

# Syncope triggered by atrial flutter in a patient with a pacemaker: cross-stimulation—a case report

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## Background

Syncope in a patient with a pacemaker is a serious event requiring urgent action to ascertain its cause. Around 5% of cases are due to a pacemaker system malfunction.

## Case summary

An 82-year-old man underwent dual-chamber permanent pacemaker implantation due to intermittent high-degree atrio-ventricular block (AVB) in sinus rhythm. Nine months later, the patient reported episodes of syncope. The chest X-ray showed both leads to be at their expected positions. The electrocardiography (ECG) showed common atrial flutter. Ventricular capture during pacing in atrial demand pacing (AAI) mode confirmed cross-stimulation due to the switching of the atrial and ventricular leads at the pacemaker header.

## Discussion

Cross-stimulation is a rare possibility in a differential diagnosis of causes of syncope. The diagnosis is frequently made during the procedure or a few hours later. The lack of symptoms during 9 months in this case was likely due to the patient having normal sinus rhythm with preserved AV conduction most of the time, as well as ventricular capture from the atrial lead related to non-sensed P waves. When atrial arrhythmias occurred, the sensing of the F waves inhibited ventricular pacing. In order to avoid this complication, in patients with intermittent bradycardia, pacing at a slightly higher heart rate during implantation of the device should be recommended to see the chamber paced with the surface ECG connected to the device interrogator. The ECG and electrogram (EGM) should correlate during device interrogation in order to identify this complication.

## Keywords

Syncope • Pacemaker dysfunction • Cross-stimulation • Case report

## Learning points

- In patients with dual-chamber pacemakers that present with syncope, the accidental switching of the atrial and ventricular leads at the pacemaker header during implantation is a possible cause.
- The perioperative interrogation of the pacemaker using surface electrocardiography (ECG) before completing the final sutures at the end of every implantation is important to avoid missing this mistake.
- The ECG and electrograms should correlate not only during device implantation but also during device interrogation at follow-up in order to diagnose this complication.

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## Introduction

In patients with a previously implanted permanent pacemaker who are admitted to the hospital with syncope, the working diagnosis is often a device malfunction until the pacemaker interrogation reveals it is functioning normally.<sup>1</sup> However, in the majority of cases, pacemaker malfunction is not the cause of syncope.<sup>1,2</sup> Reflex syncope seems to be the most frequent diagnosis.<sup>2</sup> In the Ofman *et al* study,<sup>1</sup> 4.9% of patients studied were found to have pacemaker system malfunction as a cause of syncope.

Soon after implantation, the possible technical failures might be lead displacement, perforation, or incomplete connection at the lead–pulse generator interface.<sup>2</sup> Exit block may be present early on, but it also could be present after the first month following implantation.<sup>2</sup> Later problems include lead insulation failure, lead conductor fracture or battery depletion.<sup>2</sup>

The switching of the atrial and ventricular leads at pacemaker header is another possibility in patients with dual-chamber pacemakers. This rare complication has been described as a cause of syncope attacks in the majority of the cases in the early period after implantation.<sup>3</sup> However, this did not occur in our case. Our patient came to the hospital for recurrent syncopal episodes 9 months after the implantation of the pacemaker. Even 1 month after implantation of the device, this phenomenon was not detected in the check-up at the pacemaker clinic.

## Timeline

Date	Event
30 August 2019	Our patient underwent a dual-chamber permanent pacemaker implantation due to intermittent high-degree atrioventricular block in sinus rhythm, with normal pacing, sensing and threshold acute parameters.
30 September 2019	Check-up of the device 1 month after implantation, with normal pacing, sensing and threshold parameters.
5 June 2020	The patient was admitted to our hospital with syncope. The clinical examination was normal. The electrocardiography showed common atrial flutter, and a ventricular rate of 62 b.p.m. The chest X-ray showed both leads to be at their expected positions. We checked the device. The ventricular capture during pacing in AAI mode confirmed cross-stimulation due to the switching of the atrial and ventricular leads at the pacemaker header.
6 June 2020	Cross-stimulation was corrected after opening the generator surgically. The day after, the patient was discharged.
7 July 2020	Check-up of the device 1 month after implantation, with normal pacing, sensing, and threshold parameters. The patient experienced no further syncope at follow-up.

## Case presentation

An 82-year-old man underwent dual-chamber permanent pacemaker implantation due to intermittent high-degree atrioventricular block (AVB) in sinus rhythm in August 2019, with the following acute parameters: P wave 3.6 mV; atrial impedance 512 ohms; atrial pacing threshold  $0.6\text{ V} \times 0.5\text{ ms}$ ; R wave 4.3 mV; ventricular impedance 613  $\Omega$ ; ventricular pacing threshold  $0.4\text{ V} \times 0.5\text{ ms}$ . The patient has a past medical history of hypertension and hyperlipidaemia. He was taking olmesartan and atorvastatin.

Nine months later, the patient reported episodes of syncope during the previous 2 weeks.

The clinical examination did not reveal any abnormality. The ECG (Figure 1) showed common atrial flutter, left bundle branch block morphology of the QRS complex, and a ventricular rate of 62 b.p.m. The chest X-ray showed both leads to be at their expected positions: one lead in the right atrial appendage and the other lead in the right ventricle outflow tract (Figure 2A,B).

We checked the device. It was programmed to DDD mode, with a lower rate of 60 b.p.m., the paced AV delay was 200 ms, and the sensed AV delay was 150 ms. The stored EGM showed several episodes of fast ventricular rate (Figure 3). The first part of the tracing shows the rhythm of the patient interpreted as atrial sensing-ventricular pacing (AS-VP), with one ventricular sensed beat. Then, a fast rhythm at the ventricular channel develops with a cycle length around 190 ms. For atrial pacing, the ECG showed ventricular demand pacing (VVI) pacing (Figure 4). The ventricular capture during pacing in AAI mode confirmed cross-stimulation due to the switching of the atrial and ventricular leads at the pacemaker header. This was corrected after opening the generator surgically.

One month after implantation, the check-up of the device showed normal pacing, sensing, and threshold parameters. The patient experienced no further syncope at follow-up.

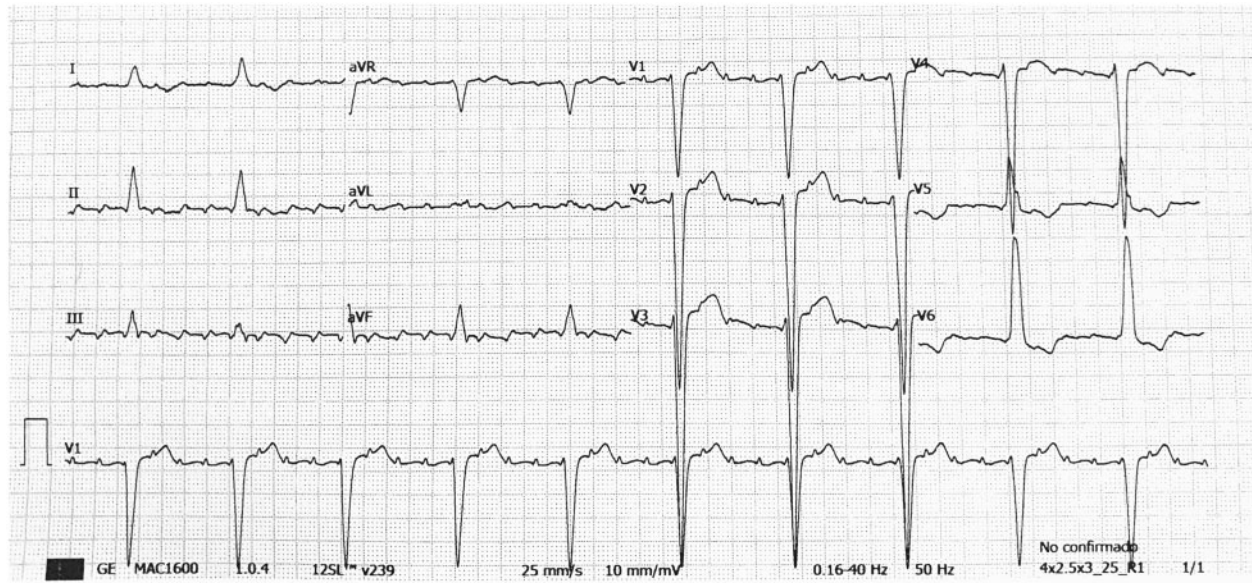
## Discussion

Cross-stimulation can be defined as stimulation of one cardiac chamber when the stimulation of the other is expected.<sup>4</sup> This was first described by Levine *et al.* in 1985.<sup>5</sup> Since then, subsequent reports have ascribed this situation to the proximity of the atrial lead to the ventricular chamber.<sup>6</sup> Other reports have attributed it to the dislodgement of the atrial lead into the ventricle or to the intrinsic design features of certain pacemakers and analysers.<sup>7</sup>

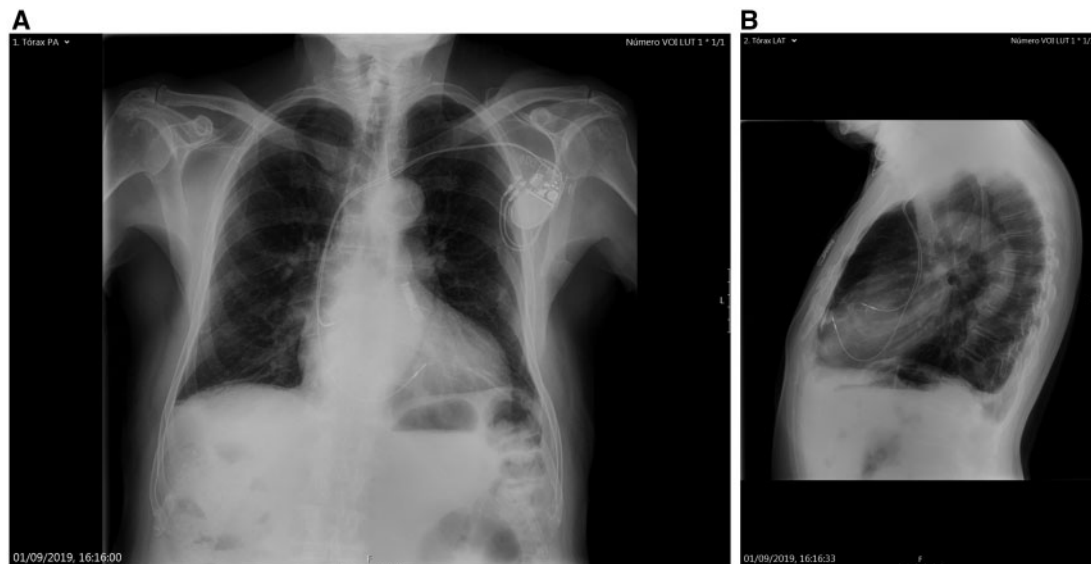
The accidental switching of the atrial and ventricular leads at the pacemaker header is another possible cause of this phenomenon.<sup>3</sup> As it is an embarrassing complication, it is probably underreported. Only a few cases have been published. Consequently, the manifestations are poorly documented.

One possible manifestation is an orthodromic endless loop tachycardia in patients with preserved anterograde conduction through the AV junction (these are patients implanted with pacemakers because of sick sinus syndrome). In patients with AVB, the lack of ventricular pacing can lead to syncopal episodes or even cardiac arrest.<sup>3</sup>

The time from implantation to the onset of symptoms varies greatly, although the diagnosis is frequently made during the procedure or



**Figure 1** The electrocardiography showed common atrial flutter, left bundle branch block morphology of the QRS complex, and a ventricular rate of 62 b.p.m.



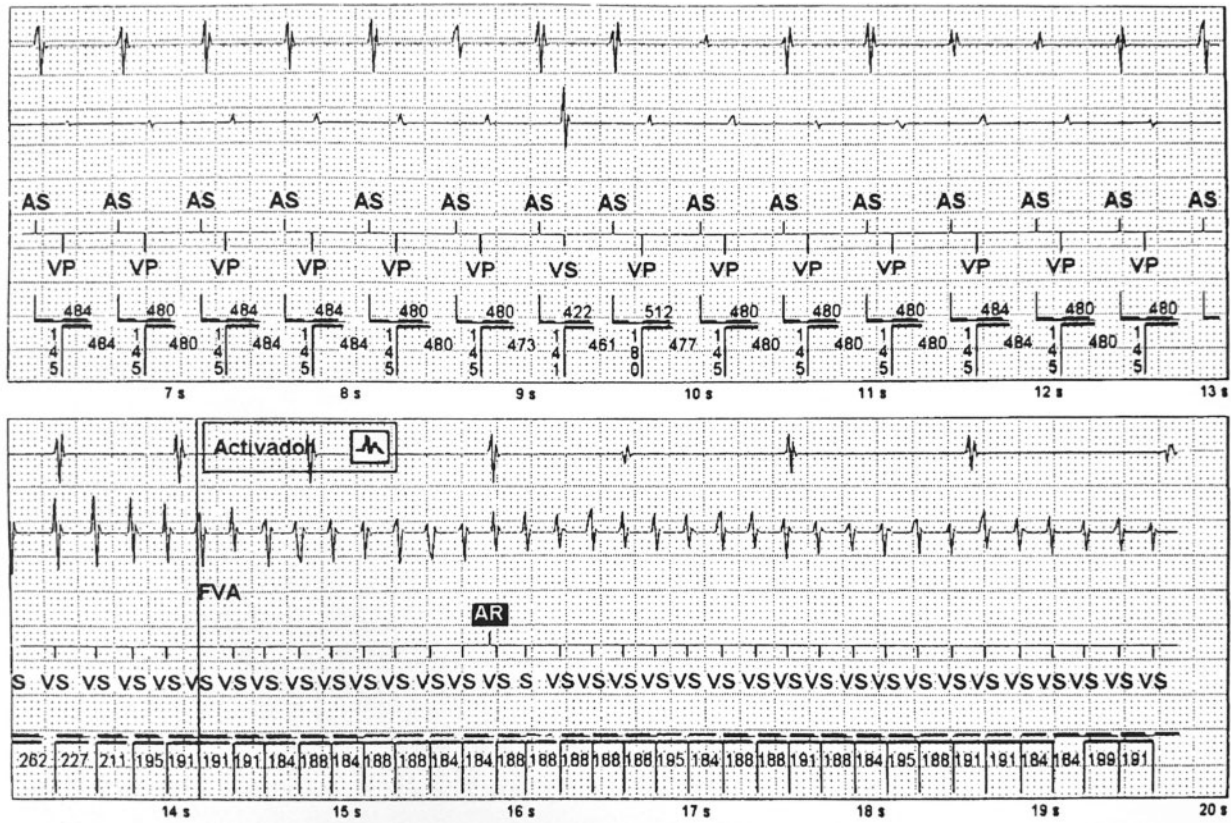
**Figure 2** (A,B) The chest X-ray showed both leads to be at their expected positions: one lead in the right atrial appendage and the other lead in the right ventricle outflow tract.

a few hours later.<sup>3</sup> However, there is a reported case of a diagnosis of this phenomenon 3 years after implantation.<sup>8</sup>

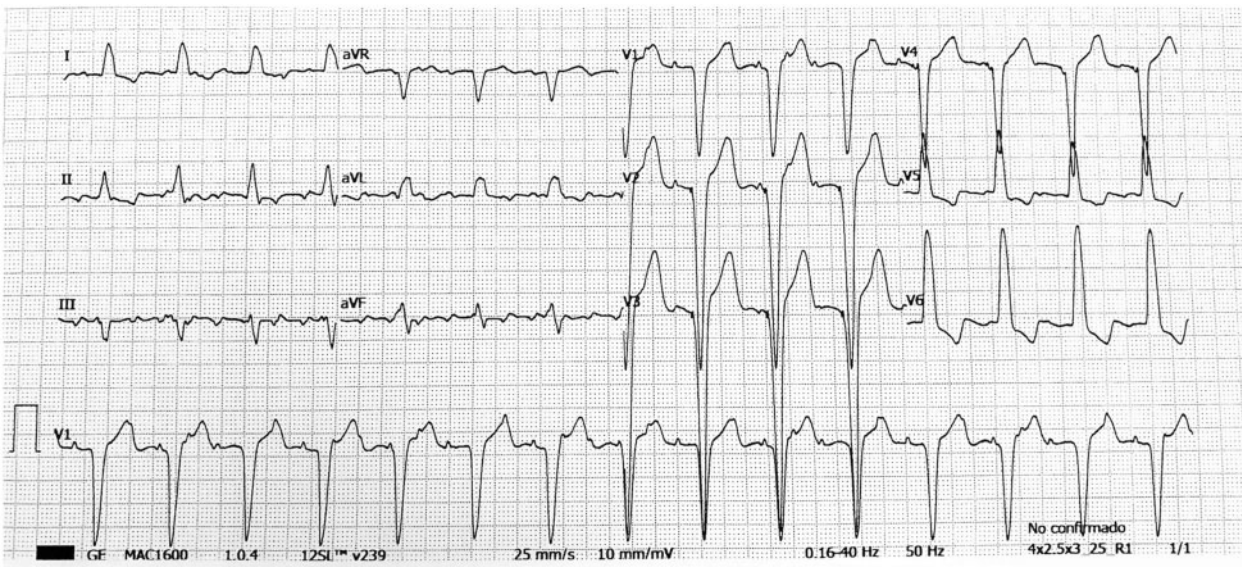
Our patient had a normal sinus rhythm at the time of implantation, with a normal PR interval and a heart rate of 80 b.p.m. When connecting both leads to the head of the pacemaker, the result was a sensed rhythm instead of a paced rhythm, and the unintentional switching of the leads was not detected.

The mistake was also not detected in the device interrogation the day after implantation. In the interrogation carried out at the pacemaker clinic, the diagnosis was not made. The P wave was non-sensed, thus keeping pacing in the atrial channel and protecting against asystole. This was probably interpreted as sick sinus syndrome. The EGM was not correlated with the ECG during the device interrogation.





**Figure 3** The stored EGM showed several episodes of fast ventricular rate. The first part of the tracing shows the rhythm of the patient interpreted as AS-VP, with one ventricular sensed beat. Then, a fast rhythm at the ventricular channel develops.



**Figure 4** For atrial pacing, the electrocardiography showed VVI pacing.

At initial presentation, the ECG showed atrial flutter (Figure 1). The first diagnostic suspicion was the dislodgement of the atrial lead into the ventricle. The chest X-ray ruled out that possibility. The ECG showed ventricular capture during pacing in AAI mode. This confirmed the cross-stimulation due to the switching of the atrial and ventricular leads at the pacemaker header.

While our patient was in sinus rhythm the ventricular chamber was paced. The P wave amplitude was lower than the sensitivity setting in the ventricular channel, protecting against asystole. The patient developed atrial flutter, and the possible loss of AV synchrony could favour the development of atrial arrhythmias. The analysis of the intracardiac EGM showed that pacing in the ventricles was inhibited primarily by the sensing of F waves with a higher amplitude, which led to the development of symptoms. Therefore, the precipitation of symptoms was due to the atrial arrhythmia.

The timing for the onset of symptoms depends on the cardiac rhythm of the patient, and the pacemaker programming. Syncope may not occur if the P wave amplitude is lower than the sensitivity setting or if the sinus rate is slower than the pacemaker lower rate, thus making the ventricular stimulation possible, or if the patient is not pacemaker-dependent (preserved anterograde AV conduction, intermittent AV block). If the amplitude and rate of P waves are high enough to be sensed and the patient is pacemaker-dependent, symptoms will occur early after implantation and the mistake could be corrected. In our case, the lack of symptoms during 9 months was likely due to the patient having normal sinus rhythm with preserved AV conduction most of the time (AVB was intermittent), as well as ventricular capture from the atrial lead related to non-sensed P waves. However, when atrial arrhythmias occurred, the sensing of the F waves inhibited ventricular pacing.

In order to avoid such an embarrassing complication, in patients with intermittent bradycardia, pacing at a slightly higher heart rate during implantation of the device should be recommended to see the chamber paced with the surface ECG connected to the device interrogator. The ECG and EGM should correlate during device implantation, but also during a device interrogation.<sup>9</sup> The correlation of the ECG and EGM during the device interrogation when the patient came to the hospital with syncope led to the correct diagnosis. If this had been done during the device interrogations, the diagnosis would likely have been made before the patient had symptoms.

In patients with dual-chamber pacemakers and intermittent AVB, cross-stimulation is a rare possibility in a differential diagnosis of causes of syncope. The timing from the implantation to the onset of symptoms could vary greatly.

The case report was approved by the hospital's investigation committee.

## Lead author biography



Olga Durán Bobin is an electrophysiologist and works in the Arrhythmia Unit of Hospital Universitario Lucus Augusti (Lugo, Spain). She had cardiology training in Hospital Universitario Salamanca (Spain).

## Supplementary material

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

**Slide sets:** A fully edited slide set detailing these cases and suitable for local presentation is available online as [Supplementary data](#).

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

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