Desynchronization in a cardiac resynchronization device induced by a pacemaker-mediated tachycardia algorithm

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This report describes the occurrence of desynchronization in a patient with a cardiac resynchronization device programmed with an active pacemaker-mediated tachycardia algorithm based on AV delay modification. Desynchronization was precipitated by sinus tachycardia and the abrupt return of the prevailing AV delay that followed the periodic prolongation of the AV delay mandated by activity of the algorithm. Prevention of desynchronization in this setting requires programming a right ventricular upper rate interval longer than the sum of the programmed ventriculoatrial interval and the AV delay.

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1. Introduction

Biotronik devices used for cardiac resynchronization therapy (CRT) can sense from the left ventricular (LV) lead [1]. 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. In Biotronik CRT devices, LV stimulation therapy does not affect traditional DDD timing cycles. However, LV sensing itself may occasionally precipitate desynchronization by realigning the LV timing cycles induced by an abrupt prolongation of the AV delay [2,3]. This report describes such an occurrence in a patient with a pacemaker-mediated tachycardia (PMT) algorithm.

2. Case report

A 63 year-old man with refractory heart failure, complete left bundle branch block and a left ventricular ejection fraction of 20% received a Biotronik Ilivia 7 HF-T device (Biotronik, Berlin, Germany) for cardiac resynchronization therapy (CRT) and defibrillation. The parameters were programmed as follows: DDD mode, biventricular pacing, lower rate 60 ppm, dynamic AV-delay (AVD) 150/120 ms with a sense compensation of - 40 ms, post-ventricular atrial refractory period (PVARP) 225 ms, right ventricular upper tracking rate 160 ppm, left ventricular (LV) maximum trigger rate 160 ppm (LV upper rate interval = LVURI = 375 ms), V−V delay 0 ms. The PMT termination algorithm was activated with a ventriculoatrial (VA) criterion of 350 ms. The device detected and stored one episode of interruption of CRT. Figs. 1 and 2 shows a part of the stored episode when desynchronization occurred. The patient had been treated with controlled release metoprolol for over a year and this was continued with 190 mg a day after device implantation. The resting heart rate at follow-up times was often satisfactory at 62–84 per min. The medical record over a period of 12 months documented that the patient had occasionally skipped several doses of metoprolol. An omitted dose of metoprolol and unusual activity most probably explain the recorded sinus tachycardia by the implanted device.

3. Background

The mechanism of desynchronization in this case is based on the design and function of a separate LV upper rate (interval, LVURI) found in Biotronik CRT devices. CRT-defibrillation (D) devices of the
Figure 1. PMT confirmation test. The marker channels are on top (A = atrium, RV = right ventricle, LV = left ventricle). The electrograms are below the marker channels. As = atrial sensed event, RVp = right ventricular paced event, LVp = LV paced event, RVs = RV sensed event, LVs = LV sensed event, VA criterion = 350 ms, AVD = programmed AV delay 80 ms, pAV = prolonged AV delay = 130 ms, LVURI = left-ventricular upper rate interval 375 ms. See text for details.

Biotronik I3-family (Intica, Ilivia and Inlexa) [2] offer trigger events for electrogram (EGM) recordings including the interruption of CRT pacing [2,3]. The detection of CRT pacing interruption is fulfilled if at least 20 LV paced events (LVp) are inhibited by the LVURI within a sliding observation window of 48 RR cycles [right-ventricular sensed (RVs) or paced events (RVp)]. The duration of the EGM recordings (initiated by interruption of CRT) is 30 seconds which gives no data about the actual duration of the desynchronization episode beyond 30 s. The DDD mode (default setting) provides an automatic algorithm to detect, confirm and terminate a pacemaker-mediated (endless loop) tachycardia (PMT). A device assumes the presence of a pacemaker-mediated tachycardia (PMT) if atrial sensed events (As) fall into the VA criterion of 350 ms (default value) after a ventricular paced event (Vp). The PMT detection algorithm collects 8 consecutive Vp-As intervals which are shorter than the VA criterion. If these 8 intervals were found, the stability of the Vp-As intervals will be checked. The average will be calculated over the 8 Vp-As intervals, and the deviations from the mean have to be less than 25 ms for each of the single Vp-As periods. If the stability criterion is met, the PMT confirmation test aims to differentiate a PMT from sinus tachycardia. The AV delay (AVD) will be prolonged once by 50 ms in order to check whether the retrograde VA conduction follows the delayed Vp event or not. If the Vp-As interval remains constant — even after a late Vp — a PMT is confirmed. Otherwise a sinus tachycardia is confirmed because the PP intervals remain unchanged. If a PMT is confirmed, the termination is based on a single initiation of an extended PVARP of 400 ms (equal to the VA criterion + 50 ms). If a PMT suspicion is discarded, and a sinus tachycardia is confirmed, the termination attempt will be skipped and pacing with the programmed AVD is continued.

4. Analysis of the recordings

In Figs. 1 and 2 the atrial intervals gradually shorten and reach values less than 430 ms. Prolongation of the AV delay is repeated every 8 cycles as long as atrial sensed events (As) fall into the VA criterion. The bold numbers on top demonstrate that all As events are detected inside the VA-criterion. The left ventricular (LV) paced event (LVp) and the right ventricular (RV) paced event (RVp) are simultaneous. On the left (A) resynchronization was not interrupted by the impact of the longer test AV delay because the LV upper rate interval (LVURI) had terminated by the time the next LVp (ending a short AV delay with RVp) was scheduled to be released. On the right side (B), the atrial intervals are slightly shorter so that the switch from the prolonged AV delay back to the programmed AV delay was associated with an early RVp event at a time when the LVURI has not timed out. The AV delay prolongation in B, has enabled a sensed conducted ventricular beat (*RVs) with a 20 ms delay in triggering an LVp. The magnification of part B in Fig. 2 shows the trigger delay of 20 ms between the RVs-event and the triggered LVp. The initiation of the LVURI is subsequently delayed by 20 ms. The following short AV delay was then associated with inhibition of LVp because the LVp was scheduled to fall within the slightly delayed LVURI. The first inhibited LVp then sets the stage for the perpetuation of desynchronization by detection of predominant LVs events. The continuation of RV pacing is possible despite the increased atrial rate because the RV upper rate interval (RVURI 375 ms) had terminated prior to the scheduled emission of RVp.

The detection of interrupted CRT is fulfilled by a 20-out-of-48 criterion, whereby at least 20 LVp events were inhibited due to LVURI (375 ms) in a sliding observation window of 48 RR cycles (RVs and RVp events; device-defined premature ventricular complexes [1] do not count). The cursive numbers count the 20 inhibited LVp events until CRT interrupt is detected (Fig. 3). Desynchronization persists even after the end of the stored recording. The oblique arrow shows the interventricular conduction delay (IVC), the interval between the RVp event to LVs event = 150 ms.

4.1. Clinical correlation

The clinical impact of the desynchronizing episode was unimpressive and unrecognized in the device statistics. The statistics encompassed 16 days during which the desynchronization episode occurred. The data showed 100% CRT pacing both in the total number of events and the trend curve. The trend curve showed no outlier. These results provide clear evidence that the period of
desynchronization was rather short and had no impact on the daily average. Clinically, the patient reported no unusual symptoms.

5. Discussion

The present observation of desynchronization involved a device from a specific family of Biotronik CRT devices. However, the potential of desynchronization secondary to LV sensing is common to all Biotronik CRT devices produced since 2006 (when the Lumax 340 HF-T device was released) because they have all been designed with the same response to LV sensing as that of devices described in this report [1,3,4]. The rather abrupt prolongation followed by shortening of the AV delay provided the disruptive event that realigned the LV timing cycles and caused desynchronization. The predominant manifestation of desynchronization consists of the release of displaced LV sensed events [2,3]. The first release of a displaced LV sensed event then starts a self-perpetuating process with the timing of these LV sensed events continually inhibiting the scheduled programmed LV paced event. Hence the designation of LVURI-lock-in Ref. [1].

5.1. At what heart rate can desynchronization be initiated?

The risk of inducing desynchronization during activity of the PMT confirmation algorithm by modification of the AV delay is obviously a fast atrial rate which may be, however, far slower than the programmed RV upper rate of 160 ppm as in this case. This critical atrial cycle length for desynchronization can be calculated.
by the following formula: $PP \leq AVD + VA$ criterion ($PP = \text{atrial cycle length}$, $AVD = \text{AV-delay}$, $VA$ criterion). By reprogramming the RV upper rate to 160 ppm, the LVURI will adapt automatically, but the intervention rate for the PMT algorithm will not adapt. The intervention rate for the PMT test was reached in this patient at 140 bpm corresponding to a cycle length of $VA$ criterion + AVD = 350 + 80 = 430 ms. The device will see this rate earlier before reaching the RV upper rate 160 ppm so that cessation of CRT occurs at a rate far below the RV upper rate. Desynchronization by the PMT protection algorithm will only occur if the upper RV rate interval is shorter (rate faster) than the sum of the $VA$ criterion plus the AV delay as it was in this case with a programmed RV upper of 160 ppm. Desynchronization will not occur with default values of an RV upper rate of 130 ppm (460 ms) and an AVD 80 ms and $VA$ criterion 350 ms, because the sum of the $VA$ criterion and the default AVD is shorter than the RV upper rate interval (460 ms). Thus, the avoidance of desynchronization in sinus rhythm requires that the RV upper rate interval be longer than the sum of the $VA$ criterion and the rate-adaptive AV delay.

5.2. At what heart rate can resynchronization return?

The interruption of CRT can persist much longer than the 30 sec desynchronization recording time (and potentially for hours?) because the sinus rate to restore CRT has to be much lower than the rate at the time of desynchronization initiation. Desynchronization will persist until the atrial rate decreases below the critical desynchronization rate [2]. This critical rate can be calculated by the following formula: $PP \geq IVC + LVURI$ ($PP = \text{atrial cycle length}$, $IVC = \text{interventricular conduction delay}$ ($RVp - LVs$)). When using the patient-related parameters ($IVC = 150$ ms, $LVURI = 375$ ms), the critical atrial cycle length = 150 ms + 375 ms = 525 ms corresponding to an atrial rate of 115 bpm which is slower than the rate in Figs. 1 and 3 [2]. The atrial rate would have to decrease below 115 bpm for resynchronization to recover.

5.3. How to prevent desynchronization by the PMT confirmation algorithm?

The need for the PMT terminating algorithm should be carefully evaluated according to the presence or absence of retrograde $VA$ conduction. This was not performed in this patient at the initial programming session when there was no evidence of sinus tachycardia. If it is necessary to retain the PMT protection algorithm despite an increased RV upper rate, the $VA$ criterion can be shortened. For example with a short $VA$ criterion of 280 ms, the sum of the AV delay + $VA$ criterion (e.g. 80 + 280 = 360 ms) would be shorter than the RV upper tracking rate interval of 375 ms. The potential interference from sinus tachycardia must always be carefully evaluated when the algorithm is active and its risk suppressed pharmacologically if necessary.

6. Conclusion

Desynchronization of a CRT device was observed in a patient without retrograde ventriculoatrial conduction who developed sinus tachycardia which activated the PMT algorithm function that had remained turned off in the default mode. Default settings are only helpful as long as the DDD- and CRT-related parameters remain unchanged. If one begins to change the RV upper rate, the LV upper rate will adapt automatically, but the intervention rate for the PMT algorithm will not. The observations underscore the importance of evaluating the status of other default parameters before ending a programming session so that an optimal resynchronization therapy can be achieved. The potential for the development of sinus tachycardia must always be ruled out.

References

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