Thus an increase in the taurine concentration in the perfusion fluid before ischemia leads to much higher residual catecholamine levels in the myocardium, and this may prove useful in ensuring more rapid restoration of cardiac function after operations under cardioplegia. The fact that taurine has an antiarrhythmic effect in the reperfusion period after ischemia provides yet another model of pathology in which this particular effect is noted. A similar effect was demonstrated previously in cases of overdosage of catecholamines and cardiac glycosides or potassium deficiency [2, 7]. This effect can be associated only partially with improved preservation of catecholamine reserves in the myocardium. Further research is required to determine the mechanism of action of taurine.

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EFFECT OF PARATHORMONE ON REACTIVITY OF THE RENAL BLOOD FLOW TO VASOPRESSIN IN NORMOTENSIVE AND SPONTANEOUSLY HYPERTENSIVE RATS

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Hypertrophy of smooth muscles in the arteries of the kidneys [13] and other organs [9] is observed in spontaneously hypertensive rats (SHR) in the prehypertensive stage, and when combined with an increase in calcium concentration in them [14], this leads to enhanced contractile activity of these muscles in response to stimulation by vasopressin [7]. Accumulation of calcium and its binding in the cardiomyocytes and smooth-muscle cells of the vessels in primary hypertension is the result of a genetically determined defect of the cell membranes [4]. Lowering of the ionized calcium level [11] and elevation of the parathormone (PTH) concentration in the blood have been observed under these circumstances [10]. It was shown previously in the writers' laboratory that PTH has a hypotensive action in SHR [1]. The same effect of PTH has been described under other conditions by different workers also [12].

The aim of this investigation was to study changes in reactivity of the renal blood flow to various doses of vasopressin (VP) under the influence of PTH in normotensive rats (NTR) and SHR.

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TABLE 1. Effect of Parathormone on Changes in BP and Cortical and Medullary Renal Blood Flow Induced by Vasopressin in Normotensive and Spontaneously Hypertensive Rats (M ± m)

Experimental conditions	Parameter			Background	Doses of vasopressin		
					minimal	average	maximal
			Normotensive rats				
Intact	Blood flow	BP, cortex	M±m	100,7±3,2	102,7±4,9	111,4±5,8	123,2±6,6*
		Medulla	M±m	237±15,2	221,9±13,3	219,5±14,2	216,9±17
			M±m	165,1±19,4	147,9±13,4	148,7±10,8	131±12,4
After receiving parathormone	Blood flow	BP, cortex	M±m	99,6±2,9	111,4±3,3*	119,9±4,1*	123,7±5,4*
		Medulla	M±m	184,9±9,5	184,4±3,8	160,4±8,8	148,8±20,4
			M±m	156,2±11,9	152,1±10,7	146,9±11,2	120,9±13,1
			Spontaneously hypertensive rats				
Intact	Blood flow	BP, cortex	M±m	136,1±5,3	136,9±4,1	147,7±6,3	152,3±6,9
		Medulla	M±m	145±10,8	105,8±6,3*	95,8±6,4*	81,6±5,7*
			M±m	127,9±12,8	121,6±6,3	95,8±5,5	80,9±9,2*
After receiving parathormone	Blood flow	BP, cortex	M±m	131,9±3,9	147,2±3,9*	155,9±4,2*	172,7±3,81*
		Medulla	M±m	137,7±15,2	105,8±10,2	103,3±10,3	79,9±7,2*
			M±m	128,1±9	106,2±9,1	109,5±11,8	94,7±12,1*

Legend. *$p < 0.05$ compared with background.

EXPERIMENTAL METHOD

The experimental animals comprised 20 SHR of the Okamoto-Aoki strain and 20 Wistar NTR aged 6-8 months (males weighing 200-250 g). The experiments were carried out under hexobarbital anesthesia (20 mg/100 g, intraperitoneally) before and 12-14 h after intramuscular injection of PTH (0.2 U/100 g). Changes in blood pressure (BP) and reactivity of the renal blood flow in response to injection of increasing concentrations of VP ("Koch-Light") into the femoral vein in doses of: 0.001 IU/100 g, 0.004 IU/100 g, and 0.008 IU/100 g body weight. The volume velocity of the local blood flow in the renal cortex and medulla was measured by the hydrogen clearance method [2]. The blood flow was expressed in ml/min/100 g weight of kidney tissue. BP was measured electromanometrically in the femoral artery. The numerical data were subjected to statistical analysis on the DVK-3 computer by Student's test.

EXPERIMENTAL RESULTS

The cortical blood flow in intact NTR was found to exceed the medullary blood flow ($p < 0.02$; Table 1), as described in [3]. The blood supply to the renal cortex of NTR was more than 50% greater than in SHR ($p < 0.001$; Table 1). The lower absolute values of blood flow in the different layers of the kidney in SHR can probably be attributed to a twofold increase in resistance of the renal vessels compared with NTR [13] and also to the smaller cardiac output [5].

Injection of PTH caused a decrease in the cortical blood flow of NTR ($p < 0.02$; Table 1), in agreement with data obtained previously in the writers' laboratory, showing an increase in total peripheral vascular resistance against the background of parathormone-induced hypercalcemia in animals of this group [5]. PTH was shown to increase the degree of reduction of the cortical blood flow in response to average and maximal doses of VP (Fig. 1a). Meanwhile PTH reduced the decrease in medullary blood flow following injection of minimal and average doses of VP (Fig. 1b). Against the background of the action of PTH in NTR in response to infusion of VP, the excess of the cortical blood flow over medullary became smaller, probably due to increased prostaglandin E2 synthesis in the medulla [15, 6]. PTH also increased the intensity of pressor responses to VP in NTR (Table 1).

In SHR, PTH reduced the relative degree of lowering of the cortical blood flow in response to injection of all doses of VP (Fig. 1c). In the medulla PTH reduced the relative fall of blood flow in response to injection of average and, in particular, of maximal concentrations of VP (Fig. 1d). Under the influence of PTH, a redistributive response was observed in SHR against the background of the action of VP, in which the medullary blood flow began to exceed the cortical blood flow, and PTH potentiated the pressor responses to VP by a greater degree in SHR than in NTR (Table 1).

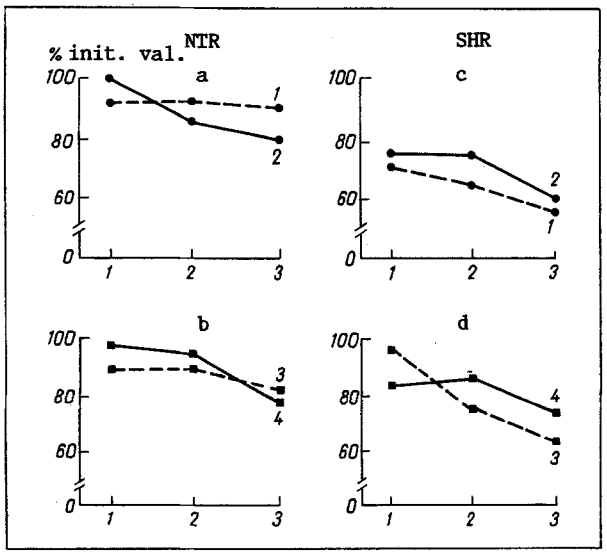

Fig. 1. Effect of parathormone on relative changes in renal blood flow induced by different doses of vasopressin in normotensive (NTR) and spontaneously hypertensive (SHR) rats. Abscissa, changes in blood flow (in percent of initial values); ordinate, doses of vasopressin: 1) 0.001 IU/100 g, 2) 0.004 IU/100 g, 3) 0.008 IU/100 g. 1) Cortical blood flow in intact animals, 2) cortical blood flow in animals receiving PTH (0.2 U/100 g), 3) medullary blood flow in intact animals, 4) medullary blood flow in animals receiving parathormone (0.2 U/100 g).

Thus in NTR differences between the cortical and medullary blood flow are reduced in response to injection of VP preceded by PTH, whereas in SHR under these conditions, the medullary blood flow actually exceeds the cortical a little. The smaller decrease in the medullary than in the cortical blood flow in SHR can evidently be attributed to increased synthesis of prostaglandin E2 in the medulla of SHR compared with NTR [8], and also, probably, to its more significant stimulation by calcium ions and vasopressin [15, 6].

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