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Recurrent shocks in a dual chamber implantable cardioverter defibrillator: Making sense of the chaos

Colin Yeo MBBS[1] | Girish M. Nair MBBS[2] | Martin Green MD[2]

[1]Department of Cardiology, Changi General Hospital, Singapore, Singapore
[2]Arrhythmia Service, Division of Cardiology, University of Ottawa Heart Institute, Ottawa, Canada

Correspondence: Colin Yeo, Changi General Hospital, Department of Cardiology, 2 Simei Street 3, Singapore 529889, Singapore (drcolinyeo@gmail.com)

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1 | CASE

A 92-year-old gentleman presented with sensation of thumping in the chest. Carelink transmission revealed that he received 15 shocks from his Medtronic Protecta XT DR implantable cardiac defibrillator (ICD) in a 24-hour period. He had ischemic cardiomyopathy and received a dual chamber ICD 12 years ago after an episode of monomorphic ventricular tachycardia (VT). Amiodarone was discontinued due to interstitial lung disease and since then he had been on metoprolol 50 mg BID. Baseline ECG showed normal sinus rhythm.

Subject had previous VT episodes which were terminated with anti-tachycardia pacing (ATP). He had not had a shock from his device over the past 12 months. Figure 1 showed the interval plot from the recorded event on the ICD. Electrograms of all 15 shocks were similar. What was the cause of his symptoms and how would you manage the patient?

2 | COMMENTARY

2.1 | Interpreting the scatterplot

The *Y* axis of Figure 1 demonstrated cycle length intervals in milliseconds (ms) and *X* axis as time in seconds (sec). Onset of the detected tachycardia was marked as 0 sec. There were three horizonal lines at 280, 320, and 530 ms, which correlated with the programmed ventricular fibrillation (VF), fast ventricular tachycardia (FVT), and VT zones.

The interval plot of the episode did not show the onset, but the subject was already in tachycardia during this recording. The solid dots, which represented the ventricular tachycardia cycle length (TCL), measuring 480 ms. It was logged within the VT zone. There was gradual acceleration of TCL peaking at 450 ms, followed by gradual deceleration, then shock. There was a time gap on the interval plot at 82 sec, annotated as by an asterix (*). The gradual shortening and lengthening of the VV intervals suggested enhanced automaticity with autonomic modulation, as the mechanism of the tachycardia. This contrasted with the usual pattern in macro re-entrant VT that usually demonstrate as a stable TCL.

White squares, representing atrial cycle length (AA interval) were tied with the VV cycle length for much of the time. However, there were two groups of AA intervals which appeared to be multiples of the VV TCL 480 ms. Morphologically, it was reminiscent of a rail road track and sometimes described as the "tram-track sign".[1] The pattern in which the AA intervals fell into two discrete TCL groups, in multiples of the ventricular TCL was suggestive of undersensing in the atrial channel, with the AA cycle length clearly linked to the VV cycle length. This suggested 1 to 1 relationship between atrium and ventricles during tachycardia.

Subject received eight RAMP+ ATP and three shocks before tachycardia was recorded as terminated by ICD. ATP episodes were denoted by sudden shortening of VV TCL where most of the VV intervals of the ATP fell in the FVT zone. During ATP, the VV intervals shortened, but AA intervals remained constant, demonstrating persistence of tachycardia despite dissociating the A from the V. This observation excluded atrioventricular tachycardia (AVRT). It was less likely VT with 1 to 1 retrograde conduction to atrium as it would be highly unlikely that atrial TCL remains unperturbed during ventricular overdrive pacing. There was no evidence of entrainment or resetting of the A from the V during ATP. At the end of each ATP, the subsequent three VV intervals were markedly slower than 480 ms, demonstrating absence of entrainment. These reproducible observations (8 times in all) helped with the conclusion that this tachycardia was highly unlikely to be macro re-entrant VT, AVRT, or atrioventricular nodal re-entrant tachycardia (AVNRT). These

observations would narrow our differential diagnosis to that of atrial tachycardia (AT) or sinus tachycardia (ST).

2.2 | Interpreting the intracardiac electrocardiogram

Figure 2 showed intracardiac electrocardiograms (EGM) from the right atrium and right ventricular leads. In the first row, marker channels were denoted by "AS" which represented sensed atrial activity by the device. However, in the same first row, there was loss of "AS" when the amplitude of the atrial EGM became smaller. This corresponded with the interval plot in Figure 1 when AA TCL suddenly increased from 490 to 1480 ms, confirming atrial undersensing. This intermittent loss of atrial sensing was repeated in the 2nd row and during the rest of the tachycardia event.

At the bottom of both rows in Figure 2, marker channel annotation "ST" indicated that the device algorithm initially diagnosed the tachycardia as sinus tachycardia. Unfortunately, later in the tachycardia, there were 16 consecutive intervals in the programmed VT zone

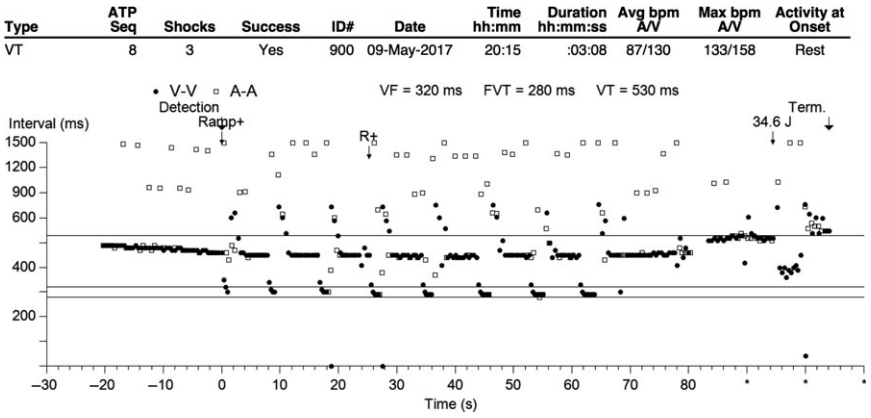

FIGURE 1 Interval plot tracing from the recorded event on the implatable cardioverter defibrillator

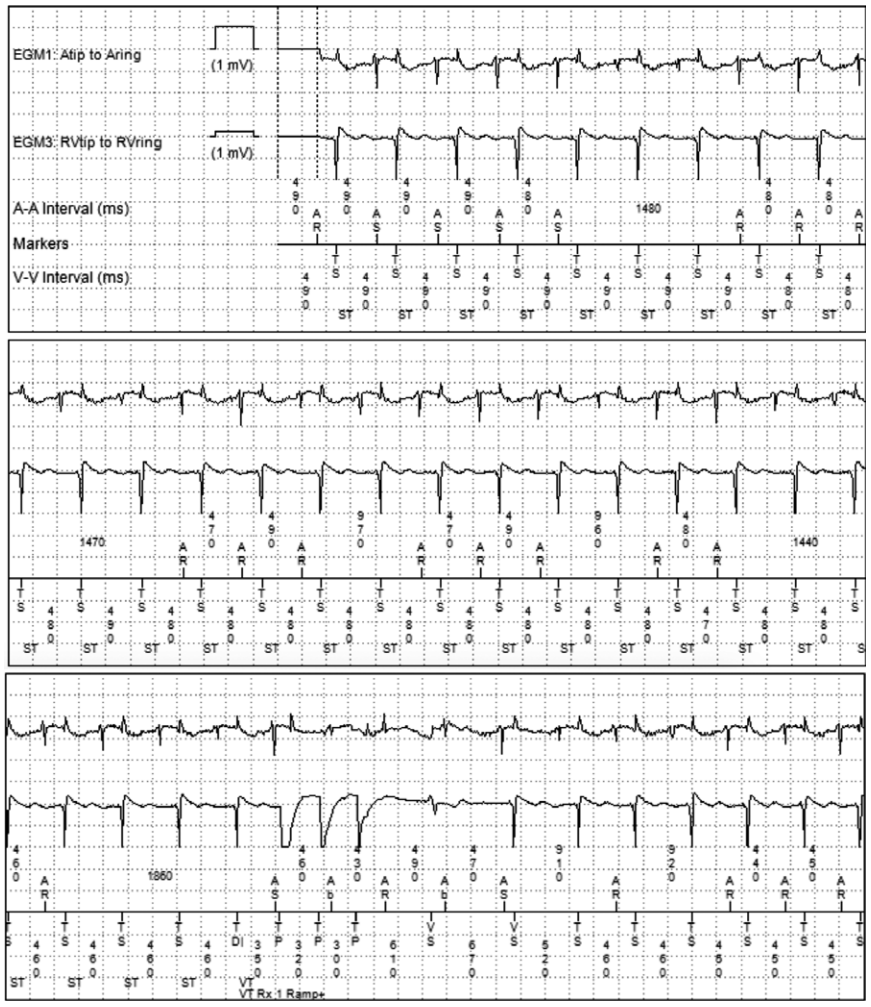

FIGURE 2 Intracardiac electrocardiogram (EGM) from the right atrium and right ventricular leads as derived the from the implantable cardioverter deibrillator

with more V EGMs than A EGMs, due to atrial undersensing, leading to reclassification of tachycardia as VT and initiation of programmed therapy.

We observed from device EGM that the atrial EGM during tachycardia was of the same morphology as the atrial EGM post termination of tachycardia. This confirmed that the algorithm was correct in initially classifying the tachycardia as sinus tachycardia, but due to atrial undersensing, it was subsequently misclassified as VT and the subject received inappropriate therapy. However, after the tachycardia slowed down it was deemed to have terminated by the device and no further therapy was delivered.

2.3 | Correlating clinical information with device interrogation findings

The subject was found to have severe anemia, which resulted in ST. VT zone was previously extended downwards due to concerns of amiodarone therapy resulting in slow VT. This resulted in overlap between VT detection zone and subject's maximum predicted sinus rates. Discontinuation of amiodarone, with resultant loss of beta-blocking action, and severe anemia resulted in ST that was misinterpreted as VT due to atrial undersensing. It is important to remember that beta-blockers and device reprogramming should be considered while discontinuing amiodarone.

2.4 | Interventions

We lowered atrial lead sensitivity from 0.9 to 0.3 mV to avoid undersensing in atrial lead. We stretched VT detection from 16 to 28 consecutive intervals and raised VT zone TCL from 530 ms to 490 ms to avoid inappropriate therapies in the VT zone. In this episode, there was intermittent atrial undersensing that triggered VT detection. Stretching detection interval reduces the chance of enough consecutive under sensed atrial EGMs to fulfill the VT criteria. We switched off shock therapies in VT zone as VT at this relative slow rate is unlikely to be hemodynamically significant thereby reducing the chance of inappropriate shocks. The subject also received appropriate therapy to correct anemia.

3 | CONCLUSION

The interval plot is a useful tool to allow physicians to interpret events at a glance. Accurate interpretation of the intra cardiac EGM would also be necessary to allow us to make an accurate diagnosis.

CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

ORCID

Colin Yeo http://orcid.org/0000-0002-1888-2246

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