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A Case Report of Hypertrophic Obstructive Cardiomyopathy Using Short-term Temporary Pacing in the Evaluation of a Non-responder for Pacing Therapy

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Summary. **Dual-chamber (DDD) pacing is a therapeutic strategy for hypertrophic obstructive cardiomyopathy (HOCM), but some patients show deterioration after implantation of the DDD pacemaker. We report a case of HOCM with shortness of breath upon physical exertion. To evaluate the suitability of pacing therapy, the acute effects of DDD pacing and short-term results using temporary VDD pacing were examined. The acute study demonstrated a mild reduction in the pressure gradient within the left ventricle. After 1 week of VDD pacing, a left ventriculography showed apical dyskinesis, with no effect on subjective symptoms or the blood concentration of brain natriuretic peptide (BNP) observed. Newly developed asynergy may be caused by changes in the contractile pattern. The patient underwent a myotomy-myectomy. After the operation, her subjective symptoms disappeared, and the hemodynamic parameters and blood concentration of BNP improved compared with those before the operation. The response to 1-week temporary VDD pacing can provide valuable data such as hemodynamics and neurohormonal changes in distinguishing between responders and non-responders for pacing therapy.**

Key words—HOCM, VDD pacing, BNP, apical dyskinesis.

INTRODUCTION

It is well known that dual-chamber (DDD) pacing is effective for the treatment of symtomatic hypertrophic obstructive cardiomyopathy (HOCM)[1-5]. However, approximately 10-30% of patients who receive pacemakers experience no symtomatic relief or show deterioration[1,4,5]. Moreover, the characteristics of non-responsive cases have not been studied in detail. In clinical trials, acute effects of DDD pacing have been studied to predict a non-response to DDD pacing therapy, but the conclusions are controversial. Some reports state that acute studies are not necessary, because symptom improvement may occur after several months, even when no improvement is apparent in acute pacing studies[1,6]. We here describe a case showing no remarkable change in hemodynamic function during a 1-week period of VDD pacing in spite of mild improvement immediately after temporary DDD pacing therapy.

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CASE REPORT

A 71-year-old woman with a 3-year history of palpitation and shortness of breath upon exertion visited another clinic in 1988. Echocardiography showed asymmetric hypertrophy of the septum and systolic anterior movement of the mitral valve. The patient was admitted to our hospital to confirm the diagnosis of HOCM and for treatment.

On physical examination, a systolic murmur was audible from the second left-sternal-border area to apex. There was no pitting edema in the lower extremities. Her blood pressure was 114/70 mmHg. Results of blood tests, biological tests, and urinalysis were almost normal. However, her plasma concentrations of brain natriuretic peptide (BNP) and human

atrial natriuretic peptide (hANP), 1461 pg/ml and 356 pg/ml respectively, were much higher than normal. Electrocardiography showed sinus rhythm and left ventricular hypertrophy. On magnetic resonance imaging, the thickness of the intraventricular septum (IVS) was 2.7 cm and that of the free wall of the left ventricle was 1.5 cm (Fig. 1). Cardiac catheter examination showed left ventricular inflow and outflow tract pressures of 250 mmHg and 82 mmHg, respectively, and a pressure gradient (PG) of 168 mmHg (Table 1). The acute effects of DDD pacing were examined with various intervals of the atrioventricular (AV) sequence. The cardiac index (CI) remained unchanged and the intraventricular PG fell slightly (Table 1). To investigate the short-term effects of AV sequential pacing, a temporary VDD pacing lead was inserted via the left internal jugular vein of the neck. One week later, cardiac catheter examination was performed again. The reduction of the PG by VDD pacing was slight, at −9% (Table 1). Pulmonary capillary wedge pressure (PCWP) increased from 16 to 20 mmHg, and CI decreased from 3.0 to 2.7 L/min/m^2. Left ventriculography (LVG) showed dyskinesis of the apex, which was not apparent in the sinus rhythm (Figs. 2 and 3). VDD pacing did not affect the patient's subjective symptoms or the concentration of BNP (Table 1). In addition, paroxysmal atrial fibrillation occurred with dizziness and a decrease in systolic blood pressure (80 mmHg). We chose myetomy-myotomy of the IVS for the following reasons: 1) if atrial fibrillation becomes permanent, it is impossible for a DDD pacemaker to operate; 2) the DDD pacing had a deteriorative effect on hemodynamic parameters.

In Sepember, Marrow's operation was undertaken successfully, and the IVS was cut by 2 cm x 3 cm x 1 cm. After the operation, the patient's symptoms improved significantly, and amelioration of the hemodynamics and BNP concentration was seen (Table 1).

DISCUSSION

DDD pacing therapy for HOCM has been applied for some 30 years[7,8]. Many studies show beneficial results of this therapy[1-5]. However, it is also a fact that non-responders from DDD pacing amount to approximately 10~30% in each study. Thus, there is a need to establish an examination for distinguishing the non-responders from candidates for DDD pacing therapy in HOCM.

Before the implantation of a permanent pacemaker, an acute study with temporary pacing is usually carried out. The reduction of the pressure

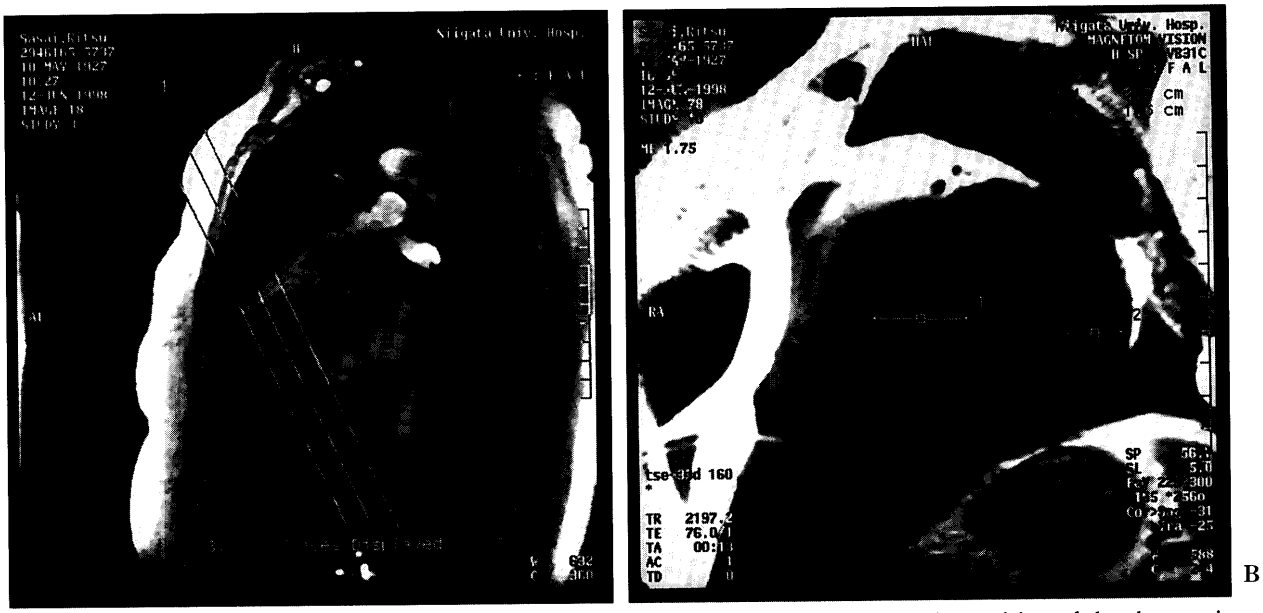

Fig. 1. Magnetic resonance imaging. (**A**) Sagittal view of the heart. Line 1 indicates the position of the short axis view (**B**). (**B**) Short axis view of the heart shows asymmetrical septal hypertrophy. Line 1 indicates the thickness of the intraventricular septum, 27 mm, and Line 2 indicates that of the free wall of the left ventricle, 15 mm.

Table 1. Hemodynamic parameters and blood BNP concentration in sinus rhythm (before and after myectomy) and pacing

	June 17, 98	June 17, 98				Aug. 5, 98	Oct. 14, 98
	Sinus rhythm	Dual chamber pacing AV sequence (ms)				VDD pacing AV sequence (ms)	Sinus rhythm after myectomy
		Before	80	120	160	100	
PCWP	(16)					(20)	(7)
PA	45/16(24)					47/17(31)	33/6(20)
RV	50/EDP=7					53/EDP=7	38/EDP=7
RA	(2)					(5)	(4)
LV (inflow)	250/EDP=23	255/EDP=28	212/EDP=18	219/EDP=20	231/EDP=18	244/EDP=23	131/EDP=12
LV (outflow)	82/EDP=23					92/EDP=21	
AO	84/51(73)	95/46(66)	82/51(65)	88/52(70)	95/54(72)	110/51(73)	138/64(93)
PG in LV(mmHg)	168	160	130[−23%]	131[−18%]	136[−15%]	152[−8%]	
CO (L/min)	4.2	3.6	3.8	3.8	3.8	3.7	4.4
CL (L/min/m^2)	3.0	2.6	2.7	2.7	2.7	2.7	3.2
LVEDVI (mL/m^2)	89					81	79
LVESVI (mL/m^2)	24					29	16
SVI (mL/m^2)	64					52	62
EF	72					64	79
BNP (pg/ml)	1461					1294	297

BNP, brain natriuretic peptide; AV, atrioventricular; PCWP, pulmonary capillary wedge pressure; PA, pulmonary arterial pressure; RV, right ventricular pressure; RA, right atrial pressure; LV, left ventricular pressure; AO, aortic pressure; PG, pressure gradient; CO, cardiac output; Cl, cardiac index; LVEDVI, left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index; SVI, stroke volume index; EF, ejection fraction; EDP, end-diastolic pressure. The numbers in paretheses are the mean pressures. The numbers in square brackets are peak gradient reduction of the PG.

gradient in the left ventricle is measured by cardiac catheterization or doppler echocardiography. However, the reduction of the pressure gradient between the acute phase and long-term phase is not always correlated. Long-term hemodynamic improvement in response to pacing therapy can be obtained in patients who do not show benefits of DDD pacing in acute studies[1,6]. It has been supposed that the acute study may be insufficient to evaluate non-responders.

One week of observation under a temporary pacemaker would be advisable for patients who are candidates for the implantation of a DDD pacemaker. There are several advantages to such a study over an acute study: the period allows us to assess changes in the hemodynamic parameters and the neurohumoral factors such as BNP. Recently, Skakai Y et al. reported the acute and chronic effect of DDD pacing therapy for HOCM[6]. They examined the reduction of PG in the acute phase in 14 patients, and showed a PG of 106 mmHg and 62 mmHg at control stages and after pacing, respectively. A permanent pacemaker was implanted in 12 of 14 patients, and the PG was measured at the control, 1 week, 6 months and 1 year stage: values were 98.5 mmHg, 21.4 mmHg, 20.8 mmHg, and 17.1 mmHg, respectively. Their observations clearly show that the reduction of PG at 1 year was different from that in the acute study, but almost the same as that at 1 week. A one week observation of pacing is considered to be a sufficient period to evaluate the non-responders. Moreover, in an acute study, the patient remains in bed, but after insertion of the temoprary pacing lead, the patient can have limited movement. This allows us to learn about changes in the subjective symptoms in the patient's daily life. We believe that the burden on the patient who undergoes pacing for one week is tolerable.

In addition, the measurement of neurohumoral factors yields important information regarding the effect of therapy for HOCM. In the present case, VDD pacing did not reduce the blood BNP concentra-

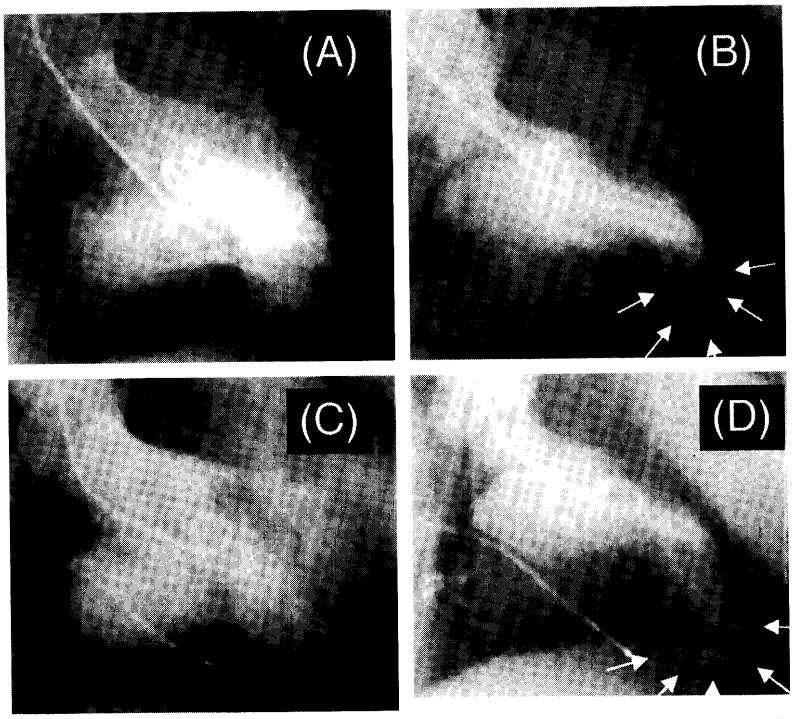

Fig. 2. Left ventriculography. (**A**) Diastolic phase and (**B**) systolic phase before VDD pacing. (**C**) Diastolic phase and (**D**) systolic phase after VDD pacing. White arrows show the outline of the apex in both the systolic and diastolic phases after VDD pacing to demonstrate dyskinesis of the apex.

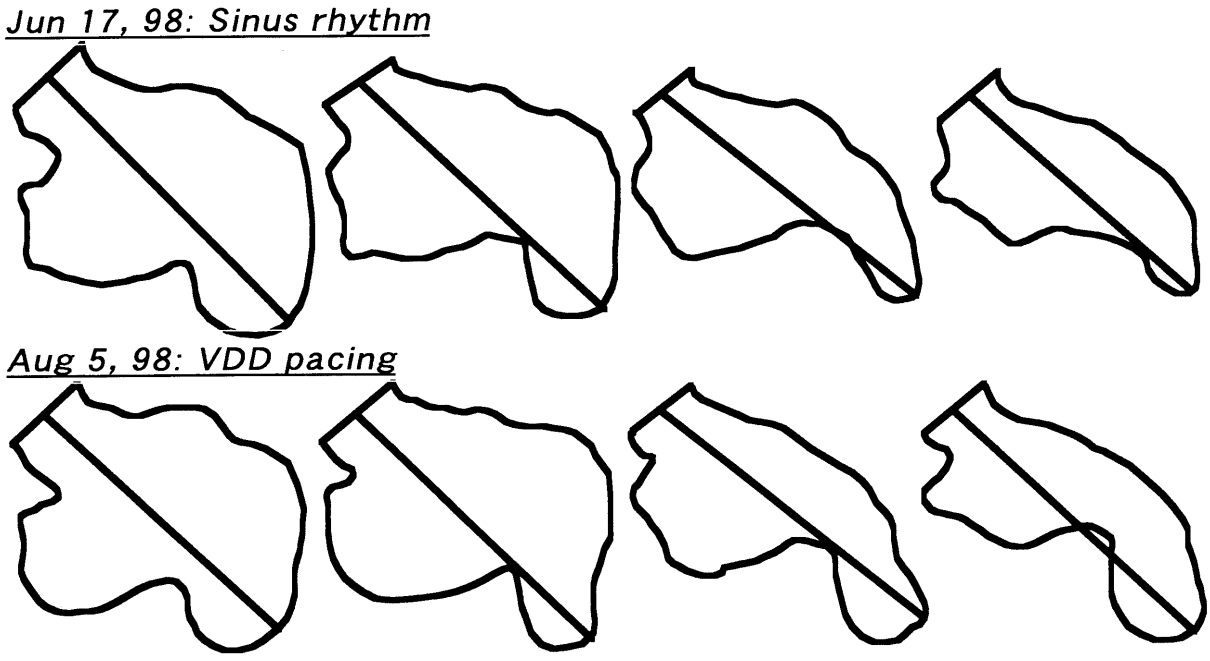

Fig. 3. Traces of left ventricular wall motion before and after VDD pacing.

tion, but after myotomy the BNP concentration fell significantly, and the patient's subjective symptoms improved (Table 1). Implantation of a permanent pacemaker has a placebo effect on symptoms in about 40% of patients[3,4]; however, we expect that the BNP level will be an objective indicator for evaluating the effectiveness of pacing theray. The BNP level is well known as a parameter that reflects the severity of symptoms and the prognosis in patients with heart failure[9].

Several papers[10-13] have reported on hemodynamic changes by DDD pacing during an acute study. In responders to DDD pacing, the CI and PCWP did not change significantly, or an increase of only PCWP was recognized. However, our case showed a decrease in CI and an increase in PCWP. According to these results and apical dyskinesis by DDD pacing, the implantation of a pacemaker was thought to be unsuitable for our case.

In our case, the pacing-induced dyskinesis of the left ventricle was considered to be undersirable, and hemodynamic parameters and the blood concentration of BNP did not improve dramatically after 1-week of pacing. Ultimately, pacing theapy was rejected. Thus, the 1-week observation of the effects of VDD pacing on hemodynamics and the measurements of the BNP level may provide an approach that identifies the non-responders to permanent DDD pacing.

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