Surgical Management of Postinfarction Ventricular Septal Rupture

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ABSTRACT  Recognition and treatment of patients with ventricular septal rupture following infarction have improved over the past 25 years to the extent that survival with good long-term palliation is achieved in the majority of patients treated surgically for this catastrophic complication of acute myocardial infarction. The small minority of patients who, by the process of selection, are seen for surgical correction of septal rupture several weeks after infarction routinely have repair of the septal defect with an operative risk of less than 10%. With increasingly early diagnosis of septal rupture, the majority of patients are seen for consideration of surgical repair often within hours after septal rupture. Most such patients seen early after septal rupture exhibit cardiogenic shock. Refinement of operative techniques both for suture repair of freshly infarcted myocardium and for repair of defects in different anatomical locations has markedly improved survival in these critically ill patients. Deferral of operation for the patient in cardiogenic shock after septal rupture represents a failed therapeutic strategy. Conversely, emergency operation for the patient with septal rupture and cardiogenic shock has markedly improved survival in this high-risk group. Prolonged intraaortic balloon pump support and deferred operation should be reserved for the uncommon patient who, because of delayed diagnosis or referral, is seen in an advanced stage of multisystem failure in which the risks of early operative intervention involve the function of organs other than the heart.

History
Ventricular septal rupture following infarction is an uncommon but serious complication of acute myocardial infarction (MI) and occurs less frequently than postinfarction left ventricular free wall rupture [1]. Latham [2] is credited with one of the first descriptions of this entity in his lectures on the heart published in 1845. In this description, he made no association between postinfarction ventricular septal defect (VSD) and coronary artery thrombosis. One of the first antemortem diagnoses of postinfarction VSD was made by Brunn [3] in 1923. Sager [4] in 1934 established specific clinical criteria for diagnosis and stressed the association of postinfarction VSD with coronary artery disease. In 1957, Cooley and colleagues [5] reported the successful surgical repair of postinfarction VSD in a patient nine weeks after the diagnosis of septal rupture.

The principal treatment of postinfarction VSD during the early 1960s, however, was aggressive medical treatment, even though it was well recognized that survivors were rare following nonoperative therapy alone. Surgical therapy was generally reserved for those patients who survived at least six weeks, purportedly to allow for scarring of the edges of the VSD. It was thought that a secure and long-lasting closure of the septal defect was dependent on this process [6–8]. By the late 1960s, early repair was introduced for patients whose condition was deteriorating despite medical therapy [9]. More recently, improved surgical techniques, newer prosthetic materials, enhanced myocardial protection with cold cardioplegic solutions, and improved perioperative mechanical and pharmacological support have led to more favorable results in the surgical management of patients with postinfarction VSD [10]. Attention is now focused on the proper timing of operative intervention.

Incidence and Pathogenesis
Autopsy studies reveal an 11% incidence of myocardial free wall rupture following acute MI [11]. Septal perforation is found much less frequently, with an incidence of 1 to 2% [12–15].

Septal rupture occurs through a zone of necrotic myocardial tissue, and is most likely to take place within the first 10 to 14 days when necrotic tissue is most abundant and before substantial ingrowth of blood vessels and connective tissue occurs [16]. Clinical studies have repeatedly confirmed the pathological findings of Mallory and co-workers [16]. Selzer and associates [17] in their review of 10 patients with postinfarction VSD reported an average time of 2.6 days (range, 1 to 6 days) from infarction to septal rupture. Ten years later, Hutchins [14] reported an average time from infarction to rupture of 4 days (range 0.5 to 7 days).

The age of patients sustaining a postinfarction VSD covers a wide range, with an average age of 62.5 years [17]. There is some evidence, however, that suggests the average age is increasing [10, 14, 18]. Men are affected more commonly than women, although septal rupture is more common in women than would be predicted based on the incidence of coronary artery disease in women [19]. In most instances, septal rupture occurs as a complication of an individual’s first MI [10, 14, 20]. Coronary anatomy studied at autopsy or by angiography usually demonstrates complete coronary artery occlusion with
Septal rupture is a serious complication of MI and has a mortality rate of 78% (81%) die within the first month. Only 7% survive 24 hours, and more than three-quarters (75%) of patients with postinfarction VSD die within the first week following an acute MI, is the most consistent physical finding of postinfarction VSD. Prior to the appearance of the murmur, patients may be having an uncomplicated recovery from acute MI. Coincident with the onset of the murmur, there is usually an abrupt decline in the patient’s clinical course with congestive failure and often cardiogenic shock. Fifty percent of patients have recurrent chest pain [14]. The differential diagnosis includes rupture of the interventricular septum versus mitral regurgitation secondary to papillary muscle rupture, dysfunction, or left ventricular dilatation. The characteristic harsh systolic murmur is best heard over a wide area, including the middle of the left sternal border and the apical area, depending on the location of the septal defect. Not infrequently the murmur radiates to the left axilla, thereby mimicking mitral regurgitation [17]. There is an associated thrill in 48 to 62% of patients [23, 28].

There are no electrocardiographic criteria for postinfarction VSD, although the electrocardiogram does provide useful information. Persistent ST segment elevation associated with ventricular aneurysm is frequently present. The electrocardiogram can also be used to predict the anatomical location of the VSD. In our review of 55 patients with postinfarction VSD, the location of the septal rupture corresponded to the territory of transmural infarction as determined electrocardiographically in all but 3 patients. Conduction defects secondary to direct involvement of the specialized conduction system are uncommon [14, 17].

On plain chest roentgenograms, 82% of patients with postinfarction VSD demonstrate left ventricular enlargement, 78% have alveolar pulmonary edema, and 64% show pleural effusions [21]. This constellation of findings, however, is nonspecific and does not distinguish between left ventricular pump failure (e.g., secondary to acute MI), ruptured papillary muscle, and ruptured ventricular septum.

The mainstay of differentiating septal rupture from mitral valve dysfunction is right heart catheterization using the Swan-Ganz catheter. This technique is quick, is relatively safe [29, 30], and can often be performed at the bedside. With septal rupture, there is an oxygen saturation “step-up” between the right atrium and the pulmonary artery. From the oxygen saturation samples, the pulmonary to systemic flow ratio can be calculated. Seldzer and associates [17] obtained flow ratios of 1.9 to 4.3, but there is a wide variation in reported values and ratios as high as 6.6 have been noted [13]. There is a rough linear correlation between shunt size and septal defect size [31]. Left-sided and right-sided pressure mea-
measurements assess the degree of biventricular failure and are helpful in monitoring the response to perioperative therapy. Right ventricular pressure, although elevated, is usually less than systemic pressure. Although biventricular failure is frequently present, right-sided failure is more prominent in patients with postinfarction VSD, whereas left-sided failure and pulmonary edema are more prominent in patients with ruptured papillary muscle. The morphology of the pulmonary capillary wedge pressure tracing is helpful in differentiating acute mitral regurgitation from septal rupture. Miller and co-workers [21] reported that one-third of patients with postinfarction VSD also have some mitral regurgitation secondary to left ventricular dysfunction; only rarely is septal rupture also associated with ruptured posterior papillary muscle [32]. Right heart catheterization does not provide information concerning left ventricular wall motion, coronary artery anatomy, or specifics of valvular dysfunction, which are all helpful in planning operative correction of septal rupture after infarction. For these reasons, we prefer to have a preoperative left heart catheterization with left ventriculography and coronary angiography when possible [10]. Chandraratna and associates [33] reported the use of echocardiography (M-mode) to examine 3 patients with postinfarction VSD. The only consistent finding was right ventricular dilatation. Normally directed septal motion was present in all patients. Other authors [34, 35] have obtained more specific information, including VSD location and its approximate size, using two-dimensional echocardiography. Not all septal defects can be visualized on echocardiography, however, as they may consist of multiple small defects or serpiginous tunnels through the septal wall. Although echocardiography does allow a quick, repeatable bedside evaluation, it should not supplant right heart catheterization.

Other diagnostic techniques include Doppler ultrasound measuring jugular venous blood flow velocity [36], radionuclide studies [37, 38], and detection of intracardiac shunts using a percutaneous platinum electrode [39, 40].

Preoperative Therapy

Patients suspected of having septal rupture should undergo right heart catheterization promptly to make the diagnosis and to monitor the patient's course and response to pharmacological or mechanical therapy or both. Digitalis and diuretic therapy are instituted. Pharmacological afterload reduction decreases the pulmonary to systemic blood flow ratio. However, this is often associated with a marked fall in mean arterial blood pressure and reduced coronary perfusion pressure, both poorly tolerated in these critically ill patients. In contrast, intraaortic balloon counterpulsation selectively decreases the pulmonary to systemic flow ratio without lowering mean aortic pressure while coronary perfusion pressure is increased [41]. Vasoconstrictors increase left-to-right shunting but may be necessary to maintain blood pressure pending placement of an intraaortic balloon pump (IABP) or surgical repair. Pharmacological therapy is intended primarily to support the patient in preparation for operation, and should not in any way delay urgent operation in the critically ill patient. Basically, our position is that there is no medical therapy for most patients with postinfarction VSD. We now admit patients with this diagnosis directly to the surgical intensive care unit rather than to the coronary care or medical intensive care unit.

Timing of Operation

Since the reported operative closure of a postinfarction VSD in 1957 [5], many advances in operative techniques, intraoperative myocardial protection, and perioperative management have been made. These advances have led to improvements in the surgical management of postinfarction septal rupture [10, 22, 41–43]. Attention is now focused on the optimal timing of surgical intervention. Guadini and associates [44] reviewed their experience with 43 consecutive patients with postinfarction VSD to determine what factors correlated with operative mortality. With multivariate analysis, two preoperative factors emerged as independent variables affecting operative mortality: multisystem failure and inferior infarction. The need for emergency operation very nearly achieved statistical significance as a determinant of operative mortality. Notably, the time interval between septal rupture and operation was not significantly related to operative mortality by their analysis. In addition, technical problems were not a major contributor to mortality or morbidity in either the early or late operative groups. These authors concluded that "the inverse relationship between early operation and chance of survival was primarily a measure of the severity of the illness in the patients who required early operation."

Radford and co-workers [28] reviewed the cases of 41 patients with a postinfarction VSD between 1971 and 1975, and obtained similar findings. They identified the presence or absence of cardiogenic shock as the single most important determinant of perioperative survival. They noted no correlation between left ventricular function and the clinical course in these patients, which contrasts with other complications of MI. Based on these findings, our management of patients with postinfarction VSD has increasingly involved prompt operative repair after an appropriate preoperative evaluation even if the condition of the patient has stabilized with pharmacological or IABP support or both. Hill and associates [22] reported the same operative...
mortality for patients operated on at any time during the
first month. They stressed the importance of early opera-
tion before the development of multisystem failure. Others [42, 43, 46, 47] have reported success with early
operative repair as well.

In 1982, we [10] reviewed our experience in the surgi-
cal management of patients with postinfarction VSD be-
fore (Group 1) and after (Group 2) 1975. Overall hospital
survival was 59% in Group 1 and 75% in Group 2. The
difference in hospital survival was greatest in those pa-
tients in cardiogenic shock preoperatively (27% for
Group 1 versus 67% for Group 2). Before 1975, operative
intervention was often delayed in patients in cardiogenic
shock, whereas after 1975, immediate operative repair
was employed. All patients in both of these subgroups
received IABP support.

These results suggest the following approach to the
surgical management of patients with postinfarction
VSD. Those patients in cardiogenic shock represent a
ture surgical emergency requiring immediate operative
repair. Death in these patients results from multisystem
failure secondary to end-organ hypoperfusion, with the
kidney, liver, lung, and brain most immediately af-
ected. Delay in operative repair in patients in car-
diogenic shock represents a "failed strategy." Also, im-
mediate operation results in a shorter period of required
IABP support postoperatively [10]. Those patients who
are in completely stable condition with no clinical deteri-
oration and who do not require any hemodynamic sup-
port can have operative repair performed when conve-
nient during that hospitalization. Patients who are in an
intermediate position between those in shock and those
in stable condition should be operated on promptly
(usually within 12 to 24 hours) after appropriate
preoperative evaluation. Those patients who are in sta-
ble condition represent 5% or less of the total population
of patients with postinfarction VSD. Therefore the vast
majority of these patients require prompt surgical treat-
ment. Physician education within referral areas is ex-
tremely important to ensure that prompt referral for
early operative intervention is possible.

In 1983, Baillot and associates [48] reported the results
in 3 patients with postinfarction VSD and in cardiogenic
shock who were treated with the IABP for approxi-
ately three weeks (range, 19 to 27 days) before surgical
repair. There were no operative deaths. The authors
concluded that prolonged IABP support is safe, allows
stabilization, and leads to more favorable operative con-
ditions. Their study must be viewed cautiously. We
think it unjustified to conclude that operative delay,
through use of pressor agents or IABP support or both,
represents an option to early surgical repair for most
patients with postinfarction VSD. These 3 particular pa-
tients are unusual in two respects. First, they were self-
selected as better-risk patients by surviving at least one
week after septal rupture without IABP support (which
was instituted between 10 and 16 days after the VSD).
Second, cardiogenic shock developed, which was pro-
longed secondary to delayed surgical repair (by referral
delay), and resulted in multisystem end-organ failure.
The chances of recovery following emergency operative
intervention in these neglected patients would indeed
be slight.

Thus, an occasional patient may be seen for surgical
therapy who, because of an imprudent delay in diag-
nosis or therapy, is already in a state of multisystem
failure (including disseminated intravascular coagule-
athy, sepsis, and hepatic, renal, and respiratory failure)
resulting from prolonged shock. Such a patient is un-
likely to survive an emergency operation and may ben-
efit from an attempt at prolonged IABP support be-
fore an attempt at operative repair. Only 1 of 55 patients
with postinfarction VSD whom we have treated was in
this category.

Operative Technique
The first operations for repair of postinfarction VSD em-
ployed an approach through the right ventricle with in-
cision of the right ventricular outflow tract, as is used in
approaching some congenital VSDs [5]. This approach
proved inadequate because of limited exposure (particu-
larly when the defect is located toward the apex of the
heart), injury to normal right ventricular muscle, inter-
ruption of coronary collaterals, and failure to eliminate
the paradoxically pulsating segment of infarcted left
ventricular wall. Subsequently, a left-sided approach
(left ventriculotomy) was described, which led to suc-
cessful results [49–51].

Techniques for closure of postinfarction VSD have
evolved based on the following eight principles: (1) ex-
pedient establishment of hypothermic total cardiopul-
monary bypass and meticulous attention to myocardial
protection; (2) transinfarction approach to the VSD with
the site of left ventriculotomy determined by the location
of transmural infarction; (3) thorough trimming of the
left ventricular margins of the infarct back to viable
muscle to prevent delayed rupture of the closure; (4) conser-
ervative trimming of right ventricular muscle as required
for complete visualization of the margins of the defect;
(5) inspection of the papillary muscles and concomitant
replacement of the mitral valve only if there is frank
papillary muscle rupture; (6) closure of the VSD without
tension, which in most instances will require the use of
prosthetic material; (7) closure of the infarctectomy out-
tension with generous use of prosthetic material as
necessary and epicardial placement of the patch to the
free wall to avoid strain on the friable endocardial tissue;
and (8) buttressing of suture lines with pledgets or strips
of Teflon felt or similar material to prevent sutures from
cutting through friable muscle.

Adherence to these principles results in individual-
ized approaches to anterior, apical, and inferoposterior
septal defects. Small defects beneath anterior infarcts
may sometimes be closed by approximation of the septal
margin of the defect to the right ventricular free wall (Fig
1). Teflon felt–pledgeted horizontal mattress sutures are
used, and all sutures are placed before any are tied
down. Larger anterior defects require patch closure to
Fig 1. Closure of small anterior septal defect. (A) The septum is approximated to the right ventricular free wall. Interrupted mattress sutures of 1-0 Tevdek are buttressed with strips of Teflon felt. (B) The ventriculotomy is closed in a similar fashion over strips of Teflon felt. (LV = left ventricle; RV = right ventricle.) (From Guyton SW, Daggett WM: Surgical repair of post-infarction ventricular septal rupture. In Cohn LH (ed): Modern Techniques in Surgery: Cardiac/Thoracic Surgery. Mt. Kisco, NY, Futura, 1983, installment 9, pp 61-1-61-15.)

Fig 2. Patch closure of larger anterior septal defect. (A) All sutures are placed through the edge of the defect and the patch before any are tied. (B) Each suture is buttressed with a pledget of Teflon felt. (C) Cross section of the completed repair shows patch on left ventricular side of the septum. (LV = left ventricle; RV = right ventricle.) (From Guyton SW, Daggett WM: Surgical repair of post-infarction ventricular septal rupture. In Cohn LH (ed): Modern Techniques in Surgery: Cardiac/Thoracic Surgery. Mt. Kisco, NY, Futura, 1983, installment 9, pp 61-1-61-15.)

Avoid tension, which may lead to disruption of the repair (Fig 2). Pledgeted mattress sutures are placed around the perimeter of the defect. On the septal edge of the defect, sutures are passed through the septum from the right side to the left. On the right ventricular edge of the defect, sutures are passed from the epicardial surface of the right ventricle to the endocardial surface. When all sutures are passed through the edge of a synthetic patch, the patch is seated on the left side of the septum. Each suture is passed through an additional pledget, and then all are tied. The remaining defect in the anterior wall of the left ventricle is then closed by approximation of the edges of the ventriculotomy with mattress sutures passed through buttressing strips of Teflon felt. An additional over-and-over running suture assures hemostasis.

The same principles are applied to the closure of apical defects. The initial incision is made through the infarcted apex of the left ventricle. Debridement of necrotic myocardium results in amputation of the apex of the left ventricle and septum. The resulting defect is closed by approximating the remaining apical portion of the left and right ventricular free wall to the apical septum. A row of interrupted mattress sutures is placed through a buttressing felt strip, the left ventricular wall, an additional strip of felt, the septum, a third strip of felt, the right ventricular wall, and a final strip of felt. The sutures are tied after all have been placed, and the closure is reinforced with an additional over-and-over suture.

The greatest technical challenge has been the successful management of inferoposterior septal defects, which are associated with transmural infarction in the distribution of the posterior descending coronary artery (Fig 3).
Early attempts at primary closure of these lesions were usually followed by reopening of the defect either intraoperatively or early postoperatively because the sutures tore out from the soft myocardium that was necessarily closed under tension. The principles already outlined have evolved in part from the analysis of such early experiences. Use of the following technique has resulted in a dramatic improvement in operative survival:

- The heart is retracted as for bypass to the posterior descending artery. The margins of the infarct are best identified prior to aortic cross-clamping, and may involve the diaphragmatic aspects of both ventricles or of the left ventricle only (Fig 3A). The left ventricular portion of the infarct is excised, thereby exposing the septal defect (Fig 3B). This may often be seen best from the patient’s left side. The papillary muscles to the mitral valve are inspected, and mitral valve replacement is planned only if there is frank papillary muscle rupture. When indicated, replacement of the mitral valve is accomplished through a separate conventional left atrial approach to avoid unnecessary tearing of friable ventricular muscle while exposing the mitral valve. Debridement of left ventricular muscle should be aimed at removal of all infarcted tissue. Debridement of right ventricular muscle can be less aggressive, the objective being complete visualization of the septal defect. Delayed rupture of the right ventricle has not been a problem.

- A small inferior septal defect may be closed by approximating the edge of the septum to the right ventricular free wall using mattress sutures and buttressing strips of Teflon felt. Larger defects require patch closure. The same principles apply as in closure of a large anterior defect. Pledged mattress sutures are placed from the right side of the septum and from the epicardial side of the right ventricular free wall (Fig 3C). All sutures are passed through the patch and then through additional pledgets (Fig 3D). The patch is seated on the left ventricular side of the septum, and all sutures are then tied (Fig 3E). Direct closure of the remaining infarctectomy is generally inadvisable because of the excessive tension necessary to close the gaping defect. Patch closure of the defect has produced improved results, but early attempts at an eversion technique for patch placement led to varying degrees of disruption because of strain on the friable endocardial surface of the ventricular muscle. To alleviate this problem, an epicardial technique has evolved. Teflon felt-pledged mattress sutures are passed from the endocardial surface to the epicardial surface around the perimeter of the infarctectomy (Fig 3F). Along the septal margin of the ventriculotomy, the sutures usually pass through the edge of the septal patch and then emerge on the epicardial surface of the right ventricle. A generously cut woven Dacron patch is tailored to overlap the edges of the infarction. Each suture is passed through the perimeter of the patch and then through an additional felt pledget. All sutures are then tied (Figs 3G, 3H).

- The approaches to anterior, apical, and inferoposterior defects, although individualized, all adhere to the basic principles we have outlined. Each technique results in elimination of both the left-to-right shunt and the paradoxically expanding segment of infarcted myocardium.

- The issue of accessory coronary artery grafting remains unsettled. Some [47, 52] argue that it provides no benefit and subjects patients preoperatively to time-consuming and potentially dangerous diagnostic studies. We [10] and others [44] are employing coronary revascularization with increasing frequency. Data from patients undergoing ventricular aneurysmectomy or valve replacement suggest that revascularization will probably be associated with enhanced survival [53]. Our policy is to place aortocoronary grafts [54] to principal epicardial coronary arteries that have severe proximal stenosis.

**Operative Mortality and Postoperative Results**

Using an early operative approach, we [10] reported an overall hospital mortality of 25% in patients treated since
1975. Mortality varied with anatomical location of the septal defect. Anterior ruptures resulted in a 15% hospital mortality whereas posterior defects resulted in a 34% mortality. Long-term results are favorable with respect to survival and functional palliation. Eighteen of the 28 patients treated since 1975 are long-term survivors. Seven are in the New York Heart Association Functional Class I, 8 are in Functional Class II, and 3 are in Class III.

Similar favorable long-term results using an early operative approach were reported by Gaudiani and associates [44]. For those patients who survived hospitalization, 88 ± 8% are alive at 5 years. Seventy-four percent of these patients are in New York Heart Association Functional Class I, 21% are in Functional Class II, and 4% are in Functional Class III.

Summary
Postinfarction ventricular septal rupture constitutes a surgical emergency. The presence of cardiogenic shock is an indication for immediate surgical repair. Initial stabilization of patients with postinfarction who are not in shock should be followed by early operative intervention. Delay in surgical treatment is reserved only for those patients who are in absolutely stable condition by clinical and laboratory criteria. This group represents a small minority of all patients with postinfarction VSD.

Operative management should involve a transinfarct approach with specific repair tailored to the anatomical location of the septal defect. Enhanced intraoperative myocardial protection is achieved with cold potassium cardioplegia.

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