Letter to the Editor

An ECG Diagnosis Only for the Cold-Hearted

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A 78-year-old woman was transferred to our emergency department in a comatose state. The paramedics of the ambulance service reported that the patient's sister found her lying unconscious on the floor of her apartment, with several injuries all over her body. At initial evaluation, the patient had non-reactive dilated pupils and was pulseless with sinus bradycardia at about 30 beats per minute on the surface electrocardiogram (ECG) (Figure 1). An advanced cardiac life-support algorithm concerning pulseless electrical activity was promptly instituted, according to the European Resuscitation Council guidelines,[1] including endotracheal intubation and intravenous administration of two consecutive doses of 1 mg of adrenaline. After 10 minutes of continuous resuscitation maneuvers, return of spontaneous circulation was achieved and the new 12-lead ECG manifested an irregular wide complex rhythm with a notch in the descending limb of the QRS resembling atypical ST-segment elevation (Figure 2).

On admission to the intensive care unit a few minutes later, the patient remained acidemic (pH: 7.05) with prominent hyperkalemia (K^+ 8.1 mEq/L), although remarkably the ST-segment elevations began to recede (Figure 3). Muscle rigidity along with cold skin suggested severe hypothermia that was confirmed by a rectal temperature measurement of 27.2°C. Based on the above physical findings, a careful reevaluation of the initial and final ECG revealed the presence of Osborn waves (Figures 1 & 3). A bedside echocardiography evaluation revealed a severely depressed ejection fraction, estimated at 20%, with no evident valvular disease or pericardial fluid.

Despite intensive core rewarming using large volumes (200 mL/h) of intravenous normal saline 0.9% warmed to 40°C, as well as administration of heated humidified oxygen, the patient expired from multiorgan failure and intractable cardiogenic shock four hours after admission.

In the meantime, the police investigation discovered that the patient was assaulted and injured by a burglar in her home and had remained unassisted for approximately 48 hours in a cold environment.

Discussion

To our knowledge this is the first case report to demonstrate the transient ECG effects associated with adrenaline infusion during resuscitation in an acidemic patient with severe hypothermia before appropriate rewarming.

Detecting life in severely hypothermic subjects with a core temperature below 30°C can be problematic in cases

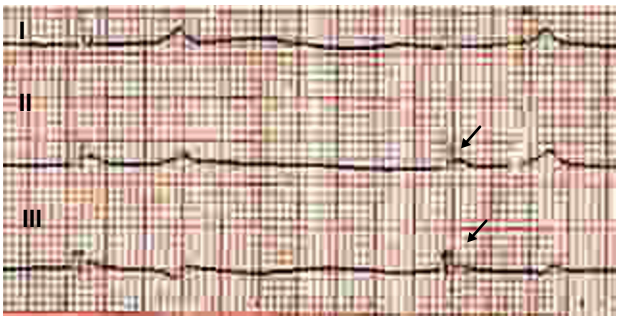

Figure 1. ECG showing sinus bradycardia with Osborn waves in leads II, III (black arrows).

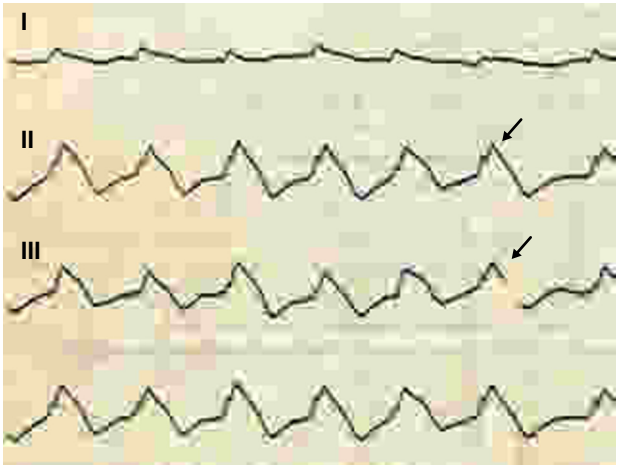

Figure 2. ECG showing atypical ST-segment elevations after adrenaline infusion in leads II, III (black arrows).

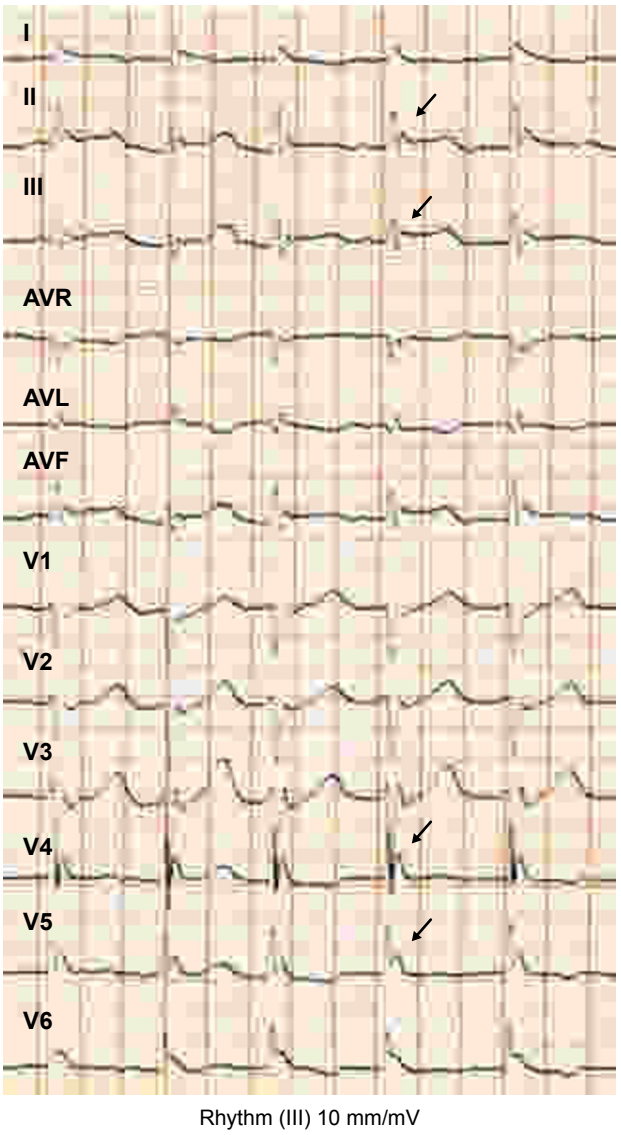

Figure 3. ECG showing sinus rhythm with Osborn waves after the disappearance of the adrenaline systemic effect in leads II, III, V_4, V_5 (black arrows).

where information about the patient's history is not available. These patients are usually unresponsive and motionless, with loss of deep tendon, skin and pupillary reflexes. In addition, blood pressure, pulse and respiration become practically undetectable.[2] The presence of electrical activity on the initial ECG in conjunction with the above mentioned characteristics may be falsely interpreted as pulseless electrical activity. This was the case in our patient, which prompted first contact medical personnel to initiate cardiopulmonary resuscitation despite severe hypothermia.

The ECG changes in our patient during the resuscitation procedure are rather interesting, and might mislead a physician engaged in such a case. ECG manifestations of hypothermia include prolonged PR and QT intervals, sinus bradycardia, atrial and ventricular dysrhythmias, and shivering artifacts (muscular tremor), which were all prominent in our case.[3] However a closer look could reveal several other observations, such as the fact that initially, although the patient was severely hyperkalemic and in an acidosis state, no manifestations of peaked T waves or widening of the QRS complex are evident, whereas prominent Osborn waves confirm the finding observed in previous case series that hypothermia can cause blunting of the expected ECG manifestations of hyperkalemia.[4] This interaction is inverted after adrenaline infusion, when a wide QRS complex tachycardia is evident. This tachycardia may be the result of the synergistic action of adrenaline and hyperkalemia,

which both prolong the QT interval, resulting in a sinusoidal ECG.

The unique aspect in our patient is the fact that, even when tachycardia was observed, the slow upright deflection between the end of QRS and the beginning of the ST segment was still apparent, indicating that the Osborn sign of hypothermia does not recede even in this condition. This is a valuable observation, since practitioners in the emergency department, considering the differential diagnosis for a patient with an incomplete medical history, could be misled into concluding that it is either ST-elevation myocardial infarction or an automatic ventricular tachycardia due to adrenaline infusion.

As the annual incidence of deaths resulting from hypothermia-related conditions is estimated at 4/1,000,000 in the United States including rural areas,[5] the above mentioned medical condition remains a challenging diagnosis. Emergency department physicians should remain "cold-blooded" and interpret the ECG findings in the context of the patient's history and clinical examination, so as to treat patients optimally, including the "cold-hearted" ones.

References

1. Deakin CD, Nolan JP, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support. Resuscitation. 2010; 81: 1305-1352.
2. Larach MG. Accidental hypothermia. Lancet. 1995; 345: 493-498.
3. Vassallo SU, Delaney KA, Hoffman RS, Slater W, Goldfrank LR. A prospective evaluation of the electrocardiographic manifestations of hypothermia. Acad Emerg Med. 1999; 6: 1121-1126.
4. Mattu A, Brady WJ, Perron AD. Electrocardiographic manifestations of hypothermia. Am J Emerg Med. 2002; 20: 314-326.
5. Baumgartner EA, Belson M, Rubin C, Patel M. Hypothermia and other cold-related morbidity emergency department visits: United States, 1995-2004. Wilderness Environ Med. 2008; 19: 233-237.