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# Case Report: Pacemaker Mimics Ventricular Extrasystoles?

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## ■ Introduction

We report about a 78 year old patient who presented himself to our clinic. He reported about palpitations, dizziness and dyspnea since dual chamber pacemaker implantation because of a bradycardia-tachycardia syndrome. The prepared resting ECG revealed an intermittent atrio-ventricular sequential pacing and ventricular premature beats. These premature beats had a left bundle-branch-block morphology and an inferior axis and were causing a peripheral pulse deficit. Under temporary deactivation of the pacemaker ventricular premature beats could not longer be documented. In the assumption that the premature beats were caused by the ventricular pacing lead, the patient was scheduled for right ventricular lead revision. Preoperatively a therapeutic approach/attempt with amiodarone was started to relieve the symptoms. The drug treatment was intended only for a short time, until an operation was scheduled. A holter electrocardiogram after amiodarone saturation showed a reduction of premature-beat burden to 1.3% with 1100 premature beats during 24 hours. At the 6-week follow up 1500 premature beats per 24 hours were documented. Under this low rate of ventricular premature beats the patient remained asymptomatic. Considering

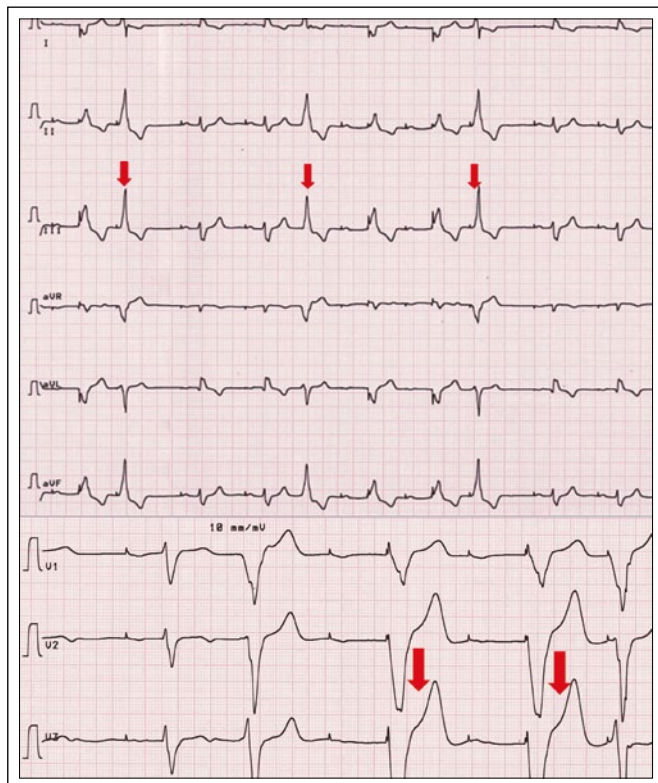
this and respecting the wish of the patient, we decided at this time to continue the antiarrhythmic therapy and to not revise the right ventricular pacing lead.

This case imposes by a rare consequence of a pacemaker implantation consisting in induced ventricular premature beats. These were probably due to an electrical irritation in the region of the right ventricular outflow tract.

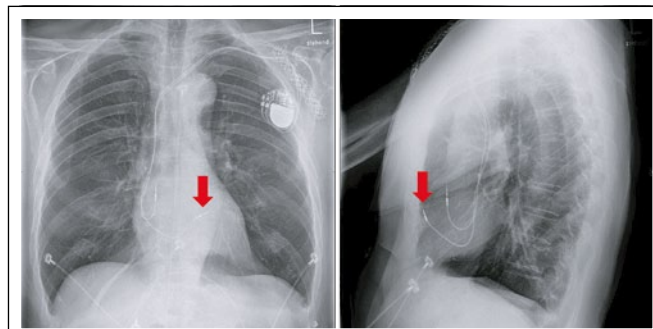
## ■ Case Report

### History

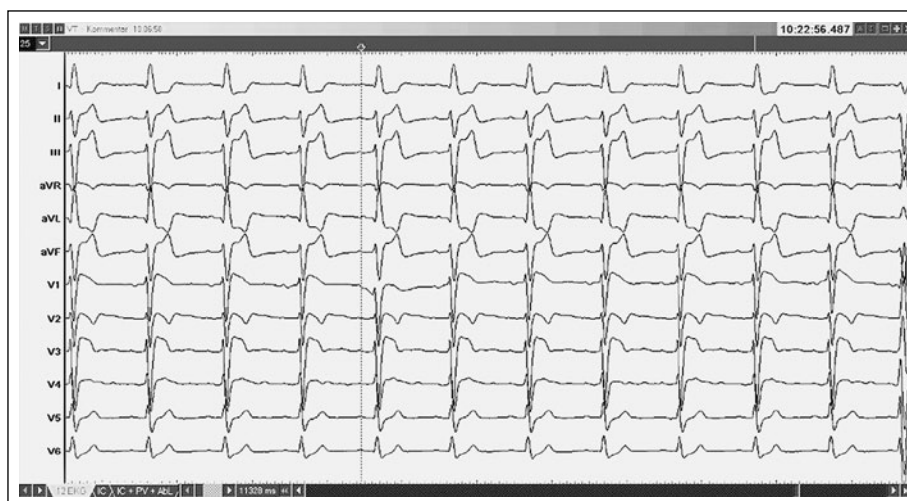
We report about a 78 year old patient who presented to our clinic earlier this year. He reported about palpitations, dizziness and dyspnea. Relevant pre-existing conditions were a coronary artery disease with status post bypass surgery. A coronary angiography in 2014 showed a good long-term outcome. In addition there was an ablation of symptomatic left ventricular extrasystoles from the region of posterolateral papillary muscle in the history of the patient. Transthoracic echocardiography showed normal systolic left ventricular function with an ejection fraction of 60% without relevant cardiac valve disorders. Because of tachyarrhythmic atrial fibrillation medication with a beta-blocker (metoprolol 47.5 mg bid) was started in the past. During the further course a dual chamber pacemaker (RELIA REDR01, Medtronic) was implanted to treat bradycardia-tachycardia-syndrome. Since the pacemaker implantation, the described symptoms like palpitations, dizziness and dyspnea occurred significantly stronger. The currently recorded resting ECG revealed an intermittent atrio-ventricular sequential pacing and ventricular premature beats (VPB). These VPB had a left bundle-branch-block morphology and an inferior axis and were causing a peripheral pulse deficit (Fig. 1). The chest X-ray brought an inconspicuous finding, with a high septal location of the ventricular lead (Fig. 2). The current pacemaker readings showed good and stable measured values. After reprogramming the pacemaker with a maximum



**Figure 1.** Resting ECG with recordings of ventricular premature beats with left bundle-branch block morphology. These are marked by the red arrow and show a partial pseudo-fusion.



**Figure 2.** X-ray image of the thorax in posterior-anterior and lateral radiation direction. It shows no pathological signs and the proper position of the pacemaker leads. The high septal ventricular lead is marked by a red arrow.



**Figure 3.** 12-lead ECG with no documentation of ventricular premature beats after temporary deactivation of the pacemaker.

atrioventricular delay of 350 ms and reduction of the right ventricular pacing percentage almost no premature beats were recorded. The patient reported about a significant symptom relief under the reprogramming and was discharged.

### Treatment and Course

After a few weeks the patient presented once more in our out-patient clinic with reoccurring palpitations, dizziness and dyspnea. The ECG revealed the previously documented premature beats with left bundle branch block morphology and inferior axis. The pacemaker interrogation showed 45% premature beats in all ventricular contractions. Under temporary deactivation of the pacemaker no ventricular premature beats could be documented (Fig. 3). Based on the assumption that the premature beats are caused by the ventricular pacing lead, the patient was scheduled for right ventricular lead revision. Preoperatively a therapeutic attempt with amiodarone was started. A holter electrocardiogram after amiodarone saturation showed a premature-beat burden of 1.3% with 1100 premature beats during 24 hours. A 6-week-follow up showed 1500 premature beats per 24 hours. The ventricular pacing rate was 100%; the programmed lower rate limit was 70 beats per minute. The patient is still asymptomatic, so that no revision of the ventricular lead was performed as of now.

### Discussion

Our case describes the possibility that premature ventricular contractions could be triggered by a septal location of the pacing lead. The patient was highly symptomatic with significant reduction in the quality of life. In the literature, the septal position is controversial. In general it is assumed that right ventricular septal pacing from mid septum improves left ventricular systolic ejection fraction and left ventricular synchrony [1]. The right ventricular apex has been the traditional position for lead placement in patients scheduled for pacemaker implantation. Pacing at the right ventricular apex may have long-term deleterious effects on left ventricular function, promoting heart failure and increasing mortality [2]. It may be difficult to distinguish between idiopathic and pacemaker induced extrasystoles. In our case, the disappearance of extrasystoles after inhibition of the pacemaker has led to the assumption that the premature beats were caused by the electric impulses of the pacemaker. In case of a mechanical trigger,

premature beats would have been expected to persist even after inhibiting the pacemaker. However, patients with ischemic cardiomyopathy appear, in general, more susceptible to ventricular arrhythmias by the modified heart muscle tissue [3].

Preoperatively a therapeutic trial with amiodarone was undertaken. A holter electrocardiogram after amiodarone saturation showed a premature-beat-burden of 1.3% with 1100 premature beats in 24 hours. A 6-week-follow up showed 1500 premature beats per 24 hours. The patient was still asymptomatic, so that no revision of the ventricular lead was performed.

Finally, we must conclude that it is still unclear whether the arrhythmia is induced by pacing or is idiopathic. The fact that the deactivation of the pacemaker abolished the extrasystoles, argues against the idiopathic mechanism. Anyway, a ventricular lead revision has been omitted in an asymptomatic patient and a reduction of premature beats under amiodarone. Due to an asymptomatic patient and a reduction of premature beats under amiodarone, a ventricular lead revision has been omitted.

### Summary

This case demonstrates a rare consequence of a pacemaker implantation consisting in induced ventricular premature beats. These were very probably due to an electrical irritation in the region of the right ventricular outflow tract.

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