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Original Article

Pathophysiology of Cardiac Output in Athletes and Non- Athletes

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***ABSTRACT*:**

Background: Overall cardiac function that includes both, flow and pressure generating capacities of the heart are known as cardiac output. Athlete heart can be differentiated morphologically depending on the time of exercise performed. **Aim:** To assess pathophysiology of cardiac output in athlete and non athlete. **Method:** A total of 200 subjects were selected for present study. Of the 200 subjects 100 were athletes and 100 were non athletes. The samples selected were aged between 18 to 45 years. Cardiac Output was calculated at rest and after exercise. Measures of hemodynamic status, cardiac structure and pumping capability were assessed by echocardiography. **Results:** Work rate at peak exercise was higher for athletes than as compared to non athletes. A significant difference was observed between work rate and peak exercise i.e. $p<0.01$. **Conclusion:** Cardiac pumping capacity was found to be higher in athletes than in non athletes. A stronger heart pumps more blood with less effort. Athletes showed lower systolic blood pressure. Further research is warranted for the same.

Key words: Cardiac output, athlete, exercise, echocardiography.

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INTRODUCTION:

The amount of blood heart pumps in 1 minute is known as cardiac output. In another words cardiac output is equal to the product of the stroke volume and the number of heart beats per minute. Whereas Cardiac power output is the product of cardiac output (CO) and mean systemic arterial blood pressure. Studies have shown that regular training and exercises is often associated with morphological changes in the heart, including increases in left ventricular chamber size, wall thickness, and mass.[1]The increase in left ventricular mass as a result of training is called athlete's heart.[2]

According to the literature studies have shown that 2 different morphological forms of athlete's heart can be distinguished: a strength-trained heart and an endurance trained heart. Long distance running and swimming tend to cause an increase in ventricular chamber dimensions. Whereas strength trained sports of weight lifting and wrestling, tend to increase wall thickness with unchanged left ventricular chamber size, which is caused by pressure overload.[3,4] Echocardiographic and ECG changes depend on the type of exercise undertaken. Very few studies are documented in literature regarding morphology of athlete heart. So we aimed to study the Pathophysiology of cardiac output in athletes.

MATERIAL AND METHOD:

A total of 100 male athletes were selected from local sport clubs and 100 healthy males were randomly selected. Participants were free from cardiovascular, pulmonary, metabolic and neuromuscular diseases and were considered apparently healthy from the point of view of cardiovascular risk factors. A detailed explained was given to the participant regarding the procedure to be performed. A written informed consent was obtained from all subjects taking part in the experiment.

Inclusion criteria included:
1. Patients aged above 18 years
2. Patients with no systemic diseases
3. Those willing to participate

Exclusion criteria included:
1. Mentally challenged patient
2. Physically challenged patient
3. Alcoholics

Exercise testing was performed on a Secacardio test cycle ergometer. Continuous electrocardiographic (ECG) monitoring was achieved with an integrated system, and heart rate was monitored. Blood pressure (BP) was recorded manually by auscultation over the left brachial artery using a stethoscope and hand-held aneroid sphygmomanometer. Selected samples were divided into two groups. Group 1 included 100 subjects those were athletes and group 2 included 100 subjects who were non athletes. All subjects were examined at rest and after exercise. Patients were advised not to consume alcohol or caffeine.

STASTICAL ANALYSIS:
Data was collected and tabulated. Data were analyzed using SPSS package descriptive data. In all cases, statistical significance was accepted at the $P<0.05$ level.

RESULTS:
A total of 200 subject both athletes and non athletes were selected for the present study. All subjects selected were aged above 18 years. Mean age of the subjects in current was found to be 24.6. Of the 200 samples selected 100 were males (50%) and 100 were females (50%) (Table1). In present study 70 subjects i.e. 35% were aged between 18 to 25 years, 80 subjects were aged between 26 to 35 years i.e. 40% and 50 were aged between 36 to 45 years i.e. 25%. In current study maximum subjects were aged between 26 to 35 years (Table 2).

Of the 200 samples selected 100 were athletes and 100 were non athletes (Graph 1). In present study no significant difference was found in age and body dimensions between athletes and non-athletes. Based on the result of our study it was found that the exercise hour for athletes were more than non athletes. Work rate at peak exercise was higher for athletes than as compared to non athletes. A significant difference was observed between work rate and peak exercise i.e. $p<0.01$.

No different was observed in heart size and volumes of all subjects measured at rest. However Left ventricle posterior wall thickness was significantly higher in athletes compared with non-athletes 9.6+1mm and 9.0+1 mm (Table 3). Comparison of IVSTs and LVIDs parameters also revealed significant difference between both groups (15.2+1.6 mm and 12.2 +1.4 mm; 36.7+ 6.5 mm and 32.1+7.1 mm, respectively). In present study it was found that Left ventricular dimensions and mass, left atrial chamber size, septal wall thickness, aortic root diameter, and right ventricular diastolic diameter in athletes were significantly increased as compared with non athletes ($p < 0.001$).

TABLE 1: Demographic age distribution

Mean age	24.6
Males	100 (50%)
Females	100 (50%)
TOTAL	n = 200

TABLE 2: Age distribution

Age group	Frequency
18-24	**70 (35%)**
25-35	**80 (40%)**
35-45	**50 (25%)**

Graph 1: Distribution of sample

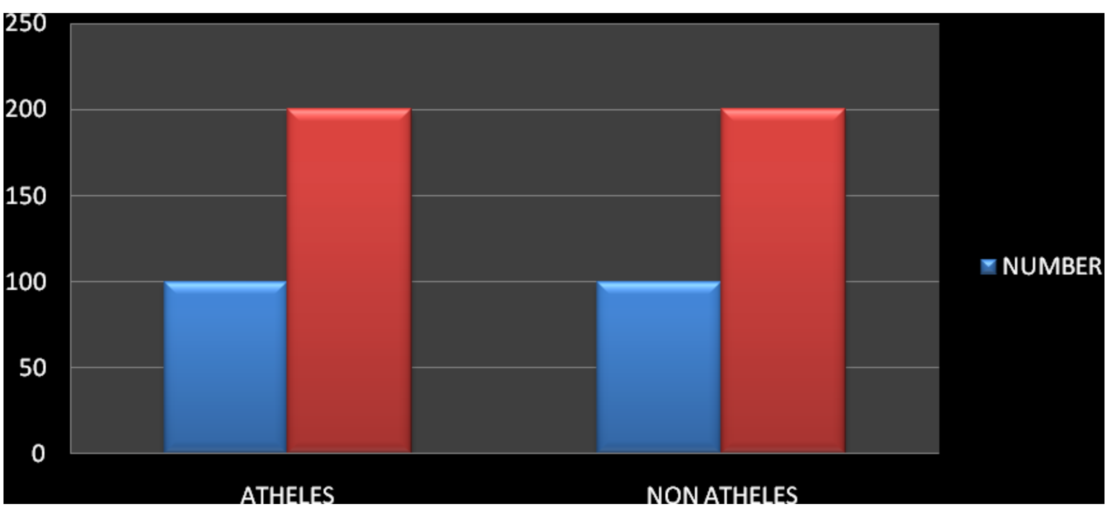

Table 3: Cardiac parameters in athletes and non athletes

	Athletes		Non athletes	
	Resting	peak exercise	Resting	peak exercise
Systolic blood pressure	110.3+ 6.4	166.4+ 12.3	119.42 +7.9	161.7+ 10.2
Diastolic blood pressure	76.5+ 7.2	86.8+ 8.1	75.8+ 7.8	80.7+8.1
Heart rate	75.2+10.5	174.7+2.4	78.5+ 11	172.4+ 2.8
Stroke volume	78.4+ 7.5	120.1+12.7	75.7+ 10.6	116.2 +12.4
Mean arterial pressure	84.4 +6.1	126.6 +7.6	86.2+8.1	123.4+ 7.1
Cardiac output	5.5+1.8	20.1+ 2.8	5.6 +1.4	18.6+ 2.2

DISCUSSION:

The heart rate is the simplest determinant to visualize cardiac output. The faster the heart beats, the more blood can be pumped over a particular period of time. Similarly, if the heart rate is slow, i.e. bradyarrhythmia, *or* is too fast, then cardiac output can be impaired. Acute supraventricular or ventricular tachycardia may also be a cause of low cardiac output, and even of cardiogenic shock.[5,6] The findings of current study suggest that athletes demonstrate greater cardiac pumping capability compared with non-athletes. Based on the results of our study we found that echocardiographic indices of cardiac structure and function is a good predictor of cardiac power output. Study conducted by Klasnja et al suggested that athletes demonstrated greater maximal cardiac pumping capability and reserve than non-athletes.[7] Various studies have found that chronic exercise induces changes in left ventricular structure, and function is consistent.[8,9] Bromley et al and Schlader et al studied Cardiac power output in their study. However cardiac power output was not examined in our study.[10]

In current study it was found that during exercise training process, there is an increased hemodynamic load on heart of the athletes. Abergel et al suggested that the mechanism behind this is myocardial hypertrophy, which is defined as an increase in the left ventricular mass.[11] in current study a significant differences in the left ventricle posterior wall thickness, interventricular septum and left ventricular size in systole. Heart size and volumes of both athletes and non-athletes were within the physiological limits of a normal population which is similar to those reported by other authors.[7]

CONCLUSION:

Within the limitation of our study we conclude that that peak exercise cardiac power output is significantly higher in athletes than in non athletes. A significant difference in the left ventricle posterior wall thickness, interventricular septum and left ventricular size in systole was observed in current study. Further studies are needed to evaluate maximal cardiac pumping capability in athletes.

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