Myocardial Ischemia due to Infrarenal Aortic Cross-clamping during Aortic Surgery in Patients with Severe Coronary Artery Disease

Rafik R. Attia, M.D., John D. Murphy, M.D., Michael Snider, M.D., Demetrios G. Lappas, M.D., R. Clement Darling, M.D., and Edward Lowenstein, M.D.

SUMMARY  Hemodynamic measurements were performed and ECG recorded before and shortly after infrarenal aortic cross-clamping during operation for abdominal aortic aneurysm in five patients without evidence of heart disease (group I) and in ten patients with severe coronary artery disease (group II). All patients sustained an increase in systemic arterial pressure. Group I demonstrated a decrease in pulmonary artery, pulmonary capillary wedge pressure (PCW), and central venous pressures when the aorta was clamped, whereas group II demonstrated an increase. The difference in response of the groups is significant (P < 0.05). All three patients who responded to cross-clamping with increases of 7 mm Hg or greater in PCW demonstrated myocardial ischemia during cross-clamping. None of the values measured prior to cross-clamping predicted with certainty the response to cross-clamping.

Sodium nitroprusside reversed the elevation of left ventricular filling pressure in all three patients, and in two patients, relieved evidence of myocardial ischemia concurrently. In the third patient, ventricular irritability was abolished by lidocaine and did not recur. We conclude that infrarenal aortic cross-clamping may cause myocardial ischemia in patients with severe coronary artery disease. This ischemia may be predicted by a rise in PCW at the time of cross-clamping, and vasodilator therapy is indicated in such patients.

INFRARENAL AORTIC CROSS-CLAMPING, required during surgical treatment of abdominal aortic aneurysm, is generally well tolerated. Occasionally, however, it is associated with severe hemodynamic disturbances. This communication compares the hemodynamic response to aortic cross-clamping during anesthesia and laparotomy in patients with and without severe coronary artery disease. The data indicate that myocardial ischemia is often produced in patients with severe coronary artery disease when the aorta is cross-clamped below the renal arteries, that this may be predicted by a rise in pulmonary capillary wedge pressure at the time of clamping, and that vasodilator therapy may prevent or ameliorate myocardial ischemia during aortic cross-clamping.

Materials and Methods

Fifteen male patients, ranging in age from 50 to 72 years, were studied. Five patients (group I) had no historical, physical, or laboratory evidence of heart disease. Ten patients (group II) had a history of severe ischemic heart disease, including previous myocardial infarction in seven, direct coronary artery surgery in three, angina pectoris in five, and congestive heart failure in eight. All had electrocardiographic evidence of myocardial ischemia.

All patients were premedicated with intramuscular morphine, 10 mg, and atropine, 0.5 mg. On arrival in the operating room, four ECG limb electrodes were attached, and appropriate cannulae were inserted for measurement of central venous pressure (CVP), arterial pressure (AP), pulmonary artery pressure (PA), and pulmonary capillary wedge pressure (PCW), an index of left atrial pressure. Pressures were recorded continuously on a Hewlett-Packard multichannel recorder. Esophageal temperature was measured using a thermistor. In group I only, cardiac output was measured by dye dilution with injection of green dye into the superior vena cava or right atrium and detection in radial artery blood.

Anesthesia was induced in all patients with 4 to 6 mg/kg thiopental. Endotracheal intubation was facilitated by 1 to 1.5 mg/kg intravenous succinylcholine, and anesthesia maintained with a minimum of 30% oxygen in nitrous oxide plus small (3 mg) increments of intravenous morphine.2 Muscular relaxation was provided by d-tubocurare or pancuronium following endotracheal intubation. Arterial blood gases and serum potassium were measured with standard techniques and corrected to the patient’s body temperature. PaO2 was above 100 mm Hg and serum potassium was maintained at normal levels throughout the procedure in all patients. Blood loss did not exceed 250 ml prior to aortic occlusion in any patient.

The study consisted of obtaining a standardized six-lead write-out of the ECG, all pressures, cardiac output (group I only), and arterial blood gases before and 1 to 3 min after aortic cross-clamping. In addition, evidence of myocardial ischemia, as estimated by ST-segment depression of the ECG and/or ventricular premature contractions, was continuously sought for on the oscilloscope tracing prior to and following aortic cross-clamping. If present, or at least once per hour if not, a standardized write-out of the ECG was obtained.

Cardiac index (CI) was derived by dividing cardiac output by body surface area, and stroke index (SI) by dividing CI by heart rate. Heart rate was derived from the ECG. Systemic vascular resistance was calculated by the formula

\[
SVR = \frac{AP - CVP}{CO} \times \frac{100 \text{ mm Hg}}{1 \text{ L per min}}
\]

where \( AP \) = arterial pressure, \( CVP \) = central venous pressure, \( CO \) = cardiac output.

The formula

\[
\text{AC} = \frac{t_2 - t_1}{R \ln P_1/P_2}
\]

(where \( P_1 \) = diastolic notch pressure, \( t_1 \) = time of opening, \( t_2 \) = time of closing) was used to estimate arteriolar resistance.

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Blood pressure, CVP, and PA/PCW before and after cross-clamping.

**Table 1. Hemodynamic Response to Infrarenal Aortic Cross-clamping in Patients without Clinical Heart Disease** (N = 6)

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>During (1-3 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RAP (mm Hg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>140.4</td>
<td>155.0</td>
</tr>
<tr>
<td>Diastolic</td>
<td>71.0</td>
<td>76.0</td>
</tr>
<tr>
<td>Mean</td>
<td>95.0</td>
<td>102.0</td>
</tr>
<tr>
<td><strong>PAP (mm Hg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>12.2</td>
<td>8.3</td>
</tr>
<tr>
<td>Diastolic</td>
<td>6.4</td>
<td>3.4</td>
</tr>
<tr>
<td>Mean</td>
<td>8.5</td>
<td>5.0</td>
</tr>
<tr>
<td><strong>CVP (mm Hg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>68.0</td>
<td>68.4</td>
</tr>
<tr>
<td>Diastolic</td>
<td>4.43</td>
<td>3.81</td>
</tr>
<tr>
<td>Mean</td>
<td>21.9</td>
<td>29.0</td>
</tr>
<tr>
<td><strong>SVR (units)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A Comp (ml/mm Hg)</td>
<td>1.42</td>
<td>1.09</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>2.66</td>
<td>2.23</td>
</tr>
<tr>
<td>SI (ml/beat/m²)</td>
<td>38.3</td>
<td>33.0</td>
</tr>
</tbody>
</table>

All values given are mean ± standard deviation.

*P < 0.05
†P < 0.001
‡P < 0.0001

**Abbreviations:** RAP = radial artery pressure; PAP = pulmonary artery pressure; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; HR = heart rate; CO = cardiac output; SVR = systemic vascular resistance; A Comp = arterial compliance; CI = cardiac index; SI = stroke index.

**Figure 1. Response to infrarenal aortic cross-clamping in one individual without heart disease.** Note the increase in arterial pressure, and the small declines in central venous, pulmonary artery, and pulmonary capillary wedge pressure. CVP = central venous pressure; PA = pulmonary artery pressure; PCW = pulmonary capillary wedge pressure.

**Results**

**Group I (table 1)**

Systolic and mean arterial pressure increased following aortic occlusion. Central venous pressure, pulmonary artery pressure, and pulmonary capillary wedge pressure decreased and heart rate was unchanged. Since cardiac and stroke indices decreased 14.0 ± 7.4 % and 19.0 ± 9.7 %, respectively, calculated systemic vascular resistance increased 33.3 ± 18.0 % and arterial compliance decreased 25.4 ± 7.0 %. One patient had a single PVC when the aorta was cross-clamped. No ECG evidence of myocardial ischemia was apparent in any time in any patient. A typical response to aortic cross-clamping in a patient without coronary artery disease is shown in figure 1.

**Group II (table 2)**

Radial artery pressure increased to the same degree following cross-clamping of the aorta in patients of group II as it had in patients of group I. In contrast to group I, however, central venous pressure, pulmonary artery pressure, and pulmonary capillary wedge pressure increased. The differences in response of all three values between the groups were significant (fig. 2).

Three of the ten patients with a history of severe coronary artery disease demonstrated signs consistent with myocardial ischemia during aortic cross-clamping: multiple unifocal premature ventricular contractions (PVC) in one patient, multifocal PVCs in one, and ST-segment depression in two. There was overlap in all variables between the seven patients with CAD who did not manifest evidence of ischemia and the three patients who did. Therefore, it is not possible to predict prior to cross-clamping those patients who will tolerate that intervention poorly. The three patients who developed ischemia, however, were those who demonstrated the greatest increases in PCW one to three minutes after the aorta was cross-clamped, and they are designated by asterisks in figure 2.

Figure 3 shows the hemodynamic response of one patient with coronary artery disease. Within ten seconds after aortic cross-clamping, both CVP and PCW increased without change in ECG. At three minutes, further elevation of PCW has occurred, although no ECG evidence of ischemia is present. Two hours post-application of the clamp, the radial artery and central venous pressures are unchanged as compared with values obtained shortly after cross-clamping but the PA and PCW are grossly elevated, and there is unequivocal evidence of myocardial ischemia on the ECG. Shortly thereafter, multifocal ventricular contractions appeared at a rate of 30/min.

**Treatment**

Therapy in each of the three cases where evidence of myocardial ischemia was noted was intravenous administration of a peripheral vasodilator, sodium nitroprusside (30 mg/250 ml 5% D/DW), by constant infusion pump. Infusion was initiated at 10 to 15 μg/min and raised at three minute intervals by 10 to 15 μg/min in order to decrease...
PCW toward normal (control) levels without producing systemic hypotension. The dose rate required ranged from 15 to 40 μg/min. In two cases this was associated with immediate relief of evidence of ischemia (fig. 4). In the third instance, in spite of reversal of the elevated PCW, the ST segments remained depressed and ventricular premature contractions persisted, though neither increased in severity. Intravenous lidocaine (bolus 100 mg, infusion 2 mg/min) caused cessation of ventricular irritability, and the ECG had reverted to the preoperative state by 12 hours postoperatively. No patient in either group had ECG changes of myocardial infarction postoperatively.

### Discussion

This study indicates that the tolerance of patients with severe coronary artery disease to the stress of infrarenal aortic cross-clamping differs from that of patients without overt coronary artery disease. Arterial blood pressure is elevated in both groups of patients. However, those with coronary artery disease sustain increases in ventricular filling pressures, while those without coronary artery disease sustain decreases. Furthermore, aortic cross-clamping in patients with severe coronary artery disease is frequently

| Table 2: Hemodynamic Response to Infrarenal Aortic Cross-clamping in Patients with Severe Coronary Artery Disease (N = 10) |
|-----------------|-----------------|-----------------|
|                 | Before          | During (1-3 min)|
| RAP (mm Hg)     |                 |                 |
| Systolic        | 139.4 ± 40.0    | 163.9 ± 40.0‡   |
| Diastolic       | 73.3 ± 10.3     | 75.0 ± 12.5     |
| Mean            | 95.7 ± 21.1     | 106.0 ± 19.1†   |
| PAP (mm Hg)     | 11.9 ± 5.6      | 14.7 ± 8.3*     |
| CVP (mm Hg)     | 4.9 ± 4.1       | 6.6 ± 5.3       |
| PCWP (mm Hg)    | 7.0 ± 5.2       | 10.5 ± 8.0†     |
| HR (mm Hg)      | 78.8 ± 11.0     | 75.8 ± 12.3     |

All values given are means ± SD.  
*P < 0.05.  
†P < 0.01.  
‡P < 0.001.  
See table 1 for abbreviations.

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**Figure 2.** Comparison of changes in central venous, pulmonary artery, and pulmonary capillary wedge pressure in patients with and without coronary artery disease. The values with asterisks are those of patients who developed evidence of myocardial ischemia during infrarenal aortic cross-clamping. The significance values refer to the comparison of group I to group II.

**Figure 3.** Response to infrarenal aortic cross-clamping of one individual with severe coronary artery disease. Note the rapid simultaneous increase in arterial, right atrial, and pulmonary capillary wedge pressure beginning immediately after cross-clamping. Two hours later, mean PCW is 30 mm Hg, and ST-segment depression indicates myocardial ischemia. RAP = right atrial pressure. For other abbreviations see figure 1 legend.
associated with myocardial ischemia, as evidenced by ST-segment depression of limb leads and ventricular irritability. No variable measured prior to cross-clamping predicted an ischemic response. Our data suggest, however, that an acute increase in excess of 5 mm Hg in CVP or 7 mm Hg in PCW within three minutes following aortic cross-clamping is likely to be associated with subsequent myocardial ischemia during the period of aortic occlusion and that vasodilator therapy may be an effective way to reverse or prevent progression of myocardial ischemia.

Only limb ECG leads were examined for evidence of ST-segment changes in this study. This is consistent with standard intraoperative anesthetic practice. It is possible that precordial electrocardiography, a more sensitive index of myocardial ischemia, would have detected changes not obvious in the limb leads. Therefore, our data may estimate a lower incidence of myocardial ischemia than actually occurs.

Infrarenal aortic cross-clamping was associated with an average increase of 33% in systemic vascular resistance and a mean decrease of 25% in systemic artery compliance in the patients measured (i.e., those without coronary artery disease). Since we did not measure cardiac output in group II, we cannot quantify changes in resistance or compliance in patients with severe coronary disease. It seems reasonable, however, to assume that a similar occlusion of the arterial tree would cause a similar direction, if not magnitude, of change in resistance and compliance.

A decrease in venous return and cardiac output following inframesenteric occlusion of the aorta has recently been described in dogs. This change was in contrast to increases observed with supramesenteric occlusion. The authors postulated that these changes were due to coexistence of parallel slow (mesenteric) and fast (extremity) time-constant venous drainage areas in the systemic circulation. The mechanism whereby infrarenal aortic cross-clamping causes a decrease in venous return is by exclusion of a portion of the fast time-constant circulation. Their postulates are consistent with the decreases in cardiac output and central venous pressure we observed in patients without coronary artery disease. The increase in left ventricular filling pressure (PCW) observed in group II probably represents a degree of left ventricular decompensation in response to a similar increase in impedance to left ventricular ejection caused by aortic occlusion, despite a decrease in venous return similar to the patients of group I.

Increased left ventricular size, as reflected by elevation of PCW, is known to be associated with increased ventricular wall stress, a cause of increased myocardial oxygen demand. In addition, increased ventricular diastolic pressure is associated with elevated intramyocardial pressure, predisposing to subendocardial ischemia. Whether the increases of PCW, which preceded ECG evidence of ischemia in all three patients, constitute a contributing cause to myocardial ischemia or reflect early ischemia, however, cannot be determined by our data. It also is not possible to rule out a change in left ventricular stiffness as contributing to the increases in PCW observed.

The association between ventricular irritability and myocardial ischemia is an important one, not limited to the situation of aortic cross-clamping. The primary treatment of irritability due to myocardial ischemia should include relief of ischemia, in addition to "symptomatic" treatment such as administration of lidocaine.

The use of vasodilators to treat an acute increase in afterload in our patients is similar to their use for "afterload reduction" in patients with pump failure following myocardial infarction. In such patients, when the left atrial pressure has become elevated, vasodilator therapy has resulted in a decrease in left ventricular filling pressure and systemic vascular resistance and an increase in cardiac output with a modest or no decrease in systemic arterial pressure. In contrast, decreases in cardiac output are common when vasodilators are administered in the absence of an elevated left ventricular filling pressure. It has therefore been suggested that a measure of left heart filling pressure is a prerequisite for decisions to use these drugs. In addition, our study reveals a lack of concordance between CVP and PCW as exemplified by the last panel in figure 3 in which a 15 mm Hg increase in PCW two hours after cross-clamping occurred without change in CVP. Both reasons convince us that left ventricular filling pressure should be monitored in patients with severe CAD undergoing abdominal aortic surgery.
ROLE OF CPK-MB IN DX OF AMI/Roark, Wagner, Izlar, Roe

References

Diagnosis of Acute Myocardial Infarction
in a Community Hospital

Significance of CPK-MB Determination

STEVEN F. ROARK, GALEN S. WAGNER, M.D.,
H. L. IZLAR, JR., M.D., AND CHARLES R. ROE, M.D.

SUMMARY Twice-daily CPK-MB determinations were performed but not made available to the physicians of 179 consecutive patients with precordial pain admitted to a community hospital to evaluate the diagnostic importance of this isoenzyme. Physician decision was based upon history and once-daily ECG and total enzymes (CPK, SGOT, LDH). Following hospital discharge, each patient’s clinical record was reviewed to determine the physician diagnostic decision. The patients were subdivided into three groups. The first group consisted of 46 patients with diagnostic QRS changes and elevated total enzymes. All 46 had physician diagnosis of acute myocardial infarction and CPK-MB was present in 44 (96%). The second group included 55 patients with nondiagnostic QRS but elevated total enzymes. Physician diagnosis was acute myocardial infarction in 28 (51%) but 16 (57%) of these had no CPK-MB. The third group contained 50 patients with nondiagnostic QRS and normal enzyme levels. Six (12%) had physician diagnosis of acute myocardial infarction but none had CPK-MB. Thus, absence of CPK-MB failed to confirm physician diagnosis of acute myocardial infarction when based upon history and total enzymes in the absence of QRS changes in 22 of 34 (65%) patients.

Since 1960 there has been a trend toward identification of a special place within the hospital for care of patients with acute myocardial infarction. These coronary care units differ from other areas by the presence of continuous ECG monitoring, maintenance of intravenous lines, maintenance of standby emergency equipment including defibrillators and pacemakers and, most importantly, by the presence of specially trained nurses.1 The difficulty of diagnosis of acute myocardial infarction from the clinical history was appreciated early.2 Subsequently, the coronary care unit has been used for management of patients with symptoms suggesting acute coronary insufficiency until either the diagnosis of acute infarction was ruled out or until the patient attained a stable condition after the diagnosis was confirmed. The 12-lead electrocardiogram has proven specific but insensitive, and total serum enzymes sensitive but nonspecific for diagnosis of acute infarction.3 Thus, when no QRS changes occur and total enzyme levels are elevated, the diagnosis is uncertain. This may result in poor utilization of coronary care beds and potentially erroneous diagnosis and treatment.

Therefore, efforts were made to identify organ specific subgroups of the total enzymes (isoenzymes).4-6 The specificity and sensitivity of these have been determined within the medical center,4 but the great majority of patients with symptoms of acute coronary insufficiency are managed in the community hospital. It is the purpose of this study to determine the incidence in which the isoenzymes of creatine phosphokinase provide information capable of altering the diagnosis and thus, potentially, the care of these patients.

Methods

All 179 patients admitted to the Watts Hospital Medical Intensive Care Unit during a six-month period with a presumptive diagnosis of acute coronary insufficiency were included in this study. Twelve-lead ECGs and serum samples were obtained at the time of admission and once daily for at least three days. All serum samples were analyzed for determination of total SGOT6 (upper limit 40), LDH6 (upper limit 225) and CPK6 (upper limit 145).
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