Optimization of Atrioventricular Delay and Follow-up in a Patient With Congestive Heart Failure With an Implanted DDD Pacemaker

—Case Report—

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It has been reported that cardiac function can be improved by implanting a DDD pacemaker (PM) and setting a short atrioventricular (AV) delay in patients with impaired cardiac function. A previous report found that the critical AV delay that induces diastolic mitral regurgitation (MR) may represent the upper limit of the optimal AV delay. The optimal AV delay can be predicted by a simple method: slightly prolonged AV delay minus the interval between the end of the atrial kick and complete closure of the mitral valve (duration of diastolic MR) at the AV delay setting. The patient was a 84-year-old man with an old myocardial infarction. He had repeated admissions to hospital for congestive heart failure. ECG showed prolongation of the PQ interval (0.28 s) and complete left bundle branch block. Cardiac function was improved by AV sequential pacing when the AV delay was set at 120 ms. After DDD-PM implantation, the cardiothoracic ratio decreased from 57 to 45% and cardiac function was improved from New York Heart Association class III to I. The AV delay was optimized during follow-up. Four years after PM implantation, the patient was in good condition without further hospital admission.

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Case Report

The patient, an 84-year-old man with an old myocardial infarction, had had percutaneous transluminal angioplasty for total occlusion of the proximal portion of the left ascending artery successfully performed in August 1994. Although there had not been restenosis or progression of the coronary artery lesion, he had repeated admissions to hospital for congestive heart failure (Fig 2A). Medications were unchanged during follow-up (oral administration of furosemide 40 mg, spironoractone 25 mg, digoxine 0.125 mg, captopril 18.75 mg, isosorbide dinitrate 60 mg, nicorandil 15 mg, aspirin 100 mg daily). He complained of dizziness, but the dose of captopril could not be increased and β-blocker could not be used because of the risk of hypotension or bradycardia.

ECG showed prolongation of the PQ interval (0.28 s) and complete left bundle branch block (Fig 2B). The His–ventricular interval was prolonged (90 ms on intracardiac electrogram) and the ejection fraction was 31%, estimated by left ventriculography. Marked diastolic MR was detected (Fig 3A) and during 80 beats/min AV sequential pacing, it was observed when the AV delay was set at 240 ms. The interval between the end of the atrial kick and complete closure of the mitral valve (duration of diastolic MR) was 120 ms (Fig 3B). Therefore, by our method, the predicted optimal AV delay was 240–120 = 120 ms, and the diastolic MR disappeared when the AV delay was set at 120 ms (Fig 3C). Cardiac output was 4.7 L/min during 80/min
atrial pacing, and was 4.7, 5.0, 5.1 and 4.8 L/min when the AV delay was set at 160, 140, 120 and 90 ms, respectively, during AV sequential pacing. Cardiac function was improved by AV sequential pacing when the AV delay was set at 120 ms.

A DDD pacemaker (PM) was implanted in May 1996, after which the cardiothoracic ratio (CTR) decreased from 57 to 45%, left atrial diameter decreased from 46 to 36 mm.
and cardiac function improved from New York Heart Association class III to I within 1 week (Fig 2C). However, the change in the diastolic left ventricular diameter was slight (65 to 63 mm). The optimal AV delay was 120 ms during P tracking ventricular pacing 1 week after pacemaker implantation. The AV delay was optimized at 100–120 ms by our method under P tracking ventricular pacing every 6 months during follow-up. Four years after PM implantation, he remained in good condition without hospitalization (Fig 2D).

Fig 3. Marked diastolic mitral regurgitation (arrow) was detected (A). During 80 beats/min AV sequential pacing, diastolic mitral regurgitation was observed when the AV delay was set at 240 ms (arrow). The interval between the end of atrial kick and complete closure of mitral valve (duration of diastolic mitral regurgitation) was 120 ms (B). The predicted optimal AV delay was 240 − 120 = 120 ms. Diastolic mitral regurgitation disappeared when the AV delay was set at 120 ms (C).
Discussion

Although the efficacy of a short AV delay in patients with severely impaired cardiac function has been reported, right ventricular pacing during AV sequential pacing has deleterious effects. It has been reported that biventricular pacing can improve the hemodynamics in patients with congestive heart failure, but the importance of optimizing AV delay is still controversial. The optimal AV delay may change from time to time with changes in cardiac function, although in previous reports, it has been fixed. However, in each patient, the optimal AV delay may not be a fixed value and thus should be adjusted constantly. Both optimization of the AV delay and shortening of the ventricular activation sequence may be important in patients with severely impaired cardiac function, and AV delay optimization may also be important during biventricular pacing.

In patients with a DDD-PM, the atrial kick may be interrupted by a too short AV delay, and filling time may be shortened by a too long AV delay. The optimal AV delay allows completion of end-diastolic filling flow prior to ventricular contraction, providing the longest diastolic filling time and occurs when the end of the A wave on transmural flow coincides with complete closure of the mitral valve. In our previous studies, diastolic MR was detected during the interval between the end of the A wave and complete closure of the mitral valve, and the critical PQ interval that induces diastolic MR may represent the upper limit of the optimal PQ interval. We devised a simple method for predicting the optimal AV delay: slightly prolonged AV delay – interval between the end of the atrial kick and complete closure of the mitral valve (duration of diastolic MR) at the AV delay setting.

DDD pacing and AV delay optimization are not always effective for congestive heart failure and the indication for this therapy remains unclear. The present patient had a prolonged PQ interval and complete left bundle branch block and was, therefore, a good candidate for short AV delay therapy. Moreover, the AV delay setting could be easily optimized by our new method during follow-up. This experience indicated that there are patients in whom heart failure can be controlled simply by DDD pacing with optimal AV delay without multisite pacing.

Although hemodynamic assessment by catheter examination provides the most reliable measurements of cardiac output and intracardiac pressures, it is invasive. The optimal AV delay is usually estimated by Doppler echocardiography, radionuclide angiography or plethysmographic impedance. Our new method is easy and not time-consuming and so AV delay optimization can be easily repeated, making it a useful method for the follow-up of patients with congestive heart failure and a DDD-PM.

In conclusion, DDD pacing with optimal AV delay is effective for some patients with severe congestive heart failure; namely, those with a prolonged PQ interval and left bundle branch block.

References

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