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IMAGES IN INTERVENTION

Hypertrophic Obstructive Cardiomyopathy and Uncontrolled Hypertension

A Therapeutic Challenge

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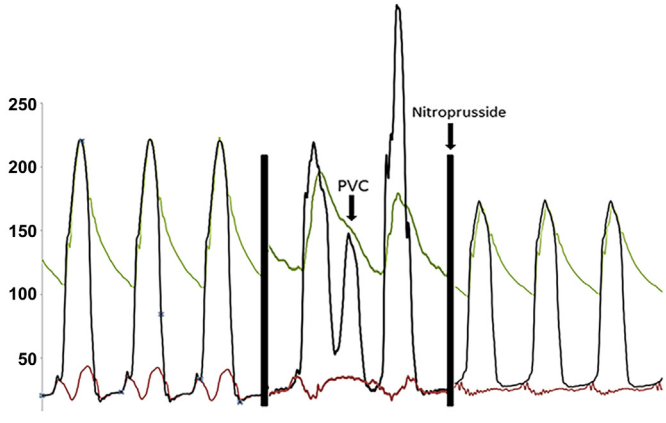
Hypertrophic obstructive cardiomyopathy and concomitant systemic hypertension can present a challenging diagnostic and therapeutic dilemma. Symptoms can occur from increased afterload from both dynamic outflow obstruction as well as the elevated systemic vascular resistance. Treatment of systemic hypertension with reduction in preload (diuretics) or afterload (vasodilators) may improve symptoms if the overall afterload on the left ventricle is reduced and also improve long-term outcome. However, treatment for hypertension may also exacerbate dynamic left ventricular outflow tract (LVOT) obstruction (1). Invasive hemodynamic assessment with nitroprusside challenge can be helpful in determining the response to medical management.

We present 2 patients with hypertrophic obstructive cardiomyopathy (Online Figures 1 and 2) and uncontrolled hypertension who presented with profound dyspnea despite maximally tolerated doses of beta-blockers. Cardiac catheterization was performed for further evaluation of their hemodynamics, using a transseptal approach to avoid catheter entrapment (2). There was severe elevation in left filling pressures in both cases with large V waves on the left atrial pressure trace. These V waves indicated impaired left

FIGURE 1 **Invasive Hemodyamic Tracing: Patient 1**



Hemodynamic tracing of patient 1 at baseline **(left)**, post-premature ventricular complex (PVC) **(middle)** and post-nitroprusside infusion **(right)**.

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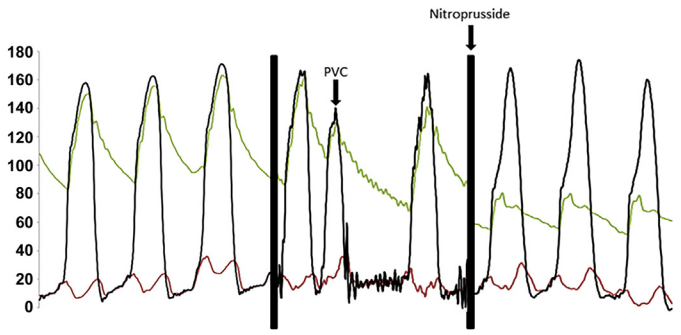
FIGURE 2 Invasive Hemodynamic Tracing: Patient 2



Hemodynamic tracing of patient 2 at baseline **(left)**, post-premature ventricular complex (PVC) **(middle)** and post-nitroprusside infusion **(right)**.

ventricular diastolic function as simultaneous echocardiography ruled out concomitant mitral regurgitation. Patient 1 (Figure 1) had severe systemic arterial hypertension with no resting LVOT gradient, but a dynamic gradient appeared after a premature ventricular contraction. With nitroprusside, both aortic and left ventricular systolic pressures decreased with no dynamic LVOT gradient and a decrease in mean left atrial pressure. Aggressive treatment of systemic hypertension was therefore recommended. Patient 2 (Figure 2) had similar baseline hemodynamics with minimal LVOT gradient at baseline but a dynamic gradient after a premature ventricular contraction. With nitroprusside, severe dynamic LVOT obstruction and a characteristic "spike-and-dome" configuration of the aortic pulse tracing developed in patient 2. Left atrial pressure remained elevated, indicating that afterload reduction would be limited by worsening of her dynamic obstruction. She underwent successful septal myectomy and subsequent treatment of systemic hypertension.

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KEY WORDS hypertension, hypertrophic obstructive cardiomyopathy, invasive hemodynamics

APPENDIX For supplemental figures, please see the online version of this article.