EFFECT OF PITUITRIN P ON SODIUM AND POTASSIUM CONTENT IN THE RABBIT ARTERIAL WALL

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Experiments on rabbits have shown that intravenous injection of pituitrin (0.8 unit/kg) results in a marked increase in arterial pressure (by 40±13.1 mm Hg) and a statistically significant decrease in the content of sodium, potassium, and water in dry specimens of the wall of the aorta and carotid and femoral arteries. Calculation of the values relative to tissue water showed that the sodium and potassium concentrations in the aortic wall are reduced by the action of pituitrin, while the concentration of these electrolytes in the femoral and carotid arteries remains unchanged.

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Data indicating the participation of electrolytes in the mechanism of contraction of smooth muscle of blood vessel walls are given in the literature [3-9, 11, et al.]. In this connection the rule of electrolytes in the mechanism of action of the hypertensive hormones is interesting.

The object of the present investigation was to study changes in the sodium and potassium concentrations in arterial walls under the influence of pituitrin P.

EXPERIMENTAL METHOD

Experiments were carried out on two groups of rabbits with 14 animals in each group. The rabbits were immobilized with tubocurarine and maintained on artificial respiration. Pituitrin P was injected into the marginal vein of the ear of the experimental group of animals in a dose of 0.8 unit/kg body weight, causing elevation of the arterial pressure. No pituitrin was given to the control animals. The arterial pressure in the left carotid artery was recorded in both groups of rabbits, and the content of sodium, potassium and water was determined in the walls of various arteries. The sodium and potassium concentrations were determined in dried tissues from the aorta and both carotid and femoral arteries. The vascular tissues were taken from the experimental animals at the time of maximal elevation of the arterial pressure caused by pituitrin. Immediately after removal of the vessel, the adventitia was stripped, and the vessels were dried with filter paper, weighed, and dried at 105° to constant weight. The dry tissue was then ground in a mortar and transferred as a powder into flasks containing 0.1 N nitric acid to extract the sodium and potassium. The concentrations of sodium and potassium in the tissue extracts were determined by a flame photometer of type PPF-UNIIZ. The water content in the arterial walls was determined from the difference in weight between the fresh and dried tissues. The significance of differences between results obtained in the experimental and control animals was assessed by Student's method.

EXPERIMENTAL RESULTS

As Table 1 shows, under normal conditions the sodium concentration in the walls of all investigated arteries was high and the potassium concentration lower. The Na/K ratio for the blood vessels was about equal, being 1.86 for the aorta, 1.93 for the carotid artery, and 1.78 for the femoral artery. The content of water in the walls of the aorta and carotid artery was similar, but much lower in the wall of the femoral artery. The arterial pressure in the control animals was 103±13.1 mm Hg.

The initial arterial pressure in the experimental rabbits was the same as in the control animals (103±12.8 mm Hg). Injection of pituitrin raised it to 143±13.1 mm Hg. The sodium and potassium concentrations in the arterial walls, calculated per unit dry weight, fell significantly after administration of

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TABLE 1. Changes in Arterial Pressure and Content of Electrolyte and Water in Arterial Walls of Rabbits under the Influence of Pituitrin P

Material investigated	Inc. in arterial pressure (in mm), M±σ	Statistical index	Na in meq/kg dry wt. of tissue		K in meq/kg dry wt. of tissue		H_2O in g/kg fresh wt. of tissue	
			cont.	expt.	cont.	expt.	control	expt.
Aorta		*n*	14	14	9	9	7	7
		M	348	270	191	139	637	580
		±σ	20,3	25,5	19,3	7,2	30,5	35,0
		Δ		**78**		**52**		**57**
Carotid artery	40±13,1	*n*	9	9	7	7	7	7
		M	340	305	172	145	657	571
		±σ	4,6	16,7	9,2	16,1	55,4	50,4
		Δ		**35**		**27**		**86**
Femoral artery		*n*	10	10	7	7	7	7
		M	343	287	193	180	591	508
		±σ	15	16	3,5	11,2	44,0	43,9
		Δ		**56**		**13**		**83**

Note. Here and in Table 2: n represents number of animals and Δ decrease in electrolyte concentration in experimental animals compared with controls. Significant differences ($P < 0.05$) are given in bold print.

TABLE 2. Changes in Electrolyte Concentration in Arterial Walls of Rabbits under the Influence of Pituitrin P Calculated Relative to Tissue Water

Material investigated	Statistical index	Na in meq/kg dry wt. of tissue		K in meq/kg dry wt. of tissue	
		control	expt.	control	expt.
Aorta	*M*	225	188	121	99
	±σ	5,1	5,4	2,1	2,48
	Δ		**37**		**22**
Carotid artery	*M*	213	217	110	103
	±σ	16,0	9,8	10,0	3,4
	Δ		**4**		**7**
Femoral artery	*M*	235	227	132	145
	±σ	12,9	19,3	12,0	10,5
	Δ		**8**		**13**

Note. In all cases n = 7.

pituitrin. Meanwhile the water content in the wall of all investigated vessels also fell by a significant amount.

Since changes in the content of electrolytes and water in the arterial wall were in the same direction, it was essential to determine whether changes in the electrolyte level were the result of changes in the water content. This is a pertinent suggestion because of published data indicating the participation of pituitrin in water metabolism [1, 2]. To verify it, the results expressed in meq/kg dry tissue were calculated relative to tissue water. These results (Table 2) showed a decrease in the concentration of sodium and potassium in the aortic wall under the influence of pituitrin, but no change in the wall of the femoral and carotid arteries. It may accordingly be considered that the decrease in sodium and potassium concentrations previously observed in the walls of the femoral and carotid arteries, in contrast to their decrease in the aortic wall, is strictly proportional to the decrease in water content in these tissues. This is an interesting fact because the aorta and the femoral and carotid arteries are vessels of different types: the aorta is a vessel of elastic type, while the femoral artery, and, to some extent, the carotid artery contain muscle cells. It can be concluded from these observations that pituitrin affects the electrolyte concentrations differently in the walls of blood vessels of muscular and elastic type.

It can also be concluded from these results that an increase in tone of the arterial walls under the influence of pituitrin, causing elevation of the arterial pressure, is accompanied by a simultaneous decrease in the sodium and potassium concentrations in the wall, associated with a decrease in the water content. The possibility of contraction of smooth muscle during a decrease in the sodium and potassium concentrations in it have also been demonstrated by the experiments of Singh [10], who described contraction of the smooth muscle of the stomach accompanying loss of these electrolytes.

It can accordingly be concluded from these results that, contrary to the opinion of some investigators [5-9, 11, et al.], an increase in arterial tone under the influence of hypertensive hormone can occur without accumulation of sodium in the arterial wall.

LITERATURE CITED

1. A. G. Ginetsinskii, Physiological Mechanisms of Water and Salt Balance [in Russian], Moscow–Leningrad (1964).
2. B. D. Kravchinskii, Modern Essentials of Physiology of the Kidneys [in Russian], Leningrad (1958).
3. E. E. Daniel, O. Dawkins, and J. Hunt, Am. J. Physiol., 190, 67 (1957).
4. E. E. Daniel and O. Dawkins, Am. J. Physiol., 190, 71 (1957).
5. E. E. Daniel, W. A. Dodd, and J. Hunt, Arch. Internat. Pharmacodyn., 119, 43 (1959).
6. S. M. Friedman, M. Nakashima, and C. D. Friedman, Circulat. Res., 4, 557 (1956).
7. S. M. Friedman, M. Nakashima, and C. D. Friedman, Am. J. Physiol., 184, 97 (1956).
8. S. M. Friedman, S. L. Wong, and G. Woo, Proc. Soc. Exp. Biol., (N. Y.), 120, 241 (1965).
9. J. D. Jamieson and S. M. Friedman, Circulat. Res., 9, 996 (1961).
10. I. Singh, Nature, 207, 1300 (1956).
11. L. Tobian and A. Fox, J. Clin. Invest., 35, 297 (1956).