PAF activity in the CSF of animals with blocked axonal transport in corticolumbar projections was determined by biological testing (Table 2).

PAF activity in intact recipients was detected between 2 and 7 days after injection of colchicine.

Unilateral blocking of axonal transport in corticolumbar projections thus leads to the same effects as destruction of brain tissue: formation of PAF, leading to the appearance of an asymmetrical functional state of the segmental apparatus. The results support the previous conclusion that the signal about injury to elements of CNS is disturbance of normal axonal transport of materials.

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MECHANISM OF THE HYPOTENSIVE EFFECT OF PARATHORMONE

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KEY WORDS: parathormone; calcium metabolism; hypotensive effect.

The parathyroid hormone is the most important regulator of calcium metabolism in the body [3]. However, it has recently been shown that it also has many other biological effects. One of these is its ability to induce vasodilatation and to lower the arterial blood pressure (BP) [6, 7-8, 10], which has been found to be true both of synthetic parathyroidin and parathyroid gland extract [13]. Although the hypotensive action of parathormone (PH) is very short in duration, the authors cited above consider that it may have definite biological importance and that it is actually older phylogenetically than its hypercalcemic effect [11].

The mechanism of action of parathyroid hormone on the vascular wall is not yet fully understood. To shed some light on this mechanism the investigation described below was undertaken.

EXPERIMENTAL METHOD

Experiments were carried out on 56 male albino rats weighing 180-200 g under thiopental anesthesia. BP was recorded by the direct method by means of an electromagnetic manometer-polygraph (Thomson, France) in the common carotid artery. Drugs for study were injected into the femoral vein. To block α-adrenoreceptors, droperidol was used [2], and verapamil was used as the calcium antagonist [12]. The action of PH was also investigated after administration of a stable Leu-enkephalin analog (D-Ala^2-Leu^5-Arg^6-enkephalin), capable of inhibiting the adenylate cyclase – cAMP system [9]. The cAMP concentration was determined in weighed samples of the diaphragmatic portion of the aorta by radioimmunoassay using kits from Amersham Corporation (England) and radioactivity was counted on a Mark III scintillation counter (USA). The numerical results were subjected to statistical analysis by Student's t test.

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EXPERIMENTAL RESULTS

Intravenous injection of two activity units of parathyroidin/100 g body weight into the rats caused the mean BP to fall after 1 min by 22.5% of its original value. BP then rose gradually: After 3 min it was already 87.3% of the initial value, and after 5 min it did not differ significantly from the control.

Intravenous injection of the α-adrenoblocker droperidol in a dose of 0.25 mg/100 g body weight into the rats lowered BP by 33.1% 1 min after injection. At the 3rd minute the BP level showed some increase, but it did not regain its original values and remained lowered until the 15th minute of the experiment.

When PH was injected in the same dose 2 min after injection of droperidol the time course of the BP changes was similar in principle to that observed in rats receiving PH alone. BP 1 min after injection of PH was 21.9% below the value recorded 2 min after injection of droperidol, i.e., immediately before the injection of PH. After 3 min BP was 15.2% lower, but after 5 min it did not differ statistically significantly from the value given above.

Preliminary injection of the α-blocker droperidol into rats thus did not alter the hypotensive action of PH. This may be evidence that this effect is not mediated through α-adrenoreceptors. The results of these experiments agree with data obtained by other workers. For instance, experiments *in vitro* on uterine muscle of guinea pigs showed that the hypotensive action of PH is not realized through adrenergic, acetylcholinergic, or histamine receptors [14].

PH exerts its effect on the main target tissues (bone, liver, kidney) through an increase in the intracellular cAMP concentration [4, 5]. Recently, however, evidence has been obtained to show that PH can influence sodium–potassium metabolism, modify cation transport along ionic channels, and thus act on the function of various cells [1].

To elucidate the mechanisms of the hypotensive effect of pH, experiments were carried out in which PH was injected after verapamil and D-Ala^2-Leu^5-Arg^6-enkephalin. The effect of PH on the cAMP content in the arterial wall also was determined.

After injection of the calcium antagonist verapamil (0.125 mg/100 g body weight) BP in the animals, just as under the influence of droperidol, was considerably lowered after 1 min and remained low throughout the subsequent period of observation. Injection of parathyroidin into rats 2 min after verapamil did not lower BP. Since blockade of the Ca channels inhibited the hypotensive effect of PH, this suggests that this effect is mediated through a change in sodium–calcium metabolism.

Experiments to study cAMP in the vascular wall showed that injection of parathyroidin caused an increase in the cAMP concentration in the diaphragmatic portion of the aorta with a maximum after 30 min (from 208.0 ± 36.5 to 367.5 ± 42.4 pmoles/g tissue, $P < 0.05$). The rise in the cAMP level induced by PH was blocked by the Leu-enkephalin analog D-Ala^2-Leu^5-Arg^6-enkephalin (183.6 ± 36.5 pmoles/g tissue, $P > 0.10$ compared with the control). Intravenous injection of D-Ala^2-Leu^5-Arg^6-enkephalin (10 μg/100 g body weight) did not cause any statistically significant changes in the BP level, and the hypotensive effect of PH was not blocked by the Leu-enkephalin analog.

The rise in the cAMP concentration in the vascular wall thus reached its maximum much later than the hypotensive effect of PH, and blocking the rise in the cAMP level did not prevent the fall of BP caused by parathyroidin. The results of these experiments can be taken as evidence that lowering of BP and accumulation of cAMP in the arterial wall under the influence of PH are two unconnected processes. The hypotensive action of PH is evidently realized through changes in sodium–calcium metabolism in the vascular wall and a decrease in the flow of ions into the cells which, in turn, leads to vasodilatation [15].

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CHARACTERISTICS OF THE SODIUM ACCUMULATING CAPACITY OF THE LIVER

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KEY WORDS: liver; sodium; deposition.

The liver is known to participate in regulation of water and electrolyte balance not only as a receptor zone [2, 5, 9-12, 14], but also as a depot organ capable of retaining an excess of water and certain ions [1, 3, 7, 11, 12].

This paper describes a study of the sodium-accumulating capacity of the liver during the creation of sodium concentration shifts in the portal system of different magnitude and duration, and an attempt is made to describe this capacity quantitatively.

EXPERIMENTAL METHOD

In the first part of the investigation, consisting of acute experiments on cats anesthetized with chloralose, the aim was to study the ability of the liver to retain Na^+ during the creation of a temporary but considerable shift of the Na concentration in blood from the portal vein. This shift was created by injecting 2 ml/kg body weight of 3% NaCl solution in the course of 1 min through a catheter fixed in one of the small mesenteric veins. Blood samples were taken simultaneously from the portal vein through an angiostomy cannula and from the posterior vena cava above the liver by venupuncture 30 sec after the beginning of injection of the solution. To prevent dilution of blood flowing from the liver the posterior vena cava was clamped below the liver during blood sampling.

The second stage of the work consisted of acute experiments on dogs (anesthetized with pentobarbital), in whose portal vein sodium shifts similar to those developing under normal physiological conditions during absorption of Na^+ from the alimentary tract, were created artificially [11]. The scheme of the experiments was as follows. After control blood samples had been taken from the portal and hepatic veins and a sample of liver tissue also had been removed, an infusion of 2% NaCl solution was given through a cannula introduced into a mesenteric vein, at the rate of 0.5 ml/min•kg for 15 min. During infusion of the solution at the 5th, 10th, and 15th minutes, and 5 min after the end of infusion of the solution, blood and liver tissue samples were taken. The sodium concentration in the blood plasma was determined by flame photometry, and in the liver tissue by extraction of the undefatted dried sample in 0.75 N nitric acid followed by flame photometry of the extract [8].

EXPERIMENTAL RESULTS

In series I there were 14 experiments. As Fig. 1 shows, the sodium concentration in the portal vein at the time of injection of the solution was considerably raised — up to 190 mM,

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