

# Cardiac adaptation to endurance training in young adult

## Abstract

**Context:** Regular physical exercise is known to cause improvement of the cardiovascular function. This adaptation is studied here with the help of non-invasive methods. **Aims:** To evaluate morphological changes in heart by echocardiography, to see the effect of exercise on autonomic function, on aerobic power and to assess the sequence of changes. **Settings and Design:** Study comprises of 12-week duration and was done on the students of physical education. **Materials and Methods:** This study was a longitudinal study in which 100 subjects (51 male, 20.18 yrs $\pm$ 1.147, 49 female, 19.91 yrs $\pm$ 1.89) were assessed using electrocardiography, echocardiography and Queen's College Step test (for  $VO_{2max}$ ) within 7 days of admission to their college and re-examined after 12 weeks. **Statistical Analysis:** Paired t-test using Graph pad prism5 software. **Results:** Electrocardiographic evaluation was suggestive of significant decrease in heart rate, significant increase in RR interval and t-wave amplitude in cardiac leads in males and similar but not significant result in females. No significant change was found in left ventricular morphology and ejection fraction after exercise program. **Conclusions:** The results of this study suggest that the exercise training over a period of 3 months does not influence cardiovascular morphology, but causes changes in parasympathetic and sympathetic tone and improves aerobic power.

### Key words:

Echocardiography, electrocardiography, heart rate,  $VO_{2max}$

## Introduction

Cardiac enlargement in athletes was recognized by the end of 19<sup>th</sup> century through the percussion of the chest in cross country skiers. This was later confirmed by radiography and evidence of necropsy. The advent of echocardiography and electrocardiography (ECG) allowed investigator to gain better insight into the changes produced in athletes. The effects of endurance training have been extensively studied in athletes, but longitudinal studies of exercise-induced cardiac changes in normal patients are limited. In the foreign countries lot of work had been carried out to assess capability of athletes and marathon runners and to study cardiac changes as a result of physical training in them.<sup>[1,2]</sup> In our country, this studies are limited hence we endeavoured to undertake a longitudinal study to assess the effect of exercise on cardiovascular system in students of physical education.

Aims and objective of the study were to evaluate morphological changes in heart by echocardiography, to see the effect of exercise on autonomic function, on aerobic power and to assess the sequence of changes.

## Materials and Methods

This was a longitudinal study in which 100 subjects (51 male, 20.18 yrs $\pm$ 1.147; 49 female, 19.91 yrs $\pm$ 1.89) were evaluated within 7 days of admission to college and after 12 weeks of exercise and these findings were compared with previous findings. All the subjects had to undergo physical training and their F.I.T.T. (i.e., frequency, intensity, time and type of physical activity) was: Ten session/wk, 40 min/session, somewhat hard intensity (RPE scale),<sup>[3]</sup> 12-week duration, and dynamic exercise in form of running. Theses exercise per session were distributed as warm up for 10 min fairly light intensity (RPE scale),

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followed by running somewhat hard intensity (RPE scale), for 30 min.

Inclusion criteria were mainly healthy volunteer, age group between 17 and 22 yrs, and non-smokers. Smokers, subjects with history cardiac, respiratory and diabetics and subjects whose clinical finding were abnormal were excluded from the study.

For maximal oxygen consumption:-Queen's College Step test<sup>[4]</sup> was used. Subjects were instructed to step at the rate of 22 steps per min (females) or 24 steps per min (male) for 3 min. The bench or stool height is 16.25 inches. After exercise, the subject remains standing, waits for 5 second, and 15-second heart rate count is taken. The  $VO_{2max}$  (ml/kg/min) is predicted using this equation.

Males

$$\text{Predicted } VO_{2max} = 111.33 - (0.42 * \text{heart rate})$$

Females

$$\text{Predicted } VO_{2max} = 65.81 - (0.1847 * \text{heart rate})$$

For echocardiography:-PHILIPS i.e. 33, S5-1 - sector array transducer. Appointments were given to eight subjects daily. They were told to take lunch, avoid any physical exertion for 4-5 hours prior to the test and assemble in Physical Education College campus at around 1 p.m. from where they were taken to Department of Cardiology by vehicle, where their echocardiography was done by the doctor on duty.

Subjects were examined and conventional two-dimensionally (2D) guided M-mode recording were recorded. A variable frequency phased array transducer was used. All the conventional acoustic window were used, and usual plane of view (parasternal long and short axes; apical four-chamber view) were registered. The gain, depth compensation, and darkest gray scale were individually adjusted according to thoracic wall thickness, depth of cardiac chamber and positional relationship between cardiac chambers and the ultrasonic beam.

The transducer was placed in the fourth intercostal space at left sternal border and was adjusted. The transducer was directed posteriorly, slightly laterally and inferiorly so as to record the clear and simultaneous echoes from left ventricular wall and interventricular septum both. Cardiac dimensions were measured in M-mode echocardiogram combined B-mode from 2D images.

At rest, left ventricular end diastolic and end systolic diameters, interventricular septum and left posterior wall thicknesses were measured from the parasternal long and short-axis view, just below the mitral valve level according to the recommendations of the American Society of Echocardiography<sup>[5,6]</sup> To assess the objectivity of

echocardiographic readings, all recording were evaluated by two independent expert.

### Calculation

Ejection fraction = end diastolic volume – end systolic volume/end diastolic volume\*100

The Devereux modified American Society of echocardiography cube formula was used to calculate LV mass.<sup>[6]</sup>

$$LV \text{ mass} = 0.8(1.04 ([LVIDD+PWTD+IVSTD]^3-[LVIDD]^3)) + 0.6 \text{ g.}$$

For recording ECG:-Cardiart 108-T-MK-VI, a single channel, 12-lead electrocardiograph was used to record the ECG (BPL Manufacturing Company, BPL limited, Bangalore, India). Resting ECG was recorded in Physical Education College, in the morning session before the start of exercise.

The subject was told to lie comfortably in supine position on the bed. Any metallic object such as watch, rings, necklace and coins were removed. Clothes were removed in a manner to expose chest, legs, and forearm. The machine was connected to the main AC power supplier. The cardio jelly was applied to over an area 2 cm in diameter of respective positions. The machine was started. The ECG of each subject was recorded on a thermo-sensitive paper 50-mm width with a speed of 25 mm/sec. All ECGs were recorded in 12 standard leads. Care was taken to ensure consistency in positioning the electrode.

Approval for the above study was taken from institutional ethics committee.

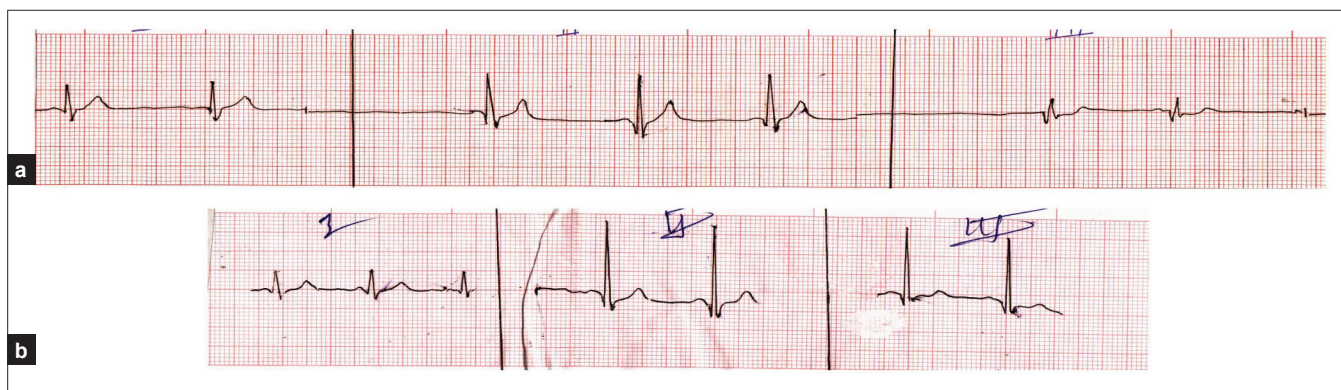
Then the data of the observation for all parameters were statistically analyzed by calculating mean and standard deviation. The data was analyzed using Graph pad prism5 software. Statistical difference between the data obtained in various groups was evaluated by paired t-test and *P*-value <0.05 was considered as statistically significant.

### Results

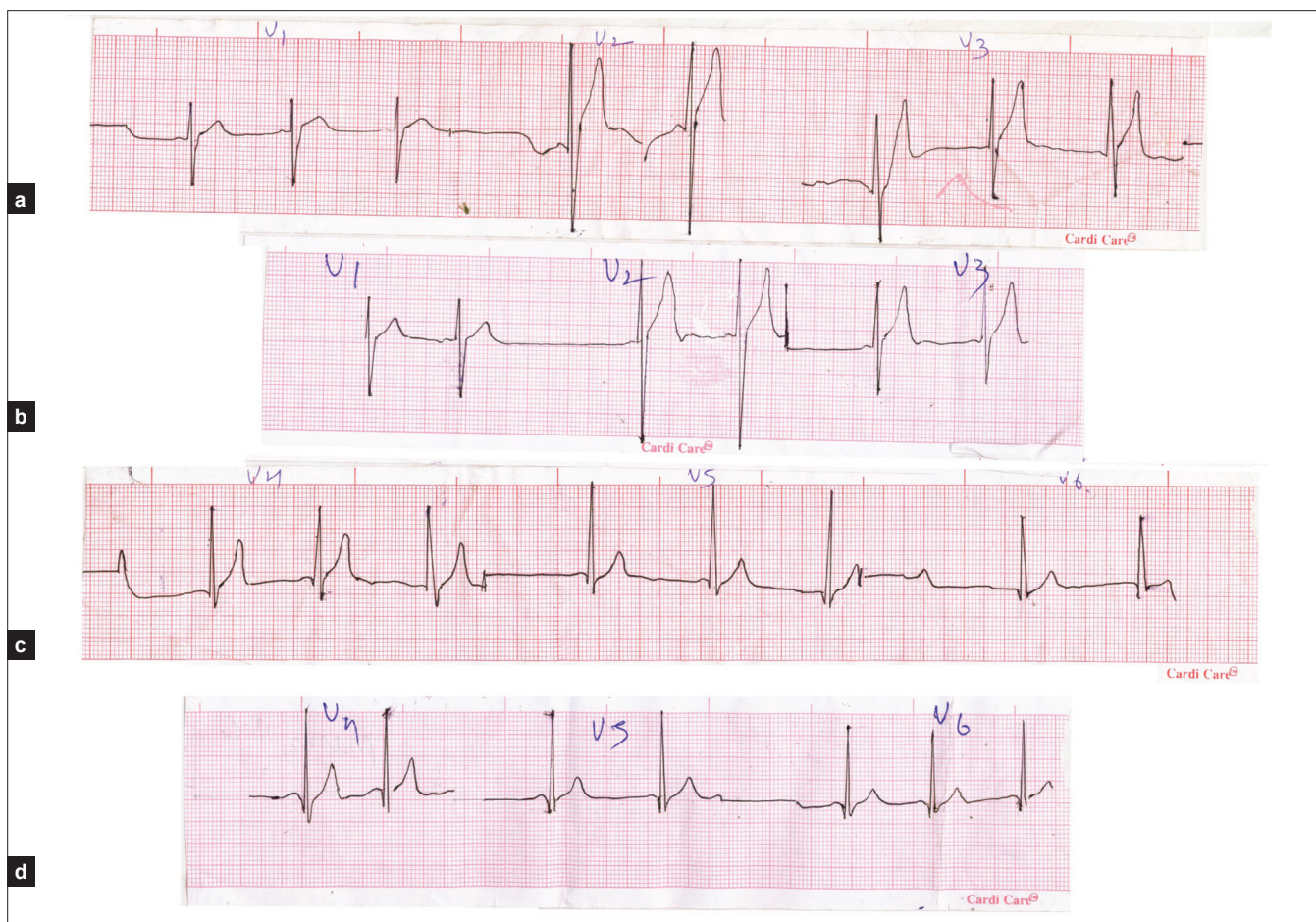
There was no significant change in anthropometric variables but there was significant improvement in aerobic power as assessed by  $VO_{2max}$  [Table 1].

There was significant decrease in heart rate, increase in RR interval and increase in T-wave amplitude in males, similar result was found in females, though they were not significant [Table 2, Figure 1].

In our study none of subject developed ST segment elevation; however, it persisted in the subject who initially were showing ST segment elevation. Peak T-waves was also found in some subject pre as well as post-exercise [Figure 2].



**Figure 1:** ECG showing decreased heart rate in bipolar leads (ECG below is pre-exercise while above is post-exercise)



**Figure 2:** ECG showing ST segment elevation and peak T-wave in pre-exercise as well as post-exercise (specially in v2 and v3 leads) (a and c post-exercise, b and d pre-exercise)

There were no significant morphological changes in heart in male as well as in females [Table 3].

### Discussion

There was significant decrease in heart rate, increase in RR interval and increase in T-wave amplitude in males, similar result was found in females, though they were not significant.

Heart rate and RR interval is having inverse relation with each other. Our finding of decrease heart rate in males is matching various longitudinal<sup>[1,2]</sup> and cross-sectional studies.<sup>[7-9]</sup> No significant change was seen in some studies.<sup>[10,11]</sup> Our finding in females is in accordance with some<sup>[10,12]</sup> and in contrast with some studies.<sup>[13,14]</sup>

Exercise training creates imbalance between the tonic activity of sympathetic accelerator and parasympathetic

**Table 1: Subjects characteristics**

Parameters	Pre-exercise male (n=51)	Post-exercise male (n=51)	P value	Pre-exercise female (n=49)	Post-exercise female (n=49)	P value
Age (yrs)	20.18±1.147	=20.18±1.147	P>0.05	19.91±1.89	=19.91±1.89	P>0.05
Height (m)	1.701±0.04568	=1.701±0.04568	P>0.05	1.581±0.05442	=1.581±0.05442	P>0.05
Weight (kg)	60.85±5.873	60.63±5.516	P>0.05	52.15±6.495	52.51±5.465	P>0.05
BSA (M <sup>2</sup> )	1.70±0.081	1.70±0.077	P>0.05	1.51±0.095	1.52±0.085	P>0.05
VO <sub>2max</sub> (ML/KG/M)	45.74±6.87	51.41±5.96***	P<0.001	38.47±3.38	41.50±2.57***	P<0.001

Values are Mean±SD; \*\*\*P<0.001; Very highly significant; Comparison between pre- and post-exercise

**Table 2: ECG changes**

Parameters	Pre-exercise male (n=51)	Post-exercise male (n=51)	P value	Pre-exercise female (n=49)	Post-exercise female (n=49)	P value
P-wave amplitude (mm)	1.12±0.32	1.19±0.40	P>0.05	1.14±0.35	1.24±0.43	P>0.05
P-wave duration (sec)	0.070±0.018	0.0713±0.021	P>0.05	0.077±0.0126	0.08±0.008	P>0.05
PR interval (sec)	0.154±0.028	0.160±0.031	P>0.05	0.176±0.024	0.179±0.02	P>0.05
QRS duration (sec)	0.058±0.013	0.058±0.008	P>0.05	0.044±0.008	0.045±0.009	P>0.05
RV1 + SV6(RVH) (mm)	5.70±3.24	6.03±3.76	P>0.05	5.77±2.7	6.28±3.66	P>0.05
RV6 + SV1(LVH) (mm)	25.41±7.13	25.53±7.58	P>0.05	22.32±5.26	24.67±6.76	P>0.05
ST segment (sec)	0.10±0.033	0.117±0.02	P>0.05	0.118±0.026	0.122±0.027	P>0.05
ST interval (sec)	0.32±0.031	0.323±0.03	P>0.05	0.336±0.025	0.337±0.024	P>0.05
T-wave amplitude (II) (mm)	3.17±1.21	3.43↑±1.3	P>0.05	3.12±0.66	3.34↑±0.59	P>0.05
T-max wave amplitude (V) (mm)	8.15±3.46	9.55↑±3.45*	P<0.05	3.92±2.01	4.16↑±1.73	P>0.05
T-wave duration (sec)	0.218±0.046	0.212±0.04	P>0.05	0.217±0.03	0.214±0.03	P>0.05
RR duration (sec)	0.89±0.178	0.991↑±0.15*	P<0.05	0.86±0.15	0.88↑±0.11	P>0.05
Heart rate (beats/min)	70.41±14.49	61.85↓±11.88*	P<0.05	71.86±12.95	68.53↓±9.08	P>0.05
QT interval (sec)	0.37±0.03	0.38±0.03	P>0.05	0.38±0.02	0.38±0.02	P>0.05
QTC (sec)	0.406±0.05	0.385±0.04	P>0.05	0.413±0.03	0.408±0.027	P>0.05
QRS axis (degree)	67.88±29.14	61.29±30.94	P>0.05	55.97±23.27	58.10±22.23	P>0.05
T-axis (degree)	45.41±23.63	44.49±22.14	P>0.05	44.63±7.79	46±12.99	P>0.05

Values are Mean±SD; \*P<0.05; Significant change; ↑ – Increase; ↓ – Decrease comparison between pre- and post-exercise

**Table 3: Result of echocardiographic dimensions**

Parameters	Pre-exercise male (n=51)	Post-exercise male (n=51)	P value	Pre-exercise female (n=49)	Post-exercise female (n=49)	P value
LVPWs (cm)	1.44±0.32	1.38±0.25	P>0.05	1.34±0.19	1.38±0.25	P>0.05
LVIDs (cm)	2.88±0.34	2.88±0.37	P>0.05	2.38±0.37	2.37±0.37	P>0.05
IVSs (cm)	1.305±0.32	1.305±0.25	P>0.05	1.28±0.207	1.271±0.296	P>0.05
LVPWD (cm)	1.041±0.1615	1.023±0.165	P>0.05	0.97±0.16	0.97±0.17	P>0.05
LVIDd (cm)	4.45±0.38	4.47±0.40	P>0.05	4.108±0.36	4.12±0.44	P>0.05
IVSd (cm)	0.98±0.14	0.99±0.15	P>0.05	0.87±0.10	0.90±0.12	P>0.05
EF (%)	61.90±9.76	63.94±8.12	P>0.05	66.91±7.75	67.24±8.77	P>0.05
LV MASS (gm)	154.44±30.21	154.70±31.36	P>0.05	118.51±19.17	122.86±24.14	P>0.05

Values are Mean±SD; No significant change; Comparison between pre- and post-exercise

depressor neuron in favour of greater vagal dominance - a response mediated primarily by increased parasympathetic activity and small decrease in sympathetic discharge.<sup>[15,16]</sup> Training also decreases the intrinsic firing rate of sinoatrial pacemaker tissue.<sup>[17]</sup>

P-wave morphology i.e., atrium morphology changes and QRS duration changes are in accordance with<sup>[8,18]</sup> but in contrast with some studies.<sup>[9,19]</sup> Bjornstad *et al.*,<sup>[9]</sup> found significant changes in both parameters in top athletes but not in athletic students.

PR interval finding are similar to our finding in some<sup>[18]</sup> but in contrast in others.<sup>[13,20]</sup>

Our finding of voltage criteria LVH is not in accordance with some studies.<sup>[8,18,19]</sup> George *et al.*, found little ECG evidence of LV hypertrophy evidence in both female power athlete and female endurance athletes.<sup>[13]</sup> Wieling *et al.*, in their longitudinal study found LVH criteria significant for freshmen oarsmen but not in senior oarsmen.<sup>[21]</sup> Bjornstad *et al.*, found significant LVH voltage criteria in athletic student but not for top athlete,<sup>[9]</sup> So the chest wall

morphology may be playing important role like the increased magnitude of QRS voltage secondary to reduced chest wall thickness.<sup>[19]</sup>

T-wave amplitude changes were matching some authors<sup>[8,19]</sup> but in contrast to some.<sup>[9]</sup>

In our study none of the subject develops ST elevation and it persisted in students who were initially showing ST elevation. ST elevation may occur as normal variant.<sup>[20]</sup> According to Bianco *et al.*,<sup>[22]</sup> early repolarization is almost always the rule in athletes but it is also frequent in sedentary males. No other repolarization changes were seen.

Corrected QT interval also does not change significantly which is in accordance with some studies<sup>[9,18]</sup> but in contrast to some authors.<sup>[8,23]</sup> Bjornstad *et al.*, demonstrated prolonged QTc in athlete compared to control inspite of lower heart rate in the athletic group demonstrate that real QTc prolongation exists in athletes.<sup>[23]</sup>

Our finding of QRS axis is matching several authors<sup>[9,18,19]</sup> but in contrast to some.<sup>[8]</sup> T-axis finding is not in contrast with some<sup>[8]</sup> but in agreement with some.<sup>[9]</sup> In Bjornstad *et al.*, study, athletes had more often T-wave axis between +30 and -180.<sup>[23]</sup>

In all above findings of ECG which we have discussed, exercise duration play an important role. Studies where physical conditioning stimulus was in years in contrast to our study, more changes were seen. According to us, bradycardia and increased T-wave amplitude are the initial ECG change but out of them, which occur first, we can not comment. Frequency and intensity also play role which will be dealt in discussion on echocardiography changes.

Also in females with 3 months of duration, no significant change was observed. In Pellica *et al.*, study, ECG pattern differed greatly with respect to gender and vast majority of female athletes showed normal ECGs.<sup>[24]</sup> According to Qi Fu and Benjamin D. Levine,<sup>[25]</sup> there are physiological and morphological gender difference exists in humans. It is likely that certain gender-specific factor such as difference in some hormonal level, menstrual cycle variability, and physical characteristics (primarily cardiac size and function) may influence the cardiovascular response to exercise in women. In females according to us exercise should be long duration and with hard intensity.

### Echocardiographic parameters

In present study as the students were doing predominantly dynamic exercise, we expect eccentric LVH, but there was no change in left ventricular dimension.

No change was found in left ventricular posterior wall thickness in systole and diastole by some (LVPWs and

LVPWd)<sup>[1,12,26,27]</sup> whereas some studies noted significant increase.<sup>[2,11,21]</sup>

Left ventricular internal diameter at end diastole (LVIDs) and at end systole (LVIDd) does not change significantly in several studies<sup>[1,2,11,12]</sup> while in some it changed significantly.<sup>[21,27-30]</sup> Inter ventricular septal thickness at end diastole (IVSd) and at end systole (IVSs) remains unchanged<sup>[1,27]</sup> but increased in some studies.<sup>[2,11,30]</sup>

EF%: Ejection fraction indicates the contractile status of heart. Some got improvement in EF<sup>[18,30]</sup> while some found no improvement in ejection fraction.<sup>[2,10,23]</sup> Bjornstad *et al.*, concluded that the systolic function at rest is similar in age-matched top athletes, athletic students and control.<sup>[9]</sup>

LV MASS: Wolfe *et al.*, found no significant change<sup>[29]</sup> while some got significant improvement in LV mass.<sup>[2,11,29,30]</sup>

If we compare all studies according to F.I.T.T., type of activity i.e., mode was predominantly dynamic as in our study, frequency is certainly more in our study, time is more or less equal, but intensity is less, so it may be the factor for no significant change. Intensity of exercise in our study was more or less equal to studies who got similar results comparable to us. So the exercise of short duration must be accompanied by very hard intensity for morphological changes to occur.

Inverse correlation between left ventricular size and heart rate was shown,<sup>[31]</sup> so bradycardia may be triggering factor for hypertrophy. Due to lower heart rate more time is allowed for ventricular filling for accommodating more volume of blood in the left ventricle, which causes stretching of myocardial fibres, this increases the end diastolic diameter of the ventricles. Increase in the size of left ventricular chamber increases the reserve capacity of chambers as well as increase force of contraction of left ventricle to pump out more volume of blood during each stroke.

### Limitations of the study

To know the minimum duration, assessment of the subjects should have done multiple times and then those readings should have compared. In the future, we would recommend multiple longitudinal studies, with not only varying duration but also with varying intensity, so that minimum duration and intensity for cardiorespiratory changes would be known.

We had taken precautions to avoid intraobserver and interobserver variations especially during echocardiography, still there is possibility of these variations.

Although we had got very few changes with 3 months of training, we can conclude that 3 months of training, is helpful in improving aerobic power of the subjects along

with the changes in parasympathetic and sympathetic tone but does not alter left ventricular morphology. Bradycardia and increased T-wave amplitude are the first changes to occur in ECG. For morphological changes to occur, physical training must be associated with very hard intensity if the duration of exercise program is short.

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