

QT and P wave dispersion

QT ve P dispersiyonu

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Heterogeneity in refractoriness and conduction velocity is the main mechanism of reentrant arrhythmias. One of the indexes which show the heterogeneity of ventricular refractoriness is QT dispersion, which is found in surface ECG leads. The most commonly used index to calculate this QT dispersion is the difference between the longest and shortest QT intervals on the 12 lead ECG, which is often adjusted for heart rate as well as number of leads sampled. Abnormally high QT dispersion has been correlated with risk of arrhythmic death in a variety of disorders (cardiac and noncardiac). Also, QT dispersion is an easy, practical and cheap parameter which has been correlated with efficacy and proarrhythmic potential of drug therapy. In this review, the measurement techniques, interpretation, clinical importance of QT dispersion and another related subject, P wave dispersion, are mentioned.

Key words : *QT dispersion, P wave dispersion, arrhythmia*

Refrakter periyodun ve iletim hızının heterojenitesi re-entran aritmilerin temel mekanizmasını oluşturur. Ventriküler refrakteritenin heterojenitesini gösteren indekslerden biri yüzey elektrokardiyogramından (EKG) elde edilen QT dispersiyonudur. QT dispersiyonunu hesaplamada en çok kullanılan yöntem 12 derivasyonlu EKG'de en uzun ve en kısa QT intervalinin farkının alınıp hıza veya kullanılan derivasyon sayısına göre düzeltilmesidir. Anormal olarak artmış QT dispersiyonu bazı hastalıklarda (kardiyak ve nonkardiyak) aritmik ölüm riskini artırır. QT dispersiyonu bazı ilaçların etkinlik ve proaritmik potansiyeli hakkında da fikir veren basit, pratik, ucuz bir parametredir. Bu derlemede QT dispersiyonunun ölçümü, yorumu, klinik önemi ve diğer bir kavram olan P dispersiyonu anlatılmıştır.

Anahtar sözcükler: *QT dispersiyonu, P dispersiyonu, aritmi*

QT dispersion

Attempts to characterize the abnormalities of ventricular repolarization from the surface electrocardiogram (ECG) can be traced back to the 1960's (1). In 1990, a report by the group of the late Professor Campbell reviewed an old idea of the interlead differences in the QT interval duration and the range of the durations, termed "QT dispersion", was proposed as an index of the spatial dispersion of the ventricular recovery times (2). It was proposed that the different ECG leads magnify the ECG signal of different myocardial regions and that, consequently, QT dispersion is almost a direct measure of the heterogeneity of myocardial repolarization. The cardiological community welcomed the idea and articles reporting QT dispersion in practically every cardiac as well as many non-cardiac diseases and syndromes have flooded the cardiological literature.

Original expression of QT dispersion

QT dispersion is the range of QT interval duration in all measurable ECG leads. It means it is the difference between longest and shortest QT interval (3). Many studies including large prospective evaluations (4,5) used the so-called "corrected QT dispersion (QTc dispersion)", i.e., the dispersion of the QT in-

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tervals corrected for heart rate by some formulas. Bazelt formula, the most accepted one, is the correction of QT interval range dividing by the square root of the R-R interval (4).

Reliability of QT dispersion assessment

Many studies have shown high inter-and intraobserver variability of manually measured QT dispersion. Relative errors of 25-40 % of inter-and intraobserver variability of manual measurement of QT dispersion have been reported (6). Substantially better reproducibility of manual measurement of QT dispersion has also been reported but a wishful bias was likely involved in these reports.

The main technical difficulty in measuring QT dispersion is very unreliable determination of T wave offset both with manual and automatic methods (7). Another one is that QT dispersion is a relatively small value compared with the QT interval. Thus, a relatively small error in QT measurement magnifies the error in QT dispersion (8). When the focus of interest is a relatively small value, the measuring tool and its resolution are most important. Manual measurement using calipers carries the highest potential for error (8). There are a few measuring methods for QT dispersion (manual measurement with caliper or ruler, application of a digitizing board with or without magnification, on screen measurement with electronic calipers, etc.). Also automated measurement systems have been developed, but problems with these systems currently exist. For manual measurement methods, increasing the paper speed is not helpful to decrease measurement error. Although it increases the resolution, increasing paper speed may make the end of the T waves more ambiguous. Another factor that can contribute to the errors made in repolarization measurements is the fact that the QT dispersion has a circadian variation. So, QT dispersion values that obtained at different times should not be compared. In a technical study, Malik and Bradford (10) showed that even the "gold standard" manual measurement using the digitizing board, can produce intraobserver variations corresponding to purely error-related QT dispersion > 40 ms. But, the currently available automated algorithms unfortunately do not perform much better than human observers (10).

Clinical studies

In 51 studies in which QT dispersion was measured in total of 8455 healthy subjects of various ages, mean QT dispersion values were found to range from 10.5 ± 10.0 ms to 71 ± 7 ms. The weighted mean \pm SD is 33.4 ± 20.3 ms (11). QT dispersion > 40 ms has 88 % sensitivity and 57 % specificity for prediction of inducibility of sustained ventricular tachycardia during an electrophysiology study (12).

Several large prospective studies published recently assessed the predictive value of QT dispersion for cardiac and all-cause mortality in the general population. In the Rotterdam Study (4), QT dispersion was found to predict cardiac mortality in a general population of 5812 adults and in the Strong Heart Study (5), the predictive value of the QTc dispersion was assessed in 1839 American Indians followed up nearly for 4 years.

In the West of Scotland Coronary Prevention Study (WOSCOPS) (13) included 6595 middle-aged men with moderately raised cholesterol but no previous Myocardial Infarction (MI), it was found that an increment of 10 ms in QT dispersion increased risk for death of coronary heart disease or nonfatal MI by 13%.

QT dispersion in cardiac disease

Majority of studies have shown that increased QT dispersion can be seen in various cardiac diseases. These are post-MI patients, patients with left ventricular hypertrophy (LVH) of various origin, patients with heart failure, including idiopathic dilated cardiomyopathy, patients with acute MI, patients with long QT syndrome of various genotype, hypertensive patients and patients with aortic stenosis.

Generally QT dispersion is increased in acute MI, although mean values from 40 ± 18 ms to 162.3 ± 64.8 ms (14). Although QT dispersion is increased in the chronic phase of MI and in other chronic forms of ischemic artery disease, there seems to be a trend towards lower values compared with the acute phase of MI (14).

Many studies tried to correlate QT dispersion with the extent or the localization of the pathological process of various diseases. Some studies have shown greater QT dispersion in anterior compared to inferior MI; correlation between QT dispersion in MI and indirect measures of infarct size, such as ejection fraction; or the amount of viable myocardium in the infarct region (15).

QT dispersion seems to undergo dynamic changes in some cardiac processes. It increases significantly during ischemia induced by balloon inflation during angioplasty, by exercise stress testing, or atrial pacing or during reperfusion following angioplasty (16).

Treatment has been shown to decrease QT dispersion. These are successful reperfusion with thrombolysis, revascularization with angioplasty or coronary artery bypass grafting surgery, treatment of heart failure with losartan, successful antihypertensive treatment of patients with LVH and beta-blocker treatment of patients with long QT syndrome (17).

QT dispersion in non-cardiac disease

Many studies have shown clinical and prognostic importance of increased QT interval and QT dispersion in various noncardiac diseases. These are type I and type II diabetes mellitus (DM), anorexia nervosa, carbon-monoxide poisoning, rheumatoid arthritis, dialysis patients, patients with electrolyte imbalance, ankylosan spondilitis, LVH of professional athletes, severe burns and recipients of renal transplantation. In type II DM, increased QT interval and QT dispersion was found to be associated with autonomic neuropathy and coronary artery disease (18). Also, increased QTc dispersion in dialysis patients has predictive value for general and cardiovascular mortality (19).

Prognostic value of QT dispersion

1. Several studies have found that patients with acute or chronic MI with ventricular arrhythmias have significantly higher QT dispersion than patients without arrhythmias (20).
2. Some studies showed that QT dispersion could predict inducibility of ventricular arrhythmias during electrophysiology study (21). Although QT dispersion is not an alternative to invasive methods of electrophysiology study, it is a useful and simple parameter for electrophysiological evaluation (8).
3. Several studies showed significant correlation between QT dispersion and outcome in patients with heart failure. Analysis from the ELITE heart failure study, in which heart failure patients treated with losartan had reduction of sudden cardiac death compared with those treated with captopril, showed that captopril but not losartan increased QT dispersion (22).
4. Several authors reported significantly higher QT dispersion in hypertrophic cardiomyopathy (HCM) patients with ventricular arrhythmias compared with those without arrhythmias (23).
5. In long-QT syndrome, patients not responding to Beta-blockers had a significantly higher QT dispersion than responders (24).
6. Effects of drugs on QT dispersion and the risk of torsades de pointes tachycardia.

QT dispersion has clinical importance for the electrocardiographic follow up of the drugs that prolong ventricular repolarization (8,25). Sicouri et al showed that an agent such as amiodarone might prolong repolarization in a more homogenous fashion. Other agent may not prolong repolarization in a similar fashion in the various tissues of the heart. Thus, the use of only QT effects is insufficient for an understanding of a pharmacologic effect on repolarization. An analysis of QT dispersion on a 24 hour basis

(26). Torres et al reported that a prolonged QT with amiodarone was associated with an improved outcome (27), but many investigators observed that a prolonged QT with quinidine, sotalol, dofetilide, propafenon and terfanadine is associated with an increased propensity for arrhythmias, especially of the torsades de pointes variety. This difference appears to be the result of different effects of the agents on dispersion in repolarization; thus, measuring QT dispersion on a 24-hour basis may be a very helpful way of assessing drug effects in an individual patient (27).

More accurate measurement and standardization techniques are needed for QT dispersion assessment. Additionally, studies are needed to be directed at assessing QT dispersion effects of drug therapy on a 24-hour basis. Most important is looking at relative changes in each patient. The current methodological problems of QT measurement are no reason to discard these useful concepts (8).

P wave dispersion

P wave dispersion is a new electrocardiography (ECG) index. It is defined as the difference between the longest and the shortest P wave duration recorded from multiple different surface ECG leads. It has a diurnal variation in healthy subjects such as shortest in summer and longest in winter (28).

Up to know the most extensive clinical evaluation of P wave dispersion has been performed in the assessment of the risk for atrial fibrillation (AF) which is characterized by inhomogeneous and discontinuous atrial conduction. Several studies showed that P wave dispersion has a predictive value for AF in patients without apparent heart disease, in hypertensives, in patients with coronary artery disease and in patients undergoing coronary artery bypass surgery. P wave dispersion has proven to be a sensitive and specific ECG predictor of AF in the various clinical settings (29).

The methods used for the calculation of P wave dispersion are manually on paper print, digital boards and on-screen methods.

Clinical situations associated with P wave dispersion

1. Ionic imbalance and dialysis itself may cause changes in P dispersion in nondiabetic patients with end stage renal failure on chronic hemodialysis (30).
2. In chronic obstructive pulmonary disease patients, presence of AF was significantly related to the prolongation of P wave dispersion (31).
3. P wave dispersion is greatest on day 2 and 3 after open-heart surgery, finding that coincide with the time of greatest risk for AF (32).

4. Inhomogeneity of atrial conduction (increased P wave dispersion) is correlated with size of defect and with degree of right atrial dilatations in children with secundum atrial septal defect (33).
5. P wave dispersion has increased during balloon-induced acute ischemia of percutaneous transluminal coronary angioplasty (34).
6. The changes in left atrial micro architecture, which concurrently decreased atrial myocardial contraction and increased P wave dispersion cause predisposition to paroxysmal atrial fibrillation (35).
7. Measurement of P wave dispersion in sinus rhythm may be a useful non-invasive clinical tool to identify patients with hypertension at risk of developing atrial electrical instability and AF (36).
8. Corrected dispersion of atrial repolarization is a useful parameter to follow-up patients with cardiac transplantation at risk of rejection (37).
9. P wave dispersion is simple electrocardiographic marker that could be used for the prediction of idiopathic paroxysmal AF (38). A P wave dispersion value of 40 msec separated patients from control subjects, with a sensitivity of 83% and a specificity of 85% (39).

In conclusion, P wave dispersion is a simple and useful parameter for the prediction of atrial arrhythmias.

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