Dramatic Response to Cardiac Resynchronization Therapy With AV Delay Optimization in Narrow QRS Heart Failure

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SUMMARY

Cardiac resynchronization therapy (CRT) has been shown to be effective for heart failure. However, as outlined in the AHA/ACC/HRS Appropriate Use Criteria, CRT is not strongly recommended for patients with a narrow QRS complex. We describe a case of dilated cardiomyopathy and narrow QRS complex in which we obtained a dramatic response to CRT by optimizing the atrioventricular (AV) delay. The patient was a 61-year-old man with intractable heart failure. Echocardiography showed a low ejection fraction of 22% but no dyssynchrony. Because he had been hospitalized many times for congestive heart failure despite β-blocker and diuretic treatment, we decided to use CRT. However, after implantation of the CRT device, the QRS complex widened abnormally, and his symptoms worsened. He was re-admitted 2 months after CRT implantation. We examined the pacemaker status and optimized the AV delay to obtain a “narrow” QRS complex. The patient’s condition improved dramatically after the AV delay optimization. His clinical status has been good, and there has been no subsequent hospitalization. Our case points to the effectiveness of CRT in patients with a narrow QRS complex and to the importance of AV optimization for successful CRT. (Int Heart J 2015; 56: 671-675)

Key words: CRT responder, Narrow QRS complex, Dilated cardiomyopathy

The effectiveness of cardiac resynchronization therapy (CRT) is well understood in cases of heart failure with a wide QRS complex ≥150 ms. Whether CRT is effective in patients with a narrow QRS complex is controversial, and its application in such patients is not usually recommended. However, in clinical practice, we often see patients with severe heart failure and a narrow QRS, and these patients are generally not considered candidates for CRT. Thus, it is difficult to manage these patients. Here, we present a case of dilated cardiomyopathy with a narrow QRS complex that was treated successfully by CRT with AV optimization. The good outcome achieved in this case points to the possibility of expanding CRT beyond current guidelines.

CASE REPORT

A 61-year-old man with dilated cardiomyopathy was referred to our hospital because of drug-refractory heart failure. Echocardiography showed an enlarged left ventricle with a low ejection fraction of 22%, and New York Heart Association (NYHA) class III heart failure was noted. His plasma B-type natriuretic peptide (BNP) concentration was 1793 pg/mL. Sinoatrial rhythm with a narrow QRS complex of 112 ms was seen on the electrocardiogram (ECG) (Figure 1). Despite β-blocker (carvedilol), diuretic (furosemide), and methyldigoxin (lanirapid) therapy, the patient had been hospitalized repeatedly for acute-congestive heart failure. The β-blocker dosage was 1.25 mg/day, which we thought might be inadequate. We attempted to increase the dosage, but the patient did not tolerate the increase well; hypotension and dizziness ensued. Therefore, although the QRS complex was narrow, we decided to apply cardiac resynchronization therapy (CRT) before trying again to increase the β-blocker dosage.

A CRT pacemaker (CRT-P, Medtronic, Minneapolis, MN, USA) was implanted without complication. After implantation of the right ventricular (RV) lead in the RV apex, coronary venography was performed, and a lateral branch was identified as a candidate vessel for left ventricular (LV) lead implantation. The LV lead was positioned at the midportion of the lateral branch, which was located at an angle opposite to that provided the most distance from the RV lead. The LV pacing threshold was 1.0 V at 0.5 ms without phrenic nerve stimulation. The right atrial (RA) lead was then positioned at the RA appendage. The device and leads used were the InSync III 8042 pulse generator, 5076 52-cm RV lead, 4196 LV lead, and 5076 45-cm RA lead (Medtronic). The RV pacing threshold was 0.5 V at 0.4 ms, and the RA pacing threshold was 1.0 V at 0.4 ms. Because sinus node function was normal, the device was programmed to DDD mode with a lower rate limit of 60 bpm, AV delay of 150 ms, and VV delay of 0 ms. This is the setting used routinely at our institution for the initial pro-
Unfortunately, after CRT-P implantation, the QRS complex widened to 142 ms (Figure 2), and the patient’s general condition worsened. Two-months later, he was re-admitted for heart failure, and intravenous dobutamine and milrinone drip were started. We checked the CRT-P status; the pacing threshold was good, and biventricular pacing was working properly. We sought to optimize the AV delay by looking at the QRS width on the ECG, and we decided to program the AV delay that yielded the narrowest QRS to achieve the maximum CRT effect. We did not optimize VV delay because an effect of VV delay optimization was unknown. We prolonged the AV delay in 10-ms increments while looking at the QRS width on the ECG and found the optimal delay to be 170 ms. Under this setting, the ECG showed a fusion beat, and the QRS width was 114 ms (Figure 3). With this AV delay optimization, the patient’s condition improved dramatically. His chest X-ray also improved in appearance (Figure 4), and he was discharged. One month after AV delay optimization, we succeeded in increasing the β-blocker dosage to 2.5 mg; however, a further increase brought about dizziness and hypotension. The patient has been free of symptoms and maintained NYHA class I functional status for 3 years. Reverse remodeling was confirmed by echocardiography, and his plasma BNP has remained under 50 pg/mL (Figure 5).

**Discussion**

Cardiac resynchronization therapy (CRT) is recognized
as a standard, effective treatment for severe heart failure. Many clinical trials have shown the benefit of CRT in patients with NYHA class III or IV functional status, an LV ejection fraction < 35%, and a QRS complex > 120 ms with left bundle branch block. A recent study indicated, however, that CRT did not reduce mortality or hospitalization due to heart failure and that it might actually increase the risk of death in patients with a narrow QRS complex.

According to the results of these trials, the “Appropriate Use Criteria” advise CRT for patients with a wide QRS complex. Yet, many heart failure patients with a narrow QRS complex are seen in clinical practice, and these patients need appropriate therapy; it should not be necessary for these patients to give up on life. Achilli, et al compared the effectiveness of CRT in patients with a wide versus those with a narrow QRS complex and reported that the clinical status and functional benefit were similar between the two groups. Our patient was turned into a CRT responder with AV delay optimization.

From our patient’s ECG, it appeared that the RV pacing may have constituted non-capturing pacing and that there was no fusion with the intrinsic conduction. In fact, the “fusion beat” derived from LV anterior fascicular pacing. AV delay optimization was successful for pacing at the selective left anterior fascicle, and this pacing narrowed the QRS complex. Recent studies have shown the LV lead position at the basal-mid lateral portion of a lateral branch to be optimal and to provide a better response to CRT than that provided by non-optimal LV lead positions. In the case we describe, positioning the LV lead at the mid-lateral portion corrected the left anterior fascicular conduction delay.

This synchronized LV pacing is beneficial for preserving LV function. van Gelder, et al studied the hemodynamic effect of intrinsic conduction during LV pacing versus biventricular pacing. The effect of LV pacing did not differ significantly from the effect of biventricular pacing; however, fusion during LV pacing made a clear and highly significant contribution to the maximum rate of LV pressure rise. The QRS duration during biventricular pacing was 219 ± 25 ms, and that during LV pacing in the presence of ventricular fusion was 198 ± 28 ms. Our case reinforces the notion that LV pacing associated with
ventricular fusion leads to improved cardiac function.

Within the last 20 years, RV pacing has been shown to lead to mechanical dyssynchrony by altering the intraventricular LV contraction sequence, resulting in deterioration of LV function. In the MOST trial, Sweeney, et al showed that ventricular dynnysynchrony induced by ventricular pacing even when AV synchrony is preserved increases the risk of hospitalization for heart failure. Thus, RV pacing should be avoided when intrinsic AV conduction is preserved, and LV pacing alone is adequate for synchronized RV-LV activation. Gasparini, et al reported that CRT provides a long-term clinical benefit in patients with a narrow QRS complex. They suggested that echocardiography did not clearly detect dyssynchrony and that careful evaluation might be needed for selection of CRT candidates. Finally, they advised caution before denying CRT to these patients on the basis of QRS width alone. In our case, cardiac dyssynchrony was not detected by echocardiography during the screening assessment due to very poor LV wall motion. Bleeker, et al evaluated cardiac echocardiography in patients with a narrow QRS complex and found severe LV dyssynchrony in a third of these patients. However, in clinical practice, it is difficult to assess cardiac dyssynchrony in patients with very poor cardiac wall motion. It is possible that cardiac dyssynchrony is underestimated when LV function is very low. Thus, it may be dangerous to select CRT candidates on the basis of either dyssynchrony or QRS width alone.

An explanation for the CRT response in our case remains speculative; however, the patient’s QRS complex was indicative of left anterior fascicular block, and this conduction disturbance may have been the obstacle to a functional response to CRT. Although the QRS morphology was not described in detail, previous studies have shown CRT to be efficacious in patients with a narrow QRS complex. Therefore, when considering candidates for CRT, we should not give up simply because of the QRS width or the existence of dyssynchrony. Moreover, especially in patients with a narrow QRS complex, AV optimization seems to be a very important factor for successful CRT.

In summary, we encountered a patient with dilated cardiomyopathy and a narrow QRS complex who responded to CRT after, and only after, optimization of AV delay. It is a well-known fact that CRT is most effective in patients with a wide QRS complex, ie, greater than 150 ms; however, our experience in this case reconfirms the effectiveness of CRT in patients with a narrow QRS complex. Our experience also reconfirms the importance of AV delay optimization for successful CRT. Furthermore, when heart failure cannot be controlled by medical treatment, CRT should be taken into consideration even in patients with a narrow QRS complex.

**DISCLOSURE**

The authors have no conflict of interest to report related to this study.

**REFERENCES**

8. Singh JP, Klein HU, Huang DT, et al. Left ventricular lead position and clinical outcome in the multicenter automatic defibrillator...