GENERAL PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY

Pathological Forms of Brain Electrical Activity and Localization of Its Sources in Patients with Trigeminal Neuralgia

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No significant differences from the typical electroencephalogram were observed in patients with trigeminal neuropathy. In patients with typical trigeminal neuralgia, the electroencephalogram variations were detected both in the changes of dominant activity and in the appearance of individual pathological phenomena. The three-dimensional localization of pathological activity generators points to the involvement of the median nonspecific brainstem structures into pathological process evoked by trigeminal neuralgia.

Key Words: *trigeminal neuralgia; electroencephalogram*

Irrespective of the nature of the initiating factor of the paroxysmal trigeminal neuralgia syndrome, the pathological basis of its realization is the pathoalgetic system formed under the action of pathological superactive neurogenerator in the trigeminal nerve system [4]. Pathological discharges produced by this generator and conveyed via specific and nonspecific conducting pathways modify the spontaneous and evoked bioelectrical cerebral activity [5,6]. There are only occasional studies analyzing spontaneous cerebral bioelectrical activity under these pathological conditions [7]. The modern computerized technique of electroencephalogram (EEG) processing makes it possible to perform simultaneous multichannel recording and processing of EEG with the help of spectral and correlation analyses, as well as to determine the three-dimensional location of the EEG-revealed sources of pathological activity [1].

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The aim of this work was to analyze EEG variations using modern computerized techniques in order to identify pathophysiological mechanisms in patients with various forms of trigeminal pathology.

MATERIALS AND METHODS

Two groups of patients with trigeminal pathology (12 men and 28 women) aged 28-61 years were examined. The first group consisted of 12 patients with persistent pain syndrome (10 of them with dental plexalgia and 2 with postherpetic ganglioneuritis). Twenty-eight patients in the second group had typical trigeminal neuralgia with paroxysmal pain syndrome lasting from 2 months to 5 years. Control group consisted of 12 healthy volunteers which had no symptoms of neural diseases. The patients were examined both in the acute period and during partial remission. EEG recording was performed by a standard electrode layout 10-20. The signals were amplified and fed into computer via a digitizer. They were analyzed with the help of EEG-processing soft-

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ware [1]. To determine the location of the bioelectrical activity sources, the rectangular reference system was used, in which x, y, and z axes were directed along, respectively, 1) the line going from the occipital tuber to the nose bridge, 2) the line connecting the external auditory meatuses and 3) the vertical line directed from basis to vertex. The coordinates of the bioelectrical activity sources were calculated with the help of algorithms described elsewhere [1].

RESULTS

The basic EEG characteristics in the control group agree with the literature data [2]. The occurrence of α-rhythm with amplitude of 85 μV and frequency of 8-12 Hz was recorded predominantly in the occipital leads was high in the control volunteers who were in the resting awareness state with closed eyes. In 8 volunteers, the α-rhythm had a spindle-like structure. The slow components of EEG (θ- and δ-activity) were observed predominantly in the frontal lobe leads as single oscillations with amplitude of no more than 30 μV. In 6 volunteers, there was a β-rhythmic high-frequency activity with frequency of 15-18 Hz and amplitude of no more than 15 μV. The basic EEG characteristics in the first group of patients with persistent pain syndrome were similar to that of the control group. Only two patients demonstrated a small decrease (by 5-15%) in the α-rhythm amplitude mainly in the sincipital and central leads in the hemisphere that was contralateral to pain localization compared with the leads in the ipsilateral hemisphere.

In contrast to the control and the first patient groups, the patients of the second group had certain disturbances in the cerebral bioelectrical activity, manifested both in variation of the character of dominant activity and in the appearance of individual pathological phenomena. Alterations of the background EEG were observed as variations of the frequency-amplitude characteristics of the dominant activity and as the disturbances of zonal EEG differences. Thus, in 7 patients of the second group the

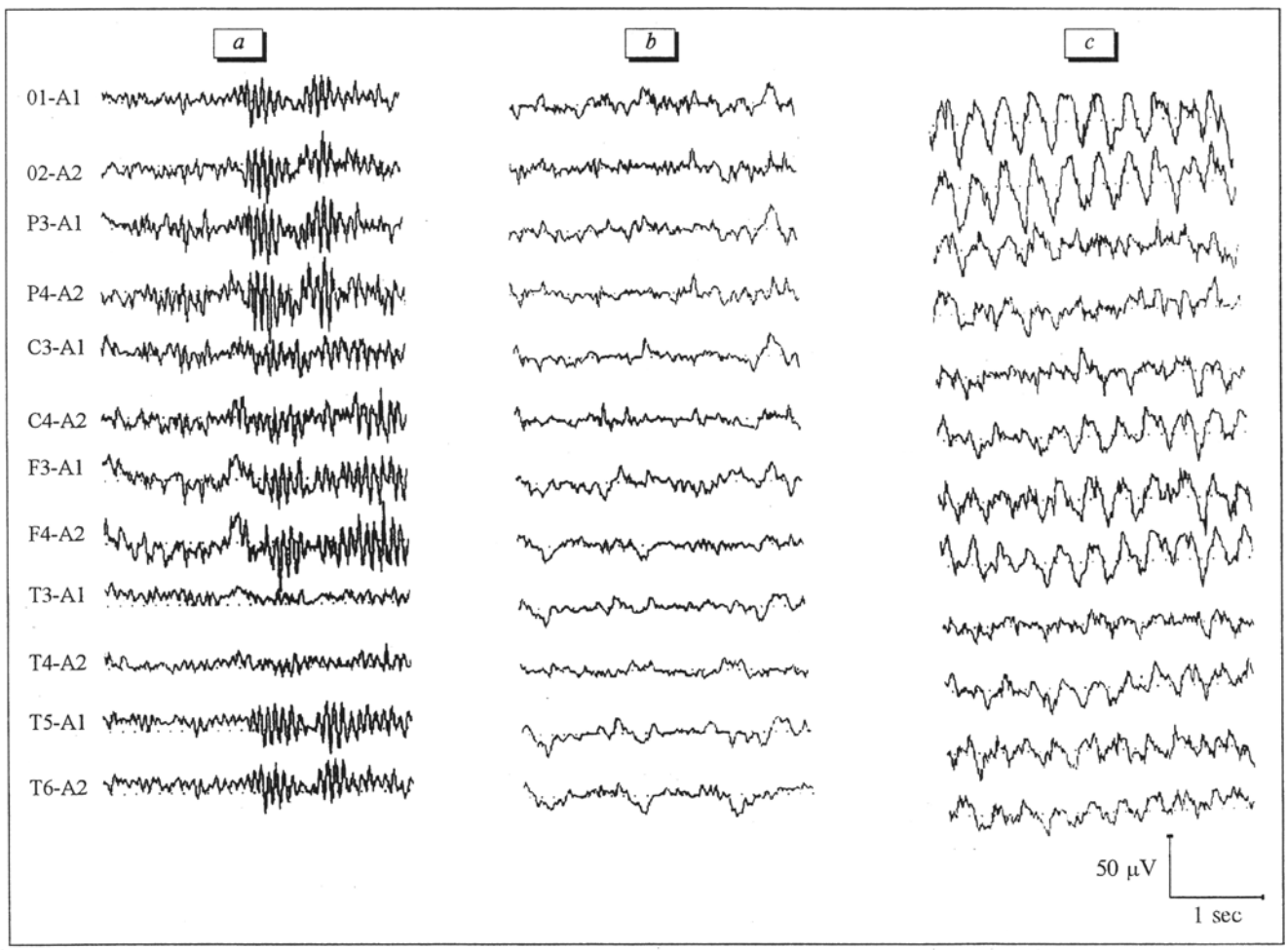

Fig. 1. Changes in the EEG dominant activity in patients with trigeminal neuralgia. *a*) an increase in the frequency of dominant activity to 14-15 Hz; *b*) "flat" low-frequency EEG; *c*) a decrease in the frequency of dominant activity. The notation corresponds to the 10-20 system.

"flat" low-amplitude EEG was recorded, which in all the leads was manifested as polymorphic activity with amplitude less than 25 μV (Fig. 1, *b*). The zonal differences were virtually absent, and there was no pronounced light-evoked activation reaction. In comparison with the control group, a significant increase in the β-rhythm power ($p<0.05$) was observed in these patients. Such variations may indicate dysfunction of the synchronizing mechanisms [2].

In 12 patients of the second group, there were variations in the dominant activity frequency. In 11 patients, the dominant activity corresponded to the β_1-frequency range of 14-17 Hz, which according to functional features could be related to as the fast mode of α-rhythm. Blockade of this activity by opening the eyes and photostimulation, as well as its transformation into the α-like spindles, seems to support this hypothesis (Fig. 1, *a*). One female patient had the dominant δ-activity with a frequency of 3 Hz, and although by functional criteria this activity could also be attributed to the slow mode of α-rhythm [2], its occurrence was low: one per 3000-4000 cases (Fig. 3, *c*). Transformation of the functional activity is not a pathological symptom, though the enhanced occurrence of such transformations in patients with typical trigeminal neuralgia points to the influence of a pathological process on the cerebral structures related to α-rhythmogenesis.

In 8 patients of the second group, the dominant activity was of the α-range, although in comparison with the control group it also demonstrated notable differences. In most cases, the α-rhythm was not structured into spindles, and α-oscillations had a spike-like shape (Fig. 2, *a*). The EEG spectrogram of these patients showed disturbances in zonal distribution of α-activity. The maximum power of this activity was observed not in the occipital lead (as in normal subjects) but in the centrosincipital lead, and it was more pronounced ipsilateral to the pain-affected side of the head (Fig. 2, *b*).

Against the background of described variations of the dominant activity, EEG in 16 patients of the second group demonstrated individual pathological phenomena manifested as: 1) the bursts of spike

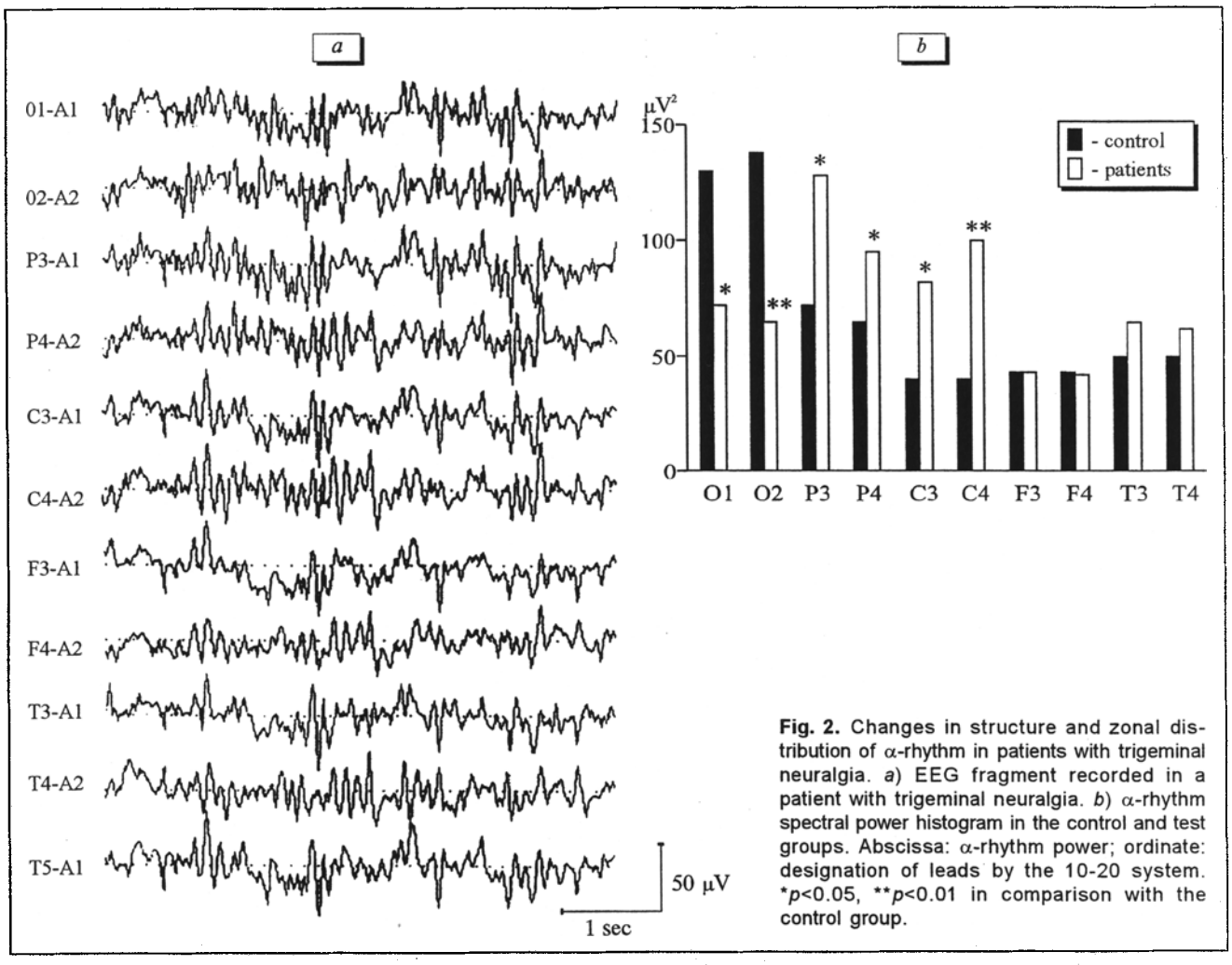

Fig. 2. Changes in structure and zonal distribution of α-rhythm in patients with trigeminal neuralgia. *a*) EEG fragment recorded in a patient with trigeminal neuralgia. *b*) α-rhythm spectral power histogram in the control and test groups. Abscissa: α-rhythm power; ordinate: designation of leads by the 10-20 system. *$p<0.05$, **$p<0.01$ in comparison with the control group.

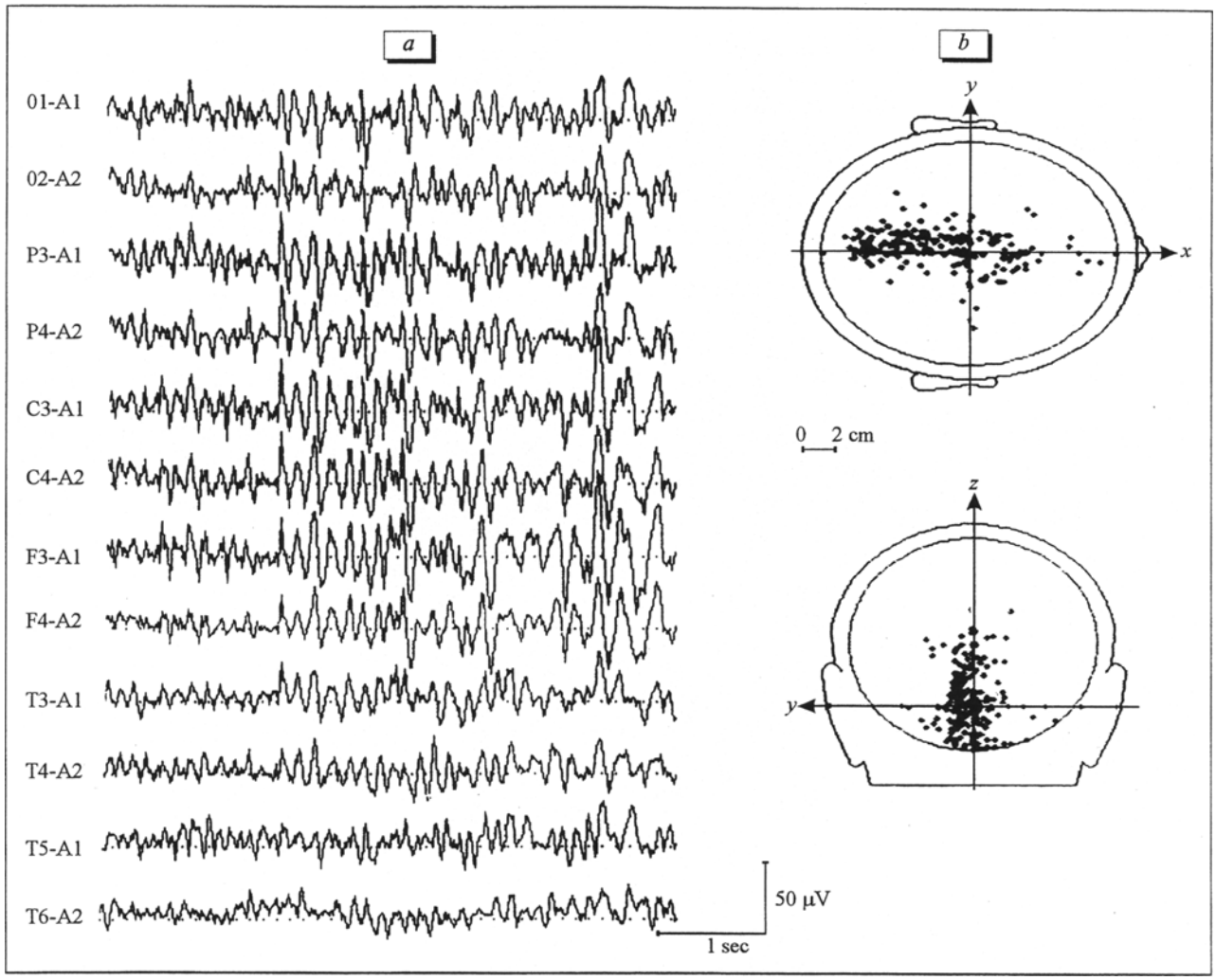

Fig. 3. EEG fragment recorded in a patient with trigeminal neuralgia who had bilateral synchronous bursts of θ- and δ-activity (*a*) and three-dimensional location of the corresponding sources (*b*).

oscillations in the frequency band of 13-18 Hz, which exceeded by 20-50% the amplitude of the background activity; 2) bilateral synchronous bursts of δ- and θ-waves, which were more pronounced in the central leads (Fig. 3, *a*); and 3) the complexes of fast wave—slow wave type. These signs may testify to pathological discharges in nonspecific median structures of the brainstem as well as to the convulsion threshold decrement, but functional loads with hyperventilation and rhythmic photostimulation did not lead to any notable increase in the intensity of these pathological signs. An attempt was made of the three-dimensional localization of this activity.

Among 16 patients of the second group, the phenomena similar to the bursts of the sharp high-amplitude β-oscillations and to the fast—slow wave complexes were found only in 8 cases. In 3 patients, these EEG phenomena occurred 0.5-1.5 sec before both the patient's report of pain appearance and the EEG motion artifact indicating a routine paroxysm. Only in 4 out of 8 patients who had EEG with these phenomena it was possible to localize the source of such an activity with a significance of 90%. In most cases the generator of this activity was located within the bottom subdivisions of the brainstem central part, either bilateral or contralateral to the pain localization.

The bursts of bilateral synchronous δ- and θ-waves were found in 9 patients. These phenomena often occurred 0.5-1 sec before the onset of pain paroxysm (Fig. 3, *a*). In 6 patients, the location of the source of this activity was determined with the significance of 90%.

Most frequently the generator of this activity was located in the nonspecific median brainstem structures contralateral (4 cases) or bilateral (2 cases) to pain localization (Fig. 3, *b*). In all other cases, the source of pathological activity could not be reliably localized (the respective location points were diffusely distributed over the entire cerebral conductor or significance of location coordinates was <80%).

Presumably, this is related to the specific algorithm of the software which is used to localize the bioelectrical activity sources. In fact, the recording of EEG from 12-16 channels provides localization of only one generator. However, if the bioelectrical activity recorded in EEG originates from two or more generators, the three-dimensional model results in only some average distribution of electric activity sources, which does not clearly show the locations of the corresponding generators [1]. According to the theory of generator and system mechanisms [4], the generator corresponding to trigeminal neuralgia and located in the brainstem nuclei of the trigeminal system, can induce formation of secondary pathological generators at higher cerebral levels. Therefore, when generators of different levels are working simultaneously, it is not always possible to localize the pathological activity sources. In addition, this activity can manifest itself in different phenomena, but in all such cases the involvement of the nonspecific median brainstem structures into the pathological process can be assumed.

In patients with trigeminal neuropathy related to disturbances in the peripheral subdivisions of the trigeminal system, the pathological process does not affect markedly the entire cerebral functional activity.

In contrast to the patients with trigeminal neuropathy, all trigeminal neuralgia patients demonstrated profound alterations in EEG. In 40% of these patients, it was possible to localize the corresponding generators. The data obtained show that the pathological algesic system incorporates the nonspecific median brainstem structures in patients with typical trigeminal neuralgia.

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