CASE REPORTS

Hyperkalemia-Induced Failure of Atrial Capture During Dual-Chamber Cardiac Pacing

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This report describes hyperkalemia-induced failure of atrial capture associated with preservation of ventricular pacing in a patient with a dual-chamber (DDD) pacemaker. This differential effect correlates with experimental and clinical observations showing that the atrial myocardium is more sensitive to hyperkalemia than is the ventricular myocardium (4–6).

Hyperkalemia increases the ventricular excitability threshold and occasionally causes failure of an artificial ventricular pacemaker (1–3). In this report, we describe hyperkalemia-induced failure of atrial capture associated with preservation of ventricular pacing in a patient with a dual-chamber (DDD) pulse generator. This differential effect correlates with experimental and clinical observations showing that the atrial myocardium is more sensitive to hyperkalemia than is the ventricular myocardium (4–6).

**Case Report**

A 75 year old man presented with intermittent atrioventricular (AV) block and received an Intermedics Cosmos unipolar DDD pulse generator (model 283-01) implanted transvenously. He was also being treated for coronary artery disease, congestive heart failure and ventricular arrhythmias with digoxin, furosemide, isosorbide dinitrate, captopril and quinidine. The long-term pacing thresholds were determined 8 weeks after pacemaker implantation. The atrial threshold was 2.7 V at 0.05 ms pulse width, and the ventricular threshold was 2.7 V at 0.10 ms pulse width. These pacing thresholds remained unchanged over the next 10 months. The pulse generator was programmed as follows: lower rate, 70 pulses/min; upper, 110 pulses/min; atrioventricular (AV) delay, 200 ms; atrial output, 2.7 V at 0.6 ms; ventricular output, 5.4 V at 0.3 ms; atrial sensitivity, 0.4 mV; ventricular sensitivity, 4 mV; postventricular atrial refractory period, 290 ms; and ventricular refractory period, 200 ms.

About a year after pacemaker implantation, the patient was hospitalized with recurrent congestive heart failure and pneumonia. The electrocardiogram (ECG) showed normal DDD pacing and sensing. The atrial and ventricular pacing thresholds were unchanged. Pertinent laboratory investigations on admission were as follows: serum potassium, 4.5 mEq/liter; blood urea nitrogen (BUN), 38 mg/dl; creatinine, 2 mg/dl; digoxin level, 1.7 ng/ml; and quinidine level, 4.3 μg/ml. The patient’s condition improved with diuretics and the pneumonia responded to erythromycin.

**Effects of hyperkalemia.** Twelve days after admission he became hypotensive with a blood pressure of 70/40 mm Hg. The ECG revealed no spontaneous atrial activity, ineffectual atrial stimuli and widening of the paced QRS complex. Atrial capture remained unsuccessful even with a maximal atrial output of 8.1 V and 1.0 ms pulse width (Fig. 1). The AV delay was programmed to 300 ms to exclude latency of atrial depolarization. Temporary reprogramming to the atrial-inhibited (AAI) mode revealed ineffectual atrial pacing stimuli, no spontaneous atrial or ventricular depolarizations and prolonged ventricular asystole. The threshold for ventricular pacing was 5.4 V at 0.2 ms pulse width, and the ventricular output of the pulse generator was therefore reprogrammed to 5.4 V and 0.6 ms pulse width. At the time, pertinent laboratory investigations revealed: serum sodium, 127 mEq/liter; potassium, 6.3 mEq/liter; CO₂, 34 mEq/liter; chloride, 80 mEq/liter; BUN, 69 mg/dl; creatinine, 4.4 mg/dl; serum quinidine, 6.3 μg/ml; and serum digoxin, 4.7 ng/ml. Five units of regular insulin were ad-
ministered intravenously followed by 50 cc of 50% glucose, also intravenously. A few minutes after the glucose infusion, the ECG revealed successful atrial capture (Fig. 2) at which point the blood pressure immediately increased to 100/70 mm Hg. At that time the threshold for atrial pacing was 5.4 V at 0.3 ms. The patient was treated with sodium polystyrene sulfonate (Kayexalate) enemas. The next day, when the serum potassium was normal, the ECG showed normal AV sequential pacing with a shorter duration of the paced P wave and QRS complex (Fig. 3). The threshold for atrial pacing was 2.7 V at 0.05 ms pulse width and the ventricular threshold was 2.7 V at 0.10 ms pulse width. Subsequent pacemaker function has remained normal and the thresholds have also remained constant.

**Discussion**

Hyperkalemia depresses impulse conduction and myocardial excitability (1–8). The myocardium is far more sensitive to the effect of hyperkalemia than are the specialized fibers of the sinoatrial node and the bundle of His (5–7). The atrial myocardium is also more sensitive to hyperkalemia than is the ventricular myocardium (4–7). In the atria, hyperkalemia causes prolongation of the PR interval and the P wave gradually flattens until it disappears from the surface ECG, when “sinoventricular” conduction may continue from the sinoatrial node to the ventricles by way of specialized atrial pathways (4–7).
Effect of hyperkalemia on the pacing threshold. The effect of potassium on the excitability threshold of atrial and ventricular myocardium is not clearly defined. Failure of impulse propagation due to depression of intraatrial or intraventricular conduction may explain the failure of pacemaker capture in hyperkalemia. The pacing threshold may also be influenced by local changes in conduction near the pacing electrodes (9). Indeed, during ventricular pacing, hyperkalemia may cause latency or an increase in the interval between the ventricular stimulus and the onset of the paced QRS complex (first degree ventricular pacemaker exit block), sometimes progressing to Wenckebach (type I) second degree pacemaker exit block or even complete exit block (3,10,11).

As far as atrial pacing is concerned, Bashour et al. (12) reported a single case of hyperkalemia-induced (K = 6.8 mEq/liter) latency (prolongation of the interval from the atrial stimulus to the paced P wave) that decreased after treatment of hyperkalemia. We could not find any previous report of hyperkalemia-induced unresponsiveness of the atria to pacing stimuli in humans. Indeed, total unresponsiveness to ventricular stimuli (that is, complete ventricular pacemaker exit block) has been reported in only three cases of hyperkalemia with plasma potassium levels of 6.6, 6.9, and 7.1 mEq/liter, respectively (1-3). The modest elevation of serum potassium in these cases and ours suggests that numerous other metabolic variables may influence the sensitivity of cardiac tissue to hyperkalemia. These include other electrolytes, acid-base balance (13), oxygen saturation (14), the rate of change of plasma potassium level, the intracellular-extracellular potassium gradient (15,16), and the etiology and severity of heart disease (7). For this reason, the cardiac manifestations of hyperkalemia in the clinical setting tend to occur at much lower potassium levels than the levels measured during the experimental infusion of potassium (1,6,7). The high serum digoxin and slightly raised quinidine levels in our patient probably contributed little to the elevation of pacing thresholds because of the immediate and dramatic restoration of effectual atrial pacing with the treatment of hyperkalemia. In addition, previous studies suggest that digitalis does not increase the pacing threshold (14,15,17).

Clinical implications. Our observations have clearly demonstrated the differential effect of hyperkalemia on the excitability threshold of the atria and ventricles in a patient with a DDD pacemaker. In patients susceptible to hyperkalemia, the relatively sudden occurrence of failure of atrial pacing with the preservation of ventricular pacing by a DDD pacemaker should not be misinterpreted as a primary or technical pacemaker problem. In patients with a DDD pacemaker, poor left ventricular function and hyperkalemia, the loss of atrial contribution to ventricular filling may lead to hemodynamic deterioration that could be potentially fatal if not promptly diagnosed as being secondary to hyperkalemia and treated accordingly.

References