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Case Report

Endless loop tachycardia below the upper tracking rate of a pacemaker: A case report

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ABSTRACT

An 82-year-old female with a history of hypertrophic cardiomyopathy (HCM), sick sinus syndrome (SSS), and an implanted DDD pacemaker was admitted to our hospital for congestive heart failure caused by rapid atrial fibrillation. After administration of amiodarone, atrial fibrillation (AF) became atrial flutter (AFL). Electrophysiological investigation revealed counterclockwise AFL. Catheter ablation of the cavotricuspid isthmus was performed. Burst pacing from the coronary sinus ostium to confirm the block line of the isthmus induced rapid, regular, ventricular pacing at a rate of 110 bpm. The differential diagnosis of this tachycardia included ectopic atrial tachycardia and pacemaker-mediated, endless loop tachycardia (ELT). We diagnosed this arrhythmia as ELT, because temporary reprogramming of the pacemaker mode from DDD to VVI terminated the tachycardia. In this patient, pacing parameters favored ELT (long atrioventricular delay [AVD] and short postventricular atrial refractory period [PVARP]), and atrioventricular and ventriculoatrial conduction time was prolonged as a result of amiodarone administration.

The ELT continued because the rate was lower than the programmed upper tracking rate. Reprogramming the parameter (decreasing AVD and increasing PVARP) resulted in termination of ELT.

1. Introduction

Endless loop tachycardia (ELT) is a well-recognized complication of dual chamber pacing systems [1,2]. Recent pacemaker systems adopt algorithms of ELT termination given that ELT is generally maintained at the programmed upper tracking rate. The rate of ELT depends on the upper tracking rate, atrioventricular delay (AVD), and ventriculoatrial (VA) conduction time. The sum of the retrograde VA conduction time and the programmed AVD is equal to the ELT cycle length with a rate below the upper tracking rate [3]. In this situation, ELT does not satisfy the termination algorithm of some pacemakers, and the tachycardia may continue. We report a case of ELT below the upper tracking rate.

2. Case report

An 82-year-old female with hypertrophic cardiomyopathy (HCM; mid-ventricular obstruction) and sick sinus syndrome (SSS) was implanted with a DDD pacemaker 15 years prior to this admission. The pacemaker generator was replaced for consumption of its battery 7 years prior (Vertus plus II, Guidant, Indianapolis, Indiana, USA), and she was followed at our pacemaker follow-up clinic. She experienced subjective symptoms such as shortness of breath, palpitation, and general fatigue in March 2011, and visited our department. Chest X-ray revealed cardiomegaly (CTR=68%) and lung congestion. The electrocardiogram (ECG) performed upon admission revealed atrial fibrillation (AF), with a heart rate of 100 bpm and a negative T-wave in the precordial leads. We diagnosed the patient with congestive heart failure because of rapid AF and HCM, and began treatment. Echocardiogram showed asymmetric septal hypertrophy, a general decline in wall motion, and an ejection fraction of 43%.

Congestive heart failure was well-controlled by the administration of angiotensin-converting enzyme inhibitor and diuretics. Because the patient had low cardiac function, amiodarone was added (a loading dose of 400 mg/day) to control AF. After administration of amiodarone, AF became atrial flutter (AFL). We therefore performed an electrophysiological study (EPS). EPS revealed cavotricuspid isthmus (CTI)—dependent, counterclockwise AFL. Linear ablation of the CTI was performed. Burst pacing from the coronary sinus ostium to confirm the block line of the isthmus induced rapid, regular, ventricular pacing at a rate of 110 bpm

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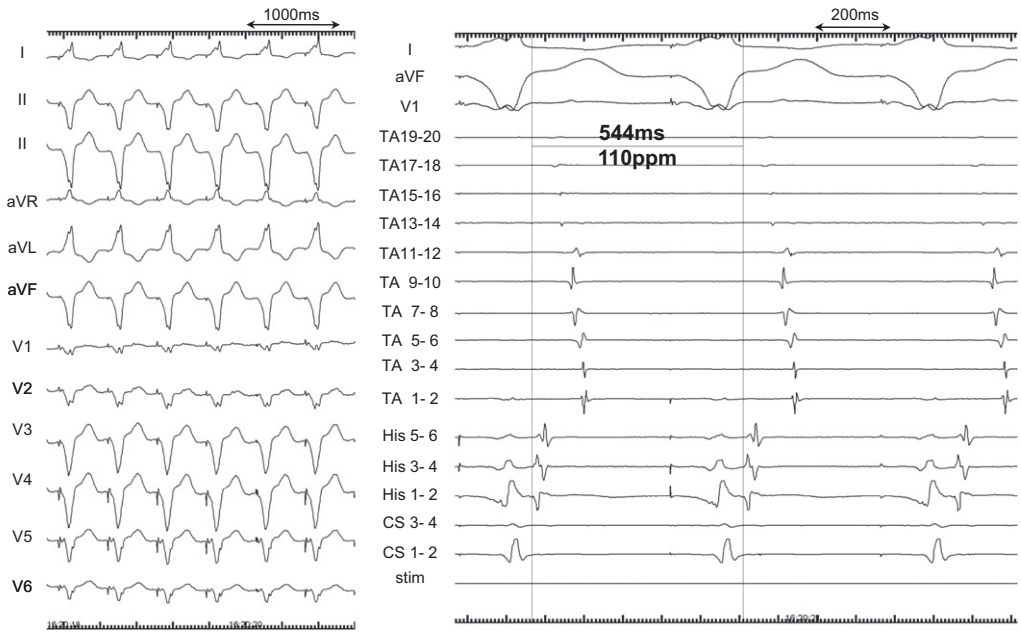

Fig. 1. (A) Electrocardiogram during rapid, regular ventricular pacing at a rate of 110 bpm. (B) Intracardiac electrogram during tachycardia. The earliest site of atrial activation was the His bundle electrogram recording site.

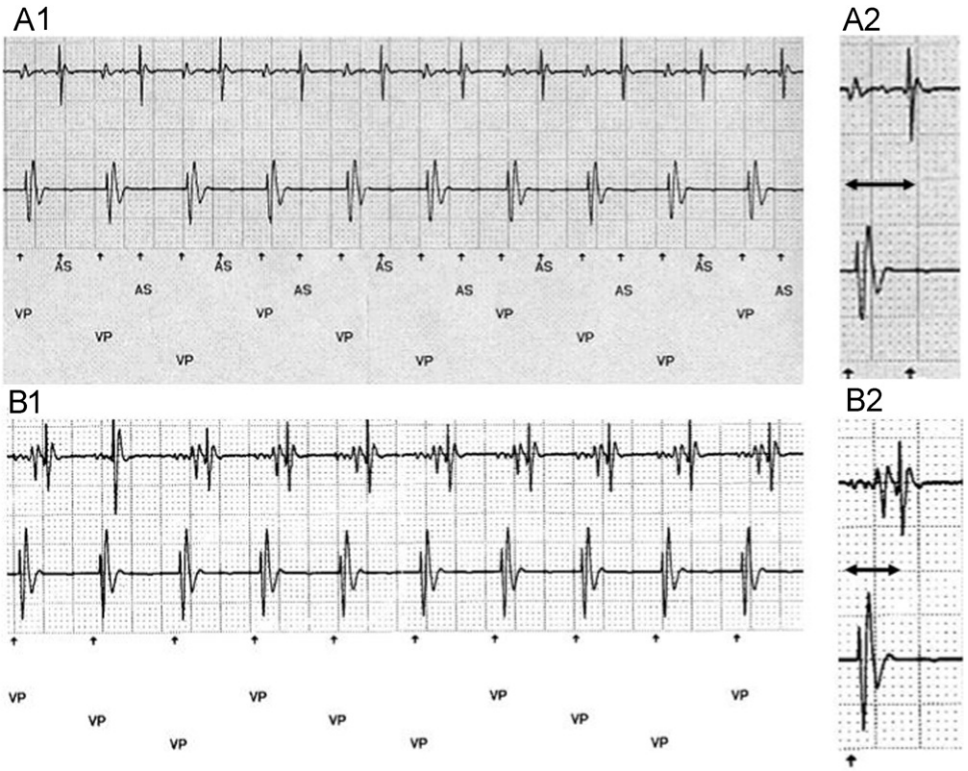

Fig. 2. (A-1) Pacemaker telemetry data during endless loop tachycardia. (B-1) Pacemaker telemetry data during rapid ventricular pacing at 110 bpm recorded after anti-arrhythmic drug exchange from amiodarone to aprindine hydrochloride. Endless loop tachycardia did not occur, even with the same programming parameters as before ablation. (A-2, B-2) Pacemaker telemetry data showed shortening of the ventriculoatrial conduction time during rapid ventricular pacing at 110 bpm after anti-arrhythmic drug exchange.

(Fig. 1A). Pacemaker telemetry data demonstrated A sense—V pace sequence during the tachycardia (Fig. 2A-1). The earliest site of atrial activation was the His bundle (HBE) recording site (Fig. 1). This tachycardia continued for 6 min until it was terminated by a premature atrial beat and was induced repeatedly. The differential diagnosis of this tachycardia included ectopic atrial tachycardia and pacemaker-mediated endless loop tachycardia (ELT). Tachycardia was terminated by reprogramming the pacemaker mode from DDD to VVI, after which rapid atrial activation also disappeared (Fig. 3). We diagnosed this arrhythmia as ELT, which was conducted to the ventricle via the pacemaker and retrogradely to the atrium via the AV node. ELT was also induced during a standard pacemaker lead threshold-check session after EPS and catheter ablation. Pacemaker parameters are

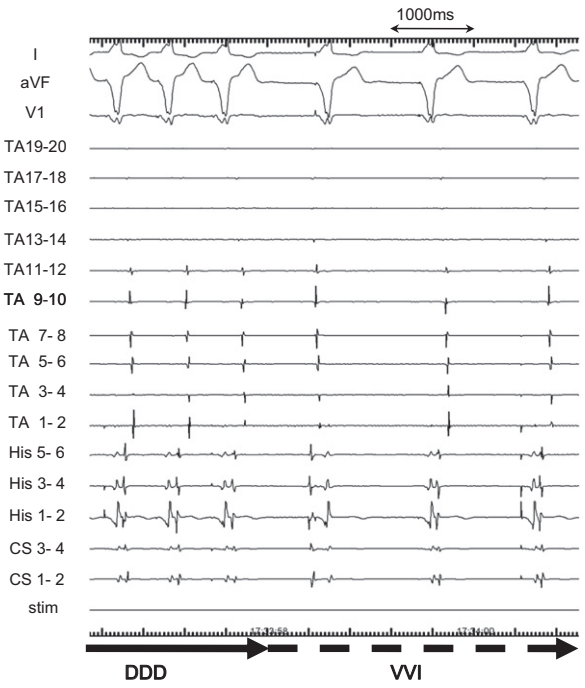

Fig. 3. Tachycardia was terminated by reprogramming the pacemaker mode from DDD to VVI; rapid atrial activation also disappeared.

Table 1
Programming data for the pacemaker. The left side shows data prior to ablation, and the right side represents data reprogramming after ablation.

Parameter	During ablation	Reprogramming after ablation
Mode	DDD	DDD
Lower rate limit (bpm)	60	60
Max tracking rate (bpm)	120	120
AV delay (paced) (ms)	300	220
AV delay (sensed) (ms)	270	190
PVARP (ms)	Dynamic PVARP	280
	Maximum 250	–
	Minimum 200	–
PVARP after PVC/PAC (ms)	400	400
PVAB (ms)	80	80
PMT termination	On	On
Atrial sensitivity (mV)	0.75	0.75
Ventricular sensitivity (mV)	2.5	2.5
A-Tachy response	On (DDI)	On (DDI)

AV=atrioventricular; PVARP=postventricular atrial refractory period; PVC=premature ventricular complex; PAC=premature atrial complex; PVAB=post ventricular atrial blanking interval; PMT=pacemaker mediated tachycardia.

listed in Table 1. Prior to this admission, the ECG of this patient always showed A pace—V sense sequence during routine pacemaker clinics. We have never detected an episode of AV block. The DDD pacemaker was programmed with long AVD (paced as AVD 300 ms, sensed AVD 270 ms), and dynamic post-ventricular atrial refractory period (PVARP) mode (maximum PVARP, 250 ms; minimum PVARP, 200 ms). Dynamic PVARP mode is a dynamic interval designed to provide a longer PVARP at slower rates to enhance protection against ELT, and a shorter PVARP to enhance atrial sensing at high rates. The upper tracking rate was 120 bpm. Pacemaker telemetry showed 250 ms of retrograde VA conduction time over programmed PVARP of approximately 208 ms at 110 bpm. We reprogrammed her pacemaker to a longer, fixed PVARP (280 ms), and a shorter AV delay (220 ms), to ensure an upper tracking rate of 120 bpm. After reprogramming the pacemaker, ELT no longer occurred. Two months later, amiodarone was discontinued because of appetite loss. We changed her prescription from 200 mg/day amiodarone to 20 mg/day aprindine hydrochloride to control paroxysmal atrial fibrillation (PAF). Pacemaker telemetry data showed shortening of the VA interval during rapid V-pacing at 110 bpm after the change in antiarrhythmic drugs (Fig. 2A-2, B-1, B-2). ELT was not induced after rapid ventricular pacing, even with the same programming parameters, before catheter ablation.

3. Discussion

ELT is a well-recognized complication of a dual chamber pacing system.[1,2] This is a wide QRS tachycardia resulting from the sensing of retrograde P-waves by the pacemaker, consequently triggering ventricular pacing beyond the programmed PVARP. Retrograde P-waves are induced by ectopic ventricular beats, ectopic atrial beats, atrial sensing failure, atrial pacing failure, and long AV delay.

The incidence of retrograde VA conduction was reported in 80% of SSS cases and in 35% of AV block cases [4]. Another study reported that the incidence of VA conduction was 32% in second degree AV block, and 14% in complete AV block [5]. Even in complete AV block, some patients who were implanted with a physiological pacemaker have a risk of ELT [6].

Thus, recent physiological pacemaker generators always have an algorithm to terminate ELT. ELT are generally maintained at the programmed upper tracking rate of the pacemaker. The generator in the current patient has an ELT detection algorithm. When 16 consecutive ventricular paces at the upper tracking rate are observed following atrial sensed events with the stability of the VA interval varying not more than 32 ms, this is detected as ELT. PVARP is then automatically extended to 500 ms for 1 cardiac cycle to avoid triggering of another ventricular event and break the ELT. In this case, ELT persisted despite the ELT termination algorithm in the generator. The pacemaker was functioning normally; however, the cycle length of this tachycardia was the sum of the intrinsic VA interval and the programmed sensed AVD, and the rate was 110 bpm—below the programmed upper tracking rate of 120 ms.

Several large clinical trials have revealed that a high percentage of ventricular pacing increases atrial fibrillation and the incidence of heart failure, thus leading to poor clinical outcome [7,8]. Recent clinical guidelines recommend the minimization of ventricular pacing in SSS patients. In some patients, prolonged pacemaker AVD is selected to preserve intrinsic ventricular beats. This patient was implanted with a DDD pacemaker because of SSS. Long AV delay (paced AVD of 300 ms, sensed AVD of 270 ms) was programmed to preserve intrinsic ventricular beats. During 100 ppm burst pacing from the coronary sinus ostium to confirm the block line of the isthmus, the Wenckebach phenomenon occurred. When the AV interval was prolonged relative to the programmed AVD, ventricular pacing began, and VA conduction beyond the atrial refractory period occurred, which initiated the ELT.

The programmed upper tracking rate is regulated by the sum of programmed AVD and PVARP. A low upper tracking rate causes a 2-to-1 AV block, or Wenckebach phenomenon, at a high sinus rhythm rate during exercise. The dynamic PVARP mode of this pacemaker is a dynamic interval designed to provide a longer PVARP at slower rates to enhance protection against ELT, and a shorter PVARP to enhance atrial sensing at higher rates. In this patient, dynamic PVARP mode (maximum PVARP, 250 ms; minimum PVARP, 200 ms) was selected to generate a higher upper tracking rate (120 bpm) just after generator replacement. The paced VA interval during ELT was approximately 250 ms, as revealed from pacemaker telemetry data. The ELT rate was 110 ppm and PVARP at this pacing rate with dynamic PVARP mode was 208 ms—below the paced VA interval. These reasons

explain why ELT continued despite a programmed ELT termination algorithm.

Prolonging PVARP to prevent sensing of the retrograde P wave is a well-known method to prevent ELT. We selected this method and succeeded in controlling the ELT. Prolonging PVARP affects the upper tracking rate of the pacemaker, which is determined by the sum of AV delay and PVARP. To ensure an adequate upper tracking rate with a long AV delay, the available width of the PVARP is limited. If ELT is to recur in this patient, and adjustment of the PVARP fails, reprogramming the pacemaker mode from DDD to DDI may be an effective option. In SSS patients, changing the pacing mode to DDI may be a simple and definitive strategy to avoid ELT.

The majority of anti-arrhythmic drugs cause block or depression of AV and VA conduction. Amiodarone has a strong effect of prolonging AV and VA conduction among anti-arrhythmic drugs [9]. Some reports suggested that anti-arrhythmic medication may be considered for patients with an ELT event to create a VA conduction block [9]. But in this case, introduction of amiodarone was the trigger of ELT initiation by prolonging VA conduction to the point that it fell beyond the PVARP. Replacement of the pacemaker generator was performed 7 years before this admission. Programming of the device was not changed from the time of generator exchange, but there was no episode of ELT before the initiation of amiodarone. After changing the patient's prescription from amiodarone to aprindine hydrochloride, her pacemaker telemetry data showed shortening of the VA interval during rapid V-pacing at 110 ppm (Fig. 2A-2, B-1 and B-2). ELT was not induced after rapid ventricular pacing, even with the same programming parameters as before catheter ablation.

In summary, programmed long AVD, prolonged VA conduction time caused by anti-arrhythmic medication, and the rate of ELT (110 bpm, below the upper tracking rate of 120 bpm) were the reason ELT continued even in the presence of a programmed ELT termination algorithm. Physicians who manage patients with DDD pacemakers should be aware that ELT does not always occur at the upper tracking rate and recognize the risk of ELT below the upper tracking rate in patients with programmed long AVD to avoid ventricular pacing. The ELT rate, which is determined by retrograde VA conduction time, may be affected by anti-arrhythmic drugs such as amiodarone.

Conflict of interest

All authors have no conflicts of interest to declare.

References

[1] Furman S, Fisher JD. Endless loop tachycardia in an AV universal (DDD) pacemaker. Pacing Clin Electrophysiol 1982;5:486–9.
[2] Bathen J, Gundersen T, Forfang K. Tachycardias related to atrial synchronous ventricular pacing. Pacing Clin Electrophysiol 1982;5:471–5.
[3] Ausubel K, Gabry MD, Klementowicz PT, et al. Pacemaker-mediated endless loop tachycardia at rates below the upper rate limit. Am J Cardiol 1988;61:465–7.
[4] Barold SS. Repetitive reentrant and non-reentrant ventriculoatrial synchrony in dual chamber pacing. Clin Cardiol 1991;14:754–63.
[5] Klementowicz P, Ausubel K, Furman S. The dynamic nature of ventriculoatrial conduction. Pacing Clin Electrophysiol 1986;9:1050–4.
[6] Duray GZ, Israel CW, Wegener FT, et al. Tachycardia after pacemaker implantation in a patient with complete atrioventricular block. Europace 2007;10:900–3.
[7] Lamas GA, Lee KL, Sweeney MO, et al. Ventricular pacing or dual-chamber pacing for sinus-node dysfunction. N Engl J Med 2002;346:1854–62.
[8] Wilkoff BL, Cook JR, Epstein AE, et al. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the dual chamber and VVI implantable defibrillator (DAVID) trial. JAMA 2002;288:3115–23.
[9] O'Mara JE, Fisher JD, Goldie SJ, et al. Effects of cardioactive medications on retrograde conduction: Continuing relevance for current devices. J Interv Card Electrophysiol 2006;15:49–55.