Electrocardiograms Recorded After Asystolic Cardiac Arrest

Solomon A. Seifu, MD, Pramilla N. Subramaniam, MD, and David Luke Glancy, MD*

A man with a history of drug abuse was found down at home and was asystolic. Following restoration of sinus rhythm, a hypothermia protocol brought his temperature to 32.5°C (90.5°F), and large Osborn waves appeared on his electrocardiogram. With rewarming the electrocardiographic signs of hypothermia diminished. Due to hypoxic brain injury during the arrest, the patient remained unresponsive and died on the fourth hospital day.
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Case Report

A 29-year-old man with a history of cocaine and heroin use was found down at home. Emergency medical services were called, responded promptly, and found the patient in asystolic cardiac arrest. Cardiopulmonary resuscitation included 2 ampules of 0.4 mg of naloxone given intravenously and 4 intravenous injections of 1.0 mg of epinephrine, which resulted in a return to sinus rhythm.

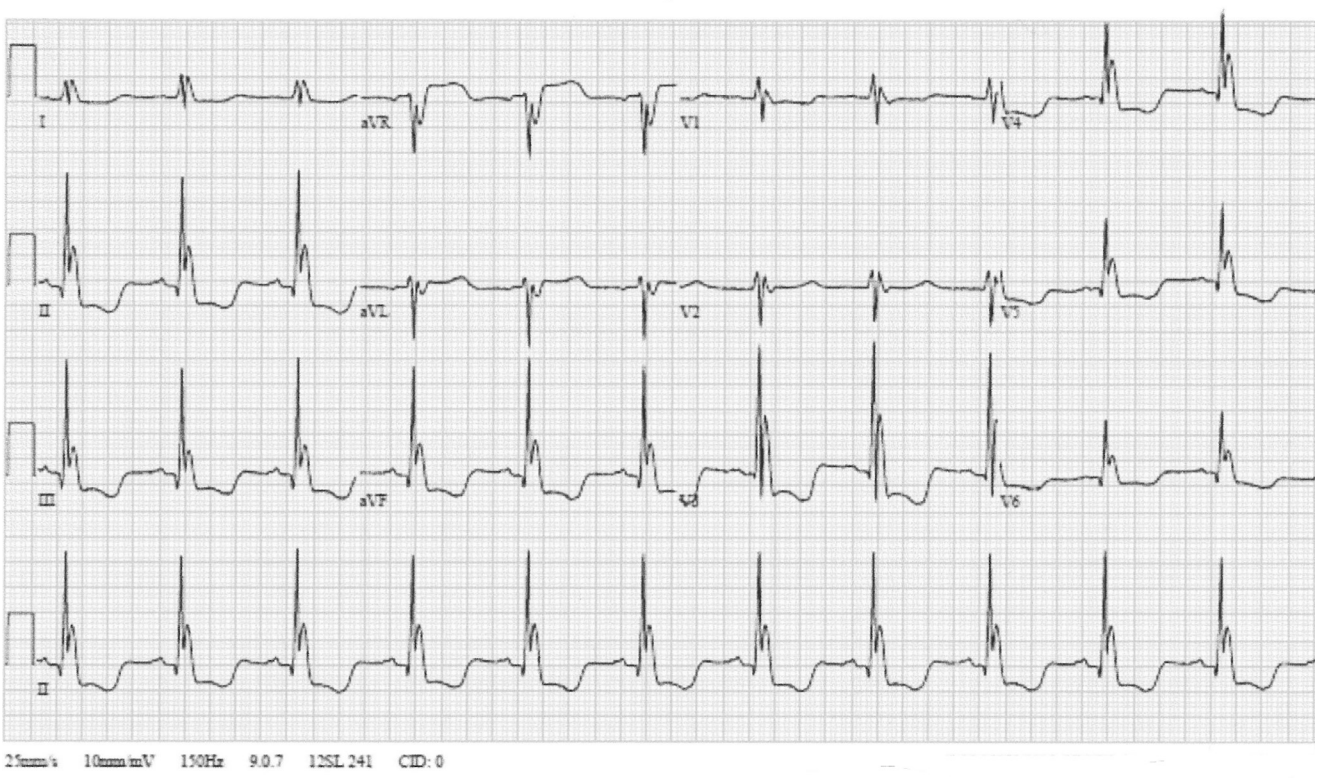

Figure 1. Electrocardiogram (ECG) recorded on the current admission when the patient's rectal temperature was 32.5˚C (91.5˚F) is markedly different from an entirely normal ECG recorded 5 years earlier. Most striking on the current ECG are the large J waves due to hypothermia (Osborn waves), the prolonged QRS duration of 170 ms, the prolonged QT interval of 546 ms (QTc = 576 ms), and the marked ST-segment depression and T-wave inversion. (The latter 2 changes are frequently seen after cardiac arrest from any cause).

In the emergency department serum laboratory values were remarkable for a troponin of 0.3 ng/ml, reference = 0.000 to 0.026 ng/ml; creatine phosphokinase of 1340 U/L, reference = 20 to 200 U/L; lactate of 12 mmol/L, reference = 0.5 to 2.2 mmol/L; urea nitrogen of 16 mg/dl, reference = 6 to 20 mg/dl; potassium of 3.9 mEq/L, reference = 3.5 to 5.1 mEq/L; a magnesium of 1.8 mg/dl,

Section of Cardiology, Department of Medicine, Louisiana State University Health Sciences Center, New Orleans, Louisiana. Manuscript received February 1, 2019; revised manuscript received and accepted February 4, 2019.

*Corresponding author: Tel: (985) 796-1550, fax: (504) 568-2127.
E-mail addresses: dglanc@lsuhsc.edu; jweber@lsuhsc.edu (D.L. Glancy).

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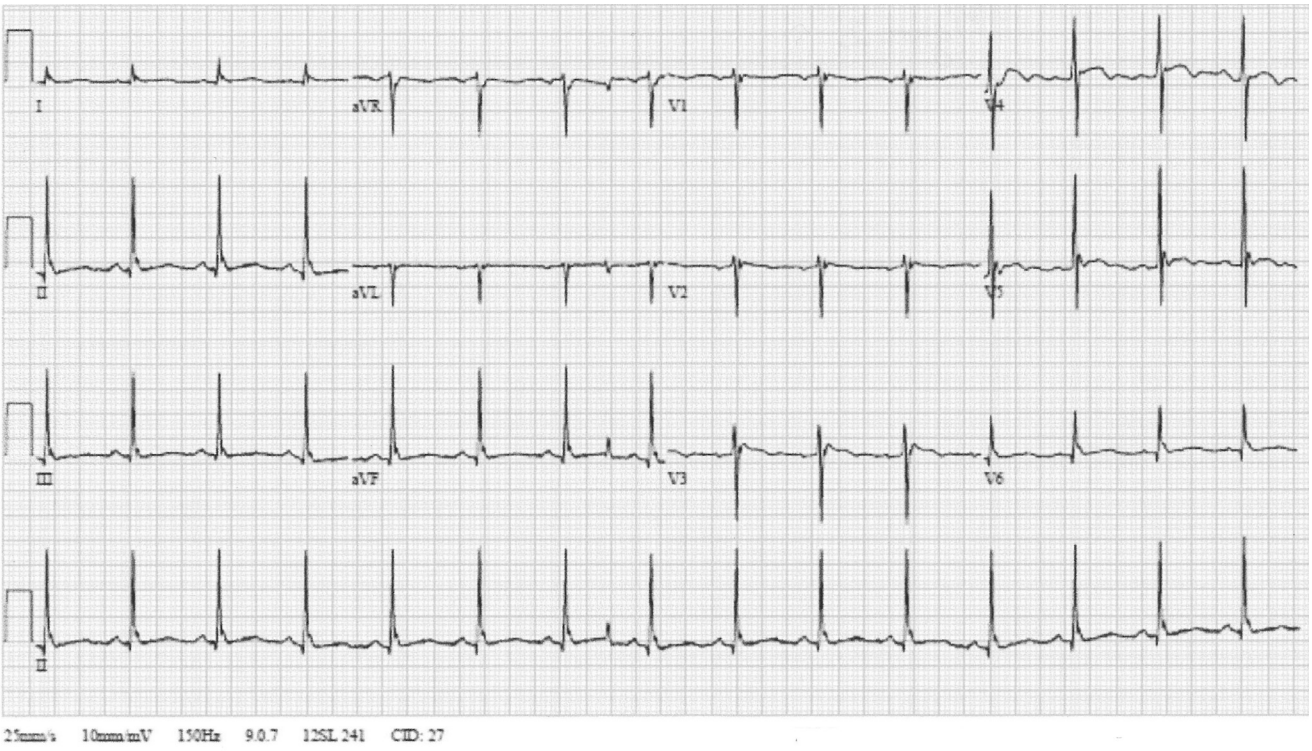

Figure 2. ECG recorded 9 hours after the ECG shown in Figure 1. The sinus rate has increased. The J waves are much smaller. The QRS duration and QT interval have decreased. The ST segment is no longer depressed, and the T-wave inversion is less marked.

reference = 1.6 to 2.6 mg/dl. An endotracheal tube was inserted, and the patient was started on isotonic intravenous fluid and a norepinephrine drip and was admitted to the intensive care unit.

A hypothermia protocol brought the patient's rectal temperature to 32.5˚C (90.5˚F) after 3 hours, and an electrocardiogram (ECG) at that time (Figure 1) was markedly different from an entirely normal ECG recorded 5 years earlier. As rewarming progressed the ECG changes of hypothermia resolved (Figure 2). A transthoracic echocardiogram showed a left ventricular ejection fraction of 50% with normal chamber sizes and valves. After full rewarming, the patient remained unresponsive due to the anoxic brain injury sustained during the cardiac arrest. He was pronounced dead on the fourth hospital day.

Apparently many observed the large J wave of hypothermia before Osborn.[1] However, his elegant study published in 1953 led to his name being attached to the wave.[2] Although J waves are seen in early repolarization, J waves of this size are virtually pathognomonic of hypothermia. Other ECG changes of hypothermia are sinus bradycardia, 1˚ A-V block, QRS prolongation, prolongation of the QT interval, atrial fibrillation, junctional rhythm, T-wave abnormalities, and baseline artifact due to shivering.[1,3] Although Osborn waves usually occur in patients with accidental hypothermia[1,3] and core temperature below 26˚C (79˚F), the current case demonstrates that Osborn waves can be seen in patients with intentionally induced hypothermia and at a core temperature of 32.5˚C (90.5˚F). Also the lower the temperature the more likely are arrhythmias to occur. Atrial fibrillation usually occurs at core temperatures between 32˚C (90˚F) and 22˚C (72˚F). Below 30˚C (86˚F) ventricular fibrillation may occur.

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