Hypothermia-related Electrocardiographic Abnormalities: Osborn Waves

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A 42-YR-OLD man with marfanoid syndrome was scheduled for repair of thoracoabdominal aneurysm. Sevoflurane/oxygen was used as the primary anesthetic. At the completion of the operation (9 h and 42 min), the temperature had decreased to 33.9°C (blood), and sinus bradycardia (heart rate 56 beats/min) was noted (Lead II) and seemed to periodically decrease to 42 beats/min.1 (The preoperative electrocardiogram demonstrated normal sinus rhythm, heart rate of 84 beats/min, and left ventricular hypertrophy.) During this episode of bradycardia, blood pressure and cardiac output remained unchanged from previously recorded measurements. Arterial blood gas analysis did not reveal any acid–base or electrolyte abnormality that might explain the presence of bradycardia. After intravenous administration of 0.4 mg atropine, there was only a minimal increase in heart rate (60 beats/min). On admission to the cardiothoracic intensive care unit (33.0°C blood temperature), bradycardia was still present; Osborn waves (J-waves) and a prolonged QT interval were noted on the electrocardiogram (fig. 1A).2,3 The Osborn waves disappeared 8 h postoperatively (36.8°C blood temperature). The Osborn waves, prolongation of QT interval, and the sinus bradycardia have disappeared (heart rate 85 beats/min).

The electrocardiographic abnormalities associated with hypothermia are Osborn waves, prolonged PR and QT interval, sinus bradycardia, atrial and ventricular dysrhythmias, and shivering artifacts (muscular tremor).2,3 Hypothermia induces a difference between epicardial and endocardial potassium channel currents. The Osborn waves represent the electrocardiographic reflection of this transmural voltage gradient.2,3 The amplitude and the duration of the Osborn waves are inversely related to body temperature.2 The Osborn waves can persist 12–24 h after resumption of normothermia. The differential diagnosis of Osborn waves includes hypercalcemia, central nervous system lesions, coronary vasospasm, left ventricular hypertrophy, Brugada syndrome, and drug abuse, and it can appear as a normal variant (early repolarization).3 As shown in this case, the electrocardiogram provides early and important end-point core organ information regarding the electrophysiologic effect of a potentially life-threatening degree of hypothermia.

References