PATHOPHYSIOLOGY AND NATURAL HISTORY

RIGHT VENTRICULAR INFARCTION

Hemodynamically important right ventricular infarction: follow-up evaluation of right ventricular systolic function at rest and during exercise with radionuclide ventriculography and respiratory gas exchange

LOUIS J. DELL’ITALIA, M.D., NICHOLAS J. LEMBO, M.D., MARK R. STARLING, M.D., MICHAEL H. CRAWFORD, M.D., RICHARD S. SIMMONS, M.D., JOHN C. LASHER, M.D., RALPH BLUMHARDT, M.D., JACK LANCASTER, M.D., AND ROBERT A. O’ROURKE, M.D.

ABSTRACT The prognosis and recovery of right ventricular systolic function in patients with hemodynamically documented right ventricular myocardial infarction (RVMI) is unclear. Therefore 27 patients who met hemodynamic criteria for RVMI were followed for at least 1 year. Four patients died within 1 year and 23 survived. Postmortem examination performed in three of the four patients showed extensive infarction of the right and left ventricles. Survivors underwent early and late follow-up resting radionuclide ventriculograms and late exercise studies. During long-term follow-up (1 to 4 years) resting radionuclide ventriculography demonstrated a significant improvement in right ventricular ejection fraction (30 ± 7% to 43 ± 8%; p < .001) and right ventricular wall motion index (2.2 ± 0.4 to 1.5 ± 0.5; p < .001) in 18 patients who survived longer than 1 year. Fourteen of these patients underwent upright bicycle exercise while off β-blocking drugs and peak radionuclide ejection fraction was acquired after anaerobic threshold was achieved. Right ventricular ejection fraction increased significantly from 41 ± 10% to 47 ± 12% (p < .001), as did the left ventricular ejection fraction (55 ± 15% to 60 ± 12%; p < .05). The direction and magnitude of change of the right ventricular ejection fraction correlated significantly with the left ventricular ejection fraction (r = .82, p < .02). Deviations from this correlation occurred in patients who had a decreased forced expiratory volume in 1 sec and an abnormal ventilatory reserve during exercise. Also, the onset of anaerobic threshold during exercise correlated with the peak exercise right ventricular ejection fraction (r = .82, p < .02). However, there was no significant correlation with the left ventricular ejection fraction. Therefore we conclude that patients with RVMI and significant left ventricular dysfunction have a poor 1 year prognosis. Otherwise, the long-term prognosis of patients who present with hemodynamically important RVMI is excellent. Furthermore, in the recovery phase, right ventricular ejection fraction increases and right ventricular functional reserve is preserved during exercise in patients not limited by angina, pulmonary disease, or left ventricular failure.


THE IDENTIFICATION of right ventricular myocardial infarction (RVMI) varies depending on the diagnostic technique used.1–8 However, only hemodynamic findings have been validated against necropsy-proven right ventricular necrosis, thereby establishing hemodynamic criteria as the most accurate markers of RVMI.9 In numerous noninvasive studies, radionuclide ventriculographic1–5 or two-dimensional echocardiographic6–7 evidence of right ventricular enlargement or decreased systolic performance has been reported to occur in 40% to 50% of patients with acute inferior myocardial infarction (IMI). In contrast, we showed that only 20% of 53 consecutive patients with acute IMI had hemodynamic findings characteristic of RVMI even after a volume challenge. Because of this discrepancy in the diagnosis of RVMI, its short-
long-term prognosis has been controversial. We9 and others9,10 have noted that patients who present with hemodynamically important RVMI have a poor short-term prognosis, whereas Haines et al.11 have reported that patients with radionuclide ventriculographic evidence of RVMI have an excellent long-term survival. In addition, other investigators have reported a marked improvement in right ventricular systolic performance in the recovery phase.4,12 However, these studies4,11,12 did not use hemodynamic criteria for the early diagnosis of RVMI and therefore may have included patients with decreased right ventricular systolic performance secondary to other causes such as chronic obstructive pulmonary disease, left ventricular dysfunction, or transient right ventricular ischemia rather than infarction. To date no study has provided long-term follow-up data on patients presenting with hemodynamically important RVMI.

Previously, we reported that hemodynamic and radionuclide ventriculographic evidence of significant left ventricular dysfunction along with persistent precordial ST segment depression were predictors of death in patients 1 year after acute IMI.13 Therefore we attempted to determine whether these same markers applied to the 13 patients with hemodynamic evidence of RVMI from the previous cohort13 and to an additional 14 patients with hemodynamically documented acute RVMI whom we studied to determine the relative efficacy of dobutamine compared with nitroprusside therapy.14 Also, follow-up studies were performed in these patients who met strict criteria for acute RVMI by both hemodynamics and radionuclide ventriculography to evaluate right ventricular systolic function and to determine the functional reserve of the right ventricle by means of rest and exercise radionuclide ventriculography and respiratory gas exchange analysis.

**Methods**

Patients. The study group consisted of 13 consecutive patients who presented to the Audie Murphy Memorial Veterans Administration Hospital between 1981 and 1983 and 14 consecutive patients who presented to Medical Center Hospital between 1983 to 1985 with hemodynamic evidence of RVMI (both a right atrial pressure [RAP] ≥ 10 mm Hg and a right atrial/pulmonary arterial wedge pressure [PAWP] ratio ≥ 0.8 either at rest or after a volume load).5 The total study population consisted of 22 men and five women, 41 to 78 years old (mean 61 ± 10 [SD]). In addition to RVMI, all had evidence of acute inferior transmural myocardial infarction. Four patients died and 23 patients were discharged from the hospital. Three of the four patients died within 72 hr of admission and the remaining patient died after presenting with acute anterior myocardial infarction 3 months later. Two patients died before radionuclide ventriculography could be performed, and the remaining two patients had right ventricular ejection fractions of 15% and 22% and left ventricular ejection fractions of 40% and 50%, respectively. All hospital survivors had both a right ventricular ejection fraction under 40% (mean 29 ± 7%, range 17% to 38%) and evidence of right ventricular akinesis or dyskinesis on wall motion analysis.5,14 Acute left ventricular ejection fraction in the 1 year survivors ranged from 40% to 75% (mean 56 ± 11%).

**Short-term studies**

**Electrocardiography.** The electrocardiogram (ECG) obtained upon admission to the coronary intensive care unit and an ECG obtained 24 hr after admission were evaluated for ECG ST segment depression in the following leads: (1) I and aVL, (2) V1 to V4, and (3) V5 or V6. An ECG ST segment abnormality was identified when 1 mm or more of ST segment depression was present at 80 msec after the J point. Also, ECG ST segment depressions were summed for the precordial leads (V1 to V4) to describe the magnitude of precordial ECG ST segment depression as follows: (1) 1 to 4 mm, (2) 5 to 10 mm, and (3) over 10 mm.

**Hemodynamics.** A No. 7 balloon-tip, flow-directed catheter (American Edwards Laboratories, Santa Ana, CA) was inserted into the pulmonary artery within 48 hr of the onset of symptoms in each patient. Cardiac index, stroke volume index, right ventricular stroke work index, systemic vascular resistance, total pulmonary resistance, and pulmonary vascular resistance were calculated in the standard fashion.5,14 A normal saline fluid challenge was given if the initial PAWP was less than 15 mm Hg.

**Radionuclide ventriculography.** Equilibrium radionuclide ventriculography was performed as previously described.5,14 Four regions of the right ventricle were evaluated for wall motion abnormalities: the apex, inferior and lateral walls, and outflow tract. Wall motion was evaluated independently by two cardiologists and scored as follows: 1 = normal, 2 = hypokinesis, 3 = akinesis, 4 = dyskinesis. The scores of all identified segments were added and the total score was divided by the total number of segments to obtain a right ventricular wall motion index. Right ventricular ejection fraction was calculated by the technique described by Maddahi et al.15

**Follow-up evaluation**

**Clinical follow-up.** All patients were followed for at least 12 months and evaluated 1 to 4 years after RVMI (2.2 ± 1.3). Two hospital survivors died of recurrent myocardial infarction 18 and 24 months after RVMI. Eighteen of the remaining 21 survivors had resting radionuclide ventriculography at follow-up. In these 21 patients, four underwent coronary artery bypass grafting and one had percutaneous transluminal coronary angioplasty of the right coronary artery before follow-up studies were performed. At the time of evaluation, 12 patients were taking calcium entry–blocking drugs, and one was taking β-blocking drugs alone. Six patients had no complaints of chest pain, whereas 14 patients complained of infrequent chest pain with activity. One patient had exertional angina with minimal activity, which progressed to angina at rest when β-blocking drugs were discontinued. Only one patient was being treated with digoxin for symptoms of congestive heart failure. Of the survivors 12 had returned to full employment.

**Exercise.** Fourteen of the 18 patients who underwent resting radionuclide ventriculography (12 men and two women) underwent graded upright bicycle exercise after β-blocking drugs were discontinued for at least 72 hr. At the time of exercise, eight patients were taking calcium entry–blocking drugs and the remaining six patients were taking no antianginal medication. Three patients did not exercise because of severe degenerative joint disease and one patient developed unstable angina when β-blocking drugs were discontinued. Before exercise all patients underwent pulmonary function testing to obtain a forced expira-
tory volume in 1 sec (FEV, in liters). Exercise was conducted 1 to 4 years after myocardial infarction (2.3 ± 1.4 years).

Exercise was started at a workload of 15 to 20 W, determined by the predicted maximum oxygen consumption of the patient, and increased at 1 min increments during exercise. An ECG including standard limb leads and precordial lead V₅ was obtained at each minute of exercise, and blood pressure was obtained by cuff sphygmomanometer. Respiratory gas exchange was performed on day 1 with a Beckman metabolic measurement cart interfaced with a Hewlett Packard 85 personal computer and programmed with a Beckman clinical exercise testing program. Gas analysis was performed with a Beckman LB-2 CO₂ and OM-11 O₂ analyzers in the metabolic cart with O₂ and CO₂ sampling from a mixing chamber every 30 sec of exercise. Maximum oxygen consumption was determined at peak exercise. Anaerobic threshold was determined by a nonlinear increase in both the minute ventilation to oxygen consumption ratio (VE/VO₂) and CO₂ production (VCO₂), the latter producing a decreased or unchanged ventilatory equivalent for CO₂ (VE/VCO₂). Oxygen consumption at anaerobic threshold was then divided by the predicted maximum oxygen consumption, and anaerobic threshold was obtained as a percent value. The anaerobic threshold was determined by one investigator blinded to the results of the exercise radionuclide ventriculography. The ventilatory reserve was determined by subtracting total ventilation from the maximum voluntary ventilation (FEV₁ × 40). Ventilatory impairment was identified when this value was less than 15 liters.

Equilibrium radionuclide ventriculography was performed on day 2, 24 to 48 hr after exercise respiratory gas exchange analysis. Two-minute acquisitions were obtained in the upright left anterior oblique view that best separated the right and left ventricles in the plane of the interventricular septum at rest and at peak exercise. The peak exercise study was acquired at one workload (15 to 20 W) beyond the anaerobic threshold that had been determined on the previous day. At this point, a 2 min acquisition was obtained where heart rate had reached a stable maximum. Frame rate was determined by the RR interval achieved at peak exercise. Right ventricular ejection fraction during exercise was calculated by the technique previously described by Maddahi et al. The response of right and left ventricular ejection fraction to upright bicycle exercise was considered normal when ejection fraction increased greater than 5 units above the resting value. No patient was limited by angina pectoris and there were no ECG changes suggestive of ischemia during exercise studies performed on days 1 or 2.

Statistical analysis. Hemodynamic values were compared between survivors and nonsurvivors by an unpaired Student's t test with a separate variance estimate. Values are reported as mean (±) 1 SD. Short-term to follow-up and rest to peak exercise right and left ventricular ejection fractions were compared with a paired Student's t test. Least-squares linear regression analysis was performed on anaerobic threshold and ejection fraction values to obtain correlation coefficients and 95% confidence intervals. A p value of <.05 was considered significant.

Results

Short-term studies

Electrocardiography. Three of four patients who died had more than 10 mm summed ST segment depression in precordial leads V₁ to V₅ and no survivor demonstrated this ECG finding (figure 1). All three patients with this particular ECG finding died within 72 hr of admission. The remaining patient had persistent ST segment depression in V₁ to V₅ and this patient died 3 months later. No 1 year survivor demonstrated persistent precordial ST segment depression.

Hemodynamic evaluation (table I). Cardiac index, stroke volume index, and left ventricular stroke work index were significantly lower in the four patients who died. Left ventricular function curves demonstrated that these nonsurvivors had a stroke volume index less than 23 ml/m² over a wide range of PAWPs, whereas patients who were alive at 1 year follow-up had stroke

![FIGURE 1. Representative ECG in a nonsurvivor demonstrating summed ST depression of greater than 10 mm in precordial leads V₁ to V₅ with minimal ST segment elevation in limb leads II, III, and aVF.](http://circ.ahajournals.org/lookup/fig/1/1)
TABLE 1
Mean hemodynamic data in four patients who died and 23 survivors before and after normal saline infusion

<table>
<thead>
<tr>
<th>Patient Type</th>
<th>RAP (mm Hg)</th>
<th>PAWP (mm Hg)</th>
<th>PA (mm Hg)</th>
<th>CI (l/min/m²)</th>
<th>SVI (ml/m²)</th>
<th>RVSWI (g/m²-m²)</th>
<th>LVSWI (g/m²-m²)</th>
<th>HR (bpm)</th>
<th>BP (mm Hg)</th>
<th>SVR (dyne-sec-cm⁻²)</th>
<th>TPR (dyne-sec-cm⁻²)</th>
<th>PVR (dyne-sec-cm⁻²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>14 ± 3</td>
<td>13 ± 5</td>
<td>24 ± 4</td>
<td>1.7 ± 0.3⁴</td>
<td>20 ± 3⁴</td>
<td>2.2 ± 0.7</td>
<td>20 ± 4⁴</td>
<td>85 ± 21</td>
<td>87 ± 19</td>
<td>2160 ± 280⁴</td>
<td>650 ± 250</td>
<td>339 ± 211</td>
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<tr>
<td>Nonsurvivors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>14 ± 2</td>
<td>13 ± 5</td>
<td>24 ± 4</td>
<td>1.5 ± 0.4⁴</td>
<td>16 ± 5⁴</td>
<td>1.5 ± 1.0⁴</td>
<td>15 ± 6</td>
<td>99 ± 29</td>
<td>87 ± 22</td>
<td>2236 ± 460</td>
<td>742 ± 140⁹</td>
<td>217 ± 18</td>
</tr>
<tr>
<td>Saline</td>
<td>17 ± 2</td>
<td>16 ± 5</td>
<td>24 ± 4</td>
<td>1.5 ± 0.4⁴</td>
<td>16 ± 5⁴</td>
<td>1.5 ± 1.0⁴</td>
<td>15 ± 6</td>
<td>99 ± 29</td>
<td>87 ± 22</td>
<td>2236 ± 460</td>
<td>742 ± 140⁹</td>
<td>217 ± 18</td>
</tr>
<tr>
<td>One year survivors</td>
<td>11 ± 4</td>
<td>12 ± 5</td>
<td>18 ± 4</td>
<td>2.1 ± 1.0⁴</td>
<td>30 ± 7</td>
<td>3.0 ± 2.6</td>
<td>35 ± 10</td>
<td>71 ± 10</td>
<td>95 ± 16</td>
<td>1710 ± 485</td>
<td>380 ± 130</td>
<td>130 ± 64</td>
</tr>
<tr>
<td>Control</td>
<td>15 ± 2</td>
<td>16 ± 2</td>
<td>22 ± 4</td>
<td>2.2 ± 0.5</td>
<td>31 ± 6</td>
<td>3.0 ± 1.5</td>
<td>36 ± 10</td>
<td>70 ± 11</td>
<td>97 ± 14</td>
<td>1600 ± 400</td>
<td>440 ± 120</td>
<td>123 ± 64</td>
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<td>Normal</td>
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</table>

RAP = right atrial pressure; PAWP = pulmonary arterial wedge pressure; PA = mean pulmonary arterial pressure; CI = cardiac index; SVI = stroke volume index; RVSWI = right ventricular stroke work index; LVSWI = left ventricular stroke work index; HR = heart rate; BP = mean arterial pressure; SVR = systemic vascular resistance; TPR = total pulmonary resistance; PVR = pulmonary vascular resistance.

*p < .001; **p < .01; *p < .05 in comparisons of nonsurvivors vs 1 year survivors, control vs control, and normal saline vs normal saline.

volume indexes above this value at baseline or after a volume infusion (figure 2).

Postmortem examination. Postmortem examination was performed in two of the three patients who died early and in the one patient who died at 3 months. All patients had extensive right ventricular necrosis and severe diffuse coronary artery disease with marked calcification of the coronary arteries. All heart specimens demonstrated extensive necrosis of the left ventricular posterior and anterior wall. The lungs demonstrated moderate-to-severe edema in all cases and there was no evidence of pulmonary emboli. Two patients had evidence of emphysematous changes in the lungs.

Follow-up evaluation of right ventricular function

Resting radionuclide ventriculography. Eighteen patients had resting, supine radionuclide ventriculograms early and at long-term follow-up. Right ventricular ejection fraction increased from 30 ± 7% to 43 ± 8% (p < .001) while left ventricular ejection fraction remained unchanged (58 ± 10% to 56 ± 12%) (figure 3). Right ventricular wall motion analysis demonstrated that the majority of akinetic and dyskinetic segments improved to normal or hypokinetic wall motion from the short-term study to follow-up analysis. Consequently, right ventricular wall motion index decreased significantly from 2.2 ± 0.4 to 1.5 ± 0.5 (p < .001).

Exercise radionuclide ventriculography. The mean upright ventricular ejection fraction increased significantly from 41 ± 10% at rest to 47 ± 12% at peak exercise (p < .001) as did the left ventricular ejection fraction.
fraction (55 ± 15% to 60 ± 12%, p < .05) (figure 4). For the group (n = 14), the magnitude and direction of change in right ventricular ejection fraction correlated with that of the left ventricle (r = .82, p < .02). The mean heart rate–blood pressure product during peak exercise was 25,356 ± 3363, which did not differ from the peak heart rate–blood pressure product during respiratory gas exchange. No patient had chest pain or ischemic ECG changes during either exercise test.

**Exercise respiratory gas exchange analysis.** The mean VO\textsubscript{2}max for the group was 18 ± 3 ml/kg/min and represented 73 ± 13% of the maximum predicted value. This lower range of VO\textsubscript{2}max can be attributed to early termination of exercise because of hypotension in seven patients and ventricular arrhythmias in one patient. In these patients, VO\textsubscript{2}max was not achieved because VO\textsubscript{2} increased greater than 1 ml/kg/min during the last minute of exercise.\textsuperscript{22}

**Correlation of exercise radionuclide ventriculography and respiratory gas exchange (table 2).** Eight of the 14 patients (Nos. 1 to 8) had a greater than 5 unit increase in right ventricular ejection fraction values from rest to peak exercise, and seven of these patients also demonstrated a similar increase in the left ventricular ejection fraction values. Three patients (Nos. 9 to 11) had a less than 5 unit increase in right ventricular ejection fraction, and their left ventricular ejection fraction response was also abnormal.

The remaining three patients (Nos. 12 to 14) had both an abnormal right ventricular ejection fraction response to exercise and a peak right ventricular ejection fraction of less than 40%, despite a normal left ventricular ejection fraction response in two patients. Two of these patients had evidence of pulmonary disease manifested by an abnormal ventilatory reserve and a decreased FE\textsubscript{V}, (28% and 48% predicted), while the remaining patient had a markedly depressed right ventricular ejection fraction of 20% that decreased with exercise. This patient required patch closure of a patent foramen ovale 48 hr after infarction to correct hypoxemia resulting from right-to-left shunting. All three of these patients had an anaerobic threshold.

**TABLE 2**

<table>
<thead>
<tr>
<th>Patient</th>
<th>RVEF</th>
<th>LVEF</th>
<th>VO\textsubscript{2}max (ml/kg/min; % predicted)</th>
<th>FEV\textsubscript{1} (liters; % predicted)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Peak</td>
<td>%</td>
<td>%</td>
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<tr>
<td>1</td>
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<td>14</td>
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<td>-1</td>
<td>43</td>
</tr>
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</table>

RVEF = right ventricular ejection fraction; LVEF = left ventricular ejection fraction; AT = anaerobic threshold; VR = ventilatory reserve.

\textsuperscript{A}Exercise study was stopped due to ventricular tachycardia.
predicted tary men, Hansen et al. \cite{16} demonstrated that all had an anaerobic threshold of 40% or more of their actual or predicted \( \text{VO}_2 \text{max} \) with a mean of 56% ± 8% for the group. In our study, the peak right ventricular ejection fraction correlated with the anaerobic threshold (\( r = 0.82, \ p < .02, \text{figure 5} \)), but the peak left ventricular ejection fraction did not (\( r = .41 \)). Because peak exercise was not achieved in all patients, the \( \text{VO}_2 \text{max} \) did not correlate with resting or peak ejection fraction values.

**Discussion**

**Short-term evaluation.** In this study, only patients who died early had over 10 mm summed ST segment depression in leads V1 to V4. Precordial ST segment elevation in V1 or V4 R has been reported as a marker of acute RVMI resulting from right ventricular injury recorded by the overlying unipolar electrode. \cite{23,24,25,26,27} Therefore a large magnitude of summed ST segment depression in the anterior precordial leads disproportionate to the amount of ST segment elevation in the inferior limb leads most likely indicates extensive left ventricular posterior and lateral wall ischemia. Furthermore, persistent ST segment depression in leads V1 to V4 24 hr after the initial ECG was found in the remaining patient who died in the 1 year follow-up, whereas no survivors demonstrated this finding. In our previous report, we found that this ECG finding was associated with a depressed left ventricular ejection fraction and increased mortality in the 1 year follow-up period in 43 consecutive patients with acute IMI and no prior history of myocardial infarction. \cite{13} Thus ECG predictors of a poor outcome were no different in the present study group, which included only patients with concomitant RVMI. In addition, acute hemodynamics in the non-survivors indicated severe left ventricular dysfunction with markedly depressed stroke volume and left ventricular stroke work indexes. Postmortem examinations also demonstrated extensive left ventricular necrosis, and in the 23 survivors who underwent early radionuclide ventriculography, the mean left ventricular ejection fraction was 56 ± 11%. These findings and the results of other series \cite{28,29,30,31} indicate that the early and 1 year mortality of RVMI is not dependent primarily on the severity of right ventricular dysfunction but rather is related to the coexistence of significant left ventricular myocardial infarction and mechanical complications of myocardial infarction, including ventricular septal defect and papillary muscle rupture or infarction.

**Follow-up evaluation**

**Resting radionuclide ventriculography.** In the survivors, right ventricular ejection fraction increased significantly (30 ± 7% to 43 ± 8%, \( \ p < .001 \)), largely due to an improvement in akinetic wall segments. Therefore, in this patient population with similar short-term hemodynamic findings of RVMI, global and regional right ventricular systolic performance improved significantly during long-term follow-up. This remarkable capacity of the right ventricle to regain systolic function after right coronary occlusion may be attributed to the rich system of thebesian veins providing a constant supply of venous oxygenated blood to the thin-walled right ventricle and the decreased myocardial oxygen demands resulting from the low-pressure pulmonary circuit. \cite{30,31} These factors may prevent the development of permanent damage until adequate left-to-right coronary collaterals are formed or until the right coronary artery is reperfused by intrinsic fibrinolysis or new bridging collaterals. \cite{34}

**Exercise radionuclide ventriculography.** The increase in right ventricular ejection fraction during exercise in this study contrasts with the results of other studies in which patients with proximal right coronary artery disease had either a decrease or no significant change in the right ventricular ejection fraction during exercise. \cite{19,20} However, the majority of patients in these studies had significant three-vessel coronary artery disease, and Berger et al. \cite{21} found that the right ventricular ejection fraction response to exercise was directly

![FIGURE 5. Relationship of peak exercise right ventricular ejection fraction (RVEF) to the anaerobic threshold in the 14 patients who underwent upright bicycle exercise with correlation coefficient (r) and 95% confidence intervals.](image-url)
related to the left ventricular ejection fraction response, as was found in our study. In addition, the ejection fraction response was not related to medical therapy at the time of exercise in our study. However, there was a mixed response with some patients failing to increase right ventricular ejection fraction appropriately despite a normal left ventricular ejection fraction response to exercise. The significance of these findings is better understood when evaluated in combination with the results of respiratory gas exchange.

Respiratory gas exchange analysis. The VO\textsubscript{2}max represents the maximum amount of oxygen that a subject can consume for a given form of exercise. In patients with cardiac disease, however, the delivery of oxygen is limited by failure of the cardiac output to increase commensurate with the needs of exercising skeletal muscle. Subsequently, there is a shift to anaerobic metabolism with production of lactic acid, which is buffered by the body’s bicarbonate pool. Because pH decreases and free CO\textsubscript{2} increases, ventilation is stimulated disproportionately to the VO\textsubscript{2} and the ventilatory equivalent for O\textsubscript{2} (VE/VO\textsubscript{2}) increases, thereby maintaining a stable pH by reducing the PACO\textsubscript{2}. At this time of isocapnic buffering, there is a nonlinear increase in the VCO\textsubscript{2} as the ventilatory equivalent for CO\textsubscript{2} (VE/VCO\textsubscript{2}) remains unchanged or decreases. Wasserman et al.\textsuperscript{18} and Weber et al.\textsuperscript{22} have shown that these changes in ventilatory kinetics correlate with increases in blood lactate levels, and this point in exercise has been referred to as the anaerobic threshold.

Numerous studies have demonstrated that the degree of PAWP elevation and the resting left ventricular ejection fraction are not predictive of exercise capacity in patients with congestive heart failure.\textsuperscript{35-40} However, Weber and associates demonstrated that peak exercise cardiac index correlated with VO\textsubscript{2}max achieved during treadmill exercise. This suggests that aerobic capacity is limited by a failure to augment stroke output that may result from a limited preload or impaired contractile function during exercise. It is of interest that Baker et al.\textsuperscript{41} found a linear correlation between the resting right ventricular ejection fraction and VO\textsubscript{2}max in patients with ischemic and nonischemic cardiomyopathies, whereas the resting left ventricular ejection fraction did not correlate with exercise capacity. These results suggest that right ventricular function may be an important determinant of augmentation of left ventricular filling and cardiac output during exercise.

Correlation of respiratory gas exchange and radionuclide ventriculography. Because only seven of 14 patients reached VO\textsubscript{2}max, this exercise variable did not correlate with ejection fraction values. Nonetheless, the results of this study are similar to those of Baker and co-workers in that peak exercise right ventricular ejection fraction correlated with the anaerobic threshold while the left ventricular ejection fraction did not. The combined results of exercise radionuclide ventriculography and gas exchange suggest that early anaerobic metabolism resulted from a failure of the right ventricle to adequately increase its stroke output during exercise. Although simultaneous right heart pressures were not obtained, increased right ventricular afterload is the most likely determinant of exercise ejection fraction. In the present study right ventricular function was normal or only slightly decreased during exercise because the majority of our patients did not have significant pulmonary disease or left ventricular dysfunction. Therefore, in the absence of these problems, right ventricular systolic function and functional capacity can be expected to be normal in the recovery phase of RVMI, and this was reflected by the extremely favorable clinical status of our patients at follow-up.

Conclusions. The results of this study indicate that patients presenting with hemodynamically important RVMI and hemodynamic or ECG markers of left ventricular dysfunction or ischemia are at higher risk for mortality. Patients not demonstrating such findings have an excellent long-term survival and have significant improvement in right ventricular systolic function. Furthermore, functional aerobic capacity is normal because right ventricular systolic performance is preserved during exercise in patients who do not have pulmonary hypertension secondary to pulmonary disease or significant left ventricular dysfunction.

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