The Clinical Picture

A homeless 63-year-old man with an abnormal electrocardiogram

**FIGURE 1.** The patient’s electrocardiogram on admission

**Q:** Police found a 63-year-old homeless man lying in the street. He was unresponsive. They brought him to the emergency department. **FIGURE 1** shows the initial 12-lead electrocardiogram (ECG) taken on admission. After intubation and appropriate care in the intensive care unit, another ECG was obtained 8 hours later (**FIGURE 2**).

What is the most likely diagnosis based on these ECGs?

- Subarachnoid hemorrhage
- Acute inferior wall myocardial infarction
- Hyperkalemia
- Hypothermia
- Digitalis toxicity

**A:** When **FIGURE 1** was recorded, the patient was still unresponsive and his temperature—measured electronically via the tympanic membrane—was 28.0°C (82.4°F). **FIGURE 2** was taken after he had regained consciousness and his temperature had warmed to 36.0°C (96.8°F). His last memory was of binge drinking outdoors.
ECG SIGNS OF HYPOTHERMIA

The most characteristic and recognizable ECG feature of hypothermia is the J wave, also called the Osborn wave, the J deflection, or the camel-hump sign. Eventually seen in about 80% of patients with hypothermia, the J wave first appears at temperatures below 33°C (91.4°F). It is a reasonably specific sign of hypothermia, but it has been reported in normothermic patients and is therefore not pathognomonic.

As shown in FIGURE 1, the J wave is the convex positive deflection at the junction of the QRS complex and the early part of the ST segment. (Perhaps a more descriptive term would be the “inverted J wave.”) Although typically most prominent in the inferior limb leads (II, III, aVF) and the lateral precordial leads (V5, V6), all leads are involved as hypothermia worsens and the deflection heightens.

Origin of the J wave. Though the cellular basis of the J wave is not yet completely understood, several experts propose the phenomenon to be due to an accentuation of the “spike-and-dome” morphology of cardiac action potentials of mid-myocardial and epicardial cells. Hypothermia markedly slows conduction velocity and delays depolarization from the endocardium to the epicardium. Coupled with a widening of epicardial cell action potentials, this slowing can unmask a J wave by moving it outside the QRS complex.

Other features. Other ECG features of hypothermia are less specific than the J wave:
• Absent P waves combine with increased atrial ectopic activity to produce atrial fibrillation with slow ventricular response; this occurs (as in FIGURE 1) in approximately 60% of patients with a body temperature below 29.0°C (84.2°F)
• PR interval prolongation leads to more severe degrees of atrioventricular block
• Prolongation of the QRS duration and QTc interval reflects the effect of hypothermia on the intraventricular conduction system and the ventricular recovery phase; this electrophyslogic “irritability” worsens progressively below a
temperature of 28.0˚C (82.4˚F), when ventricular fibrillation becomes a grave concern.

The diagnosis of hypothermia is often obvious before an ECG is obtained. Nevertheless, ECG findings typical of hypothermia may corroborate a high index of suspicion in certain high-risk patient groups, such as elderly, postoperative, hypothyroid, homeless, and alcoholic patients.

**IMPORTANCE OF MAINTAINING STABLE RHYTHM**

Hypothermia typically results in a lethal arrhythmia. Cardiac output progressively declines below 30˚C (86˚F) owing to depressed cardiac mechanics, including a reduced heart rate. However, if venous return and a stable rhythm compatible with perfusion (sinus bradycardia, atrial fibrillation) are maintained, cardiac output can be sufficient to counter the concordant decrease in oxygen demand. Therefore, maintaining a stable rhythm is essential in the management of hypothermia.

**THE WRONG ANSWERS**

As for the other answers in this quiz: Subarachnoid hemorrhage. The most characteristic ECG findings in subarachnoid and intracranial bleeding are deeply inverted T waves, prolongation of the QTc interval, and prominent U waves. Rhythm disturbances include sinus bradycardia, sinus tachycardia, wandering atrial pacemaker, AV junctional rhythm, and ventricular tachycardia. J waves with atrial fibrillation are not characteristic.

A *cute inferior wall myocardial infarction* is marked by ST-segment elevation with upward convexity in the inferior leads and reciprocal ST-segment depression in “noninfarct” leads. *Hyperkalemia.* A trial fibrillation and J waves are not features of hyperkalemia. *Digitalis toxicity.* Although atrial fibrillation with a slow ventricular response can suggest digitalis toxicity, the prolonged QTc interval and J wave in this patient suggest a different diagnosis.

**CONCLUSION OF THE CASE**

Our patient was intubated, passively warmed with external countercurrent heat exchangers, and given ample fluid replacement. After 8 hours, his temperature had risen to 36.0˚C and his ECG ([Figure 2](#)) showed conversion to sinus tachycardia, a normal QTc interval, a normal QRS duration, and no J wave. He was extubated on the following day, recovered fully, and was discharged on his 6th hospital day.

**SUGGESTED READING**


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