THE EFFECT OF THE ADRENAL GLANDS ON CHANGES IN POTASSIUM METABOLISM DURING THE PERIOD OF RECOVERY OF VITAL FUNCTIONS AFTER CLINICAL DEATH

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The mechanism for regulation of the electrolyte balance and, in the first place, potassium-sodium metabolism depends in large degree on the adrenal cortical hormones [1, 8, 14, 17]. Much interest has been aroused in changes in the possible effects of the adrenals on potassium-sodium metabolism during hypoxia, in particular upon clinical death and subsequent resuscitation, which produces, according to several writers [10, 12], hyperkalemia.* In the Soviet literature we find only one article pertaining to this question [2], in which the potassium and sodium concentration of body fluids was investigated.

TABLE 1. Effect of Adrenalectomy and Autotransplantation of the Adrenals on Potassium Concentration in Ordinary Conditions.

Group of animals	Potassium content (mg%)		
	In plasma	In cardiac tissue	In urine
		$M \pm m$	
Control	22,0±1,1	218±11,1	379±52,0
Adrenalectomized	37,2±2,2	224±15,7	568±85,4
P	<0,001	>0,05	>0,05
Animals with autotransplanted adrenal glands	42,2±5,9	204±12,6	414±76,4
P	<0,01	>0,05	>0,05

METHODS

The experiment utilized 66 cats. Adrenalectomy and autotransplantation of the adrenal glands were performed under operative conditions [4].

Autotransplantation of extirpated adrenals was immediately performed in the region of the upper pole of the corresponding kidney [4, 13]. Experiments using animals from this series began 2 weeks after operation. Clinical death (in 36 animals) was produced by lethal phlebotomy and lasted 4 min 30 sec. The usual duration of phlebotomy, which was done fractionally, with a period of hypotension was 14 min. Resuscitation was performed by the method devised by V. A. Negovskii et. al.[9]. Within an hour after the appearance of spontaneous respiration tissues from the cardiac ventricle and brain and as well as blood. (from which the plasma had been removed after centrifugation) were taken. In 30 animals of the control series of experiments plasma, urine and heart tissue were taken under the usual conditions. The concentration of potassium in plasma, urine and heart were determined in the flame photometer, type Leis Model-III.

Control standards, solutions of plasma and urine were prepared by the method of Kravchinskii [5]. Heart tissue was mineralized according to Sal'manovich [11] and made into solution.

RESULTS

The content of potassium in the plasma, urine and heart muscle of normal animals corresponded to data obtained by other authors [3, 11, 16].

In the adrenalectomized animals a marked hyperkalemia was observed (Table 1). This did not appear to be the result of potassium retention in the organism, as the potassium content of the urine not only did not fall in comparsion with controls, but even showed a tendency to rise. The hyperkalemia of adrenalectomized animals neither

*Excess of potassium [Publisher's note].

TABLE 2. Effect of Removal and of Autotransplantation of Adrenal Glands on the Potassium Concentration During Resuscitation After Clinical Death

Object of study	Intact animals		Adrenalectomized		Animals with autotransplanted adrenal glands	
	Control	Resuscitation	Control	Resusicitation	Control	Resuscitation
	Potassium content (M ± m)					
In plasma	22,0±1,1	16,6±1,4	37,2±2,2	29,3±3,9	42,2±5	28,5±3,4
P		<0,01		>0,05		<0,05
In cardiac tissue	218±6,8	286±11,1	224±15,7	258±8,2	204±12,6	258±7,3
P		<0,001		>0,05		<0,01
In urine	379±52	339±42	568±85,4	406±49,8	414±76,4	384>29,6
P		>0,05		>0.05		>0,05

appeared to be a consequence of the exit of potassium from cells in the heart tissue, as its level in the heart remained the same as in the control.

In animals with autotransplanted adrenal glands the hyperkalemia was as marked as in adrenalectomized animals ($P < 0.01$). In these experiments also it did not appear to be either a consequence of potassium retention of the result of a potassium exodus from heart muscle. Evidently, in these instances hyperkalemia may be connected with the outflow of potassium from other tissues not here studied. The presence of an excessively marked hyperkalemia in cats 2 weeks after autotransplantation of the adrenals attests to the clear inferiority of mineralocorticoid function of the transplanted glands. These data do not correspond to data obtained by other authors in experiments on mice, according to which the mineralocorticoid function of autotransplanted adrenals was recovered 2 weeks after operation [7]. The differences, probably, are related to experimentally investigated differences between species.

Under resuscitation of animals having inactive adrenal glands a decrease in the plasma potassium level was observed (Table 2). The results obtained do not correspond to the data on the presence of hyperkalemia in animals undergoing clinical death with a brief preceding period of dying [2, 10, 12]. In subsequent experiments on dogs, in which dying was produced by fractional phlebotomy, no increase in blood potassium was detected, but rather hypokalemia.

In experiments on cats in which dying was prolonged by fractional blood loss, we also observed the development of hypokalemia. The urinary concentration of potassium remained essentially unchanged during this time. During the period of resuscitation a marked increase in the potassium level of the heart tissue took place. It is possible than the decreased blood potassium during resuscitation occurred at the expense of potassium fixation in the structural tissue of the heart [15, 16, 18].

In adrenalectomized animals during resuscitation we observed a tendency toward decreased blood potassium content, although its concentration remained extremely high.

In animals with autotransplanted adrenals a true fall in plasma potassium content was observed during resuscitation in comparison with the level of this index in control animals and a distinct increase in potassium occurred in cardiac tissue.

The changes in potassium content in plasma and in heart tissue which were observed during resuscitation of animals with autotransplanted adrenal glands resembled those changes which occurred during resuscitation of animals with intact adrenals. It may be though that the hormones from the autotransplanted adrenal glands take part in the reactions of potassium transfer from the plasma to heart muscle during resuscitation. Evidently stress activity may strengthen to some degree the mineralocorticoid activity of the autotransplanted adrenal glands, which under usual conditions do not maintain support of normal levels of potassium in the blood.

The data we have obtained attests to the active influence of the adrenal glands on changes in potassium metabolism during the period of recovery of vital functions after clinical death.

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