Kinesthetic Evoked Potentials in Neurological Patients with Impaired Deep Sensitivity

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Latent periods of early components of kinesthetic evoked potentials in both hemispheres are increased in the patients with disordered deep sensitivity of the upper limbs *vs.* healthy volunteers. Disorders of proprioceptive sensitivity can be diagnosed early when clinical symptoms are not yet manifest.

Key Words: *kinesthetic evoked potentials; proprioceptive sensitivity; demyelinizing diseases; hypesthesia; lemniscus system*

Registration of somatosensory evoked potentials of the brain (SSEP) has been widely used in clinical studies of organic involvement of the nervous system. A. M. Halliday and J. E. Desmedt investigated SSEP in the involvement of peripheral nerves and found that evoked responses disappeared when peripheral nerve was completely disrupted [7], the amplitude of SSEP early components decreased, and their peak latencies increased in less grave disorders of the peripheral nerve stems [8]. Evoked responses completely disappear in some patients with Charcot–Marie disease [4]. In disseminated sclerosis, latent periods (LP) of SSEP components were prolonged and their amplitude decreased [1,3,9,10]. Decrease in amplitude, prolongation of LP, and disappearance of the early components of somatosensory evoked potentials were observed in the involvement of posterior stems of the spine [5,6].

These studies showed that SSEP recording can be used for objective diagnosis of somatosensory involvement and for studies of pathophysiological mechanisms of many nervous diseases. On the other hand, it is interesting to investigate the potentials evoked by selective stimulation of peripheral components of the kinesthetic analyzer, most of all involved in many diseases of the central nervous system.

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Our purpose was to study kinesthetic evoked potentials (KEP) in response to selective stimulation of peripheral proprioreceptors of the upper limb in patients with diseases of the central nervous system involving impairment of deep sensitivity.

MATERIALS AND METHODS

Thirty-three patients (22 men and 11 women) aged 28-57 years were examined. In 26 of them, deep sensitivity of the upper limbs was impaired as a result of demyelinizing diseases of the central nervous system and other organic involvements of the spine. In 7 patients, neurological examinations failed to find any disorders of deep sensitivity. Control group consisted of 38 volunteers: 21 men and 17 women aged 25-56 years.

A new method for selective stimulation of kinesthetic afferents of the upper limb was used for KEP recording. The method consisted in passive 50° bending of the hand in the radiocarpal joint with angular acceleration 350 rad/sec^2 [2]. Stimulation was carried out at accidental intervals every 2-4 sec. Responses were recorded by the monopolar method in both hemispheres using cup-like chlorosilver electrodes attached to the skin of the head by collodium. Active electrodes were placed at a distance of 7-8 cm from the median sagittal line and 2 cm behind the line connecting the vertex with external acoustic meatus.

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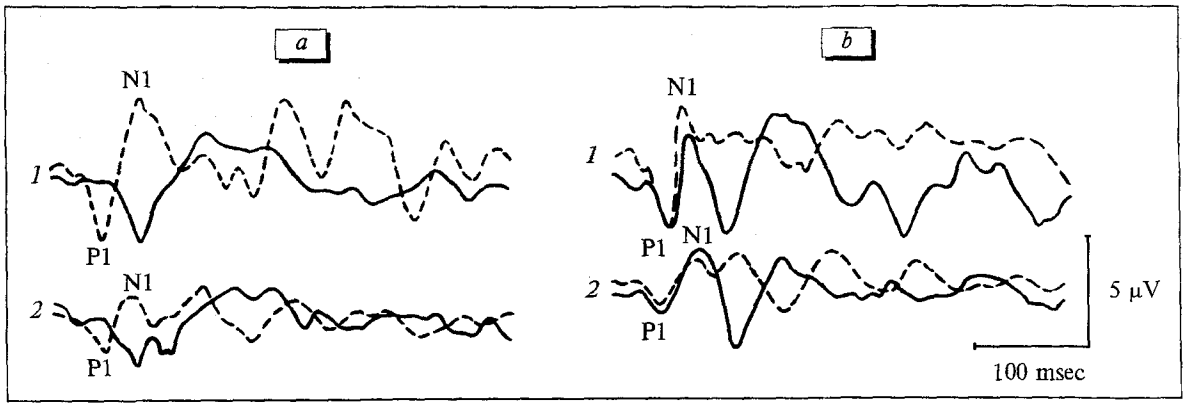

Fig. 1. Contra- (*1*) and ipsilateral (*2*) kinesthetic evoked potentials recorded in a patient with right-sided hemihypesthesia (continuous line) and in a healthy control (interrupted line) during stimulation of the right (*a*) and left (*b*) hand.

Indifferent electrodes were placed on the mastoid processes of the temporal bones. KEP registration and averaging were carried out in the 1.5-3000 Hz frequency band by Medelec Sensor and Polygraph System devices. Up to 100 individual responses were averaged with 500 msec analysis epoch. Peak latency was determined starting from the moment of stimulation. The results were processed using Student's *t* test.

RESULTS

Notable changes of KEP were revealed in patients with impaired deep sensitivity. Figure 1 shows the kinesthetic responses of a patient with impaired deep sensitivity presenting as right-sided hemihypesthesia. KEP did not differ from the norm during stimulation of kinesthetic afferents of the left hand. During stimulation of the right hand afferents, the LP of the first positive P1 component increased to 85 msec (*vs.* normal 48.6±2.4 msec) and of the second negative component N1 to 135 msec (*vs.* normal 83.5±5.1 msec). Statistical analysis of the amplitude-time KEP parameters (Table 1) showed a significant increase in LP of their early components P1 and N1 ($p< 0.001$) in both hemispheres in patients with impaired kinesthetic sensitivity *vs.* normal controls, but no differences between the amplitudes ($p>0.1$). An increase in the KEP early components LP reflects pathoanatomy of demyelinizing diseases, whhich is characterized by the priority involvement of well-myelinized rapidly conducting group A nerve fibers transferring proprioceptive information. These fibers belong to the lemniscus system and are triggered at the specific sensory relay of the thalamic postero-lateral nuclear complex. A greater increase in the LP of early components of evoked responses reflects a greater impairment of deep sensitivity. Change in the KEP early components LP permits quantitatively assess the degree of involvement and can serve as the diagnostic and prognostic sign in such diseases.

On the other hand, prolongation of LP of early components of kinesthetic P1 responses to 62 msec and of N1 to 110 msec was recorded in some patients without apparent disorders of deep sensitivity (Fig. 2). Such LP changes suggest involvement of the

TABLE 1. Characteristics of the Amplitude and Time Parameters of KEP Early Components in Patients with Impaired Deep Sensitivity (*M±m*)

Characteristics of components	Components			
	P1		N1	
	patients (*n*=26)	controls (*n*=33)	patients (*n*=26)	controls (*n*=33)
Contralateral hemisphere				
Amplitude, μV	3.9±0.6	4.9±0.3	3.4±0.5	3.5±0.3
Latent period, msec	71.3±8.1*	46.2±2.6	104.3±14.2*	81.1±5.2
Ipsilateral hemisphere				
Amplitude, μV	2.6±0.5	2.5±0.25	2.2±0.6	2.1±0.25
Latent period, msec	73.6±8.4*	48.3±2.8	111.2±15.1*	82.4±6.1

Note. *$p<0.001$ *vs.* the control.

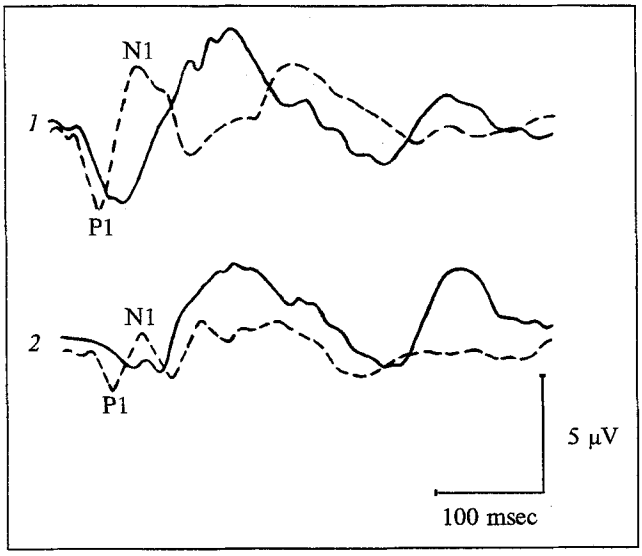

Fig. 2. Contra- (*1*) and ipsilateral (*2*) kinesthetic evoked potentials recorded in a patient in whom neurological examination revealed no disorders of deep sensitivity (continuous line) and in a healthy control (interrupted line).

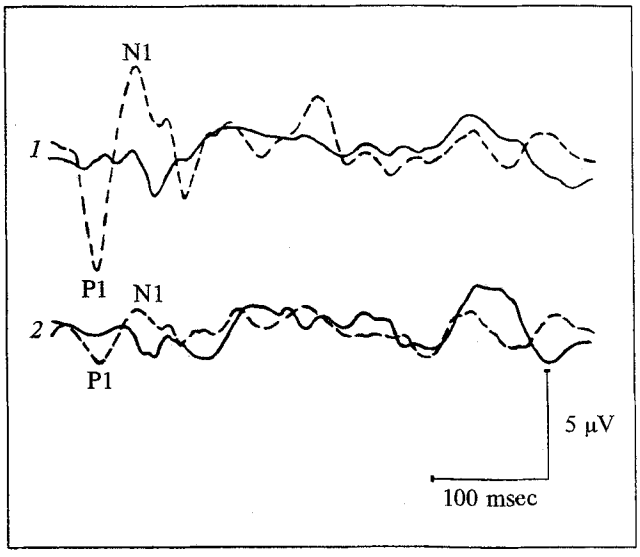

Fig. 3. Contra- (*1*) and ipsilateral (*2*) kinesthetic evoked potentials recorded in a patient with right-sided anesthesia (continuous line) and in a healthy control (interrupted line).

specific afferent systems responsible for the kinesthetic sensitivity conduction at this stage of disease. These data demonstrate the possibility of an early diagnosis of disorders of proprioceptive sensitivity, which cannot be detected by neurological examinations at this stage of the disease, by KEP recording.

Although disappearance of KEP could be expected in some patients with completely lost deep sensitivity, the LP of early component P1 increased to 95 msec and of N1 to 165 msec, while their amplitude decreased to 1.5-1.5 μV (Fig. 3). In such cases we can speak about partial retention of proprioceptive sensitivity, probably, at the expense of kinesthetic information conduction through nonspecific extralemniscus afferent systems of the brain, which confirms again the prognostic value of KEP recording.

Thus, our studies demonstrate that changes of kinesthetic responses in various types and localizations of lesions of the central nervous system leading to impairment of deep sensitivity are peculiar and reproducible, which permits the use of KEP recording as a diagnostic method.

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