Dobutamine-induced midcavitary gradients do not cause dyspnea

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Introduction

Left ventricular outflow tract (LVOT) and midcavitary gradients are common during dobutamine stress echocardiography. Some consider these gradients to be of no clinical significance.[1] However, severe dobutamine-induced obstruction has been associated with chest pain and dyspnea,[2] abnormalities in ventricular wall motion,[3] and shock.[4] These associations suggest that dobutamine-induced gradients may have a clinical significance in some patients. Right ventricular (RV) apical pacing has been reported to reduce LVOT gradients in patients with hypertrophic cardiomyopathy[5] but is currently regarded as a class IIb indication for medically refractory patients with symptoms clearly attributed to obstruction and with unfavorable characteristics for septal reduction techniques. The role of RV apical pacing in symptomatic patients with midcavitary gradients during dobutamine infusion is less well defined.

Case presentation

A 78-year-old white male complained of having experienced breathlessness since 2010. Because his ability to exercise was poor, the patient underwent a dobutamine stress echocardiogram. The ventricle was normal in size, with an estimated left ventricular ejection fraction of 60%. Mild concentric hypertrophy was noted, with impaired relaxation based on mitral inflow pattern. As there was no left atrial dilation, this observation was attributed to age. Findings of hypertrophic cardiomyopathy including focal septal hypertrophy and mitral systolic anterior motion (SAM) were not observed. An infusion of 50 μg/kg/min of dobutamine resulted in a blood pressure of 167 mm Hg and a peak heart rate of 92 beats per minute. No wall motion abnormalities were noted. A delayed peaking Doppler

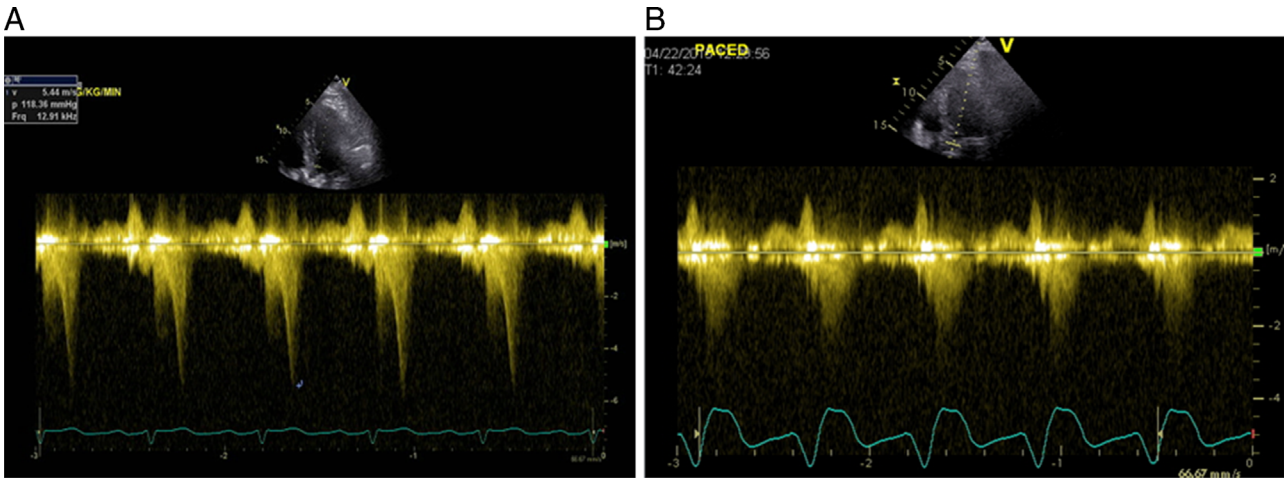

Figure 1 **A:** During infusion of dobutamine (5 mcg/kg/min) with a rate just under 1-bpm, a mid left ventricular gradient > 100mm Hg is noted by continuous wave Doppler. **B:** During ventricular pacing at 100 bpm, there is complete resolution of the midventricular gradient.

KEYWORDS Dobutamine stress echocardiography; Dynamic gradient; Left ventricular outflow tract obstruction; Pacing

ABBREVIATIONS **LVOT** = Left ventricular outflow tract obstruction; **RV** = right ventricular; **SAM** = systolic anterior motion

(Heart Rhythm Case Reports 2016;2:74–75)

Conflicts of interest: The authors have no conflicts of interest to declare.

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http://dx.doi.org/10.1016/j.hrcr.2015.10.010

KEY TEACHING POINTS

- Right ventricular apical pacing is currently regarded as a class IIb indication for hypertrophic cardiomyopathy for medically refractory patients with symptoms clearly attributed to obstruction and with unfavorable characteristics for the application of septal reduction techniques.
- Right ventricular apical pacing can eliminate severe midcavitary obstruction induced during dobutamine stress echocardiography.
- Elimination of severe midcavitary obstruction with right ventricular apical pacing, however, may not improve symptoms of dyspnea, and this therapy cannot be routinely recommended for patients with this symptom.

gradient of 180 mm Hg was observed without mitral SAM, which was consistent with transient midcavitary obstruction. The patient was treated with low doses of beta blockers and told to avoid volume depletion. Symptoms persisted. Results of a chest radiograph and pulmonary function tests were normal. The concentration of N-terminal pro-brain natriuretic peptide was 50.6 pg/mL. Coronary angiography was performed, with no lesion requiring intervention. An 18 mm Hg aortic valve gradient was recorded. Pulmonary pressures were not measured, because of the absence of significant tricuspid regurgitation; however, there was no indirect evidence for pulmonary hypertension. The patient walked 336 meters in 6 minutes.

The patient underwent a temporary dual pacemaker implantation. At rest, there was no midcavitary obstruction. During dobutamine infusion of 5 μg/kg/min, a 118 mm Hg dynamic gradient was recorded (Figure 1A). In the supine position, the patient was without symptoms. Atrioventricular pacing at 100 beats per minute immediately and completely abolished this gradient (Figure 1B). A permanent pacemaker was implanted, with the RV lead in the apex. After 2 weeks of follow-up, during which 100% ventricular pacing occurred, the patient continued to complain of dyspnea. He walked 371 m in 6 minutes. Currently, the device is programmed to minimize ventricular pacing.

This case suggests that dobutamine-induced midcavitary obstruction does not cause dyspnea.

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