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Original Article

The Central Conduction Time in Posterior Tibial and Pudendal Nerve Somatosensory Evoked Potentials

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The somatosensory evoked potentials (SEPs), following stimulation of both the posterior tibial nerve (PTSEP) and pudendal nerve (PNSEP), comprise of the lumbar negative, subcortical and cortical potential. These can be used to assess the long somatosensory pathway, including peripheral, intraspinal and intracranial conduction along the entire length. This study aimed to compare the central conduction time between the PTSEP and the PNSEP, and to investigate the relationship between the intraspinal and intracranial conduction time in the SEP pathway. The SEPs following stimulation of the posterior tibial nerve at the ankle and the pudendal nerve at the shaft of the penis were analyzed in 20 normal male subjects. The central conduction of the PNSEP was found to be slower than that of the PTSEP ($p < 0.05$). This difference is due to a delay in conduction rather than that of intracranial conduction.

Key words: Somatosensory evoked potential, central conduction time, intraspinal conduction time, intracranial conduction time

INTRODUCTION

The somatosensory evoked potential (SEP) has been used to distinguish the long somatosensory system from the peripheral nerve to the cortex.[1] The pathway of the SEP traverses the spinal cord, posterior column, nucleus gracilis and cuneatus, medial lemniscus, ventrobasal thalamic nuclei, thalamocortical tract, and terminates at the primary sensory cortex.[2-4] The recordings of the cortical, subcortical and spinal evoked potentials represent an objective measurement of the anatomic integrity of the somatosensory system and can serve to both document and localize the interruption or delay of neural conduction.[5] The central conduction time in the SEP is defined as the latency between the spinal potential recorded in the lumbar region and the cortical potential. Recording of the scalp far-field potential, which originates at the cervicomedullary junction, has been proven to give reliable and useful information for a separate assessment of both the intraspinal and intracranial SEP conduction.[6,7]

The somatosensory evoked potentials of the posterior tibial (PTSEP) and pudendal nerve (PNSEP) are comprised of the lumbar, subcortical and cortical potentials.[5,19] The similarity in configurations of the cortical, subcortical and lumbar potentials evoked by tibial and pudendal nerve stimulation might suggest a similar neural mechanism for producing both responses. The latency of the cortical potential evoked from pudendal nerve stimulation at the base of the penis is similar to that evoked by stimulation of the tibial nerve at the ankle, despite the different pathway lengths.[10] The latency of the lumbar potential following pudendal nerve stimulation occurs much earlier than that of the posterior tibial nerve stimulation.[10,11] The central conduction in the PNSEP is relatively slower than that in PTSEP.[11] There are no satisfactory explanations for this phenomenon at present. One explanation for this difference may be that fibers in the PTSEP

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pathway conduct rapidly, whereas those in the PMSEP pathway conduct more slowly.[10] The purpose of this study was to compare the central conduction time between the PTSEP and the PNSEP and to investigate the relationship of the intraspinal and intracranial conduction time between the PTSEP and PNSEP pathways.

MATERIALS AND METHODS

The subjects included 20 healthy males, aged 16 to 54 years (mean 30 years) with their heights ranging from 163 to 179 cm (mean 172 cm). Each subject lay supine on a bed and was instructed to relax with their eyes closed and the PTSEPs and PNSEPs were recorded. Stimulation was applied to the base of the penis with a bipolar ring electrode for the PNSEP and to the ankle with a bar electrode for the PTSEP. Stimulation consisted of square wave pulses of 0.2 msec. duration at a frequency of 3 Hz. The stimulation intensity for the PTSEP was increased to the point when a noticeable twitch could be observed in the muscle innervated by the nerve being stimulated. The stimulation intensity for the PNSEP was increased to a point just below the pain threshold or three times that of the sensory threshold. For the lumbar potential, the active electrode was placed over the spine at the twelfth thoracic vertebral level whilst a reference electrode was placed over the anterior iliac spine. For the subcortical far-field potential, an active electrode was placed over the Fpz (in the international 10-20 System) with a reference electrode over the fifth cervical vertebral level. For the cortical potentials, an active electrode was placed over the Cz' (2 cm behind Cz) with the Fpz used as a reference. The electrode impedance was maintained at below 5 kOhm. The filter band-pass was 10-1000 Hz. The SEPs were recorded with an Excel electromyograph. A total of 256-514 signals were averaged twice to ensure the consistency of the response. The latency of the lumbar potential was measured at the initial negative peak, LP. The latency of the subcortical potential was measured from the positive peak, P31. The latency of the cortical responses was measured to the peak of the first positive potential, P37. The interpeak latencies between P37 and LP (P37 - LP), between P37 and P31 (P37 - P31) and between P31 and LP (P31 - LP) were also measured. The mean latency values and interpeak latencies were compared between the PTSEP and the PNSEP using the paired t-test with $p < 0.05$ considered to be statistically significant.

RESULTS

The PTSEP and PNSEPs are shown as in Figures 1 and 2, respectively. These results suggest there is a similarity in the configuration of the cortical, subcortical and spinal potentials between the PTSEP and the PNSEP. The mean latencies and the central conduction time of the P37, P31, LP, P37-LP, P37-P31 and P31-LP are shown in the Table 1. The mean latency of the P37 in PNSEP was similar to or slightly longer (≈1 msec) than that in PTSEP. However, the mean latency of the

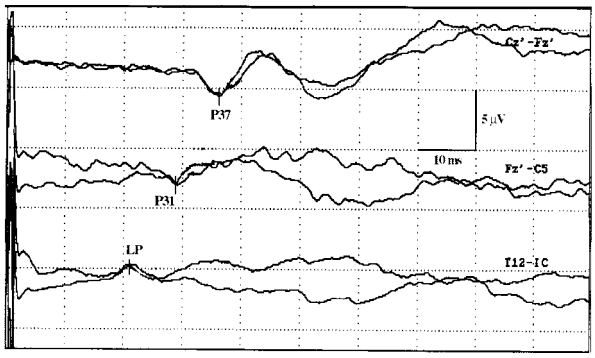

Fig. 1. Somatosensory evoked potentials following stimulation of the posterior tibial nerve; P37, cortical potential; P31, subcortical potential; LP, lumbar potential.

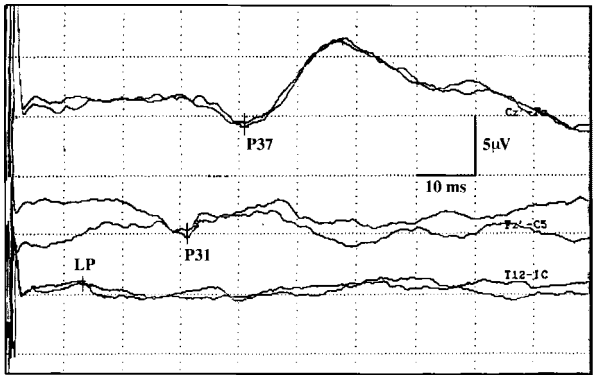

Fig. 2. Somatosensory evoked potentials following stimulation of the pudendal nerve; P37, cortical potential; P31, subcortical potential; LP, lumbar potential.

LP in the PNSEP was shorter by about 9 msec than that in the PTSEP. The mean latency of the P31 was similar in both the PNSEP and the PTSEP. There was no significant difference in the central conduction time of the P37 - P31 between the PTSEP and the PNSEP ($p > 0.05$) and in the P31 latency ($p > 0.05$), while the latency of the LP and the central conduction time of the P37-LP and the P31-LP showed a significant difference between the PTSEP and the PNSEP ($p < 0.05$).

DISCUSSION

In this investigation, there was a similar configuration in the lumbar, subcortical and cortical evoked potentials between the PTSEP and the PNSEP. The lumbar negative potential after stimulating the nerves of lower extremity and the pudendal nerve is believed to be generated within the caudal spinal cord.[8,12,13] Therefore, its latency is interpreted as the peripheral conduction time. The latency of the spinal evoked potential was approximately 12 ms after stimulation of the dorsal nerve of the penis and 21 ms after stimulation of the posterior tibial nerve at the ankle. This difference in latency could be due to either a different pathway length or a different conduction velocity. However, it is more likely to be due to a different length of the peripheral conduction pathway.

The cortical evoked potential over the scalp from the posterior tibial and pudendal nerves demonstrated a maximum response over the sensory cortex in the midline of the scalp.[14,15] This finding is consistent in that the sensory homunculus for both posterior tibial and pudendal nerve distributions are in the interhemispheric fissure.[16] The configuration and latency of the cortical evoked potentials after pudendal nerve stimulation was similar to that from the posterior tibial nerve, but with a slightly lower amplitude and a slight delay in latency. The reduced amplitude of the cortical potential in the PNSEP may be due to the deeper location of the pudenal cortical area in the interhemispheric fissure.

In this study, the latency of the cortical potential was approximately 39 - 40 ms in the PNSEP and 38-39 ms in the PTSEP. The latencies of the cortical potentials were similar and there was only a 1 ms difference between the PTSEP and the PNSEP despite the different pathway length. The central conduction time (P37 - LP) i.e. the difference between the cortical and spinal evoked potentials, was approximately 17 ms for the PTSEP and 27 ms for the PNSEP (Table 1). Therefore, the peripheral conduction time (LP) in the PNSEP was approximately 9 ms shorter than that in the PTSEP, and the central conduction time (P37 - LP) in the PNSEP was about 10 ms longer than that in the PTSEP. This suggests that the central conduction time in the PNSEP is slower than that in the PTSEP. This unexpected finding may be explained by the differences in either the routing of the pudendal afferents in the spinal cord and brainstem or both the axon spectra and the conduction velocity in the central pathway.[17]

To further clarify the difference in the central conduction time, the intracranial and intraspinal conduction time between the PTSEP and the PNSEP were compared. The significance of the far-field subcortical potential, P31 in both the PTSEP and PNSEP, as P14 of the median nerve somatosensory evoked potential, has not been

Table 1. The Mean Latencies and Mean Central Conduction Time of Posterior Tibial and Pudendal Nerve Seps

	Posterior tibial nerve SEP (n = 20) Mean ± SD	Pudendal nerve SEP (n = 20) Mean ± SD	p Values *
Spinal potenital latency (LP)	21.74 ± 1.05	12.68 ± 1.20	$p < 0.05$
Subcortical potential latency (P31)	30.77 ± 1.09	31.50 ± 2.27	NS†
Cortical potential latency (P37)	38.68 ± 1.86	39.73 ± 2.49	$p < 0.05$
Central conduction time (P37-LP)	16.91 ± 1.37	27.04 ± 2.48	$p < 0.05$
Intraspinal conduction time (P31-LP)	9.04 ± 1.11	18.80 ± 2.39	$p < 0.05$
Intracranial conduction time (P37-P31)	7.87 ± 1.45	8.22 ± 2.12	NS†

*p values computed using paired t-test. †not significant.

extensively studied.[18-20] This potential is presumed to be generated in the lower brainstem, probably before the decussation of the sensory fibers. The nucleus gracilis and medial lemniscus in the lower brainstem are probably the anatomical structures generating the potential P31.[6,7] When recorded using a noncephalic reference electrode, this potential has a wide distribution over the scalp with a slight predominance in the frontal region. Therefore, the potential, P31, has been is used to investigate the intraspinal (P31 - LP) and the intracranial conduction time (P37 - P31) separately in the somatosensory pathway.[7] In this our, the intracranial conduction time (P37 - P31) was approximately about 7 - 8 ms, which is similar for both pathways. However, there was a significant difference in intraspinal conduction time (P31 - LP), approximately 9 - 10 ms. Most of this difference occurs in the intraspinal conduction time. The delay is due to instraspinal conduction rather than in intracranial conduction. Guerit and Opsmer14 have shown that this may be explained by either the difference between the cortical and spinal generators in both the PNSEP and the PTSEP or differences in the pathway involved in spinal- cortical conduction. Although the cortical and spinal generators of both the PTSEP and the PNSEP were different, this 9 - 10 ms delay in central conduction between them could not be explained. Although there have been no animal or human studies on PNSEP pathway routing, a different conducting pathway or a different fiber population may be involved in the pathway between them. Therefore, further studies are needed to verify the various possibilities of the conducting pathway and conducting velocity in both the conducting pathway and conducting velocity in the central SEP pathway for the clinical application of the PTSEP and the PNSEP.

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