

**“EFFECT OF CONDITIONING, DECONDITIONING AND RECONDITIONING ON LVEDD, LVESD, LVEDV AND LVESV OF ADULT MALES”**

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**Abstract**

**Introduction:** Changes in echocardiographic standard measurements as a consequence of 8 weeks of conditioning 4 weeks of deconditioning and 6 weeks of reconditioning have not been comprehensively studied.

**Sample:** Twenty four residential untrained male subjects (age between 22-24 years) were selected for the study.

**Methodology:** Two-dimensional and Doppler echocardiography studies were performed for measuring 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 the subject. Before conditioning protocol of eight weeks, at the end of conditioning programme, at the end of four weeks of deconditioning and at the end of six weeks of reconditioning the criterion parameters were measured i.e. four times the data were collected from the subjects.

**Analysis of data:** Repeated measures design was applied for the study and ‘t’ tests were applied for statistical purpose. The statistical analysis was tested for significance at the 0.05 level of confidence.

**Findings and conclusions :** The t value of LVESV ( $P < 0.01$ ) decreased significantly following 12 weeks of conditioning programme where as the other variables remain unaffected. There was no change of any of the variables after 4 weeks of deconditioning programme and at the end of 6 weeks of reconditioning.

**Key words:** Left ventricular end diastolic diameter, Left ventricular end systolic diameter, Left ventricular end diastolic volumes and Left ventricular end systolic volumes.

**Introduction:**

The left ventricular size and function during exercise under different preloads have not been characterized at comparable heart rates and mean blood pressure in the same subject during trained and untrained states. Nor has the effect of deconditioning been delineated in subjects who have been training intensely for many years (Martin W. H, Coyle E.F, Bloomfield S.A, and Ehsani.A.A 1986).). The relationship does not change with training versus detraining state (Mandigou et.al. 2002). Prolonged and intense endurance training is through to promote an increase in heart mass and researchers believe detraining results in a decline in heart mass (G.Galanti, L.Scarti, S Castellani and L Toncelli 1987).). What is not clear, however, is whether the training induced increases in ventricular volume and myocardial wall thickness regress totally with inactivity. Athletes who become sedentary have enlarged hearts and an elevated  $VO_{2max}$  level in contrast to people who never trained (McArdle. WD, Katch. FL and Katch. VL. (2007).

The purpose of the study was to evaluate the effect of 8 weeks conditioning, 4 weeks deconditioning and again 6 weeks reconditioning on echocardiographically determined left ventricular end diastolic diameter, Left ventricular end systolic diameter, Left ventricular end diastolic volumes and Left ventricular end systolic volumes of adult males.

### **Methods:**

Twenty four residential untrained male subjects (age between 22-24 years) were selected for the study. Before the conditioning protocol two-dimensional and Doppler echocardiography studies were performed for measuring 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 the subject. Images of the heart were obtained in multiple cross sectional planes by using standard transducer position (*Tajik and coworkers 1978*).

After conditioning, deconditioning and reconditioning protocol the required data were collected from both the groups. Repeated measured design was applied for the study and 't' tests (Garet H. E. 1969) were applied for statistical purpose. The statistical analysis was tested for significance at 0.05 level of confidence. Before conditioning protocol of eight weeks, at the end of conditioning programme, at the end of four weeks deconditioning and at the end of six weeks reconditioning the criterion parameters were measured i.e. four times the data were collected from the subjects. Table-1 represents the conditioning protocol of the subjects.

**Table-1. conditioning protocol of the subjects.**

Treatment	Duration	Daily schedule	Weekly plan	Nature of activity
Conditioning	8 weeks	135 minutes in the morning and 90 minutes in the evening	5 days in a week	Warming up, continues run, lite apparatus drills, free hand exercises etc. Heart rate=140 btpm
Deconditioning	4 Weeks	rest	rest	rest
Reconditioning	6 Weeks	135 minutes in the morning and 90 minutes in the evening	5 days in a week	Warming up, continues run, lite apparatus drills, free hand exercises major games etc. Heart rates =140 btpm

### **Result and Discussion:**

**TABLE -1. Mean and standard deviation of the subjects**

Variables	No.	Pre Condition Test		Post Condition Test		Decondition Test		Recondition Test	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
LVEDD (cm)	24	47.89	2.75	47.83	2.72	47.86	2.71	47.8	2.63
LVESD (cm)	24	31.37	0.86	28.36	2.56	28.63	2.24	28.63	2.24
LVESV(ml)	24	48.39	12.19	42.58	16.79	42.58	16.26	42.69	16.07
LVEDV (ml)	24	106.87	13.34	106.92	15.37	106.59	15.16	106.55	15.38

**Table-2. Pair 't' test value of the subjects**

<b>Variables</b>	<b>Pre Vs Post test</b>	<b>Post Vs Decond test</b>	<b>Decond Vs Recond test</b>
<b>LVEDD</b>	<b>0.54</b>	<b>1.14</b>	<b>0.58</b>
<b>LVESD</b>	<b>5.07 *</b>	<b>0.79</b>	<b>-</b>
<b>LVESV</b>	<b>24</b>	<b>48.39</b>	<b>12.19</b>
<b>LVEDV</b>	<b>24</b>	<b>106.87</b>	<b>13.34</b>

**\* Significant at 0.01 level (2.807)**

The results of the tests shown in Table 1 and 2 which indicate that the t value of LVESD ( $P < 0.01$ ), decreased significantly following 8 weeks of conditioning programme where as the other variables remain unaffected. There was no change of any of the variables after 4 weeks of deconditioning programme and at the end of 6 weeks of reconditioning.

The changes of LVEDD and LVEDV were insignificant of subjects following three different conditioning programme of our study. In general, the training for endurance activity usually requires prolonged efforts, during which CO was sustained at high levels. The response of this type of stimulus, which may be called volume stress, might have facilitated cardiac hypertrophy through an increase in the size of the ventricular cavity. Athletes with left ventricular cavity dimension showed evidence of greater cardiac dimensional adaptation to training. In our present study, the duration of conditioning might not sufficient enough for significant increase of left ventricular end diastolic diameter and left ventricular end diastolic volume. The study of *Pelliccia (1999)* found that most of the elite athletes had absolute left ventricular cavity dimension within normal limits. The magnitude of cavity dimension seems extraordinary given the fact that in normal populations (*Knutsen and co workers 1989, Devereux and coworkers 1984, Valdeg and coworkers 1979*) or in previously sedentary persons undergoing short-term exercise training programmes (*DeMaria and co-workers 1978 and Adams and coworkers 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 myocardium remodeling response might be insufficient training programme stimulus in this regards (*Laughlin MH and McAllister RM 1992 and Urhausen A and Kindermann W 1999*). The findings of the present study was found to be in agreement with the views of *Pelliccia (1999)*, *Knutsen (1989)*, *Devereux (1984)* and *Veldeg (1979)*.

In fact, for significant development of LVEDD & LVEDV requires prolonged period. 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 finding of LVEDD was found to be in agreement with the views of *Rubal (1987)*, *Snoeckx (1982)* and *DeMaria (1978)* and the findings of LVEDV were duly supported by *Wolfe and co-workers (1979)*.

Due to conditioning programme, the subjects had to carry out a higher pre-load in regular way, for 8 weeks, which has resulted in an increase in CO during the workout. The resting bradycardia with increased SV, which needs a powerful contraction of the left ventricle. The insignificant decrease of LVESD & significant ( $p < 0.01$ ) LVESV of the experimental subjects may be due to powerful stroke output and resting bradycardia. The finding of LVESV was found to be in agreement with the views of *Estorri and co-workers (1986)*.

### **Conclusion:**

24 male residential student were selected for the study. All the students were performed 8 weeks of conditioning, 4 weeks of deconditioning and again 6 weeks of reconditioning. Before conditioning and at the end of each stage the tests were conducted for collecting the data, which were statistically treated. Within the limitations of the present study the following conclusions have been drawn,

1. LVEDD and LVESD of the subjects were insignificantly changed following 8 weeks of conditioning whereas remained unchanged after, 4 weeks of deconditioning and 6 weeks of reconditioning.
2. LVESV of the subjects were significantly decreased following 8 weeks of conditioning whereas remained unchanged after, 4 weeks of deconditioning and 6 weeks of reconditioning.
3. LVEDV of the subjects were insignificantly improved following 8 weeks of conditioning whereas remained unchanged after, 4 weeks of deconditioning and 6 weeks of reconditioning.

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