ventricular rhythm without apparent atrial activity which was interpreted as junctional in origin. Over the next several days the patient demonstrated atrioventricular nodal Wenckebach block, with gradual return to normal conduction. By Sept 24, 1976, the rhythm was normal sinus rhythm, with a P-R interval of 0.20 second.

**DISCUSSION**

Although abnormalities of cardiac conduction are well-recognized complications in both untreated and corticosteroid-treated patients with systemic lupus erythematosus,\(^3\) we believe that the resolution of this patient's atrioventricular block upon the discontinuation of therapy with clonidine indicates that this drug was most likely responsible. While no previous report of heart block due to toxic levels of clonidine has appeared in the literature, two cases of atrioventricular nodal Wenckebach block thought to be based on toxic levels of clonidine have been reported to the manufacturer of the drug (Boehringer Ingelheim Ltd., Elmsford, NY, unpublished data). A single report\(^4\) describes one patient receiving therapeutic amounts of clonidine who was noted to have transient Wenckebach block. It was postulated that the block was related to a reduction in blood pressure, since the disturbance in conduction disappeared concomitant with the return of blood pressure to "control levels." Since the presumed mechanism of clonidine is a decrease in sympathetic tone, it is tempting to speculate that relatively unopposed cardiac parasympathetic stimulation was responsible for the high-degree of atrioventricular block manifested in our patient.

The presence of renal insufficiency in our patient most likely contributed to achievement of toxic levels of clonidine. Prolongation of the radioactive half-life of clonidine beyond the normal range of 16 to 40 hours has been reported in patients with impaired renal function.\(^1\) The unfortunate coincidence of excessive administration of clonidine and diminished renal function apparently combined to produce toxic levels of this drug, which resulted in transient high-grade atrioventricular block. Fortunately, this event was without significant hemodynamic consequences to the patient described in this report.

**REFERENCES**


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**Ball Variance in a Harken Mitral Prosthesis**

**Echocardiographic and Phonocardiographic Features**

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A case of ball variance in a caged-ball prosthetic valve is presented, illustrating the echocardiographic and phonocardiographic features. The swollen silicone ball was observed on echocardiographic studies to have diminished motion and to incompletely open within its cage. On phonocardiographic studies, the Q-S; interval was prolonged, and no opening sound could be recorded. These noninvasive techniques may be helpful in predicting the need for replacement of prosthetic valves that have silicone rubber balls.

Degeneration of the silicone rubber poppet in caged-ball prosthetic cardiac valves, known as "ball variance," has become a well-known long-term complication of prosthetic valvular replacement.\(^1\) Most cases of ball variance occur in prosthetic valves placed in the aortic position, presumably because of stress due to higher pressure, but ball variance may also occur in mitral prostheses. The following case report illustrates the noninvasive diagnosis of ball variance in a patient who had undergone replacement of her mitral valve with a Harken caged-ball prosthesis 13 years earlier.

**CASE REPORT**

A 50-year-old woman with rheumatic mitral stenosis had undergone replacement of her mitral valve with a Harken caged-ball prosthesis 13 years prior to admission to our hospital. Warfarin had been administered continuously following valve replacement. Six months prior to admission, the patient had experienced progressive exercise intolerance, loss of weight, and an episode of transient dysarthria and confusion. At admission to our hospital, no localized neurologic signs were present. The fundi were normal. The first heart sound was variable in intensity. The pulmonic second sound was loud. No opening sound from the prosthetic valve could be heard. A grade 3/6 holosystolic murmur was heard maximally at the apex. A grade 2/6 diastolic rumble was present at the left sternal border. The lungs were clear on auscultation.

A chest x-ray film showed an enlarged left atrium. The

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**BALL VARIANCE IN A HARKEN MITRAL PROSTHESIS 785**
Figure 1. A (left), Simultaneous recordings of ECG, carotid pulse (CAROT), and phonocardiograms from pulmonic area (PA) and left sternal border (LSB). Interval between electrocardiographic Q wave and first heart sound (Q-1) is prolonged at 0.11 second. Holosystolic murmur (SM) is seen at left sternal border. Ratio of PEP/LVET is markedly abnormal (0.49 second). B (right), Simultaneous recordings of phonocardiograms from left sternal border (LSB) and apex, with apexcardiogram. No rapid filling wave is present in apexcardiogram, and no opening sound is present at time of 0 point. Diastolic murmur (DM) is present at left sternal border. E, Maximum systolic excursion; and sfw, slow filling wave.

Figure 2. Composite of Harken valve removed at surgery and preoperative echocardiogram. Silicone ball is misshapen, discolored, and clearly too large for its cage. On echocardiogram, leading edge of ball moves only 4 mm within cage and fails to reach apex of cage in diastole, indicating incomplete opening of valve.

electrocardiogram showed right axis deviation and sinus rhythm.

Phonocardiograms recorded from this patient are shown in Figures 1A and 1B. The interval between the Q wave on the ECG and the first heart sound on the phonocardiogram is 0.11 second, normal being 0.03 to 0.07 second. This suggests an impairment in the movement of the prosthetic ball to the closed position. The ratio of the prejection period (PEP) to the left ventricular ejection time (LVET) is 0.49 (normal, 0.345; SD ± 0.036), indicating abnormal left ventricular function. No opening sound for the prosthetic valve could be recorded. A diastolic rumble, holosystolic murmur, and accentuated pulmonic second sound were also recorded.

The preoperative echocardiogram, obtained with the transducer at the lower left sternal border, is displayed in Figure 2, together with the malfunctioning prosthetic valve, which was removed at surgery. The first linear echo encountered arises from the metal cage of the prosthetic valve and is followed by an echo originating from the leading edge of the ball poppet. Multiple echoes are recorded from the body of the ball. The trailing edge of the ball is artificially displaced posteriorly to the sewing ring of the valve, since sound is conducted more slowly through the ball than through the cardiac tissue. The most striking abnormality present is the markedly reduced movement of the ball within the cage. The ball moves 12 mm from systole to diastole, while the cage moves 8 mm as a result of motion of the mitral annulus, resulting in a net movement of the ball within its cage of only 4 mm. In addition, the leading edge of the ball fails to reach the cage during diastole, indicating failure to open properly.

At cardiac catheterization, the gradient over the mitral valve was 22 mm Hg, with a calculated valvular area of
1.3 sq cm. Moderate mitral regurgitation was seen on left ventricular cinesangiographic studies. No definite abnormalities in the nonradiopaque prosthetic ball were appreciated.

The patient underwent successful replacement of the mitral prosthesis. The malfunctioning prosthetic valve removed at surgery is shown in Figure 2. Several small friable clots were adherent to the sewing ring. The ball itself was yellow and elliptic, with linear indentations on its surface where it came into contact with the cage. The ball was soft on palpation and could barely be moved within the cage.

**DISCUSSION**

Swelling and deformation of silicone rubber prosthetic balls are well-described phenomena, caused primarily by the adsorption of lipid by the silicone ball. Motion of these balls is restricted by the metal cage of the prosthetic valve, resulting in both regurgitation of blood and impairment of ejection.

The phonocardiographic features of such a malfunctioning valve include a delayed closing sound and a diminished or absent opening sound, as well as murmurs of stenosis and regurgitation and also nonspecific elevation of the PEP/LVET ratio. The echocardiogram allows direct visualization of the impaired movement of the ball and the failure of the ball to reach maximum excursion to touch the cage. Due to the irregular nature of the ball and the distortion of reflected ultrasound by this dense structure, no precise measurement of the diameter of the ball was possible. Although no normal series exists for comparison, these phonocardiographic and echocardiographic findings are grossly outside those expected and must be abnormal. Precise definition of normal valves awaits study of a large number of patients.

Although the use of prosthetic valves with silicone rubber balls has largely given way to metal or biologic prosthetic cardiac valves, many patients still have prosthetic valves with silicone rubber balls in place and are at risk of suffering the complications of ball variance. Echocardiographic and phonocardiographic studies should be useful in following these patients and predicting the need for valve replacement.

**REFERENCES**


**An Unusual Lethal Complication Associated with Starr-Edwards Prosthetic Aortic Valve Holder***


This is the first report of an unusual fatal complication associated with the Starr-Edwards prosthetic aortic valve holder. The patient died 51 days after replacement of his aortic valve with a Starr-Edwards prosthetic aortic valve. The cause of death was coronary arterial embolus caused by a fragment broken off of the prosthetic aortic valve holder.

The common complications associated with prosthetic aortic valves are thromboembolism,1-5 hemolytic anemia,6-10 and mechanical malfunction due to ball variance.11-15 So far, there has been no report of any complication associated with the prosthetic aortic valve holder. Recently, we observed such a complication. Fifty-one days after replacement of his aortic valve with a Starr-Edwards aortic valvular prosthesis, a patient died from coronary arterial embolus caused by a fragment broken off the prosthetic aortic valve holder. We report this case in order to bring the possibility of this intraoperative hazard to the attention of the many cardiac surgeons who regularly use this device.

**CASE REPORT**

A 56-year-old man was admitted to Queen Mary Hospital, Hong Kong, in February 1975 with fever, pain in the chest, and symptoms of heart failure for two days prior to admission. His heart was enlarged, with aortic systolic and diastolic murmurs. The VDRL test and six cultures of blood were negative. An x-ray film of the chest showed cardiomegaly and calcification of the aortic valve and the aortic knuckle. An electrocardiogram revealed left ventricular hypertrophy and episodic atrial fibrillation. Cardiac catheterization and cardioangiographic studies were not performed. The patient responded to therapy with digoxin, furosemide, and a prolonged course of antibiotics.

The patient was readmitted in November 1975 with left ventricular failure. Replacement of the aortic valve was undertaken on Nov 26, 1975. Moderate hypothermia and coronary arterial perfusion was employed during the procedure for replacement of the valve. The surgical findings were (1) an aneurysm (measuring 1.5 cm in diameter) of the right coronary sinus of Valsalva, which had not ruptured into the right ventricle, and (2) a tricuspid aortic valve that was grossly calcified and predominantly stenotic. The aortic valve was excised, and the neck of the aneurysm of the right coronary sinus of Valsalva was repaired using interrupted sutures with backing of felt pledges. A size-11 Starr-Edwards aortic prosthesis (model 1280) was in-

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