Right Ventricular Dysfunction Detected by Gated Scintigraphy in Patients with Acute Inferior Myocardial Infarction

By Pierre Rigo, M.D., Malcolm Murray, M.D., Dean R. Taylor, M.D., Myron L. Weisfeldt, M.D., David T. Kelly, M.D., H. W. Strauss, M.D., and Bertram Pitt, M.D.

SUMMARY

Twenty-seven patients with acute myocardial infarction not complicated by cardiogenic shock and ten normal volunteers were studied with gated cardiac blood pool scans. The ratio of right ventricular area/left ventricular area (RVA/LVA) determined from the left anterior oblique end-diastolic scans was examined. The ratio was 1.11 ± .06 in the normal volunteers. In patients with anterior infarction the ratio fell to 0.75 ± .12 (P < .05) due to left ventricular enlargement. In those with inferior infarction the ratio was 1.12 ± .23 which was greater than in those with anterior infarction (P < .05) due to enlargement of both the left and right ventricles. Six patients with cardiogenic shock, three with inferior and three with anterior infarction were studied. The three with anterior infarction had left ventricular enlargement and a decrease in the ratio of RVA/LVA to 0.62 while the three with inferior infarction had an increase in the ratio to 2.05 suggesting right ventricular dilatation and dysfunction. These studies suggest a high incidence of right ventricular dysfunction in patients with inferior myocardial infarction.

VENTRICULAR DYSFUNCTION occurring as a result of acute myocardial infarction is usually thought to be due to left ventricular damage. At autopsy, however, concomitant right ventricular infarction is frequently seen.1–3 Recently a characteristic syndrome of low output circulatory failure attributable to right ventricular dysfunction in patients with inferior myocardial infarction has been recognized.4 In this study, gated cardiac blood pool scans were used5–8 to determine the incidence of right ventricular dysfunction in patients with acute myocardial infarction.

Methods

Thirty-three patients admitted to The Johns Hopkins Hospital Coronary Care Unit with symptoms and signs suggestive of acute myocardial infarction were studied. There were 19 males and 14 females with a mean age of 58 years. Seventeen patients had electrocardiographic evidence of inferior myocardial infarction, 15 of anterior myocardial infarction and one of an old anterior and an acute subendocardial infarction. All patients had serum enzyme evidence of acute myocardial infarction.

On admission to the Coronary Care Unit patients were clinically classified:9 class I — uncomplicated myocardial infarction; class II — evidence of mild to moderate left ventricular failure; class III — severe left ventricular failure or pulmonary edema; class IV — cardiogenic shock.

Hemodynamic studies were performed in the Coronary Care Unit as follows: a #5 or 7 Swan Ganz catheter was inserted percutaneously or by cutdown into an antecubital vein and floated into the pulmonary artery under fluoroscopic guidance. Left ventricular filling pressure was measured from the pulmonary artery occluded pressure. A short plastic catheter was placed percutaneously or by cutdown into the radial artery. Mean systolic pressure was determined by planimetric integration of the systolic arterial pressure. All pressures were measured, using the midsternal position as zero reference point and recorded employing a pressure transducer. Cardiac output was determined in duplicate by the indicator dilution technique using either indocyanine green or room temperature normal saline. Indocyanine green was injected into the pulmonary artery with sampling from the radial artery through a densitometer at a rate of 38.2 ml/min and the blood was re-infused. Dynamic on-line calibration was used.10 Room temperature saline was injected into the proximal lumen of the #7 Swan Ganz catheter and the temperature recorded with a distal thermistor.11 The thermodilution curves were analyzed using an analog computer. Stroke work index was calculated as:

\[
\text{MSP} \times \text{SI} \times 13.6 = 1000
\]

where MSP = mean systolic pressure (mm Hg), SI = stroke
volume index (ml/m²), and LVFP = left ventricular filling pressure (mm Hg).

Gated cardiac blood pool scans were performed within two hours of the hemodynamic studies. The biplane technique for measuring left ventricular volume, ejection fraction and extent of akinesis has been described previously. An index of right ventricular end-diastolic volume was obtained by calculating the area of the right ventricle in the left anterior oblique (LAO) projection and comparing it to that of the left ventricle in the same projection. The plane of the mitral and aortic valves was arbitrarily drawn as a straight line from the upper limit of the septum to the upper extent of the lateral wall. This line was perpendicular to the long axis of the ventricle in the LAO projection. The plane of the pulmonary valve was traced as a straight line perpendicular to the right ventricular outflow tract. The level of this line was adjusted so that it would cross the plane of the aortic and mitral valves at the level of the septum. Patients in whom the right ventricular contours were inadequately visualized due to superimposition of the right ventricular and hepatic blood pool were excluded from this study. Examples of left anterior oblique scans in a normal patient, a patient with an inferior myocardial infarction and right ventricular dilatation, a patient with an anterior myocardial infarction and isolated left ventricular enlargement, and a patient with biventricular enlargement are shown in figure 1.

Left ventricular volumes and ejection fraction were measured as previously described from the biplane images. In three patients with massive right ventricular enlargement evaluation of the left ventricular volume was not possible by the biplane technique because of obliteration of the margins of the left ventricle by the right ventricle.

Figure 1

Gated cardiac blood pool scans in the left anterior oblique projection in a normal patient; a patient with right ventricular dilatation secondary to an inferior myocardial infarction; a patient with left ventricular enlargement secondary to an anterior infarct; and a patient with biventricular enlargement. Line drawings of the original scans are shown for comparison. RV = right ventricle; LV = left ventricle; Ao = aorta; Pa = pulmonary artery.
in the right anterior oblique projection. In these three patients the ejection fraction was calculated from the LAO projection alone using the single plane area-length formula.

Ten normal volunteers were studied by gated cardiac blood pool scanning to determine the normal left ventricular ejection fraction and ratio of right ventricular/left ventricular area in the LAO projection. Statistical significance of the data was analyzed using the Student's t-test.

Results

The clinical, hemodynamic, and scintiphotographic data obtained in 14 patients with inferior myocardial infarction and 13 patients with anterior myocardial infarction, not in cardiogenic shock, are shown in tables 1–3. The age, sex, and racial distribution of those with inferior myocardial infarction was similar to those with anterior myocardial infarction. Incidence of previous myocardial infarction or hypertension was also similar. Two of the patients with anterior myocardial infarction had a previous history of chronic obstructive pulmonary disease. A murmur suggestive of papillary muscle dysfunction was noted in one patient with an inferior myocardial infarction and three with an anterior myocardial infarction (table 1). None had a previous history of mitral regurgitation. Eleven patients with an inferior myocardial infarction were in clinical class I, two in clinical class II, and one in clinical class III. Six patients with an anterior myocardial infarction were in clinical class I, six in clinical class II and one in clinical class III.

Systemic arterial pressure, cardiac index, right ventricular systolic pressure, right atrial mean pressure and heart rate were similar in both groups. Those with an anterior myocardial infarction had, however, significantly elevated left ventricular filling pressures and pulmonary artery mean pressures compared to those with inferior myocardial infarction (table 2).

Table 1

Clinical Characteristics of Patients Without Shock

<table>
<thead>
<tr>
<th></th>
<th>Inferior MI</th>
<th>Anterior MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>53.4</td>
<td>60</td>
</tr>
<tr>
<td>Sex: Male</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Female</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>White</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Black</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Previous MI</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Previous hypertension</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>COPD</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>MR</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>1° or 2° A-V block</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>IVCD</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

Abbreviations: MI: myocardial infarction; COPD: chronic obstructive pulmonary disease; IVCD: intraventricular conduction defect; MR: mitral regurgitation.

Table 2

Hemodynamic Data in Patients Without Shock

<table>
<thead>
<tr>
<th></th>
<th>Anterior MI</th>
<th>Inferior MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>81 ± 18.4*</td>
<td>72 ± 15.8 NS</td>
</tr>
<tr>
<td>RAM (mm Hg)</td>
<td>6.75 ± 3.8</td>
<td>5.9 ± 3.3 NS</td>
</tr>
<tr>
<td>PA mean (mm Hg)</td>
<td>24.4 ± 8.1</td>
<td>18.4 ± 5.3 P &lt; 0.05</td>
</tr>
<tr>
<td>LVFP (mm Hg)</td>
<td>17.9 ± 7.9</td>
<td>11.9 ± 4.7 P &lt; 0.05</td>
</tr>
<tr>
<td>Ao mean (mm Hg)</td>
<td>97.6 ± 13.8</td>
<td>101 ± 21 NS</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>2.65 ± 0.7</td>
<td>3.1 ± 0.6 NS</td>
</tr>
</tbody>
</table>

*Mean ± 1 standard deviation.

Abbreviations: RAM: right atrial mean pressure; PA: pulmonary artery pressure; LVFP: left ventricular filling pressure; Ao mean: mean aortic pressure; CI: cardiac index; NS: not statistically significant.

Scintiphotographic Studies (table 3)

Compared to the normal subjects both those with inferior and those with anterior myocardial infarction had a significant increase in left ventricular end-diastolic volume and decrease in left ventricular ejection fraction by the biplane gated cardiac blood pool scan technique. The area of the left ventricle normalized for body surface area, calculated from the left anterior oblique scan, was significantly increased compared to normal in both groups. There was a strong correlation between the left ventricular end-diastolic volume calculated from the biplane images and the left ventricular area obtained from the left anterior oblique scans, r = .78, suggesting that the ventricular area could be used as an index of ventricular volume.

Combining the results of the biplane calculation of the left ventricular end-diastolic volume with that of the ratio of the ventricular areas in the left anterior oblique projection one can suggest that six of the 14 patients with inferior myocardial infarction not complicated by shock had right ventricular enlargement. The right ventricular area normalized for body surface area was significantly greater (P < .05) in the 14 patients with inferior myocardial infarction than in the 13 patients with anterior infarction.

Table 3

Gated Cardiac Blood Pool Scans in Normal Volunteers and in Patients Without Shock

<table>
<thead>
<tr>
<th></th>
<th>Normal (10)</th>
<th>Anterior MI (13)</th>
<th>Inferior MI (14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF (%)</td>
<td>57.2 ± 5*</td>
<td>37.1 ± 9†</td>
<td>38.9 ± 7.5†</td>
</tr>
<tr>
<td>LVEDV (mL/m²)</td>
<td>74.2 ± 11.8</td>
<td>125.6 ± 35.6†</td>
<td>114.2 ± 3.6†</td>
</tr>
<tr>
<td>LVA (cm²/m²)</td>
<td>13.2 ± 1.9</td>
<td>18.2 ± 3.7†</td>
<td>15.7 ± 3.6†</td>
</tr>
<tr>
<td>RVA (cm²/m²)</td>
<td>14.6 ± 2.6</td>
<td>13.5 ± 3.3</td>
<td>17.0 ± 3.9†</td>
</tr>
<tr>
<td>RVA/LVA</td>
<td>1.11 ± .96</td>
<td>.75 ± .12†</td>
<td>1.12 ± .23†</td>
</tr>
</tbody>
</table>

*Mean ± 1 standard deviation.
† = different from normal P < 0.05.
‡ = different from anterior MI P < 0.05.

Abbreviations: LVEF: left ventricular ejection fraction; LVEDV: left ventricular end diastolic volume; LVA: left ventricular area; RVA: right ventricular area.

Circulation, Volume 52, August 1975
Six patients were in cardiogenic shock. The results of the gated blood pool scans in these patients are presented in table 4. Patients 1, 2 and 3 had inferior myocardial infarctions with RV enlargement. Patient 4 had evidence of an old anterior myocardial infarction and evidence of an acute subendocardial infarction, while patients 5 and 6 had acute anterior myocardial infarctions with isolated left ventricular dysfunction.

The clinical and hemodynamic data of the three patients with associated right ventricular dysfunction are presented in table 5 and in the following case descriptions.

Case 1

A 56-year-old white male was admitted to a neighboring hospital with electrocardiographic evidence of acute inferior myocardial infarction. His hospital course was complicated by bibasilar pulmonary rales for which he received intravenous diuretics. Several episodes of ventricular tachycardia and an episode of ventricular fibrillation occurred requiring cardioversion. Third degree heart block also occurred requiring insertion of a transvenous pacemaker. He developed cardiogenic shock which failed to respond to ventricular pacing and an infusion of norepinephrine was begun. At the end of the second hospital day he was transferred to The Johns Hopkins Hospital. On admission the patient was in cardiogenic shock and he had rales at the bases of his lungs. Hemodynamic evaluation (table 5) suggested inadequate plasma volume. Volume loading with plasmanate was begun and an attempt made to reduce the dose of norepinephrine. These measures failed and intra-aortic balloon counterpulsation was begun. A gated cardiac blood pool scan was performed (table 4) and revealed a left ventricle of approximately normal size with an ejection fraction of 41% with a massively dilated right ventricle. The patient died after three days of persistent shock. Postmortem examination revealed complete obstruction of the proximal right coronary artery and an acute infarction involving the posterolateral wall of the left ventricle, the posterior portion of the septum, almost the entire free wall of the right ventricle and the right atrium. The right ventricular cavity was dilated while the left ventricular cavity was of normal size. The left ventricle was hypertrophied (figs. 2 and 3).

Case 2

A 72-year-old white female was admitted to The Johns Hopkins Hospital Coronary Care Unit with

**Table 4**

Gated Cardiac Blood Pool Scans in Patients with Shock

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>RVA/LVA (cm/m²)</th>
<th>RVA (cm/m²)</th>
<th>LVA (cm/m²)</th>
<th>LVEDV Biplane (ml/m²)</th>
<th>LVEF Biplane</th>
<th>LAO</th>
<th>LVFP</th>
<th>PA</th>
<th>Ao</th>
<th>CI</th>
<th>ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56</td>
<td>M</td>
<td>2.3</td>
<td>22</td>
<td>9.3</td>
<td>-</td>
<td>41%</td>
<td>-</td>
<td>-</td>
<td>8/5/10/(6)</td>
<td>98/60</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>F</td>
<td>2.4</td>
<td>22.2</td>
<td>9.1</td>
<td>-</td>
<td>50%</td>
<td>-</td>
<td>-</td>
<td>18/10/(15)</td>
<td>76/50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>59</td>
<td>F</td>
<td>1.45</td>
<td>24.5</td>
<td>17</td>
<td>154</td>
<td>23% (+MR)</td>
<td>Old anterior MI, acute subendocardial MI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>63</td>
<td>F</td>
<td>.63</td>
<td>14.2</td>
<td>22.5</td>
<td>132</td>
<td>23% (+MR)</td>
<td>Acute anterior MI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>66</td>
<td>F</td>
<td>11.5</td>
<td>21.8</td>
<td>157</td>
<td>-</td>
<td>20%</td>
<td>Acute anterior MI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>66</td>
<td>F</td>
<td>.70</td>
<td>13</td>
<td>18.5</td>
<td>150</td>
<td>32% (+MR)</td>
<td>Acute anterior MI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** RVA: right ventricular area in LAO projection; LVA: left ventricular area in LAO projection; LVEDV: left ventricular end-diastolic volume; LAO: left anterior oblique; MR: mitral regurgitation; MI: myocardial infarction; LVEF: left ventricular ejection fraction.

**Table 5**

Clinical and Hemodynamic Data in Patients with Right Ventricular Dilatation and Shock

<table>
<thead>
<tr>
<th>Patient</th>
<th>Clinical class</th>
<th>JVD</th>
<th>Rales</th>
<th>RAM (mm Hg)</th>
<th>LVFP (mm Hg)</th>
<th>PA (mm Hg)</th>
<th>Ao (mm Hg)</th>
<th>CI (L/min/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Initial study*</td>
<td>IV</td>
<td>+</td>
<td>4</td>
<td>5</td>
<td>8/5/10/(6)</td>
<td>98/60</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>After volume expansion</td>
<td>+</td>
<td>+</td>
<td>15</td>
<td>10</td>
<td>18/10/(15)</td>
<td>76/50</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>After volume expansion</td>
<td>+</td>
<td>+</td>
<td>9</td>
<td>13</td>
<td>24/10/(13)</td>
<td>90/60</td>
<td></td>
</tr>
</tbody>
</table>

*Initial hemodynamic study performed after institution of pressor therapy.

Abbreviations: RAM: right atrial mean pressure; LVFP: left ventricular filling pressure; PA: pulmonary artery pressure; Ao: aortic pressure; CI: cardiac index; JVD: jugular venous distention.
electrocardiographic evidence of an acute inferior myocardial infarction, third degree heart block, and cardiogenic shock. Her neck veins were distended on admission and her chest clear to auscultation and percussion. A transvenous temporary pacemaker was inserted which resulted in some clinical improvement but failed to improve the signs of cardiogenic shock. Hemodynamic study (table 5) revealed a moderately elevated right ventricular end-diastolic pressure and a normal left ventricular filling pressure. The gated cardiac blood pool scan (table 4) revealed a massively dilated right ventricle with a normal sized left ventricle with an ejection fraction of 50%. Volume loading with 250 ml of plasmanate increased the right atrial pressure above the left ventricular filling pressure. The patient improved clinically and survived hospitalization. At follow-up two months after the acute myocardial infarction the patient had no clinical evidence of right ventricular failure and a gated cardiac blood pool scan revealed reduction in the right ventricular size.

Case 3

A 59-year-old white female with a past history of systemic hypertension and angina pectoris was admitted to a neighboring hospital because of unstable angina pectoris. On the first hospital day she had an acute inferolateral myocardial infarction complicated by ventricular fibrillation which responded to cardioversion. On the fourth hospital day she developed cardiogenic shock. An infusion of aramine was begun and she was transferred to The Johns Hopkins Coronary Care Unit. On admission her neck veins were distended and she had bibasilar pulmonary rales. Initial hemodynamic studies (table 5) revealed elevated right ventricular and left ventricular filling pressures. Scintigraphic studies (table 4) revealed dilatation of the right and left ventricle with a left ventricular ejection fraction of 24%. The patient slowly improved over the next few days on digoxin and diuretic therapy, but had persistent signs of left ventricular failure. A follow-up gated cardiac blood pool scan two months after myocardial infarction revealed persistent left ventricular enlargement with partial regression of right ventricular enlargement.

In contrast to patients 1, 2 and 3 in cardiogenic shock complicating their inferior myocardial infarction, patients 4, 5 and 6 did not have right ventricular dilatation but only left ventricular dysfunction as manifested by left ventricular enlargement and a reduction of their left ventricular ejection fraction.

In addition to the three patients with inferior myocardial infarction and cardiogenic shock described above, one of the 14 patients with inferior myocardial infarction without cardiogenic shock in

Figure 2

Transverse section of the heart of patient 1 with massive right ventricular and posterior-septal myocardial infarction. The entire right ventricle except the infundibular area is infarcted beginning at the arrow and extending to the posterior left ventricle.

Figure 3

A photomicrograph of the infarcted right ventricle of patient 1 shown in figure 2. The subendocardial muscle on the left is spared and the remainder is infarcted. (Hematoxylin and eosin stain, × 80)
RIGHT VENTRICULAR MYOCARDIAL INFARCTION

whom the gated blood pool scan revealed marked right ventricular dilatation is described below.

Case 7

A 41-year-old white male was admitted to The Johns Hopkins Hospital Coronary Care Unit with an uncomplicated inferior myocardial infarction. His initial hospital course was complicated by an episode of first and second degree heart block of the Wenckebach type. His jugular veins were mildly distended and his chest clear to auscultation and percussion. His hemodynamic studies included normal right and left ventricular filling pressures and cardiac output. Gated cardiac blood pool scan revealed marked right ventricular dilatation with a left ventricle of approximately normal size and an ejection fraction of 41%. Repeat hemodynamic studies after the patient received about one liter of normal saline over a 12 hour period revealed an increase of both the right and left ventricular filling pressure up to 13 mm Hg.

Discussion

The results of this study suggest a high incidence of right ventricular enlargement in patients with inferior myocardial infarction, as indicated by the significantly greater ratio of right ventricular to left ventricular end-diastolic area on the left anterior oblique gated blood pool scan in patients with inferior infarction compared to those with anterior infarction despite a similar left ventricular end-diastolic volume. The relatively high incidence of right ventricular dysfunction in patients with inferior myocardial infarction detected in this study is supported by the finding of right ventricular scarring in up to 40% of patients with inferior myocardial infarction at autopsy1-3 and of an elevated right ventricular filling pressure in relation to the left ventricular filling pressure in the present and previous studies in these patients.4 One of our patients (patient 7), with inferior myocardial infarction uncomplicated by cardiogenic shock, had marked right ventricular dilatation but would not have been recognized by pressure measurement alone since he had normal right and left ventricular filling pressures and a normal cardiac output. Only after volume loading did the right ventricular filling pressure rise to the level of the left ventricular filling pressure (13 mm Hg) suggesting right ventricular dysfunction. This patient may be a link between those asymptomatic patients with mild to moderate right ventricular dilatation and the patients with the clinical syndrome of "right ventricular infarction" described by Cohn et al.4 and exemplified by two patients in cardiogenic shock with right ventricular dilatation in the present study.

Two of our three patients with inferior myocardial infarction complicated by cardiogenic shock, one of whom died and had autopsy evidence of a massive right ventricular infarction, had marked right ventricular dilatation with a normal sized left ventricle. The third patient had enlargement of both ventricles as well as evidence of congestive heart failure. In contrast, the two patients with anterior myocardial infarction complicated by shock and one patient with an acute subendocardial and a previous anterior infarction had only left ventricular enlargement. Patients 1 and 2 presented with the clinical syndrome of right ventricular infarction characterized by electrocardiographic evidence of inferior infarction, cardiogenic shock and heart block. Patient 3 also had hemodynamic evidence of right ventricular infarction, in that the right ventricular filling pressure was elevated in relation to the left ventricular filling pressure. The initial hemodynamic findings in patient 1 were, however, not characteristic of right ventricular infarction in that both the right and left ventricular filling pressures were not elevated, possibly as a result of previous diuretic therapy. An increased right ventricular filling pressure became apparent only after volume loading. The relatively low right ventricular filling pressure in this patient in the face of evidence of massive right ventricular dilatation suggests an increased right ventricular compliance.

A similar syndrome has been described following surgery in patients with tetralogy of Fallot.12 These patients have depressed right ventricular function due to right ventriculotomy. In the presence of a diminutive left ventricle and a high left ventricular filling pressure a low output syndrome may occur in the early postoperative period.

On the basis of the observations in the present study and in recent studies6, 13, 14 the clinical spectrum of right ventricular dysfunction in patients with inferior myocardial infarction can be described. As in animal studies and in some patients with congenital heart disease, isolated right ventricular dysfunction does not result in acute deterioration of cardiac output or systemic pressure nor in signs of right ventricular failure as long as the left ventricular filling pressure and pulmonary vascular resistance remain normal.4 When the left ventricular filling pressure increases as a result of left ventricular failure and/or decreased compliance, the relative impairment of left ventricular filling and right ventricular function will determine the patient's presentation. Mild to moderate right ventricular dysfunction may result in elevation of the right ventricular filling pressure and jugular venous distension. In some patients, however, this may only be manifest after plasma volume expansion. Severe right ventricular dysfunction may lead to a low output syndrome or cardiogenic shock, unless the loss of the
systolic contribution of right and left ventricular filling is compensated for by an elevation of systemic venous pressure. The therapy of the syndrome of right ventricular dysfunction should therefore be directed toward maintaining adequate left ventricular filling. This can be achieved by increasing right atrial filling pressure to a level high enough to maintain adequate left ventricular filling regardless of initial right ventricular end-diastolic pressure. Decreasing left ventricular filling pressure by reducing left ventricular afterload may also be helpful under these circumstances.

Since cardiogenic shock due to isolated left ventricular failure is almost invariably fatal, we would suggest a therapeutic trial of volume loading in all patients with inferior myocardial infarction complicated by cardiogenic shock with careful monitoring of left ventricular filling pressure so as to avoid increasing left ventricular filling pressure over 22 mm Hg and causing pulmonary edema. If future studies reveal right ventricular infarction to be a frequent occurrence in patients with inferior myocardial infarction complicated by cardiogenic shock, the diagnostic studies outlined in this report will achieve increased importance since shock associated with right ventricular infarction is a potentially reversible situation.

References

11. FORSTER M, GANT W, DIAMOND G, MC HUGH T, CHIONETTE DW, SWAN HJC: Thermodilution cardiac output determination with a single flow directed catheter. Am Heart J 83: 906, 1972
Right ventricular dysfunction detected by gated scintiphotography in patients with acute inferior myocardial infarction.
P Rigo, M Murray, D R Taylor, M L Weisfeldt, D T Kelly, H W Strauss and B Pitt

*Circulation.* 1975;52:268-274
doi: 10.1161/01.CIR.52.2.268

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/52/2/268

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Circulation* is online at:
http://circ.ahajournals.org//subscriptions/