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# Prolonged $T_{peak}$-$T_{end}$ interval is associated with ventricular fibrillation during reperfusion in ST-elevation myocardial infarction

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ABSTRACT

*Aim:* Ventricular fibrillation (VF) during reperfusion in ST-elevation myocardial infarction (STEMI) is associated with increased in-hospital mortality. Dispersion of ventricular repolarization contributes to ventricular vulnerability during ischemia. $T_{peak}$-$T_{end}$ interval was proposed as a ventricular repolarization dispersion marker, however its value for prediction of reperfusion VF remains uncertain. We aimed to assess whether $T_{peak}$-$T_{end}$ before PCI in STEMI is associated with reperfusion VF.

*Methods:* STEMI patients admitted for primary PCI were retrospectively assessed for VF during reperfusion. Pre-PCI ECGs recorded in 40 patients with reperfusion VF (rVF group; age 65 ± 13 years, 80% male) were compared with 374 consecutive patients without reperfusion arrhythmias (No-rVF group; age 67 ± 12 years; 68% male). Digital ECGs were automatically processed and $T_{peak}$-$T_{end}$ interval computed on a per-lead basis. The global $T_{peak}$-$T_{end}$ was calculated between the earliest $T_{peak}$ and the latest $T_{end}$ in any lead, and tested for association with reperfusion VF using logistic regression analysis.

*Results:* The leftward shift of $T_{peak}$ toward QRS complex in ischemic leads resulted in $T_{peak}$-$T_{end}$ prolongation. Global $T_{peak}$-$T_{end}$ in rVF group was higher than in No-rVF group (142 ± 24 vs 130 ± 27 ms; $p = 0.007$). Global $T_{peak}$-$T_{end}$ ≥ 131 ms predicted reperfusion VF (OR = 3.41; 95% CI 1.66–7.04; $p = 0.001$) and remained a significant predictor of reperfusion VF in multivariable analysis.

*Conclusion:* $T_{peak}$-$T_{end}$ interval before PCI in STEMI was an independent predictor of reperfusion VF. Our findings warrants further research aimed at prospective validation of $T_{peak}$-$T_{end}$ as a marker of periprocedural arrhythmic risk.



## 1. Introduction

Ventricular fibrillation (VF) complicates the course of ST-elevation myocardial infarction in 3–7% [1,2], and in 1.9% occurs during reperfusion [3]. VF occurring after successful blood flow restoration in a previously occluded coronary artery is regarded as reperfusion-induced VF. The first observation of VF within seconds of blood flow restoration was made in experimental settings in 1881 [4]. Since then, experimental studies have been searching for specific mechanisms of reperfusion arrhythmias on a cellular level. The increased inhomogeneity of action potential duration in and around the previously ischemic zone immediately after abrupt restoration of blood flow was reported to enhance the likelihood for re-entry and predispose to VF [5]. The presence of multiple re-entry circuits in the ischemic area has been demonstrated during reperfusion-induced VF, however the initial ectopic impulses that induce fibrillation are usually caused by abnormal automaticity or triggered activity, but not by the re-entry mechanism [6].

The literature regarding predictors of reperfusion VF is limited and concerns mostly the clinical and angiographic characteristics of patients admitted with STEMI. In the PAMI trial, the grade 0 TIMI flow before PCI, short time from symptoms onset to emergency room and right coronary artery-related infarct were associated with VF during catheterization [7]. The data on dynamic electrocardiographic changes that can predict VF, especially VF at reperfusion, are scarce. At the same time, prediction of impending VF would have shortened the time to defibrillation, even in cardiac catheterization laboratory, and improved patient prognosis. Electrocardiography (ECG), being non-invasive, simple and commonly used in STEMI settings, represents the source for VF predictors investigation. Since ischemia and reperfusion are accompanied by profound

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alterations in ventricular repolarization dispersion [8], which is well known to predispose to arrhythmic complications [9], the variables reflecting dispersion of repolarization are of special interest.

$T_{peak}$-$T_{end}$ interval was proposed as a marker of dispersion of ventricular repolarization [10]. Prolonged $T_{peak}$-$T_{end}$ has been shown to be a marker of arrhythmogenesis in various cardiac disorders [11], but its predictive value in general population is controversial [12,13]. The prolongation of $T_{peak}$-$T_{end}$ interval was found to be an independent predictor of appropriate ICD therapy in patients with myocardial infarction history [14]. The clinical data on $T_{peak}$-$T_{end}$ in acute ischemic settings are scarce. Some case-control studies reported the prolongation of $T_{peak}$-$T_{end}$ interval in acute STEMI settings [15]. In STEMI patients, prolonged $T_{peak}$-$T_{end}$ before [16,17] and after [18] percutaneous coronary intervention (PCI) was shown to be associated with increased mortality. The value of $T_{peak}$-$T_{end}$ for prediction of reperfusion VF has not been studied yet. Our aim was to analyse the association of $T_{peak}$-$T_{end}$ with VF during reperfusion in large unselected population of STEMI patients undergoing primary PCI.

## 2. Methods

### 2.1. Study population

The study population included consecutive STEMI patients admitted to Skane University Hospital for primary PCI during 2007–2012. To select patients into the study group, the data of the Swedish National Register of Information and Knowledge about Swedish Heart Intensive Care Admissions (RIKS-HIA) and Swedish Coronary Angiography and Angioplasty Register (SCAAR) were analysed. RIKSHIA register contains information about cardiopulmonary resuscitation or defibrillation, and SCAAR register – facts on reperfusion arrhythmias during procedure. The information obtained from both registers was then crosschecked, and medical histories of patients were scrutinised to verify VF occurrence and VF timing in relation to infarct-related artery (IRA) opening. Relevant clinical information was taken from the RIKS-HIA Register, angiographic characteristics were determined from the SCAAR Register.

ECGs stored in digital format either in GE Marquette MUSE system (GE Medical Systems, Milwaukee, Wisconsin) or Infinity MegaCare ECG Management System (Dräger, Lübeck, Germany) databases were exported. An ECG recorded after onset of STEMI, but prior to coronary intervention was defined as admission ECG. If several ECGs were recorded prior to PCI, the latest ECG was considered for analysis.

The reperfusion VF group (rVF group) comprised of patients who suffered from VF during reperfusion associated with primary PCI during 2007–2012 and in whom in-hospital admission ECG was available and suitable for automatic analysis. The control group (No-rVF group) included consecutive patients without life-threatening arrhythmias admitted for primary PCI during 2007, and in whom in-hospital admission ECG was available and suitable for automatic analysis.

### 2.2. ECG analysis

The exclusion criteria for ECG analysis were pacemaker rhythm, left or right bundle brunch block. The standard interval parameters, including heart rate, PR interval, QRS duration, were calculated. The QT intervals were adjusted for heart rate using Bazett and Fredericia's corrections.

Based on the automatically identified fiducial points of the QRST complex derived by the Glasgow algorithm [19], $T_{peak}$ was determined as the moment of maximum absolute T wave amplitude in each lead. The global $T_{peak}$-$T_{end}$ was calculated as an interval between the earliest $T_{peak}$ and the latest $T_{end}$ in any lead (Fig. 1). If due to artefacts, noise or isoelectric T wave morphology, the measurements could not be accomplished in at least five leads, that should include at least one inferior (II, III or aVF), one anterior ($V_1$, $V_2$ or $V_3$) and one lateral (I, $V_5$ or $V_6$) lead, the ECG was excluded for global $T_{peak}$-$T_{end}$ calculation. No manual adjustment of automatically identified fiducial points of the QRST complex was performed.

### 2.3. Statistical analysis

To identify clinical factors associated with VF at reperfusion, relevant clinical, angiographic and electrocardiographic factors were compared across groups using chi-square or Fisher's exact test for categorical variables and Student's *t*-test for continuous variables with an approximate normal distribution, or Man-Whitney *U* test, as appropriate. Significantly associated covariates were further evaluated in univariate logistic regression models with estimation of odds ratios and likelihood-ratio tests. To determine independent factors of risk, variables significantly associated with reperfusional VF in univariate models and clinically relevant parameters were included in a stepwise regression analysis with backwards elimination. p values < 0.05 were considered significant. All analyses were performed using SPSS 22.0 (SPSS Inc., Chicago, Illinois, USA).

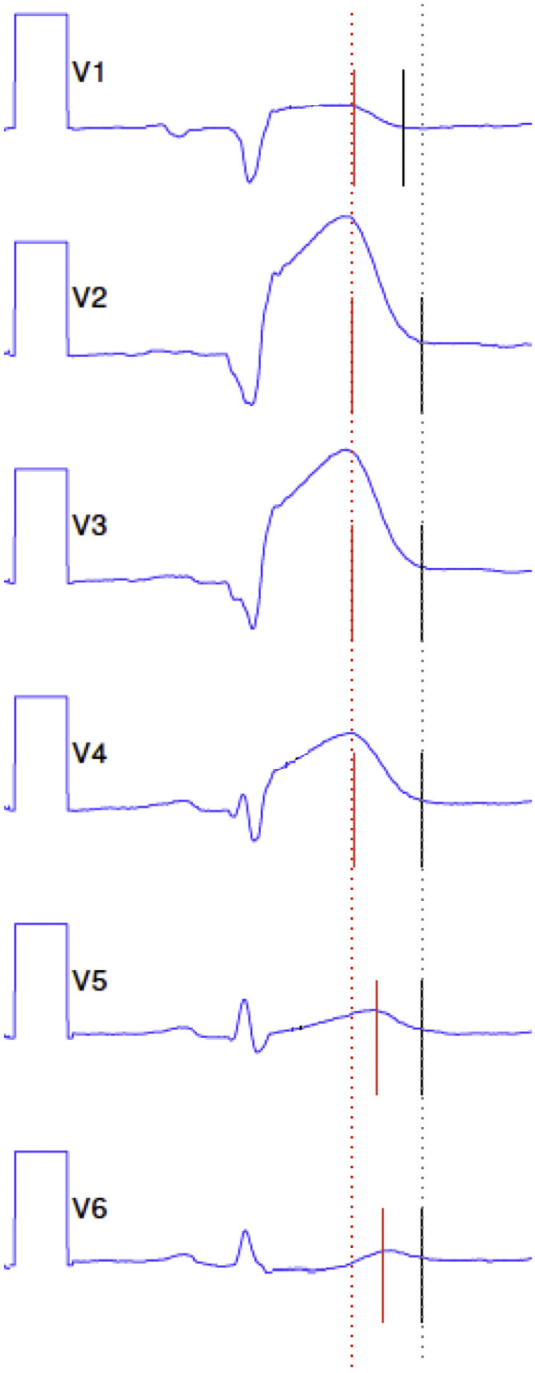


**Fig. 1.** The representative QRST complexes in precordial leads with automatic $T_{peak}$ marks (red) and $T_{end}$ marks (black). The red dashed line corresponds to the earliest $T_{peak}$ in represented leads. The black dashed line corresponds to the latest $T_{end}$ in represented leads. Note the shift of $T_{peak}$ toward QRS onset in right precordial leads corresponding to ischemic myocardial region.

## 3. Results

### 3.1. Patient characteristics

Among 3274 STEMI patients admitted for primary PCI during 6-year period from 2007 till 2012, 71 (1.9%) suffered from VF during reperfusion [3]. Admission in-hospital ECGs were available in 45 of them. Thus, 45 patients who suffered from reperfusion VF and had admission ECGs available comprised rVF group. No-rVF group comprised of 414 of

627 patients who were admitted with STEMI during 2007, not suffered from VF and had in-hospital admission ECG available for analysis. Two patients in the rVF group were excluded from electrocardiographic analysis due to ventricular paced rhythm. Patients with LBBB/RBBB were also excluded from the repolarization assessment, leaving for analysis 40 patients in rVF group and 402 patients in No-rVF group. PCI was not performed in 28 of 442 patients, all of them from No rVF group, due to technical difficulties or uncertain culprit lesion; these 28 patients were excluded. Twelve of these patients underwent subsequent coronary artery bypass graft surgery. Remaining 414 patients - 40 in rVF group and 374 in No-rVF group, formed the basis of future analysis.

Patient characteristics are presented in Table 1. Patients with inferior localization of myocardial infarction and patients with right coronary artery (RCA) as infarct related artery (IRA) more often suffered from VF during reperfusion. Patients with reperfusion VF had shorter symptom-to-baloon time. There were no differences between the groups with regard to age, gender, history of myocardial infarction, hypertension, diabetes and chronic heart failure, as well as using B-blockers and aspirin. The proportion of multivessel disease and left main stenosis did not differ between groups either (Table 1). All 40 patients who suffered from reperfusion VF, had total acute coronary occlusions, while there were no cases of non-occlusive stenosis in this group. The rate of thrombectomy in rVF group was 47% comparing to 12% in the No rVF group, $p < 0.001$. Patients suffered from VF during reperfusion had greater QRS duration before PCI. The groups did not differ in heart rate, PR duration and QTc.

### 3.2. $T_{peak}$-$T_{end}$ interval and its predictive value for VF during reperfusion

Global $T_{peak}$-$T_{end}$ in patients with VF during reperfusion was higher, than in patients without life-threatening arrhythmias (142 ± 24 vs 130 ± 27; $p = 0.007$). Using ROC curve analysis, the optimal cut-off for global $T_{peak}$-$T_{end}$ for VF prediction was identified as 131 ms (Sp = 73%; Se = 58%; AUC = 0.657, $p = 0.001$). Global $T_{peak}$-$T_{end}$ ≥ 131 ms and higher allows to predict VF at reperfusion with OR = 3.41; 95% CI 1.66–7.04; $p = 0{,}001$. Global $T_{peak}$-$T_{end}$ ≥ 131 ms remained a significant predictor of VF at reperfusion after adjusting for age, gender, smoking, myocardial infarction localization and QRS duration (OR = 3.74; 95% CI 1.70–8.14; $p = 0.001$).

$T_{peak}$-$T_{end}$/QT ratio was also higher in the rVF group (0.34 ± 0.05 vs 0.31 ± 0.06; $p = 0.001$). The optimal cut-off of $T_{peak}$-$T_{end}$/QT for VF prediction was found to be 0.32 (Sp = 65%; Se = 65%; AUC = 0.677, $p < 0.001$). $T_{peak}$-$T_{end}$/ QT >0.32 predicted VF at reperfusion with OR = 3.00; 95% CI 1.52–5.94; $p = 0{,}002$.

In patients with anterior myocardial infarction (IRA – LAD, n = 166), we observed the greater values of $T_{peak}$-$T_{end}$ in patients with rVF in the precordial leads $V_2$-$V_4$. $T_{peak}$-$T_{end}$ in lead $V_2$ was longer in the rVF compared to the No-rVFgroup (119 ± 15 ms vs 103 ± 24 ms, $p = 0.029$) and was associated with rVF (OR = 1.031; 95% CI 1.003–1.060; $p = 0.032$). In patients with non-anterior infarction (IRA – RCA or LCX, n = 218) $T_{peak}$-$T_{end}$ in lead III differed between rVF and no rVF groups (119 ± 30 vs 105 ± 30 ms; $p = 0.038$) and was associated with rVF (OR = 1.016; 95% CI 1.001–1.030; $p = 0.034$).

**Table 1**
Clinical, angiographic and electrocardiographic characteristics (the whole group, n = 442).

| | No rVF (n = 374) | rVF (n = 40) | p-Value |
|---|---|---|---|
| Age, years | 67 ± 12 | 65 ± 13 | 0.412 |
| Male gender | 253 (68%) | 32 (80%) | 0.109 |
| Myocardial infarction history | 46 (12%) | 9 (23%) | 0.085 |
| Diabetes | 41 (11%) | 2 (5%) | 0.238 |
| Hypertension | 144 (39%) | 16 (40%) | 0.853 |
| Chronic heart failure | 9 (2%) | 2 (5%) | 0.332 |
| B-blockers at admission | 97 (27%) | 14 (37%) | 0.178 |
| Aspirin at admission | 94 (25%) | 13 (34%) | 0.244 |
| Smoking | 126 (36%) | 19 (51%) | 0.057 |
| **Simptom-to-ballon time**, min | **260 (300)** | **200 (168)** | **0.001** |
| **Inferior localization** | **179 (52%)** | **28 (70%)** | **0.030** |
| Multivessel disease | 209 (58%) | 21 (55%) | 0.755 |
| Left main stenosis | 26 (7%) | 6 (15%) | 0.069 |
| **IRA RCA** | **144 (40%)** | **23 (58%)** | **0.031** |
| Characteristic of stenosis: | | | |
| Acute IRA occlusion[a] | 258 (70.5%) | 40 (100%) | |
| Chronic occlusion[b] | 2 (0.5%) | 0 | **<0.001** |
| Non-occlusive stenosis in IRA | 106 (29%) | 0 | |
| Heart rate, b.p.m. | 77 ± 20 | 71 ± 20 | 0.060 |
| PR duration, ms | 170 ± 36 | 172 ± 44 | 0.745 |
| **QRS duration**, ms | **100 ± 18** | **108 ± 20** | **0.011** |
| QTc Bazett | 435 ± 31 | 426 ± 31 | 0.071 |
| QTc Fredericia | 420 ± 29 | 417 ± 25 | 0.745 |
| **Global T peak T end** | **130 ± 27** | **142 ± 24** | **0.007** |
| **$T_{peak}$ $T_{end}$/QT** | **0.31 ± 0.06** | **0.34 ± 0.05** | **0.001** |

IRA-infarct related artery; RCA-right coronary artery.
[a] Acute IRA occlusion (less than three months prior to admission) as defined by SCAAR registry.
[b] Chronic IRA occlusion (more than three months prior to admission) as defined by SCAAR registry.

## 4. Discussion

The main findings of our study are: [1] the $T_{peak}$-$T_{end}$ before PCI in STEMI is associated with VF during reperfusion; [2] the prolongation of $T_{peak}$-$T_{end}$ is due to T peak shift toward to QRS onset in infarct-related leads.

During the last decades, $T_{peak}$-$T_{end}$ interval has been intensively investigated as an ECG marker of arrhythmic and mortality outcomes [20] in various disorders, including chronic and acute forms of ischemic heart disease. $T_{peak}$-$T_{end}$ demonstrated association with malignant arrhythmias in post-MI patients eligible for ICD therapy [14]. $T_{peak}$-$T_{end}$ measured in STEMI patients after primary PCI predicted in-hospital and long-term mortality [21]. Some studies aimed at assessment of $T_{peak}$-$T_{end}$ value before PCI and its association with arrhythmias during the early course of STEMI [15,17] and all-cause mortality [16]. According to our knowledge, we are the first to investigate the predictive value of $T_{peak}$-$T_{end}$ specifically for reperfusion VF in large non-selected population of STEMI patients treated with primary PCI.

The detection of T wave borders and analysis of $T_{peak}$-$T_{end}$ in the settings of marked ST-segment elevation may be challenging, which has in earlier STEMI studies lead to $T_{peak}$-$T_{end}$ assessment being confined to the leads unaffected by the infarction [15–17]. In the literature, the most common reported leads for $T_{peak}$-$T_{end}$ measurements were leads $V_5$ [13,16], $V_6$ [15] or $V_4$ [17]. Another approach was to choose the longest $T_{peak}$-$T_{end}$ among those measured in all 12 leads, most often in $V_2$ [14]. In experimental study on porcine model of myocardial infarction we have recently shown the increase of $T_{peak}$-$T_{end}$ during ischemia progression due to T peak shift toward QRS onset in infarct-related leads [22]. The same phenomenon was observed in the current study performed in clinical settings: T peak shifted toward to QRS onset in the infarct-related leads, leading to $T_{peak}$-$T_{end}$ prolongation confined to the affected myocardial region (Fig. 1). Therefore, in our analysis we did not limit $T_{peak}$-$T_{end}$ assessment to the non-infarcted leads, but calculated global $T_{peak}$-$T_{end}$ based on the information available from all the 12 leads.

Because of using global $T_{peak}$-$T_{end,}$ defined as an interval between the earliest $T_{peak}$ and the latest $T_{end}$ in any lead, the cut-off values of $T_{peak}$-$T_{end}$ for VF prediction in our study were higher than in the previously published literature (131 ms in our study vs mean of 103 ms in the recently published metaanalysis) [20]. At the same time, the values of $T_{peak}$-$T_{end}$ measured on per-lead basis in our study are quite comparable with previously reported ones (the mean $T_{peak}$-$T_{end}$ in $V_5$ in rVF group in our study was 101 vs 104 ms in the study in STEMI patients by Haarmark et al.) [16]. The cut-off ratio of $T_{peak}$-$T_{end}$/ QT for rVF prediction in our study is comparable to values described by Mugnai and co-authors in population of anterior STEMI (0.32) [17].

The values calculated on per-lead basis (in $V_2$-$V_4$ and in III respectively) were associated with reperfusion VF in subgroups of anterior and inferior infarctions, but not for the whole group. The rationale for the better performance of the global $T_{peak}$-$T_{end}$ was the abovementioned $T_{peak}$ shift in leads corresponding to ischemic myocardial region. Experimental studies indicated that the peak of T wave corresponds to the earliest end of repolarization in the myocardial region generating a signal in a given ECG lead [10]. Therefore, the shift of $T_{peak}$ toward to the QRS onset in ischemic leads, most likely, reflects the shortening of the earliest repolarization time in ischemic region. At the same time, T wave end remained relatively stable, thus corresponding to the unaffected end of repolarization in remote non-ischemic region. The unaffected position of the $T_{end}$ is also reflected in a relatively stable duration of QT interval during acute ischemia [22].

Several factors can predispose to reperfusion arrhythmias in STEMI, among them the size of area myocardium at risk involved in ischemia-reperfusion, the acuteness of ischemia, the localization of myocardial infarction. Prolonged $T_{peak}$-$T_{end}$ interval reflects an increased myocardial dispersion of repolarization [9,23,24], which predispose to unidirectional conduction block development leading to potential vulnerability to re-entrant ventricular arrhythmias during ischemia-reperfusion in STEMI.

## 5. Limitations

Our study was a retrospective registry-based analysis of patients admitted to our hospital with STEMI and for whom invasive strategy was chosen and initiated. Patients who might have had STEMI but died prior to PCI or for some reasons not selected for the intervention were not included in the analysis. Thus our study should be considered as a hypothesis-generating and interpreted in the view of limitations inherent to its retrospective study design.

$T_{peak}$-$T_{end}$ interval was calculated from standard 12 lead ECG taken after onset of STEMI at a certain time distance before PCI; therefore not representing the ECG information immediately before PCI.

## 6. Conclusion

In STEMI patients, the duration of the $T_{peak}$-$T_{end}$ interval before PCI is associated with VF during reperfusion. Our findings warrants further research aimed at independent verification in similar cohorts and prospective validation of $T_{peak}$-$T_{end}$ as a marker of periprocedural arrhythmic risk.

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## Conflict of interest

The authors report no conflict of interest.

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