Two-Dimensional Echocardiography and Doppler Color Flow Mapping in the Diagnosis and Prognosis of Ventricular Septal Rupture

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Doppler color flow mapping in conjunction with two-dimensional echocardiography was used to evaluate ventricular septal rupture after myocardial infarction (seven anterior and eight inferior) in 15 patients and to correlate these findings with cardiac catheterization and surgical or autopsy data. Ventricular septal rupture was diagnosed by turbulent flow traversing the ventricular septum. The direction and velocity of shunt flow was determined by color M-mode and conventional Doppler methods. In all patients, Doppler color flow mapping correctly defined the site of septal rupture, which occurred at areas of discordant septal wall motion or “hinge points” (six posterior inlet, three anterior inlet, and six apical trabecular septum). Each of three patients with moderate tricuspid regurgitation and three of four patients with right-to-left shunting during diastole died, and all had an elevated right ventricular end-diastolic pressure. Right ventricular wall motion index was significantly higher in the patients who died compared with those who survived (mean±SEM; 2.8±0.2 vs. 2.0±0.2, p=0.012), but there was no difference in left ventricular wall motion index. The rupture size measured by Doppler color flow imaging (1.7±0.1 cm) correlated with the size determined during surgery or autopsy (1.8±0.2 cm, r=0.68, p=0.022) and the pulmonic-to-systemic shunt flow ratio by cardiac catheterization (2.4:1±0.3, r=0.74, p=0.004). Color-guided continuous-wave Doppler estimates of right ventricular systolic pressure (47±2 mm Hg) correlated with cardiac catheterization measurements (48±3 mm Hg, r=0.90, p=0.0002). Two-dimensional echocardiography combined with Doppler color flow mapping allows detection, evaluation, and prognosis in ventricular septal rupture. (Circulation 1990;81:1775-1783)

Rupture of the interventricular septum after acute myocardial infarction is a life-threatening complication that requires early and accurate diagnosis for successful management. The systolic murmur that develops after a ventricular septal rupture is often indistinguishable from the systolic murmur of mitral insufficiency, and consequently, the septal rupture may remain unrecognized. Early surgical intervention improves the prognosis in these patients.1-3

The conventional method for establishing the diagnosis of ventricular septal rupture uses cardiac catheterization and, occasionally, contrast ventriculography although noninvasive methods are currently available. Two-dimensional echocardiography has been used to diagnose ventricular septal rupture after myocardial infarction,4-6 but the yield is low because of the occurrence of artifactual echo drop-outs and the often complex three-dimensional geometry of these defects.

Pulsed Doppler echocardiography also has been used to diagnose ventricular septal rupture7-10 by documenting the presence of turbulent flow at the site of the defect, but the sites of many of these lesions are frequently not visualized in the standard echocardiographic planes, making localization technically difficult. The recently introduced technique of Doppler color flow mapping allows imaging of blood flow signals superimposed on “real-time” two-dimensional echocardiographic images. The usefulness of Doppler color flow mapping in localizing congenital defects11,12 and ventricular septal ruptures13 has been previously reported. The purpose of the current study is to assess the combination of two-dimensional echocardiography and Doppler color flow mapping in detect-
ing, localizing, and estimating the size of ventricular septal ruptures after myocardial infarction.

**Methods**

**Patient Population**

The study group consisted of 15 patients (eight men, seven women) with ventricular septal rupture after acute myocardial infarction. The patients were admitted to the coronary care unit between August 1985 and October 1989, and the mean age was 66±3 years (mean±SEM; range, 44–83 years). Seven patients had anterior and eight had inferior myocardial infarctions, all documented by standard electrocardiographic and cardiac enzyme criteria. Two patients with an anterior and one with an inferior myocardial infarction had a past history of myocardial infarction. The major presenting symptoms of the 15 patients were those of congestive heart failure, and eight patients also had chest pain. Before the echocardiographic examination, four patients had established diagnoses of ventricular septal rupture as documented by a significant step-up in oxygen saturation at the ventricular level. Eight patients were referred because of the development of a systolic murmur and a suspected ventricular septal rupture. Three patients were referred for evaluation of left ventricular function or a new systolic murmur without clinical suspicion of a ventricular septal rupture (Table 1).

**Echocardiographic Studies**

Two-dimensional echocardiography with Doppler color flow ultrasound mapping was performed with commercially available systems (Irex model 880, Aloka model 860, Toshiba model 65A, Toshiba model SSH-160A, and Hewlett-Packard model 77020A) with 2.5- and 3.5-MHz phased-array transducers.

The ventricular septum was scanned in multiple standard and nonstandard two-dimensional echocardiographic imaging planes concurrently with Doppler color flow mapping. Multiple nonstandard apical planes, variants of the standard apical four-chamber view, were used extensively. Anterior angulation of the transducer from the apical position with the aorta in view allowed visualization of the anterior ventricular septum. Posterior angulation of the transducer to the inferobasal plane allowed visualization of the posterior ventricular septum. In the posterobasal plane, the echo beam exits through the basal walls of both ventricles, and the atria are not visualized. Counterclockwise rotation of the transducer, from the standard apical four-chamber position to apical two-chamber and apical long-axis views, enabled visualization of the entire ventricular septum from base to apex. In addition, standard and nonstandard subcostal views of the septum were obtained. The apical septum was interrogated with multiple angulations to ensure that unusually situated defects did not go undetected.

A ventricular septal rupture was diagnosed when turbulent blood flow signals were observed moving from the left into the right ventricle through an area of discontinuity in the ventricular septum or across an apparently intact ventricular septum. Turbulent blood flow in the right ventricle adjacent to the septum and flow signal acceleration and convergence (defined as a localized area of increased velocity and/or aliasing) along the left ventricular side of the septum also alerted the examiner to the possibility of a ventricular septal rupture in that region. Once a suspicious area was detected, the transducer was manipulated to determine the precise origin of the abnormal flow signals. The maximum color jet width

<table>
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<tr>
<th>Patient</th>
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<th>Infarct location</th>
<th>Killip class</th>
<th>Murmur onset (days after MI)</th>
<th>Reason for echo</th>
<th>VSR size at surgery (cm)</th>
<th>Surgical outcome</th>
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MI, myocardial infarction; echo, two-dimensional echocardiography and Doppler color flow mapping; VSR, ventricular septal rupture; F, female; Ant, anterior; M, male; ?, questionable; pod, postoperative day; Inf, inferior; LV, left ventricle; OR, operating room; *, at autopsy.
at the left ventricular side of the defect was measured in multiple planes and used in correlational analysis with ventricular septal rupture size measured during surgery. If multiple jets were detected, individual jet widths were added for correlational analysis. The ventricular septal rupture sites were then categorized as being in the anterior or posterior inlet and, more distally, in the anterior or posterior trabecular septum, depending on the two-dimensional echocardiographic plane (Figure 1).17

Abnormal flow signals were analyzed to characterize the timing and direction of shunt flow in each phase of the cardiac cycle by color-guided M-mode and/or pulsed- or continuous-wave Doppler methods rather than by two-dimensional color Doppler methods because of the high update rate of the former techniques.18 The cardiac cycle was divided equally into early, middle, and late systole and diastole. These flow signals were depicted by red, blue, green, and mosaic-colored signals, depending on the direction, velocity, and degree of turbulent flow across the ventricular septal rupture.14

Continuous-wave Doppler echocardiography was performed by aligning the cursor as parallel to the direction of the ventricular septal rupture jet as possible, and the maximum velocity in systole was then obtained. The pressure gradient between left and right ventricles was calculated with the simplified Bernoulli equation,19,20 and this value was subtracted from systemic cuff blood pressure to obtain right ventricular systolic pressure. Valvular regurgitation was detected by the presence of reversed or mosaic-colored signals originating from the valve and extending into the dependent chamber in the appropriate phase of the cardiac cycle. The severity of valvular regurgitation was semiquantified by previously reported criteria.21,22

Wall motion analysis was performed with a 14-segment model of the left ventricle as described by Nishimura et al.23 Wall motion analysis numerical assignments were 0=hyperkinetic, 1=normal, 2=hypokinetic, 3=akinetic, 4=dyskinetic, and 5=aneurysmal.23 The wall motion score index was calculated by dividing the sum of the numerical wall motion segments by the number of segments analyzed. Thus, a normal left ventricular wall motion index was 1.0, and higher indexes represented worse left ventricular function. Patients with a score index of more than 2.0 have been shown to have an increased risk of short-term morbidity when compared with patients with lower indexes.23 Similarly, right ventricular function was assessed with right ventricular free wall motion in three segments (proximal, middle, and distal) and the ventricular septum in two segments (basal and distal), as described by Moore et al.24 The numerical grading system was modified to that of Nishimura et al23 for ease of comparison. Left ventricular ejection fraction was calculated with a modified cylinder-ellipse algorithm.23 Right ventricular ejection fraction was not calculated.

Cardiac Catheterization and Coronary Angiography

Fourteen of 15 patients underwent left and right cardiac catheterization and coronary angiography. One patient did not undergo cardiac catheterization or coronary angiography because of the development of shock after the onset of a new systolic ejection murmur, a situation that necessitated emergent ven-
tricular septal repair after echocardiographic confirmation of the rupture. The diagnosis of ventricular septal rupture was confirmed in 10 patients by left ventriculography. Nine of these 10 patients had oxygen saturation step-ups at the ventricular level as deduced from blood sample measurements obtained through a Swan-Ganz catheter. In the remaining four patients, ventricular septal rupture was identified by an oxygen step-up only, and ventriculography was not performed because of hemodynamic instability. The magnitude of the left-to-right shunt was estimated in 13 patients by the pulmonic-to-systemic flow ratio by the standard oximetric method.26

The infarct-related artery involved the left anterior descending coronary artery in seven patients, the right coronary artery in seven, and the posterior descending artery in one. Hemodynamically significant coronary artery disease was defined by the presence of stenotic lesions obstructing 50% or more of the normal luminal caliber. Of seven patients with an anterior myocardial infarction, one had three-vessel coronary artery disease, one had two-vessel disease, and four had single-vessel disease; in the remaining patient, coronary arteriography was not performed. Four of eight patients with an inferior myocardial infarction had three-vessel coronary artery disease, and two of these four had an associated left main coronary artery lesion; three had two-vessel disease, and the remaining patient had single-vessel disease.

Seven patients died, and of these, five had an inferior and two had an anterior myocardial infarction. Two of these patients had multivessel disease, two had two-vessel disease, two had single-vessel disease, and one patient did not undergo coronary angiography. The patients who died tended to present earlier after myocardial infarction (5.0±1.4 vs. 6.6±1.7 days) when compared with those patients who survived, but this difference was not statistically significant.

Surgery

All but one patient underwent ventricular septal rupture repair. One patient died before surgery and underwent autopsy. The ventricular septal rupture size was taken from the surgical reports, and when multiple defects were present, these measurements were added to obtain the total rupture size. Eleven of 15 patients required intra-aortic balloon pump support before surgery, and six of these patients died. The five surviving patients required intra-aortic balloon pump support for a mean time of 2.5 days after surgery. Of the remaining four patients not requiring preoperative intra-aortic balloon pump support, one required intra-aortic balloon pump support during surgery, and another of these four patients died. The ventricular septal repair was approached through the infarcted area in 13 patients, and the transatrial approach was used in the remaining patient (inferior inlet septum repair). Three patients also underwent left ventricular aneurysmectomy in addition to ventricular septal repair and coronary artery bypass surgery, and one of these patients died postoperatively.

Statistical Analysis

Data are expressed as the mean±SEM. Proportions were compared by χ² analysis, and continuous variables were analyzed by Student’s t test.

Results

Site of Ventricular Septal Rupture

Doppler color flow mapping in conjunction with two-dimensional echocardiography identified and localized the ventricular septal rupture site in all patients (Table 2). The ventricular septal ruptures in anterior myocardial infarction patients were located in the anterior midinlet septum in three and in the anteroapical trabecular septum in four (Figure 2). One patient with an anteroapical rupture had three distinct, adjacent, turbulent jets entering the right ventricle across the area of rupture, which was consistent with multiple ruptures, and this finding was confirmed at surgery (Figure 2). In patients with inferior myocardial infarctions, ventricular septal ruptures were located in the posterobasal inlet septum in four (Figure 3), the posterior midinlet septum in two, and the posterior apical trabecular septum in two (Figure 4). The echocardiographically determined sites of ventricular septal rupture were confirmed at surgery or autopsy in each patient. Thirteen patients had septal rupture orifices located at the same left and right ventricular levels. The remaining two patients had serpentine tears in the septum, with openings at different left and right ventricular levels (Figure 5). Five of eight (63%) patients with posterior ventricular septal ruptures (inferior myocardial infarctions) died compared with two of seven (29%) patients with anterior ruptures (anterior myocardial infarctions), but the difference was not statistically significant.

Septal ruptures were localized to the junction of two areas with discordant wall motion or “hinge points” in all patients. The maximal wall motion discordance of adjacent segments at the ventricular septal rupture site was at least two grades in each patient, with the exception of one in whom the difference was only one grade (the ventricular septal rupture occurred at the junction of a hypokinetic and an akinetic area). Ventricular septal rupture occurred at the junction of dyskinetic or aneurysmal segments and adjacent hypokinetic segments in seven patients and at the junction of dyskinetic or aneurysmal segments and adjacent normal segments in seven patients.

Flow Acceleration

Flow acceleration proximal to the ventricular septal rupture was detected in 14 patients, and this feature aided in localization of the rupture site during the Doppler color flow examination. One patient with a large rupture (2.5 cm) had no flow acceleration.
**TABLE 2. Echocardiographic and Catheterization Data in 15 Patients With Ventricular Septal Rupture**

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<th>Patient</th>
<th>VSR site</th>
<th>VSR size (cm)</th>
<th>LVEF (%)</th>
<th>LVWMI</th>
<th>RVWMI</th>
<th>Grade</th>
<th>Doppler pressure in RV (mm Hg)</th>
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VSR, Ventricular septal rupture; LVEF, left ventricular ejection fraction; LVWMI, left ventricular wall motion index; RVWMI, right ventricular wall motion index; MR, mitral regurgitation; TR, tricuspid regurgitation; RV, right ventricle; LAD, left anterior descending; CX, circumflex; RCA, right coronary artery; QP/QS, pulmonic-to-systemic flow ratio; LV, left ventricle; RV, right ventricle; Ant, anterior; Post, posterior; 2, moderate; 1, mild; ND, not done; NA, not available.

**Size of Rupture**

The mean maximal color flow jet width at the left side of the septal rupture was 1.6±1.0 cm (range, 0.8–2.3 cm). This Doppler color flow–echocardiographic estimate of ventricular septal rupture size correlated fairly well (r=0.68, p=0.022) with the surgical or autopsy estimate of defect size (1.8±0.2 cm). The ventricular septal rupture size measured by Doppler color flow imaging also correlated fairly well (r=0.74, p=0.004) with the estimate of pulmonic-to-systemic flow ratio obtained by cardiac catheterization (2.4±0.3; range 1.2:1 to 4.4:1). The ventricular septal rupture size (1.7±0.1 vs. 1.4±0.2 cm) and pulmonic-to-systemic flow ratio (2.2±0.1 vs. 2.6±0.5) were not significantly different in those patients who survived compared with these values in the patients who died.

**Shunt Direction**

Left-to-right, turbulent, mosaic-colored flow signals across the septal rupture were present in all patients throughout systole. Low-velocity left-to-right laminar flow was observed throughout diastole in 10 patients.

**Figure 2.** Doppler color flow imaging–two-dimensional echocardiogram (modified low-parasternal long-axis view) showing mosaic-colored flow signals crossing the anterior apical ventricular septum through three defects (1–3), which were confirmed at surgery. AO, aorta; IVS, interventricular septum; LV, left ventricle; RV, right ventricle; VSD, ventricular septal defect.

**Figure 3.** Doppler color flow imaging–two-dimensional echocardiogram in ventricular septal rupture. Modified apical four-chamber plane during systole demonstrates blue flow signals in the left ventricle (LV), which cross a large postero-basal ventricular septal defect (VSD) into the right ventricle (RV). Note the presence of mosaic-colored signals in RV indicative of flow turbulence. LA, left atrium.
(Figure 6), and three additional patients had right-to-left shunting across the rupture in early to mid-diastole that interrupted left-to-right laminar diastolic flow (Figure 7). Two of the latter three patients died. One patient had pandiastolic right-to-left shunt flow and also died. No flow signals were detected during diastole in one patient with a serpiginous rupture.

**Right Ventricular Pressure Estimate**

The spectral tracings for estimation of right ventricular systolic pressure were adequate in 11 of 15 patients. There was good correlation ($r=0.90$, $p=0.0002$) between the continuous-wave Doppler-derived right ventricular systolic pressure (47±2 mm Hg; range, 30–60 mm Hg) and right ventricular pressure measurement obtained during cardiac catheterization (48±3 mm Hg; range, 30–70 mm Hg). Six of these 11 patients had right ventricular systolic pressures higher than 50 mm Hg as measured by Doppler color flow methods (range, 50–60 mm Hg).
**Atrioventricular Valvular Regurgitation**

Atrioventricular valvular regurgitation was observed in all patients. Mitral regurgitation was graded by Doppler color flow methods as mild in 13 (87%) and moderate in two (13%) patients. No mitral valve was thickened, but two patients had calcified mitral annuli. Tricuspid regurgitation was present in 14 of 15 (93%) patients and was mild in 11 and moderate in three by Doppler color flow methods. All three patients with moderate tricuspid regurgitation died (one had associated diastolic right-to-left shunting). The tricuspid valve appeared structurally normal in all patients, and no patient required concomitant valve replacement for valvular regurgitation. Ten of 11 patients with mild tricuspid regurgitation had systolic right ventricular pressures higher than 40 mm Hg (right ventricular pressures were not available in one patient). The occurrence of moderate versus mild tricuspid regurgitation was significantly increased in those patients who died (p<0.05). The occurrence of mitral regurgitation was similar in the patients who died and those who survived.

**Left and Right Ventricular Function**

Left ventricular ejection fraction in patients with anterior myocardial infarctions was significantly lower than in those with inferior myocardial infarctions (34±2 vs. 42±2%, p=0.027). The right ventricular wall motion index was more abnormal in patients with inferior myocardial infarctions than in patients with anterior myocardial infarctions (2.9±0.1 vs. 1.9±0.2, p<0.001).

Left ventricular ejection fraction in the patients who died was similar to that of surviving patients (38±2 vs. 39±3%, p=NS). The mean left ventricular wall motion index was elevated (≥2.0) in both groups of patients and was not significantly higher in the patients who died compared with those who survived (2.4±0.1 vs. 2.3±0.1, p=NS). The two patients with the poorest left ventricular wall motion index (2.7) had anterior myocardial infarctions, and both died. The right ventricular wall motion index was significantly higher in the patients who died compared with those who survived (2.8±0.2 vs. 2.0±0.2, p=0.012).

**Follow-up Studies**

Six patients had echocardiographic evaluations with two-dimensional echocardiography and Doppler color flow mapping on postoperative days 3, 6, 7, 7, 9, and 20, respectively, and one patch leak was detected. The latter patient developed a systolic murmur on the ninth day after posterobasal ventricular septal repair, and Doppler color flow mapping at that time revealed two small flow jets moving from left to right across the ventricle in the region of the ventricular septal repair, denoting a patch leak. The combined maximal color jet was approximately 4 mm wide, and the leak was managed conservatively.

**Discussion**

Ventricular septal ruptures after myocardial infarction are generally not smooth, well-defined orifices, as they are in congenital ventricular septal defects, but usually the former occur as linear lacerations, serpiginous tunnels, or multiple perforations in the necrotic myocardium.27 The reported sensitivity of two-dimensional echocardiography in detecting ventricular septal rupture ranges from 46%28 to 100%,6 but this method has limitations in detecting small, multiple, or serpiginous tears in the septum29 because of artificial dropouts and lateral resolution limitations. The recently developed technique of Doppler color flow mapping, in which color flow images are superimposed on "real-time" two-dimensional images, would be expected to obviate some of these limitations. A previous communication from our laboratory13 demonstrated the feasibility of this technique in demonstrating ventricular septal rupture by noting flow signals moving from the left to the right ventricle. The present series with a larger study population confirms the usefulness of Doppler color flow methods in making an accurate and rapid diagnosis of this entity. Doppler color flow mapping allowed rapid and reliable localization of the ventricular septal rupture site in all current study patients and, when present, demonstrated multiple perforations at the rupture site. The sites of serpiginous tears were readily demonstrated. Additionally, the color jet width at the ventricular septal rupture site had a statistically significant correlation with the rupture size measured during surgery and to the pulmonic-to-systemic flow ratio.

The use of Doppler color flow mapping conferred several advantages on the examiner when a ventricular septal rupture was suspected. The turbulent systolic blood flow across the rupture resulted in a striking display of mosaic-colored signals and aliasing within the right ventricular cavity, which was immediately apparent to the operator and helped establish the diagnosis. The convergence of flow into the rupture at the left side of the septum appeared in the form of the flow acceleration or convergence effect30 on the sector image, which was extremely useful in identifying serpiginous defects. The display of an abnormal flow stream by Doppler color flow methods also permitted parallel alignment of the continuous-wave cursor with the jet, so that a simple and reliable estimate of right ventricular and pulmonary artery systolic pressures could be obtained.31 Cursor alignment with the ventricular septal rupture flow jet associated with inferior myocardial infarction was difficult because of the location of the rupture in a nonstandard plane; therefore, the peak gradient across the defect could not be assessed in four of the current study patients.

In isolated congenital ventricular septal defects, the direction and timing of shunt flow depends primarily on the defect size,32 in contrast to defects created by ventricular septal rupture, in which right and left ventricular functions determine the direction
of shunt flow. The current study results agree with conventional Doppler studies of Bhatia et al and Miyatake et al regarding high-velocity shunt flow during systole and low-velocity shunt flow during diastole. Bhatia et al described one patient with a possible early diastolic right-to-left shunt, which was postulated to be an artifact of transducer orientation. In the current study, right-to-left shunting also occurred during diastole in four patients. Three of these patients had the highest right ventricular end-diastolic pressures (20–25 mm Hg) in our series, and each died. The remaining patient, also with a high right ventricular end-diastolic pressure (12 mm Hg), survived. Although the number of patients is small, diastolic right-to-left shunting appears to be associated with considerable right ventricular dysfunction and high mortality. In patients for whom we obtained adequate spectral tracings, the Doppler-derived right ventricular systolic pressure correlated well with the pressures recorded during cardiac catheterization. Thus, pressure hemodynamics across a ventricular septal rupture can be studied noninvasively at the bedside and may aid in therapeutic interventions.

Mitrail regurgitation due to chordal rupture or papillary muscle dysfunction also presents as a systolic murmur in the postmyocardial infarction period, and clinical differentiation from ventricular septal rupture may be difficult, which may result in the delay of appropriate treatment. Mild or moderate mitral regurgitation was present in each current study patient and was presumably due to papillary muscle dysfunction since none of these patients had chordal or papillary muscle rupture, and only two had mitral annular calcification. The Doppler color flow demonstration of flow signals across the ventricular septal rupture rapidly and easily distinguishes it from flow signals representing mitral regurgitation and thus is critical for therapeutic management. Furthermore, the severity of associated mitral regurgitation is easily graded semiquantitatively as previously described.

In most patients, the anterior two thirds of the ventricular septum is supplied by the left anterior descending coronary artery while the posterior one third is supplied by the posterior descending coronary artery. Ventricular septal rupture is expected to occur either above or below the junctional area of the anterior two thirds and the posterior one third of the ventricular septum in a horizontal J-shaped zone and will depend on whether the infarct-related artery is the left anterior descending or the posterior descending coronary artery (Figure 1). The shear and strain imparted to the infarcted myocardium and peri-infarct zone by contraction of adjacent healthy or less-involved myocardium may be responsible for septal rupture. In the current study, ventricular septal rupture occurred at junctional areas of discordant wall motion. These junctional areas or hinge points should be searched meticulously when a ventricular septal rupture is suspected and may further aid in rapid detection of this condition by Doppler color flow mapping.

Ventricular septal defects outside the infarcted region and surrounded by normally contracting ventricular segments would be expected to be congenital rather than acquired, as illustrated by the case of a 56-year-old man (not included in the present study) who presented with an inferior myocardial infarction and a loud systolic murmur clinically suggestive of ventricular septal rupture. Two-dimensional and Doppler color flow echocardiography localized the defect to the normally contracting perimembranous region remote from the site of infarction, which was consistent with a congenital origin, and unnecessary surgery was prevented.

Tricuspid regurgitation occurred very frequently in association with ventricular septal rupture and was presumably caused by high right ventricular pressure or myocardial ischemia. Although the study population is small, in two current study patients, right-to-left diastolic shunting and moderate tricuspid regurgitation did correlate with high right ventricular end-diastolic pressure, which was associated with a high mortality, suggesting that this is an important prognostic factor in ventricular septal rupture.

Doppler color flow imaging assessment of the site and size of ventricular septal rupture had no correlation with mortality. Of the two-dimensional echocardiographic findings, left ventricular ejection fraction and wall motion index did not correlate with patient outcome. Poor right ventricular wall motion indexes were associated with increased mortality, which has also been reported by other investigators.

In conclusion, two-dimensional echocardiography combined with Doppler color flow mapping is useful in the assessment of ventricular septal rupture. The latter technique allows rapid and accurate detection and characterization of ventricular septal rupture and differentiation of septal rupture from mitral regurgitation. These techniques also allow evaluation of right ventricular dysfunction, tricuspid regurgitation, and shunt direction, which appear to be important prognostic factors in ventricular septal rupture.

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Echocardiography in Ventricular Septal Rupture


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