Effect of Weight Training on Physical Work Capacity in Chronic Heart Failure

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ABSTRACT

Purpose: *This study was conducted to investigate the effect of weight training as a resisted exercise program on peak work load, peak oxygen saturation and anaerobic threshold during rehabilitation of chronic heart failure patients.* ***Subjects:*** *Thirty male patients selected from the out patient clinic of the National Heart Institute participated in the study. Their mean age was 50±6 years and the mean ejection fraction was 30-40%. They suffered from ischemic heart disease since one year ago.* ***Methods:*** *All patients were evaluated by cardiopulmonary exercise test before and after three months of training. The measuring parameters included determination of peak oxygen saturation, anaerobic threshold and peak work load. All participants received a resistive training program in the form of leg strengthening exercises.* ***Results:*** *The post treatment results of the study revealed significant increase in the peak oxygen saturation, anaerobic threshold and peak work load. Conclusion: Strengthening exercises can be considered as an effective and safely intervention in rehabilitation of chronic heart failure patients.*

INTRODUCTION

Chronic heart failure (CHF) is a major health problem with a large socio-economic burden of chronic diseases, handicap and dependency. Heart failure is not a disease but a clinical syndrome that represents a final common pathway in patients with a number of conditions that overload or damages the heart. It is considered as the fatal end result of chronic pressure or volume overload due to valvular disease, arterial hypertension and myocardial infarction[8].

Heart failure is a progressive disorder that is initiated after an event. It either damages the heart muscle with a resultant loss of functioning cardiac myocytes or disrupts the ability of the myocardium to generate force, thereby preventing the heart from being contracted normally. This index event may have an abrupt onset (myocardial infarction) or gradual onset as in volume or work overloading or it may be hereditary as in genetic cardiomyopathies. All these events produce a decline in the heart's pumping capacity. This low cardiac output gives inadequate oxygen supply and constitutes circulatory failure[11].

The signs and symptoms are caused by reduced cardiac output and increased venous pressure. CHF is a multisystem syndrome with a variety of pathophysiological changes (dyspnea, edema, vasoconstriction, and fatigue) which are often made worse by abnormalities in the lungs, kidneys, peripheral circulation and skeletal muscle leading to reduction of exercise tolerance and contribute to the symptoms of functional disability[7]. Therefore, the congestive heart failure patients suffer from fatigue and dyspnea with exercise intolerance and a poor quality of life even in asymptomatic patients[10].

Neurohumoral compensatory mechanism suggested that heart failure progress as a result of over expression of biologically active molecules, which are capable of exerting toxic effects on the heart and circulation. These proteins include norepinephrine, angiotensin II, endothelin, aldosterone and tumor necrosis factor. The biochemical properties are sufficient to contribute to disease progression from long term activation that produces direct end-organ effect within the heart and circulation[11].

In patients with heart failure, activation of the sympathetic nervous system is frequently excessive at rest. It is evidenced by increased plasma level of norepmephrine. Two potential mechanisms could mediate this effect. Firstly, excessive sympathetic vasoconstriction which could interfere with exercise capacity by impairing skeletal muscle arteriolar vasodilatation and so, limiting oxygen delivery to exercising muscles. Secondary, sympathetic activation that could adversely affect muscle performance by altering muscle metabolic behavior, increase glycogenolysis and increase muscle lactate production. These effects could adversely affect muscle efficiency and impair performance in patients with heart failure[9].

Exercise intolerance is a nearly universal problem in patients with heart failure. Maximal exercise capacity is reduced not only in symptomatic patients (exercise capacity is often <50% of normal), but also in many asymptomatic patients (averages 60-70 % of normal)[9].

Cardiopulmonary exercise testing is made by progressive increasing work rate exercise to help in determining the level of the subject's exercise limitations, adequacy of performance of the external to internal gas exchange coupling[15].

Cardiopulmonary exercise testing with metabolic gas exchange measurements is now part of the routine assessment of heart failure patients and has become an important tool for risk stratification and selection of patients for cardiac transplantation. Oxygen uptake is more stable. It measures exercise tolerance than exercise time. Peak of oxygen saturation (VO_2max) is the maximum volume of oxygen consumed by the body each minute during exercise. Because oxygen consumption is linearly related to energy expenditure, so when measuring oxygen consumption, the individual's maximal capacity to do work aerobically is indirectly measured[12].

The benefits of long term moderate exercise training in patients with stable CHF were observed after two months of training, which were maintained for one year with a supervised program of the same intensity but with a lower number of sessions per week. The benefits include improvement of the functional capacity and quality of life[2].

Focused on local strength exercise training involving lower extremities muscle groups as an alternative to dynamic exercise training in chronic heart failure patients. The results showed an increase in exercise capacity and strength as well as improvement in ventilatory response post sub maximal exercise levels. It also revealed an increase in the quality of life after knee extensor muscles training[14].

SUBJECTS, MATERIAL AND METHODS

Subjects

Thirty male patients with chronic heart failure secondary to ischemic heart disease participated in the study. They were selected according to inclusive criteria from the out patient clinic of the National Heart Institute. Their mean age was 50±6 years; the mean weight was 80±7.3 kilogram, the mean height was about 170±8.4 centimeter and the ejection fraction was between 30-40% (class II and III according to NYHA classification).

All patients were examined by cardiac physician to exclude any other medical conditions. The patients signed a medical consent for agreement to participate in the study.

Material

For evaluation

1- Oxycon pro (Jaeger - Germany), cardiopulmonary exercise test unit was used to measure oxygen saturation (VO_2), anaerobic threshold ($VO_{2\,\%}$) and workload. It consists of 12 channels of electro cardiogram (ECG), a screen indicating the percentage of carbon dioxide in breathe, cycle ergometer (Erg 900) for measurement of blood pressure and thermal printer.
2- Electrocardiogram (ECG) monitors (telemetry- Heweltt Backard, Kayak -XA). made in USA was used to monitor heart rate and rhythm.
3- Mercurial sphygmomanometer was used for periodic measurement of blood pressure.

For treatment

Free weights (2-3 kg) of sand bags were used as resistive exercises for the lower limb muscles according to each patient's tolerance.

Methods

For evaluation

Before conducting the symptom limited exercise tolerance test, all participants were instructed to visit the testing places two times to be familiarized with the equipment and to be cooperative during conducting the test.

The patient's name, address, telephone number, height, weight, sex and age were recorded. To obtain maximum patient's confidence and performance, brief explanation of the procedures was done. The patients were asked to wear loose- fitting comfortable clothes and suitable shoes for exercise. Also, patients were instructed to avoid eating a heavy meal before the exercise test. Patients continued to take routine medication before exercise testing.

Patients were asked to sit in upright position on an electronically braked computerized bicycle ergometer with a gas exchange analyzer. A face mask was applied on the patient's nose and mouth. The metabolic parameters as oxygen consumption (VO_2) and anaerobic threshold ($VO_2\%$) were firstly measured from resting position. Each patient then was asked to rotate the cycle and increasing the workload gradually according to Wasserman et al. (1994)[15] protocol. The same parameters were measured until symptom limited exercise appeared.

The recovery phase continued for about three minutes after the end of the test to record ECG and blood pressure values till reaching the resting values.

The maximum voluntary contraction for the lower limb muscles was evaluated by using free weights for one repetition maximum and then 50-60% of this weight was taken as the amount of weight to be used in the training program.

Evaluation for the measuring variables was carried out before and after three months of treatment.

For treatment

The training program was applied in the form of strengthening (resisted) exercises.

Patients were trained using lower limb strengthening exercises. Individual contractions were made at 60% of maximum voluntary contractions relative to one repetition maximum, repeated ten times, for two sets, three times a week for three months. Interval weights were readjusted from time to time according to the patient's progress. Strengthening exercises included hip flexors, extensors, abductors and adductors, knee flexors and extensors, ankle dorsi and planter flexors muscles.

Individual contractions were not held for more than five seconds to avoid an increase in cardiac load. Each exercise was performed throughout the range of motion to maximize the potential benefits. Each session began with a warming up phase of stretching and ended by a cooling down phase.

The ECG was continually monitored by using telemetry through out the training sessions. Heart rate and blood pressure were measured at rest before training, at the middle of training session and after five minutes of recovery phase.

Statistical Methods

All data were expressed as mean ± SD and comparisons between the means were done using t-test. The threshold of significance was delineated by the P-value when $P < 0.05$ is statistically significant.

RESULTS

The data collected regarding chronic heart failure patients participated in this study have been analyzed and represented as follows.

1- Peak oxygen consumption (VO_2) ml/min

Comparing the pre and post treatment mean values of peak oxygen consumption (VO_2) showed significant increase. The mean value of peak oxygen consumption pre treatment was 927±225 ml/min while the post treatment mean value was 1109.3±227 ml/min. ($P<0.0001$) table 1 and figure 1.

Table (1): Showing the mean values of peak oxygen consumption (VO_2) ml/min pre and post treatment.

Item	Statistical value	Before training	After training	t-value	P- value
Peak oxygen consumption	Mean	927.000	1109.367	20.65	< 0.0001
	±S.D.	±225.301	±227.417		

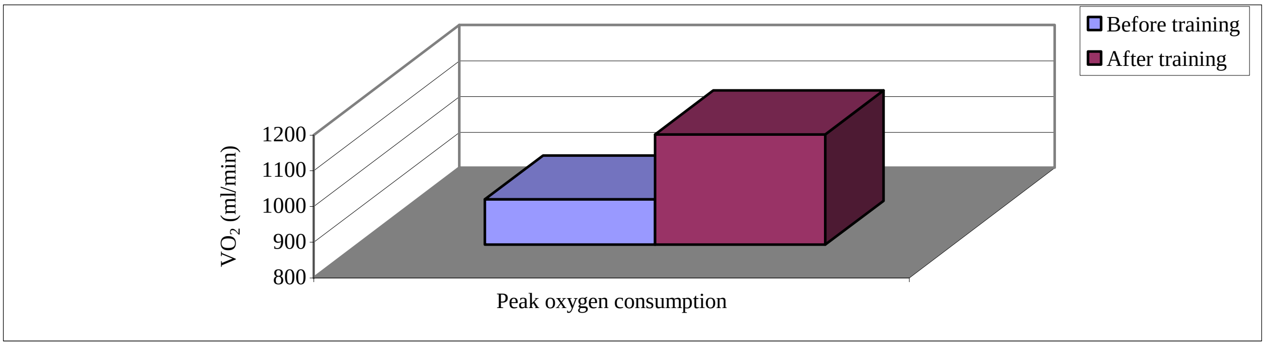

Fig. (1): Illustrating the mean values of peak oxygen consumption (VO_2) ml/min pre and post treatment.

2- Anaerobic threshold (VO_2%)

Statistical significant improvement was observed when comparing the pre and post treatment mean values of anaerobic threshold ($VO_{2\%}$). The pre treatment mean value was 36.16±8.26 ml/min%, while the post treatment mean value was 47.9±9.67 ml/min%. ($P < 0.0001$), table 2 and figure 2.

Table (2): Showing the mean values of anaerobic threshold (VO_2%) ml/min% pre and post treatment.

Item	Statistical value	Before training	After training	t-value	P-value
Anaerobic threshold	Mean	36.16	47.9	14.2	< 0.0001
	±S.D.	±8.26	±9.67		

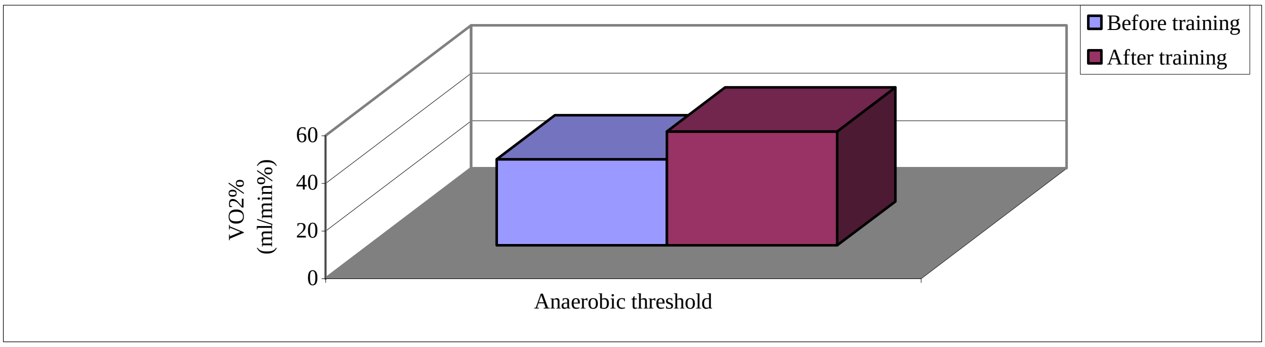

Fig. (2): Illustrating the mean values of anaerobic threshold (VO_2%) ml/min% pre and post treatment.

3- Peak work load (watt)

Significant increase was observed when comparing the pre and post treatment mean values of peak work load. The pre-treatment mean value of peak work load was 59.5±25.8 watt, while the post treatment mean value was 87.5±29.3 watt. ($P < 0.0001$), table 3 and figure 3.

Table (3): Showing the mean values of peak work load (watt) pre and post treatment.

Item	Statistical value	Before training	After training	t-value	P-value
Peak work load	Mean	59.5	87.5	16.35	< 0.0001
	±S.D.	±25.8	±29.3		

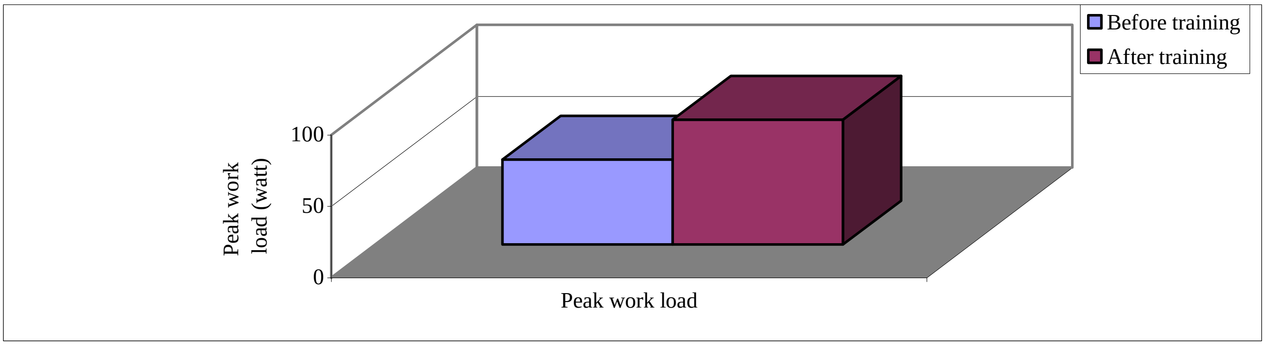

Fig. (3): Illustrating the mean values of peak work load (watt) pre and post treatment.

DISCUSSION

In the last decade, the epidemic of chronic heart failure is growing and appears to be the only cardiovascular condition whose prevalence and incidence are increased[4].

The current study was conducted to investigate the effect of weight training as a resisted exercise program on peak work load, peak oxygen saturation and anaerobic threshold during rehabilitation of chronic heart failure patients.

Cardiac rehabilitation has been shown to improve many pathohysiological changes in patient with left ventricular systolic dysfunction. Also, it was shown that the survival rate in cardiac rehabilitation participants was high in heart failure population. Exercise time, peak oxygen consumption, peak work load, anaerobic threshold are considered to be the major outcome measures that reflect the exercise tolerance, which are usually improved after exercise training[16,13].

Statistical analysis of the results of the collected data of the present study after the suggested period of treatment, showed statistical significant increase in the peak oxygen consumption (VO_2) by 20%, increase in anaerobic threshold (VO_2%) by about 32%. It also showed highly statistical significant increase in the work load by 47%.

The results of the study agree with Hambrecht et al. (1995)[5] who demonstrated that after exercise training at 40% to 80% of peak heart rate, an increase in maximum oxygen consumption and peak work load occurred. Moreover, the post treatment results also come in agreement with Hambrecht et al. (1997)[6] who studied the effect of various forms of aerobic training on nine patients with chronic heart failure due to left ventricular dysfunction with ejection fraction (26±10%) for six months and established significant improvement in peak oxygen consumption and peak work load.

The results confirm the findings of Belardine;I et al. (1999)[2] who conducted a study on 99 patients with moderate and severe CHF for 14 months. They reported highly significant (18%) increase in the exercise capacity (peak oxygen uptake) in the trained group than in the control group which was associated with increased quality of life.

The post treatment results also agree with Tyni et al. (1996)[14] who focused on local strength exercise training involving the lower extremity muscle groups as an alternative to dynamic exercise training. They found an increase in exercise capacity and strength as well as improvement in ventilatory response in sub maximal exercise levels.

The results of the current study come in agreement with Ades et al. (1996)[1] who reported an improvement in anaerobic threshold after exercise training. He attributed his findings to possible improvement in the intracellular abnormalities which lead to improvement of oxidative capacity. Improvement observed in the post treatment results may be attributed the effect of aerobic metabolism which resulted in less production of lactic acid. Also, it may be due to increase capillary density and succinate dehydrogenase activity, which indicates, increased aerobic metabolism.

On the other hand, the results of the current study contradicted with Coats, 1999[3] who studied the effect of three weeks dynamic aerobic exercise training on patients with CHF class II, III NYHA due to coronary artery disease and dilated cardiomyopathy. He found no significant increase in maximal VO_2 after dynamic aerobic exercise, although the peripheral resistance was reduced, which may be due to the short time of training.

Because of the sustained improvement in functional capacity seen in the present study after training program it can be stated that, physical training helps in reducing expensive hospitalization and improves quality of life. The difference observed after training is reflected in the patient's ability to cope with the physical demands of day to day activity, thus preventing the need for continuous bed rest and loss of independence.

Conclusion

Strengthening exercises for lower limbs can be safely enrolled in rehabilitation of chronic heart failure patients as it prevents muscle atrophy and weakness as well as reduces the relative work load on active muscle during daily activities.

REFERENCES

1- Ades, P.A., Waldmann, M.L. and Meyer, W.L.: Skeletal muscle and cardiovascular adaptation to exercise conditioning in older coronary patients. Circulation; 94: 323-330, 1996.

2- Belardinelli, R., Georgiou, D., Cianci, G. and Purcoro, A.: Randomized controlled trial of long-term moderate exercise training in chronic heart failure: effects on functional capacity, quality of life and clinical outcome. Circulation; 99: 1173-1182, 1999.

3- Coats, A.J.S.: Exercise training for heart failure, coming of age. Circulation; 99: 1138-1140, 1999.

4- Cowie, M.R., Wood, D.A. and Coats, A.J.S.: Incidence and etiology of heart failure. A population- based study. Eur. Heart J; 20: 421-428, 1999.

5- Hambrecht, R., Niebauer, J. and Fiehn, E.: Physical exercise in patients with stable heart failure. J. Am. Coll. Cardiol.; 25: 1239-1249, 1995.

6- Hambrecht, R., Fiehn, E. and Yu, J.: Effects of endurance training on mitochondrial ultra structure in skeletal muscles in patients with stable chronic heart failure. J. Am. Coll. Cardiol.; 29: 1067-1073, 1997.

7- Hasenfuss, G. and Just, H.: Myocardial phenotype changes in heart failure: cellular and sub cellular adaptations and their functional significance. Br. Heart. J.; 72 (Suppl) 10-15, 1994.

8- Hosenpud, J.D. and Greeberg, B.H.: Congestive heart failure: Pathophysiology, diagnosis and comprehensive approach to management, 2nd Ed. Lippincott Williams and Wilkins, Philadelphia;. 86: 1266-1272, 2000.

9- Lang, C.C., Rayos, G.H., Chomsky, D.B., Wood, A.J. and Wilson, J.R.: Effect of sympatho inhibition on exercise performance in patients with heart failure. Circulation; 96: 238-245, 1997.

10- Larsen, A.I., Arsland, T., Kristiansen, M., Haugland, A. and Dickstein, J.: Assessing the effect of exercise training in men with heart failure, comparison of maximal, sub maximal and endurance exercise protocols. Eur. Heart J.; 22: 684-692, 2001.

11- Mann, D.L.: Mechanisms and models in heart failure, a combinationorial approach. Circulation; 109: 999-1008, 1999.

12- Myers, J., Gullestad, L. and Vagelos, R.: Clinical Hemodynamic and cardiopulmonary exercise test determinants of survival in patients referred for evaluation of heart failure. Am. Intern. Med.; 129: 286-293, 1998.

13- Piepoli, M., Clark, A.L., Vlterrani, A., Adamopoulos, S., Sleight, P. and Coats, A.J.S.: Contribution of muscle afferent to the hemodynamic autonomic and ventilatory responses to exercise in patients with chronic heart failure, effects of physical training. Circulation; 93: 940-952, 1996.

14- Tyni, R., Gordon, A. and Sylven, C.: Improved quality of life in chronic heart failure patients following local endurance training with leg muscles. J. Card. Fail.; 2: 111-117, 1996.

15- Wasserman, K., Hansen, J.E., Sue, D.Y., Whipp, B.J. and Casaburi, J.: Measurements during integrative cardiopulmonary exercise testing. Principles of exercise testing and interpretation; 2: 52-79, 1994.

16- Wielenga, R.P., Coats, A.J.S., Mosterd, W.L. and Huisveld, J.A.: The role of exercise training in chronic heart failure. Heart; 78: 431-436, 1997.

الملخص العربي

تأثير التدريب بالأوزان علي سعه الشغل الفيزيائي في فشل عضلة القلب المزمن

تهدف هذه الدراسة إلى تحديد تأثير التدريب بالأوزان على سعة الشغل الفيزيائي في مرض فشل عضلة القلب المزمن . أجريت هذه الدراسة في معهد القلب القومي على ثلاثين مريضاً ممن كان متوسط أعمارهم خمسون عاماً.تم قياس كلاً من أقصى معدل لاستهلاك الأوكسجين ،الحد الأدنى للتنفس اللاهوائي باستخدام جهاز قياس كفائه الجهاز الدوري التنفسي وكذلك قياس الشغل المحمل الكلي قبل وبعد تطبيق البرنامج العلاجي الذي استمر ثلاثة أشهر . وقد تلقت المجموعة المختبرة برنامج تمرينات التقوية بالأوزان لجميع مجموعات عضلات الساق المختلفة. وقد أظهرت النتائج وجود فروق ذات دلاله إحصائية عند مقارنه النتائج قبل وبعد البرنامج العلاجي في صورة زيادة في معدل استهلاك الأوكسجين بنسبة20% ، زيادة في الحد الأدنى للتنفس اللاهوائي بنسبة 32% وكذلك زيادة الشغل المحمل الكلي بنسبة 47% . ومن هنا يتضح أن برنامج التقوية بالأوزان لعضلات الساق يمكن أن يكون امناً في برنامج التأهيل لمرضى فشل عضله القلب المزمن وكذلك فان البرنامج العلاجي له تأثير ايجابي على زيادة الشغل المحمل الكلي أثناء الأنشطة اليومية .