

# The Relation Between Left Ventricular Wall Stress and QT Dispersion in Hypertensive Urgency Patients

*Uç organ hasarı gelişmemiş hipertansif acillerde sol ventrikül duvar gerimi ile QT dispersiyonu arasındaki ilişki*

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Polat DURUKAN,<sup>1</sup> Murat PEKDEMİR,<sup>2</sup> Yılmaz ÖZBAY,<sup>3</sup> Ilgın KARACA,<sup>3</sup> Mustafa YILDIZ,<sup>4</sup> Mehmet AKBULUT,<sup>3</sup> Cemil KAVALCI,<sup>5</sup> Ali RAHMAN<sup>6</sup>

<sup>1</sup>Department of Emergency Medicine, Medicine Faculty of Erciyes University, Kayseri

<sup>2</sup>Department of Emergency Medicine, Medicine Faculty of Kocaeli University, Kocaeli

Departments of <sup>3</sup>Cardiology, <sup>4</sup>Emergency Medicine and <sup>6</sup>Cardiovascular Surgery, Medicine Faculty of Fırat University, Elazığ

<sup>5</sup>Ankara Atatürk State Hospital, Emergency Department, Ankara

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## SUMMARY

**Objectives:** Mortality of hypertensive patients is due to systolic and diastolic dysfunction and hypertrophy of left ventricle, coronary artery disease, arrhythmias and sudden death. Measurement of QT dispersion (QTD) is a non-invasive method for evaluation of heterogeneity of myocardial repolarization and it is thought to accompany arrhythmogenic events. Left ventricular hypertrophy is accepted to be the major factor affecting QTD in hypertensive patients. Aim of this study is to investigate the relation between QTD and left ventricular wall stress caused by an increase in afterload independent from left ventricular hypertrophy in hypertensive urgency patients.

**Materials and Methods:** Standart 12-lead surface electrocardiographic records of 76 patients matching inclusion criteria were taken in hypertensive period and period in which mean blood pressure lowered 10% by Na-nitroprussid and then QTD and corrected QT dispersion (QTcD) were calculated. Left ventricular geometric patterns were determined by echocardiographic measurements.

**Results:** Mean age of the patients was 57.29±11.3 and 68.4% of them were women. In the hypertensive period mean systolic blood pressure (SBP), QTD and QTcD were 192.11±20.2 mmHg, 45.79±14.90 msec and 54.78±17.96 msec and in lowered blood pressure (LBP) period they are 149.08±11.9 mmHg, 29.47±13.65 msec and 34.06±15.73 msec respectively (p<0.001). In 50% of patients concentric hypertrophy and in 11.8% of patients normal ventricular geometry were detected. According to left ventricular geometry mean QTD and QTcD were similar between the groups in hypertensive and LBP periods. Although mean QTDs were similar (p=0.058) in normal left ventricular geometry group in both hypertensive and LBP periods, mean QTcDs were different (p=0.036). In patient groups having other left ventricular geometric patterns mean QTD and QTcD were different in hypertensive and LBP periods (for each p<0.05). In hypertensive patients when blood pressure is high, due to an increase in afterload ventricular wall stress increases and due to myocardial ischemia QTD and QTcD lengthen, when the blood pressure comes to normal QTD and QTcD shorten.

**Conclusion:** As a result it is thought that when blood pressure changes acutely, QTD and QTcD change due to left ventricular wall stress.

**Key words:** Hypertension; QT dispersion; ventricular wall stress; ventricular.

## ÖZET

**Giriş:** Hipertansif bireylerde mortalite nedenleri sol ventrikülün sistolik ve diyastolik disfonksiyonu, hipertrofisi, koroner arter hastalığı, aritmiler ve ani ölümdür. QT dispersiyonu (QTD) ölçümü miyokardial repolarizasyon heterojenitesinin değerlendirilmesinde noninvaziv bir yöntem olarak kullanılmakta ve aritmik olaylarla birlikte olduğu düşünülmektedir. Hipertansif hastalardaki sol ventrikül hipertrofisi QTD değişimindeki major etken olarak kabul görmektedir. Çalışmanın amacı hipertansif urgency hastalarında, sol ventrikül hipertrojisinden bağımsız olarak, afterload artışına bağlı sol ventrikül duvar gerimi ve QT dispersiyonu arasındaki ilişkiyi araştırmaktır.

**Gereç ve Yöntem:** Çalışma koşullarına uygun hastalarda hipertansif dönemde ve Na-nitroprussid ile ortalama kan basıncı %10 düşürülmüş dönemde standart 12 derivasyonlu yüzey elektrokardiyografi kaydı alınarak QTD ve QTc dispersiyonu (QTcD) hesaplandı. Ekokardiyografi ile sol ventrikül geometrik yapısı saptandı.

**Bulgular:** Çalışmaya alınan 76 hastanın yaş ortalaması 57.29±11.3, %68.4'ü kadındı. Hipertansif dönemde ölçülen sistolik kan basıncı (SKB) ortalama 192.11±20.2 mmHg, QTD 45.79±14.90 msn ve QTcD 54.78±17.96 msn, kan basıncı düşürülmüş dönemde ortalama SKB 149.08±11.9 mmHg, QTD 29.47±13.65 msn ve QTcD 34.06±15.73 msn saptandı (p<0.001). Hastaların %50'sinde konantrik hipertrofi, %11.8'inde normal ventrikül

## Correspondence (İletişim)

Polat DURUKAN, M.D.

Department of Emergency Medicine,  
Medicine Faculty of Erciyes University, Kayseri.  
Tel: +90 - 352 - 437 49 01 / 22332  
Fax (Faks): +90 - 352 - 437 52 73  
e-mail (e-posta): polatdurukan@gmail.com

geometrisi saptandı. Sol ventrikül geometrisine göre gruplar arasında hipertansif ve kan basıncı düşürülmüş dönemlerdeki QTD ve QTcD ortalamaları benzerdi. Sol ventrikül geometrisi normal bulunan hastalarda hipertansif ve kan basıncı düşürülmüş dönemdeki QTD ortalamaları benzer iken ( $p=0.058$ ), QTcD ortalamaları farklı bulundu ( $p=0.036$ ). Diğer sol ventrikül geometrilerine sahip hasta gruplarında hipertansif ve kan basıncı düşürülmüş dönemdeki QTD ve QTcD ortalamaları farklıydı (her biri için  $p<0.05$ ). Hipertansif hastalarda kan basıncı yüksek iken afterload artışı ile ventrikül duvar gerimi artmakta, miyokardial iskemi nedeniyle QTD ve QTcD uzamakta ve kan basıncının normale dönmesi ile QTD ve QTcD kısalmaktadır.

**Sonuç:** Akut kan basıncı değişikliklerinde sol ventrikül duvar gerimi etkisiyle QTD ve QTcD'nin değiştiği düşünülmektedir.

**Anahtar sözcükler:** Hipertansiyon; QT dispersiyonu; ventrikül duvar gerimi; ventrikül hipertrofisi.

## Introduction

Hypertension is described as systolic blood pressure (SBP) more than 140 mmHg and diastolic blood pressure (DBP) more than 90 mmHg.<sup>[1,2]</sup> The seventh report of the joint national committee on prevention, detection, evaluation and treatment of high blood pressure (JNC-VII) guidelines for the management of hypertension classified hypertensive crises as hypertensive urgency (HTU) and hypertensive emergency (HTE). According to these guidelines in HTE, acute or continuing target organ damage accompanies serious elevations in blood pressure. In HTU there is no target organ damage, but integrity of cardiovascular system may be threatened at that time.<sup>[1]</sup> In HTU patients aim is to decrease blood pressure in 24 hours and follow up from the other day; but in HTE aim is to decrease blood pressure immediately to prevent or limit target organ damage. In HTU patients blood pressure can be decreased in 12-24 hours by oral antihypertensive agents. This treatment approach necessitates close follow up of hypertensive patient and reevaluation of blood pressure in 24 hours.<sup>[1,3,4]</sup> Such a follow up is usually not possible under emergency department situations. For this reason, to decrease the blood pressure of HTU patients sufficiently, intravenous antihypertensive agents like sodium nitroprusside and urapidil may be preferred. It was proven that this therapeutic approach is safe.<sup>[5]</sup>

It was informed by large population studies that there is high correlation between high blood pressure and stroke, myocardial infarction, heart failure, renal diseases and mortality. If blood pressure is not controlled sufficiently, overall mortality and morbidity, risk of development of serious cardiovascular, renal and cerebrovascular disease increase.<sup>[6,7]</sup> 50% of the the mortality of hypertensive population is due to hypertensive heart disease and causes of mortality are systolic and

diastolic dysfunction of the left ventricle, hypertrophy of left ventricle, coronary artery disease, arrhythmias and sudden death.<sup>[7]</sup> QT dispersion (QTD) measurement from the surface electrocardiography is being used as a safe and non-invasive method for evaluation of myocardial repolarization heterogeneity and determination of probable risk of arrhythmia. It is thought that long QTD is seen in myocardial diseases and accompanies increased incidence of arrhythmic events.<sup>[8,9]</sup>

We aimed to search the relation between left ventricular wall stress and QTD and QTcD in hypertensive and LBP period in hypertensive crisis without target organ damage (HTU).

## Methods

The study was conducted between September 2002 and February 2003 in Firat University Emergency Department in hypertensive patients. Permission was obtained from university ethic committee to conduct the study (26.09.2002, 8/2). The informed-written consent form was obtained from the patients matching inclusion criteria.

### Inclusion criteria

- Patient acceptance
- Diastolic blood pressure  $\geq 110$  mmHg or systolic blood pressure  $\geq 180$  mmHg
- Patients older than 18 years
- Absence of target organ damage:<sup>[10]</sup>
  - Cerebral infarction
  - Acute pulmonary edema
  - Hypertensive encephalopathy
- Acute coronary syndrome
- Renal failure
- Dissection of aorta
- Cerebral hemorrhage
- Preeclampsia and eclampsia

### Exclusion criteria

- Blood pressure  $< 140/90$  mmHg during the second measurement at 10th minute of admission
- Presence of target organ damage
- Allergy to nitroprusside
- Pregnancy
- Refusal of patient
- Drugs that affect QT interval (Quinidine, digoxine)
- Presence of disease that affect QT interval (Chronic renal failure, congestive heart failure, etc)
- EF  $< 40\%$

The blood pressure measurements were performed in the sitting position by aneroid machine and Korotkoff phase 1 and 5 sounds were accepted as systolic and diastolic blood pressure respectively. The second blood pressure measurement was performed after 10 minute resting and last measurement was before discharge.

Twelve lead electrocardiogram was obtained during the hypertensive period. ECG's were registered by Nihon Kohden Cardiofax ECG-9020K device. Sodium nitroprusside infusion with a dose of 0.3 µg/kg/min was started in all patients and titrated according to patient response. The treatment was stopped when the mean blood pressure was decreased by 10% or systolic blood pressure by 30%. A second 12 lead ECG was obtained when blood pressure was decreased. A blinded investigator calculated the QTD and QTcD values from the ECG's of both hypertensive and LBP period.

QT intervals were measured from at least 7 leads than QTmax and QTmin values were found. In addition, heart rate adjusted QTcmax and QTcmin values were calculated by Bazett formula.

$$QTD = QT_{max} - QT_{min} \text{ and}$$

$$QTcD = QT_{cMax} - QT_{cMin}$$

Transthoracic echocardiography was applied to patients to search the left ventricular hypertrophy (LVH) within 24 hours. Two dimensional B and M-mode echocardiography from standart apical 4 chambers and parasternal long axis view were done by the same cardiologist (Acuson® Sequoia 512 3.5 mHz 3V2c probe). Diastolic interventricular septal (IVSWT) and posterior wall thickness (LPWT), end-systolic (LVESD) and end-diastolic diameters (LVEDD) were measured. Left ventricular mass (LVM) and mass index (LVMI) were calculated by Penn formula.<sup>[11,12]</sup>

$$LVM = 1.04 \times (IVSTd + LVEDD + PWTd)^3 - (LVEDD)^3 - 13.6 \text{ gr}$$

$$\text{Body surface area (BSA)} = [(Weight \times 4) + 7] / (Weight + 90)$$

$$LVMI (g/m^2) = LVM / BSA$$

Relative wall thickness (RWT) was calculated by “(RWT)= 2 x LPWT / LVEDD” formula.<sup>[13]</sup> The patients were divided into 4 groups according to type of the left ventricular hypertrophy: Normal geometry (normal LVMI and normal RWT), concentric remodeling (normal LVMI and increased RWT), concentric hypertrophy (increased LVMI and increased RWT) and eccentric hypertrophy (increased LVMI and normal RWT) (Fig. 1).<sup>[12]</sup>

Quantitative findings were expressed as mean±standart deviation whereas qualitative ones as %. Kolmogorov Simirnov test was used to test the normal distribution of the quantitative findings. p<0.05 was accepted as statistically significant. Multiple regression models were established to show the importance of factors on QTD and QTcD during hypertensive and LBP periods.

**Results**

The study was performed with 76 HTU patients matching inclusion criteria admitted to Firat University Faculty of Medicine Emergency Department between October 2002 and February 2003. During Sodium nitroprusside treatment no complication was seen. Clinical features of the patients can be seen on Table 1.

After admission to emergency department, mean systolic blood pressure (SBP), mean diastolic blood pressure (DBP) and mean mean blood pressure (MBP) of the patients who had had a rest for 10 minutes were 192.11±20.2 mmHg, 114.54±14.8 mmHg and 140.39±14.4 mmHg respectively. Mean SBP, mean DBP and mean MBP measured just before the discharge were 149.08±11.9 mmHg, 89.08±8.8 mmHg and 109.08±8.8 mmHg respectively (p<0.001 for each).

Mean QT, QTc, QTD and QTcD measured during hypertensive and LBP period are on Table 2. Mean QTD and QTcD during hypertensive period were significantly high statistically (p<0.001 for each).

After echocardiographic evaluation QTD and QTcD values of the patients during hypertensive and LBP period in respect

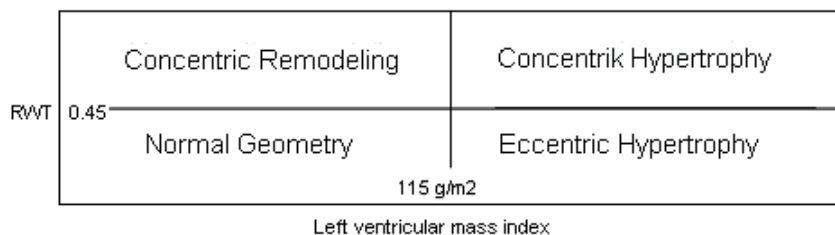


Fig. 1. Left ventricular geometry according to RWT ve LVMI.

**Table 1.** Clinical features of the patients.

Age (years)	57.29±11.3
Sex (M/F)	24 (%31.6) / 52 (%68.4)
Hypertension history (n of patients)	61 (%80.3)
Diabetes Mellitus history (n of patients)	14 (%18.4)
Newly diagnosed hypertension (n of patients)	15 (%19.7)
Hypertension history in family (n of patients)	33 (%43.4)
Use of antihypertensive agents (n of patients)	52 (%68.4)
BMI	27.91±3.8
BSA (m <sup>2</sup> )	1.85±0.15
LVMI (gr/m <sup>2</sup> )	136.11±37.3
RWT (mm)	0.50±0.09
Urea (mgr/dL)	32.69±9.8 (0-50)
Creatinine (mgr/dL)	0.84±0.2 (0.60-1.20)
Sodium (mmol/L)	136.49±3.8 (135-150)
Potassium (mmol/L)	3.93±0.4 (3.50-5.30)

M: Male; F: Female; BMI: Body mass index; BSA: Body surface area; LVMI: Left ventricle mass index; RWT: Relative wall thickness.

of left ventricular geometry and Penn formula are on Table 3.

Concentric hypertrophy was detected in 50% of the patients. In respect to left ventricular geometry, mean QTD and QTcD were similar in hypertensive and LBP period between the groups ( $p>0.05$  for each). In normal left ventricular geometry patients while mean QTD's were similar statistically in hypertensive and LBP period ( $p=0.058$ ), mean QTcD's were different statistically ( $p=0.036$ ). In patient groups having other left ventricular geometric patterns mean QTD and QTcD's were different statistically in hypertensive and LBP period ( $p<0.05$  for each).

Poor linear relation (multiple R: 0.230,  $p=0.046$ ) was found by multiple regression analysis (model 1) which evaluated the factors that may have effect on QTD in the hypertensive period. Type of LVH was detected as the effective variable of the model ( $\chi^2$ : 0.230,  $p=0.046$ ). Ineffective variables were SBP ( $p=0.937$ ), DBP ( $p=0.985$ ), MBP ( $p=0.960$ ), duration of hypertension ( $p=0.995$ ), use of antihypertensive agent ( $p=0.996$ ). Linear relation was not found (multiple R: 0.232,

$p=0.660$ ) by multiple regression analysis (model 2) that evaluated the factors that may have effect on QTcD in hypertensive period.

Poor linear relation (multiple R: 0.237,  $p=0.039$ ) was found by multiple regression analysis (model 3) which evaluated the factors that may have effect on QTD in the LBP period. MBP was the effective variable ( $\chi^2$ : -0.237,  $p=0.039$ ). Ineffective variables were duration of hypertension ( $p=0.995$ ), type of LVH ( $p=0.997$ ), use of antihypertensive agent ( $p=0.987$ ), SBP ( $p=0.299$ ) and DBP ( $p=0.136$ ). Poor linear relation was found (multiple R: 0.245,  $p=0.033$ ) by multiple regression analysis (model 4) that evaluated the factors that may have effect on QTcD in LBP period. MBP was the effective variable ( $\chi^2$ : -0.245,  $p=0.033$ ). Ineffective variables were duration of hypertension ( $p=0.995$ ), type of LVH ( $p=0.997$ ), use of antihypertensive agent ( $p=0.987$ ), SBP ( $p=0.299$ ) and DBP ( $p=0.136$ ).

## Discussion

In this study we aimed to investigate the relation between QTD and left ventricular wall stress by obtaining a controlled decrease in blood pressure by sodium nitroprusside in hypertensive urgency patients admitted to emergency department. During literature review methodologically similar studies were not found. After the study it was thought that QTD and QTcD were lengthened in high blood pressure period, when the blood pressure was decreased acutely in a controlled manner QTD and QTcD were decreased. This effect was independent of sex and more obvious in hypertrophic ventricle. Probable factor may be the ventricular wall stress which depends on the increase in afterload.

It was reported that blood pressure can be decreased in 12-24 hours, but patients should be followed up closely and blood pressure must be reevaluated in 24 hours. Because such a follow up is usually not possible in the emergency departments, to sufficiently decrease blood pressure of HTU patients intra-

**Table 2.** Mean QT, QTc, QTD ve QTcD in hypertensive and LBP periods.

Mean±SD (msc)	Hypertensive period	LBP period	p
QTmax	410.26±36.2	409.74±33.5	NS
QTmin	364.47±36.2	380.26±31.9	<0.001
QTcmax	489.84±36.1	472.52±34.2	<0.001
QTcmin	435.06±36.3	438.45±31.5	NS
QTD	45.79±14.90	29.47±13.65	<0.001
QTcD	54.78±17.96	34.06±15.73	<0.001

QTmax: QT maximum; QTmin: QT minimum; QTcmax: QTc maximum; QTcmin: QTc minimum; QTD: QT dispersion; QTcD: QTc dispersion.

**Table 3.** Mean QTD ve QTcD according to left ventricular geometry.

	Normal geometry n=9 (11.8%)	Concentric remodeling n=12 (15.8%)	Concentric hypertrophy n=38 (50%)	Eccentric hypertrophy n=17 (22.4%)	p
QTD1 (msc)	37.78±15.6	43.33±7.8	46.84±14.9	49.41±17.5	0.337
QTD2 (msc)	24.44±8.8*	25±12.4†	32.11±15.1‡	29.41±12.5‡	0.293
QTcD1 (msc)	45.59±19.3	53.93±11	55.5±17.8	58.65±21	0.624
QTcD2 (msc)	28.29±10.3**	28.83±14.7††	37.07±17.2‡	34.10±14.6‡	0.376

QTD: QT dispersion; QTcD: QTc dispersion; 1: Hypertensive period; 2: LBP period; \*p=0.058; \*\*p=0.036; †p=0.005; ††p=0.003; ‡p≤0.001 (Between QTD1 - QTD2, QTcD1 - QTcD2).

venous antihypertensive agents are safe enough.<sup>[15]</sup> In this study we aimed to put forward if the wall stress caused QTD or not; so, to obtain a standardized controlled drop in blood pressure, it was decreased to have decrease of 10% of beginning MBP or 30% of beginning SBP by sodium nitroprusside infusion. We did not have complication in any of the patients.

It was reported that increased LVM in hypertensive patients causes lengthening in QTD and QTD is longer in patients with LVH than without LVH<sup>[14]</sup> and due to nonhomogenous ventricular repolarization, hypertensive patients have longer QT and QTc durations than healthy persons.<sup>[11]</sup> LVH is microscopically characterized by myocyte hypertrophy and increase in collagen interstitial matrix. Myocyte hypertrophy can cause lengthening of action potential duration. Interstitial fibrous tissue increase can cause a decrease in amplitude of action potential and membrane potential and shortening of action potential duration or electrical stagnation.<sup>[15]</sup> For these reasons changes in variable regions of the ventricle may increase QTD and this may end with reentry circuit and ventricular arrhythmia.<sup>[16]</sup> It was also reported that after 10 year follow up of 59 adult patients with essential hypertension, SBP, DBP and QTcD were higher in patients having sudden cardiac death than alive patients; but, in spite of increased QTcD in hypertensive patients this finding was not together with risk of sudden death, only LVH and high degrees of ventricular arrhythmia caused sudden cardiac death risk.<sup>[17]</sup>

There is contradictory information about QTD and QTcD relation with blood pressure and ventricular geometry. It is reported that there is a relation between QTD, QTcD and SBP and LVMI.<sup>[18]</sup> In the hypertensive patients having LVH on ECG, not hypertension but LVMI and LVH are related to QT duration and QTD.<sup>[19,20]</sup> It was also reported that in hypertensive patients having left ventricular concentric hypertrophy, QTD is more than patients with normal geometry and after treatment there is a decrease in both ventricular hypertrophy and QTD.<sup>[21]</sup> Contrary to this, mean QTD and QTcD are similar in all of the patients having four different ventric-

ular geometry.<sup>[11,21]</sup> Also we found that mean QTD and QTcD were similar in patients having four different ventricular geometry. When blood pressures of the patients decreased by 10% of MBP, in the normal left ventricular geometry patients there was a decrease on the border of statistically significance; in the patients having other geometrical patterns there was a significant decrease in QTD and QTcD. The reason of the difference may be the fact that number of patients having normal geometry was smaller than patients with other geometric patterns. In addition, hypertrophic myocardium may give a more obvious response in repolarization heterogeneity when blood pressure changes acutely. This data make us think that effective factors in the decrease of QTD may be sodium nitroprusside or decrease of wall stress due to decreased blood pressure according to Laplace Law.

In the SILVHIA study where Irbesartan and atenolol were compared with each other for the effect on cardiac repolarization by using QTD measurement in LVH, it was reported that Irbesartan had decreased QTD and measured ventricular repolarization heterogeneity independent of LVM, blood pressure heart rate and additional antihypertensive treatment; increased myocardial stress due to increase in afterload, decreased electrical threshold of myocytes and caused an increase in risk of spontaneous depolarization.<sup>[22]</sup> It is also put forward that Irbesartan decreases QTD by a direct effect.<sup>[23]</sup> In hypertensive patients treated with enalapril for 7 years improvement in LVH, QTD and QTcD was shown. Additionally, decrease in QTD may be related to return of structural and functional changes in coronary microcirculation caused by hypertension and it was shown in human and animal studies that treatment with ACE inhibitors may improve this ischemia.<sup>[24]</sup> In a study in which Felodipin and Ramipril were used as antihypertensive agent, when the blood pressure was decreased QTD improved. Improvement in QTD was said to be due to regression of LVMI, not due to direct effect of the drugs because there was no increase in QTD and QTcD during washout period of drugs.<sup>[16]</sup> In our study 68% of the patients were using antihypertensive agents

but in spite of medical treatment blood pressure was not under control. In these patients we detected a decrease in QTD and QTcD with a 10% acute drop in MBP. We think that this shortening of QTD and QTcD is due to sodium nitropruside that we used or decrease of blood pressure, not due to direct effect of antihypertensive agents that have been used by the patients.

It was reported that QTcD was longer in patients with high blood pressure due to increased sympathetic activity than those patients without elevated blood pressure during early morning and QTcD was similar in periods when blood pressure was not different. They suggested that electrophysiologic changes resulted from afterload increase, local myocardial ischemia which causes electrical instability and LVH would be the reason.<sup>[25]</sup> QTD was lengthened independent of heart rate by afterload elevation with Fenilephrin administration in healthy individuals, this was explained by Laplace Law in which elevated afterload increases the wall stress.<sup>[26]</sup> In our study QTD and QTcD were longer in hypertensive period independent of the circadian rhythm than LBP period. QTD and QTcD may be decreased due to decrease in blood pressure hence decrease in left ventricular wall stress. Because wall stress is a major determinant of myocardial oxygen demand and it refers to the force that applied to unit myocardial area ( $\text{g}/\text{cm}^2$ ). Left ventricle systolic pressure (P), radius of left ventricular cavity (r) and LVWT are the factors that determine the wall stress according to Laplace Law.<sup>[27]</sup> Here, radius and wall thickness are constant, pressure is variable. Left ventricular wall stress increases proportionally with blood pressure for any diameter. It was hypothesised that sudden increase in blood pressure increases wall stress and this results in subendocardial ischemia as in coronary artery disease.<sup>[28,29]</sup> QTD lengthens in ischemic conditions like myocardial infarction.<sup>[30]</sup> These findings suggest that QTD may improve by antihypertensive therapy because oxygen demand decreases with decreased systolic blood pressure and myocardial perfusion improves due to decreased compression of perforator arteries. Because QTD and QTcD were similar between groups during both hypertensive and LBP periods when the patients were grouped according to left ventricular geometry, QTD and QTcD changes in healthy and hypertrophic myocardium may result from acute blood pressure changes and resultant wall stress changes. And we think that probable mechanism is myocardial ischemia caused by increased wall stress.

As a result QTD and QTcD are lengthened in HTU patients and shorten when blood pressure decreased. Due to left ven-

tricular wall stress, acute blood pressure changes should also be taken into account in shortening of QTD and QTcD which is more obvious in patients having hypertrophic left ventricle than patients having normal ventricular geometry. The relation of hypertension, left ventricular wall stress and QTD with direct indicators of ventricular wall pressure increase can be explained better with advanced studies.

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