CONTRACTILE FUNCTION OF THE MYOCARDIUM AFTER BLOOD LOSS AND REPLACEMENT

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The contractile function of the myocardium was studied during replacement of lost blood by autologous blood and blood substitutes in experiments on rabbits using the method of repeated loading by compression of the aorta. The force of contraction in all experiments was maintained for a long time and the difference from the control was small. The rate of contraction was reduced even after transfusion of autologous blood. Transfusion of blood substitutes caused an even greater decrease in the contractility index, less so with the use of dextran than of gelatinol. The decrease in contractility of the myocardium is connected with the hypoxia caused by the blood loss.

The marked increase in minute volume with a decrease in the oxygen capacity of the blood, including that arising by dilution of the blood with substitutes, has been known for a long time as the result of much research. The oxygen consumption of the heart and the total quantity of work performed by it show little change under these circumstances on account of a decrease in the general peripheral resistance [2].

In this investigation an attempt was made to determine whether partial replacement of the blood by a foreign medium leads to a decrease in the contractile power of the heart and whether the blood loss itself has a harmful action on the myocardium, leading subsequently (if the hypotension is prolonged) to the formation of the myocardium-injuring factor described earlier [5].

EXPERIMENTAL METHOD

Rabbits weighing 2-2.5 kg were anesthetized with chloralose and urethane, thoracotomy was performed and controlled respiration applied. The pressure in the left ventricle was measured with an electromanometer through a thick needle introduced into the chamber of the ventricle. The arterial pressure was measured in the femoral artery. Recordings were made on the Mingograph-81 instrument. A load was applied to the heart by repeatedly compressing the aorta [3] for 30 sec at intervals of 5 min. The aorta was compressed 14 times in each animal. The heart rate and the maximal increase in systolic and end-diastolic pressure were determined after compression for 25 sec and the contractility index (the ratio between the maximal rate of rise of pressure in the ventricle in the isometric phase of contraction and the integrative tension in the muscle in that phase) was calculated [8, 10]. The blood loss amounted to 20-25 ml/kg and it caused the arterial pressure to fall to 20 ± 5 mm Hg. The lost blood was replaced by the same volume of the rabbit's own blood (10 experiments), of dextran (10 experiments), and of gelatinol (8 experiments). The tests with compression of the aorta began 2 h after the end of blood replacement, when the animal was adapted to the new conditions. Ten control experiments (without blood loss and transfusion) also were carried out.

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TABLE 1. Changes in the Maximal Systolic Pressure (in mm Hg) during Repeated Compression of the Aorta (M ± m)

Number of consecutive compressions	Initial background	Period of clinical death: recovery period	dextran	gelatinol
1	177±7	172±6	154±7*	166±5
3	177±8	174±6	155±7	159±5
5	175±8	173±6	154±7	154±6
7	173±5	173±5	154±7	155±6
10	174±2	163±5	151±7	151±4
14	161±12	156±6	149±7	124±8*

Note: Here and in Table 2 values differing significantly from the control for the same compressions and values differing significantly from values at the first compression are marked with an asterisk.

TABLE 2. Changes in Contractility Index (in sec^{-2}) during Repeated Compression of the Aorta (M ± m)

Number of consecutive compressions	Control	After transfusion of: blood	dextran	gelatinol
1	864±95	828±43	574±102*	521±29*
3	819±37	702±43	513±93*	420±26*
5	747±39	607±41*	424±72*	328±28*
7	662±46	494±45*	394±75*	295±29*
10	552±59	381±23*	295±50*	227±22*
14	389±77	275±25	240±44	144±30*

EXPERIMENTAL RESULTS

The principal results are shown in Tables 1 and 2. Changes in the parameters studied were calculated relative to the control and also to the original data (first compression); in the latter case the statistical analysis was carried out by the difference method.

The exceptional stability of the maximal force of contractions during repeated compressions will be noted. Compared with the control there was a small decrease in the strength of contraction after transfusion of the blood substitutes, but this was statistically significant only in the experiments in which gelatinol was injected and at the 14th compression. Compared with the initial data the decrease in the strength of contraction occurred in all experiments after the 10th compression. A more complete idea of fatigue of the myocardium was given by the contractility index (Table 2). The results illustrated in Fig. 1 are expressed as percentages relative to the first compression in the control experiment, taken as 100%. In the control experiments a statistically significant decrease in the contractility index occurred after the 5th compression, but in the experiments with replacement of the lost blood by blood after the 3rd compression, the decrease in this case was more marked than in the control experiments. An even greater decrease in the contractility index was observed in animals in which the lost blood was replaced by blood substitutes.

The blood loss to which the animals were subjected thus clearly altered the functional state of the heart muscle, as was shown distinctly by the experiments in which the animals were transfused with their own blood. The cause of the phenomena observed could be disturbances of carbohydrate-phosphorus, lipid, and protein metabolism arising in the myocardium as a result of the blood loss [7, 9, 11]. The anemic hypoxia caused by transfusion of the blood substitutes reduced the reserve forces of the heart still further, but to a lesser degree after the transfusion of dextran than of gelatinol. The possibility of a harmful action of dextran on the myocardium seems unlikely. Large doses of dextran with different molecular weights have been injected repeatedly and for long periods into rabbits with the object of inducing injury to the myocardium but no such injury could be found either histologically or histochemically [6]. In other experiments in dogs changes were found in the mitochondria of the myocardium after replacement of lost blood by the substance rheopolyglucin, but the workers concerned state that the results of experiments using high dilutions of blood can be regarded as a compensatory reaction to changes taking place in oxidative metabolism rather than as a harmful action of the dextran itself on the mitochondria [4]. It is interesting to note that transfusion of rabbits with dextran leads to an increase in the noradrenalin concentration in their heart [1] which helps to maintain the force and rate of its contractions.

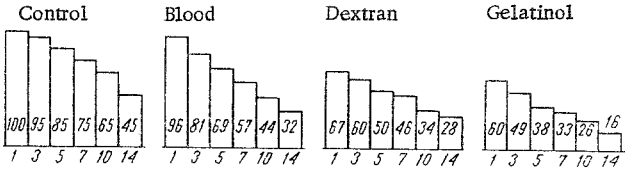

Fig. 1. Changes in contractility index (in percentage of first compression in the control). Numbers inside columns are percentages; numbers below columns represent serial no. of compression.

The cause of the more marked inhibition of the contractile function of the myocardium after transfusion of gelatinol is uncertain. It is also not clear whether gelatinol itself exerts a negative inotropic action or whether it inhibits the manifestation of positive inotropic effects.

It cannot be concluded from the small decrease in contractile power of the myocardium observed in these experiments after replacement of the lost blood by dextran that it has a harmful action on the myocardium and weakens cardiac function appreciably. This action was observed only in special experiments, in which maximal loads were applied many times in succession, under conditions of isometric contraction disadvantageous for the heart.

Under ordinary conditions after replacement of the lost blood by dextran the heart could cope fully with the load and respond with an increased minute volume. Under these conditions the work of the heart was facilitated by a considerable decrease in the resistance of the peripheral vessels.

At the same time it must be remembered that heart failure after transfusion of large doses of blood substitutes can arise if additional increased demands are presented to the heart muscle or if it is damaged by pathological processes.

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