Noninvasive Diagnosis of Left Ventricular Outflow Tract Obstruction Caused by a Porcine Mitral Prosthesis

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A patient developed shortness of breath 8 years after mitral valve replacement with a porcine prosthesis. Doppler echocardiography revealed left ventricular outflow tract obstruction created by a protruding prosthesis.

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Doppler echocardiography has proved to be a valuable noninvasive method for studying intracardiac flows. Currently available techniques allow the measurement of valvular gradients as well as left ventricular outflow tract gradients in patients with obstructive hypertrophic cardiomyopathy (1). We report a case of left ventricular outflow tract obstruction created by a porcine mitral valve prosthesis that was diagnosed by Doppler echocardiography.

Case Report

A 61 year old woman was admitted to New York University Medical Center with progressive shortness of breath. Eight years earlier, symptomatic mitral stenosis was detected and she underwent mitral valve replacement with a Hancock porcine prosthesis. She did well postoperatively until 6 months before admission, when she complained of mild shortness of breath that lessened after diuresis. However, over the 2 months preceding admission, she experienced progressive dyspnea.

On admission, a prominent apical impulse was noted for the first time, and a previously soft apical systolic murmur had increased markedly in intensity. Chest examination revealed rales in the lower two-thirds of both lung fields. The chest X-ray film demonstrated venous congestion and interstitial edema. The electrocardiogram showed normal sinus rhythm with left atrial enlargement.

Echocardiographic studies. Two-dimensional and M-mode echocardiography demonstrated biatrial enlargement and a thickened aortic valve that opened normally but exhibited mid-systolic closure (Fig. 1). In addition, a strut of the porcine prosthesis protruded into the left ventricular outflow tract and appeared to make contact with the interventricular septum during systole (Fig. 2). The left ventricular internal dimension, wall thickness and systolic function were normal.

Pulsed Doppler echocardiography revealed mild tricuspid and aortic insufficiency, but no evidence of mitral stenosis or mitral insufficiency. Continuous wave Doppler recording demonstrated a peak left ventricular outflow tract flow velocity of 3.7 m/s suggesting a maximal instantaneous outflow tract gradient of 54 mm Hg (Fig. 3). The calculated mean gradient was 39 mm Hg. The flow velocity curve exhibited a less gradual increase in velocity during systole than that characteristically seen with dynamic left ventricular outflow tract obstruction. In view of the normal aortic valve opening, the gradient was ascribed to subaortic valvular stenosis created by the mitral prosthesis, and the patient was referred for cardiac catheterization.

Cardiac catheterization. The study confirmed mild aortic regurgitation. Initially, no left ventricular outflow tract gradient could be demonstrated on pullback from the left ventricle to the aorta. However, in view of the echocardiographic findings, the patient underwent transseptal catheterization. Simultaneous recording of the left ventricular inflow tract (by anterograde transprosthetic valve catheterization) and left ventricular outflow tract (by retrograde left heart catheterization) confirmed a 60 mm Hg peak gradient across the left ventricular outflow tract. There was no evidence of mitral stenosis, mitral regurgitation or valvular aortic stenosis.

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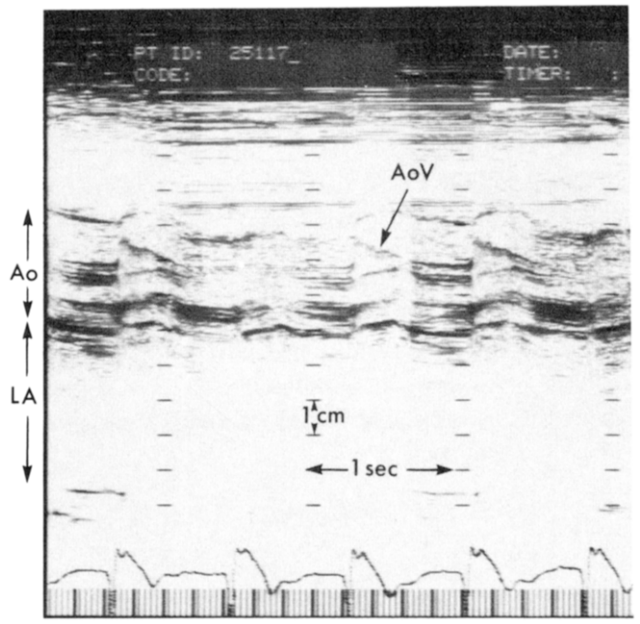


Figure 1. M-mode echocardiogram demonstrating mid-systolic closure of the aortic valve (AoV) (**arrow**). Ao = aorta; LA = left atrium.

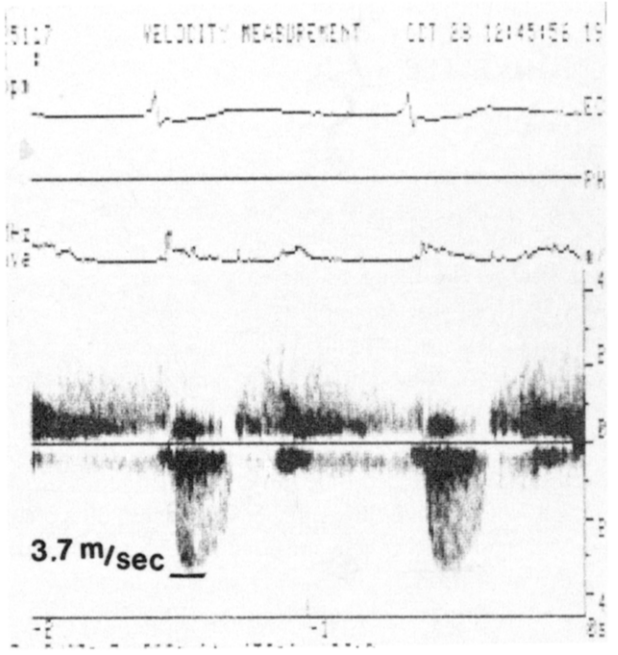
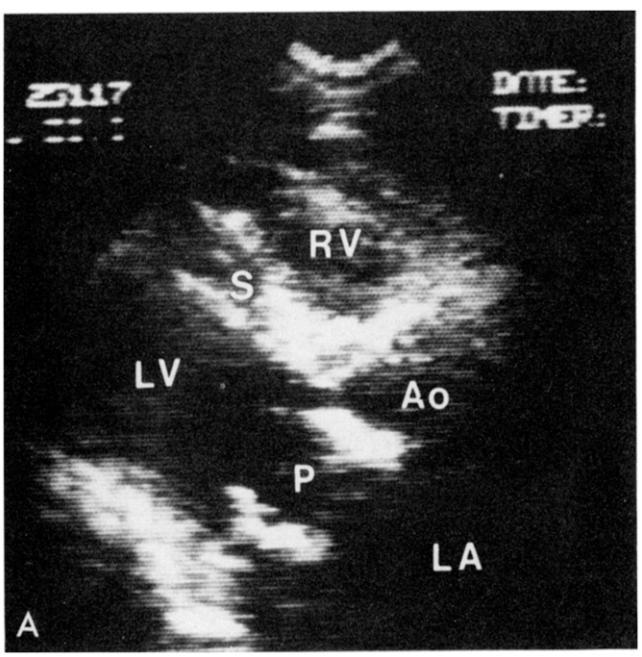


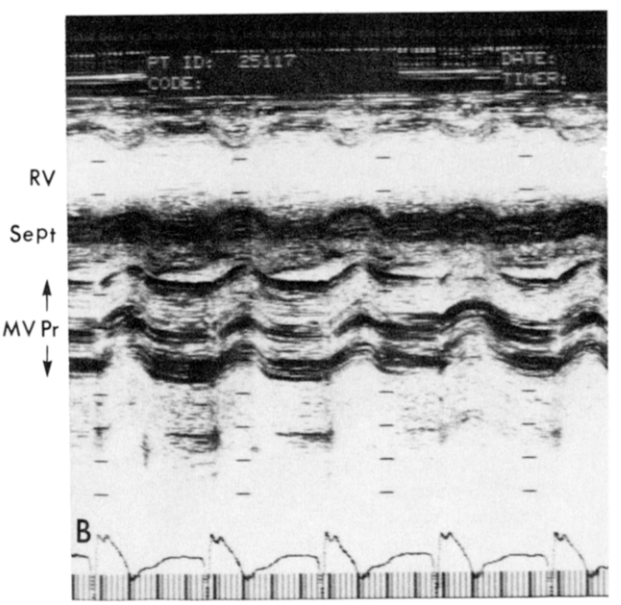
Figure 3. Continuous wave Doppler recording from the apex demonstrating a high velocity jet across the left ventricular outflow tract.

Operative findings and course. The patient was taken to the operating room for replacement of the porcine mitral valve with a low profile mechanical disc prosthesis. At surgery, there was fibrous ingrowth on the sewing ring of the bioprosthesis and its leaflets, but no mitral valve incompetence was detected. One strut, protruding into the left ventricular outflow tract, appeared to have incited a fibrous reaction in the adjacent interventricular septum. Digital examination of the septum at this site revealed a hypertrophic scar-like ridge. The mitral valve was replaced with a Björk-Shiley prosthesis.

Postoperative Doppler echocardiography demonstrated normal aortic valve excursion without mid-systolic closure, mild aortic regurgitation and a maximal instantaneous left

Figure 2. Parasternal long-axis two-dimensional (**A**) and M-mode (**B**) echocardiograms demonstrating systolic contact between the porcine mitral strut and the interventricular septum. (S, Sept). Ao = aorta, LA = left atrium, LV = left ventricle, RV = right ventricle; MV Pr and P = mitral valve prosthesis.





ventricular outflow tract gradient of only 15 mm Hg. The patient's symptoms of pulmonary congestion lessened.

Discussion

Previous reports. Left ventricular outflow tract obstruction is a recognized postoperative complication of mitral valve replacement, especially when a high profile valve is used (2–5). The relative frequency of mitral prosthesis-induced left ventricular outflow tract obstruction, particularly in patients undergoing valve replacement for mitral stenosis in which the left ventricle remains small, was an impetus for the development of low profile valves. Even these valves, however, have not prevented the phenomenon of outflow tract obstruction. Currie et al. (6) reported such a patient with a low profile Lillehei-Kastor mitral prosthesis and persistent congestive heart failure. Cardiac catheterization demonstrated a significant left ventricular outflow tract gradient; at surgery, the mitral prosthesis was found to be the cause of the gradient. Although a mechanism by which a porcine mitral prosthesis may cause obstruction, involving protrusion of the anterior portion of the stent into the left ventricular outflow tract with the tip abutting the interventricular septum, has been described briefly (7), we could not find additional clinical descriptions of this phenomenon attributed to porcine mitral valves.

Conclusion. The patient's progressive congestive heart failure was initially thought to be due to malfunction of the prosthetic valve, particularly in the face of a changing systolic murmur many years after replacement. However, she was shown convincingly, by a noninvasive technique, to have left ventricular outflow tract obstruction created by a normally functioning mitral prosthesis. Doppler echocardiography proved crucial to the correct diagnosis and more sensitive than routine cardiac catheterization in ascertaining the nature of the lesion. The noninvasive findings led to the nonroutine transseptal catheterization procedure that confirmed the subaortic valvular obstruction. Because of the fixed nature of the obstructing lesion, involving both a strut of the prosthesis and fibrous adhesions in the interventricular septum, it is not surprising that the retrograde catheter could not be passed across the obstruction. Doppler echocardiography is useful in the evaluation of prosthetic valve function. We suggest that evaluation include outflow tract hemodynamics in patients with a prosthetic mitral valve, particularly when there is clinical evidence of ongoing or recurrent congestive heart failure.

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