Treatment of Refractory Ventricular Tachycardia and Fibrillation by the Administration of Potassium and Quinidine*

Report of a Case

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Although quinidine and procaine amide are the most effective pharmacologic agents utilized in the treatment of ventricular tachycardia and ventricular fibrillation, it is not unusual to find instances of ventricular tachycardia refractory to quinidine. Because potassium is rarely used in the treatment of ventricular tachycardia,\(^1\) the following gratifying experience with this drug used in conjunction with quinidine is reported.

Case Report

A 47-year-old white man was seen in consultation because of refractory paroxysmal ventricular tachycardia and ventricular fibrillation of eight weeks duration. During this period, there were 14 episodes of ventricular tachycardia and/or fibrillation associated with frequent convulsions. Subternal "discomfort" radiating to the right shoulder and arm usually occurred immediately after the onset of ventricular tachycardia. On examination, he was a well-developed and adequately nourished, apprehensive man without dyspnea, orthopnea, edema, clubbing of digits, obesity or cyanosis. No significant arteriosclerotic changes were found in the retinal arterioles or peripheral arteries. The jugular venous pulse and thyroid gland were not abnormal, and the lung fields were clear. There was a normal left ventricular impulse palpable in the fourth left intercostal space just medial to the mid-clavicular line. The first apical sound was of good quality and there was no abnormal splitting or accentuation of the aortic or pulmonic components of the second sound. The liver and spleen were not enlarged.

Extensive serial laboratory determination including serum electrolytes, blood sugar, urea nitrogen, 5-hydroxyindole acetic acid, urinary catecholamine, 17-ketosteroids, and 11-hydroxycorticosteroids; C-reactive protein and lupus erythematosus cell preparations remained normal. Results of liver, thyroid and renal function tests were adequate, and the plasma pH was 7.41 and the serum cholesterol 220 mg. per cent. Following episodes of ventricular fibrillation and external defibrillation, liberation of serum glutamic oxalacetic transaminase increased from normal up to 255 Karmen units. Several electrocardiograms demonstrated ventricular tachycardia at a rate of 280 per minute (Fig. 1B). Serial electrocardiograms and a previous Master 2-step test were normal between the periods of ventricular tachycardia (Fig. 1A). Immediately following one episode of ventricular tachycardia, flutter and fibrillation, the ST segment and T wave changes were characteristic of acute coronary insufficiency (Fig. 1C,D). However, the electrocardiogram returned to normal within one hour (Fig. 1E). Roentgenograms of the chest, heart, gall bladder, gastrointestinal tract, and renal system demonstrated no evidence of pathology.

On the seventh hospital day, he had the first hospital episode of ventricular tachycardia. He first noted the onset of a rapid heart rate immediately followed by anterior chest pain radiating to the right arm. The pulse was fairly regular at a rate of 180 beats per minute. He then developed a generalized tonic seizure followed by apnea, deep coma, profound cyanosis and a convolution lasting for two to three minutes. The apex beat and carotid pulse were unobtainable and the pupils became fixed. After the convolution and return of consciousness, the patient became relaxed and the apical pulse, blood pressure and respirations slowly returned to normal. On several occasions, external cardiac massage was necessary for resuscitation and on three occasions, external defibrillation was required to terminate ventricular fibrillation (Fig. 1B,C). These episodes increased in frequency in spite of an intensive pharmacologic program consisting of barbiturates, chlorpromazine, papaverine, meperidine hydrochloride, warfarin sodium, diphenylhydantoin sodium, pentaerythritol tetranitrate, intravenous adrenal corticotrophic hormone, dichloroisoproterenol, procaine amide, and quinidine sulphate. On the 21st hospital day, it was suggested that all medications be discontinued. Potassium chloride, 120 mEq. daily, was initially administered intravenously and then 1.0 gm. was administered orally with 0.4 gm. of quinidine sulphate every six hours. This program raised the serum potassium level from 4.5 mEq. per liter to 6.0

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mEq. per liter and the 20 hour quinidine level to 4.6 mEq. per liter of plasma. No further episodes of tachycardia occurred. Two weeks later, inadvertently, the midnight medication was omitted and at one o'clock in the morning another episode of ventricular tachycardia developed, but spontaneously reverted to a normal rhythm after four to five minutes. During the past 18 months, 4.0 gm. of potassium chloride and 3.2 gm. of quinidine sulphate have been administered daily in divided doses without complication, and there has been no recurrence of ventricular tachycardia or chest pain. A recent electrocardiogram remains normal (Fig. 1F). Although difficult to exclude the possibility of underlying coronary artery disease in this instance, the etiology of this arrhythmia remains obscure. Ventricular tachycardia, one of the most serious, significant, and perplexing of all the arrhythmias, usually indicates serious underlying organic heart disease. On occasions, this arrhythmia occurs spontaneously in an otherwise normal heart.4

**DISCUSSION**

Recent electro-physiologic studies of cardiac muscle have demonstrated mechanisms which may be responsible for the production of ventricular tachycardia. Although intrinsic rhythmicity is present in all specialized fibers, slow diastolic depolarization is found in a few and these latent pacemakers generate ectopic activity whenever the normal pacemaker is depressed. Under normal conditions, many fibers show little evidence of intrinsic rhythmicity, but once subjected to physical or chemical stimuli, develop rapid pacemaker activity.

At a time when such sophisticated procedures as bilateral cardiac sympathectomy7 and external defibrillation6 are advocated for refractory ventricular tachycardia, it seems most important to restate the prom-

![Refractory Ventricular Tachycardia and Fibrillation Controlled](image-url)
inent role potassium plays in the function of cardiac muscle. In 1882, Ringer's classic experiments first demonstrated the beneficial effect of potassium on cardiac muscle and in 1903, Hering terminated ventricular tachycardia and ventricular fibrillation by the intravenous injection of potassium chloride in the dog. Because of the narrow margin of safety between an effective and lethal dose of potassium following intravenous administration, potassium was not utilized in the treatment of cardiac arrhythmias until Sampson and Anderson in 1930, first successfully employed potassium chloride orally in the treatment of ventricular premature contractions and ventricular tachycardia. Myocardial excitability depends upon the ability of the sensitive cell membrane to rapidly transfer potassium out with depolarization, and to return potassium back into the cell with repolarization. Digitalis reduces potassium membrane transfer rates in cardiac muscle and causes loss of cell potassium during the intermediate unsteady state. Quinidine, however, tends to stabilize membrane permeability, thereby preventing the transfer of potassium across the cell membrane. Apparently the stabilizing action of quinidine on potassium flux is more important than depression of excitability. Ventricular tachycardia and fibrillation produce myocardial damage which in turn is accompanied by an intracellular loss of potassium and a gain of sodium. The replacement of potassium to the intracellular environment promptly modifies ventricular irritability. Because potassium excretion occurs mainly by way of the urinary system and little by way of the gastrointestinal tract, there is slight danger of toxicity from oral administration providing there is an adequate renal output. If renal function is impaired, additional potassium, however, may prove lethal.

The electrocardiogram may be utilized to detect alterations of intracellular potassium concentration. Any change in potassium concentration promptly results in disturbances of cardiac conduction and potential which are readily transcribed on the electrocardiogram. References


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