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CASE REPORT

Clinical impact of increasing left ventricular pacing output in cardiac resynchronization therapy – the new optimization strategy

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Key Clinical Message

We report a patient who did not improve after standard optimization of atrioventricular and intraventricular pacing timing, but showed response to cardiac resynchronization therapy (CRT) after increasing left ventricular (LV) pacing output. Increasing LV pacing output is one of the useful optimization methods for CRT nonresponder.

Keywords

CRT, optimization, pacing output.

Introduction

Cardiac resynchronization therapy (CRT) is an established therapy for reducing the risk of mortality as well as improving symptoms in patients with severe heart failure. Based on several trials, response rates for CRT are in the range of 70% [1]. Inadequate atrioventricular (AV) and intraventricular (VV) intervals may be the potential reason for nonresponse to CRT [2]. Furthermore, a discrete region with conduction block as scar tissue at the region of the LV lead inhibits effective pacing of myocardium [3]. Optimization of pacing timing is usually performed utilizing echocardiography, and optimal AV and VV delay is determined. We report a patient with nonischemic cardiomyopathy, whose mechanical dyssynchrony did not improve after usual optimization of AV and VV pacing timing. However, he showed response to CRT after increasing left ventricular (LV) pacing output.

Case Report

A 66-year-old woman with nonischemic cardiomyopathy underwent hemodialysis due to hypertension and diabetes mellitus. She had New York Heart Association functional class III symptoms of heart failure. Her electrocardiogram (ECG) showed a complete left bundle branch block (Fig. 1A). The echocardiogram revealed concentric hypertrophy, LV dilatation, and LV dysfunction (ejection fraction using the biplane modified Simpson's rule: 26%). The coronary angiogram revealed no significant stenosis. Her blood pressure fell remarkably during hemodialysis. Since mechanical dyssynchrony was detected by tissue Doppler imaging and was recognized as a cause of LV dysfunction, we implanted a pacemaker with a function of biventricular pacing (InSync III, Medtronic, Minneapolis, MN) and initiated CRT. After pacing, the ECG showed a decrease in QRS width from 180 msec to 140 msec (Fig. 1B). However, a blood pressure (BP) fall during hemodialysis was

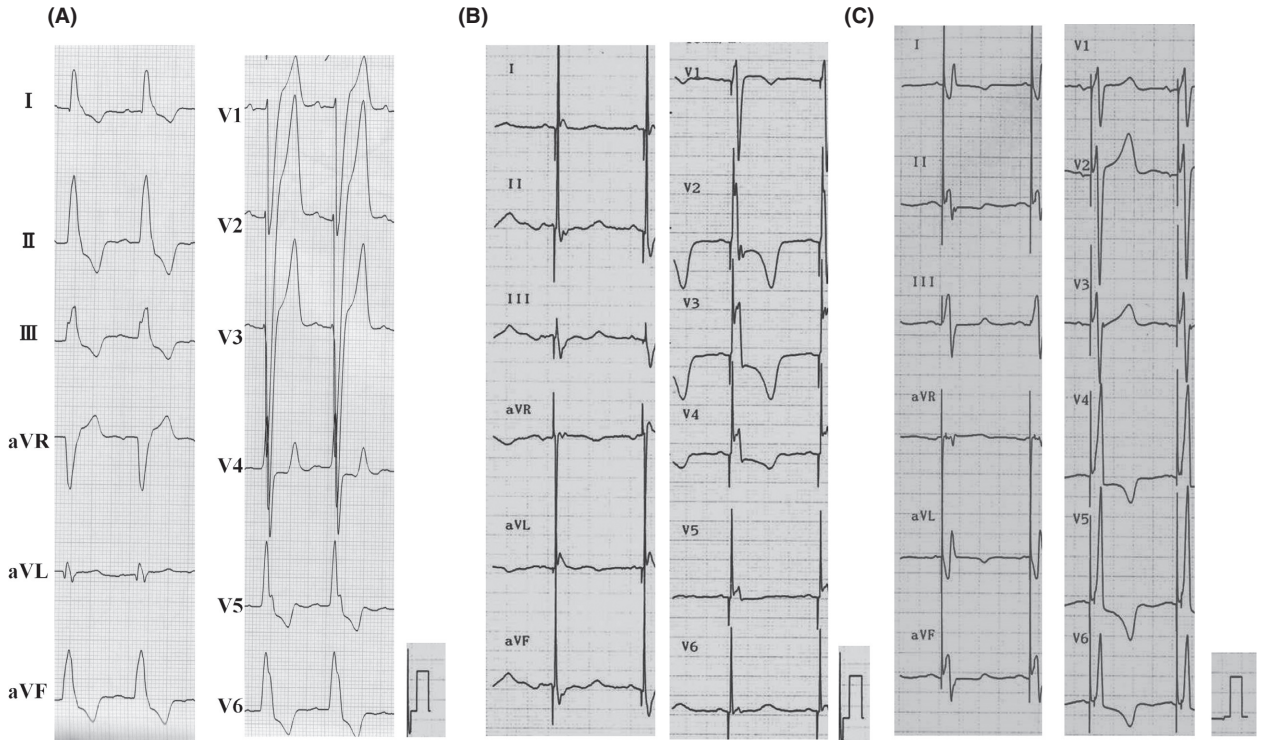

Figure 1. Twelve-lead electrocardiograms (ECG) before cardiac resynchronization therapy (CRT) (A), after CRT with low output of the left ventricular (LV) pacing (B) and with high output of the LV pacing (C). The ECG showed a complete left bundle branch block with wide QRS before CRT (A). After CRT with LV pacing stimulus output at 2.5V, the ECG showed a decrease in QRS width from 180 msec to 140 msec (B). Moreover, the morphology of the QRS had changed after increased pacing output to 3.5V without adjustment of intraventricular delay (C).

similar to that before pacing. Mechanical dyssynchrony was not improved by only adjustment of the AV and VV delay (Fig. 2A). Finally, the increase in pacing output of the LV lead from 2.5V to 3.5V improved the dyssynchrony (Fig. 2B) and also changed the ECG (Fig. 1C). After the adjustment of pacing parameters including pacing output, shortness of breath on exertion was improved, and BP fall during hemodialysis disappeared.

Discussion

Over the last decade, CRT has become an important modality in patients with severe heart failure. Up to one-third of patients may not experience any improvement in clinical status and/or reversal of cardiac remodeling after CRT based on current indication criteria [1]. One of the major potential reasons is related to suboptimal AV and VV timing [2]. Landmark CRT clinical trials such as MIRACLE [4], COMPANION [5], and CARE-HF [6] required the AV and VV optimization before discharge and at every follow-up. Based on these data, optimal AV and VV timing is needed to respond to CRT. However, in some patients, the tip of LV lead could be surrounded by scar tissue or slow impulse propagation area because LV lead implantation is often constrained by the patient's venous anatomy [3]. In these patients, electrical and mechanical dyssynchrony is not improved even with optimization of AV and VV delay. We reported a patient whose mechanical dyssynchrony did not improve after usual optimization of AV and VV pacing timing. However, he showed response to CRT after increasing LV pacing output. Underlying mechanism may be related to significant improvements in transventricular conduction time. Tedrow et al. [7] reported that paced ventricular activation can be changed by increasing stimulus strength since increased pacing stimulus strength captured an enlarged myocardial area, by producing a larger "virtual electrode". In addition Sauer et al. [8] showed that myocardial capture extends beyond a discrete slow impulse propagation area or scar tissue, which could result in ventricular activation change and more rapid conduction to a remote location. A similar report by Theis et al. [9] indicated that the LV transventricular conduction time, measured as LV stimulus artifact to first peak of the bipolar RV intracardiac electrogram, significantly improved with higher LV pacing output, and changes of QRS morphology was observed by increased LV stimulus intensity. Furthermore, we revealed that

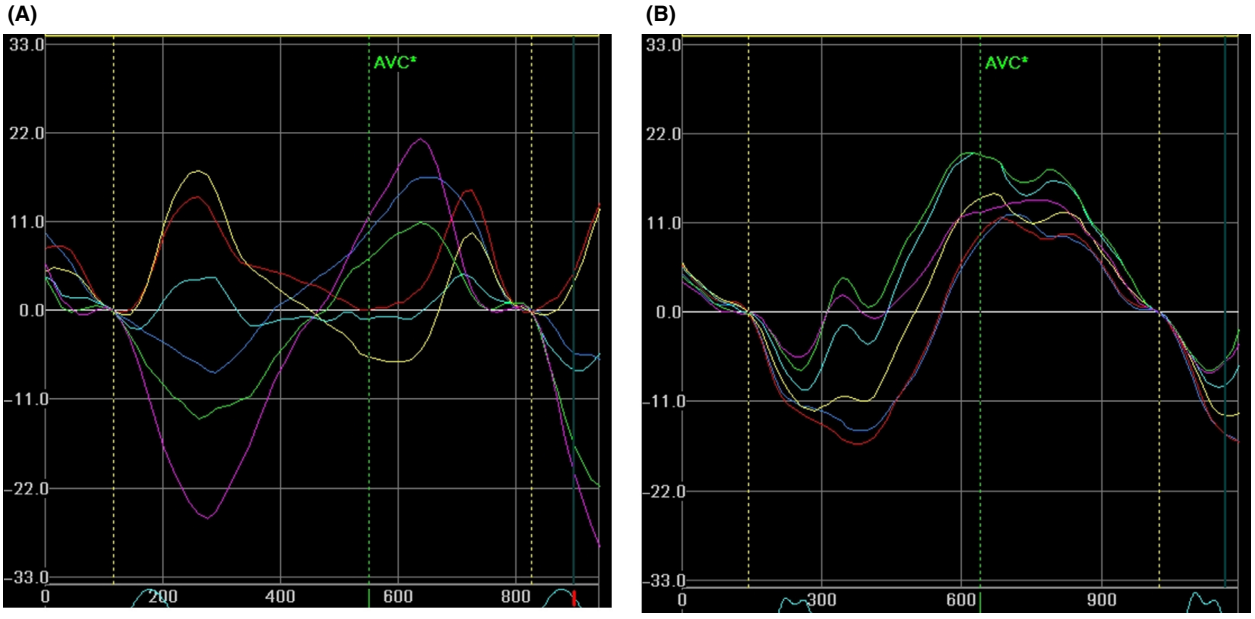

Figure 2. Velocity Graphs assessed by tissue Doppler images with low output of the LV pacing (A) and with high output of the LV pacing (B) during CRT. Improvement of the mechanical dyssynchrony with the high output at 3.5V was shown compared with the low output at 2.5V.

increasing LV pacing output in CRT improved LV mechanical dyssynchrony and cardiac function [10]. The long-term prognosis to confirm the possible positive impact of increasing LV pacing output on the response to CRT is still unclear and should be evaluated in a future study. Our patient showed that the improvement of dyssynchrony as well as the ECG morphology led to the favorable effect on symptoms such as improvement of shortness of breath and disappearance of BP fall during HD. Although there are some potential limitations of application to CRT including phrenic nerve capture and shortened device battery life at high pacing output, we strongly recommend increasing LV pacing output in CRT as a new optimization method for nonresponder.

Conflict of interest

None declared.

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