Cardiac resynchronization therapy: Interventricular delay during ventricular and supraventricular tachyarrhythmia

Stroobandt Roland, MD, Ph.D[1], Kucher Andreas, M.Sc., Ph.D[2]

From Ghent University Hospital, Heart Center [1] and BIOTRONIK SE & Co.KG, Berlin, Germany[2]

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Correspondence: *Roland Stroobandt MD, PhD , Ghent University Hospital, C. Heymanslaan, 10 B-9000 Ghent, Belgium. E-mail: roland.stroobandt@ugent.be*

Abstract

Introduction

Implantable cardioverter-defibrillators (ICDs) for cardiac resynchronization therapy (CRT-D) with the capability of LV sensing enable the assessment of interventricular delays in ventricular (VT) and supraventricular tachycardias (SVT).

Methods and Results

In total, 1078 EGM recordings of VT or VF episodes were investigated that have been transmitted via Home Monitoring®. Only those EGM recordings showing the onset of the tachyarrhythmia were used for investigation. In the 623 cases eligible for evaluation left-sided VTs could be identified in 349 cases, right-sided VTs in 252 cases. SVTs with a 1:1 antegrade conduction were found in 22 cases. VT can present three different interventricular delays whereby the right-ventricular sensed event (RVs) is either preceding the left-ventricular sensed event (LVs), or the LVs is preceding RVs, or there can be a simultaneous occurrence of RVs and LVs. In SVTs, either the LVs events were delayed or occurred simultaneously with the RVs events. SVT cases with LVs preceding the RVs events were not found.

Conclusion

The LV-EGM channel enables to distinguish between left- and right-sided premature ventricular complexes. The assessment of the interventricular delay in VTs is useful to differentiate between a possible apical-, left- or right-sided origin of the VT but not to identify SVTs.

Key Words

Implantable Cardioverter Defibrillator (ICD), Cardiac resynchronization therapy (CRT), LV sensing, RV sensing, Ventricular tachycardia, Supraventricular tachycardia, Left bundle branch block, Heart failure, Interventricular conduction delay

Introduction

Cardiac resynchronization therapy (CRT) is a well-established treatment for heart failure patients with reduced left ventricular (LV) ejection fraction. BIOTRONIK and Boston Scientific devices used for CRT can both sense from the LV lead. LV sensing was designed primarily to prevent competitive pacing outside the LV myocardial absolute refractory period and it works by inhibiting the release of an LV stimulus in the vulnerable period of the LV myocardium.[1] During the manual sensing test at sinus rhythm a sufficient separation of the right- and the left-ventricular sensed events is useful to confirm an optimal positioning of the LV lead. Patients with heart failure often present both supraventricular (SVT) and ventricular tachycardias (VT). The diagnosis of slow VTs in CRT-D devices utilizing a special desynchronization detection algorithm is possible by event-triggered EGM recordings named as CRT pacing interrupt. Such slow VTs which are usually slower than the programmed VT intervention rate and present three different interventricular delays.[2] The main goal of the study was to assess which different shapes of the interventricular delay can be observed during VT and SVT.

Methods

Methods & Study Population

In this retrospective study, we investigated anonymized EGM recordings transmitted via the Home Monitoring (HM®) system of BIOTRONIK. In total, 1078 EGM recordings of CRT-D devices (Ilivia®, Intica®, and Inlexa® of BIOTRONIK) were reviewed which have been detected either in the VT or in the VF zone. A large number of episodes (n=455) were not eligible for further evaluation as the begin of the tachyarrhythmia was not displayed because the VT detection time was longer than the maximum duration of the EGM recording prior to detection. This is typically the case if the detection time was intendedly prolonged by programming very high detection counters. The main goal was to investigate the different appearances of the interventricular delays.

Results

In the 623 cases eligible for evaluation left-sided VTs could be identified in 349 cases, right-sided VTs in 252 cases. SVTs with a 1:1 antegrade conduction were found in 22 cases (Diagram 1). VT can present three different interventricular delays whereby the RVs event is either preceding the LVs event, or the LVs is preceding RVs, or there can be a simultaneous occurrence of RVs and LVs (Table 1 shows the possible interventricular delays). An SVT shows mostly a delayed left-ventricular signal, but it is also possible to observe a simultaneous occurrence of the LVs and the RVs events. In SVT, however, it is unlikely to find an interventricular delay in which the LV event precedes the RV. The numbers in the brackets are referring to the exemplary cases.

Case 1

Figure 1. This EGM recording shows the begin of the VT. It is a right-sided VT that has been triggered by a right-ventricular premature complex (RVPC). RVs is preceding the LVs with a left ventricular electrical delay of 100ms. A VT1 detection is fulfilled, but antitachycardia pacing (ATP) - a burst with 10 biventricular pulses - has slowed the VT only slightly below the VT1 intervention rate but has not terminated the VT.

Case 2

Figure 2. This EGM recording shows the begin of the left-sided VT which has been triggered by a left-ventricular premature complex (LVPC). LVs is preceding the RVs with a right-ventricular electrical delay of 100ms. A VT1 detection is fulfilled. The first attempt of ATP - a burst with 10 right-ventricular pulses has terminated the VT.

Case 3

Figure 3. This EGM recording shows the begin of the VT. The interventricular electrical delay is 0ms with a simultaneous occurrence of LVs and RVs. An absent interventricular delay could speak in favor of an apical or septal origin of the VT. The complete course of the VT was recorded in two subsequent stored episodes. The first part of the VT was detected as CRT pacing interrupt based on a 20-out of-48 counter. Since the VT1 counter was programmed higher than 20, a VT1 detection was still fulfilled shortly after the end of the first episode. The first attempt of ATP has terminated the VT (not documented here).

Case 4

Figure 4. This EGM recording shows the begin of an SVT (1:1) triggered by repeated APCs (atrial premature complexes). RVs is preceding the LVs. The interventricular electrical delay is 100 ms between the RVs and the LVs event. The atrial tachycardia rate is very fast and detected in the VF-zone. The ATP-one-shot (before shock) has accelerated the atrial tachycardia but the 1:1 P-R conduction has changed then into a 2:1 conduction ratio. With the halved ventricular rate, the already started charging process for the shock was prematurely aborted and a termination detection could be fulfilled.

Case 5

Figure 5. This EGM recording shows the begin of an SVT (1:1) triggered by an APC. The interventricular electrical delay is almost 0ms. The simultaneous occurrence of the atrial and the ventricular signals speaks in favor of an AV-nodal reentry tachycardia (AVNRT). The atrial markers are not depicted because they occur inside the far-field blanking period of 75ms after the RVs event. The AVNRT has been terminated by the first ATP.

Figure 6. The same patient like in Fig. 5, but this episode was recorded with another configuration of the EGM channels (FF, RA, RV) instead of (RA, RV, LV). The EGM recording shows the begin of an SVT (1:1) triggered by two APCs. The narrow QRS complexes in the FF-EGM are evidence of an SVT. The interventricular electrical delay is 0ms and the occurrence of the atrial and ventricle is almost simultaneously which favors the diagnosis of an AVNRT.

Figure 7. ATP (burst with 8 biventricular pulses) has terminated the AVNRT (vertical arrow) but triggered a pacemaker-mediated tachycardia (PMT). After the ATP, a normal interventricular delay is visible between the RVp and the LVs markers due to the left bundle branch block. The PMT algorithm has terminated the PMT by prolongation of the PVARP once which has generated an Ars marker (asterisk).

Discussion

CRT-D devices capable of LV sensing allow to investigate the interventricular delays in VT and SVT. If a tachycardia is faster than the programmed VT intervention rate, biventricular pacing is suppressed because the tachycardia intervals are shorter than both the right- and the left-ventricular upper rate intervals. The typical interventricular conduction delay (RVs to LVs) caused by the left ventricular bundle branch block (LBBB) during sinus rhythm disappears and the leading tachycardia rate will be taken over by the fast reentrant circuit. In dependence of the location of the reentrant circuit the direction of the excitation spread is changing over the heart. Due to the changed excitation spread and direction the RVs and LVs events can get a different order. The different order of the RVs and LVs events can be evaluated during VT or SVT. It is a clear hint that the tachycardia is originating from the right ventricle when the RVs is preceding the LVs. On the contrary when LVs precedes RVs it suggests that the tachycardia is originating from the left ventricle. The simultaneous occurrence of the RVs and LVs speaks in favor of an apical or septal location of the reentrant circuit.

In SVT there is mostly a right-ventricular delay. During an AVNRT, the interventricular delay can also be almost 0ms. SVT cases with LVs preceding the RVs events were not found. It is to assume that almost all CRT-D devices in this study population were implanted due to a left bundle branch block (LBBB).

Theoretically an interventricular delay with LVs preceding the RVs would certainly be possible in SVT in the presence of a right bundle branch block (RBBB).

Clinical Implications

The LV-EGM may improve the diagnosis of arrhythmias. Recording the LV-EGM channel enables to distinguish between left- and right-sided premature ventricular complexes and even isolated left-ventricular premature complexes can be uncovered as described by Larsen et al.[3] and Barold et al.[4] Ventricular arrhythmias may arise from anywhere in the ventricles. Localizing the origin of arrhythmias can be challenging. In cases where the arrhythmia occurs infrequently or lasts only for a few beats the timing of the RVEGM and the LVEGM may provide guidance for ablative therapy. The LV-EGM channel is also of clinical importance to uncover dissimilar ventricular tachycardia rhythms in CRT-D devices [5; 6] where the left ventricular tachycardia rate can be faster than the tachycardia rate in the RV channel (or opposite). The faster LV VT may be undetected by the RV channel when the RV-VT remains below the intervention rate and may prevent or delay lifesaving ICD therapy.

Limitations

The anonymized EGM recordings were the single alone basis for this investigation without any clinical background information and clinical indication for CRT-D implantation. It was assumed that almost all CRT-D devices in this study population were implanted due to a left bundle branch block (LBBB). None of patients in the study population had an RBBB which can be the cause why no SVT cases with LVs preceding the RVs could be observed.

Conclusion

The information of the left ventricle is essential to understand the origin of VT and to distinguish between left- and right-sided PVCs. The evaluation of the left-ventricular vulnerable period during biventricular or triggered LV pacing on RVs events or PVCs is not possible without the knowledge about what is occurring in the LV. The assessment of the interventricular delay in VT is useful to differentiate between the possible apical-, left- or right-sided origin of the VT but can not be used to identify SVTs.

References

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Legends to the Figures

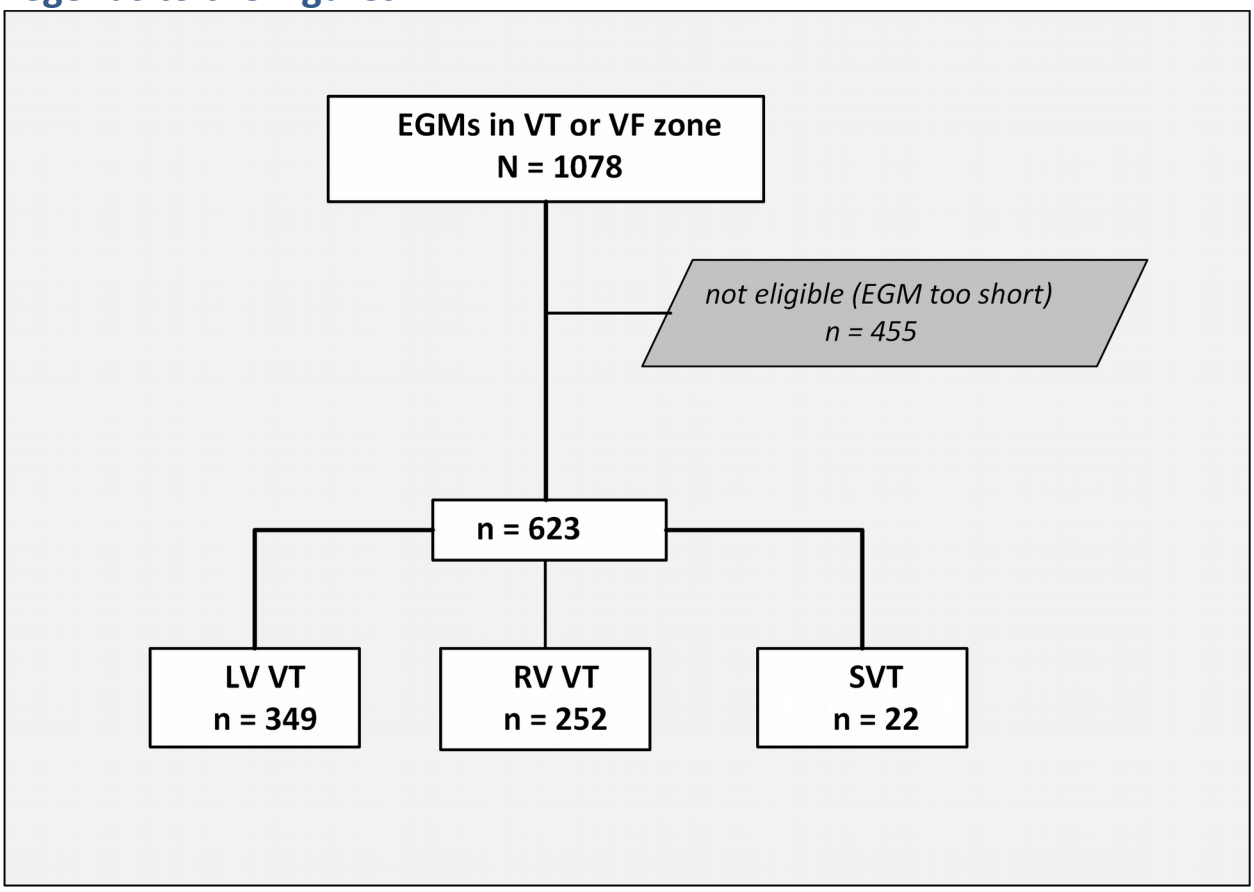

***Diagram 1**. Graphical review of the methodology and results.*

IVD	VT	SVT
RVs → LVs	• (1)	• (4)
LVs → RVs	• (2)	-
LVs and RVs simultaneous	• (3)	• (5)

Table 1. *Interventricular delays in VT and SVT. IVD = interventricular delay; VT = ventricular tachycardia; SVT = supraventricular tachycardia; RVs = right-ventricular sensed event; LVs = left-ventricular sensed event.*

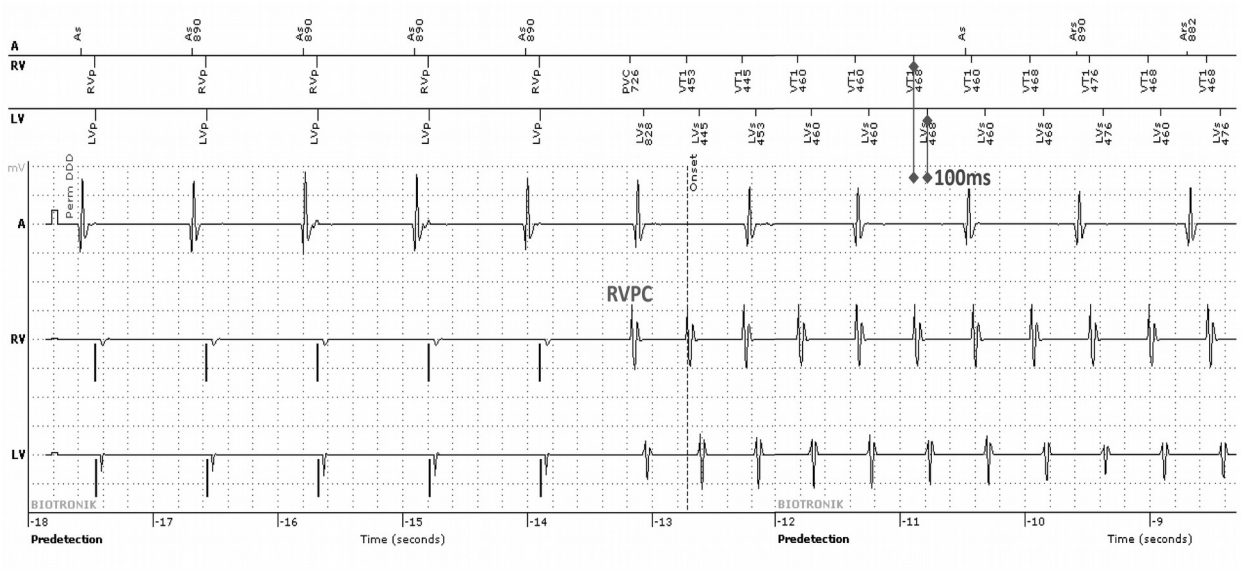

Figure 1. *Right-sided VT. The marker channels are on top: A = atrium, RV = right ventricle, LV = left ventricle. The corresponding EGM recordings are below the marker channels. As = atrial sensed event, Ars = refractory sensed atrial event, VT1 = RV sensed event detected in the VT1 zone, RVp = right-ventricular paced event; LVs = LV sensed event, LVp = left-ventricular paced event; PVC =premature ventricular complex. RVPC = right-ventricular premature complex. See text for details.*

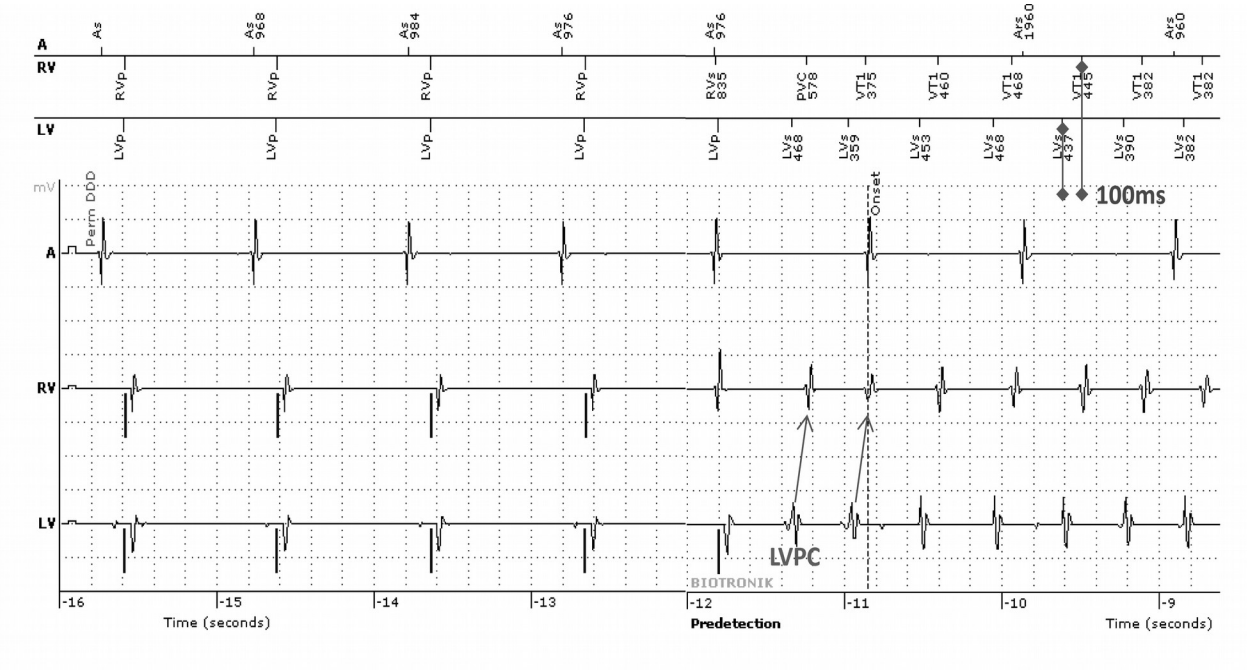

Figure 2. *Left-sided VT. Same arrangement of the markers and EGM channels as in Fig. 1.*

205 *LVPC = left-ventricular premature complex. See text for details.*

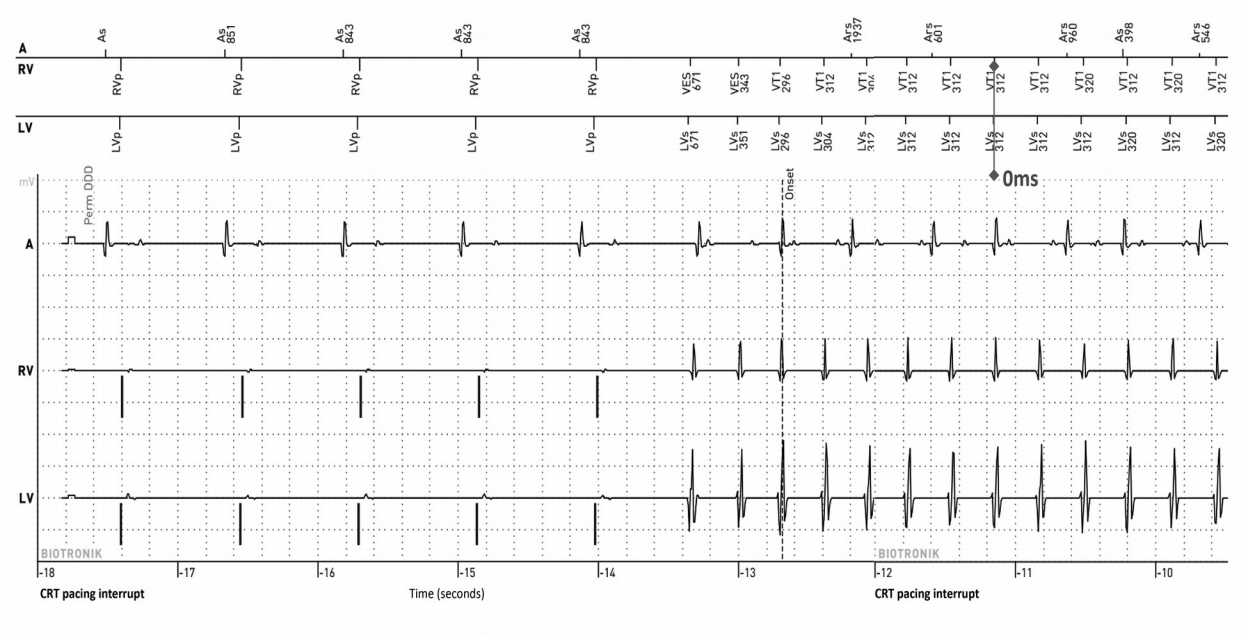

Figure 3. *Apical VT. Same arrangement of the markers and EGM channels as in Fig. 1. VES = ventricular extrasystole. See text for details.*

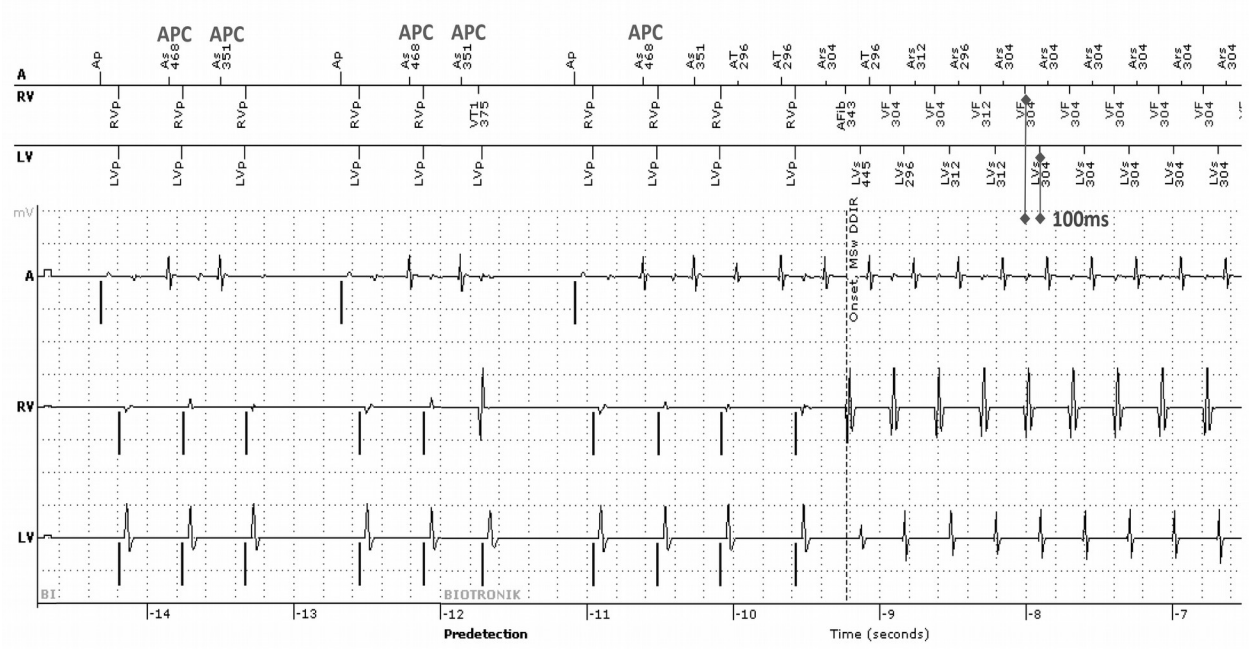

Figure 4. *Atrial reentry tachycardia. Same arrangement of the markers and EGM channels as in Fig. 1. Ap = atrial paced events; APC = atrial premature complex; VF = ventricular sensed event detected in the VF zone; Afib = ventricular sensed event classified by SMART algorithm as atrial fibrillation. See text for details.*

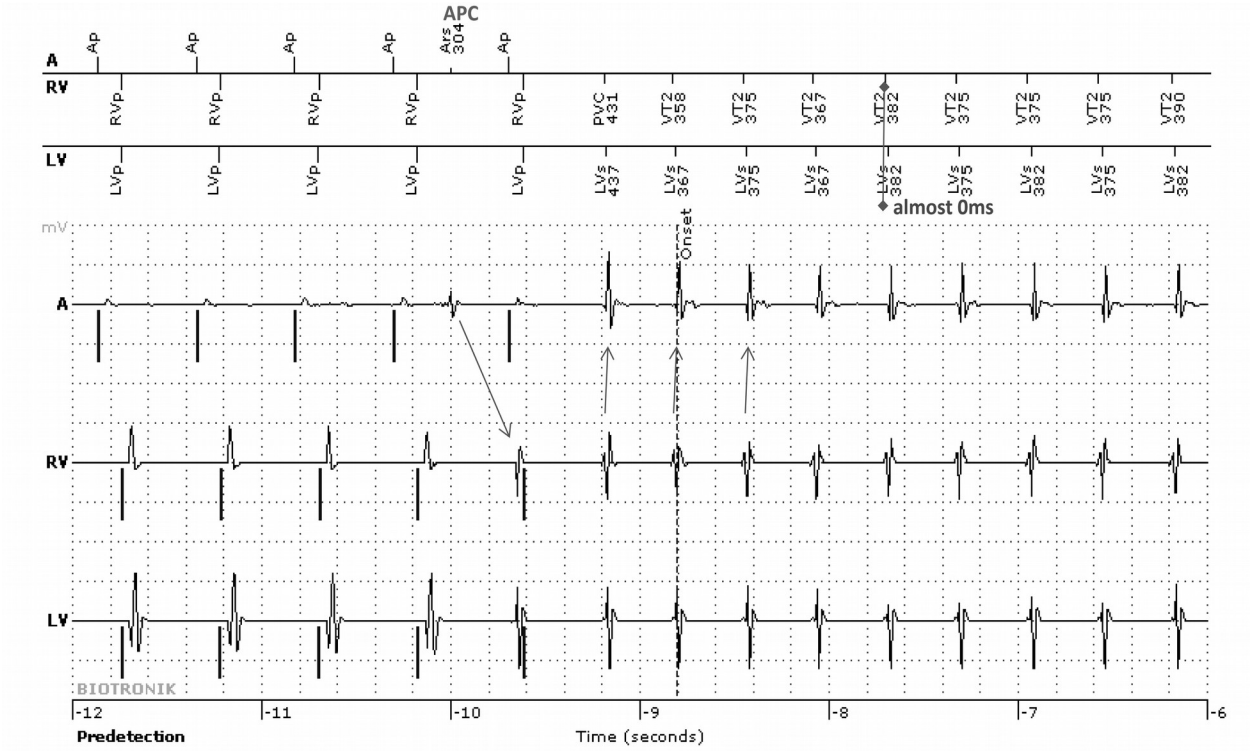

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Figure 5. *AV nodal reentry tachycardia. Same arrangement of the markers and EGM channels as in Fig. 1. Ap = atrial paced events; APC = atrial premature complex; VT2 = ventricular sensed event detected in the VT2 zone. See text for details.*

220 ***Figure 6.*** *AV nodal reentry tachycardia. Same patient as in Fig 5. The marker channels are on top: A = atrium, RV = right ventricle, LV = left ventricle. The configuration of the EGM channels has been changed. FF = far-field electrogram (EGM); A = atrial EGM; RV = right-ventricular EGM; The LV-EGM is not displayed but the LV markers. VT2 = ventricular sensed event detected in the VT2 zone. See text for details.*

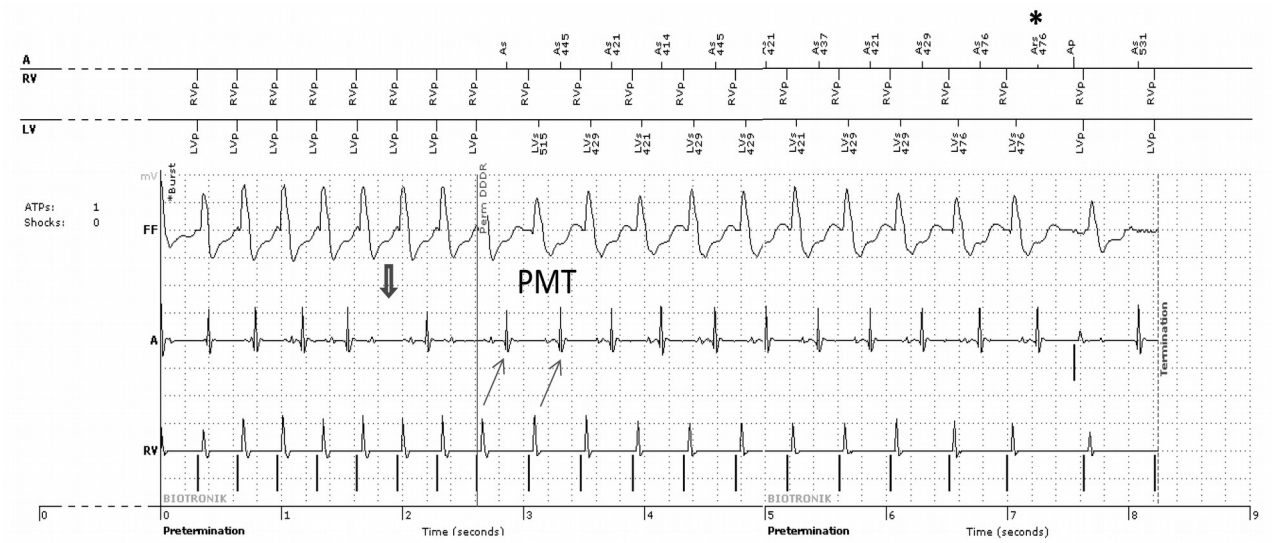

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Figure 7. *Pacemaker-mediated tachycardia (PMT) triggered by the ATP and automatic termination of the PMT. Same arrangement of the markers and EGM channels as in Fig. 6. See text for details.*

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