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Short communication

Short-latency somatosensory evoked potentials correlate with the severity of the neurological deficit and sensory abnormalities following cerebral ischemia

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Summary Short-latency somatosensory evoked potentials (SSEPs) were studied in 49 patients who had suffered hemispheric or thalamic ischemia, including 6 patients with transient ischemic attacks (TIAs) and 3 patients with reversible ischemic neurological deficits (RINDs). SSEPs were abnormal in 30 patients (61%). SSEP abnormalities correlated with the presence of sensory deficit and the degree of neurological deficit. SSEPs were normal following TIA but were abnormal in 2 of 3 patients with RINDs. SSEPs were abnormal in some patients without sensory deficits suggesting that sensory pathways may be affected when clinically inapparent.

Key words: Somatosensory evoked potentials; (Short latency); Neurological deficit; Ischemia; Sensory abnormality

Somatosensory evoked potentials have been applied in the study of stroke (Alajouanine et al. 1958; Larson et al. 1966; Liberson 1966; Miyoshi et al. 1971; Tadaharu et al. 1973; Chokroverty and Rubino 1975; Regli and Despland 1982; Robinson et al. 1985) but it is not clear how useful they are in evaluating the stroke patient. To determine the relationship between clinical findings and short-latency somatosensory evoked potentials (SSEPs) in stroke, we studied SSEPs in 49 patients who had suffered cerebral ischemia.

Methods

Description of patients and results of computerized axial tomography of the brain

All patients had a recent ischemic event with clinical signs and symptoms suggesting hemispheric involvement. These were confirmed by CT brain scanning in 34 cases. CT was normal in 14 and there were no CT data in one. CT scans demonstrated cortical (6), subcortical white matter (11), thalamic (3), and cortical-subcortical (14) lesions. Four patients had evidence of more than one event from CT evaluation. Six patients had TIAs and three had reversible ischemic neurological deficits (RINDs). The clinical profile demonstrated: mean age 54.6 (range 21–75), days elapsed from ischemic event to study 7.9 (range 1–22). Ten patients had previous TIA and eight had previous stroke. Neurological examination was done prior to SSEP testing and the presence or absence of a disturbance of pain, temperature, position, vibratory, or touch sensation was determined and the degree of weakness (mild–moderate, 4/5–3/5, or severe, 2/5–0/5) was also determined. Sensory loss was also graded as mild (slight to mild sensory deficit) or severe (marked sensory impairment). Patients were characterized as having severe or mild deficits or as being neurologically normal. Mild sensory loss or mild weakness were considered mild deficits while marked sensory impairment or severe weakness were considered severe neurological deficits. Aphasia or obtundation was considered a severe neurological deficit. Statistical analysis was performed using chi-square testing. SSEPs were interpreted without knowledge of the neurological examination.

Technique for performing SSEPs

SSEPs were done with a Nicolet II Pathfinder and a cephalic (midline frontal-Fz) reference was used. Using gold-plated cup electrodes, recordings were made from Erb's point (EP), the skin over the midline of the second cervical vertebra (CII) and modified C3 and C4 electrode positions (2 cm posterior) of the international 10-20 system. The median nerves were stimulated independently with a 0.1 msec constant current stimulus at a rate of 5.1/sec, and 1500 responses were averaged for 50 msec using 512 points. Amplification was 200,000 and bandpass was 30–1500 Hz. The following peak

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and interpeak latencies, when present, were measured: N9 (from EP), N11, N13/P13, N19, P22; N9-N13, EP-N19, EP-P22, N13-N19 (central conduction time), N13-P22. The N19 and P22 amplitudes were measured and the ratio of the N19-P22 amplitudes between the hemispheres was calculated (larger divided by smaller).

Controls

Thirty-six normal individuals (18 men, 22 women) mean age 31 years (range 19–49) served as controls for patients of age 50 or younger. Mean peak latencies and standard deviations for the EP, N13, N19 and P22 were: 9.93 ± 0.74, 13.53 ± 0.91, 19.1 ± 0.91 and 21.98 ± 0.90 msec. For the EP-N13, EP-N19, EP-P22, N13-N19 and N13-P22 interpeak latencies (IPLs) they were: 3.59 ± 0.40, 9.16 ± 0.44, 2.04 ± 0.57, 5.56 ± 0.39, and 8.44 ± 0.50 msec. Upper limits for these peak latencies and IPLs were determined by using the mean plus 3 S.D. Since the N19-P22 amplitudes and amplitude ratios did not show a gaussian distribution, a 95% tolerance limit was defined. An N19-P22 amplitude ratio (larger/smaller) was abnormal if greater than 2.55. N19 amplitudes and N19 amplitude ratios also showed a non-gaussian distribution in our controls and were highly variable. Therefore, an N19 amplitude could only be considered abnormal when absent or when the ratio of the N19 amplitude recorded from one hemisphere was 5 times that which was recorded from the other hemisphere. Likewise, P22 amplitudes were considered abnormal when absent. To define SSEP abnormalities in patients older than 50 years, a group of 12 normal individuals over 50 years (mean age 66 years) served as controls. There were slight differences in the peak and IPLs with age. For example, the upper limit for the CCT was 7.2 msec for the older control group compared with an upper limit of 6.9 in the younger group.

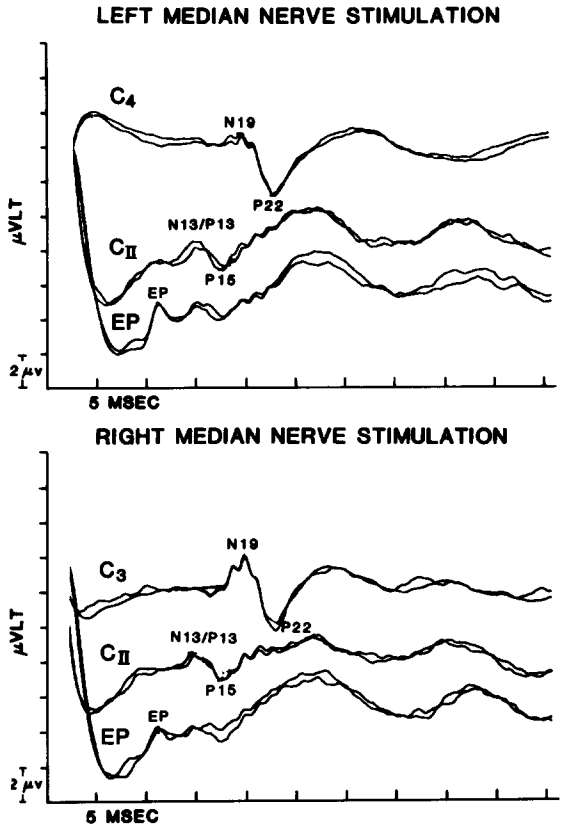

Fig. 1. This 39-year-old male suffered a stroke with expressive aphasia and right hemiparesis. CT scan was normal as were SSEPs.

TABLE I

Type of SSEP abnormalities in 30 patients. Five patients had more than 1 SSEP abnormality.

Amplitude abnormalities				Interpeak latency abnormalities				
N19	P22	N19-P22 (amplitude ratios)	Absent N19-P22 components	EP-N19	EP-P22	N13-N19 (CCT)	N13-P22	
Absent amplitude ratio (5)	Absent	2.6	3 patients	15.2	15.9	11.7	12.1 msec	
	Absent	5.18			16.9	8.3		
		2.7			17.8	12.9		
		4.0				7.4		
		3.8				9.0		
		3				9.3		
		3.2				7.7		
		3.5				9.3		
		2.9				7.6		
		6				8.4		
		2.55						
		2.6						
		4.98						
2	2	13	3	1	3	10	1	Totals

Results

Thirty of 49 patients had SSEP abnormalities. The types of abnormality are summarized in Table I. Normal SSEPs from a stroke patient are shown in Fig. 1. The most common abnormalities were those of N19-P22 amplitude ratios, 13 patients (Fig. 2), or central conduction times (CCTs), 10 patients. Five patients had more than 1 SSEP abnormality. Sixteen of 30 patients with normal sensory examination had normal SSEPs and 16 of 19 with abnormal SSEPs had abnormal sensory examination. The association between the sensory examination and SSEP abnormalities was significant, $P < 0.015$. Nineteen of 34 patients with mild neurological deficits had normal SSEPs and all 15 patients with severe neurological deficits had abnormal SSEPs.

Of the 15 patients with severe neurological deficits and abnormal SSEPs, 10 had severe motor deficits, 2 were aphasic, 2 had severe sensory loss, and 1 was obtunded. Four of the patients with severe motor deficits also had sensory loss.

The association between severity of the neurological deficit and SSEP abnormalities was also significant, $P < 0.0002$. Seven of 8 patients with prolonged CCTs had severe neurological deficits, as did 4 patients with absence of both N19 and P22 components. Four of 6 patients with lacunar infarcts had SSEP abnormalities, consisting of CCT abnormalities (2) and N19-P22 amplitude abnormalities (2). All 6 patients with TIAs had normal SSEPs. Two of 3 patients with RINDs had abnormal SSEPs (N19-P22 amplitude aymmetries). Two of 3 patients with thalamic infarcts had abnormal N19 amplitude ratios and one had an absent N19-P22 component.

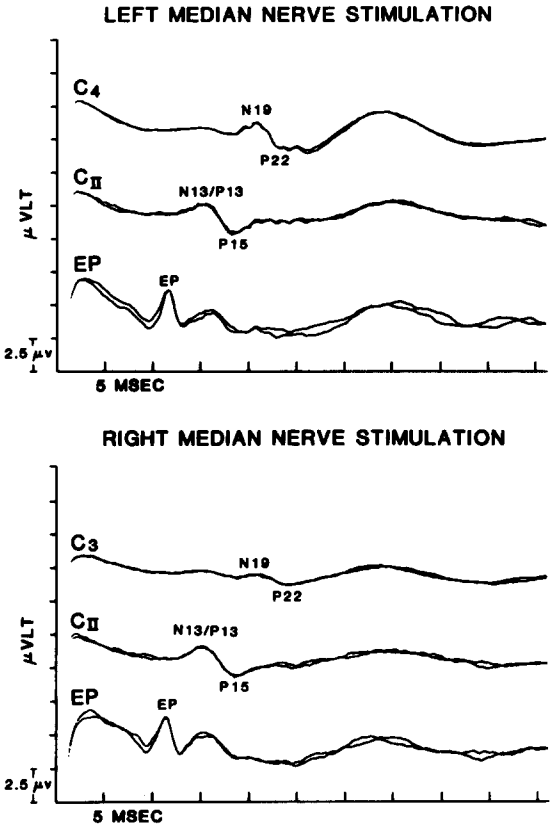

Fig. 2. This 61-year-old male developed the sudden onset of right-sided weakness and decreased sensation to pin-prick. The N19-P22 amplitude ratio is abnormal and is significantly lower with right median nerve stimulation.

Discussion

Our study suggests that SSEPs correlate with the severity of the neurological deficit and the sensory examination following stroke. SSEPs offer a non-invasive method of assessing the integrity of the posterior column mediated somatosensory pathway. CT brain scanning was more sensitive in demonstrating abnormalities compared to evoked potential testing. However, since many of our patients had earlier generation EMI scans, we cannot offer an ideal comparison of the two modalities.

The association between SEP abnormalities and sensory findings is similar to earlier reports (Alajouanine et al. 1958; Larson et al. 1966; Liberson 1966; Miyoshi et al. 1971), and our study has validated this association statistically. Our results also indicate that sensory pathway disturbances may be present when absent on clinical examination. Chokroverty and Rubino (1975) have also observed this in patients with 'pure' motor hemiplegia. The SSEP in stroke may also offer prognostic information, as N19 amplitude changes (Regli and Despland 1982) appear to be predictive of persistent sensory deficits. In this regard, it has been suggested that the SSEP might be a prognostic index for speech therapy (Liberson 1966).

Localization

Rostral to caudal localization of infarcts was difficult as we could not differentiate, using SSEPs, pure subcortical strokes from cortical strokes with subcortical involvement. We did find, however, that prolonged central conduction times were only present when there was evidence on CT scanning of subcortical involvement. Neurophysiologically this is consistent with the concept that prominent delays of the CCT will not occur in neuronal disturbances since synaptic delays would only account for a 1–2 msec delay (Desmedt and Cheron 1980). White matter conduction disturbances, conversely, can cause significant axonal conduction delays and will produce prominent CCT delays. Acutely, if blockage of axonal conduction occurs, only amplitude changes will be observed and this can also be produced by a cortical disturbance. CCT delays and absent N19-P22 components correlated strongly with severity of the neurological deficit. The CCT, therefore, may prove to have prognostic value in stroke. This may not be surprising if one considers that sensorimotor integration is highly dependent upon somatosensory input, especially positional, and that subtle changes in this system may be seen by

SSEP when absent clinically. While it remains to be seen if SSEPs are reliably predictive of long-term outcome from stroke, they do provide a sensitive and quantitative means of assessing the somatosensory pathway.

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