Abnormal Regional Metabolism and Mechanical Function in Patients with Ischemic Heart Disease

Improvement after Successful Regional Revascularization by Aortocoronary Bypass

By Kanu Chatterjee, M.D., M.R.C.P., Jack M. Matloff, M.D., H.J.C. Swan, M.D., Ph.D., F.R.C.P., William Ganz, M.D., C. Sc., V. S. Kaushik, M.D., Peter Magnusson, M.D., Marc M. Henis, M.D., and James S. Forrester, M.D.

SUMMARY

Left ventricular anterior wall metabolism was investigated concurrently with global myocardial metabolism by simultaneous preoperative sampling of anterior interventricular venous (AIV) and coronary sinus (CS) as well as arterial bloods in seven patients with severe obstructive lesions of the major coronary arteries, including left anterior descending. Postoperative study was performed two weeks to six months following successful aortocoronary artery bypass surgery. All grafts including the aorto-left anterior descending artery grafts were patent. Preoperatively in three of the seven patients, anterior wall lactate extraction (R%L) was negative at rest. The average R%L at rest (7 ± 14%) was abnormal and was negative (−49 ± 26%) at a maximum supraventricular pacing rate (MPR) of 137 ± 4.6 beats/min. Postoperatively, not only was resting R%L (39 ± 4.4%) normal but also it remained normal during atrial pacing (32 ± 8.5%) even though the postoperative MPR (164 ± 4.4 beats/min) was much higher than the preoperative MPR. Postoperatively AIV pO2 both at rest (21 ± 1.1 mm Hg) and at MPR (22 ± 1.3 mm Hg) and directly determined O2 saturations (resting: 34 ± 3.0%; MPR: 35 ± 2.1%) tended to be higher than the preoperative values (AIV pO2, resting: 18 ± 1.7; MPR: 19 ± 1.7 mm Hg; AIV O2 saturation resting: 30 ± 2.7; MPR: 33 ± 3.3%), although only differences in pO2 were statistically significant. In five of the seven patients in whom the pre and postoperative left ventricular angiograms could be compared, systolic wall motion of the left ventricular anterior wall improved markedly postoperatively.

Average global myocardial lactate extraction (G%L) preoperatively was normal (19 ± 4.8%) at rest but was negative (−22 ± 12%) at MPR. Postoperatively however, G%L both at rest (44 ± 5.5%) and at MPR (34 ± 7.9%) were normal. Coronary sinus pO2 and O2 saturation were also higher postoperatively compared to the preoperative values. Over-all left ventricular performance indicated by increase in ejection fraction also improved postoperatively. This improvement was not caused by increased coronary blood flow. Postoperative coronary sinus blood flow both at rest (114 ± 19 ml/min) and at MPR (199 ± 27 ml/min) however were less than the preoperative values (resting 136 ± 24, MPR 261 ± 40 ml/min). There was also no increase in global O2 delivery and O2 consumption despite higher heart rate and rate-pressure product achieved during postoperative pacing stress and the patients did not develop angina. These findings suggest that improved regional and global metabolism and mechanical functions observed postoperatively in these patients may be due to redistribution of blood flow to the ischemic and nonischemic myocardium following successful aortocoronary artery bypass surgery.

The metabolic derangement and mechanical dysfunction of the myocardium which results from significant obstructive coronary artery disease is, as a rule, regional and nonuniform in distribution. When regional dysfunction is severe, overt depression of over-all cardiac function with recognizable clinical manifestations occurs. If regional dysfunction is less extensive, the clinical, mechanical and metabolic abnormalities may be manifest only during stress. The objective of aortocoronary artery bypass is to restore adequate blood supply to ischemic areas of myocardium and thus to relieve the effects of inadequate coronary blood flow and segmental myocardial hypoxia at rest and under conditions of increased demand. Reports of symptomatic relief of angina, improved exercise tolerance, improvement in over-all myocardial metabolism, and mechanical performance following successful aortocoronary artery bypass suggest that this objective is achieved with surgery.

Since the coronary artery disease and the accom-
panying myocardial dysfunction are, in most instances, regional, the present study was designed to analyze the effects of revascularization on changes in regional myocardial function and metabolism. More specifically, this study reports the effects of direct revascularization on metabolism and mechanical function at rest and during atrial pacing stress in patients with prior metabolic derangement involving the anterior wall due to severe proximal disease of the left anterior descending coronary artery. Changes in regional metabolism are also correlated with regional mechanical function. The results indicate that successful regional revascularization favorably affects regional metabolism at rest or during stress in these patients. Furthermore, improvement in markers of metabolism is also accompanied by improvement in mechanical function. The anterior cardiac wall is particularly suited to study since the anterior interventricular vein drains most of the left ventricular anterior wall and systolic wall motion of the anterior wall is most easily defined by routine angiographic techniques.

Methods

Anterior Interventricular Vein Catheterization (fig. 1)

The anterior interventricular vein (AIV) drains the areas of the left ventricular (LV) muscle supplied by the left anterior descending coronary artery, i.e., most of the LV anterior wall and apex. Therefore, sampling from the proximal portion of AIV will represent sampling of effluent blood from most of the LV anterior wall and apex. Selective cannulation of the anterior inteventricular vein (AIV) which parallels the left anterior descending coronary artery was performed under fluoroscopic control. A preshaped woven-Dacron thin-walled Lehmann catheter (7F) was first placed in the coronary sinus (CS) and advanced to the junction of the AIV and the marginal artery. A J-tipped guide wire was then manipulated through the Lehmann catheter to the mouth of the AIV. The Lehmann catheter was then advanced over the guide wire in the AIV and the guide wire removed. A soft flexible radiopaque vinyl tubing (outer diameter = 0.046", inner diameter = 0.028") was then advanced through the Lehmann catheter, manipulated down the anterior interventricular vein, and the Lehmann catheter withdrawn over it.

Coronary Sinus Catheterization

A special preshaped 7F venous catheter, modified to incorporate two pacing electrodes, was placed in the coronary sinus. The coronary sinus catheter was advanced so that the external thermistor (identified by the proximal electrode-band) would lie just inside the mouth of the coronary sinus. The position of the catheter was determined by injecting contrast medium upstream in the coronary sinus in all patients and stability confirmed by the consistent recording of a stable temperature, 0.1 to 0.15°C above the fluctuant temperature characteristic of the right atrium. Coronary sinus blood flow (CSBF) was measured by the continuous infusion thermodilution technique of Ganz and expressed as ml/min. Supraventricular bipolar pacing was performed from the CS using the same catheter.

Other Measurements

Arterial pressure was monitored through a 20 gauge cannula inserted into the radial artery. Blood samples were analyzed for pH, pCO₂ and pO₂ by a pH gas analyzer Model 113 (Instrumentation Laboratories). Hemoglobin saturation was determined with a Co-oximeter, Model 182 (Instrumentation Laboratories). Samples of arterial (Art), AIV, and CS blood in duplicate were taken for determination of lactate concentration by an automated modification of the enzymatic method of Hohorst. The mean difference in lactate concentrations between paired samples was less than 5%.

Procedure

Metabolic Study

All patients were in normal sinus rhythm during both pre- and postoperative studies. Measurements of arterial pressure, coronary sinus blood flow, and the simultaneous withdrawal of blood samples from the radial artery, coronary sinus, and anterior interventricular vein were done in a stable control period at the patient’s unpaced sinus rate. The heart rate was then increasing elevated in increments of 10 beats/min at 90 second intervals by electrical pacing from the coronary sinus until the patient developed angina or maximum pacing rate (MPR) (≥ 150 beats/min) was attained. Measurements and blood samples were obtained when angina appeared and the pacing discontinued. In some patients administration of intravenous atropine, 0.4 to 0.8 mg, was necessary to permit pacing with 1:1 atrioventricular conduction at high rates. For determination of heart rate and detection of changes in ST-segment, lead V₅ of the electrocardiogram was monitored. Derived metabolic parameters were calculated as follows:

Figure 1

Chest X-ray (right anterior oblique projection) showing the positions of vinyl tubing in the anterior interventricular vein (arrow pointing right) and of the coronary sinus catheter in the mouth of the coronary sinus (arrow pointing left) for simultaneous sampling of blood from the anterior interventricular vein and the coronary sinus.

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1) Oxygen content (ml/100 ml) = hemoglobin saturation \( \% \times \) hemoglobin content (g/100 ml) \( \times 1.34 \)

2) "Global" oxygen delivery (ml/min) = CSBF (ml/min) \( \times \) art. O2 cont. (ml/100 ml)

3) "Global" myocardial O2 extraction (ml/100 ml) = art. \( - \) CS O2 cont. (ml/100 ml) \( \times 10^2 \)

4) "Regional" myocardial O2 extraction (ml/100 ml) = art. \( - \) AIV O2 content (ml/100 ml)

5) "Global" myocardial O2 consumption (MVO2) = (Art. - CSO2) \( \times \) CSBF (ml/min) \( \times 10^2 \)

6) "Global" myocardial lactate extraction (G%L) = Art. - CS lactate (mg) \( \times 100 \)

7) "Regional" myocardial lactate extraction (R%L = Art. - AIV lactate (mg) \( \times 100 \)

8) Rate-pressure product (RP) = heart rate (beats/min) \( \times \) peak systolic pressure (mm Hg) \( \times 10^3 \) (mm Hg/min)

Lactate extraction less than 10% or lactate production (negative myocardial lactate balance) was considered consistent with myocardial ischemia.21

Angiographic Study

Within one to three hours of the above study, left heart catheterization, left ventriculography, and selective coronary arteriography, and, during postoperative study, selective graft angiography, were performed by Seldinger technique. Left ventricular cineangiography was always performed prior to coronary arteriography. Left ventricular (LV) pressures were recorded through a fluid-filled loop-end catheter with a zero reference level 5 cm posterior to the sternal angle. The same catheter manometer systems were used for both pre- and postoperative studies.

For calculation of segmental wall motion and LV volumes, ectopic and postectopic cycles were eliminated. A quantitative assessment of abnormalities of LV systolic wall motion (SWM) was determined from single plane cineangiograms obtained with the patient in right anterior oblique (RAO) projection. Thirty to forty cc of 75% Hypaque were injected into the left ventricle and a filming speed of 60 frames/second was used. Left ventricular silhouettes from both pre- and postoperative cineangiograms, in all patients, were outlined by the same investigator, to minimize the observer's error in calculations of left ventricular volumes.22

Basically, systolic wall motions of the four quadrants (anterobasal, anteropapical, inferobasal, inferopapical) of the left ventricle was analyzed. Initially the end-diastolic and end-systolic outlines of the left ventricle were divided into eight anterior (designated A1-A8) and eight inferior (designated B1-B8) segments (fig. 2) which were analyzed by a Sigma 3 digital computer and displayed on an automated X-Y plotter.23 The long axis (midpoint of the aortic root to the apex) was divided by a perpendicular drawn at its midpoint and then by six additional perpendiculars, three on each side of the midpoint. The areas of each of the resulting 16 segments were then calculated from both the end-diastolic and end-systolic frames. The change in each segmental area at end-systole was expressed as the ratio \( \frac{A S - A D}{AD} \times 100 \) from the end-systolic frames, where \( A_S \) and \( A_D \) represent end-diastolic and end-systolic areas, respectively. Changes in the areas of \( A_1 - A_8 \) segments were averaged to represent anterior basal motion (ABM), in \( A_3 - A_4 \) as anteropapical (AAM), \( B_1 - B_4 \) as inferobasal (IBM), and \( B_5 - B_8 \) as representative of posterobasal motion (PBM). The left ventricular volumes were calculated from the same cineangiograms (single plane RAO) assuming the ventricle to approximate an ellipsoid of revolution. The long axis was determined directly and the minor axis was determined by the area length method of Dodge.24 The magnification factor was determined from the ratio of the projected circumference of a metal ball placed at the level of LV to its actual circumference. Angiographically determined LV volumes were corrected for body surface area. Calculated LV volumes from single plane cineangiograms in the RAO projection overestimate the actual volumes.25,26 and correction has been made by the regression equation of Herman et al.27 Since the postoperative values are compared with preoperative values for each patient, our conclusions are not dependent on the validity of this correction.

Hemodynamic parameters were calculated as follows:

1) Left ventricular stroke volume (LVSV(ml/m²) = end-diastolic volume (EDV) (ml/m²) - end-systolic volume (ESV) (ml/m²)

2) Ejection fraction (EF) = LVSV (ml/m²)/EDV (ml/m²)

3) Developed pressure (DP) (mm Hg) = aortic diastolic pressure (mm Hg) - left ventricular end-diastolic pressure (mm Hg)

Patient Population

All seven patients studied were males and their ages ranged from 40 to 58 years. The indications for surgery were

**END SYSTOLIC**

![End Systolic Calibration](frame_87.png)

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**END DIASTOLIC**

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<td>1.21</td>
<td>1.37</td>
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<td>0.55</td>
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**CALIBRATION FACTOR = 1.442**

BSA = 1.534 m²

Figure 2

Determination of left ventricular systolic wall motion with the use of digital computer and x-y plotter. Both end-diastolic and end-systolic outlines of the left ventricle are divided into the eight anterior (designated A1-A8) and eight inferior (designated B1-B8) segments and the area of each segment is then calculated. The % change in each segmental area is then calculated from end-diastole to end-systole. Changes in the areas A1-A8 segments were averaged to represent anterior basal motion (ABM), in A3-A4 as anteropapical (AAM), B1-B4 as inferobasal and B5-B8 as representative of posterobasal (PBM) motion.
chronic disabling angina in two patients, accelerated angina in four patients, and spontaneous recurrent angina at rest following recent anteroseptal myocardial infarction (12 days) in one patient. In the patient with recent infarction, heart rate was not increased by atrial pacing during preoperative metabolic study. In addition to the patient with recent infarction, two other patients had historical and electrocardiographic evidence of old myocardial infarction, preoperatively. Preoperative selective coronary arteriography demonstrated significant obstructive lesions (≥75%) involving left anterior descending (LAD), left circumflex (L CF), and right coronary artery (RCA) systems in six patients and LAD and LCF systems in the remaining patient.

Two patients had quadruple, three triple, and three double grafts and all grafts were patent at the time of the postoperative study. Three patients were studied within two weeks and the remaining four between five and 24 weeks following surgery.

Results

Pre- and postoperative clinical data are summarized in table 1. All patients were free of angina at rest and had no clinical evidence of heart failure at the time of restudy. Angina threshold determined during atrial pacing stress markedly improved postoperatively. All six patients paced preoperatively developed typical angina, at an average heart rate of 137 ± 4.6 beats/min (range 121–150 beats). Postoperatively, in all patients heart rates could be increased to a much higher level (164 ± 4.4 beats/min) and none developed angina (fig. 3). Resting heart rate postoperatively (90 ± 4.9 beats/min) was significantly higher than the preoperative resting heart rate (73 ± 5.6 beats/min) (P < 0.05). Rate-pressure product at angina (21 ± 1.3 mm Hg/min × 10³) preoperatively was also significantly lower than RP at maximum pacing rate (23 ± 1.8 mm Hg/min × 10³).

![Figure 3](image)

**Figure 3**

Pre- and postoperative maximum pacing rates (a) and rate pressure products at maximum pacing rate (b). Preoperatively all patients had angina at MPR. Postoperatively all patients were free of angina. Abbreviations: PSF = peak systolic pressure, HR = heart rate, MPR = maximum pacing rate.

| Patient | Age/sex | Inherent RP | Preop | Postop | Preop Inherent | Postop Inherent | Preop | Postop | Preop | Postop | Preop | Postop | Preop | Postop | Preop | Postop | Preop | Postop | Preop | Postop | Preop | Postop |
|---------|---------|-------------|-------|--------|---------------|----------------|-------|--------|-------|--------|-------|--------|-------|--------|-------|--------|-------|--------|-------|--------|-------|--------|-------|--------|
| DB      | 45 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |
| JS      | 58 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |
| RM      | 48 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |
| AM      | 53 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |
| JB      | 51 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |
| KB      | 53 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |
| RC      | 40 M    | Inf         | 0     | 0      | 0             | 0              | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      | 0     | 0      |

Abbreviations: Inf = STsegment depression; LAD = left anterior descending coronary artery; RCA = right coronary artery; LCF = left circumflex coronary artery.

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<table>
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<th>Preop R</th>
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<th>P</th>
<th>Preop MPR</th>
<th>Postop MPR</th>
<th>P</th>
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<td>R %L</td>
<td>+7 ± 1.4</td>
<td>+39 ± 4.4</td>
<td>NS</td>
<td>-49 ± 26</td>
<td>+32 ± 8.5</td>
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</tr>
<tr>
<td>G %L</td>
<td>+19 ± 4.8</td>
<td>+44 ± 5.3</td>
<td>0.05</td>
<td>-22 ± 12</td>
<td>+34 ± 7.9</td>
<td>0.05</td>
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<tr>
<td>AIV pO₂ (mm Hg)</td>
<td>18 ± 1.7</td>
<td>21 ± 1.1</td>
<td>0.05</td>
<td>19 ± 1.7</td>
<td>22 ± 1.3</td>
<td>0.05</td>
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<tr>
<td>AIV O₂ Sat (%)</td>
<td>30 ± 2.7</td>
<td>34 ± 3.0</td>
<td>NS</td>
<td>33 ± 3.0</td>
<td>35 ± 2.1</td>
<td>NS</td>
</tr>
<tr>
<td>CS pO₂ (mm Hg)</td>
<td>19 ± 1.3</td>
<td>22 ± 1.7</td>
<td>0.05</td>
<td>19 ± 1.8</td>
<td>21 ± 0.7</td>
<td>NS</td>
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<tr>
<td>CS O₂ Sat (%)</td>
<td>32 ± 1.8</td>
<td>36 ± 3.4</td>
<td>NS</td>
<td>33 ± 3.0</td>
<td>35 ± 1.4</td>
<td>NS</td>
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<tr>
<td>Hb (g %)</td>
<td>14.5 ± 1.4</td>
<td>11.7 ± 0.86</td>
<td>0.005</td>
<td>14.5 ± 1.2</td>
<td>11.7 ± 0.86</td>
<td>0.005</td>
</tr>
<tr>
<td>AIVT-AIV O₂ Content (ml %)</td>
<td>13.0 ± 0.028</td>
<td>9.8 ± 0.74</td>
<td>0.005</td>
<td>12.1 ± 0.31</td>
<td>10.1 ± 0.30</td>
<td>0.05</td>
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<tr>
<td>AIVT-CS O₂ Content (ml %)</td>
<td>12.7 ± 0.34</td>
<td>8.8 ± 0.67</td>
<td>0.001</td>
<td>12.1 ± 0.30</td>
<td>9.4 ± 0.87</td>
<td>0.05</td>
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<td>CSBF (ml/min)</td>
<td>136 ± 24</td>
<td>114 ± 19</td>
<td>NS</td>
<td>261 ± 40</td>
<td>199 ± 72</td>
<td>0.01</td>
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<tr>
<td>Global O₂D (ml/min)</td>
<td>25.0 ± 4.4</td>
<td>16.2 ± 2.7</td>
<td>0.005</td>
<td>49.5 ± 8.1</td>
<td>28.8 ± 4.5</td>
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<tr>
<td>MVO₂ (ml/min)</td>
<td>17.3 ± 2.7</td>
<td>10.4 ± 1.5</td>
<td>0.005</td>
<td>31.8 ± 4.7</td>
<td>19.2 ± 2.2</td>
<td>0.005</td>
</tr>
</tbody>
</table>

Abbreviations: R = at rest; CS = coronary sinus; MPR = at maximum pacing rate; Hb = hemoglobin; R %L = anterior wall lactate extraction; ART = arterial; G %L = global lactate extraction; O₂D = oxygen delivery; AIV = anterior interventricular vein; CSBF = coronary sinus blood flow; MVO₂ = myocardial oxygen consumption.

(P < 0.005) postoperatively (fig. 3). Rate-pressure product at rest postoperatively (18 ± 1.4 mm Hg/min × 10⁻⁶) was higher than the preoperative value (11.0 ± 1.2 mm Hg/min × 10⁻⁶), but the difference was not statistically significant.

Regional and Global Lactate Balance (table 2)

Preoperatively, three of seven patients had negative anterior wall lactate extraction (R%L) at rest although none of these patients complained of angina at rest. All six patients who were paced preoperatively had abnormal R%L at angina rate. Postoperatively R%L rate at rest was normal in all patients and remained normal in most patients, even when the heart rate was increased to a much higher level (fig. 4). Only in one patient did the R%L fall below 10% postoperatively; this occurred at a MPR which was much higher than the patient's preoperative angina rate (fig. 4).

Changes in global myocardial lactate extraction (G%L) (fig. 5) paralleled changes in R%L. Resting G%L was abnormal in one patient, and it became abnormal in the six patients paced preoperatively. Postoperatively, G%L was normal in all patients at rest and it remained normal in six of the seven patients even when their heart rates were increased to a much higher level compared to their preoperative angina rate.

Venous and Arterial Oxygen

Both pO₂ and directly determined O₂ saturation in AIV blood tended to be higher postoperatively both at rest and at maximum pacing rate compared to the preoperative values, although the differences were statistically significant only between pO₂ values (table 2). Similarly pO₂ and O₂ saturation in CS blood
postoperatively suggest improved coronary reserve and myocardial efficiency.

Due to increased arterial O₂ content, global O₂ delivery was less postoperatively both at rest and at maximum pacing rate compared to the preoperative O₂ delivery at rest and at angina (fig. 6).

Changes in Left Ventricular Volumes, Ejection Fraction, and Segmental Wall Motion (table 3)

Preoperative and postoperative systolic wall motion, LV volumes, and ejection fraction were compared in five of the seven patients. In the remaining two patients (numbers 3 and 7, table 1) comparisons could not be made due to inadequate left ventriculograms, either pre- or postoperative. Resting heart rates and arterial pressure during metabolic and angiographic studies before surgery were similar. Postoperatively there was no significant difference in resting heart rate and arterial pressures during metabolic and angiographic studies. Along with improved regional and global metabolism following surgery, systolic wall motion of most of the left ventricular wall segments, particularly of the anterior quadrants, improved in the majority of patients. Thus in five patients, in whom changes in systolic wall motion were determined, anterior basal, anteropapical, and inferobasal motion was improved.

**Table 3**

<table>
<thead>
<tr>
<th></th>
<th>Preop</th>
<th>Postop</th>
<th>P</th>
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<tbody>
<tr>
<td>Anterobasal syst. motion (%)</td>
<td>38.2 ± 2.5</td>
<td>56.0 ± 3.9</td>
<td>0.05</td>
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<tr>
<td>Anteropapical syst. motion (%)</td>
<td>30.2 ± 4.0</td>
<td>46.6 ± 5.4</td>
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<tr>
<td>Inferobasal syst. motion (%)</td>
<td>24.4 ± 4.0</td>
<td>40.8 ± 5.0</td>
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<td>Inferopapical syst. motion (%)</td>
<td>33.4 ± 6.8</td>
<td>51.6 ± 8.7</td>
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<td>Heart rate (beats/min)</td>
<td>72.9 ± 5.6</td>
<td>89 ± 4.9</td>
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<td>LV end-diastolic pressure (mm Hg)</td>
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<td>9.2 ± 0.9</td>
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<td>Art. syst. pressure (mm Hg)</td>
<td>135 ± 7.6</td>
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<td>Art. diastolic pressure (mm Hg)</td>
<td>83 ± 2.1</td>
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<td>Mean art. pressure (mm Hg)</td>
<td>101 ± 4.1</td>
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<td>Developed pressure (mm Hg)</td>
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<td>End-diastolic volume (mL/m²)</td>
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</table>

Abbreviations: LV = left ventricle; Art. = arterial.
Of particular importance, in the three patients with anterior wall lactate production at rest preoperatively marked anterior wall hypokinesis was also present. In all three patients normalization of anterior wall motion (figs. 7 and 8) occurred following successful aorto-LAD bypass surgery. End-diastolic volume did not change significantly postoperatively. End-systolic volume decreased, and therefore, calculated LVSV and EF improved postoperatively. There was no significant change in developed pressure postoperatively.

Discussion

Regional rather than global myocardial dysfunction is the usual consequence of segmental myocardial hypoxia that results from significant obstructive coronary artery lesions. Thus to adequately assess the functional significance of a given degree of coronary artery disease and the effectiveness of any therapy, it is necessary to evaluate changes in specific regions. This study demonstrates that successful revascularization by aortocoronary artery bypass surgery can relieve regional metabolic derangement and mechanical dysfunction. In all seven patients abnormal anterior wall lactate metabolism, present preoperatively either at rest or during atrial pacing stress, markedly improved following successful aorto-LAD bypass surgery. This improvement in anterior wall lactate metabolism was apparent not only at rest but also even when the heart rate was increased beyond rates that produced angina preoperatively. Normalization of anterior wall lactate metabolism was usually associated with decreased anterior wall myocardial oxygen extraction (Art – AIV O₂). The decreased ART – AIV O₂ was due mainly to reduced arterial oxygen content due to decreased hemoglobin concentration postoperatively. However, pO₂ and O₂ saturation in AIV blood were both usually higher postoperatively. The tendency to higher oxygen level in AIV blood in the face of reduced arterial oxygen content suggests improved and perhaps more uniform myocardial oxygen extraction following regional revascularization.

The present study indicates improvement in left ventricular segmental wall motion associated with improved regional metabolism. The anterior wall lactate production that was noted at rest in three patients preoperatively was accompanied by serious abnormalities of systolic LV anterior wall motion. Postoperatively, systolic wall motion improved with the normalization of anterior wall lactate metabolism. There was also concurrent improvement in global (coronary sinus) myocardial lactate metabolism. Abnormal global lactate extraction preoperatively became normal postoperatively in all but one patient. Art – CS O₂ content difference, i.e., global myocardial oxygen extraction, was lower postoperatively, due to both decreased arterial and usually increased CS O₂ contents. Myocardial oxygen extraction was significantly lower postoperatively due not only to decreased myocardial O₂ extraction but also due to decreased CSBF both at rest and at MPR.

Over-all myocardial function improved postoperatively. Heart rates could be increased to a much higher level in these patients and rate-pressure product was higher postoperatively. Yet myocardial O₂ consumption was less and effects of myocardial hypoxia, namely angina and abnormalities of lactate metabolism, were absent. All of these measures point to improved over-all myocardial metabolism, coronary reserve, and myocardial efficiency postoperatively. Improved myocardial efficiency was also reflected in the improvements in segmental wall motion and over-all mechanical function. In all five patients in whom

Figure 7
Pre- and postoperative left ventricular cineangiograms (RAO) in the patient with recent anteroseptal myocardial infarction. The left-hand panel shows the preoperative end-diastolic and end-systolic frames demonstrating obvious hypokinesis of the anterior wall. This was associated with negative anterior wall lactate extraction at rest. Postoperatively (right hand panel) marked improvement occurred both in anterior wall systolic wall motion and anterior wall lactate metabolism.

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Figure 8

Postoperative changes in anterobasal (a), anterogapical (b), and ejection fraction (c). In all three patients with negative anterior wall lactate extraction (●) anterior wall motion improved significantly postoperatively. Ejection fraction also improved concurrently.

pre- and postoperative left ventricular volumes and segmental wall motions were compared, ejection fraction and systolic wall motions improved.

The mechanism of improvement in angina threshold, regional and global metabolism, and mechanical function following revascularization as demonstrated in this study does not seem to be related to development of perioperative myocardial infarction. Loss of muscle mass in LV anterior wall due to perioperative anterior wall myocardial infarction will tend to cause decreased anterior wall myocardial O₂ extraction and increased lactate extraction. Therefore an apparent improvement in regional metabolism may occur postoperatively. However, none of the patients in this study whose anterior wall lactate metabolism improved had electrocardiographic evidence of new perioperative anterior wall myocardial infarction. Improved anterior wall systolic motion postoperatively further confirm improved function of previously ischemic myocardium and absence of fresh anterior wall myocardial infarction postoperatively. Therefore, improvement in anterior wall metabolism in these patients cannot be attributed to loss of anterior wall myocardial cells.

Spontaneous improvement in metabolism due to changes in myocardial O₂ demand unrelated to revascularization could explain improvement. However, this would not explain the finding that in most patients improved O₂ demand accompanied higher resting and maximal paced heart rates. The paced rates achieved were far faster than those that produced angina preoperatively.

Changes in contractile state, another determinant of myocardial O₂ demand, were not directly determined in these patients during pacing. It is not likely however that such changes decreased O₂ demand. Rather, at very high pacing rates obtained postoperatively, there may have been augmented contractility associated with tachycardia (Bowditch effect) and increase in myocardial O₂ demand. Changes in left ventricular volumes and pressures were not determined during atrial pacing either pre- or postoperatively. There was a tendency to decreased resting LVEDV, EDP and DP postoperatively, although differences were not statistically significant. This might have contributed to some extent to decreased CSBF and MVO₂ postoperatively at rest. However, it is unlikely that the changes in EDV, EDP, and developed pressure, which were only minimal, caused substantial enough changes in resting wall tensions to explain the normalization of negative anterior wall lactate extractions both at rest and during atrial pacing stress.

Although the present study supports the previous report that regional revascularization by aorto-coronary artery bypass surgery improves regional metabolism, such improvement seems to be related to changes other than improved global coronary blood flow or global O₂ delivery following surgery. In this study, postoperative coronary sinus blood flow, which approximates global coronary blood flow, did not increase. Calculated global O₂ delivery and O₂ consumption were, in fact, lower postoperatively both at rest and at MPR compared to the preoperative values. Yet the grossly abnormal anterior wall lactate metabolism, which usually results from hypoxia, documented preoperatively became normal following revascularization.

One possible explanation of such improvements in the effects of regional myocardial hypoxia is redistribution of delivery of oxygen between the previously ischemic and relatively nonischemic myocardial segments after the supplementary channels of oxygen delivery have been provided by regional revascularization. Preoperatively, even though global myocardial oxygen delivery increased in response to increase in global myocardial oxygen demand during atrial pacing stress, nonischemic myocardial segments might have received the major proportion of increased available blood flow. The ischemic myocardial segments, on the other hand, might still be deprived of adequate oxygen supply due to the presence of more severe coronary artery obstructive lesions with an increase in the severity or extent of regional mechanical dysfunction. Such redistribution of blood flow toward the nonischemic rather than ischemic areas following experimental coronary artery occlusion or during pacing induced angina has been previously suggested. Following regional revascularization a more even distribution of blood is possible. Therefore, improvement in regional metabolism may occur in the presence of unchanged or even reduced myocardial oxygen delivery. This is reflected in improvement of contractile state in the
previously ischemic myocardium and hence in the effectiveness of over-all cardiac performance. In contrast to the preoperative state, no mechanical deficit, necessitating increased work by the perfused myocardium, is present postoperatively even during stress, and hence the global metabolic requirements may be decreased. Improved myocardial efficiency postoperatively is also reflected by the fact that more mechanical work (rate-pressure product) could be performed by the left ventricle with less myocardial O2 consumption postoperatively. Thus redistribution of blood flow and improved myocardial efficiency might have been contributory to improved regional metabolism and mechanical function in these patients. However studies of regional blood flow and O2 delivery will be required to explain precisely the mechanism of improvement in regional and global metabolism and mechanical function following successful regional revascularization.

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References

27. Luzio VD, Roy PR, Sowton E: Angina in patients with

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occurred aortocoronary vein grafts. Br Heart J 36: 139, 1974


Abnormal regional metabolism and mechanical function in patients with ischemic heart
diseases: improvement after successful regional revascularization by aortocoronary
bypass.
K Chatterjee, J M Matloff, H J Swan, W Ganz, V S Kaushik, P Magnusson, M M Henis and J
S Forrester

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