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Assessment of right heart function in the athlete's heart

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Abstract It is known that the heart of an athlete has been physiologically adapted by prolonged training. There are a large number of echocardiographic studies which have focused on left ventricular wall thickness and dilatation, but there are few studies concerning right heart function in the athlete's heart. The aim of this study was to assess right heart function in elite athletes by conventional and new echocardiographic methods. The study population consisted of 36 elite highly-trained male athletes and 16 age-matched healthy sedentary controls. Right atrial, right ventricular, and inferior vena cava dimensions, and pulsed Doppler measurements of tricuspid inflow and right ventricular outflow were obtained, and systolic (preejection period, ejection time, preejection time/ejection time, QV peak, isovolumic contraction time) and diastolic (E peak, A peak, E/A ratio, decelaration time, isovolumic relexation time) function parameters were measured. The myocardial performance index was calculated as (isovolumetric contraction time + isovolumetric relaxation time)/ejection time. In addition, right ventricular systolic and diastolic functions were determined by Pulsed wave tissue Doppler imaging (S, E, and A velocities) at the lateral corners of the tricuspid annulus. The left ventricular mass index ($P < 0.005$), and right atrial ($P < 0.001$), right ventricular ($P < 0.001$), and inferior vena cava dimensions ($P < 0.001$) were significantly greater in athletes than in controls. Tricuspid E peak, A peak, E/A ratio, deceleration time, isovolumic relaxation time, preejection period, right ventricular ejection time, preejection time/ejection time, isovolumic contraction time, QV peak, and myocardial performance index were found to be similar in athletes and in controls ($P > 0.05$). Systolic, early diastolic, and late diastolic tissue Doppler imaging velocities were not significantly different in athletes and controls ($P > 0.05$). Left ventricular hypertrophy (LV mass index $>134\,g/m^2$) was found in 15 of the athletes. Right atrial dimension was greater in the athletes with left ventricular hypertrophy than in those without hypertrophy ($P < 0.05$). All right ventricular systolic and diastolic echocardiographic parameters were similar in athletes with and without left ventricular hypertrophy ($P > 0.05$). The results of this study indicate that right ventricular systolic and diastolic functions do not deteriorate in the athlete's heart despite significant chamber dilatation. They suggest that these changes are a normal physiologic adaptation to prolonged training.

Key words Athlete's heart · Myocardial performance index · Tissue Doppler imaging

Introduction

Physical activity is associated with hemodynamic changes and alters the loading conditions of the heart. Therefore the heart of the athlete has been physiolologically adapted by prolonged training. These changes include an increase in left ventricular chamber size, wall thickness and mass, and a decrease in the resting heart rate.[1–3] The development of echocardiography has allowed an understanding of the physiologic consequences of chronic training. It is known that an increased left ventricular mass occurs due to increased cavity dimensions, wall thickness, or both, in athletes who undergo sufficient intensity repetitive training, and left ventricular systolic and diastolic functions are not deteriorated in athletes despite the significant hypertrophy. Thus, this form of left ventricular hypertrophy is called physiologic hypertrophy.[4–6] Although there are many echocardiographic studies concerning left ventricular function, there are only a few studies on right heart function in the athlete's heart. Because of complex geometry of the right ventricle and its position beneath the sternum, the echocardiographic assessment of right ventricular function

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is difficult. The myocardial performance index is a new parameter assessing global ventricular function, and pulsed wave tissue Doppler imaging echocardiography (TDI) is a new technique that allows the assessment of regional myocardial velocity. The aim of this study was to assess right ventricular systolic and diastolic functions by conventional and new echocardiographic methods in elite male athletes.

Subjects and methods

Subjects

Thirty-six highly trained male athletes (mean age 21.8 ± 3.6 years) and 16 normally active age-matched control subjects (mean age 23.1 ± 0.6 years) were included. All of the athletes of the study were members of professional sports teams (14 runners, 10 wrestlers, 4 boxers, 5 basketball players, and 3 skiers). Mean athletic competition time was 7.7 ± 4.1 years and mean average training time was 11.5 ± 3.9 h/week in the athletes' group. All subjects were screened by history, physical examination, and electrocardiography, and they were all free from cardiac disease. All athletes were in an intense training period. Athletes in an off-training period or during prolonged resting (>10 days) were excluded. Subjects who had a history of cardioactive medication or anabolic steroid use were excluded from the study. The ethics committee of our institute approved the study protocol and all subjects gave written informed consent for the study.

Echocardiographic study

A Vingmed System Five Doppler echocardiographic unit (GE Vingmed Ultrasound, Horten, Norway) with a 2.5-MHz FPA probe was used. Two-dimensional and pulse-wave Doppler echocardiographic studies were performed in the left lateral decubitus position with the conventional views (parasternal long and short axis, apical four-chamber) and the supine position for the subxiphoid approach. An electrocardiogram was recorded simultaneously with the M-mode and Doppler tracings in the same monitor, and 50 mm/s M-mode sweeping speed was used for M-mode trace recording. Diastolic left ventricular septal thickness, diastolic posterior wall thickness, and left ventricular end-diastolic and end-systolic dimensions were measured in the parasternal long-axis view, and left ventricular mass was determined by the method of Devereux and Reichek, and indexed to body surface area.[7] Right atrial (RA) dimension was determined in the four-chamber view by the maximal medial to lateral dimension at end-ventricular systole (which corresponds to maximal atrial volume). Right ventricular (RV) dimension was determined in the four-chamber view with the maximal medial to lateral dimension at mid-cavity at end-diastole.[8] Tricuspid inflow velocity was recorded from the apical four-chamber view by pulsed-wave Doppler sample volume positioned at the tips of the tricuspid leaflets during diastole. Peak early (E) and late (A) tricuspid inflow velocity, E/A ratio, and deceleration time of E velocity were obtained. The RV outflow velocity was recorded from the parasternal short-axis view with the pulsed-wave Doppler sample volume positioned just below pulmonary valve. Preejection period (PEP) was measured from the onset of the QRS wave to the onset of RV ejection flow. RV ejection time (RVET) was measured from the onset to the end of RV outflow. Isovolumetric relaxation time (IRT) was obtained as the time interval from the cessation of RV outflow to the onset of tricuspid valve inflow. Isovolumetric contraction time (ICT) was determined from the cessation of tricuspid inflow to the onset of RV outflow.[9] QV peak (QVP) was measured from the onset of the QRS wave to the peak of pulmonary flow velocity.[10] Myocardial performance index (MPI) was calculated by the formula (ICT + IRT)/RVET (Fig. 1).[9,11] Tissue Doppler imaging was applied in the pulse-Doppler mode of the tricuspid annulus velocity at its lateral corners with the same echocardiographic unit, and systolic (S), early diastolic (E), and late diastolic (A) velocy was measured. Inferior vena cava diameter was determined in the subxiphoid approach 10 mm away from its junction with the right atrium as the median value between maximal inspiratory and maximal expiratory values.[8]

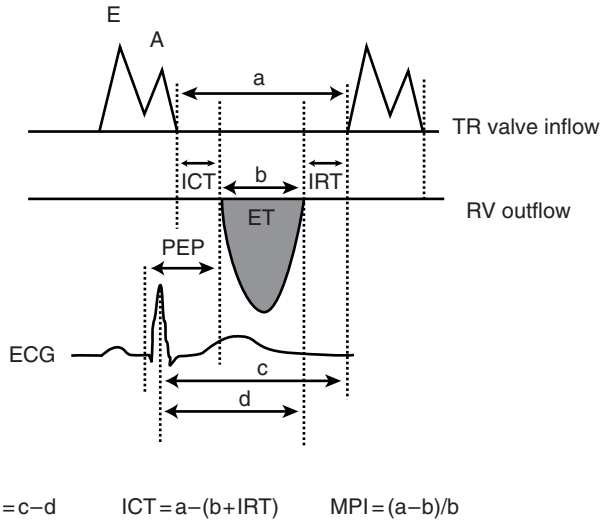

Fig. 1. Measurement of right ventricular preejection period (*PEP*), ejection time (*ET*), isovolumetric contraction time (*ICT*), isovolumetric relaxation time (*IRT*), and calculation of myocardial performance index (*MPI*)

The same investigator performed all echocardiograms. The echocardiographic measurements were repeated to assess intraobserver variability in ten subjects by the same investigator. All measurements were averaged over three cardiac cycles.

Statistical analysis

Data were expressed as mean ± SD. The differences between athletes and controls, athletes with hypertrophy, and athletes without hypertrophy were assessed with Student's

t-test. Analysis of variance (Dunnett) with Bonferroni correction was used to assess the differences between controls and athletes' subgroups. Intraobserver reproducibility was analyzed using Pearson's correlation coefficient and paired Student's *t*-test. A value of $P < 0.05$ was taken as statistically significant.

Results

The athletes and members of the control group did not differ significantly in mean age (21.8 ± 3.6 vs 23.1 ± 0.6 years, $P > 0.05$) and body surface area (1.82 ± 0.17 vs 1.89 ± 0.21 m^2, $P > 0.05$). Heart rate was significantly lower in the athletes than in the controls (57.0 ± 10.7 vs 77.5 ± 9.9 beats/min, $P < 0.001$). Systolic and diastolic blood pressure was similar between two groups ($P > 0.05$).

RA dimension ($P < 0.001$), RV dimension ($P < 0.001$), IVC diameter ($P < 0.001$), and left ventricular mass index ($P < 0.005$) were significantly greater in the athletes than in controls (Table 1). All RV systolic and diastolic function parameters detected by Doppler echocardiography in the athletes were not significantly different from those of the control group ($P > 0.05$) (Table 2).

Left ventricular hypertrophy (LV mass index >134 g/m^2) was found in 15 subjects of the athletes' group. LV mass index was 160.8 ± 21.5 g/m^2 in the athletes with left ventricular hypertrophy and 111.8 ± 17.7 g/m^2 in those without left ventricular hypertrophy ($P < 0.001$). RA dimension was greater in the athletes with left ventricular hypertrophy than in athletes without hypertrophy ($P < 0.05$). RV dimension was also greater in athletes with hypertrophy than in those without hypertrophy, but the difference was not statistically significant. All echocardiographic parameters measured in the athletes with and without left ventricular hypertrophy are presented in Table 3.

Table 1. Echocardiographic dimensions and left ventricular mass indexes in athletes and controls

	Controls ($n = 16$)	Athletes ($n = 36$)
Right atrial dimension (mm)	37.6 ± 3.3	45.6 ± 6.1**
Right ventricular dimension (mm)	35.3 ± 4.5	45.3 ± 6.2**
Inferior vena cava dimension (mm)	15.2 ± 1.7	17.8 ± 2.2**
Left ventricular mass index (g/m^2)	102.5 ± 21.6	132.2 ± 31.1*

* $P < 0.005$, ** $P < 0.001$

Table 2. Right ventricular systolic and diastolic function parameters

	Controls ($n = 16$)	Athletes ($n = 36$)	*P* value
E peak (cm/s)	50.5 ± 9.5	52.2 ± 11.0	NS
A peak (cm/s)	35.1 ± 11.6	31.9 ± 7.4	NS
E/A ratio	1.5 ± 0.5	1.7 ± 0.5	NS
Deceleration time (ms)	140.0 ± 36.6	150.9 ± 47.0	NS
Isovolumic relaxation time (ms)	74.9 ± 45.5	67.4 ± 42.3	NS
Preejection period (ms)	98.5 ± 23.5	96.7 ± 15.8	NS
Ejection time (ms)	271.5 ± 25.9	266.6 ± 43.2	NS
Preejection period/ejection time	0.37 ± 0.1	0.37 ± 0.1	NS
Isovolumetric contraction time (ms)	67.4 ± 23.0	58.6 ± 23.7	NS
QV peak (ms)	227.5 ± 18.3	225.2 ± 25.4	NS
Myocardial performance index	0.29 ± 0.1	0.31 ± 0.1	NS

NS, not significant

Table 3. Right ventricular systolic and diastolic function parameters measured in athletes with and without left ventricular (LV) hypertrophy

	Left ventricular hypertrophy		
	(−) ($n = 21$)	(+) ($n = 15$)	*P* value
LV mass index (g/m^2)	111.8 ± 17.7	160.8 ± 21.5	<0.001
Right atrial dimension (mm)	43.5 ± 5.9	48.4 ± 5.2	<0.05
Right ventricular dimension (mm)	43.7 ± 5.6	47.6 ± 6.5	0.09
Inferior vena cava dimension (mm)	1.75 ± 0.2	1.83 ± 0.3	NS
E peak (cm/s)	54.5 ± 12.2	49.0 ± 8.4	NS
A peak (cm/s)	32.0 ± 7.1	31.7 ± 7.9	NS
E/A ratio	1.8 ± 0.6	1.6 ± 0.3	NS
Deceleration time (ms)	141.7 ± 46.7	163.7 ± 45.9	NS
Isovolumic relaxation time (ms)	67.8 ± 41.1	66.9 ± 45.4	NS
Preejction period (ms)	93.5 ± 12.5	101.2 ± 18.8	NS
Ejection time (ms)	260.2 ± 43.4	275.5 ± 42.8	NS
Preejection period/ejection time	0.36 ± 0.1	0.38 ± 0.1	NS
Isovolumetric contraction time (ms)	57.0 ± 24.4	61.1 ± 23.4	NS
QV peak (ms)	218.2 ± 19.9	235.7 ± 29.6	NS
Myocardial performance index	0.31 ± 0.1	0.32 ± 0.1	NS

Table 4 summarizes the mean values of peak systolic, early diastolic, and late diastolic velocities measured by TDI. Systolic, early diastolic, and late diastolic velocities at the lateral corner of tricuspid annulus were not significantly different between the athletes and controls ($P > 0.05$). Systolic, early diastolic, and late diastolic TDI velocities were also similar in athletes with and without left ventricular hypertrophy (Table 5).

In the subgroups, RA dimension was significantly greater in runners ($P < 0.001$) and wrestlers ($P < 0.05$) than in controls. RV dimension was also significantly greater in runners ($P < 0.001$) and in wrestlers ($P < 0.01$) than in controls. Inferior vena cava diameter was significantly greater in runners ($P < 0.05$) than in controls. The systolic and diastolic function parameters measured by conventional and tissue Doppler echocardiography were not significantly different in the athlete subgroups compared with controls (Tables 6 and 7).

Table 4. Pulsed tissue Doppler imaging variables measured in athletes and controls

	Controls ($n = 16$)	Athletes ($n = 36$)	P values
Systolic velocity (cm/s)	14.46 ± 2.0	14.87 ± 2.56	NS
Early diastolic velocity (cm/s)	16.59 ± 4.73	16.97 ± 3.23	NS
Late diastolic velocity (cm/s)	11.78 ± 2.19	10.91 ± 1.94	NS
E/A ratio	1.51 ± 0.78	1.59 ± 0.35	NS

Regarding reproducibility, intraobserver variability was acceptable for all echocardiography parameters. Mean absolute differences were acceptable, and Pearson correlation coefficients were >0.90 for all parameters ($r = 0.99$, $P < 0.001$ for RV; $r = 0.94$, $P < 0.001$ for RA; $r = 0.98$, $P < 0.001$ for E; $r = 0.99$, $P < 0.001$ for A; $r = 0.97$, $P < 0.001$ for DT; $r = 0.99$, $P < 0.001$ for MPI; $r = 0.98$, $P < 0.001$ for PEP; $r = 0.98$, $P < 0.001$ for ET; $r = 0.98$, $P < 0.001$ for TDI S wave; $r = 0.97$, $P < 0.001$ for TDI E wave; $r = 0.97$, $P < 0.001$ for TDI A wave).

Discussion

The echocardiographic assessment of right ventricular function is difficult because of the complex geometry of the ventricle and its position beneath the sternum. Thus, studies indicating right ventricular function in the athlete's heart are scarce. Determination of systolic and diastolic time intervals are important because active energy cycles of contraction and relaxation occur predominantly in these periods.[11] The results of our study indicated that right ventricular systolic and diastolic time intervals were similar in the athlete's heart as compared to controls. MPI is a Doppler-derived nongeometric measure of ventricular function, and reflects both systolic and diastolic function. It was reported that MPI was a useful index for assessment of right ventricular function and appeared to be relatively independent of changes in preload or afterload in the clinical

Table 5. Pulsed tissue Doppler imaging variables measured in athletes with and without left ventricular hypertrophy

	Left ventricular hypertrophy		P value
	(−) ($n = 21$)	(+) ($n = 15$)	
Systolic velocity (cm/s)	14.6 ± 2.57	15.24 ± 2.59	NS
Early diastolic velocity (cm/s)	17.46 ± 3.61	16.27 ± 2.58	NS
Late diastolic velocity (cm/s)	10.86 ± 2.03	10.99 ± 1.86	NS
E/A ratio	1.64 ± 0.41	1.50 ± 0.23	NS

Table 6. Echocardiographic parameters measured in controls and athlete subgroups

	Controls ($n = 16$)	Runners ($n = 14$)	Wrestlers ($n = 10$)	Boxers ($n = 4$)	Basketball players ($n = 5$)	Skiers ($n = 3$)
Right atrial dimension (mm)	37.6 ± 3.3	49.1 ± 5.2***	43.8 ± 5.3*	41.5 ± 7.1	42.4 ± 6.3	45.3 ± 5.6
Right ventricular dimension (mm)	35.3 ± 4.5	48.9 ± 4.9***	44.4 ± 5.8**	41.5 ± 8.1	41.0 ± 4.5	43.0 ± 7.0
Inferior vena cava dimension (mm)	15.2 ± 1.7	18.1 ± 2.5*	17.5 ± 2.0	19.2 ± 1.8	17.5 ± 1.6	16.8 ± 0.35
E peak (cm/s)	50.5 ± 9.5	52.9 ± 10.5	50.1 ± 7.3	54.8 ± 20.6	53.2 ± 15.1	50.9 ± 3.1
A peak (cm/s)	35.1 ± 11.6	30.4 ± 8.6	32.8 ± 7.3	32.2 ± 5.1	31.5 ± 8.2	35.8 ± 2.9
E/A ratio	1.5 ± 0.5	1.8 ± 0.3	1.6 ± 0.4	1.8 ± 0.9	1.8 ± 0.7	1.4 ± 0.03
Deceleration time (ms)	140.0 ± 36.6	134.0 ± 36.9	172.4 ± 60.2	151.0 ± 11.9	140.0 ± 54.5	176.0 ± 42.1
Isovolumic relaxation time (ms)	74.9 ± 45.5	64.6 ± 43.9	54.4 ± 31.2	55.0 ± 6.0	84.8 ± 61.5	104.0 ± 56.0
Preejection period (ms)	98.5 ± 23.5	96.1 ± 14.2	104.4 ± 17.1	93.0 ± 8.9	90.4 ± 20.3	89.3 ± 16.7
Ejection time (ms)	271.5 ± 25.9	272.9 ± 40.5	272.8 ± 32.9	287.0 ± 18.3	245.6 ± 60.8	224.0 ± 64.4
Preejection period/ejection time	0.37 ± 0.1	0.37 ± 0.11	0.39 ± 0.08	0.33 ± 0.04	0.38 ± 0.08	0.41 ± 0.10
Isovolumetric contraction time (ms)	67.4 ± 23.0	54.3 ± 23.3	72.00 ± 20.56	51.0 ± 27.4	46.0 ± 27.2	61.3 ± 20.1
QV peak (ms)	227.5 ± 18.3	224.9 ± 23.7	233.6 ± 30.9	217.0 ± 20.5	221.2 ± 27.6	216.0 ± 22.3
Myocardial performance index	0.29 ± 0.1	0.30 ± 0.10	0.32 ± 0.08	0.27 ± 0.04	0.28 ± 0.07	0.42 ± 0.2

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

Table 7. Tissue Doppler parameters measured in controls and athlete subgroups

	Controls ($n = 16$)	Runners ($n = 14$)	Wrestlers ($n = 10$)	Boxers ($n = 4$)	Basketball players ($n = 5$)	Skiers ($n = 3$)
Systolic velocity (cm/s)	14.46 ± 2.0	14.90 ± 2.7	15.1 ± 3.0	13.40 ± 1.92	14.58 ± 2.41	16.37 ± 0.7
Early diastolic velocity (cm/s)	16.59 ± 4.73	16.87 ± 3.50	16.78 ± 2.98	16.95 ± 3.22	18.36 ± 4.21	15.7 ± 21.0
Late diastolic velocity (cm/s)	11.78 ± 2.19	10.89 ± 1.92	11.30 ± 1.90	10.25 ± 2.90	10.68 ± 1.88	10.97 ± 2.0
E/A ratio	1.51 ± 0.78	1.58 ± 0.36	1.50 ± 0.27	1.70 ± 0.36	1.77 ± 0.56	1.44 ± 0.07

setting.[9] We found that right ventricular MPI was also similar in athletes and controls. This result suggests that global right ventricular function does not deteriorate in the athlete's heart despite significant chamber dilatation. There are many studies indicating right ventricular dilatation in elite athletes, which is consistent with our results, but all of them show only chamber dilatation.[11–14] Our study indicated also that there was no deterioration in right ventricular systolic and diastolic functions in the athlete's heart. Tissue Doppler myocardial imaging, a new ultrasound technique, allows the quantification of myocardial velocities. Although we did not measure right ventricular wall thickness, the velocities measured at the lateral corners of tricuspid annulus by the TDI were not different in athletes compared with controls. Also, this result suggests that RV functions do not deteriorate in athletes.

During exercise, both pulmonary and systemic circulation must show the same amount of increase to meet the circulatory demands of prolonged physical exercise. Since total cardiac work is responsible for the structural changes in the heart, right ventricular enlargement in the athlete's heart probably reflects the increased hemodynamic loading caused by prolonged training. In contrast to pathologic right heart dilatation, such as chronic cor pulmonale or primary pulmonary hypertension, right ventricular systolic and diastolic functions are normal in the athlete's heart. This result suggests that this right ventricular dilatation is physiologic but not pathologic. We also found that there was a significant inferior vena cava dilatation in the athletes compared with controls which shows compatibility with the study of Goldhammer et al.[8] Inferior vena cava diameter is also greater in patients with right heart disease and pericardial diseases. Despite the absence of cardiac pathologic conditions, the dilated inferior vena cava suggests extracardiac adaptation to chronic strenuous exercise. This may be due to chronically augmented venous return from the lower part of the body, causing increased volume load with increased cardiac output in athletes.

Right ventricular and right atrial dimensions were greater in athletes with left ventricular hypertrophy than without. This may well suggest that the occurrence of left ventricular hypertrophy depends largely on the intensity of training, which ultimately induces more left ventricular hypertrophy. Thus, preload will inevitably result in enlargement of the right heart. The fact that athletes with left ventricular hypertrophy have greater right heart dimensions may be associated with the intensity of their training. The similarity between systolic and diastolic right ventricular function parameters in athletes with left ventricular hypertrophy and those without hypertrophy demonstrates that left ventricular hypertrophy does not cause a deterioration in right heart function in the athletes. Left ventricular hypertrophy (LV mass $>134\,g/m^2$) was found in 15 athletes in our study, which is consistent with earlier reports in elite male athletes. It was reported that left ventricular cavity enlargement and wall thickening was often slight and within the normal range but led to a significant elevation in some athletes.[1] The reason why all athletes do not show left ventricular hypertrophy could be the difference in athletic competition time (from 2 to 17 years) and training time (from 4 h to 21 h/week). In addition, the impact of different sports and training structure may affect this result. It is known that the athlete's heart shows physiologic and not pathologic hypertrophy, and LV systolic and diastolic function is normal. The results of our study suggest that right ventricular as well as left ventricular function does not deteriorate in athletes.

In the athlete subgroups, although the number of athletes in each subgroup was not enough to conduct a statistical analysis (three skiers, five basketball players, four boxers), right atrial and right ventricular dimensions were greater in all subgroups than in control. The fact that both conventional and tissue Doppler echocardiographic parameters of right ventricular function were not significantly different between the athlete subgroups and controls suggests also this type of training does not cause a deterioration in right ventricular function. However, the fact that each subgroup did not contain enough athletes remains the limitation of our study.

The results of this study indicated that right ventricular systolic and diastolic function does not deteriorate in the athlete's heart despite significant chamber dilatation. In spite of a significant increase in right heart dimensions, the fact that systolic and diastolic right ventricular function is normal suggests that these effects are a part of the physiologic cardiac and extracardiac adaptation to chronic strenuous exercise. We are of the opinion that these changes are normal physiologic adaptations to prolonged training.

References

1. Shapiro LM (1997) The morphologic consequences of systemic training. Cardiol Clin 15:373–379
2. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ (1999) Physiologic left ventricular cavity dilatation in elite male athletes. Ann Intern Med 130:23–31

3. Huston TP, Puffer JC, Rodney WM (1985) The athletic heart syndrome. N Engl J Med 313:4–32
4. Claessens P, Claessens C, Claessens M, Henderieckx J, Claessens J (2000) Physiological or pseudophysiological ECG changes in endurance-trained athletes. Heart Vessels 15:181–190
5. Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE (2000) The athlete's heart: a meta-analysis of cardiac structure and function. Circulation 101:336–344
6. Colan SD (1997) Mechanics of left ventricular systolic and diastolic function in physiologic hypertrophy of the athlete's heart. Cardiol Clin 15:355–372
7. Devereux RB, Reichek N (1977) Echocardiographic determination of left ventricular mass: anatomic validation of the method. Circulation 55:613–618
8. Goldhammer E, Mesnick N, Abinader EG, Sagiv M (1999) Dilated inferior vena cava: a common echocardiographic finding in highly trained elite athletes. J Am Soc Echocardiogr 12:988–993
9. Eidem BW, O'Leary PW, Tei C, Seward JB (2000) Usefulness of myocardial performance index for assessing right ventricular function in congenital heart disease. Am J Cardiol 86:654–658
10. Ohte N, Hashimoto T, Narita H, Takase R, Kobayashi K, Hayano J, Fujinami T (1990) Noninvasive evaluation of left ventricular performance with a new systolic time interval, the Q-V peak, and comparison with established systolic time intervals. Am J Cardiol 66:1018–1020
11. Tei C, Dujardin KS, Hodge DO, Bailey KR, McGoon MD, Tajik AJ, Seward SB (1996) Doppler echocardiographic index for assessment of global right ventricular function. J Am Soc Echocardiogr 9:838–847
12. Hauser AM, Dressendorfer RH, Vos M, Hashimoto T, Gordon S, Timmis GC (1985) Symmetric cardiac enlargement in highly trained endurance athletes: a two-dimensional echocardiographic study. Am Heart J 109:1038–1044
13. Sciomer S, Vitarelli A, Penco M, Liistro F, Schina M, Commisso C, Vizza CD, Dagianti A (1998) Anatomico-functional changes in the right ventricle of the athlete. Cardiologia 43:1215–1220
14. Henriksen E, Landelius J, Wesslen L, Arnell H, Nystrom-Rosander C, Kangro T, Jonason T, Rolf C, Lidell C, Hammarstrom E, Ringqvist I, Friman G (1996) Echocardiographic right and left ventricular measurements in male elite endurance athletes. Eur Heart J 17:1121–1128
15. Henriksen E, Landelius J, Kangro T, Jonason T, Hedberg P, Wesslen L, Rosander CN, Rolf C, Ringqvist I, Friman G (1999) An echocardiographic study of right and left ventricular adaptation to physical exercise in elite female orienteers. Eur Heart J 20:309–316