Rate smoothing induced ventricular arrhythmia and syncope: a new device-induced proarrhythmia

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A 74-year-old male presented to our emergency room with history of sudden onset palpitations associated with syncope. He had a single-chamber implantable cardioverter defibrillator implanted for secondary prevention of sudden cardiac death due to ischaemic cardiomyopathy. Interrogation of the device revealed episodes of non-sustained ventricular tachycardia (NSVT) at 220 ms. Post-tachycardia, another episode of NSVT with longer duration, was induced by rate smoothing pacing algorithm following premature ventricular beats. We describe this unique form of device-related proarrhythmia causing syncope.

Case report

A 74-year-old male presented to emergency room due to sudden syncope during the day. His past medical history was significant for hypertension, diabetes mellitus, hypothyroidism, coronary artery disease, and ischaemic cardiomyopathy (mid-apical anterior and basal inferolateral akinesia, ejection fraction 30%). His current medications included aspirin, metoprolol, hydrochlorothiazide, levothyroxine, lisinopril, and simvastatin. His coronary artery disease was not amenable to revascularization. He received a single-chamber implantable cardioverter defibrillator (ICD) (Vitality DS, Model T135, Boston Scientific, Natick, MA, USA) after an episode of syncope 5 years prior to this current presentation. His vitals were stable and physical exam was within normal limits except for laceration on his scalp. Electrocardiogram suggested normal sinus rhythm, first-degree atrio-ventricular (AV) block, old anterior and inferior myocardial infarction, a premature ventricular beat (PVC), and pacing secondary to Rate smoothing (RS). His electrolytes were within normal limits and myocardial infarction was ruled out. On further questioning, he remembered having palpitations that stopped and then started again before he passed out. A repeat echocardiogram did not reveal any new wall motion abnormalities.

His ICD was set up for two-zone detection (VT Zone 150–210 bpm, therapy with Burst ATP followed by cardioversion shocks, and VF Zone 210 bpm, therapy with defibrillation shocks) with bradycardia pacing at 40 bpm. He had stable sensing and pacing thresholds. Lead impedances were stable and were within acceptable limits. Device interrogation suggested the battery at middle of life (2–2.6 V) with acceptable charging time. Rate smoothing algorithm was turned on as a routine feature to prevent ventricular arrhythmias (Figure 1A). Intracardiac electrograms at the time of syncope suggested few PVCs that would trigger RS. Rate smoothing-related bradypacing induces sustained monomorphic ventricular tachycardia (VT) (CL 220 ms) leading to syncope (Figure 1B). Tachytherapies were not delivered as VT terminated before charging of the device has ended.

Coronary artery disease status was re-evaluated and the patient was deemed not to be a candidate for revascularization due to severe diffuse small vessel disease. He was started on amiodarone, beta-blocker dosage was increased, and an outpatient non-invasive programmed simulation was performed with successful termination of ventricular fibrillation (VF). An adequate acceptable safety margin...
was ensured as chronic amiodarone use can elevate defibrillation thresholds. The device was reprogrammed for aggressive ATP with cardioversion shocks between 186 and 200 bpm and as a defibrillator > 200 bpm. The rate smoothing algorithm was turned off and the patient remains asymptomatic to date.

Although RS reduced the duration of pauses in our patient, we believe that RS-related ectopy caused more pauses requiring further pacing intervention. The patient myocardium is in an exceedingly susceptible state for ventricular arrhythmias. When RS was turned off, no further ectopy and pauses were noted on device Holter monitor.

Discussion
Implantable cardioverter defibrillators remain an effective therapy for primary and secondary prevention of SCD. The benefit of mortality reduction has been proven in multiple primary and secondary prevention trials. However, ICDs also have the capacity to provoke or worsen arrhythmias. Frequently, this proarrhythmia is a consequence of inadequate ICD programming, rather than intrinsic limitation of ICDs.  

Implantable cardioverter defibrillator proarrhythmia can be classified into five categories as presented in Table 1. This proarrhythmia can cause significant morbidity and mortality to the patient, especially if the ICD was implanted for primary prevention.

Various algorithms have been published to prevent unnecessary shocks in patients with ICDs. Rate smoothing is one particular algorithm that controls pulse generator’s atrial and/or ventricular rate response to sudden changes in sensing or pacing intervals. The algorithm works on the premise that long pauses precede premature beats that frequently initiate polymorphic VT. Rate smoothing algorithm is frequently turned on to prevent ventricular arrhythmias. Prevention of pauses by RS has been shown to be highly effective in reducing shocks in patients with long QT syndromes.

The role of RS was prospectively evaluated in the prevention of ventricular arrhythmias in two randomized clinical trials (PROSPECT and VAST). The results were discordant about the effectiveness of RS for the prevention of ventricular arrhythmias. Most patients in the VAST trial did not have a short-long-short (SLS) sequence before the initiation of tachycardia. Rate smoothing algorithm did not prevent SLS sequences in patients with ventricular arrhythmias. In very few patients, RS prevented SLS preceding ventricular arrhythmias; however, it was proarrhythmic in some patients.

Detailed analysis of electrograms of patients in the VAST trial suggested that RS decreased the incidence of VT preceded by SLS; however, it did not change the incidence of VT. The authors have reasoned that pauses before VT can merely be an epiphenomenon or RS could be proarrhythmic in those patients. Importantly, the investigators have noticed similar RR sequences before VT in patients with multiple episodes.

Rate smoothing has been shown to delay or impair VT detection in a patient with ICDs due to intradevice interaction. Underdetection of VT most likely occurs in patients with long AV delays and aggressive RS (smaller per cent change). On the basis of the current evidence, we program RS only in patients with long QT syndromes or documented VF/VT therapy after an SLS sequence.

Conflict of interest: none declared.

References