Ventricular Septal Rupture: A Review of Clinical and Physiologic Features and an Analysis of Survival

Martha J. Radford, M.D., Robert Arnold Johnson, M.D., William M. Daggett, Jr., M.D., John T. Fallon, M.D., Ph.D., Mortimer J. Buckley, M.D., Herman K. Gold, M.D., and Robert C. Leinbach, M.D.

SUMMARY Forty-one patients with postinfarction ventricular septal rupture were cared for in our hospital during 1971–1975. Cardiogenic shock developed after septal rupture in 55% of these patients. Shock was unrelated to site of infarction, extent of coronary artery disease, left ventricular ejection fraction, or pulmonary-to-systemic flow ratio, but mean pulmonary artery pressure was lower in shock than in nonshock patients. These observations suggest that shock was produced mainly by right ventricular impairment. Perioperative survival was much higher in patients who did not have shock preoperatively (14 of 17 [82%]) than in those who did (three of 11 [27%]). Magnitude of shunt, left ventricular ejection fraction, extent of coronary artery disease, and performance of aortocoronary bypass grafting were not distinctly correlated with perioperative survival. After a minimum 4-year follow-up, 76% of the perioperative survivors are alive, and none suffer more than New York Heart Association functional class II disability. All 13 unoperated patients (11 in shock) died within 3 months.

AN OPERATION for repairing infarction-induced ventricular septal rupture has been available for almost 25 years1 and has been improved in the past decade.2* When in the patient's course is the best time to perform the operation is unestablished, however. Performing the operation within the first few weeks of myocardial infarction results in a high mortality rate,3,4 but whether the early operation itself causes this high mortality has not been studied. In this report we review the clinical and physiologic features of all patients in our hospital with ventricular septal rupture who were identified during the early 1970s, analyze the features most related to their course, and, based on these observations, suggest guidelines for treating patients with ventricular septal rupture.

Methods

During 1971–1975, ventricular septal rupture developed after myocardial infarction in 41 patients in our hospital. These patients were identified using three sources: a computer search of hospital records for patients with concurrent diagnoses of coronary artery disease and ventricular septal defect or rupture; the surgical log, which identified patients in whom surgical repair of the ventricular septal defect was undertaken; and pathology department records, which identified patients whose ventricular septal defect was diagnosed at necropsy. Twenty-six of the 41 patients had undergone both coronary angiography and left ventriculography, three had undergone coronary angiography alone, and two had undergone left ventriculography alone.

The diagnosis of ventricular septal rupture was established by a right atrial-to-pulmonary artery step-up in oxygen content in 38 patients. The pulmonary-to-systemic flow ratio was calculated using a standard formula.5

We define cardiogenic shock as systemic hypotension (mean arterial blood pressure ≤ 70 mm Hg) accompanied by either oliguria (urine output < 20 ml/hr for 4 hours or more) or a rising blood urea nitrogen (to > 50 mg/dl).

The left ventricular ejection fraction (left ventricular stroke volume/left ventricular end-diastolic volume) was calculated by the modified area-length method as applied to single-plane, right anterior oblique cineventriculograms.6 Whenever possible, the borders of the left ventricle were identified during the first cardiac cycle after injection of contrast medium, before the right ventricle became opacified. In some cases, the outline of the left ventricular inferior border was estimated because it was partly obscured by the opacified right ventricle. The angiographic extent of coronary artery disease was measured by the angiographic jeopardy score.7

Patients, their families, or their physicians were contacted by telephone. No patient was lost to follow-up. Survivors were questioned about activity level, cardiac medications, and intervening medical events. Follow-up was 48–91 months.

In patients in whom necropsy was performed, the proportion of the left and the right ventricular free wall and the interventricular septum that was infarcted was estimated by measuring the percentage of infarcted tissue on photographs of five cross sections of the whole heart (nine cases), or was estimated from the gross description of the heart (seven cases). Eight of the patients had undergone partial infarctectomy; our estimate of the proportion of tissue infarcted takes

From the Cardiac Units of the Departments of Medicine, Surgery, and Pathology Massachusetts General Hospital, and Harvard Medical School, Boston, Massachusetts.

Supported in part by USPHS Ischemia SCOR grant HL-17665, NIH.

Address for correspondence: Robert Arnold Johnson, M.D., Cardiac Unit, Massachusetts General Hospital, Fruit Street, Boston, Massachusetts 02114.

Received August 1, 1980; revision accepted December 19, 1980. Circulation 64, No. 3, 1981.
into consideration the approximate amount of ventricle removed during operation.

Statistical Methods

The probabilities that patients with and without cardiogenic shock were drawn from the same population with respect to each of the physiologic variables listed in table 2 were tested by the t test. The probabilities that patients who did or did not survive operation were drawn from the same population with respect to each of the physiologic variables listed in table 2 were tested by the t test. The probability that patients with or without cardiogenic shock were drawn from the same population with respect to the prevalence of anterior vs inferior infarction was tested by Fisher's exact test. The probability that patients who did or did not survive operation were drawn from the same population with respect to the prevalence of preoperative cardiogenic shock was tested by Fisher's exact test. The probability that patients who did or did not survive operation performed within 21 days of infarction were drawn from the same population with regard to the prevalence of preoperative cardiogenic shock was tested by Fisher's exact test.

Results

Clinical Characteristics

The patients were 48–74 years of age (mean 63 years). Twenty-five patients were men and 16 were women. Nine patients had a history of hypertension before myocardial infarction. In 40 of 41 patients, ventricular septal rupture was diagnosed before the patient's death.

Six patients (15%) had electrocardiographic or historical evidence for previous remote myocardial infarction. Thirteen patients (32%) had angina pectoris before the myocardial infarction causing septal rupture; in three of these, angina began less than 1 month before myocardial infarction.

The acute myocardial infarction responsible for ventricular septal rupture produced electrocardiographic Q-waves in 39 patients (transmural free wall infarction was found at necropsy in the other two patients) and a conduction defect (left bundle branch block) in one. Myocardial infarction was anterior in 22 patients and inferior in 19. In 30 patients (77%), the septal rupture occurred during the first week after acute myocardial infarction (fig. 1).

Physical examination and radiologic findings are tabulated in table 1. A new systolic murmur heralded the presence of ventricular septal rupture in all but one patient. A thrill accompanied the murmur in half of the patients. In nine patients, postinfarction chest pain immediately preceded the onset of the murmur. Three

Figure 1. Time between myocardial infarction and the appearance of the murmur of ventricular septal rupture (could be assessed in 39 of 41 patients).

patients had undergone cardiopulmonary resuscitation just before appearance of the murmur, and a temporary pacemaker had been removed from one patient the day before the murmur was heard. Systemic hypertension was recorded in four patients (mean systemic arterial blood pressure ≥ 120 mm Hg) after the onset of acute myocardial infarction but before ventricular septal rupture.

Clinical Course: Hemodynamic and Angiographic Correlations

Two patients had arterial hypotension before ventricular septal rupture. Cardiogenic shock developed after septal rupture in 22 of the 41 patients, 18 within the first week. Thirteen of these 22 had anterior myocardial infarction; nine had inferior myocardial infarction (p > 0.05). Cardiac index was measured (Fick method)† in nine patients (one in cardiogenic shock); it ranged from 2.3–4.9 l/min/m². Neither right atrial pressure, pulmonary capillary wedge pressure, pulmonary-to-systemic flow ratio, left ventricular ejection fraction, nor jeopardy score distinguished patients with from those without cardiogenic shock (table 2, figs. 2 and 3); nor did the combination of pulmonary-to-systemic flow ratio and left ventricular ejection fraction distinguish patients with from those without cardiogenic shock (fig. 4). Mean pulmonary artery pressure was lower in patients with shock than in those without shock (table 2).

Twenty-seven patients received intraaortic balloon counterpulsation, 18 of whom had cardiogenic shock, and nine of whom did not. The pulmonary-to-systemic flow ratio was reduced an average of 23% by balloon assist. In three patients, two of whom were in shock, the pulmonary-to-systemic flow ratio increased. Three serious complications resulted from intraaortic balloon placement. In one patient, it led to leg ischemia. Auxiliary-femoral bypass grafting was attempted, but the patient died during that operation.}

Another patient died of a bowel infarction; dissection

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*The t test would be expected to exaggerate type I statistical errors (finding a falsely low likelihood that the data can be explained by the null hypothesis), and our argument is based on finding a reasonably high likelihood that the data are explainable by the null hypothesis.

†Cardiac output cannot be measured by an indicator-dilution technique in the presence of a shunt.
of the aorta was found at laparotomy. In a third patient, iliac and aortic dissection were discovered at necropsy. This patient had been in cardiogenic shock and did not improve after balloon placement. Placement of an intraaortic balloon was attempted unsuccessfully in two patients.

Left ventriculography was performed in 28 patients, 15 of whom were receiving intraaortic balloon counterpulsation. Ventricular arrhythmias were frequently noted during catheterization of the left ventricle, but serious complications of angiography (death, myocardial infarction and stroke) were not encountered.

The location of ventricular septal rupture, identified by ventriculography, corresponded to the electrocardiographic location of infarction in all cases; rupture occurred in the anterior or apical septum in those patients with anterior myocardial infarction, and in the posterior septum in those patients with inferior myocardial infarction. One patient whose ECG showed inferoapical infarction demonstrated apical septal rupture.

A right ventricular aneurysm was present in seven patients, all of whom had coexisting inferior left ventricular infarction. Three of these patients had cardiogenic shock. The aneurysm occurred along the inferior right ventricular border, and was usually visible during the second cardiac cycle after the contrast agent had passed from the left ventricle into the right ventricle.

Only six patients demonstrated collateral flow to septal arteries.

Survival

In 13 patients, operative repair of the ventricular septal rupture was not attempted, and all of these patients died within 3 months. Eleven of the unoperated patients were in cardiogenic shock at the time of death; one died 1 day after intraaortic balloon pump insertion from aortic dissection and bowel infarction;
one patient died from intractable ventricular arrhythmias while awaiting operation.

**Postoperative Survival**

Operative repair of the ventricular septal defect was performed in 28 patients by methods described elsewhere.6, 7, 9 Eight of the 11 patients who had preoperative cardiogenic shock died in the perioperative period (six from heart failure); 10 of these 11 patients were operated on within 21 days of myocardial infarction (early operation).

Cardiogenic shock was thought to be impending (falling blood pressure and urine output) in six other patients who did not meet our criteria for cardiogenic shock but who were considered ill enough that repair was warranted during the first 21 days after acute myocardial infarction; one of these six died in the perioperative period ($p < 0.05$, perioperative mortality in patients with shock and early operation vs that in patients not in shock but who had early operation). The remaining 11 nonshock patients were operated on 3 weeks or more after acute myocardial infarction; two died.

Overall perioperative survival was 61%. In short, perioperative survival was much better for nonshock patients (14 of 17, 82%) than for shock patients (three of 11, 27%) ($p < 0.05$). The presence or absence of preoperative shock was the only predictor of subsequent course; right atrial pressure, pulmonary capillary wedge pressure, pulmonary-to-systemic flow ratio, left ventricular ejection fraction, extent of coronary artery disease, and the performance of aorto-coronary bypass grafting were not correlated with perioperative survival (tables 2 and 3). Two of three patients survived who underwent concurrent mitral valve replacement.

Of the 17 perioperative survivors, 13 patients (76%) were alive after a minimum 4-year follow-up, and only one of the four late deaths was directly attributable to a cardiac cause (a sudden, unexpected death 6 years after operation). Before death, the cardiac status was stable for each of the four patients who died, although one had suffered a myocardial infarction 36 months after septal repair. Of the 13 patients who are alive at the end of the follow-up period, none suffer disabling symptoms exceeding a New York Heart Association class II level, and three take no medications. Three patients work at full-time jobs. Chronic antiarrhythmic therapy is required in three patients.

All but three patients were placed on warfarin after operation. Anticoagulants were later discontinued in three patients. Five episodes of hemorrhage occurred, two of which were the cause of death.

**Necropsy Results**

The pathologic location of infarction corresponded to the electrocardiographic location in all 16 necrop-
Table 3. Number of Diseased Vessels and Number of Aortocoronary Bypass Grafts

<table>
<thead>
<tr>
<th>Vessels diseased</th>
<th>Nonsurvivors*</th>
<th>Survivors*</th>
</tr>
</thead>
<tbody>
<tr>
<td>One</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Two</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Three</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Aortocoronary bypass grafts</th>
<th>Nonsurvivors*</th>
<th>Survivors*</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>One</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

*Perioperative period.

Table 4. Infarcted Myocardium Found at Necropsy

<table>
<thead>
<tr>
<th></th>
<th>Infarcted &gt;1/3 RVFW</th>
<th>Infarcted &gt;½ septum</th>
<th>Infarcted &gt;1/3 LVFW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock</td>
<td></td>
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<tr>
<td>IMI</td>
<td>3/3</td>
<td>1/4</td>
<td>0/4</td>
</tr>
<tr>
<td>AMI</td>
<td>1/3</td>
<td>5/6</td>
<td>2/6</td>
</tr>
<tr>
<td>No shock</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IMI</td>
<td>1/2</td>
<td>0/2</td>
<td>0/3</td>
</tr>
<tr>
<td>AMI</td>
<td>ND</td>
<td>0/1</td>
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*Second number refers to total number of cases in which the size of the infarct could be evaluated.

Discussion

Incidence, Pathogenesis, and Diagnosis

Rupture of the interventricular septum is estimated to complicate 1–3% of acute myocardial infarctions, and it produces approximately 5% of perinfarction deaths.14, 16 It occurs most frequently during the first week after myocardial infarction (fig. 1),17 the infarction often having been the patient's first (85% of the patients in this series).18-20 Before operative repair was introduced by Cooley in 1957,1 septal rupture was almost always fatal: 80% of patients died within 2 months and 93% within 1 year.16, 17, 21 Only a few unoperated patients have lived for many years.22, 23

Ventricular septal rupture may occur in patients whose coronary artery disease is limited in extent.24 For example, nine of 19 patients in one series had one-vessel disease,26 and one-vessel disease was present in seven of the 30 patients in our study in whom the extent of coronary artery disease was evaluated by angiography or at necropsy. The mean jeopardy score for our septal rupture patients was 6.8, whereas the mean jeopardy score for patients with heart failure resulting from cardiomyopathic syndrome owing to coronary artery disease (multiple infarctions, either acute or remote) is 10.7.18 We found, as did Miller et al.26 that the angiographic studies of most patients with ventricular septal rupture do not show septic collateral flow. The high prevalence of first infarctions, the limited extent of coronary artery disease in some patients, and the paucity of septic collateral vessels are circumstantial evidence that an abrupt, relatively large reduction in flow, such as that produced by thrombosis or spasm, causes the infarction that is later responsible for rupture.

Hypertension before or after myocardial infarction has been postulated to be important in the pathogenesis of ventricular septal rupture.27, 28 Roberts et al. found that all of their 41 patients who died of postinfarction septal or free wall rupture had left ventricular thickening consistent with a history of hypertension.19 But our findings and those of others29, 30 suggest that the relationship between cardiac rupture and hypertension, if it exists, is not a close one. Only 25% of our patients related a history of hypertension, and of those who underwent necropsy, fewer than one-half had increased left ventricular thickness. Moreover, only four of our patients were known to be hypertensive in the immediate postinfarction period.

The onset of a new systolic murmur after myocardial infarction is the clinical sign that caused clinicians to suspect ventricular septal rupture in our patients. In rare instances, a systolic murmur is absent or overlooked,17, 31 as it was in the only one of our patients who was first diagnosed at necropsy. A thrill accompanies the murmur in approximately one-half the patients (table 1).17, 31 Neither the location of the murmur nor the existence of the thrill helps distinguish
patients with ventricular septal rupture from patients with acute mitral regurgitation owing to papillary muscle infarction or rupture.23

Right-sided heart failure overshadows left-sided heart failure in many patients with ventricular septal rupture, who almost always exhibit jugular venous and right atrial pressure elevation despite having only modest left atrial pressure elevation. For this reason, the symptoms, signs, and radiologic findings of pulmonary venous hypertension usually are not striking (tables 1 and 2).16, 19, 23, 28, 31 How sensitive or specific these observations might be for distinguishing patients with ventricular septal rupture from patients with severe mitral regurgitation owing to papillary muscle rupture or infarction has not been studied. Pulmonary edema may occur more often in the latter.17

The diagnosis of ventricular septal rupture by means of bedside right-heart catheterization and sequential oximetry was described in 1972.32 This technique has made the differentiation of septal rupture from infarction-induced acute mitral regurgitation relatively straightforward, because septal rupture usually produces a pulmonary-to-systemic flow ratio of more than 2:1 (fig. 4),19, 21 and hence it is readily detected by sequential oximetry in most instances. If right-heart catheterization cannot be performed easily, the defect sometimes can be visualized (an echo-free zone) by means of one-dimensional echocardiography33 or detected by a Doppler echocardiographic technique.34 In addition to the echo-free zone, one-dimensional echocardiography may show less specific findings suggesting ventricular septal rupture, such as right ventricular dilation, unusual mitral valve motion, diminished or paradoxical septal motion, and a diminished E-to-F slope of the tricuspid valve.35, 36-37 Two-dimensional echocardiography is likely to prove more reliable than one-dimensional echocardiography for demonstrating the site of septal rupture.38 Left-to-right shunting also may be detected by radionuclide techniques.39-41

Clinical Course and Determinants of Survival

The course after ventricular septal rupture varies.10, 17, 23, 28, 29, 42, 43 Rapid hemodynamic deterioration developed in 60% of our patients, leading to either early operative repair or death from cardiogenic shock within 1 week. Twenty percent deteriorated less rapidly, but relentlessly, until operation was performed or death ensued from cardiogenic shock. Most of the remainder stabilized hemodynamically, but were severely symptomatic from heart failure and required operation within weeks. Approximately 5% of our patients had less severe heart failure, and operative repair was carried out 2-9 months after septal rupture. One patient has been reported who survived, without operation, for 13 years after septal rupture,22 but few patients have heart failure that is mild enough to allow medical treatment as a substitute for operative repair.

Our study has two principal findings: Perioperative survival is mainly determined by whether cardiogenic shock has developed preoperatively, and if it has, it is much reduced; and the development of cardiogenic shock is not explained by the state of left ventricular performance. Some patients with relatively good left ventricular function develop cardiogenic shock and some patients with relatively poor left ventricular function do not (fig. 2). Unlike most complications of myocardial infarction, clinical outcome does not appear to depend on left ventricular function.44, 45 Nor does the volume of blood flow left-to-right through the defect, either by itself46 or considered in combination with the left ventricular ejection fraction, explain clinical outcome (fig. 3). Why, then, does cardiogenic shock develop in some patients with ventricular septal rupture and not in others? We propose that right ventricular function may be the major determinant. The lower mean pulmonary artery pressure in our patients with shock compared with those without shock is consistent with this hypothesis.

Right ventricular infarction causes cardiogenic shock and death in a small fraction of patients with inferior left ventricular myocardial infarction but without septal rupture.47 In patients with septal rupture and inferior left ventricular infarction, our necropsy findings (table 4) suggest that more right ventricular free wall was infarcted in those in whom shock had developed than in those in whom it had not. Right ventricular aneurysm formation may contribute to the inability of the infarcted right ventricle to sustain the level of pulmonary blood flow necessary to maintain an adequate systemic cardiac output when ventricular septal rupture exists. Why anterior left ventricular infarction and septal rupture would coexist with right ventricular impairment in some patients is less easily explained, because right ventricular free wall infarction is uncommon in such cases. Right ventricular free wall infarction coexisted with anterior left ventricular infarction in only one of our necropsied patients and has been noted infrequently by others.31 Massive septal infarction was common in our necropsied patients in shock after anterosetal myocardial infarction and septal rupture; this massive septal rupture may impair right ventricular function. Right ventricular function is depressed after anterosetal myocardial infarction in the porcine heart, and this depression is even more marked under conditions of volume loading.48

If right ventricular function is an important determinant of the clinical course of patients with ventricular septal rupture, intraaortic balloon counterpulsation, which directly affects only left ventricular afterload, may be expected to offer transient stabilization, at best, even though the pulmonary-to-systemic flow ratio is reduced. This response has, in fact, been observed.49, 50 Afterload reduction with vasodilator drugs has the theoretical advantage of a possible decrease in pulmonary vascular resistance, thereby improving right ventricular ejection, in addition to lowering systemic vascular resistance; the effect on the amount of shunted blood depends on the relative change in the two resistances. In two reported patients
whose septal rupture was treated with intravenous nitropresside, the systemic cardiac output improved in both, whereas the amount of shunted blood decreased in one and increased in the other.

Treatment

The best timing for operative correction of ventricular septal rupture has been a subject of debate. Because of high mortality in patients undergoing surgery soon after myocardial infarction (within 3 weeks), Giuliani et al. recommended that surgery be delayed as long as possible, ideally 3–6 weeks. This practice, of course, selects out for operation those patients with the least severe heart failure and most likely to survive without operation. Our study shows that perioperative survival is more affected by the existence of preoperative cardiogenic shock than by early timing of the operation. Early operations in patients with cardiogenic shock were associated with a high mortality, whereas early operations in patients with severe heart failure but without cardiogenic shock were associated with a much lower mortality. Several other centers are finding also that ventricular septal rupture can be repaired successfully in the early postinfarction period.

Our treatment of patients with ventricular septal rupture now is generally founded on these observations. If cardiogenic shock is present, intraaortic balloon pumping is instituted promptly; cardiac catheterization, left ventriculography, and coronary angiography are performed within 6 hours thereafter. Ventricular septal repair is carried out within the next 12 hours, even if intraaortic balloon pumping had produced apparent hemodynamic stability, because this stability is usually temporary. If the operation is delayed, the progressive azotemia, the persistent catabolic state, and the substantial risk of infection almost always present cause a further increase in the operation's risk. If intraaortic balloon pumping is unavailable, a vasodilator drug, such as nitropresside, may be instituted in some cases, but profound systemic hypotension often prevents or complicates this possibility. A vasoconstrictor drug, such as norepinephrine, may be necessary to sustain the systemic blood pressure until intraaortic balloon pumping can be instituted or ventricular septal rupture repair can be performed, even though these drugs may produce a reduction in systemic cardiac output and an increase in the left-to-right shunt. One of the advantages of intraaortic balloon pumping, even though it sometimes causes serious complications, is that it allows coronary angiography and left ventriculography to be performed in patients who otherwise might not tolerate these procedures. The risk of perioperative death is high if ventricular septal rupture has produced shock; even so, we believe that patients with shock should undergo operation (unless extreme old age or a far advanced coexisting medical condition make it ill advised), because we have not encountered any such patients who have survived without operation.

We believe that patients with progressively increasing or severe heart failure but without shock should be treated in the same way as patients with shock. Clinical and hemodynamic improvement resulting from intraaortic balloon pumping is not a reason for deferring operation. The risk of perioperative death is much lower in these patients (17% in our series) than in those with shock. In patients whose heart failure is not rapidly advancing, vasodilator drugs may produce stabilization and enough improvement that intraaortic balloon pumping is unnecessary, and angiographic studies and operative repair may be deferred for several weeks. In a few patients, perhaps 5% or less, heart failure is mild enough that operation may not be necessary. Even in these cases, however, the clinician must consider whether pulmonary arteriolar disease and, eventually, right-to-left shunting might complicate an unrepaired ventricular septal rupture; this sequence has been reported in one patient.

Several technical advances in the operation for ventricular septal rupture have been made in recent years. Placing theventriculotomy in the left ventricular infarction with an infarctectomy allows better visualization of the septal defect and avoids disrupting noninfarcted myocardium. An anterior defect can usually be closed primarily with felt-buttressed sutures; amputation of the apex and infarctectomy is often used if the defect is in the apical septum. Posterior septal defects usually require prosthetic replacement of part of the posterior wall to prevent subsequent reopening of the defect or dehiscence of the ventriculotomy. Severe mitral regurgitation, owing to papillary muscle infarction, coexisted with ventricular septal rupture in approximately 10% of our operated patients, and this combination of conditions has been reported by others. Mitral valve replacement may be performed at the time of septal repair in such instances. Functional mitral regurgitation, milder in degree, occurs more often; repair of the septal rupture alone produces prompt disappearance of regurgitation in such cases. In addition, we believe that aortocoronary bypass grafts should be placed to the largest suitable vessels that contain significant proximal stenosis in patients undergoing ventricular septal repair. This is a controversial subject, however, and no studies have been designed to test critically whether concomitant aortocoronary bypass grafting is beneficial.

Acknowledgment

The authors acknowledge the help of John B. Newell, Manager of Medical Research Applications at the Harvard Office of Information Technology.

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Hemodynamic Effects of Nitroprusside in Infants with a Large Ventricular Septal Defect

ROBERT H. BEEKMAN, M.D., ALBERT P. ROCCHINI, M.D., AND AMNON ROSENTHAL, M.D.

SUMMARY To evaluate the effects of acute vasodilator therapy, nitroprusside was administered at cardiac catheterization to five infants (ages 10 days to 6 months) with isolated ventricular septal defect and congestive heart failure. Intravenous nitroprusside was begun at a dose of 0.5 μg/kg/min and was increased by increments of 0.5 μg/kg. Hemodynamic measurements were made before nitroprusside, after 5 minutes at each dose, and 10 minutes after nitroprusside was discontinued. Baseline data were obtained before nitroprusside administration and compared with data obtained at maximal nitroprusside dose. The pulmonary-to-systemic flow ratio increased from 2.2 ± 0.2 to 3.4 ± 0.2 (mean ± SEM, p < 0.05) as a consequence of a marked decrease in systemic blood flow (5.3 ± 0.7 to 3.6 ± 0.51/min/m², p < 0.05). Pulmonary flow did not change significantly. Mean pulmonary capillary wedge and right atrial pressures decreased by 53% (10.2 ± 1.4 to 4.8 ± 1.4 mm Hg [p < 0.01] and 6.0 ± 1.4 to 2.8 ± 1.1 mm Hg [p < 0.05], respectively). Decreases in mean aortic (63.6 ± 3.0 to 54.6 ± 2.1 mm Hg, p < 0.05) and mean pulmonary artery pressure (41.4 ± 6.2 to 32.0 ± 6.7 mm Hg, p < 0.05) were also observed. An apparently paradoxical increase in systemic resistance occurred (11.7 ± 1.6 to 15.4 ± 2.4 U, p < 0.05).

Our data show that nitroprusside causes a marked decrease in systemic blood flow and an increase in the pulmonary-to-systemic flow ratio in infants with a large ventricular septal defect. These findings may be related to the hemodynamic profile of these infants, in whom ventricular function, cardiac output and systemic resistance are normal.

VASODILATOR THERAPY has assumed an important role in the medical management of many cardiovascular disorders. Agents that decrease smooth muscle tone in systemic arterioles, venous capacitance vessels, or both have been shown to produce significant hemodynamic improvement in patients with ischemic heart disease, primary cardiomyopathy, and mitral or aortic valve disease. Although the majority of these studies involved only adults, vasodilators have been used successfully in children who have poor cardiac function postoperatively or due to cardiomyopathy. In addition, experimental animal work has shown that α-blocking agents, by dilating systemic resistance vessels, can diminish the magnitude of left-to-right shunting across a ventricular septal defect (VSD). The present study was designed to evaluate the hemodynamic effects of nitroprusside in infants with a large VSD.

Methods

Five infants, three females and two males, who had a large VSD and congestive heart failure, form the basis of this report. The relevant clinical data are presented in table 1. The mean age was 2.8 months (range 10 days to 6 months). One patient had Down's syndrome. All patients were in congestive heart failure.
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_Circulation_. 1981;64:545-553
doi: 10.1161/01.CIR.64.3.545
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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