Heart Failure-related Hospitalization after the Implementation of a Ventricular Rate Modulation Algorithm

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ABSTRACT. Rate-modulated pacing algorithms have been effective to prevent bradycardia-dependent ventricular tachyarrhythmias. Implantable cardioverter-defibrillator (ICD) storage capabilities allow identification of pause-dependent initiation of ventricular tachycardias (VTs). A 64-year-old man with a severe ischemic cardiomyopathy and a single-chamber ICD was hospitalized with congestive heart failure symptoms and palpitation. Eight days prior to his hospitalization, a run of polymorphic, pause-dependent, non-sustained VT (NSVT) was observed in the device memory. The ventricular rate stabilization algorithm was turned on. The electrocardiogram showed sinus rhythm, ventricular ectopy, and right ventricular apical (RVA) paced beats. The device interrogation indicated an increasing amount of ventricular pacing that went from 0.2% to 23.8%, and frequent episodes of pacing-facilitated NSVT. The VRS algorithm was turned off and the incidence of NSVT decreased abruptly. The patient improved rapidly and was discharged on conventional heart failure therapy. A few weeks later, heart failure symptoms were resolved. In conclusion, the activation of a rate modulation algorithm may lead to heart failure due to increasing amounts of RVA pacing and ventricular tachyarrhythmias. The use of device remote monitoring may be helpful after the activation of rate modulation algorithms to follow up on the percentage of ventricular pacing and burden of ventricular arrhythmias.

KEYWORDS. heart failure, implantable defibrillator, ventricular pacing, ventricular tachycardia.

Introduction
Congestive heart failure is the leading cause of hospitalization in patients over 65 years of age, and represents a significant economic burden to our health-care system. Dedicated proprietary pacing algorithms have been effective to shorten post-extrasystolic pauses and prevent bradycardia-dependent ventricular tachyarrhythmias in patients with torsade de pointes.1,2 Implantable defibrillators’ storage capabilities allow identification of initiation mechanisms of ventricular tachyarrhythmias, and, occasionally, polymorphic ventricular tachycardia (VT) episodes are initiated in a pause-dependent manner.3 Ventricular rate stabilization (VRS) is a Medtronic (St. Paul, MN) algorithm intended to prevent long pauses after premature atrial or ventricular contractions, previously described elsewhere.4 Right ventricular apical (RVA) pacing alters left ventricular electrical activation, causing QRS duration prolongation due to slow conduction and intraventricular dysynchrony. It may have a deleterious effect in the left ventricular systolic function, and cause congestive heart failure symptoms.5-7

We present a case to report unintended consequences of the activation of a rate modulation algorithm in a patient with an implantable cardioverter-defibrillator (ICD) leading to a heart failure-related hospitalization (HFH).

Case presentation
A 64-year-old man with a history of severe ischemic cardiomyopathy, prior coronary artery bypass graft surgery, and a single chamber ICD insertion for primary
prevention of arrhythmic death presented to the emergency room with increasing dyspnea on minimal exertion, paroxysmal nocturnal dyspnea, and short-lived palpitation. The patient had previously been in stable, class I–II, chronic systolic congestive heart failure symptoms. During an office visit, days prior to his hospitalization, a

Figure 1: The interval versus time graph diagram shows a pronounced short–long–short cycle length preceding the initiation of a self-limited ventricular tachycardia.

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Figure 2: Cardiac Compass Trends by Medtronic demonstrates an abrupt increase in ventricular episodes and percentage of ventricular pacing in coincidence with the activation of the VRS algorithm (arrow). VRS: ventricular rate stabilization.

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run of a rapid, polymorphic, non-sustained VT, preceded by a short-long-short cycle was observed in the device memory (Figure 1). The Medtronic EnTrust single-chamber ICD programming at that time included a pacing mode VVI at 40 bpm, a VT detection interval equivalent to 182 bpm (24 intervals), and a ventricular fibrillation detection rate of 222 bpm (18/24 intervals). Concerns regarding pause-dependent ventricular tachyarrhythmias were raised and the VRS algorithm was turned on, with a maximum rate of 120 bpm and an interval increment of 150 ms. Eight days later the patient presented to the emergency room with the above-mentioned symptoms. The electrocardiogram showed sinus rhythm, frequent ventricular ectopic beats, and RVA paced beats. The chest X-ray showed cardiomegaly, central pulmonary vascular congestion, and interstitial edema. Laboratory examination revealed a proBNP level of 3,248. The device interrogation indicated an increasing amount of RV pacing that went from 0.2% to 23.8%, and a significant increase in episodes of non-sustained VT (NSVT) prior to hospitalization (Figure 2). Multiple, frequent runs of NSVT were seen in the telemetry monitoring. A sequence of short-long-short cycles, including ventricular pacing beats, was commonly seen preceding the initiation of NSVT, suggesting pacing-facilitated ventricular arrhythmias (Figure 3). The patient’s symptoms of acute on chronic systolic heart failure responded to diuretic therapy. The VRS algorithm was then turned off and the incidence and frequency of NSVT decreased rapidly. The patient improved and was discharged on conventional heart failure therapy with the addition of furosemide and long-acting nitrates to his medication regimen. A few weeks later, heart failure symptoms were resolved, nitrates were discontinued, and the diuretic dose reduced. The proprietary Medtronic Cardiac Compass Trends showed no further episodes of non-sustained VT, the return to a negligible amount of RVA pacing, and normalization in heart rate variability (Figure 2).

**Discussion**

In the presented case, the activation of a rate modulation algorithm increased significantly the amount of RV apical pacing and the presence of spontaneous, and pacing-facilitated non-sustained ventricular arrhythmias, leading to a HFH in a short time period. RVA pacing is a well-defined promoter of heart failure due to intraventricular dyssynchrony and AV desynchronization. These effects are more pronounced in patients with pre-existent LV dysfunction, prior heart failure history or myocardial infarction, and increased QRS duration. Pause-suppression pacing algorithms in patients with bradycardia-induced polymorphic ventricular tachyarrhythmias appear to be a reasonable approach to prevent further episodes. However, the application of rate smoothing in patients with implantable defibrillators, although safe, did not show a
reduction in ventricular episodes, with a small increase in the percentage of ventricular pacing.\(^8\)

Our patient had a history of ischemic cardiomyopathy and stable heart failure symptoms just before the VRS algorithm was turned on. His cumulative percentage of ventricular pacing went from 0.2% up to 23.8% in the few days that preceded HFH. There was also an increase in spontaneous and pacing-induced ventricular arrhythmias, including frequent participation of ventricular pacing in short–long–short cycles, suggesting an effect of a critically timed premature ventricular contraction-like effect. Premature ventricular contraction (PVC)-induced intraventricular dyssynchrony is known to compromise global cardiac function, and the effect is more deleterious with high arrhythmia burden.\(^9\)–\(^11\) In addition, the presence of NSVT, multiform PVCs and those originated in the right ventricle were associated with a decline in the LV systolic function.\(^11\) In this patient, it is reasonable to accept that a combination of increasing RVA pacing, a marked increase in ventricular ectopy, and runs of NSVT played a role in the occurrence of HFH. The near cessation of RV pacing and ventricular arrhythmias after the suppression of the VRS algorithm along with prompt symptomatic relief proved a likely cause of worsening heart failure symptoms.

In conclusion, the activation of a rate modulation algorithm to prevent pause-dependent polymorphic ventricular tachyarrhythmias in patients with ICDs may not be inconsequential, and could lead to heart failure due to increasing amounts of RVA pacing and pacing-facilitated ventricular tachyarrhythmias. Frequent device remote monitoring downloads may be appropriate after the activation of such algorithms, in order to closely follow up on the percentage of RVA pacing, burden of ventricular arrhythmias, intrathoracic impedance monitoring, and heart rate variability when available, to anticipate episodes of heart failure exacerbations and prevent hospitalizations.

References