Tp-e/QT ratio and QT dispersion with respect to blood pressure dipping pattern in prehypertension

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Introduction **Tp-e/QT, the ratio of the interval between the peak and the end of T wave to the QT interval, is a novel index of arrhythmogenesis. We investigate Tp-e/QT and QT dispersion (QTd) in prehypertensive and normotensive patients with different patterns of nocturnal blood pressure dipping.**

Patients and methods **Forty-seven prehypertensive and 37 normotensive adult patients were included. Ambulatory blood pressure monitoring recording was performed and patients were considered to be dipper if nocturnal blood pressure fall was at least 10%; nondipper if it was 0–10%; and reverse-dipper if less than 0%. Tp-e, QT intervals were assessed by 12-lead ECG and Tp-e/QT was calculated using these measurements. QTd is defined as the difference between the maximum and the minimum QT interval of the 12 leads.**

Results **Tp-e/QT was 0.22 ± 0.02 and 0.16 ± 0.01 in prehypertensives and normotensives, respectively ($P < 0.001$), whereas cQTd was 36.1 ± 6.8 and 27.2 ± 5.2 ms ($P < 0.001$). Tp-e and Tp-e/QT were the lowest in the dippers and the highest in the reverse-dippers in the prehypertensive group (Tp-e/QT dipper: 0.21 ± 0.01; nondipper: 0.24 ± 0.02; reverse-dipper: 0.25 ± 0.01; for dipper–nondipper, and dipper–reverse-dipper $P < 0.05$). However, in the normotensive group, dipping status had no effect on Tp-e/QT. There were no significant differences between dippers, nondippers, and reverse-dippers in terms of cQTd both in prehypertensives and in normotensives. There were no associations between left ventricular mass index and Tp-e, Tp-e/QT, and cQTd in both groups.**

Conclusion **Tp-e, Tp-e/QT, and cQTd are increased in prehypertensives compared with normotensives. Tp-e and Tp-e/QT are associated with the dipping status in prehypertensives.** ***Blood Press Monit*** **20:69–73**

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Introduction

Prehypertension is defined as systolic blood pressure (BP) of 120–139 mmHg and a diastolic BP of 80–89 mmHg [1]. It is recognized as a risk factor for development of overt hypertension and its consequences [2] as well as being associated with cardiovascular morbidity and mortality [3]. The prevalence of prehypertension is incontrovertible worldwide; it is reported to be 31% in the USA [4] and 14.5% in our country [5].

Circadian rhythm normally causes a fall in BP in the night time. 'Dipper' is the term to define 10% or more nocturnal BP fall, whereas the reverse is called 'nondipper'. The nondipper BP pattern is known to be associated with worse cardiovascular outcome and end-organ damage [6,7].

Myocardial repolarization has been evaluated in hypertensive patients in various studies, which highlighted that hypertension is associated with prolonged QT intervals and QT dispersion (QTd) [8,9]. Although data on myocardial repolarization in prehypertension are very limited, QT intervals and dispersion were reported to be prolonged in a prehypertensive state independent of left ventricular mass (LVM) [10].

Recently, Tp-e/QT, which is the ratio of the interval between the peak and the end of T wave to the QT interval, was introduced as a novel index of arrhythmogenesis providing an estimate of dispersion of repolarization relative to the total duration of repolarization [11]. It eliminates the confounding effects of variability of heart rate and interindividual variation of the QT interval [11].

We aimed to investigate Tp-e/QT in addition to QTd in prehypertensive and normotensive patients with different patterns of nocturnal BP dipping.

Patients and methods

Prehypertensive or normotensive adult patients who were admitted to the cardiology outpatient clinic for a general cardiovascular check-up were included. Exclusion criteria were typical chest pain, established coronary artery disease, diabetes mellitus or impaired fasting glucose, hypertension defined as BP more than 140/90 in the outpatient physical examination or antihypertensive drug use, rhythm other than sinus, U waves or negative T waves on ECG, moderate to severe valvular disease, hyper/hypothyroidism, any chronic disease, and refusal to provide written informed consent. Eighty-

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four patients (47 prehypertensive and 37 normotensive) who were eligible according to the above-mentioned inclusion and exclusion criteria were enrolled.

Office BP measurements were performed in the sitting position after 5 min of resting using the nondominant arm. BP was calculated as the average of two consecutive BP measurements taken at least 10 min apart by a physician using a mercury sphygmomanometer. Patients were re-evaluated 2 weeks after the first visit and BP measurements were repeated by the same physician at the second visit before enrollment into the study. Data were collected between March 2013 and February 2014. This study was carried out according to the recommendations of the Declaration of Helsinki on biomedical research and it was approved by the institutional ethics committee.

Ambulatory blood pressure monitoring (ABPM) was performed using Cardiospy EC-3H/ABP recorder (Labtech Ltd, Debrecen, Hungary). Each recording began in the morning between 08:00 and 09:00 a.m. Participants were asked to continue their regular activities during the recordings. Day-time was defined as 06:00 a.m.–22:00 p.m. and night time was defined as 22:00 p.m.–06:00 a.m. BP recordings were taken every 15 min throughout the day-time and every 30 min at night. Patients were considered to be dippers if nocturnal BP fall was at least 10%; nondippers if it was 0–10%; and reverse-dippers if less than 0%. The percentage of nocturnal BP variation was calculated using the following formula: $100 \times [1 - (\text{average night SBP}/\text{average day SBP})]$. ABPM recordings excluded 'white-coat hypertension' in the prehypertensive group.

The 12-lead ECG was recorded (KardioPET 600; PETAS, Ankara, Turkey) at a paper speed of 50 mm/s and amplification of 0.1 mV/mm at rest in the supine position. The QT interval was assessed as the time between the first deflection of the QRS complex and the end of the T wave. The slope intercept technique was used to determine the end of the T wave, which is identified as the intercept of the line tangential to the point of maximum T wave down-slope with the isoelectric line [12]. The QT interval was measured in as many 12 leads as possible. Corrected QT (QTc) values were calculated using Bazett's formula: QTc: $QT/\sqrt{R-R}$. The Tp-e interval was measured in the precordial leads [13]. The Tp-e/QT ratio was calculated using these measurements. QTd is defined as the difference between the maximum and the minimum QT interval of the 12 leads. All measurements were performed by two separate cardiologists to avoid error.

Echocardiographic measurements were performed using a VIVID 7 ultrasound (VIVID 7 Pro; GE, Horten, Norway) with a 2.5–3.5 MHz transducer. Parasternal and apical projections were obtained according to the recommendations of the American Society of Echocardiography [14].

Statistical analysis

The SPSS statistical software (SPSS Statistics for Windows, Version 21.0; IBM, Armonk, New York, USA) was used for all statistical calculations. The Kolmogorov–Smirnov test was used to test for a normal distribution. Continuous variables were defined as mean ± SD and categorical variables were defined as percentages. Continuous variables were compared by analysis of variance. The χ^2-test was used for the categorical variables between two groups. Pearson's correlation coefficients were calculated for the correlation between left ventricular mass index (LVMI) and Tp-e, Tp-e/QT, and cQTd. All tests of significance were two-tailed. Statistical significance was defined as P less than 0.05.

Results

The baseline characteristics of the study population are provided in Table 1. There were no differences with respect to age, sex, or BMI. Percentage of dippers, nondippers, and reverse-dippers or the extent of nocturnal BP fall were also similar. Left atrial diameter or left ventricular ejection fraction were similar, but LVMI was higher in prehypertensive patients (mean LVMI 94.0 ± 14.6 vs. 82.0 ± 17.1 g/m^2, $P = 0.001$). Table 2 shows the ECG measures in prehypertensive and normotensive patients. The mean cQTd was significantly higher in the prehypertensive group than the normotensives (36.1 ± 6.8 vs. 27.2 ± 5.2 ms, $P < 0.001$). Tp-e was significantly longer and Tp-e/QT was higher in the prehypertensive group (mean Tp-e: 93.1 ± 7.6 vs. 67.9 ± 8.1 ms, $P < 0.001$; mean Tp-e/QT: 0.22 ± 0.02 vs. 0.16 ± 0.01, $P < 0.001$) (Table 2).

Prehypertensive and normotensive patients were further analyzed with respect to their dipping status (Table 3). Tp-e and Tp-e/QT were significantly lower in the dippers compared with nondippers and reverse-dippers in

Table 1 Baseline characteristics of the study population

	Prehypertensive	Normotensive	*P*
Age (years)	53.9 ± 12.2	48.38 ± 13.8	0.055
Sex (female) (%)	51.1	62.2	0.378
BMI (kg/m^2)	26.5 ± 3	26.1 ± 2.8	0.437
Average 24 h SBP (mmHg)	130.3 ± 4.8	111.4 ± 7.6	< 0.001
Average 24 h DBP (mmHg)	81.9 ± 4.3	72.6 ± 5.3	< 0.001
Average day SBP (mmHg)	131.9 ± 4.7	112.8 ± 7.7	< 0.001
Average day DBP (mmHg)	83.1 ± 4.4	74.4 ± 5.8	< 0.001
Average night SBP (mmHg)	121.4 ± 11	102.6 ± 8.5	< 0.001
Average night DBP (mmHg)	77.5 ± 6.4	67.5 ± 6	< 0.001
Dipping status (%)			0.427
Dipper	48.9	43.2	
Nondipper	27.7	40.5	
Reverse-dipper	23.4	16.2	
Dipping (mmHg)	7.9 ± 8	9.9 ± 7.5	0.510
LA (mm)	31.2 ± 5.0	31.4 ± 4.4	0.891
LVMI (g/m^2)	94.0 ± 14.6	82.0 ± 17.1	0.001
LVEF (%)	63.0 ± 3.5	63.7 ± 3.4	0.359

DBP, diastolic blood pressure; LA, left atrium; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; SBP, systolic blood pressure.
$P < 0.05$ is considered significant.

Table 2 ECG parameters in prehypertensive and normotensive patients

	Prehypertensive	Normotensive	*P*
HR (bpm)	83.3 ± 14.3	88.9 ± 16.2	0.096
cQT max (ms)	434.9 ± 29.2	429.8 ± 42.0	0.536
cQT min (ms)	397.9 ± 28.9	402.6 ± 40.5	0.532
cQT dispersion (ms)	36.1 ± 6.8	27.2 ± 5.2	< 0.001
Tp-e (ms)	93.1 ± 7.6	67.9 ± 8.1	< 0.001
Tp-e/QT	0.22 ± 0.02	0.16 ± 0.01	< 0.001

HR, heart rate; max, maximum; min, minimum.
$P < 0.05$ is considered statistically significant.

Table 4 Pearson's correlation coefficients for the association between LVMI and Tp-e, Tp-e/QT, and cQT dispersion

	r	*P*
Prehypertensive		
LVMI		
Tp-e	0.099	0.509
Tp-e/QT	−0.008	0.960
cQT dispersion	0.378	0.09
Normotensive		
LVMI		
Tp-e	0.145	0.392
Tp-e/QT	−0.170	0.315
cQT dispersion	−0.122	0.471

LVMI, left ventricular mass index.
$P < 0.05$ is considered statistically significant.

the prehypertensive group (Tp-e: 87.3 ± 4.1; 95.8 ± 4.7; 102.1 ± 5.0 ms in dippers, nondippers, and reverse-dippers, respectively, $P < 0.001$ and Tp-e/QT: 0.21 ± 0.01; 0.24 ± 0.02; 0.25 ± 0.01 in dippers, nondippers, and reverse-dippers, respectively, $P < 0.001$). However, there were no significant differences in terms of Tp-e and Tp-e/QT between dippers, nondippers, or reverse-dippers in the normotensive group (Tp-e: 67.5 ± 7.3; 68.4 ± 7.7; 67.5 ± 11.9 ms in dippers, nondippers, and reverse-dippers, respectively, $P = 0.949$ and Tp-e/QT: 0.16 ± 0.02; 0.16 ± 0.02; 0.16 ± 0.01 in dippers, nondippers, and reverse-dippers, respectively, $P = 0.891$).

Although the mean cQTd was higher in prehypertensives than normotensives, there were no significant differences between dippers, nondippers, or reverse-dippers both in prehypertensive and in normotensive patients.

It was searched whether there were any associations between LVMI and electrocardiographic indices of myocardial repolarization. Table 4 shows Pearson's correlation coefficients for the associations between LVMI and Tp-e, Tp-e/QT, and cQTd both in prehypertensive and in normotensive groups. It was shown that there were no significant correlations between LVMI and the above-mentioned indices.

Discussion

In this study, it was shown that prehypertensive individuals have longer Tp-e, higher Tp-e/QT, and longer cQTd than normotensives. In addition, in prehypertensives, Tp-e and Tp-e/QT were significantly augmented in nondippers and reverse-dippers than dippers. However, cQTd was similar in prehypertensive dippers, nondippers, and reverse-dippers despite being longer compared with normotensives.

Inhomogeneity of ventricular repolarization and augmentation of ventricular repolarization dispersion are associated with malignant arrhythmias and has prognostic importance in terms of mortality and sudden cardiac death [15,16].

Although there are studies that highlight increased QTd in hypertension especially in the presence of left ventricular hypertrophy and increased LVM [17], data on arrhythmogenic potential in prehypertensive patients are limited. Doğru *et al.* [10] reported that QTd was prolonged in prehypertensives compared with normotensives and these changes were independent of LVM. However, another study on black Nigerian prehypertensive individuals found that prehypertensives had similar QTd compared with

Table 3 Demographic, hemodynamic, and ECG findings of the prehypertensive and normotensive patients given separately for dippers, nondippers, and reverse-dippers

	Prehypertensive				Normotensive			
	Dipper (*N* = 23)	Nondipper (*N* = 13)	Reverse-dipper (*N* = 11)	*P*	Dipper (*N* = 16)	Nondipper (*N* = 15)	Reverse-dipper (*N* = 6)	*P*
Age (years)	50 ± 12.9	55.5 ± 8.6	60.3 ± 11.9	0.056	48.2 ± 12.5	47.9 ± 14.1	50.2 ± 18.4	0.943
Sex (female) (%)	52.2	61.5	36.4	0.465	50	80	50	0.182
BMI (kg/m^2)	22.6 ± 2.4	25.8 ± 3.4	26.5 ± 2.0	0.882	26.1 ± 2.4	25.8 ± 3.4	26.5 ± 2.0	0.882
Dipping (mmHg)	14.6 ± 3.9	5.4 ± 3.4	−2.7 ± 2.6	< 0.001[a]	15.9 ± 4.4	6.1 ± 2.3	−2.0 ± 2.0	< 0.001[a]
HR (bpm)	82.3 ± 16.2	85.3 ± 11.0	82.9 ± 14.5	0.835	87.2 ± 18.4	93.0 ± 15.7	83.2 ± 8.8	0.398
cQT max (ms)	436.0 ± 33.0	433.2 ± 27.7	434.6 ± 24.3	0.963	432.6 ± 42.6	426.7 ± 40.6	430.2 ± 50.6	0.929
cQT min (ms)	400.5 ± 32.5	397.8 ± 24.6	392.7 ± 27.3	0.772	405.5 ± 42.4	399.5 ± 37.4	402.7 ± 49.2	0.923
cQT dispersion (ms)	35.5 ± 8.0	35.4 ± 6.2	38.2 ± 4.6	0.518	27.1 ± 4.6	27.1 ± 6.4	27.5 ± 4.1	0.988
Tp-e (ms)	87.3 ± 4.1	95.8 ± 4.7	102.1 ± 5.0	< 0.001[a]	67.5 ± 7.3	68.4 ± 7.7	67.5 ± 11.9	0.949
Tp-e/QT	0.21 ± 0.01	0.24 ± 0.02	0.25 ± 0.01	< 0.001[b]	0.16 ± 0.02	0.16 ± 0.02	0.16 ± 0.01	0.891
LVMI (g/m^2)	91.4 ± 14.7	96.4 ± 13.5	96.5 ± 16.1	0.510	80.3 ± 17.6	77.9 ± 16.6	96.8 ± 9.8	0.060

HR, heart rate; LVMI, left ventricular mass index; max, maximum; min, minimum.
[a]Significant difference between dipper–nondipper, dipper–reverse-dipper, and nondipper–reverse-dipper.
[b]Significant difference between dipper–nondipper and dipper–reverse-dipper.
$P < 0.05$ is considered significant.

normotensives [18], although their study participants had far higher LVMI than the participants in the study by Doğru *et al.* [10] and the participants of our study.

Tp-e and Tp-e/QT are recently introduced indices of arrhythmogenesis [11]. The Tp-e interval has been shown to be well correlated with transmural repolarization dispersion [19]. As Tp-e/QT is an estimate of dispersion of repolarization relative to the total duration of repolarization, it places the confounding effect of heart rate aside [11]. Two studies on ST-elevation myocardial infarction (STEMI) patients showed that a higher Tp-e/QT ratio was associated with death, cardiac death, major adverse cardiac events [20], and malignant ventricular arrhythmias [21]. Erikssen *et al.* [22] reported that Tp-e was a strong predictor of mortality during the first year after acute myocardial infarction and it was also strongly associated with fatal cardiac arrhythmias. Demiri *et al.* [23] found that the Tp-e/QT ratio was correlated with increased frequency of arrhythmic events and shock therapy administered to ventricular arrhythmias in heart failure patients who had implantable cardioverter defibrillators [23].

Although there is no clear-cut normal range for Tp-e/QT, Gupta *et al.* [11] defined a range of 0.15–0.25 in normal healthy individuals. In patients with LQTS, a ratio of more than 0.28 was associated with a risk of torsades de pointes [24]. Demiri *et al.* [23] proposed a cut-off value of more than 0.25 in heart failure patients with ICD, and Zhao *et al.* [20] proposed more than 0.29 in patients with STEMI undergoing PCI. Taking these ranges or cut-off values into consideration, the values we report in prehypertensives remain in the normal range despite being higher compared with normotensives.

Demir and Uyan [25] have recently shown that QTd, Tp-e, and Tp-e/QT are prolonged in nondipper hypertensives compared with dipper hypertensives. Karaagac *et al.* [26] have reported that Tp-e, Tp-e/QT, and Tp-e/QTc were higher in nondipper hypertensives with metabolic syndrome than dippers. To our knowledge, there is no study on Tp-e/QT and dipping status in the literature carried out on prehypertensive patients. This study shows that inhomogeneity in ventricular repolarization may occur in the prehypertensive stage, and in addition, non-dipping (and/or reverse dipping) status in prehypertensive patients is associated with further prolongation in Tp-e and Tp-e/QT ratio. A nondipper BP pattern is associated with higher cardiovascular risk as reported elsewhere [6,7]. Left ventricular hypertrophy, left ventricular diastolic dysfunction, autonomic nervous system dysfunction, and increased inflammatory activity play a role in the increase in cardiovascular mortality and morbidity in nondippers [26]. Activation of the sympathetic nervous system and the renin–angiotensin–aldosterone system may induce atrial and ventricular fibrosis and cause cellular electrophysiological alterations [27] that may contribute toward impaired electrocardiographic indices of ventricular repolarization in nondipper and reverse-dipper prehypertensives compared with dippers.

Myocardial hypertrophy in hypertension is known to be associated with prolonged QT maximal duration and QTd [28] that may cause malignant arrhythmias. We have not detected prolonged QT duration, although QTd was prolonged. However, in this study, prehypertensive patients had normal LVMI, although higher than normotensives. We may tentatively conclude that in prehypertension, electrophysiologic alterations precede anatomical changes.

In the literature, there are studies that report normal QT durations and QTd in the presence of myocardial hypertrophy in the athlete's heart [29,30]. This means that the underlying pathology such as autonomic nervous system dysfunction or renin angiotensin system activation [31] may lead to the prolongation of QT duration rather than increased myocardial mass. These studies are supportive of our thesis that electrophysiologic alterations may be responsible from QT alterations rather than anatomical change in LVM. To further clarify this, we also searched for the associations between LVMI and Tp-e, Tp-e/QT, and QTd, and we found no significant correlation. Of course, it should be kept in mind that although prehypertensive patients had higher mean LVMI compared with normotensives, they were in the normal range or mildly higher than normal in the present study.

One of the limitations of the study was the relatively small number of participants. Another point is the cross-sectional design, which precludes inference of causality. Long-term prospective studies are needed if a prolonged Tp-e interval and a higher Tp-e/QT ratio observed in prehypertensive and especially nondipper and reverse-dipper prehypertensive patients compared with normotensives lead to any clinical consequences in terms of arrhythmia.

Conclusion

Novel indices of myocardial repolarization, Tp-e and Tp-e/QT ratio, are increased in prehypertensives compared with normotensives. This increase is more evident in nondipper and reverse-dipper prehypertensives than dippers. Prospective studies are needed to clarify whether these findings will be associated with an increased risk of arrhythmia.

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Conflicts of interest

There are no conflicts of interest.

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