

CASE REPORT

CLINICAL CASE

Cardiac Resynchronization Therapy-Induced Proarrhythmia

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ABSTRACT

BACKGROUND Cardiac resynchronization therapy (CRT)-induced proarrhythmia is a serious and an unpredictable clinical entity, which is usually manifested by recurrent sustained ventricular tachycardia or arrhythmic storm, refractory to antiarrhythmic therapy. It typically develops early after CRT implantation.

CASE SUMMARY We describe a unique case of CRT-induced proarrhythmia with first ventricular tachycardia episodes occurring >4 years after CRT implantation due to the unfortunate combination of coincidences. Programming optimization led to the disappearance of arrhythmic episodes.

TAKE-HOME MESSAGES The case report proves that sometimes even dramatic clinical conditions, such as an arrhythmic storm, can be quickly resolved by modifying programming. (JACC Case Rep. 2026;■:107282)

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HISTORY OF PRESENTATION

A 75-year-old man with mixed cardiomyopathy (ischemic and nonischemic) in NYHA functional class III was referred for electrophysiological study (EPS) and ablation of recurrent ventricular tachycardias (VTs) at the beginning of 2024.

PAST MEDICAL HISTORY

The cardiac resynchronization therapy with defibrilator was implanted in February 2018. A quadripolar (LV) lead was placed in the anterior branch of the coronary sinus. The reason for choosing this suboptimal LV lead position was not docu-

mented. The absence of a suitable posterolateral branch comes into consideration. Despite the suboptimal position, the QRS duration width narrowed by 20 milliseconds after initiation of biventricular pacing. In addition, 6 months after the cardiac resynchronization therapy (CRT) implantation, a cardiac magnetic resonance showed reverse remodeling, with a reduction of end-diastolic volume and an improvement in LV ejection fraction from the initial 20% to 38%. Transmural late gadolinium enhancement of postischemic fibrotic origin on the inferior wall remained stable (Figures 1 and 2).

First VT episodes occurred in June 2022, >4 years after CRT implantation. Thereafter, new treated VT episodes were documented at each follow-up.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

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**ABBREVIATIONS
AND ACRONYMS****AV** = atrioventricular**CRT** = cardiac
resynchronization therapy**EPS** = electrophysiological
study**LV** = left ventricular**PES** = programmed electrical
stimulation**PVC** = premature ventricular
complex**RV** = right ventricular**RVOT** = right ventricular
outflow tract**VT** = ventricular tachycardia

All device parameters were within the normal range, as was the QTc interval. The pharmacotherapy of heart failure was evaluated as optimal and well tolerated.

INVESTIGATIONS AND MANAGEMENT

Programmed electrical stimulation (PES) was performed using an 8-beat drive train at 3 different cycle lengths (500, 427, and 373 milliseconds). This was followed by up to 3 extrastimuli delivered down to the ventricular refractory period (with a minimum coupling interval of 200 milliseconds). PES within the area of the apex and right ventricular outflow tract (RVOT) did not induce

any VT. The procedure was performed using a Stereotaxis magnetic navigation system and the CARTO mapping system. An electroanatomic map of the left ventricle was created, revealing areas of fractionated potentials and late potentials on the inferior wall. Radiofrequency ablation homogenization of the scar on the inferior wall with eradication of late potentials followed. At the end of the procedure, a negative PES was performed again.

After EPS, repeated appropriate shocks occurred in the ward. Amiodarone therapy was initiated and recoronarography followed. It showed chronic total occlusion of the right coronary artery, unchanged from previous findings and unsuitable for revascularization.

A new PES was indicated but again yielded negative results. Because no VT was induced during EPS, repeated radiofrequency ablation of fractionated potentials on the inferior wall with transition to the septum followed. This time, the procedure was performed using the CARTO mapping system and a

TAKE-HOME MESSAGES

- CRT-induced proarrhythmia is a serious and unpredictable clinical entity, which is usually manifested early after CRT implantation by recurrent sustained ventricular tachycardia or arrhythmic storm, refractory to pharmacotherapy, and often accompanied by acute heart failure frequently leading to cardiogenic shock.
- This case emphasizes the importance of individual device programming and the necessity of the physician's expertise in troubleshooting because sometimes even dramatic clinical conditions, such as an arrhythmic storm, can be quickly resolved by modifying the programming.

SmartTouch ablation catheter. At the end of the procedure, pacing spikes on the T-wave of premature ventricular complex (PVC) triggering a nonsustained VT were observed ([Figure 3](#)). On closer inspection of the CRT device recordings, a diagnosis of CRT-induced proarrhythmia with a multifactorial mechanism was established. Although targeting the premature ventricular contractions was a potential strategy, we opted against immediate comprehensive mapping and ablation. This decision was based on the fact that the ventricular ectopy was highly infrequent and predominantly polymorphic during the procedure, initiating the sustained arrhythmia in only a single instance. Consequently, we prioritized implantable cardioverter-defibrillator programming optimization, deferring PVC ablation to a subsequent procedure if required. After adjusting the programming of RV-Blank after A-pace, no more VTs occurred and the patient was discharged. At 6-month follow-up, no new arrhythmic events were observed.

VISUAL SUMMARY Timeline of the Case

Timeline	Events
February 9, 2018	CRT-D implantation.
August 6, 2018	CMR showing reverse remodeling with a reduction of EDV and an improvement in LV ejection fraction.
June 2022-January 2024	Repeated VT episodes recorded by the device.
June 22, 2024	First EPS with a negative PES at the end of the procedure. After the EPS, repeated adequate shocks occurred in the ward
February 28, 2024	A new EPS was indicated; however, it was negative again. At the end of the procedure, pacing spikes on the T-wave of PVC triggering a nonsustained VT were observed. Optimization of the device programming followed.
July 25, 2024	Follow-up. No new VT recordings since the programming optimization.

CMR = cardiac magnetic resonance; CRT-D = cardiac resynchronization therapy with defibrillator; EDV = enddiastolic volume; EPS = electrophysiological study; LV = left ventricular; PES = programmed electrical stimulation; PVC = premature ventricular complex; VT = ventricular tachycardia.

DISCUSSION

CRT-induced proarrhythmia is a rare complication of CRT, which should be included in the differential diagnosis of recurrent sustained VTs. However, if an arrhythmic event does not occur early after the device implantation and/or the morphology of the arrhythmia lacks a right bundle branch block pattern (which points to the origin in the left ventricle), the CRT-induced proarrhythmia is usually no longer considered. Several possible pathophysiological mechanisms have been described.

PACING FROM WITHIN OR ADJACENT TO AN EPICARDIAL SCAR. Pacing directly within an arrhythmogenic substrate can lead to the formation of a re-entry circuit and is typically manifested by monomorphic sustained VT. The second mechanism is reverse ventricular activation and prolongation of transmural dispersion of repolarization. In the case of increased dispersion of repolarization, prolongation of action potentials leads to early subsequent depolarization and to re-entry arrhythmia. Thus, increased dispersion of repolarization increases the risk of adequate antitachycardia therapy.¹

Pacing adjacent to a slow conduction area appears to be a predictor of an arrhythmic event. Triggered VT is also predominantly monomorphic. Ablation of areas of late potentials and slow conduction is an effective treatment. Despite the theoretical risk of damage to the LV lead, the ablation does not lead to a significant deterioration of the pacing threshold. This type of CRT-induced proarrhythmia occurs more often in nonischemic cardiomyopathies with an epicardial located scar.²

CRT-INDUCED PROARRHYTHMIA DUE TO THE LV LEAD POSITION. An important determinant for the arrhythmia's occurrence is the position of the LV lead itself. According to the MADIT-CRT trial, anterior LV lead placement increases the risk of arrhythmic events in comparison with the posterior and lateral placement. In addition, patients with anterior LV lead position had an increased incidence of myocardial infarctions.³

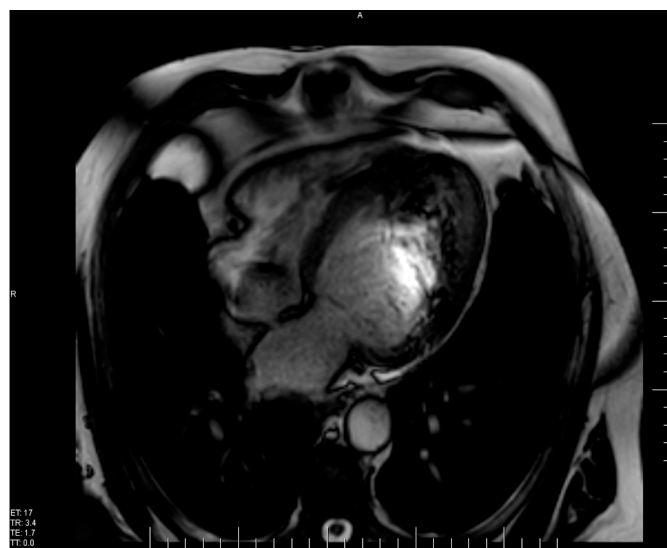
However, even in the case of optimal lead position, local mechanical irritation may occur. Remodeling and scarring of vulnerable tissue at the site of the LV lead due to the mechanical irritation is proposed as one of the possible mechanisms which lead to changes in conduction and/or myocardial repolarization. Mechanically induced VT has the same morphology as a paced QRS duration in the LV only

FIGURE 1 Cardiac Magnetic Resonance Before CRT Implantation

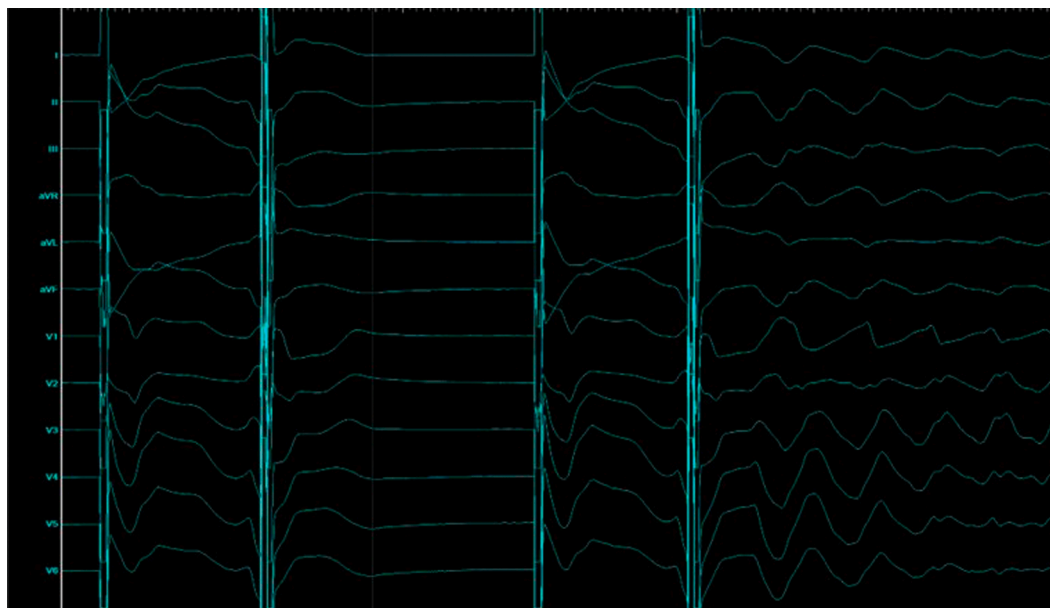
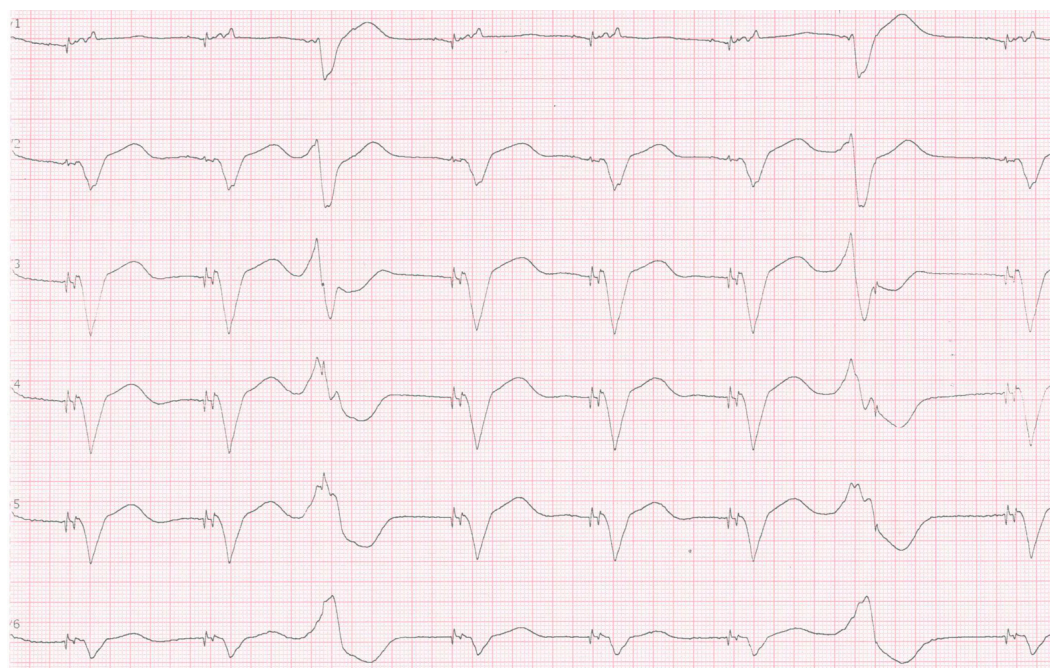


Examination showed dilated left ventricle with severe systolic dysfunction (ejection fraction: 20%, end-diastolic volume: 379 mL). CRT = cardiac resynchronization therapy.

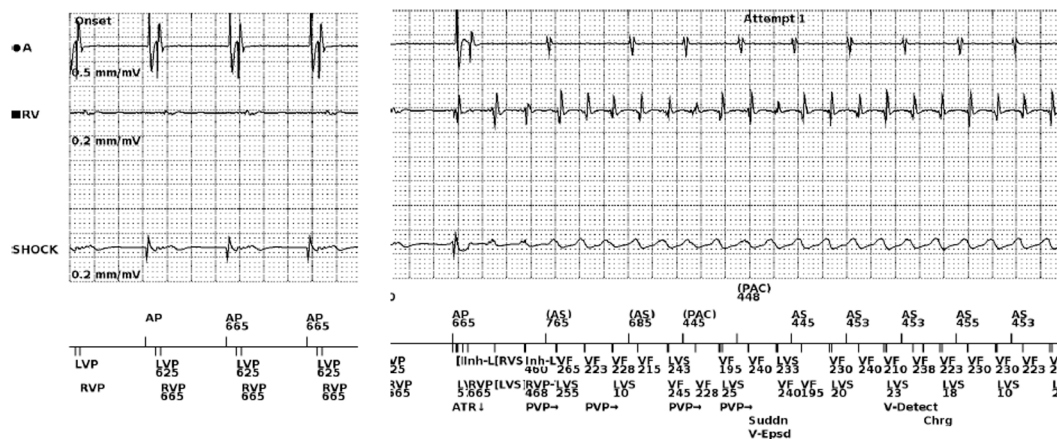
FIGURE 2 Cardiac Magnetic Resonance 6 Months After CRT Implantation



The examination showed reverse remodeling with the reduction of end-diastolic volume (193 mL) and the improvement of left ventricular systolic function (ejection fraction: 38%).

FIGURE 3 Ventricular Tachycardia Triggered by Pacing Spike Within the Premature Ventricular Complex's T-Wave**FIGURE 4** Electrocardiogram With Documented Pacing Spikes in the Beginning of the Premature Ventricular Complex T-Wave

As evident from the tracing, pacing spikes in the T-wave do not trigger ventricular tachycardia in this electrocardiographic recording.

FIGURE 5 EGM Recording of the Pacing-Induced VT With Sweep Speed 25 mm/s

Left ventricular sensed event occurs very shortly after atrial pace, which has already initiated right ventricular (RV) blanking period. The major part of the premature ventricular complex (PVC) falls into the RV blanking period. Subsequently, RV pacing triggers ventricular tachycardia (VT) by hitting the T-wave of the PVC. EGM = electrogram.

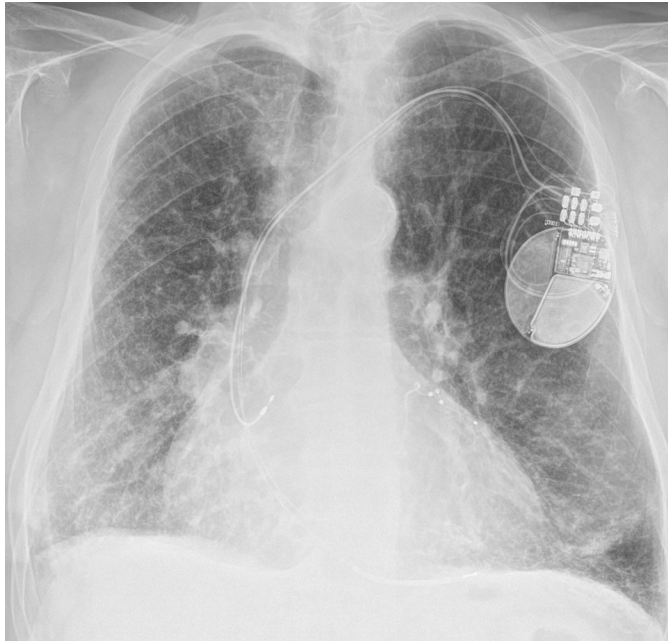
setting, and modification of the programming does not lead to the disappearance of arrhythmic events.⁴

CRT-INDUCED PROARRHYTHMIA DUE TO THE DEVICE PROGRAMMING. Various CRT functions and algorithms may induce a VT under certain circumstances.

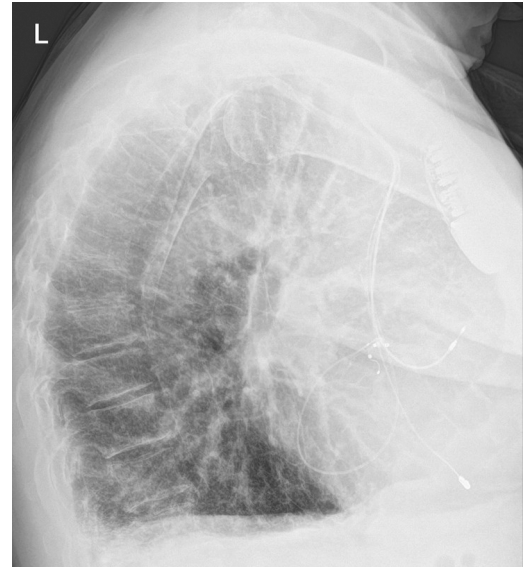
Typically, this phenomenon is described in adaptive atrioventricular (AV) shortening algorithms. The adaptive AV shortening algorithm searches the intrinsic AV conduction by prolonging the AV interval. If the intrinsic AV conduction is not detected

FIGURE 6 EGM Recording of the Pacing-Induced VT With Sweep Speed 100 mm/s

Same event as in the [Figure 5](#). In the first PVC, note the atrial pacing (marker AP), which occurs at the time of PVC onset. The sensed event from the right ventricle falls into the blanking period after AP and is marked as RVS. The LVS marker indicates sensing by the left ventricular lead, which results in inhibition of left ventricular pacing after the programmed AV delay (marker Inh-LVP), followed by RV pacing (marker RVP), after which VT is induced. In Boston Scientific CRT devices, overall ventricular timing is determined by the right ventricle, therefore neither LVS nor RVS prevents RV pacing, which in this particular case fell into the vulnerable phase of PVC. AV = atrioventricular other abbreviations as in [Figure 5](#).

FIGURE 7 Chest Radiograph: Anteroposterior View

Atrial lead is in the right atrial appendage, with the right ventricular lead in the apex and the left ventricular lead in the anterior branch of the coronary sinus.

FIGURE 8 Chest Radiograph: Lateral View

within the programmed time interval, the RR interval will be suddenly shortened by pacing. Significant shortening of the RR interval, can lead to PVC.⁵

In all previously mentioned mechanisms of CRT-induced arrhythmias, VT appears typically early after device implantation. This case report is unique because the first VT occurred years after the implantation. Even within CRT-induced proarrhythmia, this is a very rare phenomenon. After analysis of the CRT recordings and the surface electrocardiogram, a pacing spike within T-wave of PVC was identified as the trigger of VT (**Figures 4 to 6**). Even a low output pacing during the vulnerable period represents a known risk of malignant arrhythmia (the so-called R-on-T phenomenon). The search for the cause of the pacing spike on the T-wave led to an interesting finding. PVCs had left bundle branch block morphology and superior axis with the origin most probably in the RVOT. However, the CRT recordings showed PVCs that were first sensed by the LV lead. This discrepancy can be explained by the anterior position of the LV lead, which was therefore more proximal to the RVOT area (**Figures 7 and 8**).

Further analysis of the CRT recordings showed LV sensed PVC, which was followed by right ventricular

(RV) pacing, which fell into the vulnerable phase of the PVC.

Now, why did the device pace shortly after the sensed ventricular event?

Closer inspection of the recordings showed atrial pacing shortly before the LV sensed PVC. Hence, the RV lead was not able to sense the PVC due to the blanking period after the atrial pacing. In Boston Scientific CRT devices, overall ventricular timing is determined by the right ventricle. Therefore, the only explanation is that the PVC from the RVOT occurred very shortly, almost simultaneously, after atrial pacing, which already started the blanking period. PVC was sensed by the anterior LV lead and its whole QRS duration was undersensed by the right ventricle due to the ongoing blanking period after atrial pacing. Finally, RV pacing followed, hitting directly the T-wave of the PVC, and triggered VT.

In Boston Scientific CRT devices, inhibition of RV sensing is a consequence of the RV blank after A-pace setting, which is an interventricular blanking period designed to promote adequate sensing of RV events. It prevents oversensing of interventricular events after atrial pacing. However, in the case of PVC, the probability of undersensing of R-wave after atrial pacing increases.

For CRT Autogen X4, the RV blank after A-pace is nominally set to 65 milliseconds. After adjusting the blanking period from 65 milliseconds to Smart mode, pacing into the T-wave no longer occurred despite the persistence of frequent PVCs. The Smart option

EQUIPMENT LIST

CRT-D Boston Scientific Autogen X4 G179

- Integrated bipolar single-coil RV lead Reliance 4-Front 0696
- Quadripolar LV lead Acuity X4 Spiral S 4674
- Atrial lead Ingevity MRI 7741

EPS

- SJM Inquiry Decapolar L
- SJM Inquiry Quadripolar L
- ACUSON AcuNav intracardiac ultrasound probe
- Carto Biosense Webster 3D navigation system
- Pentaray mapping catheter
- Stereotaxis system
- NaviStar RMT ThermoCool

CRT-D = cardiac resynchronization therapy with defibrillator;
EPS = electrophysiological study.

allows the automatic gain control sensing threshold to automatically increase when the Smart blanking period expires. This promotes sensing of R waves that would otherwise fall within the blanking period. The Smart blanking function does not change the programmed sensitivity setting.⁵

It was a truly unfortunate combination of coincidences: the occurrence of PVCs from the right ventricle, sensed by the LV lead and the accidental timing of atrial pacing simultaneously with PVC. Due to the absence of PVC-related symptoms and the disappearance of VT episodes after the programming optimization, ablation of PVCs was not indicated. The optimal effect of CRT with proven reverse remodeling despite the anterior position of the LV lead can be explained by the anterior localization of the latest electrical activation. In daily routine, a purely anatomically guided implant strategy is

mainly used; however, more individual strategies should be considered. Implantation of the LV lead with a favorable Q-LV interval was shown to be independently associated with greater reverse remodeling and symptomatic improvement in 6 months.⁶ Although it was repeatedly shown that the lateral-posterolateral region is the most beneficial for both acute and long-term clinical response in CRT patients, some patients might benefit from anterior LV lead position.⁷

CONCLUSIONS

CRT-induced proarrhythmia is a rare complication of CRTs, which should be included in the differential diagnosis of a recurrent VT, especially in case of its manifestation early after the implantation. Several mechanisms were described; however, the exact pathophysiology is still unclear. We describe a unique case of CRT-induced proarrhythmia occurring years after CRT implantation due to the unfortunate combination of coincidences.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS cardiac pacemaker, cardiac resynchronization therapy, ventricular tachycardia