

## Treatment of Ventricular Tachycardia Storm with Transvenous Overdrive Pacing in a Patient with a Normal QT Interval

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### Abstract

**Background:** Ventricular tachycardia (VT) storm is usually treated with anti-arrhythmics; however, it is unclear how to treat refractory cases.

**Case Summary:** A 65-year-old man with CAD s/p CABG and ICM presented after multiple VF shocks from a wearable cardiac defibrillator, 3 weeks post-NSTEMI that was non-revascularizable. He received a subcutaneous implantable cardioverter defibrillator (S-ICD). Subsequently, he went into medication-refractory polymorphic VT storm. Given the appearance of R-on-T phenomenon during episodes of sinus bradycardia despite a normal QTc interval, a TVP was emergently placed, and the VT was eliminated with overdrive pacing. Reduction of the backup pacing rate reinitiated VT. The S-ICD was exchanged for a transvenous ICD, with no further VT or ICD shocks.

**Conclusion:** This case highlights the diagnostic and therapeutic value of overdrive pacing in medication-refractory VT storm, even without QT prolongation, by demonstrating how pacing can suppress PVC-triggered arrhythmias by modifying myocardial excitability. Overdrive pacing should be considered early in refractory VT storm—particularly when PVC-triggered arrhythmias occur in the setting of bradycardia.

**Keywords:** Ventricular tachycardia; Overdrive pacing; Temporary transvenous pacemaker

### Introduction

Ventricular tachycardia (VT) storm can be a life-threatening arrhythmic emergency and is associated with significant morbidity, recurrent hospitalizations, and increased mortality, particularly in patients with ischemic cardiomyopathy [1,2]. Despite advances in device therapy and pharmacologic management, VT storm remains a challenging clinical problem.

The underlying pathophysiology of VT storm is multifactorial and often involves a vulnerable myocardial substrate combined with transient triggers such as ischemia, electrolyte abnormalities, autonomic imbalance, or premature ventricular complexes (PVCs) [1,2]. Bradycardia may further facilitate arrhythmogenesis by prolonging repolarization and increasing susceptibility to R-on-T-mediated ventricular arrhythmias [3].

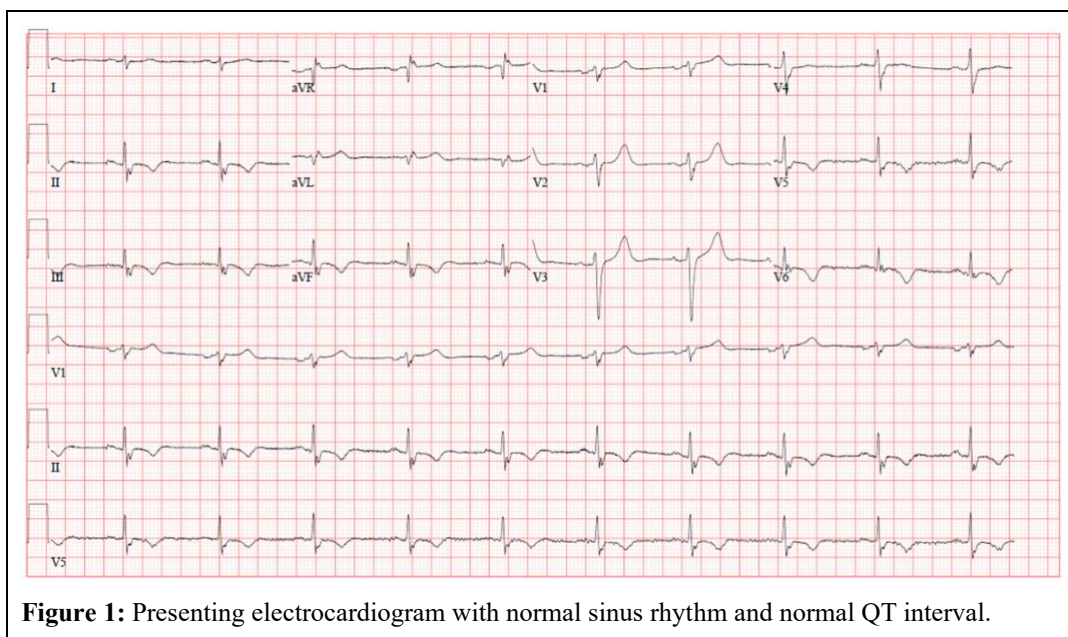
Standard management of VT storm includes correction of reversible triggers, anti-arrhythmic drug therapy, beta-blockade, sedation, and defibrillation [1,2,4,5]. In refractory cases, catheter ablation, autonomic modulation, or mechanical circulatory support may be required [1]. Overdrive pacing is a well-established therapy in congenital or acquired long QT syndrome; however, its role in suppressing VT storm in patients with normal QT intervals is less clearly defined and remains underrepresented in contemporary literature [6-9].

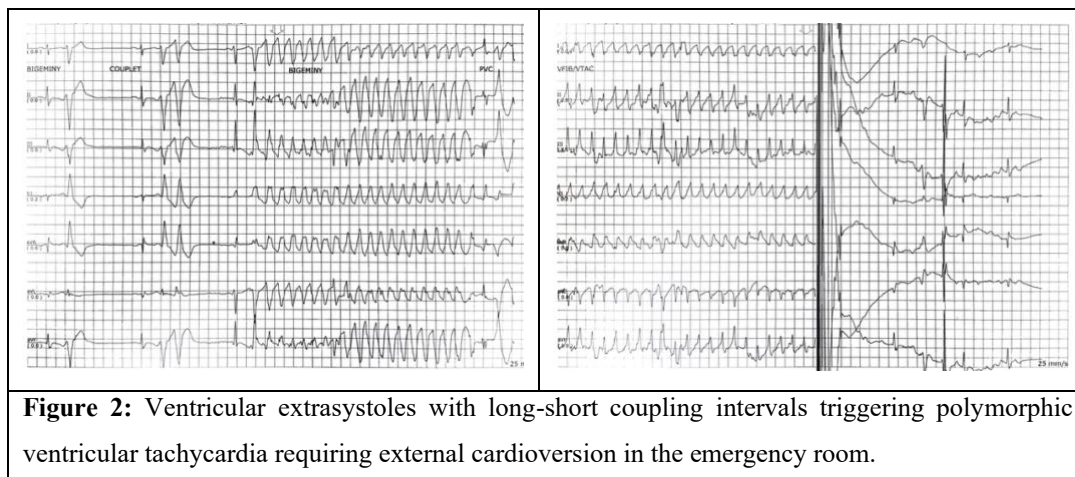
Here, we report a case of medication-refractory polymorphic VT storm triggered by short-coupled PVCs in the setting of sinus bradycardia and a normal QT interval, successfully treated with transvenous overdrive pacing.

### Case Presentation

A 65-year-old man with coronary artery disease status post coronary artery bypass graft (2006), ischemic cardiomyopathy, hypertension, hyperlipidemia, and a recent admission for non-ST elevation myocardial infarction presented for multiple shocks from a wearable cardiac defibrillator (WCD).

The patient had three episodes of lightheadedness without syncope followed by appropriate shocks from his WCD for ventricular fibrillation. Physical examination was unremarkable. Presenting labs were notable for a high-sensitivity troponin level of 275 ng/L (normal  $\leq 22$  ng/L)—which was less than the prior admission's peak of 5406 ng/L, potassium level of 5.2 mmol/L (normal  $\leq 5.1$  mmol/L), and magnesium level 2.6 mg/dL (normal range 1.6-2.6 mg/dL). Initial 12-lead electrocardiogram (EKG) showed sinus rhythm at 62 beats per minute (bpm), QT/QTc intervals of 442/448 milliseconds, and no acute ischemic ST-segment changes (Figure 1). While in the emergency room, he had PVCs with right bundle morphology and superior axis inducing polymorphic VT requiring external cardioversion to sinus rhythm (Figure 2). He was bolused with intravenous amiodarone 150 milligrams twice with a continuous drip initiated at 1 milligram/minute and lidocaine 100 milligrams once with a continuous drip also at 1 milligram/minute. He was admitted to the cardiac intensive care unit for further work-up and management.

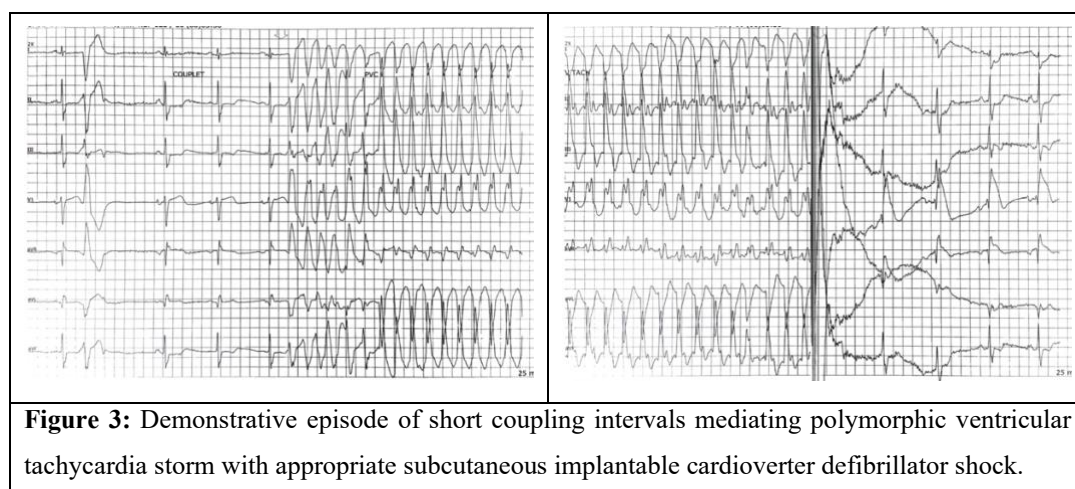




**Figure 2:** Ventricular extrasystoles with long-short coupling intervals triggering polymorphic ventricular tachycardia requiring external cardioversion in the emergency room.

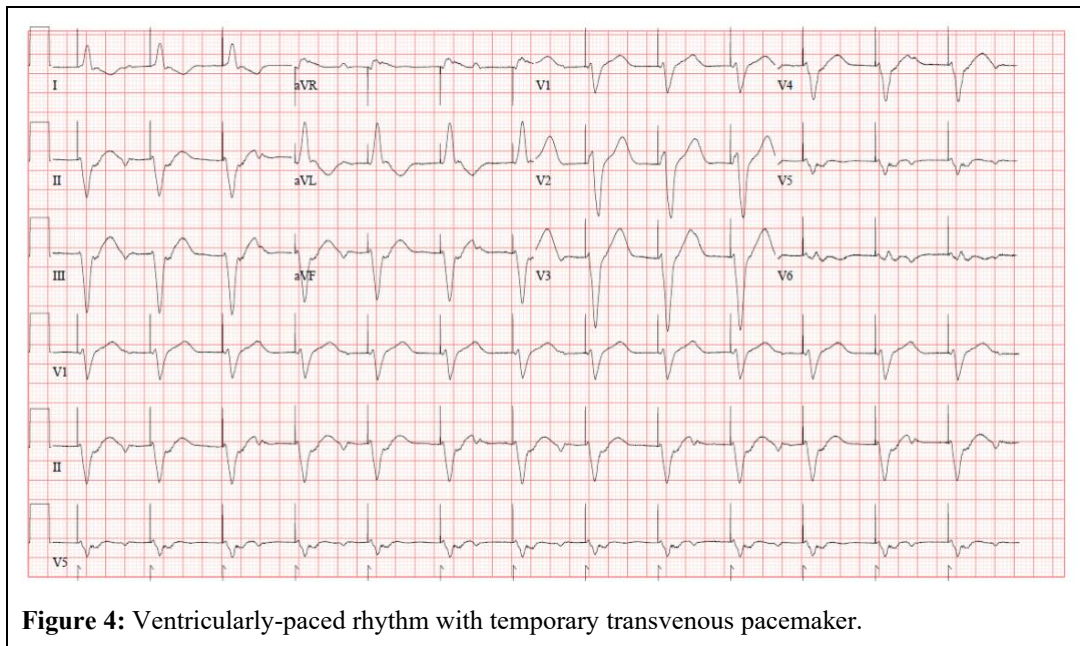
Two weeks prior to his current admission, the patient presented for chest pain and was found to have non-ST elevation myocardial infarction. Left heart catheterization at that time revealed a patent left internal mammary artery graft to his left anterior descending coronary artery, completely occluded venous grafts to the right coronary and first obtuse marginal arteries, and a severely obstructed venous graft to the ramus intermedius coronary artery. The acute ramus intermedius graft lesion was unable to be wired, and therefore no revascularization was ultimately able to be performed. Transthoracic echocardiogram demonstrated severe left ventricular dysfunction with an ejection fraction of 24%. His guideline-directed medical therapy was up titrated, and he was discharged with a WCD (ASSURE<sup>®</sup>, Kestra Medical Technologies, Kirkland, Washington).

Over the next 24 hours of his current admission, he was transitioned from intravenous amiodarone and lidocaine to oral amiodarone and mexiletine while carvedilol was initiated as guideline-directed medical therapy. Telemetry during this time remained electrically quiet. A subcutaneous implantable cardioverter defibrillator (Emblem<sup>™</sup> S-ICD, Model A219, Boston Scientific Corporation, Marlborough, Massachusetts) was implanted on hospital day 2 for secondary prevention. Overnight, the patient went into polymorphic VT storm requiring ten appropriate shocks from his S-ICD, refractory to multiple intravenous boluses of amiodarone, lidocaine, and propranolol. All episodes were hemodynamically stable with associated lightheadedness and without syncope. Telemetry revealed multifocal triggers of ventricular extrasystoles with short coupling intervals, sinus bradycardia with heart rate 50-55 bpm, and a normal corrected QT interval (Figure 3).

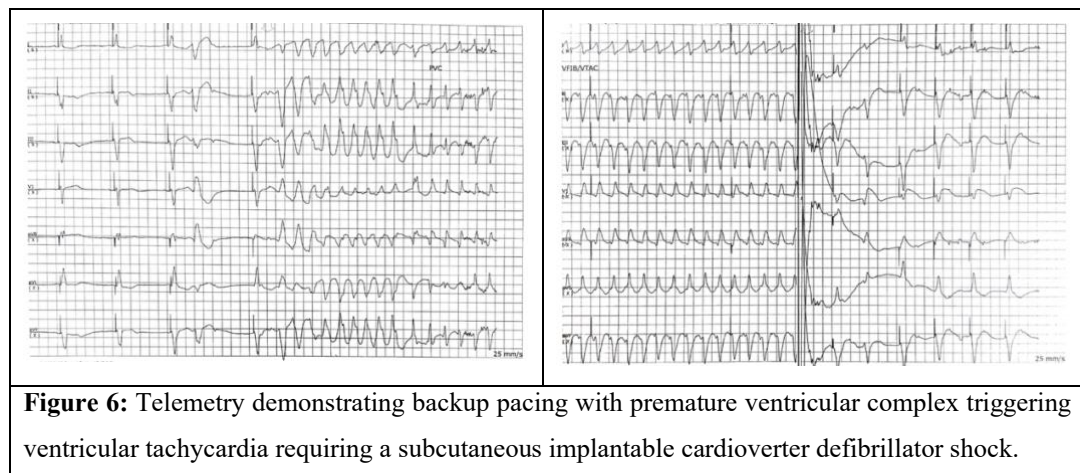
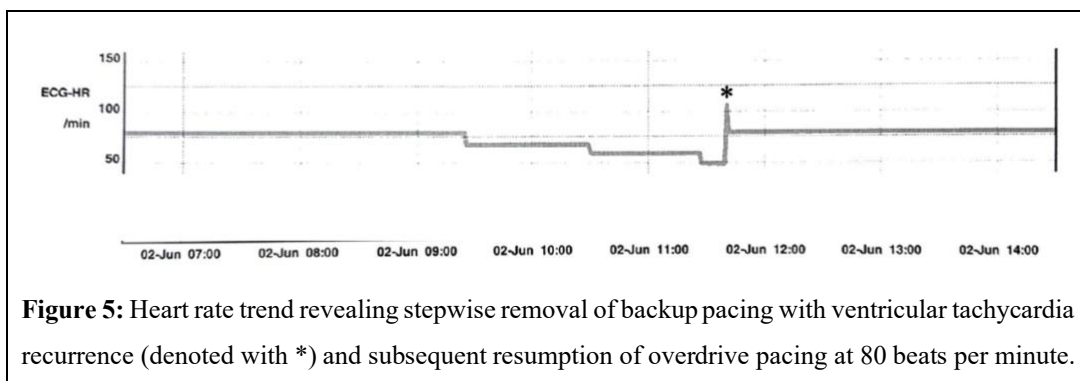


**Figure 3:** Demonstrative episode of short coupling intervals mediating polymorphic ventricular tachycardia storm with appropriate subcutaneous implantable cardioverter defibrillator shock.

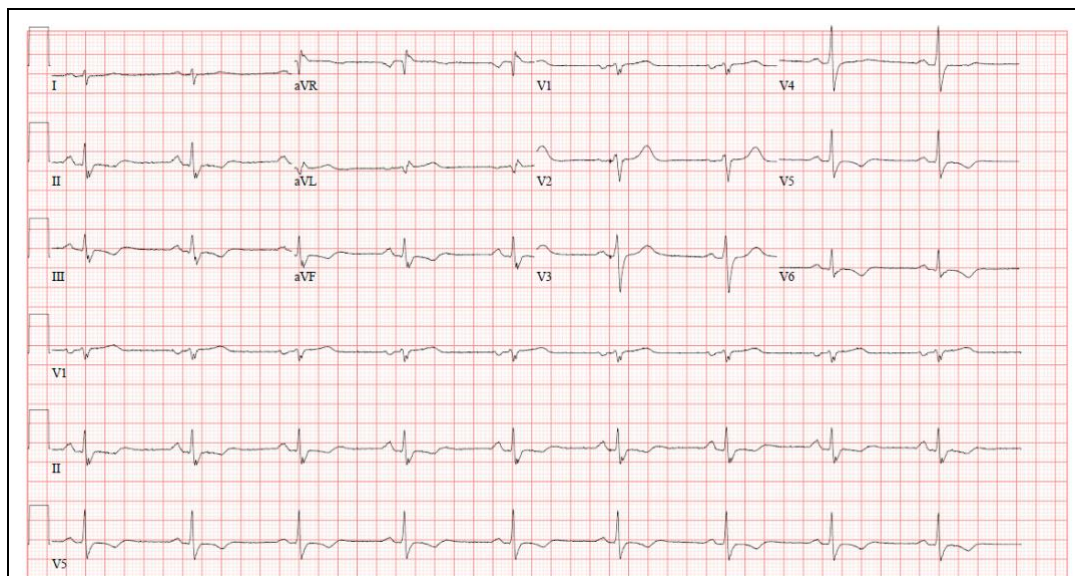
Given the underlying sinus bradycardia and the appearance of R-on-T phenomenon despite a normal QTc interval, a decision was made to emergently place a temporary transvenous pacemaker (TVP) to overdrive pace the patient’s sinus bradycardia to shorten the effective refractory period. After placement of the TVP set to 80 bpm, there was no recurrence of VT for over 24 hours (Figure 4).



On hospital day 4, the lower rate limit (LRL) of the backup pacing was reduced in a stepwise fashion (Figure 5). At a backup rate of 50 bpm, an untimely PVC once again degenerated the rhythm into sustained VT requiring an S-ICD shock (Figure 6).



On hospital day 5, the S-ICD was explanted. Given a QRS intermittently greater than 130 milliseconds (Figure 7) on a background of non-revascularizable ischemic cardiomyopathy, an endocardial cardiac resynchronization therapy defibrillator (RESONATE™ X4 CRT-D, Model G447, Boston Scientific Corporation, Marlborough, Massachusetts) was implanted and set to DDD mode with a LRL of 80 bpm. Throughout the remainder of the admission, there was no recurrence of sustained VT nor any ICD shocks. The patient was ultimately discharged on hospital day 8 on amiodarone, mexiletine, and carvedilol. He has had only one recurrence of ventricular tachycardia in over a year of follow-up when amiodarone was temporarily discontinued in the setting of worsening subclinical hypothyroidism with subsequent initiation of levothyroxine after resumption of amiodarone.



**Figure 7:** Electrocardiogram with QRS interval of 132 milliseconds and severely reduced left ventricular systolic function necessitating biventricular implantable cardioverter defibrillator implant.

## Discussion

Acute management options for hemodynamically unstable VT include anti-arrhythmics and cardioversion [1,2,4,5]. Our patient had VT storm that was refractory to multiple anti-arrhythmics. In our case, given the patient's underlying sinus bradycardia and appearance of R-on-T initiation of VT, overdrive pacing—a strategy that is well-described to shorten the QT interval in long QT syndrome—to decrease the effective refractory period provided a simple yet elegant solution. What was exceptional to this patient's case is that overdrive pacing was successful despite a normal QT interval. Overdrive pacing outside of that scenario is unusual, and we offer two hypotheses of why it may work. The first hypothesis is that this is a mechanism that likely reflects cycle-length dependence of calcium channel recovery and potassium current activation. By shortening the diastolic interval, overdrive pacing limits recovery of L-type calcium channels (ICa-L), reducing early and delayed afterdepolarizations, while maintaining activation of repolarizing potassium currents (IKr, IKs). These combined effects shorten the action potential duration and effective refractory period, thereby suppressing PVC triggers and interrupting re-entrant circuits. The second hypothesis is simply that the rate of overdrive pacing suppressed the PVCs; however, that seems less likely based on the short coupling intervals of the demonstrated PVCs (Figures 2 and 3). Regardless of the true mechanism, ultimately, overdrive pacing was successful as our patient had no further VT events.

Regarding prior similar cases, multiple reports have described overdrive pacing to treat ventricular arrhythmias from reversible etiologies such as post-coronary revascularization, long QT from new medications, or electrolyte abnormalities—with none of them requiring permanent device implant [3,6-9]. One example case describes a patient in his 50s presenting with an anterior ST-elevation myocardial infarction and VT who had a complete occlusion of his left anterior descending coronary artery; post-revascularization, the patient had VT storm requiring defibrillations that was ultimately successfully treated with overdrive pacing from a TVP at 100 bpm [6]. There were, however, other patients who did ultimately necessitate permanent device implant [10-15]. A retrospective study of one tertiary center's coronary care unit over six months saw 7 patients admitted for VT storm, with 3 receiving overdrive pacing via TVPs and the remainder receiving medical therapy alone [16].

In our patient, alternative management options such as intubation/sedation, radiofrequency ablation of the PVCs, or further medication therapy were considered. Given increased sympathetic activity contributing to VT storm, the first option could have been utilized to suppress adrenergic tone. Second, while radiofrequency ablation was not feasible overnight, it was ultimately deferred during admission given multifocal PVC triggers without a clear dominant trigger as well as removal of overdrive pacing leading to recurrence of VT likely proving a bradycardia-mediated phenomenon. Third, leadless atrial pacing could have been an elegant option rather than exchanging the whole device, however this was not available at our institution at that time. Finally, quinidine in a patient post-myocardial infarction with possible His-Purkinje PVC-triggered VT could have been considered, however risking possibly prolonging the QT interval, potentially exacerbating the VT storm. Chronotropic agents such as isoproterenol or epinephrine could have also been considered, though these medications are relatively contraindicated early post-myocardial infarction. However, a well-placed temporary wire may offer more reliability than medical therapy. Given the likely PVC-mediation of VT storm, it was reasonable to consider overdrive pacing as a temporary bridge to a more definitive solution of permanent endocardial device implant.

Ultimately, while pacing for PVC-induced VT may not be novel, this case contributes educational value by demonstrating its utility in a real-world scenario outside of long QT contexts and emphasizing thoughtful consideration of device strategy in refractory VT management.

## Conclusions

Overdrive pacing via a TVP successfully terminated medication-refractory VT storm triggered by short-coupled PVCs in a patient with bradycardia and a normal QT interval, underscoring the importance of recognizing pacing as a possible therapeutic tool in select patients with refractory VT.

**Abbreviations:** bpm = beats per minute; EKG = electrocardiogram; ICD = implantable cardioverter defibrillator; LRL = lower rate limit; PVC = premature ventricular complex; S-ICD = subcutaneous implantable cardioverter defibrillator; TVP = transvenous pacemaker; VT = ventricular tachycardia; WCD = wearable cardiac defibrillator

**Patient Perspective:** “My experience with the episode that brought me to the hospital was at times nerve-racking, filled with uncertainties and unknowns. While I was always comfortable with the level of care I was being provided at Stony Brook, it was Dr. Zhang who, in my humble opinion, made all the difference with his intervention. I don't know how many more shocks my heart was going to be able to withstand, but when Dr. Zhang explained to me what he wanted to do, it made perfect sense to me, and I had complete confidence in his abilities. I remain eternally grateful for his intervention.”

The patient provided written consent for publication of this case report.

**Running Title:** VT overdrive pacing

**Financial disclosures:** None.

**Conflict of Interest:** None.

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