Echocardiographic Observations on Ventricular Septal Rupture Complicating Acute Myocardial Infarction

By P. A. N. Chandraratna, M.D., M.R.C.P., P. K. Balachandran, M.D., P. M. Shah, M.D., and M. Hodges, M.D.

SUMMARY

Echocardiograms were performed on three patients with ventricular septal rupture complicating myocardial infarction. The pulmonary artery mean pressure was 90 mm Hg or more in all three patients. The size of the ventricular septal defect, determined at operation or autopsy, was 2 cm or greater in each patient. The salient echocardiographic abnormality was dilatation of the right ventricle. The direction of septal motion was normal in all the patients. The left atrial diameter was slightly increased in one patient and was normal in the other two. In one patient, an unusual pattern of mitral valve motion was seen. Complete closure of the mitral valve after its initial opening in early diastole was observed and this was followed by almost complete reopening of the valve. This pattern was suggestive of increased blood flow through the mitral valve. Although some of these findings are nonspecific, the combination of echocardiographic findings may provide useful clues to the diagnosis of septal perforation.

Additional Indexing Words:
Ultrasound Myocardial infarction Ventricular septal defect

ECHOCARDIOGRAPHY was introduced by Edler and Hertz\textsuperscript{1} as a useful means of studying mitral valve function. This technique has subsequently been widely used to investigate various intracardiac structures. Right and left ventricular dimensions, left atrial size and aortic root diameter can all be accurately measured using ultrasound.\textsuperscript{2, 3} The range of normal values for these various structures has been established; and the abnormalities that occur in different forms of heart disease have been described.\textsuperscript{4-8}

Ventricular septal rupture is a rare and ominous complication of acute myocardial infarction. Since surgical correction of the defect is sometimes feasible, accurate diagnosis of septal rupture is important. The hemodynamic and echocardiographic findings in three patients with ventricular septal defect secondary to myocardial infarction are presented. Although many of these features are nonspecific, the combination of echocardiographic findings may provide useful clues to the diagnosis of ventricular septal rupture.

Materials and Methods

Three patients with acute myocardial infarction and ruptured ventricular septum were studied. Two of these patients were admitted to the University of Rochester Medical Center and the third was seen at the Mount Sinai Medical Center. None of them had a past history of a heart murmur or congestive cardiac failure. Cardiac catheterization was performed shortly after admission in all three patients. Informed consent was obtained from each patient prior to cardiac catheterization.

Echocardiography was performed using a commercially available ultrasonoscope and a 0.5 inch, 2.25 MHz transducer. With each patient in the supine position, the transducer was placed in the third or fourth interspace at the left sternal edge and the mitral valve, aortic root, left atrium, and left ventricle echograms were recorded.

Results

The clinical data on our patients are presented in table 1. The first patient (WF) sustained an inferior infarct, the second (DW) had an infero-lateral infarct, and the third (SK) had an antero-septal myocardial infarct. Ventricular septal rupture occurred on the fifth, second and third day, respectively. The patients presented with chest pain, hypotension or congestive cardiac failure. A loud systolic murmur and systolic thrill were noted at the lower left sternal border in two patients (WF, SK). The third patient (DW) had a somewhat atypical grade III/VI mid-systolic murmur.

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\textsuperscript{3} Received August 30, 1974; revision accepted for publication October 11, 1974.
which was of equal intensity at the left sternal edge and the apex. The first patient had emergency surgery and died at operation. Successful repair of the ventricular septal defect was achieved in the second patient five weeks after septal perforation. The third patient died of intractable cardiac failure and cardiogenic shock after nine days.

The hemodynamic data of the three patients are shown in table 2. All three patients had a pulmonary artery mean pressure of 30 mm Hg or more. Patients DW and SK were noted to have left-to-right shunts at the ventricular level of 3.2:1, and 2.4:1, respectively. The left ventricular angiogram in the first and second patients showed a large ventricular septal defect in the lower part of the muscular septum and a markedly enlarged right ventricle. Coronary arteriography in patient WF revealed significant occlusions in the right coronary artery, first marginal artery and circumflex artery. Patient DW had total occlusion of the distal circumflex and left anterior descending coronary arteries.

The operative or autopsy data are outlined in table 3. Patient WF had an infarct involving the inferior wall and the posterior half of the interventricular septum. There was a 2 cm ventricular septal defect (VSD) in the inferior part of the muscular septum. A total occlusion of the left main coronary artery was found at autopsy in patient SK. She had a 2.5 cm VSD and massive infarction of the septum.

The echocardiographic findings in our patients are

### Table 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>ECG</th>
<th>Day of rupture</th>
<th>Presentation</th>
<th>Physical signs</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>WF</td>
<td>59</td>
<td>M</td>
<td>Inferior infarct</td>
<td>5</td>
<td>Chest pain, hypotension</td>
<td>Systolic thrill and grade V/VI holosystolic murmur at the lower left sternal border</td>
<td>Emergency surgery. Operative death</td>
</tr>
<tr>
<td>DW</td>
<td>62</td>
<td>M</td>
<td>Inferolateral infarct</td>
<td>2</td>
<td>Chest pain, CCF, low output state</td>
<td>Grade III/VI harsh mid-systolic murmur at the left sternal border and apex</td>
<td>Surgery five weeks after septal rupture. Successful repair of VSD</td>
</tr>
<tr>
<td>SK</td>
<td>70</td>
<td>F</td>
<td>Antero-septal infarct</td>
<td>3</td>
<td>CCF</td>
<td>Thrill and grade IV/VI holosystolic murmur at the lower left sternal border</td>
<td>Died after nine days</td>
</tr>
</tbody>
</table>

Abbreviations: CCF = congestive cardiac failure; VSD = ventricular septal defect.

### Table 2

<table>
<thead>
<tr>
<th>Patient</th>
<th>RA (mm Hg)</th>
<th>RV (mm Hg)</th>
<th>PA (mm Hg)</th>
<th>PAW</th>
<th>SA (mm Hg)</th>
<th>LV angio</th>
<th>Coronary angio</th>
</tr>
</thead>
<tbody>
<tr>
<td>WF</td>
<td>11</td>
<td>43/13</td>
<td>43/20</td>
<td>30</td>
<td>108/64</td>
<td>93/17</td>
<td>Large VSD in the inferior portion of septum, mild mitral incompetence, slight reduction of LV contractility, dilated RV</td>
</tr>
<tr>
<td>DW</td>
<td>—</td>
<td>—</td>
<td>58/24</td>
<td>34</td>
<td>120/78</td>
<td>82</td>
<td>3.2:1</td>
</tr>
<tr>
<td>SK</td>
<td>—</td>
<td>—</td>
<td>60/24</td>
<td>35</td>
<td>108/75</td>
<td>90</td>
<td>2.4:1</td>
</tr>
</tbody>
</table>

Abbreviations: RCA = right coronary artery; CCA = circumflex coronary artery; LCA = left coronary artery.

*Circulation, Volume 51, March 1975*
Table 3
Operative or Autopsy Data

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>WF</td>
<td>Marked dilatation of the heart; infarction of the posteroinferior surface and one-half of the interventricular septum; 2 cm VSD in the inferior part of the muscular septum.</td>
</tr>
<tr>
<td>DW</td>
<td>Inferior wall infarct; 5 cm dyskinetic area on the inferior wall; 2.5 cm VSD adjacent to the inferior wall.</td>
</tr>
<tr>
<td>SK</td>
<td>Occlusion of the left main coronary artery; normal dominant right coronary artery; massive infarction of the interventricular septum with a septal rupture 2.5 cm in diameter; normal free wall of the left ventricle.</td>
</tr>
</tbody>
</table>

summarized in table 4. Marked dilatation of the right ventricle was a salient feature in all the patients. It is of interest that the left atrium was enlarged in only one patient. Figure 1 illustrates the aortic root and the normal sized left atrium in patient WF. An unusual pattern of mitral valve movement was seen in this patient (fig. 2). The mitral valve was seen to close completely shortly after it opened in diastole, and the valve then reopened almost completely. A markedly dilated right ventricle was present. Figure 3 shows the echocardiogram of patient SK. The tricuspid valve is seen within the dilated right ventricle. The direction of septal motion was normal in all three patients.

Discussion

Rupture of the interventricular septum is an uncommon complication of myocardial infarction, which carries an ominous prognosis. It occurs in 0.5 to 1% of cases of acute myocardial infarction. In one series only 11% survived more than two months.

The characteristic physical signs of septal rupture consist of a holosystolic murmur best heard at the left sternal border, without radiation to the axilla; and a systolic thrill in 50% of cases. The electrocardiogram frequently shows conduction disturbances. In contrast, acute mitral regurgitation due to myocardial infarction is usually associated with a systolic murmur that is best heard at the apex. However, the clinical differentiation between septal rupture and acute mitral regurgitation is not always easy. In Selzer's series of ten cases, the murmur of septal rupture was apical or was of equal intensity at the apex and the left sternal border in half the cases. In one of our patients (DW) a harsh mid-systolic murmur was equally as loud at the apex as at the left sternal border. The diagnosis of septal perforation in this patient could not be made confidently on clinical grounds.

Dilatation of the right ventricle was a consistent echocardiographic feature in all three patients. This is readily explained on the basis of combined volume and pressure overload of the right ventricle. In contrast, the echocardiographic right ventricular dimension was normal in four patients with pulmonary edema and acute mitral incompetence complicating myocardial infarction, studied in our laboratory (fig. 4) (unpublished data). Thus, the presence of right ventricular dilatation on the echocardiogram may be useful in differentiating between septal rupture and acute mitral regurgitation in the setting of an acute myocardial infarct.

The unusual pattern of mitral valve movement seen in patient WF may also be of some diagnostic significance. Complete closure of the valve after its initial opening early in diastole may be explained by very rapid ventricular filling producing a steep rise in the left ventricular diastolic pressure. The valve then reopens almost completely. This probably denotes torrential flow through the mitral valve.

Table 4
Echocardiographic Findings

<table>
<thead>
<tr>
<th>Patient</th>
<th>RV (cm)</th>
<th>LVED (cm)</th>
<th>LA (cm)</th>
<th>Direction of septal motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>WF</td>
<td>3.0</td>
<td>4.8</td>
<td>3.5</td>
<td>Normal</td>
</tr>
<tr>
<td>DW</td>
<td>2.7</td>
<td>5.2</td>
<td>4.2</td>
<td>Normal</td>
</tr>
<tr>
<td>SK</td>
<td>3.2</td>
<td>5.4</td>
<td>2.8</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Abbreviation: LVED = left ventricular end-diastolic dimension.

Figure 1
Echocardiogram of patient WF, showing the aortic root and the left atrium. CW = chest wall; RVO = right ventricular outflow tract; AW Ao = anterior wall of aorta; PW Ao = posterior wall of aorta; LA = left atrium.

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Ultrasound recording from patient WF illustrating the dilated right ventricle and the abnormal pattern of movement of the mitral valve. CW = chest wall; RV = right ventricle; IVS = interventricular septum; ECG = electrocardiogram; AMV = anterior leaflet of mitral valve; PMV = posterior leaflet of mitral valve; PLV = posterior wall of left ventricle.

Echocardiogram of patient SK demonstrating the dilated right ventricle (RV) and the tricuspid valve (TV). PLV = posterior wall of left ventricle; IVS = interventricular septum.

Echocardiogram from a patient with acute mitral incompetence complicating myocardial infarction. There is excessive movement of the interventricular septum (IVS) and dilatation of the left ventricle. The right ventricular dimension is normal. LVPW = posterior wall of left ventricle.
The left atrial diameter was slightly increased in one patient and was within normal limits in the other two. These findings are consistent with poor compliance of the left atrium in the acute situation.

In summary, the echocardiographic findings in three patients with ventricular septal rupture complicating acute myocardial infarction are presented. Since the complication cannot always be distinguished from acute mitral regurgitation, the echocardiogram may provide useful clues in support of this diagnosis.

References
Echocardiographic observations on ventricular septal rupture complicating acute myocardial infarction.
P A Chandraranta, P K Balachandran, P M Shah and M Hodges

Circulation. 1975;51:506-510
doi: 10.1161/01.CIR.51.3.506

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