A New Strategy for the Attenuation of Left Ventricular Pressure Gradient in Patients with HOCM

Key words: hypertrophic obstructive cardiomyopathy, left ventricular pressure gradient, left ventricular epicardial pacing

A recent study elucidated that left ventricular pressure gradient (LVPG) is related even to the prognosis in patients with hypertrophic cardiomyopathy (HOCM) (1). Therefore, it is an important strategy to attenuate LVPG and to decrease mitral regurgitation in patients with hypertrophic obstructive cardiomyopathy (HOCM), especially in patients with idiopathic hypertrophic subaortic stenosis. Several strategies such as drugs (2, 3), surgical therapies (4), DDD pacing therapy (5, 6) and transcoronary ablation of the septal hypertrophy (7, 8) have been performed to attenuate LVPG. Both β blockers and calcium antagonists are effective in attenuating LVPG, but both are often insufficient. We previously reported that cibenzoline, which has little anticholinergic activity, could attenuate LVPG (3). Mainly in the United States of America, ventriculomyectomy is now performed. Transcoronary ablation of septal hypertrophy is performed worldwide to attenuate LVPG.

Several papers have reported that DDD pacing therapy could also attenuate LVPG in patients with HOCM. However, recent studies (9, 10) reported that DDD pacing was not always effective to attenuate LVPG in patients with HOCM. Ommen et al (9) reported that DDD pacing is not effective in most patients with HOCM, and Maron et al (10) reported that this method is effective only in old patients with HOCM. In this issue of the Journal, Yufu et al (11) reported the usefulness of left ventricular apex epicardial pacing in a 55-year-old women with LVPG of about 200 mmHg, whose LVPG was not attenuated by right dual-chamber pacing. They speculated the following mechanisms of efficacy of left ventricular apex epicardial pacing for the attenuation of LVPG in this patient. First, in this patient mid-ventricular hypertrophy was strong and the degree of LVPG of this region was markedly decreased. Second, the alterations of left ventricular wall motion by an inversion of left ventricular activation sequence may have been related to the marked decrease in LVPG. Although the precise mechanisms remain to be determined, it is certain that this provides a new method to attenuate LVPG in patients with HOCM.

Recent studies show that the elevation of intracellular Ca\(^{2+}\) in myocytes and activation of calcineurin in the cytoplasm are closely related to cardiac hypertrophy including patients with HCM (12, 13). Intracellular Ca\(^{2+}\) overload is also known to be closely related to diastolic dysfunction in HCM (14). In addition, it is suspected that the decrease in intracellular Ca\(^{2+}\) overload results in improvement of left ventricular diastolic dysfunction (3, 15) and attenuation of left ventricular hypertrophy (16). In view of these findings, we, cardiologists, look forward to future studies to elucidate whether or not the decrease in LVPG due to left ventricular apex epicardial pacing is associated with improvement of left ventricular diastolic dysfunction.

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References


