of neurophysiology are trying to reduce waiting lists by introducing technician led carpal tunnel screening clinics.

However, assessing the evidence for electrodiagnostic tests in carpal tunnel syndrome is bedevilled by the lack of a universal gold standard for compression mononeuropathy. Many of studies are based on comparing the nerve conduction studies with the outcome of surgery, and whilst there are obvious disadvantages such as the placebo effect of surgery, this approach has the advantage of being practical and clinically relevant. One such study that looked at a large number of patients (c3000) demonstrated that

nerve conduction studies were a powerful predictor of the outcome of decompressive surgery (Bland JD, *Do nerve conduction studies predict the outcome of carpal tunnel decompression?* Muscle Nerve. 2001 Jul;24(7):935-40). So in my opinion there are three good reasons why all patients with CTS should be investigated neurophysiologically:

- the nerve conduction studies can grade the severity of the carpal tunnel syndrome and help predict the outcome of surgery;
- Nerve conduction studies and EMG can detect other disorders with similar presentation for example, brachial plexopathy, cervical radiculopathy and generalised neuropathy;
- If surgery for carpal tunnel syndrome fails it is very useful to have baseline preoperative tests to monitor progress and assess the effect for surgery.

Similar arguments also apply to the other compression mononeuropathies. For instance the presentation of ulnar neuropathy is very similar to lower cervical radiculopathy or lower trunk plexopathy, and these can only be distinguished with electrophysiology.

So in my opinion carpal tunnel syndrome cannot be managed without electrodiagnostic tests, even though we don't often discover something not already suspected by the clinicians. However, I will finish my argument for this approach with a *bete noire* of the evidence police, case anecdotes.

On a number of occasions I have seen patients with hand symptoms and MRI proven cervical disc disease, some of whom have had carpal tunnel syndrome and whose symptoms resolved with decompression and were thus saved from having a needless procedure.

Polyneuropathy

- To try to assess polyneuropathy without nerve conduction studies and EMG is like trying to navigate Irish roads without a route map. Nerve conduction studies can determine the extent and severity of the neuropathy. They are essential for classifying the neuropathy (demyelinating or axonal; mononeuritis multiplex or symmetrical polyneuropathy) and in some cases nerve conduction studies can confirm the exact type of neuropathy (Guillain Barre syndrome for instance). There is good evidence that the great majority of neuropathies can be successfully investigated with clinical assessment, nerve conduction studies and routine blood tests (Muscle Nerve. 2002;26:288-90. *Evaluating patients with*



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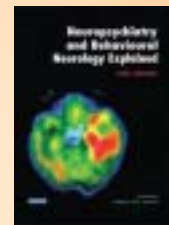
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suspected peripheral neuropathy: do the right thing, not everything. Pourmand R). In many cases there is no need for CSF examination or nerve biopsy. But do we ever find something not suspected by clinicians? You can be sure we do and here are a few examples of new diagnoses that have emerged following nerve conduction studies that I have performed:

- demyelinating neuropathies - acquired and inherited;
- anti-MAG neuropathy diagnosed as a result of disproportionately long distal motor latencies;
- a paraneoplastic sensory neuropathy missed because of an asymmetric presentation;
- mononeuritis multiplex in someone referred with Saturday night palsy and severe generalised neuropathy presenting as foot drop.

Motor Neurone Disease

The aim of electrodiagnostic studies in motor neurone disease is twofold - firstly to exclude any treatable peripheral neuropathy and secondly to help confirm the clinical suspicion of a widespread anterior horn cell disease. The first step is a fairly thorough neuropathy screen (we routinely do sural, radial, median and ulnar sensory studies and common peroneal, posterior tibial, median and ulnar motor studies, with tibial and median F wave studies). The next step is again a fairly thorough EMG examination of all four limbs with at least one other site (I favour tongue and facial muscles). To make a confident diagnosis of motor neurone disease:

- sensory nerve conduction studies need to be within acceptable limits for the age group;
- there may be some reduction in compound motor action potentials but conduction velocities need to be within acceptable limits and there should be no other evidence of demyelination such as dispersion or conduction block;
- evidence of chronic partial denervation (typically fibrillations with large, polyphasic motor units and a reduced interference pattern), and these signs ought to be in 2 of the three site examined (eg lower limbs and tongue for instance).

To be fair, rarely are results unexpected in these circum-

stances and it is my own experience that when the clinical signs are clear the neurophysiology is also clear cut. I do not routinely perform root stimulation to examine for proximal conduction block, and I would say that this uncomfortable procedure should be saved for those cases where there is no upper motor neurone signs and strong clinical suspicion of multifocal motor neuropathy with conduction block. In this last respect the criteria for MMNCB should be motor conduction block in two nerves outside of normal entrapment site (Muscle Nerve. 2003;27:117-21. *Consensus criteria for the diagnosis of multifocal motor neuropathy.* Olney RK, Lewis RA, Putnam TD, Campellone JV Jr; American Association of Electrodiagnostic Medicine). If there is conduction block in one site then it is fair to consider this as possible MMNCB and manage accordingly.

Other Conditions

EMG is an essential part of the investigation and management of myopathy, although the gold standard investigation remains muscle biopsy. EMG can confirm the presence, extent, and severity of myopathy. Sometimes there are useful clues to the aetiology for instance the presence of fibrillations with myopathic motor units can be a clue to an inflammatory myopathy. Neuromyotonia is a clinical condition defined by the presence of a characteristic sign and the first step in the investigation of myotonic disorders is the detection of myotonia on EMG. In my experience the first clue to Lambert-Eaton Myaesthetic syndrome can be the presence of characteristic features on motor nerve conduction studies and repetitive stimulation. Because single fibre EMG is a sensitive test, it can be a useful way of excluding neuromuscular junction disorders.

Conclusion

I could not be viewed as an objective judge of the role of neurophysiology, but for what it's worth I do not think it is possible to manage disorders of nerve and muscle without access to nerve conduction studies and EMG. As you can see I haven't over-endowed this piece with references but a good single textbook of EMG is by Preston and Shapiro. It is an easy read and has plenty of diagrams to explain how to perform the various tests.

Reference:

David C. Preston, MD and Barbara E. Shapiro, MD, PhD (1997) *Electromyography And Neuromuscular Disorders.* Butterworth-Heinemann

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