# Single Lead Cardiac Resynchronization in the Presence of RBBB

**Paul A. Levine, M.D., FACC**
**Vice President, Medical Services**
**St. Jude Medical CRMD**
**Clinical Professor of Medicine**
**Loma Linda University School of Medicine**

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The utilization of specific sites of stimulation is becoming recognized as a valuable adjunct to rate and the AV interval in optimizing hemodynamics for patients requiring permanent pacing therapy. This has led to the concept of cardiac resynchronization therapy in patients with a marked intraventricular conduction abnormality who have end-stage dilated cardiomyopathy. When the etiology of dilated cardiomyopathy is either idiopathic or ischemic, the most common intraventricular conduction abnormality is LBBB. Given that pacing from an RV endocardial location also results in a LBBB pattern, standard dual chamber pacing can do little with respect to optimizing the ventricular activation sequence and may even contribute to ventricular dysfunction developing on a chronic basis. The growing appreciation that a disordered activation sequence, most commonly of the LBBB type, may significantly compromise hemodynamics led to the concept of biventricular pacing. Simultaneous stimulation of both ventricles results in fusion with normalization of the QRS although it rarely becomes absolutely normal. The effective normalized activation sequence results in improved hemodynamics, a reduction in mitral regurgitation and overall improvement in cardiac function. The role of biventricular pacing in the treatment of end-stage dilated cardiomyopathy is growing in response to this observation.

As the role of ventricular activation sequence becomes better appreciated, it seems reasonable to attempt to normalize this wherever possible. For study purposes, the role of pacing in the management of congestive heart failure is restricted to those patients who do not require a pacemaker for standard bradycardia indications so as to not confuse the analysis in patients who require pacing for standard indications. However, the lessons from the biventricular CHF pacing studies should be applied to patients who have a generally accepted indication for pacing therapy. The following case is a prime example of this and reflects the growing appreciation of the role of the ventricular activation sequence.

The patient is a 60+ year old man whose pacemaker was implanted for symptomatic sinus node dysfunction. He had intact AV nodal conduction with a right bundle branch block ventricular activation pattern. His pacemaker, St. Jude Medical's Trilogy DR+ ® model 2364 had been programmed to the DDDR mode with a long AV delay to allow for intact AV nodal conduction. This resulted in effective inhibition of the ventricular channel with the

beneficial effect of reducing overall battery current drain and increasing device longevity. However, even more important than device longevity is optimization of ventricular function. On the most recent visit, the growing appreciation of the role of ventricular activation sequence was taken into consideration and the AV delay adjusted to force ventricular fusion. The LBBB pattern associated with RV apical pacing combined with the patient's RBBB ventricular activation pattern resulted in a near-normal QRS pattern.

Figure 1:

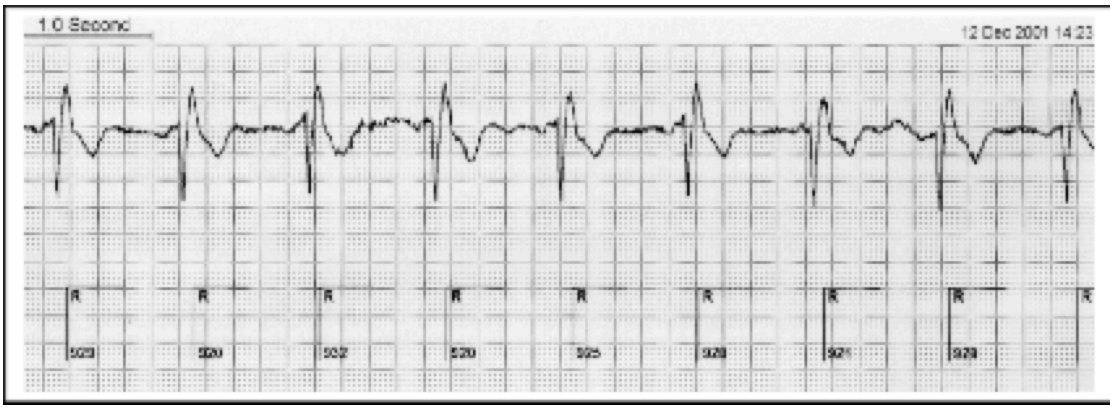


**Figure 1 was recorded with the pacemaker programmed to the VVI mode and a low rate to allow AV conduction and a demonstration of his usual ventricular activation pattern. The PR interval is 160 ms and the QRS duration is 160-180 ms with a RBBB pattern. The monitor lead is a modified V1.**

Figure 2:

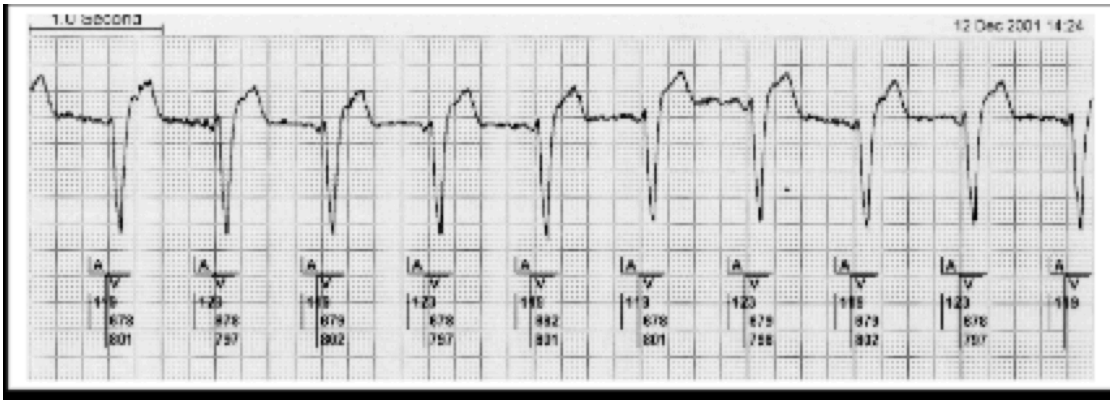


**Figure 2 was recorded just prior to the evaluation of the ventricular capture threshold. The AV delay was shortened to 125 ms to force ventricular capture at an interval shorter than his intrinsic AV nodal conduction. This resulted in the expected LBBB pattern associated with RV apical pacing. Even this complex, however, represents some degree of ventricular fusion as the QRS duration is not as wide as commonly seen with RV apical pacing.**

Figure 3:

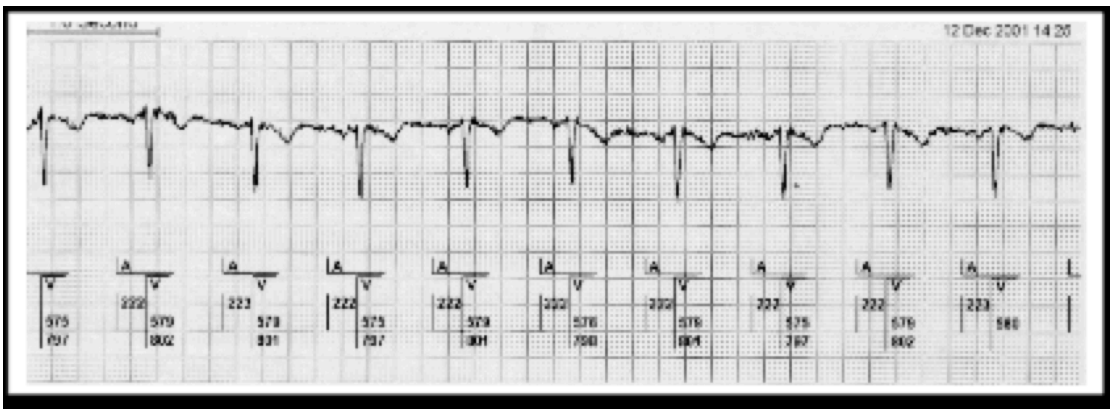

**Figure 3 represents fusion between the native conduction pattern and RV apical pacing. The ECG was monitored while adjusting the AV delay. In this case, the AV delay that provided the narrowest QRS was 225 milliseconds.**

As many physicians and support staff might be misled by the above complex and the rationale for this specific AV delay, the "Patient Information" in the pacemaker was modified so that this would be explained (Figure 4).

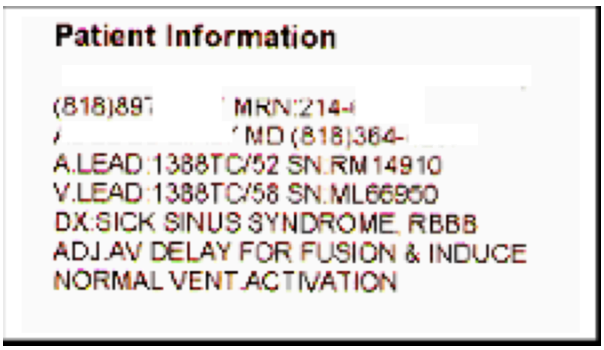


**Figure 4: There is limited space in the Patient Information section. As such, the information needs to be cryptic but free text is allowed. The patient's and physician's names have been redacted. The A and V leads are identified as is the diagnosis. It is also noted that the patient has RBBB and that the AV delay was adjusted to intentionally create fusion in order to induce a normal ventricular activation sequence.**

**Summary:**

In addition to selecting the optimal resting base rate, the rate-modulated settings and the AV delay, one should begin to think about ventricular activation sequence. When there is normal ventricular function, a normal QRS complex and intact AV nodal conduction at a normal PR interval, a long AV delay (either fixed or preferably achieved with AV/PV Hysteresis [AutoIntrinsic Conduction Search]) is associated with optimal hemodynamics. When there is an intrinsic intra- or interventricular conduction abnormality, careful attention to placement of the ventricular lead is being recognized as being of increasing value. In those patients who have a RBBB with intact AV nodal conduction, careful adjustment of the AV delay with a standard right

ventricular endocardial lead can result in consistent fusion that effectively normalizes the ventricular activation sequence.