**J Waves in Accidental Hypothermia**

– Body Temperature and Its Clinical Implications –

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**Background:** The J wave is an ECG marker of ventricular fibrillation. However, the prevalence and clinical implications of J waves in hypothermic patients remain unclear.

**Methods and Results:** We evaluated the clinical characteristics and ECGs of patients who were admitted for accidental hypothermia (<35.0°C). J waves were defined as notches or slurs in the terminal part of the QRS complex with an amplitude ≥0.1 mV. We analyzed the prevalence of J waves and the relationship between body temperature (BT) and J wave amplitude. We also examined the augmentation of J waves following variable R-R intervals in patients with atrial fibrillation. Furthermore, we assessed the incidence of ventricular arrhythmias. A total of 60 hypothermic patients were recruited (mean age, 64±9 years; 97% male). The mean BT was 31.3°C (range, 29.4–33.5°C). J waves, which disappeared after rewarming, were observed in 30 patients (50%), with a higher frequency in patients with lower BT. Higher amplitude of J waves was associated with lower BT (P<0.001). Of the 8 patients with J waves and atrial fibrillation, 4 exhibited an augmentation of J waves following a short R-R interval. Only 1 patient with J waves developed ventricular tachycardia during rewarming.

**Conclusions:** The prevalence of J waves and their amplitude increased with the severity of hypothermia. The temporal development of J waves might not be associated with fatal arrhythmic events. (Circ J 2014; 78: 128–134)

**Key Words:** Hypothermia; J wave; Ventricular fibrillation; Ventricular tachycardia

The J wave, also known as the Osborn wave,1 is a positive deflection at the terminal part of the QRS complex. Although J waves are often observed in hypothermic patients,2 few reports have revealed the relationship between the presence of J waves and body temperature (BT). The exact BT at which J waves develop in clinical settings has not yet been determined.

The J wave is noted not only in hypothermia but also in some nonhypothermic conditions such as Brugada and early repolarization syndromes. The presence of J waves in such syndromes is related to the occurrence of ventricular fibrillation (VF), leading to sudden death. However, whether hypothermic patients with J waves are susceptible to fatal ventricular arrhythmia or ventricular tachycardia (VT)/VF remains undetermined. Indeed, some previous reports have shown that the incidence of VT/VF is unexpectedly low in hypothermic patients with J waves, varying from 0% to 2%.3-5 The augmentation of J waves following a long R-R interval is reported to be a marker for VF6,7 because the heterogeneity of membrane potentials is expressed as augmentation of J waves and can induce VF due to phase 2 reentry.8 A previous report demonstrated that VF developed after sudden augmentation of J waves in idiopathic VF, which was induced following a long coupling interval.6 Such an event in hypothermic patients would serve as a warning sign for identifying patients at high risk of developing VF.

In the present study, we sought to clarify the relationship between the incidence of J waves and BT, as well as the clinical implications of J waves in patients with accidental hypothermia. We also attempted to determine the augmentation of J waves following a long R-R interval in patients with atrial fibrillation (AF).

**Methods**

**Study Design**

We studied 64 consecutive patients who were admitted to hospital for accidental hypothermia between 2002 and 2012. Of these, 63 were homeless people admitted during the winter. The remaining patient was not homeless, but succumbed to hypothermia in a cold house because of depression. We reviewed all the patients’ medical records to evaluate clinical characteristics such as age, sex, vital signs, cause of hypothermia, ECG findings, and laboratory data on admission. Hypothermia was defined as a rectal temperature <35.0°C.9 Of the 64 patients, 4...
J Waves in Hypothermia

QT and QTc intervals. In patients with AF, the R-R interval was measured as the average of 3 continuous beats. The PR interval was measured only in sinus rhythm. The QTc interval was calculated using Bazett’s formula.

J waves were defined as notches or slurs in the terminal part of the QRS complex, with an amplitude ≥0.1 mV above the isoelectric line in at least 2 contiguous leads. Persistent notches and slurs after rewarming to 35.0°C were not regarded as J waves related to hypothermia. Typical J waves on serial ECGs of a hypothermic patient are shown in Figure 1. We examined the relationship between the J-wave amplitude and BT at which the J wave appeared. The J-wave amplitude was measured as the difference between the top of the J wave and the isoelectric line. We measured the highest amplitude in the leads in which the J waves were recorded. The location of the J waves was classified into 4 sites: inferior (II, III, aVF), right precordial (V1–3), left precordial (V4–6), and high lateral (I, aVL). We evaluated the site where J waves appeared most frequently and were excluded because of right bundle branch block. No patient had left bundle branch block or preexcitation syndrome. Thus, a total of 60 patients were analyzed in the present study. All patients were rewarmed within 6 hours of arrival. We compared the clinical characteristics and basic ECG parameters of patients with and without J waves. In addition, we classified the patients into 3 groups on the basis of the severity of hypothermia:10 mild (BT ≥32.0°C), moderate (BT, 28.0–32.0°C) and severe (BT <28.0°C); we assessed the characteristics of the J waves in these 3 groups.

The study was approved by the institutional ethical review board of the hospital, which with financial support from the Japanese government, has developed special care units for homeless people who need medical treatment.

ECG Analysis

The following ECG parameters were analyzed: cardiac rhythm, R-R interval, PR interval, duration of the QRS complex, and QT and QTc intervals. In patients with AF, the R-R interval was measured as the average of 3 continuous beats. The PR interval was measured only in sinus rhythm. The QTc interval was calculated using Bazett’s formula.

J waves were defined as notches or slurs in the terminal part of the QRS complex, with an amplitude ≥0.1 mV above the isoelectric line in at least 2 contiguous leads. Persistent notches and slurs after rewarming to 35.0°C were not regarded as J waves related to hypothermia. Typical J waves on serial ECGs of a hypothermic patient are shown in Figure 1. We examined the relationship between the J-wave amplitude and BT at which the J wave appeared. The J-wave amplitude was measured as the difference between the top of the J wave and the isoelectric line. We measured the highest amplitude in the leads in which the J waves were recorded. The location of the J waves was classified into 4 sites: inferior (II, III, aVF), right precordial (V1–3), left precordial (V4–6), and high lateral (I, aVL). We evaluated the site where J waves appeared most frequently and...
Clinical Outcome

We assessed the survival and major adverse cardiovascular events such as fatal ventricular arrhythmias or VT/VF in hypothermic patients during the follow-up period.

Statistical Analysis

Numerical data are presented as mean ± standard deviation if the data followed a normal distribution. Otherwise, data are shown as median and interquartile range (quartile 1–quartile 3). Categorical variables are expressed as absolute numbers or percentages. Continuous variables were analyzed using unpaired Student’s t-test, Mann-Whitney test, or Kruskal-Wallis rank test. Fisher’s exact test or the χ² test was used for categorical variables. Bonferroni corrections were used for post-hoc multiple tests. The relationship between the amplitude of the J wave and BT and the correlation among the number of J waves and the severity of hypothermia (mild, moderate, and severe) were assessed by a nonparametric test of trend. A P value <0.05 was considered statistically significant. All statistical analyses were performed with Stata software (version 10; Stata Corp, College Station, TX, USA).

Results

Clinical Characteristics and J Waves Related to Accidental Hypothermia

A total of 60 patients with accidental hypothermia were examined (mean age, 64±9 years; 97% male). The median BT was

Table 1. Patients’ Characteristics and J Waves

<table>
<thead>
<tr>
<th></th>
<th>All (n=60)</th>
<th>J wave (n=30)</th>
<th>No J wave (n=30)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>64.4±9.0</td>
<td>65.5±8.2</td>
<td>63.2±9.8</td>
<td>NS</td>
</tr>
<tr>
<td>Male</td>
<td>58 (97%)</td>
<td>29 (98%)</td>
<td>29 (98%)</td>
<td>NS</td>
</tr>
<tr>
<td>Body temperature (°C)</td>
<td>31.3 (29.4–33.5)</td>
<td>29.4 (27.0–30.5)</td>
<td>33.4 (32.3–34.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>115±12</td>
<td>111±23</td>
<td>120±26</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>75 (56–83)</td>
<td>59 (47–78)</td>
<td>79 (71–83)</td>
<td>0.002</td>
</tr>
<tr>
<td>Laboratory data</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na (mEq/L)</td>
<td>142±7</td>
<td>141±5</td>
<td>143±8</td>
<td>NS</td>
</tr>
<tr>
<td>K (mEq/L)</td>
<td>4.2±1.0</td>
<td>4.4±1.0</td>
<td>4.0±1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Cr (mg/dl)</td>
<td>1.1 (0.8–1.7)</td>
<td>1.1 (0.8–1.7)</td>
<td>1.1 (0.8–1.4)</td>
<td>NS</td>
</tr>
<tr>
<td>Alb (mg/dl)</td>
<td>3.3±0.6</td>
<td>3.3±0.6</td>
<td>3.3±0.7</td>
<td>NS</td>
</tr>
<tr>
<td>Ca (mg/dl)</td>
<td>8.3 (7.6–9.0)</td>
<td>8.4 (8.0–9.1)</td>
<td>8.2 (7.3–8.8)</td>
<td>NS</td>
</tr>
<tr>
<td>corrected Ca (mg/dl)</td>
<td>9.1 (8.4–9.6)</td>
<td>9.3 (8.7–9.5)</td>
<td>8.7 (8.3–9.7)</td>
<td>NS</td>
</tr>
<tr>
<td>pH</td>
<td>7.35 (7.30–7.45)</td>
<td>7.33 (7.31–7.41)</td>
<td>7.39 (7.29–7.47)</td>
<td>NS</td>
</tr>
<tr>
<td>ECG characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sinus rhythm</td>
<td>42 (70%)</td>
<td>17 (57%)</td>
<td>25 (83%)</td>
<td>0.047</td>
</tr>
<tr>
<td>Junctional rhythm</td>
<td>5 (8%)</td>
<td>5 (16%)</td>
<td>0 (0%)</td>
<td>NS</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>13 (22%)</td>
<td>8 (27%)</td>
<td>5 (17%)</td>
<td>NS</td>
</tr>
<tr>
<td>PR interval (ms)</td>
<td>180 (160–200)</td>
<td>200 (180–200)</td>
<td>160 (160–180)</td>
<td>0.013</td>
</tr>
<tr>
<td>QRS interval (ms)</td>
<td>100 (80–160)</td>
<td>160 (140–200)</td>
<td>80 (80–100)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>QT interval (ms)</td>
<td>480 (440–520)</td>
<td>520 (440–560)</td>
<td>440 (420–480)</td>
<td>0.0011</td>
</tr>
<tr>
<td>QTc interval (ms)</td>
<td>519 (492–539)</td>
<td>520 (500–542)</td>
<td>511 (480–537)</td>
<td>NS</td>
</tr>
<tr>
<td>Cause of hypothermia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exposure to cold</td>
<td>32 (53%)</td>
<td>13 (43%)</td>
<td>19 (63%)</td>
<td>NS</td>
</tr>
<tr>
<td>Infection</td>
<td>19 (32%)</td>
<td>13 (43%)</td>
<td>6 (20%)</td>
<td>NS</td>
</tr>
<tr>
<td>Alcohol</td>
<td>7 (12%)</td>
<td>3 (10%)</td>
<td>4 (13%)</td>
<td>NS</td>
</tr>
<tr>
<td>Others</td>
<td>2 (3%)</td>
<td>1 (3%)</td>
<td>1 (3%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are n (%), mean±SD, or median (interquartile range). P<0.05 is considered statistically significant. NS, not significant. Continuous variables were analyzed using unpaired Student’s t-test, Mann-Whitney test, or Kruskal-Wallis rank test. Categorical variables were analyzed using the χ² tests or Fisher’s exact test.

Augmentation of J Waves

We defined the augmentation of J waves as an increment of 0.1 mV compared with the preceding beat. Hypothermia can induce AF, which is characterized by variable R-R intervals. We recorded the amplitude of the J wave and the preceding R-R interval in AF patients with J waves. We investigated whether the augmentation of J waves following a long R-R interval would occur in the same manner as that observed in patients with idiopathic VF. Furthermore, to determine whether the J-wave augmentation is affected by variation of the preceding R-R intervals, we calculated a ratio of the J-wave amplitude of the corresponding beat to that of the preceding beat, which was defined as an augmentation ratio, as well as a ratio of the preceding R-R interval to the following. The correlation between the augmentation ratio and the preceding R-R interval ratio was assessed in all the ECGs in which J waves and AF were recorded.

Laboratory Data

Blood samples were drawn on each patient’s arrival and were analyzed inhouse. If serum albumin was <4.0 mg/dl, the corrected serum calcium was calculated: calcium (mg/dl) +4.0 – albumin (mg/dl).

the association between the number of sites and the severity of hypothermia.

Two experienced cardiologists read, measured, and interpreted all ECGs.

Clinical Outcome

We assessed the survival and major adverse cardiovascular events such as fatal ventricular arrhythmias or VT/VF in hypothermic patients during the follow-up period.

Statistical Analysis

Numerical data are presented as mean ± standard deviation if the data followed a normal distribution. Otherwise, data are shown as median and interquartile range (quartile 1–quartile 3). Categorical variables are expressed as absolute numbers or percentages. Continuous variables were analyzed using unpaired Student’s t-test, Mann-Whitney test, or Kruskal-Wallis rank test. Fisher’s exact test or the χ² test was used for categorical variables. Bonferroni corrections were used for post-hoc multiple tests. The relationship between the amplitude of the J wave and BT and the correlation among the number of J waves and the severity of hypothermia (mild, moderate, and severe) were assessed by a nonparametric test of trend. A P value <0.05 was considered statistically significant. All statistical analyses were performed with Stata software (version 10; Stata Corp, College Station, TX, USA).

Results

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A total of 60 patients with accidental hypothermia were examined (mean age, 64±9 years; 97% male). The median BT was
J Waves in Hypothermia

31.3°C (interquartile range, 29.4–33.5°C). J waves were observed in 31 patients on admission. Such J waves completely disappeared after rewarming in 30 patients. One patient who had persistent J waves was not included in the J-wave group, because the amplitude (0.1 mV) and location (inferior) of the J waves did not change after rewarming. Therefore, in our study, 30 patients (50%) exhibited hypothermia-related J waves. Clinical and ECG characteristics are shown in Table 1. There was no significant difference between patients with and without J waves in terms of age, sex, blood pressure, electrolytes, pH, or cause of hypothermia. Patients with J waves had decreased heart rates compared with those without J waves (P=0.002). The presence of J waves was associated with longer PR interval (P=0.013), longer QRS interval (P<0.0001) and lower frequency of sinus rhythm (P=0.047). QT interval was also longer in patients with J waves than those without J waves (P=0.0011), although the QTc interval was similar in both groups. Among the 30 patients who exhibited J waves, hypothermia was caused by exposure to cold (n=13), infection (n=13), alcohol (n=3), and adrenal failure (n=1).

Table 2. J-Wave Characteristics and Severity of Hypothermia

<table>
<thead>
<tr>
<th>ECGs, n</th>
<th>All</th>
<th>Mild (A) (32.0°C–34.9°C)</th>
<th>Moderate (B) (28.0°C–31.9°C)</th>
<th>Severe (C) (&lt;28.0°C)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>J wave</td>
<td>60</td>
<td>28</td>
<td>20</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Location of J wave, n</td>
<td>Inferior</td>
<td>21</td>
<td>3</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>High lateral</td>
<td>4</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Right precordial</td>
<td>8</td>
<td>0</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Left precordial</td>
<td>21</td>
<td>1</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>No. of J-wave sites</td>
<td>1</td>
<td>15 (25%)</td>
<td>2 (7%)</td>
<td>8 (40%)</td>
<td>5 (42%)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>8 (13%)</td>
<td>1 (4%)</td>
<td>4 (20%)</td>
<td>3 (25%)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>5 (8%)</td>
<td>0 (0%)</td>
<td>3 (15%)</td>
<td>2 (17%)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>2 (3%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>2 (17%)</td>
</tr>
</tbody>
</table>

Values are n (%). P<0.05 is considered statistically significant for Kruskal-Wallis rank test or np trend test. Bonferroni's correction was applied for P value from Fisher’s exact test for comparison between groups. NS, not significant.

Relationship Between J Waves and BT

As shown in Table 1, patients with J waves had lower BT than those without J waves (P<0.001). Figure 2 shows that all pa-
J-wave characteristics are presented for comparison among patients whose BT was <30.0°C had J waves. None of the patients with BT >33.5°C developed J waves. Furthermore, the amplitude of J waves increased with decreasing BT ($\rho=-0.75$, $n=60$, $P<0.001$). Of note, the amplitude of the J waves was as high as 1.0 mV or more in 3 patients whose BT was <28.5°C.

**J-Wave Characteristics and Severity of Hypothermia**

J-wave characteristics are presented for comparison among patients...
the 3 groups that were divided on the basis of the severity of hypothermia (Table 2). J waves were present in 100% patients in the severe group, in 75% of the moderate group, and in only 10.7% patients in the mild group. In addition, J waves were noted at 1 (n=15), 2 (n=8), 3 (n=5), and at all sites (n=2). J waves were evident most frequently in the left precordial and inferior leads (n=21 for both), followed by the right precordial (n=8) and high lateral leads (n=4). The number of sites where J waves appeared was significantly higher in the severe group than in the other groups (P<0.0001).

**Augmentation of J Waves**

The augmentation of J waves was analyzed exclusively in hypothermic patients with AF. A total of 13 patients were in AF rhythm and 8 of them had J waves. The augmentation of J waves never occurred following a longer R-R interval. Conversely, 4 ECGs showed augmentation of J waves following a shorter R-R interval (Figure 3A) and the other 4 ECGs did not show any augmented J waves (Figure 3B). When the relationship between augmentation of the J wave and the preceding R-R interval was assessed in the 8 patients with AF, the augmentation ratio inversely correlated with the preceding R-R interval ratio (r=−0.45, n=90, P<0.0001) (Figure 4). These data indicate that hypothermia-related J waves tend to be augmented following a relatively short R-R intervals in AF patients.

**Clinical Outcome**

During hospitalization, only 1 patient with J waves developed sustained VT, which spontaneously converted to sinus rhythm, while being treated by rewarming (BT was 27.8°C). This patient had no structural heart disease and normal left ventricular function on echocardiography. None of the patients developed VF during follow-up. After rewarming, 1 patient with J waves died of acute kidney injury, which was unrelated to heart disease or hypothermia. With regard to severe complications accompanying hypothermia, 1 patient had septic shock and 2 had acute respiratory distress syndrome, but all recovered completely.

**Discussion**

A major finding of the present study is that the frequency of J waves increased with decreasing BT in patients with accidental hypothermia. It is noteworthy that J waves appeared definitively as BT fell below 30.0°C. Furthermore, the number of sites where J waves appeared on the ECG and the amplitudes of the J waves positively correlated with the severity of hypothermia.

A decrease in temperature can accelerate inactivation of the Na channel and decrease the amplitude of the action potential in the epicardium but not in the endocardium. This difference in action potential activity leads to the development of a transmural voltage gradient that manifests as a prominent J wave on ECG. This mechanism can lead to phase 2 reentry. Severe hypothermia may increase the transmural voltage gradient, resulting in a higher amplitude of J waves in more areas. Our study showed a decrease in the height of the J wave as patients recovered from hypothermia. It is possible that warming reduces the difference between the epicardium and the endocardium in the action potential.

In the early repolarization and Brugada syndromes, J waves rise in response to bradycardia or a pause. However, our study did not demonstrate an augmentation of the J wave following a long R-R interval. On the contrary, some ECGs revealed augmentation of J waves following a short R-R interval. This discrepancy suggests a possible mechanistic difference between a hypothermia-induced J wave and a J wave formed in the early repolarization and Brugada syndromes. An increase in the transient outward potassium current (I_{to}) induced by bradycardia is mainly responsible for a prominent action potential dome, leading to augmentation of the J wave. A plausible reason why the J waves were not augmented after a long R-R interval in our study might be suppression of I_{to} in the setting of hypothermia. Li et al reported that the amplitude and density of I_{to} decreased at 23°C as compared with 36°C in isolated myocytes from human hearts. The time-dependent inactivation and reactivation of I_{to} was attenuated at lower temperature, suggesting an inhibitory effect of low temperature on I_{to}. In addition, a possible explanation for J-wave augmentation after a short R-R interval would be that action potential delay might be more involved in the manifestation of hypothermia-induced J waves, rather than repolarization abnormalities related to prominent I_{to}. This is suggested by our finding that J waves were augmented following long–short sequences of the preceding R-R intervals, which resembles the characteristic refractory period that can lead to aberrant conduction known as the Ashman phenomenon. Augmented J waves after a relatively short R-R interval could be explained by conduction delay induced by suppression of Na channels, although the precise electrophysiological mechanisms remain unclear.

Although we detected J waves in 30 hypothermic patients, only 1 patient developed sustained VT. Therefore, we could not determine the relationship between the presence of J waves and the incidence of fatal ventricular arrhythmias in accidental hypothermia. It is possible that the majority of hypothermic patients in our study did not have the substrate for malignant ventricular tachyarrhythmias that would be unmasked by a trigger such as hypothermia. There are some reports of patients with early repolarization syndrome and J waves in whom therapeutic hypothermia increased the J-point elevation with multiple recurrent episodes of VF. Spontaneous or therapeutic hypothermia may serve as a trigger for VF in patients with early repolarization syndrome, in whom bradycardia or a pause induces J-wave augmentation, suggesting a rate-dependent precipitating factor for phase 2 reentry. In our study, hypothermia caused bradycardia and QT prolongation with prominent J waves on ECG, but these changes were normalized after rewarming. Because patients with early repolarization syndrome have a relatively short QTc interval, even in the clinical setting of hypothermia, we postulate that a relatively long QTc interval in healthy subjects exposed to a cold environment may have a protective effect against the development of phase 2 reentry, presumably through action potential delay. Thus, we would consider that hypothermia-induced J waves in healthy subjects are different from those in early repolarization and Brugada syndromes and idiopathic VF in terms of the electrophysiological mechanisms and arrhythmogenic characteristics.

Our study population mainly comprised homeless people. Previous studies did not necessarily include people who were subjected to a cold environment but those suffering from severe systemic diseases that could affect the ECG findings. ECG findings can be also modified by comorbidities, particularly severe heart disease. Ingestion of large amounts of alcohol and/or illegal drugs such as cocaine can also impair the myocardium, thus affecting the ECG findings. One report focused on homeless people with hypothermia, who were also alcoholics or drug users. Patients with drug addiction are uncommon in Japan, but there are many such people in other regions such as North and South America and central and western Europe.
Indeed, our study included only 3 alcoholic patients. Furthermore, patients with infections accounted for less than one-third of our study population. Therefore, it is assumed that the ECG changes observed in our study were less influenced by concomitant factors other than hypothermia.

Despite the number of cases of severe hypothermia, the resultant mortality was very low (1.7%). This finding is not consistent with other previous reports, which indicate a mortality rate between 12% and 38%.\textsuperscript{3,5,13} This difference in results may be explained by different etiologies of hypothermia. Vassal et al. reported 38% mortality in patients with severe underlying conditions such as cardiogenic shock, cardiopulmonary arrest, and accidental falls,\textsuperscript{5} which were not present in our study patients. In contrast, the main etiology of hypothermia in our study was not a serious life-threatening condition but exposure to a cold environment, which can be readily treated by rewarming. Thus, we would speculate that most of the patients in our study were essentially healthy except for being hypothermic.

\textbf{Study Limitations}

Our sample size may not have been adequate to allow the detection of significant differences in event rates because of its low frequency. Because early rewarming was achieved in all patients, the duration of hypothermia may have been too short to induce fatal tachyarrhythmias. If the patients' hypothermia had lasted for longer, they would have been more susceptible to VT/VF. In addition, the accurate fatal arrhythmic event rate is still uncertain, because hypothermic patients who died outside of the hospital were not included in the present study. Further research with a sufficient number of hypothermic patients may be required to confirm our findings.

In the present study, we interpreted the ECG findings based on interpatient and not intrapatient comparisons. Findings based on intrapatient comparisons will probably enable more precise interpretations regarding the characteristics of J waves.

\textbf{Conclusions}

In this study, J waves were found in 50% of a series of hypothermic patients. All the patients whose BT was less than 30.0°C developed J waves. Furthermore, the amplitude of the J waves and the number of sites where J waves appeared was related to the severity of hypothermia. However, the temporal development of J waves might not be associated with fatal arrhythmic events.

\textbf{Disclosures}

Financial support for this study was not provided. The authors report no conflicts of interest.

\textbf{References}