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Title : DOES DELIBERATE HYPOTENSION PRODUCE MYOCARDIAL ISCHEMIA WHEN THE CORONARY ARTERY IS STENOTIC?

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Introduction. Deliberately inducing hypotension during anesthesia permits certain surgical procedures to be performed with increased patient safety. Ischemic heart disease is considered a contraindication to the use of deliberate hypotension, the rationale being that the lowered perfusion pressure produced both by a narrowed coronary artery and by hypotension would prove inadequate, and that myocardial ischemia would occur. The purpose of this study is to determine the effect of deliberate hypotension on regional myocardial blood flow and oxygenation distal to a coronary artery stenosis.

Method. Five mongrel dogs underwent left thoracotomy with halothane-oxygen anesthesia. After exposure of the left anterior descending coronary artery (LAD), an electromagnetic flow transducer and a small plastic clamp were placed on the artery near its origin. Small catheters were placed in the distal LAD and in the great cardiac vein (GV). The GV drains the area of myocardium perfused by the LAD. The left ventricle, the left atrium, the coronary sinus (CS), and the central aorta were also catheterized. Regional blood flow was measured by injecting 9 and 15 μ radioactive microspheres. Measurements were made of oxygen and lactate concentrations across the heart. Electrocardiographic leads were inserted into the epicardium and the endocardium of the myocardium perfused by the LAD. Measurements were made of heart rate (HR), cardiac output (CO), left-ventricular end-diastolic pressure (LVEDP), left atrial pressure (LAP), CS pressure, LAD pressure, LAD flow, and regional myocardial flow. Hemodynamic and metabolic measurements were made at eight time periods: control, after 15 and 30 min of stenosis, sequentially during 2 hr of hypotension (mean aortic pressure, 50 torr), and after restoration of normal pressure. Hypotension was induced with deep halothane. Two degrees of stenosis were studied, moderate and severe. Moderate stenosis did not reduce resting coronary flow but prevented any increase in flow in response to 10 sec of coronary artery occlusion. This stenosis is defined as a critical stenosis and has been shown by Elzinga[1] to represent a 75 per cent reduction in coronary artery diameter. Severe stenosis reduced resting flow by 40 per cent. After the study ended, the dogs were killed, and the LAD and circumflex arteries were perfused with different colored dyes. The left ventricle was serially sectioned and divided into inner, middle, and outer layers.

Results. Neither degree of stenosis changed regional myocardial flow, lactate extraction, EKG, or CS or GV oxygen saturation during normotension. Inducing hypotension with halothane required increasing halothane (mean ± SE) from 1.2 ± 0.1 to 2.1 ± 0.2 MAC. This increase in halothane concentration resulted in decreases (mean ± SE) in CO to 35.5 ± 5.1 per cent, HR to 90 ± 6.0 per cent, and myocardial oxygen consumption to 40 ± 10 per cent control. Neither LVEDP nor LAP changed. Dogs with moderate (critical) stenosis showed no evidence of myocardial ischemia. These dogs had no changes in lactate extraction, EKG, or CS or GV oxygen saturation. Dogs with moderate stenosis had a marked reduction in regional myocardial flow, but this reduction was proportionate to the reduction in myocardial oxygen consumption during hypotension. Furthermore, regional myocardial flow was the same in the myocardium perfused by the stenotic artery as in the normal myocardiums. Dogs with severe stenosis developed myocardial ischemia during hypotension, as evidenced by EKG, lactate production, and regional blood flow (table 1).

Discussion. The combination of severe coronary artery stenosis and reduction in blood pressure by deep anesthesia produces endocardial ischemia. Deep anesthesia provides a measure of protection by reducing MVO_2. The reduced flow requirement can be seen in Table 1 by examining the flow to the normal myocardium. This protection is only partial, as ischemia does occur in the area supplied by the stenotic coronary artery. This ischemia is demonstrated by the extremely low endocardial flow, lactate production, and EKG changes.

Reference

1. Elzinga WE, Skinner DB: Hemodynamic characteristics of critical stenosis in canine coronary arteries. J Thorac Cardiovasc Surg 69:217-222, 1975

Table 1A. Severe Stenosis - Hemodynamics

	CO (l/m)	HR (beats/min)	Diastolic Pressure Aorta (torr)	Diastolic Pressure LAD (torr)
Control	3.17	107	92.0	91.7
Stenosis	2.95	106	85.7	53.0
Stenosis plus hypotension	1.5	106	42.0	20.6

*Pressure measured distal to stenosis.

Table 1B. Severe Stenosis - Regional Flow and Lactate Extraction

	Endocardial Blood Flow N	Endocardial Blood Flow I	Epicardial Blood Flow N	Epicardial Blood Flow I	Lactate Extraction (%) A-CS	Lactate Extraction (%) A-GV
Control	0.874	0.802	0.756	0.828	37.3	45.7
Stenosis	0.625	0.578	0.568	0.563	31.2	48.6
Stenosis plus hypotension	0.347	0.100	0.373	0.210	20.6	-34.5

N = myocardium perfused by normal coronary artery; I = myocardium perfused by stenotic coronary artery; A = artery; CS = coronary sinus; and GV = great vein.