Hellenic Journal of Cardiology (2016) 57, 267–270

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: http://www.journals.elsevier.com/hellenic-journal-of-cardiology/

CASE REPORT

Electrocardiographic Manifestations in three Psychiatric patients with Hypothermia — Case Report

Eleftherios Pelechas [a,*], Nikolaos Tsigaridas [b], Sofia Kyrama [c], Stratos Trogganis [d], Christoforos Kardamis [e]

[a] Accident and Emergency Department, Scarborough General Hospital, United Kingdom
[b] Department of Cardiology, Chatzikosta Hospital, Ioannina, Greece
[c] Department of Cardiology, General Hospital of Arta, Greece
[d] Department of Cardiology, General Hospital of Kastoria, Greece
[e] Department of Cardiology, General Hospital of Corfu, Greece

Received 17 May 2014; accepted 26 June 2015
Available online 8 September 2016

KEYWORDS
Hypothermia;
Osborn waves;
Electrocardiographic changes;
Psychiatric patients

Abstract Hypothermia occurs when the core body temperature falls below 35°C, which, in severe cases, can lead to electrocardiographic changes. Several conditions that occur in the psychiatric population increase the risk of hypothermia. This risk can be further increased by the use of several classes of medications such as antipsychotics, beta-adrenergic antagonists and benzodiazepines. We report on three psychiatric patients who were admitted for hypothermia and developed electrocardiographic manifestations (sinus bradycardia, QT prolongation and Osborn waves), which completely resolved after treatment.

1. Introduction

Hypothermia is associated with a spectrum of electrocardiographic changes.[1] In addition, the degree of hypothermia leads to different electrocardiographic manifestations.[2] In mild hypothermia (35°C – 32°C), the electrocardiogram (ECG) is usually normal, but it can show J waves (Osborn waves) in rare situations.[3] Osborn waves in inferior and lateral leads, in combination with the appearance of other electrocardiographic manifestations such as prolonged PR and QT intervals, increased QRS complex duration, decreased amplitude of P and T waves and frequent supraventricular arrhythmias, are noted in moderate hypothermia (32°C – 28°C).[4–7] In severe hypothermia (<28°C), additional ECG changes such as J waves in all leads, absence of P waves

* Corresponding author. Eleftherios Pelechas, Cherry Tree Avenue 21, Scarborough, North Yorkshire, YO12 5DX, United Kingdom. Tel.: +44 00306979868855, +44 04407455949968.
E-mail address: pelehas@yahoo.com (E. Pelechas).
Peer review under responsibility of Hellenic Cardiological Society.

http://dx.doi.org/10.1016/j.hjc.2015.06.003

Table 1 Medical and drug history of the three patients with hypothermia.

	Age (in years)	Drug history	Core body temperature
Case 1	50	Diazepam, haloperidol, zolpidem	27,2°C
Case 2	61	Diazepam, haloperidol, zolpidem, clozapine, biperiden	27,6°C
Case 3	64	Levodopa, memantine, amlodipine/valsartan, lorazepam	28,8°C

and frequent ventricular arrhythmias may been seen.[8–9] However, the Osborn wave is considered the most specific ECG change seen in hypothermia patients.[10–12]

2. Case presentation

Three psychiatric inmates who presented in one month (December) were transferred to our emergency department by ambulance for low responsiveness (two patients) and coma (third patient). The patients' medical and drug histories are presented in Table 1. Their electrocardiograms all featured sinus bradycardia (38 bpm — 43 bpm), QT prolongation (.52 sec — .72 sec) and Osborn waves (see Figures 1, 2 and 3). A "shivering artifact" was also seen in the electrocardiograms of the first and the third patients.

All three patients were undergoing treatment with benzodiazepines (diazepam and lorazepam). One patient was also suffering from enuresis. The other two patients had poorly controlled Parkinson's disease. Blood counts, urea and electrolytes and chest x-rays did not show any evidence of infection. Because we had three patients in such a short period of time, we discovered that the heating system was not working properly. This heating system issue, combined with the enuresis in the first patient or the shakings in the Parkinson disease patients, may have contributed to the patients' hypothermia.

The patient in the first case was taking oral diazepam and haloperidol (which both cause sedation and drowsiness when combined with zolpidem), increasing the risk of central nervous system depression.

The second patient was using the same medications as the first patient, as well as clozapine. The combination of diazepam, haloperidol and clozapine may produce sedation and drowsiness while also increasing the risk of cardiac or respiratory failure.

For the third patient, the only possible drug interaction responsible for the ECG changes was amlodipine, valsartan and levodopa. This drug combination can produce added drug effects and should be monitored closely.

After gradually rewarming the patients using special rewarming blankets, the electrocardiographic manifestations resolved. All three patients were discharged a few days later.

3. Discussion

The J wave was initially reported by Kraus in 1920. It was reported again by Kraus in 1922 in a patient with hypercalcemia as well as by Tomazewski (1938) in a patient with hypothermia.[13–15] John Osborn reported experimentally induced J waves in hypothermic dogs in 1953. He described the J wave as a "current of injury" and postulated its occurrence was secondary to hypothermia-induced

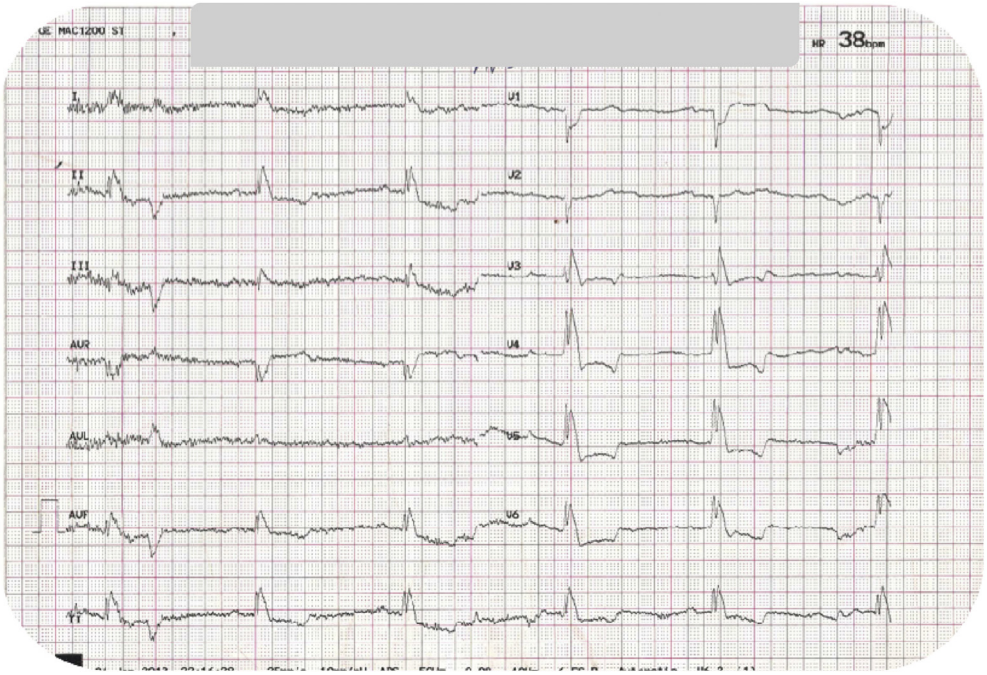

Figure 1 Electrocardiogram in severe hypothermia (27,2°C): sinus bradycardia (38 bpm), QT prolongation, J waves, and shivering artifact are seen.

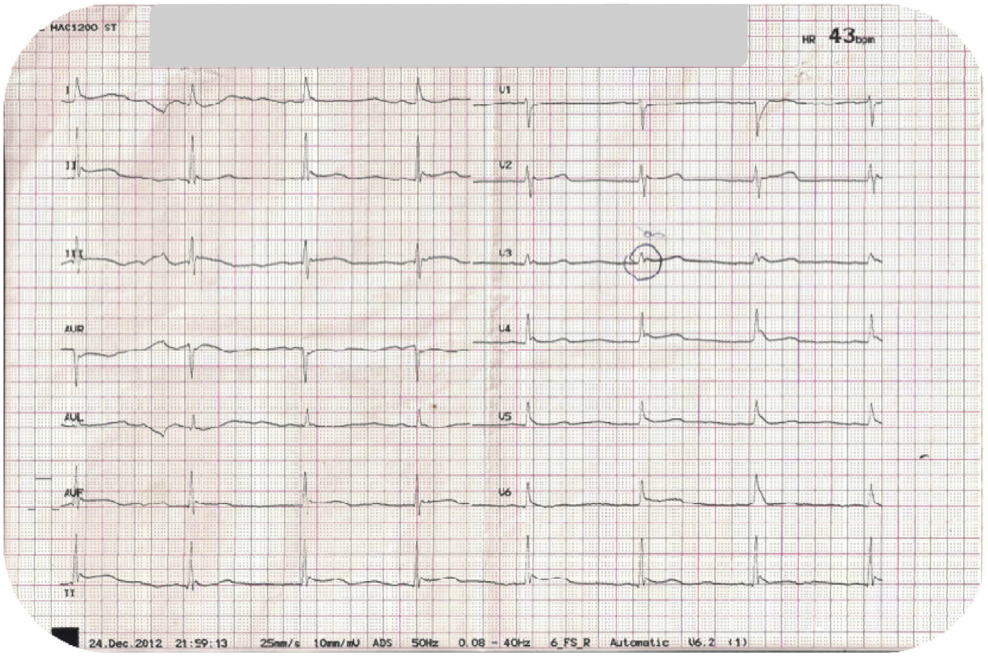

Figure 2 Electrocardiogram in severe hypothermia (27,6°C): sinus bradycardia (43 bpm), QT prolongation, J waves and myocardial infarction-like ST elevation in leads $V_4 - V_6$ are seen.

acidosis.[16] The Osborn wave (also known as camel-hump sign, late delta wave, hathook junction, hypothermic wave or prominent J wave), is a positive deflection occurring at the junction between the QRS complex and the ST segment, where the S point (which is also known as the J point) has a myocardial infarction-like elevation.[17–18] The mean vector axis of the J wave is oriented anteriorly, inferiorly and leftward across the left ventricle and septum.[6,19] J waves are present in 80% of patients with temperatures less than 35°C.[2] There is no consensus on the prognostic significance of J waves. The physiological basis of J wave has been described by Antzelevitch and colleagues.[19] The presence of a 4-aminopyridine-sensitive transient outward potassium current is responsible for the characteristic spike and dome pattern of the action potential in the ventricular epicardial and endocardial cells.[20]

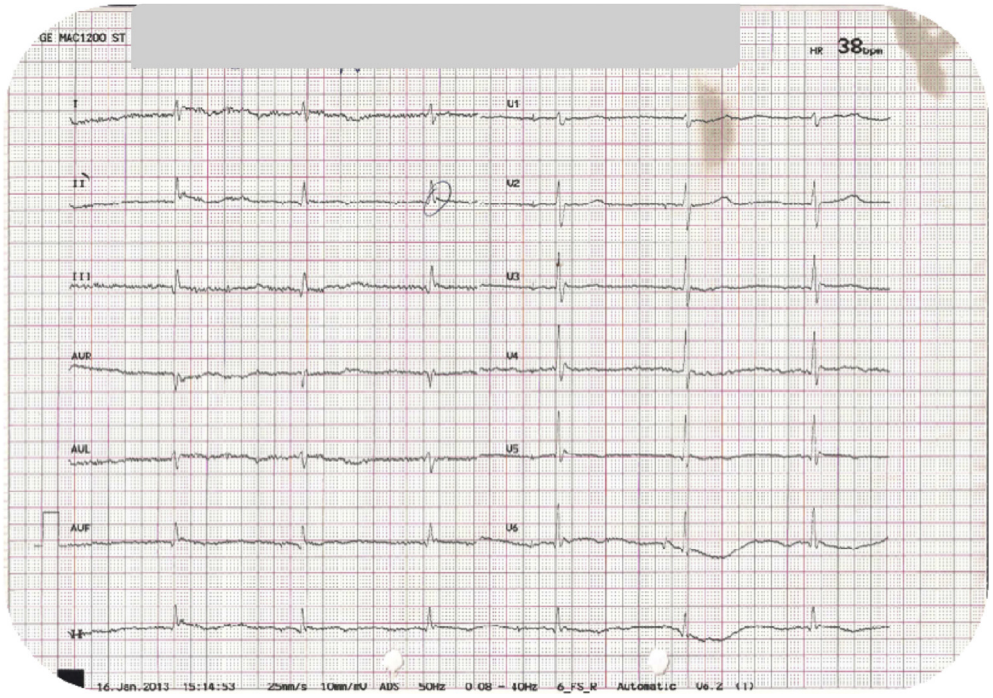

Figure 3 Electrocardiogram in moderate to severe hypothermia (28,8°C): sinus bradycardia (38bpm), QT prolongation, J waves in inferior leads (II, III, avf), shivering artefact.

Table 2 Known causes of Osborn waves in normothermic patients.

Acute ischemic events
Cocaine use
Haloperidol overdose
Left ventricular hypertrophy
Hypercalcemia
Brugada's syndrome
Central nervous system injury
After resuscitation of cardiac arrest

This current is more prominent in the ventricular epicardium compared to the endocardium, creating a voltage gradient between the epicardial and endocardial cells.[21] This voltage gradient across ventricular myocardium is accentuated by hypothermia and results in prominent Osborn waves.

Osborn waves are not pathognomonic of hypothermia; normothermic patients can also present with these waves. Some known causes of Osborn waves in normothermic individuals are presented in Table 2.[22–26] Although the arrhythmogenic implications of Osborn waves are not fully understood, the presence of this characteristic deflection may reflect an underlying critical condition. Different medical backgrounds of different patients could lead to ventricular arrhythmias, but each situation should be considered on a case-by-case basis. Understanding the true significance of the Osborn wave requires further study in order for it to be considered a potentially defining characteristic for patients who present with it.

Several conditions that occur in the psychiatric population increase the risk of hypothermia. Mental retardation, debilitating physical illness, nocturnal enuresis and seizure disorders are some examples of conditions that may predispose patients to hypothermia. This risk can be further increased by the use of several classes of medications used to treat psychiatric disorders, including benzodiazepines, antipsychotics, and beta adrenergic antagonists.[27–28] As a result, poor supervision of the room temperature for these patients may lead to hypothermia.

Financial support

The authors have no financial interests or financial arrangements to disclose.

References

1. Gavaliatsis IP. Electrocardiographic issues related to action potential phases 1 and 2 on the occasion of a case of accidental mild hypothermia. *Int J Cardiol*. 2001;77:81–86.
2. Cheng D. The EKG of hypothermia. *J Emerg Med*. 2002;22:87–91.
3. Aslam AF, Aslam AK, Vasavada BC, Khan IA. Hypothermia: Evaluation, Electrocardiographic Manifestations, and Management. *Am J Med*. 2006;119:297–301.
4. Anand K, Radhakrishnan S, Radhakrishnan A. The First Case Report of Accidental Severe Hypothermia from Tropical South India. *World J Med Sci*. 2014;10:446–451.
5. Schmidt-Schweda S, Ohler A, Post H, Pieske B. Moderate hypothermia for severe cardiogenic shock. *Resuscitation*. 2013;84:319–325.
6. Mustafa S, Naushad SN, Gowda RM, Khan IA. Electrocardiographic Features of Hypothermia. *Cardiology*. 2005;103:118–119.
7. Mattu A, Brady WJ, Perron AD. Electrocardiographic manifestations of hypothermia. *Am J Emerg Med*. 2002;20:314–326.
8. Ansari E, Cook JR. Profound hypothermia mimicking a Brugada type ECG. *J Electrocardiol*. 2003;36:257–260.
9. Graham CA, McNaughton GW, Wyatt JP. The electrocardiogram in hypothermia. *Wilderness Environ Med*. 2001;12:232–235.
10. Junttila MJ, Sager SJ, Tikkanen JT, Anttonen O, Huikuri HV, Myerburg RJ. Clinical significance of variants of J-Points and J-waves: early repolarization patterns and risk. *Eur Heart J*. 2012;33:2639–2643.
11. Antzelevitch C, Yan G. J wave syndromes. *Heart Rhythm*. 2010;7:549–558.
12. Edelman ER, Joynt K. J waves of Osborn revisited. *J Am Coll Cardiol*. 2010;55:2287.
13. Kraus F. Ueber die Wirkung des Kalziums auf den Kreislauf. *Dtsch Med Wochensch*. 1920;46:201–203.
14. Kraus F, Zondek SG. Uber die Durchtrankungsspannung. *Klin Wochensch I Jahrgang*. 1922;36:1778–1779.
15. Tomaszewski W. Changements electrocardiographiques observes chez un homme mort de froid. *Arch Mal Coer*. 1938;31:525.
16. Osborn JJ. Experimental hypothermia: Respiratory and blood pH changes in relation to cardiac function. *Am J Physiol*. 1953;175:389–398.
17. Maruyama M, Kobayashi Y, Kodani, et al. Osborn waves: History and Significance. *Indian Pacing Electrophysiol J*. 2004;4:33–39.
18. Gussak I, Bjerregaard P, Egan TM. ECG phenomenon called the J wave: history, pathophysiology, and clinical significance. *J Electrocardiol*. 1995;28:49–58.
19. Yan GX, Antzelevitch C. Cellular basis for electrographic J wave. *Circulation*. 1996;93:372–379.
20. Antzelevitch C, Sicouri S, Litovsky SH, et al. Heterogeneity within the ventricular wall: Electrophysiology and pharmacology of epicardial, endocardial and M cells. *Circ Res*. 1991;69:1427–1449.
21. Litovsky SH, Antzelevitch C. Transient outward current prominent in canine ventricular epicardium and not endocardium. *Circ Res*. 1988;62:116–126.
22. Patel A, Getsos J, Moussa G. The Osborn wave of hypothermia in normothermic patients. *Clin Cardiol*. 1994;17:273–276.
23. Otero J, Lenihan DJ. The "Normothermic" Osborn Wave Induced by Severe hypercalcemia. *Tex Heart Inst J*. 2000;27:316–317.
24. Alings M, Wilde A. Brugada's syndrome: clinical data and suggested pathophysiological mechanism. *Circulation*. 1999;99:666–673.
25. De Sweit J. Changes simulating hypothermia in the electrocardiogram in subarachnoid hemorrhage. *J Electrocardiol*. 1972;5:193–195.
26. Jain U, Wallis DE, Shah K, Blakeman BM, Moran JF. Electrocardiographic J waves after resuscitation from cardiac arrest. *Chest*. 1990;98:1294–1296.
27. Marum RJ, Wegewijs MA, Loonen AJ, Beers E. Hypothermia following antipsychotic drug use. *Eur J Clin Pharmacol*. 2007;63:627–631.
28. Hagg S, Mjorndal T, Lindqvist L. Repeated episodes of hypothermia in a subject treated with haloperidol, levomepromazine, olanzapine and thioridazine. *J Clin Psychopharmacol*. 2001;21:113–115.