

# Broad complex tachycardia in a patient with a pacemaker: What is the mechanism?



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## Introduction

Permanent pacemakers have the capacity to sense, detect, and store tachyarrhythmia episodes, providing clinicians with a valuable diagnostic tool that can be utilized for decision making and patient management. Algorithms predominantly use rate and regularity for detection, with electrograms (EGMs) helping in discrimination between ventricular (VT) and supraventricular tachycardias (SVT).<sup>1</sup> The case we present seeks to highlight a potentially common pitfall that should be recognized, owing to its clinical implications.

## Case report

A 75-year-old woman presented to a rural hospital with syncope. A dual-chamber pacemaker had been implanted for impaired atrioventricular (AV) nodal conduction following aortic valve replacement for infective endocarditis 15 years prior. On arrival to the emergency department, the patient was in a state of cardiovascular collapse with a broad complex tachycardia with a rate exceeding 200 beats per minute (bpm) (Figure 1A). She was promptly electrically cardioverted with a single 150-joule synchronized shock. A 12-lead electrocardiogram (ECG) was not performed prior to cardioversion.

The pulse generator had been replaced 6 years before presentation because of battery depletion. The current device was a Versa VEDR01 (Medtronic Inc, Minneapolis, MN), with Guidant (Indianapolis, IN) atrial (4480) and ventricular (4457) bipolar leads. The pacemaker was programmed to DDD mode, with a lower rate of 60 bpm, atrial sensitivity of 0.3 mV, and atrial high rate (AHR)/mode switch detection rate of 150 bpm. Device parameters had been stable on annual interrogations.

Following cardioversion, the ECG showed a paced atrial rhythm with first-degree AV block, right bundle branch block, and left anterior fascicular block. Pacemaker interro-

gation showed P waves of 2.0 mV and R waves of 8.0 mV with satisfactory threshold and impedance measurements. The clinical episode was logged as a ventricular high rate (VHR) event, with the ventricular EGM (VEGM) rate exceeding that of the atrial EGMs (AEGM) (Figure 1B). Transthoracic echocardiography demonstrated normal left ventricular size with moderate impairment of systolic function and a normally functioning aortic valve prosthesis. Given the findings above, what is the most likely tachycardia mechanism?

## Discussion

The differential diagnoses in this circumstance include the following:<sup>2</sup>

- VT, with:
  - Ventriculoatrial (VA) block
  - 1:1 VA conduction and true or functional atrial undersensing
- Rarer possibilities, such as atrial (or upper common pathway) block in association with:
  - AV nodal reentrant tachycardia
  - Junctional tachycardia
  - Intra-hisian reentrant tachycardia<sup>3,4</sup>
  - Reentrant tachycardia utilizing a concealed nodoventricular or nodofascicular pathway
- Atrial tachycardia or flutter with atrial undersensing.

A cardiac electrophysiology study was performed. Apart from an HV interval of 65 ms, AV and VA conduction were normal and via the normal conduction system. Rapid atrial pacing induced a broad complex arrhythmia with 1:1 AV association. The cycle length was 315 ms and His bundle potentials preceded each ventricular depolarization (Figure 2A). Administration of adenosine (18 mg) during tachycardia resulted in AV block with no change in the atrial cycle length (Figure 2B).

Concurrent device interrogation with both intracardiac EGMs and marker channels activated showed AEGMs and VEGMs in a 1:1 ratio, but most atrial events were not displayed on the marker channel because they fell within the post-ventricular atrial blanking (PVAB) period. A peculiarity

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**KEY TEACHING POINTS**

- Functional atrial undersensing may be part of “normal” behavior in older dual-chamber devices and may confound discrimination between supraventricular and ventricular rhythms, with detected atrial activity falling within post-ventricular atrial blanking periods.
- Physicians should be aware of this phenomenon and be vigilant in patients with a history of atrial arrhythmias.
- If suspicion arises, the post-ventricular atrial blanking period should be shortened and the stored “EGM type” should be changed to “AEGM” to improve detection and discrimination.

of this particular pacemaker is that it does not notate events falling within the PVAB. Occasional atrial events fell within the post-ventricular atrial refractory period and were marked as an atrial refractory event (Figure 3A). These findings were consistent with an atrial tachycardia, with functional undersensing of atrial activity.

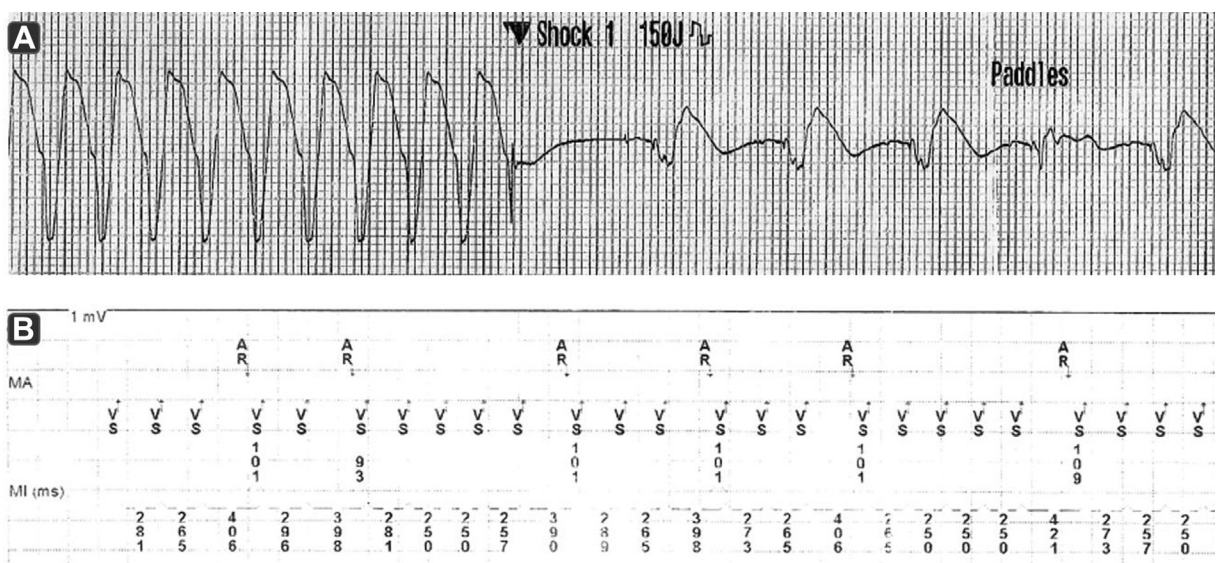
Multiple different atrial arrhythmias with atrial rates of 140–220 bpm were subsequently induced with rapid atrial stimulation at baseline and following isoproterenol administration. Ventricular arrhythmias were not induced with rapid ventricular stimulation from the right ventricular apex or with programmed stimulation with up to 4 extra-stimuli in the baseline state or following isoproterenol administration.

Conservative management with oral metoprolol was employed in the first instance. The PVAB was shortened to 100 ms (from 180 ms) in order to improve arrhythmia discrimination. The post-ventricular atrial refractory period was left unchanged (250 ms). AEGM was selected as preferred “EGM type” for recording of both AHR and VHR episodes, having previously only had the marker channel activated. Over the ensuing months, the patient continued to have recurrent atrial arrhythmias with rapid ventricular response despite optimized medical therapy, and AV node ablation was subsequently performed. She has remained well since.

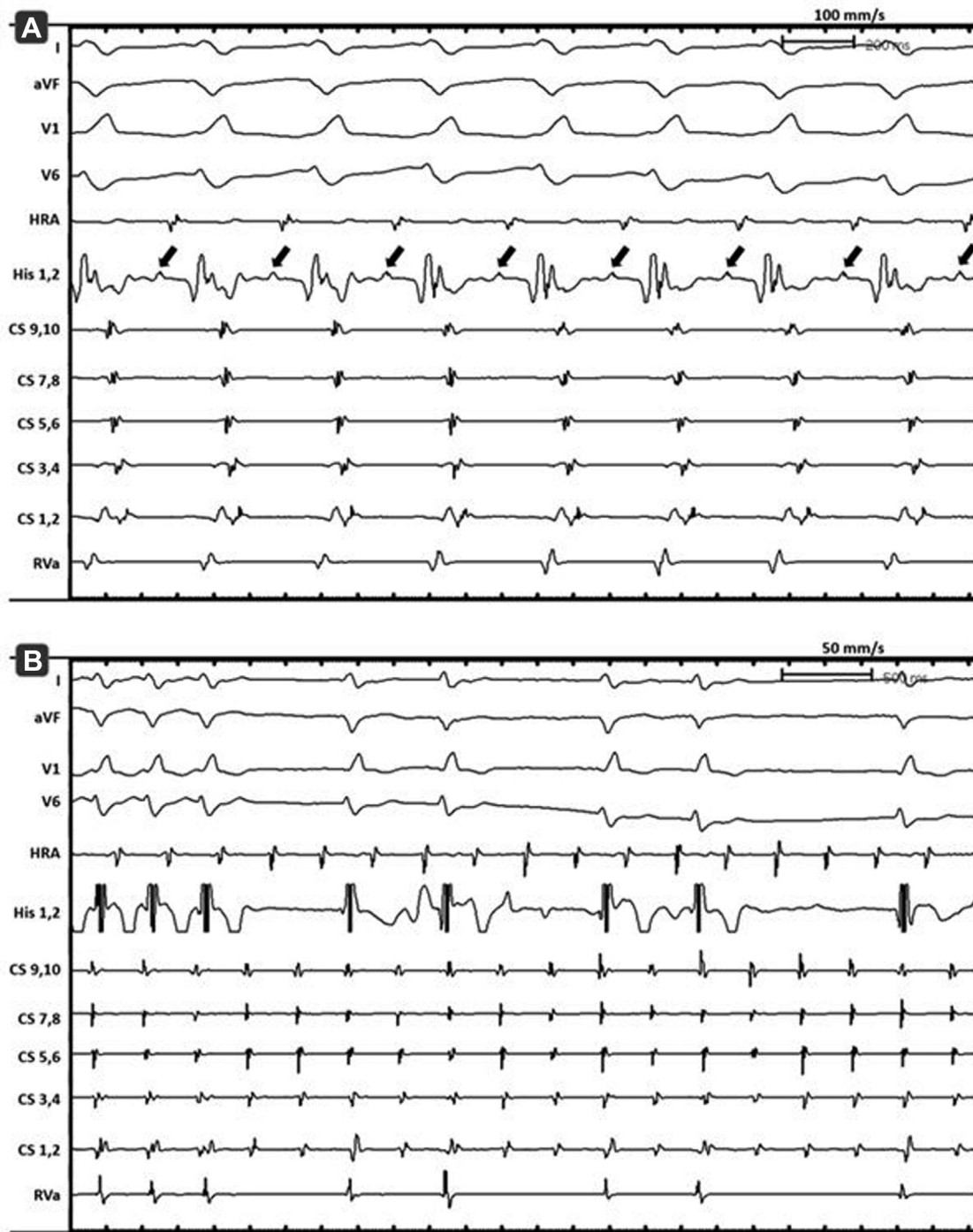
This case highlights a situation in which a device-recorded ventricular rate appeared to exceed the atrial rate, in association with a hemodynamically compromising broad complex tachycardia.

The predominant theme from the differential diagnosis list above is the atrium not being an obligatory part of the tachycardia. In our case, the converse was true. Multiple atrial tachycardias were induced during the electrophysiology study with the atrial rhythm unperturbed following administration of adenosine and the ventricle shown not to participate in the tachycardia.

While the clinical tachycardia was not captured on a 12-lead ECG, the marker channel recording demonstrated a similar pattern to the strip recorded by the device during the initial clinical event. AEGMs were clearly present during tachycardia, as illustrated in Figure 3. They were of an adequate amplitude above the programmed sensitivity, but the activity fell predominantly within the PVAB period and, as such, was not properly sensed by the device. This resulted in the tachycardia being classified as a ventricular, rather than atrial, high rate episode. The suspicion that the original rhythm was supraventricular, despite AEGMs not



**Figure 1** A: Trace from the external defibrillator demonstrating a regular tachycardia of over 200 beats per minute, and successful 150 joule synchronized shock. B: Recording from the pacemaker of the clinical arrhythmia. No electrograms from the episode were stored or were available on interrogation. The strip demonstrates ventricular events exceeding atrial rates, suggesting a ventricular tachycardia. The atrial marker channels showed intermittent atrial events, detected within the refractory period. Note: Panels A and B are not on the same scale and are not concurrent.



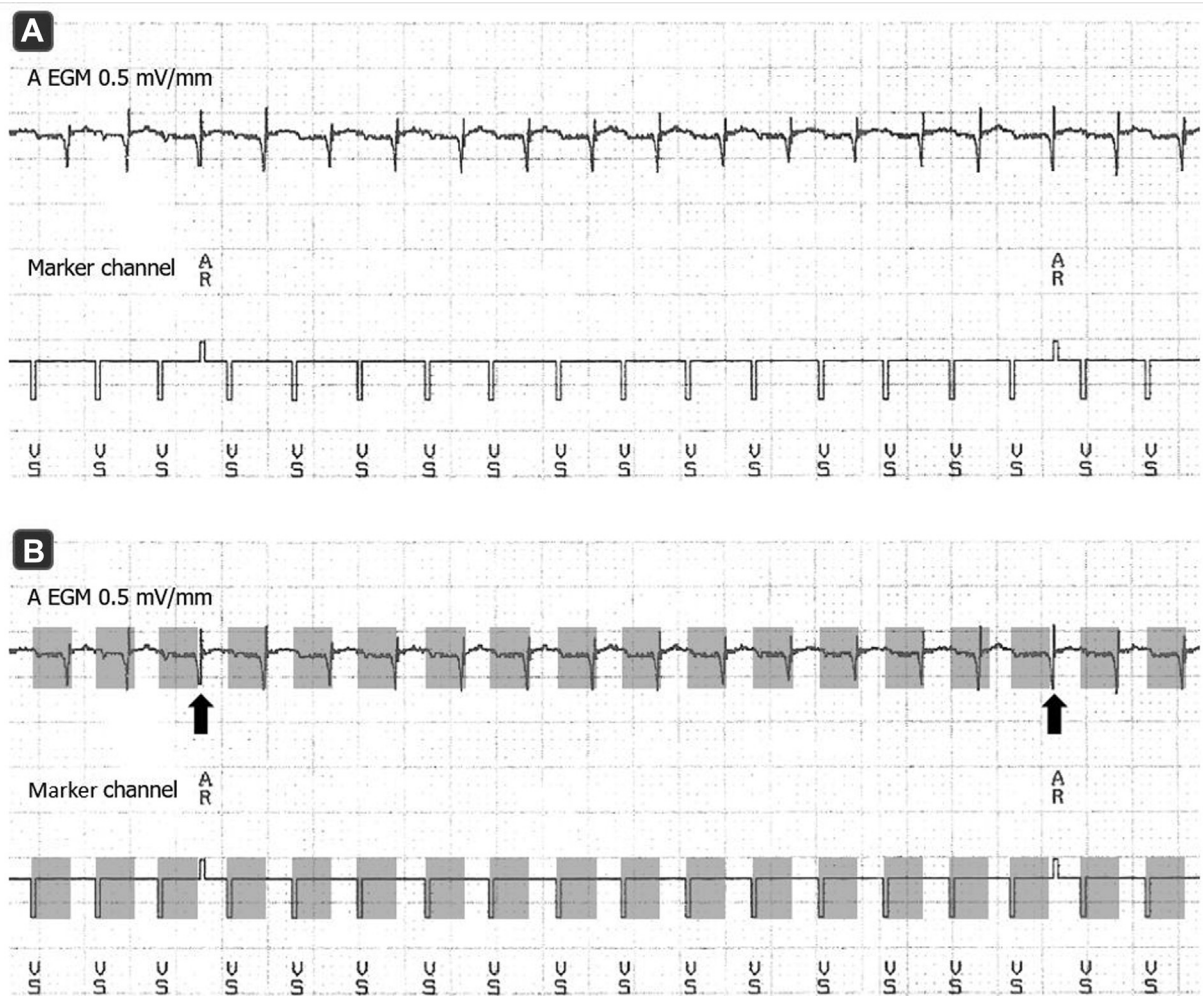
**Figure 2** A: Intracardiac electrograms (EGMs) from high right atrial (HRA), His bundle, coronary sinus (CS), and right ventricular apex (RVa) catheters. Broad complex tachycardia with cycle length of 315 ms, with His bundle EGMs (arrow) preceding ventricular EGMs and HV interval of 65 ms. B: Intracardiac EGMs following administration of adenosine 18 mg, demonstrating atrioventricular dissociation with no perturbation of atrial rate.

being available, was raised as perturbation in the ventricular intervals appeared to unmask atrial refractory events on the atrial marker channel.

This was an example of functional atrial undersensing, where atrial signals are “detected” but “unsensed” because they fall within a blanking or refractory period.<sup>5</sup> This con-

trasts with the more common form of undersensing, when the signal amplitudes fall below the programmed device sensitivity.

As illustrated in this case, functional atrial undersensing may confound discrimination between SVT and VT, and result in the following:



**Figure 3** **A:** Device interrogation corresponding to Figure 2A at sweep speed of 25 mm/s with atrial electrograms (EGMs) displayed in addition to the marker channel, demonstrating 1:1 atrioventricular association but with no atrial sensed events and only 2 atrial refractory events displayed on the marker channel. The amplitude of atrial EGMs exceeded the programmed sensitivity of 0.5 mV, but events were not recorded because they fell within the post-ventricular atrial blanking (PVAB) period. **B:** Corresponding to Figure 3A, shaded boxes denote the PVAB period, with atrial events in the “A EGM” line marked AR (labeled with arrows) falling just outside the PVAB window.

- Misinterpretation of an SVT as a VT, which clinically may lead to:
  - Inappropriate referrals for (and insertion of) implantable cardioverter-defibrillators (ICDs) for secondary prevention, particularly if there are preceding indications for primary prevention
  - Inappropriate therapy being delivered by ICDs<sup>6</sup>
- Underdetection of atrial tachyarrhythmias that may warrant rhythm management as well as anticoagulation for thromboembolic prophylaxis.

The issue described in this case is more evident in older devices, in which the marker channel may be the only recording strip available, or if EGMs are not turned on during device programming for high rate episode recordings. Crucially, it should be recognized that atrial activity within blanking periods may not be recorded on the marker channel.

In devices from the Medtronic Versa range, EGM storage has to be switched on, specifying high rate type (AHR, VHR, or both) and the EGM type (AEGM, VEGM, or summed EGM) to be collected.<sup>6</sup> In cases like these, AEGM or summed EGM should be used as preferred EGM type for recording high rate episodes, rather than the marker channel alone. Should a suspected episode of undersensing occur, the PVAB should be shortened to improve sensing of atrial activity. Fortunately, ICDs have more sophisticated discrimination algorithms to prevent inappropriate therapy in similar circumstances.

Several other limitations of the device beyond the blanked AEGMs were raised.

First, storage space for EGM collection is restricted to 4 episodes, and if both AHR and VHR episodes are specified for collection, potentially only 2 of each will be recorded.<sup>7</sup>

Second, if EGM data are being collected for an AHR or VHR episode while an episode of the other type occurs, no EGM data are collected for the second episode.<sup>7</sup> This would appear to have been a likely reason EGMs were not available in our patient. When the atrial arrhythmia started, atrial rates exceeding the mode switch rate of 150 bpm would have triggered recording of an AHR and EGMs of this would have been collected by the device. This, however, would have precluded collection of EGMs of the VHR that “followed,” when the rate exceeded the ventricular detection rate of 180 bpm.

Finally, episodes from previous interrogations are not available on subsequent interrogations and, as such, would be dependent upon the operator of the programmer at the time to print the data for a specific arrhythmia episode, failing which, the data are no longer retrievable. In this case, it led to the inability to examine the episode shown in [Figure 1B](#) during the interrogation at our center, and reliance solely upon the printout that was provided.

## Conclusion

Functional atrial undersensing may occur as a result of “normal” pacemaker or ICD behavior. It should be identified

as a possible confounder in the recognition and discrimination of SVT and VT, particularly in patients with a history of atrial arrhythmias with older-generation devices. Shortening blanking periods and altering EGM storage properties can improve the detection, accuracy, and utility of stored tachyarrhythmia events.

## References

1. Gillberg J. Detection of cardiac tachyarrhythmias in implantable devices. *J Electrocardiol* 2007;40:S123–128.
2. Kose S, Amasyali B, Aytemir K, Kilic A, Iyisoy A, Celik T, Hurkan K, Isik E. Ventricular block during atrioventricular nodal reentrant tachycardia suggesting existence of an upper common pathway. *Int Heart J* 2005;46:333–338.
3. Lau EW. Infraatrial supraventricular tachycardias: mechanisms, diagnosis, and management. *Pacing Clin Electrophysiol* 2008;31:490–498.
4. Morady F. Ventriculoatrial Block During a Narrow-QRS Tachycardia: What Is the Tachycardia Mechanism? — IV. *J Cardiovasc Electrophysiol* 1996;7:174–177.
5. Barold SS, Van Heuverswyn F, Timmers L, Stroobandt RX. Functional atrial undersensing associated with switching to a tracking mode of pacing. *Pacing Clin Electrophysiol* 2012;35:1188–1193.
6. Barold SS, Kucher A, Menne J, Stroobandt RX. Electrical Atrial Alternans Recorded by Cardiac Rhythm Devices during Atrial Flutter. *Pacing Clin Electrophysiol* 2015;38:1231–1235.
7. Medtronic Inc. ADAPTA / VERSA / SENSIA / RELIA - A family of implantable pulse generators: Pacemaker Reference Guide [Internet]. 2016 (cited 10 November 2017). Available at [http://manuals.medtronic.com/content/dam/emanuals/crdm/CONTRIB\\_244793.pdf](http://manuals.medtronic.com/content/dam/emanuals/crdm/CONTRIB_244793.pdf).