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CASE REPORT
Electrophysiology

Case report: successful termination of ventricular tachycardia by intrinsic anti-tachycardia pacing beyond conventional anti-tachycardia pacing

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Background Anti-tachycardia pacing (ATP) is a pain-free alternative to defibrillation shock for monomorphic ventricular tachycardia (VT). Intrinsic ATP (iATP) is a novel algorithm of auto-programmed ATP. However, the advantage of iATP over conventional ATP in clinical cases is still unknown.

Case summary A 49-year-old man with no significant past medical history was transferred to our institution with sudden-onset fatigue from working on a farm. A 12-lead electrocardiogram showed monomorphic sustained wide QRS tachycardia with a right bundle branch block pattern and superior axis deviation with a cycle length (CL) of 300 ms. Sustained monomorphic VT originating from the left ventricle due to underlying vasospastic angina was diagnosed by contrast-enhanced cardiac magnetic resonance imaging, coronary angiography, and the acetylcholine stress test, and implantable cardioverter defibrillator implantation was performed. Nine months later, a clinical VT episode with a CL of 300 ms was observed, which could not be terminated by three sequences of conventional burst pacing. Ventricular tachycardia was finally terminated by a third iATP sequence without any acceleration.

Discussion Although standard burst pacing by conventional ATP reached the VT circuit, it failed to terminate the VT. Using the post-pacing interval, iATP automatically calculated the appropriate number of S1 pulses required to reach the VT circuit. In iATP, the S2 pulses are delivered with a calculated coupling interval based on the estimated effective refractory period during tachycardia. In this case, iATP might have led to less aggressive S1 stimulation, followed by aggressive S2 stimulation, which probably helped terminate the VT without any acceleration.

Keywords Case report • Implantable cardioverter defibrillator • Anti-tachycardia pacing • Intrinsic anti-tachycardia pacing • Ventricular tachycardia • Sudden cardiac arrest

ESC Curriculum 5.6 Ventricular arrhythmia • 5.10 Implantable cardioverter defibrillators

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Learning points

- Intrinsic anti-tachycardia pacing is a novel algorithm of auto-programmed anti-tachycardia pacing (ATP).
- Intrinsic anti-tachycardia pacing allows the ATP to definitively reach the ventricular tachycardia (VT) re-entrant circuit, resulting in an appropriate coupling interval of extraventricular stimuli to create a conduction block in the VT circuit.
- Intrinsic anti-tachycardia pacing might improve the rate of successful termination of VT when compared with conventional ATP.

Introduction

Implantable cardioverter defibrillators (ICDs) are known to improve survival following sudden cardiac death in high-risk patients.[1] Anti-tachycardia pacing (ATP) is a kind of ICD therapy that delivers pacing pulses to interrupt ventricular tachycardia (VT) and restore normal sinus rhythm. There are two main types of ATP: burst and ramp. Burst pacing is a sequence of pulses with fixed coupling interval (CI). Ramp pacing is a sequence of pulses with an auto-decremental interval, leading to progressive shortening of the CI.[2] Successful ATP for VT is reportedly not significantly associated with mortality in ICD recipients, and it can terminate regular VT painlessly, even for fast VT.[2,3] Hence, ATP is the preferred therapy over appropriate shocks for monomorphic VT.[4] On the other hand, although the success rate of ATP has been reported to be 61–96%, adverse effects of ATP, such as acceleration to rapid VT or ventricular fibrillation (VF), have also been observed.[4]

Intrinsic ATP (iATP) is a novel algorithm of auto-programmed ATP.[5] This algorithm allows the ATP to definitively reach the VT re-entrant circuit, resulting in an appropriate CI of extraventricular stimuli to create a conduction block in the VT circuit. Intrinsic ATP has two important features. First, the propagation time from the pacing site to the entrance of the VT circuit is calculated using the post-pacing interval (PPI), which is used to determine the number of pulses required to reach the VT circuit. Second, the S2 (and S3) pulses are delivered with a calculated CI based on the estimated effective refractory period (ERP) during tachycardia. Ventricular tachycardia can be terminated when the antidromic waves of S2 (and S3) collide with the active front of the previously stimulated beat and the orthodromic wave collides with the relative refractory period.[6]

A previous virtual modelling study showed that iATP more successfully improves the rate of successful termination of VT when compared with conventional ATP.[5] Several case reports have also shown the efficacy of iATP in different clinical settings with similar results.[7–9] However, the advantage of iATP over conventional ATP in clinical cases is still unknown. We experienced a case in which conventional ATP failed to terminate VT, but in which subsequent iATP within the same sequence of VT led to successful VT termination.

Timeline

Day 0: sustained ventricular tachycardia (VT) with a heart rate of 200 b.p.m. → lidocaine

During hospitalization

Day 8: contrast-enhanced cardiac magnetic resonance imaging: late gadolinium enhancement in the inferoposterior septum of the left ventricular apex

Day 11: coronary angiography: no significant stenosis

Day 11: acetylcholine stress test: positives

→ Diagnosis of sustained monomorphic VT originating from the left ventricle with underlying vasospastic angina

Continued

Continued

Day 15: implantable cardioverter defibrillator (ICD) was implanted

Nine months after the ICD implantation: sustained VT with a heart rate of 200 b.p.m.

Day 296: three sequences of conventional burst pacing failed to terminate VT

→ VT was terminated by a third intrinsic anti-tachycardia pacing (iATP) sequence in the same VT sequence

Follow-up

Day 354: iATP was programmed as the first therapy followed by conventional anti-tachycardia pacing

Day 585: VT was terminated by the second iATP sequence

Case presentation

The patient was a 49-year-old man with no significant past medical or family history. He was transferred to our hospital with sudden-onset fatigue from working on a farm. At admission, his level of consciousness was alert, his blood pressure was 170/120 mmHg, and his pulse rate was 200 .b.p.m.. On physical examination, S1 and S2 on cardiac auscultation were rhythmic, with no murmur. Coarse crackles were heard in the lung bilaterally. There was no oedema of the lower limbs. A 12-lead electrocardiogram showed monomorphic sustained wide QRS tachycardia with a right bundle branch block pattern and superior axis deviation with a cycle length (CL) of 300 ms (*Figure 1*). Intravenous administration of 50 mg lidocaine terminated the tachycardia. Transthoracic echocardiography demonstrated a preserved left ventricular (LV) ejection fraction of 64% and a slightly enlarged right ventricle (RV). We performed cardiac magnetic resonance imaging (MRI) to further assess for any evidence of underlying cardiomyopathy. A contrast-enhanced cardiac MRI did not show any specific features of arrhythmogenic cardiomyopathy with only late gadolinium enhancement in the inferoposterior septum of the LV apex and a slightly enlarged RV with reduced RV function (*Figure 2*). Hence, we decided to perform coronary angiography. Angiography did not show any significant stenosis of the coronary artery. However, acetylcholine injection into the left anterior descending artery and right coronary artery evoked significant diffuse stenosis with vasospasm. According to the result of coronary angiography, we confirmed that the substrate of VT was myocardial infarction due to vasospasm in this patient. After obtaining the patient's informed consent, a transvenous ICD was implanted. Dual-chamber ICD was chosen for the diagnosis of supraventricular tachycardia despite no necessity for atrial pacing at the moment. The ICD (Cobalt XT DR ICD: Medtronic, Inc., Minneapolis, MN, USA) was successfully placed by a standard transvenous procedure with the appropriate pacing and sensing thresholds for atrial and ventricular pacing. The tip of the shock lead was placed in the RV apex.

At the time of the patient's discharge from the hospital, ICD therapy was programmed as shown in *Figure 3*, as in the usual clinical set-up.

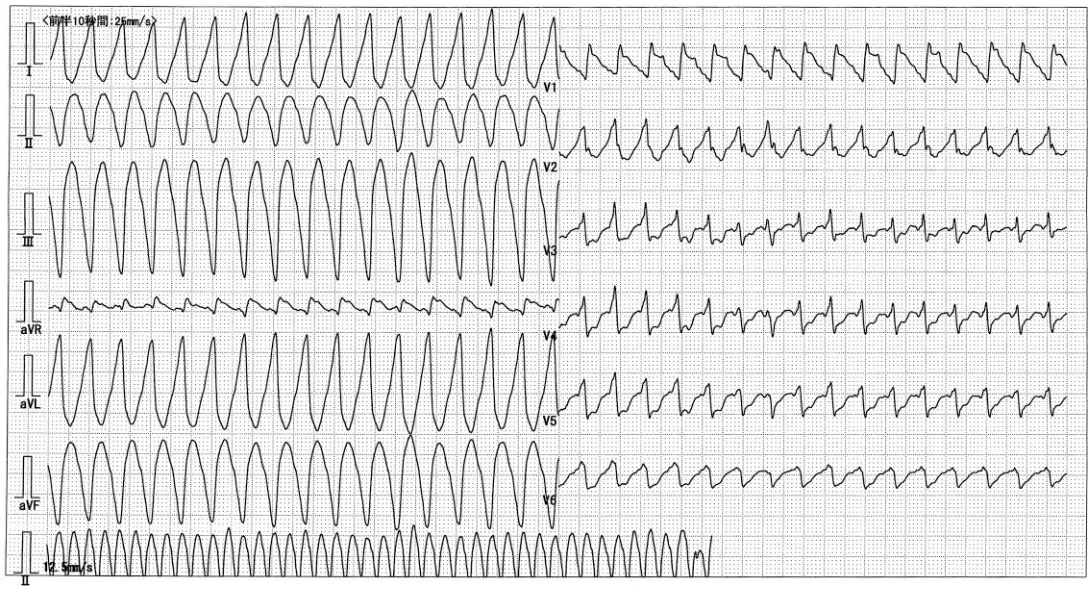

Figure 1 A 12-lead electrocardiogram during ventricular tachycardia. A 12-lead electrocardiogram showed monomorphic sustained wide QRS tachycardia with a right bundle branch block pattern and superior axis deviation with a cycle length of 300 ms.

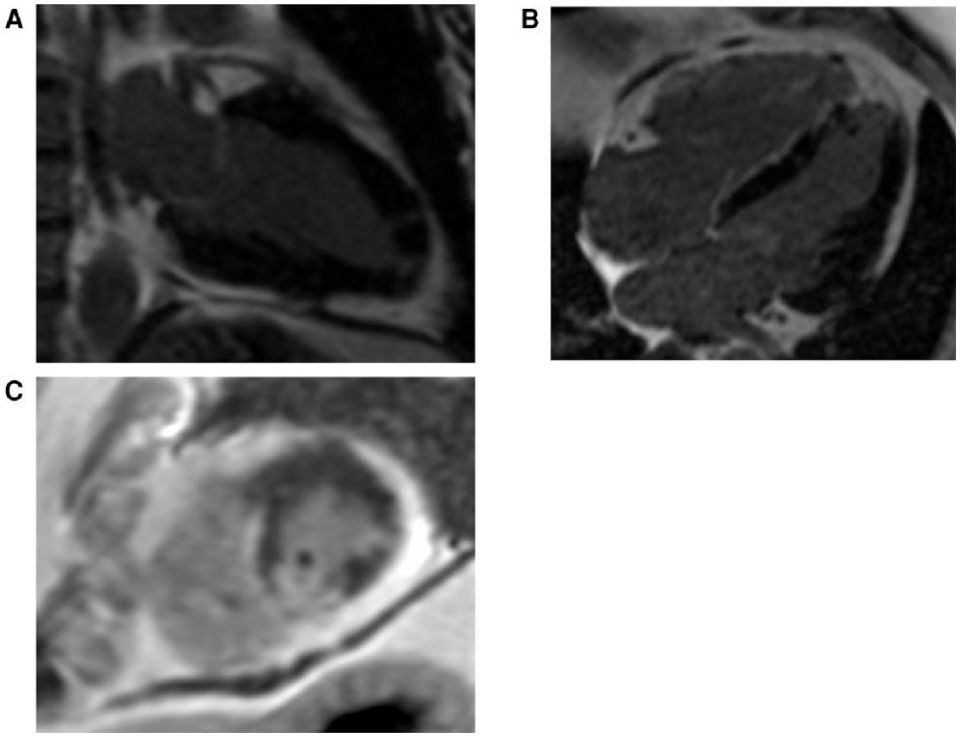

Figure 2 Late gadolinium enhancement of contrast-enhanced cardiac magnetic resonance imaging. (*A*) Two-chamber view of contrast-enhanced cardiac magnetic resonance imaging. Late gadolinium enhancement was positive at the left ventricular apex. (*B*) Four-chamber view of contrast-enhanced cardiac magnetic resonance imaging. Late gadolinium enhancement was positive at the left ventricular apex. The right ventricle was slightly enlarged, although there was no late gadolinium enhancement in the right ventricle. (*C*) Short-axis view of contrast-enhanced cardiac magnetic resonance imaging at the mid-apex level. Late gadolinium enhancement was positive in the inferoposterior septum of the left ventricular apex.

VT/VF Detection

		V. Interval (Rate)	Initial	Redetect
VF	On	270 ms (222 bpm)	30/40	12/16
FVT	Off			
VT	On	440 ms (136 bpm)	12	12
Monitor	Off	450 ms (133 bpm)	32	

VT Therapies	Rx1	Rx2	Rx3	Rx4	Rx5	Rx6
VT Therapy Status	On	On	On	On	On	On
Therapy Type	Burst	iATP	Ramp+	CV	CV	CV
Energy				30 J	40 J	40 J
Pathway				B>AX*	B>AX*	B>AX*
# Sequences	3	10	3			
S2/S3 Minimum		160 ms				
Initial # Pulses	10		10			
R-S1 Interval=(%RR)	88 %		88 %			
S1S2(Ramp+)=(%RR)			84 %			
S2SN(Ramp+)=(%RR)			81 %			
Interval Dec	10 ms					
Smart Mode	Off	Off	Off			

Shared V. ATP

V-V Minimum ATP Interval	200 ms
V. ATP Amplitude	8 V
V. ATP Pulse Width	1.5 ms
V. ATP Pace Blanking	170 ms

Shared V. Therapies

Active Can/SVC Coil	SVC Off
Progressive Episode Therapies	Off
Confirmation+	On

Figure 3 Settings of the implantable cardioverter defibrillator for ventricular tachycardia therapy. The cycle length in the ventricular tachycardia zone was <420 ms. Anti-tachycardia pacing for ventricular tachycardia was programmed as Rx.1. Conventional anti-tachycardia pacing comprised 3 sequences of 10 pulses at 88% of the ventricular tachycardia cycle length. Intrinsic anti-tachycardia pacing was programmed as Rx.2. The minimum V–V interval was set as 200 ms, and the minimum coupling interval of S2/S3 was 160 ms.

Sequential ATP was programmed to evaluate the efficacy of iATP compared with standard ATP. Standard burst pacing with 3 sequences of 10 pulses at 88% of VTCL was programmed as the first therapy (Rx.1). Subsequently, iATP was programmed as the second therapy (Rx.2).

Nine months after the ICD implantation, a home-monitoring system recorded a clinical VT episode with a VTCL of 300 ms in this patient (*Figure 4*), which could not be terminated by the three sequences of conventional burst pacing. Subsequently, the first sequence of iATP was delivered with nine S1 pulses (88% of VTCL), followed by an S2 pulse with a CI of 260 ms (*Figure 5A*). The second iATP was delivered with four S1 pulses of 280 ms and one S2 pulse of 240 ms. Ventricular tachycardia was finally terminated by a third iATP sequence of four S1 pulses and one S2 pulse, with CIs of 280 and 220 ms, respectively (*Figure 5B*).

During this episode, the patient did not feel any discomfort. To compare the efficacy of ATP with iATP, we considered to program iATP as the first therapy. Ten months after the first episode, VT episode with a VTCL of 310 ms was observed, and the VT was terminated by the second sequence of iATP without any acceleration. No anti-arrhythmic drug was administered; however, no shock therapy was delivered until today.

Discussion

We report a case that demonstrates the advantage of iATP compared with conventional ATP therapy in the same VT sequence in a clinical situation. Other conventional forms of ATP, such as burst pacing, ramp pacing, and scan pacing, have not previously shown any clinical benefit in terms of VT termination when directly compared against each other.[2,10,11] Anti-tachycardia pacing therapy is the preferred therapy over appropriate shock for VT, although the termination rate is not satisfactory and sometimes leads to an acceleration of the rhythm to rapid VT or VF.[4]

To resolve these issues, iATP has been introduced, which has two important features that improve the success rate of VT termination: (i) the appropriate number of S1 pulses required to reach the VT circuit is automatically calculated using the PPI; and (ii) the S2 (and S3) pulses are delivered with a calculated CI based on the estimated ERP during tachycardia. The efficacy of iATP has already been reported in a virtual modelling study.[5] That study showed that the success rate of ATP was 73% with iATP and 56% with conventional ATP, although the acceleration rate was not significantly different.[3] However, although there are few previous case series, no large clinical trial has shown the advantage of iATP compared with conventional ATP.[6,12]

In iATP, the propagation time from the pacing site to the entrance of the VT circuit is calculated as (PPI—VTCL)/2. In this case, PPI was 330 ms and PPI—VTCL was 30 ms. The propagation time was, thus, only 15 ms, suggesting the close proximity of the distal electrode of the RV lead and the VT circuit, and that only two pacing pulses would be needed to reach the VT circuit. Based on the programmed algorithm, four S1 pulses is the minimum number that should have been administered to reach the VT circuit in the present case. The S2 and S3 pulses terminated VT in this case by collision of the antidromic wave of S3 with the wavefront propagated by the S1 pulse, and accession of the orthodromic wave to the refractory period following the S1 wavefront.

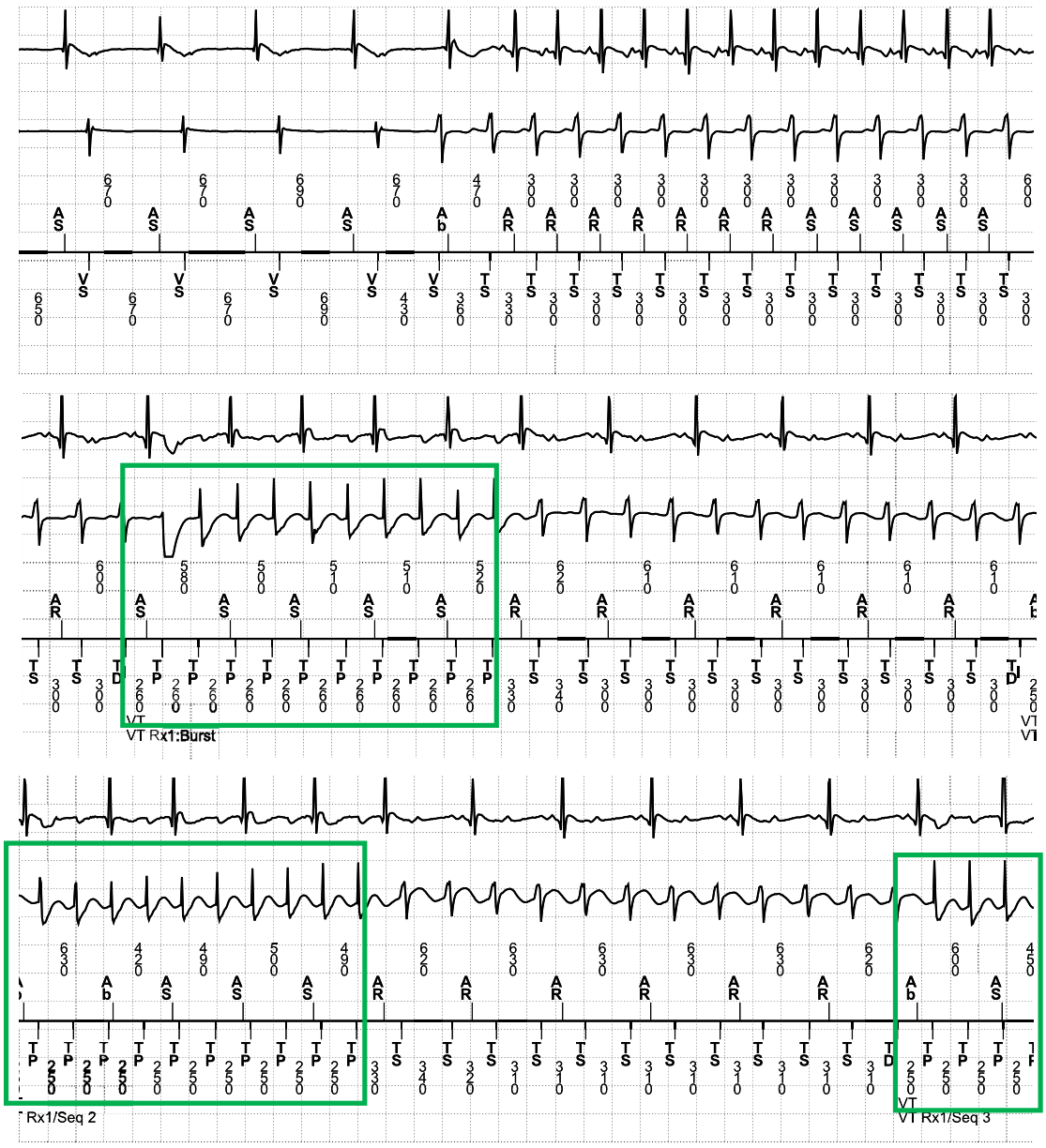

Figure 4 Intracardiac electrogram during ventricular tachycardia. The green squares show conventional anti-tachycardia pacing. Ventricular tachycardia with a cycle length of 300 ms was detected. Conventional anti-tachycardia pacing was attempted, which resulted in resetting the ventricular tachycardia, suggesting that the anti-tachycardia pacing reached the ventricular tachycardia circuit. The second anti-tachycardia pacing was decremented by 10 ms, leading to prolongation of the ventricular tachycardia cycle length to 310 ms.

In this case, standard burst pacing by conventional ATP did not terminate the VT, but prolonged the VTCL from 300 to 310 ms, suggesting that although these pulses reached the VT circuit, they could not terminate the VT.

There are two possible reasons for this observation: (i) the last orthodromic wave of conventional ATP might have reset the VT, such that it was not able to coincide with the relative refractory period; and (ii) the last orthodromic wave of conventional ATP might have induced another VT. Hagiwara *et al.*[6] showed that a short pacing interval might be needed when the conduction block is created by burst or ramp ATP, and that these pacings are associated with the potential risk of VT acceleration. Swenson *et al.*[5] showed that less aggressive S1 stimulation followed by aggressive S2 and S3 stimulation improves the efficacy of ATP without increasing the acceleration rate.

To the best of our knowledge, this is the first report comparing conventional ATP with iATP during the same episode of VT. However, our report has several limitations. Specifically, conventional ATP prolonged the VTCL from 300 to 310 ms, and we were unable to prove whether these two VTs with different CLs were the same VT despite the similarity in the intracardiac electrograms. Further studies are required to determine whether this algorithm is more effective for terminating VTs.

In conclusion, we report a case in which iATP successfully terminated VT following the failure of termination by conventional ATP.

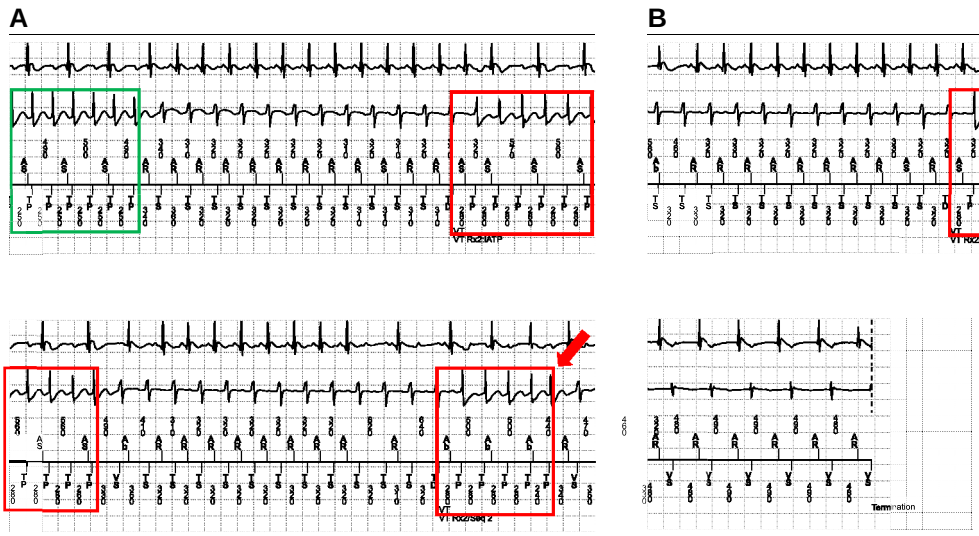

Figure 5 Intracardiac electrogram when ventricular tachycardia was terminated. (*A*) The green square shows conventional anti-tachycardia pacing, and the red square shows intrinsic anti-tachycardia pacing. A ventricular tachycardia cycle length of 310 ms was detected when the first intrinsic anti-tachycardia pacing was delivered. The first intrinsic anti-tachycardia pacing sequence consisted of nine S1 pulses with a coupling interval of 88% of the ventricular tachycardia cycle length, the same as that of conventional anti-tachycardia pacing, followed by an S2 pulse with a coupling interval of 260 ms that resulted in prolongation of ventricular tachycardia cycle length to 320 ms. The number of S1 pulses was calculated based on the default propagation time setting that was defined as 150 ms. The red arrow shows the second intrinsic anti-tachycardia pacing sequence that was determined using the prior failed anti-tachycardia pacing sequence. It consisted of only four S1 pulses, which was the programmed minimum number of S1 pulses. This indicates that the distal electrode of the shock lead was in close proximity to the ventricular tachycardia circuit, so that S1 pulses easily reached the ventricular tachycardia circuit. Hence, the S2 pulse was decremented by 20 ms. (*B*) The red square shows intrinsic anti-tachycardia pacing, and the blue arrow shows the third intrinsic anti-tachycardia pacing sequence. Ventricular tachycardia was successfully terminated by an S2 pulse that was further decremented by 20 ms.

Lead author biography

Shuichiro Kazawa graduated from Tokyo Medical University and received his medical degree in 2012. He worked as a Cardiology senior resident at Tokyo Medical University Hospital from 2014, a Cardiology fellow at Tachikawa General Hospital from 2015, and as a Cardiology fellow at Tokyo Medical University Hospital from 2017. He became an EP fellow at the Heart Rhythm Management Center in UZ Brussel from 2019. From 2021, he has worked as a Cardiology fellow at the Tokyo Medical University Hachioji Medical Center.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for the submission and publication of this case report, including the images and associated text, has been obtained from the patient in line with COPE guidance.

Conflict of interest: K.S. and S.K. have received honoraria from Medtronic. The other authors declare no conflicts of interest in association with this manuscript.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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