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Case Report

Unique Interaction Between an Atrial Single-Chamber Pacemaker and a Ventricular Defibrillator

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Abstract. **A well described interaction between an antibradycardia pacemaker and a ventricular defibrillator is sensing of pacemaker stimuli by the ventricular defibrillator. This report describes an interaction between an atrial demand pacemaker and a ventricular defibrillator that resulted in ventricular asystole and polymorphic ventricular tachycardia. In this case, the ventricular defibrillator sensed atrial pacing stimuli when complete atrioventricular block with a slow ventricular escape rate developed. Defibrillator-based ventricular demand pacing was inhibited, resulting in prolonged periods of ventricular asystole, polymorphic ventricular tachycardia, and multiple defibrillator shocks. Ventricular defibrillator sensing of atrial pacemaker stimuli in the setting of complete atrioventricular block and ventricular asystole cannot be simulated during defibrillator implantation when atrioventricular conduction is intact. Therefore, a pacemaker programmed to atrial demand pacing in a patient with a ventricular defibrillator can result in inappropriate inhibition of ventricular pacing in the setting of complete heart block. Furthermore, this interaction can be avoided with a dual-chamber pacing ventricular defibrillator.**

Key Words. **complete atrioventricular block, pacemaker–defibrillator interaction, ventricular fibrillation**

Introduction

In the era of ventricular defibrillators with rate-responsive dual-chamber pacing, implantation of a single chamber ventricular defibrillator might still be considered in a patient with a preexisting rate-responsive pacemaker system or in a patient in whom battery longevity is a critical issue. Numerous adverse interactions in patients with a pacemaker and a ventricular defibrillator have been described [1–3]. This report describes an interaction between an atrial demand pacemaker and a ventricular defibrillator that occurred in a patient in whom complete atrioventricular block with prolonged periods of ventricular asystole developed and resulted in sustained polymorphic ventricular tachycardia.

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Case Report

A 73 year old woman was admitted to an outside hospital with a chief complaint of multiple ventricular defibrillator discharges. The patient had a past medical history of coronary artery disease, myocardial infarction, diabetes mellitus, rheumatic disease of the mitral and tricuspid valves, congestive heart failure, and a left ventricular ejection fraction of 0.35. Eight years prior to admission an epicardial ventricular defibrillator system was implanted for syncope and ventricular tachycardia. Two weeks later, she developed sinus bradycardia, long-short sequences, and polymorphic ventricular tachycardia which resulted in multiple defibrillator shocks. The patient was treated with an atrial demand pacemaker. Four years later, the ventricular defibrillator and the pacemaker systems became infected and were explanted. After an appropriate course of antibiotic therapy, a defibrillator lead (Guidant, St. Paul MN, model 0072) was positioned in the patient's right ventricular apex and attached to a ventricular defibrillator pulse generator (Guidant, model 1746). An atrial pacemaker lead (Guidant, model 4269) was positioned in the right atrial appendage and connected to a bipolar SSI pacemaker pulse generator (Guidant, model 446). Intraoperatively, sensing of the atrial pacemaker stimulus by the ventricular defibril-

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lator was observed at an amplitude of 3.75 V with a pulse width of 0.4 ms. The atrial pacemaker stimulus was not sensed at an amplitude of 2.5 V and a pulse width of 0.6 ms. The ventricular defibrillator sensed ventricular fibrillation when the pacemaker was programmed to asynchronous atrial pacing with an amplitude of 2.5 V and a pulse width of 0.6 ms. The pacemaker was chronically programmed to the AAI mode with an amplitude of 2.5 V and a pulse width of 0.6 ms. Amiodarone at a daily dosage of 200 mg was initiated subsequently for atrial fibrillation.

Interrogation of the patient's ventricular defibrillator upon presentation to the University of Michigan Medical Center demonstrated 19 episodes of polymorphic ventricular tachycardia, 12 of which were sustained and resulted in shocks. During AAI pacing at 85 beats per minute, the patient developed complete atrioventricular block (Fig. 1). The ventricular defibrillator sensed pacing stimuli from the atrial pacemaker which inhibited VVI pacing. Long-short sequences occurred, and polymorphic ventricular tachycardia resulted. Normal post-shock VVI pacing was observed (Fig. 1). However, after non-sustained episodes of polymorphic ventricular tachycardia with diverted shocks, the ventricular defibrillator sensed the atrial pacing stimuli, defibrillator-based VVI pacing was inhibited, and ventricular asystole resulted (Fig. 2).

Programmed settings for the atrial pacemaker were AAI pacing at 85 beats per minute with an output amplitude of 2.5 V with a pulse width of 0.6 ms. The pacemaker was immediately deactivated and the ventricular defibrillator's VVI pacing rate was increased to 85 beats per minute. Amiodarone, desethylamiodarone and digoxin concentrations were 1.1 μg/ml, 0.9 μg/ml, and 1.7 ng/ml, respectively. Within hours of discontinuing amiodarone and digoxin, atrioventricular conduction resumed. The patient received a DDDR ventricular defibrillator 8 days later without complication.

Discussion

Main Finding

Oversensing of atrial pacemaker stimuli by a ventricular defibrillator, with subsequent inhibition of defibrillator-based VVI pacing in the setting of atrioventricular block, has not been

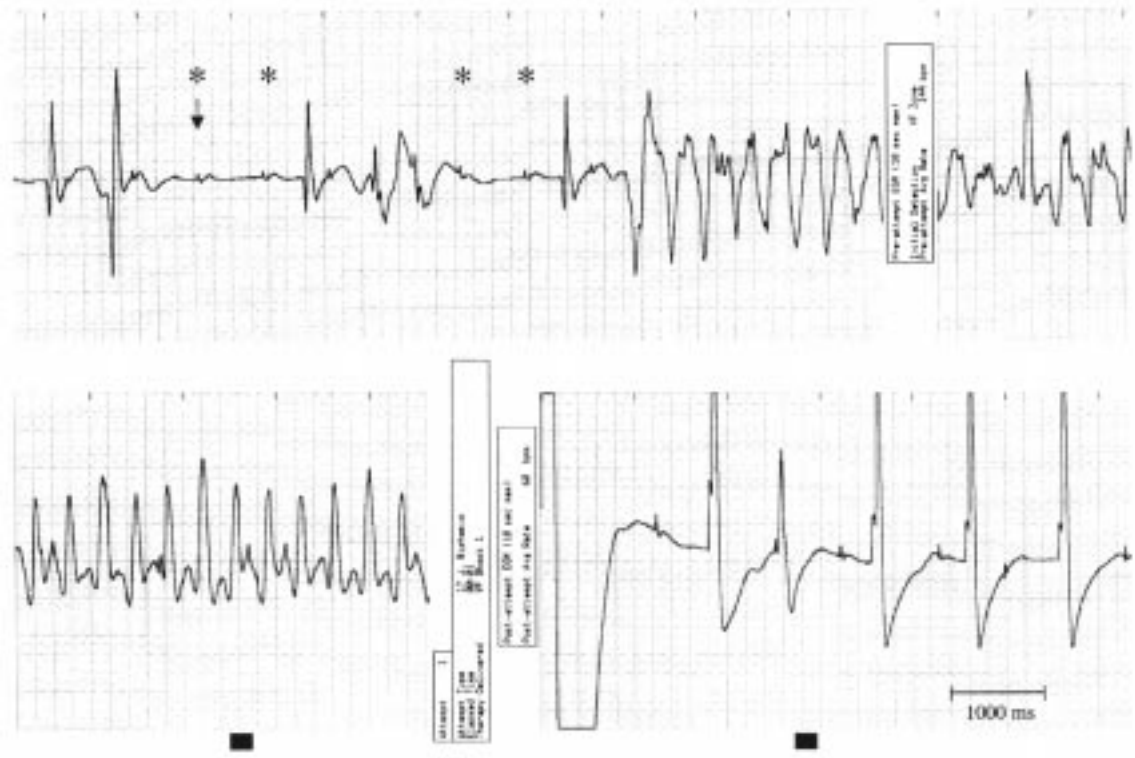

Fig. 1. *This intracardiac electrogram from the ventricular defibrillator demonstrates atrial pacing and capture (arrow) in the setting of complete atrioventricular block. Defibrillator-based VVI pacing is inhibited due to sensing of the atrial pacemaker stimuli (*). A long-short sequence results in polymorphic ventricular tachycardia. A 17 joule shock terminated the tachycardia. Normal post-shock defibrillator-based VVI pacing at 60 beats per minute occurs. (Note that ventricular paced beats have no visible pacing artifacts.)*

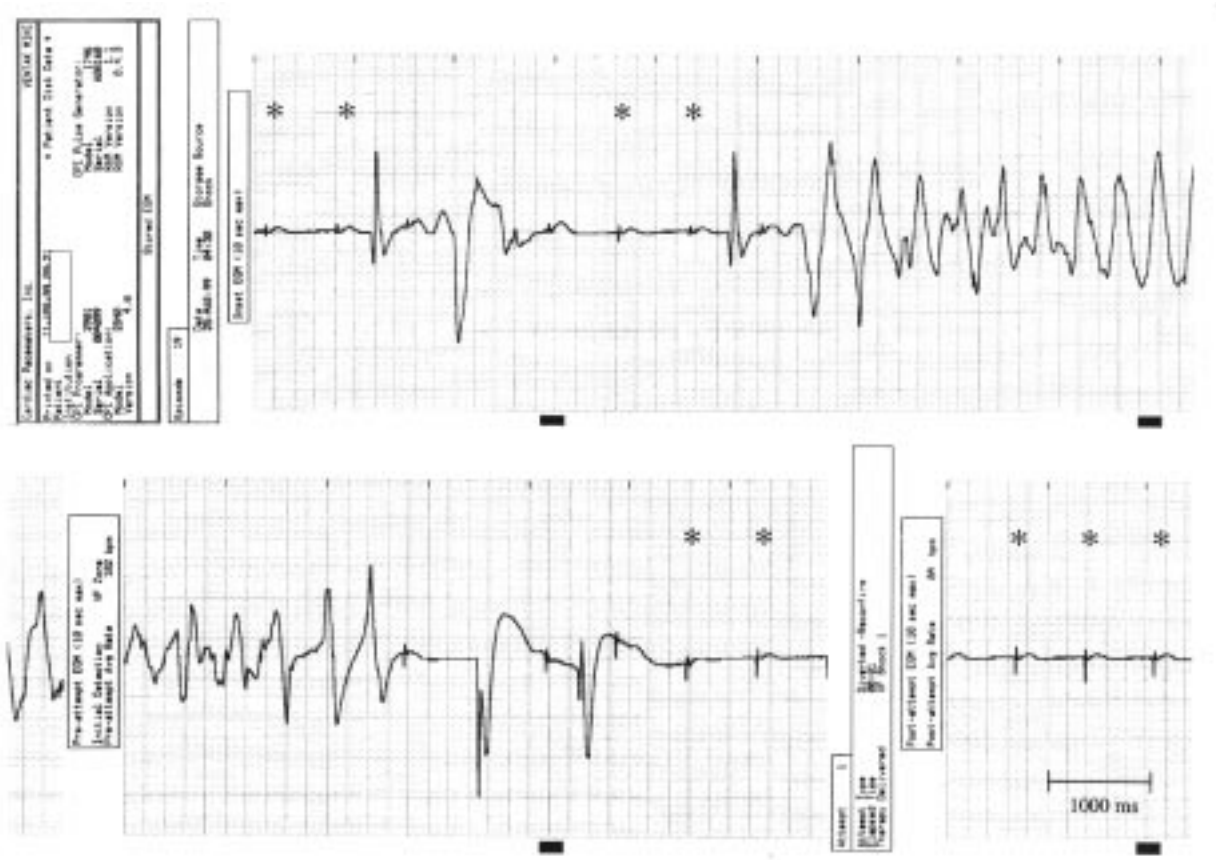

Fig. 2. *An intracardiac electrogram showing a nonsustained episode of polymorphic ventricular tachycardia. The defibrillator senses the atrial pacemaker stimuli (*), defibrillator-based VVI pacing is inhibited, long-short sequences occur, and nonsustained polymorphic ventricular tachycardia results. Atrial pacing stimuli are sensed following the spontaneous termination of the polymorphic ventricular tachycardia, defibrillator-based VVI pacing is inhibited, and ventricular asystole results.*

described as a pacemaker–defibrillator interaction.

Mechanism

The pacemaker–defibrillator interaction described herein is most likely due to the defibrillator's capability to automatically adjust sensitivity. These sensing amplifiers and algorithms differ among different companies. In our case, after a sensed event, the defibrillator automatically adjusted the sensitivity to 75% of the amplitude of the sensed signal. After the ventricular refractory period, the sensitivity doubled every 156 ms until either a signal was sensed or the maximum sensitivity was reached. In this patient, an atrial pacemaker stimulus was not sensed by the defibrillator immediately after the onset of complete atrioventricular block. Without sensed ventricular events, the defibrillator's sensitivity increased such that the next atrial pacing stimulus was sensed. Since the ventricular escape interval was programmed to 1500 ms, which was much longer than the atrial escape interval of 706 ms, defibrillator-based ventricular demand pacing was continuously inhibited. This resulted in severe bradycardia and polymorphic ventricular tachycardia. Post-shock VVI pacing, in contrast, occurred with a fixed sensitivity of 2.5 mV, which was inadequate for sensing atrial pacing stimuli. However, after a nonsustained episode of polymorphic ventricular tachycardia, the sensitivity remained at maximum, the atrial stimuli were sensed, and defibrillator-based VVI pacing was inhibited.

Previous Studies

Numerous reports indicate that implantation of a defibrillator and a separate pacemaker is safe if intraoperative testing fails to demonstrate oversensing or undersensing [4–6]. For instance, sensing of a pacemaker stimulus during ventricular fibrillation may lead to inhibition of ventricular fibrillation therapy [1]. Conversely, sensing of the pacemaker stimulus and the associated evoked potential may lead to inappropriate shocks [7–9].

Clinical Implications

Based on this case report, previously described pacemaker-defibrillator interactions can be safely excluded during intraoperative testing only if the pacemaker is programmed to a mode that incorporates ventricular sensing. As many as 12% of patients with normal AV-conduction at the time of AAI pacemaker implantation will develop second or third degree AV block [10]. This report highlights the potential for serious device interactions in patients with a defibrillator and pacemaker-based AAI pacing. This interaction can be avoided with a dual-chamber pacing ventricular defibrillator.

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