

# Dispersion of QT interval in premature ventricular beats is not an independent marker for inducible sustained ventricular tachycardia

Ventriküler prematüre atımların QT interval dispersiyonu indüklenebilir sürekli ventriküler takikardi için bağımsız bir gösterge değildir

Fatih Sinan Ertaş, Çağdaş Özdöl, Timuçin Altın, Yusuf Atmaca, Ömer Akyürek, Güneş Akgün, Remzi Karaoğuz, Muharrem Güldal, Çetin Erol

Department of Cardiology, Ankara University School of Medicine

**Aim:** Variability in QT interval duration on the different leads of the 12-lead ECG has been proposed as an indicator of risk for ventricular arrhythmias in different clinical settings, but the value of QTd-V is not clear yet. The aim of this study was to estimate the value of QT dispersion in ventricular premature beats (QTd-V) in identifying patients susceptible to reentrant ventricular tachyarrhythmias (VT).

**Materials and Methods:** We compared the performance of precordial QTd-V, late potentials on the signal-averaged electrocardiogram and reduced left ventricular ejection fraction for identification of inducible ventricular tachycardia in 34 patients undergoing electrophysiologic study.

**Results:** QTd-V in 12 patients with inducible VT ( $110 \pm 50$  msec) was found to be greater than that in 22 patients without inducible VT ( $65 \pm 38$  msec,  $p=0.006$ ). Multivariate analysis including ejection fraction and presence of late potentials showed that QTd-V was not an independent factor in identifying the susceptible patients to ventricular tachyarrhythmias.

**Conclusion:** Increased QTd-V is related to susceptibility to reentrant ventricular tachyarrhythmias, however does not appear to provide additional diagnostic information to that provided by late potentials and left ventricular ejection fraction.

Key words: **QT dispersion, ventricular premature beat, electrophysiologic study**

**Amaç:** 12 derivasyonlu EKG nin farklı derivasyonları arasında QT interval süresinde değişkenlik birçok klinik durumda ventriküler aritmiler için bir risk göstergesi olarak öngörülmüşken, QTd-V nin değeri henüz açık değildir. Çalışmanın amacı reentran ventriküler takiaritmilere (VT) duyarlılığı olan hastaları belirlemede ventriküler premature atımlarda QT dispersiyonu'nun (QTd-V) değerini araştırmaktır.

**Gereç ve Yöntem:** Elektrofizyolojik çalışma yapılmış 34 hastada indüklenebilir VT nin tahmini için prekordiyal QTd-V, sinyal ortalamalı EKG de geç potansiyeller ve azalmış sol ventrikül ejeksiyon fraksiyonu karşılaştırıldı.

**Bulgular:** İndüklenebilir VT li 12 hastadaki QTd-V ( $110 \pm 50$  msec) VT nin indüklenemediği 22 hastadaki QTd-V den ( $65 \pm 38$  msec,  $p=0.006$ ) daha büyük bulundu. Ejeksiyon fraksiyonu ve geç potansiyel varlığının dahil edildiği çok değişkenli analiz QTd-V nin ventriküler takiaritmilere duyarlı hastaları belirlemede bağımsız bir faktör olmadığını gösterdi.

**Sonuç:** Artmış QTd-V tek başına reentran ventriküler takiaritmilere duyarlılıkla ilintili olsa da, geç potansiyeller ve sol ventrikül ejeksiyon fraksiyonu hesaba katıldığında ilave tanısal bilgi sağlama-dığı gözükmemektedir.

Anahtar sözcükler: **QT dispersiyon, ventriküler prematüre atım, elektrofizyolojik çalışma**

Received: 03.01.2006 • Accepted: 16.01.2006

Corresponding author

Dr. Fatih Sinan Ertaş  
Bilkent-3 Ufuk Sitesi D1/06800 Ankara, Turkey  
Tel : +90 (312) 508 25 23  
Fax : +90 (312) 312 52 51  
E-mail adress : ertas@medicine.ankara.edu.tr

Experimental studies have provided powerful evidence of the dispersion of myocardial recovery times for the occurrence ventricular arrhythmias (1-4). Measurement of the variability in QT interval duration among the different leads of the standard 12-lead electrocardiogram (ECG) (i.e., QT dispersion) has been proposed as a noninvasive method for detecting the inhomogeneity of ventricular recovery times (5-9).

Observations have related an increase in QT dispersion on the surface ECG to increased risk of clinically important ventricular arrhythmias (10-14). Useful

prognostic value of abnormally increased QT dispersion has been found in patients with acute myocardial infarction (10), cardiomyopathy (11), the long QT syndrome (10,15), drug induced torsades de pointes (16) and sudden cardiac death (17).

The presence of late potentials on the signal-averaged ECG (6,18,19,20,21,22) and abnormal left ventricular ejection fraction (LVEF) (23) also have been associated with an anatomic substrate for reentrant ventricular arrhythmias and inducible ventricular tachycardia (VT) at electrophysiologic study (EPS).

There are only several reports studying the clinical significance of QT interval dispersion measured in spontaneous ventricular beats (QTd-V) (24-26) showing significant relationship between QTd-V and the risk of arrhythmic events.

The present study was designed to examine relation of measures of precordial QT dispersion in premature ventricular beats and inducible ventricular arrhythmias and the independence of these findings from late potentials and ventricular function.

## Materials and Methods

Patients who underwent electrophysiologic testing because of symptomatic ventricular arrhythmia at our center were enrolled into the study. The study group consisted of 34 patients (21 men, 13 women) who had premature ventricular beats on a 12-lead standard ECG and signal averaged ECG's before electrophysiologic study. The mean age of the patients was 54 years.

### Electrocardiography

Standard ECG's were recorded with a 12-channel ECG recorder at a paper speed of 25mm/sec. QT interval, QT dispersion, QRS complex duration, QTd-V and duration of the QRS complex of ventricular extrasystole measured manually. The QT intervals were measured from the onset of the QRS complex to the end of the T wave by means of tangential method. When U waves were present, the QT interval was measured to the nadir of the curve between T and U waves, also with the aid of tangential method. The QRS complex duration of normal and ventricular premature beats were measured from the beginning of the QRS complex to its end. QTd-V was defined as difference between the maximum and minimum QT interval measured in ventricular premature beats across the 12-lead ECG. A minimum 10-leads was required for QTd-V to be calculated. Index of prematurity of VPBs was obtained by dividing the coupling interval time of ventricular premature beat (VPB) by the QT interval duration of preceding normal beat.

### Signal averaged ECG

After careful preparation of the skin and with the patient lying quietly in the supine position, three orthogonal X,Y and Z leads were acquired with an operator selected template at a frequency of 2000 Hz. Digital filtering was performed on averaged, orthogonal-lead complexes with a fourth-order 40 to 250 Hz bandpass bidirectional digital filter. Late potentials were defined as present when the filtered vector QRS duration was >114 msec and either the root-mean square voltage of the terminal 40 msec of the vector QRS was < 20  $\mu$ V or the low-amplitude signal was > 38 msec.

### Electrophysiologic study

Electrophysiologic testing included programmed ventricular stimulation using up to three extrastimuli and two basic drive cycle lengths (600 and 400 ms) from the right ventricular apex and outflow tract. Ventricular tachycardia was defined as 1) sustained when its duration was >30s or if defibrillation was required for its termination; and 2) as non-sustained if it lasted >5 beats but <30s.

### Statistical analysis

Data are expressed as mean value and standard deviation. Paired and unpaired t tests were used for quantitative data where applicable. The Pearson correlation coefficient was used to estimate univariate correlations between the variables. Logistic multiple regression analysis was used to evaluate the independent values of the different variables in differentiating the patient groups with and without susceptibility to ventricular tachyarrhythmias. Differences were considered significant when  $p < 0.05$ .

## Results

### Group characteristics

Clinical characteristics of the study population groups relevant to the inducibility of VT at EPS are summarized and compared in Table 1. Patients with positive EPS were slightly older than those with negative EPS ( $57 \pm 15$  vs  $52 \pm 15$  years,  $p=0.37$ ). There were no significant sex differences among the groups. Patients with inducible VT were more likely to have coronary artery disease or cardiomyopathy than those with negative EPS. There were significant difference between the patient groups in terms of the left ventricular ejection fraction ( $59 \pm 12$  vs  $45 \pm 14$  respectively,  $p=0.005$ ).

### QT measurement comparisons:

Table 2 summarizes and compares the resting electrocardiographic findings in two groups. QRS duration of the patients with positive EPS was significantly greater than that of the patients with negative EPS, but the QRS duration of ventricular premature beat was not significantly

different between the groups. Patients with inducible ventricular arrhythmias had a longer QT dispersion of normal QRS complexes and ventricular premature beats ( $p=0.001$  and  $p=0.006$  respectively). The prematurity index did not differ between the groups.

**Signal averaged electrocardiogram measurements:**

Table 3 summarizes and compares the signal averaged ECG findings in the groups. The QRS duration of the patients with inducible ventricular arrhythmias was significantly longer ( $p=0.003$ ). The presence of ventricular late potentials were much more common in EPS positive patients than EPS negative patients (20% vs 88%,  $p=0.002$ ).

**Regression analysis:**

There were no significant correlation between EF and QTd-V in EPS negative patients ( $r=0.13$ ,  $p=0.9$ ) or in EPS positive patients ( $r=0.12$ ,  $p=0.7$ ). A correlation was found between QTd-V and QTd in EPS positive patients ( $r=0.7$ ,  $p=0.01$ ) but no correlation was found in negative EPS patients ( $r=0.3$ ,  $p=0.1$ ). The relationship of QT-V dispersion to the presence of late potentials and an abnormal LVEF who underwent EPS is examined by logistic regression analysis. Among these patients, QTd-V, late potentials ( $p=0.07$ ) and ejection fraction ( $p=0.3$ ) were not independently associated with susceptibility to ventricular tachyarrhythmias.

**Discussion**

The identification of patients who are at high risk for ventricular tachycardia and sudden death is of great importance. The strategies such as ventricular ectopic activity and spontaneous arrhythmias can not effectively identify subjects at high risk (27). The newer noninvasive methods such as signal-averaged electrocardiography, heart rate variability and baroreceptor reflex sensitivity offer improved risk stratification (28). However, the positive predictive accuracy of each of these methods is still limited with regard to identifying individual patients for therapeutic interventions; it is possible that the combination of noninvasive methods may result in better accuracy (29).

The present study demonstrates that prolonged QT dispersion of normal and ventricular premature beats on the standard resting ECG can identify patients with inducible VT. These findings confirm the previous report of Dabrowski that demonstrate a significant relationship between QTd-V and risk of arrhythmic events (24-26). These observations are consistent with the hypothesis that QT dispersion on the standard ECG is a marker of underlying regional inhomogeneity of ventricular repolarization that can be associated with reentrant VT.

Difference from the previous studies from Dabrowski (24-26) showing QT dispersion of ventricular premature beat is

**Table 1.** Clinical characteristics of the study patients

	Negative EPS patients (n=22)	Positive EPS patients (n=12)
Age (yr)	52±15	57±15
Sex (male/female)	14/8	7/5
LVEF	59±12 %	45±14
CAD	2 ( 9.1%)	4 ( 33.3%)
HT	6 ( 27.3%)	None
DCMP	3 (13.6%)	3 (25%)
VHD	4 ( 18.2%)	None
ICMP	1 (4.5%)	5 ( 41.7%)
none	6 (27.3%)	none

\*Data are expressed as mean ± SD  
**\*\*CAD:** coronary artery disease; **DCMP:** dilated cardiomyopathy; **ICMP:** ischemic cardiomyopathy; **EPS** electrophysiologic study; **HT:** hypertension; **LVEF:** left ventricular ejection fraction; **VHD:** valvular heart disease;

**Table 2.** Summary of electrocardiographic measurements

	Negative EPS patients (n=22)	Positive EPS patients (n=12)	p
QRS duration	99±6	147±32	0.001
QT dispersion	41±15	75±37	0.001
QTd-V	67±38	110±50	0.006
QRS-V	157±36	163±30	NS
Prematurity index	1.8±0.9	1.2±0.9	NS

\***QTd-V, QT:** dispersion of ventricular premature beat; **QRS-V, QRS:** complex duration of ventricular premature beat; **NS:** non significant

**Table 3.** Signal averaged ECG findings in patient groups

	Negative EPS patients (n=22)	Positive EPS patients (n=12)	p
LAS	32.7±17.8	65±25	0.006
QRS duration	112±37	164±22	0.003
RMS	43.9±27.8	14.2±13	0.01
Late potentials	20%	88%	0.002

\***LAS:** low amplitude signal duration; **RMS:** root mean square voltage

an independent risk for arrhythmic events, the present study demonstrates that QTd-V had a similar accuracy compared with that of other noninvasive methods used in previous studies for discriminating between patients with different susceptibilities to ventricular tachyarrhythmias and does not provide

additional independent diagnostic information. However, measurement of QT dispersion is easy, inexpensive and non-invasive but because measurement of QT interval and its dispersion are subject to interobserver and interobserver variability (28,29) and the problems with identification of the end of the T wave and as QTd-V is a dependent risk factor, the measurement of QTd-V should not be included in the non-invasive evaluation of arrhythmic risk.

### Limitations

Our major limitations were small size of study population and the inhomogeneity of the study groups. Prospective

studies of larger and homogeneous groups of patients are therefore needed to confirm the present data.

### Conclusion

Our findings demonstrate that precordial dispersion of ventricular premature beat on the resting ECG is associated with the presence of inducible reentrant VT in symptomatic patients undergoing EPS, however QT dispersion of ventricular premature beat does not appear to provide additional independent diagnostic information to that provided by late potentials and left ventricular ejection fraction.

### References

- Han J, De Jalon PG, Moe GK. Adrenergic effects on ventricular vulnerability. *Circ Res* 1964; 14:516-24.
- Han J, Moe GK. Non-uniform recovery of excitability in ventricular muscle. *Circ Res* 1964; 14:44-60.
- Merx W, Yoon MS, Han J. The role of local disparity in conduction and recovery time on ventricular vulnerability to fibrillation. *Am Heart J* 1977; 94:603-10.
- Kuo CS, Munokata K, Reddy CP et al. Characteristics and possible mechanisms of ventricular arrhythmia dependent on the dispersion of action potential durations. *Circulation* 1983; 67:1356-7.
- Day CP, McComb JM, Campell RWF. QT dispersion: an indication of arrhythmic risk in patients with long QT intervals. *Br Heart J* 1990; 63:342-4.
- Day CP, McComb JM, Campell RWF. QT dispersion in sinus beats and ventricular extrasystoles in normal hearts. *Br Heart J* 1992; 67:39-41.
- Dritsas A, Gilligan D, Nichoyannopoulou P et al. Amiodarone reduces QT dispersion in patients with hypertrophic cardiomyopathy. *Int J Cardiol* 1992; 36:345-9.
- Cowan JC, Yusoff K, Moore M, et al. Importance of lead selection in QT interval measurement. *Am J Cardiol* 1988; 61:83-7.
- Day CP, McComb JM, Matthews J et al. Reduction of QT dispersion by sotalol following myocardial infarction. *Eur Heart J* 1991; 12:423-7.
- Van de Loo A, Arendte W, Hohnloser S. Variability of QT dispersion measurements in the surface electrocardiograms in patients with acute myocardial infarction and in normal subjects. *Am Heart J* 1994; 74:1113-8.
- Pye M, Quinn A, Cobbe A. QT interval dispersion: a non-invasive marker of sustained ventricular arrhythmias? *Br Heart J* 1994; 71:511-4.
- Sylvén J, Horacek M, Spencer C et al. QT interval variability on body surface. *J Electrocardiol* 1984; 17:179-88.
- Goldner B, Brandspiegel H, Horwitz L et al. Utility of QT dispersion combined with the signal averaged electrocardiogram in detection patients susceptible to ventricular tachyarrhythmia. *Am J Cardiol* 1995; 76:1192-4.
- Perkiomaki J, Koistinen M, Yli-Mayry S et al. Dispersion of QT interval in patients with and without susceptibility to ventricular tachyarrhythmias after previous infarction. *J Am Coll Cardiol* 1995; 26:174-9.
- Priori S, Napolitano C, Dielhl L et al. Dispersion of the QT interval: a marker of therapeutic efficacy in the idiopathic long QT syndrome. *Circulation* 1994; 89:1681-9.
- Hii J, Wyse D, Gillis A et al. Precordial QR interval dispersion as a marker of torsades de pointes: disparate effects of class Ia antiarrhythmic drugs and amiodarone. *Circulation* 1992; 86:1376-82.
- Zareba W, Moss A, Le Cessie S. Dispersion of ventricular repolarization and arrhythmic cardiac death in coronary artery disease. *Am J Cardiol* 1994; 74:550-3.
- Cortina A, Ambrose J, Priet-Granada J et al. Left ventricular function after myocardial infarction: clinical and angiographic correlations. *J Am Coll Cardiol* 1985; 5:619-24.
- Merri M, Benhorin J, Alberti M et al. Electrocardiographic quantitation of ventricular repolarization. *Arrhythmia* 1989; 80:1301-8.
- Garson A. How to measure the QT interval: what is normal? *Am J Cardiol* 1993; 72:14B-16B.
- Bazett H. An analysis of time relations of electrocardiograms. *Heart* 1920; 7:353-70.
- Lander P, Berari E, Rajagopalan C et al. Critical analysis of the signal-averaged electrocardiogram: improved identification of late potentials. *Circulation* 1993; 87:105-17.
- Barr CS, Naas A, Freeman M et al. QT dispersion and sudden death in heart failure. *Lancet* 1994; 343:327-9.
- Dabrowski A, Kramarz E, Piotrowicz R. Dispersion of QT interval in premature ventricular beats as a marker of susceptibility to arrhythmic events. *Journal of Cardiovascular risk* 1998; 5:97-101.
- Dabrowski A, Kramarz E, Piotrowicz R. Dispersion of QT interval following ventricular premature beats and mortality after myocardial infarction. *Cardiology* 1999; 91:75-80.
- Dabrowski A, Kramarz E, Piotrowicz R et al. Predictive power of increased QT dispersion in ventricular extrasystoles and in sinus beats for risk stratification after myocardial infarction. *Circulation* 2000; 101:1693-7.
- Turitto G, El-Sherif N. Complex ventricular arrhythmias and nonsustained ventricular tachycardia: risk stratification and management. In: el-Sherif N, Samet P, editors. *Cardiac Pacing and Electrophysiology*. Philadelphia: Saunders, 1991; 217-33.
- Farrell TG, Bashir Y, Cripps T et al. Risk stratification for arrhythmic events in postinfarction patients based on heart rate variability, ambulatory electrocardiographic variables and the signal averaged electrocardiogram. *J Am Coll Cardiol* 1991; 18:687-97.
- Bigger JT Jr, Steinberg JS. Risk stratification for arrhythmic death after MI: an overview. In: El-Sherif N, Samet P, eds. *Cardiac Pacing and Electrophysiology*. Philadelphia: Saunders 1991; 303-23.
- Kautzner J, Yi G, Camm AJ et al. Short and long term reproducibility of QT, QTc and QT dispersion measurement in healthy subjects. *PACE* 1994; 17:928-37.
- Ahnve S. Methodological aspect of QTc interval determination. In: Butrous CG, Schwartz PJ, eds. *Clinical aspects of Ventricular repolarization*. London: Farrand press, 1989; 1-16.