Underdetection of Ventricular Tachycardia by Algorithms to Enhance Specificity in a Tiered-Therapy Cardioverter-Defibrillator

CHARLES D. SWERDLOW, MD, FACC,*† THOMAS AHERN, MD, FACC,‡ PENG-SHENG CHEN, MD, FACC,* CHUN HWANG, MD,* ELI GANG, MD, FACC,* WILLIAM MANDEL, MD, FACC,* ROBERT M. KASS, MD,* C. THOMAS PETER, MD, FACC* Los Angeles, California and Las Vegas, Nevada

Objectives. The goal of this study was to determine the incidence and clinical significance of underdetection in 125 patients treated with a tiered-therapy cardioverter-defibrillator, the Medtronic PCD.

Background. Underdetection, distinct from undersensing, is a unique, potential complication of new algorithms that enhance specificity in tiered-therapy cardioverter-defibrillators. These algorithms may delay or prevent recognition of ventricular tachycardia even though electrograms are sensed accurately and RR intervals meet the programmed interval criterion.

Methods. Underdetection was defined as delay in detection >5 s at electrophysiologic study or symptomatic delay or detection failure at follow-up of 15 ± 8 months.

Results. We identified six specific mechanisms of underdetection caused by algorithms to discriminate sustained ventricular tachycardia from sinus tachycardia, atrial fibrillation, ventricular fibrillation and nonsustained ventricular tachycardia. Underdetection caused detection delays in 13 (1.9%) of 677 induced ventricular tachyarrhythmias in 12 patients (9.6%). During follow-up, underdetection occurred in 7 (9.9%) of 71 patients in whom ventricular tachycardia therapies were programmed. Failure to detect ventricular tachycardia occurred in 6 (0.6%) of 988 spontaneous ventricular tachycardia episodes in four patients (5.6%); 2 episodes required external cardioversion. After defibrillator reprogramming, underdetection did not occur.

Conclusions. Algorithms to enhance specificity cause underdetection of ventricular tachycardia in a significant minority of patients with tiered-therapy cardioverter-defibrillators. Optimal programming can minimize underdetection.

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Inappropriate detection of ventricular tachycardia is an important problem for tiered-therapy cardioverter-defibrillators because it may cause painful and dangerous inappropriate therapies or aborted shocks, with consequent battery depletion (1-13). The designs of tiered-therapy defibrillators include algorithms to enhance specificity for detection of ventricular tachycardia and thereby minimize inappropriate detection. Underdetection, distinct from undersensing, is a unique, potential complication of these algorithms. They may delay or prevent recognition of ventricular tachycardia even though electrograms are sensed accurately and the tachycardia cycle lengths meet the programmed interval criterion. We evaluated prospectively the incidence and clinical significance of underdetection in consecutive patients who underwent implantation of a tiered-therapy cardioverter-defibrillator (Medtronic model 7217B PCD).

Methods

Table 1 summarizes the clinical characteristics of the 125 study patients. Patients gave written, informed consent according to a protocol approved by the Human Subjects Committee of each participating institution. The PCD is a multiprogrammable, tiered-therapy cardioverter-defibrillator that has been described in detail (11,14,15). It distinguishes tachyarrhythmias in two zones, referred to as ventricular tachycardia and ventricular fibrillation, on the basis of the tachycardia cycle length. The PCD stores limited diagnostic data, including the 20 intervals (detection sequence) before the last detected arrhythmia.
Clinical arrhythmia criterion is designed to avoid detection of a two-zone device in 71 patients. In these patients, the long-term ventricular fibrillation suspension of ventricular tachycardia was selected because it represents detection or detection failure during follow-up. The value of 330 ms (range 320 to 370) was used to detect was 12 or 18.

During long-term follow-up, we programmed the PCD as a one-zone device in 71 patients. In these patients, the ventricular tachycardia-detection interval was programmed 50 ms greater than the longest cycle length of spontaneous or induced ventricular tachycardia if the cycle length was ≤450 ms and 20 to 40 ms greater if it was >450 ms. The median value was 440 ms. Programmed stability was 40 ms, and the programmed onset ratio was 87% (16). The onset criterion was designed to avoid detection of sinus acceleration. It detects initiation of ventricular tachycardia only if the ratio of an interval to the mean of four previous intervals is less than a programmed onset ratio and either the second or third preceding interval exceeds the ventricular tachycardia-detection interval. In these 71 patients, the median ventricular fibrillation-detection interval was 320 ms, range 280 to 360 ms. The number of intervals to detect was 12 or 16 for ventricular tachycardia and 12 or 18 for ventricular fibrillation.

In the remaining 54 patients, the PCD was programmed as a one-zone device, with therapy only for device-detected ventricular fibrillation. The long-term ventricular fibrillation-detection interval was programmed 50 ms longer than the anticipated ventricular tachycardia cycle length. The median value was 330 ms (range 320 to 370). The number of intervals to detect was 12 or 18.

Follow-up. The 125 patients were followed up for a total of 1,888 patient-months from the date of electrophysiologic study until the date of study closure, death (eight patients) or cardiac transplantation (two patients). Mean duration of follow-up was 15.1 ± 8.1 months. The 71 patients in whom ventricular tachycardia therapies were programmed were followed up for 1,136 patient-months.

Data analysis. Significant underdetection was defined prospectively as delay in detection >5 s at intra- or postoperative electrophysiologic study or symptomatic delay in detection or detection failure during follow-up. The value of 5 s was selected because it represents an approximate doubling of the detection time when 12 to 16 intervals are required to detect ventricular tachycardia. This definition was applied prospectively to intraoperative and postoperative electrophysiologic studies and stored detection intervals and electrocardiographic (ECG) recordings from spontaneous episodes of device-detected arrhythmias. We used previously defined criteria for the appropriateness of detected ventricular tachycardia (16).

We reviewed the 20 stored detection intervals from episodes of device-detected ventricular tachycardia to estimate the frequency of asymptomatic delays in detection. For episodes in which the number of intervals to detect was 12, the 8 intervals preceding the 12 detection intervals were analyzed. We defined the onset of ventricular tachycardia to be present if the ratio of any four intervals to the four preceding intervals was <90% (16).

Results

We identified six causes of underdetection. Two of these, failure to satisfy the onset and stability criteria, have been reported as part of a comprehensive analysis of these criteria for the first 100 patients (16). The four previously undescribed causes of underdetection were isolated long intervals during ventricular tachycardia, competition between ventricular tachycardia and ventricular fibrillation detection zones, failure to reconfirm ventricular tachycardia and post-ventricular fibrillation suspension of ventricular tachycardia detection to avoid detection of nonsustained ventricular tachycardia.

Table 2 shows the incidence of each cause of underdetection. Overall, underdetection caused detection delays in 13 (1.9%) of 677 induced ventricular tachycardia and ventricular fibrillation episodes in 12 patients (9.6%). During follow-up, underdetection of ventricular tachycardia occurred in 9 (0.9%) of 988 ventricular tachycardia episodes in 7 (9.9%) of 71 patients in whom ventricular tachycardia.

Table 1. Clinical Characteristics of 125 Study Patients

| Age (yr) | 61 ± 11 |
| Gender | | |
| Male | 102 (82%) |
| Female | 23 (18%) |
| Coronary artery disease | 104 (83%) |
| Myocardial or valvular disease | 23 (18%) |
| Primary electrical disease | 1 (1%) |
| Clinical arrhythmia | | |
| Sustained monomorphic ventricular tachycardia | 82 (66%) |
| Ventricular fibrillation | 31 (25%) |
| Both | | |
| Left ventricular ejection fraction* | 0.32 ± 0.11 |
| PCD implant technique | | |
| Epicardial | 45 (36%) |
| Transvenous | 80 (64%) |

*Measured by contrast angiography (82 patients) or radionuclide ventriculography (38 patients). Data presented are mean value ± SD or number (% of patients).

Table 2. Mechanisms of Underdetection of Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>EPS</th>
<th>Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Onset not satisfied</td>
<td>NA</td>
<td>4 (0.1)</td>
</tr>
<tr>
<td>2. Stability not satisfied</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>3. Isolated long interval</td>
<td>4</td>
<td>0.2</td>
</tr>
<tr>
<td>4. VT-VF competition</td>
<td>5</td>
<td>0.9</td>
</tr>
<tr>
<td>5. Failure to reconfirm VT</td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>6. Post-VF suspension of VT detection</td>
<td>4</td>
<td>1.4</td>
</tr>
</tbody>
</table>

*Denominators vary. For mechanisms 1, 2, 3 and 5, the denominator is 401 induced ventricular tachycardia (VT) episodes. For mechanism 4, it is 137 induced ventricular tachycardia episodes in which automatic cardioversion therapy was delivered. For mechanism 5, it is 276 induced ventricular fibrillation (VF) episodes. Mechanisms 2 and 3 both occurred in three episodes, and mechanisms 2 and 5 occurred in one additional episode. The total number of episodes of underdetection during follow-up was nine. CE = cannot evaluate; EPS = electrophysiologic study; NA = not applicable.
therapies were programmed. Failure to detect ventricular tachycardia occurred in six (0.6%) episodes in four patients (5.6%), and delays in detection >5 s were documented in three (0.3%) additional episodes in three patients (4.2%). Underdetection of ventricular fibrillation was not recorded.

Onset. The four episodes of failed detection caused by gradual acceleration of hemodynamically stable ventricular tachycardia have been reported elsewhere (15) but are included here for completeness. In each case, the ventricular tachycardia-detection interval had been programmed only 30 ms greater than the longest cycle length of spontaneous or induced ventricular tachycardia, and it was reprogrammed to a higher value.

Isolated long intervals during ventricular tachycardia. The PCD algorithm for detection of ventricular tachycardia requires that consecutive intervals be less than the ventricular tachycardia detection interval. A single long interval resets the ventricular tachycardia-interval count to zero. This provides a degree of protection against inappropriate detection of atrial fibrillation independent of the stability criterion and is highly effective when combined with that algorithm (16-18). Isolated long intervals may cause underdetection if undersensing occurs during ventricular tachycardia, or ventricular tachycardia intervals are unstable. Abrupt doubling of stored detection intervals, indicating undersensing, was recorded during one unmonitored spontaneous ventricular tachycardia, resulting in a delay of at least 6 s (Fig. 1). At subsequent electrophysiologic testing, the telemetered R wave was 7 mV in sinus rhythm and 8 mV in ventricular tachycardia. Underdetection could not be reproduced, and reprogramming was not performed. Isolated long intervals during irregular ventricular tachycardia caused a 6-s asymptomatic delay in detection of one induced ventricular tachycardia and contributed to underdetection of two spontaneous ventricular tachycardias (see Interval stability).

Interval stability. Five ECG documented episodes of underdetection due to the stability criterion occurred in four patients. These included a 10-s delay in detection of hypotensive ventricular tachycardia at electrophysiologic testing; 7- and 10-min delays in detection of asymptomatic, spontaneous, normotensive ventricular tachycardia; and failure to detect two episodes of hypotensive, spontaneous, polymorphic ventricular tachycardia. During three of the spontaneous episodes, mean cycle length was near the ventricular tachycardia-detection interval, and some of the detection delay was caused by isolated or frequent long intervals that reset the ventricular tachycardia interval count independent of the stability criterion. In the remaining spontaneous episode, competition between ventricular tachycardia and ventricular fibrillation zones contributed to underdetection (see later).

Each patient had an antiarrhythmic drug effect at the time of interval-unstable ventricular tachycardia: amiodarone (200 mg daily, n = 2), procainamide (level 7.0 μg/ml) and propafenone (450 mg daily). In each patient, the PCD detected multiple episodes of interval-stable, spontaneous and induced ventricular tachycardia in the control state without delay. The delay at electrophysiologic testing occurred in a patient in whom amiodarone had been discontinued for 2 days. At repeat testing after amiodarone had been discontinued for 2 months, six induced interval-stable ventricular tachycardias were detected, with a maximal delay of 2.8 s. Review of this patient's induced tachycardia before amiodarone therapy demonstrated a cycle length stability of 20 ms. The 10-min delay in detection occurred perioperatively in a patient receiving amiodarone therapy. The 7-min delay occurred 1 h after an intravenous loading dose of procainamide administered because of frequent, spontaneous, interval-stable ventricular tachycardia that had been detected by the PCD. In these two patients, the telemetered R waves were 17 and 14 mV, respectively, in sinus rhythm immediately after the underdetected arrhythmias. During an electrophysiologic study performed <24 h later, the telemetered marker channel showed that both patients had sensing of all QRS complexes during induced ventricular tachycardia. The final patient was admitted to hospital because of

**Figure 1.** Underdetection caused by an isolated long interval. The 20 consecutive intervals preceding detection of spontaneous, unmonitored ventricular tachycardia are shown. They were stored by the PCD and retrieved through telemetry. Intervals are numbered in reverse time order from detection so that interval 0 corresponds to detection, and interval 19 was recorded first. The ventricular tachycardia-detection interval was 450 ms. Asterisk indicates abrupt doubling of intervals due to undersensing. Undersensing did not occur during multiple other episodes of spontaneous and induced ventricular tachycardia in this patient.
frequent, interval-stable ventricular tachycardia that was
detected by the PCD. After initiation of propafenone ther-
apy, two episodes of polymorphic ventricular tachycardia
were not detected. Both required cardioversion, and one
required cardiopulmonary resuscitation. The telemetered R
wave was 6 mV in sinus rhythm. Electrophysiologic testing
during propafenone therapy demonstrated reliable sensing of
ventricular tachycardia and ventricular fibrillation.

Of the 71 patients in whom ventricular tachycardia ther-
apiies were programmed during follow-up, unstable intervals
caused underdetection of ventricular tachycardia in 4 (24%)
of 17 patients treated with type I or type III antiarrhythmic
drugs versus 0 (0%) of 54 patients who did not receive
antiarrhythmic drugs (p < 0.001). In these four patients,
PCDs were reprogrammed with stability off. In the patients
who had long intervals, the ventricular tachycardia-
detection interval was increased, and the number of intervals
to detect ventricular tachycardia was decreased.

Competition between ventricular tachycardia and ven-
tricular fibrillation zones. Tiered-therapy cardioverter-
defibrillators classify tachyarrhythmias into different zones
on the basis of cycle length to deliver therapy appropriate for
the specific arrhythmia. If the cycle length of a tachycardia
varies around the boundary between these two zones, de-
tection may be delayed. The maximal possible delay
depends on the specific method used for incrementing or
decrementing the interval counters for each zone. The
PCD’s ventricular tachycardia-interval counter is neither
incremented nor decremented by ventricular fibrillation
intervals. In contrast, detection of ventricular fibrillation
requires only that 75% of the intervals in a rolling detec-
tion window be less than the ventricular fibrillation-detection
interval. Delay in detection due to competition between
ventricular tachycardia and ventricular fibrillation detection
zones may occur if a tachycardia accelerates across the
ventricular fibrillation-detection interval boundary, deceler-
ates across the boundary or oscillates around the boundary.
This type of underdetection was observed in six induced
ventricular tachycardias, two due to acceleration, one due
to deceleration and three due to oscillation (Fig. 2). All except
the tachycardia that accelerated were detected as ventricular
tachycardia. The maximal delay was 6.5 s. Zone competition

Figure 2. Competition between ventricular tachycardia (VT) and
ventricular fibrillation (VF) detection zones at electrophysiologic
testing. Eight surface electrocardiographic leads, the electrogram
from a temporary pacing catheter in the right ventricular apex
(RVA) and the telemetered PCD marker channel are shown. The
ventricular tachycardia interval counter is reset to zero by intervals
greater than the programmed PCD marker interval (400 ms) and suspended, but not reset, by intervals less than
the ventricular fibrillation-detection interval (280 ms). Ventricular
tachycardia is detected when the ventricular tachycardia-interval
counter equals 12, the programmed number of intervals to detect
(FID). In contrast, the ventricular fibrillation-interval count is the
number of ventricular fibrillation intervals in the last 16 intervals.
Detection of ventricular fibrillation requires that 12 of these 16
intervals (75%) be less than the ventricular fibrillation-detection
interval. The last cycle of a 400-ras drivetrain followed by three
extrasystoles is shown on the left. Long single marks denote ventric-
ular pacing (VP). The first six induced intervals are ventricular
tachycardia intervals (TS) (intermediate-length double marks), and
the seventh is a ventricular fibrillation interval (FS) (short double
mark). The eighth interval (asterisk) is sensed appropriately (VS)
(intermediate-length single mark), but resets the ventricular
tachycardia-interval counter because it differs from the preceding
interval by more than the programmed stability value of 40 ms. After
this interval, 28 sequential ventricular tachycardia and ventricular
fibrillation intervals are required to detect an arrhythmia (ventricular
tachycardia) because of zone competition. Antitachycardia pacing is
followed by 6 beats of nonsustained ventricular tachycardia and
termination of the arrhythmia.
Figure 3. Underdetection caused by interaction between unstable intervals and competition between ventricular tachycardia and ventricular fibrillation detection zones. A, Induced ventricular tachycardia. A surface electrocardiographic lead and the PCD marker channel are shown. The tachycardia cycle length varies around the ventricular fibrillation detection interval of 320 ms. The arrow marks reset of the ventricular tachycardia-interval counter by an unstable interval (VS) (two intermediate-length single marks). As in Figure 2, intermediate-length double marks (TS) denote ventricular tachycardia intervals, and short double marks (FS) denote ventricular fibrillation intervals. The total delay in detection is 8.5 s. The 0.6 J cardioversion pulse (CD) (long triple down-mark followed by long triple up-mark) accelerated the tachycardia, which was then detected rapidly as ventricular fibrillation and defibrillated successfully with 34 J. FDI = ventricular fibrillation-detection interval; NID = number of intervals to detect; TDI = ventricular tachycardia detection interval. B, Spontaneous polymorphic ventricular tachycardia during treatment with propafenone. The ventricular fibrillation detection interval is 320 ms; the number of intervals to detect is 16 for ventricular tachycardia and 18 of 24 for ventricular fibrillation (VENT FIB). This arrhythmia was not detected, so stored intervals were not available for analysis. The tachycardia cycle length is difficult to measure precisely because of the polymorphic complexes, but is near the ventricular fibrillation detection interval. The 360-ms interval at the right side resets the ventricular tachycardia-interval counter because it differs from the second preceding interval (300 ms) by more than the programmed stability value of 40 ms.

was corrected by changing the ventricular fibrillation-detection interval or decreasing the number of intervals to detect ventricular tachycardia and ventricular fibrillation.

Figure 3A illustrates an interaction between zone competition and the stability criterion. Zone competition delayed detection until an unstable interval resets the ventricular tachycardia-interval count to zero. An interaction of this type prevented detection of one of the previously discussed spontaneous ventricular tachycardias (Fig. 3B).

Failure to reconfirm ventricular tachycardia. The capability to abort shocks if ventricular tachycardia terminates during capacitor charging (noncommitted shocks) requires reconfirmation of the persistence of ventricular tachycardia either during or immediately after charging. The PCD reconfirms ventricular tachycardia at the end of the capacitor charge period during a brief time window that is also used to synchronize the cardioversion shock (synchronization period). Failure to confirm ventricular tachycardia results in an aborted shock and relaunch of the ventricular tachycardia detection sequence. During electrophysiologic testing, five shocks were aborted appropriately in 137 automatic cardioversion therapies, and inappropriate failure to reconfirm ventricular tachycardia occurred once. This was caused by a...
complex interaction between the automatically adjusting sensing threshold and the reconfirmation algorithm. To avoid undersensing low amplitude electrograms during ventricular fibrillation, the sensing threshold increases abruptly after each sensed event and then exponentially decays to the programmed setting. During ventricular tachycardia, the sensing threshold is high because of high amplitude ventricular electrograms. Because detection is suspended during capacitor charging, the sensing threshold is at a minimum (corresponding to maximal sensitivity) at the end of the charge cycle. This change in sensing threshold permitted R wave double sensing, which resulted in an aborted shock (Fig. 4).

Postventricular fibrillation suspension of ventricular tachycardia detection. Shocks that terminate ventricular fibrillation may initiate ventricular tachycardia. Antitachycardia pacing delivered into postshock ventricular tachycardia that would otherwise have terminated spontaneously may initiate sustained ventricular tachycardia. To avoid detection of postshock nonsustained ventricular tachycardia, the PCD's algorithm for detection of ventricular tachycardia is disabled for 64 ventricular events (bradycardia pace, sense and ventricular fibrillation therapy) after detection of ventricular fibrillation. During this period, intervals are classified as ventricular tachycardia intervals if they fall into the ventricular tachycardia zone, but the ventricular tachycardia interval counter is not incremented. During 276 episodes of induced ventricular fibrillation, postshock suspension of ventricular tachycardia detection caused underdetection of four episodes of sustained ventricular tachycardia that required termination. In two episodes, ventricular tachycardia accelerated into the programmed ventricular fibrillation zone after 6.2 and 7.5 s, respectively, resulting in detection as ventricular fibrillation. One episode was cardioverted manually because of hypotension. The final episode required 20 s for detection (Fig. 5). Postventricular fibrillation suspension of ventricular tachycardia detection also caused clinically appropriate inhibition of ventricular tachycardia therapy in 13 episodes of nonsustained postshock monomorphic ventricular tachycardia with cycle length shorter than the programmed ventricular tachycardia-detection interval that lasted >5 s. Only two episodes lasted ≥20 intervals, with the longest lasting 28 intervals. Because postventricular fibrillation suspension of ventricular tachycardia detection is not

Figure 4. Interaction between oversensing and reconfirmation of ventricular tachycardia. A surface electrocardiographic lead and the telemetered PCD marker channel are shown. The paper speed is 12.5 mm/s. Cardioversion of ventricular tachycardia is noncommitted. At the completion of the charging period, cardioversion is delivered if the interval from the first nonrefractory sensed event to the next sensed event falls within the synchronization period >200 ms and less than the ventricular tachycardia detection interval plus 60 ms. Otherwise, cardioversion is aborted, and the ventricular tachycardia interval counter is reset to zero. The ventricular tachycardia detection zone was 410 to 270 ms, the number of intervals to detect ventricular tachycardia 16 and the programmed sensitivity 0.3 mV. After an ineffective 1-j shock for ventricular tachycardia with cycle length 300 ms, redetection of ventricular tachycardia occurs (TS) (intermediate-length double markers), followed by normal suspension of telemetry during a 4.5-s capacitor charge to 34 J. After charging ends (CE) (double long marker), the first nonrefractory sensed event is designated VS (intermediate-length single marker). The interval beginning with this event ends with a short single marker denoting oversensing in the refractory period. This results in failure to reconfirm ventricular tachycardia and reset the ventricular tachycardia interval counter. Redetection of ventricular tachycardia was required. The oversensing at the end of the charging period was caused by changes in the automatically adjusting sensitivity. Because the sensing threshold was at a minimum at the end of the charging period, the next QRS complex (VS) was sensed slightly earlier in the electrogram. The postoversensing blanking period (120 ms) then began and ended slightly earlier. This permitted double-sensing of the QRS complex for that one cycle (arrow) in the refractory period (VR) (121 to 200 ms), which aborted the shock. The doublesensed QRS complex in turn raised the sensing threshold to a high value. Note that the QRS complex after the double-sensed QRS complex was undersensed (asterisk), probably because the sensing threshold had not decayed sufficiently to permit sensing. The second charge time (from the last TS to CE markers) was short (0.6 s) because the capacitor was precharged. The cardioversion pulse (CD) (34 J) ended ventricular tachycardia. Because of oversensing in the critical reconfirmation interval, a total of 50 ventricular tachycardia intervals (15.6 s) were required for detection of ventricular tachycardia, rather than 16 intervals (5.0 s).

programmable, it can be avoided only by programming the ventricular fibrillation-detection interval longer than the cycle length of ventricular tachycardia.

Accuracy in spontaneous ventricular tachycardia. Onset of ventricular tachycardia was identified between the twelfth and sixteenth intervals preceding detection in 77% of the 92 episodes with number of intervals to detect of 12. Because of the PCD’s limited memory, the reason for additional delay in 23% of episodes could not be determined. No patient had unexplained syncope or undiagnosed self-terminating sustained palpitations lasting >1 min. Underdetection did not contribute to any of the eight deaths during follow-up.

Discussion

Previous studies have reported undersensing by cardioverter-defibrillators caused by fixed gain (19), specific limitations of electrodes (20) and automatically adjusting gain or sensitivity (21), but underdetection has not been studied. Underdetection of ventricular tachycardia is a consequence of imperfect algorithms designed to discriminate sustained ventricular tachycardia from sinus tachycardia, atrial fibrillation, nonsustained ventricular tachycardia, and ventricular fibrillation in tiered-therapy cardioverter-defibrillators. We have previously analyzed the value and limitations of onset and stability algorithms (16). The present study addresses all causes of underdetection by obligatory or optional algorithms to enhance specificity. Our principal finding is that clinically significant underdetection of ventricular tachycardia is infrequent, but serious consequences can occur rarely. Reprogramming can prevent most underdetection. Underdetection of ventricular fibrillation did not occur because the PCD’s algorithms to enhance specificity do not apply to detection of ventricular fibrillation. 

Unstable intervals. Cycle length instability may cause underdetection by one or both of two mechanisms designed to prevent detection of atrial fibrillation, isolated intervals longer than the ventricular tachycardia-detection interval and failure to satisfy the stability criterion. Underdetection due to interval instability occurred only during antiarrhythmic drug treatment in patients whose ventricular tachycardia had stable intervals in the control state. This is a previously undescribed interaction between antiarrhythmic drugs and implantable cardioverter-defibrillators. Antiarrhythmic drugs have been reported to increase cycle length variability at the termination of ventricular tachycardia (22,23), but their effect on increasing cycle length variability during ongoing, sustained ventricular tachycardia has not been reported. One explanation may be oscillation of the ventricular refractory period caused by the sudden decrease in cycle length at the onset of ventricular tachycardia to a value close to the sinus rhythm refractory period (24). Drug-induced prolongation of refractoriness may cause the tachycardia cycle length to encroach on the refractory period, and drug-enhanced slowing of conduction in the relative refractory period may permit conduction of unstable intervals without block of short intervals that would otherwise cause termination of tachycardia.

Our findings support reevaluation of interval stability or programming stability off whenever antiarrhythmic drug therapy is initiated. Other solutions to the problem of interval instability include increasing the ventricular tachycardia-detection interval, decreasing the number of intervals required to detect ventricular tachycardia, programming the PCD as a single-zone device using the less restrictive ventricular fibrillation detection criteria or using a different tiered-therapy defibrillator with a less restrictive algorithm for detection of ventricular tachycardia. However, less restrictive algorithms do not protect against detection of atrial fibrillation (18) and can result in the delivery of inappropriately slow antitachycardia pacing if undersensing occurs (21).

Rejection of nonsustained ventricular tachycardia. Two mechanisms of underdetection relate to algorithms designed to withhold therapy from nonsustained ventricular tachycardia, failure to reconfirm ventricular tachycardia and postventricular fibrillation suspension of ventricular tachycardia detection. Although the mechanism of failure to reconfirm ventricular tachycardia identified in this study is specific to the PCD confirmation algorithm, the problem is a general one for defibrillators that deliver noncommitted shocks, and each reconfirmation algorithm has specific limitations (25). Sensing errors caused by automatically adjusting sensitivity or gain are unique to advanced cardioverter-defibrillators (21). In this study, failed reconfirmation was due to an interaction between such a sensing error and the reconfirmation algorithm.

Postventricular fibrillation suspension of ventricular tachycardia detection is designed to withhold therapy from postshock nonsustained ventricular tachycardia. Our findings suggest that the duration for which postshock detection is suspended in the PCD may be longer than optimal.

Dissimination between ventricular tachycardia and ventricular fibrillation. Tiered-therapy defibrillators discriminate between tachyarrhythmias in different rate zones to ensure that delivered therapy will be optimal for the specific arrhythmia. Although most documented delays due to competition between ventricular tachycardia and ventricular fibrillation detection were short, these delays may be symptomatic because they occur during hemodynamically unstable ventricular tachycardia. Algorithms that analyze a combination of the counters in each tachycardia zone should minimize these delays.

Interactions between zone competition and unstable intervals were rare but prevented detection of one episode of ventricular tachycardia. This emphasizes the potential for complex interactions between different mechanisms of underdetection. We have recommended previously that stability not be programmed in patients with hypotensive ventricular tachycardia (16).

Underdetection of ventricular tachycardia after ventricular fibrillation therapy is also a consequence of the capability
Figure 5. Postventricular fibrillation suspension of ventricular tachycardia detection. Detection of ventricular tachycardia is disabled for 64 ventricular events (bradycardia pace, sense and therapy) after detection of ventricular fibrillation (not programmable). Detection of ventricular tachycardia requires 16 intervals $<400 \text{ ms}$, and detection of ventricular fibrillation requires 12 intervals $<260 \text{ ms}$. A surface electrocardiographic lead and the telemetered PCD marker channel are shown. AC denotes application of alternating current to induce ventricular fibrillation. Intermediate-length single marks (VS) immediately after AC denote intervals that are sensed but not detected as ventricular tachycardia or ventricular fibrillation because detection is inactive. The DU (data uplink) marker shows activation of detection. Rapid detection of ventricular fibrillation is indicated by short double marks (FS). Telemetry is suspended during the capacitor charge cycle that results in delivery of programmed ventricular fibrillation therapy, a 16-J shock. The postshock rhythm is monomorphic ventricular tachycardia that stabilizes with cycle length 280 ms. Intervals are identified as ventricular tachycardia intervals (TS) (intermediate-length double marks), but detection of ventricular tachycardia is suspended for 64 ventricular events after ventricular fibrillation detection. Thus postventricular fibrillation detection of ventricular tachycardia requires 80 (64 + 16) intervals. Telemetered markers were recorded for 76 of the 80 postventricular fibrillation events (70 TS, 5 FS, one cardioversion), and two of the TS marks were cut inadvertently during mounting. The telemetry marker indicating the end of the ventricular fibrillation therapy capacitor charge and markers for four other intervals around delivery of ventricular fibrillation therapy were not recorded. Detection of ventricular tachycardia is followed by delivery of the first programmed ventricular tachycardia therapy, a 0.64-J cardioversion (CD) (long double downmark followed by long double up-mark), which is ineffective. The asterisk identifies the long double marker for capacitor charge corresponding to the therapy. VR (short single mark) denotes a sensed event in the postshock refractory period. With detection, active ventricular tachycardia is redetected rapidly and terminated with the second programmed ventricular tachycardia therapy, a 5-J cardioversion. Excluding delays due to ventricular fibrillation intervals, postventricular fibrillation detection of ventricular tachycardia requires 20 s.

Conclusions. Underdetection of ventricular tachycardia is a consequence of imperfect algorithms to enhance specificity for detection of ventricular tachycardia. In this study, underdetection was infrequent. Serious consequences were
rare and were prevented by reprogramming. Algorithm improvements and specific failsafe mechanisms may be beneficial. Future algorithms, including electrogram configuration (5), dual-chamber analysis (27) and hemodynamic sensing (28), may further increase specificity but may also harbor unforeseen problems.

References