Recurrent loss of pacemaker capture following premature ventricular contractions

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Introduction

Exit block is a phenomenon classically defined as failure of an impulse to excite the surrounding tissue when falling outside of the refractory period of the heart.[1] Pacemaker exit block typically occurs as a result of inflammation and/or fibrosis at the electrode-myocardium interface.[2] Here, we report a unique case of pacemaker exit block caused by premature ventricular contractions (PVCs) and discuss its clinical implications.

Case report

A 57-year-old male patient underwent a mechanical aortic valve replacement with ascending aortic root replacement for severe aortic stenosis in the setting of a bicuspid aortic valve. He had no other past medical history, and there was no personal or family history of presyncope, syncope, or sudden cardiac death. His preoperative electrocardiogram demonstrated normal sinus rhythm with a right bundle branch block (RBBB) (Figure 1). His initial postoperative electrocardiogram was unchanged. Transthoracic echocardiography (TTE) demonstrated a normal left ventricular ejection fraction (LVEF). He developed a brief episode of paroxysmal atrial fibrillation on the fourth post-operative day and was subsequently started on amiodarone and bisoprolol. Later that day, he developed ten seconds of paroxysmal atrioventricular (AV) block and syncope. Amiodarone was discontinued. On telemetry review, a PVC immediately preceded AV block and ventricular asystole (Figure 2). The epicardial pacing wires, which had been disconnected and insulated earlier that day in anticipation of removal the following day, were reconnected to the temporary external pacemaker.

It was determined that the patient had developed PVC-induced paroxysmal AV block (Phase 4 AV block), supported by the presence of underlying conduction system disease (RBBB). A dual-chamber permanent pacemaker was implanted (Medtronic Astra XT DR MRI SureScan X2DR01; Medtronic 4074-58 centimeter (cm) CapsureSense MRI SureScan right ventricular [RV] lead; Medtronic 4076-52 cm CapsureFix Novus MRI SureScan right atrial lead; Medtronic, Minneapolis, MN), with appropriate pacing and sensing characteristics (right ventricle: impedance 760 ohms, R wave 6.5 mV, capture 0.75 V at 0.4 ms; right atrium: impedance 456 ohms, P wave 2.6 mV, capture 0.5 V at 0.4 ms).

Within 24 hours after pacemaker implantation, there were episodes of loss of ventricular capture post-PVCs. Chest radiography demonstrated appropriate pacemaker lead positioning without evidence of lead dislodgment (Supplemental Figure 1). Laboratory investigations did not demonstrate any electrolyte or acid-base disturbances, and the patient was not hypoxemic. Device interrogation showed both unipolar and bipolar RV capture thresholds at less than 0.5 V at 0.4 ms. Pacemaker output needed to be programmed to a maximum output of 8 V at 1.2 ms to maintain consistent capture. However, at these settings, the pulse generator battery longevity was only anticipated to be 2 months.

KEY TEACHING POINTS

- Paroxysmal pacemaker loss of capture may occur despite appropriate pacemaker lead testing.
- Transient pacemaker exit block should be considered on the differential diagnosis when paroxysmal pacemaker loss of capture occurs.
- Pacemaker exit block may be induced by locoregional premature ventricular contractions (PVCs), adding to its differential diagnosis.
- PVC-induced loss of capture may require pacemaker lead repositioning.
- Electrophysiology studies may be helpful in demonstrating the level of atrioventricular node dysfunction in cases of paroxysmal pacemaker loss of capture.

KEYWORDS Pacemaker; Loss of capture; Premature ventricular contraction; Atrioventricular block; Exit block

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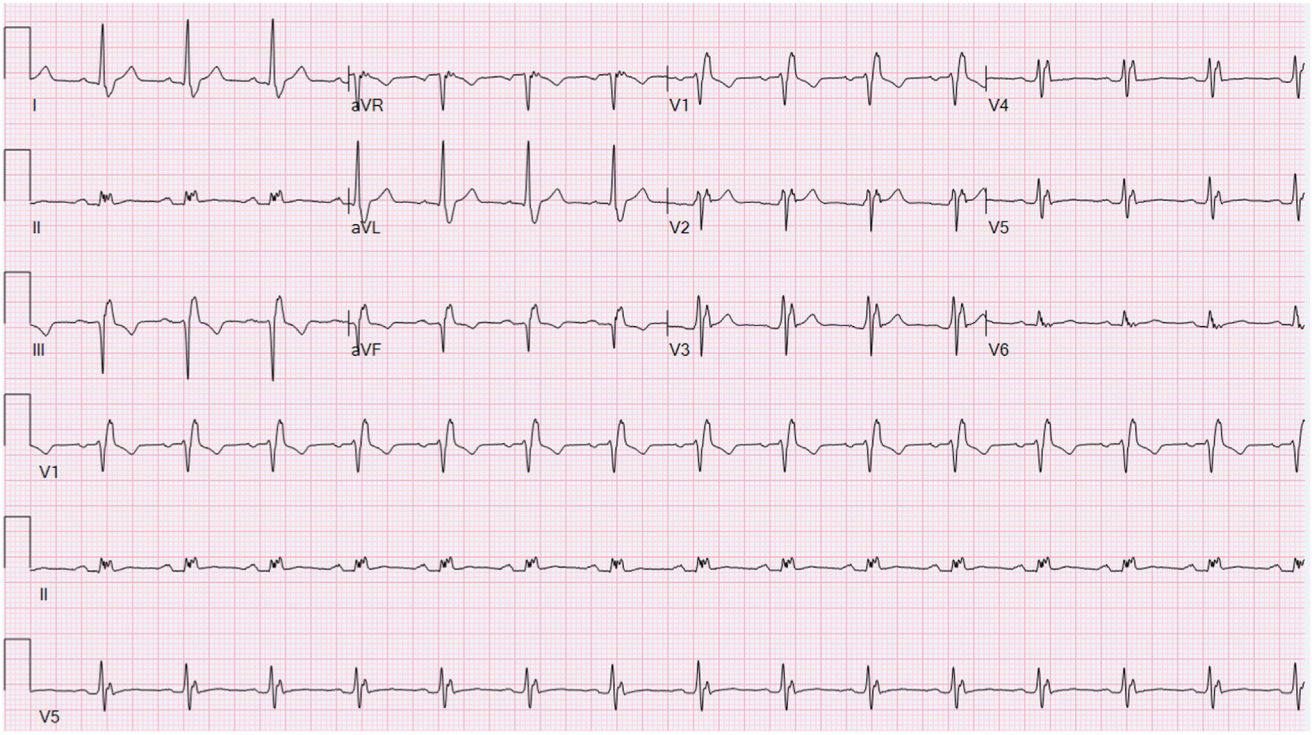

Figure 1 Preoperative 12-lead electrocardiogram demonstrating normal sinus rhythm with a right bundle branch block.

Further detailed review of telemetry identified that pacemaker loss of capture (LOC) only occurred after left bundle branch block (LBBB) morphology PVCs (Figure 3A) and *not* with either RBBB morphology PVCs or normal intrinsic conduction (Figure 3B). Coupling intervals for LBBB and RBBB morphology PVCs are displayed in Figure 3. Shorter PVC coupling intervals (less than 400 ms) were often seen with LBBB morphology PVCs that induced pacemaker LOC. RBBB morphology PVCs with similar coupling intervals did not result in pacemaker LOC. Also, this phenomenon was documented to occur with both the temporary epicardial pacing lead (located on the right ventricle) and the permanent transvenous RV lead. This was suggestive of localized RV PVC-induced pacemaker exit block. Sotalol was started for PVC suppression and the pacing output was reduced to 3.5 V at 0.4 ms. At 3 months of follow-up, ventricular pacing threshold remained at less than 0.5 V at 0.4 ms, and no further episodes of syncope or documented paroxysmal atrioventricular block have been observed.

Discussion

The response to a pacing stimulus, in which a cathode stimulus delivers electrical impulses to the surrounding myocardium, is an "all or none" phenomenon.[3] Numerous factors determine whether an electrode will consistently depolarize and "capture" the nearby myocardium. Failure of normally elaborated impulses to capture

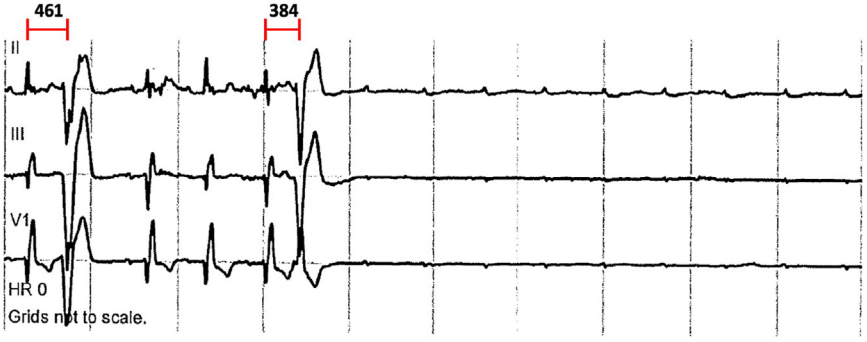

Figure 2 Telemetry with epicardial pacemaker wire in situ but disconnected from temporary pacemaker pack. A right bundle branch block (RBBB) premature ventricular contraction (PVC) induces complete atrioventricular block and ventricular asystole. PVC coupling intervals are marked with calipers and presented in ms. Left bundle branch block PVC coupling interval 461 ms and RBBB PVC coupling interval 384 ms.

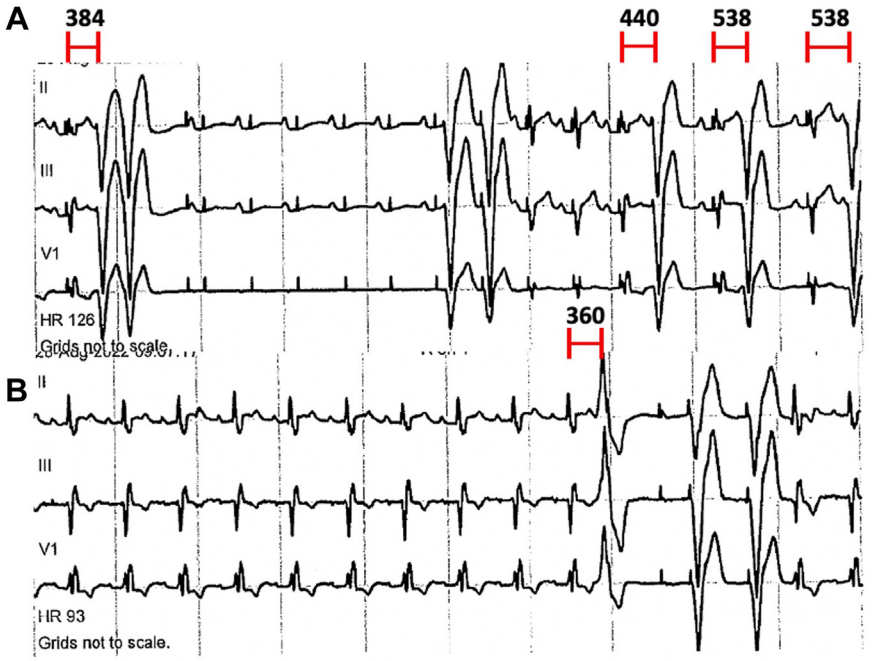

Figure 3 **A:** Telemetry post–permanent pacemaker implantation. Left bundle branch block morphology premature ventricular contraction (PVC) produces both paroxysmal atrioventricular (AV) block and loss of pacemaker capture. **B:** Telemetry post–permanent pacemaker implantation. Right bundle branch block morphology PVC produces paroxysmal AV block but does not cause loss of pacemaker capture. PVC coupling intervals measured in ms.

the myocardium is termed "loss of capture," in which there is a pacing spike followed by no P wave or QRS complex. Its causes can include lead dislodgment or malposition, premature lead failure, premature battery depletion, programming errors with suboptimal output, lead fracture, pacemaker exit block from myocardial fibrosis and/or inflammation, cardiomyopathy, breach of insulation, end of battery life, electrolyte disturbances (in particular hyperkalemia), acidemia, hypoxia, medication-induced alterations of the capture threshold, and electrical cardioversion.[2,4–6]

Local pacemaker exit block occurs when capture of myocardium immediately at the cathode fails to conduct to the surrounding myocardium, and may be divided into 2 types: "type 1," in which there is a progressive delay in conduction prior to loss of capture, and "type 2" in which loss of capture occurs without warning.[7] To date, the literature has only described nearby myocardial fibrosis and inflammation as etiologies of local pacemaker exit block.[4] This novel case elucidates a unique etiology of pacemaker exit block—PVCs. There were no metabolic or structural (either myocardial or pacemaker lead–related) factors or medications that may have contributed to exit block. With PVC suppression, there was no further pacemaker exit block. We hypothesize that LBBB PVCs originating close to the RV apical pacing site were able to cause local refractoriness and resultant exit block, whereas RBBB PVCs originating at more remote sites failed to do so. This is further supported by LBBB morphology PVC coupling intervals, as shorter intervals (less than 400 ms) appear more likely to result in pacemaker LOC. Given that the patient had already demonstrated a tendency to develop phase 4–mediated block prior to pacemaker implantation, it is plausible that LBBB PVCs were able to similarly induce local phase 4 block. To our knowledge, there have been no reported cases of PVCs as the culprit for local pacemaker exit block.

Conclusion

Paroxysmal pacemaker LOC, and its possible resultant symptoms, may occur despite appropriate pacemaker lead testing. Transient pacemaker exit block should be considered on the differential diagnosis when this phenomenon occurs. To date, this is the first case demonstrating that pacemaker exit block may be induced by locoregional PVCs, adding to its differential diagnosis. In such cases, PVC suppression, either pharmacologically or with ablation, may prevent LOC.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.09.006.

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