Defibrillation testing is mandatory in patients with subcutaneous implantable cardioverter–defibrillator to confirm appropriate ventricular fibrillation detection

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BACKGROUND The subcutaneous implantable cardioverter–defibrillator (S-ICD) remains a new technology requiring accurate assessment of the various aspects of its functioning. Isolated cases of delayed sensing of ventricular arrhythmia have been described.

OBJECTIVE The purpose of this multicenter study was to assess the quality of sensing during induced ventricular fibrillation (VF).

METHODS One hundred thirty-seven patients underwent induction of VF at the end of the S-ICD implantation.

RESULTS VF induction was successful in 133 patients (97%). Mean time to first therapy was 16.2 ± 3.1 seconds, with a substantial range from 12.5 to 27.0 seconds. Four different detection profiles were arbitrarily defined: (1) optimal detection (n = 39 [29%]); (2) undersensing with moderate prolongation of time to therapy (<18 seconds; n = 68 [51%]); (3) undersensing with significant prolongation of the time to therapy (≥18 seconds; n = 19 [14%]); and (4) absence of therapy or prolonged time to therapy related to noise oversensing (n = 7 [6%]). In some of the patients in the last group, despite induction of VF the initial counter was never filled, the device did not charge the capacitors, and the shock was not delivered because of a sustained diagnosis of noise (n = 5). A manual shock by the device or an external shock had to be delivered to restore the sinus rhythm.

CONCLUSION Our study demonstrated a marked sensing delay leading to prolonged time to therapy in a large number of S-ICD patients. A few worrisome cases of noise oversensing inhibiting the therapies were detected. These results support the need for systematic intraoperative defibrillation testing.

KEYWORDS Complication; Defibrillation testing; Noise; Subcutaneous implantable cardioverter–defibrillator; Undersensing

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Introduction

The subcutaneous implantable cardioverter–defibrillator (S-ICD) represents an efficient alternative to a transvenous device in patients who do not require pacing and who are at risk for device-related complications over their lifetime.1–3 The S-ICD is entirely extrathoracic and leaves the heart and vasculature untouched. Avoiding the intravascular space with an S-ICD completely modifies the sensing characteristics compared to the “near-field” sensing of a transvenous system because subcutaneous signals have lower amplitude, longer duration, and lower frequency

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Patient baseline characteristics are summarized in Table 1. One hundred thirty-seven consecutive S-ICD systems were implanted in 4 institutions and were retrospectively analyzed. Patients provided informed consent for S-ICD implantation and testing.

Mean ejection fraction (%) 47
Noncompaction cardiomyopathy 2 (2%)
Long QT syndrome 4 (3%)
Myocarditis 6 (4%)
Idiopathic VT/VF 17 (12%)
Brugada syndrome 17 (12%)
Hypertrophic cardiomyopathy 18 (13%)
Ischemic heart disease 52 (38%)

DFT in S-ICD patients are limited.6,7 Extensive data on the quality of sensing during intraoperative testing by delivery, via the programmer, of a 50-Hz DC burst for 16–20 seconds. During the induction, the number of zones (1 single shock zone or 1 shock zone and 1 conditional zone) and the zone cutoffs were programmed according to the physician’s choice as follows: (1) 1 single zone programmed from 170–200 bpm (n = 77); (2) 2 zones with a conditional zone from 170–200 bpm and a shock zone from 200–230 bpm (n = 56); and (3) 2 zones with a conditional zone at 220 bpm and a shock zone at 240 bpm (n = 4). The first shock energy was programmed to 65 J, and the second shock energy was programmed to 80 J in reversed polarity followed by external rescue shocks if ineffective.

Sensing with an S-ICD
The S-ICD system senses subcutaneous signals from a dipole defined as primary (proximal electrode ring to can), secondary (distal electrode ring to can), or alternate (distal to proximal electrode) vector. After implantation, the system automatically selects the optimal vector for detection and gain combination based on the R- to T-wave amplitude ratio to avoid QRS and T-wave oversensing.

To minimize undersensing of VF and to prevent T-wave oversensing, the device operates with a low sensing floor (0.08 mV or 80 μV) and a low high-pass filter (3 Hz) that cannot be altered in any manner.

The S-ICD sensing algorithm comprises 3 phases:
1. The sensed event detection phase filters the input signal and generates sensed events for further analysis. The S-ICD uses automatic sensitivity adjustment to reduce T-wave oversensing and sensing refractory periods to prevent R-wave double-counting with different profiles. The sensing threshold is adjusted based on the amplitude of the preceding 2 QRS complexes. Once an elevated heart rate is certified, threshold stringency is progressively relaxed as the heart rate increases. The refractory period and the decay profile are more sensitive in the shock zone than in the conditional zone. Therefore, addition or removal of a tachycardia detection zone alters the sensing profile on a beat-to-beat basis.
2. The certification phase classifies the sensed events as certified QRS complexes or as suspected oversensing events and calculates an accurate ventricular rate. A waveform algorithm uses frequency and slew rate analysis to ensure the signal is cardiac in origin and to reject myopotentials and electromagnetic interference, corresponding to the “N” (noise) marker on the electrogram (EGM). The intervals associated with the noise events are discarded. The remaining sensed events are then passed through 4 certification algorithms to recognize and correct for R-wave double-counting and T-wave oversensing. A dot “●” marker on the EGM labels the uncertain “oversensed” events. The S-ICD measures heart rate as the rolling average of 4 consecutive certified intervals.
3. The decision phase detects VF and ventricular tachycardia (VT) and discriminates the latter from a supraventricular tachycardia (SVT). In the shock zone, the device detects VF using only rate and duration. In the conditional zone, the device also uses SVT–VT discrimination based on “static” EGM morphology (comparison with sinus template), QRS duration, and “dynamic” EGM morphology.

Methods
Patients
The study was approved by the Institutional Committee on Human Research at the authors’ institution, and all patients provided informed consent for S-ICD implantation and testing. One hundred thirty-seven consecutive S-ICD systems were implanted in 4 institutions and were retrospectively analyzed. Patient baseline characteristics are summarized in Table 1.

Implantation procedure
Implantation was performed with the patient under general anesthesia using a technique involving 2 or 3 incisions and placement of the midaxillary pulse generator under the subcutaneous tissue or intermuscularly.8 In this multicenter observational study, the quality of sensing in 137 consecutive patients with an S-ICD undergoing intraoperative defibrillation tests was systematically evaluated.

Intraoperative defibrillation test
The device automatically selected the sensing vector. All patients underwent standardized intraoperative defibrillation test by delivery, via the programmer, of a 50-Hz DC burst for 4–10 seconds. During the induction, the number of zones (1 single shock zone or 1 shock zone and 1 conditional zone) and the zone cutoffs were programmed according to the physician’s choice as follows:

<table>
<thead>
<tr>
<th>No. of patients</th>
<th>137</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48 ± 15</td>
</tr>
<tr>
<td>Male</td>
<td>96 (70%)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>52 (38%)</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>18 (13%)</td>
</tr>
<tr>
<td>Brugada syndrome</td>
<td>17 (12%)</td>
</tr>
<tr>
<td>Idiopathic VT/VF</td>
<td>17 (12%)</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>16 (12%)</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>6 (4%)</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>5 (4%)</td>
</tr>
<tr>
<td>Long QT syndrome</td>
<td>4 (3%)</td>
</tr>
<tr>
<td>Noncompaction cardiomyopathy</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Mean ejection fraction (%)</td>
<td>47 ± 16</td>
</tr>
<tr>
<td>Primary prevention implant</td>
<td>77 (56%)</td>
</tr>
</tbody>
</table>

Values are given as mean ± SD or n (%) unless otherwise indicated. VF = ventricular fibrillation; VT = ventricular tachycardia.
The last criterion classifies detected tachyarrhythmias as shockable if their beat-to-beat morphology varies sufficiently to indicate that the rhythm is polymorphic.

The detection zone can be programmed from 170–250 bpm in 10-bpm intervals, with the option to program a 2-zone therapy: a shock zone, in which the only criterion is the actual heart rate, and an optional conditional zone with a lower rate cutoff between 170 and 240 bpm to distinguish supraventricular from ventricular tachyarrhythmias with the INSIGHT rhythm discrimination algorithm (Boston Scientific, MN, USA).9

The system uses initial nonprogrammable 18 of 24 duration criteria before capacitor charging. The charge confirmation algorithm is the final analysis performed before the capacitors are charged for therapy delivery and requires that at least 16 T markers are contained within the 24-beat window and that the 2 most recent sensed signals are fast

A confirmation algorithm is also used after completion to capacitor charge to ensure persistence of the ventricular arrhythmia before shock delivery.

**Measurements**

Time to charge and time to therapy for induced episodes were defined as the interval starting 2 seconds after the end of the 50-Hz DC burst (a delay to allow recovery from amplifier saturation) and ending at the first marker C (charge) and at the marker corresponding to the shock delivery.

**Statistical analysis**

Continuous variables are reported as mean ± and were tested for normality using the Shapiro–Wilk test. Comparison between groups of continuous variables was performed using either 1-way analysis of variance or the Kruskal–Wallis test. Categorical variables are given as count and percentage, and comparison between groups was performed using the Fisher exact test. A 2-sided P < .05 was considered significant, and Bonferroni correction was applied for multiple tests. Statistical analyses were performed using SPSS software, version 18.0 (SPSS Inc, Chicago, IL).

**Results**

**Inducibility**

All implantation procedures were completed without acute complications. At the end of the procedure, all 137 patients underwent VF induction. The automatically selected vectors were as follows: primary (n = 69 [50%]), secondary (n = 54 [40%]), and alternate (n = 14 [10%]). VF induction was successful in 133 patients (97%); 4 patients were not inducible despite numerous attempts with DC bursts of different duration.

**Reliability of induced-arrhythmia detection**

In 5 patients (4%), the initial counter was never filled, and the device did not charge the capacitors because of noise oversensing. In the other patients (n = 128 [96%]), mean time to first therapy was 16.2 ± 3.1 seconds, with a widely varying range from 12.5 to 27.0 seconds. Four different detection protocols were arbitrarily defined according to the occurrence of undersensing and according to the time to first therapy. The groups did not differ statistically in terms of age, sex, ejection fraction, or sensing vector programmed. In contrast, there was a statistically significant difference in the programmed limit of the shock zone between the different groups (group 1: 191 ± 26 bpm; group 2: 199 ± 27 bpm; group 3: 216 ± 22 bpm; group 4: 210 ± 28 bpm; P < .01 among all groups and P < .01 between groups 1 and 3).

**Optimal detection**

In these patients (n = 39 [29%]), 4–6 S markers were initially observed, followed by the first T marker and then no further S markers until shock delivery (excluding the S marker(s) occurring just before shock delivery). A few signals could be undersensed but without sufficient impact on the heart rate to change the classification of the cycles (from T to S). Mean time to therapy in this group was 13.7 ± 0.8 seconds (Figure 1).

**Undersensing with moderate prolongation of time to therapy (time to first therapy <18 seconds)**

In these patients (n = 68 [51%]), 1 or more S markers were observed after the occurrence of the first T marker. This undersensing resulted in a modest delay in time to first therapy (<18 seconds). Mean time to therapy was 15.8 ± 1.5 seconds.

**Undersensing with significant prolongation of time to therapy**

In these patients (n = 19 [14%]), the time to first therapy was >18 seconds (Figure 2). Mean time to therapy was 22 ± 1.8 seconds, with >21 seconds in 12 patients and >24 seconds in 1 patient. In this latter patient, the physician decided to induce a second episode using a different vector, with a return to a better (<18 seconds) time to first therapy.

In only 2 patients of this group, the conditional zone was programmed >200 bpm. In these specific patients, programming a zone from 200 bpm would have further increased the sensitivity and probably would have led to better detection and classification of more events as tachycardia, decreasing the time to therapy.

**Absence of therapy or prolonged time to therapy related to noise oversensing**

In 5 patients (4%), despite induction of VF the initial counter was never filled, the device did not charge the capacitors, and the shock was not delivered because of a sustained diagnosis of noise (repeated N markers) with visualization of fine noise on the EGM superimposed over the fast ventricular events (Figures 3 and 4). The intervals associated with the noise events were discarded, explaining the absence of detection of the arrhythmia. A manual shock by the device or an external shock had to be delivered to restore sinus rhythm after a mean VF duration of 30.5 ± 5.1 seconds. The source of the noise was deeply investigated with the help of Boston Scientific technical service. However, no definite conclusion could be drawn from the tracing analysis. Electromagnetic interference was highly unlikely based on the slow and random frequency content of the noise. Far-field sensing of
diaphragmatic muscular activities remained the most likely hypothesis.

In these 5 patients, the sensing vector was the primary vector, after which the sensing vector was changed to secondary in 4 patients, and VF induction testing was repeated. In 1 patient, VF induction was repeated with the primary vector. For all of these patients, appropriate detection and treatment of VF were noted during this second induction, with mean time to therapy of 17.1 ± 1.2 seconds.

In 2 additional patients (2%; 1 with a primary vector and 1 with an alternate vector), the presence of noise immediately after induction caused a delay in VF detection with a time to therapy of 20.0 and 24.0 seconds, respectively.

**Efficacy of defibrillation**

The S-ICD successfully restored sinus rhythm in 118 of the 128 patients (92%) with the first 65-J shock. In 7 patients (6%), the first internal shock was ineffective, and a further 80-J defibrillation with reversed polarity was effective. In 3 patients (2%), after failure of these 2 shocks, delivery of an external shock was required to restore sinus rhythm. These patients underwent fluoroscopic examination searching for potential improvement of the lead/can positioning or shock vector. Repositioning of the sensing lead and/or the pulse generator under fluoroscopic control was performed, with the can being moved to a more cranial position in 1 patient, whereas in 1 patient the lead was moved to a right parasternal position, which resulted in a successful defibrillation. The S-ICD system has remained implanted in all patients.

**Discussion**

In the field of defibrillation, development of the S-ICD represents the most striking evolution in the past 10 years. This technology is seemingly able to avoid a large number of
complications associated with the presence of endocardial leads that impair both the quality of life and the prognosis of implanted patients.\textsuperscript{10–12} Although the potential benefits of the S-ICD seem obvious in terms of preventing complications, the prerequisite for its broad dissemination is demonstration of at least an equivalent efficacy compared to the transvenous ICD in terms of the 2 fundamentals of this type of therapeutic treatment: namely, the ability to correctly detect, without delay, a ventricular rhythm disorder, followed by the ability to reduce the latter...
with an electric shock. The first clinical results seem highly encouraging, although the description of a few isolated clinical cases suggests some instances with a more or less marked sensing delay. 

Our study focused on the quality of sensing during induced arrhythmia in patients implanted with an S-ICD. Our main findings are as follows. (1) With the S-ICD, the average interval between the onset of the arrhythmia and the electric shock
was 16 seconds, which is much longer than for a transvenous ICD with standard programming. It should be emphasized that given the shock energy tested during induction was 65 J, the charge time as a result of a spontaneous arrhythmia might be longer, the energy delivered being 80 J. (2) There was considerable interindividual variability in the time to therapy. This variability seemed to be much greater than that observed with a transvenous ICD. (3) There was a relatively large subgroup of patients with significantly prolonged time to therapy. The policy to adopt in the presence of a prolonged sensing delay is not standardized. (4) In a limited number of patients, we found a worrisome absence of therapy delivered during a true ventricular arrhythmia secondary to a classification of cycles as noise.

**Noise detection**

By design, S-ICDs are more exposed to the risk of oversensing of myopotentials and of electromagnetic interference.
because the sensing electrodes are more spaced and at greater distances from the ventricular myocardium than for a transvenous ICD. Sophisticated algorithms have been developed to filter noise from the true cardiac signals. In some instances, we found oversensing of contemporaneous noise of the induced arrhythmia, leading to an absence of sensing and therefore to an absence of delivered therapy in some patients or leading to a marked sensing delay in others. An external shock or manual shock with the device had to be delivered in order to terminate the ventricular arrhythmia. One of the specificities of the S-ICD is recording of an episode only if the device has charged its capacitors, which will not be the case if the noise is sustained and prevents the arrhythmia counters from incrementing. Therefore, it is not possible to assess the true incidence of this type of problem as a result of spontaneous arrhythmia. The question is whether this type of extremely worrisome tracing is a problem only observed during the postimplant induction procedure (what we believe) or whether it can be observed during a spontaneous arrhythmia and thus indicates danger to the survival of these patients. For the 5 patients without delivered therapy, the sensing vector was always the primary vector. The noise only appeared immediately after induction and was not found on the cycles preceding the burst. The arrhythmia was induced using a 50-Hz-type burst delivered between the can and the coil. Although hypothetical, we could speculate that this 50-Hz burst induced the arrhythmia but also a muscle spasm of the diaphragm, the diaphragmatic myopotentials being superimposed on top of the ventricular arrhythmia tracing. If the problem is induced by the burst (i.e., the most likely hypothesis), the risk of occurrence during a spontaneous arrhythmia seems to be limited. This hypothesis would also explain the apparent gap between our finding and the many other reassuring reports in the literature on the S-ICD.  

Sensing delay with an S-ICD

Recognition of a malignant ventricular arrhythmia by the S-ICD system is totally original. Rather than analyzing rhythms on a beat-to-beat basis, S-ICDs are primarily designed for VF detection, for which accurate classification of individual cardiac EGMs is not critical. In the present study, there were no observed failures in defibrillation threshold testing due to undersensing. On the other hand, a prolonged delay in sensing the arrhythmia was found, with significant interindividual variability. During tachycardia and after the first T marker, the sensing profile is modified to become more sensitive. Therefore, after the first T marker, the presence of any S cycles is unexpected but was observed in a large number of patients. It has been acknowledged recently that there is a need to increase detection intervals and upgrade therapy zones in order to reduce the occurrence of both inappropriate therapies and avoidable therapies in response to benign or spontaneously terminating arrhythmias. The time to therapy intervals observed for 65-J shocks were systematically longer than all of the times measured in studies that led to the recommendation to increase the initial sensing counters. For an S-ICD, at what point is the time to therapy considered too long? The question at the time of induction also aims to determine the degree of undersensing to be attained for which a change lead positioning or a reprogramming is required. Empirically modifying the position of the lead in order to improve the quality of sensing is difficult at best. Moreover, reprogramming options aimed at optimizing the quality of the signal are limited. Therefore, what are the other options?

1. S-ICD subjects systematically undergo prescreening, an essential step in an attempt to ensure they have adequate surface EGMs before to implantation. After the implant, an automatic vector selection algorithm (based on a weighted combination of the QRS/T-wave ratio and R-wave amplitude for each vector) selects the best sense vector and gain combination. If the selected vector is associated with a “substantial” sensing delay or noise, it is possible to change the vector manually and to reinduct with the new vector, bearing in mind that this second vector was initially considered by the device as being less effective than the first in terms of ratio between sensitivity and specificity.

2. The sensing characteristics of the S-ICD are affected by the detection rate programmed for its therapy zones. Increasing the programmed detection rate reduces the sensitivity of the device. Conversely, reducing the detection rate reduces its specificity and increases the risk of inappropriate therapies related to T-wave oversensing. In the present study, the programings used varied according to the practices of the centers. However, they were relatively low so it is difficult to incriminate the zones programming to explain the prolonged detection times.

3. Two detections zones are programmable. The conditional shock zone has a longer refractory period and longer time constant for automatic adjustment of sensitivity than the shock zone. Thus, programming a conditional shock zone alters sensing on a beat-to-beat basis. For an identical cutoff zone, programming a conditional shock zone renders the device less sensitive, but it also allows for reducing the risk of oversensing of the T wave or of double-counting of the R wave. In our study, the programing of the conditional zone was relatively standard, and no programming margin seemed to be available for this parameter. There were no differences between patients of group 1 (perfect detection) and patients of group 3 (delayed detection) in terms of the number of zones or the lower limit of the conditional zone. In contrast, there was a statistically significant difference in the programmed limit of the shock zone between the 3 groups (higher limit for group 3). As explained in section “Sensing with an S-ICD”, programming a lower limit for the shock zone seems to be associated with an improved quality of sensing during an episode of VF but may be associated with an increased risk of inappropriate therapies.
4. The S-ICD remains a relatively new therapy. Future modifications of the sensing algorithm could allow for reducing this sensing delay without altering its specificity. The question also arises as to whether, in the future, additional parameters will need to be programmable (as with the possibility of using several vectors simultaneously). Similarly, the possibility of coupling the S-ICD with a leadless pacemaker (potentially providing antitachycardia pacing), with detection of the arrhythmia remaining under the control of the S-ICD. 

Consideration could thus be given to assigning sensing to the pacemaker as opposed to the S-ICD, which would allow for near-field sensing should be explored.

Value of intraoperative induction

The results of the SIMPLE study question the value of perioperative DFT in individuals undergoing transvenous ICD implantation, and many practitioners now elect to forego DFT testing. In contrast, there are no current data in S-ICD recipients regarding the safety and efficacy of not performing DFT. In addition to concerns regarding the reliability of the efficacy of defibrillation (8% failure with the first 65-J shock), delivering a shock remains the only way to measure lead impedance with the S-ICD. Finally, our study suggests that delivering a shock remains the only way to measure lead recipients regarding the safety and efficacy of not performing DFT testing.

Conclusion

Although there are numerous objective reasons for being enthusiastic about the S-ICD, this particular therapy remains relatively new, and there is still a need for accurate analysis of the various components of its functioning. Although it is recognized that the detection time is longer for an S-ICD compared to a transvenous defibrillator, the present study nonetheless reveals a certain number of patients with significant or even excessive sensing delay as well as some worrisome cases of oversensing of noise inhibiting the therapies. The present findings also argue in favor of systematically performing intraoperative DFT at the end of the implantation procedure and encourage the development of event monitoring functions.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2018.02.013.