Focus on ECGs: Case #2

Evaluation of Suspected Device Malfunction on ECG

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A 62-year-old man with nonischemic cardiomyopathy, a history of Boston Scientific biventricular implantable cardioverter-defibrillator placement (in 2011), ventricular tachycardia after radiofrequency ablation (April and November 2014), paroxysmal atrial fibrillation, and severe mitral regurgitation presented with acute exacerbation of heart failure. A resting electrocardiogram (ECG) showed normal sequential atrioventricular (AV) pacing at a heart rate of 63 beats/min. Baseline device settings were DDD with a lower rate of 60 beats/min, an upper rate of 115 beats/min, a minimum sensed AV delay of 135 ms, and a minimum paced AV delay of 180 ms. The patient underwent mitral valve replacement. Three days later, the pacemaker rate was increased to 80 beats/min, and an ECG showed pacing concomitantly within the T wave in beats 5 and 13 of the rhythm strip (Fig. 1).

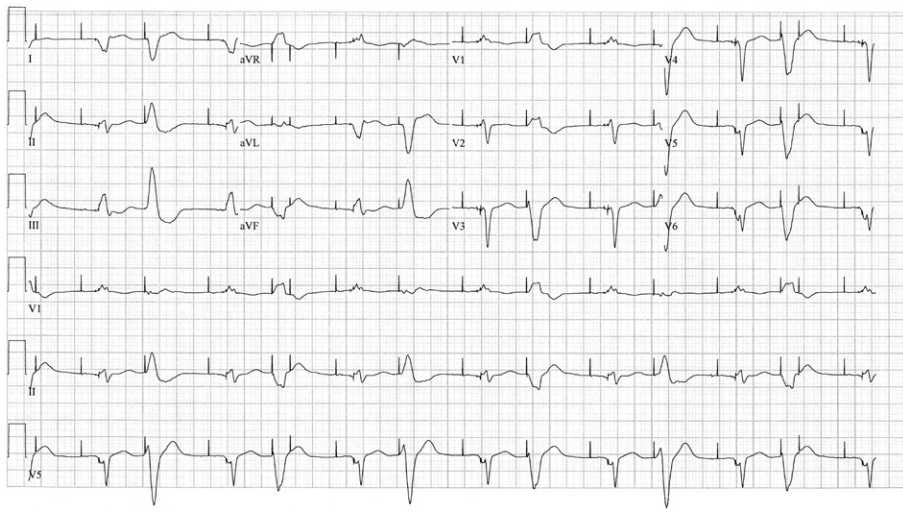

Fig. 1

Should pacemaker malfunction be suspected?

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.*

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Answer

The ECG is compatible with normal function of the device. Biventricular pacemakers frequently have features that attempt to promote pacing and thus ventricular resynchronization. Ventricular sense response resynchronizes ventricular depolarization upon frequent changes in R-R intervals, particularly in patients with atrial fibrillation or frequent premature ventricular contractions (PVCs). On a surface ECG, these events appear as pacing stimuli within a native ventricular depolarization during the attempt to resynchronize the ventricle. This can lead to fusion or pseudofusion beats, depending on the effect of pacing on the depolarization. Typically, these resynchronization stimuli occur 1.25 ms after the initial ventricular stimulus and would not be expected during the T wave.[1]

In this ECG, the AV delay is near the programmed minimum at 200 ms in sequentially paced beats (as shown on beat 2). On beat 3, an atrial stimulus and a PVC appear to inhibit further pacer output. On beat 5, an atrial stimulus and PVC appear almost simultaneously. A pacemaker stimulus follows, notably after a 200-ms delay. This suggests that the PVC occurred during the device's ventricular blanking period, during which the ventricular lead is "blinded" to native ventricular depolarization and thus continues programmed pacing. Therefore, an appropriate 2nd stimulus attempts to pace the ventricle. Pacing stimuli on a T wave can theoretically produce ventricular tachycardia or fibrillation and should prompt device evaluation.[2,3]

In many devices, when a PVC occurs immediately after the post-atrial ventricular blanking (PAVB) period, the PVC usually falls within the crosstalk zone and leads to ventricular-safety pacing. This sequence is initiated after an atrial-paced stimulus, after which comes a sensed event in the ventricular channel. The AV delay of the safety-paced beat is usually much shorter than the programmed AV delay. However, on beat 2 of our patient's ECG, the expected safety pacing was absent, because this manufacturer's pacing algorithm does not use a crosstalk zone for safety pacing. There is only a programmable PAVB period, selected manually (range, 30–200 ms).

Of note, PVCs occurred in a similar manner in our patient, after which a 2nd pacemaker stimulus did not occur on the T wave. Upon review of the timing and morphology of each PVC, ECG beats 3, 7, and 11 had a similar morphology: negative deflection in lead aVL, positive deflection in leads II through aVF, and an early R wave in lead V_1. This suggests a left lateral location—spatially the farthest from the RV lead, allowing delayed arrival outside the blanking period of the RV lead and thus inhibiting stimuli output. The remaining PVCs, in contrast, appear to have had a positive deflection in lead aVL and a negative deflection in leads II through aVF, suggesting an inferoapical left ventricular location much closer to the RV lead. Earlier transit might have caused the inferoapical PVCs to fall within the device's blanking period and led to the discharge seen on the T wave. The exception is beat 9, on which the PVC appears to have occurred about 40 ms later (after the atrial stimuli) than on beats 5 and 13—allowing the signal to fall outside the blanking period and leading to inhibited ventricular-stimulus output.

In this patient, device interrogation showed normal function, no undersensing episodes, and stable lead impedances. No atrial fibrillation or high ventricular rates were recorded. Postoperatively, amiodarone was started, and no further pacemaker stimuli were seen within the T wave.

References

1. Lloyd MS, El Chami MF, Langberg JJ. Pacing features that mimic malfunction: a review of programmable and automated device functions that cause confusion in the clinical setting. J Cardiovasc Electrophysiol 2009;20(4):453-60.
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3. Conover MB. Understanding electrocardiography. 8th ed. St. Louis: Mosby; 2002. p. 245.

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