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Current Issues in ICD SVT-VT Discrimination: Pacing for SVT-VT Discrimination

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1. Introduction

The stored electrograms (EGMs) retrieved from the ICD provide a unique and useful source of information regarding the mechanism of the underlying arrhythmia.

Current ICD algorithms discriminate VT from SVT on the basis of **passive analysis** of detected **rhythms** with positive predictive values of greater than 90% [1].

Despite this, the incidence of inappropriate therapies for SVT discrimination still remains high and varies from 16% to 31% as quoted in prior studies [2]. In many ways, the ICD bears a resemblance to a diagnostic electrophysiological study. The underlying cardiac rhythm is analysed and acted on often with the delivery of anti-tachycardia pacing (ATP) if the threshold is met. This therapeutic interaction by the ICD with the underlying arrhythmia can also be interpreted as a diagnostic manoeuvre similar to the pacing techniques employed in the electrophysiology laboratory before arriving at the diagnosis (figure 1). The success or failure of ATP to terminate the underlying rhythm may both have value.

1.1. Anti-tachycardia pacing

Anti-tachycardia pacing has been demonstrated to be a safe, effective and painless therapy in randomized controlled multicentre trials [3,4]. In the PAINFREE trial two sequences of ATP were delivered before a shock in the fast ventricular tachycardia (FVT) zone. A total of 446 FVT episodes with a mean cycle length of 301 ± 24 msec were documented in 52 patients. A total of 396 of these FVT episodes were terminated by ATP alone with an adjusted efficacy of 77% (95% CI 68% to 83%) [5]. Acceleration of the VT by ATP occurred in only 10 (4%) FVT episodes but these went on to delivery of a definitive shock aborting the episode (figure 2).

Figure 1. The ICD bears a resemblance functionally to a diagnostic electrophysiology suite

Arrhythmia related syncope that may have been from the marginal delay during delivery of ATP occurred in just 4 patients (2%) and merely involved 9 device episodes.

The PAINFREE Rx II randomized ICD patients to 2 arms: standardised empirical ATP in the FVT zone ($n=313$) before shock or directly to shock in the control group ($n=321$) [6]. Anti-tachycardia pacing was effective in 229 of 284 episodes in the ATP arm thus yielding an adjusted efficacy of 72%. The episode duration, incidence of arrhythmic syncope and acceleration of VT was similar in both arms.

In their evaluation of ATP as first line therapy, Schoels and coworkers evaluated 760 ventricular arrhythmia episodes in 128 patients. Five hundred were appropriately detected (82 patients) [7,8].

Their analysis however showed that with conventional ICD programming and detection there were 260 episodes that were inappropriately treated. Of these 224 (57 patients) were atrial tachycardia or atrial fibrillation (AT/AF) while the remaining 36 episodes (19 patients) were due to sinus tachycardia.

This suggests that conventional device detection algorithms are prone to misdiagnosis for supraventricular arrhythmias in a significant proportion of patients. In the case of devices programmed with ATP as first line therapy it would be painless and would not result in significant morbidity. This does not hold true if there was an inappropriate shock delivered.

Since pacing is a common way of differentiating arrhythmias in an electrophysiological study, the response to this form of pacing in the ICD, by deduction may therefore hold clues as to the mechanism of the underlying detected rhythm. This then has diagnostic potential for the device specialist evaluating stored EGMs in a clinic setting and possibly has the potential for further algorithm development.

Figure 2. A. An episode of VT in the FVT zone terminated by a burst of ATP. B. The first burst of ATP in a detected VT episode fails to terminate the VT so a second burst at a shorter cycle length is delivered resulting in acceleration of the VT which then leads to a shock from the device terminating the episode.

2. Pacing to discriminate between atrial tachycardia and re-entrant SVT

Ventricular pacing and an evaluation of the atrial response after advancement of the A during retrograde conduction is a conventional manoeuvre of differentiating AT from a re-entrant SVT either AVNRT or AVRT. Knight et al. demonstrated that an A-A-V response after 1:1 VA conduction after ventricular pacing during ongoing tachycardia had a specificity and sensitivity for diagnosing AT figure 3 [9,10].

An A-V response on cessation pacing, however, suggests either AVNRT or AVRT as the underlying mechanism (figure 4). This interpretation is based on condition that the A is advanced during V pacing and that the underlying tachycardia continues unperturbed post pacing.

Using this data, it therefore seems fairly intuitive to apply these atrial responses to the interpretation of device EGMs after ATP. If there is consequent conduction to the atrium in a 1:1 fashion with advancement of the A, then the return response after pacing maybe diagnostic as discussed above [11].

This concept was applied by Ridley and co-workers to the interpretation of ICD EGMs from dual chamber ICDs (Medtronic, MN, USA) [12]. The evaluation of responses, however, was

Figure 3. An A-A-V response after V pacing and 1:1 VA conduction with ongoing tachycardia suggests AT as the underlying mechanism.

based on the interval plot summary of episodes and not on the intracardiac signals. These were categorized as a type 1 response if the ventricle (V) was dissociated from the atrium (A)

Figure 4. An A-V response on cessation of V pacing with 1:1 VA conduction suggests either AVNRT or AVRT.

defining the rhythm as an AT (figure 5A). A type 2 response was due to variable VA conduction and therefore leading to an inconclusive A response (figure 5B).

Figure 5. A. Type 1 response: the ventricular EGMs are dissociated from the atrial events during ATP. This is consistent with a diagnosis of AT. B. Type 2 response: ATP results in a variable atrial response and therefore is inconclusive.

A type 3A response occurs if the post pacing phenomenon is a V-A-A-V which is essentially an A-A-V if the last paced V, is not taken into account, as was encountered in the Knight et al. study mentioned above (figure 5C). The overall sensitivity was 71.9% (95% CI 67.1-73.6) and a specificity of 95% (95% CI 83.5-99.1). A Type 3B or V-A-V response was felt to be less conclusive for a SVT and the authors felt this did not exclude VT. This study however was

based on the the interval plot as opposed to the EGMs. In our opinion, by reviewing both near and farfield EGMs in conjunction with the scatterplot, a reasonable clinical deduction can be made with regards a V-A-V response to suggest either AVNRT/AVRT. A device-based algorithm might combine pacing response with EGM morphology to discriminate the Type 3B response.

A limitation in this study was the high exclusion rate of cases since 45.1% of data could not be reliably analysed for various reasons. In 74.5% of these cases the tachycardia was terminated by the ATP as well. This, in itself, does not imply that all these episodes were VT since SVTs may also terminate with ventricular pacing. The flow diagrams in the diagnostic approach discussed later in this chapter discuss how termination of tachycardia can be evaluated to obtain a rhythm diagnosis.

Figure 6. A type 3A response shows a VAAV pattern which is consistent with AT and a type 3B response where a VAV pattern is observed.

3. Pitfalls in interpreting the VAAV/VAV response

In order to evaluate the atrial response after V pacing, it is important to confirm that the atrium was indeed advanced during ongoing tachycardia.

Iso-arrhythmic dissociation of the ventricle at the atrial tachycardia rate may mimic 1:1 VA conduction resulting in a misdiagnosis of a VAV interval on cessation of pacing.

The next common problem is to recognise the pseudo VAAV pattern. This could occur coincidentally post ATP delivered in the ventricle with VA dissociation during an episode of AT (figure 11).

A long VA interval during an SVT as is the case in atypical AVNRT or the so called “fast-slow” variant can also yield a pseudo “VAAV” response (figure 7).

4. Limitations

The following device related limitations need to be borne in mind:

1. Over/undersensing producing an incorrect A or V response on the interval plot.
2. Timing with automatic, decaying threshold sensing may not be accurate resulting a blanking of the sensed event.
3. The 10 ms resolution in Medtronic ICDs leading to an inherent error in the estimated intervals.

Issa, Z. F. and Mansour, I. N. *J.Cardiovasc.Electrophysiol.* 18(5), 548-549, 2007.

Figure 7. a). This SVT was determined to be an atypical AVNRT using a fast-slow re-entrant substrate. This was inappropriately detected and ATP was delivered by the device. On cessation of pacing a “VAAV” response is seen – or is it? (b). The same EGM is shown above. This time the arrows show each pacing spike delivered in the ventricle and the corresponding atrial signal. The last entrained atrial event (A) event is late and closer to the next ventricular sensed event (VS) because of retrograde conduction up the slow pathway. This in reality is a VAV sequence and not a VAAV response!

*1:1 Tachycardias	N:1
ST	AF
AT	Aflutter
Aflutter	AT
AVNRT	
AVRT	
VT with retrograde conduction	

*Challenge for passive ICD algorithms with variable ability to discriminate.

Table 1. Classification of Device Intracardiac EGMs

The tachycardias detected by the ICD therefore can be broadly classified as either 1:1 ($A = V$) tachycardias or N:1 tachycardias (ie. $A > V$).

The 1:1 tachycardias are difficult for both device algorithms as well as the observer to resolve as either retrograde VA conduction during VT as opposed to a SVT (table 1). However, 1:1 VA conduction only occurs in about 10% of detected VTs and therefore has a low probability [13, 14]. However, the consequences of misdiagnosis of VT is much greater than the misdiagnosis of SVT. If this is misclassified as an SVT, then the result may well be a withholding of appropriate device therapies which is best avoided.

It seems rather intuitive to distinguish N:1 tachycardias as either AF, A flutter or AT if there are accompanying atrial EGMs on which to base this interpretation. Single chamber ICDs are frequently implanted if the indication is a primary prevention strategy or in the presence of persistent atrial tachycardias so that the atrial signals are lacking in these device EGMs. This then does not permit the use of algorithms that rely on A and V patterns of association in order to discriminate the rhythm. The result may well be inappropriate therapies in the form of ATP and/or shocks. It also makes it difficult for the device specialist when it comes to analysing the tracings [11].

In the example in figure 8, it is evident that the underlying arrhythmia is rapidly conducted AF. In the following clinical scenario, a single chamber ICD (figure 9), it is difficult to be certain that this is indeed an episode of VT. It may well be an organized atrial tachycardia or an episode of paroxysmal AF which is pseudo regularised and detected in tachycardia zone. Anti-tachycardia pacing is elicited as it is the first line therapy programmed in this detection zone of the device. It was deemed ineffective and therefore there was escalation to a shock. After each burst of ATP, a pause (arrows) is evident before resumption of the tachycardia. We postulated that the pause duration may help predict the chamber of origin of the tachycardia, namely VT vs AT/AF [15].

Figure 8. An interval plot showing uncontrolled atrial fibrillation detected in the VF zone of the ICD leading to ATP in the form of a ramp and then progression to sequential inappropriate shocks.

Figure 9. A treated episode in a single chamber ICD detected as VT but may also be a regular SVT with rapid onset. The absence of an atrial EGM makes it difficult to be absolutely certain.

5. The post pacing interval after ATP as discrimination tool

The mechanism of the observed pause after an episode of ATP for true VT, in fact, would represent “entrainment” provided that the VT is advanced by the ATP and then continues unchanged post ATP. This pause then can thus be referred to as the post pacing interval (PPI). The difference between the PPI and the ambient tachycardia cycle length (TCL) has been established as indication of the proximity of the pacing source to the tachycardia circuit and is a fundamental electrophysiological concept [16,17] (figure 10).

In the case of AF or AT the tachycardia source is in a relatively distant chamber, namely the atria. The pause following ATP would represent retrograde invasion of the infra nodal conducting system and concealed penetration of the AV node.

This illustration demonstrates the concept of the PPI. After delivery of ATP and the absolute PPI and the difference between the PPI and ambient TCL (PPI-TCL) is used to predict the source of the tachycardia (figure 11).

Episodes of failed ATP for detected tachycardias in a heterogenous cohort of 250 patients receiving dual chamber and biventricular ICDs were evaluated at our centre. Fifty one events ($n=18$ AT/AF and $n=33$ VT) were eventually compared after excluding episodes in which ATP terminated or altered the $TCL \geq 50$ ms ie. a significant perturbation of the underlying tachycardia.

The mean PPI after failed episodes of ATP for VT and AF/AT were 512 ± 88 ms vs 693 ± 96 ms ($p < 0.01$). Thus a significant difference was observed in the pause intervals for appropriately and inappropriately delivered ATP which is understandable given the different mechanisms accounting for the PPI in each context.

Figure 10. The pacing source is described as a distance X away from a macro-re reentrant circuit of tachycardia cycle length A + B + C. The PPI is therefore equal to the sum of 2 x X and the TCL (A+B+C).

Figure 11. An episode of a 1:1 AT is inappropriately treated by the ICD. The A EGMs are dissociated from the V during ATP (arrows). There is an apparent long PPI with a pseudo VAAV response.

Figure 12. ROC curves for PPI and PPI-TCL and the cut-offs for each are indicated.

The same observation was made for the PPI-TCL difference for VT and AF/AT: 179 ± 103 ms vs 330 ± 97 ms ($p < 0.01$), respectively.

The ROC identified cut off values of 615ms or greater for the PPI predicting AF/AT with a sensitivity of 77.8% (95% CI 58.6%-97.0%) and a specificity of 87.5% (95% CI 76.0%-99.0%) (figure 12).

A PPI-TCL ≥ 260 ms also predicted AF/AT with a sensitivity of 72.2% (95% CI 51.5%-92.9%) and a specificity of 78.1% (95% CI 63.8% - 92.4%) (figure 12).

The use of the pause interval represents a form of active discrimination in that it relies on the response to ATP rather than a passive evaluation of EGMs which is the conventional method employed by device algorithms. This also represents a “downstream” evaluation after detection of the tachycardia by the device has already occurred.

Saba and colleagues in the Dynamic Discrimination Download Study (DD) presented a paradigm shift from the “diagnose before treating” to a “treat first and diagnose what is left” algorithm design in Medtronic dual chamber ICDs (MN, USA) [18,19]. Here, once a tachycardia was detected, ATP was applied with the delivery of 8 pulses of ATP in the atrium and ventricle.

These were delivered either simultaneously with no AV delay (SAV) or with a Convergent AV delay (CAV) decrementing to 0ms which was thought to be less proarrhythmic (figure 13).

Saba S et al. Circulation 2010;121:487-497

Figure 13. The two methods by which ATP was delivered in the Dynamic Discrimination Download Study (DD).

The ATP was applied initially on detection and advanced discriminators in the form of PR logic (Medtronic, MN, USA) were only applied thereafter on the remaining rhythm disorder. If the tachycardia was not terminated by ATP, the chamber in which the first sequence of tachycardia was redetected post ATP, determined whether the rhythm was classified as SVT or VT (figure 14).

Saba S et al. Circulation 2010;121:487-497

Figure 14. In this example from the DD Study, ATP is applied in the CAV (convergent AV delay) format. The tachycardia resumes post ATP with the first event being an atrial sensed (AS) EGM. This then fulfills the criteria of being an SVT.

The authors argued that if ATP terminated the tachycardia, then the time to effective therapy was shortened. If tachycardia was ongoing, then there was still no appreciable delay after the initial 8 pulses of ATP in making a definitive diagnosis. The DD algorithm terminated or correctly classified 1379/1381 SVT episodes with an overall specificity of 99.9% and 23/26 VT episodes with a sensitivity of 88.5%. There was no significant difference in the effectiveness between the SAV and CAV ATP schemes ($p>0.5$). This upfront method of ATP delivery did not induce any atrial arrhythmias in the cohort studied but there was one episode of slow VT induced which spontaneously terminated.

The use of ATP as a means of pacing for SVT-VT discrimination does present some clinical challenges:

1. In the case of ventricular deliver of ATP, retrograde AV nodal conduction and advancement of the atrial EGM must be observed in order to interpret VAAV/VAV responses.
2. ATP itself may induce premature atrial on ventricular complexes that truncate pause intervals and/or influence the assessment of the chamber of origin of tachycardia.
3. ATP must capture ventricular myocardium during the drive train.
4. ATP may accelerate or decelerate the existing tachycardia or may induce further arrhythmia.

Some possible solutions to obviate these problems would be to:

1. Deliver the ATP in multiple sequences in order to insure myocardial capture.
2. To deliver the ATP at high pacing output
3. To automatically set variable blanking periods after the delivery of ATP in order not to sense induced premature beats.

6. General considerations when approaching device based tracings

1. The chamber of onset of the tachycardia (if observed) in a dual chamber ICD has been implanted helps discriminate SVT from VT.
2. Although VT tends to be a stable rhythm, cycle length variations may occur during onset of the tachycardia or in the presence of anti-arrhythmic drugs.
3. SVTs may also present as regular tachycardias and AF may show pseudoregularisation at rapid rates.
4. If A>V events are noted this usually defines an SVT (AT, AF or A Flutter) except in the case of dual tachycardias.
5. If V>A events are noted this suggests VT although AVNRT with intermittent retrograde block should be considered but this is fairly uncommon.

6. 1:1 tachycardias are difficult to differentiate but VT with retrograde conduction is only observed in 10-30% of VT episodes.

These points are summarised in the following flow chart:

* Be aware of dual tachycardias in the A > V arm

Figure 15. A broad classification of rhythm disorders according to the A and V electrogram relationship.

In 1:1 tachycardias, the atrial EGM in dual chamber devices, during the delivery of ventricular ATP and the EGM response post ATP with ongoing tachycardia can be evaluated for the atrial response:

1. VAAV response suggests AT, unless it is a pseudo VAAV which may occur in atypical AVNRT
2. VA dissociation suggests AT
3. VAV response is compatible with AVNRT/AVRT. It does not completely rule out VT but a VVA response is suggestive of VT.

These points are summarised in the following flow chart:

Figure 16. Arrhythmia classification based on atrial response post ATP.

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