CASE REPORT

Tachycardia after pacemaker implantation in a patient with complete atrioventricular block

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The atrioventricular (AV) node allows ante- and retrograde conduction between atria and ventricles. It is commonly assumed that these AV nodal conduction properties go hand in hand. However, ante- and retrograde AV conduction can be completely independent from each other in individual patients. We report about a patient with permanent AV block III° requiring implantation of a pacemaker. As soon as a dual-chamber device was connected to the implanted leads, a tachycardia started at the maximum tracking rate, which was subsequently reprogrammed from 120 to 170 bpm. Non-invasive electrophysiologic testing showed that this patient demonstrated 1:1 ventriculoatrial (VA) conduction up to 170 bpm leading to endless loop tachycardia (ELT) while the antegrade AV block III° persisted. This case impressively illustrates that one has to take into account that patients with antegrade AV block III° may still have a high VA conduction capacity leading to ELT. Dual-chamber devices therefore have to be programmed accordingly, activating dedicated reactions after ventricular premature beats and automatic ELT detection and termination algorithms.

KEYWORDS
AV block; Retrograde conduction; Endless loop tachycardia; Pacemaker mediated tachycardia; Pacemaker circus movement tachycardia; Dual-chamber pacing

The atrioventricular (AV) node has several unique electrophysiological properties: it provides inverse rate-dependent conduction, antegrade and retrograde conduction, allows concealed conduction invisible from the surface ECG, may be functionally divided into slow and fast pathways, and is capable of escape rhythms, may show conduction block of different degrees and can sustain re-entrant tachycardia. The following case well illustrates the retrograde AV conduction behaviour in a patient with third degree AV block receiving a pacemaker.

Case presentation

A 63-year-old male was referred to our department with complete AV block, an atrial rate of ~95 bpm and a ventricular escape rhythm of ~20 bpm (Figure 1). The patient was haemodynamically compromised, acute myocardial ischaemia was excluded, and a temporary right ventricular pacing lead inserted. After transfer to the coronary care unit, echocardiography revealed hypertensive heart disease with a massive concentric left ventricular hypertrophy (interventricular septum 19 mm). After initial stabilization, a dual-chamber pacemaker was implanted (Identity ADx XL DR 5386, St Jude Medical, Sylmar, CA, USA) while the patient was still in complete AV block. Immediately after connecting the electrodes to the generator and inserting it into the pacemaker pocket, rapid regular ventricular pacing at a rate of 120 bpm ensued (Figure 2, programmed values see Table 1). After skin closure, the implanting physician reprogrammed the device (Table 1); however, the tachycardia continued to come and go and seemed to be initiated and stopped repeatedly by premature atrial beats (Figures 3 and 4). Upon increasing the maximum tracking rate, the tachycardia followed the upper rate up to 170 bpm (Figure 5).

The differential diagnosis of this tachycardia included sinus or ectopic atrial tachycardia (at the time of implantation, the patient was still on i.v. catecholamines), and pacemaker-mediated endless loop tachycardia (ELT). By reprogramming the pacemaker to DDI 30 bpm, frequent atrial premature beats were registered; however, no underlying sustained or non-sustained atrial tachycardia was present. On the other hand, programming the pacemaker to VVI 80–170 bpm showed 1:1 retrograde AV conduction with a ventriculoatrial (VA) interval of 270–290 ms. Therefore, the reproducibly documented tachycardia represented ELT caused by atrial premature beats, which were conducted to the ventricle via the pacemaker and retrogradely to the atrium via the AV node.
Discussion

ELT has been observed and understood early after the introduction of dual-chamber pacing.\textsuperscript{1–3} ELT necessarily requires retrograde AV nodal conduction. There is only scanty data on the incidence of intact retrograde conduction in patients undergoing pacemaker implantation. Although retrograde VA conduction was described in 80% of patients with sinus node disease vs. 35% in patients with AV block,\textsuperscript{4,5} another study reported retrograde VA block in 62% of patients with first degree AV block, 80% of patients with second degree AV block, and 100% of patients with third degree AV block.\textsuperscript{6} In infranodal AV block, second degree AV block was associated with 50% and third degree AV block with 85% retrograde VA block.\textsuperscript{6} At higher rates (140–160 bpm), retrograde conduction has been described in only 20% of patients at implantation.\textsuperscript{7}

However, antegrade AV and retrograde VA conduction can be totally independent from each other in individual patients: in the present case, permanent complete antegrade AV block with a ventricular escape rhythm of 20 bpm occurred together with a retrograde 1:1 VA conduction capacity of the AV node of up to 170 bpm. Since this high VA conduction capacity could have been associated with the use of i.v. catecholamines, assessment was repeated one week later when the patient was in stable condition and off any positive dromotropic drugs. There was still complete antegrade AV block associated with retrograde 1:1 VA conduction up to 170 bpm, the VA interval ranging from 274 to 297 ms.

The second interesting observation in this patient relates to the occurrence of ELT of different rates, ranging from 110 to 170 bpm (depending on the programmed upper tracking limit). This is in contrast to the widespread perception that ELT occurs always at an identical rate. The latter depends (as in AV junctional tachycardia) on the conduction velocity of the antegrade and retrograde limbs of the reentry circuit: the antegrade conduction time depends on the programmed sensed AV delay and the retrograde conduction time on the intrinsic VA interval. Intrinsic VA intervals ranging from 100 to 400 ms have been described\textsuperscript{4,8} and may be influenced by autonomic tone (retrograde conduction present during exercise, absent at rest).

Figure 1  ECG on admission. There is complete atrioventricular block with a broad-complex ventricular escape rhythm of <20 bpm and an atrial rate of ~95 bpm. Paper speed 25 mm/s.

Figure 2  Tachycardia after electrode connection and device implantation. Paper speed 25 mm/s.
anti-arrhythmic drugs, and by premature ventricular beats, which may facilitate conduction through the His–Purkinje system or AV node depending on their coupling interval.9

In our patient, however, there were only subtle changes in VA conduction times of $\pm$20 ms but the rate of the ELT varied between 110 and 170 bpm. Therefore, the rate change of the ELT was predominantly determined by the antegrade conduction. In fact, Figure 4 shows a sensed AV delay ranging from 203 to 219 ms, despite the fact that the sensed AV delay was programmed to 180 ms. This is caused by the fact that at this time, the upper tracking limit was programmed to 120 bpm and the device attempted to induce a Wenckebach behaviour by prolonging the AV delay. However, in ELT, the VA interval remains stable—unlike sinus tachycardia with AV block III where the P wave would successively approach the preceding ventricular event until it is no longer tracked. A dependency of the ELT rate from programming the upper tracking limit has been observed earlier.10

In Figure 3, ELT starts with a rate of 128 bpm (RR interval 470 ms) and accelerates to 170 bpm (353 ms), the programmed upper tracking limit. In parallel, the sensed AV interval decreases from 180 to 70 ms. This is caused by the rate-adaptive AV interval, which had been activated, and provides an AV interval of 180 ms at 60 bpm and a minimum sensed AV delay of 70 ms at the upper tracking limit.

There are several automatic pacemaker algorithms for detection and termination of ELT. Most ELT detection algorithms are activated if a certain number of cycles show atrial-sensed events at a pre-specified rate (e.g. upper tracking limit) or with a pre-specified VA interval (e.g., 400 ms). In this case, the post-ventricular atrial refractory period (PVARP) is extended (e.g. to 400 ms), thus interrupting the antegrade limb of the ELT. These algorithms have several weaknesses: they typically fail to distinguish sinus tachycardia (particularly with AV block I) from ELT and may report thousands of inappropriate ELT detections upon interrogation. Clinically even more important, they do not prevent the cause of ELT but can (at best) only terminate

![Figure 3](link) Initiation of tachycardia. An atrial premature beat starts a regular tachycardia with intrinsic atrial and paced ventricular events. In this case, a sensed atrioventricular delay of 180 ms (rate adaptive, minimum 70 ms) and an upper tracking limit of 170 bpm (353 ms) were programmed. Note that the atrioventricular delay is the same during sinus rhythm and the first cycles of ELT (172–179 ms), most likely due to concealed conduction to the atrioventricular node during sinus rhythm and complete antegrade block after the atrial premature beat. Paper speed 25 mm/s, simultaneous registration of lead II, marker annotations (P, atrial sensed event; V, ventricular paced event), cycle length annotations (AV delay, VA delay, and RR interval) in milliseconds, bipolar atrial electrogram, bipolar ventricular electrogram.

![Figure 4](link) Termination of tachycardia. An atrial premature beat occurs in the post-ventricular atrial refractory period (white P in black box, see arrow), is thus not tracked, and the tachycardia stops. In this case, the upper tracking limit was programmed to 120 bpm (sensed AV delay 180 ms). Legend as in Figure 3; A, atrial paced event.
the ELT after a minimum of 8–10 cycles. If patients have frequent triggers of ELT (e.g. atrial oversensing, atrial premature beats, non-capturing atrial stimuli, very long programmed AV delays) as in the present patient with frequent atrial premature beats, ELT may almost instantly start again. Atrial pacing within the myocardial refractory period (e.g. due to undersensing or shortly after a P wave in the atrial refractory or blanking period of the pacemaker) may cause asynchronous ventricular pacing with VA conduction, particularly if a long programmed AV delay opens the AV node for retrograde conduction. The effect is similar to an ELT but due to the lack of reentry termed 'repetitive non-reentrant VA synchrony'.

ELT detection algorithms may fail; e.g. in the case of variable or slow VA conduction, or due to interaction with other automatic features such as a rate-adaptation of PVARP or AV interval. Therefore, if ELT has been documented, the permanent setting should be programmed in order to avoid ELT: prevention of atrial oversensing, ensuring atrial capture, appropriate AV delay, upper tracking limit, and most importantly an appropriate PVARP that should be programmed either long or sensor-varied, combining a long PVARP at rest (e.g. 400 ms) with a shorter PVARP (e.g. 250 ms) if the sensor confirms exercise, thus permitting AV conduction of sinus tachycardia during exercise.

In the present case with frequent atrial premature beats as the cause of ELT, we programmed a lower rate limit of 80 bpm, which successfully suppressed atrial ectopy, and a PVARP of 350 ms.

**Conclusion**

This case illustrates three important clinical features:

(i) Antegrade and retrograde conduction through the AV node may occur completely independent from each other.

(ii) It remains unclear how many patients with AV block of different degree and different sites of block have retrograde VA conduction. In patients with complete AV block, retrograde conduction may lead to pacemaker syndrome in single- and to ELT in dual-chamber pacemakers. The presence of retrograde conduction should be considered and checked if symptoms are reported during pacemaker therapy.

(iii) In contrast to the common belief, ELT rate depends on the patient’s VA interval and the AV delay of the pacemaker that may depend on the pacing rate and that may be extended by the programmed upper tracking rate. Dedicated algorithms for ELT detection need to be optimized in the case of new-onset tachycardias after pacemaker implantation.

**Conflict of interest:** C.W.I. is currently conducting research sponsored by Medtronic Inc. and St. Jude Medical Inc. He is member of the advisory board and speaker’s bureau for Boston Scientific, Redfronc Inc., Sorin Group and St. Jude Medical. S.H.H. is member of the speaker’s bureau of St. Jude Medical. He is currently conducting research sponsored by Medtronic Inc and St. Jude Medical. G.Z.D. and F.T.W. have no conflicting economic interests to declare.

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