Sensory evoked potentials in Guillain-Barré polyneuropathy

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SUMMARY In 11 patients with acute Guillain-Barré polyneuropathy examined within 2 weeks of the onset of the paralysis, sensory evoked potential techniques were able to demonstrate a proximal conduction delay between Erb's point and the cervical cord in 10 subjects; while sensory conduction distal to Erb's point was much more commonly normal in the early period. Sensory evoked potential techniques provide therefore a valuable way to demonstrate proximal conduction velocity slowing early in the course of this disease.

The lesions in Guillain-Barré polyneuropathy are distributed in most patients throughout the peripheral nervous system.¹⁻³ In others, however, the changes are most prominent in the proximal peripheral nerves.⁴ In keeping with the last, conduction block and slow conduction in the proximal peripheral nerves are demonstrable sometimes when more distal conduction is normal or very nearly normal.⁵⁻⁸

Because the assessment of motor conduction over the proximal portions of the peripheral nervous system is difficult we wanted to learn whether somatosensory evoked potential (SEP) techniques would offer any advantage, especially in the early course of Guillain-Barré polyneuropathy when both motor and sensory conduction over more distal segments of the peripheral nervous system are often normal.

Our results have shown that proximal conduction abnormalities are commonly demonstrable with the SEP technique and suggest that these regions may be amongst the earliest affected in Guillain-Barré polyneuropathy.

Methods

Subjects

Eleven patients meeting the NINCDS criteria⁹ for the

diagnosis of Guillain-Barré polyneuropathy in whom the sensory and motor conduction studies were carried out within the first two weeks of the onset of the paralysis were included in this study.

Electrophysiological tests

SEP measurements: The median nerve was stimulated at the wrist and the evoked potentials recorded at the proximal upper arm, Erb's point, cervical (C6 spine) and contralateral cortical (C3-4) sites by surface electrodes except at the scalp where needle electrodes were used. FZ served as the reference electrode for all sites.

Stimulus intensities were adjusted to be just supra-maximal for the 2nd or 3rd digit antidromic sensory nerve action potentials. Maximum sensory conduction velocities (MSCV) were measured between the wrist and proximal arm, proximal arm and Erb's point and Erb's point to the cervical cord. For these, the latencies were measured to the initial positive peak in the case of the nerve trunk potentials, the onset of the cervical potential and to the N 20 peak. Calipers were used to measure the distance between Erb's point and the C6 dorsal spine.

Motor: For comparison with conduction in motor fibres the maximum motor conduction velocities (MMCVs) were also assessed in the median nerve by recording the thenar maximum "M" potential and stimulating the median nerve at the levels of the proximal upper arm, elbow and wrist. Stimulation at Erb's point was excluded because the stimulus at this site unavoidably may extend to both ulnar and median motor axons innervating muscles in the thenar group.

To assess motor conduction between Erb's point and the proximal upper arm, the recordings were made from the hypothenar muscle group because there is very little pickup of electrical potentials generated by the median innervated muscles at this site and so extension of the stimulus to median nerve innervating the thenar and first one or two

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lumbrical muscles does not cause any appreciable errors in the assessment of this proximal conduction velocity. Conduction velocities in ulnar motor axons distal to the upper arm however, were notably less useful compared to median conduction assessments between the upper arm and wrist because of the propensity to local conduction abnormalities in the ulnar nerve at the level of the elbow even early in the course of the Guillain-Barré polyneuropathy, possibly because of compression injury to the nerve at this site in the paralysed limb.

Results

The results in the 11 patients in this study are listed in the table.

In all but one of the 11 patients, sensory conduction was abnormal within the first 2 weeks after the onset of the weakness. For example, the MSCVs were below the 3 SD control lower limit between Erb's point and the cervical cord in 10 patients, and below this limit between Erb's point and the proximal upper arm in two patients. In contrast, the MSCVs between the proximal arm and wrist were within the normal range in all but one patient in the first 2 weeks.

The peak to peak (p-p) amplitudes of the compound nerve action potentials recorded at the level of the proximal arm were in the normal range in 10 of the 11 patients, but at Erb's point, (5 of 11) and at the cervical site (1 of 11) the amplitudes were well below those in all normal subjects. Moreover, in six patients, the p-p duration of the proximal arm potential exceeded the 3 SD upper limit of control (fig).

In six of the 10 patients in whom the MSCV between Erb's point and the cervical region was

abnormally slow (< 3 SD below control values), the MMCVs in ulnar motor axons between Erb's point and the proximal arm was also shown to be slow. In the remaining four of the above 10 patients the motor conduction velocity was either normal (in two), not assessed or could not be assessed properly because of the very low amplitude of the "M" potentials. The latencies of the cortical potentials were prolonged (> 2 SD upper limit) in only three of the 11 patients.

Discussion

The most important observation in this SEP investigation was the clear demonstration of slowed conduction across the roots and proximal brachial plexus at times when motor and sensory conduction velocities over more distal segments were normal. This pattern was established within the first two weeks following the onset of the weakness in this disease. In some of the patients there was also slow conduction in motor axons between the proximal arm and Erb's point, but in only one patient were the maximum conduction velocities, sensory or motor, abnormal distal to the proximal arm in this early period.

Other abnormalities included the increases in the p-p durations of the recorded nerve trunk potentials at the proximal arm site and reduced (p-p) amplitudes of the potentials at the Erb's and cervical sites in some patients. The increase in p-p duration points to an increase in temporal dispersion among single fibre potentials which contribute to the main nerve trunk potential. However, it is impossible, with SEP techniques, to assess conduction block

Table Sensory evoked potentials. Maximum conduction velocities and peak to peak amplitudes of the compound nerve action potentials

	W-PA (m/s)	PA (mV)	PA-Erb's (m/s)	Erb's (mV)	Erb's-cervical (m/s)	Cervical (mV)
Control (n = 30)	64.7	0.013	74.8	0.006	79.6	0.002
± 1 SD	4.0	0.006	5.0	0.001	6.9	0.0005
least	59.0	0.0065	70	0.0025		0.001
normal	52.7		59.8		58.9	
- 3 SD	62.5	0.008	54.5	0.002	28.6	0.001
Patients	62.1	0.006	67.6	0.002	40.3	0.001
	64.9	0.014	73.4	0.004	47.8	0.002
	60.2	0.008	48.9	0.003	32.4	0.001
	62.1	0.009	65.0	0.002	42.9	0.001
	64.0	0.009	73.1	0.003	64.7	0.002
	61.6	0.007	72.2	0.002	26.0	0.003
	62.1	0.009	65.0	0.003	42.9	0.001
	65.1	0.007	55.9	0.001	33.3	0.001
	72.6	0.009	65.0	0.002	30.6	0.001
	56.1	0.002	57.1	0.001	36.6	0.001

W-PA = wrist to proximal arm.

PA-Erb's = proximal arm to Erb's point.

Erb's-cervical = Erb's point to cervical cord.

Fig Illustration of the compound nerve trunk potentials recorded at the upper arm and Erb's point in response to a stimulus (S) delivered at the wrist just supramaximal for the antidromic sensory nerve action potential (digit II or III). Shown too are the cervical (C6 spine) and contralateral cortical evoked potentials. The reference was FZ. Gains same for both control (left) and Guillain-Barré polyneuropathy subjects (right). Note the broader upper arm potential, lower amplitude of the cervical potential and the later onset of the latter in respect to the Erb's point potential in the Guillain-Barré polyneuropathy patient.

because the potentials generated by single nerve fibres as recorded in the volume conductor presented by the extracellular space about the nerve trunk, are triphasic and the durations of such potentials are short. This, and the wide differences among their conduction velocities (25–70 m/s) could reduce the area of the recorded compound nerve action potentials through phase cancellations between the various single fibre potentials. This makes it impossible therefore, to assess conduction block properly in such nerves.

The main technical problems in these investigations were uncertainties about the precise site of origin of the brachial plexus potential in relation to the Erb's point electrode, lack of a sharp onset of the cervical potential in some patients and the inability to measure directly the distances between the sites of origin of the brachial plexus and cervical potentials. The onset of the cervical potential probably does, however, correspond to the arrival of orthodromic impulses at the root entry zone and the earliest onset of pre- and post-synaptic activity in

the spinal cord.

The wisdom of calculating a conduction velocity between Erb's point and the cervical potential is open to question because the distance between these two sites cannot be measured properly. Nonetheless, these velocities were calculated for continuity in comparison to the velocities across more distal segments. As well, those judged to be normal or abnormal were precisely the same whether the assessments were based on the latencies between Erb's point and the cervical potential alone or the derived conduction velocities over the same segment.

Even though stimulation of the median nerve at the wrist results in antidromically conducted potentials in some motor axons as well as the orthodromically conducted potentials in sensory afferents, the maximum conduction velocities as assessed by the SEP technique must have represented the latter rather than the former because the maximum motor conduction velocities as measured in the median nerve over the same segments were in almost all

instances 5–10 m/s lower than those assessed by study of the SEP potentials.

In Guillain-Barré polyneuropathy the conduction velocity between Erb's point and the cervical cord could be reduced where conduction in the normally fastest conducting nerve fibres was slowed, or the impulses in enough of them were blocked to make it impossible to detect the earliest onset of the cervical potential. Both the above could coexist in the same patient.

The finding of proximal conduction abnormalities in excess of those in more distal segments of the peripheral nervous system, suggests that the former are more liable to damage in Guillain-Barré polyneuropathy. In this regard, it was of interest that in all but one of the 11 patients, the distal sensory nerve action potentials were all in the normal range within the first two weeks. This and the normal MSCVs between the wrist and proximal arm studied by the SEP technique suggests that the earliest conduction abnormalities in the larger myelinated sensory fibres are truly proximal in location.

Whether or not there were conduction abnormalities in the very terminal portions of peripheral sensory fibres similar to those postulated to occur in motor axon terminals and pre-terminals because of deficiencies in the peripheral barrier at these sites,¹⁰ could not be excluded because the sensory nerve fibres were not stimulated through their terminals or receptors.

The fact that the cortical potentials were only delayed in three of the patients, whereas proximal conduction delays were demonstrable in 10 patients could be explained if it were assumed that fewer fastest conducting afferents were necessary to evoke the cortical potential than the cervical potential.

Even though "F" response latencies have been shown to be prolonged in many patients with Guillain-Barré polyneuropathy,^{5,6} unfortunately in our experience the "F" response is often absent very early in the course of the disease, at the stage of maximum paralysis, perhaps because of conduction block over the more proximal segments of the peripheral nervous system in this disease.

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