

Atrioventricular Plane Displacement in Untrained and Trained Females

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ABSTRACT

SLØRDAHL, S. A., V. O. E. MADSLIEN, A. STØYLEN, A. KJOS, J. HELGERUD, and U. WISLØFF. Atrioventricular Plane Displacement in Untrained and Trained Females. *Med. Sci. Sports Exerc.*, Vol. 36, No. 11, pp. 1871–1875, 2004. **Purpose:** Female athletes often demonstrate changes in cardiac dimensions that are less prominent than in male athletes, and results from longitudinal studies are conflicting. The atrioventricular plane displacement (AVPD) in the heart is used as an index of left ventricular systolic function with the assumption that it is a more sensitive method for measuring myocardial contractility compared with left ventricular ejection fraction. The aim of the present study was to determine the effect of a short period of endurance training on cardiac dimensions in sedentary female subjects and to measure the AVPD at rest and during submaximal workload. **Methods:** Twelve sedentary female subjects (21.9 ± 1.3 yr, 168.8 ± 3.5 cm, 64.0 ± 6.6 kg, and 42.6 ± 2.9 mL·kg⁻¹·min⁻¹ in maximal oxygen uptake) were examined with echocardiography before and after a period of interval training (varying from 2 to 5 min at 90–95% of maximal heart rate, 3 d·wk⁻¹, 8 wk). **Results:** Maximal oxygen uptake increased by 18% to 50.4 ± 3.1 mL·kg⁻¹·min⁻¹ ($P < 0.001$). Left ventricular mass increased from 123.9 to 139.2 g ($P = 0.007$). There was a significant increase in posterior wall thickness but no change in cavity size. The AVPD did not change at rest but increased significantly from 15.6 to 17.6 mm ($P < 0.001$) during exercise at 85–90% of maximal heart rate. **Conclusion:** This study shows that a short period of aerobic endurance training induces changes in the female heart, both in cardiac dimensions at rest and in left ventricular systolic function at submaximal workload. AVPD during submaximal exercise discriminate well between the untrained and trained healthy heart. **Key Words:** LEFT VENTRICULAR FUNCTION, EXERCISE, ULTRASOUND, FEMALE ATHLETES, ATHLETES HEART

Long-term training is associated with an increase in cardiac dimensions in both males (18,25) and females (9–11,20,26). Female athletes often demonstrate changes in cardiac dimensions that are less prominent than in male athletes. Pelliccia et al. (17) found that the absolute left ventricular cavity size exceeded normal limits in only a minority (8%) of the female athletes, and all had left ventricular wall thickness within normal limits. Longitudinal studies have shown inconsistent verification of training-induced increase in left ventricular cavity size and wall thickness in females (4,16,21,30). In previously sedentary

subjects, Cox et al. (4) found adaptive changes in left ventricular dimensions after 7 wk of intense endurance training: 6 d·wk⁻¹ with alternating days of continuous cycling (40 min) and interval running (5 × 5-min bouts).

Traditionally, intrinsic contractile function of the left ventricle has been measured by ejection fraction and fractional shortening. However, because Lundbäck (15) described the heart works as a displacement pump with atrioventricular plane displacement (AVPD), the magnitude of AVPD has routinely been used to estimate left ventricular function with the assumption that AVPD is a sensitive method for measuring myocardial contractility (13). Several studies have addressed Lundbäck's hypothesis of the heart as a displacement pump, using AVPD as an index of left ventricular systolic function, and found that AVPD decreases as left ventricular systolic function deteriorates (1,2,24). Although AVPD corresponds with the pump function of a failing heart, it is unclear whether improved cardiac function, as in endurance-trained athletes, can be assessed by changes in AVPD. In a recent study, we found that the AVPD decreased during physical activity in endurance-trained female athletes, indicating that the AVPD may not

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TABLE 1. Physical and physiological characteristics before and after the training period.

Variable	Before	After	P
$\dot{V}O_{2\max}$ (mL·kg ⁻¹ ·min ⁻¹)	42.6 ± 2.9	50.4 ± 3.1	<0.001
Body weight (kg)	64.0 ± 6.6	63.9 ± 6.8	NS
Hematocrit (%)	38.5 ± 1.7	39.1 ± 1.4	NS
Hemoglobin (g·L ⁻¹)	13.0 ± 0.4	13.2 ± 0.5	NS
Resting heart rate (beats·min ⁻¹)	71.3 ± 9.0	59.3 ± 6.7	<0.05
Submaximal heart rate (beats·min ⁻¹)	182.8 ± 18.3	176.9 ± 14.5	NS

Data are mean ± SD; *N* = 12; $\dot{V}O_{2\max}$, maximal oxygen uptake.

be an important mechanism of increased cardiac pumping during exercise (29). However, that study had methodological limitations and needs verification. Furthermore, there exist no reports on the effect of aerobic endurance training on the AVPD. Therefore, in the present study we determined the effect of a short period of endurance training on the AVPD at rest and during exercise, and whether training could induce changes in left ventricular dimensions in young sedentary females.

METHODS

Twelve sedentary females (age, 21.9 ± 1.3 yr; height, 168 ± 3.5 cm) were examined with echocardiography before and after 8 wk of aerobic interval training. None of the subjects had been engaged in a formal training program for at least 3 months before this study. The subjects had a maximal oxygen uptake of 42.6 ± 2.9 mL·kg⁻¹·min⁻¹ and a maximal heart rate of 202.5 ± 7.1 beats·min⁻¹. The regional medical research ethics committee approved the study, and written informed consent was obtained from all subjects. Physical and physiological characteristics of the subjects are presented in Table 1.

Training program. The subjects exercised under the supervision of one of the authors (V. O. E. Madslie) three times per week for 8 wk. Each exercise started with a warm-up period of 15–20 min at an exercise intensity corresponding to 60–70% of maximal heart rate. Then the interval period lasted about 25 min and consisted of walking/running up a steep hill for 2–5 min with an exercise intensity corresponding to 90–95% of maximal heart rate, with a 2- to 3-min active recovery at 50–60% of maximal heart rate between the intervals. Heart rates were measured manually or by short-range radio telemetry (Polar Sportster, Polar Electro, Finland). Each subject reported all exercise in a training diary as easy exercise with heart rates between 60 and 80% of maximal value, as moderate with heart rates between 80 and 90% of maximal, or as hard with heart rates above 90% of maximal. Mean exercise time per subject during the 8-wk period was 23 ± 5.8 h; 29% of this was reported as hard exercise and 23% as moderate exercise.

Maximal oxygen uptake. $\dot{V}O_{2\max}$ and maximal heart rate was measured by running on a treadmill (Woodway PPS 55) at a 5.5° incline as previously described (29). Oxygen uptake, minute ventilation, and breathing frequency were measured using an Ergo Oxyscreen (Jaeger EOS Sprint, Höchberg, Germany). Blood was drawn from a fin-

gertip after the $\dot{V}O_{2\max}$ test, and blood lactate concentration was analyzed immediately with a YSI Model 1500 Sport Lactate Analyzer (Yellow Springs Instruments, Yellow Springs, OH). All subjects had an unhemolized lactate concentration above 6.60 mmol·L⁻¹ (mean 9.17 ± 1.84 mmol·L⁻¹). Hemoglobin and hematocrit were measured before and after the training period. Minimal or resting heart rate was measured after 10 min of supine rest preceding the treadmill tests, or, if lower, the heart rate was measured during the resting echocardiographic examination.

Echocardiographic measurements. Echocardiographic recordings were performed by an experienced cardiologist using a Vingmed System Five scanner (GE Vingmed Ultrasound, Horten, Norway) with a 2.5-MHz transducer. Echocardiographic data were transferred to a computer and analyzed off-line using EchoPAC software (GE Vingmed Ultrasound). Resting examinations in a supine position were performed in the left lateral decubitus position. Examinations during supine bicycle exercise were performed with the upper part of the body elevated at about 20°. Cine-loops of parasternal long-axis views, parasternal M-mode, cine-loops of the apical four-chamber, and two-chamber views were recorded. Left ventricular (LV) dimensions were calculated from the M-mode recordings by the leading-edge to leading-edge convention of the American Society of Echocardiography (12). LV mass was determined from the M-mode measurements by Devereux and Reichek's formula (6). Longitudinal M-mode recordings were generated off-line from the two-dimensional cine-loops. A cursor was drawn from the apex through the mitral ring immediately adjacent to the valve. AVPD was defined as the distance from end diastolic to end-systolic position of the plane, which represents the total motion of that point during systole, in the apical direction. The procedure was performed in the septal and lateral points of the four-chamber plane, and in the anterior and posterior points of the two-chamber plane. The two points in the same plane are thus simultaneous; the two planes, however, are sequentially recorded. The average of the four points' motion represents the average apical motion of the atrioventricular plane (AVPD). The method is similar to that described by Höglund et al. (13) and has earlier been found to be both valid and reliable (24). Cine-loops of the two- and four-chamber planes were transferred to the computer for later analysis. An interobserver analysis was performed in the present study for AV plane displacement and left ventricular mass, which includes the measurement of chamber dimensions and wall thickness. The coefficient of variation was 8.7% for left ventricular mass, 3.1% for AV plane displacement at rest, and 7.3% during activity.

Intra- and interobserver variability. Intra- and interobserver variability are presented according to the requirement from MSSE® (<http://www.sportsci.org/resource/stats/relycalc.html#excel>). The intraobserver variability is presented below: change in mean AVPD 0.01 mm (confidence limits -0.01 to 0.05), typical error 0.02 mm (0.02–0.06), intraclass *r* = 0.98; changes in mean septum diameter in diastole 0.01 mm (-0.02 to 0.05), typical error 0.03

TABLE 2. M-mode measurements before and after the training period.

Variable	Before	After	P
Left ventricular mass (g·kg ⁻¹)	1.94 ± 0.45	2.20 ± 0.48	<0.01
Left ventricular internal diameter, end-diastole (mm)	48.1 ± 0.48	49.4 ± 0.26	NS
Left ventricular internal diameter, end-systole (mm)	31.9 ± 0.32	32.6 ± 0.20	NS
Septum thickness, end-diastole (mm)	8.3 ± 0.21	7.8 ± 0.13	NS
Septum thickness, end-systole (mm)	10.1 ± 0.22	10.0 ± 0.14	NS
Posterior wall thickness, end-diastole (mm)	6.0 ± 0.11	7.1 ± 0.11	<0.05
Posterior wall thickness, end-systole (mm)	12.2 ± 0.13	12.7 ± 0.13	NS
Left ventricular ejection fraction, rest (%)	0.70 ± 0.04	0.69 ± 0.05	NS
Left ventricular ejection fraction, activity (%)	0.72 ± 0.05	0.70 ± 0.05	NS
Atrioventricular plane displacement rest (mm)	15.3 ± 1.6	14.8 ± 1.0	NS
Atrioventricular plane displacement exercise (mm)	15.6 ± 0.8	17.6 ± 0.8	<0.05

Data are mean ± SD.

(0.02–0.06), intraclass $r = 0.94$; changes in mean left ventricular internal diameter 0.01 mm (–0.04 to 0.06), typical error 0.06 (0.04 – 0.09), intraclass $r = 0.98$; changes in mean posterior wall diameter in diastole 0.004 mm (–0.03 to 0.04), typical error 0.04 (0.03 – 0.06), intraclass $r = 0.78$.

The interobserver variability in our laboratory was: change in mean AVPD –0.05 mm (–0.12 to 0.02), typical error 0.08 mm (0.05–0.13), intraclass $r = 0.86$; changes in mean septum diameter in diastole –0.013 mm (–0.06 to 0.03), typical error 0.05 mm (0.04–0.08), intraclass $r = 0.84$; changes in mean left ventricular internal diameter 0.03 mm (–0.02 to 0.08), typical error 0.06 mm (0.04–0.10), intraclass $r = 0.98$; changes in mean posterior wall diameter in diastole –0.03 mm (–0.06 to 0.00), typical error 0.04 mm (0.03–0.06), intraclass $r = 0.73$.

Protocol. The resting heart rate and $\dot{V}O_{2\max}$ were measured before and after the 8-wk training period. In addition, the cardiac dimensions and the AVPD were measured simultaneously, and before and during submaximal exercise. To maintain a submaximal heart rate during the echocardiographic examination, the subjects ran from the treadmill (after measuring the $\dot{V}O_{2\max}$) to the echocardiographic examination and kept the heart rate high by supine bicycle exercise. The aim was to keep the heart rate above 85% of maximal heart rate.

Statistical analyses. The results are reported as mean and standard deviation (SD). A paired Student's *t*-test was used to determine differences between the measurements before and after the training period. A *P* value of < 0.05 was considered to represent a significant difference.

RESULTS

The AVPD at submaximal heart rate increased significantly after the training period from 15.6 ± 0.8 to 17.6 ± 0.8 mm ($P < 0.001$). Submaximal heart rate was not different before and after the training period (182.8 vs 176.9 beats·min⁻¹, $P = 0.43$, 90 vs 87% of maximal heart rate). The AVPD was the same at rest before and after the training period. The left ventricular ejection fraction (LVEF) did not change during the training period (0.70 ± 0.04 vs 0.69 ± 0.05) and was the same during activity. LVEF did not correlate to AVPD.

The left ventricular mass increased significantly from 123.9 ± 22.7 to 139.2 ± 23.8 g ($P = 0.007$). The posterior

wall thickness increased in end-diastole (Table 2) but not in end-systole. Left ventricular internal diameter did not change at end-diastole or at end-systole during the period.

The 8-wk training period gave a significant increase in $\dot{V}O_{2\max}$ from 42.6 ± 2.9 to 50.4 ± 3.1 mL·kg⁻¹·min⁻¹, which is a mean increase of 18.3% ($P < 0.001$). The body weight, hemoglobin, and hematocrit were the same before and after the training period (Table 1). The resting heart rate decreased significantly from 71 ± 9 to 59 ± 7 beats·min⁻¹ ($P < 0.001$).

DISCUSSION

This study shows that a short period of aerobic endurance training increases AVPD during submaximal workload in untrained females. Increased AVPD indicates improved intrinsic myocardial contractility during exercise. This is in accordance with Lundbäck's hypothesis (15,28), where AVPD contributes increasingly and Frank-Starling activation decreasingly to enhanced cardiac output at high exercise levels. Thus, AVPD during exercise is a good estimate of left ventricular systolic function, because it discriminates well between the healthy untrained and trained heart.

In a recent study (29), however, we observed a 15% decrease in AVPD in both sedentary and trained female subjects during exercise at 95% of maximal heart rate, whereas the LVEF increased by 25% in both groups. In this previous study, the resting values were obtained in a supine position while the exercise was done in a sitting position which obviously could influence the results. AVPD in the present study was in the normal range reported for healthy individuals in the same age group (13).

At rest, we found similar results in this and our previous study (29), with no difference in AVPD before and after a training period in sedentary females, and no difference between female athletes and controls. Similarly, we found, as in our previous study, no difference in LVEF at rest in female athletes compared with sedentary female controls. This is consistent with previous studies in both males and females reporting similar ejection fraction (3,7,21) and fractional shortening (5,9,22,30) in athletes and controls. However, Cox et al. (4) found an increased LVEF after the training period. Based on most previous studies, ejection fraction and AVPD at rest seem to be unaffected by the trained state of the subject. However, endurance training

improves left ventricular function at rest by reducing resting heart rate, which is an indicator of increased stroke volume (9,10). We observed a reduction in resting heart rate after the training period, but we do not know whether we obtained the heart rate during a true resting condition because it was done on the test days. Only a few studies (e.g., (10)) have examined the prevalence of sinus bradycardia in female athletes. A lower resting heart rate in endurance-trained females may result from reduced intrinsic heart rate, enhanced vagal tone, or both.

This study confirms that a short period of aerobic endurance training induces changes in cardiac dimensions. The results are similar to findings by Cox et al. (4), who found an increase in ventricular wall thickness after 7 wk of intense endurance training in sedentary subjects (six females, five males). It is important to emphasize that we only obtained significant changes for the posterior wall and that the calculation of left ventricular mass is dependent on these measurements. The present study shows the efficiency of aerobic interval training at high intensity. It should be noted that the training program used in the present study with only three exercise sessions per week for 8 wk gave an increase in maximal oxygen uptake of more than 18%, changing the subjects' condition from being untrained to well trained. The rationale behind the present training regimen is based upon the fact that $\dot{V}O_{2max}$ offers a precise measure of the capacity to transport and use oxygen, and reflects the functional capacity of the lungs, cardiovascular system, and muscle mitochondria combined (19,23). At maximal aerobic exercise, the majority points to a $\dot{V}O_{2max}$ that is oxygen-supply limited (23,27). This appears to be evident in highly trained athletes (20) and in average fit humans (14). Consequently, cardiac output has a major influence on $\dot{V}O_{2max}$ (23,27). Furthermore, because it is known that the stroke volume does not reach a plateau before an exercise intensity corresponding to $\dot{V}O_{2max}$ (~95% of maximal heart rate), we chose an interval training intensity that requires maximal stroke volume, and thereby put pressure upon AVPD. The present data support the notion that high intensity aerobic interval training is effective in improving $\dot{V}O_{2max}$, probably by increased cardiac output.

Marsh et al. (16) found that increasing moderate endurance running from 15 to 62 miles·wk⁻¹ did not change cardiac dimensions or $\dot{V}O_{2max}$. Ten sedentary female students were endurance-conditioned by 10 wk of running in another study (21). The training program consisted of running every other day for a minimum of 30 min at a heart rate above 70% of the maximal value. They found a tendency, though not a significant one, toward increased cardiac dimensions, whereas they did observe a significant increase in $\dot{V}O_{2max}$ of 20%. Similarly, Wolfe et al. (30) found a significant increase in $\dot{V}O_{2max}$ of 10.9% in seven sedentary females that participated in an 11-wk running program (4–5 sessions per week). They also found an increase in wall thickness and left ventricular mass, but mean changes in the training group were not significantly different from those observed in the control group for all echocardiographic

variables. Training intensity was 80–85% of maximal heart rate reserve, and started with 20-min sessions that were lengthened 5 min per session every 2 wk. The explanation of these results compared with the studies with significant changes in cardiac dimensions can be that the intensity of the training was lower. On the other hand, the changes in dimensions that have been demonstrated in females after a training period are not very prominent, and the explanation can be that the accuracy of echocardiography has not been good enough to show marginal changes.

Left ventricular cavity diameter did not change in the present study despite decreased heart rate at rest. Eshani et al. (8) found that cavity size increased before changes in ventricular wall thickness. The main reason for differences between studies in which cavity changes occur first is probably the relatively small size of the groups and problems in detecting small changes with echocardiography during a short training period. Fagard et al. (9) found a similar ratio of wall thickness to internal radius in female runners and nine matched controls. Similarly, Riley-Hagan et al. (20) found using magnetic resonance imaging in female endurance athletes that the increase in left ventricular mass was proportional to the increase in left ventricular end-diastolic volume compared to sedentary controls. We believe that the main reason for increased $\dot{V}O_{2max}$ after the training period is a little increase in ventricular volumes in spite of the fact that we did not achieve significant changes and increased ejection fraction as shown by changes in AVPD. Female endurance athletes are shown to have lower heart rates than control groups (10). A reduction in heart rate at rest after the training period in this study and the significant increase in $\dot{V}O_{2max}$ can also in part be a consequence of adaptations in the skeletal muscles as the muscle's ability to deliver, extract, and use oxygen increases.

Limitations of the study. Echocardiographic recording with the subjects exercising in a semisupine position might not be representative of submaximal exercise performed in the upright position. The nature of the echocardiography, on the other hand, requires a semisupine position to get reliable data. Furthermore, testing conditions were similar at pre- and posttests, and therefore suited to measure exercise-induced adaptation in the female heart.

CONCLUSIONS

A short period of aerobic endurance training induces changes in the female heart, both in cardiac dimensions at rest and in left ventricular function evaluated by AVPD during submaximal exercise. AVPD during submaximal exercise may be used to discriminate between a healthy untrained and trained heart.

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