

## Review Article

*Current Concepts***VENTRICULAR SEPTAL RUPTURE  
AFTER ACUTE MYOCARDIAL  
INFARCTION**YOCHAI BIRNBAUM, M.D., MICHAEL C. FISHBEIN, M.D.,  
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**W**HEN ventricular septal rupture complicates acute myocardial infarction, the mortality is high. Reperfusion therapy has reduced the incidence of septal rupture. However, rapid diagnosis, aggressive medical management, and surgical intervention are required to optimize recovery and survival. This review summarizes information on septal rupture in both the era before thrombolytic therapy and after the advent of reperfusion therapy.

**INCIDENCE**

In the era before reperfusion therapy, septal rupture complicated 1 to 3 percent of acute myocardial infarctions.<sup>1-5</sup> Among the 41,021 patients in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial, ventricular septal rupture was suspected in 140 patients (0.34 percent) and confirmed by a retrospective review in 84 (0.2 percent).<sup>6</sup> Thus, reperfusion therapy has decreased the incidence of septal rupture.<sup>6</sup>

**RISK FACTORS**

Septal rupture occurs more frequently with anterior than other types of acute myocardial infarction.<sup>2,6-9</sup> Risk factors for septal rupture in the era before thrombolytic therapy included hypertension,<sup>10,11</sup> advanced age (60 to 69 years),<sup>10</sup> female sex,<sup>10,12</sup> and the absence of a history of angina or myocardial infarction.<sup>1,2,13-15</sup> Angina or infarction may lead to myocardial preconditioning as well as to the development of coronary

collaterals, both of which reduce the likelihood of septal rupture.<sup>15</sup> In patients undergoing thrombolysis, advanced age, female sex, and the absence of smoking are often associated with an increased risk of septal rupture,<sup>6</sup> whereas the absence of antecedent angina has not been associated with an increased risk.<sup>8</sup> In the GUSTO-I trial, there was a nonlinear relation between the systolic and diastolic blood pressures at enrollment and septal rupture, since hypertension (a blood pressure of more than 130/75 mm Hg) and extensive myocardial infarction and right ventricular infarction (which are causes of hypotension) are also risk factors for septal rupture.<sup>6</sup>

**PATHOGENESIS**

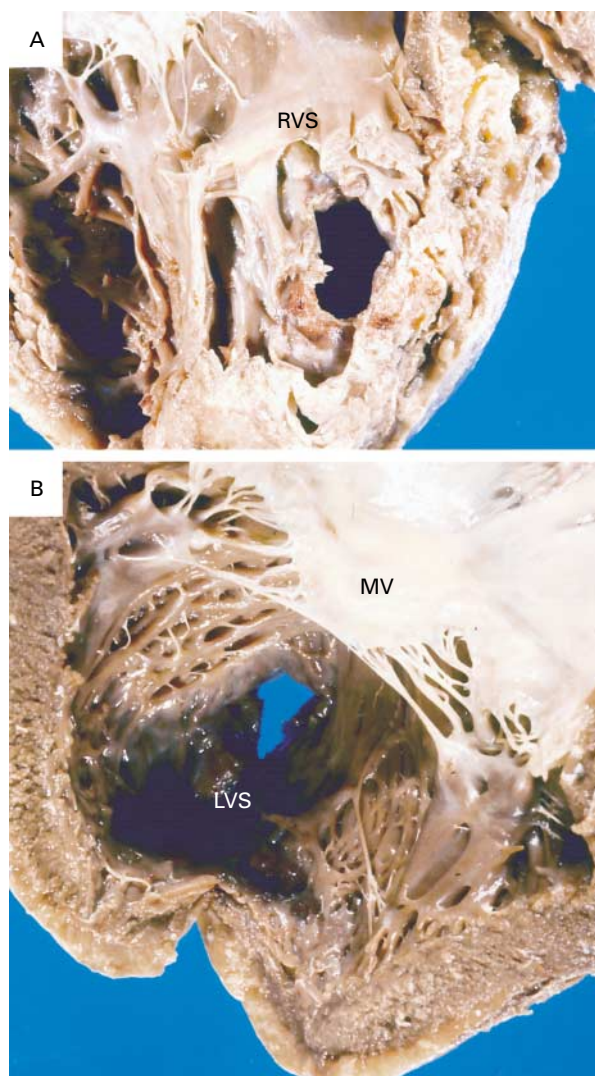
The septum adjacent to the rupture is often thin and necrotic. Without reperfusion, coagulation necrosis develops within the first three to five days after infarction, with numerous neutrophils entering the necrotic zone. The neutrophils undergo apoptosis and release lytic enzymes, hastening the disintegration of necrotic myocardium.

The pathogenic process of the rupture changes over time. During the first 24 hours, coagulation necrosis is just beginning and there are relatively few neutrophils within the infarcted tissue. Early ruptures occur in infarcts with large intramural hematomas that dissect into tissue and rupture. If patients survive for several weeks, the septum becomes fibrotic.

Becker and van Mantgem classified the morphology of free-wall rupture into three types, which are also relevant to ventricular septal rupture<sup>16</sup>: type I ruptures have an abrupt tear in the wall without thinning; in type II, the infarcted myocardium erodes before rupture occurs and is covered by a thrombus; and type III has marked thinning of the myocardium, secondary formation of an aneurysm, and perforation in the central portion of the aneurysm.

The size of septal rupture ranges from millimeters to several centimeters. Morphologically, septal rupture is categorized as simple or complex. Figure 1 shows a simple septal rupture with a discrete defect and a direct through-and-through communication across the septum. The perforation is at the same level on both sides of the septum. Extensive hemorrhage with irregular, serpiginous tracts within necrotic tissue characterizes complex septal rupture (Fig. 2).<sup>7,13</sup> Septal ruptures in patients with anterior myocardial infarction are generally apical and simple (Fig. 1 and 3). Conversely, in patients with inferior myocardial infarction, septal ruptures involve the basal inferoposterior sep-

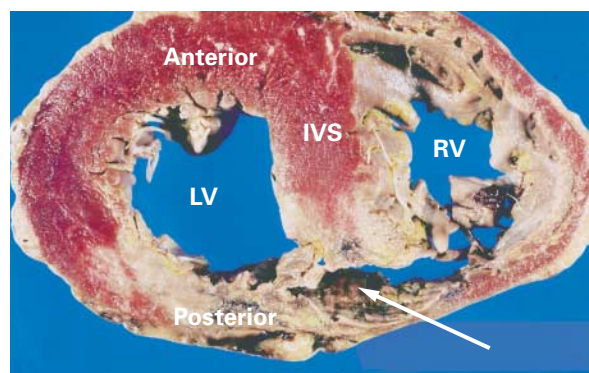
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**Figure 1.** Findings at Autopsy in a Patient with a Simple Ventricular Septal Rupture.

There is a discrete defect with a direct through-and-through communication across the septum. The perforation is at the same level on both sides of the septum: the left ventricular aspect of the interventricular septum (LVS), and the right ventricular aspect of the interventricular septum (RVS). MV denotes mitral valve.

tum and are often complex (Fig. 2 and 4; also see the video clips in the Supplementary Appendix available with the full text of this article at <http://www.nejm.org>).<sup>3,7,13</sup> Occasionally, muscles of the ventricular free wall or papillary muscles may tear,<sup>7,14,17,18</sup> especially in the case of complex septal ruptures.<sup>7</sup> Ventricular septal ruptures associated with an inferior or anterior myocardial infarction generally involve right ventricular infarction.<sup>12</sup>



**Figure 2.** Gross Findings in a Patient with a Posterior Ventricular Septal Rupture.

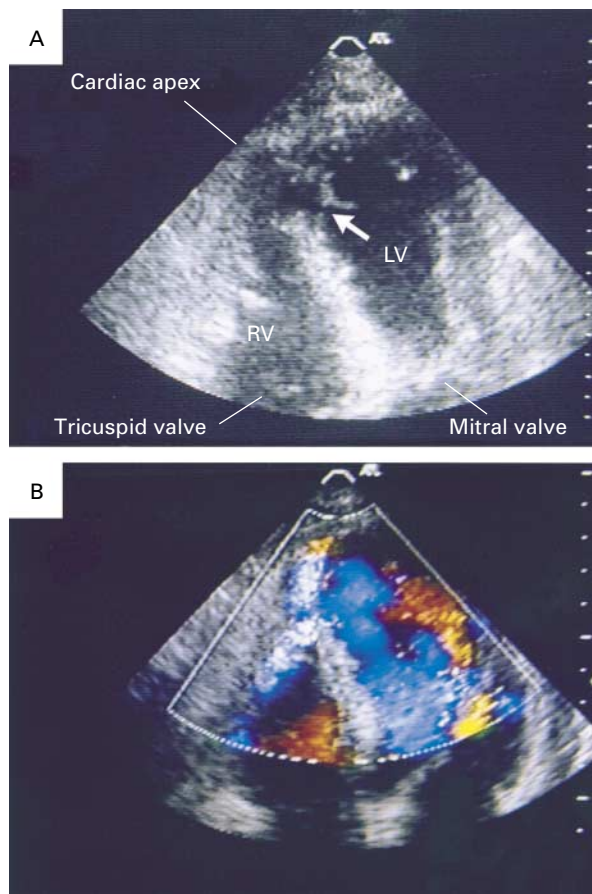
There is an infarction involving the basal inferior septum, the basal posteroinferior wall, and the right ventricle (RV). The ventricular septal rupture (arrow) is complex, with an irregular, ser-piginous tract at the junction of the inferior wall and the inter-ventricular septum (IVS). LV denotes left ventricle.

## HEMODYNAMICS

Septal rupture results in a left-to-right shunt, with right ventricular volume overload, increased pulmonary blood flow, and secondary volume overload of the left atrium and ventricle. As left ventricular systolic function deteriorates and forward flow declines, compensatory vasoconstriction leads to increasing systemic vascular resistance, which, in turn, increases the magnitude of the left-to-right shunt. The degree of shunting is determined by the size of the septal rupture, the level of pulmonary vascular resistance and systemic vascular resistance and the ratio of the two, and left ventricular and right ventricular function. As the left ventricle fails and the systolic pressure declines, left-to-right shunting decreases and the fraction of the shunt diminishes.

## ANGIOGRAPHIC FINDINGS

Some studies have found that septal rupture is associated with multivessel coronary artery disease.<sup>2,7</sup> However, others found a high prevalence (54 percent) of single-vessel disease among patients with ventricular septal rupture.<sup>8,19</sup> Ventricular septal rupture is likely to be associated with total occlusion of the infarct-related artery.<sup>3,6,8</sup> In the GUSTO-I study, total occlusion of the infarct-related artery was documented in 57 percent of patients with ventricular septal rupture, as compared with 18 percent of those without ventricular septal rupture.<sup>6</sup> Collaterals are less often evident in patients with ventricular septal rupture,<sup>2,8,15</sup> supporting the hypothesis that collateral circulation reduces the risk of rupture of the cardiac free wall as well as septal rupture.<sup>15</sup>

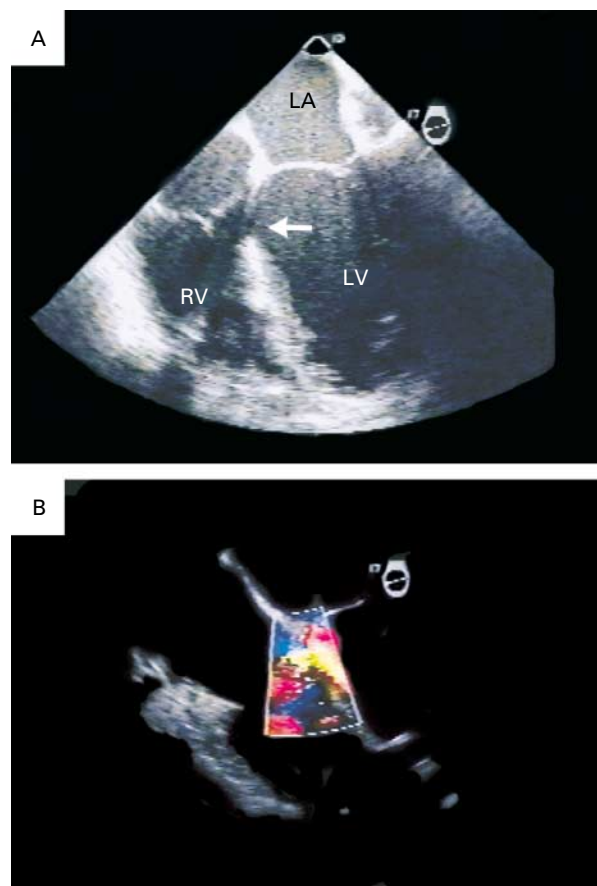


**Figure 3.** Transthoracic Two-Dimensional Apical Four-Chamber Views in a Patient with Ventricular Septal Rupture Complicating an Anterior Acute Myocardial Infarction.

In Panel A, the ventricular septal rupture is apical and simple (arrow). RV denotes right ventricle, and LV left ventricle. The sites of both the right and the left ventricular perforation are at the same level. In Panel B, color Doppler ultrasonography shows turbulent flow (bright blue mosaic) from the left ventricle to the right ventricle through the interventricular septal defect.

### TIME COURSE

Without reperfusion, septal rupture generally occurs within the first week after infarction.<sup>3,5,7,19,20</sup> As explained above, there is a bimodal distribution of septal rupture, with a high incidence on the first day and on days 3 through 5 and rarely more than two weeks after infarction. The median time from the onset of symptoms of acute myocardial infarction to rupture is generally 24 hours or less in patients who are receiving thrombolysis.<sup>21</sup> The median time from the onset of infarction to septal rupture was 1 day (range, 0 to 47; 94 percent of cases were diagnosed within 1 week) in the GUSTO-I trial<sup>6</sup> and 16 hours in the Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock (SHOCK) trial.<sup>22</sup> Al-



**Figure 4.** Transesophageal Four-Chamber Views in a Patient with a Ventricular Septal Rupture Complicating an Acute Posterior Myocardial Infarction.

In Panel A, the ventricular septal rupture (arrow) is basal and at the junction between the basal inferior septum and the inferoposterior wall. In Panel B, a color Doppler image shows the shunt across the ventricular septal rupture. LA denotes left atrium, LV left ventricle, and RV right ventricle.

though thrombolytic therapy reduces the size of the infarct, it may in some cases promote hemorrhagic dissection in the myocardium, accelerating the onset of septal rupture.

### CLINICAL MANIFESTATIONS

Symptoms of septal rupture include chest pain, shortness of breath, and those associated with low cardiac output and shock.<sup>2,14</sup> Acute septal rupture produces a harsh, loud holosystolic murmur along the left sternal border, radiating toward the base, apex, and right parasternal area, and a palpable parasternal thrill in half of patients.<sup>19,23</sup> With cardiogenic shock and a low-output state complicating septal rupture, there is rarely a thrill, and the murmur is difficult to identify

because turbulent flow across the defect is reduced. Right and left ventricular S<sub>3</sub> gallops are common. The pulmonic component of the second heart sound is accentuated by pulmonary hypertension. Tricuspid regurgitation may also be present. Biventricular failure generally ensues within hours or days.

As compared with acute mitral regurgitation, septal rupture has a loud murmur, a thrill, and right ventricular failure but is less often characterized by severe pulmonary edema. In patients with a low cardiac output, distinguishing between these two entities can be difficult. In addition, severe mitral regurgitation may occur in 20 percent of patients with septal rupture.<sup>24-26</sup>

### DIAGNOSIS

Pump failure in patients with myocardial infarction may be related to the major mechanical complications, such as ventricular septal rupture, papillary-muscle rupture, or free-wall rupture (Table 1). Alternatively, it results from the infarction or ischemia of a large area, ischemic mitral regurgitation, right ventricular dysfunction, or hypovolemia. Doppler echocardiography is generally diagnostic (Fig. 2 and 4; also see the video clips at <http://www.nejm.org>).<sup>26,27</sup> Doppler techniques can be used to define the site and size of septal rupture, left and right ventricular function, estimated right ventricular systolic pressure, and the

left-to-right shunt.<sup>28</sup> The sensitivity and specificity of color Doppler echocardiography have been reported to be as high as 100 percent.<sup>26,27</sup> In severely ill patients who are receiving assisted ventilation, the image quality of transthoracic echocardiography may not be sufficient for diagnosis, and transesophageal echocardiography is more sensitive (Fig. 2).<sup>29-31</sup>

Pulmonary-artery catheterization can be helpful. In patients with a septal rupture, the increase in oxygen saturation occurs within the right ventricle and not only in the pulmonary artery. Severe mitral regurgitation may result in an increase in oxygen saturation in the peripheral pulmonary arteries.<sup>32</sup> The presence of large V waves in the pulmonary-capillary wedge tracing is a nonspecific finding that also occurs with mitral regurgitation and with poor left ventricular compliance.<sup>33</sup>

Left ventriculography can also be used to diagnose septal rupture. Coronary angiography is useful for assessing the coronary anatomy if concomitant revascularization is being considered. Radionuclide scintigraphy is an alternative noninvasive technique for diagnosing septal rupture, assessing ventricular function, and calculating the size of the intracardiac shunt.<sup>34</sup>

### MEDICAL THERAPY

Medical therapy consists of mechanical support with an intraaortic balloon pump, afterload reduc-

**TABLE 1.** CHARACTERISTICS OF VENTRICULAR SEPTAL RUPTURE, RUPTURE OF THE VENTRICULAR FREE WALL, AND PAPILLARY-MUSCLE RUPTURE.\*

CHARACTERISTIC	VENTRICULAR SEPTAL RUPTURE	RUPTURE OF VENTRICULAR FREE WALL	PAPILLARY-MUSCLE RUPTURE
Incidence	1–3% without reperfusion therapy, 0.2–0.34% with thrombolytic therapy, 3.9% among patients with cardiogenic shock	0.8–6.2%, thrombolytic therapy does not reduce risk; primary PTCA seems to reduce risk	About 1% (posteromedial more frequent than anterolateral papillary muscle)
Time course	3–7 days without reperfusion therapy; median, 24 hr with thrombolysis	1–7 days without reperfusion therapy; mean, 2.7 days with thrombolysis	Median, 1 day (range, 1–14)
Clinical manifestations	Chest pain, shortness of breath, hypotension	Anginal, pleuritic, or pericardial chest pain, syncope, hypotension, arrhythmia, nausea, restlessness, hypotension, sudden death	Abrupt onset of shortness of breath and pulmonary edema; hypotension
Physical findings	Harsh holosystolic murmur, thrill (+), S <sub>3</sub> , accentuated 2nd heart sound, pulmonary edema, RV and LV failure, cardiogenic shock	Jugulovenous distention (29% of patients), pulsus paradoxus (47%), electromechanical dissociation, cardiogenic shock	A soft murmur in some cases, no thrill, variable signs of RV overload, severe pulmonary edema, cardiogenic shock
Echocardiographic findings	Ventricular septal rupture, left-to-right shunt on color flow Doppler echocardiography through the ventricular septum, pattern of RV overload	>5 mm pericardial effusion not visualized in all cases, layered, high-acoustic echoes within the pericardium (blood clot), direct visualization of tear, signs of tamponade	Hypercontractile LV, torn papillary muscle or chordae tendineae, flail leaflet, severe mitral regurgitation on color flow Doppler echocardiography
Right-heart catheterization	Increase in oxygen saturation from the RA to RV, large V waves	Ventriculography insensitive, classic signs of tamponade not always present (equalization of diastolic pressures among the cardiac chambers)	No increase in oxygen saturation from the RA to RV, large V waves, very high pulmonary-capillary wedge pressures

\*PTCA denotes percutaneous transluminal coronary angioplasty, RA right atrium, RV right ventricle, and LV left ventricle.

tion, diuretics, and usually, inotropic agents.<sup>35,36</sup> Oxygenation should be maintained with the administration of oxygen by mask, continuous positive airway pressure, bilevel positive airway pressure, or intubation with mechanical ventilation.<sup>37</sup> Nitroprusside may reduce left-to-right shunting and improve cardiac output, but it may also cause hypotension. Its use is contraindicated in patients with acute renal failure. Patients with hypotension often need inotropic agents and vasopressors to maintain arterial blood pressure. However, an increase in left ventricular pressure increases left-to-right shunting. Attempts to stabilize the patient's condition with medical therapy are only temporizing, because most patients have a rapid deterioration and die. Most patients require surgical intervention. Even patients whose condition appears to be clinically stable are at risk for abrupt hemodynamic deterioration, because the size of the septal rupture can increase without warning.<sup>3</sup> The mortality rate among patients with septal rupture who are treated conservatively without mechanical closure is approximately 24 percent in the first 24 hours, 46 percent at one week, and 67 to 82 percent at two months.<sup>38,39</sup> Lemery et al. reported a 30-day survival rate of 24 percent among medically treated patients, as compared with a rate of 47 percent among those treated surgically.<sup>19</sup>

### MECHANICAL CLOSURE

It was long believed that shortly after an acute myocardial infarction, the myocardium was too fragile for the safe repair of the septal rupture. A waiting period of three to six weeks before surgery was standard to allow the margins of the infarcted muscle to develop a firm scar to facilitate the surgical repair.<sup>40</sup> However, many patients died while awaiting surgery or underwent emergency surgery after sudden decompensation.<sup>19,37</sup> A 1977 series of 43 patients reported an increased survival rate after early surgical repair,<sup>37</sup> and these findings have since been confirmed by others.<sup>41</sup>

Current guidelines of the American College of Cardiology–American Heart Association for the treatment of patients with acute myocardial infarction recommend immediate operative intervention in patients with septal rupture, regardless of their clinical status.<sup>36</sup> Surgical management is based on six goals. Hypothermic cardiopulmonary bypass with optimal myocardial protection should be promptly established.<sup>35</sup> The septal rupture should be approached through the infarct, and all necrotic and friable margins of the septum and ventricular walls should be excised to avoid postoperative hemorrhage, a residual septal defect, or both.<sup>35</sup> Prosthetic material should be used to reconstruct the septum and the ventricular walls, and the geometric configuration of the ventricles and function of the heart should be preserved.<sup>35</sup> The septal

rupture should be closed by a method chosen according to its location — apical, anterior, or posterior.<sup>35</sup> The mitral valve should undergo concomitant repair or replacement if indicated. Coronary-artery bypass grafting should be performed in patients with multivessel coronary artery disease, although there is no need to bypass the artery responsible for the infarcted septum.<sup>35</sup> Newer surgical techniques, which avoid direct incision of the ventricles, can be used in selected patients. Exposure of the septum through the right atrium may reduce the risk associated with early surgery by averting additional damage to the left ventricle and decreasing the risk of postoperative bleeding.<sup>42</sup>

In selected patients, percutaneous closure of septal rupture with catheter-based devices may be an alternative to surgical repair.<sup>43-45</sup> Although only a few case reports have been described to date, several points should be stressed. As the site of the septal rupture in patients with myocardial infarction becomes surrounded by fragile necrotic tissue, attempts to pass the closure device through the site may increase the size of the rupture. The septal rupture in patients with anterior infarction is usually near the apex, whereas in patients with inferior infarction it is usually near the base of the right and left ventricular free wall. Thus, it may not be possible to open the wings of catheter-based closure devices such as the Amplatzer (AGA Medical) completely without distorting the right or left ventricle. Moreover, in patients with inferior infarction, the septal ruptures are usually basal and thus close to the tricuspid and mitral valves. Consequently, positioning and opening the sealing devices may markedly impinge on these valves and cause tricuspid or mitral regurgitation (or both).

### POSTOPERATIVE CARE

Postoperative care is directed toward reversing cardiogenic shock and incipient multiorgan failure, particularly in elderly patients. The management of right ventricular failure is aimed at reducing afterload while maintaining systemic arterial pressure. Optimal management includes continuation of an intraaortic balloon pump, pharmacologic inotropic support, control of arrhythmias, optimization of volume status, correction of metabolic acidosis and coagulopathy, institution of dialysis for oliguric renal failure, reversal of the catabolic state with nasogastric-tube feeding, and slow weaning from ventilatory support once all hemodynamic and metabolic variables have been stabilized.

Echocardiography is essential to assess the completeness of the repair, to determine whether septal rupture has recurred as a result of dehiscence of the interventricular patch, and to evaluate right and left ventricular function, since such knowledge will be used to guide pharmacologic and mechanical support.<sup>46</sup> Patients with severe and persistent hypoxemia



and systemic organ desaturation should be evaluated for a patent foramen ovale.<sup>47</sup>

### PROGNOSIS

In the prethrombolytic era, outcomes after septal rupture were extremely poor, with an in-hospital mortality rate of approximately 45 percent among surgically treated patients and 90 percent among those treated medically.<sup>3,48,49</sup> In the SHOCK trial, the in-hospital mortality rate was significantly higher among patients in cardiogenic shock as a result of septal rupture than among patients with all other categories of shock (87.3 percent, as compared with 59.2 percent among those with pure left ventricular failure and 55.1 percent among those with acute mitral regurgitation).<sup>50</sup> Surgical repair was performed in 31 patients with septal rupture (56 percent), 21 of whom underwent concomitant bypass surgery, and 6 of whom (19 percent) survived. Of the 24 patients who were treated medically, only 1 survived.<sup>22</sup> Pretre et al. reported that among 54 patients who underwent surgical repair of a ventricular septal rupture, 28 underwent concomitant coronary-artery bypass surgery (52 percent), 14 died after surgery (26 percent), and 19 (35 percent) died during follow-up (mean follow-up, 42 months).<sup>51</sup> The cumulative survival rate (including perioperative deaths) was 78 percent at 1 year, 65 percent at 5 years, and 40 percent at 10 years. Thus, the mortality rate among patients with ventricular septal rupture remains extremely high, even in the reperfusion era. In the GUSTO-I trial, the 34 patients who underwent surgical repair had a lower 30-day mortality rate than the 35 patients who were treated medically (47 percent vs. 94 percent,  $P < 0.001$ ) as well as a lower 1-year mortality rate (53 percent vs. 97 percent,  $P < 0.001$ ).<sup>6</sup> However, selection bias may have accentuated the differences in the rates.

For patients who survive surgery, the long-term prognosis is relatively good.<sup>6,47</sup> Crenshaw et al. reported a mortality rate of only 6 percent among patients who survived the first 30 days after surgery.<sup>6</sup> Among 60 patients who survived surgical repair, the 5-year survival rate was 69 percent, the 10-year survival rate was 50 percent, and the 14-year survival rate was 37 percent.<sup>49</sup> Eighty-two percent of these patients were in New York Heart Association class I or II at follow-up, and angina and other medical problems were not prevalent.

The immediate preoperative hemodynamic status is a major determinant of the postoperative outcome,<sup>2,37</sup> rather than the ejection fraction or the size of the intracardiac shunt. In the GUSTO-I trial, all 8 patients with septal rupture who were in Killip class III or IV at presentation died, as compared with 53 of 74 patients (72 percent) who were in Killip class I or II at presentation.<sup>6</sup> Among patients who are undergoing

surgical repair, the prognosis is associated with the preoperative systolic blood pressure and right atrial pressure and the duration of cardiopulmonary bypass.<sup>35,52</sup> Patients whose systemic arterial blood pressure remained high had the best prognosis. The combination of an elevated right atrial pressure with a low systemic blood pressure was associated with an extremely poor prognosis.<sup>2,52</sup> Right ventricular function is also a predictor of survival.<sup>5</sup> Others report that renal failure<sup>20</sup> and diabetes mellitus<sup>35</sup> are strong negative predictors of survival after surgery. Patients with septal rupture complicating inferior rather than anterior myocardial infarction have the poorest outcome.<sup>2,5,20</sup> No correlation has been demonstrated between the risk of early death and age or sex.<sup>20</sup> Blanche et al. found that preoperative use of an intraaortic balloon pump reduced immediate postoperative mortality, but it was not associated with an improved long-term prognosis.<sup>35</sup>

The development of a residual or recurrent septal defect is reported in up to 28 percent of patients who survive repair and is associated with high mortality.<sup>47</sup> In asymptomatic patients who have a small residual left-to-right shunt, conservative therapy may be warranted.<sup>47</sup> In patients who have clinical heart failure or a pulmonary–systemic shunt fraction of more than 2.0, repeated surgical intervention is clearly indicated to improve the outcome.<sup>47</sup>

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### REFERENCES

1. Pohjola-Sintonen S, Muller JE, Stone PH, et al. Ventricular septal and free wall rupture complicating acute myocardial infarction: experience in the Multicenter Investigation of Limitation of Infarct Size. *Am Heart J* 1989;117:809-18.
2. Radford MJ, Johnson RA, Daggett WM Jr, et al. Ventricular septal rupture: a review of clinical and physiologic features and an analysis of survival. *Circulation* 1981;64:545-53.
3. Topaz O, Taylor AL. Interventricular septal rupture complicating acute myocardial infarction: from pathophysiologic features to the role of invasive and noninvasive diagnostic modalities in current management. *Am J Med* 1992;93:683-8.
4. Hutchins GM. Rupture of the interventricular septum complicating myocardial infarction: pathological analysis of 10 patients with clinically diagnosed perforations. *Am Heart J* 1979;97:165-73.
5. Moore CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS. Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. *Circulation* 1986;74:45-55.
6. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. *Circulation* 2000;101:27-32.
7. Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. *Am J Cardiol* 1984;54:1201-5.
8. Skehan JD, Carey C, Norrell MS, de Belder M, Balcon R, Mills PG. Patterns of coronary artery disease in post-infarction ventricular septal rupture. *Br Heart J* 1989;62:268-72.
9. Birnbaum Y, Wagner GS, Gates KB, et al. Clinical and electrocardiographic variables associated with increased risk of ventricular septal defect in acute anterior myocardial infarction. *Am J Cardiol* 2000;86:830-4.
10. Shapira I, Isakov A, Burke M, Almog CH. Cardiac rupture in patients with acute myocardial infarction. *Chest* 1987;92:219-23.

11. Oskoui R, Van Voorhees LB, DiBianco R, Kiernan JM, Lee F, Lindsay J Jr. Timing of ventricular septal rupture after acute myocardial infarction and its relation to thrombolytic therapy. *Am J Cardiol* 1996;78:953-5.
12. Cummings RG, Reimer KA, Califf R, Hackel D, Boswick J, Lowe JE. Quantitative analysis of right and left ventricular infarction in the presence of postinfarction ventricular septal defect. *Circulation* 1988;77:33-42.
13. Mann JM, Roberts WC. Acquired ventricular septal defect during acute myocardial infarction: analysis of 38 unoperated necropsy patients and comparison with 50 unoperated necropsy patients without rupture. *Am J Cardiol* 1988;62:8-19.
14. Figueras J, Cortadellas J, Soler-Soler J. Comparison of ventricular septal and left ventricular free wall rupture in acute myocardial infarction. *Am J Cardiol* 1998;81:495-7.
15. Prêtre R, Rickli H, Ye Q, Benedikt P, Turina MI. Frequency of collateral blood flow in the infarct-related coronary artery in rupture of the ventricular septum after acute myocardial infarction. *Am J Cardiol* 2000;85:497-9.
16. Becker AE, van Mantgem JP. Cardiac tamponade: a study of 50 hearts. *Eur J Cardiol* 1975;3:349-58.
17. Mann JM, Roberts WC. Fatal rupture of both left ventricular free wall and ventricular septum (double rupture) during acute myocardial infarction: analysis of seven patients studied at necropsy. *Am J Cardiol* 1987;60:722-4.
18. Aravot DJ, Dhalla N, Banner NR, Mitchell A, Rees A. Combined septal perforation and cardiac rupture after myocardial infarction: clinical features and surgical considerations of a correctable condition. *J Thorac Cardiovasc Surg* 1989;97:815-20.
19. Lemery R, Smith HC, Giuliani ER, Gersh BJ. Prognosis in rupture of the ventricular septum after acute myocardial infarction and role of early surgical intervention. *Am J Cardiol* 1992;70:147-51.
20. Loisanse DY, Lordez JM, Deleuze PH, Dubois-Rande JL, Lellouche D, Cachera JP. Acute postinfarction septal rupture: long-term results. *Ann Thorac Surg* 1991;52:474-8.
21. Westaby S, Parry A, Ormerod O, Gooneratne P, Pillai R. Thrombolysis and postinfarction ventricular septal rupture. *J Thorac Cardiovasc Surg* 1992;104:1506-9.
22. Menon V, Webb JG, Hillis LD, et al. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. *J Am Coll Cardiol* 2000;36:Suppl A:1110-6.
23. Perloff JK, Talano JV, Ronan JA Jr. Noninvasive techniques in acute myocardial infarction. *Prog Cardiovasc Dis* 1971;13:437-64.
24. Gowda KS, Loh CW, Roberts R. The simultaneous occurrence of a ventricular septal defect and mitral insufficiency after myocardial infarction. *Am Heart J* 1976;92:234-6.
25. Amico A, Iliceto S, Rizzo A, Cascella V, Rizzon P. Color Doppler findings in ventricular septal dissection following myocardial infarction. *Am Heart J* 1989;117:195-8.
26. Smyllie JH, Sutherland GR, Geuskens R, Dawkins K, Conway N, Roelandt JR. Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. *J Am Coll Cardiol* 1990;15:1449-55.
27. Fortin DF, Sheikh KH, Kisslo J. The utility of echocardiography in the diagnostic strategy of postinfarction ventricular septal rupture: a comparison of two-dimensional echocardiography versus Doppler color flow imaging. *Am Heart J* 1991;121:25-32.
28. Vargas Barron J, Sahn DJ, Valdes-Cruz LM, et al. Clinical utility of two-dimensional Doppler echocardiographic techniques for estimating pulmonary to systemic blood flow ratios in children with left to right shunting atrial septal defect, ventricular septal defect or patent ductus arteriosus. *J Am Coll Cardiol* 1984;3:169-78.
29. Obariski TP, Rogers PJ, Debaets DL, Murcko LG, Jennings MR. Assessment of postinfarction ventricular septal ruptures by transesophageal Doppler echocardiography. *J Am Soc Echocardiogr* 1995;8:728-34.
30. Ballal RS, Sanyal RS, Nanda NC, Mahan EF III. Usefulness of transesophageal echocardiography in the diagnosis of ventricular septal rupture secondary to acute myocardial infarction. *Am J Cardiol* 1993;71:367-70.
31. Harpaz D, Shah P, Bezante GP, Meltzer RS. Ventricular septal rupture after myocardial infarction: detection by transesophageal echocardiography. *Chest* 1993;103:1884-5.
32. Tatoes CJ, Gault JH, Mason DT, Ross J Jr. Reflux of oxygenated blood into the pulmonary artery in severe mitral regurgitation. *Am Heart J* 1968;75:102-6.
33. Fuchs RM, Heuser RR, Yin FC, Brinker JA. Limitations of pulmonary wedge V waves in diagnosing mitral regurgitation. *Am J Cardiol* 1982;49:849-54.
34. Wynne J, Fishbein MC, Holman BL, Alpert JS. Radionuclide scintigraphy in the evaluation of ventricular septal defect complicating acute myocardial infarction. *Cathet Cardiovasc Diagn* 1978;4:189-97.
35. Blanche C, Khan SS, Matloff JM, et al. Results of early repair of ventricular septal defect after an acute myocardial infarction. *J Thorac Cardiovasc Surg* 1992;104:961-5.
36. Ryan TJ, Antman EM, Brooks NH, et al. 1999 Update: ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol* 1999;34:890-911.
37. Daggett WM, Guyton RA, Mundth ED, et al. Surgery for post-myocardial infarction ventricular septal defect. *Ann Surg* 1977;186:260-71.
38. Gray RJ, Sethna D, Matloff JM. The role of cardiac surgery in acute myocardial infarction. I. With mechanical complications. *Am Heart J* 1983;106:723-8.
39. Fox AC, Glassman E, Isom OW. Surgically remediable complications of myocardial infarction. *Prog Cardiovasc Dis* 1979;21:461-84.
40. Giuliani ER, Danielson GK, Pluth JR, Odyniec NA, Wallace RB. Postinfarction ventricular septal rupture: surgical considerations and results. *Circulation* 1974;49:455-9.
41. Scanlon PJ, Montoya A, Johnson SA, et al. Urgent surgery for ventricular septal rupture complicating acute myocardial infarction. *Circulation* 1985;72:Suppl II:II-185-II-190.
42. Massetti M, Babatasi G, Le Page O, Bhoyroo S, Saloux E, Khayat A. Postinfarction ventricular septal rupture: early repair through the right atrial approach. *J Thorac Cardiovasc Surg* 2000;119:784-9.
43. Lee EM, Roberts DH, Walsh KP. Transcatheter closure of a residual postmyocardial infarction ventricular septal defect with the Amplatzer septal occluder. *Heart* 1998;80:522-4.
44. Landzberg MJ, Lock JE. Transcatheter management of ventricular septal rupture after myocardial infarction. *Semin Thorac Cardiovasc Surg* 1998;10:128-32.
45. Pesonen E, Thilen U, Sandstrom S, et al. Transcatheter closure of post-infarction ventricular septal defect with the Amplatzer Septal Occluder device. *Scand Cardiovasc J* 2000;34:446-8.
46. Blanche C. Surgery for mechanical complications of myocardial infarction. In: Crawford MH, DiMarco JP, eds. *Cardiology*. St. Louis: Mosby, 2001:171-178.
47. Blanche C, Khan SS, Chau A, Matloff JM. Postinfarction ventricular septal defect in the elderly: analysis and results. *Ann Thorac Surg* 1994;57:1244-7.
48. Heitmiller R, Jacobs ML, Daggett WM. Surgical management of post-infarction ventricular septal rupture. *Ann Thorac Surg* 1986;41:683-91.
49. Davies RH, Dawkins KD, Skillington PD, et al. Late functional results after surgical closure of acquired ventricular septal defect. *J Thorac Cardiovasc Surg* 1993;106:592-8.
50. Hochman JS, Buller CE, Sleeper LA, et al. Cardiogenic shock complicating acute myocardial infarction — etiologies, management and outcome: a report from the SHOCK Trial Registry. *J Am Coll Cardiol* 2000;36:Suppl A:1063-70.
51. Prêtre R, Ye Q, Grunenfelder J, Lachat M, Vogt PR, Turina MI. Operative results of "repair" of ventricular septal rupture after acute myocardial infarction. *Am J Cardiol* 1999;84:785-8.
52. Held AC, Cole PL, Lipton B, et al. Rupture of the interventricular septum complicating acute myocardial infarction: a multicenter analysis of clinical findings and outcome. *Am Heart J* 1988;116:1330-6.

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