

# The Relationship Between QT Dispersion and Left and Right Ventricular Diastolic Dysfunction in Patients With Myocardial Infarction

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**Objective:** The purpose of this study was to investigate the relation between electrical dispersion and impairment of ventricular filling in patients with acute myocardial infarction (MI).

**Methods:** Thirty patients with recent myocardial infarction (17 patients with anterior and 13 patients with inferior MI) were included in the study. QT dispersion (QTd) was defined as maximum minus minimum QT interval durations. Flow propagation velocity measured by color m-mode echocardiography was used to determine diastolic function.

**Results:** There was a positive correlation between isovolumic relaxation time and QTd, as well as negative correlation existed between left ventricular flow propagation velocity (LVFPV) and QTd. The QTd was greater and LVFPV was lower in patients with anterior myocardial infarction as compared with those with inferior MI.

**Conclusion:** There is an association between electrical dispersion and left ventricular filling abnormalities in patients with acute myocardial infarction. (*Ana Kar Der, 2001; 1:266-271*)

**Key Words:** Color M-mode echocardiography, QT dispersion, myocardial infarction

## Introduction

Different electrocardiographic (ECG) leads magnify the ECG signal of different myocardial regions and consequently QT dispersion is supposed to be a measure of the heterogeneity of myocardial repolarization (1). It is proposed that left ventricular filling abnormalities may be caused by increased electrical dispersion since the relationship between asynchronous movement of the left ventricular (LV) walls and parameters of diastolic filling suggests a link between heterogeneous myocardial repolarization and left ventricular filling (2). Mitral flow propagation velocity defined by color M mode echocardiography has been proposed as a preload independent index of ventricular relaxation (3-7).

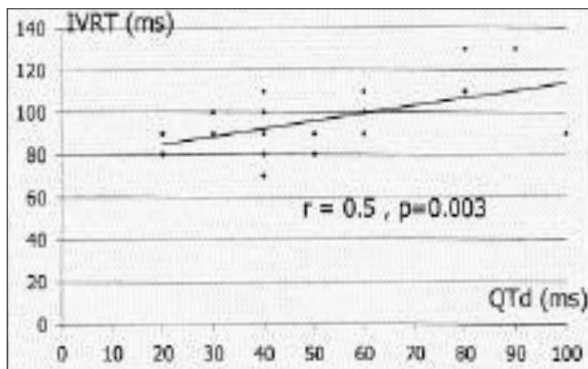
The aim of this study was to investigate the relation between electrical dispersion and impairment of filling in patients with acute myocardial infarction (MI) where abnormal LV filling is frequently seen (8-11). Flow propagation velocity (FPV) was used in addition

to known methods to assess diastolic function.

## Material and Methods

**Study population:** Thirty patients (mean age 51.5±0.71 years) with recent MI (10±2 days) were included in the study. Patients with chronic atrial fibrillation and bundle branch block were excluded from the study. Basic characteristics (age, gender, risk factors), localization of MI and in-hospital treatment modalities were evaluated for each patient. Before discharge from the hospital patients underwent a detailed echocardiographic examination to assess ventricular function and a 12-lead electrocardiogram with speed of 50 mm/sec for the assessment of QT dispersion. ECG recordings were done prior to the echocardiographic examination. According with the localization of MI all patients were divided into two groups: group A – 17 patients with anterior MI and group B - 13 patients with inferior MI.

**QT dispersion:** QT dispersion (QTd) was defined as maximum minus minimum QT interval durations. QT intervals were measured in all 12 leads of a standard ECG. The QT interval was defined as the dis-



**Figure 1. Correlation between isovolumic relaxation time and QT dispersion.**

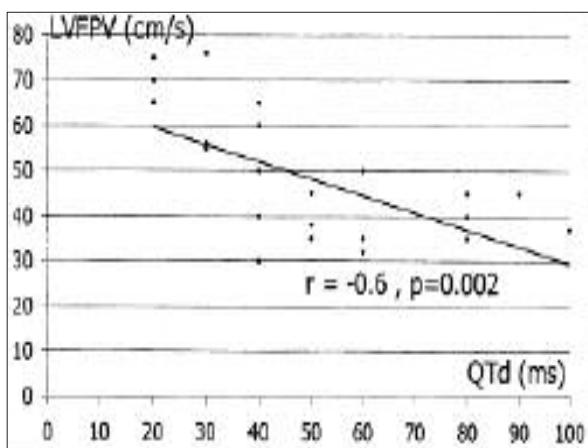
tance from the beginning of QRS complex to the end of the T wave. End of the T wave was determined as the point where T wave returned to the TP baseline. The average QT interval was calculated from the successive heart cycles in each lead and then corrected for heart rate using Bazett formula. QT dispersion and corrected for heart rate QTd were further included into the analysis.

**Echocardiographic Study:** A Vingmed CFM 725 machine with a 3.5 MHz transducer was used and echocardiographic examinations were done according to the recommendations of the American Society on Echocardiography (12). Left ventricular volumes and ejection fraction (LVEF) were measured by

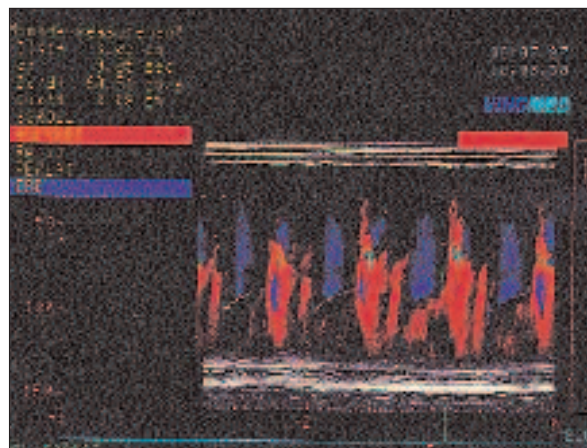
**Table 1: Demographic and other characteristics**

PARAMETERS		Group A(n=17)	Group B(n=13)
	n(%)	n(%)	n(%)
Age, years	54.8±9.9	52.2±10.8	57.4±9.0
Gender (male)	27(93)	17(100)	10(76.9)
Smoking	19(63)	8(47)	11(84.6)
Heredity	6(20)	4(23)	2(15.4)
Hypertension	9(30)	5(29)	4(30.7)
Hyperlipidemia	7(23)	4(23.5)	3(23)
Diabetes Mellitus	5(16.6)	3(17.6)	2(15.4)
Thrombolysis	14(46)	9(52.9)	5(38.5)
Beta Blocker	17(56.7)	10(58.8)	7(53.8)
Ca-antagonist	2(6.7)	1(5.8)	1(7.6)
QT dispersion, ms	47.3±21.3	57.0±20.0	30.0±13.0*
QTc dispersion, ms	48.6±21.6	58.6±22.0	35.8±12.9*

\* - differences between group A and B are significant – p=0.03  
Data are presented as mean value or number (%) of patients



**Figure 2. Correlation between left ventricular flow velocity propagation and QT dispersion.**



**Figure 3. Color M-mode sample of LV flow propagation in patients with anterior myocardial infarction.**

Simpson's modified biplane method (12). Mitral, tricuspid and pulmonary venous flow velocities were studied by pulsed wave Doppler. Five consecutive beats obtained during quiet respiration were measured and averaged for each Doppler variable and the peak velocities during early (E) and atrial contraction (a) filling, their ratio (E/a), deceleration time E (dtE) and isovolumic relaxation time (IVRT) for both transmitral and transtricuspidal Doppler flows, the peak systolic (S) and diastolic (D) velocities of pulmonary vein flow, their ratio (S/D) were calculated. Color M-mode echocardiography was done in the apical four-chamber view, with the cursor aligned parallel with LV and RV inflows. LV and right ventricular (RV) FPV were measured by the linear slope of the color front

bolytic therapy. However, in general, the baseline and clinical characteristics were similar in both study groups. Study groups also did not differ as regards to treatment by beta-blockers and calcium antagonists. Fifty-eight percent of patients in group A and 53.8% of patients in group B were receiving beta-blockers, whereas only 5.8% of group A and 7.6% of group B patients were taking calcium antagonists during evaluation. None of the patients received nitrates and use of ACE inhibitors was similar in both groups.

All patients were in sinus rhythm during ECG recording: mean heart rate did not differ between groups. QT dispersion values (absolute and corrected

**Table 2: Echocardiographic findings**

Parameters	Group A	Group B	p
LA (mm)	32.9+8.6	33.5+3.4	<0.82
LV ESD, (mm)	38.4+6.2	34.5+5.7	<0.09
LV EDD, (mm)	51.9+4.8	48.0+5.3	<0.05
LVEF, (%)	47.9+9.3	56.2+8.2	<0.018*
E/A(mitral)	1.38+0.6	1.25+0.3	<0.4
DT, (ms)	171+42	182+35	<0.45
IVRT, (ms)	97.5+17.3	90.8+6.7	<0.2
E/A (tricuspid)	1.15+0.3	1.2+0.2	<0.6
S/D	1.08+0.2	1.25+ 0.3	<0.16
LVFPV, (cm/s)	44.2+10.0	55.6+16.4	<0.03*
RVFPV, (cm/s)	36.0+8.0	50.0+14.0	<0.012*

\* - differences are significant

LA- left atrial diameter, LVESD – left ventricular end-systolic dimension, LVEDD - left ventricular end-diastolic dimension, LVEF – left ventricular ejection fraction, E/A – ratio of early to late filling velocities, DT – deceleration time, IVRT – isovolumic relaxation time, S/D – systolic to diastolic pulmonary venous flow velocities ratio, LVFPV – left ventricular flow propagation velocity, RVFPV – right ventricular flow propagation velocity

wave while the cursor was placed between the level of annulus and 2 cm in depth of LV or RV or the slope of the first aliasing velocity from the mitral annulus in early diastole to 4 cm distally into the ventricular cavity.

**Statistical Analysis:** Two-tailed Student's t test and Pearson's correlation test were used. Values of  $p < 0.05$  were considered as significant.

## Results

Clinical characteristics of the study groups are displayed in Table 1. Mean age of patients included into the study was 54.8+9.9 years and 93% of them were male. There were more smokers in group B, while more patients of group A underwent throm-

bolitic therapy) were greater in group A as compared with group B (57.0+20.0 ms vs. 30.0+13.0 ms,  $p=0.03$  and 58.6+22.0 ms vs. 35.8+12.9 ms,  $p=0.03$ , respectively).

When two groups were compared by means of left atrial size and LV dimensions there were no significant differences (Table II), however LVEF was lower in group A than in group B (47.9+9.3% vs. 56.2+8.2 %,  $p < 0.018$ ). Mitral and tricuspid inflow velocities, pulmonary vein flow velocities, dtE and IVRT were similar in both groups. However, LVFPV and RVFPV were lower (44.2+10 cm/sec vs. 55.6+16.4 cm/sec,  $p=0.03$  and 36+8 cm/sec vs. 50+14 cm/sec,  $p=0.01$ , respectively) in patients with anterior MI than in those with inferior one.

There was a positive correlation between QTd and

IVRT ( $r=0.5$ ,  $p=0.003$ ) (Figure 1). On the other hand, there was a negative correlation between QTd and mitral E/A ratio ( $r=-0.5$ ,  $p=0.003$ ), LVFPV ( $r=-0.6$ ,  $p=0.002$ ) and RVFPV ( $r=-0.5$ ,  $p=0.02$ ), while there was no correlation between QTd and mitral dtE (Figure 2).

## Discussion

QT dispersion by definition is the inter-lead differences in QT interval duration. Range of durations has been proposed as an index of the spatial dispersion of the vectorial recovery times. Majority of studies have shown increased QT dispersion in various cardiac diseases (1). Compared with healthy controls an increased QT dispersion has been reported in heart failure and left ventricular dysfunction of various etiology: LV hypertrophy, arterial hypertension irrespective of presence of LV hypertrophy and hypertrophic cardiomyopathy (13-25).

Generally, QT dispersion is increased in acute MI (26) and alternating values from 40 ms to 162 ms have been reported. Several studies have shown greater QT dispersion in anterior compared to inferior MI (27-29). QT dispersion in MI has been found to correlate with indirect measures of infarct size such as EF (30). It is proposed that there is a relationship between QT dispersion and relaxation abnormality (2).

Assessment of relaxation abnormalities can be accomplished by measurement of mitral inflow patterns. Impairment of LV relaxation results in prolongation of IVRT, decrease in early transmitral flow velocity and prolongation of the dtE (31).

Since 1990's a new method has been used in measurement of flow propagation by color M-mode technique (32-35). Flow propagation velocity  $<45$  cm/sec has been considered as a positive sign of relaxation abnormality (36). Early diastolic interventricular filling pattern was investigated by Steine et al. (37) in acute MI by color M-mode echocardiography. They measured the difference between occurrence of peak flow velocity at the mitral tip and in the apical region by a blinded analysis. The study showed slowing of early diastolic mitral to apical flow propagation (37).

A correlation between prolonged QT dispersion and shortening of the E wave deceleration time has been found by Szymanski et al. (2). They suggested that greater QT dispersion might reflect more advanced impairment of left ventricular filling in MI patients. Asynchronous movement of the ventricular walls concerns both electrical and mechanical functions of the left ventricle leading to the impairment of left ventricular filling (38).

The main finding of our study is demonstration of association of increased QT dispersion with ventricular relaxation abnormalities in acute MI patients. There were positive correlation between QTd and IVRT, negative correlation between QTd and LVFPV. At the same time, QT dispersion was greater in patients with anterior MI than in patients with inferior MI.

These findings are in agreement with results reported by Szymanski et al. (2), confirming that greater electrical dispersion is associated with greater diastolic dysfunction, validated by the assessment of IVRT and mitral flow propagation dynamics. Diastolic dysfunction observed in acute MI may lead to electrical dispersion as well. Main difference between our and previous study (2) is that they found a correlation of QT dispersion with restrictive pattern of diastolic dysfunction (shortened dtE), whereas we demonstrated an association with relaxation abnormality (increased IVRT and shortened FPV). The region of MI is also important too, since anterior MI's manifest with more severely depressed LV function. Therefore, both electrical and mechanical dysfunctions are greater in anterior than in inferior MI.

In conclusion, this study demonstrates that there may be a relationship between QT dispersion and ventricular relaxation abnormalities in patients with acute myocardial infarction.

Limitations of the study. QT dispersion is affected by drugs and our patients were receiving beta-blockers. Secondly, our patients had a relatively preserved LV systolic function. Whether presence of both systolic and diastolic dysfunctions is accompanied by expected greater increase in QT dispersion should be validated in larger series.

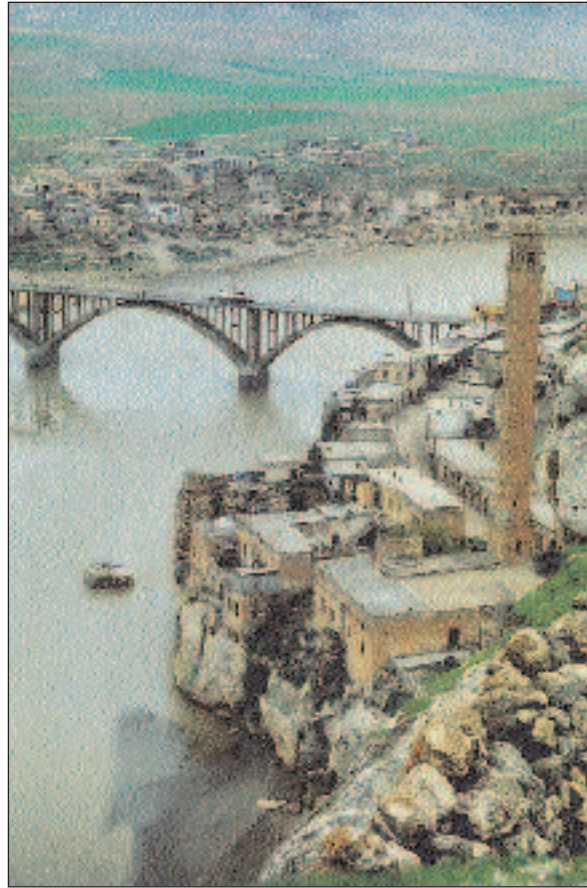
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