

Decrease in QRS amplitude in juvenile female competitive athletes during the initial twenty-one months of intensive training

Ljuba Bacharova¹, Martina Tibenska², Dana Kucerova², Olga Kyselovicova³,
Helena Medekova³ and Jan Kyselovic²

¹International Laser Centre, Bratislava, Slovak Republic

²Faculty of Pharmacy, Comenius University, Bratislava, Slovak Republic

³Faculty of Physical Education and Sport, Comenius University, Bratislava, Slovak Republic

Abstract

Background: *The aim of this project was to study changes in QRS amplitude in junior female athletes during the initial twenty-one months of competitive training programme in aerobic gymnastics.*

Methods: *Somatometric parameters, heart rate (HR), blood pressure (BP) and 12-lead ECGs were recorded in 12 female athletes, aged 13–17 years (average 13.8) at 3-month intervals over a period of 21 months. The Sokolow-Lyon index (SLI) and the maximum QRS spatial vector magnitude (QRSmax), approximated from RV5, RaVF and SV2 voltages, were analyzed.*

Results: *The mean values of QRSmax and SLI decreased gradually during the study period. The difference between the mean QRSmax values at the beginning and at the end of the study period was -0.8 mV (30.8%), $p < 0.001$, and between the initial and final values of SLI was -0.6 mV (24%), $p < 0.001$. The somatometric parameters changed only slightly, HR and systolic BP values did not change significantly.*

Conclusions: *This study showed that 21 months of competitive aerobic gymnastics training led to a decrease in the QRSmax magnitude. This finding is in contrast with the classical hypothesis on the ECG diagnostics of LVH and is in agreement with an alternative hypothesis on the relative voltage deficit during the early stage of LVH development. (Cardiol J 2007; 14: 260–265)*

Key words: electrocardiogram, QRS voltage, juvenile athletes, female

Editorial p. 220

Introduction

Regular physical training results in an increase in left ventricular mass (LVM), which is known as physiological left ventricular hypertrophy (LVH), or athlete's heart [1, 2]. 12-lead ECG in athletes frequently shows an increased QRS voltage and these QRS changes are attributed to the physiological adaptation of the heart that occurs as a consequence of systematic physical training. However, the increased QRS amplitude is observed only in a proportion of athletes with increased LVM [3–6]. There is also poor agreement between the QRS voltage and the size and morphology of the left ventricle [6–9].

Address for correspondence:

Assoc. Prof. Ljuba Bacharova, MD, PhD, MBA
International Laser Centre
Ilkovicova 3, 812 19 Bratislava, Slovak Republic
Tel: +421 2 654 21 575; fax: +421 2 654 23 244
e-mail: bacharova@ilc.sk

This study was supported, in part, by the grant VEGA 1/3406/06 from The Science Grant Agency (VEGA), Slovak Republic.

Received: 26.02.2007

Accepted: 03.04.2007

In our previous papers, we have pointed out that the left ventricular mass does not always need to be the major determinant of the QRS voltage [10, 11]. We have shown a decrease in both absolute and relative values of QRS amplitude in the initial stage of experimental models of LVH due to volume and pressure overload, respectively [12, 13]. A similar decrease in QRS amplitude is also observed in the experimental model of exercise-induced left ventricular hypertrophy in swimming normotensive rats [14]. We have concluded that the decrease in the QRS amplitude could be an early sign of the hypertrophic rebuilding of myocardium, reflecting the changes in the electrical properties of myocardium.

In this study, we tested the hypothesis that the early period of intensive physical training is associated with a decrease in QRS amplitude. The changes in QRS amplitude in junior female athletes were analyzed during the course of 21 months training after their entry into a competitive aerobic gymnastics program.

Methods

A group of 12 girls aged 13–17 years (average age 13.8 years) was followed up for 21 months. This group of girls entered a newly designed intensive training program in competitive aerobic gymnastics at the Faculty of Physical Education and Sport of Comenius University, Bratislava, three months prior to the study period. Before entering the aerobic gymnastic program, most of the girls practiced early stage rhythmic or artistic gymnastics, or modern dance with a low level training load in the sport category of pupils (maximum 3 hours per week).

The girls were routinely screened in 3-month intervals. None of the girls had any history or symptoms of underlying cardiovascular disease, or of a family history of premature death from cardiovascular disease. None was taking any form of prescribed cardiovascular drug treatment.

During the study period, the number of training sessions was five to ten per week (2 hours per training session, 4 to 5 days per week). The overall design of the training programme contained the following components: aerobic activities (about 30 percent on average), an aerobic activities (about 20 percent on average), dynamic strength (10 percent), dynamic and static strength (10 percent), flexibility and coordination (about 30 percent on average).

The following anthropometric parameters were measured and calculated:

- body weight, height, body mass index (BMI);
- body fat percent (BF%) calculated as $BF\% = 0.365 \times (TS + SSS + SIS + MCS) + 0.62$,

where TS is the triceps skinfold width, SSS is the subscapular skinfold width, SIS is suprailiacal skinfold width and MCS is medial calf skinfold width;

- the absolute active body mass (ABM) calculated as $ABM = \text{bodyweight} - (BF\% \times BW/100)$;
- the relative active body mass (%ABM), calculated as $\%ABM = 100 - BF\%$.

Blood pressure was recorded in sitting position after 5 minutes of rest, using the automatic barometer OMRON M4-I, Omron Matsusaka, Japan.

Then standard 12-lead ECG was recorded in supine position using the electrocardiograph SEIVA EKG, Czech Republic. Each ECG was recorded for 15 s, the average value of the first three QRS complexes were used for further calculation. This electrocardiograph works in a semi-automated mode, e.g. the onset and the end of the QRS complexes can be corrected manually, and the QRS amplitude and the heart rate (HR) are given automatically. All electrocardiograms were evaluated by one blinded researcher.

The following QRS voltage parameters were calculated and analyzed:

- the Sokolow-Lyon index, calculated as the sum of SV2 plus RV5,6 [15];
- the approximated maximum spatial QRS vector magnitude (QRSmax), calculated using the following formula:

$$QRS_{\max} = \sqrt{RV5^2 + RaVF^2 + SV2^2}$$

Data are presented as mean and standard deviation (SD), or standard error of the mean (SEM), respectively. The differences between the values at particular time intervals were tested using the Friedman test. A probability value $p < 0.05$ was accepted as significant.

This study was approved by the Ethics Committee of the Faculty of Physical Education and Sports of the Comenius University, Bratislava. The girls' parents gave their informed consent for the inclusion of the girls in the study.

Results

Table 1 presents the basic statistics of the anthropometric variables in the study group. At the end of the study period the height of the girls increased by an average of 2 cm, this increase was statistically significant as compared to the initial values. In addition, the values of body weight, BMI and ABM increased significantly during the second half of the follow-up period compared to the initial values.

The values of HR, as well as of systolic BP, did not change significantly during the study period with respect to the initial values (Table 2).

Table 1. Anthropometric variables: height, body weight (BW), body-mass index (BMI), the body-fat percentage (BF%), the absolute active body-mass (ABM) and the relative active body-mass (%ABM), in the group of female athletes during the study period (mean ± SD are presented).

	Height [cm]	BW [kg]	BMI [kg/m ²]	BF%	ABM [kg]	%ABM
Sep 03	159.7 ± 4.3	46.2 ± 5.5	18.0 ± 1.5	12.4 ± 2.4	40.7 ± 4.1	87.6 ± 2.4
Dec 03	159.7 ± 4.3	46.2 ± 5.5	18.0 ± 1.5	11.3 ± 2.1	41.2 ± 4.2	88.7 ± 2.1
Apr 04	159.8 ± 4.3	46.2 ± 5.5	18.0 ± 1.5	11.5 ± 2.2	41.1 ± 4.2	88.5 ± 2.2
Jul 04	160.2 ± 4.6	47.3 ± 5.0	18.4 ± 1.3	11.5 ± 2.5	41.8 ± 3.9	88.5 ± 2.5
Sep 04	160.4 ± 4.6	48.8 ± 4.8*	18.9 ± 1.3*	12.3 ± 2.3	42.7 ± 3.5	87.7 ± 2.3*
Dec 04	160.8 ± 4.7	49.6 ± 4.4***	19.2 ± 1.2**	12.3 ± 2.3	43.4 ± 3.1	87.7 ± 2.3***
Apr 05	161.0 ± 4.6**	50.6 ± 4.4***	19.5 ± 0.9***	12.7 ± 1.9	44.1 ± 3.4**	87.3 ± 1.9***
Jul 05	161.1 ± 4.6***	50.7 ± 4.7***	19.5 ± 1.0***	12.6 ± 2.0	44.3 ± 3.6**	87.5 ± 2.0***

*p < 0.05, **p < 0.01, ***p < 0.001 (vs. 1st measurement)

Table 2. The values of systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) in the group of female athletes during the study period (mean ± SD are presented).

	SBP [mm Hg]	DBP [mm Hg]	HR [bpm]
Sep 03	116.8 ± 7.0	67.1 ± 7.9	77.3 ± 14.2
Dec 03	118.2 ± 8.2	66.5 ± 7.1	73.2 ± 14.1
Apr 04	115.2 ± 6.8	69.9 ± 7.5	76.0 ± 14.2
Jul 04	116.2 ± 5.6	67.4 ± 5.9	69.3 ± 12.2
Sep 04	115.8 ± 6.9	66.1 ± 6.5	75.8 ± 14.8
Dec 04	111.4 ± 11.1	67.3 ± 6.0	74.9 ± 16.3
Apr 05	114.3 ± 5.2	65.0 ± 7.2	72.3 ± 12.6
Jul 05	116.3 ± 4.1	68.8 ± 6.1	68.3 ± 10.8*

*p < 0.05 vs. 1st measurement

The changes in the QRS voltage criteria under study are shown in Figure 1. The mean values of QRSmax and of SLI decreased gradually in the study period, and the values at the end of the study period differed significantly with respect to the initial values. The difference between the mean QRSmax values at the beginning and at the end of the study period was -0.8 mV (30.8%), and the difference between the initial and final values of SLI was -0.6 mV (24%).

Discussion

The main results of this study were the significant decrease in the QRSmax and SLI values, respectively. These findings were in contrast with the findings of increased QRS amplitude in athletes.

Increased QRS voltage is more frequently found in highly trained athletes as compared to sedentary controls [16, 17], and was shown to be enhanced with increased level of training [18]. However, the proportion of athletes exceeding the upper normal

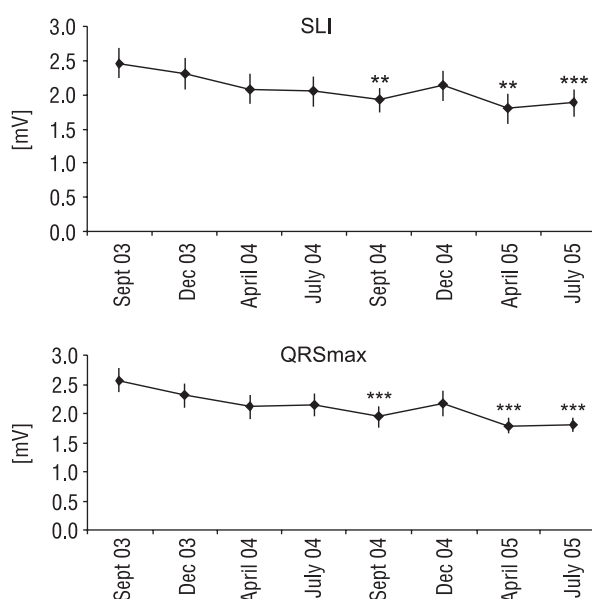


Figure 1. Values of Sokolow-Lyon index (SLI) and of approximated maximum spatial QRS vector magnitude (QRSmax) during the follow-up period. Values are presented as mean, error bar indicates standard error of the mean; **p < 0.01, ***p < 0.001 (vs. 1st measurement).

limits is up to 47 percent in highly trained athletes [5], which means, on the other hand, that more than half of the results are within normal limits. In addition, there is a poor correlation between echocardiographic LVH and ECG LVH [6]. The classical reasoning for the poor performance of voltage criteria in athletes includes the influence of anthropometric parameters, the effects of increased parasympathetic tone on myocardium, the type of exercise and the changes in chamber filling.

In this study, the girls were taller at the end of the follow-up period and they increased their body

weight and BMI due to active body mass. However, the increase in height in the healthy population is associated with the augmentation of QRS; similarly, the body weight shows the same association [19, 20]. The increased BMI and obesity are reported to decrease significantly the QRS voltage [21–23]. However, Eisenstein et al. [24] and Frank et al. [25] show that low voltage is not a significant feature in the ECG of obese people. Furthermore, the increase in BMI in our study group was not due to increased percentage of fat, but to increased active body mass. Therefore, we do not suppose that the decrease in QRS voltage found in this study can be attributed to the changes in anthropometric parameters.

The decrease in HR (bradycardia), as a consequence of increased parasympathetic tone, is regularly seen in top athletes, and is considered to be a sign of well-trained athletes [26, 27]. However, in this study, a significant decrease in HR was observed only at the end of the follow-up period after 21 months of intensive training so the average value of 68 bpm cannot be classified as bradycardia. Therefore, we have no support for attributing the changes in QRS voltage to the changes in parasympathetic tone.

With respect to the type of exercise, two different structural forms of athletes' hearts can be defined, depending on the predominant type of exercise undertaken [28]. In the isotonic type of exercise, the left ventricular chamber size increases with normal wall thickness resulting in an increase in LVM. In isometric type (strength trained) athletes, an increased LVM is observed as a result of increased wall thickness and normal mean LV end-diastolic volume. Some sports involve combined isotonic and isometric types of exercise and are expected to show intermediate changes [29]. Fagard [30] points out that the development of so-called eccentric or concentric LVH, according the type of sport, cannot be regarded as an absolute and dichotomous concept since the training regimens and sports activities are not exclusively dynamic or static, and the load on the heart is not purely of the volume or the pressure type [31]. In addition, Bjornstad et al. [18] show that the differences in ECG findings are relatively minor and do not distinguish the type of sports activity. The nature of exercise undertaken in aerobic gymnastics can be defined as combined exercise; therefore, changes in both LVM and LV dimensions could be expected. So far, no data have been obtained specifically on aerobic gymnastics since this discipline has been established rather recently (the first World Championship

organized by the International Federation of Gymnastics was held in 1995). Nevertheless, none of the changes in heart geometry in athletes was reported to lead to a decrease in QRS amplitude.

The effect of the changes in ventricular blood filling is usually interpreted by the application of the Brody effect [32]. However, there is a certain inconsistency in the application of the Brody effect. First, the original Brody effect considers the changes in conductivity of the intracavitary blood, and not the changes in intracavitary volume; second, the interpretation of the Brody effect is controversial, as is shown in the analysis of its citation [10]. Therefore, the application of the Brody effect to the explanation of changes in QRS voltage due to change in ventricular volume we do not consider plausible.

In this study, two QRS voltage parameters were used. The Sokolow-Lyon index represents a classical clinical ECG voltage parameter for LVH detection. It is calculated as the sum of two defined leads, i.e. it reflects changes in the cardiac electric field only in the plane defined by these two particular leads — in the horizontal plane. The reasoning for the use of the maximum spatial QRS vector is its rationality: firstly, it involves changes in all three spatial components (anterio-posterior, vertical and left-to-right), and secondly, it does not depend on the position of the heart.

The lack of the evidence of the influence of the “classical” factors on the QRS amplitude in this study favours the alternative explanation we hypothesize. The alternative hypothesis considers the false negative ECG results in LVH diagnostics as the reflection of the relative voltage deficit caused by changes in active and passive electrical properties of myocardium in LVH [11]. In our previous studies, we have shown the decrease in the absolute QRS amplitude as well as in the QRS to LVM ratio (the specific potential of myocardium) in the early stage of experimental models of pathological LVH, both volume and pressure overload induced [12, 13]. Similar results, e.g. the decrease in QRS amplitude and in the SP, are observed also in the early stage of exercise-induced left ventricular hypertrophy in swimming normotensive rats [14]. In the literature, we have found so far only one paper reporting an initial decrease in QRS amplitude due to training — in obese children who participated in a jogging program. Hayashi et al. [33] report a decrease in SV1 + RV5 voltage after 3 months of exercise training, and a return to pre-training values is observed one year after training.

Clinical implications

Papers on ECGs in athletes are focused mainly on the clinical significance of the wide range of ECG abnormal patterns that may be found in trained athletes. In addition, the recommendations on the systematic ECG screening are focused mainly on the identification of ECG abnormalities [34–36]. Little attention is paid to changes within normal limits, which might bear additional relevant information on the status of the athlete's heart in the early phase of the intensive training.

Limitations of the study

The number of girls in this study is relatively small. While at the beginning, 17 girls entered the program, during the two-year follow-up period the number decreased to 12. Only those girls who participated continuously in the competitive training during the whole study period were included in this study. In this arrangement, the girls served as their own control and the influence of inter-individual variability was reduced.

Echocardiography was not performed to quantify the changes in LVM and in the morphology of the heart since only a slight increase in LVM might be theoretically expected in this study, which would not explain the decrease in QRS voltage. It was shown that regular intensive physical training in highly trained adults is associated with a modest increase in cardiac dimensions [1, 2, 37]. Sharma [37] showed that highly trained adolescents have only a slightly greater dimension of LV and LVM — they are physically less mature and have been training intensively for a shorter period.

In the design of this study, we did not use a control group. We wanted to avoid biases possibly arising from the different constitutional characteristics, incomparable life style, physical load and psychological stress between competitive athletes and sedentary controls. Furthermore, the number of girls was relatively small, which could additionally lead to a bias by selection in the control group.

Conclusions

In this study, we showed that a 21-month competitive training programme in young female athletes did not lead to the classically expected increase in QRS voltage. On the contrary, the results supported the hypothesis that there would be a decrease in the QRS amplitude at the early stages of intensive training. This decrease could not be attributed to changes in anthropometric parameters, increased parasympathetic influence, or the

type of training. We suppose that the decrease in the QRS amplitude could be an early sign of the rebuilding of myocardium, reflecting the changes in electrical properties of myocardium at the early stage of LHV development. These results stress the need to pay attention also to decreasing QRS values, to the changes “within normal limits”.

References

1. Fagard R. Athlete's heart. *Heart*, 2003; 89: 1455–1461.
2. Oakley D. General cardiology: The athlete's heart. *Heart*, 2001; 86: 722–726.
3. Peronnet F, Perrault H, Cleroux J et al. Electro- and echocardiographic study of the left ventricle in man after training. *Eur Appl Physiol Occup Physiol*, 1980; 45: 125–130.
4. Zakyntinos E, Vassilakopoulos T, Mavrommati I, Filappatos G, Roussos C, Zakyntinos S. Echocardiographic and ambulatory electrocardiographic findings in elite water-polo athletes. *Scand J Med Sci Sports*, 2001; 11: 149–155.
5. Bjornstad H, Smith G, Storstein L, Meer HD, Hals O. Electrocardiographic and echocardiographic findings in top athletes, athletic students and sedentary controls. *Cardiology*, 1993; 82: 66–74.
6. Somauroo JD, Pyatt JR, Jackson M, Perry RA, Ramsdale DR. An echocardiographic assessment of cardiac morphology and common ECG findings in teenage professional soccer players: reference ranges for use in screening. *Heart*, 2001; 85: 649–654.
7. Douglas PS, O'Toole ML, Hiller WD, Hackney K, Reichek N. Electrocardiographic diagnosis of exercise-induced left ventricular hypertrophy. *Am Heart J*, 1988; 116: 784–790.
8. George KP, Wolfe LA, Burggraf GW, Norman R. Electrocardiographic and echocardiographic characteristics of female athletes. *Med Sci Sports Exerc*, 1995; 27: 1362–1370.
9. Kansal S, Roitman DI, Sheffield LT. A quantitative relationship of electrocardiographic criteria of left ventricular hypertrophy with echocardiographic left ventricular mass: a multivariate approach. *Clin Cardiol* 1983; 6: 456–463.
10. Bacharova L. Evidence-based medicine: A lesson for electrocardiography? *Arq Bras Cardiol*, 2003; 81: 102–110.
11. Bacharova L, Kyselovic J. Electrocardiographic diagnosis of left ventricular hypertrophy: is the method obsolete or should the hypothesis be reconsidered? *Med Hypotheses*, 2001; 57: 487–490.
12. Bacharova L, Bernadic M, Fizekova A. Electrocardiographic manifestation of experimental left ventricular

- hypertrophy. In: Jagielski J, Gornicki M eds. *Electrocardiology* 91. World Scientific Publ Co, Singapore 1992: 29–32.
13. Bacharova L, Kyselovic J, Klimas J. The initial stage of left ventricular hypertrophy in spontaneously hypertensive rats is manifested by a decrease in the QRS amplitude/left ventricular mass ratio. *Clin Exp Hypertens*, 2004; 26: 557–567.
 14. Bacharova L, Michalak K, Kyselovic J, Klimas J. The relation between QRS amplitude and left ventricular mass in the initial stage of exercise-induced left ventricular hypertrophy in rats. *Clin Exp Hypertens*, 2005; 27: 533–541.
 15. Sokolow M, Lyon TP. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am Heart J*, 1949; 37: 161–186.
 16. Bjornstad H, Storstein L, Meen HD, Hals O. Electrocardiographic findings in athletic students and sedentary control. *Cardiology*, 1991; 79: 290–305.
 17. Sharma S, Whyte G, Elliott P et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med*, 1999; 33: 319–324.
 18. Bjornstad H, Storstein L, Dyre Meen H, Hals O. Electrocardiographic findings according to level of fitness and sport activity. *Cardiology*, 1993; 83: 268–279.
 19. Ishikawa K. Correlation coefficients for electrocardiographic and constitutional variables. *Am Heart J*, 1976; 92: 152–161.
 20. Norman JE Jr, Levy D. Improved electrocardiographic detection of echocardiographic left ventricular hypertrophy: results of a correlated data base approach. *J Am Coll Cardiol*, 1995; 26: 1022–1029.
 21. Levy D, Labib SB, Anderson KM, Christiansen JC, Kannel WB, Castelli WP. Determinants of sensitivity and specificity of electrocardiographic criteria for left ventricular hypertrophy. *Circulation*, 1990; 81: 815–820.
 22. Norman JE Jr, Levy D. Adjustment of ECG left ventricular hypertrophy criteria for body mass index and age improves classification accuracy. The effect of hypertension and obesity. *J Electrocardiol*, 1996; 29 (suppl): 241–247.
 23. Okin PM, Roman MJ, Devereux RB, Kligfield P. Electrocardiographic identification of left ventricular hypertrophy: test performance in relation to definition of hypertrophy and presence of obesity. *J Am Coll Cardiol*, 1996; 27: 124–131.
 24. Eisenstein I, Edelstein J, Sarma R, Sanmarco M, Selvester RH. The electrocardiogram in obesity. *J Electrodiol*, 1982; 15: 115–118.
 25. Frank S, Colliver JA, Frank A. The electrocardiogram in obesity: statistical analysis of 1,029 patients. *J Am Coll Cardiol*, 1986; 7: 295–299.
 26. Viitasalo MT, Kala R, Eisalo A. Ambulatory electrocardiographic findings in young athletes between 14 and 16 years of age. *Eur Heart J*, 1984; 5: 2–6.
 27. Hanne-Paparo N, Drory Y, Schoenfeld Y, Shapira Y, Kellermann JJ. Common ECG changes in athletes. *Cardiology*, 1976; 61: 267–278.
 28. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. *Ann Intern Med*, 1975; 82: 521–524.
 29. Spirito P, Pelliccia A, Proschan MA et al. Morphology of the „athlete’s heart” assessed by echocardiography in 947 elite athletes representing 27 sports. *Am J Cardiol*, 1994; 74: 802–806.
 30. Fagard RH. Impact of different sports and training on cardiac structure and function. *Cardiol Clin*, 1997; 15: 397–412.
 31. Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE. The athlete’s heart. A meta-analysis of cardiac structure and function. *Circulation*, 2000; 101: 336–344.
 32. Brody DA. The theoretical analysis of intracavitary blood mass influence on the heart-lead relationship. *Circ Res*, 1956; 4: 731–738.
 33. Hayashi T, Fujino M, Shindo M, Hitoki T, Arakawa K. Echocardiographic and electrocardiographic measures in obese children after an exercise program. *Int J Obes*, 1987; 11: 465–472.
 34. Bader RS, Goldberg L, Sahn DJ. Risk of sudden death in young athletes: which screening strategies are appropriate? *Pediatr Clin North Am*, 2004; 51: 1421–1441.
 35. Wingfield K, Matheson GO, Meeuwisse WH. Pre-participation evaluation: an evidence-based review. *Clin J Sport Med*, 2004; 14: 109–122.
 36. Corrado D, Pelliccia A, Bjornstad HH et al.; Study Group of Sport Cardiology of the Working Group of Cardia Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology: Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*, 2005; 26: 516–524.
 37. Sharma S. Athlete’s heart-effect of age, sex, ethnicity and sporting discipline. *Exp Physiol*, 2003; 88: 665–669.