

Title: FACTORS AFFECTING ENDOCARDIAL CIRCULATION
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Introduction. Left ventricular ischemia and infarction in the presence of patent coronary arteries is a frequent cause of cardiac failure following cardiopulmonary bypass (1) or hemorrhagic shock. The endocardial viability ratio (EVR), a ratio of diastolic pressure time index to tension time index (DPTI/TTI), has been used to define the endocardial perfusion. However, this parameter has been proven to be not sufficiently sensitive in detecting endocardial ischemia. Direct measurements of endo/epimyocardial blood flow ratio may help to elucidate the pathophysiological mechanism of the endocardial ischemia under various experimental conditions.

Methods. Thirty-two pentobarbitalized dogs (10-20 kg) were used: 20 for hematocrit (Hct) alteration (ranged 12 - 78%), 6 for nitroprusside induced hypotension, 3 for aortic occlusion and 3 for hemorrhage experiments. Hemodilution was induced with isovolemic exchange of whole blood with donor plasma, and hemoconcentration with donor packed cells. Nitroprusside was infused IV at a constant rate to lower the blood pressure first to 75% and then to 50% of control blood pressure. An umbilical tape was threaded around the ascending aorta, proximal to major branches, to effect partial aortic occlusion. Hemorrhage was induced by gradual bleeding with approximately 10 ml/kg at each step. Microspheres (15 ± 1 μ, labeled with radionuclides ⁵⁷Co, ¹¹³Sn, ¹⁰³Ru, ⁹⁶Nb and ⁴⁶Sc) were injected into the left ventricle to determine the cardiac output and regional myocardial blood flows in the control state and 30 min after each experimental condition has been established.

Results. Marked epi- and endocardial vasodilation was noted in extreme hemodilution and hemoconcentration, with endocardium showing less vasodilation than the epicardium. As a result, the endo/epi flow ratio decreased when the Hct was altered towards either extreme. Similar results were obtained from nitroprusside induced hypotension, aortic occlusion and hemorrhage experiments. In all experimental conditions, the endo/epi flow ratio shows close correlation with coronary vascular hindrance (coronary flow resistance/blood viscosity) but not with EVR (Fig. 1). The correlation is less marked when coronary flow resistance (vascular hindrance x blood viscosity) is used instead of vascular

hindrance.

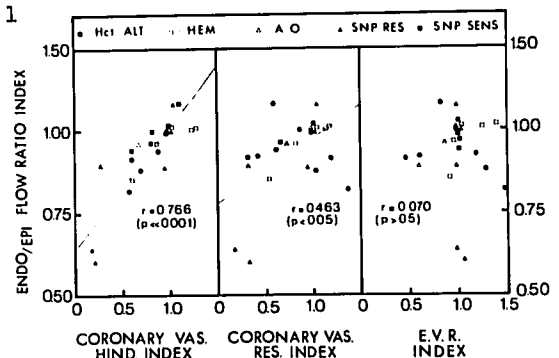
Discussion. The greater vulnerability of the subendocardial muscle to ischemia than the epicardial muscle can be attributed to the following reasons: First, subendocardial muscle might have a greater oxygen requirement, therefore it is more vulnerable to a decrease of oxygen supply. Second, the endocardial blood vessels are normally more dilated than the epicardial ones. Therefore, any further decrease in oxygen supply/demand ratio, which is generally accompanied by a marked coronary vasodilation, can result in endocardial ischemia. The EVR, frequently utilized to estimate the endocardial oxygen supply/demand ratio, cannot properly predict the endocardial hemodynamics. These results indicate the importance of relative regional changes in coronary vascular geometry in the occurrence of endocardial ischemia. An understanding of the overall and regional coronary hemodynamics, including the endo/epi blood flow ratio, may help to further our understanding of the mechanism of development of endocardial ischemia.

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References.

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Fig. 1



Relationship between the endo/epi flow ratio index and the coronary vascular hindrance index, the coronary vascular resistance index, and the EVR index. Hct alt = Hematocrit alteration. HEM = Hemorrhage. A.O. = Aortic occlusion. SNP RES = Nitroprusside resistant, and SNP SENS = Nitroprusside sensitive.