

[CASE REPORT]

Twitching of the Pacemaker Pocket Induced by Radiofrequency Energy Delivery to the Cavotricuspid Isthmus

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Abstract:

An 82-year-old man with a permanent pacemaker (PM) implanted for sick sinus syndrome complained of palpitation due to paroxysmal atrial fibrillation and flutter. During extensive pulmonary vein isolation, the atrial lead was dislodged to the level of the tricuspid annulus. Radiofrequency energy delivery to the cavotricuspid isthmus reproducibly caused twitching of the PM pocket. The atrial lead was repositioned to the right atrial appendage, PM check revealed no functional change in the PM or lead performance. This is the first reported case of twitching of the PM pocket due to electromagnetic interference.

Key words: pacemaker, twitching, electromagnetic interference, radiofrequency

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Introduction

The safety and efficacy of catheter ablation of tachyarrhythmia in patients with permanent pacemaker (PM) implantation have been established (1). It has been reported that radiofrequency energy delivery may cause pacemaker dysfunction, such as inhibition of pacing, switching to backup mode, pacemaker mediated tachycardia, over- or undersensing, due to electromagnetic interferences (2).

We describe a case in which twitching of the PM pocket was reproducibly observed during radiofrequency energy delivery to the cavotricuspid isthmus, where the dislodged atrial lead lay.

Case Report

An 82-year-old man with a permanent PM (Medtronic, ADAPTA DR, atrial lead: CapSure FIX ventricular lead: CapSure FIX) that had been implanted 4 years earlier for sick sinus syndrome presented to our hospital with palpitation and was found to be in atrial fibrillation (AF) and flutter (AFL) (Fig. 1). Despite medical therapy, paroxysmal tachycardia occurred frequently. He was admitted to our hospital for radiofrequency ablation of refractory AF and AFL. Chest X-ray showed a dual-chamber PM with two leads: one in the right atrial appendage and one in the right ventricular apex (Fig. 2). Transthoracic echocardiography revealed no structural heart disease, no atrial or ventricular dilatation, no myocardial hypertrophy, and a preserved left ventricular function. Transesophageal echocardiography excluded an intracardiac thrombus.

After written informed consent was obtained, an electrophysiological study was performed in the postabsorptive state under light sedation and while free of antiarrhythmic agents. After internal jugular and femoral vein punctures were performed, a heparin bolus (100 U/kg) was administered, and afterward, continuous infusion of heparin was provided, maintaining an activated clotting time value between 250 and 300 seconds. Surface electrocardiogram (ECG) and bipolar endocardial electrograms were continuously monitored and stored on a computer-based digital amplifier/recorder system for an offline analysis (Bard Electrophysiology). Intracardiac electrograms were filtered from 30

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Figure 1. Twelve-lead electrocardiogram. Upper panel: sinus rhythm, Middle panel: atrial fibrillation, Lower panel: atrial flutter.

to 500 Hz.

Three long sheaths were introduced to the left atrium under intracardiac echocardiographic guidance. During simultaneous left and right pulmonary vein angiography, the tip of the atrial lead was dislodged from the right atrial appendage to the level of the tricuspid annulus (Fig. 2). The pacing mode was changed from DDD to VVI with a lower rate of 40 paces per minute without a rate response function. Extensive encircling pulmonary vein isolation was performed using the double-ring catheter method. Subsequently, ablation for atrial flutter was performed. A duodecapolar Halo catheter (Irvine Biomedical, Irvine, USA) was placed around the tricuspid annulus. A quadripolar catheter was positioned to record the His potential. An ablation catheter (Fantasista M/

L with an 8-mm-tip electrode) was introduced via the femoral vein. During constant pacing from the coronary sinus ostium, radiofrequency ablation targeted to the cavotricuspid isthmus (maximum tip temperature set at 55 °C, maximum output 40 watts) induced twitching of the PM pocket (Fig. 3), immediately stopping energy delivery within 10 seconds. After confirming the tip of the ablation catheter was separated from the dislodged atrial lead by more than 5 mm, radiofrequency ablation reproducibly induced twitching of the PM pocket.

The atrial lead was repositioned to the right atrial appendage, and PM check revealed no functional change in the PM or in the atrial or ventricular lead impedance, pacing threshold, or sensing threshold. The patient remained free of ar-

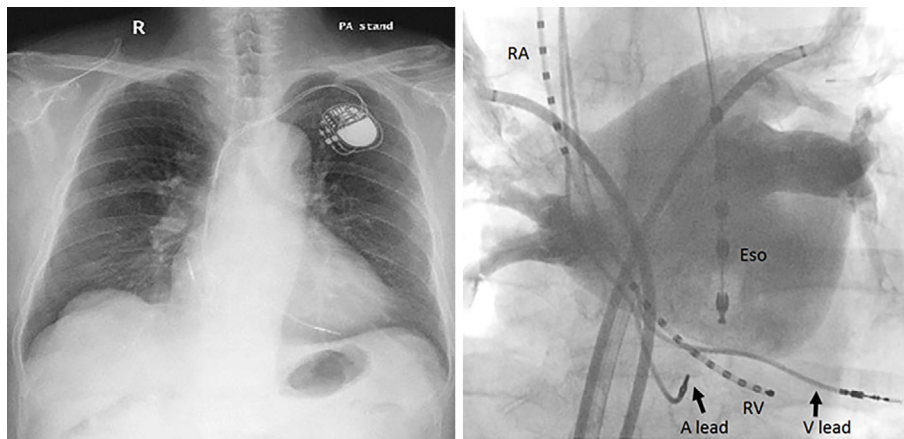


Figure 2. Chest X-ray and angiography. Left panel: Chest X-ray, Right panel: Pulmonary vein and left atrial angiography. A lead: dislodged atrial lead; Eso: tripolar electrode catheter of the esophagus, RA: decapolar electrode catheter of the right atrium, RV: decapolar electrode catheter of the right ventricle, V lead: ventricular lead

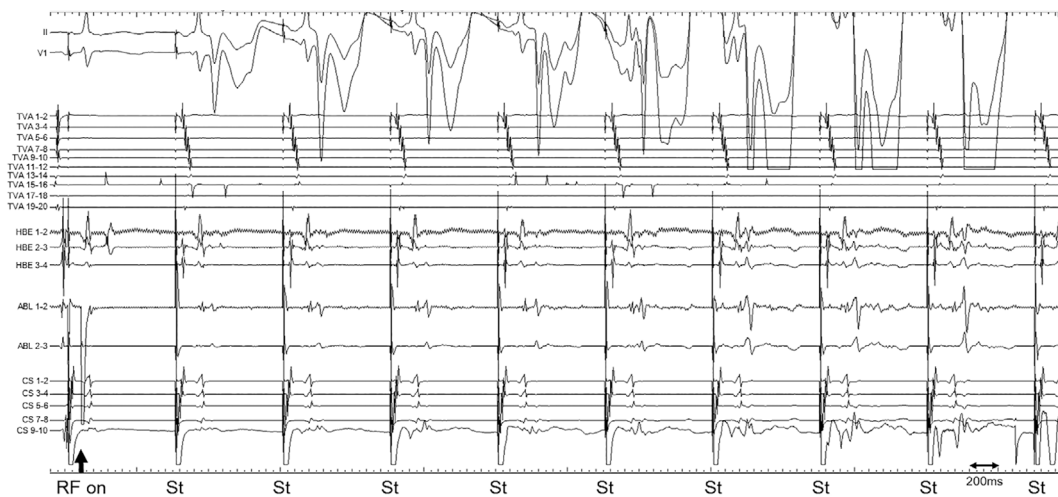


Figure 3. Intracardiac electrogram. The surface electrogram drifted due to the twitching of the pacemaker pocket during radiofrequency ablation, but the intracardiac electrograms were captured by constant stimulation with a pacing cycle length of 750 ms from the coronary sinus ostium. II, V1, surface electrocardiographic leads II, V1; ABL 1-2, distal electrogram of the ablation catheter; ABL 3-4, proximal electrogram of the ablation catheter; CS: coronary sinus electrogram, HBE: His bundle electrogram, RF: radiofrequency, St: stimulus artifact, TVA: tricuspid valve annulus electrogram

rhythmias over a 12-month follow-up period.

Discussion

In this case, radiofrequency ablation to the cavotricuspid isthmus near the dislodged atrial lead reproducibly caused twitching of the PM pocket. This phenomenon was considered to be due to electromagnetic interference.

It has been reported that catheter ablation for patients with permanent PM may cause electromagnetic interference, including PM inhibition, untoggled backup mode, pacemaker tachycardia, oversensing, and transient loss of ventricular capture (2-5). Such interference produced by radiofrequency current has been shown to be transient, and most

devices automatically toggled back to their full function, so catheter ablation for patients with permanent PM is considered safe. In our case, a PM check before and after ablation revealed no functional change in the PM or lead performance.

Extracardiac stimulation, such as in the PM pocket due to radiofrequency (RF) ablation, is very unusual, and the present case is the first one reported in the literature (according to a PubMed search). The mechanism underlying the twitching of the PM pocket is unknown. We speculate the mechanism of this phenomenon to be as follows: i) the radiofrequency current interfered with the tip of the atrial lead which then became dislodged around the tricuspid annulus, ii) the electric current was conducted through the atrial lead

in a retrograde direction to the PM can, iii) the electric current passed through the can due to the Zener diode function and thus caused the stimulation of the left greater pectoral muscle. The Zener diode is a semiconductor that is designed to shunt unusually large currents away from the PM circuitry and protect it in the event of PM malfunction (6). In our case, radiofrequency energy delivery reproducibly induced twitching of the PM pocket. It is important to check the PM function and the lead performances to prevent further complications, such as PM malfunction, pacing failure, and ablation of the right ventricular muscle where the ventricular lead is placed. If the atrial lead had not been dislodged from the right atrial appendage in our case, twitching of the PM pocket might not have occurred.

Conclusion

We reported a rare case of twitching of the PM pocket during radiofrequency ablation due to electromagnetic interference.

The authors state that they have no Conflict of Interest (COI).

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