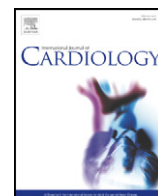




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## Effectiveness of subcutaneous implantable cardioverter-defibrillator testing in patients with hypertrophic cardiomyopathy

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### ABSTRACT

**Background:** Subcutaneous ICD (S-ICD) is a promising option for Hypertrophic Cardiomyopathy (HCM) patients at risk of Sudden Cardiac Death (SCD). However, its effectiveness in terminating ventricular arrhythmias in HCM is yet unresolved.

**Methods:** Consecutive HCM patients referred for S-ICD implantation were prospectively enrolled. Patients underwent one or two attempts of VF induction by the programmer. Successful conversion was defined as any 65 J shock that terminated VF (not requiring rescue shocks). Clinical and instrumental parameters were analyzed to study predictors of conversion failure.

**Results:** Fifty HCM patients (34 males,  $40 \pm 16$  years) with a mean BMI of  $25.2 \pm 4.4$  kg/m<sup>2</sup> were evaluated. Mean ESC SCD risk of was  $6.5 \pm 3.9\%$  and maximal LV wall thickness (LVMWT) was  $26 \pm 6$  mm. In 2/50 patients no arrhythmias were inducible, while in 7 (14%) only sustained ventricular tachycardia was induced and cardioverted. In the remaining 41 (82%) patients, 73 VF episodes were induced (1 episode in 14 and >1 in 27 patients). Of these, 4 (6%) spontaneously converted. In 68/69 (98%) the S-ICD successfully cardioverted, but failed in 1 (2%) patient, who needed rescue defibrillation. This patient was severely obese (BMI 36) and LVMWT of 25 mm. VF was re-induced and successfully converted by the 80 J reversed polarity S-ICD.

**Conclusions:** Acute DT at 65 J at the implant showed the effectiveness of S-ICD in the recognition and termination of VT/VF in all HCM patients except one. Extreme LVH did not affect the performance of the device, whereas severe obesity was likely responsible for the single 65 J failure.

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### 1. Introduction

Hypertrophic Cardiomyopathy (HCM) is the most common inherited heart muscle disorder and a leading cause of sudden cardiac death (SCD) in young adults [1,2]. Patients at high risk of SCD benefit from primary prophylaxis with an implantable cardioverter defibrillator (ICD). However, because of their young mean age at implantation, HCM

patients are more likely to suffer device-related complications and up to 4% intravascular lead related complications [3–6]. The subcutaneous ICD (S-ICD) [7] eliminates the need for lead placement in the heart and is expected to eliminate intravascular lead-related complications and lead malfunctioning at follow up [8,9].

However, compared with other arrhythmogenic conditions, HCM possesses unique features that might influence the efficacy of the device, such as increased left ventricular (LV) mass and unpredictable electrical substrate [10]. Some clinicians express concerns that the defibrillation threshold (DFT) in HCM may be higher than in other cardiomyopathies, and may increase over time in relation to LV mass

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and extent of myocardial fibrosis [11]. Furthermore, because of the subcutaneous parasternal placement of the leads, the S-ICD requires greater shock energy compared to transvenous ICDs, in order to convert potentially lethal arrhythmias [3]. This uncertainty is similar to the debate that occurred in the early transvenous ICD days, and threatens to hinder the clinical use of S-ICD in HCM patients, in the absence of convincing evidence supporting its effectiveness in this particular population. Thus, we felt it timely to assess post-implantation DFT testing in a cohort of HCM patients, in order to evaluate S-ICD effectiveness in the detection and termination of induced ventricular fibrillation (VF) and assess potential predictors of failure.

## 2. Patients and methods

### 2.1. Study population

Consecutive HCM patients referred for S-ICD implantation for both primary and secondary prevention at seven Italian Centers from June 2014 to May 2016 were prospectively enrolled. The diagnosis of HCM was based on ultrasound characteristics: a hypertrophied, non-dilated left ventricle (wall thickness of at least 15 mm) in the absence of another cardiac or systemic disease capable of producing a similar degree of hypertrophy [1]. For all patients, informed consent to participate in the study was obtained and the Ethics Committee of each Center approved the study protocol.

### 2.2. Electrocardiogram collection, 24-h Holter monitoring and screening test

Standard 12-lead ECG was performed in all patients, and the presence of atrioventricular and interventricular conduction delays, ST segment abnormalities were assessed. The Sokolow LV hypertrophy index was calculated. Moreover, 24-H Holter monitoring recordings were obtained for all patients in order to assess the presence of Non Sustained Ventricular Tachycardia (NSVT) or other ventricular arrhythmias. Patients with pacing indication due to bradyarrhythmias were excluded. All patients were eligible for S-ICD implantation according to the manufacturer screening tool and rules.

### 2.3. Echocardiography

Comprehensive two-dimensional and Doppler echocardiographic studies were performed using commercially available instruments. LV hypertrophy was assessed by two-dimensional echocardiography, and the site and extent of LV maximal wall thickness were identified. Peak instantaneous LV outflow gradient, due to mitral valve systolic anterior motion and mitral-septal contact, was estimated with continuous wave Doppler under standard conditions [1].

### 2.4. Cardiac magnetic resonance

CMR examination was performed using commercially available scanners and workstations. LV end-diastolic and end-systolic volumes, LV mass, and maximal LV wall thickness were obtained and the presence of delayed enhancement was assessed by visual inspection 15 min after intravenous administration of gadolinium-based contrast agent. LV mass indexed to BSA [12] was derived by the summation of discs method and multiplying myocardial muscle volume by 1.05 g/cm<sup>3</sup>.

Normal LV mass was defined by LV mass Index values < 81 g/m<sup>2</sup> for males and < 62 g/m<sup>2</sup> for females, mildly increased when 81–91 g/m<sup>2</sup> for males and 62–69 g/m<sup>2</sup> for females and markedly increased if > 91 g/m<sup>2</sup> for males and > 69 g/m<sup>2</sup> for females [13].

### 2.5. Sudden death prediction model

The validated ESC HCM Risk-SCD algorithm was used to estimate the risk of Sudden Cardiac Death (SCD) [1]. Based on the resulting 5-year risk estimate, patients were defined as low risk if < 4%, intermediate if ≥ 4–< 6% and high-risk if ≥ 6%.

Furthermore, a comparative characterization of the arrhythmic risk was performed using the ACC/AHA risk stratification model for HCM [14].

### 2.6. Acute defibrillation threshold testing and VF termination

DFT testing was routinely performed in all patients at the time of S-ICD implantation, following one or two attempts of VF induction, by delivering via the programmer a 50 Hz DC burst for a maximum length of 10 seconds under deep sedation or general anesthesia. Detection of VF was performed automatically by the device and successful conversion was defined as any 65 J shock that terminated VF and did not require any external rescue shocks. In case of failure of VF conversion at 65 J, after restoration of sinus rhythm with an external defibrillator, a subsequent attempt at maximal energy available was performed.

### 2.7. Statistical analysis

Continuous and normally distributed data were represented as mean ± SD; categorical data were expressed both as numbers and as percentages. The SPSS software (version 21.0, SPSS Inc., Chicago, Illinois) was used for statistical analysis.

## 3. Results

### 3.1. Demographics and clinical profile

We evaluated 50 consecutive HCM patients with a mean age of 40 ± 16 years (Table 1). Fifteen were females (30%), 22 (42%) were < 40 years and 28 (58%) were ≥ 40 years. Mean BMI was 25.2 ± 4.4 kg/m<sup>2</sup> [18.1–36], with 4 obese (BMI ≥ 30) patients. Eight patients (16%) were in NYHA class > I, mean LV ejection fraction was 62 ± 8% and 8 patients had history of Atrial Fibrillation. Seven (14%) patients had basal Left Ventricular Outflow Tract Obstruction (LVOTO) of at least 30 mm Hg and 1 patient (2%) had a prior surgical myectomy. Two patients (4%) had been resuscitated from cardiac arrest due to VF, whereas 48 (96%) were implanted for primary prevention. Only 1 patient had been previously implanted with a transvenous ICD (Table 1).

Maximal LV wall thickness (LVMWT) was 26 ± 6 mm, with 13 patients (26%) < 20 mm, 23 (46%) between 20 and 29 mm and 14 (28%) ≥ 30 mm. Moreover, in the 38 patients (76%), who had their indexed LV mass measured by CMR, only 3 patients (8%) were in the normal range, whereas 35 (91%) had a marked increase of LV mass (indexed LV mass > 91 g/m<sup>2</sup> for males and > 69 g/m<sup>2</sup> for females).

### 3.2. ICD indications

Mean calculated risk for SCD at 5 years by the ESC score was 6.5 ± 3.9%. Specifically, 26 (52%) patients were estimated to be at high risk, 8 (16%) at intermediate/high risk and 16 (32%) at low risk. However, of the 16 low SCD risk patients according to the ESC score, all but one had at least one major risk factor according to the 2013 ACC/AHA Guidelines (Fig. 1). The only patient with no SCD risk factors had been referred following a transvenous lead fracture implanted 10 years before.

### 3.3. Post-implantation defibrillation threshold testing

S-ICD implantation was uneventful in the 50 study patients. At the end of the procedure, all underwent VF induction according to the protocol. In 2 patients (4%) no arrhythmias could be induced, while in 7

**Table 1**  
Baseline characteristics of the cohort.

Variable	Total implants (n = 50)
<b>Demographic</b>	
Age (years)	40 ± 16 [15–65]
Male sex (n)	35 (70%)
BMI (kg/h <sup>2</sup> )	25.2 ± 4.4 [18.1–37]
BMI > 30 (n)	4 (8%)
BSA (m <sup>2</sup> )	1.84 ± 0.21 [1.5–2.2]
<b>Medical history</b>	
NYHA > I (n)	8 (16%)
Atrial fibrillation (n)	8 (16%)
LVOT gradient (mm Hg)	16 [5 to 64]
LVOT gradient > 30 mm Hg (n)	7 (14%)
Myectomy (n)	1 (2%)
Prior transvenous ICD (n)	1 (2%)
ICD primary prevention (n)	48 (96%)
ICD secondary prevention (n)	2 (4%)
Genotype positive (n) <sup>a</sup>	24 (51%)
Syncope (n)	6 (12%)
LGE on CMR <sup>b</sup>	24 (63%)
Abnormal BP response to exercise (n) <sup>c</sup>	4 (9%)
Fx of SCD (n)	20 (40%)
<b>Medications</b>	
Beta-blockade (n)	36 (72%)
ACE/ARB (n)	9 (18%)
Calcium channel blockers (n)	10 (20%)
Antyarrhythmics (n)	7 (14%)
<b>Electrocardiographic</b>	
Pathologic Q waves (n)	6 (12%)
Positive Sokolow index (n)	25 (50%)
ST/T alterations (n)	34 (68%)
Nonsustained VT (n)	30 (60%)
<b>Echocardiography</b>	
LA diameter (mm)	46 ± 7
LV ED volume (cc)	65 ± 22
LV EF (%)	62 ± 8
End-stage	3 (6%)
LV MWT (mm)	26 ± 6
<b>ESC HCM SCD risk</b>	
Low-intermediate ESC risk (n)	24 (48%)
High ESC risk (n)	26 (52%)
Individual risk at 5 years (%)	6.7 ± 4.1

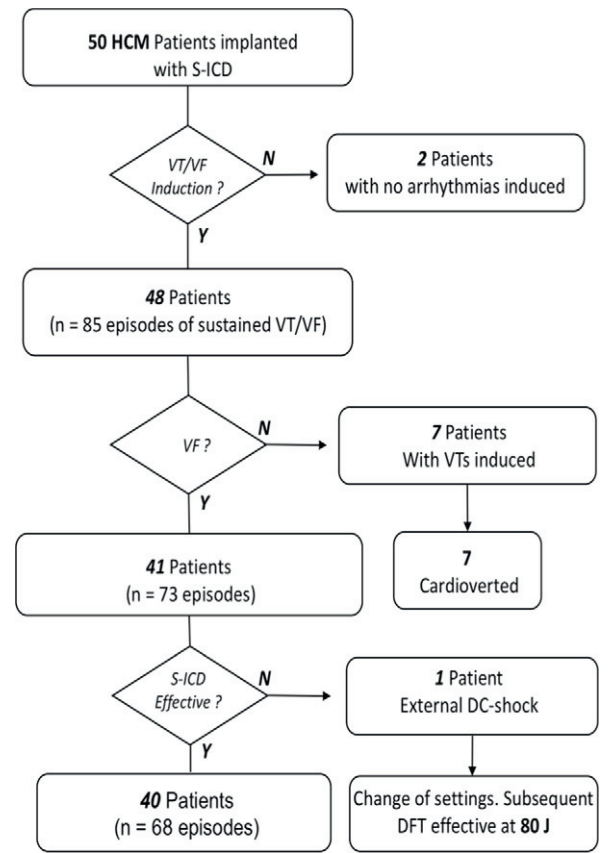
<sup>a</sup> Gene testing available for 47/50 patients.

<sup>b</sup> CMR data available for 38/50 patients.

<sup>c</sup> Abnormal BP response was available for 45 patients.

VT: Ventricular Tachycardia

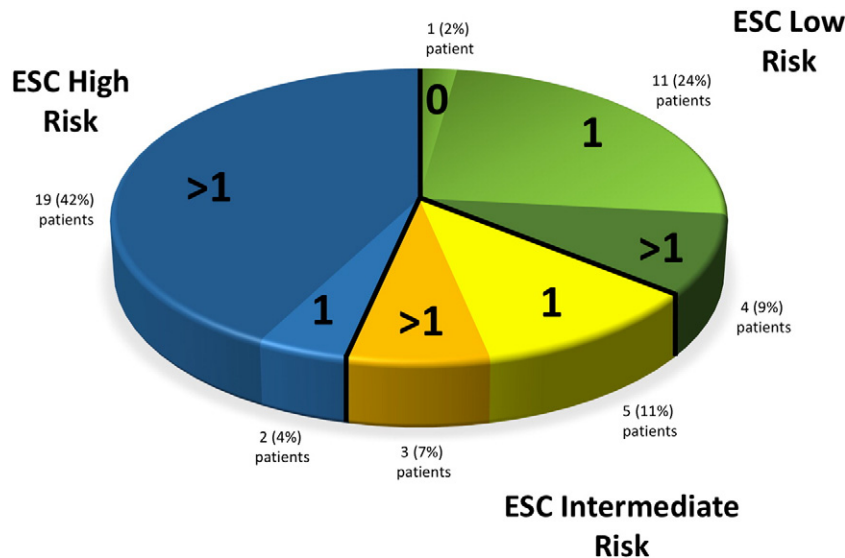
VF: Ventricular Fibrillation



**Fig. 2.** Flow chart of post-implantation DFT testing. VT: Ventricular tachycardia. VF: Ventricular fibrillation.

(14%) polymorphic sustained ventricular tachycardia (SVT) was induced and correctly identified and cardioverted by the S-ICD (Fig. 2).

In the remaining 41 (82%) patients, a total of 73 VF episodes were induced (1 episode in 14 patients and > 1 in 27 patients) and all were correctly detected by the device (Fig.2). Of the 73 VFs, 4 (6%) terminated



**Fig. 1.** Distribution of arrhythmic risk according to the ESC-SCD score. Within each ESC Risk group class, are also reported the number of patients with 0, 1 and >1 ACC/AHA 2013 guidelines.

spontaneously into sinus rhythm. The S-ICD successfully restored sinus rhythm in 68 of the 69 remaining episodes (98.5%). Of note, all patients with massive LVH (LVMWT  $\geq 30$  mm) and extreme LV mass (LV mass  $\geq 120$  g/m<sup>2</sup>) were successfully cardioverted with an induction to S-ICD shock time  $< 21$  s in 93% of patients (mean  $17 \pm 2$  s [12–27]). The only Defibrillation Testing failure at 65 J (2%) occurred in a severely obese patient (BMI of 36 kg/m<sup>2</sup>; BSA 2.3 m<sup>2</sup>) implanted in primary prevention, who required an external rescue defibrillation despite device and lead optimal anatomic positioning (Fig. 3). Patient's LVMWT was 25 mm at the level of the anterior septum and he had mild LVOT obstruction at rest (peak LVOTO 30 mm Hg). VF was induced for a second time and successfully converted by the 80 J reversed polarity S-ICD.

#### 4. Discussion

The entirely subcutaneous ICD represents an appealing alternative to a transvenous device in HCM patients, who are often young, do not require pacing, and face considerable device-related complications over their lifetime, including those related to lead substitutions and infections [3–5]. However, a perception persists among some clinicians that the unique features of HCM, such as extreme increase in LV wall thickness and mass and unpredictable electrical substrate, may affect the effectiveness of the S-ICD in the conversion of lethal arrhythmias. However, the present study shows that these concerns are largely unjustified. We observed a 98.5% effectiveness of the S-ICD in the recognition and termination of induced VF at the time of implant in an HCM cohort largely represented by moderate- to high-risk patients. These results were consistent across the diverse phenotypic spectrum of HCM, including an important subset with extreme LVH (i.e. 28% of patients with LVMWT  $\geq 30$  mm and 91% with a marked increase of LV mass). In such extreme cases, the S-ICD successfully converted at first attempt all the induced VF. Overall, these data are in line with a pooled sub-analysis of EFFORTLESS and IDE registry [15], showing a 95.5% effectiveness in post-implant cardioversion of VT/VF at  $\leq 65$  J in 99 consecutive

HCM patients. Likewise, in a recent series of 23 high-risk HCM patients [16] there was a 100% efficacy in VF termination at 65 J by the S-ICD. With respect to the EFFORTLESS/IDE cohort [15], our patients showed a more severe phenotype with a greater maximal LV thickness ( $26 \pm 6$  mm vs  $21 \pm 6$  mm), more frequent NSVTs (60% vs 34%) and higher individual ESC-arrhythmic risk score ( $6.7 \pm 4\%$  vs  $5 \pm 3.5$ ). Moreover,  $>75\%$  of our patients were studied with a CMR, whereas such exam was not included in the EFFORTLESS/IDE protocol.

Of note, the only defibrillation failure at 65 J in our cohort appeared largely attributable to severe obesity and its consequences on defibrillation threshold, rather than reflect an adverse profile of the HCM. Obesity has previously been associated with conversion failure by the S-ICD in two independent series [15,16]. In the IDE registry, the failure rate of VF conversion at 65 J was 17% in obese patients compared to only 5% in normal weight individuals [17]. In the small series reported by Weinstock and colleagues, higher BMI were associated with defibrillation failure at the lower DFT of 50 J [16]. It is reasonable to postulate that a greater amount of body fat between the can and the parasternal lead coil might be responsible for dumping of energy delivery to the myocardium. However, a practical solution to such problem can be offered by a 'deeper' positioning of the parasternal coil in obese patients, since the nearer to the heart the pulse generator and coil are placed, the more the shock vector efficacy may be improved. Furthermore, in obese patients the implanting physician needs to carefully ensure the visualization of the fascia overlying the ribs, to tunnel and place the parasternal coil over the rib and the muscular fascia and not through the subcutaneous fat [18].

This hypothesis bears direct relevance to clinical practice, given the growing epidemics of obesity in the western population, and should be validated further.

Our data emphasize the importance of DFT testing in S-ICD implants, with specific regard to HCM, in order to overcome potential issues related to lead and device positioning and optimize sensing and shock delivery. Routine patients as well as high-risk subgroups, such as

IVS= interventricular septum

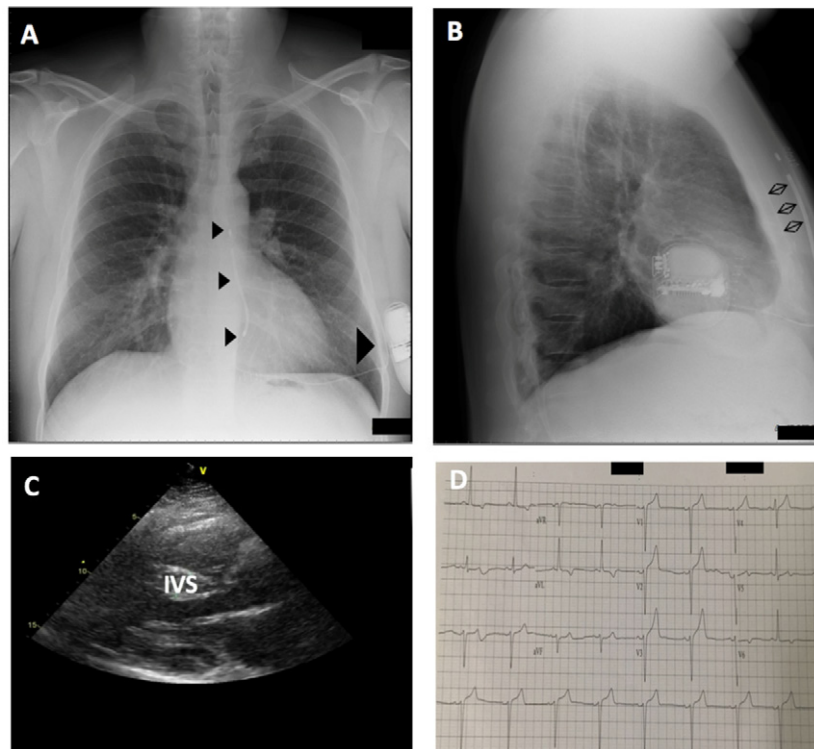


Fig. 3. Antero-Posterior Chest X-ray showing device positioning (A); Lateral chest X-ray showing superficial anterior Lead positioning on sub-sternal fat (B); echocardiographic parasternal long axis (C) and basal electrocardiogram (D) of the patient with failed VF conversion at 65 J. IVS = interventricular septum.



obese one, require greatest attention, given the lack of evidence to support a 'non-DFT policy'.

At present, the S-ICD limits two important classes of complications in HCM patients: the morbidity related to transvenous lead malfunctions (recurrent lead replacement/extraction, venous thrombosis and obstruction) and endocarditis [15,16]. Such advantages, together with the high acute induced VF conversion effectiveness in CM, cannot be ignored and should be considered in device selection for young HCM patients at risk for SCD.

The development of new technologies, such as leadless pacemakers compatible with the S-ICD system [19], together with a better management of inappropriate shocks [20], opens promising perspectives for SCD management, thereby widening the subset of patients who might benefit from SCD prophylaxis.

## 5. Conclusions

Acute defibrillation testing at 65 J at the implant showed the effectiveness of S-ICD in the recognition and termination of VT/VF in all HCM patients except one. Extreme LVH did not affect the performance of the device, whereas severe obesity was likely responsible for the single 65 J direct polarity failure observed in our series. Actually HCM patients, who do not require pacing, or those who are at higher risk for transvenous ICD related complications, may benefit from long-term protection guaranteed by a subcutaneous device (avoiding morbidity associated with endocavitary devices).

## Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

## Acknowledgments

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