The Effects of Assisted Circulation by Intraaortic Balloon Pumping and Shunting of the Left Ventricle on Myocardial Damage after Coronary Artery Ligation in Dogs

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SUMMARY

The purpose of this study is to determine the effects of different kinds of assisted circulation (AC) on the intensity of ischemic and necrotic processes in experimental myocardial infarction in dogs. Three types of experiments were carried out. In the first group, the infarction was modelled by surgical ligation of a branch of the descending artery. In the second group, AC by intraaortic balloon counterpulsation was started 15 min after coronary occlusion and lasted for 2 hours. In the third group, AC was carried out by means of left ventricular drainage with transaortic insertion of a catheter, and a subsequent forcing of the blood into the femoral artery. During the experiment, 10 epicardial leads were recorded and the number of points registering a considerable displacement of segment ST (nST) as well as the total displacement of segment ST (Σ ST) were analysed. The size of the necrotic zone in the myocardial wall was identified macroscopically.

The results testify to the favourable effects of both methods of AC on the intensity of destructive myocardial processes. The effect on ST was recorded soon after the beginning of AC. The effect on Σ ST was less pronounced and was observed at a later stage (after 30–45 min). In the histochemical investigation of the necrotic zone of the myocardial wall, the positive effects were more marked in the extracorporeal bypass of the left ventricle, which showed the size of myocardial infarction to be much smaller ($p < 0.05$) than in the group with intraaortic balloon pumping. These experimental data are indicative of a significant positive effects of AC. In particular, shunting of the left ventricle caused a marked decrease in myocardial necrosis.

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ASSISTED circulation (AC) is an alternative method of treating acute cardiac insufficiency.¹⁾ The present means of assisting the weakened myocardium can be grouped according to two principles: 1) diminished after-load (decreased aorta impedance) and 2) decreased preload.^{2),3)} Intraaortic balloon counterpulsation is the best example of the first type of AC method. Its simplicity, safety, and high effectiveness have made it a standard for the usefulness of other assisted circulation methods. The second type of AC method consists of shunting part of the blood from the left atrium or left ventricle to the aorta or a peripheral artery, thus diminishing the work of the left ventricle. Research in this field was initiated by Dennis et al,⁴⁾ who published a method for unloading the left ventricle via a rigid cannula, passed through the septum into the left atrium. A number of reports have been published on the use of blood drainage from the left atrium⁵⁾ or left ventricle,^{6),7)} and transarterial unloading of the left ventricle has been used clinically with considerable success.⁸⁾

The aim of our experiments was to compare the efficiency of two methods of AC: intraaortic balloon counterpulsation and transarterial blood drainage from the left ventricle. The efficiency of these methods was compared by electrocardiographic recordings of the displacement of segment ST from the epicardial leads, as well as by macroscopic identification of the necrotic lesions of the myocardium.

MATERIALS AND METHODS

Twenty-six mongrel dogs (mainly male) weighing from 24 to 35 Kg (average 27.8 Kg) were used in our experiments. They were divided into 3 groups: a control group and 2 groups for investigation of 1) the influence of AC by intraaortic counterpulsation and 2) blood drainage from the left ventricle. The control group consisted of 10 dogs. After premedication with neuroleptanalgesic drugs (fentanyl 0.01 mg/Kg, droperidol 0.25 mg/Kg) and atropine (6–8 mg), they were anaesthetized by repeated fractional administration of hexenal. Respiration was maintained by endotracheal intubation with an air-oxygen mixture. A left thoracotomy was performed in the fourth or fifth intercostal space and the pericardium was opened. The apical branch leading from the descending coronary artery was ligated. The following parameters were recorded simultaneously: electrocardiogram, left ventricular pressure (via a catheter passed through the left auricle), arterial pressure in the aortic arch (via a catheter passed through the brachial artery), and cardiac output with an electromagnetic flowmeter. The heparinization of dogs was performed at a rate of 2 mg/Kg of body weight. Blood samples were

drawn periodically to investigate a number of biochemical indices.

Epicardial ECG leads from the frontal surface of the left ventricle were recorded by the methods of Maroko et al⁹⁾ with special electrodes located at 10 sites. Six recording sites were in the immediate proximity of the ischemic area. Epicardial leads were registered immediately after the ligation of a branch of the descending coronary artery, and additional records were taken every 15 min for 2 hours and 15 min. After completion of recording from the epicardial leads, the chest was sutured layer by layer leaving a drainage in the pleural cavity. A pathomorphological investigation of the heart was carried out after the dog's death, but not later than 24 hours after the ligation of the coronary artery.

The size of necrosis arising after the ligation of the coronary artery was determined by the procedure of Nachlas and Schnitka.¹⁰⁾ The myocardial sections (1 cm thick) were stained, the necrotic area was excised, weighed and its volume was determined by means of removing liquid from a measuring cavity. Similar studies were performed on the whole left ventricle. The projection area of the zone of myocardial infarction upon the frontal surface of the left ventricle was measured in accordance with the spread of the necrosis in the myocardial wall.

The preparatory stages in the second group of experiments (8 dogs) was analogous to those for the control group. In addition, one of the femoral arteries or the common iliac artery of the animal was exposed for inserting the intraaortic balloon. The end of the balloon was situated just below the bifurcation of the left subclavian artery. The balloon-pump was synchronized with the "R" wave of ECG. Intraaortic balloon counterpulsation started 15 min after the ligation of a branch the descending coronary artery and lasted for 2 hours. Intraaortic balloon pumping was realised by a dual-chambered balloon with 23 cm³ volume. The systolic pressure of balloon inflation was about 150 mmHg, the diastolic balloon pressure was created by 40 mmHg of vacuum.

The third group also consisted of 8 dogs on whom a bypass shunting of the left ventricle was performed. Blood was drawn through a catheter with a 4 mm inner diameter, inserted through the left subclavian artery into the left cardiac ventricle. The blood from the left ventricle was collected into a cylinder and was forced into the femoral artery by means of a roller pump. The mean output of the pump was 1,100 ml/min (from 31 to 45 ml/min/Kg), equivalent to about 44% of cardiac output. A reservoir included in the circuit of the outer shunt served to control the volume of the extracorporeal shunt and as an air trap.

The following indices have been analysed in the present study: total rise

of segment ST in epicardial leads (Σ ST), number of recording points of epicardial leads where the displacement of segment ST was equal or exceeded 2 mm (nST), and the size of myocardial necrosis in the left ventricular wall.

RESULTS

The following data were obtained from the first group. Immediately after ligation of the coronary artery, the total displacement of segment ST was equal to 4.4 ± 0.5 cm (Table I). The maximum value of this index was recorded 75 and 90 min after coronary occlusion. Subsequently, ST decreased and was equal to its initial value (3.8 ± 0.4 cm) in 2 hours and 15 min. The number of points with considerable segment ST displacement behaved similarly. The maximum nST rise was recorded between 1 hour and 1 hour 45 min after ligation and fluctuated around 9.0, and decreased towards the end of the observation period (Table II).

The following data were obtained from the second group with intra-aortic counterpulsation. The maximum Σ ST rise was recorded 15 min after the ligation of the coronary artery (6.7 ± 0.6 cm). At a later stage, when intraaortic counterpulsation was started, a progressive decrease of this index was noted. Starting with the 45th min after the ligation of the coronary artery or at the 30th min of AC, Σ ST fluctuated between 3.6 ± 0.3 cm and 4.1 ± 0.9 cm. In this group, nST increased sharply within 15 min after the ligation of the coronary artery, and from with the 30th min and up to the end of AC, this index stabilized at an average of about 6.0.

In the group with a bypass of the left ventricle, the maximum value of Σ ST was recorded immediately after ligation (5.1 ± 0.9 cm) and prior to the onset of AC (5.8 ± 1.4 cm). While the shunt was functioning, this index decreased gradually and stabilized at about 4.0 cm, especially in the second half

Table I. Changes of Σ ST in the Control and Assisted Circulation Groups in Dogs
(Mean $\pm$ S.E.)

Time Σ ST	Ligation of coronary artery	15 min	30 min	45 min	60 min	1 h 15 min	1 h 30 min	1 h 45 min	2 h	2 h 15 min
Control (cm) M	4.4	6.0	5.6	6.0	5.9	7.4	7.5	7.0	4.9	3.8
S.E.	0.5	1.2	0.9	1.4	0.9	0.8	0.5	0.8	1.2	0.4
IABP (cm) M	4.0	6.7	6.3	4.8	4.0	3.9	3.6	4.0	4.1	3.7
S.E.	0.6	0.6	0.1	0.6	0.8	0.4	0.3	0.6	0.9	0.3
LVB (cm) M	5.1	5.8	4.7	3.7	5.0	4.3	4.0	3.9	3.7	3.7
S.E.	0.9	1.4	0.7	1.2	0.1	0.7	0.6	0.6	1.3	6.6

of the experiment. An hour after the ligation of the coronary artery Σ ST and nST increased to 5.0 ± 0.1 cm and 8.2 ± 1.4 respectively. The number of points with a considerable displacement of segment ST during AC decreased from 7.0 ± 1.6 at the beginning of the experiment to 6.2 ± 0.6 at the end. The change was not significant

The data obtained by macroscopic identification of necrotic changes in the whole ventricular wall had some interesting features (Table III). In the control group, the mass of the infarction was 14.34 ± 1.98 Gm per 100 Gm of left ventricular myocardium, and the infarction mass volume was 16.60 ± 2.02 cm³ per 100 cm³ of left ventricular volume. The projection of the necrotic zone onto the frontal surface of the left ventricle had an area of 14.4 ± 2.9 cm². The following data demonstrated the beneficial effects of intraaortic balloon pumping on myocardial necrosis: the infarction mass was 10.70 ± 1.49 Gm per 100 Gm left ventricular myocardium and the infarction mass volume was 9.32 ± 1.33 cm³ per 100 cm³ of left ventricular volume. The area

Table II. Changes of nST in the Control and Assisted Circulation Groups in Dogs

Time nST	Ligation of coronary artery	15 min	30 min	45 min	60 min	1 h 15 min	1 h 30 min	1 h 45 min	2 h	2 h 15 min
Control (cm) M		7.0	7.6	7.8	8.0	9.2	8.4	9.0	9.0	7.2
S.E.		1.4	0.5	1.3	1.5	0.8	1.5	0.5	0.5	1.1
IABP (cm) M		6.8	8.3	8.3	7.0	6.0	6.0	5.0	7.3	6.3
S.E.		0.5	0.9	0.5	0.7	0.7	0.7	0.6	0.4	0.9
LVB (cm) M		7.0	7.2	7.4	6.6	8.2	5.8	5.7	6.6	6.5
S.E.		1.6	1.0	1.1	1.5	1.4	1.1	0.9	0.7	0.6

IABP=intraaortic balloon pumping; LVB=left ventricular bypass.

Table III. Influence of Assisted Circulation on the Intensity of Necrosis in the Ventricular Wall

	Control	IABP	LVB	P ₁	P ₂	P ₃
Infarction mass per 100 Gm of myocardium (Gm)	14.34 ± 1.98	10.70 ± 1.49	8.10 ± 1.31	>0.05	<0.55	>0.05
Infarction mass volume per 100 cm ³ of myocardium (cm ³)	16.60 ± 2.02	9.32 ± 1.33	5.92 ± 1.31	<0.05	<0.05	<0.05
MI area on LV surface (cm ²)	14.4 ± 2.9	9.9 ± 2.8	7.2 ± 1.5	>0.05	<0.05	>0.05

P₁=significant difference between control and intraaortic balloon pumping values; P₂=significant difference between control and left ventricular bypass values; P₃=significant difference between IABP and LVB values. MI=myocardial infarction; LV=left ventricle; IABP=intraaortic balloon pumping; LVB=left ventricular bypass. Mean $\pm$ S.E.

of the necrotic lesion projected upon the frontal wall of the left ventricle was $9.9 \pm 2.8 \text{ cm}^2$. In the group with transaortic shunting of the left ventricle, more convincing data on the reduction of the necrotic lesion zone were obtained. The infarction mass was minimal in that group of animals: $8.10 \pm 1.31 \text{ Gm}$ per 100 Gm of left ventricular myocardium. The volume of infarction mass was also minimal ($5.92 \pm 1.31 \text{ cm}^3$ per 100 cm^3 of left ventricular volume). The projection of the necrotic zone on the surface of the left ventricle was $7.2 \pm 1.5 \text{ cm}^2$.

DISCUSSION

Although intraaortic balloon pumping has been in use as a method of assisted circulation on a large scale in cardiosurgical practice, as well as in the treatment of acute myocardial infarction and unstable angina,¹¹⁾⁻¹⁴⁾ it has certain disadvantages. Intraaortic counterpulsation assumes *a priori* cardiac work (in heart massage using a aortic pressure monitor as a source of synchronized impulses). Therefore, the use of AC methods is promising in resuscitation, because it may fully or partly take over the pumping function of the heart. The methods should be simple in construction and quick in application. The drainage of the left ventricle initiated by Dennis et al⁴⁾ meets these needs to a certain degree. However the method of inserting a rigid cannula into the left atrium by perforating interatrial septum from the right atrium causes trauma and is rather dangerous. That is why the insertion of a cannula for collecting blood from the left ventricle through the arterial system seems to be more promising. There are reports by Zwart et al⁸⁾ on the use of this method with relatively positive results.

We have chosen two methods for comparing the effectiveness of the two procedures for AC: the degree of ischemia in the area of experimental myocardial infarction was estimated from recordings from epicardial leads and macroscopic determination of the size of myocardial necrosis. The method of recording displacements of segment ST is attractive because of its simplicity, safety, and speed in the interpretation of final results. Thus it is very convenient for a comparative estimation of therapeutic measures under investigation.¹⁵⁾⁻¹⁸⁾ However, some reports state that there is no close link between the displacement of segment ST and the intensity of biochemical shifts in the myocardial tissue.¹⁹⁾ Therefore, only a direct recording of the viability of cardiac cells can serve as an absolute index of destruction of the heart tissue. Recordings of dehydrogenase activity in functionally healthy tissue and its absence in the necrotic zone gives a possibility to delineate the damaged area in the ventricular wall.^{10),20)}

Our data show that assisted circulation, both by intraaortic counterpulsation and by shunting the left ventricle, favourably affects the myocardium and lowers the displacement of segment ST in epicardial leads. An analysis of data obtained from recording epicardial leads does not reveal significant differences in groups with AC. However, the results of macroscopic investigations of necrosis in groups with AC show a considerable decrease both of the necrotic zone in the ventricular wall and of the zone of the necrosis projected on the surface of the left ventricle, especially when shunting the left ventricle.

Thus, our investigation has established that the left ventricular bypass compared to intraaortic balloon pumping has a marked effect on the intensity of experimental myocardial infarction. At the same time it has been established that the dynamic control of the displacement level of segment ST in the epicardial leads may serve as an indication of the intensity and direction of pathological processes in the myocardial wall. However, this method does not give a strict quantitative evaluation of the size of the necrotic zone. The latter can be more adequately obtained by histochemical methods.

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