The successful treatment of end stage of heart failure associated with complete right bundle branch block with biventricular pacemaker placement

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Abstract

Biventricular pacing has been used to treat patients with symptomatic heart failure, systolic left ventricular dysfunction and intraventricular conduction delays. This modality is usually reserved for the treatment of patients with a left bundle branch block pattern on electrocardiogram. We report the successful use of biventricular pacing in a patient with heart failure and a right bundle branch block conduction delay.

Introduction

Biventricular pacing (BiVP) or the simultaneous pacing of the left and right ventricle, has been used in the treatment of patients with severe left ventricular systolic dysfunction, intraventricular conduction delays and progressive heart failure.[1] This treatment may result in improved cardiac performance and functional status.[1] It is postulated that BiVP results in resynchronization of left ventricular contraction to allow coordinated contraction of the left ventricular septum and lateral wall. Left bundle branch block (LBBB) often causes asynergistic contraction between the septum and the left ventricular lateral wall.[2,3] Accordingly, BiVP has been predominantly used in patients with LBBB.[2,3] Although patients with right bundle branch block (RBBB) may also demonstrate intraventricular dysynchrony, the benefit of BiVP in patients with heart failure and RBBB has been less clearly established.[4] We report a case of a patient with nonischemic cardiomyopathy and RBBB in whom BiVP resulted in improved functional status and cardiac performance.

Case report

A 38-year old man presented to our institution with progressive heart failure and renal insufficiency. He had been diagnosed to have nonischemic cardiomyopathy at the age of 34. At that time, echocardiographic examination demonstrated dilated cardiomyopathy with left ventricular ejection fraction of 25%. Coronary angiography revealed the absence of significant coronary obstructive disease. The patient was treated with furosemide, valsartan, carvedilol and spironolactone. Three months prior to admission, the patient was diagnosed as having decompensated congestive heart failure. Outpatient diuretic therapy was attempted. The patient developed progressive worsening of dyspnea and was admitted to our institution.

On admission, the patient had overt congestive heart failure and a low output cardiac state. The blood pressure was 110/70 mm Hg and pulse was 102 beats per minute. On initial physical examination, the patient was documented as having elevated jugular venous filling pressure, pulmonary rales and a summation gallop. Laboratory studies showed normal blood count and electrolytes. Blood urea nitrogen and serum creatinine levels were elevated at 57 mg/dl and 2.8 mg/dl, respectively. A chest radiograph demonstrated congestive heart failure. An electrocardiogram (ECG) revealed the presence of sinus tachycardia, left axis deviation and RBBB with QRS duration of 190msec (Fig.1).

Right heart catheterization was performed. Hemodynamic pressure measurement showed a blood pressure 97/50 mmHg, a heart rate 109 beats/min, a right atrial pressure 18 mmHg, a pulmonary artery pressure 56/32 mmHg, a pulmonary artery wedge pressure 29 mmHg, and a cardiac index 2.0 L/min/m2 while on milrinone.

He was treated with intravenous diuretics, nesiritide and inotropic support with milrinone. However, he continued to manifest congestive heart failure and low cardiac output. Right heart pressure measurement documented right atrial pressure 24 mmHg, pulmonary artery pressure 61/40 mmHg, pulmonary artery wedge pressure 24 mmHg, and cardiac index 2.6 L/min/m2.

An echocardiography showed left ventricular enlargement, moderate to severe mitral insufficiency and global hypokinesis of the left ventricle with left ventricular ejection fraction of 10%. Septal contraction occurred 420 msec after the onset of the surface QRS complex and was 100 msec after contraction of the left ventricular lateral wall (Fig 2), demonstrating left ventricular dysynchroniza-

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tion. Therefore, resynchronization treatment was considered. A biventricular pacemaker was placed. Ventricular leads were placed in the right ventricular apex and the posterolateral branch of the coronary sinus.

After placement of the biventricular pacing system, the patient had marked clinical improvement with a change in functional status from NYHA IV to II. Serum chemistries revealed a decrease in creatinine from 2.8 to 1.3 mg/dl. The echocardiography performed after pacemaker placement documented synchronous contraction of the septum and lateral wall and increase in left ventricular ejection fraction from 10 to 20%. It also showed significant decrease of mitral regurgitation.

He was discharged and has not required hospitalization for heart failure in nine months of follow-up. His functional status remains NYHA II.

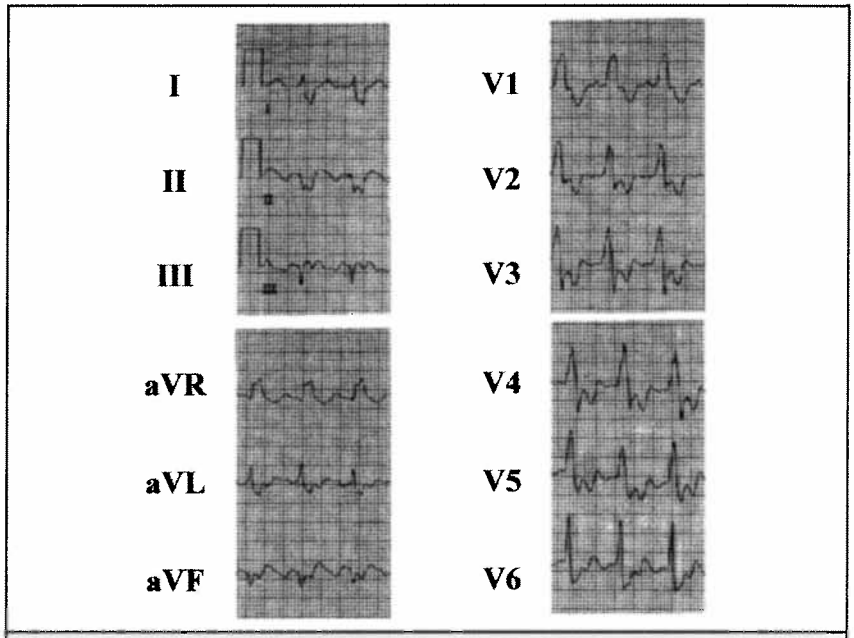

Figure 1.— A 12-lead surface electrocardiogram (25mm/s) on admission.

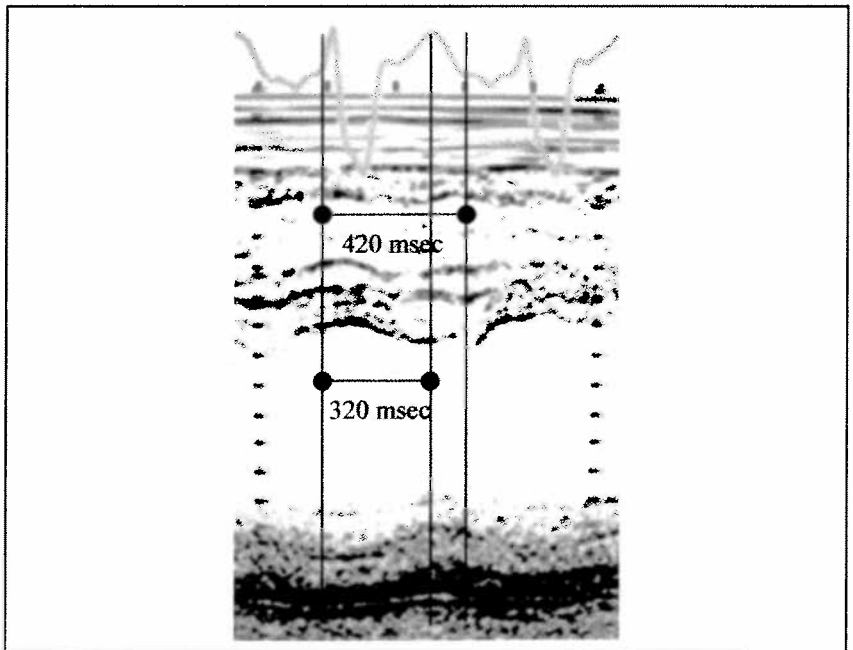

Figure 2.— M-mode echocardiographic view of the interventricular septum and posterolateral wall before biventricular pacemaker placement.

Discussion

Nearly 5 million persons living in the USA have heart failure, with 550,000 new patients diagnosed annually.[5] Despite substantial advances in drug therapy, heart failure was associated with 287,000 deaths and nearly 1 million hospital admissions in the USA in 1999.[6] A common finding in advanced heart failure is abnormal electrical activation of the ventricles or electrical ventricular dysynchrony, which is manifested in ECG as prolongation of QRS duration, often in the pattern of LBBB. This has been reported to be associated with diminished cardiac function[2,7] and increased mortality.[3] Recently, devices that make use of atrial-synchronized BiVP to coordinate right and left ventricular contraction have been developed. This device enhances cardiac function, reduces myocardial oxygen consumption, and improves exercise capacity, functional status, and quality of life. A meta-analysis of randomized controlled trial showed reduced mortality from heart failure.[8]

In the Multicenter InSync Randomized Clinical Evaluation (MIRACLE) study, 440 patients with marked functional limitations with NYHA functional class III or IV associated with an ejection fraction of 35% or less and a QRS interval of 130 msec or more were randomly assigned to BiVP or to a control group.[9] Patients treated with BiVP were noted to have improvement in functional class, increased exercise capacity and decreased rates of hospitalization. While improvement was noted in 66% of patients in this trial, a minority of patients did not improve. A hypothesis for failure to improve with BiVP was inclusion of patients with RBBB into the study population. This study included patients with both right and left bundle branch block as well as patients with nonspecific intraventricular conduction delay patterns.

We contend that improvement noted with BiVP in the treatment of patients with advanced heart failure and conduction abnormalities is not dependent upon the type of conduction abnormality but rather related to the extent of septal and left ventricular lateral wall asynergy. Despite the electrocardiographic finding in our patient of RBBB and therefore presumed isolated right ventricular asynergy, our patient demonstrated left ventricular dysynergy on echocardiographic studies. BiVP in our patient resulted in improved left ventricular contractile coordination and associated clinical status. We recommend that baseline echocardiographic testing be performed in patients with cardiomyopathies and conduction delays. BiVP should be considered in patients with conduction delays and associated left ventricular dysynergy.

See ***Successful Treatment****, p. 165*

Successful Treatment from p. 156

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