

Review Article

Beneficial Effects of Physical Training and Methodology of Exercise Prescription in Patients with Heart Failure

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The heart failure (HF) syndrome is rapidly becoming one of the most prevalent cardiovascular diseases in recent years and has a high mortality rate. Depending on the age group, the prevalence of HF may vary from 1-2% in persons aged 55-64 to 7-10% for those >75 years of age. One out of two HF patients usually dies within 5 years from the onset of the symptoms.¹

Prior to the late 1980s bed rest and restricted physical activity were recommended for all stages and forms of HF. However, prolonged bed rest and physical inactivity can lead to skeletal muscle atrophy, pulmonary embolism, venous thrombosis, and a further reduction in exercise tolerance and exacerbation of symptoms.² The concept of exercise training (ET) in patients with chronic HF developed in the late 1980s. Coats et al, in the first randomised study of training patients with stable HF, showed that eight weeks of ET led to an increase in exercise capacity and to an improvement of the abnormal sympathovagal balance.³ Research over the past 15 years has expanded our understanding and knowledge of the role of ET in patients with left ventricular dysfunction and HF. Exercise and physical activity were completely readdressed and revised in this setting.^{2,4,5} The purpose of

this review is to provide a brief overview of the beneficial effects of physical training and to discuss the current developments in the methodology of exercise prescription for HF patients.

Beneficial effects of physical training

Exercise intolerance and peripheral skeletal muscle myopathy are two of the most important clinical and physical symptoms of HF syndrome and are primarily attributed to peripheral limitations, such as endothelial and vascular dysfunction, persistent inflammation, decreased metabolic and oxidative capacity of the skeletal muscles, and reduced musculoskeletal blood flow and O₂ supply to active tissues.⁶⁻⁸ In addition, autonomic nervous system imbalance⁹ and weakened central haemodynamics¹⁰ form the pathophysiological basis of the impaired cardiovascular function and decreased physical capacity of HF patients.

Physical training results in significant peripheral adaptations and in limited but important central haemodynamic changes, which contribute to curbing or even reversing the development of the HF syndrome. There follows a brief outline of the beneficial effects of ET in chronic HF (Figure 1).

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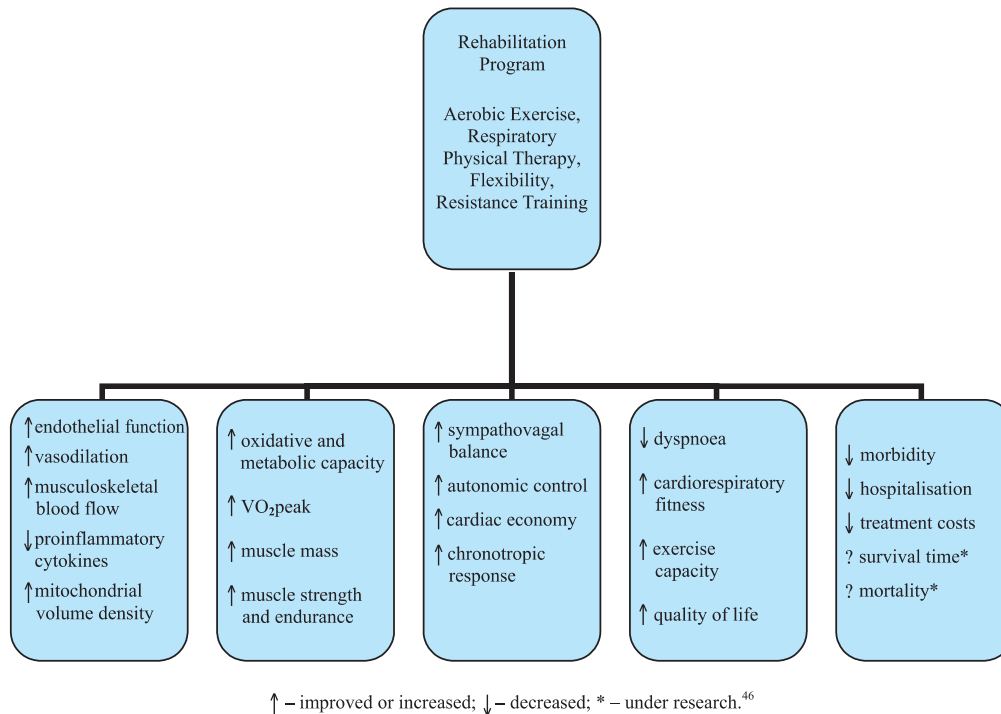


Figure 1. Beneficial effects of physical training in heart failure.

Vascular, metabolic and anti-inflammatory effects (Table 1)

Systematic aerobic exercise training (AT) increases capillary density, promotes the synthesis and release of nitric oxide, improves angiogenesis, vasodilation and endothelial function, reduces oxidative stress and peripheral vascular resistance in active tissues, and increases metabolic capacity and musculoskeletal blood flow.^{6,7,11}

Exercise training of HF patients is associated with lower blood lactate threshold and blood lactate con-

Table 1. Vascular, anti-inflammatory and metabolic effects of exercise training in heart failure.

- Decreased oxidative stress
- Improved vasodilatory capacity and endothelial function
- Reduced systemic vascular resistance
- Increased musculoskeletal blood flow
- Inhibition of elevation of proinflammatory cytokines
- Reduced skeletal myocyte apoptosis
- Increased IGF-1 expression in skeletal muscles
- Attenuation and/or reversal of skeletal muscle atrophy
- Increased mitochondrial volume density
- Increased citrate synthase activity
- Improved oxidative and metabolic muscle capacity

centration at standardised submaximal workload levels,¹² reduced depletion and more rapid resynthesis of phosphocreatine,⁶ and increased citrate synthase activity.⁷ Following AT, the mitochondrial total volume density is increased, mitochondrial enzymatic activity and skeletal muscles' oxidative capacity are enhanced.^{7,12,13} In addition, recent data indicate that continuous AT improves phase II O₂ kinetics, which are an indirect index of muscle oxidative capacity.¹⁴ In some studies, a shift from IIb to IIa muscle fibre type⁷ and a modest increase in type I muscle fibres¹⁵ have been reported. Combination training (CT), that is AT combined with resistance training (RT), improves both oxidative and glycolytic skeletal muscle capacity,¹⁶ increases the cross-sectional area of both these muscle types,¹² and improves neuromuscular function, all leading to a significant increase in muscle strength and endurance.^{16,17}

It is believed that persistent inflammation may contribute to skeletal muscle myopathy and the pathogenesis of chronic HF. Long-term physical training, as opposed to acute bouts of exercise, seems to have important anti-inflammatory effects.^{6,8,18} Recent data indicate that exercise has a potent downregulatory effect on platelet-mediated inflammation, decreasing plasma levels of soluble CD40 ligand and P-selectin in

chronic HF patients.¹⁹ Physical training seems to modulate beneficially the immune responses of HF, as expressed by elevated circulating proinflammatory cytokines.²⁰ This in turn results in increased anti-apoptotic factors and pronounced sensitisation of insulin-like growth factor-1 (IGF-1) activity, known to be associated with reduced skeletal myocyte apoptosis and reversal of muscle atrophy.^{6,21}

Molecular effects of exercise training

So far there are only a limited number of available studies that investigated the molecular effects of physical training and the exercise-induced cardiac and skeletal muscle cell remodelling in HF.

Animal studies demonstrated that ET attenuates the extent of apoptosis and alters the expression of contractile and heat shock proteins.²² Treadmill exercise has been reported to activate the c-jun n-terminal kinase (JNK) signalling pathway—a family of intracellular signalling molecules that are activated in response to cellular stresses—in the rat heart.²³ Systematic ET may have the potential to reduce the expression of the atrophy-related ubiquitin-proteasome pathway, which is vital for the breakdown of contractile proteins.²⁴

Physical training seems to improve muscle protein synthesis through activation of the P13K/Akt molecular signalling pathway as a result of an increase of IGF-1 expression in skeletal and cardiac muscles.⁷ In addition, ET may attenuate protein degradation by inhibiting fork-head transcription factors (FOXO), which are related to proteolysis-mediated muscle atrophy.⁷ Exercise training activates several favourable signalling pathways that lead to increased peroxisome proliferator-activated receptor gamma co-activator (PGC-1 α) expression in skeletal and cardiac muscles, a transcriptional factor which in turn regulates mitochondrial biogenesis and fatty acid oxidation.²⁵ Finally, ET is associated with increased expression of vascular endothelial growth factor (VEGF), promoting angiogenesis and relieving hypoxia, to which cardiac and skeletal muscles are prone in HF.^{7,25}

Impaired calcium cycling may underlie contractile dysfunction of the failing heart.²⁶ However, little is known about possible ET effects on sarcoplasmic reticulum calcium regulation in HF. Elucidating the mechanisms responsible for disturbed excitation-contraction coupling in the failing heart²⁶ and understanding the signalling pathways of the exercise-induced adaptations in skeletal and cardiac muscle cells will probably contribute to the development of

novel molecular and genetic therapies for cardiac disease in the future.

Improvement of sympathovagal balance and chronotropic response to exercise

Physical training results in a significant decrease of muscle sympathetic activity, lower levels of circulating catecholamines, increased vagal tone, and improved baroreceptor sensitivity.^{6,9} In addition, new evidence indicates that the exercise-induced enhanced central antioxidant mechanisms and improved endothelium-dependent vasodilation activate favourable biological and genetic adaptations that may lead to a decrease of sympathetic outflow and normalisation of autonomic function.^{11,27} Heart rate (HR), both at rest^{3,28,29} and at submaximum workload,^{3,30,31} declines, while the rate-pressure product at submaximum workload increases more gradually and to lower levels.^{3,30} The chronotropic response to exercise is improved: HR is higher at maximum workload^{32,33} and lower during recovery.³⁴

Cardiac function

Most studies agree that the impact of ET on central haemodynamics is either modest or unimpressive.⁵ Moreover, little correlation exists between functional capacity and ejection fraction or other haemodynamic indices at rest.⁷ These underline the argument that the peripheral vascular and metabolic adaptations are the major priority in HF interventions. On the other hand, central haemodynamics at peak effort correlate well with exercise capacity and their improvement must also be an important target for HF rehabilitation programmes. Indeed, AT has been reported to improve cardiac performance and to increase stroke volume and cardiac output at peak effort.^{10,32} In addition, AT may attenuate or even reverse left ventricular remodelling, and improve systolic and diastolic function.^{30,33,35} Recent data are inline with these findings, indicating that AT decreases end-diastolic and end-systolic volumes and increases left ventricular ejection fraction.^{11,36,37} However, other reports have pointed out that the exercise-induced increase in ejection fraction is either not significant or absent.^{29,31,32}

Increase in exercise capacity and peak O₂ uptake

Exercise training prolongs phase I O₂ kinetics,¹⁴ which are a valuable cardiodynamic index, and increases the oxygen uptake efficiency slope, which is an objective

measure of cardiopulmonary function reserve and has a high prognostic value in chronic HF.³⁸ Overall, AT, alone or combined with RT, significantly increases exercise capacity and VO_2peak .³² Indeed, there is accumulating evidence that CT maximises the beneficial effects of physical training on exercise capacity and VO_2peak .^{7,16} The exercise-dependent increase in VO_2peak usually ranges from 10-30%^{5,28-30} and rarely rises to 40-45%.¹¹ In addition, there have been reports of significant increases in VO_2 at the anaerobic threshold (up to 25%), in exercise time, and in maximum workload achieved (up to 40%).^{27,29,30,32,33} Patients with diastolic dysfunction may also benefit from ET; it was shown recently that physical training improved their functional capacity and increased VO_2peak by 30%.³⁹

Improvement of respiratory function

Aerobic training leads to a decrease of minute ventilation (VE) at submaximum workload, a lower respiratory rate, a lower tidal volume, a lower ventilatory equivalent for CO_2 (VE/ VCO_2) slope value, and a rise in the anaerobic threshold.^{28,29,33} In addition, CT of the upper extremities improves ventilation during exercise as well as respiratory muscle endurance, resulting in relief of breathlessness.⁴⁰ Exercise training reduces the perceived sense of dyspnoea both at rest and during exercise.² Inspiratory training increases the inspiratory muscles' strength and endurance⁴¹ and improves exercise capacity and quality of life.⁴²

Psychosocial benefits

Exercise training is an effective therapeutic approach to reducing the dyspnoea and pronounced muscle fatigue of HF patients, promoting their sense of independence and psychosocial health.^{2,13} Such a response, together with the markedly improved quality of life,^{11,28,30,32,43} is associated with less chronic illness behaviour, less depression, and improvement of their general sense of wellbeing. Recent findings show that ET may also be beneficial for patients with dyspnoea and diastolic dysfunction, significantly increasing their functional capacity and improving their quality of life.³⁹ The data so far indicate that exercise increases the survival time and reduces the mortality of heart failure patients.^{28,44,45} It is hoped that the large, randomised, multi-centre trial HF-ACTION, which is nearly completed and will soon report its findings,⁴⁶ will confirm the early results and allay the reservations held by some researchers.^{31,47}

It is worth stressing that most ET studies report great benefits to one or more quality of life parameters.^{43,48} Morbidity, hospital admissions, length of stay, and treatment costs are all significantly reduced for patients who participate in structured ET programs, compared to those who receive the usual clinical care for HF.^{43,44,47}

Exercise prescription

Patients with mild to moderate and stable HF, with an exercise capacity >3 metabolic equivalents (METs), may participate in rehabilitation programmes (Tables 2, 3). The majority of exercising HF patients have VO_2peak 11-19 $\text{mlO}_2/\text{kg}/\text{min}$, ejection fraction 20%-40%,

Table 2. Heart failure: relative criteria necessary for the initiation of exercise training.²

- Compensated heart failure for at least 3 weeks
- Ability to speak without dyspnoea (respiratory rate <30 breaths/min)
- Resting heart rate <110 bpm
- Less than moderate fatigue
- Cardiac index ≥ 2.1 L/min/m² (for invasively monitored patients)
- Central venous pressure <12 mmHg (for invasively monitored patients)

Table 3. Relative and absolute contraindications for exercise training in patients with stable chronic heart failure.²

Relative Contraindications

1. ≥ 1.8 kg increase in body mass over previous 1 to 3 days
2. Concurrent continuous or intermittent dobutamine therapy
3. Decrease in systolic blood pressure with exercise
4. New York Heart Association Functional Class IV
5. Complex ventricular arrhythmia at rest or appearing with exertion
6. Supine resting heart rate ≥ 100 bpm
7. Pre-existing comorbidities

Absolute Contraindications

1. Progressive worsening of exercise tolerance or dyspnoea at rest or on exertion over previous 3 to 5 days
2. Significant ischaemia at low rates (<2 METS, ≈ 50 W)
3. Uncontrolled diabetes
4. Acute systemic illness or fever
5. Recent embolism
6. Thrombophlebitis
7. Active pericarditis or myocarditis
8. Moderate to severe aortic stenosis
9. Regurgitant valvular heart disease requiring surgery
10. Myocardial infarction within previous 3 weeks
11. New onset atrial fibrillation

and usually belong to New York Heart Association (NYHA) classes II-III.^{2,45,47,49}

Exercise prescription should be individualised, tailored to the outcome of a symptom-limited cardiopulmonary exercise test and to the patient's clinical status. The main goals are the dynamic enhancement of cardiorespiratory fitness and the improvement of muscle strength and endurance, without excessive pressure or volume cardiac overload (Table 4).^{2,4,5}

All HF patients who are enrolled in rehabilitation programmes undergo cardiopulmonary exercise testing, based on protocols with gradually increasing intensity and a duration sufficient to elicit a full range of metabolic responses.⁵⁰ Cardiopulmonary exercise test measurements are of particular importance for the assess-

ment of ventilatory abnormalities, risk stratification, definition of the symptoms that set the upper limit of the exercise intensity, and precise determination of VO_2peak and HRpeak achieved.⁵¹ Treadmill exercise protocols are frequently chosen for patients who are to be enrolled in a continuous AT programme, while in the case of interval AT the Meyer's bicycle steep ramp test⁵² is usually used.

Throughout the course of the programme, the six-minute walk test is used as a simple and inexpensive assessment of functional capacity and physical symptoms. However, it is questionable whether the changes in six-minute walk are reliably connected with changes in VO_2peak and/or changes in physical symptoms in all categories of HF patients.^{53,54} Standardised questionnaires, such as the "Minnesota Living with Heart Failure", the "Chronic Heart Failure Questionnaire", the "Kansas City Cardiomyopathy Questionnaire", and the "SF-36" are usually used to assess the changes in clinical status and quality of life of HF patients.^{48,55} It must be pointed out, though, that there are no comparative evaluations of these instruments, no such instrument has measured quality of life in HF trials in a reliable or reproducible fashion, and their usefulness has been hampered by a lack of sensitivity to changes in health status.^{48,56}

Dyspnoea is usually assessed with the American Thoracic Society's 5-grade scale, or with the 10-grade Borg scale.⁵⁷ However, the reproducibility and the ability of these scales to evaluate changes in symptoms reliably is questionable. Recently, a new dyspnoea self assessment instrument – the Provocative Dyspnoea Assessment – has been proposed, which uses a five-point Likert scale to yield a final Dyspnoea Severity Score.⁵⁸

Most published ET programmes last 8-26 weeks, and the mode of exercise employs aerobic activities of continuous or interval type and often combines aerobic with resistance training.^{51,59-61} High intensity interval AT versus moderate continuous AT,¹¹ and CT versus AT alone have been found to be more effective in increasing VO_2peak , muscle strength and endurance, and to improve the overall functional capacity of HF patients.^{7,16,62,63} The intensity of exercise in continuous AT usually ranges from 60 to 80% of HRpeak , or 50 to 70%, and more rarely up to 80% of VO_2peak .^{45,59,61} When interval AT is chosen, exercise intensity may be increased up to 90% of HRpeak ,¹¹ or up to 70-80% of maximum short time exercise capacity,^{2,52} as determined by Meyer's steep ramp test. In a number of studies, periods of intensive ET are followed by long periods of moderate exercise in or-

Table 4. Heart failure: outline of exercise training.

Phase I: Adaptation to exercise

- Hospitalisation (if necessary, acute heart failure)
- Inpatient physical training under supervision
- Respiratory physical therapy, inspiratory muscle training
- 3-15 min of exercise for 2-4 s/d, 2-3 METs, for 3-5 weeks

Phase II: Improvement of cardiorespiratory fitness

- Outpatient physical training under supervision
- Respiratory physical therapy, inspiratory muscle training
- Flexibility - stretching exercises, main muscle groups,
- 2-4 repetitions for 10-20 seconds each, mild discomfort
- Moderate aerobic training at 40-70% of VO_2peak
- 20-40 min of exercise, 3-4 s/w, for 3-4 months.
- Callisthenics - Mild strengthening exercises, 4-6 muscle groups,
- 1-2 sets of 10 repetitions each, at 40-50% of 1-RM

Phase III: Dynamic increase of physical capacity

- Outpatient physical training - rehabilitation centre
- Vigorous continuous aerobic training at 60-80% of VO_2peak
- Intense interval training at 70-90% of VO_2peak
- 30-50 min of exercise, 4-5 s/w for 3-6 months
- Flexibility - stretching exercises, main muscle groups,
- 3-5 repetitions for 15-30 seconds each
- Moderate strengthening exercises, 6-10 muscle groups,
- 2-3 sets of 10-15 repetitions each, at 50-60% of 1-RM

Phase IV: Maintenance of the training effects

- Long-term community or home training - independent exercise
- Moderate aerobic exercise at 60-70% of VO_2peak
- 30-40 min of exercise, 3-4 s/w
- Moderate resistance training
- Leisure time activities - recreational sports

MET – metabolic equivalent of the task, s/d – sessions per day; s/w – sessions per week; 1-RM – one repetition maximum.

der to maintain the beneficial effects of physical training.^{47,49,61}

Inpatient rehabilitation - phase I

Phase I involves heart failure patients who need hospitalisation. It commences immediately after their haemodynamic stabilisation in the intensive care unit and continues in the hospital's rehabilitation department. Stable HF patients may also participate in a phase I ET programme as outpatients, as a way of gradually adapting to exercise demands. By hospital discharge, patients should demonstrate an understanding of physical activities that may be inappropriate, be counselled to identify abnormal signs and symptoms suggesting the need for medical review, and be educated in the management of possible limitations in their daily life activities.

During phase I, the rehabilitation programme aims to counter the deleterious effects of bed rest, improve functionality, achieve a safe exercise capacity >3 METs, and prepare the patient for a safer return to daily life activities. Exercise prescription must define the appropriate modes, intensity, duration, frequency and rate of progress (Table 5):

- **Modes** of exercise include self-care activities, simple voluntary exercises with increasing range of motion, and aerobic activities, such as walking and mild interval cycling.
- Exercise **intensity** ranges between 1-3 METs. The HR during exercise must not exceed 20 bpm above HR_{rest}. The rating of perceived exertion, based on the 6 to 20 Borg scale, should not exceed 11 (light exertion).⁵⁷
- The **duration** of exercise initially ranges from 3-5 minutes and increases progressively to 15 minutes.
- The **frequency** of exercise in the intensive care unit is 3-4 short lasting sessions per day, followed by 1-2 sessions per day in the physical therapy department.^{57,64}

Exercise is terminated immediately if HR increases more than 20-30 bpm above HR_{rest}, or on the appearance of signs and symptoms that suggest cardiovascular insufficiency and deterioration in the patient's clinical condition (Table 6).

Outpatient rehabilitation - phase II

All HF patients who satisfy the criteria listed in Tables 2 and 3, who have met the (inpatient) phase I rehabilitation goals, and who have an exercise capacity >3-4 METs, may continue exercising in phase II.⁶⁴

Table 5. Phase I: Activity guidelines for hospitalised heart failure patients at different Rate of Progress levels.

Level	Activity
1	Bed rest, supplemental O ₂ , continuous positive airway pressure (CPAP) or biphasic positive airway pressure (BiPAP).
2	Bed rest, respiratory physical therapy, positioning, relaxation techniques.
3	Bed rest, range of motion exercises, inspiratory muscle training.
4	Chair, mild resistance and flexibility exercises.
5	Room ambulation.
6	Hallway ambulation, stationary cycling: ± 2 METs, 1-5 min, 2 s/d.
7	Physical Therapy Dept, warm-up and cool-down exercises, stationary cycling: ± 2 METs, 3-8 min, 2 s/d.
8	Physical Therapy Dept, stationary cycling: ± 3 METs, 3-10 min, 2 s/d, stretching and strengthening exercises.
9	Physical Therapy Dept, stationary cycling: 3-4 METs, 5-15 min, 2 s/d. Climbing stairs (1 to 2 floors). Management of possible limitations in daily life activities.
10	Understanding of physical activities that may be inappropriate. Patient discharged, participation in phase II rehabilitation program.

MET – metabolic equivalent of the task; s/d – sessions per day.

After a thorough and detailed evaluation, the participants are trained, as outpatients, in an appropriately organised and equipped rehabilitation department under medical and clinical supervision. Medical supervision is strongly suggested, since apart from the patient's precarious clinical condition a variety of symptoms may appear, including hypotension, arrhythmias, exercise-induced angina, or other symptoms of cardiovascular

Table 6. Physical training in heart failure: criteria indicating the need for exercise discontinuation.^{2,50,65}

- HR during exercise > HR_{rest} + 20 bpm
- Plateau or decrease in SBP >10 mmHg
- Poor pulse pressure (<10 mmHg difference between SBP and DBP)
- Respiratory rate of >40 breaths.min⁻¹ during exercise
- Development of an S₃ or pulmonary rales
- Increase in pulmonary rales
- Increase in the second component of the second sound (P₂)
- Significant ventricular or atrial dysrhythmias
- Second or third-degree heart block
- Onset of angina, ischaemic ECG changes
- Marked dyspnoea or fatigue (≥14 on Borg scale)
- Diaphoresis, pallor or confusion

DBP – diastolic blood pressure; HR – heart rate; SBP – systolic blood pressure.

decompensation.^{2,65} Medical supervision should comprise: pulmonary and cardiac auscultation; check for body weight, peripheral oedema; monitoring of HR, blood pressure, rhythm and symptoms (before and during training); pulmonary and cardiac auscultation (after training).⁶⁵

The main goals of phase II rehabilitation are the enhancement of cardiorespiratory and muscle fitness, attainment of a safe exercise capacity >5-6 METs, limitation of dyspnoea and early muscle fatigue, psychological support and improvement of the patient's quality of life. The essential components of a systematic, individualised exercise prescription include the following.

Modes of exercise

During warm up, range of motion and flexibility exercises are used. Flexibility training involves stretching the main muscle groups with 2-4 repetitions each, for 2-3 times per week, holding at a position of mild discomfort for 15-30 s.^{64,65} Throughout the dynamic phase of the session, combined callisthenics and aerobic exercises are prescribed. The use of a bicycle ergometer at a power setting up to 60-75 W (4-5 METs) is the ideal means of exercising for most HF patients, especially when interval AT is chosen.⁵² Alternatively, walking on a treadmill ergometer at a speed up to 6-7 km/h (4-5 METs) can be recommended. Mild strengthening exercises, for 1-2 times per week, can be added as phase II progresses.

Other popular free activities, such as running or intense outdoor cycling, are not advisable for HF patients, because they are associated with exaggerated cardiovascular and metabolic responses, corresponding to bicycle ergometer exercise at a power of 80-150 W.² Patients with chronic diastolic or systolic dysfunction should refrain from swimming and water exercises with head-out water immersion, since the increased hydrostatic pressure results in left ventricular overload and in increased pulmonary capillary wedge pressure.^{9,60,66}

Initially, patients with limited exercise tolerance cannot exercise continuously for a satisfactory length of time, and for this reason interval AT is suggested, with the continuous method reserved for later. Although the majority of published exercise trials use continuous AT, it has been shown that intensive interval AT causes a stronger peripheral muscle stimulus and a more rapid improvement in VO_2peak and in the anaerobic threshold, without inducing excessive cardiovascular stress.^{2,11,49,52}

Intensity of exercise

During phase II, the intensity of effort in continuous AT should range from 40-70% of VO_2peak and/or 50-70% of heart rate reserve (HRR).^{5,49,51,60,65} In interval treadmill exercise, intensity may increase up to 80% of VO_2peak or 80% of HRR for 3-5 min, followed by walking periods of mild intensity (<50% of HRR).¹¹ The rating of perceived exertion, based on the 6 to 20 Borg scale, must not exceed 13 (somewhat hard).^{5,49} Dyspnoea during exercise should not exceed +2 (mild, some difficulty) on the five-grade dyspnoea scale,⁶⁷ while on the 10-grade scale it should be less than 3 (moderate).⁵⁷

The regulation of exercise intensity should preferably be based on VO_2peak attained during cardiopulmonary testing. Monitoring intensity based only on rating of perceived exertion or on sole HR measurements, especially in patients who are taking beta-blockers, should be avoided, since HF patients have a poor chronotropic response to exercise.^{5,67} The exercise intensity must be prescribed with care, so that it will not lead to myocardial ischaemia, a decrease in ejection fraction, pulmonary capillary wedge pressure >20 mmHg, or exceeding the anaerobic threshold (Table 6).

In stationary cycling interval AT is recommended, with an exercise/relaxation cycle of 30/60 s and an exercise intensity of 50-60% of the maximum short time exercise capacity, based on Meyer steep ramp test. During cool down the patient continues to exercise, but at a very low intensity (10-15 W). This cycle can be repeated 10 times in a 15-minute session. On the treadmill, the patient can carry out interval AT initially with a 60/60s cycle, progressing to exercise cycles of 2-10 min with relaxation periods of 1-2 min. An indicative recommendation is a 60 s exercise cycle at a speed of 4 km/h followed by another 60 s of low-intensity exercise at a speed of 1.5 km/h.^{2,52,60}

Duration and frequency of exercise

Heart failure patients may exercise 3-5 times per week, for 20-40 min per session.^{51,60} Initially, the total duration of the session should not exceed 10 to 20 min (moderate interval AT). The session duration is gradually prolonged until continuous AT is given for 15-20 min, during the dynamic part of the session. Heart failure patients tend to have slower cardiovascular adaptation to exercise and slower recovery from a given work rate.⁵¹ Thus, the training plan should allow for long warm-up and cool-down periods (8-12 min),

in order to avoid blood pressure, ischaemic and cardiac rhythm disturbances.^{60,67} In general, progression of ET should be followed in the order: prolong the duration, then increase frequency, then augment intensity.

Phase II may last as long as 3-4 months. Usually, the initial benefits in aerobic capacity and muscle strength become apparent after the first month, while the improvement in the patient's functional capacity peaks between the 16th and 26th week of the programme (phase III).⁴⁹

Respiratory physical therapy in heart failure

Respiratory physical therapy is particularly useful and supplements the treatment protocol during rehabilitation phases I and II. The primary goals are the prevention of pulmonary complications, an increase in the respiratory muscles' strength and endurance, a lower respiratory rate, and the improvement of exercise capacity with less dyspnoea.⁶⁵ For outcome assessment, ratings of dyspnoea during exercise, maximum inspiratory and expiratory pressure should be measured before and after the training period.⁴² For this purpose, inspiratory muscle training via the "threshold" device is prescribed at an intensity of 15-60% of maximal inspiratory pressure, with a duration of 20-30 min, for 3-5 sessions per week, and for a period of 2-3 months.^{2,65,68} The programme may be supported with expiratory muscle training,⁶⁸ while in some centres yoga breathing has been used successfully, with a controlled inspiration-expiration rhythm and a respiratory rate of 15, 10, or 6 breaths per minute.^{2,60,65} Yoga exercises mobilise the diaphragm, abdominal and chest muscles in sequence. This may help to improve awareness of breathing, decrease resting respiratory rate, increase tidal volume and, as a consequence, help to diminish atelectasis.²

Outpatient rehabilitation - phases III and IV

All stable HF patients who have met phase I and II rehabilitation goals, with an exercise capacity >5-6 METs, may continue exercising in phase III. The main goals are the dynamic increase of cardiorespiratory and muscle fitness and the further improvement of patients' overall physical capacity.

In phase III, HF patients may be trained in rehabilitation centres outside hospital, under clinical supervision. Flexibility and continuous and/or interval AT with moderate strengthening exercises are used.⁶⁹ The exercise intensity ranges from 60-80% of VO_2 peak,

or 60-80% of HRR, when the continuous mode of training is selected.^{51,57,64,65} Interval AT in the form of stationary cycling is also used, with an exercise/recovery cycle of 15/60 s or 10/60 s 10-12 times in a 15 min session, up to an intensity of 70-80% of the maximum short time exercise capacity.^{2,70} Horizontal cycling at 12-13 km/h (4-5 METs) in normal weather conditions can be prescribed in stable HF patients with an exercise capacity >7 METs.^{2,60} Exercise duration ranges from 30 to 50 min, 4-5 times per week.

Discontinuation of physical training, or return to physical inactivity after completion of the ET program, is followed by a rapid loss of the physiological and functional benefits within several weeks or months.⁶⁰ In accordance with this, recent data indicate that the significant anti-inflammatory effects of a 20-week ET programme disappeared after one year without systematic exercise.¹⁹

In phase IV, systematic exercise aims to maintain the developed beneficial effects of physical training. In case of community or home exercise programmes, frequent communication with the treating physician is essential, while transtelephonic electrocardiographic monitoring may also be used. The HR during exercise and the energy expenditure are monitored with standardised HR watches and walking pedometers.⁷¹ In phase IV, 30-40 min of moderate exercise (brisk walking, callisthenics, stationary cycling and treadmill exercise) for 3-4 days per week and frequent bouts of leisure time activities and recreational sports are considered adequate for the long-term maintenance of the improved cardiorespiratory fitness and the increased physical capacity developed in the previous phases.

Resistance training in heart failure

Until the late 1990s, RT was contraindicated for HF patients, and the existing guidelines still view it with great caution.^{2,5,17,72} However, there is a growing body of evidence that RT is indeed safe for most HF patients^{7,16} and may even be more effective than AT alone in reversing peripheral metabolic limitations and skeletal muscle myopathy, and in increasing overall functional capacity.^{16,62,63} Patients with chronic, low-risk HF, in NYHA classes I-III, with moderate to good left ventricular function, without angina or ischaemic ECG disturbances and dysrhythmias, and without uncontrolled hypertension or moderate to severe aortic stenosis, may follow a moderate RT program. Mild strengthening exercises may be prescribed as soon as possible. How-

ever, essential prerequisites, such as AT for at least several weeks and an exercise capacity >5 METs, must be satisfied before the initiation of a more dynamic and structured RT programme for HF patients.^{17,72}

The European Society of Cardiology suggests the performance of rhythmic strengthening exercises using short bouts of work phases, with small numbers of repetitions, and a work-recovery ratio of 1:2.² The rhythmic sequence of the exercise, the unilateral activation of the upper and lower limbs, and the involvement of preferably just one muscle group per activity, ensure the gradual strengthening of the body without either marked increments in systemic vascular resistance or a significant reduction in left ventricular ejection fraction and stroke work index indicating an acute overloading of the left ventricle.^{2,49,60} In addition, shortening the isometric and lengthening the isotonic phase of the exercise may help to avoid haemodynamic strain.⁷² For increased patient safety, ECG, blood pressure and body weight must be monitored during the first 4-6 weeks of RT.

Depending on NYHA class, the strengthening of 4-10 muscle groups of the upper body, lower body and the trunk is suggested. Resistance training consists of 1-3 sets of at most 10-15 repetitions for each muscle group (or weight station), a contraction duration of 4-6 s (2-3 s for concentric, 2-3 s for eccentric phase), with adequate rest intervals (1-2 min) between the series, at an intensity of 40-60% of 1-repetition maximum (1-RM).^{16,17,63,72} The RT session may last 10-30 min and be repeated on 2-3 days per week. It must be pointed out that during effort breath holding (Valsava manoeuvre) must be avoided. In addition, regulating the intensity of RT based on maximum voluntary contraction should not be used. This method of intensity monitoring is associated with isometric training and requires a maximum isometric contraction test.

Finally, it is worth noting that the use of neuromuscular electrical stimulation in HF patients seems to improve muscular⁷³ and endothelial function,⁷⁴ and increase exercise capacity.⁷³

Conclusions

Structured and systematic physical training must be an essential part of the overall clinical care of chronic heart failure syndrome. Long-term exercise results in peripheral vascular, oxidative and metabolic adaptations, in improvement of autonomic control, and in limited central haemodynamic changes. These beneficial effects may contribute to curbing and/or reversing the pathophysiological mechanisms that lead to the

occurrence or the deterioration of HF syndrome. The primary goal of exercise prescription is to develop an individualised exercise programme combining aerobic with resistance training in order to enhance cardiorespiratory and muscular fitness, increase total physical capacity, and improve the quality of life of HF patients. Exercise makes a decisive contribution to reducing morbidity, hospitalisation, treatment costs and a likely improvement in the patient's survival expectancy and mortality.

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