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**Hyperkalemic Cardiac Arrhythmia Secondary to Spironolactone**

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Although multiple adverse reactions can occur during the use of spironolactone, the most serious complication is related to its potassium-sparing effect which may induce dangerous and even fatal hyperkalemia. One patient is presented who developed severe hyperkalemia after spironolactone resulting in ventricular standstill and survival following treatment with sodium bicarbonate.

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**CASE REPORT**

A 69-year-old man was brought to the emergency room of Methodist Hospital of Brooklyn in a state of cardiovascular collapse, intensely cyanotic, without pain, and with a level of consciousness inconsistent with his clinical state. A brief physical examination revealed blood pressure 60 mm Hg by palpation, and an irregular heart rate of 20-25 beats per minute. Because of the fear of impending cardiac arrest, resuscitative measures were instituted. Following initial electrocardiogram (Fig 1A) which revealed the pattern of a dying heart with a ventricular rate of 13 beats per minute, atropine sulfate, 0.4 mg, was given intravenously and an isoproterenol drip was started. Almost simultaneously, sodium bicarbonate, 44.6 mEq was given intravenously to correct probable metabolic acidosis in the setting of clinical shock. A similar dose was repeated after a five minute period. After the second dose of bicarbonate, the electrocardiogram revealed bizarre QRS complexes occurring with such regularity as to suggest the presence of an AV junctional tachycardia.

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**FIGURE 1A** Emergency room electrocardiographic strip revealing the pattern of a dying heart with a ventricular rate of 13 beats/min. B. AV junctional rhythm approximately five minutes after 44.6 mEq of intravenous sodium bicarbonate. C. AV junction tachycardia ten minutes later—note the decreased widening of the QRS complex.

Skin rash, gastrointestinal irritation, hirsutism, gynecomastia, and muscular weakness are adverse reactions related to the use of the potassium-sparing diuretic, spironolactone. The side effects are usually infrequent and mild. The most serious complication is related to its potassium-sparing effect which may induce dangerous and even fatal hyperkalemia. The following case report presents an occurrence of severe hyperkalemia after the excessive use of spironolactone, resulting in ventricular standstill, and survival after treatment with sodium bicarbonate.
There was no atrial activity noted (Fig 1B). The isoproterenol was discontinued and a sodium bicarbonate infusion of 44.6 mEq in 500 ml of 5 percent D/W was started. The clinical status within ten minutes greatly improved with the blood pressure reading 120/90 mm Hg and the heart rate being 60-65 beats per minute. Electrolytes, drawn initially and repeated within one half hour revealed serum potassium, sodium, and chloride to be 7.9, 127, and 88 mEq/L, respectively. The electrocardiogram (Fig 1C) now revealed a more rapid AV junctional tachycardia at 110 beats per minute with slightly less widening of the QRS complex.

A detailed history obtained after clinical improvement revealed the ingestion of spironolactone, 150 mg per day for the past four months, orange juice, one quart daily, and digoxin 0.25 mg daily, all for the treatment of congestive heart failure. The excessive dosage of spironolactone was secondary to self-treatment with the medication supplied by his pharmacist's son. Prior to admission, muscular weakness was prominent. One hour after admission the serum potassium was 7.1 mEq/L. Exchange resin, sodium polystyrene sulfonate (Kayexalate), and sorbitol were started orally. Six hours later the serum potassium was 6.5 mEq/L. The electrocardiogram (Fig 2) showed atrial fibrillation with a moderate ventricular response and normal QRS complexes. The only other pertinent findings were a slightly elevated BUN of 33 mg percent and a urine creatinine clearance of 45.8 ml/min/m².

**FIGURE 2.** Six hours after admission the electrocardiogram showed atrial fibrillation with normal ventricular response and normal QRS complexes.

**DISCUSSION**

The use of sodium bicarbonate in unmasking severe hyperkalemia is clearly demonstrated by this patient. The diagnosis was suggested by the rapid improvement in the electrocardiogram from a very slow ventricular rate with bizarre QRS complexes to atrial fibrillation with normal intraventricular conduction and a rapid improvement in clinical status following intravenous bicarbonate. It is of interest that this patient was conscious despite serious electrocardiographic changes of a dying heart and severe serum electrolyte abnormalities. Confirmation of the diagnosis was obtained by serum electrolyte analysis and history of excessive ingestion of a potassium-sparing diuretic and large volumes of orange juice daily.

The effect of hyperkalemia on the action potential of cardiac muscle has been described. Progressive increase in extracellular potassium concentration produces progressive decrease in the resting membrane potential. This, in turn, decreases the upstroke velocity and results in a slow intraventricular conduction and increased duration of the QRS complex on conventional electro-
Pacemaker Function in Relation to Electroconvulsive Therapy

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The use of electroconvulsive therapy (ECT) is reported in a patient with a permanent transvenous demand pacemaker (Medtronic 5841). Although electrocardiographic monitoring by telemetry did not demonstrate arrhythmias, ECT is capable of interfering with the proper function of certain types of pacemakers because of the electrical frequencies employed. Those patients who require ECT should undergo careful evaluation of possible contributing risk factors.

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There are many electrical phenomena that may interfere with pacemaker function. These include medical and surgical diathermy, ultraviolet and neon light, ignition systems, betatron units, microovens, power transmission lines, radio frequency transmissions, including radio telemetry, and radar warning systems. In view of the number of permanent pacemakers that are being implanted, it is important to evaluate those patients who are exposed to therapeutic electrical appliances. We wish to report our experiences with a patient who had repeated electroshock therapy since there are little data available indicating the safety of this type of treatment.

CASE REPORT

The patient, a woman aged 71, had a permanent transvenous demand pacemaker unit (Medtronic 5841) implanted subcutaneously in a right subclavicular pocket. The bipolar pacing catheter was wedged at the apex of the right ventricle. This was for Mobitz type 2 atrioventricular block associated with episodes of syncope. One month later she was noted to be severely depressed and underwent ECT on 12 occasions. Methohexital sodium (Brevital) and succinylcholine were the anesthetic and muscle relaxant used.

The method of electroshock delivery was as follows: A Reiter electrostimulator (CW 47) was used with the electrodes applied to the right temporal region in a coronal direction. Initial setting of 30 ma was gradually reduced to 10 ma over a period of 40 seconds. Using a 1500 ohm load, the average potential difference at the output terminals of this machine at 30 ma was 27 v with a peak of 500 v. The electrical frequency of the current was 28 Hz and the maximal applied energy was 12.5 joules per second. During this time the ECG was monitored using an RKG radioelectrocardiography system with a bipolar lead placement, one lead over the xiphisternum and the other at the angle of the left scapula. The demand mode of the pacemaker was in constant operation prior to the procedure. During the time of application of electroshock the ECG could be monitored only crudely, but there was no evidence of interference with pacemaker initiation (Fig 1). A battery-operated ECG machine was also used as an alternative method of monitoring but was unsuccessful due to gross interference.

DISCUSSION

It is inevitable that patients with pacemakers will be considered for ECT and it is therefore necessary to identify any problem that could arise. The only previous report available was that of Youmans and co-workers who observed no complications during ECT in a patient who had an epicardial asynchronous fixed-rate pacemaker (Medtronic 5870). The Medtronic system is built to withstand 500 v and currents of 1 to 2 amp and has appropriate shielding with the use of shunt diodes on pacemaker input-output terminals. Nevertheless a break in insulation or a fractured electrode wire could certainly create a potential hazard. The situation is quite different in the case of synchronous pacemakers whose operation is controlled by electric signals picked up by the stimulating electrodes. In the true demand pacemaker potentials of less than 0.5 mv which lie in the frequency range of the QRS complex (20 to 40 Hz) will inhibit the pacemaker for as long as they are present plus about 0.8