Nonphysiological prolongation of PR intervals can lead to palpitations and shortness of breath similar to those seen with pacemaker syndrome. This has been shown to be related to diastolic mitral regurgitation. We present a case of intermittent first-degree AV block that was caused by premature ventricular contraction (PVC)–induced slow pathway conduction. Catheter ablation of the slow pathway and the PVC focus normalized the PR interval and abolished all symptoms.

Case History
The patient is a 74-year-old woman with a structurally normal heart who presented with palpitations, shortness of breath, dizziness, and decreased exercise tolerance. Event monitoring disclosed no sustained arrhythmia but revealed frequent PVCs. The PVCs had left bundle-branch block morphology and a normal QRS axis. The baseline PR interval was normal; however, prolonged periods of first-degree AV block (PR>500 ms) were noted. First-degree AV block was consistently initiated by PVCs (Figure 1A). Interestingly, appropriately timed PVCs just as consistently led to normalization of the PR interval (Figure 1B). In the absence of such appropriately timed PVCs, slow pathway conduction persisted. She did not respond to therapy with calcium channel blockers, β-blockers, or low dose flecainide. An electrophysiology study was undertaken. Baseline conduction intervals were within normal limits. Programmed atrial stimulation revealed dual AV nodal pathways with a fast pathway effective refractory period of 560 ms. No arrhythmia was induced with up to double atrial premature beats. Spontaneous PVCs consistently led to prolongation of the AH interval from 102 to 560 ms (Figure 2A). There was no change in the HV interval. During periods with long AH intervals, spontaneous or timed PVCs led to normalization of the PR interval (Figure 2B). These findings confirmed the observations from the event monitor noted above. Slow pathway ablation was undertaken. Application of radiofrequency energy at the classical slow pathway location using a 3-dimensional electroanatomic mapping system (CARTO) induced transient junctional rhythm. After ablation, the patient continued to have frequent PVCs with VA conduction, but these did not lead to prolongation of the AH interval (Figure 2C). There was no remaining evidence of dual AV nodal pathways. Regarding the still frequent PVCs, it was noted that the ventricular electrogram at the His location was earlier than the electrogram in the right ventricular apex or outflow tract. A 3D activation map of PVCs was created. The earliest ventricular activation was found to be at the posteromedial aspect of the RV just distal to the tricuspid ring. The local electrogram at that site during the PVC preceded the onset of the surface QRS by 35 ms and showed a negative unipolar electrogram. A single lesion at this site abolished the PVCs. After ablation, no PVCs were seen, despite programmed ventricular stimulation with up to triple premature beats and isoproterenol infusion. All medications were discontinued.

After ablation, the patient no longer complained of the above symptoms. Two weeks of event monitoring demonstrated a consistently normal PR interval and no ventricular prematurity beats. No symptoms or arrhythmias were subsequently documented.

Discussion
This patient presented with intermittent first-degree AV block caused by conduction down the slow pathway. We speculate that during sinus rhythm rapid conduction down the fast pathway (normal PR and AH intervals) led to concealed retrograde conduction up the slow pathway. Appropriately timed PVCs with retrograde conduction led to block in the fast pathway because of collision proximal to the turn-around site in the AV node, thus eliminating block in the slow pathway. The subsequent sinus beat conducted down the slow pathway, leading to long PR and AH intervals. Slow pathway conduction was maintained by retrograde concealed conduction in the fast pathway. During persistent slow pathway conduction, appropriately timed PVCs with retrograde conduction, by the same mechanism, led to collision and block in the slow pathway, thus preventing retrograde concealed conduction in the fast pathway, thereby making the fast pathway once again available for anterograde conduction. The next sinus beat and subsequent beats, therefore, conducted down the fast pathway with normal PR and AH intervals. The elimination of intermittent first-degree AV block after slow pathway ablation, despite the persistence of PVCs, supports this mechanism.
The prevalence of first-degree AV block, which rises with age, is estimated as 2% to 4%. Although first-degree AV block is generally seen as a benign condition, prolongation of the PR interval can be associated with atrioventricular dysynchrony. Symptoms similar to those in our patient caused by slow pathway mediated first-degree AV block have been described. Our case illustrates that intermittent first-degree AV block can be related to slow pathway conduction caused by ventricular premature beats with retrograde conduction. This is an interesting and unusual indication for slow pathway ablation.

Disclosures
None.

References

Key Words: atrioventricular block ■ atrioventricular node ■ catheter ablation ■ ventricular premature complexes

Figure 1. Event monitoring revealing first-degree AV block initiated by premature ventricular contraction (PVC; A) and normalization of PR interval caused by appropriately timed PVC (B). These phenomena occurred frequently and consistently.

Figure 2. Intracardiac electrograms confirm phenomenon documented by event monitor. A, premature ventricular contractions (PVCs) result in prolongation of AH interval. B, PVCs result in normalization of AH interval. C, After ablation PVCs conduct retrograde but do not lead to prolongation of AH interval. CS indicates coronary sinus; HIS, bundle of his; HRA, high right atrium; and RV, right ventricle.
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