

## Assessment of Tpeak-Tend/QT Ratio for Predicting Malignant Arrhythmias among ST Segment Elevation Myocardial Infarction Cases Undergoing Primary Coronary Intervention

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### ABSTRACT

**Background:** In individuals with ST-segment elevation myocardial infarction, the most telling sign of arrhythmogenesis was a lengthening of the Tpeak-Tend (Tp-e) interval.

**Objective:** To evaluate the Tpeak-Tend/QT ratio in predicting malignant arrhythmias among cases who had myocardial infarction (elevated ST segment) and underwent primary percutaneous coronary intervention.

**Subjects and methods:** In a prospective cohort study; we classified the selected cases into 2 groups: Group A: included 78 cases with complete-ST segment resolution (CSTR), which meant they got a successful complete reperfusion. Group B: included 42 patients with incomplete-STR (ISTR), meant they got an unsuccessful complete reperfusion.

**Results:** Significant lower values of ST elevation, ventricular rate, and QT interval were found among CSTR group than ISTR group, significant lower corrected QT was found among CSTR group than ISTR group. Significant lower Tp-Tec, and Tp-e/QT ratio were found among CSTR group than ISTR group. AUC of ROC curve of Tp-e/QT and Tp-Tec were 0.746, 0.838. At cutoff  $\geq 0.305$  and  $\geq 133.5$ , the sensitivity was 55.0% and 85.7% and specificity was 62.8% and 64.1% respectively. **Conclusion:** The electrophysiologic feature that represents transmural dispersion of cellular repolarization (TDR) in STEMI was similar in nature for both an elevated ST-segment and an elevated Tp-ec. Patients with STEMI after PPCI had an increased STR and Tp-e/QT ratio, both of which predicted arrhythmia and had predictive value for malignant arrhythmia.

**Keywords:** ST Segment Elevation Myocardial Infarction, Primary Coronary Intervention (PCI).

### INTRODUCTION

Coronary artery diseases, that contribute to myocardial infarction (MI), is a major etiology for death as well as disability in modern civilization. Arrhythmogenic substrates, such as extended QT intervals and T wave alternans, have been the focus of electrophysiological characterization investigations in patients with myocardial infarction <sup>(1)</sup>.

Several ion channel illnesses have been linked to an increase in the time it takes for the T wave to complete its cycle from peak to end (Tpeak-Tend interval [Tp-e]). Malignant arrhythmia and sudden cardiac death (SCD) are thought to be more likely as a result of this rise. The Tp-e interval, calculated from an electrogram taken across the wedge, has been shown to correlate well with the transmural dispersion of cellular repolarization in experiments using rabbit as well as canine left ventricular wedge models <sup>(2)</sup>.

Cases who had congenital long QT syndrome (LQTS) or short QT syndrome (SQTS) frequently exhibit a prolonged Tp-e interval, which plays a role in the arrhythmogenesis of these conditions. This evidence lends credence to the hypothesis that the Tp-e/QT ratio is a reliable biomarker of ventricular repolarization variability that is independent of HR. Recent researches have revealed that Tp-e/QT ratios, rather than only QT, QTc, or Tp-e intervals, are the best predictors of ventricular arrhythmias <sup>(3)</sup>. Patients reporting chest pain should have urgent coronary revascularization if they have an elevated ST segment on the ECG, the most important clinical criterion for this decision. This is due

to the fact that acute transmural myocardial infarction is characterized by an elevated ST segment. The degree to which STR resolves after reperfusion therapy among cases with ST-segment elevation myocardial infarction (STEMI) has been demonstrated in numerous studies to be predictive of left malignant arrhythmia, ventricular function preservation, and sudden cardiac death <sup>(4)</sup>.

Recent research has demonstrated that STR is incomplete in ECG leads even when significant epicardial vascular recanalization has been achieved, indicating that even if myocardial microcirculation disruption is present, myocardial tissue perfusion may not be adequate. Brugada syndrome (Brs) and ST-elevation myocardial infarction (STEMI) are characterized by an elevated Tpeak-Tend (Tp-e) interval in electrocardiogram (ECG) leads, which represents phase 2 reentry in ischemic cellular models and is the fundamental cause of arrhythmogenesis in these patients <sup>(5)</sup>.

We aimed at this work to assess the Tpeak-Tend/QT ratio in prediction of malignant arrhythmias among cases who had ST segment elevation myocardial infarction and underwent primary percutaneous coronary intervention.

### PATIENTS AND METHODS

This is prospective cohort study that was done at Coronary Intensive Care Unit and of Cardiology Department at Zagazig University Hospital and continued for follow up for 6 months.

**Selected cases were divided into 2 groups:**

**Group A:** included 78 cases with complete-STR (CSTR), (Successful reperfusion).

**Group B:** included 42 cases with incomplete-STR (ISTR), (Incomplete reperfusion).

**Inclusion Criteria:**

- Adult patients aged > 18 years old.
- Typical chest pain of 1<sup>st</sup> attack of STEMI presented <12 hours and lasting >30 minutes, having an ST-segment elevation of more than 0.1 mV in two consecutive limb leads or more than 0.2 mV in two consecutive precordial leads.
- Serum troponin I levels that are abnormally high compared to reference intervals.

**Exclusion Criteria:**

- Cases who had left or right bundle branch block.
- Ventricular pacing cases.
- Corresponding artery restenosis after myocardial infarction, which was confirmed by a subsequent coronary angiogram.
- Electrolyte disturbances that could affect QT interval.
- Patients receiving antiarrhythmic drugs that could affect QT interval.

**The following was done to all of the groups:**

**1. History taking:**

A complete medical history was obtained, including but not limited to gender, age, antibiotics, tricyclic-antidepressants, antihistamines, antipsychotics, and antiarrhythmics are all examples of conditions and drugs that can alter QT and Tp-e intervals.

**2- Clinical examination**

There was a thorough evaluation of cardiac health both systemically and locally.

**3- Lab investigations:**

- Blood sugar.
- Complete blood count.
- High sensitive serum cardiac troponin I.
- Serum creatinine.
- Serum sodium and potassium.

**4- Electrocardiogram (ECG):**

▪ **(1<sup>st</sup> ECG):**

A 12-lead resting electrocardiogram (ECG) was obtained from the patient while they were laying supine in the hospital using a Fukuda Denshi CardimaxFX-2111 Electrocardiograph (USA). The paper speed was 25 mm/s, and the standardisation was 10 mm. The ECG data were used to calculate the patient's resting heart rate and Tp-e as well as QT intervals were measured manually on electrocardiograms using slide calipers and a magnifying glass to reduce the likelihood of human error. Three separate tests were averaged for their respective values. The QT interval was calculated by timing how long it took for the tangent of the T wave's steepest descending limb to cross the isoelectric baseline from the start of the QRS complex.

- **100 minutes after primary coronary intervention (PCI), an electrocardiogram was taken (2<sup>nd</sup> ECG).**
- Both the QT and Tp-e intervals underwent intensive manual inspection. Using the Bazett formula, we calculated the corrected QT (QTc) and pause-to-peak (Tp-e) intervals (Tp-ec). The QT interval was calculated by counting the number of seconds between the start of the QRS complex and the end of the T-wave in the electrocardiogram. T-wave duration was determined by recording its rise and fall times (the Tp-e interval). If the T wave was negative or biphasic, the peak was located by measuring the height from the highest to lowest point.
- Three consecutive heartbeats were averaged for each parameter, and the highest values for QTc and Tp-ec in ST-segment raised leads were used for the analysis.
- The maximal ST-segment elevation between the first and second ECGs was measured to determine the degree of STR.
- Complete STR (CSTR) patients had a decrease of at least 50% in ST-segment elevation from the baseline to the second ECG, while incomplete STR (ISTR) patients had no such decrease.

**5- Coronary Angiography:**

Coronary angiography and PPCI outcomes were classified based on TIMI flow <sup>(6)</sup>.

**6. Echocardiography:**

After 24 hours of reperfusion, left ventricular ejection fraction (LVEF percent) was measured in all patients using the Simpson's method.

**Evaluation and follow up:**

At 6 months, the key outcome was the occurrence of malignant arrhythmia episodes, which included ventricular tachycardia/fibrillation, syncope, and attempted sudden cardiac death. For patients who have several adverse events, the initial event was utilized to calculate the composite clinical endpoint.

**Ethical considerations:**

**The Institutional Review Board at Zagazig University offered their approval, and all patients provided informed consent. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.**

**Statistical analysis**

The dependent-samples Student t test was used to do comparisons between continuous variables expressed as means and standard deviations. Youden index = Max ([sensitivity] + [specificity] - 1) was utilized for determination of optimal cutoff value, which is the value with the highest sensitivity and specificity. SPSS 20.0.0 was used for the statistical analysis (IBM Inc, Armonk, NY). Z test was used to compare area under ROC curves (AUCs) using MedCalc15.2.2. P value, 0.05 was considered significant.

**RESULTS**

Statistically, in terms of age and gender, there was no discernible difference among 2 studied groups (Table 1).

**Table (1): Demographics of the participated groups**

Variable		Group A n= 78	Group B n= 42	P value
Age (years)	Mean ± SD	56.4± 11.8	57.9± 11.3	0.494
Gender	Male, n (%)	49 (62.8)	25 (59.5)	0.723
	Female, n (%)	29 (37.2)	17 (40.5)	

Significant lower values of ST elevation, ventricular rate, and QT interval were found among CSTR group than ISTR group. Significant lower Corrected QT was found among CSTR group than ISTR group. Significant lower Tp-Tec, and Tp-e/QT ratio were found among CSTR group than ISTR group (Table 2).

**Table (2): ECG among the participants**

Variable		Group A n= 78	Group B n= 42	P value
Symptom to balloon (minutes)	Mean ± SD	257.1 ± 110.7	262.6 ± 112.7	0.728
Max. ST elevation (mm)	Mean ± SD	4.7± 2.3	5.8± 2.8	<b>0.021*</b>
Ventricular rate	Mean ± SD	82.6± 8.1	89.1± 8.9	<b>&lt;0.001*</b>
100 min. post PCI QT interval (ms)	Mean ± SD	375.3± 17.8	382.4± 18.1	<b>0.039*</b>
100 min. post PCI Corrected QT (ms)	Mean ± SD	435.4± 38.1	463.8± 18.5	<b>&lt;0.001*</b>
Tp-Te	Mean ± SD	109.1± 10.7	122.9± 12.5	<b>&lt;0.001*</b>
Tp-Tec	Mean ± SD	127.9± 13.3	149.4± 14.9	<b>&lt;0.001*</b>
Tp-Te/QT	Mean ± SD	0.291± 0.025	0.322± 0.032	<b>&lt;0.001*</b>

\*: Significant.

In the CSTR group, EF was noticeably higher. While CSTR has a much lower LVEDD. In the CSTR group, the LVESD was drastically reduced. Neither the RV nor the LA size were significantly different between the CSTR and ISTR groups (Table 3).

**Table (3): Echocardiography of the participated groups**

Variable		Group A n= 78	Group B n= 42	P value
EF (%)	Mean ± SD	49.4± 7.3	45.8± 5.9	<b>0.007*</b>
LVEDD (mm)	Mean ± SD	48.2± 2.7	49.5± 3.3	<b>0.022*</b>
LVESD (mm)	Mean ± SD	35.3± 3.0	37.5± 2.6	<b>&lt;0.001*</b>
RV size (mm)	Mean ± SD	24.5± 4.3	25.4± 3.8	0.251
LA size (mm)	Mean ± SD	33.5± 5.5	34.6± 4.8	0.309

EF; Ejection fraction, LVEDD; left ventricular end diastolic dimension, LVESD; left ventricular end systolic dimension, \*: Significant.

Non-significant differences were found among coronary arteries i.e., RCA, LCX, LAD, or Culprit artery (Table 4).

**Table (4): TIMI flow grade among the two studied groups**

Variable		Group A (n= 78)	Group B (n= 42)	P value
LCX	TIMI 2, n (%)	6 (7.7%)	3 (7.1%)	>0.999
	TIMI 3, n (%)	60 (76.9%)	32 (76.2%)	
RCA	TIMI 2, n (%)	9 (11.5%)	4 (9.5%)	0.786
	TIMI 3, n (%)	41 (52.6%)	25 (59.5%)	
LAD	TIMI 2, n (%)	13 (16.7%)	3 (7.1%)	0.218
	TIMI 3, n (%)	27 (34.6%)	17 (40.5%)	
Culprit artery	Left anterior descending artery	38 (48.7%)	22 (52.4%)	0.862
	Left circumflex artery	12 (15.4%)	7 (16.7%)	
	Right coronary artery	28 (35.9%)	13 (31.0%)	

LCX; left circumflex artery, RCA; right circumflex artery, LAD; left anterior descending coronary artery

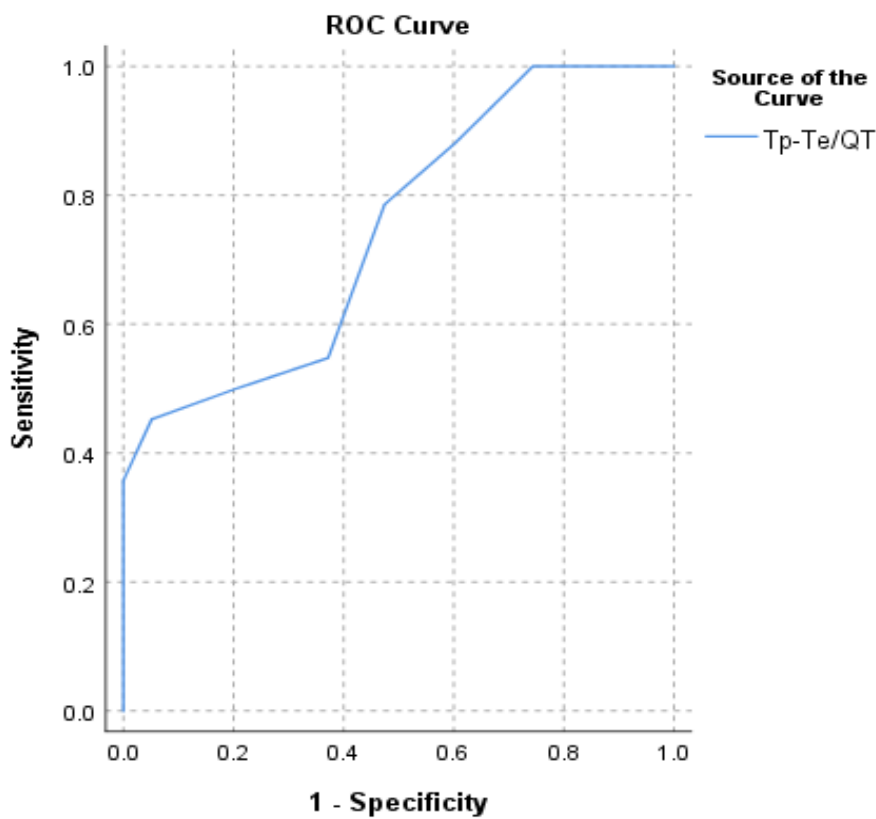
QTc was found to be significantly related to STR. However, STR was substantially linked to elevated Tp-ec and Tp-e/QT ratio (Table 5).

**Table (5): Univariate logistic binary regression of ST resolution**

Variable	OR univariate (95% CI)	P value
QT corrected	1.018 (0.987-1.049)	0.264
Tp-ec	1.349 (1.170-1.556)	<0.001*
Tp-e/QT	0.00 (0.000-0.000)	0.004*

\*: Significant.

Tp-e/sensitivity, QT's specificity, and optimal cutoff values for predicting STR were evaluated using ROC analysis. There was a 0.746 AUC for ROC. With a cutoff of 0.305, we found that the sensitivity was 55.0% and the specificity was 62.8% (Figure 1).



**Figure (1): Tp-e/QT Roc Curve in for STR Prediction**

The sensitivity, specificity, and optimal cutoff values for Tp-ec in predicting STR were determined by ROC curve analysis. The area under the curve for ROC was 0.838. At cutoff  $\geq 133.5$ , the sensitivity was 85.7% and specificity was 64.1% (Figure 2).

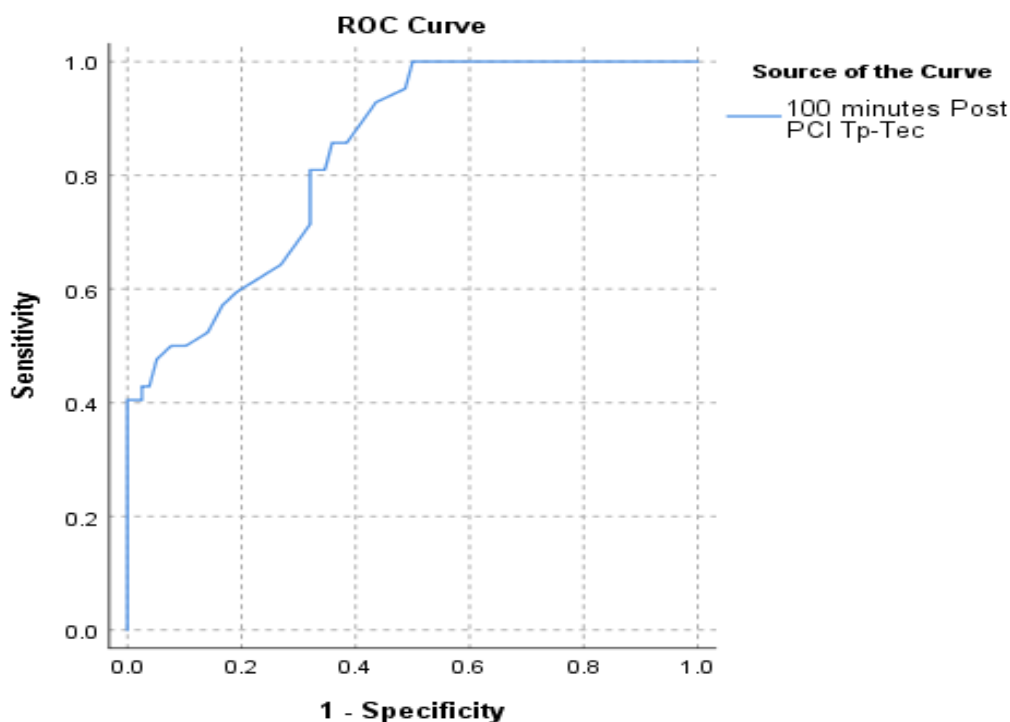


Figure (2): Tp-ec Roc Curve for STR Prediction

Statistically, there was statistical significance association between Tp-e/QT and the endpoints during follow up in hospital (Table 6).

Table (6): Comparing endpoints in hospital follow up according to Tp-e/QT

Variable		Tp-e/QT <0.305 N=68		Tp-e/QT >0.305 N= 52		P value
Total complications	No	66	97.1%	44	84.6%	0.02*
	Yes	2	2.9%	8	15.4%	

\*: Significant.

There was statistically significant association between Tp-e/QT and the endpoints during 6 months follow up (Table 7).

Table (7): Comparing endpoints in after 6 months follow up according to Tp-e/QT

Variable		Tp-e/QT <0.305 N=68		Tp-e/QT >0.305 N= 52		P value
Total complications	No	63	92.6%	41	78.8%	0.028*
	Yes	5	7.4%	11	21.2%	

\*: Significant.

There was no statistically significant association between Tp-ec and the endpoints after 6 months follow up (Table 8).

Table (8): Comparing endpoints after 6 months follow up according to Tp-ec

Variable		Tp-ec <133.5 N=56		Tp-ec >133.5 N=64		P value
Total complications	No	52	92.9%	52	81.3%	0.104
	Yes	4	7.1%	12	18.8%	

## DISCUSSION

Recent researches have suggested that the Tpeak-Tend interval (Tp-e) can be used to predict malignant arrhythmia as well as sudden cardiac death (SCD) in certain ion channel disorders. Starting at the T wave's apex and finishing at its trough establishes its duration. Transmural dispersion of cellular repolarization is strongly reflected in wedge electrograms (Tp-e interval), as shown in studies using canine and rabbit left ventricular wedge models (TDR). Circulation, a scholarly publication, published this study<sup>(2,7)</sup>.

Arrhythmogenesis, in which a prolonged Tp-e interval plays a role, is common in patients with long QT syndrome (LQTS) and short QT syndrome (SQTS) (also known as TDR at the cellular level). These data indicates that the Tp-e/QT ratio has potential as an independent indicator of ventricular repolarization variability. Recent researchers have demonstrated that a more accurate predictor of ventricular arrhythmias is the Tp-e/QT ratio than the QT interval, QTc, or Tp-e interval alone<sup>(3,8)</sup>.

As regard to our results we got, age and gender did not differ significantly among 2 groups. In agreement with our results, **Wang et al.**<sup>(4)</sup> who were interested in learning more about the electrophysiological relationship between an increased ST segment and a prolonged Tp-e interval and the predictive utility of MACE in STEMI during PCI. Differences in age or sex between the groups did not reach statistical significance.

In the current study, significant lower values of ST elevation, ventricular rate, and QT interval were found among CSTR group than ISTR group. Significant lower corrected QT was found among CSTR group than ISTR group. Tp-Tec and Tp-e/QT ratio were both significantly lower in the CSTR group compared to the ISTR group. The CSTR group had significantly better EF. While CSTR has a much lower LVEDD. In the CSTR group, the LVESD was drastically reduced. RV and LA sizes did not differ significantly between the CSTR and ISTR groups, statistically speaking.

**Reza et al.**<sup>(9)</sup> found that the average ratio of Tp-e to QT in group 1 was  $0.196 \pm 0.029$ , while in group 2 it was  $0.309 \pm 0.053$ . The Tp-e/QT ratio differed significantly among the 2 groups. Cases who had arrhythmias had a significantly higher mean Tp-e/QT ratio than those without arrhythmias ( $0.29 \pm 0.067$  vs.  $0.242 \pm 0.068$ ;  $p=0.001$ ).

**Zhao et al.**<sup>(10)</sup> conducted a study with 338 individuals (N = 338) who had STEMI and were treated with PPCI. Electrocardiograms were used to measure Tpe and Tpe/QT ratio among cases who had ST-segment elevation. Patients who died or had MACE had a considerably higher Tpe/QT ratio, indicating that both mortality and major adverse cardiovascular events were more likely to occur in this group (area under ROC curve 0.88;  $P < 0.001$ ).

In the current study, RCA, LCX, LAD, and culprit artery did not differ significantly among studied groups.

In comparison with our results, **Wang et al.**<sup>(4)</sup> found no statistically significant differences ( $P > 0.05$ ) between the groups with respect to the type of lesion considered to be the cause of the symptoms and the time it took to undergo revascularization. Patients in both the CSTR and ISTR groups received the same secondary preventive treatment as advised by guidelines since there was no clinically relevant difference between the treatment alternatives they had ( $P > 0.05$ ).

In our study, the univariate binary logistic regression study that looked at the ECG parameters found no significant association between QTc and STR. Tp-Tec and Tp-Tec both went up.

**Reza et al.**<sup>(9)</sup> found that the Tp-e/QT ratio, with an odds ratio (OR) of 3.845, was found to be the independently significant predictors of malignant ventricular arrhythmia in a multivariate logistic regression analysis of risk variables for this condition.

The sensitivity, specificity, and optimal cutoff values for Tp-ec in predicting STR were determined, in the current study, by ROC curve analysis. The area under the curve for ROC was 0.838. At cutoff  $\geq 133.5$  the sensitivity was 85.7% and specificity was 64.1%.

In comparison with our results, **Wang et al.**<sup>(4)</sup> showed that the Tp-e/QT ratio had an AUC greater than 0.883, but the Tp-ROC ec had an AUC less than 0.692. We discovered that the Tp-e/QT interval AUC was significantly different from the Tp-ec interval AUC using the MedCalc Z test ( $P < 0.05$ ). The most sensitive and specific cutoffs were determined to be  $Tp-ec > 139$  ms and  $Tp-e/QT > 0.31$ . Patients undergoing PCI for STEMI had a significantly higher AUC-ROC for Tp-e/QT compared to other ECG parameters. The most accurate predictor of STR was found when the Tp-e/QT ratio after PCI was combined with the findings of the binary logistic regression analysis.

**Zhao et al.**<sup>(10)</sup> reported an 86% sensitivity and 83% specificity for death with a Tpe/QT ratio  $> 0.29$ , and an 88% sensitivity and 81% specificity for significant adverse cardiac events (MACE) with a Tpe/QT ratio  $> 0.29$ .

Our results showed that there was statistically significant association between Tp-Tec/QT and the endpoints during follow up in hospital.

There was statistically significant association between Tp-Tec/QT and the endpoints during after 6 months follow up. There was no statistically significant association between Tp-Tec and the endpoints after 6 months follow up.

In comparison with our results, **Wang et al.**<sup>(4)</sup> showed that patients with STEMI had their ECGs obtained twelve hours after the onset of a myocardial infarction and again 100 minutes following PCI. The QTc interval was shorter after PCI ( $436 \pm 32.9$  vs. 402

$\pm 30.1$ ,  $P = 0.042$ ). Myocardial infarction substantially delayed Tp-ec interval, which was  $159 \pm 26.9$  before doing PCI versus  $126 \pm 22.5$  after doing PCI, with a  $P$  value  $< 0.001$ , as well as QTc dispersion, which was  $92 \pm 23.5$  before doing PCI versus  $68 \pm 18.4$  after doing PCI, with  $P$  value  $< 0.001$ , and significantly elevated Tp-e/QT ratio was  $0.366 \pm 0.09$  before-PCI versus  $0.292 \pm 0.08$  after-PCI, with  $P$  value  $< 0.001$ , and it was partially recovered by revascularization therapy of PCI.

Previous studies by **Yan *et al.*** <sup>(11)</sup> and **Antzelevitch *et al.*** <sup>(12)</sup> found that the transmural dispersion of cellular repolarization has been shown to correlate well with the Tpe interval in an electrogram obtained across a canine or rabbit left ventricular wedge model (TDR). The Tp-e interval can also be used to evaluate repolarization variability throughout the membrane, from base to top, and around the entire heart. However, like the QT interval, the Tpe interval increases linearly with increasing body mass. In addition, the Tpe interval reduces linearly from 40 to 110 ms as HR rises from 60 to 100 beats per minute (bpm). While HR has varied and the Tp-e/QT ratio has remained very stable between 0.15 and 0.25.

#### STUDY LIMITATIONS

Some research has shown that manual measurements of ECG parameters have low inter- and intra-observer repeatability. Similar errors were seen in the ST-segment and Tp-e measures of STEMI patients. There has been debate about the best way to assess T-wave markers in cases of an elevated ST-segment, as alternative designs of the T-wave have been used to measure these parameters. We measured using techniques established by recent experimental research that provide light on this.

There were several examples where it was too challenging to locate the T-peak, wave's so we didn't include them. Another flaw was that the cellular electrophysiologic mechanisms underlying the clinical findings were inferred.

#### CONCLUSION

TDR in STEMI was electrophysiologically represented by an elevated ST-segment and an elevated Tp-ec. Patients with STEMI after PPCI had an increased STR and Tp-e/QT ratio, both of which predicted arrhythmia and had predictive value for malignant arrhythmia.

**Supporting and sponsoring financially:** Nil.

**Competing interests:** Nil.

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