

CARDIAC STRUCTURE AND FUNCTION IN YOUNG ENDURANCE ATHLETES AND NONATHLETES

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The purpose of this study was to examine, in male adolescents, the effects of long-term endurance training on cardiac structure and function, by adopting a cross-sectional comparison with a nonathletic control group. A total of 13 endurance-trained (EX) and seven untrained (CON) male adolescents (mean \pm SE, age = 15.3 ± 0.3 and 15.2 ± 0.28 yrs, respectively) underwent echocardiography at rest to determine left ventricular end-diastolic dimension (LVD_d), left ventricular end-systolic dimension (LVD_s), left ventricular posterior wall thickness (LVPW), stroke volume (SV), and cardiac output (CO). On separate days, incremental exercise tests were conducted on a cycle ergometer to measure peak oxygen uptake ($\dot{V}O_{2max}$) and anaerobic power. $\dot{V}O_{2max}$ was greater in the endurance group (54.4 ± 1.8 mL min^{-1} kg^{-1}) than in the control group (45.8 ± 1.6 mL min^{-1} kg^{-1} ; $p < 0.05$). Mean exercise time was longer in EX (12.9 ± 0.7 min) than CON (10.4 ± 0.8 min; $p < 0.05$). No significant differences were noted between the two groups in resting heart rate, maximal heart rate, LVD_d, LVD_s, LVPW, SV, SV indexed, CO, and CO indexed, or in the anaerobic strength. These data provide evidence that endurance-trained adolescent males develop superior exercise performance before the cardiac remodeling that is evident in trained adult athletes.

Keywords: endurance training, cardiac structure and function, exercise performance, oxygen uptake, male adolescents

Introduction

Cardiorespiratory function is critical to the performance of many sports and physical activities, such as long-distance running and swimming. In endurance-trained adult athletes, the cardiorespiratory system typically shows evidence of having undergone a series of favorable adaptations (Ikaheimo et al. 1979), which are thought to contribute to performance. These adaptations include left ventricular (LV) enlargement, chamber wall

thickening, an increase in cardiac contractility, an increase in cardiac output (CO), and electrocardiographic changes (Smith et al. 1994; Stork et al. 1992). However, there are almost no data on endurance-trained children and adolescents to inform scientists, coaches, and athletes about the normal sequence of adaptive changes that manifest in adult athletes. Timing of cardiovascular adaptations in young people is of great interest across a range of issues, including athletic performance, talent identification, growth and development, and cardiovascular health in adulthood. In one of the few studies on adolescent athletes, some differences in cardiac structure and function were reported between trained and untrained volunteers (Ricci et al. 1982). The differences included an increase in the LV mass in the groups trained for endurance (10%) and those trained for strength

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(4%). A major confounding issue in the interpretation of both physiologic and structural responses to exercise training in children and adolescents is that these responses invariably take place in the context of growth and maturation. This raises methodologic and, in some cases, even ethical difficulties with conducting these investigations. Possibly as a corollary, children are encouraged to delay specialized intensive training for specific sports until they reach early to middle adolescence (American Academy of Pediatrics 2000).

Many important questions arise about the possible effect of exercise training on the cardiac structure and function of children and adolescents (Baxter-Jones et al. 2003). The studies focusing on the associations between long-term exercise training and cardiac structure and function in young athletes during rest and exercise may provide answers to some of these questions. Most previous studies (Stork et al. 1992; Ricci et al. 1982) comparing cardiac structure and function in adult athletes and nonathletes were cross-sectional, and, thus, it was not possible to determine whether the exercise training was responsible for all, or some, of the traits. There are three main hypotheses to explain the interactions of exercise training and cardiac structure and function (Stolt et al. 2000; Karjalainen et al. 1997; Smith et al. 1994). First, exercise training is responsible for all of the changes. Alternatively, some athletes may have pre-existing cardiac advantages and engage in self-selection to endurance-type activities. Third, cardiac profiles in athletes reflect a combination of exercise training and pre-existing cardiac structure and function. To thoroughly test these three hypotheses would require extensive longitudinal studies. In the current research, cross-sectional observations of cardiac structure and function at rest and during exercise were made for young male adolescent endurance-trained athletes and for sedentary controls matched for age and gender. If the differences between the groups were less than they are for adults, or even absent, then this disparity lends some indirect support to the hypothesis that exercise training contributes to the improvements in cardiac structure and function that are commonly seen in endurance-trained athletes.

Therefore, the purpose of this study was to compare exercise responses and cardiac structure and function at rest in endurance-trained (EX) and untrained (CON) male adolescents aged 14–16 years.

Methods

This study examined the cardiac structure and function, as well as the maximal oxygen consumption ($\dot{V}O_{2max}$) during exercise testing of 13 EX male adolescents and seven CON male adolescents (age range, 14–16 yrs).

EX adolescents volunteered from an elite swimming squad and an athletics club. Each had participated in organized endurance training, including swimming or running, at least three times per week for the past year. Each training session lasted 1–2 hours. Work intensity was self-rated using a scale of 1–5, when 1 = light training that does not result in perspiration or make you feel tired; 2 = training that results in a little perspiration and minimal fatigue (recovery takes only a few minutes); 3 = training that results in profuse perspiration, a heart rate increase ($140\text{--}150\text{ beats}\cdot\text{min}^{-1}$), and considerable exhaustion (recovery takes a few minutes to 15 minutes); 4 = training that results in profuse perspiration, a considerable heart rate increase ($> 150\text{ beats}\cdot\text{min}^{-1}$), and more exhaustion (recovery takes $> 15\text{ min}$); 5 = hard work sessions that result in maximal heart rate (MHR) increase ($> 180\text{ beats}\cdot\text{min}^{-1}$), abundant perspiration, and complete exhaustion. Most of the trained adolescents in this study had a work intensity score above 3. It is generally acknowledged that exercise should be performed at an average intensity of at least 70% of the MHR, at least three times per week and at least 30 minutes per session to elicit improvements in aerobic capacity (Leicht et al. 2003; Hoffman 2002).

The CON group came from peers of the EX group or some high schools in Melbourne. The volunteers did not belong to any athletics club and had not participated in regular specific endurance training during the past year.

Before any testing, all participants were informed of the risks of the procedures, for which they and their parents gave written consent. The study protocol was approved before the procedures began by the relevant Victoria University Human Research Ethics Committee and conformed to the National Health and Medical Research Council (Australia) Statement on Ethical Conduct of Research in Humans (1999). All participants were free of injuries at the time of testing and for 3 months before participation, and were not taking any prescribed medications that may have affected exercise performance.

All participants were measured for height, body mass with a portable digital scale, and four skinfold sites (Harpenden, British Indicators Ltd, West Sussex, UK). Anthropometric measures were used to calculate body surface area (BSA), the body composition percentages of lean body mass (fat-free mass, FFM) and body fat by Body Composition Analysis program Series A, Repco, Melbourne, Australia.

$\dot{V}O_{2max}$ tests were conducted using a symptom-limited incremental protocol (commencing at 25 W with increments of 25 W min^{-1}) on an electronically braked cycle ergometer (Excalibur, Lode, Groningen, The Netherlands). Cadence was self-selected in the range of 80–100 revolutions per minute. Cycling continued until volitional fatigue occurred. Expired air was analyzed online using open-circuit spirometry (Applied Electrochemistry S-3A and CD-3A analyzers, Ametek, Pittsburg, PA, USA). All instrumentation used in the measurement of $\dot{V}O_{2max}$ was calibrated using standard methods before and immediately after each test. Criteria for establishing that $\dot{V}O_{2max}$ had been reached consisted of a plateau in $\dot{V}O_2$ for two successive increments of power (Bickham et al. 2002). The criterion used for reaching a plateau in $\dot{V}O_2$ was that further increases in exercise intensity did not lead to an additional increase in oxygen consumption. If the subject did reach a plateau, the secondary criterion was that the heart rate reached 195 beats/minute.

During aerobic testing, each participant's heart rate was recorded each minute and rhythm was monitored continuously using a telemetry electrocardiograph (X-Scribe Stress Test System, Mortara Instrument Inc, Milwaukee, WI, USA).

Echocardiography was used to measure cardiac structure and function at rest. During this procedure, participants were asked to lie on a bed for as long as 60 minutes, in the left lateral decubitus position. A 3.5-MHz transducer was placed in the fourth or fifth intercostal space adjacent to the left sternal border. Using the parasternal long-axis view, an average of 3 measurements by M mode of the interventricular septum (end diastole), LV cavity size (end diastole and end systole), and LV posterior wall thickness (LVPW) (end diastole) were made. From the parasternal long-axis view, LV outflow tract (LVOT) diameter was measured by two-dimensional imaging, using an average of three measurements. The left ventricle ejects a volume of blood into the cylindrical

aorta on each beat. The base of this cylinder is the systolic cross-sectional area of the aorta, and its height is the distance the average blood cell traveled during ejection for that beat. This distance is expressed as the velocity-time integral (VTI). VTI was taken to be the average of measurements from three consecutive heartbeats, except for irregular rhythms, where VTI was calculated from the average of five beats. VTI was also used in the calculation of stroke volume (SV) and CO.

$$SV = LVOT \text{ area} \times LVOT \text{ VTI}$$

$$CO = SV \times \text{heart rate}$$

t tests were used to determine significant differences between the two groups for height, weight, body composition, exercise heart rates, $\dot{V}O_{2max}$, and cardiac indices. Descriptive statistics are reported as means and standard deviation of the mean (mean \pm SD). Statistical analysis was made using SPSS for Windows (Version 10.0.5; SPSS Inc. Headquarters, Chicago, IL, USA). For all variables, the alpha level of $p < 0.05$ was considered significant.

Results

The anthropometric values for the two groups (EX and CON) are shown in Table 1. These characteristics were similar for the two groups. Although EX showed a lower mean value for percentage body fat than CON, this was not statistically significant.

$\dot{V}O_{2max}$ was significantly higher in the endurance group ($54.41 \pm 1.79 \text{ mL min}^{-1} \text{ kg}^{-1}$) than the untrained group ($45.83 \pm 1.6 \text{ mL min}^{-1} \text{ kg}^{-1}$, $p < 0.05$; Table 2), with corresponding differences in time to fatigue (12.9 ± 0.7 minutes for EX, 10.4 ± 0.8 minutes for CON, $p < 0.05$;

Table 1. Characteristics of young endurance athletes and nonathlete control subjects*

	EX (<i>n</i> = 13)	CON (<i>n</i> = 7)
Age (yrs)	15.3 \pm 0.3	15.20 \pm 0.28
Height (cm)	174.2 \pm 2.8	171.2 \pm 3.3
Body mass (kg)	60.8 \pm 2.9	58.7 \pm 3.3
BSA (m ²)	1.7 \pm 0.2	1.7 \pm 0.2
Percentage body fat	8.9 \pm 0.7	11.8 \pm 1.7
Percentage FFM	91.2 \pm 0.6	88.2 \pm 1.7

* Values are mean \pm SD.

EX = experimental group; CON = control group; BSA = body surface area; FFM = fat-free mass.

Table 2). Absolute $\dot{V}O_{2\max}$ was not significantly different between the two groups (Table 2). MHRs were not significantly different between the groups, as well.

Resting heart rates were not significantly different between EX and CON (Table 3). LV end-diastolic dimension (LVD_d), LV end-systolic dimension (LVD_s), LVPW, SV, SV indexed, CO, and CO indexed were not significantly different between EX and CON (Table 3).

Discussion

Several studies have demonstrated large percentage improvements in $\dot{V}O_{2\max}$ in physically conditioned adults (Gravelle et al. 2000; Yamazaki et al. 2000), due to changes in both the cardiovascular system and skeletal muscle cells. Several earlier investigators (Obert et al. 1998; Rowland et al. 1998; Unnithan et al. 1996) had also provided evidence indicating that prepubertal endurance athletes would attain a greater $\dot{V}O_{2\max}$ than untrained prepubertal children. Some researchers reported an average training improvement in $\dot{V}O_{2\max}$ in approximately 10% of adolescents (Naughton et al. 2000). The present study found a greater relative $\dot{V}O_{2\max}$ (mL kg⁻¹ min⁻¹) in EX male adolescents than in CON male adolescents, but no significant difference in absolute $\dot{V}O_{2\max}$ (L min⁻¹) in EX males and CON males. This result is probably due to the effect of body composition on $\dot{V}O_{2\max}$ results. Previous studies indicated that the mean value of $\dot{V}O_{2\max}$ showed a significant negative relationship with body fat percentage ($r = -0.55$) (Al-Hazzaa et al. 1994). In our study, both groups of adolescent males were similar in age, height, weight and BSA. The EX group showed a lower mean value in body-fat percentage (8.89%) than

Table 2. Results of symptom-limited graded exercise test*

	EX (n = 13)	CON (n = 7)
$\dot{V}O_{2\max}$ (L min ⁻¹)	3.3 ± 0.19	2.7 ± 0.19
$\dot{V}O_{2\max}$ (mL min ⁻¹ kg ⁻¹)	54.41 ± 1.79 [†]	45.83 ± 1.60 [†]
$\dot{V}O_{2\max}$ (mL min ⁻¹ kg ⁻¹ FFM)	59.69 ± 2.00 [†]	52.19 ± 2.40 [†]
MHR (beats min ⁻¹)	194.38 ± 3.66	188.71 ± 5.50
Time to fatigue (min)	12.85 ± 0.69 [†]	10.43 ± 0.75 [†]

*Values are mean ± SD.

[†]Significant difference between groups ($p < 0.05$).

EX = experimental group; CON = control group; $\dot{V}O_{2\max}$ = peak oxygen uptake; FFM = fat-free mass; MHR = maximal heart rate.

Table 3. Cardiac structure and function at rest in young endurance athletes (EX) and nonathlete control subjects (CON)*

Parameter	EX (n = 13)	CON (n = 7)
LVD _d (mm)	5.26 ± 0.13	5.38 ± 0.20
LVD _d /BSA (mm/m ²)	3.05 ± 0.02	3.12 ± 0.02
LVD _s (mm)	3.59 ± 0.12	3.40 ± 0.23
LVD _s /BSA (mm/m ²)	2.08 ± 0.02	1.96 ± 0.02
LVPW (mm)	0.80 ± 0.02	0.79 ± 0.02
LVPW/BSA (mm/m ²)	0.47 ± 0.02	0.46 ± 0.02
Resting heart rate (beats min ⁻¹)	61.00 ± 1.19	60.00 ± 3.50
SV (mL beat ⁻¹)	91.23 ± 5.41	92.42 ± 3.95
SV indexed (mL min ⁻¹ m ²)	52.35 ± 2.48	54.72 ± 2.30
CO (L min ⁻¹)	5.89 ± 0.37	5.51 ± 0.19
CO indexed (mL min ⁻¹ m ²)	3.40 ± 0.17	3.25 ± 0.18

*Values are mean ± SD.

LVD_d = left ventricular end-diastolic dimension; BSA = body surface area; LVPW = left ventricular posterior wall thickness; LVD_s = left ventricular end-systolic dimension; SV = stroke volume; CO = cardiac output.

did the control group (11.80%), although a significant difference could not be demonstrated. The FFM encompasses all of the body's nonfat tissues, including the skeleton, water, muscle, connective tissue, organ tissues and teeth. The use of FFM to normalize $\dot{V}O_2$ is more appropriate for research questions examining physiologic changes during growth and maturation than it is for questions examining endurance performance in weight-bearing activities. The results of the present study confirm that the greater $\dot{V}O_2$ of FFM (mL min⁻¹ kg⁻¹ FFM) occurs in trained male adolescents.

Superior aerobic strength was not accompanied by significant differences, or even trend differences, between the two groups for LVD_d, LVD_s, and LVPW, even when the latter are expressed relative to BSA. Therefore, these data suggest that superior $\dot{V}O_{2\max}$ in endurance-trained adolescents who do not have cardiac hypertrophy occurs, either as a result of the training, or as an inherent trait. There is evidence that trained adult athletes develop LV enlargement, chamber wall thickening, and increased cardiac contractility and CO (Smith et al. 1994; Stork et al. 1992), suggesting that these adaptations become manifest because of the functional improvements that result from long-term endurance training. We acknowledge, however, that the phase of rapid physical growth during adolescence increases the complexity of studying the effects of exercise train-

ing in people in this developmental phase. During adolescence, substantial growth and development of the cardiorespiratory and musculoskeletal systems occur. Therefore, cardiorespiratory function may be related to the combination of maturation, with its rapid changes in body mass and body composition, and the physical training. The purpose of using a control group and cross-sectional comparison design in this study was to eliminate the influence of these variables.

$\dot{V}O_{2max}$ is directly related to maximal CO, arteriovenous oxygen saturation, pulmonary capacity of oxygen transport, oxygen muscular extraction, and metabolic rate. $\dot{V}O_{2max}$ and maximal CO are linearly correlated ($r = 0.84$) (Bello et al. 1995), and the increases in $\dot{V}O_{2max}$ with training result primarily from increases in maximal CO, not from increases in maximal arteriovenous oxygen differences (Yamazaki et al. 2000). In the present study MHRs were similar in both groups, indicating that the higher $\dot{V}O_{2max}$ in the EX individuals was predominantly due to higher maximal SVs in the athletes. This finding is supported by earlier work, showing that improvements in maximal CO resulted almost entirely from increased SV, with little change in MHR (Rowland 1992). Furthermore, SV and CO are similar for trained and untrained volunteers at rest (Rowland et al. 1998; John 1969).

Cardiovascular adaptations related to endurance training are well known among male adult athletes. During endurance exercise, the heart improves its ability to pump blood mainly by increasing its SV. This occurs because of an increase in end-diastolic volume, LV contractility, and LV muscle mass. Endurance training is thought to cause LV enlargement characterized both by dilation, due to increased blood volume loads (preload), and by hypertrophy, in response to increased contractility and force of contraction (afterload). These modes of cardiac enlargement are distinct from those occurring in heart failure, with endurance training being associated with improved systolic LV ejection (Giorgi et al. 2000; Ikaheimo et al. 1979) rather than the impaired ventricular function that is characteristic of heart failure. Endurance-exercise training may increase preload by several mechanisms, including increases in circulating blood volume (Ricci et al. 1982), or increased filling times at rest and submaximal exercise (manifested as lower heart rates) to lead to increased SVs. EX adult athletes

showed elevated values for LV chamber dimension, LV mass, LV mass index, and SV index when compared with untrained subjects, and the extent of these adaptations was related to training volumes (Stork et al. 1992). The cardiac structural changes during rest are adaptation responses to long-term exercise training. These adaptations add to the efficiency of the heart's energy utilization and may improve the maximum CO, the $\dot{V}O_{2max}$, and endurance performance during exercise (Giorgi et al. 2000). In contrast to adult studies, the extent to which endurance training can alter cardiac function and size in children and adolescents is not well understood. Some recent research (Obert et al. 1998; Rowland et al. 1998) has shown that the cardiac structure and function of children as well as adults may incur adaptive changes with exercise training, although these changes might be quantitative differences in the absolute values during rest and exercise between children and adults. In contrast to the findings with the child athletes, adult endurance athletes demonstrated a greater SV than nonathletes at rest, as well as during exercise. The above findings have suggested that there might be quantitative differences in the absolute values measured in some of the cardiac responses during rest and exercise between children and adults. In the current study, LVD_d , LVD_s , and LVPW were not significantly different between EX and CON male adolescents. Previous reports of cardiac adaptations with growth indicated that if long-term training is a stimulus for cardiac hypertrophy, the adaptation is slow and mild in young people (Bale 1992). Children may need greater exercise intensity than adults to trigger cardiovascular adaptations to training (Baxter-Jones et al. 2003), and requires further study. The degree of improvement is thought to be dependent on such variables as training volume, training modes, and the initial physical capacities of the child or adolescent. After 8 weeks of endurance training, LV internal dimension and LV wall thickness did not change in 16-year-old adolescent boys (Ricci et al. 1982). The brief duration of the intervention possibly contributed to the absence of hypertrophic changes in the heart in that investigation. In the present cross-sectional study, the duration that participants had been engaged in endurance training was at least 12 months, yet cardiac structure and function at rest were not different from their age-matched peers. Effective responses to endurance training depend on

sufficient training stimuli, comprised of the intensity, duration, and frequency of training. The role of genetics in facilitating observed adaptations is also considered to be important. Insufficient long-term exposure to endurance-training stimuli, in combination with genetic factors and/or the slowness of resting adaptations to cardiovascular structure and function in the athletic adolescents, may explain the absence of differences in the current data. That the cardiac structural variables measured during rest in our study were not accompanied by significantly increased $\dot{V}O_{2\max}$ and endurance performance, may suggest the potential role of variables extrinsic to the cardiovascular system that may be supportive of endurance performance and increased $\dot{V}O_{2\max}$ (Cameron et al. 1980). Such extrinsic variables, including the active muscle mass, increased oxidative capacity of skeletal muscle, increased muscle blood flow and possible increased total blood volume, mental capacity to resist the symptoms, and physical manifestations of fatigue, may play important roles in the development of maximal gas transport and consumption in young athletes (St Clair Gibson et al. 2001).

Furthermore, it might be difficult to expose more profound cardiovascular differences in trained versus untrained children, because most 'untrained' children are also active, through active recreation habits, compulsory school sports, or physical education classes. Bale (1992) commented that, in most studies, the effects of exercise training on children can only be assessed in weeks or months, yet in adults the effects are more easily monitored over longer periods of time. Thus, the assumption that physical activity, exercise, or training explains the differences between inactive and athletic children in functional capacity, over and above growth and maturation, should consider not only the differences of exercise training but also the influence of active recreation habits, compulsory school sports, or physical education classes.

Conclusions

In conclusion, the EX male adolescents had higher $\dot{V}O_{2\max}$ during exercise testing than the CON adolescent males, although height, weight, body composition, and FFM were similar for both.

EX adolescent males did not show greater SVs, CO at rest, or cardiac dimensions than CON male adolescents, as was hypothesized. This observation suggests that endurance-training stimulus may have to be of greater intensity, duration, and frequency than was evident for this cohort. The observation that these changes are evident in adults suggests that if long-term training is a stimulus for cardiac hypertrophy, the adaptation is slow and modest in young people. Alternatively or in addition, the CON group may have been engaged in sufficient exercise that may have masked any differences that otherwise may have existed.

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