

# electrocardiogram of the month

## Transient Digitoxic Double Tachycardia\*

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**S**imultaneous atrial and ventricular bidirectional tachycardia is a serious arrhythmia that is frequently induced by excessive digitalis administration and is associated with high mortality.<sup>1-8</sup> In this report, we describe such an arrhythmia that occurred transiently during intravenous (IV) administration of a therapeutic dose of digitalis. The arrhythmia resolved when further digitalis administration was discontinued and the patient was hydrated.

### CASE REPORT

The patient, a 62-year-old man, was admitted to the Brooklyn VA Medical Center at 12:00 AM with a two-week history of increasing shortness of breath, paroxysmal nocturnal dyspnea, leg edema, palpitation, cough, and weight gain of 10 kg. He was a nonsmoker and nondiabetic, with no history of chest pain, prior myocardial

infarction, renal disease, thyroid disorder, or rheumatic fever, but he had a five-year history of hypertension. The only medication the patient was taking at the time of admission was furosemide. Physical examination revealed an emaciated elderly man in moderate respiratory distress. His blood pressure was 115/65 mm Hg, pulse rate 150 beats/minute, irregularly irregular; respiratory rate 30 to 35/minute; and temperature 36.8°C (rectal). There was no jugular venous distention or carotid bruit. Examination of the chest revealed dullness to percussion of both lung bases and decreased vocal fremitus and breath sounds with a few scattered rales. On cardiac examination the patient had variable S<sub>1</sub>, increased P<sub>2</sub>, S<sub>3</sub> gallop, but no murmur or click. Liver span was 11 cm, and the lower extremities had pitting edema up to the knees.

The chest roentgenogram showed cardiomegaly, bilateral pleural effusion, and pulmonary vascular congestion. A 12-lead ECG showed an old anteroseptal infarction. The cardiac rhythm was atrial fibrillation with an average ventricular response of 150 beats/minute. The patient was given 0.5 mg of digoxin IV, followed by 0.25 mg every two hours to slow the ventricular response. Figure 1A was obtained after 0.75 mg of digoxin was given and shows atrial fibrillation with moderate slowing of ventricular rate to 115 beats/minute. Figure 1B was obtained after a total of 1.25 mg of digoxin was

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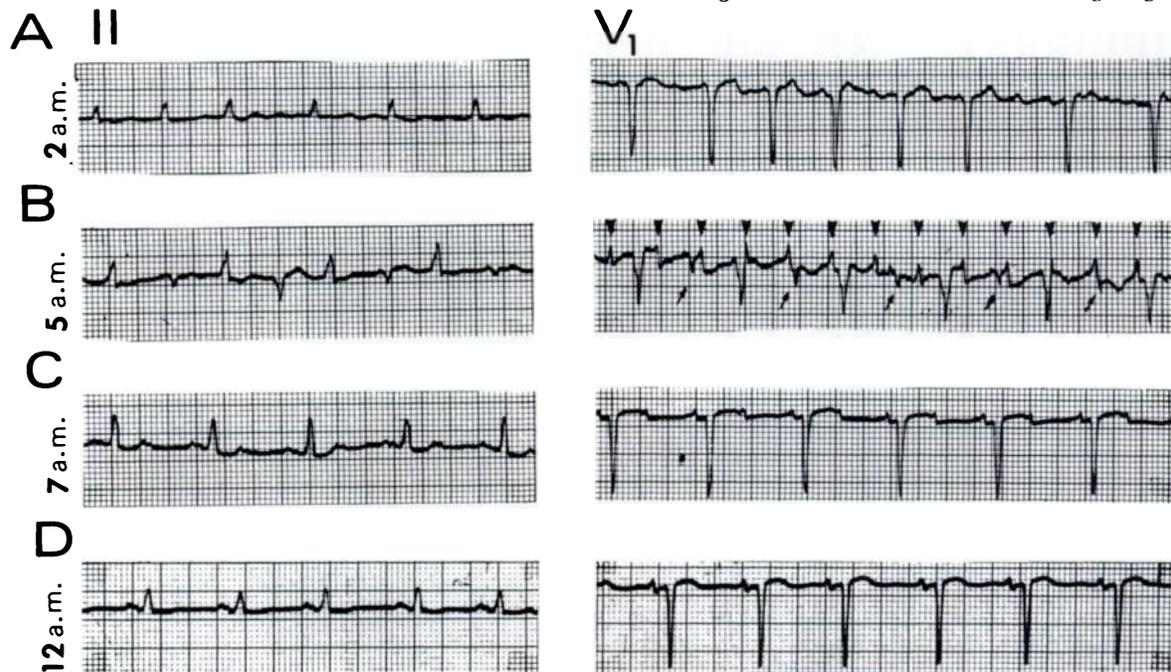


FIGURE 1. Rhythm strips (leads 2 and V<sub>1</sub>) recorded at various times after digitalis administration. Panel A, rhythm strips obtained after IV administration 0.75 mg of digoxin; panel B, after 1.25 mg of digoxin, panels C and D, two and seven hours later, during which digoxin was withheld. Arrowheads (panel B) indicate P waves, and small arrows, one set of the alternating QRS complexes masked by P waves.

given and shows the development of simultaneous atrial tachycardia and bidirectional ventricular tachycardia. The atrial rate was 180 beats/minute and the P waves were best seen in lead V<sub>1</sub>. The atrial activity was completely dissociated from the slower ventricular activity at a rate of 162 beats/minute. The ventricular rhythm was regular and the alternately directed ventricular complexes had a frontal plane QRS axis of +95° and -40°. At that time the laboratory data were as follows: pH, 7.46; PaO<sub>2</sub>, 66 mm Hg; PaCO<sub>2</sub>, 36 mm Hg; HCO<sub>3</sub><sup>-</sup>, 19 mEq/L; BUN, 106 mg/dl; serum creatinine, 2.6 mg/dl; serum potassium, 6 mEq/L; serum sodium, 135 mEq/L; glucose, 184 mg/dl. Hemoglobin concentration was 16.5 g/dl; WBCs, 12,600/mm<sup>3</sup>, with 79 percent polymorphonuclear cells, 5 percent stabs, and 16 percent lymphocytes. The renal insufficiency was thought to be secondary to marked dehydration, and the cardiac rhythm was thought to be due to toxicity to digitalis toxicity. The patient was transferred to the coronary care unit, digoxin treatment was withheld, and blood was drawn for digoxin level.

In the coronary care unit the ECG showed atrial tachycardia with 2:1 atrioventricular conduction (Fig 1C). The rate of the atrial rhythm of 160 beats/minute was slower compared with that of Figure 1B, and the configuration of the P waves was different. The bidirectional ventricular tachycardia had spontaneously terminated. The patient was hydrated, and in the next few hours a repeated ECG showed resumption of sinus rhythm (Fig 1D). The serum digoxin level at the time of double tachycardia turned out to be 3.5 ng/ml.

The following day, with adequate hydration, BUN was 27 mg/dl; creatinine, 1.9 mg/dl; serum potassium level, 3.7 mEq/L; and repeated digoxin level was 2.2 ng/ml. The patient was then transferred out of the coronary care unit.

## DISCUSSION

There is a general agreement that bidirectional ventricular tachycardia is an advanced digitoxic rhythm associated with severe underlying myocardial dysfunction in the overwhelming majority of the cases. In a review of cases by Hellman and Lind,<sup>1</sup> 30 of 34 cases had fatal outcome. In 26 cases, the deaths occurred soon after the arrhythmia was diagnosed. By 1973, 72 cases of bidirectional tachycardia was reported in the literature.<sup>2</sup> Forty-four percent of the patients had atrial fibrillation, and 82 percent had been receiving digitalis. Like bidirectional tachycardia, double tachycardia (defined as simultaneous organized atrial and junctional or ventricular tachycardia with atrioventricular dissociation) has been recognized as a distinct entity since first described by Gallavardin et al in 1920.<sup>3</sup> In 1952 Bernstein et al<sup>4</sup> presented seven cases, and in 1960 Castellanos et al<sup>5</sup> reviewed 35 previously described cases and presented 15 new cases. In 1972, Wishner et al<sup>6</sup> reviewed 11 more cases of double tachycardia and a few additional case reports were published recently.<sup>7,8</sup> Castellanos et al<sup>5</sup> described three patients with simultaneous atrial tachycardia and bidirectional tachycardia similar to the one in the present report. One of their patients had received a small dose of ouabain, and the double tachycardia was thought to be a hypersensitivity reaction. As in bidirectional tachycardia, double tachycardia is predominantly a digitoxic arrhythmia (over 75 percent)<sup>5</sup> with mortality in excess of 73 percent.<sup>6</sup>

The present case report highlights certain features of digitoxic rhythm not generally appreciated. Simultaneous atrial tachycardia and bidirectional ventricular tachycardia were precipitated during IV administration of a standard digitalizing dose for the control of atrial fibrillation with rapid ventricular response. A dosage of 1.25 mg digoxin over

several hours to control atrial fibrillation with rapid ventricular response in a patient who gave no history of being on digitalis is considered within therapeutic range. At that time the renal insufficiency secondary to marked dehydration was not appreciated. However, renal insufficiency does not usually require modification of the loading dose of digitalis, but rather the maintenance dose.<sup>9</sup> To our knowledge simultaneous precipitation of both these arrhythmias in a patient with atrial fibrillation has not been reported. Also, both of these arrhythmias resolved spontaneously with resumption of sinus rhythm over a short period without electrolyte manipulation and/or pharmacologic intervention. This suggests that the development of digitoxic cardiac rhythm involves not only well-known factors, such as the serum digoxin level and the relationship of extracellular to intracellular potassium level (not always reflected by the serum potassium level), but also some poorly understood factors such as individual susceptibility as well as increased vulnerability of the enlarged and diseased heart.

Atrial tachycardia with atrioventricular block and bidirectional tachycardia as manifestations of digitalis toxicity also highlight two characteristic pathophysiologic effects of the drug, *ie*, acceleration of ectopic pacemakers and impairment of atrioventricular conduction.<sup>10</sup> Acceleration of ectopic pacemaker may be due to enhanced diastolic depolarization or digitalis-induced oscillatory afterpotential (triggered activity).<sup>11-13</sup> The present report suggests that digitoxic double tachycardia may not always have the grave prognosis reported in the literature as long as digitalis toxicity is suspected, digitalis is discontinued promptly, and fluid and electrolyte imbalance is corrected.

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