Left Ventricular Dimensions of Adolescent Males: A 12 Weeks Interval Training Report

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Abstract

The purpose of the study was to find out the effect of 12 weeks of interval training with 60-80% intensity for 5-15 repetitions of 400meters on left ventricular end diastolic diameter (LVEDD), left ventricular end diastolic volumes (LVEDV), left ventricular end systolic diameter (LVESD) and left ventricular end systolic volumes (LVESV) of adolescent males. Echocardiographic measurements in athletes should take into account the specific sport and the quantity and quality of training. Sixteen non-residential untrained male subjects (8 Experimental & 8 Control) ranging between 14-16 years were selected for the study. Before the 12 weeks training protocol two-dimensional and Doppler echocardiography was performed for measuring LVEDD, LVESD, LVEDV and LVESV of adolescent boys. Changes in all the parameters were insignificant in case of control group as the initial and final test means yielded lesser values than the tabulated value. The initial and final test means of LVEDD and LVEDV of the experimental group were 44.75cm, 45.5cm and 91.75ml and 95.38ml respectively and the mean difference were insignificant ($p>0.05$) whereas, 't' value of LVESD and LVESV of the experimental group were 3.77 ($p <.05$) and 3.64 ($p <.05$) respectively. The result indicates that the 12 weeks interval training was effective for significant reduction of LVESD and LVESV values of the adolescent boys.

Key wards: LVEDD, LVESD, LVEDV and LVESV.

Introduction

Regular physical exercise induces changes in the body that are termed as physiological adaptations to increased loads. In general, these adaptations are favorable and enable the individual to increase physical performance capacity (*Macfarlance et al, 1991*). Adaptations of training include the structure and function of cardiovascular system in addition to its functional control (*Urhausen and Kindermann, 1992*). Strength training induces changes to pressure loads, whereas endurance training requires volume loads and elicits an increased maximal cardiac output, by increasing stroke volume (*Andersen et al, 2000 and Astrand et al, 2003*). It has been found that sports performance and training induced adaptations are determined mainly by genetic factors and to a limited extent by training (*Kuipers, 2005*).

All forms of athletic training are associated with left ventricular hypertrophy (LVH). However, the exact effects on cardiac structure and function depend upon the type of training (*Gilbert, 1977; Nishimura, 1980*). Endurance training exerts a volume overload on the left ventricle and produces left ventricular cavity enlargement with proportional increases in myocardial thickness (*Morganroth et al, 1975 and Longhurst, 1981*). Long-term athletic training is associated with cardiac morphological changes, including increased left ventricular cavity dimension, wall thickness and calculated mass that are commonly described as "athlete's heart" (*Rost and Hollmann, 1983; Hutson et al, 1985; Maron, 1986; and Spirito et al, 1994*). These changes seem to present adaptations to the hemodynamic load produced by long term, frequent, intensive exercise programmes (*Longhurst et al, 1980; Keul et al, 1981 and Longhurst et al, 1981*). The extent to which left ventricular cavity dimensions is increased by systematic training is modest in most athletes (*Astrand et al, 2003*).

Echocardiography has become firmly established in cardiological diagnostics in last few years. Two-dimensional echocardiography yields important information, not only about pathological changes, but also about structural and functional adaptations about healthy hearts. It is useful to the sports cardiologists as it is non-invasive and is repeatable (*Urhausen and Kindermann, 1992*).

The purpose of the present study was to evaluate the effect of 12-weeks of interval training on echocardiographically determined left ventricular end diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD), left ventricular end diastolic volume (LVEDV) and left ventricular end systolic volume (LVESV) of adolescent boys.

Material & Method

Sixteen non-residential untrained male subjects ranging between 14-16 years were selected for the study. Out of these 8 subjects served as control and other 8 were included in the experimental group. Before and after the training protocol two-dimensional Doppler echocardiography tests were performed for measuring LVEDD, LVESD, LVEDV and LVESV of al the boys. Images of the heart were obtained in multiple cross sectional planes by using standard transducer position (*Tajik et al, 1978*).

Twelve weeks training was imparted to the experimental group. Initially general conditioning programme was imparted for a period of four weeks to the experimental group and for the next 12 weeks the interval training method was adopted for development of cardio-vascular endurance as per the details given in table 1. Before each training session 20 minutes of general warming up and after the training session 20 minutes of cool down protocol was followed. For the control group no such training was applied.

TABLE – 1: Weekly Schedule of Training Programme

Method	Interval training
Intensity	60 – 80%
Duration	70 to 90 sec
Distance	400 meters
Repetitions	5 – 15
Recovery	Active and incomplete, the next repetition was started when Minute Heart Rate dropped to 110 – 120 beats.
Load Frequency	Thrice a week

The initial and final test scores were compared for significance using t-test (*Garet, 1969*). The statistical analysis was tested for significance at 0.05 level of confidence.

Results and Discussion

LVEDD of experimental and control subjects before and after the training were 44.75, 45.38 and 45.5, 45.25 cm respectively. Table-2 reveals that the t-ratios obtained for the mean differences in the initial and final values for the experimental and control groups yielded insignificant values of 1.05 and 0.22 respectively, since both these values were lesser than the ‘t’ value of 2.36 required for significance at 0.05 level. LVESD of the subjects before and after interval training were 29.13 and 27.13 cm (experimental) and 28.75 and 28.75 cm (control). T-ratio for the experimental group showed significant decrease in LVESD in contrast to the control group. The t-ratios obtained for the mean differences between initial and final tests of the experimental and the control groups in LVEDV were 1.14 and 0.04 respectively, which were not significant, whereas LVESV showed significant decrease in case of experimental group ($P<0.05$).

TABLE 2: Significance of differences between the initial and final test means of experimental and control groups in LVEDD, LVESD, LVEDV and LVESV

Parameters	Initial Test	Final Test	DM	t-ratio
Exp. LVEDD (cm)	44.75	45.50	0.75	1.05
Cont. LVEDD (cm)	45.38	45.25	0.13	0.22
Exp. LVESD (cm)	29.13	27.13	2.00	3.77*
Cont. LVESD (cm)	28.75	28.75	0.00	0.00
Exp. LVEDV (ml)	91.75	95.38	3.63	1.14
Cont. LVEDV (ml)	95.00	94.88	0.12	0.04
Exp. LVESV (ml)	33.00	27.75	5.25	3.64*
Cont. LVESV (ml)	32.13	32.38	0.25	0.12

Exp. = Experimental Group while Cont. = Control Group
$t_{.05}$ (7) =2.36, * Significant at 0.05 level

Exercise training causes a number of well-known physiological changes in the heart: an increase in LVEDD and LV wall thickness that lead to increased left ventricular mass, stroke volume is increased and heart rate is decreased in resting conditions (*Brornstad et al, 1993 and Fagard, 1997*). The values obtained by echocardiography for cardiac dimensions and wall thickness for athletes do not provide a distinct data set or a bimodal distribution and are usually within the ranges accepted as normal. Although such values are usually significantly different from the normal in statistical terms, the reports on echocardiography findings in athletes are somewhat contradictory, possibly because of varying methodology (*Turpeinen, 1996 and Fagard, 1997*).

Left ventricular end diastolic diameter (LVEDD) and left ventricular end diastolic volume (LVEDV) changes were insignificant in the case of experimental subjects following interval training in the present study. Interval training requires a prolonged effort to have an effect on the LVEDD and LVEDV in an individual. The training for endurance activity is invariably of long durations during which cardiac output is sustained at high levels. The response to this type of stimulus, which may be called volume stress, may facilitate cardiac hypertrophy through an increase in the size of the ventricular cavity.

In the present study, the duration of interval training might not have been sufficient enough to cause significant increase in left ventricular end diastolic diameter and left ventricular end diastolic volume. *Pelliccia (1999*) reported that most of the elite athletes had absolute left ventricular cavity dimensions within normal limits. The magnitude of cavity dimension seems extraordinary given the fact that in normal populations (*Knutsen et al, 1989, Devereux et al, 1984, Valdeg et al, 1979*) or in previously sedentary persons undergoing short-term exercise training programmes (*DeMaria et al, 1978 and Adams et al, 1981*), it is necessary to point out that hypertrophy of the myocardium does not manifest in every endurance-trained athlete. One of the reasons of the varying myocardium remodeling response might be inadequate training programme stimulus in this regard (*Laughlin and McAllister, 1992 and Urhausen & Kindermann, 1999).*

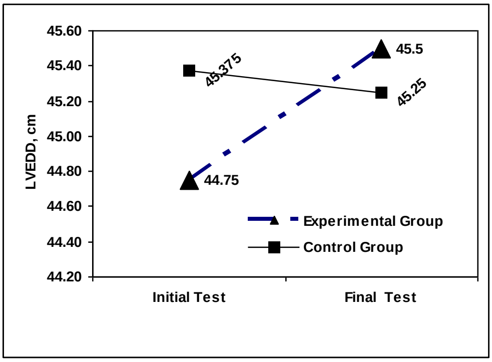

Figure 1: Comparison of LVEDD in the control and Experimental groups after 12 week interval training

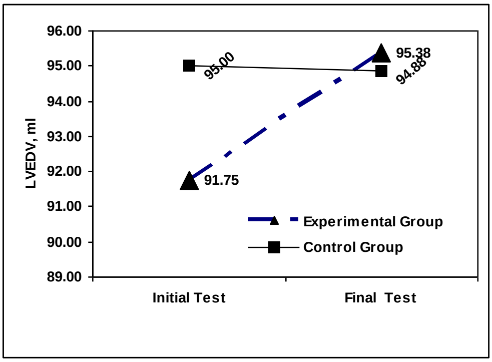

Figure 2: Comparison of LVEDV in the control and Experimental groups after 12 week interval training

In fact, for significant development of LVEDD & LVEDV prolonged period is required. The duration of training employed in this study might have been inadequate and hence there was no significant increase in LVEDD & LVEDV of the experimental subjects. The LVEDD & LVEDV of the subjects are graphically represented in Figures 1 & 2. The finding related to LVEDD was found to be in agreement with the views of *Rubal (1987), Snoeckx (1982)* and *DeMaria (1978)* and the findings of LVEDV duly support the findings of *Wolfe and co-workers (1979*).

Figures 3 & 4 represent the changes in LVESD and LVESV in the subjects after the training. Due to the interval mode of endurance training, the experimental group had to carry out a higher pre-load in regular way, for 12 weeks, which has resulted in an increase in CO during the workout.

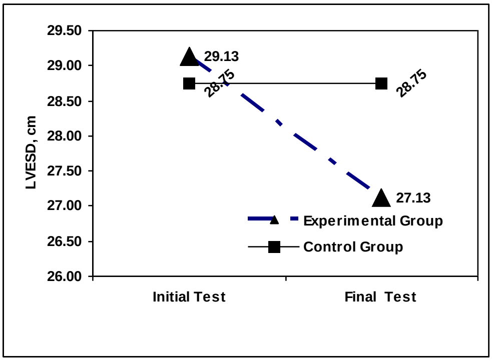

Figure 3: Comparison of LVESD in the control and Experimental groups after 12 week interval training

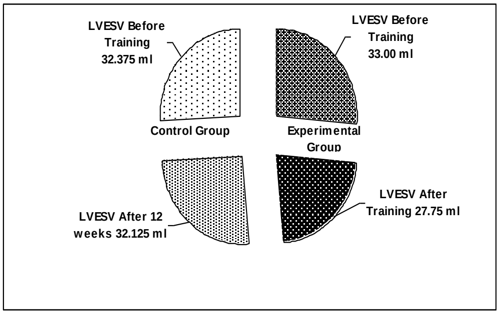

Figure 4: Comparison of LVESV in the control and Experimental groups after 12 week interval training

The resting bradycardia with increased stroke volume needs a powerful contraction of the left ventricle with every beat of the heart. The significant decrease of LVESD & LVESV in the experimental subjects may be due to the forceful stroke output resulting in the resting bradycardia. The results of LVESD were found to be in agreement with the similar results reported by *DeMaria (1978)* and the findings of LVESV were found to be concurring with the views of *Astorri and co-workers (1986)*.

Conclusions

The interval training of moderate to long duration with 60 to 80% intensity for a period of 12-weeks was successful in significantly decreasing Left Ventricular End Systolic diameter and Volume whereas it failed to cause statistically significant changes in the Left Ventricular End Diastolic diameter and Volume in the experimental subjects.

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