Role of right ventricular infarction in cardiogenic shock associated with inferior myocardial infarction

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SUMMARY In order to assess the role of right ventricular and left ventricular dysfunction in patients with cardiogenic shock associated with combined right ventricular and inferior myocardial infarction, we reviewed the clinical records and haemodynamic data of 18 patients with inferior myocardial infarction and cardiogenic shock. Six of these patients had haemodynamic evidence of right ventricular dysfunction. There was no significant difference in left ventricular stroke work index between patients with $18 \pm 3$ g m per m$^2$ and without $14 \pm 2$ associated right ventricular dysfunction. Though pulmonary artery wedge mean pressure was significantly lower in patients with right ventricular dysfunction ($10 \pm 1$ mmHg) than in those without right ventricular dysfunction ($18 \pm 2$), this difference is probably the consequence of the right ventricular damage, since all patients with right ventricular dysfunction had a wedge pressure $11$ mmHg or less compared with only three of 12 patients without right ventricular impairment. Necropsy observations in eight patients including two with right ventricular dysfunction support this concept. All patients had $40$ per cent or more infarction (old plus new) of the left ventricle. However, the two with right ventricular infarction had wedge pressures $10$ mmHg or less whereas the other six without right ventricular infarction all had wedge pressure $14$ mmHg or more. In addition, while all five patients with right ventricular dysfunction who were treated by plasma volume expansion had a rise in wedge pressure ($16 \pm 1$ after fluid therapy) only two survived. Mortality in the group with right ventricular dysfunction (three of six) was similar to that in the group without right ventricular dysfunction (seven of 12). We conclude that in patients with cardiogenic shock associated with combined right ventricular and inferior myocardial infarction, (1) low pulmonary artery wedge pressure is the result primarily of right ventricular dysfunction and does not necessarily imply that left ventricular damage is slight and (2) severe left ventricular damage, often with a relatively low wedge pressure, is more common than has been previously reported in these patients.

Recently, a number of investigators (Rigo et al., 1975; Sharpe et al., 1978; Tobinick et al., 1978) have drawn attention to the frequency with which inferior myocardial infarction is complicated by right ventricular infarction. The typical haemodynamic feature of this entity is a raised mean right atrial pressure which equals or exceeds mean pulmonary artery wedge pressure (Cohn et al., 1974; Rotman et al., 1974). Furthermore, some authors (Cohn et al., 1974; Rigo et al., 1975) have reported data from a few patients suggesting that

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of right ventricular and left ventricular dysfunction in the pathogenesis of cardiogenic shock associated with combined right ventricular and inferior myocardial infarction.

**Patients and methods**

**PATIENT SELECTION**

The computer files of all patients admitted to the MGH MIRU-ISA* for haemodynamic monitoring between January 1969 and July 1977 were searched for patients with a diagnosis of cardiogenic shock in association with inferior myocardial infarction. The diagnosis of cardiogenic shock was based on previously published clinical and haemodynamic criteria (Dunkman et al., 1972; Leinbach et al., 1973). Patients with shock resulting from mechanical lesions (ventricular septal defect, acute mitral regurgitation, tamponade, or cardiac rupture) or rhythm disorders were excluded. Twenty-four patients were identified in the category of inferior myocardial infarction with cardiogenic shock of whom 18 had complete records available for analysis. Six of the 18 patients had haemodynamic evidence of right ventricular dysfunction with a raised right atrial mean pressure equal to or greater than pulmonary artery wedge mean pressure and a right ventricular stroke work index which was less than 2.0 g m per m\(^2\). The records of all 18 patients were reviewed and pertinent clinical, haemodynamic, and electrocardiographic data abstracted and entered on standard forms designed for this study. When available, data from cardiac catheterisation, including coronary arteriography and left ventriculography, and post-mortem examination were also reviewed and included for each patient.

A second group of 27 patients with acute inferior myocardial infarction without shock was also assembled for comparison with the group with inferior myocardial infarction with shock. These patients were selected in the same fashion as the previously described group. All patients in this category had haemodynamic monitoring and complete records were available for analysis in 17 of them. Haemodynamic monitoring was done for one of two reasons: (A) as part of an infarct size reduction study; or (B) to assess treatment for recurrent ischaemia in patients with acute inferior myocardial infarction.

**HAEMODYNAMIC DATA**

Haemodynamic monitoring was initiated in all patients soon after admission to the MIRU-ISA using previously described techniques (Dunkman et al., 1972). Cardiac output was determined by dye dilution or thermodilution method. In addition to directly measured pressures on the right and left sides of the heart, the following haemodynamic indices were derived using the following formulæ: (A) Cardiac index (CI) = cardiac output/BSA (ml per m\(^2\))

(B) Stroke volume index (SVI) = stroke volume/BSA (ml per m\(^2\))

(C) Left ventricular stroke work index (LVSWI) = [systemic arterial mean pressure (MAP) – pulmonary artery wedge mean pressure (PAWP)] x SI x 0.0136 (g m per m\(^2\))

(D) Right ventricular stroke work index (RVSWI) = [pulmonary artery mean pressure (PA) – mean right atrial mean pressure (RA)] x SI x 0.0136 (g m per m\(^2\))

(E) Systemic vascular resistance (SVR) = [(MAP – RA)/cardiac output] x 80 (dynes s cm\(^{-2}\))

(F) Pulmonary vascular resistance (PVR) = [(PA – PAWP)/cardiac output] x 80 (dynes s cm\(^{-2}\)).

**STATISTICAL ANALYSIS**

The group means for continuous variables were assessed for significant differences using a computer programme for one way analysis of variance and the Newman-Keuls procedure for multiple comparisons (Snedecor and Cochran, 1967). Discontinuous variables were assessed with the aid of another computer programme for Fischer’s exact test. All data are presented as mean ± standard error of the mean (SEM).

* Massachusetts General Hospital, Myocardial Infarction Research Unit, Intensive Study Area.

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**Table 1 Clinical, electrocardiographic, and coronary arteriographic data in patients with inferior myocardial infarction**

<table>
<thead>
<tr>
<th></th>
<th>No. of patients</th>
<th>Mean age (range)</th>
<th>History of left ventricular failure</th>
<th>Previous anterior myocardial infarction</th>
<th>Triple vessel coronary artery disease</th>
<th>Hospital mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock patients</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With RV dysfunction</td>
<td>6</td>
<td>59 (44-70)*</td>
<td>1*</td>
<td>1*</td>
<td>4/4†</td>
<td>3†</td>
</tr>
<tr>
<td>Without RV dysfunction</td>
<td>12</td>
<td>54 (45-66)</td>
<td>3</td>
<td>5</td>
<td>7/8</td>
<td>7</td>
</tr>
<tr>
<td>Patients without shock</td>
<td>17</td>
<td>44 (28-64)</td>
<td>0</td>
<td>4</td>
<td>3/12‡</td>
<td>1**</td>
</tr>
</tbody>
</table>

*P = NS vs other groups. †P = NS vs shock patients without right ventricular dysfunction. ‡P < 0.02 vs shock patients with or without right ventricular dysfunction. *P < 0.05 vs shock patients with right ventricular dysfunction; and P < 0.01 vs shock patients without right dysfunction.
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Results

CLINICAL, ELECTROCARDIOGRAPHIC, AND CORONARY ARTERIOGRAPHIC DATA

The data for patients with cardiogenic shock both with and without right ventricular dysfunction and for patients without shock are summarised in Table 1. Mean age, past history of left ventricular failure, and electrocardiographic evidence of previous anterior myocardial infarction did not differ significantly in the three groups. The extent of coronary artery disease (defined as reduction in luminal diameter of 50% or more) was known for 12 patients with shock (eight without right ventricular dysfunction and four with right ventricular dysfunction) and 12 patients without shock. In patients with shock, the incidence of triple vessel coronary artery disease was comparable in those patients with and without right ventricular dysfunction but triple vessel disease was significantly less frequent in the patients without shock. Likewise, hospital mortality was comparable in the two groups of patients with cardiogenic shock.

Patients with shock and right ventricular dysfunction had by definition significantly higher right atrial mean pressure than all other patients. In addition, pulmonary artery and pulmonary artery wedge mean pressures and right ventricular stroke work index were significantly lower in patients with shock and right ventricular dysfunction than in patients with shock but without haemodynamic evidence of right ventricular dysfunction. Furthermore, all patients with right ventricular dysfunction had a wedge pressure 11 mmHg or less compared with only three of 12 patients with shock but without right ventricular dysfunction (P < 0.01). However, there was no significant difference in left ventricular filling pressure between the six patients with right ventricular dysfunction and the 17 patients without shock. Depression of left ventricular function as assessed by left ventricular stroke work index in patients with shock was similar in those with and without right ventricular dysfunction even though pulmonary artery wedge pressure was significantly lower in the group with right ventricular dysfunction. Systemic vascular resistance was similar in all groups. There was no significant difference in pulmonary vascular resistance between patients without shock and those with shock and right ventricular dysfunction.

Table 2  Haemodynamic data in patients with inferior myocardial infarction

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Right atrium (mean)</th>
<th>Pulmonary artery (mean)</th>
<th>Pulmonary artery wedge (mean)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>15</td>
<td>10</td>
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<tr>
<td>2</td>
<td>15</td>
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<td>40</td>
<td>27</td>
</tr>
<tr>
<td>18</td>
<td>7</td>
<td>31</td>
<td>26</td>
</tr>
</tbody>
</table>

HAEMODYNAMIC DATA ON ADMISSION

Data for patients with cardiogenic shock with and without right ventricular dysfunction are shown in Table 2. These data were obtained within 24 to 48 hours of the onset of circulatory failure in all patients except one in whom data were obtained approximately 72 hours after the onset of clinical shock. Mean data for the two groups of patients with cardiogenic shock and for the group of patients without shock are given in Table 3.

RESPONSE TO PLASMA VOLUME EXPANSION

Five of six patients with shock and right ventricular dysfunction were treated initially by plasma volume expansion with salt poor albumin or normal saline, in addition to catecholamine infusions. In three
Table 3  Group mean (±SEM) haemodynamic data

<table>
<thead>
<tr>
<th></th>
<th>Pressure (mmHg)</th>
<th>Stroke work index (g m per m²)</th>
<th>Resistance (dynes s cm⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right atrium (mean)</td>
<td>Pulmonary artery (mean)</td>
<td>Pulmonary artery wedge (mean)</td>
</tr>
<tr>
<td>Cardiogenic shock patients</td>
<td>With right ventricular dysfunction</td>
<td>14 ± 2*</td>
<td>16 ± 1†</td>
</tr>
<tr>
<td></td>
<td>Without right ventricular dysfunction</td>
<td>8 ± 1</td>
<td>25 ± 3</td>
</tr>
<tr>
<td>Patients with inferior myocardial infarction without cardiogenic shock</td>
<td>5 ± 1</td>
<td>19 ± 2</td>
<td>12 ± 1</td>
</tr>
</tbody>
</table>

* P < 0.01 vs other groups. † P < 0.03 vs shock patients without right ventricular dysfunction; and P = NS vs patients without shock. ‡ P < 0.01 vs shock patients without right ventricular dysfunction; and P = NS vs shock patients with right ventricular dysfunction.

patients (cases 1, 2, and 6) there was a rise in pulmonary artery wedge pressure from 9 or 10 mmHg to between 15 and 19 mmHg without any associated rise in cardiac index or decrease in pressor requirements. All three died despite the addition of intra-aortic balloon pumping in two of them (cases 2 and 6). In contrast two other patients (cases 3 and 5) showed a rise in cardiac index from 1.3 to 1.5 and from 1.5 to 2.7 litre/min per m², respectively, in response to plasma volume expansion sufficient to raise wedge pressure from 8 to 17 mmHg in case 3 and from 9 to 14 mmHg in case 5. Intra-aortic balloon pumping was also used in case 5. Both these patients survived. Case 4 also survived with intra-aortic balloon pumping but was not treated by plasma volume expansion.

Five other patients with shock but without evidence of right ventricular dysfunction (cases 7, 9, 10, 14, and 16) also survived. Two (cases 9 and 10) were treated with plasma volume expansion plus catecholamine infusion and did not require intra-aortic balloon pumping. In these two patients fluids were given continuously for 12 to 24 hours after admission. Though wedge pressure did not rise in either patient, cardiac index increased from 1.6 to 2.3 and from 2.8 to 3.1 litre/min per m², respectively. Moreover, the rise in cardiac index in each patient occurred during the time of weaning off and discontinuance of catecholamines. The remaining three surviving patients were treated only with catecholamines and the balloon pump.

NECROPSY FINDINGS

Eight patients, including two (cases 2 and 6) with right ventricular dysfunction, had a post-mortem examination. All the hearts were hypertrophied and in all 40 per cent or more of the left ventricle was infarcted (old plus new) (Fig. 1). Infarction of the posterior interventricular septum was present in both patients with right ventricular functional impairment and in four of six patients without right ventricular dysfunction; the extent of posterior septal involvement was comparable in both groups. Gross evidence of right ventricular infarction was present in case 2, but not in case 6, and extended to the posterior free wall of the right ventricle (Fig. 1). Of patients without evidence of right ventricular dysfunction only case 13 had gross evidence of right ventricular infarction; in this patient, the necrosis was confined to a small basal region of the right ventricular posterior wall.

Microscopical examination of the right ventricle in patients with right ventricular impairment showed multiple and often confluent areas of acute necrosis, primarily involving subendocardial and mid-myocardial zones throughout the right ventricle (Fig. 2). Similar findings in patients with right ventricular infarction have been reported previously (Laurie and Woods, 1963). In contrast, the right ventricle of patients without right ventricular dysfunction (Fig. 3) had no (case 12) or only a few isolated foci of necrosis (cases 13, 17, and 18). In two other patients (cases 11 and 15), right ventricular damage could not be assessed because the relevant sections were not available for review.

Discussion

Several recent studies (Cohn et al., 1974; Rigo et al., 1975; Sharpe et al., 1978; Tobinick et al., 1978) have focused attention on the role of right ventricular infarction when cardiogenic shock is associated with inferior myocardial infarction. In the cases described in these reports the extent of coexistent left ventricular damage has been thought to be modest (Cohn et al., 1974; Rigo et al., 1975). The fact that several of the patients studied have had relatively low pulmonary artery wedge pressures and responded well to fluid administration and/or vasodilator treatment has been taken to support...
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This view. It has therefore been postulated that the occurrence of cardiogenic shock in this situation is the consequence of the inability of the severely damaged right ventricle to pump adequate amounts of blood into the left side of the heart (Cohn et al., 1974; Rigo et al., 1975). This concept is not new and was first proposed by Zaus and Kearns in 1952.

In the present study, however, some observations indicate that in patients with cardiogenic shock in association with combined inferior and right ventricular myocardial infarction, left ventricular damage may be more extensive than has been reported previously. For example, the depression of left ventricular stroke work index in patients with shock was similar in those with and in those without right ventricular infarction. The lower pulmonary artery wedge pressure in patients with right ventricular infarction probably results from right ventricular dysfunction rather than less extensive left ventricular damage. This interpretation is supported by the fact that pulmonary artery wedge pressure was 10 mmHg or less in the two patients with right ventricular infarction studied at necropsy but was 14 mmHg or more (median 26 mmHg) in the six patients without right ventricular dysfunction. Nevertheless, in both groups total extent of infarction (old plus new) was 40 per cent or more of the left ventricular myocardium.

Further, despite initial wedge pressures of 11 mmHg or less in all patients with haemodynamic evidence of right ventricular infarction, only two of five treated by plasma volume expansion responded with an increase in cardiac index and both severe left ventricular damage is usually incompatible with survival; in another group, there is less severe left ventricular damage and sufficient viable myocardium to permit them to respond favourably to plasma volume expansion and to other treatment. However, because of right ventricular dysfunction, both groups may have relatively low left-sided filling pressures.

These findings have a number of implications for the management of patients with cardiogenic shock and right ventricular infarction. Thus, if plasma volume expansion is used in an attempt to relieve circulatory failure, close attention should be directed toward the response of left ventricular stroke work to an increase in pulmonary artery wedge pressure induced by the increased plasma volume. It is possible that these patients may have more left ventricular damage than their initial wedge pressures suggest and thus may not respond favourably to volume loading. This was the case...

Fig. 1 Post-mortem section of heart from case 2. There is recent infarction involving posterior septum (A), posterior wall (B), and lateral wall (C) of left ventricle. In addition, posterior wall of right ventricle (D) is involved by recent infarction. The remainder of the right ventricular free wall appears grossly uninvolved by infarction. Total left ventricular necrosis totalled 40 per cent.
in three of five patients with right ventricular infarction in this series. Likewise, if vasodilators are used then agents that work predominantly on the arterial side of the circulation may be the most appropriate choice since excessive venodilatation and consequent preload reduction may have an adverse effect in this situation. In addition, in view of the frequent occurrence of severe left ventricular damage, early consideration of intra-aortic balloon pumping also appears warranted (Leinbach et al., 1978). Finally, because the distinction of those patients with very severe left ventricular damage...

Fig. 2 Photomicrograph of representative section of lateral right ventricular wall from case 2 shows extensive necrosis of myocytes and early infiltration by polymorphonuclear leucocytes. (H. and E. × 80.)

Fig. 3 Photomicrograph of representative section of right ventricle from case 17, without haemodynamic evidence of right ventricular dysfunction. Only isolated foci of myocyte necrosis (arrows) are present. (H. and E. × 80.)
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from those with less severe damage may be impossible from haemodynamic observations alone, it may be useful to obtain radionuclide ventriculograms in these patients in order to make this important distinction (Rigo et al., 1974).

In summary, the present study indicates that when cardiogenic shock is associated with combined right ventricular and inferior myocardial infarction, this may be associated with more severe left ventricular damage than has previously been reported. Furthermore, right ventricular dysfunction in this situation may cause relatively low left-sided filling pressures even in the presence of extensive left ventricular infarction. This may be a cause of error in the clinical and haemodynamic assessment of the extent of left ventricular damage present. Since the extent of left ventricular damage will ultimately not only determine the response to treatment but also prognosis (Sobel et al., 1972; Weber et al., 1978), it is clearly important to attempt to distinguish those patients with very severe left ventricular infarction from those in whom left ventricular damage is less extensive. Radionuclide ventriculograms may prove useful for this purpose and should, therefore, be obtained as early as possible in the patient’s illness.

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References


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