THE OXYGEN SUPPLY OF THE MYOCARDIUM IN ACUTE ANEMIA CAUSED BY PARTIAL REPLACEMENT OF THE BLOOD BY POLYGLUCIN

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The threat of acute anemic hypoxia arises after therapeutic transfusions of blood substitutes, when the oxygen capacity of the circulating blood is sharply reduced although its normal volume is restored.

It has been shown that in acute normovolemic anemia the minute volume is considerably increased [1,11,14, 15], the coronary blood flow is increased [4,10], and the metabolism of the myocardium is modified [15].

The question naturally arises how the heart muscle receives its oxygen supply in these conditions and whether any threat of anoxia of the myocardium develops.

To investigate this problem, the oxygen supply of the myocardium was studied in acute anemia caused by replacement of part of the blood by polyglucin.

EXPERIMENTAL METHOD

Experiments were carried out on 10 dogs. The animals were anesthetized with Nembutal (40 mg/kg) and transferred to artificial respiration using a mixture containing 45% oxygen, after which the thorax was opened by sawing through the sternum. Clotting of the blood was prevented by heparin. The coronary blood flow was determined by the method of Morawitz and Zahn as modified by N. V. Kaverina. The arterial pressure and the coronary blood flow were recorded on the paper of a kymograph. The oxygen tension (pO_2) was determined continuously in relative units (as a percentage of the initial level) using a polarographic method with a bare platinum electrode implanted into the anterior wall of the left ventricle [3]. The oxygen concentration in the arterial and venous blood (from the coronary sinus) was determined by Van Slyke's method. The values of pO_2 in the arterial and venous blood were measured by a polarographic method using Clark's electrode [7]. The carbon dioxide tension (pCO_2) was determined by the principle of Show and Randall [18]. The degree of dilution of the blood with the blood substitute was verified from the hemoglobin concentration and the hematocrit index. The blood flow and oxygen consumtpion were estimated per 100 g left ventricle, and the weight of the ventricle was determined from data obtained by Hermann [12]. The mean capillary pO_2 in the myocardium, the resistance to the coronary blood flow, and the percentage utilization of oxygen by the myocardium were calculated. Besides the determination of pO_2 by the polarographic method, this index was also calculated by the method of Thews [19] in the venous portion of a tissue cylinder bordering on the capillaries.

Blood samples were taken from the artery and coronary sinus in the initial state, at the height of the blood loss of 30 ml/kg (in the conditions of the open-thorax experiment this blood loss was serious) and 10-15 min and 1 h after replacement of the lost blood by an equal volume of polyglucin. The numerical results were analyzed by statistical methods.

EXPERIMENTAL RESULTS AND DISCUSSION

Data showing the changes in the hemodynamics and the overall gas exchange of the heart muscle are given in Table 1.

In the initial state, the arterial pressure was relatively low, on account of the severity of the operation, the depth of the anesthesia and, possibly, the action of an increased oxygen concentration in the inspired air [8]. This last factor also led to a slight decrease in the coronary blood flow and in the oxygen consumption, as other authors also have observed [8,17].

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TABLE 1. Hemodynamics and Oxygen Consumption of the Heart Muscle

	Initial data	After blood loss	After replacement by an equal volume of polyglucin	1 h after replacement of lost blood
Arterial pressure (in mm)	82	7 ± 3.1 P < 0.001	88 ± 3.6 P > 0.25	90 ± 5 P > 0.05
Coronary blood flow (in ml/100 g weight of left ventricle)	45	15.2 ± 6.4 P < 0.02	80 ± 5.4 P < 0.001	91 ± 5 P < 0.001
Oxygen consumption of myocardium (in ml/100 g weight of left ventricle)	6.8	2.5 ± 0.73 P < 0.001	7.1 ± 0.55 P > 0.25	—
Resistance of coronary blood flow	2.0	0.5	1.2 ± 0.1 P < 0.001	1 ± 0.2 P < 0.001
Heart rate	153	85 ± 44	121 ± 21	145 ± 21
Oxygen tension in heart muscle (in %)	100	54	93 ± 4.8 P > 0.1	93 ± 5.7 P > 0.5
pO_2 in venous end of tissue cylinder (in mm Hg)	16	7 ± 1.6 P < 0.002	20 ± 1.8 P > 0.05	16

As a result of the blood loss, the coronary blood flow was reduced to one-third, the oxygen consumption of the heart also was reduced almost to one-third, and the value of pO_2 in the myocardium fell only to one-half its initial value.

Transfusion of polyglucin against this background led to a gradual increase in the value of pO_2 in the heart muscle, reaching the initial level only a few minutes after the end of the transfusion, in contrast to the value of pO_2 in the brain tissues, which was restored much faster [5]. The arterial pressure returned to its initial level. The oxygen consumption was restored, as a result of a corresponding increase in the coronary blood flow. The resistance of the coronary blood vessels was reduced.

The changes in the gaseous composition of the blood, explaining the pattern of the oxygen supply of the heart after blood loss and replacement, are shown in Table 2.

In the initial state, the results showing the gaseous composition of the arterial blood corresponded to the normal physiological values, with the exception of the increased pO_2, which was due to inhalation of air containing 45% oxygen.

At the height of the blood loss, very considerable changes were observed in the venous blood. The oxygen concentration in volumes % fell to an extremely low value and pO_2 fell by approximately the same amount as in the heart muscle, i.e., by 50%. The percentage utilization of oxygen was 91 compared with 74 in the initial state. The concentration of CO_2 and the value of pCO_2 in the venous blood were practically unchanged, but the veno-arterial difference of CO_2 was increased, a sign of respiratory and, possibly, of tissue acidosis. The presence of severe hypoxia of the myocardium was confirmed by the calculated values of pO_2 at the venous end of the tissue cylinder, which fell from 16 to 7 mm. According to data in the literature, this value is close to the limit beyond which the activity of the oxidative enzyme systems is disturbed [6].

Immediately after replacement of the lost blood, the restoration of the normal oxygen consumption of the myocardium and of the CO_2 level in the heart muscle was accompanied by changes in the gaseous composition of the blood. The hemoglobin concentration fell to 6.6 g % (initially 13.3 g %), and the hematocrit index fell by approximately 50%. The oxygen concentration in the arterial blood fell, while the other indices of the gaseous composition of the blood returned to their initial values. In the venous blood, the oxygen concentration fell in proportion to the dilution of the blood. The arterio-venous difference was reduced. The value of pO_2 rose slightly, possibly as a result of the increased blood flow and of a shift to the right of the hemoglobin dissociation curve.

This last mentioned phenomenon has also been described in similar circumstances for mixed venous blood [13, 16]. The value of pO_2 in the venous end of the tissue cylinder was slightly higher than initially.

TABLE 2. Gaseous Composition of Arterial Blood and of Blood Flowing from Sinus Venosus

Index	Initial data	After blood loss	After replacement of lost blood by an equal volume of polyglucin	1 h after replacement of lost blood
Concentration of O_2 (in vols. %)*	20,3 5,1	19,1±1,8 1,7±0,55 $P<0,01$	12,4±1,2 3,3±0,57 $P<0,05$	
Arterio-venous difference (in vols. %)	15,2	17,4	9,1	
Concentration of CO_2 (in vols. %)	19,3	14,5±1 $P<0,01$	18,5±0,9 $P>0,2$	
	32,9	30±0,7 $P<0,02$	25,2±1,2 $P<0,02$	
pO_2 (in mm)*	126	103±5,2 $P<0,01$	127±7,9 $P>0,5$	114±6,9 $P>0,1$
	21	12±1,3 $P<0,001$	25±2,0 $P<0,05$	21±2,7 $P>0,2$
pCO_2 (in mm)*	27	21±1,7 $P<0,05$	24±2,1 $P>0,2$	26±3,6 $P>0,5$
	42	45±2,4 $P>0,2$	43±2,3 $P>0,5$	38±2,9 $P>0,5$
Mean capillary pO_2 (in mm)	57	44±1,0 $P<0,001$	55±1,6 $P>0,5$	52±2,5 $P>0,2$
Percentage utilization of O_2	74	91±3,6 $P<0,01$	73±4,7 $P>0,2$	—
Hemoglobin (in g %)	13,3	—	6,6	7,9
Hematocrit index	43	—	22	26

*Above — index of arterial blood; below — of blood flowing from sinus venosus.

One hour after replacement of the lost blood, the value of pO_2 in the venous blood was the same as initially. The polarographic findings showed that pO_2 in the heart muscle was maintained at the level it had reached and calculations showed that it returned to its initial value.

The results of these experiments thus show that, despite the development of acute normovolemic anemia, judging by the oxygen consumption and the pO_2 levels in the heart muscle and in the venous blood, the oxygen supply of the myocardium was undisturbed.

At the same time, it was found that inhalation of a mixture with an increased oxygen concentration does not portect the myocardium against severe hypoxic disorders in the presence of marked disturbances of the circulation and a fall in the volume of the coronary blood flow.

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