

Right atrial appendage pacing may promote atrial fibrillation in patients treated with cardiac resynchronization therapy due to interatrial conduction prolongation

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Summary

The case report focuses on unfavorable hemodynamic consequences of the right atrial appendage (RAA) pacing in patients treated with cardiac resynchronization therapy (CRT-D) and shows how to assess this with echocardiography. A patient with artificial mitral and aortic valves and DDD pacemaker was upgraded to CRT-D due to severe heart failure. Under CRT the patient improved, left ventricular ejection fraction increased from 28 to 40%, right ventricular pressure normalized however, the frequency of atrial fibrillation episodes increased. The RAA-paced rhythm resulted in significant prolongation of the interatrial delay, which precluded optimal paced atrio-ventricular delay (AVD) programming, as with an increase in AVD-paced the biventricular capture was lost. We suspect that increment in left atrial pressure due to suboptimal AVD resulted in progression to chronic AF. *Geriatrics 2011; 5: 65-69.*

Keywords: cardiac resynchronization therapy, atrial pacing, right atrial appendage, atrial fibrillation, Doppler echocardiography, atrio-ventricular delay

Introduction

Cardiac resynchronization therapy (CRT) has become an approved treatment strategy for patients with heart failure, depressed left ventricular (LV) function, and prolonged QRS [1]. The most common arrhythmia in heart failure is atrial fibrillation (AF), as chronically elevated filling pressures contribute to the pathogenesis of AF. Right atrial appendage (RAA) is the conventional atrial pacing site, however, it can induce prolongation of atrial conduction time, which is a well known factor predisposing to AF development and maintenance. The present case report focuses on unfavorable hemodynamic consequences of RAA pacing in CRT recipients and shows how to assess this with echocardiography.

Case report

A 64-year-old woman, who had underwent the artificial mitral and aortic valves and DDD pacemaker

implantation (paroxysmal perioperative 3rd degree AV block) 3 years before, was admitted to our department due to chronic heart failure symptoms (NYHA class III) on optimal medical treatment. The patient had frequent episodes of paroxysmal AF. The ECG showed atrially-triggered ventricular pacing (VAT) 80 bpm. Coronary angiography revealed no abnormalities. Echocardiography showed normal artificial mitral and aortic valve function, normal LV end-diastolic diameter (52 mm), enlarged left atrium (53 mm), depressed LV ejection fraction (28%), severe tricuspid regurgitation, and increased right ventricular systolic pressure (68 mmHg). Mechanical dyssynchrony was observed [2]: “septal flash” phenomenon was present within the distal interventricular septum, septo-lateral difference in peak S'-wave in apical 4-chamber view was 90 ms, and interventricular mechanical delay (IVMD) was 70 ms. The atrio-ventricular synchrony was preserved: LV diastolic filling time (DFT_{LV}) was 440 ms = 48% of the RR interval. Global interatrial delay (IAD) during VAT pacing was prolonged to 40 ms (Figure 1a).

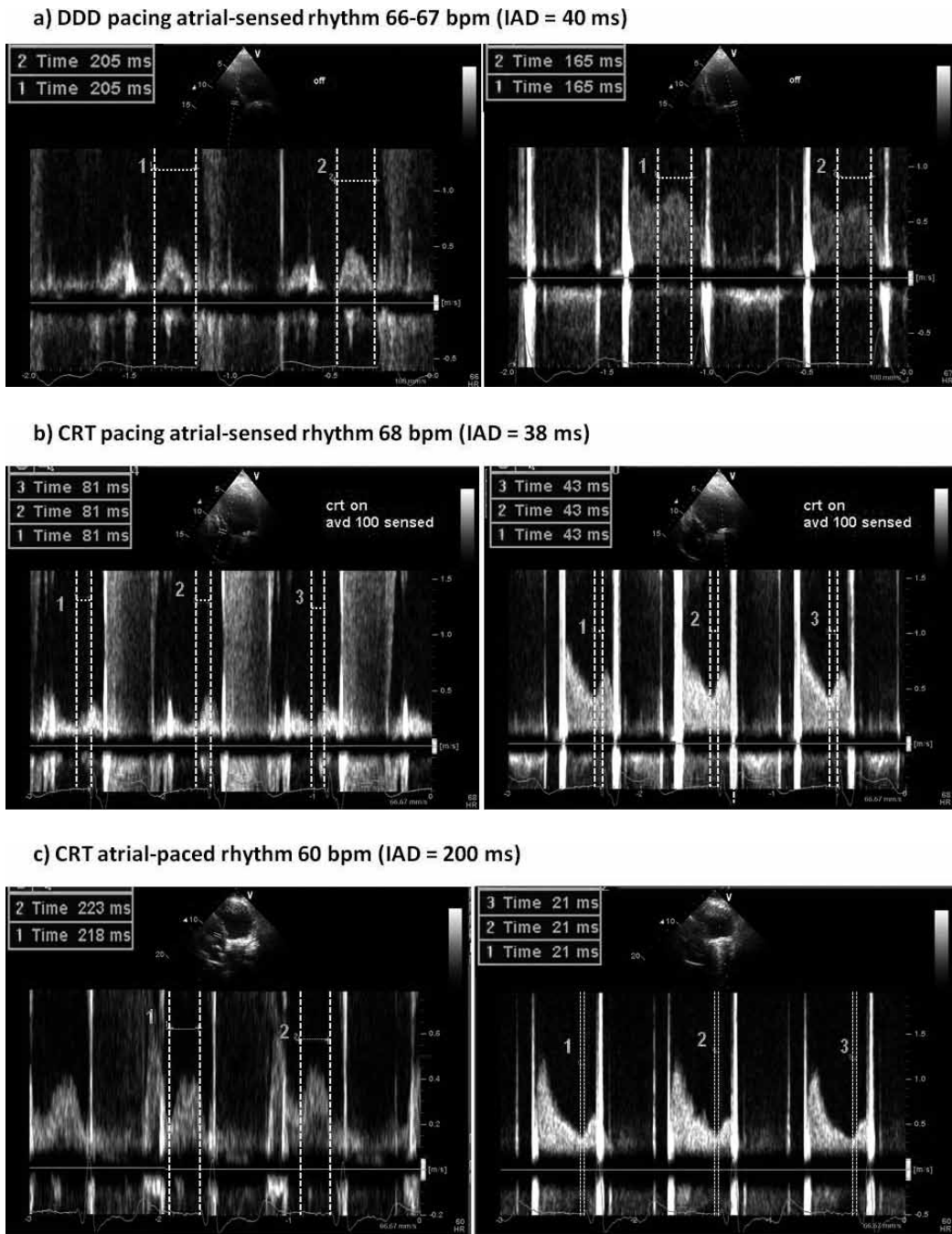


Figure 1. Global interatrial delay (IAD) assessment. Registration of the tricuspid (left) and mitral (right) inflow during apnoea and the difference between the beginning of the right and left atrial A-wave was calculated: a) before CRT upgrade: VAT pacing (67 bpm) IAD = 205 ms – 165 ms = 40 ms, b) after CRT upgrade: CRT atrial-sensed rhythm (66 bpm) IAD = 81 ms - 43 ms = 38 ms, c) CRT atrial-paced rhythm (60 bpm) with the long AVD paced of 200 ms IAD = 221 ms – 21 ms = 200 ms (this setting resulted in the loss of biventricular capture)

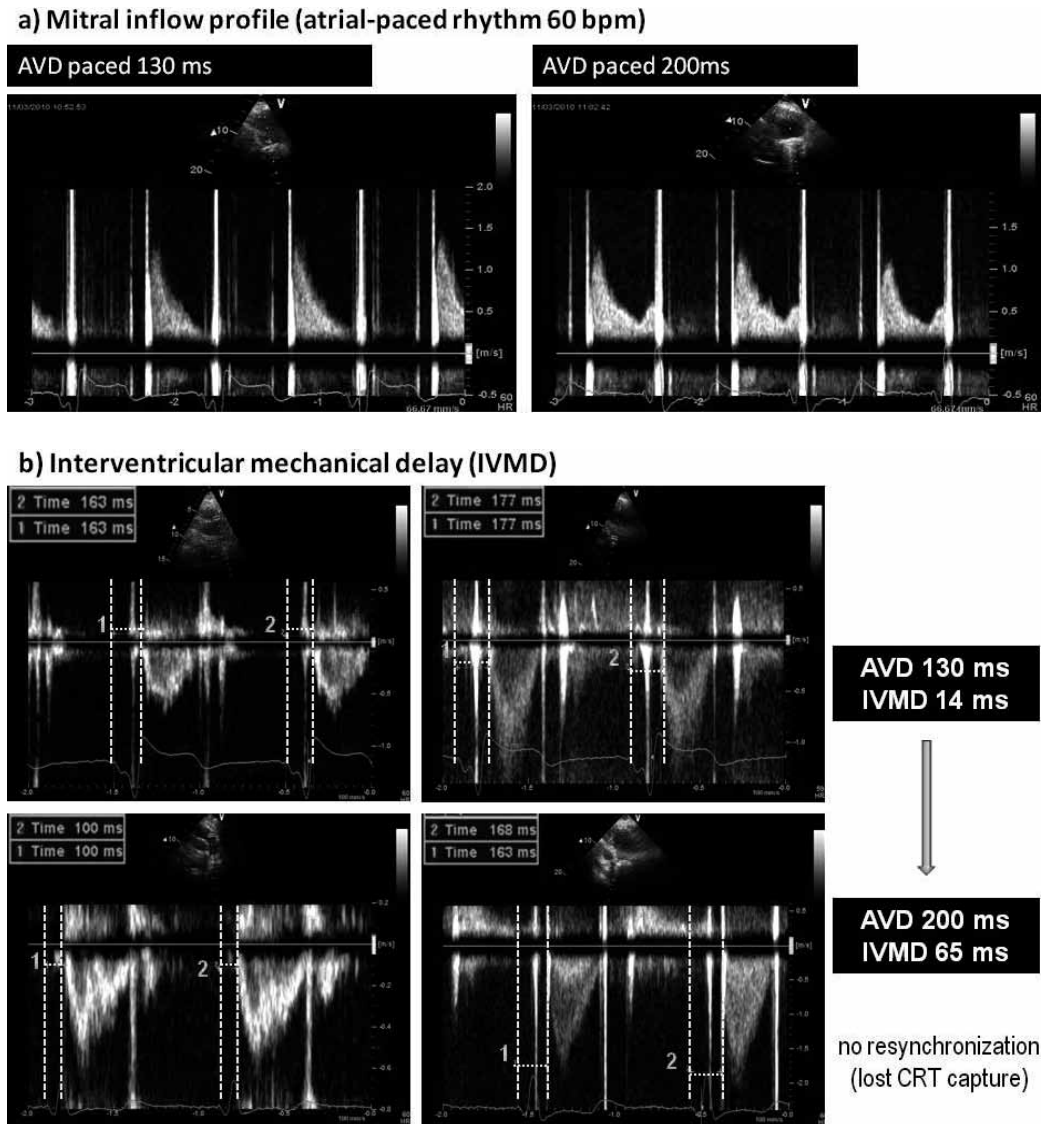


Figure 2. Atrio-ventricular delay (AVD) optimization during the CRT atrial-paced rhythm (60 bpm)

- a) mitral inflow profile, left: the AVD paced of 130 ms results in monophasic mitral inflow, right: the AVD paced of 200 ms was the one when a tiny mitral A-wave appeared,
- b) determination of the interventricular delay (IVMD) – time difference between right and left pre-ejection times, revealed that with the AVD paced of 130 ms (top) the IVMD was 177 ms – 163 ms = 14 ms, while with the AVD paced of 200 ms (bottom) the IVMD was 165 ms – 100 ms = 65 ms, which is a proof of lost resynchronization. Note also the change in QRS morphology.

The patient successfully underwent an upgrade to CRT-D system (Medtronic, InSync III) with the LV electrode placed in the postero-lateral vein, defibrillating electrode in the right ventricular apex and the previously implanted RAA lead. After implantation

the device was programmed to DDD-R mode with basic rate 60 bpm and the sensed atrio-ventricular delay (AVD) was optimized under echocardiography during 68 bpm VAT pacing. The longest DFT_{LV} (450 ms) without the A-wave truncation was provided by AVD

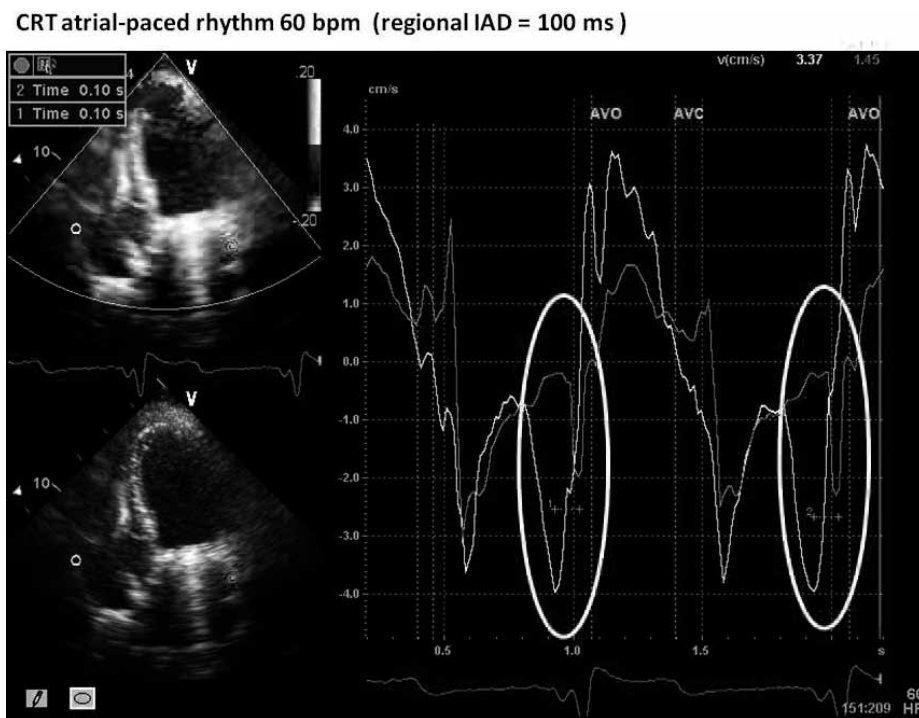


Figure 3. Regional IAD (during CRT atrial-paced rhythm 60 bpm and the AVD paced of 130 ms), defined as the time difference between the peak of tissue Doppler atrial A'-wave in the right and left atrial lateral wall, was determined of 100 ms

sensed of 100 ms (Figure 1b) [3]. The 30 ms nominal value of the sensed-paced AVD offset was programmed. Pre-excitation of the right ventricle with 10 ms provided the highest aortic velocity time interval. The degree of mechanical dyssynchrony diminished: no "septal flash" phenomenon was present, septo-lateral difference in peak S'-wave was 50 ms, the IVMD was 30 ms. After the CRT-D upgrade the patient improved (NYHA class II), LV ejection fraction increased to 32%, however the patient continued with frequent episodes of paroxysmal or persistent AF despite different pharmacological strategies, and underwent three electrical cardioversions. Frequent episodes of atrial tachyarrhythmia and atrial stunning after cardioversion precluded renewed estimation of the AVD setting under echocardiography. However, one year after the upgrade the patient was free from AF during echocardiography. It revealed the increase of the LV ejection fraction to 40%, and the right ventricular systolic pressure normalization. During the examination the 60 bpm CRT pacing was recorded with AVD-paced of 130 ms. As the mitral inflow was monophasic the AVD-paced was progressi-

vely increased until 200 ms, when a tiny mitral A-wave appeared (Figure 2a). Unfortunately, with AVD-paced of 200 ms the biventricular capture was lost: QRS morphology changed and the IVMD increased from 14 to 65 ms (Figure 2b). In order to reveal the mechanism of the monophasic mitral inflow during CRT pacing, the global and regional IAD was determined. The global IAD defined as the time difference between the beginning of the tricuspid and mitral A-wave was prolonged to 200 ms. It was calculated during 60 bpm atrial-paced CRT with AVD of 200 ms (with subsequent loss of biventricular capture). It was impossible to determine the global IAD during CRT under the atrial-paced rhythm, as no mitral A-wave was present (Figure 2a left). An attempt was made to measure the regional IAD, defined as the time difference between the peak of the tissue Doppler atrial A'-wave between the right and left atrial lateral wall (Figure 3). During CRT with atrial-paced rhythm of 60 bpm (AVD-paced of 130 ms) the late mitral A'-wave was detected and regional IAD of 100 ms calculated. The AVD was programmed short (130/100 ms), as biventricular capture

was our basic aim – it increased LV ejection fraction by 12% in 1 year. After several months the patient was still in NYHA class II heart failure, however developed permanent AF.

Discussion

There is no doubt that CRT has revolutionized the treatment of patients with heart failure and QRS prolongation. However, one-third of the patients do not derive clinical benefit from CRT implantation, or can even deteriorate [1]. Furthermore, in those who appear to respond it is not known whether or not they experience maximal benefit from CRT. Optimal lead placement and device settings are important factors which can maximize the response to the therapy. Up till now the main emphasis is put on the LV electrode position, and only occasionally on atrial function. However, there are reports on deleterious effects of RAA pacing, which can aggravate interatrial conduction abnormalities. *Adelstein et al.* showed that incidence of AF in CRT recipients increases twice when atrial-paced instead of atrial-sensed mode dominates [4]. In this study in all patients the atrial lead was placed in the RAA.

Our patient had several factors predisposing to atrial tachyarrhythmias, however in our opinion after upgrading to CRT the major risk factor for AF was RAA pacing, which induced important IAD with the truncation of the mitral A-wave. Under the CRT therapy the patient's LV ejection fraction increased from 28 to 40%, the right ventricular pressure normalized, however, the frequency of AF episodes increased

despite of pharmacological treatment modification. The RAA-paced rhythm resulted in the significant IAD prolongation, and it was impossible to program the optimal paced AVD, as the increase of AVD-paced precluded biventricular capture. The nominal paced-sensed AVD offset in most pulse generators is 30 ms. Previous studies [5] have underlined the possibility that a greater offset (60-70 ms) could be hemodynamically beneficial in CRT recipients, but it varies widely among patients. Atrial lead placement in the Bachmann's bundle region [6] could have diminished the IAD and enabled hemodynamically efficient programming of AVD-paced, however our patient has refused the reoperation. In our opinion, in CRT recipients, and especially in those with chronotropic incompetence and high probability that atrial-paced rhythm will dominate after implantation, special care should be focused to the atrial lead position. The RAA can induce or aggravate the IAD and preclude the optimal AVD programming. Implantation of the right atrial lead in the Bachmann's bundle region should be considered in these patients to obviate this problem.

The authors declare no conflict of interests

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