

EFFECT OF PRELIMINARY CASTRATION ON SYNTHESIS OF PROTEINS, RNA, AND PHOSPHOLIPIDS IN THE MYOCARDIUM DURING COMPENSATORY CARDIAC HYPERFUNCTION

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In castrated animals in the emergency stage of compensatory cardiac hyperfunction, the increase in the rate of synthesis of proteins, RNA, and phospholipids is less, while the RNA and phospholipid content in the myocardium is lower than in animals in which coarctation of the aorta was not preceded by castration. Protein synthesis in the myocardium in castrated animals 180 days after coarctation of the aorta is inhibited to a lesser degree than in noncastrated animals.

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The androgens are anabolic steroids and participate in the regulation of synthesis of nucleic acids and proteins in various organs [9, 10-12].

In this investigation the effect of exclusion of gonad function by castration on structural metabolism in the myocardium was studied in the presence of compensatory cardiac hyperfunction produced by coarctation of the aorta.

EXPERIMENTAL METHOD

Coarctation of the aorta by Beznak's method as modified by Kogan [4] was carried out 2 weeks after castration of male rats weighing 150 g. By this time the characteristic changes following castration had become stabilized [2]. Protein synthesis was studied with the aid of methionine- S^{35} , and RNA and phospholipid synthesis by means of radioactive phosphorus P^{32} . Proteins were isolated from the muscle of the left ventricle by the usual method [5, 6, 8]. RNA and phospholipids were isolated by the method of Vladimirov and co-workers [3]. Phosphorus of RNA and phospholipids was determined quantitatively by the Fiske-Subbarow method as modified by Braunshtein [1]. Synthesis of RNA and phospholipids was assessed on the basis of relative specific activity [7]. Protein synthesis was studied 4, 30, and 180 days after coarctation of the aorta, and synthesis of RNA and phospholipids 4 days after coarctation.

TABLE 1. Relative Weight of Left Ventricle and Intensity of Protein Synthesis in Myocardium during Compensatory Cardiac Hyperfunction

Group of animals	Relative weight of left ventricle $\cdot 10\ 000$			Activity per 20 mg dry protein (pulses/100 sec)		
	Day after coarctation of aorta					
	4-th	30-th	180-th	4-th	30-th	180 -th
1. Control	21 \pm 0,4	22 \pm 0,5	21 \pm 0,5	364 \pm 9	455 \pm 18	483 \pm 9
2. Coarctation	31 \pm 0,9	34 \pm 2,0	33 \pm 1,5	514 \pm 23	480 \pm 21	423 \pm 13
3. Castration	21 \pm 0,4	22 \pm 0,9	22 \pm 0,8	342 \pm 7	442 \pm 11	482 \pm 18
4. Castration + coarctation	27 \pm 0,8	33 \pm 1,8	32 \pm 1,5	419 \pm 11	496 \pm 13	464 \pm 11
P_{1-2}	<0,001	<0,001	<0,001	<0,001	>0,5	<0,01
P_{3-4}	<0,001	<0,01	<0,001	<0,001	<0,05	>0,5
P_{2-4}	<0,05	>0,05	>0,1	<0,001	>0,1	>0,05
P_{1-4}	<0,001	<0,01	<0,01	<0,001	<0,02	>0,05

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EXPERIMENTAL RESULTS AND DISCUSSION

The role of male sex hormones in structural metabolism of the myocardium during normal physiological loading of the heart is unimportant: castration has no significant effect on the rate of synthesis or the content of RNA, phospholipids, and proteins in the myocardium. The relative weight of the left ventricle in castrated animals was indistinguishable from the control at all periods of observation (Table 1). However, in animals with compensatory cardiac hyperfunction 4 days after coarctation of the aorta, a statistically significant decrease in the rate of protein synthesis by 29%, RNA synthesis by 19%, and phospholipid synthesis by 16% was observed in the castrated animals compared with those in which coarctation of the aorta was not preceded by castration. The content of RNA and phospholipids in the castrated animals with coarctation of the aorta at this period was lower than in noncastrated animals with coarctation by 22 and 16%, respectively. The degree of hypertrophy of the myocardium, determined from the relative weight of the left ventricle, was 18% lower in the castrated rats 4 days after coarctation of the aorta than in noncastrated rats ($P < 0.05$). By 30 days after coarctation of the aorta, protein synthesis in the myocardium of the noncastrated animals had returned to normal. In the castrated rats at this time protein synthesis was still 11% above the control level. This resulted in further development of the hypertrophy, and the relative weight of the left ventricle in the castrated rats with coarctation of the aorta on the 30th day was now equal to the relative weight of the left ventricle in noncastrated animals.

In the castrated animals 180 days after coarctation of the aorta a less marked decrease in the rate of protein synthesis was found than in the noncastrated animals. A possible explanation of this is that exclusion of gonad function reduces the level of activity of the organism as a whole, with a consequent lowering of the intensity of work of the heart. Wear and tear of the myocardium in the course of compensatory cardiac hyperfunction may thus develop more slowly in castrated than in noncastrated rats.

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