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THE SCALP TOPOGRAPHY OF EARLY COMPONENTS OF SOMATOSENSORY EVOKED POTENTIAL (SEP) RECORDED SIMULTANEOUSLY FROM 16 RECORDING ELECTRODE LOCATIONS

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Abstract

The scalp somatosensory evoked potential (scalp-SEP) to median nerve stimulation was recorded simultaneously from 16 recording electrode locations in 31 normal subjects with MULTI-PURPOSE BIOPHYSICAL DATA PROCESSOR. The scalp topographies of the latencies of the P14, N18 and P25 components, and of the peak-to-peak amplitudes of the P14-N18 and N18-P25 were studied.

According to the previous studies the Nu-N13 of nuchal-SEP was closely related to the P14 of the scalp-SEP, but in this study the latencies of the P14 recorded at frontal region were smaller than those of the Nu-N13 in some subjects. The Nu-P10 of nuchal-SEP was approximately 4 msec less in latency compared with the P14 of scalp-SEP. This latency difference would allow to cover the distance from upper cervical cord to the cerebral cortex. It seemed that the P14 potential of scalp-SEP was more closely related to the Nu-P10 than the Nu-N13 component.

The P14 potential was widely distributed over the scalp and its peak latency was different at each recording location in the same subject. The peak latency of this potential was greatest at the somatosensory area (C3 or C4) contralateral to the stimulation and the greater was the increase in the distance from that area the greater the decrease in its peak latency.

The N18 and P25 potentials were also widely distributed over the scalp and their peak latencies increased from front to back. Especially these differences were greatest across the central sulcus. In coronal recordings on the two sides of the scalp, there were no apparent differences over the anterior region of the central sulcus, but over the posterior region of the central sulcus, the peak latencies

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were smaller over the ipsilateral hemisphere than the contralateral one to the side of stimulation.

The maxima of peak-to-peak amplitudes of the P14-N18 and N18-P25 were localized on the posterior contralateral quadrant of the scalp extending from Rolandic locations back to the occiput.

INTRODUCTION

It had been believed that the somatosensory evoked potential (SEP) could be recorded solely from the contralateral somatosensory area. However, more recent observations showed that it appeared widely over the scalp not only contralaterally but also ipsilaterally.[1~5)]

In early investigations, SEPs were recorded from restricted number of electrode simultaneously in each session which was repeated in the same subject and finally the scalp topography of SEP was studied in total of these sessions. However, these studies could not eliminate the effect of timelag in each session, so differences in the wave form characteristics of the scalp recorded SEP have been reported by many laboratories. These differences may be attributed to differences in stimulus parameters, variability contributed by back ground EEG activity, fluctuation in subject arousal level and contamination by potentials of extracranial origin. So, there was a certain limitation to complete more detailed exploration.[3,6~9)]

In this study, the scalp SEP to median nerve stimulation was recorded simultaneously from 16 recording electrode locations with MULTI-PURPOSE BIOPHYSICAL DATA PROCESSOR, then the scalp topography of the latencies of the P14, N18 and P25 components, and of the peak-to-peak amplitudes of the P14-N18 and N18-P25 was able to investigate more accurately.

METHODS AND MATERIALS

Observations were made on 31 normal young adults in total. These subjects were divided into two groups. The group A consisted of 16 subjects (7 females, 9 males) ranging in age from 20 to 27 years with the average of 21.6 years and group B consisted of 15 subjects (10 females, 5 males) ranging in age from 19 to 25 years, with the average of 20.6 years. All subjects were of right handed and revealed no EEG abnormalities during SEP recording.

Subjects were placed in a supine position with eyes closed in a semi-dark, quiet, and electrically-shielded room and encouraged to awake and relax. The EEG was continuously recorded to minimize muscle activity, drowsiness, eyeblinks, etc.

Two disc electrodes were attached on the skin over the median nerve just proximal to the wrist and used as stimulating electrode. The cathode was

placed 3 cm proximal to the anode. The stimulus was a 0.2 msec square wave pulse with an intensity of about 10 volts above thumb twitch threshold. The inter-stimulus interval was randomly varied from 1 to 4.5 sec. The averaged responses to the right and left median nerve stimulation were analyzed in each group separately.

K-M type (pad) recording electrodes were used on the scalp and disc electrodes were used on the skin over the nasion, mastoid and nuchal area. Recordings were obtained simultaneously from 16 electrode locations, including scalp placements of the 10-20 system. The other locations were selected to examine the potentials: nasion (N); Mastoid process (M1 or M2); and the suboccipital depression just above the spine of the second cervical vertebra in the midline (Nu).[10] In group A (Fig. 1), the all electrodes were placed on the contralateral cerebral hemisphere to the stimulated side, for the study of the anterior-posterior plane (A-P plane) differences. In group B (Fig. 1), the electrodes were placed on the bilateral scalp symmetrically, so favorable for the study of the coronal plane differences.

Recording was referential to the right or left earlobe, contralateral to the stimulated side and the negativity at the active electrode was upward. Input from the recording electrodes was led to conventional EEG amplifiers. The entire system had a time constant of 0.1 sec and a filter of 60 Hz. The output was summated by a MULTI-PURPOSE BIOPHYSIOLOGICAL DATA PROCESSOR SM-01 (SAN-EI INSTRUMENT Co., LTD.) and then displayed on the section paper through an X-Y plotter. The computer was triggered by the stimulator. Routinely 250 responses were summated and the first 102.4 msec following the stimulus were analyzed.

The method of naming of evoked potential components was based on the "Committee on the Methods at the International Symposium on Cerebral Evoked Potential in Man," Brussels, 1974.

Response peak latencies at each recording location were measured up to 0.01 msec for nuchal (Nu)-P10, Nu-N13, and P14 components and up to 0.1 msec for N18 and P25 components. These were not measured, when peaks were blunt or difficult to be identified from the ongoing background activity. The peak-to-peak method was used for the analysis of the component amplitude (Broughton),[11] then P14-N18 and N18-P25 at each recording location were measured in this study.

RESULTS

1. Representative Records

Typical records of group A and group B were shown in Fig. 1. The

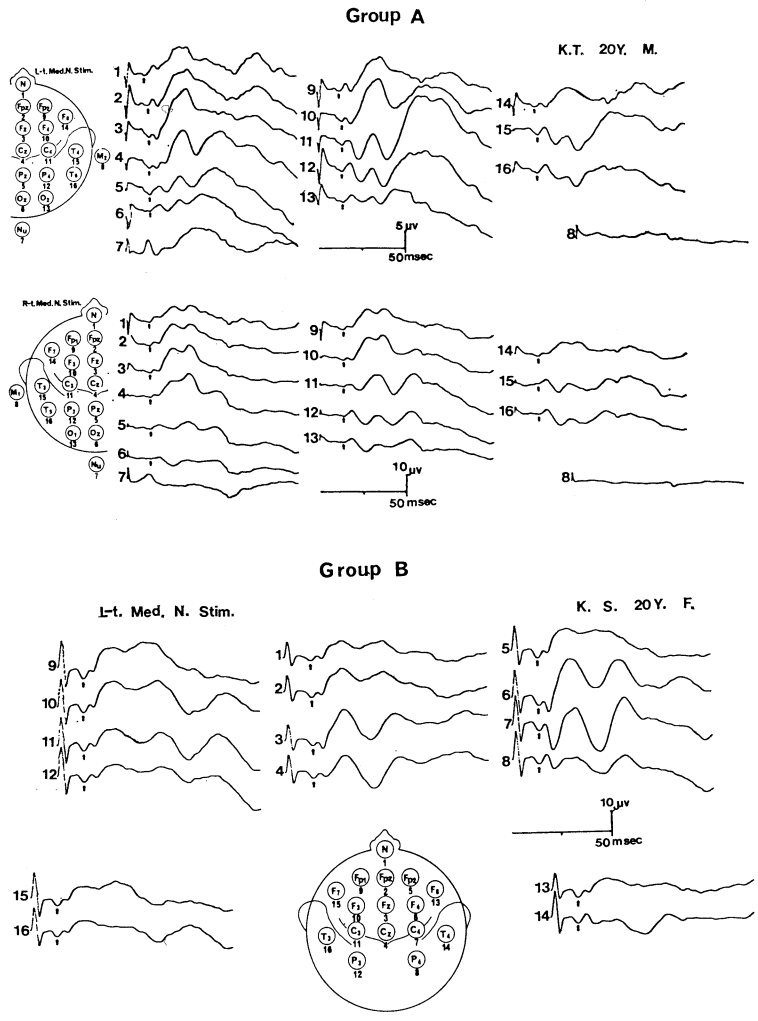

Fig. 1. Distribution of SEPs in a representative subject. Arrows indicate P14 components. In this and all succeeding figures, upward deflections indicate negativity at recording electrode, stimulus occurring at trace onset, and each trace is the average of 250 responses.

early components of SEP were distributed widely over the scalp except for the nuchal (Nu) and mastoid (M1 or M2) areas.

2. Nuchal Evoked Potentials

In group A, nuchal evoked potentials consisted of a triphasic potential, initially positive. But in this paper, I studied only the first positive potential

(Nu-P10) and the following negative potential (Nu-N13). The peak latencies of Nu-P10 and Nu-N13 were 9.67±1.29 ($\bar{\chi}$±S.D.) msec and 13.20±1.32 msec to the left median nerve stimulation and 9.89±0.97 msec and 13.18±1.19 msec to the right median nerve stimulation, respectively.

3. Topographical Comparison of P14 Peak Latencies

This potential (P14) may correspond with P15 by Goff *et al.* (1977).[4] This potential was recorded consistently in all subjects and distributed widely over the scalp except for the mastoid and nuchal electrode locations. The mean peak latencies of P14 at each recording location were shown in Fig. 2. In Fig. 3, the peak latencies at each locations were compared with the peak latency at C3 (or C4) where was the nearest position to the primary sensory area.[11]

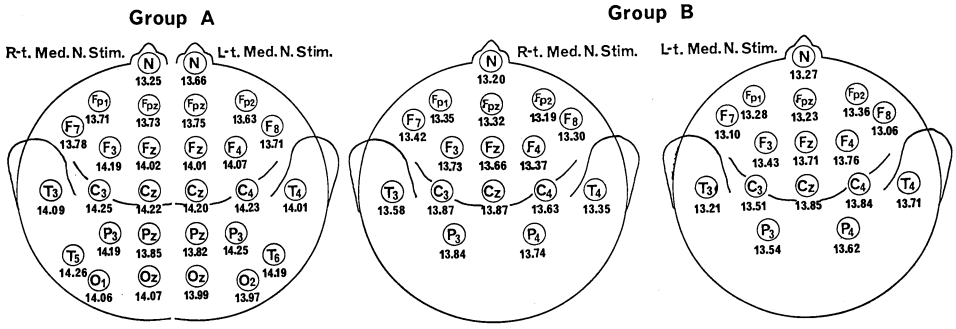

Fig. 2. Topographies of mean peak latencies of P14 components. (msec)

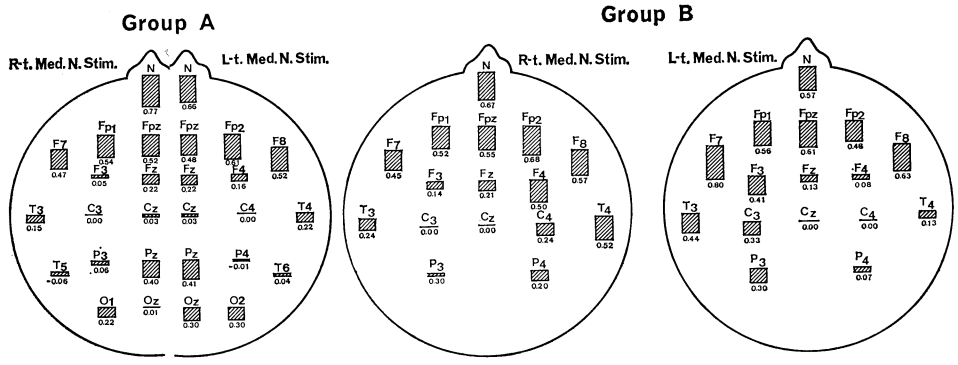

Fig. 3. Comparison of P14 peak latency differences among C3 (or C4) and other recording electrode locations. Peak latencies were greatest at the C3 (C4) or P3 (P4) and decreased slightly from these areas to the frontal, lateral and occipital ones progressively. (msec)

In group A, the potential showed consistent A–P latency differences. The peak latencies were greatest at the C3(C4) or P3(P4) and slightly decreased from these areas to the frontal, lateral and occipital ones, progressively. Especially in the frontal area, the peak latencies were smallest.

In group B, the P14 peak latency was also greatest at the C3(C4) or P3(P4) contralateral to the side of stimulation and the greater was the increase in the distance from this area the greater the decrease in the peak latencies. In coronal referential recordings on the two sides of the scalp, the peak latency of P14 decreased over the hemisphere ipsilateral to the stimulated median nerve. However, these peak latency differences were all within the narrow range of one msec.

4. Topographical Comparison of N18 and P25 Peak Latencies

The mean peak latencies of N18 and P25 at each recording location were shown in Fig. 4 and 5.

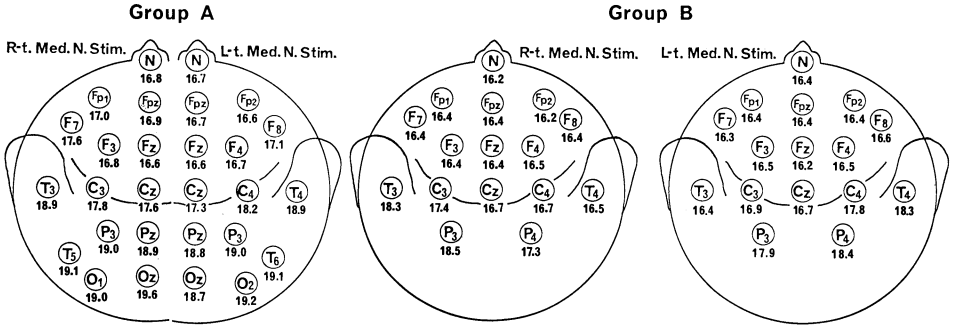

Fig. 4. Topographies of mean peak latencies of N18 components. (msec)

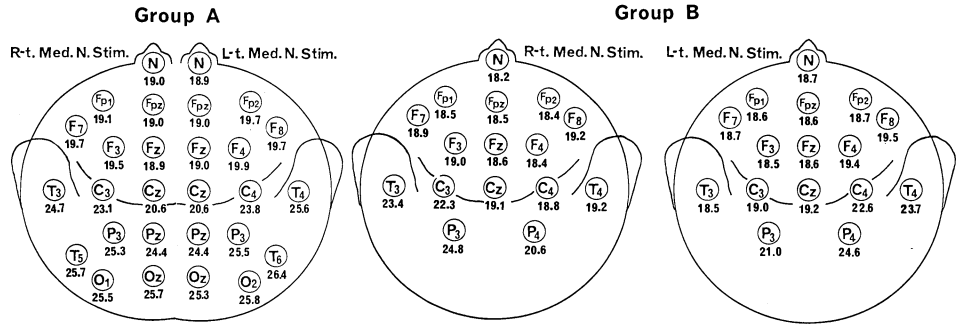

Fig. 5. Topographies of mean peak latencies of P25 components. (msec)

In group A (Fig. 4-A and 5-A), the peak latencies of these components increased from front to back and the peak latency differences were greatest across the central sulcus.

In group B (Fig. 4-B, 5-B and 6), the A-P latency differences were the same as mentioned above and the coronal latency differences were as the followings. There were no apparent differences over the anterior region of the central sulcus. However, over the posterior region of the central sulcus, the peak latencies were smaller on the ipsilateral hemisphere than the contralateral one to the side of stimulation, and were greater at lateral than at medial locations in the contralateral hemisphere. These peak latency differences were more prominent in P25 than in N18 (Fig. 6).

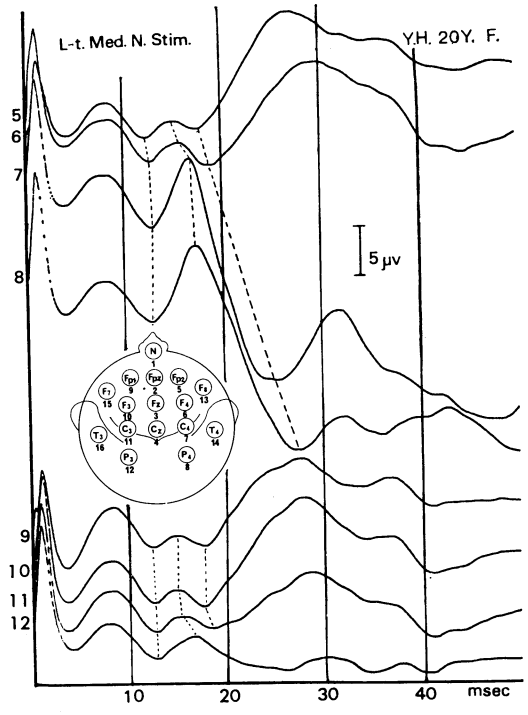

Fig. 6. Partial illustrative presentation of SEPs (group B). The progressive latency shifts of the N18 and P25 components were recorded from both hemispheres in the A-P plane, especially across the central sulcus. In the coronal plane, there was no apparent difference over the anterior region of the central sulcus. However, over the posterior region of it, the peak latencies were smaller on the ipsilateral hemisphere to the side of stimulation.

5. Topographies of Peak-to-peak Amplitude of P14-N18 and N18-P25

Topographical mappings of peak-to-peak amplitude of P14-N18 and N18-P25 were illustrated in Fig. 7 and 8.

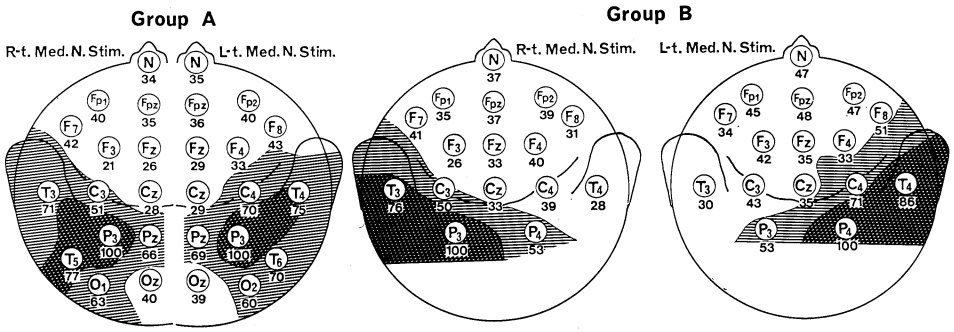

Fig. 7. Topographies of P14-N18 peak-to-peak amplitude. In this and following figures, double crosshatching indicates all locations at which the component was 75 % or more of its maximal amplitude, and the single crosshatching indicates 50-75 % region. (%)

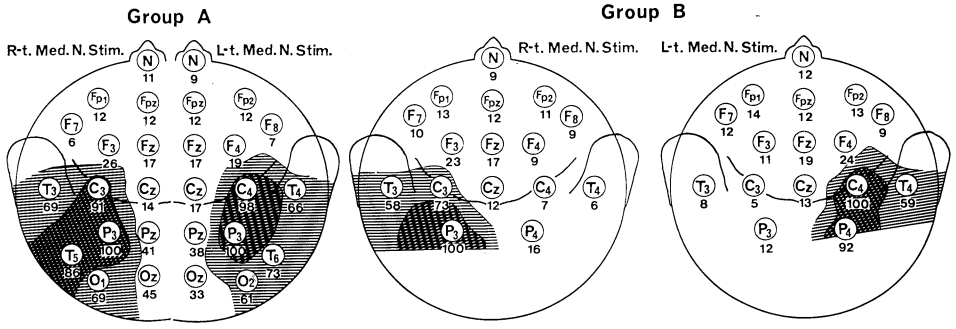

Fig. 8. Topographies of N18-P25 peak-to-peak amplitude. (%)

In group A (Fig. 7-A and 8-A), the distributions of these amplitudes were localized mainly on the posterior contralateral quadrant of the scalp extending from Rolandic locations back to the occiput.

In group B (Fig. 7-B and 8-B), there were no significant differences between ipsi- and contra-lateral sides in the anterior region of central sulcus. In the posterior region of central sulcus, the amplitudes were larger in the contralateral than the ipsilateral hemisphere. The distributions of N18-P25 amplitudes were relatively restricted at the parieto-occipital region comparing with those of P14-N18 amplitudes.

6. Statistical Analysis

Statistical comparisons were done with the two-way analysis of variance for subjects and electrode locations concerning the peak latencies of P14, N18 and P25 components, and the amplitudes of P14–N18 and N18–P25, respectively. The study revealed significant differences at the 1.0 % level among subjects and electrode locations about each peak latency and amplitude.

DISCUSSION

Since Dawson (1947)[12] introduced averaging techniques by recording the somatosensory evoked potential (SEP) 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.[1,13,14] However, more recent observations show that these components are distributed widely over the scalp,[1~5] suggesting the complex nature of the neuronal substrate which underlies the SEP in man.

1. Nuchal-SEP

Giblin[15] and Halliday *et al.*[16] recorded SEPs in healthy subjects and in patients with lesions of the nervous system. In their study, when sensory loss was slight the potentials appeared normal or slightly delayed, but when it was severe the evoked potential from the damaged hemisphere was absent or markedly abnormal. These abnormal responses highly associated with proprioseptive loss, while apparently normal records were obtained from the patient with loss of pain and temperature perception only. Furthermore, Larson *et al.*[17] reported in a patient that the latency, configuration, and amplitude of the cerebral evoked potentials secondary to transcutaneous stimulation of the sciatic nerve were the same before and after a spinothalamic tractotomy. The above findings suggest that the afferent volley travels by the dorsal column pathways. In fact, some workers[10,18,19] recorded somatosensory evoked response over the spine. Cracco,[10] Mortillaro and Emser[18] recorded SEPs from multiple sites over the spine and suggested that the evoked potential traveled in the dorsal column with progressive increase in latency at more rostral recording location. Mathews *et al.*[19] noted no such increase in latency of evoked potential between the lower and upper neck, and concluded that the components were generated from fixed sites, possibly including dorsal root ganglia, spinal cord interneurons, dorsal column nuclei, and cerebellum. Kimura *et al.*[20] observed that the negative peak, N0, of the nuchal-SEP (Nu-N13 in this study) was 0.9 ± 0.6 msec less in latency compared with the initial positive peak, P0, of the scalp-SEP (C3-P14 or C4-P14 in this study). From the results, they

suggested that N0 was generated primarily from the dorsal column nuclei and P0 from either lemniscal inflow or cerebellar or thalamic nuclei.

Recently we also observed[21,22] progressive increases in latency at more rostral recording locations over the spine and supported Cracco's notion[10] that the nuchal-SEP was a traveling wave in the dorsal column. In this study, the estimated latency of Nu-N13 (13.20 ± 1.32 or 13.18 ± 1.19 msec) was 1.04 ± 0.39 or 1.07 ± 0.38 msec less than the latency of C4-P14 (14.23 ± 1.04 msec) or C3-P14 (14.25 ± 1.37 msec) to the stimulation of the left or right median nerve, and this result was almost the same as those reported by Kimura *et al.*[20] Comparing with the peak latency of Nu-N13 with N-P14 (P14 recorded at Nasion), the latency of N-P14 was smaller than Nu-N13 in 12 subjects out of 31 subjects. Thus, it seems unreasonable that the P14 of scalp-SEP is an evoked potential following Nu-N13.

On the other hand, if Nu-P10 is accepted as traveling wave which ascends in the posterior column, this potential may be equivalent to the activity generated from the dorsal column nuclei. Furthermore, we have shown that the peak latency of Nu-P10 (9.67 ± 1.29 or 9.89 ± 0.97 msec) was 4.60 ± 0.59 or 4.56 ± 0.89 msec less than the latency of C4-P14 or C3-P14 to the stimulation of the left or right median nerve, respectively. The latency difference of approximately 4.6 msec between Nu-P10 and P14 potential would allow to cover the distance for an impulse to travel from the upper cervical cord to the cerebral cortex. Thus, the Nu-P10 component recorded over the high cervical area may be assumed as activities of dorsal column nuclei and the P14 component of scalp-SEP may be more closely related to the Nu-P10 comparing with the Nu-N13 component. The results mentioned above would be consistent with the interpretation that P14 potential might not be subcortical origin.

2. P14 Component of Scalp-SEP

The P14 potential has not been observed consistently, but using ear reference recording, it appears constantly over the scalp. It is distributed widely over the scalp. The area of its potential maxima has not been 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.[2,8,13,23] Goff *et al.*[8] 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 (Broughton).[13] Allison *et al.*[23] stated that it represented the VPL response and the ensuing thalamo-cortical volley. Nakanishi *et al.*[24] 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 absence 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 lemniscal systems from the medulla to the thalamus. Greenberg *et al.*[25] 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 the P14 potential recorded from parietal scalp was the afferent volley of depolarization conducted in the lemniscal 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. Cracco[3] reported the following results. In the central-coronal plane the P14 potential was most prominent at midline and at parasaggital recording locations contralateral to the stimulated median nerve. The side of its 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. Although we did not measure the amplitude of the P14 potential itself, we found the peak latency to be greatest at the somatosensory area contralateral to the stimulus and smallest at the frontal region. These results might be explained from unhomogeneity of the brain tissue as a volume conductor which existed between the generator of the P14 and each recording electrode.

On the other hand, the existence of core of amplitude and the smallest latency at the frontal area suggested that the origin or the function of the P14 potential might be closely related to this area. It was conceded generally that there was a close relation between the sensory system and the frontal lobe.[26] Chatrian *et al.*[27] postulated that the anterior part of the cingulate gyrus plays a role in emotional behavior and provides a neural basis for the aversive drive and affects what is referred to as the motivational-affective dimension of pain. Cracco *et al.*[28] mentioned on the mechanism which underlay the genesis of the earlier oscillatory potentials evoked by visual stimulation and suggested that these wavelets might carry coded information which was of value to the responding organism or they might be involved in

setting the state of general excitability of the visual system. We also suppose the similar significance of the P14 potential evoked by somatosensory stimulation.

3. N18 and P25 Components of Scalp-SEP

The scalp distribution of N18 and P25 components has been reported by several authors. Broughton[13] and Allison *et al.*[23] recorded N18 and P25 potentials of approximately the same latency but of opposite polarity, suggesting a polarity inversion across the contralateral central sulcus. Broughton[13] explained these potentials as arising from a horizontally oriented "Dipole" generator, probably the primary somatosensory cortex folded into the posterior wall of the central sulcus. On the other hand, Goff *et al.*[4] and Cracco[2] observed that the peak latencies of these components increased front to back, especially across the central sulcus.

Studies of ipsilateral SEP responses to the peripheral nerve stimulation in man have been scarce in number.[2,4,5,17,29~31] Larson *et al.*[17,31] reported that the ipsilateral responses might be (1) a reflection of volume conduction, (2) mediated by the corpus callosum, or (3) due to uncrossed fibers, and suggested that the volume conduction was most likely. On the other hand, Liberson[29] and Williamson *et al.*[30] proposed the transmission to the ipsilateral hemisphere from the contralateral cortex through the corpus callosum. Tamura[5] showed that the peak latencies of the ipsilateral responses were about 4-5 msec longer than those of the contralateral ones and he observed that in patients with unilateral sensory impairment, stimulation of the normal side evoked normal responses over the contralateral and ipsilateral hemispheres but when the affected side was stimulated all SEPs were markedly reduced in amplitude or abolished over both hemispheres. So he concluded that ipsilateral responses were the result of subsequent activation of the ipsilateral hemisphere via an interhemispheric pathway, possibly involving the corpus callosum. In animals, SEPs of similar latency have been recorded from separate, independent cortical area (Rose and Mountcastle)[32] and the existence of uncrossed fibers in somatosensory pathways has been verified.[33~35] In human case, Penfield and Jasper[36] reported that electrical stimulation of the cerebral cortex of conscious patients seemed to demonstrate the existence of a second sensory somatic representation.

The apparently progressive latency shifts over the scalp in the anterior-posterior plane, especially across the central sulcus,[2,4] was also observed in this study. In coronal plane, there was no apparent difference over the anterior region of the central sulcus. However over the posterior region of the central sulcus, the peak latencies were smaller on the ipsilateral hemisphere than the

contralateral one to the side of stimulation (Fig. 6). The last result is not consistent with previous reports[2,5] and could be explained by neither volume conduction nor transcallosal transmission. Traveling waves of the SEP may be the result of the algebraic summation of the activities of multiple cerebral generators activated non-simultaneously by thalamic, callosal or other numerous afferent system (Cracco).[2]

4. Peak-to-Peak Amplitudes of P14-N18 and N18-P25

Concerning the distribution of the amplitudes of these early components, many authors reported that they were projected mainly to the posterior contralateral quadrant of the scalp extending from Rolandic locations back to the occiput and occasionally to anterior contralateral locations. Goff *et al.*[4] reported about the scalp topography of human SEP in detail. They measured base-to-peak, but in the present study peak-to-peak distance was used to evaluated SEP amplitudes. The scalp topographies of N20, and P25 or P30 amplitudes by Goff *et al.*[4] well corresponded with those of P14-N18 and N18-P25 amplitudes in this study. The site of maximum amplitude of the potentials was located on the posterior contralateral quadrant of the scalp, where there might be a number of generators.

From the above results, it is suggested that the early component (P14, N18, and P25) of scalp-SEP may be diffusely distributed over the cortex under each recording electrode location and may be focused on the posterior contralateral quadrant hemisphere of the somatosensory cortex closely related to somestic sensorium. Namely, these waves may carry coded information which is of value to the responding organism or they may be involved in setting the state of general excitability of the somatosensory system, as associating with protective movement or arousal of attention and memory related to the stimulation. These findings emphasize the complex nature of neuronal substrate which underlies the SEP in man.

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