EFFECT OF REVERSIBLE EXCLUSION OF THE ANTERIOR PART OF THE CEREBRAL CORTEX ON REGULATION OF ARTERIAL PRESSURE AFTER BLOOD LOSS

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Stimulation of various parts of the cerebral cortex may produce different patterns of change in the circulation [4, 7, 9, 15-17], and views have been expressed in the literature concerning localized vascular centers [3], the inclusion of cortical cells in the arc of unconditioned circulatory reflexes [1], and the absence of cortical control of vascular tone [2, 10, 11].

The authors have attempted to study this problem by investigating the effect of a reversible cold block of the motor and premotor areas on the dynamics of recovery of the arterial pressure after blood loss. These zones are one of the three cortical regions influencing autonomic functions [19]. In control experiments the cold block was applied to the posterior portions of the hemispheres, which are "silent" so far as vascular effects are concerned [7].

EXPERIMENTAL METHOD

Experiments were carried out on 37 cats. The motor and premotor cortex was subjected to a reversible bilateral functional block by passing cold water (2-6°) through the frontal sinus, and the inner wall of which in the cat is in direct contact with the sensorimotor and premotor areas of the cortex. The operation of preparing the sinus and inserting the Plexiglas plug with the afferent and efferent tubes was carried out 3-5 days before the acute experiment. The communication between the sinus and the nose was blocked with acrylic glue. The criterion of the functional blocking of the appropriate areas of the cortex was an increase in the threshold of the motor reaction to stimulation of the projection zone of the forelimb through epidural silver electrodes. The occipital regions of the cortex were cooled by means of chromium-plated copper capsules 10 and 15 mm in diameter, implanted above the posterior portions of the hemispheres [6]. The degree of cooling was judged from the readings of graduated thermocouples inserted above the surface of the cooled and uncooled parts of the cortex. In the acute experiments the vessels were dissected under transient ether anesthesia reinforced by injection of curare-like drugs and artificial respiration. Bleeding to the extent of 1-2.5% of the body weight was carried out from the femoral artery, and reinfusion took place into the femoral vein. The arterial pressure was recorded with a mechanically sensitive pickup [5] connected to a loop oscillograph. Bleeding began 10 min after cold was applied to the cortex.

EXPERIMENTAL RESULTS

The control experiments showed that 10 min after the beginning of cooling of the cortex the threshold of the motor reaction was increased 3-6 times. In the first 2-3 min of cooling the increase in threshold took place faster than subsequently. The temperature of the appropriate portions of the cortex was reduced by 12-15° at the 7th-8th min of cooling, and was maintained steadily at this level during 30-40 min of cooling. When cooling stopped, the original threshold of the motor reaction and the original brain temperature were restored after 3-5 min.

A preliminary series of experiments on 15 cats showed that during repeated (as many as 60) bleedings and reinfusions, with or without hexobarbital anesthesia, and also after bilateral denervation of the carotid reflexogenic zones and bilateral vagotomy, the arterial pressure fell in the course of each bleeding to the same level and returned during each reinfusion to the original level, with a maximal deviation of

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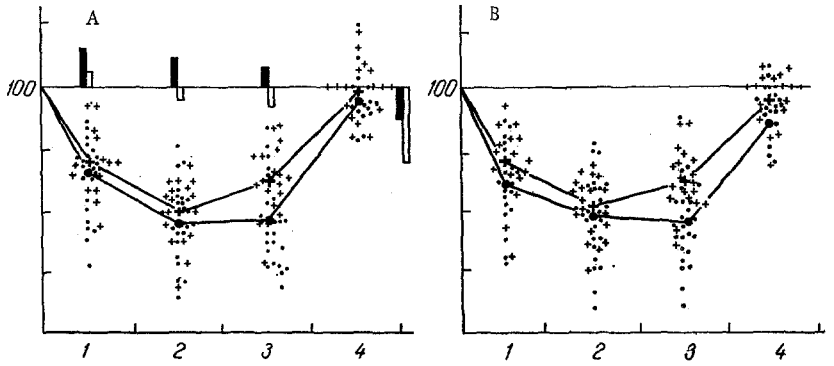

Fig. 1. Dynamics of systolic (A) and diastolic (B) pressure, taken as 100%, and pulse rate during bleeding and reinfusion. The dots denote the results of individual measurements of the pressure with an "intact" cortex and the corresponding mean values; the crosses, the same after cold blocking of the anterior portions of the cortex. The black columns in A denote the pulse rate with an "intact" cortex; the white columns, the same after cold blocking of the anterior part of the cortex. All indices are expressed as percentages of the corresponding initial values (before bleeding). 1) 15 sec after beginning of bleeding; 2) toward the end of bleeding; 3) 5 min after end of bleeding; 4) after reinfusion of blood.

±10-12%. This meant that the bleeding and reinfusion could be repeated over and over again in the experiments with functional blocking of the cortex, for the repetition of the bleeding, provided that the whole of the removed blood was reinfused, was not reflected in the values of the blood pressure.

Cooling of the anterior portions of the cortex led in 10 of the 12 experiments to a more rapid recovery of the arterial pressure after bleeding to the extent of 1-1.5% of the body weight. As the figure shows, when the anterior part of the cortex was blocked, the mean values of the systolic and diastolic pressures 15 sec after the beginning of bleeding and near its end were 3-7% higher than before the action of cold. The clearest (and statistically significant) differences were observed in the 5-min interval between the end of bleeding and the beginning of reinfusion. During this time the systolic pressure of the animals with an "intact" cortex hardly increased, while the diastolic pressure continued to fall,falling from 57.4 to 54.8% of its original value before bleeding. Against the background of the cold block of the anterior part of the cortex, the systolic pressure of these same animals rose from 59.6 to 70% and the diastolic pressure from 60.5 to 69.1%. The difference between the magnitude of the spontaneous rise of arterial pressure with or without the cold block of the cortex in the 5-min interval between the end of bleeding and the beginning of reinfusion averaged 13.1 ± 3.42% of its initial level for the systolic pressure and 14.1 ± 3.28% for the diastolic (analysis of the corresponding data by means of the t-test; $P < 0.002$). The pulse pressure recovered by 11.8% of its initial value in the same time interval after the cold block of the anterior portion of the cortex, but by only 2.1% without the block.

Bleedings repeated 30 min after the cessation of cooling followed the same course as the bleedings before cooling: hardly any spontaneous recovery of the arterial pressure took place.

A clearer recovery of the arterial pressure following blocking of the anterior portion of the cortex was associated with the absence of tachycardia, such a characteristic feature of the usual reaction of the cardiovascular system to bleeding, and even with a tendency toward bradycardia. Reinfusion, leading in normal conditions to a slowing of the heart rate, was accompanied by a more marked bradycardia when the motor and premotor areas were blocked (see figure).

If the arterial pressure was below 80-90 mm, blocking of the anterior portion of the cortex did not give rise to a spontaneous increase of pressure, but only a distinct bradycardia. Spontaneous recovery of the arterial pressure after bleeding was absent also against the background of cold blocking of the occipital regions of the hemispheres, although it was accompanied by marked bradycardia.

The results of these experiments may be interpreted formally as the result of liberation of the lower centers of sympathetic vasomotor innervation from the inhibitory influence of the frontal portions of the cortex [4, 14, 18]. However, the absence of tachycardia and, conversely, the tendency for the heart rate to slow during bleedings against the background of blocking of the anterior portions of the cortex do not permit the results to be attributed to an increase in the activity of the sympathetic system. Probably the tone of the vagus nerves was increased at the same time, in accordance with the results of experiments demonstrating the inhibitory effect of the cortex on the vagus innervation of the heart [12]. The results of the present experiments agree with those obtained by Popova [8], who showed that application of 20% KCl solution to the motor cortex of the cat intensifies both the depressor and the pressor responses to stimulation of corresponding points of the medulla. At the same time, these results may be compared with the intensification of the interoceptive reflexes after decerebration [13], demonstrated in Chernigovskii's laboratory, and also with the well-known increase in the proprioceptive reflexes and postural reflexes in lesions of the motor and premotor areas [15, 16]. They do not, however, agree with the views expressed by Belenkov and co-workers [10, 11, 12], who claim that blocking of the motor cortex is not reflected in the reflex regulation of the blood pressure.

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