DDD Pacing Therapy Could Serve as a Dual Purpose Treatment in Hypertrophic Obstructive Cardiomyopathy—A Case Report Which Suggests the Importance of Lead Position and the Mechanism—

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We treated a patient with hypertrophic obstructive cardiomyopathy (HOCM) who underwent DDD pacing therapy. He suffered from attacks of paroxysmal atrial fibrillation (PAF) complicated by sick sinus syndrome. Initially, we were unable to decrease the left ventricular outflow tract (LVOT) gradient by pacing from the mid-distal portion of the right ventricular (RV) septum. However, by changing the pacing site to the apical portion guided by right ventriculography, it was possible to decrease the LVOT gradient and at the same time reduce the mitral regurgitation. Tissue Doppler imaging (TDI) revealed a marked motion delay of the ventricular septum during DDD pacing. The mechanism of the therapy for HOCM provided by the DDD pacing was clearly confirmed by TDI. Furthermore, a dramatic effect of preventing symptomatic PAF with the use of overdrive pacing in the region of Bachmann’s bundle was also observed. This case report provides new insight into DDD pacing therapy for patients with HOCM.

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Introduction

Currently permanent pacemakers should be considered as an adjunctive therapy for HOCM. Although therapies such as percutaneous myocardial ablation and surgical manipulation effectively reduce the LVOT gradient, they carry a risk of mortality and morbidity. On the other hand, pacing therapy has is less invasive than the therapies mentioned above because it causes no rapid or irreversible anatomical changes. However, results of recent randomized studies have not been uniform and are considered controversial.

This case report describes a patient with HOCM in whom a reduction in the LVOT gradient was achieved with appropriate insertion of a right
ventricular (RV) lead guided by a hemodynamic study. Tissue doppler imaging (TDI) revealed a more marked motion delay of the ventricular septum during dual-chamber (DDD) pacing than atrial (AAI) pacing, and frequent paroxysmal atrial fibrillation (PAF) attacks were greatly suppressed by overdrive Bachmann’s bundle pacing.

**Case Report**

A 62-year-old man with a history of precordial chest pain during a PAF attack was admitted to our hospital. Drug therapy including beta-blockers (metoprolol 240 mg/day), cibenzoline (300 mg/day) and verapamil (120 mg/day) could not reduce the LVOT gradient or suppress the PAF attacks. In the 2 weeks that followed, he had a pre-syncopal episode. The ECG demonstrated a sinus pause after the PAF had terminated. The physical examination revealed a grade 4/6 systolic murmur, best heard from the apex. A 12-lead electrocardiogram (ECG) demonstrated sinus rhythm with left ventricular hypertrophy. Transthoracic echocardiography revealed asymmetric septal hypertrophy (interventricular septum, 23 mm vs. posterior wall, 19 mm), systolic anterior motion of the mitral valve, and moderate mitral regurgitation (MR). Continuous wave (CW) Doppler revealed a significant dynamic LVOT gradient of 103 mmHg. Implantation of a permanent DDD pacemaker was performed in order to maintain rhythm and to reduce the LVOT gradient.

During the cardiac catheterization the coronary arteriogram did not reveal any organic stenosis. Without discontinuing the oral drugs from the above description, a hemodynamic study was performed before the implantation. The left ventricular and aortic pressures were recorded simultaneously and the difference was used as a surrogate of the LVOT gradient. The LVOT gradient was maintained (110 mmHg) by pacing from the mid-distal portion of the RV septum (Figure 1). However, pacing from the apical portion of the RV guided by right ventriculography (RVG) reduced the LVOT gradient (to 44 mmHg) when the interval of the atrioventricular delay was set at 100 msec (Figure 2). Changes in the AV delay (from 80 to 150 msec) did not influence the LVOT gradient with the DDD pacing. After these measurements were made, separate pacemaker leads were inserted in the RA septum around Bachmann’s bundle (BB) and the RV apex region. The RV pacemaker lead, a model 5068 ‘CAPSUREFIX’ bipolar screw-in lead (Medtronic, Inc, Minneapolis, MN, USA), was advanced to the apical portion of the RV.

![Figure 1](image1.png)

**Figure 1** Simultaneous recordings of the left ventricular and aortic pressures.
A: Unpaced, pressure gradient of 113 mmHg.
B: Dual chamber (DDD) pacing, with the right ventricular (RV) catheter located in the mid-distal portion of the RV septum, pressure gradient of 110 mmHg.
C: The RV catheter is located in the mid-distal portion of the RV septum.
An echocardiographic examination performed 2 weeks after the pacemaker implantation revealed that the LVOT gradient was 38 mmHg and there was a mild degree of MR. All echocardiographic measurements were obtained using the Vivid 7 echocardiographic scanner (GE-Vingmed Ultrasound AS, Horten, Norway) with a 2.5-MHz transducer. We recorded TDI images of the 4-chamber views with AAI and DDD pacing, respectively. At least 3 consecutive beats were stored digitally for post-processing. The gain settings, filters, and pulse repetition frequency were adjusted to optimize the color saturation. The TDI data were analyzed using the software program provided with the echocardiographic machine (Tissue Tracking mode). TDI clearly revealed a motion change in the ventricular septum during DDD pacing rather than during AAI pacing (Figure 3).

After three-weeks of follow-up, the patient continued to have symptomatic PAF. Overdrive pacing in the region of BB (DDD minimum HR: 75bpm) could suppress the symptomatic episodes of the PAF, detected by the device-monitored diagnostics (Figure 4). After six-months of follow-up, the patient has remained asymptomatic.

**Discussion**

The key findings in the present case were as follows: (1) The first observation in this study was that a reduction in the pressure gradient was not achieved with the RV septal pacing; (2) The optimal RV pacing site guided by the RVG and hemodynamic study could improve the LVOT gradient with a reduction in the MR; (3) The overdrive BB pacing could prevent symptomatic PAF.

DDD pacing has been reported to be effective in HOCM patients by altering the pattern of the septal contraction and causing a delay in the onset of the mitral leaflet movement and septal contraction during right ventricular stimulation.5) In our patient, the indication for permanent pacemaker implantation was sick sinus syndrome. It was very important to choose an effective pacing site in order to decrease the LVOT gradient. The cardiac apex should be visualized with contrast by RVG because an overhang of the ventricular septum often makes positioning of the RV lead difficult. Further, the RV lead should certainly be inserted guided by a hemodynamic study to acquire the greatest effect of this therapy. The selection of the pacing site needs to be carefully evaluated.
Figure 3  Tissue Doppler imaging (Upper), 2-D color Doppler echocardiography (Lower) and continuous wave Doppler echocardiography (Lower) were recorded 1 month after the pacemaker implantation. The tissue Doppler images were recorded from the 4-chamber view with AAI and DDD pacing. The Doppler points (yellow, light blue) are positioned at the basal- and mid-septum, and the others (green, red) at the basal- and mid-lateral wall. TDI clearly revealed a greater motion delay in the ventricular septum during DDD pacing rather than during AAI pacing. The pressure gradient at the LVOT decreased from 123 mmHg to 38 mmHg. Note that the mitral regurgitation area visualized during the DDD pacing is dramatically reduced compared to that during AAI pacing (lower).

Figure 4  Overdrive pacing in the region of Bachmann’s bundle could suppress the symptomatic episodes of PAF, detected by device-monitored diagnostics.
TDI has been used to assess the regional cardiac function and synchrony before and after pacing.6) Recently, Ito et al.7) reported the efficacy of TDI for DDD pacing therapy in HOCM. In our case, TDI revealed a more marked motion delay in the interventricular septum during DDD pacing than it did during AAI pacing (Figure 3). This mechanism could be explained by the following. At first, the interventricular septum contracted earlier during the pacing stimulation than did the lateral wall (Figure 3 right upper panel), but the septum could not contract longitudinally due to its hypertrophy, and therefore the lateral wall contracted earlier than the septum. The dysssynchrony, that is to say the septum contraction during the early diastolic phase, was caused by the delayed longitudinal contraction that Sogaard et al. reported.8) We also found that DDD pacing reduced the MR in comparison to AAI pacing. The possibility was suggested that by directly altering the mitral valve complexes or improving the mitral valve systolic anterior movement by decreasing the Venturi effect, a reduction in the MR could be achieved (Figure 3).

Atrial fibrillation (AF) is the major cardiac arrhythmia in severe HOCM. The effect of BB pacing and overdrive pacing in the prevention of symptomatic PAF was reported.9,10) In the present case, the frequent PAF attacks were greatly suppressed by overdrive pacing in the BB region (Figure 4). The possibility of the synergistic effect of the reduction in the MR is also suggested. To the best of our knowledge, there have been no reports in which overdrive pacing in the BB region could suppress symptomatic episodes of PAF in HOCM. This therapy could serve a dual purpose, by providing a hemodynamic effect and preventing atrial tachyarrhythmias.

Pacing therapy may demonstrate a significant advantage over the other therapies in that a detailed hemodynamic study can be performed before the implantation, and the use of various drugs can be increased in the presence of bradycardia after the implantation. Further studies may be necessary in order to establish this therapy. We believe this “cardiac desynchronization therapy” for HOCM has further possibilities.

References