Is this pacemaker working properly?
Prolongation of the atrial escape interval following activation of the ventricular safety pacing algorithm in a dual-chamber implantable cardioverter defibrillator with atrial-based, lower rate timing

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CASE PRESENTATION

A 78-year-old woman was referred to the Cardiac Rhythm Device Clinic at the Kingston General Hospital (Kingston, Ontario) due to ischemic cardiomyopathy and symptoms of chronic congestive heart failure. A resting radionucleotide scan demonstrated an ejection fraction of 30%. A dual-chamber implantable cardioverter defibrillator (ICD) (7274 Marquis DR; Medtronic Inc, USA) was implanted.

The ICD was programmed to a lower rate of 60 beats/min (atrial-based), a paced atrioventricular delay (AVD) of 180 ms and a sensed AVD of 150 ms. Rate response was ‘on’ and ventricular safety pacing was activated. The postventricular atrial refractory period was 250 ms, the premature ventricular complex (PVC) response was ‘on’, atrial sensitivity was at 0.3 mV and ventricular sensitivity was at 0.3 mV.

During one of the patient’s routinely scheduled follow-up appointments, a 12-lead ECG was obtained (Figure 1A).

As seen in the rhythm strip of Figure 1A (lead V1), the first and second complexes were due to an atrial paced event followed by a ventricular paced event with an AVD of 180 ms (Figure 1B). The ventricular rate was 900 ms with an atrial escape interval (AEI) of 720 ms. There was a PVC with a fixed coupling interval (600 ms) following the two normally paced complexes that led to activation of the ventricular safety pacing mechanism. An atrial pacing stimulus was delivered within the initial portion of the PVC at 900 ms from the previous atrial spike. The tail of the PVC was sensed within the ventricular safety pacing window. The ICD, instead of inhibiting ventricular pacing, delivered a ventricular pacing stimulus at a shortened AVD.

Key Words: Atrial escape interval prolongation; Electrocardiography; Pacemakers; Pacing; Safety pacing

Figure 1) A 12-lead electrocardiogram obtained during a routine follow-up.
B Detailed analysis of intervals. A-A Paced atrial beat to paced atrial beat interval; AEI Atrial escape interval; AVD Atrioventricular delay; SP Safety pacing

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(110 ms). A shortened AVD decreases the likelihood of a ventricular paced event occurring during ventricular repolarization, minimizing the risk of ventricular proarrhythmia. This ventricular pacing stimulus fails to capture because it occurs during the physiological refractory period of the ventricle. Although safety pacing triggered an earlier ventricular paced event, the timing cycle of the next event was not reset based on the delivered ventricular stimulus. Instead, the lower rate was determined by the atrial-based timing.

The lower rate limit can be ventricular-based and controlled by ventricular events (paced or sensed). In ventricular-based timing, a paced ventricular stimulus resets the timing cycle, whereas the AEI remains fixed (1). The ECG in our example demonstrated atrial-based timing. In the present case, the interatrial (A-A) interval was fixed at 900 ms, and after activation of ventricular safety pacing, the AEI was extended from 720 ms to 790 ms. The ventricular paced stimulus delivered at the shortened AVD during the ventricular safety pacing window did not reset the interventricular interval. Instead, because the A-A interval was fixed, the AEI was extended by an amount equal to the decrease in AVD due to ventricular safety pacing (Table 1). This mechanism (early sensing of the ventricular spike) was facilitated by high atrial sensitivity.

TABLE 1
Calculation of the extension of the atrial escape interval (AEI)

<table>
<thead>
<tr>
<th></th>
<th>Without safety pacing</th>
<th>With safety pacing</th>
</tr>
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<tbody>
<tr>
<td>AVD</td>
<td>180 ms</td>
<td>110 ms</td>
</tr>
<tr>
<td>AEI</td>
<td>900 ms – 180 ms = 720 ms</td>
<td>900 ms – 110 ms = 790 ms</td>
</tr>
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AVD Atrioventricular delay

Ventriculoatrial crosstalk can be triggered by ventricular sensed or paced events. In atrial-based timing systems, crosstalk may result in prolongation of the AEI to maintain the timing according to the lower rate limit. A previous report from Barold (2) demonstrated prolongation of the AEI following far-field R wave (premature ventricular contraction [PVC]) sensing. High atrial sensitivity facilitates this mechanism. In our case, a patient with a dual-chamber ICD experienced ventricular trigeminy, which triggered the ventricular safety pacing mechanism with subsequent prolongation of the AEI.

CONCLUSION
The pacemaker/ICD in the present case was functioning normally. Automatic extension of the AEI to maintain a fixed A-A interval in atrial-based timing pacemakers is one of the new features of the latest generation of pacemakers and ICDs. Careful analysis of the 12-lead ECG of patients with cardiac devices will lead to improved recognition of normal functioning, minimizing additional testing and consultation.

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