

# Effectiveness of changing conventional implantable cardioverter-defibrillator settings to intrinsic antitachycardia pacing–like settings in the treatment of ventricular tachycardia in patients with cardiac failure



Motomi Tachibana, MD,<sup>\*</sup> Akihiro Hayashida, MD,<sup>\*</sup> Kimikazu Banba, MD,<sup>\*</sup> Masahisa Arimichi, ME,<sup>†</sup> Yoshimasa Tsushima, MD,<sup>‡</sup> Atsushi Hirohata, MD<sup>\*</sup>

From the <sup>\*</sup>Department of Cardiology, Sakakibara Heart Institute of Okayama, Okayama, Japan,

<sup>†</sup>Department of Medical Engineering, Sakakibara Heart Institute of Okayama, Okayama, Japan, and

<sup>‡</sup>Department of Cardiovascular Surgery, Sakakibara Heart Institute of Okayama, Okayama, Japan.

## Introduction

Intrinsic antitachycardia pacing (iATP) is a new implantable cardioverter-defibrillator (ICD) program for the treatment of ventricular tachycardia (VT) based on electrophysiological methods. Although the efficacy of iATP has been reported,<sup>1,2</sup> at present, most patients with a previously implanted ICD cannot use the iATP program because of the limitations of these models. The iATP program immediately and automatically designs the next antitachycardia pacing (ATP) sequence based on the postpacing interval (PPI), the interval between ATP pulse administration for VT and the resumption of VT. Although the full formula is not publicly available, the most appropriate number of ATP pulses can be predicted from the approximate formula using the iATP program from Medtronic. As the calculation is simple, we adjusted the ATP pulse number settings of conventional ICD to the most appropriate pulse number using the formula. Here we present the case of a patient using a conventional ICD model that could not use the iATP program; conversion to an iATP-like pulse number setting calculated by the formula resulted in the effective treatment of VT.

## Case report

A 68-year-old man with an implantable cardiac resynchronization therapy defibrillator (CRT-D) was referred to our hospital with VT storm. At 62 years of age, the patient underwent

**KEYWORDS** Intrinsic antitachycardia pacing; Antitachycardia pacing; Implantable cardioverter-defibrillator; Ventricular tachycardia; Heart failure (Heart Rhythm Case Reports 2022;8:353–356)

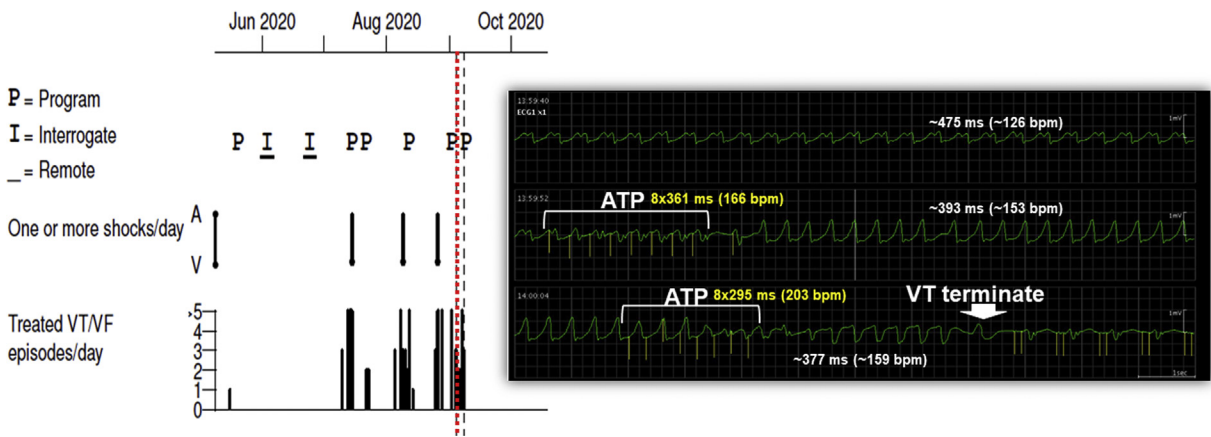
Funding Sources: None. Disclosures: The authors declare no conflicts of interest. **Address reprint requests and correspondence:** Dr Motomi Tachibana, Department of Cardiology, Sakakibara Heart Institute of Okayama, 2-5-1 Nakai-cho, Kita-ku, Okayama 700-0804, Japan. E-mail address: [motomitachibana@yahoo.co.jp](mailto:motomitachibana@yahoo.co.jp).

## KEY TEACHING POINTS

- Using an approximation of the intrinsic antitachycardia pacing (iATP) formula, the number of antitachycardia pacing (ATP) pulses required to reach the ventricular tachycardia (VT) circuit (the number of pulses required for S1) can be calculated.
- Determining the number of S1 pulses would help calculate whether the VT circuit can be reached with the set number of ATP pulses, even for patients using conventional implantable cardioverter-defibrillators and cases wherein iATP cannot be used.
- Knowledge of the minimum number of ATP pulses necessary (up to the number of pulses in S1 + 2 pulses) may help prevent VT acceleration by ATP.

aortic valve replacement and mitral valvuloplasty for severe aortic stenosis and severe mitral regurgitation. At 64 years of age, he started hemodialysis for renal failure. Echocardiography revealed reduced cardiac function with a left ventricular ejection fraction of 23% on echocardiography. At the age of 66 years, the patient underwent CRT-D implantation for repeated episodes of cardiac failure and sustained VT (Claria MRI Quad CRT-D DTMA2QQ; Medtronic Minneapolis, MN).

On admission, a 12-lead electrocardiogram showed atrial and biventricular pacing rhythms with a heart rate of 80 beats per minute. Electrocardiogram monitoring and device checks revealed that ATP was frequently but not successfully activated for VT with a cycle length of 460–500 ms. Eventually,

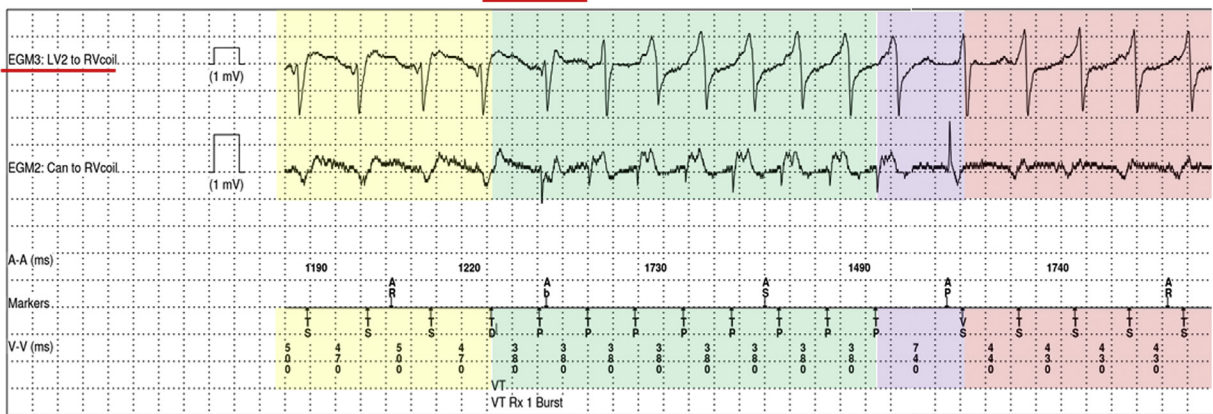


**Figure 1** Electrocardiography monitoring and device introductions on admission. (Right) Electrocardiography monitoring on admission. Antitachycardia pacing (ATP) was performed on ventricular tachycardia (VT) at 126 beats per minute but was unsuccessful. The VT was accelerated after ATP and then terminated spontaneously. (Left) Intra-cardiac data from the device. Frequent VT episodes were being treated. The red dotted line indicates the date of admission.

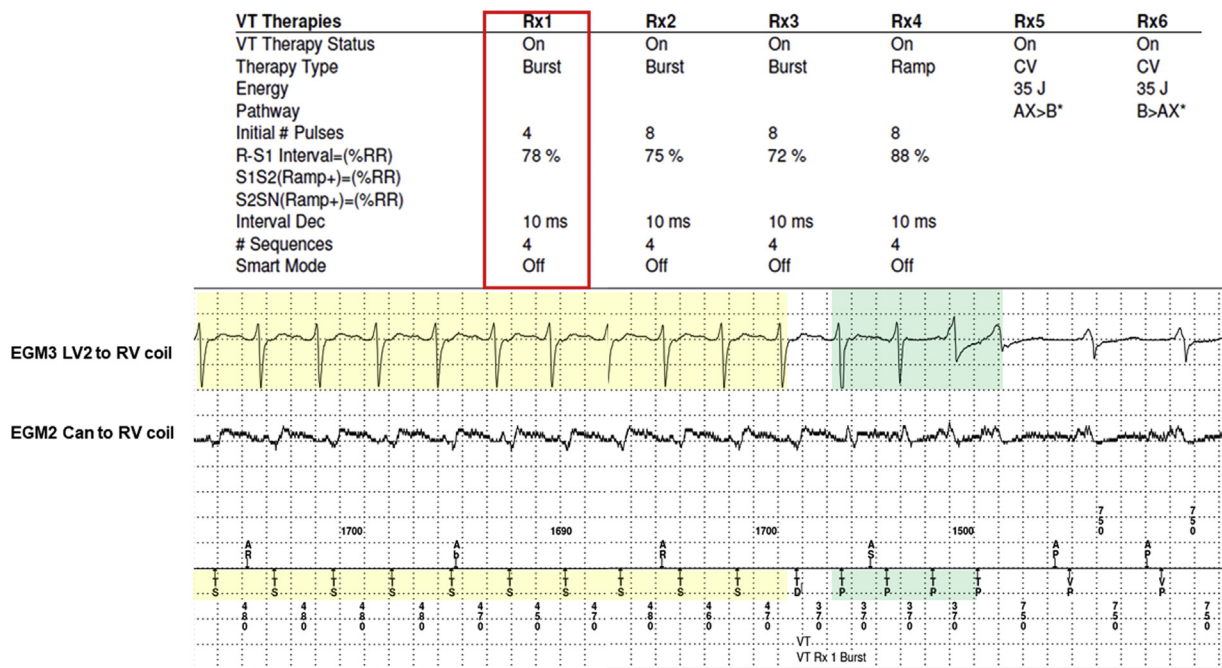
the VT self-terminated (Figures 1 and 2). The patient was already receiving standard therapy for cardiac failure and 20 mg of carvedilol and 200 mg of amiodarone for VT. His cardiac failure gradually worsened owing to repeated episodes of VT. Since conventional ATP had no effect on the VT, the patient was treated with frequent electrical shock defibrillation. This suggested that the number of ATP pulses was not appropriate for the VT; therefore, we changed the number of ATP pulses to that determined using the iATP program's formula.<sup>1,3</sup>

The initial treatment sequence of CRT-D for VT was set to 8 pulses at a 78% burst, which seemed effective in the early phase of treatment after implantation. According to the iATP program, the propagation time is the PPI minus the VT cycle length (VTCL) divided by 2 (A); because the ATP-VT wavefront collision advancement with 1 pacing pulse is the VTCL minus the ATP interval divided by 2 (B), the number of ATP pulses required to reach the VT circuit (the number of S1) can be calculated as A/B. Using this formula, we calculated the number of pulses needed to reach the VT circuit at 2.4, where

VT Therapies	Rx1	Rx2	Rx3	Rx4	Rx5	Rx6
VT Therapy Status	On	On	On	On	On	On
Therapy Type	Burst	Burst	Burst	Ramp	CV	CV
Energy					35 J	35 J
Pathway					AX>B*	B>AX*
Initial # Pulses	8	8	8	8		
R-S1 Interval=(%RR)	78 %	75 %	72 %	88 %		
S1S2(Ramp+)=(%RR)						
S2SN(Ramp+)=(%RR)						
Interval Dec	10 ms	10 ms	10 ms	10 ms		
# Sequences	4	4	4	4		
Smart Mode	Off	Off	Off	Off		



**Figure 2** Before changing antitachycardia pacing (ATP) pulse number setting (initial ATP pulse number was 8). ATP fails to stop ventricular tachycardia (VT); rather, it accelerates it. The patient's mean VT cycle length (VTCL) was 487 ms, ATP interval was 380 ms (burst, %RR = 78% of VTCL), and postpacing interval (PPI) was 740 ms. The area highlighted in yellow shows the VT before the application of ATP, green indicates ATP (8 pulses), purple indicates PPI, and red indicates VT after the delivery of 8 pulses of ATP.



**Figure 3** After changing antitachycardia pacing (ATP) pulse number setting (initial ATP pulse number was 4). Yellow indicates ventricular tachycardia (VT) before ATP application, while green indicates ATP (4 pulses). The VT was terminated by the first ATP, followed by the A and V paces. RV = right ventricular.

PPI was 740 ms, VTCL was 487 ms, and ATP interval was 380 ms. The calculation indicated that at least 3 ATP pulses were required. In the iATP program, if VT was not terminated by S1, up to 2 pulses (S2 and S3) would be added to the ATP; thus, in this case, the number of ATP pulses was set at 3–5. Additionally, the minimum number of pulses in iATP was 4. Furthermore, this formula does not include a myocardial conduction delay, so it is possible that a few more ATP pulses may actually be needed. Therefore, we set the number of ATP pulses to 4, which was a reduction from the initial 8 pulses (Figures 2 and 3). After the ATP pulse number setting was changed, the probability of VT termination by the first ATP would increase.

To assess the efficacy of the reduction of ATP pulses from 8 to 4 for VT termination, we compared the effects of pre- and post-ATP changes, with 8 pulses and 4 pulses, respectively, on each of the 21 VT episodes. Efficacy was assessed as follows: the 21 VT episodes that occurred on the day prior to changing the settings were compared to the 21 VT episodes that occurred on the day after the settings were changed. The success rate of the initial ATP tended to increase after the settings were changed compared to the initial setting. Moreover, the number of ATP sequences required for VT termination was reduced in the period after the settings were changed to 4 ATP pulses. VT acceleration by ATP was seen in 7 of 21 episodes (33%) when the number of ATP pulses was 8, and in 3 of 21 episodes (14%) when the number of ATP pulses was 4 (Table 1). VT acceleration was seen in 3 episodes in the iATP-like setting (ATP with 4 pulses), but the VTCL of these 3 episodes was as fast as 400 ms, which was much faster than VT targeted in the iATP-like setting. The improved control of VT achieved by the new settings

improved the control of cardiac failure, and the patient was discharged.

## Discussion

Recent clinical trials showed that frequent cardiac defibrillation is associated with increased mortality.<sup>4,5</sup> There is also renewed focus on delaying the time to ICD detection of ventricular arrhythmias to avoid treating self-terminating episodes; therefore, optimal programming of ATP pulses to maximize efficacy and minimize VT acceleration or degeneration to ventricular fibrillation is required.

The automated ATP operates on 2 principles: entry into the VT circuit (by S1) and termination of VT by closing the excitable gap (by S2) without accelerating VT (by S1 not being aggressive). In a conventional ICD, depending on the model, the number of ATP pulses can be set at 1–15 without information concerning the most appropriate number of pulses. No information on the most appropriate number of ATP pulses sometimes results in a failure to discontinue VT

**Table 1** Difference in antitachycardia pacing efficacy before and after change to an intrinsic antitachycardia pacing-like setting

	Conventional ATP	iATP-like setting
Number of initial ATP pulses	8	4
Initial ATP success, n (%)	10 (48)	13 (62)
Total ATP sequence, n ± SD	2.0 ± 3.1	1.5 ± 1.4
VT acceleration <sup>†</sup> by ATP, n (%)	7 (33)	3 (14)

ATP = antitachycardia pacing; iATP = intrinsic antitachycardia pacing; VT = ventricular tachycardia.

<sup>†</sup>VT acceleration was defined as a VT cycle length that was 10% faster immediately after ATP than that before ATP.

effectively. Moreover, if the number of ATP pulses is more than necessary to terminate VT, VT may be triggered again after termination. These findings suggest that unnecessary surplus ATP pulses after reaching the VT circuit cannot terminate VT effectively and could sometimes accelerate VT. However, using the formula of the iATP program, it is possible to calculate the number of ATP pulses necessary to reach the VT circuit, even if the ICD model itself cannot use iATP.

The iATP is programmed to appropriately calculate the number of S1 ATP pulses required plus 2 when necessary. Because the iATP program showed a low rate of VT acceleration,<sup>1</sup> it was reasonable to consider that up to the calculated number of pulses to reach 1 VT circuit plus 1 or 2 pulses might not accelerate VT. In this case, the calculation predicted that 3 pulses of ATP were required to reach the VT circuit. Although we used 487 ms as a VTCL for calculation, the patient's actual VTCL was 460–500 ms, so we added 1 pulse to the calculated minimum number as a margin. In this case, the number of ATP pulses was changed from 8 to 4 (3 plus 1 pulse). As a result, VT termination by ATP became more successful, and the control of the cardiac failure improved.

As for the fundamental treatment of VT, it is necessary to consider medications such as antiarrhythmic drugs, control of cardiac failure, and catheter ablation. Catheter ablation in particular is effective in acute suppression of electrical storm, often represents a life-saving therapy, and may effectively reduce frequent ATP or shock of ICD.<sup>6,7</sup> However, in a patient with VT that is uncontrolled by a conventional ICD, it may be worthwhile to try resetting the ATP to an appropriate number of pulses using this formula when the previous pulse number was suspected to be inadequate. Resetting of the ATP pulse number in a conventional ICD can be easily and quickly performed, even in the emergency room.

In our patient, 3 episodes of VT were accelerated by our modified ATP program, but VTCL before modified ATP in all 3 episodes was 400 ms, which was shorter than expected. The iATP was designed to adapt immediately and automatically to the most appropriate ATP pulse number for the next VT. However, conventional ICDs do not possess this function. Therefore, even after changing to an iATP-like setting, if the VT that appears different from the target VTCL, a conventional ICD would not effectively treat it. This problem remains an important limitation of conventional ICD models.

The refractory period of the myocardium is proportional to the length of the preceding cardiac cycle, and states with a short preceding cardiac cycle will have a short refractory period. In the case of the present patient, the cycle length of VT was relatively long, 460–500 ms; thus, the excitation gap of VT is also considered relatively long, and for this reason, it may be possible to stop the tachycardia with S1 alone. Therefore, if the VT cycle length is shorter, it is possible that the effect of this setting change will be lesser. Furthermore, in this case the R-S1 setting of ATP therapy was unusual in that it was a 78% burst, shorter than the conventional setting of approximately 88%; thus, it may have quickly reached the VT circuit and played a role in closing the excitable gap. In addition, although there was a marked therapeutic effect on VT with the change in ICD settings, the fact that this was a successful case in only 1 patient is also a limitation. Studies with a larger number of patients are required.

## Conclusion

Changing conventional ICD settings to iATP-like settings allowed us to treat VT more effectively with ATP. In addition, VT acceleration by ATP also decreased after the pulse number was changed to an iATP-like setting.

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