ESC European Society of Cardiology

European Heart Journal - Case Reports
doi:10.1093/ehjcr/ytab096

CASE REPORT
Arrhythmias/electrophysiology

A case report of inappropriate inhibition of ventricular pacing due to a unique pacemaker electrogram storage feature

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Received 15 September 2020; first decision 27 October 2020; accepted 25 February 2021

Background Modern permanent pacemakers (PPMs) have individual features designed to identify cardiac rhythm abnormalities and improve their performance. Inappropriate pacing inhibition may be an undesired outcome from these features and cause symptoms in patients who require frequent pacing, leading to dizziness, and syncope. Inappropriate inhibition can be difficult to identify in circumstances that are intermittent and difficult to reproduce.

Case summary A 57-year-old female underwent a mitral valve replacement (MVR) for severe mitral stenosis. One month following MVR, she presented with symptomatic third-degree atrioventricular block, and a dual-chamber PPM (Advisa^{TM}, Medtronic, Minneapolis, USA) was implanted and programmed DDD 50–130 b.p.m. At the 3-month follow-up, she reported frequent episodes of lightheadedness. She was found to have intermittent ventricular pacing inhibition on a 48-h Holter monitor due to an internal function of the Advisa^{TM} series of PPMs that attempts to store an electrogram (EGM) every 1 h and 30 s. During the EGM storage, an amplified signal from the storage capacitor can result in oversensing by the ventricular channel and inappropriate pacing inhibition.

Discussion To rectify the issue, the ventricular lead sensitivity value was increased from 0.9 mV to 1.2 mV. No instances of inappropriate ventricular pacing inhibition were noted on follow-up. To our knowledge, this is a rare case of inappropriate ventricular pacing inhibition caused by a combination of PPM self-adjusting sensitivity algorithm and oversensing every 1 h and 30 s from an amplified storage capacitor. Physicians should be aware of this possible complication and differentiate it from device or lead malfunction.

Keywords Pacemaker • Pacing inhibition • Advisa • Case report • Oversensing • Dizziness

Learning points

- This case highlights an important cause of inappropriate inhibition of ventricular pacing due to a non-programmable feature of a pacemaker and can be translated to other manufacturers that might have other proprietary algorithms with similar effect.
- If a regular inhibition of ventricular pacing is seen in a Medtronic Advisa^{TM} pacemaker, the electrogram storage feature resulting in inhibition should be considered as a cause and sensitivity reduced as a treatment option.

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Handling Editor: Christoph Sinning
Peer-reviewers: Christian Sohns; Konstantinos Iliodromitis; Fabian Barbieri; David Duncker and Richard Ang
Compliance Editor: Edwina McNaughton
Supplementary Material Editor: Nida Ahmed

Introduction

All modern permanent pacemakers (PPMs) can function in a demand mode, meaning that pacing stimuli are inhibited when the patient's intrinsic heart rate exceeds the programmed lower rate limit or when activity is sensed in the implanted cardiac chamber. There are numerous PPM manufacturers with various individual features designed to identify cardiac rhythm abnormalities and improve the devices' performance.[1,2] Inappropriate pacing inhibition may be an undesired outcome from these features and cause symptoms in patients who require frequent pacing. Inappropriate pacing inhibition can be challenging to identify in circumstances that are intermittent and difficult to reproduce. We identified a rare case of inappropriate pacing inhibition caused by a non-programmable internal PPM feature resulting in the patient experiencing dizziness.

Timeline

Day 0	A 57-year-old woman with severe mitral stenosis underwent mechanical mitral valve replacement.
Day 30	She presented with symptomatic intermittent complete heart block requiring implantation of a dual-chamber pacemaker (Advisa^{TM}).
Day 122	She complained of intermittent unprovoked lightheadedness and was identified to have inappropriate pacing inhibition on a 48-h Holter monitor.
Day 162	Readjustment of ventricular chamber sensitivity value and repeat ambulatory electrocardiogram monitor showed resolution of the problem.
Day 256	No further pacing inhibition on monitor and symptoms resolved.

Case presentation

A 57-year-old Caucasian woman underwent a mitral valve replacement (MVR) for severe mitral stenosis. Her past medical history included obstructive sleep apnoea, Hashimoto's thyroiditis, and gastro-oesophageal reflux disease. The regular medications included thyroxine 50 mcg daily, warfarin, and Aspirin 81 mg daily. One month following MVR, she presented with symptomatic third-degree atrioventricular (AV) block, and a dual-chamber PPM (Advisa^{TM}, Medtronic, Minneapolis, USA) was implanted as per guidelines and programmed DDD 50–130 b.p.m.[3,4] However, several months after her device implantation, she reported frequent episodes of abrupt lightheadedness that were not orthostatic. Device interrogation parameters were unchanged from the time of implant. P waves were 2.8 mV and R waves were 18 mV. Right atrial and right ventricular lead impedances were 531 and 862 Ohms, respectively. Capture thresholds on both leads were <1 V/0.5 ms. Provocation testing in the clinic with deep respiration and active movement of the arms and the generator pocket did not evoke pacing inhibition or show artefact on either atrial or ventricular leads. Twelve-lead electrocardiogram (ECG) showed atrial tracking with ventricular pacing and no loss of capture. Chest X-ray showed no obvious lead dislodgement or fracture.

Since she was stable and had no syncope, she was ultimately sent home with an ambulatory Holter monitor. She had intermittent ventricular pacing inhibition, as shown in *Figure 1* documented only on the Holter monitor. Ventricular pacing inhibition was seen with sinus rate tracking of ≤75 b.p.m., was not preceded by any visualized artefact, and did not correspond to standard device features such as Managed Ventricular Pacing or Capture Management. AV delay remained the same during ventricular capture, excluding AV hysteresis.

The pacing inhibition's recurrent and predictable nature every 1 h and 30 s suggested a device feature as opposed to a hardware malfunction. This was reported to the device manufacturer and discussed with a technical expert from the company. In this case, it was caused by an internal function of the Advisa^{TM} series of PPMs that attempts to store an electrogram (EGM) every 1 h and 30 s. During EGM storage, an amplified signal from the storage capacitor can result in oversensing by the ventricular channel and inappropriate ventricular pacing inhibition. To rectify the issue, the ventricular lead sensitivity value was increased from 0.9 mV to 1.2 mV. No further instances of inappropriate ventricular pacing inhibition were noted on the follow-up ambulatory monitor at 3 months, and the patient's symptoms resolved.

Discussion

Dizziness or syncope is a concerning symptom in patients with PPMs, requiring urgent attention and thorough investigation. Although there are many potential causes uncovered through careful history and review of medications, additional tests such as a 12-lead ECG, chest X-ray, and device interrogation with provocative manoeuvres may help rule out PPM hardware malfunction (*Figure 2*).[5,6] Ambulatory monitoring is useful in identifying causes of 'pseudo-malfunction' as a result of increasingly complex, proprietary device features.[7] These features can cause confusion and sometimes lead to inappropriate pacing inhibition as seen in our patient who was pacing-dependent.[1,2]

Pacemaker oversensing occurs due to multiple factors that can be categorized as extrinsic or intrinsic. Extrinsic factors include lead noise or artefact caused by electromagnetic interference from machinery and sources of high voltage.[5–8] Intrinsic factors can be from physiologic or non-physiologic sources. Physiologic sources include cross-talk due to oversensing of signal in the opposite chamber.[5,7,8] Non-physiologic sources include lead fracture or a misaligned connection of the lead connector pin into the device connector block.[7] If all of these causes are ruled out, then 'pseudo malfunction' due to device-specific features should be considered.[1,2] This case report highlights a rare cause of intrinsic non-physiologic noise wherein a

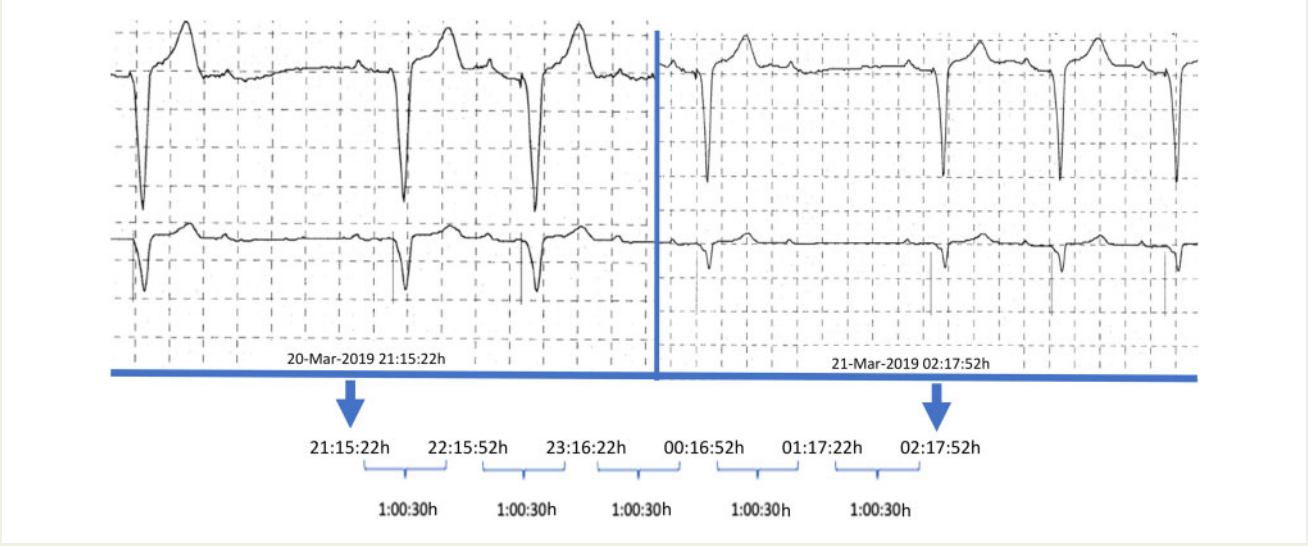

Figure 1 Holter monitor showing two time points of pacing inhibition related to intermittent oversensing in the ventricular lead. This was due to a repetitive feature of electrogram collection every 1 h and 30 s.

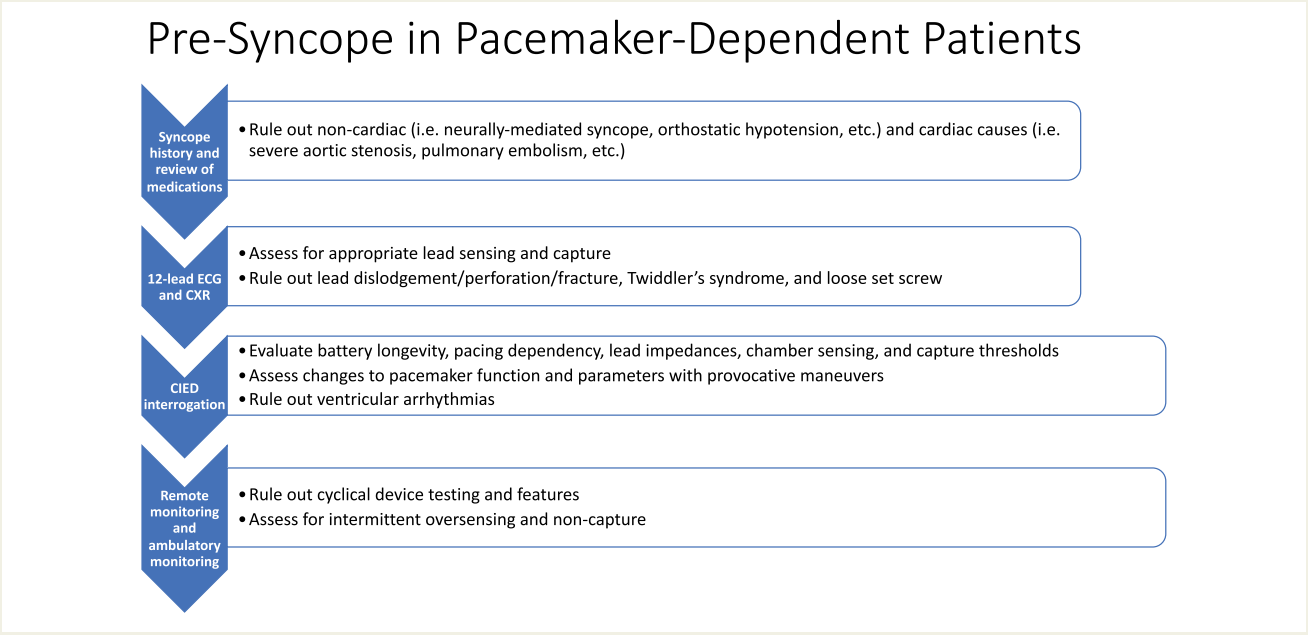

Figure 2 Stepwise approach to pre-syncope in pacemaker-dependent patients.

signal is generated by a circuit within the PPM that interferes with normal device function. In the Advisa^{TM} series of PPMs, ventricular lead sensing is managed by a self-adjusting function that was originally developed for implantable cardioverter-defibrillators to identify tachyarrhythmias. Sensing filters are usually present in defibrillators to allow for an adjustment of ventricular sensitivity with decay for a programmed length of time (nominally 450 ms).[9] The self-adjusting feature is based on a time-dependent sensitivity decay after a ventricular sensed event. After each ventricular sensed event, the sensitivity decay starts at either 75% of the R wave (18 mV for our case) or a value calculated as 9 times the programmed sensitivity value (0.9 mV in our case = 8.1 mV), whichever is greater during diastole.[10]

The Advisa^{TM} model of PPMs also has a feature that collects EGMs every 1 h and 30 s, resulting in intermittent ventricular pacing inhibition as recorded on the Holter monitor in our patient (*Figure 1*). As the EGM storage feature turned ON and OFF every 1 h and 30 s, there were instances of an amplified signal generated from the storage capacitor resulting in ventricular oversensing when the self-adjusting sensitivity feature of the ventricular channel approached 0.9 mV in late diastole. The EGM storage feature cannot be

programmed OFF in the Advisa^{TM} model and therefore the device manufacturer needs to be made aware of this to fix the underlying problem. The only immediate solution to ventricular oversensing was increasing the ventricular sensitivity value from 0.9 mV to 1.2 mV. This change in sensitivity was possible without any adverse impact on device function as the patient was pacing-dependent, and the ventricular-sensed amplitude was 18 mV. A repeat Holter monitor was performed after 3 months, and there were no further episodes of pacing inhibition.

This is a rare case of inappropriate ventricular pacing inhibition caused by a combination of PPM self-adjusting sensitivity feature and oversensing of a recurrent signal produced by the EGM storage amplifier every 1 h and 30 s. Despite thousands of implanted Advisa^{TM} PPMs, this is the second case report of this subtle anomaly that can cause significant symptoms in pacing-dependent patients.[10] Although several proprietary device features can cause unexpected pacing inhibition, the one highlighted in this article is non-programmable and easily missed without further ambulatory monitoring. Technical assistance from the device manufacturer should be sought after when these issues are identified. Cardiologists should be aware of this possible complication and differentiate it from device or lead malfunction.

Lead author biography

Dr Habib R. Khan graduated from University of Peshawar, Pakistan in 2002. After completing resident training, he trained in medicine in Ireland and obtained MRCP Ireland and UK. He is certified by European Examination in General Cardiology, and Cardiac Rhythm Management certification in Devices by British Heart Rhythm Society. He is currently appointed as an Assistant Professor at University of Western, London-Ontario, Canada. Dr Khan regularly publishes in field of cardiovascular medicine.

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.

Funding: None declared.

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