Case Report

A Case with no Hemodynamic Benefit from Right Ventricular Anodal Capture during Biventricular Pacing

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This case report describes a patient with a biventricular pacing system in whom right ventricular anodal capture had no hemodynamic benefit. While controlling the ventricular output, three morphologies of the paced QRS complex were obtained: right ventricular stimulation, biventricular stimulation, and biventricular pacing with additional stimulation from the anodal electrode in the right ventricle. While the QRS duration was 5 ms longer, the left ventricular systolic pressure and dP/dt_{max} during biventricular pacing without anodal capture of the right ventricle were greater than that during biventricular pacing with anodal capture. To avoid useless high output settings, the hemodynamic and clinical data should be compared with and without right ventricular anodal capture in each individual patient.
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Key words: Anodal stimulation, Biventricular pacing, dP/dt, Triple-site pacing

Introduction

Cardiac resynchronization therapy (CRT) has been shown to improve the symptoms in patients with moderate to severe heart failure.[1–4] Recently, anodal capture of the right ventricle (RV) has been described in patients with CRT-pacemakers[5,6] and CRT systems associated with defibrillators.[7] Bulava et al.[6] reported the incidence of the anodal capture of the RV and its impact on the effects of cardiac resynchronization by means of tissue Doppler imaging (TDI) analysis. However, the hemodynamic and clinical benefit of the anodal capture of the RV remains undetermined. We report a case without any hemodynamic benefit from RV anodal capture during biventricular pacing.

Case Report

A 70-year-old man had an extensive anterior myocardial infarction in 1982. He underwent coronary artery bypass graft surgery in 1994 and implantation of a dual chamber pacemaker (model 213, ELA Medical, Montrouge, France) for complete atrioventricular block in 2003. At that time, conventional bipolar leads (models 4092 and 4592, Medtronic, Minneapolis, MN, USA) were positioned in the RV apex and right atrial appendage. Despite

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optimal drug treatment, he developed congestive heart failure in 2004. During RV pacing the QRS complex exhibited a left bundle branch block configuration, QRS width of 190 ms, and inferior to superior ventricular activation, compatible with RV apical pacing. The intraventricular conduction delay between the left ventricular (LV) septal and lateral walls measured by tissue Doppler imaging was 253 ms. Therefore, an upgrade to a biventricular pacemaker system was performed. The patient's functional class was New York Heart Association (NYHA) class III, cardiothoracic ratio (CTR) of 48%, LV ejection fraction (EF) of 23%, and B-type natriuretic peptide (BNP) level of 614 pg/ml.

After an occlusive coronary sinus (CS) venogram, a unipolar LV lead (model 2187, Medtronic, Minneapolis, MN, USA) was placed via the CS into the antero-lateral vein. The LV threshold was 0.8 V, 1.0 mA at a pulse width of 1.0 ms, R wave amplitude 10.9 mV, and lead impedance 985 ohm. The RV threshold was 0.5 V, 1.0 mA at a pulse width of 1.0 ms, R wave amplitude 18.8 mV, and lead impedance 586 ohm. All leads were connected to a biventricular pulse generator (model 8040, Medtronic, Minneapolis, MN, USA) that was implanted in the left pectoral region. During the ventricular threshold test, the 12-lead ECG exhibited 3 different QRS configurations (**Figure 1**). At an output setting between 0.5 V and 1.5 V with a pulse width of 0.4 ms, only the RV was captured (**Figure 1A**). At an output setting between 2.0 V and 4.0 V, the RV and LV were captured (**Figure 1B**). Further, at an output setting of $\geq$5.0 V, both ventricular cathodes and the anodal electrode of the RV were captured (**Figure 1C**). The difference between the two QRS configurations during biventricular pacing was determined to be due to the additional stimulation from the anodal electrode in the RV, because the same change in the QRS morphology was observed during bipolar and unipolar RV pacing at an output setting of $\geq$5.0 V. The QRS width was 190 ms during RV pacing, 135 ms during biventricular pacing without anodal capture of the RV, and 130 ms during biventricular pacing with additional anodal capture of the RV. During biventricular pacing with additional anodal capture of the RV, the R-wave amplitude in leads I and aVL increased and the R/S ratio in lead V1 decreased. The distance from the tip of the RV lead to the ring was 17 mm.

Five days after the implantation, the hemodynamic effect of the triple-site pacing, i.e. standard biventricular cathodal pacing from the RV and LV plus additional anodal capture of the RV, was evaluated. A standard 5-Fr pigtail catheter was advanced to the LV apex to measure the ventricular pressure. The hemodynamic data was obtained after 2 minutes of steady-state pacing and the results reflected the values derived from an average of at least 15 sequential cycles. The LV dP/dt was calculated during real time and its maximal value (dP/dt_{max}) was determined. The LV systolic pressure and dP/dt_{max} were 104 mmHg and 665 mmHg/s during cathodal RV pacing, 115 mmHg and 853 mmHg/s during biventricular pacing without anodal capture of the RV, and 109 mmHg and 821 mmHg/s during biventricular pacing with additional anodal

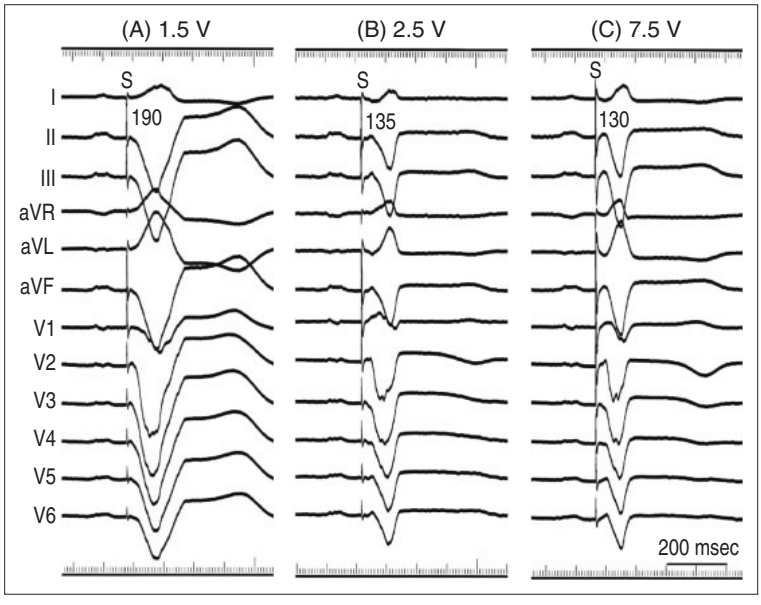

Figure 1 Twelve-lead ECGs recorded with different ventricular output settings.
(A) At an output setting between 0.5 V and 1.5 V and pulse width of 0.4 ms, only the right ventricular cathode captured and the QRS width was 190 ms. (B) At an output setting between 2.0 V to 4.0 V with a pulse width of 0.4 ms, the right and left ventricular cathodes captured and the QRS width was 135 ms. (C) At an output setting of $\geq$5.0 V and a pulse width of 0.4 ms, both ventricular cathodes and the anodal electrode of the right ventricle captured and the QRS width was 130 ms.
S = pacing stimulus.

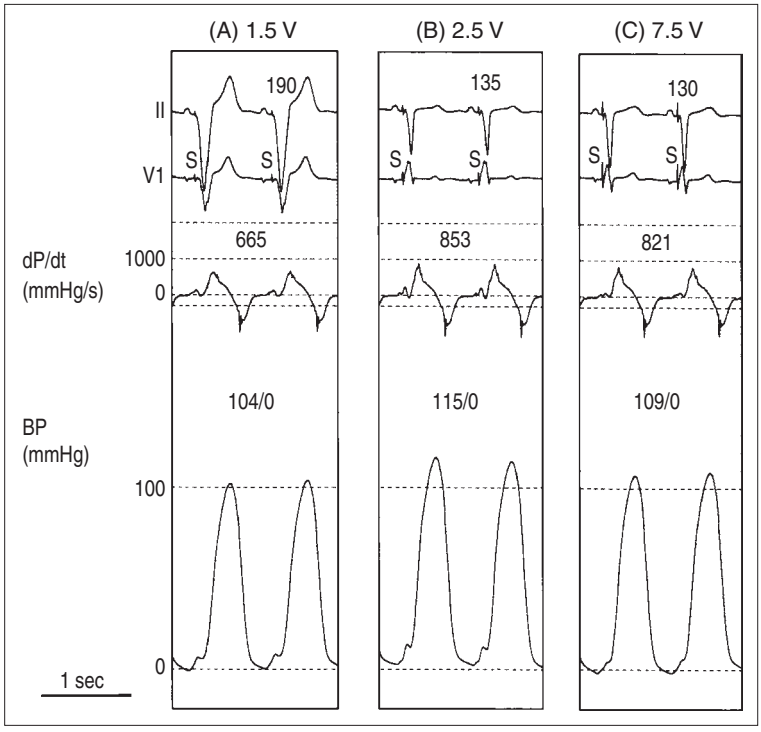

Figure 2 The hemodynamic data during the three different QRS morphologies.
The surface ECGs (leads II and V1), left ventricular dP/dt, and left ventricular pressure were simultaneously recorded. The left ventricular systolic pressure and dP/dt_{max} were 104 mmHg and 665 mmHg/s during cathodal right ventricular pacing (A), 115 mmHg and 853 mmHg/s during biventricular pacing without anodal capture of the right ventricle (B), and 109 mmHg and 821 mmHg/s during biventricular pacing with additional anodal capture of the right ventricle (C), respectively. While the QRS duration was 5 ms longer, the LV systolic pressure and dP/dt_{max} during biventricular pacing without anodal capture of the RV were greater than that during biventricular pacing with anodal capture.

capture of the RV, respectively (**Figure 2**). While the QRS duration was 5 ms longer, the LV systolic pressure and dP/dt_{max} during biventricular pacing without anodal capture of the RV were greater than that during biventricular pacing with anodal capture. Therefore, the ventricular output was programmed to 4.0 V to obtain biventricular pacing without anodal capture of the RV. After that, we performed TDI analysis but there was no significant difference between CRTs with and without RV anodal capture. After 6 months of effective resynchronization therapy, the patient's functional class improved from NYHA III to II, CTR decreased from 48% to 44%, LVEF increased from 23% to 45%, and BNP decreased from 614 pg/ml to 40 pg/ml.

Discussion

This case report demonstrates that anodal capture of the RV during biventricular pacing does not always have a hemodynamic benefit. While the QRS duration was 5 ms longer, the LV systolic pressure and dP/dt_{max} during biventricular pacing without anodal capture of the RV were greater than that during biventricular pacing with anodal capture.

In the first-generation devices with common right and left ventricular channels, a bipolar configuration was used for the pacing impulse to simultaneously stimulate the RV and LV using the distal tip of the electrodes as the cathodes and the ring of the RV lead as the anode. At higher pacing outputs, three sites were captured, i.e. by cathodal stimulation from the tips of the RV and LV leads and additional anodal capture of the RV inferior wall or septum, depending on the position of the ring of the RV lead. Bulava et al.[6] recently reported the incidence of anodal capture by the RV lead in biventricular pacing systems, the effect on the ventricular depolarization pattern, and its impact on cardiac resynchronization, by means of TDI analysis. They reported that this phenomenon was found in 26% of cases with the first-generation biventricular devices. In almost all patients with this phenomenon, the QRS duration decreased from 10 to 20 ms and the QRS amplitude in leads I and aVL increased. In their study the TDI analysis of the LV basal segments exhibited a significant shortening of the systole period, together with a corresponding prolongation of the diastolic period, at the inferior wall of the LV, during triple-site pacing as compared to standard biventricular pacing in all patients. However, the hemodynamic and clinical data were not compared in their study.

While the reason for no hemodynamic benefit during triple-site pacing in our patient was undetermined, several factors can be considered. We believe that stimulating a later-activated LV region produces a larger response in CRT because it more effectively

restores the regional activation synchrony. Therefore, shortening of the systolic period in the inferior wall of the LV during triple-site pacing, which was observed in the study by Bulava et al.,[6] might be less effective or even worse in patients with normal inferior LV wall motion and a delayed systolic period in the anterior or lateral wall. Another aspect is the optimal site of the RV pacing lead for the biventricular pacing system. This is still undetermined and probably depends on the site of the LV pacing lead. We speculated that an anatomically and electrically distant site from the LV pacing site might be more effective. Therefore, anodal capture of the RV would have a benefit if the RV proximal electrode was located more distant from the LV lead than the RV tip electrode, however, it would have no benefit if the RV tip electrode was more distant from the LV lead than the RV proximal electrode.

While Bulava et al. reported some beneficial effect of RV anodal capture during biventricular pacing, there are several problems in this phenomenon. First, ignoring it could lead to inadequate programming of the device. A loss of RV anodal capture could be mistaken for loss of LV capture and the LV output being programmed at unnecessarily high level.

Another important disadvantage of RV anodal capture during biventricular pacing is its incompatibility with interventricular delay (V-V delay) programming. While the generator in this report was an old model without a V-V delay programming function, a problem will be encountered at the time of generator replacement in the future.

In conclusion, RV anodal capture during biventricular pacing does not always have a hemodynamic benefit. To avoid useless high output settings, the hemodynamic and clinical data should be compared with and without RV anodal capture in each individual patient.

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