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Review Article

Response of Left Ventricular Volumes and Ejection Fraction during Different Modes of Exercise in Health and CAD Patients - a

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ABSTRACT

Different studies support the notion that chronic aerobic, anaerobic and resistive exercise bouts can influence Left Ventricular (LV) function contractility state and volumes. Exercise training in health and disease brings about an improvement in quality of life; alleviate the negative impact of age and pathological state on the maximal work capacity. This review provides an overview of the latest findings in this research field on humans. The aim of this review is to elaborate on what is known about the physiological mechanisms affecting LV volumes following single bout of exercise and exercise training. The different modes of exercise and their impact on LV in young adult, athletes, untrained young adults, trained, untrained healthy older adults and coronary artery disease patients, are reviewed and discussed. Together these findings suggest that long lasting aerobic exercise training, is probably one factor that encounter the aging and coronary artery disease process' by enhancement LV volumes. However, this is not the case during anaerobic and resistive and following training in which the opposite might be true. The purpose of this review was to provide a current clarification for the role of different exercise intensities, and modes on human health.

Keywords: Exercise training; Left ventricular function; Aging; Anaerobic exercise; Coronary artery disease; Elite athletes

INTRODUCTION

The complex interplay of the different physiological responses to exercise with genetic polymorphism can be found in the origins of LV volumes and Ejection Fraction (EF) in health and in Coronary Artery Disease (CAD) patients. It has been assumed that resistive exercise can be very demanding due to the presser response [1, 2] which might have a significant effect on LV volumes [3]. However, in healthy subjects, the effects of successful adaptations to pressure overload training are still controversial, as pressure per se does not always produce intrinsic depression of the myocardial inotropic state. Though, this is not the case in patients with heart disease and hypertension in which the adaptation to pressure overloads is a pathological.

Although hypertrophy is thought to compensate for hemodynamic stress, Echocardiography documented myocardial hypertrophy is an independent risk factor for cardiovascular morbidity and mortality [4]. Pressure overload leads to a marked increase in LV mass of the myocardium, which is probably an adaptive reaction aimed at normalizing the increased wall stress thus, reduce LV volumes and EF [5].

On the other hand, endurance training improves maximal work capacity as evidenced by higher maximal oxygen uptake, Stroke Volume (SV), LV End Diastolic Volume (EDV), EF, cardiac output and by lower End Systolic Volume (ESV), heart rate and blood pressure [6].

Aerobic exercise, such as running, walking, cycling and swimming, involves movement of large muscle groups. The marked arteries' dilatation I.e. volume overload that produces LV eccentric hypertrophy by increasing venous return and consequently, LV EDV is increased and LV ESV is reduced and hence, SV and EF are augmented [7]. This physiological hypertrophy is characterized by chamber enlargement and a proportional change in wall thickness. In contrast, isometric, static exercise or resistive training, involves sharp increase in after load rather than volume overload resulting in LV concentric hypertrophy [8]. Physiological hypertrophy in athletes is an unusual adaptation by which the myocardium compensates for an increased in volume overload. At some point this adaptation can be defined as an athletic heart in healthy subjects or, as pathological in cardiac patients. An important problem is determining when LV hypertrophic ceases to be physiological and begins to be pathological. However, Animal studies have demonstrated that physiological and pathological hypertrophy has distinct structural and molecular bases [9,10]. These LV Hypertrophies change volumes, mass and function in relation to the mode of exercise emphasizing the role of exercise in epigenetic.

LV volumes and EF

LV systolic function is generally assessed by measuring the extent and velocity of fibre shortening, EDV, ESV, EF and velocity of circumferential fibre shortening [11]. LV EF is a measurement of the fraction or ratio of blood ejected from EDV leaving the ventricles with each contraction [12]. LV EF represents the volumetric fraction of blood pumped out of the ventricle every heartbeat. It is calculated by subtracting ESV from EDV divided by EDV, and is an essential parameter for the evaluation of LV function [13]. In addition, the relationship between LV EDV and its SV refers to the capacity of the LV to increase contractility state at systole, i.e., ejection indices, which can be obtained by echocardiography and radionuclide methods [1,14]. Higher EF values mean an increase in LV contractility.

LV SV is an absolute measurement as cardiac output where LV EF is a relative quantity, and it is the amount of blood pumped past the aortic valve into the aorta and then into the systemic circulation, while right ventricular SV is driven through the pulmonary valve into the pulmonary circulation.

At rest and at peak aerobic exercise, left ventricular contractility and volume measurements can be obtained in a clinical setting by noninvasive methods [15]. LV EF is expressed as a percentage measured by several methods which include: a. Echocardiogram b. Magnetic resonance imaging and c. Multiple gated acquisition radio nuclear scan, probably more reliable than echo with a smaller variation [16].

If the LV EF is reduced below normal values it can be a sign of myocardial dysfunction. Ranges of LV EF to defined health status are as follow: a. 50 – 75% is a normal range, b. 40-49% slightly below normal and, c. 39% and below, mild-to-moderate-to-sever heart failure. This is with the understanding that mitral regurgitation and segmental wall motion abnormalities are not present. The accuracy in the determination LV volumes which is a function of chamber size and shape is very dependent on the experience of the technologist or cardiologist and thus influence the determination of LV EF. A change in LV EF of 5-10% is likely to represent a genuine fall in LV EF. Anything < 5% is likely to be within the limits of test-retest reproducibility for the measurement of LV EF >10% is highly likely to represent a real decline.LV EF is a routine clinical standard to assess cardiac function. It is valuable to determined LV function and contractility in health and disease. It is often useful in determining contractility in the basal state of chronic heart disease [17]. LV EF is

a complex function of LV EDV, ESV and SV which are preload and after load dependent [17,18]. Thus, it is understandable why LV EF would not detect subtle changes in myocardial contractile state.

LV EF and volumes responses during aerobic exercise

Aerobic exercise alters the loading conditions of the myocardium. The main changes are in heart rate, LV EDV, ESV and SV the components of cardiac output. Total peripheral resistance declines with a slight to moderate rise in systolic blood pressure while diastolic blood pressure decrease or remain unchanged. Therefore, the contracting LV works against volume overload. Following physical activity or inactivity LV function, EDV, ESV, SV and EF may adjust subsequent the changes in muscle mass. [19]. in healthy young individuals, LV function indices measured have been shown to increase with aerobic exercise and training [20]. In healthy subjects during aerobic exercise the male's SV exceeded that of the female due to gender differences in LV geometry and variance in heart size [21]. During maximal aerobic exercise, in healthy young males and healthy young women oxygen uptake values and SV are significantly higher in men while, LV EF increases similarly in both groups from rest to maximal effort [22]. At peak exercise, men and women increase EDV and decrease ESV (Figure 1) and thus, increase in SV (Figure 2) and EF (Figure 3) is noted [23]. Such responses may reflect both an increase in myocardial inotropic state and a significant reduction

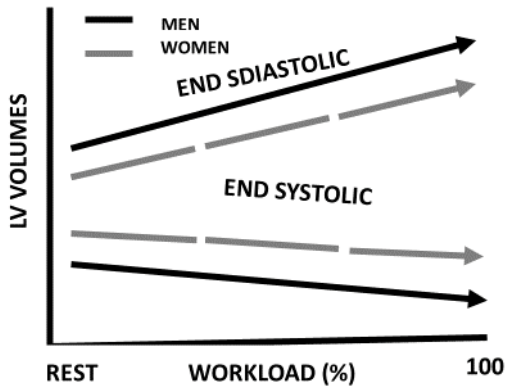

Figure 1: LV volumes responses during aerobic exercise in healthy men and women well trained and sedentary subjects.

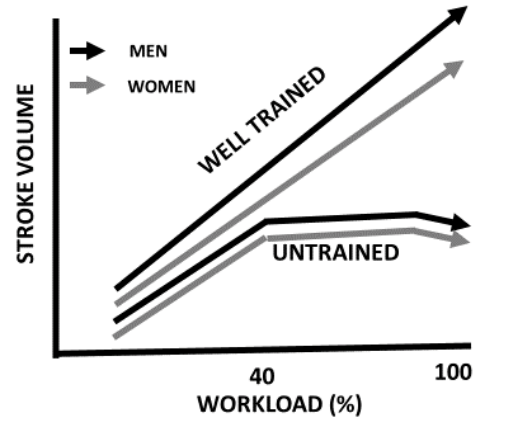

Figure 2: Stroke volume responses during aerobic exercise in healthy men and women well trained and sedentary subjects.

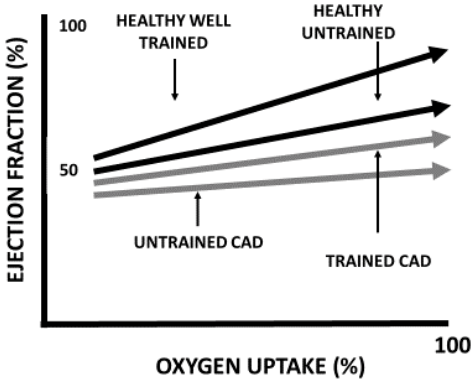

Figure 3: Ejection fraction changes during aerobic exercise in health and disease. No differences between men and women.

in total peripheral resistance [24]. Therefore, the increase in cardiac output during aerobic exercise was due to an increase in heart rate and SV in both men and women [25].

EF during dynamic aerobic exercise measured at maximal oxygen uptake may reach 80% and 90% in top aerobic athletes. In normal active subjects' contractility and SV are increased along with EF reaching 70% - 80% [15]. During sub maximal exercise, however, the decrease in ESV is less in women than in men.

Healthy aging appears not to affect LV EF, EDV, ESV and SV at rest. However, during exercise the EF is increase decrease or reminds unchanged depends on the individual's physical fitness. Aging reduces LV function due to environmental influence, genetics and changes attributed to normal physiological aging [26]. Prior to training of the elderly subjects LV EF, increase only modestly during exercise from resting values. Following sub maximal endurance exercise training in the healthy elderly, LV EF increase significantly at peak exercise compared to that observed before the initial training program [27,28]. The increase in LV contractility and the reduction in total peripheral resistance following training bringing about a significant reduction in ESV and thus, increase SV which correlates with an increase in venous return [29].

Cardiovascular system, morphological and physiological changes have been identified in the CAD patients. Important factor that influence the physical performance of the cardiac patient is aging associated with a shift in mechanism by which cardiac output is maintained during sub- maximal exercise. Myocardial functional changes include declines in maximum heart rate, SV, LV function and an increase in total peripheral resistance. In spite of having lower early diastolic filling rate LV response to sub maximal exercise in CAD patients with normal resting LV EF showed an increase in LV EF and LV ejection rate during aerobic exercise. The LV EF response of CAD patients to aerobic exercise is a significant increase values from rest to exercise. In addition, during sub maximal exercise, EDV is maintained and even increased. As a result, CAD patients appear to have a greater reliance on the Frank-Starling mechanism for the maintenance of cardiac output during sub maximal exercise [30].

Exercise-based cardiac rehabilitation increases peak oxygen uptake and improves work capacity in cardiac patients. Following 6 months, exercising cardiac patients compare with controls improve SV, EDV and EF with reduced total peripheral resistance at rest [31].

Marked changes are notable in the CAD patients during maximal effort in which EF, SV, cardiac output, contractility, and oxygen uptake are increased, while total peripheral resistance, systolic and diastolic blood pressure are decreased, thus, lowering after-load which in turn facilitate LV systolic and diastolic function [32]. Furthermore, a decrease in resting and during sub maximal exercise in heart rate, systolic and diastolic blood pressure is observed.

LV volumes and EF during resistive exercise

During static exercise force is developed without or with minimal muscle shortening. Cardiac output is increased slightly from 5 to 10-12 L·min^{-1}, mainly by the increase of heart rate. In addition, sharp increases in systolic, diastolic and mean arterial blood pressures are seen. These blood pressure elevations bring about a pressure overload on LV. This presser response may be important in maintaining perfusion of the active muscle groups during sustained contraction However, it opposes LV ejection.

Resistive training known as power and strength training improve work capacity in sport and daily tasks. Also, it may attenuate the aging effects on muscle strength and mass. Resistive Training of small muscle mass, such as the handgrip manoeuvre affects LV function in variable manner both in healthy young men and women. EDV, ESV, SV and EF have been found [33]. To increase, decrease, or remain unchanged (Figure 4). By contrast, the corresponding effects of resistive training with active large muscle mass, is related to the size of the activated muscle mass, intensity and duration of contraction [33,34]. No changes were noted in males and females in SV and EDV. However, EF rise by exercise only in the males due to decrease in ESV while it reminded unchanged in the females.

During resistive bouts in elderly men, In the face of the presser response, EF remains unchanged while it increases significantly in the young subjects. In elderly and young individuals EDV and ESV significant increase are observed from rest to moderate resistive exercise. However, during heavy isometric exercise such as dead lift, ESV is significantly reduced from resting values in the young group while in the elderly; ESV is increased [34]. The effects of long lasting resistive training on power athletes show a slight or minor changes in LV end diastolic and systolic dimensions results in a slight decrease or unchanged LV EF [35].

LV function is normally maintained or improves in normal subjects and cardiac patients with mild impairment of LV contractility. CAD patients with poor left ventricular function may show deterioration during isometric exercise, although this pattern of response is difficult to predict from resting studies. Many CAD patients who show ischemic ST depression or angina during dynamic exercise may have a reduced ischemic response during isometric or combined isometric and dynamic exercise [36]. Patients with CAD have an impaired health status and reduced exercise work tolerance due to LV dysfunction and reduced in peripheral adjustment ability. Therefore, Isometric exercise is usually discouraged for patients with CAD because of the possible adverse effects of increased blood pressure on LV function [36]. In addition, it presents several constrains in the assessment of cardiac function in CAD patients. Furthermore, even if resting LV volumes and LV EF could give a complete picture of LV systolic function during exercise, it would not simultaneously quantify LV diastolic function in heart failure [37,38]. In patients documented CAD or myocardial infarction which were in a supervised aerobic exercise program, had a significantly lower LV EF at rest than normal subjects, and did not have significant changes in LV volumes and EF during handgrip or dead lift. These suggest that LV responses are similar in normal and exercise-trained CAD patients during upright sub maximal isometric exercise utilizing small or large muscle mass [1].

LV EF and volumes responses during anaerobic exercise

Anaerobic effort is characterized by exposing the subject to a very high degree of sudden strenuous all-out exercise therefore, it is not recommended for CAD patients. It consists of short intense bouts of physical activity, such as weightlifting and sprints, where oxygen demand exceeds oxygen supply. While aerobic exercise energy supply relies on oxygen, anaerobic exercise utilizes through glycol sis. Although one would expect to obtain increases in LV function with increase in adrenergic state produced by anaerobic exercise intervention, echocardiography indices and blood pressure response resembles those seen during isometric exercise [2]. Indicating that auto-regulation is not involved while performing sudden strenuous all-out exercise. During all-out anaerobic effort in healthy young men and women values for cardiac output are low compared to what one would expect during maximal dynamic aerobic exercise; 14.4 L·min^{-1} for men and 12.3 L·min^{-1} for women [39]. The explanation for the lower cardiac output might be related to the short duration of the tests, in turn it did not bring about the appropriate adjustment of the LV volumes and LV EF and due to the relatively high total peripheral resistance (Figure 5) which was not as low as seen during aerobic exercise [39].

The assumed luck of the auto-regulation mechanism may indicate that the reduced SV and LV EF in men is due to the relatively

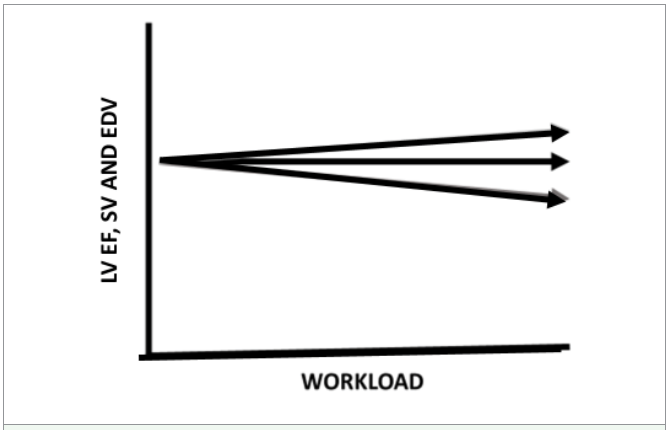

Figure 4: LV EF, SV and EDV responses during resistive training in healthy and disease men and women.

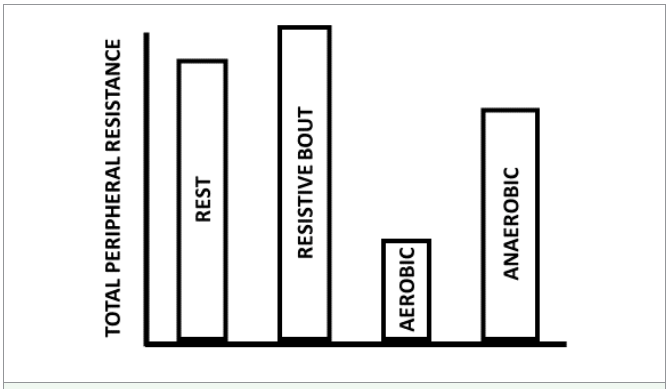

Figure 5: Different responses of total peripheral resistance in health and disease and in men and women.

high pressure opposing LV ejection although they increased LV contractility. On the other hand, women keep their SV and LV EF values at peak all-out anaerobic exercise as at rest probably due to the lesser increase in mean arterial blood pressure along with increased contractility. Previous report suggested that during strenuous exercise well trained healthy young adult subjects did not increase SV and LV EF and sometimes SV was reduced [15,40].

Following the Wingate Anaerobic Test, in older subjects, few elder lies may experience ECG abnormalities thus; all-out anaerobic exercise may be dangerous for the older population, due to hypoxia and inappropriate blood pressure response [41]. During exercise in the elderly, compared to resting values, LV EF and ESV are unchanged while, EDV and SV are lower. However, it is very important to emphasise that these elder lies were with work capacity of 12.5 METs. These LV function responses in the elderly are most likely due the relatively higher total peripheral resistance opposing the LV ejection [42]. Data suggest that during all-out anaerobic exercise, forces opposing ejection were not reduced enough to avoid reduced left ventricular function in the older subjects [43]. This is attributed to functional changes with age in the myocardium and associated blood vessels, leading to impairment of left ventricular function and blunted inotropic and chronotropic responses to catecholamine. Therefore, an all-out anaerobic-type effort should not be given to an older subject due to the great hazardous potential.

CONCLUSIONS

The above data indicate that individuals retain a high degree of trainability in the life cycle. LV function, contractility, EDV, ESV, SV and EF improve significantly following aerobic exercise training while during resistive training there is an improvement in strength with slight changes in LV function and volumes. Anaerobic exercise is not a recommended tool for young, elderly and prohibited for CAD to improve fitness. This mode of exercise serves the needs of elite competing athletes. Worth to note, following training much of the improvements happens peripherally in the elderly and CAD patients, just as in younger individuals. Physical activity or exercise can improve immediate and long-term health status, quality of life and reduce the risk of developing several diseases such as CAD.

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