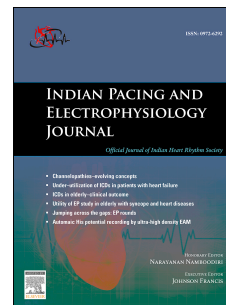


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Sahra Jama, Benedict M. Wiles, Magdi M. Saba, Michael C. Waight, Anthony C. Li



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Ventricular arrhythmia during automated lead testing: what is the mechanism?

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CASE PRESENTATION

A 49-year-old female with cardiac sarcoidosis, previous septal ablation for ventricular tachycardia, severe LV systolic dysfunction (EF 20%), complete heart block and a secondary prevention CRT-D (Brava Quad, Medtronic, apical ICD lead) presented with syncope four weeks after an elective generator change for battery depletion.

Sarcoidosis had been diagnosed a decade earlier, after an initial presentation with uveitis and arthralgia complicated by a ventricular fibrillation cardiac arrest. Scarring of the basal septum had been demonstrated on PET-CT and during invasive mapping, and the patient had previously responded to CRT therapy, both clinically and by echocardiographic parameters. There was a history of shock therapy for ventricular tachycardia, but not since her successful ablation several years earlier.

Interrogation of her CRT-D device revealed a tachycardia episode for which shock therapy had been delivered. The electrograms from this episode are shown in Figure 1 and device settings at the time of the event are shown in Figure 2. Since the generator change normal lead parameters and 100% biventricular pacing had been noted, with no additional arrhythmia episodes.

Importantly, ventricular fibrillation requiring emergency external defibrillation had occurred during the recent generator change, which was undertaken with continuous ECG monitoring. To facilitate lead transfer during this procedure the biventricular pacing rate had been very gradually reduced to 40bpm to encourage intrinsic rhythm. When no underlying rhythm was detected, pacing had been reprogrammed to RV only. A 7-lead ECG showing the final beat of biventricular pacing is shown in Figure 1 (top panel). The episode of ventricular fibrillation, which occurred within 30 seconds of cessation of biventricular pacing, is also shown (bottom panel). Post defibrillation full recovery ensued and after a period of observation, during which there was no further ventricular ectopy, the patient was discharged.

What is the most likely cause of the tachycardia episode that resulted in shock therapy? What programming alteration, made after the generator change, may have prevented the second event from occurring?

COMMENTARY

The EGM in Figure 1 shows an appropriately sensed episode of ventricular fibrillation, which appears to have been triggered by ventricular ectopics. The ectopics are denoted by sensed ventricular events (Vs) on the marker channel and have been further highlighted by annotation with a star. 'Capture Management' can also be seen at the start of the marker channel. This informs us that an automated lead threshold test is being performed during arrhythmia onset. The device settings, shown in Figure 2, confirm that it is the atrial lead that is being tested, as capture management was turned 'off' on both ventricular leads. The start of the algorithm is annotated with an arrow. From this point forward the the 'Vp' events on the marker channel become RV-only paced beats as in Medtronic CRT devices, biventricular pacing is automatically switched to RV-only pacing during atrial threshold testing. Where atrial capture management is turned on, this is a non-programmable system function.¹

The ECG in Figure 3 shows RV-only paced complexes followed by polymorphic ventricular ectopics with significant fractionation. Within a short time period, these ectopics result in triggered ventricular fibrillation, with R on T phenomenon occurring after a short-long-short sequence. The ectopics appear immediately after cessation of biventricular pacing and were not observed during biventricular pacing, even with extended ECG monitoring post procedure monitoring. Ectopic activity was also not observed during follow up, where 100% biventricular pacing was achieved.

Ventricular fibrillation requiring shock therapy has therefore occurred on two separate occasions, both of which correspond to transient periods during which biventricular pacing was replaced by RV-only pacing. We therefore deduce that RV-only pacing is pro-arrhythmic in this individual. Switching automated threshold testing 'off' on all of the leads, immediately after the generator change, may therefore have prevented the second event. Unfortunately, it is not widely known that automated atrial threshold testing results in a temporary cessation of biventricular pacing.

Fortunately, the overall incidence of single chamber ventricular pacing resulting in ventricular dysrhythmia is low.^{2,3} We believe that patients at greater risk of this phenomena would include those with myocardial scar in close proximity to the RV pacing lead, either due to myocardial pathology of prior ventricular ablation, and patients in whom events like this have been previously documented. In such individuals, careful device programming is required, as is a comprehensive understanding of automated algorithms that might inadvertently result in RV-only pacing.

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FIGURE LEGEND**Figure 1**

The onset of an arrhythmia episode downloaded from a Medtronic Bravia CRT-D. The bottom panel immediately follows the top chronologically. The atrial EGMs, ventricular EGMs and marker channels (for both the top and bottom panels) are identified on the left side of the top panel. The following additional annotations have been added to aid understanding: 'Arrow' – onset of 'Capture Management' (atrial) algorithm, 'Star' - sensed ventricular events (triggered ectopics), 'Opaque Star' the first beat of device detected tachycardia.

Figure 2

Device settings showing how the device was programmed after the generator change

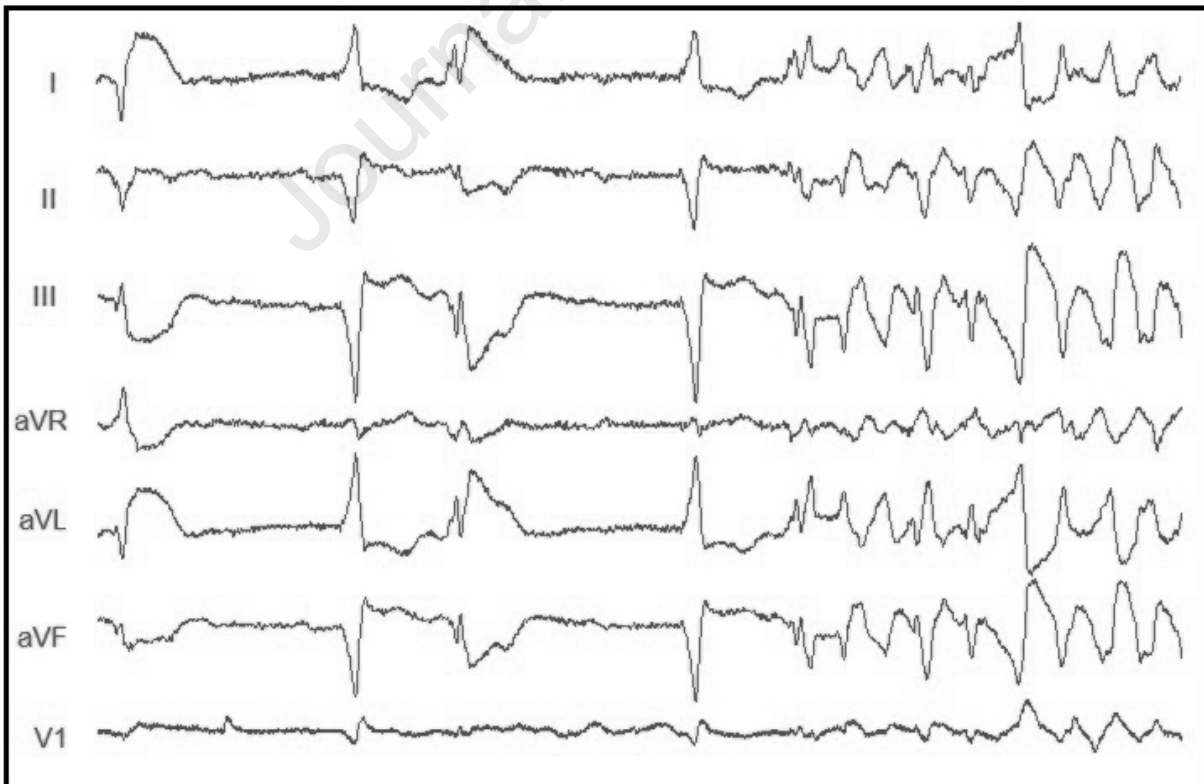
Figure 3

7 lead ECG recorded during the generator change procedure. The top and bottom panels are not continuous chronologically, as the bottom panel was recorded around 20 seconds after the top panel

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Pacing Summary					
Mode		Rates		CRT	
Mode	DDD	Lower	80 bpm	V. Pacing	LV->RV
Mode Switch	171 bpm	Upper Track	140 bpm	V-V Pace Delay	0 ms
		Upper Sensor	120 bpm	Paced AV	120 ms
				Sensed AV	100 ms
Pacing Details		Atrial	RV	LV	
Amplitude	2.00 V	2.00 V	1.50 V		
Pulse Width	0.40 ms	0.40 ms	0.40 ms		
Capture Management	On	Off	Off		
Sensitivity	0.30 mV	0.30 mV			
Pace Polarity	Bipolar	Bipolar	LV1 to LV2		
			Tip	LV1	LV2
				LV3	LV4
Sense Polarity	Bipolar	Bipolar			



Conflict of Interest:

No funding was received for this manuscript. The authors would like to declare the following conflicts of interest; MW and BW have received funding from the Advanced Ventricular Arrhythmia Training and Research Program administered by St. George's Hospital Charity. BW has received unrestricted fellowship funding and consultancy payments from Boston Scientific. AL and MS have received unrestricted grants from Abbott Labs.

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