Unique Sensing Errors in Third-Generation Implantable Cardioverter-Defibrillators

DAVID J. CALLANS, MD,* BRUCE G. HOOK, MD, ROBERT B. KLEIMAN, MD, RAMAN L. MITRA, MD, BELINDA T. FLORES, RN, MSN, FRANCIS E. MARCHLINSKI, MD, FACC

Philadelphia, Pennsylvania

Objectives. Third-generation cardioverter-defibrillators appear to be susceptible to unique sensing errors. This study was performed to determine the incidence and types of sensing errors in combination therapy implantable devices.

Background. One of the advantages offered by third-generation implantable cardioverter-defibrillators is the combination of bradycardia and antitachycardia pacing and cardioversion-defibrillation capabilities in a single device. The potential for unique sensing errors, those caused by the conflicts presented by combining bradycardia and tachycardia sensing and therapy algorithms in the same device, has not been previously addressed.

Methods. To determine the incidence of important sensing errors, 61 patients with a combination therapy device (Cadence [Ventritex] and PCD [Medtronic]) were studied for a 25-month period. In addition to surface electrocardiographic recordings during implantation and routine device testing, real-time and stored electrograms recorded from the rate-sensing leads (Cadence) and real-time marker channel recordings (PCD) were reviewed to diagnose sensing errors that resulted in symptoms, device inefficacy or delivery of inappropriate therapy. After recognition, specific reprogramming steps were performed in an attempt to avoid recurrent sensing errors.

Results. A total of 13 sensing errors were diagnosed in 12 patients (19.7%); the incidence was similar in both devices. Five distinct categories of sensing errors were identified. After device reprogramming, only one recurrent error occurred in 98 patient-months of follow-up.

Conclusions. Important sensing errors occur in approximately 20% of patients with third-generation combination therapy cardioverter-defibrillators. Prompt diagnosis of sensing errors can lead to specific reprogramming steps to avoid recurrent errors.

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Figure 1. This sequence begins with electrograms identical to those registered during real-time recordings of sinus rhythm in this patient (arrows). A 6-beat run of nonsustained ventricular tachycardia (VT) with a different electrogram morphology and large amplitude signals occurs. The next electrogram, which follows at a long interval, is identical in morphology to the sinus beats at the beginning of the sequence but has a much smaller amplitude. This beat is not sensed, as evidenced by the delivery of a pacing stimulus (s) at the programmed ventricular-inhibited (VVI) pacing interval (50 beats/min [bpm]/1200 ms) from the large amplitude electrogram. This long-short coupling sequence resulted in the initiation of ventricular tachycardia, which was subsequently terminated by the device.

Device implantation. In seven patients a nonthoracotomy lead system (PCD), which consisted of endocardial energy-delivering-pacing-sensing leads with or without a subcutaneous patch lead, was implanted. Six patients had a new generator connected to existing energy-delivering and rate-sensing lead systems. Most patients received two epicardial patch energy-delivering leads and a rate-sensing-pacing lead system consisting of an endocardial in-line bipolar lead (16 patients) or two epicardial screw-in electrodes (32 patients). Before implantation, the following specifications were documented: 1) an R wave amplitude >5 mV during spontaneous rhythm (>4 mV for chronic lead systems), 2) a pacing threshold ≤1.5 V at 0.5-ms pulse width (≤2 V in chronic lead systems), and 3) a defibrillation threshold ≤550 V (Cadence) or 18 J (PCD). Device efficacy for detection and termination of all inducible ventricular arrhythmias was assessed intraoperatively. Detailed testing was also performed 1 and 6 weeks postoperatively, with induction of ventricular arrhythmias using noninvasive programmed stimulation.

Data acquisition and analysis. In addition to surface electrocardiographic recordings during implantation and routine device testing, the following information was reviewed for diagnosis of sensing errors: for PCD, direct recordings from the rate-sensing and energy-delivering lead during device implantation and real-time marker channel recordings during device testing; for cadence, direct recordings from the rate-sensing and energy-delivering lead systems during device implantation, real-time electrograms recorded from the rate-sensing leads during device interrogations and stored electrograms recorded from the rate-sensing leads during events leading to therapy delivery for spontaneous arrhythmias. The utility of stored electrogram analysis in the diagnosis of spontaneous arrhythmias has been previously reported (6). After recognition of a sensing error that resulted in significant symptoms, device inefficacy or inappropriate delivery of therapy, a specific programming intervention was performed, if possible, to prevent recurrent errors.

Results

Five different categories of sensing errors were observed: 1) transient sensing failure during sinus rhythm caused by an abrupt change in the amplifier gain setting, 2) transient sensing failure during sinus rhythm caused by spontaneous variation in signal amplitude, 3) inappropriate inhibition of bradycardia pacing due to T wave oversensing, 4) inappropriate antitachycardia therapy caused by T wave oversensing during sinus rhythm, and 5) ineffective antitachycardia pacing due to inaccurate detection of the tachycardia cycle length.

Transient sensing failure during sinus rhythm caused by an abrupt change in the amplifier gain setting (Fig. 1). The sense amplifier adapts continuously to the characteristics of the incoming signal by adjusting either the gain setting (Cadence) or the sensing threshold (PCD). The adjustments are not instantaneous but require either a fixed number of signals of significantly different amplitude or the passage of a fixed amount of time before going into effect. If a series of large amplitude signals interrupts sinus rhythm (for example, nonsustained ventricular tachycardia), the gain will be decreased to prevent amplifier saturation. This adjustment can lead to a transient inability to sense the next sinus signal. In the absence of a sensed R wave, a pacing stimulus is delivered at the programmed escape interval after the last large amplitude beat. The resulting long-short stimulation sequence can result in device-mediated initiation of tachycardia in susceptible patients.

Two patients had sustained ventricular tachycardia initiated by inappropriate pacing stimuli delivered in association with this sensing error. Both tachycardias were promptly detected and terminated by device therapy. The bradycardia pacing rate was increased in both cases in an attempt to avoid duplicating the coupling interval sequence that resulted in arrhythmia induction.
Transient sensing failure during sinus rhythm caused by spontaneous variation in signal amplitude (Fig. 2). As described earlier, the sense amplifier adjusts to establish a relatively stable gain/threshold setting for the sinus rhythm signal. A single large amplitude signal from a ventricular or a conducted atrial premature beat will not produce a change in the amplifier gain setting but does cause a transient alteration in sinus cycle length. The sinus electrogram after a premature beat can be significantly lower in signal amplitude than the preceding sinus beats (7). This spontaneous change in signal amplitude can cause transient failure to sense the sinus signal, resulting in the delivery of inappropriate pacing stimuli, potentially causing the induction of ventricular arrhythmias.

Three patients had episodes of device-mediated tachycardia induction associated with this sensing error. The tachycardias were appropriately detected and terminated with device therapy. To prevent the coupling interval sequence that resulted in tachycardia induction, the bradycardia pacing rate was increased.

Inappropriate inhibition of bradycardia pacing due to T wave oversensing (Fig. 3). When the lead system is used for pacing, sensing function is intermittently suspended (blanking). During intermittent ventricular pacing, this is not problematic because the amplifier adjusts to the amplitude of the spontaneous R waves. In 100% paced rhythm, the only signal that is available for sensing is the T wave and local afterdepolarization. The sense amplifier adjusts to detect these low amplitude events, usually by operating at maximal gain or minimal threshold. This amplification can result in inappropriate sensing of the T wave as a separate event, which inhibits delivery of the next pacing stimulus and lengthens the effective pacing escape interval.

Inappropriate inhibition of bradycardia pacing occurred...
in four pacing-dependent patients, resulting in symptoms due to sustained heart rates of ≤40 beats/min or frequent pauses ≥2 s in duration. The postpacing refractory period was increased in these patients to decrease the likelihood of T wave oversensing.

Inappropriate antitachycardia therapy caused by T wave oversensing during sinus rhythm (Fig. 4). Appropriate sensing depends on distinguishing R waves from T waves on the basis of amplitude differences with or without the use of a time-dependent change in the sensing threshold down to a programmable “floor” value (PCD). If the amplitude of the T wave-afterdepolarization complex is not significantly less than the R wave amplitude, it will be interpreted as an R wave. The resultant “double-counting” can result in spurious tachycardia detection.

Device therapy was delivered during sinus rhythm at rates well below the cutoff for tachycardia detection because of T wave oversensing in two patients. In one patient, the sensing threshold was increased to prevent recurrence. No intervention was possible in the other patient with this problem.

Ineffective antitachycardia pacing due to inaccurate detection of the tachycardia cycle length (Fig. 5 and 6). Two different sensing errors occurred in this category. In the first, appropriate antitachycardia pacing was transiently inhibited due to T wave oversensing (Fig. 5). In the second, antitachycardia pacing protocol was VVI burst pacing, 10 stimuli at 84% of the tachycardia cycle length. The marker channel demonstrates that the T wave after the 3rd paced beat is counted as an R wave (tachycardia rate zone, sensed beat [TSI]). The next tachycardia beat is sensed (fibrillation rate zone, sensed beat [FSI]), which results in inhibition of the 4th beat of the burst. The remainder of the burst is delivered but is unsuccessful in terminating the tachycardia. VP = ventricular paced event; VR = ventricular sensed event within sensing refractory period; VS = ventricular sensed event.

Figure 4. This sequence of stored electrograms begins with sinus tachycardia (133 beats/min, 450 ms), well below the rate cutoff for detecting ventricular fibrillation (VF) (270 ms). Note the relatively large amplitude deflections between R waves, which represent local T waves. There are two increases in the amplifier gain setting, indicated by the straight arrows. The gain steps are apparently performed to better interpret the low amplitude portions of the signal to prevent missing a diagnosis of fine ventricular fibrillation. The T wave signal was eventually magnified sufficiently to cause double counting, resulting in spurious detection of ventricular fibrillation and the delivery of a 500-V shock. NSR = normal sinus rhythm.

Figure 5. A real time surface electrocardiographic (ECG) lead and marker channels from the pacing-sensing lead during an induced episode of ventricular tachycardia. The antitachycardia pacing protocol was VVI burst pacing, 10 stimuli at 84% of the tachycardia cycle length. The marker channel demonstrates that the T wave after the 3rd paced beat is counted as an R wave (tachycardia rate zone, sensed beat [TSI]). The next tachycardia beat is sensed (fibrillation rate zone, sensed beat [FSI]), which results in inhibition of the 4th beat of the burst. The remainder of the burst is delivered but is unsuccessful in terminating the tachycardia. VP = ventricular paced event; VR = ventricular sensed event within sensing refractory period; VS = ventricular sensed event.
Cardiac pacing was delivered at a cycle length greater than the tachycardia cycle length. If 5 beats are averaged together to determine the tachycardia cycle length, one interval is falsely sensed as 820 ms because of signal dropout, then antitachycardia pacing will proceed at 430 ms. T = time line.

Figure 6. Surface electrocardiographic leads I, II, and V1 during an episode of induced ventricular tachycardia (VT) with a stable cycle length of 410 ms. After tachycardia detection, antitachycardia pacing (programmed for a burst of 12 stimuli at 85% of the tachycardia cycle length) ensues at a cycle length of 430 ms (i.e., longer than the tachycardia cycle length). If 3 beats are averaged together to determine the tachycardia cycle length, and one interval is falsely sensed as 410 ms because of signal dropout, then antitachycardia pacing will proceed at 430 ms.

Sensing errors caused by the special complexities of third-generation devices and no specific programming changes were performed.

Follow-up after device reprogramming. A total of 13 sensing errors resulting in significant symptoms or inappropriate device therapy were seen in 12 (19.7%) of the 61 patients. The incidence was similar in both devices: 10 of 48 patients with the Cadence and 2 of 13 patients with the PCD experienced problems because of sensing errors. In 10 of the 13 episodes, which occurred over a period of 73 patient-months after implantation, a specific change in device programming was performed in an attempt to prevent recurrence. Only one recurrent error occurred after reprogramming over a period of 98 patient-months. The single recurrence was an episode of ventricular tachycardia caused by an inappropriate paced beat after a nonsensed sinus complex, which was again treated by an adjustment in the bradycardia pacing interval.

Discussion

Major findings. In our study, unique sensing errors resulting in symptoms from bradycardia or tachyarrhythmias, device inefficacy or delivery of inappropriate therapy occurred in nearly 20% of patients with combination therapy devices. The recognition of these sensing errors was facilitated by the enhanced diagnostic capabilities available in the Cadence and PCD. Finally, in most cases, recurrent errors were avoided by specific programming interventions.

Unique requirements of combination therapy devices. Sensing errors caused by the inability of present devices to distinguish ventricular from supraventricular tachyarrhythmias (4,5,6,8-14) and those imposed by the limitations of available lead technology (13,16) have been well documented. This is the first comprehensive study of sensing errors caused by the special complexities of third-generation combination devices. The requirements for cardioverter-defibrillator sensing systems are more demanding than those developed for bradycardia pacemakers. Accurate detection of ventricular fibrillation requires rapid adaptation to a >10-fold change in signal amplitude, a situation that essentially invalidates the incorporation of a fixed, programmable sensing threshold. In addition, tachycardia-sensing algorithms cannot include post-sensing refractory periods, which bradycardia pacemakers utilize to avoid T wave oversensing. Although present tachycardia-sensing algorithms are able to meet these specifications, the addition of bradycardia pacing presents seemingly unavoidable contradictions. For example, in the absence of sensed complexes, two potentially life-threatening diagnoses with different treatment strategies must be considered: heart block requiring bradycardia pacing and ventricular fibrillation requiring amplifier gain adjustments for proper detection.

Several of these sensing errors are also observed in bradycardia pacing systems. Combination therapy devices are considerably more complicated because device reprogramming steps, even if effective in preventing recurrent errors, often compromise either bradycardia- or tachycardia-sensing functions. For example, T wave oversensing can be easily managed in bradycardia pacing systems by readjusting the sensing threshold. Raising the sensing threshold (PCD) to prevent T wave oversensing during sinus rhythm imposes potential limitations on the speed and accuracy of detecting ventricular fibrillation. Lengthening the post-sensing refractory period to eliminate T wave oversensing during paced rhythms (Cadence) reduces the amount of time available for sensing and could conceivably prevent or delay detection of tachycardia. Obviously, device testing is essential after significant changes in programming to ensure adequate tachycardia sensing function.

Conclusions. We found important sensing errors, resulting in symptoms, device inefficacy or delivery of inappropriate therapy in nearly 20% of patients with third-generation combination therapy devices. This represents a considerable improvement over the results of physically separate bradycardia pacemakers and cardioverter-defibrillators (1-3,5) but demonstrates the difficulty in combining bradycardia- and tachycardia-sensing algorithms in a single system. Recognition of sensing errors, facilitated by the enhanced diagnostic capabilities of third-generation devices, allows reprogramming steps to avoid recurrent sensing errors.
References


