LITERATURE CITED

1. I. A. Arshavskii, Outlines of Age Physiology [in Russian], Moscow (1967).
2. T. P. Zhukova and V. R. Purin, Vopr. Okhr. Materin. Det., No. 8, 19 (1972).
3. Z. N. Kiseleva, Byull. Éksp. Biol. Med., No. 4, 115 (1960).
4. B. N. Klosovskii and E. P. Kosmarskaya, Vestn. Akad. Med. Nauk SSSR, No. 1, 18 (1969).
5. E. M. Mastyukova, "Characteristics of mental development and neuropsychic deviations in children exposed to asphyxia at birth," Author's Abstract of Candidate's Dissertation, Moscow (1965).
6. N. S. Sazonova and L. A. Ermolina, in: Current Problems in Oligophrenia [in Russian], Moscow (1975), pp. 206-215.
7. H. B. Benaron, B. E. Tucker, J. P. Andrews, et al., Am. J. Obstet. Gynecol., 80, 1129 (1960).
8. M. E. Jarvik and R. Kopp, Phychol. Rep., 21, 221 (1967).
9. B. Hellstrom and B. Jonsson, Acta Paediatr. Scand., 42, 398 (1953).
10. W. F. Windle (editor), Neurological and Psychological Deficits of Asphyxia Neonatorum, Thomas CC, Springfield (1958), pp. 31-43.

DYNAMICS OF DENDRITIC AND TRANSCALLOSAL CORTICAL POTENTIALS DURING HYPERTHERMIA

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Dendritic and transcallosal potentials in the course of hyperthermia and after restoration of temperature homeostasis were investigated in experiments of waking rabbits exposed in a heat chamber to an air temperature of 45°C. The effect of the high temperature was accompanied by marked depression of the amplitude of the dendritic potential, although by a lesser degree than the transcallosal response. The results are evidence of the direct inhibitory effect of heat on neurons in the various layers of the cortex. Restoration of the function of the cortical neurons after a high degree of hyperthermia is observed 24 h after normothermia is reached.

KEY WORDS: *Hyperthermia; cerebral cortex; evoked potentials.*

Experimental and clinical evidence have now been obtained to show that during hyperthermia the rhythm of the global electrocorticogram (ECoG) is slowed and its amplitude increased [3-5, 7, 10, 13, 15] and evoked potentials in specific cortical projection zones in response to adequate stimulation of receptors are sharply inhibited [4, 8]. However, it is impossible from these data to assess objectively the effect of a high temperature directly on cortical neurons, for the changes recorded largely reflect the functional state of subcortical structures also.

In the investigation described below the method of recording dendritic and transcallosal potentials was used in a direct study of cerebral cortical function in the course of hyperthermia and after restoration of temperature homeostasis.

EXPERIMENTAL METHOD

Experiments were carried out on 15 adult rabbits without the use of anesthetics or muscle relaxants. In most cases two experiments were carried out on each rabbit at an interval of 1 week. Two days before the experiment, under superficial pentobarbital anesthesia, stainless steel needle electrodes with an active tip 100 μ in diameter and an interelectrode

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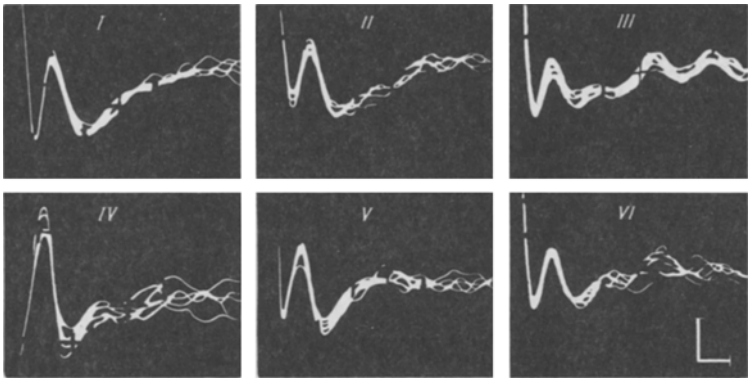

Fig. 1. Changes in parameters of DP depending on body temperature and voltage of stimulating current. I, II, III) DPs with output voltage of 3 V and body temperature of 38.5, 42, and 43°C respectively; IV, V, VI) DP with output voltage of 6 V and body temperature of 38.5, 42, and 43°C respectively. Calibration: amplification 100 μV, time marker 10 msec.

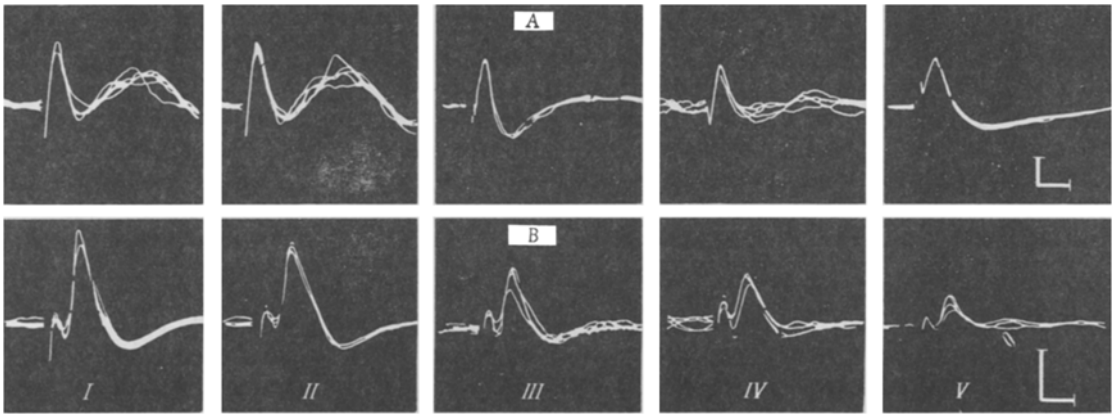

Fig. 2. Changes in parameters of DP and TCR during hyperthermia. A) DP; B) TCR. I) 39°C, II) 39°C, III) 40°C, IV) 42°C, V) 43.4°C. Calibration: amplification 100 μV, time marker 10 msec.

distance of 2 mm were implanted subdurally into strictly symmetrical areas of the sensomotor cortex of both hemispheres. The cortex was stimulated through bipolar electrodes with single square pulses 0.05-0.1 msec in duration from an ÉSU-1 electronic stimulator with radiofrequency outputs. A monopolar recording technique was used with the reference electrode inserted into bone over the frontal sinus. To reduce the loop of stimulating current a scheme of stimulation described by Roitbak [6] was used. Bioelectrical responses were recorded from the screen of a two-channel cathode-ray oscilloscope by superposition during stimulation of the cortex at a frequency of once every 5 sec. Upward deflection of the beam corresponded to negativity beneath the active electrode.

Hyperthermia was produced by placing the waking rabbit in a heat chamber where the air temperature was maintained automatically at 45°C. The degree of hyperthermia was recorded by an electrothermometer inserted into the rectum to a depth of 5 cm. Dendritic and transcallosal potentials were investigated 5 min after the animal had been placed in the chamber, when its body temperature had risen by 0.5-1 and 2-3°C, and in the terminal phase of hyperthermia when the rectal temperature was 42.6-43.4°C. The period of recovery of the parameters of the cortical electrical responses was studied after preliminary elevation of the body temperature to 42°C, after which the hyperthermia was immediately stopped. The results were subjected to statistical analysis on the "Odra" computer.

TABLE 1. Dynamics of Amplitude Parameters of DP and TCR in Sensomotor Cortex during Hyperthermia (M±m)

Rectal temperature, °C		DP	TCR	
		amplitude of negative phase of response, μV	amplitude of positive phase of response, μV	amplitude of negative phase of response, μV
39		291,0±13,8	133,9±7,9	226,1±16,7
39		277,1±11,1	118,7±5,6	212,2±19,8
	P	>0,05	>0,05	>0,05
40		257,6±13,0	99,4±8,0	168±9,1
	P	>0,05	>0,05	<0,05
41		201,6±9,8	80,3±7,2	133,7±9,1
	P	<0,001	<0,001	<0,005
42		161,1±7,4	69,8±6,4	100,4±7,1
	P	<0,001	<0,001	<0,001
43		122,7±5,8	42,2±5,4	76,4±7,1
	P	<0,001	<0,001	<0,001

Legend. Mean results of 10 experiments shown.

EXPERIMENTAL RESULTS AND DISCUSSION

The dendritic potential (DP) in the waking rabbits consisted of a negative wave with a latent period of 0.5-1.5 msec and an amplitude of 250-350 μV, and with a duration of not more than 20 msec. The transcallosal response (TCR) consisted of a positive–negative complex with a latent period of 3-4 msec, and with an amplitude and duration of the negative component of 200-250 μV and 15-20 msec respectively. The principal characteristics of the DP and TCR recorded in these experiments were similar to those described elsewhere [1, 2, 6, 9, 12, 14].

In the initial state doubling the intensity of the stimulus compared with the background value caused an increase in amplitude of the DP by 180-200%. With an increase in the body temperature to 42°C and, in particular, in the terminal stage of hyperthermia, this effect no longer exceeded 20-40% (Fig. 1).

The increase in amplitude of DP during an increase in the strength of cortical electrical stimulation is known to be due chiefly to the involvement of a larger number of synaptic contacts, converging on different cortical cells, in the excitation process [6]. It can accordingly be postulated that the observed effect is largely due to the inhibitory influence of the high temperature on synaptic transmission of excitation.

The action of a high temperature is accompanied by regular changes in cortical potentials. The dynamics of amplitude parameters of DP and TCR during hyperthermia is illustrated by Table 1. DP and TCR 5 min after the animals have been placed in the heat chamber, when their body temperature remains the same as before, were characterized by comparatively stable parameters (Fig. 2). However, after an increase in the rectal temperature by 0.5-1°C a tendency was observed for the amplitude of DP and TCR to fall; the latent period, duration, and configuration of the cortical electrical responses, however, showed no substantial change. With a further disturbance of the temperature regime, starting with a rise of body temperature above 40°C, reduction of DP and TCR continued progressively, but their parameters showed definite quantitative differences. When the body temperature rose to 42°C and, in particular, in the terminal phase of hyperthermia the amplitude of TCR as a rule was depressed much more than the amplitude of DP. Comparison of the rate of extinction of the cortical responses to direct and transcortical stimulation in the agonal stage of hyperthermia showed that the DP was most resistant. The higher sensitivity of TCR than of DP observed during a disturbance of the temperature regime was evidently due to the participation of spike activity of the bodies of the cortical neurons (beneath the stimulating electrodes) in the formation of the TCR.

Further investigations were aimed at studying the evolution of evoked cortical electrogenesis during elevation of the body temperature to 42°C and subsequent cessation of hyperthermia. As the results of 10 experiments showed, normalization of the heat balance after a high degree of hyperthermia was not accompanied by synchronized restoration of the postsynaptic activity of the different cortical cells. Not until 24 h after normothermia of the body had been reached were the parameters of DP and TCR virtually indistinguisable from those found previously.

When analyzing the possible causes of inhibition of the cortical dendritic apparatus it can be assumed that the depression of DP observed during the action of heat may be due to functional inhibition of axodendritic synapses, through which the apical dendrites are depolarized under the influence of nonspecific afferent impulses primarily from the brain-stem reticular formation, in the plexiform layer of the cortex. Evidence of the mainly direct inhibitory effect of a high temperature on postsynaptic activity of the special dendrites is given by the observations of Chang [11], who found reduction of the DP during local elevation of the cortical temperature above 40°C. The changes observed in the parameters of DP could be connected with a decrease in the sensitivity of the postsynaptic chemoreceptive membrane to the action of the mediator or (and) it might be the result of a decrease in the number of quanta of mediator liberated in response to direct electrical stimulation of the cortex during hyperthermia.

On the basis of the view that the formation of the TCR is unconnected with the activity of subcortical structures [12] and reflects processes of synaptic transmission of excitation entirely at the cortical level, it can be postulated that the observed depression of TCR is a reflection of the direct inhibitory action of heat on neurons in the various layers of the cortex. The direct sensitivity of cortical neurons to the effect of high temperature is confirmed in experiments to study the bioelectrical activity of an isolated strip of cortex [5]. The changes in DP and TCR may thus be evidence of a direct cortical mechanism for the action of heat and they may be linked with the rise of body temperature and with the functional characteristics of the brain.

LITERATURE CITED

1. T. M. Darbinyan and V. B. Golovchinskii, Mechanisms of Narcosis [in Russian], Moscow (1972).
2. V. V. Zakusov and R. U. Ostrovskaya, Byull. Éksp. Biol. Med., No. 11, 85 (1967).
3. Z. A. Iskhakov, in: Electrophysiology of the Nervous System (Proceedings of the 4th All-Union Electrophysiological Conference) [in Russian], Rostov-on-Don (1963), p. 73.
4. O. V. Levchuk, Zh. Vyssh. Nervn. Deyat., No. 4, 717 (1966).
5. V. D. Lindenbraten, Patol. Fiziol. Éksp. Ter., No. 3, 79 (1972).
6. A. I. Roitbak, Bioelectrical Phenomena in the Cerebral Cortex [in Russian], Tbilisi (1965).
7. B. A. Saakov, Mechanisms of General Complications of Thermal Injuries [in Russian], Kiev (1963).
8. Z. T. Tursunov and M. V. Abdusamatova, Abstracts of Proceedings of the 2nd Conference of Physiologists of Uzbekistan [in Russian], Tashkent (1973), pp. 235-236.
9. L. M. Chuppina, Fiziol. Zh. SSSR, No. 6, 686 (1969).
10. I. K. Yaichnikov, "Analysis of the effect of an increased temperature on brain electrical activity in rabbits," Author's Abstract of Candidate's Dissertation, Leningrad (1973).
11. H. T. Chang, Cold Spring Harbor Symp. Quant. Biol., 17, 189 (1952).
12. H. T. Chang, J. Neurophysiol., 16, 117 (1953).
13. H. Gäenshirt, W. Krenkel, and W. Zylka, Electroencephalogr. Clin. Neurophysiol., 6, 409 (1954).
14. B. Grafstein, J. Neurophysiol., 22, 504 (1959).
15. M. Lennox, W. Sibley, and H. Zimmerman, J. Pediatr., 45, 179 (1954).