

Tachycardia in the Presence of Ventricular Pacing

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A 61-year-old man with coronary artery disease presented with volume overload caused by medication noncompliance. His medical history included percutaneous coronary intervention; ischemic cardiomyopathy (left ventricular [LV] ejection fraction, <0.15); and placement of a biventricular implantable cardioverter-defibrillator (ICD), model and programming unknown. Chest radiographs showed properly positioned ventricular and right atrial leads. Figure 1 shows the patient's electrocardiogram (ECG) on presentation.

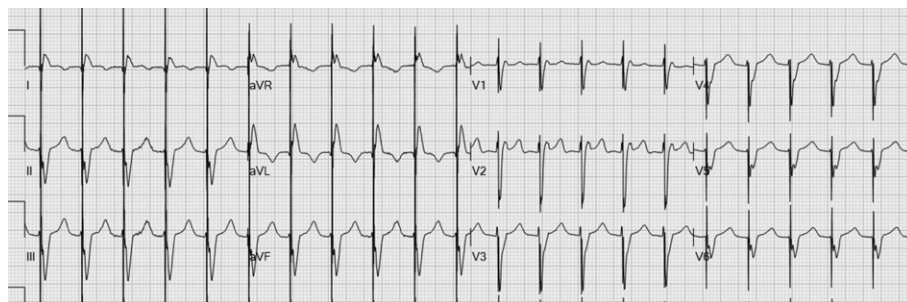


Fig. 1

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On the basis of the ECG, what is the patient's diagnosis?

- A) Supraventricular tachycardia with biventricular pacing response
- B) Pacemaker-mediated tachycardia
- C) Sinus tachycardia with normal atrioventricular delay programming
- D) Antitachycardia pacing

See next page for the answer, as well as a link to the Focus on ECGs blog, where you can participate in a moderated discussion.

FOCUS ON ECGs: ANSWER #17

Answer

A) Supraventricular tachycardia with biventricular pacing response.

The ECG shows a relatively narrow QRS complex at a ventricular rate of 100 beats/min. Notably, an rSR' configuration in leads V₁ and V₂ (Fig. 2A, arrows) represents a retrograde P wave that suggests supraventricular tachycardia—most likely atrioventricular nodal reentry tachycardia, not sinus tachycardia with normal atrioventricular delay programming.¹ The ventricular pacemaker stimuli after each intrinsic QRS complex are probably from responsive biventricular ICD pacing. After the patient's tachycardia resolved, we observed sinus rhythm, normal right bundle branch configuration, and normal biventricular pacing (Fig. 2B).

When the ICD senses intrinsic activity in one ventricle, it immediately paces the other ventricle, to better synchronize LV contraction. However, the initially narrower QRS configuration suggests that the LV pacing stimuli were not capturing the already depolarized ventricular myocardium. Responsive biventricular ICD

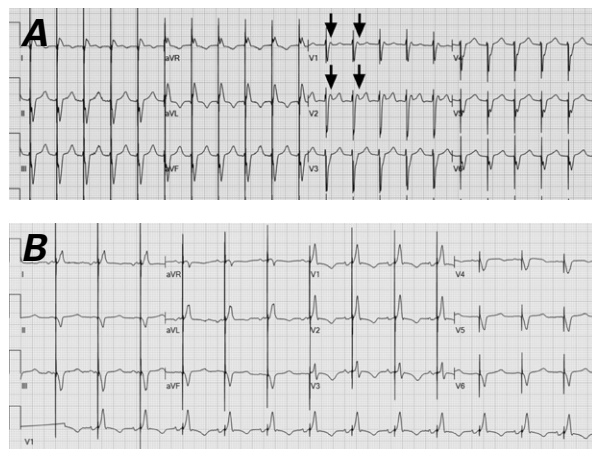


Fig. 2

pacing is expected to produce fusion complex morphology, as in cardiac resynchronization therapy, and indeed the wider QRS complex after conversion to sinus rhythm indicates fusion from His–Purkinje system depolarization with coinciding pacing stimuli.

In Figure 1, the response is not antitachycardia pacing, because the pacing stimuli occur before the QRS complexes; moreover, there was no burst of pacing stimuli faster than the spontaneous rate.¹ Nor is the response pacemaker-mediated tachycardia, because the pacing stimuli occur after the QRS complexes begin.^{2,3} The P waves at the end of the QRS complexes might be retrograde conduction from the pacemaker stimulus; however, the very long subsequent PR intervals suggest another mechanism.

References

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