JACC: CASE REPORTS VOL. 3, NO. 1, 2021

MINI-FOCUS ISSUE: ELECTROPHYSIOLOGY

ADVANCED

CASE REPORT: CLINICAL CASE

Atrioventricular Node Ablation to Enhance Resynchronization Therapy Response in a Patient With Complete Atrioventricular Block

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ABSTRACT

Up to one-third of patients who undergo cardiac resynchronization therapy do not obtain clinical benefit. A systematic approach can identify treatable causes in many nonresponding patients. We present a case of nonresponse to cardiac resynchronization therapy that resolved by ablation of the atrioventricular node in a patient with complete atrioventricular block. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:150–5)

A 70-year-old man was referred to the cardiac device clinic for lack of response to cardiac resynchronization therapy (CRT).

PAST MEDICAL HISTORY

Four years before presentation, the patient had a dual chamber pacemaker implanted for complete atrioventricular (AV) block. One year later, he had an anterior wall myocardial infarction. Despite percutaneous revascularization, dilated cardiomyopathy developed, with an ejection fraction of 34% and New York Heart Association functional class II heart failure symptoms on optimal medical therapy. Upgrade to a CRT defibrillator system was performed, along with extraction of the ventricular pacemaker lead.

One year after the procedure, the patient's clinical status had not improved. He was referred for assessment and optimization of device function.

LEARNING OBJECTIVES

- To highlight the importance of a stepwise assessment of patients who do not respond to cardiac resynchronization therapy.
- To discuss available management options for patients with device-related rhythm abnormalities.

DIFFERENTIAL DIAGNOSIS

Clinical response to CRT depends on multiple factors, starting from patient selection to implant issues and appropriate programming. Common causes of nonresponse at short-term follow-up include loss of left ventricular capture secondary to lead dislodgment, low percentage of biventricular pacing, and inadequate AV or interventricular timing.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received August 13, 2020; revised manuscript received October 14, 2020, accepted November 2, 2020.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2020.11.009

INVESTIGATIONS

The 12-lead electrocardiogram showed a regular atrial rhythm at 80 beats/min, with a low-amplitude P-wave, and biventricular pacing with a paced QRS complex duration of 150 ms (Figure 1). Lead parameters were normal. The chest radiograph showed adequate lead position, with the left ventricular lead in a posterolateral vein (Figure 1). Echocardiography showed an unchanged left ventricular ejection fraction (36%) and an abnormal transmitral filling pattern with fusion of the E and A waves.

The programmed AV interval was 150 ms. Attempts to improve left ventricular filling were made by shortening the AV interval. At different values of AV interval, the filling pattern remained unchanged, but a change in heart rate was noted, with faster heart rate at shorter AV intervals and vice versa (Figures 2A to 2C).

This finding raised the suspicion that the atrial activity was not sinus rhythm, but rather retrograde activation from the paced QRS complex, so that the ventriculoatrial interval

ABBREVIATIONS AND ACRONYMS

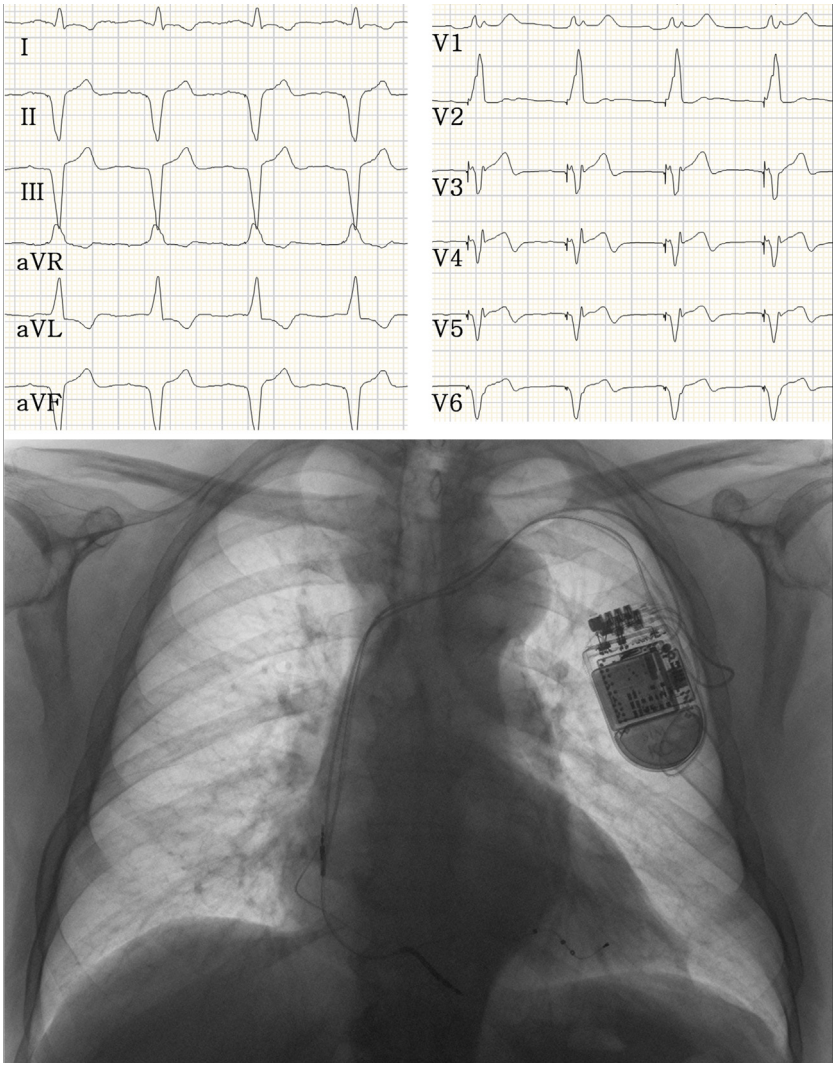
AV = atrioventricular

CRT = cardiac resynchronization therapy

PMT = pacemaker-mediated tachycardia

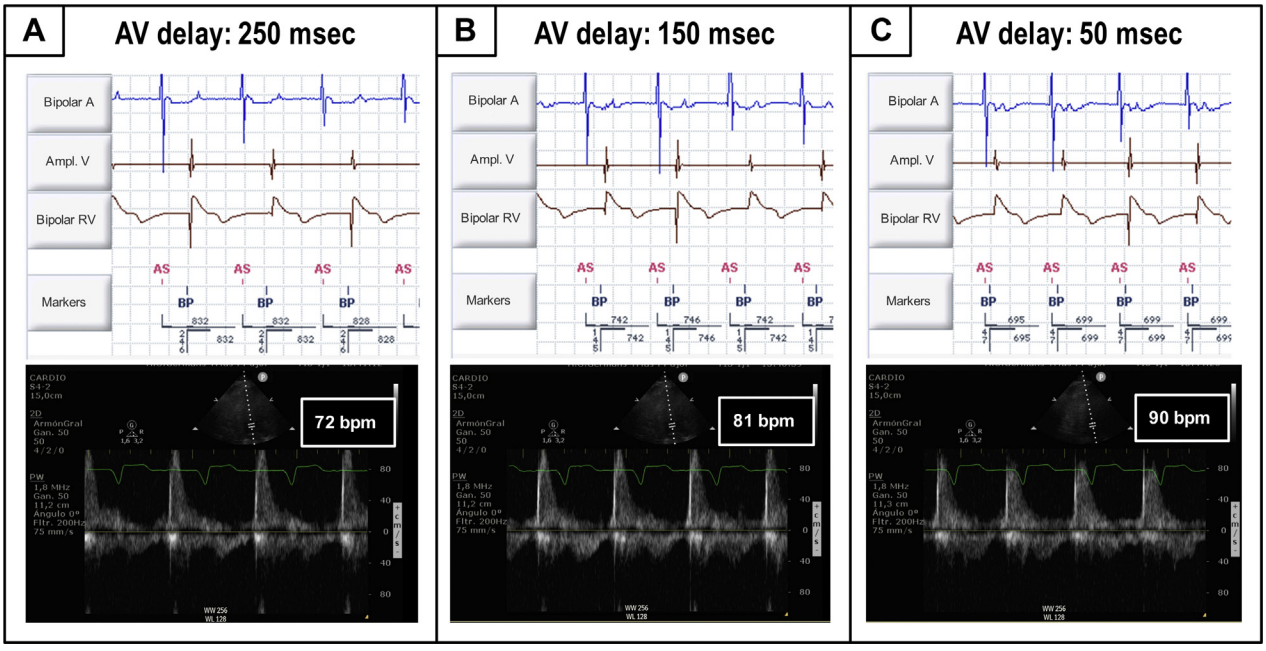
PVARP = post-ventricular atrial refractory period

FIGURE 1 Findings on Presentation



(Top) The 12-lead electrocardiogram on presentation shows an atrial rhythm of 80 beats/min, with a low-amplitude P-wave, followed by biventricular pacing. **(Bottom)** The chest radiograph shows adequate lead positions.

FIGURE 2 **Intracardiac Electrograms, Device Markers, and Transmitral Filling Patterns on Pulsed-Doppler Echocardiography at Different Programmed AV Intervals**



(A–C) Atrial sensing and biventricular pacing are shown. A monophasic filling wave (fusion of the E and A waves) is present. Shortening of the atrioventricular (AV) interval does not result in separation of the E and A waves, but an increase in heart rate is observed. A = atrium; Ampl. = amplitude; AS = atrial sensing; BP = biventricular pacing; RV = right ventricle; V = ventricle.

remained constant. This would explain the constant E/A-wave relationship and the higher heart rates at shorter AV intervals. During device interrogation, baseline complete antegrade AV block was documented, whereas ventricular pacing in the VVI mode showed retrograde conduction with a very long ventriculoatrial interval. The patient was in slow, incessant, pacemaker-mediated "tachycardia" with the AV node as the retrograde limb. The unusually low heart rate and the low P-wave amplitude in the surface electrocardiogram made it possible for the condition to be mistaken for sinus rhythm.

MANAGEMENT

Because the retrograde conduction time was 600 ms, which exceeded the maximum programmable postventricular atrial refractory period (PVARP), it was not possible to manage this case by means of device reprogramming. AV node catheter ablation was considered the only option to avoid pacemaker-mediated re-entry. During continuous ventricular pacing (VVI mode), the earliest retrograde activation was mapped in the compact AV node area, and radiofrequency delivery at this site blocked retrograde conduction with resumption of normal sinus rhythm (**Figures 3A and 3B**).

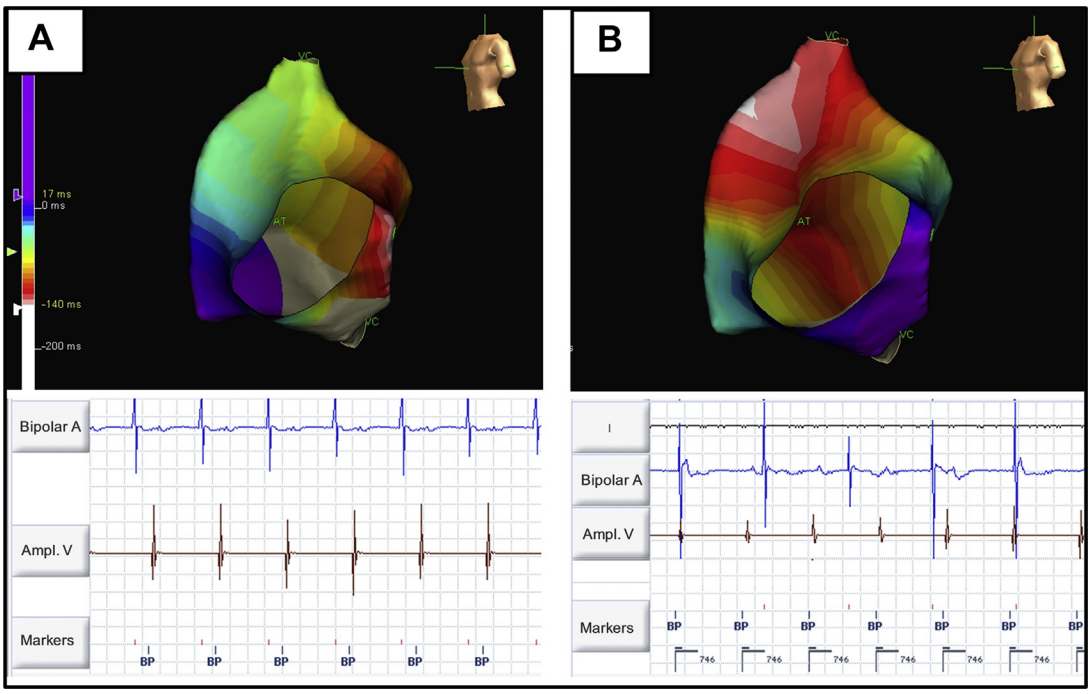
After AV node ablation, it became possible to adjust the AV interval so that an optimal left ventricular filling pattern with separation of the E and A waves could be obtained (**Figure 4**).

DISCUSSION

CRT is a well-established therapy for patients with heart failure and electrical dyssynchrony induced by left bundle branch block or right ventricular pacing. However, about one-third of patients do not benefit from CRT. Multiple causes of CRT nonresponse exist, from inadequate patient selection to issues related to implant technique and device programming.

Suboptimal AV or interventricular synchrony is among the most common causes of nonresponse to CRT (1). Although routine echocardiographic optimization has not been shown to be superior to empirical or algorithm-based optimization, a trial of echocardiographic optimization is warranted in nonresponding patients. Adequate AV synchronization optimizes the left atrial contribution to left

FIGURE 3 **Electroanatomic Mapping**



(A) Electroanatomic map of right atrial activation during VVI pacing. Constant retrograde conduction is seen with a long ventriculoatrial (VA) interval, with earliest activation at the anteroseptal tricuspid annulus (atrioventricular [AV] node region). After atrioventricular node ablation, there is **(B)** ventriculoatrial dissociation, and the atrial activation is consistent with sinus rhythm (earliest activation at the superior vena cava–right atrial junction). Abbreviations as in Figure 2.

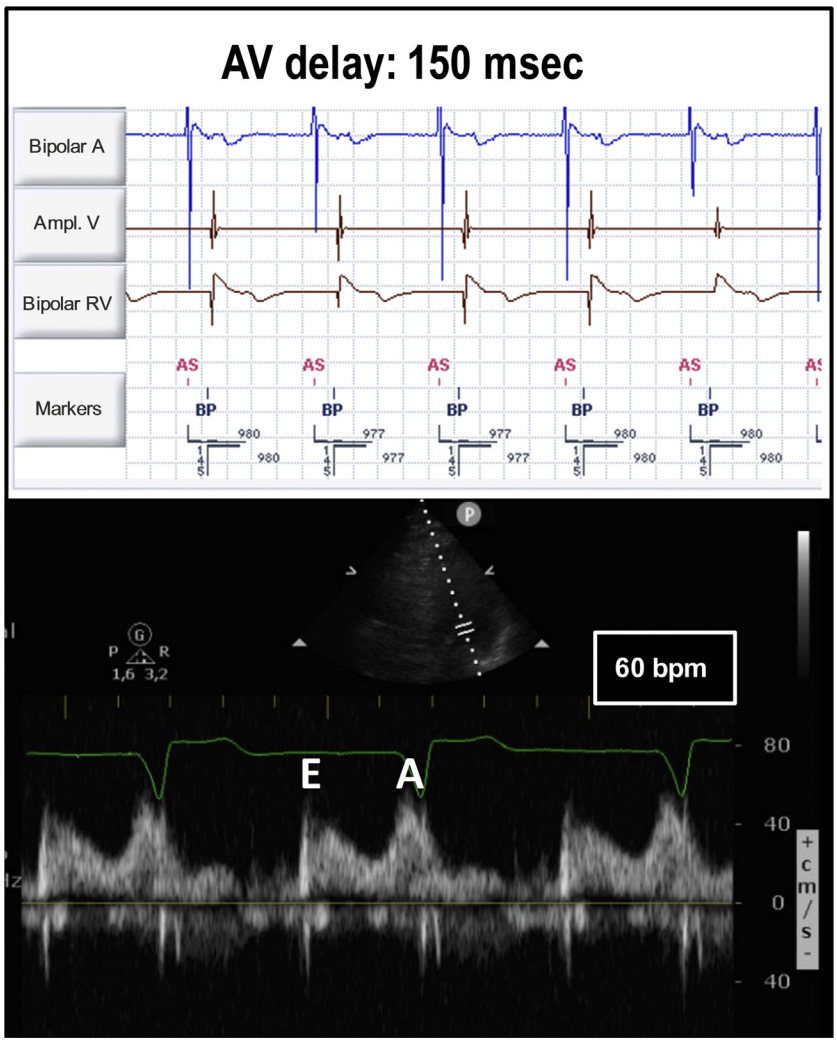
ventricular filling. A prolonged AV delay produces fusion of the E and A waves because the atrial contraction occurs during the early diastolic filling phase. This shortens left ventricular filling time, which reduces cardiac output and can provoke diastolic mitral regurgitation. Whenever fusion of the E and A waves is encountered, the AV delay should be shortened to advance the E-wave and separate it from the A-wave of the following heartbeat. Excessive AV delay shortening must also be avoided to prevent ventricular contraction before the completion of atrial emptying, thus resulting in a truncated A-wave.

In this case, AV interval shortening did not cause separation of the E and A waves, but rather an increase in the heart rate. This finding suggested that the atrial activation was "linked" to the ventricular activation. Continuous retrograde conduction through the AV node (despite baseline complete antegrade AV block) resulted in a permanent form of pacemaker-mediated tachycardia (PMT) that was difficult to recognize. Typical PMT can be recognized by a sequence of atrial sensing: ventricular pacing at or close to the device upper tracking rate, with a negative P-wave indicating retrograde atrial activation. In our case, the P-wave was of low amplitude, and retrograde conduction was so slow that the resulting heart rate was mistaken for normal sinus rhythm.

PMT can usually be managed by programming algorithms to prevent initiation or perpetuation of the tachycardia (2). Prolonging the PVARP avoids PMT by making the retrograde atrial activation fall into the pacemaker refractory period so that it is not tracked. However, a long PVARP limits the maximum tracking rate, which can be deleterious in active patients. In addition, very slow retrograde conduction can exceed the maximum programmable PVARP. In our case, retrograde conduction time was approximately 600 ms, and the maximum programmable PVARP was 500 ms. Modern devices have algorithms to identify and terminate PMT, but in the case of very slow retrograde conduction, the device cannot identify the rhythm as PMT.

AV node ablation in patients with antegrade AV block has been reported in pacemaker recipients with symptomatic PMT in which device programming

FIGURE 4 AV Interval Programming



After atrioventricular (AV) node ablation, a physiologic atrioventricular interval could be programmed with an optimal transmitral filling pattern (separation of E and A waves). Abbreviations as in Figure 2.

options were considered inappropriate (3,4). To our knowledge, no previous case has been reported in which retrograde conduction did not cause clinical tachycardias, but instead a lack of response to CRT secondary to impaired AV synchrony, which could be successfully managed by AV node ablation. It is possible that this is an underrecognized cause of CRT nonresponse.

FOLLOW-UP

The patient's clinical status improved almost immediately, with increased exercise tolerance. Follow-up echocardiography 4 months after the procedure showed reverse remodeling with a decrease in left ventricular end-diastolic and end-systolic diameters and a significant increase in left ventricular ejection fraction (from 36% to 52%).

CONCLUSIONS

In patients with a lack of response to CRT, identification of treatable causes is required. AV dyssynchrony is common and can usually be solved by echocardiographic or algorithm-based optimization. A systematic approach can help to identify unusual causes of poor response to CRT, which may require invasive management.

AUTHOR DISCLOSURES

This study did not receive any specific funding. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS ablation, cardiac resynchronization therapy, echocardiography