Somatosensory evoked potential from S1 nerve root stimulation

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Received: 20 April 2010 / Revised: 19 November 2010 / Accepted: 7 March 2011 / Published online: 10 May 2011
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Abstract The objective of this study was to detect cerebral potentials elicited by proximal stimulation of the first sacral (S1) nerve root at the S1 dorsal foramen and to investigate latency and amplitude of the first cerebral potential. Tibial nerve SEP and S1 nerve root SEP were obtained from 20 healthy subjects and 5 patients with unilateral sciatic nerve or tibial nerve injury. Stimulation of the S1 nerve root was performed by a needle electrode via the S1 dorsal foramen. Cerebral potentials were recorded twice to document reproducibility. Latencies and amplitudes of the first cerebral potentials were recorded. Reproducible cerebral evoked potentials were recorded and P20s were identified in 36 of 40 limbs in the healthy subjects. The mean latency of P20 was 19.8 ± 1.6 ms. The mean amplitude of P20–N30 was 1.2 ± 0.9 μ V. In the five patients, P40 of tibial nerve SEP was absent, while

well-defined cerebral potentials of S1 nerve root SEP were recorded and P20 was identified from the involved side. This method may be useful in detecting S1 nerve root lesion and other disorders affecting the proximal portions of somatosensory pathway. Combined with tibial nerve SEP, it may provide useful information for diagnosis of lesions affecting the peripheral nerve versus the central portion of somatosensory pathway.

Keywords Somatosensory evoked potentials · Intra-operative monitoring · S1 nerve root · Nerve root stimulation

Introduction

The major clinical application of somatosensory evoked potential (SEP) is to detect functional or dysfunctional transmission through somatosensory pathway from the peripheral nerve, root, spinal cord, brainstem to the cerebrum. Routine SEP techniques are performed following stimulation of the common peroneal nerve at the fibular

Part of the present study was presented at the 34th ISSLS meeting in Hongkong in July 2007 (No. 144) and presented at the AOCCN2009 in Seoul in April 2009 (No. PO3.17).

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head or tibial nerve at the ankle, approximately 50% of the total conduction time is consumed at the periphery. SEP obtained following proximal stimulation of sciatic nerve [1] and femoral nerve [2] can elicit reproducible cerebral and spinal potentials. The techniques bypass much of the peripheral nerve in the limb, therefore, maximize the central component in the latency. Bamford et al. [1] reported that sciatic nerve SEP can be used in patients with severe distal nerve dysfunction which invalidates conventional tibial nerve SEPs. Robinson et al. [2] demonstrated that femoral nerve SEP was useful for intraoperative monitoring during spinal surgery involving T12–L4 vertebral levels. SEP obtained from proximal stimulation of S1 nerve root is another useful SEP technique, which has rarely been reported in previous studies. We report a method of recording cerebral evoked potentials following proximal stimulation of the S1 nerve root at the S1 dorsal foramen.

Materials and methods

Radiological study in localization of S1 dorsal foramen

We first conducted a pilot study in localization of S1 dorsal foramen by radiographs. The anterior–posterior pelvis films were performed on 40 healthy volunteers (24 male

and 16 female). The radiological study showed that S1 dorsal foramen lies lateral to the midpoint of the posterior superior iliac spine (PSIS) and the median sacral crests. In the coronal plane, S1 dorsal foramen lies 7.3 ± 2.8 mm lateral to the midpoint, 25.6 ± 2.8 mm lateral to the median sacral crests and 16.1 ± 2.5 mm distal to the S1 superior endplate. These anatomical data suggest that a lateral approach to the midpoint should be advocated for S1 nerve root stimulation (Fig. 1).

S1 nerve root SEP study

Written informed consent was obtained prior to the study, which was approved by the Ethics Committee of Fudan University. Sixteen male and nine female volunteers participated in the SEP study. The ages varied from 22 to 56 years (mean 41 years) and the heights varied from 153 to 184 cm (mean 169 cm).

The subjects included 20 healthy volunteers (thirteen male and seven female) and 5 patients (three male and two female) with unilateral sciatic nerve or tibial nerve injury over a year earlier. None of the healthy subjects had a history of radicular pain, peripheral neuropathy, back pain, alcoholism, limb trauma, diabetes mellitus or Guillain–Barré syndrome. Two of the three patients who had a unilateral sciatic nerve injury had posterior dislocation of hip joint and the other one had laceration of the sciatic

Fig. 1 The anatomical relationship of S1 dorsal foramen and the adjacent structures on the pelvic anterior–posterior film. Note that the S1 dorsal foramen lies lateral to the midpoint of the PSIS and the median sacral crest

nerve. The other two patients who had a unilateral tibial nerve injury had lower limb trauma. All the patients were free of other neurological diseases. The sciatic nerve and tibial nerve injuries were confirmed by clinical and electrophysiologic findings.

SEPs were recorded using an electromyogram (Nihon Kohden MEB-9400, Japan). Routine posterior tibial nerve SEPs were obtained following stimulation of posterior tibial nerve at the ankle with current sufficient to induce a clearly visible flexion of the toes. The stimulus intensity ranged from 12 to 20 mA, the stimulus duration was 0.2 ms and its frequency was 3 Hz.

The dorsal ramus of the first sacral nerve root was stimulated by means of a 50 mm monopolar teflon-coated stainless-steel needle electrode with an 1 mm bare tip, which was inserted into the first dorsal sacral foramen located lateral to the midpoint of the PSIS and the median sacral crest [3]. A surface electrode was placed ventrally at the inguinal groove to complete the stimulus circuit. The needle was advanced into the sacral foramen while a low-intensity (less than 5 mA) repetitive stimulus was being applied. As soon as a repetitive plantar flexion was noted of the toes or ankles, the needle was secured into position. At the optimal site, a S1 nerve root H-reflex was induced from soleus muscle to ensure that the afferent fibers of S1 nerve root were stimulated [4, 5] (Fig. 2).

For SEPs elicited from S1 nerve root stimulation, the stimulus duration was 0.2 ms and frequency was 3 Hz. The intensity was adjusted to 20% above the motor threshold of S1 nerve root-innervated muscles, and ranged from 6 to 18 mA.

For the soleus H-reflex from S1 nerve root stimulation, the stimulus duration was 0.2 ms and the intensity ranged from 10 to 15 mA.

Fig. 2 Soleus H-reflexes from electrical stimulation of S1 nerve root at the S1 dorsal foramen from both sides of a normal subject. The intensity of the nerve stimulus is to the right of the traces

Cerebral potentials were recorded from subcutaneous needle electrodes at Cz' referenced to Fpz. Electrode impedances were maintained close to one another and measured below 2 k Ω . Recordings were made with a sensitivity of 2.5 μ V/division for tibial nerve SEP and 1 μ V/division for S1 nerve root SEP. The filter settings were 50 Hz and 2 kHz. A ground electrode was placed in the popliteal fossa for the tibial nerve SEP and in the lumbar area for S1 nerve root SEP. Over 250 responses were averaged for each tracing. At least two tracings were obtained to document reproducibility.

All subjects underwent the procedures of bilateral tibial nerve SEPs and S1 nerve root SEPs. Scalp SEPs were recorded from 50 lower limbs in the 25 individuals.

Statistics

SPSS 12.0 was used for statistical analysis. The means and standard deviations were calculated for latency and amplitude of the first cerebral potentials of tibial nerve SEPs and S1 nerve root SEPs in the 20 healthy subjects.

Results

S1 nerve root SEP study in healthy subjects

The procedures were tolerated well by all subjects. No one complained of suffering or pain after the procedure.

In the 20 healthy subjects, the latency of P40 and amplitude of P40–N50 of the tibial nerve SEP were almost identical on the two sides. The mean latency of P40 was 38.8 ± 2.0 ms. The mean amplitude of P40–N50 was 3.2 ± 1.8 μ V (Table 1).

In the healthy subjects, for S1 nerve root SEP tests, reproducible cerebral evoked potentials with several components were recorded bilaterally from 16 subjects and unilaterally from four subjects. The first positive potential P20 was a consistent cerebral positivity with a stable latency at around 20 ms followed by a negativity N30 with a more varying latency (Figs. 3, 4). The latencies of P20 ranged from 17.2 to 23.4 ms (mean 19.8 ± 1.6 ms). The amplitudes of P20–N30 ranged from 0.2 to 3.3 μ V (mean 1.2 ± 0.9 μ V) (Table 1). The amplitude of P20–N30 was less than 1 μ V in 18 healthy limbs and was equal or greater than 1 μ V in 18 healthy limbs.

In the four healthy subjects (subject 9, 11, 12 and 14), for S1 nerve root SEPs, cerebral potentials and P20 component were absent on one side of each subject. S1 nerve root H-reflex could only be elicited with a higher stimulus intensity (more than 25 mA) in these limbs.

Table 1 The mean values and inter-leg difference of latencies and amplitudes of P40 component of tibial nerve SEP and P20 component of S1 nerve root SEP in healthy subjects

Parameter	Tibial nerve SEP			S1 nerve root SEP		
	Range	Mean	SD	Range	Mean	SD
Latency (ms)	34.3–42.3	38.8	2.0	17.2–23.4	19.8	1.6
Amplitude (μ V)	1.2–8.8	3.2	1.8	0.2–3.3	1.2	0.9
Inter-leg latency difference (ms)	0–1.4	0.6	0.5	0.2–1.6	0.5	0.4
Inter-leg amplitude difference (μ V)	0.1–1.0	0.4	0.3	0–0.7	0.2	0.2

Fig. 3 a Cerebral potentials elicited by stimulation of posterior tibial nerve at the ankle from both sides of subject 1. **b** Cerebral potentials elicited by stimulation of S1 nerve root from both sides of subject 1

S1 nerve root SEP study in patients with peripheral nerve injury

In the patients with unilateral sciatic nerve or tibial nerve injury, all the patients had XR to assist localization of the S1 dorsal foramen, because S1 nerve root H-reflex could not be induced from soleus muscle on the involved side. P40 of tibial nerve SEP was absent from the involved side in each patient. However, well-defined cerebral potentials of S1 nerve root SEP were recorded and P20 component was identified from the involved side with a normal amplitude and latency (Fig. 5) (Table 2).

Discussion

In this study, reproducible cerebral evoked potentials were recorded following needle stimulation of the S1 nerve root at the S1 dorsal foramen in normal subjects. Cerebral potentials of S1 nerve root SEP were also recorded when P40 of tibial nerve SEP was absent in patients with sciatic nerve or tibial nerve injury. This SEP technique bypasses much of the peripheral nerve in the lower limb, thus it provides a new approach to quantify the physiologic function of the proximal portion of somatosensory pathway. Electrical stimuli eliciting peripheral nerve SEP and magnetic stimuli eliciting muscle SEP both lack the

specificity to detect a single nerve root lesion or deficit, because they are conducted by multiple segments of nerve roots. Several investigators have reported that dermatomal spinal SEP was specific in detecting acute lumbosacral nerve root injury [6, 7]. S1 nerve root SEP also shares the specificity for evaluating conduction of a single nerve root, owing to a near-nerve electrical stimulation of the nerve root.

The first positive cerebral potential, P20

In this study, P20, a positive potential peaking at latency around 20 ms, was the first cerebral potential to S1 nerve root SEPs. It has been described that after stimulation of tibial nerve at the ankle, the lumbar N22 was recorded at the spinal level of T12 or L1 [8], N19 was recorded at the spinal level of L5 [1] and N17 was recorded at S1 dorsal foramen. The expected latency of the first cerebral potential of S1 nerve root SEPs can be calculated as the P40 latency of tibial nerve SEP subtracting N20 latency of the lumbar CNAP, which is consistent with a latency of 20 ms for the first cerebral potential to S1 nerve root SEP.

A poor signal-to-noise ratio (SNR) was a limitation of this technique. In this study, the amplitude of P20–N30 was less than 1 μ V in 18 healthy limbs and P20 component of S1 nerve root SEP could not be identified from four healthy limbs. We had postulated that a stimulus of sufficiently

high intensity would depolarize afferents from both the proximal and distal parts of the leg, and should lead to large SEPs. However, the scalp field distribution of SEP to

Fig. 4 Cerebral potentials of S1 nerve root SEP from six limbs of six normal subjects. Note the reproducibility of P20 potentials and the following components

Fig. 5 Cerebral potentials of bilateral tibial nerve SEP and S1 nerve root SEP from a patient with unilateral sciatic nerve injury. **a** Tibial nerve SEPs. **b** S1 nerve root SEPs. Note the absence of P40 of tibial nerve SEP and the robust P20 of S1 nerve root SEP from the involved side

the proximal leg stimulation is different from that of the distal leg stimulation [9]. The sensory input of the distal leg projects approximately to the inferior portion of the paracentral lobule, while the proximal leg is represented either close to the superior lip of the interhemispheric fissure or over the lateral convexity. Simultaneous activation of separate SI areas and the different orientations of individual P20 dipole vectors might lead to a low amplitude or the occasional absence of an identifiable P20 in a poorly defined cerebral evoked potential. In addition, there might be occlusion due to convergence between the afferent inputs from proximal and distal parts of leg along the somatosensory pathway [10, 11]. Furthermore, the poor SNR might also be due to anomalous innervation patterns in the lumbosacral spine. SEPs may be mediated from a post-fixed distribution of lower sacral nerve roots (e.g. S2–S3) or a pre-fixed distribution of higher lumbar nerve roots (e.g. L4–L5) [7]. Variations in innervation patterns can also occur within subjects, this may account for the lack of unilateral SEPs in the four healthy subjects.

Importantly, a weak electrical current was utilized for nerve location in S1 nerve root stimulation by needle electrode. Motor responses elicited by very low stimulus intensity indicated close proximity to or contact between needle tip and nerve [12–14]. Based on Coulomb's law, increase of stimulus intensity allows stimulation of a nerve at greater distance. By contrast, use of low stimulus intensity leads to greater accuracy in ultimate position of the needle tip relative to the nerve [15, 16]. In the four healthy subjects, S1 nerve root H-reflex could only be elicited with a higher stimulus intensity (more than 25 mA). This indicated that the needle tip was not advanced into the S1 dorsal foramen to get proximity to the S1 nerve root, therefore, a sufficiently high stimulus intensity would be required to allow activation of the S1 nerve root, this will inevitably depolarize the adjacent sacral nerve roots (S2–S4). We postulated that a non-specific stimulation of multi-level sacral nerve roots (including

Table 2 Latencies and amplitudes of the Cz'-Fpz cerebral potentials to P40 for tibial nerve SEP and P20 for S1 nerve root SEP in patients with peripheral nerve injury

Subject	Side	P40 Lat. tibial (ms)	P40 Lat. Diff.	P40–N50 Amp. tibial (μV)	P40–N50 Amp. Diff.	P20 Lat. S1 (ms)	P20 Lat. Diff.	P20–N30 Amp. S1 (μV)	P20–N30 Amp. Diff.
1	R	–	–	–	–	21.8	0.4	1.5	0.3
	L	36.7		2.7		22.2		1.8	
2	R	–	–	–	–	18.4	1.2	0.5	0.2
	L	38.6		3.0		17.2		0.3	
3	R	40.1	–	2.4	–	20.2	0.6	1.1	0.1
	L	–		–		19.6		1.2	
4	R	–	–	–	–	18.8	0.5	0.6	0.2
	L	39.2		1.8		18.3		0.4	
5	R	38.4	–	38.4	–	18.9	0.4	0.9	0.3
	L	–		–		19.3		1.2	

–, Not detected

S1 nerve root) was able to induce H-reflex from soleus muscle, but was unlikely to elicit reproducible cerebral evoked potentials.

The potential applications of S1 nerve root SEP technique

The technique of S1 nerve root SEP may provide useful information for diagnosis of lesions affecting the peripheral nerve versus the central portion of somatosensory pathway. In the five patients with unilateral sciatic nerve injury, the tibial P40 was absent from the involved side. However, the S1 nerve root P20 was identified bilaterally (Fig. 5). Since S1 nerve root SEP is performed by proximal stimulation at the S1 dorsal foramen, the method bypasses lesions affecting the peripheral nerve. Therefore, the abnormal tibial nerve SEP and normal S1 nerve root SEP indicates a lesion affecting the peripheral portion of somatosensory pathway. On the other hand, an abnormal tibial nerve SEP and an abnormal S1 nerve root SEP would indicate a lesion affecting the central portion of somatosensory pathway.

The method of S1 nerve root stimulation could also be utilized for S1 nerve root H-reflex. Hoffmann et al. first described the routine H-reflex occurring in calf muscles of humans on stimulation of tibial nerve at the popliteal fossa. Recent studies have documented that the soleus H-reflex can be elicited by magnetic or high voltage electrical stimulation at the S1 dorsal foramen [4, 5]. This study also proved that the soleus H-reflex could be elicited by near-nerve electrical stimulation. Valls-Solé J et al. [17] has indicated that magnetic stimulation and near-nerve electrical stimulation activated different nerve afferents, the former probably activated both various lumbosacral sensory afferents and intramuscular nerve afferents, the latter might preferentially activated large muscle afferents.

In summary, reproducible cerebral evoked potentials could be recorded following needle stimulation of the S1 nerve root at the S1 dorsal foramen in normal subjects. This method may be useful in detecting S1 nerve root lesion and other disorders affecting the proximal portions of somatosensory pathway. Combined with tibial nerve SEP, it may provide useful information for diagnosis of lesions affecting the peripheral nerve versus the central portion of somatosensory pathway.

Finally, the major limitation of this study was the small sample size. In S1 nerve root SEP tests, cerebral evoked potentials were absent from 4 healthy limbs in the 20 healthy subjects. However, reproducible cerebral evoked potentials of S1 nerve root SEP were recorded bilaterally in the 5 patients. This small sample size may induce low statistical power and bring false-negative error to the results.

Acknowledgments The authors thank Dr. David Burke for his helpful review and comments of the manuscript.

Conflict of interest None.

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