The results suggest that in experimental hypoparathyroidism there is not only a direct effect of hypocalcemia on transmitter exocytosis, but there are also substantial changes in the apparatus of secretion and, possibly, of accumulation of transmitter and (or) Ca^{2+} by the terminal, secondary to the hypocalcemia. Correlation discovered between disturbances of calcium homeostasis at organism and cell level under these conditions is particularly interesting.

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CHANGES IN AREA OF MYOCARDIAL DAMAGE DURING POSTISCHEMIC REPERFUSION

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KEY WORDS: reperfusion; ischemia; myocardium.

The change in area of myocardial injury during postischemic reperfusion is not only of theoretical, but also of practical interest because of the introduction of thrombolytic and revascularization methods of treatment of ischemia and infarction of coronary origin into clinical practice. Data in the literature on this question are contradictory. Some workers state that after ischemia ranging in duration from 14-30 min [7] to 1-6 h [6] reperfusion leads to a decrease in size of the infarct, whereas others [4] state that reperfusion after ischemia for 5 h causes an increase in size of the infarct in most cases. To test which of these conclusions is correct the investigation described below was carried out.

EXPERIMENTAL METHOD

Experiments were carried out on 88 noninbred male albino rats weighing 180-200 g. At each stage of the experiment 5-8 rats were used. Permanent and transient ischemia were simulated by ligating the left coronary artery 2-3 mm below the left inferior angle of the

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conus arteriosus [2, 3]. In the model of transient ischemia the artery was ligated together with a plastic tube which could easily be removed together with the divided ligature when it was necessary to start reperfusion. The operations were performed under endotracheal ether anesthesia: in one (for permanent coronary occulsion) and two stages (transient ischemia). After each stage of the operation the chest wall was closed without drainage and the rats placed in a large cage in which other rats undergoing the same operation were kept. The animals were killed with ether vapor. To measure the zone of damage the reaction with nitro-BT for dehydrogenase followed by morphometry and weighing of the zone of necrosis was used [8]. The reaction with nitro-BT is based on the fact that it stains normal cardiomyocytes, containing dehydrogenases, blue but does not stain damaged cardiomyocytes. To carry out the nitro-BT reaction, the heart was taken from the chest, washed to remove blood, the left and right ventricles were separated, and the left ventricle divided starting from the apex into transverse segments 2 mm thick. Usually four or five segments were obtained and the last segment could be under 2 mm thick. All segments from one heart were joined together freely by means of a thread passed through their chambers, and immersed in 0.1% nitro-BT solution in phosphate buffer, pH 7.4, for 15 min. Segmental morphometry was carried out with the MBS-1 stereoscopic microscope equipped with ocular micrometer, for which the value of one scale division had previously been determined. The area of the zone of injury of each segment was measured on the lower and upper surfaces (in mm^2). The total absolute area of the zone of injury of the left ventricle was found by adding together the areas of the zone of injury in all segments. The whole area of the left ventricle was found in a similar manner for each segment and subsequently added together. The resulting total absolute area of the zone of necrosis was converted to a percentage relative to the whole area of left ventricular myocardium.

EXPERIMENTAL RESULTS

If the level of occlusion of the left coronary artery remained the same, with an increase in duration of continuous occlusion from 30 min to 1 h and to 2 h the area of the zone of injury increased from 13 ± 1.6 to 41 ± 2.4% ($P < 0.001$) and to 53 ± 2.5% ($P < 0.001$), respectively. If the duration of occlusion exceeded 2 h the area of the zone of injury did not increase further: after 4 h it was 53 ± 1.9%.

These results show that groups (families) of cardiomyocytes differing in resistance to ischemia are evidently present in the zone of occlusion, and they agree with data in the literature on preservation of viable (metabolically active) complexes of cardiomyocytes in the wall of a postinfarct aneurysm formed from scar tissue, comparatively poor in vessels [1, 5].

The time course of changes in area of the zone of injury during reperfusion, following ischemia of conventionally "average" duration (30 min) was phasic in character. Initially the area of the zone of injury increased — from 13 ± 1.6% immediately after ischemia to 40 ± 0.6% ($P < 0.001$) after 30 min of reperfusion, to 41 ± 2.1% ($P < 0.001$) after 1-1.5 h of reperfusion, and to 49 ± 3.3% ($P < 0.001$) after 3.5 h of reperfusion, whereas after 23.5 h of reperfusion it again decreased relatively to 38 ± 0.5% ($P < 0.001$). On reperfusion after longer (1 h or more) previous ischemia, the phasic character of the changes was less marked or absent. In these cases the area of injury reached a maximum soon after the beginning of reperfusion (1 h after ischemia 1 h in duration) from 41 ± 2.42 to 47 ± 1.6%, but later, after 4 h, it had not changed significantly. It can be concluded from these results that after conventionally "average" (30 min) coronary occlusion, leading to ischemia, the initial period of reperfusion until 3.5 h demands special attention in clinical practice, and that the initial increase in the area of damage during reperfusion is partly reversible.

If the area of injury after a combined period of ischemia and postischemic reperfusion (from 1 to 24 h) is compared with the area after ischemia alone, without subsequent reperfusion, for an equal length of time, as a rule the area after combined ischemia and reperfusion is less than that after ischemia alone. For example, the area of the zone of injury after 30 min of ischemia and 1.5 h of reperfusion was 41 ± 2.1%, compared with 53 ± 2.5% after 2 h of continuous ischemia ($P < 0.001$); the area after 30 min of ischemia and 23.5 h of reperfusion was 38 ± 0.5%, compared with 53 ± 1% after 24 h of continuous ischemia ($P < 0.001$), and after 1 h of ischemia and 3 h of reperfusion it was 45 ± 0.5% compared with 53 ± 1.9% after 4 h of continuous ischemia ($P < 0.01$). With an increase in duration of ischemia the area of damage caused by ischemia and reperfusion gradually approached the area caused by ischemia alone of the same duration.

For example, whereas after 30 min of ischemia and 23.5 h of reperfusion the area of injury was 38 ± 0.5%, after 1 h of ischemia and 23.5 h of reperfusion it was 47 ± 1.22%, after 4 h of ischemia and 20 h of reperfusion 52 ± 1.5%, and after 24 h of continuous ischemia 53 ± 1%.

The results are thus evidence that a combination of ischemia lasting between 30 min and 4 h with reperfusion leads as a rule to the formation of a zone of injury that is smaller than or, sometimes, equal to the area of that found after ischemia of the same duration unaccompanied by reperfusion.

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COMPENSATORY RECOVERY OF FUNCTIONS IN CATS AFTER SOMATOSENSORY TRACTOTOMY: ROLE OF THE CEREBRAL COMMISSURES

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There is evidence of the important role of the cerebral commissures in transmission of somatosensory impulses from peripheral receptor to cerebral cortex [1, 8]. Besides classical pathways, primary somatosensory influences have been shown to be conducted through the corpus callosum and interthalamic commissure [2, 3, 6, 7]. It is evident that these commissural pathways can provide the structural basis for compensatory and recovery processes in the CNS when the classical somatosensory pathways are injured.

The object of this investigation was to study the character of compensation of kinesthetic functions in cats when classical commissural projections of the somatosensory system are blocked.

EXPERIMENTAL METHOD

Experiments were carried out on 36 adult cats divided into five groups depending on the character of neurosurgical operations: 1) intact, 2) after division of the right tegmentum mesencephali, 3) after combined division of the right tegmentum mesencephali and left optic tract, 4) after combined division of the left tegmentum mesencephali and left optic tract, 5) after combined division of the right tegmentum mesencephali and cerebral commissures. Divisions of half the tegmentum mesencephali involved the classical somatosensory pathways (spino—cervico—thalamic, posterior funiculus, and spino—reticulo—thalamic tracts). Additional division of the optic tract in the animals of groups 3 and 4 prevented visual control over ipsilateral (group 3) or contralateral (group 4) tegmental part of the body as the result of homonymous tract hemianopia. In the animals of group 5 commissures of the telencephalon, diencephalon, and mesencephalon (corpus callosum, anterior and hippocampal commis-

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