Selective Embolization of the Coronary Arteries. A Hemodynamic, Metabolic and Radiologic Study.* (29582)

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The advantage of producing myocardial infarction by selective injection of emboli into the coronary circulation has been previously emphasized(1). In the present study myocardial infarction was produced by injecting relatively large (1.1 mm in diameter) steel ball bearings into the circumflex branch of the left coronary artery. The ball bearings are large enough to remain in the coronary system and to be easily seen by fluoroscopy and radiography. Coronary arteriograms were obtained before and after embolization, to determine the extent of the coronary vascular system occluded by the emboli and to attempt to demonstrate changes in the caliber of the coronary arteries.

The changes in hemodynamics and myocardial metabolism occurring as a result of the myocardial infarction produced are reported here.

Material and methods. Twenty-three experiments were carried out. Large mongrel dogs weighing 18 kg or more were used. Anesthesia was produced by Pentobarbital (25 mg/kg of body weight) given intravenously. Catheters were introduced through the left carotid artery, external jugular vein, and right brachial vein and artery. Pressures were obtained from the right and left atrium and ventricle, ascending aorta and pulmonary artery. Statham strain gauges and a Sanborn Polybeam recorder were used for registering the pressures. A dye dilution curve was recorded by injecting 5 mg of indocyanine green into the right atrium with continuous sampling from the aorta through a Gilford densitometer(2). The cardiac output was calculated by the Hamilton method(3). The coronary sinus was then intubated and simultaneous arterial and coronary venous blood was drawn for determination of coronary blood flow(4), oxygen(5), glucose(6), lactate(7), pyruvate(8), and free fatty acids (9). The circumflex branch of the left coronary artery was then catheterized with a No. 7 aortographic catheter, under fluoroscopic control. A coronary arteriogram was obtained by injecting 2 cc of sodium iothalamate (Angioconray) and utilizing the spot film device of the Picker image amplifier. Steel ball bearings (1.1 mm in diameter) were then injected into the intubated coronary artery. In 12 experiments, 2 to 3 ball bearings/kg of weight were injected, in the remaining, 4-5 ball bearings/kg of weight. Three cubic centimeters of normal saline solution was used to carry the ball bearings through the catheter. Further flushing of the catheter, when needed, was done by repeatedly drawing and reinjecting blood from the cannulated coronary artery. A second coronary arteriogram was obtained from 1 to 5 minutes following the embolization. The catheter was then withdrawn into the aorta. In 4 experiments the second coronary arteriogram was obtained at the end of the procedure. After 45 minutes to 1 hour, all basal determinations were repeated. The blood volume was maintained constant in all experiments where blood was drawn. Metabolic studies were done in only the first 10 experiments.

Results. All animals surviving the experiment were sacrificed 3-5 days later and the heart examined. Large infarcts of both ventricles could easily be seen macroscopically (Fig. 1).

The degree of coronary occlusion caused by the ball bearings was clearly demonstrated by the coronary arteriograms (Fig. 2). No change in diameter of the coronary arteries could be seen.

The hemodynamic changes are summarized in Table I. A fall in cardiac output occurred in all experiments (3.13 to 2.13 l/min). Mean aortic pressure fell in most experiments (123 to 99 mm Hg); in 3 instances

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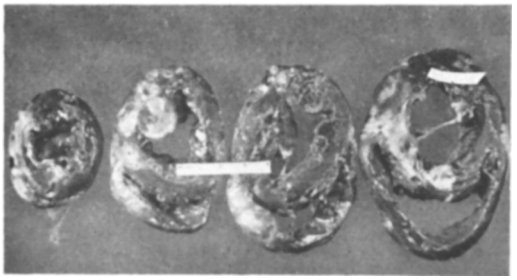

FIG. 1. Heart of a dog sacrificed 4 days after production of myocardial infarction.

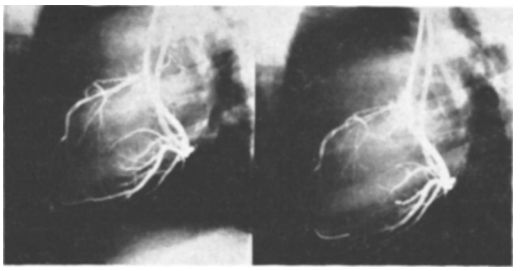

FIG. 2. Coronary arteriograms obtained before and after coronary embolization.

it increased. Peripheral vascular resistance increased in 16 of the 23 experiments. A significant fall in coronary blood flow (104 to 66 cc/100 g of lv/min) occurred in all 8 experiments where it was determined. No significant change in coronary vascular resistance was observed. A well developed picture of congestive heart failure with high left ventricular end-diastolic, left atrial and pulmonary artery pressures occurred in 3 of 12 experiments where 4 or more ball bearings/kg of body weight were injected. In the remaining observations the mean pressure in the pulmonary artery fell (16 to 13 mm Hg) and no significant change in the end-diastolic pressure of either ventricle could be observed.

The metabolic findings are grouped in Table II. A significant increase in the arterial level of lactate (9.56 to 13.84 mg%) and pyruvate (.337 to .470 mg%) was observed. Myocardial oxygen extraction increased (14.10 to 15.10 vol%). No consistent change in myocardial extraction of glucose, lactate, pyruvate and free fatty acids was observed.

Discussion. It has been frequently reported that myocardial infarction causes a decrease in cardiac output(10-16). The frequent fall in systemic arterial pressure and

TABLE I. Experimental Myocardial Infarction —Hemodynamic Changes

	Before MI	After MI	p Value
Rate, b/min	148	145	>.60
CO, l/min	3.13	2.13	<.001
MAP, mm Hg	123	99	<.001
TPR, mm Hg/l/min	40.1	45.3	>.10
CBF, cc/100 g LV/min	104	66	<.005
CVR, mm Hg/cc/100 g LV/min	1.12	1.33	>.90
LAP, mm Hg	5	6	>.40
PAP, mm Hg	16	13	<.05

MI, myocardial infarction; CO, cardiac output; MAP, mean aortic pressure; TPR, total peripheral resistance; CBF, coronary blood flow; CVR, coronary vascular resistance; LAP, left atrial pressure; PAP, pulmonary arterial pressure.

increase in total peripheral resistance are also well established facts(10-13,17,18). Although it is known that heart failure can be present as a result of experimentally produced myocardial infarction(11,12,19), the degree of myocardial necrosis necessary to produce it has not been emphasized. In this series of experiments, heart failure characterized by high end-diastolic left ventricular, left atrial and pulmonary artery pressures was a rare occurrence and only observed when practically all branches of the circumflex artery had been occluded. Decrease of ejection power of the right ventricle(20) was probably the reason for the fall in the pulmonary artery pressure, since an infarct of

TABLE II. Experimental Myocardial Infarction —Metabolic Changes.

	Before MI	After MI	p Value
Oxygen, A – V, vol %	14.10	15.10	<.05
FFA, μEq/l			
BA	716	749	>.50
Δ A – V	214	225	>.80
Glucose, mg %			
BA	88	87	>.70
Δ A – V	14	9	>.70
Lactate, mg %			
BA	9.56	13.84	<.05
Δ A – V	6.10	6.60	>.70
Pyruvate, mg %			
BA	.337	.470	<.05
Δ A – V	.056	.024	>.70

MI, myocardial infarction; BA, arterial; ΔA–V, arterial minus coronary sinus.

the right ventricle was present in all examined specimens. The reported changes in coronary blood flow following experimental myocardial infarction have been contradictory(10,21). Our finding of a decrease in coronary blood flow is correlated with the increase in myocardial oxygen extraction, which compensates for the diminished blood supply to the myocardium. The cardiac output and coronary perfusion pressure, two important determinants of the coronary blood flow were decreased. The observed changes in coronary vascular resistance were not consistent and no change in the caliber of the patent coronary arteries was seen radiographically.

The only consistent metabolic change observed was the increase in the arterial level of lactate and pyruvate, probably as a result of an increased rate of systemic glycolysis. No definite evidence of myocardial glycolysis was detected. Changes in the myocardial extraction of glucose, lactate, pyruvate and free fatty acids were not consistent, suggesting that the non-infarcted portion of the myocardium derives its energy from the same substrates and possibly through the same pathways utilized by myocardium of the normal heart.

Summary. A method for production of myocardial infarction by injecting steel ball bearings into the coronary arteries of closed chest dogs is described. Hemodynamic changes resulting from the myocardial infarcts were: A fall in the aortic pressure, cardiac output and coronary blood flow; a picture of congestive heart failure was observed in a few cases of severe infarction. Metabolic changes observed were increased myocardial oxygen extraction and increased arterial levels of lactate and pyruvate. No consistent change in myocardial extraction of carbohydrates and free fatty acids was observed.

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