Benefits of exercise training in chronic heart failure


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Summary Exercise training performed in cardiac rehabilitation centres is an adjuvant therapy in chronic heart failure patients with left ventricular dysfunction; it decreases the deleterious consequences of chronic heart failure. Exercise training attenuates neurohormonal stimulation, the production of proinflammatory cytokines and natriuretic peptide overexpression. Trained patients showed a significant decrease in the peripheral organ injuries encountered in chronic heart failure, with a reduction in vascular resistance and improvements in endothelial dysfunction and the oxidative capacity of peripheral muscles, without a deleterious effect on left ventricular remodelling. Ultimately, exercise training leads to a notable improvement in ventilatory capacity. These beneficial effects are accompanied by improvements in symptoms at rest, exercise capacity and quality of life. Several training programmes are in current use: exercise training sessions always include endurance exercise performed either at a constant load intensity or with interval training, combining periods of exercise performed at high intensity with periods performed at low intensity. Most of the time, training programmes also include resistance training sessions, which improves large muscle strength. Exercise training programmes seem to have a favourable effect on prognosis, even if the results of Heart Failure: a Controlled Trial Investigating Outcomes of Exercise Training (HF-ACTION) remain controversial, emphasizing the difficulty in monitoring observance and the importance of compliance with a long-term exercise training programme. Patients who do not improve their exercise capacity significantly after an exercise training programme have a poorer prognosis.

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Résumé  Le réentraînement physique pratiqué en centre de réadaptation cardiovasculaire fait partie intégrante du traitement de l’insuffisance cardiaque par dysfonction ventriculaire gauche. Il permet en effet de lutter efficacement contre les conséquences délétères de l’insuffisance cardiaque. Le réentraînement physique réduit la stimulation neurohormonale et la production de cytokines pro-inflammatoires et de peptides natriurétiques. Il corrige les anomalies périphériques vasculaires et musculaires en réduisant les résistances périphériques, en améliorant la dysfonction endothéliale et en restaurant les capacités oxydatives, sans altérer le remodelage ventriculaire gauche. Enfin, il améliore les capacités ventilatoires. Ces effets bénéfiques s’accompagnent d’une amélioration fonctionnelle au repos, de la tolérance à l’effort et de la qualité de vie. Plusieurs protocoles de réentraînement sont actuellement utilisés. Les séances comportent obligatoirement des exercices d’endurance, réalisés soit à intensité constante, soit en créneaux en alternant des phases à hautes et à faibles intensités. Elles sont le plus souvent couplées à des séances de gymnastique segmentaire qui renforcent les principaux groupes musculaires. Le réentraînement physique semble avoir un effet bénéfique sur le pronostic des patients même si celui-ci reste plus difficile à authentifier comme le démontrent les résultats de l’étude HF-ACTION, et ce, probablement en raison de la difficulté de contrôler l’observance et la compliance de l’exercice physique à long terme. Les patients qui n’améliorent pas leur tolérance à l’effort après réalisation d’un programme de réadaptation ont un pronostic plus péjoratif à court et moyen terme.

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Abbreviations

HF  heart failure
LVEF  left ventricular ejection fraction
NYHA  New York Heart Association
$V_{CO_2}$  CO$_2$ production
VE  pulmonary ventilation
$V_O_2$  oxygen consumption

Cardiac limitation to exercise in heart failure patients: physiopathological benefits of exercise training

Irrespective of aetiology, chronic HF begins with a pump function injury. However, symptoms and disease progression also involve alteration of peripheral organs and neurohormonal activation. Both HF and peripheral organ injuries account for patients’ exercise intolerance. Exercise training facilitates the correction, in part, of most of the peripheral abnormalities and tends to decrease the neurohormonal stimulation in chronic HF patients without having a deleterious effect on left ventricular remodelling (Fig. 1).

Heart rate response

In general, heart rate is increased at rest and decreased at peak exercise in chronic HF patients, leading to a reduction of the chronotropic reserve. This is accounted for mainly by the desensitization of $beta$-adrenergic receptors. Furthermore, the decrease in heart rate immediately after exercise (heart rate recovery), which is considered to be an indicator of parasympathetic nervous system tone, is reduced in chronic HF patients. The benefit of exercise training on heart rate response to exercise has been well documented. Exercise training leads to a decrease in heart rate at rest and to increases in both the chronotropic reserve and heart rate recovery [9,10], particularly via a beneficial effect on the sympathetic nervous system, even in patients receiving a beta-blocker [10,11].

Stroke volume response

Stroke volume is decreased at rest in HF. During exercise, it increases less than in healthy subjects or, more often, decreases. The benefits of exercise training on left
Exercise training in chronic HF remains controversial. Randomized studies showed either no effect [1,4,12,13] or a small beneficial effect on left ventricular remodelling, with a slight but significant increase in LVEF [11,14]. Wisloff et al. suggested that an interval training programme is associated with a greater improvement in LVEF than a continuous or steady-state programme [15]. Furthermore, improvement of left ventricular function could be related to promotion of the coronary collateral development, with a significant reduction in residual ischaemia in ischaemic cardiomyopathy [12], a reduction in peripheral arterial resistance observed after exercise training [4] and an improvement in left ventricular diastolic filling.

Neurohormonal activation

Neurohumoral activation, including activation of the sympathetic nervous and renin-angiotensin systems, is the rule in advanced HF, and patients with the greatest sympathetic activation have the poorest prognosis. More recently, proinflammatory cytokines and brain natriuretic peptide have been identified as cardiac markers in various HF settings.

Exercise training reduces norepinephrine levels at rest and during exercise [4], decreases central sympathetic nerve outflow measured directly by microneurography, enhances vagal control with a shift away from sympathetic activity, and improves heart rate variability with a return to a more balanced sympathetic-vagal tone [16]. Moreover, it allows a significant reduction in the local expression of the cytokine tumour necrosis factor-alpha, interleukin-1-beta, interleukin-6 and inducible nitric oxide synthase in the skeletal muscle of chronic HF patients [17], and has a beneficial effect on peripheral inflammatory markers reflecting monocyte/macrophage-endothelial cell interaction [18]. These local anti-inflammatory effects of exercise may attenuate the catabolic wasting process associated with the progression of chronic HF.

Finally, numerous studies have shown a reduction in natriuretic peptide overexpression with a significant reduction in brain natriuretic peptide and N-terminal prohormone brain natriuretic peptide levels after endurance exercise training [11,19]. The effect of combined endurance/resistance training on natriuretic peptide levels remains more controversial [13,20].

Peripheral limitation to the exercise response in chronic heart failure patients

The recognition of the importance of muscular and vascular abnormalities, at the level of the peripheral vessels and the muscles, has been a major breakthrough in the understanding of the physiology of the limitation of the exercise response in chronic HF. Simple experiments suggest that it is usually the periphery and not the heart that limits the exercise capacity. For example, when cardiac output during exercise is increased by dobutamine infusion in patients with severe HF, maximal VO₂ increases minimally because arteriovenous oxygen difference does not increase in parallel [21].

Vascular abnormalities

Vasodilatory response during exercise is altered in patients with chronic HF. The main abnormalities in arterial response during exercise seem to be a profound alteration in flow-dependent vasodilatation, mainly at the level of the resistive arteries. Abnormalities in endothelium- and flow-dependent vasodilatation are a key phenomenon in the blunted vasodilatory response to exercise in chronic HF patients. A significant improvement in endothelium-
dependent relaxation has been observed in trained patients [22]. Exercise training enables the improvement of both basal endothelial nitric oxide formation and agonist-mediated endothelium-dependent vasodilation of the skeletal muscle vasculature in chronic HF patients [22,23].

Peripheral muscle abnormalities

Peripheral muscle abnormalities are another principal reason for the reduced exercise capacity of chronic HF patients. Muscle atrophy and structural modifications occur frequently, linked especially to malnutrition, deconditioning and the toxic action of cytokines.

Fatty infiltration is sometimes observed macroscopically, meaning that functional muscle mass is reduced to a greater extent than it appears. Microscopically, muscle fibre distribution is modified: type IIb fibres (glycolytic fibres) are increased at the expense of type I oxidative fibres; mitochondrial density is decreased and, in parallel, there is a selective reduction in enzymes involved in the Krebs cycle (i.e., the oxidative pathway), such as citrate synthase or succinyl CoA dehydrogenase.

Exercise training leads to a significant increase in muscle aerobic capacities, with a dramatic increase in myofibril cross-sectional area, mitochondrial density, volume density of cytochrome c oxidase-positive mitochondria and capillary density [24,25]. These modifications appear even with low-intensity endurance exercise training (i.e., 40% of peak \( \dot{V}O_2 \)) and allow a significant improvement in both peak \( \dot{V}O_2 \) and ventilatory threshold [24,26,27]. However, a higher intensity level of exercise training (70% of peak \( \dot{V}O_2 \)) seems to be necessary to obtain a significant reshift to type I fibre, with a significant increase in type I fibre and a significant decrease in type II fibre.

Abnormalities of the ventilatory response to exercise

Exertional dyspnoea is far from being related only to pulmonary wedge pressure; it has been related to \( CO_2 \) output, pulmonary dead space, pulmonary blood flow and activation of peripheral muscle chemoreceptors. The slope relating ventilation to oxygen uptake (\( VE/VCO_2 \) slope) is increased in chronic HF patients and is a potent prognostic factor.

Both endurance exercise training and local respiratory muscle training improve ventilatory capacities.

Mancini et al. [28] studied the effect of respiratory muscle training in a set of 14 patients with chronic HF. Maximal sustainable ventilatory capacity increased from 48.6 to 76.9 L/min with an increase in inspiratory and expiratory respiratory muscle strength. Chiappa et al. reported recently an improvement in blood flow to resting and exercising limbs after inspiratory muscle training [29]. Likewise, Van Laethem et al. showed a significant improvement in the oxygen uptake efficiency slope, a new variable assessing the linear measure of the ventilatory response to exercise, after 6 months of combined strength and aerobic exercise training in 35 patients with stable chronic HF [30]. In these two studies, the improvement in ventilatory capacity after exercise training was closely correlated with the improvement in peak \( \dot{V}O_2 \).

The benefit of exercise training on the \( VE/VCO_2 \) slope remains more controversial. Davey et al. [31] described a significant decrease in the \( VE/VCO_2 \) slope after exercise training, indicating an improvement in ventilatory efficiency, while Passino et al. [11] did not.

Exercise training modalities

In published studies, exercise training regimen levels have varied from low to moderate; interval training at various intensities (up to 95% of peak heart rate) has also been shown to be beneficial [15]. Moreover, it has never been shown clearly that training intensity influences directly the magnitude of the increase in exercise tolerance in chronic HF patients.

In a survey conducted by the Groupe exercice réadaptation et sport of the French Society of Cardiology in 2005, 40 cardiac rehabilitation centres described their exercise training modalities in chronic HF patients [32]. A cardiopulmonary exercise test was performed in 86% of the cases and three different training sessions of varying intensities were then prescribed: a training heart rate equal to the heart rate observed at the ventilatory threshold (50% of the rehabilitation centres); a training session driven by the patients’ feelings (grade 12—14 on the Borg scale; 32% of the rehabilitation centres); or a training session at the load observed at the ventilatory threshold (prescription in watts: 18% of the rehabilitation centres).

This heterogeneity is in keeping with guidelines [8]. Although gas exchange measurement seems to offer an objective assessment of functional capacity and should be used when feasible to derive the exercise prescription [8], it should be emphasized that the exercise training modality prescribed varies widely between studies.

The intensity range used most frequently has been 70—80% of peak \( \dot{V}O_2 \) (60% to initiate the programme in very debilitated patients), but it is not clear whether this percentage of peak \( \dot{V}O_2 \) is prescribed by training heart rate or training power.

In Heart Failure: A Controlled Trial Investigating Outcomes of Exercise Training (HF-ACTION), exercise training modalities were described very clearly (Table 1) [33]. The exercise training sessions were purely moderate, continuous training (60—70% of heart rate reserve). Interval and resistance training were forbidden, so that the treatment could be translated easily into clinical practice in daily life. Nevertheless, in specialized cardiac rehabilitation centres, these two exercise training session types can be associated with classical continuous training.

Interval training

Aerobic interval training comprises short bouts of high muscular loading alternating with rest periods (Fig. 2). It appears to be as safe as steady-state exercise training with respect to left ventricular function, and a recent study [15] has shown its efficiency. Twenty-seven stable, elderly, post-infarction chronic HF patients were randomized to a moderate, continuous training group (70% of exercise peak heart rate), an aerobic interval training group (95% of peak heart rate) or a control group for 12 weeks. The increase in peak \( \dot{V}O_2 \)
Exercise training in chronic HF

<table>
<thead>
<tr>
<th>Training phase</th>
<th>Location</th>
<th>Week</th>
<th>Weekly sessions</th>
<th>Aerobic duration (min)</th>
<th>Intensity (% of heart rate recovery)</th>
<th>Mode of exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial supervised</td>
<td>Clinic</td>
<td>1–2</td>
<td>3</td>
<td>15–30</td>
<td>60</td>
<td>Walk or cycle</td>
</tr>
<tr>
<td>Supervised</td>
<td>Clinic</td>
<td>3–6</td>
<td>3</td>
<td>30–35</td>
<td>70</td>
<td>—</td>
</tr>
<tr>
<td>Maintenance</td>
<td>Clinic/home</td>
<td>7–12</td>
<td>3/2</td>
<td>30–35</td>
<td>70</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Home</td>
<td>13 end</td>
<td>5</td>
<td>40</td>
<td>60–70</td>
<td>—</td>
</tr>
</tbody>
</table>

Sixty percent of heart rate recovery = resting heart rate + 0.6 (peak heart rate – resting heart rate).

was greater with interval training (46%) than with continuous training (14%; \( p < 0.001 \)). Moreover, interval training was associated with reverse left ventricular remodelling (a significant decrease in diastolic and systolic left ventricular volume and an increase of 35% in LVEF \( p < 0.01 \)), which was not observed in the continuous training or control groups.

**Resistance training**

The rationale for resistance training as an adjunct to an exercise-based rehabilitation programme stems from several lines of evidence: lack of strength is an important problem in amyotrophic chronic HF patients because equilibrium and most leisure and occupational tasks require a minimum degree of strength in legs and arms. Moderate-to-high intensity resistance training performed 2–3 days per week (for 3–6 months) improves muscular strength and endurance in men and women of all ages by 25–100%. Moreover, because the pressure response to resistance exercise is proportionate to the percentage of maximal voluntary contraction as well as the muscle mass involved, increased muscle strength results in an attenuated heart rate and blood pressure response to any given load, because the load now represents a lower percentage of the maximal voluntary contraction [34].

Programmes include a set of eight to ten different exercises that train the major muscle groups, performed 2–3 days per week. A repetition range of ten to 15 at a low relative resistance is recommended for cardiac patients. Small free weights (0.5–2 kg), elastic bands or a weight machine can be used. The weight can be adjusted in accordance with the patient’s feelings (13–16 on the Borg scale) and must always be less than 50% of the maximum weight that could be used to complete one repetition.

**Clinical benefits of exercise training**

**Quality of life**

For the most part, dedicated studies found a significant improvement in quality of life after completion of an exercise training programme [1,35,36]. This improvement was observed in young and elderly patients [37], with various exercise training programme settings, including endurance exercise only, endurance and resistance [13], aerobic interval training [15,38], local muscle training [36] and even an exercise training programme performed at low intensity [1].

More recently, the HF-ACTION study confirmed a significant improvement in quality of life in a randomized study that included a large population of chronic HF patients. Patient’s health status, assessed by the Kansas City Cardiomyopathy Questionnaire, improved on average by five points prematurely at 3 months in the exercise arm vs three points in the control arm; this significant difference was sustained over time [39].

**Exercise capacity**

Improvement in exercise capacity is related to the improvement in neurohormonal activation, peripheral abnormalities and ventilatory function, and has been assessed by a
or combined aerobic and strength training, with an increase involved either continuous or intermittent aerobic exercise or combined aerobic and strength training, with an increase in peak $\dot{V}_{O_2}$ from 12 to 31% was observed. A recent meta-analysis suggested that the greatest mean increase in peak $\dot{V}_{O_2}$ was identified in the 40 studies that involved either continuous or intermittent aerobic exercise and functional capacity in the existing trials are therefore incremental to standard therapy, and some studies have documented ongoing efficacy despite beta-blocker therapy [50].

The incorporation of beta-blockers into standard therapy for HF is a relatively recent development and many studies preceded this era. Improvements in cardiac function and functional capacity in the existing trials are therefore incremental to standard therapy, and some studies have documented ongoing efficacy despite beta-blocker therapy [52].

Finally, a local muscle training programme also leads to a significant improvement in exercise capacity. There were no reports of deaths related directly to exercise during more than 60,000 patient-hours of exercise training [48].

Survival benefits

As with every effective treatment, the benefits of exercise training on survival are not related to exercise training prescription but are related directly to exercise training realization. This suggests that exercise training will only have a positive effect on prognosis when performed in a cardiac rehabilitation centre, because the main problem with home training sessions is the lack of compliance.

In a randomized study that included 110 chronic HF patients, Belardinelli et al. [1] reported for the first time a favourable outcome after 3.3 years of follow-up after 12 months of exercise training. The rates of all cardiac events, cardiac deaths and hospitalization for HF were reduced significantly in trained patients vs a control group (log rank test = 14.29, 6.24 and 5.78, respectively). The authors emphasized the possible role of improvement in both exercise capacity and myocardial perfusion through vessel neoformation and the reduction of endothelial dysfunction. In this study, exercise training was performed in a cardiac rehabilitation centre. By contrast, McKelvie et al. [53], in a randomized, controlled, single-blind trial comparing 3 months of supervised training followed by 9 months of home-based training with usual care in 181 chronic HF patients, did not find any significant differences regarding rates of mortality, the composite clinical outcome of total deaths or hospitalization for HF, or the composite clinical outcome of total deaths or worsening HF, over a similar follow-up period. However, the compliance with exercise training was probably worse in this study (30% of the patients attended 55% of the sessions and the 9 months of home-based training were not under control) than in the study by Belardinelli et al., in which all sessions were held at the hospital gymnasium under the supervision of a cardiologist. Indeed, it was shown recently that home-based exercise training programmes were associated with poor compliance and may not be appropriate for community based chronic HF patients [54].

The Exercise Training Meta-Analysis of Trials in Patients with Chronic Heart Failure (ExTraMATCH), which included nine randomized studies with 801 chronic HF patients, also suggested that exercise training has a beneficial effect on outcome. Overall, there were 88 deaths in the exercise arm (median time to event, 618 days) and 105 deaths in the control arm (median time to event, 421 days). The mortality rate and the death or readmission to hospital rate were significantly lower in the exercise group (heart rate 0.65 and 0.72, respectively; $p = 0.05$). These results imply that 17 patients need to be treated to prevent one death in 2 years [55].

By contrast, in a meta-analysis that included 30 randomized parallel-group trials, Smart et Marwick [48] reported that there was a non-significant mortality reduction trend during the training and follow-up periods (mean duration, 5.9 months), with 26 deaths among the 622 subjects in the exercise group and 41 deaths among the 575 sedentary control subjects (odds-ratio 0.71; $p = 0.06$). Finally, HF-ACTION (a large, international, multicentre, National Heart, Lung and Blood Institute-sponsored, randomized trial) was conducted to determine whether exercise training reduces all-cause mortality or all-cause hospitalization of symptomatic chronic HF patients with left ventricular systolic dysfunction [56]. Patients in the exercise training arm ($n = 1159$) attended 36 supervised, facility-based, exercise training sessions. After completing 18 sessions, patients started home-based exercise. Then there was a transition to just home-based exercise after completion of all 36 sessions. Patients in the control group ($n = 1172$) received the usual treatment and self-management education and were contacted regularly by telephone. After an average follow-up period of 2.5 years, no significant difference was found (in the intention-to-treat population) in the primary endpoint (all-cause mortality and all-cause hospitalization rate, hazard ratio 0.93; $p = 0.13$) or in the secondary endpoints (cardiovascular mortality and cardiovascular hospitalization rate, hazard ratio 0.92; $p = 0.14$; cardiovascular mortality and HF hospitalization rate, hazard ratio 0.87; $p = 0.06$). After adjustment for prespecified prognostic factors (cardiopulmonary exercise testing exercise duration, LVEF, Beck Depression Inventory and history of atrial fibrillation or flutter), both the all-cause mortality and all-cause hospitalization rate (hazard ratio 0.89; $p = 0.03$) and the cardiovascular mortality and HF hospitalization rate (hazard ratio 0.85; $p = 0.03$) were reduced significantly compared with the control group, although several major concerns may limit the results.
Firstly, in keeping with the American College of Cardiology/American Heart Association guidelines, the HF-ACTION education manual recommends that control chronic HF patients perform 30 min (or as long as can be tolerated) of moderate intensity activity on most days of the week, so the control group was not without any physical activity. Secondly, compliance with exercise training was relatively low: close to 60 min/week on average of exercise time in the trained group instead of the 120 min/week expected. This may explain the relative low gain in peak $V_O^2$ in the training group (0.6 mL/kg/min vs 17% on average observed in other controlled studies [48]). Finally, patients received optimal medical treatment including beta-blockers (95%), angiotensin-converting enzyme inhibitors or angiotensin receptor blockers (92%), implantable cardioverter defibrillators (40%) or biventricular pacing (18%).

Exercise capacity improvement after exercise training programme completion: a strong prognostic factor

In the study by Belardinelli et al. [1], the results suggested that an improvement in prognosis was only possible in patients with a significant improvement in peak $V_O^2$ after completion of the training programme, but the number of trained patients ($n=50$) was not sufficiently high for conclusions to be drawn. We conducted a bicentric study to assess the prognostic value of improvement (or lack of improvement) in exercise capacity, after an exercise training programme in patients with chronic HF. One hundred and fifty-five patients were enrolled prospectively. Patients underwent 3–5 training sessions per week for 4–8 weeks in a cardiac rehabilitation centre. Improvement in exercise capacity was assessed by the change in peak $V_O^2$ and percentage of maximal predicted peak $V_O^2$ between admission and after completion of the training programme. After a follow-up period of $16 \pm 6$ months, 27 cardiac events occurred: 12 deaths, five cardiac transplantations and ten hospitalizations for acute HF. Both univariate and multivariable analyses showed that brain natriuretic peptide concentration at admission and change in exercise capacity were the strongest prognostic factors, independent of well-known variables such as LVEF, functional state at rest and the presence of atrial fibrillation. The risk ratio of cardiac events for non-responsive patients (defined as patients in whom the percentage of theoretical peak $V_O^2$ was increased less than the median value of 6% after the training programme) vs responsive patients was 8.2 ($p = 0.0006$) (Fig. 3) [10]. Chronic HF patients who do not show a significant improvement after the exercise training programme are therefore also

![Figure 3. Kaplan-Meier event-free survival curves of chronic heart failure patients according to their exercise response to training. Those who improved (delta percentage of predicted peak $V_O^2$ greater than 6%) had a better outcome than those who did not improve (delta percentage of predicted peak $V_O^2$ less than 6%) [10]. CV, cardiovascular; $P_{V_O^2}$, peak oxygen consumption; RR, risk ratio.](image-url)
at high risk of cardiac events and should be monitored carefully.

**Indication for exercise training**

Most studies have included chronic HF patients with LVEF less than 40% in NYHA functional class II or III. Guidelines recommend cardiac rehabilitation in such patients [7,8,10]. No data are available concerning chronic HF patients with preserved left ventricular function. Most severe patients, in NYHA functional class IV, are excluded currently from cardiac rehabilitation programmes, although a training programme that includes segmental muscle exercise is safe and effective in such patients [55,57,58]. On the other hand, the benefit of exercise training in asymptomatic chronic HF patients has not been evaluated. Finally, while HF with preserved left ventricular function occurs frequently, especially in elderly patients, no data are available regarding the effect of an exercise training programme in this population. Exercise training programmes are efficient both in ischaemic and non-ischaemic cardiomyopathy, although improvement in peak VO2 has been reported to be greater in patients with non-ischaemic cardiomyopathy [44].

Although elderly patients represent the largest population with chronic HF, patients who are included in cardiac rehabilitation programmes are currently younger. Exercise training is efficient and safe in elderly patients when adapted protocols are used [37]. In the HF-ACTION study, 25% of the patients (i.e., 582 patients) were aged greater than 68 years. Young and old patients underwent the same protocol (moderate continuous training at 60-70% of heart rate reserve) and no significant subgroup interaction regarding quality of life improvement for age was found [39]. In our study of 155 patients, 19% were aged greater than 65 years and the improvement in exercise capacity was not influenced by age [10]. Finally, Wisloff et al. reported recently in elderly, post-infarction patients, the superiority of aerobic interval training over moderate continuous training for improving left ventricular remodelling, aerobic capacity, endothelial function and quality of life [15]. Even if exercise capacity in elderly patients was more often limited by a non-cardiovascular cause, the benefit was similar to that in younger chronic HF patients.

**Conclusion**

Exercise training allows the correction, in part, of most of the peripheral abnormalities encountered in chronic HF and tends to decrease neurohormonal stimulation without a deleterious effect on left ventricular remodelling. Exercise training programmes usually include endurance and resistance exercise, leading to a significant improvement in quality of life and exercise capacity and a trend towards improvement in the prognosis of chronic HF patients. Exercise training appears, therefore, to be a safe, non-costly strategy that can be integrated into the overall drug and non-drug management of chronic HF patients with low ejection fraction: its effects on symptoms and quality of life are at least equal to those produced by other therapies. Its effects on outcome are probably largely dependent on the long-term adherence to an exercise-based way of life after a hospital-based rehabilitation programme and are probably not negligible, even on top of optimal therapy.

**Conflicts of interest**

None.

**References**


