Osborn waves due to severe hypothermia

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Case description

A 60-year-old woman, known for illicit drug use and treated for severe depression was found unconscious at her home with a body temperature 24.5 °C due to intoxication of opiates and multiple psychotropic drugs (flurazepam, citalopram, levomepromazinum, clonazepam). An electrocardiogram (ECG) was obtained at her admission showing large J waves (or Osborn waves) in the 12 leads, with sinus bradycardia at a rate at 48/min and a prolongation of the PR, QRS and QT intervals (fig. 1). She was intubated due to hypoventilation and active rewarming measures were initiated, which consisted of administration of warmed intravenous saline, heated humidified oxygen and use of a warming blanket.

Four hours after admission her body temperature had risen to 29° C and a repeat ECG showed a slight regression of the abnormalities (fig. 2).

The last ECG, done when the hypothermia was corrected, showed disappearance of the J waves, normalisation of the QRS and QT intervals and acceleration of the sinus rate to 111 beats/min (fig. 3). The patient made full neurological recovery.

Discussion

Hypothermia causes characteristic ECG changes. It decreases spontaneous depolarisation of cardiac pacemaker cells and prolongs myocardial action potential duration.

Classic electrocardiographic manifestations [1] of hypothermia include:

- shivering artefacts
- sinus bradycardia
- PR / QRS / QT prolongation
- J waves (Osborn waves)
- atrial arrhythmias (common when the temperature falls to below 32 °C)
- ventricular fibrillation and asystole (especially if the temperature is <28 °C)

The Osborne wave is the most striking ECG feature of hypothermia, first described by John J. Osborn in 1953 [2]. It is a "hump-like" deflection between the QRS complex and the early part of the ST segment [3] and represents distortion of the earliest phase of membrane repolarisation. It is present in approximately 80% of hypothermic patients when the temperature is below 30 °C. The amplitude of the Osborne wave is roughly proportional to the degree of hypothermia [4].

Recent findings suggest that hypothermia increases the epicardial potassium current relative to the current in the endocardium during ventricular repolarisation [5]. The transmural voltage gradient is reflected on the surface electrocardiogram as a prominent J wave or Osborn wave. The J wave is most prominent in leads facing the left ventricle and in the inferior limb leads [6] and disappears with rewarming but can persist for 12–24 hours after restoration of body temperature [4].

The J wave is not pathognomonic of hypothermia but also occurs in other conditions such as hypercalcaemia [7], certain CNS lesions (subarachnoidal haemorrhage or cerebral injuries) [8], in the Brugada Syndrome [9] and also in healthy persons with early repolarisation [10].

There is no conflict of interest.

References

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Figure 1
12-lead ECG obtained at core body temperature 24.5 °C, showing sinus bradycardia at 48/min, a prolonged QT interval at 560 ms and large J waves more remarkable in inferior and in V_3–V_5 leads.

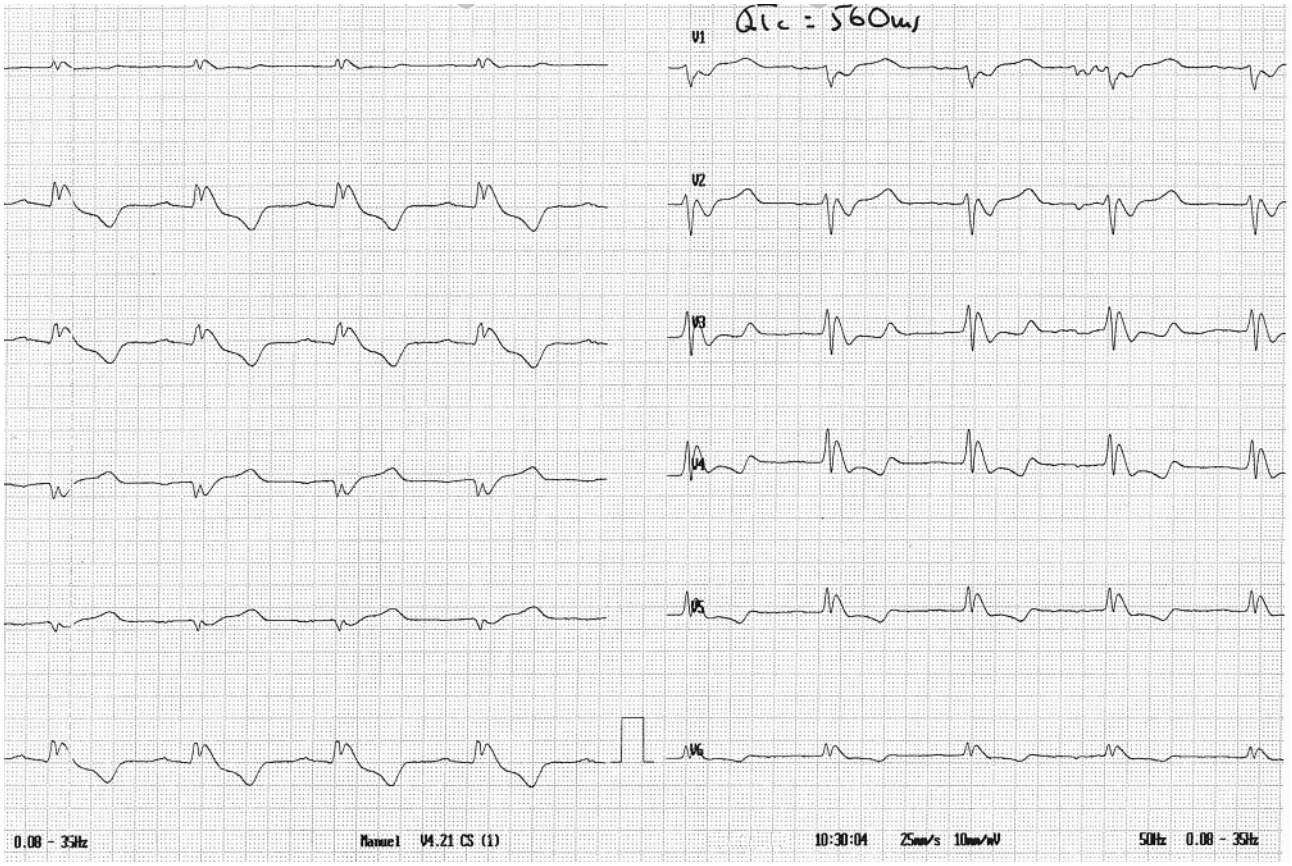

Figure 2
12-lead ECG obtained at core body temperature 29 °C, showing persistance of sinus bradycardia at 50/min, a prolonged QT interval at 480 ms and smaller amplitudes J waves.

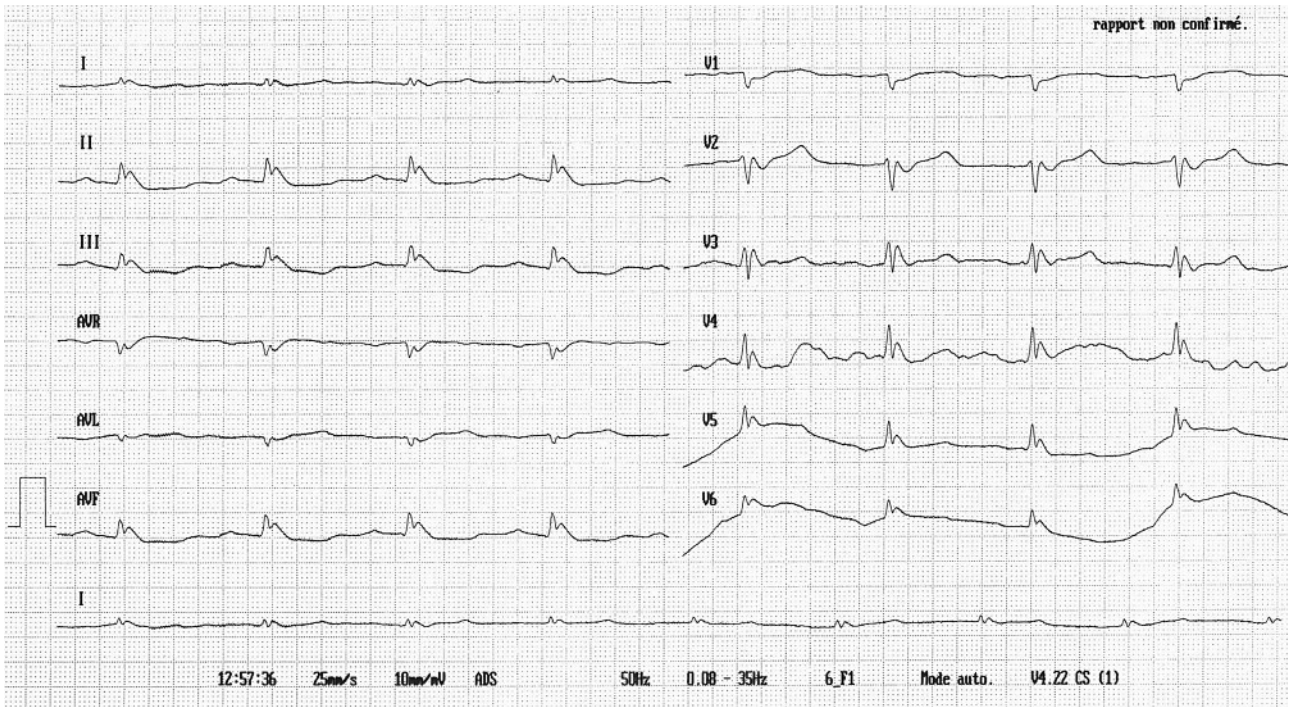

Figure 3
12 lead ECG obtained after rewarming (core body temperature 35.9 °C) with disappearance of the J waves and normalisation of QT interval.