A 60-year-old man with nonischemic cardiomyopathy and atrial fibrillation is started on sotalol, receives a dual chamber implantable cardioverter defibrillator, and undergoes electrical cardioversion. The bradycardia parameters are set as follows:

- Mode: DDDR 80 to 140 beats per minute
- Atrioventricular delay 220 to 400 ms
- Postventricular atrial refractory period 200 to 350 ms
- Ventricular refractory period 210 to 250 ms
- Postventricular atrial refractory period after premature ventricular complex 400 ms
- A blank after V pace 85 ms
- V blank after A pace (crosstalk window) 65 ms

Post-implant, there are multiple episodes of ventricular pacing with ventricular pacing burden 14%. Which of the following statements is FALSE for the representative episode for which the surface rhythm tracing is shown?

**Answer Options**

A. The first ventricular paced event occurs because of functional ventricular undersensing
B. The episode initiates with retrograde ventriculoatrial conduction
C. Retrograde P waves fall in postventricular atrial refractory period
D. Ventricular pacing induces antegrade atrioventricular conduction conduction block
E. Antegrade conduction of a premature atrial complex interrupts retrograde ventriculoatrial conduction

**Figure.** A representative episode of ventricular pacing on 3-lead cardiac telemonitor.
ANSWER TO JANUARY 8th QUESTION

E. Upgrade to DDDR system

The 12-lead ECG and device ECG (Can—SVC coil) show presence of discrete P waves (Figure 1, arrows) occurring after each biventricular-paced QRS complex, suggesting absence of atrial fibrillation and retrograde 1:1 ventriculoatrial conduction. Figure 2 shows device ECG and ventricular electrogram; biventricular pacing is inhibited after the initial 3 QRS complexes. P waves are shown with arrows. With inhibition of pacing, native P waves are seen to march across and generate intrinsically conducted QRS complexes. Even though this patient had a history of permanent atrial fibrillation and was appropriately implanted with a cardiac resynchronization therapy—defibrillator without an atrial lead, he is now in sinus rhythm. Atrial fibrillation may have terminated with the defibrillator shocks that were previously delivered for ventricular arrhythmia. In the absence of an atrial lead and with underlying sinus rhythm, biventricular pacing results in atrioventricular dyssynchrony, and retrograde ventricular atrial conduction can lead to pacemaker syndrome. Furthermore, the sinus rhythm is competing with biventricular pacing and is likely responsible for the reduced biventricular pacing at 52%. Limited delivery of biventricular pacing and atrioventricular dyssynchrony may be the cause for persistent heart failure symptoms (New York Heart Association class III).

Triggered biventricular pacing in response to ventricular sensing (Option A) will increase the biventricular pacing percentage reported by the device. However, because the bulk of the left ventricle may already be depolarized from intrinsic conduction before biventricular pacing is triggered, this may only provide little or no resynchronization. Maximizing β-blockade and increasing rate responsiveness (Option B), atrioventricular node ablation (Option C), or amiodarone with increase in lower pacing rate (Option D) may prevent competition from intrinsic sinus rhythm to allow delivery of higher percentage of biventricular-paced beats. However, this will lead to continued atrioventricular dyssynchrony. There is no evidence provided in the question for ventricular ectopy as a cause of reduced biventricular pacing to indicate amiodarone as stated in Option D. The optimal solution in this case is to add a right atrial lead and upgrade to DDDR system to provide atrioventricular synchrony, as well as maximize biventricular pacing (tracking atrial rhythm with biventricular pacing).

REFERENCES


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