FUNCTIONAL STATE OF THE CEREBRAL CORTEX AND MESENCEPHALIC RETICULAR FORMATION IN THE COURSE OF TRAUMATIC SHOCK

S. P. Matua

UDC 616-001.36:612.825.001.6

KEY WORDS: traumatic shock; evoked potential; seizure response; activation response, brain.

Data in the literature on functional changes in the cerebral cortex and mesencephalic reticular formation (RF) in traumatic shock are highly contradictory [3, 6, 7, 9-11, 14, 16]. Electrophysiological studies of the functional properties of the sensomotor cortex and mesencephalic RF [1, 2, 3, 5, 8, 12, 15] have demonstrated extensive two-way connections of these structures and other parts of the brain, and their important role in processes of integrative brain activity so that their essential role in reactions of the body to all extremal factors can be postulated.

The object of this investigation was to make a combined study, by modern electrophysiological methods, of the functional state of these vital brain structures during the course of traumatic shock.

EXPERIMENTAL METHOD

Acute experiments were carried out on cats weighing 2.5-4 kg, anesthetized intraperitoneally with pentobarbital (20-25 mg/kg) or a mixture of chloralose and pentobarbital (40-45 and 10-15 mg/kg, respectively). Steel needle electrodes (diameter of the active tip 100-150 μ, interelectrode distance 1 mm) were implanted by means of a stereotaxic apparatus of the MV-4101 type, in accordance with coordinates taken from an atlas for the cat [13]. Tripolar electrodes were inserted into the sensomotor and visual cortex and bipolar electrodes into the mesencephalic RF. A monopolar system was used for recording, the reference electrode being located in the frontal bones. The functional state of the cortex was assessed by determining thresholds of the seizure response (TSR) induced by direct electrical stimulation of different cortical zones by square pulses with a frequency of 60-100 Hz, a duration of 0.1-0.5 msec, and a voltage of 1-15 V, applied for a period of 5 sec, and also from evoked potentials (EP) recorded in the sensomotor projection cortex during electrical stimulation of the skin of the forelimb. The functional state of RF was judged from thresholds of the activation response (RA) on the electrocorticogram (ECoG) of the sensomotor cortex, produced in response to direct high-frequency (150-200 Hz) electrical stimulation of RF, and also from EP recorded in it synchronously with primary responses (PR) in the cortex to electrical stimulation of the skin. A two-channel ESU-2 electronic stimulator with radiofrequency attachments was used for stimulation. The ECoG was recorded on an 8-channel Medicor electroencephalograph. The derived EP were led to symmetrical inputs of type UBP-02 AG amplifiers with transmission band of 1 to 10,000 Hz and recorded from the screen of a type S1-18 dual-beam CRO by means of the SOR-2 photographic recorder by superposition during stimulation with a frequency of once in 5 sec. The mean arterial blood pressure (BP) was recorded in one of the common aortic arteries by means of a mercury manometer. Shock was induced by Cannon's method. The results were subjected to statistical analysis on the Odra computer.

EXPERIMENTAL RESULTS

Simultaneous investigation of EP in the sensomotor projection cortex and in the mesencephalic RF revealed differences in their evolution during the course of shock. The PR appeared after a latent period of 13.4 ± 2.4 msec and consisted of an initial positive wave

Department of Pathological Physiology, Rostov Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR V. K. Kulagin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 91, No. 3, pp. 296-298, March, 1981. Original article submitted July 21, 1980.

 0007-4888/81/9103-0300$07.50

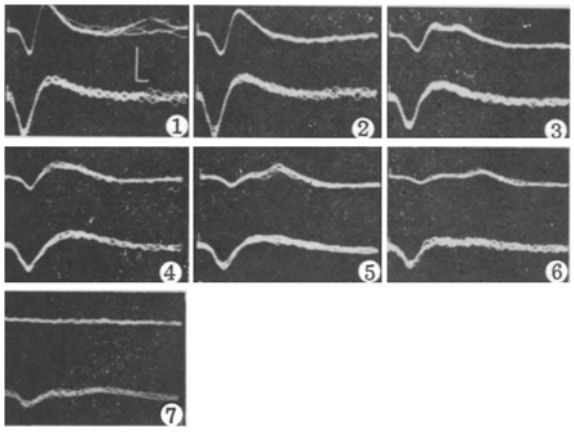

Fig. 1

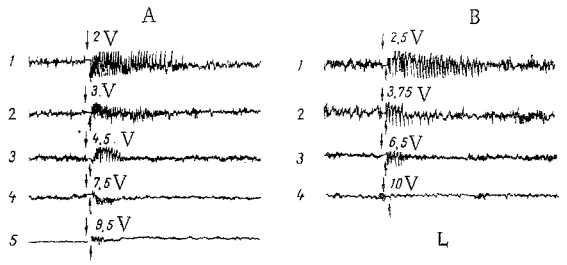

Fig. 2

Fig. 1. Changes in EP in sensomotor cortex and mesencephalic FR during electrical stimulation of skin of contralateral forelimb during course of traumatic shock. 1) Initial background (BP 130 mm Hg), 2) immediately after trauma (BP 50 mm Hg), 3) after 20 min of shock (BP 85 mm Hg), 4) after 40 min of shock (BP 75 mm Hg), 5) after 60 min of shock, (BP 60 mm Hg), 6) after 90 min of shock (BP 45 mm Hg), 7) after 120 min of shock (BP 40 mm Hg). Death after 150 min. Calibration: for amplification 100 μV, for time 20 msec.

Fig. 2. Changes in TSR in sensomotor (A) and visual (B) areas of cortex during course of traumatic shock. 1) Initial background (BP 120 mm Hg), 2) immediately after trauma (BP 60-55 mm Hg), 3) after 20 min of shock (BP 70 mm Hg), 4) after 40 min of shock (BP 60-55 mm Hg), 5) after 60 min of shock (BP 45 mm Hg). Death after 85 min. Calibration: for amplification 100 μV, for time 1 sec.

(amplitude 85.2 ± 14.6 μV, duration 25.4 ± 3.8 msec), followed by a negative wave (amplitude 83.6 ± 12.5 μV, duration 58.8 ± 2.4 msec). EP in the mesencephalic RF consisted of a postive–negative complex with latent period of 8.5 ± 1.7 msec and a combined amplitude of response of 163.5 ± 12.4 μV. After the first minutes of the posttraumatic period (Fig. 1) marked depression of the amplitude of PR was observed, whereas the amplitude of the reticular EP remained relatively stable. By the 20th minute of shock the combined amplitude of the cortical potentials was 40-50% of its initial level, but by the 60th minute it was only 10-20%, and the latent period of PR was lengthened by 2.5-3 times. Depression of reticular responses was less marked and was observed in most experiments only after the 20th-30th minutes of shock. A significant decrease in amplitude by 40-50% and lengthening of the latent period by 2-2.5 times were observed by the 60th-90th minutes of shock, but even in the late stages of shock hypotension (120 min, BP 40-45 mm Hg) the reticular response could still be recorded in some experiments, when PR had disappeared. The greater resistance of the positive component and increasing lengthening of the latent periods in the course of shock were characteristic of the evolution of EP in both structures. The observed lengthening of the latent periods of EP could indicate the onset of synaptic delay in traumatic shock at different levels of the afferent component of the somatosensory system which, as will be clear from the results, was more marked in the specific sensory pathways. In addition, the greater resistance of the positive component of PR observed during shock, according to existing views [4, 5, 8, 12], is evidence that the cortical neurons can still generate a postsynaptic slow potential, whereas the sharp depression of the negative component of PR points to suppression of the discharge of these neurons.

An objective method of evaluating the functional state of the cerebral cortex is by investigating electroencephalographic thresholds of evoked seizure activity [4]. The results of these experiments showed that functionally different cortical areas (sensomotor and visual) had different initial TSR values (2.0 ± 0.2 and 2.5 ± 0.2 V, respectively). Immediately after trauma an increase of TSR in the cortex by 0.5-2.0 V was observed. In 8 of 17 experiments a tendency toward their recovery was observed immediately after the primary increase in TSR, and this was accompanied (between the 20th and 40th minutes of shock) by stabilization of BP at the 80-90 mm Hg level. Later, however, there was a steady rise ($P < 0.01$) of the thresholds and simultaneous shortening of the duration of the epidischarges (Fig. 2), or even their

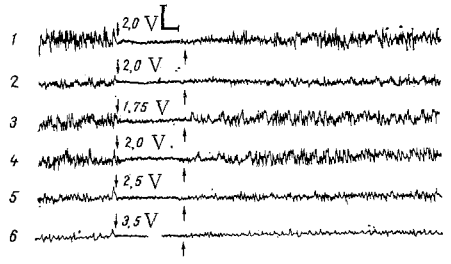

Fig. 3. Changes in thresholds of AR in sensomotor cortex during direct high frequency (150 Hz) electrical stimulation of mesencephalic RF. 1) Initial background (BP 135 mm Hg), 2) immediately after trauma (BP 55 mm Hg), 3) after 20 min of shock (BP 90 mm Hg), 4) after 40 min of shock (BP 80 mm Hg), 5) after 120 min of shock (BP 50 mm Hg), 6) after 180 min of shock (BP 35 mm Hg). Death after 217 min. Calibration: for amplification of 200 μV, for time 1 sec. Arrows indicate beginning and end of stimulation.

complete disappearance in later (60-90 min) stages of shock. These changes were more marked and took place considerably earlier in the visual cortex. Furthermore, whereas paroxysmal activity in the initial state spread from the test areas to other parts of the brain and was accompanied by a behavioral seizure response in the animals, as shock developed the seizure discharges in the cortex became local in character and were not manifested in external behavior. In most experiments a direct relationship was found between the ability of the cortex to give an evoked electroconvulsive response and the length of survival of the animals after trauma, i.e., the more marked the increase in TSR in the cortex and the sooner it occurred, the more rapidly the animals died.

The results thus point to considerable inhibition of the functional state of the cerebral cortex (more marked in its visual area) during shock and dependence of its course on the degree of depression of functional activity of this structure.

To assess the functional state of RF and the character of reticulocortical interrelations during the course of traumatic shock, thresholds of the AR were studied in the cortex during direct electrical stimulation of the mesencephalic RF.

The initial threshold for AR was 2.0-3.0 V. During the first 20 min after trauma (Fig. 3) the threshold of AR fell by 0.25-0.5 V, and this was accompanied by elevation of BP and its stabilization at the 85-90 mm Hg level, possible evidence of an increase in the excitability of the mesencephalic RF at this stage of shock. Before the 60-90th minutes of shock the thresholds of AR remained stable at the initial level, and only in the later stages (120-180 min) was a significant ($P < 0.05$) increase in threshold by 0.5-1.5 V observed. However, the possibility that AR can be produced under these conditions (BP 35-40 mm Hg) itself suggests that reticular neurons retain their ability to function at a sufficiently high level. Another interesting fact is that the activating effects of the mesencephalic RF also were preserved during a time of sharp depression of spontaneous cortical electrical activity.

The disparity found between the results obtained by the use of two different methods of assessing the functional state of RF (comparatively early depression of EP and the fairly long-lasting stability of the thresholds of AR) can be explained on the grounds that infliction of massive mechanical trauma leads primarily to blocking of somatosensory efferents before their entry into that structure, whereas it has no significant effect on the reticular neurons themselves.

The results as a whole are evidence that in traumatic shock the blockade of afferent pathways and inhibition of the functional state of the cerebral cortex, detectable comparatively early and associated with a relatively high level of functional activity of brain structures (mesencephalic RF) can be regarded as interconnected links of a single compensatory and adaptive mechanism, responsible for emergency control of initially important autonomic functions of the body under extremal conditions.

LITERATURE CITED

1. P. K. Anokhin and K. V. Sudakov, Dokl. Akad. Nauk SSSR, 187, 681 (1969).
2. A. S. Batuev, Cortical Mechanisms of Integrative Brain Activity [in Russian], Leningrad (1978).
3. A. N. Berkutov, S. N. Tsybulyak, N. I. Egurnov, et al., Éksp. Khir., No. 1, 58 (1973).
4. V. I. Gusel'nikov, Electrophysiology of the Brain [in Russian], Moscow (1976).
5. R. A. Durinyan, Cortical Control of Nonspecific Brain Systems [in Russian], Moscow (1975).
6. M. P. Zyablov, "Electrophysiological analysis of functions of the nervous system in the course of traumatic shock," Candidate's Dissertation, Rostov-on-Don (1977).

7. V. K. Kulagin, The Pathological Physiology of Trauma and Shock [in Russian], Leningrad (1978).
8. A. I. Roitbak, in: Current Problems in Electrophysiological Investigations of the Nervous System [in Russian], Moscow (1964), pp. 164-219.
9. B. A. Saakov and S. A. Eremina, Vestn. Khir., No. 1, 112 (1970).
10. D. M. Sherman, The Problem of Traumatic Shock [in Russian], Moscow (1972).
11. Yu. M. Shtykhno and B. T. Chuvin, in: Problems in the General Theory of Disease [in Russian], No. 1, Moscow (1976), p. 154.
12. H. T. Chang, Cold Spring Harbor, Symp. Quant. Biol., 17, 189 (1952).
13. H. Jasper and C. Ajmone-Marsan, A Stereotaxic Atlas of the Diencephalon of the Cat, Ottowa (1954).
14. A. G. Kovach and P. Sandor, Ann. Rev. Physiol., 38, 571 (1976).
15. H. H. Sheard and S. P. Flynn, Brain Res., 4, 324 (1967).
16. H. B. Stoner, Fed. Proc., 20, 38 (1961).

IONIC COMPOSITION AND POTASSIUM PERMEABILITY OF ERYTHROCYTE MEMBRANES FROM PATIENTS WITH THE INFLAMMATORY-DYSTROPHIC FORM OF PERIODONTOSIS

L. V. Peshkova, O. L. Orlova,
T. A. Kas'yanova, V. P. Trifonova,
and Yu. V. Evtodienko

UDC 616.314.17-008.1:616.155.126.31

KEY WORDS: erythrocytes; permeability; potassium content; periodontosis.

During the development of pathological processes considerable disturbances of acid–base balance in the tissues and of the ionic composition of the erythrocytes take place [1, 5, 7, 8]. In periodontosis, a stomatologic disease with most uncertain etiology and one which is most difficult to treat, energy metabolism in the tissues of the mouth undergo substantial changes [2, 3, 6]. One cause of the disturbance of function and energy metabolism of the tissues, it can be tentatively suggested, is a change in the ionic permeability of biomembranes and in the ionic homeostasis of the cells.

Accordingly it was decided to make a comparative study of the ionic composition and permeability of erythrocyte membranes in the capillary blood during the development of the inflammatory-dystrophic form of periodontosis.

EXPERIMENTAL METHOD

Erythrocytes were isolated from blood taken from the gums of a patient with periodontosis during curettage and also from blood taken from a finger. At the same time blood was taken from a finger for general analysis. Blood taken from the finger of clinically healthy persons served as the control.

The erythrocytes were separated, washed, and incubated in the same medium of the following composition: 0.3 M sucrose, 10 mM Tris-HCl, 50 units/ml heparin, pH 7.5. Blood in a volume of 0.1 ml was introduced into a centrifuge tube containing 1 ml of isolation medium and centrifuged for 10 min at 1000 rpm on the TsLK-1 centrifuge; the supernatant was drawn off and the residue was washed twice with 10 volumes of medium, after which the erythrocytes were sedimented by centrifugation.

The concentration of potassium ions passing into the incubation medium on the addition of erythrocytes was measured by means of a K^+-selective membrane electrode [4]. In the experiments with valinomycin the K^+ concentration was 1 μg/ml incubation medium. The coefficient

Laboratory of Biochemistry, Odessa Research Institute of Stomatology, and Department of Biochemical Regulation, Institute of Biological Physics, Academy of Sciences of the USSR, Pushchino. (Presented by Academician of the Academy of Medical Sciences of the USSR S. E. Severin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 91, No. 3, pp. 298-300, March, 1981. Original article submitted July 18, 1980.

