Editorial Comment

Hypertrophic Cardiomyopathy: When Do You Diagnose Midventricular Obstruction Versus Apical Cavity Obliteration With a Small Nonobliterated Area At the Apex of the Left Ventricle?*

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The present study. In this issue of the Journal, Nakamura et al. (1) describe an interesting and potentially important phenomenon in hypertrophic cardiomyopathy, that is, diastolic flow from an asynergic or aneurysmal apical cavity directed toward the base of the left ventricle through an area of midventricular narrowing (obstruction). This apex to base diastolic flow begins with isovolumetric relaxation, continues for almost 60% of diastole and is associated with a diastolic decrease in the size of the apical cavity. At the same time, base to apex mitral inflow is associated with an increase in the size of the basal part of the left ventricle. The apical cavity fills in late diastole, after cessation of the diastolic flow from apex to base and also during isovolumetric systole. During systolic ejection, systolic flow from apex to base is either abruptly halted by midventricular obstruction or attenuated in the latter half of systole.

Nakamura et al. (1) have prospectively examined 198 patients with hypertrophic cardiomyopathy for evidence of this phenomenon, which was originally described by Zoghbi et al. (2) and by the authors (3,4) in 1988 and 1989. There were 124 patients with asymmetric septal hypertrophy, 59 of whom had evidence of apical involvement. 54 patients with apical hypertrophy and 20 patients with diffuse or atypical hypertrophy. Fifty-five of these 198 patients had evidence of cavity obliteration and it is important to note that this was defined as ‘‘complete end-systolic cavity obliteration at the papillary muscle level in the short-axis view.’’ Twenty of the 55 patients with evidence of midventricular cavity obliteration had evidence of paradoxic diastolic jet flow, and in these patients the pattern of left ventricular hypertrophy by echocardiography was described as apical in 8, asymmetric septal and apical in 7, asymmetric septal only in 2, midventricular in 2 and diffuse in 1 (1).

Two-dimensional echocardiography detected no apical cavity in 13 of 20 patients, an apical cavity in 4 and an apical aneurysm in 3. Right anterior oblique left ventricular angiography in 16 of these 20 patients detected an apical aneurysm in 11, apical dyskinesia in 3 and apical akinesia in 2. In 4 of the 16 patients, special angiographic techniques were required to detect the apical cavity, which in cases of apical hypertrophy tended to be ‘‘a small outpouching connected to the main chamber by a narrow muscular tunnel that completely disappeared during contraction.’’ In contrast, ‘‘some [apical] aneurysms were so large that, together with the main chamber, they formed an hourglass-like ventricle,’’ and in three such patients, thrombus was detected in the aneurysm.

The importance of recognizing paradoxic diastolic jet flow by Doppler techniques, according to Nakamura et al. (1), is that it reveals the presence of an apical cavity that may be concealed, that is, not detected by echocardiography or even angiographic techniques. Patients with diastolic jet flow had a higher incidence of systemic embolism, ventricular arrhythmias and apical thallium perfusion defects than did the patients without this abnormality. However, Nakamura et al. (1) do not indicate whether these risks are present in all patients with this abnormal diastolic flow pattern or only in those with an apical aneurysm.

Midventricular obstruction with apical aneurysm. This study points out a number of unanswered questions in regard to apical hypertrophic cardiomyopathy and the syndrome of midventricular obstruction with or without apical infarction and aneurysm formation. Fixed or reversible apical thallium perfusion defects, or both, were reported in apical hypertrophic cardiomyopathy (1,5) and Webb et al. (5) described a patient with apical infarction with aneurysm formation in association with this entity. The postinfarction angigram in this patient showed an hourglass deformity with marked midventricular narrowing caused by a noninfarcted area of midventricular hypertrophy between the base and the aneurysmal apex of the left ventricle. Perhaps this is one way in which the syndrome of midventricular obstruction with apical aneurysm may develop (6,7). On the other hand, we (6,7) and others (8) described patients with midventricular obstruction whose condition progressed to apical infarction and aneurysm formation. However, many such patients first present with midventricular obstruction and apical aneurysm, leaving some doubt as to the pathogenetic mechanism responsible for the observed abnormalities.

A midventricular obstruction may be very long or very localized and the apical chamber may be very small or very large and aneurysmal. With this tremendous variation in angiographic appearance, it is sometimes difficult to know whether the term ‘‘midventricular obstruction’’ applies or whether it is a case of cavity obliteration in which a small

*Editorials published in Journal of the American College of Cardiology reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

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pocket of angiographic dye happens to remain at the apex. In time, perhaps criteria will be established for making this distinction.

**Midventricular obstruction versus apical cavity obliteration and catheter entrapment.** Another point worth commenting on is the difficulty in positioning a catheter in the apical chamber in patients with midventricular obstruction. In the present study (1) this was possible in only 3 of 16 patients. The difficulty arises in passing the catheter through the area of midventricular hypertrophy, which may be impossible or may initiate serious ventricular arrhythmias. Nakamura et al. (1) report that the elevated apical pressure decreases after the pressure decreases in the basal part of the left ventricle; thus, there is both a systolic and a diastolic pressure difference from apex to base, and the latter accounts for the paradox diastolic jet flow from the apical cavity through the midventricular obstruction toward the base, which is the focus of the present study. This late decrease in apical pressure must be distinguished from catheter entrapment in the myocardium, which is also associated with a late reduction in the elevated systolic pressure (9). The means by which these pressure differences may be distinguished from each other and from the outflow tract pressure gradient in hypertrophic cardiomyopathy were described previously (7,9,10). In the management of patients with obstructive hypertrophic cardiomyopathy a clear distinction must be made between outflow tract obstruction and midventricular obstruction (7,10), especially in patients who have both types of obstruction (7).

The observations of Zoghbi et al. (2) and Nakamura et al. (1,3,4) draw attention to the need to define what is midventricular obstruction (with or without apical infarction and aneurysm) in contrast to apical cavity obliteration in which a small cavity remains in an otherwise totally obliterated apex. This distinction is complicated by the fact that apical cavity obliteration may progress to midventricular obstruction after infarction of the hypertrophied apex. It will also be important to determine whether the increased risk of arterial embolism and ventricular arrhythmias is present in all patients with diastolic jet flow or only in those with an apical aneurysm. Thirty-five years ago Brock (11) and Teare (12) drew modern attention to hypertrophic cardiomyopathy. We continue to learn.

**References**