HOW TO GET THE MOST OUT OF NERVE CONDUCTION STUDIES AND ELECTROMYOGRAPHY

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Why should we think hard when requesting nerve conduction studies (NCS) and electromyography (EMG)? Aside from the reasons that should determine our use of all investigations there are two particular issues that relate to these tests:

- NCS/EMG is at best uncomfortable, at worst painful, for the patient—despite the neurophysiologist's best efforts
- NCS/EMG are expensive, although this cost is borne by the health care system. They take 30–60 minutes of a neurophysiologist's time, a scarce and expensive resource.

ELECTRODIAGNOSTIC STUDIES ARE AN EXTENSION OF CLINICAL EXAMINATION

Very early on in just about every textbook or article on NCS/EMG the following two observations are made:

- The tests do not replace a careful history and examination of the patient
- NCS/EMG are an extension of the clinical assessment.

These observations may seem implausible to those who do not perform these tests and view electrodiagnostic tests as a "black box" from which answers magically appear. However, neurophysiology is very definitely used in the same way as clinical examination to solve clinical problems, and complements the clinical evaluation rather than replacing it. In clinical examination you determine the site of the lesion by assessing the distribution of weakness, reflex changes, and sensory loss. Neurophysiologically you not only examine the distribution but also the type of abnormalities detected in the nerve conduction studies and EMG. Neurophysiology can be thought of as the clinical examination with the ability to "probe" nerves and muscles in a different manner.

There are a few obvious parallels with clinical examination:

- It is usually more straightforward to localise a significant weakness clinically than a milder degree of weakness where you tend to be less certain in distinguishing mild weakness from normal strength. Similarly "soft" sensory signs tend to be more difficult to localise. These same problems arise neurophysiologically—a more significant lesion is easier to localise as the neurophysiological abnormalities are more clear cut.
- If it is difficult to obtain cooperation in the clinical examination it is likely to be more so when doing neurophysiology—especially EMG which requires a high level of patient cooperation.
- When examining a patient you are often able to discount certain clinical findings that relate to known prior pathology—for example, a mild foot drop from a previous L5 radiculopathy; the neurophysiologist has to do the same so please tell them about it.
- NCS/EMG and clinical examination are both operator dependant.

NCS/EMG is particularly helpful in localising a peripheral nervous system deficit found on clinical examination. Thus:

- If you cannot frame your question in anatomical terms, which is how the neurophysiologist will try to answer it, then wonder why you are ordering the test at all.
- If you can localise the lesion with confidence on clinical examination alone, will you gain additional useful information from requesting NCS/EMG?

Often neurophysiological studies can determine the site of the lesion more precisely than examination alone, but they do not determine the cause. Usually this will require other investigations and it may be that the neurophysiological studies add nothing to the diagnosis if these other tests are diagnostically positive. For example, neurophysiological confirmation of an L5 radiculopathy may contribute little if the magnetic resonance image (MRI) of the lumbosacral spine clearly demonstrates an L5 root compression. If you are going to do other tests, consider whether NCS/EMG will add to the diagnosis. In the era of the "programmed investigation approach" remember that "block booking" tests and thus failing to appreciate the value of an investigative pathway is a poor rather than efficient use of diagnostic resources.

Before considering the role NCS/EMG play in different clinical situations it is worthwhile considering a little about the tests themselves and making some observations about how we use investigations in general.

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TESTING THE TESTS

Evidence based medicine suggests that we should understand the particular features of diagnostic tests, such as the sensitivity (the ability to detect those with disease) and specificity (the ability to detect those without). To calculate these we require the test to be compared to an accepted gold standard.

This leads to the first problem for NCS/EMG. For some conditions—for example, chronic inflammatory demyelinating polyradiculopathy (CIDP) or multifocal motor neuropathy with conduction block—NCS/EMG is an essential part of definition of these conditions making sensitivity and specificity calculations somewhat circular, though attempts are made to assess different sets of diagnostic criteria.[1] For other conditions, such as carpal tunnel[2] or ulnar neuropathy in the cubital tunnel,[3] while NCS/EMG are not the only way of confirming the diagnosis, they are probably the most robust non-invasive method. In other situations another modality can provide a more definitive diagnosis—for example, cervical radiculopathies where MRI or surgical findings can provide the gold standard.[4]

Paradoxically, this means that the conditions where there is the best published evidence on sensitivity and specificity are those where there is another more definitive method of achieving a diagnosis, this being used as the gold standard against which NCS/EMG can be compared. Cervical and lumbar radiculopathy are clear examples of this.

The next problem is that NCS/EMG is not a single investigation but an evolutionary one in which a series of tests can be applied to a clinical problem. The tests that are used will be dictated by the clinical presentation but also are adjusted according to the results of the other tests as the examination proceeds. Thus a patient referred with possible ulnar nerve lesion with weakness and wasting in the first dorsal interosseous and found to have normal ulnar motor and sensory studies prompts a search for dennervation in other T1 muscles, and next a more widespread search of dennervation that might suggest anterior horn cell disease.

As can be readily appreciated, this means that the tests are significantly operator dependant, particularly the EMG element of the examination. This process of ongoing detection reflects the skill and expertise of the operator, particularly in the assessment of more complex clinical problems. While quantitative EMG is being used more extensively, most EMG assessments depend on a large element of judgement on the part of the neurophysiologist.

NCS/EMG are not just normal or abnormal, there is a significant range of normality, which can be wide for some measures. Also there are different degrees of abnormality, from something that just falls outside the normal range, to the clear cut. Generally this will produce difficulties when deficits are subtle. Some examples:

- the absence of a medial plantar sensory response can be taken as an indication of a neuropathy—but this is also absent in 8% of normal subjects[5]
- finding fibrillation potentials in the small muscles of the feet can be interpreted as indicating a length dependant motor neuropathy—but are found in 20% of normal subjects.[6]

As can be appreciated, the specificity and positive predictive value of a very clear cut abnormality is much greater than for a milder one. By analogy, a haemoglobin concentration of 5 g/dl is clear evidence of anaemia, while a

Box 1

An illustration of how the prevalence of the condition being tested for profoundly alters the way results should be interpreted. The same test, test A for condition X, which has a sensitivity of 85% and specificity of 97%, is used in three different populations.

Population 1: screening of general population: prevalence of condition X is 3%—300 in 10 000.

10 000 tests:

	Has condition X (300)	Does not have condition X (9700)
Test positive	255	291
Test negative	45	9409

Positive predictive value = 47%
Negative predictive value = 99%

Despite this being a test with high specificity in this population less than half those with a positive test have condition X.

Population 2: testing in a population with symptoms suggestive of the condition drawn from the general population: Prevalence of condition X is 20% in this population—40 in 200.

	Has condition X (40)	Does not have condition X (160)
Test positive	34	5
Test negative	6	155

Positive predictive value = 87%.
Negative predictive value = 96%.

In this setting with a higher prevalence of the condition the test is much more useful—though there are still a significant number of false positives and false negatives—which might result in 1 in 6 operations being unnecessary.

Population 3: diagnostic testing in a patient thought to be quite likely to have the condition: prevalence of condition X is 50%—100 in 200.

200 tests:

	Has condition X (100)	Does not have condition X (100)
Test positive	85	3
Test negative	15	97

Positive predictive value = 97%
Negative predictive value = 86%

With a high clinical likelihood this test is very helpful.

Same test, different setting, very different results.

Test A is comparison of median and ulnar sensory latency from ring finger (AAN practice parameters).

Condition X is carpal tunnel syndrome. The prevalences given are those of general population,[13] population of symptomatic patients from general population,[13] and a hypothetical situation with a pre-test probability of 50%.

Table 1 Methods used in localising upper limb focal mononeuropathies

Nerve	Site of lesion	Demonstration of focal slowing	Finding changes in CMAP and SAP	Dennervation changes in nerve distribution	Comments
Common					
Median	Carpal tunnel	+++	+++	+	EMG not usually needed
Ulnar	Elbow (cubital tunnel)	+	+++	++	
Uncommon					
Radial	Upper arm	+	++	+++	
Axillary	Humeral head	NA	NA	++	
Ulnar	Wrist (Guyon's canal) or hand	+	++	+++	Depends on other ulnar studies being normal
Long thoracic	Not clear	NA	NA	++	Limited usefulness; small risk of pneumothorax
Rare					
Anterior interosseous branch of median nerve	Just below the elbow	NA	NA	++	Depends on other median studies being normal
Posterior interosseous branch of radial nerve	As nerve enters supinator muscle	NA	NA	++	Depends on other radial nerve studies being normal
Suprascapular	Suprascapular notch	NA	+	+++	
Musculocutaneous		NA	+	++	

+++ high likelihood; ++ probable finding; + possible finding; – unlikely finding.
Findings will be dependant on severity of lesion.
CMAP, compound motor action potential; NA, not applicable; SAP, sensory action potential.

haemoglobin of 10.9 g/dl falls outside the normal range but its clinical significance is less certain. This question of certainty has perhaps been best explored in relation to carpal tunnel syndrome.[7]

The significance of a test result will also vary depending on how likely it is that a patient has the condition being tested for. This can be easily illustrated by considering the impact of using a sensitive test, which is also reasonably specific, in populations with different prevalences of the condition being tested for (box 1). While this is easy to understand when applied to a population, the same applies when you consider the pre-test probability of having a diagnosis on clinical grounds—a pre-test probability of 20% equates to a population prevalence of 20%. A test is much more useful in patients who are likely to have the condition being tested for—if they are unlikely to have the condition the number of false positive results will lead to inappropriate diagnoses and investigations.

It is also important to consider whether the degree of abnormality found is consistent with the clinical picture. Using the anaemia analogy again, haemoglobin of 5 g/dl is a reasonable explanation for lethargy and fatigue, while haemoglobin of 10.9 g/dl is probably not.

PRAGMATIST OR COMPLETIST

A test should not be considered in isolation. We must also consider where it fits into the best diagnostic strategy—the strategy by which you get to the diagnosis and make the best management decisions as efficiently and effectively as possible. This involves thinking not only about the test in question but also other investigations and the potential therapeutic and prognostic implications of the results of these investigations.

Most research is focused on particular tests and there is less research into the effectiveness of diagnostic strategies, something most clinicians develop through clinical practice, though studies in this are beginning to appear.[8 9]

Neurologists fall into a spectrum that can be characterised as running from extreme "pragmatists", whose objective is to do as few investigations as possible, preferably none, to solve the immediate clinical management problem, to extreme "completists", who wish to eliminate every possibility, however remote, and document every deficit as completely as possible, even when this has no immediate therapeutic implications. Most neurologists lie somewhere in between, and reflect their role. Tertiary centre neurologists are probably more "completist", while those in secondary centres are more likely to be "pragmatists". Other factors such as the culture they work in, subspecialisation, medico-legal experiences, and their own personality will influence their approach.

The "pragmatist" will develop a strategy that uses tests that provide unique and definitive information. The "completists" will use investigations to build up and document the condition. Both will run into different problems:

- The pragmatist is at risk of misdiagnosing conditions, particularly those that are rare and mimic a more common alternative.
- The completist is at risk of misdiagnosing conditions because they may be misled by false positive results, particularly when using tests in situations where there is a low prevalence of the condition in question (true positives).

You will appreciate this spectrum of approach informs on the uptake of NCS/EMG that we now consider in certain clinical settings.

LEVEL OF THE PERIPHERAL NERVOUS SYSTEM

Focal mononeuropathies

Requests for studies in patients with suspected focal mononeuropathies are a common reason for referral for neurophysiology, and of these carpal tunnel syndrome and ulnar mononeuropathies are the most common.

Neurophysiological studies may be able to demonstrate an abnormality in the nerve, the site of involvement either:

- directly, if the site of involvement is anatomically located, to allow direct study of the nerve at that site, or
- by inference, if no direct study of the nerve at that site is possible, but changes consequent on the lesion, either changes in motor or sensory responses or dennervation changes, lead to the deduction of the lowest possible site of involvement.

They also determine whether there is evidence of more widespread subclinical peripheral nerve abnormalities.

They can be helpful for:

- Diagnosis—if you are unsure of the localisation on clinical grounds.
- Management—if you are considering surgical decompression then the most accurate localisation possible is desirable. Generally it would seem prudent to undertake a non-invasive test to document a focal neuropathy before an invasive procedure even if the diagnosis is secure on clinical grounds.
- Prognosis—if you wish to determine the degree of peripheral nerve damage. This will tend to be helpful in patients with a more severe peripheral nerve lesion—usually of more sudden onset, for example, trauma or compression—and distinguish between neuropraxia and axonotmesis as well as those situations where the nerve is no longer in continuity. Often this distinction cannot be made acutely, when the distinction would be most helpful. If there is real concern the nerve may not be in continuity, surgical exploration may be needed.

Tables 1 and 2 give a list of common mononeuropathies indicating how electrodiagnostic studies localise different focal neuropathies. Those where focal abnormality can be directly demonstrated provide the greatest diagnostic accuracy and diagnostic certainty. Those nerves where there are changes in the motor responses and on EMG provide a lower level of diagnostic confidence and localisation, and those with EMG changes alone lower still.

RADICULOPATHIES

In the majority of patients with symptoms or signs of a cervical or lumbosacral radiculopathy the diagnostic issue is whether there is radicular compression, usually related to disc disease, and the subsequent management revolves around the role of surgery. For such patients imaging, in particular MRI, is the most efficient investigation. In the past, when myelography was the imaging modality of choice, neurophysiological investigations were important in selecting patients for whom the small risks of contrast myelography were worth taking. However, the low risk of spinal MRI has changed this and the role for neurophysiology is reduced.

Diagnosis of a radiculopathy with electrodiagnostic studies depends on there being an axonal motor deficit sufficient to produce dennervation changes that EMG can detect in muscles within a specific myotome. Localisation with EMG can identify the myotome involved; however, the spinal level at which the nerve root involved exits the spinal canal is less certain as nerve roots commonly exit the spinal canal a level above (pre-fixed) or below (post-fixed) the expected spinal level.

In the small number of patients where imaging is equivocal, neurophysiology may be valuable in distinguishing radiculopathies from plexopathies. However, for both radiculopathies and plexopathies the localisation of the lesion does not provide an aetiological explanation for the deficit—this will only be provided by other factors, such as the time course, further imaging, or other investigative findings.

Plexopathies

Neurophysiological studies of plexopathies can demonstrate the distribution and severity of any dennervation, with involvement of muscles beyond the distribution of single nerve roots. Here the neurophysiologists must have a detailed knowledge of neuroanatomy to interpret these localising findings. There may be abnormalities of sensory responses as plexus lesions are post-ganglionic. EMG of paraspinal muscles should be normal as these are innervated by posterior nerve roots originating proximal to the plexus.

As you will appreciate these studies can support the plexus as the site of the lesion but again do not provide an aetiological diagnosis. However, there are traps, particularly if the lesion is mild. Absence of involvement of paraspinal muscles does not exclude a radiculopathy. Not all plexus lesions will lead to changes in those sensory responses that are accessible to measurement. If you remain in doubt then alternate diagnoses need to be excluded by other modalities, particularly multiple radiculopathies with MRI.

Table 2 Methods used in localising lower limb focal mononeuropathies

Nerve	Site of lesion	Demonstration of focal slowing	Finding changes in CMAP and SAP	Dennervation changes in nerve distribution	Comments
Common					
Lateral cutaneous nerve of the thigh	Inguinal ligament	NA	–	NA	Electrodiagnostic studies limited in this common mononeuropathy
Common peroneal	Fibular head	++	+++	++	
Interdigital	Between heads of metatarsals	NA	–	NA	Difficult nerves to study
Uncommon					
Femoral		NA	NA	+++	Distinguish from plexopathy/radiculopathy
Sciatic	Pelvis, buttock or thigh	NA	++	+++	
Tibial, at ankle	Tarsal tunnel	+	++	++	
Perineal	Alcock's canal	NA	NA	NA	
Saphenous	Thigh or knee	NA	+	NA	
Rare					
Tibial, at knee		NA	++	+++	
Posterior cutaneous nerve of the thigh		NA	NA	NA	Limited use
Obturator	Obturator foramen	NA	NA	++	
Gluteal		NA	NA	++	
Sural		NA	+++	NA	

+++ high likelihood; ++ probable finding; + possible finding; – unlikely finding.
Findings will be dependant on severity of lesion.
CMAP, compound motor action potential; NA, not applicable; SAP, sensory action potential.

Peripheral neuropathies

Nerve conduction studies can categorise neuropathies according to distribution (generalised or multifocal, or mono-neuritis multiplex), if they are sensory, sensorimotor or motor, and whether they are axonal or demyelinating. Studies may, however, be normal in small fibre neuropathies.

There are many aetiologies to neuropathies and these differ according to their varying neurophysiological findings. One approach described in an excellent review[10] is to use NCS/EMG as the primary method of classification of a patient suspected of having a neuropathy. From an epidemiological point of view this means that the majority of patients tested will have a distal symmetrical axonal polyneuropathy, most associated with diabetes or alcohol, where the diagnosis is straightforward.[11]

An alternative strategy is to focus on what additional information the NCS/EMG provide and recognise that for many patients with a typical chronic neuropathy other factors, such as diabetes, history of alcohol or drug exposure lead to the diagnosis and the NCS/EMG adds nothing to the clinical assessment. A study of this strategy found half of neurophysiological studies performed added nothing to the eventual diagnosis in the investigation of patients with a distal symmetrical polyneuropathy of more than six weeks duration.[8] This study also reassuringly found that those patients with demyelinating neuropathies were recognised on clinical grounds.

If the role of nerve conduction studies and EMG in chronic neuropathies is disputed there is no such controversy in their use in acute neuropathies, asymmetrical or multifocal neuropathies, or in any severe disabling neuropathy. Here the differential diagnosis includes acute and chronic demyelinating polyneuropathies, and vasculitic neuropathies where electrodiagnostic tests lead either to a diagnosis and treatment or strengthen the indication for nerve biopsy. Any patient with a neuropathy that is not entirely typical for the putative cause should also be considered for neurophysiological assessment.

For the most part changes in nerve conduction studies and EMG lead to a clear categorisation of neuropathy. However, there are potential traps:

- As the fibres tested are large myelinated fibres a small fibre neuropathy may not be associated with changes and other diagnostic tests such as thermal thresholds may help.
- In patients with demyelinating neuropathy (for example, early Guillain-Barré syndrome) investigated early in disease course, changes can be relatively subtle or absent.

Anterior horn cell disease

Neurophysiology serves two purposes in anterior horn cell disease. Firstly, it documents that the deficit relates to the anterior horn cell by demonstrating dennervation changes with normal motor conduction and normal sensory studies, and eliminates alternative purely lower motor disorders, in particular multifocal motor neuropathy with conduction block. Secondly it can demonstrate evidence of dennervation in clinically normal muscles and by demonstrating a wider distribution of lower motor neurone abnormalities so corroborate the diagnosis of amyotrophic lateral sclerosis (ALS).[12]

Thus in a patient with disease limited to one body region or with a purely lower motor presentation, neurophysiology has the potential for substantially altering patient management.

Nerve conduction studies (NCS) and electromyography (EMG): key points

- The tests are there to help you (and if they won't help don't do them)
- NCS and EMG are an extension of clinical examination.
- Generally NCS and EMG are effective at localising the level of the nervous system involved or the site of the lesion but not the cause
- Sometimes NCS can be misleading—for example, finding abnormalities that occur within the general population or misattributing myopathic and neuropathic weakness
- Tests other than NCS or EMG will usually be needed to determine the cause of a problem

In a patient with evidence of widespread upper and lower motor neurone signs the study will simply document the deficit and confirm the clinical diagnosis.

Neuromuscular junction disorders

Neurophysiology has been pivotal in defining disorders of the neuromuscular junction and is the test that characterises these conditions. The investigations are straightforward in situations where the clinical diagnosis is obvious but become increasingly difficult in patients with more subtle or limited problems.

The role of neurophysiology will vary. In a patient with clear clinical evidence of myasthenia and a positive anti-acetylcholine receptor antibody, repetitive nerve stimulation or single fibre may corroborate—though would not exclude the diagnosis if normal. However, in a patient with a suggestive clinical picture but a negative antibody test then neurophysiology may diagnostic. The pragmatist might argue the studies add nothing in the first situation and are thus unnecessary.

Neurophysiology provides the definitive diagnosis for patients with Lambert-Eaton myasthenic syndrome (LEMS).

Nerve conduction studies and EMG are helpful in distinguishing botulism from an acute demyelinating neuropathy.

Muscle disease

Electrodiagnostic studies can find evidence of a myopathy, eliminating alternative diagnoses, and document the distribution of myopathic changes. Some findings can point to specific diagnoses—for example, fibrillations suggesting an inflammatory myopathy, myotonia suggesting a myotonic disorder—though for all these conditions other tests provide the gold standard for diagnosis.

How should neurophysiology fit into the diagnostic strategy for a patient with a suspected myopathy? Pragmatists would argue that a patient with the clinical features of an inherited muscle disorder for which there is a simple genetic test, such as myotonic dystrophy or fascioscapulohumeral muscular dystrophy, should have appropriate genetic studies as neurophysiology would add nothing if the clinical diagnosis is confirmed—though may be useful if it is not. Likewise if a patient has features of an inflammatory myopathy with raised inflammatory markers and raised creatine kinase in whom a muscle biopsy will provide the definitive diagnosis, then this is the test that should be done.

Neurophysiology is likely to be particularly helpful if you are unsure whether the weakness is neurogenic (increased amplitude, increased duration units, with fewer units firing

at a high rate), or myopathic (short duration, small units with early recruitment to a full interference pattern of reduced amplitude)—and thus will direct your diagnostic strategy.

However, the distinction is not always clear cut and there are traps:

- Low amplitude polyphasic short duration motor units, normally indicative of a myopathic abnormality, can occur in early reinnervation following dennervation.
- In severe myopathies loss of whole motor units can lead to limited recruitment mimicking a neuropathic lesion.
- Longer duration motor units, normally a feature of a neuropathic weakness, can be seen in myopathies with regeneration.

Thus there is a risk that EMG can misattribute the level of weakness, especially if the results are taken in isolation and the possibility of this misattribution is not considered. If the EMG result is at odds with other elements of the clinical picture then think again.

Syndrome hunting

Sometimes NCS/EMG can be helpful in the differential diagnosis of patients who present with a more complicated widespread neurological problem. They can provide objective evidence of involvement of peripheral nerve or muscle in a patient with a predominantly upper motor neurone deficit or even dementia, which changes the differential diagnosis. For example, finding of widespread dennervation in a patient with upper motor neurone syndrome without sensory loss or in a patient with fronto-temporal dementia can lead to a diagnosis of motor neurone disease.

RECOMMENDED READING

- Kimura J. *Electrodiagnosis in diseases of nerve and muscle*, 3rd ed. Oxford University Press 2001. **A magisterial overview of peripheral neurophysiology.**
- Staal A, van Gijn J, Spaans F. *Mononeuropathies; examination, diagnosis and treatment*. London: WB Saunders, 1999. **An excellent and very accessible text on mononeuropathies.**

REFERENCES

1 **Haq UR**, Fries TJ, Pendelbury WW, *et al*. Chronic inflammatory demyelinating polyradiculoneuropathy; a study of proposed electrodiagnostic and histologic criteria. *Arch Neurol* 2000;**57**:1745–50.
2 **Jalecki CK**, Andary MT, Floeter MK, *et al*. Practice parameter: electrodiagnostic studies in carpal tunnel syndrome. *Neurology* 2002;**58**:1589–92.
3 **American Association of Electrodiagnostic Medicine,** American Academy of Neurology, American Academy of Physical Medicine and Rehabilitation. Practice parameter for electrodiagnostic studies in ulnar neuropathy at the elbow: summary statement. *Muscle Nerve* 1999;**22**:408–11.
4 **Levin KH**, Maggiano HJ, Wilbourn AJ. Cervical radiculopathies: comparison of surgical and EMG localisation of single root lesions. *Neurology* 1996;**46**:1022–5.
5 **Antunes AC**, Nobrega JAM, Manzano GM. Nerve conduction study of the median and lateral plantar nerves. *Muscle Nerve* 2000;**40**:135–8.
6 **Boon AJ**, Harper CM. Needle EMG of abductor hallucis and peroneus tertius in normal subjects. *Muscle Nerve* 2003;**27**:752–6.
7 **Nodera H**, Herrmann DN, Holloway RG, *et al*. A Bayesian argument against rigid cut-offs in electrodiagnosis of median neuropathy at the wrist. *Neurology* 2003;**60**:458–64.
8 **Rosenberg HR**, Portegies P, de Visser M, *et al*. Diagnostic investigation of patients with chronic polyneuropathy: evaluation of a clinical guideline. *J Neurol Neurosurg Psychiatry* 2001;**71**:205.
9 **Haig AJ**, Huey-Ming T, LeBreck DB. The value of electrodiagnostic consultation for patients with upper extremity nerve complaints: a prospective comparison with history and physical examination. *Arch Phys Med Rehabil* 1999;**80**:1273–81.
10 **England JD**, Asbury AK. Peripheral neuropathy. *Lancet* 2004;**363**:2151–61.
11 **MacDonand BK**, Cockerell OC, Sander JWAS, *et al*. The incidence and lifetime prevalence of neurological disorders in a prospective community based study in the UK. *Brain* 2000;**123**:665–76.
12 **Brooks BR**. El Escorial World Federation of Neurology criteria for the diagnosis of amyotrophic lateral sclerosis. Subcommittee on motor neuron diseases/amyotrophic lateral sclerosis of the World Federation of Neurology Research Group on Neuromuscular Diseases and the El Escorial ''Clinical limits of amyotrophic lateral sclerosis'' workshop contributors. *J Neurol Sci* 1994;**124**(suppl):96–107.
13 **Atroshi I**, Gummesson C, Johnsson R, *et al*. Prevalence of carpal tunnel syndrome in a general population. *JAMA* 1999;**282**:153–8.

''EEG HAPPY FAMILIES''

Answers

EEG number	Request form	Technician's report	Conclusion
1	B	iv	f
2	C	v	a
3	D	i	g
4	E	ii	h
5	F	iii	d
6	A	vii	c
7	G	viii	e