

Double P-wave phenomenon and new-onset heart failure in a patient with previous heart surgery, permanent pacemaker, and repeated catheter ablations for right atrial flutters: What is the cause?



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Introduction

The atriotomy scars after heart surgeries have been demonstrated as an anatomical substrate for arrhythmias.^{1,2} Catheter ablation of typical and atypical atrial flutters is frequently required in these patients owing to heart failure deterioration. However, not only arrhythmias but also conduction disturbances may occur after cardiac interventions, requiring the implantation of a pacemaker.³ In the present case, both catheter ablations and pacemaker implantation followed a myxoma excision. However, new-onset heart failure was presented with a confusing electrocardiogram (ECG), the interpretation of which was crucial for the explanation of the clinical deterioration and further evaluation.

Case report

A 74-year-old woman was referred to electrophysiology study and, potentially, catheter ablation for sustained palpitations. She had a history of an operated left atrial myxoma via right atrial atriotomy and septotomy followed by repeated catheter ablations of an isthmus-dependent flutter and an atypical flutter dependent to the atriotomy. Moreover, a dual-chamber pacemaker (Reply 200 DR W2; Sorin CRM SAS, Clamart, France) had been implanted for infra-Hisian conduction disease. Sensing failure of the atrial lead had been managed with implantation of a new lead with active fixation while the previous one was abandoned. Remarkably, the patient presented a new-onset heart failure and was recently hospitalized for pulmonary edema. Echocardiography revealed only a mildly hypertrophic left ventricle with preserved ejection fraction attributed to hypertension.

The surface ECG on admission demonstrated ventricular apical stimulation following a discreet P wave corresponding

KEY TEACHING POINTS

- A high prevalence of arrhythmias is observed after heart surgeries (as for excisions of myxomas), related to the anatomical substrate of the atriotomy scars, requiring catheter ablation.
- Surgical injury to the cardiac conduction system should also be considered in these patients, as conduction disturbances may occur after cardiac interventions, requiring the implantation of a pacemaker.
- Although pacemaker syndrome has mainly been described in VVIR-paced patients and sinus rhythm, it can also be presented with atrial-based pacing owing to interatrial conduction disturbances.

to the intrinsic atrial rhythm (red arrows in [Figure 1](#)). Moreover, a new-onset P wave of negative deflection in the inferior leads was superimposed to the T wave (blue arrows in [Figure 1A](#)).

Device interrogation revealed an atrial-based pacing (DDDR) with A-sensed V-paced rhythm in a 1:1 ratio. Furthermore, no atrial high rate episodes had been recorded. Cessation of stimulation led to an intrinsic non-sinus node atrial rhythm (red arrows in [Figure 1B](#)) conducted to the ventricles with prolonged PR interval and 1:1 atrioventricular association. However, rhythm detection via device interrogation revealed atrioventricular dissociation without any anterograde or retrograde conduction, further impeding interpretation ([Figure 1C](#)).

Discussion

How ECG on admission could be interpreted and how this discordance between surface ECG and device intracardiac recordings could be explained

In order to explain this discordance as well as the incomprehensible ECG on admission, an electrophysiology study was

KEYWORDS Pacemaker syndrome; Interatrial block; Conduction disturbances; Atrial flutter; Cardiac surgery
(Heart Rhythm Case Reports 2023;9:954–958)

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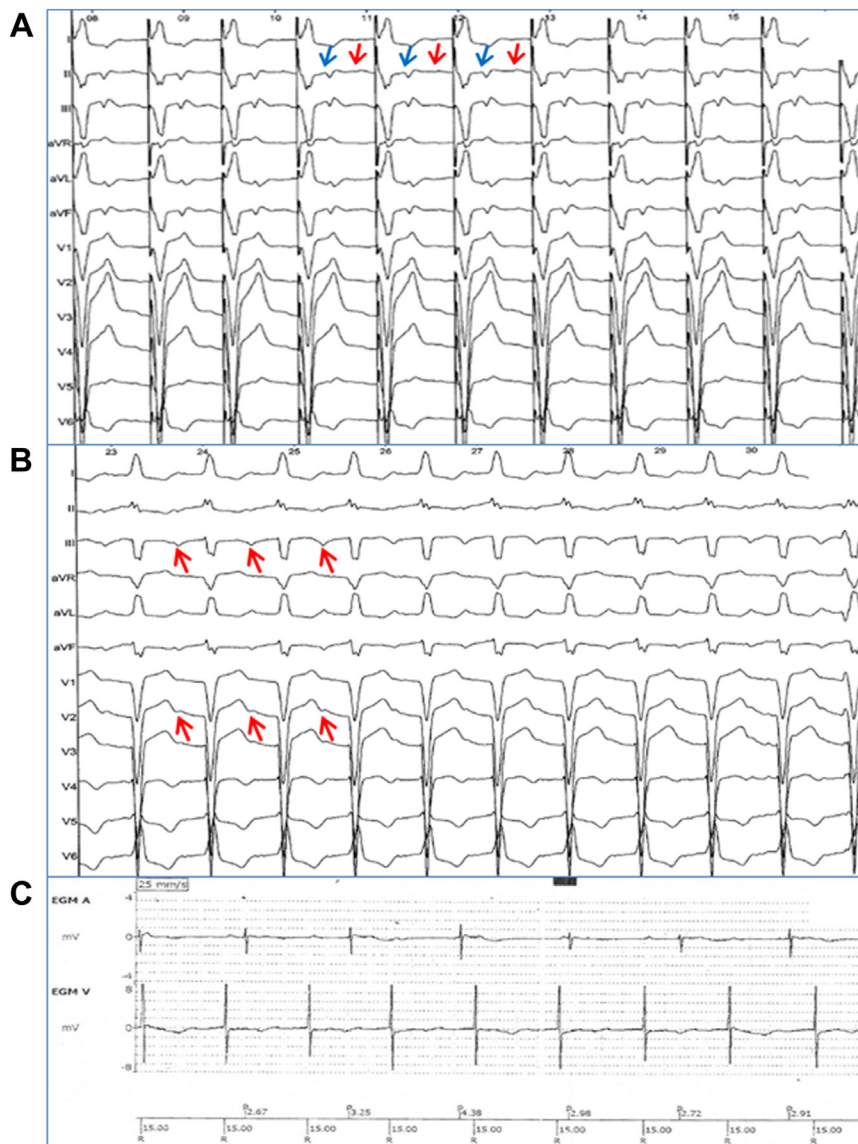


Figure 1 A: Electrocardiogram on admission. B: Intrinsic rhythm after cessation of stimulation. C: Rhythm detection via device interrogation.

performed using 2 quadripolar catheters (VIKINGTM diagnostic catheter; Boston Scientific, Marlborough, MA) positioned in the right atrium and in the coronary sinus (CS) and by programming the device at VVI mode.

Bidirectional interatrial conduction block was revealed and confirmed with pacing from the right atrium (RA), as the stimuli were not able to capture the atrium in the CS, and vice versa. Moreover, both anterograde and retrograde 1:1 atrioventricular conduction was observed between the left atrium (LA) and the ventricles (Figure 2A and 2B). Interestingly, programmed atrial stimulation induced a nonsustained right atrial tachycardia conducted neither to the LA nor to the ventricles (Figure 2B). Complete interatrial conduction block explained the discordance between surface ECG and intracardiac recordings.

Interatrial conduction block has mainly been described after atriotomies and/or complex catheter ablations, whereas it

is rare in unoperated patients.⁴⁻⁷ Digitalis intoxication and amiodarone administration have also been reported as possible causes.⁴ The sites of interatrial conduction in the normal atria are presumed to be along the Bachmann bundle, interatrial septum, and the musculature of the proximal CS.⁸ In our case, disruption and/or dissociation of the above-mentioned structures could be caused by the previous interventions.

Endocardial activation mapping of the right atrium, performed using the IntellaMap OrionTM mini-basket mapping catheter and the ultra-high-density RhythmiaTM mapping system (Boston Scientific, Marlborough, MA), demonstrated a complete dissociation between a large part of the RA and the interatrial septum and the LA (Figure 3). The dissociated part of the RA was including the free wall and the usual site of sinus node as well as the appendage where the atrial lead had been implanted. Importantly, His electrogram was recorded

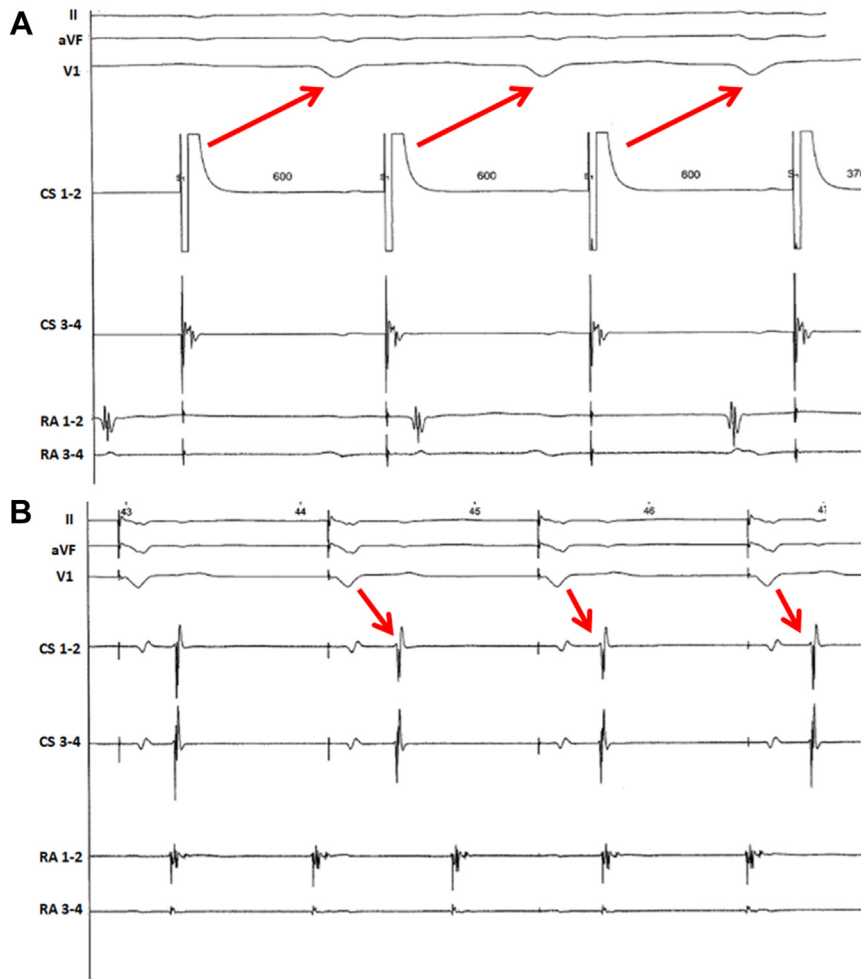


Figure 2 A: Stimulation from the distal bipole of the coronary sinus. B: Ventricular pacing during a dissociated right atrial tachycardia. CS = coronary sinus; RA = right atrium.

at a more septal location compared to the line of block, and conduction appeared to be interrupted before reaching the atrioventricular node, explaining atrioventricular dissociation between the RA and the ventricles. The latter, in addition to the complete interatrial block, had led to a complete electrical dissociation of the part of the RA that was containing the sinus node and the atrial pacing site. On the other hand, both anterograde and retrograde conduction between the LA and the ventricles were preserved.

Based on these findings, an explanation of ECG on admission was feasible. Spontaneous sinus rhythm followed by ventricular pacing was leading to retrograde left atrial conduction that was displaying as a retrograde P wave superimposed to the T wave.

How could we explain patient's symptoms?

Taking into consideration the preserved ejection fraction of the left ventricle, the fact that no arrhythmia events were detected, and the retrograde conduction to the LA during RV pacing, pacemaker syndrome could explain or contribute (along with hypertensive cardiomyopathy) in the new-onset

heart failure and pulmonary edema. A previous ECG revealed 1 biphasic p wave in lead V₁ containing both components of right and left atrial depolarization (Supplemental Figure 1). As ECG modification was followed by heart failure onset, pacemaker syndrome appeared to be the most relevant diagnosis. Severe pacemaker syndrome has been described to be developed in nearly 20% of VVIR-paced patients and sinus rhythm with congestive signs and symptoms associated with retrograde conduction.⁹ Pacemaker syndrome can also occur under certain circumstances with atrial-based pacing owing to inadequate timing of atrial and ventricular contraction, as in the presence of an abnormally long intra-atrial conduction time or if the AV delay is programmed either too long or too short.¹⁰ In our case, complete interatrial conduction block is firstly described as a cause of pacemaker syndrome despite DDD pacing. Right atrial sensing was followed by ventricular stimulation and subsequent retrograde activation of the left atrium. In this way, LA contraction was not contributing in the ventricular preload.

A high prevalence of arrhythmias has been reported after heart surgeries (as for congenital heart diseases and excisions

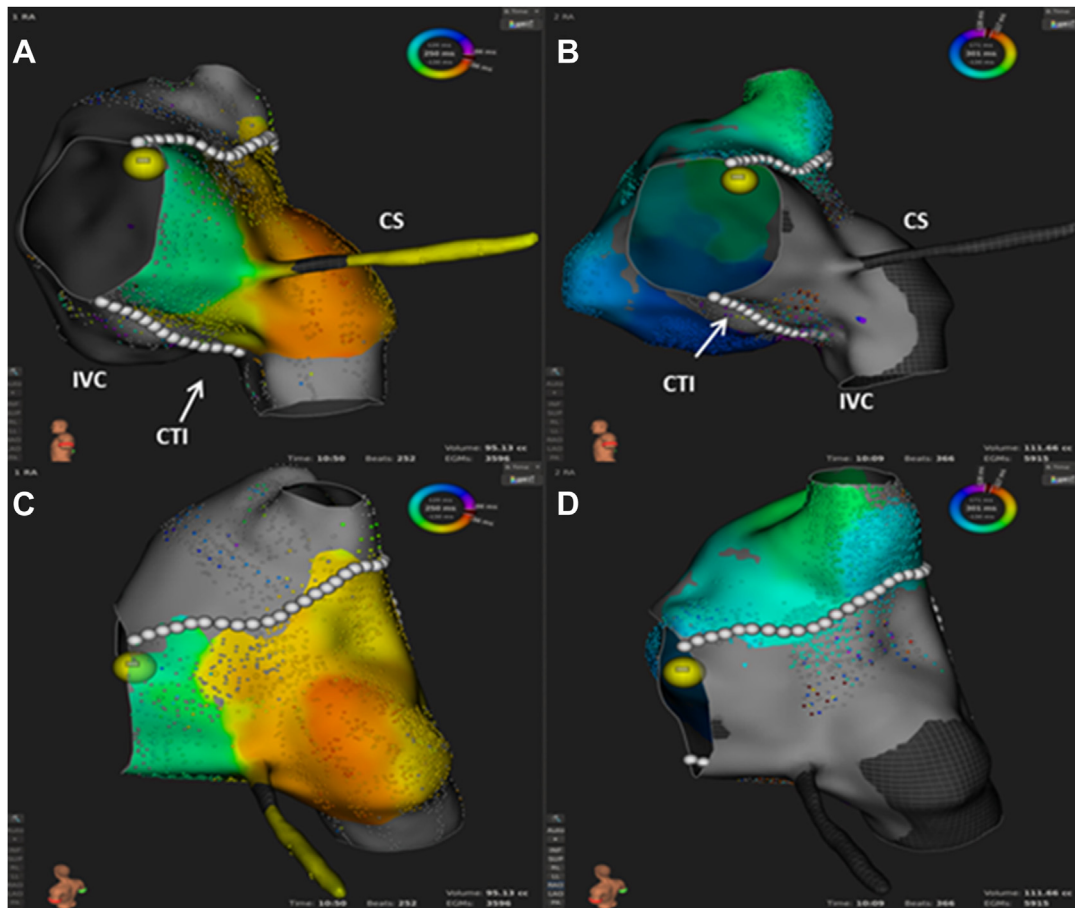


Figure 3 Endocardial activation mapping during pacing from the coronary sinus (A and C) and from the high right atrium (B and D). Yellow spot indicates His electrogram recording. CS = coronary sinus; CTI = cavotricuspid isthmus; IVC = inferior vena cava.

of myxomas), related to the anatomical substrate of the atriotomy scars.^{1,2} In the present case, a cavotricuspid isthmus-dependent and an atypical right flutter dependent to the atriotomy were previously treated with catheter ablation. However, surgical injury to the conduction pathways should also be considered in these patients. Notably, device interrogation did not reveal any recorded arrhythmias.

How could we manage this patient?

In order to block the retrograde activation of the LA, ablation of the atrioventricular node was decided, followed by programming the device at DDDR mode while LA remained electrically active, although isolated. This approach led to improvement of heart failure symptoms and palpitations. On the other hand, a potential disadvantage of the subsequent isolation of the LA could be an increase in thromboembolic risk. It is known that electrical isolation of the left atrial appendage can harbor significant thromboembolic risks.¹¹ However, whether total LA isolation has a similar effect remains unclear, especially when isolation does not result in an electrically silent LA, as in our case.¹²

Atrial lead placement at the lower atrial septum (near the CS ostium) and dual-site atrial pacing with the help of a bifur-

cated Y-connector could also be a potential strategy, although this approach has rarely been described.^{13,14} Batrial pacing has also been demonstrated to be feasible using specifically designed CS leads for left atrial pacing.¹⁵ However, a right atrial lead had already been abandoned in this patient, and an additional intervention was discouraged. Nevertheless, the above-mentioned techniques could be a supplementary approach in case of sustained pacemaker syndrome owing to dissociated LA contraction. Finally, no intervention and programming the device to VVI mode with a slow baseline rate could allow the intrinsic rhythm to be present. However, sustained bradycardia or a pseudo-pacemaker syndrome owing to the delayed atrioventricular conduction were possible and could lead to an uncertain outcome.

Funding Sources: None.

Disclosures: None.

Appendix Supplementary Data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2023.09.008>.

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