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# Clinical Application of Peripheral and Central Conduction Velocities Using Somatosensory Evoked Potentials

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**ABSTRACT.** The peripheral conduction velocity (PCV) and central conduction velocity (CCV) were measured using the cerebral and spinal somatosensory evoked potentials. Reduced PCVs and/or CCVs significantly correlated with sensory disturbances due to neuromyelopathy, neuropathy and myelopathy. PCV and CCV are useful as indicators of functional disturbances of the central and peripheral sensory pathways.

**Key words : SEP — peripheral nerve conduction velocity— —central nerve conduction velocity**

The short latency components of the cerebral somatosensory evoked potential (CSEP) have been used to detect lesions of sensory pathways throughout the peripheral and central nervous system. Especially, in some diseases such as multiple sclerosis or polyneuropathies, peak delays of the short latency CSEP have been used routinely to explore the peripheral and spinal cord lesions and to monitor the clinical course. Cortical neuronal mass activity, which may be likened to a biological amplifier, has enabled us to record clearly such previously indistinguishable potentials as dispersed nerve action potentials at the level of the peripheral nerve or the spinal cord. The short latency components are very stable, being unaffected by drowsiness, sleep, thought processes and most medications including those inducing light anesthesia.

Measurement of the peripheral sensory nerve conduction velocity using SEP was attempted by Ball et al.[1] Conduction velocity of the spinal cord was determined by Cracco,[2] Jones and Small,[3] Terao et al.[4] and Kakigi et al.[5] in normal individuals using surface recording of the spinal somatosensory evoked potential (SSEP).

By simultaneous recording of the SSEP and CSEP following tibial nerve stimulation, Delbeke et al.[6] estimated grossly the conduction velocity between the T12 spinous process and the contralateral somatosensory cortex in normal subjects. Recently, with a similar technique, Eisen and Odusote[7] measured central and peripheral conduction times in multiple sclerosis patients gaining significant results useful for diagnosis. In this newly designed study, peripheral and central conduction velocities in normal subjects and in four groups of neurological patients were examined by simultaneously recording the lumbar SSEP and parietal scalp CSEP. Though preliminary in nature, this study

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indicated the practicality of applying the methods clinically.

## SUBJECTS AND METHODS

Observations were made on 30 normal young male adults, ranging in age from 18 to 26 years (mean 20.1 years) and 23 patients with various neurological disorders. Recording procedures were based on our previously reported routine techniques[4] for recording CSEPs and SSEPs, with reference to the methods of Eisen and Odusote[7] (Fig. 1).

Subjects relaxed in a supine position with their eyes closed in a semidark, quiet and electrically shielded room with an ambient temperature between 25°C and 29°C. Bilateral simultaneous electric shocks, or unilateral stimulations on either side, were given percutaneously over the posterior tibial nerve at the ankles. Point "F" was the proximal electrode position. The stimulus was a square wave current pulse of 0.6 to 1.0 msec duration applied at a rate of 2/sec, and the strength was adjusted to 10 V above the motor threshold of the stimulated muscle so as to provide equal contraction bilaterally.

For recording SSEP, a pair of silver chloride disc electrodes was attached to the skin over the thoracolumbar spines 5 cm apart. In our previous study[4] on human SSEPs using a multi-channel bipolar surface recording technique, a phase reversal of the SSEP was noted near the lst or 2nd lumbar vertebra, just level with the lower sacral segment. The electrodes were placed rostral to this phase reversal point, the upper electrode at point "L" 3 tenth of the distance from the spinous process of the 4th lumbar vertebra to the suboccipital depression where the peripheral stimuli are thought to arrive in the spinal cord. The CSEP was recorded from a scalp needle electrode placed 2 cm posterior to the vertex (point "C"), with a reference electrode on the ear lobe.

The responses were amplified with an EEG machine filtered at 60 Hz with a time constant of 0.1 sec for CSEP and 0.001 sec for SSEP. Averaging was accomplished by summing the responses to 250 stimuli in bilateral stimulations and to 500 stimuli in unilateral stimulations with a Signal Processor 7T07A (SAN-EI). The analysis time was 100 msec. CSEPs and SSEPs were recorded at least twice in each stimulation session and were plotted graphically by an X-Y plotter.

A positive peak at 20 msec (P20) and two positive peaks at 30 and 38 msec (P30 and P38) occurred in SSEPs and CSEPs respectively, from which peak latencies, PCVs (F-L interval) and CCVs (L-C interval) were calculated as follows :

$$\text{PCV} = \frac{\text{distance F - L}}{\text{latency P20}}$$

$$\text{P30 CCV} = \frac{\text{distance L - C}}{\text{latency P30 - latency P20}}$$

$$\text{P38 CCV} = \frac{\text{distance L - C}}{\text{latency P38 - latency P20}}$$

The distances F-L and L-C were measured with the person standing straight as if taking his height. Two different CCVs (P30 CCV and P38 CCV) were determined using the two peak latencies of the CSEP.

## RESULTS

The peak latencies, PCV and CCVs of 30 normal subjects, are shown in Table 1. The PCV was 52.2±2.4 m/sec, and the P30 CCV and P38 CCV were

TABLE 1. Peak latencies, PCV and CCVs in 30 normal subjects

| | Bilat. St. | L. Tib. N. St. | R. Tib. N. St. |
|---|---|---|---|
| $P_{20}$ (msec) | 19.9 ±1.1 | 19.8 ±1.2 | 19.7 ±1.1 |
| $P_{30}$ (msec) | 30.0 ±1.3 | 30.3 ±1.4 | 30.1 ±1.2 |
| $P_{38}$ (msec) | 38.2 ±1.7 | 38.3 ±1.6 | 38.2 ±1.8 |
| PCV (m/sec) | 52.2 ±2.4 | 52.1 ±2.2 | 52.6 ±2.7 |
| CCV $P_{30}$–$P_{20}$ (m/sec) | 55.8 ±4.5 | 54.5 ±5.1 | 54.3 ±4.6 |
| CCV $P_{38}$–$P_{20}$ (m/sec) | 30.9 ±2.0 | 30.6 ±1.9 | 30.7 ±2.3 |

55.8±4.5 m/sec and 30.9±2.0 m/sec, respectively. There were no differences in the peak latencies between bilateral and unilateral stimulations, and the peaks were noted in all cases, though they were more conspicuous with the bilateral simultaneous stimulation. The SSEP and CSEP of a normal 19-year-old male are shown in Fig. 1. In the normal control group, the mean of F–L was 103.2 cm, of L–C 56.4 cm and height 170.1 cm.

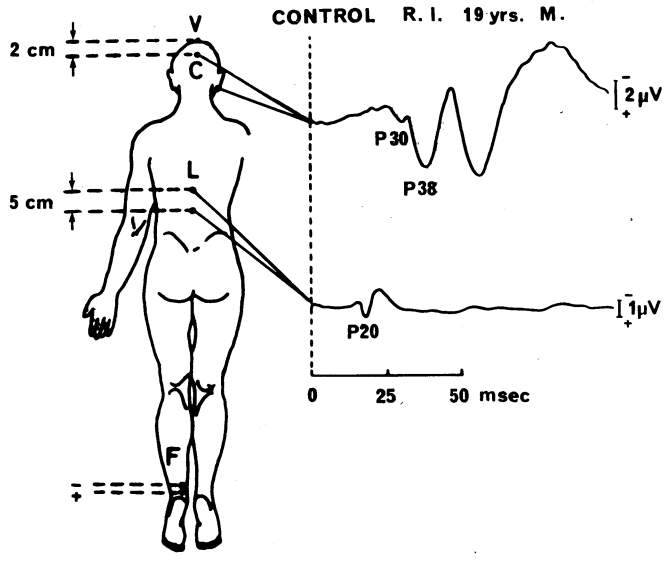


Fig. 1. Diagram showing the method of recording CSEP and SSEP following tibial nerve stimulation. "L" is the point 3 tenths of the distance from the 4th lumbar vertebra to the suboccipital depression, "F" the point over the posterior tibial nerve, and "V" the vertex.

Representative cases of the patient group are reported below.

Case 1 (Fig. 2) : A 52-year-old female with diabetic polyneuropathy. She had paresthesia and sensory disturbances in the distal part of all four limbs. The peak latencies of P20, P30 and P38 were all within normal limits (±2SD). However, the height of the patient was 154.3 cm and the distance F - L was 92.3 cm, the latter being shorter than the control group mean by 10.9 cm. A low PCV of 44.0 m/sec was calculated, though P30 and P38 CCV were within normal limits.

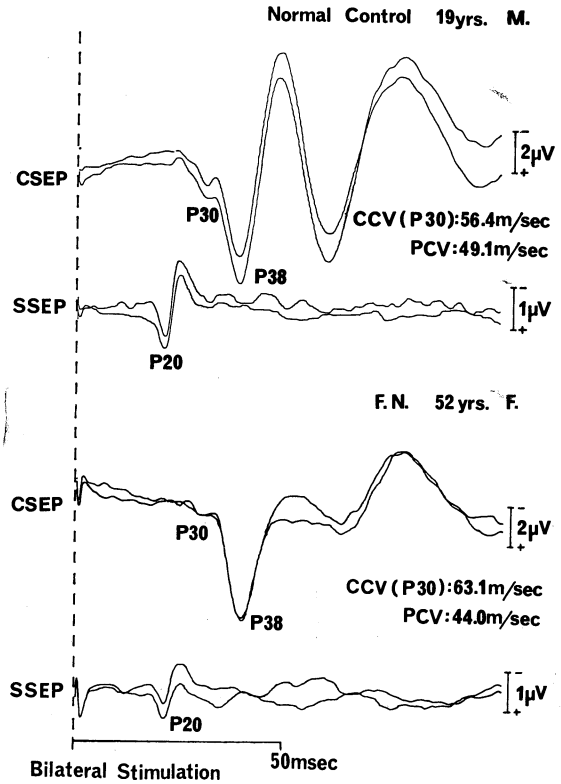


Fig. 2. Simultaneous CSEP and SSEP recording of a 52-year-old female with diabetic polyneuropathy.

Case 2 (Fig. 3) : An 18-year-old male with multiple sclerosis (Devic type). He had paraparesis with sensory disturbances below the high thoracic level. P20 was 20.7 msec and the PCV was 58.8 m/sec, both within normal limits, P30, however, was unidentifiable, and P38 was quite delayed at 44.5 msec and, as were the succeeding peaks, was deformed with a low amplitude. P38 CCV was consistently low at 22.2 m/sec.

Case 3 (Fig. 4) : A 48-year-old male with cervical spondylotic myelopathy and polyneuropathy. He had spastic paraparesis in addition to paresthesia and sensory disturbances in the distal parts of all four limbs. The peak latencies of P20, P30 and P38 were all prolonged; 22.6 msec, 38.0 msec and

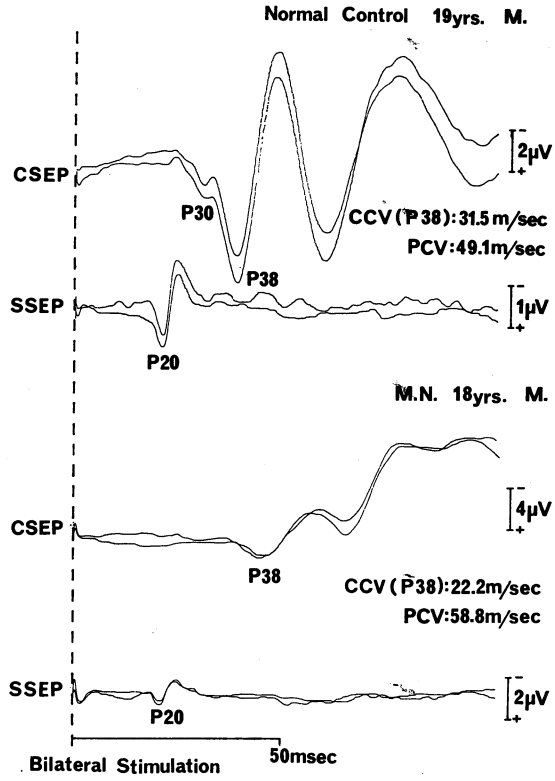


Fig. 3. Simultaneous CSEP and SSEP recording of an 18-year-old male with the Devic type of MS.

45.3 msec, respectively. Both PCV and P30 CCV were decreased; 45.9 m/sec and 37.7 m/sec, respectively.

In addition, twenty-three patients were classified into 4 groups : neuropathy, myelopathy, neuro-myelopathy and others. The P20, P30 and P38 latencies, and PCV, P30 and P38 CCV in each group were compared statistically with those in the normal control group (Fig.5).

a) Neuropathy group

This group included 8 patients with polyneuropathies. The peak latency of P20 was delayed but showed no significant difference. P30 and P38 were prolonged ($p<0.05$), and PCV and P38 CCV were low ($p<0.02$ and $p<0.05$).

b) Myelopathy group

This group included 4 patients with myelopathies. The peak latencies of P20 and P30 showed no significant differences, but P38 was prolonged ($p<0.05$). PCV was not significantly different; P30 and P38 CCV were low ($p<0.05$). A patient with MS was excluded because the CSEP wave pattern was unclear.

c) Neuro-myelopathy group

This group included 4 patients with myelopathies in addition to polyneuropathies. The peak latencies of P20 and P30 were delayed but showed

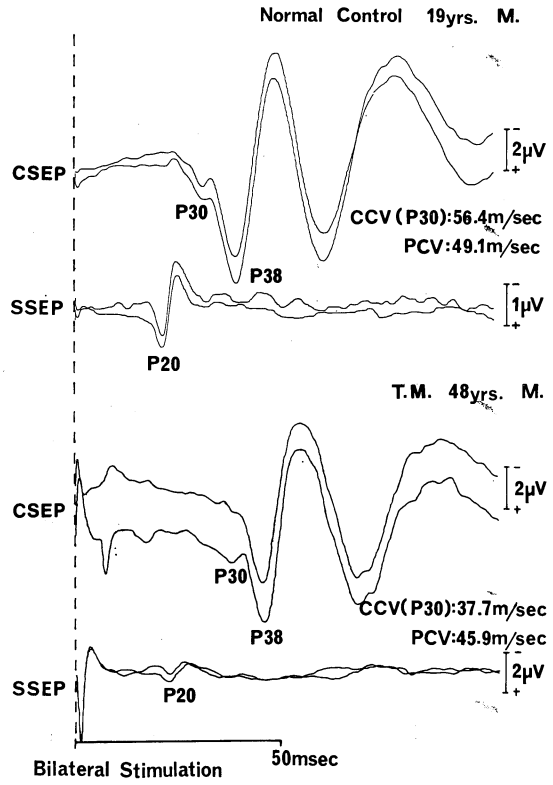


Fig. 4. Simultaneous CSEP and SSEP recording of a 48-year-old male with cervical spondylotic myelopathy and polyneuropathy.

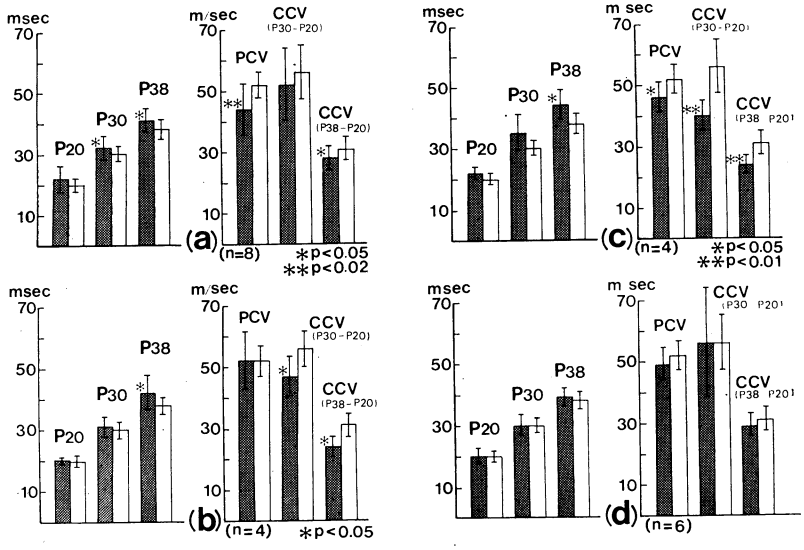


Fig. 5. Statistical comparison of peak latencies, PCV and CCVs between normal subjects (□) and the four patient groups (▨) ; a) neuropathy, b) myelopathy, c) neuromyelopathy, and d) others. Bars indicate the mean ±2SD.

no significant differences, P38 was prolonged ($p<0.05$), and PCV, P38 and P38 CCV all were low ($p<0.05$ and $p<0.01$).

d) Others

This group included 6 miscellaneous cases including motor neuron diseases, Parkinson disease, cerebellar ataxia and myotonia congenita. In this group, no significant differences among the indicators were observed.

## DISCUSSION

In the present study, PCV reflects the sensory conduction velocity (SCV) along the whole length of the peripheral nerve. The clinical usefulness of the PCV may be greater than the conventional measurement, SCV, which is restricted to the distal parts of the peripheral nerves. In our previous study,[4] the PCV was found to be 57.5 ± 2.5 m/sec, and in this study a slower 52.2 ± 2.4 m/sec. The discrepancy may be explained by the difference in the way the distance between points F and L was measured; in the previous study, F – L was measured following the contour of the body, but in this study as a straight line between the two points, resulting in a shorter length. Using a needle electrode, Buchthal and Rosenfalck[8] reported that the sciatic nerve SCV between the popliteal fossa and buttock was 56.8 ± 1.1 m/sec which accords better with our previous result.

Cracco et al.[2] described a precise method for recording SSEP using surface electrodes. In a previous study the present authors[4] adopted a multi-channel surface SSEP recording to bilateral simultaneous tibial nerve stimulation. The measurement of the conduction velocity of the spinal cord was highly favorable because the distribution of the SSEP, which progressively increased in latency at more rostral recording locations, could be recognized more easily. The triphasic waves recorded on the lower lumbar spines may correspond with the lumbar nerve root potentials (LNRP) named by Liberson et al.,[9] and the ascending responses rostral to the triphasic waves may represent the evoke potential travelling over the spine. From the latency difference in the ascending initial positive peaks, the spinal conduction velocity between the sacral and upper cervical segment was determined to be 70.1 ± 8.4 m/sec.

Recently, Kakigi et al.[5] reported short latency SSEPs and CSEPs following posterior tibial nerve stimulation. They stated that bipolar derivation is advantageous to monopolar derivation since there is much less interference by artefacts. However, the response recorded by bipolar derivation is obviously a potential difference between two active electrodes, and, therefore, they concluded that monopolar derivation is ideal when one is interested in rapidly propagating responses such as the SSEP. In their study, the mean conduction velocity of the spinal cord was 70.5 ± 11.2 m/sec when it was calculated using the onset latency of the initial negative peak, and 73.5±7.8 m/sec when using the peak latency. The former is very similar to values determined in our previous study.[4]

Phillips and Daube[10] studied precisely the lumbosacral SSEP responses to peroneal and tibial nerve stimulation using surface electrodes over the spinous processes S1 to T11 with monopolar and bipolar derivations. Bipolar recording showed a phase reversal of the cord peak with either peroneal or tibial nerve

stimulation, which is in accord with our previous report[4] on the same phenomenon. In their study, two well defined negative peaks were demonstrated. The first one was explained as a travelling depolarizing wave in afferent fibers of the cauda equina, and the second as a volume-conducted response arising from the spinal cord. They noted that bipolar sequential electrode linkages and averaging of many responses was needed to follow the waves travelling up the spinal cord.

In previous reports, the spinal conduction velocity was found to be approximately 65 m/sec,[2] 60-65 m/sec,[3] 70.1 m/sec[4] and 70.5 m/sec.[5] Ertekin[11,12] measured the propagation velocity of the volleys along the dorsal funiculus of the human spinal cord using an intrathecal recording technique. If two intrathecal electrodes were situated behind the cord dorsum at the lower cervical and lower thoracic levels respectively, almost the same type of potentials were evoked to both rostral and caudal stimulations. The conduction velocity in both rostral and caudal directin, averaged about 45 to 47 m/sec. He noted that the conduction velocity was often above 60 m/sec if lateral recording and stimulating was performed. He suggested that the increased speed may have been due to the stimuli passing through the neural structures adjacent to the cord dorsum. It then becomes controversial whether or not the spinal conduction velocity measured by the interpeak latency of the wave travelling over the spine reflects directly the conduction velocity of the cord dorsum. However, the speed which is calculated is assumed to be the maximal spinal conduction velocity.

The evaluation of spinal cord lesions using the spinal conduction velocity is a fascinating possibillity. In our previous study,[13] however, the velocity could not be measured in patients with spinal cord lesions, because the appearance of SSEP peaks were greatly restricted over the spine, especially at the thoracocervical level. The authors, therefore, planned the simultaneous recording of the CSEP and SSEP after the method of Eisen and Odusote.[7] As already mentioned, two positive peaks, P30 and P38, on the scalp were used as indicators to measure CCV. P30 has not been noticed very often, though it was described in a few reports.[7] Using a noncephalic reference, Kakigi et al.[5] found 4 components, P25; N27, P28 and N31, preceding the major positive peak, P40 (P38 in this study), in response to posterior tibial nerve stimulation at the ankle. The 4 components were all considered to be generated in the deep structure. P30 in this study may correspond with P28 in the report by Kakigi et al.[5] P40 has been observed distinctly with conventional recording techniques. Tsumoto et al.[14] analysed CSEP to lateral popliteal nerve stimulation, and that the primary sensory area of the lower limb was responsible for this P40 component that was generated from the focus lying posterior to the rolandic sulcus. That P40 was distributed to some extent ipsilateral to the stimulation side was unexpected. The same phenomenon, however, was observed by us in our previous study[15] with scalp topographic mapping of the CSEP. Cruse et al.[16] also noted the paradoxical lateralization of the CSEP evoked by stimulation of the posterior tibial nerve.

From the peak latencies (P20, P38 and P38) and peak to peak distances (F - L and L - C), the authors determined two different CCVs, P30 CCV which was 55.8 $\pm$ 4.5 m/sec and P38 CCV which was 30.9 $\pm$ 2.0 m/sec. In

almost all previous reports, central conduction time has been calculated from inter-peak latency without referring to conduction velocity, which is reasonable because of the difficulty of measuring accurately the length of the sensory pathway under the skin and of the ambiguity of where the generation site for each peak is. However, the peak latency of CSEP varies significantly according to age, body height or length of the lower limbs, and among races and individuals, so that the conduction time may not be an adequate indicator of deteriorated conduction. It is for this reason that the authors decided the calculation of the conduction velocity may be more useful clinically.

As mentioned above, P30 is considered to be generated in the deep structure and to be a far-field potential, but its source remains to be discovered. In the present study, P30 was observed in all normal controls, but Eisen and Odusote[7] reported that the P30 component following tibial nerve stimulation could not be recognized with confidence in 40% of the recordings of normal subjects. In pathological cases, P30 is often difficult to detect as shown in Case 2 (Fig. 3), in which the CCV of 22.2 m/sec was calculated using P38 instead of P30. P30 CCV in normal control was 55.8 m/sec in our study, and Delbeke et al.[6] calculated a CCV of ca. 60 m/sec between the SSEP peak on the Th12 spinous process and CSEP peak considered to be P30. It can be assumed that errors were introduced naturally when we consider that the distance L - C measured as a straight line is shorter than the actual nerve pathway, and that the origin P30 is subcortical.

P38 was conspicuous enough to be identified even in pathological cases, and, thus, its clinical significance may be great. P38 CCV was 30.9 m/sec in normal controls, much slower than the spinal coduction velocity of 45-47 m/sec determined by Ertekin.[11,12] The slower velocity can be explained, through inadequately, by differences in methods and conducting fibers. P38 has been considered to be an initial cortical event generated in the foot sensory area. However, it is widely accepted that the initial cortical potential following the stimulation of the median nerve is the negative one, N20 (N18 in our previous report[17]). Recently, Berić and Prevec[18] recorded an early negative potential (N37) following stimulation of the posterior tibial nerve, but only from the contralateral hand sensory area against the reference Fz, and they proposed that negative potential to be an initial cortical response.

Though there are a few problems to be overcome, the measurement of PCV and CCV was shown to be valuable in the diagnosis of peripheral and central neuronal lesions. This study is the first step toward applying these conduction velocities clinically, and we hope to develop a more refined procedure in the near future.

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