COMPLICATIONS AFTER MITRAL VALVE SURGERY

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DISCLOSURES

- NONE
OBJECTIVE

• To understand some of the complications that can occur after mitral valve repair/replacement
• To be able to quote the incidence of complications after mitral valve surgery
• To understand the management and outcomes of complications after mitral valve surgery
Operative outcomes in mitral valve surgery: Combined effect of surgeon and hospital volume in a population-based analysis

Arman Kilic, MD, Ashish S. Shah, MD, John V. Conte, MD, William A. Baumgartner, MD, and David D. Yuh, MD

Objective: We evaluated the combined effect of hospital and surgeon volume on operative outcomes of mitral valve surgery in the United States.

Methods: The Nationwide Inpatient Sample was used to identify adult patients undergoing isolated mitral valve surgery for mitral regurgitation from 2003 to 2008. Hospitals and surgeons were separately stratified into equalize tertiles according to annual overall mitral valve operative volumes. Multivariate logistic regression analysis was conducted, adjusting for multiple patient, hospital, and operative data, to determine the separate and combined effects of hospital and surgeon volume on operative outcomes.

Results: A total of 50,152 eligible patients were identified during the study period. Although both hospital and surgeon volume correlated significantly with operative mortality in separate risk-adjusted analyses, only lower surgeon volume persisted as a significant risk factor in the combined risk-adjusted analysis. Moreover, although hospital volume only accounted for 10.7% of the surgeon volume effect on increased mortality for low-volume surgeons, surgeon volume accounted for 74.5% of the hospital volume effect on increased mortality in low-volume hospitals. Surgeon, but not hospital, volume correlated with inpatient costs. Also, significant trends were seen with repair rates, with increasing surgeon volume demonstrating a relatively stronger correlation with the odds of repair ($P < .001$) than hospital volume ($P = .01$).

Conclusions: The effect of hospital volume on operative outcomes of mitral valve surgery was largely driven by the individual surgeon volumes within that hospital. Conversely, surgeon volume affected these outcomes independently of hospital volume. Identifying the processes by which higher volume surgeons attain better outcomes in mitral valve surgery would therefore be prudent. (J Thorac Cardiovasc Surg 2013;146:638-46)
MORTALITY WITH MV SURGERY “REAL WORLD”

**FIGURE 2.** Operative mortality for various combinations of surgeon and hospital volume.

\( p\text{-value} < 0.001 \) for decreasing trend in operative mortality.
QUESTION 1:

- All of the following are possible complications of Mitral valve Repair/Replacement Surgery except:

A) Injury to the bundle of His
B) Left ventricular outflow tract obstruction
C) Left Ventricular dysfunction
D) Injury to the right coronary artery
E) Aortic Regurgitation
F) Rupture of Atrio-Ventricular Groove or left ventricle
QUESTION 1:

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D) Injury to the right coronary artery
E) Aortic Regurgitation
F) Rupture of Atrio-Ventricular Groove or left ventricle
COMPLICATIONS

• 1. Heart Block

• 2. Ventricular Dysfunction

• 3. Systolic Anterior Motion

• 4. Atrioventricular Groove/LV Rupture
ANATOMY: “TIGER COUNTRY”
HEART BLOCK
HEART BLOCK
HEART BLOCK ETIOLOGY
INJURY TO AV NODE/BUNDLE OF HIS
HEART BLOCK ETIOLOGY
INJURY TO SINUS NODE ARTERY

The Clinical Anatomy of the Sinus Node Artery
Denis Berdajs, MD, Lajos Patonay, MD, DD, and Marko I. Turina, MD

Institute of Anatomy, Histology, and Embryology, Laboratory for Applied and Clinical Anatomy, Semmelweis University Budapest, Budapest, Hungary, and Department of Cardiovascular Surgery, University Hospital Zürich, Zürich, Switzerland

Background. Our basic aim was to describe the topographic relation between the sinus node artery and the superior posterior border of the interatrial septum with regard to the sinus node dysfunction that follows the superior transseptal approach to the mitral valve.

Methods. During our study 50 human hearts without previous pathologic alterations were analyzed. The position of the sinus node and the course of the sinus node artery were investigated. For identification of the origin of the artery, selective coronary angiograms were performed. The course of sinus node artery and its topographic relation to the interatrial septum was identified by the dry dissections of the hearts. Based on histologic and dry dissected specimens the exact position of the sinus node was determined.

Results. We found that the sinus node artery originates from the right coronary artery in 66% of examined cases and from the left coronary artery in 34% of cases. The sinus node artery crosses the superior posterior border of the interatrial septum in 54% of cases.

Conclusions. Our results were compared with clinical studies focusing the incidence of the sinus rhythm disturbance after the superior transseptal approach. The incidence of rhythm disturbance varies from 52% to 60% of cases. Comparing our morphologic and clinical results we can state that the risk for intraoperative damage to the sinus node artery during the superior transseptal approach to the mitral valve is high.

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HEART BLOCK ETIOLOGY
INJURY TO SINUS NODE ARTERY

66% RCA Origin

34% LCA Origin

Incidence and pathophysiology of atrioventricular block following mitral valve replacement and ring annuloplasty

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Received 27 August 2007; received in revised form 12 March 2008; accepted 31 March 2008; Available online 15 May 2008

Abstract

Background: In this retrospective study we evaluate the causative mechanisms underlying postoperative atrioventricular block (AVB) following mitral valve replacement and mitral valve annuloplasty. Methods: Between January 1990 and December 2003, 391 patients underwent mitral valve replacement or ring annuloplasty and quadrangular resection. Exclusion criteria were preoperative AV block, two or three valvular procedures, reoperations and procedures combined with coronary artery bypass grafting. The presence of the postoperative AVB was compared with preoperative and intraoperative variables. On 55 post-mortem specimens the relationship between the AV node, AV node artery and mitral valve annulus was investigated. Results: The mean age was 59 ± 14 years and 44% of patients were female. Postoperatively AVB occurred in 92 (23.5%) patients. AVB III was found in 17 (4%) patients, in whom a pacemaker was implanted within median interval of 4 days. Second degree AVB occurred and first degree AVB in five (1.3%) and in 70 (18%) patients respectively. In dry dissected human hearts in 23% of investigated cases the AV node artery was discovered to run close to the annulus of the mitral valve. Conclusions: Data collected in this study showed that, sotalol and amiodarone as well as a prolonged cross-clamp time may slightly influence the 23% incidence of postoperative AVB. The morphological investigation showed that the AV node artery runs in close proximity to the annulus in 23% of cases. We speculate that damage of the AV node artery may play a role in development of AVB.

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Keywords: Mitral valve surgery; Mitral valve reconstruction; Postoperative rhythm disturbances; Atrioventricular block; Heart conducting system
Incidence and pathophysiology of atrioventricular block following mitral valve replacement and ring annuloplasty.*

Conduction disturbances as compared following mitral valve replacement or mitral valve annuloplasty

<table>
<thead>
<tr>
<th>Conduction disturbances</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVB I</td>
<td>70 (17.9%)</td>
</tr>
<tr>
<td>AVB II</td>
<td>5 (1.5%)</td>
</tr>
<tr>
<td>AVB III</td>
<td>17 (4.3%)</td>
</tr>
<tr>
<td>Right bundle branch block</td>
<td>18 (4.6%)</td>
</tr>
<tr>
<td>Left bundle branch block</td>
<td>10 (2.6%)</td>
</tr>
<tr>
<td>Left anterior hemi block</td>
<td>30 (7.7%)</td>
</tr>
<tr>
<td>Total</td>
<td>150 (38.3%)</td>
</tr>
</tbody>
</table>

Conduction disturbances after mitral valve replacement or after mitral valve annuloplasty (n = 391).

Berdajs et al. EJCTS 2008;34:55-61
Predictors of Permanent Pacemaker Implantation
during the Early Postoperative Period after Valve Surgery

Maqsood M. Elahi, MD
Darren Lee, MD
Ramana Rao V.
Dhannapuneni, MD

The ability to preoperatively identify patients who may require permanent pacemaker implantation is rather poorly understood. The aim of this study is to determine the current incidence of permanent pacing after valve surgery and to determine which factors place the heart valve patient at risk of requiring permanent pacemaker implantation. We audited the records of 2,392 consecutive adult patients who underwent cardiac valve surgical procedures by the same surgical team from 25 April 1998 through 31 March 2003. Of these, 118 patients (group A) required the postoperative implantation of permanent pacemakers during the same hospitalization; they were compared with 1,959 heart valve patients (group B) who did not require pacemaker placement. Multivariate logistic regression analysis showed that reoperations (odds ratio [OR], 8.23; P <0.001), longer cumulative cross-clamp times (OR, 5.9; P <0.001), multiple-valve surgical procedures (OR, 3.46; P <0.05), and absence of preoperative sinus rhythm (OR 2.52; P <0.001) were independent predictors of the need for permanent pacemaker implantation after valve surgery. These results suggest that patients who display these risk factors for arrhythmias that require permanent pacemaker implantation receive closer observation and advance counseling about the likelihood of such implantation. (Tex Heart Inst J 2006;33:455-7)
HEART BLOCK

Predictors of Permanent Pacemaker Implantation during the Early Postoperative Period after Valve Surgery

Maqsood M. Elahi, MD
Darren Lee, MD
Ramana Rao V. Dhannapuneni, MD

<table>
<thead>
<tr>
<th>Variables</th>
<th>Odds Ratio</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reoperation</td>
<td>8.23</td>
<td>0.001</td>
</tr>
<tr>
<td>Cross-clamp time (min)</td>
<td>5.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Multiple-valve surgery</td>
<td>3.46</td>
<td>0.024</td>
</tr>
<tr>
<td>Absence of sinus rhythm</td>
<td>2.52</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Maqsood et al. Tex Heart J 2006;33:455-7
Permanent Cardiac Pacing After a Cardiac Operation: Predicting the Use of Permanent Pacemakers

Richard S. Gordon, BSc, Joan Ivanov, MSc, Gideon Cohen, MD, and Anthony L. Ralph-Edwards, MD
Division of Cardiovascular Surgery, The Toronto Hospital, Toronto, Ontario, Canada

Background. The need for permanent cardiac pacing after cardiac operations is infrequent but associated with increased morbidity and resource utilization. We identified patient risk factors for pacemaker insertion to enable development of a predictive model.

Methods. Data were collected prospectively for 10,421 consecutive patients who had cardiac operations between January 1990 and December 1995. Two hundred fifty-five patients (2.4%) were identified as having received a permanent pacemaker during the same hospitalization. Logistic regression analysis was performed to determine the independent, multivariate predictors of permanent pacing. The predictive accuracy and precision of the logistic regression model was evaluated in the 1996 database of 2,236 consecutive patients by the calculation of Brier scores.

Results. Eight independent predictors of permanent pacemaker requirement were identified. The factor-adjusted odds ratios (OR) with 95% confidence interval (CI) associated with each predictor are as follows: (1) valve replacement surgery (aortic: OR 5.8, CI 3.9–8.7; mitral: OR 4.9, CI 3.1–7.8; tricuspid: OR 8.0, CI 5.5–11.9; double: OR 8.9, CI 5.5–14.6; and triple: OR 7.5, CI 2.9–19.3); (2) repeat operation: OR 2.4, CI 1.8–3.3; (3) age 75 years or older: OR 3.0, CI 2.0–4.4; (4) ablative arrhythmia operation: OR 4.2, CI 1.9–9.5; (5) mitral valve annular reconstruction: OR 2.4, CI 1.4–4.2; (6) use of cold blood cardioplegia: OR 2.0, CI 1.2–3.6; (7) preoperative renal failure: OR 1.6, CI 1.0–2.6; and (8) active endocarditis: OR 1.7, CI 0.9–3.0. A model for postoperative permanent pacemaker requirement using the eight predictors was formulated and tested (Brier score = 0.017 ± 0.003; Z = 0.18).

Conclusions. The proposed predictive model correlated highly with actual pacemaker use, which suggests that the requirement for pacing results from either operative trauma or increased ischemic burden. Preoperative identification of patients at increased risk of conduction disturbances may allow for earlier detection and improved treatment. Patients requiring postoperative pacing had increased morbidity and length of stay.

Table 4. Multivariate Analysis for Operating Room Pacing

<table>
<thead>
<tr>
<th>Variable</th>
<th>Regression Coefficient</th>
<th>SE</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-5.3570</td>
<td>0.170</td>
<td>0.005</td>
<td>0.004–0.007</td>
</tr>
<tr>
<td>Procedure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVR</td>
<td>1.7590</td>
<td>0.204</td>
<td>5.81</td>
<td>3.90–8.66</td>
</tr>
<tr>
<td>MVR</td>
<td>1.5870</td>
<td>0.238</td>
<td>4.89</td>
<td>3.07–7.79</td>
</tr>
<tr>
<td>TVR</td>
<td>2.0870</td>
<td>0.199</td>
<td>8.06</td>
<td>5.46–11.9</td>
</tr>
<tr>
<td>Double</td>
<td>2.1870</td>
<td>0.251</td>
<td>8.91</td>
<td>5.45–14.6</td>
</tr>
<tr>
<td>Triple</td>
<td>2.0100</td>
<td>0.486</td>
<td>7.46</td>
<td>2.88–19.3</td>
</tr>
<tr>
<td>Reoperation</td>
<td>0.8822</td>
<td>0.156</td>
<td>2.42</td>
<td>1.78–3.28</td>
</tr>
<tr>
<td>Age (y)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65–74</td>
<td>0.6161</td>
<td>0.151</td>
<td>1.85</td>
<td>1.38–2.49</td>
</tr>
<tr>
<td>≥75</td>
<td>1.0960</td>
<td>0.194</td>
<td>2.99</td>
<td>2.04–4.38</td>
</tr>
<tr>
<td>Ablative arrhythmia operation</td>
<td>1.4410</td>
<td>0.414</td>
<td>4.22</td>
<td>1.87–9.52</td>
</tr>
<tr>
<td>MV annulus reconstruction</td>
<td>0.8606</td>
<td>0.287</td>
<td>2.36</td>
<td>1.35–4.15</td>
</tr>
<tr>
<td>Cold blood cardioplegia</td>
<td>0.7168</td>
<td>0.292</td>
<td>2.05</td>
<td>1.16–3.63</td>
</tr>
<tr>
<td>Preoperative renal failure</td>
<td>0.4802</td>
<td>0.232</td>
<td>1.62</td>
<td>1.03–2.55</td>
</tr>
<tr>
<td>Active endocarditis</td>
<td>0.5080</td>
<td>0.303</td>
<td>1.66</td>
<td>0.918–3.01</td>
</tr>
</tbody>
</table>

OR 7.5, CI 2.9–8.3; (3) age 75+; (4) valve annular se of cold blood cooperative renal endocarditis; OR for permanent pacemakers was 3.17 ± 0.003; Z = model correlated with blood pressure and congestive heart failure. The model suggests that either operative or nonoperative identification of conduction block and indeterminate pacemaker status was important.  

AVR = aortic valve replacement/repair; Double = two valve operation; GOF = goodness of fit; MV = mitral valve; MVR = mitral valve replacement or repair; OR = odds ratio; SE = standard error; Triple = three valve operations; TVR = tricuspid valve replacement or repair.

Richard S. C. Gordon
Division of Cardiovascular Surgery

Background. After cardiac open heart surgery, increased mortality and morbidity can be expected due to the development of ventricular tachyarrhythmias. The purpose of the study was to determine the independent risk factors for ventricular tachyarrhythmias and to identify the predictive factors for the development of ventricular tachyarrhythmias. Methods. Data were collected on consecutive patients who underwent open heart surgery at the University of California, San Francisco, between January 1990 and December 1992. Logistic regression was used to determine the independent risk factors for ventricular tachyarrhythmias. The results of the logistic regression analysis were used to calculate the odds ratios and 95% confidence intervals. Results. Eight significant risk factors were identified: age, gender, preoperative renal function, preoperative endocarditis, active endocarditis, AV block, ventricular tachyarrhythmias, and MV annulus reconstruction. AV block was associated with a OR of 4.9, 95% CI: 1.9–12.9.

Regression Coefficient SE OR 95% CI
Constant -5.3570 0.170 0.005 0.004–0.007
AVR 1.7590 0.204 5.81 3.90–8.66
MVR 1.5870 0.238 4.89 3.07–7.79
TVR 2.0870 0.199 8.06 5.46–11.9
Double 2.1870 0.251 8.91 5.45–14.6
Triple 2.0100 0.486 7.46 2.88–19.3
Reoperation 0.8822 0.156 2.42 1.78–3.28
Age (y) 0.6161 0.151 1.85 1.38–2.49
≥75 1.0960 0.194 2.99 2.04–4.38
Ablative arrhythmia operation 1.4410 0.414 4.22 1.87–9.52
MV annulus reconstruction 0.8606 0.287 2.36 1.35–4.15
Cold blood cardioplegia 0.7168 0.292 2.05 1.16–3.63
Preoperative renal failure 0.4802 0.232 1.62 1.03–2.55
Active endocarditis 0.5080 0.303 1.66 0.918–3.01

AVR = aortic valve replacement/repair; Double = two valve operation; GOF = goodness of fit; MV = mitral valve; MVR = mitral valve replacement or repair; OR = odds ratio; SE = standard error; Triple = three valve operations; TVR = tricuspid valve replacement or repair.
VENTRICULAR DYSFUNCTION
SURGICAL CONSIDERATIONS FOR LV DYSFUNCTION

1. Myocardial Protection
2. Cardiopulmonary bypass
3. Coronary Anatomy
MYOCARDIAL PROTECTION: THE ROUTE

- Antegrade vs. Retrograde
MYOCARDIAL PROTECTION: THE ROUTE

- Antegrade:
- Requirements:
  1. Competent Aortic Valve
  2. No high grade stenoses in L/R main coronary arteries
MYOCARDIAL PROTECTION: 
THE ROUTE

• Retrograde:
• Requirements:
  1. You can get the catheter in the coronary sinus
  2. No unrecognized Left SVC

Always Confirm its Efficacy!
CARDIOPULMONARY BYPASS:

- Key elements to protect heart
  1. Mild hypothermia
  2. Minimizing blood return to heart
Right Coronary Artery Injury After Tricuspid Valve Repair

Robin Varghese, MD, MS, FRCSC, Adanna Akujuo, MD, and David H. Adams, MD

Tricuspid valve repair can result in right coronary artery injury secondary to valve annuloplasty. We report a case in which a patient developed right coronary artery occlusion because of tricuspid valve repair and review management options. An 83-year-old gentleman with a past medical history of mitral valve prolapse developed Class III symptoms of congestive heart failure in addition to atrial fibrillation and pulmonary hypertension. Transthoracic echocardiogram revealed preserved ventricular function and severe mitral regurgitation secondary to annular dilation with minimally restricted leaflet motion. In addition, moderate tricuspid regurgitation was evident secondary to annular dilation. Preoperatively, the intrinsic cardiac rhythm was a slow junctional escape at 35 beats/min; therefore, the patient was ventricular paced at 90 beats/min. The chest was closed without issue. The patient remained on low-dose vasopressin and epinephrine. During transport of the patient from the operating room to the intensive care unit, new 3-mm ST-segment elevation was noted in lead II of the electrocardiogram. On arrival in the intensive care unit, a 12-lead electrocardiogram revealed 5-mm ST-segment elevation in leads II, III, and avF, and a transthoracic echocardiogram demonstrated a hypovascular inferior wall. The patient was taken to the cardiac catheterization suite for urgent left heart catheterization.
Surgical Considerations

Coronary Anatomy

David H. Adams, MD

Myth was a slow junctional; therefore, the patient was 110 beats/min. The chest x-ray showed no abnormalities. During transport to the operating room, the patient’s ECG showed ST-segment elevation in leads II, III, and aVF. An echocardiogram confirmed hypokinetic inferior wall. The cardiac catheterization suite was not available.
CAN THE LV DECLINE DESPITE GOOD SURGERY?
Determinants of early decline in ejection fraction after surgical correction of mitral regurgitation

Rakesh M. Suri, MD, DPhil, Hartzell V. Schaff, MD, Joseph A. Dearani, MD, Thoralf M. Sundt III, MD, Richard C. Daly, MD, Charles J. Mullany, MB, MS, Maurice E. Sarano, MD, and Thomas A. Orszulak, MD

**Objective:** We sought to echocardiographically examine the early changes in left ventricular size and function after mitral valve repair or replacement for mitral regurgitation caused by leaflet prolapse.

**Methods:** Preoperative and early postoperative echocardiograms of 861 patients with mitral regurgitation caused by leaflet prolapse who underwent mitral valve repair or replacement (with or without coronary revascularization) were studied. Among the patients, 625 (73%) were men and 779 (90%) had mitral valve repair.

**Results:** The rate of valve repair increased from 78% in the first decade of the study to 92% in the second decade. At early echocardiography (mean, 5 days postoperatively), we observed significant decreases in left ventricular ejection fraction (mean, −8.8) and left ventricular end-diastolic dimension (mean, −7.5). The magnitude of the early decline in ejection fraction was similar in patients who had mitral valve repair and replacement. The decrease in postoperative ejection fraction was independently associated with a lower preoperative ejection fraction, the presence of atrial fibrillation, advanced New York Heart Association functional class, greater left ventricular end-diastolic and end-systolic dimensions, and larger left atrial size.
DOES IT HAPPEN?

Determinants of early decline in ejection fraction after surgical correction of mitral regurgitation

Rakesh M. Suri, MD, RTI Health Solutions, Raleigh, North Carolina; Herting M. Dietz, MB, BS, MD, RTI Health Solutions, Raleigh, North Carolina; Christian C. Colan, MD, PhD, RTI Health Solutions, Raleigh, North Carolina; Edward D. Kim, MD, RTI Health Solutions, Raleigh, North Carolina; Richard C. D'Cunha, MD, PhD, RTI Health Solutions, Raleigh, North Carolina; and Edward R. Lawrence, MD, PhD, RTI Health Solutions, Raleigh, North Carolina


TABLE 2. Echocardiographic changes in the early postoperative period

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preoperative*</th>
<th>Postoperative*</th>
<th>Overall change*</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF, %</td>
<td>62.9 ± 9.9</td>
<td>53.1 ± 11.3</td>
<td>-8.8 ± 10.7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>69.9 ± 7.6</td>
<td>53.1 ± 7.7</td>
<td>-7.5 ± 7.3</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVESD, mm</td>
<td>36.9 ± 7.1</td>
<td>36.6 ± 8.2</td>
<td>-0.5 ± 6.5</td>
<td>.26</td>
</tr>
<tr>
<td>Left atrial size, mm</td>
<td>52.3 ± 9.2</td>
<td>48.0 ± 8.7</td>
<td>-5.0 ± 7.4</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

LVEDD, Left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension. *Expressed as mean ± standard deviation.

62% 53% 9% DECLINE
DOES IT HAPPEN?


### TABLE 1. Baseline demographic data

<table>
<thead>
<tr>
<th>Variable</th>
<th>All patients (n = 632)</th>
<th>No (n = 390)</th>
<th>Yes (n = 242)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics and comorbidity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>57.0 ± 13.2</td>
<td>56.0 ± 13.1</td>
<td>58.7 ± 8.1</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Female gender</td>
<td>229 (36.2)</td>
<td>148 (37.9)</td>
<td>81 (33.5)</td>
<td>.255</td>
</tr>
<tr>
<td>Body surface area (m²)</td>
<td>1.90 ± 0.25</td>
<td>1.89 ± 0.24</td>
<td>1.92 ± 0.25</td>
<td>.193</td>
</tr>
<tr>
<td>Hypertension</td>
<td>290 (45.9)</td>
<td>179 (45.9)</td>
<td>111 (45.9)</td>
<td>.994</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>163 (25.8)</td>
<td>105 (26.9)</td>
<td>58 (24.0)</td>
<td>.409</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>20 (3.2)</td>
<td>8 (2.1)</td>
<td>12 (5.0)</td>
<td>.042</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>74 (11.7)</td>
<td>38 (9.7)</td>
<td>36 (14.9)</td>
<td>.051</td>
</tr>
<tr>
<td>Obstructive lung disease</td>
<td>19 (2.9)</td>
<td>1 (0.8)</td>
<td>4 (2.9)</td>
<td>.234</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>7 (1.1)</td>
<td>6 (1.5)</td>
<td>1 (0.4)</td>
<td>.288</td>
</tr>
<tr>
<td>Renal failure</td>
<td>2 (0.3)</td>
<td>2 (0.5)</td>
<td>0 (0)</td>
<td>.260</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
<td>14 (2.2)</td>
<td>6 (1.8)</td>
<td>8 (3.3)</td>
<td>.225</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>366 (57.9)</td>
<td>228 (58.5)</td>
<td>138 (57.0)</td>
<td>.722</td>
</tr>
<tr>
<td><strong>Preoperative echocardiographic parameters</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>60.7 ± 7.5</td>
<td>61.5 ± 6.7</td>
<td>59.3 ± 8.4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVEF &lt; 50%</td>
<td>65 (10.3)</td>
<td>28 (7.2)</td>
<td>37 (15.3)</td>
<td>.001</td>
</tr>
<tr>
<td>LVESE (mm)</td>
<td>35.8 ± 6.8</td>
<td>34.6 ± 6.2</td>
<td>37.8 ± 7.3</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVEDD &gt; 40 mm</td>
<td>159 (25.2)</td>
<td>74 (19.0)</td>
<td>85 (35.1)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PA systolic pressure (mm Hg)</td>
<td>34.6 ± 13.3</td>
<td>32.3 ± 11.6</td>
<td>38.0 ± 14.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>56.0 ± 7.9</td>
<td>54.9 ± 7.4</td>
<td>58.0 ± 8.1</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PASP &gt; 50 mm Hg</td>
<td>82 (13.0)</td>
<td>39 (10.0)</td>
<td>43 (17.8)</td>
<td>.005</td>
</tr>
</tbody>
</table>
Does it matter?

Echocardiographic Prediction of Left Ventricular Function After Correction of Mitral Regurgitation: Results and Clinical Implications

Figure 1. Survival after postoperative echocardiography according to postoperative ejection fraction (EF). The two rows of numbers at the bottom of the graph are the number of patients at each interval.

- Postoperative EF ≥ 50%: 69% (8 ± 5% survival at 5 years)
- Postoperative EF < 50%: 38% (38% survival at 5 years)

Enriquez-Sarano et al. JACC. 1994;24:1536-43
Predicting early left ventricular dysfunction after mitral valve reconstruction: The effect of atrial fibrillation and pulmonary hypertension

Robin Varghese, MD, MS, FRCSC, Shinobu Itagaki, MD, Anelechi C. Anyanwu, MD, MSc, FRCS, Federico Millia, MD, and David H. Adams, MD

Objectives: The preoperative ejection fraction (EF) and left ventricular (LV) end-systolic dimension are known predictors of postoperative LV dysfunction after mitral valve repair. We investigated the effect of a preoperative history of atrial fibrillation and moderate pulmonary hypertension (defined as pulmonary artery systolic pressure >50 mm Hg) on early postoperative LV dysfunction.

Methods: From 2003 to 2010, 632 patients who had undergone successful mitral valve repair surgery for degenerative disease were included in the present study. The preoperative and postoperative echocardiographic data and postoperative outcomes were collected retrospectively. We analyzed the demographic, hemodynamic, and echocardiographic parameters to assess the predictors of early postoperative LV dysfunction, defined as an LVEF <50%.

Results: The mean age of the cohort was 57 ± 13 years. All patients had less than mild mitral regurgitation on postoperative echocardiography. After mitral valve repair, a significant decrease in the LVEF (60% ± 8% to 54% ± 9%), LV end-systolic diameter (36 ± 7 mm to 33 ± 7 mm), and LV end-diastolic dimension (56 ± 8 mm to 48 ± 7 mm) was observed at early postoperative echocardiography (P < .001). On multivariate regression analysis, preoperative atrial fibrillation, pulmonary hypertension, and LV end-systolic dimension were independent predictors of the postoperative LVEF (P = .035 and P < .001, respectively). Preoperative atrial fibrillation (odds ratio, 1.97; 95% confidence interval, 1.28-3.02; P = .002) and pulmonary artery systolic pressure >50 mm Hg (odds ratio, 1.82; 95% confidence interval, 1.11-2.97; P = .017) increased the risk of postoperative LV dysfunction by almost twofold.

Conclusions: In addition to the established predictors of postoperative LV dysfunction, the presence of preoperative pulmonary hypertension and a history of atrial fibrillation in patients undergoing mitral valve repair surgery increased the risk of early postoperative LV dysfunction by almost twofold. (J Thorac Cardiovasc Surg 2014;148:422-7)
LV DYSFUNCTION RISK STRATIFICATION

Predicting early left ventricular dysfunction after mitral valve reconstruction: The effect of atrial fibrillation and pulmonary hypertension

TABLE 3. Predictors of postoperative left ventricular ejection fraction (continuous variables)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate</th>
<th>Multivariate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>-0.092</td>
<td>.021</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>0.209</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>ESD (mm)</td>
<td>-0.337</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PASP (mm Hg)</td>
<td>-0.205</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Left atrial diameter (mm)</td>
<td>-0.185</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Crossclamp time (min)</td>
<td>-0.006</td>
<td>.652</td>
</tr>
</tbody>
</table>

TABLE 4. Predictors of postoperative left ventricular dysfunction (categorical variables)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate</th>
<th>Multivariate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>1.02 (1.00-1.03)</td>
<td>.014</td>
</tr>
<tr>
<td>Female gender</td>
<td>0.82 (0.59-1.15)</td>
<td>.255</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>0.94 (0.68-1.30)</td>
<td>.722</td>
</tr>
<tr>
<td>History of AF</td>
<td>2.49 (1.66-3.75)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PASP &gt; 50 mm Hg</td>
<td>1.95 (1.22-3.10)</td>
<td>.005</td>
</tr>
<tr>
<td>Concomitant CABG</td>
<td>1.57 (0.91-2.70)</td>
<td>.105</td>
</tr>
<tr>
<td>Concomitant TVR</td>
<td>1.19 (0.85-1.67)</td>
<td>.314</td>
</tr>
</tbody>
</table>

In the final model, Hosmer-Lemeshow = 0.814; c-statistic = 0.680. OR, Odds ratio; CI, confidence interval; AF, atrial fibrillation; PASP, pulmonary artery systolic pressure; CABG, coronary artery bypass grafting; TVR, tricuspid valve repair.

Conclusion: In addition to the established predictors of postoperative LV dysfunction, the presence of preoperative pulmonary hypertension and a history of atrial fibrillation in patients undergoing mitral valve repair surgery increased the risk of early postoperative LV dysfunction by almost twofold. (J Thorac Cardiovasc Surg 2014;148:422-7)
Predicting early left ventricular dysfunction after mitral valve reconstruction: The effect of atrial fibrillation and pulmonary hypertension

Robin Varghese, MD, MS, FRCSC, Shinobu Itagaki, MD, Anelechi C. Anyanwu, MD, MSc, FRCS, Federico Milla, MD, and David H. Adams, MD

**TABLE 5. Prevalence of postoperative LV dysfunction according to presence of preoperative guideline parameters**

<table>
<thead>
<tr>
<th>Preoperative guideline parameters</th>
<th>Postoperative LV dysfunction (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF &lt; 60% only</td>
<td>26/76 (34.2)</td>
</tr>
<tr>
<td>LVEF &lt; 60% + ESD &gt; 40 mm</td>
<td>26/54 (48.1)</td>
</tr>
<tr>
<td>LVEF &lt; 60% + ESD &gt; 40 mm + AF or PHT</td>
<td>17/24 (70.8)</td>
</tr>
<tr>
<td>LVEF &lt; 60% + ESD &gt; 40 mm + AF + PHT</td>
<td>4/6 (66.7)</td>
</tr>
</tbody>
</table>

Data in parentheses are percentages. $P = .010$ for all. LV, Left ventricular; LVEF, left ventricular ejection fraction; ESD, end-systolic diameter; AF, atrial fibrillation; PHT, pulmonary hypertension.

Preoperative pulmonary hypertension and a history of atrial fibrillation in patients undergoing mitral valve repair surgery increased the risk of early postoperative LV dysfunction by almost twofold. (J Thorac Cardiovasc Surg 2014;148:422-7)
ATRIOVENTRICULAR GROOVE & VENTRICULAR RUPTURE
FIBROUS SKELETON

Heart in diastole: viewed from base with atria removed
AV GROOVE
QUESTION 2:

After elective Mitral valve Repair, in an active 75 yo female with normal EF, excessive bleeding is noted when weaning from cardiopulmonary bypass. A rupture of the atrioventricular groove is suspected. The most appropriate plan of action is:

- A) Lift the heart up to identify the site of bleeding and place pledgeted suture around the tear.
- B) Reapply the cross clamp, open the left atrium, and attempt repair through the annuloplasty ring.
- C) Reapply the cross clamp, open the left atrium remove the ring, patch the tear with pericardium and replace the annuloplasty ring back.
- D) Reapply the cross clamp, open the left atrium remove the ring, patch the tear with pericardium and replace the valve with a bioprosthetic valve.
QUESTION 2:

After elective Mitral valve Repair, in an active 75 yo female with normal EF, excessive bleeding is noted when weaning from cardiopulmonary bypass. A rupture of the atrioventricular groove is suspected. The most appropriate plan of action is:

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D) Reapply the cross clamp, open the left atrium remove the ring, patch the tear with pericardium and replace the valve with a bioprosthetic valve
TYPES OF LV RUPTURE

- **Type I**: At the Atrioventricular Groove
  - Calcified posterior annulus
  - Elderly female patient
  - Posterior annular abscess
  - Lifting of the heart after MV prosthesis in place
TYPES OF LV RUPTURE

• **Type 2:** At the base of the Papillary Muscles:
  - Excessive resection of posterior papillary muscle
  - More common in rheumatic disease with calcified chordae
TYPES OF LV RUPTURE

- **Type 3**: Posterior LV wall between the annulus and Papillary Muscles:
  - Incorrect valve replacement suture placement deep in ventricle
  - Injury from valve strut of bioprosthetic valve (immediate or delayed)
DIAGNOSIS?

- Bleeding from the posterior pericardial well.....and lots of it.
- Difficult to identify exact location of tear
- Lifting heart aggressively will make it worse
REPAIR OF THE AV GROOVE

• 2 OPTIONS:

• External Repair
  • From lateral wall of heart

• Internal Repair
  • Within left atrium
MANAGEMENT: EXTERNAL REPAIR

• External suture repair is Difficult:
  • Easy to worsen the tear as the heart is lifted up
  • Difficult to pinpoint the source of tear
  • Injury to circumflex more common

Ann Thorac Sx 1998;46:491-4
A New Technique for Repair of Atrioventricular Disruption Complicating Mitral Valve Replacement

Rosauro Mejia, MBBS, and Duncan S. Thomson, MD

Department of Cardiothoracic Surgery, John Hunter Hospital, Newcastle, New South Wales, Australia

Atrioventricular disruption is an uncommon but often lethal complication in mitral valve replacement. We present the case of a 79-year-old woman in whom the disruption after mitral valve replacement was successfully repaired using BioGlue surgical adhesive, bovine pericardium, and polytetrafluoroethylene (Teflon) patch. (Ann Thorac Surg 2003;75:1973–4) © 2003 by The Society of Thoracic Surgeons

In severe mitral annulus calcification, mitral valve replacement can be complicated by rupture or dehiscence of the posterior atrioventricular groove. Atrioventricular disruption is a difficult and major complication of mitral valve replacement. This condition occurs rarely (1% to 2%) but is usually fatal with an operative mortality rate of 50% [1, 2].

The patient was placed back onto bypass and cooled, and the heart was perfused with cardioplegic solution. BioGlue was then applied to the external surface of the dehisced margins of the atrioventricular groove, and a patch of bovine pericardium was placed over the glue before it set, with a total circulatory arrest time of 2 minutes to ensure a dry field.

The circulation was then restarted, but further bleeding complicated attempted weaning from cardiopulmonary bypass. Therefore, the pericardium was removed, and 2-0 Ticron polytetrafluoroethylene (Teflon) bolstered sutures were used to attempt to close the defect. However, the sutures themselves caused additional bleeding from the damaged tissues. Consequently, a further attempt to achieve hemostasis with the BioGlue surgical adhesive was performed with the heart arrested and perfused with cardioplegic solution again.

The BioGlue was applied under and over the Teflon-pledged sutures, and another patch of Teflon felt was glued on top of the Teflon-pledged sutures. It was held firmly in place for 2 minutes to achieve good adhesion, and then the aortic cross-clamp was released. Fortunately, this second attempt resulted in a dry operative field with complete control of the bleeding. The patient spontaneously went into sinus rhythm after the cross-clamp was released, and bypass was withdrawn without inotropic support. The patient was deliberately kept hypotensive overnight with a mean blood pressure less than 70 mm Hg. She then made an uneventful recovery. Before discharge an echocardiograph showed no periavalvar leak from either the mitral or aortic valve.
MANAGEMENT: INTERNAL REPAIR

1. Reapply Cross clamp and arrest heart
2. Remove prosthesis/ring
3. Identify site of injury (type I, 2, 3).
4. Patch repair is usually required
5. Replacement of valve usually with a smaller prosthesis
Rupture of the Posterior Wall of the Left Ventricle after Mitral Valve Replacement: Etiological and Technical Considerations

Mike Azariades, M.D., and Stuart C. Lennox, F.R.C.S.

ABSTRACT Rupture of the posterior wall of the left ventricle after mitral valve replacement, although infrequent, may be a highly lethal complication. Controversy exists regarding the etiology of this complication. Suggested causative factors include the type and extent of the valvular disease, type and size of the prosthesis, and the surgical techniques used.

Our experience over a 20-year period includes 10 patients with rupture of the left ventricle following mitral valve replacement. In all patients, both mitral leaflets were excised together with the attached chordae. Three patients survived after repair of the rupture. Repair consisted of compressing the area between the left atrium and the base of the papillary muscle using two strips of Teflon and deep mattress sutures passed beneath the coronary vessels in the atrioventricular groove.

Since 1983 we have routinely preserved the posterior leaflet of the mitral valve with its attached chordae to maintain a “tethered loop” between the mitral valve and ventricle. No further ruptures have occurred. The technique used for repair represents reconstitution of the divided loop between the ventricle and the mitral valve.

Despite overall improvement in the results of valve repair, the midpor the papillary the atrioventricular annulus; (2) leading to tension of the bioprosthesis; (3) excision of para-nuluses and "tions; (4) the ventricles stretching; and (11) the muscle and the anatomy posterior left ventricular rupture.

We repaired wall of the with an em

Fig 3. Lateral view of repair of posterior left ventricular rupture. (LV = left ventricle; LA = left atrium; RV = right ventricle; RA = right atrium; Ao = aorta.)
Endoventricular Pocket Repair of Type I Myocardial Rupture After Mitral Valve Replacement: A New Technique Using Pericardial Patch, Teflon Felt, and Bioglu

Saqib Masroor, MD, John Schor, MD, Roger Carrillo, MD, and Donald B. Williams, MD
Mount Sinai Medical Center, Miami Beach, and University of Miami/Jackson Memorial Hospital, Miami Beach, Florida

Left ventricular (LV) rupture is an infrequent but potentially lethal complication of mitral valve replacement and repair. We report the case of an 82-year-old man who underwent mitral valve replacement and the repair of an atrial septal defect. Both valve leaflets were excised and the annulus was extensively decalcified, followed by the implantation of a bioprosthetic valve. LV rupture was diagnosed after weaning from cardiopulmonary bypass (CPB). CPB was resumed and the bioprosthetic valve was removed. The patient then underwent a unique repair using a pericardial patch, Teflon felt (Meadox Medical Inc, Oakland, NJ), and BioGlue (Cryolife Inc, Kennesaw, GA). A second valve was implanted with a successful outcome.


Fig 3. Cross-section through the atriocavitricular groove at the repair site. The valve stiches go through the pericardial pocket. (Teflon felt: Meadox Medical Inc, Oakland, NJ; BioGlue: Cryolife Inc, Kennesaw, GA.)
Autotransplantation of Heart for Repair of Left Ventricular Rupture After Mitral Valve Replacement

J. Wei, C. Wu, G. Hong, D.Y. Tung, C.Y. Chang, and Y.C. Chuang

THREE types of left ventricular rupture following mitral valve replacement have been described.\textsuperscript{1,2} However, attempts at repair are difficult. According to a collective review\textsuperscript{3} of a large series of mitral valve replacement at 10 institutions, the incidence of left ventricular rupture after mitral valve replacement varied from 0.5\%\textsuperscript{4} to 14\%,\textsuperscript{5} with an average of 1.2\%. The mortality rate of this complication was 75\% or higher, and it is responsible for 18\% of all deaths from mitral valve replacement.\textsuperscript{3} 

operation. One and a half months later, the patient complained of dizziness and EKG showed a ventricular rate of 50/min. A VDD permanent pacemaker (Medtronic Inc, Minneapolis, Minn) was implanted on February 1, 1999. Follow-up echocardiogram on June 10, 2000. 7.5 months postoperatively, showed a well-functioning heart with aortic valve pressure gradient of 35 mm Hg, mitral valve area of 3.2 cm\textsuperscript{2}, LVIDd 29 mm, LVIDd 47 mm, mild aortic regurgitation, and no mitral regurgitation.

Case 2
OUTCOMES

- Mortality estimated at 50-75%
- Negative results often not published
WHY ARE OUTCOMES POOR

1. ACCESS – Difficult to place sutures deep in LV for patch
2. LV muscle often weak tissue and can tear
3. Once heart starts beating and LV fills sutures can tear
4. Injury to circumflex coronary artery
5. Complex repair leading to long cross clamp times and CPB times.
6. Patients are often elderly
SYSTOLIC ANTERIOR MOTION
Simply put,

A mismatch between the mitral valve annular dimension and the leaflet tissue.
Mechanism of Outflow Tract Obstruction Causing Failed Mitral Valve Repair
Anterior Displacement of Leaflet Coaptation

Kamthorn S. Lee, MD; William J. Stewart, MD; Harry M. Lever, MD; Paul L. Underwood, MD; Delos M. Cosgrove, MD

Background. Systolic anterior motion of the mitral valve causing left ventricular outflow tract obstruction occurs in 1% to 2% of patients having mitral valve repair, in some cases requiring further surgery to relieve the obstruction, but the mechanism and the geometry involved are not certain.

Methods and Results. We studied 14 patients who developed systolic anterior motion and left ventricular outflow tract obstruction, all after posterior leaflet resection and annuloplasty, in whom a second repair eliminated systolic anterior motion by complete (n=6) or partial (n=8) ring removal. Intraoperative transesophageal echocardiography was recorded before pump, after failed repair during left ventricular outflow tract obstruction, and after a corrective second pump run to relieve the systolic anterior motion. Systolic anterior motion occurred when the mitral valve coaptation to septum distance was reduced (before, 26.5±4.3; during systolic anterior motion, 17.4±4.4 versus after second pump, 23.4±6.9 mm) and the mitral valve coaptation to posterior mitral annulus distance was greater (before, 18.9±3.4; during systolic anterior motion, 22.2±4.6 versus after second pump, 17.4±3.6 mm), both P<.01. Comparing dimensions before pump, during systolic anterior motion, and after the second pump, there were no differences in left ventricular cavity diameter in systole or diastole, the septum to posterior annulus distance, or the angle between the aortic and mitral annular planes.

Conclusions. After mitral repair, left ventricular outflow tract obstruction occurs when the mitral coaptation line is displaced anteriorly. When systolic anterior motion occurs, reduction of the amount of annuloplasty or use of the posterior leaflet sliding procedure may eliminate this problem. Understanding the geometry of this phenomenon may facilitate preoperative echo selection of high-risk patients (those with large redundant posterior leaflets and relatively normal ventricular size) and modification of surgical technique to avoid the problem of outflow tract obstruction after mitral valve repair. (Circulation. 1993;88[part 2]:24-29.)

KEY WORDS • mitral valve • echocardiography • valvuloplasty

Fig 3. Diagrams show positions of mitral coaptation during stages of surgery. Left, Before repair: Mitral valve (MV) with flail. Ao indicates aorta; LA, left atrium; and LV, left ventricle. Middle, During systolic anterior motion (SAM). Right, After second repair.
Obstructive Hypertrophic Cardiomyopathy: Echocardiography, Pathophysiology, and the Continuing Evolution of Surgery for Obstruction

Mark V. Sherrid, MD, Farooq A. Chaudhry, MD, and Daniel G. Swistel, MD
Divisions of Cardiology and Cardiovascular and Thoracic Surgery, St. Luke’s-Roosevelt Hospital Center, Columbia University, College of Physicians and Surgeons, New York, New York

Our understanding of the pathophysiology of obstruction in hypertrophic cardiomyopathy has evolved since initial descriptions in the late 1950s. This review addresses the cause of obstruction, from early ideas that a muscular outflow tract sphincter was the cause, through the discovery of systolic anterior motion (SAM) of the mitral valve, to current understanding that flow drag, the pushing force of flow, is the dominant hydrodynamic mechanism for SAM. The continuing redesign and modification of surgical procedures to relieve outflow obstruction have corresponded to ideas about the cause of this condition. In this review we discuss the evolution of surgical procedures to relieve obstruction and review modern surgical approaches. Medical and nonsurgical methods for reducing obstruction are reviewed, as well as efforts to prevent sudden arrhythmic cardiac death. Echocardiography has become central to understanding this complex phenomenon, and for clinical diagnosis, operative planning and intraoperative management.

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Management and decision-making strategy for systolic anterior motion after mitral valve repair

Giuseppe Crescenzi, MD,∗ Giovanni Landoni, MD,∗ Alberto Zanigrillo, MD,∗ Fabio Guarracino, MD,∗ Concetta Rosica, MD,∗ Giovanni La Canna, MD,† and Ottavio Alfieri, MD‡

Objective: Systolic anterior motion can complicate mitral valve repair. It can have no clinical consequence or cause low cardiac output syndrome and hypotension. The management of systolic anterior motion in the operating room remains controversial: some groups advocate nonsurgical management, and others propose immediate surgical correction. Conventional hemodynamic measures require time and can be unsuccessful. While describing our experience, we propose a simple and innovative management and classification of this complication.

Methods: Presenting the data of 608 consecutive patients who underwent mitral valve repair for degenerative mitral valve disease, we describe a novel 2-step conservative management consisting of intravascular volume expansion and discontinuation of inotropic drug (step 1) and increasing afterload by means of ascending aortic stents (step 2). We also describe a novel classification of systolic anterior motion (SAM) (1), difficult to revert (responding to step 2), or persistent. Systolic anterior motion was 9.8% (60/608): 40 patients had easy-to-revert systolic anterior motion. Five patients had a persistent SAM within 48 hours. SAM is a relatively frequent complication after MVR and can occur intraoperatively or postoperatively. A systematic approach addressing perioperative SAM after MVR yields excellent mid-term results.

8.4%

Crescenzi et al JTCVS 2009;137:320-5

6.6%

Varghese et al JTCVS 2012;143:S2-7

4.3%

Brown et al JTCVS 2007;133:136-43
QUESTION 3:

After elective Mitral valve repair in a 40 year old male, SAM with moderate MR and LVOT gradient of 60mmHg is found after discontinuing bypass. Medical therapy is initiated in the operating room which results in a decrease in LVOT gradient to 40mmHg and moderate MR. The next optimal step is to:

A) Start an esmolol infusion and phenylephrine infusion to increase MAP and allow bradycardia and then proceed with closing with a plan to wean the infusions postop.

B) Return to CPB, remove the ring and if the valve competent proceed to close.

C) Return to CPB and displace the posterior leaflet into the ventricle using a neochordae.

D) Remove the ring and replace the mitral valve with posterior leaflet sparing

E) No further intervention is required as the SAM will improve with time
DOES ALL SAM RESOLVE WITH MEDICAL THERAPY?

Figure 2. The incidence of SAM and LVOTO at intraoperative TEE, postmedical management, dismissal, and late follow-up. SAM, Systolic anterior motion; TEE, transesophageal echocardiography; LVOTO, left ventricular outflow tract obstruction; TTE, transthoracic echocardiography.
Management of systolic anterior motion after mitral valve repair: An algorithm

Robin Varghese, MD, MS, FRCSC, Anelechi C. Anyanwu, MD, MSc, FRCS, Shinobu Itagaki, MD, Federico Milla, MD, Javier Castillo, MD, and David H. Adams, MD

Objective: To evaluate the effectiveness and outcomes of an intraoperative and postoperative algorithm for managing systolic anterior motion (SAM) after mitral valve repair (MVRr).

Methods: All consecutive patients who underwent MVRr for degenerative disease from January 2002 to June 2011 were included, with the data collected retrospectively. Patients who underwent MVRr for primary SAM were excluded from the study. Patients who developed SAM after the repair were systematically treated according to the algorithm. The intraoperative algorithm first involved medical management techniques, followed by surgical correction for significant SAM (mild or greater mitral regurgitation, left ventricular outflow tract gradient > 50 mm Hg). The postoperative algorithm focused on medical management and symptoms to guide the treatment decisions.

Results: The overall in-hospital incidence of SAM was 6.6% (52/785). In 41 patients, SAM was identified in the operating room, and in 11 patients, it was found postoperatively on the predischarge echocardiogram. Of the 41 patients with intraoperative SAM, 35 (85.4%) had resolution with medical management and 6 (14.6%) required surgical repeat repair while in the operating room. No patient required mitral valve replacement for persistent SAM. Postoperatively, 11 new cases were identified, and 7 cases of resolved intraoperative SAM recurred. These postoperative cases of SAM were managed according to the postoperative SAM algorithm. At last follow-up, 17 (94.4%) of 18 patients had resolution of SAM and 1 (5.6%) patient had mild SAM (less than mild mitral regurgitation, peak left ventricular outflow tract gradient < 50 mm Hg) and were asymptomatic. No patients with postoperative SAM required reoperation after their initial surgery. The median echocardiographic follow-up was 1.3 years. During follow-up, 1 early death (noncardiac) and 2 late deaths (1 noncardiac, 1 of unknown etiology) occurred.

Conclusions: SAM is a relatively frequent complication after MVRr and can occur intraoperatively or postoperatively. A systematic approach addressing perioperative SAM after MVRr yields excellent mid-term results.

(J Thorac Cardiovasc Surg 2012;143:S2-7)
**SAM ALGORITHM**

**Management of Intracoperative Systolic Anterior Motion**

1. Technically appropriate repair with Prosthetic Leaflet height <5mm and appropriately sized ring.
2. SAM during weaning from CPB?
   - Yes: \( \text{Postop SAM} \)
   - No: \( \text{Proced to closure} \)
3. Echocardiogram on POD 4-6, Beta blocker commencement as soon as possible.
   - No Further Management
   - \( \text{Postop SAM} \)
4. Minimal MR, LVOT gradient <50mmHg
   - Degree
     - Yes: More than minimal MR/LVOT Gradient >50mmHg, Structural SAM
     - No: Resume CPB and perform further repair to address SAM. Check coaptation depth <10-12mm with ink-test after re-repair.
     - Persistent SAM?
       - Yes: Increase beta blocker, administer fluids, avoid diuretics, allow hypotension and repeat echo in 48 hours.
       - No: None or Minimal MR, LVOT gradient <50mmHg

**Management of Postoperative Systolic Anterior Motion**

1. Pre-Discharge Echocardiogram demonstrates SAM
   - Degree
     - Yes: More than minimal MR/LVOT gradient >50mmHg, hemodynamic instability or symptoms
     - No: Increase beta blocker therapy, avoid diuretics, repeat echocardiogram in 6 weeks.
2. Beta-blocker therapy, avoid diuretics, repeat echocardiogram in 6 weeks.
Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring

SAM during weaning from CPB?

Yes

Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachycardia

No

SAM during weaning from CPB?

Minimal MR, LVOT gradient < 50mmHg

Degree

Yes

More than minimal MR/LVOT Gradient > 50mmHg or "Structural SAM"

No

Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

No

Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

No Further Management

Postop SAM?

No

Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.

Yes

Move to Postop SAM Algorithm

Postop SAM?

Yes

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
Management of Intraproperative
Systolic Anterior Motion

Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring

SAM during weaning from CPB?

No

Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachycardia

Yes

Persistent SAM?

No

More than minimal MR/LVOT Gradient > 50mmHg or “Structural SAM”

Yes

Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.

No Further Management

Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

Postop SAM?

No

Degree

Yes

Minimal MR, LVOT gradient < 50mmHg

Resume CPB ...

Structural SAM

No

Move to Postop SAM Algorithm

No Further Management

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
Management of Intraproductive Systolic Anterior Motion

- Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring

1. SAM during weaning from CPB?
   - Yes
     - Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachcardia
   - No
     - Persistent SAM?
       - Yes
         - More than minimal MR/LVOT Gradient > 50mmHg or "Structural SAM"
       - No
         - Minimal MR, LVOT gradient < 50mmHg
           - Degree
             - Yes
               - Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.
             - No
               - Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible
                 - No Further Management
                 - Yes
                   - Postop SAM?
                     - No
                       - Resume CPB
                     - Yes
                       - Move to Postop SAM Algorithm

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
Management of Intraproperative Systolic Anterior Motion

Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring

SAM during weaning from CPB?

Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachycardia

Persistent SAM?

More than minimal MR/LVOT Gradient > 50mmHg or “Structural SAM”

Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.

Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

Minimal MR, LVOT gradient < 50mmHg

Degree

Yes

No

Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

Postop SAM?

Yes

No

Move to Postop SAM Algorithm

No Further Management

Postop SAM?

Yes

No

Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring

Management of Intraproductive Systolic Anterior Motion

- Proceed to closure

- Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

- Minimal MR, LVOT gradient < 50mmHg

- Degree

- No

- Postop SAM?

- Yes

- No Further Management

- Move to Postop SAM Algorithm

- Persistent SAM?

- Yes

- Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.

- More than minimal MR/LVOT Gradient > 50mmHg or "Structural SAM"

- Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachycardia

- SAM during weaning from CPB?

- Yes

- No

- No

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
AFTER COMING OFF BYPASS...
Management of Intraoperative Systolic Anterior Motion

- Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring
  - SAM during weaning from CPB?
    - Yes
      - Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachycardia
    - No
      - Minimal MR, LVOT gradient < 50mmHg
        - Degree
          - Yes
            - Persistent SAM?
              - No
                - Proceed to closure
              - Yes
                - Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.
          - No
            - More than minimal MR/LVOT Gradient > 50mmHg or "Structural SAM"

- Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible
  - Postop SAM?
    - No
      - No Further Management
    - Yes
      - Move to Postop SAM Algorithm

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
Management of Intraproperative Systolic Anterior Motion

Technically appropriate repair with Posterior Leaflet height <15mm and appropriately sized ring

SAM during weaning from CPB?

Yes

Wean from CPB avoiding inotropes, keep MAP 80-90, increase preload, avoid tachycardia

No

SAM during weaning from CPB?

No

Proceed to closure

Echocardiogram on POD #2/3, Beta blocker commencement as soon as possible

Minimal MR, LVOT gradient < 50mmHg

No

Degree

Yes

More than minimal MR/LVOT Gradient > 50mmHg or "Structural SAM"

Persistent SAM?

No

Resume CPB and perform further repair to address SAM. Check coaptation depth < 10-12mm with ink-test after re-repair.

Yes

Postop SAM?

No

Move to Postop SAM Algorithm

Yes

No Further Management

Varghese R, Anyanwu AC et al JTCVS 2012;143:S2
## BACKGROUND

**TABLE 2. Details of 6 patients requiring intraoperative repeat repair for systolic anterior motion**

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>Etiology of MR</th>
<th>Valve lesion</th>
<th>Initial repair technique</th>
<th>Ring type and size</th>
<th>Repeat repair technique</th>
<th>SAM resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Barlow’s disease</td>
<td>Ruptured A2 chord, restricted calcified P3 chord</td>
<td>Triangular resection of A2, annuloplasty ring</td>
<td>C-E Physio 40 mm</td>
<td>Posterior leaflet shortening by horizontal resection at annulus</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>Barlow’s disease</td>
<td>P3 chordal elongation</td>
<td>P3 resection and sliding plasty</td>
<td>C-E Physio 38 mm</td>
<td>P1 shortening by horizontal resection at annulus</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>Forme fruste</td>
<td>P2 chordal elongation</td>
<td>P2 triangular resection</td>
<td>C-E Physio 32 mm</td>
<td>Triangular resection of A2 tip and cutting of abnormal chord; P2 Gore-Tex chord to lower height</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>Forme fruste</td>
<td>P2 chordal elongation</td>
<td>P2 quad resection, horizontal, vertical compression with sliding plasty, P2 neochordae</td>
<td>C-E Physio II 32 mm</td>
<td>Shortening of P2 neochordae and addition of chordal transfer</td>
<td>Yes</td>
</tr>
<tr>
<td>5</td>
<td>Fibroelastic deficiency</td>
<td>P3 chordal elongation</td>
<td>Chordal transfer to P3 and placement of neochordae to P3</td>
<td>C-E Physio 30 mm</td>
<td>Short neochordae to P2 and P3 to shorten leaflet height, magic suture to anterior commissure</td>
<td>Yes</td>
</tr>
<tr>
<td>6</td>
<td>Fibroelastic deficiency</td>
<td>P2 ruptured chord</td>
<td>P2 triangular resection</td>
<td>C-E Physio 26 mm</td>
<td>Short neochordae to P2</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*Pt. no., Patient number; SAM, systolic anterior motion; C-E Physio, Carpenter-Edward Physio Ring (Edwards Life Sciences, Irvine, Calif).*
Predicting systolic anterior motion after mitral valve reconstruction: using intraoperative transoesophageal echocardiography to identify those at greatest risk

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Abstract

OBJECTIVES: We set out to determine if intraoperative pre-bypass transoesophageal echocardiography could assist in predicting which patients are at greatest risk for systolic anterior motion (SAM) after mitral valve repair (MVR).

METHODS: Three hundred and seventy-five consecutive patients who underwent reconstructive MVR surgery for degenerative disease were included. Data were collected using intraoperative echocardiographic images taken prior to the initiation of cardiopulmonary bypass. Based on the physiology of SAM, we postulated that 11 parameters could be potential risk factors for SAM: left ventricular ejection fraction (LVEF), left ventricular end-systolic dimension, left ventricular end-diastolic dimension (LVEDD), basal septal diameter (basal-interventricular septal diameter in diastole [IVDD]), mid-ventricular septal diameter (mid-IVDD), coaptation-septal distance (c-sept), anterior leaflet height, posterior leaflet height, aorto-mitral angle, mitral annular diameter and left atrial diameter. These parameters were measured and recorded by a blinded single operator. Independent predictors of SAM were identified using multiple logistic regression analysis.

RESULTS: Of the 375 patients, 345 (92%) did not develop SAM (No-SAM group), while 30 (8%) developed intraoperative or postoperative SAM (SAM group). The mean age was 56.8 ± 12.8 and 56.7 ± 13.8 in the No-SAM and SAM groups, respectively. The incidence of fibroelastic deficiency, form fruste and Barlow’s disease was similar in both groups. All patients received a complete annuloplasty ring as part of the repair. There was no statistical difference in the mean ring size used in each group. EF was similar in the No-SAM (56.2% ± 8.1) and SAM (57.0% ± 9.2) P = 0.63) groups. Independent predictors of developing SAM after valve repair were: EDD <45 mm [odds ratio (OR) 3.90; P = 0.028], aorto-mitral angle <120° (OR 2.74; P = 0.041), coaptation-septal distance <25 mm (OR 5.09; P = 0.003), posterior leaflet height >15 mm (OR 3.80; P = 0.012) and basal septal diameter ≥15 mm (OR 3.63; P = 0.039).

CONCLUSIONS: The risk for SAM can be predicted using intraoperative transoesophageal echocardiography. The combination of a smaller left ventricle, tall posterior leaflet, narrow aorto-mitral angle and enlarged basal septum significantly increases the risk for SAM. Knowing these parameters prior to valve repair can assist the surgeon in adjusting their repair technique to minimize the risk.

Keywords: Mitral valve repair • Systolic anterior motion
Results:
Leaflet(s) Repaired

Leaflet(s) by Presence of SAM

- Posterior: P=0.07
- Anterior: P=0.42
- Bileaflet: P=0.17
Results:
Repair Techniques

- Posterior leaflet resection: P=0.84
- Poster leaflet sliding plasty: P=0.46
- Neochordal placement: P=0.37
- Commisuroplasty: P=0.06
Results:
Cardiac Chamber Measurements (Univariate Analysis)

Mean Cardiac Chamber Measurements

- Ejection fraction (%)
  - All Patients: 56.3
  - No SAM: 56.2
  - SAM: 57
  - P = 0.61

- LVESD (mm)
  - All Patients: 32.6
  - No SAM: 32.8
  - SAM: 28.9
  - P = 0.02

- LVEDD (mm)
  - All Patients: 54.5
  - No SAM: 55
  - SAM: 48.4
  - P = 0.001

- Left atrial diameter (mm)
  - All Patients: 54.6
  - No SAM: 54.6
  - SAM: 54.1
  - P = 0.82
Results:

Septal Diameter (Continuous Variables, Univariate)

Mean Septal Diameter Measurements

- Basal septal diameter (mm):
  - No SAM: 11.2 mm
  - SAM: 13.1 mm
  - $P=0.003$

- Mid-LV septal diameter (mm):
  - No SAM: 9.5 mm
  - SAM: 10.8 mm
  - $P=0.005$
Results:
Mitral Valve Measurements (Continuous Variables)

Mean Mitral Valve Measurements

- Anterior leaflet length: 29.8, 29.6, 31.9, P=0.06
- Posterior leaflet length: 15.1, 14.9, 16.5, P=0.03
- Coaptation-septum distance: 26.7, 27.1, 20.1, P=0.001
- Mitral annular diameter: 39.2, 39.2, 38.2, P=0.31
- Annuloplasty ring size: 31.5, 31.5, 31.7, P=0.76
Results:
Aorto-mitral Angle

No SAM | SAM

Mean Aorto-mitral Angle

P=0.002

Degrees

110 112 114 116 118 120 122 124 126 128

116.1 127.6
Results:

Independent Predictors as Categorical Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate</th>
<th>Multivariate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P-Value</td>
</tr>
<tr>
<td>Ejection fraction &gt; 65%</td>
<td>1.38 (0.54 – 1.38)</td>
<td>0.51</td>
</tr>
<tr>
<td>End-systolic diameter &lt; 35mm</td>
<td>2.51 (0.99 – 6.34)</td>
<td>0.053</td>
</tr>
<tr>
<td>End-diastolic diameter &lt; 45mm</td>
<td>3.21 (1.37 – 7.57)</td>
<td>0.007</td>
</tr>
<tr>
<td>Septal diameter (basal) ≥ 15mm</td>
<td>4.78 (2.06 – 11.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Coaptation-septum distance &lt; 25mm</td>
<td>8.40 (3.27 – 22.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anterior leaflet length ≥ 25mm</td>
<td>4.10 (0.96 – 17.6)</td>
<td>0.058</td>
</tr>
<tr>
<td>Posterior leaflet length ≥ 15mm</td>
<td>2.61 (1.18 – 5.79)</td>
<td>0.018</td>
</tr>
<tr>
<td>Aortomitral angle &lt;120 °</td>
<td>3.49 (1.62 – 7.56)</td>
<td>0.001</td>
</tr>
</tbody>
</table>
QUESTION 3:

After elective Mitral valve repair in a 40 year old male, SAM with moderate MR and LVOT gradient of 60mmHg is found after discontinuing bypass. Medical therapy is initiated in the operating room which decreases the MR to mild and LVOT gradient to 40mmHg. The next optimal step is to:

A) Start an esmolol infusion and phenylephrine infusion to increase MAP and allow bradycardia and then proceed with closing with a plan to wean the infusions postop.

B) Return to CPB, remove the ring and if the valve competent proceed to close.

C) Return to CPB and displace the posterior leaflet into the ventricle using aPTFE neochord.

D) Remove the ring and replace the mitral valve with a mechanical/bioprosthesis.

E) No further intervention is required as the SAM will improve with time.
THANK YOU