Answer

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The correct answer is C: Hyperkalemia. Upon the arrival of the patient, one of our quick-thinking nurses obtained a stat serum potassium level, as part of his "weakness" workup. Shortly after his arrest and rapid return of spontaneous circulation, that level was reported as severely elevated at 8.3 mmol/L. He immediately received sodium bicarbonate, rapid acting insulin and dextrose, and calcium chloride. His dysrhythmia quickly improved. (An ECG taken a few minutes after treatment is shown in Fig. 1.) His vital signs quickly stabilized, and he soon regained full consciousness and was extubated.

His hyperkalemia, as well as his new onset acute renal failure (the serum creatinine was 218 mmol/L) both resolved with discontinuation of his quinapril. He was eventually discharged home without deficit. His potassium and creatinine were 3.8 mmol/L and 98 mmol/L, respectively, at follow-up.

Hyperkalemia is reported to occur in 1 to 2 percent of hospitalized patients, but the incidence in the general population can only be estimated. It may arise as a result of increased potassium intake or absorption, decreased renal excretion, or trans-cellular shifts.[1] The most common clinical manifestations are ECG abnormalities and neuromuscular symptoms, such as weakness. The latter are usually overshadowed by the more urgent cardiac instability.

Cardiac electrical dysfunction, and the resulting ECG changes, arises due to partial depolarization of resting membrane potentials in excitable tissues.[2] A higher con-

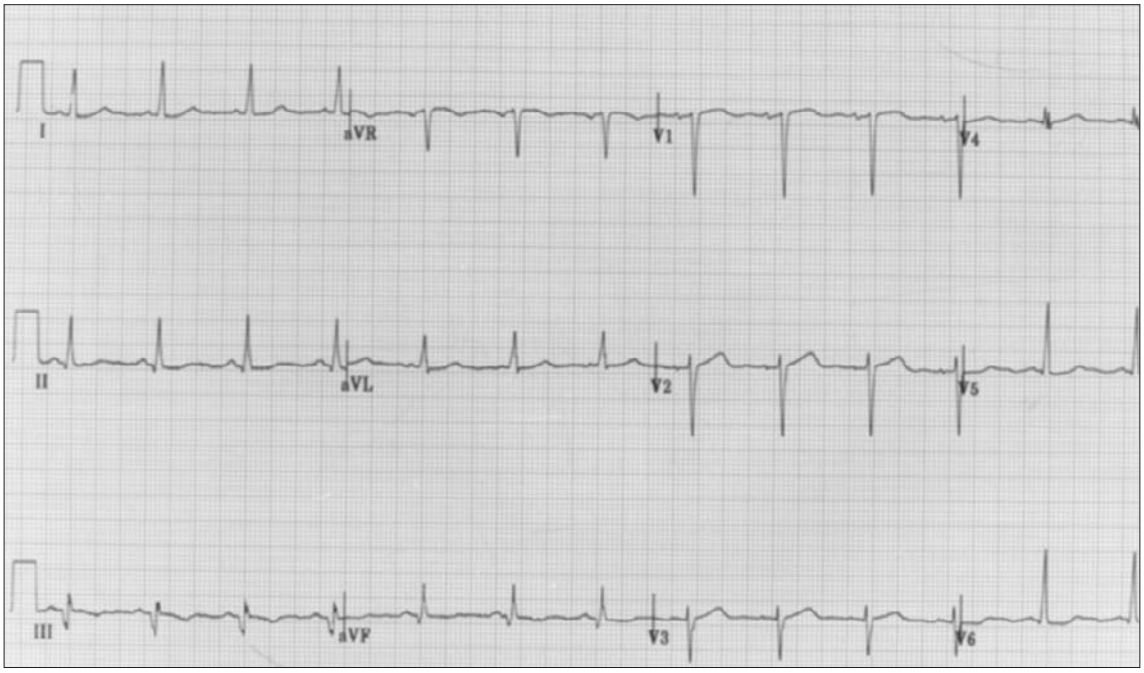

Fig. 1. Repeat twelve-lead ECG taken shortly after potassium-lowering treatment showing normal sinus rhythm. Specifically, there is no evidence of acute ischemia.

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centration of extracellular potassium will result in a larger extracellular-to-intracellular gradient, and a greater *potassium diffusion potential* (KDP). The KDP is responsible for the *resting membrane potential* (RMP), and the greater the KDP is, the less negative the RMP will be. The closer the RMP is to threshold (i.e., the less negative it is), the easier it will be to generate an action potential (AP). This explains the cardiac irritability often associated with hyperkalemia.

In general, progressive increases in serum potassium produce characteristic ECG changes and can provide valuable clues to the presence of hyperkalemia.[3] As the potassium level increases, peaking of the T waves is usually the first characteristic manifestation, followed by loss of P waves, and then widening and slurring of the QRS complexes. Eventually the tracing assumes a sine wave appearance, followed by ventricular fibrillation or asystole.

Our patient, however, presented with a bradycardia, likely junctional in origin, that rapidly progressed to asystole. His ECG showed a narrow QRS complex, without T-wave peaking. The same partial depolarization that raises the RMP closer to the threshold for generation of an AP, also closes the inactivation gates on the sodium channels, making it less likely that an AP will be generated, a process called *accommodation.*[2] This may explain the less irritable, but no less life-threatening, rhythms that may also occur, such as in our patient. As well, other factors such as the use of beta blockade and acid-base status may play a contributing role.

Keep in mind that the potassium level does not always correlate with the ECG, as life-threatening hyperkalemia can exist without these usual ECG findings. A normal ECG does not preclude treatment of known or suspected hyperkalemia, as normal cardiac conduction can quickly degenerate into a fatal arrhythmia.[3]

Competing interests: None declared.

References

1. Rosen P, Barkin R, Danzl DF, Hockberger RS, Ling LJ, Markovchick V, et al. Emergency medicine: concepts and clinical practice, 4th ed. St. Louis: Mosby; 1998. p. 2438-9.
2. Costanzo LS. Physiology, 2nd ed. Philadelphia: Saunders; 2002. p. 20.
3. Szerlip HM, Singer I. What's the significance of hyperkalemia. Diagnosis 1987;Nov:174-85.

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