PHYSIOLOGY

Kinesthetic and Somatosensory Evoked Potentials in Patients with Parkinson's Syndrome

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In patients with Parkinson's syndrome the early components of kinesthetic evoked potentials recorded from the brain differ significantly from those in healthy subjects, whereas the components of somatosensory evoked potentials do not. The observed differences appear to reflect enhanced pyramidal descending influences on the transmission of inhibitory kinesthetic afferent impulses in order to compensate for the excessive excitation in the extrapyramidal system.

Key Words: *kinesthetic evoked potentials; somatosensory evoked potentials; Parkinson's syndrome; extrapyramidal and pyramidal systems*

Mechanisms by which afferent impulses are transmitted in patients with extrapyramidal disorders of the sensorimotor function constitute an important area of study in neurophysiology and clinical neurology. In their study of evoked potentials (EP) in patients with Parkinson's syndrome, Zenkov *et al.* [4] discovered that the amplitudes of intermediate and late components of these evoked responses were increased as compared to those in normal subjects, whereas the early components remained unchanged. These results were confirmed by Gnezditskii and Arkhipova [1], who found the areas of EP components in parkinsonian patients to be enlarged in the same range of latencies. Other authors, however, did not record any changes in the amplitude or temporal parameters of evoked responses in parkinsonian patients [5].

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The purpose of the present study was to examine, in patients with the akinetic-rigid (Parkinson) syndrome, the kinesthetic EP (KEP) arising in response to selective stimulation of upper limb afferents and the traditionally recorded somatosensory EP (SEP) elicited by transcutaneous electrostimulation of the median nerve.

MATERIALS AND METHODS

A total of 15 parkinsonian patients aged 48-69 years and 19 healthy subjects aged 39 to 62 were examined.

To record KEP, a method of appropriate selective stimulation of kinesthetic upper limb afferents was used, in which one wrist is bent passively at the radiocarpal joint at an angle of 50° with a large angular acceleration (350 rad/sec^2) [2]. For recording SEP, the median nerve was stimulated at the wrist with electrical pulses of 0.1 msec duration at an intensity corresponding to the threshold for a motor response from muscles of the th-

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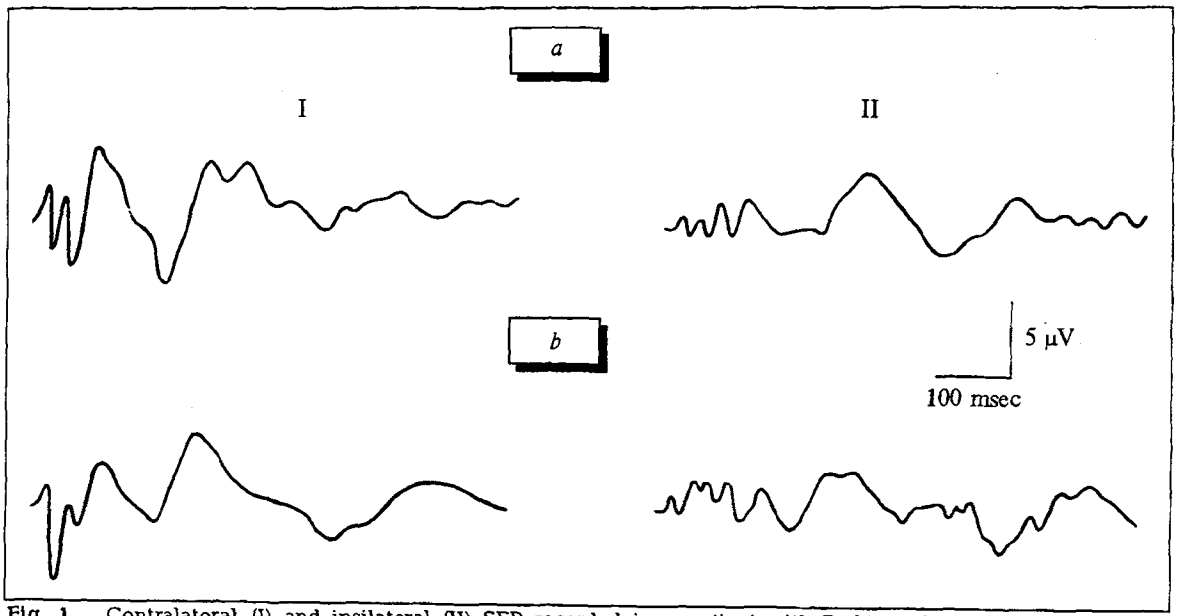

Fig. 1. Contralateral (I) and ipsilateral (II) SEP recorded in a patient with Parkinson's syndrome (*a*) and in a healthy subject (*b*).

enar eminence (≈20 μA). The stimuli were delivered at random intervals once every 2 to 4 sec. The responses were recorded from both hemispheres by means of monopolar cuplike silver-chloride electrodes attached to the scalp with collodion. The "active" electrodes were positioned on the head at a distance of 7-8 cm from the median sagittal line and 2 cm posterior to the line connecting the vertex with the external auditory meatus, while the "indifferent" electrodes were placed on the mammillary processes of the temporal bones. The EP were registered and averaged within a pass band of 1.5 to 3000 Hz in a Polygraph System (Japan); from 64 to 128 solitary responses were averaged with an epoch of analysis equal to 500 msec. Peak latencies were determined from the moment stimulation was started. The results were treated statistically using Student's *t* test.

RESULTS

The SEP recorded from both hemispheres in parkinsonian patients did not differ much in their characteristics from those registered in the healthy

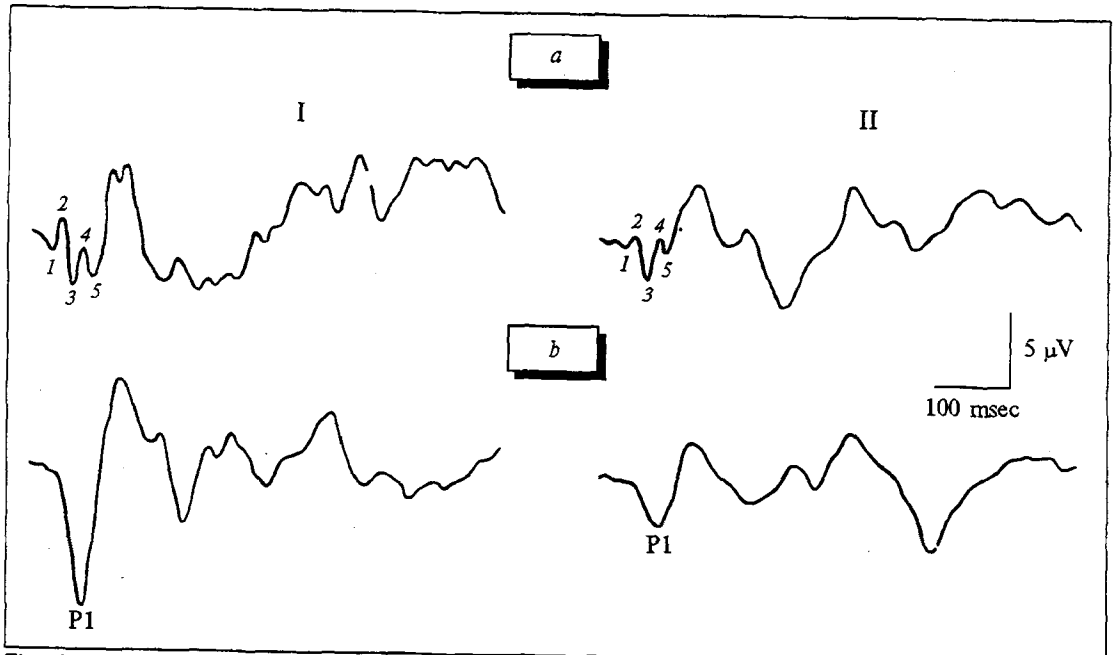

Fig. 2. Contralateral (*I*) and ipsilateral (*II*) KEP recorded in a patient with Parkinson's syndrome (*a*) and in a healthy subject (*b*). Response components: *1*) PO1; *2*) NO1; *3*) PO2; *4*) NO2; *5*) PO3.

TABLE 1. Characteristics of the Early KEP Components in Patients with Parkinson's Syndrome. The Values are Means±SEM; $n = 15$

Characteristic	Component				
	PO1	NO1	PO2	NO2	PO3
	Contralateral hemisphere				
Amplitude, μV	1.4±0.3	1.6±0.4	2.9±0.7	2.2±0.7	1.9±0.6
Latency, msec	25.2±3.3	30.9±3.5	38.7±3.9	54.7±6.1	67.4±9.2
	Ipsilateral hemisphere				
Amplitude, μV	0.6±0.3	0.8±0.3	1.9±0.5	1.6±0.6	1.1±0.5
Latency, msec	26.4±4.4	32.7±4.9	42.4±5.2	56.3±7.6	69.8±9.9

subjects (Fig. 1). The difference between these two groups in the mean amplitudes and latencies of SEP was statistically insignificant ($p>0.1$). In contrast, the KEP recorded in the patients from both hemispheres (contralateral and ipsilateral relative to the stimulation site) showed several differences from those in the healthy subjects, the main differences being found for the early components of these responses. Thus, instead of only one positive component, P1, which in the healthy subjects had an amplitude of 5.2±0.3 μV and a latency of 44.8±2.4 msec in the contralateral hemisphere and 3.4±0.2 μV and 46.3±2.6 msec in the ipsilateral, a complex of low-amplitude positive-negative oscillations, PO1-NO1-PO2-NO2-PO3, was recorded from both hemispheres in an interval of 25 to 70 msec (Fig. 2). The amplitudes and latencies of these components are shown in Table 1.

The question arises as to why kinesthetic evoked responses are altered in patients with Parkinson's syndrome. There is a large body of evidence in the literature indicating that corticofugal inhibitory influences are exerted on the transmission of afferent impulses and in particular on the evoked activity of various structures of the cutaneous motor analyzer [5-8, 10]. It is also known that phasic movements and postural tonus are controlled by two distinct functional systems - pyramidal and extrapyramidal - that are, in a certain measure, in reciprocal relationships [3]. In parkinsonism, which is a syndrome involving abnormal functioning of the extrapyramidal system, the content of dopamine in the caudate nuclei is reduced [9], so that processes of cholinergic excitation predominate in the striatal system and the characteristic symptoms such as rigidity and tremor develop as a result. This suggests that the changes we observed in the early KEP components reflect enhanced pyramidal descending inhibitory influences that can partially block the kinesthetic afferent impulse traffic so as to compensate for the excessive excitation in the extrapyramidal system of patients with Parkinson's syndrome.

The results of this study permit us to conclude that the recording of kinesthetic evoked responses provides a more refined and appropriate method for the study of somatosensory functions in patients with extrapyramidal disorders than the recording of somatosensory evoked responses.

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