Reverse Wenckebach Block and Complete Atrioventricular Dissociation due to Potassium and Digitalis

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The relation of digitalis to potassium was studied by administering potassium intravenously before and after digitalization. The disturbances of conduction due to potassium and those occurring after digitalis were observed and are here discussed.

Potassium is an effective agent for the control of arrhythmias associated with digitalis and hypopotassemia and of many arrhythmias of obscure etiology.1-5 The therapeutic effect of potassium in cardiac irregularities caused by digitalis has been ascribed to a digitalis and potassium "antagonism." For a demonstration of this antagonism, potassium was administered intravenously to patients with atrial fibrillation after they were "fully" digitalized. The end point of digitalization was shown by a marked depression of atrioventricular (A-V) conduction and the appearance of A-V nodal escape beats, A-V nodal rhythm, ventricular bigeminy with varying configuration of the ectopic beats and, finally, failure to speed significantly the ventricular rate by intravenous administration of 1.25 to 2.0 mg. of atropine. It was assumed that in such patients, after treatment with potassium, the digitalis-potassium antagonism would be manifested by an increase of conduction via A-V node with disappearance of A-V nodal arrhythmias and speeding of ventricular rate. Surprisingly, however, in the majority of patients the conduction in the A-V node was depressed further with the appearance of complete A-V dissociation, ventricular escape beats, and idioventricular rhythm. In short, the electrocardiogram looked as if the digitalis had exerted a greater effect and not been antagonized by the potassium.6

The case reported in this paper is one of a large number of cases of atrial fibrillation, treated with potassium over the past 2 years. It is considered that this particular case is worthy of detailed description as it demonstrates (1) reverse Wenckebach periods, an extremely rare phenomenon, (2) the ability of potassium to cause complete A-V dissociation, a finding not previously observed in human beings in the presence of sinus rhythm,7-8 (3) the obvious "additive effect" of potassium and digitalis, and (4) hazards of rapid administration of potassium to an overdigitalized patient, as contrasted with one who is not digitalized.

CASE REPORT

An 82-year-old man was admitted to Indianapolis General Hospital for the control of atrial fibrillation caused by coronary heart disease. The patient had not been previously treated for heart disease and had never received digitalis. Significant physical findings were atrial fibrillation and moderate cardiac enlargement. No clinical signs of heart failure were noted. Laboratory procedures, including complete blood count, urinalysis and blood urea nitrogen were normal. Plasma serum potassium was 4.5 mEq./L. An electrocardiogram showed atrial fibrillation.

On May 8, 1958, 40 mEq. of an isotonic solution of potassium phosphate in distilled water was administered intravenously over a period of 50 minutes. The rate of infusion varied from 30 drops to 180 drops per minute, depending on the severity of pain at the site of injection. Figure 1 shows the effect of potassium, Strips 1 and 2 (control tracing) disclose atrial fibrillation with a ventricular rate of 90 beats per minute and aberrant ventricular conduction. Within 3 minutes of onset of rapid infusion (180 drops per minute) the aberrant conduction became less frequent (strip 3), finally disappeared and did not reappear during the period of observation. The conduction through the A-V node and the ventricular rate remained unchanged (strip 4).

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In the next week the patient was given 2.0 mg. of digitoxin. On May 15, 1958, the day the infusion of potassium was to be repeated, the patient exhibited sinus rhythm with delayed A-V conduction (fig. 2). The P-R interval measured 0.24 to 0.30 second. Two Wenckebach periods were noticed (strip 1). Because it was thought that the electrocardiographic and clinical findings were those of digitalis toxicity, potassium was administered in the manner described. Strip 2 illustrates changes during rapid infusion of potassium with resultant slowing of the ventricular rate from 100 to 80 beats per minute without any change of the P-R interval. This effect was probably mediated through the vagus nerve. Shortly after the infusion was slowed, the P waves became lower in amplitude, and periods of complete A-V dissociation occurred with an A-V nodal rate of 60 beats per minute and a retrograde P wave after the ninth ventricular complex (strip 3). These changes persisted for a few minutes, after which sinus rhythm with first-degree block returned. At that time the solution was changed to isotonic potassium phosphate in 5 per cent glucose, to diminish the pain at site of injection; for a short period of time rapid infusion was re instituted (strip 4). The A-V dissociation with A-V nodal rhythm promptly reappeared, P waves became smaller, and 2 ventricular escape beats were observed (beats 3 and 9). At this point the infusion was slowed from 180 drops per minute to 30 drops per minute and this rate was maintained until the entire 40 mEq. were given. Strip 5 shows the persistence of complete forward block but with preservation of retrograde conduction, as shown by the appearance of retrograde P waves after

![Fig. 1 Top. Electrocardiogram May 8, 1958.](http://circ.ahajournals.org/)

![Fig. 2 Bottom. Electrocardiogram May 15, 1958.](http://circ.ahajournals.org/)
complexes 2 and 9 with an R-P of 0.40 to 0.48 second measured from beginning of R to nadir to P. The R-P interval following the last beat in this strip is 0.40 second. Strip 6 shows complete unidirectional block. Retrograde P waves are present after ventricular beats 3, 4, and 5 with a R-P of 0.24, 0.40 and 0.48 second respectively, and again after beats 9 and 10 with a R-P of 0.40 and 0.46 second. It is uncertain whether or not a retrograde P wave is buried in the S-T segment of ventricular complex 8. Shortly after the infusion was discontinued sinus rhythm returned, first alternating with second-degree block and stabilizing 30 minutes after the infusion was discontinued as a first-degree block with occasional Wenckebach periods (a situation identical to that present before infusion of potassium). This rhythm remained unchanged during the period of observation (strip 7).

COMMENT

There is no doubt that this tracing demonstrates periods of unidirectional (forward) heart block as well as periods of complete block. In the former the retrograde conduction from the A-V node to the atria has the classical Wenckebach structure. In strip 6, 3 consecutive retrograde P waves are visible; the largest increment in time of conduction toward atria is between the first and second complex (0.16 second), and this increase is less between the second and third P wave (0.08 second). The disturbance of A-V conduction was probably as much a result of the vagal as of the direct depressing effect of potassium on the conduction tissue.

It was thought that the failure to depress the A-V conduction, and thus to slow the ventricular rate before the patient was digitalized, was of some clinical significance. The disappearance of aberrant conduction during the initial infusion without slowing of the ventricular rate suggests improvement rather than depression of impulse conduction. The failure to slow appreciably the ventricular rate in undigitalized or poorly digitalized patients with atrial fibrillation has been confirmed many times during our study.9 One cannot escape the thought that in this case the digitalis has in some way potentiated the toxic effects of potassium. That the reverse is less likely is attested to by the transient nature of the changes observed. The substitution of isotonic potassium phosphate in 5 per cent glucose for the same in distilled water does not confuse the immediate results. The slowing of sinus rhythm, complete A-V dissociation and retrograde conduction appeared before the solution was changed (strips 1-3). It is impossible to state with certainty whether or not the glucose, by combining with potassium subsequently affected the serum or intracellular myocardial potassium, and thus contributed to the toxic effects observed.

It seems reasonable to assume that digitalis toxicity, which becomes manifest as a result of potassium depletion,10 may represent an entirely different problem from toxicity due primarily to administration of large doses of digitalis. In the former potassium is beneficial; in the latter, however, it may aggravate the situation by its well-known nonspecific depressing effect on the heart. Further study of this problem is essential.

SUMMARY

A case is presented in which (1) the toxic effects of potassium on the myocardium became manifest only after digitalization, (2) the effect of potassium was one of depression of atrioventricular conduction to the point of complete atrioventricular dissociation, with periods of retrograde conduction manifesting the classical Wenckebach structure. The "additive" effects of potassium and digitalis are stressed.

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SUMMARIO IN INTERLINGUA

Es presentate un caso in que (1) le effectos toxic de kalium super le myocardio deveniva manifeste solmente post digitalisation e in que (2) le effecto le kalium consisteva in le depression del conduction atrioventricular usque a su dissociation complete, con periodos de conduction retrograde exhibiente le classic structura de Wenckebach.

Le effectos "additive" de kalium e digitalis es ablineate.
REFERENCES


Rupture of a papillary muscle of the heart has been considered an unusual complication of myocardial infarction. Although first recognized as early as 1803, only 56 cases have been reported to date. Five additional cases are presented in this paper. Rupture of the posterior papillary muscle of the left ventricle is 6 to 12 times more common than rupture of the anterior one, and the former is usually associated with posterior myocardial infarction. Diagnosis of rupture of the papillary muscle should be considered in the presence of acute myocardial infarction, when there suddenly develops a loud apical systolic murmur with sudden onset of shock, dyspnea, and pulmonary edema. The prognosis is extremely poor. Differential diagnosis includes acute cardiac dilatation with mitral insufficiency and perforation of the interventricular septum. Anterior myocardial infarction is found in 75 per cent of the cases of septal perforation, while posterior infarction is just as common among cases of papillary muscle rupture.

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