A case of paroxysmal atrioventricular block–induced cardiac arrest

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Introduction
Premature ventricular contractions (PVCs) have been shown to cause paroxysmal first-degree,1,2 second-degree,3,4 and complete5–7 atrioventricular (AV) block. We present a case of PVC-induced paroxysmal AV block (PAVB) resulting in cardiac arrest that was managed with implantation of a leadless pacemaker.

Case report
A 66-year-old man with a past medical history of hyperlipidemia and left bundle branch block (Figure 1) was admitted to the hospital for an esophagectomy for esophageal cancer. The postoperative course was complicated by acute renal failure requiring renal replacement therapy. On postoperative day 5, he suffered an asystolic cardiac arrest requiring cardiopulmonary resuscitation (CPR). The rhythm strip from the arrest revealed sinus tachycardia and left bundle branch block, then a series of PVCs, followed by a 20-second period of ventricular asystole due to complete AV block (Figure 2). The patient regained spontaneous circulation after 2 minutes of CPR. Prior to the cardiac arrest, his labs were notable for a pH of 7.29 and a potassium level of 5.6 mmol/L. The same day he was taken for an exploratory laparotomy for abdominal distention, at which viable small and large bowel were found. The patient remained on mechanical ventilation and was closely monitored in the intensive care unit.

A transthoracic echocardiogram performed later demonstrated normal left ventricular chamber size and function with no regional wall motion abnormalities. Cardiac magnetic resonance imaging showed no evidence of infiltrative, inflammatory, or ischemic heart disease and no delayed myocardial enhancement. During his stay at hospital he had infrequent PVCs but no further episodes of asystole, syncope, or dizziness.

The PAVB had occurred in the setting of preexisting bundle branch block, suggestive of underlying conduction disease. Due to an unpredictable recurrence long term, a decision was made to place a single-chamber permanent pacemaker. He had a prior tunneled right internal jugular vein port for chemotherapy and a recent intravenous line–associated left upper extremity deep vein thrombosis extending to the axillary vein, thus limiting options for traditional transvenous pacing. Therefore, a leadless pacemaker (Micra, Medtronic Inc, Minneapolis, MN) was placed in the right ventricle (RV) to treat future PAVB episodes (Figure 3A and B). There were no procedural complications.

Discussion
PAVB is an abrupt, unexpected, repetitive block of atrial impulses as they propagate to the ventricles. It is likely a rare cause of sudden cardiac death. The true incidence of this phenomenon is unknown. It has been observed in association with distal conduction disease at baseline, most commonly right bundle branch block.8 PAVB has been reported as both bradycardia and tachycardia dependent.9 It occurs in the setting of a diseased conduction system, in which cells with a less negative resting membrane potential have impaired excitability, and post-polarization refractoriness.9 The mechanism remains unsettled, with phase 3 block, phase 4 block, and concealed conduction all proposed.8–10 Management requires permanent pacing due to the unpredictable nature of recurrence of the block and the associated risk of sudden cardiac death.

Radiofrequency ablation of the PVC in a patient with PVC-induced first-degree AV block has been previously described.1

KEY TEACHING POINTS
● Paroxysmal atrioventricular block (PAVB) is an uncommon cause of cardiac arrest.
● Permanent pacing is indicated in most cases due to its unpredictable course.
● A leadless pacemaker can successfully be used to manage PAVB in case of limited vascular access.

KEYWORDS Conduction system; Leadless pacemaker; Paroxysmal atrioventricular block; Premature ventricular contractions; Sudden cardiac arrest (Heart Rhythm Case Reports 2018;4:383–385)

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This was not possible in our case due to significant comorbidities, a paucity of PVCs, and the severe nature of the AV block resulting in cardiac arrest that necessitated CPR.

A traditional transvenous pacemaker was not an ideal choice in our case due to the presence of a thrombus in the left axillary venous system and a chronic indwelling catheter in the right internal jugular vein. Leadless pacemakers can be implanted using a single femoral site puncture and have emerged as a promising alternative in patients with limited vascular access. They have a significantly reduced risk of
complications compared to historical transvenous controls, but currently offer RV-only pacing. Chronic RV pacing is known to increase the risk of atrial fibrillation and cardiomyopathy in patients with intact AV conduction. Dual-chamber leadless pacemakers are under development, but are not currently available. In our patient, apparently infrequent, relatively brief, but potentially catastrophic AV block was followed by normal AV conduction, suggesting that long-term single-chamber antibradycardia support was sufficient to prevent adverse clinical events. During 1-month follow-up the ventricular pacing was less than 1% and there were no symptoms to suggest pacemaker syndrome.

To our knowledge, this case is the first to use a leadless pacemaker to manage PAVB.

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