Review

Exercise training in heart failure

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Abstract

Chronic heart failure (CHF) is a common condition with a poor prognosis. It is associated with poor exercise tolerance and debilitating symptoms. These symptoms appear to be associated with pathophysiological changes that occur systemically in the patient with CHF. Exercise training in carefully selected patients has been shown to be safe and to improve exercise capacity. Many of the pathophysiological abnormalities of CHF are improved by training. Some studies have suggested a possible improvement in morbidity and mortality with training. This review analyzes the controlled clinical trials of exercise training in CHF published to date.

Keywords: exercise tolerance, exercise training, heart failure, rehabilitation

Introduction

Chronic heart failure is a common condition with a poor prognosis. It is associated with debilitating limiting symptoms even with optimal modern medical management. One of the most prominent symptoms of CHF is severe exercise intolerance with marked fatigue and dyspnea at low exercise workloads.

The severity of symptomatic exercise limitation varies between patients with CHF and appears to be unrelated to the extent of left ventricular systolic dysfunction at rest. Peripheral changes commonly occur in patients with CHF and have been implicated in the development of symptoms in this condition. The reason for fatigue in CHF has been more readily attributed to persistent vasoconstrictor drive, endothelial dysfunction, and a wide range of structural and functional abnormalities of skeletal muscle than to ventricular dysfunction per se [1]. Similarly, dyspnea appears to be more closely related to enhanced ventilatory effort than pulmonary congestion in most well-treated non-edematous patients [2]. This may be related to augmented reflex control systems, such as the arterial chemoreflexes and the muscle ergoreflexes (metaboreflexes) [3,4].

Interest has been shown in the possibility that some forms of exercise therapy, either localized or systemic, could benefit the peripheral manifestations of CHF and thereby modify the symptoms of patients with CHF. Recently it has even been suggested that regular training could have beneficial effects on disease progression and survival. This review looks at the controlled clinical trial evidence for the role of exercise therapy in the management of patients

CHF = chronic heart failure.

Table 1

Randomized controlled parallel group trials of exercise training in patients with impaired left ventricular function

Authors	*n*	Exercise Program	Outcome versus Control
Jette 1991 [17]	1. LVEF <30% Control *n*=10 ExT *n*=8 2. LVEF 31–50% Control *n*=10 ExT *n*=11	Supervised in hospital for 4 weeks a.m. – run 5 min × 3/ week at 70–80% max HR calisthenics 30 min cycle 15 min at 70–80% max HR p.m. – walk (graded paths) 30–60 min	LVEF <30% group Peak VO_2 increased by 221 ml/min ($P<0.05$) Peak WL increased by 13 W ($P<0.05$) LVEF 31–50% group Peak VO_2 increased by 85 ml/min (P=NS) Peak WL increased by 9 W (P=NS)
Koch 1992 [15]	1. ExT *n*=12 2. Control *n*=13	40 sessions over 90 days, graded program	QOL score increased by 52% ($P<0.0001$)
Belardinelli 1992 [18]	1. ExT *n*=10 2. Control *n*=10	Supervised 8 week program Cycle at 60% VO_{2peak} × 3/week	Ex tolerance increased by 45% ($P<0.005$) Peak O_2 uptake increased by 20% ($P<0.001$)
Kostis 1994 [19]	1. Placebo *n*=6 2. Digoxin *n*=7 3. ExT n=7	12 week program Walk/row/cycle/stair climb 60 min × 3–5/week at 50–60% max HR Cognitive behavioral sessions Diet modification	Ex tolerance increased by 182 s ($P<0.05$)
Kiilavuori 1995 [20]	1. ExT *n*=8 2. Control *n*=12	Supervised for 3 months Cycle 30 min × 3/week at 50–60% max VO_2	Ex tolerance increased by 71% at submaximal WL (P=0.01) High frequency component of HR variability increased by 22–55% during the day (P=0.0001)
Hambrecht 1995 [21]	1. ExT n= 12 2. Control n=10	Supervised for 3 weeks 10 min × 6/day at 70% max VO_2 then home-based until 6 months: cycle 40 min/day at 70% max VO_2 walk/calisthenics/ball games 60 min × 2/week	Peak VO_2 increased by 31% ($P<0.01$) Muscle mitochondria volume density increased ($P<0.05$)
Belardinelli 1995 [22]	1. ExT *n*=36 2. Control *n*=19	Supervised in hospital Calisthenics warm up 15 min × 3/week Cycle 40 min × 3/week at 60% max VO_2	Peak VO_2 increased by 12% ($P<0.001$) Improved indices of diastolic function
Hambrecht 1997 [23]	1. Ext *n*=9 2. Control n= 9	Supervised for 3 weeks 10 min × 6/day at 70% max VO_2 then home-based until 6 months: cycle 40 min/day at 70% max VO_2 walk/calisthenics/ball games 60 min × 2/week	Enhanced oxidative enzyme activity and improved mitochondrial size and number of cistae in skeletal muscle biopsies
Dubach 1997 [24]	1. ExT *n*=12 2. Control *n*=13	Supervised for 8 weeks Walk 60 min × 2/day Cycle 40 min × 4/week at 80% max VO_2	Peak VO_2 uptake increased Peak WL increased Ex time increased
Reinhart 1998 [25]	*n*=25	8 week residential program Cycle 40 min at 70–80% maximal exercise capacity × 4/week and walk 2 × 1 h/day	Increased maximal cardiac output and peak O_2 uptake

Table 1

Continued

Authors	*n*	Exercise Program	Outcome versus Control
Wielanga 1998, 1999 [26,27]	1. ExT *n*=41 2. Control *n*=39	Supervised for 12 weeks 3×10 min cycle/walk/ball games 5 min rest Exercise at target heart rate (resting HR + 60% difference between resting and maximum HR)	Feelings of being disabled decreased as shown by? Self-Assessment of General Well-Being (SAGWB) Ex time increased by 21.4% ($P<0.0001$) Anaerobic threshold increased by 12.5% ($P<0.05$) Peak O_2 uptake not significantly changed
Callaerts-Vegh 1998 [28]	1. ExT *n*=9 2. Control *n*=8	8 weeks intensive training	Peak O_2 uptake increased by 30.9% Urinary nitrate elimination decreased in controls but unchanged in ExT group
Willenheimer 1998 [29]	1. ExT *n*=22 2.Control *n*=27	Supervised for 16 weeks. Interval training: cycle 60 s at 80% VO_{2max} then 30 s rest week 0–7 15 min×2/week week 7–16 45 min×3/week	Peak ex WL increased by 7 W ($P<0.01$)
Belardinelli 1999 [16]	1. ExT *n*=50 2. Control *n*=49	Supervised 8 week program Cycle at 60% VO_{2peak}×3/week Supervised 12 month maintenance program: 2 sessions/week: 20 min stretching exercises; 40 min cycle	Peak O_2 uptake increased by 18% at 2 months ($P<0.001$) Lower mortality (n=9 versus *n*=20, RR=0.37; 95% CI, 0.17 to 0.84; $P=0.01$) Fewer hospital readmissions for heart failure (5 versus 14; RR=0.29; 95% CI, 0.11 to 0.88; $P=0.02$)
Sturm 1999 [30]	*n*=26	12 weeks of 50% maximal exercise capacity training progressing to 100 min step aerobics/week and 50 min cycle/week	Peak O_2 uptake increased by 23.3% ($P=0.001$ Peak WL increased by 26% ($P=0.0001$)
Keteyian 1999 [31]	1. ExT *n*=21 2. Control *n*=22	Warm up 5 min, cool down 11 min 33 min×3/week at 60–80% max HR (11 min×3 ergometers: cycle, row, arm, treadmill)×24 weeks	Peak O_2 uptake increased ($P<0.05$) Improved chronotropic response to exercise
Quittan 1999 [32]	1. *n*=25	Aerobics 3 h/week	Improved QOL ($P=0.0001$), physical role fulfillment ($P=0.001$), physical ($P=0.02$) and social ($P=0.0002$) functioning Peak O_2 uptake and ex time increased ($P<0.01$)
Hambrecht 2000 [33]	1. ExT *n*=36 2. Control *n*=37	2 weeks×10 min cycle 4–6/week followed by 6 months home-based cycle×20 min at 70% peak O_2 uptake	NYHA class improved Ex time increased LVEF increased from 0.30 to 0.35 ($P=0.003$) Peak ex TPR decreased ($P=0.003$)

ex=exercise, ExT=exercise training, HR=heart rate, LVEF=left ventricular ejection fraction, NYHA=New York Heart Association functional class, peak O_2 uptake=peak oxygen consumption, QOL=quality of life, RR=relative risk, TPR=total peripheral resistance, WL=work load

with CHF. It will concentrate on generalized exercise training programs and will not look at selective respiratory muscle training.

The beneficial effects of exercise training in cardiac patients without symptomatic heart failure have been well described and will not be reviewed here. This review focuses on the evidence for training as a therapeutic option in patients with stable symptomatic CHF and refers specifically to studies on post-infarct left ventricular dysfunction or selective muscle training where necessary.

Exercise training in chronic heart failure

Participation in an exercise program was historically considered to be ill-advised for patients with significant left ventricular impairment. Until the late 1980s, avoidance of physical exercise was the standard recommendation for all patients suffering from heart failure. This was initially challenged by uncontrolled studies that showed that selected patients with significantly impaired left ventricular function could increase their exercise tolerance after a period of exercise training [5,6,7]. There was no detectable deterioration in left ventricular function in patients included in these studies.

It was not until the end of the 1980s that reports were published showing that physical training could increase exercise capacity in patients with CHF. Sullivan and colleagues [8,9] found that patients with severe left ventricular dysfunction, some of whom had previously suffered heart failure, improved their maximal exercise performance after a prolonged regime of physical training. They demonstrated an increased blood flow to exercising muscle and an increased ability of skeletal muscle to extract oxygen from the nutritive blood flow. Ventilatory function was also improved, with a reduction in the respiratory exchange ratio at sub-maximal exercise and a delay in the anaerobic threshold. These authors showed no improvement in exercise cardiac output, so the training-induced benefits appeared to be mainly caused by peripheral adaptations.

The beneficial effects of training were confirmed in a controlled cross-over trial in 11 patients with stable class II-III CHF [10]. These carefully selected patients could exercise without serious ventricular arrhythmias and had no other medical condition that limited their exercise tolerance. After baseline evaluation and familiarization with laboratory procedures, all patients completed 8 weeks of exercise training and 8 weeks without exercise in a randomized cross-over study. The training regime produced a 20–25% increase in exercise tolerance and peak oxygen consumption. There was also a significant reduction in questionnaire-rated symptoms attributable to heart failure and a coincident increase in the extent and ease of performing daily activities. Since these early reports, numerous trials of similar design have confirmed these benefits and have shown many additional advantages of training in patients with heart failure. The randomized trials of parallel group design are summarized in Table 1 and those of cross-over design are in Table 2.

Training for heart failure: development of an idea

The earliest reports of training patients with significant left ventricular impairment were case reports or small series. The first report of exercise training of patients with heart failure was in 1987, in Russia [11]. This study compared the effects of exercise training with no exercise in post-myocardial infarction patients who showed clinical signs of heart failure. The training continued for 11 months and included exercise therapy, cycle ergometry and walking. The patients who exercised showed a considerable increase in physical working capacity, a more favorable hemodynamic response to stress, a higher stroke index, a reduced heart rate increment and a greater reduction in systemic peripheral resistance compared with the control patients.

Further physiological investigation of the training-induced improvements in exercise capacity was undertaken by Sullivan and colleagues [8,9]. This was closely followed in the early 1990s by the first randomized controlled trials of training in patients with chronic stable heart failure [10,12], which showed an increase in exercise capacity and improved symptomatic status.

In the rest of the 1990s, an increasing number of larger and better designed studies in patients with heart failure demonstrated a consistent increase in exercise capacity and a bewildering array of physiological benefits with exercise training. A summary of trials performed by a collaborative European group [13] and an overview of all trials published up to 1998 [14] have shown a consistent 15–20% increase in exercise capacity in a broad range of patients with heart failure. The beneficial effects of training have included improvements in hemodynamic responses, myocardial perfusion, diastolic function, skeletal muscle function and histological and biochemical responses, ventilatory control, peripheral vascular and endothelial function and neurohormonal and autonomic function.

Global exercise capacity was improved even with only low intensity training by relatively small muscle groups, which shows the primary importance of peripheral training mechanisms in even severe heart failure [15]. Although the majority of these training effects are likely to be beneficial for a patient with CHF, none of the trials to date has been designed specifically to address the question of prognosis or alteration in disease progression. The largest reported controlled trial [16] included almost 100 patients and showed a statistically significant improvement in survival and a statistically significant reduction in hospital readmissions for heart failure. The results of this study, however, must be regarded as an exciting possibility of benefit rather than proof. A sufficiently powered trial is required to evaluate the proposal that training may improve survival.

Table 2

Randomized controlled cross-over trials of exercise training in patients with impaired left ventricular function

Authors	*n*	Exercise Program	Outcome versus Control
Coats 1990, 1992 [10,12]	ExT *n*=17	Home-based 8 week program. Cycle for 20 min × 3/week at 60–80% max HR	Ex tolerance increased by 18.7% ($P<0.001$) Peak O_2 uptake increased by 18.2% ($P<0.01$) Other physiological benefits included decreased ventilation and improved heart rate variability
Adamopoulos 1993 [34]	ExT *n*=12	Home-based 8 week program. Cycle for 20 min × 3/week at 60–80% max HR	Reduced intramuscular phosphocreatine depletion and ADP accumulation ($P<0.003$)
Radaelli 1996 [35]	ExT *n* = 6	Home-based 20 min cycle × 5 days/week × 5 weeks	Peak VO_2 increased by 15% ($P<0.05$) Improved autonomic control of HR and peripheral vessels
Meyer 1997 [36]	ExT *n*=18	Interval cycle and treadmill training for 3 weeks	Ex distance up 65% ($P<0.001$)
Tyni-Lenne 1997 [37]	ExT *n*=16 (women)	Knee extensor endurance training for 8 weeks	Citrate synthase increased by 44% ($P<0.0001$), lactate dehydrogenase increased by 23% ($P<0.002$), oxidative capacity in relation to glycolytic capacity increased by 23% ($P<0.002$) Peak O_2 uptake increased by 14% ($P<0.0005$) Peak work rate increased by 43% ($P<0.0001$) Increased distance ambulated during 6 min ($P<0.03$) Improved overall ($P<0.01$), physical ($P<0.05$), and psychosocial ($P<0.03$) health-related quality of life
Ohtsubo 1997 [38]	ExT *n*=7	Unilateral calf plantar flexion exercise	Smaller reduction in standardized intramuscular phosphocreatine and intracellular pH after training ($P<0.05$) New Borg scale improved ($P<0.05$)
Taylor 1999 [39]	ExT *n*=8	Training 3/week × 8 weeks	Ex time increased by 7.4%, peak O_2 uptake increased by 17.6% ($P<0.05$), peak cardiac index increased by 10% ($P<0.05$)
Owen 2000 [40]	ExT *n*=22 elderly patients (15 completed)	Training 1/week × 12 weeks	Distance walked in 6 min increased by 20% ($P<0.012$)
Maiorana 2000 [41]	ExT *n*=13	Circuit weight training program for 8 weeks	Peak O_2 uptake increased by 11.8% ($P<0.01$) Ex time increased by 18.4% ($P<0.01$) Maximal skeletal muscle isotonic voluntary contractile strength increased by 17.9% ($P<0.001$)

ex=exercise, ExT=exercise training, HR=heart rate, peak O_2 uptake=peak oxygen consumption, WL=work load

Unanswered questions

Although there are many reports of training, few have prospectively compared the elements of the training program. Important points that still need to be considered include the optimal frequency and intensity of training, the necessary duration for training effects and how long these effects last after cessation of a formal program. Other points that need to be addressed in future controlled trials include details of the most appropriate selection of patients, the elements of the training program and possible interactions with modern heart failure medications. Although cardiovascular medicine has greatly improved in the last decade as a result of randomized controlled trials of effective treatments, this oldest of physical therapies remains extremely difficult to evaluate. This is at least partly because our modern system of funding trials is firmly in favor of commercial patentable treatments. A program of government support for randomized non-commercial treatment trials is needed if these important questions and considerations are to receive a timely answer.

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