The Silence of the Atria

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A 78 year old woman with a history of hypertension, hyperlipidemia and diastolic heart failure presented with profound bradycardia. She had been complaining of general weakness and headache, without chest pain or dyspnea. She was taking angiotensin-converting enzyme inhibitors, spironolactone, beta-blockers, calcium channel blockers, aspirin, furosemide and statins. The dosage of ACE inhibitors was doubled a week prior to her admission. She had been consuming large amounts of bananas and citrus fruits, known to be rich sources of potassium.

On physical examination, she was fully conscious and hemodynamically stable. An ECG demonstrated severe bradycardia of 33 bpm with normal QRS axis, narrow QRS complexes and absence of P waves – "silence of the atria" [Figure 1]. Laboratory studies revealed a serum potassium level of 7.9 mEq/L, serum urea 101 mg/dl and serum creatinine 2.1 mg/dl (compared to her baseline of 1.1 mg/dl). The patient was treated with intravenous fluids, calcium gluconate, insulin, furosemide and kayexalate with a reduction of the potassium level to 5.7 mEq/L within 6 hours. A subsequent ECG revealed sinus bradycardia of 47 bpm with reappearance of P waves, no changes in QRS axis or width, and only a minimal change in T wave morphology [Figure 2].

Electrocardiographic manifestations of hyperkalemia commonly include peaked T waves, widening of the QRS complexes, prolongation of the PR interval, flattening or absence of the P wave, and a "sine wave" appearance at severely elevated levels [1]. Sinus tachycardia and bradycardia, idioventricular rhythm and first, second and third-degree atrioventricular block have all been described as possible manifestations of hyperkalemia [2]. When a patient presents with bradycardia it is likely that the ECG will be available before the serum potassium findings. Clinicians should be aware that life-threatening hyperkalemia may cause profound bradycardia which, as in our case, may superficially resemble complete AV block. However, true second and third-degree AV block are uncommon in hyperkalemia, because the P waves usually disappear before such advanced AV block occurs [3]. If the ECG suggests life-threatening hyperkalemia, one should consider giving calcium gluconate, without waiting for serum electrolyte results.

ACE = angiotensin-converting enzyme
AV = atrioventricular

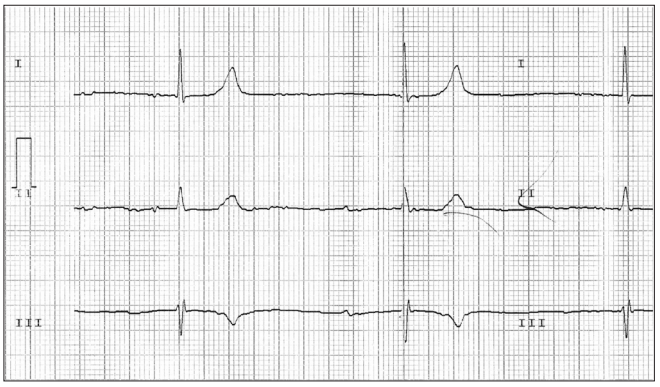

Figure 1. ECG during hyperkalemia (potassium 7.9 mEq/L) showing bradycardia of 33 bpm and absence of P waves ("silence of the atria"). Note the normal QRS axis and the narrow QRS complexes.

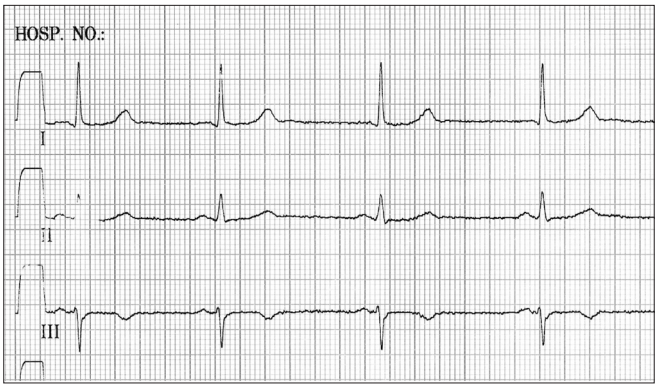

Figure 2. ECG of the same patient after treatment for hyperkalemia (potassium 5.7 mEq/L), showing sinus bradycardia of 47 bpm with reappearance of *P* waves.

References

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