Incidence and Clinical Relevance of Slow Ventricular Tachycardia in Implantable Cardioverter-Defibrillator Recipients
An International Multicenter Prospective Study

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Background—This study aims to assess the incidence and clinical relevance of slow ventricular tachycardia (VT) and the effectiveness and/or deleterious effects of antitachycardia pacing in slow VT in implantable cardioverter-defibrillator recipients.

Methods and Results—This multicenter prospective randomized study included 374 patients (326 men) without prior history of slow VT (<148 bpm) implanted with a dual-chamber implantable cardioverter-defibrillator. Patients had a 3-zone detection configuration: a slow VT zone (101 to 148 bpm), a conventional VT zone (>148 bpm), and a ventricular fibrillation zone. Patients were randomized to a treatment group (n=183) with therapy activated in the slow VT zone or a monitoring group (n=191) with no therapy in the slow VT zone. During follow-up (11 months), 449 slow VTs occurred in 114 patients (30.5% slow VT incidence); 181 VTs (54 patients) occurred in the monitoring group; 3 were readmitted to the hospital; and lightheadedness and palpitations occurred in 4 and 250 (60 patients) in the treatment group treated by antitachycardia pacing (89.8% success rate) and shock delivery (n=2). There were 10 crossovers from the monitoring to treatment group and 3 crossovers from the treatment to monitoring group (P=0.09). Quality of life scores were not different between groups.

Conclusions—Slow VT incidence (<150 bpm) is high (30%) in implantable cardioverter-defibrillator recipients without prior history of slow VT, has limited clinical relevance, and is efficiently and safely terminated by antitachycardia pacing. (Circulation. 2005;112:946-953.)

Key Words: arrhythmia ■ defibrillation ■ tachyarrhythmias ■ tachycardia

Implantable cardioverter-defibrillators (ICDs) efficiently terminate ventricular tachycardia (VT) and ventricular fibrillation (VF), thus prolonging life in patients at high risk of ventricular arrhythmias. Their efficacy is linked to their ability to appropriately detect ventricular arrhythmia. Primary VT detection is based on the tachycardia detection rate (TDR), representing the rate above which therapy is applied. In clinical practice, TDR is usually programmed with a safety margin of 20 bpm below the clinical arrhythmia.1

Some patients may exhibit slow VT (below the TDR), sometimes causing symptoms such as syncope, palpitations, and congestive heart failure, leading to a high incidence of hospital readmission2-3 and sometimes death.3

Programming a low TDR decreases the incidence of undetected slow VTs but may lead to inappropriate detection and treatment of supraventricular tachycardia (SVT), usually sinus tachycardia (ST).4 Inappropriate VT therapy during SVT is particularly distressing because the full sequence of ICD therapies, including painful unnecessary shocks, is often delivered.

To overcome this limitation, detection specificity has been enhanced by additional criteria such as sudden onset,4 stability,4,5 ventricular electrogram (EGM) morphology,6 or EGM vector timing and correlation7 and, in the case of dual-chamber systems, by the relationship between the atrium and the ventricle.8-13

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The Data Supplement, which contains an Appendix listing the participating investigators and institutions, can be found at http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.533513/DC1.

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The PARAD algorithm implemented in dual-chamber ICDs from Ela Medical has a high sensitivity (98.5%) in ST and/or atrial flutter and atrial tachycardia detection. We have recently reported in a preliminary study that programming a low (128 bpm) TDR was not associated with inappropriate SVT detection.

The aim of this prospective multicenter international 2-arm randomized study was to assess the incidence and clinical relevance of slow VT (101 to 148 bpm) in ICD recipients, the safety of programming a low TDR (101 bpm), and the effectiveness and/or potential deleterious effects of ATP therapy in slow VT. It was approved by all appropriate ethics committees of the enrolling medical centers. Written informed consent was duly obtained from each study participant. After implantation, all patients had a 3-zone detection configuration programmed as follows: (1) a slow VT zone, ranging from 101 to 148 bpm; (2) a conventional VT zone, ranging from 148 to at least 183 bpm (up to 213 bpm); and (3) and a VF zone.

Patients were randomized to 2 groups: a treatment group with ATP (mandatory, but ATP parameters left to investigator’s discretion) with or without cardioversion (optional) activated in the slow VT zone or a monitoring group with no therapy programmed in the slow VT zone. Conventional ICD therapy was programmed in the conventional VT zone (ATP and shock) and VF zone (shock).

### Pulse Generators, PARAD and PARAD+ Algorithms, Programming Parameters, and Detection Duration

A Defender IV was implanted in 321 patients; the remaining 53 patients received an Alto DR. Both include the PARAD/PARAD+ detection algorithms, which have been previously described and are briefly summarized below. PARAD operates only within the tachycardia zones (ie, in the slow VT and conventional VT zones) to differentiate ST or SVT from VT. The first step is classification of each cycle as slow (below the TDR), tachycardia (above the TDR and below the VF detection rate), or fibrillation (above the VF detection rate). A minimum of 6 of 8 consecutive cycles falling within the tachycardia zone is necessary to activate PARAD, which further refines the tachycardia diagnosis on the basis of 3 criteria: stability, PR (AV) association, and acceleration and its chamber of origin (atrium or ventricle) if present. To store the arrhythmic event in the device memory, the rhythm classification must be confirmed, after initial detection, over a minimum of 12 cycles. Left to investigator’s discretion, the algorithm could also be programmed in the PARAD+ mode, which withholds VT therapy in case a long
ventricular cycle is detected. The detection algorithms were programmed in the standard AV mode.

When programmed with a 2-VT-zone configuration, the ICD detection configuration allows overlap between the lower limit of the VT detection rate and the pacemaker upper tracking rate. This allowed programming of the VT detection rate to 101 bpm, even in patients with second- or third-degree AV block (35 of 374) or with sinus node disease (77 of 374) (Table 1).

**Patient Follow-Up**

Patients were seen in the outpatient clinic at 1 month and every 3 months for 12 months (complete study follow-up duration) or whenever the device was activated. At these times, statistics and Holter data were retrieved and stored. Early study termination included death, patient or physician’s decision, severe ICD complications, and technical reasons.

**Quality of Life**

Quality of life (QOL) was measured with the Minnesota Living With Heart Failure Questionnaire before implantation and at each 3-month follow-up. The Minnesota questionnaire is a patient self-assessment that includes 21 items with a 6-point response format, ranked from 0 to 6. Lower scores represent better QOL. QOL scores were compared between the 2 groups at baseline, at the end of follow-up, and within groups at each follow-up (see Statistical Analysis).

**Data Analysis**

All episodes detected within the VF zone were excluded. Every single sustained (>12 seconds) and/or treated spontaneously fully documented episode detected either in the slow VT zone or in the conventional zones and stored in the Holter memory was analyzed. This 12-second value was chosen because it represents the time necessary to detect 6 of 8 RR cycles plus 12 RR cycles at the longest VT cycle length (ie, 600 ms), which are necessary to activate PARAD, to confirm the tachycardia classification, to treat when necessary, and to store the event. Each episode was reviewed by the study investigators. When the arrhythmia classification was considered inappropriate or equivocal, the data were submitted to an expert panel that assigned the ultimate diagnosis. Sensitivity was calculated for all episodes sensed within the tachycardia zones and defined as true-positive/all-clinical VT episodes in which true positive is a clinical VT episode correctly classified by the device as VT. Specificity was calculated for all episodes sensed within the tachycardia zones and defined as true-negative/all-clinical ST/SVT episodes in which a true negative is a clinical SVT or ST episode correctly classified by the device as SVT/ST.

**Statistical Analysis**

All statistical tests were 2 sided and performed on an intention-to-treat approach. A value of \( P<0.05 \) was considered statistically significant. Because a single patient may exhibit multiple episodes, which may introduce a possible bias in the calculation of sensitivity and specificity, algorithm performance was calculated not only on a per-episode basis, in which all episodes are considered independently regardless of the number of episodes per patient, but also on a per-patient basis with the generalized estimating equation. Sensitivity and specificity are expressed with a 95% bilateral CI. Fisher’s exact test was used to compare the incidence of severe adverse events in each group. Student \( t \) test for matched series was used to compare changes in QOL between baseline and 1 year in each group, and Student \( t \) test for independent series was used to compare each group at each follow-up. When the intragroup assumptions of normality or homogeneity of variances did not hold, the nonparametric version of these tests was retained (Mann-Whitney).

**Results**

Data were collected over a mean follow-up of 11±3 months (range, 24 days to 13 months). There were 183 patients in the ATP group and 191 patients in the monitoring group (\( P=NS \)) with no differences between the 2 groups (Table 1). Antiarrhythmic drug therapy remained stable over the course of the study (Table 2).

**Patient Survival**

During the period of observation, 23 patients died: 10 in the monitoring group and 13 in the therapy group (\( P=NS \)) (Table 3). The main cause of death was congestive heart failure (n=9; 5 in the treatment group, 4 in the monitoring group; \( P=NS \)).

**Spontaneous Arrhythmic Events**

**Slow VT Incidence and Detection Algorithm Performance**

A total of 6763 episodes (Figure 1) were detected in 337 of the 374 patients, of which 6621 were documented in 336 patients in the conventional or slow VT zones. Among these 6621 episodes, 769 were detected in the conventional VT zone (>148 bpm). The remaining 5852 (6621−769) episodes were detected in the slow VT zone in 328 patients, among which 449 (in 114 patients) were classified as slow VT by the investigators with an appropriate device classification in 431 episodes (18 VT episodes inappropriately classified as SVT) (Table 4). Algorithm sensitivity in the slow VT zone was 96% (431 of 449), and the slow VT incidence was 30.5% (114 of 374, ie, number of patients with slow VT in the overall population). The mean rate of these 449 episodes was 127±13 bpm (range, 101 to 147 bpm). The incidence of slow VT according to rate is given in Figure 2.

The other 5403 (5852−449) episodes detected in the slow VT zone were classified as SVT (13%) or ST (87%) by the investigators with an appropriate device classification in 5166 cases (237 SVT episodes inappropriately classified as slow VT) (Table 4). Algorithm specificity in the slow VT zone was 96% (5166 of 5403). Specificity and sensitivity according to various slow VT cutoff rates are given in Figure 3. It can be seen that although the VT cutoff rate increases, specificity remains relatively stable, whereas sensitivity progressively increases to reach 100% above 140 bpm.
On a per-patient basis, the algorithm sensitivity and specificity are 95.7% (95% CI, 91.5 to 97.9) and 95.4% (95% CI, 93.7 to 96.6), respectively.

Clinical Tolerance of Slow VT, Efficacy, and Side Effects of Slow VT Therapy

Slow VT tolerance, as well as efficacy and side effects of slow VT therapy, were assessed in the 431 slow VT episodes detected by the device.

In the monitoring group, 181 slow VTs were documented in 54 patients. In 26 episodes (14 patients), VT started in the slow VT zone but progressively accelerated and was eventually detected in the conventional VT zone and hence treated. The rate of these 26 episodes at onset was 139 ± 20 bpm; their duration was 55 ± 200 minutes (range, 16 seconds to 16 hours). The rate and duration of the 155 remaining episodes were 126 ± 11 bpm (range, 104 to 142 bpm) and 15 ± 113 minutes (range, 12 seconds to 24.3 hours), respectively. The longest event (1 day) led to emergency hospital admission. It was interrupted by manual ATP via the programmer. Two other patients were admitted as an emergency for symptomatic slow VT. Four other patients complained of palpitations and lightheadedness related to slow VT on Holter, but the symptoms were not severe enough to lead to emergency hospital admission.

In the treatment group, 250 slow VTs were documented in 60 patients (mean rate, 127 ± 13 bpm; range, 101 to 147), among which 245 were treated and 5 spontaneously decelerated below the TDR. Initial therapy always consisted of ATP, with a success rate of 89.8% (220 of 245) with no deleterious ATP effects except for 1 episode accelerated by ATP to the conventional VT zone and subsequently interrupted by ATP in this zone. In 13 of the 25 episodes (6 patients) with ATP failure, 5 were interrupted by shocks (2 patients), whereas in the 4 patients with no shocks programmed in the slow VT zone, spontaneous VT termination occurred for the 8 remaining episodes, which lasted from 1 minute to 3.5 hours. In the remaining 12 episodes (5 patients), the VT rate progressively accelerated to the conventional VT zone where it was detected and then treated.

There was no statistical difference in the number of slow VT episodes between the 2 groups, but there was a statistical difference in symptoms (P = 0.02) in favor of the treatment group.

Clinical Relevance of Inappropriate Slow VT Detection and Inappropriate Slow VT Therapy

Among the 18 slow VT episodes inappropriately classified as SVT by the device (false negatives), 6 occurred in the monitoring group, and 12 were in the treatment group. These
18 misclassified episodes lasted from 12 seconds to 42 minutes; 13 episodes lasted <2 minutes. They were totally asymptomatic.

Among the 237 SVTs inappropriately classified as VT by the device (false positives), 157 (33 patients) occurred in the monitoring group, and 80 (30 patients) occurred in the treatment group. In the latter group, inappropriate VT detection led to ATP therapy with no deleterious effects in 26 patients and to shock delivery after ATP failure (n=5) in 4 patients. As expected, the difference in terms of shocks favors the monitoring group (P=0.03) because no slow VT therapy was activated in this group.

**Crossover Before the End of the Study**

There were 10 patients for whom investigators requested crossover from the monitoring to the treatment group for slow VT. These 10 patients included the 3 patients with emergency hospital admission, the 4 patients with symptomatic slow VT (see above), and 3 other patients in whom VT therapy was activated by the investigators in the slow VT zone because of slow VT stored in the device memories despite lack of symptoms.

There were 3 investigator requests for crossover from the treatment to the monitoring group because of inappropriately detected atrial fibrillation treated by ATP in 3 patients with no ATP side effects (no shock delivered or VT acceleration).

No crossover was requested by the investigators for inappropriate shocks in SVT (see above).

The difference in crossover rate between the 2 groups does not reach statistical significance (P=0.09).

**QOL Assessment**

QOL score was lower at baseline in the monitoring group than in the treatment group (26.0±18.7 versus 29.3±22.0; P=NS), and the difference remained stable (22.7±19.2 in the monitoring group versus 24.7±20.3 in the treatment group at 1-year follow-up; P=NS). QOL continuously and significantly improved throughout the study in each group (P=0.002 in the monitoring group, P=0.0007 in the treatment group from baseline to 12 months). Statistical analysis did not reveal any statistical difference between the 2 arms during follow-up.

**Discussion**

The main findings of this study are that almost one third (30.4%) of ICD recipients without prior symptomatic or ECG documented slow VT history exhibited slow VT during a 1-year follow-up. These slow VTs can be efficiently interrupted by ATP but seem to have limited clinical relevance.

**Incidence of Slow VT**

This study reports a 30.4% incidence of slow VT in ICD recipients without a prior history of symptomatic or ECG-
documented slow VT. Data are scarce on the incidence of slow VT in ICD recipients. In a retrospective study including 154 patients, Nunain et al report, over a mean follow-up of 15.3 months, a 7.9% incidence of symptomatic VT occurring below the programmed TDR. Bänsch et al, in a study that included 659 patients with a follow-up of 31 months, found an incidence of VT occurring below the TDR ranging from 2.7% to 3.5% per year during the first 4 years after ICD implantation. The discrepancies between these 2 studies and the present one can be explained easily. In the present study, all episodes >101 bpm and lasting >12 seconds were recorded and analyzed. In the work by Nunain et al, only symptomatic episodes were analyzed, and in the Bänsch et al study, only episodes with ECG documentation, 12-channel ECG, or 24-hour Holter recordings, as well as symptomatic episodes leading to hospital readmission, were included. With the same inclusion criteria, the number of patients with slow VT in the present study would have been 15 (the 7 symptomatic patients in the monitoring group plus the 8 patients, 4 in each group, in whom VT accelerated to the conventional VT zone), with a 1-year slow VT incidence of 4% (15 of 374). Another possible explanation is the unusually low TDR programmed in the present study, not only for the lower limit of the slow VT zone (101 bpm) but also for the lower limit of the conventional zone (148 bpm). Since the studies by Bänsch et al and Nunain et al were retrospective and nonrandomized, there are no data on the cutoff value for VT detection, but it is likely that the devices were programmed in a conventional manner, with TDR >150 or 160 bpm. In fact, in the Bänsch et al study, the mean rate of undetected VT was 159 bpm. It is very likely that slow VT incidence was probably identical in these 3 studies, but the unusually low VT detection rate in the present study provides a level of surveillance that did not exist in the 2 preceding studies.

Clinical Relevance of Slow VT

There was no statistical difference in the clinical outcome of patients in the monitoring group compared with patients in the treatment group. This is surprising because slow VT can be a harbinger of clinical deterioration or an indication of electrolyte abnormalities or ischemia. Hence, treating slow VT would seem appropriate. However, although untreated slow VTs were sometimes symptomatic, emergency hospital admission was required in only 3 patients. This reason may be that most of these untreated VTs did not last long enough (mean duration, 15 minutes) to become symptomatic or that the follow-up was too short (11 ± 3 months). For example, in study by Bänsch et al, the follow-up was 3 times longer (33 versus 11 months). In our experience, incessant, slow VT in ICD recipients is not unusual. In a study from our institution reporting the results of slow VT ablation (mean VT cycle length, 432 ms) in 21 patients with coronary artery disease, 10 were ICD recipients, of whom 6 had symptomatic, incessant, slow VT below the detection rate that led to emergency hospital admission. Another possible explanation is the value of the ejection fraction of the patients included in the study (39%), a higher value than in most ICD trials of primary or secondary prevention. This may explain in part the lack of symptoms of untreated slow VT episodes in the monitoring group because symptoms are related not only to the arrhythmia rate but also to the quality of the pump function.

The nonclinical relevance of slow VT in this study is further enhanced by the lack of difference in the Minnesota QOL scores between the 2 groups. Once again, this finding may be due to an insufficient follow-up period. Another
explanation may be that the Minnesota questionnaire,\(^{19}\) developed to assess patients with heart failure,\(^ {20}\) is not appropriate for detecting small variations in QOL in ICD recipients in whom heart failure is not always the main symptom.

**Safety of Programming a Low Detection Rate and Efficacy of Slow VT Therapy**

Although it is accepted that programming a low TDR increases the rate of inappropriate detection and hence inappropriate therapy, it was not associated with a high incidence of inappropriate therapy in the present study. The overall specificity was 96%, a value that probably is overestimated because of the detection of numerous ST episodes (87% of all ST/SVT episodes) that are very efficiently detected by the PARAD/PARAD+ algorithms with an ST specificity detection of 98.5%.\(^ {10}\)

On the other hand, decreasing the detection rate while preserving high specificity may decrease the sensitivity and may be harmful to the patient, as was the case in this study in which sensitivity was only 96%, with 18 slow VT episodes incorrectly classified as SVT by the algorithm. This is further underlined by the fact that sensitivity was related to the tachycardia rate, with the lowest value (93%) for the lowest VT cutoff rate (101 bpm) (Figure 3). This is way below 100%, the goal for all detection algorithms in the conventional ICD era. Because slow VTs do not appear to be an emergency life-threatening situation and are most often short lasting, such a “low” sensitivity value may be acceptable.

To the best of our knowledge, when programmed with a 2-VT-zone configuration, only the ELA Medical ICD detection configuration allows overlap between the lower VT detection rate limit and the pacemaker upper tracking rate. In other words, the slow VT detection rate can be programmed to 101 bpm while the pacemaker upper tracking rate can be programmed to a maximum value of 142 bpm. This allows programming of such a slow VT detection rate even in pacemaker-dependent patients. This unique feature is due to the fact that paced ventricular cycles are ignored in the count of cycles necessary to fulfill the tachycardia detection criteria and hence do not jeopardize tachycardia detection.

Slow VTs are efficiently terminated by ATP, with an ATP success rate of 90% in this study. ATP was not associated with deleterious effects such as VT acceleration except in 1 case in which VT was accelerated to the conventional VT zone, subsequently detected, and efficiently terminated by ATP.

**Study Limitations**

Only 12 to 25 fully documented tachycardia episodes could be retrieved from the Holter memory at each follow-up visit for each patient. Hence, the data collected represent only a sample of the total number of events occurring among the patient population. Because of the low TDR programmed, ST is by far the most frequent “arrhythmia” detected in the slow VT zone. This high number of ST episodes may have erased true slow VT from the Holter memory. This may be particularly relevant in the monitoring group in which slow VT, even when appropriately detected, was not treated and hence not kept as priority episodes in the Holter memory. This could explain why there is a trend for a higher incidence of slow VT in the treatment group. This Holter saturation by physiological arrhythmia may also lead to an underestimation of slow VT incidence.

Because a certain proportion of slow VTs are short lasting, the ATP success rate of slow VT may be slightly overestimated in that ATP is delivered in VTs that would have ended spontaneously a few seconds later. Indeed, it has been shown in an article reporting the effects of ATP in fast VT that approximately one third of fast VTs terminate during capacitor charging, whereas ATP is delivered immediately after detection criteria have been fulfilled.\(^\text{21}\) This ATP efficacy overestimation, however, probably is marginal because in this study the time from VT onset to VT detection and hence to VT therapy in the treatment group ranges from >8 seconds for the fastest VT (just below 148 bpm) to 12 seconds for the slowest VT (just above 101 bpm), whereas stored episodes in the monitoring groups lasted ≥12 seconds.

**Conclusions**

This study shows that slow VT (<150 bpm) is a common feature in ICD recipients. Most of these slow VTs are short lasting and are associated with few or no symptoms. ATP efficiently terminates slow VT and is not associated with deleterious effects. Considering that most slow VT episodes do not lead to a life-threatening situation, slow VT therapy should consist of ATP therapy only, whereas ATP followed by shocks should be programmed only in the conventional VT zone.

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**References**

Few data are available on the incidence and clinical relevance of slow ventricular tachycardia (VT) in ICD recipients, the safety of programming a low VT detection rate, and the usefulness of treating such arrhythmic episodes. We addressed these issues in a prospective multicenter 2-arm randomized study including 374 patients without prior history of slow VT (<148 bpm) implanted with a dual-chamber implantable cardioverter-defibrillator (ICD) and programmed with a slow VT zone (101 to 148 bpm), a conventional VT zone (>148 bpm), and a ventricular fibrillation zone. Patients were randomized to a treatment group (n=183) with therapy activated or a monitoring group (n=191) with no therapy activated in the slow VT zone. Approximately one third of ICD recipients exhibited slow VTs during an 11-month follow-up; most of these were short lasting (mean, 15 minutes) and associated with few or no symptoms. In the treatment group, the ATP success rate was 90% with no side effect. Programming a low VT detection rate was not associated with a reduction in VT detection specificity (>90%) but was associated with a reduction in VT detection sensitivity, which ranged from 93% for the lowest VT rates (<110 bpm) to ~100% for the highest VT rates (>148 bpm). The limited clinical relevance of these slow VTs may be due to the insufficient follow-up period. It is conceivable that in the future, with the improvements in detection algorithms, tachycardia detection rates will be programmed at lower values to treat slow VT.
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