Quantitative Assessment of the Effects of Therapeutic Hypothermia on Early Repolarization in Idiopathic Ventricular Fibrillation Survivors - A Seven Year Cohort Study

**Running title:** Williams et al.; Early repolarization in therapeutic hypothermia

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**Journal Subject Codes:** [171] Electrocardiology, [5] Arrhythmias, clinical electrophysiology, drugs
Abstract:

**Background** - The early repolarization (ER) pattern on the electrocardiogram (ECG) is associated with an increased risk of idiopathic ventricular fibrillation (VF). Hypothermia is known to result in similar electrocardiographic changes. In this retrospective cohort study we examine the impact of therapeutic hypothermia on ER in survivors of cardiac arrest attributed to idiopathic VF (ID-VF) and draw comparisons with a control group who experienced coronary artery disease related VF (CAD-VF).

**Methods and Results** - All patients who suffered cardiac arrest and were treated with therapeutic hypothermia over a 7 year period were considered for inclusion in the study. 43 patients were identified with ID-VF or CAD-VF arrest. ECGs were obtained during cooling and again after rewarming. ECGs were digitized and assessed for the presence of ER by two independent observers. Cooling significantly increased the prevalence (74 % during cooling vs. 51 % at baseline temperature, \( P=0.044 \)) and mean amplitude (0.78±0.10 mV during cooling vs. 0.56±0.09 mV at baseline temperature, \( P=0.038 \)) of ER in the overall cohort. During cooling ER was more common among survivors of idiopathic VF than of CAD-VF (100 % vs. 67 %, \( P=0.043 \)). ER magnitude was significantly greater among idiopathic VF survivors than CAD-VF survivors both during cooling (1.16±0.18 mV vs. 0.70±0.11 mV, \( P=0.044 \)) and at baseline temperature (1.02±0.21 mV vs. 0.42±0.09 mV, \( P=0.005 \)).

**Conclusions** - Hypothermia increases both the prevalence and magnitude of ER in cardiac arrest survivors. Despite the association of ER with idiopathic VF, therapeutic hypothermia only increases ER amplitude in CAD-VF survivors.

**Key words:** repolarization, hypothermia, ventricular fibrillation, sudden cardiac death, arrhythmia, idiopathic ventricular fibrillation, early repolarization syndrome, J wave, therapeutic hypothermia
Introduction

Early repolarization (ER) is a common electrocardiographic finding, being seen in 5-13% of Europeans.\textsuperscript{1, 2} While long felt to be a benign variant, or even a marker of cardiovascular health,\textsuperscript{3} robust data now point to such changes as a marker of elevated risk of ventricular fibrillation (VF) and cardiac arrest. This is true in both otherwise healthy individuals (so-called idiopathic VF)\textsuperscript{1, 2, 5-7} and in those with coronary artery disease.\textsuperscript{8-10} Risk appears to vary markedly with the degree of ER, quantified as the magnitude of J-point elevation (JPE). Thus patients with a JPE magnitude of $>0.2$ mV are at around twice the risk of cardiac arrhythmic death as compared to those with JPE defined using a more conventional cut off of $>0.1$ mV.\textsuperscript{1} Furthermore, patients with ER who have experienced an episode of idiopathic VF have a mean JPE magnitude of around twice that seen in those without arrhythmia.\textsuperscript{11}

Early repolarization syndrome is one of a broader set of conditions, including the Brugada syndrome, collectively termed the J-wave syndromes.\textsuperscript{12} These conditions are defined by elevation of the J-point, slurring of the terminal part of the QRS complex and ST segment elevation. All appear to be attributable to a similar ionic mechanism: an enhanced net repolarizing current gives rise to a marked epicardial action potential notch and hence a J-wave.\textsuperscript{13} The increased transmural dispersion of repolarization this reflects results in an increased propensity to ventricular arrhythmia. Such J-waves are also a well established feature of hypothermia.\textsuperscript{14, 15} While similarities and differences between the various J-wave syndromes have been a topic of recent interest,\textsuperscript{16} interactions between ER and hypothermia have not previously been studied in a clinical context.

In this study we hypothesized that therapeutic hypothermia (defined as core temperature 33-36°C) would have a greater effect on the magnitude of JPE in idiopathic VF (ID-VF) than
coronary artery disease-related VF (CAD-VF) arrest survivors. We therefore used data obtained from a population of adult early survivors treated with therapeutic hypothermia following cardiac arrest to investigate the potential interactions between ER and hypothermia.

**Methods**

All survivors of sudden cardiac arrest with an initial rhythm of VF treated at the critical care unit at St. Thomas’ Hospital between 2005 and 2012 were assessed for inclusion. Data collection and analysis for this study were part of ongoing real-time data collection processes during physician-directed patient care.

*Patient Selection*

Cases were eligible for inclusion if: 1) their management included therapeutic hypothermia, 2) ECGs obtained at baseline temperature and during cooling were available and 3) sufficient data (echocardiograms, coronary angiograms, cardiac magnetic resonance imaging (MRI) studies and/or electrophysiology studies) were available to determine whether the arrest was attributable to myocardial ischemia. Cases were excluded if ventricular conduction abnormalities, other underlying arrhythmic disorders or clear confounding factors such as recent cardiothoracic surgery or cocaine use were present.

Cases were classified as ID-VF if criteria specified in guidelines were met, that is there was no structural heart disease (as indicated by an echocardiogram showing normal biventricular dimensions and function), no coronary artery disease (as indicated by a normal coronary angiogram) and no known repolarization abnormalities. Cases were classified as CAD-VF if A) there was a pre-existing diagnosis of coronary artery disease or B) significant coronary artery disease was demonstrated on an angiogram, a significant troponin rise from baseline was
observed and regional wall motion abnormalities were present either on echocardiography or MRI.

Data Collection and Analysis

ECGs were obtained close to the mid-point of the period of therapeutic hypothermia (‘cooled’ ECG), during which patients’ core temperatures fell to between 32 and 34°C, and again prior to discharge (‘baseline’ ECG). Records were digitally scanned (300dpi, 24-bit color) and read at 4x magnification by two independent observers. ECGs were assessed for the presence of ER and the magnitude of JPE, manifest as slurring or notching of the downslope of the R wave in at least two contiguous inferior (II, III, aVF) or lateral (I, aVR, aVL, V4-6) leads.3, 5 ER was assessed at typical (≥0.1mV) and high-risk (>0.2mV) amplitudes, as well as on morphological criteria alone (JPE <0.1mV). The following data were recorded for each patient: the number and location of leads displaying ER, the mean and maximum amplitudes of JPE in each lead, PR interval, QRS duration and QTc interval (derived using Bazett’s formula). For each ECG displaying ER ≥0.1mV, ST segment morphology was classified as “concave/rapidly ascending” when there was >0.1mV ST segment elevation within 100ms after the J-point and the ST segment merged gradually with the T wave, or “horizontal/descending” when the ST segment elevation was ≤0.1mV within 100ms of the J-point and continued as a flat segment until T wave onset.18, 19

Statistical analysis was performed using SPSS (SPSS Inc., Chicago, IL). Categorical variables were compared using a two-tailed Fisher’s Exact test or an exact McNemar test for paired data as appropriate. Continuous variables were compared using a two-tailed Student’s T-test (paired or unpaired as appropriate) and reported as means ± standard errors. Owing to the nature of the study, no adjustments were made for multiple comparisons. All tests were two-sided, and P values <0.05 were considered statistically significant.
Results

Of the 89 cases reviewed, 43 met the inclusion criteria. Reasons for exclusion were: paired ECGs not available (n=21), abnormal baseline ECG (n=17: left bundle branch block, right bundle branch block, paced rhythm, Brugada syndrome), insufficient clinical details to confirm VF etiology (n=4), other confounding factors (n=4: dilated cardiomyopathy, cardiothoracic surgery, drug use, myocarditis). Baseline characteristics are shown in Table 1.

Effect of cooling on ER in the overall cohort

In the cohort overall, the ER pattern was present in a significantly greater proportion of patients during cooling than at baseline (32/43 during cooling vs. 22/43 at baseline, P=0.024). The mean J-point elevation (JPE) amplitude for the inferolateral ECG leads was significantly greater during cooling than at baseline (0.78±0.10 mV during cooling vs. 0.56±0.09 mV at baseline, P=0.038). The maximum JPE amplitude seen in any single lead of each ECG was greater during cooling than at baseline (1.15±0.15 mV during cooling vs. 0.68±0.11 mV at baseline, P=0.002). Figure 1 illustrates typical ID-VF and CAD-VF cases in which ER morphology was augmented by cooling.

Temperature-dependent prevalence of ER in ID-VF and CAD-VF arrest survivors

When defining the presence of ER as any JPE (manifest as terminal QRS notching or slurring) with or without ST segment elevation, ER was more common among survivors of ID-VF as compared to CAD-VF both at baseline (80.0% vs. 42.4%) and during cooling (100% vs. 66.7%), but the difference reached statistical significance only during cooling (Figures 2A, 2B). In contrast, JPE of a magnitude ≥0.1mV was present significantly more frequently in the ID-VF as compared to the CAD-VF group at baseline (70.0% vs. 27.3%, Figure 2C), but the significance threshold was not met during cooling (80.0% vs. 42.4%, Figure 2D). Finally, there was no
significant difference in the prevalence of ER between ID-VF and CAD-VF at baseline (20.0% vs. 3.0%) or during cooling (40.0% vs. 15.2%) when defining ER as a JPE magnitude >0.2mV (Figure 2E and 2F). Cooling was not associated with the *de novo* development of ER in either those with ID-VF (8/10 vs. 10/10, $P=0.480$) or CAD-VF (14/33 vs. 22/33, $P=0.061$) arrest.

**ST segment morphology in idiopathic VF and CAD-VF arrest survivors with ER**

Considering only the cases with JPE ≥0.1mV (Figure 3), a greater proportion of the ID-VF group than the CAD-VF group (71.4% vs. 33.3%) displayed horizontal/downsloping ST segments at baseline temperature. Similarly, in the CAD-VF group horizontal/downsloping ST segments were more prevalent during hypothermia than at baseline temperature (53.8% vs. 33.3%).

Neither of these differences reached statistical significance, possibly reflecting the small sample size. In contrast, there was no difference between the ID-VF and CAD-VF groups in the proportion of patients with horizontal/downsloping ST segments during hypothermia, or within the ID-VF group on cooling.

**Temperature-dependent amplitude of JPE in idiopathic VF and CAD-VF arrest survivors**

Mean and maximum JPE amplitude were significantly greater amongst the ID-VF group, as compared to the CAD-VF group, both at baseline and during cooling (Table 2). Maximum, but not mean, JPE magnitude increased significantly on cooling in the ID-VF group (Figure 4A). Both mean and maximum JPE magnitude increased on cooling in the CAD-VF group (Figure 4B). There was no difference in the proportion of patients in the ID-VF group as compared to the CAD-VF group in whom JPE magnitude increased on cooling (6/10 vs. 16/33, $P=0.72$ for mean and 9/10 vs. 18/33, $P=0.063$ for maximum). Similarly, there was no difference in the magnitude of the increase in either mean or maximum JPE on cooling between the groups (ΔJPE, Table 2).
QRS duration and QTc intervals

QRS duration and QTc interval increased significantly on cooling in both the ID-VF and CAD-VF groups (Table 3). There were no differences in QRS duration or QTc interval between ID-VF and CAD-VF groups either during cooling or at baseline (all \( P > 0.20 \)).

Discussion

The main findings of this study are as follows. 1) Overall, ER was more common and of greater amplitude during cooling than at baseline. 2) ER amplitude was significantly greater in survivors of ID-VF arrest than survivors of CAD-VF arrest at all temperatures. 3) Whilst the prevalence of ER was not significantly increased in either group on cooling, mean JPE was increased by cooling only in survivors of CAD-VF arrest.

In recent years robust population-based studies have established an association between the early repolarization (ER) pattern and ventricular fibrillation (VF), whether idiopathic in nature or secondary to ischemia.\(^{5, 6, 8}\) At the same time, the tendency for hypothermia to result in similar electrocardiographic changes is well established.\(^{14, 15}\) Furthermore, in isolated cases therapeutic hypothermia itself has been described as potentially pro-arrhythmic.\(^{20}\) The now-common use of therapeutic hypothermia following cardiac arrest presents an important opportunity to examine in a systematic manner the impact of cooling on ER morphology in patients who have suffered either ID-VF or CAD-VF.\(^{21}\) This retrospective cohort study therefore assessed the impact of therapeutic hypothermia on the prevalence, morphology and magnitude of ER in all ventricular fibrillation patients receiving therapeutic hypothermia at our critical care unit over a 7-year period.

A number of differences exist between this cohort and previously published descriptions of ER. Firstly, the overall frequency of ER in our study population is rather higher than that
reported elsewhere. At baseline temperature 51% showed morphological ER. Even limiting to those patients with JPE ≥0.1 mV, a widely accepted definition,5 the overall prevalence of ER in this cohort at baseline was still 37.2%. This prevalence is substantially higher than that seen in the European population overall (5 – 13%)1,2 and also markedly higher than that described in a recent study of patients who had suffered cardiac arrests attributable to a variety of causes by Rolfast et al (3%).22 The former likely reflects the high-risk nature of the patients in our study population and the latter differences in underlying causes for VF between our cohort and that reported in Rolfast et al's study. During cooling 74% of our study population showed the ER pattern (51.2% at the 0.1 mV JPE threshold). The prevalence of ER is well established to vary with core temperature23: case series of patients with accidental hypothermia suggest that, given the relatively mild degree of hypothermia used in a therapeutic setting, the prevalence of ER would be expected to be close to zero.23,24 Rolfast et al report a 30% prevalence in their study population during cooling.

Secondly, similarities and differences emerge between patients whose arrest was attributable to ID-VF, as compared to CAD-VF. Perhaps unsurprisingly, patients who suffered an episode of ID-VF were younger and apparently healthier than those who experienced CAD-VF. There were no differences in QRS duration or QTc between groups, though both parameters increased significantly on cooling. The latter finding has previously been established,24 with increased QTc having been described in the context of therapeutic hypothermia.22 The ER pattern was more common among survivors of ID-VF, as compared to CAD-VF, during cooling, though not at baseline temperature. Furthermore, JPE was of a greater magnitude in patients with idiopathic VF at both temperatures studied. Large magnitude JPE was seen more frequently among patients with ID-VF. This finding reached significance at baseline temperature but
became less marked with the compounding influence of cooling. These observations are in keeping with a previous suggestion that the presence of ER in patients resuscitated following cardiac arrest is suggestive of a non-ischemic etiology and that large-magnitude JPE is associated with a particularly high risk of idiopathic VF.\textsuperscript{1,25}

The present study was intended to discern differences in response to cooling between patients suffering ID-VF and CAD-VF. In this regard we note that cooling-related increases in J-point elevation were significant in the CAD-VF group but not the ID-VF group. It is tempting to speculate that this surprising finding is attributable to differences in the specific ion channel disruptions giving rise to ischemic or idiopathic ER. On one hand, ischemic ER is most likely attributable to local activation of ATP-sensitive K\textsuperscript{+} currents by ischemic mediators.\textsuperscript{12,26} Hypothermia is generally also thought to produce ER as a result of activation of K\textsuperscript{+} currents, most importantly the transient outward current.\textsuperscript{27} On the other, idiopathic ER may arise as a result of dysfunction of one of several depolarizing or repolarizing ion currents and hence interactions with hypothermia may be less predictable.\textsuperscript{16}

\textbf{Limitations}

While we report the association between early repolarization and both idiopathic and CAD-related VF, we cannot be clear from these data as to the mechanisms underlying arrhythmia in either setting. Traditionally ER has been associated with arrhythmia initiated by phase II re-entry.\textsuperscript{13,28} It is certainly possible, however, that patients in the CAD-VF group who had previously experienced a myocardial infarction (one third of this group) might in fact have suffered an episode of scar-related VT which subsequently deteriorated into VF. Patients in the idiopathic VF group were younger than those in the CAD-VF group. Since the prevalence of early repolarization decreases with age,\textsuperscript{29,30} it is possible that this confounding factor could have
contributed to some of the differences observed between the ID-VF and CAD-VF groups.

Finally, the baseline (normothermic) ECGs were obtained following rewarming after the arrest. Whether the same results would have been seen had pre-morbid baseline ECGs been available is not known.

Conclusions

Early repolarization is more strongly associated with idiopathic VF than with coronary artery disease related VF. Hypothermia increases both the prevalence and magnitude of early repolarization in cardiac arrest survivors: however, the mean amplitude of J-point elevation is increased in survivors of coronary artery disease related VF, but not of idiopathic VF.

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Conflict of Interest Disclosures: None.

References:


**Table 1.** Demographic data

<table>
<thead>
<tr>
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<th>ID-VF (n = 10)</th>
<th>CAD-VF (n = 33)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>42.9 ± 4.7</td>
<td>61.5 ± 2.3</td>
<td>0.0004 *</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>8 (80%)</td>
<td>31 (94%)</td>
<td>0.23</td>
</tr>
<tr>
<td>Females</td>
<td>2 (20%)</td>
<td>2 (6%)</td>
<td>0.23</td>
</tr>
<tr>
<td><strong>Co-morbidities</strong></td>
<td></td>
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<tr>
<td>Hypertension</td>
<td>1 (10%)</td>
<td>14 (42%)</td>
<td>0.13</td>
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<tr>
<td>Hypercholesterolemia</td>
<td>0 (0%)</td>
<td>11 (33%)</td>
<td>0.043 *</td>
</tr>
<tr>
<td>Pre-existing coronary artery disease (previous MI or stent)</td>
<td>0 (0%)</td>
<td>11 (33%)</td>
<td>0.043 *</td>
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<tr>
<td>Diabetes mellitus</td>
<td>0 (0%)</td>
<td>5 (15%)</td>
<td>0.320</td>
</tr>
<tr>
<td>Family history of ischemic heart disease</td>
<td>0 (0%)</td>
<td>2 (6%)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

VF indicates ventricular fibrillation; MI, myocardial infarction; ID-VF, idiopathic VF; CAD-VF, coronary artery disease-related VF; * indicates statistical significance.
Table 2. Magnitude of J-point elevation

<table>
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<tr>
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<th>Mean J-point elevation (mV)</th>
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<tr>
<td></td>
<td>ID-VF</td>
<td>CAD-VF</td>
<td>P value</td>
<td></td>
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<tr>
<td>Baseline</td>
<td>1.02 ± 0.21</td>
<td>0.42 ± 0.09</td>
<td>0.005 *</td>
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<tr>
<td>Mean</td>
<td>1.16 ± 0.18</td>
<td>0.70 ± 0.11</td>
<td>0.044 *</td>
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<tr>
<td>Δ JPE</td>
<td>1.38 ± 5.26</td>
<td>2.79 ± 7.33</td>
<td>0.577</td>
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<tr>
<td>Baseline</td>
<td>1.18 ± 0.24</td>
<td>0.53 ± 0.12</td>
<td>0.015 *</td>
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<tr>
<td>Maximum</td>
<td>1.69 ± 0.29</td>
<td>0.99 ± 0.16</td>
<td>0.042 *</td>
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<tr>
<td>Δ JPE</td>
<td>5.17 ± 6.51</td>
<td>4.57 ± 10.05</td>
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</table>

VF indicates ventricular fibrillation; ID-VF, idiopathic VF; CAD-VF, coronary artery disease-related VF; * indicates statistical significance.

Table 3. Other ECG parameters

<table>
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<th>Duration (ms)</th>
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<th></th>
<th></th>
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</thead>
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<tr>
<td></td>
<td>Baseline</td>
<td>Cooled</td>
<td>P value</td>
<td></td>
</tr>
<tr>
<td>QRS duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ID-VF</td>
<td>89.1 ± 2.3</td>
<td>97.9 ± 3.4</td>
<td>0.002 *</td>
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<tr>
<td>CAD-VF</td>
<td>94.3 ± 2.2</td>
<td>100.5 ± 2.5</td>
<td>0.016 *</td>
<td></td>
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<tr>
<td>QTo interval</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ID-VF</td>
<td>380.6 ± 37.9</td>
<td>493.2 ± 16.5</td>
<td>0.048 *</td>
<td></td>
</tr>
<tr>
<td>CAD-VF</td>
<td>420.7 ± 4.6</td>
<td>472.6 ± 7.8</td>
<td>&lt;0.001*</td>
<td></td>
</tr>
</tbody>
</table>

VF indicates ventricular fibrillation; ID-VF, idiopathic VF; CAD-VF, coronary artery disease-related VF; * indicates statistical significance.

Figure Legends:

Figure 1. Early repolarization developing during hypothermia. The early repolarisation pattern is minimal during normothermia (temperature 37 °C, leftmost ‘baseline’ ECGs of each panel) but
substantially augmented during hypothermia (33 °C, rightmost ‘cooled’ ECGs of each panel). (A) In a single ID-VF patient, terminal QRS slurring combined with ST segment elevation is seen in the inferior leads and terminal QRS notching is seen in the lateral leads during hypothermia. (B) In a single CAD-VF patient, terminal QRS slurring develops in the lateral leads during hypothermia. Baseline/normothermic ECGs were obtained following rewarming after the period of therapeutic hypothermia.

**Figure 2.** Prevalence of early repolarisation in the four subgroups. Number of cases displaying early repolarisation (ER) at normothermia (left column) and when cooled (right column) using thresholds of all ER (A and B), ER≥0.1mV (C and D) and ER≥0.2mV (E and F). Baseline/normothermic ECGs were obtained following rewarming after the period of therapeutic hypothermia. VF indicates ventricular fibrillation; ID-VF, idiopathic VF; CAD-VF, coronary artery disease-related VF; * indicates statistical significance.

**Figure 3.** ST-segment morphology in idiopathic and coronary artery disease related ventricular fibrillation survivors with early-repolarization (J-wave amplitude ≥0.1mV). Proportion of cases with horizontal/downsloping (high risk) vs. concave/upsloping ST segments (benign) in each group at normothermia and while cooled are shown. ST segment morphology was defined according to criteria in Ref 19. Baseline/normothermic ECGs were obtained following rewarming after the period of therapeutic hypothermia. VF indicates ventricular fibrillation; ID-VF, idiopathic VF; CAD-VF, coronary artery disease-related VF.

**Figure 4.** J-point elevation among patients with idiopathic and coronary artery disease related
ventricular fibrillation at baseline temperature and during hypothermia. Mean (circles) and maximum (triangles) J-point elevation at normothermia and during cooling in (A) ID-VF and (B) CAD-VF cases are shown as individual data points. Baseline/normothermic ECGs were obtained following rewarming after the period of therapeutic hypothermia. VF indicates ventricular fibrillation; ID-VF, idiopathic VF; CAD-VF, coronary artery disease-related VF; * indicates statistical significance.
A. ID-VF Group

Baseline  Cooled  Baseline  Cooled
B. CAD-VF Group

Baseline

Cooled

Baseline

Cooled
Presence of ER

Baseline

Cooled

% of cases

p = 0.069

p = 0.043*

% of cases

p = 0.024*

p = 0.069

% of cases

p = 0.130

p = 0.177

% of cases

ID-VF

CAD-VF

ID-VF

CAD-VF

ID-VF

CAD-VF

ID-VF

CAD-VF
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