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# What is the rhythm: ventricular or supraventricular?

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### PACEMAKER/ICD PROBLEM OF THE MONTH

# What is the rhythm: Ventricular or supraventricular?

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#### **Case summary**

A 45-year-old man with hypertrophic cardiomyopathy and syncope underwent implantation of a dual-chamber internal cardioverter-defibrillator (ICD; Marquis DR 7274, Medtronic, Inc., Minneapolis, MN, USA). A follow-up office interrogation revealed an episode of monomorphic ventricular tachycardia, which was terminated by antitachycardia pacing (ATP) therapy (Figure 1). Six months after implantation, the patient presented to the emergency department with multiple ICD discharges (Figure 2). What is the mechanism of the arrhythmia?

#### Commentary

Figure 1 shows that the patient had a sustained ventricular tachycardia (VT) that was terminated by ATP therapy to normal sinus rhythm (NSR). Note 2:1 ventriculoatrial (VA) conduction at the end of ATP therapy and the differences in ventricular electrogram (EGM) between VT and NSR. Figure 2 shows long RP tachycardia (cycle length 350 ms) with 1:1 AV or VA association. The differential diagnosis include (1) VT (note that the ventricular local EGM in Figure 2 is different than that of NSR in Figure 1) with retrograde 1:1 VA conduction; (2) any supraventricular rhythm with aberrancy; or (3) isorhythmic double tachycardia. Given that atrial and ventricular rates are exactly the same before and after ATP therapy, even after perturbation of the atrium (1:1 VA conduction at the end after initial VA dissociation during ATP), isorhythmic double tachycardia is very unlikely. At the end of the ATP therapy (arrow), there is atrial capture at the rate of ventricular pacing cycle length (300 ms). After cessation of ATP therapy, a "VAAV response" is noted.

This phenomenon is noted repeatedly in multiple other stored EGMs with the same recovery time (440 ms) after cessation of ATP therapy, suggesting that the atrium is driving the ventricle rather than the other way around. Again, AV dissociation during ATP (before the arrow),

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maintenance of the exact atrial rate (350 ms) before and after ATP, and VA wobble after ATP therapy suggest that it is unlikely that V is driving A. Bypass tract-mediated tachycardia or atypical AV nodal reentrant tachycardia can be ruled out based on the facts that there is VA dissociation during the initial portion of ATP therapy and that there is "VAAV response" after cessation of ATP therapy. One may question the short AV delay in the first atrial and ventricular beat after termination of ATP therapy as qualifying for a "VAAV response." This short AV delay can be explained by distant location of the atrial electrode from the atrial focus of arrhythmia and the fully recovered AV node (710 ms), which allows rapid conduction down the AV node. After ATP therapy, due to decremental conduction of the AV node, the AV delay becomes progressively longer to a final constant AV delay. The other unlikely possibility is that the first ventricular beat after ATP therapy is a junctional beat (same ventricular EGM as the others), which had to occur repeatedly as noted in other multiple ATP therapies.

Therefore, we are left with atrial tachycardia/flutter or sinus tachycardia. Additional clinical history revealed that the patient was chasing his runaway dog when he experienced multiple ICD discharges. The patient did not have a history of atrial arrhythmia. Figure 3 shows that both ventricular and atrial rates are below the set 370-ms detection rate for VT, and the rhythm continued even though the ICD had exhausted therapies.

This is an example of inappropriate ICD discharges due to sinus tachycardia. Interestingly, the ventricular EGM during sinus tachycardia shown in Figure 2 is different than that of NSR in Figure 1. This is due either to sinus tachycardia with aberrancy or to change of local ventricular EGM polarities after multiple shock therapies from the previous episode. Indeed, the ventricular EGM during sinus tachycardia from the previous episode (before shock therapy was delivered) had the same ventricular EGM as recorded in NSR.

Inappropriate ICD discharges are unacceptably common, ranging from 15% to 20%.<sup>2</sup> They occurred in the index patient even though all the PR logic parameters were set to "on." This patient did have a documented VT (cycle length 310–340 ms) that was terminated by ATP therapy. There-

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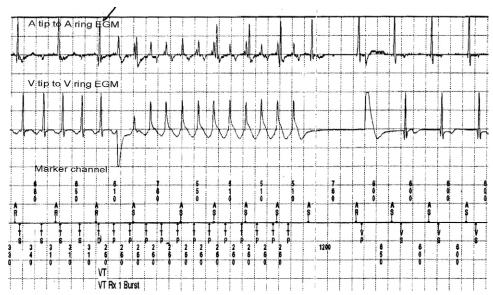


Figure 1 Stored electrogram from the patient's ICD during a routine follow up. EGM = electrogram.

fore, to prevent inappropriate ICD discharges but still treat VT in this patient, we increased his beta-blocker dose and change the VT zone from 300 to 370 ms with ATP therapy

without any shock therapy. For fast VT via the ventricular fibrillation zone from 260 to 300 ms, ATP therapy followed by shock therapies was programmed. For the ventricular

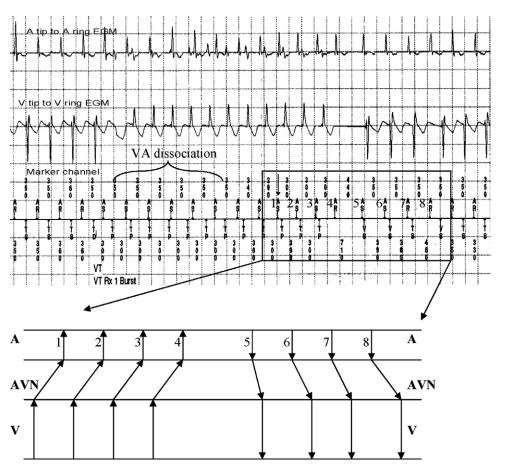


Figure 2 Stored electrogram of the most recent episode and the ladder diagram of key beats during and after ATP therapy.

ICD Model: Marquis DR 7274 Serial Number: PKC133591H 9966 Software Version 4.0 Copyright Medtronic, Inc. 2001

## VT/VF Episode #9 Report

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ID#	Date/Time	Туре	V. Cycle	Last Rx	Success	<b>Duration</b>
9	Mar 26 09:27:55	VT	350 ms	VT Rx 6	No	1.6 min

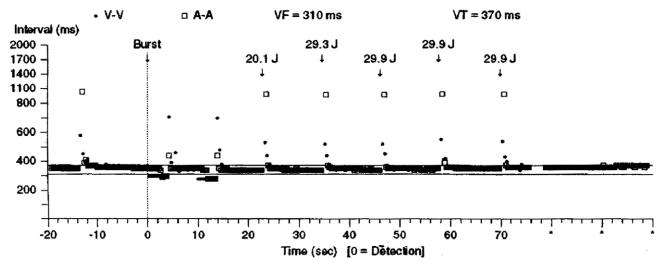


Figure 3 Episode summary.

fibrillation zone <260 ms (230 bpm), only shock therapies were programmed.

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