

EFFECT OF HYPERFUNCTION ON THE CATECHOLAMINE CONTENT IN HEART MUSCLE WHEN THE THYROID HORMONE BALANCE IS DISTURBED

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Hyperfunction of the heart caused by coarctation of the abdominal aorta in animals receiving moderate doses of thyroid (20 mg/100 g body weight daily) considerably reduces the catecholamine content in the myocardium. Small doses of thyroid (1.5 mg/100 g body weight) prevent the decrease in the noradrenalin content in the myocardium during hyperfunction for 4-45 days.

Investigations have shown that the adrenalin and noradrenalin content in heart muscle is reduced in hyperthyroidism [2, 3] and in compensatory hyperfunction of the heart in rabbits [9, 11].

In the investigation described below the effect of various doses of thyroid on the adrenalin and noradrenalin content in the myocardium was studied in hyperfunction of the heart.

The character of the effect of thyroid hormones is largely determined by the dose given [1, 9, 10]. Since the cardiogenic effect of large (toxic) doses of thyroid has been adequately studied, the effect of small (1.5 and 5 mg/100 g) and average (20 mg/100 g) doses of thyroid was investigated.

EXPERIMENTAL METHOD

In experiments on albino rats hyperfunction of the heart was induced by fitting a metal spring to the subdiaphragmatic part of the abdominal aorta (coarctation of the aorta) [4]. Hyperthyroidism was induced by daily administration of thyroid in 1% starch mucilage by gastric tube for 2 weeks. The aorta was then constricted to a third or quarter of its original lumen. After the formation of coarctation of the aorta, administration of thyroid continued, and the myocardium was investigated on the 4th, 14th, or 45th day after the operation. Control animals received 1% starch mucilage. The animals were killed by decapitation. Adrenalin and noradrenalin in the heart muscle were determined by a fluorometric method [5].

EXPERIMENTAL RESULTS

Thyroid, in a dose of 20 mg/100 g body weight reduced the concentrations of adrenalin (from 0.134 ± 0.009 to 0.092 ± 0.11 $\mu\text{g/g}$) and noradrenalin (from 1.068 ± 0.09 to 0.747 ± 0.12 $\mu\text{g/g}$; $P < 0.05$) in the heart muscle, in agreement with results obtained by other investigators [2, 3].

In animals receiving thyroid in a dose of 5 mg/100 g the adrenalin concentration in the myocardium was increased (to 0.191 ± 0.028 $\mu\text{g/g}$), while the noradrenalin concentration was essentially unchanged. Administration of thyroid in doses of 1.5 mg/100 g body weight daily had no effect on the concentration of adrenalin and noradrenalin in the heart muscle. A decrease in the concentration of noradrenalin and adrenalin in the myocardium was observed 14 and 45 days after coarctation of the aorta in the control rats (Table 1).

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TABLE 1. Concentrations of Catecholamines in the Myocardium during Hyperfunction of the Heart with Disturbance of the Thyroid Hormone Balance (in $\mu\text{g/g}$ tissue)

Group of animals	Without stenosis		Duration of stenosis of aorta					
	NA	A	4 days		14 days		45 days	
			NA	A	NA	A	NA	A
1. Intact rats	1,068±0,09	0,134±0,009	0,764±0,122 $P_{2-1}>0,1$	0,184±0,029 $P_{2-1}>0,4$	0,520±0,185 $P_{2-1}<0,025$	0,070±0,019 $P_{2-1}<0,005$	0,404±0,054 $P_{2-1}<0,001$	0,072±0,014 $P_{2-1}<0,005$
2. Stenosis of the aorta without thyroïd								
3. Stenosis + thyroïd (20 mg/100 g)								
4. Stenosis + thyroïd (5 mg/100 g)								
5. Stenosis + thyroïd (1.5 mg/100 g)								
			0,609±0,110 $P_{3-2}>0,2$ $P_{3-1}<0,05$	0,186±0,02 $P_{3-2}>0,5$ $P_{3-1}>0,1$	0,368±0,067 $P_{3-2}>0,1$ $P_{3-1}<0,001$	0,094±0,006 $P_{3-2}>0,1$ $P_{3-1}<0,05$	0,263±0,009 $P_{3-2}<0,05$ $P_{3-1}<0,001$	0,071±0,011 $P_{3-2}>0,4$ $P_{3-1}<0,005$
			0,678±0,085 $P_{4-2}>0,4$ $P_{4-1}<0,01$	0,183±0,015 $P_{4-2}>0,1$ $P_{4-1}>0,1$	0,459±0,09 $P_{4-2}>0,2$ $P_{4-1}<0,001$	0,060±0,001 $P_{4-2}>0,2$ $P_{4-1}<0,001$	0,557±0,166 $P_{4-2}>0,1$ $P_{4-1}<0,005$	0,084±0,018 $P_{4-2}>0,2$ $P_{4-1}<0,05$
			0,791±0,081 $P_{5-2}>0,2$ $P_{5-1}>0,1$	0,072±0,007 $P_{5-2}<0,001$ $P_{5-1}<0,001$	0,659±0,010 $P_{6-2}>0,4$ $P_{6-1}<0,05$	0,099±0,009 $P_{6-2}>0,2$ $P_{6-1}<0,01$	0,724±0,065 $P_{6-2}<0,005$ $P_{6-1}>0,4$	0,083±0,014 $P_{6-2}>0,2$ $P_{6-1}<0,01$

Hyperfunction of the heart, against the background of thyroid administration (20 and 5 mg/100 g) led to an equal, or even more marked, decrease in the noradrenalin and adrenalin concentrations in the myocardium.

Conversely, small doses of thyroid (1.5 mg/100 g body weight) prevented the marked decrease in the noradrenalin concentration in the myocardium observed during hyperfunction of the heart and maintained an adequately high level of this catecholamine (Table 1).

The results thus confirm earlier observations [3, 6-8] showing that the concentrations of catecholamines in the heart muscle are lowered in hyperfunction of the myocardium.

According to Parin and Meerson [6, 7], the decrease in concentration of the catecholamines in the heart muscle during prolonged, exhausting work is one of the causes of the developing heart failure.

It is possible that small doses of thyroid, by stimulating the synthesis of myocardial nucleic acids and proteins, provide an adequate supply of structural materials for the developing hyperfunction and thereby prevent the decrease in concentration of catecholamines in the myocardium.

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