Enhanced AAIR→DDDR pacing (atrial-based Managed Ventricular Pacing [MVP]) is a novel pacing mode that facilitates intrinsic conduction whenever possible to mitigate the deleterious effects of prolonged duration right ventricular (RV) electric stimulation on left ventricular contractility. The following case illustrates uncommon ECG and hemodynamic consequences of this algorithm in a patient with marked atrioventricular (AV) conduction delay.

Brief Case Description
A 58-year-old man with a history of acute myeloid leukemia, subsequent bone marrow transplantation, nonablated paroxysmal atrial flutter, and tachy-brady syndrome underwent implantation of a Medtronic Adapta ADDR01 permanent dual-chamber pacemaker (Medtronic Inc., Minneapolis, Minn.) for symptomatic junctional bradycardia with leads positioned in the right atrial (RA) appendage and RV apex. After implantation, flecainide 100 mg twice daily was added to extended-release diltiazem 240 mg daily with a resultant absence of arrhythmia on serial pacemaker telemetry interrogations. His pacemaker was programmed with enhanced AAIR→DDDR pacing enabled at a lower rate limit of 60 bpm. A routine ECG obtained 1 month later (Figure 1) demonstrated atrial pacing and capture in the terminal portion of the preceding T wave with pronounced 1° AV block. Closer scrutiny of the tracing reveals 2 separate atrial depolarization waves. Repolarization abnormalities and QT interval prolongation were attributed to the effects of intermittent RV pacing and the concomitant administration of flecainide, fluoxetine, sulfamethoxazole-trimethoprim, and azithromycin. Two-dimensional echocardiography at that time demonstrated biatrial and RV enlargement with normal left ventricular and RV regional and global function.

Figure 2 displays the temporal relationship between the RA and left atrial (LA) depolarization in leads V1 through V3. The right atrial pacemaker stimulus artifact is followed by immediate RA capture. A second atrial deflection with opposite polarity representing LA depolarization is noted ∼200 ms after RA activation and is followed 240 milliseconds later by an intrinsically conducted narrow QRS complex.

Figure 1. Twelve-lead ECG.
Figure 3 displays contemporaneous transtricuspid and mitral continuous-wave Doppler velocity-time integrals aligned using the atrial pacemaker stimulus artifact. The top panel demonstrates merging of the transtricuspid valve rapid inflow (E wave) velocity-time integral with the RA A wave. The initiation of RA contraction is coincident with RA pacing, and anterograde flow across the transtricuspid valve continues into early ventricular systole. The stimulus-to-QRS interval is ≈400 milliseconds. The bottom panel depicts the transmitral valve inflow velocity-time integral. A nearly normal temporal relationship (minimal fusion) of the E and A waves is demonstrated. Baseline artifact and low-amplitude signals hamper definitive identification of the P-wave onset on the ECG rhythm strips displayed; however, the LA A-wave-to-QRS interval is ≈185 milliseconds. The paced cycle length difference between the upper and lower panels is 50 milliseconds.

**Discussion**

The presence of interatrial conduction delay has received much attention in the literature as a risk factor for the development of atrial tachyarrhythmias. However, it has been underappreciated as an important mediator of optimal myocardial performance in patients with dual-chamber pacemakers and implantable cardioverter-defibrillators. Several factors contribute to the ECG findings in this case. RA appendage pacing produces marked alterations in intraatrial and interatrial impulse transmission that impairs coordinated atrial activation.1 Enhanced AAIR<=>DDR pacing (MVP) is designed to reduce high-density RV pacing in patients with sinus rhythm and intact AV conduction through extension of the AV delay after a paced or sensed atrial event.2,3 However, this programming may generate markedly prolonged AV intervals that in some instances produce “pseudo-pacemaker syndrome.”4 Flecainide and diltiazem slow conduction in the atria, AV node, and His-Purkinje system and further contribute to abnormal interatrial and AV conduction.

**Conclusions**

Despite an effective PR interval approximating 400 milliseconds, the combination of marked interatrial and AV conduction delay paradoxically “normalizes” LA transport function. Patients with dual-chamber pacemakers and implantable cardioverter-defibrillators programmed to allow permissive extension of the AV interval and who receive conduction-altering medications may benefit from periodic echo Doppler evaluations at incremental paced atrial rates to determine the integrity of interatrial and AV conduction and to optimize these relationships when appropriate.
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Disclosures
None.

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