Ventricular Septal Defect After Myocardial Infarction: Diagnosis by Two-dimensional Contrast Echocardiography

Milutin Drobac, M.D., Brian Gilbert, M.D., Robert Howard, M.D., Ronald Baigrie, M.D., and Harry Rakowski, M.D.

SUMMARY Thirteen patients who had ventricular septal defects (VSDs) after myocardial infarction (MI) underwent two-dimensional echocardiography (2-D echo), with confirmation of the VSD by oximetry. Eight of the patients were male and five were female, ages 51–76 years. Five had anterior and eight inferior MIs. Two-dimensional echocardiography revealed akinesis or dyskinesis of the interventricular septum (IVS) in all 13 patients. In only six could a defect in the IVS be directly visualized. Two-dimensional echocardiographic left ventricular (LV) wall motion abnormalities correlated with ECG and angiographic site of infarction in all patients. Twelve patients had adequate saline contrast studies. Positive LV contrast (microbubbles entering the left ventricle through the VSD) was seen in 11 patients, and negative right ventricular (RV) contrast (washout of the RV bubbles by LV blood crossing the VSD) in five patients; at least one abnormality was present in every patient. The location of the VSD was determined by visualizing a VSD or by the site of the positive LV or negative RV contrast. Oximetry showed VSD shunts of 1.4:1 to 7:1, with no correlation between the degree of negative RV contrast and shunt size. Surgical or pathologic confirmation of VSD was obtained in 12 patients, with agreement of VSD location by 2-D echo in all. Four of the 11 patients who underwent surgical repair died, and two patients died before surgery could be attempted. We conclude that 2-D echo is a sensitive, rapid and safe technique for diagnosing VSD after MI. Positive LV contrast, with or without negative RV contrast, is more sensitive in the diagnosis and localization of post-MI VSD than direct echocardiographic visualization of the defect.

ACUTE rupture of the interventricular septum (IVS) is a life-threatening complication that occurs in 0.5–1% of patients with acute myocardial infarction (MI). Typically, a patient presents with biventricular failure, shock and a new pansystolic murmur usually within the first week after MI. Successful management depends on prompt recognition and hemodynamic stabilization, followed by surgical repair.

Clinically, a postinfarction ventricular septal defect (VSD) may be easily confused with acute mitral regurgitation, because the location of the murmur and the ECG site of infarction are usually similar. Initial diagnosis of VSDs has depended on oximetry studies showing a stepup in oxygen saturation in the left ventricle. Left ventricular (LV) cineangiography is carried out to assess LV function and to localize the anatomic site of septal rupture aiding the surgeon in the surgical approach. This procedure may carry significant risk to the typically unstable patient.

Two-dimensional echocardiography (2-D echo) is a safe and simple method of tomographically imaging the heart and allows visualization of the entire length of the IVS. This technique is useful for directly visualizing congenital membranous VSDs. Recently, direct visualization of postinfarction VSDs was reported.

However, since postinfarction VSDs may be small or multiple, or consist of a linear tear, they may not be readily visualized directly. Previous studies have demonstrated the value of contrast saline echocardiography in the diagnosis of intracardiac shunts. We therefore assessed the ability of 2-D echo to localize the site of acute postinfarction VSD by both direct visualization and by saline contrast studies.

Patients and Methods

All patients in our coronary care unit who presented with a new systolic murmur and acute hemodynamic deterioration after a recent MI underwent 2-D echo with saline contrast studies. Of 28 patients studied, 14 had a proved postinfarction VSD. Interpretable 2-D echo studies were obtained in 13 of the VSD patients, who formed this study group (table 1). In one patient, an adequate 2-D echo study could not be obtained. Eight patients were male and five were female, ages 51–76 years. Two of the patients had previous MIs. Five patients had acute anterior and eight acute inferior MIs, all with diagnostic electrocardiographic and enzymatic changes. All patients deteriorated acutely from 1–10 days after infarction, with clinical findings of pulmonary edema and a new pansystolic murmur (table 1). The remaining 14 patients did not have any echocardiographic, oximetric or pathologic evidence of a VSD and are not included in this study.

Echocardiography

Two-dimensional echocardiography was performed using a Varian V-3000 or V-3400 real-time, phasedarray ultrasonoscope. All patients were studied acutely in the coronary care unit within 15 minutes to 2 hours of admission or development of symptoms and findings suggestive of acute VSD, and before oximetry. Attempts were made to record parasternal long- and short-axis, apical two- and four-chamber and subcostal views. Particular emphasis was placed on visualizing the IVS in the four-chamber view. Attempts were made in all views to directly visualize the VSD. If a defect was not visualized in the standard views, the transducer was angulated through an arc sweeping the

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### Table 1. Clinical Data in 13 Patients with Postinfarction Ventricular Septal Defects

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Location of infarction</th>
<th>Clinical findings</th>
<th>Days post-MI</th>
<th>Operation</th>
<th>Procedure</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>68</td>
<td>F</td>
<td>Inf</td>
<td>PSM</td>
<td>NYHA</td>
<td>VSD</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>63</td>
<td>M</td>
<td>Inf</td>
<td>+</td>
<td>—</td>
<td>7</td>
<td>Died preoperatively</td>
</tr>
<tr>
<td>3</td>
<td>69</td>
<td>M</td>
<td>Inf</td>
<td>+</td>
<td>+</td>
<td>10</td>
<td>Died intraoperatively</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>M</td>
<td>Inf</td>
<td>+</td>
<td>+</td>
<td>7</td>
<td>VSD closure, posterior pseudoaneurysm closed, ACB; survived</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>M</td>
<td>Inf</td>
<td>+</td>
<td>-</td>
<td>1</td>
<td>Infarct resection with VSD patch, ACB; survived</td>
</tr>
<tr>
<td>6</td>
<td>51</td>
<td>F</td>
<td>Inf</td>
<td>+</td>
<td>+</td>
<td>1</td>
<td>Died intraoperatively</td>
</tr>
<tr>
<td>7</td>
<td>53</td>
<td>M</td>
<td>Inf</td>
<td>+</td>
<td>+</td>
<td>6</td>
<td>VSD patch, ACB; survived</td>
</tr>
<tr>
<td>8</td>
<td>76</td>
<td>F</td>
<td>Inf</td>
<td>+</td>
<td>+</td>
<td>3</td>
<td>Died preoperatively</td>
</tr>
<tr>
<td>9</td>
<td>58</td>
<td>M</td>
<td>Ant</td>
<td>+</td>
<td>-</td>
<td>1</td>
<td>Aneurysmectomy + VSD closure; died postoperatively</td>
</tr>
<tr>
<td>10</td>
<td>62</td>
<td>M</td>
<td>Ant</td>
<td>+</td>
<td>+</td>
<td>35</td>
<td>Aneurysmectomy + VSD closure; survived</td>
</tr>
<tr>
<td>11</td>
<td>67</td>
<td>M</td>
<td>Ant</td>
<td>+</td>
<td>-</td>
<td>4</td>
<td>Died intraoperatively</td>
</tr>
<tr>
<td>12</td>
<td>73</td>
<td>F</td>
<td>Ant</td>
<td>+</td>
<td>+</td>
<td>5</td>
<td>VSD patch, ACB; survived</td>
</tr>
<tr>
<td>13</td>
<td>69</td>
<td>F</td>
<td>Ant</td>
<td>+</td>
<td>+</td>
<td>7</td>
<td>VSD patch, ACB; survived</td>
</tr>
</tbody>
</table>

Abbreviations: INF = inferior; ANT = anterior; PSM = pansystolic murmur; NYHA = New York Heart Association functional classification; ACB = aortocoronary bypass; VSD = ventricular septal defect; + = present; = absent.

### Table 2. Two-dimensional Echocardiographic, Catheterization and Surgical Data

<table>
<thead>
<tr>
<th>Pt</th>
<th>Wall motion</th>
<th>VSD visualization</th>
<th>Contrast saline</th>
<th>Angiography</th>
<th>Coronary lesions (75%)</th>
<th>Shunt</th>
<th>Surgical pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Inferior + septal akinesis</td>
<td>No</td>
<td>+ apical</td>
<td>—</td>
<td>—</td>
<td>7:1</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>Inferior akinesis, septal dyskinesis</td>
<td>1 cm</td>
<td>+/- posterobasal</td>
<td>Inferior akinesis</td>
<td>RCA</td>
<td>2.6:1</td>
<td>4 x 3-cm posterobasal VSD</td>
</tr>
<tr>
<td>3</td>
<td>Inferior akinesis, septal dyskinesis</td>
<td>No</td>
<td>+/- posterior midventricular</td>
<td>Inferior akinesis</td>
<td>RCA, LAD</td>
<td>2.3:1</td>
<td>Irregular posterior septal tear</td>
</tr>
<tr>
<td>4</td>
<td>Inferior akinesis with pseudoaneurysm, septal dyskinesis</td>
<td>2 cm</td>
<td>+ apical</td>
<td>Inferior pseudoaneurysm</td>
<td>RCA</td>
<td>4.5:1</td>
<td>4 x 5-cm pseudoaneurysm 2-cm VSD</td>
</tr>
<tr>
<td>5</td>
<td>Posterobasal akinesis + septal dyskinesis</td>
<td>2 cm</td>
<td>+ midventricular</td>
<td>Inferior akinesis</td>
<td>LAD, RCA</td>
<td>2.3:1</td>
<td>Posterior 4 x 1.5 cm VSD (tear)</td>
</tr>
<tr>
<td>6</td>
<td>Septal dyskinesis, posterior akinesis</td>
<td>1-2 cm</td>
<td>+ posterobasal</td>
<td>Posterobasal akinesis</td>
<td>LAD, RCA, Cx</td>
<td>1.4:1</td>
<td>Posterobasal 1.5-2-cm VSD</td>
</tr>
<tr>
<td>7</td>
<td>Septal dyskinesis</td>
<td>2 cm</td>
<td>+ posterobasal</td>
<td>Apical dyskinesis</td>
<td>LAD, RCA, Cx</td>
<td>4:1</td>
<td>Posterobasal septal tear</td>
</tr>
<tr>
<td>8</td>
<td>Septal + posteromedial dyskinesis</td>
<td>No</td>
<td>Inadequate study</td>
<td>—</td>
<td>—</td>
<td>1.4:1</td>
<td>Posteroseptal serpentine tract (postmortem)</td>
</tr>
<tr>
<td>9</td>
<td>Apical dyskinesis, septal akinesis</td>
<td>No</td>
<td>+/- apical</td>
<td>Apical dyskinesis</td>
<td>LAD, RCA</td>
<td>4:1</td>
<td>Apical VSD (2 cm)</td>
</tr>
<tr>
<td>10</td>
<td>Apical + septal dyskinesis</td>
<td>No</td>
<td>+ apical</td>
<td>Apical dyskinesis</td>
<td>LAD, RCA</td>
<td>4:1</td>
<td>Apical VSD (1-1½ cm)</td>
</tr>
<tr>
<td>11</td>
<td>Apical + septal dyskinesis</td>
<td>No</td>
<td>+/- apical</td>
<td>Apical dyskinesis</td>
<td>LAD</td>
<td>2:1</td>
<td>Apical VSD, irregular (1 cm)</td>
</tr>
<tr>
<td>12</td>
<td>Apical + septal dyskinesis</td>
<td>No</td>
<td>- apical</td>
<td>Apical dyskinesis</td>
<td>LAD, RCA</td>
<td>3:1</td>
<td>Apical VSD (1-2 cm)</td>
</tr>
<tr>
<td>13</td>
<td>Posterobapical + septal akinesis</td>
<td>2 cm</td>
<td>+ posterobapical</td>
<td>Poster + apical akinesis</td>
<td>LAD, RCA</td>
<td>4.3:1</td>
<td>Infrapapillary VSD (2 cm)</td>
</tr>
</tbody>
</table>

Abbreviations: + = positive contrast; = negative contrast; RCA = right coronary artery; LAD = left anterior descending coronary artery; Cx = circumflex; VSD = ventricular septal defect.
septum to see as much of the septal anatomy as possible and search for a defect. Rapid multiple injections of 10 ml of normal saline were made into a peripheral vein (11 patients) or into the right atrium through a Swan-Ganz catheter (three patients). Injections were made through a three-way stopcock placed at the proximal end of the i.v. or Swan-Ganz line. Simultaneous 2-D echo images were recorded. If adequate microbubbles were not initially observed, 1 ml of room air was added to the syringe and agitated with 10 ml of normal saline. After visible air was expelled from the system, the injection was repeated. Positive LV contrast was present when microbubbles from the right ventricle were seen to cross the IVS into the left ventricle. Negative right ventricular (RV) contrast was considered present when RV microbubbles were consistently washed out by blood from the left ventricle, crossing the IVS. The saline study was considered negative if positive LV or negative RV contrast was not seen during three consecutive injections. Real-time and stop-frame analysis was performed by two or more independent reviewers.

The presence of a VSD was confirmed in all patients by oximetry during Swan-Ganz catheterization. Eleven of the patients underwent LV angiography and coronary arteriography. Two patients died before angiography could be done. Surgical repair was attempted in these 11 patients 4–60 days after infarction (table 1). Invasive studies were performed and interpreted independent of the echocardiographic data.

**Results**

**Echocardiography**

**Wall Motion Abnormalities**

Two-dimensional echocardiographic assessment of LV wall motion abnormalities was in complete agreement with abnormalities documented by angiography and ECG site of transmural infarction (table 2). Four of five patients with anterior MIs had apical LV aneurysms by both 2-D echo and angiography. Two-dimensional echocardiography further revealed that the apical dyskinesis involved most of the IVS in three patients, with an akinetic IVS in one patient. One patient with an anterior MI had extensive apical and septal akinesis. Seven of eight patients with inferior MIs had akinetic inferior walls by 2-D echo. One patient had normal posterior wall motion, with dyskinesis involving the posterior region of the IVS. All eight patients exhibited abnormal septal motion, seven with septal dyskinesis and one with septal akinesis. Wall motion abnormalities were in agreement with those documented by angiography in six patients (table 2).

**Direct Visualization of VSD**

In six patients, a VSD in the IVS was directly visualized (table 2, figs. 1 and 2). Five of these six had inferior MIs and large pathologic defects. One patient had an anterior MI with a defect visualized in the posteroapical region of the IVS. One of the six (patient 4) had concurrent rupture of the posterior wall with pseudoaneurysm formation, which was diagnosed by 2-D echo. Echocardiography underestimated the size of the defects in four of the patients.

In the other seven patients, despite adequate visualization of the IVS, a definite defect distinguishable from echo dropout could not be documented.

**Saline Contrast Studies**

Saline contrast studies were diagnostic of VSDs in 12 patients. In one patient, adequate saline studies could not be obtained. Positive LV contrast (microbubbles crossing from the right to the left ventricle) was seen in 11 patients (figs. 3, 4 and 5). In seven patients, five with anterior and two with inferior MI, one patient had normal posterior wall motion, with dyskinesis involving the posterior region of the IVS. All eight patients exhibited abnormal septal motion, seven with septal dyskinesis and one with septal akinesis. Wall motion abnormalities were in agreement with those documented by angiography in six patients (table 2).

**Figure 1.** Diastolic (A) and systolic (B) still frames taken in the short-axis view at the level of the papillary muscles from patient 2, who had an inferior myocardial infarction. The posterior region of the septum bulges acutely into the right ventricle (RV), with a 1-cm defect visualized at the apex of the bulge (arrow). Panels C and D are diagrammatic representations of A and B. LV = left ventricle; A = anterior; P = posterior; M = medial; L = lateral; P in panel C = papillary muscles.
In three patients, all with anterior infarction, this originated from the apical portion of the IVS washing out RV microbubbles, from apex to base. In two patients with inferior infarction, negative contrast originated from the posterobasal septum. Negative contrast was not observed in seven patients despite repeated adequate injections. The presence or degree of negative contrast did not correlate with shunt size (table 2).

**Invasive Studies**

All 13 patients underwent Swan-Ganz catheterization, with oximetry studies demonstrating left-to-right shunts at the ventricular level of 1.5:1–7:1 (table 2). Eleven patients had cardiac catheterization with LV angiography and selective coronary arteriography. Anatomic localization of the VSD was in agreement with 2-D echo in all patients, with six patients showing apical defects and five posterior defects. Eight of 11 patients had severe two- and three-vessel coronary artery disease and three had one-vessel disease (table 2). Although all patients showed significant hemodynamic impairment, further deterioration did not appear to be precipitated by cardiac catheterization.

**Pathology**

Seven of 13 patients underwent successful infarctectomy with VSD repair and survival at 2–18 months of follow-up (table 1). Four patients died perioperatively of pump failure and two died within hours of presentation despite aggressive attempts to stabilize them. Operative or pathologic confirmation of post-MI VSD was obtained in 12 patients. All defects were single, the edges showing various degrees of maturation, depending on the duration of the defect. The five patients with anterior MI had defects localized to the apical region of the IVS, the largest 2 cm in diameter.

In patients with inferior MIs, defects were located in the posterior region of the IVS, although in one, the defect was in the apical half of the left ventricle. Three patients with inferior MIs had irregular tears at the

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**FIGURE 2.** Apical four-chamber views demonstrating visualization of a posterocapical ventricular septal defect in patient 13. Panel A is the standard apical four-chamber view. In panels B, C and D, the plane of the transducer is progressively swept posteriorly, imaging the interventricular septum through a right angle. The apical region of the interventricular septum progressively bulges into the right ventricle (RV), until a posteriorly located defect is visualized in panel D (‘off axis’). RA = right atrium; LA = left atrium; LV = left ventricle.

this occurred in the apical region of the septum. Five patients with inferior MI showed positive LV contrast in the posterior septal region (table 2). Marked RV negative contrast was seen in five of 12 patients (fig. 4).
point of septal attachment to the posterior wall, and one had an inferior pseudoaneurysm (table 2). Patient 8 had a serpentine tract through the posterior LV wall, which tracked behind the IVS and opened into the right ventricle.

**Discussion**

Because of the usually life-threatening and deteriorating course in patients with postinfarction VSDs, a rapid and safe method of accurate diagnosis is important. VSD closure with or without infarctectomy may be life-saving. Using 2D echo, we rapidly and accurately diagnosed the presence of post-MI VSDs in 12 patients. The location of the VSD was correctly described by 2-D echo in 11 cases in which confirmatory pathology was available.

Although direct visualization of post-MI VSDs has been reported, this has not been a definitive diagnostic feature in our experience, unless the defect is particularly large.

Echocardiography consistently underestimated the size of the VSD, where visualized, and failed to visualize defects in seven patients. This may be explained by a number of factors. Although axial resolution is 2 mm, lateral resolution is as much as 1 cm, and depends on the gain setting used as well as the depth of the given study. The study technique may also have limitations. Patients are acutely ill and frequently in respiratory distress or intubated. This may limit both viewing time and quality.

As perforations may occur in regions of the IVS not normally visualized in the standard views, it may be necessary to angulate the transducer “off axis” to find a defect (fig. 2). Adequate saline contrast studies demand good image resolution with a minimum of background noise. In our experience, the apical four-chamber view most often allowed adequate studies. The anatomy of the defect itself may make its echocardiographic visualization difficult. Pathologically, the defects, when acute, have ragged edges, may have a serpentine course through the septum, and sometimes consist of multiple smaller defects. Defects may be obscured by RV trabeculations surgically, if approached through the right ventricle.

Saline contrast 2-D echo provided the most accurate means of diagnosis in this study. Positive LV contrast (microbubbles entering the LV) was seen in 11 of the initial 14 patients. Of the three other patients, in one echocardiography did not produce adequate images, one had an inadequate saline study and one had a true-negative study. Despite large left-to-right shunts, the defects allowed a significant number of microbubbles to cross into the left ventricle in diastole. This most likely represents admixture of RV and LV blood in diastole, rather than significant right-to-left shunting. Negative contrast has been reported as a feature of post-MI VSD; however, seven patients with large left-to-right shunts did not have negative RV contrast in this study. Although the other five patients had

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**Figure 4.** Still frames in the four-chamber view from patient 9 before and after saline contrast. After injection, microbubbles fill the right atrium (RA) (B) and right ventricle (RV) (C). In the next systole, they are completely washed out of the RV from the apex to base (E). During the next diastole, microbubbles again fill the RV (G), and microbubbles spill over into the left ventricle (LV) through the ventricular septal defect at the apex of the interventricular septum (arrow, panel G, and H). LA = left atrium.
marked negative contrast, this was not predictive of the size of the left-to-right shunt. Only one patient had negative contrast alone. Visualization of negative contrast may be affected by many factors. A large number of dense microbubbles entering the right ventricle or the delay in delivery of the saline bolus to the right ventricle, which may be visualized in patients with a prolonged circulation time, may obscure negative contrast. Furthermore, the left-to-right jet of blood across the VSD may be directed outside of the echocardiographic plane being viewed. The site of the VSD was accurately predicted in all cases by visualizing the region in which microbubbles crossed into the left ventricle or the origin of the RV negative contrast.

Two-dimensional echocardiography, therefore, provides a reliable means of rapidly diagnosing the presence and location of a VSD. It can also accurately demonstrate LV wall motion abnormalities\(^\text{22, 23}\) and the presence of other complications. If adequate 2-D echo images with saline contrast studies are obtained, the need for LV angiography is questionable. The benefits of aortocoronary bypass at the time of VSD repair have not been demonstrated. However, we believe that coronary arteriography is indicated when time allows. In hemodynamically unstable patients, the combination of oximetry (Swan-Ganz catheterization) and 2-D echo may represent adequate investigation in preparing these acutely ill patients for corrective surgery.

References

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**Figure 5.** Three still frames, with diagrammatic representations below, in the subcostal view from patient 10. Panels A and D show all four chambers of the heart, with arrows indicating a left ventricular apical aneurysm. After injecting 10 ml of normal saline (B), the right ventricular cavity (RV) is clearly outlined by microbubbles and a bulge at the apical portion of the interventricular septum is noted (arrows in panel E). In the subsequent diastole, a jet of microbubbles can be seen crossing an apically located ventricular septal defect and entering the apical portion of the left ventricle (LV) (arrows in panel F). Eff = effusion; RA = right atrium; LA = left atrium.
Two-dimensional Echocardiographic Diagnosis of Left Atrial Thrombus in Rheumatic Heart Disease

A Clinicopathologic Study

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with the technical assistance of Mae R. Palileo, RMT, Luz W. Esmele, RMT, Alejandro C. Caballero, RMT, and Rodel L. Leano, RMT

SUMMARY Two-dimensional echocardiographic studies were performed in 293 patients with rheumatic heart disease who underwent open-heart mitral valve surgery during an 18-month period. Diagnostic confirmation of a left atrial thrombus was based on direct inspection of the left atrium during surgery and histopathologic examination. Two-dimensional echocardiographic recordings were reviewed. Of the 293 patients, 33 had left atrial thrombi by two-dimensional echocardiographic criteria. This diagnosis was confirmed at surgery and histopathologic study in 30 (specificity 98.8%). A thrombus was not found in three patients. In 21 other patients, left atrial thrombi were present but were not detected by two-dimensional echocardiography (sensitivity 58.8%). Ten of these 21 had thrombi in the left atrial cavity. In 11 patients, thrombi were located in the left atrial appendage, all of which were missed by two-dimensional echocardiography. Excluding these 11 left atrial appendage thrombi, the sensitivity of two-dimensional echocardiography for detecting left atrial cavity thrombi was 75.0%.

ALTHOUGH the grave prognostic significance of left atrial thrombus was recognized almost a century ago,1-3 a definite clinical demonstration of left atrial thrombus became possible only recently, with left atrial angiograms.4-5 However, transseptal atrial septostomy, used for left atrial angiography, has been associated with significant risk to the patient.6,7 Pulmonary arteriography with levophase left atrial angiography is safer, but is not sensitive enough to detect the thrombus.8,9

Because of the success of M-mode echocardiography in demonstrating left atrial myxoma,10,11 it was hypothesized that left atrial clots might be demonstrable noninvasively.12,13 However, M-mode echocardiography is unreliable for diagnosing relative-

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