

# IMPACT OF DIFFERENT SPORTS AND TRAINING ON CARDIAC STRUCTURE AND FUNCTION

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There is no doubt that the heart can adapt to an increased hemodynamic load. In disease states, it is generally recognized that two types of chronic overload can effect the heart in different ways.<sup>47</sup> A volume load, as in aortic or mitral insufficiency, leads to an enlargement of the left ventricular internal diameter and a proportional increase of wall thickness. This type of adaptation is called *eccentric left ventricular hypertrophy*. A pressure load, as in aortic stenosis or hypertension, is associated with a thickening of the ventricular wall and an unchanged internal dimension, or *concentric left ventricular hypertrophy*. It was suggested that these cardiac adjustments serve to normalize systolic wall stress.<sup>47</sup>

Physical activity is associated with hemodynamic changes and alters the loading conditions of the heart.<sup>68</sup> In dynamic exercise, the emphasis is on movement with no or minimal development of force. The main hemodynamic features are increases in heart rate and stroke volume, the two components of cardiac output. Systemic vascular resistance drops, but the net result is a slight to moderate rise in blood pressure. The load on the heart is predominantly of the volume type. In static exercise, force is developed with no or minimal movement. The hemodynamic consequences involve a slight elevation of cardiac output, owing to the increase of heart rate,

and a more pronounced rise of blood pressure, resulting in a pressure load on the heart. From a theoretical point of view, the hemodynamic alterations and the ventricular loading conditions during exercise could in the long run lead to left ventricular hypertrophy. Although the load on the heart is continuous in pathologic conditions, however, it is not in the athlete.

Several questions therefore arise: (1) Does the heart of athletes differ from that of non-athletes, and are cardiac adaptations related to the type of physical exercise? (2) Are structural differences associated with alterations in cardiac function? (3) Are differences between athletes and nonathletes due to training per se, to other factors, or a combination of training and other factors?

## CARDIAC STRUCTURE IN ATHLETES

As reviewed by Rost,<sup>92, 93</sup> the first indication of an enlarged heart in athletes was given by Henschen at the end of the nineteenth century through carefully performed percussion of the chest in cross-country skiers. Cardiac enlargement was later confirmed by use of radiography and by evidence from autopsy. The advent of echocardiography allowed clini-

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cians to gain a better insight into the heart of the living athlete. Wall thicknesses and internal dimensions could be studied in detail, and estimations of left ventricular mass became possible. There is now overwhelming evidence that the heart of athletes may differ from that of sedentary subjects.

To test the hypothesis that exercise-related volume and pressure loads are associated with different cardiac adaptations, one should ideally dispose of athletes engaged in purely dynamic or static sports. In addition, the load on the heart should be of sufficient duration and intensity. Although athletic conditioning is rarely purely dynamic or static and the training programs of different athletes may overlap, long distance running comes close to an ideal example of dynamic exercise. Other predominantly dynamic aerobic or endurance sports are swimming, rowing, and cycling. The dynamic activity can be of short duration but high intensity, calling on anaerobic metabolism, as is the case in sprinters. Several sports are categorized as predominantly static or involve power training, such as weight lifting, body building, wrestling, and throwing events such as shot putting. The training regimens of these athletes are, however, not uniform. The power training can be static but is sometimes described as dynamic involving only light-to-moderate weights. The actual duration of the static activity and therefore of the pressure load on the heart may be brief. Some of these athletes also engage in dynamic activities such as running. Finally, the activities involved in most ball sports are combinations of several types of exercise.

Numerous studies have applied echocardiography in athletes engaged in these various sports and compared the results to those of nonathletes. An important question arises: Who should be in the control group? Ideally, these subjects should be normally active and matched for age, gender, and anthropometric characteristics. Particularly the last aspect poses a problem. Even if athletes and nonathletes are matched for height and weight, body composition may be different. Athletes usually have less fat and a higher lean body mass. The author considers that matching for height and weight is adequate because small differences in lean body mass did not reach statistical significance in studies that applied this matching procedure and estimated lean body mass.<sup>31, 50, 59, 66</sup> There is no evidence that the fat mass does not affect the heart because

obesity per se is associated with left ventricular hypertrophy. In several studies, however, athletes and control subjects were not adequately matched for body size. These authors simply divided the echocardiographic variables by an index of body size, usually body surface area. This procedure assumes that the ratio between the studied variable and the index of body size is a constant and independent of body size, which has not been proved.

This article is restricted to studies in which athletes and nonathletes were matched for anthropometric characteristics or in which body size was similar in the two groups. Because many reports mention only body surface area, or the latter can be calculated from reported data on height and weight, the selection of studies was based on this index, and a maximal deviation of 5% was allowed. Furthermore, the average age of the groups could not differ by more than 5 years, the subjects had to be of the same gender, and the athletes of any group had to perform the same type of sport. In addition, the reported echocardiographic data should include data on left ventricular wall thickness and on internal diameter. Study groups or subgroups of athletes that did not fulfill these selection criteria were not considered for the comparison of cardiac structure of athletes and nonathletes,\* unless in domains in which few data are available. Whenever possible, meta-analytical techniques were applied to compare groups of athletes and matched controls.<sup>35</sup> Advantages of meta-analyses are the increased statistical power and the greater precision in the estimates than in individual studies; disadvantages are the potential heterogeneity of the study populations and differences in methodology in the various reports.<sup>36</sup> Finally, this article is limited to data on the left ventricle, but a larger right ventricular dimension<sup>43, 71, 91, 113, 127, 128</sup> and left atrial size<sup>6, 10, 50, 52, 113, 121, 127, 128</sup> have been reported in athletes.

## **Athletes Engaged in Predominantly Dynamic Aerobic Sports**

### *Long Distance Runners*

Table 1 summarizes the results of the meta-analysis of echocardiographic data of young male long distance runners,<sup>35</sup> which involved

\*References 1, 6, 11, 13, 15, 16, 18, 19, 21, 37-40, 42, 45, 48-50, 52, 64, 65, 69, 71-75, 77, 78, 80, 81, 83, 87, 96, 97, 99, 101, 102, 105, 109, 124, 125.

**Table 1.** RESULTS OF THE META-ANALYSIS OF LONG DISTANCE RUNNERS AND MATCHED CONTROLS\*

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From Fagard RH: Athlete's heart: A meta-analysis of the echocardiographic experience. *Int J Sports Med* 17(suppl 3): S140, 1996; with permission.

135 athletes and 173 control subjects from 10 reports.<sup>19, 28, 43, 52, 59, 69, 79, 105, 113, 128</sup> The runners were competitive athletes, who had trained for several years and ran on average about 100 km/week; aerobic power was approximately 40% higher than in controls. Although athletes and nonathletic controls did not differ in age, body surface area was slightly but significantly lower in the runners, despite the strict selection criteria for inclusion in this analysis; this highlights the difficulty of finding suitable control subjects for runners. Heart rate was 14 beats/minute lower in athletes than in controls, left ventricular internal diameter was 4.8 mm higher, and the septum and the posterior wall were thicker by 1.5 and 1.6 mm. Calculated left ventricular mass was larger by 67 g or 48% ( $P < .001$ ). The increase in mass appeared to be more pronounced in ultraendurance than in amateur long distance runners.<sup>108</sup> The higher left ventricular mass of runners was confirmed by use of nuclear magnetic resonance techniques; the percentage difference of 29% is, however, difficult to interpret because the control subjects were heavier than the athletes.<sup>66</sup>

It is noteworthy that the meta-analysis on runners<sup>35</sup> reveals that relative wall thickness, that is, the ratio between wall thickness and internal diameter,<sup>47</sup> was slightly but significantly higher in the runners than in the controls; the percent difference averaged 7.8% ( $P \leq .05$ ). Therefore, the increase in wall thickness was somewhat more pronounced than expected in the long distance runners, who have generally been considered to develop pure eccentric left ventricular hypertrophy. These findings can most likely be explained

by the fact that training regimens and sports activities are not exclusively dynamic<sup>68</sup> and that the load on the heart is not purely of the volume type. The data thus indicate that male long distance runners have a larger left ventricular mass than nonathletic control subjects because of a larger left ventricular internal dimension and a slightly disproportionate thickening of the left ventricular wall, compatible with predominantly eccentric left ventricular hypertrophy. The term *hypertrophy*, however, should be used with caution because the results of most athletes fall within normal limits.

Few data are available in female long distance runners and matched controls. Both echocardiography<sup>31, 65</sup> and magnetic resonance imaging<sup>69</sup> revealed higher left ventricular mass and internal dimension in athletes than in controls, similar to the results in men. Spirito and colleagues,<sup>106</sup> however, noted that female athletes have smaller left ventricular cavity dimension and smaller wall thickness than males of the same age and body size who were training in the same sport.

### Swimmers

In 1975, Morganroth and associates<sup>69</sup> reported that left ventricular mass was significantly elevated by 45% in male swimmers at the collegiate level who were training for at least 3 years. The left ventricular internal diameter was higher in the athletes; small differences in wall thickness were not significant. The slightly higher left ventricular mass, internal diameter, and wall thickness of adult female swimmers were not significantly different from well-matched controls in another

study.<sup>98</sup> The sparse information on the training regimen of these athletes mentions that they participated in aerobic conditioning for a minimum of 6 months; it is possible that the total training quantity was insufficient to elicit a clear effect on the heart, in agreement with the insignificantly lower resting heart rate. Left ventricular mass and internal diameter, but not posterior wall thickness, were significantly higher in swimmers who participated in competitions for at least 5 years,<sup>19</sup> but these results are more difficult to interpret because of the larger body size of the athletes. Swim training starts at an early age. Medved and colleagues<sup>63</sup> and Özer and associates<sup>76</sup> studied the heart of 7- to 15-year-old swimmers, who had been training for an average of about 2.5 years, and found that both the left ventricular internal diameter and wall thickness were larger than in well-matched controls. The data on school-age and adult swimmers are compatible with the notion that this type of training is associated with eccentric left ventricular hypertrophy.

### Cyclists

Table 2 summarizes the results of the meta-analysis of male cyclists and matched controls.<sup>35</sup> There were 69 athletes and 65 controls from four studies.<sup>28, 32, 67, 79</sup> Age and body surface area were similar in athletes and controls. Heart rate was lower by 16 beats/minute in the cyclists. Left ventricular internal diameter and septal and posterior wall thickness were larger by 4.5, 2.6, and 2.7 mm. The difference in left ventricular mass averaged 102 g or 64% ( $P < .01$ ). The percentage difference in mass was somewhat lower when assessed by nuclear magnetic resonance imaging, 37%<sup>66</sup> and 44%,<sup>86</sup> but it cannot be ascer-

tained whether this is due to the technique or to differences in the selection of the athletes. Also, female cyclists had larger left ventricular mass, internal dimension, and wall thickness than matched sedentary controls.<sup>89</sup> The higher left ventricular mass seems to be restricted to competitive cyclists and was not observed in recreational cycle tourists.<sup>29</sup>

In the meta-analysis of male cyclists, the athletes' relative wall thickness exceeded that of the control subjects by 19% (see Table 2), indicating that cycling is not only associated with an increase of the internal diameter, but also with a disproportionate thickening of the wall. This could be explained by the fact that cycling involves static activity of the upper part of the body. Magnetic resonance imaging revealed that cyclists had slightly, but not significantly, higher left ventricular mass-to-end-diastolic volume ratio, when compared to controls.<sup>86</sup> In other studies on cyclists, the athletes' weight or body surface area was significantly larger than in the control subjects.<sup>6, 72, 105</sup> The study by Nishimura and co-workers<sup>72</sup> is nevertheless of interest because they examined three age groups: 20- to 29-year-old, 30- to 39-year-old, and 40- to 49-year-old cyclists. The left ventricular internal dimension was enlarged in all age groups, but wall thickness was only significantly elevated in the older athletes. Calculated relative wall thickness was 0.33, 0.31, and 0.39. Although the same athletes were not followed longitudinally, this could suggest that wall thickening takes more time or total training quantity to develop than the cavity enlargement. In a longitudinal study of cyclists, wall thickness increased significantly from the resting to the competitive season, but the internal diameter, which was larger than control in each period, did not.<sup>27</sup> In an analysis of 947

**Table 2.** RESULTS OF THE META-ANALYSIS OF CYCLISTS AND MATCHED CONTROLS\*

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elite athletes, Pelliccia and colleagues<sup>81</sup> observed that absolute wall thickness was independently associated with body surface area, age, and male sex as well as with certain sports such as rowing and cycling. They did not analyze relative wall thickness as the dependent variable in multiple regression analysis, however.

Cyclists are particularly suited to address the question of the upper limit of physiologic left ventricular hypertrophy. Pelliccia and colleagues<sup>81</sup> reported that a left ventricular wall thickness of 13 mm or greater is uncommon and that it reaches 16 mm at the most. The author pooled the results of male road cyclists,<sup>27, 28, 32</sup> average age 22 years, who cycled 590 km/week on average at the time of the echocardiographic study and had been involved in competitive cycling for a mean of 6.4 years. Left ventricular internal diameter and mean wall thickness (i.e., the average of septal and posterior wall thickness) amounted to  $54.0 \pm 4.7$  (SD) mm and  $12.4 \pm 1.6$  mm. A thickness of 16 mm was not observed, but it reached 13 mm or greater in 14 of the 45 athletes. Rodriguez Reguero and colleagues<sup>90</sup> studied 40 professional road cyclists, mean age 26 years, and found that 21 had a ventricular thickness equal to or greater than 13 mm and that it was even greater than 16 mm in 3 athletes, with an upper limit of 19 mm.

The various studies on cyclists agree that left ventricular mass is larger than in matched nonathletes because of a larger left ventricular internal diameter and wall thickness. A large training quantity may lead to a disproportionate increase in wall thickness, which could be related to the static component involved in cycling.

### *Triathletes*

In triathletes, who combine swimming, running, and cycling, left ventricular mass was significantly elevated by 58%<sup>24</sup> and 31%.<sup>46</sup> This was due to a nonsignificant increase in left ventricular internal dimension and a significant increase in wall thickness. Consequently, relative wall thickness averaged 0.41 and 0.38 in the triathletes and 0.33 and 0.34 in the controls. The difference was significant only in the study by Douglas and associates,<sup>24</sup> which should be interpreted with caution because the athletes comprise men and women and their anthropometric data were not reported. Although it is tempting to speculate that the cycling component is responsible for

a disproportionate increase in wall thickness, more studies are needed in this type of athlete.

### *Veteran Athletes*

Data are available in active veteran long distance runners,<sup>74, 101</sup> cyclists,<sup>119</sup> marathon runners and triathletes combined,<sup>40</sup> and oarsmen.<sup>48</sup> Although body size was not reported<sup>40, 101</sup> or greater in control subjects than in athletes,<sup>48, 74</sup> all results are compatible with a larger left ventricular mass in the veteran endurance athlete.<sup>40, 48, 74, 101, 119</sup> Although the left ventricular wall was significantly thicker in athletes than in controls in each report, there was less evidence of an increased internal diameter. The difference with the nonathletes was significant only in the cyclists,<sup>119</sup> but similar although nonsignificant tendencies were present in two other groups of athletes,<sup>48, 101</sup> in whom the left ventricular internal diameter was about 3 mm larger than in controls. It appears therefore that the heart of older athletes is more apt to increase in wall thickness than in internal dimension in response to endurance training.

### **Athletes Engaged in Predominantly Dynamic Anaerobic Sports**

Sprinting involves predominantly dynamic exercise of high intensity. Left ventricular mass of 8 young female college sprinters (163 g) was significantly larger than in 40 matched control subjects (126 g). The left ventricular internal dimension tended to be greater in the athletes; relative wall thickness was not significantly different.<sup>98</sup> Male sprinters and control subjects were not well matched in two other studies,<sup>15, 52</sup> but they suggested a significantly higher left ventricular mass, internal dimension, and wall thickness in the athletes. The results are compatible with the notion that sprint training may be associated with eccentric left ventricular hypertrophy.

### **Athletes Engaged in Predominantly Static Sports**

The training regimens of a number of sports involve predominantly static exercise. A drawback of several studies or of subgroups is the mismatch of athletes and control subjects, probably as a result of difficult

ies arising from the unusual body size of athletes. Table 3 summarizes the results of the meta-analysis, which comprises data of athletes who were predominantly engaged in strength training and whose body surface area was comparable to that of the control subjects.<sup>35</sup> The six selected studies<sup>17, 59, 69, 82, 99, 116</sup> involved 42 weight or power lifters, 40 body builders, 50 wrestlers, 25 throwers, and 21 bobsledders, a total of 178 athletes, and 105 control subjects of similar age and body size. Heart rate was not significantly different between the groups. The left ventricular internal diameter, septal thickness, and posterior wall thickness were larger by 1.3, 1.4, and 1.2 mm and left ventricular mass by 39 g or 25% ( $P < .05$ ) in the athletes. Their relative wall thickness was 12% ( $P < .05$ ) higher than in controls. Relative wall thickness was also significantly elevated in judokas<sup>109</sup> and wrestlers,<sup>16</sup> but their anthropometric characteristics were not reported<sup>109</sup> or athletes and nonathletes were not well matched.<sup>16</sup> The question arises whether use of anabolic steroids is involved in the cardiac adaptation of strength athletes. Urhausen and colleagues<sup>114</sup> suggested that the intake of anabolic steroids combined with intense body building may induce a minor concentric increase in the left ventricular wall thickness, but others did not find evidence of an effect of these drugs on left ventricular structure.<sup>99, 112, 125</sup>

In general, there is no convincing evidence of an increased septal-to-posterior wall ratio in strength athletes, as has been claimed by Menapace and co-workers.<sup>64</sup> This study should, however, be interpreted with caution because of inadequate matching for body size, age, and gender; the weight lifters were all men, whereas half of the control subjects were women.

The data on athletes in whom strength

training is an important component of the training regimen suggest that predominantly concentric left ventricular hypertrophy may be observed, but the increase in left ventricular mass is small. Both absolute and relative wall thickness are higher than in controls, but the combined analysis also reveals a slightly higher left ventricular internal diameter. These results can be explained by the fact that training regimens and sports activities are not purely static<sup>68</sup> and that the duration of the actual load on the heart is usually limited.

### Athletes Engaged in Ball Sports

Apart from technical skills, most ball sports involve predominantly dynamic exercise, both aerobic and anaerobic. Reliable data are available in male basketball players<sup>91, 115</sup> and female softball<sup>98</sup> and field hockey<sup>127</sup> players (Table 4). Heart rate was usually lower and peak oxygen uptake higher in the athletic groups. Left ventricular mass was larger in the athletes. Despite some inconsistencies, both left ventricular internal diameter and wall thickness appeared to be higher in athletes than in controls, with relatively similar calculated or reported relative wall thickness. The results are compatible with eccentric left ventricular hypertrophy in athletes engaged in the reported ball sports.

## CARDIAC FUNCTION IN ATHLETES

### Left Ventricular Systolic Function

Left ventricular systolic function has most often been studied by use of echocardiography or radionuclide ventriculography and expressed as fractional shortening of the left

**Table 3.** RESULTS OF THE META-ANALYSIS OF STRENGTH ATHLETES AND MATCHED CONTROLS\*

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**Table 4. CROSS-SECTIONAL COMPARISON OF ATHLETES INVOLVED IN BALL SPORTS AND CONTROL SUBJECTS**

Authors	Subject	Sex	Number	Age (y)	BSA (m <sup>2</sup> )	HR (beats/min)	LVID <sub>d</sub> (mm)	IVST <sub>d</sub> (mm)	PWT <sub>d</sub> (mm)	LVM (g)	h/R	FS (%)	EF (%)	Vo <sub>2</sub> Peak (mL/min/kg)
Roeske et al, 1976 <sup>91</sup>	C	Male	10	26.5	2.20	66	49.9	12.8	9.8	(214)	(0.45)	—	76	—
	BAS	Male	10	25.4†	2.19†	53*	53.7*	13.7†	11.1*	(274)	(0.46)	—	79†	—
Zeldis et al, 1978 <sup>27</sup>	C	Female	25	22.1	1.56	71	42.3	8.7	10.3	128	(0.45)	—	75	41
	FH	Female	10	20.1	1.62†	59*	47.3*	8.3†	10.7†	178*	(0.40)	—	76†	52*
Rubal et al, 1981 <sup>98</sup>	C	Female	10	19–24	†	71	44	7.5	7.3	123	(0.34)	34	—	40
	SOF	Female	9	19–23	†	51*	48*	8.9*	8.2†	168*	(0.36)	33†	—	55*
Van Decker et al, 1989 <sup>115</sup>	C	Male	11	31	2.3	63	57	9.5	9.3	201	0.34	34	54	—
	BAS	Male	12	26†	2.4*	61†	59†	11.4*	11.4*	284*	0.38†	34†	52†	—

Statistical analysis: comparison between athletes and controls: \* $P \leq .05$ , † $P > .05$ .

‡Matched for age and body size.

— = Not reported; ( ) variables between parentheses have been calculated.

FS = Fractional shortening; EF = ejection fraction; C = control subjects; BAS = basketball; FH = field hockey; SOF = softball.

For all other abbreviations, see Table 1.

ventricular internal dimension or ejection fraction. The meta-analysis on long distance runners, cyclists, and strength athletes revealed that these indices of systolic function were usually not different between athletes at rest and matched control subjects.<sup>35</sup> Results were similar in swimmers,<sup>63, 76, 97</sup> triathletes,<sup>46</sup> ball players,<sup>91, 98, 115, 127</sup> sprinters,<sup>97</sup> veteran endurance athletes,<sup>40, 74, 101, 119</sup> and female athletes in general.<sup>42</sup> In addition, the increase of fractional shortening or ejection fraction on dynamic exercise was not different from controls in long distance runners<sup>33, 61</sup> or in aerobically trained subjects.<sup>12</sup> In some studies, however, fractional shortening or ejection fraction at rest was significantly higher<sup>48, 52, 100</sup> or depressed<sup>19, 43, 79</sup> in athletes, but these values were still within normal limits. Other indices of systolic function, such as the peak posterior wall velocity,<sup>28, 31, 43, 52, 113, 116</sup> the peak velocity of the internal diameter change,<sup>28, 31, 116</sup> or peak ejection rate,<sup>12</sup> were similar in athletes and controls. The data therefore suggest a normal left ventricular systolic function in athletes, but according to preliminary evidence, left ventricular systolic performance at peak exercise may be enhanced in older endurance trained athletes.<sup>100</sup>

The evaluation of contractility in humans is complicated, however, by the fact that contractile indices are influenced by afterload, preload, and heart rate. In cyclists, the relationship between the fractional shortening index and systolic wall stress was similar to that obtained in matched sedentary subjects.<sup>32</sup> Further appreciation of subtle changes is difficult, however, from noninvasive studies alone.

### Left Ventricular Diastolic Function

Left ventricular diastolic function has been studied by use of mechanocardiography, ra-

dionuclide techniques, imaging echocardiography, and Doppler velocimetry. At rest, the atrial wave on the left ventricular apex cardiogram of cyclists<sup>117</sup> and the left ventricular filling rate by radionuclide measurements in runners<sup>45</sup> and aerobically trained subjects<sup>12</sup> were similar to control values. Imaging echocardiography revealed that peak rates of chamber enlargement or filling, posterior wall movement, or wall thinning were not different from controls in runners,<sup>28, 31, 33</sup> swimmers,<sup>18</sup> cyclists,<sup>28</sup> triathletes,<sup>24</sup> throwers,<sup>116</sup> power lifters,<sup>18</sup> and basketball players.<sup>115</sup> The ratio of the transmitral Doppler peak flow velocity during atrial contraction (A) to the peak flow velocity during rapid left ventricular filling (E) (or vice versa) was normal in runners,<sup>1, 31</sup> swimmers,<sup>76</sup> cyclists,<sup>32</sup> triathletes,<sup>46</sup> weight-lifters,<sup>1, 80, 125</sup> throwers,<sup>116</sup> and basketball players.<sup>115</sup> In other studies of runners<sup>37</sup> and triathletes,<sup>24</sup> however, the A-wave was proportionately lower than the E-wave. This can probably be ascribed to the lower heart rate, which prolongs the diastolic filling period and reduces the atrial component. Heart rate is a significant determinant of the A/E ratio in multivariate regression analysis.<sup>32</sup> There has been concern about left ventricular diastolic function in athletes with pronounced left ventricular hypertrophy. Table 5 summarizes the results in 45 professional road cyclists, divided according to left ventricular wall thickness. The results indicate that the mitral inflow A/E ratio was similar in the 14 athletes with wall thickness greater than or equal to 13 mm, when compared to the 31 cyclists with wall thickness less than 13 mm.

The overall evidence obtained with various techniques therefore suggests that left ventricular diastolic function at rest is similar in athletes and nonathletes. Evidence is accumu-

**Table 5.** LEFT VENTRICULAR STRUCTURE AND SYSTOLIC AND DIASTOLIC FUNCTION IN COMPETITIVE ROAD CYCLISTS, DIVIDED IN TWO GROUPS, ACCORDING TO MEAN WALL THICKNESS\*

	Mean Wall Thickness		P
	<13 mm	≥13 mm	
Number	31	14	—
MWT <sub>d</sub> (mm)	11.7 ± 1.1	14.3 ± 0.8	—
LVID <sub>d</sub> (mm)	53.0 ± 5.0	55.4 ± 3.9	0.11
FS (%)	31.4 ± 4.7	32.1 ± 5.6	0.68
A/E ratio	0.42 ± 0.11	0.40 ± 0.11	0.70

\*Values are means ± SD.

MWT<sub>d</sub> = Mean wall thickness at end-diastole; LVID<sub>d</sub> = left ventricular internal diameter at end-diastole; FS = fractional shortening of the left ventricular internal diameter; A/E ratio = ratio of transmitral peak flow velocity during atrial contraction to early filling peak flow velocity.



lating, however, that left ventricular diastolic function, assessed by radionuclide ventriculography, imaging, or Doppler echocardiography, is enhanced in the exercising endurance-trained athlete<sup>12, 33, 44, 61, 73</sup> as compared with untrained control subjects, which favors adequate filling of the ventricle when the diastolic period gets shorter at higher heart rates. In addition, preliminary evidence suggests enhanced diastolic function in older master endurance athletes as compared to age-matched sedentary controls, so that diastolic dysfunction associated with normal aging appears to be less pronounced in exercise-trained persons.<sup>40, 110</sup>

## ROLE OF GENETIC ENDOWMENT AND ENVIRONMENTAL FACTORS

### Athletes Engaged in Predominantly Dynamic Sports

Differences in cardiac structure between athletes and nonathletes cannot necessarily be ascribed to the training per se. There is evidence that maximal aerobic power, which is universally high in endurance athletes, has a strong genetic component.<sup>29</sup> Because a high maximal aerobic power is partly the result of a high cardiac output and stroke volume, an inherited larger heart could contribute to the superior maximal oxygen uptake. In a study of young monozygotic and dizygotic male twins, however, the genetic variance of cardiac variables was much smaller than that of peak oxygen uptake.<sup>30</sup> Another study revealed that the weight-adjusted inheritance amounted to 29% and 68% for septal and posterior wall thickness but was nil for the left ventricular internal diameter.<sup>9</sup> Adams and colleagues<sup>3</sup> concluded from twin studies that

cultural familial influences are more important in determining echocardiographic cardiac size than nonfamilial or genetic influences. It seems therefore that genetic factors are not of major importance for the explanation of *athlete's heart*, particularly not for the eccentric type of hypertrophy. This assertion is in agreement with the observation of Bielen and Fagard,<sup>7</sup> who found similar cardiac sizes on chest radiograms in present athletes and nonathletes when they were 7 years old. In addition, echocardiographic cardiac structure was not different from controls in 12-year-olds who trained regularly and intensively, despite their higher peak oxygen uptake.<sup>5</sup> Rowland and associates<sup>95</sup> found no difference in echocardiographic structure in 12-year-old runners compared to matched controls involved in a variety of organized sports or exercise programs but not in endurance training. By contrast, 10- to 11-year-old swimmers consistently showed an elevated left ventricular internal dimension and wall thickness,<sup>4, 63, 76, 94</sup> which could be related to the early start of intensive training in this sport.

Three types of studies are available to assess the cardiac effects of changes in physical activity per se: the evaluation of athletes in training and nontraining periods, the results of longitudinal training studies of sedentary subjects, and observations in spinal cord-injured patients. The author identified nine studies<sup>10, 14, 25, 27, 56, 60, 79, 105, 121</sup> in which 11 groups of competitive athletes, engaged in predominantly dynamic sports, were assessed in different training states.<sup>35</sup> There were 22 long distance runners, 19 swimmers, 12 cross-country skiers, 63 cyclists, and 35 rowers, a total of 151 athletes, whose weighted age averaged 22.6 years (Table 6). Peak oxygen uptake was 4.8 mL/minute/kg higher in the trained state, heart rate 3.3

**Table 6.** RESULTS OF THE META-ANALYSIS OF LONGITUDINAL OBSERVATIONS IN 11 GROUPS OF ATHLETES: DATA FROM THE INACTIVE PERIOD AND CHANGE FROM THE INACTIVE TO THE ACTIVE PERIOD

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**Table 7.** DATA OF LONGITUDINAL DYNAMIC TRAINING STUDIES IN NONATHLETIC SUBJECTS

Authors	Characteristics of Subjects				Training Program				
	Sex	Group	Number Entered	Age (y)	Duration (wk)	Weekly Frequency	Time (min)	Intensity	Methods
Wolfe et al, 1979 <sup>122</sup>	Male	TG	20	36.8	26	4	10–30	60–80% of HR <sub>m</sub>	jog (by)
Stein et al, 1980 <sup>107</sup>	Male + female	TG	14	20	14	3	34	70–90% of HR <sub>m</sub>	by
		CG	10	19					
Adams et al, 1981 <sup>2</sup>	Male	TG	25	22	12	5	50	85% of HR <sub>m</sub>	jog
		CG	11	21					
Greenen et al, 1982 <sup>41</sup>	Male + female	TG	38	6.8	32	4	25	HR = 150–185 regular physical education	games, dance
		CG	41	7.1	32	2	25		
Landry et al, 1985 <sup>57</sup>	Male + female	TG	20	25	20	4–5	40–45	60–80% of HR <sub>r</sub>	by
		CG	5						
		TG	20	20					
Cox et al, 1986 <sup>20</sup>	Male + female	TG	11	22.5	7	6	40	85–100% of V <sub>O<sub>2m</sub></sub>	by, run
		CG	5	27.9					
Morrison et al, 1986 <sup>70</sup>	Female	TG	22	51.1	32	3	40	65–75% of HR <sub>r</sub>	walk, jog
		CG	10	51.5					
Rubal et al, 1987 <sup>97</sup>	Female	TP	10	19–31	10	3.5	≅ 30	>70% of HR <sub>m</sub>	run
		DP			10				
		TP	10	19–30	10		≅ 30		
Vanhees et al, 1992 <sup>118</sup>	Male	TG	30	38	16	3	60	70% of HR <sub>r</sub>	by, jog, cal
		CG	15	—					
Dart et al, 1992 <sup>22</sup>	Male + female	TP	10	20–30	4	3	30	70% of V <sub>O<sub>2m</sub></sub>	by
		DP			4				
Wolfe et al, 1992 <sup>123</sup>	Female	TG	12	19.3	11	3–5	20–45	80–85% of HR <sub>r</sub>	run
		CG	4	21.3					
McDonald et al, 1993 <sup>82</sup>	Male	TG	9	22–34	10	3	60	60% of V <sub>O<sub>2m</sub></sub>	by
		CG	4						

Statistical analysis: comparison of posttraining vs pretraining in exercise and control groups separately: \* $P \leq .05$ ; † $P > .05$ ; comparison of changes in exercise group and control group: ‡ $P \leq .05$ ; § $P > .05$ .

||LVM in  $\text{gim}^2$  BSA.

— = Not reported.

V<sub>O<sub>2m</sub></sub> = Maximal oxygen uptake; HR = heart rate; LVID<sub>d</sub> = left ventricular internal diameter at end-diastole; IVST<sub>d</sub> = interventricular septal thickness at end-diastole; PWT<sub>d</sub> = posterior wall thickness at end-diastole; LVM = left ventricular mass; FS = fractional shortening; EF = ejection fraction; TG = training group; CG = control group; TP = training phase; DP = detraining phase; HR<sub>m</sub> = maximal heart rate; HR<sub>r</sub> = heart rate reserve; jog = jogging; by = bicycle; run = running; cal = calisthenics.

beats/minute lower, the left ventricular internal diameter 1.1 mm larger, and the septum and the posterior wall 0.7 and 0.5 mm thicker. The difference in left ventricular mass amounted to 26 g or 13% ( $P \leq .01$ ). Relative wall thickness had risen by 4.6% ( $P = .05$ ) in the active period. Systolic left ventricular function remained unchanged ( $P = .85$ ). In addition, Crouse and associates<sup>21</sup> followed female basketball players during the competitive season and found increases of the left ventricular internal diameter, wall thickness, and mass.

Longitudinal dynamic training studies of healthy, nonoverweight nonathletes, in which a nontraining control group<sup>2, 20, 41, 57, 62, 70, 107, 118, 122, 123</sup> or an inactive phase was included,<sup>22, 97</sup> are summarized in Table 7. Noncontrolled studies or studies in which detraining consisted of only a reduction in training quantity

are not considered in this article.<sup>23, 26, 51, 53, 58, 84, 85, 88, 103, 111, 121, 126</sup> The training characteristics are given in Table 7. A significant increase of maximal oxygen uptake was achieved in each group of subjects, a reduction in heart rate in most. In adults, training induced a significant increase in left ventricular internal diameter,<sup>2, 20, 22, 57, 70, 107</sup> wall thickness,<sup>20, 57, 118</sup> or left ventricular mass<sup>20, 57, 118</sup> in several training groups but was not associated with changes in the others.<sup>62, 97, 122, 123</sup> When compared with control subjects, 7-year-old children involved in an aerobic exercise program for 8 months showed a progressive increase in left ventricular mass.<sup>41</sup> On average, the net difference between the fit and the unfit state amounted to +2.7% for the left ventricular internal dimension and to +6.2% for wall thickness; the difference averaged +11.6% for reported or calculated left ventricular mass. These results

**Table 7.** DATA OF LONGITUDINAL DYNAMIC TRAINING STUDIES IN NONATHLETIC SUBJECTS (Continued).

Results																
n	VO <sub>2</sub> Peak (mL/min/kg)		HR Rest (beats/min)		Echocardiography											
	pre	post	pre	post	LVID <sub>d</sub> (mm)		IVST <sub>d</sub> (mm)		PWT <sub>d</sub> (mm)		LVM (g)		FS (%)		EF (%)	
					pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
12	42	50*	65	56*	52.2	52.9†	8.3	8.4†	7.8	7.9†	148	152†	—	—	74	76†
10	48	48†	61	60†	51.4	51.5†	8.7	8.7†	9.0	8.9†	171	170†	—	—	77	77†
14	29	38*	74	67*	47	50*	—	—	—	—	—	—	32	35*	—	—
10	28	27†	—	—	47	47†	—	—	—	—	—	—	30	31†	—	—
22	49	56‡	63	54‡	45.7	49.6‡	10.7	10.4§	10.9	10.2§	(175)	(186)	29	32§	—	—
9	56	55	60	57	48.4	49.3	10.6	10.7	9.7	11.0	(176)	(198)	27	27	—	—
38	—	—	84	89§	40	40§	3.9	4.7§	3.9	4.7‡	21.2	27.4‡	40	38§	—	—
41	—	—	85	84	39	40	4.4	4.6	4.3	4.6	23.4	25.8	38	38	—	—
20	37	48*	—	—	47.1	47.9*	8.1	8.9*	7.5	8.3*	91	106	—	—	—	—
5	no significant changes															
20	44	50*	—	—	45.6	47.1†	8.2	8.6†	7.5	7.6†	87	97†	—	—	—	—
8	no significant changes															
11	41	53*	69	61*	49.6	51.3*	10.4	11.5*	10.9	11.2†	272	231*	—	—	62	68*
5	51	48†	—	—	51.8	51.7†	10.8	10.4†	10.4	10.2†	(208)	(200)	—	—	—	—
17	27.3	30.8*	—	—†	46	48*	8	9†	8	9†	(117)	(146)	—	—	66	74*‡
8	23.7	24.4*	—	—†	49	49†	9	10†	8	8†	(141)	(152)	—	—	77	71†
10	38	46*	75	66†	47	49†	5.9	7.1†	6.0	6.9†	106	140†	—	—	69	72†
38	—	—	69	—	48	—	6.5	—	6.5	—	128	—	—	—	65	—
10	42	50*	75	65†	54	57†	7.8	8.4†	7.5	8.1†	180	220†	—	—	64	67†
43	—	—	76	—	52	—	7.4	—	7.5	—	169	—	—	—	65	—
23	38	44*	66	58*	50.5	50.1†	10.3	11.5*	9.8	11.1*	187	215*	30	30†	—	—
12	—	—	65	66†	50.5	50.2†	10.5	10.0†	10.1	10.2†	193	186†	33	30†	—	—
10	38	44*	67	60*	49.8	51.1*	8.3	8.4†	8.2	8.0†	166	172†	40	40†	—	—
39	—	—	64	—	50.5	—	8.2	—	8.2	—	167	—	39	—	—	—
7	41	46‡	68	68§	45.8	46.9§	7.4	7.4§	7.2	7.3§	116	124§	33	33§	—	—
4	45	42	67	71	45.5	46.8	7.3	6.5	7.6	7.2	117	110	33	35	—	—
(9)	46	50*	62	57*	52.0	52.6†	8.9	9.0†	9.0	9.0†	(167)	(171)	38	39†	—	—
(4)	43	39*	65	68†	52.0	51.7†	9.0	10.0†	9.3	9.7†	(171)	(188)	42	39†	—	—

therefore suggest that predominantly dynamic exercise can alter left ventricular structure. In addition, left ventricular mass index was smaller in patients with spinal cord injuries, in whom selection bias is virtually excluded, in comparison to normally active subjects.<sup>55, 120</sup>

The results in athletes and nonathletes are, however, not always in agreement with the notion that dynamic training leads to eccentric left ventricular hypertrophy, which could be due to the limitations of echocardiography in relation to the small changes of cardiac structure. Fractional shortening or ejection fraction remained unchanged with training in most studies<sup>2, 22, 25, 41, 62, 97, 118, 121–123</sup> and was slightly impaired<sup>60, 79</sup> or improved in others.<sup>10, 14, 20, 27, 70, 107</sup> One could speculate that the natural endowment for endurance performance, including a superior aerobic power, facilitates exercise and allows prolonged and intensive endurance training in the motivated subject, leading to cardiac adjustments, a secondary rather than a primary phenomenon in the high-level athlete.

### Athletes Engaged in Predominantly Static Sports

Genetic factors could be involved in concentric left ventricular hypertrophy in view of the significant heritability of weight-adjusted absolute and relative wall thickness but not of the left ventricular internal dimension.<sup>9</sup> Data on strength training are scarce and not controlled. The results of Kanakis and Hickson<sup>54</sup> are compatible with the development of concentric left ventricular hypertrophy. They studied the left ventricular response to a 10-week program of lower-limb strength training in nine healthy male subjects aged 18 to 27. Left ventricular mass increased by 13% ( $P < .05$ ) and wall thickness by 12% ( $P < .05$ ) with no changes in internal dimension. Twenty weeks of weight lifting in 12 adolescent boys increased left ventricular mass by 4.3% ( $P < .05$ ), but the changes in internal dimension and wall thickness did not reach statistical significance.<sup>88</sup> Smith and colleagues<sup>104</sup> examined seven college wrestlers during preseason and 4 months later at the

season's end. Left ventricular mass and wall thickness increased by about 20% ( $P < .01$ ), whereas the internal dimension remained unchanged.

## Conclusion

Studies on the genetic variance of cardiac structure indicate that the genetic component is small, particularly for the left ventricular internal diameter. It is therefore unlikely that preexisting genetic factors are important to explain differences in internal diameter that exist between endurance athletes and sedentary subjects. Differences in wall thickness may be partly genetically determined. Training per se may cause changes in cardiac structure, although the changes that have been observed are smaller than differences between athletes and nonathletes. This may obviously be due to the training regimen of athletes, which cannot be equaled in sedentary subjects, and to the fact that regression during the resting season in athletes is incomplete. The role of nontraining environmental factors such as nutrition is not well understood.

## SUMMARY

There is overwhelming evidence, particularly from echocardiography, that the heart of competitive athletes may differ from that of nonathletes, matched for age, gender, and body size. A larger left ventricular mass has been shown in athletes performing predominantly dynamic aerobic and anaerobic sports, in athletes engaged in static training, and in players of ball sports. Enlargement of the left ventricular internal diameter was most pronounced and reached about 10% in athletes performing predominantly dynamic sports; mainly strength training athletes had a lesser increase of the internal dimension, which was limited to 2.5%. Also the left ventricular wall appeared to be thickened in all types of athletes compared with controls. In sports with high dynamic and low static demands, wall thickness was proportionate or slightly disproportionate to the size of the internal diameter so that relative wall thickness was not different from controls or slightly increased (predominantly eccentric hypertrophy). In strength athletes, the disproportionate increase of wall thickness averaged about 12%

(predominantly concentric hypertrophy). In sports with high dynamic and high static demands and requiring prolonged training, such as cycling, the increases of absolute and relative wall thickness reached 29% and 19% and were more pronounced than in runners (mixed hypertrophy). A plausible interpretation of these results is that the development of so-called eccentric or concentric left ventricular hypertrophy according to the type of sports cannot be regarded as an absolute or dichotomous concept because training regimens and sports activities are not exclusively dynamic or static and because the load on the heart is not purely of the volume or the pressure type. Most studies agree that left ventricular systolic and diastolic function is normal in the athlete at rest, whereas diastolic function seems to be enhanced in the exercising endurance athlete. The consistency of the results of studies on athletes in the competitive and the resting season, of training of sedentary subjects, and of spinal cord-injured patients suggests that variations in physical activity can alter left ventricular structure; genetic factors do not seem to be involved in the size of the left ventricular internal diameter but have to be taken into account to interpret wall thickness.

## ACKNOWLEDGMENT

The author gratefully acknowledges the secretarial assistance of N. Ausselees.

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