

Treatment of symptomatic bradycardia due to T-wave oversensing with implantation of a new generator incorporating delayed decay and threshold start sensitization algorithms



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Introduction

T-wave oversensing (TWO) can lead to a reduction in biventricular pacing as well as bradycardia in pacemaker-dependent patients. We present a case of a 58-year-old woman with hypertrophic cardiomyopathy (HCM) and atrioventricular nodal (AVN) ablation who presented with severe bradycardia due to overcounting of T waves. Multiple reprogramming strategies were attempted without success. This is the first reported treatment of TWO by change-out to a generator utilizing decay delay and threshold start features.

Case report

A 58-year-old woman with a history of HCM and permanent atrial fibrillation (AF) presented to the office with persistent dyspnea on exertion and inability to raise her heart rate with exercise. Eight years ago, she had episodes of nonsustained ventricular tachycardia and syncope, prompting implantation of a left-sided dual-chamber implantable cardioverter-defibrillator (ICD) (Medtronic Secura DR; Medtronic, Minneapolis, MN) with a Medtronic CapSureFix 5076 lead in the right atrium and Medtronic Sprint Quattro 6947 lead in the right ventricular (RV) apex. Over the next 5 years, she had multiple admissions for symptomatic AF requiring cardioversion. AF persisted despite 2 attempts at pulmonary vein isolation and multiple antiarrhythmic drugs. She was found to have worsening left ventricular (LV) function with a drop in her ejection fraction to 20%. Three years ago, she therefore underwent AVN ablation with upgrade to a biventricular ICD (Medtronic Viva Quad

KEY TEACHING POINTS

- T-wave oversensing in implantable cardioverter-defibrillators is due to the specific requirement of such devices to detect low-amplitude and low-frequency signals.
- T-wave oversensing can lead to inhibition of bradycardia pacing and cardiac resynchronization therapy.
- Multiple reprogramming options should be considered in the event of T-wave oversensing, including adjustments to the ventricular sensitivity, postpacing blanking period, and lead sensing configuration.
- In the event of refractory T-wave oversensing, generator change should be considered, including change-out to a device utilizing decay delay and threshold start features.

CRT-D; Medtronic, Minneapolis, MN) with a Medtronic Attain Performa 4298 lead placed in a lateral vein through the coronary sinus. Following these interventions, her ventricular function normalized on subsequent echocardiograms. However, she began to experience severely limiting dyspnea on exertion, noting that her heart rate would not increase with exercise despite rate-responsive programming. She also reported 2 episodes of severe symptomatic bradycardia with heart rates in the 40s while undergoing physical therapy at a rehabilitation center.

Her 12-lead electrocardiogram in the office revealed underlying AF and biventricular pacing (Figure 1). Device interrogation revealed an underlying rhythm of AF with complete heart block. Bradycardia pacing was programmed in the VVIR mode with a lower rate of 70 beats per minute

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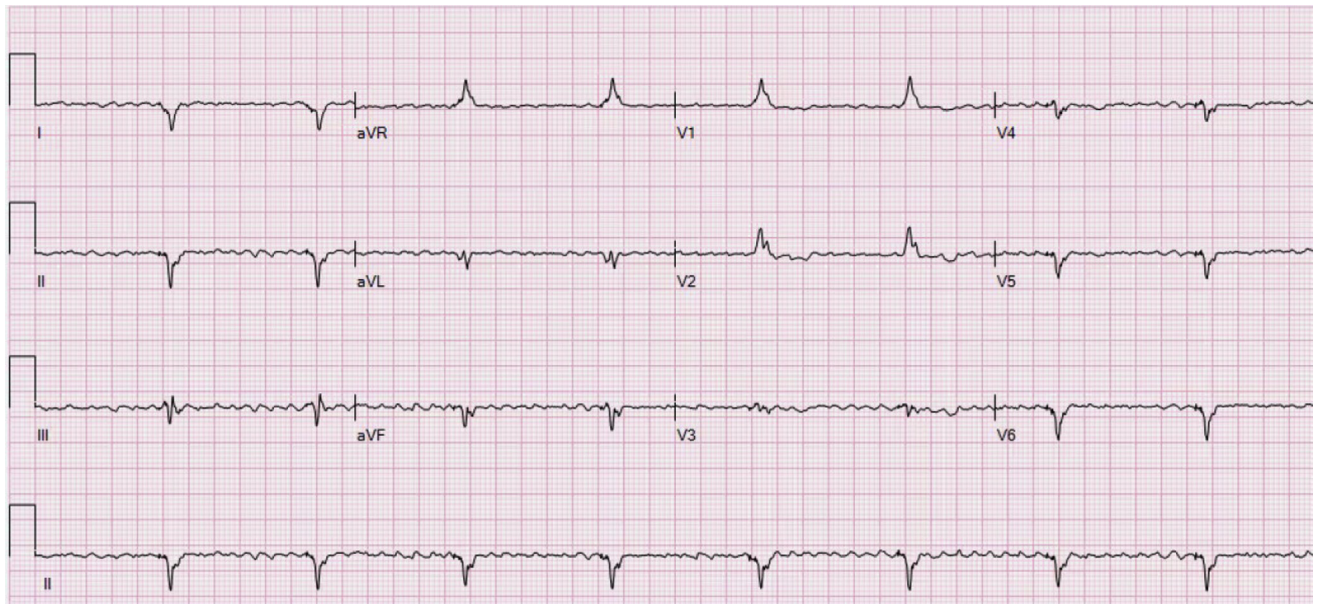


Figure 1 Twelve-lead electrocardiogram at baseline showing underlying atrial fibrillation and biventricular pacing. The device is programmed to VVIR at 70 beats per minute but is pacing at a lower rate owing to T-wave oversensing.

(bpm) and an upper rate of 120 bpm. Despite a lower programmed rate of 70 bpm, pacing at 52 bpm was noted (Figure 1). AF burden was 100% and she was ventricularly paced 95.6% of the time. Her atrial, RV, and LV lead impedances were within normal limits and no episodes

of ventricular tachyarrhythmias were detected. Device electrograms were consistent with TWO during ventricular pacing at 70 bpm (Figure 2). Electrolytes were within normal limits and a 2-view chest radiograph ruled out lead dislodgement, fracture, or malpositioning. There was an increase in



Figure 2 Medtronic electrograms showing high-amplitude postpacing T wave. T-wave oversensing persisted despite adjustments to sensitivity up to 1.2 mV, increase in postpacing blanking period to 400 ms, and changing lead sensing configuration from dedicated bipolar to integrated bipolar.

TWO during exercise, which led to “double counting” of the QRS complex with inhibition of pacing.

Several programming adjustments were attempted to reduce or eliminate TWO. The postpacing blanking period was increased to over 400 ms. This maneuver failed to reduce TWO and was limited by the pacing rate, which did not allow appropriate programming of rate response at such a high blanking period. Her postpacing blanking period was thus left at 320 ms. Ventricular sensitivity was adjusted to 0.6 mV, 0.9 mV, and 1.2 mV in both true (dedicated) bipolar and integrated bipolar configurations. ICD function was tested in the lab to ensure proper detection at these lower sensitivities. There was no change in TWO with lower sensitivity up to 1.2 mV and there was in fact more TWO noted with integrated bipolar sensing. Therefore, the device was left with a sensitivity of 0.6 mV in a true bipolar configuration. LV paced polarity was adjusted as well, given the fact that the patient described more

symptoms after upgrade to CRT-D. LV paced polarity was tested at all viable configurations, but none eliminated TWO. Therefore, paced polarity was left at LV1 to LV2 with LV first by 20 ms. Simultaneous LV and RV pacing also failed to reduce TWO.

Several options were considered and discussed with the patient. This included insertion of a new dedicated RV sensing lead (which would represent a fourth lead), lead extraction and placement of a new ICD lead on the septum, or a change to a new Abbott generator that would utilize decay delay and threshold start features to reduce TWO. The patient elected for change-out of the pulse generator to minimize interventions and potential risks, especially since her current generator was nearing the elective replacement indicator in several months. An Abbott Quadra (Abbott, Abbott Park, IL) was successfully implanted and connected to the prior right atrial, RV, and coronary sinus leads. Pacemaker maximum sensitivity was 2.0 mV with postpaced

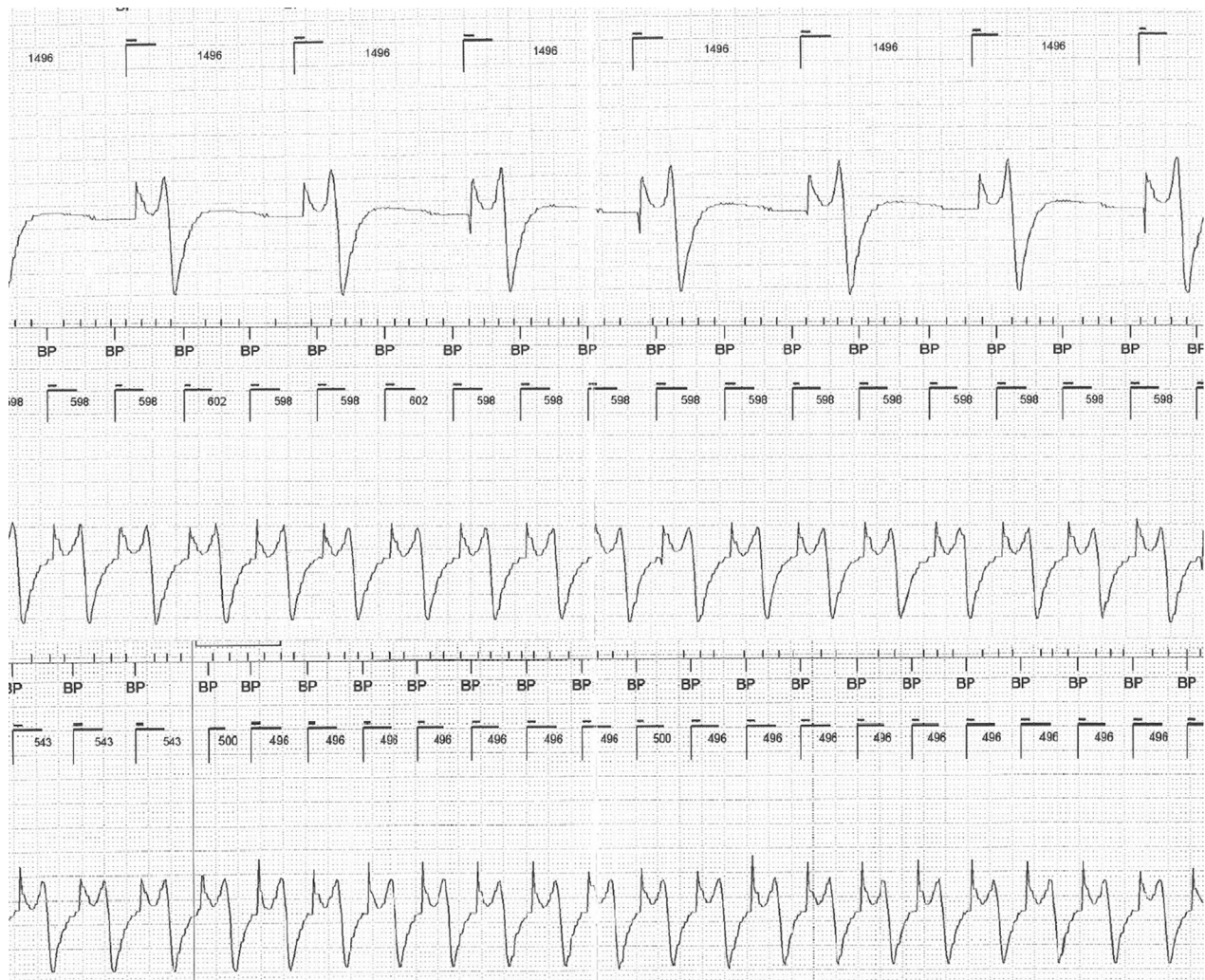


Figure 3 Postimplant Abbott (Abbott Park, IL) electrograms (auto-gained right ventricular signal). Prior to programming, there is intermittent overcounting of the sensed T wave (top pane) at VVI 30 beats per minute (bpm). Following programming of decay delay at 220 ms and threshold start at 1.0 mV of the paced beat there is elimination of T-wave oversensing at both 40 bpm and 130 bpm.

threshold start set to 1.0 mV and postpaced decay delay set to 220 ms. Postsensed decay delay was set to 60 ms with a threshold start of 62.5% (Figure 3). There was no further TWO detected immediately post implant or 6 months later.

Discussion

Proper functioning of ICDs requires both high sensitivity and short refractory periods to ensure prompt detection of the rapid, low-amplitude signals of ventricular fibrillation (VF). This requirement can lead to oversensing of non-QRS signals, particularly the T wave. When TWO is detected, the clinician should first rule out organic and mechanical causes unrelated to device programming, including lead malpositioning and electrolyte abnormalities. The clinical importance of TWO has been discussed primarily in the context of inappropriate ICD therapy and reduction in CRT-D; however, in this case the only manifestation of TWO was symptomatic bradycardia due to inhibition of pacing. In fact, our patient only manifested TWO after AVN ablation and upgrade to CRT-D. Despite the observation that TWO might be more common in paced beats than in native beats,¹ there have been relatively few reports of TWO leading to symptomatic bradycardia owing to inhibition of pacing.^{2,3}

As a result, most programming adjustments reported to fix the problem of TWO have been described in the context of inappropriate shocks and reduction of CRT. In this case we attempted many programming adjustments. The first was to decrease the ventricular sensitivity to exclude non-QRS signals.^{1,4} This carries the drawback of potential underdetection of VF. We verified adequate ICD function by induction of VF in the lab at lower programmed R-wave sensitivities. Despite this effort, reducing sensitivity from 0.6 mV to 1.2 mV did not prove to reduce TWO. This may have been owing either to increased paced repolarization amplitude (absolute size of the T wave) or to decreasing R-wave amplitude relative to the T wave, both of which may be of particular importance in the setting of HCM.^{2,5}

The next maneuver we attempted was to increase the postpacing blanking period so that the oversensed T wave would not be counted by the device. In a study of 50 patients with older devices, increasing the bradycardia pacing refractory period to 386 ± 32 ms eliminated TWO in paced beats.⁶ This maneuver failed to solve the problem in our case, as any increase in the postpacing blanking period was limited by the pacing rate and disallowed rate responsiveness. In fact, we found no examples of an increase in the postpacing refractory period solving the problem of TWO in contemporary devices.

We then attempted to adjust the lead sensing configuration from a true bipolar configuration to an integrated bipolar configuration. There are varying data on the effect of integrated bipolar vs true bipolar sensing configuration on TWO.^{7,8} Weretka and colleagues⁸ suggest that an integrated

bipolar configuration may be up to 5 times more likely to result in TWO than a true bipolar configuration, perhaps owing to the proximal position and large surface area of the RV coil, which serves as 1 of the sensing anodes in an integrated bipolar configuration. Other data from Brugada patients have shown a higher degree of TWO with true bipolar sensing, with the problem of TWO in 2 patients with Medtronic ICDs solved by switching from a true bipolar to integrated bipolar sensing configuration.⁷ However, it has been called into question how much this increased incidence in TWO is due to the sensing configuration itself vs the particular sensitization algorithm employed by a given generator.^{2,7} In this case, adjusting the sensing configuration from integrated bipolar to true bipolar resulted in a greater degree of TWO. We also attempted simultaneous pacing of the left and right ventricle, which has been reported to eliminate TWO in cases of CRT-D loss owing to its effect on repolarization and T-wave morphology,⁹ but it was of no benefit here.

Given the degree of morbidity experienced by our patient and the failure of reprogramming, we then considered invasive options. One possibility was addition of a specialized RV pacing lead. In a case reported by Kapa and colleagues,⁵ an HCM patient suffering from inappropriate shocks due to TWO was successfully treated with placement of a dedicated sensing lead in the mid-septal outflow region. Although this option was considered, the risks attendant with placing an extra pacing lead were unwarranted, given the high likelihood that the problem could be fixed with simply changing the specific ICD generator.

Changing the specific ICD generator has been shown to reduce TWO in several case reports. Depending on the manufacturer, contemporary ICDs employ various means to reduce TWO, which are largely based on (1) time-dependent adjustment of sensitivity following a sensed or paced R wave through auto-gain control or T-wave rejection algorithms and (2) bandpass filtering to reduce sensing of low-frequency T waves relative to high-frequency R waves. In prior cases of generator change fixing TWO, the T-wave amplitude seems to have decreased on device electrograms following implantation of a new generator.^{2,10,11}

This is the first case to report treatment of TWO by change-out to a generator utilizing the decay delay and threshold start T-wave rejection algorithms. Decay delay allows the sensitivity to start at a programmable percentage of a sensed paced or native R wave, remain fixed for a certain period of delay, then increase gradually to a programmable minimal sensitivity. The rate of decay is set to 1 mV / 312 ms and is nonprogrammable. Threshold start is the maximum programmable sensing threshold beginning at the end of the blanking period and is derived as a percentage of the R wave. It is important to note that not all devices are able to apply their specific T-wave rejection features to both paced and sensed beats. It has been reported that Medtronic's T-wave rejection algorithm may only apply to sensed events² and

may be a reason for the persistent TWO encountered prior to reimplant. The Abbott device used in our case also utilizes a “split” programming feature, allowing one to program separate sensitivity parameters for paced beats without compromising the detection of sensed low-amplitude signals seen in VF. It should be noted that the favorable response obtained with the new generator may have been due to a change in band-pass characteristics rather than the decay delay algorithm. Such differences in the sense amplifier may result in a change in the detection interval from the paced biventricular complex to the T wave, which may have come into play here.

Conclusion

This case illustrates a unique way to treat TWO by change-out to a generator utilizing the decay delay and threshold start T-wave rejection algorithms in a situation where multiple programming options were ineffective in eliminating symptomatic TWO. As ICDs continue to evolve, it is important that the electrophysiologist have a basic understanding of the various means of addressing TWO, including device-specific programming options available at the current time. Furthermore, the clinician should be aware of other nonprogrammable factors, such as band-pass characteristics, which may result in TWO.

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