Unusual Electrocardiogram During Cardiac Resynchronization. What is the Mechanism?

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ABSTRACT

Figure 1 shows the ECG of a patient with a cardiac resynchronization therapy (CRT) device coming back for follow-up 3 months after implantation. The device was programmed to the VVI mode to rule out fusion with the conducted spontaneous QRS complex. Should you be concerned that the tracing during pacing shows a left bundle branch block (LBBB) with right inferior axis deviation in the frontal plane rather than the classic pattern of a right bundle branch block pattern (defined as a dominant R wave) in lead V1?

KEYWORDS
Cardiac resynchronization, left ventricular stimulation, left bundle branch block, right bundle branch block
Discussion

There is no problem with the CRT system. The right ventricular (RV) lead was placed in the RV outflow tract and the left ventricular (LV) lead was placed in a posterolateral coronary vein. Fig 2 shows the ECG during VVI pacing of the LV showing the expected right bundle branch block (RBBB) pattern and Fig 3 shows the ECG during VVI pacing of the RV outflow tract.

Biventricular pacing with the RV lead at the apex

Lead V1 is often positive during biventricular (BiV) pacing with the RV lead located at the apex. 1 The frontal plane QRS axis during BiV pacing usually moves superiorly from the left (from the usual site of monochamber RV apical pacing) to the right superior quadrant in an anticlockwise fashion if the ventricular mass is predominantly depolarized by the LV pacing lead. The frontal plane axis may occasionally reside in the left superior rather than the right superior quadrant during uncomplicated BiV pacing.

Causes of a Negative QRS Complex in Lead V1 During CRT

A negative QRS complex in lead V1 during uncomplicated BiV pacing (with an apical RV lead) probably reflects a different activation of an heterogeneous BiV substrate (ischemia, scar, His-Purkinje participation in view of the varying patterns of LV activation in spontaneous LBBB, etc.), and does not necessarily indicate a poor (electrical or mechanical) contribution from LV stimulation. Although the lack of a dominant R wave in lead V1 may be of no importance, it requires investigation to rule out the problems listed below. This is especially important in the presence of a QS complex in lead V1 suggesting that RV pacing depolarizes most, if not all, of the LV.

A negative paced QRS complex may occur in the following circumstances: Incorrect pla-
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Cement of lead V₁ (too high on the chest), lack of LV capture, LV lead displacement, marked LV latency (exit block), (2) CRT with the RV lead in the outflow tract, marked delay in the local propagation of LV activation from the stimulation site (with or without abnormal latency), (3) ventricular fusion with the conducted QRS complex, coronary venous pacing via the middle cardiac vein (also the anterior cardiac vein), or even unintended placement of 2 leads in the RV.

Biventricular pacing with the RV lead in the outflow tract In our experience with over 100 cases, we have found that during biventricular pacing with the RV lead in the septal area or outflow tract, the paced QRS in lead V₁ is often negative and the frontal plane paced QRS axis is often directed to the right inferior quadrant (right axis deviation). This may create a problem in troubleshooting because the ECG may resemble that of simple monochamber RV septal or outflow tract pacing with a LBBB pattern and right inferior frontal axis deviation mimicking loss of LV pacing.

**Left Ventricular Latency**

The interval from the pacemaker stimulus to the onset of the earliest paced QRS complex is called latency. An isoelectric onset of the QRS complex in one or only a few leads can mimic latency. Consequently the demonstration of latency requires a 12-lead ECG taken at fast speed for diagnosis. During RV pacing this interval normally measures <40 ms. A prolonged latency interval represents first-degree pacemaker exit block. At physiologic rates pronounced latency is uncommon during RV pacing but may be more prevalent during LV pacing because of LV pathology including scars. The causes of abnormal LV latency include scarring, ischemic myocardium, nonischemic cardiomyopathy, hyperkalemia and antiarrhythmic drugs. Prolonged LV latency delays LV depolarization during simultaneous BiV pacing and can produce an ECG pattern dominated by the pattern of RV pacing (2). Prolonged LV latency intervals during stimulation from within epicardial cardiac veins may be due to interposed tissue and electrode proximity to a scar preventing direct contact between electrode and healthy LV myocardium. The QRS complex in a conventional surface ECG cannot differentiate failure of excitation (latency) from delayed propagation in the myocardium around the electrode (without latency). This concept was illustrated in the case of Grimley et al (3) where a CRT patient with ischemic cardiomyopathy where BiV pacing (RV lead at the apex) showed a LBBB pattern identical to monochamber RV pacing. There was no evidence of latency. The observations were attributed to delay or the emerging LV impulse in a region of scarring as evidenced by the LBBB pattern (configuration varied from a rate of 75 ppm to 85 ppm) during monochamber LV pacing.

**Effect of Stimulus Amplitude And Pacing Rate**

An increase in the pacing rate may prolong the abnormal stimulus to QRS interval during RV and LV stimulation while a prolonged latency interval may shorten by slowing the pacing rate (2). An increase in stimulus amplitude may shorten the stimulus-QRS interval and a decrease accentuates the latency interval (2). In this respect, some investigators have shown that increasing the LV stimulus output decreases interventricular conduction time (4,5). Investigations with temporary unipolar LV pacing (anode in the inferior vena cava) have shown...
that patients with an LV scar or infarction near the pacing site may exhibit a change in paced QRS configuration, a decreased latency interval, shorter QRS duration and conduction time to the RV when the LV output is increased (4). These changes were independent of RV anodal stimulation. Increasing the LV output strength probably works by decreasing the latency interval and/or enlarging the area of myocardial capture beyond a site of conduction block creating a larger virtual electrode. In patients with implanted CRT devices (unipolar LV lead and anode in the RV proximal electrode), increasing the LV output may also reduce the paced QRS duration, the conduction time from LV to RV and may alter QRS configuration by a combination of RV anodal pacing and a larger virtual electrode effect (5,6). A larger virtual electrode may be of particular importance during pacing of diseased myocardium but may be complicated by phrenic stimulation, rapid battery depletion and RV anodal capture. Bipolar LV leads are needed to show the true impact of increasing the LV output because they are not associated with RV anodal capture.

**Programming the Interventricular (V-v) Interval**

Prolonged LV latency intervals with delayed LV activation can result in a suboptimal hemodynamic CRT response that is potentially correctable by advancing LV stimulation (before RV stimulation) via a programmable interventricular (V-V) delay. The hemodynamic consequences depend on the difference (delta latency) between right and left sided latency intervals during BiV pacing rather than absolute values. Occasionally, programming the V-V delay cannot pre-excite the LV sufficiently to provide optimal hemodynamics. In this situation the RV channel should be turned off.

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**FIGURE 1**

*Presenting ECG showing pacing with a left bundle branch block pattern and right axis deviation in the frontal plane. The device was programmed to the VVI mode to exclude fusion with the conducted spontaneous QRS complex.*
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**REFERENCES**

**FIGURE 2**

ECG during VVI pacing of the left ventricle showing a right bundle branch block pattern with right axis deviation.

**FIGURE 3**

ECG during VVI pacing of the right ventricular outflow tract showing pacing with a left bundle branch block pattern and right axis deviation in the frontal plane. Note the QRS duration is longer than that in Fig 1.