An 82-year-old man with a history of diabetes mellitus, hypertension, chronic kidney disease, polycythemia, hypothyroidism, T-cell lymphoma, and prostate cancer presented with complaints of dyspnea on exertion for several months.

He underwent extensive evaluation including an echocardiogram, which showed normal left ventricular systolic function with an ejection fraction of 63%, left ventricular hypertrophy, moderate aortic stenosis, and mild to moderate aortic regurgitation. A cardiac catheterization showed normal filling pressures with moderate aortic stenosis. He was thought to have tachy-brady syndrome because of episodes of rapid atrial fibrillation and sinus bradycardia at rest. Because of exercise associated fatigue and dyspnea, the patient was referred for pacemaker implantation.

Careful inspection of hospital telemetry revealed frequent atrial premature contractions (APCs), often in a bigeminal pattern, which blocked in the A V node and caused compensatory reset of the sinus node, resulting in heart rates in the 40s (Figure 1). The patient also had periods of atrial fibrillation with rapid ventricular rates that appeared to be initiated by APCs identical to those nonconducted APCs that caused bradycardia (Figure 2). With ambulation, the patient’s heart rate abruptly and reproducibly decreased from 80 to 40 bpm as a result of the blocked APCs, and correlated with his symptoms of fatigue and dyspnea.

Because of the close coupling interval of his APCs (160 ms), pacemaker implantation was not be feasible because it would require programming a short postventricular atrial refractory period (< 160 ms) as well as a high tracking rate (>135 bpm), which would have been inappropriate for this elderly patient. Therefore, the decision was made to proceed with an invasive electrophysiology study and catheter ablation of the APC.

The site of origin of the APCs was localized to the anteroseptal mitral annulus (Figure 3). Radiofrequency ablation at the site of origin permanently eliminated the APCs within 5 seconds of the onset of energy (Figure 4). Additional radiofrequency delivered in this area (insurance burns) induced short paroxysms of atrial fibrillation. There was no evidence of sinus node dysfunction as determined by sinus node recovery time. Immediately after the procedure, the patient had complete resolution of his symptoms and prolonged ambulatory ECG monitoring revealed normal sinus rhythm, resolution of APCs, and an absence of prolonged pauses and atrial arrhythmias.

This case highlights the deleterious and myriad arrhythmogenic potential of seemingly benign but closely coupled APCs. As shown herein, APCs can sometimes be a subtle and easily overlooked cause of bradycardia (via block in the AV node and sinus node reset), in addition to being a well known trigger of atrial fibrillation. Both of these arrhythmic problems (bradycardia and atrial fibrillation) were abolished with a single radiofrequency burn of a focal, mitral annular arrhythmogenic source.

References

Disclosures
None.
Figure 1. ECG shows bradycardia. Arrows highlight the atrial premature contractions (APCs) that occur with close coupling to the sinus beats and which block in the AV node and reset the sinus node. The final APC conducts to the ventricle after a longer coupling interval. The APCs are more apparent in leads III and aVF.

Figure 2. Telemetry monitor recording shows initiation of atrial fibrillation from an identical atrial premature contraction (APC). Arrows highlight the APCs. The first one is blocked in the AV node and the subsequent APCs trigger atrial fibrillation.

Figure 3. Electroanatomical map in the left anterior oblique projection showing the site of origin of the APC along the antero-septal aspect of the mitral annulus (teal color). Coronary sinus catheter is shown in red, and yellow dot represents His bundle along the anteroseptal tricuspid annulus. White circle denotes the relative position of the mitral annulus (MA).
Figure 4. Elimination of the atrial premature contraction (APC) with ablation. **Left,** Sinus beat followed by APC with earliest activation preceding surface P wave and intracardiac electrograms by 30 ms. **Right,** APCs (red ovals on surface ECG and asterisks on intracardiac electrograms) occurring in a trigeminal pattern before ablation at the site of origin resulted in elimination of the APCs and restoration of sinus rhythm within 5 seconds (**right**). ABL indicates ablation; CS, coronary sinus; d, distal; lat, lateral; p, proximal; RA, right atrium; and sept, septum.
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Circulation. 2014;130:e148-e150
doi: 10.1161/CIRCULATIONAHA.114.011155
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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