STATE OF THYROID FUNCTION IN ACUTE ANOXIC ANOXIA

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In rats developing acute anoxic anoxia after a stay of 30 min at a pressure of 360 mm Hg an increase in the concentration of protein-bound iodine was found in the blood (from 2.38 ± 0.2 to 4.09± 0.2 μg%) and morphological changes were demonstrated in the thyroid gland indicating stimulation of its function. Similar results were obtained in rats with pulmonary edema due to ammonia and also in experiments on dogs in which pulmonary edema was produced by administration of chloramine.

Depression of thyroid activity is found in persons living at high altitudes and in animals kept under chronic anoxic conditions [1-8, 11-13]. Insufficient attention has been paid to the study of thyroid function in acute anoxia, and the findings reported in the literature are contradictory.

The object of this investigation was to study thyroid function in acute anoxic anoxia. The level of thyroid function was estimated from the protein-bound iodine of the blood [10] and the morphological picture of the gland.

EXPERIMENTAL METHOD AND RESULTS

Acute anoxic anoxia was produced in 20 rats by keeping them for 30 min in a vacuum apparatus at 260 mm Hg (PO_2 = 70 mm Hg); to allow adaptation of the animals, they were given a rest of 5 min when the pressure had been reduced to 560 mm Hg.

The second model of acute anoxic anoxia was pulmonary edema, which was produced in rats (20 experiments) by intraperitoneal injection of 6% ammonium chloride solution (40 mg/100 g body weight) and in dogs (10 experiments) by intravenous injection of 10% chloramine solution (60 mg/kg body weight). The development of pulmonary edema and its severity were assessed from the pulmonary index (PI) and the microscopic picture of the lungs.

The concentration of protein-bound iodine in the blood (PBI) was determined before and at the height of the pulmonary edema and in the experiments on dogs the oxygen saturation of the arterial blood was also determined by means of a combined oxyhemometer.

In the animals with acute anoxic anoxia produced by "altitude sickness" blood was taken at the end of the experiment for investigation of the PBI, after which the animals were sacrificed and the thyroid gland investigated histologically.

In the rats with acute anoxia due to "altitude sickness" an increase in the PBI concentration was observed (Table 1), and microscopic changes were found in the thyroid gland, indicating stimulation of its function: the colloid in most of the follicles was liquid, it stained a pale-pink color with eosin, and resorption vacuoles were present at the periphery. Desquamation of the thyroid epithelium was observed in the lumen of the follicles (Fig. 1), further histological evidence of increased thyroid function, accompanied by increased liberation of thyroid hormones into the blood stream [9].

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TABLE 1. Concentration of PBI, Pulmonary Index, and Oxygen Saturation of Arterial Blood in Acute Anoxic Anoxia (M ± m)

Experimental conditions	PBI in μg%	Pulmonary index	Oxygen saturation of arterial blood
Exprmts. on rats			
Control	2,38±0,21	0,62±0,4	
Altitude sickness $PO_2 \approx$ ≈70 mm Hg	4,09±0,2 $P_1<0,001$	—	—
Pulmonary edema due to ammonia	5,18±0,24 $P_1<0,001$	1,56±0,16 $P_1<0,001$	—
Exprmts. on dogs			
Pulmonary edema due to chloramine: original data	3,3±0,26 $P_2<0,05$	—	68,1±1,8 $P_2<0,001$
expt.	4,4±0,28		35,7±3,4

Note: P_1 calculated relative to control; P_2 calculated relative to initial state.

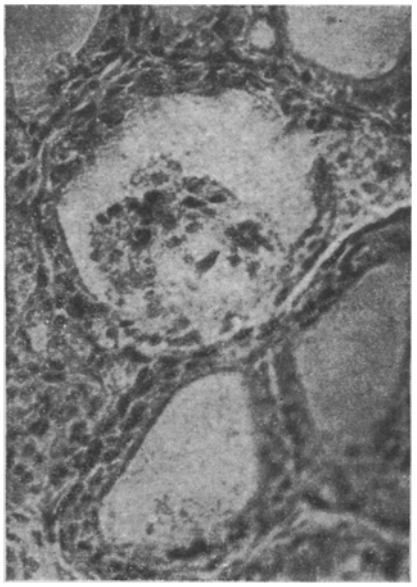

Fig. 1. Thyroid gland of rat with experimental altitude sickness. Hematoxylin-eosin, MBI-6, 560×.

Injection of ammonium chloride led to toxic pulmonary edema in all the rats, accompanied by an increase in the pulmonary index and also by changes in the microscopic picture characteristic of pulmonary edema. Just as during "altitude sickness," the PBI concentration was higher than in the control.

In the dogs with acute anoxic anoxia, when the oxygen saturation of the arterial blood had fallen from 68.1 ± 1.8 to 35.7 ± 3.3%, an increase in the PBI concentration was observed, just as in the experiments on rats. Microscopic examination of the thyroid gland revealed the changes described above, evidence of stimulation of the function of the gland.

The results thus demonstrate increased function of the thyroid gland in acute anoxic anoxia. The stimulation of the thyroid gland, the hormones of which modify the intensity of metabolism, observed in these experiments is evidently a component of the complex adaptive reactions of the body maintaining the constancy of the internal medium in acute anoxia.

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