Acute Improvement of Left Ventricular Functions by Biventricular Pacing in a Patient with Dilated Cardiomyopathy and Permanent Atrial Fibrillation - A Case Report

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Summary

Simultaneous right and left ventricular pacing was performed in a 76-year old man with severe congestive heart failure (CHF). Differential diagnosis revealed dilated cardiomyopathy, a left bundle branch block, and permanent atrial fibrillation. For left ventricular pacing, the transvenous lead Corox LV (BIOTRONIK) was introduced through the coronary sinus into the lateral coronary vein and connected to the atrial channel of a dual-chamber pacemaker. The patient clinically improved from borderline NYHA class III/IV to NYHA class II with biventricular pacing, but not with standard right ventricular VVI pacing. This case report gives evidence that CHF patients with permanent atrial fibrillation might benefit from biventricular pacing.

Key Words

Biventricular pacing, congestive heart failure (CHF)

Introduction

Despite advances made in drug treatment, the prognosis and quality of life for patients with severe chronic congestive heart failure (CHF) is very poor. Promising results for the treatment of CHF have recently been reported with simultaneous right and left ventricular (LV) pacing combined with conventional drug treatment. Biventricular pacing is capable of improving the synchronization of the ventricular function in patients with dilated caradiomyopathy and left bundle branch block (LBBB). In these patients, biventricular pacing improves the cardiac output, lowers the functional NYHA classification, and improves the quality of live. Especially those patients with moderate to severe CHF, severe LV systolic dysfunction, and predominantly LV conduction disturbances will benefit from this therapy [1-5].

Pacing of the left ventricle can be achieved either with epicardial leads using thoracotomy/thoracoscopy or via the coronary venous system using endovenous leads. Due to the technical limitations of leads not designed for a transvenous approach, the latter was not always feasible. The development of new, more maneuverable pacemaker leads specially designed for transvenous LV pacing has led to a marked improvement. Left ventricular pacing is becoming a technology with a status that is no longer experimental.

In this article, we report the outcome of biventricular pacing in a patient with severe CHF and permanent atrial fibrillation.

Case-Report

A 76-year old, male CHF patient with permanent atrial fibrillation was hospitalized for the treatment of CHF. Although the patient was under permanent CHF drug treatment, he showed additional signs of severe heart failure. He was symptomatic with exertion dyspnea and cardiac arrhythmia at rest (60 bpm). The cardiologic status of the patient resembled borderline NYHA class III to IV.

Radiography of the chest showed an enlarged heart shadow. The appearance of Kerley lines in the X-rays indicated a bilateral edema of the basal pulmonary lobes (Figure 1). Twelve-lead ECG recordings showed

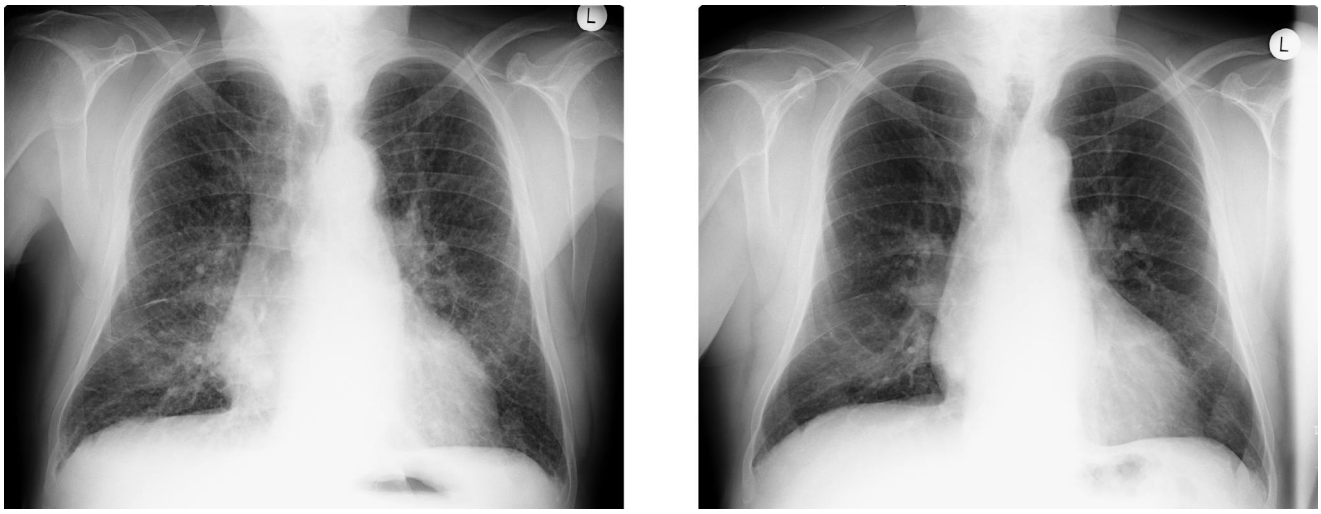

Figure 1. Anterior-posterior chest X-rays taken at admission (left) and one day prior to implantation (right) showing Kerley lines and an enlarged heart shadow.

atrial fibrillation, LBBB, and a prolonged QRS duration of about 160 ms. The 24 h ECG revealed episodes of bradyarrhythmia with a heart rate of about 31 bpm. While the right heart appeared normal, echocardiography showed a dilated left ventricle (end diastolic diameter = 71 mm), a severe systolic dysfunction with a LV ejection fraction of about 25%, a mild mitral insufficiency (Grade I), a dilated left atrium (38 mm), and a left atrial thrombus. Recompensation of the CHF was achieved with diuretics, ACE-inhibitors, and beta-blockers. In view of the patient's critical hemodynamic condition in combination with bradyarrhythmia and LBBB, it was decided to pace the left and right ventricle to achieve biventricular pacing.

The transvenous lead system Corox LV (BIOTRONIK) was chosen for LV pacing. The Corox LV is a silicon-coated unipolar electrode offering the use of a mandrin. A ring just behind a soft distal tip serves as the stimulating electrode. On a length of about 1 cm proximal to the ring, the silicon surface is screw-like to ensure proper fixation. The LV lead was introduced through the subclavian vein and placed via the coro-

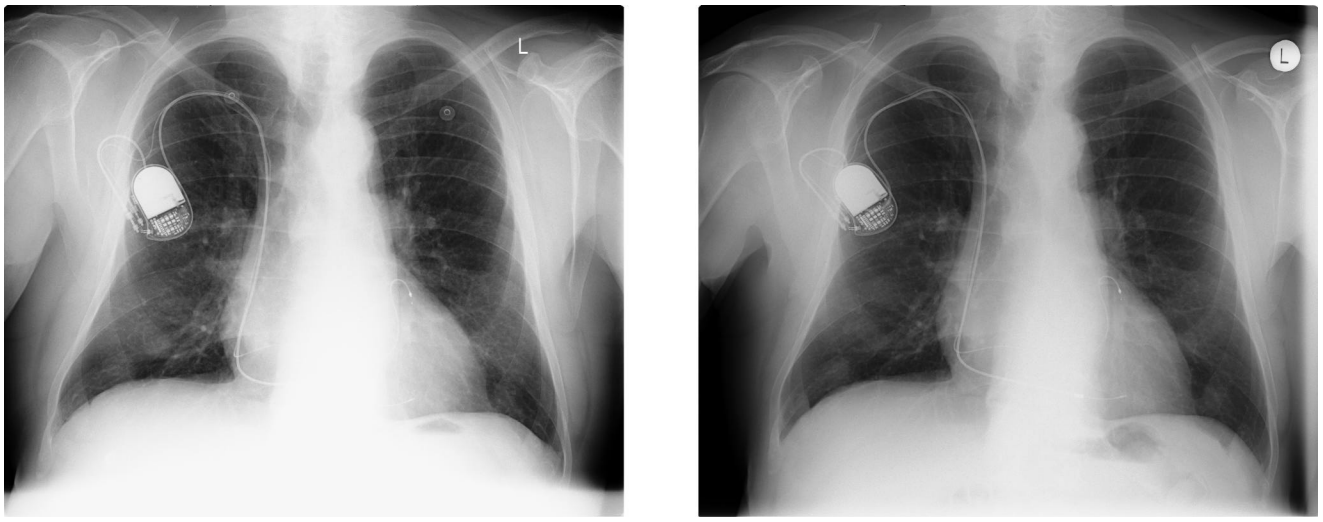

Figure 2. Anterior-posterior chest X-rays taken at implantation (left) and one week after implantation (right). Note the reduction of Kerley lines one week after implantation.

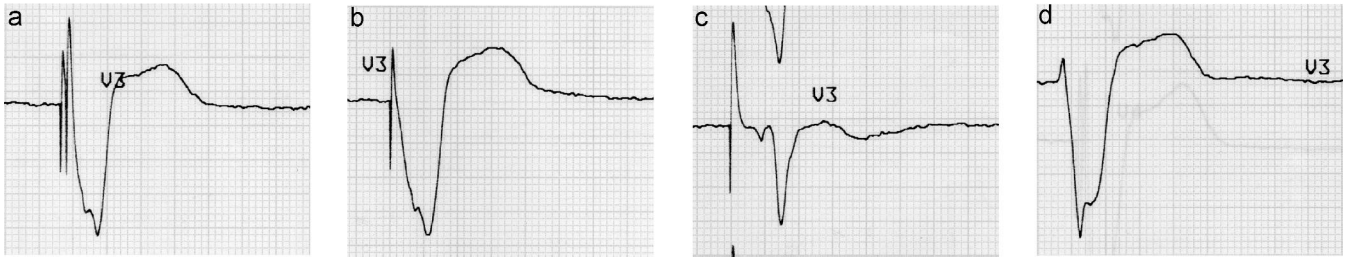

Figure 3. QRS complex in lead III ECG recordings (50 mm/s) after a) biventricular pacing, b) right ventricular pacing, c) left ventricular pacing, and d) at spontaneous rhythm.

nary sinus into the lateral coronary vein at a high basomedial level (Figure 2). At implantation, the pacing threshold was 2.6 V at a pulse width of 0.4 ms. The R-wave amplitude was above 20 mV, and the impedance of the electrode was 700 Ω. For right ventricular pacing, a second lead (Polyrox, BIOTRONIK) was placed via the subclavian vein into the apex of the right ventricle.

For biventricular pacing, the LV lead was connected to the atrial channel of the dual-chamber pacemaker Actros DR (BIOTRONIK), and the right ventricular lead was connected to the ventricular channel. To ensure simultaneous pacing of both ventricles, the pacemaker was set to the DDD mode and programmed to the shortest AV delay possible (15 ms).

We observed a slight decrease in QRS duration with LV and biventricular pacing (Figure 3). In spontaneous rhythm and with right ventricular pacing, the QRS duration was 160 ms, and with LV and biventricular pacing, the QRS duration was about 140 ms.

One day after implantation, we noticed a rise in the LV pacing threshold and a decrease in the LV sensing amplitude. Successful permanent LV pacing was obtained at 7 V, and the R-wave amplitude had decreased to 2.9 mV. Radiography showed no dislocation of the LV lead (Figure 2). We therefore interpreted the increase in threshold as the result of a physiologic process at the electrode. Similar effects are known from conventional endocardial leads. These electrodes show a transient increase in pacing threshold after implantation. Despite the increase in LV pacing threshold, biventricular pacing was successfully continued for the next eight days. During this period, the patient's clinical status improved impressively. X-rays taken one week after the implantation showed no signs of edema. Additional echocardiography showed a slight increase in the ejection fraction from 25% at admission to 30% at discharge, and the LV enddiastolic diameter had decreased from 71 to 63 mm. The NYHA classification of the patient had improved from borderline NYHA class III-IV to NHYA grade II.

Eight days after implantation, the pacing threshold remained high. In view of the high battery drain due to the high LV pacing threshold, we decided to switch from biventricular to right ventricular pacing. Therefore we reprogrammed the pacemaker from DDD to VVI, the latter ensuring right ventricular pacing. Two weeks after discharge, the patient returned to our ambulatory care center with signs and symptoms of severe CHF. Despite continous drug therapy, the patient had become symptomatic at rest and fulfilled the criteria of the NYHA classification IV. We promptly reestablished biventricular pacing by reprogramming the pacemaker from VVI to DDD, and the clinical status of the patient improved within several hours. Since the LV pacing threshold had decreased to 2.8 V, we decided to continue biventricular pacing. Since then the patient's clinical status has been stable and is classified as NYHA class II.

Discussion

This case report underlines the importance and value of biventricular pacing in patients with severe heart failure. Most convincing was the unexpected rapid improvement of the patient's clinical status when the patient returned in a decompensated state. After switching from right ventricular to biventricular pacing, the patient became unsymptomatic within hours. From this observation, it is evident that resynchronization of the ventricular events is of beneficial value for the hemodynamic condition of CHF patients with permanent atrial fibrillation, dilated left ventricle, and LBBB.

In the past, technical limitations in placing a permanent LV pacing lead prevented the widespread use of the biventricular pacing therapy. Recent advantages in transvenous lead design solved many of the problems encountered with epicardial leads. The approach via the coronary sinus for LV pacing offers the advantage of a minimal invasive procedure.

Successful LV pacing was achieved with the LV lead Corox LV (BIOTRONIK) placed at a high basomedial level of the lateral cardiac vein. At implantation, the pacing threshold and R-wave amplitude were within the expected range of 2 to 3 V [3]. Compared to conventional right ventricular pacing leads, transvenous lead systems tend to have somewhat higher pacing thresholds. Endovenous leads use a ring instead of a tip to stimulate the underlying tissue. Most of the ring surface is exposed to the venous lumen and gives rise to a considerable amount of current spread into the surrounding blood.

The increase in LV pacing threshold noted one day after implantation was probably due to a physiologic process of unclear origin. Although the increase pointed to a lead dislocation, post implantation X-rays confirmed the proper location of the LV lead. In right ventricular pacing, a transient rise in the pacing threshold is a well-known phenomenon due to inflammatory processes at the site of the electrode. In transvenous lead pacing, similar processes might occur. Three weeks post-implantation, the pacing threshold had decreased to a value close to the implantation level, and continuous LV pacing could be achieved with a moderately high amplitude.

Several studies propose biventricular pacing for additional treatment of severe heart failure to improve the severely depressed LV systolic function. Our patient fulfilled two criteria for biventricular pacing. According to the literature, biventricular pacing improves the LV systolic function in patients with dilated cardiomyopathy and LBBB [1-5].

In the presence of LBBB, the usual pattern of contraction is greatly disturbed. Even in apparently normal hearts, LBBB causes interventricular asynchrony and cardiac abnormalities [6][7]. In addition, the usual sequence of ventricular events is reversed with the right ventricular systole and diastole markedly preceding the LV events. In patients with LBBB, the mechanical asynchrony of the left and right ventricle leads to a reduction in the ejection fraction [6]. In CHF patients with dilated cardiomyopathy and LBBB, biventricular pacing is thought to resynchronize the ventricular activation sequence and to ameliorate the consequences of LBBB on the ejection fraction. Furthermore, biventricular pacing not only increases the patient's ejection fraction but improves the cardiac output by optimizing the diastolic filling pattern (for discussion see [8]). One potential mechanism is the improvement in diastolic relaxation due to the improved relaxation synchrony brought about by pacing. In our patient, we chose the lowest interventricular delay possible. In this patient, the programmed delay of 15 ms had no negative effects on the outcome of biventricular pacing therapy.

It has been reported recently that an appropriate AV delay optimizes the diastolic filling time and improves the hemodynamics of biventricularly - paced CHF patients [8]. Since our patient suffered from permanent atrial fibrillation, AV-synchronous pacing could not be achieved. Our therapeutic approach shows that resynchronization of the ventricular action might be sufficient to improve the hemodynamic state and the quality of live of CHF patients with permanent atrial fibrillation, dilated left ventricle, and LBBB significantly.

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