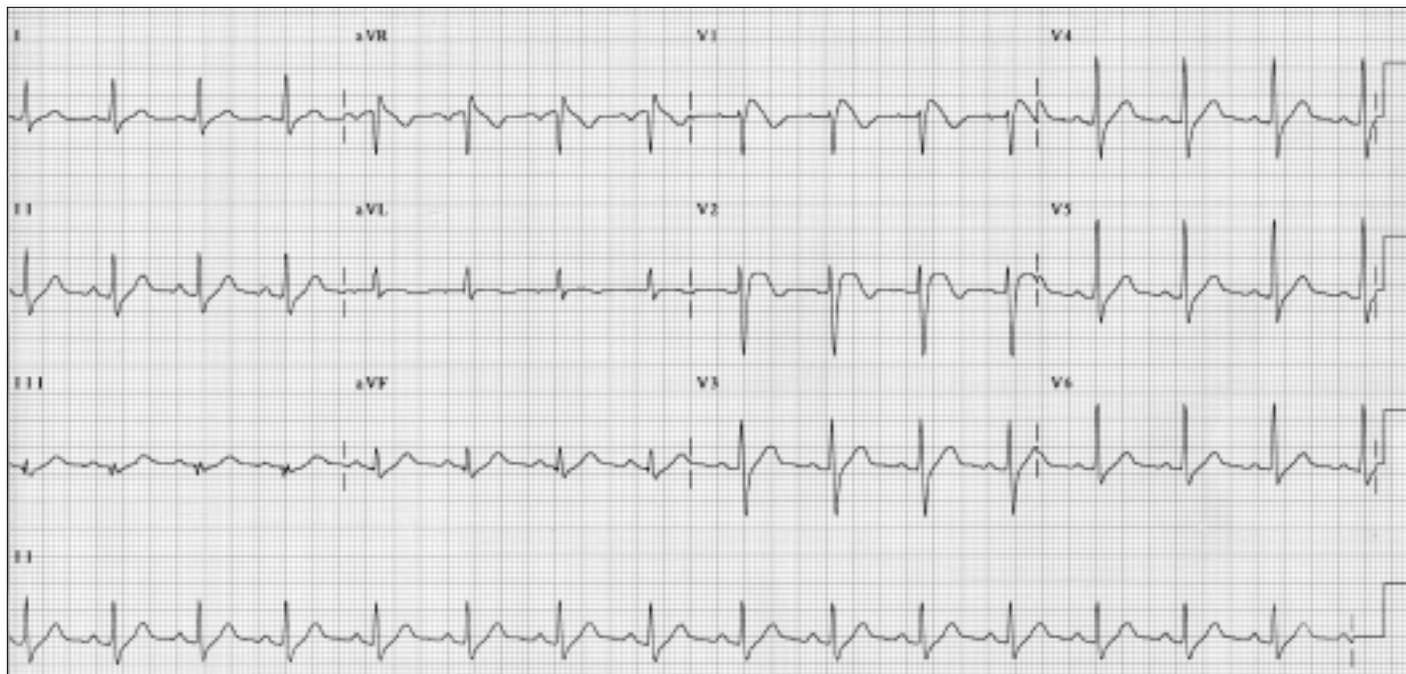


## Syncope in a young man

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**Figure 1.** Electrocardiogram recorded in the emergency department shows incomplete right bundle branch block and elevated and coved ST segments with inverted T waves in leads  $V_1$  and  $V_2$ .

**A** 33-year-old man awakened early with warmth, diaphoresis, and palpitations. He went to the kitchen to drink water, felt light-headed, and fell to the floor. When he got up, he lost consciousness completely and fell again, lacerating his chin and chipping his tooth. On awakening, he felt cold and clammy and went to the emergency department.

History revealed that 2 days earlier the patient had been started on treatment for depression with citalopram, a selective serotonin reuptake inhibitor, and trazodone, a tricyclic antidepressant. Aside from the evidence of trauma, physical examination findings were normal, including vital signs. Blood pressure did not fall when he stood. Blood counts, serum electrolytes, and thyroxine and thyroid-stimulating hormone levels were normal. A urinary toxicology screening test was negative, including no evidence of tricyclic antidepressants or cocaine. Initial values of creatine kinase (CK) (170 IU/L) and its MB fraction (CK-MB) (1.4 ng/mL) were normal. The next day, both were slightly elevated (249 IU/L and 13.8 ng/mL, respectively). An electrocardiogram did not show evidence of myocardial infarction but suggested the Brugada syndrome (Figure 1). An echocardiogram

showed normal left ventricular function with no significant wall-motion abnormality.

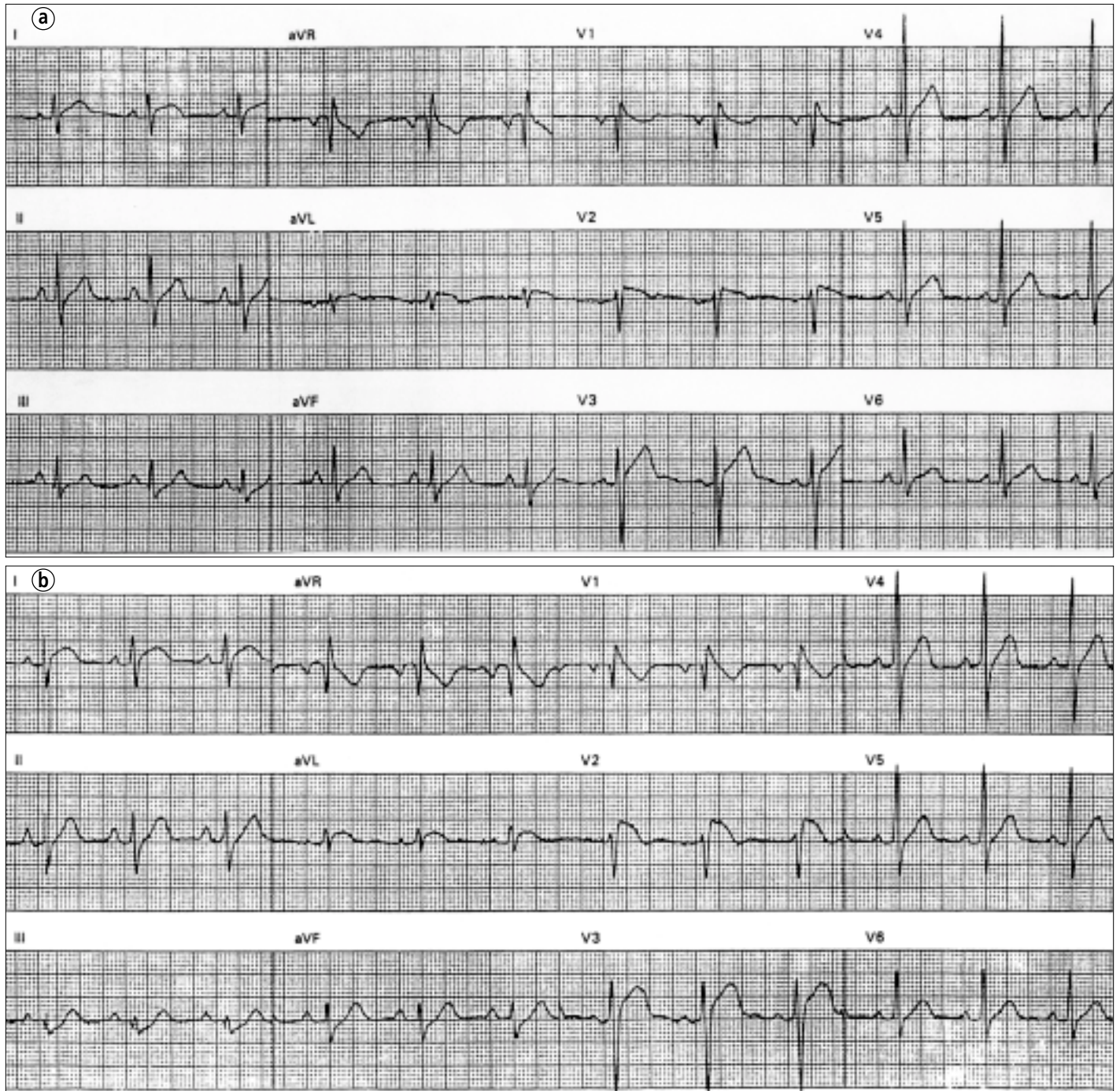
Angiography revealed normal coronary arteries and a left ventricular ejection fraction of 50% to 55% with a small area of inferoapical hypokinesis. During electrophysiologic study, ventricular tachycardia was not induced. Characteristic of Brugada syndrome, 1 g of procainamide infused intravenously over 30 minutes caused an increase in ST-segment elevation in electrocardiographic leads  $V_1$  to  $V_3$  (Figure 2).

The causes of syncope are myriad, and several possibilities may apply to a given patient. In this patient, the antidepressant

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Supported by an unrestricted grant from the Medical Center of Louisiana Foundation.

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**Figure 2.** Five days after admission, electrocardiograms were recorded (a) before and (b) after intravenous infusion of 1 g of procainamide over 30 minutes. In (a), ST-segment elevation is no longer seen in lead V<sub>1</sub> but has appeared in V<sub>3</sub>. In (b), ST-segment elevation has increased in leads V<sub>1</sub> through V<sub>3</sub> (especially in V<sub>2</sub>), a finding typical of Brugada syndrome. Although all prior electrocardiograms showed some ST-segment elevation in the anterior precordial leads, 3 hours after this tracing the ST segment was isoelectric in V<sub>1</sub>, V<sub>2</sub>, and V<sub>3</sub>.

drugs are potential reasons for syncope. The selective serotonin reuptake inhibitors have been associated with orthostatic hypotension or severe sinus node slowing, although rarely, and the tricyclic antidepressants have produced multiple adverse cardiovascular effects: orthostatic hypotension, conduction disturbances, and ventricular ectopy, including ventricular tachycardia and torsades de pointes (1, 2). QRS prolongation and rightward deviation of the terminal QRS forces have been described with overdoses of tricyclic antidepressants (1). Among the tricyclic antidepressants, however, trazodone is the one nearly free of

cardiovascular side effects except for orthostatic hypotension (2), and this could not be elicited in the emergency department. Furthermore, evidence of tricyclic antidepressant drugs could not be found in the urinary toxicology screening test. The slight rise in CK and CK-MB suggests the possibility of a ventricular tachyarrhythmia due to myocardial ischemic injury, but the absence of chest pain, the presence of normal coronary arteriograms, and the lack of evidence of cocaine use make ischemia-induced arrhythmia an unlikely cause of syncope in this patient.

The significant findings on the electrocardiogram are a right bundle branch block pattern and ST-segment elevation in leads V<sub>1</sub> and V<sub>2</sub>, electrocardiographic features identified by Brugada and Brugada as harbingers of sudden death due to ventricular tachyarrhythmias (3). The elevated ST segment may be seen in V<sub>1</sub> to V<sub>2</sub> or V<sub>1</sub> to V<sub>3</sub>, may be coved and end in an inverted T wave (as in *Figure 1*), or may be notched. The pattern is mimicked by a variety of conditions, including early repolarization, myocardial ischemia or infarction, hypothermia, electrolyte disturbance, and tricyclic antidepressant overdose (4). In patients with the Brugada syndrome, the electrocardiographic pattern may come and go, and sodium channel-blocking drugs will accentuate the pattern (*Figure 2*) (5).

The Brugada syndrome occurs worldwide; it is frequently familial, and in some patients the mutation has been identified. Most often this has involved SCN5A, a gene located on chromosome 3 and encoding the cardiac sodium channel (6, 7). In this patient with palpitations suggesting a tachyarrhythmia followed by trauma-producing syncope, an electrocardiographic pattern of the Brugada syndrome, and accentuation of the pattern by procainamide, the decision to implant an automatic cardioverter defibrillator transvenously was a relatively easy one. The broader, less clear-cut issues of the magnitude of risk faced by the patient with this electrocardiographic pattern who is asymptomatic and has no family history of sudden death, and consequently of that patient's proper management, have been debated vigorously (6–9).

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