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CASE REPORT
Arrhythmias/electrophysiology

Dual atrial rhythms: a case report of an unusual cause of pacemaker syndrome

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Background Atrial dissociation (AD) is described as the existence of two simultaneous electrically isolated atrial rhythms. Theoretically, detection of dual atrial rhythms with a sufficiently high rate by pacemaker can lead to automatic mode switching and associated pacemaker syndrome. Such a clinical observation has not been reported before in the literature.

Case summary An 87-year-old female with Ebstein's anomaly status post-tricuspid valve annuloplasty and tricuspid valve replacement and a dual-chamber pacemaker presented with congestive heart failure 1 week after undergoing atrial lead revision. Interrogation of her dual-chamber pacemaker revealed two atrial rhythms: sinus or atrial-paced rhythm and electrically isolated atrial tachycardia (AT). Sensing of both atrial rhythms by the pacemaker led to automatic mode switching, which manifested as ventricular paced rhythm with retrograde P waves on electrocardiogram. Adjusting the atrial lead sensitivity to a level higher than the sensing amplitude of AT restored atrial paced and ventricular sensed rhythm, which resulted in resolution of heart failure symptoms.

Discussion Regardless of the cause of AD, there must be electrical insulation between the two rhythms for their independent coexistence in the atria. Atrial dissociation can lead to pacemaker syndrome from automatic mode switching. If the sensing amplitude during sinus rhythm is significantly larger than that of AT, adjusting the atrial lead sensitivity would solve the issue, as in the present case. Otherwise, atrial lead revision, pharmacotherapy, or AT ablation should be considered.

Keywords Atrial dissociation • Dual atrial rhythms • Atrial tachycardia • Pacemaker syndrome • Case report

ESC Curriculum 5.5 Supraventricular tachycardia • 5.9 Pacemakers • 6.1 Symptoms and signs of heart failure

Learning points

- Two electrically separated rhythms can simultaneously coexist in the atria, which is termed as atrial dissociation or dual atrial rhythms.
- Dual atrial rhythms with a combined rate above the preset mode switch rate can lead to automatic mode switch and associated pacemaker syndrome. Adjustment of atrial lead sensitivity to 'ignore' atrial tachycardia is the most expeditious mean to avoid mode switch in the presence of dual atrial rhythms.

Introduction

Atrial dissociation (AD) is defined as the existence of two simultaneous electrically isolated atrial rhythms. Atrial dissociation is an uncommon condition that has been reported in patients post-heart transplant and in various other cardiac surgical procedures.[1,2] Atrial dissociation could lead to clinical consequences such as symptomatic atrioventricular (AV) dissociation or pseudo ventricular tachycardia (VT).[1] We report the first case of AD leading to pacemaker syndrome and congestive heart failure (CHF) from automatic mode switching.

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Timeline

4 years prior	Single-chamber atrial pacemaker implant for sinus node dysfunction.
3 years prior	Tricuspid valve replacement. Addition of a right ventricular epicardial lead. Decreased atrial lead sensing and increased pacing threshold were noted during subsequent outpatient follow-up.
5 weeks prior	Complete loss of sensing and capture of right atrial lead were noted.
2 weeks prior	Patient underwent atrial lead revision.
1 week prior	Increased dyspnoea and fatigue started.
Index hospital visit	Dual atrial rhythms leading to 'inappropriate' automatic mode switching were identified. After atrial lead sensitivity was adjusted, atrial-paced rhythm was restored.
Day 1	Heart failure symptoms resolved.
One month after discharge	The patient remained asymptomatic with atrial-paced rhythm.

Case presentation

An 87-year-old female presented with chief complaints of dyspnoea and fatigue for 1 week. Two weeks prior, the patient underwent atrial lead revision of her dual-chamber pacemaker due to atrial lead failure. The new lead was placed in the right atrial appendage and the old atrial lead was abandoned. The pacemaker was programmed to DDDR mode with automatic mode switching to VDIR on detection of an atrial arrhythmia with a rate above 170 b.p.m. An electrocardiogram (ECG) before discharge revealed atrial paced and ventricular sensed rhythm.

The patient has a past medical history of Ebstein's anomaly complicated by severe tricuspid regurgitation status post-tricuspid valve annuloplasty 13 years prior, paroxysmal atrial tachycardia (AT), atypical atrial flutter, and sinus node dysfunction (SND) requiring the implantation of a single-chamber atrial pacemaker. Three years prior, the patient underwent redo tricuspid valve repair and replacement with an addition of a right ventricular (RV) epicardial lead and upgrade to a dual-chamber pacemaker. In-office device interrogation after the surgery revealed significantly decreased sensed atrial activity and elevated atrial lead thresholds. Five weeks prior to the index emergency department visit, the patient had increased weakness and dyspnoea. Interrogation of the pacemaker showed loss of sensing and failure to capture of the right atrial lead. The patient underwent atrial lead revision as mentioned above. Her outpatient medications included nebivolol 5 mg daily, amiodarone 100 mg daily, and warfarin. Amiodarone had been on hold for ~2 months prior to the presentation due to concern for possible contribution to progressive weakness.

Physical examination revealed an elderly woman in no acute distress. Blood pressure was 128/78 mmHg, heart rate was regular at 70 beats per minute (b.p.m.), respiratory rate was 16 per minute, and temperature was 98.2°F. Pulse oximeter showed 95% oxygen saturation. Grade 3/6 pan-systolic murmur at left parasternal border was noted. Neck veins were not distended. Fine crackles on both lung bases were noted. There was no lower extremity oedema. Complete blood count and basic metabolic panel were unremarkable. B-type natriuretic peptide was 925 pg/mL (normal: ≤100 pg/mL). Troponin I was <0.03 ng/mL (normal < 0.05 ng/mL). Chest X-ray showed bilateral lower lung interstitial infiltrates. Electrocardiogram showed ventricular paced rhythm at 70 b.p.m. with retrograde P waves (*Figure 1*).

The chief differential diagnosis was CHF. Due to recent pacemaker lead revision and upgrade, complications related to the procedure such as pericardial effusion, pacemaker malfunction, and pacemaker syndrome were considered.

Transthoracic echocardiogram was unchanged from prior and was significant for left ventricular ejection fraction (LVEF) of 65%, dilated right ventricle with decreased systolic function, and status post-bioprosthetic tricuspid valve replacement with moderate tricuspid regurgitation. There was no significant pericardial effusion.

Pacemaker malfunction was suspected due to unexpected ventricular paced rhythm with visible retrograde P waves on ECG. In the absence of obvious atrial arrhythmia, one would expect to see atrial or AV sequential pacing. The pacemaker was now apparently functioning in either VOO or VVI mode.

Pacemaker interrogation was performed. Intracardiac electrograms (EGMs) revealed two independent simultaneous atrial rhythms and ventricular paced rhythm (*Figure 2*). The first atrial rhythm has a relatively larger amplitude of ~1 mV at a cycle length of 855 milliseconds (ms) and coincides with the retrograde P waves seen on surface ECG (double-ended arrows). The other atrial rhythm has a smaller amplitude of ~0.3 mV at a shorter cycle length of 318 ms, likely representing AT (small arrows). Atrial tachycardia is electrically isolated with conduction exit block as it does not influence the cycle lengths of the retrograde atrial rhythm, nor does it have any association with the ventricular activity in this patient with no history of AV conduction block. The pacemaker was able to sense both atrial rhythms (marked as 'AS' on EGMs) with the combined rate above the preset mode switch rate of 170 b.p.m., leading to automatic mode switch.

Pacemaker was temporally programmed to AAI at 30 b.p.m. to check for sinus rhythm and intrinsic AV conduction. The atrial lead sensitivity was decreased from 0.25 mV to 0.5 mV. Sinus bradycardia with anterograde AV conduction was noted on EGMs (*Figure 3*). Atrial tachycardia exhibited no influence on sinus rhythm or ventricular activities. Atrial tachycardia was no longer recognized by the device after sensitivity change. On the surface ECG, sinus bradycardia with intermittent atrial pacing coexisted with AT (*Figure 4A*, amplitude 20 mm/mV). Atrial tachycardia activity can be seen most clearly in lead V3 (*Figure 4B*, arrows) with the cycle length identical to that seen on the intracardiac EGMs.

Because the amplitude of AT was relatively small, atrial lead sensitivity was decreased to ignore AT and DDDR mode was restored (*Figure 5A*). The patient's symptoms subsided shortly afterwards. Amiodarone was resumed. Upon outpatient follow-up, she remained asymptomatic and AT was no longer present (*Figure 5B*).

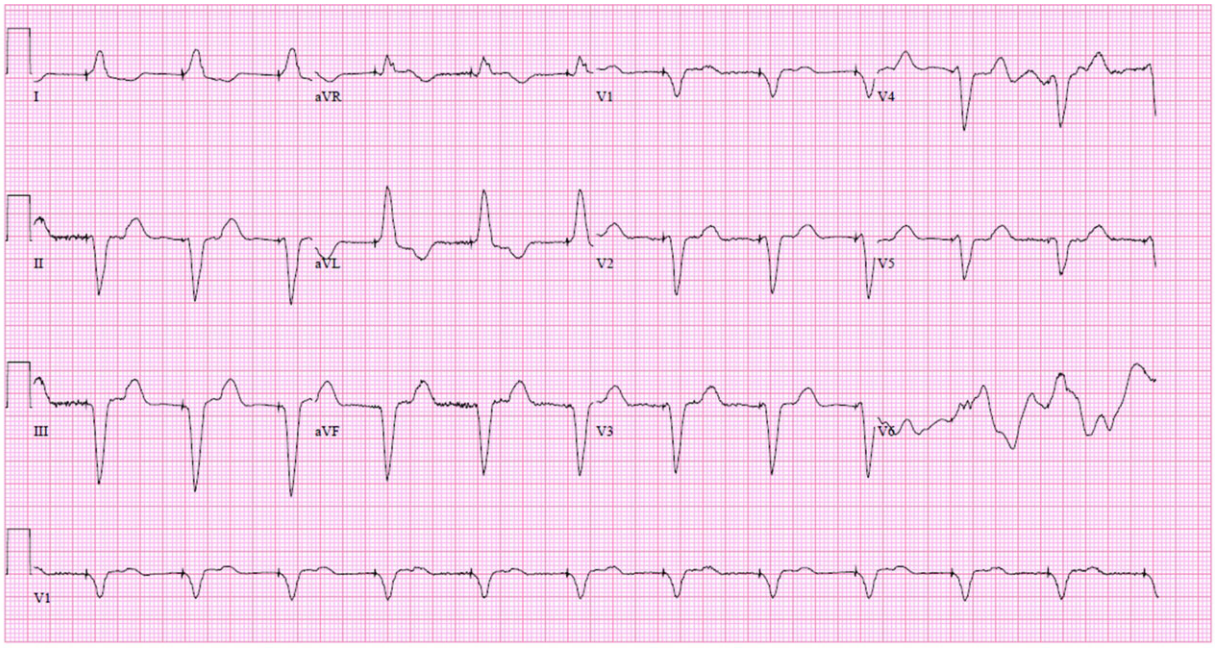


Figure 1 The presenting electrocardiogram showed ventricular paced rhythm at 70 b.p.m. with retrograde P waves most clearly visible in the inferior leads.

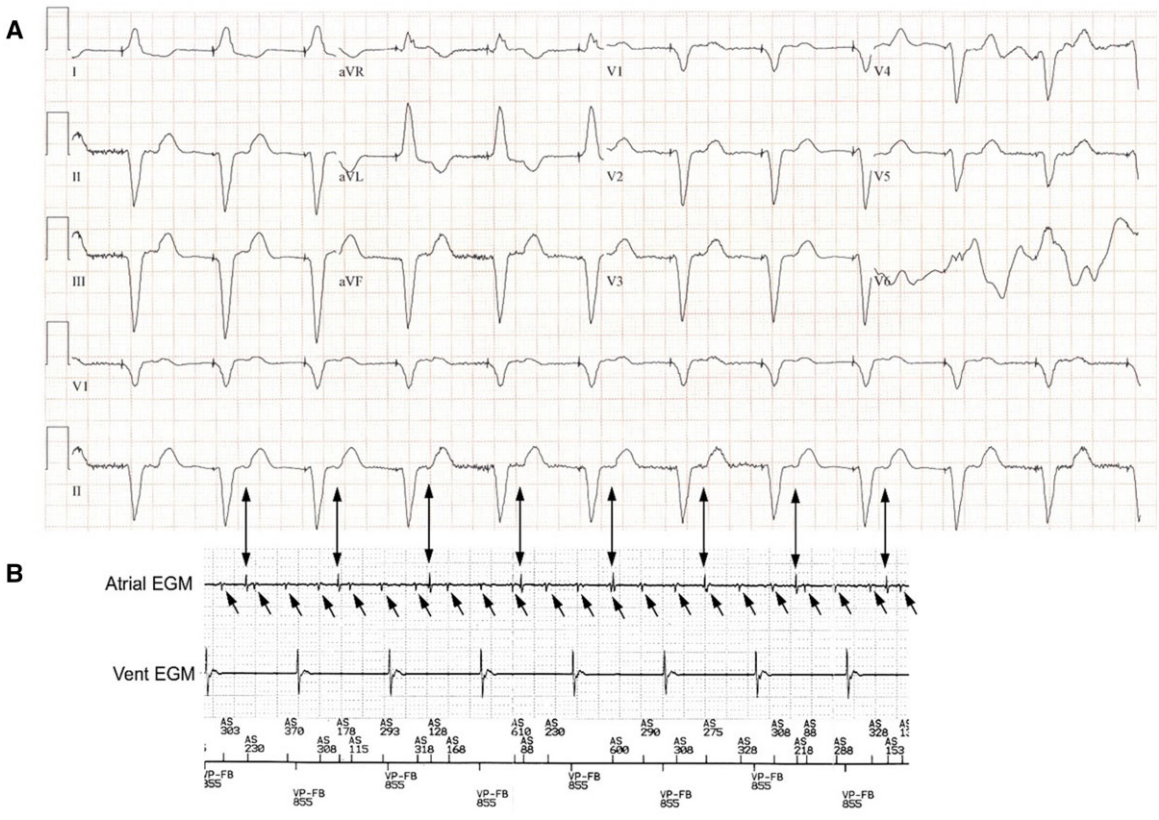


Figure 2 (*A*) Presenting electrocardiogram. (*B*) Atrial and ventricular electrograms from pacemaker interrogation revealing two independent simultaneous atrial rhythms and ventricular paced rhythm. Note retrograde P waves (double-ended arrows) and atrial tachycardia (small arrows). Atrial electrogram: 5 mm/mV; ventricular electrogram: 1 mm/mV.

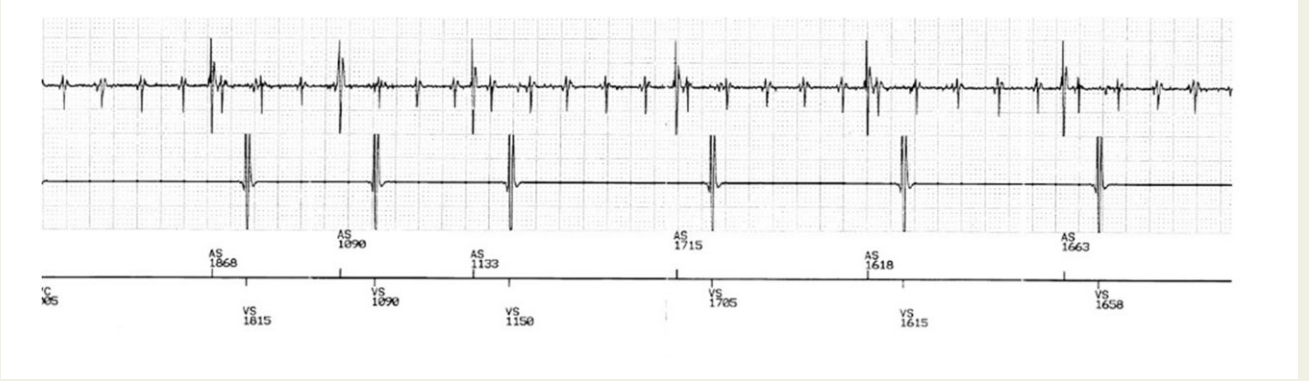


Figure 3 Atrial and ventricular electrograms showed sinus bradycardia with atrial premature depolarization (marked as 'AS') with anterograde atrioventricular conduction. Atrial tachycardia is electrically isolated and not recognized by the device. Atrial electrogram: 20 mm/mV; ventricular electrogram: 1 mm/mV.

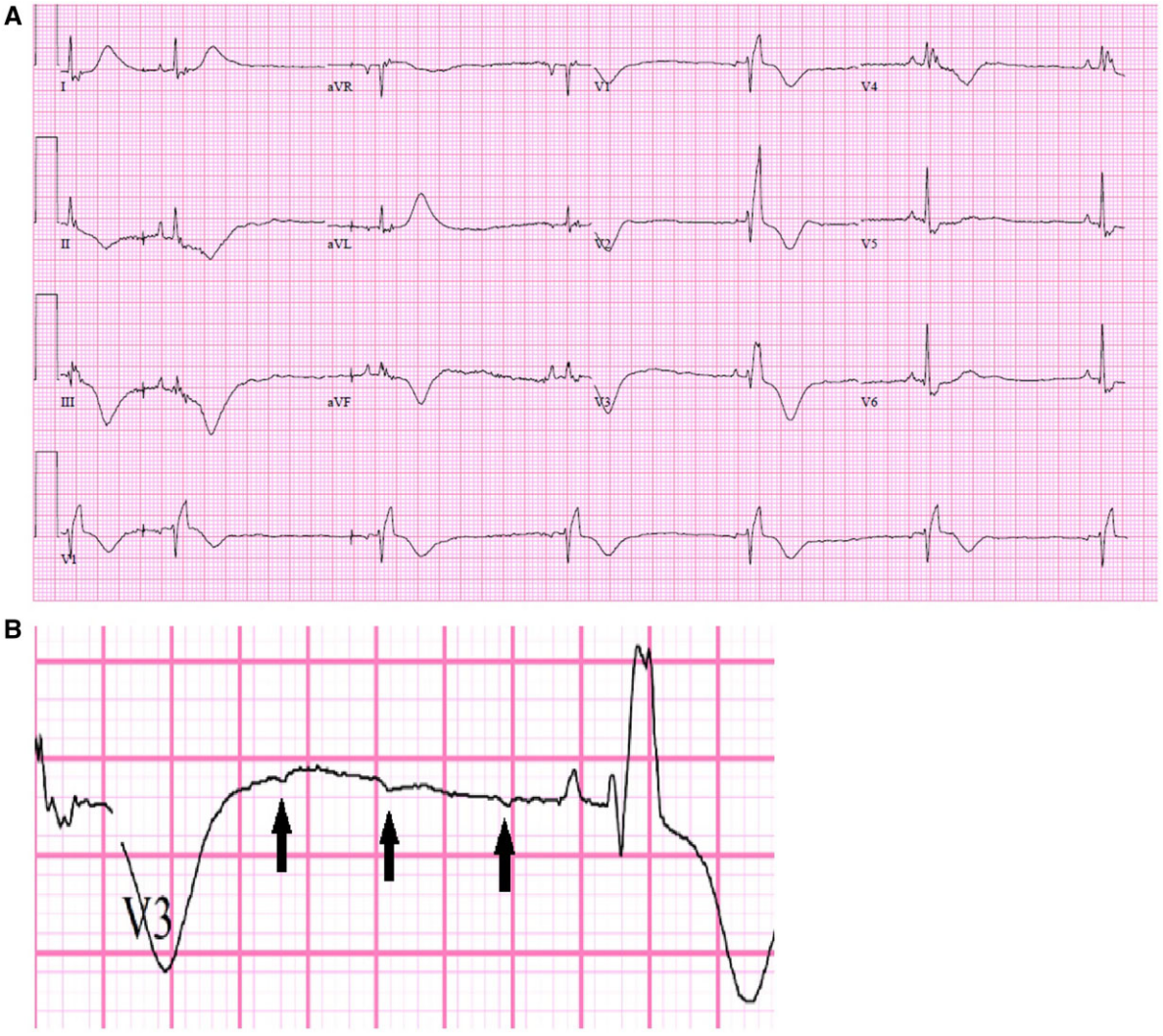


Figure 4 (*A*) Surface electrocardiogram after atrial lead sensitivity adjustment after the pacemaker was temporarily programmed to AAI at 30 b.p.m. (*B*) Magnified lead V3 from *A* showing AT activity.

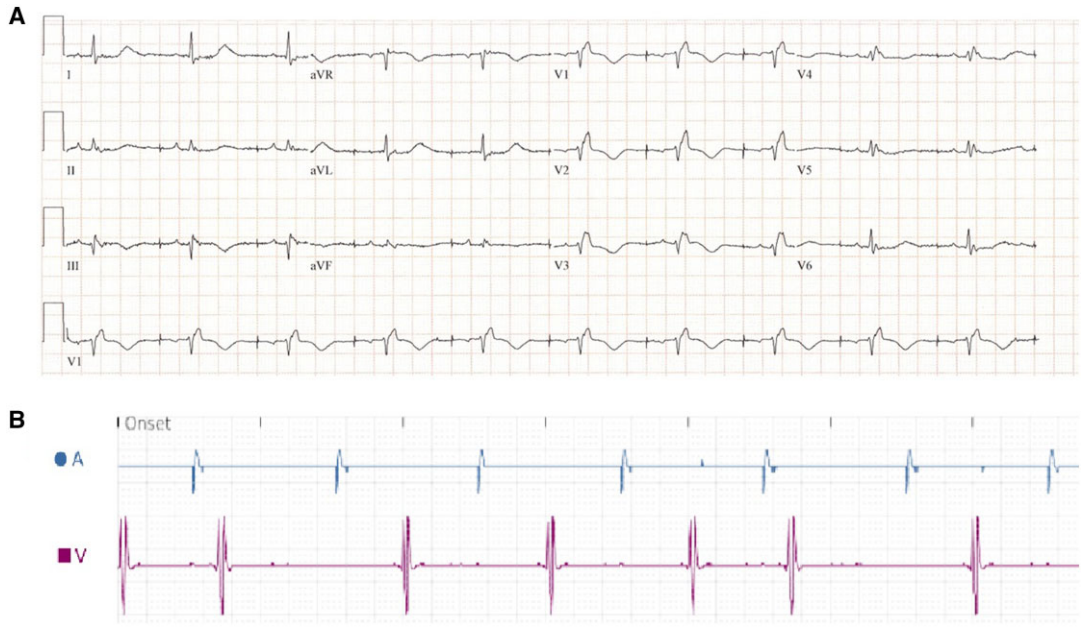


Figure 5 (*A*) Electrocardiogram after atrial lead sensitivity adjustment. AAIR mode, backup with DDDR mode, was restored. The rhythm was atrial-paced and ventricular-sensed. (*B*) Electrograms at outpatient follow-up no longer showed atrial tachycardia.

Discussion

Patient's presentation is consistent with pacemaker syndrome due to loss of AV synchrony. In this case, loss of AV synchrony is due to 'inappropriate' mode switching triggered by electrically isolated AT. Common causes of automatic mode switch are atrial tachyarrhythmia, far-field R wave sensing, noise oversensing, and battery depletion.

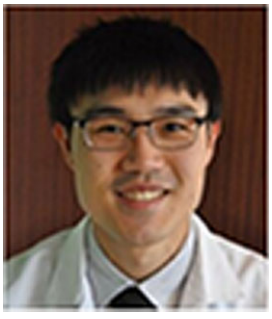
Symptoms of pacemaker syndrome range from fatigue, shortness of breath, palpitation, lightheadedness to syncope, or heart failure which are traditionally attributed to loss of AV synchrony as seen in single-chamber RV pacing.[3] Dual-chamber pacing reduces pacemaker syndrome and is recommended over single-chamber RV pacing in patients with SND with or without AV block.[4] Right ventricular pacing also causes loss of interventricular synchrony similar to that caused by left bundle branch block and has been proposed to be a contributor to pacemaker syndrome.[5] Right ventricular pacing may also lead to atrial fibrillation and it is recommended to programme the pacemaker to minimize ventricular pacing in patients with SND.[4] To maintain physiologic ventricular activation, His bundle pacing may be considered in patients with AV block and LVEF >40% who are anticipated to have >20% ventricular pacing.[4]

This patient had two electrically isolated atrial rhythms defined as AD. Regardless of the cause there must be electrical insulation or reduced electrical coupling between the two rhythms for their independent coexistence in the atria. Atrial dissociation might be incidentally seen on surface ECG without symptoms.[6,7] Invasive electrophysiologic study might be needed to identify AD in many cases.[1,7] Atrial dissociation may present with AV dissociation when there is electrical isolation between the SA node and the AV node.[1,2] Some patients will need a permanent pacemaker and identification of AD has a very important implication on the location of atrial lead placement, i.e. the atrial lead has to be placed in the site electrically connected to the AV node. Atrial dissociation could present as pseudo VT leading to misdiagnosis and unnecessary treatment.[1]

To the best of our knowledge, pacemaker syndrome as a result of AD has not been reported previously. We suspect that AD in this patient is the result of prior tricuspid valve interventions that created an electrically isolated zone in the right atrium close to where the new atrial lead was implanted.

In this case, the amplitude of the intracardiac AT signals is significantly lower than that of the sinus signals and changing the atrial lead sensitivity could provide the solution for inappropriate mode switch. If the amplitudes of both signals are similar, then more aggressive antiarrhythmic treatment, atrial lead revision, or ablation of atrial arrhythmias might be necessary to restore AV synchrony.

Lead author biography



Dr Natee Sirinvaravong, MD, is a cardiology fellow at Lankenau Medical Center in Wynnewood, PA, USA. He is passionate about cardiac electrophysiology and ECG teaching. He is planning to pursue further career in Clinical Cardiac Electrophysiology.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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