Original Research Article

Correlation of exercise capacity with Cardiovascular systolic function

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Abstract

Introduction: The analysis of systolic function in echocardiography may be useful to explain the correlation between the presence of systolic function and maximal exercise capacity on exercise test.
Objectives: To correlate exercise capacity with the cardiovascular systolic function.
Material and methods: Necessary information as per our study was collected from the hospital record of the study participants undergoing routine measurements of systolic function by 2D echocardiography and exercise capacity (VO_2max) by treadmill test using bruce protocol.
Results: Karl Pearson correlation analysis showed that cardiovascular systolic functions in our group do not correlate with exercise capacity. There was no correlation between exercise capacity and end systolic volume index (r=-0.171, p<0.089), between exercise capacity and ejection fraction (r=0.069, p<0.495), between exercise capacity and fractional shortening (r=0.074, p<0.467), between exercise capacity and stroke volume index (r=-0.092, p<0.o.361), and cardiac index (r=-0.072, p<0.475) also showed no significant association with exercise capacity.
Conclusions: Correlation analysis showed that cardiovascular systolic functions in our group do not correlate with exercise capacity.

Key words

Exercise capacity, Cardiovascular systolic function, Correlation.

Introduction

Exercise capacity is defined as the maximal oxygen uptake for a given workload and can be expressed either as metabolic equivalents (METs or multiples of the basal rate of oxygen consumption when a person is at rest i.e. 3.5 ml/kg/min for an average adult) or VO_2max (the highest rate at which oxygen can be taken in and utilized by the body during exercise). VO_2max

increases linearly with intensity of exercise and attains its highest value close to or at maximal exercise. Individual's maximal capacity to do work aerobically is measured indirectly when we measure oxygen consumption as oxygen consumption is linearly related to energy expenditure.

Many factors including age, female sex, body mass index, and co morbid medical conditions, are known to be associated with a decrement in exercise capacity, as reflected by a decrease in maximal workload achieved or maximal oxygen consumption [1-6]. Aerobic exercise capacity decreases progressively with age and is associated with reductions in functional capacity, increases in disability and decreases in independence and quality of life. Determining the most important parameters affecting exercise performance, especially in relation to age, is complex, given the numerous confounding factors.

The most consistently reported mechanism contributing to this decrease in exercise capacity with aging is reduction in maximal heart rate. This appears to be non-modifiable and inevitable consequence of aging. Identifying potential reversible mechanisms underlying the maximal heart rate; this appears to be a non-modifiable and in evitable consequence of aging [7]. Previous studies have suggested that measurements of left ventricular systolic function do not predict maximal exercise time in individuals with normal or impaired left ventricular systolic function [8, 9]. However, differences in exercise capacity related to small changes in ejection fraction within normal range require evaluation in large population within the normal range.

When physicians have a better understanding of the physiology of exercise intolerance, they can then translate that understandings to patients in the office settings or at the bedside.

Materials and methods

The study participants were selected from Haldiram and Moolchand Govt. Centre of Cardiovascular Science and Research, PBM Hospital, Sardar Patel Medical College, Bikaner. This study was approved by the institutional ethical board, and verbal consent was obtained at the time of echocardiogram.

The Study was conducted on 100 subjects, necessary information as per my study were collected from the hospital record of the study participants undergoing routine measurements of left ventricular systolic function by 2D echocardiography and treadmill test by bruce protocol.

Participants

For this analysis, we excluded patients who had 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; suffering from any chronic or acute disease; taking any drugs that could affect the heart.

Anthropometric variables like height and weight were obtained and BMI and BSA were calculated from them. BMI was calculated from the formula = weight (kg)/ height $(m)^2$. BSA was calculated by formula = 0.0001x71.84 x (weight in kg) $^{0.425}$ x (height in cm) $^{0.725}$.

Echocardiography

First a resting echocardiography was performed on Phillips ultrasound machine on M-mode, 2D mode to evaluate cardiovascular systolic function. After echocardiography the subjects were underwent Bruce protocol treadmill test on CTMT 12 lead system.

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/m^2) = 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.

VO_2 max in male $=14.8 - (1.379 \times T) + (0.451 \times T^2) - (0.012 \times T^3)$

VO_2 max in women $=4.38 \times T - 3.9$ ("T" is the total time on treadmill measured as fraction of minutes).

Blood pressure, ECG and pulse rate were recorded immediately on termination of exercise and after 1 minute and 5 minutes of recovery. 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.

Results

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. Statistical analysis was done using MS excel and SPSS version 23.0.data was expressed as mean and standard deviation.Vo2 max was calculated using bruce protocol. 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)**
End systolic volume index	17±3.22
Ejection fraction	63.72 ± 5.85
Fractional shortening	34.55 ± 4.33
Stroke volume index	30.12 ± 5.26
Cardiac index	2.35 ± 0.62
VO2MAX	33.30 ± 7.47

Table - 2: Correlation of VO2max with Systolic function parameters of the subjects.

Parameters	**r**	**p**
End systolic volume index	-0.171	0.089
Ejection fraction	0.069	0.495
Fractional shortening	0.074	0.467
Stroke volume index	-0.092	0.361
Cardiac index	-0.072	0.475

Karl Pearson 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 (r=_0.171, p<0.089), between exercise capacity and ejection fraction (r=0.069, p<0.495), between exercise capacity and fractional shortening (r=0.074, p<0.467), between exercise capacity and stroke volume index (r=-0.092, p<0.361), and cardiac index (r=-0.072, p<0.475) also showed no significant correlation with exercise capacity as per **Table - 2**.

Discussion

In our study, we observed no correlation between left ventricle systolic function within normal range and exercise capacity which is comparable to findings by Grewal, et al. [10]. We found that

exercise capacity is not importantly influenced by variations of ejection fraction within the broad range of normal values; for example, an ejection fraction of 70% would not portend a better exercise capacity than an ejection fraction of 55%.

End systolic volume is usually regarded as an index of left ventricular ejection performance which is known to have very little if any influence on VO_2 max in normal subjects. Ehsani, et al. (1978) [11] reported a parallel reduction in left ventricular dimensions and exercise capacity among endurance athletes.

Jean Louis, et al. (1993) [12] observed a significant positive correlation between left ventricular volumes (at both end systole and end diastole) and VO_2 max, which on multivariate analysis showed only end systolic volume as independent correlate of VO_2 max.

In contrast to previous studies by Ehsani, et al.(1978) [11] where endurance athletes exhibit larger cardiac volumes than untrained individuals, Jean Louis, et al. [12] found higher volumes even in untrained individuals. They suggested that as there was no correlation of VO_2max with left ventricular ejection phase indexes in their study in normal individuals, the physiological significance of end systolic volume is related to more cardiac size than to ejection performance.

Left ventricular function is usually established by computing or estimating ejection fraction. Ejection fraction is the percentage of blood ejected from the ventricle during systole in relation to the total end-diastolic volume. Visually we judge left ventricular function on the basis of how much smaller the ventricle becomes during systole.

When the function of the heart is impaired, less blood will be ejected and the ejection fraction will fall. Ejection fraction is also a function of ventricular size. When the ventricle is large (in athletes, for instance), ejection fraction will drop. This is pure mathematics. When the ventricle is large, even a small systolic reduction in ventricular size (and a relatively low ejection fraction) will yield a sufficient stroke volume to perfuse the body. Conversely, when the ventricle is small (also known as hypovolemia), it will compensate by increasing its contractility. In this condition the ejection fraction will be higher than normal.

During conventional echocardiography, left ventricular function is assessed at rest. Therefore we have no clue as to the "functional capacity" and contractile reserve of the ventricle. Obviously it would be hazardous to rely on ejection fraction alone. We will see later how this can affect our interpretation under various conditions.

Conclusion

In the range of normal values, the ejection fraction does not correlate with cardiovascular exercise capacity.

A reduction in ejection fraction is simply a consequence of ventricular dysfunction. When contractility drops, the heart has to compensate. This is achieved by several means:

- Elevation in sympathetic activity (which increases heart rate and contractility);
- Increase in salt and water retention (which increases blood volume and elevates preload);
- Dilatation of the ventricle - which, by the Frank starling mechanism, increases contractility. More volume is ejected relative to the degree of inward motion of the walls.
- When certain segments reveal poorer contractility, other areas will compensate by contracting "more than normal" (hypercontractility)

Since exercise capacity is associated with prognosis both in healthy individuals and in patients with heart diseases. Work needs to be done to see if we can modify this age related

decline in exercise capacity. Identification of factors that modify the exercise capacity is highly relevant, because its reversal can improve the quality of life of the patient and have an effect on survival.

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