

Efficiency of quantification of cardiac electrical heterogeneity: via QT dispersion, transmural dispersion, or both

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CITATION: Tokatli A, Yiginer O, Haholu A, Uzun M. Efficiency of quantification of cardiac electrical heterogeneity: via QT dispersion, transmural dispersion, or both *Biol Sport*. 2015;32(2):175–176.

Received: 2014-10-17; Reviewed: 2014-12-12; Re-submitted: 2014-12-12; Accepted: 2014-12-12; Published: 2015-03-17.

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Key words:

athletes

heart

repolarization

Tp-e interval

Tp-e/QT ratio

LETTER TO THE EDITOR

We read with great interest the article entitled 'A 24-hour ambulatory ECG monitoring in assessment of QT interval duration and dispersion in rowers with physiological myocardial hypertrophy' by Lutfullin IY et al. [1] published in the *Biology of Sport* journal. In the study, the effects of myocardial hypertrophy on cardiac repolarization parameters were investigated in endurance athletes using both 12-lead ECG and 24-hour ECG monitoring. In the conclusion of the article, it was stated that myocardial hypertrophy observed in athletes does not lead to an increase in corrected QT interval (QTc) duration, QT dispersion (QTd) or corrected QT dispersion (QTcd). QT interval (QTI) duration was found to be increased in athletes with myocardial hypertrophy, but it was attributed to the increased vagal tone of the endurance athletes.

Regular intensive training may lead to cardiac hypertrophy known as athlete's heart. Athlete's heart is associated with alterations in cardiac structure and electrophysiological properties [2]. As expected, heterogeneity in the duration of the ventricular repolarization phase leading to arrhythmias may also be seen in athlete's heart. As used in this study, QTd is the most frequently used parameter to detect ventricular inhomogeneity. However, as also mentioned in this article, varying results have been reported in the studies related to QTI [3]. This may be due to the technical limitations in the QTI measurement. QTI can be measured either manually or automatically. It is well known that the reproducibility of QTI measurements is low in both manual and automatic measurements [4]. Additionally, in manual measurements, inter- and intra-observer variability of QTd is very high [3].

Quantifying the inhomogeneity of the myocardium, transmural dispersion of repolarization (TDR) has also been used since the beginning of the 2000s in addition to QTd [5]. There are three cell types having different electrophysiological properties in the ventricu-

lar myocardium: the endocardial, epicardial and midmyocardial M cells. Repolarization of these three different cell types creates the T wave of the ECG. The epicardial cells are repolarised early, which coincides with the peak of the T wave, but repolarization of M cells is the latest and coincides with the end of the T wave. Therefore, the time between the peak and end of the T wave is called the Tp-e interval, as an index of TDR. The ratio of the TDR to the total duration of repolarization (Tp-e/QT ratio) has also been used as an ECG index of ventricular arrhythmogenesis [6]. The role of the TDR in the determination of the arrhythmic risk has been demonstrated in coronary artery disease and in the Brugada, short QT and long QT syndromes [7,8]. Previously, we found that TDR was increased in patients with obstructive sleep apnoea, chronic arsenic exposure and in long-term heavy cigarette smokers [9,10]. Increased TDR has also been demonstrated in subjects with hypertension, obesity and in animal models of ventricular hypertrophy [11-13]. Additionally, it was found in different species with varying body sizes that both QTI duration and TDR increase as the body mass increases. As it may be concluded from this observation, increased left ventricular mass causes increased TDR leading to prolongation of QTI. This situation suggests that the thickening layer of the heart is mostly the midmyocardial layer, which leads to prolongation of the Tp-e interval. In athletes, left ventricular hypertrophy may also occur mostly in the midmyocardial layer in order to meet the increased demands of the body during endurance physical activity.

Regarding these observations, if Lutfullin et al. measured the Tp-e interval in their study, they might find increased TDR in rowers with myocardial hypertrophy. Thus, considering all the data about QTI duration, QTd and TDR, the study might have completely revealed the effects of athlete's heart on electrical heterogeneity of the myocardium in many respects.

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