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ORIGINAL SCIENTIFIC PAPER

Repolarisation abnormalities and outcomes among patients with cardiac arrest

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Aims Changes in ventricular repolarisation, observed as QTc prolongation, are frequently observed following cardiac arrest. The T-peak to T-end (TpTe) interval represents a period of increased susceptibility to ventricular arrhythmia. We posit that TpTe prolongation may be associated with adverse clinical outcomes in patients resuscitated from cardiac arrest.

Methods and results We included patients aged ≥18 years with both out-of-hospital and in-hospital cardiac arrest following return of spontaneous circulation (ROSC) who had an electrocardiogram (ECG) obtained within 24 h following ROSC. The first ECG obtained was evaluated to determine the QTc and TpTe intervals. Hierarchical logistic regression was used to evaluate the association between prolongation of the QTc and TpTe intervals and clinical outcomes (in-hospital mortality and favourable neurologic outcome at hospital discharge). We included 443 patients, with a median age of 61 years (IQR: 50–72 years), 60.5% male, 65.7% OHCA, and 29.8% with initial shockable rhythm. Overall, 310 patients had QTc prolongation (70.0%), and 284 had TpTe prolongation (64.1%). Patients with TpTe prolongation had a greater incidence of initial shockable rhythm (35.6% vs. 19.5%, $P < 0.001$) and higher initial lactate (8.6 vs. 7.4 mmol/L, $P = 0.03$). QTc prolongation was not associated with in-hospital mortality [odds ratio (OR):1.27, 95% confidence interval (CI): 0.75–2.14, $P = 0.37$] or favourable neurologic outcome (OR: 0.88, 95% CI: 0.50–1.54, $P = 0.65$). TpTe prolongation was independently associated with in-hospital mortality (OR: 1.69, 95% CI: 1.01–2.85, $P = 0.05$) but not favourable neurologic outcome (OR: 0.78, 95% CI: 0.45–1.37, $P = 0.39$).

Conclusion TpTe interval prolongation, but not QTc interval prolongation, was associated with increased in-hospital mortality in patients resuscitated from cardiac arrest.

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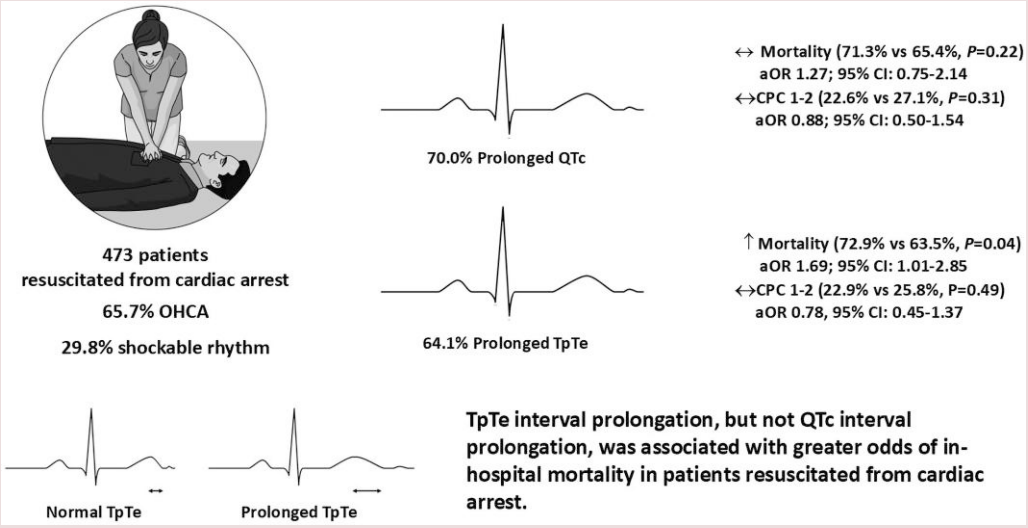
Graphical Abstract



aOR, adjusted odds ratio; CI, confidence interval; CPC, Cerebral Performance Category; OHCA, out-of-hospital cardiac arrest. Figure created with Mind the Graph.

Keywords Cardiac Arrest • Electrocardiogram • QT prolongation • TpTe interval

Introduction

Both out-of-hospital cardiac arrest (OHCA) and in-hospital cardiac arrest (IHCA) are associated with significant morbidity and mortality.[1–3] Despite advancements in post-resuscitation care, early risk stratification remains challenging.[4–7] Although the primary cause of death is anoxic brain injury, arrythmias represent up to 11% of deaths.[8,9]

Changes in ventricular repolarisation, observed as QTc prolongation, are frequently observed in patients resuscitated from cardiac arrest.[10–16] QTc prolongation in these patients is thought to be multifactorial, with contributions from myocardial injury, hypothermic therapies, electrolyte derangements, and the administration of QTc-prolonging medications.[16] Unique components of ventricular repolarisation may be differentially affected following cardiac arrest, with prior studies demonstrating QTc prolongation without significant change in the T-peak to T-end (TpTe) interval.[11,13] The TpTe interval represents transmural dispersion of repolarisation in the left ventricle and is a period of increased susceptibility for ventricular arrhythmia.[17] TpTe interval prolongation in patients resuscitated from OHCA was not found to be associated with core body temperature but was associated with risk for ventricular arrhythmia in the Targeted Temperature Management (TTM) trial.[11] However, this study did not report the association between TpTe and mortality.

The goal of this study was to evaluate the association between prolongation of the QTc and TpTe intervals and clinical outcomes among patients resuscitated from cardiac arrest. We posit that because the TpTe interval represents a period of increased susceptibility to ventricular arrhythmia, that TpTe prolongation may be associated with adverse outcomes in patients resuscitated from cardiac arrest.

Methods

Study population

The present study was a retrospective analysis of patients enrolled in the Multimodal Outcome CHAracterisation in Comatose Cardiac Arrest Patients (MOCHA) Registry at a single tertiary care centre between 2011 and 2022. We included patients aged ≥18 years with OHCA or IHCA who achieved sustained return of spontaneous circulation (ROSC) (≥20 min after cardiopulmonary resuscitation), and had an electrocardiogram (ECG) obtained within the first 24 h following ROSC. The present study was approved by the Yale University Institutional Review Board.

ECG analysis

We evaluated the first 12-lead ECG obtained following ROSC. Each ECG was manually interpreted by two trained assessors (CS and SB). A third reviewer adjudicated cases with interrater disagreement (PEM), which occurred in 38 ECGs (8.6%). Each assessor was blinded to the others' assessments. The QT interval, defined as the interval from the onset of the QRS complex to the end of the T wave, representing the time from earliest electrocardiographic evidence of ventricular depolarisation to the earliest electrocardiographic evidence of ventricular repolarisation,[18] was measured using the tangent method.[19,20] The corrected QT interval (QTc) was calculated using Bazetts formula.[18] QTc prolongation was defined as a corrected QT interval of ≥ 460 ms in females or ≥ 450 ms in males.[18] The QTpeak interval was measured from the onset of the QRS complex to the peak or nadir point of the T wave. The TpTe interval was calculated as the difference between the QT interval and the QTpeak interval. TpTe prolongation was defined as a TpTe interval of ≥ 90 ms.[21] All measurements were made in lead II. For patients with atrial fibrillation, intervals were averaged over four consecutive beats, and the mean heart rate was used for heart rate correction.

Variables of interest

Demographic variables included age, sex, race and ethnicity. Medical comorbidities included coronary artery disease (CAD), heart failure, chronic kidney disease (CKD), hypertension, and diabetes. Arrest characteristics included arrest location (OHCA or IHCA), initial non-perfusing rhythm (shockable or non-shockable), whether the arrest was witnessed, whether bystander cardiopulmonary resuscitation (CPR) was administered, and the Pittsburgh Cardiac Arrest Category (PCAC), a validated illness severity tool for predicting survival and good functional outcome after cardiac arrest.[22] Laboratory data included initial lactate and pH, obtained immediately

Table 1 Patient demographics and clinical characteristics for patients with cardiac arrest stratified by QTC and TpTe

Patient characteristics	Overall ($n = 443$)	QTc prolongation ($n = 310$)	No QTc prolongation ($n = 133$)	*P*-value	TpTe prolongation ($n = 284$)	No TpTe prolongation ($n = 159$)	*P*-value
Demographics							
Age (years)	61 (50–72)	63 (52–73)	59 (46–68)	0.01	63 (53.5–75)	59 (44–67)	<0.001
Sex							
Male	268 (60.5)	184 (59.4)	84 (63.2)	0.45	172 (60.6)	96 (60.4)	0.97
Female	175 (39.5)	126 (40.7)	49 (36.8)		112 (39.4)	63 (39.6)	
Race and ethnicity							
White	276 (62.3)	190 (61.3)	86 (64.7)	0.09	172 (60.6)	104 (65.4)	0.15
Black	106 (23.9)	83 (26.8)	23 (17.3)		75 (26.4)	31 (19.5)	
Hispanic	50 (11.3)	31 (10.0)	19 (14.3)		28 (9.9)	22 (13.8)	
Other	11 (2.5)	6 (1.9)	5 (3.8)		9 (3.2)	2 (1.3)	
Medical comorbidities							
CAD	115 (26.0)	89 (28.7)	26 (19.6)	0.04	87 (30.6)	28 (17.6)	0.003
Heart failure	79 (17.8)	60 (19.4)	19 (14.3)	0.20	56 (19.7)	23 (14.5)	0.17
CKD	75 (16.9)	60 (19.4)	15 (11.4)	0.04	51 (18.0)	24 (15.1)	0.44
Hypertension	279 (63.0)	204 (65.8)	75 (56.4)	0.06	189 (66.6)	90 (56.6)	0.04
Diabetes	141 (31.8)	102 (32.9)	39 (29.4)	0.46	91 (32.0)	50 (31.5)	0.90
Arrest characteristics							
OHCA	291 (65.7)	211 (68.1)	80 (60.2)	0.11	190 (66.9)	101 (63.5)	0.47
Initial shockable rhythm	132 (29.8)	94 (30.3)	38 (28.6)	0.71	101 (35.6)	31 (19.5)	<0.001
Witnessed arrest	327 (73.8)	234 (75.5)	93 (69.9)	0.22	206 (72.5)	121 (76.1)	0.41
Bystander CPR	234 (52.8)	162 (52.3)	72 (54.1)	0.72	145 (51.1)	89 (56.0)	0.32
PCAC				0.69			0.67
1	52 (12.6)	34 (11.8)	18 (14.3)		33 (12.4)	19 (12.9)	
2	31 (7.5)	22 (7.6)	9 (7.1)		19 (7.1)	12 (8.2)	
3	75 (18.1)	56 (19.4)	19 (15.1)		53 (19.9)	22 (15.0)	
4	256 (61.8)	176 (61.1)	80 (63.5)		162 (60.7)	94 (64.0)	
Laboratory data							
Initial lactate (mmol/L)	8.2 (5.0–11.4)	8.2 (4.9–11.2)	8.4 (5.3–11.7)	0.45	8.6 (5.4–12)	7.4 (4.5–10.5)	0.03
Initial pH	7.28 (7.16–7.37)	7.28 (7.16–7.38)	7.28 (7.15–7.37)	0.50	7.27 (7.13–7.36)	7.30 (7.21–7.38)	0.01
Procedures							
LHC	49 (11.1)	33 (10.7)	16 (12.0)	0.74	38 (13.4)	11 (6.9)	0.05
PCI	35 (7.9)	25 (8.1)	10 (7.5)	0.85	27 (9.5)	8 (5.0)	0.09
TTM	258 (58.2)	182 (58.7)	56 (57.1)	0.76	163 (57.4)	95 (59.8)	0.63

CPR, cardiopulmonary resuscitation; LHC, left heart catheterisation; OHCA, out-of-hospital cardiac arrest; PCAC, Pittsburgh Cardiac Arrest Category; PCI, percutaneous coronary intervention; TTM, targeted temperature management (33–36°C at the enrolling institution).

following ROSC. We additionally included procedures during admission, such as left heart catheterisation (LHC), percutaneous coronary intervention (PCI) and TTM.

Outcomes

The primary outcome of interest was in-hospital mortality. We also assessed for favourable neurologic outcome, defined as a cerebral performance category score of 1–2 at the time of hospital discharge.[23] We report the proportion of patients who underwent withdrawal of life-sustaining therapies (WLST), and the median time to WLST. We additionally report recurrent cardiac arrest and antiarrhythmic drug use within the first 5 days of admission.

Statistical analysis

Baseline characteristics were reported for the entire cohort and stratified by QTc and TpTe intervals. Continuous variables were reported as medians and interquartile ranges (IQR), and categorical variables were reported as frequencies and percentages. The Wilcoxon rank-sum test and chi-squared

Table 2 Unadjusted clinical outcomes for patients with cardiac arrest stratified by QTC and TpTe

Outcome	Overall ($n = 443$)	QTc prolongation ($n = 310$)	No QTc prolongation ($n = 133$)	*P*-value	TpTe prolongation ($n = 284$)	No TpTe prolongation ($n = 159$)	*P*-value
In-hospital mortality	308 (69.5)	221 (71.3)	87 (65.4)	0.22	207 (72.9)	101 (63.5)	0.04
WLST	259 (58.5)	185 (59.7)	74 (55.6)	0.43	178 (62.7)	81 (50.9)	0.02
Time to WLST (days)	4 (2–8)	4 (2–9)	4 (2–7)	0.40	4 (2–9)	5 (2–8)	0.78
WLST among pts who died	256 (83.1)	183 (82.8)	73 (83.9)	0.82	176 (85.0)	80 (79.2)	0.20
Favourable neurologic outcome	106 (23.9)	70 (22.6)	36 (27.1)	0.31	65 (22.9)	41 (25.8)	0.49
Recurrent cardiac arrest	49 (11.1)	30 (9.7)	19 (14.3)	0.16	29 (10.2)	20 (12.6)	0.45
Recurrent cardiac arrest with initial shockable rhythm	7 (1.6)	4 (1.3)	3 (2.3)	0.46	6 (2.1)	1 (0.6)	0.23
Antiarrhythmic drug administration	118 (26.6)	86 (27.7)	32 (24.1)	0.42	89 (31.3)	29 (18.2)	0.003

WLST, withdrawal of life-sustaining therapy.

test were used for continuous and categorical variables, respectively. Hierarchical logistic regression models were constructed to evaluate the association of QTc prolongation and TpTe prolongation with in-hospital mortality, including an unadjusted model (Model 1), and additionally adjusting for patient demographics (age, sex, race, and ethnicity) (Model 2), medical comorbidities (heart failure, CKD, diabetes) (Model 3), and arrest characteristics (arrest location, initial rhythm, witnessed status, bystander CPR, lactate) (Model 4). Sensitivity analyses were conducted for patients with initial shockable rhythm and OHCA. Covariates were selected based on clinical significance. Models were assessed for multicollinearity using the variance inflation factor. Interobserver variability was assessed using the Intra-class correlation coefficient (ICC) and Bland-Altman plots. All analyses were performed on STATA 16.0 (Stata Corp, College Station, TX) with statistical significance considered at a two-tailed $P < 0.05$.

Results

Of 471 patients enrolled in the MOCHA registry at our centre, we included 443 (94.1%) patients with an ECG obtained within 24 h. The median time to ECG acquisition was 52 min (IQR: 30–139 min). Interobserver variability, measured as median difference and IQR between assessors, was −0.2 ms (IQR: −4.3 to 4.1 ms; ICC: 0.98, $P < 0.001$) for the QT interval and 1.2 ms (IQR: −5.3 to 5.8 ms; ICC: 0.91, $P < 0.001$) for the QTpeak interval. Similarly, Bland-Altman plots showed good agreement (see Supplementary material online, *Figure S1*).

Baseline characteristics for the entire cohort and stratified by QTc prolongation and TpTe prolongation are shown in *Table 1*. Patients had a median age of 61 years (IQR: 50–72 years), and 60.5% were male. The median QTc was 483 ms (IQR: 446–530 ms), and the median TpTe was 100 ms (IQR: 79–124 ms). Overall, 310 (70.0%) patients had a QTc prolongation and 284 (64.1%) patients had a TpTe prolongation. Patients with QTc prolongation were older (63 vs. 59 years, $P = 0.01$), and had a higher proportion of CAD (28.7% vs. 19.6%, $P = 0.04$) and CKD (19.4% vs. 11.4%). Arrest characteristics, including arrest location, initial rhythm, the proportion of witnessed arrests, the proportion of arrests with bystander CPR, and PCAC score did not differ between those with and without QTc prolongation (all, $P > 0.05$). Similarly, the rate of LHC, PCI, and TTM did not differ between those with and without QTc prolongation (all, $P > 0.05$).

Patients with TpTe prolongation were older (63 vs. 59 years, $P < 0.001$), and had a higher proportion of CAD (30.6% vs. 17.6%) and hypertension (66.6% vs. 56.6%, $P = 0.04$). Patients with TpTe prolongation had a greater incidence of initial shockable rhythm (35.6% vs. 19.5%, $P < 0.001$) and higher initial lactate (8.6 vs. 7.4 mmol/L, $P = 0.03$). The PCAC score, and proportion undergoing LHC, PCI, and TTM were not statistically different (all, $P > 0.05$).

Overall, 308 (69.5%) patients died prior to hospital discharge (*Table 2*). WLST occurred in 259 (58.5%) patients, and the median time to WLST was 4 days (IQR: 2–8 days). The proportion of patients undergoing WLST who died did not differ either by QTc group or TpTe group. In-hospital mortality did not significantly differ between those with and without QTc prolongation (71.3% vs. 65.4%, $P = 0.22$). In multivariable analysis, QTc prolongation was not associated with in-hospital mortality after sequentially adjusting for demographic characteristics, medical comorbidities, and arrest characteristics (Model 4: OR 1.27; 95% CI: 0.75–2.14, $P = 0.37$) (*Figure 1A*). Similarly, a favourable neurologic outcome occurred in 106 (23.9%) patients, and did not significantly differ between those with and without QTc prolongation (22.6% vs. 27.1%, $P = 0.31$), including after multivariable analysis (OR 0.88; 95% CI: 0.50–1.54, $P = 0.65$) (*Figure 1B*). Recurrent cardiac arrest (9.7% vs. 14.3%, $P = 0.16$), recurrent cardiac arrest with initial shockable rhythm (1.3% vs. 2.3%, $P = 0.46$), and antiarrhythmic drug administration (27.7% vs. 24.1%, $P = 0.42$) did not differ between groups.

In contrast, in-hospital mortality was higher among patients with TpTe prolongation (72.9% vs. 63.5%, $P = 0.04$). The association between TpTe prolongation and in-hospital mortality persisted in our fully adjusted model (OR 1.69; 95% CI: 1.01–2.85, $P = 0.05$) (*Figure 2A*). The proportion of patients with a favourable neurologic outcome did not significantly differ between those with and without TpTe prolongation (22.9% vs. 25.8%, $P = 0.49$), including after multivariable analysis (OR 0.78, 95% CI: 0.45–1.37, $P = 0.39$) (*Figure 2B*). While recurrent cardiac arrest did not differ between groups (10.2% vs. 12.6%, $P = 0.45$), recurrent cardiac arrest with initial shockable rhythm was numerically higher amongst patients with TpTe prolongation [6 (2.1%) vs. 1 (0.6%), $P = 0.23$]. Antiarrhythmic drug administration was higher among patients with TpTe prolongation (31.3% vs. 18.2%, $P = 0.003$).

In sensitivity analysis including only patients with a shockable rhythm, TpTe prolongation was not significantly associated with mortality (OR 0.87; 95% CI: 0.33–2.32, $P = 0.78$) or favourable neurologic outcome (OR 1.00; 95% CI: 0.37–2.68, $P = 0.99$). Similarly, in this subgroup, QTc prolongation was not associated with mortality (OR 0.88; 95% CI: 0.35–2.18, $P = 0.78$) or favourable neurologic outcome (OR 0.92;

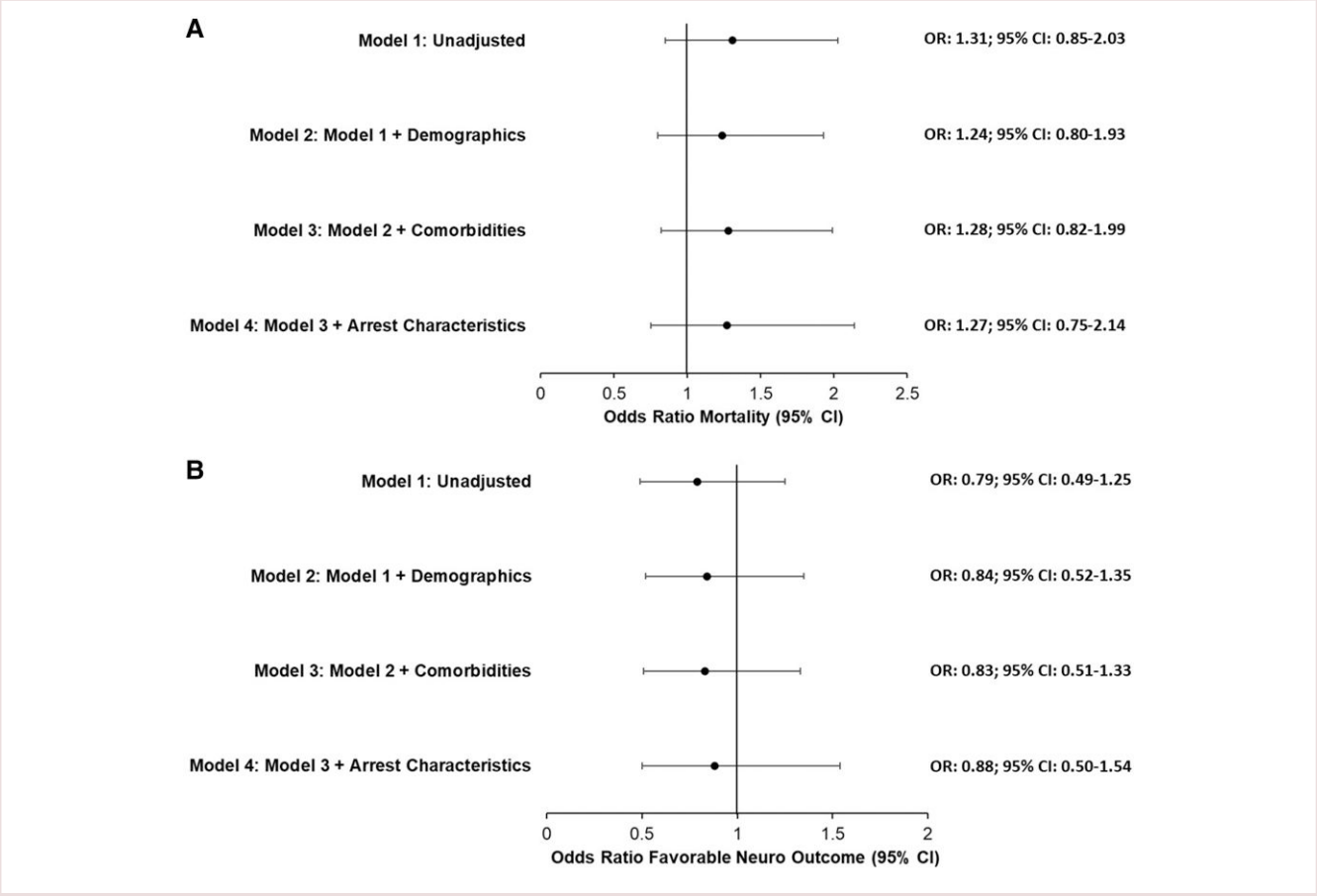


Figure 1 Odds ratio and 95% confidence intervals of the association between QTc prolongation and (*A*) in-hospital mortality and (*B*) favourable neurologic outcome. In-hospital mortality occurred in 71.3% (221/310) patients with QTc prolongation and 65.4% (87/133) patients without QTc prolongation. Favourable neurologic outcome occurred in 22.6% (70/310) patients with QTc prolongation and 27.1% (36/133) without QTc prolongation.

95% CI: 0.37–2.30, $P = 0.85$). In the subgroup of patients with OHCA, TpTe prolongation was not associated with greater odds of mortality (OR 1.60; 95% CI: 0.77–3.32, $P = 0.20$) or favourable neurologic outcome (OR 0.75; 95% CI: 0.33–1.66, $P = 0.47$). QTc prolongation was not significantly associated with mortality (OR 0.76; 95% CI: 0.35–1.62, $P = 0.47$) or favourable neurologic outcome (OR 1.62; 95% CI: 0.69–3.79, $P = 0.27$) in this subgroup.

Discussion

In this retrospective, single-centre study of patients resuscitated from cardiac arrest, we report several important findings. First, we found that repolarisation abnormalities are very common following cardiac arrest, occurring in approximately two-thirds of all patients. Second, we found unique patients characteristics between repolarisation groups, specifically a higher proportion of patients with a shockable rhythm for those with TpTe prolongation. Third, TpTe prolongation but not QTc prolongation was independently associated with in-hospital mortality, though neither were associated with neurologic outcomes or WLST. While the association between TpTe prolongation and in-hospital mortality did not persist in the subgroups, including patients with initial shockable rhythm or OHCA, these sensitivity analyses were limited by small sample size and should be considered exploratory. Overall, we found that repolarisation prolongation is common in patients post-arrest achieving ROSC and that those with TpTe prolongation may represent a patient population at increased risk of short-term mortality.

TpTe prolongation has previously been described as a risk factor for ventricular arrhythmia and death in several patient populations, including the general population,[17] patients with heart failure with reduced ejection fraction,[24] acute myocardial infarction,[25] and hypertrophic cardiomyopathy.[26] In particular, a post-hoc analysis from the TTM trial found that TpTe prolongation was associated with increased risk of ventricular arrhythmia in comatose patients resuscitated from OHCA.[11] However, the association between TpTe and clinical outcomes in patients with cardiac arrest is unclear. In the present study, we provide additional evidence that TpTe prolongation is associated with adverse outcomes, and may be a useful marker of increased risk.

We found that QTc prolongation was not associated with mortality or neurologic outcome. Prior studies have similarly shown that QTc prolongation in patients resuscitated from cardiac arrest was not associated with mortality or ventricular arrhythmia.[10,11,14] Various reasons, including lower temperature targets during TTM may contribute to QTc prolongation via preferential prolongation of early ventricular repolarisation.[11] In contrast TpTe does not appear to be affected by targeted body temperature.[11] As maintenance of normothermia during the post-arrest period is adopted, QTc prolongation may be less significant.[27]

Risk stratification among patients with cardiac arrest remains challenging in contemporary critical care practice.[5–7] As brain injury is the

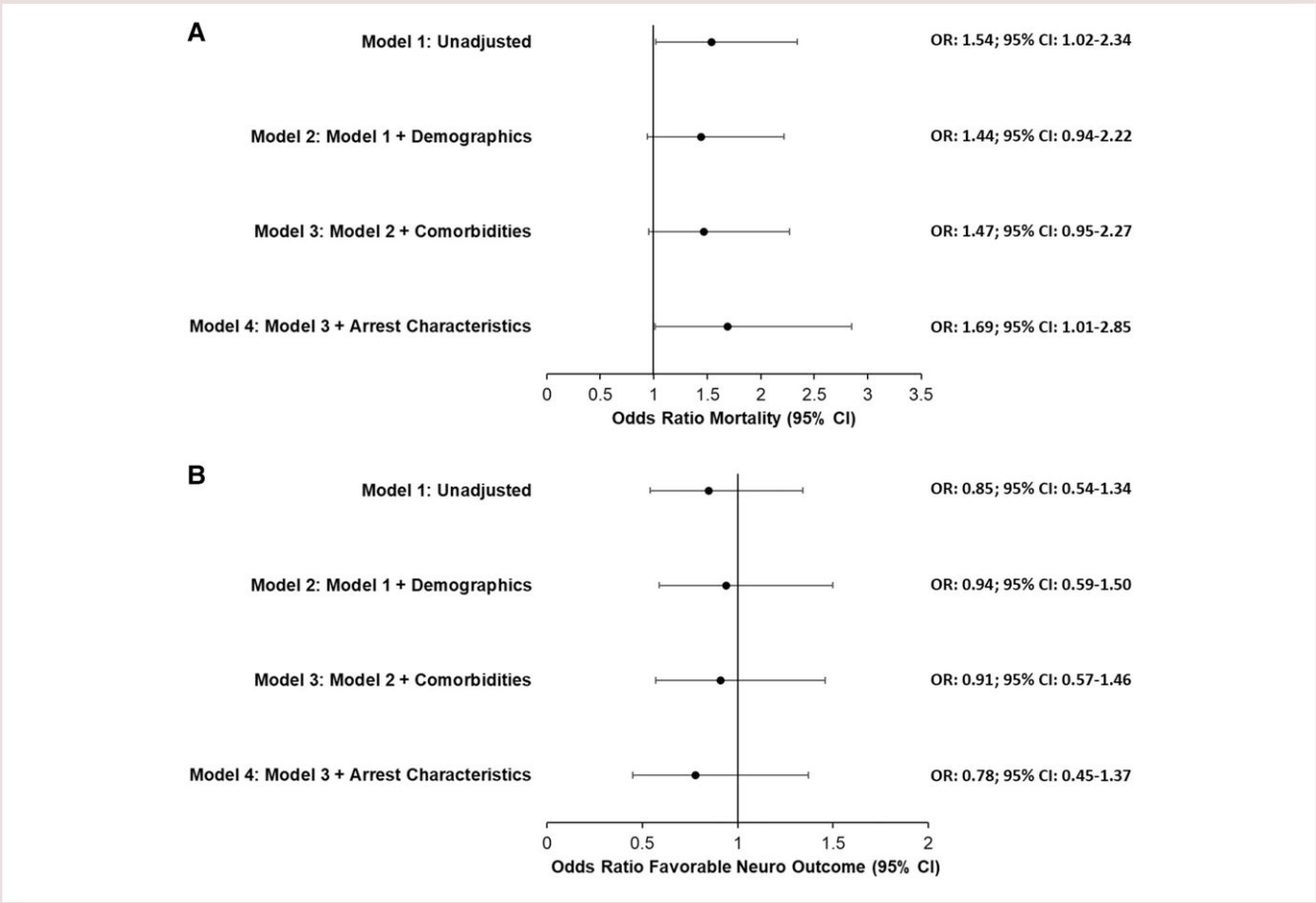


Figure 2 Odds ratios and 95% confidence intervals of the association between TpTe prolongation and (*A*) in-hospital mortality and (*B*) favourable neurologic outcome. In-hospital mortality occurred in 72.9% (207/284) patients with TpTe prolongation and 63.5% (101/159) patients without TpTe prolongation. Favourable neurologic outcome occurred in 22.9% (65/284) patients with TpTe prolongation and 25.8% (41/159) patients without TpTe prolongation.

leading cause of death in comatose patients resuscitated from cardiac arrest,[8] much of the research on risk stratification in this population centres on markers of neurologic injury.[5,6] However, up to one-third of patients die of cardiovascular causes including shock and arrhythmia.[8,9] Synthesis of both markers of neurologic injury and markers of non-neurologic organ dysfunction, including electrocardiographic signs of cardiac dysfunction, may further improve risk stratification in this challenging and heterogenous patient population.[28]

Our findings should be interpreted considering several limitations, including our single-center and retrospective, observational study design. Our study included a heterogenous patient population with both OHCA and IHCA, and further studies may seek to evaluate if the observed associations between ventricular repolarisation and clinical outcomes are consistent across arrest subgroups (e.g. location cardiac arrest, initial rhythm, presumed aetiology). Additionally, the present study specifically investigates the association between repolarisation after ROSC and clinical outcomes. The effect of repolarisation abnormalities detected preceding cardiac arrest on clinical outcomes was outside of the scope of the present study. While interobserver variability is a source of error in manual ECG interpretation, interobserver agreement was excellent. An important limitation of our study is the lack of data on mode of death, which limits our ability to potentially identify associations/mechanisms between TpTe prolongation and the aetiology of increased in-hospital mortality (e.g. higher rates of ventricular arrhythmia). Furthermore, this limits potential clinical application beyond identifying a higher risk cohort.

Conclusions

TpTe interval prolongation, but not QTc interval prolongation, was associated with greater odds of in-hospital mortality in patients resuscitated from cardiac arrest. The TpTe interval may represent an electrocardiographic biomarker for adverse outcomes in patients resuscitated from cardiac arrest.

Supplementary material

Supplementary material is available at *European Heart Journal: Acute Cardiovascular Care* online.

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Author contributions

Christopher Schenck (Methodology, Formal analysis, Writing—original draft), Soumya Banna (Writing—original draft), Noah Kim (Data curation), Christine Nguyen (Data curation), Emily J. Gilmore (Writing—review & editing), David M. Greer (Writing—review & editing), Rachel Beekman (Conceptualization, Supervision, Writing—review & editing), and P. Elliott Miller (Conceptualization, Supervision, Methodology, Formal analysis, Writing—review & editing).

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Data availability

In order to protect individual patient confidentiality, supporting data is not available.

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