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THE INITIAL POSITIVE COMPONENT OF THE SCALP-RECORDED SOMATOSENSORY EVOKED POTENTIAL IN NORMAL SUBJECTS AND IN PATIENTS WITH NEUROLOGICAL DISORDERS *

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The first event of the scalp-recorded somatosensory evoked potential (SEP) to median nerve stimulation is usually a small positive potential with a peak latency of 13—17 msec (p_{15}) in routine recordings (Allison 1962; Goff et al. 1962, 1966, 1969, 1977; Allison et al. 1963, 1974; Broughton 1969; Cracco 1972; Tamura 1972; Tsumoto et al. 1973; Halliday 1975). Some workers have suggested that the p_{15} potential, the subsequent negative potential and inconstantly appearing notch on the next positive component are all generated in thalamocortical axons (Goff et al. 1962, 1966). But others have recently proposed a subcortical origin for the p_{15} potential (Broughton 1969; Cracco 1972; Allison et al. 1974). Thus there is no agreement about the neural systems that give rise to the p_{15} potential. Many workers have studied SEPs to median nerve stimulation in patients with neurological disorders (Halliday and Wakefield 1963; Giblin 1964; Domino et al. 1965; Liberson 1966; Larson and Sances 1968; Stohr and Goldring 1969; Williamson et al. 1970; Tamura 1972; Tsumoto et al. 1973; Noël and Desmedt 1975; Green and Hamilton 1976). However, alterations of the p_{15} potential have not been described yet in their studies.

From a clinical point of view, the present study has been undertaken with the aims: (1) to study general characteristics of the p_{15} potential in normal subjects as control; (2) to examine alterations of the p_{15} potential in selected patients with neurological disorders; and then (3) to elucidate the origin for the p_{15} potential.

Methods and materials

The individuals examined were eight normal young volunteers, six male and two female, ranging in age from 20 to 26 years, and 12 selected patients with unilateral sensory impairment of joint position, vibration, tactile and temperature due to neurological disorders. These patients were divided into 3 groups: group I consisted of seven cases with lesions at the thalamus; group II two cases with lesions above the thalamus; group III three cases with lesions below the thalamus. The nature of pathology in these cases was established on the basis of all the information available in each case; clinical, angiographic, computed tomography and postmortem examinations.

Recordings of SEP were made while the subjects were in a supine position with eyes closed. The stimulating electrodes (2.5 mm × 25 mm) were attached over the median nerve just proximal to the wrist with the cathode about 2 cm proximal to the anode. Electrical stimuli were applied percutaneously at a rate of 1/1.5 sec or occasionally at a rate of cardiac pulse by using the ECG to minimize the cardiac-related activity. The stimuli were 0.2 msec rectangular pulses and were adjusted to produce a small twitch of the thumb. Recording electrodes consisted of thin steel needles. Evoked potentials were usually obtained from 4 derivations on all subjects: (1) bipolar recordings over the brachial plexus where one electrode was placed at Erb's point (the junction of the posterior border of the sternocleidomastoid and the superior margin of the clavicle) and the other 3 cm proximal to it; (2) the suboccipital depression just above the supine of the second cervical vertebra in the midline — earlobe recordings; (3) C_3—A_1 or C_4—A_2 recordings; and (4) C_3—F_3 or C_4—F_4 recordings (International 10—20 system). In normal subjects, moreover, monopolar (ear reference) recordings were made from a symmetrical array of 12 scalp electrodes (F_3, C_3, P_3, O_1, F_z, C_z, P_z, O_z, F_4, C_4, P_4, O_2), and bipolar scalp recordings were also performed. Evoked potentials were amplified with an EEG machine (Sanei Sokki EG-130) and were then fed into a 4-channel averaging computer (Sanei Sokki Mediac 7T07). In all recordings relative negativity in grid 1 had resulted in an upward deflection. The recording system had a time constant of 0.3 sec and a frequency response flat to 2 kc within 3 dB. Usually, 200 consecutive responses were summated and the first 90 msec and 40 msec following the stimulus were analyzed.

Results

Normal subjects

The median nerve action potential over the brachial plexus at Erb's point (MNP), the suboccipital evoked potential (SOP) and the SEPs to median nerve stimulation in a normal subject are shown in Fig. 1. The MNP, the SOP and the p_{15} potential recorded with an ear reference electrode were obtained in all eight subjects, but the p_{15} potential recorded with bipolar scalp electrodes could be rarely distinguished. In ear reference recordings the p_{15} potential was followed by negative-positive diphasic potentials. Table I shows observed values for the peak latencies of the MNP, the SOP and the p_{15} potential recorded with ear reference electrode to right median nerve stimulation. There were no significant differ-

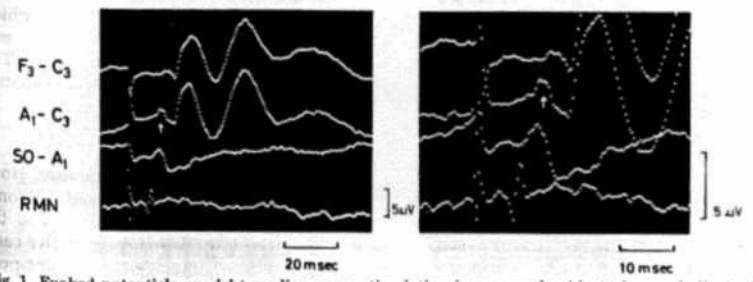

Fig. 1. Evoked potentials on right median nerve stimulation in a normal subject. Arrows indicate the p_{15} potential. In this and all subsequent figures, negativity in grid 1 produces upward deflections. Abbreviations in this and following figures: SO, suboccipital depression; RMN, right median nerve; LMN, left median nerve; R, right; L, left; St., stimulation.

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TABLE I

Mean peak latencies and base-to-peak amplitudes of the median nerve action potential obtained over the brachial plexus (MNP), the suboccipital evoked potential (SOP) and the P_{15} potential recorded with ear reference electrode to right median nerve stimulation in normal subjects.

	Median nerve potential (MNP)		Suboccipital evoked potential (SOP)		Initial positive potential (P_{15})	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Peak latency (msec)	9.3	0.5	13.1	0.7	14.2	1.0
Amplitude (base-to-peak) (μV)	3.6	0.8	1.6	0.4	0.9	0.3

ences between these evoked potentials to right side stimulation and those to left side stimulation. The peak latency of the P_{15} potential ranged between 12 and 15 msec with a mean of 14.2 msec, which was 1.1 msec greater than that of the SOP and was 2.1 msec less than the onset latency of the initial negative component of the SEP recorded with bipolar C_3—F_3 leads to the right median nerve stimulation. The mean amplitude of the P_{15} potential (measured base-to-peak) was $0.9 \pm 0.3\,\mu V$. The P_{15} potential was widely distributed over the scalp in ear reference recordings. Although the subsequent responses of the SEP were largely limited in their distribution to the scalp contralateral to the stimulated median nerve, the P_{15} potential was obtained over the scalp at the same electrode locations to both contralateral and ipsilateral median nerve stimulations (Fig. 2). Furthermore, in the sagittal plane and in the central-coronal plane, it was also recorded with an ear reference electrode (Fig. 3A), but in bipolar scalp recordings it underwent a cancellation effect (Fig. 3B).

Patients with neurological disorders

Group I. Patients with lesions at the thalamus. This group consisted of a case with glioblastoma of the thalamus (case 1) and 6 cases with vascular lesions in the thalamus (cases 2—7) (Table II). Case 1, a 55-year-old female, had begun to experience numbness and weakness on the left side 5 months prior to the time of testing, when sensory impairment of joint position and vibration, especially joint position, and tactile and temperature was revealed on the left side. There were flaccid hemiplegia and exaggerated deep tendon reflexes with a Babinski sign on the left side. Cerebral angiograph and brain scanning showed a mass in the right thalamus. The evoked potentials to stimulation of the right (non-affected) side were of normal configuration and amplitude. To stimulation of the left (affected) side, however, only the P_{15} potential was observed in scalp recordings, whereas the MNP and the SOP were obviously normal (Fig. 4). The patient died 7 months later. At autopsy, glioblastoma was found in the right thalamus and also invading the hypothalamus, internal capsule and midbrain on the right side. There were six cases (3 male and 3 female, ranging in age from 53 to 64 years) with thalamic vascular lesions which were all defined by clinical signs and symptoms and computed tomography. The SEPs were recorded 2 months—10 years after the onset, when sensory impairment for all modalities of the unilateral extremities was revealed in all cases. Stimulation of the non-affected side evoked normal responses. However, to stimulation of the affected side only the P_{15} potential was well defined, and the subsequent responses were absent in five cases with severe sensory impairment of joint position (cases 2—6) and were significantly altered in latency and amplitude in a case with moderate sensory impairment (case 7).

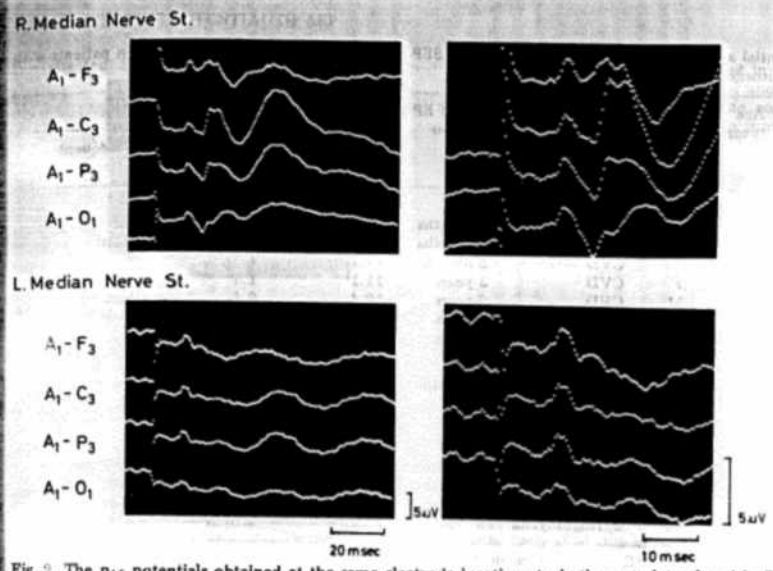

Fig. 2. The P_{15} potentials obtained at the same electrode locations to both contralateral and ipsilateral median nerve stimulations. Note that latencies and amplitudes of the P_{15} potentials are almost equal at all electrode locations.

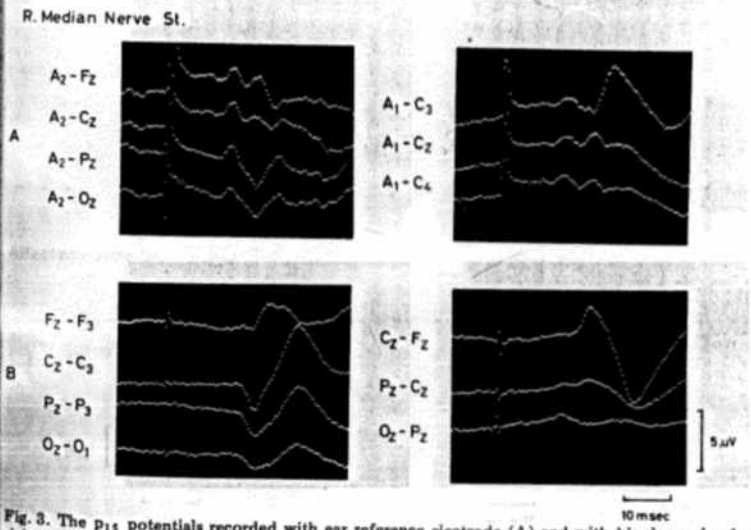

Fig. 3. The P_{15} potentials recorded with ear reference electrode (A) and with bipolar scalp electrodes (B). The right median nerve was stimulated. Note that the P_{15} potential was well developed in ear reference recordings (A), but it underwent a cancellation effect in bipolar scalp recordings (B).

TABLE II

The P_{15} potential and the subsequent responses of the SEP to stimulation of the affected side in patients with neurological disorders.

	Age (years)	Sex	Clinical diagnosis	Time of EP test after onset	P_{15} potential		
					Peak latency (msec)	Amplitude (μV)	Subsequent responses
Group I							
Case 1	55	F	Glioblastoma	5 months	13.3	1.6	absent
Case 2	59	F	CVD	6 months	14.3	0.9	absent
Case 3	53	F	CVD	2 months	14.4	1.0	absent
Case 4	55	F	CVD	3 years	11.1	1.3	absent
Case 5	53	M	CVD	2 years	12.4	0.4	absent
Case 6	63	M	CVD	10 years	12.6	1.1	absent
Case 7	64	M	CVD	7 years	13.3	0.6	abnormal
Group II							
Case 8	60	M	Metastatic brain tumor	2 months	12.2	1.3	abnormal
Case 9	33	F	Meningioma	7 years	12.1	1.1	abnormal
Group III							
Case 10	32	M	CVD	6 months	?	?	ill-defined
				8 months	14.3	0.5	diminished
Case 11	47	M	CVD	9 months	absent	absent	absent
Case 12	43	M	Syringomyelia	10 years	absent	absent	absent

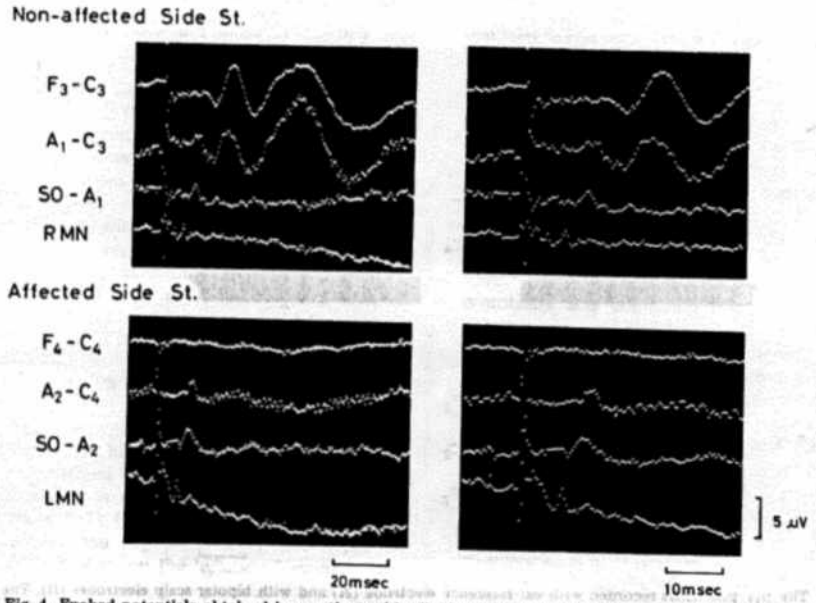

Fig. 4. Evoked potentials obtained in a patient with glioblastoma in the right thalamus. Note that only the P_{15} potential was well developed without the subsequent responses to stimulation of the affected side.

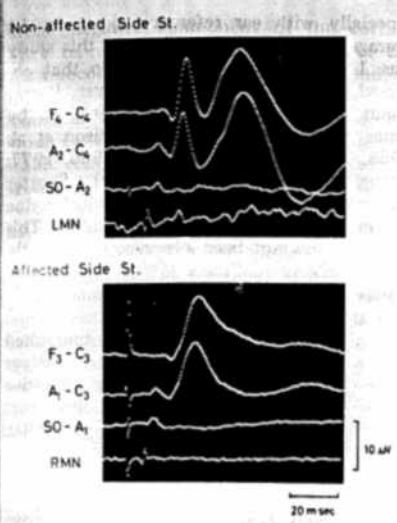

Fig. 5. Evoked potentials obtained in a patient with metastatic tumors in the parietal and frontal lobes (case 8). Note the absence of the second negative-positive component of the SEP to stimulation of the affected side.

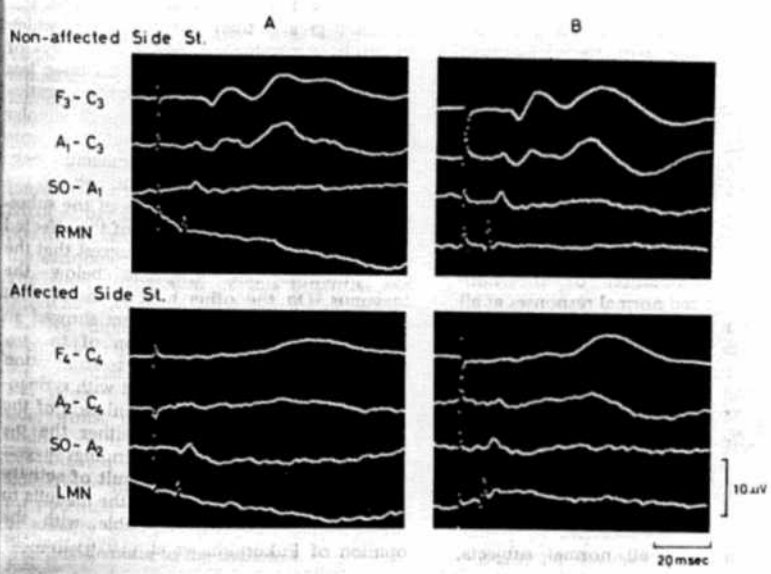

Fig. 6. Evoked potentials obtained in a patient with vascular lesion in the brain stem (case 10). Note the ill-defined P_{15} potential to stimulation of the affected side at the first time of testing (A). Two months later, however, a relatively normal P_{15} potential appeared with diminished subsequent responses to stimulation of the affected side (B).

Group II. Patients with lesions above the thalamus. This group consisted of a 60-year-old man with metastatic tumors in the left parietal and frontal lobes (case 8) and a 33-year-old woman with a meningioma in the left parietal convexity (case 9), which were identified at the time of operation. They had sensory impairment for all modalities of the unilateral extremities. In these cases, stimulation of the affected side evoked a P_{15} potential which had normal configuration and latency, but the subsequent responses showed a profound change in the second negative-positive component, which was almost absent. When the non-affected side was stimulated, however, normal responses were seen at all electrode locations as shown in Fig. 5.

Group III. Patients with lesions below the thalamus. This group consisted of two cases with vascular lesions of the brain stem (cases 10 and 11) and one case with syringomyelia of the cervical cord (case 12), which were identified by clinical examination and computed tomography. They had sensory impairment for all modalities of the unilateral extremities. Both cases with vascular lesions of the brain stem showed an absence or profound reduction of the SEPs, including the P_{15} potential to stimulation of the affected side. Case 10 showed an ill-defined P_{15} potential to stimulation of the affected side at the first time of testing, but 2 months later, when joint position sense had recovered markedly, a relatively normal P_{15} potential appeared at a given latency with diminished subsequent responses. But stimulation of the non-affected side produced normal responses at all electrode locations (Fig. 6). On the other hand, in a patient with syringomyelia who had a sensory loss for all modalities on the right upper limb, no recognizable potentials except for the MNP could be evoked to stimulation of the affected side.

Discussion

As Cracco (1972) reported, the P_{15} potential was recorded in all normal subjects, especially with ear reference electrode. Its average peak latency obtained in this study was 14.2 msec and was similar to that obtained by Tamura (1972). However, it was about 1 msec less than those described by other workers (Allison 1962; Allison et al. 1963, 1974; Goff et al. 1962, 1966, 1972; Broughton 1969; Cracco 1972). Shorter latency obtained in this study is probably due to smaller size of the body examined. This potential has not been observed consistently by all workers. As Cracco (1972) pointed out, however, it may be due to the small amplitude of this potential and recording technique. This potential could be hardly distinguished with bipolar scalp recordings, but not with ear reference recordings. This finding and positive polarity of the P_{15} potential also indicate that the earlobe could be active for this potential. Some workers have suggested that the initial triphasic response of the SEP including the P_{15} potential is generated in the thalamocortical axons (Goff et al. 1962, 1966). However, the peak latency of the P_{15} potential was 1.1 msec greater than that of the SOP which might be generated in afferent pathways of the upper spinal cord and was 2.1 msec less than the onset latency of the initial negative component of the SEP recorded with bipolar C_3—F_3 electrodes. Furthermore, in patients with lesions at or above the thalamus, only the P_{15} potential was recorded in spite of the absence or profound alterations of the subsequent responses to stimulation of the affected side (Table II). These findings suggest that the P_{15} potential may originate below the thalamus. On the other hand, patients with vascular lesion in the brain stem showed an absence or profound alteration of the P_{15} potential with the normal SOP to stimulation of the affected side. In a patient with syringomyelia of the spinal cord, stimulation of the affected side could evoke neither the P_{15} potential nor the SOP. These findings suggest that the P_{15} potential is the result of activity of the afferent pathways from the medulla to the thalamus. It is compatible with the opinion of Fukushima et al. (1976) that the initial negative component of the cortical SEPs represents the afferent volley of the thalamocortical fibers. On the other hand, all SEP components are mediated by the medial lemniscal system as defined by Rose and Mountcastle (1959). Thus the P_{15} potential would reflect activity of the medial lemniscal systems in the brain stem. Recently, auditory evoked potentials generated in the brain stem have been recorded over the human scalp and used for evaluation of clinical problems (Jewett et al. 1970; Starr and Hamilton 1976; Stockard and Possiter 1977). The P_{15} potential recorded with ear reference electrode may be useful in diagnosis and prognosis of the brain stem lesions involving the sensory pathways.

Summary

The initial positive component of the scalp-recorded somatosensory evoked potential to median nerve stimulation was studied in 12 patients with sensory impairment for all modalities of the unilateral extremities due to lesions at or above or below the thalamic level, taking the potentials obtained from eight normal subjects as control. In normal subjects, this potential could be easily obtained in ear reference recordings with a peak latency of 12—15 msec. This finding and positive polarity of this potential indicate that the earlobe could be active for this potential. The wide distribution of this potential was different from the subsequent negative-positive diphasic components. In patients with lesions at or above the thalamic level the P_{15} potential was of normal configuration and latency to stimulation of the affected side, whilst in patients with lesions in the brain stem or in the cervical cord it could not be obtained to stimulation of the affected side. These findings and short latency of the P_{15} potential suggest that it may be the result of activity of the medial lemniscal systems from the medulla to the thalamus.

Résumé

Ondes positives initiales du potentiel évoqué somato-sensitif enregistrées sur le scalp chez des sujets normaux et des malades atteints de troubles neurologiques

L'onde positive initiale du potentiel somato-sensitif évoqué par stimulation du nerf médian et enregistré sur le scalp a été étudiée chez 12 malades avec troubles sensitifs d'une extrémité pour toutes les modalités, par lésion située soit au-dessus soit au-dessous du niveau thalamique, en utilisant les potentiels obtenus chez 8 sujets normaux comme contrôle. Chez les sujets normaux, ce potentiel pouvait être facilement obtenu sur des enregistrements avec référence à l'oreille avec une latence de pic de 12—15 msec. Ces données ainsi que la polarité positive de ce potentiel indiquent que le lobe de l'oreille pourrait être actif en ce qui le concerne. La large distribution de ce potentiel est différente de celle de la composante diphasique négative-positive consécutive. Chez les malades avec lésions au niveau ou au-dessus du niveau thalamique, le potentiel P_{15} est de configuration et de latence normales à la stimulation du côté atteint alors que chez des malades présentant des lésions du tronc cérébral ou de la moelle cervicale il pourrait ne pas être obtenu par stimulation du côté atteint. Ces faits, et la latence courte du potentiel P_{15}, suggèrent qu'il peut être le résultat de l'activité des systèmes lemniscaux médian du bulbe au thalamus.

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