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THE SCALP TOPOGRAPHIC MAPPING OF HUMAN SOMATOSENSORY EVOKED POTENTIALS

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Abstract

The scalp topographic mappings of somatosensory evoked potential (SEP) to the median and posterior tibial nerve stimulation were recorded from 16 recording electrode locations in 23 normal subjects with MULTI-PURPOSE BIOPHYSICAL DATA PROCESSOR, TOPOGRAPHY-SYSTEM 500 (SAN-EI).

Po (P14), the earliest SEP component to the median nerve stimulation was distributed diffusely with frontal dominancy on the scalp. N1, P1, P2, P3 and N4 were best seen in the contralateral parietal area to the stimulated nerve. N2 and N3 were observed dominantly in the frontal area without significant laterality. The last component, P4 within 200 msec of analysing time had a highly maximum amplitude in the fronto-central region at midline.

Pl (P37) component to the posterior tibial nerve stimulation was best seen in the ipsilateral parieto-occipital area to the stimulated nerve in all subjects. The subsequent peaks were observed in the parietal area maximally at midline.

The topographic SEP mappings revealed a dynamic and systematic evolution on the scalp in normal individuals, and the diagnostic procedures may expand the clinical utility, especially as regards functional lesions.

INTRODUCTION

The clinical utilization of somatosensory evoked potential (SEP) to assist in the diagnosis of various neurological diseases has increased. Nevertheless, the clinical analysis of SEP is still performed on the limited region of the hemisphere. For instance, most reports of SEP abnormalities in patients with cerebral lesions have described the SEP changes on the contralateral parietal

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area to the stimulated peripheral nerves.

From our previous study[1] and the review of the papers concerning scalp distribution of SEP in man[2–4], it is clear that SEP distributes diffusely on the scalp and affords an important spatiotemporal information of cerebral function. However, a large amount of data containing multi-channel recordings have placed limits on manual approaches in our studies. Recently, brain electrical activity mapping has been introduced as a method for extending clinical utility of EEG and evoked potential data[5]. This report describes the methods and results of our study on the scalp topographic mapping of normal adult SEP.

MATERIALS AND METHODS

Twenty-three subjects ranged in age from 19 to 28 years. They were in good health and without history of neurological disease, and were all of right handed. Recording was carried out in an electrically-shielded chamber. The EEG was continuously monitored to minimize muscle activity, drowsiness, eyeblinks, etc.

In the first 13 subjects, seven males and six females, electric shock was given percutaneously over the median nerve at the wrist, and in the second 10 subjects, six males and four females, it was given over the posterior tibial nerve at the ankle. Stimulation was given usually on the right side, and in some cases

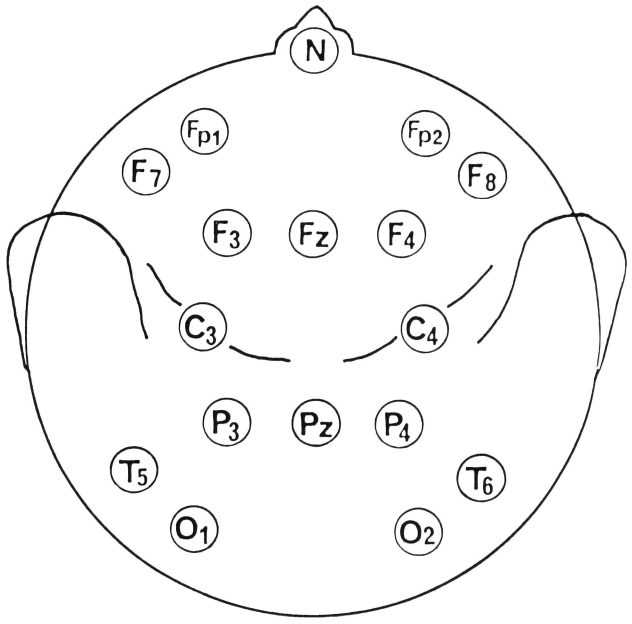

Fig. 1. Electrodes placements over the scalp.

it was also given on the left side successively. The duration of the stimulus was 0.2 msec in the first group, 0.6 msec in the second one respectively. The stimulus strength was adjusted 10 V above the motor threshold of the stimulated muscle. The inter-stimulus interval was randomly varied from 1 to 4.5 sec.

K-M type (pad) recording electrodes were used on the scalp. Recordings were obtained simultaneously from 16 electrode locations of the scalp placements of the 10-20 system as in Fig. 1. Recording was referential to the right or left earlobe, contralateral to the stimulated side. Input from the recording electrodes was led to conventional EEG amplifires. The entire system had a time constant of 0.1 sec and a filter of 60 Hz. The output was summated and then the SEP topographic mappings were displayed as color televison images by means of a MULTI-PURPOSE BIOPHYSICAL DATA PROCESSOR, TOPOGRAPHY-SYSTEM 500, SAN-EI INSTRUMENT Co. LTD.

The computer was triggered by the stimulator and routinely 250 responses were summated. In parallel with the procedure, SEPs were plotted graphically on millimeter paper by a X-Y plotter. The baseline was estimated and set manually as an average of the activity occurring the first 10 msec after stimulus following Goff et al.[4] (Fig. 2).

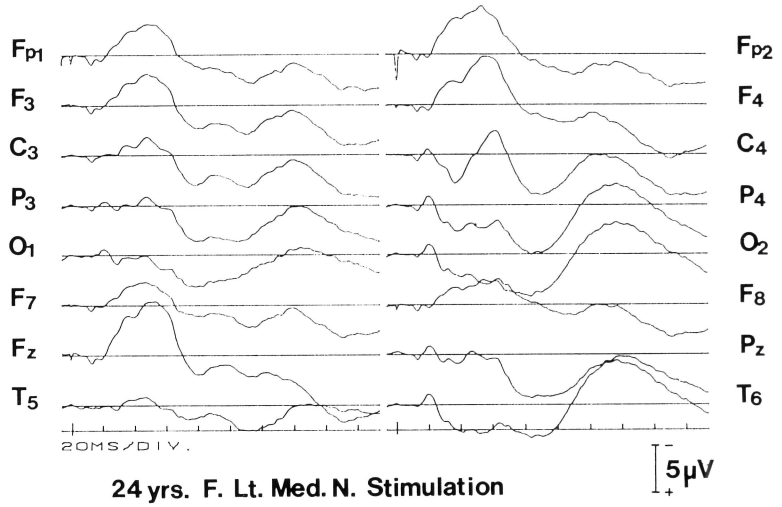

Fig. 2. Examples of 16 scalp SEPs ; A 24-year-old normal adult Female. The baseline was estimated and set manually as an average of the activity occurring the first 10 msec after stimulus. The topogrpahic mappings in this case are shown in Fig. 4.

RESULTS

In the median nerve stimulation, the response within 200 msec has five

TABLE 1

Mean values and standard errors for latencies and appearance rate of each component of parietal SEP to the median (A) and tibial (C) nerve bilateral simultaneous stimulation in normal subjects.

Wave	Latency (msec)		Appearance (%)	
	Rt. Hemisphere (Hs)	Lt. Hs	Rt. Hs	Lt. Hs
A				
P_0	13.3± 0.8	13.3± 0.9	100.0	100.0
N_1	18.2± 1.3	18.1± 1.1	100.0	100.0
P_1	24.0± 2.6	24.0± 2.5	93.3	93.3
N_2	32.3± 2.7	32.0± 2.5	90.0	93.3
P_2	42.6± 3.2	43.0± 3.0	100.0	100.0
N_3	61.0± 6.7	61.2± 7.7	93.3	96.7
P_3	96.2± 9.5	96.4± 8.6	90.0	90.0
N_4	126.3± 8.0	126.8± 7.7	86.7	83.3
P_4	163.6±13.3	162.8±13.8	50.0	50.0
C				
P_1	37.6± 2.1	37.4± 2.1	100.0	100.0
N_1	47.6± 2.2	47.5± 2.2	100.0	100.0
P_2	59.2± 2.6	59.1± 2.7	100.0	100.0
N_2	74.3± 4.0	74.2± 4.1	100.0	100.0
P_3	95.3± 6.3	95.1± 6.2	76.7	76.7
N_3	111.8±11.0	111.8±11.1	50.0	50.0
P_4	123.0± 5.1	123.0± 4.9	33.3	33.3
N_4	142.3± 4.6	141.6± 4.5	26.7	26.7

positive peaks and four negative ones beginning with a small positivity (Po) with the latency of about 14 msec, and in the posterior tibial nerve stimulation, the response within 200 msec has four positive peaks and four negative ones beginning with a positivity (P1) with the latency of about 37 msec[6] (Table 1). These peak latencies were used as indicators to identify each peak in this study.

Then, serial topographic mappings at each parietal (C3 and C4 to the right and left median nerve stimulation and Pz to the posterior tibial nerve stimulation) SEP peak latency were visualized on TV screen. The mappings were all photographed on the color films, and red repesented positive and blue represented negative activity with respect to the ear reference. The topographic mappings were presented with grading of ten color tints from blue (−5) to green (−1) and from yellow (+1) to red (+5) respectively (Fig. 3).

Representative topographic maps are shown for the median nerve stimulation in Fig. 4. and for the posterior tibial nerve stimulation in Fig. 5. In the median nerve stimulation, Po (P14), the earliest SEP component was small and was distributed diffusely with frontal dominancy on the scalp. N1, P1,

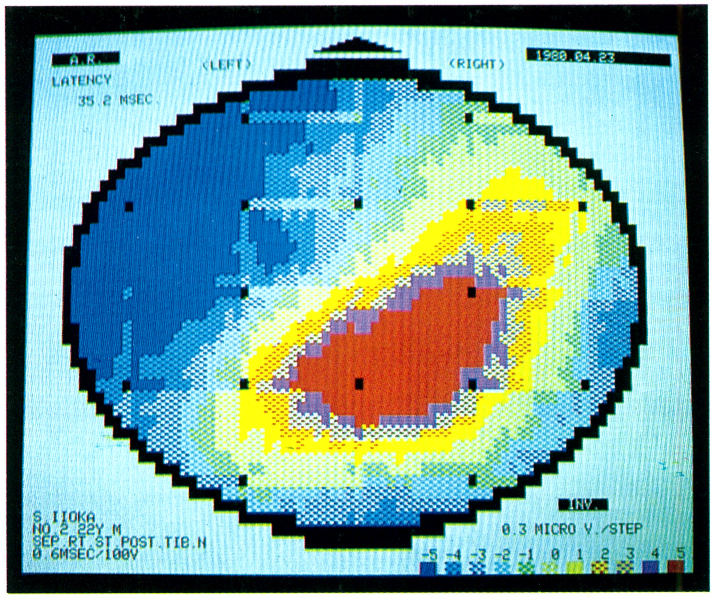

Fig. 3. A topographic SEP mapping to the right posterior tibial nerve stimulation at the parietal (Pz) P1 peak latency of 35.2 msec ; A 22-year-old normal adult male. The mapping is presented with grading of 10 color tints from blue to green (negative activity) and from yellow to red (positive activity).

P2, P3 and N4 were best seen in the contralateral parietal area to the stimulated nerve. On the other hand, N2 and N3 were observed dominantly in the frontal area without significant laterality. The last component P4 had a highly maximum amplitude in the fronto-central region at midline.

In the posterior tibial nerve stimulation, P1 (P37) component was best seen in the ipsilateral parieto-occipital area to the stimulated nerve in all subjects. The other successive peaks were observed in the parietal area maximally at midline, however, some of them were difficult to identify.

DISCUSSION

Since Dawson[7] introduced averaging techniques by recording the somatosensory evoked potential from the human scalp, a considerable number of studies have been reported by many workers. As yet, there is no established theory about the origin of each component. Early workers suggested that the early components of SEP were all generated in thalamocortical axons[2,8,9]. However, more recent observations showed that these components are distributed widely over the scalp[1–4,10,11] suggesting the complex nature of the neural

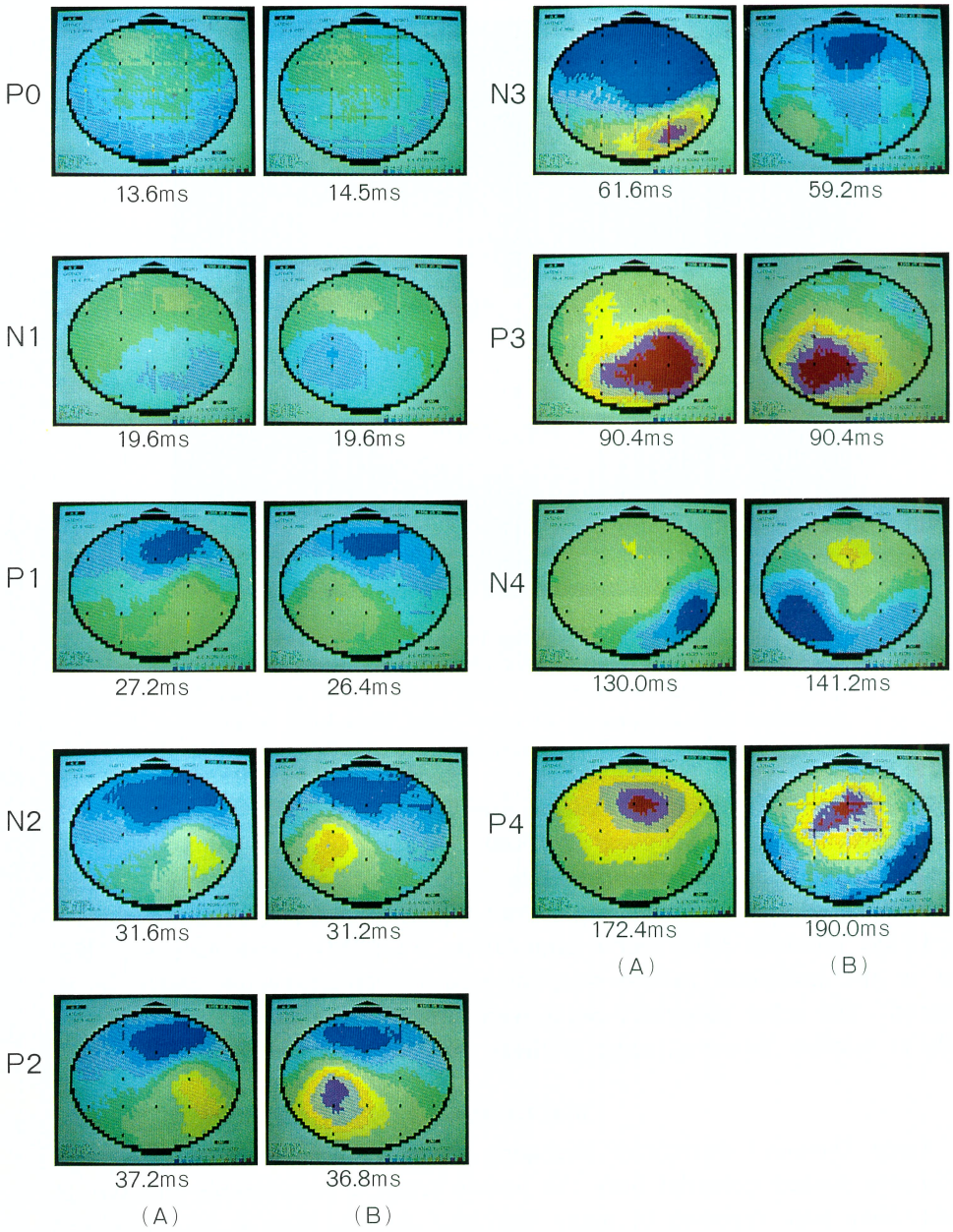

Fig. 4. Example of a series of topographic SEP mapping at each parietal (C4 and C3) peak latency to the left (A) and right (B) median nerve stimulation; A 24-year-old normal adult female. N1, P1, P2, P3 and N4 are best seen in the contralateral parietal area to the stimulated nerve. N2 and N3 are observed dominantly in the frontal area without significant laterality. P4 has a highly maximum amplitude in the fronto-central area at midline. Each peak latency is shown in msec (ms) under the mapping.

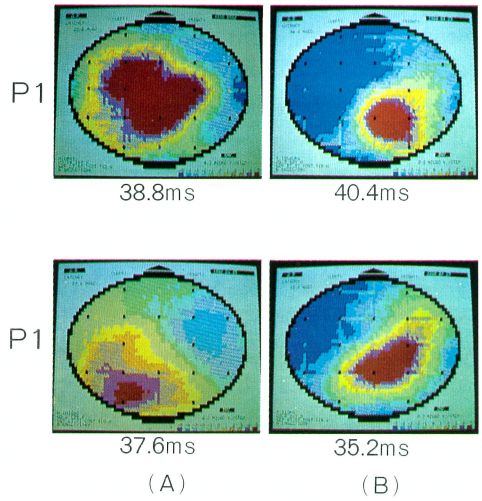

Fig. 5. Topographic SEP mappings at the parietal (Pz) P1 peak latencies to the left (A) and right (B) posterior tibial nerve stimulation in four normal subjects. Note the appearance of positive potential maxima in the ipsilateral parieto-occipital area to the stimulated nerve in all of them.

substrate which underlies the SEP in man.

The P14 potential has not been observed constantly, but using ear reference recording, it appears steadily over the scalp. It is distributed widely over the scalp. The area of its potential maxima has not clearly defined. As the latency of this potential was uniformly the same at all recording locations, its subcortical origin has been believed by several authors[3,8,12,13]. Goff et al.[12] suggested that the potential was generated in thalamo-cortical axons. The ascending thalamo-cortical radiation volley or a ventrobasal thalamic potential was also a likely candidate[8]. Allison et al.[13] stated that it represented the VPL response and the ensuing thalamo-cortical volley. Nakanishi et al.[14] observed that in patients with lesions at or above the thalamus, only the P14 potential was of normal configuration and latency in spite of the abscence or profound alterations of the subsequent responses to stimulation of the affected side, and that in patients with lesions in the brain stem or in the cervical cord it could not be obtained to stimulation of the affected side. They concluded that the P14 potential might be the result of activity of the medial leminiscal system from the medulla to the thalamus. Greenberg et al.[15] showed that it could be consistently recorded even in patients who were areflexic and who had electroencephalograms with minimal or no cerebrally originating electrical activity. They believed that P14 potential recorded from parietal scalp was the afferent volley of depolarization conducted in the leminiscal system.

Therefore, it has been postulated that the P14 potential originates from the brain stem and is recorded widely and synchronously over the scalp as volume conduction or far field potential. Then, the P14 potential recorded over the scalp should change in amplitude in proportion to the distance from the generator and should have uniformly the same peak latency at all recording sites. However, Goff et al.[4] showed that the amplitude of the P14 was greatest at the frontal region which was most distant from the brain stem generator. The simulate finding was observed in the present study. Cracco[10] reported the following results. In the centralcoronal plane the P14 potential was most prominent at midline and at parasaggital recording locations contralateral to the stimulated median nerve. The side of potential maxima could be shifted from left to right by stimulating the right or left median nerve respectively. In the anterior-posterior plane, it was greatest at posterior frontal-parietal locations. These results might be explained from unhomogenity of the brain tissue as a volume conductor which existed between the generator of the P14 and each recording electrode.

The scalp distribution of N1 (N18) and P1 (P25) components has been reported by several authors. Broughton[8] and Allison et al.[13] recorded N18 and P25 potentials of approximately the same latency but of opposite polarity, suggesting a polarity inversion across the contralateral central sulcus. Broughton[8] explained these potentials as arising from a horizontally oriented "Dipole" generator, probably the primary sensory cortex folded into the posterior wall of the central sulcus. On the other hand, Cracco[3] and Goff et al.[4] observed that the peak latencies of these components increased from front to back, especially across the central sulcus. The scalp topographies of N20, P25, N35, P45, N70, P100, N140 and P190 by Goff et al.[4] fairly well corresponded with N1, P1, N2, P2, N3, P3 and P4 in our study respectively. The last peak P190 or P4 had a maximum amplitude in the central region at midline, and it may reveal the vertex potential.

P1 (P37) component to the posterior tibial nerve stimulation was best seen in the ipsilateral parieto-occipital area. The finding was much contrasted with the above results to the median nerve stimulation. Tsumoto et al.[16] reported a neurophysiological analysis of SEP evoked by common peroneal nerve stimulation and noted that P1 (P34) and N1 (N45) components slightly lateralized ipsilaterally to the stimulated nerve. They stated that the paradoxical phenomenon might be explained by the fact that the parietal electrode ipsilateral to the stimulated nerve reflected potential maxima from the contralateral leg-foot parietal sensory area situated vertically in the saggital plane.

The topographic mapping system described here revealed a dynamic and

systematic evolution of SEP on the scalp in normal individuals, and may greatly expand the clinical utility, especially as regards functional lesions[5].

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