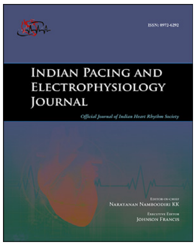
INDIAN PACING AND ELECTROPHYSIOLOGY JOURNAL 15 (2015) 236–244

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/IPEJ



ICD discrimination of SVT versus VT with 1:1 V-A conduction: A review of the literature

Rhanderson N. Cardoso, Chris Healy, Juan Viles-Gonzalez, James O. Coffey[*]

Cardiovascular Division, Department of Medicine, University of Miami, Miller School of Medicine, Miami, USA

ARTICLE INFO

Article history:
Available online 12 February 2016

Keywords:
Implantable cardioverter defibrillator
Supraventricular tachycardia
Atrioventricular nodal reentrant tachycardia
Inappropriate shock
Rhythm discrimination algorithms

ABSTRACT

Inappropriate ICD shocks are associated with increased mortality. They also impair patients' quality of life, increase hospitalizations, and raise health-care costs. Nearly 80% of inappropriate ICD shocks are caused by supraventricular tachycardia. Here we report the case of a patient who received a single-lead dual-chamber sensing ICD for primary prevention of sudden cardiac death and experienced inappropriate ICD shocks. V-A time, electrogram morphology, and response to antitachycardia pacing suggested atrioventricular nodal reentry tachycardia, which was confirmed in an electrophysiology study. Inspired by this case, we performed a literature review to discuss mechanisms for discrimination of supraventricular tachycardia with 1:1 A:V relationship from ventricular tachycardia with 1:1 retrograde conduction.

Contents

Introduction

Implantable cardioverter defibrillator (ICD) use has been shown to reduce mortality among patients with heart failure and left ventricular systolic dysfunction [1–6]. However, up to 13% of patients who receive an ICD can receive inappropriate shocks and as much as 31% of total shocks delivered by ICDs are considered inappropriate [7,8]. Nearly 80% of inappropriate ICD shocks are caused by supraventricular tachycardia (SVT), which includes atrial fibrillation (AF), atrial flutter,

---

[*] Corresponding author. 4062 University of Miami Hospital – East Building, 1400 NW 12th Avenue, Miami, FL 33136, USA. Tel.: +1 305 243 7040.

E-mail address: jamesodellcoffey@gmail.com (J.O. Coffey).

Peer review under responsibility of Indian Heart Rhythm Society.

http://dx.doi.org/10.1016/j.ipej.2016.02.006

sinus tachycardia, atrial tachycardia, atrioventricular (AV) reentrant tachycardia, and AV nodal reentrant tachycardia (AVNRT) [8]. AF is known to be a common cause of ICD therapies, but the rhythm irregularity and other factors facilitate fairly accurate discrimination and avoidance of ICD shocks via device programming [8–10]. At the same time, all SVT entities with the exception of AF can exist with a 1:1 A-V relationship, thereby presenting a potential diagnostic challenge.

Discrimination of ventricular tachycardia (VT) from such arrhythmias is believed to be facilitated by the presence of dual-chamber detection [11,12]. However, the Centers for Medicare & Medicaid Services does not reimburse the addition of an atrial lead for that purpose alone in patients who have no documented SVT prior to device implantation [13], because there is evidence that SVT-induced ICD shocks can be avoided just as successfully with a single lead device and optimal device programming [14–16]. Furthermore, implanting an atrial lead solely for that purpose adds unnecessary risk to the procedure, particularly dislodgement, perforation, and vascular injury [17–19].

It is in that context that the FDA recently approved a novel ICD lead that enables two-chamber sensing without requiring a separate atrial lead [20]. Herein we present a patient who received an ICD using such a lead but nevertheless experienced inappropriate ICD shocks secondary to SVT, consistent with typical slow-fast AVNRT. This case inspired a literature review of the discrimination mechanisms designed to differentiate SVT with a 1:1 relationship from ventricular tachycardia (VT) with 1:1 retrograde conduction.

Case report

A 57-year-old man with non-ischemic dilated cardiomyopathy and an ejection fraction of 20% for several years despite optimal medical management received an ICD for primary prevention of sudden cardiac death. The implanted device employed a single lead with atrial sensing capabilities (BIOTRONIK Lumax 740 VR-T DX, BIOTRONIK SE & Co KG, Berlin, Germany). Of note, although he had experienced palpitations in the past, at the time of device implantation he had no documented history of tachyarrhythmias. Several months after the implant, he presented to the electrophysiology clinic with recurrent ICD shocks. The patient reported multiple episodes of palpitations and lightheadedness, several of which were terminated by ICD shocks. On these occasions, he was fully conscious when shocked and was clearly emotionally impacted by the events, as he was now complaining of fear, anxiety, and a sense of impending doom. Device interrogation revealed multiple episodes of tachycardia with a fast ventricular rate (205–225 bpm), a 1:1 V-A relationship, and a V-A time of 50 ms (msec) (Fig. 1). In several cases, antitachycardia pacing (ATP) was able to successfully terminate the arrhythmia (Fig. 2). At other times, despite ventricular capture, ATP was unable to entrain the tachycardia. In those instances, the tachycardia persisted after ATP (Fig. 3). On two occasions, the tachycardia fell into the ventricular fibrillation (VF) zone, resulting in ICD shocks. Table 1 illustrates the device settings at the time of shock.

A diagnosis of AVNRT was strongly suspected on the retrospective review of the tachycardia episode, based on the short V-A time, the unchanged ventricular morphology on intracardiac electrograms, and the response to ATP. The patient was, therefore, scheduled for an electrophysiology (EP) study and possible radiofrequency catheter ablation. Meanwhile, in order to avoid further inappropriate shocks while awaiting the EP study, the VF zone was increased to greater than 233 bpm. At the EP study, dual AV nodal physiology was in fact revealed. A narrow complex tachycardia was reproducibly induced with single atrial extra-stimuli (Fig. 4). The tachycardia had a 1:1 VA relationship, a negative V-A time, and concentric atrial activation. Entrainment maneuvers were consistent with typical AVNRT. Slow pathway modification was performed, following which tachycardia was no longer inducible. Post-ablation, the device settings were returned to the primary prevention settings standard for our practice. On follow-up device interrogations, there have been no further episodes of tachycardia. The patient is relieved, but states that the anxiety caused by this experience has not completely resolved.

Discussion

The aim of this analysis is to highlight potential difficulties in device discrimination of non-AF SVT from VT and to review what is known about existing options to prevent inappropriate treatment in such cases. The negative consequences of inappropriate shocks are several-fold. A single inappropriate shock results in increased mortality, with a hazard-ratio (HR) of 1.6. The risk further increases with each subsequent shock until up to a HR of 3.7 after 5 inappropriate shocks [7]. Significant behavioral disorders, psychological distress, and a negative impact on quality of life have also been described following ICD shocks [21–24]. Furthermore, inappropriate shocks are pro-arrhythmic and have the potential to cause malignant ventricular arrhythmias [25,26]. Finally, they also lead to more frequent clinic visits and hospitalizations, with a subsequent increase in healthcare costs [27,28].

An observational analysis of 426 patients reported that 13.6% of inappropriate ICD shocks were attributed to AVNRT; the incidence of AVNRT among ICD recipients was approximately 3.5% [29]. Current multi-society guidelines give a Class I indication to catheter ablation for the treatment of symptomatic AVNRT [30]. Catheter ablation targeting the slow pathway of the AV node has a success rate greater than 95%, with a risk of heart block requiring pacemaker implantation of only about 1% [31,32]. In other words, identifying ICD patients with AVNRT has the potential to reduce or eliminate inappropriate shocks, thereby improving patients' quality of life and possibly even their survival.

The commercially available algorithms used to discriminate SVT from VT differ depending on whether dual- or single-chamber sensing is available. In single-chamber sensing, the most used criteria are *electrogram morphology*, *interval stability*, and *suddenness of onset*. Both AVNRT and VT with 1:1 VA conduction typically have a *sudden onset* and *high interval stability*. Therefore, in single-chamber sensing, electrogram morphology is the only criterion capable of

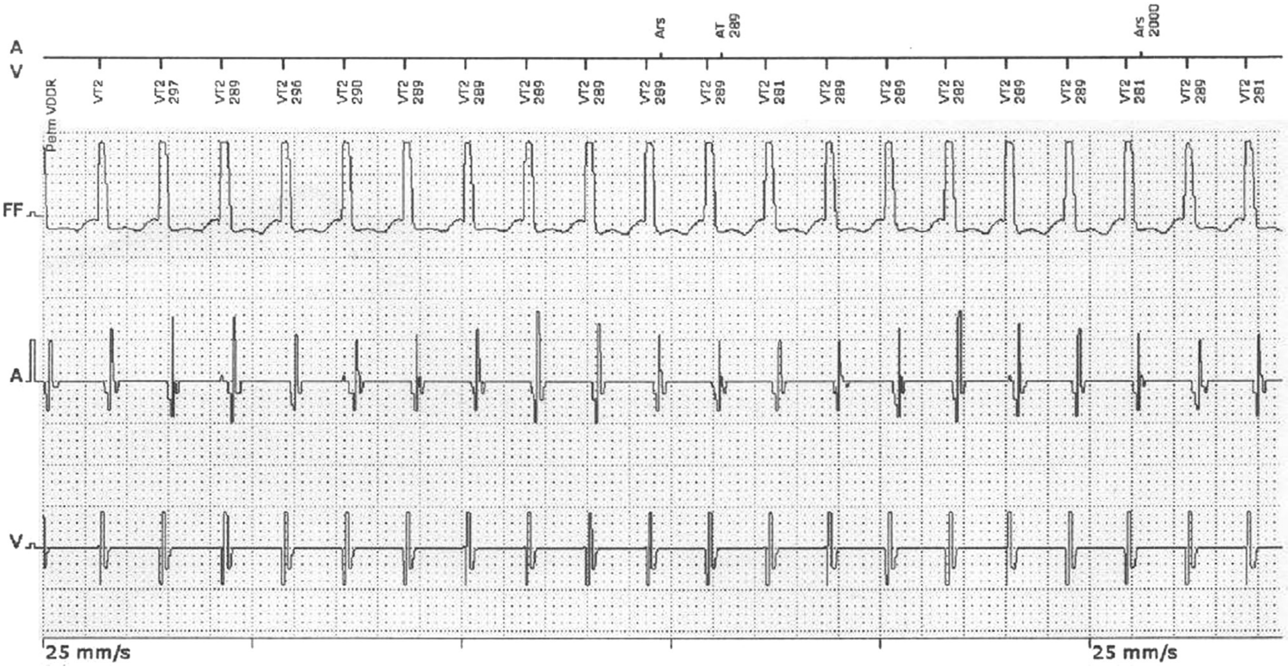


Fig. 1 – A short VA interval (50 msec) is observed in the tachycardia which resulted in ICD shock. A: atrial electrogram; AV: marker channel; FF: far-field electrogram; V: ventricular electrogram.

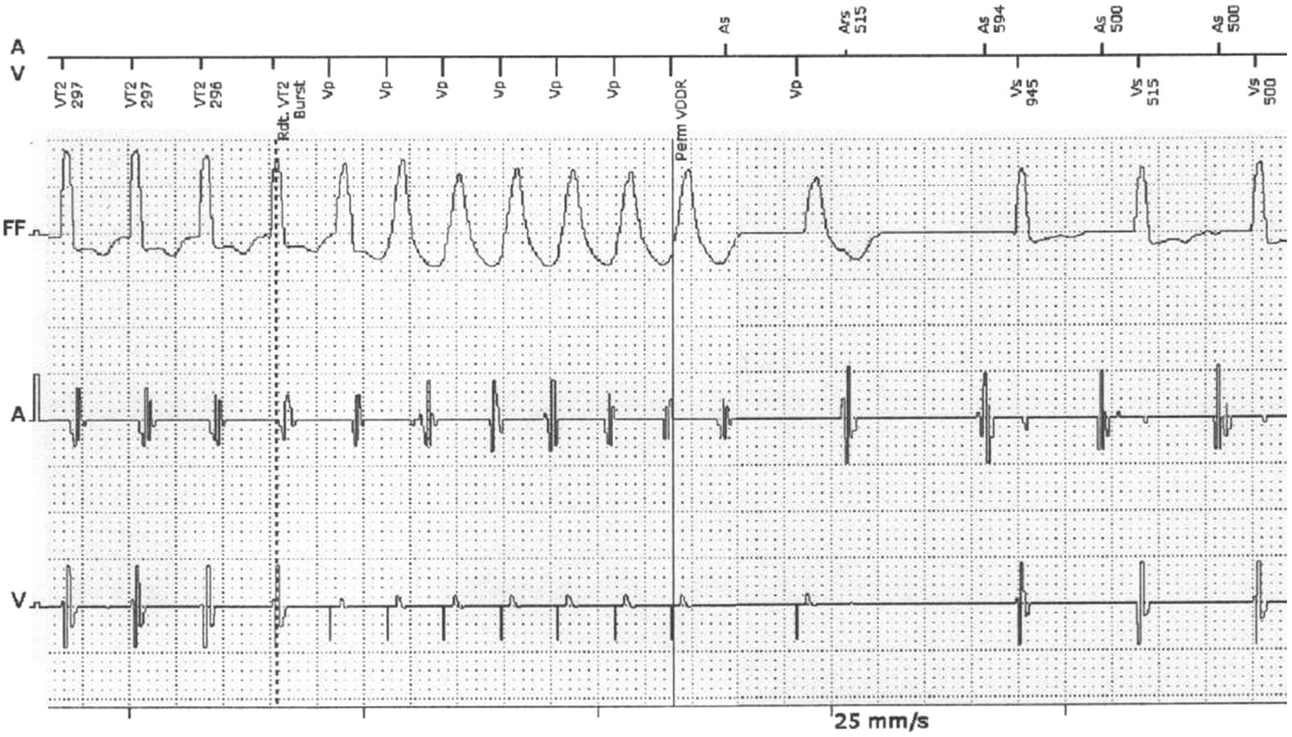


Fig. 2 – Antitachycardia pacing with entrainment of the tachycardia, as evidenced by an atrial cycle length (CL) which is (1) shorter than the tachycardia CL and (2) exactly the same as the ventricular paced CL. In this case, ATP terminates the tachycardia after entrainment. This response does not differentiate AVNRT from VT with 1:1 retrograde conduction (see text for full explanation). A: atrial electrogram; AV: marker channel; F: far-field electrogram; V: ventricular electrogram.

discriminating between VT and abrupt-onset regular SVTs, such as AVNRT. While this distinction can be easily made in the setting of "normal" conduction, it is not uncommon for AVNRT to be associated with rate-related bundle branch block, thereby removing the usefulness of *electrogram morphology* [10].

In dual-chamber sensing, additional criteria for SVT-VT distinction include *comparison of atrial and ventricular rates*,

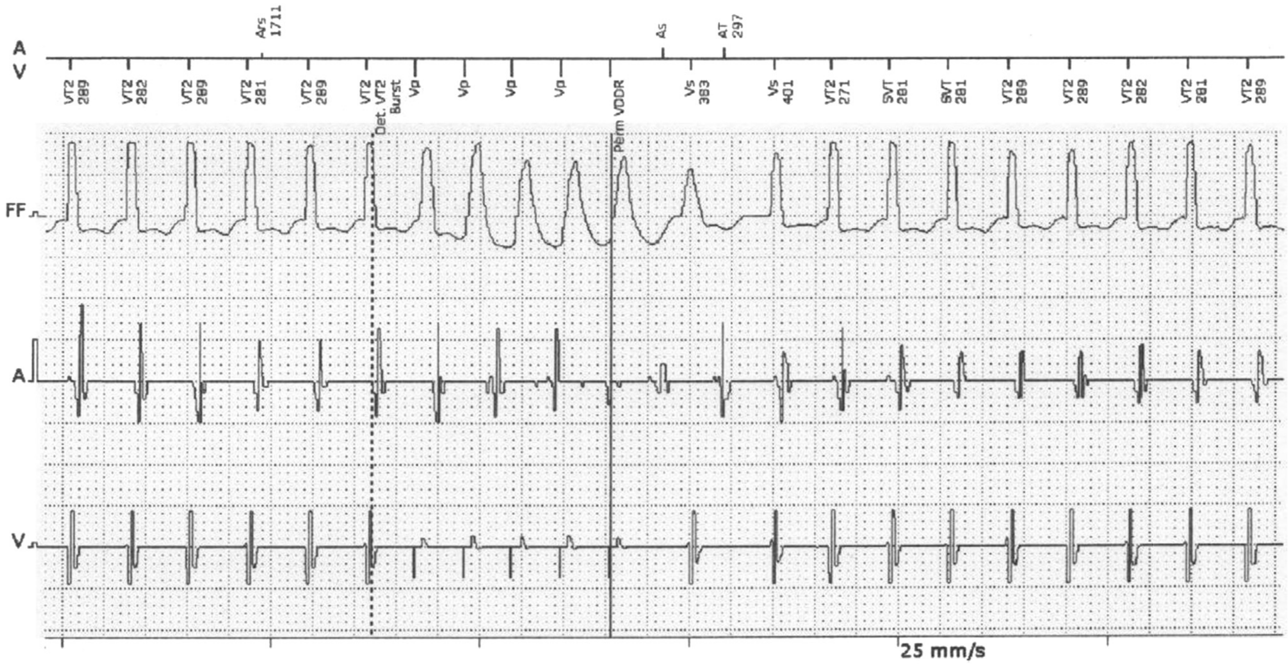


Fig. 3 – Antitachycardia pacing (ATP) with AV dissociation. The shortening of the ventricular cycle length (CL) and the change in QRS morphology seen on the far-field electrogram confirm ventricular capture. The atrial CL, however, remains unchanged throughout and after ATP, so entrainment has not occurred. In the setting of ventricular ATP with AV dissociation, persistence of the tachycardia after ATP is finished is suggestive of an atrial (or sinus) tachycardia or atrioventricular nodal reentrant tachycardia. This finding excludes atrioventricular reentrant tachycardia and VT with 1:1 VA conduction (see text for full explanation). A: atrial electrogram; AV: marker channel; F: far-field electrogram; V: ventricular electrogram.

AV association, and *P:R pattern*. [10] More than 90% of VTs are identified by a ventricular rate > atrial rate [10]. However, up to 20% of patients with VT demonstrate retrograde conduction and thus have the potential to develop VT with 1:1 retrograde conduction [33,34]. As in AVNRT, VT with 1:1 VA conduction presents with AV association and equal atrial and ventricular rates. Therefore, the distinction between AVNRT and VT with 1:1 VA conduction is highly dependent on timing relationships between atrial and ventricular electrograms, which is called *P:R pattern* analysis [10]. The *V-A interval*, measured from the onset of ventricular depolarization to the subsequent earliest demonstration of atrial activation, is used to characterize the P:R pattern [35]. Atrial and ventricular conduction occur almost concurrently in AVNRT; the V-A interval is therefore shorter than in VT [35]. Indeed, the V-A interval is typically longer than 80 msec in VT with retrograde conduction [36]. In the patient we present here, therefore, the V-A time of 50 msec is consistent with AVNRT.

While the use of V-A interval is obviously crucial in distinguishing between AVNRT and VT, it may not be sufficient for discrimination. Given the importance of atrial sensing in dual-chamber discriminators and the increased sensitivity required to detect low-amplitude atrial electrograms, there is a potential for oversensing of far-field R wave signals and subsequent overestimation of the atrial rate [10,37]. This phenomenon may lead to inappropriate rejection of VT as SVT or vice versa [10,38–40]. To prevent atrial oversensing of far-field R waves, some dual-chamber ICDs have a post-ventricular atrial blanking period, in which the atrial lead is unable to sense any events for up to 200 ms [10,41]. Undersensing of atrial events in the blanking period also has the potential to cause inappropriate shocks, because the ventricular rate can be mistakenly identified as greater than the atrial rate [10,14,41]. In fact, in the case here presented, atrial events were not recognized by the device, as they occurred within the postventricular blanking period. Thus, the ventricular rate was interpreted as greater than the atrial rate, and VT was (mistakenly) recognized.

Atrial undersensing (and misclassification of tachycardia as ventricular rate > atrial rate) is a major limitation of dual-

Table 1 – Device parameters at time of shock.

SAV	300–260 msec (60–130 bpm)[a]
PVAB	75 msec
PVARP	225 msec
PVARP after a PVC	375 msec
VT1 zone	160–179 bpm; monitor only
VT2 zone	180–224 bpm; ATP, shock x 5
VF zone	≥225 bpm; ATP during charging; shock x 5

[a] The SAV decreases from 300 to 260 msec as the patient's heart rate increases from 60 to 130 bpm. SAV: Sensed AV delay; PVAB: Post-Ventricular Atrial Blanking; PVARP: Post-Ventricular Atrial Refractory Period; PVC: Premature Ventricular Contraction.

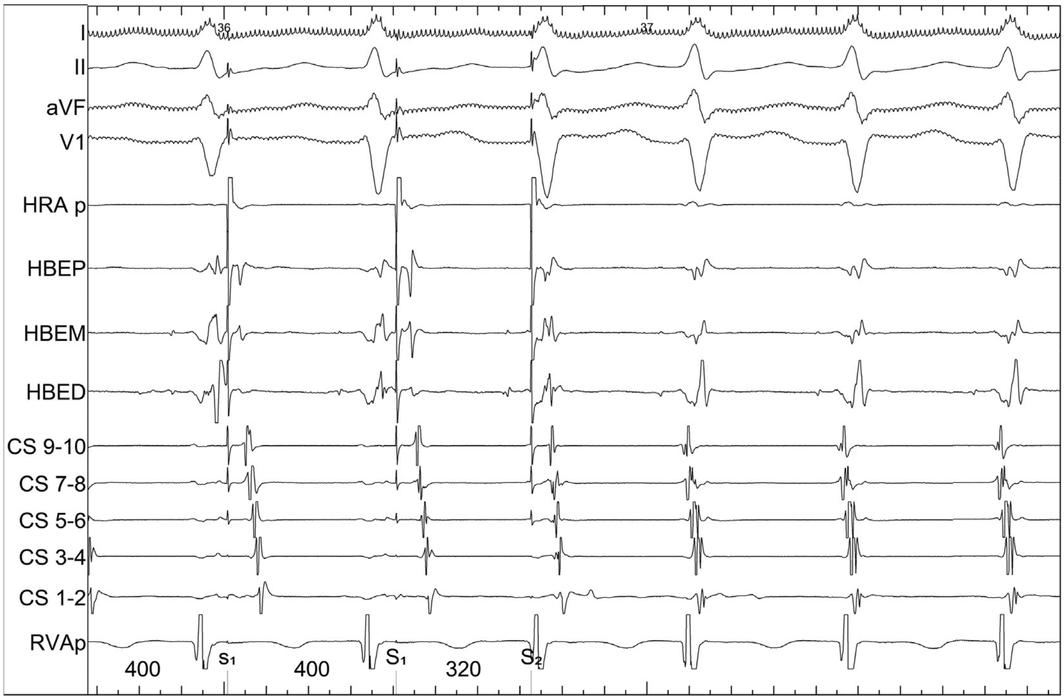


Fig. 4 – The above narrow complex tachycardia was reproducibly induced following an AH jump during an EP study. The characteristics of the tachycardia included a 1:1 V-A relationship, a negative V-A time, concentric atrial activation, and entrainment intervals consistent with typical AVNRT, thereby confirming the suspected diagnosis. Following slow pathway modification the tachycardia was no longer inducible. I, II, aVF, and V1 = surface electrograms; HRA p = high right atrial intracardiac electrogram; HBEP, HBEM, and HBED = His intracardiac electrograms (proximal, mid, and distal); CS 9–10, CS 7–8, CS 5–6, CS 3–4, and CS 1–2 = coronary sinus intracardiac electrograms (proximal to distal); RVAp = right ventricular apical intracardiac electrogram.

chamber ICDs, and may be accountable for the absence of benefit in avoiding inappropriate therapies when compared to single-chamber devices. In a randomized trial comparing single to dual-chamber ICDs, 75% of inappropriate therapies in the dual-chamber group were secondary to atrial sensing problems, mostly undersensing [15]. Manufacturers have developed different strategies to overcome the challenges of setting the post-ventricular atrial blanking period. In Biotronik SMART algorithm, the blanking period can be adjusted to a minimal value, in which case sensing of atrial events is not affected, thereby enabling accurate determination of the atrial rate and the V-A interval [42]. A similar option is available in St. Jude Medical and Boston Scientific devices [43]. The Medtronic PR Logic and ELA/Sorin algorithms maintain atrial sensing during the PVAB for the purpose of SVT-VT discrimination. When a 2:1 AV rhythm is identified, these algorithms analyze A-V and V-A intervals to reject far-field R waves and therefore prevent atrial oversensing [43].

Different manufacturers use the various discriminators in distinct sequences to establish an algorithm for VT/SVT discrimination. The DX Biotronik ICD system utilizes the SMART algorithm (Fig. 5), which includes heart rate, interval stability, A:V association, and P:R pattern analysis. Atrial and ventricular rates are analyzed first. If the ventricular rate is faster (Fig. 5, line 1), the rhythm is classified as VT. If the atrial rate is faster (Fig. 5, line 2), the rhythm is identified as SVT if either the RR is unstable (suggests variable AV conduction) or the A/V relationship shows an integral conduction ratio (e.g., 2:1, 3:1). If the ventricular and atrial rates are equal (Fig. 5, line 3), the system checks for stability and association. If the RR is stable but the PP is not, there is AV dissociation and VT is identified. If both RR and PP are stable, the rhythm is classified as VT if either the PR changes (AV dissociation) or if there is suddenness of onset. If the RR is unstable, a stable PR (AV association) indicates SVT, whereas an unstable PR (AV dissociation) indicates VT [44]. As shown in the bold sequence of Fig. 5, this algorithm cannot reliably differentiate AVNRT from VT with retrograde 1:1 conduction, given that both arrhythmias present with equal atrial and ventricular rates, stable RR and PP intervals (stability), no PR change (AV association), and sudden onset. In this particular sequence, the SMART system identifies VT. Therefore, the electrophysiologist should have increased awareness of the possibility of AVNRT and apply the concepts discussed here to successfully discriminate this potentially curable arrhythmia.

Unlike the SMART system, St. Jude Medical, Boston Scientific Rhythm ID, and Medtronic PR Logic algorithms utilize morphology assessments to discriminate SVT from VT [45]. In Medtronic devices that utilize PR Logic, SVT-VT discrimination can still occur in the VF zone. PR Logic uses three patterns of discriminators to identify SVTs (Table 2). Each of these rules is individually programmable and can be turned off. As in other dual-chamber sensing algorithms, a ventricular rate faster than the atrial rate identifies VT. If the rhythm cannot

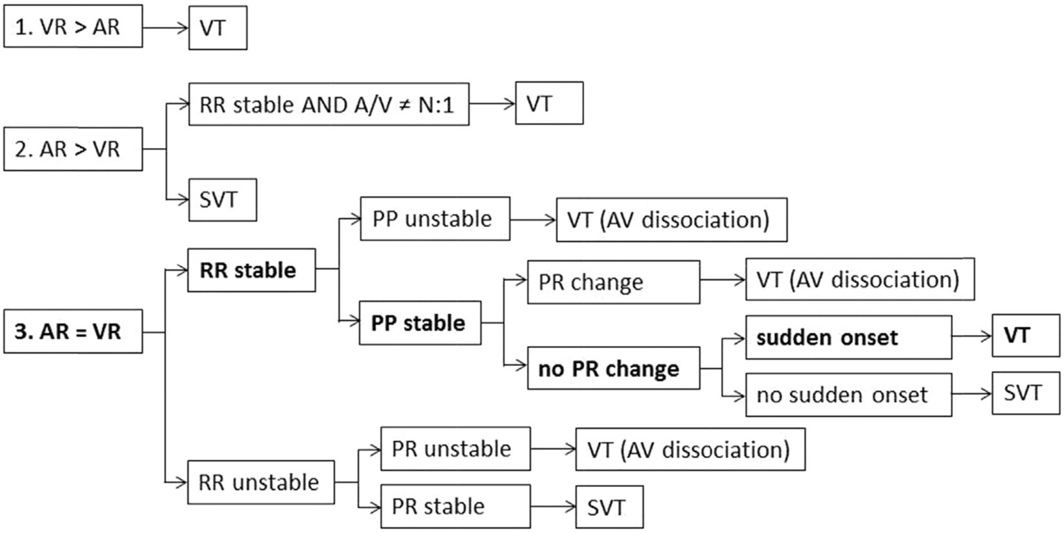


Fig. 5 – SMART algorithm for tachycardia analysis used in the Biotronik DX single-lead ICD. In our patient, atrial activity occurred during the post-ventricular atrial blanking period. Thus, the device identified VR > AR (Line 1), and (mistakenly) called the rhythm VT. In case the device had correctly identified VR = AR, the bold sequence illustrates how the rhythm still would have been called VT. Notice this sequence is unable to discriminate AVNRT from VT with retrograde 1:1 conduction (see text for full explanation). Modified from: Lori et al. Implantable cardioverter defibrillator system with floating atrial sensing dipole: A single-center experience. *Pacing Clinc Electrophysiol.* 2014;37:1265–1273. AR = atrial rate; A/V ≠ N:1 = conduction ratio not integral; SVT = supraventricular tachycardia; VR = ventricular rate; VT = ventricular tachycardia.

Table 2 – PR Logic algorithm for tachyarrhythmia discrimination (Medtronic Inc.).

SVT rule		Device classification
A:V = 1 AND near simultaneous activation of A & V		1:1 SVT
A:V = 1 AND gradual onset AND AV interval consistent with antegrade conduction		Sinus tachycardia
Atrial rate > Ventricular rate	Irregular VV OR AV association AND regular VV	Atrial fibrillation/atrial tachycardia
	AV dissociation AND regular VV	Double tachycardia (coexisting atrial and ventricular tachyarrhythmias)

be classified as VT based on rates, and none of the three SVT rules can be identified, PR Logic applies morphologic criteria for discrimination. This is done by checking for concordance between the unknown tachyarrhythmia and the baseline ventricular depolarization morphology [45–47].

In addition to the previously mentioned V-A interval, the response to ATP may also help in discriminating AVNRT from VT with retrograde 1:1 conduction. Overdrive pacing by ATP with a cycle length (CL) that is slightly shorter than the tachycardia CL can lead to one of three responses in AVNRT. *Entrainment* occurs when pacing is able to continuously reset the full tachycardia circuit with each paced beat. In entrainment, the atrial CL exactly matches the paced CL (Fig. 2). *AV dissociation* develops when ventricular capture occurs, but the nodal tachycardia circuit is maintained; in this case, the ventricular CL matches the paced rhythm, but the atrial CL remains unchanged from the original tachycardia. Finally, *termination* of AVNRT by ATP is also a possibility. Up to 20% of SVTs can be terminated by ATP [48–50]. Termination and entrainment also occur in VT; therefore, these responses to ATP cannot reliably discriminate AVNRT from VT with 1:1 VA conduction [51,52]. However, maintenance of the same tachycardia CL in the atria after ATP ventricular capture (*AV dissociation*; Fig. 3) and after ATP is finished demonstrates that atrial depolarization during the tachycardia is independent of ventricular origin. Thus, this finding excludes the possibility of VT with 1:1 retrograde conduction and atrioventricular reentrant tachycardia using a usual atrioventricular accessory pathway. In VT with retrograde 1:1 conduction, when ATP ventricular capture occurs, the CL in the atria either (1) follows the CL of the ventricular paced rhythm (if entrainment occurs), or (2) returns to a sinus/atrial paced rhythm if the paced stimuli are unable to reach the atria. The CL in the atria cannot, however, remain the same as the tachycardia CL, given that atrial depolarization arises from a ventricular stimulus in VT with 1:1 VA conduction.

Importantly, RR intervals falling into the ventricular fibrillation (VF) zone are not considered for discrimination by the Biotronik SMART algorithm. At ventricular rates >200–230 bpm, distinction between VF and SVT is not accurate due to the following: (1) RR intervals in AF can become more regular [53]; (2) both VF and rapidly conducting AF present with AV dissociation [38]; and (3) irregular RR intervals are also observed in polymorphic VT and VF, which could potentially misclassify the rhythm as SVT [10]. Interestingly, up to 42% and 10% of ICD therapies for ventricular arrhythmias occur at rates > 188 bpm and >250 bpm, respectively, and thus have the potential to fall under the VF zone [54]. Ricci et al. showed that 50% of AF-related inappropriate shocks occur at rates >200 bpm [55]. The concern for VF zone inappropriate ICD therapy in SVT is particularly worrisome in younger patients with rapidly conducting AV nodes, where the heart rate can reach nearly 250 bpm [29,56]. The patient in our case had received 2 ICD shocks for SVT with rates in the VF zone.

Conclusion

Differentiating SVT from VT in patients with ICDs is important in the optimization of clinical outcomes, but this can be challenging in rhythms with a 1:1 VA relationship. Programming of the post-ventricular atrial blanking period can result in undersensing of atrial activity (long PVABP) or oversensing of far-field R waves as atrial activity (short PVABP), which can misclassify a 1:1 rhythm into ventricular rate > atrial rate, or vice-versa, respectively. Setting dual-chamber devices to a minimal blanking period can allow accurate sensing during the refractory period and precise determination of the V-A interval, which can be extremely useful in distinguishing AVNRT (short V-A interval) from VT with 1:1 retrograde conduction. The response to ATP may also assist in discriminating such arrhythmias. Particularly, the persistence of the tachycardia CL in the atria concomitantly with ATP ventricular capture (*AV dissociation*) is highly suggestive of AVNRT. Catheter ablation is a class I indication for the treatment of symptomatic AVNRT. Inappropriate shocks due to AVNRT in patients with ICDs provide further support for this potentially curative treatment. In this scenario, the procedure has the added benefit of preventing further inappropriate shocks and their negative consequences.

REFERENCES

[1] Zipes DP, Wyse DG, Friedman PL, Epstein AE, Hallstrom AP, Greene L, et al. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. N Engl J Med 1997;337:1576–83.

[2] Bardy GH, Lee KL, Mark DB, Poole JE, Packer DL, Boineau R, et al. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med 2005;352:225–37.

[3] Bristow MR, Saxon LA, Boehmer J, Krueger S, Kass DA, De Marco T, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. N Engl J Med 2004;350:2140–50.

[4] Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, et al. Canadian Implantable Defibrillator Study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. Circulation 2000;101:1297–302.

[5] Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: the Cardiac Arrest Study Hamburg (CASH). Circulation 2000;102:748–54.

[6] Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877–83.

[7] van Rees JB, Borleffs CJ, de Bie MK, Stijnen T, van Erven L, Bax JJ, et al. Inappropriate implantable cardioverter-defibrillator shocks: Incidence, predictors, and impact on mortality. J Am Coll Cardiol 2011;57:556–62.

[8] Daubert JP, Zareba W, Cannom DS, McNitt S, Rosero SZ, Wang P, et al. Inappropriate implantable cardioverter-defibrillator shocks in Madit II: frequency, mechanisms, predictors, and survival impact. J Am Coll Cardiol 2008;51:1357–65.

[9] Wolpert C, Jung W, Spehl S, Schimpf R, Omran H, Schumacher B, et al. Incidence and rate characteristics of atrial tachyarrhythmias in patients with a dual chamber defibrillator. Pacing Clin Electrophysiol 2003;26:1691–8.

[10] Swerdlow CD. Supraventricular tachycardia-ventricular tachycardia discrimination algorithms in implantable cardioverter defibrillators: state-of-the-art review. J Cardiovasc Electrophysiol 2001;12:606–12.

[11] Friedman PA, McClelland RL, Bamlet WR, Acosta H, Kessler D, Munger TM, et al. Dual-chamber versus single-chamber detection enhancements for implantable defibrillator rhythm diagnosis: the detect supraventricular tachycardia study. Circulation 2006;113:2871–9.

[12] Bansch D, Steffgen F, Gronefeld G, Wolpert C, Bocker D, Mletzko RU, et al. The 1+1 trial: a prospective trial of a dual- versus a single-chamber implantable defibrillator in patients with slow ventricular tachycardias. Circulation 2004;110:1022–9.

[13] Centers for Medicare & Medicaid Services. Coverage determinations: Implantable automatic defibrillators. In: Medicare National Coverage Determinations Manual. Baltimore, MD. Available at: http://www.cms.gov/medicare-coverage-database/details/ncd-details.aspx?NCDId=110&ncdver=3&CoverageSelection=National&KeyWord=defibrillator&KeyWordLookUp=Title&KeyWordSearchType=And&bc=gAAAABAAAAAAAA%3d%3d&. [accessed 19.01.15].

[14] Kuhlkamp V, Dornberger V, Mewis C, Suchalla R, Bosch RF, Seipel L. Clinical experience with the new detection algorithms for atrial fibrillation of a defibrillator with dual chamber sensing and pacing. J Cardiovasc Electrophysiol 1999;10:905–15.

[15] Deisenhofer I, Kolb C, Ndrepepa G, Schreieck J, Karch M, Schmieder S, et al. Do current dual chamber cardioverter defibrillators have advantages over conventional single chamber cardioverter defibrillators in reducing inappropriate therapies? A randomized, prospective study. J Cardiovasc Electrophysiol 2001;12:134–42.

[16] Theuns DA, Klootwijk AP, Goedhart DM, Jordaens LJ. Prevention of inappropriate therapy in implantable cardioverter-defibrillators: results of a prospective, randomized study of tachyarrhythmia detection algorithms. J Am Coll Cardiol 2004;44:2362–7.

[17] Ghani A, Delnoy PP, Ramdat Misier AR, Smit JJ, Adiyaman A, Ottervanger JP, et al. Incidence of lead dislodgement, malfunction and perforation during the first year following device implantation. Neth Heart J 2014;22:286–91.

[18] Antonelli D, Feldman A, Schliamser JE, Militianu A, Turgeman Y. Acute pericardial tamponade due to screw-in atrial lead heart perforation. Europace 2012;14:453–5.

[19] Takahashi T, Bhandari AK, Watanuki M, Cannom DS, Sakurada H, Hiraoka M. High incidence of device-related and lead-related compared with the single-chamber version. Circ J 2002;66:746–50.

[20] Safak E, Schmitz D, Konorza T, Wende C, De Ros JO, Schirdewan A. Clinical efficacy and safety of an implantable cardioverter-defibrillator lead with a floating atrial sensing dipole. Pacing Clin Electrophysiol 2013;36:952–62.

[21] Luderitz B, Jung W, Deister A, Marneros A, Manz M. Patient acceptance of the implantable cardioverter defibrillator in ventricular tachyarrhythmias. Pacing Clin Electrophysiol 1993;16:1815–21.

[22] Vlay SC, Olson LC, Fricchione GL, Friedman R. Anxiety and anger in patients with ventricular tachyarrhythmias. Responses after automatic internal cardioverter defibrillator implantation. Pacing Clin Electrophysiol 1989;12:366–73.

[23] Thomas SA, Friedmann E, Kao CW, Inguito P, Metcalf M, Kelley FJ, et al. Quality of life and psychological status of patients with implantable cardioverter defibrillators. Am J Crit Care 2006;15:389–98.

[24] Mark DB, Anstrom KJ, Sun JL, Clapp-Channing NE, Tsiatis AA, Davidson-Ray L, et al. Sudden Cardiac Death in Heart Failure Trial I. Quality of life with defibrillator therapy or amiodarone in heart failure. N Engl J Med 2008;359:999–1008.

[25] Messali A, Thomas O, Chauvin M, Coumel P, Leenhardt A. Death due to an implantable cardioverter defibrillator. J Cardiovasc Electrophysiol 2004;15:953–6.

[26] Mitchell LB, Pineda EA, Titus JL, Bartosch PM, Benditt DG. Sudden death in patients with implantable cardioverter defibrillators: the importance of post-shock electromechanical dissociation. J Am Coll Cardiol 2002;39:1323–8.

[27] Bhavnani SP, Giedrimiene D, Coleman CI, Guertin D, Azeem M, Kluger J. The healthcare utilization and cost of treating patients experiencing inappropriate implantable cardioverter defibrillator shocks: a propensity score study. Pacing Clin Electrophysiol 2014;37:1315–23.

[28] Pereferrer Kleiner D, Sicras Mainar A, Villuendas Sabate R, Alcalde Rodriguez O, Labata Salvador C, Bayes-Genis A. Do inappropriate implantable cardioverter-defibrillator shocks generate additional costs? Rev Esp Cardiol 2014;67:65–6.

[29] Goldberger JJ, Passman R, Arora R, Kadish AH. A higher than expected prevalence of AV nodal reentrant tachycardia in patients receiving implantable cardioverter-defibrillators. Pacing Clin Electrophysiol 2011;34:584–6.

[30] Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, Alpert JS, Calkins H, Camm AJ, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias–executive summary. J Am Coll Cardiol 2003;42:1493–531.

[31] Clague JR, Dagres N, Kottkamp H, Breithardt G, Borggrefe M. Targeting the slow pathway for atrioventricular nodal reentrant tachycardia: Initial results and long-term follow-up in 379 consecutive patients. Eur Heart J 2001;22:82–8.

[32] Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. Pacing Clin Electrophysiol 2000;23:1020–8.

[33] Militianu A, Salacata A, Meissner MD, Grill C, Mahmud R, Palti AJ, et al. Ventriculoatrial conduction capability and prevalence of 1:1 retrograde conduction during inducible sustained monomorphic ventricular tachycardia in 305 implantable cardioverter defibrillator recipients. Pacing Clin Electrophysiol 1997;20:2378–84.

[34] Jongnarangsin K, Pumprueg S, Prasertwitayakij N, Crawford TC, Mukerji S, McLemore-McGregor R, et al. Utility of tachycardia cycle length variability in discriminating atrial tachycardia from ventricular tachycardia. Heart Rhythm 2010;7:225–8.

[35] Katritsis DG, Camm AJ. Atrioventricular nodal reentrant tachycardia. Circulation 2010;122:831–40.

[36] Thompson JA, Jenkins JM. Ventriculoatrial conduction metrics for classification of ventricular tachycardia with 1:1 retrograde conduction in dual-chamber sensing implantable cardioverter defibrillators. J Electrocardiol 1998;31(Suppl.):152–6.

[37] Swerdlow CD, Schsls W, Dijkman B, Jung W, Sheth NV, Olson WH, et al. Detection of atrial fibrillation and flutter by a dual-chamber implantable cardioverter-defibrillator. For the Worldwide Jewel AF Investigators. Circulation 2000;101:878–85.

[38] Wilkoff BL, Kuhlkamp V, Volosin K, Ellenbogen K, Waldecker B, Kacet S, et al. Critical analysis of dual-chamber implantable cardioverter-defibrillator arrhythmia detection: results and technical considerations. Circulation 2001;103:381–6.

[39] Dijkman B, Wellens HJ. Dual chamber arrhythmia detection in the implantable cardioverter defibrillator. J Cardiovasc Electrophysiol 2000;11:1105–15.

[40] Safak E. Clinical efficacy and safety of an implantable cardioverter defibrillator lead with a floating atrial sensing dipole. Pacing Clin Electrophysiol 2014;37:130–1.

[41] Kamalvand K, Tan K, Kotsakis A, Bucknall C, Sulke N. Is mode switching beneficial? A randomized study in patients with paroxysmal atrial tachyarrhythmias. J Am Coll Cardiol 1997;30:496–504.

[42] Sinha AM, Stellbrink C, Schuchert A, Mox B, Jordaens L, Lamaison D, et al. Clinical experience with a new detection algorithm for differentiation of supraventricular from ventricular tachycardia in a dual-chamber defibrillator. J Cardiovasc Electrophysiol 2004;15:646–52.

[43] Koneru JN, Swerdlow CD, Wood MA, Ellengogen KA. Minimizing inappropriate or "unnecessary" cardioverter-defibrillator shocks: appropriate Programming. Circ Arrhythm Electrophysiol 2011;4:778–90.

[44] Iori M, Giacopelli D, Quartieri F, Bottoni N, Manari A. Implantable cardioverter defibrillator system with floating atrial sensing dipole: a single-center experience. Pacing Clin Electrophysiol 2014;37:1265–73.

[45] Madhavan M, Friedman PA. Optimal programming of implantable cardiac-defibrillators. Circulation 2013;128:659–72.

[46] Biffi M. ICD programming. Indian Heart J 2014;66(Suppl. 1):S88–100.

[47] Thachil A, Chennapragada S, Calambur N. Inappropriate detection of a supraventricular tachycardia as dual tachycardia by the pr logic algorithm. Indian Pacing Electrophysiol J 2014;14:161–4.

[48] Singh DK, Viswanathan MN, Tanel RE, Lee RJ, Lee BK, Marcus GM, et al. Heart Rhythm 2014;11:1327–35.

[49] Veenhuyzen GD, Quinn FR. Principles of entrainment: diagnostic utility for supraventricular tachycardia. Indian Pacing Electrophysiol J 2008;8:51–65.

[50] Arenal A, Ortiz M, Peinado R, Merino JL, Quesada A, Atienza F, et al. Differentiation of ventricular and supraventricular tachycardias based on the analysis of the first postpacing interval after sequential anti-tachycardia pacing in implantable cardioverter-defibrillator patients. Heart Rhythm 2007;4:316–22.

[51] Stevenson WG, Friedman PL, Sager PT, Saxon LA, Kocovic D, Harada T, et al. Exploring postinfarction reentrant ventricular tachycardia with entrainment mapping. J Am Coll Cardiol 1997;29:1180–9.

[52] Josephson ME, Almendral J, Callans DJ. Resetting and entrainment of reentrant ventricular tachycardia associated with myocardial infarction. Heart Rhythm 2014;11:1239–49.

[53] Swerdlow CD, Chen PS, Kass RM, Allard JR, Peter CT. Discrimination of ventricular tachycardia from sinus tachycardia and atrial fibrillation in a tiered-therapy cardioverter-defibrillator. J Am Coll Cardiol 1994;23:1342–55.

[54] Wathen MS, DeGroot PJ, Sweeney MO, Stark AJ, Otterness MF, Adkisson WO, et al. Prospective randomized multicenter trial of empirical antitachycardia pacing versus shocks for spontaneous rapid ventricular tachycardia in patients with implantable cardioverter-defibrillators: pacing fast ventricular tachycardia reduces shock therapies (painfree rx ii) trial results. Circulation 2004;110:2591–6.

[55] Ricci RP, Pignalberi C, Landolina M, Santini M, Lunati M, Boriani G, et al. Ventricular rate monitoring as a tool to predict and prevent atrial fibrillation-related inappropriate shocks in heart failure patients treated with cardiac resynchronisation therapy defibrillators. Heart 2014;100:848–54.

[56] Orejarena LA, Vidaillet Jr H, DeStefano F, Nordstrom DL, Vierkant RA, Smith PN, et al. Paroxysmal supraventricular tachycardia in the general population. J Am Coll Cardiol 1998;31:150–7.