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Right ventricular myocardial performance index and exercise capacity in athletes

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Abstract Right ventricular function is important for exercise capacity in athletes. The aim of this study was to investigate the effects of habitual exercise training on right ventricular global function. Fifty-two male athletes (25 runners, 27 wrestlers) and 43 age-matched sedentary male subjects were studied. All subjects in the study underwent an echocardiography examination and cardiopulmonary exercise test. Maximal oxygen consumption, right ventricular cavity diameters, and diastolic parameters were higher in the athletes than in controls. However, the right ventricular myocardial performance index was lower in athletes compared with controls. Therefore, the right ventricular myocardial performance index showed a negative correlation with maximal oxygen consumption ($r = -0.61$; $P < 0.001$). The right ventricular myocardial performance index may reflect changes in right ventricular function and exercise capacity in athletes.

Key words Right ventricle · Athlete's heart · Myocardial performance index · Maximal oxygen consumption

Introduction

"Athlete's heart" is an adaptation to long-term training, resulting in an increase in cardiac dimensions and stroke volume as well as in a lower heart rate.[1] A considerable body of echocardiographic studies have described how athletic training induces morphological adaptation of the left ventricle (LV) in athletes.[1–3] On the other hand, diseases of the right ventricle (RV) are also important in athletes, and it has been suggested that structural RV disease, such as RV cardiomyopathy, is a cause of sudden death in athletes.[3]

However, only a few reports have dealt with the echocardiographic estimation of RV adaptation in athletes.[4,5] This is not easy to achieve due to the complex anatomy and geometry of the right ventricle, making the evaluation of its function limited. Therefore, a simple reliable and easy method is needed. Recently, a conceptually new Doppler index that combines the assessment of systolic and diastolic RV performance was proposed by Tei et al.[6] More recently, the myocardial performance index (MPI) has shown promise in the assessment of RV function in adults with various heart diseases.[7–9]

The aims of this study were (1) to compare endurance-trained and strength-trained athletes with untrained controls regarding RV morphology and MPI of RV (RV-MPI), and (2) to assess the relationship between RV-MPI and exercise capacity values in athletes.

Methods

Study population

We screened 30 male Caucasian runners, 32 male Caucasian wrestlers, and 50 Caucasian sedentary males for this study; exclusion criteria led to 25 runners, 27 wrestlers, and 43 sedentary subjects being included in the analyses. We regarded the subjects as athletes when they had been in training for at least 10 h per week for at least 8 years. Subjects were regarded as sedentary controls when they exercised (walking) for less than 2 h per week. Subjects having acute or chronic illnesses in their history or on physical examination were excluded. No subject had a history of hypertension, diabetes mellitus, or alcohol abuse, and none had electrolyte disturbances. No subjects were taking any form of medication. All subjects were free of cardiovascular

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disease as determined by a detailed history and physical examination.

The investigation conformed to the principles outlined in the Declaration of Helsinki and was approved by the local Research Ethics Committee. Written informed consent was obtained from all subjects before participation.

Procedures

All subjects underwent resting Doppler echocardiography and graded treadmill exercise to measure maximal aerobic capacity on the same day. At 08.00–10.00 h, we performed an echocardiography examination then cardiopulmonary exercise testing for each subject.

Echocardiographic measurements

All echocardiographic examinations were performed and measured with a Vingmed System 5 Doppler echocardiographic unit (GE Vingmed Ultrasound, Horten, Norway) by two cardiologists blinded to the grouping of the study subjects. Echocardiography was performed in the lateral decubitus position, and left ventricular dimensions were obtained based on the standards of the American Society of Echocardiography.[10] Left ventricular mass was calculated by using the method of Devereux and Reichek.[11]

Right ventricular cavity (from endocardial border to endocardial border of the RV) measurements were obtained according to the protocol of Foale et al.[12] On the parasternal long-axis view, the RV diameter (RVd_1) was measured from the anterior right ventricular wall to the septum. In the apical four-chamber view, the RV diameter (RVd_2) was measured from the right ventricular apex to the mid-point of tricuspid valve annulus. The RV diameter (RVd_3) was measured from the RV free wall to the septum in the middle of the RV.

The mitral and tricuspid inflow velocity pattern was obtained from the apical four-chamber view, with the sample volume of the pulsed wave Doppler positioned at the tips of the mitral and tricuspid leaflets during diastole. Early diastolic peak flow velocity (E), late diastolic peak flow velocity (A) (cm/s), and the E/A ratio were measured. Deceleration time of the E wave (DT) was measured. DT was corrected for heart rate[13]: $DTc = DT/RR^{1/2}$, where DT is expressed in milliseconds and RR in seconds.

Doppler intervals were measured from the tricuspid inflow and RV outflow velocity time intervals. The interval a is the duration from the cessation to the onset of tricuspid inflow, and the interval b (RV ejection time) is the duration of the RV outflow velocity profile. Intervals a and b were corrected for heart rate. The MPI was defined as (a − b)/b (Fig. 1). The reproducibility of the method has been described previously.[14] M-mode and Doppler measurements were obtained during three consecutive cardiac cycles and averaged.

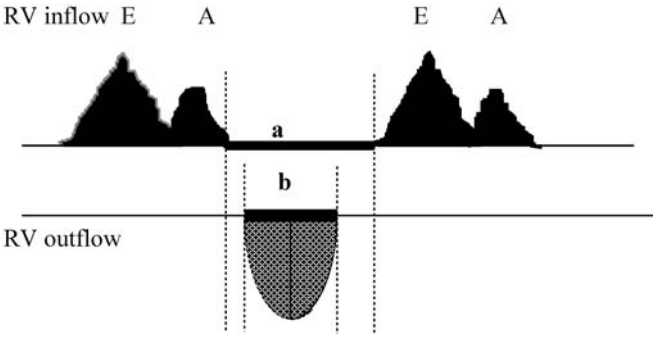

Fig. 1. This diagram shows how to calculate the myocardial performance index of the right ventricle (RV-MPI). The interval *a* is from cessation to onset of right ventricular inflow; the interval *b*, from onset to cessation of RV outflow, is the ejection time. RV-MPI is calculated as (a − b)/b

Cardiopulmonary exercise testing

The exercise was performed in a quiet air-conditioned room with an average temperature of 21° ± 2°C and full resuscitation facilities. All participants underwent a standard Bruce multistage maximal treadmill protocol with metabolic measurements. The exercise was discontinued due to fatigue, symptoms, or other criteria.[15] A standard 12-lead electrocardiogram was monitored continuously and was recorded at rest, during the last 10 s of each exercise stage, at the end of the graded exercise test, and at the end of each minute of the subsequent 3-min recovery. Oxygen consumption was measured every 10 s using a metabolic chart (2900C B × B, Sensormedics, Yorba Linda, CA, USA). Respiratory gas was analyzed using a zirconium oxygen analyzer and a nondispersive infrared sensor for carbon dioxide. Before each test, the gas analyzers were calibrated with two mixtures of gases of known oxygen and carbon dioxide concentration. Basic gas and flow measurements were also corrected for ambient temperature, barometric pressure, and water vapor. Subjects breathed ambient air through a Rudolph two-way valve during the exercise. To ensure that maximal oxygen consumption ($\dot{V}O_{2max}$; ml/kg per minute) was reached, three criteria had to be met: (1) a leveling off of $\dot{V}O_2$ despite an increase in exercise power over the final stages of the test, (2) attainment of age-predicted maximal HR [(210 − 0.65 × age) ± 10 beats/min], and (3) R (respiratory exchange ratio) ≥ 1.10.[16]

Statistical analysis

Values are expressed as the mean ± SD and SE. The independent unpaired *t*-test was used for all athletes when compared with controls. An analysis of variance test was used for comparison of the three groups. Post hoc analysis was done by the Bonferroni test. The relation between RV-MPI and $\dot{V}O_{2max}$ was tested by linear regression analysis. *P* values of less than 0.05 were considered significant.

Table 1. Characteristics of athletes and control subjects

	Athletes (n = 52)	Controls (n = 43)	P value
Age (years)	23.0 ± 3.6	22.1 ± 2.2	0.17
Height (cm)	172.8 ± 6.7	174.8 ± 8.8	0.22
Weight (kg)	71.3 ± 11.5	68.6 ± 9.5	0.22
BSA (m^2)	1.79 ± 0.13	1.82 ± 0.15	0.37
HR (beats/min)	54 ± 9	76 ± 10	<0.001
$\dot{V}O_{2max}$ (ml/kg/min)	64.5 ± 3.8	43.4 ± 3.2	<0.001

Variables are expressed as mean ± SD
BSA, body surface area; HR, heart rate; $\dot{V}O_{2max}$, maximal oxygen consumption

Table 2. Measurements of the left ventricle in athletes and controls

	Athletes (n = 52)	Controls (n = 43)	P value
LVM (g)	218 ± 57	156 ± 11	<0.001
LVMI (g/m^2)	120 ± 29	86 ± 7	<0.001
FS (%)	34.4 ± 4.7	30.7 ± 4.7	<0.001
Em (cm/s)	92.6 ± 11.5	76.4 ± 10.3	<0.001
Am (cm/s)	51.0 ± 7.3	48.1 ± 8.3	0.07
Em/Am	1.9 ± 0.3	1.6 ± 0.3	<0.001
DTm (ms)	205 ± 16	168 ± 13	<0.001
DTm_c (ms)	193 ± 14	189 ± 13	0.10

Variables are expressed as mean ± SD
LVM, left ventricular mass; LVMI, left ventricular mass index; Em, early diastolic peak mitral flow velocity; Am, late diastolic peak mitral flow velocity; DTm, deceleration time of mitral flow; DTm, heart-rate-corrected deceleration time of mitral flow; DTm_c, heart-rate-corrected DTm

Intraobserver and interobserver variabilities

Intraobserver and interobserver reproducibilities were assessed for the RV-MPI in 40 randomly selected subjects (20 athletes, 20 controls). Agreement between the two observers was verified using the Bland–Altman method.[17] The variability was determined as a mean percent error, derived as the absolute difference between two observations divided by the mean of the two observations, and expressed as a percentage.

Results

The two study groups were similar with regard to age, weight, height, and body surface area. The $\dot{V}O_{2max}$ was significantly higher in the athlete group than in the control group (43.39 ± 3.26 vs 64.54 ± 3.84, $P < 0.001$) (Table 1).

Left ventricular mass and mass index, fractional shortening, and stroke volume were higher in the athletes than in controls. Transmitral flow velocity parameters, i.e., Em wave, the Em/Am ratio, and DTm, were higher in the athletes than in the controls (Table 2).

Right ventricular measurements are summarized in Tables 3 and 4. The RV diameters of athletes were larger than those of controls. However, these cavity measurements were higher in the endurance-trained athletes compared with the strength-trained athletes. Transtricuspid flow velocity in the athletes showed greater values in Et, Et/At, and DTt than those of controls.

Table 3. Measurements of the right ventricle in athletes and controls

	Athletes (n = 52)	Controls (n = 43)	P value
RVd_1 (cm)	2.9 ± 0.2	2.0 ± 0.2	<0.001
RVd_2 (cm)	2.4 ± 0.2	1.5 ± 0.2	<0.001
RVd_3 (cm)	5.0 ± 0.2	4.1 ± 0.2	<0.001
Et (cm/s)	73.8 ± 13.1	56.4 ± 10.3	<0.001
At (cm/s)	41.1 ± 7.4	38.1 ± 8.3	0.07
DTt (ms)	333 ± 12	289 ± 15	<0.001
DTt_c (ms)	315 ± 26	324 ± 20	0.09
Et/At	1.8 ± 0.3	1.5 ± 0.4	<0.001
a (ms)	375 ± 35	425 ± 35	<0.001
b (ms)	284 ± 26	299 ± 20	<0.01
RV-MPI	0.32 ± 0.01	0.43 ± 0.02	<0.001

Variables are expressed as mean ± SD
RVd, right ventricular cavity diameter; Et, early diastolic peak tricuspid flow velocity; At, late diastolic peak tricuspid flow velocity; DTt, deceleration time of tricuspid flow; DTt_c, heart-rate-corrected DTt; a, duration from the cessation to the onset of tricuspid inflow; b, duration of the RV outflow velocity profile; RV-MPI, myocardial performance index of right ventricle

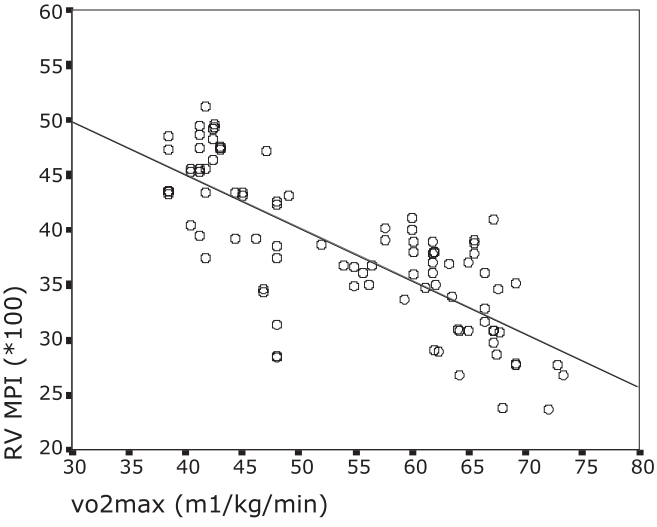

Fig. 2. Relation between the right ventricular performance index (*RV MPI*) and maximal oxygen consumption (*VO2max*) (coefficient = −0.61, $P < 0.01$)

The RV-MPI of the athletes was significantly lower than that of the controls (0.32 ± 0.01 vs 0.43 ± 0.02, respectively; $P < 0.001$) (Tables 3 and 4). However, RV-MPI was lower in the endurance-trained athletes than in the controls. Furthermore, there was a negative correlation between RV-MPI and $\dot{V}O_{2max}$ ($r = -0.61$, $P < 0.01$) (Fig. 2).

Reproducibility

The absolute difference for the same observer was 0.02 ± 0.04 for RV-MPI. The absolute difference between

Table 4. Comparison of right ventricular measurements in control subjects and endurance- and strength-trained athletes

	Endurance-trained athletes (n = 25)	Strength-trained athletes (n = 27)	Controls (n = 43)	F value	P value
RVd_1 (cm)	3.08 ± 0.04	2.80 ± 0.03	1.96 ± 0.03	26.73	<0.001
RVd_2 (cm)	2.58 ± 0.04	2.30 ± 0.03	1.46 ± 0.03	27.69	<0.001
RVd_3 (cm)	5.19 ± 0.03	4.86 ± 0.03	4.07 ± 0.02	32.48	<0.001
Et (cm/s)	72 ± 2	75 ± 2	56 ± 2	25.37	<0.001
At (cm/s)	42 ± 2	41 ± 2	38 ± 1	1.72	0.18
Et/At	1.79 ± 0.07	1.85 ± 0.06	1.53 ± 0.05	8.30	<0.001
DTt (ms)	332 ± 3	335 ± 3	289 ± 2	29.26	<0.001
DTt_c (ms)	313 ± 5	317 ± 4	324 ± 4	2.10	0.12
RV-MPI	0.28 ± 0.01	0.36 ± 0.01	0.43 ± 0.02	50.42	<0.001

Variables are expressed as mean ± SE

RVd, right ventricular cavity diameter; Et, early diastolic peak tricuspid flow velocity; At, late diastolic peak tricuspid flow velocity; DTt, deceleration time; DTt_c, heart-rate-corrected DTt; RV-MPI, myocardial performance index of right ventricle

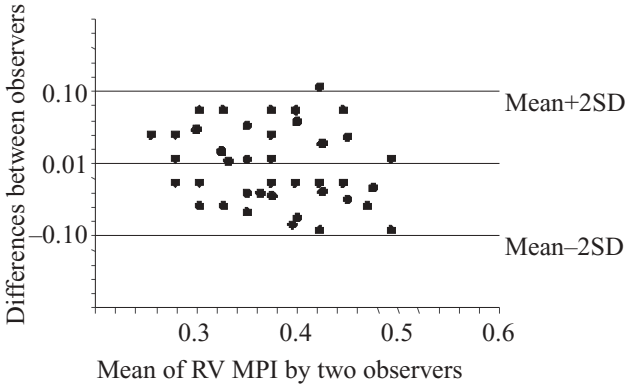

Fig. 3. Interobserver variabilities of myocardial performance index of the right ventricle (*RV MPI*) measurements in echograms of 40 randomly selected subject, as assessed by the Bland–Altman method

observers was 0.03 ± 0.05 for RV-MPI (Fig. 3). Intraobserver and inter-observer variability (expressed as the absolute difference divided by the mean of measurements) was 1% ± 2% and 2% ± 3%, respectively, for RV-MPI.

Discussion

Morganroth et al.[18] were the first to postulate that two different morphological forms of athlete's heart can be distinguished: a strength-trained and an endurance-trained heart. Recent studies on this topic have confirmed how endurance- and strength-training may determine either extreme volume or pressure load, thus explaining the athlete's increase in LV internal dimension and/or wall thickness.[19,20] However, RV adaptation in the athlete has not been explained precisely due to the difficulty of obtaining readily standardizable views.

Right ventricular dimensions in athletes

The findings of this study suggests that RV enlargement occurs in athletes. Henriksen et al.[4] also reported similar results in endurance athletes. However, the RV diameters in strength-trained athletes were lower than those of endurance athletes. Right ventricular adaptation to intense and prolonged physical exercise may be expected to increase the RV cavity dimensions, with a concomitant increase in RV contractile reserve.[5] Interestingly, Douglas et al.[21] found that RV dilatation was developed while the LV became smaller during extreme exercise in elite athletes. This finding suggests that the disproportionate increases in RV and LV afterload during exercise could be the cause of the differences in ventricular size.

Previous studies showed that pulmonary vascular resistance is reduced less than systemic resistance.[22–24] The relative increase in pulmonary artery pressure is greater than the increase in systemic arterial pressure. These changes suggest that the relative increase in cardiac load imposed on the right ventricle is greater than that in the left ventricle during exercise.[22–24] It has been reported that pulmonary artery pressure was increased by an average of 60% while systemic pressure was increased by 11% to 45% after exercise.[22–24]

Furthermore, we also found that RV cavity diameters in endurance-training athletes were higher than in strength-training athletes. We speculated that volume overload exercise training (isotonic type) causes a greater increase in the diameter of the RV cavity compared with volume overload exercise training (isometric type). Hammermeister and Morrisson[25] also explained that RV dilatation could be compensated as a result of increasing venous return during exercise.

RV-MPI and $\dot{V}O_{2max}$ in athletes

To the best of our knowledge, this is the first study that shows a relationship between RV-MPI and $\dot{V}O_{2max}$ in athletes. The results of the present study emphasize the importance of a better functional RV in the maintenance of

endurance capacity. It has been reported that RV work was increased 3.6- to 5.2-fold, while LV work increased only 2.1- to 2.8-fold over baseline during the same exercise load.[22–24] The greater increase in hemodynamic load imposed on the RV may explain that although there are close interactions (morphologic and functional) for each ventricle, the response of the RV is somewhat different to that of the LV.

Other relaxation parameters, i.e., Et, At, Et/At, and DTt, are measured using transtricuspid flow, but these diastolic parameters are affected by many factors, such as age, gender, respiration stage, and heart rate. However, RV-MPI has been reported to be independent of heart rate and blood pressure.[6,14] This method is a noninvasive, easily calculated, and reproducible method for evaluating combined systolic and diastolic function by simple cardiac time interval analysis with Doppler echocardiography.[6,14] The MPI is calculated by isovolumic contraction time, which is important in the assessment of systolic function, and by isovolumic relaxation time, which is used to assess diastolic function.[26,27] Moreover, we previously reported that MPI of LV was lower in endurance athletes than in controls,[28] which suggested that increased global LV function in athletes supports a better exercise capacity compared with sedentary subjects. Furthermore, Erol and Karakelleoglu[29] found that RV-MPI was similar in athletes and controls. They explained that global RV function does not deteriorate in the athlete's heart despite significant chamber dilatation.

In the present study, we found that there was a significant negative correlation between RV-MPI and maximal aerobic capacity. An increase in LV function was a prerequisite for the increase in $\dot{V}O_{2max}$ resulting from training. To explain these data, one could hypothesize that better RV function in athletes may contribute to enhanced endurance capacity via increased LV filling and performance.[30]

Previous studies have shown that patients with RV pressure or volume overload often have affected LV function that is related to the geometric distortion of the left ventricle.[31,32] Since both ventricles share a common ventricular septum and pericardium, alterations in RV function may influence left ventricular function. Substantial experimental evidence suggests that RV hemodynamics influence LV pressure–volume relationships.[30] These phenomena are compatible with known structural and functional interrelationships of the two ventricular chambers, such that changes in RV volume and pressure may result in altered transmural stresses across the interventricular septum, thus modifying the LV pressure–volume relationship.[31,32]

Right ventricular performance and heart rate

In addition, our study suggests that the maintenance of cardiac performance at a lower heart rate in athletes may lead to a better force–frequency relationship in the myocardium, related to a restoration of cardiac performance. There is little information on the influence of variation in the resting heart rate within the normal range on RV function. It has been established previously that Doppler-derived transtricuspid flow velocity is affected by heart rate. During tachycardia the peak velocity of the A wave and right atrial filling fraction are reduced, without any effect on peak velocity of the E wave.[33] The available evidence suggests that the RV adapts to chronic endurance training in a manner analogous to that of the LV.[34] A slow heart rate may allow greater ventricular filling and a subsequent increase in ventricular ejection fraction through the Frank–Starling effect.[35] It is most likely that the long-term reduction in heart rate and associated changes in diastolic function and myocardial metabolism are of major importance for myocardial recovery.[35]

Limitations of the study

The estimation of morphologic and functional parameters of RV by two-dimensional echocardiography is difficult. The entire RV cannot be visualized because of the inaccessible intrathoracic position and irregular geometric shape. Tracing techniques also cause errors because of developing noise from the RV endocardial surface. Therefore, measurement and functional assessment of the RV are much more difficult compared with the LV. Furthermore, unfortunately, echocardiographic measurements were not repeated at peak exercise due to technical difficulties. We believe that this would be a logical step if one is to correlate $\dot{V}O_{2max}$ to indices of RV function.

Conclusions

Increasing RV function may support the development of greatly increased cardiac output during exercise. The right ventricle, as well as the left, must alter its performance to meet the increased circulatory demands of exercise. We can conclude that in athletes, regular and intensive exercise training leads to RV dilatation and decreased RV-MPI (supra-normal RV systolic and diastolic function) with increased $\dot{V}O_{2max}$. RV-MPI may provide a simple and noninvasive measure of right ventricular global function and exercise capacity in athletes.

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