Review Article

An Umbrella Review Article on a Correlation of Osborn Waves with Hypothermia Induced Left Ventricular Systolic Dysfunction

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ABSTRACT

The purpose of this review article is an overall finding from published studies on hypothermic causes of the Osborn waves induced lv systolic dysfunction and related topics are discussed. A systematic literature search was performed in the PubMed and Embase databases and collected 100 articles, out of which 28 were removed after peer review. We found 100 articles, out of which a total of 72 cases were considered. When analyzing only cases with more than one reported ECG, there was a strong inverse correlation between J wave, body temperature and left ventricular dysfunction. Electrocardiographic manifestations of hypothermia may assist in timely diagnosis and management of hypothermic patients. Even though prominent J-waves are the hallmark of hypothermic patient's ECG, they are not pathognomonic, as they have been associated with other inherited or acquired conditions, many of which are highly arrhythmogenic.

Keywords: Hypothermia, left ventricular systolic dysfunction, Osborn waves, Heart rate, prolonged ejection time, Left ventricular ejection fraction.

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INTRODUCTION

Hypothermia is a core body temperature below 35 ° C (95 ° F) that causes cardiac tissue generalized slowing of impulse condition and can be seen in ECG and electrocardiogram as prolongation of ejection time, widening of QT intervals, RR, PR and QRS [1,2,3]. As part of the stress response, sinus tachycardia occurs in the early phases of hypothermia. Sinus bradycardia combined with gradual prolongation of the PR interval, QRS complex, and QT interval, survives when the temperature drops below 90 ° F [4,5,6]. ECG abnormalities associated with J Waves can cause hypothermia and can lead to Left ventricular dysfunction. (Figure 1).

Osborn wave

The electrocardiogram (ECG) J point (Figure 2) is the point where the ST-segment is joined by the QRS complex and marks the end of depolarization and the beginning of repolarization. Several causes can lead to the J point being deviated from the baseline.

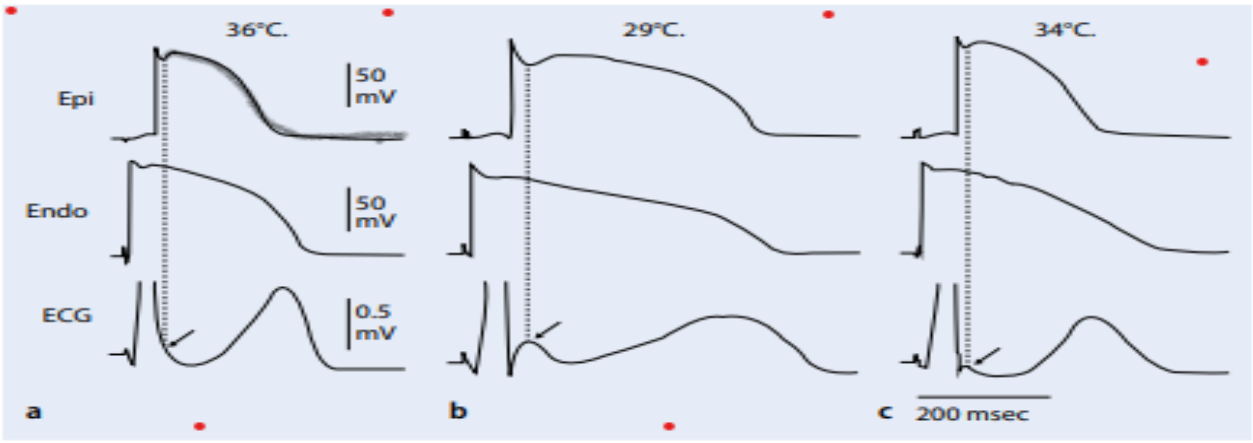

Figure 1: ECG Abnormalities associated with (J WAVES): Effect of hypothermia on action potential and ECG morphology

These include early repolarization, acute pericarditis, myocardial infarction, branch block of the right or left bundle, hypertrophy of the right or left ventricle, or digital effect [7,8]. When the J point is significantly deviated from the baseline, the J wave is formed. After John J. Osborn work on hypothermic dogs in 1953, this wave acquired a

number of names, including the camel hump symbol, late delta wave, J point wave, hat-hook junction, K wave, H wave, J wave, and the Osborn wave. The Osborn wave is not considered a marker of hypothermia only, as it is often encountered in a number of other nonhypothermic pathologies [9,10]. Although the Osborn wave is usually an unusual warning, it has important consequences for its detection in the 12-lead ECG and during telemetry monitoring. The clinical benefit of understanding the Osborn wave well exceeds its potential to indicate low core body temperature or overcooling in patients experiencing therapeutic hypothermia in hypothermic subjects but extends in some clinical settings to be a predictor of mortality [11,12]. For example, the occurrence of the Osborn wave was found to be a predictor of ventricular fibrillation (VF) in patients with acute coronary syndrome (ACS). This was shown by Aïssou and colleagues, who found that there was a higher frequency of ventricular fibrillation (VF) and cardiac arrest and in-hospital mortality in patients with acute coronary syndrome (ACS) and apparent Osborn waves in their ECG. The Osborn wave's major diagnostic and prognostic effects warrant its acknowledgment by numerous health care professionals expected to experience it and not just cardiologists [13,14]. A comprehensive analysis of the Osborn wave is provided here: its history, clinical relevance, features of the ECG, electrophysiological basis, and prognostic implications [15].

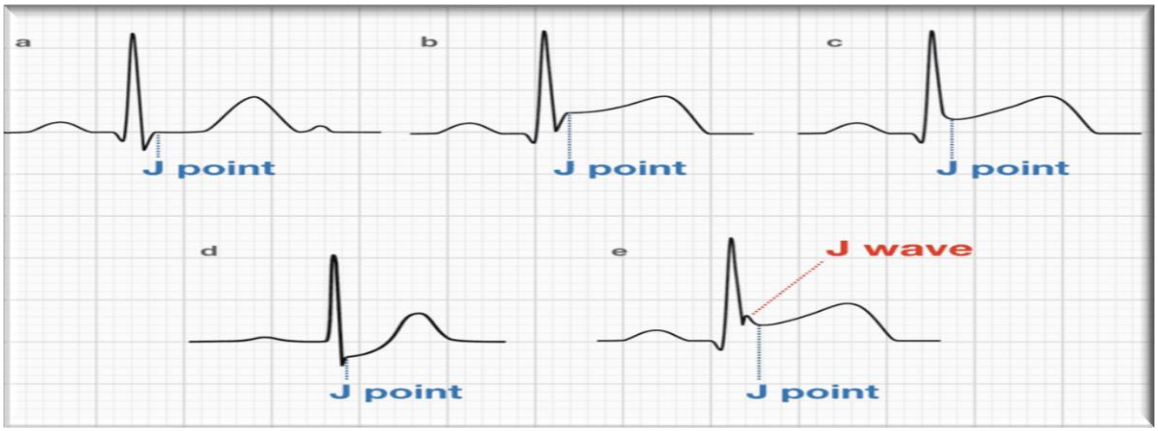

Figure 2: J Point in Electocardiograph

Mechanism via Electrocardiograph and Electrophysiological basis

Evidence based Electrophysiological basis of the Osborn wave

A dramatic shift in understanding the electrophysiologic mechanism responsible for the Osborn wave was demonstrated in 1988 by Litovsk and Antzelevitch. Epicardial action potential, unlike that of endocardium, displays a "spike and dome" morphology that becomes progressively more accentuated at slower stimulation rates (thus explaining the Osborn wave prominence with slower heart rates) [16,17]. To further confirm this theory, the spike and dome morphology and the rate-dependent changes observed in the epicardial action potential were tested after addition of 4-aminopyridine (4-AP, a transient outward current blocking agent)[18]. In epicardium, the spike and dome morphology were greatly attenuated with 1 mM of 4-AP and was totally abolished with 5 mM 4-AP. These observations provided support for the hypothesis that the transient outward current that is prominent in the epicardium more than in the endocardium causes the spike and dome configuration of the epicardial action potential creating a transmural voltage gradient manifesting itself as the Osborn wave in the ECG. In 1996, the excellent work performed by Yan and Antzelevitch provided the first direct evidence supporting the hypothesis that heterogeneous distribution of a transient outward current mediated spike and-dome morphology of the action potential across the ventricular wall is the underlying electrophysiological mechanism explaining the Osborn wave[19,20].This was concluded after an Osborn wave was observed at the R-ST junction of the ECG in 17 of 20 adult dogs after isolation and arterial perfusion of the right or left ventricular wedge and recording transmembrane action potentials from the epicardial, M region, and endocardial transmural sites with three floating microelectrodes[21,22]. They observed that the notched configuration of the action potential is more prominent in the epicardium than the endocardium and the end of phase 1 of the epicardial action potential coincides with the peak of the J-point elevation. A decrease in the temperature to 29 °C caused a more prominent Osborn wave and rewarming caused a reduction in the amplitude of the Osborn wave and that of the notch of the epicardial action potential [23,24]. This observation was shown clinically in many case reports and studies that demonstrated the increase in the amplitude of the Osborn wave with progression of hypothermia and the gradual resolution of the Osborn wave with rewarming. Electrocardiographic characteristics of the Osborn wave Hypothermia remains the main cause for encountering an Osborn wave in the ECG. The Osborn wave is reversible with correction of the precipitating factor for its development[25,26,27]. It manifests as a positive deflection with a dome-shaped configuration evident at the R-ST junction of the ECG. Yan and Antzelevitch showed experimentally that the Osborn wave emerges in ECG leads in which the mean

vector axis is transmurally oriented across the left ventricle and septum and is therefore usually evident in leads II, III, aVF, and V3–V6. Vectorcardiography indicates that the Osborn wave forms an additional loop that happens at the junction of the QRS and T loops, which is directed leftward and anteriorly explaining its prominence in leads associated with the left ventricle[28,29]. This observation was evident in many case reports illustrating the ECG distribution of the hypothermic Osborn wave. Higuchi and colleagues found that Osborn waves were evident in the inferolateral leads in 70% of their cases[30]. As the core body temperature decreases, the Osborn waves become evident in more ECG leads and not just the inferolateral leads. It was found that the number of ECG leads with an evident Osborn wave were significantly higher in the severe hypothermia compared with the mild and moderate hypothermia groups[31,32].

Electrocardiographic characteristics of the Osborn wave

Hypothermia remains the primary reason for witnessing an Osborn wave in the ECG. The Osborn wave is reversible with correction of the precipitating factor for its growth. At the R-ST junction of the ECG, it appears as a positive deflection with a dome-shaped configuration. The Osborn wave appears in ECG leads where the mean vector axis is transmurally oriented across the left ventricle and septum, as Yan and Antzelevitch demonstrated experimentally, and is thus typically visible in leads II, III, aVF, and V3–V6. The Osborn wave forms an extra loop at the junction of the QRS and T loops, which is guided leftward and anteriorly, explaining its prominence in leads connected with the left ventricle, according to vectorcardiography. Many case studies demonstrating the ECG distribution of the hypothermic Osborn wave rendered this finding. In 70% of the cases studied, Higuchi and colleagues discovered Osborn waves in the inferolateral leads. The Osborn waves appear in more ECG leads, not just the inferolateral leads, as the core body temperature rises. When compared to the mild and moderate hypothermia groups, the number of ECG leads with an obvious Osborn wave was substantially higher in the extreme hypothermia community.

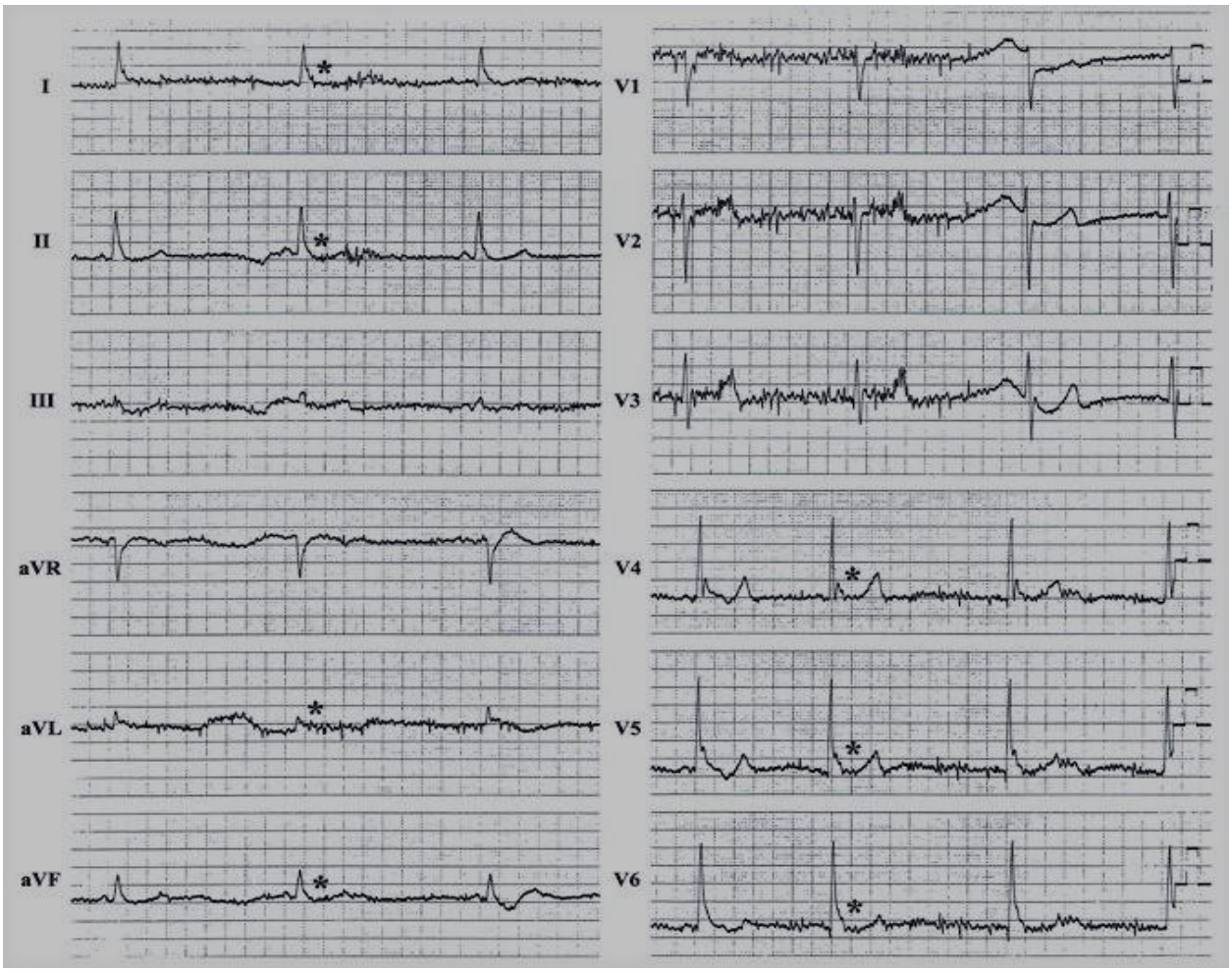

Figure 3: Twelve-lead ECG in a patient with osborn waves fluctuations.

Hypothermia Induced ECG Abnormalities (J WAVES)

The J waves (FIGURE 3) finding of hypothermia include: The "Osborne wave" in the QRS complex is characterized by a label in the downward part of the R wave and is associated with hypothermia, hypocalcemia and Bragada syndrome; Due to obesity, chronic obstructive pulmonary disease or COPD, pericardial effusion, low voltage on the ECG may occur; Osbornwaves are responsible for sinus bradycardia, junctional bradycardia, atrial fibrillation with sluggish

ventricular response or higher-grade AV blocks; Baseline hypothermia signs are shivering and shivering [33,34].

Osborn wave hypothermia has been reported to decrease systolic and diastolic left ventricular function. A case involved a patient with hypothermia with reduced heart rate, pulse rate, respiratory rate, elevated systolic pressure, and prolonged progression of the ejection period to left ventricular systolic dysfunction with low ejection fraction [35,36].

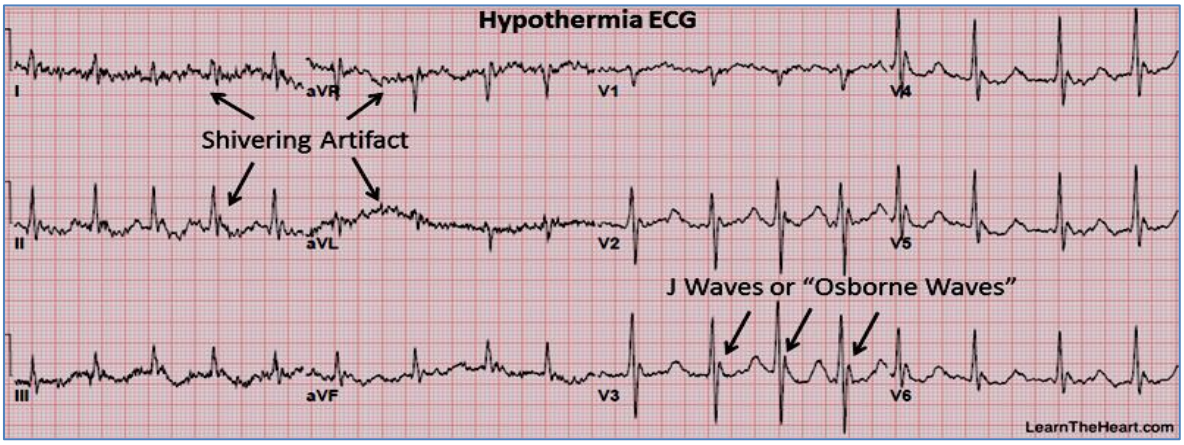

Figure 4: Hypothermia Induced ECG Abnormalities (J WAVES)

The ischemic Osborn waves

The ischemic Osborn wave was observed in several reports of patients with ST-segment elevation myocardial infarction (STEMI)[37,38]. In the study by Aïssou and colleagues, 47% (74/159) of patients with ACS (94% of whom had STEMI) had ischemic Osborn waves. Osborn waves were also reported during vasospastic angina[39]. This was shown in case reports by Maruyama and colleagues and Sacha and colleagues, who both the cellular basis for ST-segment changes observed during ischemia was defined by Di Diego and colleagues. During step 1 of repolarization, acute ischemia opens the KATP channels (creating Ito current) inside the epicardium [40,41]. The action potential of the endocardium shows a much smaller Ito current. This difference creates a transmural voltage gradient during early ventricular repolarization leading to the development of the Osborn waves [42,43]. Persistence of ischemia results in loss of epicardial nerve impulse dome increasing the transmural voltage gradient reflected as ST-segment elevation within the ECG with amalgamation of the "J wave" into the elevated ST-segment [44,45,46].

Association of the Osborn wave and core body temperature

The fact that the Osborn wave amplitude is inversely associated with the core body temperature has been illustrated by published case reports, leading to an increase in its amplitude with a decrease in temperature and a decrease in amplitude with re-warming accompanied by its absence when approaching normothermia [48,49]. To further highlight the intensity of this association, by evaluating all recorded cases of hypothermic Osborn waves, we have recently published the results of the correlation between the amplitude of the Osborn wave and core body temperature [50,51]. There was a clear inverse association between the Osborn wave amplitude and temperature when looking only at cases with more than one recorded ECG during the index hospitalization and the respective core body temperature [52]. Osborn waves were evident in 80 and 51,6 percent of patients who presented with accidental hypothermia, respectively, in the studies by Okada and colleagues and Higuchi and colleagues. The Osborn wave presence frequency in the above study was 100% with extreme hypothermia, 75% with moderate hypothermia, and 10.7% with mild hypothermia. In addition to hypothermia, there are potentially other variables that relate to the appearance of Osborn waves [53,54]. In his research, John Osborn found that in hypothermic animals, the Osborn wave reflected a current of injury caused by acidosis [55,56]. He noticed that when the pH was maintained in the normal range through mechanical ventilation, these waves were absent. Edelman and Joynt also identified this finding when they reported the case of a hypothermic (33.3ºC) and profoundly academic (pH 7.03) patient after cardiac arrest who displayed Osborn wave on the initial ECG that, despite the same degree of hypothermia, disappeared after improvement of acidosis through mechanical ventilation [57,58,59].

Osborn waves effect Cardiovascular System

Tachycardia reflects the early cardiovascular response to hypothermia. However, the heart rate slows and cardiac production decreases as the body cools below 35 ° C [60,61]. Hypotension, cardiac contractility and intravascular volume can occur secondary to decreased cardiac performance [62,63]. Electrocardiographic results are non-specific and include prolongation of the intervals of PR, QRS, and QTc, as well as shifts in the ST-segment and T-wave [64,65].

Materials and Methods

The PubMed and Embase databases were used to conduct a systematic literature search. A summary of all articles published between 1950 and 2014 that recorded

hypothermic J waves for patient populations, core body temperature in degrees Celsius (°C), J wave amplitude in millimetres (mm), J wave lead with the highest amplitude, presence of acidosis, PO2, electrolytes, and outcome[66,67]. The core body temperature and J wave amplitude of each electrocardiogram (ECG) were reported in cases where there were multiple ECGs [68]. The main goal of the analysis is to see if there's a connection between J wave amplitude and core body temperature in the admission ECG [69]. We have also examined the strength of this relationship in cases with more than one ECG. We attempted to find the most frequent lead that recorded the highest amplitude of the J wave in addition to the correlation between the amplitude of J wave and Ph. All identified articles were screened and checked for eligibility [70,71,72].

Articles identifying through database searching n=100 (Pubmed, Embase, Scopus)

⇩

Article included after screening for tittle n= 72

⇩

Articles only based on osborn waves and related to cardiovascular disease were included

⇩

Articles which are irrelevant to cardiovascular disease were excluded

DISCUSSION

Imad A. Alhaddad et al., concluded a study on Osborn Waves of Hypothermia. Osborn waves, also known as J waves, camel-hump waves, and hypothermic waves, are best seen in the inferior and lateral precordial leads, according to this analysis. They become more noticeable as the body temperature decreases, and they eventually fade as the body temperature rises.

Takanao Mine et al., concluded a case report on Left ventricular systolic dysfunction in a patient with accidental hypothermia. Hypothermia, according to this research, can inhibit left ventricular contraction, resulting in a longer ejection time and a lower heart rate. These results may be indicative of hypothermia-induced cardiac dysfunction.

Okada M et al., concluded a study on The J wave in accidental hypothermia. This research suggests that even after normothermia was restored, a small J wave remained in many instances. It was also difficult to tell the difference between these small J waves and small notches at the QRS-ST junction, which are often seen in healthy people who haven't been exposed to hypothermia.

Hesham R. Omar MD et al., concluded a study on The Osborn wave: what have we learned. This study suggests that further research is needed to determine whether the Osborn wave occurs in hypothermic states predisposes to VF or not. It's also important to keep in mind that ECG abnormalities in hypothermia could signal myocardial ischemia. This awareness will help to avoid the overuse of potentially risky medications like anticoagulant or thrombolytic therapy in hypothyroid patients that could become coagulopathic as a result of their condition.

Mitsunori Maruyama et al., concluded a study on Osborn Waves: History and Significance In their paper, this research suggests that the frequency of ventricular fibrillation was linked to the augmentation of the Osborn waves; a similar phenomenon was recently reported in a patient with a non-Q wave myocardial infarction due to extreme coronary vasospasms.

Hurst JW et al., concluded a study on Abnormalities of the S-T segment, part I. Clin Cardiol this study suggests that the method of identifying primary and secondary S-T segment anomalies, as well as their importance. With a few exceptions, it is part of the repolarization mechanism and thus part of the T wave when a mean vector constructed for the S-T segment displacement seen in 12 ECG leads is relatively parallel with a mean vector representing the T wave.

Abbott JA et al., concluded a study on the nonspecific camel-hump sign. According to this study, hyopthermia is the cause of camel hump, which results in ECG shifts, cardiovascular disease, and celebral injury.

Douglas J.A. Brown et al., concluded a study on Accidental Hypothermia. This study suggests that advances in the safety and availability of rewarming techniques have improved the prognosis for hypothermia patients, especially those who are treated with extracorporeal rewarming after cardiac arrest.

Tetsuji Shinohara et al., concluded a study on Characterization of J wave in a patient with idiopathic ventricular fibrillation. Hypothermia, according to this research, can inhibit left ventricular contraction, resulting in a longer ejection time and a lower heart rate.

D.Emsile smith et al., concluded a study on the significance of changes in the electrocardiogram in hypothermia. According to this study, death from ventricular fibrillation is the most serious risk associated with the current use of hypothermia in heart and brain surgery. In order to better understand the importance of this deflection, it was observed in hypothermic patients with normal hearts and hypothermic dogs using direct epicardial electrodes.

Gustavo X. Morales et al., concluded a study on Giant J-wave (Osborn wave) unrelated to hypothermia. This study suggests that J-waves in hypercalcaemia are thought to be caused by a rise in the calcium-activated outward current and a decrease in the inward calcium current, according to this report. During ventricular repolarization, this resulted in all-or-none repolarization of the action potential (end of

Phase 1 in the epicardium), resulting in an Ito channel-mediated transmural voltage gradient.

José M Di Diego et al., concluded a study on Cellular basis for ST-segment changes observed during ischemia. This study suggests that the cellular basis for ischemia-induced ST-segment elevation was investigated using an isolated arterially perfused canine ventricular wedge preparation. Transmembrane action potentials (AP) from epicardial (Epi) and endocardial (Endo) regions, a pseudo-electrocardiogram (ECG), and five intramural unipolar electrograms were reported simultaneously at a simple cycle duration of 800 or 2,000 ms.

M Davidson et al., concluded a study on Accidental hypothermia: a community hospital perspective. This study suggest that Diabetes and alcohol dependence tend to be risk factors for hypothermia, as they were found in 18 (30%) and 14 (23%), respectively, of our patients. Every emergency department should have a plan in place for identifying and managing hypothermia victims so that effective rewarming procedures can be implemented quickly.

Lenihan DJ et al., concluded a study on the "Normothermic" Osborn Wave Induced by Severe Hypercalcemia. This study suggest that the electrocardiographic J wave has been associated with hypothermia since the early studies by Osborn 1 and is commonly referred to as an Osborn wave, according to this research. Several subsequent studies have indicated that this electrical anomaly may be caused by factors other than hypothermia.

Cardenas GA et al., concluded a study on Osborn waves in sepsis. This study suggests that the electrophysiologic mechanisms of Osborn waves, as well as the differential diagnosis of this electrocardiographic finding and other hypothermia-related findings

CONCLUSION

There are diagnostic and prognostic consequences of the emergence of the Osborn wave in the ECG that illustrate the significance of its identification by different health care providers. Even if the diagnosis of hypothermia is initially missed with the widely used oral or tympanic thermometer, the hypothermic ECG features allow for early detection and treatment of hypothermic patients, and particularly because hypothermia can present with misleading symptoms, such as confusion, dizziness or dyspnea. Its arrhythmogenic potential is more significant, especially in patients with myocardial ischemia, where it has been shown to be a precursor of malignant ventricular arrhythmias, and hence the importance of continuous monitoring of telemetry in these subjects. Whether or not the occurrence of the Osborn wave predisposes to VF in hypothermic states still needs further study. Knowledge of this will avoid the excessive administration of potentially dangerous drugs in subjects that could be coagulopathic to hypothermia, such as anticoagulant or thrombolytic therapy and also Hypothermia can impair left ventricular contraction and may specifically result in prolongation of the ejection time and reduced heart rate. These findings may be a characteristic finding of hypothermia-induced cardiac dysfunction

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