CONGESTIVE HEART FAILURE after acute myocardial infarction usually indicates extensive muscle necrosis not remediable by surgical intervention. By contrast, the appearance of a new, loud systolic murmur in this clinical setting suggests the possibility of a surgically correctable lesion, and further study is mandatory. Fox et al.\(^1\) stated that the exact anatomic diagnosis of a correctable early complication of myocardial infarction requires invasive study, and preliminary screening of these patients requires bedside hemodynamic monitoring.\(^2\) The development of two-dimensional echocardiography and its application to patients with coronary artery disease gives the clinician an alternative, noninvasive bedside method of studying these critically ill patients.

Materials and Methods

During a 3-year period, 17 patients developed or were referred to our institution with congestive heart failure and a new, loud (at least grade III/VI) systolic murmur after an acute myocardial infarction (table 1). Two-dimensional echocardiographic study was performed prospectively on 14 of these patients before invasive study. Studies were performed using either a Grumman Health Systems RT-400 or a Varian V-3000 phased-array sector scanner. Complete echocardiographic studies included the parasternal long-axis and short-axis views, the apical long-axis and four-chamber views, and the subxiphoid four-chamber view.\(^3\) Careful attention was paid to transducer position and the sector was deliberately angulated both on-axis and off-axis to evaluate the entire interventricular septum and the entire mitral valve apparatus. Off-axis views were obtained by first placing the transducer at a standard window and obtaining a standard view. Then the transducer was angulated through an arc perpendicular to the plane of the two-dimensional sector. Alternatively, once the standard view was obtained, the transducer was rotated slightly clockwise or counterclockwise. Studies were reviewed by two interpreters. Contrast studies were not performed.

All patients had invasive diagnostic studies. Twelve had surgical confirmation of the anatomic pathology.

Results

Papillary Muscle Rupture

Four patients had a rupture of a part of a papillary muscle. Each had an inferior myocardial infarction. The parasternal and apical long-axis views were most valuable in detecting the mitral valve abnormality. In each case, there was a flail anterior mitral leaflet.

During systole, the tip of the anterior mitral leaflet went beyond the posterior leaflet into the left atrium; maximal abnormal motion occurred at the tip of the leaflet with loss of systolic leaflet coaptation. The length of the anterior leaflet segment involved in the “flailing” systolic motion was unusually long (figs. 1A–1D), or the tip of this segment was abnormally thick. In one patient, the posteromedial papillary muscle appeared truncated, and a definite mass was attached to the anterior leaflet (fig. 1E). (At surgery, this mass was the detached head of the posteromedial papillary muscle.) In each patient, the posterior mitral leaflet moved normally and the interventricular septum appeared intact.

All four patients had severe mitral insufficiency documented by left ventriculography. In only one patient did angiography detect a ruptured papillary muscle. All had a mitral valve replacement; three survived. At surgery, all had rupture of one or two heads of the posteromedial papillary muscle.

Papillary Muscle Dysfunction

Five patients had papillary muscle dysfunction. Each had an inferior myocardial infarction. The parasternal and apical long-axis views and the para-
sternal short-axis views were of greatest value in detecting the mitral valve abnormality. No patient had either a flail anterior or a flail posterior mitral leaflet; systolic coaptation of the leaflets was preserved. Systolic and diastolic excursion of the anterior leaflet was normal; however, during systole and diastole, motion of the posterior leaflet was reduced; it appeared to be retracted apically and held in a rigid position. On parasternal and apical long views, the posterobasal left ventricular wall was akinetic or dyskinetic. This abnormal segment involved the insertion of the posteromedial papillary muscle in three patients. On short-axis views, motion of the posteromedial papillary muscle was reduced. On all views, the posteromedial papillary muscle appeared more dense than the anterolateral papillary muscle (fig. 2).

All five patients had severe mitral insufficiency documented by left ventriculography (table 1). All had mitral valve replacement; three survived. At surgery, papillary muscles and chordae were intact, but the posteromedial papillary muscle was scarred.

**Ventricular Septal Rupture**

Eight patients had ventricular septal rupture; five were studied by two-dimensional echocardiography; three died before a study could be performed. Of the
patients who survived, four had an inferior myocardial infarction and one had an anterior infarction. Of the patients who died, two had an anterior infarction and one had an inferior infarction. The apical or sub-xiphoid four-chamber view or the parasternal short-axis view detected the septal defect. Occasionally, it was necessary to direct the transducer off-axis to find the septal defect. In each case, the site of septal rupture was located in the center of a septal aneurysm; septal tissue immediately adjacent to the defect

### Table 1. Clinical Data on 14 Patients Who Developed Congestive Heart Failure and a Loud Systolic Murmur After Myocardial Infarction

<table>
<thead>
<tr>
<th>Ruptured papillary muscle</th>
<th>Age (year)</th>
<th>Sex</th>
<th>Acute infarction</th>
<th>Old infarction</th>
<th>Interval from infarction to complication</th>
<th>CAD</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>63</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>5 days</td>
<td>RCA</td>
<td>MVR (died)</td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>M</td>
<td>Inferior</td>
<td>No</td>
<td>2½ weeks</td>
<td>LCF</td>
<td>MVR</td>
</tr>
<tr>
<td>3</td>
<td>65</td>
<td>M</td>
<td>Inferior</td>
<td>No</td>
<td>6 weeks</td>
<td>LAD, RCA</td>
<td>MVR</td>
</tr>
<tr>
<td>4</td>
<td>49</td>
<td>M</td>
<td>Inferior</td>
<td>No</td>
<td>6 weeks</td>
<td>LCF, RCA</td>
<td>MVR</td>
</tr>
<tr>
<td>Papillary muscle dysfunction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>Unknown</td>
<td>RCA</td>
<td>MVR (died)</td>
</tr>
<tr>
<td>6</td>
<td>72</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>3 weeks</td>
<td>RCA</td>
<td>MVR</td>
</tr>
<tr>
<td>7</td>
<td>67</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>2 weeks</td>
<td>LAD, LCF, RCA</td>
<td>MVR (died)</td>
</tr>
<tr>
<td>8</td>
<td>66</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>4 weeks</td>
<td>LAD, LCF, RCA</td>
<td>MVR</td>
</tr>
<tr>
<td>9</td>
<td>60</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>4 weeks</td>
<td>RCA</td>
<td>MVR</td>
</tr>
<tr>
<td>Ventricular septal rupture</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>62</td>
<td>M</td>
<td>Inferior</td>
<td>No</td>
<td>6 weeks</td>
<td>RCA</td>
<td>Septal patch (died)</td>
</tr>
<tr>
<td>11</td>
<td>61</td>
<td>M</td>
<td>Inferior</td>
<td>No</td>
<td>10 days</td>
<td>LAD, RCA</td>
<td>Septal patch</td>
</tr>
<tr>
<td>12</td>
<td>62</td>
<td>M</td>
<td>Anterior</td>
<td>No</td>
<td>Unknown</td>
<td>LAD, LCF, RCA</td>
<td>No</td>
</tr>
<tr>
<td>13</td>
<td>67</td>
<td>M</td>
<td>Inferior</td>
<td>No</td>
<td>2 weeks</td>
<td>LAD, LCF, RCA</td>
<td>Septal patch (died)</td>
</tr>
<tr>
<td>14</td>
<td>72</td>
<td>F</td>
<td>Inferior</td>
<td>No</td>
<td>1 day</td>
<td>RCA</td>
<td>No</td>
</tr>
</tbody>
</table>

Abbreviations: CAD = coronary artery disease (greater than 75% narrowing of left anterior descending [LAD], left circumflex [LCF], or right coronary artery [RCA]); MVR = mitral valve replacement.
Figure 2. A parasternal short-axis view of a patient with severe mitral insufficiency from acute dysfunction of the posteromedial papillary muscle (MPM). The posteromedial papillary muscle is fibrotic and appears more dense than the anterolateral papillary muscle (LPM). A = anterior; P = posterior; R = right; L = left; RV = right ventricle; LV = left ventricle.

Discussion

Drobec et al. reported 30 patients with a complicated acute myocardial infarction studied by two-dimensional echocardiography; two had a ventricular septal defect and one had a flail mitral valve. Heger et al. reported 23 patients studied by two-dimensional echocardiography; six had either acute mitral insufficiency or a ventricular septal defect. We have had no false-positive or false-negative diagnoses of septal or papillary muscle rupture.

Papillary muscle rupture is unusual; it is a complication in 1% of acute myocardial infarctions. Each papillary muscle provides chordae tendineae to each leaflet. The posteromedial papillary muscle is involved 2.5 times more often than the anterolateral papillary muscle. Thus, as in our four patients, an inferior myocardial infarction can cause a ruptured posteromedial papillary muscle, which can produce a flail anterior mitral leaflet. We did not observe a flail posterior leaflet in any of these patients. There are a number of possible explanations. First, our population size is small. Second, the coronary distribution and site of occlusion determine the location of the infarct and the site of papillary muscle rupture. Thus, rupture of a different head of the posteromedial papillary muscle could produce a flail posterior leaflet. Third, the anatomic differences between the two leaflets, the relative sizes and the number of cordal attachments could produce greater posterior than anterior leaflet stability in the event of papillary muscle rupture.

Rupture of an entire papillary muscle is usually fatal; most patients who survive the acute event have rupture of only one or two heads of one of the papillary muscles. Overall, papillary muscle rupture is an unusual cause of acute mitral insufficiency except in the setting of an acute myocardial infarction; in this setting, a ruptured muscle and ruptured chordae can produce a flail mitral leaflet detectable by two-dimensional echocardiographic study. It may not always be possible to determine the precise site of rupture after an acute infarction. However, in our four patients, the length of the flail leaflet segment was longer and the tip of the segment thicker than usual in ruptured chordae; this suggests that part of a papillary muscle was attached to the leaflet. Furthermore, in one of these patients, the papillary muscle was truncated. Although the exact sensitivity and specificity of these findings in differentiating a ruptured papillary muscle from ruptured chordae tendineae is unknown, the clinical implications of each are similar. Recently, Wei et al. suggested that a ruptured papillary muscle usually occurs with a small myocardial infarction and that early surgical intervention be considered in all of these patients. The surgical prognosis depends on the degree of left ventricular scarring.

The acute development of irreversible papillary muscle dysfunction and severe mitral insufficiency is an unusual, not-well-recognized, extreme form of papillary muscle dysfunction. Experimental studies indicate that papillary muscle infarction does not produce significant mitral insufficiency unless both papillary muscles or left ventricular myocardium adjacent to a single infarcted papillary muscle is also involved. Indeed, Shelburne et al. suggested that in humans, severe left ventricular dysfunction must coexist to produce significant valvular insufficiency. However, in some cases papillary muscle necrosis can lead to acute fibrosis, shortening, and retraction of one of the mitral leaflets into the body of the left ventricle. The resulting disruption of the normal line of anterior and posterior leaflet closure could cause severe mitral insufficiency. Glancy et al. suggested that these patients are indistinguishable clinically from patients with papillary muscle rupture. However, by two-dimensional echocardiography, the two lesions can be differentiated; but patients with papillary muscle fibrosis and severe, surgically remediable, valvular insufficiency cannot be separated by two-dimensional study from patients with papillary muscle dysfunction and mild or moderate insufficiency. This requires invasive study.

Septal rupture occurs in approximately 1% of acute...
Correctable Complications of AMI/Mintz et al.

Infarctions. The rarity of this event is explained by the septum's dual blood supply; furthermore, transseptal pressure is countered by tension on the right ventricular side.\textsuperscript{18, 19} Rupture may occur with either anteroseptal or inferior infarction;\textsuperscript{20} survival depends on severity of left ventricular failure, a function of infarct and shunt size.\textsuperscript{1-21}

Scanlan et al.\textsuperscript{22} and Farcot et al.\textsuperscript{23} both reported the two-dimensional echocardiographic detection of septal rupture. In our study, we had no difficulty visualizing the site of rupture. The necrotic and fibrotic septal tissue adjacent to the defect probably enhances visualization; the density difference is greater than between normal myocardium and a defect (as would occur in a congenital hole). However, perforations may also present as multiple holes, as a serpigenous tunnel from the left to the right ventricle, or a jagged linear laceration,\textsuperscript{1} so two-dimensional echocardiography may not detect all cases of septal rupture. Strict adherence to technique is important; the transducer must be angulated off-axis in each standard view to maximize detection. The use of peripheral contrast injection may be helpful; a negative contrast effect in the right ventricle or a positive contrast effect in the left ventricle may confirm the presence of a shunt or detect an unsuspected one.

Therapy of a ruptured interventricular septum differs from the therapy of acute mitral insufficiency. Most series\textsuperscript{1, 21-24} report enhanced survival if surgery can be delayed 3 weeks. Delay often is not possible. In a recent study, Montoya et al.\textsuperscript{25} stated that early surgery carries a relatively low mortality and that delay causes unacceptable risks and unpredictable mortality. In our series, three patients died before we could study them.

The ability of two-dimensional echocardiography to visualize the entire left ventricular myocardium as well as the spatial relationships of the intracardiac structures is a significant advance in noninvasive diagnosis. Early complications of myocardial infarction can be diagnosed and surgically correctable causes of congestive heart failure can be identified. The identification of a remediable cause of heart failure in these patients mandates surgical intervention. In addition, two-dimensional echocardiog-

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure3}
\caption{Sequential systolic frames from a two-dimensional echocardiographic study of a patient with an acute ventricular septal defect after myocardial infarction. The apical four-chamber view is shown. At the onset of systole (A), there is no defect. During systole (B and C), the septum bulges into the right ventricle (RV). The open white arrow indicates the defect in the middle of this septal aneurysm. R = right; L = left; LV = left ventricle; RA = right atrium; LA = left atrium.}
\end{figure}
raphy allows quantitation of residual left ventricular function, an important prognostic parameter in the therapy of patients with early complications of myocardial infarction. This approach may select patients requiring an aggressive therapeutic approach. It may also obviate the need for left ventricular angiography, a hazardous procedure in these patients.

References

Two-dimensional echocardiographic identification of surgically correctable complications of acute myocardial infarction.

G S Mintz, M F Victor, M N Kotler, W R Parry and B L Segal

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