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QT dispersion and maximum QT interval – an attempt at complex analysis of the parameters in patients with previous myocardial infarction

The phenomenon of inter-lead variability of QT intervals in a standard 12-lead surface electrocardiogram (ECG) was first noticed and reported by Campbell et al. (3) in 1985. Five years later Day et al. (6) put forward a hypothesis that this observation might represent regional, physiologic inhomogeneity of myocardial refractoriness rather than be a pure recording artifact, which was later substantiated by animal experiments conducted by Zabel et al. (42). Thus, the concept of a new electrocardiographical index was conceived and subsequently named 'QT dispersion'.

The QT interval dispersion is defined as the difference between the maximum and the minimum QT interval duration in a standard 12-lead ECG tracing and indirectly reflects differences in repolarisation period of particular areas in ventricular myocardium (28, 32).

Since the initial research, growing attention has been focused on the QT dispersion phenomenon, which resulted in revealing that a scope of abnormal conditions can be characterised by an increase in QT dispersion values. These, most of all, include diverse heart muscle pathology, yet they are not restricted solely to cardiovascular diseases. Diabetes (25) is a case in point, but also chronic obstructive pulmonary disease (30), rheumatoid arthritis (12), Duchenne muscular dystrophy (41), though a plethora of others may still remain unknown. As far as cardiovascular conditions are concerned, increased QT dispersion can be associated with the following: chronic heart failure (2), hypertrophic cardiomyopathy (8), congenital heart diseases (28) (e.g. mitral valve prolapse, tetralogy of Fallot), hypertension (37), ischaemic heart disease (31, 40) and acute myocardial infarction (9, 11, 15, 23, 26, 33).

The effect of time on QT dispersion in myocardial infarction plays an important role. During the first few days, QT dispersion increases with time elapsing since the onset of the acute coronary syndrome and after the acute phase, usually decreases (11, 15, 26). Nonetheless, this course of events can be modified by the agents such as ACE inhibitors, β-blockers, rest, successful thrombolysis and angioplasty which reduce QT dispersion and others like restenosis and exercise, which tend to act the opposite (28). The processes lying behind these changes in QT dispersion along with time are probably due to a sequence of alterations in the structure of myocardium occurring during the acute phase and recovery (i.e. ischaemia, necrosis, scar). Diverse spatial distribution of these pathologic areas as well as the fact that all of them can coexist in one myocardium may lead to inhomogeneity and loss of synchronization in the repolarisation process. That, in turn, is likely to additionally aggravate the Autonomic Nervous System dysfunction.

Recent research suggests that the QT dispersion increase in patients after myocardial infarction may correlate with incidence of life-threatening ventricular heart rhythm disorders or higher risk of sudden cardiac death (23, 33). Despite the doubtless fact that QT dispersion decreases in the recovery phase of acute myocardial infarction, it is still argued whether it ever manages to reach the values typical of healthy population. Therefore, we set out to examine whether the increased QT dispersion is still present in patients who had developed myocardial infarction at least one year prior to our examination.

The aim of this study was to determine whether or not any difference can be traced in values of QT interval dispersion (QTd), corrected QT interval dispersion (QTdc), maximum QT interval (QT_{max}) and corrected maximum QT interval (QTc_{max}) in patients with previous myocardial infarction compared to the respective values in healthy population. Furthermore, the differences in QTd, QTdc, QT_{max} and QTc_{max} were assessed regarding age, gender and myocardial infarction location.

MATERIAL AND METHODS

The research was carried out on a group of 28 patients aged 48 to 84 having referred to a Cardiologic Consulting Room organized as part of the Socio-Scientific Camp (Zwierzyniec, July 2004), each at least one year after having developed a myocardial infarction. The study group was, subsequently, divided into 2 subgroups according to the location of the myocardial infarction – 13 patients aged 49 to 72 after anterior wall or anteroseptal infarction and 15 patients aged 48 to 82 after inferior myocardial infarction. In order to determine QTd, QTdc, QT_{max} and QTc_{max} values in healthy population, further to be compared with respective values in the study groups, a control group of 26 healthy people aged 27 to 73 was selected (Table 1).

Table 1. Characteristics of the study and control groups

	Myocardial infarction groups			Control group
	total	anterior MI	inferior MI	
Number of patients	28	13	15	26
Age (yr, mean ± SD)	64.4 ± 10.54	63.7 ± 10.7	65.0 ± 11.1	49.8 ± 17.2
Gender				
male	19	11	8	8
female	9	2	7	18
RR interval (ms, mean ± SD)	831.7 ± 179.2	842.1 ± 220.3	822.6 ± 141.9	916.2 ± 148.7
Heart rate (beats/min, mean ± SD)	76 ± 19	77 ± 23	75 ± 15	68 ± 14

Moreover, patients from the myocardial infarction group and the control group were arranged into 4 subgroups in terms of gender with the aim of establishing whether it exerts any influence on the QT dispersion values (Table 2).

Table 2. Characteristics of the MI group and the control group in terms of gender

	MI males	MI females	Healthy males	Healthy females
Number of patients	19	9	8	18
Age (yr, mean ± SD)	64 ± 10.6	65.2 ± 11.5	47.8 ± 10.7	50.7 ± 14.1
RR interval (ms, mean ± SD)	823.2 ± 189.8	849.5 ± 163.6	979.8 ± 119.7	887.9 ± 154.5
Heart rate (beats/min, mean ± SD)	77.3 ± 20.7	73.1 ± 14.3	62.0 ± 7.5	70.0 ± 15.1

The following stage of the study involved establishing existence of any changes in the QT dispersion occurring along with age. A new division of patients was set to serve this purpose (Table 3).

Table 3. Characteristics of the MI group and the control group in terms of age

	Healthy group < 50 yr	Healthy group ≥ 50 yr	MI group < 60 yr	MI group ≥ 60 yr
Number of patients	11	15	11	17
Age (yr, mean ± SD)	37.9 ± 8.6	58.5 ± 7.4	53 ± 4.1	71.8 ± 6.1
Gender				
male	4	4	5	14
female	7	11	6	3
RR interval (ms, mean ± SD)	908.2 ± 178.5	922.0 ± 128.9	810.5 ± 132.2	845.4 ± 206.8
Heart rate (beats/min, mean ± SD)	68.9 ± 16.1	66.6 ± 11.9	75.8 ± 12.0	76.0 ± 22.4

Individuals with heart rhythm disorders in ECG tracings or in anamnesis, as well as those undergoing antyarrhythmic therapy with sodium or potassium channel blocking drugs, were excluded from all the study and the control groups. For the QT intervals measuring purposes, we applied a half-automated procedure conducted as follows. The QT interval dispersion in patients from all groups was evaluated in standard 12-lead ECG tracings which had been recorded using the Medea computer set. The ECG tracings with the leading sinus rhythm, exclusively, were analysed. In order to perform the measurements, we implemented the M_EKG software. This software permits precise calculations of the QT intervals duration times on the basis of an electrocardiographical chart of a computer-performed cycle averaging. Nonetheless, this method is not devoid of potential human errors projecting onto the ultimate measurement result as the application requires manual placement of the interval markers further to be used in computer calculations of the intervals, thus making them vulnerable to some inaccuracies. Whereas usually no considerable problem exists in terms of determining the onset of the QT interval, yet some difficulties emerge as far as the end point of the interval is concerned. Regarding that, certain rules for establishing the end point of the QT interval, which have already proved to be efficient enough in this respect, were adopted in order to avoid the risk of the markers' misplacement as well as to provide sufficient reproducibility:

- in tracings with either negative (inverted) or no visible U wave, the end of the QT interval was marked by the intersection of the descending arm of a T wave with the isoelectric line,
- a similar rule was also the case with biphasic (negative-positive) TU complexes on the stipulation that the marker position was set in point where the ascending instead of the descending arm of a T wave intersected the isoelectric line,
- if the normal fall of the descending arm of a T wave was disrupted by the onset of a U wave, the marker was placed on the intersection of the tangent line to the final segment of the descending arm of the T wave with the hypothetical isoelectric line.

Those leads in which the determination of the onset or the end of the QT interval according to the above rules was impossible or likely to be unreliable were omitted. Aditionally, only those tracings in which the quality of the tracing as well as the morphology of QT intervals in at least ten out of twelve leads permitted reliable assessment were subjected to further analysis.

On account of numerous reports on physiological diurnal changes in the QT interval dispersion, all the ECG tracings were recorded at the same time of the day, i.e. between 9 a.m. and 1 p.m., which was to prevent these diurnal fluctuations from influencing and, eventually, disturbing the results. Also, in order to avoid the risk of any measurement inaccuracies resulting from misplacement of markers as well as to provide sufficient reproducibility, the measurements were performed by

two people working independently. The values obtained this way were averaged respectively and subsequently used in calculations.

The following indices were calculated: maximum QT interval (QT_{max}), corrected maximum QT interval (QTc_{max}), minimum QT interval (QT_{min}), corrected minimum QT interval (QTc_{min}), QT interval dispersion (QTd) and corrected QT interval dispersion (QTdc). Since the duration of QT intervals varies with heart rate, it is necessary to correct those values with some correction formula in order to make them comparable. We employed the Bazett's formula:

$$QTc = \frac{QT}{\sqrt{RR}};\ QTc - \text{corrected QT interval, } QT - \text{QT interval, } RR - \text{mean RR interval (sec), } HR - \text{heart rate}$$

The values of the analysed variables are presented as mean value ± standard deviation. Distributions of the variables were checked using the Shapiro-Wilk normality test and homogeneity of variance with the F and the Levene tests. Provided the criteria of both normal distribution and homogeneity of variance were fulfilled, the unpaired two-sample Student t test was applied to compare two studied groups, in other cases we employed the Mann-Whitney U test. Whenever more than two groups were subjected to comparisons, the tests were performed using analysis of variance (ANOVA) and the *post hoc* Scheffé test. A probability value of $p < 0.05$ was considered significant.

RESULTS

In the first stage of the study we compared 28 patients after myocardial infarction regardless of its location and 26 representatives of healthy population, all characterized in Table 1, in terms of QT dispersion and maximum QT interval. The mean values of QTd and QT_{max} before and after correction as well as the results of statistical analysis are shown in Table 4 and Figure 1. The values of QTd and QTdc were significantly greater in the MI group with $p < 0.001$. Although no statistical difference was observed in QT_{max}, the difference in QTc_{max} proved to be significant ($p < 0.01$).

Table 4. QTd, QTcd, QT_{max} and QTc_{max} in MI and control groups

	MI group	Control group	p
QTd	74.1 ± 24.9	49.6 ± 19.2	0.000184
QTdc	83.3 ± 32.1	51.5 ± 17.8	0.000045
QT_{max}	437.4 ± 56.3	433.6 ± 43.2	0.77837
QTc_{max}	482.9 ± 37.5	454.5 ± 22.7	0.001566

When the location of the myocardial infarction was taken into consideration, QT dispersion (both corrected and uncorrected) differed significantly between the anterior MI group and the control group as well as between the inferior MI group and control group, whereas the differences in QTd and QTdc between the anterior and inferior MI groups failed to demonstrate any statistical significance. It is, however, worth pointing out that the mean values of QT interval dispersion tended to be greater in the anterior MI group compared to those in the inferior MI group.

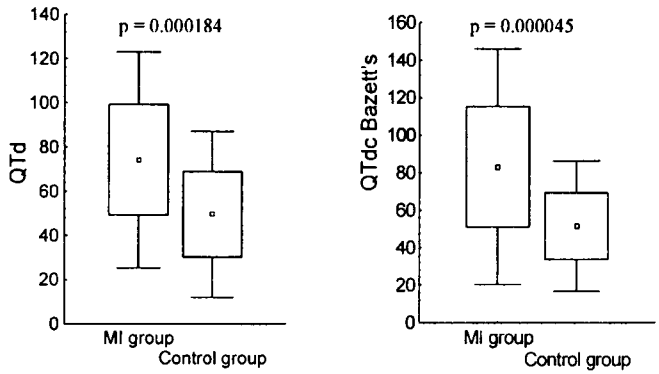

Fig. 1. QTd and QTdc in MI and control groups

As for the maximum QT interval, the values showed no correlation with the myocardial infarction location except for QTc_{max} in the anterior MI patients which, exclusively, was significantly greater compared to the respective value in the control group ($p < 0.05$). Again, despite the lack of statistical differences in QT_{max} between the two study groups as was the case with QTd, the mean QT_{max} values in the anterior MI group were greater than respective values in the inferior MI group. The characteristics of patients from all the three groups are displayed in Table 1 and the results of the above comparisons are gathered in Table 5 and Figure 2.

Table 5. QTd, QTdc, QT_{max} and QTc_{max} in anterior MI, inferior MI and control groups

	Anterior MI group (1)	Inferior MI group (2)	Control group (3)	p 1 vs 3	p 2 vs 3	p 1 vs 2
QTd	77.9 ± 22.8	70.8 ± 22.5	49.6 ± 19.2	0.002176	0.019541	0.703753
QTdc	88.7 ± 39.5	78.5 ± 24.4	51.5 ± 17.8	0.000533	0.009811	0.592059
QT_{max}	442.6 ± 69.4	432.9 ± 43.9	433.6 ± 43.2	0.870778	0.999282	0.880726
QTc_{max}	486.5 ± 40.9	479.8 ± 35.4	454.5 ± 22.7	0.016063	0.054878	0.853870

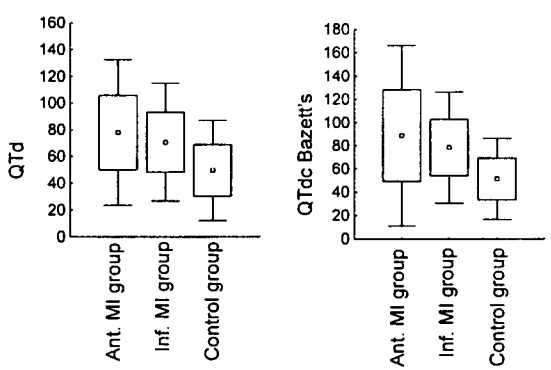

Fig. 2. QTd and QTdc in anterior MI, inferior MI and control groups

Another factor generally suspected of exerting certain influence on QT interval dispersion is gender. In this respect, the data obtained in this study seemed encouraging, at first. The mean values of QTd and QTdc tended to be greater in the MI females group compared to the MI males group while in healthy population, they took a reverse order (QTd and QTdc were greater in healthy males). Yet the application of the Analysis of Variance failed to statistically support this observation as being of significance in both comparisons. Surprisingly, the result of statistical analysis was no different when the MI and the healthy males were compared though, again, the mean QT dispersion value as well as all the mean corrected QT dispersion values presented a tendency to be greater in men after myocardial infarction. However, some statistically significant results ($p < 0.01$) were brought by the comparison of QTd and QTdc in females after myocardial infarction and females free of any cardiovascular diseases with all the values significantly smaller in the latter group.

As we analysed the QT_{max} and QTc_{max} values, they did not fall into any logical pattern nor did they manifest any statistically significant differences.

Table 2 shows the main characteristics of the patients from the four investigated groups and Table 6 discloses mean values of the studied indices and the results of all the comparisons performed in order to determine the effect of gender on QT dispersion and maximum QT interval in two populations (Myocardial Infarction and Healthy).

Table 6. QTd, QTdc, QT_{max} and QTc_{max} in MI and healthy patients grouped regarding gender

	MI males (1)	MI females (2)	Healthy males (3)	Healthy females (4)	p 1 vs 2	p 3 vs 4	p 1 vs 3	p 2 vs 4
QTd	70.5 ± 25.7	81.7 ± 22.6	56.4 ± 27.4	46.6 ± 14.2	0.671717	0.778598	0.525347	0.004085
QTdc	79.7 ± 33.5	90.7 ± 29.3	56.6 ± 25.8	49.2 ± 13.1	0.788736	0.933185	0.238392	0.004332
QT_{max}	430.8 ± 56.4	451.4 ± 56.6	439.7 ± 38.5	430.8 ± 45.9	0.800520	0.982229	0.981776	0.804846
QTc_{max}	478.8 ± 37.8	491.7 ± 37.6	444.7 ± 23.5	458.9 ± 21.5	0.792304	0.766859	0.094953	0.098819

The analysis of the data received after arranging the patients from the healthy population and those affected by myocardial infarction into age groups revealed no statistically significant differences between the two age groups in both populations.

The essential characteristics of patients from the studied age groups are presented in Table 3. Table 7 displays all the results obtained after analysis and comparisons of the relevant data in terms of patients' age.

Table 7. QTd, QTdc, QT_{max} and QTc_{max} in different age groups of the MI and healthy populations

	MI groups		Healthy groups		p 1 vs 2	p 3 vs 4
	< 60 yr (1)	≥ 60 yr (2)	< 50 yr (3)	≥ 50 yr (4)		
QTd	78.9 ± 22.9	71.0 ± 26.4	46.3 ± 14.7	51.9 ± 22.1	0.427300	0.469891
QTdc	88.6 ± 27.7	79.8 ± 35.0	48.2 ± 12.5	53.9 ± 20.9	0.489887	0.426485
QT_{max}	438.4 ± 44.4	436.8 ± 64.1	429.1 ± 47.8	436.9 ± 40.8	0.944716	0.657537
QTc_{max}	488.9 ± 37.4	479.1 ± 38.2	452.5 ± 20.5	456.0 ± 24.7	0.511667	0.701968

DISCUSSION

The main challenge which our study faced was to reveal any tendencies of QT dispersion in patients after myocardial infarction to take on any abnormal values. Yet, its physiological range is still a matter of contention in numerous publications (28, 32). This is partly due to different measurement techniques employed, namely automatic, manual and many modifications of these two basic methods (4, 11, 14, 16, 21, 24), and partly due to the controversy of T wave end definition (16, 28, 32). We decided to apply the so-called tangent method in identifying T wave end which seems to be slowly gaining advantage over the second most commonly used definition assuming the nadir between T and U waves as the boundary of QT intervals (28, 32). Also, our choice of manual measurement was dictated by the McLaughlin's et al. (22) claim of automatic techniques being less accurate in cardiac patients and the implication made by Charbit et al. (4) of the need to additionally verify tracings of patients with abnormal QT intervals when measured automatically.

The range of normal values of QT dispersion encountered in representatives of healthy population varies considerably between different studies owing to the reasons stated above. However, certain general conclusions can be drawn from a large collection of available data as was the case with the hypothesis formed by Sahu et al. (28) based on the analysis performed by Statters et al. (34) and Surawicz et al. (35). The latter found the average value of QT dispersion in normal subjects being less than 40 ms in 13 studies but exceeding 40 ms in 8 studies. Taking both the research works mentioned into consideration, Sahu concluded that QT dispersion in healthy individuals should not go beyond 50 ms. Soler et al. (32) approaches the problem more strictly setting the physiological range within the limit of 40–50 ms whereas Surawicz et al. (35) proposed that 65 ms might be a reasonable upper limit of normality and what exceeds that value up to 115 ms, most certainly, conveys the information of structural heart disease and carries a risk of ventricular arrhythmia or sudden cardiac death.

In our study, the average QT dispersion in the control group amounted to 49.6±19.2 ms before correction and 51.5±17.8 ms when corrected using the Bazett's formula which stays in perfect accord with the research of Hashimoto et al. (13) who found this corrected index to be 53.0±17.6 ms in their control population. What entitles us to make such a comparison is the fact that in both studies similar methods of measurement and similar criteria of establishing the end of T waves were employed. However, most of other extensive studies on the subject of QT dispersion show lower values. This may result from application of fully automated measurement techniques in most of them and different criteria of determining the T waves end.

It is worth pointing out that QT dispersion is not a constant value in an individual but it undergoes significant changes throughout the day. As observed by Yetkin et al. (39), QT dispersion is the most prominent in the morning hours, but is also increased in the afternoon. Taking that into consideration, all the recordings of ECG tracings in our study were performed from 9 a.m. to 1 p.m. Mention was made earlier of the variety of diseases associated with increased QT dispersion. The reports on the subject concern, most of all, patients affected with coronary artery disease and acute myocardial infarction.

Regretfully, patients in studies of acute myocardial infarction are not usually followed beyond the day of discharge. Nonetheless, there is enough evidence for claiming that QT dispersion continues to maintain its abnormal values at least a year after the onset of the coronary incident, despite the fact that isolated discrepancies occur in the specialist literature. An example of such may be the work of Murray et al. (24) who, admittedly, found greater QT dispersion in postinfarct patients compared to healthy population, yet the difference failed to prove any statistical significance. This is absolutely contradicted by Macfarlane et al. (19) whose research on a group of 361 individuals previously affected by myocardial infarction reveals that QTd in those patients is not only greater but also the divergence of the results between studied

groups holds extremely high significance ($p<0.0001$). The application of a fully automated method of measurement by Macfarlane renders the numerical values of QTd completely incomparable with those obtained in our study though the conclusions which can be drown from the distribution of the results coincide. Another aspect which both works share is the similar level of probability.

Hashimoto et al. (13), who adopted similar to ours methodology of measurement, divided patients into two groups in terms of location of myocardial infarction. The values of QT dispersion corrected with the Bazett's formula in that study in patients with anterior MI, inferior MI and healthy individuals were 69.9±21.5, 62.2±26.9 and 53.0±17.6 respectively which stays close to our own results – 88.7±39.5, 78.5±24.4 and 51.5±17.8 respectively. While in Hashimoto's study the only statistical difference can be observed between the anterior MI group and the control subjects, the overall outcome of our work is that regardless of the location of myocardial infarction QTd is significantly greater in comparison with healthy subjects. At the same time, mean QT dispersion does not differ significantly between the anterior and the inferior MI group, albeit a tendency of QTd to be slightly higher in patients with previous anterior MI is discernible in both studies. The finding is also substantiated by Macfarlane et al. (19), while Średniawa et al. (36) report the difference to be statistically significant. This may rather result from the ECG lead placement than from any underlying pathophysiological process, since the anterior wall of heart muscle is represented in more leads of standard electrocardiogram than the inferior wall is.

Many research projects have been designed to identify agents having impact on QT dispersion in myocardial infarction patients. These also included factors which cannot be influenced such as age and gender. All unanimously agree to the point that QT dispersion is neither age- nor gender--dependent which is also consistent with our own findings.

More interestingly though, when patients were grouped according to their gender, it was discovered in our study that the only significant difference existed between healthy and MI affected women. That suggests that the difference in QTd between the total MI and control groups may, to a large extent, result from disparity between healthy women and those having previously developed myocardial infarction. Such a distribution of results in women might be caused by certain, still unknown disposition of female myocardium towards greater viability when afflicted with ischaemia or infarction. This hypothesis, however, requires thorough verification on a much larger sample.

As far as maximum QT interval is concerned, after numerous comparisons we performed, the sole considerable relationship was between the control group and individuals with previous myocardial infarction. However, the relationship was statistically significant only when the intervals were corrected with Bazett's formula but not before correction. Hashimoto et al. (13), dividing patients in terms of infarction location, found that maximum QT interval was significantly greater in the anterior MI patients compared to the control group. The QT intervals, in that study, were corrected with Bazett's formula which, as it has already been mentioned, is strongly criticized in many publications. In our work the statistical significance also existed when the intervals were corrected with Bazett's formula but not with other formulae or uncorrected which renders the findings of Hashimoto et al. (13) questionable. Again, this controversy requires further investigation on a larger group of patients.

STUDY LIMITATIONS

The main limitation of our study was a relatively small sample size. This is due to the fact that patients undergoing antiarrhythmic therapy were not included. Additionally, the criterion of exclusion of those ECG tracings where assessment of QT intervals was possible in less than 10 leads was also a major stumbling-block.

CONCLUSIONS

In conclusion, in patients with previous myocardial infarction values of QT dispersion and corrected QT dispersion are significantly greater compared to the control patients. Also, anterior MI and inferior MI groups revealed statistical differences in QTd and QTdc in comparison with the control group but not when compared with each other. As to maximum QT interval, the values of the parameter were significantly greater in all patients after myocardial infarction and patients with anterior MI only when corrected with the Bazett's formula. No differences in QTd, QTdc, QT_{max} and QTc_{max} were discovered in terms of age and gender.

The issue of QT dispersion and maximum QT interval is still a matter of controversy in many aspects and requires further investigation, especially as to its predictive value and clinical utility. The thing of utmost importance is establishing certain standards of QT interval measurement and referential values of QTd and QT_{max}. Our opinion is, however, that QT dispersion might be used for monitoring the effects of treatment of individual patients with myocardial infarction.

REFERENCES

1. Aytemir K. et al.: Comparison of formulae for heart rate correction of QT interval in exercise electrocardiograms. Pacing Clin. Electrophysiol., 22, 1397, 1999.
2. Barr C. S. et al.: QT dispersion and sudden unexpected death in chronic heart failure. Lancet, 343, 327, 1994.
3. Campbell R. W.: Measurement of the QT interval. Eur. Heart J. 6 (suppl.), 81, 1985.
4. Charbit B. et al.: QT interval measurement: evaluation of automatic QTc measurement and new simple method to calculate and interpret corrected QT interval. Anesthesiology, 104, 255, 2006.
5. Davey P.: A new physiological method for heart rate correction of the QT interval. Heart, 82, 183, 1999.
6. Day C. P. et al.: QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. Br. Heart J., 63, 342, 1990.
7. Dogan A. et al.: Comparison of the four formulas of adjusting QT interval for the heart rate in the middle-aged healthy Turkish men. Ann. Noninvasive Electrocardiol., 10, 134, 2005.
8. Dritsas A. et al.: QT-interval abnormalities in hypertrophic cardiomyopathy. Clin. Cardiol., 15, 739, 1992.
9. Gabrielli F., Balzotti L., Bandiera A.: QT dispersion variability and myocardial viability in acute myocardial infarction. Int. J. Cardiol., 61, 61, 1997.
10. Gang Y. et al.: Computerised measurements of QT dispersion in healthy subjects. Heart, 80, 459, 1998.
11. Glancy J. M. et al.: Dynamics of QT dispersion during myocardial infarction and ischaemia. Int. J. Cardiol., 57, 55, 1996.
12. Goldeli O., Dursun E., Komsuoglu B.: Dispersion of ventricular repolarisation: a new marker of ventricular arrhythmias in patients with rheumatoid arthritis. J. Rheumatol., 25, 447, 1998.
13. Hashimoto N. et al.: Relationship between infarction location and size to QT dispersion in patients with chronic myocardial infarction. Jpn. Heart J., 43, 455, 2002.

14. Hunt A. C.: Accuracy of popular automatic QT interval algorithms assessed by a 'gold standard' and comparison with a Novel method: computer simulation study. BMC Cardiovasc. Disord., 5, 29, 2005.
15. Kabakci G. et al.: What is the optimal evaluation time of the QT dispersion after acute myocardial infarction for the risk stratification? Angiology, 52, 463, 2001.
16. Kautzner J.: QT interval measurements. Card. Electrophysiol. Rev., 6, 273, 2002.
17. Kobusiak-Prokopowicz M. et al.: Roczna obserwacja dyspersji odstępu QT całkowitej i skorygowanej u chorych z przebytym zawałem mięśnia serca. Przeg. Lek., 60, 85, 2003.
18. Luo S. et al.: A comparison of commonly used QT correction formulae: the effect of heart rate on the QTc of normal ECGs. J. Electrocardiol., 37 Suppl., 81, 2004.
19. Macfarlane P. W. et al.: Influence of lead selection and population on automated measurement of QT dispersion. Circulation, 98, 2160, 1998.
20. Malik M. et al.: Relation between QT and RR intervals is highly individual among healthy subjects: implications for heart rate correction of the QT interval. Heart, 87, 220, 2002.
21. Malik M., Batchvarov V. N.: Measurement, interpretation and clinical potential of QT dispersion. J. Am. Coll. Cardiol., 36, 1749, 2000.
22. McLaughlin N. B., Campbell R. W., Murray A.: Accuracy of four automatic QT measurement techniques in cardiac patients and healthy subjects. Heart, 76, 422, 1996.
23. Mulay D. V., Quadri S. M.: QT dispersion and early arrhythmic risk in acute myocardial infarction. Indian Heart J., 56, 636, 2004.
24. Murray A., McLaughlin N. B., Campbell R. W.: Measuring QT dispersion: man versus machine. Heart, 77, 539, 1997.
25. Naas A. A. et al.: QT and QTc dispersion are accurate predictors of cardiac death in newly diagnosed non-insulin dependent diabetes: cohort study. BR Med. J., 316, 745, 1998.
26. Parale G. P., Adnaik A. R., Kulkarni P. M.: Dynamics of QT dispersion in acute myocardial infarction. Indian Heart J., 55, 628, 2003.
27. Puddu P. E. et al.: Evaluation of 10 QT prediction formulas in 881 middle-aged men from the seven countries study: emphasis on the cubic root Fridericia's equation. J. Electrocardiol., 21, 219, 1988.
28. Sahu P. et al.: QT dispersion in medicine: electrophysiological Holy Grail or fool's gold? QJM, 93, 425, 2000.
29. Sarma J. S. et al.: An exponential formula for heart rate dependence of QT interval during exercise and cardiac pacing in humans: reevaluation of Bazett's formula. Am. J. Cardiol., 54, 103, 1984.
30. Sarubbi B., Esposito V., Ducceschi V. et al. Effect of blood gas derangement on QTc dispersion in severe chronic obstructive pulmonary disease: evidence of an electropathy? Int. J. Cardiol., 58, 287, 1997.
31. Sheehan J. et al.: QT dispersion, QT maximum and risk o cardiac death in the Caerphilly Heart Study. Eur. J. Cardiovasc. Prev. Rehabil., 11, 63, 2004.
32. Soler-Soler J., Galve E.: QT dispersion after myocardial infarction with heart failure: additional prognostic marker? Eur. Heart J., 20, 1146, 1999.
33. Spargias K. S., Lindsay S. J., Kawar G. I.: QT dispersion as a predictor of long-term mortality in patients with acute myocardial infarction and clinical evidence of heart failure. Eur. Heart J., 20, 1158, 1999.
34. Statters D. J. et al.: QT dispersion: problems of methodology and clinical significance. J. Cardiovasc. Electrophysiol., 5, 672, 1994.

35. Surawicz B.: Will QT dispersion play a role in clinical decision-making? J. Cardiovasc. Electrophysiol., 7, 777, 1996.
36. Średniawa B., Jarski P. et al.: Dyspersia QT u chorych po zawale serca w zależności od zachowanej żywotności mięśnia sercowego. Kardiol. Pol., 56, 399, 2002.
37. Tomiyama H. et al.: Left ventricular geometric patterns and QT dispersion in borderline and mild hypertension: their evolution and regression. Am. J. Hypertens., 11, 286, 1998.
38. van de Loo A., Arendts W., Hohnloser S. H.: Variability of QT dispersion measurements in the surface electrocardiogram in patients with acute myocardial infarction and in normal subjects. Am. J. Cardiol., 74, 1113, 1994.
39. Yetkin E. et al.: Diurnal variation of QT dispersion in patients with and without coronary artery disease. Angiology, 52, 311, 2001.
40. Yilmaz R., Demirbag R., Gur M.: The association of QT dispersion and QT dispersion ratio with extent and severity of coronary artery disease. Ann Noninvasive Electrocardiol., 11, 43, 2006.
41. Yotsukura M., Yamamoto A., Kajiwara T. et al.: QT dispersion in patients with Duchenne-type progressive muscular dystrophy. Am. Heart J. 137, 672, 1999.
42. Zabel M., Portnoy S., Franz M. R.: Electrocardiographic indexes of dispersion of ventricular repolarisation: an isolated heart validation study. J. Am. Coll. Cardiol., 25, 746, 1995.
43. Zaidi M. et al.: Dispersion of ventricular repolarisation: a marker of ventricular arrhythmias in patients with previous myocardial infarction. Heart, 78, 371, 1997.

SUMMARY

QT dispersion is the difference between the maximum and the minimum QT interval in standard 12-lead ECG and reflects differences in repolarisation period of particular areas in ventricular myocardium. Its value is reported to be elevated in various cardiovascular and other diseases. The matter of QT dispersion in myocardial infarction focuses much attention and raises controversy. The aim of the study was to discover any differences in QTd, QTdc, QT_{max} and QTc_{max} in patients with previous myocardial infarction compared to normal subjects. The patients were grouped regarding presence of previous MI, its location, age and gender. The measurements were performed using a half-automated method. The above parameters were calculated and then corrected with the Bazett's correction formula. QTd as well as QTdc is significantly greater in all MI patients (74.1±24.9 vs 49.6±19.2), anterior and inferior MI subjects (77.9±22.5, 70.8±22.5 vs 49.6±19.2) compared to the control group. Moreover, QT_{max} corrected with the Bazett's formula was significantly higher in all MI patients and in anterior MI patients. In contrast, no statistically significant differences were traced in QTd between anterior and inferior MI groups, and in QT_{max} and QTd regarding age and gender.

Dyspersja odstępu QT i maksymalny odstęp QT – próba kompleksowej analizy tych parametrów u pacjentów z przebytym zawałem serca

Dyspersja QT to różnica między największym a najmniejszym odstępem QT w standardowym 12-odprowadzeniowym elektrokardiogramie. Odzwierciedla ona różnice w okresach repolaryzacji poszczególnych obszarów w mięśniu komór. Jej wartość jest podwyższona w różnych chorobach układu krążenia i innych. Dyspersja QT i jej kliniczna użyteczność skupia na sobie coraz więcej uwagi, jak również budzi kontrowersje. Celem pracy było wykazanie istnienia różnic w wartościach

QTd, QTdc, QT_{max} i QTc_{max} u pacjentów po przebytym zawale mięśnia serca w porównaniu z osobami zdrowymi. Pacjenci z zawałem zostali podzieleni na grupy zależnie od lokalizacji zawału, wieku i płci. Pomiary zostały przeprowadzone przy użyciu metody półautomatycznej, następnie policzono powyższe parametry i skorygowano je za pomocą wzoru Bazetta. Wartości QTd i QTdc były znacząco większe w całej grupie pacjentów po zawale jak też w grupie pacjentów z przednim oraz z dolnym zawałem w porównaniu z grupą kontrolną. Ponadto QTc_{max} był znacząco większy w całej grupie z zawałem oraz u pacjentów z zawałem ściany przedniej. Jednocześnie nie stwierdzono statystycznie istotnych różnic w QTd między grupą z przednim a dolnym zawałem oraz w QT_{max} i QTd w zależności od wieku i płci.