Somatosensory evoked potentials in syringomyelia

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SUMMARY The two types of upper limb somatosensory evoked potential abnormality observed in nine patients with syringomyelia were reduced amplitude or absent cervical potentials and an abnormal central conduction time. Although this pattern of abnormalities resembles that observed in other intrinsic spinal cord lesions, it differs from peripheral nerve diseases and cervical radiculopathy in which the central conduction time is normal.

Somatosensory evoked potentials (SEPs) have been used extensively in the investigation of diseases of the peripheral and central nervous systems.¹ However, reports of SEP findings in patients with syringomyelia have been rare and, with one exception,² have described only one or two individual cases.³⁻⁸ Accordingly the findings that may be expected in patients with syringomyelia have not been clearly defined. Since the clinical presentation of syringomyelia may resemble that of other spinal cord lesions and peripheral nerve diseases, it is important to be aware of the types of SEP abnormality that can be observed in this group of patients.

We report the results of SEP studies in nine patients with syringomyelia.

Methods

SEPs were recorded while the subjects sat on a semi-reclining couch in a quiet, electrically shielded room. Stimuli consisting of 0.15 ms square wave pulses were delivered at a rate of 4.1 Hz via silver cup electrodes, saline soaked pads or subdermal needles placed over the median and ulnar nerves at the wrists. The cathode was 2.5 cm proximal to the anode. The stimulus intensity was adjusted to produce a twitch in the appropriate distal muscles. Both upper extremities were studied individually in all patients. Recordings were made with platinum alloy subdermal needle electrodes placed over the midpoint of the clavicle (Erb's point), over the spinous processes of the seventh (CVII) and second (CII) cervical vertebrae and over the scalp, contralateral to the side of stimulation. The scalp electrodes were placed 2 cm behind the C3 and C4 positions in accordance with the international 10-20 system. A mid-forehead reference electrode (Fpz) was

used for all recordings and the patient was grounded through an additional scalp electrode. Electrode impedance was maintained below 5,000 ohm.

Four channels of data were recorded simultaneously through high input impedance biological amplifiers using a bandpass of 10 Hz to 3,000 Hz at -3 db. EMG, EEG and ECG signals were automatically rejected if their amplitudes exceeded 50 μ V. A Tracor Northern TN 3,000 computer collected and averaged 40 ms samples following each stimulus. Two sets of 1,000 samples were made for each electrode derivation and if the averaged potentials were not reproducible, a third set of 1,000 samples was recorded. A potential was regarded as present if it was reproducible on two or more runs. The sets of 1,000 samples were added together and averaged before measurement of the amplitude and latency of each individual potential.

The amplitudes of the Erb's point and cervical potentials were measured from the preceding baseline to the peak of the negative potential while scalp potential amplitudes were measured between the peaks of the negative potential and the following positivity. The amplitudes of the potentials were considered abnormal if they were outside the range obtained in normal subjects. Latencies were measured to the peaks of the negative potentials. The central conduction time⁹ (defined as the interpeak latency between the major potentials at the cervical spine and scalp) was calculated for each nerve tested. Absolute latencies and central conduction times were considered abnormal if they were more than three standard deviations longer than the mean value for normal subjects.

Subjects

The normative data for median and ulnar nerve SEPs have been reported elsewhere;¹⁰ relevant values are seen in the tables of results. The median nerve central conduction times were compared with previously published normal data from this laboratory.⁹

We studied nine patients with syringomyelia (table 1). There were eight females and one male, aged 16 to 65 (mean 41) years. All patients had neurological findings typical of syringomyelia with lower motor neuron signs in the upper limbs, impaired pain and temperature sensation in cervical and/or thoracic dermatomes and, in six cases, upper

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Received 5 July 1985 and in revised form 4 February 1986.
Accepted 24 March 1986.

Table 1 Clinical and radiological findings in nine patients with syringomyelia

Patient	Sex	Age (yr)	Clinical features			Radiological findings	
			Cranial nerve signs	Lower motor neuron signs in arms	Abnormal posterior column sensation	Spinal cord size	Tonsillar herniation
1	F	37	0	Left only—Mild	0	Normal	+
2	F	43	0	Right > Left—Moderate	Right hand	Normal	0
3	F	59	0	Bilateral—Severe	All limbs	Atrophic	+
4	F	29	+	Bilateral—Moderate	0	Normal	+
5	F	34	0	Absent right finger jerk only	0	Dilated	+
6	F	36	+	Right > Left—Severe	Right hand	Dilated	+
7	M	50	+	Bilateral—Moderate	0	Atrophic	+
8	F	65	+	Bilateral—Severe	All limbs	Dilated	+
9	F	16	0	Right only—Mild	0	Dilated	+

M = Male, F = Female, 0 = Absent, + = Present.

motor neuron signs in the legs. The lower motor neuron signs were unilateral in three patients and, although bilateral in six cases, in two of these the changes were significantly asymmetric.

All patients were investigated with myelography, using water soluble contrast material in seven cases and iophendylate in the other two. This was combined with computerised tomography (CT) of the foramen magnum and spinal cord in seven. The radiological findings were characteristic of syringomyelia in all cases^{11 12} (table 1); they included the demonstration of a spinal cord cavity in all patients who had CT scans.

In two patients (Patients 6 and 8) posterior fossa surgery had been performed 15 and 11 years respectively before the study. In both cases the foramen magnum was decompressed and a muscle plug was placed in the proximal part of the central canal of the medulla. Another two patients (Patients 1 and 4) had a posterior fossa operation and decompression of the foramen magnum after SEP investigations. The intraoperative findings in each of these

four cases confirmed the presence of a Type 1 Chiari malformation.

Brainstem auditory evoked potentials recorded in four patients (Patients 1, 4, 5 and 7), two of whom had cranial nerve signs, were normal, as were pattern-reversal visual evoked potentials in Patient 4.

Results

Median nerve (table 2) and ulnar nerve (table 3) SEPs were recorded in all patients. Six had abnormally low amplitude or absent cervical potentials but in all cases the latencies of the cervical potentials were normal. The abnormalities of the cervical potentials were significantly asymmetric in five of the six cases, with the more abnormal findings corresponding to the side of greater clinical involvement. In Patient 7 the Erb's point potential was absent after stimulation of the

Table 2 Latencies and amplitudes of median nerve somatosensory evoked potentials in nine patients with syringomyelia

Patient		Erb's point		CVII		CII		C3/C4		CCT	Comment
		Lat	Amp	Lat	Amp	Lat	Amp	Lat	Amp		
1	R	9.7	3.8	13.3	1.8	13.4	1.6	18.6	4.2	5.2	Normal
	L	9.6	3.2	13.1	1.2	13.2	0.8	18.4	4.7	5.2	
2	R	10.3	4.1	14.4	0.3*	14.5	0.5*	20.9	1.2	6.4	Reduced amplitude cervical potentials
	L	10.3	1.4	14.2	0.7*	14.1	1.2	20.2	2.3	6.1	
3	R	9.1	4.5	13.4	0.6*	13.4	0.6*	20.3	1.2	6.9	Reduced amplitude cervical potentials; prolonged CCT
	L	9.0	2.6	12.7	1.1	12.7	1.2	20.1	2.0	7.4*	Asymmetric CCT
4	R	NR	NR	NR	NR	13.5	0.8	18.0	1.7	4.5	
	L	NR	NR	NR	NR	13.0	1.9	19.3	1.8	6.3*	
5	R	10.0	4.8	14.2	3.8	14.1	3.7	19.7	5.0	5.6*	Asymmetric CCT
	L	9.5	4.0	14.1	3.5	14.1	3.3	18.8	4.9	4.7	
6	R	7.3	11.1	11.3	0.2*	11.4	0.2*	17.2	2.1	5.8	Reduced amplitude cervical potentials; prolonged CCT
	L	7.8	7.5	11.5	1.0	11.5	1.1	19.5	1.6	8.0*	Borderline asymmetric CCT
7	R	11.0	3.3	13.5	3.2	13.6	2.8	19.5	1.7	5.9*	
	L	10.4	2.8	14.1	2.7	14.1	2.5	19.2	1.8	5.1	
8	R	9.6	5.6	13.8	2.0	13.6	1.0	19.7	1.2	6.1	Left—absent cervical potentials
	L	9.8	2.9	Abs*	—	Abs*	—	20.1	0.8	—	
9	R	9.1	4.1	NR	NR	12.0	2.5	18.1	6.8	6.1*	Asymmetric CCT
	L	9.2	6.4	NR	NR	12.7	3.5	17.9	8.2	5.2	
Normal		10.0†	1.2§	13.2†	0.8§	13.3†	0.7§	19.1†	0.7§	5.75†	Mean right—left CCT difference = 0.25 ± 0.18 ms
		± 0.6‡	—10.6	± 0.8‡	—3.2	± 0.8‡	—3.8	± 0.9‡	—8.3	± 0.46‡	

CCT = Central conduction time; * = Abnormal result; R = Right; NR = No recording from this site; Lat = Latency (msec); L = Left; Abs = Absent potential; Amp = Amplitude (microvolts); † = mean; ‡ = SD; § = range.

Table 3 Latencies and amplitudes of ulnar nerve somatosensory evoked potentials in nine patients with syringomyelia

Patient		Erb's point		CVII		CII		C3/C4		CCT	Comment
		Lat	Amp	Lat	Amp	Lat	Amp	Lat	Amp		
1	R	9.6	1.6	13.8	1.1	13.9	0.6	19.3	1.7	5.4	Normal
	L	10.4	3.1	14.0	1.1	14.1	0.9	19.2	3.1	5.1	
2	R	11.4	1.4	16.6	0.5	16.7	0.6	22.5	0.8	5.8	Absent left Erb's point potential
	L	Abs*	—	16.6	0.6	16.6	1.0	22.0	1.5	5.4	
3	R	Abs*	—	Abs*	—	Abs*	—	Abs*	—	—	Bilateral absent cervical potentials; absent right Erb's point and left scalp potentials
	L	9.3	2.1	Abs*	—	Abs*	—	20.9	0.9	—	
4	R	NR	NR	NR	NR	13.0	0.4	18.4	1.1	5.4	Reduced amplitude cervical potential
	L	NR	NR	NR	NR	13.7	0.3*	20.0	1.3	6.3	
5	R	10.7	1.5	15.4	3.0	15.3	2.5	20.2	2.4	4.9	Normal
	L	10.2	2.8	14.4	3.1	14.5	2.8	19.5	2.4	5.0	
6	R	7.8	3.1	12.2	0.6	12.4	0.7	17.4	3.2	5.0	Normal
	L	9.0	1.1	12.8	1.3	12.7	1.2	17.6	2.2	4.9	
7	R	Abs*	—	14.8	1.6	Abs*	—	20.6	0.8	—	Absent right Erb's point and CII potentials
	L	11.8	1.3	16.1	0.7	16.0	1.0	19.9	1.5	3.9	
8	R	10.0	1.1	Abs*	—	Abs*	—	Abs*	—	—	Cervical and scalp potentials absent bilaterally
	L	10.2	1.8	Abs*	—	Abs*	—	Abs*	—	—	
9	R	9.7	0.8	NR	NR	13.6	0.5	19.4	3.6	5.8	Normal
	L	9.5	1.0	NR	NR	13.0	0.6	18.0	4.9	5.0	
Normal		10.5†	0.6§	14.1†	0.5§	14.2†	0.4§	19.6†	0.6§		
		±0.8‡	—4.4	±1.1‡	—2.7	±1.0‡	—2.7	±1.1‡	—5.3		

CCT = Central conduction time; * = Abnormal result; R = Right; NR = No recording from this site; Lat = Latency (msec); L = Left; Abs = Absent potential; Amp = Amplitude (microvolts); † = mean; ‡ = SD; § = range.

right ulnar nerve but he had neurophysiological evidence for a coexistent ulnar nerve lesion at the elbow. Erb's point potentials were absent after stimulation of one ulnar nerve in Patients 2 and 3 but there was no other evidence for an associated peripheral nerve lesion in these two cases.

Six patients had abnormal median nerve central conduction times (CCT). In two cases (Patients 3 and 6) the CCT to one hemisphere was prolonged, while in four (Patients 4, 5, 7 and 9) there was significant asymmetry in the CCT to the two hemispheres. In all of these cases a Type 1 Chiari malformation was present.

Upper limb SEPs were entirely normal in only one patient (Patient 1). This patient had mild lower motor neuron signs confined to the left arm associated with cervical syringomyelia and a Type 1 Chiari malformation.

Peroneal nerve SEPs were performed in one patient (Patient 4). The lumbar potentials were normal but the scalp potential following right peroneal nerve stimulation was of low amplitude.

Discussion

Previous reports of SEPs in patients with syringomyelia have described attenuation or absence of cervical potentials on the clinically affected side.³⁻⁷ However, other patients with syringomyelia have had normal upper limb SEPs.^{4,5} Stockard and Iragui described a patient with prolonged cervical potential latencies.⁸ Riffel and associates recorded tibial nerve SEPs in nine patients with syringomyelia;² the cortical potentials had normal or only slightly prolonged

latencies but amplitudes were frequently reduced.

Two types of abnormal findings were seen in upper limb SEPs in our patients with syringomyelia. Six patients had reduced amplitude or absent cervical potentials. These findings are similar to those described in earlier reports.³⁻⁷ We also observed an abnormal CCT in six patients, all of whom had tonsillar herniation at the foramen magnum. The presence of an abnormal CCT in syringomyelia may be due to compression of the medulla and upper cervical spinal cord at the foramen magnum, either in the past or at the time of investigation. Therefore a prolonged or asymmetric CCT in a patient with syringomyelia may provide a clue to the presence of a Type 1 Chiari malformation, but as this SEP abnormality was not seen in one patient with tonsillar herniation, it cannot be used to exclude this disorder. Spinal interneurons contribute to the cervical potential¹³ and it is therefore possible that the spinal cord syrinx alone may result in an increase in the CCT. However, it is more likely that the syrinx would cause attenuation or loss of the cervical potentials before it affects the CCT.

Only one patient in this group had lower limb SEPs and it is difficult to be certain of the usefulness of this investigation in syringomyelia. It is possible that lower limb SEPs may provide information on the anatomical extent of the spinal cord cavity and may help to differentiate syringomyelia from diseases of the peripheral nervous system but the fact that lumbar and cervical potentials may be of low amplitude or difficult to obtain in normal people¹⁴ limits the usefulness of this technique.

The SEP studies described in this report used electrical stimulation which predominantly activates

large, fast-conducting fibres that mediate light touch and proprioceptive sensation. Since pain and temperature sensation are predominantly affected in syringomyelia, recording of SEPs using stimulus modalities that preferentially activate smaller diameter unmyelinated or poorly myelinated fibres might be expected to demonstrate more prominent abnormalities. However such techniques may be uncomfortable and are not practical in routine evaluation of these patients.

The SEP abnormalities recorded in syringomyelia may resemble those observed in other neurological diseases, especially other intrinsic lesions of the spinal cord. The combination of low amplitude or absent cervical potentials and an abnormal CCT closely resembles the findings seen in patients with multiple sclerosis⁸ and similar changes may also be seen in patients with myelopathy associated with cervical spondylosis.¹⁵ Prolongation of the latency between Erb's point and the scalp has been described in patients with foramen magnum compression associated with achondroplasia¹⁶ and theoretically other abnormalities at the cervico-medullary junction may be expected to prolong the CCT. The importance of the pattern we have described is that it may assist in differentiating syringomyelia from various types of peripheral nerve disease and cervical radiculopathy in which the CCT is not prolonged.^{15 17 18}

The assistance of Dr E B Walker and Miss S Russell is gratefully acknowledged.

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