

All in the Head?

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A 40-year-old woman is found unconscious at her home and brought to the hospital ED. No past medical history is available. On examination, she is comatose with normal vital signs and no lateralizing neurological findings. An ECG (Figure 1) is obtained.

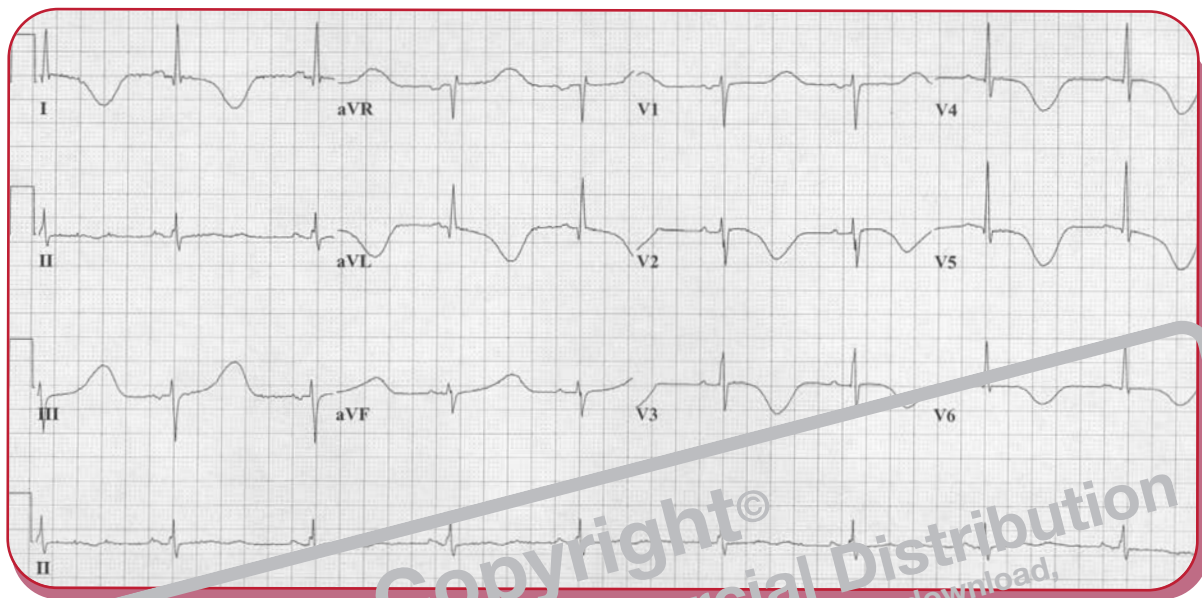


Figure 1. Initial ECG.

1. What are the abnormal ECG findings?

The ECG shows sinus bradycardia at approximately 50 bpm. There are striking repolarization abnormalities. There is impressive, deep, symmetric T wave inversions in leads I, aVL and V2 to V6. In addition, there is marked prolongation of the QT interval, which exceeds 700 milliseconds (ms) in some leads.

Even when appropriately corrected for the accompanying bradycardia, the corrected QT interval (QTc) is approximately 650 ms, far exceeding the 440 ms to 450 ms usually accepted as the upper limit of normal.

2. What is the most likely diagnosis?

The main diagnostic consideration is whether the ECG changes shown reflect an acute intracranial event or are due to a primary cardiac condition predisposing the patient to arrhythmia and loss of consciousness. Patients with congenital QT prolongation syndromes may experience life-

threatening ventricular tachyarrhythmias causing syncope or sudden death. Acute severe myocardial ischemia may also cause marked repolarization abnormalities on the ECG and serious arrhythmias. Many drugs cause QT prolongation, with an associated risk of *torsades de pointes*.

ECG

of the Month

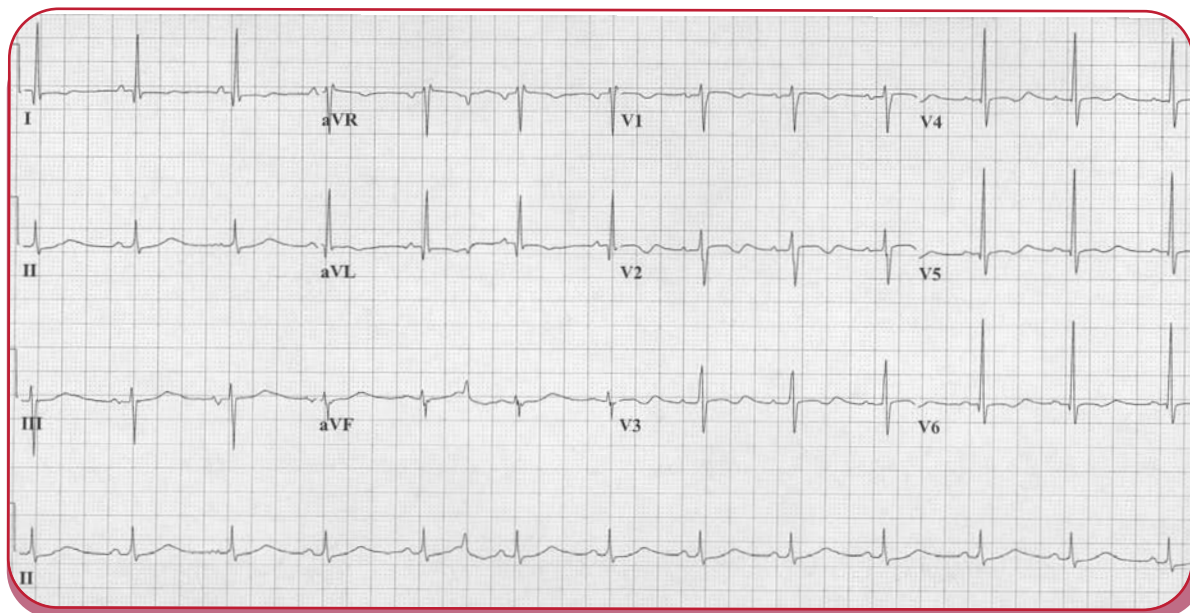



Figure 2. Repeat ECG.

A CT scan of the head in this patient demonstrated the presence of a large subarachnoid hemorrhage. Her condition gradually improved over the next several days and a repeat ECG (Figure 2) showed marked resolution of the initial repolarization changes.

ECG abnormalities are often seen following subarachnoid hemorrhage and stroke. QT prolongation and large inverted T waves are the most common abnormalities described, but ST segment changes and pathologic Q waves may also occur. Life-threatening arrhythmias, such as

polymorphous ventricular tachycardia, are seen infrequently. The mechanism of the ECG changes is not fully established, but is thought most likely to be due to subendocardial myocardial injury, resulting from a centrally-mediated release of catecholamines. In support of this viewpoint is the fact that many such patients show elevations in serum levels of markers of myocardial damage, such as troponins. Occasionally, regional wall motion abnormalities may be seen on echocardiography. Autonomic dysfunction is probably also a contributing factor. 

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Publication Mail Agreement No.: 40063348
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