EFFECT OF HYPERVENTILATION ON CHRONAXIE OF THE MOTOR CORTEX

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Hyperventilation is shown to cause an increase in chronaxie of the cortex.

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The effect of hyperventilation on cerebral cortical function has been studied chiefly from the aspect of changes in its electrical activity [1, 2, 9, 10]. We know of no investigations of the effect of hyperventilation on the chronaxie of the cortex. There are isolated reports of an increase in the motor chronaxie of muscles and also of the cutaneous and optic chronaxie under the influence of hyperventilation [7, 8].

It was therefore of interest to investigate the chronaxie of the motor cortex and to study its changes in relation to changes in the ventilation of the lungs in the conditions of an open and closed thorax.

EXPERIMENTAL METHOD

Sixteen acute experiments were performed on adult dogs weighing from 6-25 kg anesthetized with morphine and urethane. The skull was trephined in the region of the sigmoid gyri; the dura was opened, and the exposed area of the brain was covered with a swab in warm physiological saline. The chronaxie was determined by means of a condenser chronaximeter, starting 30-40 min after trephining. The indifferent electrode (anode) was fixed to a shaved area of the animal's body; the active electrode (cathode) was applied to a point on the brain in the region of the sigmoid gyrus corresponding to the neck muscles. The rheobase was determined before and after investigation of the chronaxie; values of the chronaxie were taken into consideration when the theobase values coincided or differed by not more than 3 V. Each determination of rheobase and chronaxie was undertaken 3 min after a change in the operating conditions of the artificial respiration apparatus. The brain temperature, tracheal respiration, and arterial pressure were recorded throughout the experiment. In nine experiments the composition of the blood gases was determined by Van Slyke's method. Blood was taken from the carotid artery and jugular vein.

EXPERIMENTAL RESULTS

Results of experiments with typical changes in chronaxie under the influence of hyperventilation of the lungs to an extent of 50-100% above the normal pulmonary ventilation are given in Table 1. They show that apnea developed in the experiments as a result of a small decrease in the CO_2 concentration in the arterial blood (by not more than 1%). However, the chronaxie was increased every time during apnea caused by hyperventilation. These changes in chronaxie were regularly found in connection with hyperventilation, and they were observed also in our other experiments.

On changing from hyperventilation to natural (in closed chest experiments) or to the initial volume of ventilation of the lungs (in open chest experiments), the chronaxie was restored to the original (or close to the original) values.

The mean chronaxie during normal respiration was 0.13 msec, rising to 0.154 msec during hyperventilation ($P < 0.05$). The difference was significant if the results of the two groups of experiments (open and closed chest) were analyzed separately or together.

No direct relationship could be found between the changes in chronaxie and the CO_2 concentration in the arterial blood. The degree by which the chronaxie was lengthened during hyperventilation varied with

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TABLE 1. Chronaxie of Motor Cortex and Composition of Blood Gases during Normal Pulmonary Ventilation and Hyperventilation

Expt. No.	Experimental conditions	Rheobase (in V) before and after determining chronaxie	Chronaxie (in msec)	Content of gases in arterial blood (in %)		Respiration rate per min	Pulse rate per min	Arterial pressure (mm Hg)
				CO_2	O_2			
2	Natural respiration	69; 69 70; 70	0.144 0.140	37.98	22.87	16	100	100-94
	Hyperventilation 800 cm^3	50; 49 49; 49 45; 45	0.156 0.160 0.160	37.18	24.59	Apnea	80	100-102
	Natural respiration	62; 60 60; 60	0.148 0.148	37.98	23.36	16	120	84-88
	Hyperventilation 800 cm^3	49; 47 45; 46	0.168 0.168	–	–	Apnea	140	72-80
	Natural respiration	62; 60 62; 60	0.144 0.148	–	–	18	140	100
	Hyperventilation 700 cm^3	70; 70	0.156	–	–	14	120	–
3	Natural respiration	45; 46 46; 44	0.180 0.180	35.14	22.05	12	160	84
	Hyperventilation 700 cm^3	50; 52 54; 52	0.212 0.210	34.61	23.03	Apnea	140	56
	Natural respiration	50; 49 48; 49	0.188 0.192	35.94	22.05	12	140	74
	Hyperventilation 700 cm^3	54; 60 60; 60	0.264 0.260	–	–	Apnea	140	42
	Natural respiration	56; 55 54; 55	0.200 0.200	–	–	9	140	80

the volume of lung ventilation. This was seen in experiments in which the lungs were ventilated artificially. For example, during hyperventilation at the rate of 15 liters/min the chronaxie of the motor cortex was 0.176-0.172 msec, and with hyperventilation of 10 liters/min it fell to 0.156 msec. The brain temperature remained unchanged.

Comparison of the observed changes in chronaxie and variations of the arterial pressure showed that in eight experiments the increase in chronaxie during hyperventilation was accompanied by a decrease in arterial pressure by 10-52 mm, in two experiments the arterial pressure rose by 15-28 mm, and in two it was unchanged. The consistent increase in chronaxie during hyperventilation was thus observed when the arterial pressure changed in different directions, and it was therefore not due to changes of arterial pressure.

Hyperventilation of the lungs caused a decrease in the CO_2 concentration in the arterial blood and, as a result, the rhythmic activity of the respiratory center ceased and apnea supervened. Meanwhile the afferent flow from the respiratory center to the cortex ceased. In our earlier investigations we found that during inhibition of the respiratory center, when afferent influences of the respiratory center on the cortex were removed, the chronaxie of the motor cortex was lengthened [5, 6], while the cortical electrical activity was depressed both during inhibition of the respiratory center caused by stimulation of the central end of the divided vagus nerve and during hyperventilation apnea [4].

In the present investigation the increase in chronaxie of the motor cortex could have been due either to a direct lowering of the CO_2 concentration in the arterial blood or to abolition of afferent impulses from the respiratory center as a result of apnea.

There are reports in the literature of the appearance of slow waves on the electroencephalogram during hyperventilation [9]. Our investigations [3] also showed that in hyperventilation apnea, besides depression of cortical electrical activity, fast waves disappear from the EEG and slow waves appear. These changes in chronaxie and in the EEG, illustrating a decrease in cortical lability, are due both to the direct effect of a fall in the blood CO_2 concentration and to removal of the influence of afferent impulses from the respiratory center.

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