# Induction of a fast ventricular tachycardia by a CRT-D device. What is the mechanism? Device Rounds

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**Abstract**

A fast ventricular tachycardia was apparently induced in a patient with a CRT-D device by the delivery of a pacemaker stimulus whose timing corresponded with the timing of the QRS complex in the far-field electrogram. Appropriate programming of the device might have prevented this complication.

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**Abstract**

A fast ventricular tachycardia was apparently induced in a patient with a CRT-D device by the delivery of a pacemaker stimulus whose timing corresponded with the timing of the QRS complex in the far-field electrogram. Appropriate programming of the device might have prevented this complication.

**Key words.** Ventricular tachycardia, cardiac resynchronization, implantable cardioverter-defibrillator, left ventricular sensing, electrogram.

*Abbreviations*

As = Atrial sensed event, AT = Atrial tachycardia, Ars = Atrial sensed event in the atrial refractory period, RV = Right ventricle, LV = Left ventricle, RVp = Right ventricular paced event, LVp = Left ventricular paced event, LVs = Left ventricular sensed event, RVs = Right ventricular sensed event, PVARP = Post ventricular atrial refractory period, VF = Ventricular tachycardia interpreted as ventricular fibrillation by the device, FF = Far field.

Biotronik CRT-D devices with LV sensing have added a new dimension to programmability and evaluation of pacemaker function (1-4). Left ventricular sensing was designed to prevent the delivery of a left ventricular stimulus into the left ventricular vulnerable period.

The parameter *"LV T-wave protection "* (LVTP) can either be turned on or off. If LVTP is on, the left-ventricular upper rate interval (LVURI) will be initiated by LVs-events, LVp-events and inhibited LVp events. If LVTP is off, the LVURI will still be initiated by LVp events or inhibited LVp events but not by LVs events. In this respect, some workers have advocated turning off the LVTP function to prevent desynchronization or so-called CRT pacing interrupt (3).

In this case, a CRT-D device (Ilivia 7 HF-T QP, Biotronik) was implanted according to standard guidelines in a patient with episodic VT. The device was programmed with a VT zone at 154 bpm and a VF zone at 200 bpm. The LV pacing voltage was 4.5 V with a pulse width of 1.0 ms. The LV maximum trigger rate was 150 bpm (corresponding to an LVURI of 400 ms) and LVTP was turned off. With the LVTP turned off, LV sensing is still depicted on the marker channel as LVs, but this representation is used only for diagnostic purposes because LVs cannot start an LVURI.

*Triggering* upon right ventricular sensing (RVs) was also programmed as shown by the delivery of a left ventricular pacing (LVp) event upon all the right ventricular sensed (RVs) events (Figure 1A). Triggering did not occur upon sensing VF events during VT because of the occurrence of repeated inhibited LVp events initiating an LVURI.

What is the pacing mode?

The device is functioning with biventricular pacing in the DDIR mode at 75 bpm in response to automatic mode switching initiated by atrial fibrillation and with no sensor activity at this time. The lower rate interval of 800ms timed out after the first RVs event, hence RVp and LVp were released simultaneously. There was also intermittent failure of LV capture despite the increased pacing output. The first stimuli probably captured only the RV. The PVARP was 225ms. If the PP interval is shorter than the atrial tachycardia intervention interval (here 300 ms), the As- or Ars annotation will be replaced by AT markers. The origin of the first LVs event is puzzling.

Was ventricular tachycardia induced by a ventricular stimulus falling in the vulnerable period of LVs 156?

After the simultaneous delivery of RVp and LVp, RVs 398 triggers an LVp event which is followed by an LV sensed event (LVs 156). This LVs marker did not start an LVURI because LVTP was turned off.

A closer look at only the marker channels in Figure 1A and B might suggest that a fast VT was induced by LVp (triggered by RVs 414) falling into the LV vulnerable period of an LV event (LVs 156). One might also wonder whether LVp triggered by RVs 398 actually failed to capture the LV resulting in RVs 398 being conducted to the LV as LVs 156 after an intraventricular delay of 156 ms. With the absence of a corresponding LV electrogram, the FF electrogram shows no deflection corresponding to LVs 156. Consequently, LVs 156 appears to have originated from sensing the tail end of the QRS complex linked to the slow VT. The third LVp fell within the QRS complex of the slow VT as shown in the FF electrogram (Fig 1B). This event seems have caused an acceleration of the slow VT (as manifested by a shorter cycle) identical to that of the subsequent stable but faster VT. This is discussed in more detail in the answer to question 3.

Was ventricular tachycardia induced by the delivery of a pacemaker stimulus whose timing corresponded with the QRS complex in the far-field electrogram?

In Figures 1A and B, a slow VT starts in the middle of the tracing with RVp-LVp whose cycle ends with the second LVp (triggered from RVs 398) and this LVp falls before the QRS complex in the FF channel. The second LVp starts a cycle that is shorter than the first cycle. This cycle ends with the third LVp (triggered by RVs 414) which is delivered on the QRS complex specifically during the descending limb of the QRS complex (Fig 1 B). The third LVp (triggered by RVs 414) starts a cycle whose duration is similar to that of the subsequent induced fast and stable VT (cycle length 257 to 281 ms). The second LVp event may have influenced the duration of the second cycle but it is the third LVp that seems to have initiated the fast VT.

**Discussion**

Although the fast VT might have been coincidental, one could postulate that its induction mechanism was related to the altered electrophysiologic arrhythmic milieu produced by the third LVp (regardless of LV capture) falling within the QRS complex of the slow VT an effect similar to the phenomenon originally described by Luceri et al (5). The shortening of the second cycle of the VT was probably also produced by the above mechanism. In another episode shown in Figure 2A and B, the stable and fast VT was induced basically by the same manner as in Figure 1A and B.

Based on the above mechanism, prevention of the fast VT would entail turning off the triggering function and/or prolonging the LVURI longer than the slow VT cycle so that a triggered LVp would be inhibited and therefore not released. In addition, LVTP could be turned on as the initiation of LVURI by LVs events would also contribute to inhibit triggered LVp events which could meet either the LV vulnerable phase or upon right ventricular QRS complexes.

The consequences of a ventricular stimulus falling on a QRS complex has been largely ignored. Nevertheless, it should be considered in puzzling cases as a cause of VT initiation or acceleration in CRT-D devices programmed with a triggering function but deactivated LVTP.

**Conclusion**

The recordings show how a CRT-D device could have initiated ventricular tachycardia in a predisposed patient by the delivery of a pacemaker stimulus into the QRS complex of a spontaneous beat when the LVPT is programmed off. In this case, turning the LVTP on with a relatively long LVURI and/or turning triggering off, might have prevented this complication. This case demonstrates that the LVTP function can be beneficial in special circumstances despite the report of Haeberlin et al (3) who advocated the routine deactivation of the LVTP function.

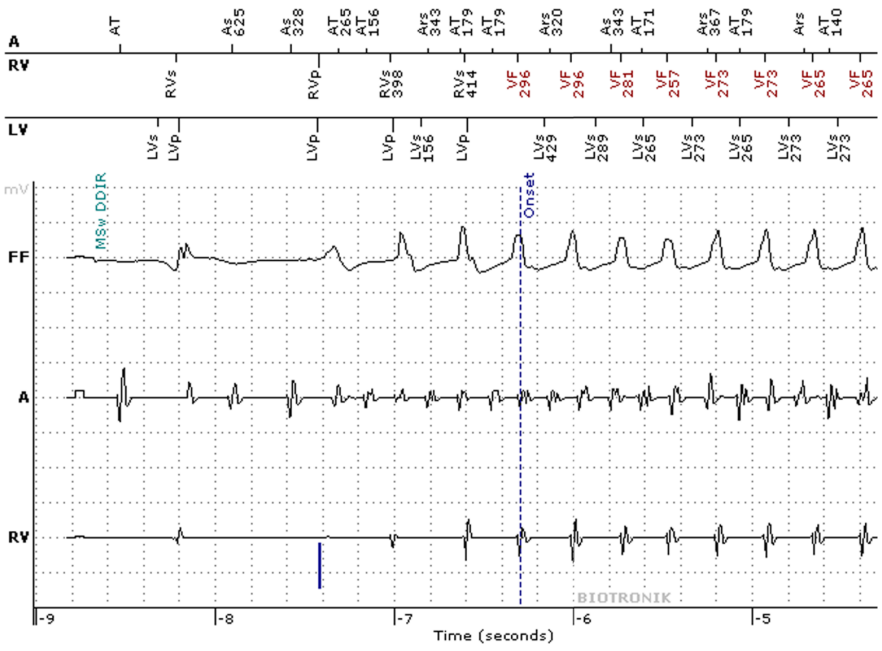


Figure 1A

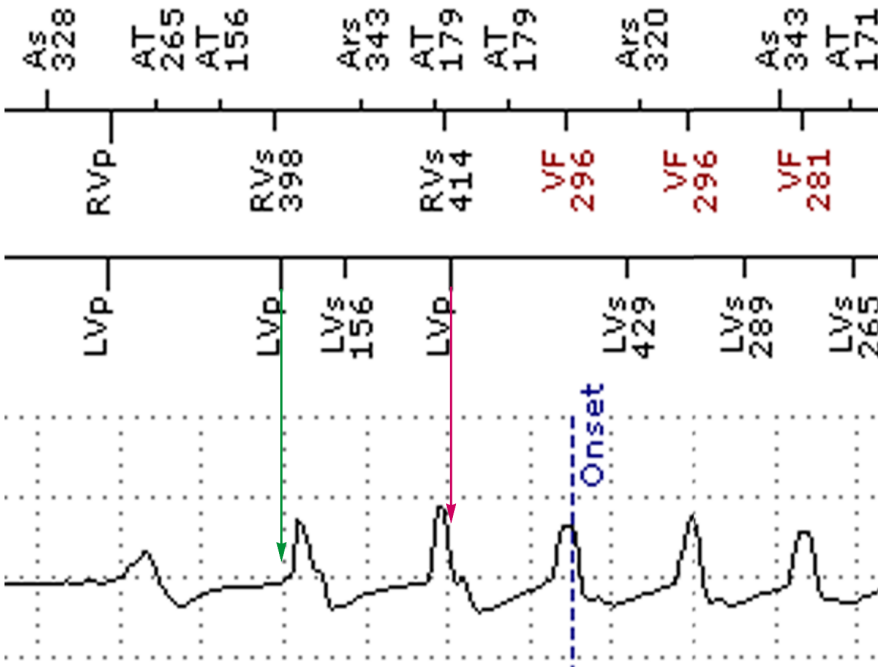


Figure 1B

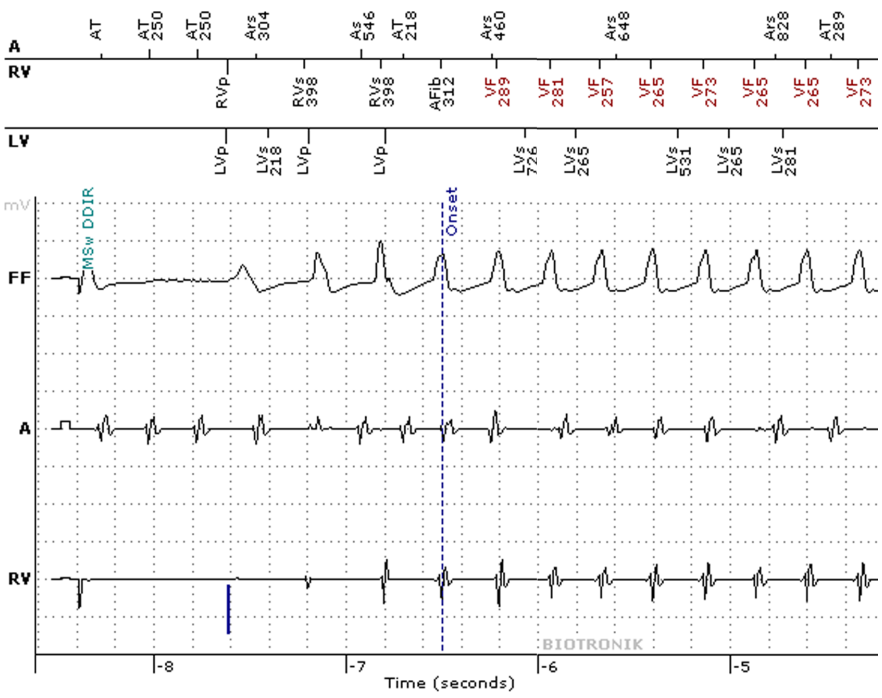


Figure 2A

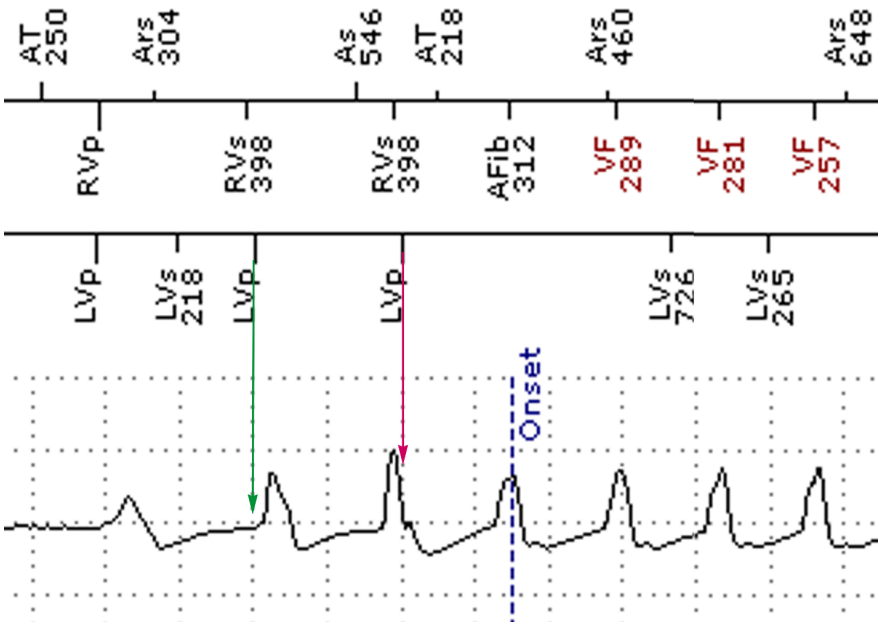


Figure 2B

**Legends**

Figure 1A. Induction of VT. Starting at the top, A = atrial marker channel, RV = right ventricular marker channel, and LV = left ventricular marker channel; then FF = far-field electrogram, A = atrial electrogram, and RV = right ventricular electrogram. Note, the left ventricular electrogram is not displayed.

AT = atrial sensed event classified in the atrial tachycardia zone. As = atrial sensed event, Ars = atrial sensed event in the post-ventricular atrial refractory period (PVARP). RVs = right ventricular sensed event, RVp = right ventricular paced event, VF = right ventricular sensed event classified in the VF zone, LVs = left ventricular sensed event, and LVp = left ventricular paced event. MSw DDIR indicates that the device functions with automatic mode switching. See text for details.

Figure 1B. Magnified portion of Figure 1A. Abbreviations as in Figure 1A. The triggered LVp event on RVs 398 is placed prior to the QRS complex (first vertical arrow). The triggered LVp event on RVs 414 is delivered on the QRS complex specifically during the descending limb of the QRS complex (second vertical arrow). The descending limb of the QRS complex shows a notch which is absent in the other QRS complexes. If LVTP would have been turned on, the triggered LVp during the descending limb of the QRS complex would not have been released.

Figure 2A. Induction of VT. Abbreviations as on Figure 1A. There are unsensed low amplitude LV signals which fall intermittently below the sensing threshold of 1.6 mV. An Afib marker (Afib 312) will be displayed if a right-ventricular sensed event is detected inside the VT zone, and if the SVT discrimination algorithm "SMART" has classified the rhythm as atrial fibrillation. LVs 218 appears to represent sensing of the tail end of the QRS complex. See text for details.

Figure 2B. Magnified portion of Figure 2A. Abbreviations as in Figure 1A. Note how the third LVp falls on the QRS complex as shown in the FF channel.

**References**

1. Haeberlin A, Ploux S, Noel A, Chauvel R, Welte N, Marchand H, Haissaguerre M, Ritter P, Bordachar P.J. Left ventricular sensing in cardiac resynchronization devices-opportunities and pitfalls for device programming. J Cardiovasc Electrophysiol. 30:1352-1361, 2019.

2. Barold SS. Left ventricular sensing by cardiac resynchronization devices. Pacing Clin Electrophysiol.42:1081-1085. 2019.

3. Haeberlin A, Ploux S, Noel A, Chauvel R, Welte N, Marchand H, Haïssaguerre M, Ritter P, Eschalier R, Bordachar P. Causes of impaired biventricular pacing in cardiac resynchronization devices with left ventricular sensing. Pacing Clin Electrophysiol.3:332-340, 2020.

4. Barold SS, Kucher A. Understanding the timing cycles of a cardiac resynchronization device designed with left ventricular sensing. Pacing Clin Electrophysiol.37:1324-37, 2014.

5. Luceri RM, Ramirez AV, Castellanos A, Zaman L, Thurer RJ, Myerburg RJ. Ventricular tachycardia produced by a normally functioning AV sequential demand (DVI) pacemaker with "committed" ventricular stimulation. J Am Coll Cardiol. 1:1177-.9, 1983.