# CORRESPONDENCE

## Patient Lives Almost 50 Years after Aortic Valve Replacement with a Starr-Edwards Caged-Ball Valve

*To the Editor:*

Yalcinkaya and colleagues[1] reported the case of a patient whose mitral Starr-Edwards caged-ball valve (SECBV) was still working after 41 years. The authors referred to 2 other mitral SECBV carriers alive at 44 and 45 years. From their own experience, Starr and Grunkemeier[2] reported their longest-term survivors at 44.4 years (mitral position) and 51.7 years (aortic position). We also had an aortic valve recipient who lived for nearly 50 years.

On 17 April 1964, a 10-year-old boy underwent aortic valve replacement with an SECBV at a different hospital. After discharge, all his postoperative monitoring occurred in our hospital. Through the years, transthoracic echocardiograms (TTEs) showed good performance of the prosthetic aortic valve. In 2009, when the patient underwent successful ablation of supraventricular tachycardia at age 45 years, TTE and cardiac catheterization revealed good aortic valve function. On 19 January 2014, he underwent urgent cardioversion of ventricular tachycardia. Results of radiographic and fluoroscopic examination indicated normal valvular function. A TTE revealed a peak gradient of 41 mmHg, a mean gradient of 21 mmHg, and normal left ventricular function. Four days later, the 59-year-old patient died of refractory ventricular fibrillation—49 years, 9 months, 7 days after valve replacement. No autopsy was performed.

Given so few reports of well-functioning SECBVs beyond 40 years,[3-5] we think that our patient's survival for almost 50 years without valvular dysfunction is noteworthy.

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## Evaluating Coronary–Cameral Fistulas

*To the Editor:*

Having read the interesting case report by Banerjee and Patra[1] in the August 2016 issue of the *Journal*, we wish to contribute to their discussion on coronary–cameral fistulas (CCFs).

Acquired CCFs may be iatrogenic or post-traumatic. For a CCF to be classified as acquired, it must not have been present at birth, it must not have been seen on prior angiographic images, and its cause must be known. Potential causes are previous myocardial infarction; a diagnostic or interventional endovascular procedure, such as percutaneous transluminal coronary angioplasty or stenting; aortic or mitral valve surgery; coronary artery bypass grafting; congenital heart surgery for tetralogy of Fallot, ventricular septal defect (VSD), double-chambered right ventricle, or transposition of the great arteries with VSD; septal myectomy in the treatment of hypertrophic cardiomyopathy; chest trauma; permanent pacemaker implantation; and complications after endomyocardial biopsy.[2,3]

Although most CCFs are asymptomatic, the size of the fistula and severity of the left–right shunt might cause symptoms. Clinical findings include a continuous cardiac murmur; difficulty breathing; exertional dyspnea; high-flow heart failure caused by the left–right shunt (except when fistulas open to the left ventricle [LV]); pulmonary hypertension; cardiac arrhythmias; stroke; endocarditis; chest pain or myocardial infarction caused by coronary steal; cardiac tamponade from fistula aneurysm, dissection, or rupture; and sudden cardiac death.[2-4] Because the left–right shunt fraction can be restricted by high intracavitary pressure, substantial hemodynamic changes might not be observed when CCFs open to the LV. However, ischemic chest pain or myocardial infarction might develop in these patients because of coronary steal.[4]

Regardless of size, CCFs carry a risk for endocarditis.[4] Fistulas that appear to be relatively harmless can lead to congestive heart failure caused by progressive and increasing left–right shunts, LV dysfunction and volume overload, aneurysms, myocardial ischemia, or endocarditis. For these reasons, asymptomatic CCFs should be treated.[2-4]

The treatment choice depends on whether the CCF is congenital or acquired. Whereas multiple congenital coronary–ventricular microfistulas can be treated medically with β-blockers or calcium channel blockers, large solitary macrofistulas that lead to severe hemodynamic shunts (Qp:Qs ratio, 1.5) should be closed by transcatheter or surgical means.[2-4]

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