Intern. J. Neuroscience, 114:977–997, 2004

ISSN: 0020-7454 / 1543-5245 online
DOI: 10.1080/00207450490450154

Taylor & Francis
healthsciences

SIMULTANEOUS RECORDING OF EEG AND DIRECT CURRENT (DC) POTENTIAL MAKES IT POSSIBLE TO ASSESS FUNCTIONAL AND METABOLIC STATE OF NERVOUS TISSUE

S. E. MURIK

Irkutsk State University
Irkutsk, Russia

A. G. SHAPKIN

Irkutsk State Medical University
Irkutsk, Russia

It has been proposed to assess functional and metabolic state of the brain nervous tissue in terms of bioelectrical parameters. Simultaneous recording of the DC potential level and total slow electrical activity of the nervous tissue was performed in the object of study by nonpolarizable Ag/AgCl electrodes with a DC amplifier. The functional and metabolic state of the brain was determined in terms of enhancement or reduction in the total slow electrical activity and positive or negative shifts in the DC potential level.

Keywords cyclopentyladenosine, electroencephalogram, DC potential, functional and metabolic state of the nervous tissue, ischemia, Nembutal, steady potential

Received 26 September 2003.

Address correspondence to S. E. Murik, Physiology Dept., Irkutsk State University, Sukhe-Bator St. 5, Irkutsk 664003, Russia. E-mail: murik@ic.isu.ru

One of the problems of neurology and neurophysiology is the development of efficient minimal invasive methods of assessment of the functional and metabolic state of the nervous tissue. There are not very many available methods (Demchenko, 1976; Rusinov, 1987; Buchsbaum et al., 1989) that make it possible to diagnose precisely the state of the brain nervous tissue in a wide range of adaptive responses of the organism and at the same time being convenient for both experimental and clinical use.

One of the characteristics of the functional state of the nervous tissue is its bioelectrical activity in the EEG form (Zhirmunskaya et al., 1977; Rusinov, 1987; Zhirmunskaya, 1989). However at present there is no unanimous opinion about diagnostic potentials of this method. Similar EEG changes are often found in different psychophysiological states (attention, emotions, sleep, and fatigue). At the same time identical effects can result in different EEG responses. Thus, according to some data hypoxia and ischemia are followed by increasing power of the rhythms, particularly in delta- and theta-ranges (Von der Drift & Kok, 1973; Kauser-Gatchalian & Neundörfer, 1980) or increasing beta-activity (Daniyarov & Vilenskaya, 1986) and by depression of the EEG according to other evidence (Kauser-Gatchalian & Neundörfer, 1980). As a result the use of the EEG method alone is not sufficient for precise identification of the functional and metabolic state of the nervous tissue. However, activation processes in the nervous system are often followed by an increase in the index of rapid rhythms, and the EEG method is used for estimation of the functional state of the brain (Ingvar et al., 1976).

The recording of the steady potential level also called DC potential is often used to study the development of such unfavorable states of the brain as ischemia (Rogers et al., 1990; Mies et al., 1993). According to present opinions the DC potential reflects relatively stable changes of potential of the rest of the membrane of neuron and glia cells (Rebert, 1978; Elbert, 1993; Marczynski, 1993). The resulting development of depolarization processes causes a negative shift of the DC potential. Numerous experimental data obtained at present indicate that the level of the membrane potential of neurons closely correlates with neurons' metabolic state (Ingvar et al., 1976; Yanvareva & Kuzmina, 1985; Rogers et al., 1990; Li, J. et al., 2000). In particular, a deficit in macroergs causes disturbances

in ionic homeostasis resulting in membrane depolarization. Therefore, the level of the membrane potential of neurons can serve as an indicator of their metabolic state. However, this method is not acceptable for performing precise functional diagnostics. Ultimately there are no practical methods at present for simultaneous assessment of both metabolic and functional state of the nervous tissue with a sufficient accuracy.

One of the approaches to solve this problem can be parallel recording of the DC potential level as an indicator of polarization processes and the EEG as a characteristic of the functional activity. The simultaneous recording of the EEG and DC potential of the nervous tissue could specify as well the character of functional and metabolic processes proceeding during various adaptive responses of the organism.

The aim of the present work was to study diagnostic potential of the method of simultaneous recording of the EEG and DC potential for assessment of the functional and metabolic state of the brain during development of brain ischemia of different severity, Nembutal narcosis, and intracerebroventricular injection of cyclopentyladenosine (CPA).

METHODS

The experiments were performed on nonpedigreed white rats. The EEG and DC potential were simultaneously recorded with one and the same electrode by a unipolar method with a 4-channel DC gauge amplifier of 1 MOm input resistance and 0–40 Hz band frequency. An indifferent electrode was attached to the nasal parts of the skull. Nonpolarizable Ag/AgCl wire electrodes with a tip diameter of 0.25 mm were used for experiments. Bioelectrical potentials were digitized with 100 Hz frequency and were entered into the computer for further mathematical processing. Successive nonartefact regions of EEG of 1 s duration were used to analyze rhythms and DC potential. The values of the DC potential were obtained by averaging EEG potentials during these periods. The rhythm spectrum and its power were determined with the "Fourier" transformation. The values of the rhythm power spectrum were neutralized according to 5 ranges:

delta-1—0.2–1 Hz, delta-2—1–4 Hz, theta—4–8 Hz, alpha—8–13 Hz, and beta—13–32 Hz rhythm.

To assess the functional and metabolic state of the nervous tissue by the authors' method, the DC potential and EEG were studied in the first series of the experiments by modeling acute circulatory ischemia of different severity. The brain ischemia was developed by bilateral occlusion of both common carotids and intravasal occlusion of the left internal carotid and median brain arteries. The ischemia was modeled by the two ways in the same rats during a single experiment in succession. Recording electrodes were preliminarily implanted under skull bones above the frontal and parietal cortex of the right hemisphere (RH) and the left hemisphere (LH) in the rats of either sex (n = 2) of 150–200 g weight under Nembutal narcosis. The electrode withdrawals were attached to the skull with quickly hardening plastic. Two to three days later the rats were operated on under Nembutal narcosis (40 mg/kg) to model the ischemia. The bioelectrical activity of the brain was started and recorded before narcosis and continued during the whole experiment.

The first model of ischemia ("Ischemia-1") consisted in binding up both common carotids and limiting blood flow in Willis artery vessels. The duration of the isolated effect of this model of ischemia was 20 min, whereupon an original occluder (Sufianova, 2001) being able to self-accumulate (at the expense of swelling in a liquid medium) was additionally inserted in the median brain artery (MBA) of the LH of the rats up to complete cutting off the break of the vessel (Ischemia-2). Combined modeling of the two kinds of circulatory ischemia was performed during 60 min, whereupon the occluder was removed and the recording of the DC-potential and EEG was performed for the next 16 min.

In a particular series of the experiments (n = 10) the DC potential and the EEG rhythm power were studied in the frontal and parietal cortex of the RH and LH following the intracerebroventricular injection of cyclopentyladenosine (CPA). CPA (25 μg/kg) was injected under narcosis (40 mg/kg Nembutal) into the median brain ventricle of the RH.

The functional and metabolic state of the nervous tissue was also assessed by the authors' method after injection of Nembutal. The scheme of the experiment was the following: after connection of

plugs with wires from an amplifier to electrode withdrawals attached to the rat head ($n = 22$), the animal was placed into an experimental chamber where biopotentials were initially recorded for 5–10 min after animal calming; then the rat was taken out of the chamber and it was injected intraperitoneally with Nembutal (40 mg/kg) holding it in the hands. Then the rat was returned to the chamber and the biopotentials were again recorded as long as 15–20 min after injection. The localization of electrodes was the same as in the earlier experiments.

All the results were analyzed statistically by the methods of parametric and nonparametric statistics for dependent and independent samplings: Student *t*-test, sign criterion, Wilcoxon test and Wilcoxon-Mann-Whitney test.

RESULTS

Figure 1 shows changes in the EEG power and DC potential level during ischemia modeling by two methods. The binding up of both common carotids ("Ischemia-1") is seen to result in an increase in the EEG power in the frontal and parietal cortex of the LH and RH. The increase in the rhythm power spectrum in all derivations was 14.04 ± 1.75% ($p < .001$) for the whole rat sampling. The highest rise in the amplitude (20.04 ± 3.2%) was observed in the alpha-range, where it increased from 23.71 ± 0.86 to 28.46 ± 1.09 μV ($p < .001$). In case of the theta-rhythm the rise was 16.31 ± 3.15% and it changed from 46.52 ± 2.03 to 54.01 ± 2.08 μV ($p < .01$). The rise in the amplitude for delta-rhythms was 10.61 ± 2.79%, that is, from 106.85 ± 3.96 to 118.38 ± 3.74 μV ($p < .01$). The least changes were recorded for beta-rhythm. Its power increased as little as 9.21 ± 4.33% from 7.31 ± 0.34 to 7.98 ± 0.33 μV. Nevertheless, this increase was statistically reliable and a dependent *t*-test showed differences at $p < .01$. Simultaneously with the increase in the EEG rhythm power a slight negative shift in the DC potential level was observed, which in the end of the period reached 1222.51 ± 290.1 μV ($p < .01$).

An additional insertion of the occluder in the MBA of the LH on the background of the development of the total depression of the

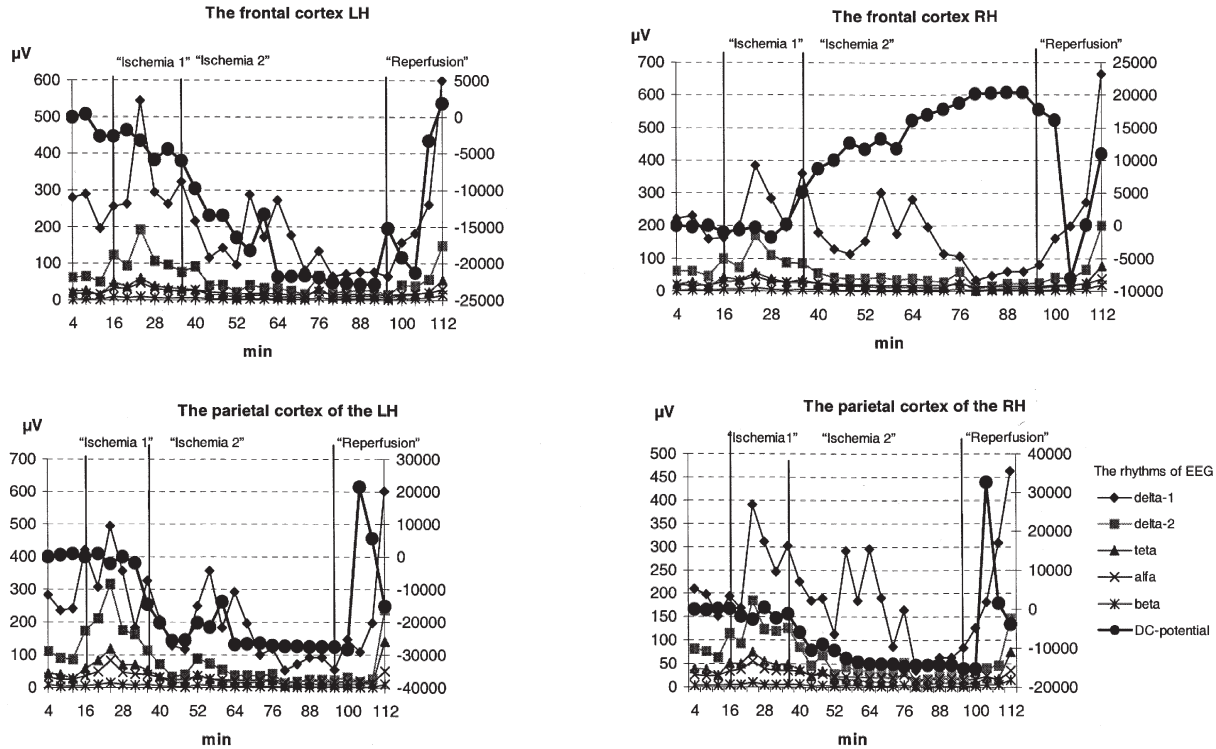

FIGURE 1. Changes in the DC potential and EEG in the rat neocortex during modeling of brain ischemia by different methods. "Ischemia-1" is binding up of common carotids; "Ischemia-2" is insertion of the occluder into MBA of the LH; "Reperfusion" is the removal of the occluder from MBA. EEG rhythm amplitude is shown in the left scale of the Y axis, and the DC potential level (μV), in the right one. DC potential values in the period preceding "Ischemia-1" are taken as a zero.

EEG rhythms resulted in greater changes in the DC potential in the neocortex having both positive and negative direction (Figure 1, "Ischemia-2"). In the LH a significant (some tens of millivolt) negative shift in the DC potential level was observed both in the frontal and parietal cortex. An average depression in the EEG rhythms in the LH amounted to 25.62 ± 2.41 in the frontal cortex and 22.74 ± 1.94% in the parietal one. Slow frequencies were mostly changed (see Table 1). In the RH a decrease in the rhythms was reliably less than in the LH and amounted to 14.28 ± 2.49% for the frontal cortex and 13.03 ±2.19% for the parietal cortex. The analysis of EEG changes in the RH by particular ranges (see Table 1) showed that the decrease affected only delta-rhythm. In other frequency ranges the insertion of the occluder in the MBA of the LH failed to result in reliable changes in the rhythms as compared to the period preceding brain ischemia. Differences in the biopotential changes in the RH were also true for the DC potential. In the parietal cortex of the RH a negative shift was almost three times less than in the LH (see Figure 2). In the frontal cortex of the RH the DC potential shift was of a positive direction.

Thus, the development of the circulatory ischemia according to model 1 was followed by a rather low negative shift in the DC potential level and by an increase in the EEG rhythm power in all derivations. The use of occlusion of MBA of the LH in model 2 resulted in a significant additional negativation of the DC potential level of the LH and a depression on this background of the EEG in it.

It is also seen in Figure 2 that after removing the occluder the changes recorded in the DC potential in derivations were opposite to those observed when the occluder was inserted in the MBA. On the average in the whole group of animals the potential had a positive shift during 16 minutes in the frontal and parietal cortex of the LH by 42.7 ± 10% ($p < .05$) and 40.0 ± 22% ($p < .05$), respectively. The removal of the occluder caused a decrease in the DC potential by 23.8 ± 12.7% ($p < .05$) in the frontal cortex of the RH. A slight positivation of the DC potential amounting to 17.2 ± 11.2% was observed in the parietal cortex of the same hemisphere.

The analysis of the resulting rhythm changes showed that reperfusion caused an increase in the EEG power in all channels and all frequencies by 11.6 ± 2.03% ($p < .001$) on average. A comparison

TABLE 1. The change in the rhythm power (%) of different ranges in the "Ischemia-2" period relative to the period preceding "Ischemia-1"

	Left hemisphere					*Right hemisphere*				
	Delta-1	*Delta-2*	*Theta*	*Alpha*	*Beta*	*Delta-1*	*Delta-2*	*Theta*	*Alpha*	*Beta*
Frontal cortex	*** –50.83 ± 3.52	*** –38.10 ± 5.07	*** –34.10 ± 2.41	** –11.76 ± 4.09	–8.45 ± 6.22	*** –51.86 ± 2.44	*** –23.45 ± 3.17	+3.45 ± 2.20	+1.39 ± 2.62	–7.18 ± 5.94
Parietal cortex	*** –44.84 ± 4.38	*** –21.77 ± 3.31	*** –21.17 ± 3.31	** –13.03 ± 2.90	* –17.09 ± 5.20	– –42.94 ± 1.60	** –18.35 ± 3.24	–4.94 ± 3.39	–2.63 ± 3.43	–4.23 ± 1.89

"–", decrease in the rhythm amplitude; "+", increase in the rhythm amplitude.
*$p < .05$, **$p < .01$, ***$p < .001$.

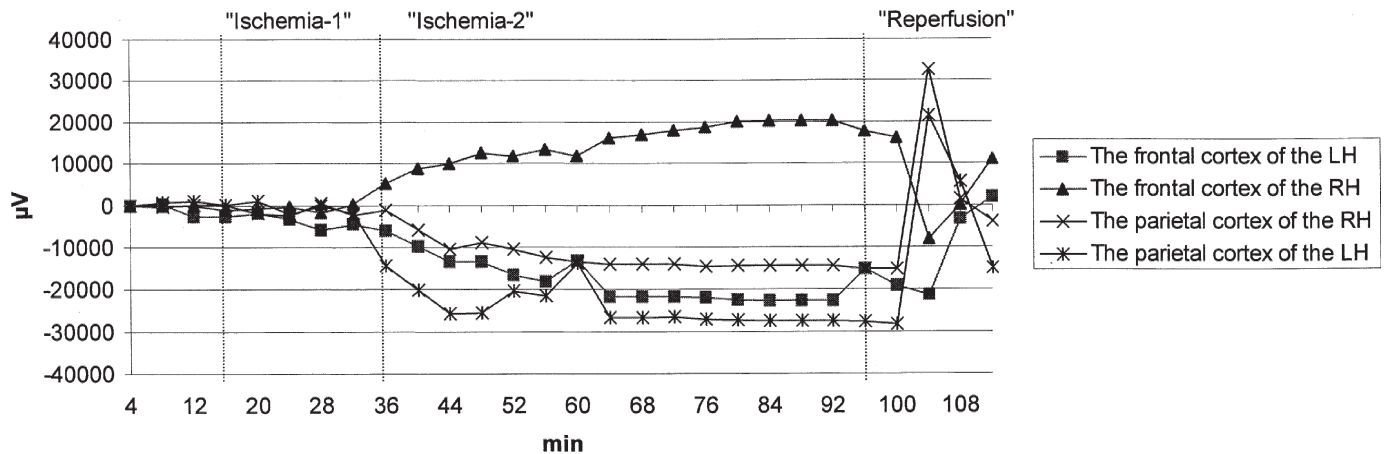

FIGURE 2. Changes in the DC potential level in the rat neocortex during modeling of ischemia by different methods. DC potential values in the period preceding "Ischemia-1" are taken as a zero.

of the EEG changes in the RH and the LH revealed an increase in the rhythm power mainly in the parietal cortex of the RH and in the frontal cortex of the LH. The greatest increase in the average amplitude of all rhythms was observed in the parietal cortex of the RH and amounted to 29.08 ± 2.86% ($p < .001$), alpha- and beta-ranges being mostly increased by 31.47 ± 5.14% ($p < .002$) and 36.13 ± 9.3% ($p < .004$), respectively, as compared to 20.25 ± 3.05% ($p < .01$) for delta and 18.98 ± 2.86% ($p < .01$) for theta-rhythms. The increase of the rhythm power in the frontal cortex of the LH was 16.59 ± 2.89% ($p < .001$) without an apparent prevalence in any range. In the frontal cortex of the RH there was only a tendency to amplitude increase in the theta-rhythm range. In the parietal cortex of the LH with an increase in the activity in the corresponding area of the RH no reliable changes were found in any frequency.

Thus, according to EEG data reperfusive changes affected chiefly the parietal cortex of the RH, in which the power of all rhythms increased. In the LH some increase in the amplitude of the rhythms was observed in the frontal cortex. A comparison of EEG developed during 16 min after reperfusion with EEG observed in these animals prior and during "Ischemia-1" showed that the RH approached the period of "Ischemia-1" by characteristics of the main rhythms, although the amplitude was a little lower, whereas in the LH the rhythm power as in the case of ischemia remained in an average lower by 18.89 ± 2.63% ($p < .001$) as compared to a preoperative period. Furthermore, the right hemisphere domination of the average power of all rhythms by 10.37 ± 1.14% recorded during ischemia following the removal of the occluder not only retained (in spite of some increase of the rhythm amplitude in the frontal cortex of the LH) but further increased up to 26.6 ± 6.04%. An examination of interhemisphere asymmetry in the rhythm power after reperfusion in the frontal and parietal cortex singly showed that in the frontal cortex after removal of the occluder from the left internal carotid there were no actual differences in all the rhythms between hemispheres. The rhythms dominated in the RH by 2.56 ± 3.63% ($p > .05$). However, the asymmetry was found when the ranges were considered separately. Thus, theta- and alpha-rhythm dominated in the RH by 13.45 ± 8.34 and 8.25 ± 3.30%, respectively, whereas beta-rhythm prevailed in the LH by 11.63 ± 6.10%. In the

parietal cortex the power of all rhythms in the RH was higher than in the LH by 52.69 ± 8.45%.

Therefore, reperfusion related to the removal of the occluder from the left internal carotid was followed by maintenance of a general picture of interhemisphere differences in the rhythm power observed during ischemia, that is, the rhythm amplitude of the RH dominated over the amplitude of the LH; however, it was of a more complex character. Thus, in the parietal cortex the asymmetry increased in all frequencies, while in the frontal cortex the amplitude of rather rapid rhythms became dominating in the LH and rather slow rhythms remained more expressed in the RH as it was during modeling of the "Ischemia-2."

A comparison of the character of changes in the EEG power and DC potential level after reperfusion showed that the resulting increase in the rhythm power occurred during both positive and negative shifts of the DC potential. In the LH the withdrawal of the occluder resulted in positivation of the DC potential in the frontal and parietal cortex and in an increase in the amplitude of EEG rhythms in the frontal cortex. In the RH the rhythm power increased both during positive (parietal cortex) and negative (frontal cortex) shifts of the DC potential.

Figure 3 shows the change in the DC potential and EEG in the rats after intracerebroventricular CPA injection in four derivations. The CPA injection is seen to cause in all cases a positive shift in the DC potential with an initial activation of the power of the most (delta-1, delta-2, and beta) of EEG rhythms changed (in 5–10 mm) for their depression (Figure 4). The highest positivation of the DC potential was observed in the RH, that is, at the side of the injection.

Therefore, the CPA injection resulted in due time in differently directed changes in the EEG rhythm power along with a positive deviation of the DC potential. Right after preparation injection the rhythm power increased and then in 5–10 min it was inhibited.

Figure 5 shows the changes in the recording biopotentials in case of interperitoneal injection of Nembutal in rats. At the beginning, right after the injection, a negative shift in the DC potential ($p < .001$) and an increase in the EEG power ($p < .001$) were observed. One to two minutes later a positive shift in the DC potential appeared with maintaining an enhanced rhythm power (Figure 5, "Presleep").

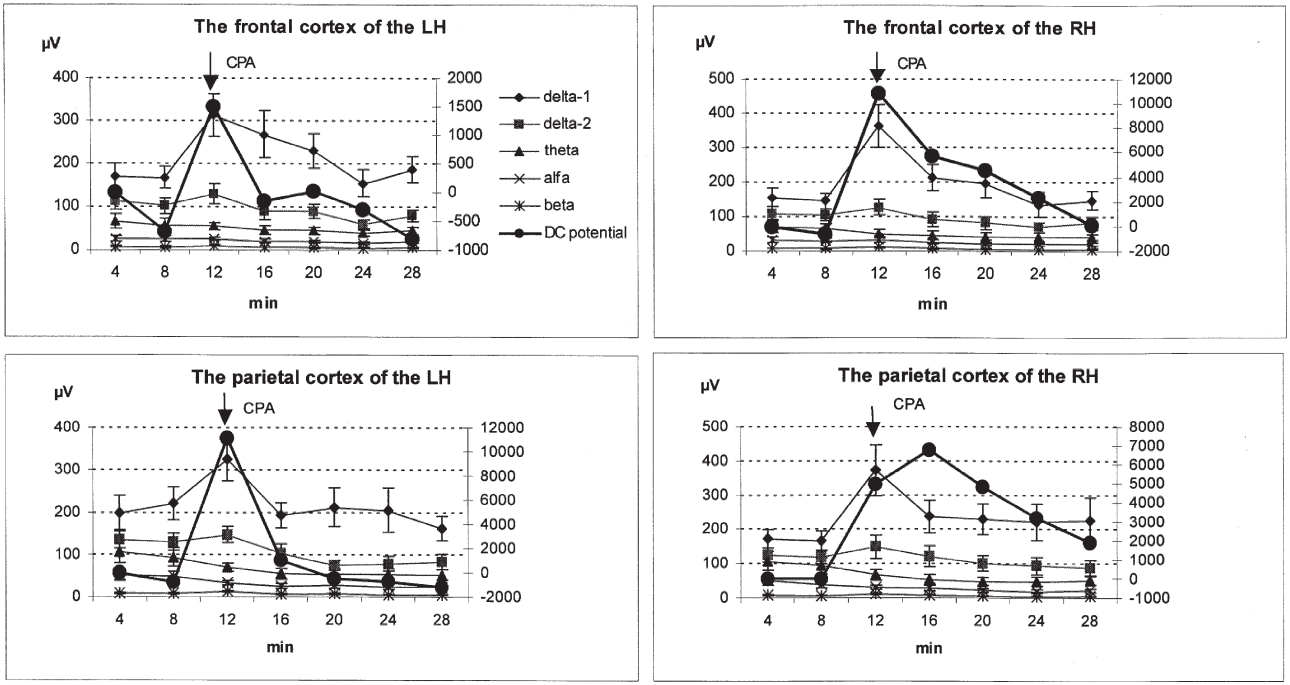

FIGURE 3. Changes in the DC potential level and EEG rhythm power in rats after intracerebroventricular injection of CPA. The arrow shows the moment of the CPA injection. For the rest of designations see Figure 1.

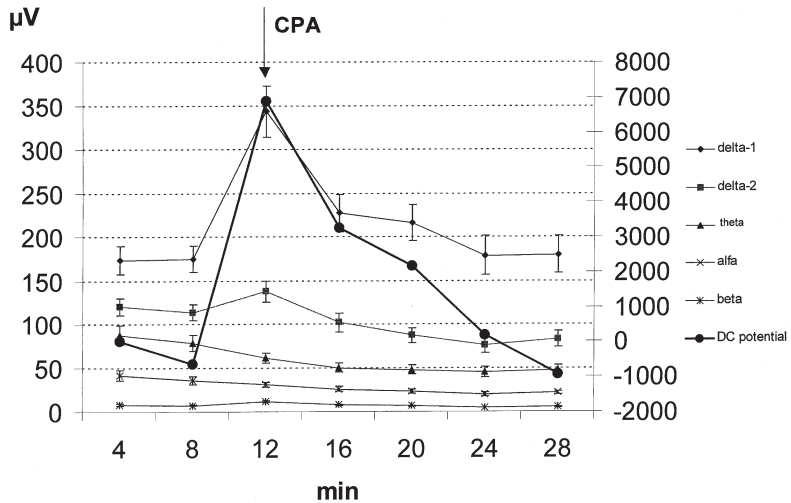

FIGURE 4. Changes in the DC potential level and EEG rhythm power as an average in all derivations after intracerebroventricular injection of CPA.

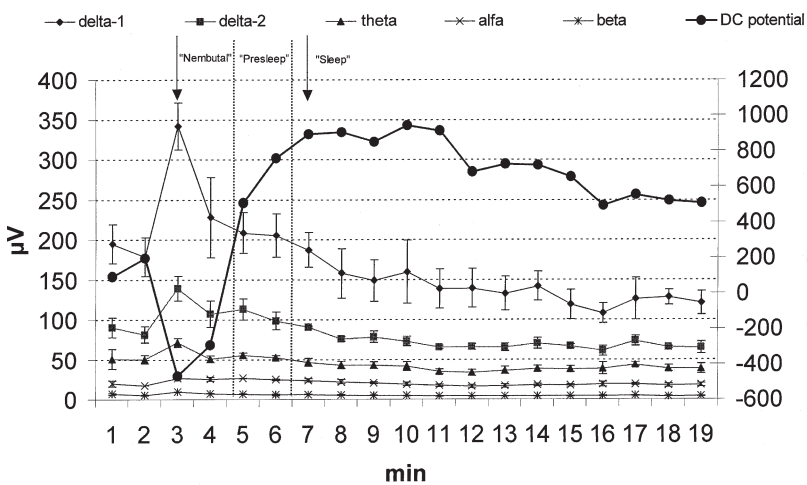

FIGURE 5. Changes in the DC potential level and EGG rhythm power after intraperitoneal injection of 40 mg/kg Nembutal.

The disappearance of pain sensitivity and falling asleep of the animal proceeded with a further positive shift in the DC potential with a simultaneous decrease in the amplitude of the EEG rhythms that were inhibited at a maximum during further sleep.

As a whole the results of this study showed that complex interrelations between DC potential level and EEG rhythms demonstrated in the ischemia model and intracerebroventicular injection of CPA were also observed after interperitoneal injection of Nembutal.

DISCUSSION

Different character of interrelations between changes in the DC potential and EEG are the main facts revealed in these experiments. Positive and negative shifts in the DC potential can be followed by both an increase and a decrease in the EEG power. The analysis of the data available in the literature and the changes in the complex of bioelectrical parameters (EEG and DC potential) obtained in these experiments during rat narcotization with Nembutal, intracerebroventricular injection of CPA, binding up of common carotids, MBA occlusion and reperfusion allow the authors to make a definite conclusion on the functional and metabolic changes proceeding in the brain nervous tissue. As shown in these experiments, the limitation of the total brain blood flow by binding up of the common carotids ("Ischemia 1") results in an increase of the EEG rhythm power and development of a negative shift of the DC potential. According to the literature the increase in the EEG rhythm power reflects the development of activation processes (Ingvar, 1967; Moskalenko et al., 1969), while a negative shift in the DC potential indicates depolarization of neuron elements (Marczynski, 1993; Kohling, et al., 1996). Thus, as a whole the character of the changes of biopotentials in this model of ischemia suggests the development of the functional state of a catelectrotonus type in the nervous cells.

The aggravation of the ischemic state in the LH after introduction of the occluder in the MBA revealed a depression in the EEG rhythms on the background of still greater negativation of the DC potential. According to literature data (Kosmotiani et al., 1969; Gurvich et al., 1969; Chen et al., 1993; Mies et al., 1993), a negative shift in

the DC potential and EEG depression indicate the development of a deep brain ischemia. It allows us to consider that in the LH a severe brain ischemia was modeled during "Ischemia-2," which is supported by a histological analysis (Sufianova et al., 2001). Such a state of the nervous tissue can be characterized as a state of parabiosis (Vvedensky) or cathodic Verigo depression.

The changes in the EEG and DC potential observed in the RH during "Ischemia-2" appear to be related to a redistribution of the blood flow at the expense of activation of the mechanisms of collateral blood circulation. There is evidence on an enhancement of the blood flow in the basin of symmetrical arteries of the hemisphere contrary to insult (Pokrovsky et al., 1989). Simultaneous negative shifts in the DC potential observed in the LH and positive shifts of the DC potential in the frontal cortex of the RH indicate an increase of the blood flow in this part of the brain and development of repolarization processes here, whereas a decrease in the EEG rhythm power indicates an improvement of metabolic and functional state of the nervous tissue impaired in the period of "Ischemia-1." In other words, in the frontal cortex of the RH, a state close to a preoperative one was formed after introduction of the occluder. It is supported by a histological analysis of the nervous tissue of the RH (Sufianova et al., 2001). The parietal cortex of the RH appeared to be partially "robbed" at the expense of activation of the blood supply of the frontal cortex of this hemisphere. A negative shift in the DC potential in the parietal cortex of the RH along with some decrease of rhythm power in it indicates the formation of a relatively unfavorable metabolic and functional state of the nervous tissue during "Ischemia-2."

Therefore, evidences of the experiments with the "Ischemia-2" model demonstrated that the depression of the EEG rhythms could follow both development of the unfavorable functional and metabolic state of the nervous tissue and returning to the state close to an optimum, and their differentiation is possible only by the character of resulting changes in the DC potential. The development of the unfavorable functional state is followed by a negative deviation of the DC potential.

A comparison of the results of the changes of the biopotentials of the two models of ischemia makes it possible to consider "Ischemia-

1" as a model of a mild ischemia. It has been shown in a similar model of ischemia (Dirlam, 1994) that in spite of the resulting inactivation of barrier-transport systems of the hematoencephalic barrier, neurons maintain a high level of oxidative metabolism due to the existence of intracellular compensatory-adaptive mechanisms in particular. As is evidenced (Yanvareva & Kuzmina, 1985), neurons affected by hypoxia initially respond by depolarization of the rest potential and by activation of the impulse activity changing as hypoxia became more pronounced for its depression of a parabiotic type. Most likely the activation of EEG and negativation of the DC potential observed during binding up of common carotids reflect an initial exaltation of brain cell excitability of a catelectronic type.

The withdrawal of the occluder and reperfusion of the brain along the MBA caused an increase in the EEG rhythm power and a positive shift in the DC potential in the frontal and parietal cortex of the LH. A similar picture was observed in the parietal cortex of the RH. In the frontal cortex of the RH the reperfusion of MBA of the LH resulted in an increase of the EEG power at the background of a negative shift in the DC potential. To put it otherwise, the restoration of the regime of blood flow corresponding to the period of "Ischemia-1" in the LH according to electrophysiological data reflects an improvement of metabolic and functional state of the nervous tissue. Similar processes occurred in the parietal cortex of the RH, whereas in the frontal cortex the removal of the occluder reducing collateral blood flow impaired its metabolic state, thus activating it again.

The data on reperfusion demonstrate that the brain activation revealed by the EEG characteristics is possible both with negative and positive shifts of the DC potential. In the first case it suggests an impairment of the functional and metabolic state (as during "Ischemia-1"), whereas in the second case it suggests its improvement because of leaving the state of cathodic depression with increasing blood flow.

In the experiments with CPA injection a positive shift of the DC potential was recorded followed by an initial activation of the EEG power with its further inhibition. According to the literature (Kostopoulos & Phillis, 1977; Shefner & Chiui, 1986), adenosine and its analogs (including CPA) inhibit the impulse activity of neurons and cause

membrane hyperpolarization. The positivation of the DC potential demonstrated in the present experiment also indicates neuron hyperpolarization. However, brain hyperpolarization can combine both with an increase in the rhythm power and its depression. The initial activation of the EEG in delta- and beta-ranges (Figure 4) with a positive shift in the DC potential apparently suggests the development of the functional state similar to anode exaltation with increasing metabolic need at the background of maintaining ionic homeostasis, which gradually changed for hyperpolarization inhibition. A decline in the rhythm power developing after CPA injection was steady and continued for 60 min observation.

Simultaneous recording of the DC potential level and EEG during narcotization showed at least three subsequent stages of the change in the functional and metabolic state of the brain. A negative emotional excitability developing apparently in animals after puncturing the integument with a needle and the preparation injection was followed by negativation of the constant potential and an increase in the EEG power, presumably reflecting development of depolarizing exaltation of neuron excitability and intensification of metabolism of the nervous tissue. As Nembutal was sucked in the blood the DC potential showed a positive shift with maintaining an enhanced EEG power (Figure 5, "Presleep"). The character of the change of the DC potential indicates the development of re- and hyperpolarization processes and an enhanced amplitude of the rhythms suggests the absence of hyperpolarization inhibition at this period. The data are available on hyperpolarization changes of the membrane potential of neurons affected by Nembutal (Sato et al., 1967). A comparison of the DC potential level and EEG characteristics makes it possible to consider in neurons in the "Presleep" period the development of the functional state corresponding to anode exaltation and intensification of metabolism of the nervous tissue at this period. Finally, an onset and development of the narcotic sleep was followed by still greater positive shift of the DC potential with resulting inhibition of the EEG power. The functional state observed presumably reflects further hyperpolarization of the cells and the onset of hyperpolarization inhibition with decreasing metabolism of the nervous tissue.

A comparison of these data with the results of biopotential changes

after CPA injection indicates their principle similarity. In both cases a positive shift in the DC potential was followed by an initial activation of EEG with its subsequent decrease. It suggests that in both cases generally similar functional and metabolic processes developed.

In conclusion, the analysis of the data available in the literature and of the character of the changes of the complex of bioelectrical parameters (EEG and DC potential) obtained in these experiments allows for a conclusion on resulting functional and metabolic processes in the brain nervous tissue following these changes:

- Negative shifts in the DC potential and an increase in the EEG power occur in the process of depolarization of the neuron membrane followed by an increase of excitability and metabolic need of the nervous tissue. Similar changes in the EEG and DC potential are observed, particularly during development of the reaction of activation.
- Negative shifts in the DC potential and depression in the EEG power occur in the process of still greater depolarization of neurons, development of depolarizing inhibition (by a parabiotic or cathodic type), and a decrease in the level of their metabolism.
- Positive shifts in the DC potential and an increase in the EEG power occur in the process of repolarization of the cell membrane in connection with their getting out of the cathodic depression or parabiosis or during hyperpolarization and development of hyperpolarizing exaltation of excitability. In both cases it is related to metabolism activation.
- Finally, positive shifts in the DC potential and depression in the EEG power occur in the processes of repolarization of the cell membrane in connection with returning of the membrane potential to the level of the rest potential after depolarizing exaltation of excitability or during hyperpolarization of the cell membrane and development of the hyperpolarizing depression. In both cases it is related to a decline in the metabolic need of the nervous tissue.

The results of the experiments described demonstrated great diagnostic opportunities of the method proposed. Neither EEG nor DC potential separately could perform precise differentiation of the

functional and metabolic state of the nervous tissue. An advantage of the method is also its relative simplicity. The method proposed makes it possible to record and differentiate the transfer of one functional and metabolic state into another, thus increasing the precision of diagnostics of pathological and physiological states, to carry out adequate drug therapy of pathological states; for instance, during ischemia aimed at restoration of functional and metabolic state of the nervous tissue, to assess prognosis and validity of medical treatment following particular therapy, as well as to study the effect of extreme factors on the human organism.

CONCLUSIONS

1. An intricate character of the changes in the bioelectrical parameters (EEG and DC potential) is shown in rats during Nembutal narcotization, intracerebroventricular injection of CPA, binding up of both carotids, MBA occlusion, and reperfusion. Negative and positive shifts of the DC potential can be followed by both an increase and a decline in the EEG rhythm power reflecting the development of different functional and metabolic states of the nervous tissue.
2. The character of the changes of the complex of the bioelectrical parameters (EEG and DC potential) makes it possible to assess functional and metabolic states of the brain nervous tissue:
   - a negative shift in the DC potential and an increase in the EEG rhythm power reflect the development of depolarizating activation of neurons and enhanced metabolism of the nervous tissue;
   - a negative shift in the DC potential and a decline in the EEG rhythm power reflect the development of depolarizating inhibition and a decline in the metabolism of the nervous tissue;
   - a positive shift in the DC potential and an increase of the EEG rhythm power reflect the development of repolarizating and hyperpolarizating activation of neurons and enhanced metabolism of the nervous tissue;
   - a positive shift in the DC potential and a decline in the EEG rhythm power reflect the development of repolarizating or

hyperpolarizating inhibition of neurons and a decline in metabolism of the nervous tissue.

REFERENCES

Buchsbaum, M. S., Gillin, J. C., Wu, J., Hazlett, E., Sicotte, N., Dupont, R. M. et al. (1989). Regional cerebral glucose metabolic rate in human sleep assessed by positron emission tomography. *Life Sciences, 45*(15), 1349–1356.

Chen, Q., Chopp, M., Bodzin, G., & Chen, H. (1993). Temperature modulation of cerebral depolarization during focal cerebral ischemia in rats: Correlation with ischemic injury. *Journal of Cerebral Blood Flow and Metabolism, 13*(3), 389–394.

Daniyarov, S. B., & Vilenskaya, E. M. (1986). The effect of high mountain hypoxia on the human EEG. *Journal of Higher Nervous Activity, 30*(2), 337–343.

Demchenko, I. T. (1976). The methods of investigation of the brain circulation. In B. I. Tkachenko (Ed.), *The methods of investigation of the circulation* (pp. 104–125). Leningrad: Nauka.

Dirlam, G. G. (1994). The reactivity of capillaries and pyramid neurons of brain cortex in rats under conditions of acute reduction of blood stream. *The Bulletin of Experimental Biology and Medicine, 5,* 558–560. [in Russian]

Elbert, T. (1993). Slow cortical potentials reflect the regulation of cortical excitability. In W. C. McCallum & S. H. Curry (Eds.), *Slow potential changes in the human brain* (pp. 235–251). New York: Plenum Press.

Gurvich, A. M., Shikunova, L. G., Novoderzhkina, I. S., & Bulanova, O. N. (1969). The role of posthypoxic changes of metabolism and brain edema in the dynamics of the restoration of functions of the central nervous system following long-term period of the complete cessation of circulation. In G. I. Mchedlishvili (Ed.), *The correlation of blood supply with metabolism and function* (pp. 233–240). Tbilisi: Metsniereba.

Hockaday, I. M., Potts, F., Epstein, E., Bonazzi, A., & Schwab, R. (1965). EEG changes in acute cerebral anoxia from cardiac or respiratory arrest. *Electroencephalography and Clinical Neurophysiology, 18*(6), 575–586.

Ingvar, D. H. (1967). Cerebral metabolism, cerebral blood flow end EEG. *Electroencephalography and Clinical Neurophysiology, 25,* 102–106.

Ingvar, D. H., Sjolund, B., & Arbo, A. (1976). Correlation between dominant EEG frequency cerebral oxygen upstake and blood flow. *Electroencephalography and Clinical Neurophysiology, 41*(3), 268–276.

Kauser-Gatchalian, M. C., & Neundörfer, B. (1980). The prognostic value of EEG in ischemic cerebral insults. *Electroencephalography and Clinical Neurophysiology, 49*(5–6), 608–617.

Kohling, R., Schmidinger, A., Hulsmann, S., Vanhatalo, S., Lucke, A., Straub, H. et al. (1996). Anoxic terminal negative DC-shift in human neocortical slices in vitro. *Brain Research, 741*(1–2), 174–179.

Kosmotiani, P. A., Chikvaidze, V. N., Svanidze, I. K., & Mchedlishvili, G. I. (1969). The effect of ischemia on some metabolic processes in the central nervous system. In G. J. Mchedlishvili (Ed.), *The correlation of blood supply with metabolism and function* (pp. 201–210). Tbilisi: Metsniereba.

Kostopoulos, G. K., & Phillis, J. W. (1977). Purinergic depression of neurons in different areas of the rat brain. *Experimental Neurology, 55,* 719–724.

Li, J., Takeda, Y., & Hirakawa M. (2000). Threshold of ischemic depolarization for neuronal injury following four-vessel occlusion in the rat cortex. *Journal Neurosurgical. Anesthesiology, 12*(3), 247–254.

Marczynski, T. J. (1993). Neurochemical interpretation of cortical slow potentials as they

relate to cognitive processes and a parsimonious model of mammalian brain. In W. C. McCallum & S. H. Curry (Eds.), *Slow potential changes in the human brain* (pp. 253–275). New York: Plenum Press.

Marczynski, T. J. (1993). Neurochemical interpretation of cortical slow potentials as they relate to cognitive processes and a parsimonious model of mammalian brain. In W. C. McCallum & S. H. Curry (Eds.), *Slow potential changes in the human brain* (pp. 253–275). New York: Plenum Press.

Mies, G., Iijima, T., & Hossmann, K. A. (1993). Correlation between peri-infarct DC shifts and ischemic neuronal damage in rat. *Neuroreport, 4*(6), 709–711.

Moskalenko, Yu. E., Demchenko, I. T., Savich, A. A., & Vainshtein, G. B. (1969). On particular relations between local blood stream and some parameters of the functional state of limited parts of the brain. In G. I. Mchedlishvili (Ed.), *The correlation of blood supply with metabolism and function* (pp. 154–163). Tbilisi: Metsniereba.

Pokrovsky, A. V., Yakhno, N. N., Kuntsevich, G. I., Lavrentieva, M. A., & Malkova, M. V. (1989). The characteristics of intrabrain hypodynamics in occlusing damages of the main brain arteries. *The Journal of Neuropathology and Psychiatry,* **89**(9), 7–11. [in Russian]

Rebert, C. S. (1978). Electrogenesis of slow potential changes in the central nervous system: A summary of issues. In D. Otto (Ed.), *Multidisciplinary perspectives in event related brain potential research* (pp. 3–11). Cincinnati: U.S. Environmental Protection Agency.

Rogers, H., Birch, P. J., & Hayes A. G. (1990). Effects of hypoxia and hypoglycaemia on DC potentials recorded from the gerbil hippocampus in vitro. *Naunyn-Schmiedebergs Archives of Pharmacology, 342*(5), 547–553.

Rusinov, V. S. (1987). *The biopotentials of the human brain. Mathematical analysis.* Moscow: Meditsina.

Sato, H., Austin, G., & Yai, H. (1967). Increase in permeability of the postsynaptic membrane to potassium produced by "Nembutal." *Nature, 215*(109), 1506–1508.

Shefner, S. A., & Chiui, R. H. (1986). Adenosine inhibits locus coereleus neurons: An intracellular study in a rat brain slices preparation. *Brain Research, 366*(1–2), 364–1368.

Sufianova, G. Z., Usov, L. A., Sufianov, A. A. et al. (2001). The minimal invasive model of the focal brain ischemia in rats. *The Experimental and Clinical Pharmacology, 64*(4), 63–67. [in Russian]

Von der Drift, I. H. A., & Kok, N. K. D. (1973). The value of the EEG for the prognosis of cerebrovascular disease. In J. S. Meyer, H. Lechner, & M. Reivich (Eds.), *Cerebral vascular disease* (pp. 127–135). Salzburg.

Yanvaneva, I. N., & Kuzmina, T. R. (1985). On mechanisms of disturbances of the functional state of the central nervous system under oxygen deficiency of the brain. In N. P. Mowchan (Ed.), *The physiological mechanisms of the main nervous processes (Leningrad naturalists association).* Leningrad, *75*(5), 71–77.

Zhirmunskaya, E. A. (1963). *The normal electrical activity of the brain in the hypertonic disease and brain insult*. Moscow: Nauka.

Zhirmunskaya, E. A. (1989). *The functional interrelations of the human cerebral hemispheres*. Leningrad: Nauka.

Zhirmunskaya, E. A., Gutman, S. R., Maslov, V. K., Mezentsev, V. V., & Ustinova N. S. (1977). On the recognition of the state of the person under investigation by characteristics of his electroencephalogram. In V. S. Rusinov (Ed.), *The functional importance of electrical processes of the brain* (pp. 274–282). Moscow: Nauka.