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Posterior tibial nerve somatosensory evoked potentials in slowly progressive spastic paraplegia: a comparative study with clinical signs

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Abstract. Clinical and neurophysiological examinations were performed on seven patients with hereditary spastic paraplegia and on eight patients with primary lateral sclerosis. The results were compared with those obtained from a group of 39 control subjects. Prolonged latency times and decreased amplitudes of the posterior tibial nerve (PTN) somatosensory evoked potentials (SEPs) were found in the majority of the patients. The SEP changes occurred without sensory impairment or with loss of vibration sense only. There was no significant relation between the PTN SEP abnormalities and the severity of pyramidal signs for the whole patient group, nor longitudinally for the individual subjects. Analyses of PTN SEPs in patients suffering from slowly progressive spastic paraplegia (SP), therefore, seem to be a method to indicate a feature of spinal cord dysfunction that is not related to the severity of clinical signs. Considering the neuropathology of the spinal cord in SP patients, we furthermore argue that the ascending spinal pathway involved in conducting impulses for PTN SEPs probably uses other routes as well as the funiculus gracilis.

Key words: Somatosensory evoked potentials – Posterior tibial nerve – Spastic paraplegia

Introduction

Joint position and vibration sense are commonly considered to be the specific modalities transmitted via the dorsal column, and somatosensory evoked potentials (SEPs) are assumed to travel primarily over the same pathway. However, more recently traditional views concerning the function of the dorsal columns in vibratory sensation have been questioned [6, 11, 18, 28] and, in addition, some studies [11, 22] have not been able to confirm a correlation between vibration sense deficits and peroneal or posterior tibial nerve (PTN) SEP impairment. Paradoxically, Greenberg and Erwin [10] reported PTN SEP changes to be related to the pyramidal syndrome. However, heterogeneous patient groups have been investigated in previous studies and neurological signs were not systematically analysed. Therefore, possible relationships remain questionable.

The clinical picture of the syndrome of slowly progressive spastic paraplegia (SP) is quite homogeneous, with an insidious onset and signs limited to the pyramidal tracts. The SP syndrome, therefore, forms a good model for relating clinical signs to SEP findings.

In the present study a group of 15 patients with slowly progressive SP was investigated. Seven patients had the hereditary form (HSP) [4, 5, 25] and 8 had SP without any identifiable cause or a positive family history, diagnosed as primary lateral sclerosis (PLS) according to the criteria set out recently by Younger et al. [29]. All patients were classified neurologically and neurophysiologically. The purpose of our study was to determine the frequency of abnormal PTN SEP features in relation to classified neurological signs occurring in our group of SP patients. In 8 of them the studies were repeated after 1–3 years.

Materials and methods

Normal values for PTN SEP features were derived from findings obtained in a control group consisting of 39 subjects, 28 females and 11 males, aged 12–60 years (mean 35 years). The height of each person was measured. In 5 control subjects SEP studies were repeated after 2–5 years.

Observations were made on 15 patients with SP, 7 females and 8 males, aged 25–75 years (mean 47 years). Seven patients had HSP and 8 PLS [3, 8, 24, 29]. The diagnosis of the former cases was based on the presence of a positive family history. In the latter cases there was no family history of similar disease, symptoms were restricted to progressive gait disorders and signs were re-

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stricted to corticospinal tract signs. Specific causes for pyramidal tract involvement were ruled out by extensive laboratory, radiological (MRI) and other neurophysiological investigations, including visual evoked potentials, electroneurography and EMG studies [29]. Examinations were repeated in 5 HSP and 3 PLS patients after 1–3 years.

A clinical examination was carried out just prior to the neurophysiological testing. Clinical examination and neurophysiological studies were performed by different investigators. The presence and severity of features were categorized in a clinical, practical manner. An increase in calf muscle tone was investigated by passive dorsiflexion of the ankle joint and was graded according to the Ashworth scale [2]: O, normal tone; 1, tone slightly increased, giving a catch on abrupt passive stretch of the triceps surae muscle; 2, tone increased; 3, tone markedly increased; 4, passive movement of the ankle barely possible. The strength of the triceps surae was judged according to the Medical Research Council (MRC) scale [16] and graded in four categories: 0, normal (MRC 5); 1, slight weakness (MRC 4); 2, moderate weakness (MRC 2 or 3); 3, severe weakness (MRC 0 or 1). Achilles tendon and knee reflexes were graded as: 0, diminished or absent; 1, normal; 2, increased; 3 clonus. Plantar reflex was scored as: 0, flexor; 1, equivocal; 2, extensor. Sensation of pain, vibration and joint position were classified as: 0, no deficit; 1, vibration loss only; 2, vibration loss, combined with deficits for pain or joint position; 3 deficits for pain, vibration and joint position. Vibration sense was investigated by use of a tuning fork.

A scale for PTN SEP latency time, corrected for height and according to the standard deviation (SD) limits of the control group, was made: 0, normal; under the upper limit of two times SD; 1, moderately delayed, between two and three times SD; 2, markedly delayed, between three and four times SD; 3, extremely delayed, more than four times SD; 4, no recordable potential.

For statistical analysis, values obtained on the most affected limb or when both sides were equally disturbed, only values obtained on the right side were used. Patients without a detectable SEP potential were excluded from ANOVA, while SEP amplitudes were set at 0.1 μV for statistical inquiry. If the SEP potential was absent for one leg, SEP parameters of the leg with a detectable SEP were taken for ANOVA.

Recording of the SEPs was carried out with the subject in a supine position in a quiet and dimly lit room. The subjects were encouraged to relax completely with eyes closed. The constant current stimuli were wave electrical pulses of 0.2 ms duration, delivered three times per second to the PTN at the medial side of the ankle, using a bipolar surface electrode. Stimulus intensity was adjusted to produce a small muscle twitch for PTN stimulation. Two series of 400 potentials were recorded, averaged and superimposed with a Nicolet Pathfinder 1 averager and checked for reproducibility. A blank recording was generated in advance without stimulation in order to estimate the signal to noise ratio. SEPs were recorded with surface electrodes over Cz′, 3 cm posterior to Cz and referred to Fpz, C3 and C4. Electrode impedances were less than 5 kΩ. The overall bandpass was 0.8 Hz to 3.3 kHz, the analysis time 100 ms, including a 10-ms prestimulus interval, with 512 points/channel.

PTN SEP recordings were performed on both limbs. The latency time was measured at P37. The amplitude was taken from the baseline to the positive peak.

Peripheral sensory nerve conduction in the PTN was assessed in both legs by placing a surface electrode in the fossa poplitea to record the mixed nerve potential. Furthermore, the soleus H-reflex was recorded as a measure of proximal nerve function. Formulae for normal latency time of the soleus H-reflex have been reported earlier [23]; the formula as a function of age (years) is $28.55 \pm (0.349 \times \text{age}) \pm 2.02$ (SD) and as a function of height (cm) $11.84 \pm (0.1056 \times \text{height}) \pm 1.80$ (SD).

The BMDP statistical computer program was chosen to computerize and analyse all the data obtained. To compare the patients with the control group an analysis of variance (ANOVA) was performed. We used the Tukey studentized range method (7D) for parametric and the Kruskal-Wallis and Mann-Whitney tests (3S) for non-parametric analyses. Parametric test results were checked by non-parametric statistics. To find correlations between clinical signs and SEP disturbances the Fisher test was computed in a two-way table (4F). *P*-values smaller than 0.05 were considered to be significant.

Results

Controls

The mean latency time for PTN SEP in the control group was 40 ms (range 35–46, SD 2.5) and the mean amplitude was 3.0 μV (range 0.5–7.1, SD 1.8). These results were similar to those reported by other investigators [13–15, 20]. A significant correlation ($r = 0.7$, $P < 0.001$) between latency time and height of the controls existed in our study and, therefore, a correction for height was made. The regression line for the PTN SEP was $9.4948 + 0.18068 \times$ height (cm). No other correlations, particularly for age, were found. Results for both limbs were similar and SEP figures of the right limb were taken for statistical analysis.

The mean PTN conduction velocity was 47 m/s (range 42–53, SD 3.0). No correlation with age was found and a velocity of 41 m/s (47 m/s − 2 SD) was used as the lower limit of normal.

In 5 healthy controls, in whom SEP studies were repeated after 2.5 years, the mean latency change was +0.6 ms (range −0.8 to +2.0, SD 0.9) and the mean amplitude change −0.9 μV (range −2.0 to +0.6, SD 0.9). The SEP altered in both directions. In 3 subjects the amplitude diminished slightly, while the latency time showed a slight increase in 2 and no change in 1. Two other subjects showed a higher amplitude, a decreased latency time for one leg and an increased latency for the other side. The SEP latency times remained between the range of two times SD. There is a wide range for normal SEP amplitude values, but amplitudes were not under the lower limit of the control mean minus the SD (1.2 μV) in the repeated investigations.

Patients

Clinical data and PTN SEP findings are presented in Table 1. There were no differences between the HSP and PLS group for the results of clinical and neurophysiological examinations. Signs of an upper motoneuron lesion existed, in various combinations, in all patients. All had normal strength (MRC 5, 7 patients) or slight weakness (MRC 4, 8 patients) of the legs. In 8 patients this was combined with a normal or slightly elevated muscle tone (Ashworth 0, 2 patients and Ashworth 1, 6 patients) and in 7 with a moderate to severely increased tone (Ashworth 2, 5 patients and Ashworth 3, 2 patients). Plantar responses were extensor in all patients. Hyperreflexia of the legs was present in 14 subjects (grade 2, 7 patients and grade 3, 7 patients) and in 1 subject Achilles tendon and knee reflexes were normal (grade 1). All patients were able to walk at least a few metres with assistance. No deficits were found for pain and joint position.

Table 1. Presentation of clinical data and posterior tibial nerve somatosensory evoked potential (SEP) findings in 15 patients with spastic paraplegia (SP), 7 with the hereditary form of the disease (HSP) and 8 with primary lateral sclerosis (PLS). A scale for latency, corrected for height, is given in parentheses; 0, within two times standard deviation (SD); 1, between two and three times SD; 2, between three and four times SD; 3, more than four times SD; 4, no recordable (nr) SEP potential

Patient no.	SP form	Age (years)	Height (cm)	Sex	Muscle tone (Ashworth)	Vibration loss	SEP	
							Latency (ms)	Amplitude (μV)
1	PLS	74	163	f	0	0	45 (1)	3.0
2	HSP	49	190	m	3	1	45 (0)	0.5
3	PLS	44	179	m	2	0	nr (4)	0.1
4	HSP	50	170	m	1	0	38 (0)	0.8
5	PLS	46	183	m	1	0	47 (0)	0.7
6	PLS	48	182	m	2	0	47 (1)	0.7
7	HSP	52	170	f	1	0	51 (3)	0.5
8	PLS	49	186	m	2	0	49 (1)	0.2
9	HSP	43	168	f	1	0	42 (0)	1.3
10	HSP	43	172	f	2	0	46 (1)	0.3
11	HSP	59	174	m	3	1	42 (0)	1.2
12	PLS	31	181	m	2	0	45 (0)	1.5
13	HSP	37	163	f	1	0	nr (4)	0.1
14	PLS	25	165	f	0	0	48 (2)	0.1
15	PLS	52	181	f	1	0	50 (2)	0.9

Table 2. Changes of spasticity and of posterior tibial nerve SEP latency in 8 patients who were re-examined after 1–3 years

SEP latency	Spasticity intensity (Ashworth)			Total
	Increase	Stable	Decrease	
Increase	2	1	1	4
Decrease	1	2	1	4
Total	3	3	2	8

Table 3. Posterior tibial nerve SEP latency times, corrected for height [9.4948 + 0.18068 × height (cm)] found in the controls and patients. The mean value is indicated by "M"

Latency (ms)	Controls	Patients
12		
11		*
10		
9		*
8		*
7		*
6		**
5	*	M*
4	*	
3	**	**
2	*	**
1	******	
0	M********	
−1	*******	
−2	**	*

A vibratory disturbance (score 1) was present in 4 patients for at least one leg. In 2 of them the leg with vibration loss was used for ANOVA (Table 1). In 11 patients no vibration deficit was present.

In the 8 patients who were re-examined after 1–3 years, the following was found; 2 showed a decrease of their lower limb spasticity, according to the Ashworth scale (1 from grade 1 to 0 and 1 from grade 2 to 1), 3 remained stable (1 with Ashworth 1, 1 with Ashworth 2 and 1 with Ashworth 3) and 3 patients showed an increase (1 from grade 2 to 3 and 2 from grade 1 to 2) (Table 2). Weakness of the lower limbs increased from 1 to 2 in 1 patient and tendon reflexes become more abnormal in 5. Sensory functions according to the clinical classification improved in 2 patients. The other clinical modalities did not change in any of the patients.

Latency times and amplitudes of PTN SEPs are shown in Table 1. A scale for SEP latency time, according to normal SDs and corrected for height, is given in parentheses. The mean latency time was 45 ms (range 38–51) and the mean amplitude 0.7 μV (range 0.1–3.0). In two patients no SEP potentials could be recorded (scale 4) on either side. In 6 patients (Table 1) latency time fell within two times SD and was regarded as normal (grade 0); 4 patients had latency times between two and three times SD (grade 1, moderately delayed); 2 patients scored between three and four times SD (grade 2, markedly delayed); and 1 patient had the most significant delay, being outside the range of four times SD (grade 3). Peripheral PTN conduction velocity (mean 47 ms, range 41–53 ms) and soleus H-reflexes were normal in all patients.

Tables 3 and 4 present PTN SEP latency times (corrected for height) and amplitudes in the controls and patients. Differences in both SEP parameters between the two groups were significant ($P < 0.01$).

Abnormal PTN SEPs were present in our patients without sensory disturbance or with loss of vibration sense only (Fig. 1). The PTN SEP impairment was neither related to weakness of the legs, nor to increased tendon

Table 4. Posterior tibial nerve SEP amplitudes found in the controls and patients. The mean value is indicated by "M"

Amplitude (μV)	Controls	Patients
7.5		
7.0	**	
6.5		
6.0	*	
5.5		
5.0	*	
4.5	*	
4.0	**	
3.5	**	
3.0	M	*
2.5	******	
2.0	*******	
1.5	*****	**
1.0		M**
0.5	*	*****
0.0		****

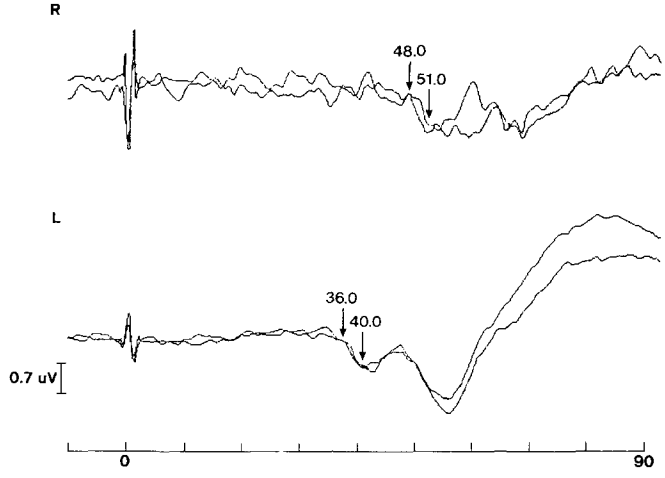

Fig. 1. The posterior tibial nerve somatosensory evoked potential (SEP) in a patient (Table 1; no. 7), with a delayed SEP latency at P37 (51.0 ms) and an amplitude of 0.5 μV on the right side (R) and a normal SEP latency (40.0 ms) and an amplitude of 0.7 μV on the left (L). Muscle tone was slightly (Ashworth 1) elevated on the right and normal (Ashworth 0) on the left side. There was no weakness of leg muscles or sensory impairment

reflexes. There was no significant correlation (Fisher score $P = 0.5$) between abnormal muscle tone and the presence or absence of PTN SEP (Table 5). In a two-way table an absent SEP potential in at least one leg (5 cases) did not correlate with severe spasticity (Ashworth 2 or 3; 3 cases), while not all patients without or with slight spasticity (Ashworth 0 or 1; 8 cases) had the PTN SEP potential present (6 cases). No correlation was found between SEP abnormalities and disease duration.

In the 8 SP patients, 5 HSP and 3 PLS patients, in whom neurological and neurophysiological examinations were repeated, the mean latency change was −2.6 ms (range −5.4 to +2.6) and the mean amplitude change

Table 5. Severity of spasticity and reproducibility of posterior tibial nerve SEPs

SEP	Spasticity intensity (Ashworth)		Total
	Slight	Severe	
Absent	2	3	5
Present	6	4	10
Total	8	7	15

−0.2 μV (range −0.4 to +0.1). In 4 patients the latency time changed more than the normal SD value of 2.5 ms compared with the first examination. In 2 of them the latency time decreased from grade 1 to grade 0 and from grade 3 to 0, respectively. In the other 2 patients the latency time increased from grade 1 to grade 3. The latency times of the remaining 4 subjects stayed within the SD scale seen in the first SEP measurement. Amplitude changes were small, but all patients already had low amplitudes (0.2–1.2 μV, mean 0.7) at the first SEP examination. One patient, in whom latency time decreased, showed no loss of vibration sense during the repeated investigation, while a deficit for vibratory sensation was observed in one leg during the first examination. The other patients showed no change in vibratory sense. Therefore, no relation between vibration loss and SEP change chould be established. Change of spasticity was not correlated with change of PTN SEP latency time or with its amplitude. In 2 patients SEP potentials disappeared; 1 of them showed an increase and the other a decrease of spasticity, according to the Ashworth scale. Latency time increased in 2 subjects; spasticity worsened in 1 and remained stable in the other. Latency time decreased in 1 who worsened, in 2 who remained stable and in 1 patient with a decrease in Ashworth scale grade (Table 2). No correlation could be established between SEP changes and change of reflexes or strength of the triceps surae.

Discussion

There are two important observations from the present study. Firstly, although the preponderance of evidence favours vibration and joint position sense as being most related to SEP changes [1, 9, 17, 27, 30], our study demonstrated that significant SEP changes can occur in spastic paraparesis without sensory impairment or with loss of vibration sense only, observed with the routine neurological examination. This contradiction may, at least partly, be explained by the fact that in most studies heterogeneous patient populations were investigated without accurately defining both sensory and motor impairment. Secondly, no significant correlation was established between PTN SEP absence or presence and severity of spasticity for the whole patient group. Our SEP findings are similar to the PTN SEP abnormalities in spastic patients with sensory deficits reported by Greenberg et al. [10, 11]. However, in the present study no relation

between PTN SEP abnormalities and severity of the pyramidal syndrome was found.

PTN SEP latencies in our 5 control subjects showed a sufficient intraindividual reproducibility over a long period of time. In some of our patients in whom studies were longitudinally repeated, latency changes were present outside the normal upper limits of SD without progressive decline of their disease. Others, who worsened clinically, showed even shorter latency time during repeated measurements. Therefore, as in the patient group, in the individual patients also there was no relation between changes of PTN SEPs and clinical signs. Amplitude reproducibility of PTN SEP is not consistent, considering the variety of amplitudes in our controls after 2.5 years. Consequently, amplitude changes are less useful as a quantitative measure in repeated studies.

With regard to our 7 HSP patients, PTN SEPs with decreased amplitudes and normal latency times have been reported by several authors [12, 19, 26]. In other studies [7, 21], however, both decreased amplitudes and prolonged latency times were found. Our results, suggesting demyelinization and axonal degeneration of fibres conducting impulses for PTN SEPs, are more in accordance with the findings reported by the latter investigators. Pedersen and Trojaborg [19] and Thomas et al. [26] made no correction for height and Imai et al. [12] studied a small group of five young siblings (10–18 years) and, therefore, in these studies prolonged latency times could have been missed.

Neuropathological studies in HSP have revealed bilateral degeneration of the corticospinal tract and adjacent structures that increases from the cervical to the lower lumbar level, while degeneration of the funiculus gracilis in both posterior columns increases from the lumbar to the upper cervical level [4, 5, 25]. Conversely, in PLS no or only slight degeneration of both posterior columns has been found with severe degeneration of the lateral corticospinal tracts through the entire spinal cord [3, 8, 29]. Because of the similar PTN SEP abnormalities in HSP and PLS and the distinctive involvement of the posterior columns, it may be suggested that impulses for PTN SEPs may use other routes through the spinal cord than the funiculus gracilis alone.

In summary, in a group of SP patients we found prolonged latency times and decreased amplitudes of PTN SEPs. These SEP changes were not significantly related to pyramidal signs, and were present without sensory impairment or with vibration loss only. PTN SEP abnormalities, therefore, may indicate a feature of spinal cord dysfunction that is not related to the severity of clinical signs observed by the routine neurological examination. Further studies in selected patient groups are necessary to draw conclusions on possible locations of spinal pathways for impulse conduction of various SEP cortical potentials, elicited by electrical stimulation of lower limb nerves.

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