Right ventricular only pacing for cardiac resynchronization therapy

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We evaluated a 55-year-old male patient with ischaemic cardiomyopathy and a history of coronary artery bypass grafting for optimization of his cardiac resynchronization therapy (CRT). During follow-up over 12 years, left ventricular (LV) ejection fraction remained severely reduced at 23% with a severely dilated left ventricle (end-diastolic volume 185 mL, end-systolic volume 142 mL) and the patient symptomatic New York Heart Association (NYHA) III despite optimal medical therapy and consistently 99% biventricular pacing. While 48 h electrocardiogram (ECG) monitoring did not reveal any episodes of atrial fibrillation or other arrhythmias, slight variations in QRS morphology were observed throughout the day. Cardiopulmonary exercise testing showed a severely reduced functional capacity (VO₂ max 8.7 mL/min/kg, max. workload 92 W). QRS duration varied throughout the test and was shortest during higher heart rates, suggesting progressive fusion with intrinsic atrioventricular (AV) conduction. Chest X-ray demonstrated a midventricular lateral position of the epicardial LV lead and the right ventricular (RV) lead in a midventricular septal location (Figure 1A and B). CRT interrogation showed uneventful findings with 98% CRT pacing, of which 32% was biventricular and 68% LV only pacing; the latter resulted from the ‘adaptive CRT’ algorithm, which automatically adjusts atrioventricular intervals (AVIs) and biventricular/LV only pacing.

During CRT optimization, the patient was in sinus rhythm with complete right bundle branch block (RBBB) and a QRS duration of 160 ms (Figure 1C, a). 12-lead ECGs during RV, LV, and biventricular pacing with different modalities as well as during intrinsic rhythm were obtained (Figure 1C a–d). During biventricular pacing with adaptive AVI, the ECG reflected combined wave fronts of RV and LV pacing with no signs of fusion with intrinsic AV conduction at rest. A 12-lead ECG– and echocardiography-guided (transmitral inflow profile) evaluation of different manually programmed AV and interventricular (VV) intervals led to identification of the shortest QRS of 115 ms with RV-only pacing and an AV-delay of 130 ms (Figure 1C, d), resulting in a fusion of intrinsic AV conduction with RV pacing. This setting effectively led to resolution of the RBBB.

Of note, RV only stimulation without fusion with intrinsic AV conduction (VVI stimulation at a rate of 80 b.p.m.) was associated with a QRS duration of 184 ms, indicating myocardial capture outside the intrinsic right bundle. Fusion-optimized AVI with RV pacing demonstrated a significantly shorter QRS duration and an optimal transmitral inflow profile compared to the best biventricular pacing configuration with optimized interventricular intervals (RV pre-excitation of 40 and 60 ms relative to the left ventricle).
At follow-up 2 months later, the patient reported a reduction in exertional dyspnoea with a jump from NYHA class III to II and improved exercise capacity with increase of maximal workload from 92 to 104 W. Echocardiography demonstrated beginning left ventricular reverse remodelling with a decrease in left ventricular end-systolic volume from 142 to 122 mL as well as a decrease in end-diastolic volume from 185 to 170 mL and an increase in ejection fraction from 23% to 28%.

In summary, it is essential to carefully manage CRT patients following CRT implantation. Although algorithms for automated CRT optimization exist, one size does not fit all. During RBBB, RV pacing with careful manual AVI programming can lead to more effective resynchronisation than biventricular pacing.

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