J wave and hypothermia

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Abstract

Hypothermia can result from exposure to a cold environment (e.g., accidental drowning) or it can be induced and used as a brain protection strategy (e.g., therapeutic hypothermia). One common ECG presentation with hypothermia is the J wave, which is related to the altered cellular activities during hypothermia. A case study is used in this article to illustrate the presentation of a J wave with a patient experiencing hypothermia.

Hypothermia is defined as a core temperature of equal to or less than 35.0 °C (Petersdorf, 1994). Hypothermia can result from an accident (accidental hypothermia) such as near drowning in cold water or it can be used as a brain protection strategy (therapeutic hypothermia) for patients with head injury or stroke (Harries, 2005; Krieger et al., 2001; Marion et al., 1997). As body temperature decreases during hypothermia, cellular activity is altered (Keamy & Hall, 1992) and the patient may present with various alterations. One common ECG change in hypothermia is the appearance of a J wave. In this article a patient with hypothermia and showing ECG changes is presented.

Case scenario

A 21-year-old man suffered a severe head injury after a single vehicle rollover. On admission, his body temperature was 32.5 °C. His blood gases were normal with a serum potassium level of 3.1mmol/L. The ECG (see Figure One) showed sinus bradycardia with a prolonged QT interval (520 msec; QTc 481 msec). J waves were noted in limb leads II, III, aVF and precordial leads V3 to V6.

J waves

A J wave (see Figure Two) is also known as an Osborn wave. It is a hump-like deflection at the J point (the point where the QRS complex ends and joins with the ST segment). A J wave is usually associated with hypothermia, but it can also be a normal variant (early repolarization syndrome) or exist in

![Figure One: Patient’s ECG on admission.](image-url)
some abnormal conditions such as Chagas disease, Brugada syndrome, electrolyte imbalances such as hypercalcemia, brain injury, subarachnoid hemorrhage, or central nervous system disorders especially in the hypothalamic region (Anguera & Valls, 2000; Heckmann, Lang, Neundörfer, Ropers, & Werner Moshage, 2001; Mieghem, Sabbe, & Knockare, 2004).

In hypothermia, ion channels in the epicardium stay open longer than normal and more potassium efflux occurs, which creates a voltage difference between the epicardium and endocardium. This transmural voltage difference results in a spike-and-dome action potential morphology (see Figure Three) (Anguera & Valls, 2000; Krantz, 2005; Mieghem et al., 2004), which manifests on the ECG as a J wave. The ventricular activation is from endocardium to epicardium. A thicker left ventricular wall makes the impulse transmission longer than the right. As a result, the J wave is more prominent on the inferior leads (II, III, aVF), and mid to left precordial leads (V3 to V6) (Yan & Antzelevitch, 1996). Since the right ventricular wall is thinner, the impulse transmission time from endocardium to epicardium is shorter, and the J wave may not be obvious.

The ECG changes related to hypothermia are reversible (Yan & Antzelevitch, 1996). Once the body temperature rewarms to the normal range, the amplitude of the J wave will be reduced or will disappear. The patient in this scenario was rewarmed gradually to 36°C. Following rewarming, his ECG was back to a normal sinus rhythm and the J wave disappeared.

References