PHYSIOLOGY

Analysis of Changes in the Electroencephalogram Power Spectrum on a New Model of Brain Ischemia in Rats

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UDC 615.22.015.4.07

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny,* Vol. 118, № 12, pp. 565-567, December, 1994
Original article submitted March 9, 1994

The bioelectrical activity is studied in the left and right parietal cortex by recording the power spectrum of the electroencephalogram in brain ischemia caused by complete ligation of the left common carotid artery and 50% reduction of the blood flow in the right common carotid artery in experiments carried out on nonnarcotized Wistar rats. Ischemia results in marked and stable disorders in the bioelectrical activity manifested in a decrease of the total EEG power, depression of the dominating frequency in the Θ-range, increase of the δ-range power, and interhemispheric asymmetry of some spectrogram parameters.

Key Words: *electroencephalogram; brain ischemia*

The magnitude and nature of electroencephalogram (EEG) changes in cerebrovascular disorders correlate with the clinical state of patients [5,7], making it possible to assess the degree of functional disorders of the brain according to the change of EEG parameters. This is especially important in experiments carried out on animals, where it is impossible to assess accurately the severity of damage to brain functions according to neurological symptoms [12]. In view of this and of the possibility of using certain EEG parameters as markers of the degree of insufficiency of blood supply to the brain, the aim of the present study was to perform dynamic examination of the EEG power spectrum in the brain cortex of alert rats using a new model of brain ischemia.

MATERIALS AND METHODS

Experiments were carried out on 12 alert male Wistar rats weighing 200-250 g. For EEG recording, Nichrome electrodes were implanted 6-7 days prior to the experiment in the region of the parietal cortex of the right and left hemispheres in anesthetized animals (sodium ethaminal, 50 mg/kg i.p.). Brain ischemia was induced in 6 rats under ether anesthesia by ligation of the left common carotid artery and by a 50% reduction of the blood flow in the right common carotid artery (under MFV-1100 blood flow rate control). Only isolation of the carotid arteries was performed in 6 sham-operated rats.

EEG recording and spectral analysis were performed using an O.T.E. Biomedica neurophysiological kit. The epoch of the analysis was 248 sec with time constant 0.03 sec. The values of the relative total power of the EEG spectrum were calculated as well as the powers of individual ranges (0.5-4.0 for δ, 4-8 for Θ, 8-12 for α, and 12-22 Hz for β_1) and the changes of these indexes as compared to the initial values. Results were processed statistically using the Student *t* test.

RESULTS

The initial values of the relative power in individual ranges (27-28 for δ, 41-43 for Θ, 14 for α, and 11-

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0007-4888/94/0012-1257$12.50

TABLE 1. Changes of Relative Power of Total Power Spectrum Ranges (Σ) of EEG (%) in Rats after Brain Ischemia

Hemisphere	Range	Initial level	Period of ischemia, days					
			1	2	3	4-5	7	10
Left								
	δ	26.3±1.7	−9.3±2.7*	−13.5±3.4*	−8.6±4.8	−15.3±3.3*	−15.7±2.2*	−18.8±2.6*
	Θ	43.0±4.0	−25.7±4.1*	−26.3±7.6*	−28.6±3.1*	−30.1±5.4*	−31.3±4.9*	−31.7±4.5*
	α	13.3±2.1	−1.9±1.1	−2.7±2.8	−5.2±2.5	−3.6±1.9	−7.9±1.1*	−5.7±1.1
	β_1	10.3±0.3	−0.1±1.2	−0.6±2.0	−1.8±2.7	−0.1±0.5	−4.7±2.0	−3.4±2.2
	Σ	94.3±2.9	−36.6±4.3*	−42.6±15.2*	−44.5±6.1*	−54.4±8.8*	−60.1±6.1*	−60.0±5.8*
Right								
	δ	26.8±1.1	−8.3±2.1*	−10.8±3.6*	−4.7±5.5	−9.9±5.1	−10.8±3.4*	−15.0±4.0*
	Θ	43.5±2.3	−21.2±4.0*	−21.8±6.9*	−22.2±5.6*	−20.5±8.5*	−25.1±6.5*	−26.9±5.4*
	α	14.3±2.7	+1.5±2.5*	−1.5±2.1	−2.5±4.4	−1.8±3.4	−5.7±3.1	−3.7±2.6
	β_1	12.5±2.7	+0.9±2.3	−0.2±1.5	−0.1±4.7	−1.6±4.0	−4.0±4.2	−2.5±3.8
	Σ	99.5±3.1	−26.7±6.8*	−34.0±13.2*	−29.7±15.6	−33.8±20.3	−46.5±15.3*	−49.5±13.1*

Note. An asterisk denotes $p<0.05$ as compared to the initial level.

16% for β_1) in sham-operated rats corresponded to the values in the group of test rats (Table 1). Such a nature of EEG spectral power is in agreement with the data of other authorities [6,10], who have noted the domination of the Θ-range activity in animals. Moderate aftereffects of the operative trauma were found in sham-operated animals namely, a short-term (just on the 1st day) decrease of the total power (by 23 and 19% in the left and right hemispheres, respectively). On the 4th-5th and 10th day the total power rose moderately (by 10-17%), but the correlation of the powers of individual ranges remained stable during the whole period of observation (10 days) (data not presented).

Brain ischemia resulted in marked changes of the EEG (Tables 1 and 2). The most regular were the decrease of the total EEG power, lowering of the Θ-range power, increase of the δ-range portion in the spectrum, and the greater magnitude of the pathological shifts in the cortex of the left, more afflicted, hemisphere. The described shifts were stable but modulated by transient changes in individual ranges of the EEG frequency spectrum that might have been due to both compensatory and pathological reactions induced in the brain tissue by the acute ischemia. Thus, the significant rise of the $\alpha+\beta_1/\Theta$ coefficient on the 1st and 2nd day in both hemispheres evidently attests that disorders in functional activity do not attain any marked intensity in the early period of ischemia, and therefore the brain is able to react with an enhanced excitation in response to hypoxia. The existence of a phase of EEG desynchronization in the initial period of hypoxia is confirmed on the model of unilateral insult in dogs [10]. The rise of the power of the α_1- and β_2-ranges of the spectrogram in patients with ischemic insult in the early periods is related to the compensatory hyperactivation of the ascending reticular systems [3].

The total EEG power fell on the 3rd day by 29.7 and 44.5% in the right and left cortex, respectively; δ-activity dominated in the cortex of both hemispheres and its absolute power was close to the initial (Table 1). This is probably related to the maximal degree of brain tissue edema in this period [1], as is confirmed by the close correlation ($r=0.81$, $p<0.05$) between the changes of the δ-range power and the values of active resistance in the left-hemisphere cortex. The latter is in good agreement with clinical observations on the

TABLE 2. Changes of Coefficients of Asymmetry in the Power of Spectrum Ranges and Total Power (Σ) of EEG in Rats after Brain Ischemia

Index	Initial level	Period of ischemia, days					
		1	2	3	4-5	7	10
δ_s/δ_d	0.98±0.40	- 0.07±0.05	- 0.15±0.02	- 0.2±0.05*	- 0.26±0.05*	- 0.26±0.07	- 0.25±0.07
Θ_s/Θ_d	0.98±0.06	- 0.19±0.06*	- 0.20±0.06*	- 0.23±0.10	- 0.33±0.10*	- 0.28±0.09*	- 0.22±0.10
α_s/α_d	0.97±0.06	- 0.25±0.08*	- 0.16±0.10	- 0.20±0.11	- 0.25±0.09*	- 0.26±0.10*	- 0.11±0.12
β_{1s}/β_{1d}	0.88±0.06	- 0.11±0.05	- 0.10±0.06	- 0.18±0.08	- 0.24±0.07*	- 0.16±0.9	- 0.11±0.12
Σ_s/Σ_d	0.96±0.05	- 0.15±0.05	- 0.16±0.06	- 0.20±0.09*	- 0.29±0.08*	- 0.25±0.09*	- 0.17±0.10

Note. An asterisk denotes $p<0.05$ as compared to the initial level; s refers to the left side, d to the right side.

interrelation between the aggravation of the state of ischemic insult patients, features of brain edema, and the increase of the δ-range power [3]. The period of the maximal of asymmetry in spectrogram indexes (the 4th-5th day after ischemia) coincides with the end of the period of the stable reduction of the circulation, after which the blood supply of the brain tends to be restored [5]. However, calculation of the correlation between the changes of local brain circulation and the main indexes of the EEG power spectrum in the postischemic period did not point up any regularity in a single case. This may be further proof of the phenomenon of disconnection of the circulation and the functional activity of the brain shortly after acute ischemia [9].

Thus, gross bilateral functional disturbances attesting to marked damage of the brain tissue in ischemia were stable during the whole period of observation, despite the previously noted tendency toward restoration of the local brain circulation from the 7th day after ischemia [1]. Analogous marked changes of the total power of individual ranges of the EEG spectrum are demonstrated in a 2-hour occlusion of 4 magistral arteries and unilateral embolic insult in dogs [10,11]. A diffuse lowering of the brain's electroproduction with an increase of the relative magnitude of pathological slow-wave activity is a reliable criterion of brain ischemia [5,8]. Together with this, the totality of the findings regularly reproduces the noted shifts in spectrograms only in patients with ischemic insult in the carotid system [3]. Bilateral depression of the total spectrum power with features of asymmetry was found in the majority of patients (in 18 out of 20) together with a redistribution of the power of individual ranges in the total spectrum due to a lowering of the power of the dominant α-range and an increase of the power of slow-wave Θ- and, particularly, δ-activity. In another kinds of damage to the blood circulation the spectrograms and other EEG indexes either differed significantly from the above, or showed no regular pattern of changes [2,3,5,8].

From the analysis of the EEG power spectrum it may be concluded that the proposed model of brain ischemia in rats corresponds to the ischemic insult developing due to thrombosis or embolism of the carotid artery. The sable, long-lasting disorders of brain functional activity permit us to recommend using this model of ischemia to study the pathogenesis of ischemic damage to the brain and the protective effect of new preparations.

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