References


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QT dispersion and sudden cardiac death

See page 281 for the article to which this Editorial refers

Sudden cardiac death due to malignant ventricular arrhythmias is a considerable health problem in Europe and rest of the world. The article by Fu et al. in this issue focuses on dispersion of repolarization as a main electrophysiological factor for sudden death in patients with coronary artery heart disease and dilated cardiomyopathy.

Strong experimental evidence links ventricular fibrillation to an increased temporal dispersion of the recovery of excitability. Increased non-uniform recovery time may result from inhomogeneity of repolarization duration or from local delay in activation. The development of ventricular fibrillation seems to be dependant on several electrophysiological mechanisms, for instance the timing and the starting point of an extrasystole. When the distance to a preceding action potential is short, a marked shortening of the following action potential is observed. This phenomenon is described as the proximity effect and has been found in isolated heart preparations, in beating dog hearts and in patients with coronary artery disease. There is reason to believe that an early extrasystole induces ventricular fibrillation when it starts in an area with short action potentials and moves to an area with long action potentials or moves from an area with normal conduction to an area with delayed conduction.

Neural mechanisms have a significant role to play in the regulation of ventricular repolarization and that process may in itself increase or decrease dispersion of refractoriness. In patients with cardiomyopathy, sympathetic innervation has been found to be heterogeneous and seems to be related to inhomogenous refractoriness. Local delay in activation may be due to earlier myocardial infarction, scars etc.

At present it is impossible to measure exactly dispersion of repolarization in the ventricles non-invasively. In experimental studies in dogs the end of the T wave in the ECG corresponds largely to the end of the latest action potentials obtained on the epicardial surface of the heart.

In the 12-lead ECG, an interlead difference between the end of the T wave is known for some time. In the last few years it has been thought that this lead difference between the ends of the T waves, expressed as QT or JT dispersion, in some way reflects repolarization inhomogeneity, even though the methodology has not been adequately tested against invasively obtained monophasic action potentials.

The long QT time syndrome demonstrates dramatically increased QT dispersion, and this variable is probably related to sudden death and life threatening arrhythmias. Its decrease may be linked to the antiarrhythmic effect of beta-blockers. There is reason to believe that the arrhythmogenic effect...
of certain antiarrhythmic drugs are also related to increased dispersion of refractoriness.

The underlying mechanisms of arrhythmogenesis in ischaemic and non-ischaemic heart failure are still incompletely understood, and might involve a complex set of pathogenic factors. In the present paper[1] patients with dilated cardiomyopathy and chronic ischaemic heart disease are mixed together, which potentially might weaken this report. However, the incidence of sudden cardiac death has been found to be similar in these groups of patients, pointing to a common electrophysiological mechanism behind sudden cardiac death in cardiac failure. Arguments for more complexity are many. Left ventricular ejection fraction has, in numerous studies, been shown to be related to cardiac death, while repolarization variables, as measured in the present study, are not closely correlated to ejection fraction. It is well documented that plasma epinephrine and norepinephrine levels, platelet aggregability and coronary artery resistance are important in the occurrence of ventricular fibrillation. The role of dispersion of refractoriness in this respect is largely unknown.

QT dispersion was initially defined as the difference between the maximum and the minimum QT intervals measured from the standard 12 ECG leads. It has been suggested that JT interval dispersion is a better predictor of sudden cardiac death in patients with myocardial infarction. JT measurements are described carefully in the present paper[1]. Heart rate corrected QT and JT intervals were calculated by Bazett’s formula and written as QTc and JTc. These formulae show the relationship between heart rate and QT and JT time. The relationship is, however, influenced by several factors, such as slow adaptation to change in heart rate, exercise, cardiac drugs etc.

In the present paper[1] 163 patients with impaired ventricular function, defined as ejection fraction lower than 40%, were included. Forty-nine patients died of a primary cardiac event during a follow-up period averaging 26 months. Twenty four patients died suddenly. Thirty fulfilled endpoint criteria (24 sudden cardiac death and 10 sustained ventricular tachyarrhythmias). JTc-d was found to be the most powerful prognostic factor, with values above 85 ms yielding high sensitivity and specificity for endpoint events. These data suggest that the test may have dramatic consequences for a large population group.

Limitations of the present study, as a guideline for clinical decision making, are many. It is a retrospective study with all its pitfalls. Dispersion of JTc was measured manually with the possibility of bias. A prospective study is therefore urgently needed to confirm the present data before definite therapeutic consequences are taken in individual patients. It is, of course, of interest to explore the effect of beta-blockers, ACE inhibitors etc. Lots of research has to be done.

In a study by Fei et al. published last year[7], QT dispersion in patients with congestive heart failure secondary to idiopathic cardiomyopathy did not predict cardiac death. In that study, few patients died suddenly and 55% of the patients were excluded due to atrial fibrillation or bundle branch block. Other studies on QT dispersion are more in agreement with the present study[8].

The mechanism of sudden cardiac death in patients with severe cardiac failure is approximately 50% ventricular tachyarrhythmias and 50% bradyarrhythmias. In patients with less severe heart failure, ventricular tachyarrhythmias are relatively more common as the cause of death. While defibrillators are presumed to abort sudden death from tachyarrhythmias, their role in reducing the total death rate in patients with severe cardiac insufficiency has been questioned. It is therefore, in my opinion, not justified to implant defibrillators in patients with cardiac insufficiency and JTc-d above 85 ms before more research has been done in this very important field of electrophysiology. We also hope for better tools for measuring dispersion of refractoriness non-invasively in man. Meanwhile we have to rely on the classical ECG intervals measured manually with a paper speed of 50 mm · s⁻¹ with all its limitations.

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References