Malfunction of the Mitral Valve Prosthesis (Cutter-Smeloff)

Clinical and Hemodynamic Observations in Three Cases

By Simon J. K. Lee, M.D., F.R.C.P.(C), A. J. Zaragoza, M.D., J. C. Callaghan, M.D., F.R.C.S.(C), C. M. Couves, M.D., F.R.C.S.(C), and L. P. Sterns, M.D., F.R.C.S.(C)

SUMMARY

Three cases of intermittent obstruction of the mitral prosthesis (Cutter-Smeloff) due to ball variance have been reported. The typical findings were marked variation in the arterial pulse and the intensity of the mitral closing sound and intermittent absence of the opening click. The phonocardiography revealed varying intervals from aortic closing sound to the opening click (A2 - OC interval). Hemodynamic studies revealed fluctuating aortic and pulmonary artery wedge pressures in spite of normal sinus rhythm, due to impaction of the ball into the prosthetic ring, opening only with severe elevation of the left atrial pressure. At operation, the balls showed yellowish discoloration, but no disruption of sutures nor thrombosis of the prosthesis was found. The incidence of this phenomenon was 9% with the Cutter-Smeloff mitral prosthesis, but no such case was seen among patients with the Starr-Edwards mitral prosthesis.

Additional Indexing Words:
Mitral ball variance
Hemodynamics
Mitral valve surgery
Phonocardiography

Ball-valve malfunction of the Starr-Edwards aortic prosthesis due to swelling, fissuring, fragmentation, and fracture, which may lead to embolization or impaction of the poppet and sudden death, is being recognized with increasing frequency.1-8 However, mechanical failure of the mitral ball prosthesis appears to be rare, as only a few cases have been reported,9,10 and they have involved the Cutter-Smeloff prosthesis. A few cases of poppet changes without hemodynamic complications have been observed with the Starr-Edwards mitral prosthesis, but these have been incidental findings.11

The purpose of this communication is to report three cases of Cutter-Smeloff mitral ball-valve malfunction and to present the clinical and hemodynamic features of the sticky mitral ball-valve syndrome. We will attempt to explain the reasons for the mechanical obstruction occurring in the Cutter valve but not in the Starr-Edwards prosthesis.

Methods

Cardiac catheterization was carried out under fluoroscopic control after the patient had had a light breakfast. ECG, phonocardiographic findings, and pressures were recorded by a multi-channel recorder.* Statham pressure transducers (P23Db) were directly connected to a N6 Cournand catheter for pressure recordings on the venous side and a N7 NIH angio catheter for the arterial side. The reference point for zero pressure

From the Departments of Medicine and Surgery, University of Alberta Hospital, Edmonton, Alberta, Canada.

Work was carried out during Dr. Lee's tenure of a senior research fellowship from the Canadian Heart Foundation.

Received November 5, 1969; accepted for publication November 26, 1969.

Circulation, Volume XLI, March 1970 479

*DR-8, Electronics for Medicine, Inc., White Plains, New York.
was the midthoracic level. Respiration (case 3) was recorded by a mercury-filled strain-gauge plethysmograph.* Cardiac output was measured using direct Fick principle. Oxygen uptake was estimated by collecting expired gas in Douglas bags with analysis for oxygen and carbon dioxide by Beckman E-2 oxygen analyzer* and CO₂ capnograph.† Arterial and pulmonary artery blood were analyzed for oxygen by the method of Van Slyke and Neill.

Report of Cases

Case 1: L.H. (No. 106270)

This 55-year-old man had mitral valve replacement with a Cutter-Smeloff (no. 9 M) prosthesis in January 1968. Prior to surgery, he complained of severe exertional dyspnea, orthopnea, and atrial fibrillation. Cardiac catheterization revealed reduced cardiac output, and elevated pulmonary artery (PA) wedge pressure with giant V waves (table 1). At operation, the mitral valve was found to be grossly incompetent and was replaced by a no. 9 Cutter prosthetic valve. The patient made an uneventful recovery and experienced symptomatic improvement.

However, 10 months after surgery, he was readmitted because of increasing dyspnea and episodes of pulmonary edema. On examination, the pulse volume was grossly irregular in spite of a regular sinus rhythm, the jugular venous pressure was elevated to 10 cm, and marked hepatomegaly was found. The first heart sound was variable in intensity, and the opening click of the prosthesis was heard intermittently. A short apical systolic murmur was heard. Chest x-rays revealed cardiac enlargement with pulmonary venous congestion.

Repeated heart catheterization revealed hemodynamic deterioration since the preoperative study (table 1). The most significant findings were marked variation in both pulmonary artery wedge pressure from 22 mm Hg to 50 mm Hg and in aortic systolic pressure from 63 mm Hg to 90 mm Hg (fig. 1). An inverse relationship was found between these two pressures; i.e., with increasing wedge (left atrial) pressure the aortic pressure decreased, while a sudden drop in the wedge pressure was followed by an abrupt increase in the aortic pressure. These findings are consistent with intermittent obstruction of the mitral prosthesis.

Reoperation was carried out. An excerpt of the surgical report follows: "... exploration of the left atrium was made through a left atriotomy. At first examination, the ball appeared to move well

Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>Preop and</th>
<th>VO₂</th>
<th>CO</th>
<th>RA</th>
<th>PA</th>
<th>PA wedge</th>
<th>Aorta</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PO (mol%)</td>
<td>(ml/min)</td>
<td>(L/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Preop</td>
<td>163</td>
<td>2.37</td>
<td>9</td>
<td>46/21 (28)</td>
<td>(23)</td>
<td>121/86</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>307</td>
<td>2.60</td>
<td>11</td>
<td>86/48 (59)</td>
<td>(22)-(50)</td>
<td>63/52-90/65</td>
</tr>
<tr>
<td></td>
<td>Preop</td>
<td>150</td>
<td>2.91</td>
<td>6</td>
<td>41/15 (28)</td>
<td>(29)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>23</td>
<td>163</td>
<td>2.71</td>
<td>12</td>
<td>29/13 (20)</td>
<td>(14)</td>
<td>99/52</td>
</tr>
<tr>
<td></td>
<td>36</td>
<td>156</td>
<td>3.12</td>
<td>15</td>
<td>57/38 (46)</td>
<td>(15)-(40)</td>
<td>86/68-130/70</td>
</tr>
<tr>
<td></td>
<td>Preop</td>
<td>312</td>
<td>6.40</td>
<td>6</td>
<td>45/22 (35)</td>
<td>(29)</td>
<td>106/57</td>
</tr>
<tr>
<td>3</td>
<td>22</td>
<td>287</td>
<td>6.37</td>
<td>4</td>
<td>24/10 (18)</td>
<td>(10)</td>
<td>92/64</td>
</tr>
<tr>
<td></td>
<td>43</td>
<td></td>
<td></td>
<td>5</td>
<td>67/36</td>
<td>(10)-(30)</td>
<td>77/67-118/81</td>
</tr>
</tbody>
</table>

Abbreviations: PO = postoperative; CO = cardiac output; RA = right atrium; PA = pulmonary artery; numbers in parentheses are the mean pressures.

*Beckman Instruments, Inc., Fullerton, California. †Goddart Valve Corp., Worcester, Massachusetts.

Figure 1

Case 1. ECG, aortic pressure, and pulmonary artery wedge (PAW) pressures.
and the procedure was to be terminated. However, when the patient was weaned-off the pump, the pulmonary artery pressure was noted to increase and palpation at this time revealed the prosthetic valve to be non-functional. Bypass was re instituted, and the left atriotomy reopened, and the ball was seen to 'stick'. The silastic ball was yellowish, but neither thrombosis nor disruption of the sutures was found. The whole Cutter prosthesis was removed and replaced with a 33-mm Cooley-Bloodwell prosthesis. The patient recovered from surgery.

**Case 2: P.J. (No. 185504)**

This 46-year-old woman underwent mitral valve replacement (Cutter-Smeloff no. 6 M) for severe mitral regurgitation due to rheumatic heart disease. Prior to surgery, her symptoms were dyspnea on mild exertion, paroxysmal nocturnal dyspnea, and hemoptysis. A clinical diagnosis of mitral and tricuspid regurgitation was confirmed by hemodynamic studies (table 1). During mild exercise, the mean pulmonary artery wedge pressure increased from 29 mm Hg to 35 mm Hg with giant V waves.

At operation, the mitral valve was grossly regurgitant and was replaced, but the tricuspid valve lesion was not severe enough to warrant surgical measures. At routine postoperative checkup 23 months after surgery, she reported marked symptomatic improvement. On examination, the heart was in normal sinus rhythm, and heart sounds of the prosthetic valve were normal with a short mid systolic murmur best heard at the apex. Repeated hemodynamic studies revealed a significant improvement (table 1).

Three years after the operation, the patient was readmitted complaining of increasing difficulty with dizziness and shortness of breath on exertion which had been present for 3 months. She also had become aware of a variation in the sounds of her valve. On examination, the peripheral pulse was grossly irregular in spite of the sinus rhythm. The first heart sound over the apex varied in intensity, and the opening click (OC) was intermittently absent. Repeated hemodynamic studies revealed again a marked fluctuation in both wedge and aortic pressure with intermittent opening click and variable $A_2 - OC$ interval (fig. 2).

At reoperation, the ball was yellowish and was found to stick in the closed position. However, no
ingrowth of tissue over the struts was seen, and good epithelialization was found over the sewing ring. The ball was irregular in shape with "bubbles" beneath the surface. A hairline crack, 5 mm in length and 4 mm in depth, was also found. After extracting the ball through an apical ventriculotomy, a new ball was successfully placed in the cage.

Case 3: H.C. (No. 256967)

This 40-year-old man underwent mitral valve replacement with a Cutter-Smeloff no. 8 M prosthetic valve, for rheumatic mitral valve disease. Before surgery, his difficulties were shortness of breath on exertion and hemoptysis. A clinical diagnosis of mitral stenosis and regurgitation was confirmed by hemodynamic studies (table 1). On mild exercise, the mean PA wedge pressure increased from 29 mm Hg to 40 mm Hg. At operation, the mitral valve was severely regurgitant and calcified.

On a routine check-up 22 months after surgery, the patient was asymptomatic and was able to participate in sports without any difficulty. Hemodynamic studies at this time revealed significant improvement (table 1). On heavy exercise that increased his oxygen consumption from a resting 287 to 1,978 cc/min, the pulmonary artery pressure only increased from 20/13 (20) mm Hg to 57/26 (40) mm Hg, and the mean PA wedge pressure increased from fourteen mm Hg to 28 mm Hg. Cardiac output increased from a resting value of 6.37 L/min to 13.17 L/min.

Forty-three months after the surgery, he was readmitted complaining of severe shortness of breath and hemoptysis which developed in a period of 2 weeks. On examination, the peripheral pulse was grossly irregular in volume despite sinus rhythm. As with the previous two cases, the first heart sound over the apex varied in intensity, and the opening click was intermittently absent. Hemodynamic studies at this time revealed findings similar to those in the two previous cases (table 1). Again, a marked fluctuation of both wedge and aortic pressure with the intermittent opening click was found (fig. 3). The time interval between the second heart sound and the opening click varied from 0.08 sec to 0.13 sec.

Figure 3

Case 3. Opening click (OC) present intermittently. Marked fluctuation in aortic and PA wedge pressure not related to the respiration. An abrupt increase in aortic pressure is preceded by a high PA wedge pressure, and an opening click.
which was attributed to changing left atrial pressure.

At operation, the ball was yellowish without any crack. The ball was replaced at first with a new ball from a no. 8 Cutter prosthesis, but was found to stick again into the ring, and it was decided to replace the valve with the largest toroidal valve (20.5 mm) available. The patient made a satisfactory recovery from this surgery.

Discussion

An early diagnosis of mitral ball-valve malfunction due to ball variance is critical. Due to the lower pressure generation of the left atrium in comparison to the ventricle, any impingement of the ball into the ring during systole would result in obstruction of the diastolic filling and is likely to have a far more serious consequence than the aortic prosthesis variance. Phonocardiography12 and sound spectroscopy13 were useful in detecting aortic ball-valve variance, but similar studies have not been reported for the mitral ball variance, although absence of the opening click has been described in dehiscence14 and massive thrombosis of the mitral prosthesis.15

All three of our patients presented unique clinical and hemodynamic findings of intermittent mitral valve obstruction and the diagnosis of malfunction of the mitral ball valve is easy to make in the presence of sinus rhythm. The typical physical findings are fluctuating pulse volume and arterial pressure accompanied by varying intensity of the mitral closing sound, intermittent opening click, and the variable second sound-opening click duration. When the arterial pressure was recorded simultaneously with pulmonary artery wedge pressure, an increasing PA wedge (left atrial) pressure was accompanied by decreasing aortic pressure, while a sudden drop in the wedge pressure was followed by an increase in the aortic pressure, indicating that the ball valve opens intermittently only with the increased left atrial pressure; hence, the term “sticky” valve has been suggested. The phonocardiographic findings of an intermittent opening click and varying intensity of the closing sound also confirm the diagnosis.

The intermittent obstruction appears to be the result of impaction of the ball into the prosthetic ring due to systolic pressure. Observations at operation and examination of the removed prosthesis did not show evidence that the poppet was impinged in the struts (cage). Had this been the case, mitral regurgitation would have resulted.

The Cutter prosthesis has been designed to allow almost one half of the ball to enter the ring during systole in order to increase the size of the effective orifice and to lower the height of the struts (fig. 4). This fact, in combination with the softening of the silastic rubber of the ball, appears to be the reason for the impaction during systole, as the increased size alone without changing the consistency would decrease the tendency for impaction into the seating ring.

The balls removed in cases 1 and 3 were analyzed by the Cutter Laboratories. In case 1, the mean diameter was increased by 0.007 inches with a 140-mg increase in weight. The total extractable material was 4% by weight (1.5% in simple and 2.5% in complex lipids). In case 3, the increase in mean diameter was 0.019 inches with the total extractable material of 2.85% (2.51% in lipids and 0.34% in silicone). Thus, the change in diameter and weight was minimal. The finding of an increased lipid content is similar to the observations made in cases of aortic (Starr-Edwards) ball variance.8

Figure 4

Comparison of the normal Cutter-Smelloff (left) and the Starr-Edwards (right) mitral prosthesis in the systolic position. Note that about half of the ball passes beyond the valvular ring (during systole) in the Cutter-Smelloff prosthesis, while little engagement is seen in the Starr-Edwards prosthesis.

Circulation, Volume XLI, March 1970
Prior to 6 months before this review, a total of 32 Cutter mitral prostheses had been used in this center, including multi-valve replacement. Therefore, the apparent incidence of mechanical failure has been 9% in this group. In contrast, a total of 84 mitral Starr-Edwards prostheses had been used, excluding the multi-valvular replacement prior to 1967, with no case of mechanical failure in this group. In our opinion, this is due to the difference in mechanical design of the Starr-Edwards prosthesis (fig. 4) in which the ball does not enter the valvular ring during systole as changes in the silastic ball of this prosthesis have been reported to occur. From this experience, further use of the Cutter-Smeloff mitral prosthesis appears inadvisable.

Acknowledgment

We are grateful to Drs. R. E. Rossall and J. Dvorkin for making their patients available for this study.

References

Malfunction of the Mitral Valve Prosthesis (Cutter-Smeloff): Clinical and Hemodynamic Observations in Three Cases

SIMON J. K. LEE, A. J. ZARAGOZA, J. C. CALLAGHAN, C. M. COUVES and L. P. STERNS

_Circulation_. 1970;41:479-484
doi: 10.1161/01.CIR.41.3.479

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1970 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/41/3/479

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

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

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