Tachycardia Induction Due to Inappropriate Implantable Cardioverter Defibrillator Therapy: What Is the Mechanism?

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Antitachycardia therapies administered by implantable cardioverter defibrillators (ICDs) can occasionally prove proarrhythmic due to induction of ventricular tachyarrhythmias. In this report we present the case of a tachycardia induction as a result of inappropriate therapy delivery in an ICD recipient. Detailed analysis of the stored electrograms facilitated the delineation of the underlying mechanism of the recorded tachycardia.

Case presentation

A 73-year-old patient had received a dual-chamber ICD (St Jude Medical Epic DR) with the indication of ischaemic cardiomyopathy, severely impaired systolic function (ejection fraction 25%), syncope occurrence and induction of syncope ventricular tachycardia during an electrophysiological study. The initial device programming is presented in Figure 1. During an elective patient follow up, episode retrieval reported the occurrence of a “ventricular tachycardia” (VT) in the slow VT zone, which was initially treated unsuccessfully with two consecutive antitachycardia therapies. The administration of the next programmed therapy (5J shock) was aborted because the tachycardia rate fell below the programmed limit of the slow VT detection zone.

Discussion

A detailed analysis of the stored electrograms can facilitate the interpretation of the detected tachyarrhythmias (Figures 2 & 3). Initially, the stored rhythm strip presents a tachyarrhythmia with equal atrial and ventricular rate but varying cycle length. During the stored tachycardia, changes in the A-A interval precede changes in the V-V interval, resulting in a stable A-V interval but a varying VA interval. These findings are indicative of a supraventricular rhythm with 1-to-1 atrioventricular conduction, while the finding that the atrial activation is not linked to the ventricular activation is indicative of an atrial tachycardia. With acceleration of the atrial tachycardia and maintenance of 1-to-1 atrioventricular conduction, the ventricular rate falls within the predetermined slow VT detection zone. Furthermore, the detected tachycardia satisfies the sudden onset criterion, which is the sole discriminator activated in the V=A branch (equal atrial and ventricular rate).
Tachycardia Due to Inappropriate ICD Therapy

![Figure 1. Programmed parameters as retrieved during the patient’s follow-up visit.](image)

Based on the programmed settings (Figure 1), therefore, since the sudden onset criterion is indicative of VT, the programmed therapies are delivered stepwise with an initial delivery of an 8-pulse burst ventricular pacing train. During the first burst (cycle length of 328 ms) the ventricle is captured, as evidenced by a change in the ventricular electrogram morphology, but the atrial rhythm remains unaffected, with an unchanged atrial electrogram morphology. The inability to entrain the atrium during the tachycardia with ventricular overdrive pacing usually occurs when the ventriculo-atrial block cycle length is longer than the tachycardia cycle length, and this finding has been reported to associate with an 80% positive predictive value for atrial tachycardia. After the first burst therapy, the tachycardia remains unaffected, with its rate remaining within the slow VT zone, and therefore the second burst therapy is delivered at a slower pacing rate than the first burst therapy (cycle length of 332 ms). It should be noted that the second antitachycardia pacing therapy is administered without re-evaluating the satisfaction of the sudden onset criterion, since when the sudden onset criterion has already been met within an episode, it is considered met for the whole duration of the index episode. The second burst train is delivered and the ventricular overdrive pacing also results in acceleration of the atrial rate to the pacing rate in the last four beats, since the coupling interval of the last four A-A intervals equals the pacing cycle length (STIM-STIM interval in Figure 2), while the atrial electrogram morphology is different than the initial form, denoting the retrograde atrial depolarisation.

After the termination of the second burst pacing train, a tachycardia with the same ventricular electrogram morphology persists within the slow VT detection window but gradually decelerates and stabilises at a cycle length of 414 ms. Therefore, the administration of the next programmed therapy, a 5-Joule energy shock, is aborted and the patient remains in a tachycardia slower than the programmed detection rate.

The tachycardia following the second antitachycardia pacing therapy is characterised by equal atrial and ventricular rates (V=A rate branch). The differential diagnosis would include a supraventricular tachycardia with 1-to-1 atrioventricular conduction or a ventricular tachycardia with 1-to-1 ventriculo-atrial conduction. The ventricular electrogram morphology during the finally recorded tachycardia is identical to that during the baseline supraventricular rhythm, thus strongly disfavouring the probability of a ventricular tachycardia. The occurrence of a ventricular tachycardia with a similar morphology to the intrinsic rhythm, as would be the case in a septal VT, is highly unlikely, taking into consideration the identical ventricular electrogram morphology.

Therefore, the finally recorded tachycardia is of supraventricular origin and the differential diagnosis includes the initially recorded atrial tachycardia, a different type of atrial tachycardia, an atrioventricular nodal re-entry tachycardia and an atrioventricular
Figure 2. Retrieval of the index tachyarrhythmia episode.
re-entry tachycardia. The atrial electrogram morphology of the second tachycardia is distinctly different from the initially recorded atrial electrogram, thus excluding the persistence of the initial type of atrial tachycardia. The almost simultaneous atrial and ventricular depolarisation also excludes atrioventricular re-entry tachycardia.\textsuperscript{1,4,5} The analysis of tachyarrhythmia initiation facilitates the elucidation of the underlying mechanism. The last retrograde atrial beat is conducted to the ventricle with a characteristically long atrioventricular delay (Figure 3, red circle and arrow) of 620 ms (suggestive of a “jump” in atrioventricular conduction), where simultaneous atrial activation results in an atrial electrogram morphology that is identical to that during retrograde atrial activation by the second burst train. These characteristics are typical of atrioventricular nodal re-entry tachycardia, with activation of the ventricle through the slow pathway and retrograde atrial activation via the fast pathway (slow-fast).

Figure 3. Episode retrieval (continued).
The patient was offered the option of catheter ablation of the atrioventricular nodal re-entry tachycardia but he refused. Therefore, we proceeded with titration of the administered beta-blocker dosage and re-programming of the slow VT detection zone at higher ventricular rates. In the next patient follow up, which occurred 3 months later, no tachyarrhythmia episodes were recorded.

**Conclusion**

Atrioventricular nodal re-entry tachycardia is occasionally the cause of delivery of inappropriate therapies among ICD recipients. To our knowledge, this is the first case report describing the induction of an atrioventricular nodal re-entry tachycardia by an inappropriate antitachycardia therapy. The diagnosis was based on the mode of tachycardia initiation, the timing of atrial and ventricular activation and the evaluation of ventricular as well as atrial electrogram morphology. Thorough interpretation of stored electrograms facilitates the diagnosis of tachyarrhythmias in ICD recipients and thus contributes to their improved management and the avoidance of inappropriate therapies.

**Reference**