Hyperkalemia has several electrophysiologic effects on cardiac tissues that allow for its ready detection on a standard electrocardiogram. The diagnosis is easiest when the baseline tracing is normal with sinus rhythm and a normal QRS duration. We report this case of hyperkalemia which was diagnosed in the presence of ventricular pacing and atrial fibrillation and which resulted in the conversion of the atrial fibrillation to normal sinus rhythm.

**CASE REPORT**

A 39-year-old white man was admitted in June, 1978 for revision of his pacemaker pocket. He had been well until March, 1973 when he developed dyspnea on exertion. Right heart failure was detected after he had had an upper respiratory infection. Cardiac catheterization in May, 1973 revealed hemodynamics consistent with a restrictive cardiomyopathy, moderate pulmonary hypertension, and 2+ aortic insufficiency. In April, 1978, a permanent transvenous pacemaker was inserted to control atrial fibrillation with a slow ventricular response and syncope. Renal function was mildly abnormal with the serum creatinine ranging from 1.8-2.9 mg/dl and the blood urea nitrogen from 38-97 mg/dl. Serum potassium was normal on several determinations.

He subsequently developed a large hematoma at the pacemaker site and was re-admitted on June 15, 1978. The initial ECG (Fig 1A) revealed coarse atrial fibrillation with occasional paced beats. The blood urea nitrogen was 72 mg/dl, serum creatinine was 2.6 mg/dl, and the serum potassium was 4.4 mEq/L. On June 16, 1978 he underwent revision of his pacemaker pocket under local anesthesia. Postoperation he developed increasing fatigue, lethargy, and slurred speech. An ECG was obtained (Fig 1B) which demonstrated loss of atrial fibrillatory activity and a paced rhythm with marked widening of the QRS complexes, findings suggestive of hyperkalemia. Laboratory data revealed a serum potassium of 8.9 mEq/L, blood urea nitrogen of 189 mg/dl, and serum creatinine of 7.5 mg/dl. After treatment with calcium, sodium bicarbonate, glucose, insulin, and Kayexalate, the serum potassium decreased to 4.1 mEq/L. A repeat ECG the following day (Fig 1C) revealed normal sinus rhythm with first degree AV block and a normal QRS duration. Renal function returned to baseline after a short course of peritoneal dialysis, and the patient remained in normal sinus rhythm.

**DISCUSSION**

Hyperkalemia has been shown to lower resting

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**FIGURE 1** Serial electrocardiogram leads 2, V2, and V3. A. Serum potassium level 4.4 mEq/L. B. Serum potassium level 8.9 mEq/L. C. Serum potassium level 4.1 mEq/L. Changes in QRS duration are best appreciated by comparison of the paced complexes in lead V2, whereas changes in atrial activity can be seen best in leads 2 and V3.
membrane potential, shorten action potential duration, and decrease the velocity of phase 0 of the action potential, thereby decreasing the velocity of conduction in the myocardium. This results in several changes on the standard electrocardiogram including peaked T waves, shortening of the QT interval, QRS prolongation, left axis deviation, decreased P wave amplitude, and sinoventricular conduction.

Atrial muscle has been shown to be especially sensitive to depolarization by potassium, and consequent loss of atrial activity may be apparent even in patients with atrial fibrillation. Although conversion to normal sinus rhythm after treatment of hyperkalemia has been previously described, there has been only one prior report documenting the transitional tracing of atrial arrest. In the present case, hyperkalemia was recognized, in spite of ventricular pacing, by the disappearance of atrial activity and by an increase in the duration of the paced QRS complexes from 180 msec to 240 msec. The ability to make this diagnosis in the presence of a paced rhythm may require careful comparison with a previous tracing.

REFERENCES

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