Pacemaker reprogramming

A wide-complex tachycardia in a 90-year-old man with a dual-chamber pacemaker

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Case presentation

A 90-year-old man with ischaemic heart disease with preserved ejection fraction, paroxysmal atrial flutter and sinus sick syndrome requiring dual-chamber pacemaker implantation 2 years earlier (Sorin KORA 250 DR, atrial lead VEGA R52, ventricular lead VEGA R58; stimulation mode AAIR-DDDR, lower rate limit 60 beats/min, maximum tracking rate 130 beats/min), was brought to the emergency department because of ongoing palpitations for the past 10 days. His medication included rivaroxaban, amiodarone, bisoprolol and torasemide. Vital parameters showed a heart rate of 130 beats/min, blood pressure of 114/89 mm Hg, SpO_2 of 95% and tympanic temperature of 36.0°C. The patient was in moderate distress. The rest of the physical examination, including chest and lung auscultation, was unremarkable. A 12-lead electrocardiogram (ECG) was obtained (fig. 1). High sensitivity cardiac troponin T was 52 ng/l (normal value: <14 ng/l) without any significant change on repeat measurement. Electrolyte levels and thyroid-stimulating hormone value were within normal range.

What is your diagnosis? What would be your next step in managing this patient?

Solution

The ventricular rate is regular at a rate of about 130 beats/min. No evident P waves can be spotted. QRS width is about 160 ms with left axis deviation (QRS axis –60°). A QS pattern in the precordial leads V1–V2, with late transition to an RS pattern in V6, give a left bundle branch block (LBBB) morphology. There is physiological ST segment discordance with the QRS, in the setting of LBBB. T waves appear peaked in the precor-

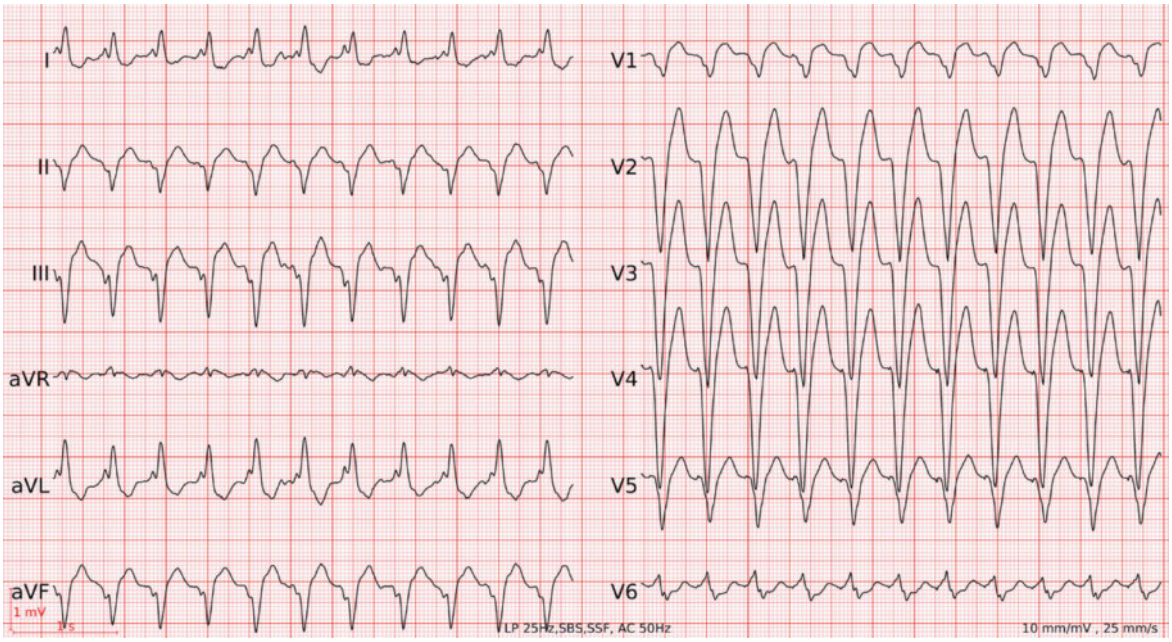

Figure 1: 12-lead ECG obtained at admission.

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dial leads with a corrected QT value of 447 ms according to Bazett's formula [1]. In summary, there is a regular wide complex tachycardia at a rate of 130 beats/min without evidence of sinus node activity.

Differential diagnosis includes ventricular tachycardia and supraventricular tachycardia with aberration. The threshold to suspect ventricular tachycardia should be particularly low, especially in this patient with ischaemic heart disease. Advanced algorithms to differentiate ventricular tachycardia from supraventricular tachycardia (i.e., Brugada algorithm, Vereckei algorithm) exist, but their use in the emergency department and by non-cardiologists may be difficult. Nevertheless, there is no evidence of fusion complex, capture complex, atrioventricular (AV) dissociation, concordance in the precordial leads or extreme right axis deviation, to support the diagnosis of ventricular tachycardia.

Importantly, in the presence of LBBB-like QRS complexes in patients with pacemakers, right-ventricular pacing should be suspected. The absence of pacing spikes does not rule out pacemaker stimulation [2]. The ECG shown in figure 2 was obtained after activating the pacemaker enhancement function of the computerised ECG reading, where a pacing spike preceding QRS is now evident. Therefore, in the setting of an inappropriately elevated heart rate, at or below maximum tracking rate, a pacemaker-mediated tachycardia should be considered.

Pacemaker-mediated tachycardia concerns patients with dual-chamber pacemakerss, with the classical form being endless loop tachycardia with a reentry mechanism [3]. In the presence of retrograde AV nodal conduction, retrograde P waves are tracked and thus lead to ventricular stimulation mostly at maximum tracking rate. The pacemaker is therefore considered the anterograde limb of the reentry circuit. Appropriate pacemaker programming and specific algorithms usually prevent endless loop tachycardia.

In the setting of suspected pacemaker-mediated tachycardia, the most appropriate next step would be to place a magnet on the pacemaker generator. This has the advantage of being easily accessible to non-cardiologists and of being both diagnostic and curative for a pacemaker-mediated tachycardia. Most pacemakers are nominally programmed to be "magnet responsive" and placing a magnet over the pacemaker generator will convert the programmed pacing mode to an asynchronous one (AOO or VOO in single-chamber pacemakers, depending on the functioning lead; DOO in a dual-chamber one). The pacing rate may vary between 85 and 100 beats/min depending on the manufacturer [4]. Importantly, placing a magnet on the generator of an implantable cardioverter defibrillator will deactivate only the anti-tachycardia therapy function, without any modification of pacing.

Figure 3 shows the ECG obtained following placement and removal of a magnet from the pacemaker generator. An atrial activity at about 260 beats/min is now visible (red arrows), corresponding to flutter waves. The ventricular activity has become slower and irregular, at about 84 beats/min. Spontaneous ventricular ac-

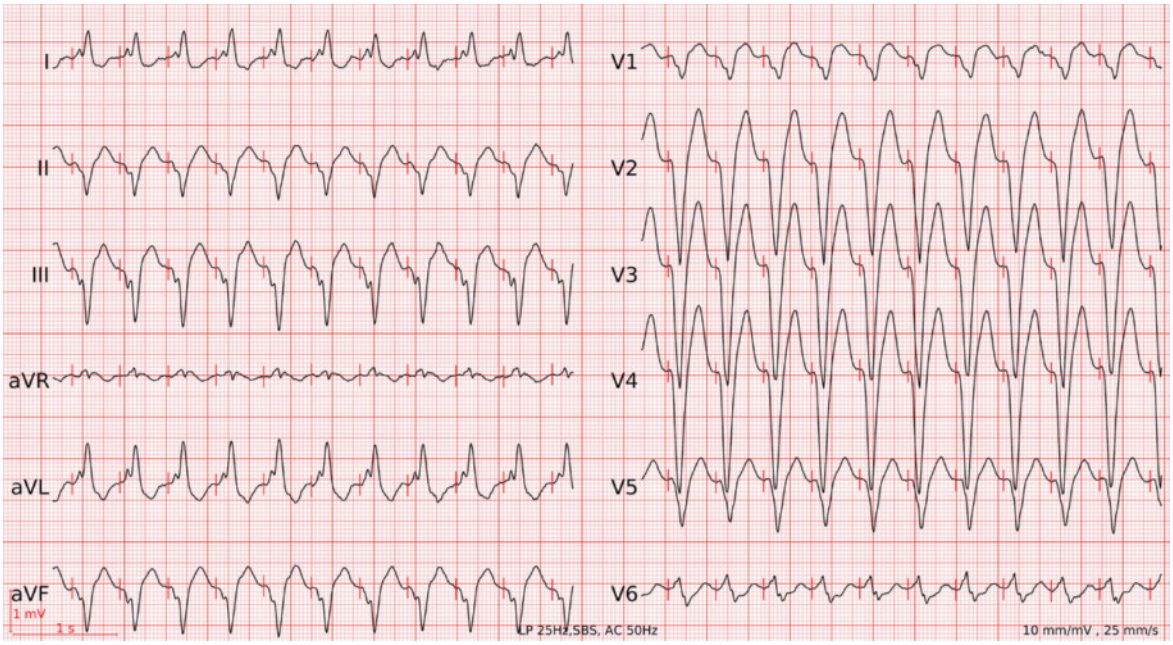

Figure 2: 12-lead ECG obtained after turning on the pacemaker enhancement function of the computerised ECG reading. A pacing spike preceding each QRS complex is visible.

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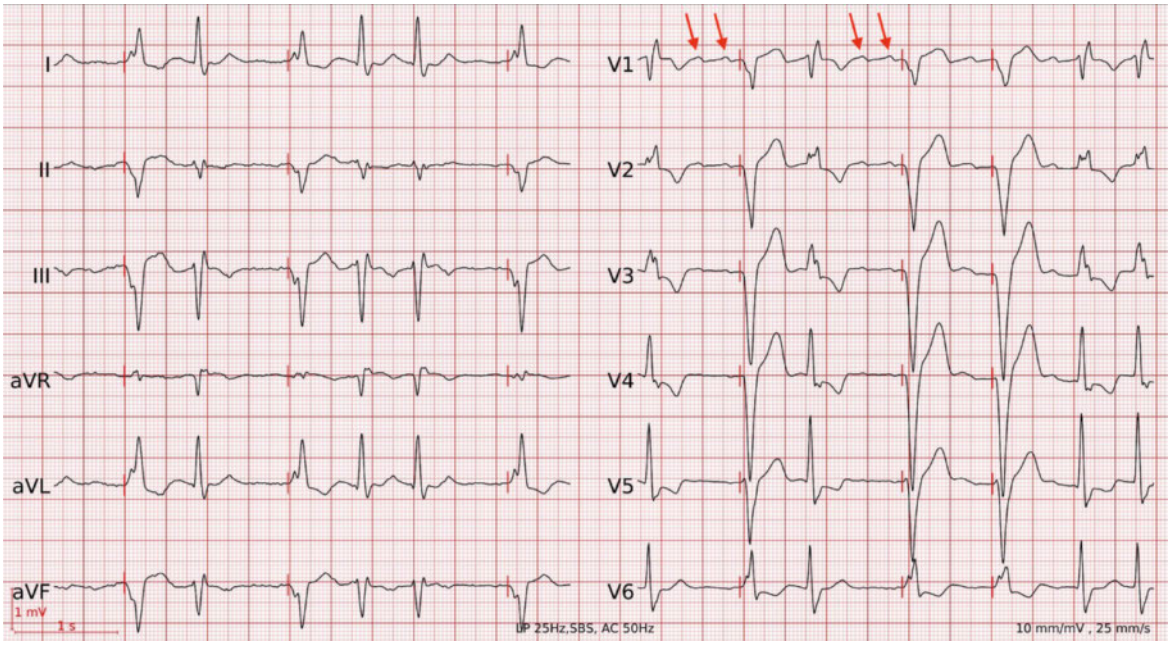

Figure 3: 12-lead ECG obtained after placement and removal of magnet from the pacemaker generator. Atrial activity at about 260 beats/min is visible (red arrows). There is alternation between spontaneous, AV-conducted ventricular activity with left anterior fascicular block and right bundle branch block, and right-ventricular stimulation denoted by LBBB morphology, at a rate of about 84 beats/min.

tivity shows right bundle branch block QRS morphology with left axis deviation in the setting of concomitant left anterior fascicular block. AV conduction is irregular, paced ventricular complexes (LBBB morphology, preceding pacing spike) are also present.

Figure 4 shows the ECG obtained following pacemaker reprogramming by the cardiologist, who confirmed an ongoing atrial flutter on pacemaker analysis. The ECG shows an atrial flutter with approximately 2:1 AV conduction at 107 beats/min with and left anterior fascicu-

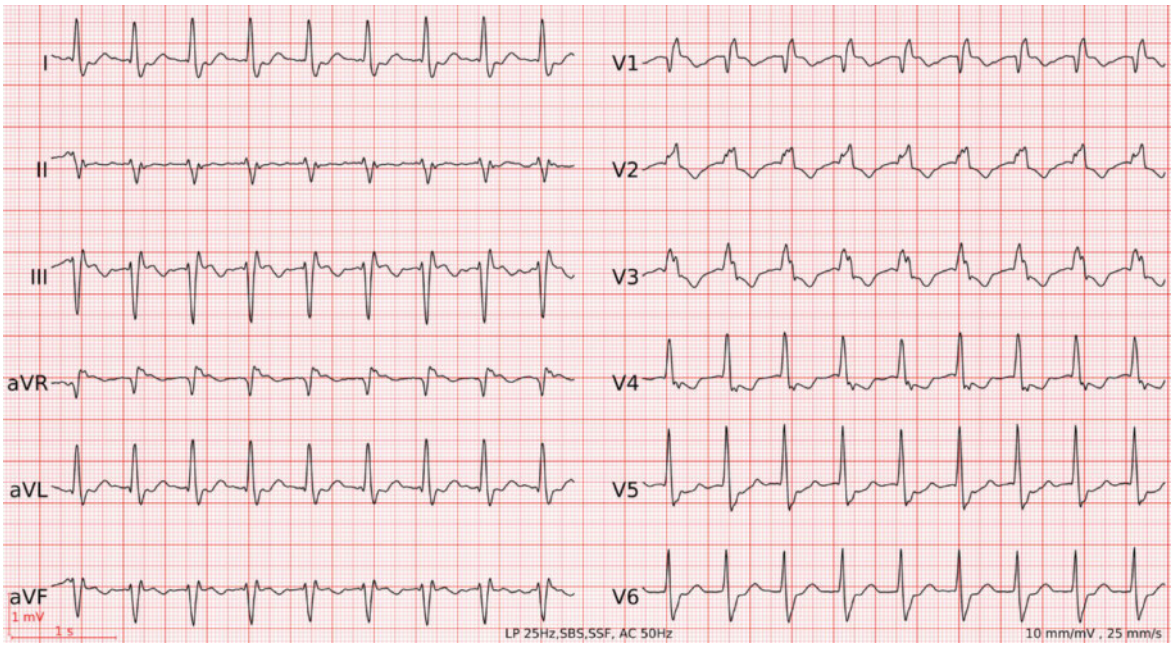

Figure 4: Final 12-lead ECG obtained after pacemaker adjustments were made. There is a regular wide-complex (right bundle branch block and left anterior fascicular block morphology) tachycardia at 107 beats/min, confirmed to be an atrial flutter with approximately 2:1 conduction on pacemaker analysis.

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lar block. Atrial overdrive using the pacemaker was unsuccessful in converting the atrial flutter.

Final diagnosis was a "2:1 lock-in" phenomenon with ventricular tracking at 130 beats/min, in the setting of atrial flutter. This is a particular form of pacemaker-mediated tachycardia in atrial flutter, with failure to mode switch. Automatic mode switch is a programmable function in most dual-chamber pacemakers, which prevents tracking of rapid atrial activity above a prespecified rate is detected by converting pacing mode to DDI(R) and avoiding rapid ventricular stimulation [5]. At a specific rate, the atrial signal may fall in the post-ventricular atrial blanking period (PVAB) and be undetected by the pacemaker. Thus, the pacemaker will only sense every other atrial signal (2:1), falsely interpreting the rhythm as sinus tachycardia, as illustrated in figure 5, resulting in ventricular tracking at half the atrial rate.

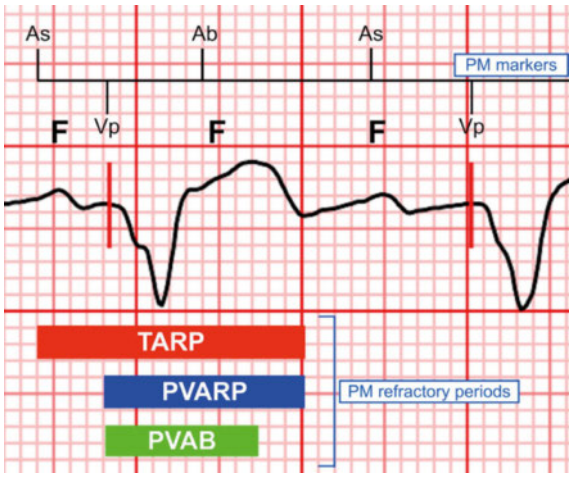

Figure 5: "2:1 lock-in" in atrial flutter. PVAB: post-ventricular atrial blanking period; PVARP: post-ventricular atrial refractory period; TARP: total atrial refractory period; As: atrial sensed event; Ab: atrial sense in PVAB; Vp: ventricular paced event; F: flutter wave. An atrial event (As) is, per definition, tracked and leads to a ventricular stimulus (Vp) in the absence of a spontaneous ventricular event following a defined atrio-ventricular (AV) delay. In the setting of , the second flutter wave (F) may fall in the PVAB, where it would be "blanked" (Ab) and not sensed by the pacemaker. This flutter wave cannot lead to spontaneous ventricular activity, even if AV conduction is preserved, as the ventricle is refractory at that moment. The pacemaker then detects the next flutter wave and the cycle repeats itself at a ventricular paced rate, which would be half the flutter rate. An atrial event falling in the PVARP, outside the PVAB, will be sensed and counted (i.e., for the purpose of mode switch) but ton tracked. TARP is the sum of the AV interval and PVARP and limits the maximum tracking rate of a pacemaker.

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For the latter to occur, programmed maximum tracking rate must be ≥ atrial rate/2. Some pacemakers have dedicated algorithms to automatically detect "2:1 lock-in" and counter it [6].

To avoid "2:1 lock-in" recurrence, parameters such as timing cycle (AV delay and PVAB), maximum tracking rate and/or tachycardia detection rate need to be modified. In our case, mode switch failure was favoured by intermittent underdetection of flutter waves. The atrial electrode's sensitivity was indeed programmed too high, hindering appropriate recognition of abnormal atrial activity. The sole reduction of atrial sensitivity threshold (the pacemaker was made more sensitive to atrial activity) was sufficient to allow adequate mode switch.

Interestingly, an increase in heart rate can be noted between the ECG obtained after magnet removal (fig. 3) and the one obtained after pacemaker reprogramming (fig. 4). This is in fact independent of programming and denotes variable AV conduction in this patient under bisoprolol and amiodarone treatment.

Following the above mentioned pacemaker reprogramming, the patient could be discharged under his usual medication regimen and was doing well at 1-month follow-up.

Disclosure statement
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