Regional Myocardial Blood Flow in Patients with Residual Anterior and Inferior Transmural Infarction

By Edward M. Dwyer, Jr., M.D., Ralph B. Dell, M.D., and Paul J. Cannon, M.D.

SUMMARY
Regional myocardial perfusion rates were estimated in ten patients with a residual transmural anterior wall infarction and ten patients with a residual transmural inferior infarction. The results in these two groups of patients were compared to the regional myocardial perfusion patterns obtained in 25 patients with normal coronary arteriograms who failed to demonstrate any electrocardiographic evidence of a myocardial infarction. Clearance rates of xenon-133 from multiple areas of the heart were monitored externally with a multiple-crystal scintillation camera following selective injection of the isotope into either coronary artery. Local myocardial blood flow rates were calculated by the Kety formula.

In patients with normal coronary arteriograms, mean myocardial perfusion rates in the left ventricle (62 ml/100g/min) exceeded flow rates in the right ventricle and atrium. There was slight inhomogeneity of local perfusion rates but no significant differences among major subregions (anterior descending, diagonal, or circumflex) within the left ventricle.

In the group with an anterior transmural infarction, myocardial blood flow rates were significantly depressed (44 ml/100g/min) and asynergy was present in the region of the left ventricle supplied by the anterior descending artery, which was >80% narrowed. Diminished capillary blood flow was found in the right ventricular region in the patients with an inferior transmural infarction and right coronary artery narrowing of >80%. There was no evidence of nutrient flow to the inferior surface of the left ventricle after right coronary 133Xe injection.

The data indicate that myocardial perfusion was reduced in regions of the heart which correspond to the electrocardiographic and angiographic location of residual transmural infarction. The perfusion rates observed in areas showing electrocardiographic evidence of transmural infarction and regional ventricular asynergy, although subnormal, were surprisingly high and suggested the presence of residual viable myocardial cells.

Additional Indexing Words: Coronary artery disease  Scintillation camera Radioisotope study  Xenon-133

Knowledge of the adequacy of capillary perfusion within infarcted regions of the left ventricle has assumed increasing importance in recent years. Medical and surgical approaches are currently being proposed which attempt to improve survival of ischemic myocardium within and around areas of acute infarction. In addition, the success of aortocoronary bypass grafts in restoring blood flow to ischemic heart tissue in patients with coronary artery occlusions may depend, to a great extent, upon the presence of viable myocardium and an adequate capillary bed in the region distal to the graft.

Electrocardiographic changes diagnostic of a transmural infarction have heretofore been considered to be evidence of a myocardial infarction which extends completely across the ventricular wall. However, recent autoradiographic and pathological studies have demonstrated that islands of viable myocardium often persist in the area of the infarct.

From the Roosevelt Hospital and the Departments of Pediatrics and Medicine of the College of Physicians and Surgeons, Columbia University, New York, New York.

This work was supported by U.S. Public Health Service Grants HL 14148, HL 14236, and D 03993 from the National Institutes of Health. Dr. Dell is a Career Scientist of the Health Research Council of the City of New York, 1-743. Dr. Cannon is the recipient of a Research Career Development Award, HL 15031-08, from the National Heart and Lung Institute.

Address for reprints: Dr. Edward M. Dwyer, Jr., Department of Medicine, Cardiovascular Laboratory, Roosevelt Hospital, 428 West 59th Street, New York, New York 10019.

Received April 9, 1973; revision accepted for publication June 8, 1973.

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MYOCARDIAL PERFUSION IN TRANSMURAL MI

In a previous report, we described a method for quantitatively measuring the local capillary perfusion rates in multiple regions of the myocardium in patients who are undergoing diagnostic coronary arteriography. The method calculates myocardial blood flow from the clearance rate of xenon-133. Xenon-133 is selectively injected via a catheter into a coronary artery, and isotope washout curves from multiple discrete areas of the myocardium are measured externally with a multiple-crystal scintillation camera. Rate constants for isotope washout from heart muscle are calculated using a monoexponential model, and the myocardial capillary blood flow rates in multiple regions of the heart are calculated from these rate constants by the Kety formula. The pattern of regional myocardial perfusion rates so obtained is superimposed upon a tracing of the patient's coronary arteriogram obtained during the same study. Using this method, the pattern of myocardial perfusion rates observed in patients with normal and abnormal coronary arteriograms has been described. In addition, diffuse reductions of total left ventricular myocardial perfusion were noted in patients with significant radiographic narrowing of two or more branches of the left coronary artery.

The present study represents an extension of our investigations of myocardial perfusion in patients with coronary artery disease. It was undertaken to ascertain what alterations of regional myocardial blood flow patterns result from a previous transmural myocardial infarction. Regional myocardial blood flow rates were measured in 20 patients with electrocardiographic evidence of a residual transmural anterior or inferior myocardial infarction. Results were then compared to the perfusion patterns obtained in 25 patients with normal coronary arteriograms lacking electrocardiographic evidence of a myocardial infarction.

Methods

Subjects and Criteria for Selection

The infarction group was composed of 20 patients, each of whom was selected for this study on the basis of an electrocardiogram which showed a typical residual anterior or inferior myocardial infarction. Nine patients from this group were described and included in an earlier report. There were 19 males and one female whose ages ranged from 40 to 69 years. Each of the patients related a history compatible with a previous myocardial infarction. All were regarded as potential candidates for coronary artery surgery and were therefore studied.

Criteria for diagnosis of a transmural infarction were as follows: 1) an anterior infarction was diagnosed if a Q wave of 0.03 sec or greater was present in any of the leads V1-V4. By this criterion ten patients had an anterior infarction. In seven of ten patients, a Q-S pattern was present in leads V1-V6; 2) an inferior infarction was diagnosed in ten patients who showed a Q wave of 0.03 sec or greater in lead aV1 with additional Q waves in leads II and III.

The control group was composed of 25 patients whose electrocardiograms showed no evidence of a myocardial infarction. There were 16 men and nine women ranging in age from 40 to 88 years. Nine of the patients had rheumatic or calcific valvular heart disease; ten had primary myocardial disease of unknown etiology and six had no evidence of heart disease. All underwent coronary arteriography at the time of diagnostic cardiac catheterization, which was performed for clinical indications. Right and left coronary arteriograms were interpreted to be normal in all 25 patients. Fifteen of those subjects were included in a previous report.

Procedure

Each patient was brought to the laboratory in the postabsorptive state after premedication with oral pentobarbital (100 mg). A right and left heart catheterization was carried out, followed by left ventriculography. Pressures from the right atrium, pulmonary artery, left ventricle, and brachial artery were obtained. Selective coronary arteriography was performed according to the technique of Sones. Cine films were taken at 64 frames/sec using a 6-in image intensifier with a 35 mm camera. Details of this procedure have been outlined in a previous report.

Obstructive lesions in the right coronary artery and in the three major branches of the left coronary artery which were apparent on the arteriograms were graded on a 0 to 5 basis (0 = no occlusive disease; 1 = luminal irregularity; 2 = narrowing of 25 to 49%; 3 = narrowing of 50–70%; 4 = narrowing of 75 to 90%; and 5 = total occlusion).

Measurement of Regional Myocardial Perfusion

A detailed description of the equipment and procedures involved in the determination of regional myocardial perfusion with xenon-133 and a multiple crystal scintillation camera has been presented previously. An abbreviated description is outlined below.

At the conclusion of diagnostic coronary arteriography, a shallow (25°) LAO cineangiogram of either the left or the right coronary artery was obtained for later superimposition on the pattern of local myocardial perfusion rates. Upon completion of the arteriogram, 5-10 min were allowed to elapse before measurements of myocardial blood flow were made. Without any changes in the patient's position, the cine camera was then removed and replaced by the multiple-crystal scintillation camera and multichannel collimator which was positioned in the same plane and location over the patient's precordium. The camera consists of a

*Baird Atomic Autofluoroscope Model 5600.
rectangular grid composed of 294 individual NaI (TI) scintillation crystals arranged in 21 columns of 14 crystals.

Twenty to twenty-five mCi of xenon-133, dissolved in 1-2 ml of sterile pyrogen-free saline, were then injected over 1-2 sec into the main left or right coronary artery and gamma radiation emitted by xenon-133 in myocardial cells was monitored by means of the scintillation camera. Counts from each of the 294 crystals were recorded each second for 7 min onto magnetic tape.

Myocardial blood flow rates in the areas viewed by the multiple scintillation crystals were calculated from the rate constants by the Kety formula. The assumptions of this method, along with derivation of the equations which are employed, have been reported in detail previously.\(^9\) Capillary blood flow \((F)\) in ml/100 g of myocardial tissue/min in each region was calculated by the formula: \(F = k \times \lambda / \rho \times 100\) where \(k\) is the experimentally determined rate constant for xenon-133 washout from the region, \(\lambda\) is the myocardium to blood partition coefficient \((0.72)\) for xenon-133 which was obtained by Conn in normal dog heart,\(^10\) and \(\rho\) is the specific gravity of the tissue \((1.05)\).\(^11\) The standard deviation of each flow measurement \((\text{SDM})\) was also calculated for each crystal based upon the scatter of original data points about the fitted line. The regional perfusion measurements were printed out by the computer in 21 rows of 14 numbers with each number in the printout corresponding to the location in the rectangular detector of the crystal from which the data was obtained.

**Localization of Myocardial Regions**

Upon completion of the isotope study, a tracing of the patient's coronary arteriogram \((\text{LAO position})\) was made after it had been displayed on a screen at a known magnification. The computer printout of local myocardial perfusion rates was similarly enlarged and superimposed upon the tracing. Proper alignment of the computer printout and the arteriogram tracing was achieved through use of radioactive-radiopaque markers which appeared on both. The local myocardial perfusion rates were then printed onto the tracing in appropriate locations to form a myocardial perfusion pattern.

Figure 1 shows the perfusion pattern obtained after the injection of xenon-133 into the arteriographically normal dominant right coronary artery of a control subject. In right coronary studies, different myocardial regions were distinguished by landmarks provided by the coronary arteriogram. Mean perfusion rates in three

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**Figure 1**

This study illustrates the perfusion rates obtained following injection of xenon-133 into the right coronary artery. The pattern was characteristic of the group of patients with a normal dominant right coronary artery. Myocardial blood flow \((\text{ml/100g/min})\) was significantly higher in the region of the inferior left ventricle than in either the right atrial or right ventricular region.
regions were calculated in each right coronary study by averaging the local perfusion rates obtained by crystals overlying: 1) the right atrial area, 2) the region of the right ventricle, and 3) the inferior portion of the left ventricle. Crystals overlying the right atrium were located to the patient's right of the proximal right coronary artery on the perfusion pattern. Crystals overlying the right ventricle were located between the proximal right coronary artery and the interventricular septum; and those over the inferior left ventricle were located to the patient's left of the A-V node artery and the arteriographic "blush" produced by angiographic dye in the interventricular septum.

Figure 2 shows the perfusion pattern obtained after injection of xenon-133 into the main left coronary artery of another control subject whose coronary arteriogram was normal. A small but significant inhomogeneity of local perfusion rates was observed in this and other patients with normal left coronary arteriograms. A mean left ventricular perfusion rate was calculated from each left coronary study by averaging the local perfusion rates overlying the left ventricle. In addition, three left ventricular subregions were analyzed, each representing an area of the myocardium supplied by one of the three major branches of the left coronary artery (anterior descending, diagonal, and circumflex branches). For each subregion a mean perfusion rate was calculated by averaging the local flow rates of crystals whose area on the perfusion pattern was crossed by the tracing of the left coronary branch vessel.

**Results**

The scoring of the coronary arteriograms in ten patients with anterior myocardial infarction, along with the mean regional myocardial blood flow rates obtained in the left ventricle and in the left anterior descending, diagonal, and circumflex subregions, are presented in table 1. The results of studies of the right coronary artery in ten patients with an inferior myocardial infarction are presented in table 2. The mean regional perfusion values obtained from studies in 25 control patients without electrocardiographic evidence of infarction with angiographically normal coronary arteries are presented at the bottom of each table.

**Control Studies**

The left coronary artery was studied in 15 of the control subjects; an average of 36 crystals recorded myocardial xenon-133 washout curves in each. The mean left ventricular myocardial perfusion rates in this group of patients averaged 62 ml/100g/min;

![Figure 2](image)

*The computer printout of myocardial blood flow rates (ml/100g/min) obtained following selective injection of xenon-133 into the left coronary artery. The computer printout has been magnified, aligned, and superimposed on a tracing of the patient's left coronary arteriogram. A slight inhomogeneity of perfusion rates among the three subregions was noted in this patient.*

*Circulation, Volume XLVIII, November 1973*
Table 1

Angiographic Data and Regional Myocardial Blood Flow Rates in Patients with Anterior Transmural Infarctions Studied by Left Coronary Artery Injection

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Left ventriculography</th>
<th>Coronary artery anatomy</th>
<th>Regional MBF (ml/100 g·min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ant Desc</td>
<td>Diag</td>
</tr>
<tr>
<td>9</td>
<td>75</td>
<td>M</td>
<td>Apical-lateral dyskinesis</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>M</td>
<td>Normal</td>
<td>4</td>
<td>0</td>
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<tr>
<td>14</td>
<td>70</td>
<td>M</td>
<td>Antero-apical aneurysm</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>27</td>
<td>80</td>
<td>M</td>
<td>Postero-apical aneurysm</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>33</td>
<td>85</td>
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<td>Antero-apical aneurysm</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>49</td>
<td>75</td>
<td>M</td>
<td>Anterior aneurysm</td>
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<td>2</td>
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<tr>
<td>70</td>
<td>90</td>
<td>M</td>
<td>Antero-apical dyskinesis</td>
<td>4</td>
<td>0</td>
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<tr>
<td>72</td>
<td>80</td>
<td>F</td>
<td>Anterior aneurysm</td>
<td>3</td>
<td>5</td>
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<td>75</td>
<td>75</td>
<td>M</td>
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<td>0</td>
</tr>
<tr>
<td>84</td>
<td>75</td>
<td>M</td>
<td>Antero-lateral akinesis</td>
<td>5</td>
<td>4</td>
</tr>
</tbody>
</table>

Mean ± sd

| (N) | 4.2 (10) | 1.3 (7) | 3.5 (10) | 44 ± 13 (10) | 58 ± 13 (7) | 49 ± 17 (10) | 49 ± 12 (10) |

Normal mean value ± sd

| (N) | 0 (14) | 0 (14) | 0 (14) | 60 (14) | 70 (14) | 62 ± 12 (15) |

Abbreviations: Ant Desc = anterior descending; Diag = diagonal; Circ = circumflex; LV = left ventricle; Regional MBF = regional myocardial blood flow.

Coronary Arteriogram Grading: 0 = normal; 1 = luminal irregularity; 2 = 25-40% narrowing; 3 = 50-70% narrowing; 4 = 75-90% narrowing; 5 = total occlusion.

*Denotes not included due to overlap with another subregion.
†Significantly different from control value (P < 0.01).
‡Significantly different from control value (P < 0.02).

and there were no statistically significant differences
between the three left ventricular subregions.

An average of 28 crystals recorded myocardial xenon-133 washout curves in the 12 control studies of patients with normal right coronary arteriograms. In this group, the average myocardial capillary perfusion rate recorded by crystals overlying the right ventricle was 48 ml/100g/min. This was significantly below the average mean myocardial rate of 68 ml/100g/min recorded by crystals overlying the inferior left ventricular myocardium in ten patients who had dominant right coronary

Table 2

Angiographic Data and Regional Myocardial Blood Flow Rates in Patients with Inferior Transmural Infarctions Studied by Right Coronary Artery Injection

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Left ventriculography</th>
<th>Coronary artery anatomy</th>
<th>Regional MBF (ml/100 g·min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RCA</td>
<td>Ant Desc</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>M</td>
<td>Posterior wall dyskinesis</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>55</td>
<td>M</td>
<td>Posterior wall hypokinesis</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>55</td>
<td>M</td>
<td>Normal</td>
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<td>4</td>
</tr>
<tr>
<td>15</td>
<td>55</td>
<td>M</td>
<td>Posterior wall hypokinesis</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>29</td>
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<td>M</td>
<td>Infero-apical hypokinesis</td>
<td>5</td>
<td>3</td>
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<tr>
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<td>55</td>
<td>M</td>
<td>Inferior wall dyskinesis</td>
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<td>4</td>
</tr>
<tr>
<td>63</td>
<td>55</td>
<td>M</td>
<td>Normal</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>64</td>
<td>45</td>
<td>M</td>
<td>Posterior wall hypokinesis</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>67</td>
<td>45</td>
<td>M</td>
<td>Inferior wall hypokinesis</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>80</td>
<td>45</td>
<td>M</td>
<td>Normal</td>
<td>5</td>
<td>4</td>
</tr>
</tbody>
</table>

Mean ± sd

| (N) | 4.9 (10) | 3.9 (10) | 1.6 (10) | 2.5 (10) | 35 ± 6 (10) | 37 ± 6* (10) |

Normal Mean Value ± sd

| (N) | 0 (10) | 0 (10) | 0 (10) | 34 ± 10 (10) | 48 ± 11 (10) | 68 ± 12 (10) |

Abbreviations: See table 1; RCA = right coronary artery; RA = right atrium; RV = right ventricle; Inf LV = inferior left ventricle.

Coronary Arteriogram Grading: see Table 1.

*Significantly different from control value (P < 0.05).
arteries which supplied this region. The average perfusion rate recorded by crystals overlying the right atrial area was the lowest, 34 ml/100g/min.

**Anterior Myocardial Infarction Studies**

Severe obstructive disease of the anterior descending artery was present in all ten patients with an anterior myocardial infarction as shown by a score of 4 or 5 for this vessel (table 1). Five of the patients in this group had an anterior wall aneurysm and four had lesser abnormalities of wall motion. There tended to be disease in the other two major branches of the left coronary artery in these patients. The mean score for the diagonal was 1.3 with three patients having a score of 4 and the others a score of 0, while the mean score for the circumflex was 3.5 (range 0-5).

The myocardial perfusion pattern obtained from study of patient No. 75 with an anterior myocardial infarction is shown in figure 3. In this patient there was an 80% narrowing of the anterior descending artery along with an occlusion of the circumflex. Myocardial perfusion in the anterior descending subregion was low (33 ml/100g/min), a pattern which was typical of the patients in this group.

The group mean myocardial perfusion rate in the anterior descending subregion of 44 ml/100g/min (table 1) was significantly less \( P < 0.01 \), two-tailed \( t \) test than the mean flow of 63 ml/100g/min observed for the corresponding region in the control group. The perfusion rate was not significantly different between the patients with an infarction and the control group for the diagonal and circumflex subregions although, on average, the infarct group tended to have lower flows. The overall flow rate for the left ventricle of 49 ml/100g/min was significantly less \( P < 0.02 \) than the total left ventricular flow rate for the control group.

**Inferior Myocardial Infarction Studies**

In nine of ten patients with an inferior myocardial infarction, there was total occlusion of the

![Figure 3](http://circ.ahajournals.org/)

**Figure 3**

This study illustrates observations which were typical of patients with an anterior transmural infarction. Coronary arteriography in this patient (75) demonstrated an 80% narrowing of the anterior descending artery and total occlusion of the circumflex artery. In the subregion supplied by the anterior descending artery, the mean perfusion rate was significantly reduced below the mean value obtained in the diagonal subregion.

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right coronary artery; in the remaining patient there was 90% narrowing of the right coronary (table 2). Abnormalities of posterior-inferior left ventricular wall motion were observed in seven patients in this group. Hypokinetic motion was seen in five patients and dyskinesis was present in two. The other three patients had normal left ventricular wall motion.

The myocardial perfusion pattern observed during right coronary artery studies on a patient with an inferior infarction is shown in figure 4. No washout of xenon-133 was observed in the inferior left ventricle after a bolus of xenon-133 dissolved in saline was injected into the occluded right coronary artery. This result was observed in nine out of ten patients with diseased right coronary arteries and inferior transmural infarction; in the tenth patient, the mean myocardial perfusion rate recorded over the inferior left ventricle after a right coronary tracer injection was 42 ml/100g/min, a value markedly lower than that found in similar studies of the control group (table 2).

Collateral blood vessels, bridging the proximal right coronary artery occlusion and supplementing perfusion to right ventricular myocardium, were visualized arteriographically in five of nine patients with inferior transmural infarction. Despite the presence of a collateral blood supply, the average mean right ventricular myocardial perfusion rate in the inferior infarction group was 37 ml/100g/min, a value significantly less ($P < 0.05$) than the value of 48 ml/100g/min for right ventricular flow observed in the control group (table 2).

**Discussion**

In the present study, regional myocardial perfusion rates were measured in 20 patients with electrocardiographic evidence of transmural myocardial infarction and compared to results obtained in 25 control patients without evidence of infarction who had normal coronary arteriograms.

**Control Group**

The control group included six patients without evidence of heart disease and 19 patients with primary myocardial or valvular heart disease. The results of regional myocardial perfusion measurements in the control studies extend our previous

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**Figure 4**

The perfusion pattern in this patient (#10) with a total occlusion of the right coronary artery and an inferior wall infarct shows an absence of significant isotope washout from the inferior left ventricle. Mean myocardial blood flow rates were normal in the right atrial region but reduced in the area of the right ventricle.

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observations made with a smaller number of patients. Slight inhomogeneity of local myocardial blood flow rates was found in the left ventricle of patients with normal left coronary arteriograms, but there were no significant differences between the average myocardial perfusion rates in the sub-regions supplied by the anterior descending, diagonal, and circumflex arteries. The average mean left ventricular myocardial blood flow rate of 63 ml/100g/min found in the control group is similar to that reported by other workers monitoring single washout curves ($^{133}$Xe, H$_2$) from the left ventricle of patients with radiographically normal coronary arteries.

In studies of the control subjects with normal right coronary arteriograms, differences in regional perfusion were more pronounced. The mean myocardial blood flow (68 ml/100g/min) in the inferior portion of the left ventricle supplied by a dominant right coronary artery was significantly higher than the mean myocardial perfusion rate in the region of the right ventricle (48 ml/100g/min) which, in turn, exceeded that measured in the right atrial area (34 ml/100g/min). Similar differences between atrial, right, and left ventricular blood flow rates have been reported by Domenech et al. in canine experiments, using radioactive microspheres. Since myocardial oxygen demand is determined largely by pressure generation in the ventricle, the data suggest that because of its greater pressure work, the left ventricle requires not only a greater muscle mass, but also a larger blood supply per unit tissue than the right ventricle.

**Anterior Transmural Infarction**

Severe stenosis of the anterior descending artery was present in each patient with a residual anterior transmural infarction; the mean arteriographic score for this vessel in the group was 4.2. Diminished capillary perfusion was found in the ventricular subregion supplied by the diseased anterior descending artery. The group mean perfusion rate in the anterior descending region, 44 ml/100g/min, was significantly lower than that observed in the same region in the control subjects without infarction that had normal coronary arteriograms. These observations confirm previous studies indicating that the degree of isolated coronary artery obstruction usually exceeds 75% before resting blood flow is reduced. Until recently, observations on isolated lesions of the coronary circulation were confined to experimental models. Smith et al., however, measured the pressure gradients across lesions of an isolated coronary artery branch and the nutrient flow to the myocardial region supplied by that branch at the time of coronary bypass surgery. They found that significant pressure gradients across the lesion and diminished distal perfusion occurred only when preoperative arteriography demonstrated a proximal lesion which was narrowed greater than 80%. They found mean blood flow rates ($^{133}$Xe washout technique) in the area of myocardium supplied by the anterior descending artery they found were considerably less than perfusion rates in our ten patients with similar anterior descending artery lesions and residual anterior infarction who were studied at the time of coronary arteriography. The different circumstances of the two studies and/or the different methods of curve analysis may account for this discrepancy.

Regional asynergy in the anterior-apical left ventricle was found in nine of ten patients with anterior transmural infarction. It is possible that a reduced level of capillary perfusion in myocardium distal to the anterior descending lesions was associated with reversible localized abnormalities of ventricular contraction in some of the patients. In support of this hypothesis are reports that myocardial blood flow (assessed from $^{133}$Xe or H$_2$ washout curves after instillation of inert gas into the graft) was restored to normal values by successful aortocoronary bypass grafts. Several other investigators have described improvement of regional left ventricular contraction abnormalities following surgical relief of acute or chronic myocardial ischemia. However, this appears to occur less frequently than an improvement in general cardiac function as assessed by exercise testing, standard hemodynamics, and ventriculography ejection fraction. In the present series of patients, none of the eight patients with anterior transmural infarction who had bypass surgery were restudied to ascertain whether or not there was a return of function to normal in a previously asynergic region. Aneurysmectomy confirmed that the wall motion abnormalities in three of the patients were primarily due to scar. A systematic attempt to distinguish between scar containing islands of ischemic myocardium and a chronically ischemic full thickness myocardium was not made in this study.

Multiple vessel involvement was frequently apparent on the arteriograms of the patients with anterior transmural infarction. The group mean arteriographic scores were 1.6 for the diagonal and 3.1 for the circumflex branches. This is consistent with observations by Likoff et al. that multiple
vessel involvement is the most frequent finding in patients undergoing arteriography for symptomatic coronary artery disease. The average mean myocardial perfusion rates in the diagonal and circumflex ventricular subregions were reduced below the values found in the control groups, but the reductions did not obtain statistical significance. However, when the local perfusion values from all crystals overlying the entire left ventricle were averaged for each patient in both groups, it was found that the average mean left ventricular myocardial perfusion rate was significantly lower in the anterior transmural infarction group than in the control group with radiographically normal left coronary arteries.

These results in a group of patients with multiple vessel coronary disease are consistent with our previous finding⁶ that mean left ventricular perfusion was significantly reduced in a group of patients with greater than 75% obstructions of two or more branches of the left coronary artery. Several other groups, however, using inert gas techniques in which a single washout curve from the entire left ventricle is measured, found that total left ventricular perfusion was normal in patients with radiographically documented coronary artery disease.²⁵ ²⁶ This may have occurred because areas of reduced perfusion were obscured by much larger regions with normal perfusion when only a single myocardial washout curve was measured. Such a suggestion is supported by the results obtained in patient No. 49 (table 1). In this patient with an isolated left anterior descending obstruction, the myocardial perfusion rate was reduced in the anterior descending subregion, but was normal or increased elsewhere in the ventricle; the mean perfusion rate for the entire left ventricle of this patient was comparable to that found in the control studies of patients without demonstrable coronary artery disease.

**Inferior Transmural Infarction**

Nine of the ten patients with residual inferior infarction exhibited a total occlusion of the right coronary artery. The tenth had a 90% obstruction. The most striking difference from controls, apparent on the perfusion patterns of nine of these patients, was the absence of detectable xenon-133 washout curves from the inferior left ventricle after tracer was injected into the right coronary artery. In the other patient, inferior left ventricular perfusion was detected but was subnormal. Klocke et al. have presented calculations which indicate that areas of myocardium with reduced perfusion rates might not be fully loaded with inert gas after a bolus injection.¹⁴ With our present measurement technique, isotope delivery to a myocardial region can be assessed from the computer printout of the peak count observed by each scintillation crystal and time after start of the study that the peak count occurred. If too few counts arrive in a region, the peak count is low and the scatter of the points comprising the local washout curve so wide that the computer is unable to apply a monoexponential equation to the data. This occurred in the nine studies. These results suggest that diffusible nutrients are not effectively supplied by bridging right coronary collaterals to the inferior left ventricle in the patients with transmural inferior infarction.

From the absence of detectable xenon-133 washout curves over the inferior left ventricle after right coronary isotope injection, one need not conclude that there is no capillary perfusion in this region. This only implies that the bolus of xenon-133 did not reach the inferior wall of the left ventricle in sufficient quantity to permit acceptable washout curves. Most likely, some capillary flow was available to the inferior left ventricle via collaterals from the left coronary artery. Collateral blood vessels from the left coronary artery to this area were visible on the arteriograms of several of our patients, and Williams et al.²⁷ have demonstrated during bypass surgery that intercoronary collateral flow to tissue supplied by diseased right coronary arteries may be provided from the left circumflex or anterior descending vessels with blood flow values ranging between 10 and 20 ml/min.

Theoretically, if xenon-133 had been injected into the left coronary artery of one of the patients with inferior infarction and absent perfusion by the right coronary study, it is likely that there would have been delivery of adequate amounts of isotope via collaterals to the inferior left ventricle so that capillary perfusion in the region could be estimated. Unfortunately, when the patient is positioned in a LAO position and the injection made in the left coronary artery, measurement of inferior wall washout is complicated by radioactivity from deposition of xenon-133 in the anterior wall.

In five of the patients with inferior infarction and occluded proximal right coronary arteries, the arteriograms showed extensive collateral vessels which originated from the proximal branches (sinus node, right ventricular, and marginal vessels) and

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*(Circulation, Volume XLVIII, November 1973)*
formed bridges around the occlusion. The regional perfusion measurements suggested, however, that the capillary blood supply provided by these collateral vessels was normal only in the right atrial region. The average mean right ventricular myocardial perfusion rate of 37 ml/100g/min was significantly lower than in the control group and perfusion was not detected in the inferior wall of the left ventricle. Despite subnormal myocardial perfusion to the right ventricle, none of the patients had an increased right ventricular end-diastolic pressure or evidence of right heart failure. These results in man therefore are similar to observations of Brooks et al. who found that under control conditions total right coronary occlusion in dogs caused no change in cardiac output or right and left ventricular pressures even though there was a decline in right ventricular contractility. In contrast, with an elevated pulmonary artery pressure, the over-all cardiac function was increasingly dependent upon the performance of the right ventricle, which in turn was directly related to the level of right coronary artery flow and inversely related to the degree of right coronary constriction.

Unfortunately, there are no available data which confirm or exclude the presence of right ventricular infarction in this group (table 2). Focal myocardial damage rather than extensive infarction of the right ventricle has been the more common finding in autopsy studies. The uncommon occurrence of right ventricular infarction suggests that despite subnormal right ventricular perfusion generally accompanying right coronary artery occlusion and inferior infarct, the lower pressure demands put upon the right ventricle permit it to retain viability and normal pump function.

Critique of the Method

The study of regional myocardial perfusion in patients with residual myocardial infarction is accompanied by several problems related to the measurement technique.

1) Because of the spherical geometry of the heart, two myocardial surfaces may be viewed by a single crystal which monitors xenon-133 washout from all the tissue within its field of view. In an attempt to minimize overlap of the anterior and posterior wall of the left ventricle, the present studies were performed in the left anterior oblique view. This displaces the region supplied by the anterior descending vessel in such a way that there is minimal overlap of other myocardial tissue supplied by the left coronary artery and brings most of the circumflex subregion from a posterior to a lateral location. In right coronary studies, the inferior left ventricle is also displaced in the left anterior oblique view with minimal overlap of other tissue supplied by the same vessel.

2) The primary data, i.e., the rate constants for isotope removal, were multiplied by a factor, $\lambda/\rho$, to determine myocardial blood flow. The value used for $\lambda$ in the calculations was 0.72, the value found by Conn in static studies of normal canine myocardium. Thus the expression of the results in terms of nutrient blood flow (ml/100g/min) must be interpreted with caution since they depend upon an assumed value for the myocardial:blood partition coefficient for xenon-133. Flow studies in isolated dog hearts have shown an excellent correlation between flow/gram calculated from the labeled inert gas washout curve using 0.72 for $\lambda$ and flow/gram measured with a flowmeter. It is not possible to exclude local alterations of the partition coefficient in areas of infarction due to fibrosis or fatty infiltration.

As discussed in more detail elsewhere, the presence of fat in the tissue under study alters the tail of a xenon-133 washout curve. Monoexponential analysis of the initial portion of each washout curve, as used in these studies, was selected in part to minimize any effect of altered tissue composition upon the measurement of flow/gram in the myocardial muscle within the field of view of the detector. The tissue flow values (30-50 ml/100g/min) obtained in areas of transmural infarction in the present studies are ten- to twentyfold the values which would be obtained in fat or scar tissue from published t 1/2 of xenon-133 washout from these tissues; thus the flow values probably reflect the nutrient blood supply to the residual islands of ventricular muscle and not to fat or scar.

3) The myocardial perfusion rates obtained with the inert gas measurement techniques yield a value of flow per unit volume or mass of tissue (ml/100g/min). It is likely that not only the clearance rate but also the mass of tissue are reduced in a region of infarction. Unfortunately, precise quantification of regional myocardial mass in human subjects is not possible with current techniques. Therefore, total flow to a myocardial region cannot be determined.

Despite these limitations of the present method for estimating regional myocardial perfusion in its current state of development, the basic approach
and observations (i.e., simultaneous measurement of isotope washout from multiple regions of the heart of a patient with coronary disease with a multichannel collimator and multiple detectors) retain validity which are independent of the form of mathematical analysis or of assumptions involved in expressing the data in terms of nutrient blood flow.

Applications

The present results suggest that there is a surprising high level of capillary perfusion in the surviving myocardium of patients with a residual anterior transmural infarction. Figure 5 shows a portion of an aneurysm, resected from the left ventricular wall of patient No. 72, containing strands of viable myocardial fibers with a surrounding meshwork of scar. The basis by which this residual myocardial muscle survived the acute injury is not known. Both inherent cellular differences and uneven collateral circulation to different regions have been postulated. The demonstration of residual, albeit subnormal, myocardial perfusion rates in myocardial subregions which were the site of infarction should provide support for current attempts to improve survival of ischemic tissue within and around an acute infarct. In addition, preoperative data documenting residual viability of myocardium in the region of an infarct may be helpful in decisions regarding the advisability of coronary bypass surgery on an artery which supplies the site of an old infarction.

References


Regional Myocardial Blood Flow in Patients with Residual Anterior and Inferior Transmural Infarction

EDWARD M. DWYER, JR., RALPH B. DELL and PAUL J. CANNON

Circulation. 1973;48:924-935
doi: 10.1161/01.CIR.48.5.924

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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