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# Effect of Maximal Exercise on the Heart Rate, Blood Pressure and Level of Cardiac Remolding in Elite Athletes

#### Hafeezullah Wazir Ali<sup>1</sup>, Abdullah Saleh Alanazi<sup>2</sup>, Mohammad Aslam<sup>3</sup>, Muhammad Mazhar Hussain<sup>4</sup>

<sup>1</sup>MBBS, MPhil, FCPS, Department of Physiology, Northern Border University, Ar'ar, Saudi Arabia
 <sup>2</sup>MBBS, SBFM, University health center, Northern Border University, Ar'ar, Saudi Arabia
 <sup>3</sup>MBBS, MPhil, PhD, FCPS, Professor of Physiology, Shifa Tameer e Millat University, Islamabad, Pakistan
 <sup>4</sup>MBBS, MPhil, FCPS, Professor and Head of Physiology Department Vice Principal Fazaia Med College, Air University, Islamabad

#### ABSTRACT

**Aim / Objective:** The purpose of this study was to determine how intense exercise effect the blood pressure, heart rate and the extent upto, which the cardiac remolding may occur in elite athletes. **Location of Study:** This study was conducted in the Department of Physiology, Army Medical College, along with the Armed Forces Institute of Cardiology (AFIC) in Rawalpindi.

**Methodology:** There were 40 control participants and 40 elite endurance athletes. Clinical tests were performed on all the subjects to rule out an underlying medical condition or disease. Auscultatory method was used to measure the heart rate before exercise and immediately after exercise, Similarly the blood pressure was examined before and within 3 minutes after the physical activity. The left ventricular end-diastolic internal diameter (LVIDd), diastolic posterior wall thickness (PWTd), diastolic interventricular septal thickness (IVSTd), and left ventricular mass (LVM) were measured by echocardiography and determined using the Devereux formula. Before breakfast, the participants engaged in maximum physical activity on an electronic ergometer cycle until they were exhausted.

**Results:** It was observed that when compared to the control group, the heart rates and blood pressure of athletes were noticeably lower. In contrast, LVIDd, IVSTd, PWTd, and LVM were higher in athletes than in healthy controls...

**Conclusion:** The long term athletic training causes remodeling of the heart with increased dimensions of the left ventricle which reduces the heart rate and blood pressure in endurance trained athletes.

#### ARTICLE DETAILS

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**KEYWORDS:** The heart of trained athletes, left ventricular remodeling, Blood pressure and heart <u>http</u> rate.

#### INTRODUCTION

Having a healthy circulatory system is essential for supplying enough blood to fulfill the thermoregulatory requirements and metabolic demands of exercise. During 1890s, by simply auscultating and percussing the chest, doctors in Sweden and the USA found increased heart dimensions in elite athletes [1]. Which was followed by the first reports of athletic bradycardia in marathon runners from the Boston [2]. Subsequently, with improved imaging technologies, many studies revealed the structural and functional adaptations observed in the human heart in response to athletic activities. The endurance athletes have large remodeled hearts, with large ventricular volumes, modest wall thickening, and a low relative wall thickness associated with reductions in resting heart rate [3]. It has been recognized that regular endurance exercise training promotes healthy physical activity associated with

multiple benefits and a longer life expectancy [4]. The most dramatic rise in left ventricular mass is observed in the first few months of training, and it is closely correlated with the amount of exercise performed. This rise in cardiac volume improves the heart's flexibility, which leads to a higher stroke volume [5], on the other hand 'bed rest' leads to the reversal of these changes and reduction in volume in two weeks[6]. Heart rate and arterial blood pressure decreases indicate that the degree of adaptation depends on how hard the training program is used. Notably, the maximal heart rate stays unaffected by this [7]. Bilateral dilatation of the heart chambers, supernormal diastolic function, and improved arterial vasomotor function are the hallmarks of exercise-induced cardiovascular remodeling (EICR) [8]. Though there is continuous debate over physical exercise, these cardiac adaptations-which include both structural and functional changes—are usually regarded as benign [9]. Although there might be controversies about the physical activities, There is scientific proof that physical activity, regardless of intensity,. is much better for health than leading a sedentary lifestyle of the individual[10]. The prolonged response to intense exercise on heart includes phenotypic and physiologic adaptive changes that improve myocardial ability to meet oxygen demands [11]. The heart with these adaptive changes lead to the lowering of the heart rate (HR) and arterial blood pressure (BP). These cardiac remodeling (CR) and hemodynamic adaptations are physiological which result due to the increased demands of blood flow and oxygen during exercise [12]. The studies have proved an inverse relationship between physical activity and blood pressure (BP) pointing that exercise lower the systolic and diastolic BP. However, the maximum HR in athletes remain unchanged during or after exercise with protective effects on their cardiovascular health [13]. However, light intensity exercise may contribute to the sustained (up to 19 hours) decreased ambulatory systolic BP during the day and night compared to the moderate intensity [14]. Therefore, the European Society of Cardiology and the American Heart Association recommend exercise for the prevention and therapy of hypertension [15]. The effect of regular physical exercise on reduction in resting BP is not as acute as the reduction detected in HR. In non-exercising sedentary hypertensive people, clinically significant decreases in BP can be achieved with moderate increases in physical activity above sedentary levels [16]. Many studies have established the long-term impact of physical activities in athletes on left ventricle (LV) size, wall thickness, and it's functioning [17]. Cardiac adaptations in athletes mainly depend upon the characteristics, intensity, and cumulative duration of training protocols, with a 'dose-effect' relation [18]. The endurance training on dynamic exercise has been associated with a substantial increase in cardiac output and reduction in peripheral vascular resistance and BP [19]. Dilation of the LV is common and should be considered as a normal finding in

cardiac adaptation. Mild thickening of LV walls, with or without left ventricular volume increase, may develop in cardiac remodeling. Balanced LV wall thickening and chamber dilation and a relative wall thickness is common cardiac adaptions in endurance trained athletes. Similarly it has been documented that inter-ventricular septal thickness increases in athletes, accompanied by the significantly higher left and right ventricular mass index and larger left atrial chamber [20].

#### **OBJECTIVE**

The present study was carried out to determine the cardiac adaptive changes in endurance trained elite athletes and evaluated the effects of maximal exercise on heart rate, arterial blood pressure and echocardiographic changes in elite athletes and sedentary groups.

#### METHODOLOGY

This project was carried out in the Department of Physiology, Army Medical College, along with the Armed Forces Institute of Cardiology (AFIC) in Rawalpindi. Overall 80 people were included in the study: 40 were top endurance athletes, while the remaining 40 acted as controls by being inactive but from the same work and living conditions.

#### SAMPLING TECHNIQUE

In this study, the non-probability purposive sampling was used to specifically choose individuals based on attributes that were significant to the research goals. This strategy was adopted in order to guarantee a focused representation of both healthy inactive controls and top endurance athletes, which is consistent with the study's emphasis on the cardiovascular changes linked to varying degrees of physical exercise. In the context of this investigation, the inclusion of elite endurance athletes and sedentary controls was deliberate as it aimed to discern distinctive cardiac features associated with varying physical activity levels, accurately representing the populations of interest and enhancing the study's internal validity. Purposive sampling enabled us to select individuals who meet this specific criterion.

#### INCLUSION CRITERIA

The study comprised of two groups:

1. **GROUP-I:** this group contained Elite Endurance Athletes (n=40) who were recruited from Pakistan Army. All the recruited members were between the age of 18 to35 years old with a BMI of 19-22 kg/m<sup>2</sup>.

2. GROUP-II: This group Included the healhy sedentary controls with the same age and BMI as that of group-I (n=40) **EXCLUSION CRITERIA:** Based on their medical history, physical examination (blood pressure  $\leq 140/90$  mm Hg), and echocardiography, all participants were passed through a clinical examination to rule out lung and cardiovascular

disorders. A positive family history in this study includes occurrences of coronary artery disease, cardiomyopathy, severe arrhythmias, or any other debilitating cardiovascular illness.

#### STUDY DESIGN

To evaluate and compare cardiovascular parameters in top endurance athletes and inactive controls at a specific moment in time, a cross-sectional comparative research design was selected. This allowed for insights into the immediate differences and correlations between the two groups. This method provides useful snapshot insights into potential correlations and disparities while enabling efficient evaluation of the research variables

#### DATA COLLECTION PROCEDURE

- 1. General physical examination:
  - a) Height (m) and Weight (kg)
  - b) Body mass index (BMI):  $BMI = Weight in Kg \div Height in meters^2 (Kg/m^2)$
  - c) Heart Rate: Heart rate was measured by auscultatory method at mid clavicular line in left fourth intercostal space before and within 3 minutes after exercise.
  - **d) Blood Pressure:** Arterial blood pressure (mm Hg) was measured after 5 minutes of rest in a seated position by mercury sphygmomanometer.

#### 2. M-mode echocardiography:

Toshiba Power Vision 6000 echocardiograph with 3.7 MHz transducer was used for echocardiographic and Doppler tests. The subjects' left lateral location was set at a 45° angle. An integrated M-mode and two-dimensional examination was carried out to measure the diastolic interventricular septal thickness (IVSTd), left ventricular posterior wall thickness (PWTd), and left ventricular end-diastolic cavity dimension (LVIDd) in order to prevent trabeculations in the wall thickness measurements. Initially, a parasternal long-axis view of a twodimensional targeted M-mode recordings was acquired. Next, using expanded two-dimensional images, the thickness of the septal and posterior walls was measured in the parasternal longaxis view between the papillary muscle and the tips of the mitral valve. The real thicknesses of the posterior wall and septum were acknowledged to be represented by smaller figures from two-dimensional or M-mode measurements. Left ventricular mass was (LVM) calculated by using the Devereux formula [21].

LVM= 0.8 [1.04 (LVIDd + PWTd + IVSTd)  $^{3}$ -(LVIDd)  $^{3}$ ] + 0.6 g

3. **Bicycle Exercise Test:** The participants completed a maximal exercise test until they were exhausted while using an electronically controled bicycle ergometer. Starting with a 50W load, the exercise was then increased to a 10W load every five minutes until exhaustion. Heart rate and arterial blood pressure were measured during the exercise test. The individuals' blood pressure was taken at three points firstly after a five-minute supine rest period than during the peak of the activity, and lastly right before they were allowed to halt cycling [22].

#### 4. STATISTICAL ANALYSES

The collected data was analyzed by an in-depth analysis using the computer program SPSS Version-22. For each relevant variable, the means and standard deviations ( $\pm$ SD) were computed using descriptive statistics. The difference in means between elite endurance athletes and inactive controls was then determined using inferential statistics, which used a paired student's t-test with a 95% confidence level. The study employed a rigorous statistical technique to identify significant differences and associations between the two groups, so assuring a comprehensive and precise interpretation of the data.

#### RESULTS

The major goal of this study was to understand the connection between blood pressure, heart rate, and wall thickness of the chambers of heart. Interestingly, as compared to controls, the heart rates and blood pressure of the athletes were noticeably lower. In contrast, as compared to controls, top athletes showed higher mass, diastolic posterior wall thickness, end-diastolic internal diameter, and diastolic interventricular septal thickness in the left ventricle. A thorough summary of the study outcomes is offered in Tables 1 to 3.

**Table -1:** The physical characteristics of top athletes and controls, such as mean age and BMI, are shown in table 1. The statistical analysis did not show any significant differences between these parameters obviously because the controls were carefully matched,.

**Table-2** The heart rate, diastolic blood pressure (DBP), and systolic blood pressure (SBP) of top athletes and controls before and after exercise are presented in table 2. The mean heart rate of the post-exercise controls was greater than that of the pre-exercise controls. Athletes, on the other hand, continuously showed lower heart rates than sedentary controls. Additionally, athletes' post-exercise mean heart rates were greater than their pre-exercise heart rates, but they were lower than the heart rates of controls following the same activity. SBP and DBP readings showed identical patterns. In comparison to the controls, the heart rate and blood pressure of top athletes were found to be considerably lower. The results of the paired sample 't' test as shown in table 2 showed statistical significance (p < 0.001 and p < 0.008), suggesting that top athletes had significantly lower blood pressure and heart rates than controls.

**Table-3** Table 3 provides additional information about the left ventricular parameters, including left ventricular internal diameter in diastole (LVIDd), diastolic inter-ventricular septal

thickness (IVSTd), diastolic posterior wall thickness (PWTd), and left ventricular mass (LVM) in top athletes and controls. The paired sample 't' test revealed that all these characteristics were statistically substantially higher in elite athletes than in controls (p < 0.001 and p < 0.008), suggesting considerable anatomical changes in elite athletes' hearts.

Variables	Controls (N=40)	Elite Athletes (N=40)
Age (years)	22.63	22.49
	±2.89	$\pm 2.78$
BMI	20.41	20.86
	±2.56	$\pm 0.96$
Height (m)	1.74	1.76
	$\pm 0.45$	$\pm 0.87$
Weight (kg)	64.48	63.58
	$\pm 2.40$	$\pm 4.47$

Table 1:	Comparison	of the ages a	and BMI b	between	controls and	elite athlete	s
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The values of age and BMI are given as mean ±SD

None of the differences is statistically significant

## Table 2: Comparison of diastolic, systolic and mean arterial blood pressure and heart rate, before and after exercise in controls and athletes

Subjects (n=80)	Variables	Controls (n=40)	Athletes (n=40)
Pre-exercise	Heart rate	75.19	55.96 **
		±3.16	±3.47
	BP Systolic (mmHg)	117	107.25 **
		±5.12	±5.01
	BP Diastolic	76.55	58.19**
	(mmHg)	±5.25	±2.56
	Mean Arterial blood pressure (mmHg)	96.00	83.58**
		±21.05	±24.86
Post-exercise	Heart rate	136.00	110.82**
		±6.87	±8.27
	BP Systolic (mmHg)	139.32	121.36*
		±9.04	±5.16
	BP Diastolic (mmHg)	70.68	56.14**
		±5.19	±3.76
	Mean Arterial blood pressure (mmHg)	105	92.75**
		±35.06	±37.86

The values are given as mean  $\pm$  SD

The data compared between athletes and controls before and after exercise

\* The difference is statistically significant at p<0.008

\*\* The difference is statistically significant at p<0.001

Parameter	Controls	Elite Athletes
	( <b>n=40</b> )	( <b>n=40</b> )
LV end-diastolic internal diameter (LVIDd) (mm)	39.68	54.73**
	±2.28	±1.59
Diastolic inter-ventricular septal thickness (IVSTd) (mm)	8.52	9.72**
	±0.37	±0.89
Diastolic posterior wall thickness (PWTd) (mm)	8.04	8.63*
	±0.32	±0.73
Left Ventricular Mass (LVM) (gm)	102.5	176.36**
	±11.01	±22.86

Table 3: Comparison of left ventricular echocardiographic variables between elite athletes and controls

The values are given as mean  $\pm$  SD

\* The difference is statistically significant at p<0.008

\*\* The difference is statistically significant at p<0.001

#### DISCUSSION

The most of the studies in highly trained athletes have been conducted on Caucasian populations; therefore, the effect of racial variation on heart have not been yet established. No such type of work has so far been reported in Pakistan. In present study the heart rate (HR) and blood pressure (BP) of athletes were significantly less than the controls, while left ventricular end-diastolic internal diameter (LVIDd), diastolic inter-ventricular septal thickness (IVSTd), diastolic posterior wall thickness (PWTd), and left ventricular mass (LVM) were greater in athletes than the sedentary healthy controls. Due to the endurance exercise the blood pressure (BP) decreases most probably due to decrease in sympathetic vasomotor control and/or due to increased venous return. Similarly, heart rate (HR) also decreases probably due to same reasons. It was suggested by the experts that physical activity might have beneficial effects on BP. Until recently Published metaanalyses have also confirmed the efficacy of physical activity in lowering BP in athletes as compared to their sedentary counterparts. In present study systolic blood pressure (sBP), diastolic blood pressure (dBP) and HR were measured before exercise and just after maximal exercise. Their means values were calculated and compared to sedentary controls and the means values of HR, dBP and sBP were compared with pre and post exercise states in endurance athletes. In present study, the post exercise HR was significantly more in sedentary controls (p<0.001) as were sBP and dBP. Post exercise mean values of same parameters were increased in elite athletes as well, although this increase was substantially less as compared to that seen in sedentary controls. Almost same findings have been reported by other workers as well. Like Pentikäinen et al calculated the mean HR as  $60.0 \pm 8.9$  per minute, the sBP as  $114.3 \pm 9.7$  mm Hg, the dBP as  $64.1 \pm 7.1$  mm Hg [23]. Deus et al found the HR;  $58.94 \pm 12.75$  beats/min; (P<0.001). Similarly sBP and dBP were significantly reduced (P<0.001) [24]. Huang et al calculated (mean  $\pm$  SD) HR to be  $60.88 \pm 2.65$  beats/min (*P*<0.001), systolic blood pressure  $110.79 \pm 1.85$  (*P* = 0.049) diastolic blood pressure  $63.79 \pm 1.99$  (P < 0.001) [25]. Lee et al reported that stroke volume was significantly higher while heart rate was lower in master athletes compared with aged-matched sedentary subjects. He found that resting HR was lower 58.62 $\pm$ 8.38 per minute (P < 0.001) during the intensive training phase in elite athletes [26]. Santoro et al calculated that resting heart rate was 56.3±6 beats/minute in elite athletes as compared to sedentary controls  $(73.3\pm5.7 \text{ beats/min; } P<0.001)[27]$ . Similarly Yahia M in 2019 found out that athletes had significant lower resting heart rate in comparison with control participants (56.5±10 versus 63.5±7.3 per minute, p=0.002) [28]. This value is comparable to the results of our study. Churchill et al recorded the same heart rate ( $58\pm9$  per minute) in elite female and male soccer players of US Soccer national teams [29].

Several studies have shown that athletes generally experience a decreased mortality, and that endurance athletes in particular have an increased longevity. Many studies have been conducted in the last 20 years, particularly in the last 5 years, to investigate how long-term endurance exercise impacts the size and mass of the heart. It has been established that consistent training in endurance sports is linked to a physiological increase in the heart's mass and volume [30]. According to the current study, athletes had higher LVIDd values than sedentary individuals do. The dimensions of the left ventricle end-diastolic cavity (LVIDd) in top athletes were found to be around 54.63 mm±1.79 mm in elite athletes, This value was substantially greater than that of the sedentary controls, who had an estimated value of 40.63 mm  $\pm$  2.08 mm.The same pattern with an ; LVIDd of  $56 \pm 5.6$  mm in endurance trained athletes was noted by D'Andrea et al [31]. Similarly according to Beaumont et al., athletes had higher LV mass (72 g), stroke volume (13.59 mL), posterior wall thickness (1.20 mm), interventricular septal thickness (1.23 mm,), and LV end-diastolic diameter (3.65 mm) (all p < 0.01) [32]. These figures closely match the

study's current data. This dilation of the ventricle is most likely an extreme physiologic response to intense physical conditioning when there exists no systolic dysfunction. In our investigation, we discovered that top athletes had considerably higher diastolic posterior wall thickness (PWTd) and diastolic interventricular septal thickness (IVSTd) as compared to controls[33]. The average (±SD) PWTd and IVSTd values of our top athletes showed a noticeable rise; however, after examining each case separately, it became clear that some athletes had normal IVSTd and PWTd values (mean IVSTd in athletes was 9.86 
0.89 mm versus 8.56 0.37 mm; p<0.001). The average  $(\pm SD)$  PWTd and IVSTd values of our top athletes showed a noticeable rise; however, after examining each case separately, it became clear that some athletes had normal IVSTd and PWTd values (mean IVSTd in athletes was 9.86 
0.89 mm versus 8.56 ±0.37 mm; p<0.001). This may be the result of adaptation to training-induced hemodynamic overload. In this investigation, the mean PWTd was determined to be  $8.63 \pm 0.73$ mm, a considerably greater value than the mean PWTd of controls which was  $8.04 \pm 0.32$  mm (p  $\leq 0.008$ ). Pellicia et al. observed similar results in their research of 40 male professional athletes, measuring the PWTd at 9.3±1.4mm, which is similar to the level we observed in this investigation[34]. Additionally, PWTd had been evaluated in a study by De Bosscher et al. which showed that 8.7% had an LV wall thickness of >13 mm and 51.4% had a dilated LV of >62 mm (p < 0.001). Given that Caucasian people had larger body surface areas than Asian populations, these high numbers are most likely the result of racial differences.

To sum up, our well-researched work, it offers important new understandings into the cardiac adaptations of high-level athletes performing prolonged endurance training. When combined with increased diastolic posterior wall thickness (PWTd) and interventricular septal thickness (IVSTd), the measured increase in left ventricular end-diastolic cavity dimensions (LVIDd) in athletes indicates notable structural changes brought on by rigorous athletic conditioning. The physiological changes in heart size and mass that top athletes exhibit are a complex reaction to the hemodynamic demands of extended endurance training. These results are consistent with previous research and provide credence to the idea that consistent endurance training modifies heart shape in a meaningful way.

Even though our study offers thorough information on the cardiac characteristics of top athletes, more research is necessary to fully understand the functional and long-term effects of these changes. It is essential to comprehend the complex interaction between cardiac morphology and continuous endurance exercise in order to optimize training regimens and protect athletes' cardiovascular systems.

#### CONCLUSION

Long-term athletic training results in remodeling of the heart, enlarging the left ventricle, which lowers blood pressure and heart rate in endurance-trained athletes. According to current studies, top athletes may exhibit increased LVM together with reduced systolic and diastolic blood pressure and heart rate as genuine physiological responses to endurance training.

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