

zlocillin, and cefaperazone, but more detailed information is required.

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Anesthesiology  
72:563-566, 1990

## Severe Dynamic Left Ventricular Outflow Tract Obstruction Following Aortic Valve Replacement Diagnosed by Intraoperative Echocardiography

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Left ventricular hypertrophy may develop as a consequence of longstanding aortic stenosis. In patients with severe fixed aortic valvular stenosis, secondary left ventricular hypertrophy may also cause dynamic left ventricular outflow tract obstruction.<sup>1,2</sup> Recovery from aortic valve replacement may be complicated by the development of symptomatic outflow tract obstruction presenting weeks to months after surgery. We report a case in which severe dynamic left ventricular outflow tract obstruction developed in the immediate postoperative period in a patient who underwent aortic valve replacement for severe aortic stenosis. Intraoperative epicardial echocardiography was used to make the diagnosis and guide management.

#### CASE REPORT

A 71-year-old woman was referred to the Cleveland Clinic Foundation where she presented with the recent onset of congestive heart failure. She had a history of hypertension, but was otherwise free of previous cardiac symptoms.

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Key words: Heart: aortic stenosis; hypertrophic cardiomyopathy. Measurement technique: color flow doppler. Monitoring: echocardiography.

On admission, clinical features of severe aortic stenosis were present, including a grade III/VI harsh systolic ejection murmur at the right upper sternal border. Preoperative EKG showed normal sinus rhythm, right bundle branch block with a left anterior hemiblock, and Q waves in V<sub>4</sub>-V<sub>6</sub> and AVL. Preoperative 2-D/Doppler/echocardiography demonstrated severe concentric left ventricular hypertrophy; left ventricular chamber size and systolic function were normal. The aortic valve was heavily calcified with a maximum Doppler derived gradient of 125 mmHg and a Doppler estimated aortic valve area of 0.65 cm<sup>2</sup>. Cardiac catheterization showed mild (+1) aortic insufficiency and no significant coronary artery disease.

Because of severe aortic stenosis with recent congestive failure, the patient underwent aortic valve replacement with a 21-mm Carpentier-Edwards porcine bioprosthesis. Anesthetic management included fentanyl (75 µg/kg), diazepam, pancuronium, and metubine. Intraoperative monitors included a central venous catheter inserted prior to induction, and a left atrial catheter inserted prior to separation from cardiopulmonary bypass. Total bypass time was 114 min, and cross-clamp time was 74 min. The patient separated easily from cardiopulmonary bypass without inotropic support or vasodilators. On arrival in the intensive care unit (ICU) the mean arterial pressure (MAP) was 86 mmHg, central venous pressure (CVP) was 16 mmHg, and left atrial pressure (LAP) was 14 mmHg; nitroprusside was initiated and titrated to keep MAP 70-90 mmHg. Initial urine output was 80 ml/h. Approximately 8 h postoperatively, her urinary output decreased to 25 ml/h with CVP and LAP unchanged. Dopamine (4 µg · kg<sup>-1</sup> · min<sup>-1</sup>) was begun for renal effect. Urine output did not improve and CVP and LAP began to increase. As ionized calcium was low, a bolus of calcium (500 mg CaCl<sub>2</sub>) was given, and a dobutamine infusion was started at 5 µg · kg<sup>-1</sup> · min<sup>-1</sup>. At this time, MAP declined precipitously to 55 mmHg in spite of discontinuing nitroprusside, and the CVP and LAP rose to 20 and 44 mmHg, respectively.

These hemodynamic developments, coupled with decreased chest tube drainage, led to a provisional diagnosis of cardiac tamponade and prompted a return to the operating room for re-exploration of the mediastinum. Upon opening the chest, there was no blood in the pericardial cavity and no significant bleeding was found. The heart was not enlarged and appeared to be contracting vigorously. Boluses of epinephrine were given and an infusion was started to maintain MAP over 60 mmHg. A pulmonary artery thermistor was inserted and the

initial cardiac output was 3.4 l/min with a heart rate of 118 beats per min. Hypotension persisted, prompting further inotropic support with epinephrine, while the dopamine dose was increased to 15  $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ . LAP, however, remained high (peak of 52 mmHg) and either prosthetic valve dysfunction or acute mitral regurgitation (MR) was suspected at this point. Intraoperative echocardiography was performed as preparations to initiate cardiopulmonary bypass were underway.

The intraoperative epicardial echo/Doppler study revealed a left ventricular cavity of normal size with vigorous systolic function. There was severe left ventricular hypertrophy (LVH) with some asymmetrical septal hypertrophy. Severe systolic anterior motion (SAM) of the anterior mitral valve leaflet was noted producing dynamic left ventricular outflow tract (LVOT) obstruction (fig. 1a) with a maximum LVOT gradient of 64 mmHg. There was severe mitral regurgitation by color flow Doppler (fig. 1b). The planes of epicardial echo/Doppler interrogation that were used for this exam are illustrated in figure 2. The Carpentier-Edwards aortic prosthesis appeared to be functioning appropriately with normal leaflet motion and no aortic regurgitation detected.

The intraoperative echo demonstrated the etiology of the patient's postoperative hemodynamic deterioration as dynamic LVOT obstruction due to systolic anterior motion of the mitral valve with associated severe mitral regurgitation. In retrospect it was clear that the patient's hemodynamic compromise was aggravated by progressively increasing inotropic support. The pathophysiology was similar to that seen with *denovo* hypertrophic obstructive cardiomyopathy, necessitating measures to decrease contractility, increase preload, and increase afterload to keep the outflow tract open in systole and allow anterograde left ventricular ejection.

The dobutamine, dopamine, and epinephrine infusions were discontinued and a brief trial of substituting norepinephrine for epinephrine was attempted with no improvement in hemodynamics. The norepinephrine infusion was discontinued and esmolol and phenylephrine infusions were started, while administering 5% albumin and packed red blood cells. Heart rate decreased from 122 to 84. Initial efforts to increase afterload with phenylephrine were accompanied by right ventricular dilatation, and CVP rose to 28 mmHg (equal to LAP at this point) as cardiac index dropped to 1.9 l/min. This prompted switching the phenylephrine infusion to the LA catheter with a subsequent increase in MAP to 100 mmHg, and improved RV function as visualized in the surgical field. CVP decreased to 7 mmHg while LAP decreased to 24 mmHg over a period of 30 min. A second echo exam was performed at this point, showing a significant decrease in SAM severity, a maximum LVOT/aortic valve velocity of 3 m/s (normal for a 21-mm Carpentier-Edwards valve), and only minimal mitral regurgitation by color flow Doppler imaging (fig. 1c).

The patient was returned to the ICU in stable condition and the

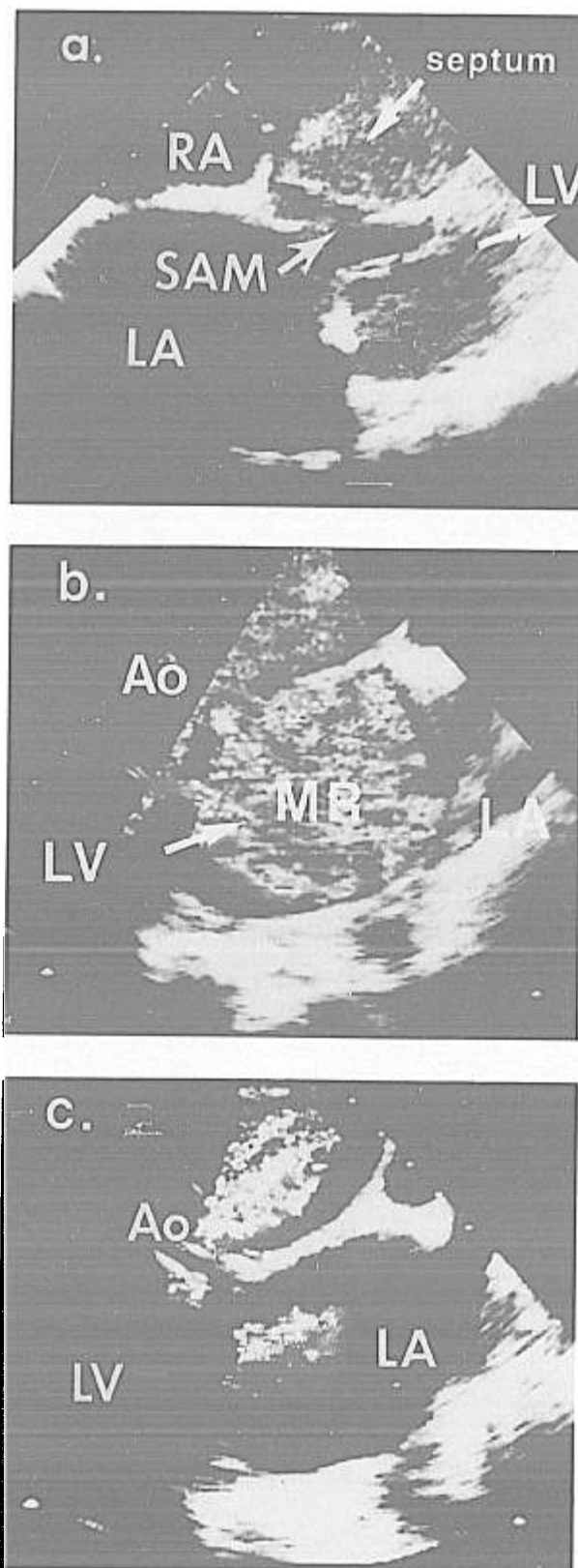


FIG. 1. Epicardial 2-D echo/Doppler views from a subcostal window (see fig. 2 for schematic demonstrating plane of interrogation) at the time of diagnosis (a) demonstrates systolic anterior motion (SAM) of the anterior mitral leaflet and occlusion of the left ventricular outflow tract, the interventricular septum appears thickened. Similar epicardial transaortic window views (see figure 2 for demonstration of this plane) of the left atrium (LA) demonstrate the marked difference in severity of mitral regurgitation as evidenced by a large multicolored aliased jet (MR) encompassing the entire left atrium (LA) at the time of diagnosis (b); and a marked reduction in the size of the jet after withdrawal of inotropic agents and institution of appropriate medical therapy for dynamic left ventricular outflow tract obstruction (c).

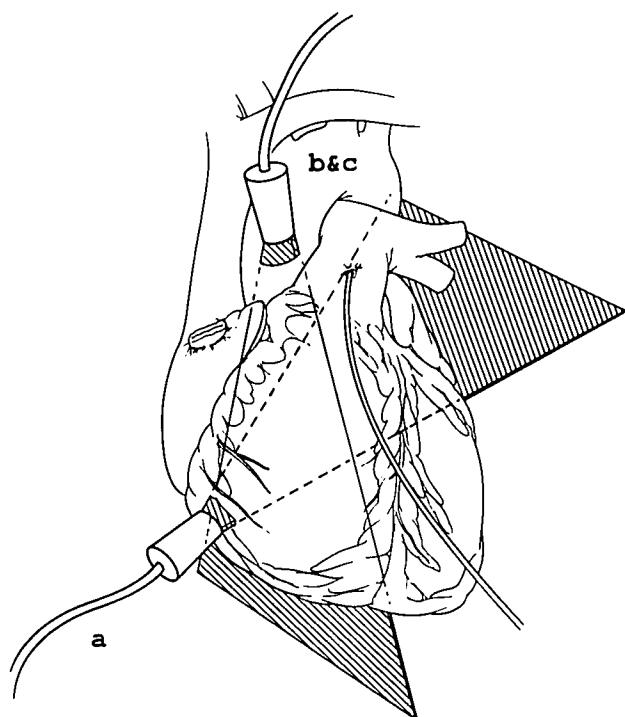


FIG. 2. Schematic drawing demonstrating the planes of interrogation used in the epicardial echo/Doppler photographs in figure 1. Subcostal window view used in figure 1a to demonstrate systolic anterior motion (SAM) of the mitral valve (a). Aortosuperior vena caval sulcus view used in figures b and c to visualize the left atrium (LA) and demonstrate the relative severity of mitral regurgitation (MR) (b and c).

esmolol and phenylephrine infusions were slowly withdrawn over the next 36 h. Extubation of the trachea was accomplished on the second postoperative day. A persistent third-degree AV block was present postoperatively requiring insertion of a permanent pacemaker on the fourth postoperative day. An Echo/Doppler study done on the 11th postoperative day revealed severe LVH with normal systolic function, mild (1+) MR, no systolic anterior motion of the mitral valve, and normal prosthetic valve function. Doppler flow velocity in the LVOT was normal, ruling out significant obstruction. She was discharged on the 15th postoperative day on verapamil (80 mg tid), propranolol (10 mg qid), coumadin (2 mg qd), and a thiazide diuretic.

#### DISCUSSION

The association of aortic stenosis and hypertrophic cardiomyopathy is well recognized in the literature.<sup>1-5</sup> While hypertrophic obstructive cardiomyopathy may occur with aortic stenosis as a separate entity,<sup>3,4</sup> it is far more likely that dynamic left ventricular outflow obstruction occurs in patients with severe aortic valve stenosis as a result of secondary left ventricular hypertrophy in response to the chronically elevated left ventricular afterload presented by the stenotic valvular lesion.<sup>1-3</sup> Preoperatively, the diagnosis of significant subvalvular obstruction may be quite difficult since the hemodynamic findings

may be masked by the coexistent aortic stenosis.<sup>4,5</sup> The echocardiogram may show asymmetrical hypertrophy of the septum (septum: free wall of LV ratio > 1.6), SAM of the mitral valve and a narrow LVOT dimension.<sup>5</sup> These findings may not be apparent unless provocative maneuvers (amyl nitrite, isoproterenol, Valsalva) are employed; however, severe valvular aortic stenosis is a relative contraindication to these maneuvers. Doppler echocardiography may be helpful in demonstrating an unusually increased velocity in the LVOT<sup>6</sup> (normally less than 1.0 m/s). This may be detected either by pulsed-wave Doppler interrogation showing an aliased signal, or by continuous-wave Doppler interrogation showing both LVOT and aortic valve velocities superimposed with a significantly higher-than-normal LVOT velocity. Cardiac catheterization may suggest the presence of hypertrophic obstructive cardiomyopathy when a subvalvular gradient is detected on careful pullback from left ventricular apex to ascending aorta or rarely when a gradient is demonstrated in the pulmonary outflow tract (RVOT obstruction by the bulging septum protruding into the RV).<sup>4</sup> In our patient, the preoperative evaluation did not show the findings described above, indicating that the subvalvular obstruction was latent, masked by her stenotic aortic valve.

Replacement of the aortic valve may worsen the obstructive pattern of dynamic LVOT obstruction; this may be due in part to the decrease in afterload of the LV, which unmasks the underlying functional obstruction.<sup>4,5</sup> The development of symptomatic dynamic LVOT obstruction has been reported to occur several months after aortic valve replacement; however, the presentation of dynamic LVOT obstruction acutely after valve replacement has not been reported. In retrospect, the clinical course in the early postoperative period was typical of dynamic LVOT obstruction. With the LV afterload reduced to normal by valve replacement, and the myocardium recovering from the effects of crossclamping the use of inotropic agents in the form of dopamine, dobutamine, and calcium precipitated functional obstruction. It is possible that timely application of transesophageal echocardiography in the ICU may have saved our patient a trip to the operating room to make the diagnosis, although the Doppler-derived flow velocity would have been more difficult to obtain with the less favorable angle of interrogation of the LVOT afforded by TEE.\*\*

The mitral regurgitation observed in the first echo was probably secondary to the SAM of the mitral valve and associated disruption of leaflet coaptation, since it dra-

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matically improved once correct therapy for the dynamic outflow obstruction was instituted. Once the diagnosis is made the appropriate therapy consists of maintaining left ventricular volume by augmenting preload and afterload, and withdrawal of stimulation to contractility.<sup>7,8</sup> The use of these measures in combination with esmolol to depress contractility corrected the dynamic LVOT obstruction and SAM but resulted in clinically significant right ventricular dysfunction. It was hoped that switching the phenylephrine infusion to the LA catheter would allow us to specifically target the  $\alpha$  agonist on the systemic vasculature, with systemic clearance resulting in less drug presented to the pulmonary vascular bed, as has been demonstrated with LA norepinephrine.<sup>††</sup> The return of the hemodynamic parameters to normal and the repeat echo provided us with confirmation of the efficacy of the treatment by demonstrating a decrease in LVOT gradient, decrease in SAM of the mitral valve, and reduced severity of mitral regurgitation. Although myomectomy at the time of aortic valve replacement has been advocated,<sup>9</sup> in our case this would have necessitated re-replacement of the aortic prosthesis. The risk of myomectomy was felt to be unwarranted at that time, particularly in light of the response to medical management.

In conclusion, we stress that a high index of suspicion of dynamic LVOT obstruction is warranted when a patient with long-standing severe aortic stenosis has hemodynamic deterioration early after aortic valve replacement

and fails to improve or worsens with inotropic treatment. Echo cardiographic exam performed at the bedside, if the patient's condition allows it, or intraoperatively as in our case, will provide the diagnosis and guide appropriate therapy.

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Anesthesiology  
72:566-570, 1990

### Prolonged Neuromuscular Blockade after Long-Term Administration of Vecuronium in Two Critically Ill Patients

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In some patients with severe respiratory failure, administration of neuromuscular blocking drugs may im-

prove gas exchange, probably by increasing chest wall compliance and improving ventilation/perfusion match-

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Received from the Department of Anesthesia, University of Cali-

fornia San Francisco, San Francisco, California. Accepted for publication October 24, 1989. Supported in part by NIH grant ROI GM 26403.

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Key words: Complications: prolonged neuromuscular blockade. Intensive Care. Monitoring: neuromuscular function. Neuromuscular relaxants: 3-desacetylvecuronium; vecuronium.