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| 著者  | Author(s)                                                       |
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| Konishi, Hiroki / Fukuzawa, Koji / Mori, Shumpei / Kiuchi, Kunihiko / Hirata, Ken-ichi |

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The limitations and potential adverse effects of the premature ventricular contraction response

Hiroki Konishi MD\textsuperscript{1} | Koji Fukuzawa MD\textsuperscript{1,2} | Shumpei Mori MD\textsuperscript{1} | Kunihiko Kiuchi MD, FHIRS\textsuperscript{1,2} | Ken-ichi Hirata MD\textsuperscript{1,2}

\textsuperscript{1}Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Kobe, Japan
\textsuperscript{2}Section of Arrhythmia, Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Kobe, Japan

Correspondence
Koji Fukuzawa, Section of Arrhythmia, Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Kobe, Japan.
Email: kfuku@med.kobe-u.ac.jp

Abstract
A 69-year-old man, who had undergone surgery for mitral and tricuspid regurgitation with the Maze procedure for paroxysmal atrial fibrillation, was admitted with an episode of syncope due to sick sinus syndrome. Three days after implantation of a dual-chamber pacemaker (Accent MRI\textsuperscript{\textregistered}, St. Jude Medical Inc.), ventricular pacing on T-wave was recorded multiple times. St. Jude Medical Inc. pacemakers have a unique additional algorithm, called premature ventricular contraction response, related to preventing pacemaker-mediated tachycardia. This algorithm was determined to be a cause of ventricular pacing on T-wave. We report the limitations and potential adverse effects of such automated algorithms.

KEYWORDS
pacemaker, pacemaker mediated tachycardia, premature ventricular contraction response, ventricular pacing on T-wave

1 | INTRODUCTION

The basic concept of pacemaker operating settings to prevent pacemaker-mediated tachycardia (PMT) is similar among each manufacturer, including an algorithm extending the postventricular atrial refractory period (PVARP) after a premature ventricular contraction (PVC). St. Jude Medical Inc. developed a unique algorithm, called PVC response, for a part of algorithms to prevent PMT. We report a case with ventricular pacing of T-wave caused by this PVC response algorithm.

2 | CASE REPORT

A 69-year-old man was admitted to our hospital because of an episode of syncope. Two years earlier, he had undergone surgery for mitral and tricuspid regurgitation, with the Maze procedure for paroxysmal atrial fibrillation. A 12-lead electrocardiogram showed regular rhythm. P-wave was unremarkable because of previous Maze procedure (Figure 1A). Twenty-four-hour ambulatory monitoring revealed sinus arrest lasting eight-seconds following the termination of paroxysmal atrial tachycardia. Subsequent to a diagnosis of sick sinus syndrome, a dual-chamber pacemaker (Accent MRI\textsuperscript{\textregistered}, St. Jude Medical Inc., Minneapolis, MN, USA) was implanted.

At the time of implantation, the P-wave amplitude was 2.0 mV during sinus rhythm. The atrial pacing threshold was 0.75 V/0.4 ms, and the atrial lead impedance was 380 Ω. The R-wave amplitude was 10.2 mV, the ventricular pacing threshold was 1.0 V/0.4 ms, and the ventricular lead impedance was 530 Ω.

Pacemaker programming was set as follows: pacing mode, DDD; tracking rate, 60 to 130 ppm; paced atrioventricular (AV) delay, 200 ms; sensed AV delay, 150 ms; postventricular atrial blanking period (PVAB), 150 ms; and PVARP, 275 ms. Ventricular intrinsic preference, the program to avoid ventricular pacing, was enabled to extend AV delay for another 100 ms (Figure 1B). Ventricular blanking after atrial pacing was programmed to 44 ms. Auto-sensing, PMT response, and PVC response were also activated. The auto-mode
switch was programmed to convert to DDI mode with a rate of 60 ppm when the pacemaker confirmed atrial fibrillation.

Three days after the pacemaker implantation, ventricular pacing on T-wave was recorded multiple times in ambulatory monitoring (Figure 2), always during the paroxysmal atrial tachycardia of 125 bpm with oscillation. 12-lead electrocardiograms during atrial tachycardia were also devoid of apparent P-wave (Figure 1C).

3 | DISCUSSION

A ventricular spike on a T-wave usually occurs in the undersensing of the preceding R-wave. In the present case, however, R-wave undersensing was unlikely as suspected by optimal and stable right ventricular lead location in frontal and lateral chest radiographs and stable R-wave amplitude without any change in ventricular pacing threshold that was confirmed by multiple device checks. In this
FIGURE 3  Premature ventricular contraction response algorithm. In addition to extending postventricular atrial refractory period (PVARP) after premature ventricular contraction (PVC), atrial pacing (AP) is applied 330 ms after the sensed atrial event (AR) within the extended PVARP to prevent compensatory pause following PVC. AS: atrial sensing, ECG: electrocardiogram, VP: ventricular pacing, VS: ventricular sensing.

FIGURE 4  Internal device electrogram and the diagram of device program operation. Single asterisks indicate atrial events that are undersensed due to the postventricular atrial blanking (PVAB) during atrial tachycardia. The ventricular events following the undersensed atrial events were recognized as premature ventricular contraction (PVC), which extends the postventricular atrial refractory period (PVARP). When an atrial event (AR) happened to be recorded within this extended PVARP after PVAB, PVC response algorithm was activated, by applying atrial pacing (AP) 330 ms after the AR (gray arrow). Because the intrinsic ventricular excitation (double asterisk) happened to occur within the ventricular blanking (VB) after this AP, it was masked. Then, the ventricular pacing (VP) was applied after a programmed paced atrioventricular delay, resulting in the VP on the T-wave. Conversely, AP based on PVC response was precluded after the second AR because intrinsic ventricular sensing happened to be recorded 321 ms after the AR. ECG: electrocardiogram, VS: ventricular sensing.
setting, a particular ventricular blanking program presumably masked the R-wave.

When retrograde atrial excitation, caused by the preceding PVC, is recognized as an intrinsic atrial event, it increases the risk of PMT. An algorithm extending the PVARP after a PVC is commonly applied to mask the retrograde atrial event. In addition, as shown in Figure 3, PVC response delivers atrial pacing applied 330 ms after the sensed atrial event within the extended PVARP. This unique atrial pacing algorithm is effective to prevent compensatory pause after the initial PVC. Furthermore, this atrial pacing can preclude the next retrograde atrial excitation caused by second continuous PVC.

Figure 4 shows the internal device electrogram of the present case during atrial tachycardia, showing the mechanism of ventricular pacing on T-wave. When paroxysmal atrial tachycardia occurred, several atrial events were undersensed because of PVAB of the preceding intrinsic ventricular event. Thus, even in the situation without frequent PVC, most intrinsic ventricular events were mistakenly counted as PVC in the present case. Consequently, the ventricular events recognized as PVCs extended the PVARP from 275 ms to 475 ms. Furthermore, PVC response algorithm delivered atrial pacing applied 330 ms after the sensed atrial event which was happened to be recorded within this extended PVARP after PVAB. The intrinsic ventricular excitations were masked in the ventricular blanking provided during 44 ms from the atrial pacing delivered by PVC response. Accordingly, the ventricular pacing applied after a programmed paced AV delay (200 ms) resulted in the ventricular pacing on the T-wave of masked intrinsic ventricular excitation.

In the setting of current pacing on T-wave due to PVC response algorithm, the interval between the masked R-wave and inappropriate ventricular pacing spike is expected to be fixed approximately around 200 ms (programmed paced AV delay). Conversely, in the setting of R-wave undersensing, the interval between the R-wave and inappropriate ventricular pacing spike could be variable.

In the setting of atrial tachycardia, therefore, PVC response can be activated even without any PVCs (Figure 2). The first early spike in Figure 2 (white arrow) on T-wave was applied because the preceding intrinsic R-wave was masked within the ventricular blanking after the atrial pacing triggered by PVC response (atrial pacing spike was not recorded in this monitor strip). The R-wave (gray arrow) following the first spike was consequently masked by the ventricular refractory period after the first ventricular pacing spike. The second late spike (black arrow) on T-wave was triggered by atrial sensing, which was expected to be located just after the preceding R-wave (gray arrow). It was delivered after an appropriate interval from the first spike, determined by the limitation of maximum tracking rate.

In this case, the rate of the atrial tachycardia, as well as the prolongation of AV conduction time during atrial tachycardia due to decremental conduction property, would determine the frequency of ventricular events, overlapped by subsequent atrial events. Furthermore, oscillation in the rate of the atrial tachycardia would determine the frequency of atrial events that were recorded within the extended PVARP after PVAB. Considering these multiple factors involved in this case, the present situation would be infrequent.

As demonstrated in the present case, PVC response may have a potential risk of ventricular pacing on the T-wave, especially in the setting of atrial tachyarrhythmia with oscillation and AV conduction prolongation. Although pacing-induced ventricular fibrillation is rare, the potentially life-threatening risk should not be underestimated. In the present case, PVC response was turned off, which successfully rendered ventricular spikes on T-wave undetectable. This case highlights the limitations and potential adverse effects of such automated algorithms as PMT prevention/intervention, which could have resulted in ventricular proarrhythmia.

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CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

ORCID

Hiroki Konishi http://orcid.org/0000-0001-5365-6239
Kunihiko Kiuchi http://orcid.org/0000-0002-9305-4854

REFERENCES

1. St. Jude Medical Inc.: Merlin™ Patient Care System Help Manual. St. Jude Medical. Bradycardia and tachycardia devices, bradycardia parameters. 61–84.
2. El-Damaty A, Gray C, Sharma R, Sapp J. Atrial pace on PVC algorithm inducing ventricular fibrillation. Pacing Clin Electrophysiol. 2012;35:749–51.
3. Delacretaz E. Asynchronous ventricular pacing triggering ventricular fibrillation. J Cardiovasc Electrophysiol. 2004;15:963–4.

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