Cardiac Arrest due to Hyperkalemia following Intravenous Penicillin Administration*

Charles W. Mercer, M.D. and Joseph R. Logic, M.D.

Two episodes of cardiopulmonary arrest occurred after the rapid intravenous administration of potassium penicillin G in a 60-year-old black woman with streptococcal endocarditis and sepsis. Asystole, ventricular fibrillation, and atrioventricular block suggested a positive relationship of these abnormalities to the rapid change in concentration of serum potassium produced by the injections. The fact that $1 \times 10^6$ units of potassium penicillin G contain 1.7 mEq K+ suggests that caution should be employed in the rapid intravenous administration of this antibiotic even in patients with normal serum electrolytes and a normal electrocardiogram.

The electrophysiologic effects of acute hyperkalemia on the conduction system of the heart have been studied extensively1,2 and the hazards of intravenous administration of potassium, even with electrocardiographic monitoring, have been emphasized properly.3 However, the occurrence of high grade atrioventricular block and asystole probably resulting from the potassium in an intravenous penicillin injection prompts the report of this case, and a brief review of the cardiac consequences of acute hyperkalemia.

CASE REPORT

A 60-year-old black woman was admitted to the City of Memphis Hospital medical service on September 25, 1972. On a previous admission for trauma, the presence of organic heart disease and congestive heart failure with cardiomegaly and a murmur of mitral insufficiency were noted. During the week before admission, the patient developed recurrent epistaxis and progressively increasing weakness, malaise, anorexia, decreased responsiveness and shortness of breath. The past medical history revealed heavy use of alcohol.

Physical findings included clinical evidence of dehydration, blood pressure 110/70 mm Hg, respiratory rate 24/min, pulse 100/min, and oral temperature of 100° F. Petechiae were not observed in the optic fundi and the pharynx showed no active inflammatory changes. The lungs contained medium rales at both bases. Examination of the heart localized the point of maximal impulse in the left anterior axillary line with a left ventricular heave. There was a grade 4/6 pansystolic murmur of mitral insufficiency with an associated apical

![Electrocardiogram tracings](image-url)
HERITABLE Q-T PROLONGATION

systolic thrill. Although mild epigastric and right upper quadrant tenderness was present, the liver could not be palpated, but the splenic tip was. Neither splinter hemorrhages nor peripheral edema was noted.

Laboratory data showed a hypochromic, macrocytic anemia, with hematocrit 24 percent. Initial blood urea nitrogen was 84 mg percent and serum electrolytes were normal. ASO titer was 166 Todd units. Urinalysis showed no red cells or casts. Arterial blood gases demonstrated mild alkalosis and arterial desaturation. Admission 12-lead electrocardiogram was within normal limits. The spinal fluid was normal.

The initial problems were those of sepsis and organic heart disease of unknown cause with mitral insufficiency; bacterial endocarditis was considered probable. Multiple blood cultures grew out group A, beta hemolytic streptococci, for which penicillin therapy was indicated. Hydration and correction of the anemia was accomplished within the initial 36 hospital hours.

Antibiotic therapy was initiated on the third hospital day with 5 x 10^6 units of aqueous potassium penicillin G given intravenously every 12 hours during a 45-60 minute period. On the fourth hospital day, while the penicillin was inadvertently given as an intravenous "push," the patient's eyes and head deviated to the right and she became unresponsive. An electrocardiogram revealed ventricular fibrillation from which she was quickly resuscitated, but because of persistent mild hypotension and hypoxia, she was transferred to the intensive cardiopulmonary care unit. Throughout this acute episode there was no clinical evidence to suggest the occurrence of systemic anaphylaxis or acute pulmonary embolization. The post-arrest ECG and chest x-ray film were unchanged from those on admission. On the eighth hospital day, she again received 4 x 10^6 units of the same penicillin preparation intravenously in a drip inadvertently given rapidly in a 10-15 min period. Another episode of cardiorespiratory arrest occurred during which the electrocardiographic rhythm strips in Figure 1 were obtained.

Further resuscitation was not necessary. Arterial gases obtained at the time of arrest showed only mild respiratory alkalosis (pH 7.51, PCO2 25 mm Hg). Serum electrolytes obtained earlier in the day reported a serum potassium concentration of 4.0 mEq/L. The patient continued to improve with appropriate daily penicillin therapy for bacterial endocarditis.

The potassium content of the injection given was determined by diluting 1 x 10^6 units of penicillin to a volume of one liter. The concentration, as expected, was 1.7 mEq K+/L.4

**DISCUSSION**

The occurrence of cardiopulmonary arrest during the relatively rapid injection of penicillin-G containing a total of 6.8 and 8.5 mEq of potassium (K+) on two different occasions requires the reemphasis of the consequences of rapidly changing serum K+ concentration on the cardiac conducting and pacemaker fibers of the heart. It has been shown that differences in sensitivity to the absolute levels and rate of change of levels of serum K+ exist for various cardiac tissues. The S-A nodal and the larger fibers of the internodal and intraventricular conducting systems are the most resistant to block due to K+ excess.5 This may account for the occurrence of sinoventricular conduction with absent P waves in hyperkalemia. Experimental studies have shown that block can occur at any level of the conduction system during hyperkalemia2 and that rapid increases in serum K+ can result in total suppression of pacemaker activity.3 In this patient, high grade A-V block, a Wenckebach type of A-V conduction delay, intraventricular block, asystole and ventricular fibrillation occurred when penicillin containing K+ was given intravenously. This complication of the intravenous administration of penicillin G could easily have been fatal. Sodium penicillin G would probably have been a safer drug were there sufficient cause for such large amounts of penicillin being given intravenously as a bolus.

**REFERENCES**


Heritable Q-T Prolongation without Congenital Deafness
(Romano-Ward Syndrome)*

Miklós Csanády, M.D., and Zoltán Kiss, M.D.

An additional case of the Romano-Ward syndrome is reported. In three members of a family, the Q-T intervals on the ECGs were found to be prolonged. One of the members was resuscitated on several occasions from Adams-Stokes attacks which occurred the first day postpartum and in subsequent premenstrual periods; these attacks were caused by ventricular fibrillation. The beta-adrenergic blocking drug propranolol proved to be an effective agent in preventing attacks. Given in a relatively small maintenance dose, the patient remained free from complaints, despite the unchanged prolongation of the Q-T interval.

There are only a few reported families in which the effected members frequently suffer from ventricular arrhythmias and sudden death occurs most often at an early age. The T and U waves of the electrocardiogram produce a common complex and exhibit a bizarre shape; the Q-T or Q-U interval is prolonged. There are no other accompanying familiar anomalies (such as deafness, for example, or a tendency to extracellular hypokalemia).

*From the First Department of Medicine, University Medical School, Szeged, Hungary.

Reprint requests: Dr. Csanády, First Department of Medicine, 6720 Szeged, Hungary