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

Patrick A. Calvert, PhD, MRCP; James Cockburn, MD, MRCP; Dylan Wynne, PhD, FRACP;
Peter Ludman, MD, FRCP; Bushra S. Rana, MD, MRCP; David Northridge, MD;
Michael J. Mullen, MD, FRCP; Iqbal Malik, PhD, FRCP; Mark Turner, MD;
Saib Khogali, MD, FRCP; Gruschen R. Veldtman, MD, FRCP, MD; Martin Been, MD;
Rob Butler, MD; John Thomson, MD, FRCP; Jonathan Byrne, PhD, FRCP;
Philip MacCarthy, PhD, FRCP, Lindsay Morrison, MD, FRCP, Len M. Shapiro, MD, FRCP;
Ben Bridgewater, MD; Jo de Giovanni, MD; David Hildick-Smith, MD, FRCP

Background—Postinfarction ventricular septal defect 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—Postinfarction ventricular septal defect closure was attempted in 53 patients from 11 centers (1997–2012; aged 72±11 years; 42% female). Nineteen percent had previous surgical closure. Myocardial infarction was anterior (66%) or inferior (34%). Time from myocardial infarction to closure procedure was 13 (first and third quartiles, 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 (23%), partial (62%), or none (15%). Median length of stay after the procedure was 5.0 (2.0–9.0) days. Fifty-eight percent survived to discharge and were followed up for 395 (63–1522) days, during which time 4 additional patients died (7.5%). Factors associated with death after postinfarction ventricular septal defect closure included the following: age (hazard ratio [HR]=1.04; \( P=0.039 \)), female sex (HR=2.33; \( P=0.043 \)), New York Heart Association 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 myocardial infarction (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 postinfarction ventricular septal defect 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. (Circulation. 2014;129:2395-2402.)

Key Words: Amplatzer occluder device | death | myocardial infarction | percutaneous administration | ventricular septal defect

Clinical Perspective on p 2402

Ventricular septal rupture after myocardial infarction carries a grim prognosis. Survival to 1 month without intervention is 6%.

The online-only Data Supplement is available with this article at http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIRCULATIONAHA.113.005839/-/DC1.

Received August 28, 2013; accepted March 21, 2014.

From the University Hospitals Birmingham National Health Service Foundation Trust, Birmingham, UK (P.A.C., P.L., J.d.G.); University of Cambridge, Cambridge, UK (P.A.C.); Papworth Hospital National Health Service Foundation Trust, Cambridge, UK (P.A.C., B.S.R., L.M.S.); Sussex Cardiac Centre, Brighton and Sussex University Hospitals, Brighton, UK (J.C., D.W., D.H.-S.); Edinburgh Royal Infirmary, Edinburgh, UK (D.N.); The Heart Hospital, University College London, London, UK (M.J.M.); Imperial College Healthcare National Health Service Trust, London, UK (I.M.); University Hospitals Bristol National Health Service Foundation Trust, Bristol, UK (M.T.); Heart and Lung Centre, New Cross Hospital, Wolverhampton, UK (S.K.); University Hospital Southampton National Health Service Foundation Trust, Southampton, UK (G.R.V.); Walsgrave Hospital Coventry, Coventry, UK (M.B.); University Hospital North Staffordshire National Health Service Trust, Stoke-on-Trent, UK (R.B.); Leeds Teaching Hospitals National Health Service Trust, Leeds, UK (J.T.); King’s College Hospital National Health Service Foundation Trust, London, UK (J.B., P.M.); Liverpool Heart and Chest Hospitals National Health Service Foundation Trust, Liverpool, UK (L.M.); and University Hospital of South Manchester, Manchester, UK (B.B.).

Correspondence to David Hildick-Smith, MD, FRCP, Sussex Cardiac Centre, Brighton and Sussex University Hospitals, Eastern Road, Brighton, BN2 5BE, UK; E-mail david.hildick-smith@bsuh.nhs.uk

© 2014 American Heart Association, Inc.

Circulation is available at http://circ.ahajournals.org

DOI: 10.1161/CIRCULATIONAHA.113.005839
advent of the Amplatzer family of ventricular septal defect (VSD) 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 postinfarction VSD (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 PIVSD cases in which percutaneous intervention had been attempted in the United Kingdom.

Methods

Device implantation for PIVSD has been attempted in the United Kingdom since 1997. Units report numbers of cases undertaken to the British Cardiovascular Intervention Society; this information is published annually at http://www.bcis.org.uk. All centers (n=14) where PIVSD activity has been reported in the United Kingdom 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 centers, 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, preprocedural 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 >24 mm in diameter (the largest available device).

Cardiogenic shock was defined according to the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial. Coronary artery disease was defined as >70% stenosis in 1 of the 3 major epicardial coronary arteries.

Procedure

The majority of procedures in the United Kingdom were done by 1 of 2 operators (J.D.G., D.H.-S.) on a visiting basis. Procedures were usually done with the patient under general anesthesia with transesophageal echocardiographic imaging (Figure 2). Once femoral arterial access was gained, the VSD was crossed left to right in the majority of cases, and a wire was 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 the left ventricle with the wire snared in the aorta (Figure 3B), but given the trabeculated nature of the right ventricle 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 with the use of transesophageal echocardiography to measure the dimensions of the defect both on 2 dimensions and on color flow mapping. The PIVSDs were often complex in shape, and, when available, 3-dimensional transesophageal echocardiography 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 in cases in which this was not used, the device was oversized compared with the transesophageal echocardiography measurements, with 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, 24 versus 18 mm, respectively) and also has a thicker waist (10 versus 7 mm) and therefore was more suited to larger PIVSD or hypertrophied ventricular septa. The device was then delivered via a TorqueVue sheath (SJM) 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 to gain complete closure. Whether a second device was implanted depended on the size and shape of the residual defect and also on whether there was a rim present. In cases in which 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 a circumstance, the device was oversized in an attempt to splay the discs of the device against the free wall 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

Figure 1. Pathological specimens of implanted Amplatzer postinfarction ventricular septal defect devices. A, View of 24-, 18-, and 16-mm Amplatzer postinfection ventricular septal defect devices used to close a large postinfarction ventricular septal defect. B, Amplatzer postinfection ventricular septal defect device seen with organized thrombus filling the nintinol meshwork.

Figure 2. Echocardiographic images of ventricular septal defects (single arrow). A, Transesophageal echocardiographic 4-chamber view. B, Transesophageal echocardiographic 4-chamber view with color flow Doppler. C, Transthoracic parasternal short-axis view. D, Deployment of a 16-mm Amplatzer postinfarction ventricular septal defect device (SJM, Plymouth, MN; double arrow), still on delivery cable (triple arrow). The device is deployed in an apical ventricular septal defect, and 2 additional ventricular septal defects are evident close to the mitral valve (single arrow).
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 with the use of SPSS version 19.0 (SPSS Inc, Chicago, IL). Only first attempts at percutaneous PIVSD closure were included in the analysis. Normally distributed data are presented as mean±SD, and nongaussian data are presented as median (first and third quartiles). Categorical data are presented as frequency (percentage). Clinical and procedural parameters were tested for an association with death during follow-up with the use of univariable Cox proportional hazards regression.

Results

Demographics

Between December 1997 and January 2012, 58 attempted PIVSD closure procedures were reported in 53 patients among 11 centers in the United Kingdom. In 5 patients, the percutaneous closure attempt failed, and the patients underwent repeated percutaneous closure attempts. These 5 repeated percutaneous closures attempts were excluded from the analysis for statistical reasons. The median number of procedures performed per center was 5 (first and third quartiles, 1–8), with 1 center performing 14 procedures. Only first attempts at percutaneous PIVSD closure were included in the analysis (n=53).

Ten patients (19%) 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 to closure was 13 (5–54) days and was not associated with late mortality (hazard ratio [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 the following: 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 device.

Table 1. Clinical Parameters (n=53)

<table>
<thead>
<tr>
<th>Clinical Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>72.2 (10.8)</td>
</tr>
<tr>
<td>Female sex, No. (%)</td>
<td>22 (42)</td>
</tr>
<tr>
<td>Height, mean (SD), cm</td>
<td>169.7 (8.6)</td>
</tr>
<tr>
<td>Weight, mean (SD), kg</td>
<td>74.5 (12.8)</td>
</tr>
<tr>
<td>Diabetes mellitus, No. (%)</td>
<td>11 (21)</td>
</tr>
<tr>
<td>Hypertension, No. (%)</td>
<td>36 (68)</td>
</tr>
<tr>
<td>Smoking, No. (%)</td>
<td>15 (28)</td>
</tr>
<tr>
<td>Hypercholesterolemia, No. (%)</td>
<td>27 (51)</td>
</tr>
<tr>
<td>Initial MI treatment, No. (%)</td>
<td>None (62)</td>
</tr>
<tr>
<td>PCI</td>
<td>15 (28)</td>
</tr>
<tr>
<td>CABG</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Rescue PCI, No. (%)</td>
<td>3 (6)</td>
</tr>
<tr>
<td>PCI of infarct-related artery (any stage), No. (%)</td>
<td>15 (28)</td>
</tr>
<tr>
<td>Previous surgical closure, No. (%)</td>
<td>10 (19)</td>
</tr>
<tr>
<td>Infarct territory, No. (%)</td>
<td>Inferior (35)</td>
</tr>
<tr>
<td></td>
<td>Anterior (18)</td>
</tr>
<tr>
<td>Vessels with CAD, No. (%)</td>
<td>31 (58)</td>
</tr>
<tr>
<td></td>
<td>17 (32)</td>
</tr>
<tr>
<td></td>
<td>5 (9)</td>
</tr>
<tr>
<td>NYHA classification, No. (%)</td>
<td>I (0)</td>
</tr>
<tr>
<td></td>
<td>II (15)</td>
</tr>
<tr>
<td></td>
<td>III (40)</td>
</tr>
<tr>
<td></td>
<td>IV (45)</td>
</tr>
<tr>
<td>Cardiogenic shock, No. (%)</td>
<td>26 (49)</td>
</tr>
<tr>
<td>Creatinine, mean (SD), μmol/L</td>
<td>161 (83)</td>
</tr>
<tr>
<td>IABP, No. (%)</td>
<td>33 (62)</td>
</tr>
<tr>
<td>MI to procedure time, median (quartiles 1–3), d</td>
<td>13 (5–54)</td>
</tr>
</tbody>
</table>

CABG indicates coronary artery bypass graft; CAD, coronary artery disease; IABP, intra-aortic balloon counterpulsation pump; MI, myocardial infarction; NYHA, New York Heart Association; and PCI, percutaneous coronary intervention.
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 3 of 7 patients in whom it was attempted. Failure to deliver multiple devices was due to failure to cross 1 or more defects (n=2) and unstable device position (n=2). Immediate complete or partial shunt reduction was seen in 12 (23%) and 33 patients (62%), respectively (Table 3). No shunt reduction occurred in 8 patients (15%).

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 on to have a surgical repair after attempted percutaneous closure because of hemolysis, device embolization, and failure of percutaneous closure attempt.

Follow-Up
Eighteen patients (34%) died before discharge, with 31 patients (58%) surviving to discharge. Long-term follow-up among patients who survived to hospital discharge was for 395 (first and third quartiles, 63–1522) days. During this time, only an additional 4 patients died.

Factors Associated With Long-Term Death
Factors associated with death on long-term follow-up included the following: age, female sex, New York Heart Association class IV, cardiogenic shock, creatinine, size of defect, absence of revascularization therapy for presenting myocardial infarction, and use of inotropes (Figures 4 and 5). Prior surgical closure of the VSD and immediate reduction in shunt were associated with survival (Figures 4 and 5). Kaplan–Meier cumulative mortality curves are displayed in Figures 6 and 7.

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

PIVSD in the United Kingdom
The UK Myocardial Ischemia National Audit Project reported 32439 ST-segment elevation myocardial infarctions for the year April 2011 to March 2012. The incidence of PIVSD is 0.2% in the era of reperfusion. If we assume similar ST-segment elevation myocardial infarction rates for the 14 years under consideration, the overall number of PIVSD cases would be expected to be 908. During this same 14-year period, 743 patients underwent surgical repair of PIVSD, and 53 underwent percutaneous repair; therefore, there may
be some selection bias in the cases presented in this series. Our data demonstrate a 58% discharge survival among these relatively selected patients having percutaneous treatment. Survival from surgical closure series ranges between 20% and 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 and 5). This is perhaps to be expected, given that although 18 patients (34%) died during the index admission, once patients achieved discharge, only an additional 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 semipermeable at implantation and may take a number of days to completely occlude with organized thrombus (Figure 1B).18 Although this is acceptable and even useful in the elective setting, these highly unstable patients cannot necessarily tolerate persistent shunting after 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, although it has been reported.19 Expert consensus suggests that if the shunt is not reduced by at least two thirds, the patient is most

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

**Figure 5.** Procedural factors associated with mortality on long-term follow-up. Data plotted are unadjusted hazard ratios (box) and 95% confidence intervals (stems). MI indicates myocardial infarction; and Prev surg, previous surgical.
unlikely to survive either to surgery or to discharge, and our data support this consensus.

Many patients (47%) did not receive any form of reperfusion therapy, usually because of delayed presentation, and consequently they 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 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.\(^4\,17\,20\) 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-comer 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 nonsignificant trend in this direction (HR=0.99 [0.98–1.001]; \( P = 0.077 \)).

**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, particularly 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 because 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 because of 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 (Figure I in the online-only Data Supplement). However, the measure of a successful closure was shunt reduction without embolization alone and not cosmesis. Our experience suggests that arteriovenous loops are invaluable, as are braided guiding catheters to prevent kinking. In the 5 patients in whom an arteriovenous 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 because of extended rupture, free wall rupture, renal failure, inflammation, sepsis, vascular access complications, and multiorgan failure.

Our data confirm that the overall outlook for postinfarction ASD is poor but that selected cases can be successfully treated. In the SHOCK and Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO) trials, mortality in patients with PIVSD and cardiogenic shock was 87% to 100%.

Existing Literature on PIVSD Closure

Series on postinfarction septal closure are few. Thiele et al reported 29 cases in a single center over a 6-year period. Their “warts and all” publication exposes the limitations of the percutaneous approach in this setting. Closure was attempted 1 to 3 days after diagnosis. Eighty-six percent of patients had a technical success of device implantation, but 17% died in the catheter laboratory, 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 because of complications of the disease process itself. Our data corroborate this, with 89% device implantation success but only 59% survival to discharge.

The largest series before the present analysis was a single-center series of 40 closures in 30 patients over a 20-year period. Sixty percent of these patients were being treated for residual leaks after surgery, and these patients seem to form a lower-risk cohort, judging by both their own exemplary mortality rate of 23% and our own data in which 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 extensive use of an externalized guidewire; however, unlike our cohort, this was often a venous loop rather than an arteriovenous loop.

Although the parameters tested in the present analysis and the study by Assenza et al differ, both investigators found similar trends: 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 centers over a 3-year period, in the majority of whom the procedure was attempted >2 weeks after the infarction. Procedural success was 16 of 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 modeling identified age, female sex, shock, preoperative aortic counterpulsation balloon, redo surgery, emergency status, preoperative 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 the retrospective nature of our study, 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 (ie, those who underwent conservative or surgical management). Finally, it is possible that our report has not captured all cases of percutaneous VSD repair in the United Kingdom because 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 survive to discharge have excellent outcomes on long-term follow-up.

Acknowledgments

Thanks to Simon Bond, MA, PhD (Cambridge Clinical Trials Unit and Medical Research Council Biostatistics Unit), for statistical advice.

Disclosures

Drs Hildick-Smith and de Giovanni are proctors for St Jude AGA Medical. The other authors report no conflicts.

References


**CLINICAL PERSPECTIVE**

Postinfarction ventricular septal defect (PIVSD) is a catastrophic complication of myocardial infarction that is associated with poor outcomes, especially if treated conservatively. The current American College of Cardiology Foundation/ American Heart Association and European Society of Cardiology guidelines both recommend early surgery. However, surgery is often withheld or delayed, and outcomes after surgery remain poor, with repeat intervention often required for dehiscence. This study assesses the long-term mortality of those patients with PIVSD who underwent attempted percutaneous closure in the United Kingdom between 1997 and 2012. In those with technically successful device closure of the PIVSD (89%), there was an immediate reduction in shunt in 96%. Major immediate complications included procedural death (3.8%) and emergency cardiac surgery (7.5%). Survival to discharge occurred in 58%, with only an additional 4 patients (7.5%) dying thereafter. Factors associated with long-term death included age, female sex, raised serum creatinine, defect size, New York Heart Association class IV dyspnea, cardiogenic shock, and use of inotropes. Prior surgical closure, revascularization therapy, and immediate shunt reduction were all associated with long-term survival. Percutaneous PIVSD closure is a technically challenging procedure. Our experience suggests that arteriovenous loops are invaluable, as are braded guiding catheters to prevent kinking. The defects are often complex, serpiginous, multiple, and difficult to size. Three-dimensional transesophageal echocardiography is useful in understanding the anatomy. The rims are usually friable, and significant oversizing of the device is recommended. Percutaneous device closure of PIVSD is a challenging but viable option for these critically unwell patients.
Percutaneous Closure of Postinfarction Ventricular Septal Defect: In-Hospital Outcomes and Long-Term Follow-Up of UK Experience

Patrick A. Calvert, James Cockburn, Dylan Wynne, Peter Ludman, Bushra S. Rana, David Northridge, Michael J. Mullen, Iqbal Malik, Mark Turner, Saib Khogali, Gruschen R. Veldtman, Martin Been, Rob Butler, John Thomson, Jonathan Byrne, Philip MacCarthy, Lindsay Morrison, Len M. Shapiro, Ben Bridgewater, Jo de Giovanni and David Hildick-Smith

_Circulation_. 2014;129:2395-2402; originally published online March 25, 2014; doi: 10.1161/CIRCULATIONAHA.113.005839

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/129/23/2395

Data Supplement (unedited) at:
http://circ.ahajournals.org/content/suppl/2014/03/24/CIRCULATIONAHA.113.005839.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/
SUPPLEMENTAL MATERIAL
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.