

# A CASE OF SEVERE SAM FOLLOWING A DAVID PROCEDURE

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## Abstract

We report a case of hemodynamically significant systolic anterior motion of the mitral valve following a David procedure. Although systolic anterior motion of the mitral valve has been reported following mitral valve repair or replacement and aortic valve replacement, it has not been previously described following isolated ascending aortic surgery.

## Introduction

Systolic Anterior Motion (SAM) of the mitral valve leaflets and chordae with associated turbulent flow through the left ventricular outflow tract (LVOT) as well as mitral regurgitation (MR) has been described following cardiac surgery particularly mitral valve repair or replacement<sup>1</sup> and aortic valve replacement<sup>2,3</sup>. It has not been previously described following isolated ascending aortic surgery. We present a patient who developed SAM with left ventricular outflow obstruction and mitral regurgitation following the David procedure<sup>4</sup>.

## Case Report

A 47-year-old gentleman with history of hypertension and a recent episode of chest pain and shortness of breath was found to have an ascending aortic aneurysm. Preoperative transthoracic echocardiography revealed a 5.7 cm ascending aorta and aortic root aneurysm. The aortic valve was trileaflet with moderate aortic valve insufficiency. No SAM and only trivial MR was seen (fig. 1). No significant subaortic gradient was measured. A valve sparing root replacement with coronary artery reimplantation as well as replacement of the ascending aorta with a graft (David procedure) was performed. Following separation from cardiopulmonary bypass, transesophageal echocardiography revealed normal biventricular function without the aid of inotropic support. However, turbulent flow was seen in the LVOT with a gradient of 64 mm Hg demonstrated by continuous flow Doppler with coexistent severe mitral regurgitation. SAM of the anterior mitral leaflet and part of the subvalvular apparatus was seen (fig. 2). This was associated with increasing pulmonary arterial pressures and profound systemic arterial hypotension (62/33 mm Hg). The patient was treated by decreasing the paced heart rate to 70 beats per minute, increasing the mean arterial pressure and volume expansion. A mean arterial pressure of greater than 80 mm Hg was required to prevent SAM with resolution of the LVOT obstruction and the MR.

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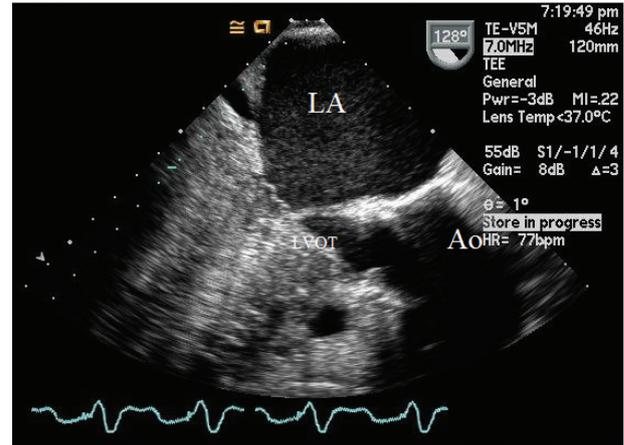
Fig. 1

Transesophageal echocardiography. Midesophageal aortic valve long axis view demonstrating an unobstructed left ventricular outflow tract. LA-left atrium, LVOT-left ventricular outflow tract, Ao-Ascending aorta



Fig. 2

Transesophageal echocardiography. Midesophageal aortic valve long axis view demonstrating an obstructed left ventricular outflow tract due to systolic anterior motion of the mitral valve. LA-left atrium, LVOT-left ventricular outflow tract, Ao-Ascending aorta



## Discussion

SAM of the mitral leaflets and chordae as seen in the midesophageal views on transesophageal echocardiography causing LVOT obstruction and MR is often found in patients with hypertrophic cardiomyopathy<sup>9</sup>. Other echocardiographic features of SAM include turbulent flow through the LVOT seen with color flow Doppler and high outflow velocity seen with continuous wave doppler which is usually described as late peaking. The mechanism by which this occurs has been described as a high velocity flow caused by the narrowed LVOT creating a Venturi effect on the mitral valve leaflets, causing the leaflets to move anteriorly thereby further narrowing the LVOT and causing obstruction<sup>1</sup>. It has also been described to occur after cardiac surgical procedures including mitral valve repair<sup>1</sup> and aortic valve replacement particularly for aortic stenosis<sup>2,3</sup>. A number of predictors of SAM after mitral valve surgery have been elucidated. These include an anterior leaflet length greater than 1.8 cm and less than 2.5 cm distance between the coaptation point and the septum<sup>5</sup>. The presence of SAM following AVR has been attributed to the increase in blood velocity through the LVOT once the obstruction due to the stenotic valve has been relieved and end systolic pressures fall<sup>6</sup>.

We believe that this is the first report of SAM occurring after a David procedure. While the valve is not replaced in a David procedure, it is reimplemented inside a tube graft<sup>3</sup>. This reimplantation may change the relationship between the aortic and mitral valves. Veronisi and colleagues<sup>11</sup> have described a phenomenon of mitral-aortic coupling which demonstrated that both valves have synchronous and reciprocal dynamic behavior. Further work showed that this relationship was altered by the placement of an annuloplasty ring on the mitral valve<sup>11</sup>. Perhaps performing a David procedure similarly alters this relationship and in so doing can lead to the development of SAM.

The cardiac anesthesiologist and the echocardiographer need to be able to anticipate SAM as its presence after cardiac surgery has been correlated with increased mortality<sup>12</sup>. The treatment of SAM is to increase both preload and afterload, thereby increasing left ventricular size, and preventing obstruction. This can be accomplished with the use of beta-blockers which both decrease heart rate increasing preload and decrease the force of left ventricular ejection, alpha-1 vasoconstrictors which increase afterload and volume expansion.<sup>2</sup> The use of positive inotropes, diuretics, or vasodilators needs to be avoided as much as possible.

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