Case report

Biventricular pacing and atrioventricular junction ablation as treatment of low output syndrome due to refractory congestive heart failure and chronic atrial fibrillation

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Key words:
Heart failure; Pacing.

A 71-year-old male patient with end-stage heart failure, atrial fibrillation, congestive and low output symptoms, underwent biventricular pacing and atrioventricular junction ablation while anuric and hypotensive. Following atrioventricular junction ablation blood pressure increased by 20 mmHg during biventricular but not during right ventricular apical pacing. A rapid clinical improvement was observed and the patient was discharged from the hospital in NYHA functional class III.

(Ital Heart J 2000; 1 (12): 844-847)

Received July 3, 2000; revision received September 29, 2000; accepted October 6, 2000.

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Introduction

Biventricular and single-site left ventricular (LV) free wall pacing can acutely improve cardiac hemodynamics and contractile function in congestive heart failure (CHF) patients with intraventricular conduction delay, during sinus rhythm[1-3] as well as during atrial fibrillation[1,4]. The preliminary results of several ongoing long-term studies using biventricular or LV pacing in such patients all show an improvement in the patients' clinical conditions as evaluated by NYHA functional class and the Minnesota Living with Heart Failure score, oxygen consumption during cardiopulmonary test and the distance covered during the 6-min walk test[5-7]. The majority of patients enrolled in these studies were in sinus rhythm and in a relatively stable NYHA functional class III or IV at study entry, with only a few exceptions of class IV patients with chronic atrial fibrillation requiring intravenous inotropic support[7].

We describe a case of refractory CHF with marked intraventricular conduction delay, chronic atrial fibrillation, congestive and low output symptoms despite intravenous inotropes in whom biventricular pacing and atrioventricular junction ablation were performed as rescue treatment when the patient was anuric and hypotensive. Cardiac performance immediately improved allowing the patient to recover from an apparently irreversible low output syndrome.

Case report

A 71-year-old man with a relevant medical history (coronary artery disease with prior myocardial infarction, LV aneurysm, moderate tricuspid, mitral and aortic regurgitation, severe LV dysfunction, chronic atrial fibrillation, episodes of both acute pulmonary edema and CHF since 1997) was admitted to our hospital in August 1999, due to a new episode of cardiac decompensation. He presented with dyspnea at rest, orthopnea, fatigue and weakness, anxiety and anorexia. The physical examination revealed moist pulmonary rales over both lung fields, jugular vein distension, edema in both legs, congestive hepatomegaly, gallop sound, and an apical grade 2 systolic murmur. Blood pressure was 100/80 mmHg and he had an irregular rhythm at about 150/min. Rest ECG showed atrial fibrillation with rapid ventricular response, marked intraventricular conduction delay (QRS duration of 229 ms) and chronic ST-segment elevation in the precordial leads (Fig. 1). A chest X-ray revealed cardiac enlargement, vascular redis-

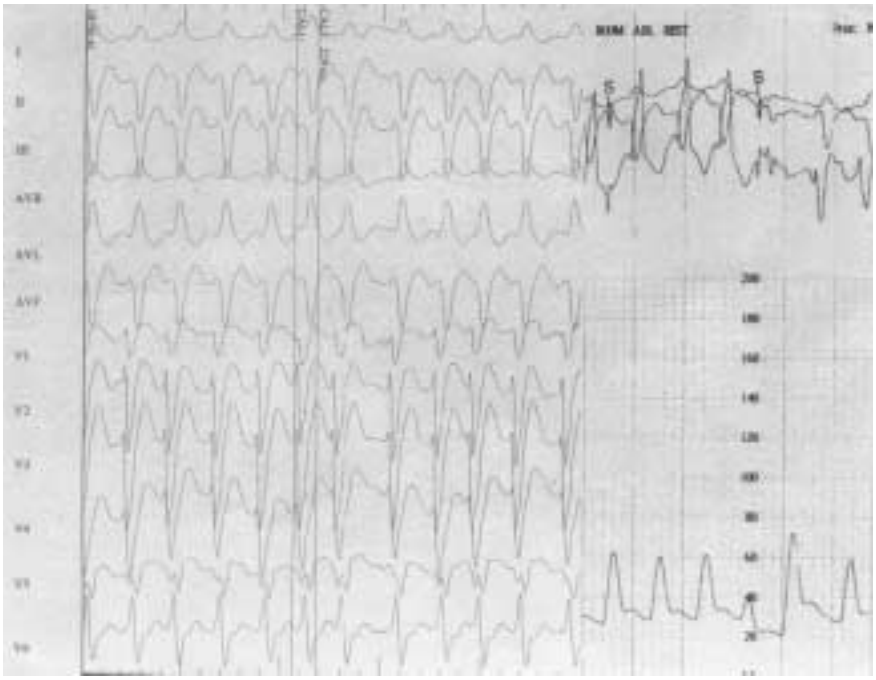

Figure 1. *Resting ECG showing atrial fibrillation with rapid ventricular response of 140 beats and marked intraventricular conduction delay (QRS duration of 229 ms).*

tribution, bilateral alveolar edema, and right pleural effusion (Fig. 2).

Two-dimensional Doppler echo examination showed a dilated, poorly contracting left ventricle (LV end-diastolic diameter 84 mm; LV ejection fraction 17%), a fibrotic and thin interventricular septum, a large apical aneurysm, moderate mitral, aortic and tricuspid regurgitation, and severe pulmonary hypertension (maximum regurgitant tricuspid flow velocity 3.8 m/s). Despite optimal medical therapy with digoxin, angiotensin-converting enzyme inhibitors, furosemide, canrenone, carvedilol, amiodarone and intravenous nitroglycerin heart failure became refractory and hemodynamic compensation required the association of intravenous dopamine and dobutamine. Several attempts of weaning the patient from inotropic support failed in a time-period of 1 month. In the meantime dobutamine was replaced by intravenous milrinone due to excessively high ventricular rate and hypotension. Heart transplant was excluded due to the advanced age of the patient. On the evening of October 3rd 1999, the clinical picture worsened. Orthopnea and mental confusion developed and the patient became pale, diaphoretic and anuric. Progressive hypotension developed during that night requiring nitroglycerin and milrinone discontinuation. In the morning of October 4th, the mean ventricular rate was 150/min, systolic blood pressure was around 60 mmHg, oxygen saturation was 86%. An emergency atrioventricular junction ablation and biventricular pacing were attempted based on the hypothesis that cardiac performance would have been improved by both ventricular rate slowing and regularization[8,9] and by LV resynchronization[1-4]. A Medtronic Attain 2187 lead model (Medtronic Inc., Minneapolis, MN, USA) was positioned into a large lateral marginal vein through the coronary sinus. The acute ventricular stimulation threshold was 2 V. Pacing at more anterior sites and into the anterior interventricular vein resulted in no ventricular capture. It was impossible to insert the lead into any posterior vein. A Vitatron Slimtime S lead model (Vitatron Medical B.V., Dieren, The Netherlands) was positioned at the apex of the right ventricle. The two leads were connected to a Medtronic InSync model 8040 pulse generator to simultaneously pace both ventricles. Ablation of the atrioventricular junction was then performed with two applications of radiofrequency current. Following atrioventricular node ablation, systolic blood pressure instantaneously increased by 20 mmHg, from the baseline value of 60 to about 80 mmHg, during biventricular but not during right ventricular pacing alone (Fig. 3). This

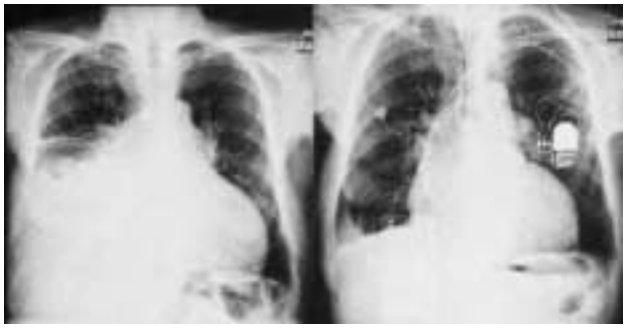

Figure 2. On the left: *chest X-ray before implant shows cardiac enlargement, vascular redistribution, bilateral alveolar edema and right pleural effusion.* On the right: *at pre-discharge chest X-ray, a marked reduction in both cardiac size and pulmonary vascular congestion is present compared with baseline.*

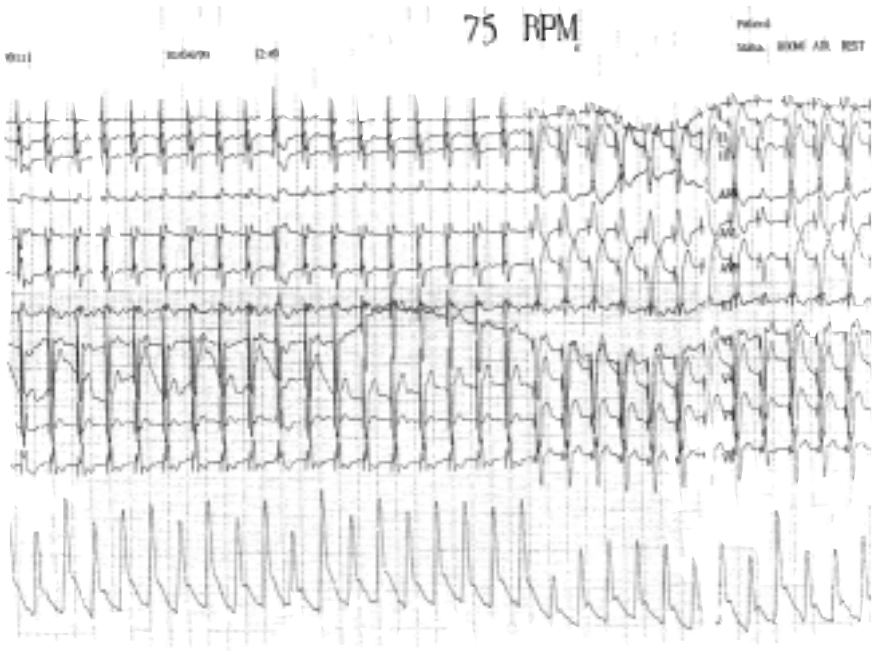

Figure 3. *After atrioventricular node ablation systolic blood pressure instantaneously increased by 20 mmHg, from the baseline value of 60 to about 80 mmHg during biventricular* (on the left, continuous tracing) *but not during right ventricular pacing alone* (on the right, continuous tracing).

was associated with a narrowing of the paced QRS duration of 44 ms (from the baseline value of 229 to 185 ms). The interventricular mechanical delay, echocardiographically derived from the delay measured between the onset of pulmonic and aortic flow, shortened from 62 ms (baseline value) to 30 ms during biventricular pacing, at the patient's discharge. The patient's clinical conditions improved very quickly and he was discharged from hospital 11 days after the procedure in NYHA functional class III with the same initially ineffective therapy. Pre-discharge chest X-ray showed a marked reduction in both cardiac size and pulmonary vascular congestion compared with baseline (Fig. 1). Patient functional class remained stable during 4-month follow-up.

Discussion

Multisite ventricular pacing has recently been proposed as an additional treatment for patients with severe CHF and intraventricular conduction delay, based on the results of several studies showing a significant acute improvement of cardiac hemodynamics and contractile function during biventricular pacing compared with baseline. Blanc et al.[1], reported a significant increase in systolic blood pressure and a decrease in pulmonary capillary wedge pressure and V-wave amplitude, compared with baseline, during either biventricular or single site LV but not during right ventricular pacing in 27 patients with severe CHF and sinus rhythm. Leclercq et al.[2] compared the acute hemodynamic effects of biventricular DDD pacing compared with baseline AAI and optimal right ventricular DDD pacing in 18 patients with severe CHF, major intraventricular conduction block and stable sinus rhythm. A highly significant increase in cardiac output (35%), an 18.5% decrease in mean pulmonary capillary wedge pressure, and a 21% reduction in V-wave amplitude were observed during biventricular DDD compared with AAI pacing mode. A much smaller increase in cardiac output was observed during optimal right ventricular DDD pacing. Kass et al.[3] compared hemodynamic data under normal sinus rhythm and during ventricular pacing (VDD) at varying sites and atrioventricular delays in 18 heart failure patients with intraventricular conduction delay. In this study LV free-wall and biventricular VDD pacing significantly raised dP/dt max, peak systolic pressure and arterial pulse pressure, whereas right ventricular VDD pacing did not. In contrast to systole, pacing had little effect on diastolic function. To evaluate if the acute hemodynamic benefit reported with LV pacing also applies to heart failure patients with chronic atrial fibrillation, Etienne et al.[4] compared LV and biventricular pacing with baseline sinus rhythm or right ventricular apical pacing in 28 patients, 17 in sinus rhythm and 11 with atrial fibrillation. Biventricular and LV pacing yielded similar results in both groups of patients, with or without atrial fibrillation. All the patients enrolled in these acute studies were in relatively stable clinical conditions. No data are available about the acute effects of LV based pacing in CHF patients and unstable hemodynamic conditions, as was the case of our patient. In our case atrial fibrillation with uncontrolled ventricular rate might also have contributed to the exacerbation of low output symptoms. Indeed cardiac performance has been reported to be improved by both ventricular rate slowing and regolarization in CHF patients with atrial fibrillation[8,9]. In our

patient, systolic blood pressure increased by 20 mmHg following atrioventricular node ablation during biventricular pacing but not during right ventricular pacing. This suggests that it was the ventricular resynchronization by biventricular pacing, rather than the ventricular rate control and regolarization by atrioventricular node ablation, to exert the most positive effects on LV performance in this patient. This is in agreement with a previous report[3] of a strong relationship between the extent of intraventricular conduction delay, which was 229 ms in our patient, and the favorable effects of LV pacing.

This is the first description of reversion of an apparently irreversible low output syndrome in a patient with refractory CHF and chronic atrial fibrillation by biventricular pacing combined with atrioventricular node ablation.

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