Cardiac Rehabilitation for Patients with Chronic Heart Failure.

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Introduction
Heart failure is a major problem in public health. Several advances in treatment and management of heart failure have been regularly introduced including new pharmacological therapies and non-pharmacological strategies (surgery, ventricular assistance and resynchronization). However, even if the prognosis of heart failure is improved, the individual exercise tolerance and the social repercussions of this disease remained important. Cardiac rehabilitation is an additive but essential part of the therapeutic strategy for stable chronic heart failure patients. So, it is now demonstrated that exercise training improves exercise tolerance, corrects some peripheral abnormalities and may improve the prognosis.

Heart failure abnormalities
The prognosis of heart failure is related to the left ventricular function, to the level of exercise capacity and to the arrhythmic risk. However, there is no correlation between the systolic left ventricular function and the effort tolerance. Figure 1:

![Relationship between peak VO₂ and systolic ventricular function in CHF patients](image)

**Figura 1:** Absence of correlation between left ventricular ejection fraction and exercise tolerance

Symptoms are usually represented by dyspnea and/or fatigue during exercise, consequences of complex mechanisms involving heart, lungs and muscles. The exercise intolerance force patients to reduce their activities, which, in turn, worsens muscular deconditioning and ventilatory abnormalities.
So, a vicious circle is initiated, which may lead to a worsening of the prognosis.

Heart failure due to left ventricular systolic dysfunction is associated with cardiac and peripheral abnormalities. Overlap mechanisms are involved in the physiopathology of dyspnea and fatigue. Reduced cardiac output is the primary factor of the disease, but it can not by itself explain the exercise intolerance. Effort intolerance seems to be related also to peripheral changes in blood flow, endothelial and muscles function.

The mechanisms of the dyspnea include: abnormalities in pulmonary blood flow distribution at rest and at maximal exercise, excessive production of lactates, increase in the pulmonary deadspace, fatigue of the respiratory muscles [6], bronchiolar hyper reactivity and finally, hyperventilation [1,2].

Fatigue and muscular strength's decrease are explained by the early anaerobic metabolism during exercise in heart failure. This is due in part to low muscular blood flow, but also to muscular deconditioning and to abnormal oxygen utilization. It is associated with structural modifications: decrease in oxidative fibers (type I and IIa), increase in glycolytic fibers (type IIb), reduction in the capillary density and decrease of mitochondrial activity [3,4].

Neuro-hormonal modifications include excessive sympathetic stimulation, activation of renin-angiotensin system and increasing levels of endothelin. Finally, impairment of endothelium-dependent vasodilation contributes to reduce exercise tolerance in heart failure.

**Effects of physical training**

The beneficial effects of cardiac rehabilitation are widely accepted for coronary artery diseases: reduction of 25 % of the cardiovascular mortality at 3 years after myocardial infarction, improvement of the exercise tolerance and quality of life [5]. Moreover, patients who have left ventricular dysfunction post MI (ejection fraction < 45 %) improve their exercise tolerance (+ 23 % in peak VO₂) after training without significant deleterious ventricular remodeling [6].

**Exercise tolerance**

One of the most important beneficial effect of training was the improvement in exercise tolerance. Exercise training improves not only exercise duration, but also peak VO₂ and anaerobic threshold (table 1).
In summary, exercise training allows an improvement from 20 to 30 % of the duration of exercise and peak VO2. These effects seem to be related to the compliance of patients to the rehabilitation program.

More importantly, sub-maximal performances are improved, which may explain a symptomatic improvement assessed by NYHA class and quality of life scores reduction [7,8].

**Muscular effects**

Exercise training improves muscular strength, particularly for most deconditioned patients. Improvements are related with the reduction of vascular resistances and reduction of lactate production for sub-maximal exercise. Training leads to an increase of the mitochondrial density (correlated with the improvement of peak VO2), of oxidative fibers and of the capillary density [9]. However, the part of muscular effects in the improvement of exercise capacity for a given patient is not easily demonstrable because of obvious interactions with the other systems. For instance, training reduces the stimulation of muscular ergoreceptors and consequently attenuates hyperventilation and vasoconstriction [10].

**Ventilatory effects**

The favorable effects of exercise training on ventilatory abnormalities were reported by some studies: reduction of hyperventilation, improvement in oxygen extraction and in ventilatory efficiency [7-11]. Furthermore, selective respiratory muscle training in a small study had demonstrated an improvement of inspiratory and expiratory muscle strength and an increase in peak VO2 [12].

**Effects on autonomous nervous system**

The enhanced parasympathetic tonus with a decrease in sympathetic and renin-angiotensin systems activations are widely accepted effects of exercise training. In heart failure patients, results were debatable. The majority of the studies found a decrease levels of plasma catecholamines and an improvement in heart rate variability [7,8-13]. This decrease in sympathetic tonus may participate to a lower arrhythmic risk, but this is not yet demonstrated.

**Effects on the endothelial function**

In heart failure, exercise training improves flow-dependent relaxation and decreases peripheral vascular resistances. Hornig et al showed that training restores endothelium-dependent relaxation imputed to the NO endothelium release [14]. Hambrecht et al, confirmed more recently these results; they found a correlation between the changes in endothelium-dependent vasodilation and the improvement in exercise capacity [15].

<table>
<thead>
<tr>
<th>Author</th>
<th>Year of publication</th>
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<th>EF (%)</th>
<th>Intensity (%)</th>
<th>Duration (weeks)</th>
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<td>17 #</td>
<td>20</td>
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Table 1: Randomized studies and results on exercise tolerance after cardiac rehabilitation in CHF patients.

#: cross-over study. *: % of peak VO2. °: % maximal heart rate.

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Effects on cardiac function
In chronic heart failure patients, resting heart rate was decreased after training. However, it has not been proved that left ventricular ejection fraction significantly increases due to physical training. Some authors suggest that an improvement of the coronary collaterals might improve systolic function in long-term follow-up. Although, Dubach et al. showed an increase in maximal cardiac output and a reduction in the maximal arterio-venous oxygen difference after a two-weeks, high intensity training program [16]. In the same way, some authors suggested that exercise training could have beneficial effects on diastolic function by a diastolic wall-stress reduction [17]. The improvement in peak VO$_2$ in dilated cardiomyopathy may be predicted by a peak ventricular filling rate assessed by Doppler echocardiography [18].

Survival effects
By now, as regards outcome implications, only one randomized study demonstrates a reduction of the morbidity and mortality at 3.3 years of follow up [19]. In this study, including 99 patients, relative reduction of the cardiac mortality and rehospitalizations for heart failure were 37% and 29% respectively. More recently EXERT study, not designed for this purpose, did not confirm these results [20]. In our knowledge two large randomized trials (HF-ACTION and EFICACIA) are currently organized in order to investigate the prognostic impact of exercise training in heart failure patients.

Selection of the patients
Cardiac rehabilitation is clearly recommended for patients with left ventricular dysfunction in post-infarction. According to the European Society of Cardiology, American Heart Association and Agency for Health Care Policy and Research guidelines [21,22], a global component cardiac rehabilitation program is indicated for heart failure patients with ischemic or dilated cardiomyopathy, in NYHA class II and III.

Exercise training is also recommended before and after cardiac transplantation, for patients implanted with a left ventricular assist device, and for post-operative patients with left ventricular dysfunction.

Contraindications for exercise in heart failure patients are listed in a Figure 3.

- Acute heart failure
- Unstable angina
- Complex ventricular arrhythmias
- Pulmonary arterial pressure > 60 mmHg at rest
- Intra cardiac thrombus
- Moderate to severe pericardial effusion
- Myocarditis in evolutive phase
- Symptomatic obstructive cardiomyopathy or aortic stenosis
- Venous thrombosis
- Orthopedic or neurologic problems limiting exercise possibilities

Figura 3: Contraindications to exercise training in heart failure.

Nevertheless, inclusion of a given patient in a rehabilitation program needs to be extremely careful and needs a precise evaluation before exercise training.

Age and sex do not seem to have any incidence on benefits of exercise training, but there are only few studies concerning these subgroups of patients. In women, the compliance to the program (98%) and the improvement of the muscular enzyme activity are comparable to those obtained in programs including a majority of males [23].

The left ventricular ejection fraction is neither a selection criteria nor a predictive factor of results of training [8].
Severity of heart failure have to be considered before the inclusion in exercise training program. Only NYHA class II and III are recognized indications in published guidelines. However, Meyer [24], and Stevenson [25] studies have demonstrated that the most severe patients such as those awaiting cardiac transplantation can benefit from exercise training. Belardinelli [18] found that patients with relaxation abnormalities assessed by Doppler echocardiography have a higher benefit than other patients. The same authors showed that presence of hibernating myocardium and changes of LVEF during echo-Dobutamine test were the most predictive variables of benefits of exercise training [26].

In our experience, baseline low exercise tolerance and compliance to the program are independent predictive factors of the improvement after rehabilitation [27]. The most deconditioned heart failure patients, who have the weakest baseline performances, should be targeted for inclusion in supervised programs of rehabilitation.

**The practical modalities**
Cardiac rehabilitation includes not only exercise training, even if exercise represents an essential and original part in the management of heart failure patients.

**Education, risk factors control and psychological help** remain essential points for these chronic disease patients. It is moreover the purpose of the studies of the American, Australian and Swedish nurses who show a reduction of the number of hospitalizations due to a rigorous follow-up of these patients [28,29,30]. DIAL study, recently presented at scientific sessions of AHA, demonstrates that supervision of patients by phone contact reduces significantly the number of re hospitalizations for heart failure (-29%). Eckman [30], shows however that only 13 to 17% of the old persons are candidates for a supervising program at home, because of a more difficult follow-up in this group of patients with a lesser participation.

**Physical training** aims to improve the exercise capacities, to decrease the oxygen myocardial consumption at rest and at effort and to limit sympathetic activation and arrhythmias.

The practice of physical activities requires personalized programs. Evaluation before beginning a program, including cardio respiratory stress test evaluation, left ventricular function and arrhythmic risk assessment, is required to estimate prognosis and for guidance of exercise training prescriptions. Currently exercise training intensity is adjusted at anaerobic threshold or from 60 to 80% of the peak VO2 level obtained during the first test, and reevaluated during the follow up [22,31]. Generally, training is practiced on cyclo ergometer or treadmill, completed by sessions of outdoor walking and/or swimming. The described benefits appear gradually from 8 to 40 weeks, with training sequence of 3 to 5 sessions a week. The dynamic resistance training for individual muscle groups produces improvements in muscular strength and endurance [32]. This training mode, with a minimal cardiac workload, seems more adapted for the most advance heart failure patients in whom loss in skeletal muscle volume and low strength was frequent.

Advanced heart failure patients need beginning exercise training in a supervising center; at the opposite ambulatory training may be preferred after a period of training without events and for low risk patients.

**Risks**
In Changes study [33], a randomized study including 80 patients in class II and III, the improvement in exercise duration, in anaerobic threshold and of peak VO2 were 21, 12 and 10% respectively after a 12 weeks exercise training program. No significantly differences in cardiac events or side effects was noted.

More recently, a review of 64 studies concludes that exercise training is safe in CHF patients without deaths in 59 000 patients-hours and 1 adverse event every 3 300 hours [34]. The most common events include arrhythmias and worsening symptoms.

**Unresolved questions**

The benefits of this original approach in heart failure are proved, but physiological aspects, practical modalities and prognostic value remain still to clarify.

These points include the role of training on ventricular remodeling, on diastolic function, on coronary and peripheral vessels and on the neuro-hormonal regulation.

Moreover, exercise prescription protocol must be adapted for every group of patients, but this is not yet codified.

Importantly, cost and reimbursement are different among countries and may interfere with heart failure
management.

It remains to define effects and interest of physical training in association with the other non pharmacological approaches proposed in heart failure (ventricular assist devices, cardiac resynchronization).

Maintenance phase possibilities and long-term effects of exercise training needs to be studied. It remains to clarify by controlled and unquestionable studies the effects of exercise training on cardiac morbidity and mortality.

Conclusion
Exercise training, integrated into a global comprehensive rehabilitation program, including pharmacological adaptations, control of risk factors, psychological counseling and education, increases exercise tolerance in patients with stable chronic heart failure.

References


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