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Reversal of Left Ventricular Intracavitary Gradient With Intracavitary Diastolic Regurgitation in Hypertrophic Obstructive Cardiomyopathy

RICHARD G. TROHMAN, MD, FACC, ANDRES R. PALOMO, MD, NGAI X. NGUYEN, MD, ROBERT J. MYERBURG, MD, FACC, KENNETH M. KESSLER, MD, FACC

Miami, Florida

A 73 year old man presented with angina and nonsustained ventricular tachycardia. Cardiac catheterization revealed the dynamic systolic intracavitary gradient of hypertrophic obstructive cardiomyopathy. Abnormal isovolumetric relaxation resulted in the development of a diastolic gradient from the left ventricular outflow tract to the left ventricular apex accompanied by intracavitary regurgitation of contrast material from the outflow tract to the left ventricular body during left ventriculography. This case provides hemodynamic and angiographic confirmation of abnormal isovolumetric relaxation in this syndrome and insight into its mechanism.

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Dynamic systolic outflow tract obstruction and reduced diastolic compliance of the left ventricle are the characteristic findings of hypertrophic obstructive cardiomyopathy (1). Abnormally slow isovolumetric relaxation appears to play a role in reducing compliance (2,3). We report a new angiographic and hemodynamic finding during isovolumetric relaxation in an elderly patient with this syndrome.

Case Report

Clinical history and physical findings. A 71 year old white man was admitted to the coronary care unit because of short runs of nonsustained ventricular tachycardia. He reported progressive rest and nocturnal anginal chest pain. He had had dizziness for years and had experienced two syncopal episodes. He denied dyspnea on exertion, but reported the recent onset of pedal edema. Cardiac catheterization 21 years before admission had revealed hypertrophic obstructive cardiomyopathy. The status of his coronary arteries at that time was unknown. Risk factors included cigarette smoking and a family history positive for coronary artery disease.

From the Division of Cardiology, University of Miami School of Medicine and the Veterans Administration Medical Center, Miami, Florida.

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Address for reprints: Richard G. Trohman, MD, Electrophysiology Department, Veterans Administration Medical Center, Medical Service (IIIA), 1201 Northwest 16th Street, Miami, Florida 33125.

His past medical history was remarkable for three poorly documented myocardial infarctions, mild biopsy-proven hepatic cirrhosis thought to be secondary to non-A, non-B hepatitis, peptic ulcer disease and lumbar laminectomy. Medications included propranolol, 40 mg orally twice a day, sublingual nitroglycerin and acetaminophen as needed.

Physical examination revealed the apical impulse to be within the midclavicular line. It was not abnormally forceful or enlarged. A grade 2/6 systolic ejection murmur was heard in the aortic area and increased in intensity with the Valsalva maneuver. A grade 3/6 holosystolic murmur radiated from the apex to the axilla. A prominent fourth heart sound was heard. Carotid upstrokes were normal. No edema was noted.

Noninvasive studies. Electrocardiography revealed left axis deviation and left ventricular hypertrophy. The chest X-ray film suggested left ventricular prominence. M-mode and two dimensional echocardiography revealed a thickened, hypokinetic interventricular septum and systolic anterior motion of the mitral valve, compatible with hypertrophic obstructive cardiomyopathy.

Cardiac catheterization. Because of the history of hypertrophic obstructive cardiomyopathy, increased chest pain and apparent myocardial infarctions, cardiac catheterization was performed. Propranolol was discontinued 48 hours before catheterization.

Right and left heart catheterization was performed. An 8F USCI Sones catheter was advanced using a right brachial arteriotomy to the left ventricular apex. An 8F Cordis multipurpose catheter was passed percutaneously through the

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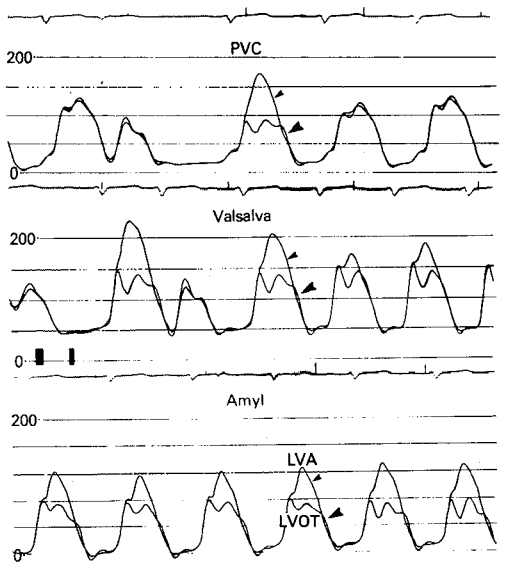

Figure 1. Left ventricular apical (LVA) pressures and systolic gradient are noted by **small arrows**. The left ventricular outflow tract (LVOT) pressures and reversed diastolic gradient are noted by **large arrows**. The findings are present after a premature ventricular contraction (PVC), during the Valsalva maneuver and with amyl nitrite inhalation.

right femoral artery (using the Seldinger technique) and positioned in the left ventricular outflow tract. Hemodynamics at rest and during the Valsalva maneuver, during amyl nitrite inhalation and with postextrasystolic potentiation were assessed. The multipurpose catheter was then withdrawn to the ascending aorta and the measurements were repeated.

The rest hemodynamics revealed no intracavitary or aortic valve gradient. Left ventricular end-diastolic pressure was 18 mm Hg. The coronary arteries were normal. A systolic intracavitary gradient of 50 to 80 mm Hg was reproducibly

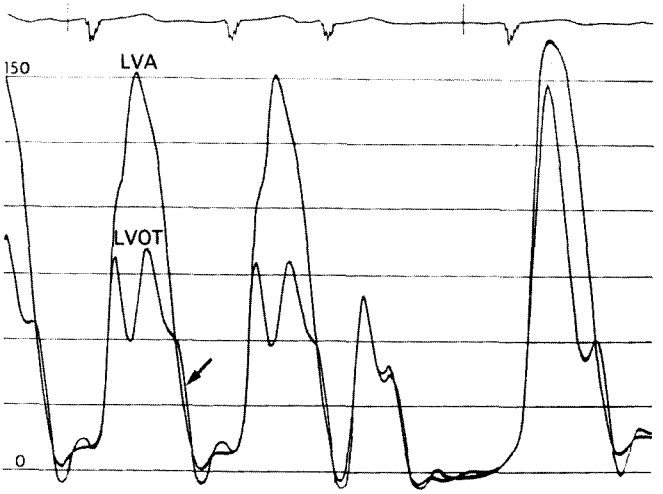

Figure 2. A diastolic gradient (**arrow**) is noted between the left ventricular outflow tract (LVOT) and the left ventricular apex (LVA).

present with all provocative techniques (Fig. 1). *This gradient reversed during isovolumetric relaxation* (Fig. 2) Hemodynamic features of catheter entrapment were absent (4).

Biplane left ventriculography was performed (Fig. 3). Systolic anterior motion of the mitral valve was clearly present. The most striking finding was the asynchronous isovolumetric muscular relaxation accompanied by "regurgitation" of angiographic contrast material from the left ventricular outflow tract into its body and apex. This "regurgitation" clearly preceded the opening of the mitral valve in early diastole.

Hospital course. The patient's clinical symptoms and nonsustained ventricular tachycardia resolved with adequate beta-adrenergic blockade and disopyramide therapy.

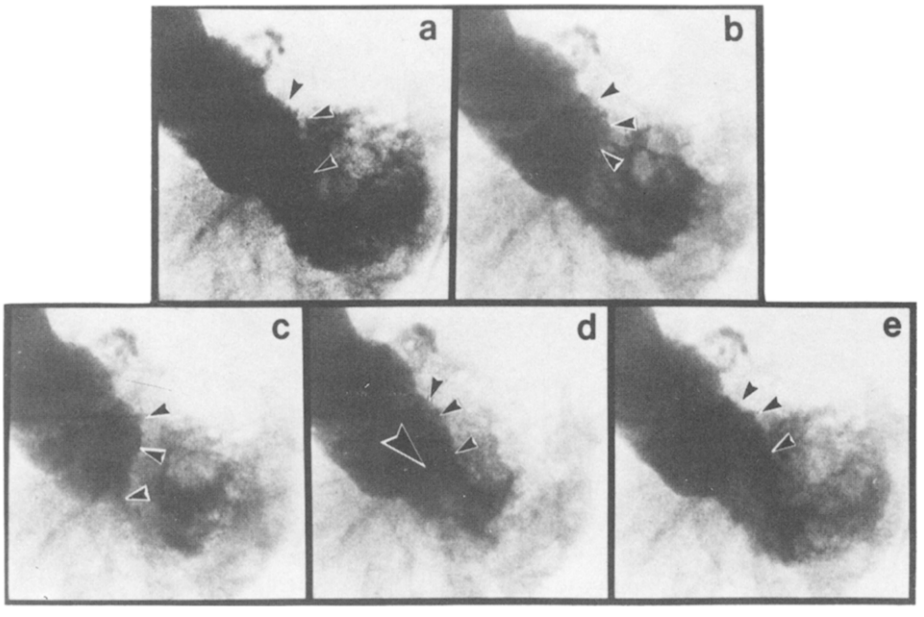

Figure 3. Left ventriculography (left anterior oblique position). **a**, End-diastole; **b**, early systole; **c**, systolic anterior motion of the mitral valve; **d**, isovolumetric relaxation with diastolic regurgitation (**large arrow**); **e**, initial diastolic opening of mitral valve. The position of the anterior leaflet of the mitral valve is outlined by **small arrows** during each portion of the cardiac cycle.

Discussion

Diastolic dysfunction in hypertrophic cardiomyopathy. Hemodynamic, echocardiographic and angiographic findings in hypertrophic obstructive cardiomyopathy are well described. Because the outflow tract obstruction is a systolic event, left ventricular ejection dynamics have been studied extensively (5,6). In addition, symptoms of pulmonary congestion are clearly correlated with diminished diastolic compliance of the hypertrophied ventricles. Diastolic dysfunction in hypertrophic obstructive cardiomyopathy has been attributed primarily to marked muscular hypertrophy associated with diminution in intraventricular cavity size.

It is well known that diastolic distensibility may be altered by retardation of the rate or other abnormalities of isovolumetric relaxation. Noninvasive studies have suggested that isovolumetric relaxation is prolonged in hypertrophic obstructive cardiomyopathy. The finding of intraventricular gradient reversal during early diastole expands our understanding of the diastolic dysfunction in this syndrome.

Mechanism. The systolic intraventricular gradients in our patient appear to be secondary to the classic separation of the left ventricle into high and low pressure zones. The intracavitary isovolumetric regurgitation found in our patient (with gradient reversal) suggests that isovolumetric relaxation may be significantly asynchronous. Here, the left ventricular body relaxes before the outflow tract and the slowest area of relaxation coincides with the region of outflow tract obstruction. Although the mechanism of this asynchrony and asymmetry of relaxation is uncertain, it is interesting to speculate that it relates to the abnormal, disarrayed, hypertrophied muscle found in this syndrome. Active relaxation may be less effective in these disorganized cells. Alternatively, the two chambers may be sensing different systolic loads and their load-dependent isovolumetric relaxation may differ.

Diastolic murmurs in hypertrophic obstructive cardiomyopathy are most often attributed to increased transmitral flow in patients with marked mitral regurgitation. Aortic insufficiency is rare unless there has been prior corrective surgery or bacterial endocarditis (7–9). Subvalvular regurgitation might explain the distolic murmur heard in some of these patients.

Implications. This finding has implications for the past and future study of diastolic function in this syndrome because, like systole, diastole does not appear to be necessarily homogeneous.

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