Pacemaker repetitive nonreentrant ventriculoatrial synchrony. Why did automatic mode switching occur?

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Abstract

Repetitive nonreentrant ventriculoatrial synchrony during dual-chamber pacing is characterized by long intervals alternating with short intervals. This arrangement activated automatic mode switching in a St Jude dual-chamber pacemaker in which the algorithm requires an atrial sensed event for automatic mode switching initiation. Automatic mode switching activation by an atrial sensed event (retrograde P wave) was puzzling because the programmed postventricular atrial period was longer than the retrograde ventriculoatrial conduction time. The explanation is presented in the form of questions and answers to facilitate the understanding of pacemaker function and complex timing cycles.

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Automatic mode switching; Cardiac pacing; Retrograde ventriculoatrial conduction

A Zephyr XL DR DDDR pacemaker (St Jude Medical, Sylmar, CA) was implanted 3 years ago in a 72-year-old man with sick sinus syndrome. Figs. 1 and 2 were recorded when the programmed parameters were as follows: low rate, 60 beats/min; sensor on; upper tracking rate, 140 ppm; paced atrioventricular (AV) delay, 150 milliseconds; sensed V delay, 120 milliseconds (rate response AV delay off); postventricular atrial refractory period (PVARP), 350 milliseconds; postventricular atrial blanking period, 125 milliseconds; ventricular refractory period, 250 milliseconds; atrial tachycardia detection rate, 180 beats/min; automatic mode switching (AMS) base rate, 80 ppm; AMS mode, DDDR; pacemaker-mediated tachycardia detection rate, 120 ppm; and premature ventricular complex atrial pace response on. Telemetry revealed 470 AMS episodes over a period of 7 months; the longest recorded episode was 8 seconds. The total AMS duration for the 7 months was 38 minutes 48 seconds.

Why was AMS activated by long and short atrial intervals?

Automatic mode switching in St Jude devices provides a variety of destinations: DDD(R) → DDI(R) and DDD → DDI in implantable cardioverter-defibrillators and pacemakers and additional options for pacemakers with VDD(R) → VVI(R) and VDD → VVI. Rather than the actual atrial rate or atrial interval, the AMS algorithm in St Jude devices uses a “running average” rate or the filtered atrial rate interval (FARI), also known as the mean atrial rate, filtered or matched atrial rate. The device continuously monitors the spontaneous atrial interval and generates a related but different atrial interval (FARI) that changes according to the duration of the detected prevailing atrial cycle. The device compares the current spontaneous atrial interval with the FARI, which is being continually updated. The FARI is adjusted with every atrial interval composed of sensed (AS) and paced events (AP) in various combinations. Hence, all AS-AS, AS-AP, AP-AS, and AP-AP intervals are counted toward the FARI. P waves detected in the PVARP (but not in the postventricular atrial blanking period) are used as part of the atrial intervals to calculate the FARI. When the current spontaneous atrial interval shortens, the FARI also shortens by 39 milliseconds for every atrial interval shorter than the FARI. When the spontaneous atrial interval lengthens to a value longer than the FARI, the FARI itself lengthens by 25 milliseconds. The device moves toward AMS as long as the spontaneous atrial rate remains relatively fast. Activation of AMS requires the FARI to shorten to the programmed atrial tachycardia detection rate or interval. Upon termination of the rapid spontaneous atrial rate, the device eventually returns to the standard tracking mode when the FARI becomes longer than...
the programmed upper tracking rate interval or the maximum sensor rate interval.

Why did the pacemaker not activate AMS simply on the basis of alternating long and short intervals?

During repetitive nonreentrant ventriculoatrial synchrony (RNRVAS), there were only 2 atrial events: “(AS)” and AP. The short interval of 120 milliseconds (alternating with the long interval) should have carried the FARI to the AMS threshold. However, AMS did not occur because in St Jude devices, it can only be activated by an atrial sensed event (AS). Automatic mode switching was, therefore, activated when rate-adaptive abbreviation of the PVARP released AS for sensing. The sensed atrial event initiated a VP-VP interval of 429 milliseconds (equal to the upper tracking interval). Automatic mode switching then started after the first AS-VP delay. During AMS in this device in the DDIR mode, the PVARP shortens to the postventricular

Fig. 1. Stored recording of an episode of RNRVAS. The electrograms are on top with the ventricular below the atrial electrograms. A ventricular paced event “VP” induces retrograde VA conduction from VP to “AS.” The latter sits in a box with a black background displaying white letters spelling “AS.” This arrangement locates the “AS” in the PVARP. “AS” in the PVARP is designated as “(AS)” for simplicity and to differentiate it from the AS marker, which indicates sensing outside the PVARP. The atrial paced event (AP) is ineffectual because it occurs too close to the atrial myocardial refractory period generated by the preceding (AS) event. RNRVAS continues until the emergence of an atrial sensed event AS beyond the PVARP. This AS event immediately activates AMS. During AMS in the DDIR mode, the rhythm is equivalent to functional VVIR pacing with preserved retrograde VA conduction. The refractory markers (horizontal line below the markers) indicate that AMS shortens the PVARP to the duration of the programmed shorter postventricular atrial blanking period (125 milliseconds). See text for details.
atrial blanking period interval by design. The entire duration of the PVARP is enabled once AMS has terminated.

What is the arrhythmia?

The arrhythmia is RNRVAS.3-6 The predisposing factors include a relatively long retrograde ventriculoatrial (VA) conduction time, a long PVARP, and a relatively short atrial escape interval (often during a sensor-driven lower rate). In this case, a retrograde P wave (labeled AS but depicted in white letters AS on a black background; it indicates an atrial event detected in the atrial refractory period) is trapped in the PVARP, and the subsequent AP is ineffectual because it falls in the atrial myocardial refractory period generated by the preceding P wave. The (AS)-AP interval measures about 120 milliseconds. Therefore, the retrograde VA conduction time is 570 ms - 120 ms = 450 ms, which is basically the same as the calculated value of 286 milliseconds, considering the margin of error in measuring the AR-P interval manually. By using calipers, all the retrograde VA conduction times were equal.

Why did the pacemaker sense the atrial event labeled AS?

How is this possible with a PVARP programmed to 350 milliseconds because the VA conduction time was 296 milliseconds? The PVARP must have been shorter than 350 milliseconds for the pacemaker to sense the P wave. Actually a rate-responsive PVARP had been programmed.1 The shortest duration was programmed at 170 milliseconds. The rate-responsive PVARP in St Jude devices automatically changes the PVARP in response to increases or decreases in the Atrial Fibrillation Suppression Algorithm rate, sensor-indicated rate or the FARI used for AMS (all functions of St. Jude devices). The algorithm begins to operate when the intrinsic rate exceeds 90 beats/min. A low setting (programmed in this case) changes the PVARP by 1 millisecond for each 1-min\(^{-1}\) change in the pacing rate. A high setting changes the PVARP by 3 milliseconds for each 1-min\(^{-1}\) change in the pacing rate. The duration of the ventricular refractory period follows suit and moves toward a minimum value identical to that of the minimum PVARP. Thus, as pacing rates rise, the PVARP shortens until the maximum
sensor rate, maximum tracking rate, or the shortest PVARP setting is reached.

**Where does AMS start and finish?**

The AMS label changed somewhat in St Jude devices over the years as far as placement in recordings. In this generation, the “AMS” label starts with the first P wave that is sensed (and the FARI is already at atrial tachycardia detection interval). Automatic mode switching really starts after the VP linked to the first AS. Any atrial events after the start of AMS label will no longer tracked (D. Davis, personal communication, 2011). In contrast to the initiation of AMS, AS is not required for the termination of AMS. At the end of the AMS sequence, the last “AMS” label is placed on the last ventricular event that is not tracked (ie, the P wave preceding this last ventricular event was also not tracked). Tracking can then occur beyond this point, which is outside AMS. All P waves will be tracked if VA conduction time is less than PVARP.

**What happened during and after AMS?**

When AMS occurred, there was no rhythm to justify its existence, and it terminated quickly. The rhythm during AMS was most probably functional VVIR pacing (in the DDIR mode) with regular, unchanged retrograde VA conduction, although sinus rhythm with first-degree AV block could not be excluded. As soon as AMS was terminated, the device tracked a P wave (at 9.8 seconds) that induced a physiologic AV delay and closed a ventricular interval conforming to the maximum tracking interval of 429 milliseconds (140 ppm). This VP appears to give rise to retrograde VA conduction with a duration similar to that seen before AMS.

**Can RNRVAS continue indefinitely without activating AMS?**

It is theoretically possible. Most cases occur during an activity with a sensor-driven increase in the pacing rate. As shown with the present case, the atrial escape interval terminated with an atrial stimulus too close to the P wave trapped in the PVARP, and AP was, therefore, ineffectual. Upon cessation of the activity, slowing of the sensor-driven rate produces a longer atrial escape interval that will displace AP away from the P wave in the PVARP. This permits atrial capture and restoration of AV synchrony. This mechanism is the reason why RNRVAS episodes on activity are usually self-limited. However, with fixed (non–rate adaptive) timing cycles at rest, RNRVAS could theoretically continue indefinitely. However, it usually terminates because of a perturbation of the timing cycles by spontaneous atrial or ventricular activity.

**Can RNRVAS occur at rest?**

Repetitive nonreentrant ventriculoatrial synchrony can occur at rest according to the programmed parameters and the VA conduction time. Assume a low rate of 80 ppm, AV delay of 250 milliseconds, atrial escape interval of 500 milliseconds, and VA conduction time of 300 milliseconds contained in a PVARP greater than 300 milliseconds. The atrial stimulus would fall 200 milliseconds from the P wave in the PVARP and in all likelihood in its atrial myocardial refractory period. This problem can be prevented by appropriate programming either by a slower lower rare and/or a shorter rate-adaptive AV delay. This increases the duration of the atrial escape interval, pushing the atrial stimulus further away from the P wave in the PVARP.

**How can RVRVAS be prevented on exercise when the atrial escape interval shortens by sensor activity?**

This case suggests that a rate-adaptive PVARP with the shortest minimum PVARP should be able to abort RNRVAS occurring with activity by promoting atrial sensing by virtue of a shorter PVARP.

**Is this a form of preformed mode switching?**

No. The FARI is continually updating on every atrial event, so it does not latch or lock into an “AMS” position when it first reaches ATDI. In our patient, FARI hovered around the ATDI according to the rules governing its shortening or prolongation. If the continuing rolling average of FARI was shorter than the ATDI at the point where the P wave was sensed, then AMS occurred. The peak atrial rate indicates that the fastest atrial to atrial interval AFTER AMS has been initiated.

**Does this patient help us understand how RNRVAS starts?**

Fig. 3 shows the termination of RNRVAS by the above mechanisms. The first atrial sensed event AS at the onset of AMS triggers a ventricular paced event at the ventricular upper tracking interval of 429 milliseconds. This VP rolls out a full PVARP somewhat shorter than 350 milliseconds because of rate adaptation. An atrial event “(AS)” falls again into the PVARP. The next atrial stimulus (AP) occurs too close to the atrial myocardial period generated by the preceding P wave. Therefore, AP is ineffectual and RNRVAS restarts. In this way, RNRVAS begets RNRVAS in certain circumstances governed by the programmed timing cycles. In the absence of a substantial increase in the pacing rate, ventricular premature complexes are the most common initiating and terminating mechanisms.

**Is RNRVAS a rare pacemaker arrhythmia?**

Repetitive nonreentrant ventriculoatrial synchrony has become more common in the last few years because of the importance of minimizing right ventricular (RV) pacing. In pacemakers without special algorithms to reduce or minimize RV pacing, the AV delay is lengthened in the hope of reducing RV pacing. A long AV delay and greater...
use of sensor function on exercise for rate increase predispose to RNRVAS.

**What is the treatment of RNRVAS?**

Most RNRVAS cases seem to occur with activity because of the faster pacing rates. The observations in this case suggest that sensor-driven RNRVAS may be quickly terminated or prevented by programming a rate-dependent PVARP with the shortest minimum duration. The rate-adaptive PVARP was important in this patient because of the relatively long retrograde VA conduction time that required a relatively long PVARP at rest to contain the retrograde P waves.

Although it could be argued that the brief and asymptomatic RNRVAS episodes require no treatment or parameter change, an attempt should be made to eliminate them to avoid attributing all AMS events to RNRVAS, thereby potentially masking more important causes of AMS such as atrial tachyarrhythmias and atrial sensing of far-field R waves. All AMS episodes should be evaluated in details if the pacemaker design allows it to search for these other AMS causes. In this respect, careful attention must be paid to event counter diagnostics.

The situation is compounded by the atrial tachycardia/atrial fibrillation (AT/AF) monitor of St Jude pacemakers. The AT/AF algorithm does not rely on the detection of consecutive intervals shorter than the tachycardia detection interval whose duration is shared by both the AMS and the AT/AF algorithms. The AT/AF algorithm interprets the alternating short and long intervals of RNRVAS in a complex formula, provided the short interval is shorter than the tachycardia detection interval. In our case, the AS-AP interval was about 170 milliseconds, and the tachycardia detection interval was 333 milliseconds. For this reason, many AT/AF counts were registered in the absence of clinical events. In conclusion, RNRVAS can induce AMS, and when AMS is inactive during RNRVAS from a lack of AS events, the pacemaker can register AT/AF episodes. Thus, it would be wise to eliminate all the RNRVAS episodes so that other causes of AMS and AT/AF counting can be detected without interference.

**References**


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Should the cardiac catheterization laboratory be activated?

Answers:

1. An acute inferior myocardial infarction
   Acute inferior myocardial infarction should be suspected in patients with a suggestive history and whose ECG demonstrates ST segment changes, including depression, elevation, and/or dynamic T wave changes. Elevation of serum cardiac enzymes provides support for acute myocardial infarction. Q waves are evidence for transmural ischemia, which would be accompanied by the expected ST-T changes, and for prior infarction.

2. An ectopic atrial rhythm
   Ectopic atrial rhythms originate from foci within the atria and lead to varying p wave morphologies including, but not limited to, inverted p waves in the frontal leads if the ectopic pacemaker’s location is near the atrioventricular (AV) junction. The inverted p wave indicates that the atria are being depolarized retrogradely. Ectopic atrial or junctional rhythms may terminate pauses (escape foci and rhythms) and may occur normally or result from sinus nodal dysfunction or from any condition that accelerates their rate of depolarization (“accelerated” rhythms) (Fig. 2). Depending on their cause and rate, these rhythms may become the dominant cardiac pacemaker. In this ECG, what appear to be q waves due to an inferior wall infarct in leads II, III, and F actually represent retrograde (inverted) p waves.

3. Accelerated ventricular rhythm
   Accelerated ventricular rhythms are rhythms originating in ventricular tissue with a rate faster than the intrinsic (“idioventricular”) ventricular rhythm (30 to 40 per minute) but slower than ventricular tachycardia (100 per minute). They are characterized by abnormal QRS complexes not conducted from the atria, and therefore, AV dissociation is expected to be present. Although this rhythm may be associated with myocardial infarction, acute or old, it is not usually associated with ischemia. This patient’s rhythm is not an accelerated ventricular rhythm because the p waves that precede each QRS complex are clearly not part of the QRS complex (Figs. 2 and 3) and both the QRS complexes and the ST segments are the same as in sinus rhythm.

4. WPW conduction
   WPW conduction involves ventricular preexcitation by AV conduction over an accessory pathway as well as through the normal AV node-His-Purkinje system; each pathway may be used to varying degrees. WPW conduction is classically characterized on the ECG by a shortened PR interval (usually <0.12 second) and a delta wave representing preexcitation, thus widening the QRS complex. The delta wave is a slurred deflection that deforms the initial part of the QRS and typically lasts 0.03 to 0.06 seconds. In the tracing above, the PR interval is normal in duration, there is no evidence for a delta wave, and the QRS complex is narrow.

![Fig. 3. Enlargement of lead II from EKG seen in Fig. 2. Color illustration online.](image-url)

References