COMPARATIVE STUDY OF THE EFFECT OF DECEREBRATION,
HEXOBARBITAL, AND ETHER ON THE CONSTANT POLARIZATION
POTENTIALS OF THE CAT'S BRAIN

M. Kh. Starobinets

UDC 612.822.3-06; [612.82-089+612.82.014. 46; [615.787+615.781.41

The comparative investigation of the cortical electrical activity during barbiturate and ether anesthesia reveals differences in the electroencephalogram (EEG) in these conditions [6, 8]. Intercollicular division of the brain stem causes the same changes in the EEG as sedative doses of barbiturates [7]. It has been shown by the method of recording the constant potentials that barbiturates produce hyperpolarization and ether—depolarization of the cerebral cortex [4,9]. No agreement has however been reached regarding the cause of the differences in the electrophysiological characteristics of barbiturate and ether anesthesia.

The object of the present investigation was to make a comparative study of the changes in the constant polarization potential (CPP) and the EEG of the cerebral hemispheres of the cat during production of hexobarbital anesthesia, during administration of narcotic doses of ether by inhalation and by subcutaneous injection, and after decerebration. The effect of ether and asphyxia on the CPP of the cerveau isolé preparation was also studied.

EXPERIMENTAL METHOD

Investigations were carried out on tracheotomized cats. The CPP of the sensorimotor area of the cortex were recorded by means of nonpolarizing electrodes (Zn-ZnSO₄), and the wick of the indifferent electrode was fixed to the nasal bone from which the periosteum had been stripped. The CPP were recorded by a compensation method (accuracy of measurement up to 0.1 mV) by means of a high-ohmic potentiometer and a mirror galvanometer. In some experiments the CPP were recorded automatically by means of a type N-373-1 dc self-writing apparatus. The EEG of the sensorimotor cortex was recorded periodically.

EXPERIMENTAL RESULTS

The effect of decerebration on the CPP of the cerebral hemispheres was studied in 16 cats. In 15 experiments during the intercollicular division of the brain stem an electropositive jump of the potentials by 2-7 mV was observed, followed by a slow decrease over a period of 3-8 min to the initial level and sometimes below it. This electropositive wave is not an artifact because cutting with a scapel when the hand was electrically insulated, and with a bone knife produced this phenomenon equally. After restoration of the original value of the CPP, a prolonged secondary hyperpolarization developed $(2.0-6.2 \text{ mV} \cdot \text{mean } 3.96\pm0.44 \text{ mV})$, decreasing after 40-90 min. The hyperpolarization observed during division of the brain stem coincided in time with the appearance of slow waves on the EEG, interrupted by bursts of spindles (Fig. 1). Only in one experiment was no change in the level of polarization of the cortex caused by division of the mesencephalon.

The effect of hexobarbital on the CPP of the cerebral hemispheres was investigated in 10 cats. Because of the massive doses of the drug injected (200-300 mg/kg) a state of deep anesthesia lasting many hours was produced. In all the experiments, 5-20 min after injection of the hexobarbital the animals developed hyperpolarization of the cortex. The hyperpolarization increased during the next 15-50 min and reached 1-3 mV (mean $2.12\pm0.22 \text{ mV}$), and remained at this level for 20-80 min. Later, as a rule, the magnitude of the electropositive potential decreased, and somethimes fell below the initial level (Fig. 2).

Department of Human and Animal Physiology, O. V. Kuusinen Petrozavodsk University (Presented by Active Member of the Academy of Medical Sciences of the USSR S. R. Mardashev). Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 63, No. 3, pp. 19-22, March, 1967. Original article submitted July 30, 1965.

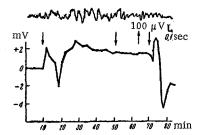


Fig. 1. Effect of intercollicular division of the brain stem on the constant polarization potentials of the cat's cortex and the subsequent action of ether and asphyxia on the level of polarization of the cortex of a cerveau isolé preparation. Along the axis of abscissas - time (in min); along the axis of ordinates -level of polarization (in mV). Bottom curve -CPP of cerebral cortex, top curve -EEG. Arrows: 1st-decerebration, 2nd - beginning of inhalation of ether, 3rd-end of inhalation of ether, 4th - compression of the trachea (asphyxia).

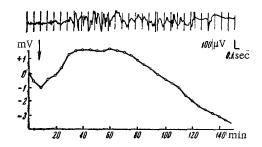


Fig. 2. Effect of hexobarbital on the constant polarization potentials of the cat's cerebral cortex. Arrow—injection of hexobarbital. Remainder of legend as in Fig. 1.

In all the experiments a constant correlation was observed between the level of polarization of the cortex and the EEG. Slow waves appeared on the EEG before the increase in the electropositive potential. In the period of hyperpolarization, however, the amplitude of these slow waves increased and spindles periodically appeared. With a decrease in the hyperpolarization the bioelectrical activity of the brain was

depressed. In the experiments in which hyperpolarization changed into depolarization, the EEG consisted of the isoelectric line, interrupted by occasional "discharges" with a frequency of 0.5-2 per second (Fig. 2).

Against the background of hyperpolarization of the cortex caused by hexobarbital, when small chronic convulsions were observed although the animal did not awake following injection of bemegride—a barbitu-rate antagonist—in a dose of 5-6 mg/kg, the EEG showed an increase in the frequency of the rhythm, and a decrease in the amplitude of the waves. In 6 of the 10 tests in which bemegride was injected the level of the CPP fell, while in 4 tests the development of hyperpolarization stopped.

The effect of inhalation of ether on the level of the cortical CPP was studied in 22 test on 12 cats. In all cases anesthesia caused by inhalation of ether was accompanied by marked depolarization of the hemisphere (from -0.7 to - 7 mV, mean -2.91±0.43 mV). Depolarization appeared 1-3 min after the inhalation of ether was started and it was maintained throughout the period of anesthesia (5-15 min). When the inhalation of ether was discontinued, the CPP of the brain returned to their initial level after 2-5 min (Fig. 3). During the depolarization caused by ether anesthesia, a slowing of the rhythm of the electrical activity and an increase in the amplitude of the waves were observed on the EEG. However, these changes were much less marked than during barbiturate anesthesia.

Because the inhalation of ether is accompanied by irritation of the receptors of the respiratory passages, 10 experiments were carried out in which a narcotic dose of ether was injected subcutaneously. In 9 experiments, 13-20 min after injection of an ether-oil mixture (8-10 ml/kg body weight), depolarization of the cortex developed (from -1.6 to -5.1 mV, mean -3.12±0.31 mV). This depolarization lasted for 20-60 min, throughout the period of narcotic sleep. When the animal awoke the CPP returned to their original level. Only in one experiment did the subcutaneous injection of ether cause an increase in the electropositive potential of 3.4 mV.

In experiments on cats with cerveau isolé, inhalation of ether was given against the background of maximal hyperpolarization of the cortex, and also during the period of decrease of the electropositive potential and of repolarization. In all these 20 tests the level of the CPP and the pattern of the EEG was unchanged by the action of ether (Fig. 1).

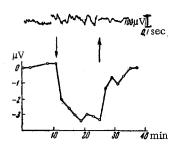


Fig. 3. Effect of ether on the constant polarization potentials of the cat's cerebral cortex. Arrows: 1st-beginning of inhalation of ether, 2nd-end of inhalation of ether. Remainder of legend as in Fig. 1.

Eight tests were performed on the same decerebrate cats in which the trachea was compressed to study the effect of asphyxia on the CPP of the "sleeping brain" preparation. In all the experiments the level of polarization of the cerebral hemispheres exhibited changes of phase. Initially it changed toward hyperpolarization, from 2.1 to 5.5 mV (mean 3.07 ± 0.58 mV), but 2-5 min later the electropositive potential fell sharply by 6.1-9.5 mV (mean 8.1 ± 0.39 mV: Fig. 1).

A fact which deserved attention was the uniform direction of the change in the level of the CPP of the cortex during barbiturate anesthesia and in the cerveau isole preparation. The hyperpolarization observed in these states, taken in conjunction with the uniform changes on the EEG, suggests that barbiturate anesthesia develops on account of exclusion of the tonic influence from the reticular formation. Barbi-

turates have a direct action on the cortical elements only in the late stages of anesthesia, as is confirmed by the secondary depolarization reported by several authors [1,3] and by myself, coinciding in my investigations with the period of depression of the fast electrical activity.

The depolarization of the cortex during ether anesthesia was not due to irritation of the receptors of the cranial nerves, because it developed also after subcutaneous injection of the ether-oil mixture. In this respect my results conflict with those obtained by A. I. Volegov [2]. Since inhalation of narcotic doses of ether was not reflected in the level of the cortical CPP of the cerveau isolé preparation, while asphyxia usually had a negativizing effect [5, 10], it can be assumed that ether anesthesia is due to the action of the volatile anesthetic on the reticular formation of the brain stem. However, a final solution of this problem of the character and localization of the action of ether must await further investigation.

LITERATURE CITED

- 1. I. A. Arshavskii, Uspekhi Sovr. Biol., 41, No. 2, 193 (1956).
- 2. A. I. Volegov, In the book: Electrophysiology of the Nervous System [in Russian], p. 82, Rostov-on-Don (1963).
- 3. I. P. Mezhulis, In the book: Problems in Balneology [in Russian], 4, 227, Riga (1957).
- 4. O. P. Minut-Sorokhtina, G. N. Sorokhtin, and Yu. B. Temper, Fiziol. Zh. SSSR, No. 6, 638 (1962).
- 5. O. P. Minut-Sorokhtina, G. N. Sorokhtin, and Yu. B. Temper, Fiziol. Zh. SSSR, No. 8, 893 (1962).
- 6. E. D. Adrian and B. H. C. Matthews, J. Physiol. (London), 81, 440 (1934).
- 7. F. Bremer, C. R. Soc. Biol., 122, 460 (1936).
- 8. A. Forbes, A. J. Derbyshire, and B. Rempel, et al., Am. J. Physiol., 113, 43 (1935).
- 9. S. Goldring, J. S. Metcalf S. H. Huang, et al., J. Nerv. Ment. Dis., 128, 1 (1959).
- 10. W. Weinstein, J. H. Kendig, S. Goldring, et al., Arch. Neurol. Chic., 4, 441 (1961).