Dysfunction of Mitral Ball Valve Prosthesis

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Three case histories of patients with malfunctions of Smeloff-Cutter mitral ball valve prostheses are presented here —one underwent successful replacement. The clinical diagnosis was made by observing a marked variation in the A_2OC interval and intermittent absence of the opening click. Phonocardiograms were diagnostic in all three cases, while echocardiograms and even angiograms did not uniformly diagnose prosthetic valve dysfunction. Periodic phonocardiographic evaluations may be helpful in early detection of the prosthetic valve dysfunction. Once the diagnosis is established, immediate surgical treatment is imperative to prevent sudden death.*

Introduction

Phonocardiography has been of major importance in the diagnosis of prosthetic mitral valve dysfunction by documenting changes in the intensity and timing of the prosthetic mitral valve opening click (OC) (1,6,8,11-14). The normal interval from the aortic valve closing sound to the opening click (A_2OC interval) in patients with ball valve prostheses ranges from 70-170 msec (11,15-18), with a beat-to-beat variation of up to 20 msec, if the patient is in sinus rhythm.

The three cases presented malfunctions of Smeloff-Cutter valves in the mitral position. One occurred as a result of fibrous tissue overgrowth on the valve ring, while the poppet itself remained normal (6,8-10). Phonocardiography confirmed these suspected malfunctions, while one case was not detected by echocardiography and angiography. Immediate diagnosis and prompt surgical management of prosthetic valve dysfunctions have prevented fatal outcomes in some cases (6,8,9).

Case Reports

Case 1

A 63 year-old man was admitted to the hospital in May 1970 with a sudden onset of right hemiparesis and dysphasia. Rheumatic mitral stenosis and regurgitation had been diagnosed in 1966, and he had undergone a mitral valve replacement with a No. 8 Smeloff-Cutter ball valve prosthesis. Since the valve replacement, he had done well on warfarin sodium with prothrombin times in the therapeutic range. In March 1977 a cardiac catheterization confirmed a paravalvular leak, which was closed surgically.

Physical examination showed dysphasia and a right hemiparesis. Cardiac examination revealed a regular rhythm and a blood pressure of 160/95 mmHg. The A_2-OC interval was prolonged, with a marked beat-to-beat variation. A phonocardiogram confirmed the variation of the A_2-OC interval (Fig. 1). On cardiac catheterization, pulmonary artery (PA) pressure was 38/16 mmHg (mean 25), and pulmonary capillary wedge pressure was 14 mmHg with no significant mitral valve gradient. The left ventricular angiogram revealed no mitral regurgitation, and the left ventricular contraction was normal. The function of the mitral valve prosthesis was felt to be abnormal.

At surgery, the prosthesis showed impaired mobility of the poppet due to formation of a fibrin clot along the posterior strut on the ventricular side. The poppet itself appeared normal. The valve was replaced by a No. 7 Smeloff-Cutter prosthesis. Following surgery, the patient developed a low output syndrome and died later the same day.

Fig. 1
Phonocardiogram showing variable A_2-OC interval. OC = opening click, S_1 = first heart sound, S_2 = second heart sound.

Case 2

A 35 year-old woman was admitted in August 1972 with congestive heart failure. Her medical history included mitral valve replacement (No. 9 Smeloff-Cutter ball valve prosthesis) in February 1966. In 1970, she had developed an acute inferior wall myocardial infarction, possibly due to coronary artery embolism, with an uneventful recovery. She did well on adequate anticoagulation, with prothrombin times in the therapeutic range, until she developed acute congestive heart failure and was admitted to another hospital in July 1972. Her symptoms improved when she received digitalis and diuretics, but she continued to have dyspnea at rest.

She had a regular heart rhythm and a blood pressure of 110/90 mmHg. Her neck veins were markedly distended at 45°, and she had 4+ pretibial edema. Her first and second heart sounds were accentuated. The prosthetic mitral valve opening click (OC) was markedly delayed and at times absent. There was a 3/6 holosystolic murmur best heard at the apex. An electrocardiogram showed a regular sinus rhythm and evidence of an old inferior wall myocardial infarction. Chest fluoroscopy revealed gross cardiac enlargement; however, the mitral valve poppet was thought to move normally.
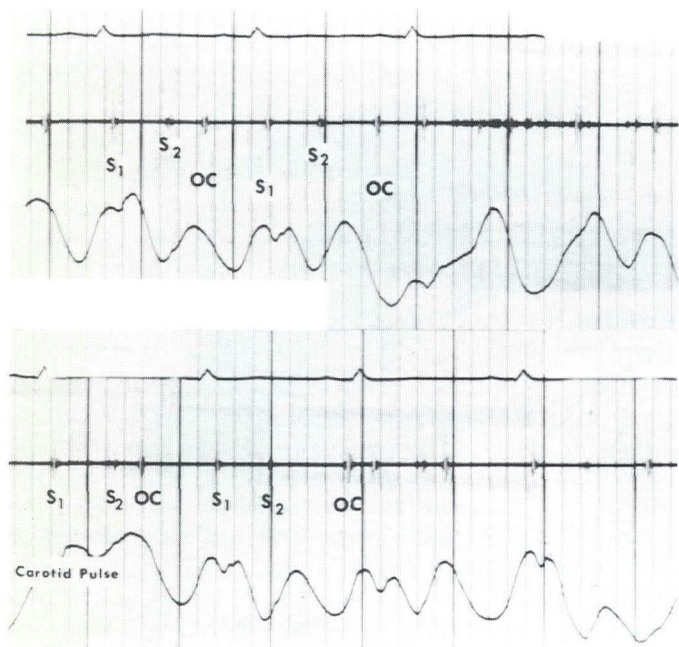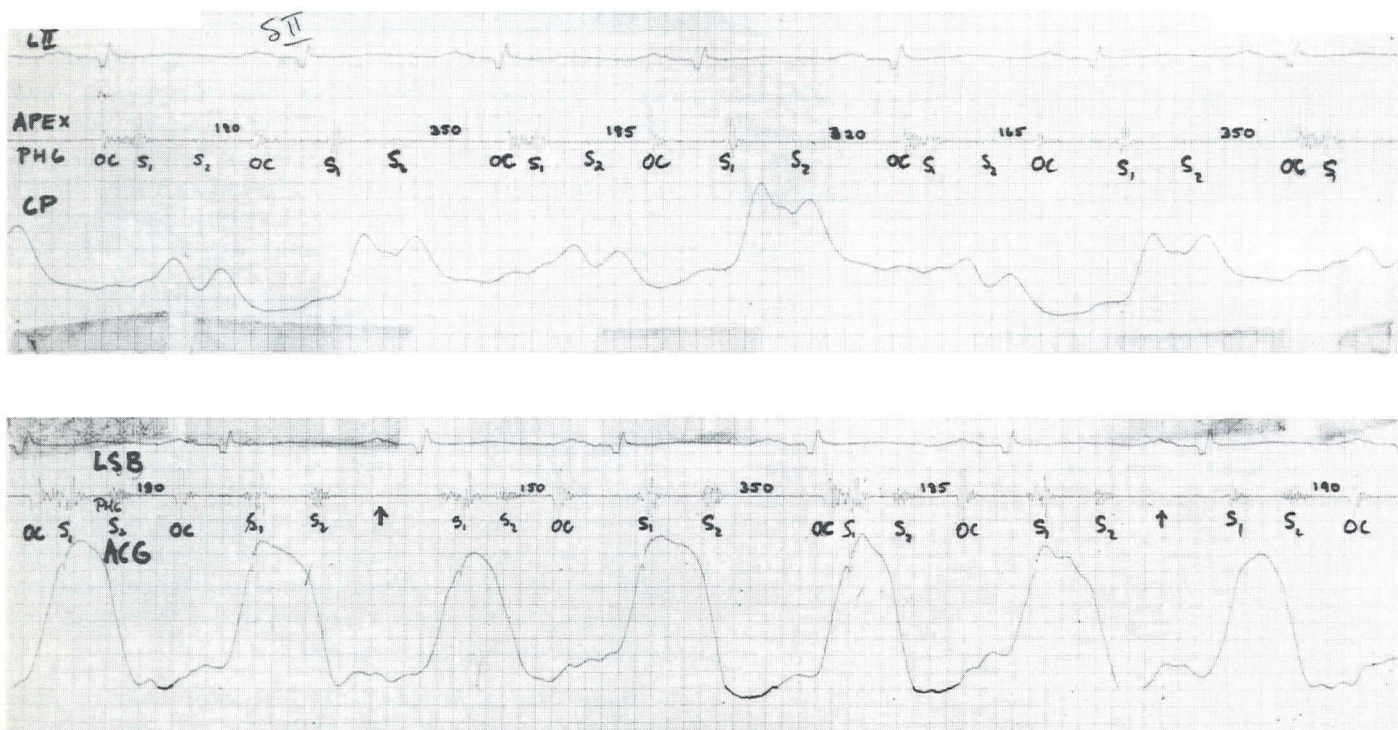

Fig. 2
Phonocardiogram showing variable S_2OC interval between 150-350 msec. There is diminished carotid pulse wave amplitude following a cycle with the delayed opening of the mitral valve poppet (upper panel). Arrows (lower panel) indicate where there was a complete absence of the OC due to the sticking of the mitral valve poppet. ACG = apex cardiogram, CP = carotid pulse tracing, LSB = left sternal border, OC = opening click, PHG = phonocardiogram, S_1 = first heart sound, S_2 = second heart sound.

A phonocardiogram revealed a prolonged A_2-OC interval with significant variations (15-380 msec) between beats. The carotid pulse tracing showed variable amplitude with each small pulse wave being preceded by a prolonged A_2-OC interval (Fig. 2, upper panel). At times, the opening click was totally absent (Fig. 2, lower panel). During her stay in the hospital, she had several episodes of near syncope which were believed due to low cardiac output.

A cardiac catheterization done one week later revealed a large, poorly contracting left ventricle with moderate mitral regurgitation. The mitral valve poppet movement was irregular. There was moderate pulmonary hypertension (PA pressure 74/41 mmHg, mean 51 mmHg) and a mitral valve gradient of 16mmHg. A diagnosis of sticking mitral valve poppet was made, and the near syncopal episodes were believed due to episodes of acute reduction of cardiac output, resulting from valvular dysfunction. Surgery to replace the valve was scheduled, but on the day before surgery she was found dead in bed. Autopsy permission could not be obtained.

Case 3

A 41 year-old woman was admitted in November 1976 with acute congestive heart failure. In May 1971, she had undergone mitral valve replacement with a No. 5 Smeloff-Cutter ball valve prosthesis for severe rheumatic mitral stenosis. Subsequently, she did well on adequate anticoagulation with warfarin sodium until her second admission in November 1976. She had developed progressive dyspnea three days before admission. Her symptoms improved with digitalis and diuretics; her cardiac rhythm was regular at 110/min, and blood pressure was 110/70 mmHg. Her neck veins were flat at 45°, but the hepatojugular reflux was positive. She had bilateral moist basal crepitations and scattered rhonchi. Auscultation of the heart revealed a loud

S_1, normal S_2, and a loud opening click. A 2/6 midsystolic murmur was heard at the apex. Pulmonary vascular congestion with Kerley 'B' lines was present on x-ray; and an electrocardiogram showed a regular sinus rhythm with right axis deviation and nonspecific ST-T wave changes. Her symptoms improved on medical treatment. On repeat examination a day later, the A_2OC interval was prolonged and varied from beat-to-beat. A phonocardiogram confirmed the variation of the A_2OC interval (Fig. 3) from 95-215 msec. The echocardiogram failed to reveal any abnormality of the mitral valve poppet motion (Fig. 4). A cardiac catheterization four days after admission revealed moderate pulmonary hypertension (PA 55/19 mmHg, mean 14 mmHg) with no significant mitral valve gradient. There was mild mitral regurgitation. The mitral valve poppet seemed to be moving normally. Because of the persistent variation of the A_2-OC interval, mitral valve prosthesis dysfunction was diagnosed. She underwent successful mitral valve replacement with a Bjork-Shiley prosthesis. The removed Smeloff-Cutter valve had an overgrowth of dense fibrous tissue along the rim of the struts, and the ball was stuck to the rim. The poppet itself was normal (Fig. 5). She has continued to do well on long-term anticoagulants.

Discussion

Malfunction of the mitral valve prosthesis is commonly recognized only after complications such as systemic embolism and/or congestive heart failure develop. Because of the serious morbidity and mortality often associated with systemic emboli, congestive heart failure, and the danger of sudden death, early detection of prosthetic valve dysfunction is of obvious importance. Malfunction of the mitral ball valve prosthesis can be suspected by the presence of delayed mitral valve opening click and variation of the A_2OC interval in a patient with regular sinus rhythm.
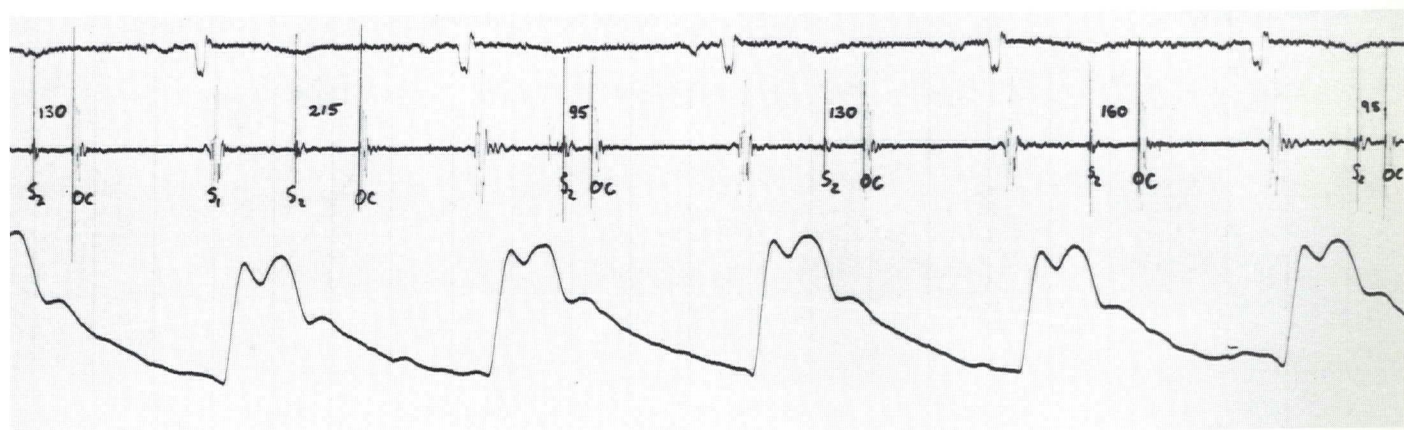

Fig. 3
Phonocardiogram showing variation of the S_2-OC interval from 95-215 msec. OC = opening click, S_1 = first heart sound, S_2 = second heart sound.
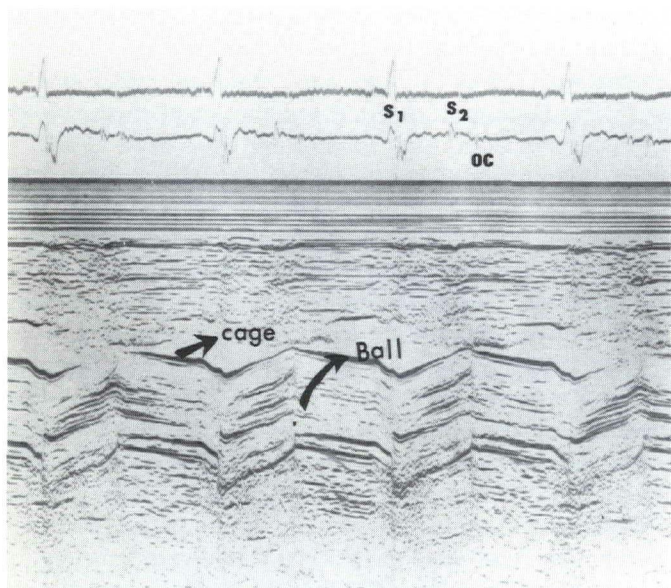
Fig. 4
Echocardiogram showing normal mitral valve poppet motion.

Fig. 5
Smeloff-Cutter valve showing overgrowth of the fibrous tissue along the rim as well as some adherent blood clot. The poppet appears normal.

At times the opening click may be absent, as illustrated in our second case. Intermittent and progressive lengthening of the A_2-OC interval may be associated with variation in the pulse volume or pulsus alternans. Although phonocardiograms and, at times, echocardiograms are useful in the diagnosis of prosthetic mitral valve dysfunction, a thorough history and physical examination should enable one to suspect and diagnose the disorder. Once the diagnosis is made, corrective measures should be taken immediately to prevent a fatal outcome.

The Smeloff-Cutter valve allows almost one half of the poppet to enter the ring during systole, which thus makes it more susceptible to impaction in the seating ring than the Starr Edwards valve.
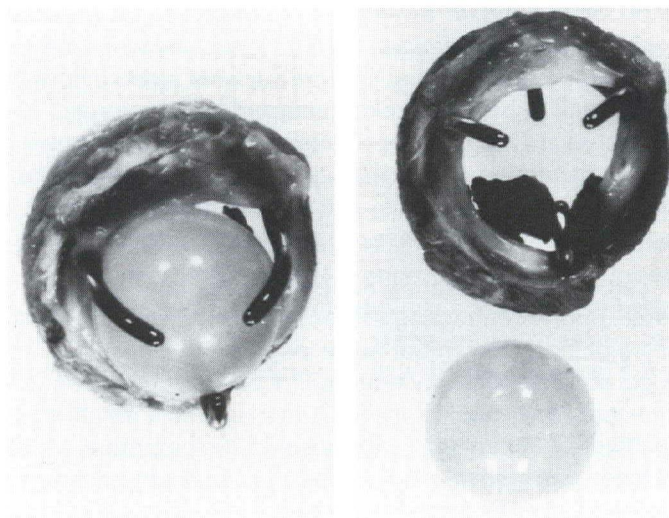
The three cases reported here demonstrate the value of phonocardiography in diagnosing dysfunction of the Smeloff-Cutter mitral ball valve prosthesis. Periodic phonocardiographic evaluations following mitral valve replacement are of value in early detection of valve dysfunction. Sudden onset of congestive heart failure, systemic embolization, or syncopal episodes in a patient with a prosthetic mitral valve who had been doing well before should suggest prosthetic valve dysfunction, and immediate action should be taken to prevent a fatal outcome.

Acknowledgment

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