Percutaneous Closure of Post-Infarction Ventricular Septal Defect:  
In-Hospital Outcomes and Long-Term Follow-Up of UK Experience

Running title: Calvert et al.; Percutaneous post-infarct VSD closure

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Abstract

**Background**—Post-infarction ventricular septal defect (PIVSD) carries a grim prognosis. Surgical repair offers reasonable outcomes in patients who survive a healing phase. Percutaneous device implantation represents a potentially attractive early alternative.

**Methods and Results**—PIVSD closure was attempted in 53 patients from 11 centers (1997-2012) (age 72±11 years; 42% female). 19% had previous surgical closure. Myocardial infarction (MI) was anterior (66%) or inferior (34%). Time from MI to closure procedure was 13 [Q1-Q3: 5-54] days. Devices were successfully implanted in 89% of patients. Major immediate complications included procedural death (3.8%) and emergency cardiac surgery (7.5%). Immediate shunt reduction was graded as complete (22%), partial (63%) or none (16%). Median length of stay post-procedure was 5.0 [2.0-9.0] days. 58% survived to discharge and were followed up for 395 [63-1522] days, during which time four further patients died (7.5%). Factors associated with death following PIVSD closure included: age (HR=1.04, p=0.039), female sex (HR=2.33, p=0.043), NYHA class IV (HR=4.42, p=0.002), cardiogenic shock (HR=3.75, p=0.003), creatinine (HR=1.007, p=0.003), defect size (HR=1.09, p=0.026), inotropes (HR=4.18, p=0.005) and absence of revascularization therapy for presenting MI (HR=3.28, p=0.009). Prior surgical closure (HR=0.12, p=0.040) and immediate shunt reduction (HR=0.49, p=0.037) were associated with survival.

**Conclusions**—Percutaneous closure of PIVSD is a reasonably effective treatment for these extremely high-risk patients. Mortality remains high but patients who survive to discharge do well in the longer term.

**Key words:** postmyocardial infarction ventricular septal defect, percutaneous closure, myocardial infarction, death, Amplatzer device
Introduction

Ventricular septal rupture after myocardial infarction carries a grim prognosis. Survival to one month without intervention is 6%. Surgical repair of the ventricular septum has been the mainstay of structural management, but despite current guidelines, which recommend immediate surgical repair, surgical preference is often to allow initial healing for at least two weeks. This introduces a significant selection bias into surgical series, artificially inflating survival rates. The advent of the Amplatzer™ family of ventricular septal defect closure devices offers a potentially attractive alternative to surgical repair with multiple device implants possible if required (Figure 1A). A few series of selected cases exist, with good results reported but there are no large series with early as well as delayed intervention. The largest series of percutaneous PIVSD closure reported on 40 closure procedures in 30 patients from a single center. However, the devices used in this series (CardioSEAL and STARFlex; NMT Medical, Boston, MA) are no longer commercially available. We sought to categorize and analyze all post-infarction VSD cases where percutaneous intervention had been attempted in the UK.

Methods

Device implantation for post-infarction VSD (PIVSD) has been attempted in the UK since 1997. Units report numbers of cases undertaken to the British Cardiovascular Intervention Society (BCIS) and is published annually at www.bcis.org.uk. All centers (n=14) where PIVSD activity has been reported in the UK were contacted to participate in this retrospective study and all agreed to participate but only 11 provided data. An electronic case record form was circulated to all centres, and data were acquired from medical and electronic records. Vital status was obtained via the Office of National Statistics.
Data were collected regarding patient demographics, clinical features, pre-procedure clinical condition, echocardiographic features, procedural characteristics, procedural complications, in hospital outcomes and vital status. The only absolute exclusion criterion for attempted percutaneous closure was a defect measuring greater than 24mm in diameter (the largest available device).

Cardiogenic shock was defined according to the SHOCK trial. Coronary artery disease was defined as >70% stenosis in one of the three major epicardial coronary arteries.

**Procedure**

The majority of procedures in the UK were done by one of two operators (JdG, DHS) on a visiting basis. Procedures were usually done under general anesthesia with transesophageal echo (TEE) imaging (Figure 2). Once femoral arterial access was gained, the VSD was crossed left-to-right in the majority of cases and a wire passed into the pulmonary artery, where it was snared from the venous circulation (either internal jugular or femoral) and an arteriovenous rail was formed. Some operators preferred to cross from the right to left ventricle with the wire snared in the aorta (Figure 3B) but given the trabeculated nature of the RV and the often serpiginous nature of the VSD, this frequently resulted in difficulties in manipulating the wire into the VSD itself. The device was sized using TEE to measure the dimensions of the defect both on two dimensions and on color flow mapping. The PIVSDs were often complex in shape and where available, three dimensional TEE was used to better understand the defect. In some cases a sizing balloon was used to determine the correct device size to implant, but this risked significant enlargement of the defect and where this was not used the device was oversized compared to the TEE measurements, the amount depending on the anatomy of the defect and the firmness of the rims. The Amplatzer™ PIVSD device (SJM, Plymouth, MN) is available in larger sizes than the
Amplatzer™ muscular VSD device (maximum waist diameter 24mm vs 18mm respectively) and also has a thicker waist (10mm vs 7mm) and therefore was more suited to larger PIVSD or hypertrophied ventricular septa. The device was then delivered via a TorqueVue™ sheath (SJM, Plymouth, MN) to the interventricular septum and the device was positioned straddling the defect (Figure 3). After device release the degree of immediate shunt reduction was assessed angiographically and echocardiographically. In some cases multiple devices were required in order to gain complete closure. Whether a second device was implanted depended on the size and shape of the residual defect and also whether there was a rim present. Where no rim was present (between the septum and the free wall), it was technically more challenging to appropriately size and place the device securely. In such circumstance the device was oversized in an attempted to splay the discs of the device against the free wall in order to gain stability and a good seal against the wall, although this was often difficult to achieve. The size and type of additional devices deployed depended on these factors as well as the strength of the surrounding tissue.

Procedural complications were recorded, including device embolization, cardiac tamponade, stroke, and procedural death.

Statistical methods

Statistical analysis was performed using SPSS v19.0 (SPSS Inc. Chicago, IL). Only first attempts at percutaneous PIVSD closure were included in the analysis. Normally distributed data are presented as mean ± standard deviation and non-Gaussian data presented as median [first and third quartile (Q1-Q3)]. Categorical data are presented as frequency (percentage). Clinical and procedural parameters were tested for an association with death during follow-up using univariable Cox proportional hazard regression.
Results

Demographics

Between December 1997 and January 2012, 58 attempted PIVSD closure procedures were reported in 53 patients between 11 centers in the UK. In five patients the percutaneous closure attempt failed and they underwent repeated percutaneous closure attempts. These five repeated percutaneous closures attempts were excluded from the analysis for statistical reasons. The median number of procedures performed per center was 5 [Q1-Q3: 1-8], with one center performing 14 procedures. Only first attempts at percutaneous PIVSD closure were included in the analysis (n=53). 10 (19%) patients had previously undergone attempted surgical closure. In these patients the reason for percutaneous closure was either patch dehiscence or failure of closure of separate PIVSDs. The mean patient age was 72±11 years and 22 (42%) were female. Time from myocardial infarction (MI) to closure was 13 [5-54] days and was not associated with late mortality (HR = 0.99 [0.98-1.001], p = 0.077). The territory of the infarction was anterior in 35 patients (66%) and inferior in 18 patients (34%) (Table 1). The defect site was evenly distributed between anterior, apical and inferior positions (Table 2). Devices were successfully implanted in 47 patients (89%) (Table 3). Reasons for failure to successfully deploy a device included: inability to cross the PIVSD with the wire (n=1), inability to cross the defect with the delivery sheath (n=1), a diffuse defect unsuitable for percutaneous closure (n=2) and a defect too large to retain the largest device available (n=2). The clinical profiles of patients at presentation are displayed in Table 1.

Procedural factors

Multiple devices were successfully placed in three out of seven patients in whom it was attempted. Failure to deliver multiple devices was due to failure to cross one or more defects
(n=2) and unstable device position (n=2). Immediate complete or partial shunt reduction was seen in 11 (22%) and 32 (63%) patients respectively (Table 3). No shunt reduction occurred in 8 (16%) patients.

**Complications**

Major immediate complications included 2 deaths (3.8%) and need for emergency cardiac surgery in 4 patients (7.5%). The need for surgery was as a result of device embolization in 2 patients (3.8%). Blood transfusion was required in 4 patients (7.5%). Six patients went onto have a surgical repair after attempted percutaneous closure due to hemolysis, device embolization and failure of percutaneous closure attempt.

**Follow-up**

18 (34%) of patients died before discharge with 31 (58%) of patients surviving to discharge. Long-term follow-up among patients who survived to hospital discharge was for 395 [Q1-Q3: 63-1522] days. During this time only an additional four patients died.

**Factors associated with long-term death**

Factors associated with death on long-term follow up included: age, female sex, NYHA class IV, cardiogenic shock, creatinine, size of defect, absence of revascularization therapy for presenting MI and use of inotropes (Figures 4 & 5). Prior surgical closure of the VSD and immediate reduction in shunt were associated with survival (Figures 4 & 5). Kaplan-Meier cumulative mortality curves are displayed in Figures 6 and 7.

**Discussion**

This series of unselected cases undergoing percutaneous VSD repair following post-infarction septal rupture shows that overall the outlook remains poor for these patients. Amongst those who
have a successful procedure, in hospital mortality remains high due to the underlying condition. However, those who survive to hospital discharge have a good long-term outlook (Figure 6B).

Post-myocardial infarction ventricular septal rupture in the UK

The UK Myocardial Ischaemia National Audit Project (MINAP) reported 32439 ST-elevation myocardial infarctions (STEMI) for the year April 2011 to March 2012. The incidence of PIVSD is 0.2% in the era of reperfusion.1 Assuming similar STEMI rates, for the 14 years under consideration, therefore the overall number of PIVSD cases would be expected to be 908. During this same 14 year period, 743 patients underwent surgical repair of PIVSD15 and 53 underwent percutaneous repair, therefore there may be some selection bias in the cases presented in this series. Our data demonstrate a 59% discharge survival among these relatively selected patients having percutaneous treatment. Survival from surgical closure series ranges between 20-87% depending on case selection 5, 16, 17.

Can we predict mortality in PIVSD?

Parameters reflecting the critical clinical condition of these patients were associated with long-term mortality (Figures 4 & 5). This is perhaps to be expected, given that although 18 patients (34%) died during the index admission, once patients achieved discharge, only a further 4 patients (7.5%) died in the follow up period.

Our data suggest that complete closure of the defect is a good determinant of success. Unfortunately the Amplatzer™ VSD occluder is semi-permeable at implantation and may take a number of days to completely occlude with organized thrombus (Figure 1B).18 While this is acceptable and even useful in the elective setting, these highly unstable patients cannot necessarily tolerate persistent shunting following closure, even for a few days. Unfortunately, because of the rarity of the condition, there is little financial imperative to produce devices more
suited to septal rupture. The concept of reducing the shunt as a bridge to surgery is seldom effective, though it has been reported. Expert consensus suggests that if the shunt is not reduced by at least two thirds, the patient is most unlikely to survive either to surgery or to discharge, and our data would support this consensus.

Many patients (47%) did not receive any form of reperfusion therapy, usually due to delayed presentation, and consequently were more likely to die (HR = 3.28, p = 0.009). Reperfusion of the infarct-related vessel may be of benefit in establishing blood flow to areas of watershed ischemia. This may render defect margins more viable and therefore reduce risk of defect extension after device implantation. However, contrast nephropathy is a major concern in these patients and any benefit in revascularization remains hypothetical. Although an appropriately designed trial randomizing patients to reperfusion or not would be required to directly address this question, such a trial is unlikely ever to be conducted.

A number of markers of a critical clinical condition were associated with long-term mortality on univariable analysis. However, this study was not adequately powered to determine which of these factors (if any) may help to predict adverse outcomes. A larger study with appropriate statistical design would be required to provide more insight into this.

Myocardial infarction to procedure time

It is clear from the published surgical series that operative mortality is reduced by delaying surgery. However, this surgical “selection of the fittest” is naturally at the expense of the majority who do not survive long enough on medical therapy. The question is whether all-comers survival can be improved by early closure? Although our series cannot directly address this question, our data suggest that patients with PIVSD treated with percutaneous device closure relatively early do well, especially if they survive to discharge. Although increased time to
closure was not associated with reduced death, there was a non-significant trend in this direction (HR = 0.99 [0.98-1.001] p=0.077).

The technical aspects of percutaneous PIVSD closure

From the technical point of view, percutaneous PIVSD closure is a demanding procedure, requiring expertise and collaboration between interventionists, anesthetists and imaging specialists. Factors that confound early surgical closure are also important for percutaneous closure, in particular, lack of firm tissue on which to seat the device. As a consequence, patch dehiscence is a common reason for the need for percutaneous closure in those with previous surgical correction of PIVSD. The partially dehisced patch can increase the technical difficulty of the procedure as the wire can get trapped in blind-ending pockets created by the dehisced patch. Furthermore, the fact that PIVSDs are often serpiginous or multiple can make effective device closure difficult. Balloon “sizing” of the VSD can be useful to confirm that the wire rail has passed through the major part of the defect but is no longer widely used due to excellent imaging modalities and the risk of balloon enlargement of the defect. Often, it was difficult to accurately predict the most appropriate size of device and a “trial and error” approach was frequently necessary. The rims were usually friable and hence, if doubt existed, larger devices were selected. The serpiginous and complex nature of the defects meant that the devices were sometimes unable to conform to their intended shape (supplemental figure 1). However, the measure of a successful closure was shunt reduction without embolization alone and not cosmesis. Our experience suggests that arterio-venous loops are invaluable as are braided guiding catheters to prevent kinking. In the five patients were an arterio-venous loop was not fashioned, the operator was able to negotiate the delivery catheter sufficiently far into the ventricle to permit delivery of the device.
Patients are usually fragile and manipulation of the heart, systemic hypotension and contrast load contribute to renal dysfunction, hypothermia, acidosis and hypoperfusion. Patients who survive the procedure remain at risk of subsequent demise due to extended rupture, free wall rupture, renal failure, inflammation, sepsis, vascular access complications and multi-organ failure.

Our data confirm that the overall outlook for post-infarction VSD is poor, but that selected cases can be successfully treated. In the SHOCK and GUSTO trials, mortality in patients with PIVSD and cardiogenic shock was 87-100%. 1,13

Existing literature on PIVSD closure

Series on post-infarction septal closure are few. Thiele et al reported 29 cases in a single centre over a six year period.21 Their “warts and all” publication exposes the limitations of the percutaneous approach in this setting. Closure was attempted 1-3 days after diagnosis. 86% of patients had a technical success of device implantation, but 17% died in the catheter lab, 41% had major complications and survival to 30 days was only 35%. This study demonstrated that technical success in implanting a device is only half the battle. Many patients still die early due to complications of the disease process itself. Our data corroborate this, with 89% device implantation success but only 59% survival to discharge.

The largest series prior to the present analysis was a single center series of 40 closures in 30 patients over a 20 year period.12 60% of these patients were being treated for residual leaks following surgery, and these patients seem to form a lower risk cohort, judging both by their own exemplary mortality rate of 23% and from our own data where percutaneous closure of VSD after a prior surgical closure was associated with reduced long-term mortality (HR=0.12 [0.02-0.91] p=0.040). Like our cohort, there was an extensive usage of an externalised guide-wire
however, unlike our cohort, this was often a veno-venous loop rather than an arterio-venous loop. Although the parameters tested in the present analysis and that by Assenza et al differ, both investigators found similar trends, that is there is a positive relationship between mortality and larger defects and also between mortality and a more critical clinical state.

Holzer et al described the US experience with the procedure in 18 patients from different centres over a three year period, in the majority of whom the procedure was attempted more than two weeks after the infarction. Procedural success was 16/18 and 30-day survival was 72%. This clearly demonstrates the statistical value of delayed intervention but at a cost to overall survival.

A large surgical series (n=2876) from the Society of Thoracic Surgeons National Database has given some important insight into this condition. Overall operative death was 42.9%. Multivariable logistic regression modelling identified age, female sex, shock, pre-operative aortic counterpulsation balloon, redo surgery, emergency status, pre-operative dialysis and mitral insufficiency as independent predictors of operative mortality. With the exception of prior surgical closure, these findings are broadly in agreement with the findings in the present analysis.

**Study limitations**

Although this is the largest series of percutaneous PIVSD closure, it is still relatively small. This, combined with our study’s retrospective nature, limits the conclusions that can be drawn. This is a registry of patients who actually underwent attempted percutaneous closure of PIVSD. As such it does not include those patients who were considered for, but did not undergo, attempted percutaneous closure of PIVSD (i.e. those who ended up with conservative or surgical management). Lastly, it is possible that our report has not captured all cases of percutaneous VSD repair in the UK as cases were not submitted on a compulsory basis.
Conclusions

Ventricular septal rupture is a mortal complication of myocardial infarction. Our data demonstrate that in selected patients, device closure is possible. However, in-hospital mortality is high, even after apparently successful procedures. We believe that consideration should be given to early attempted percutaneous closure in patients presenting with this calamitous condition. Patients who do survive to discharge have excellent outcomes on long-term follow-up.

Acknowledgments: Thanks to Dr Simon Bond MA PhD (Cambridge Clinical Trials Unit and MRC Biostatistics Unit) for statistical advice.

Conflict of Interest Disclosures: DHS and JdG are proctors for St Jude AGA medical

References:


15. NICOR. The national adult cardiac audit registry; national institute of cardiovascular


Table 1. Clinical parameters.

<table>
<thead>
<tr>
<th>Clinical parameters</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>72.2 (10.8)</td>
</tr>
<tr>
<td>Female sex</td>
<td>22 (42%)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>169.7 (8.6)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>74.5 (12.8)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>11 (21%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>36 (68%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>15 (28%)</td>
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<tr>
<td>Hypercholesterolemia</td>
<td>27 (51%)</td>
</tr>
<tr>
<td>Initial MI treatment:</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>26 (49%)</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>10 (19%)</td>
</tr>
<tr>
<td>PCI</td>
<td>15 (28%)</td>
</tr>
<tr>
<td>CABG</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Rescue PCI</td>
<td>3 (6%)</td>
</tr>
<tr>
<td>PCI of IRA (any stage)</td>
<td>15 (28%)</td>
</tr>
<tr>
<td>Previous surgical closure</td>
<td>10 (19%)</td>
</tr>
<tr>
<td>Infarct territory:</td>
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<tr>
<td>Inferior</td>
<td>35 (66%)</td>
</tr>
<tr>
<td>Anterior</td>
<td>18 (34%)</td>
</tr>
<tr>
<td>No vessels with CAD:</td>
<td></td>
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<tr>
<td>1VD</td>
<td>31 (58%)</td>
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<tr>
<td>2VD</td>
<td>17 (32%)</td>
</tr>
<tr>
<td>3VD</td>
<td>5 (9%)</td>
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<td>NHYA:</td>
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<tr>
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<td>0 (0%)</td>
</tr>
<tr>
<td>2</td>
<td>8 (15%)</td>
</tr>
<tr>
<td>3</td>
<td>21 (40%)</td>
</tr>
<tr>
<td>4</td>
<td>24 (45%)</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>26 (49%)</td>
</tr>
<tr>
<td>Creatinine (μmol/L)</td>
<td>161 (83)</td>
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<tr>
<td>IABP</td>
<td>33 (62%)</td>
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<tr>
<td>MI to procedure time (days)</td>
<td>13 [5-54]</td>
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</tbody>
</table>

MI (myocardial infarction), PCI (percutaneous coronary intervention), CABG (coronary artery bypass surgery), IRA (infarct related artery), CAD (coronary artery disease), VD (vessel disease). IABP (intra-aortic balloon counterpulsation pump)
Table 2. Procedural parameters.

<table>
<thead>
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<tr>
<td>Defect site</td>
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<tr>
<td>anterior</td>
<td>19 (36%)</td>
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<tr>
<td>apical</td>
<td>16 (30%)</td>
</tr>
<tr>
<td>inferior</td>
<td>18 (34%)</td>
</tr>
<tr>
<td>LV function</td>
<td></td>
</tr>
<tr>
<td>LVEF&gt;50%</td>
<td>16 (30%)</td>
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<tr>
<td>LVEF: 30-50%</td>
<td>26 (49%)</td>
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<tr>
<td>LVEF&lt;30%</td>
<td>11 (21%)</td>
</tr>
<tr>
<td>RV function</td>
<td></td>
</tr>
<tr>
<td>RV good</td>
<td>22 (42%)</td>
</tr>
<tr>
<td>RV dilated and hyperkinetic</td>
<td>9 (17%)</td>
</tr>
<tr>
<td>RV dilated and hypokinetic</td>
<td>22 (42%)</td>
</tr>
<tr>
<td>Size of defect (per mm)</td>
<td>18 [16-24]</td>
</tr>
<tr>
<td>General anesthesia</td>
<td>43 (81%)</td>
</tr>
<tr>
<td>Access</td>
<td></td>
</tr>
<tr>
<td>Femoro-femoral</td>
<td>24 (45%)</td>
</tr>
<tr>
<td>Femoral-jugular</td>
<td>25 (47%)</td>
</tr>
<tr>
<td>Single femoral</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Single-jugular</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Radio-jugular</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Inotropes</td>
<td>30 (57%)</td>
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<tr>
<td>AV loop</td>
<td>48 (91%)</td>
</tr>
<tr>
<td>Balloon sizing</td>
<td>26 (49%)</td>
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</table>

LV (left ventricle), LVEF (LV ejection fraction), RV (right ventricle), mm (millimeters), AV (arterio-venous)
Table 3. Procedural parameters.

<table>
<thead>
<tr>
<th>Procedural parameters</th>
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<tr>
<td>No of device placements attempted</td>
<td></td>
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<tr>
<td>0</td>
<td>3 (6%)</td>
</tr>
<tr>
<td>1</td>
<td>43 (81%)</td>
</tr>
<tr>
<td>2</td>
<td>6 (11%)</td>
</tr>
<tr>
<td>3</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>No of device placements successful</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>6 (11%)</td>
</tr>
<tr>
<td>1</td>
<td>44 (83%)</td>
</tr>
<tr>
<td>2</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>3</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Device type used</td>
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<tr>
<td>Muscular VSD</td>
<td>17 (32%)</td>
</tr>
<tr>
<td>PIVSD</td>
<td>34 (64%)</td>
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<tr>
<td>other</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Largest device size (mm)</td>
<td>18 (5)</td>
</tr>
<tr>
<td>Immediate reduction in shunt</td>
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<tr>
<td>No reduction</td>
<td>8 (15%)</td>
</tr>
<tr>
<td>Partial reduction</td>
<td>33 (62%)</td>
</tr>
<tr>
<td>Complete closure</td>
<td>12 (23%)</td>
</tr>
<tr>
<td>Procedure duration (min)</td>
<td>136 [104-172]</td>
</tr>
<tr>
<td>Fluoroscopy time (min)</td>
<td>136 [104-172]</td>
</tr>
</tbody>
</table>

VSD (ventricular septal defect), PIVSD (post-infarct VSD), mm (millimeter), min (minutes)

Figure Legends:

Figure 1. Pathological specimens of implanted Amplatzer™ post-infarct VSD devices. A) 24mm 18mm and 16mm Amplatzer™ post-infarct VSD devices used to close a large PIVSD. B) Amplatzer™ post-infarct VSD device seen with organized thrombus filling the nitinol meshwork. PIVSD (post-infarction ventricular septal defect).

Figure 2. Echocardiographic images of ventricular septal defects (single arrow). A) Transesophageal echo (TEE) 4-chamber view. B) TEE 4-chamber view with color flow Doppler.
C) Transthoracic parasternal short axis view. D) Deployment of a 16mm Amplatzer™ post-infarct VSD device (SJM, Plymouth, MN) (double arrow), still on delivery cable (triple arrow). Note that the device is deployed in an apical VSD whilst two further VSDs are evident close to the mitral valve (single arrow).

**Figure 3.** Fluoroscopic images in postero-anterior projection of percutaneous post-infarct VSD closure. A) Right ventriculogram showing contrast passing through an apical ventricular septal defect (VSD) into the left ventricle. B) The VSD has been crossed with a hydrophilic (Terumo Medical Corporation, Somerset, NJ) wire (double arrow) passing from the right to left ventricle. This has been captured in the descending aorta by a snare pass from the right femoral artery (single arrow) to form an arterio-venous rail to support advancement of the delivery sheath. C) The left ventricular disc of a 16mm post-infarction VSD Amplatzer™ device (SJM, Plymouth, MN) (single arrow) has been deployed through a 9F Shuttle Sheath (Cook Medical Inc, Bloomington, IN). D) Amplatzer™ post-infarct VSD device.

**Figure 4.** Clinical factors associated with mortality on long-term follow up. Data plotted are unadjusted hazard ratio (box) and 95% confidence interval (stems). HR (hazard ratio), No of cor As (number of coronary arteries), NHYA (New York Heart Association classification).

**Figure 5.** Procedural factors associated with mortality on long-term follow up. Data plotted are unadjusted hazard ratio (box) and 95% confidence interval (stems). MI (myocardial infarction), HR (hazard ratio), mm (millimeter), min (minute).
Figure 6. Kaplan-Meier cumulative mortality curves. Kaplan-Meier estimate of long-term mortality for (A) all patients and (B) those that survived to discharge from hospital.

Figure 7. Kaplan-Meier cumulative mortality curves according to A) revascularization therapy for presenting myocardial infarction and B) usage of inotropes.
Figure 1
Figure 4

Unadjusted hazard ratio for survival and mortality.

- Age (per year)
  - HR 1.04 [1.002-1.09] p=0.039
- Female
  - HR 2.33 [1.03-5.26] p=0.043
- Diabetes
  - HR 0.68 [0.23-2.00] p=0.48
- Hypertension
  - HR 1.11 [0.46-2.70] p=0.82
- Smoking
  - HR 0.78 [0.29-2.10] p=0.62
- No revascularization therapy
  - HR 3.28 [1.34-7.99] p=0.009
- No. cor As with >70% stenosis
  - HR 1.47 [0.83-2.58] p=0.18
- NYHA IV
  - HR 4.42 [1.70-11.51] p=0.002
- Creatinine (per micromol/ml)
  - HR 1.007 [1.003-1.012] p=0.003
Unadjusted survival mortality hazard ratio

- Cardiogenic shock: HR 3.75 [1.55-9.09] p=0.003
- MI to closure time (days): HR 0.99 [0.98-1.001] p=0.077
- Defect site: HR 1.17 [0.73-1.85] p=0.52
- Size of defect (per mm): HR 1.09 [1.01-1.17] p=0.026
- Inotropes: HR 4.18 [1.55-11.26] p=0.005
- No of devices placed: HR 1.56 [0.61-4.11] p=0.34
- Largest device (per mm): HR 1.07 [0.98-1.18] p=0.13
- Immediate shunt reduction: HR 0.49 [0.25-0.96] p=0.037
- Prev surg closure: HR 0.12 [0.02-0.91] p=0.040

Figure 5
Figure 6

A

Kaplan-Meier cumulative mortality for all patients

Cumulative death (fraction)

Follow up from procedure (years)

Number at risk

53 18 11 9 9 5 4 4 2 2 2

B

Kaplan-Meier cumulative mortality for patients discharged from hospital

Cumulative death (fraction)

Follow up from discharge (years)

Number at risk

31 16 11 9 9 5 4 4 2 2 2
Figure 7

A

Cumulative death (fraction) vs. follow up from procedure (days)

- No revascularization therapy
- Revascularization therapy

Log rank = 0.005

No revascularization, number at risk
- 26
- 11
- 9
- 9
- 9
- 9
- 9

Revascularization, number at risk
- 27
- 21
- 16
- 14
- 11
- 11

B

Cumulative death (fraction) vs. follow up from procedure (days)

- Inotropes
- No inotropes

Log rank = 0.002

Inotropes, number at risk
- 30
- 14
- 10
- 9
- 7
- 7

No inotropes, number at risk
- 23
- 17
- 15
- 13
- 12
- 12
Percutaneous Closure of Post-Infarction Ventricular Septal Defect: In-Hospital Outcomes and Long-Term Follow-Up of UK Experience

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Figure Legend:

**Supplemental Figure 1.** A malconformed post-infarction VSD Amplatzer™ device (SJM, Plymouth, MN) (single arrow) is seen sitting within a serpiginous PIVSD. Although the device was still attached to the delivery cable and therefore under tension, its conformation was little altered after release from the cable.