

Original research article: Study of Effect of exercise on left ventricle systolic function in person without any heart disease

Dr Ekta Khurana, Assistant Professor, Department of Physiology, Pacific medical college and hospital, Udaipur, Rajasthan, India

Corresponding Author: Dr Ekta Khurana

BHK-A, Quarter no 202, Pacific medical college and hospital campus, bhilo kableda, udaipur,313001,rajasthan,India, E mail:khuranaekta11111@gmail.com

ABSTRACT:

Background: Regular training, in particular endurance exercise, induces structural myocardial adaptation, so-called "athlete's heart". In addition to the 2D standard echo parameters, assessment of myocardial function is currently possible by deformation parameters

Objective: To determine the impact of measures of cardiac function assessed by echocardiography on exercise capacity and to determine if these associations are modified by sex or advancing age.

Methodology: The Study was conducted on 100 subjects at Haldiram and Moolchand Heart Centre, PBM Hospital, S.P. Medical College, Bikaner. First a resting echocardiography was performed to evaluate cardiovascular diastolic function. After echocardiography the subjects were underwent Bruce protocol treadmill test.

Result & Conclusion: There was no correlation between exercise capacity and end systolic volume index ($p=0.089$), between exercise capacity and ejection fraction($p=0.459$), between exercise capacity and fractional shortening($p=0.467$), between exercise capacity and stroke volume index($p=0.361$), between exercise capacity and cardiac index($p=0.475$).

Key words: Exercise, Echocardiography, trade mill test

INTRODUCTION:

There is solid proof that actual shortcoming made by illness diminishes opposition different sicknesses, expands hazard of numerous infections like diabetes, coronary illness and hypertension are among the main sources of death and disability.[1,2] The significance of standard active work is underlined to keep a sound life and to have both preventive and recuperating impacts for some illnesses. It is realized that cardiovascular gamble factors are enormously worked on through the cardiovascular changes that happen with normal and long haul work out. [3,4]

During the previous ten years, practice tests and actual preparation programs have been involved with expanding recurrence in the analysis and the executives of coronary conduit infection (CAD). Despite the fact that extreme limitations in active work were recently prescribed to such patients, preparing studies have exhibited that appropriately executed preparing programs really further develop the activity resilience in most of CAD patients. [5] while causing just little dangers. Moreover, practice tests performed by a reasonable convention give significant

symptomatic data regarding the useful limit of the cardiovascular framework and structure a judicious reason for suggestions on active work in these patients. Investigations of the cardiovascular acclimations to exercise and preparing have principally been worried about focal circulatory boundaries. Essential to an undeniable degree of perseverance execution is an incredible limit of the oxygen transport framework. For instance, maximal oxygen take-up has been corre lated to heart volume, blood volume, and the aggregate sum of hemoglobin. [5,6] Moreover, the cardiovascular stroke volume (SV) gives off an impression of being a practical cir culatory boundary that most plainly isolates thoroughly prepared competitors from inactive subjects. The observing that SVexpansions because of preparing has zeroed in consideration on the significance of the heart as a siphon. The assessment that superior circulatory acclimation to submaximal practice and expanded maximal oxygen consuming limit are both connected with essential focal cardiovascular variations has consequently won. [7]

MATERIAL AND METHOD:

This Cross sectionanl study was conducted at Haldiram and Moolchand Heart Centre, PBM Hospital, S.P. Medical College, Bikaner,Rajasthan ,India from 1 Aug 2017 to 1 Aug 2018.

Study involves total 100 participants.

Demographic characteristic of all participants was taken including weight,height,Age etc.

BMI was calculated by using formula.(BMI =weight (kg)/ height (m)²)

Left ventricular Syastolic function was assayed by 2D echocardiography and followed by treadmill test by Bruce protocol of all participants.

Exclusion criteria: participants having atrial fibrillation/flutter at the time of exercise; had moderate or severe valvular heart disease; had poor image quality, which prohibited a final impression; had ejection fractions of less than 50%; or had echocardiographic evidence of exercise induced myocardial ischemia; any respiratory disease; suffering from any chronic or acute disease; taking any drugs that could affect the heart .

Evaluation of cardiovascular systolic function

LV end systolic and end diastolic internal dimensions (LVIDs and LVIDd).

Stroke volume (SV) was measured by Teicholtz method.

Cardiac output CO (L/min) = stroke volume (SV) X heart rate (HR).

Stroke volume index (SVI) = stroke volume / BSA.

Cardiac index CI (L/m²) = CO/BSA. LV systolic (ejection) function assessed by endocardial fractional shortening (FS) and ejection fraction (EF) using the Teicholtz method.

Tread mill exercise test The Bruce protocol (Bruce RA., 1972) for multistage treadmill testing of maximal exercise will be used. Resting ECG, pulse rate and blood pressure was measured. Then a standing ECG, pulse rate and blood pressure were recorded. Subject was explained and demonstrated the procedure and instructed to inform immediately if anything happens or any problem occurs. VO₂ max was calculated by following formula. VO₂ max in male =14.8 - (1.379 × T) + (0.451 × T²) - (0.012 × T³) VO₂ max in women =4.38 × T - 3.9 ("T" is the total time on treadmill measured as fraction of minutes).

RESULT:

Mean value of VO₂max, end systolic volume index, ejection fraction, fractional shortening, stroke volume index was observed as per **Table - 1**.

Table 1: Mean left ventricular systolic function and exercise capacity.

Parameters	Values (mean ± SD)
Stroke volume index	30.12 ± 5.26
Fractional shortening	34.55 ± 4.33
End systolic volume index	17±3.22
Cardiac index	2.35 ± 0.62
Ejection fraction	63.72 ± 5.85
VO2MAX	33.30 ± 7.47

After obtaining Vo2 max (exercise capacity) from bruce protocol and cardiovascular function i.e. left ventricle systolic function from echocardiography, correlation was seen between them through appropriate statistical analysis. Analyses were conducted to determine the strongest correlates of exercise capacity.

The correlation analysis showed that cardiovascular systolic function in our group had no correlation with exercise capacity. There was no correlation between exercise capacity and end systolic volume index (p=0.089), between exercise capacity and ejection fraction (p=0.459), between exercise capacity and fractional shortening(p=0.467), between exercise capacity and stroke volume index(p=0.361), between exercise capacity and cardiac index(p=0.475)(**Table 2**)

Table 2: correlation of vo2max with systolic function parameters of the subjects

Parameters	P VALUE
Stroke volume index	0.361
Fractional shortening	0.467
End systolic volume index	0.089
Cardiac index	0.475
Ejection fraction	0.459

DISCUSSION:

Maximum O₂ uptake capacity (VO₂max), the best available objective measure of aerobic exercise capacity, is generally lower in patients with coronary artery disease than in age-matched healthy subjects.² Many of these patients also exhibit impaired left ventricular function in response to exercise.^[8] However, recent studies have reported a poor correlation between Vo₂max and left ventricular performance.^[9] Furthermore, pharmacologic interventions that improve left ventricular function in patients with heart failure may not necessarily result in increased maximal exercise capacity.^[10] This would imply that left ventricular contractile function has little if any effect on maximal exercise capacity in cardiac patients. However, since VO max is sensitive to changes in cardiac output and left ventricular contractile function is one of the major determinants regulating cardiac output during maximal exercise.

The fall in ejection fraction during exercise may be caused by the following changes during exercise: (1) an inadequate rise in stroke volume with an inappropriately large end-diastolic

volume, (2) no change in stroke volume with a larger end-diastolic volume, or (3) a decrease in stroke volume with a minimal change in end-diastolic volume. Our results suggest that the fall in ejection fraction in our patients was associated with a lack of increase in stroke volume from the resting level and a small but statistically insignificant increase in end-diastolic volume.

The increase in cardiac output is a product of changes in heart rate and left ventricular stroke volume during exercise. Dynamic exercise is characterized by a large increase in cardiac output, while static exercise usually results in a more modest increment. The most important determinant for the increase in cardiac output is the exercise-related change in heart rate.^[11] There is a linear increase in heart rate with progressively increasing workloads until a maximal heart rate is attained. Maximal dynamic exercise causes a significantly greater rise in heart rate than maximal static exercise, in large part accounting for the different magnitudes of increase in cardiac output during the two types of exercise. The patient's age and degree of conditioning" both affect heart rate response to exercise, the maximal heart rate attained varying inversely with increasing age and training. The type of exercise performed (i.e., arm versus leg) also significantly influences the observed heart rate response to exercise. The heart rate response for arm exercise is significantly higher than for leg exercise at an equivalent workload.^[12]

In contrast to heart rate, there is no consistent exercise-related increase in stroke volume.^[13] Stroke volume is influenced by the net effect of changes in contractility (related to elevated blood catecholamines), after load (the tension which must be generated for ejection of blood from the left ventricle), heart rate and preload (the end-diastolic stretch of the left ventricle). Each factor in turn is influenced by the degree of exertion and the type of exercise performed, determining whether an increase, decrease or no change in stroke volume occurs.

CONCLUSION:

There was no correlation between exercise capacity and end systolic volume index ($p=0.089$), between exercise capacity and ejection fraction($p=0.459$), between exercise capacity and fractional shortening($p=0.467$), between exercise capacity and stroke volume index($p=0.361$), between exercise capacity and cardiac index($p=0.475$).

REFERENCES:

1. Hermansen, L. and Saltin, B.: Oxygen uptake during maximal treadmill and bicycle exercise. *J. Appl. Physiol.* 26:31- 37, 1969.
2. Mitchell, J.H., Reardon, W.C., McCloskey, D.I. and Wildenthal, K.: Possible role of muscle receptors in the cardiovascular response to exercise. *Ann. N. Y. Acad. Sci.* 301:23242, 1977
3. Woo JS, Derleth C, Stratton JR, Levy WC. The influence of age, gender, and training on exercise efficiency. *J Am Coll Cardiol.* 2006;47(5):1049- 1057.
4. Hossack KF, Bruce RA. Maximal cardiac function in sedentary normal men and women: comparison of age-related changes. *J Appl Physiol.* 1982;53(4): 799-804.
5. Weiss EP, Spina RJ, Holloszy JO, Ehsani AA. Gender differences in the decline in aerobic capacity and its physiological determinants during the later decades of life. *J Appl Physiol.* 2006;101(3):938- 944.
6. Franciosa JA, Pork M, Levine TB. Lack of correlation between exercise capacity and indexes of resting left ventricular performance in heart failure. *Am J Cardiol.* 1981;47(1):33-39.
7. Higginbotham MB, Morris KG, Cohn EH, Coleman RE, Cobb FR. Determinants of variable exercise performance among patients with severe left ventricular dysfunction. *Am J Cardiol.* 1983;51(1):52-60.

8. Okura H, Inoue H, Tomon M, et al. Impact of Doppler-derived left ventricular diastolic performance on exercise capacity in normal individuals. *Am Heart J.* 2000;139(4):716-722.
9. Vanoverschelde JJ, Essomri B, Vanbutsele R, d'Hondt A, Cosyns JR, Detry JR. Contribution of left ventricular diastolic function to exercise capacity in normal subjects. *J Appl Physiol.* 1993;74(5):2225- 2233.
10. Skaluba SJ, Litwin SE. Mechanisms of exercise intolerance: insights from tissue Doppler imaging. *Circulation.* 2004;109(8):972-977.
11. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: a comparative simultaneous Doppler-catheterization study. *Circulation.* 2000; 102(15):1788-1794
12. Higginbotham MB, Morris KG, Cohn EH, Coleman RE, Cobb FR. Determinants of variable exercise performance among patients with severe left ventricular dysfunction. *Am J Cardiol.*, 1983; 51(1): 52-60..
13. Jasmine Grewal, Robert B, Garvan Kane, Carolyn Lam, Patricia A. Pellika. Left ventricle function and exercise capacity. *JAMA*, 2019; 301(3): 286-294