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QT Dispersion and T-Loop Morphology in Late Pregnancy and After Delivery

M. LECHMANOVÁ, O. KITTNAR, M. MLČEK, J. SLAVÍČEK, A. DOHNALOVÁ, Š. HAVRÁNEK, J. KOLAŘÍK, A. PAŘÍZEK[1]

Institute of Physiology and [1]Department of Obstetrics and Gynecology, First Faculty of Medicine, Charles University, Prague, Czech Republic

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Summary

The aim of the study was to detect changes of both the QT dispersion and T-loop morphology resulting from the changed spatial position of the heart during pregnancy. Electrocardiographic and vectorcardiographic recordings were obtained from 37 healthy women 19-36 years old in the 36th to 40th week of physiological pregnancy and 2 to 6 days after delivery. The same recordings were obtained from 18 healthy women of the same age. The average QT dispersion (±S.D.) in normal subjects was significantly lower (34±12 ms) than in those in late pregnancy (73±18 ms) ($P<0.001$). The average amplitude of T-loop (Ta) in women in late pregnancy was significantly ($P<0.001$) smaller (532±98 μV) and the width of T-loop (Tw) was wider (21.24±11.48 deg) than in the control group (793±114 μV and 7.17±3.02 deg, respectively). The partial post-partum restoration of all parameters was not significant. In all groups, the QT dispersion was significantly correlated with Tw but not with Ta. According to these results we can conclude that the QT dispersion is an indirect reflection of the complete process of ventricular repolarization, reflected in the morphology of the T-loop.

Key words
QT dispersion • Electrocardigraphy • Vectorcardiography • T-loop morphology • Pregnancy

Introduction

According to experimentally obtained data (Han *et al.* 1964, Merx *et al.* 1992), increased temporal dispersion of refractoriness has been suggested to be very closely related to the increased vulnerability of the ventricular myocardium to serious tachyarrhythmias. The duration of the myocardial refractoriness (the time between depolarization of cells by an activating wavefront and repolarization) is thought to be represented by the interval between the onset of the Q wave and the end of the T wave on the ECG curve. Delayed cardiac electrical recovery (repolarization) could therefore be detectable by the prolongation of the QT interval. Each of the ECG leads is aimed to derive information from a different aspect of the ventricular myocardium. Therefore, each ECG lead represents another region of the heart muscle mass. As the myocardium is generally an inhomogeneous tissue there are regional differences in action potential duration. Therefore, one lead can represent the area of earliest repolarization by the shortest QT interval whilst another one indicates the area of the

Fax+420 249 20 590
E-mail physres@biomed.cas.cz http://www.biomed.cas.cz/physiolres

heart which is last to be depolarised by the longest QT interval (Fig. 1). The difference between them is known as QT dispersion which is thought to be representative of the overall variability in the timing of repolarization (Day *et al.* 1992).

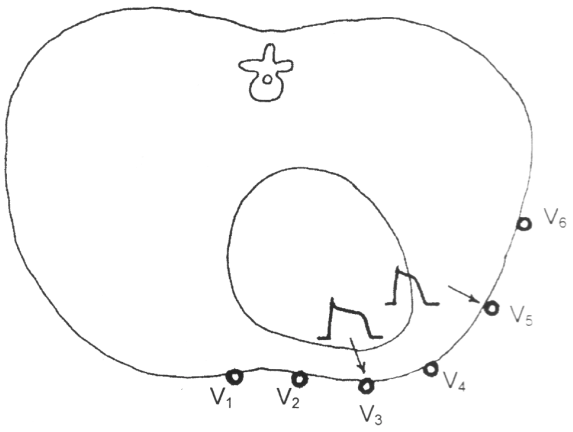

Fig. 1. *Standard interpretation of QT dispersion: Each lead represents another area of myocardium. If there is a difference in two areas as far as the duration of the action potential is concerned, there will also be a different QT interval duration on the recording leads.*

However, another concept of QT dispersion origin has been discussed recently, suggesting that rather than regional heterogeneity of myocardial repolarization, different projections of the 3-dimensional T-wave loop into individual ECG leads accounts for the possible differences in QT interval duration (Kors *et al.* 1999, Batchvarov and Malik 2000). According to this concept, there is only one end of repolarization, which is the final common termination for all leads together, when the electric field dissolves and all potential differences vanish. If the electrical potential recorded on any ECG lead equals zero this means that there is no difference between two electrodes composing the lead (including precordial leads), because the central terminal by no means constitutes a zero potential (Burger 1955). In other words, the projection of the instantaneous heart dipole is perpendicular to the given lead. Consequently, the shortest QT duration can be expected to occur in the lead where the terminal part of the T loop is perpendicular to the lead axis (Fig. 2). According to Kors *et al.* (1999) several factors come into play to explain the relation between QTd and T loop morphology. Especially two parameters of the T loop could be crucial: 1) spatial T loop amplitude (Ta) and 2) T loop width (Tw).

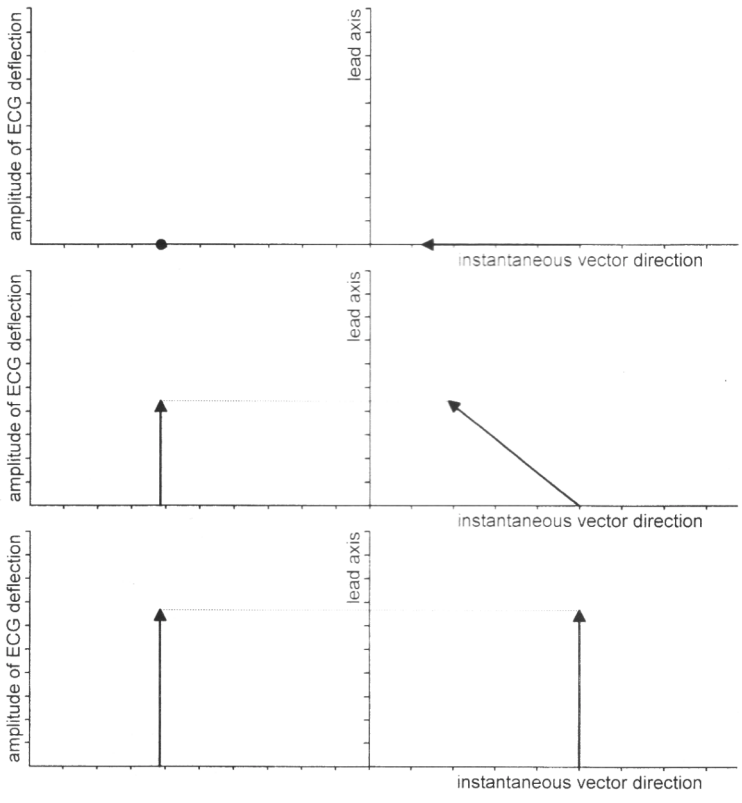

Fig. 2. *Different amplitudes registered on a lead due to different direction of instantaneous vector.*

Nevertheless, many studies have demonstrated a direct relation between increased QT dispersion and different cardiovascular pathologies. Mirvis (1985), using body surface mapping, detected increased QT dispersion in patients after acute myocardial infarction, and his finding was repeatedly confirmed (Cowan *et al.* 1988, van de Loo *et al.* 1994). Later, increased QT dispersion was found in arrhythmia patients with long QT intervals (Day *et al.* 1990, Linker *et al.* 1992, Priori *et al.* 1994), in patients with drug-induced torsades de pointes tachycardias (Hii *et al.* 1992, Hohnloser *et al.* 1993) and also in patients with lethal arrhythmias (Somberg *et al.* 1985, Barr *et al.* 1994). Sporton *et al.* demonstrated in 1997 that an acute increase in QT dispersion can be caused by induced myocardial ischaemia.

Although it seems safe to conclude on the basis of these studies that QT dispersion is of major importance in detecting repolarization abnormalities, yet its mechanism is still not clear. While QT dispersion could result from underlying heterogeneity of ventricular repolarization (i.e. as a direct measure of the recovery time dispersion), it also could result from a variable projection of single three-dimensional vector dynamics onto different ECG leads (i.e. as an indirect measure of general repolarization abnormalities).

In order to contribute to solve this question, we have used late pregnancy as a model of the physiologically changed spatial arrangement of thoracic organs accompanied by deviated morphology of the T-loop. Thus, this model has enabled us to study the relation between QT dispersion and spatial aspects of the T- loop.

Patients and Methods

Patients

Electrocardiographic recordings were obtained from 37 healthy non-obese women aged between 19-36 years with physiological pregnancy in the 36th to 40th week and in the 2nd to 6th day after spontaneous physiological delivery. The same recordings were obtained from the control group containing 18 volunteers, healthy non-pregnant, non-obese women aged between 20-36 years.

A healthy person was defined for the purposes of this project according to the following criteria: negative cardiological family and personal history, normal arterial blood pressure, normal glycemia, normal cholesterolemia, normal ECG, non-smoker, normal body weight, negative neurological and endocrinological personal history, no cardiological medication.

Measurements

The examination was carried out under standard conditions by only one examiner in order to assure full compatibility and reproducibility of the obtained results in all the examined groups. Data acquisition was performed in the morning and the examined women were allowed to relax for a few minutes before the recordings. While examined they were asked to remain still and to avoid any movement.

Electrocardiographic and vectorcardiographic recordings were obtained simultaneously using the Cardiac 112.2 device (Kittnar *et al.* 1993). The QT interval was measured from 80 unipolar chest leads used for body surface potential mapping. It was measured from the start of the Q wave to the end of the T wave, each QT interval was corrected for the patient's heart rate (QTc) using Bazett's formula (van de Loo *et al.* 1994). QT dispersion was then defined as the difference between the maximal and minimal QT interval in any of the leads measured. Accordingly, QTc dispersion was defined as the difference between maximal and minimal heart rate-corrected QT interval QTc. Vectorcardiographic recordings were obtained from Frank's orthogonal leads, angles and amplitudes of the T-loop were measured at 3 time instants (1/4, 1/2 and 3/4 of the whole T-loop duration) in all three perpendicular planes (frontal, transversal, and left sagital). From the measured data the spatial T-loop amplitude (Ta) and T-loop width (T w) were then calculated. Tw was defined as the space angle between T vectors at the 1/4 and 3/4 instants of the T-loop (Fig. 3).

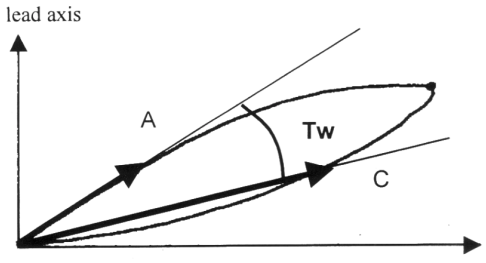

Fig. 3. *The estimation of the width of T-loop (Tw).*

Data analysis

The computer program of the Cardiac 112.2 device was used for the processing of the electrocardio-

graphic and vectorcardiographic data. This program determines the common wave onsets, offsets and amplitudes for all 95 leads in one representative beat. The set of all leads comprised: 12 standard ECG leads, 3 orthogonal Frank's vectorcardiographic leads and 80 regularly placed unipolar body surface leads (Kittnar and Šťovíček 1993).

The QT intervals were measured manually by a single observer from curves on the device screen. The cursor indicated the start of the Q wave and the end of the T wave. The curves were displayed on the screen at a speed corresponding to a paper shift of 50 mm/s and a gain of 1mV/cm. To check the reproducibility of the measurement we assessed both intraobserver and interobserver variability. For determining the intra-observer variability, all ECG tracings were evaluated by the same investigator on two different occasions. To assess the interobserver variation, all ECG tracings were analyzed by a second independent investigator who was ignorant of the results obtained by the other. T-loop parameters were also measured manually from curves on the device screen. The cursor was used to indicate the three crucial instants of the T-loop (1/4, 1/2 and 3/4 of the T-loop duration), the amplitudes and angles were then automatically calculated by the computer program.

Statistical evaluation of the assessed data was performed using the ANOVA method for repeated measurements.

Table 1. Statistically significant differences in electrocardiographic and vectorcardiographic parameters, QT and QTc dispersion and T-loop parameters (p<0,05).

	Control group (n=18)		**Late pregnancy** (n=37)		**After delivery** (n=37)	
Heart rate (1/min)	75.2	(11.5)	102.1	(18.2)	97.2	(16.3)
QT interval (ms)	365.2	(30.5)	323.7	(29.3)	334.3	(19.0)
QTc interval (ms)	405.3	(20.7)	442.3	(28.9)	418.5	(22.0)
QT dispersion (ms)	34	(12)	73	(18)	64	(20)
QTc dispersion (ms)	36	(13)	79	(21)	62	(23)
T wave$_f$ axis (deg)	27.6	(14.8)	18.8	(9.1)	16.0	(11.9)
QRS-T_f space angle (deg)	25.6	(20.9)	37.8	(19.7)	45.3	(34.0)
Ta (μV)	792.9	(104.3)	532.1	(98.4)	561.8	(117.2)
Tw (deg)	7.17	(3.02)	21.24	(11.48)	14.05	(12.32)

Numbers are the mean values± S.D., f – frontal plane, other abbreviations see text.

Results

Reproducibility of the determination of QT dispersion was high in both intraobserver and interobserver comparisons. In absolute numbers, the difference between the first and second determination of QT dispersion in the same ECG tracing (intraobserver variability) ranged between 0 and 18 ms, with an average value of 8 (±3) ms. The values for interobserver variability varied between 0 and 21ms, with a mean value of 9 (±5) ms.

The average QT dispersion (±S.D.) in the 18 normal subjects was significantly lower [34 (±12) ms] than in women in the late period of pregnancy [73 (±18) ms] (P<0.001). The results were very similar using rate corrected values with average QTc dispersion values of 36 (±13) ms for normal subjects and 79 (±21) ms for those in late pregnancy (P<0.001). In spite of the reduction of QTc interval after delivery, both QT and QTc dispersions were not significantly changed on the 2nd to 6th day after spontaneous physiological delivery and neither was there any significant change in the heart rate (Table 1).

Table 2. Parameters of the correlation between QTd and Tw

	Control group (n=18)	**Late pregnancy** (n=37)	**After delivery** (n=37)
Coefficient r	0.827	0.752	0.767
P	< 0.001	< 0.001	< 0.001
Regression equation	QTd = 18.0+2.2xTw	QTd = 60.7+0.6xTw	QTd = 51.6+0.8xTw

r - Pearson´s coefficient of correlation

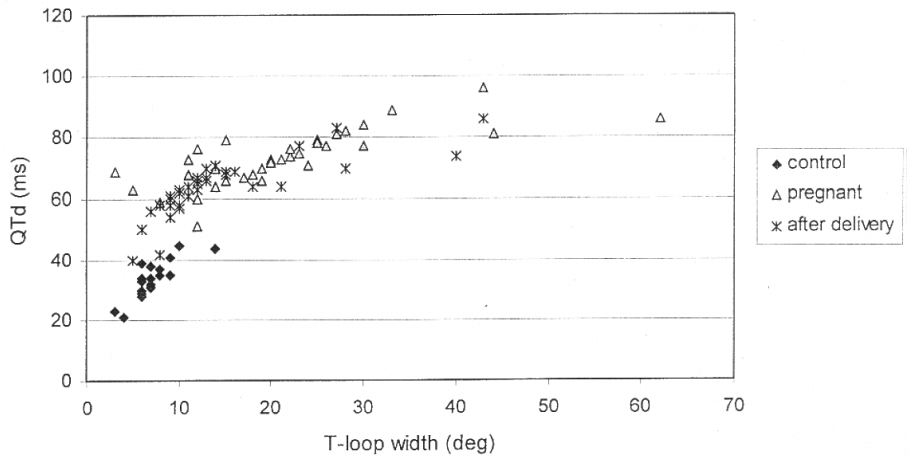

Fig. 4. *QT dispersion (QTd) versus Tw plots in the control group and in the group of pregnant women both before and after delivery.*

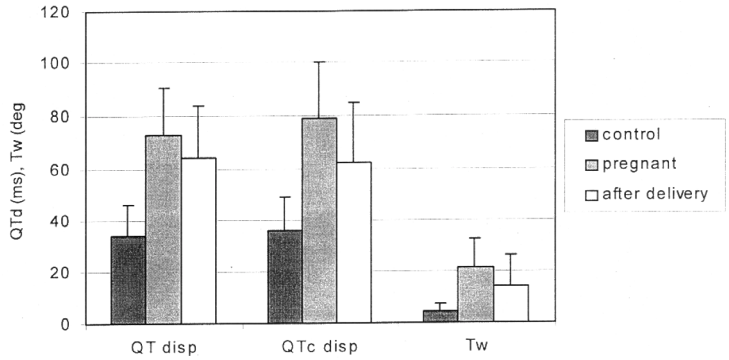

Fig. 5. *Changes of QT dispersion, QTc dispersion and Tw in late pregnancy and after delivery.*

The average Ta in women in the late period of pregnancy was significantly (P<0.001) smaller [532 (±98) μV] and the Tw wider [21.24 (±11.48) deg] than in the control group [793 (±114) μV and 7.17 (±3.02) deg respectively]. The partial post-partum restorations of both parameters were not significant (Table 1, Fig. 5). In the control group, as well as in the group of pregnant women both before and after delivery, the QT dispersion was correlated well with Tw (Table 2, Fig. 4) but not significantly with Ta.

Discussion

The present study was aimed to determine the possible changes in QT and QTc dispersion in late pregnancy and after delivery from ECG curves using body surface potential mapping. The employment f a greater number of leads for determinating QT dispersion appears to characterize the QT dispersion more accurately than assessment in only 12 or even 6 precordial leads. The use of a small number of leads was undoubtedly the

main cause of the repeatedly suggested poor reproducibility (Day *et al.* 1990, Kautzner *et al.* 1994). The enhanced accuracy for QT dispersion assessment from 12-lead ECG in comparison with only 6 precordial leads was also reported (van de Loo *et al.* 1994, Higham and Campbell 1994). Moreover, the reproducibility could be influenced by the scale of the ECG curve (paper speed and gain) and especially the lower time resolution (25 mm/s) was suggested to be an important reason for the poor reproducibility (Glancy *et al.* 1996). Both the intra- and interobserver variability of QT and QTc dispersion assessed in this study permits the use of this method to determine changes in QT dispersion as the detected changes lie well above the errors encountered in this study.

The present measurements indicate that the late pregnancy *per se* causes both a prolongation of QTc interval and an increase in the QT as well as QTc dispersion and that in spite of the reduced QTc interval both QT and QTc dispersion remain increased even after delivery (Table 1, Fig. 5). These findings are in close agreement with those of Mshui *et al.* (1999) in patients with simple obesity before and after diet therapy.

Our results suggest that it is necessary to re-evaluate the possible clinical importance of QT dispersion. While regional changes in action potential duration and conduction (increased electrical inhomogeneity of the myocardial tissue) can play an important part as QT dispersion determinants, other factors that could occur both in the heart itself and in the tissues lying between the ventricular myocardium and measuring electrodes on the body surface may well alter the QT dispersion. It must be emphasized that the surface electrocardiographic QT interval "reflects complex and interrelated aspects of cardiac electrophysiology, cardiac geometry, torso shape, tissue impedance, and biological signal processing" (Higham and Campbell 1994).

To study the possible physiological determinants of QT dispersion, we have evaluated electrocardiologic changes due to altered thoracic geometry. Changed spatial arrangement of abdominal organs during pregnancy results in increased pressure of the viscera on the diaphragm and thoracis organs. This is undoubtedly associated with the changed spatial position of the heart which is then accompanied by an "abnormal" pattern of the electrical heart field configuration, including the changed spatial relations between the heart and any ECG lead. Consequently, the morphology of vector-cardiographic loops must be changed as well. Change in the T wave axis in the frontal plane suggests a more horizontal position of the heart; this change, nevertheless, is not accompanied by a corresponding change in the QRS axis. This is apparently the cause of the increased QRS-T space angle in the frontal plane (Table 1). Our results suggest that it is necessary to re-evaluate the possible clinical importance of QT dispersion.

Nevertheless, in addition to the changed spatial arrangement of the chest organs two other factors could contribute to the electrocardiographic changes during pregnancy:

Changed electrical properties of the myocardium due to changed both sympathetic and hormonal modulation (epinephrine, progesteron) of the electrical heart activity. This can be to a certain extent in accordance with the hypothesis that increased QT dispersion is a marker of enhanced risk of arrhythmias as similar changes of neurohumoral cardiac modulation contribute very probably to such an increased risk and could also be involved in a delay of cardiac electrical recovery. This explanation appears to be in accord with our findings of changed electrical heart field in late pregnancy and after delivery (Lechmanová *et al.* 2001).

The changed impedance of tissues lying between the ventricular myocardium and measuring electrodes on the body surface is due to the gradual accumulation of sodium and total body water, most of which is extracellular during pregnancy. This explanation corresponds to our recent findings (Lechmanová *et al.* 2001) that most hemodynamic parameters changed in late pregnancy are completely restored in 4 days after delivery with only two exceptions: the heart rate and the thoracic fluid capacity. The persisting significant increase of thoracic fluid capacity can be caused by elevated blood flow in the thorax due to increased heart rate as well as by persistent increase in the total body fluid.

All electrocardigraphic changes detected in late pregnancy are not partially restored until 4 days after delivery. This finding is in accordance with not fully restored body weight within 4 days after labor, probably due both to a persisting increase of total body fluid (increased thoracic fluid capacity) and to increased sympathetic activity. The latter could explain some changes of other variables, for instance increased heart rate.

As mentioned above. it has been hypothesized recently (Kors *et al.* 1999) that two parameters of the T-loop morphology (Ta and Tw) could affect QT dispersion.

Overall decreased spatial amplitude (Ta) of the T-loop will result in decreased amplitude of the T wave

which could undoubtedly lead to increased QT dispersion because of the increased uncertainty in determining the end of low T waves (Kautzner *et al.* 1994, Murray *et al.* 1997). Although we have found increased QT dispersion corresponding to the decreased Ta in late pregnancy, there was poor correlation between both parameters.

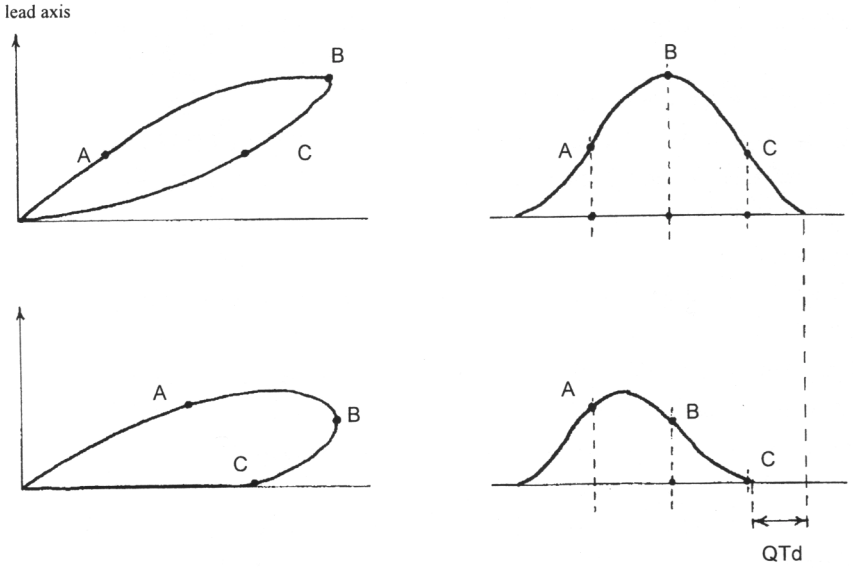

Fig. 6. *Relation between morphology of T-loop projection onto the lead axis and shortening of QT duration. Wider T-loop can have its terminal part perpendicular to lead axis, which causes a shortening of T wave (modified according to Kors et al. 1999).*

The wider the T-loop the higher the probability that the terminal T axis is perpendicular to the lead axis and consequently, the shorter the QT duration (Fig. 6). It may be questionable whether this fact alone could be sufficient to explain the difference in QT dispersion between pregnant women and the control group in our study. However, the lead axes of 80 precordial leads placed orderly on the whole torso surface are distributed in 80 different directions and they therefore receive different projections of the T-loop. The direction of the terminal T axis determines which lead will have the shortest duration and which will have the longest one. If the direction of the T-loop is changed without any change of its shape, the leads with the shortest and longest QT will change, but the difference between the longest and shortest QT (QT dispersion) will remain the same. However, if the width of the loop increases, the QT interval in corresponding leads will be shortened and the QT dispersion will be increased. This theoretical consideration is supported by the very good correlation between QT dispersion and Tw. Nevertheless, it would be erroneous to simply conclude that there is a causal relation between QT dispersion and Tw as both parameters represent an enormous simplification of the electrical heart field that is very complex and spreads towards the measuring electrodes through the inhomogeneous environment of the human thorax. In our opinion, both QT dispersion and T-loop morphology are able to reflect similar disorders of the repolarization pattern. In other words, we can conclude that the QT dispersion is an indirect reflection of the complete process of ventricular repolarization, reflected in the morphology of the T-loop. Moreover, the findings observed in our study suggest that the complexities of factors involved in the overall dispersion of repolarization needs to be explored systematically. The QT dispersion can reflect not only increased risk of serious tachyarrhythmias, especially due to myocardial ischaemia, but it was also proved in our study to be increased physiologically. Can such an increase be caused just by a changed spatial arrangement of the chest organs including the heart, or can it be due to changed patterns of neurohumoral cardiac modulation? This merits further investigation. However, we may conclude that the prolonged QT dispersion should be interpreted simply just as "an unspecific sign of changed course of repolarization".

Acknowledgements

Informed Consent

The study was performed in compliance with Human Studies Guidelines of the First Faculty of Medicine, Charles University, Prague. The Ethical Committee of the Faculty of Medicine approved all procedures used in the study. The subjects were informed about all procedures and an informed consent was obtained from all subjects involved in the study after the nature of the administrations had been explained. A preliminary communication (Lechmanová *et al.* 2000) was presented at the Meeting of Czech and Slovak Physiological Societies, Hradec Králové, February 2-4, 2000. Supported by the Grant MSM 11110008.

References

BARR CS, NAAS A, FREEMAN M, STRUTHERS AD: QT dispersion and sudden unexpected death in chronic heart failure. *Lancet* **343:** 327-329, 1994.

BATCHVAROV V, MALIK M: Measurement and interpretation of QT dispersion. *Prog Cardiovasc Dis* **42:** 325-344, 2000.

BURGER HC: The zero of potential: a persistent error. *Am Heart J* **49:** 581-586, 1955.

COWAN JC, YUSOFF K, MOORE M, AMOS PA, GOLD AE, BOURKE JP, TANSUPHASWADIKUL S, CAMPBELL RW: Importance of lead selection in QT interval measurement. *Am J Cardiol* **61:** 83-87, 1988.

DAY CP, McCOMB JM, CAMPBELL RWF: QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J* **63:** 342-344, 1990.

DAY CP, McCOMB JM, CAMPBELL RWF: QT dispersion in sinus beats and ventricular extrasystoles in normal hearts. *Br Heart J* **67:** 39-41, 1992.

GLANCY JM, WESTON PJ, BHULLAR HK, GARRATT CJ, WOODS KL, DE BONO DP: Reproducibility and automatic measurement of QT dispersion. *Eur Heart J* **17:** 1035-39, 1996.

HAN J, MOE GK: Non-uniform recovery of excitability in ventricular muscle. *Circ Res* **14:** 44-60, 1964 .

HIGHAM PD, CAMPBELL RWF: QT dispersion. *Br Heart J* **71:** 508-510, 1994.

HII JTY, WYSE GD, GILLIS AM, DUFF HJ, SOLYLO MA, MITCHELL BL: Precordial QT interval dispersion as a marker of torsades de pointes. *Circulation* **86:** 1376-82, 1992.

HOHNLOSER SH, VAN DE LOO A, KALUSCHE D, ARENDTS W, QUART B: Does sotalol-induced alteration of QT-dispersion predict drug effectiveness or proarrhythmic hazards? (abstr.) *Circulation* **88 (suppl. I):** I-397, 1993.

KAUTZNER J, GANG YI, CAMM AJ, MALIK M: Short- and long-term reproducibility of QT, QTc and QT dispersion measurement in healthy subjects. *PACE* **17:** 928-937, 1994.

KITTNAR O, SLAVÍČEK J, VÁVROVÁ M, BARNA M, DOHNALOVÁ A, MÁLKOVÁ A, ASCHERMANN M, HUMHAL J, HRADEC J, KRÁL J: Repolarization pattern of body surface potential maps (BSPM) in coronary artery disease. *Physiol Res* **42:** 123-130, 1993.

KITTNAR O, ŠŤOVÍČEK P: Contemporary body surface potential mapping in electrocardiology and its perspectives. *Physiol Res* **42:** 141-143, 1993.

KORS JA, van HERPEN G, van BEMMEL JH: QT dispersion as an attribute of T loop morphology. *Circulation* **99:** 1458-1463, 1999.

LECHMANOVÁ M, PAŘÍZEK A, HALAŠKA M, SLAVÍČEK J, KITTNAR O: Changes of the electrical heart field and hemodynamic parameters in the 34th to 40th weeks of pregnancy and after delivery. *Arch Gynecol Obstet*, in press, 2001.

LECHMANOVÁ M, KITTNAR O, SLAVÍČEK J, PAŘÍZEK A: Electrical heart field configuration and QT dispersion in the late pregnancy and after delivery. *Physiol Res* **50:** P16, 2000.

LINKER NJ, COLONNA P, KEKWICK CA, TILL J, CAMM AJ, WARD DE: Assessment of QT dispersion in symptomatic patients with congenital long QT syndromes. *Am J Cardiol* **69:** 634-638, 1992.

MERX W, YOON MS, HAN J: The role of local disparity in conduction and recovery time on ventricular vulnerability to fibrilation. *Am Heart J* **94:** 603-610, 1977.

MIRVIS DM: Spatial variation of the QT intervals in normal persons and patients with acute myocardial infarction. J *Am Coll Cardiol* **5:** 625-631, 1985.

MSHUI ME, SAIKAWA T, ITO K, HARA M, SAKATA T: QT interval and QT dispersion before and after diet therapy in patients with simple obesity. *Proc Soc Exp Biol Med* **220:** 133-138, 1999.

MURRAY A, McLAUGHLIN NB, CAMPBELL RWF: Measuring QT dispersion: man versus machine. *Heart* **77:** 539-542, 1997.

PRIORI SG, NAPOLITANO C, DIEHL L, SCHWARTZ PJ: Dispersion of the QT interval: a marker of therapeutic efficacy in the long QT syndrome. *Circulation* **89:** 1681-89, 1994.

SOMBERG J, TEPPER D, WYNN J: Prolonged repolarization: a historical perspective. *Am Heart J* **109:** 395-398, 1985.

SPORTON SC, TAGGART P, SUTTON PM, WALKER JM, HARDMAN SM: Acute ischaemia: a dynamic influence on QT dispersion. *Lancet* **349:** 306-309, 1997.

VAN DE LOO A, ARENDTS W, HOHNLOSER SH: Variability of QT dispersion measurements in the surface electrocardiogram in patients with acute myocardial infarction and in normal subjects. *Am J Cardiol* **74:** 1113-1118, 1994.

Reprint requests

O. Kittnar, Institute of Physiology, First Faculty of Medicine, Charles University, Albertov 5, 128 00 Prague 2, Czech Republic. e-mail: okittnar@lf1.cuni.cz