Osborne waves in the hot summer
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ABSTRACT
Osborne waves are produced when the J-point deviates from baseline. While there are many known causes of Osborne waves, hypothermia remains the most common. Previous studies have been inconsistent about the risk of Osborne waves progressing to a deadly arrhythmia. Commonly, once patients are rewarmed, they no longer exhibit Osborne waves or experience cardiac arrhythmias. This patient presented with hypothermia on a hot, humid August day demonstrating two factors known to cause Osborne waves – hypothermia and hypocalcemia. While replenishing the calcium was beneficial, providing ventilator support and active rewarming remained the mainstays of treatment.

Key Words: Arrhythmias, hypothermia, J-waves, Osborn waves, summer

INTRODUCTION
Hypothermia most commonly occurs in the homeless population, individuals trapped in cold environments and those who have substance abuse, cardiovascular, or neurologic disorders. Up to 80% of patients with hypothermia demonstrate Osborne waves, also known as J-waves.[1] J-waves are produced when the J-point, where the QRS segment joins the ST, deviates from baseline.[2] They lead to repolarization abnormalities causing atrial and ventricular arrhythmias which can ultimately lead to death.[3-4] Given that Osborne waves are more common in hypothermic patients, it is rare that a patient with hypothermia would present to the hospital during the warmer months. We present an unusual case of Osborne waves in an unresponsive patient presenting with hypothermia during the summer. This illustrates the importance of obtaining a detailed patient history to understand an unusual clinical picture.

CASE REPORT
Our patient is a 30-year-old female with a history of polysubstance abuse, anxiety, and depression who was brought to our Emergency Department (ED) unresponsive. On the day of admission in August, the local temperature was 32.2°C (90°F) with 59% humidity. Emergency Medical Services (EMS) found her in a bathtub where she had been placed by friends to wake her up after she had injected intravenous (IV) heroin. Per EMS, the patient was unresponsive, cold, and had pinpoint pupils. They administered NARCAN, 4 mg intranasally, in the field with no improvement of her respiratory status or level of consciousness.

On arrival to the ED, the patient did not respond to verbal or painful stimuli and was intermittently moaning. A total of 4 mg of NARCAN were administrated intravenously with no significant improvement in mental status. She was intubated for airway protection due to decreased level of consciousness with 20 mg of etomidate and 100 mg of succinylcholine. Physical examination revealed initial axillary temperature of 25.8°C (78.4°F), pulse rate of 55, respiratory rate 16, and a blood pressure 94/40. Her wet clothes were removed, and warm blankets were applied. A temperature-sensing Foley was inserted with 35 ml of clear yellow urine returned. Topical warming with a Bair Hugger system (3M, Maplewood, MN, USA) and warm saline bags was placed in both axillae and on both groins. She was given a total of 2 L of warmed IV normal saline through large-bore peripheral IVs.

Initial laboratory work was significant for hyperkalemia at 5.6 mmol/L, creatinine 1.16 mg/dL, hyperglycemia at
385 mg/dL, hypocalcemia at 7.2 mmol/L, and leukocytosis at 16,000.

An electrocardiogram (EKG), obtained on arrival, demonstrated atrial fibrillation and Osborne waves [Figure 1].

Central venous access was obtained, and the patient was transferred to the intensive care unit for continued warming with warm IVs fluids and bladder irrigation. Ten units of regular insulin as well as calcium gluconate were administered for hyperkalemia and hypocalcemia, respectively. When she became normotensive and normothermic, a repeat EKG was obtained and revealed a normal rate, rhythm, and QRS complex. Her Osborne waves had resolved. She was extubated the following day, following commands, and answering questions appropriately. Addiction medicine was consulted to help facilitate transition back to outpatient care. She was discharged home on hospital day 3.

On her presentation – and critical to the clinical scenario – in discussion with her friends who found her, they reported hearing medical stories of patients being kept alive after overdoses with induced cooling. As such, when she was found unresponsive and before calling EMS, her friends placed her in the bathtub and filled the tub with as much ice as available. In addition, to reduce her core temperature, they reported inserting multiple ice cubes rectally.

**DISCUSSION**

Osborne waves appear on an EKG when the J-point deviates from baseline. Although seen in conditions such as sepsis, hypothyroidism, and diabetic ketoacidosis, hypothermia remains the most common cause of Osborne waves, with accidental hypothermia being the main cause closely followed by therapeutic hypothermia. Hypothermia therapy has been well documented for both neurological and cardiac protection and has been used in patients who have brain injuries or suffered cardiac arrest. In terms of neuroprotection, hypothermia reduces the cerebral metabolism of glucose and consumption of oxygen which decreases intracellular acidosis and free radical oxygen production eventually leading to a decrease in cerebral edema and minimizing risk of thrombosis. In cardioprotection, hypothermia protocol has been used to decrease infarct size and reduce myocardial injury. It has been proposed that in a hypothermic state metabolic demand is reduced, adenosine triphosphate is preserved, and there is improved blood flow through the myocardial microvasculature.

While the benefits of using hypothermia are numerous, physicians have to be cognizant of many complications. During induction of hypothermia, patients are at risk for bradycardia and subsequently reduced cardiac output. Cardiac patients have a tendency to become hypotensive when hypothermia is used which can exacerbate underlying causes of cardiac arrest. In addition, infection and bleeding need to be monitored since in a hypothermic state cellular and humoral immunity are impaired, and enzymes within the coagulation cascade are nonfunctioning. When a patient is cooled to <35°C, they start to show signs of insulin resistance, decreased gastrointestinal mobility, have reduced cytochrome P450 activity leading to decreased clearance of certain medications, and demonstrate electrolyte abnormalities.

One must also be careful not to rewarm patients too quickly. Ideally, patients should be rewarmed 0.2°C-0.5°C per hour to prevent hypoglycemia and electrolyte imbalances. Rewarming too quickly has been shown to increase the risk of neurocognitive complications and decrease or lose any clinical benefit for which the procedure was used.

Methods of rewarming are both passive and active. Passive rewarming includes the removal of wet and/or cold clothes. Active rewarming utilizes warm blankets or warming tools such as a Bair Hugger and warmed IV fluids which can be placed in peripheral lines or used as heating packs in the groin and axilla. IV fluids can also be used as irrigation through an orogastric tube or a Foley catheter.

The literature has been inconsistent as to if Osborne waves have the potential to predict cardiovascular death both in the immediate hospital stay and long term. This case is unique for many reasons. It provides a prime example of how a detailed history
can help understand an unusual clinical picture. It is rare that a patient presents with hypothermia on a hot, humid summer day, and it would have been easy to attribute her altered mental status solely to her drug use. In addition, the patient also exhibited multiple factors that could have contributed to the development of Osborne waves – hypothermia and hypocalcemia. Nevertheless, by actively rewarming as well as correcting her electrolyte abnormality, the patient’s EKG normalized and was free of arrhythmias and Osborne waves which is consistent with previous literature.

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**Conflicts of interest**
There are no conflicts of interest.

**REFERENCES**

1. Hudzik B, Gasior M. J-waves in hypothermia. CMAJ 2017;189:E1461.
2. Aslam AF, Aslam AK, Vasavada BC, Khan IA. Hypothermia: Evaluation, electrocardiographic manifestations, and management. Am J Med 2006;119:297-301.
3. Delhaye C, Mahmoudi M, Waksman R. Hypothermia therapy: Neurological and cardiac benefits. J Am Coll Cardiol 2012;59:197-210.
4. Mattu A, Brady WJ, Perron AD. Electrocardiographic manifestations of hypothermia. Am J Emerg Med 2002;20:314-26.
5. Omar HR. The osborn wave: What have we learned? Herz 2016;41:48-56.
6. Hadziselimovic E, Thomsen JH, Kjaergaard J, Kober L, Graff C, Pehrson S, et al. Osborn waves following out-of-hospital cardiac arrest-effect of level of temperature management and risk of arrhythmia and death. Resuscitation 2018;128:119-25.
7. Pargaonkar VS, Perez MV, Jindal A, Mathur MB, Myers J, Froelicher VF, et al. Long-term prognosis of early repolarization with J-wave and QRS slur patterns on the resting electrocardiogram: A cohort study. Ann Intern Med 2015;163:747-55.
8. Tikkanen JT, Anttonen O, Junttila MJ, Aro AL, Kerola T, Rissanen HA, et al. Long-term outcome associated with early repolarization on electrocardiography. N Engl J Med 2009;361:2529-37.