HEART FAILURE

COMPLEX CASE STUDY

Failure of Cardiac Resynchronization Therapy due to Inappropriate Inhibition of Left Ventricular Pacing during Biventricular Pacing

ROBERT H. HELM, MD, ADVAY G. BHATT, MD and KEVIN M. MONAHAN, MD

Boston University School of Medicine, Boston, MA

ABSTRACT. Cardiac resynchronization therapy (CRT) has been shown to be an effective therapy in patients with cardiomyopathy and left bundle branch block; however, 20% to 30% of patients receiving CRT fail to respond, and this maybe in part due to poor therapy implementation. Recent data suggest that a basal left ventricular (LV) lead location is more efficacious than apical placement. We describe a case in which CRT exacerbated heart failure because of the combined effect of a basal lead location and left ventricular sensing algorithm. Disabling this sensing algorithm improved CRT delivery.

KEYWORDS. biventricular pacing, cardiac resynchronization therapy, left atrial far-field oversensing, left ventricular lead placement, left ventricular protection period.

Introduction

Cardiac resynchronization therapy (CRT) has emerged as a highly effective therapy in patients with cardiomyopathy and left bundle branch block. Unfortunately, 20% to 30% of patients receiving the therapy fail to demonstrate clinical improvement. Basal left ventricular (LV) lead location has been shown to be more efficacious than apical. We describe a case where implementation of CRT was detrimental because of the combined effect of basal lead location and left ventricular sensing.

Case report

A 65 year-old male with non-ischemic cardiomyopathy was referred for upgrade of his implantable cardioverter-defibrillator (ICD) for progressive heart failure despite an optimized heart failure regimen. He was quite functionally limited and had New York Heart Association class III symptoms. His electrocardiogram showed non-specific intraventricular conduction delay with a QRS duration of 166 ms. He was electively brought to the electrophysiology laboratory, and the pocket was opened. The coronary sinus was accessed, and a balloon-occlusive venography of the coronary sinus showed a small lateral branch of the coronary sinus. A Medtronic 4136-88 (Medtronic Inc, St. Paul, MN) was advanced into the lateral branch vessel, and suitable lead parameters were found. The pacing impedance was 1,146 ohms, and the capture threshold was 0.4 volts at 0.5 ms. There was no phrenic nerve or atrial capture with high-output pacing. The lead connected to a Boston Scientific COGNIS TM 100-D generator (model # N119; Boston Scientific Corp., Boston, MA), and the wound was closed. The following day, posterior-anterior and lateral chest renographs showed stable basal LV lead position (Figure 1a,b). Interrogation of the device also demonstrated stable LV lead parameters, and the patient was discharged.

Two weeks later, he was admitted to our institution with acute decompensated heart failure. A chest renograph showed stable LV lead position (Figure 1c,d). Interrogation of his ICD showed unchanged LV lead parameters. The ventricular pacing burdens were 66% and 82% for the left ventricular (LV) and right ventricular (RV), respectively. The discrepancy was due to inhibition of LV pacing during DDD pacing (Figure 2a). When pacing was inhibited, two
electrical potentials were observed on the LV channel that corresponded to atrial and ventricular activation (Figure 2b). The device sensed the first component (far-field left atrial component) as LV activation and inhibited the scheduled LV pace event to avoid pacing in the vulnerable recovery period. The scheduled RV pace event was delivered. We concluded that the lack of ineffective biventricular pacing and burden of RV-only pacing contributed to heart failure exacerbation in our patient. Disabling the LV sense feature restored effective biventricular pacing.

Discussion
Identifying causes of non-response to CRT can be challenging. Assuring adequate therapy implementation is important before classifying the patient as a non-responder. The present case report highlights an unusual situation where device programming and lead placement led to poor CRT implementation. Left atrial far-field sensing has been previously reported in CRT systems that shared a common ventricular port (i.e., RV and LV leads connected via a Y-adaptor). In
conventional CRT systems with dedicated LV ports, left atrial sensing has only been reported in a patient with new onset atrial flutter that resolved with restoration of sinus rhythm. Our case is the first to demonstrate this phenomenon occurring in sinus rhythm, and this situation may not be as easily recognized as atrial flutter. Pacing within a chamber without sensing has the theoretical risk of arrhythmia induction. Boston Scientific cardiac resynchronization devices have independently programmable RV and LV pacing and sensing. While RV sensing is used for RV-based timing and RV pacing inhibition, LV sensing is only used to inhibit a scheduled LV pace event. There have been rare case reports of CRT having a pro-arrhythmic effect. Thus, following a sensed LV event, Boston Scientific incorporated a programmable protection feature known as the Left Ventricular Protection Period (LVPP). The LVPP inhibits a scheduled pace event for 300 to 500 ms to avoid LV pacing during the vulnerable recovery period (T-wave). This feature is independent of RV sensing and does not reset the lower rate timer. Among other cardiac device manufacturers including Medtronic, St. Jude, and Biotronik, Biotronik is the only corporation that has a similar LV sense feature, and it is referred to as LV T-wave protection. Basal LV lead location is desirable, and in our patient it resulted in a dual-component electrogram corresponding to atrial and ventricular activation. With the LVPP parameter turned “ON,” the left atrial far-field electrogram was sensed as an LV event, and the scheduled LV pace event was inhibited during biventricular pacing. The RV pace event was not inhibited. This resulted in resynchronization failure, a high burden of RV pacing, and acute decompensated heart failure. Disabling the LVPP feature, which is nominally turned “ON,” achieved appropriate biventricular pacing with simultaneous LV and RV pace events. An alternative treatment strategy would have been to change the LV sense configuration to an extended bipolar configuration using the distal LV dipole and RV coil. The distal dipole is further from the mitral annulus and is less likely to record atrial activity (Figure 3). Inhibition of LV pacing during CRT pacing was not observed with the extending LV bipolar configuration in sinus rhythm.
In conclusion, a basal LV lead location with far-field left atrial sensing in the presence of LV sensing (LVPP) may lead to inappropriate inhibition of LV pacing and poor implementation of CRT therapy.

References