Chapter 11

# Management And Controversies of Post Myocardial Infarction Ventricular Septal Defects

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Additional information is available at the end of the chapter

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## 1. Introduction

Acute myocardial infarction (AMI), despite advances in health care delivery systems, education, and primary prevention still remains a significant problem. Fortunately, with these advances and early interventions, there has been a decline in the incidence of mechanical complications. Unfortunately, while becoming less common, when mechanical complications occur and despite advances and evolving techniques in the surgical management of these problems, morbidity and mortality remain high. Post-myocardial infarction ventricular septal rupture (PI-VSD) has challenged and intrigued 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 otherwise be doing well. In addition, while less common, some patients might not present until weeks, if not longer, after their AMI with symptoms prompting a work-up that might reveal a chronic PI-VSD. Early PI-VSDs tend to be catastrophic and can result in death. The pathology is also variable and complex, but common themes include:

1. acute right ventricular (RV) failure from a sudden increase in pressure, volume, and flow from left to right shunts,
2. pulmonary hypertension also from the acute increase in RV work and flow and
3. worsening cardiac output, often with manifestations of shock and end-organ damage, from acute left ventricular (LV) dysfunction and left-right shunting.

Definitive management remains surgical, however, controversies continue to exist regarding the timing of surgery, the role of concomitant coronary revascularization, and the evolving

role of percutaneous closure devices. Unfortunately, despite early repair and improvement in techniques and peri-operative management, the short and long-term outcomes remain less than ideal.

## 2. History

Post-myocardial infarction ventricular septal defects (PI-VSD) were described first at autopsy [30]. It was not until 1923 that the pre-mortem pathophysiology was understood [7]. [40], described the association with coronary artery disease and acute myocardial infarction [40]. The first report of a surgical repair came in 1956 when Denton Cooley described a patient 9 weeks after the initial diagnosis who underwent operative intervention [12]. With advances in the peri-operative and intra-operative management of the cardiovascular surgery patients there were reports of survival in what was previously felt to be an inherently fatal problem. Most of the successful operative cases occurred in patients who presented in congestive heart failure weeks after their initial acute event. Based upon these experiences, for many years it was the belief that operative management should be delayed as long as possible to allow for scaring of the necrotic myocardium to provide for a more stable repair. As experiences grew early repair was advocated, particularly in stable patients before hemodynamic deterioration and associated multi-organ failure.

## 3. Clinical Presentation

The incidence of PI-VSD has decreased dramatically over the years with advances in myocardial reperfusion and early revascularization strategies. Historically, up to 5% of AMI were associated with mechanical complications such free-wall ruptures, papillary muscle rupture, and PI-VSD [1]. With advanced in therapies that advocate early and aggressive attempts at reperfusion of the acute ischemic myocardial – such as thrombolytic therapy, early percutaneous interventions with coronary stenting (PCI), and, rarely, emergent coronary artery bypass surgery (CABG) – the overall presentation of mechanical complications, such as PI-VSD, has decreased significantly. Large multi-center studies evaluating the pathophysiologies of acute myocardial infarctions have shown a current incidence of approximately 0.2% of all AMI. In patients who present late or in whom there is a delay in therapy and there is a resulting increase in myocardial damage, this incidence increases up to 2%. Despite the low risk of developing a PI-VSD, it accounts for a disproportionally high mortality rate. Five percent of all early deaths after AMI are attributed directly to the complications of PI-VSD [36].

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

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Risk factors include gender, with men at a greater risk than women (3:2 ratio), increasing age, and current smoking history. In the GUSTO trial, the mean age of presentation with a PI-VSD was 62.5 years and ranged from 44 to 81 years [13].

## 4. Diagnosis

The diagnosis of a PI-VSD must be considered in any patient presenting with hypotension, cardiogenic shock, or respiratory failure, particularly in the setting of a patient who otherwise had been doing well, either during or after an AMI. 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 tamponade, or severe LV failure and pulmonary edema. The initial diagnosis must be suspected during initial investigations during a comprehensive work-up.

Patients often complain of recurrent chest pain. The characteristics of the pain are often different than their initial presentation and are typically related to the onset or recurrence of myocardial necrosis. Often a new systolic murmur will develop and it 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. Hemodynamic deterioration can be quick and there can be a rapid progression to cardiogenic shock.

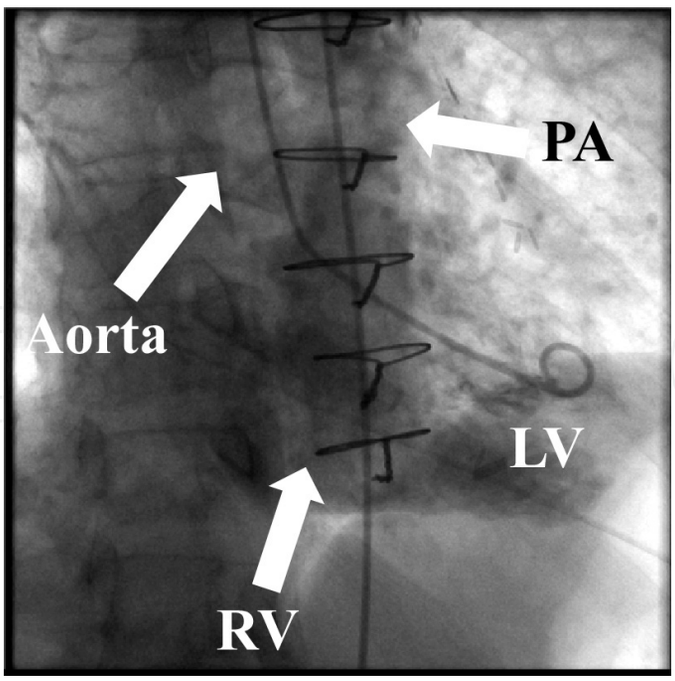


**Figure 1.** Representative cardiac catheterization in which contrast is injected during left ventriculography crosses the defect into the right ventricle. Contrast flowing into the pulmonary artery is then diagnostic for a ventricular septal defect.

With the acute clinical decompensation, a rapid evaluation of potential causes 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 can correlate with the size, and more importantly – the physiologic consequences, of the defect.

**4.1. Cardiac Catheterization:**

Without a doubt, the value of early cardiac catheterization and coronary angiography in the setting of acute myocardial ischemia is well established and standard of care. In patients whom the diagnosis of a PI-VSD is suspected this test can also be diagnostic. Because a septal defect is often with extensive and acute ischemia of a large territory of myocardium, it is not uncommon that the catheterization findings are different from patients with a history of chronic coronary artery disease in which compensatory development of septal collaterals have had time to develop. 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. The left anterior descending (LAD) artery is often the culprit vessel and this explains why anterior or apical septal defects are found in 60% of cases. Conversely, acute occlusions of a dominant right coronary or circumflex artery accounts for the remaining cases involving the posterior septum. Seven percent have concomitant double vessel disease, and 29% have triple vessel disease.

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 left to right shunting. Left ventricular contrast injections, although less likely to be performed in a deteriorating patient secondary to the concern that additional contrast might further injure already compromised renal function, can be diagnostic of a PI-VSD. Contrast injected into the LV will cross the defect (left-right shunt) and flow into the pulmonary arterial tree. This "pulmonary arteriogram" is characteristic for a VSD (Figure 1, see above)

Arguments against mandatory catheterization suggest that in a clinically deteriorating patient in whom the diagnosis is clear it only 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 a risk factor for a poor outcome [33]. Despite these theoretical 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 impaired coronary blood flow – particularly given the importance of optimal and complete revascularization. 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 (or repeat catheterization) is rarely encountered. However, as many of the patients develop septal defect several days (or weeks)

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after their initial acute coronary insult, it is hard to argue the need for repeat cardiac catheterization if the diagnosis is clear and the coronary anatomy is defined. Conversely, repeat catheterization might suggest an alternative, and potentially more likely, diagnosis such as acute stent thrombosis, coronary dissection, or disruption of an already unstable plaque.

### 4.2. Echocardiography:

Transthoracic echocardiography (TTE) remains the cornerstone of the non-invasive assessment of PI-VSD [28]. TTE is indicated in any patient who presents with acutely impaired ventricular function or in unexplained hemodynamic deterioration [9]. Echocardiography has the benefit of being able to assess both left and right ventricular function, the presence of co-existing and confounding valvular diseases – typically mitral and/or tricuspid regurgitation, and with color flow imaging it can be 100% specific and sensitive in diagnosing a PI-VSD. Despite the utility of transesophageal echocardiography in the acute assessment of an unstable 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.

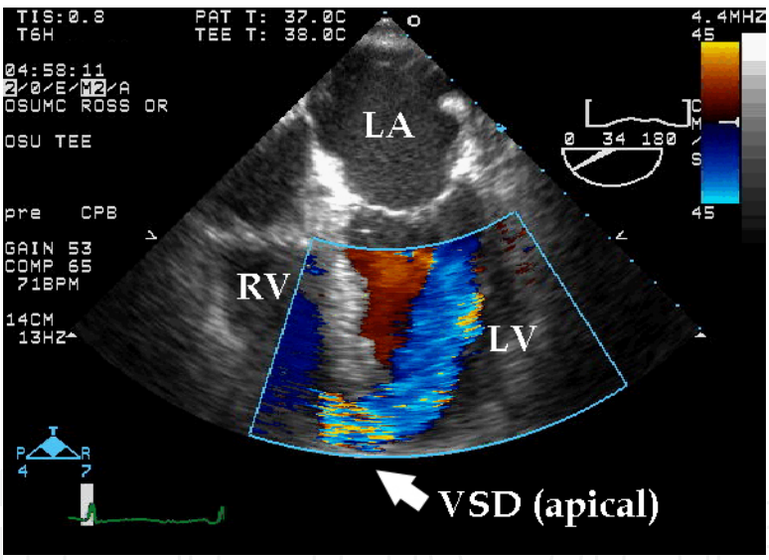


**Figure 2.** Transesophageal echo, 4-chamber view, demonstration an apical VSD with shunt from the left ventricle (LV) to the right ventricle (RV). The left atrium (LA) is also shown to illustrate the typical relationship of the defect to the mitral valve.

### 4.3. Cardiac Magnetic Resonance Imaging:

While the diagnosis is often made at the time of initial cardiac catheterization or echocardiography, occasionally a PI-VSD may be encountered as an incidental finding during other diagnostic imaging. While patients might be too hemodynamically unstable or the presence of an intra-aortic balloon pump might contraindicate cardiac 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 cardiac 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 experience describing the role of cardiac MRI in PI-VSDs, using concepts derived from the literature on congenital shunts and defects, cardiac MRI might be of value in assisting in defining the extent of the defect, the shunt fraction, right ventricular function, and other associated pathophysiology [17]. Cardiac MRI might be of additional value in situations of questionable catheterization or echocardiographic results or in the assessment of the post-operative patient when a residual shunt is suspected. Nevertheless, MRI is, in general, not considered a first-line diagnostic imaging tool.

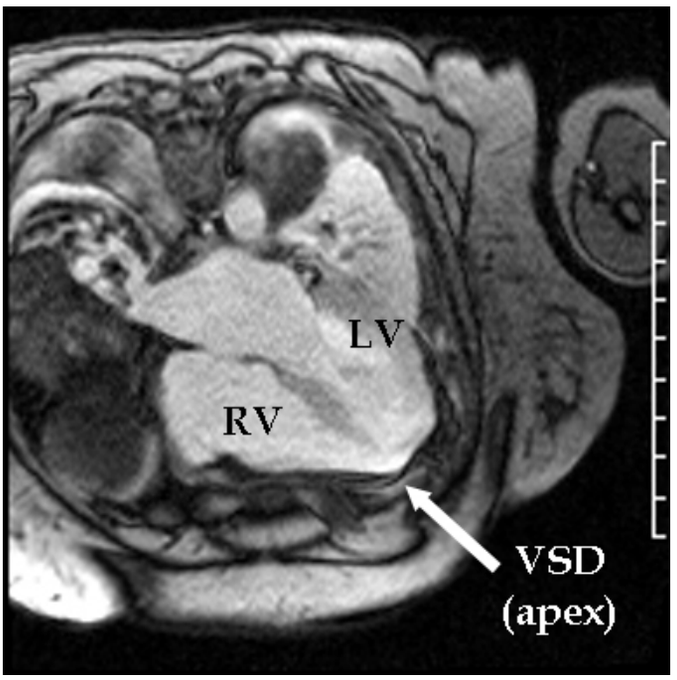


**Figure 3.** Cardiac MRI demonstrating an apical defect. Gated cine images indicated a left to right shunt in which quantitative assessment can be used to calculate the shunt fraction and size of the defect.

## 5. Pathophysiology

The pathophysiology reflects two different types of defects. The first, a simple rupture, is a direct through-and-through defect that is typically located anteriorly when associated with a LAD territory infarct. Alternatively, complex defects 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. Obviously, with unpredictable injury to the septum, there can be a combination of the 2 different pathologies. Multiple defects are found in 5-11% of cases and emphasized the need for a complex pre-operative and intra-operative assessment of all

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pathways to insure a complete repair [18] and the observation that most defects are probably larger than they initially appear. 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 and potentially the annular structures of the mitral valve. 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.

Interestingly, and clearly an area of further study, the pathologic consequences and outcomes of surgery of anterior and posterior defects are different in ways beyond what can be explained by the varying degree of shunting. 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 [15]. Particularly considering the acute pressure/volume overload and associated RV failure, it becomes understandable why posterior based defects are associated with 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 such as AMI. In general, 25% of patients with PI-VSDs die within the first 24 hours [6]. Death is most commonly related to pre-existing comorbidities and the potentially irreversible and severe heart failure that comes from not only the acute pump failure from the inciting infarct but also the significant acute left to right heart shunting that only 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 [31]. It is easy to appreciate that those patients who survive the first month may have inherently favorable variables 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 in these rare cases.

## 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 [38]. 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 (IABP) should be placed. Coronary augmentation will assist the ischemic and injured myocardium. More importantly, an IABP will unload the left ventricle and improve cardiac output, and end-organ perfusion. By decreasing afterload, there will also be an improvement in 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 finishing the pre-operative assessment.

While some advocate a strategy of delayed repair, this approach is rarely successful. The hypothesis of this management plan 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, in theory, but it is rare that patients remain stable or can be supported during this time period. While guidelines for delayed surgical management are lacking, this might be an option in those who are hemodynamically or 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" approach in those with minimal shunting and with strict attention to medical comorbidities and nutrition a period of close careful waiting may be clinically successful. This approach may also be used to justify waiting in patients who have other severe comorbidities precluding intervention and would, in theory, require optimization prior to surgery. Nevertheless, it is hard to argue that any other comorbidities would improve enough 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 also suggest that in these patients, unless early surgical repair is clearly contraindicated, that their physiologic reserve combined with a minimal pathophysiologic insult might predispose them to a good outcome regardless of whether an early or late repair is performed.

Of growing concern regarding the timing of surgery and the implications of peri-operative management is the use of potent, and often irreversible, anti-platelet inhibitors and/or anti-coagulants. The data and experience on operating on patients with some of these newer agents, the impact on the ability – or lack thereof – to achieve surgical hemostasis with such drugs is both limited and evolving. As many of these patients might have already been pre-treated with P2Y12 inhibitors such as clopidogrel, or the more potent agents such as prasugrel or ticagrelor, the impact on bleeding and the timing of surgery can be worrisome. Furthermore, other agents used to facilitate coronary interventions, such as Gp IIb/IIIa inhibitors such as eptifibatide, or direct thrombin inhibitors such as bivalirudin might require an appropriate 'wash-out' period. The risk for a massive transfusion (particularly with platelets) at the time of surgery with these agents still active cannot be understated. In fact, at patient who already might be considered physiologically high-risk might be considered inoperable in the setting of recent Dabigatran (Pradaxa) exposure due to the risk of catastrophic, irreversible, surgical hemorrhage. In the post-operative period, the decision to

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restart a P2Y12 inhibitor, if indicated (i.e. for recent coronary stents), clearly needs to be a balance between the risk of bleeding and the risk of a stent thrombosis or recurrent ischemia. Hopefully, newer agents, such as cangrelor, which is an IV, short-acting, reversible, anti-platelet agent might lead to protocols that can assist in bridging patients to such high-risk surgical interventions – such as an acute PI-VSD [2].

## 8. Operative Management

The initial surgical techniques for PI-VSDs followed a surgical approach similar to that for congenital ventricular septal defects. The approach was through a ventriculotomy in the right ventricular outflow track (RVOT) [12]. It was quickly realized that this approach had significant drawbacks. Firstly, in an already dysfunctional and acutely injured right ventricle, the outflow tract incision only further reduced residual, crucial RV function. As importantly, while suited for many common types congenital septal defects near the aortic valve, the RVOT incisions offered poor exposure of defects that tended to be much further down the septum towards the apex. Most importantly, since 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, extension of the defect, and clinical failure. Pioneering animal studies by [22] advocated an approach to the VSD thru the left ventricle in the region of the culprit vessel through infracted myocardium – specifically, anterior defects approached through the anterior wall while posterior defects through the inferior wall. These techniques addressed many of the deficiencies of a RVOT approach [22]. The benefits of these animal studies were subsequently validated clinically within several years [26][8]

### 8.1. Basic Principles and Considerations

After the patient arrives in the operative room, routine anesthesia is induced. 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. Since these patients have left-to-right shunting, it is of critical importance to avoid pharmacologic agents that cause pulmonary vasodilation. Such agents would worsen the shunt, increase 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 fatal. 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 unless clearly indicated by pre-operative studies, this is not necessary. Cold-blood antegrade and retrograde cardioplegia is delivered via conventional root and coronary sinus catheters. Topically cooling to further reduce the metabolic demands of the already compromised heart is also liberally used. This author routinely uses ice-slush wrapped in gauze to further cool the right ventricle to assist in reducing the temperature and assists further in myocardial protection – a key component in minimizing post-operative biventricular dysfunction. Active or passive systemic cooling is performed with an ideal temperature of 25º-28ºC to further assist end-organ protection. Regardless, the key concept is an appropriate and well thought out approach to myocardial 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. The "Sandwich Technique" approach has been described to minimize the tension and risk for leakage of the final repair. In this technique, a patch is positioned on the RV side and re-enforced with pledgets. The defect, much like a "pot-hole" in the road, is then filled a gelatin-resorcin-formalin (GRF) glue and then covered on the left ventricular side with a similar patch and pledgets. The proposed advantage of this technique is that the glue serves as a cement to re-enforce the repair, minimize tension on the sutures lines, attempts to preserve ventricular geometry, and most importantly, reduces the risk of leaking. Early [24] and midterm results [25], although in limited studies, support this adjuvant to traditional patch repair techniques as described below. Although the "sandwich technique" advocated a repair via a right ventricular incision, such an approach, for reasons already outlined, may not be desirable – nevertheless, experience with this technique is growing and this might be a reasonable approach in selected patients [4]. 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 peri-valvular tissues is involved, mitral valve replacement may be required. The choice of prosthesis is up to the surgeon, but given the 5-year limited survival of these patients in general and the post-operative challenges that might come from the need for anticoagulation with a mechanical valve, a tissue valve in these cases is a reasonable option regardless of the patient's age and co-morbidities.

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 defects involve the apical portion of the right ventricle, septum, and the left ventricle. As mentioned, such defects are typically the result of acute occlusion of the distal left anterior descending artery. Daggett and colleagues first described the technique of apical amputation and repair of the PI-VSD in 1970. The initial incision is created through the

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infracted tissue of the cardiac apex. The necrotic myocardium is then 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. While meticulous hemostasis is critical, too much tension on the suture repair can tear through the muscle and result in uncontrolled postoperative hemorrhage.


**Figure 4.** 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.

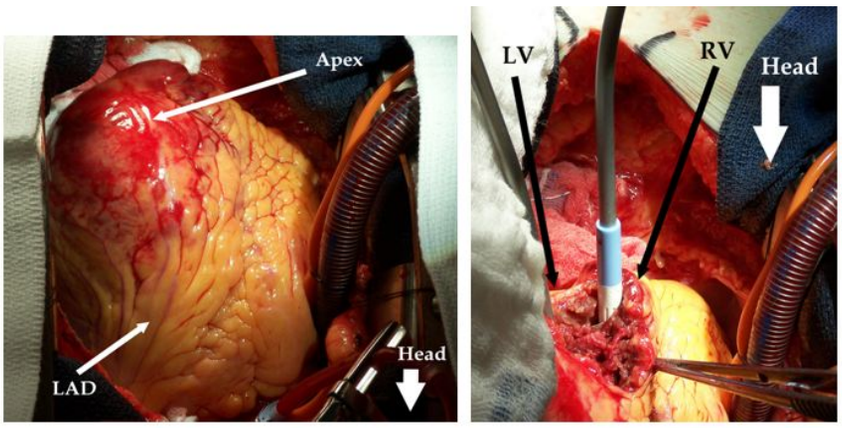


**Figure 5.** Left: Intra-operative view of the extensive apical infarction that has resulted in the echocardiographic findings demonstrated in Figure 2. The left anterior descending artery (LAD) is shown. Right: The same patient after opening and debridement of the infracted apex. The necrotic septum 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 defect involves the anterior septum as well as the anterior left ventricular free wall. This, as discussed earlier, is typically a result of acute infarct of the LAD territory. The initial approach is via an incision in the left ventricular myocardium parallel to the LAD and through the non-viable or ischemic tissue. The infracted 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. How-

ever, great care must be taken if the defect is noted to be tracking through the myocardium – a finding that might not be obvious. In general, the larger the patch the better - as a too small of a patch is more likely to pull through and dehisce.

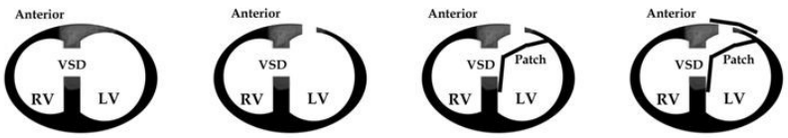


**Figure 6.** Picture representation of the various steps used to repair an anterior defect with the patch excluding the necrotic septum from the higher pressure left ventricular cavity. The incision is through the infracted muscle on the anterior wall, parallel to the left anterior descending artery.

Small defects can be plicated with a primary repair to the right ventricular free wall using interrupted, pledgeted suture as first suggested by Shumacker [42]. For anything other than very small (<1.0 cm) defects, most anterior septal infarcts will require repair with a patch. This is fashioned in a manner that will allow for a tension-free repair. Excess tension in the repair can lead to devastating consequences either with acutely life threatening bleeding or a delayed dehiscence and residual shunt.

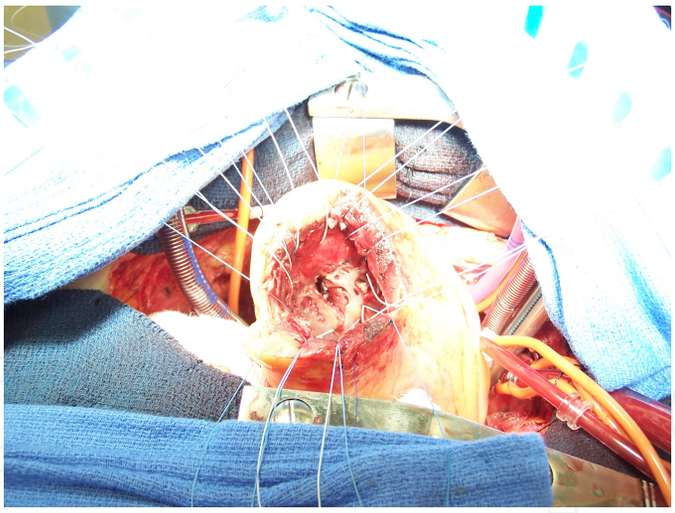

**Figure 7.** Intra-operative picture with the apex of the heart elevated (head and aortic cannula to the right). The incision is through the infarcted anterior wall. The septal defect is shown in the middle of the cavity with circumferential sutures around a wide margin. The sutured will then be placed through a pericardial patch to exclude the infracted septal muscle and defect from the left ventricular cavity.

Larger defects require the use of a prosthetic patch that is anchored to the posterior wall of the septum using interrupted, pledgeted Tycron or Proline suture to distribute the tension. The suture is passed from the RV through the LV so that the patch-septum interface lies in the left ventricle, as opposed to the RV. The anterior sutures are placed through the right

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ventricular free wall and tagged with hemostats (Figure 7). 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 similar to apical repairs. A second layer of running suture, often with a strip of felt to distribute the tension of the closure, is placed for reinforcement.

### 8.4. Posterior Defects

The posterior or inferior septal defect involves a transmural infarction of the myocardium in the posterior descending artery 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 immediate, and potentially fatal, consequences. Hence, posterior/inferior septal defects 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 which is critical for a durable repair. The papillary muscles are inspected. If the base is involved in the infarct resulting in ruptured papillary muscle, then mitral valve replacement is indicated. If a small posterior defect (<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.

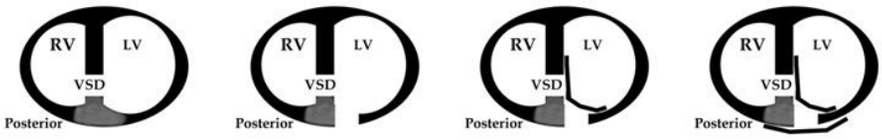


**Figure 8.** Similar to anterior repairs, the high pressure left ventricular cavity is isolated from the necrotic septum with a patch repair. The incision is along the distal right coronary along and parallel to the posterior descending artery through the infracted basal muscle.

As with anterior defects, most posterior 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 defect. 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.

### 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. Biologic glues can be liberally used for small suture leaks, but it should be avoided in more significant bleeding as this might suggest a less than stable closure.

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 [19].

Although the benefits of concomitant revascularization on long-term outcomes are debatable, complete coronary revascularization, if possible, is typically advocated [23]. 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 refute the benefits of revascularization in the setting of an already acutely and chronically ischemic myocardium.

## 9. Post-operation Management

The post-operative management of patients following successful repair should be similar to that of other high-risk cardiac surgery patients. However, there are several key principles that must be remembered. 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 common and there should be a low threshold for placement of an intra-aortic balloon pump (IABP), particularly if one was not placed pre-operatively. While, as discussed below, the use of an IABP is often

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associated with worse outcomes, the relationship to a poor outcome is the need for its use and the potential delay in initiating therapy rather than the therapy itself that influences the adverse outcome. Right heart failure is common and often these patients require considerable therapies directed specifically at assisting in right heart management. 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 [37]. 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 important as even transient elevations in blood pressure can result in disruptions in either the patch repair or the ventriculotomy closure suture line that might precipitate uncontrolled and fatal cardiac 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. Any post-operative coagulopathy must be aggressively corrected. Although recovery in these patients is unpredictable, it may be prolonged. A slow wean of inotropes may be required and there should be a low threshold for repeat and/or frequent echocardiographic evaluations in a patient who is not improving as anticipated. Repeat echocardiography might show a residual shunt or valvular dysfunction, more importantly may identify 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. Even though several large single center experiences and outcomes have been reported, most summarize years of experience and may not take into consideration the improvements in peri-operative management, surgical skills, and – probably most importantly – the clinical judgment necessary for the management of these critically ill patients.

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 treatment 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 [13]. All patients who presented in Class III or IV heart failure died.

Deja and colleagues reported their experience with 117 patients from the Glenfield General Hospital in England. In their series, there were 76 anterior defects and 34 posterior defects. The mean age was 65 ± 8 years and 43 of the 117 were females. One third of patients were in

cardiogenic shock at the time of presentation. The average time from AMI to the development (or diagnosis) of a PI-VSD was 6 days. The time interval from the point of diagnosis to surgical intervention was 9 days. The overall mortality was 37% and this does not include the 6.4% intra-operative mortality. Forty percent had evidence of a residual left-right shunt with 13 patients undergoing early re-operation. In patients undergoing re-operative surgery, mortality was 30%. Table 1 summarizes their overall results. The predictors of post-operative mortality included:

1. shock at time of surgery,
2. clinical deterioration while awaiting surgery,
3. need for concomitant CABG, and
4. pre-operative renal failure (as a marker for shock and organ failure).

The obvious criticism of this report is the long interval from the time of diagnosis to the time of surgery as earlier intervention, as advocated, might have resulted in less end-organ damage in an already compromised patient (Deja MA, 2002).

| | |
|---|---|
| Overall surgical mortality (%) | 37% |
| Intra-operative mortality (%) | 6.4 % |
| Need for extra-corporeal membrane oxygenation (ECMO) | 2 % |
| Re-exploration/bleeding | 5 % |
| Average ICU Stay | 4.8 days |
| Average Ventilator Time | 40 hours |
| Major inotropic support | 90 % |
| Use of Intra-aortic balloon pump | 75 % |
| Tracheostomy | 5% |
| Continuous renal replacement | 16 % |
| 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 this uncommon problem. In a report from Sweden, outlining the national experience with 189 patients, several factors were found to be predictive of favorable vs. unfavorable short (< 30 days) and long-term (> 30 days) outcomes (Tables 2 and 3) [27].

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| **Favorable Predictors** |
|---|
| Short time from MI to Diagnosis |
| Short time from Diagnosis to Operating Room |
| Short time from MI to Operating Room |
| Pre-Op catheterization |
| Anterior rupture |
| **Unfavorable Predictors** |
| IABP |
| Stroke/Coma |
| Renal failure |
| Re-Op for bleeding |
| **No effect on outcome** |
| Age |
| Gender |
| Pre-Op IABP |
| Pre-Op Lytic therapy |
| 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, IABP: Intra-aortic balloon pump, MI: Myocardial infarction.

| **Favorable Predictors** |
|---|
| Younger age |
| **Unfavorable Predictors** |
| Pre-Op IABP |
| Pre-Op catheterization |
| Need for CABG |
| Renal Failure requiring dialysis |
| Residual shunt |
| **No effect on outcome** |
| Anterior rupture |
| Time from MI to Operating Room |
| Post-Op stroke |
| Pre-Op Lytic therapy |
| Post-Op IABP |
| Re-Op for bleeding |

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

The Italians also presented their experience with the outcomes of 58 patients treated between 1992 and 2000 [10]. The mean age was 73 years. Thirty-six percent presented in acute renal failure, 33% were in atrial fibrillation, and 22% were insulin dependent diabetics. Most (57%) were in NYHA Class IV heart failure and 41% were in cardiogenic shock. Intra-aortic balloon pumps were used in only 20% of patients. Sixty percent had associated 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).

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

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

In a series of 50 patients, operated on over 19 years (1983-2002), Mantovani et al reported their single center experience. The mean age of 66 ± 9 years (range: 45-81) who presented with either anterior (n=30, 60%) or posterior (n=20, 40%) PI-VSDs. Most patients developed their defects within the first week (76%) with an average of 4 days post-AMI. Only 2 patients presented after 2 days. Cardiac catheterization was performed in 98% of patients. Coronary angiography revealed single vessel disease in 51% of patients, double vessel disease in 35%, and 14% had triple vessel disease. Pre-operative IABP was used in 56% of patients and 74% underwent surgery within 2 days of diagnosis of a PI-VSD. Operative mortality (within 30 days) was 36% with 6 operative deaths. Posterior defects were associated with 50% mortality versus 25% for anterior. Other univariate risk factors for early death included:

cross-clamp time >100 minutes (p=0.035);

emergent surgery (p=0.02); and delayed surgical intervention (>3 days post diagnosis, p=0.0055).

Interestingly, in their experience factors not associated with operative/post-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

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emergent surgery (odds ration: 10.23) and a delayed treatment (OR: 4.03) were the only predictors of early post-operative or operative mortality. Long-term survival was 76.5 ± 7.8 and 56.1 ± 11.5% at 5 and 10 years suggesting a reasonable outcome for this catastrophic problem. No significant predictors of long-term survival were found in their analysis.

However, patients with residual myocardium at risk from incomplete revascularization tended to have a worse long-term prognosis [32].

A review of the Society of Thoracic Surgeons National Database has also provided some valuable insight into the management and outcomes of the problem of PI-VSD. Arnaoutakis retrospectively reviewed all adult patients who underwent surgical repair of a PI-VSD between 1999 and 2010 [3]. In this review, there were 2,876 patients – reflecting probably the largest series reviewed. In this cohort, 56.5% were males, almost half (49.7%) underwent emergent surgical intervention, and 65% had a pre-operative IABP. One third (33%) had undergone a previous percutaneous coronary intervention and 7.5% had undergone a previous CABG. The annual incidence of PI-VSD was relatively constant and ranged from 232-297 cases/year and implies that few reporting centers have a significant experience with this complex problem - of the 666 centers reporting data to the database the experience ranged from 0.09 to 3.7 cases/center/year. Consistent with previous reports, the overall operatively mortality was 42.9%. They found an inverse relationship between the timing of surgery and survival. Patients operated on within 6 hours of presentation had a 54% mortality. Survival was ~50% for the patients operated on between 1 and 7 days. However, of the 513 patients operated on >21 days after presentation had a <20% mortality – again, suggesting that those who present late and remain hemodynamically stable have a better long-term prognosis. Furthermore, when classified as elective, survival was 87% versus 20% for salvage operations. Refractory cardiac failure was the most common cause of death post-operatively. Clearly, maintaining hemodynamic and physiologic stability is key to a good outcome. Table 5 lists the predictors of a good outcome.

| Favorable | Unfavorable |
|---|---|
| **FAVORABLE FACTORS** | **UNFAVORABLE FACTORS** |
| Male gender | Older age |
| Hypertension | Chronic kidney disease |
| Smoking | Need for pre-operative IABP |
| Chronic lung disease | Pre-operative shock |
| Pre-operative NYHA Class IV | Previous CABG |
| Pre-operative beta-blocker | Triple vessel CAD |
| Pre-operative lipid agent | Lower Ejection Fraction |
| Elective repair | Salvage surgery |
| Short bypass time | Longer aortic cross-clamp time |

**Table 5.** Adopted from Society for Thoracic Surgeons National Registry Database on favorable vs. unfavorable predictors of survival after repair of PI-VSD in 2,876 patients.

In a multi-variate analysis of the clinical characteristics, the following variables were found to be predictors of post-operative death:

1. Advancing age (>65 years/old)
2. Lower Ejection Fraction
3. Female (vs. male)
4. Pre-operative cardiogenic shock
5. Pre-operative need for intra-aortic balloon pump
6. Peripheral vascular disease
7. Percutaneous coronary intervention within 6 hours of surgery
8. Re-do cardiac surgery
9. Emergent/Salvage surgery
10. Pre-operative dialysis
11. Pre-operative mitral regurgitation

Of all of the variables, the strongest predictor of a poor outcome was pre-operative renal failure requiring dialysis. Interestingly, favorable variables included a history of hypertension, congestive heart failure, and need for concomitant CABG. The authors emphasized patients, due to their physiologic status, who required earlier intervention tended to have worse outcome. Obviously, the controversies regarding the balance between the timing of intervention and clinical optimization continue.

Unfortunately, there is little data reporting long-term survival. In their report of 68 patients undergoing surgery for PI-VSD between 1988 and 2007, Fukushima and colleagues from Brisbane, Australia provide some valuable insight and predictors of long-term outcomes [21]. In their report, 85% of patients underwent urgent surgery within 48 hours of diagnosis, 71% had concomitant CABG, and 30-day mortality was 35%. The mean follow-up was 9.2 years. Overall short-time outcomes and predictors of survival were similar to previous reports (as discussed above). The actuarial survival at 1 year was 67%, 63% at five years, 51% at 10 years, and 36% at 15 years. However, freedom from main adverse coronary events of the survivors was 91%, 61%, 40%, and 19% at 30 days, 1 year, 5 years, and 15 years respectively. At 5 years, freedom from congestive heart failure was 70% and 85% for ventricular arrhythmias – while at 10 years, 54% were free of heart failure and 71% from arrhythmias. For the cumulative survival analysis, there were 43 patients alive a 1 year, 34 at 5 years, 22 at 10 years, and 6 at 15 years.

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## 11. Controversial Topics

### 11.1. Percutaneous Closure devices

Successful application of less invasive non-surgical options and closure devices in children with congenital VSDs has prompted enthusiasm for the use of similar closure devices in patients with PI-VSDs. 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 [41].

However, because of the appeal of a less-invasive, non-surgical, option for these critically ill patients, investigators continue to try and define the role of septal occluder devices in patients with PI-VSDs. Attia recently reviewed the literature of such devices [5]. Thirty manuscripts were reviewed, but only 5 studies, consisting of approximately 100 patients, were felt to provide some insight into a "best practice" recommendation – despite numerous case reports. The general recommendations were that 1) surgical management still should be considered the 'gold standard' for patients with PI-VSDs but occluder devices might have a role in small defects (< 15 mm diameter) and in patients who present late (> 3.5 weeks after the index event). Attia also suggested a potential role in attempting to minimize a significant shunt in patients who are too ill to survive surgery as temporizing and potential means of stabilizing a patient prior to urgent surgical intervention.

While conceptually promising, the complex nature of acute defects as compared to congenital defects has tempered some of the early enthusiasm as early experiences were discouraging and improvements in outcomes were not observed [35]. Difficulties in covering not only the actual defect, but also the residual necrotic myocardium predisposed to early recurrence. Challenges remain in the technology because of problems positioning the devices and adequately covering potentially complex defects.

### 11.2. Mechanical Support

Despite advances in surgical and post-operative management, operative mortality is still high and depending on clinical presentation can vary between 10-60%[14]. 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 therapy may be indicated for those with irreversible ventricular failure. Since early acute co-morbidities and associated cardiogenic shock predict poor outcomes, there is some evidence and support for pre-operative biventricular mechanical support to allow for clinical optimization and stabilization as a bridge to definitive repair [11].

In cases in which there is extensive ventricular infarction and acute heart 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 as a bridge

to recovery before exposing the compromized left ventricle to systemic pressures and contractile function [19].

Right ventricular mechanical 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 guide decision making other than clinical judgment and center experience with management of acute post-cardiotomy right heart failure.

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 [39]. If residual shunts are significant then biventricular support, either with long-term ventricular assist devices or extra-corporeal membrane oxygenation, may allow for a period of recovery and stabilization prior to an attempted repair in an otherwise very high-risk surgical patient [44]. The decision to intervene surgically on residual shunts, because of the extremely high operative mortality as discussed above, must clearly be made in the context of the overall clinical condition of the patient. Small defects can be managed medically and can be surprisingly well tolerated physiologically for years.

The need for mechanical support, while attractive in unstable post-operative patients, is also not without substantial risks. Often there is need for aggressive anti-coagulation, multiple surgical procedures (i.e. device change-outs, explants, re-operation for bleeding, etc), and overall patient recovery is more difficult when tethered to external VAD controllers. In addition, the risks for infectious complications with long-term support are considerable.

A total artificial heart, by definition, eliminates native cardiac recovery and mandates cardiac transplantation, nevertheless, it may be an option with appropriate resources and experience in highly selected patient with few other comorbidities and in general, is probably a poor idea.

### 11.3. Residual/Recurrent Defects

Residual shunts are found in up to 25% of patients after definitive repair [43]. 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 [27] and surgery is reserved for patients in heart failure failing medical management or those with large shunts (Qp:Qs>2.0) [34]. Because of the high operative mortality with repairing residual or recurrent shunts there has been interest, but limited success, with percutaneous closure devices [41]. Nevertheless, the role of percutaneous closure and the ideal devices are undefined [35] and probably best reserved for use in those centers with extensive experience in the closure of congenital VSDs.

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## 12. Conclusions

Ventricular septal defects after acute myocardial infarction are rare events. With modern reperfusion strategies, septal defects occur in up to 0.02% of acute myocardial infarctions. Despite advances and experiences in the management of these complex patients, operative mortality still approaches 50% with major risks including cardiogenic shock, renal failure, right and/or left ventricular failure, size of the defect with degree of shunting, posterior/inferior locations, and residual post-repair shunting. While some patients may present late or benefit from watchful waiting and 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 while avoiding additional injury to the already compromised left and right ventricles. Post-operative management is typically challenging considering the inherent pre-operative biventricular dysfunction and often encountered end-organ dysfunction. 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.

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