Mechanical Increase of Vascular Resistance in Experimental Myocardial Infarction with Shock

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ABSTRACT

The hemodynamic and cardiac metabolic effects of increasing central aortic pressure by obstructing the abdominal aorta with a balloon catheter introduced via a femoral artery were determined in 28 anesthetized dogs with acute myocardial infarction and shock produced by coronary embolization with plastic spheres. Following coronary embolization, aortic pressure, cardiac output, and left ventricular mechanical efficiency declined in all animals; left ventricular “excess lactate” appeared in about one-half. With abdominal aortic obstruction for 1 hour, there were significant elevations of postembolic aortic pressure, coronary flow, cardiac output, left ventricular oxygen consumption and mechanical efficiency. Left atrial pressure rose slightly but not beyond normal limits. Arterial-coronary sinus oxygen difference diminished, and left ventricular excess lactate diminished or disappeared in 60% of the animals in which it was noted in the postembolic state. After 1 hour of abdominal aortic obstruction, cardiac output, central aortic pressure, and coronary flow diminished moderately. It is concluded that mechanical increase of vascular resistance for periods up to 1 hour may improve myocardial performance in acute myocardial infarction with shock. The increased left ventricular oxygen needs are met adequately by the increase of coronary flow associated with the increase of perfusion pressure.

ADDITIONAL KEY WORDS
coronary flow myocardial metabolism
cardiac output myocardial lactate dogs

In the treatment of patients with acute myocardial infarction and shock, it appears rational to use measures which may increase the acutely reduced cardiac output and lessen myocardial ischemia. In such patients, it has been traditional to attempt to raise arterial blood pressure and hence, coronary flow, with various pressor agents having both inotropic and peripheral vasoconstrictive effects; beneficial results have been reported from their use (1-5). However, more recently, it has been suggested that other therapeutic approaches may be preferable, including the use of vasodilating agents (6, 7), which may actually lower coronary perfusion pressure, and of agents that have only an inotropic effect (8). Advocates of these measures have stressed the potential harm in raising the pressure load against which the acutely infarcted left ventricle must pump.

In this study, we analyzed the hemodynamic and cardiac metabolic effects of increasing coronary perfusion pressure and coronary blood flow in dogs with acute myocardial infarction and shock by mechanically increasing central aortic pressure. Our goal was to determine whether the considerable increase of coronary flow resulted in an improvement of myocardial performance despite the concomitant increase of left ventricular “pressure...
VASCULAR RESISTANCE IN EXPERIMENTAL MYOCARDIAL INFARCTION

FIGURE 1
Schematic representation of the experimental arrangement. See text for details.

Methods
We studied 28 dogs anesthetized with a solution of 0.4% chloralose and 4% urethane given intravenously in doses sufficient to produce light anesthesia. Three hundred ml of this solution was infused rapidly. This was followed generally by a slow infusion of 50 to 75 ml, depending on the response of the animal. The total dose that was administered ranged from 1.2 to 1.4 g of chloralose and 12 to 14 g of urethane. A thoracotomy was performed in the 4th right interspace and artificial ventilation with air was maintained through an endotracheal tube. Figure 1 is a schematic representation of the experimental arrangement. After a small incision was made in the pericardium, a large polyethylene catheter with a flanged end was inserted via the right atrial appendage approximately 2 cm into the mouth of the coronary sinus and sutured in place. This catheter was connected by a small length of Tygon tubing to a large-bore Bardic catheter which was inserted into a femoral vein. A three-way stopcock was placed in the coronary sinus-femoral vein circuit for periodic sampling of coronary sinus blood and for measurement of coronary sinus flow by gravity drainage. Preliminary experiments with this tubing arrangement indicated that the coronary sinus flow measured at the far end of the Bardic catheter was the same as that measured with only the short polyethylene catheter introduced into the coronary sinus. In addition, in a few experiments, coronary sinus flow was measured by an electromagnetic flowmeter applied to the tubing system; this flow was the same as that obtained from the end of the short catheter introduced directly into the coronary sinus. Therefore, although the resistance to coronary sinus outflow imposed by the shunt from the coronary sinus to the femoral vein was not determined directly, the shunt did not elevate resistance to coronary sinus outflow sufficiently to affect coronary sinus flow.

Through the same chest incision, polyethylene tubing was inserted through a segmental pulmonary vein into the left atrium for measurement of left atrial pressure. The zero reference level for left atrial pressure was the midleft atrium as determined by direct inspection. A thin-walled Lehman aortographic catheter was passed in a retrograde direction from a femoral or carotid artery into the ascending aorta just distal to the coronary arterial orifices. The catheter was first inserted into the left ventricle and then withdrawn until an aortic pressure pulse appeared on the monitoring screen; it was used for injection of plastic microspheres, measurement of central aortic pressure, withdrawal of arterial blood for sampling and cardiac output determinations.

Myocardial infarction with shock was produced by our modification (9) of the technique of Agress and co-workers (10). Polystyrene microspheres 325 μm in diameter, 3 to 4 mg/kg, were injected with a pressure injector into the ascending aorta during transient asystole produced by rapid intravenous injection of 0.4 mg/kg acetylcholine.

At least a 30% fall in cardiac output and mean central aortic pressure, persisting for 30 min following coronary embolization, was required for the animal to be considered in “shock” and for further studies to be performed. About 75% of all animals developed shock with only one injection of microspheres; several required two or three injections of microspheres before the desired hemodynamic alterations could be produced. Some did not develop shock even after multiple injections of microspheres.

This method consistently produces diffuse subendocardial infarction involving both ventricles in those that survive for 6 or more hours after coronary embolization (11). The animals in this study were killed after the experiment, before...
sufficient time had elapsed to develop these pathological changes.

Obstruction of the abdominal aorta was produced by inflating a balloon catheter which had been inserted via a femoral artery to the most caudal point of the abdominal aorta at which obstruction produced a significant rise of central aortic pressure. To achieve this it was necessary to place the balloon just above the renal arteries and below the superior mesenteric artery in 22 of 28 animals and below the renal arteries in 6 of 28 animals. After each experiment, we opened the abdomen and noted the exact position of the balloon catheter. The change in cardiac output, central aortic pressure, left atrial pressure and coronary sinus flow before or after aortic obstruction was not significantly different in these two groups of animals.

Cardiac output, coronary sinus flow, central aortic and left atrial pressure were first determined after anesthesia; then 30 min after coronary embolization when there was a stable hemodynamic state; 15, 30 and 60 min after aortic obstruction; and 15 min after slow deflation of the balloon. Samples were drawn simultaneously from the aorta and coronary sinus at these intervals for determination of pH, Po2, and the concentration of hemoglobin, lactate and pyruvate.

Pressures were determined with Statham strain gauges and were recorded on a multichannel, photographic oscillographic recorder. Cardiac output was measured by the dye-dilution technique; indocyanine green dye was injected into a central vein or the right atrium and blood samples were drawn from a carotid artery through a densitometer by a constant-speed motor-driven syringe. The resultant curve was replotted on semilog paper. Circulation time from the right atrium to the aorta was measured from the onset of injection of the dye to the initial recorder deflection indicating the appearance of dye at the ascending aorta. Po2 and pH were determined by Instrumentation Laboratory electrodes, and hemoglobin by a Coleman Universal spectrophotometer.

Lactate and pyruvate were measured by the methods of Barker and Summerson (12) and Friedemann and Haugen (13). "Excess lactate" of the left ventricle was calculated by the method of Huckabee (14). It was assumed that the left ventricle produced the excess lactate determined by Huckabee's formula, \( XL = (L_a - L_v) - (P_a - P_v) \times (L_a/P_v) \), where \( XL \) = "excess lactate"; \( v \) = coronary sinus; \( a \) = aorta; \( L \) = mm lactate; and \( P \) = mm pyruvate.

Systemic vascular resistance, left ventricular work, left ventricular oxygen consumption, coronary vascular resistance and "mechanical efficiency" of the left ventricle were calculated from the above measurements, using conventional formulas. Left ventricular weight was calculated from the body weight, using the ratio derived from the experiments of Herrmann (15). We assumed that the coronary sinus drained blood from most of the left ventricle, although the relative amount of left ventricular myocardium and of other cardiac chambers drained by the coronary sinus may vary among different animals.

Oxygen consumption of the left ventricle was calculated as the product of coronary flow in milliliters per 100 grams left ventricle per minute and the left ventricular arteriovenous oxygen difference (milliliters of oxygen per 100 milliliters of blood). Oxygen saturation of blood from the coronary sinus and aorta was calculated from the measured oxygen tension (after correcting for the temperature and pH of each sample) and the oxygen dissociation curve of hemoglobin. Oxygen capacity was calculated from the measured hemoglobin concentration assuming that each gram of hemoglobin, when fully saturated, carried 1.34 ml of oxygen. By knowing the percentage of oxygen saturation and oxygen capacity, the oxygen content of the specimen could then be determined.

Left ventricular "mechanical efficiency" was determined by dividing the work of the left ventricle (in kg-m/min) by the energy cost of the left ventricular work. The latter was calculated as left ventricular oxygen consumption (ml/min) \( \times 2.06 \); this is the energy equivalent in kilograms of 1 ml of oxygen consumption.

Ten dogs were studied to determine the effects of suprarenal aortic obstruction for a period of 1 hour on renal function and structural abnormalities. The same anesthesia was given, but the chest was not opened and both ureters were cannulated with polyethylene tubing via flank incisions. The balloon was positioned just above the renal arteries. Urine flow was measured and, in 5 animals, blood urea nitrogen was determined preoperatively and 24 and 48 hours postoperatively. The animals were sacrificed at periods varying from 1 hour to 1 week following the experiment, and the kidneys were examined by a pathologist.

Results

The hemodynamic effects are indicated in Table 1 and Figure 2. Following coronary embolization and before balloon inflation there were significant declines of cardiac output, stroke volume and aortic pressure. Coronary sinus flow did not decrease significantly; left atrial pressure increased slightly but not above normal limits. The heart rate slowed moderately. The left atrial and left ventricular end-diastolic pressure remained normal initially.
**TABLE 1**

**Hemodynamic Effects of Mechanical Increase of Vascular Resistance Following Coronary Embolization**

<table>
<thead>
<tr>
<th></th>
<th>No. of dogs†</th>
<th>Preembolic</th>
<th>Before balloon inflation</th>
<th>After balloon inflation</th>
<th>15 min</th>
<th>30 min</th>
<th>60 min</th>
<th>Balloon deflated</th>
</tr>
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<tbody>
<tr>
<td>Cardiac output</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(ml/kg/min)</td>
<td>21</td>
<td>97</td>
<td>63</td>
<td>93*</td>
<td>89*</td>
<td>79*</td>
<td>58</td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>28</td>
<td>144</td>
<td>125</td>
<td>122</td>
<td>119</td>
<td>107</td>
<td>105</td>
<td></td>
</tr>
<tr>
<td>(beats/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central aortic pressure</td>
<td>28</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mm Hg)</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>(4)</td>
<td>114</td>
<td>75</td>
<td>129</td>
<td>126</td>
<td>117</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Diastolic</td>
<td>(3)</td>
<td>76</td>
<td>50</td>
<td>98*</td>
<td>98*</td>
<td>89*</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>(3)</td>
<td>91</td>
<td>64</td>
<td>117*</td>
<td>117*</td>
<td>106*</td>
<td>56</td>
<td></td>
</tr>
<tr>
<td>Stroke volume</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(ml)</td>
<td>21</td>
<td>15</td>
<td>11</td>
<td>17*</td>
<td>17*</td>
<td>16*</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Left atrial mean pressure</td>
<td>27</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>(mm Hg)</td>
<td>(0.5)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td></td>
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<tr>
<td>Systemic vascular resistance</td>
<td>21</td>
<td>2836</td>
<td>2783</td>
<td>4256*</td>
<td>4662*</td>
<td>4457*</td>
<td>2530</td>
<td></td>
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<tr>
<td>(dynes sec cm⁻⁵)</td>
<td>(722)</td>
<td>(902)</td>
<td>(1485)</td>
<td>(485)</td>
<td>(400)</td>
<td>(30)</td>
<td></td>
<td></td>
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<tr>
<td>Circ. time</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>(RA-aorta) (sec)</td>
<td>20</td>
<td>12</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>15</td>
<td></td>
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<tr>
<td>Cor. sinus flow</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(ml/100 g LV/min)</td>
<td>25</td>
<td>52</td>
<td>49</td>
<td>91*</td>
<td>92*</td>
<td>84*</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Cor. vasc. resist.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>(dynes sec cm⁻⁵)</td>
<td>22</td>
<td>137793</td>
<td>97833</td>
<td>112990</td>
<td>125392</td>
<td>111612</td>
<td>82675</td>
<td></td>
</tr>
<tr>
<td>Left ventric. work</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(kg-m/min)</td>
<td>21</td>
<td>2.06</td>
<td>0.76</td>
<td>2.53*</td>
<td>2.54*</td>
<td>1.93*</td>
<td>0.71</td>
<td></td>
</tr>
<tr>
<td>(kg-m/min/kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>0.091</td>
<td>0.037</td>
<td>0.117*</td>
<td>0.114*</td>
<td>0.087*</td>
<td>0.036</td>
<td></td>
</tr>
<tr>
<td>Left ventric. stroke work</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(kg-m/beat)</td>
<td>21</td>
<td>0.014</td>
<td>0.007</td>
<td>0.027*</td>
<td>0.022*</td>
<td>0.018*</td>
<td>0.006</td>
<td></td>
</tr>
</tbody>
</table>

Mean values with standard error in parentheses.

*Change from postembolic level obtained prior to balloon inflation: P < 0.05.

†Mean weight = 23.1 (±1.1) kg.
Obstruction of the abdominal aorta resulted in rise of central aortic pressure, coronary sinus flow, cardiac output, stroke volume and calculated left ventricular work. Left atrial pressure rose in several animals but still remained within normal limits. Changes of left atrial pressure in the group as a whole were not of statistical significance.

The responses to aortic obstruction of 15 animals that had no or less than 10% rise in systemic vascular resistance following coronary embolization before aortic obstruction, were compared to those in 6 animals showing a greater rise. Cardiac output increased in the two groups, as did central aortic pressure and vascular resistance. Coronary flow increased even though portions of the peripheral coronary arterial tree were occluded by the microspheres.

Release of the balloon at the end of 1 hour resulted in return to the postembolic levels of cardiac output, aortic pressure and coronary sinus flow obtained prior to balloon obstruction.

**EFFECTS ON RENAL FUNCTION**

No urine flow was detected during aortic obstruction in the animals in which bilateral ureteral catheterization was performed; anuria persisted for 2 to 4 hours after the balloon was deflated. There was no elevation of blood urea nitrogen determined 24 and 48 hours postoperatively in any of the 5 animals studied. One day to 1 week after abdominal aortic obstruction for 1 hour, no significant gross or microscopic renal abnormality was apparent in these animals. In animals sacrificed earlier than this, focal areas of renal hemorrhagic congestion were noted.

**METABOLIC EFFECTS**

Alterations of left ventricular oxygen consumption, efficiency and lactate extraction are
Effects on Cardiac Metabolism of Mechanical Increase of Vascular Resistance Following Coronary Embolization

<table>
<thead>
<tr>
<th>Arterial-cor. sinus</th>
<th>No. of dogs</th>
<th>Preembolic</th>
<th>Before balloon inflation</th>
<th>Postembolic</th>
<th>Postembolic</th>
<th>After balloon inflation</th>
<th>Balloon deflated</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂ diff. (ml O₂/100 ml blood)</td>
<td>22</td>
<td>13.84</td>
<td>13.79</td>
<td>7.75*</td>
<td>7.15*</td>
<td>7.25*</td>
<td>9.14</td>
</tr>
<tr>
<td>LV consumpt. (ml O₂/100 g LV/min)</td>
<td>22</td>
<td>5.61</td>
<td>4.19</td>
<td>5.41*</td>
<td>5.22*</td>
<td>4.64*</td>
<td>3.09</td>
</tr>
<tr>
<td>Hemoglobin (g/100 ml)</td>
<td>23</td>
<td>16.4</td>
<td>16.9</td>
<td>16.2</td>
<td>15.0</td>
<td>14.4</td>
<td>15.4</td>
</tr>
<tr>
<td>LV mechanical efficiency (%)</td>
<td>16</td>
<td>21.1</td>
<td>12.3</td>
<td>25.5*</td>
<td>25.5*</td>
<td>21.2*</td>
<td>13.2</td>
</tr>
<tr>
<td>Arterial blood lactate (mm)</td>
<td>16</td>
<td>.61</td>
<td>.77</td>
<td>.88</td>
<td>.91</td>
<td>.96</td>
<td>1.04</td>
</tr>
<tr>
<td>Coronary sinus blood lactate (mm)</td>
<td>16</td>
<td>.50</td>
<td>.87</td>
<td>.83</td>
<td>.84</td>
<td>.91</td>
<td>.96</td>
</tr>
<tr>
<td>Arterial blood pyruvate (mm)</td>
<td>16</td>
<td>.027</td>
<td>.029</td>
<td>.031</td>
<td>.032</td>
<td>.031</td>
<td>.03</td>
</tr>
<tr>
<td>Coronary sinus blood pyruvate (mm)</td>
<td>16</td>
<td>.018</td>
<td>.023</td>
<td>.028</td>
<td>.028</td>
<td>.022</td>
<td>.02</td>
</tr>
<tr>
<td>LV &quot;excess lactate&quot;</td>
<td>16</td>
<td>.00</td>
<td>+.03</td>
<td>-.01</td>
<td>.00</td>
<td>-.05</td>
<td>-.10</td>
</tr>
</tbody>
</table>

Mean values with standard error in parentheses.
*Change from postembolic level obtained prior to balloon inflation: P < 0.05. For discussion of alterations of arterial and coronary sinus lactate, see text.

The degree of change of left ventricular oxygen consumption and mechanical efficiency compared to alterations of cardiac output, central aortic pressure and coronary sinus flow are shown in Figure 2.

Following coronary embolization and prior to abdominal aortic obstruction, left ventricular oxygen consumption diminished, but less than the work of the left ventricle; the efficiency of the left ventricle declined significantly. Arterial-coronary sinus oxygen difference narrowed somewhat concomitant with the fall of coronary vascular resistance and relatively good maintenance of coronary sinus flow. Alterations of left ventricular lactate extraction or production were variable. In 10 of 16 animals, left ventricular excess lactate production developed in the postembolic state, whereas in the remaining 6 animals there was no appreciable change of arterial-coronary sinus lactate difference and no excess lactate production.

Following obstruction of the abdominal aorta, left ventricular oxygen consumption increased; this was apparently met adequately by the very large increase of coronary flow associated with the rise of coronary perfusion pressure. Arterial-coronary sinus oxygen difference diminished during the period of abdominal aortic obstruction; cardiac output rose; and calculated mechanical efficiency of the left ventricle increased considerably. Left ventricular excess lactate production varied. In 6 of the 10 animals that had left ventricular excess lactate production in the postembolic state before aortic obstruction, the lactate disappeared or diminished after abdominal aortic obstruction; 4 showed no significant change. Of the 6 animals with no postembolic left ventricular excess lactate, there was initial appearance of left ventricular excess lactate during aortic obstruction in 1 and no change in 5. Central aortic pressure levels were not higher in this animal than in the others. Arterial
lactate rose moderately, but gradually, during and following aortic obstruction.

**EFFECTS OF DIFFERENT LEVELS OF ARTERIAL PRESSURE**

An attempt was made to determine the effects of different degrees of central aortic pressure elevation on hemodynamic and metabolic alterations. Responses did not vary significantly with the absolute level of aortic pressure. However, when the animals were divided into one group that showed a 50% or greater increase over control mean central aortic pressure during aortic obstruction and one group that showed less increase than this, certain differences in the responses of these two groups could be ascertained (Fig. 3).

The procedure of aortic occlusion was the same in these two groups. There was no consistent difference between them in postembolic cardiac output prior to aortic obstruction or in the site of aortic obstruction. Postembolic central aortic pressure and systemic vascular resistance were somewhat lower in the group responding to abdominal aortic obstruction with a relatively greater rise of central aortic pressure.

Cardiac output increased to a slightly greater extent in the group with higher central aortic pressure. Differences between the groups were not of statistical significance 15 min after balloon obstruction of the aorta, but were at 30 and 60 min. Coronary sinus flow was also significantly greater in the group with relatively higher central aortic pressure. Left ventricular oxygen consumption increased more in this group, which performed greater left ventricular work, but left ventricular mechanical efficiency was also significantly greater.

Left atrial pressure rose to a greater degree during aortic obstruction in those ani-
VASCULAR RESISTANCE IN EXPERIMENTAL MYOCARDIAL INFARCTION

 Alterations of left atrial pressure in the two groups of dogs depicted in Figure 3.

Discussion

Our studies have indicated that in the dog with acute myocardial infarction and shock, increase of coronary perfusion pressure by obstruction of the abdominal aorta for a period of 1 hour was associated with an increase of coronary flow, cardiac output, mechanical efficiency of the left ventricle and in some instances, reversal of excess lactate production by the left ventricle. This did not result in widening of arterial-coronary sinus oxygen difference or rise of left atrial pressure above normal limits. These results indicate that in this type of acute myocardial infarction with shock, left ventricular function may be favorably affected, over this period of time, by a pronounced increase of coronary flow despite a concomitant increase of the pressure load against which the left ventricle must pump. This is consistent with the findings of other investigators that have related deterioration of left ventricular performance to diminished coronary flow (16, 17) and enhancement of left ventricular function to increase of coronary blood flow (18, 19) and suggests that this may also hold in animals with acute myocardial infarction with shock. It has been emphasized previously by Weisberg and his associates (18) that under conditions of hypoxemia cardiac performance may be coronary flow dependent. The response of the acutely ischemic myocardium in these experiments is consistent with those findings.

Caution is, of course, necessary in transferring the conclusions derived from our data in experimental myocardial infarction to a clinical situation in humans. Diffuse coronary embolization with many microspheres may produce different alterations of coronary flow and oxygen diffusion than the acute occlusion of a single large artery in human myocardial infarction. In addition, therapy in humans with
acute myocardial infarction and shock may be of long duration and the prolonged effects of vasoconstriction and organ ischemia may be more deleterious than those seen after 1 hour of abdominal aortic obstruction.

The effects on ventricular function of increasing outflow resistance have been reported by several investigators studying preparations without acute myocardial infarction. Experimental arrangements and conclusions from the experimental data have not been uniform. Increase of cardiac output following thoracic aortic obstruction has been noted by Barcroft (20) and by Gupta and Wiggers (21). Reduction of cardiac output following both short term and longer occlusion of the aorta with a variety of experimental techniques has also been reported (22-28). Sonnenblick and Downing (27) have demonstrated that a relatively constant stroke volume may be maintained over a broad range of aortic pressure without much change in left ventricular end-diastolic pressure. Similarly, Imperial and his associates (28) found stroke work to be unaffected by slight to moderate increases of outflow resistance, but reported an inverse relationship between stroke work and outflow resistance with more severe augmentation of resistance. Adaptive mechanisms permitting the left ventricle to respond to an increase of outflow resistance with enhanced myocardial contractility, thereby preventing an associated rise of left ventricular end-diastolic pressure. Additionally, Rose and Braunwald (32) have indicated that patients with cardiac disease respond to an increase of systemic vascular resistance produced by angiotensin with small changes of stroke work and large elevations of left ventricular end-diastolic pressure. However, their patients did not have apparent prior hypotension or reduction of coronary flow as might individuals with acute myocardial infarction and shock. Goodyer and associates (33) did demonstrate an improvement of ventricular performance in the dog when an initially low coronary perfusion pressure (as in hemorrhage) was increased by aortic obstruction and Villagraña et al. (34), after ligation of a coronary artery, found that constriction of the ascending aorta augmented coronary flow and decreased the area of myocardial infarction compared to that in control dogs without constriction of the aorta. Decreased left ventricular oxygen extraction (as in our experiments) with compression of the aorta in normal dogs has been reported by Feinberg et al. (35). This suggests that the increase of coronary flow produced this way is sufficient to provide the increased oxygen needs of the heart associated with increased outflow resistance, although it has been recognized that the oxygen and lactate concentrations of coronary sinus blood may not reflect uniform venous return from the ischemic and nonischemic areas of the left ventricular myocardium (36, 37). Quite apart from what would appear to be favorable alterations of coronary sinus oxygen and left ventricular lactate extraction during abdominal aortic obstruction in our experiments, improvement of myocardial performance as judged by several other parameters suggests that oxygen delivery to ischemic or partially ischemic areas of the myocardium was enhanced by the increased coronary flow.

Therefore it appears that, at least in acute myocardial infarction, the level of the arterial pressure and the coronary flow are of importance in determining left ventricular response to an increased resistance and that beneficial effects on left ventricular function may be obtained when aortic pressure is raised from shock levels and coronary (and...
cerebral) flow are thereby increased, despite the concomitant increase of left ventricular work. Conclusions from our experiments do not necessarily apply to the hemodynamic situation that obtains in hemorrhagic or septic shock in animals.

Because the pronounced rise of coronary flow and central aortic pressure produced by temporary, mechanical abdominal aortic obstruction for short periods may be beneficial to the acutely infarcted left ventricle, the use of such a technique, perhaps combined with a small shunt from the superior vena cava to the distal aorta, may be helpful in the treatment of patients with myocardial infarction and shock who do not respond satisfactorily to the available pharmacologic agents.

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References

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