VSD surgical closure: better results justify increased invasiveness

Panagiotis Dedeilias, MD, PhD, FACTS
Evangelismos Hospital, Athens, Greece
**Latham**
First described at autopsy in 1845

**Cooley**
First successful surgical repair in a patient after 9 weeks post detection of VSR in 1956

**Heimbecker, Allen, Woodwark, Iben**
Surgery for acute phase in the late 1960s
General Statistics

**Incidence**: 1-3% of MI without reperfusion therapy

- 0.2-0.34% in fibrinolytic therapy *Antman, Anbe, Armstrong et al*

- 0.23% in prim PCI *Yip et al 0.17% (APEX-AMI)*

- 3.9% in patients with cardiogenic shock

**Male : Female = 3:2** *Grenshaw 2000*

- 1 vessel (50%), 2 vessels (40%), 3 vessels (10%) *Gusto-I Circ. 2000*

**Time course**: Bimodal peak = within 24 hours and 3-5 days

**Chronic VSR** = more than 4-6 weeks
ΣΤΑΤΙΣΤΙΚΑ ΣΤΟΙΧΕΙΑ II

- GUSTO – I (Global Utilization of Streptokinase and t-PA for Occluded coronary arteries).
- 41.000 ασθενείς με MI
- Ποσοστό VSD: 0.2%

Murday A. heart 2003.
ΣΤΑΤΙΣΤΙΚΑ ΣΤΟΙΧΕΙΑ ΙΙΙ

45.000 OEM/χρόνο
90 VSD’s/χρόνο
45 → χειρουργείο
ΘΕΣΗ VSD

• 60% πρόσθιο-κορυφαίο τμήμα διαφράγματος.

• 20-40% οπίσθιο διάφραγμα μετά από κατωτεροδιαφραγματικό OEM από απόφραξη επικρατούσας δεξιάς ή περισσότερο σπάνια, επικρατούσας περισπωμένης αρτηρίας.
Μηχανισμός πρόκλησης

- Οξεία πλήρης απόφραξη μεγάλου αγγείου που τροφοδοτεί το διάφραγμα με ταυτόχρονη έλλειψη παράπλευρου δικτύου.
- Διολίσθηση μυοκυττάρων, έλλειψη συνδετικού ιστού, αποδόμηση υαλίνης μυοκυττάρων, ενζυμική κυτταρόλυση.

David TE: Mechanical Complications of Myocardial infarction 1993; p175
Από τους επιζήσαντες 35-68% θα εξελιχθούν σε ανεύρυσμα λόγω αναδιαμόρφωσης.

*Preffer MA, Brawnwald E: Circulation 1990*
30% των οπισθίων-κατωτέρων post ΜΙ VSD’S σχετίζονται με MR
Παθοφυσιολογία

L -> R shunt + Ελάττωση συσταλτικότητας LV

Οξεία Αύξηση Πνευμονικής Κυκλοφορίας Οξεία ελάττωση Συστηματικής κυκλοφορίας

+ Ανεπάρκεια Μιτροειδούς — CO
Διάγνωση

1. Νέο συστολικό φύσημα (90%).
2. Υποτροπή θωρακικού άλγους.
3. Οξεία αιμοδυναμική επιδείνωση.
4. Νέο block.
5. x-Ray: αυξημένη πνευμονική αγγείωση.
8. ECHO + έγχρωμο Doppler.
9. Καθετηριασμός, στεφανιογραφία, κοιλιογραφία.
ΠΡΟΕΓΧΕΙΡΗΤΙΚΗ ΑΝΤΙΜΕΤΩΠΙΣΗ

1. Μεταφορά σε Μονάδα Εντατικής θεραπείας.
2. Τοποθέτηση Swan-Ganz.
3. Ελάττωση SVR και L->R shunt με αγγειοδιασταλτικά εφ’ όσον το επιτρέπει η Α.Π. του ασθενούς.
4. Διατήρηση CO με ινότροπα.
5. Χρήση IABP για ελάττωση μεταφορτίου, ελάττωση απαιτήσεων O₂ από το μυοκάρδιο, αύξηση ροής στεφανιαίων.
6. Μηχανικός αερισμός αν απαιτείται.
7. ECHO + έγχρωμο doppler για καθορισμό θέσης και μεγέθους VSD.
8. Καθετηριασμός στεφανιαίων.
ΦΥΣΙΚΗ ΕΞΕΛΙΞΗ

Χωρίς χειρουργική αντιμετώπιση
25% καταλήγουν σε 24 ώρες
50% καταλήγουν σε 1 εβδομάδα
65% καταλήγουν σε 2 εβδομάδες
80% καταλήγουν σε 1 μήνα

Μόνο 7% ζουν πάνω από 1 έτος
Outcome and profile of ventricular septal rapture with cardiogenic shock after myocardial infarction

Menon V, Webb J, et al

55 cases. Mε post MI VSD

Μέσος χρόνος από MI έως VSD : 7-3 hours

24 cases. Φαρμακευτικά, IABP, κλπ

31 χειρουργείο, υψηλού κινδύνου

Επέζησαν μόνον 7, οι 6 από τους οποίους είχαν υποβληθεί σε χειρουργική διόρθωση
ΠΟΤΕ
ΔΕΝ ΘΑ ΧΕΙΡΟΥΡΓΗΘΟΥΜΕ;
ΜΟΝΟΝ όταν SMOF είναι δεδομένη

A. Murday, Heart 2003
ΧΕΙΡΟΥΡΓΕΙΟ ΑΜΕΣΑ Ή OΧΙ;

Μέχρι 1982: 26 ασθενείς με post MI VSD.
2 σταθεροί αιμοδυναμικά, όχι χειρουργείο
6 θάνατοι προ χειρουργείου
3 θάνατοι μετά χειρουργείο
Χειρουργική θνητότητα 17% συνολική θνητότητα 38%

Μετά 1982: 29 ασθενείς με post MI VSD
2 σταθεροί που δεν χρειάστηκαν χειρουργείο
2 θάνατοι σε ασταθείς αιμοδυναμικά που δεν χειρουργήθηκαν
7 θάνατοι μετά το χειρουργείο
Χειρουργική θνητότητα 32%, συνολική θνητότητα 33%

Morell et al Eur Heart 1987
Επαναγγείωση λοιπών ισχαιμικών αλλοιώσεων

- ΝΑΙ γιατί βελτιώνει την άμεση και απώτερη επιβίωση.
- Πρέπει οι περιφερικές αναστομώσεις να προηγούνται της διόρθωσης του VSD.
- ΟΧΙ μοσχεύματα στην εμφραγματική περιοχή.

Cox, Plokker et al, European Heart J, 1996
Repair of post-infarct ventricular septal defect with or without coronary artery bypass grafting in the northwest of England: a 5-year multi-institutional experience

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Received 1 May 2003; received in revised form 17 July 2003; accepted 18 July 2003
Follow Up (months)

<table>
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<th>30-Day</th>
<th>12-months</th>
<th>24-months</th>
<th>48-months</th>
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</thead>
<tbody>
<tr>
<td>CABG – Yes</td>
<td>36</td>
<td>30</td>
<td>28</td>
<td>16</td>
</tr>
<tr>
<td>CABG - No</td>
<td>17</td>
<td>15</td>
<td>10</td>
<td>6</td>
</tr>
</tbody>
</table>

Observed mid-term survival based on whether concomitant CABG performed.

Freedom from death (%)

- Concomitant CABG
- No concomitant CABG

p = 0.62
Mid-term survival based on whether concomitant CABG performed, adjusted for unstable angina, current smoking, and total occlusion of infarct related artery.
### Αποσύνδεση από εξωσωματική

<table>
<thead>
<tr>
<th>Ενδείξεις τοποθέτησης LVAD</th>
<th>Ενδείξεις για RVAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>CI &lt;1-8 L/min-m²</td>
<td>CI &lt;1-8 L/min-m²</td>
</tr>
<tr>
<td>LA pressure &gt;18-25 mmHg</td>
<td>LA pressure &lt;15 mmHg</td>
</tr>
<tr>
<td>RA pressure &lt;15 mmHg</td>
<td>RA pressure &gt;25 mmHg</td>
</tr>
<tr>
<td>Σ.Α.Π.&lt;90 mmHg</td>
<td>Σ.Α.Π.&lt;90 mmHg</td>
</tr>
</tbody>
</table>

*COHN’S CARDIAC SURGERY IN THE ADULT 2013*
Principles of repair of postinfarction ventricular septal defects

1. Expeditious establishment of total cardiopulmonary bypass with moderate hypothermia and meticulous attention to myocardial protection.
2. Transinfarct approach to ventricular septal defect with the site of ventriculotomy determined by the location of the transmural infarction.
3. Thorough trimming of the left ventricular margins of the infarct back to viable muscle to prevent delayed rupture of the closure.
4. Conservative trimming of the right ventricular muscles as required for complete visualization of the margins of the defect.
5. Inspection of the left ventricular papillary muscles and concomitant replacement of the mitral valve only if there is frank papillary muscular rupture.
6. Closure of the septal defect without tension, which in most instances will require the use of prosthetic material.
7. Closure of the infarctectomy without tension with generous use of prosthetic material as indicated, and epicardial placement of the patch to the free wall to avoid strain on the friable endocardial tissue.
8. Buttressing of the suture lines with pledgets or strips of Teflon felt or similar material to prevent sutures from cutting through friable muscle.

The American Heart Association recommends an urgent closure of the rupture in all patients.

Emergency surgical repair is necessary, even in hemodynamically stable patients because the rupture site can expand abruptly, resulting in sudden hemodynamic collapse in previously stable patients.
ACC/AHA Guidelines for the management of acute heart failure 2013
Early surgery is associated with a high mortality rate, reported as 20-40%, and a high risk of recurrent ventricular rupture, while delayed surgery allows easier septal repair in scarring tissue but carries the risk of rupture extension and death in all patients with severe heart failure that does not respond rapidly to aggressive therapy, but delayed elective surgical repair may be considered in patients who respond well to aggressive heart failure therapy.

Percutaneous closure of the defect with appropriately designed devices may soon become an alternative to surgery.
ΧΕΙΡΟΥΡΓΙΚΕΣ ΤΕΧΝΙΚΕΣ

1. Κορυφαία ρήξη διαφράγματος.
2. Πρόσθια ρήξη.
3. Κατώτερη – οπίσθια.
4. Διαδερμική σύγκλειση.
5. Διακολπική (ΔΕ κόλπος).
6. Δια του RVOT.
Πρώιμη χειρουργική αντιμετώπιση της μετεμφραγματικής ρήξης του μεσοκοιλιακού διαφράγματος

Daggett et al, 1977
Nonexposed

David et al, 1995
Exposed technic
Surgery of postinfarction ventricular septal rupture: The effect of David infarct exclusion versus Daggett direct septal closure on early and late outcomes

Objective: David infarct exclusion and Daggett direct septal closure are alternative techniques to repair postinfarction ventricular septal rupture. The aim of the present study was to compare the 2 methods with regard to postoperative morbidity, 30-day mortality, and long-term survival.

Methods: From May 1981 to December 2010, 110 patients underwent surgery for postinfarction ventricular septal rupture. Data were collected on the clinical, angiographic, and echocardiographic findings, operative procedures, early morbidity, and survival time. The epidemiologic design was of an exposed (David infarct exclusion, n = 42) versus a nonexposed (Daggett direct closure, n = 68) cohort with 3 endpoints: postoperative morbidity, 30-day mortality, and long-term survival. The crude effect of the repair technique versus the endpoint was estimated using univariate statistics. Stratification analysis using the Mantel-Haenszel method was done to quantify the confounders and pinpoint the effect modifiers. Adjustment for confounders was performed using logistic regression and Cox regression analysis, and with propensity score stratification statistics. Survival curves were analyzed using the Breslow test and log-rank test.

Results: The surgical technique had no influence on postoperative morbidity. The 30-day mortality was 16.7% in the David group and 48.5% in the Daggett group (P = .000). Long-term survival was greater after David than after Daggett, with 5- and 10-year survival of 69% versus 38% and 48% versus 27%, respectively (P = .004). Total coronary revascularization improved survival more in the David than in the Daggett group.

Conclusions: David infarct exclusion was superior to Daggett direct septal closure for early and late survival after surgery for postinfarction ventricular septal rupture. Total coronary revascularization improved survival more in the David than in the Daggett group. (J Thorac Cardiovasc Surg 2014;148:2736-42)

Runar Lundblad, MD, PhD, and Michel Abdelnoor, MPH, PhD
Repair of posterior postinfarction ventricular septal defect (VSD). **A,** The heart is lifted out of the pericardial cavity, and the VSD is approached through a vertical incision in the infarcted left ventricular myocardium. **B,** Infarcted tissue (right ventricle [RV], left ventricle [LV], and septum) is excised. Dashed lines indicate limits of excision. **C,** Septal patch is placed on LV side of the septum and secured using polyester mattress sutures, with pledgets placed on RV side of the septum.

Key: IVC, inferior vena cava; LV, left ventricle; RV, right ventricle.

Continued
If a second patch is required to close the free wall, the free edge of the septal patch is sutured to the free wall of the left ventricle (LV). The patch for closure of the RV is attached to the septal patch already in position and to the free wall of the right ventricle (RV) (see text for details). E, If extensive infarction is present, no infarcted muscle on the free wall of the RV or LV is excised. The septal patch is sutured to the RV edge of the incision that was made to expose the septum. This suture line incorporates the infarcted but intact LV free wall and a patch of polyester placed over the infarcted muscle (see text for details). F, Completed repair, with interrupted sutures placed through the myocardium, the external patch, and a strip of polytetrafluoroethylene felt.

Key: IVC, Inferior vena cava; LV, left ventricle; PTFE, polytetrafluoroethylene; RV, right ventricle.
Repairs of an anterior postinfarction ventricular septal rupture using the technique of infarct exclusion. (A) The standard ventriculotomy is made in the infarcted area of left ventricular free wall. An interior patch of Dacron (Meadox Medicals Inc., Oakland, NJ), polytetrafluoroethylene, or glutaraldehyde-fixed pericardium is fashioned to replace and/or cover the disease areas (septal defect. Septal infarction, or free wall infarction). (B) The internal patch is secured to normal endocardium with a continuous monofilament suture, which may be reinforced with pledgeted mattress sutures. There is little, if any, reception of myocardium and no attempt is made to close the septal defect.
Double-Patch Repair of Postinfarction Ventricular Septal Defect
Surgical Repair of Ventricular Septal Defect After Myocardial Infarction: Outcomes From The Society of Thoracic Surgeons National Database


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George J. Arnaoutakis, MD, Yue Zhao, PhD, Timothy J. George, MD, Christopher M. Sciortino, MD, PhD, Patrick M. McCarthy, MD, and John V. Conte, MD
**Background.** The development of a ventricular septal defect (VSD) after myocardial infarction (MI) is an uncommon but highly lethal complication. We examined The Society of Thoracic Surgeons database to characterize patients undergoing surgical repair of post-MI VSD and to identify risk factors for poor outcomes.

**Methods.** This was a retrospective review of The Society of Thoracic Surgeons database to identify adults (aged ≥18 years) who underwent post-MI VSD repair between 1999 and 2010. Patients with congenital heart disease were excluded. The primary outcome was operative death. The covariates in the current The Society of Thoracic Surgeons model for predicted coronary artery bypass grafting operative death were incorporated in a logistic regression model in this cohort.

**Results.** The study included 2,876 patients (1,624 men [56.5%]), who were a mean age of 68 ± 11 years. Of these, 215 (7.5%) had prior coronary artery bypass grafting operations, 950 (33%) had prior percutaneous intervention, and 1,869 (65.0%) were supported preoperatively with an intraaortic balloon pump. Surgical status was urgent in 1,007 (35.0%) and emergencies in 1,430 (49.7%). Concomitant coronary artery bypass grafting was performed in 1,837 (63.9%). Operative mortality was 54.1% (1,077 of 1,990) if repair was within 7 days from MI and 18.4% (158 of 856) if more than 7 days from MI. Multivariable analysis identified several factors associated with increased odds of operative death.

**Conclusions.** In the largest study to date to examine post-MI VSD repair, ventricular septal rupture remains a devastating complication. As alternative therapies emerge to treat this condition, these results will serve as a benchmark for future comparisons.

## Summary of reported clinical experience with surgical repair of postinfarction VSD

<table>
<thead>
<tr>
<th>Institution</th>
<th>City</th>
<th>Year</th>
<th>N</th>
<th>Hospital mortality</th>
<th>5-y survival</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Massachusetts General Hospital</td>
<td>Boston</td>
<td>2002</td>
<td>114</td>
<td>37%</td>
<td>45%</td>
<td>184</td>
</tr>
<tr>
<td>Papworth Hospital</td>
<td>Cambridge</td>
<td>2002</td>
<td>25</td>
<td>48%</td>
<td>-</td>
<td>185</td>
</tr>
<tr>
<td>University Hospital</td>
<td>Zurich</td>
<td>2000</td>
<td>54</td>
<td>26%</td>
<td>52%*</td>
<td>186</td>
</tr>
<tr>
<td>Sakurabashi Watanabe Hospital</td>
<td>Osaka</td>
<td>2000</td>
<td>16</td>
<td>33%</td>
<td>-</td>
<td>187</td>
</tr>
<tr>
<td>Glenfield General Hospital</td>
<td>Leicester</td>
<td>2000</td>
<td>117</td>
<td>37% (30 day)</td>
<td>46%</td>
<td>188</td>
</tr>
<tr>
<td>Evangelismos General Hospital</td>
<td>Athens</td>
<td>1999</td>
<td>14</td>
<td>50%</td>
<td>-</td>
<td>189</td>
</tr>
<tr>
<td>Texas Heart Institute</td>
<td>Houston</td>
<td>1998</td>
<td>126</td>
<td>46%</td>
<td>-</td>
<td>124</td>
</tr>
<tr>
<td>The Toronto Hospital</td>
<td>Toronto</td>
<td>1998</td>
<td>52</td>
<td>19%</td>
<td>65%*</td>
<td>190</td>
</tr>
<tr>
<td>Southampton General</td>
<td>Southampton</td>
<td>1998</td>
<td>179</td>
<td>27%</td>
<td>49%</td>
<td>121</td>
</tr>
<tr>
<td>Cedars – Sinai</td>
<td>Los Angeles</td>
<td>1998</td>
<td>31</td>
<td>32%</td>
<td>-</td>
<td>191</td>
</tr>
<tr>
<td>Mid America Heart Institute</td>
<td>Kansas City</td>
<td>1997</td>
<td>76</td>
<td>41%</td>
<td>41%</td>
<td>192</td>
</tr>
<tr>
<td>St. Anthonius Hospital</td>
<td>Nieuwegein</td>
<td>1996</td>
<td>109</td>
<td>28% (30 day)</td>
<td>-</td>
<td>193</td>
</tr>
<tr>
<td>Green Lane Hospital</td>
<td>Auckland</td>
<td>1995</td>
<td>35</td>
<td>31% (30 day)</td>
<td>60%*</td>
<td>194</td>
</tr>
<tr>
<td>Hospital Cardiologique du Haut – Lévèque</td>
<td>Bordeaux</td>
<td>1991</td>
<td>62</td>
<td>38%</td>
<td>44%</td>
<td>35</td>
</tr>
<tr>
<td>CHU Henri Mondor</td>
<td>Créteil</td>
<td>1991</td>
<td>66</td>
<td>45%</td>
<td>44%</td>
<td>195</td>
</tr>
</tbody>
</table>

* Value estimated from published graphical or tabular data.

Note: Series with less than 10 patients were excluded from the table.
Time-related survival after repair of postinfarction ventricular septal defect at the Massachusetts General Hospital (MGH, n=114). Note that the horizontal axis extends to 20 years. Circles represent each death, positioned on the horizontal axis at the interval from operation to death, and actuarially (Kaplan-Meier method) along the vertical axis. The vertical bars represent 70% confidence limits (± 1 SD). The solid line represents the parametrically estimated freedom from death, and the dashed lines enclose the 70% confidence limits of that estimate. The table shows the nonparametric estimates at specified intervals.
Survival in patients who were discharged after repair of postinfarction ventricular septal defect (MGH, n=72). The horizontal axis is expanded and represents the time from hospital discharge to death. The depiction is otherwise similar to to figure.

A. Murday Heart 2003, 1462-6
ΧΕΙΡΟΥΡΓΙΚΗ ΘΝΗΤΟΤΗΤΑ ΜΕΤΕΜΦΡΑΓΜΑΤΙΚΗΣ ΡΗΞΗΣ ΣΤΗΝ ΑΓΓΛΙΑ

**Miscellaneous procedures: Mortality; financial years 2004-2008**

- **Without CABG**
- **With CABG**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Without CABG</th>
<th>With CABG</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV aneurysmectomy</td>
<td>10%</td>
<td>25%</td>
</tr>
<tr>
<td>Acquired VSD</td>
<td>20%</td>
<td>35%</td>
</tr>
<tr>
<td>Atrial myxoma</td>
<td>15%</td>
<td>30%</td>
</tr>
<tr>
<td>Pericardieotomy</td>
<td>10%</td>
<td>20%</td>
</tr>
</tbody>
</table>

Crude mortality rates.
Post infarction ventricular septal defect – can we do better?☆

Marek A. Deja*, Jacek Szostek, Kazimierz Widenka, Bartlomiej Szafron, Tomasz J. Spyt, Mark St.J. Hickey, Andrzej W. Sosnowski

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Received 8 September 1999; received in revised form 2 May 2000; accepted 10 May 2000
Abstract

Objective: To identify predictors of early and late outcome among 117 consecutive patients who underwent postinfarction ventricular septal defect (VSD) repair over a period of 12 years. Methods: A retrospective analysis of clinical data was performed. Mean age was 65.5 ± 7.8. There were 43 females. Full data were obtained in 110 patients. Of these, 76 patients presented with anterior and 34 with posterior VSD. Thirty-three patients were operated in cardiogenic shock. Mean time between myocardial infarction (MI) and VSD development was 5.6 ± 7.8 days (median 4) and from VSD to surgery 9.0 ± 28.1 (median 2). Sixty-six patients had intraaortic balloon pump (IABP) inserted, and 15 were ventilated preoperatively. Logistic regression and Cox regression were used for multivariate analysis. Results: Thirty days mortality was 37% Among 110 patients, in whom complete analysis was possible, 38 died within 30 days (35%). Mortality in the posterior VSD group was 35% and in the anterior VSD group 34% (NS). In 44 patients (40%) a residual shunt was found on postoperative echocardiography. This required reoperation in 13 patients (four deaths). Cardiogenic shock prior to surgery adversely influenced early survival – odds ratio (OR) 5.7 (confidence interval (CI) 2.1–16.0) (P = 0.0008). Deterioration of haemodynamic status in between admission and surgery was stronger predictor of mortality than shock on admission – OR 6.0 (CI 1.6–22.6) (P = 0.008) vs. 3.1 (CI 1.0–9.3) (P = 0.049). A longer time between MI and surgery favoured survival – OR 0.1 (CI 0.03–0.4) (P = 0.002). The time period from the infarct to the septal rupture, but not from the rupture to surgery, appeared to be a significant predictor of survival – OR 0.2 (CI 0.05–0.6) (P = 0.008). Five years survival was 46 ± 5%. Preoperative cardiogenic shock affected late survival – OR 2.7 (CI 1.5–4.9) (P = 0.001). Of 72 patients who survived 30 postoperative days, 12 (17%) were in New York Heart Association (NYHA) class III or IV and five (6.9%) in Canadian Cardiovascular Sociey (CCS) class III or IV at the last follow-up. Conclusions: Preoperative cardiogenic shock and early postinfarction septal rupture carry a grave prognosis. Achieving haemodynamic stability prior to surgery may be beneficial but prolonged attempts to improve patients’ cardiovascular state are hazardous. © 2000 Elsevier Science B.V. All rights reserved.
Surgical repair of post infarction ventricular septal defects: a national experience

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Abstract

Objectives: Ventricular septal rupture is a rare but feared complication after acute myocardial infarction. Most reports about outcome after surgical treatment are single center experiences. We investigated the results after surgical repair in all patients in Sweden during a 7-year period. Methods: All patients undergoing surgical repair 1992-1998 were identified with the aid of the Swedish Heart Surgery Registry. The patients (n=189, 63% men, mean age 69 ± 8 years) were operated at 10 different centers. Pre-and peri-operative variables were collected from the Registry and individual patient charts. Mortality was calculated and a Cox proportional hazards regression model was used to identify independent predictors for early and late mortality. Mean follow-up was 2.4 years. Results: Seventy-seven of the 189 patients died within 30 days (41%). Urgent repair (Risk Ratio 4.2 (2.0-8.9), P<0.001) and posterior rupture (RR 2.1 (1.3-3.4), P=0.002) were independent predictors of 30-day mortality. Total cumulative survival (Kaplan-Meyer) was 38% at 5 years. For patients that survived the first 30 days (n=112), 5 year cumulative survival was 67%. Independent predictors for mortality after 30 days were number of concomitant coronary anastomoses (RR 1.5 (1.2-2.0), P=0.001), residual postoperative shunt (RR 2.7 (1.4-5.4), P=0.004) and postoperative dialysis (RR 3.4 (1.5-7.5), P=0.003). Conclusions: Early mortality after surgical repair of post infarction septal rupture is still considerable. Early repair and posterior rupture are predictors of early mortality. Long-term survival in patients surviving the immediate postoperative period is limited by pre-existing coronary artery disease, postoperative renal failure and the presence of a residual postoperative shunt.

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Ανεξάρτητοι παράγοντες κινδύνου/θανάτου

1) Προεγχειρητικό καρδιογενές shock: OR 5.7, P=0.0008
2) Ταχεία επιδείνωση απο τη διάγνωση: OR 6.0 P=0.008
3) Χρόνος μεσολάβησης απο MI->VSD vs VSD-> Surgery OR 2.0 P=0.008
4) Προυπάρχουσα στεφανιαία νόσος
5) Εναπομείναν μεγάλο VSD ή υποτροπή VSD
6) Νεφρική ανεπάρκεια
1. Cardio SEAL

Διπλή ομπρέλλα από νιτινόλη επικεκαλημένη με DACRON που κλείνει σαν όστρακο.


2. Amplatzer septal occluder

3. Rashkind double umbrella
**Background.** We reviewed our experience at the Montreal Heart Institute with early surgical and percutaneous closure of postinfarction ventricular septal defects (VSD).

**Methods.** Between May 1995 and November 2007, 51 patients with postinfarction VSD were treated. Thirty-nine patients underwent operations, and 12 were treated with percutaneous closure of the VSD.

**Results.** Half of the patients were in systemic shock, and 88% were supported with an intraaortic balloon pump before the procedure. Before the procedure, 14% of patients underwent primary percutaneous transluminal coronary angioplasty. The mean left ventricular ejection fraction was 0.44 ± 0.11, and mean Qp/Qs was 2.3 ± 1. Time from acute myocardial infarction to VSD diagnosis was 5.4 ± 5.1 days, and the mean delay from VSD diagnosis to treatment was 4.0 ± 4.0 days. A moderate to large residual VSD was present in 10% of patients after correction. Early overall mortality was 33%. Residual VSD, time from myocardial infarction to VSD diagnosis, and time from VSD diagnosis to treatment were the strongest predictor of mortality. Twelve patients were treated with a percutaneous occluder device, and the hospital or 30-day mortality in this group was 42%.

**Conclusion.** Small or medium VSDs can be treated definitively with a ventricular septal occluder or initially to stabilize patients and allow myocardial fibrosis, thus facilitating delayed subsequent surgical correction.

Συμπερασματικά η μετεμφραγματική ρήξη του μεσοκοιλιακού διαφράγματος αν και εμφανίζεται σπανιότερα απ' ότι στο παρελθόν αποτελεί μια επιπλοκή που έχει υψηλή θνητότητα και νοσηρότητα και πρέπει να αντιμετωπίζεται χειρουργικά με προτεραιότητα.
ΕΥΧΑΡΙΣΤΩ ΠΟΛΥ