Short Latency Somatosensory Evoked Potentials in a Patient with a Cervical Neurinoma

—Case Report—

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Abstract

A 34-year-old male with a right C4 root neurinoma was operated on and the tumor was totally removed. Short latency somatosensory evoked potentials (SL-SEPs) recorded before operation revealed only P₁ (P₉) and N₁ potentials. SL-SEPs were sequentially recorded and compared with changing symptoms and signs in an attempt to identify the primary generator of P₂ (P₁₁) potential. The recovery of SL-SEPs largely corresponded to recovery of vibration and position sensations; P₂ and following potentials appeared with improvement of these deep sensations. These findings suggest that P₂ potential may originate in the dorsal column or in a site rostral to the tumor.

Key words: spinal root neurinoma, evoked potentials, sensory disturbance, SL-SEPs

Introduction

Goff *et al.*⁹⁾ first recorded a positive potential with a peak latency of 13 to 17 msec following stimulation of the median nerve with a cephalic reference. Two additional positive potentials preceding this potential were first recorded by Cracco^{5,6)} with a noncephalic reference.

After Cracco reported his finding of short latency somatosensory evoked potentials (SL-SEPs) in response to median nerve stimulation in man, many investigators^{1,8,12-16,19)} attempted to locate the primary generators of these potentials. Most of these studies employed normal subjects, and the origins of these potentials are still a matter of controversy. Only a few investigators^{2,4,17)} have recorded SL-SEPs in patients with well localized lesions, and their reports are brief. Study of the recovery of SL-SEPs in the

context of clinical signs in patients with well localized lesions is necessary, not only to identify the primary generators but also to determine the usefulness of SL-SEPs in clinical practice.

For this purpose we sequentially recorded SL-SEPs in a patient with a neurinoma of the right C4 dorsal root and studied the relationship between SL-SEPs and the clinical findings.

Case Report

A 34-year-old male noticed sensory and motor disturbances of the right upper extremity in February, 1982. The disturbances had extended to his right lower extremity by September of that year. By March of 1983, his left upper and lower extremities were also involved.

On admission to our hospital on April 14, 1983, he

Received January 14, 1985; Accepted August 19, 1985

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Fig. 1 Metrizamide CT myelography at the C4 level. Note the dumbbell-shaped mass enlarging the right intervertebral foramen and compressing the spinal cord.

had sensory disturbances below the C4 level; deep sensations (position and vibration) were markedly disturbed compared to superficial sensations. Sensation in the sacral region was spared. Mild motor weakness in the right upper and lower extremities were observed. Bilateral patellar tendon reflexes were increased and clonus was elicited in both ankles.

Computerized tomographic myelography revealed a dumbbell-shaped mass lesion at the C3–C4 level (Fig. 1). On May 25, 1983, a neurinoma arising from

Fig. 2 Photograph taken during surgery (left) and corresponding diagram (right) show that this tumor was compressing the spinal cord ventromedially.

Fig. 3 Pre- and postoperative course (signs and symptoms). Note the prolonged recovery of position and vibration sensations on the right side.

the right C4 root and compressing the spinal cord ventromedially was totally removed (Fig. 2). The patient's clinical course is illustrated in Fig. 3. The motor weakness in the right upper and lower extremities gradually disappeared over the 2 weeks following surgery. During that period, the decreased superficial pain and touch sensations also improved. The lowered position and vibration sensations on the left side gradually improved to normal within 3 weeks after surgery; those on the right side improved somewhat more slowly. However, restoration of position and vibration sensations in the right hand and foot took 4 months after operation, and the thumb and fingers recovered slightly faster than the toes.

Recording procedure and findings of SL-SEPs: SL-SEPs were recorded on five occasions while the patient was hospitalized and twice thereafter. At each recording the left and right median nerves were independently stimulated at the wrist, *via* an isolation unit (Nihon Koden), with a 0.3 msec square pulse wave at a rate of 3.7 pulses per second. The stimulus intensity was just enough to produce a twitch of the thumb. The recording electrode was placed on the scalp at Cz point (international 10–20 system) and the noncephalic reference electrode on the left knee.

The input from the recording electrode was amplified by a differential preamplifier with a band width of 100 Hz to 1 kHz, and 1,000 samples were summated by a Sanei 7T08 signal processor over an analysis time of 51.2 msec. The summated responses were superimposed to ensure reproducibility and were recorded by a X-Y plotter.

The SL-SEPs were classified and labeled as follows. The four positive potentials were P₁, P₂, P₃, and P₄ and the three negative ones were N₁, N₂, and N₃. Normal peak latencies for these potentials were obtained from 23 healthy subjects and are listed in

Fig. 4 Classification of potentials and their peak latencies. P_1 : 7.9 ± 0.5 msec, N_1 : 9.6 ± 0.5 msec, P_2 : 10.4 ± 0.6 msec, N_2 : 11.4 ± 0.6 msec, P_3 : 12.8 ± 0.8 msec, N_3 : 15.7 ± 0.8 msec, P_4 : 19.1 ± 1.1 msec.

Fig. 4. Our P_1 , P_2 , and P_3 approximately correspond to P_9 , P_{11} , and P_{13} in Anziska and Cracco's study.^{2,3)}

The first SL-SEPs were recorded on May 10, about 2 weeks before surgery, and on both sides only P_1 and N_1 potentials with prolonged latencies were identified (Table 1 and Fig. 5). On the fifth recording (June 17), potentials appearing after N_1 and having quite prolonged peak latencies were first recognized on the left side. Position and vibration sensations on the left side had already improved by this time.

The right position and vibration sensations had reached the normal range by September 5, when potentials following N_1 appeared on the right side. All peak latencies had approached normal ranges on the left side, but not on the right. The last recording, on October 21, showed that peak latencies for all potentials on both sides were within normal ranges, with the exception of P_3 .

Discussion

Since Cracco^{5,6)} first recorded SL-SEPs in man, using a noncephalic reference, many researchers^{1,8,12-16,19)} have attempted to ascertain the primary generators of these potentials. Their results suggest that SL-SEPs are generated primarily by sources within the dorsal column medial lemniscal system. This can be theoretically determined by calculating the conduction velocity of the stimulated nerve and spinal cord.^{7,10)}

In our patient, position and vibration sensations were more severely disturbed and took a longer time to improve after surgery than did motor function and superficial sensations. Sequential recordings of SL-SEPs in this patient revealed that the recovery of SL-SEPs paralleled the improvement in vibration and position sensations but not that of motor function and superficial sensations. Clinical improvement in vibration and position sensations was observed prior to the recovery of peak latencies of the respective potentials. These findings indicate that SL-SEPs are more closely related to deep sensation, which is conveyed in the dorsal column medial lemniscal system. We postulate that the tumor compressing the spinal cord at the C4 level impaired the dorsal column more than other structures, such as the lateral corticospinal tract and the spinothalamic system.

During the period in which deep sensation was severely compromised, only P_1 and N_1 potentials were recorded in the SL-SEPs. Our P_1 component is analogous to the P_9 potential, which is widely considered to originate from the brachial plexus or peripheral nerve. On the other hand, our P_2 potential

Table 1 Peak latencies of SL-SEPs in our patient

Date	Stimulation side	P_1 (7.9 ± 0.5)	N_1 (9.6 ± 0.5)	P_2 (10.4 ± 0.6)	N_2 (11.4 ± 0.6)	P_3 (12.8 ± 0.8)	N_3 (15.7 ± 0.8)	P_4 (19.1 ± 1.1)
May 10, '83	R	8.8	10.4	—	—	—	—	—
	L	9.0	10.6	—	—	—	—	—
May 26	R	8.6	10.6	—	—	—	—	—
	L	8.8	10.6	—	—	—	—	—
May 31	R	9.0	10.8	—	—	—	—	—
	L	9.2	10.8	—	—	—	—	—
June 4	R	8.6	10.4	—	—	—	—	—
	L	8.6	10.0	—	—	—	—	—
June 17	R	9.2	10.6	—	—	—	—	—
	L	9.4	10.8	12.4	13.6	15.2	17.8	21.2
September 5	R	9.0	11.4	12.4	13.6	15.8	19.0	21.8
	L	9.0	10.6	11.6	13.0	15.0	17.6	20.0
October 21	R	8.8	9.5	10.5	11.4	15.0	17.0	20.6
	L	8.8	10.0	10.6	11.6	14.2	16.8	20.6

Fig. 5 left: Recording of SL-SEPs. right: Peak SL-SEP latencies. Shaded zones indicate the range of standard deviation in normal subjects. Note that all components of SL-SEPs on the patient's left side were first recorded on June 17 and those on the right side on September 5.

corresponds to the P_{11} potential,^{2,3)} which may be primarily generated from the root entry zone¹⁹⁾ or the dorsal column.⁴⁻⁷⁾ In our case, removal of the tumor was followed by clinical improvement in deep sensation and the appearance of P_2 and other later potentials. This suggests that P_2 potentials may originate in the dorsal column or its nuclear structures. Another possibility is that excision of the tumor restored axonal conduction to the primary generator of P_2 , which is located rostral to the tumor.

We reviewed the literature on SL-SEPs in patients with cervical lesions and found that only two other cases have so far been reported.^{11,18)} These cases involved a dislocation fracture of the odontoid process and an intramedullary lesion. In these patients the P_2 , or P_{11} , potential was not detected; however, neither the precise location of the lesions nor the clinical signs were clearly described. We feel that

more reports on SL-SEPs in patients with well localized lesions and well documented clinical data are necessary to identify the primary generators and to define the clinical role of SL-SEPs.

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