Hepatic blood flow in acute myocardial ischemia

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Summary. Hepatic blood flow was monitored in cats during myocardial ischemia (MI). Increased plasma CPK activity, the ST segment of the electrocardiogram, and hepatic flow was reduced by 5 h to 40% of control. The results suggest that MI can influence organs distant from the original ischemic episode.

Myocardial ischemia (MI) is a complex disease entity involving a variety of cardiac and extracardiac phenomena. Information regarding the cardiac processes responsible for the spread of ischemic damage in acute myocardial ischemia is becoming better understood. However, extracardiac processes involved in the pathogenesis of acute myocardial ischemia remain poorly understood. The purpose of this study was to determine if myocardial ischemia, uncomplicated by cardiogenic shock, alters liver blood flow. If liver blood flow is compromised it could induce tissue injury resulting in a deficit in energy metabolism, as well as an impairment in phagocytosis of the reticuloendothelial system.

Methods. Male cats (3.8-5.3 kg) were anesthetized with...
sodium pentobarbital (30 mg/kg, i.v.). The right carotid artery and left jugular vein were cannulated. Mean arterial blood pressure (MABP) and central venous pressure (CVP) were recorded from the carotid arterial and jugular venous catheters respectively, using a Grass Model 7 oscillographic recorder. Lead III of the scalar electrocardiogram was also monitored. All cats were tracheotomized to allow for positive pressure ventilation after thoracotomy. A mid-line laparotomy was performed with subsequent isolation of the hepatic artery and hepatic portal vein. A noncannulating electromagnetic flow probe (i.d. = 1.5 mm) was carefully positioned around the common hepatic artery to ensure that flow through the hepatic artery perfused only the liver. In addition, a cannulating electromagnetic flow probe (i.d. = 3.0 mm) was placed in the lumen of the hepatic portal vein for measurement of portal venous flow (PVF). This procedure necessitated ligation of the right gastric and gastroduodenal veins for placement of the flow probe. Heparin (250 U/kg) was administered i.v., and the abdomen was closed. Subsequently, a mid-sternal thoracotomy was performed, and the exposed pericardial sac was incised and retracted. This exposed the left coronary artery and its branches. Myocardial ischemia (MI) was induced via ligation of the left anterior descending (LAD) coronary artery 10-14 mm from the coronary ostium. This ligation was accomplished by passing a 3-0 silk ligature around the LAD coronary artery and tying the vessel securely. Myocardial ischemia was initiated after a 15 min stabilization period. In sham-operated controls, the identical experimental maneuvers were performed except that the LAD artery was not occluded.

**Sampling protocol and biochemical assays.** 3 ml venous blood samples were drawn just prior to occlusion and every hour thereafter for 5 h. The blood was centrifuged at 2500 x g and 4 °C for 20 min. The plasma was collected for the determination of plasma protein concentration, and creatine phosphokinase (CPK) activity. Saline (6 ml of 0.9% NaCl) was given to replace blood lost by sampling. The biuret method of Gornall et al. was used to determine plasma protein concentrations. Plasma creatine phosphokinase activity was determined by the method of Rosalki.

**Results.** The cardiovascular and biochemical responses of cats subjected to myocardial ischemia and their controls are summarized in the table. In both the sham-operated and myocardial ischemia groups, MABP did not change significantly. In contrast, the plasma CPK activity and the S-T segment of the electrocardiogram increased progressively in the MI group over the 5 h observation period. No significant changes in plasma CPK activity or S-T segment voltage were observed in sham-operated controls. These findings indicate myocardial ischemia existed in cats subjected to coronary artery occlusion, and was not present in sham-operated controls.

Total hepatic blood flow for cats subjected to either acute MI or sham-MI for the 5 h observation period is depicted in the figure. The initial blood flow for both the control and experimental groups was 28.5 and 27.3 ml/kg/min. In the sham-operated control cats, no significant changes in hepatic blood flow were observed over the 5 h experimental period. In contrast, liver blood flow decreased in MI cats within 2 h of coronary artery ligation and progressively declined over the remaining 3 h of the experimental period, so that flow had declined by 60%, 5 h after coronary artery occlusion. Both hepatic portal flow and hepatic arterial flow declined proportionally so that the decreased total hepatic flow was equally distributed between its 2 vascular supplies.

**Discussion.** Although there is considerable information available on the cardiac adjustments following myocardial ischemia, a paucity of information exists on the extracardiac mechanisms activated in response to acute MI. Our findings indicate myocardial ischemia initiates vascular changes in the liver, an organ distant from the original ischemic episode.

The data presented in the table indicate coronary occlusion resulted in a significant degree of MI after ligation of the left anterior descending coronary artery. The changes noted in plasma CPK activity and the S-T segment of the electrocardiogram are similar to those known to occur in cats following coronary artery occlusion. The stable blood pressure observed in the MI group indicates that uncomplicated myocardial ischemia without cardiogenic shock was induced in these cats. In addition, in similar experiments, Spath and Lefer have reported a 35% reduction in aortic blood flow at 5 h of post myocardial ischemia. Thus, the 60% decrease in hepatic blood flow we observed was not a consequence of severely compromised cardiac performance, aortic blood flow or hypotension which occurs in hemorrhagic and other types of shock.

The control liver flows obtained in the experiments are comparable to those reported by Nxumalo et al. and Lautt. The reduction in liver blood flow in response to

![Diagram of liver blood flow over time](image)

**Myocardial ischemia induced changes in mean arterial blood pressure, S-T segment and plasma creatine phosphokinase activity**

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Time (h)</th>
<th>MABP</th>
<th>CPK</th>
<th>S-T</th>
<th>SABP</th>
<th>CPK</th>
<th>S-T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sham-MI</td>
<td>6</td>
<td>0</td>
<td>135 ± 11</td>
<td>3.1 ± 0.9</td>
<td>0.01 ± 0.01</td>
<td>116 ± 6</td>
<td>3.8 ± 0.7</td>
<td>0.01 ± 0.01</td>
</tr>
<tr>
<td>MI</td>
<td>7</td>
<td>0</td>
<td>127 ± 8</td>
<td>2.8 ± 0.7</td>
<td>0.01 ± 0.01</td>
<td>119 ± 5</td>
<td>9.6 ± 0.7 ∗</td>
<td>0.17 ± 0.05 ∗</td>
</tr>
</tbody>
</table>

MABP = mean arterial blood pressure expressed in mm Hg; CPK = creatine phosphokinase activity of the plasma expressed in IU/mg protein × 10²; S-T segment change in lead III of EKG expressed in mV. * p < 0.02 compared to zero time value.