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).[1] 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 interrogation 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:[2]

- 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[3,4]
  - 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

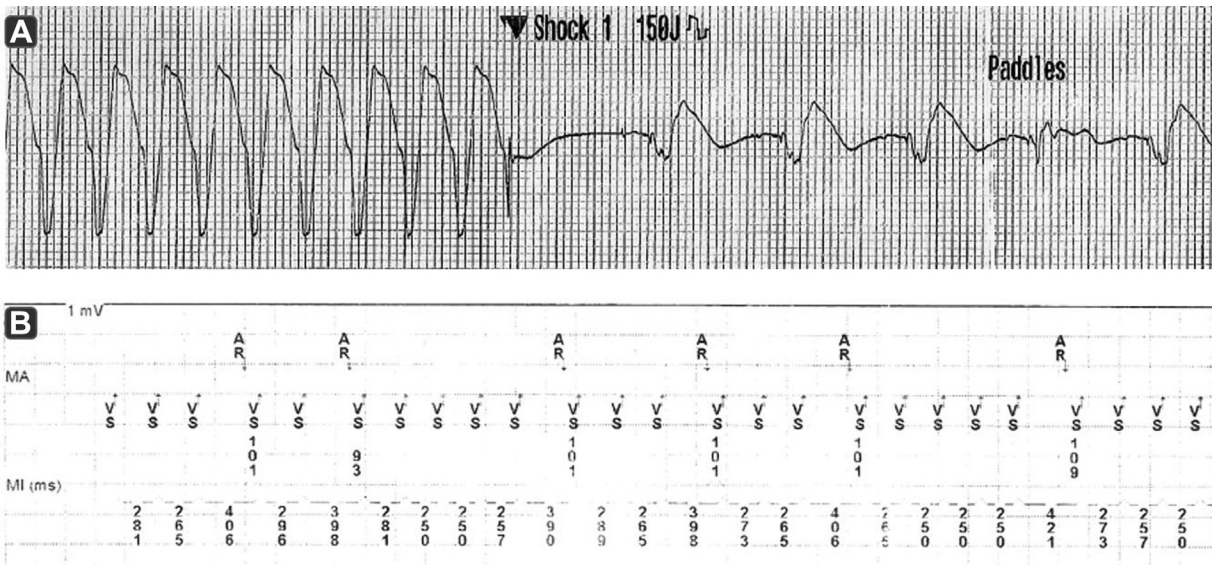

Figure 1 **A:** Trace from the external defibrillator demonstrating a regular tachycardia of over 200 beats per minute, and successful 150 joule synchronized shock. **B:** Recording from the pacemaker of the clinical arrhythmia. No electrograms from the episode were stored or were available on interrogation. 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.