Original Paper DOI: 10.5604/20831862.1077547 Biol. Sport 2013;30:237-241

A 24-HOUR AMBULATORY ECG MONITORING IN ASSESSMENT OF QT INTERVAL DURATION AND DISPERSION IN ROWERS WITH PHYSIOLOGICAL MYOCARDIAL HYPERTROPHY

Accepted for publication 26.08.2013

AUTHORS: Lutfullin I.Y.[1,2], Kim Z.F.[3], Bilalova R.R.[4], Tsibulkin N.A.[2], Almetova R.R.[1], Mudarisova R.R.[1], Ahmetov I.I.[1,3]

[1] Volga Region State Academy of Physical Culture, Sport and Tourism, Kazan, Russia
[2] Kazan State Medical Academy, Kazan, Russia
[3] Kazan State Medical University, Kazan, Russia
[4] Emergency Care Hospital of Kazan #1, Kazan, Russia

Reprint request to:
Ildus Y. Lutfullin
Volga Region State Academy of Physical Culture, Sport and Tourism
E-mail: lutfullin@list.ru

ABSTRACT: Myocardial hypertrophy (MH) due to cardiac pathology is characterized by an increase in QT interval duration and dispersion, while the findings for exercise-induced myocardial hypertrophy are contradictory. The majority of published research findings have not explored this relationship, but there have only been a few conducted studies using 24-hour ECG monitoring. The aim of the study was to determine the QT interval duration and dispersion in short-term and 24-hour ECG in endurance athletes with myocardial hypertrophy and without it. Methods: A total of 26 well-trained rowers underwent a resting 12-lead ECG, 24-hour ECG monitoring and echocardiography. Results: Athletes with MH (n = 7) at rest did not show any increase in QTc interval duration and dispersion, or mean and maximal QTc duration in Holter monitoring compared to athletes without MH (n = 19). Left ventricular mass was not significantly correlated with any QTc characteristics. Furthermore, athletes with MH had significantly longer mean QT (P = 0.01) and maximal QT (P = 0.018) intervals in Holter monitoring and higher 24-hour heart rate variability indexes due to stronger vagal effects. Conclusions: The present study demonstrated that athlete's heart syndrome with myocardial hypertrophy as a benign phenomenon does not lead to an increase in QT interval duration, or increases in maximal and mean duration in a 24-hour ECG. An increase in QT interval duration in athletes may have an autonomic nature.

KEY WORDS: athletes, myocardial hypertrophy, left ventricular mass, QT interval, Holter monitoring

INTRODUCTION

Regular exercise may lead to functional and structural adaptations that improve cardiac function. The increase in ventricular wall thickness before reaching the level of hypertrophic cardiomyopathy or a "gray zone" is considered to be a benign physiological phenomenon, also known as "athlete's heart syndrome". The prevalence of myocardial hypertrophy (MH) in athletes differs depending on age, gender, ethnicity, type of sport and the extent and intensity of training [11,22]. The relationship between MH and the electrophysiology of the myocardium differs between physiological and pathological variants. For instance, MH in hypertrophic cardiomyopathy or essential arterial hypertension is characterized by a long QT interval and increases in QT dispersion [14], while the findings for exercise-induced MH are contradictory. The majority of published research findings have not explored this relationship [8,17,18,27], and few studies have supported this observation [16,24].

QT interval prolongation is vitally important, as it is an independent risk factor of ventricular tachycardia and sudden cardiac death regardless of the reason of prolongation, including inborn long QT syndrome or acquired forms due to cardiac pathology and drug side effects [28]. Some authors suggest that the autonomic nervous system may also cause QT interval prolongation and increase its dispersion [2].

Holter monitoring (a 24-hour ECG) is used for expanded evaluation of QT interval duration [12]. Despite the well-known difficulties in QT interpretation in Holter monitoring (due to signal filtering and recording methods), evaluation of possible long QT is a class I indication for ambulatory ECG [5].

The aim of the present study was to determine the QT interval duration and dispersion in short-term and 24-hour ECG in endurance athletes with MH and without it.

MATERIALS AND METHODS

Twenty-six well-trained male and female rowers aged from 17 to 22 years (18.9±2.0) took part in the study. The athletes were divided into two groups according to their left ventricular mass index: athletes with left ventricular hypertrophy (LVH) (LVH Group, n = 7) and

athletes with normal ventricular myocardial mass (NVMM Group, n = 19). The survey was performed prior to training. All athletes were training at the same rowing centre. The Ethics Committee of the Kazan State Medical Academy approved the study and written informed consent was obtained from each participant. All experiments were performed in accordance with the ethical standards of the Helsinki Declaration.

Standard 12-lead ECG was recorded with a paper speed of 50 mm·s^{-1} and amplification of 0.1 mV·mm^{-1}. The duration and dispersion of QT and QTc were measured accurately using sport-specific recommendations for interpretation [13]. Mean, maximum and minimum duration of QT and QTc intervals in a 12-lead 24-hour ECG were measured automatically after manual exclusion of non-sinus events, noise, intermittent data and similar conditions typical of Holter recordings [5].

Echocardiography was performed in an integrated M-mode and two-dimensional study to determine septal wall thickness, posterior wall thickness, left ventricular (LV) end-diastolic diameter and LV end-systolic diameter, consistent with the recommendations of the American Society of Echocardiography [6]. Overlying trabeculations were excluded in the ventricular septum or posterior wall measurements, to avoid overestimating wall thickness. The LV mass was calculated using the formula of Devereux [7]. Measurements were quoted with body surface area calculated using the formula of Dubois and Dubois. According to the American Society of Echocardiography/ European Association of Echocardiography recommendations we considered myocardial hypertrophy at 95 g·m^{-2} in females and 115 g·m^{-2} in males [15]. Left ventricular ejection fraction was determined by the Teichholz method [26].

The 24-hour heart rate variability (HRV) was studied using the following indexes: SDNN (standard deviation of all NN intervals) – total power of HRV, pNN50% (percentage of differences between adjacent NN intervals that are greater than 50 ms) and rMSSD (root mean square of the successive differences) – short-term components of HRV, SDNNidx (standard deviation of the average of NN intervals in all 5-minute segments of a 24-h recording) – long-term components of HRV [25].

All analyses were conducted using SPSS for Windows version 20 software. Results are presented as mean ± SD. Categorical variables were compared by using the Mann-Whitney U-test, for continuous variables, while the difference between the two groups was assessed using an unpaired t-test. Correlations were calculated by Pearson and Spearman's rank correlation coefficient. A P value < 0.05 was considered statistically significant.

RESULTS

Echocardiographic surveys showed significantly lower ejection fraction and shortening fraction (within normal limits) in LVH Group (P = 0.031 and P = 0.001, respectively) in comparison with NVMM Group. Other values were not significantly different between the two groups (Table 1).

TABLE 1. MAIN ECHOCARDIOGRAPHIC VALUES IN TWO GROUPS OF ATHLETES

Echocardiographic parameters	LVH Group (n=7)	NVMM Group (n=19)	P
Left ventricular mass index, g·m^{-2}	94.9 ± 11.4	110.2 ± 6.83	p<0.05
Aortic root diameter, mm	2.88 ± 0.12	2.89 ± 0.14	ns
Aortic valve opening, mm	1.92 ± 0.13	1.95 ± 0.16	ns
Left atrium diameter, mm	3.08 ± 0.17	3.10 ± 0.18	ns
Right atrium size, mm	2.25 ± 0.30	2.23 ± 0.23	ns
Ejection fraction, %	65.3 ± 1.30	66.95 ± 1.50	p<0.05
Shortening fraction, %	35.50 ± 0.63	37.11 ± 1.20	p<0.01

Note: LVH - left ventricular hypertrophy; NVMM - normal ventricular myocardial mass, ns - not statistically significant

TABLE 2. ELECTROCARDIOGRAPHIC CHARACTERISTICS IN TWO GROUPS OF ATHLETES

	LVH Group (n=7)	NVMM Group (n=19)	P
PQ, ms	143.2 ± 15.3	173.0 ± 33.9	ns
QRS, ms	98.5 ± 11.5	94.5 ± 4.4	ns
QT interval, ms	393.2 ± 30.6	419.5 ± 25.0	ns
QTc interval	412.3 ± 32.0	406.0 ± 36.8	ns
QRS axis, 0	65.2 ± 2.1	71.6 ± 32.0	ns
ΔQT, ms	38.4 ± 13.3	31.7 ± 6.2	ns
ΔQTc	41.2 ± 14.5	30.8 ± 8.5	ns

Note: LVH - left ventricular hypertrophy; NVMM - normal ventricular myocardial mass; ns - not statistically significant

The duration and dispersion of QT and QTc intervals at rest were not significantly greater in LVH Group (P = 0.13 and P = 0.77, respectively). These values were within normal limits among all athletes (Table 2). In correlation analysis of the data, QT and QTc did not significantly correlate with LV mass and LV mass index. Furthermore, we also observed a positive relationship between ΔQT and ΔQTc at rest and aortic root diameter (r = 0.54, r = 0.52, respectively, P < 0.05).

Moderate positive relationships were revealed between QT characteristics observed at rest and during Holter monitoring. The QT interval duration of ECG correlated with mean QT duration during waking periods (r = 0.75, P < 0.01) and over 24-hour periods (r = 0.66, P < 0.05); the correlations for QTc interval were r = 0.56 and r = 0.48, respectively (P < 0.05). However, QT and QTc at rest did not significantly correlate with sleep period QT and QTc characteristics.

TABLE 3. MEAN, MAXIMAL AND MINIMAL QT AND QTc DURATIONS OF ECG IN TWO GROUPS OF ATHLETES

ECG parameter	Period	LVH Group (n=7)	NVMM Group (n=19)	P
mean QT	24-hour	394.5 ± 17.2	422.0 ± 12.6	p<0.05
	Awake period	362.8 ± 14.8	389.5 ± 12.0	p<0.01
	Sleep period	418.8 ± 22.5	451.5 ± 13.7	p<0.01
mean QTc	24-hour	405.8 ± 13.4	412.0 ± 20.3	ns
	Awake period	406.0 ± 12.8	413.0 ± 16.3	ns
	Sleep period	405.8 ± 14.1	410.0 ± 23.1	ns
maximal QT	24-hour	462.3 ± 17.7	488.3 ± 7.2	p<0.05
	Awake period	419.6 ± 17.7	452.0 ± 25.0	*p=0.07*
	Sleep period	462.3 ± 22.7	488.3 ± 7.2	p<0.05
maximal QTc	24-hour	457.1 ± 20.8	469.5 ± 11.3	ns
	Awake period	446.5 ± 16.4	464.5 ± 13.2	ns
	Sleep period	455.9 ± 20.4	466.3 ± 16.7	ns
minimal QT	24-hour	308.8 ± 39.3	304.8 ± 12.6	ns
	Awake period	308.8 ± 27.1	304.8 ± 12.6	ns
	Sleep period	360.5 ± 39.3	392.5 ± 31.0	ns
minimal QTc	24-hour	361.2 ± 25.1	367.8 ± 20.4	ns
	Awake period	368.6 ± 18.8	372.3 ± 21.9	ns
	Sleep period	364.6 ± 25.5	376.3 ± 22.2	ns

Note: LVH - left ventricular hypertrophy; NVMM - normal ventricular myocardial mass; ns - not statistically significant

TABLE 4. CORRELATIONS BETWEEN MEAN AND MAXIMAL QTc AND QT

ECG parameter	Period	LV mass	LV mass index
mean QT	24-hour	0.78**	0.71**
	Awake period	0.70**	0.67**
	Sleep period	0.77**	0.72**
maximal QT	24-hour	0.76**	0.68**
	Awake period	0.72**	0.69**
	Sleep period	0.76**	0.68**
mean QTc	24-hour	0.43	0.25
	Awake period	0.21	0.37
	Sleep period	0.38	0.21
maximal QTc	24-hour	0.76**	0.63*
	Awake period	0.66**	0.47
	Sleep period	0.77**	0.66**

Note: * P < 0.05; ** P < 0.01;

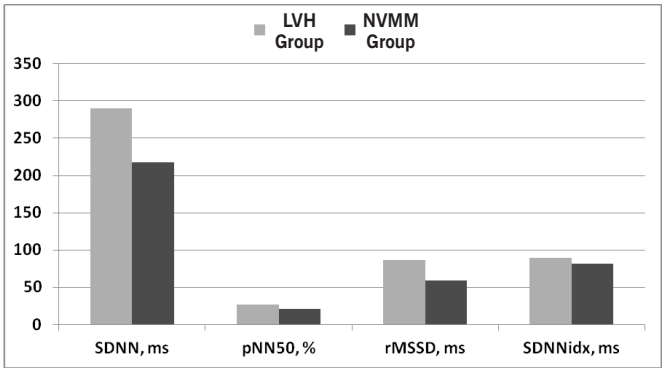

FIG. 1. 24-HOUR HRV INDEXES IN TWO GROUPS OF ATHLETES. Note: LVH - left ventricular hypertrophy; NVMM - normal ventricular myocardial mass; *P < 0.05, statistically significant differences between two groups.

In evaluating the mean, minimal and maximal duration of QTc interval during sleep, while awake and over 24-hour periods, no significant differences between LVH Group and NVMM Group were revealed (Table 3). In contrast, mean duration of QT interval of the same ECG monitoring periods was significantly longer in LVH Group (Table 3). Moreover, LVH Group athletes had significantly longer maximal QT intervals (P = 0.02), while minimal QT was not significantly different. Mean and maximal QT and maximal QTc durations positively correlated with LV mass and LV mass index (Table 4).

The 24-hour HRV survey revealed significantly higher HRV indexes in LVH Group, namely, higher SDNN (290.5 ± 48.4 ms vs. 217.5 ± 33.5 ms; P = 0.048), RMSSD (86.7 ± 27.9 ms vs. 59.4 ± 18.0 ms; P = 0.044) and pNN50 (26.7 ± 4.8% vs. 20.8 ± 10.9%; P = 0.035) (Figure 1), reflecting higher levels of total power and short-term components of power of HRV.

The relationships between QT and QTc interval duration and autonomic influences on heart electrophysiology are of particular interest. We did not find any significant correlations between 24-hour HRV indexes (SDNN, rMSSD, pNN50, SDNNidx) and QT and QTc intervals duration at rest, or between 24-hour maximal and mean QTc and mean QT. Maximal 24-hour QT interval positively correlated

TABLE 5. CORRELATIONS BETWEEN ΔQT AND ΔQTC PARAMETERS OF ECG AT REST AND 24-HOUR HRV INDEXES

Period	HRV index	ΔQT	ΔQT
Day	SDNN, ms	0.45	0.47
	pNN50, %	0.64**	0.52*
	SDNNidx, ms	0.68**	0.61*
Night sleep	SDNN, ms	0.33	0.39
	pNN50, %	0.02	-0.12
	SDNNidx, ms	0.49	0.49

Note: * $P < 0.05$; ** $P < 0.01$;

with SDNN ($r = 0.64$, $P < 0.05$). Furthermore, ΔQT ΔQTc at rest positively correlated with pNN50 and SDNNidx during wake periods, while sleep period HRV indexes did not show any significant relationships (Table 5).

DISCUSSION

QT and QTc interval duration represents electrical depolarization and repolarization of the left and right ventricles myocardium. Besides QT and QTc interval, the electrical properties of myocardium can be characterized by QT and QTc interval dispersion, which is known to be a marker of myocardial inhomogeneity [29]. Myocardial hypertrophy is a well-known cause of increase in QT interval duration and dispersion [14]. Prolonged QTc is considered to be unrelated to sports and an uncommon ECG phenomenon in athletes [3,22]. However, the role of exercise-induced MH in QT prolongation is still unclear. Tanriverdi et al. observed an increased ΔQTc and positive correlation between ΔQTc and LV mass in 56 endurance athletes compared to controls ($r = 0.3$, $P = 0.024$) [24]. The opposite results were obtained by Halle et al., when 26 endurance athletes with MH had lower QTc dispersion compared to controls and a negative correlation with LV mass was revealed ($r = -0.38$; $P = 0.002$) [10]. Zoghi et al. obtained similar results when 147 males with MH of varying etiologies (including long-term training) were examined and athletes had no significant QTc prolongation compared to controls [30]. Another study showed that QTc was significantly longer in 76 athletes with increased LV mass compared to 76 controls calculated with Fridericia and Hodges correction formulas [16]. However, most authors have demonstrated an increased QTc interval duration as an uncommon observation in athletes with MH [17,18,27], including elderly and young athletes. Accordingly, Galetta et al. [8] did not find an increase in QT duration or dispersion in 16 elderly athletes (aged 67.6±4.5 years) with LV myocardial hypertrophy. Alchaghouri et al. [1] did not reveal any relationship between MH and QTc interval duration in young athletes aged 16.4±0.76 years. Moreover, Rajappan et al. showed that the heart rate/QT recovery disturbance, typical for long QT syndromes, was also atypical in athletes. Fifteen highly trained rowers showed quick QT interval recovery after physical exercise, which was interpreted as a high myocardial fitness marker [21].

In our study we did not find any relationship between resting ECG and Holter monitoring QTc interval duration or dispersion and LV wall thickness, end-diastolic dimension or LV mass in general. The only echocardiographic value correlating with ΔQTc and ΔQT at rest was aortic root diameter. As shown before in sedentary and newly diagnosed arterial adult hypertensives, QTd is independently related to aortic strain ($P = 0.043$) and aortic distensibility ($P = 0.037$) calculated echocardiographically from the derived ascending aorta diameters [9]. In addition, it was shown that the aorta has an afterload mediated influence on ΔQT, independent from MH [19]. This phenomenon requires further evaluation, especially in athletes with increased aortic root dimensions.

24-hour monitoring may provide additional information on QT interval characteristics. In our study moderate correlations between resting ECG and Holter monitoring values of QT and QTc intervals were obtained, excluding the sleep period. The latter may indicate a change in QT duration regulation during sleep. We found only a few studies examining QT interval with Holter monitoring in athletes. More specifically, Palatini et al. showed a prolonged QTc interval in 30 highly trained boys aged 10-14 compared to 30 age-matched untrained controls [20]. However, we did not reveal any influence of athlete's heart syndrome (including benign MH) on mean and maximal QTc duration in a 24-hour ECG in this study. This viewpoint is consistent with the benign nature of athlete's heart syndrome [4]. One might suggest that higher duration of mean and maximal QT in athletes with MH can be explained by the manifestation of autonomic influences, taking into account higher levels of 24-hour HRV indexes in these athletes. Moreover, SDNN was positively correlated with maximal QT, but not with mean and maximal QTc, which are clinically significant.

Our study does have limitations. Firstly, both manual and automatic measurement of QT and QTd may be subject to error. Secondly, the study had a relatively small sample size. For this reason, these findings cannot be generalized to the broader community based on this study alone.

CONCLUSIONS

The present study demonstrated that athlete's heart syndrome with myocardial hypertrophy does not lead to increase in clinically significant QT interval duration, or increases in maximal and mean duration in a 24-hour ECG. The increase in QT interval duration in athletes may have an autonomic nature.

ACKNOWLEDGMENTS

This work was supported by a grant of the Volga Region State Academy of Physical Culture, Sport and Tourism (2012-2013).

Conflict of interest

The authors report no conflicts of interest.

REFERENCES

1. Alchaghouri S., Wong K.Y., Perry R.A., Ramsdale D.R., Somauroo J.D., Pyatt J.R. QT peak prolongation is not associated with left ventricular hypertrophy in teenage professional football players. Ann. Noninvasive Electrocardiol. 2007;12(2):104-110.
2. Baranowski R., Buchner T., Poplawska W., Rydlewska-Sadowska W. Sex differences in 24 H QT analysis in normal subjects. Abstr. of the 9 th Congress of the International Society for Holter and Noninvasive Electrocardiology & International Congress on Cardiac Pacing and Electrophysiology. Istanbul, Turkey 23-27 September 2000. Annals of Noninvasive Electrocardiology 2000; 5(4) (part 2):186, s.47.
3. Basavarajaiah S., Wilson M., Whyte G., Shah A., Behr E., Sharma S. Prevalence and significance of an isolated long QT interval in elite athletes. Eur. Heart J. 2007;28(23):2944-2949.
4. Biffi A., Maron B.J., Culasso F., Verdile L., Fernando F., Di Giacinto B., Di Paolo F.M., Spataro A., Delise P., Pelliccia A. Patterns of ventricular tachyarrhythmias associated with training, deconditioning and retraining in elite athletes without cardiovascular abnormalities. Am. J. Cardiol. 2011;107(5):697-703.
5. Crawford M.H., Bernstein S.J., Deedwania P.C., DiMarco J.P., Ferrick K.J., Garson A. Jr., Green L.A., Greene H.L., Silka M.J., Stone P.H., Tracy C.M., Gibbons R.J., Alpert J.S., Eagle K.A., Gardner T.J., Gregoratos G., Russell R.O., Ryan T.H., Smith S.C. Jr. ACC/AHA Guidelines for Ambulatory Electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the Guidelines for Ambulatory Electrocardiography). Developed in collaboration with the North American Society for Pacing and Electrophysiology. J. Am. Coll Cardiol. 1999;34(3):912-948.
6. Devereux R.B., Liebson PR, Horan MJ. Recommendations concerning the use of echocardiography in hypertension and general population research. Hypertension.1987;9(Pt 2):97-104.
7. Devereux R.B., Alonso D.R., Lutas E.M., Gottlieb G.J., Campo E., Sachs I., Reichek N. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am. J. Cardiol.1986; 57(6):450-458.
8. Galetta F., Franzoni F., Santoro G., Prattichizzo F., Femia F.R., Pastine F., Pentimone F. QT dispersion in elderly athletes with left ventricular hypertrophy. Int. J. Sports Med. 2003; 24(4):233-237.
9. Gur M., Yilmaz R., Demirbag R., Yildiz A., Akyol S., Polat M., Bas M.M. Relationship between the elastic properties of aorta and QT dispersion in newly diagnosed arterial adult hypertensives. Anadolu Kardiyol. Derg. 2007;7(3):275-280.
10. Halle M., Huonker M., Hohnloser S.H., Alivertis M., Berg A., Keul J. QT dispersion in exercise-induced myocardial hypertrophy. Am. Heart J. 1999;138(2 Pt 1):309-312.
11. Hosseini M., Piri M., Agha-Alinejad H., Haj-Sadeghi Sh. The effect of endurance, resistance and concurrent training on the heart structure of female students. Biol. Sport 2012;29(1):17-21.
12. ICH Harmonized Tripartic Guideline E14 (2005). The clinical evaluation of QT/QTc interval prolongation and proarrhythmic potential for non-antiarrhythmic drugs. Available at: http://www.fda.gov/RegulatoryInformation/Guidances/ucm129335.html
13. Johnson J.N., Ackerman M.J. QTc: how long is too long? Br. J. Sports Med. 2009;43(9):657-662.
14. Kahan T., Bergfeldt L. Left ventricular hypertrophy in hypertension: its arrhythmogenic potential. Heart. 2005;91(2):250-256.
15. Lang R.M., Bierig M., Devereux R.B., Flachskampf F.A., Foster E., Pellikka P.A., Picard M.H., Roman M.J., Seward J., Shanewise J.S., Solomon S.D., Spencer K.T., Sutton M.S., Stewart W.J.; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J. Am. Soc. Echocardiogr. 2005;18(12):1440-1463.
16. Lengyel C., Orosz A., Hegyi P., Komka Z., Udvardy A., Bosnyák E., Trájer E., Pavlik G., Tóth M., Wittmann T., Papp J.G., Varró A., Baczkó I. Increased short-term variability of the QT interval in professional soccer players: possible implications for arrhythmia prediction. PLoS One. 2011;6(4):e18751.
17. Lonati L.M., Magnaghi G., Bizzi C., Leonetti G. Patterns of QT dispersion in athletic and hypertensive left ventricular hypertrophy. Ann. Noninvasive Electrocardiol. 2004;9(3):252-256.
18. Mayet J., Kanagaratnam P., Shahi M., Senior R., Doherty M., Poulter N.R., Sever P.S., Handler C.E., Thom S.A., Foale R.A. QT dispersion in athletic left ventricular hypertrophy. Am. Heart J. 1999;137(4 Pt 1):678-681.
19. Orlowska-Baranowska E., Baranowski R., Kusmierczyk B., Sepiska J. Reduction of the QT interval dispersion after aortic valve replacement reflects changes in electrical function rather than structural remodeling. J. Heart Valve Dis. 2005;14(2):181-185.
20. Palatini P., Maraglino G., Mos L., Munari L., Ronsisvalle G., Calzavara A., Lusiani L., Libardoni M., Pessina A.C., Dal Palù C. Effect of endurance training on Q-T interval and cardiac electrical stability in boys aged 10 to 14. Ventricular arrhythmias in trained boys. Cardiology. 1987;74(5):400-407.
21. Rajappan K., O'Connell C., Sheridan D.J. Changes in QT interval with exercise in elite male rowers and controls. Int. J. Cardiol. 2003;87(2-3):217-222.
22. Sharma S. Athlete's heart – effect of age, sex, ethnicity and sporting discipline. Exp. Physiol. 2003;88(5):665-669.
23. Steriotis A.K., Nava A., Rigato I., Mazzotti E., Daliento L., Thiene G., Basso C., Corrado D., Bauce B. Noninvasive cardiac screening in young athletes with ventricular arrhythmias. Am. J. Cardiol. 2013;111(4):557-562.
24. Tanriverdi H., Kaftan H.A., Evrengul H., Dursunoglu D., Turgut G., Kiliç M. QT dispersion and left ventricular hypertrophy in athletes: relationship with angiotensin-converting enzyme I/D polymorphism. Acta Cardiol. 2005;60(4):387-393.
25. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation 1996;93 (5):1043-65.
26. Teichholz L.E., Kreulen T., Herman M.V., Gorlin R. Problems in echocardiographic volume determinations: echocardiographic-angiographic correlations in the presence or absence of asynergy. Am. J. Cardiol. 1976;37:7-11.
27. Turkmen M., Barutcu I., Esen A.M., Ocak Y., Melek M., Kaya D., Karakaya O., Saglam M., Basaran Y. Assessment of QT interval duration and dispersion in athlete's heart. J. Int. Med. Res. 2004;32(6):626-632.
28. Viskin S. Long QT syndromes and torsade de pointes. Lancet 1999;354:1625-1633.
29. Whitsel E.A., Raghunathan T.E., Pearce R.M., Lin D., Rautaharju P.M., Lemaitre R., Siscovick D.S. RR interval variation, the QT interval index and risk of primary cardiac arrest among patients without clinically recognized heart disease. Eur. Heart J. 2001;22:165–173.
30. Zoghi M., Gürgün C., Yavuzgil O., Akilli A., Türkoglu C., Kültürsay H., Akin M. QT dispersion in patients with different etiologies of left ventricular hypertrophy: the significance of QT dispersion in endurance athletes. Int. J. Cardiol. 2002;84(2-3):153-159.