Device interaction between cardiac contractility modulation (CCM) and subcutaneous defibrillator (S-ICD)

Luca Trolese MD | Thomas Faber MD | Alexander Gressler MD | Johannes Steinfurt MD | Judith Stuplich MD | Eike Jordan MD | Christoph Bode MD | Manfred Zehender MD | Ingo Hilgendorf MD

Abstract
Combined implantation of cardiac contractility modulation (CCM) with subcutaneous implantable cardioverter-defibrillator (S-ICD) appears a suitable option to reduce the amount of intracardiac leads and complications for patients. Here we report on a patient with ischemic cardiomyopathy carrying an S-ICD in which a CCM device was implanted. During crosstalk testing post-CCM implantation, the S-ICD misannotated QRS complexes and T waves. The problem was solved through reprogramming the CCM, while preserving S-ICD functionality and improving heart failure symptoms. In conclusion, S-ICD combined with CCM seems to be a good and safe option for patients when device interference is being ruled out.

KEYWORDS
CCM, crosstalk test, heart failure, interferences, noise, S-ICD, T wave oversensing

1 | INTRODUCTION
Cardiac contractility modulation (CCM) is a device-based treatment option for patients with chronic heart failure with reduced ejection fraction (HFrEF) improving exercise capacity and reducing the rates of cardiovascular deaths and hospitalization for heart failure.1,2 The device is implanted similar to a pacemaker, with two bipolar leads are being positioned at the right ventricular septum (local sense [LS] and right ventricle [RV]) delivering nonexcitatory high voltage biphasic electrical impulses during the absolute refractory period.

Since many patients receiving CCM present with a left ventricular ejection fraction (LVEF) ≤ 35%, concomitant and prophylactic placement of an implantable cardioverter-defibrillator (ICD) needs to be considered according to guideline recommendations.3 However, currently no device combines CCM and ICD capabilities in one device, requiring the transvenous implantation of at least two leads into the heart.

Combining transvenous implantation of CCM with the subcutaneous placement of an ICD (S-ICD) appears as a suitable option for those patients who do not require pacemaker stimulation, and which may benefit from a reduction in implanted lead-related complications. In the following, we report a case of unexpected electrical interference when combining CCM and S-ICD placement in one patient.

2 | CASE REPORT
A 59-year-old male patient with ischemic cardiomyopathy, LVEF of 20%, narrow QRS complex (113 ms), and oligosymptomatic heart failure (NYHA class I-II) received transvenous ICD implantation in 2014 for primary prevention. In 2017, the device and leads were...
explanted in the context of staphylococcus aureus endocarditis, and subsequently, an S-ICD device (Boston Scientific; A209, Electrode 3401 SQ-1) was implanted (Figure 1).

As heart failure progressed to NYHA class II-III despite optimal medical treatment and revascularization, a CCM (Impulse Dynamics; Model: Optimizer smart) was implanted in 2018 (Figure 2).

Post CCM implantation and activation, false annotations were registered by the S-ICD for multiple vector configurations tested. The QRS complex was misannotated with N (noise) and the T wave with S (sensed) (Figure 3).

When the CCM was reprogrammed applying either single LS or single RV maximal output stimulation (7.5 V), or different delays between LS and RV stimulation, intermittent misannotations were still recorded via the S-ICD. A progressive reduction of QRS notching was observed when CCM pacing voltage was lowered step by step. Selective LS or RV stimulation did not alter QRS notching in the intracardiac electrogram (IEGM) effectively (at the same voltage level). Finally, following the stepwise reduction (steps of 0.5 V) in voltage output to 4.5 V with simultaneous RV and LS stimulation, all QRS complexes and T waves were annotated correctly. The CCM voltage reduction and stimulation protocol combined with S-ICD vector programming is summarized in Table S1.

In the IEGM, notching of the QRS complex was present with high-voltage CCM stimulation and vanished when CCM voltage output was lowered to 4.5 V, indicating cessation of device interference (Figure 2). The delivery time of CCM therapy was increased empirically to 10 h (from 8 h default setting). Finally, in the S-ICD, the alternative vector was programmed and a dual shocking zone at 220/bpm (conditional zone) and 250/bpm (shock zone) was set. No defibrillation threshold (DFT) test was performed again. With the above-mentioned CCM settings, no misannotations were registered by the S-ICD during device crosstalk testing, and during the ergometric test on the day post-CCM implantation.

Moreover, the patient did not experience misannotation or inadequate S-ICD shocks within the first 6 months follow-up. Following CCM placement, and despite low voltage output, heart failure symptoms improved according to the patient (NYHA class I-II) at the 6 months follow-up.
DISCUSSION

To our knowledge, we report the first documentation of noise (N = QRS) and T wave oversensing (S) of an S‐ICD combined with a CCM. Inadequate noise annotation by the S‐ICD, when combined with CCM in the same patient, is a rare and usually noncritical malfunction.4 When noise occurs, the respective annotation (N) should not limit tachycardia detection of the S‐ICD, but there is no experience when noise annotation coincides with T wave oversensing. By modifying CCM programming (see description above) the problem was resolved and the therapy was clinically effective, nevertheless. Probably, the reduced CCM output, although excitatory, resulted in a less deformed QRS complex morphology (Figure 3) which was then adequately recognized by the S‐ICD. In our patients, reduction of pacing amplitude does not influence the clinical outcome of CCM. The lowest threshold of CCM voltage output sufficient to impose a therapeutic effect has not been tested systematically in trials. However, we and others have seen that CCM voltage amplitude can be reduced, occasionally, to prevent palpitations due to high output pacing without jeopardizing the clinical benefit. In these cases, we empirically increase the duration of daily delivered (from 8 to 10 h) CCM therapy to compensate for the lower amplitude, in accordance with the manufacturer’s recommendation. We acknowledge that Klopee et al.5 described CCM therapy to be similarly effective over a range of shorter (5 h) to longer (12 h) daily periods of stimulation, but limited clinical data exists.

In our patient, DFT was tested after the initial S‐ICD placement in 2017. Following the voltage adaptation of CCM pacing that eliminated the interference with S‐ICD sensing during crosstalk testing, we did not repeat DFT testing again. The CCM device contains a built‐in algorithm which inhibits CCM stimulation in case the irregular electrical activity is being detected, including ventricular extrasystoles and ventricular tachycardia. Roeger et al.1 reported that during S‐ICD testing, CCM signal delivery stopped immediately during (VT)/VF detection and the S‐ICD recognized the arrhythmia adequately in all 20 patients. For this reason, we regarded DFT testing not necessary, but we acknowledge that other operators may prefer to repeat the test. The manufacturer does not offer an official recommendation for such rare complications.

In conclusion, S‐ICD combined with CCM appears as an effective and safe option for patients suffering from chronic HFrEF. Crosstalk testing to assure adequate S‐ICD detection is of high importance when CCM and S‐ICD are being combined.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

ORCID

Luca Trolese http://orcid.org/0000-0001-7067-8261

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