Indian Pacing and Electrophysiology Journal 17 (2017) 44–47

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

Low-energy cardioversion of ventricular tachycardia: When less is more

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ARTICLE INFO

Article history:
Received 12 February 2017
Accepted 13 February 2017
Available online 20 February 2017

Keywords:
Ventricular tachycardia
Implantable cardioverter-defibrillator
Pro-arrhythmia
Shock

ABSTRACT

Herein we report the case of a patient who was admitted in ventricular tachycardia after having received multiple ineffective (and sometimes pro-arrhythmic) high-energy internal shocks from his ICD and who was finally successfully treated by a commanded low-energy internal cardioversion of 0.6 J. This article revisits the use of low-energy shocks and discusses their electrophysiogical mechanisms and clinical implications.

1. Case description

A 70-year-old man with ischemic heart disease followed-up at another centre and equipped with an implantable cardioverter-defibrillator (ICD) for sustained ventricular tachycardia (VT), was admitted after having experienced multiple shocks.

Upon arrival, the patient was in VT at 170bpm, but clinically stable. Extracts from ICD interrogation are shown in Fig. 1, and the device parameters are shown in Table 1. Device interrogation revealed that in total, the patient had experienced three separate VT episodes with ineffective ATP and multiple shocks that were either appropriate but ineffective (2 × 31J and 1 × 41J), appropriate and effective (1 × 31J and 4 × 41J), inappropriate and delivered during sinus tachycardia without any effect (1 × 31J and 1 × 41J), or with induction of VT (1 × 31J and 2 × 41J). The inappropriate shocks all followed effective shocks which converted the VTs to sinus tachycardia falling in the VT-1 zone. These shocks were delivered because the rhythm discrimination algorithm does not use morphology analysis after shocks (as the EGM may be modified). The patient was admitted in VT due to the maximum number of shocks (6) having been delivered for the event, with the last shock having been pro-arrhythmic (induction of the VT observed at admission).

Options were 1) to administer amiodarone (which may have been ineffective or resulted in delayed cardioversion), 2) external cardioversion (requiring deep sedation), or 3) commanded therapy via the ICD. As all VT episodes had been refractory to ATP and sometimes to high-energy shocks, we opted to deliver a low-energy internal cardioversion at 0.6 J after administering Fentanyl 100 mcg iv. This successfully terminated the VT (Fig. 2) and was reported by the patient to be considerably less painful than the preceding shocks. The patient was thereafter stable, without recurrence of arrhythmias. The patient was discharged after 5 days under amiodarone and admitted one month later for radiofrequency ablation of VT. Programmed ventricular stimulation induced multiple VTs, localized at the inferior and apical walls of the left ventricle. Electro-anatomic CARTO (Biosense Webster, Diamond Bar, CA) mapping revealed a large scar in the inferior and apical left ventricle. Substrate modification targeting abnormal local ventricular activity and late potentials in this territory was performed. At the end of the procedure, VT could only be triggered using an aggressive induction protocol. The patient did not have any recurrence of VT during one year of follow-up under amiodarone.

2. Discussion

To the best of our knowledge, this is the first report of a very low-energy (0.6 J) ICD shock successfully terminating VT after failed ATP and multiple high-energy (31–41J) shocks. Failure of high-energy shocks to cardiovert VT is however not exceptional. In a cohort of 2000 patients from the ALTITUDE remote monitoring

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Peer review under responsibility of Indian Heart Rhythm Society.

http://dx.doi.org/10.1016/j.ipej.2017.02.002

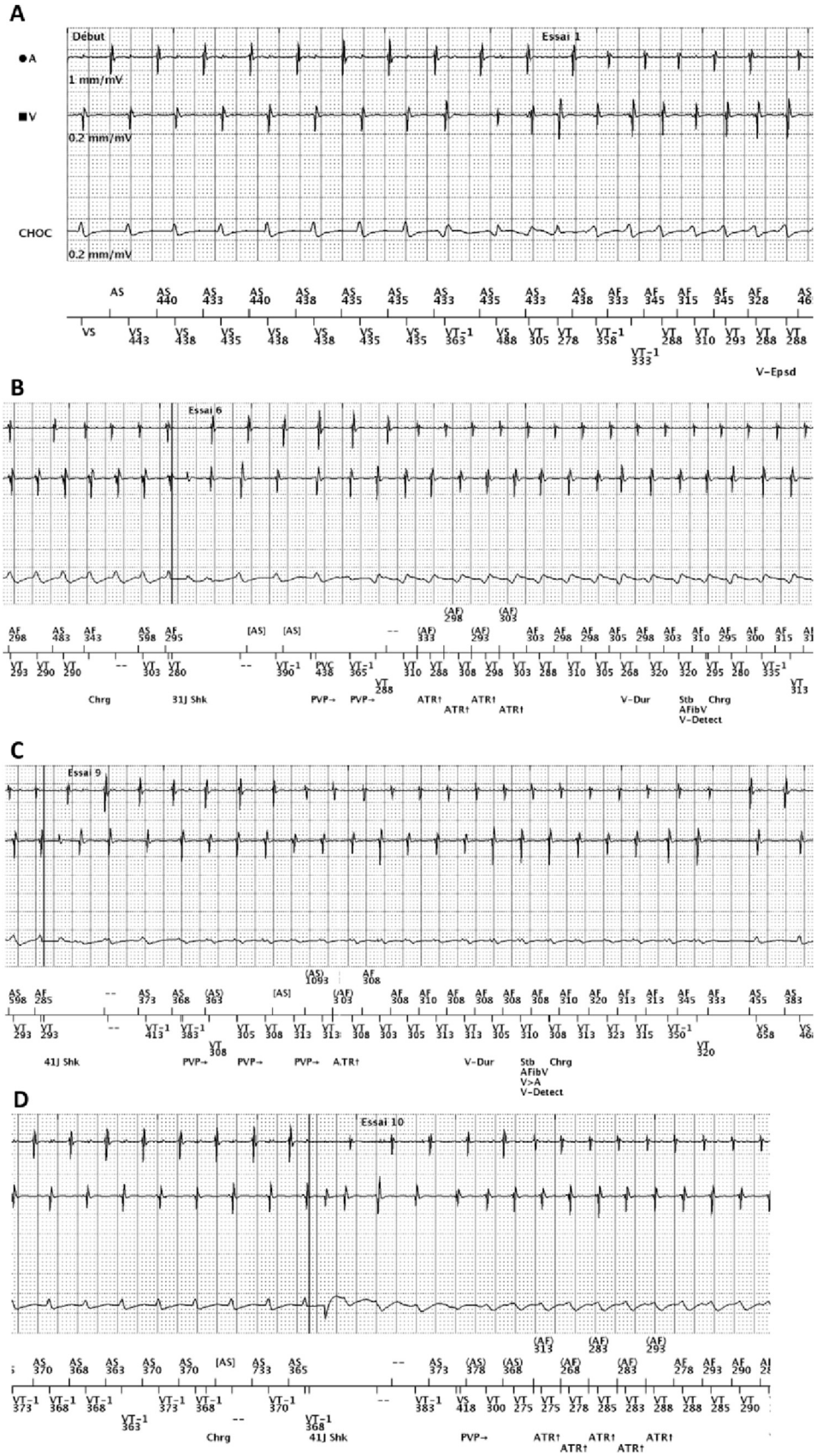

Fig. 1. Samples of the episode retrieved by ICD interrogation at admission showing **A.** Onset of VT. Note the change in QRS morphology in the choc electrogram (EGM) and V > A **B.** After 8 failed ATP attempts (5 bursts and 3 ramps), an appropriate but ineffective 31 J cardioversion is delivered which changes the VT morphology and cycle length **C.** Delivery of a 41J synchronized shock resulting in non-sustained VT and return to sinus rhythm (last 2 cycles) **D.** Inappropriate shock during sinus tachycardia (falling in the VT-1 zone), initiating a new VT.

Table 1
Device parameters (Boston Scientific TELIGEN and MarlboroughMA).

Parameter	Setting
Bradycardia mode (50–130 bpm)	DDD (AAI with VVI backup)
VT-1 Zone (140–185 bpm)	5 s duration Discriminator: Rhythm ID ATP: 5 bursts, 3 ramps Shocks: 2 × 31J + 4 × 41J
VT Zone (185–240 bpm)	2.5 s duration Discriminator: Rhythm ID ATP: 3 bursts Shocks: 31J + 5 × 41J
VF Zone (>240 bpm)	1 s duration Quick Convert ATP Shocks: 8 × 41J

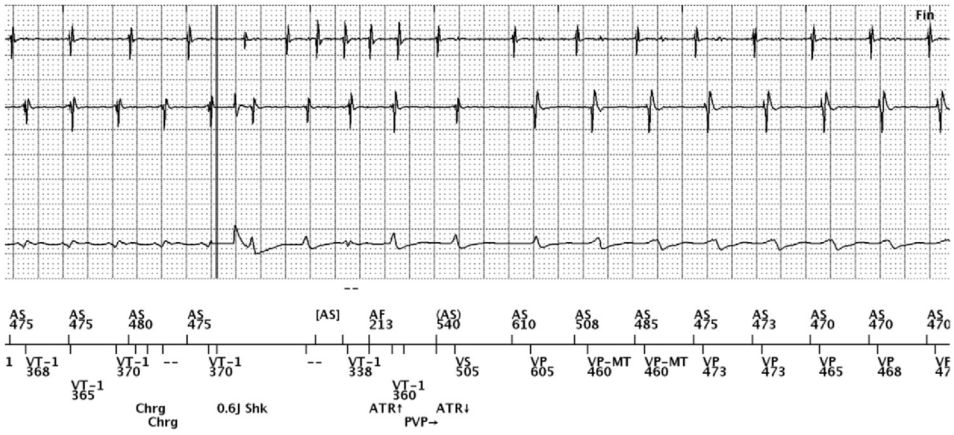

Fig. 2. Intracardiac electrogram showing successful termination of ventricular tachycardia after a commanded internal electrical shock of 0.6 J during the emergency room visit.

registry, 362 patients had 811 monomorphic VT episodes with unsuccessful ATP. The first shock (mean 23J) failed in 15.7% of cases, and required ≥ 3 shocks (mean 34J) in 7.5% of cases [1].

The physiopathology explaining our findings is speculative. Experiments on post-shock arrhythmogenesis and vulnerability have centered around re-initiation of ventricular fibrillation after defibrillation shocks, leading to the concept of the upper limit of vulnerability [2]. Animal models have shown that virtual electrodes (formed by dissipation of post-shock polarization) and break-excitation wavefronts in border zones of scar regions may re-induce arrhythmias [3,4]. Disruption of the Purkinje system may also play a role in initiating and facilitating re-entry of post-shock arrhythmias, as suggested by computer modeling [5]. Low-energy shocks may affect these mechanisms differently compared to high-energy shocks (e.g. by less extensive depolarization of myocardial tissue resulting in a smaller virtual electrode effect, or by less impact on the Purkinje system).

Low-energy shocks for treating VT have been studied as early as 1982 [6] and evaluated as an alternative to high-energy shocks for ICD therapy in several historical studies in the 1990's. Bardy et al. [7] performed a cross-over study, randomizing ramp ATP and synchronized low-energy cardioversion (titrated to 0.2–2 J) in 24 epicardial ICD patients (16 with coronary artery disease) to treat induced monomorphic VT. Low-energy cardioversion was successful in 75% of the patients (with a threshold of only 0.46 ± 0.39 J), without any difference in terms of efficacy or acceleration to VT/VF (observed in 21% of patients) compared to ramp ATP. Half of the cases with successful low-energy cardioversion had some polymorphic repetitive ventricular responses before restoration of SR (otherwise known as type II break, as was the case in our patient) which was also observed in two-thirds of cases with effective ATP. No patient factors were found to be predictive of low-energy shock efficacy. Lauer et al. [8] studied 40 ICD patients with prior MI with a step-down protocol of low-energy shocks (0.1–4J) for induced monomorphic VT. Successful VT cardioversion with <1 J was observed in 50% of patients, but 35% of patients developed VF during low-energy shocks (only 1 of these 14 cases had VF induced with an energy of <1 J). More recently, Sivagangabalan et al. [9] reported a cohort of 602 ICD patients programmed with VT zones between 150 and 300 bpm with ATP + initial low-energy (5 J) shocks followed by high-energy shocks. During follow-up, 142 patients had ventricular arrhythmias, of which slow VT (150–180 bpm) was treated successfully by low-energy shocks after 3 failed ATP attempts in 44/60 (73%) episodes, and fast VT (180–240 bpm) in 21/39 (54%) episodes. Acceleration of ventricular arrhythmia secondary to the low energy shock was noted in 8% and 39% of slow and fast VT episodes respectively.

Our case also illustrates the pro-arrhythmic effect of shocks delivered during sinus tachycardia despite synchronization with the R-wave. It also highlights the potential hazard of programming low VT zones, because therapy will continue to be delivered after effective shocks which convert the arrhythmia to sinus tachycardia falling in a VT zone, as discrimination algorithms are less stringent after shock delivery.

Potential benefits of low-energy over high-energy shocks include reduced charge time, less battery drain, and avoidance of adverse hemodynamic effects [10]. Pain, caused by skeletal muscle contraction, is unlikely to be reduced in most patients by low-energy shocks (patients were unable to distinguish pain levels between 0.4 J and 2 J shocks for device-based internal atrial

defibrillation [11]), although individual nociception is variable and some individuals may benefit from very low (<1 J) outputs. However, a failed initial shock may lead to increased pain perception of subsequent shocks [11]. It should also be borne in mind that the number of shocks for a given episode is limited (usually to a maximum of 5–8 shocks depending on the VT zone and the manufacturer). Programming an initial low-energy shock therefore reduces the total number of available high-energy shocks. The minimum programmable shock energy varies from 0.1 J (e.g. Boston Scientific) to 2 J (Biotronik, Berlin). Current ICD programming guidelines [12] do not make any recommendation regarding the initial shock energy in VT zones, although it is recommended that maximum energy be programmed in the VF zone to increase the likelihood of first shock termination of ventricular arrhythmia (and also of supra-ventricular arrhythmias in case of inappropriate shocks).

For the above-mentioned reasons, we do not recommend routine programming of an initial low-energy (<1J) shock for VT. However, in case of VT resistant to ATP and to at least one high-energy shock, this option should be borne in mind.

Conflict of interests

There are no conflicts of interest to report.

Funding support

This report did not receive any external funding.

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