
Working Group Report

Recommendations for exercise training in chronic heart failure patients

Working Group on Cardiac Rehabilitation & Exercise Physiology and Working Group on Heart Failure of the European Society of Cardiology*

Historical background

The treatment of heart failure has changed dramatically during the years. The radical shift in concept is illustrated by the change from sympathomimetic drugs to beta-blocking agents, representing a striking example of how medical management of heart failure has evolved in an unanticipated fashion. Exercise and physical activity is another treatment area being completely readdressed and revised in this setting. Prior to the late 1980s, cardiac enlargement, decreased left ventricular systolic function and heart failure were considered absolute or relative contraindications to exercise training. Previous treatment strategies involved restriction of physical activity and bed rest for all stages and forms of heart failure^[1]. During acute exacerbation of the disease, rest was considered to be beneficial by causing increased renal blood flow, enhanced urine output, and pharmacological diuresis. Once stable, patients were advised to avoid exercise fearing a further decline in cardiac function. However, prolonged rest or inactivity can lead to skeletal muscle atrophy, further reduction in exercise tolerance, venous thrombosis, pulmonary embolism, decubitus, and exacerbation of symptoms.

The concept of exercise training in patients with heart failure developed in the 1980s, following a period of intense evaluation of the safety and efficacy of exercise rehabilitation in patients with stable coronary artery disease and based on a better understanding of peripheral adaptation in heart failure. Initial studies among coronary patients with significant left ventricular dysfunction reported a significant improvement in exercise capacity without major detrimental effects^[2–4]. Research over the past 10 years expanded our understanding and knowledge about the role of exercise training in patients with left ventricular dysfunction and heart failure.

It is now well established that exercise intolerance in patients with chronic heart failure has a multifactorial aetiology and that changes in the periphery rather than left ventricular performance itself are important determinants of exercise capacity^[5]. In addition, an increasing body of evidence has demonstrated that neurohormonal interaction between the periphery and the heart are also important determinants of symptoms and prognosis in these patients^[6,7]. Indeed, therapeutic interventions that alter these interactions are now accepted as valuable therapeutic approaches: exercise should be considered one of these. During the last decade a long list of small trials has been published, all indicating a variety of impressive benefits that could be achieved with physical training^[8–28]. These included increased peak oxygen consumption (VO_2), improvement in the respiratory function and in the autonomic control of the circulation associated with reduction in sympathetic nervous system activity and enhancement in vagal activity^[8–14]. More recent reports have documented an increase in endothelial function and in skeletal muscle biochemical and histological characteristics^[12,15–18]. All these effects lead to significant improvement in exercise tolerance, partial relief of symptoms such as dyspnoea, fatigue, sleep disturbance, and muscle weakness, improved New York Heart Association (NYHA) functional class and perception of quality of life and symptom severity. No significant deleterious effects, or significant deterioration in central haemodynamics have been reported with exercise training^[8,9,11,22–27] and a possible attenuation of unfavourable left ventricular remodelling has also been documented^[22,23]. Without question, selected patients can safely undergo and derive benefit from an exercise training programme.

Despite these experiences in small series of dedicated expert groups in the field, the effect of exercise training on morbidity and mortality in this patient population is limited^[28]. Given that both the inability to increase cardiac output during exercise and a reduction in peak VO_2 are associated with haemodynamic dysfunction and poor short-term survival, one might speculate that an intervention capable of improving exercise tolerance and at the same time of attenuating the neuroendocrine over-activity which is characteristic of

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the syndrome would have a favourable impact on prognosis. Such a relationship, however, has not yet been fully demonstrated.

Exercise training effects in chronic heart failure

Exercise capacity

Exercise duration was used as an initial measure of exercise capacity in patients with heart failure. However, due to concerns surrounding accuracy and reliability, it is now common practice to assess exercise capacity by determination of peak VO_2 . Compared with age-matched normal controls, peak VO_2 is reduced in patients with heart failure^[29,30]. Reasons for the reduced VO_2 include a reduced exercise cardiac output response, reduced nutritive blood flow to the active skeletal muscles, and skeletal muscle abnormalities such as a reduced percentage of Type I fibres, diminished oxidative capacity, and decreased capillary density. Metabolic abnormalities have also been described, including early dependence on anaerobic metabolism, excessive early depletion of high energy phosphate bonds and excessive early intra-muscular acidification^[31,32]. Biopsy studies have confirmed defects in oxidative and glycolytic enzymes which may explain these metabolic abnormalities^[32,33]. These histological and metabolic disorders are in part similar to those seen in severe deconditioning, and can be at least partially prevented by regular exercise training^[12,16–18]. Because of these changes, sub-clinical muscle wasting (atrophy) and muscle function abnormalities in terms of early fatigability and reduction in maximal strength are very common in heart failure^[34–36]. One underlying mechanism appears to be a progressive decrease in physical activity to avoid symptoms; however, a disease-related increase in plasma cytokine levels has been suggested. Indeed, insulin resistance, elevated levels of tumour necrosis factor- α , and excessive noradrenaline levels may also contribute to skeletal muscle abnormalities, which may lead to severe skeletal muscle wasting that is characteristic of the catabolic state of the syndrome of cardiac cachexia in advanced heart failure^[36].

Early respiratory muscle de-oxygenation and fatigue have also been described in heart failure^[37]. These changes may lead to excessive ventilatory effort, to inefficient ventilation with ventilation/perfusion mismatch and unpleasant dyspnoea. Although the respiratory muscle changes are similar to those in the skeletal muscle, cytokine or neuroendocrine catabolic effects, rather than inactivity alone, are more likely to contribute to these abnormalities.

Initial uncontrolled reports have indicated that patients with heart failure show improved exercise tolerance, leg blood flow and parameters of ventilatory function after exercise training^[8,9]. Since then, numerous randomized trials in patients with stable class II-III

heart failure showed improvement in peak VO_2 with exercise training^[10–13]. These findings were confirmed in many subsequent reports^[19–28] evaluating the mechanisms of the training-induced improvements in exercise capacity. Importantly, most of these trials involved patients taking ACE inhibitors, diuretics, and digitalis, and relatively few patients were receiving beta-adrenergic blockade therapy. Thus the 15% to 25% increase in exercise capacity that occurs with exercise training is in addition to what is expected with standard medical therapy. The mechanisms responsible for the exercise training-induced increase in peak VO_2 represent an integration of several factors. Myocardial function is improved, as evidenced by increases in peak cardiac output response^[8,11,25,27]. However, changes in central transport do not solely explain the increase in peak VO_2 after training. Less vasoconstriction of the nutritive arterioles within the active skeletal muscles, together with improved oxygen extraction and metabolic function at the active skeletal muscle, are also involved^[8,12,17]. In fact, a lower blood lactate concentration at a fixed submaximal work rate can be documented after exercise training. Time to ventilatory-derived anaerobic threshold and oxygen consumption at this threshold are also increased with training, suggesting delayed reliance on anaerobic pathways for energy production^[8,9,12,19,38].

Improvements in peak exercise capacity and an improved ability to tolerate submaximal exercise represent clinically important and expected outcomes that occur in response to exercise training in stable patients with mild-to-moderate heart failure.

Myocardial function

Improved maximal cardiac output has been reported in some but not all studies of training in heart failure. A factor contributing to the increase in peak cardiac output is partial reversal of the chronotropic incompetence that is characteristic of patients with heart failure: a modest 4% to 8% increase in peak heart rate after exercise training has been documented^[12,13,25,26,38]. The training-induced increase in stroke volume is also modest and may be due to left ventricular dilation^[27]. By contrast, an improvement in diastolic filling rate has been reported that was significantly correlated with an increase in cardiac index at peak exercise^[20]. Left ventricular contractility after training appears unchanged both at rest and during exercise^[22,23,26]; however, a significant improvement in thallium uptake and in left ventricular contractile response to low-dose dobutamine infusion has been documented^[39]. No effect on left ventricular ejection fraction has been reported in stable heart failure. Importantly, in contrast to early negative observations in a small series of patients early post infarction^[40] and contrary to widespread concerns in the cardiology community, no significant changes in left ventricular volumes, wall thickness and thinning of the infarcted area have been documented^[22,26], and a

possible attenuation of unfavourable left ventricular remodelling with long-term exercise training has been suggested in patients with left ventricular dysfunction after myocardial infarction^[23].

Peripheral blood flow

Patients with heart failure exhibit a subnormal peripheral blood flow response to exercise. Skeletal muscle blood flow is normal, or slightly reduced at rest, but the increase during both submaximal and maximal exercise is 20% to 40% below that of normals^[29,41]. This limitation is due to an impaired ability to vasodilation (or to less vasoconstriction) during exercise in the metabolic active skeletal muscle. Two primary reasons for this reduction in nutritive blood flow are a decreased release of endothelium-derived relaxing factor (nitric oxide vasodilator system) from arterioles supplying the metabolic active muscles, and an exaggerated increase in vasoconstrictive neurohormones such as endothelin, norepinephrine, renin, angiotensin II, and vasopressin (vasoconstrictor system)^[42]. Although not exhaustively studied in heart failure, there is evidence that regular physical exercise can improve endothelium dysfunction, reduce peripheral vascular resistance and increase skeletal muscle blood flow^[8,15,43]. Whether this is predominantly obtained by reducing vasoconstrictor tone, by improving large vessel function or by enhancing endothelial vasodilator function is not known.

Skeletal muscle function

Partial corrections of skeletal muscle abnormalities in heart failure have been demonstrated with exercise training. These changes have been seen in histology, mitochondrial structure, oxidative enzymes and in magnetic resonance spectroscopy. Several trials using a limb-training model have documented significant improvements in muscle function as measured by muscle strength, exercise time, phosphocreatine resynthesis and depletion rates, adenosine diphosphate concentration, intracellular pH, and inorganic phosphate/phosphocreatine ratio^[8,12,16–18]. The over-activity of the skeletal muscle ergoreflex effects on haemodynamic and ventilatory control during exercise have been shown to be partially corrected by single limb training^[44].

Skeletal muscle wasting appears to be reversed with physical training, although this has not been firmly documented as yet. Although the favourable changes in skeletal muscle abnormalities have been related to training-induced improvements in VO_2 at both ventilatory-derived anaerobic threshold and peak exercise, the relevance of skeletal muscle improvements to improved exercise tolerance has not been fully elucidated (despite likely interaction).

Ventilatory function

Patients with heart failure have altered ventilatory responses that contribute to the early onset of dyspnoea

with exercise. However, arterial PO_2 , PCO_2 , and O_2 saturation are usually maintained in the normal range during exercise unless significant baseline pulmonary disease is present^[45,46]. Although the origin of dyspnoea is multifactorial, the increased ventilation (VE) plays a role. In patients with heart failure, VE during exercise is somewhat greater per unit of VO_2 , which is achieved by a higher breathing frequency. In addition, measurements of ventilatory exchange during exercise suggest that the ratio of dead space/tidal volume (VD/VT) is increased. These findings infer that mild ventilation/perfusion defects develop during exercise, contributing to a higher VE to carbon dioxide production (VCO_2) relationship observed in these patients^[45,46]. Changes in the skeletal muscle lead to the early onset of acidosis during exercise and result in increased VCO_2 . This in turn may lead to peripheral chemoreceptor stimulation of central ventilatory control centres, causing an increase in VE and dyspnoea during exercise. In addition, early activation of muscle ergoreflex contributes to an excessive increase in sympathetic activation and exercise hyper-ventilation^[6,44].

Exercise training reduces ventilatory abnormalities in patients with heart failure, decreasing VE at a fixed submaximal work rate and normalizing the slope relating VE to VCO_2 ^[11,19,38]. These changes lead to substantial improvements in overall ventilatory efficiency, and a reduction in the perceived sense of dyspnoea. Respiratory muscle training has been reported to give specific benefits and is associated with improved exercise tolerance^[47], either when integrated in a generalized training protocol of whole body dynamic exercise or as specific respiratory muscle training.

Neuroendocrine and autonomic nervous system activity

Early compensatory responses in heart failure are provided by neuroendocrine activation, which leads to characteristic remodelling of both the peripheral vascular system and cardiac chambers, progressive attenuation of the arterial and cardiopulmonary baroreflexes, and downregulation of cardiac beta1 receptors. In addition, cardiac parasympathetic activity is inhibited, which contributes to sinus tachycardia, loss of RR interval variability, and attenuation of heart rate and vasomotor responses to baroreflex stimuli.

Exercise training among patients with chronic heart failure reduces the activity of the sympathetic and renin-angiotensin system^[10,11,14]. Abnormalities in nor-adrenaline spillover, heart rate variability and heart rate responses during exercise are at least partially corrected. However, the impact of exercise training in patients on beta-blockers remains incompletely evaluated at this time and needs further evaluation.

Health-related quality of life

Patients with heart failure experience progressive disability and decline in health-related quality of life, both

Table 1 Relative and absolute contraindications to exercise training among patients with stable chronic heart failure

Relative contraindications	Absolute contraindications
<ol style="list-style-type: none"> 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 beats \cdot min⁻¹ 7. Pre-existing comorbidities 	<ol style="list-style-type: none"> 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

of which are associated with dyspnoea and fatigue during routine activities of daily living. In addition, feelings of depression and isolation are common. Even in optimally treated patients, symptom relief is often not complete and quality of life suffers. No strong correlation exists between measures of health-related quality of life and either central haemodynamic abnormalities or exercise intolerance^[5]. Exercise training is an effective treatment to improve quality of life. As patients become more tolerant of exertion, they experience less fatigue and dyspnoea and become more comfortable performing tasks of daily living. Such a response is associated with increased independence, less chronic illness behaviour, less depression, and improvement of their general sense of well-being^[10,11,23,48].

Exercise programming

Despite the increased attention to moderate exercise training in the management of patients with stable chronic heart failure, standardized guidelines for exercise training in these patients have not been previously established, although some recommendations have been suggested^[49]. Exercise training methods used for studies and clinical practice have been largely based on recommendations for fitness training or on rehabilitation experience with cardiac patients with good exercise tolerance and arbitrarily modified for chronic heart failure patients^[50-54].

Any recommendation for exercise training in heart failure should be based on the particular pathology of the patient, the individual's response to exercise (including heart rate, blood pressure, symptoms, and perceived exertion), and measurements obtained during cardiopulmonary exercise testing. Additionally, patient's individual status including current medication, risk factor profile, behavioural characteristics, personal goals, and exercise preferences should be taken into consideration when exercise training is recommended. Based on the central, peripheral and metabolic alterations in chronic heart failure, the premise of rehabilitation is to apply both aerobic and strength training, with sufficient

exercise stimuli to skeletal muscles, without producing a significant load on the cardiovascular system.

Eligibility criteria and patient evaluation

All published studies of exercise training in heart failure have enrolled patients with stable chronic heart failure. No trial to date has evaluated exercise training for patients with unstable heart failure, nor has any study recruited a significant number of patients with NYHA class IV heart failure. Relative and absolute contraindication to exercise training are listed in [Table 1](#). Eligible patients are those with stable chronic heart failure identified as NYHA class II or III. A limited number of carefully selected and motivated Class IV patients free of dyspnoea at rest might also be eligible to participate; however, a great deal of supervision during exercise is mandatory in such patients. The recommendations for developing an exercise prescription are typically aimed at chronic heart failure patients with an ejection fraction less than 40%, consistent with the inclusion criteria of the majority of the exercise training studies conducted in patients with heart failure ([Table 2](#)). However, by no means do we intend to imply that patients with an ejection fraction above 40% do not benefit from an exercise training programme. No minimal left ventricular ejection fraction criteria for exercise training have been published. The reported mean ejection fraction ranged from 18% to 35%, with a range down from 5% to 11%, and in a controlled post-infarct study patients with the lowest ejection fraction benefited most from training^[55]. It appears that the benefits of training are not dependent on the aetiology of heart failure. However, specific contraindications include obstructive valvular disease, especially aortic stenosis or active myocarditis, either viral or autoimmune.

Little is known about exercise training in heart failure due to regurgitant valvular disease. Significant valvular heart disease should be corrected by surgery first and then the patient may be considered for exercise training when left ventricular dysfunction is still severe and associated with heart failure. Secondary mitral valve

Table 2 Summary of randomized exercise training studies in (LV) dysfunction and heart failure

Study	n pts	Clinical characteristics
Coats 1990 ^[10]	11	Stable CHF, IHD, synus rhythm, NYHA II–III
Jetté 1991 ^[55]	18	10 wk after the 1st anterior MI no previous history of
Koch 1992 ^[76]	25	Stable CHF, IHD or IDCM, NYHA II–III
Meyer 1991	12	Stable CHF, sinus rhythm, NYHA II–III
Coats 1992 ^[11]	17	Stable CHF, secondary to IHD sinus rhythm, NYHA
Davey 1992	22	Stable CHF, NYHA II–III
EAMI Study, Giannuzzi 1993 ^[22]	31	5 wk after 1st anterior MI, NYHA II–III
Adamopoulos 1993 ^[17]	12	Stable CHF, NYHA II, III
Belardinelli 1995 ^[13]	27	Mild CHF, IHD or IDCM, NYHA II–III
Belardinelli 1995 ^[13]	55	Stable CHF, IHD or IDCM, NYHA II–III
Hambrecht 1995 ^[12]	22	Stable CHF, IHD or IDCM, NYHA II–III
Kiilavouri 1995 ^[14]	20	Stable CHF, IHD or IDCM, NYHA II–III
Kiilavouri 1995 ^[14]	27	Stable CHF, IHD or IDCM, NYHA II–III
Meyer 1996 ^[19]	18	Stable CHF, IHD or IDCM, NYHA II–III
Meyer 1996 ^[19]	16	Stable CHF, IHD or IDCM, NYHA II–II
Piepoli 1996 ^[44]	12	Stable CHF, NYHA II–III
Magnusson 1996	11	Stable CHF, IHD or IDCM, NYHA II–IV (1 pt)
Hambrecht 1997 ^[18]	18	Stable CHF, IHD, NYHA II–III
ELVD Study, Giannuzzi 1997 ^[23]	77	3–4 wk after 1st MI, no history of HF
Willenheimer <i>et al.</i> 1998	49	Stable CHF, IHD or IDCM, 8 'Boston points;
Belardinelli 1999	99	Stable CHF, IHD or IDCM, NYHA II–III
CHANGE study, Wielenga <i>et al.</i> 1999	80	Stable CHF, IHD or IDCM, NYHA II–III
CHANGE study, Wielenga 1999 ^[64]	80	Stable CHF, IHD or IDCM, NYHA II–III
ELVD CHF just completed, Giannuzzi 1999	86	Stable CHF, IHD or IDCM, NYHA II–III

regurgitation is common in severely dilated left ventricles but can be operated on with reasonable peri-operative risk provided that basic contractility and cardiac output is not too severely depressed (i.e. cardiac index $>2.5 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$).

Since exercise may predispose to ventricular arrhythmias which are already very common in heart failure, patients with evidence of ventricular tachycardia or other serious ventricular arrhythmias on exercise or on 24-hour electrocardiographic monitoring have been generally excluded from controlled randomized studies. Until further information is available it seems prudent not to recommend exercise in heart failure patients who clearly demonstrate exercise-induced serious ventricular arrhythmias. Other common abnormalities that may limit responses to exercise training should be thoroughly evaluated. These include exercise-induced angina or silent ischaemia, marked hypotension and atrial arrhythmias. It is likely that patients with both angina and heart failure would also benefit from training. However, the possibility exists that exercise-induced functional deterioration in the presence of ischaemia may make the patient unable to exercise sufficiently to derive training benefits or may predispose to ventricular arrhythmias; such patient groups needs specific investigation before entering an exercise training programme with any confidence. In any case, all patients with heart failure should have their clinical status carefully reviewed by the cardiologist before starting an exercise programme. In addition to the history and physical examination to identify cardiac and non-cardiac problems that might limit exercise participation and other factors possibly contributing to exercise intolerance (anaemia, reversible

airway disease, peripheral vascular disease), a blood test for basic chemistries and electrolytes and a cardiopulmonary exercise test are indicated.

Modality

Aerobic exercise

Modes of exercise. In previous exercise training studies, and in clinical practice, walking, jogging, cycling, swimming, rowing, and calisthenics have been applied. Despite this wide range of activities, the pathology and exercise tolerance of patients with chronic heart failure only allow a few such selected activities to be performed.

Cycle ergometer training allows exercising at very low workloads, exact reproducibility of a prescribed and/or tolerated workload, and continuous monitoring of heart rate, rhythm, and blood pressure. Additionally, cycle ergometry was demonstrated to be ideal for applying the interval training method. These facts suggest that cycle ergometer training is to be recommended as the most favourable type of aerobic exercise for chronic heart failure patients in general, and particularly for those patients with severe exercise intolerance, a history of serious rhythm disorders, frequent need for change in diuretics, as well as those with obesity, orthopaedic and neurological, and/or age-related limitations for other types of exercise.

The application of a tolerated workload from cycle ergometer training to outdoor cycling is not possible because of environmental factors influencing cardiovascular stress (e.g. head wind, slopes). Even outdoor cycling on a plain track and at a very slow speed

(12 km . h⁻¹) requires almost 1000 ml . min⁻¹ VO₂, corresponding to approximately 50–60 W^[54]. This suggests that even outdoor cycling with minimal environmental stress factors can be recommended only for a small population of chronic heart failure patients (clinically long-term stable; high exercise capacity/tolerance).

Jogging at a speed of 80 m . min⁻¹ is the lower limit of speed which allows a comfortable movement. Because even this speed requires an oxygen consumption of 1200 ml . min⁻¹ or a corresponding exercise tolerance of ≥1 W . kg⁻¹ body weight, jogging is not advisable for chronic heart failure patients.

In contrast, a wide range of workloads in walking offers a promising applicability for patients at a broad range of exercise tolerance: low speeds of <50 m . min⁻¹, corresponding to 650 ml . min⁻¹ VO₂, required a low exercise tolerance of 0.3 W . kg body weight^[56], and a faster speed of 100 m . min⁻¹, corresponding to 900–1000 ml . min⁻¹ VO₂, required an exercise tolerance of 0.8–0.9 W . kg⁻¹^[54]. During swimming, head-up immersion and the hydrostatically-induced volume shift result in an increased volume loading of the left ventricle, with increase of heart volume, and pulmonary capillary wedge pressure. Swimming slowly (20–25 m . min⁻¹) results in measurements of heart rate, blood lactate, and plasma catecholamines similar to those measured during cycle ergometry at work loads of 100–150 W^[57]. Because of these findings, chronic heart failure patients with diastolic and systolic dysfunction should refrain from swimming.

Interval vs steady state exercise. The rationale for developing interval training methods for cardiac patients was to apply a more intense exercise stimuli on peripheral muscles than that obtained during steady-state training methods, but without inducing greater cardiovascular stress. This is possible by using short bouts of work phases in repeated sequence, followed by short recovery phases. In coronary patients with low exercise capacity^[58] aerobic exercise training using an interval method resulted in a more pronounced effect on the subjects' exercise capacity than using a steady-state training method. Additionally, chronic heart failure patients with very low baseline aerobic capacity demonstrated an improvement in ventilatory threshold (24% on average) and peak VO₂ (20% on average) after only 3 weeks of interval cycle ergometer training^[19]. This finding is similar to that reported by other studies but after much longer training periods (8–24 weeks) using steady-state method training^[11–13,21,39,59].

From a practical point of view, work phases of 30 s and recovery phases of 60 s are useful, using an intensity of 50% of maximum short term exercise capacity for work phases. During the recovery phase, patients may pedal at 10 W^[60]. There are other combinations of work/recovery phases such as 15 s/60 s and 10 s/60 s with 70% and 80% of maximum short-term exercise capacity, respectively, which were proven to be tolerable^[61]. During the initial three work phases, work rate should be successively increased to reach the final rate by the

fourth work phase. Depending on the work/recovery interval chosen, about 10–12 work phases are performed per 15-min training session^[61].

Although work rate is markedly higher during interval work phases than during steady-state exercise, during the course of a 15-min interval training, left ventricular ejection fraction increased significantly and by the same magnitude as that seen during steady-state exercise performed at the same average power output. Additionally, mean arterial blood pressure and heart rate are also similar to that seen in steady-state exercise, while blood lactate is significantly higher during interval exercise, indicating a greater peripheral training stimulus^[62]. Thus, interval exercise allows more intense exercise stimuli on peripheral muscles with no greater left ventricular stress than when using a steady state training method. Therefore, interval exercise is recommended for aerobic training in patients with significant baseline limitations related to chronic heart failure.

Although cycle ergometer is preferred for applying interval training, this method can also be performed on a treadmill. In this case, a practical way is to chose work and recovery phases of 60 s each. During work phases, walking speed is adjusted to that heart rate tolerated by the patient during interval cycle ergometer training. During recovery phases, walking speed should be as slow as possible^[63].

Determination of the work rate for interval work phases. Work rate for interval work phases is determined by a steep ramp test^[60]. The steep ramp test enables the determination of a patient's maximum short time exercise capacity.

For this steep ramp test, patients start with unloaded pedalling for 3 min, and the work rate is then increased by 25 W every 10 s. Because of the rapid increase of work rate, many patients can duplicate the maximum work rate from an ordinary ramp test (e.g. 10 W . min⁻¹ increments), that means they can perform 150 to 200 W during exercise periods of 60 to 90 s, without any complications^[60].

Intensity recommendation for steady-state exercise. There is no consensus, at present, as to which parameter is optimal for measuring intensity; likewise, controversy exists about the 'optimal' intensity which should be used for aerobic exercise training in chronic heart failure patients. At least three approaches have been used: % peak VO₂, % peak heart rate, and rating of perceived exertion.

In terms of VO₂, an intensity of 40–80% of peak VO₂ was applied successfully^[11–13,21,39,59], indicating that patients with a low initial exercise tolerance respond to a low exercise intensity. Since intensity and duration of exercise training are closely interrelated in terms of training effect expected, low intensity can partly be compensated for by training sessions of longer duration, or higher frequency.

The rationale for using heart rate as a guide to exercise intensity is based on the relatively linear

relationship between heart rate and VO_2 ^[54] exercise training programme. Intensities of 60–80% heart rate reserve^[21,64] and 60–80% of the predetermined peak heart rate were applied^[11,17]. In chronic heart failure patients, these heart rate recommendations did not take into account the impaired force–frequency relationship in myocardial performance^[65,66]. Since a long-term reduction in heart rate (associated with a change in diastolic function and myocardial metabolism) is of major importance for myocardial recovery^[65,67], the training heart rate should be as low as possible. To follow this rule, interval training is preferable because it allows high peripheral exercise stimuli without significant increase of heart rate.

In healthy subjects, training intensities of 40–80% peak VO_2 were demonstrated to correlate positively with ratings of perceived exertion (6–20 Borg Scale) between 12–15 (light-moderate/heavy)^[54,68]. In chronic heart failure patients, exercise intensities corresponding to rating of perceived exertion <13 (<somewhat hard) have been reported to be well tolerated, and successfully applied^[21].

Nevertheless, in chronic heart failure, patients' rating of perceived exertion should be considered merely as an adjunct to a training intensity determined by % peak VO_2 , heart rate, blood pressure, and symptom monitoring because the rating of perceived exertion determined during graded cardiopulmonary exercise testing may not consistently translate to the same intensity as that during exercise training, and a certain percentage of patients are unable to reliably use the rating of perceived exertion scale. Since during exercise, ratings of fatigue and dyspnoea are often differently perceived, both symptoms should be monitored separately.

Specifics. A small systolic blood pressure increase of only 10–20 mmHg might be acceptable if no concomitant symptoms such as dyspnoea, fatigue, and dizziness, unknown from previous exercise training sessions, occur.

More important than any intensity recommendation, exercise training must be diligently monitored, and terminated when an acute decrease in blood pressure, onset of angina, significant dyspnoea and fatigue, a feeling of exhaustion, and/or serious exercise-induced rhythm disorders are observed.

Duration and frequency

Intensity and duration of exercise are closely interrelated and frequency is related to both. In training studies with chronic heart failure patients, there has been an extensive variability in duration and frequency of exercise applied ranging from 10 min to 60 min sessions, and performed between 3 to 7 times per week^[11–13,19,21,59]. Duration and frequency for an individual patient depends on his baseline clinical and functional status. According to general principles of exercise prescription^[54], chronic heart failure patients with functional capacity of <3 METS (which approximates 25–40 W) seem to benefit from multiple short daily exercise

sessions of 5–10 min each^[12,19]. For those with a functional capacity of 3–5 METS (corresponding to approx. 40–80 W), one to two sessions/day of 15 min each seems to be appropriate. In patients with functional capacity of >5 METS 3–5 sessions per week for 20–30 min each are recommended.

Rate of exercise progression

Initial improvements of aerobic capacity and symptoms, in traditional programmes, occurred at 4 weeks. The maximum time required to attain peak responses in physical and cardiopulmonary variables was 16 and 26 weeks, respectively, then responses plateau^[59]. In the first weeks of training, clinically stable patients with very low exercise capacity demonstrated faster and more pronounced adaptation to exercise training than patients with higher baseline exercise tolerance^[69], but also no relationship was found^[64]. According to these observations, the rate of training progression has to be tailored individually, with respect to baseline functional capacity, clinical status, individual adaptability to a training programme, possible secondary disease, and (biological) age. Three stages of progression have been observed: an initial stage, improvement, and maintenance stage.

(a) Initial stage: intensity should be kept at a low level (e.g. 40–50% peak VO_2) until an exercise duration of 10–15 min is achieved. Exercise duration and frequency of training is increased according to symptoms and clinical status.

(b) During the improvement stage, the gradual increase of intensity (50%→60%→70% and even >0%, if tolerated, peak VO_2) is the primary aim; prolongation of a session to 15–20 min, and if tolerated up to 30 min, is a secondary goal. On the basis of repeated exercise testing, readjustments of training intensity is recommended when a patient is able to perform a given exercise intensity at a decreased rating of perceived exertion, and/or an improved exercise tolerance is assumed compared to baseline. In general, progression of exercise training should be followed in the order: duration, then frequency, then intensity.

(c) The maintenance stage in exercise programmes usually begins after the first 6 months of training^[54]. Further improvements may be minimal, as seen in the study by *Kavanagh et al.*^[59]. Continuing individual tailored training enables clinically stable patients to maintain their exercise capacity, and/or slow down or delay the muscle wasting and loss of aerobic capacity typical of progressive heart failure. The effects of a 3 week residential training programme were lost after only 3 weeks of activity restriction^[19], suggesting the need for implementing long-term exercise training into the therapy management of chronic heart failure.

Callisthenics

Callisthenics are one of the most common components of exercise training programmes in chronic heart failure. Primary goals are the improvement of musculoskeletal flexibility, movement coordination, muscle strength, and

respiratory capacity, as well as the ability to cope with activities of daily life. There is a lack of recommendations on the specifics of callisthenics. Thus, methods should be adopted from callisthenic programmes for cardiac patients with good exercise tolerance and modified for chronic heart failure patients. Exercise position and type of exercises should be chosen to avoid a significant increase of pre- and afterload, meaning that exercises in the sitting position and, while exercising, keeping arms at body level, are preferred. A moderate to slow speed, combined with normal rhythmic breathing is also recommended. The number of separate exercises per session and repetitions per set has to be determined by the patient's individual disease status, functional capacity, and response to exercise.

Resistance exercise training

Up to now, there has been a reluctance to apply resistance exercise. Primarily, this is based on potentially harmful cardiovascular responses which were found primarily during sustained isometric hand grip exercise lasting >3 min^[70-72]. In these studies, marked increments in systemic vascular resistance and a decrease in left ventricular ejection fraction and stroke work index, among others, was found, indicating an acute overloading of the left ventricle. In contrast, during rhythmic single leg press exercise, performed by two sets of 10 repetitions each at load, 70% of 1-repetition-maximum, heart rate and rate-pressure-product were lower, and systolic blood pressure, ejection fraction, diastolic and systolic volume were no greater than those found during cycling exercise at intensity 70% of peak VO_2 ^[73].

Additionally, during rhythmic double leg press exercise, at loads of 60% and 80% maximum voluntary contraction using interval modes with 60 s work phases of 12 repetitions each and 120 s rest phases, chronic heart failure patients demonstrated increased left ventricular stroke work index and decreased systemic vascular resistance, suggesting enhanced left ventricular function^[74].

This might be due to a rhythmic sequence of submaximal isometric muscle contractions, which help to maintain venous return, reduce systemic vascular resistance, maintain muscle blood flow, and meet muscle metabolic need.

The amount of cardiovascular stress expected during resistance exercise also depends on the muscle group/mass involved (single leg/arm vs double leg/arm). Double arm exercise with a given load resulted in greater cardiovascular stress than single arm exercise. Thus, in chronic heart failure patients at an advanced stage of heart failure and/or a very low exercise tolerance, resistance exercise training can be applied in a segmental fashion, meaning contractions of small muscle groups or muscle mass^[75,76]. In summary, rhythmic strength exercise appears to be a promising training method for chronic heart failure patients when small muscle groups are involved, using short bouts of work phases, small numbers of repetitions, and a work/recovery ratio of 1:2, for example, is applied. However, experience with this

approach is limited and more experience in larger patient populations is needed before a general recommendation can be given.

Respiratory training

Respiratory muscle strength and endurance can be increased with resistive inspiratory muscle training. Inspiratory training using e.g. the THRESHOLD inspiratory muscle trainer, at a minimum of 20–30 min \cdot day⁻¹ for 3–5 days weekly has been used in patients with chronic heart failure or chronic obstructive pulmonary disease^[45,77,78]. The intensity should be 25–35% of maximum inspiratory pressure (PI max) measured at functional residual capacity. If this cannot be achieved for 15 min, intensity can be reduced. For outcome assessment, ratings of dyspnoea, maximum inspiratory and expiratory pressure, and exercise performance should be measured before and after the training period. Exercises to strengthen abdominal muscles are also recommended. For example, in the supine position while exhaling, the head and shoulders are raised, and the abdomen pulled in as close as possible to the spinal column^[45,54]. Similar exercises can be carried out in the sitting position.

Yoga exercises have been demonstrated to improve coordination of the respiratory muscles and of the diaphragm, and to slow down the respiratory rate^[79]. In chronic heart failure patients, inspiration and expiration at controlled frequency (e.g. 15, 10, or 6 breaths \cdot min⁻¹) mobilize diaphragm, abdominal muscles, and lower and upper chest muscles in sequence. This may help to improve awareness of breathing, decrease resting respiratory rate, and increase tidal volume, and, as a consequence help to diminish atelectasis.

Safety

In most exercise training studies, patients with left ventricular dysfunction, with and without signs of heart failure, were lumped together, and thus not properly categorized. Additionally, patients involved were highly selected, using exclusion criteria associated with high risk, such as documented myocardial ischaemia and malignant rhythm disorders. On the basis of the currently available clinical research supervised, in-hospital training programmes are recommended, especially during the initial phases, in order to verify individual responses and tolerability, clinical stability and promptly identify signs or symptoms indicating the need to modify or terminate the exercise programme (Tables 3 and 4). The supervision should include: pulmonary and cardiac auscultation; check for body weight, peripheral oedema; monitoring of heart rate, blood pressure, and rhythm (before training); monitoring of heart rate, blood pressure, rhythm and symptoms (during training); pulmonary and cardiac auscultation (after training). On the other hand, according to the individual clinical response, a combined supervised/non-supervised exercise training programme may be possible for certain patients who exhibit long-term stability and excellent

Table 3 Relative criteria necessary for the initiation of an aerobic exercise training programme

- Compensated heart failure for at least 3 weeks
- Ability to speak without dyspnoea (with a respiratory rate of <30 breaths \cdot min⁻¹)
- Resting HR of <110 beats \cdot min⁻¹
- Less than moderate fatigue
- Cardiac index of ≥ 2.1 \cdot min⁻¹ \cdot m⁻² (for invasively monitored patients)
- Central venous pressure of <12 mmHg (for invasively monitored patients)

Table 4 Relative criteria indicating the need to modify or terminate the training programme

- Marked dyspnoea or fatigue (≥ 14 on Borg scale)
- Respiratory rate of >40 breaths \cdot 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)
- Poor pulse pressure (<10 mmHg difference between systolic and diastolic BP)
- Decrease in BP (of >10 mmHg) during progressive exercise
- Increased supraventricular or ventricular ectopy during exercise
- Diaphoresis, pallor or confusion

compliance. In addition to supervised sessions, intermittent sessions of unsupervised exercise training can begin when the safety of training within the prescribed range of exercise has been demonstrated^[53,80]. The combination of some supervised and some unsupervised exercise training is most likely to favour a more strict adherence to the exercise prescriptions.

Conclusion and future directions

Exercise training, especially as part of a comprehensive cardiac rehabilitation programme, can increase exercise capacity in compensated stable chronic heart failure patients. This is associated with improvements in peripheral vascular, muscular and metabolic function. Indices of sympatho-vagal balance may be improved and this suggests a potential protective role with respect to progression of left ventricular dysfunction and symptoms of heart failure. All of these effects would be beneficial in the broader range of heart failure patients. Although the role of physical training on morbidity and mortality has not been fully elucidated, there are no subsets of patients other than those with contraindications mentioned above, which should not be offered these benefits. There is evidence that patients with at least moderate, and possibly severe but stable heart failure can benefit from exercise rehabilitation, provided it is tailored to the clinical status and functional capacity of the patient and certain safety measures are taken into account. The growing literature on exercise training in heart failure, however, while supplying several fundamental answers, raises a number of important questions. In particular, the possible interaction between pharmacological therapies, especially beta-blockade,

and exercise training and the training effects in selected populations (elderly, females, with angina) should be more thoroughly evaluated. Questions about optimal training modality and intensity also remain unanswered. As yet, the most appropriate forms of exercise therapy are not known. The application and role of physical activity in more advanced heart failure patients remain to be specifically addressed. In addition, whether the training effects could be maintained over the long term, and whether exercise training is practicable in multiple medical settings outside of the enthusiastic and well motivated specialist clinics should be definitely established. Finally, sufficiently powered trials are needed to assess morbidity, mortality, and the cost-effectiveness end-points relative to exercise training in patients with heart failure. To answer some of the previously mentioned questions, the Working Group on Heart Failure together with the working Group on Cardiac Rehabilitation and Exercise Physiology of the European Society of Cardiology encourage participation in future trials aimed to evaluate whether long-term physical training improves the functional status, morbidity and mortality of stable chronic heart failure patients.

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