CHANGES IN THE MYOGLOBIN CONTENT IN THE HEART IN EXPERIMENTAL MYOCARDIAL INFARCTION AND ATHEROSCLEROSIS

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Myoglobin is one of the principal factors in the complex system supplying the heart with oxygen. This is because it possesses a number of important properties: it has an extremely high affinity for oxygen, it can combine with oxygen and liberate it quickly at a comparatively low partial pressure, and it is present in heart muscle in a high concentration. The distinctive properties of myoglobin determine its role in the heart muscle as an oxygen reserve, maintaining an adequate rate and intensity of oxidative processes in the period of contraction of the heart [2, 8]. The role of myoglobin in the heart muscle is thus extremely important, and this accounts for the interest shown in its study.

In the present investigation the myoglobin content in the heart of normal animals was studied. Its content was determined in animals of different ages, and in different subdivisions and areas of the heart muscle. The effect of experimental atherosclerosis and of an acute disturbance of the coronary blood flow on the content of this chromoprotein was also investigated.

EXPERIMENTAL METHOD

Experiments were carried out on albino rats of three age groups (under 1 month, 8-12 months, and 24-36 months) and on mongrel dogs weighing 12-16 kg. Experimental myocardial infarction was produced in the dogs by ligation of the descending branch of the left coronary artery (86 animals). At different times after the operation (2-5 and 10-12 h, 1, 3, 5, 10, and 30 days) the region of the infarct and a remote area, designated the intact zone of the heart (usually an area in the posterior wall of the left ventricle), were investigated. Experimental atherosclerosis was produced in seven dogs by the Steiner–Kendall principle, by the combined administration of cholesterol and methylthiouracil for 9-15 months [4]. Myoglobin was determined spectrophotometrically by Björck's method [5].

EXPERIMENTAL RESULTS

The myoglobin content in the heart muscle of the rats in the first month of life was 0.46±0.002 g %. The highest content was found in the heart of middle-aged rats (1.26±0.002 g %). A significant fall in the myoglobin content was observed in the myocardium of the old animals (0.96±0.006 g%), evidently one of the causes of the decrease in intensity of tissue respiration observed in old age.

Investigation of the topography of the metabolic processes in the heart is of great importance to our understanding of the link between the functions of the various parts of the heart and its metabolism. The highest myoglobin content was discovered in the left ventricle (1.42±0.09 g%), which performs the most work. It is characteristic that the highest intensity of oxidative metabolism is found in the left ventricle [3]. The myoglobin content in the right ventricle (1.03±0.08 g%) was lower than in the left, but higher than in the atria (0.46±0.1 g% in the left and 0.81±0.09 g%). Moreover, in the same subdivision of the heart (the left ventricle), differences in the myoglobin content were found in different areas and levels.

There is wide discussion at the present time of the problem of the blood supply to the various divisions of the heart at the moment of systole and diastole. Many authors state that the inner layers of the myocardium are less well supplied with blood at the moment of systole than the more superficial layers [6, 7]. The results of the experiments now described show that the myoglobin content is in fact higher (1.66±0.11 g%) in the inner layer (next to the endocardium). The myoglobin content in the outer layer is

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TABLE 1. Myoglobin Content in the Heart (M±m) at Various Times during Development of Experimental Myocardial Infarction in Dogs (in g/100 g Dry Tissue)

Duration of infarct	Zone of infarct	Intact zone
From 2-5 h	0,81±0,12	1,12±0,23
10—12 h	0,71±0,13	1,20±0,17
1 day	0,57±0,05	1,18±0,11
2 days	0,53±0,10	1,23±0,13
3 »	0,37±0,08	0,90±0,11
5 »	0,60±0,10	1,15±0,14
10 »	0,64±0,12	1,41±0,82
30 »	0,99±0,09	1,05±1,07
Normal	1,42±0,09	

TABLE 2. Myoglobin Content in the Heart and Some Biochemical Indices of the Blood Serum in Dogs with Experimental Atherosclerosis

Experimental dogs	Myoglobin in the heart (in g/100 g dry tissue)	Serum			
		cholesterol (in mg %)	lecithin (in mg %)	liproproteins (in % of total content)	
				α	β
V'yunok	0,77	1250	502	4,1	95,9
Pirat	0,56	1376	620	9,9	90,1
Ral'f	0,84	520	452	12,8	87,2
Seryi	0,62	1000	1350	2,5	97,5
Pluton	0,88	2296	515	0	100
Tsygan	1,10	576	625	16,6	83,4
Pegas	0,60	1904	707	18,0	82,0
Normal	1,42	155	187	76	24

1.2±0.13 g%. Evidently, the higher myoglobin level in the inner layers of the left ventricle is a special mechanism of adaptation to the conditions of blood supply determined by the cardiac activity.

The important role of myoglobin is also seen in chronic anoxia, for example when the partial pressure of oxygen is lowered, and this is accompanied by a marked increase in the myoglobin concentration in the heart [1]. However, no figures are available for the quantitative changes in the myoglobin concentration in acute anoxia.

Experiments were carried out to study the effect of ligation of the left coronary artery on the myoglobin content at various times during the development of myocardial infarction. As the results given in Table 1 show, 2-5 h after the development of acute ischemia of the myocardium the myoglobin content began to fall in the zone of the infarct. The lowest myoglobin content in the myocardium was found after 3 days, when it had fallen to one-quarter of its normal value. On the 5th-10th day the myoglobin level was still definitely low. Thirty days after production of myocardial infarction the normal myoglobin concentration in the heart muscle still had not been restored, demonstrating the stability of the changes taking place. In the intact zone of the heart the myoglobin content was within the lower limit of normal almost throughout the period of observation, except on the 3rd and 30th days, when it was lowered by a statistically significant amount.

The myoglobin content also was investigated in the hearts of 7 dogs with experimental atherosclerosis at the height of development of the lesion, as reflected by the characteristic blood changes: hypercholesteremia, phospholipidemia, an increase in the β-lipoprotein fraction, etc.

It is clear from Table 2 that the degree of lowering of the myoglobin content in the individual animals varied, but not to the same extent as in myocardial infarction.

The variation in the character of the changes in the myoglobin content in the heart—from a considerable increase in chronic anoxia to a sharp decrease in myocardial infarction and an appreciable decrease in experimental atherosclerosis—is evidently determined by the degree and the rate of development of the tissue anoxia and the structural changes taking place in the tissues. In turn, the changes in the myoglobin content may exert an effect on the course of oxidative processes in the myocardium in these conditions.

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