Effect of Pacing Rate in Pacing Therapy in Hypertrophic Obstructive Cardiomyopathy

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The effects of dual-chamber pacing therapy in patients with hypertrophic obstructive cardiomyopathy (HOCM) have been reported in short- and long-term studies. Almost all of these studies have reported that the key factor in pacing therapy is the setting of the atrioventricular (AV) interval. However, studies focusing on the effects of pacing rate on the hemodynamic state are rare. In this study, cardiac catheterization was performed in 2 patients during temporary pacing at various rates and AV intervals. When the pacing rate was increased slightly (to 70–90/min), AV sequential pacing decreased peak subaortic pressure gradient and increased systolic aortic pressure without increase in pulmonary capillary wedge pressure, left ventricular end-diastolic pressure, and the time constant of isovolumetric relaxation. In another case, of a patient who became refractory to AV sequential pacing therapy at an optimum AV interval, pacing at a slightly higher rate relieved syncope. Thus, AV sequential pacing therapy performed at a slightly higher rate than normal in a patient with HOCM may lead to a decreased subaortic pressure gradient and relief of symptoms without noticeable deterioration in cardiac function. (*Jpn Circ J* 1998; 62: 546–548)

Key Words: Hypertrophic obstructive cardiomyopathy; Dual-chamber pacing; Pacing rate

The effects of dual-chamber pacing therapy in patients with hypertrophic obstructive cardiomyopathy (HOCM) have been reported in short- and long-term studies.^{1–4} Almost all of these studies reported that the key factor in pacing therapy is the setting of the atrioventricular (AV) interval. In this study, we examined the effect of pacing rate on acute hemodynamic changes in 3 patients with HOCM.

Case Report

Patient 1

A 40-year-old woman on beta-blockers and calcium antagonists was admitted to hospital with chest pain. Her blood pressure was 98/60 mmHg and heart rate 62/min. A grade 3/6 systolic murmur at the left sternal border and a grade 2/6 holosystolic murmur at the apex were observed. Twelve-lead electrocardiography revealed a prolonged QRS interval. Echocardiography demonstrated asymmetric septal hypertrophy, narrowing of the left ventricular outflow tract, abnormal systolic anterior motion (SAM) of the anterior leaflet of the mitral valve, and mitral regurgitation. Left ventriculography (LVG) demonstrated an abnormal “ballerina foot” configuration, anterior movement of the anterior leaflet during systole, and grade 2/4 mitral regurgitation. A coronary arteriogram (CAG) was normal. A cardiac catheterization study demonstrated that the peak-to-peak pressure gradient in the left ventricular outflow tract was 86 mmHg. A cardiac catheterization study during VVI or AV sequential temporary pacing (DDD mode) at various

pacing rates (from a sinus rate of 50 to a pacing rate of 90/min) and various AV intervals (from 60 to 100 msec) was performed (Fig 1). Each different pacing setting was maintained for 5 min to allow equilibration of hemodynamics. The ventricular electrode was placed as close to the right ventricular apex as possible. DDD mode pacing decreased the peak subaortic pressure gradient, stroke volume, and left ventricular end-diastolic pressure (LVEDP) and increased the systolic aortic pressure, cardiac output, and pulmonary capillary wedge pressure (PCWP). DDD mode pacing also reduced the time constant of isovolumetric relaxation () when the pacing rate was increased to 80/min. However, at a rate of 90/min, pacing with both VVI and DDD mode caused an increase in LVEDP, PCWP and time. The patient experienced chest pain at this rate. VVI-mode pacing decreased cardiac output and increased PCWP more than did DDD mode pacing.

Patient 2

A 72-year-old woman on beta-blockers and calcium antagonists was admitted to hospital and diagnosed as having syncope. Vital signs included a pulse of 60/min and a blood pressure of 120/60 mmHg. A grade 2/6 systolic murmur was noted at the left sternal border. An echocardiogram showed asymmetric septal hypertrophy, narrowing of the left ventricular outflow tract, SAM of the mitral valve, and mitral regurgitation. An LVG showed an abnormal configuration and grade 2/4 mitral regurgitation. The CAG was normal. A cardiac catheterization study demonstrated that the peak subaortic pressure gradient in the left ventricular outflow tract was 50 mmHg. During DDD mode pacing, the pressure gradient decreased and the systolic aortic pressure increased, both when the pacing rate was increased from 60 to 70/min with 140 msec AV interval, and from 80 to 90/min with 100 msec AV interval (Table 1). A DDD permanent pacemaker was implanted.

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Fig 1. Hemodynamic changes in patient 1 before and during the pacing study. SR, normal sinus rhythm at baseline study; bpm, beats per minute; peak PG, peak-to-peak pressure gradient in the left ventricular outflow tract; sys-BP, systolic aortic blood pressure; CO, cardiac output; SV, stroke volume; LVEDP, left ventricular end-diastolic pressure; m-PCWP, mean pulmonary capillary wedge pressure; τ , time constant of isovolumetric relaxation; AV, atrioventricular interval.

Table 1 Hemodynamic Findings in Patient 2 Before and During the Pacing Study

Mode	Sinus rhythm	DDD pacing				
Rate	51	60	70	80	90	
AV interval (msec)		140	140	100	100	
Ao (mmHg)	80/49	90/44	100/46	104/52	106/56	
Peak PG (mmHg)	50	40	24	16	14	
Mean PCWP (mmHg)	14	12	10	10	11	

AV, atrio-ventricular; Ao, aortic pressure; PG, pressure gradient; PCWP, pulmonary capillary wedge pressure.

The lower rate was programmed to 80/min and the upper rate to 120/min. The programmed AV delay was decreased from 100 msec to 80 msec, which permitted ventricular pre-excitation during daily activity on propranolol (60 mg/day) and verapamil (120 mg/day). DDD pacing led to a reduction in the inward systolic movement of the interventricular septum with significant reduction of systolic anterior motion and subaortic pressure gradient on echocardiographic examination. The patient has been followed up in our outpatient department for 3 years with no episodes of syncope.

Patient 3

A 70-year-old man was admitted to the hospital with frequent episodes of syncope and convulsions. Vital signs included a pulse of 78/min, a blood pressure of 120/72 mmHg, and a grade 2/6 systolic murmur at the left sternal border. An echocardiogram showed asymmetric septal hypertrophy, narrowing of the left ventricular outflow tract, SAM of the mitral valve, and mitral regurgitation. A LVG showed grade 2/4 mitral regurgitation. The CAG was normal. A cardiac catheterization study demonstrated a peak subaortic pressure gradient of 60 mmHg. When the patient experienced spontaneous syncope and convulsions during the catheterization study, increased subaortic pressure gradients of more than 110 mmHg, decreased systolic aortic pressures, and ST-segment depressions in all leads on the 12-lead electrocardiogram were detected. An echocardiogram, during spontaneous syncope and convulsions, detected increased SAM followed by closure of the left ventricu-

lar outflow tract and increased mitral regurgitation. Beta-blocker therapy with propranolol (240 mg/day), subsequent atrial synchronized right ventricular pacing, and AV sequential pacing at an AV interval of 100 msec (which was the optimum AV interval defined by an echocardiographic study), and various pacing rates (50, 70, and 80 /min) failed to relieve the syncope. On echocardiographic study, the subaortic pressure gradient was 31 mmHg at a pacing rate of 50/min and 21 mmHg at 70/min. However, AV sequential pacing at a pacing rate of 90/min relieved the syncope and the patient experienced no episodes on a reduced dose of propranolol (40 mg/day) for 15 days until undergoing a mitral valve replacement. The echocardiographic subaortic pressure gradient was 15 mmHg at a pacing rate of 90/min.

Discussion

The symptomatic and hemodynamic benefits of dual-chamber pacing therapy in patients with HOCM have been reported previously¹⁻⁴ and the influence of the AV interval^{1,4} and the right ventricular pacing site^{5,6} has been emphasized. Reduction in the subaortic pressure gradient during right ventricular apical pacing may be due to an altered pattern and an abolition of the early systolic intra-ventricular obstruction! However, studies focusing on the effects of pacing rate on the hemodynamic state are rare.^{2,7,8} A previous study using pacing rates of 100, 120, and 150 /min during dual-chamber pacing² and right atrial pacing⁷ reported deterioration of hemodynamic indexes. The study using dual-chamber pacing reported that pacing at a rapid heart rate resulted in modest reductions in pressure gradient with marked increases in filling pressures and reductions in cardiac output? However, in these previous studies the hemodynamic changes using a pacing rate lower than 100 /min were not examined. The study of pacing rate from 80 to 110/min reported a decrease in τ during right atrial pacing in hypertrophic cardiomyopathy.⁸ In our reported patients, increasing the pacing rate (but not exceeding 100 /min) improved circulatory conditions. The acute hemodynamic change observed during this pacing study and the patients described in this and in previous studies provide

some invaluable insights. First, pacing therapy performed at a slightly higher rate (70–90/min) may decrease subaortic pressure gradient without noticeable deterioration in cardiac function. Second, pacing at a rate higher than 100/min may cause a deterioration in cardiac function, probably because of a disturbance in ventricular filling and an elevation in both diastolic pressure in the ventricle and atrial pressure. Several proposed mechanisms of these effects of a slightly higher pacing rate are: (1) decreasing the stroke volume may reduce the Venturi effect followed by a decrease in the degree of outflow obstruction, and this reduction in contraction load may account for the lack of deterioration of cardiac output and diastolic function; (2) a higher pacing rate increases coronary flow,⁹ which may aid diastolic function. Thus, our experience suggests that patients who become refractory to dual-chamber pacing therapy at an optimum AV interval may experience beneficial effects at a slightly higher rate. However, it is not clear if these effects associated with pacing rate are found in all patients with HOCM or if they have long-term benefits.

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