Short-latency somatosensory evoked potentials (SSEPs) of the tibial nerves in spinal cord injuries

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Stimulations of the tibial nerves of 76 spinal cord injured patients revealed short-latency somatosensory evoked potentials (SSEPs) especially P37 recorded from the scalp. The SSEPs findings can be classified by degrees of impairment into 5 different types as follows:

Type 1: Absence of P37. This was found in all patients with complete cord lesions as well as patients with loss of joint sense.

Type 2: Decreased amplitude and prolonged latency. This was elicited in patients with moderate to severe central cord lesions.

Type 3: Prolonged latency only.

Type 4: Decreased amplitude only.

Type 5: Normal P37, which was mostly seen in patients with mild central cord lesion.

The study demonstrated that SSEPs of the tibial nerves are related to joint sense and seem to relate to the extent of cord damage, especially of central cord and complete cord lesions. It can be done as early as during the first week post injury, particularly in an uncooperative patient suspected of having spinal cord injury, in which case the clinical assessment may be difficult and unreliable.

Key words: short-latency somatosensory evoked potentials (SSEPs); spinal cord injury.

Introduction

After using computer averaging technique, cerebral response and somatosensory evoked potentials (SEPs) following electrical stimulation of peripheral nerve in man were first reported by Dawson.¹ Thereafter, many studies of SEPs have been published concerning techniques, their origins and clinical uses.

Early study in patients with spinal cord injuries showed that no SEPs were elicited in complete cord lesion whilst in some patients with an incomplete lesion, such potentials were present.² Subsequently, others demonstrated the presence of SEPs in cases with a complete lesion,^{3,4} and concluded that there was a better prognosis of recovery.^{5,6} This was opposed by others. The presence of SEPs does not always predict recovery, it only permits quantitative evaluation of the somatosensory sys-

tem, especially of joint sense.^{7,8} This was confirmed by the study in those with a Brown-Séquard lesion showing impairment of cortical SEPs after stimulation of the leg ipsilateral to the side of cord injury,⁹ and by animal studies showing that dorsal column, dorsolateral and dorsal spinocerebellar tracts are responsible for SSEPs; whereas the anterolateral (spinothalamic) tract affects later waves.^{10,11,12}

This investigation was to study whether SSEPs would show any relation to different clinical cord lesions, and may give enough evidence regarding the integrity of the spinal cord after injury.

Methods

Neurological examination was performed on every spinal injured patient with a neurological deficit admitted to the Maharaj

Nakorn Chiang Mai Hospital. Motor and sensory modalities were graded according to the severity of impairment (Table I). Then, clinical cord lesions were identified regarding neurological deficits (Table II).

Using a Neuromatic 2000C, SSEPs were recorded with bandpass filter set at 20–2000 Hz. Constant current square-wave pulses of 200 microsecond duration at a rate of 3 per second were delivered by stimulating electrodes placed over the tibial nerve behind the medial malleolus. Current strength was determined for each subject until movements of the toes were seen or a maximum at 20 mAmp. Lumbar and scalp SEPs were recorded from the first lumbar spine referenced to the contralateral iliac crest and from Cz referenced to Fz (International 10–20 system) respectively by using needle recording electrodes. This cephalic reference (Fz) was chosen to minimise contamination by artifacts.¹³ Patients should be relaxed in the supine position to reduce myogenic potentials.¹⁴ At least 200 sweep repetitions were averaged for each

trial and 2 replications were performed to identify the exact responses. Amplitude and latency of lumbar potential (LP) and SSEPs, ie. the first positive (downward) peak labelled P37,¹⁴ were then compared with the control data of our previous study¹⁵ as follows:

Mean predictive LP latency = $-5.939 + 0.155\text{Height}(\text{cm})$; SE = 0.981.

Mean LP amplitude (baseline to peak) = 1.24 microV; SD = 0.38.

Mean predictive P37 latency = $-6.159 + 0.256\text{Height}(\text{cm})$; SE = 1.404.

Mean P37 amplitude (baseline to peak) = 1.97 microV; SD = 0.83.

The latency is prolonged if it is more than mean + 3SE, while amplitude is decreased if it is less than mean – SD. Due to the correlation between latency and height,¹⁵ we compared the patients' data with the predictive values corrected by height as shown above.

Table I Grading of motor and sensory impairments below level of injury

Grade	Motor	Pinprick	Joint sense
0	loss	loss	loss
1	useless	diminished	appreciation of movement
2	useful	normal	appreciation of movement and direction, but less reliable
3	normal		normal

Table II Clinical cord lesions according to neurological impairments

Lesions	Degrees of neurological impairments		
	Motor	Pinprick	Joint sense
Complete cord transection	0	0	0
Anterior cord	0–1	0–1	3
Central cord			
severe	0	0–1	0–2
moderate	1	1	1–2
mild	2	1–2	1–3
Posterior cord	3	2	0–1
Others			
motor preserved only	1–2	0	0
unclassified	1–2	1	0

Results

There were 100 patients in this study but only 76 spinal cord injured patients were included. The others were excluded due to abnormal or absence of LP suggesting conus medullaris or cauda equina lesions. Forty-seven patients were quadriplegic and 29 were paraplegic. There were 36 complete and 40 incomplete lesions. Average age was 36 years. Most patients were evaluated within the first month and only 8 cases were studied within the first week post injury.

According to SSEPs, there were 5 different types of tibial nerve P $\overline{37}$ findings among these patients concerning the severity of impairment as follows (Fig 1):

Type 1: absence of P $\overline{37}$.

Type 2: decreased amplitude and prolonged latency.

Type 3: prolonged latency only.

Type 4: decreased amplitude only.

Type 5: normal P $\overline{37}$.

The relationships between P $\overline{37}$ findings

and joint sense as well as clinical cord lesions are shown in Table III and IV respectively.

Type 1 was found in all who had complete cord lesions (36 cases) as well as in patients with either total loss or moderate to severe impairment of joint sense.

Type 2 was mostly elicited in patients with moderate to severe impairment of joint sense, and in cases with moderate to severe central cord lesions.

In one patient with useful motor and diminished pinprick sensation but loss of joint sense, type 2 was detected. This was done on the eighth day post injury. On the other hand, there were 7 cases (13 responses) whose joint sense was preserved but impaired, where P $\overline{37}$ could not be identified. In only 2 of these, the SEPs studies were done within the first week after injury. While 6 out of 8 cases retaining joint sense were also examined during the first week, P $\overline{37}$ was still preserved.

Type 3 was seen in patients with mild to moderate impairment of joint sense, and

Figure 1 Five different types of P $\overline{37}$ findings in spinal cord injuries. Each pair of traces depict repetition of evoked responses from the same side.

Table III The relation between joint sense and SSEPs (P37) findings

Joint sense Grade	Types of P37	No. of responses				
		1	2	3	4	5
0		80	1			
1		11	13	1	4	3
2		2	2	3	1	3
3			2		7	19

There were 2 responses for each patient and a total number of 152 responses were evaluated.

Table IV The relation between clinical cord lesions and SSEPs (P37) findings

Lesions	Total cases	Types of P37	No. of responses				
			1	2	3	4	5
Complete cord	36		36				
Anterior cord	1				1		
Central cord							
severe	5			4	1		
moderate	11		3	3		2	3
mild	19		2	1	1	3	12
Motor preserved only	2		2				
Unclassified	2		2				

Numbers in this table indicate the summation of the best responses between the two sides of P37 findings in each case.

types 4 and 5 were usually elicited in cases of mild central cord lesions or in patients with normal to mild impaired joint sense.

Discussion

The results of this study showed that SSEPs are related to joint sense and also confirmed that the less the degree of joint sense impairment is, the more likely will the P37 findings tend to be normal (from type 1 to 5). In mild degree impairment, either amplitude or latency of P37 is affected, while with severe impairment both parameters are usually affected.

This study advocated that the dorsal spinocerebellar tract carrying impulses from receptors in the joints and muscles was responsible for 'joint sense' of the lower or hind limb,¹⁶ and is the main pathway of propagation of the evoked responses.¹²

Regarding those with a complete cord lesion, many studies have advocated its relation to the loss of SSEPs,^{2,8} while others reported the presence of SSEPs in some

cases with a clinical lesion.⁴ In this study, no patients with complete cord lesion with preservation of P37 were found. It indicates that if SSEPs are absent, either a complete or a severe central cord lesion with severe impairment of joint sense should be considered. The presence of SSEPs reflect an incomplete lesion. However, the type of SSEPs findings may help in predicting the degree of joint sense impairment and the severity of the clinical cord lesion, especially the extent of central cord damage and complete cord transection.

The time at which the test (SEPs) was done did not seem to affect the evoked responses. Although some patients, those with preserved joint sense, were examined within the first week after injury, SSEPs were still elicited. Oedema at the site of injury did not alter the result, as reported previously.¹⁷ However, if there is clinical improvement regarding joint sense, the SSEPs will improve in latency and/or amplitude. Because of the rarity of posterior cord lesion, the persistence of absence of

SSEPs may be used to predict complete cord lesion or severe cord damage.

Although this investigation did not study long-latency SEPs which were said to propagate via the anterolateral tract in animal studies,^{10,11,12} there was one case with loss of joint sense but preserved pinprick sensation, showing abnormal low amplitude and prolonged latency of P37. However, we could not conclude that the spinothalamic tract was responsible for this finding.

To date, the study of SSEPs is an adjunctive procedure in the evaluation of joint

sense and of the integrity of the spinal cord in those whose clinical assessment may be difficult and unreliable. It can be performed with expected accurate results as early as in the first week after injury and can be repeated later to confirm a definite interpretation. The presence or the improvement of SSEPs findings do not ensure a good prognosis of functional recovery because SSEPs only represent an intact sensory pathway in the spinal cord. However, the degree of SSEPs impairment also seems to relate to the severity of a central cord lesion.

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