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Review

The EKG in hypothermia and hyperthermia

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Abstract Hypothermia and hyperthermia are relatively common clinical conditions that are associated with significant morbidity and mortality, especially if not promptly recognized and treated. Both of these conditions associated with extreme alterations in core body temperatures can be accompanied by alteration in cardiac function, often with manifest EKG changes. However, some of the EKG changes associated with hypothermia and heat stroke are non-specific and lead to diagnostic dilemmas. We here present 2 clinical cases, one each for hypothermia and hyperthermia to describe the EKG changes associated with these clinical conditions. We also present a review of available literature on these subjects.

Keywords: Hypothermia; Hyperthermia; ECG changes

Body temperature is controlled by the hypothalamus and by integration of efferent and afferent signals by the neurons in the preoptic anterior and posterior hypothalamus. The core body temperature is maintained in the range of 36.5–37.5 °C (97.7–99.5 °F) despite significant environmental variations. However, exposure to extremes of temperature can manifest as hypothermia or hyperthermia, both of which are associated with significant alterations in normal physiology which can be fatal. Alteration in cardiovascular function is often associated with changes in conduction properties of cardiac tissue, manifested as characteristic changes on surface EKG. We present here two cases associated with these extremes of temperature to illustrate the associated EKG changes and review the existing literature on the subject.

Hypothermia

For this relief much thanks; 'tis bitter cold And I am sick at heart.
[-Hamlet Act I, Scene I, 10.]

Hypothermia is defined at a drop in body's core temperature below 35 °C or 95 °F, with temperatures between 32.2 and 35 °C referred to as mild hypothermia, 28–32.1 °C as moderate and <28 °C as severe hypothermia. Hypothermia may be result from accidental prolonged exposure to cold, or may be from metabolic or therapeutic causes. EKG changes associated with hypothermia include the characteristic "Osborne waves" or J waves, initially described in 1953 [1]. Prolonged PR and QT intervals, increased QRS duration, repolarization abnormalities mimicking acute coronary syndrome (ST depressions, ST elevations) and atrial and ventricular arrhythmias have also been described in the literature as associated with hypothermia [2,3]. In the acute setting, these EKG changes may be interpreted as diagnostic of other conditions that may not co-exist: indeed suspicion of acute coronary syndrome/ST elevation myocardial infarction and Brugada syndrome have been raised by similar EKG findings [4,5]. We here describe a case that highlights this diagnostic dilemma.

Case report

An 85-year-old female with a reported medical history of mild cognitive impairment and osteoporosis, but no other significant medical history was found down by her van in her unheated garage by her neighbors in the middle of a cold, Midwest winter. Average outside temperature that day was 4 °F (−15 °C). When EMS arrived at the scene, she was found to be unresponsive with a Glasgow Coma Scale of 6, spontaneous respirations and exhibited a core (rectal) body temperature 26.7 °C (80.1 °F). She was immediately transported to a local emergency room. A CT scan of the head and cervical spine done in the ER did not show any acute intracranial process or spinal fracture. She was also hypotensive with a blood pressure of 77/53 mm Hg and a heart rate of 59/min and was started on IV fluids. Re-warming was initiated with forced-air warming blankets (Bair-Hugger). She was intubated for airway protection and was subsequently transferred to our institution for further care.

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On arrival, her vitals had improved with a blood pressure of 90/54 mm Hg, and a heart rate of 76/min. She was mechanically ventilated and unresponsive to external stimuli. A 12 lead EKG (Fig. 1) was obtained which demonstrated sinus or junctional rhythm (with poorly defined atrial activity), a right bundle branch block pattern with diffuse ST depressions in multiple leads and characteristic Osborne waves (arrows). The QTc was prolonged, calculated at 742 milliseconds.

An initial troponin T obtained was positive at 0.36 ng/mL (reference <0.10 ng/mL). Other labs obtained subsequently were notable for a normal renal function with blood urea nitrogen of 21 mg/dL and serum creatinine of 0.5 mg/dL. She was noted to have hypokalemia with a potassium level of 2.8 mEq/L. TSH was 1.66 μIU/mL (reference range 0.27–4.20 μIU/mL). Her EKG changes were attributed to severe hypothermia and hypokalemia. She was subsequently admitted to the intensive care unit.

Over the next few hours, her vitals stabilized with supportive care. Her mental status improved as well. She was gradually rewarmed and her core temperature improved to 37.1 °C (98.9 °F) over the next few hours. Serial EKGs obtained about 10 hours post her previous EKG showed sinus rhythm (now apparent), appearance of ST elevation in the anterior-septal leads (arrows), ST depression and T wave abnormalities in inferior leads with disappearance of the Osborne waves and right bundle branch block (Figs. 2 and 3).

Her troponin T increased to 0.70 ng/dL. It was not immediately apparent if some of her EKG changes could be attributed to her hypokalemia and hence she was taken to the cardiac catheterization laboratory for coronary angiography. Her coronary angiogram revealed essentially normal coronary arteries. An echocardiogram obtained subsequently showed normal bi-ventricular systolic function and no significant valvular abnormalities. She improved gradually with supportive care, with normalization of mental status and was successfully extubated. Her subsequent hospital course was uneventful and she was discharged home 4 days post admission with involvement of social services. An EKG obtained prior to her discharge showed improvement in her previously noted repolarization abnormalities (Fig. 4).

Discussion

The effects of hypothermia on the function of the heart and the conduction system (with focus on EKG manifestations) have been a subject of interest for over 100 years [6–8]. Multiple animal studies and human observations have revealed a multitude of EKG changes that accompany progressive hypothermia. While EKG changes such as SA (sino-atrial) exit block, PR prolongation, QRS widening, ST depression, atrial and ventricular fibrillation have been described, they are not specific for hypothermia and may be observed in a variety of different clinical scenarios. In contrast, Osborne waves defined as J-point elevation or a 1-mm in height elevation at the end of the QRS complex that occurs in two consecutive beats are a relatively specific finding in hypothermia, described in the literature as occurring in about 80% patients with a core temperature of <90 °F (32.2 °C). A prospective study of serial patients presenting with hypothermia to an emergency department found Osborne waves to be present in 100% of patients with a core body temperature of ≤87 °F (≤30.5 °C) [3]. The Osborn wave as it is called today was first described in 1938 in a patient with accidental hypothermia as a repolarization abnormality present at the end of the QRS [9]. Subsequent papers described this finding in patients with intentional and unintentional hypothermia in different settings [10,11]. Animal studies on experimental hypothermia done in the same time frame also described this characteristic deflection at the end of QRS. This was attributed to myocardial anoxia, though not definitively proved [12–16]. These EKG characteristic changes were subsequently described by J.J.

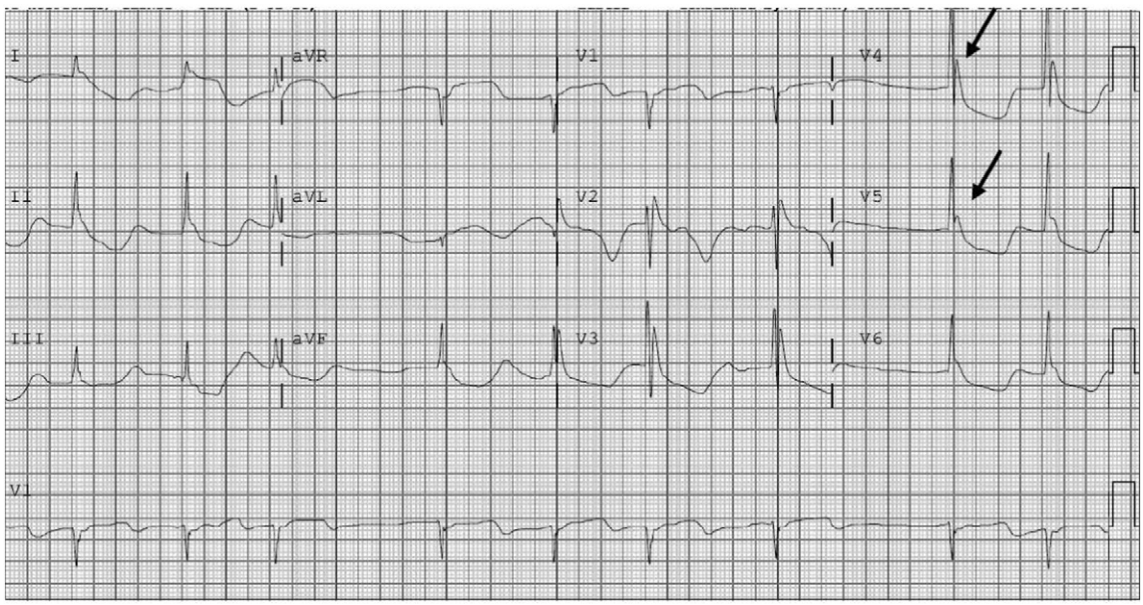

Fig. 1. Note the marked ST depressions, prolonged QT interval and Osborne waves (arrows). Atrial activity is not immediately apparent on this EKG.