

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800

Open access books available

122,000

International authors and editors

135M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Post Myocardial Infarction Ventricular Septal Defect

Michael S. Firstenberg and Jason Rousseau
*Division of Cardiac Surgery, The Ohio State University Medical Center, Columbus,
USA*

1. Introduction

With advances in the management of acute myocardial infarction (AMI) the incidence of mechanical complications continues to decline. Nevertheless, when they occur, unfortunately, despite similar advances and growing experiences in the surgical management of these problems, morbidity and mortality remain high. Post-myocardial infarction ventricular septal defects (PI-VSD) have fascinated and challenged clinicians for years. The timing of presentation can be quite variable, as they tend to occur in patients several days after their initial cardiovascular insult (acute PI-VSD) - and unfortunately, they can occur in patients who appear to have been making significant progress on the road to recovery. In addition, although more rare, some patients might not present until weeks, if not longer, after their infarction with symptoms that prompt the discovery of a chronic PI-VSD. Early PI-VSDs tend to be catastrophic and typically result in early death. The pathophysiology is also variable and complex, but common themes include: 1) worsening cardiac output, often with manifestations of shock and end-organ damage, from acute left ventricular (LV) dysfunction and from increased left-right shunting, 2) acute right ventricular (RV) dysfunction from a sudden increase in pressure, volume, and flow from left to right shunts, and 3) pulmonary hypertension also from the increase in RV flow. Definitive management remains surgical, however controversies continue to exist regarding the timing of surgery and the role of concomitant coronary revascularization. Unfortunately, despite early repair and standardization of techniques, both short and long-term outcomes remain less than ideal.

2. History

As with many cardiovascular conditions, post-myocardial infarction ventricular septal defects (PI-VSD) were described first at autopsy (Latham, 1845) and then pre-mortum in 1923, many years before the pathophysiology was understood (Brunn, 1923). It was not until 1934 that the association with coronary artery disease was described (Sager, 1934). The first report of a surgical repair came in 1956 when Denton Cooley described the surgical management in a patient 9 weeks after the initial diagnosis (Cooley, 1956). With advances in cardiovascular surgery and peri-operative management of the cardiac surgery patient there were increasing reports of survival in what was previously felt to be a lethal problem. Most of the successful cases occurred in patients who presented in congestive heart failure many

weeks after their initial acute event. It was these experiences that set the foundation for the belief that operative management should be delayed as long as possible to allow for scarring of the necrotic myocardium to provide for a more stable repair. As experiences grew – in terms of the initial diagnosis and surgical management – early repair was advocated, particularly in patients who were stable before hemodynamic deterioration and subsequent end-organ failure.

3. Clinical presentation

The incidence of PI-VSD has decreased considerably over the years with advances in myocardial reperfusion strategies. Historically, up to 5% of all myocardial infarctions were associated with mechanical complications such free-wall rupture, papillary muscle rupture, and PI-VSD (Agnihotri, 2008). With current treatment algorithms that advocate early and aggressive attempts at revascularization of the acute ischemic myocardial – such as thrombolytic therapy, early percutaneous interventions with coronary stenting (PCI), and, less frequently, emergent coronary artery bypass surgery (CABG) – the overall incidence has dropped significantly. Large multi-centers studies evaluating the pathophysiologies of acute myocardial infarctions have shown a current incidence of approximately 0.2% of all AMI. With delays in therapies, or late clinical presentation, and the resulting increase in myocardial damage, this incidence increases up to 2%. Despite the relatively low risk of developing a PI-VSD, it account for a disproportionally high risk of mortality. Over 5% of all early deaths after AMI are attributed directly to the pathophysiologic complications of PI-VSD (Poulsen, 2008).

The timing of the development of a PI-VSD can be quite variable with the average time to clinical presentation is between 2 and 4 days, however presentation can be as few as a few hours after AMI or as long as several weeks.

Patient risk factors include gender, with men at a greater risk than women (3:2 ratio), increasing age, and current smoking history. The mean age of presentation in GUSTO was 62.5 years and ranged from 44 to 81 years (Crenshaw, 2000).

4. Diagnosis

The diagnosis of a PI-VSD must be considered in the differential in any patient presenting with hemodynamic impairment, particularly in the context of a sudden deterioration in a patient who otherwise had been doing well, either during or after an AMI. As a PI-VSD presents in a similar manner as other mechanical complications of AMI, such a papillary muscle rupture (with acute mitral regurgitation), free wall rupture (with contained tamponade), or severe LV failure and pulmonary edema the initial diagnosis is often suspected during initial investigations and confirmed with additional imaging.

The typical presentation occurs in a patient who is otherwise recovering after initial management of an uncomplicated AMI. Patients often complain of recurrent chest pain from what is most likely new onset or recurrence of myocardial necrosis and will develop a new systolic murmur that can be harsh, pansystolic, and often-best auscultated at the left lower sternal border. Patients can often have a bundle branch block from disruption of the septal conduction system and will quickly deteriorate hemodynamically with findings suggestive of acute cardiogenic shock.

With the acute clinical deterioration, a rapid assessment of the etiology is critical. Unlike other mechanical complications, such as papillary muscle rupture, PI-VSDs will have imaging confirming a left to right shunt – such as contrast injected into the left ventricle during catheterization crossing the defect into the RV and entering into the pulmonary arteries (Figure 1). Likewise, oxymetric assessment with right heart catheterization will demonstrate a “step-off” from the mixing of de-oxygenated RV blood with the oxygenated LV blood. Quantitative assessment of Qp:Qs will correlate with the size of defect.

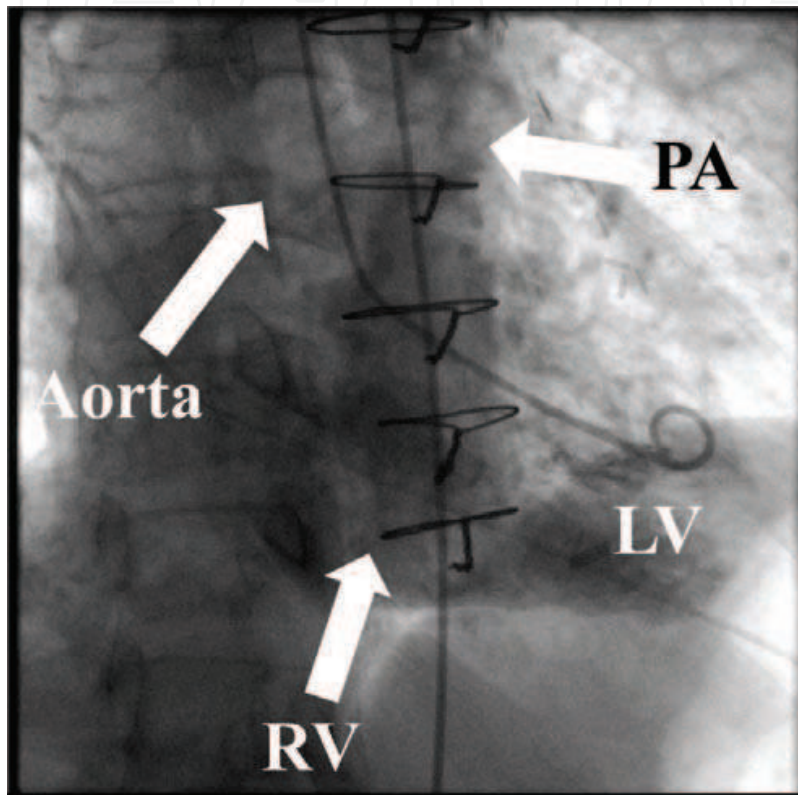


Fig. 1. Representative cardiac catheterization in which contrast is injected into the left ventricular cavity and then crosses the defect into the right ventricle. Contrast flowing into the pulmonary artery is then diagnostic for a ventricular septal defect.

4.1 Echocardiography

Transthoracic echocardiography (TTE) remains the cornerstone of the non-invasive assessment of PI-VSD (Kishon, 1993). TTE is indicated in any patient who presents with evidence of acutely impaired ventricular function or in unexplained hemodynamic deterioration suggests a mechanical complication following an acute myocardial infarction (Buda, 1991). Echocardiography has the benefit of being able to assess both left and right ventricular function, the presence of potentially co-existing and confounding valvular diseases – typically mitral regurgitation, and with color flow imaging it can be 100% specific and sensitive in diagnosing a PI-VSD. Despite the utility of TEE in the acute assessment of a deteriorating patient, a high index of suspicion is needed when looking for a PI-VSD as traditional echo windows might miss a small or apical defect. Large pericardial effusions might suggest an associated free wall rupture.

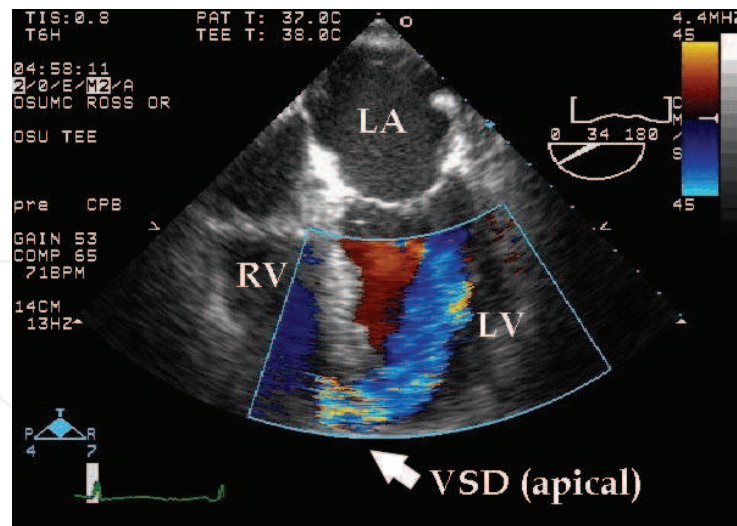


Fig. 2. Transesophageal echo, 4-chamber view, demonstration an apical VSD with flow from from the left ventricle (LV) to the right ventricle (RV). The left atrium (LA) is also shown to illustrate the typical relationship of the defect to the mitral valve.

4.2 Cardiac catheterization

Since patients who present with ECG and laboratory evidence of myocardial ischemia often undergo early cardiac catheterization with coronary angiography, this test often represents the initial study to suggest or confirm a diagnosis. Because the pathophysiology typically is associated with extensive and acute ischemia of a large territory of myocardium, it is not surprising that the findings at catheterization are often different than what would be expected for patients with a history of chronic coronary artery disease in which compensatory development of septal collaterals has had time to developed. During the acute presentation, the findings often suggest a complete occlusion of a large coronary artery in the setting of relatively minimal disease. Single vessel disease is found in 64% of patients and since the left anterior descending (LAD) artery is often the culprit vessel this explains why antero-apical septal VSDs are found in 60% of cases. Conversely, acute occlusions of a dominant right coronary or circumflex artery accounts for the remaining cases that occur in the posterior septum. Seven percent have concomitant double vessel disease, and 29% have triple vessel disease.

More importantly is that the cardiac catheterization might be the initial confirming diagnostic test. As mentioned above, quantitative assessment of oxygen step-offs, when performed, will demonstrate an increase in the partial pressure of oxygen (PaO_2) between the right atrium and ventricle - diagnostic of the left to right shunt. Left ventricular contrast injections, although less likely to be performed in a deteriorating patient secondary to the concern that additional contrast might further injure an already compromised renal function, is critical in patients with a suspected PI-VSD. Contrast injected into the LV will cross the defect (left-right shunt) and enter into the pulmonary arterial tree. This "pulmonary arteriogram" is characteristic for a VSD (Figure 1, see above).

Despite what may be perceived as the obvious value of early catheterization, the value is debatable. Opponents of mandatory catheterization advocate that in a clinically deteriorating patient in whom the diagnosis is clear it delays surgical management, the dye load may worsen already impaired renal function, and some reports suggest that considering the patterns of coronary disease typically encountered that coronary revascularization is actually a risk factor for a poor outcome (Muehrcke DD, 1992). Despite these theoretically arguments,

from a practical standpoint it is hard to argue the clear benefits of defining the coronary anatomy prior to a surgical intervention aimed at treating a complication of coronary – particularly given the long-established importance of optimal or complete revascularization. Again, since these patients have already undergone catheterization as part of the initial management of their initial ischemic event the question whether to proceed with catheterization is rarely encountered. However, as many of the patients develop septal rupture several days (or weeks) after their initial acute coronary insult, it is hard to argue the need for repeat cardiac catheterization if the diagnosis is clear, particularly if the nuances of the coronary anatomy have recently been determined. Conversely, in less clear clinical situations, repeat catheterization might suggest an alternative, and potentially more likely, diagnosis such as acute stent thrombosis or disruption of an already unstable plaque.

4.3 Magnetic Resonance Imaging

Although the diagnosis is often made at the time of initial cardiac catheterization or echocardiography, occasionally a PI-VSD may be encountered during other diagnostic imaging. While patients might be too hemodynamically unstable or the presence of an intra-aortic balloon pump (or other ferrous containing or non-MRI safe device) might preclude MRI, with the growing indications and utilization of MRI for operative planning, PI-VSD might be encountered. Patients with low ejection fractions, cardiomyopathies, or suspicion for unusual cardiac anatomy, might have MRI performed to assess for myocardial viability, fibrosis, or valvular pathology. In these patients a PI-VSD may be an unsuspected finding (Figure 3). Although there is little literature describing the utility of MRI in PI-VSDs, using concepts derived from the literature on congenital shunts and pathology, MRI might be of value in assisting in defining the extent of the defect, the shunt fraction, right ventricular function, and other associated pathophysiology (Didier, 1986). MRI might be of additive value in cases of questionable catheterization or echocardiographic studies or in assessing the post-operative patient in which a residual shunt is suspected. Nevertheless, MRI is, in general, not considered a first-line imaging diagnostic tool.

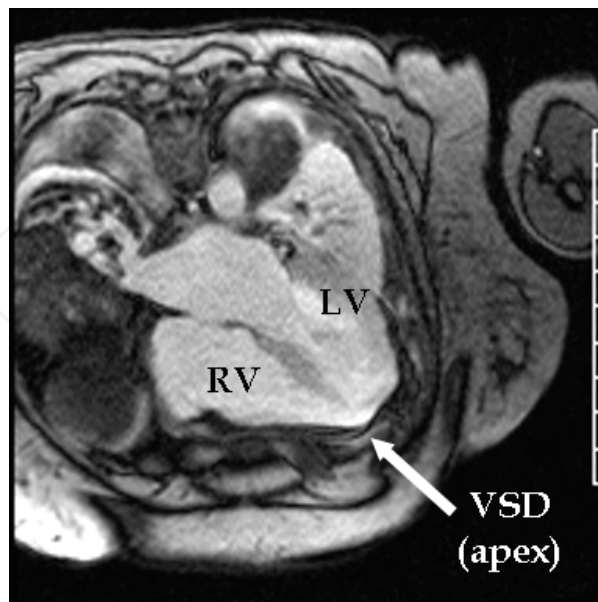


Fig. 3. Cardiac MRI demonstrating an apical defect. Gated cine images indicated a left to right shunt in which quantitative assessment can be used to determine the shunt fractions and size of the defect.

5. Pathogenesis

The pathogenesis reflects two different types of rupture. The first, a simple rupture, is a direct through-and-through defect that is typically located anteriorly (when associated with a LAD territory infarct). Conversely, complex ruptures are believed to result from tracking of blood as it dissects thru the septum with left ventricular entry sites remote from right ventricular exit sites – these tracks then enlarge over time due to the pressure gradient between the left and right ventricle. Multiple defects are found in 5-11% of cases and emphasized the need for a complex pre-operative and intra-operative assessment of all pathways to insure a complete repair (Edwards, 1984). Incomplete closure of residual or secondary defects can account for post-operative recurrences. Transmural infarcts can be quite extensive with defects developing to several centimeters in diameter and can often involve extensive areas of the left ventricular free wall. For complex defects, as blood dissects through the necrotic myocardium there can be further expansion and damage with loss of cellular integrity. With local cellular destruction there is fragmentation with degeneration of myocytes with enzymatic digestion and destruction. In patients who survive the acute presentation, up to 66% develop chronic ventricular aneurysms and a third will have significant functional mitral regurgitation from the secondary effects on the ventricular free wall.

The pathologic consequences and outcomes of surgery of anterior and posterior defects appear to be different in ways beyond what can be explained by the degree of shunting. Earlier autopsy studies have shown that anterior PI-VSDs were associated with 33% of the LV and only 10% of the RV being infarcted, while posterior defects were associated with only 20% of the LV and 33% of the RV being infarcted (Cummings, 1977). Particularly considering the acute pressure/volume overload and associated RV failure, it becomes understandable why posterior based defects have a worse prognosis.

6. Natural history

The natural history of untreated PI-VSD is also poorly understood. As advances in the acute and chronic management of coronary artery disease continues to evolve, so does complications of CAD. In general, 25% of patients with PI-VSDs die within the first 24 hours (Berger, 1992). Death is most likely a function of pre-existing comorbidities and potentially irreversible and catastrophic heart failure that comes from not only the acute pump failure from the precipitating infarct but also the massive acute left to right heart shunting that compromises systemic perfusion further. The sudden increase in pulmonary overcirculation also contributes to the development of significant right heart failure. For those patients who survive the acute event, 1, 2, and 4-week survival is 50%, 35%, and 20% respectively (Lemery, 1992). It is easy to appreciate that those patients who survive the first month may have inherently favorable factors that might further self-select for a good post-operative outcome. Prolonged untreated survival has been reported with up to 7% of patient surviving to 1 year – obviously the physiologic insult and over-circulation is minimal.

7. Timing/indications for surgery

The mere presence of a PI-VSD is considered an indication for surgery with the majority of patients undergoing urgent or emergent operative intervention. The primary goal of VSD closure is to reduce the end-organ damage from the combined insults of acute right ventricular overload/failure and systemic cardiogenic shock.

As soon as the diagnosis is made an intra-aortic balloon pump should be placed. Coronary augmentation will assist the ischemic and compromised myocardium, but more importantly it will unload the left ventricle and improve cardiac output, end-organ perfusion, and reduce pulmonary shunting and over-circulation. However, the physiologic improvements with IABP and other inotropic or vasoactive medications should only be viewed as transient and allow for finishing the pre-operative assessment.

Although some advocate a strategy of delayed repair, this approach is rarely successful. The theory is to give the friable necrotic myocardium time (3-6 weeks) to fibrosis thereby allowing for an easier and more secure repair. The scarred tissue will better hold suture and less likely to tear apart and result in an early post-operative failure. This approach appears reasonable, but it is rare that patients remain stable or can be support during this time period. While guidelines for delayed surgical management are lacking, this might be an option in those who are hemodynamically/physiologically stable with a delayed presentation, have no or minimal signs of pulmonary hypertension or over-circulation, and have a stable fluid balance with good renal function. Unfortunately, such patients are rare and less than 5-10% of all PI-VSD patients will survive to allow for delayed repair. Such an approach may represent a "survival of the fittest" perspective in those with minimal shunting and with strict attention to medical comorbidities and nutrition a period of close watchful waiting may work. This approach may also be used to justify waiting in patients who have other severe comorbidities preclude intervention and would in theory require optimization prior to surgery. Nevertheless, it is hard to argue that any other problem would improve to the point of making surgery safer in the setting of worsening right ventricular heart failure - a problem that by itself is very difficult to treat both pre and post-operatively. Although, one can argue that in these patients, unless early surgical repair is clearly contraindicated, that their physiologic reserve combined to a minimal pathophysiologic insult predisposes to a good outcome regardless of whether an early or late repair is performed.

8. Operative management

The initial attempts at repairing PI-VSD followed a surgical approach similar to that for congenital VSDs - through a ventriculotomy in the right ventricular outflow track (RVOT) (Cooley, 1957). It was quickly appreciated the significant limitations of this approach. Firstly, in an already compromised right ventricle, the outflow tract incision only further reduced residual RV function. In addition, while suited for many common types congenital VSD's near the aortic valve, the RVOT incision offered poor exposure of defects that tended to be much further down the septum towards the apex. Most importantly, because the patch and suture line was on the RV side, the defect was still exposed to LV pressures and consequently was at increased risk for patch dehiscence, early recurrence, and extension of the defect. Pioneering work by animal studies by Heimbecker in 1968 advocated an approach to the VSD thru the left ventricle in the region of the culprit vessel through infarcted myocardium - i.e., anterior defects approached through the anterior wall while posterior defects through the inferior wall. These techniques addressed the deficiencies of a RVOT approach (Heimbecker, 1968). The benefits of these animal studies were subsequently validated clinically within several years (Javid, 1972)(Buckley, 1971)

8.1 General considerations

After the patient arrives in the operating room, the patient is routinely anesthetized and pharmacologically paralyzed. If not already in place, all patients should have arterial monitoring lines and a pulmonary artery (Swan-Ganz) catheter inserted. Pressures and

oxygen saturations should be obtained to determine shunt fractions and to assist in determining the completeness of repair at the end of the procedure. Given that this subset of patients possesses a left-to-right shunt, it is of utmost importance to avoid pharmacologic agents that cause pulmonary vasodilation, thus worsening the shunt, increasing pulmonary over-circulation, and potentially worsening right heart dysfunction. Preoperative antibiotics including a first generation cephalosporin, such as cefazolin, and vancomycin are administered. In cases of antibiotic allergies, appropriate alternatives should be chosen.

Median sternotomy is performed and the patient is prepared for cardiopulmonary bypass. Minimally invasive techniques are typically not advocated for this type of extensive and complex procedure in which complete exposure of heart is helpful. However, in situations of re-operative surgery, depending on surgeon preferences, consideration should be given to peripheral cannulation (i.e. femoral or axillary) prior to sternotomy as a re-entry injury to an already compromised and dilated RV can be catastrophic. The patient is heparinized prior to standard aortic - right atrial cannulation. Some advocate routine bicaval venous drainage, but typically, as procedures on the tricuspid or mitral valves are not performed, this is not necessary. Cold-blood antegrade and retrograde cardioplegia is delivered via conventional root and coronary sinus catheters. Topical cooling to further reduce the metabolic demands of the already compromised heart is also liberally used. This author also routinely uses ice-slush wrapped in gauze to further cool the right ventricle to assist in reducing the temperature and thereby assisting in myocardial protection. Active or passive systemic cooling is performed with an ideal temperature of 25°-28°C to further assist end-organ protection.

In general, once the defect is identified, a piece of either glutaraldehyde-fixed bovine pericardium or Dacron is cut to not only cover the defect but a generous rim of surrounding, and potentially non-viable, myocardium. Continuous suture or interrupted pledgeted sutures are used to suture the patch to the residual septum. Tension on the repair must be avoided to minimize the risk of the sutures tearing through once the ventricle is pressurized and begins to contract. For posterior defects the patch might require anchoring to the annulus of the mitral valve. For cases in which the annulus of the mitral valve and/or perivalvular tissues is involved, mitral valve replacement may be required.

The intra-operative approach and management of the defect is based upon the location of the VSD and, the need for concomitant procedures. The pre-operative assessment of the location of the defect is critical in determining the optimal approach to closing it.

8.2 Apical defects

Apical septal ruptures involve the cardiac apex, which includes the apical portion of the right ventricle, septum, and the left ventricle. Daggett and colleagues described the technique of apical amputation and repair of the PI-VSD in 1970. The initial incision is created through the infarcted tissue of the cardiac apex. The necrotic myocardium is then

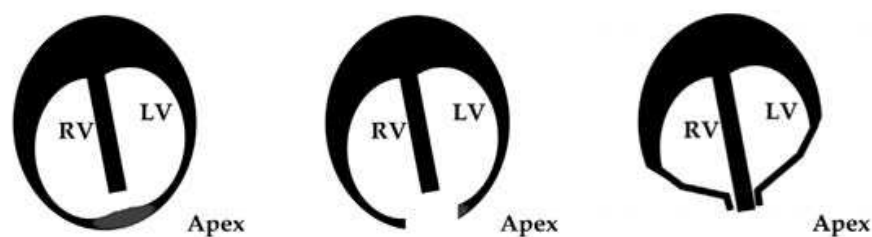


Fig. 4. Repair of apical repair involves excising the apical defect and bringing together the residual edges of the left and right ventricular walls using a primary repair reinforced with pledgets.

excised until healthy muscle is exposed and deemed adequate for repair. The healthy tissues of the right and left ventricle are then approximated to the septum using interrupted, felt pledgeted, heavy Tycron suture in a mattress fashion (Figure 4). Felt strips are placed along the right and left septal walls during this process to create a 'felt sandwich'. The apical repair can be reinforced with a second layer of suture. Meticulous hemostasis is critical.

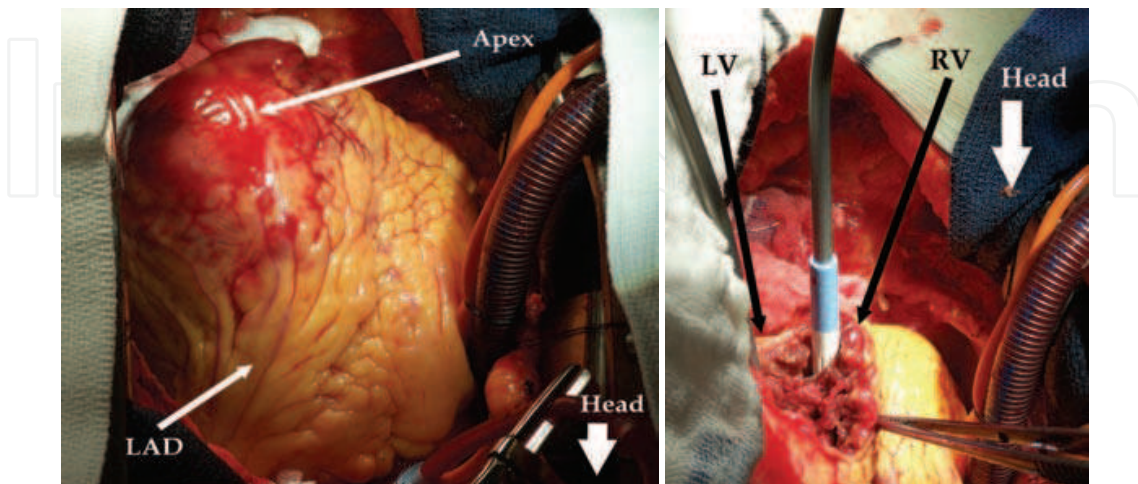


Fig. 5. Left: Intra-operative view of the extensive apical infarction that has resulted in the the echocardiographic findings demonstrated in Figure 2. The left anterior descending artery (LAD) is shown. Right: The same patient after opening and debridement of the infarcted apex. The necrotic septum is visualized with a probe in the left ventricle (LV) and the bypass "pump sucker" is in the right ventricle (RV).

8.3 Anterior defects

The anterior septal rupture involves the anterior septum as well as the anterior left ventricular free wall. This, as discussed earlier, is a typically a result of acute infarct of the LAD territory. The initial approach is via an incision in the infarcted left ventricular myocardium. The infarcted area is then excised and debrided back to healthy, viable myocardium. The septum is then inspected and necrotic tissue is excised in the same fashion. This is can be straightforward in a single, obvious defect. However, great care must be taken if the defect is noted to be tracking through the myocardium - a finding that might not be obvious and hence a larger patch may be used than what would be employed to cover the defect.

Small defects can be plicated to the right ventricular free wall using interrupted, pledgeted suture as first suggested by Shumacker (Shumaker, 1972). For anything other than very small (<1.0 cm) defects, most anterior septal infarcts will require repair with a patch. This is created in a fashion that will allow for a tension-free repair. Excess tension in the repair can lead to devastating consequences and a return trip to the operating room.

The more complex approaches use a prosthetic patch that is anchored to the posterior wall of the septum using interrupted, pledgeted Tycron or Prolene suture. The suture is passed from a right to left direction so that the patch-septum interface lies in the left ventricle, as opposed to the right. The anterior sutures are placed through the right ventricular free wall and tagged with hemostats (Figure 6). All sutures are placed prior to placing them through the patch. They can now be placed through the designated anterior region of the prosthetic patch and then through a second pledget if desired prior to tying the suture knots. The left ventricular free wall is then re-approximated using interrupted, mattress suture. A second layer of running suture is placed for reinforcement.

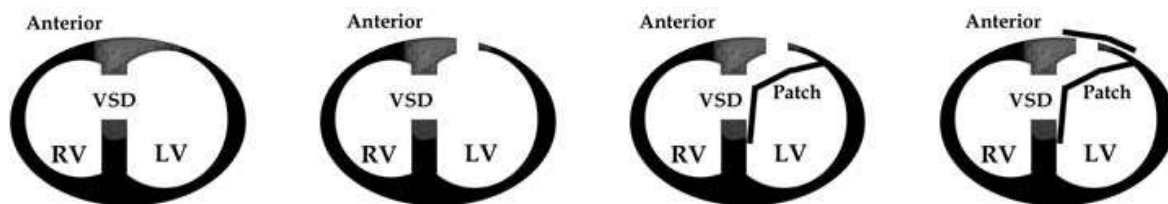


Fig. 6. Picture representation of the various steps used to repair an anterior defect with the patch excluding the necrotic septum from the higher pressure left ventricular cavity. The incision is through the infarcted muscle on the anterior wall, parallel to the left anterior descending artery.

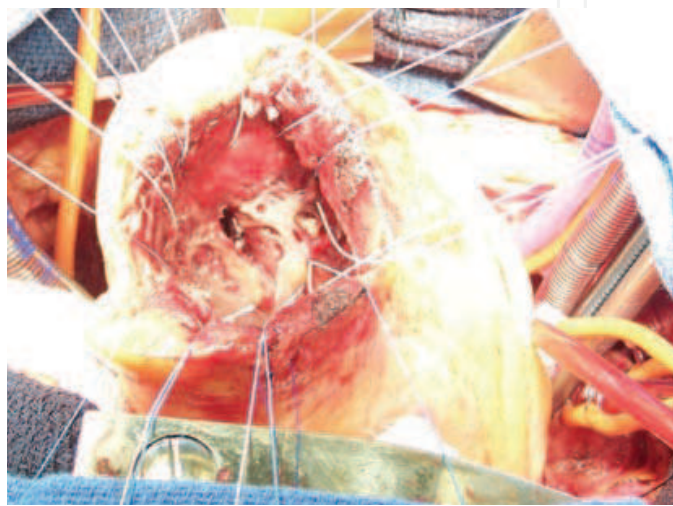


Fig. 7. Intra-operative picture with the apex of the heart elevated (head and aortic cannula to the right). The incision is through the infarcted anterior wall. The septal defect is shown in the middle of the cavity with circumferential sutures around a wide margin. The sutures will then be placed through a pericardial patch to exclude the infarcted septal muscle and defect from the left ventricular cavity. Note: This is from a different patient than from the images above

8.4 Posterior defects

The posterior or inferior septal rupture involves a transmural infarction of the myocardium in the posterior descending arterial distribution. The inferior wall is often thin and after infarction, is quite friable. For this reason, primary repair is not a durable option and is rarely successful. Attempt at primary repair, in which the myocardium is placed under tension, can have disastrous and immediate, and potentially fatal, consequences. Hence, posterior/inferior septal ruptures are the most technically demanding of the PI-VSDs.

After the heart is arrested, the inferior wall is lifted out of the pericardial well and exposed. The transmural infarct may involve both ventricles, or the septum and left ventricle alone. The transinfarct incision is created in a longitudinal fashion in the left ventricle. The nonviable myocardium is excised, which will create adequate exposure of the septal defect. The papillary muscles are inspected. If the base is involved in the infarct resulting in ruptured papillary muscle, then mitral valve replacement is warranted. If a small posterior VSD (<1.0 cm) is identified, primary repair to the ventricular free wall using pledgetted suture as described earlier is satisfactory - but this situation is very rare and placement of a small patch may result in a more durable outcome than risking a primary repair involving ischemic myocardial tissue.

Large defects will require a tension free repair by utilizing a patch closure. This technique often necessitates the use of two separate patches, one dedicated to the septal repair and the other to the wall of the ventricle. Principles as described previously apply. The pledgetted mattress sutures are placed from the right ventricle to the left along the circumference of the VSD. The sutures are passed through the contoured patch and tied down. Great care should be taken to avoid lacerating the myocardium. Some authors suggest placing a second pledget on the patch side of the repair to minimize this risk. The posterior ventricular wall is repaired with the second patch using mattress sutures. Occasionally, depending on the size and quality of free wall myocardium, the free-edges can be approximated and closed primarily (and re-enforced with a pericardial or felt strip) rather than using a second patch.

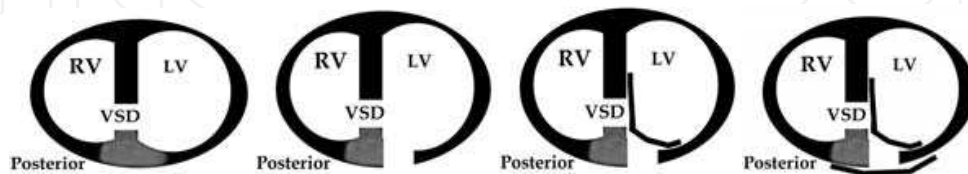


Fig. 8. Similar to anterior repairs, the high pressure left ventricular cavity is isolated from the necrotic septum with a patch repair. The incision is along the distal right coronary along and parallel to the posterior descending artery through the infarcted basal muscle.

8.5 General principles

Closure of the ventriculotomy is performed by folding the free edge of the patch to the edge of the ventricle to exclude it from the circulation. The ventriculotomy repair is then closed with a primary closure re-enforced with strips of either Teflon felt or pericardium. Exclusion of the necrotic myocardium from the left ventricular is also important in minimizing the risk of small debris breaking off at any point and causing a systemic embolism.

Regardless of the location of the ventriculotomy, it cannot be emphasized enough the importance of a tension free closure. Any unnecessary tension through injured or friable myocardium may predispose to catastrophic and potentially fatal post-operative bleeding once the ventricle becomes pressurized. In extreme cases involving extensive myocardial (free wall and septal) damage, temporary mechanical support with either extra-corporeal membrane oxygenation (ECMO) or a left ventricular assist device may help unload the ventricle to assist in recovery. The hypothesis behind this approach is by reducing the LV pressure, it will encourage recovery, reduce the pressure on the repair, and allow for further decision making in patients in whom there is extensive ventricular destruction and residual ventricular function may not be adequate to support physiologic needs (Firstenberg, 2009).

Although the benefits of concomitant revascularization on long-term outcomes are debatable, complete coronary revascularization, if possible, is typically advocated (Heitmiller, 1986). As with other risk models for outcomes after surgery (e.g. EuroScore and STS models), it is the need for revascularization and the extent of underlying CAD that defines the long-term outcome rather than the actual performing of the procedure. Overall, the paradigm of complete and/or optimal revascularization should apply in cases of PI-VSD management. It is hard to argue the conceptual benefits of revascularization in the setting of an already acutely and chronically compromised myocardium.

9. Post-operation management

The post-operative management of patients following successful repair should be similar to that of other high-risk surgical patients. However, there are several key points that need to

be considered. As these patients often present and are taken to the operating room in acute decompensated heart failure, strict attention to optimizing biventricular function is critical. Post-operative left ventricular dysfunction is not uncommon and, unless already in place, there should be a low threshold for placement of an intra-aortic balloon pump (IABP). While, as discussed later, the use of an IABP is often associated with worse outcomes, the relationship is the need for it and the potential delaying in initiating its use rather than the therapy itself that influences the outcomes. Right heart failure is common and often these patients require considerable therapies directed specifically at assisting in right heart recovery. Conventional intravenous agents such as epinephrine, milrinone, and dobutamine are often required – and sometimes at high doses. Inhaled agents that selectively reduce pulmonary vascular resistance and assist in reducing RV afterload such as inhaled nitric oxide (20-80 ppm) or Epoprostenol (2,500 – 20,000 ng/min) may be required (Rich, 1993). Ventricular arrhythmias are also common from the residual ischemic/necrotic myocardium (as well as secondary to the ventriculotomy) and anti-arrhythmic medications, such as Amiodarone, should be used liberally. In addition, as the repaired septal defect and free wall are often quite friable, strict attention to avoid hypertension is critical as even transient elevations in blood pressure can result in disruptions in the patch repair that might precipitate uncontrolled and fatal LV bleeding. Any acute increase in chest tube drainage should raise the concern for ventricular suture line dehiscence and there should be a low threshold for returning the patient to the operating room for re-exploration – however, excess manipulation of the heart in the search for bleeding should be avoided at the risk of catastrophic suture-line tearing in a beating and pressurized ventricle. Although recovery in these patients is unpredictable and may be prolonged and a slow wean of inotropes may be required, there should be a low threshold for repeat and/or frequent echocardiographic assessment in a patient who is not improving as predicted. Repeat echocardiography might show a residual shunt or valvular dysfunction, more importantly would be identification easy to correct problems, such as tamponade.

10. Outcomes: predictors of survival

The increasing rarity of PI-VSD implies that few centers are able to report an extensive series. While several large single center experiences and outcomes have been reported, most summarize years of experience and may not consider the ongoing evolution in the peri-operative management and improvement in surgical skills and judgment of these critically ill patients.

Deja and colleagues reported their experience with 117 patients from the Glenfield General Hospital in England. The mean age was 65 ± 8 years and there were 43 females. Of the 117, 76 were anterior defects while 34 were posterior. One third of patients presented in cardiogenic shock. The average time from AMI to the development (or diagnosis) of a PI-VSD was 6 days and there was an additional 9-day average interval before surgical intervention. Their overall mortality was 37% - not including a 6.4% intra-operative mortality. 40% have evidence of a residual left-right shunt with 13 patients undergoing early re-operation with 30% mortality in the re-op group. Table 1 summarizes their overall results. Their overall predictors of post-operative mortality include: shock at time of surgery, clinical deterioration while awaiting surgery, need for concomitant CABG, and pre-operative renal failure (as a marker for shock and organ failure). Criticisms of these results include that earlier surgical intervention after initial diagnosis might have resulted in less end-organ damage and an already compromised patient (Deja MA, 2002).

Overall surgical mortality (%)	37%
Intra-operative mortality (%)	6.4 %
ICU Stay (days)	4.8 days
Major inotropic support	90 %
Used of Intra-aortic balloon pump	75 %
Need for extra-corporeal membrane oxygenation (ECMO)	2 %
Ventilator Time (hours)	40 hours
Tracheostomy	5%
Continuous renal replacement	16 %
Re-exploration/bleeding	5 %
Stroke	5 %
Residual shunt	40%

Table 1. Post-Operative Complications Following Surgical Repair (From Deja MA, 2002)

National registry data has proven useful to define the real-world experiences with the presentation and management of PI-VSD. In the report of 189 patients from Sweden, several factors were able to predict favorable vs unfavorable short (<30 days) and long-term (>30) outcomes (Tables 2 and 3) (Jeppsson, 2005).

Favorable Predictors	Unfavorable Predictors	No effect on outcome
Short time from MI to Dx	IABP	Age
Short time from Dx to OR	Stroke/Coma	Gender
Short time from MI to OR	Renal failure	Pre-OP IABP
Pre-operative catheterization	Re-op for bleeding	Pre-OP Lytic therapy
Anterior rupture		Concomitant CABG
		Residual shunt

Table 2. Predictors of short-term (<30-day) survival based on National Swedish Experience (Adapted from Jeppsson et al. Euro J Cardiothor Surg 2005;27:216-221). CABG: Coronary artery bypass surgery, Dx: Diagnosis, IABP: Intra-aortic balloon pump, MI: Myocardial infarction.

Favorable Predictors	Unfavorable Predictors	No effect on outcome
Younger age	Pre-Op IABP	Anterior rupture
	Pre-Op Cath	Time from MI to OR
	Need for CABG	Post-Op stroke
	Renal Failure requiring dialysis	Pre-OP Lytic therapy
	Residual shunt	Post-Op IABP
		Re-op for bleeding

Table 3. Predictors of Long-Term (>30 day). See above for legend.

In a similar national registry experience, Cerin and colleagues reported the outcomes in 58 patients treated with PI-VSD from 1992 to 2000 in Italy (Cerin, 2003). The mean age was 73

years/old. Thirty-six percent presented in acute renal failure, 33% were in atrial fibrillation, and 22% were insulin dependent diabetics. 57% were in NYHA Class IV heart failure with 41% in cardiogenic shock. Intra-aortic balloon pumps were used in only 20% of patients and 60% presented with significant mitral regurgitation. The timing of surgery was 14 ± 12 days from the acute event with 76% undergoing surgery within the first 3 weeks and 31% within the first 24 hours. A key point is again emphasizing the importance of early diagnosis and surgery before the onset of shock and organ failure (Table 4).

	Survivors (n=28)	Death (n=30)
Time to OR (d)	21 ± 13	11 ± 8
OR < 24hrs	18 %	43 %
Pre-Shock	28 %	57 %
Pre-sPAP (mmHg)	42 ± 11	56 ± 14
CPB time (time)	95 ± 28	126 ± 35
Post-IABP	39 %	90 %
Post-LVEF (%)	45 ± 2	29 ± 2
Post-Op Renal Failure	25 %	66 %

Table 4. Italian Registry Data. Legend: CPB: Cardiopulmonary bypass, IABP: Intra-aortic balloon pump, LVEF: Left ventricular ejection fraction, sPAP: Systolic pulmonary artery pressures. Table adapted from Cerin et al. *Inter Soc Cardiovasc Surg* 2003;11:149-154

More recently, Mantovani et al reported their 19-year, single center, experience in 50 patients. Between 1983 and 2002, 50 consecutive patients with a mean age of 66 ± 9 years (range: 45-81) who presented with either anterior (n=30, 60%) or posterior (n=20, 40%) PI-VSDs. Patients developed their defects on average 4 days post-AMI with most within the first week (76%) and only 2 patients presenting after 2 days. Cardiac catheterization was performed in 98% of patients (51% single vessel disease, 35% double, and 14% triple). 56% of patients required a pre-operative IABP and 74% underwent emergent surgery on average 2 days after diagnosis of a PI-VSD. Operative mortality (within 30 days) was 36% with 6 dying in the operating room. Posterior defects were associated with 50% mortality versus 25% for anterior. Other univariate risk factors for early death included: emergent surgery (p=0.02); cross-clamp time >100 minutes (p=0.035); and delayed surgical intervention (>3 days post diagnosis, p=0.0055). Interestingly, in their experience factors not associated with operative mortality included: gender, extent of CAD (single vs triple vessel disease), need for CABG, age (>65 years), or the year of operation (before/after 1992). In a logistic regression analysis, only emergent surgery (odds ratio: 10.23) and a delayed treatment (OR: 4.03) were the only predictors of early mortality. Long-term survival was 76.5 ± 7.8 and $56.1 \pm 11.5\%$ at 5 and 10 years. No obvious predictors of long-term survival were found in their analysis although patients with residual myocardium at risk from unrevascularized regions tended to have a worse long-term prognosis (Mantovani, 2006).

In the GUSTO trial in which 41,021 patients were randomized to different strategies of reperfusion during AMI, 84 developed a PI-VSD. 34 of these were managed surgically, with 31 (90%) undergoing early treated and 3 (10%) undergoing delayed surgery. Survival in the surgical group was 53% at 30 days and 47% at 1 year. Conversely, for those treated medically, as an indicator of the lethality of this problem, survival at 30 days and 1 year was 6% and 3%, respectively (Crenshaw, 2000). All patients who presented in Class III or IV heart failure died.

11. Controversial topics

11.1 Percutaneous closure devices

Successful application of closure devices in children with congenital VSDs combined with the morbidity and mortality associated with surgical management has prompted enthusiasm for the use of percutaneous closure devices. The role of such devices has been proposed for both the primary closure of acute defects and to assist in the closure of recurrent or residual shunts (Shah, 2005).

While conceptually promising, initial experiences were discouraging and improvements in outcomes were not observed (Pienvichit, 2001). Difficulties in covering not only the actual defect, but also the residual necrotic myocardium predisposed to early recurrence. Early devices tended not to be large enough and were very difficult to position (or re-position if necessary).

In addition, the lack of an overall survival benefit further illustrates that clinical outcomes are not only dictated by closure of the VSD, but sometimes more importantly, a function of the extent of the myocardial infarction – which might be inherently so extensive as to preclude survival.

11.2 Mechanical support

As discussed, despite advances in surgical and post-operative management, operative mortality is still 10-60%. Even with early intervention biventricular failure is often a significant factor in early post-operative deaths. Short and long-term mechanical support, beyond intra-aortic balloon counterpulsation, is a reasonable option in patients with post-operative ventricular (left, right, or bi) failure and who are felt to be salvageable. Short-term support may be required as a bridge to recovery, while long-term device may be indicated for those with irreversible ventricular failure.

In cases in which there is extensive ventricular infarction/failure, associated free-wall rupture, or when there is excessive bleeding or tension from the ventriculotomy temporary left ventricular support should be considered. With LVAD inflow drainage from the left atrium, the LV is unloaded (i.e. 'atrialized') and may allow time for recovery/healing prior to exposing the injured LV to system pressures and contractile function (Firstenberg 2009).

Right ventricular support is also difficult following the acute volume/pressure overload of a PI-VSD with recovery unpredictable and potentially prolonged. Unfortunately, there is little data to support this use in this application other than clinical judgment and center experience.

Residual shunts after repair pose a unique challenge for patients requiring mechanical support. Careful attention to left and right ventricular flows and pressures are critical to compensate for the residual shunt – and prevent worsening of over-circulation (Sai-Sudhakar, 2006). If residual shunts are significant then biventricular support may allow for a period of recovery and stabilization prior to an attempted repair in an otherwise very high-risk surgical patient.

The need for mechanical support, while attractive in unstable post-operative patients, is also associated with difficult problems. Often there is need for aggressive anti-coagulation, the need for multiple surgical procedures (i.e. device change-outs, explants, etc), and overall patient recovery is more difficult when tethered to external VAD controllers.

A total artificial heart precludes native cardiac recovery and obligates transplantation, nevertheless, it may be an option with appropriate resources and experience in highly selected patient with few other comorbidities.

11.3 Residual/recurrent defects

Residual shunts are found in up to 25% of patients after definitive repair (Skillington, 1990). The etiology of residual shunts is either a missed defect at the time of initial repair, dehiscence of a patch (sewn to necrotic or friable tissue), or further extension of the initial defect. Fortunately, most residual shunts tend to be physiologically tolerated and spontaneous closure has been reported. Operative re-intervention is associated with a >60% mortality (Jeppsson, 2005) and surgery is reserved for patients in heart failure failing medical management or those with large shunts ($Q_p:Q_s > 2.0$) (Murashita, 2010). Because of the high operative mortality with repairing residual or recurrent shunts there has been interest, but limited success, with percutaneous closure devices (Shah, 2005). Nevertheless, the role of percutaneous closure and the ideal devices are undefined (Pienvichit, 2001) and probably best reserved for use in those centers with extensive experience in the closure of congenital VSDs.

12. Conclusions

Post-myocardial infarction ventricular septal defects complicate up to 0.02% of acute myocardial infarctions. Despite advances in the surgical care of these moribund patients, operative mortality still approaches 50% with major risks including cardiogenic shock, renal failure, right and/or left ventricular failure, size of VSD, posterior/inferior locations, and residual VSD. While some patient may present late or benefit from a delayed repair, typically surgical intervention is indicated prior to irreversible end-organ damage. Repair techniques emphasize closure of the defect and protecting the injured septum from left ventricular pressures. Post-operative management is typically challenging considering the inherent pre-operative biventricular dysfunction and associated end-organ damage. Those who survive their initial event and operation tend to have favorable 5 and 10-year survivals.

13. Conflicts of interest

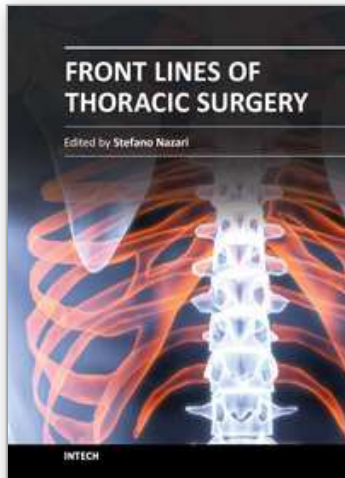
The authors have no conflicts of interest or disclosures related to any of the topics or technologies discussed in this manuscript.

14. References

- Agnihotri AK, Madsen JC, Daggett WM. Surgical Treatment of Complications of Acute Myocardial Infarction: Postinfarction Ventricular Septal Defect and Free Wall Rupture. Cohn L, ed. *Cardiac Surgery in the Adult*. New York: McGraw-Hill, 2008:753-784.
- Berger TJ, Blackstone EH, Kirklin JW. Postinfarction ventricular septal defect, in Kirklin JW, Barratt-Boyes BG (eds): *Cardiac Surgery*. New York, Churchill Livingstone, 1993; p 403.

- Brunn F. Diagnostik der erworbenen ruptur der kammerscheidewand des herzens. *Wien Arch Inn Med* 1923; 6:533.
- Buckley MJ, Mundth ED, Daggett WM, DeSanctis RW, Sanders CA, Austen WG. Surgical therapy for early complications of myocardial infarction. *Surgery*. 1971;70(6):814-20
- Buda AJ. The role of echocardiography in the evaluation of mechanical complications of acute myocardial infarction. *Circulation*. 1991;84(3 Suppl):I109-21.
- Cerin G, Di Donato M, Dimulescu D, Montericcio V, Menicanti L, Frigiola A, De Ambroggi L. Surgical treatment of ventricular septal defect complicating acute myocardial infarction. Experience of a north Italian referral hospital. *Cardiovasc Surg*. 2003 Apr;11(2):149-54.
- Cooley DA, Belmonte BA, Zeis LB, Schnur S. Surgical repair of ruptured interventricular septum following acute myocardial infarction. *Surgery*. 1957;41(6):930-7.
- Crenshaw BS, Granger CB, Birnbaum Y. et al. Risk factors, angiographic patterns and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-1 (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries). Trial Investigators. *Circulation*. 2000; 101(1):27-32.
- Cummings RG, Califf R, Jones RN, Reimer KA, King YH, Lowe JE. Correlates of survival in patients with postinfarction ventricular septal defect. *Ann Thorac Surg* 1989;47:824-30 (update: 1997;63:1508-9).
- 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(1):33-42.
- Deja MA, Szostek J, Widenka K, et al. Post infarction ventricular septal defect – can we do better? *Eur J Cardiothorac Surg*. 2000; 18:194
- Didier D, Higgins CB. Identification and localization of ventricular septal defect by gated magnetic resonance imaging. *Am J Cardiol*. 1986;57(15):1363-8.
- 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.
- Firstenberg MS, Blais D, Crestanello J, Sai-Sudhakar C, Sirak J, Louis LB, Vesco P, Sun B. Long-term mechanical support for complex left ventricular postinfarct pseudoaneurysms. *Heart Surg Forum*. 2009;12(5):E291-3.
- Heimbecker RO, Lemire G, Chen C. Surgery for massive myocardial infarction. An experimental study of emergency infarctectomy with a preliminary report on the clinical application. *Circulation*. 1968;37(4 Suppl):II3-11.
- Heitmiller R, Jacobs ML, Daggett WM. Surgical Management of Postinfarction Ventricular Septal Rupture. *Ann Thorac Surg*. 1986; 41(6):683-691.
- Javid H, Hunter JA, Naiafi H, Dye WS, Julian OC. Left ventricular approach for the repair of ventricular septal perforation and infarctectomy. *J Thorac Cardiovasc Surg*. 1972 Jan;63(1):14-24.
- Jeppsson A, Liden H, Johnsson P, Hartford M, Radegran K. Surgical repair of post infarction ventricular septal defects: a national experience. *Euro J Cardiothor Surg*. 2005;27: 216-221.
- Kishon Y, Iqbal A, Oh JK, Gersh BJ, Freeman WK, Seward JB, Tajik AJ. Evolution of echocardiographic modalities in detection of postmyocardial infarction ventricular

- septal defect and papillary muscle rupture: study of 62 patients. *Am Heart J.* 1993;126(3 Pt 1):667-75.
- Labrousse L, Choukroun E, Chevalier JM, et al. Surgery for post-infarction ventricular septal defect (VSD): risk factors for hospital death and long term results. *Euro J Cardiothor Surg.* 2002;21:725-732.
- Latham PM. Lectures on Subjects Connected with Clinical Medicine Comprising Diseases of the Heart. London, Longman Rees, 1845.
- 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
- Mantovani V, Mariscalco G, Leva C, Blanzola C, Sala A. Surgical repair of post-infarction ventricular septal defect: 19 years of experience. *International Journal of Cardiology.* 2006;108: 202-206.
- Muehrcke DD, Daggett WM, Buckley MJ, et al: Postinfarct ventricular septal defect repair: effect of coronary artery bypass grafting. *Ann Thorac Surg.* 1992; 54:876.
- Murashita T, Komiya T, Tamura N, Sakaguchi G, Kobayashi T, Sunagawa G. The clinical challenge to reduce the postoperative residual shunt in surgical repair of postinfarction ventricular septal perforation. *Interact Cardiovasc Thorac Surg.* 2010;11(1):38-41.
- Pienvichit P, Piemonte TC. Percutaneous closure of postmyocardial infarction ventricular septal defect with the CardioSEAL septal occluder implant. *Catheterization and Cardiovascular Interventions.* 2001;54:490-494.
- Poulsen SH, Praestholm M, Munk, Wierup P, Egeblad H, Nielsen-Knudsk JE. Ventricular septal rupture complicating acute myocardial infarction: Clinical characteristics and contemporary outcome. *Ann Thorac Surg.* 2008;85:1591-1596.
- Rich GF, Murphy GD Jr, Roos CM, Johns RA. Inhaled nitric oxide. Selective pulmonary vasodilation in cardiac surgical patients. *Anesthesiology.* 1993;78(6):1028-35.
- Sai-Sudhakar CB, Firstenberg MS, Sun B. Biventricular mechanical assist for complex, acute post-infarction ventricular septal defect. *J Thorac Cardiovasc Surg.* 2006;132(5):1238-9
- Sager R. Coronary thrombosis: perforation of the infarcted interventricular septum. *Arch Intern Med.* 1934; 53:140
- Shah NR, Goldstein JA, Balzer DT, Lasala JM, Moazami N. Transcatheter repair of recurrent postinfarct ventricular septal defects. *Ann Thorac Surg.* 2005;80(5):1907-9.
- Shumacker H. Suggestions concerning operative management of postinfarction ventricular septal defects. *J Thorac Cardiovasc Surg.* 1972; 64:452
- Skillington PD, Davies RH, Luff AJ, et al. Surgical treatment for infarct-related ventricular septal defects. Improved early results combined with analysis of late functional status. *J Thorac Cardiovasc Surg.* 1990;99(5):798-808.



Front Lines of Thoracic Surgery

Edited by Dr. Stefano Nazari

ISBN 978-953-307-915-8

Hard cover, 412 pages

Publisher InTech

Published online 03, February, 2012

Published in print edition February, 2012

Front Lines of Thoracic Surgery collects up-to-date contributions on some of the most debated topics in today's clinical practice of cardiac, aortic, and general thoracic surgery, and anesthesia as viewed by authors personally involved in their evolution. The strong and genuine enthusiasm of the authors was clearly perceptible in all their contributions and I'm sure that will further stimulate the reader to understand their messages. Moreover, the strict adhesion of the authors' original observations and findings to the evidence base proves that facts are the best guarantee of scientific value. This is not a standard textbook where the whole discipline is organically presented, but authors' contributions are simply listed in their pertaining subclasses of Thoracic Surgery. I'm sure that this original and very promising editorial format which has and free availability at its core further increases this book's value and it will be of interest to healthcare professionals and scientists dedicated to this field.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Michael S. Firstenberg and Jason Rousseau (2012). Post Myocardial Infarction Ventricular Septal Defect, Front Lines of Thoracic Surgery, Dr. Stefano Nazari (Ed.), ISBN: 978-953-307-915-8, InTech, Available from: <http://www.intechopen.com/books/front-lines-of-thoracic-surgery/post-myocardial-infarction-ventricular-septal-defect>

INTECH
open science | open minds

InTech Europe

University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China

Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

© 2012 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen