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 on atrial and ventricular excitability during cardiac pacing correlates with the well known clinical and experimental observation that the atrial myocardium is more sensitive to hyperkalemia than is the ventricular myocardium. (J Am Coll Cardiol 1987;10:467-9)

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-
Figure 1. ECG (leads V1 to V6) showing hyperkalemia-induced failure of atrial capture during dual-chamber (DDD) pacing. The paced QRS complex is widened to 0.36 second. Pacemaker variables: lower rate, 70 pulses/min; atrioventricular delay, 200 ms; atrial output, 8.1 V at 1.0 ms pulse width; and ventricular output, 5.4 V at 0.6 ms pulse width.

Figure 2. ECG (leads V1 to V6) showing restoration of atrial capture a few minutes after treatment of hyperkalemia. The interval from the onset of the atrial stimulus to the isoelectric segment of the PR interval measures approximately 0.22 second and represents delay in intraatrial conduction. The duration of the paced QRS complex has shortened to 0.30 second. Pacemaker variables: lower rate, 70 pulses/min; atrioventricular interval, 300 ms; atrial output, 8.1 V at 1.0 ms pulse width; ventricular output, 5.4 volts at 0.6 ms pulse width.

Figure 3. ECG (leads V1 to V6) recorded 24 hours after Figures 1 and 2 when the serum potassium was normal. The interval from the onset of the atrial stimulus to the isoelectric segment of the PR interval has shortened to 0.16 second and the paced QRS complex exhibits further shortening to 0.24 second. Pacemaker variables: lower rate, 90 pulses/min (increased from 70 because of congestive heart failure and ventricular ectopic activity); atrioventricular interval, 300 ms; atrial output, 5.4 V at 0.6 ms pulse width; ventricular output, 5.4 V at 0.6 ms pulse width.

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. 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 pace block), sometimes progressing to Wenckebach (type I), 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. Am J Cardiol 1986;57:337–8.


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References


