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Atrial fibrillation in a dual chamber ICD recipient with activation of the ventricular intrinsic preference algorithm: what is the mechanism?

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Case presentation

A 72-year-old man with ischemic cardiomyopathy and sinus node dysfunction was implanted with a dual chamber implantable cardioverter defibrillator (ICD) (St Jude Medical Ellipse DR 2277-36, St. Paul, MN, USA) for secondary prevention. The programmed parameters were as follows: DDDR 50-130 bpm, paced atrioventricular (AV) delay (AVD) = 150 ms, atrial pacing threshold = 1 V, atrial detection = 2.5 mV, ventricular pacing threshold = 0.75 V, ventricular detection = 12 mV, post-ventricular atrial refractory period (PVARP) = 275 ms. The “Ventricular Intrinsic Preference” (VIP) mode was programmed with the following settings, VIP extension: 200ms, search interval: 30sec, search cycles: 3 beats. Ten months after implantation, patient was asymptomatic. Atrial and ventricular pacing burdens were 73% and <1%, respectively. No VT episodes occurred during follow-up, but device interrogation revealed 24 episodes of atrial fibrillation (AF), as shown in Figure 1. What is the mechanism of AF initiation? How could you reprogram the device to avoid these AF episodes?

Commentary

Many studies demonstrated that right ventricular (RV) pacing can induce dyssynchronous left ventricular contraction and precipitate left ventricular dysfunction, resulting in adverse outcomes such as AF or heart failure.[1] Few strategies can reduce RV pacing burden in dual chamber systems, i.e. programming DDD pacing with a fixed long AV delay, AV hysteresis or specific AAI-DDD mode switch algorithms.

All device brands developed their own mode switch algorithm, most of them based on an AAI pacing mode while monitoring AV conduction. The pacemaker switches to DDD pacing mode in case of persistent loss of AV conduction, while returning to AAI mode if AV

conduction resumes. One of the major drawbacks of such algorithms is that long AV delays are permitted, leading to AV dyssynchrony, diastolic mitral regurgitation, or benign arrhythmias such as AV nodal reentrant tachycardia as previously described.[2] Non-conducted atrial sensing/pacing events may also occur without mode switching to DDD mode, resulting in short-long-short ventricular sequences. Such short-long-short cycles have been described to infrequently trigger malignant ventricular arrhythmias in patients with cardiomyopathies. [3-4]

The VIP algorithm functioning is different since non-conducted atrial events are not tolerated permitted and the algorithm works like a periodic AV-delay hysteresis. Consequently, every single atrial event will be followed by a ventricular sensed or paced beat and short-long-short ventricular cycles cannot happen. Three parameters have to be programmed, namely the “VIP extension”, the “search interval” and the “search cycles”. Periodically (“search interval”, from 30 sec to 30 min), the device extends the programmed AV interval by the “VIP extension” (from 50 to 200 ms) for a determined number of beats (“search cycles”, 1, 2 or 3 consecutive beats). When an intrinsic R wave is sensed by the device within the AV delay + VIP extension, spontaneous ventricular activations occurs and the device functions as a single chamber atrial pacemaker. When no intrinsic R wave is sensed within the AV delay + VIP extension, the AV delay shortens after the programmed “search cycles” to its original programmed value. Consequently, the device is designed to sustain long AV delays, which may favor retrograde VA conduction and initiate a pacemaker-mediated tachycardia (PMT), as previously described. [5]

Initially, as shown in figure 2, the atrium is paced since the rate response mode is activated (SIR = Sensor indicated rate), with intrinsic ventricular activation occurring after a long AV delay (mark 1, Figure 2). The third ventricular beat is paced since no intrinsic R wave was detected within the AV delay + VIP extension (mark 2, Figure 2). One can appreciate that

after this paced beat, a retrograde atrial conduction occurs, detected by the device but not taken into account, since it occurs in the PVARP. An AV delay is not triggered after this atrial event and the device continues to inadvertently and ineffectively pace the atrium. After the third “VP” with long AV delay (= programmed AV delay + VIP extension), the AV delay returns to its original programmed value (i.e. 150 ms, mark 3, Figure 2), still responsible for a clear retrograde atrial conduction (mark 4, Figure 2). At this time, a “pseudo-PMT” is triggered, the ventricular pacing favoring retrograde VA conduction, which constantly occurs during the PVARP, and the device continues to inadvertently and ineffectively pace the atrium (since the atrial pacing spike occurs during the absolute refractory period of the atrium) which perpetuate the phenomenon. Progressively, the atrial pacing rate slowly decreases (up to 707 ms), and at one given time (mark 5, figure 2), the atrial pacing spike captures the atria, probably during the vulnerable window, which consequently triggers AF (mark 6, Figure 2), activating the device mode switching (“CAM”).

In our case, we describe for the first time the occurrence of episodes of AF initiated by the VIP algorithm activation. Long AV delay favored retrograde VA conduction, initially occurring during the PVARP, and not triggering an AV delay. Consequently, the device continued to inadvertently pace the atrium without capturing it, the atrium being in absolute refractory period. Once the pacing spike occurred in the vulnerable window of the refractory period and captured the atrium, an episode of AF occurred. In our patient, reprogramming a shorter VIP extension was sufficient to avoid retrograde VA conduction, while maintaining spontaneous ventricular activation without significant increase in ventricular pacing burden. One may assume that shortening the PVARP could have avoided AF episodes, since the phenomenon would have generated a PMT, detected and stopped by the device after the ninth “AS-VP” at maximal tracking rate.

To note, if one looks closely at the ventricular EGMs during the “pseudo-PMT”, there is variation in the “Vp” complexes morphology: VP#4 and Vp#12 and Vp#13 (first two complexes of the bottom strip of Figure 1) appear to be fully paced while Vp#5-11 appear to be different and may represent varying levels of fusion. Therefore, it cannot be excluded that this may represent a form of atrio-ventricular nodal reentrant tachycardia (AVNRT, slow-fast or slow-intermediate) that would have persisted even in the absence of ventricular pacing. However, spontaneous AVNRT had never been observed in this patient, and this hypothesis, although theoretically possible, is unlikely.

In summary, we describe here a new pro-arrhythmic effect of the VIP algorithm, generating AF in a patient with no history of atrial arrhythmias. Clinicians should be aware of such potential events, since the reprogramming of the device may prevent new episodes and avoid the initiation of otherwise unnecessary anticoagulation.

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Figure legends:

Figure 1: Initiation of an AF episode. From top to bottom: atrial EGM, ventricular EGM, markers and delays (AA, VV, and AV delays, respectively).

Ap: Atrial pacing; As: Atrial sensing; CAM: Automatic mode switching; EGM: Electrograms; SIR: Sensor indicated rate; VIP: ventricular intrinsic preference; Vp: ventricular pacing; Vs: Ventricular sensing

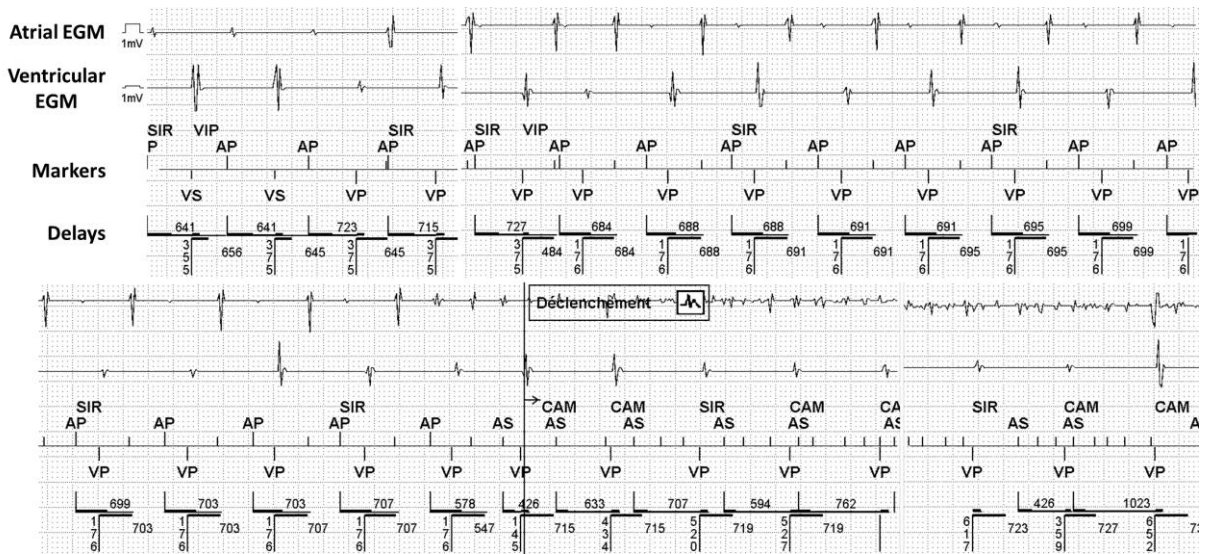


Figure 2: See text for details.

