Atrioventricular crosstalk inhibition following sensed atrial events, a rare phenomenon

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Introduction

Atrioventricular (AV) crosstalk inhibition is an uncommon occurrence in the modern era. This phenomenon is seen in DDD pacing systems. The most common scenario is when atrial pacing artifact is inappropriately sensed by the ventricular-sensing amplifier, resulting in ventricular pacing inhibition. We present a rare and challenging case of AV crosstalk inhibition following sensed, intrinsic atrial events (P waves).

Case report

Our patient was a 73-year-old man who presented with a cardiac sounding syncope. His past medical history included type 2 diabetes, hypertension, and hyperlipidemia. His presenting electrocardiogram demonstrated complete heart block requiring the insertion of an urgent temporary pacing wire.

A transthoracic echocardiogram revealed normal left ventricular size, mild systolic dysfunction (left ventricular ejection fraction of 50%), and no significant valvular abnormalities. He underwent implantation of a dual-chamber pacemaker. The intraoperative pacing lead parameters were satisfactory. Several hours postoperatively, the incident shown in Figure 1 was noted on telemetry. This rhythm strip demonstrates a series of P waves which are not followed by paced-QRS complexes. This is consistent with AV crosstalk inhibition. A subsequent chest radiograph confirmed that the leads were in the appropriate positions and there was no evidence of obvious lead dislodgment (Figure 2).

Adjusting the sensitivity setting in the right ventricular channel was unable to fix the issue. As a result, the patient underwent right ventricular lead repositioning that was able to resolve the issue.

KEY TEACHING POINTS

- Atrioventricular (AV) crosstalk is most commonly a result of inappropriate sensing of atrial pacing artifact, leading to right ventricular pacing inhibition.
- Factors predisposing to crosstalk sensing include increased atrial outputs (amplitude and pulse width), increased sensitivity setting in the ventricular channel, unipolar electrode configuration in either atrial or ventricular channels, increased pacing rates, and a short post–atrial blanking period.
- At present there is no programming available to prevent AV crosstalk due to sensing of intrinsic atrial events.

Discussion

AV crosstalk inhibition is a rare and dangerous phenomenon that can occur in DDD pacing systems. It can pose significant risks in pacemaker-dependent patients with AV conduction abnormalities, where the effective paced rate may be slower than the programmed base rate.[1–5] Factors that may predispose to crosstalk sensing include increased atrial outputs (amplitude and pulse width), increased sensitivity setting in the ventricular channel, unipolar electrode configuration in either atrial or ventricular channels, increased pacing rates, dislodgment of the atrial or ventricular leads into the opposite chamber, and a short post–atrial blanking period.[1,3,6,7]

In addition to adjusting the above parameters, programming of modern pacemakers to extend the ventricular blanking period and crosstalk sensing windows can help to prevent ventricular inhibition.[1,7] However, these methods only apply to situations where AV crosstalk inhibition occurs following a sensed atrial pacing artifact and not a sensed intrinsic P wave. As such, we were unable to use these options to

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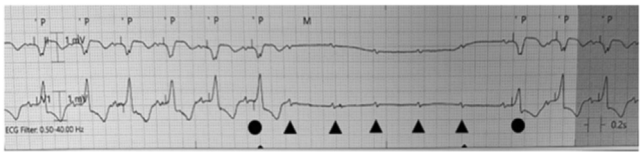

Figure 1 Rhythm strip. Circle indicates atrial sensed beat followed by a ventricular paced event. Arrow indicates sensed P wave, with no ventricular pacing owing to atrioventricular crosstalk inhibition.

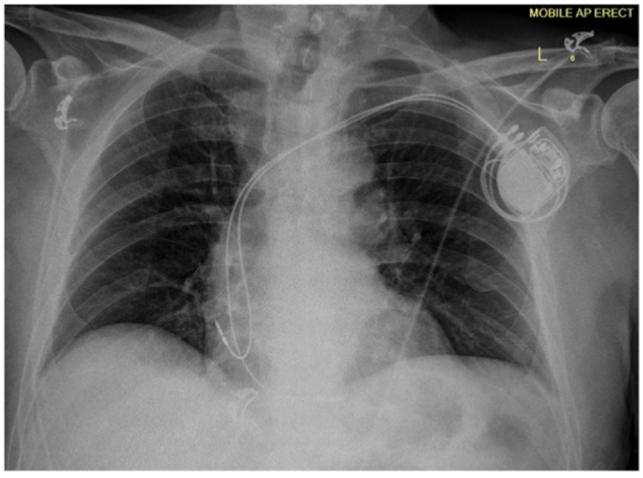

Figure 2 Anteroposterior chest radiograph demonstrating an implanted dual-chamber pacemaker device with atrial lead (right atrial appendage) and ventricular lead (mid right ventricular septum) in appropriate positions.

reprogram the device. If this rare phenomenon is noted during intraoperative lead assessment, we would suggest to avoid implanting the right ventricular (RV) lead in the RV outflow tract or basal RV septal areas, such as deep septal pacing. These areas are near to the atria and, as such, have a higher chance of P-wave oversensing. In this case, implanting the RV lead more apically is reasonable.

We propose that the pacemaker manufacturers develop programming algorithms that could be used to tackle this rare phenomenon so that reoperation can be avoided.

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