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Effect Of Conditioning, De Conditioning And Reconditioning On Left Ventricular Functions Of Adult Males

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Introduction: Changes in echo cardio graphic standard measurements as a consequence of 8 weeks of conditioning 4 weeks of reconditioning 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 colour Doppler echocardiography studies were performed for measuring left ventricular end systolic volume (LVESV), left ventricular end Diastolic volume (LVEDV), Ejection Fraction (EF), Fractional shortening (FS), Stroke volume (SV) and resting heart rates (RHR) of the subjects. Four times the data were collected from the subjects i.e. before conditioning protocol, at the end of eight weeks conditioning programme, at the end of four weeks of deconditioning and at the end of six weeks of reconditioning. **Analysis of data:** Repeated measured design was applied for the study and 't' tests were applied for statistical purpose. The statistical analysis was tested for significance at 0.05 level of confidence. **Findings and conclusions :** All the parameters except LVESV were significantly changed following conditioning protocol , only SV, FS, RHR were significantly differ in case of reconditioning protocol and RHR and HRsub were significantly changed after conditioning, reconditioning and reconditioning.

Key words: Left ventricular End systolic Volume (ml), Left ventricular End Diastolic Volume (ml) , Ejection Fraction (%), Fractional Shortening (%), Stroke Volume(ml) and Resting and sub-maximal heart rates (beats/min).

Introduction :

Assessment of echocardiographic measurements in athletes should take into account the specific sport and the quantity and quality of training. All parts of athlete's heart are enlarged and its performance increases. Highly trained endurance athletes show the most enlarge hearts [Urhausen. A and Kindermann. W. (1992)]. Regular physical exercise induces changes in the body that are a physiological adaptation to increased loads. In general, these adaptations are favourable and enable the individual to increase physical performance capacity. Adaptations of training also include the structure and function of cardiovascular system in addition to its functional control [McArdle. WD, Katch. FL and Katch. VL. (2007)]. 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. JL, Scherling. P and Saltin. B. (2000), Astrand. P.O, Rodahl. K, Dahl H.A and Stromme. S.B. .(2003)]. It has become clear that sports performance and training induced adaptations are determined mainly by genetic factors and to a limited extent by training [Kuipers Harm. (2005)]. 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 [Huston. TP, Puffer. JC and Rodney. WM. (1985), Maron BJ. (1986), Pelliccia. A, Maron. BJ. (1997) and Tajik et. al. (1978)]. These changes seem to present adaptations to the hemodynamic load produced by long term, frequent, intensive exercise programmes [Keul. J, Dickhuth. HH, Simon. G and Lehmann. JH. (1981), Kuipers Harm. (2005) and Longhurst. JC, Kelly. AR, Gonyea. WJ and Mitchell. JH. (1980)]. Echocardiography has become firmly established in cardiological diagnostics in last few years. 2-dimentional 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 cardiology as it is non-invasive and is repeatable [Urhausen. A and Kindermann. W. (1992)].

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)]. The volume of the heart was measured for the first time probably by Stephen Hales (1941) in the eighteenth century, to investigate the capacity of the heart chamber. The measured subject was the heart of a horse, one of the greatest athletic animals. Hales filled it with melted bees wax and then calculated the displacement of the solidified mass according to the Archimides Principle. Later, similar experiments were made on the hearts from cadavers by Hiffelshien and Robin (1864) and by Hochrein (1927). Some others made models of the heart with the aid of projections in several planes, which took a considerable time to prepare. The greatest development in this field was the introduction of the calculation of heart volume from the frontal and sagital orthodiagrams, independently by Rohrer(1916) and Kahlstorf (1932). The shape of the heart was assumed to be between a parabolid and an ellipsoid. The reliability of this method was demonstrated by Kahlstorf himself and was confirmed by several other researchers. Musshoff and Reindell (1956) . Introduced some modifications in this method and improved the formula of Rohrer-Kahlstorf into the form used by most of the investigators today.

The purpose of the study was to evaluate the effect of 8 weeks conditioning, 4 weeks deconditioning and again 6 weeks reconditioning on echocardioghaphically determined left ventricular LVESV, LVEDV, FS, EF, SV, RHR and HRsub of adult males.

Methods:

Twenty four residential untrained male subjects (age between 22-24 years) were selected for the study. Two dimensional and Doppler echocardiography studies were performed for measuring left ventricular End systolic volume, left ventricular End Diastolic Volume, Ejection Fraction, Fractional Shortening, and Stroke Volume. Images of the heart were obtained in multiple cross sectional planes by using standard transducer position by using the formula proposed by Devereux Rb. (1987).

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.

Results and Discussions:

The experimental protocol was followed as per the guideline of Table -1. 8 weeks of conditioning 5 days in a week, followed by 4 weeks of rest and again 6 weeks of reconditioning were applied for the study and at the beginning of the treatment the desirable data were collected.

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

Table-2. 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
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
SV (ml)	24	57.42	18.13	67.35	11.90	59.21	15.98	59.12	15.88
EF (%)	24	64.89	8.66	69.73	6.69	67.98	6.06	67.98	6.05
FS (%)	24	34.14	4.39	39.64	5.61	35.06	3.44	34.98	3.4
RHR (btpm)	24	74.75	4.51	66.66	6.31	68.92	3.26	64.5	5.01
HRsub (btpm)	24	149.83	9.15	139.83	11.23	142.08	13.52	137.5	7.78

Table-3. Pair 't' test value of the subjects

Variables	Pre Vs Post test	Post Vs Decond test	Decond Vs Recond test
LVESV	0.05	1.88	0.09
LVEDV	3.14**	0.02	1.44
SV	3.99**	3.55**	1.44
EF	3.16**	1.71	-
FS	5.8**	5.10**	1.44
RHR	4.43**	2.26*	5.15**
HRsub	4.36**	1.23	2.10*

* Significant at 0.05 level (2.069)
** Significant at 0.01 level (2.807)

From Table 2 and 3 it is found that LVEDV, SV, EF and FS were significantly improved where as RHR and HRsub were significantly decreased after conditioning. SV, FS were decreased and RHR was significantly improved following 4 weeks of rest, in the reconditioning phase only RHR and HRsub were significantly decreased.

The response of training stimulus, which may be called volume stress, might have facilitated cardiac hypertrophy through an increase in the size of the ventricular cavity. Due to conditioning, the subjects of the study 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 was evident with increased SV of the experimental subjects, which needs a powerful contraction and significant expansion of the left ventricle. The significant improve of

LVEDV of the experimental subjects may be due to powerful stroke output and resting bradycardia. The findings of LVEDV were found to be in agreement with the views of DeMaria.

Ejection Fraction of the subjects ware significantly changed following the training. EF is dependent upon, LVEDV and LVESV of an individual. As LVEDV was significantly increased and LVESV was slightly decreased after conditioning, the difference of LVEDV and LVESV increased significantly. In the resting state the EF of an individual is related to the contractility of the left ventricular muscle. As FS of the subjects increased significantly, the contractility of left ventricular muscle increased, which might have facilitated the significant increment of EF of the experimental subject. The EF is also related to FS, SV and RHR of the subjects. In this case FS and SV increased significantly in the subjects. So, the significant increment of EF of trainees may be due to the adaptation to training. The findings of this parameters was found to be in agreement with the views of Estorri (1986), Vieweg (1975) and Dressendorfer (1986).

In general, the SV increases significantly following endurance training and thus endurance athlete have an increased ventricular cavity, so as to allow more blood to fill the ventricle during diastole, provided training duration is of optimum span, thus resulting in a increased Stroke Volume. But in the case of observation made by the scholar increased Stroke Volume following conditioning may due to increased myocardial contractility. The increased in EF and FS of the subjects following conditioning might have facilitated SV increment. However, more recently it has been shown that without increasing diastolic volume, a stronger contraction could, as much as, double the SV by more completely emptying the ventricle, thus myocardial contractility is a key factor for increase the SV of an individual. The experimental subjects showed a significant increased in FS after conditioning period, which indicate a significant increase in myocardial contractility and thus, an adaptive increase of SV and decrease of RHR and HRsub were evident the conditioning protocol. In the deconditioning phase SV and FS decreased and RHR significantly improved because of the withdrawal of conditioning loads. Furthermore, only RHR and HRsub were significantly decreased after reconditioning protocol, which indicated the bradycardia and propositional relationship of HRH and HRsub with training loads.

The findings of this parameters was found to be in agreement with the views of Schuster and Brode (1990), DeMaria et.al. (1978), Wolfe and co-workers (1979) and Gilbert et.al. (1977).

Conclusions:

Within the limitations of the present study the following conclusions may be drawn

1. There was no significant difference of LVESV between conditioning, deconditioning and reconditioning of adult males.
2. There was significant improvement of LVEDV between pre and post conditioning but remain unchanged after deconditioning and reconditioning of adult males.
3. SV was significantly improved following conditioning and decreased after deconditioning but remain unaffected following reconditioning.
4. EF was significantly improved following 8 weeks of conditioning and remains unaffected following deconditining and reconditioning of the subjects f the study.
5. FS was significantly improved following conditioning and decreased after deconditioning but remain unaffected following reconditioning.
6. RHR was reduced significantly following conditioning and improved after deconditioning and again decreased significantly after 6 weeks of reconditioning.
7. Hrsub was reduced significantly following conditioning and unaffected following deconditioning and again reduced significantly following 6 weeks of reconditioning.

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