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QT Dispersion in Single-Vessel Coronary Artery Disease: Is There Relation Between QT Dispersion and the Diseased Coronary Artery or Lesion Localization?

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İZOLE TEK DAMAR KORONER ARTER HASTALIĞINDA QT DİSPERSİYONU: TUTULAN DAMAR YA DA LEZYON YERLEŞİMİ İLE QT DİSPERSİYONU ARASINDA İLİŞKİ VAR MI?

ÖZET

İskemik ataklar veya akut miyokard infarktüsü sırasında QT dispersiyonunun (QTD) arttığı gösterilmiştir. Bununla birlikte, tutulan koroner arter ya da lezyon yerleşimi ile QTD arasında ilişki bulunup bulunmadığı konusunda yeterli bilgi yoktur. Bu çalışmada, izole tek damar hastalığı saptanan ve daha önceden miyokard infarktüsü bulunmayan hastalarda, QTD ile damar tutulumu ya da lezyon yerleşimi arasındaki ilişki egzersiz stres testi (EST) ile araştırılmıştır.

Çalışmaya, KAH şüphesi nedeniyle önce EST, daha sonra koroner anjiyografi uygulanan ve koroner arterleri normal bulunan 53 birey ile tek damar hastalığı saptanan 119 hasta alınmıştır. QT ölçümleri her iki grupta test başlangıcı ve bitiminden 2 dakika sonraki dönemlerde (rec-2) yapılmıştır. Tek damar hastalığı olan gruplarda [sol ön inen arter (LAD), sirkumfleks arter (CX), sağ koroner arter (RCA)] dinlenim halindeki düzeltilmiş QT dispersiyonunun (QTcD) kontrol grubuna göre anlamlı derecede yüksek olduğu gözlenmiştir (kontrol grubunda 33±12 ms, LAD grubunda 49±13 ms, CX grubunda 45±10 ms ve RCA grubunda 44±11 ms, p<0.05). Rec-2 döneminde yapılan ölçümlerde ise QTcD değerlerinin yine kontrol grubuna oranla anlamlı derecede yükselmiş olduğu (kontrol grubunda 38±12 ms , LAD grubunda 68±18 ms, CX grubunda 59±17 ms ve RCA grubunda 61±18 ms, p<0.005), bununla birlikte QTcD ile tutulan damar ya da lezyon yerleşimi arasında herhangi bir ilişki bulunmadığı saptanmıştır. Ayrıca rec-2 dönemindeki QTD'ndaki artışlar ile ST segment depresyonu arasında yakın bir korelasyon olduğu da gözlenmiştir (r=0.706, p<0.001)

Sonuç olarak, izole tek damar hastalıklarında tutulum gözlenen damara ya da proksimal ve distal yerleşime göre QTD artışının anlamlı bir farklılık göstermediği, tek damar hastalığı bulunan hastaların kontrol grubuna göre daha yüksek bazal QTD değerlerine sahip olduğu ve bu farkın egzersiz ile daha fazla arttığı gözlenmiştir. Bu bulgularımız, QT dispersiyonu üzerine koroner arter hastalığının yaygınlığı ve ciddiyetinden çok, bölgesel iskeminin etkili olduğunu savunan görüşleri desteklemektedir.

Anahtar kelimeler: *Bölgesel iskemi, izole tek damar koroner arter hastalığı, QT dispersiyonu*

QT dispersion (QTD), defined as the difference between the longest and the shortest QT interval on the standard 12-lead electrocardiogram (ECG) has been suggested to reflect regional variation in ventricular repolarization and cardiac electrical inhomogeneity [1,2]. Conditions such as long QT syndrome [3], hypertrophic cardiomyopathy [4], acute myocardial infarction [5] and congestive heart failure [6] have been shown to cause an increase in QTD, the risk of serious arrhythmias and sudden death. This parameter has also been measured in patients with coronary artery disease (CAD). In studies performed with atrial pacing [7,8] or exercise stress test (EST) [9-11] it has been shown that acute ischemia caused a significant increase in QT dispersion. Lowe et al. reported that stable triple-vessel CAD with or without inducible ischemia was associated with wider QT dispersion [12].

However, no extensive data on single-vessel CAD and QT dispersion and the relation between the coronary artery involved or the lesion localization and the degree of the QTD changes during rest and exercise are available. Therefore, we designed this study to clarify the QTD changes during rest and exercise and to examine the relation of QTD with the coronary artery involved and lesion localization in patients with single-vessel CAD.

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METHODS

Patients

We studied 172 patients retrospectively, 119 with single-vessel CAD (59 with left anterior descending artery (LAD), 36 with circumflex artery (CX), 34 with right coronary artery (RCA) disease (mean age 51±9 years, 21 women and 98 men), and 53 patients without coronary artery disease (mean age 48±12 years, 14 women and 39 men). Subjects with evidence of myocardial infarction, congestive heart failure, valvular heart disease, left ventricular hypertrophy, severe hypertension, uncontrolled diabetes mellitus, atrial fibrillation, left or right bundle branch block, patients receiving class I or class III antiarrhythmic agents were excluded from the study. The patient characteristics are summarized in Table 1.

Exercise stress test protocol

All patients underwent exercise stress test (EST) with modified Bruce protocol initially. 12-lead electrocardiograms were recorded at rest and after each stage of the exercise protocol with Quinton Q4500 treadmill equipment at a paper speed of 25 mm/sec. All medications except nitrates and acetyl-salicylic acid were stopped before EST for at least >5 half-lives. The test ended in the presence of the following criteria; angina pectoris, ST-segment depression, serious arrhythmias, reaching peak heart rate, systolic arterial pressure above 220 mmHg or severe hypotension. Exercise test responses were considered positive and the test was stopped when there was >1 mm additional horizontal or downsloping ST segment depression at 80 ms after J point. The ECG recordings at rest and at the 2nd minute of recovery period (rec-2) were used for comparison of QT interval changes.

Coronary angiography

All patients underwent coronary angiography after EST with standard techniques for the reason of having an abnormal EST or for ruling out the diagnosis of CAD. The coronary angiograms were reviewed by an expert observer blinded to the result of the study. Significant CAD was defined when ≥70% luminal diameter narrowing of a major coronary artery in any projection. The lesion localization were then classified in 3 parts in LAD and CX groups as proximal, mid (between 1. and 2. diagonal branch of LAD and between 1. and 2. obtuse marginal branch of CX respectively) and distal part. The RCA lesions were also classified as proximal (above right ventricular branch) and distal (below right ventricular branch).

QT measurements

All the QT interval measurements were made on the rest and post-exercise period (2nd minute of the recovery period) electrocardiograms by a single observer who is blinded to patient's data. After magnifying the ECG's 2 fold by using a magnifying glass, QT intervals were measured from the beginning of the inscription of the QRS complex to the point at which the T wave returned to the isoelectric line from the three consecutive beats. If a U wave was present, the termination of the T wave was defined as the nadir between the T and U waves. Leads where the T wave ends or T wave morphology could not be clearly observed was excluded from analysis. The PR segment was taken as the baseline to solve the difficulty in identifying the end of the T wave in the presence of ST-segment depression mostly during post-exercise period measurements. A minimum of 9 ECG leads (mean 10.2) was analyzed. ECG's with fewer than 8 measurable leads were excluded from the study.

QT parameters

QT dispersion (QTD) was calculated as the difference between the longest (QTmax) and the shortest QT (QTmin) intervals recorded. The QT interval was corrected (QTc) for the heart rate by using Bazett's formula (QTc= QT/ square root of R-R interval in seconds). Corrected QT dis-

Table 1. Patient Characteristics for the Four Groups

	Control (n=53)	LAD (n=59)	CX (n=36)	RCA (n=34)	p value
Age (years)	48±12	53±9	51±10	50±11	NS
Men/Women	42/11	41/8	29/7	28/6	NS
Risk Factors					
Smoking	17 (32%)	29 (49%)*	16 (47%)*	18 (53%)*	<0.005
Hyperchlosterolemia	8 (15%)	31 (53%)*	15 (42%)*	17 (50%)*	<0.001
Systemic hypertension	8 (15%)	13 (22%)	7 (19%)	6 (18%)	NS
Diabetes mellitus	7 (13%)	11 (19%)	8 (2%)	7 (21%)	NS
Medications					
Nitrates	11 (21%)	52 (88%)*	29 (81%)*	25/34 (74%)*	<0.0001
B-blocker#	6 (11%)	12 (20%)	9 (25%)	7 (21%)	NS
Calcium-channel blockers#	7 (13%)	18 (31%)*	7 (19%)	6 (18%)	<0.01

LAD: patient group with left anterior descending artery obstruction ≥%70
CX: patient group with circumflex artery obstruction ≥%70
RCA: patient group with right coronary artery obstruction ≥%70
** Statistically different from control group.*
 # Stopped before exercise stress test for at least >5 half-lives.

persion (QTcD) was defined as the difference between the maximum and the minimum QTc for a given heart rate. The percentage changes in QTD in response to increasing heart rate was defined as the percent change in QTD (%QTD). It was calculated as the difference in the QTD at rest and post-exercise period divided by the difference between the respective RR intervals multiplied by 100 [9]. This index reflects the percent change in QT interval dispersion for each 100 sec decrease in the RR interval. Similar values were also calculated for QTcD.

Intraobserver variability in measurements of QT dispersion

Intraobserver variability in measurements of QT dispersion was determined from blinded repeat interpretation of 45 randomly selected ECG's. The mean difference between the first and second measurements of the same observer was 9.4±4.2 ms and linear regression analysis yielded minimal intraobserver variation with a correlation coefficient of 0.92 ($p<0.0005$).

Statistical analysis

Data are given as mean values±SD. One-way analysis of variance (ANOVA) was used to determine the difference between the four groups (Control, LAD, CX, RCA) and Chi-squared or unpaired t test were used for a difference between two groups. The relation between QT dispersion and lesion localization in the related coronary artery was evaluated by Pearson's correlation analysis. A p value of <0.05 was considered statistically significant.

RESULTS

Study population

As shown in Table 1, there was no statistically significant difference in age and gender between all four groups. There were fewer patients who smoked in the control group than in the other groups ($p<0.005$). Approximately half of the patients with single-vessel disease were found to have increased cholesterol levels (above 240 mg/dl) when compared to control group ($p<0.001$). Nitrate and calcium-channel blocker usage was also higher in single-vessel disease groups when compared to the control group ($p<0.01$).

QTD and QTcD at rest

QTD at rest ranged from 10 to 60 ms (mean 31±10 ms) in the control group, from 30 to 90 ms (mean 47±14 ms) in the LAD group, from 25 to 85 ms (mean 41±11 ms) in the CX group, from 25 to 90 ms (mean 43±9 ms) in the RCA group with a significant difference between the control and other three groups ($p<0.05$; Table 2). QTcD at rest was 33±12 ms in the control group, 49±13 ms in LAD group,

Table 2. QT Interval Parameters (ms) at Rest and Post-exercise Period

		Control (n=53)	LAD (n=59)	CX (n=36)	RCA (n=34)	Cont vs. LAD	Cont vs. CX	Cont vs. RCA	LAD vs. CX vs. RCA
Rest	RR	789±72	812±64	865±46	830±65	NS	NS	NS	NS
	QTmax	385±29	385±34	381±28	387±29	NS	NS	NS	NS
	QTmin	357±25	340±28	341±24	345±25	0.01	0.05	0.05	NS
	QTD	31±10	47±14	41±11	43±9	0.01	0.05	0.05	NS
	QTcmax	407±31	410±34	413±30	407±32	NS	NS	NS	NS
	QTcmin	376±25	360±29	365±31	362±32	0.05	0.05	0.05	NS
	QTcD	33±12	49±13	45±10	44±11	0.05	0.05	0.05	NS
Post-exercise#	RR	492±35	521±41	535±38	528±42	NS	NS	NS	NS
	QTmax	339±21	345±19	336±17	341±18	NS	NS	NS	NS
	QTmin	308±17	285±21	283±18	285±20	0.05	0.05	0.05	NS
	QTD	34±11	59±16	54±14	56±13	0.01	0.05	0.02	NS
	QTcmax	418±27	417±19	402±24	412±19	NS	NS	NS	NS
	QTcmin	379±24	340±21	344±23	350±24	0.05	0.05	0.05	NS
	QTcD	38±12	68±18	59±17	61±18	0.001	0.005	0.005	NS
% change	QTD	2±2	12±3	9±3	10±3	0.01	0.01	0.01	NS
	QTcD	3±2	15±4	11±3	12±4	0.01	0.05	0.05	NS

Cont: *control group,* ***RR:*** *RR interval*
 # Second minute of recovery period
%change: *percentage changes in QT dispersion between rest and post-exercise period for each 100 ms increase in heart rate Other abbreviations were given in Table 1.*

45±10 ms in the CX group and 44±11 ms in the RCA group with a significant difference between control and other three groups ($p<0.05$; Table 2).

QTD and QTcD at post-exercise period

QTD at post-exercise period (2nd minute recovery) ranged from 15 to 65 sec (mean 34±11 ms) in the control group, from 35 to 110 ms (mean 59±16 ms) in the LAD group, from 30 to 85 ms (mean 54±14 ms) in the CX group, from 30 to 90 ms (mean 56±13 ms) in the RCA group with a significant difference between the control and other three groups ($p<0.05$; Table 2). QTcD at post-exercise period was 38±12 ms in the control group, 68±18 ms in LAD group, 59±17 ms in the CX group and 61±18 ms in the RCA group with a significant difference between control and other three groups ($p<0.005$, Table 2). The increase in QTcD between rest and post-exercise period was statistically significant in LAD, CX and RCA group ($p<0.05$), but not in control group (Fig.1).

Fig.2 shows the changes in QTcD as lines, which connects the resting and post-exercise period values. Although most patients in the control group showed small changes in the dispersion of QT, the magnitude of these changes were much greater in patients with single-vessel CAD. The effect of ischemia localization as proximal, mid and distal part of the related coronary artery on QTD, QTcD and % change in QTcD were also investigated and no correlation was found ($p>0.05$) (Table 3). When the relation between the QTD and ischemic ECG changes during the post-exercise period was investigated, it was seen that the increase in QTD is well correlated with ST-segment depression ($r=0.706$, $p<0.001$).

% change in QTD and QTcD

The percent change in QTD was 2±2 in control group, 12±3 in LAD group, 9±3 in CX group and 10±3 in RCA group with a significant difference between the control and other three groups ($p<0.01$; Table 2). The percent change in QTcD was 3±2 in control group, 15±4 in LAD group, 11±3 in CX group and 12±4 in RCA group with a significant difference between the control and other three groups ($p<0.05$; Table 2).

DISCUSSION

QT interval reflects the total duration of depolarization and repolarization of the ventricular muscle. The difference between the maximum and minimum QT interval measured from 12-lead ECG is called

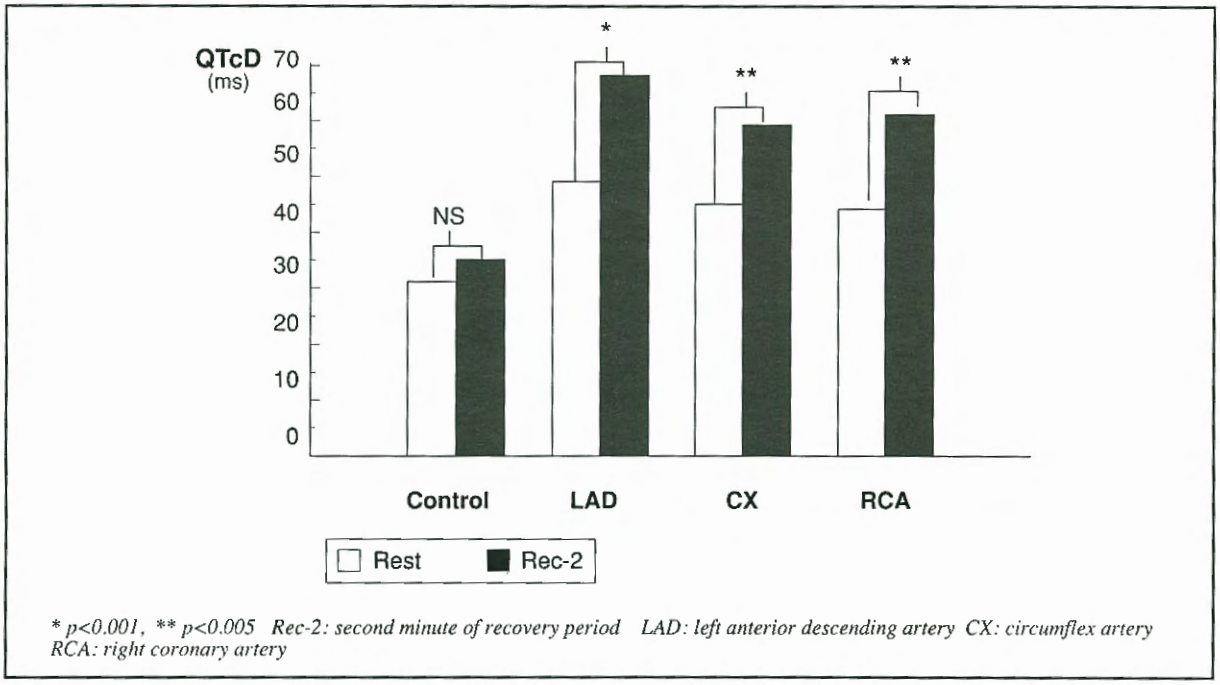

Figure 1. Bar diagram showing the changes in corrected QT dispersion (QTcD) at rest and 2nd minute recovery (Rec-2) period in all four groups. Increase in QTcD is seen to be much larger in LAD, CX, and RCA group in compared with that in control group

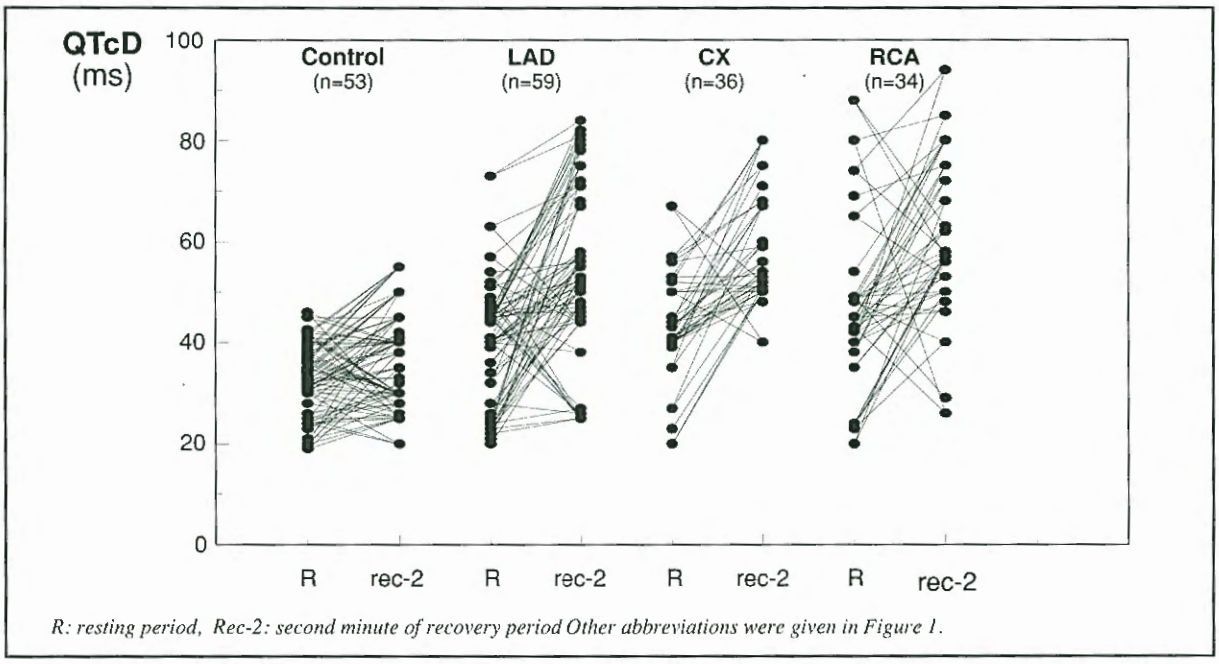

Figure 2. Corrected QT dispersion (QTcD) changes in all four group of patients were shown as lines connecting the QTcD data points during rest and 2nd minute recovery (Rec-2) period. It is seen that magnitude of changes in QTcD is greater in LAD, CX and RCA groups when compared to control group.

Table 3. QTD (ms), QTcD (ms), and % changes in QTcD in Single-vessel Disease Groups Classified According to the Lesion Localization

		LAD			CX			RCA		
		Prx (n=9)	Mid (n=39)	Distal (n=11)	Prx (n=7)	Mid (n=17)	Distal (n=12)	Prx (n=23)	Distal (n=11)	p value
Rest	QTD	51±15	48±13	47±14	45±19	41±9	43±12	47±16	42±13	NS
	QTcD	54±16	49±12	46±11	48±15	41±9	43±13	42±11	46±12	NS
Post-exercise	QTD	62±21	57±16	58±15	59±14	54±13	50±12	59±14	55±14	NS
	QTcD	71±23	68±19	65±15	58±15	61±20	54±16	63±18	61±17	NS
% change	QTcD	15±5	11±4	13±3	14±4	10±4	12±3	14±4	11±3	NS

Prx: proximal localization of stenosis Other abbreviations were given in Table 1.

QT dispersion. This term is used to describe the heterogeneity of ventricular repolarization [1,2] and an increase in QTD has been shown to increase the risk of serious arrhythmias and sudden cardiac death [13-15]. The main reason for investigating QTD changes is to find a method to predict the risk of arrhythmogenesis and sudden death.

There are several conditions, which lead to QT prolongation. Among these conditions myocardial ischemia has been shown to be an important predisposing factor for increased QT dispersion. In the studies performed with atrial pacing, Stierle et al., Sporton et al. and Lowe et al. showed that QTD was increased significantly in those with coronary artery disease (CAD) but not in those without coronary artery disease [7,8,12]. Lowe et al. also found that patients with triple-vessel disease had higher QTD at rest and there was no correlation between QTD and number of diseased vessels [12]. However, Sporton et al. found a positive correlation between the extent of CAD and the extent of QTD but the number of patients in this study was only eighteen [8].

Changes in QTD during EST was also investigated in a few studies with the intention to differentiate CAD from healthy subjects and conflicting results were reported [16,17]. In recent three studies per-

formed with EST, Roukema et al. and Stoletniy et al. reported that in ischemic heart disease QTD at rest and peak exercise is greater when compared to control group [9-11]. In an other recent study, Strutters et al. examined exercise induced changes in QTD by EST in order to differentiate different grades of CAD and found that in resting conditions there was no difference in QTD between control group and patients with CAD [18]. They supposed that an exercise-induced 16 ms or greater QTD had an 88% sensitivity and a 95% negative predictive accuracy in identifying triple-vessel disease. In all of these studies performed with atrial pacing or EST, QTD changes were investigated mostly in multi-vessel CAD. However, the relation of QTD to lesion localization or association between QTD and involved coronary artery has not yet been fully elucidated.

To our knowledge this is probably the first study investigating the association between QT dispersion and the involved coronary artery and lesion location during rest and exercise in single-vessel disease systematically and extensively. The first finding of our study is that, single-vessel groups had a wider baseline QTcD which increased further with exercise compared to control group. The control group had a lower baseline QTcD that showed little or no increase during exercise.

Our finding of increased resting QTD in CAD is consistent with the finding of Roukema et al., Stoletniy et al. and Lowe et al. [9,10-12] although their patients mostly were in multi-vessel disease group. Furthermore, in some studies it was reported that baseline QTD did not distinguish patients with CAD from healthy subjects [7,8,12]. In a report by Bonow et al., it was shown that abnormalities of regional diastolic functions have been observed in patients with single-vessel CAD and normal resting systolic wall motion [19]. Therefore the finding of prolonged resting QTD in these patients is an acceptable finding and not suprising.

The second finding of the present study is that, there was high a correlation between the QTD increase and ST-segment depression during the post-exercise period. The third finding of our study is that, there is no association between QTD and the coronary artery involved. In all single-vessel groups, QTD, QTcD and % change in QTcD increased in similar percentages in response to exercise when compared to control group which no significant increase was observed.

The last finding of the present study is that, there is also no relation between lesion localization and QTD in the related coronary artery as proximal, mid and distal. Since QTD is supposed to be dependent on regional differences in action potential duration [20], severity of localized ischemia rather than extend of CAD would be expected to have greater influence on inducible QTD [12]. From this point of view, the lack of difference in QTD between the proximal, mid or distal location of the lesion in the related artery in our study is a reasonable finding. We observed that the increase in QTD and QTcD during exercise was mainly due to a decrease in QT-min and QTc-min while QT-max and QTc-max remained constant. This finding was also reported by some other investigators [7,21,22]. This observation was supported by Kleiman et al. who showed that acute ischemia is associated with local shortening of the action potential duration [23].

Study Limitations

This study has some limitations. First, all stenosis above %70 were taken into the same category and not classified in subgroups and so we need other studies which will clarify the relation of degree of stenosis with QT dispersion. Second, in our study QT measurements were done manually with the aid of a magnifying glass by a single observer. Although it is conceivable to think that automatic measurement of QT dispersion may be superior to manual measurement in reproducibility, there is also evidence that manual measurements is superior to automatic measurement of QTD which usually need some manual editing and give different results [24,25]. In our study, also the leads in which the T wave amplitude was less than 0.1 mV or in which termination could not be clearly identified were excluded from the study in order to prevent erroneous results. The intraobserver variation for QTD was within 9 ms and a high intraobserver correlation was found ($p<0.0005$). Third, to examine the exercise-induced QTD changes, second minute of the recovery period was selected because at very fast heart rates as in peak exercise period, T and P waves may be fused so the QT interval may be difficult to measure.

For this reason, we prefer to measure QT intervals at an early period of recovery period. So it can be suggested that our results for QTD measurements at post-exercise period may not reflect the values for maximal ischemic condition but, in a previous study it was shown that QTD changes returned to baseline values within 5 minutes after cessation of pacing (7). So, we supposed that to measure QT interval changes in the early period of the recovery period also is reliable and reflect the changes in QTD due to exercise in patients with CAD.

Conclusion

Our study indicates that the patients with single-vessel CAD had wider resting QT dispersion when compared to control group, which further increased significantly with exercise and QT dispersion is unrelated to lesion localization or to which coronary artery is involved. We also observed that QTD increase is well correlated with ST segment depression during exercise. These findings support the opinion that severity of localized ischemia rather than extent of CAD would be expected to have greater effect on inducible QT dispersion.

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