Postural change-dependent T-wave oversensing resulting in the administration of inappropriate shocks

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1. Introduction

T-wave oversensing in implantable cardioverter defibrillators remains a problem that requires a solution [1–5]. Although this phenomenon may be a manufacturer-related technical problem, several situations have been reported to cause or contribute to the onset of T-wave oversensing [6–16]. We report here a case of inappropriate shock administration that was caused by T-wave oversensing and that was induced by the patient’s postural changes.

2. Case report

A 68-year-old man with dilated cardiomyopathy (left ventricular ejection fraction, 15%) and non-sustained ventricular tachycardia received an implantable cardioverter defibrillator. Even though his cardiac status had greatly improved 2 years later after β-blocker therapy, he experienced 2 episodes of sudden shock when he was squatting in a bathroom without any preceding symptoms. His serum electrolyte and plasma glucose levels were normal. Interrogation of the device revealed that the shock was caused by sinus tachycardia and T-wave oversensing. A number of episodes of non-sustained ventricular tachycardia due to T-wave oversensing was also recorded. Follow-up interrogation of the device with the patient in the supine position could not reproduce the T-wave oversensing. We were able to elicit T-wave oversensing only after reproduction of the patient’s clinical situation using isoproterenol and postural changes (i.e., sinus tachycardia and squatting). This case suggests that sudden increases in non-sustained ventricular tachycardia events may be caused by T-wave oversensing, and postural changes should be taken into consideration in such situations.

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of 113 beats/min, where the R–T and T–R intervals were approximately 250 ms. Premature ventricular contractions interrupted consecutive counting repeatedly during sinus tachycardia and lasted >2.5 min so that inappropriate therapies were not administered. Interrogation of the ICD in the supine position did not reveal T-wave oversensing (Fig. 2). The intrinsic ventricular amplitude had been 8–9 mV at the time of implantation and was 5 mV after the event, although the surface electrocardiogram (ECG) showed no change. We decreased the sensitivity from 0.3 mV to 0.5 mV, increased the detection rate of the ventricular tachycardia zone from 150 to 162 beats/min, increased the number of intervals to detect (NID) of VF and FVT from 18/24 to 24/32, and increased the dose of carvedilol to 30 mg in order to slow the sinus rate. However, multiple episodes of nonsustained ventricular tachycardia that were caused by T-wave oversensing were still recorded during a 1-week period, and the patient complained of dizziness because of orthostatic hypotension.

We next reviewed the shock episodes and intracardiac electrograms and found that the episodes occurred only when the patient had sinus tachycardia and was squatting. Several methods were considered to troubleshoot this problem. We decreased the sensitivity and increased the NID and detection rate of ventricular tachycardia in order to satisfy the combined count criterion; however, a number of nonsustained ventricular tachycardias with short R–T and T–R intervals (about 250 ms) were also recorded. Therefore, changes in these parameters could not sufficiently resolve the problem. In addition, these methods can also cause delayed detection of true fatal ventricular events. Medication to slow the heart rate was administered for a week, but the patient could not tolerate it. Repositioning of the lead was a possible solution, but endothe-

### Table 1

<table>
<thead>
<tr>
<th>Detection</th>
<th>Rate (bpm)</th>
<th>NID initial</th>
<th>Rx 1</th>
<th>Rx 2</th>
<th>Rx 3</th>
<th>Rx 4</th>
<th>Rx 5</th>
<th>Rx 6</th>
</tr>
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<tr>
<td>VF</td>
<td>220–250</td>
<td>18/24</td>
<td>30 J</td>
<td>30 J</td>
<td>30 J</td>
<td>30 J</td>
<td>30 J</td>
<td>30 J</td>
</tr>
<tr>
<td>FVT via VF</td>
<td>222–240</td>
<td>18/24</td>
<td>Burst (1)</td>
<td>Burst (1)</td>
<td>30 J</td>
<td>30 J</td>
<td>30 J</td>
<td>30 J</td>
</tr>
<tr>
<td>VT</td>
<td>150–222</td>
<td>24</td>
<td>Burst (3)</td>
<td>Burst (1)</td>
<td>Burst (3)</td>
<td>Ramp (3)</td>
<td>Ramp (3)</td>
<td>30 J</td>
</tr>
</tbody>
</table>

SVT Criteria On: AF/AFL, Sinus Tach, 1:1 SVTs.

![Fig. 1.](image)

(A) An example of inappropriate shock therapy caused by T-wave oversensing. Although the R–T intervals were about 360 ms and the T–R intervals were about 250 ms, the implantable cardioverter defibrillator (ICD) incorrectly detected ventricular fibrillation with the combined count criterion, and shock therapy was the first therapy administered according to therapy protocol. Note that the inappropriate shock was delivered on a T-wave. (B) An example of pseudo nonsustained ventricular tachycardia at a sinus rate of 106 beats/min. Note that both the R–T and T–R intervals were about 250–270 ms. This episode continued longer than 2.5 min. In the tracing, TP was incorrectly inscribed instead of VP. This kind of error sometimes occurs just after anti-tachycardia therapy in GEM2 ICD models. Abbreviations used are as follows: AR, atrial sensing during refractory period; VS, ventricular sensing; VR, ventricular refractory after charge; CE, charge energy; CD, cardioversion; TP, anti-tachycardia pacing; VP, ventricular pacing.

3. Discussion

We herein report a case of inappropriate shock therapy that was caused by T-wave oversensing and that occurred when the patient had sinus tachycardia and was squatting. Several methods were considered to troubleshoot this problem. We decreased the sensitivity and increased the NID and detection rate of ventricular tachycardia in order to satisfy the combined count criterion; however, a number of nonsustained ventricular tachycardias were still recorded. Further shortening of the detection intervals of the ventricular tachycardia zone would not be sufficient because a number of nonsustained ventricular tachycardias with short R–T and T–R intervals (about 250 ms) were also recorded. Therefore, changes in these parameters could not sufficiently resolve the problem. In addition, these methods can also cause delayed detection of true fatal ventricular events. Medication to slow the heart rate was administered for a week, but the patient could not tolerate it. Repositioning of the lead was a possible solution, but endothe-
lization of the lead body may have prevented manipulation.
because 2 years had passed since ICD implantation in the present case. An additional lead for ventricular sensing or replacement of the ICD with one from another manufacturer or from the new generation of Medtronic ICDs was a desirable solution in such situations [3–8,12–16,18,19]. However, all of these methods are expensive and require additional operations, which may cause complications [20]. The patient’s cardiac status had improved so significantly that aggressive intervention for ventricular tachycardia to prevent sudden cardiac death was no longer necessary, although the possibility of sudden cardiac death could not be excluded. We decided to stop the therapy except in the ventricular fibrillation zone. Consequently, his clinical course has been good.

The most important aspect in this case was how to make a correct diagnosis. The sudden appearance of nonsustained ventricular tachycardia 1 month before inappropriate shock therapy may have been caused by T-wave oversensing. Although the diagnosis of inappropriate shock therapy was finally made by intracardiac electrograms, interrogation of the device in the supine position did not demonstrate T-wave oversensing. We then reproduced the clinical situation by isoproterenol infusion and a postural change (i.e., sinus tachycardia and squatting position) to finally demonstrate T-wave oversensing. There were 3 important factors involved in the observed phenomenon: sinus tachycardia, squatting, and a decrease in R-wave amplitude. In addition to T-wave oversensing, sinus tachycardia was required to satisfy the detection rate. A decrease in R-wave amplitude has been reported to be an important factor for T-wave oversensing [2,12–15]. Although the present patient’s cardiac function had significantly improved and his surface ECG showed no changes, the intrinsic R-wave amplitude had decreased from 8–9 mV to 5 mV. Interestingly, sinus tachycardia increased the T-wave amplitude. Adrenergic stimulation causes sinus tachycardia and may have affected depolarization and local electrograms. In addition, squatting reduced the R-wave amplitude further, resulting in T-wave oversensing. Regarding the lead system, a true bipolar lead reportedly lowers the risk of T-wave oversensing [20]. Although the lead used was true bipolar, an active fixation lead was screwed into the upper part of the interventricular septum of the right ventricle in this patient. The distal tip of an active fixation lead may be less endothelialized and more flexible than that of a passive fixation lead because the lead can be fixed only by screwing. In addition, postural change may affect the angle between the distal tip of the lead and the local myocardium and cause electrogram variation. Although the true mechanism responsible for these variations in electrograms is unknown, this phenomenon should be taken into consideration when T-wave oversensing is suspected or when intermittent reductions of R-wave amplitudes are observed [13,14]. In addition, multiple episodes of nonsustained tachycardia were recorded before the episodes of inappropriate shock therapy. Therefore, when multiple nonsustained ventricular tachycardia events without suspicious causes are newly detected or the frequency suddenly increases, the possibility of T-wave oversensing should be considered.

Conflict of interest

All authors have no conflicts of interest to declare.

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References


