



Closing the Loop

Keith J.C. Finnie, MB, ChB

Vignette

A 69-year-old man experiences two episodes of syncope. A Holter monitor recording demonstrates intermittent complete heart block. A dual chamber permanent pacemaker is implanted, following which, he is free of syncopal episodes. A few months later, he complains of occasional palpitations. The electrocardiogram (ECG) shown in Figure 1 is recorded during one such episode.

Questions

1. What is the arrhythmia mechanism in this man?
2. What measures may be taken to correct the problem?

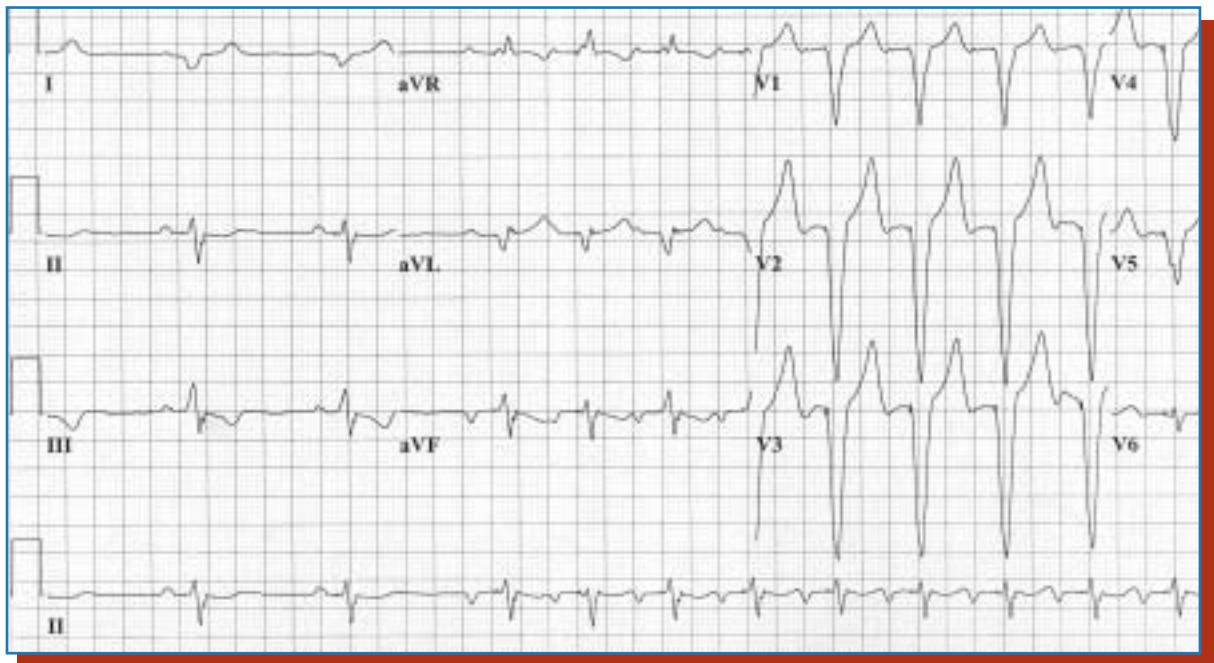


Figure 1. ECG recorded during an episode of palpitations.

Dr. Finnie is a professor of medicine, University of Western Ontario, and site chief of cardiology, London Health Sciences Centre, Victoria Campus, South Street, London, Ontario.

Answers

1. The first two beats in the ECG are of sinus origin. Each P wave is sensed by the pacemaker and followed by a ventricular paced beat after the programmed atrioventricular (AV) delay has elapsed. The pacemaker spikes are so small as to be almost invisible, which is often the case with a bipolar pacemaker. The third P wave of the sequence is an atrial extrasystole and it too is sensed by the pacemaker, which dutifully delivers another ventricular paced beat. Following this, a regular tachycardia at 100 beats per minute emerges. The QRS complexes during this tachycardia are also paced, the pacemaker spikes being easily seen in leads V₃ to V₅.

Close inspection of the lead II rhythm strip at the bottom of the recording reveals that the morphology of the T waves during the tachycardia is quite different from the first two paced beats prior to the arrhythmia. This is because there is a retrograde P wave occurring almost exactly midway between each QRS complex. In effect, the pacemaker is functioning as an artificial "bypass tract". Each ven-

tricular paced beat is conducting retrogradely to the atria. The beat is being sensed by the atrial lead and initiating another ventricular paced beat to maintain AV synchrony according to the programming parameters. The result is a "loop tachycardia". For this to have occurred, there must have been some change in the ability of

the AV node to conduct retrogradely, as the first two paced beats on the ECG show no evidence of retrograde atrial activation.

Lengthening the period before which the pacemaker is not permitted to sense a P wave should prevent the pacemaker from sensing the retrograde P wave and tracking on it.

2. This problem can usually be corrected by reprogramming some of the pacemaker parameters. Lengthening the period before which the pacemaker is not permitted to sense a P wave should prevent the pacemaker from sensing the retrograde P wave and tracking on it. Reducing the sensitivity of the atrial lead may work if

the retrograde P wave has a significantly lower amplitude than the sinus P wave. Finally, programming the pacemaker into the ventricular demand mode and eliminating atrial sensing completely could be considered, although this would mean sacrificing the physiologic benefits of AV synchrony. **DX**