Cardiac structure and function in cyclists and runners

Comparative echocardiographic study

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SUMMARY Twelve cyclists and 12 long distance runners matched for age, height, and weight with two control groups of 12 non-athletes were studied echocardiographically to evaluate cardiac structure and function. Runners weighed 8 kg less than cyclists, but age and height were similar. Peak oxygen uptake per kg body weight was higher in athletes than in the control subjects but was similar in the cyclists and in the runners. The athletes' hearts had a larger end diastolic left ventricular internal diameter, mean wall thickness, and cross sectional area of the left ventricular wall than those of the respective control subjects. Nevertheless, whereas the left ventricular internal diameter was not different between the cyclists and runners, mean wall thickness and cross sectional area of the left ventricular wall were greater in the cyclists even after adjustment for weight. The ratio of wall thickness to left ventricular internal radius was significantly larger in cyclists than in their control group, but the ratio was similar in runners and their control group. The echocardiographic indices of left ventricular function were similar in the athletes and the control groups. Systolic left ventricular meridional wall stress was lower in the cyclists than in the runners.

The data suggest that runners develop an increase in left ventricular wall thickness which is proportionate to the internal diameter but that in cyclists the increase is disproportionate because of the isometric work of the upper part of the body during cycling.

Long distance cycling and running are two predominantly isotonic endurance sports. Cycling, however, comprises isometric work of the upper part of the body. Ultrasound imaging of the heart of athletes has shown that isotonic and isometric training lead to different adaptations of cardiac structure—that is, mainly left ventricular eccentric hypertrophy in the former¹⁻¹¹ and concentric hypertrophy in the latter.¹⁷⁻¹¹ Whether these adaptations of the heart also differ between long distance cyclists and runners has not often been studied. Only Snoeckx *et al* studied both types of athletes and found that calculated muscle mass was significantly greater in the cyclists.¹¹ Furthermore, data on both groups of athletes suggest that the structural adaptations of the heart are more

pronounced in cyclists¹²⁻¹⁴ than in runners.¹⁻¹⁰ Cyclists, however, generally weigh more than runners, which could affect differences in cardiac structure between the two types of athletes.

In the present study we assessed echocardiographically the cardiac structure and function of long distance cyclists and runners and of two groups of non-athletic control subjects matched for age, weight, and height for either the cyclists or the runners.

Subjects and methods

Twelve male cyclists (aged 18-35 years) and 12 male runners (aged 19-40) were studied. The cyclists had been involved in competitive cycling for a mean of 8.0 (range 4-11) years and the runners for 6.6 (range 2-14) years. As indicated by a questionnaire the cyclists covered between 10 000 and 47 000 km a year during training and competition and the runners distances between 3300 and 8000 km. All athletes were

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studied during periods of active training. Twelve male non-athletes were matched for age, height, and weight with the runners and another 12 with the cyclists; they were not involved in competitive sports, but moderate recreational sports were not a criterion for exclusion.

ELECTROCARDIOGRAPHY

Each subject was studied in the morning. A standard 12 lead resting electrocardiogram was recorded on an Elema-Schönander Mingograf 34 three channel electrocardiograph at a paper speed of 25 mm/s. QRS voltages were measured and the magnitude and direction of the mean electrical axis (frontal plane) calculated.¹⁵

ECHOCARDIOGRAPHY

Echocardiography was performed, always by the same investigator and with the subject rotated slightly on his left side, using a 2.25 MHz transducer and a commercially available Irex system II ultrasonic unit with photographic paper. Measurements were recorded at end expiration. The transducer was angulated to ensure simultaneous visualisation of the left ventricular posterior wall and the interventricular septum just below the mitral valve apparatus—that is, at the level of the posterior chordae tendineae. The echocardiograms were processed using a digitiser and computer program.¹⁶ The echocardiographic measurements were analysed on each of three successive heart beats, and the values were then averaged. Both left ventricular dimensions and dimensional changes were calculated. Wall thickness and cavity dimensions were measured using the leading edge method (from the most anterior edge of the endocardial and epicardial lines). The following echocardiographic measurements were obtained: (a) left ventricular end diastolic total and internal diameter (LVTd and LVIDd), measured at the onset of the QRS complex, and left ventricular systolic internal diameter (LVIDs) taken as the minimal left ventricular dimen-

sion; (b) posterior wall and interventricular septal thickness at the onset of QRS (PWTd and IVSTd) and the maximal thickness during systole (PWTs and IVSTs); and (c) peak velocities of the LVID change and of the posterior wall endocardial displacement during systole and during relaxation. The percentage shortening of the LVID was calculated as $((\text{LVIDd} - \text{LVIDs})/\text{LVIDd}) \times 100$, mean wall thickness at end diastole (MWTd) as $(\text{IVSTd} + \text{PWTd})/2$, and mean wall thickness during systole (MWTs) as $(\text{IVSTs} + \text{PWTs})/2$. The cross sectional area (CSA, cm^2) of the left ventricular wall in a transverse plane was determined from the echocardiographic left ventricular internal dimension and wall thickness at end diastole as $\pi(\text{LVIDd}/2 + \text{MWTd})^2 - \pi(\text{LVIDd}/2)^2$. This index of left ventricular myocardial mass does not entail dubious assumptions regarding ventricular geometry.¹⁷ The ratio of wall thickness to internal radius (h:R) was calculated as $\text{MWTd}/(\text{LVIDd}/2)$. Systolic left ventricular meridional wall stress (mm Hg), a quantitative index of myocardial afterload, was calculated as $0.334 \times P \times \text{LVIDs}/\text{MWTs} \times (1 + \text{MWTs}/\text{LVIDs})$, where P is the systolic blood pressure.^{18 19}

BICYCLE ERGOMETRY

Each subject underwent a graded uninterrupted exercise test on a bicycle ergometer (Siemens 380 B) until exhaustion. The initial external workload was 20 W and was increased by 30 W every four minutes. Pulmonary ventilation ($\dot{V}_E$) was continuously measured by a pneumotachograph, and oxygen uptake ($\dot{V}O_2$) was determined from the measurement of oxygen in the expired air (Siemens FD 84). Heart rate was calculated from the electrocardiogram.

DATA ANALYSIS

All data were analysed at the end of data collection. The echocardiograms were interpreted blind in a ran-

Table 1 Comparison of two values of left ventricular (LV) function and structure from 19 echocardiograms analysed blind by one observer. Values are means (SEM)

	First value	Second value	% difference
LV internal diameter			
Diastole	51.5 (1.0)	51.2 (0.9)	1.2 (0.2)
Systole	33.3 (0.7)	33.4 (0.7)	0.9 (0.2)
Interventricular septal thickness			
Diastole	10.3 (0.4)	10.2 (0.4)	7.0 (1.7)
Systole	14.8 (0.5)	14.4 (0.4)	4.5 (1.6)
Posterior wall thickness			
Diastole	10.7 (0.4)	10.7 (0.4)	2.3 (0.5)
Systole	17.6 (0.5)	17.0 (0.5)	4.8 (1.3)
Peak velocity of internal diameter change			
Systole	180 (7.1)	179 (7.4)	11.7 (2.2)
Relaxation	114 (4.3)	110 (4.7)	11.0 (2.0)
Peak velocity of posterior wall displacement			
Systole	77 (3.5)	75 (4.0)	8.3 (1.8)
Relaxation	164 (7.0)	157 (6.5)	12.5 (1.8)

Table 2 Clinical, exercise testing, and electrocardiographic data in 12 cyclists (group 1), 12 matched controls (group 2), 12 runners (group 3), and 12 matched controls (group 4). Values are means (SEM)

	Group 1	Group 2	Group 3	Group 4	Intergroup comparison (p)*		
					1 v 2	3 v 4	1 v 3
Clinical data							
Age (y)	23.3 (1.3)	24.5 (1.6)	26.3 (1.7)	26.5 (2.2)	NS	NS	NS
Height (m)	1.79 (0.01)	1.79 (0.02)	1.75 (0.02)	1.78 (0.02)	NS	NS	NS
Weight (kg)	71.3 (1.5)	71.2 (1.9)	63.1 (1.7)	65.4 (1.5)	NS	NS	<0.05
Resting systolic blood pressure (mm Hg)	123 (2)	123 (3)	129 (3)	122 (2)	NS	NS	NS
Exercise data							
Peak oxygen uptake (l/min)	4.57 (0.12)	3.13 (0.15)	3.80 (0.18)	3.19 (0.11)	<0.001	<0.05	<0.01
Peak oxygen uptake (ml/min/kg)	64.1 (1.5)	44.0 (1.7)	60.0 (1.6)	48.9 (1.7)	<0.001	<0.001	NS
Heart rate sitting (beats/min)	61 (3)	73 (4)	59 (2)	78 (1)	<0.05	<0.001	NS
Heart rate at 140 W (beats/min)	104 (3)	148 (5)	116 (5)	147 (4)	<0.001	<0.001	NS
Peak heart rate (beats/min)	183 (4)	194 (3)	178 (3)	193 (4)	NS	<0.05	NS
Electrocardiographic data							
R wave (lead V5) (mV)	2.33 (0.38)	1.21 (0.10)	2.13 (0.23)	1.19 (0.15)	<0.05	<0.05	NS
R wave (lead V6) (mV)	1.54 (0.29)	0.80 (0.05)	1.44 (0.25)	0.79 (0.09)	NS	NS	NS
Mean QRS axis (°)	55.3 (6.1)	56.8 (4.9)	62.8 (4.2)	62.4 (5.5)	NS	NS	NS
Mean QRS voltage (mV)	1.49 (0.13)	1.44 (0.13)	1.62 (0.15)	1.19 (0.15)	NS	NS	NS

*None of the variables is significantly different between group 2 and group 4.

dom order by one observer. Nineteen echocardiograms from the present study were analysed by the same observer on another occasion. Table 1 shows the means of the two values and the percentage difference calculated as: ((highest value minus lowest value)/mean of the two values) $\times$ 100.

For intergroup comparisons of data one way analysis of variance was applied using a Scheffé test for the comparison of means. Furthermore, when appropriate, analysis of covariance with weight as the covariate was used for intergroup comparison; adjusted means were compared with a Scheffé test when weight was found to be a significant covariate.²⁰

Results

CHARACTERISTICS OF THE SUBJECTS

Cyclists, runners, and the control groups of the cyclists and of the runners did not differ in age and height (Table 2). Weight was similar in the athletes and their matched controls but was greater in the cyclists than in the runners ($p<0.05$). Peak oxygen uptake, adjusted for body weight, was higher in the athletes than in their matched controls ($p<0.05$) but was similar in cyclists and runners and in the two control groups (Table 2). Resting and submaximal heart rates at 140 W were lower in the athletes than in the controls ($p<0.05$) but did not differ between the two groups of athletes and between the control groups. Peak heart rate did not differ between the groups except that the runners had a significantly lower heart rate than their control group ($p<0.05$) (Table 2).

ELECTROCARDIOGRAPHIC DATA

A comparison of voltage criteria in various leads (R

waves in peripheral leads and V5–6 and S waves in V1–2) showed a higher R wave in lead V5 in the two groups of athletes than in the controls ($p<0.05$) (Table 2). Other differences were not significant. Also the mean QRS axis and the mean QRS voltages were similar in the four groups.

ECHOCARDIOGRAPHIC DATA

Cardiac structure

Table 3 summarises the echocardiographic data on cardiac structure. LVTd, LVIDd, IVSTd, PWTd, and MWTd were higher ($p<0.05$) in cyclists than in their control group and in runners than in their control group, except that PWTd was not different between the two latter groups. The ratio of IVSTd to PWTd was close to 1 and not different between the four groups. The ratio of mean wall thickness to the left ventricular internal radius at end diastole (h:R) was higher in the cyclists than in their controls ($p<0.05$) but did not differ between the runners and their controls (Fig. 1). The cross sectional area of the left ventricular wall (CSA) was greater ($p<0.001$) in both groups of athletes than in the controls. In cyclists, LVIDs, IVSTs, and PWTs were greater ($p<0.05$) than in their controls, but only IVSTs was significantly different between the runners and their controls.

None of the variables differed between the two control groups. When cyclists were compared with runners, however, some of the dimensions were larger in the cyclists—namely, LVTd, PWTd, MWTd, PWTs, and CSA. In the analysis of covariance, weight was a significant covariant for LVTd ($p<0.01$) and for CSA ($p<0.05$) but not for the other echocardiographic variables. After adjustment for weight the dif-

Table 3 Echocardiographic data on cardiac structure and function in 12 cyclists (group 1), 12 matched controls (group 2), 12 runners (group 3), and 12 matched controls (group 4). Values are means (SEM)

	Group 1	Group 2	Group 3	Group 4	Intergroup comparison (p)*		
					1 v 2	3 v 4	1 v 3
Cardiac structure							
Heart rate (beats/min)	52 (2)	63 (3)	51 (3)	62 (2)	<0.05	<0.05	NS
During diastole:							
LV diameter (mm)							
Total (LVTd)	80.0 (1.2)	68.6 (0.9)	74.0 (1.2)	66.4 (0.9)	<0.001	<0.001	<0.01
Internal (LVID)	55.2 (1.0)	49.5 (0.8)	52.5 (1.0)	48.5 (0.7)	<0.01	<0.05	NS
IVS thickness (IVSTd) (mm)	12.1 (0.5)	9.3 (0.2)	10.9 (0.5)	8.5 (0.3)	<0.001	<0.01	NS
Posterior wall thickness (PWTd) (mm)	12.7 (0.5)	9.8 (0.4)	10.5 (0.4)	9.3 (0.5)	<0.001	NS	<0.05
Mean wall thickness (MWTd)	12.4 (0.3)	9.6 (0.2)	10.7 (0.4)	8.9 (0.4)	<0.001	<0.01	<0.01
IVSTd/PWTd	0.97 (0.06)	0.97 (0.05)	1.04 (0.06)	0.94 (0.05)	NS	NS	NS
Wall thickness: internal radius ratio	0.45 (0.01)	0.39 (0.01)	0.41 (0.02)	0.37 (0.02)	<0.05	NS	NS
Cross sectional area (cm ²)	26.45 (0.91)	17.75 (0.49)	21.35 (0.89)	16.12 (0.76)	<0.001	<0.001	<0.001
During systole:							
LVIDs (mm)	36.2 (1.2)	32.2 (0.8)	36.0 (0.9)	33.2 (0.8)	<0.05	NS	NS
IVSTs (mm)	16.3 (0.6)	13.8 (0.4)	14.7 (0.7)	12.4 (0.4)	<0.05	<0.05	NS
PWTs (mm)	19.8 (0.5)	15.8 (0.4)	15.7 (0.5)	15.0 (0.7)	<0.001	NS	<0.001
Cardiac function							
During systole:							
% Shortening of LVID (%)	34.5 (1.5)	35.0 (1.2)	31.3 (1.7)	31.6 (1.2)	NS	NS	NS
Peak velocity of:							
LVID (mm/s)	115 (6)	105 (5)	106 (8)	98 (7)	NS	NS	NS
LVPW (mm/s)	79 (3)	72 (6)	67 (3)	62 (3)	NS	NS	NS
During relaxation:							
Peak velocity of:							
LVID (mm/s)	189 (8)	173 (11)	158 (17)	157 (9)	NS	NS	NS
LVPW (mm/s)	165 (5)	150 (9)	131 (15)	126 (7)	NS	NS	NS
Systolic LV meridional wall stress (mm Hg)	55.4 (3.0)	61.6 (2.6)	73.4 (4.6)	71.7 (5.4)	NS	NS	<0.05

*None of the variables is significantly different between groups 2 and 4.

ference between cyclists and runners was no longer significant for LVTd ($p=0.07$) but persisted for CSA ($p<0.01$) (Fig. 2).

Cardiac function

The various indices of myocardial function during systole or during relaxation or both—that is, the percentage shortening of LVID and the peak velocities of change in LVID and of PW endocardial movement—did not differ between the groups. Systolic left ventricular meridional wall stress was not different between the athletes and control subjects but was lower ($p<0.05$) in the cyclists than in the runners (Table 3).

Discussion

The cardiac adaptations of endurance trained athletes have been well described since ultrasound cardiac imaging has become available.¹⁻¹⁴ For the left ventricle, both an increase in left ventricular internal diameter and thickening of the muscular wall have been observed. It is assumed that the increase of wall thickness is proportionate to the dilatation of the left ventricle, but the ratio of wall thickness to internal radius is not often reported. Furthermore, there may be subtle differences in the cardiac adaptation to dif-

ferent types of endurance training. Snoeckx *et al*, for example, found a significantly greater calculated muscle mass in cyclists than in runners.¹¹ From the reports on cyclists¹¹⁻¹⁴ and on runners,¹⁻¹¹ it appears that the left ventricular internal diameter is always enlarged, except in one study.² Posterior wall thickness on the other hand was increased in all studies of cyclists, except in a study from Japan,¹² but this variable did not differ between the runners and non-athletes in several reports.^{1,3,7}

We, therefore, undertook a comparative study of the cardiac adaptations in cyclists and runners. For such a comparison to be valid the aerobic capacity of both groups of athletes should be similar. In the present study no attempt was made to determine true maximal oxygen uptake, but peak oxygen uptake for a prolonged uninterrupted exercise test on the bicycle was similar in both groups of athletes, at least when oxygen uptake was adjusted for body weight. The slightly, although not significantly, lower peak $\dot{V}O_2$ per kg of the runners in contrast to that of the cyclists can be attributed to the fact that the runners were unused to this form of testing.²¹ Also the similar heart rate at the submaximal work rate of—for example, 140 W—suggests a comparable exercise capacity for both groups of athletes.

Fig. 1 Ratio of mean wall thickness to internal radius in cyclists, runners, and two control groups for cyclists and runners. Values are means (SEM). Values inside each set of horizontal brackets are not significantly different but differ ($p < 0.05$) from values outside the bracket.

Cyclists weigh more than runners. The data for each group of athletes were, therefore, compared with those for non-athletes matched for age, height, and weight. The importance of adequate control groups is emphasised by the observation that several measurements, which differed significantly between runners and their matched controls, did not differ between runners and the somewhat heavier cyclist control group; this was true for LVIDd, IVSTd, MWTd, and IVSTs. Furthermore, for intergroup comparisons data were adjusted for weight when it was a significant covariate.

The present study confirms that left ventricular hypertrophy develops in cyclists and in runners. Indeed, electrocardiographic voltage criteria, at least in lead V5, and the cross sectional area of the left ventricular wall were larger in both groups of athletes than in the respective control groups. This area is an index of left ventricular myocardial mass, and its use is preferred because it does not entail dubious assumptions regarding ventricular geometry as in the calculation of myocardial mass from the M mode echocardiogram. Moreover, in agreement with another study,¹¹ we found that the cross sectional area was larger in the cyclists than in the runners. Snoeckx *et al* attributed the difference to the more intense training of the cyclists.¹¹ In the present study peak oxygen uptake was similar in both groups of athletes, but differences in training intensity can, of course,

Fig. 2 Left ventricular total dimension during diastole and cross sectional wall area at end diastole in cyclists, runners, controls for cyclists, and controls for runners. Values are means (SEM) adjusted for weight. Values inside each set of brackets are not significantly different but differ ($p < 0.05$) from values outside the bracket.

not be excluded, even supposing that training regimens of cyclists and runners can be compared. In addition, the larger weight of the cyclists must be considered, but the difference in wall cross sectional area persisted after adjustment for weight. A third possibility is that cyclists and runners develop a different type of left ventricular hypertrophy.

Eccentric left ventricular hypertrophy is characterised by an increase in the internal diameter of the left ventricle with a proportionate increase in wall thickness; this is attributed to volume overload. Concentric hypertrophy, on the other hand, does not produce changes in the internal diameter, but left ventricular wall thickness is increased as a result of pressure overload.¹⁸ The runners clearly develop eccentric hypertrophy as evidenced by the increased left ventricular internal diameter with an unchanged ratio of wall thickness to internal radius compared with the matched control subjects. This is compatible with the repeated volume overloading of predominantly isotonic endurance training. The cyclist's heart, however, is characterised by an increased internal diameter but a disproportionate increase in wall thickness. This mixed eccentric-concentric type of hypertrophy may be the result of both volume and pressure overload in the cyclists as a result of a combination of mainly isotonic exercise with isometric work of the arms and the upper part of the body. Athletes who are only strength trained do indeed develop left ventricular concentric hypertrophy.¹⁹⁻²² Five of the cyclists did engage in mild strength training, mainly in the winter rest period, but it is unlikely that this affected our results since the ratio of left ventricular wall thickness to the internal radius was identical in these cyclists and in those who did not include weight lifting in their training programme.

The disproportionate increase in wall thickness in

the cyclists led to a smaller systolic left ventricular meridional wall stress in these athletes than in the runners. Sugishita *et al* argued that the hearts of athletes participating in isometric sports activities have hypertrophied in response to the high blood pressure resulting from the isometric exercise.⁹ At rest, however, when blood pressure is within normal limits, wall stress becomes abnormally low. It is possible that this mechanism operates to some extent in cyclists. This would, however, require a higher blood pressure in cycling cyclists than in running runners, but such a comparison is, to the best of our knowledge, not available.

The various echocardiographic indices of myocardial function showed that during systole the fractional shortening of the left ventricular internal diameter and the peak velocities of dimension change and of posterior wall endocardium displacement did not differ between the athletes and the control subjects nor between the cyclists and the runners. Furthermore, during left ventricular relaxation the peak velocities were similar in the four groups. Most studies of cyclists and runners show that the indices of left ventricular function are normal, although some report a slightly depressed,^{2,9} and others a somewhat enhanced, left ventricular function.^{5,6}

In conclusion, the cardiac function of both cyclists and runners, studied during their active season, is characterised by left ventricular dilatation and an increase in wall thickness. Whereas the increase of wall thickness in relation to the internal dimension appears to be proportionate in runners, it is disproportionate in cyclists, probably because of the isometric work component during cycling. Even after adjustment for weight, muscle mass is larger in cyclists than in runners despite a comparable aerobic capacity. Left ventricular function is similar in athletes and non-athletes.

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