

# Cardiac Rehabilitation in Heart Failure: A Brief Review and Recommendations

Ileana L. Piña

Published online: 18 March 2010  
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**Abstract** The number of heart failure patients continues to increase for men and women. Our current medical therapy includes a variety of agents that can reduce morbidity and mortality. However, many patients remain limited, in part, due to deconditioning. Adding to the functional loss are the multiple hospitalizations and even recommendations from providers to maintain bed rest and avoid physical activity. The multiple studies involving conditioning and exercise for patients with heart failure have mostly shown improvements, not only in function but also in quality of life and other physiologic parameters that should be of benefit to these patients. The HF ACTION (Heart Failure: A Controlled Trial Investigating Outcomes of Exercise Training) trial, which randomized 2,231 patients to a formal prescribed exercise or control, demonstrated the safety of aerobic training in this population. The benefits on the hard end points of mortality and hospitalizations were modest but significant when adjusted for prognostic factors. The guidelines currently recommend that activity be recommended in conjunction with medical therapy. A program can be adjusted to the patient's need but should include intensity, frequency, and duration, as well as a guide to progression.

**Keywords** Heart failure · Exercise ·  $\text{VO}_2$

## Introduction

The prevalence of heart failure (HF) continues to increase [1••]. Hospitalizations have increased to over 600,000 per year. HF constitutes the number 1 discharge diagnosis in the United States for Medicare beneficiaries, accounting for more than 700,000 admissions each year [2]. HF carries significant mortality and morbidity as well as a high cost to the health care system. Parallel to this increase in prevalence over the past 10 years, there has been a wealth of new information about pharmacologic therapy that decreases hospitalizations and improves survival in these patients. However, in spite of excellent medical therapy, some patients remain chronically fatigued and are unable to perform a myriad of activities due to symptoms and may remain as the most symptomatic of patients with chronic disease. The inability to perform even activities of daily living can lead to poor quality of life and ultimately to feelings of sadness and depression. Because of the vicious cycle of frequent hospital admissions, these patients go through periods of decompensation during which symptoms limit activity and deconditioning worsens. It may be difficult to distinguish the symptoms of fatigue and dyspnea due to deconditioning from the HF syndrome itself (i.e., fatigue with activity).

Because the patients in this population may be hospitalized often, deconditioning may gradually appear and become insidious, especially if the patient is advised to get “bed rest.” A common misconception still exists within professional and nonprofessional circles that exercise is detrimental to patients with HF—that the heart may be further injured by activity. In a European survey, 60% of the population believed that HF patients should get bed rest and avoid any physical activity [3]. Physical activity in the form of exercise training has been shown to be safe, in a

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I. L. Piña (✉)  
Department of Medicine and Epidemiology/Biostatistics,  
Case Western Reserve University, Cleveland VA Medical Center,  
2627 Fairmount Boulevard,  
Cleveland, OH 44106, USA  
e-mail: ilppina@aol.com  
e-mail: ileana.pina@case.edu

supervised setting or in the home, for the majority of stable, well-medicated HF patients [4••]. This review describes the effects of deconditioning, and presents a basic understanding of the physiology of exercise in normal individuals and in HF patients and the rationale for exercise training followed by suggested approaches. The recently completed and published HF ACTION trial, sponsored by the US National Heart, Lung, and Blood Institute, will be referred to often because it is the largest, prospective, randomized, controlled trial of exercise training in 2,231 patients with class II to IV HF due to systolic dysfunction. This review does not cover exercise training in preserved systolic function HF because the HF ACTION trial did not include that population and the data are less plentiful. Nonetheless, it is recognized that by signs, symptoms, and deconditioning, preserved systolic function HF may be identical to HF with systolic dysfunction [5]. Therefore, the Heart Failure Society of America (HFSA) guidelines have recommended a similar approach to these patients as to those with systolic dysfunction [6].

### Definition of Functional Capacity

Functional capacity, or the ability to perform activity, can be estimated or actually measured. The most common description of functional capacity is the New York Heart Association (NYHA) classification of I to IV, which is based on an individual's perception of activity tolerance and the interpretation of that perception by an interviewer. A more accurate measurement of functional capacity is the measurement of activity by exercise testing. Exercise intolerance or loss of functional capacity is defined as the reduced ability to perform activities, which involve dynamic movement of large skeletal muscles due to symptoms of dyspnea or fatigue. Exercise intolerance can be described by considering the activities that produce symptoms, or often by the level of function compared with a normal individual of similar age, weight, and gender. A commonly used expression that describes exercise intolerance in patients with HF is their peak oxygen uptake ( $VO_2$ ) in relation to their normal predicted. Peak  $VO_2$  is one of the best measures of functional capacity and is discussed below.

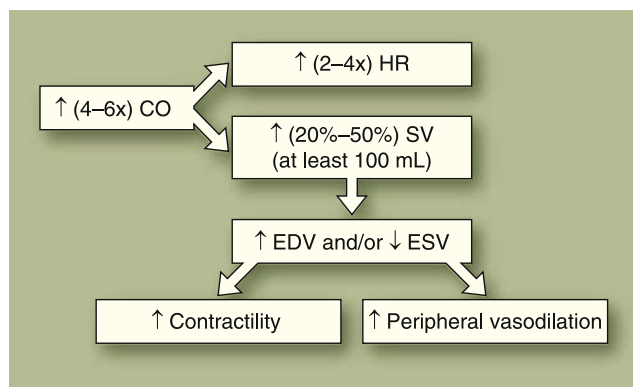
### Exercise Physiology in Normal Individuals

The capacity for performing activity depends upon the ability of the heart to increase cardiac output to improve blood flow to the exercising muscles, and the ability of these muscles to use oxygen from the peripheral blood. Maximal oxygen uptake ( $VO_{2max}$ ) is the product of cardiac

output and peripheral arterial-venous difference. Cardiac output is the product of heart rate and stroke volume. Thus,  $VO_{2max} = \text{cardiac output} \times \text{arteriovenous oxygen difference}$ . The increase in cardiac output during maximal upright exercise is typically four- to sixfold in normal individuals. This is accomplished by a two- to fourfold increase in heart rate and a 20% to 50% augmentation of stroke volume. Figures 1 and 2 demonstrate these parameters in normal individuals and patients with HF [7]. Age, gender, and genetics also influence normal functional capacity. Age alone will change the components of this physiologic response. Maximum heart rate declines with increasing age. However, stroke volume should not change appreciably, although left ventricular end-systolic volume may not decrease as much, thereby needing preload more [8].

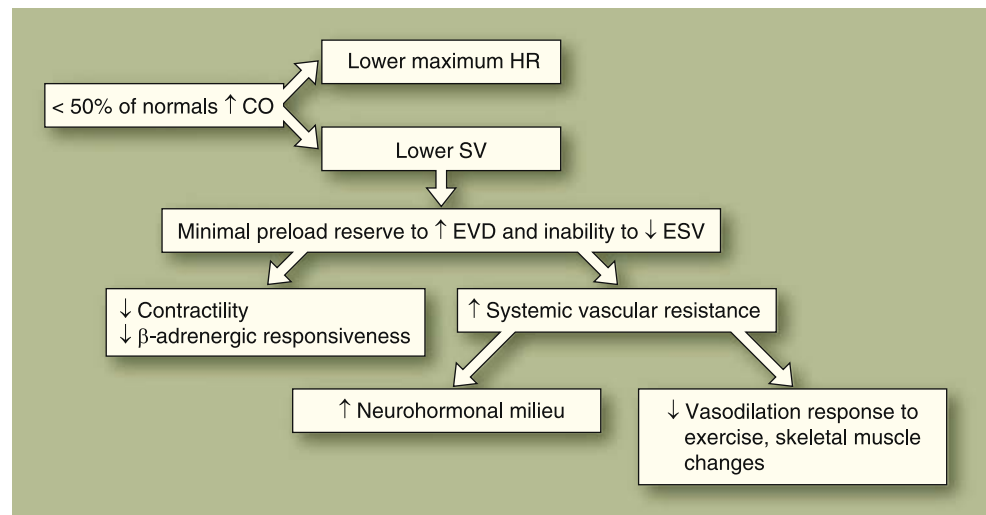
### Deleterious Effects of Bed Rest

Because of periods of bed rest, the HF patient who is frequently hospitalized will be more deconditioned and debilitated than the NYHA class II patient who remains out of the hospital. Bed rest is deleterious to normal subjects and it is certainly deleterious to patients with chronic diseases such as HF. Even normal individuals who are subjected to bed rest experience a loss of functional capacity (ability to do activity) after only 10 days. Functional capacity can decrease by as much as 8.4% in men and 6.8% in women, and total exercise tolerance decreases by 8.1% in men and 7.3% in women [9]. Peak heart rate increases in men and women. In addition, there is a direct correlation between the drop in function and the number of days of bed rest [10]. Not only is there a loss of functional capacity, but changes in muscle and bone also



**Fig. 1** Exercise physiology: normals. Maximal oxygen uptake ( $VO_{2max}$ ) =  $CO (HR \times SV) \times D(A-V)O_2$ . (A-V) $O_2$ —arteriovenous oxygen difference; CO—cardiac output; EDV—end-diastolic volume; ESV—end-systolic volume; HR—heart rate; SV—stroke volume;  $VO_2$ —peak oxygen uptake. (Adapted from Piña et al. [7])

**Fig. 2** Exercise physiology: heart failure. Maximal oxygen uptake ( $VO_{2max}$ ) = CO (HR × SV) × D(A-V)O<sub>2</sub>. (A-V) O<sub>2</sub>—arteriovenous oxygen difference; CO—cardiac output; EDV—end-diastolic volume; ESV—end-systolic volume; HR—heart rate; SV—stroke volume; VO<sub>2</sub>—peak oxygen uptake. (Adapted from Piña et al. [7])



occur, particularly in the lower extremities. Inactivity also increases maximal heart rate, with a decrease in vagal tone, increased sympathetic catecholamine secretion, and enhanced  $\beta$ -receptor sensitivity to circulating catecholamines. Although heart rate is elevated, the functional capacity drops due to a lower stroke volume and a drop in cardiac output [11]. The patient with HF can suffer all of these consequences of bed rest, and in addition may experience the changes in muscle fibers that occur with the disease alone. Therefore, alterations in skeletal muscle structure, fiber type, and mitochondrial oxidative enzymes further augment the exercise intolerance suffered by HF patients [12, 13].

### Exercise Intolerance in Heart Failure

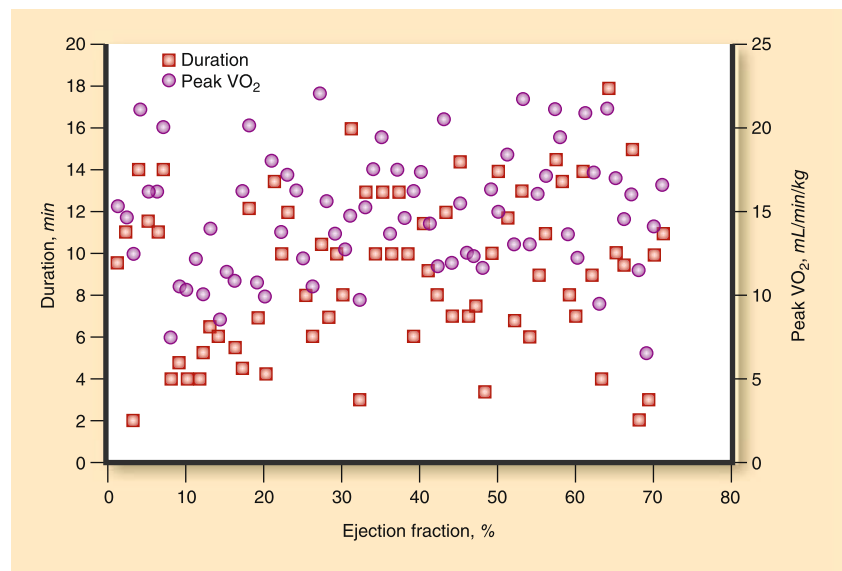
HF can be defined as the inability of the heart to meet the metabolic demands of the tissues, thereby producing symptoms of fatigue and/or dyspnea on exertion that can progress to dyspnea at rest. Intuitively, it would be expected that patients with HF, especially with low ejection fraction, would be unable to perform activity. However, there is no relationship between resting ventricular function and functional capacity whether measured by a simple exercise test or by measuring the true  $VO_2$  (Fig. 3) [14]. Therefore, restricting patient activity by only evaluating resting ventricular function is not supported by studies. Dyspnea on exertion may be exacerbated by volume retention. The inability to perform exercise without discomfort may be one of the first symptoms experienced by HF patients and is often the primary reason for the patients to seek medical care. In some cases, family members or close friends may notice the patient's symptoms of dyspnea. Therefore, exercise intolerance is closely linked to the diagnosis of HF.

Patients with incipient HF may slowly and unknowingly self-limit their activity to avoid symptoms of dyspnea or fatigue. If asked about the time that it takes to perform activities of daily living, patients may admit that it takes them longer than in previous months or years. Symptom assessment should include questions such as how long does it take to perform their morning routine and are there recreational or work-related activities that they have abandoned or curtailed. This type of questioning will often detect that symptoms existed far before the actual first episode of recognized HF. Patients will also often describe the need to totally avoid activity the day following a more unusually active day. Fatigue can be related to low cardiac output and may include difficulty in concentrating.

In contrast to the normal exercise physiology noted in Fig. 1, the cardiac output of a patient with left ventricular dysfunction may be limited in several ways (Fig. 2). The heart rate reached will be lower commensurate with a lower workload. In addition, the current use of  $\beta$  blockers as indispensable therapy for HF will also limit the heart rate achieved. Stroke volume will be limited by the inherent loss of contractility (systolic dysfunction) and inability to augment preload. Peripheral changes are also present with alterations in the skeletal muscle, coupled to peripheral vasoconstriction [13, 15]. Skeletal muscle changes suggest that alterations may contribute to abnormal oxygen extraction or substrate delivery/utilization and may further limit exercise tolerance in HF.

Thus, the exercise intolerance of patients with HF seems to be multifactorial but, nevertheless, the functional capacity is impaired and can be measured by cardiopulmonary exercise testing to determine the peak  $VO_2$ . As in normal individuals, the peak  $VO_2$  is lower in women with HF than in men [16]. The peak  $VO_2$  seems to “track” the NYHA class. In the HF ACTION trials, the peak  $VO_2$  of women versus men was significantly lower (13.35 [CI,

**Fig. 3** Relationship between ventricular function and exercise capacity.  $\text{VO}_2$ —peak oxygen uptake



10.80, 16.30] vs. 14.90 [CI, 11.90, 18.20];  $P < 0.001$ ). The difference between men and women was greatest for the NYHA class II patients. Of note, the peak  $\text{VO}_2$  has been used as a strong predictor of outcomes, including mortality, and often becomes a deciding factor in the listing of HF patients for cardiac transplantation [15]. Analysis of the extensive database of HF ACTION continues and will produce further information about the predictive nature of this parameter.

It is recommended that before prescribing an exercise training or cardiac rehabilitation program for patients with HF, that at a minimum a standard exercise test be performed. The gold standard would be measurement of peak  $\text{VO}_2$ , which would serve the purpose of prognostication and to set an exercise prescription. An exercise test will also support the patient's ability to do activity and help allay the fears and concerns of family members.

### Exercise Training in Heart Failure

In the 1960s, exercise was contraindicated for patients with HF. In the 1970s and 1980s, a series of studies demonstrated that ventricular function at rest could not predict exercise capacity and that patients with HF could perform physical activity [17–20]. Other studies pointed out physiologic beneficial effects of exercise training. The potential benefits of exercise training in HF as reported were multiple and included the following: an improvement in function, a heightened sense of quality of life, improved ventilatory parameters, improved muscle function, improvement in endothelial function, and a potential drop in neurohormones [15, 21]. Improvements in peak  $\text{VO}_2$  have varied from 10% to 30%, as reported in small trials [15, 22,

23]. The improvements in women may be less than those of men, but this needs to be confirmed in further analysis of the HF ACTION trial [4•, 16]. Indices of submaximal exercise, such as the 6-minute walk or the ventilatory threshold, can also improve [4•, 23]. However, improvement in peak  $\text{VO}_2$  may not translate to improved survival. The effects of mortality and hospitalizations were somewhat controversial. A small study of 99 patients randomized to exercise or no exercise in retrospect showed a reduction in mortality and hospitalizations, whereas another larger study failed to show any benefit [24, 25]. A meta-analysis of 11 studies comprising 729 patients reported that

**Table 1** Components of an exercise prescription for heart failure patients

- Intensity
  - Peak HR (from an exercise test)
  - HR reserve
    - =  $x\%(\text{peak HR} - \text{rest HR}) + \text{rest HR}$
  - Borg scale RPE
  - HR at ventilatory threshold
- Duration
  - Per session
    - Consider warm up and cool down
  - Number of weeks
- Frequency
  - Number of times/week
  - Daily
  - Most days of the week
- Progression
  - Consider low level early
  - Increase as tolerated

HR heart rate, RPE rate of perceived exertion, based on the Borg scale

**Table 2** Borg scale: 15 point

■ 6–20% effort
■ 7–30% effort (very, very light [rest])
■ 8–40% effort
■ 9–50% effort (very light [gentle walking])
■ 10–55% effort
■ 11–60% effort (fairly light)
■ 12–65% effort
■ 13–70% effort (somewhat hard [steady pace])
■ 14–75% effort
■ 15–80% effort (hard)
■ 16–85% effort
■ 17–90% effort (very hard)
■ 18–95% effort
■ 19–100% effort (very, very hard)
■ 20—Exhaustion

the results showed a beneficial effect of exercise training on mortality [26].

Using all these studies as background, the HF ACTION trial was launched to address the question of outcomes in a large group of ambulatory, well-medicated patients with HF randomized to a formal exercise program or to control. In the HF ACTION trial, exercise training was shown to have a modest but not statistically significant effect on all-cause mortality or all-cause hospitalization [4••]. When adjusted for various factors, the results became statistically significant, although still modest. However, the trial suffered from less than optimal adherence to the exercise program by the group of patients randomized to exercise training. In spite of the suboptimal adherence to training, the study did show a statistically significant improvement in health status as demonstrated by the Kansas City Cardiomyopathy Questionnaire in all its domains. At 3 and 12 months, more patients had attained a five or greater point improvement [27]. The HF ACTION trial did confirm the safety of training in a large group of patients; therefore, patients and

their caregivers can be reassured of the extremely low event rates.

Both the American Heart Association/American College of Cardiology and the HFSA guidelines for HF recommend that exercise training in the form of rehabilitation “should be considered as part of the routine therapeutic approach in the majority of patients with HF due to left ventricular systolic dysfunction.” However, if the patients are congested or dyspneic at rest, these symptoms need to be controlled prior to the patient embarking on the exercise program. It is also strongly recommended that, as in the HF ACTION trial, the training be initiated in a supervised setting. The question of whether these patients should be monitored has often arisen. In HF ACTION, electrocardiogram (ECG) monitoring was not a requirement and with the low rate of events and the high safety profile, there is no basis for demanding ECG monitoring. In the current economic environment with lack of coverage by Centers for Medicare & Medicaid Services (CMS) or insurers for cardiac rehabilitation programs if the diagnosis is HF, a home program may become a necessity. However, the recommendations for exercise prescription still apply.

### The Exercise Prescription

As in any exercise program, the exercise prescription should include intensity (dose), frequency, duration, and the often forgotten, progression. The recommendation should also include the type of exercise prescribed (i.e., aerobic, resistive, or both). The HF ACTION trial did not test resistive training, only aerobic. However, there are no data to date that show deleterious effects of resistive training for muscle conditioning. The prescription should also give a guide to the type of aerobic training recommended (e.g., treadmill vs. bicycle ergometry or some other form of equipment, such as gliders and rowers). For patients exercising only at home or continuing at home after a supervised program, walking is a widely accepted

**Table 3** Exercise training program: HF ACTION

Training phase	Location	Week <sup>a</sup>	Sessions/ week	Aerobic, <i>min</i>	Intensity (HR reserve, %)	Training mode
Initial supervised	Rehabilitation center	1–2	3	15–30	60	Walk/cycle
Supervised	Rehabilitation center	3–6	3	30–35	70	Walk/cycle
Supervised and home	Rehabilitation center and home	7–12	3 and 2	30–35	70	Walk/cycle
Maintenance	Home	13–end	5	40	60–70	Walk/cycle

<sup>a</sup> Week intervals shown are goals and may vary for individual participants

HF ACTION heart failure: a controlled trial investigating outcomes of exercise training, HR heart rate

(Data from O'Connor et al. [4••])

and achievable, inexpensive form of activity. Resistive training can take the form of elastic bands, or small free weights. Warm up and cool down are equally essential, as in any exercise regimen. Warm up and cool down can be achieved by stretching, or performing the same aerobic activity at a lower intensity.

Generally, 20–30 min of the desired intensity are recommended and added to the warm-up and cool-down period. Programs generally last 4–6 weeks, or can be counted by number of sessions. Thirty-six sessions have been commonly used. The intensity can be defined by a heart rate percentage, or a heart rate reserve percentage (Table 1), or by a rate of perceived exertion (Borg scale; expressed as RPEs). A heart rate of 60% to 70% of maximum is commonly used. The heart rate reserve approach takes into consideration the heart rate achieved during exercise testing, which in the current therapeutic  $\beta$ -blocker milieu may be helpful. A 70% of the heart rate reserve is also reasonable. Table 2 illustrates the ratings of the Borg scale. Using RPE may help patients achieve the recommended intensity without concern about measuring their pulse rate. A Borg rating of 13–14 may coincide with the level of the ventilatory threshold, which occurs at 60% to 80% of the maximum  $\text{VO}_2$ . The most frequently recommended duration of a formal program is three times per week. Table 3 depicts the strategy used in the HF ACTION trial. If patients are very debilitated and deconditioned, a slow start of lower intensity broken by periods of rest is reasonable. Progression implies that as the patient improves, function, intensity, and duration can be increased gradually to reach the assigned target in the exercise prescription.

Patients should also be encouraged to incorporate activity into their daily lives and even into activities of daily living. Hobbies should also be encouraged. Should a patient with HF wish to return to work, an evaluation of the metabolic requirements of the occupation is certainly warranted. An exercise test may assist in decision making on return to the work environment. Employment that involved mainly low-level activity may be completely safe. It may be suggested that a part-time schedule be initiated and can be increased if the patient does well. Active engagement in job or hobby activity can do nothing but benefit the patient by improving quality of life and self-assurance. There are many tables and descriptions of the metabolic ( $\text{VO}_2$ ) requirements of occupations [28, 29].

### Conclusions: Additional Benefits

At this point, only the exercise portion of cardiac rehabilitation has been discussed above. However, formal cardiac rehabilitation programs offer much more than only

exercise. The patient education in these programs includes dietary guidelines, risk factor modification, secondary prevention, education about the disease processes and when to call for symptoms, smoking cessation counseling, and personal counseling, if needed. The recently published Performance Measures for Cardiac Rehabilitation did not include HF as an indication because the totality of the HF ACTION trial was still pending [30]. Future endeavors include approaching CMS to consider coverage and funding for this important component of total HF care.

Exercise training should be an integral part of the care program for patients with HF. The HF ACTION trial has provided the assurances of safety that were so badly needed. The benefits of training on survival and hospitalizations are modest but the enhancement of health status is very significant. In addition, there are many physiologic benefits to increased activity as noted above. The HF ACTION trial did not directly address the physiologic changes that have been previously reported in the literature and, therefore, the reports stand as valid. CMS and insurance coverage in addition to education for providers is critical in extending this type of treatment to the general HF population.

**Disclosure** Dr. Ileana L. Piña has been on the speakers' bureau for Novartis, and is a consultant for the US Food and Drug Administration for devices and Sanofi-Aventis. She has also received a grant from the US National Institutes of Health and the Office of Women's Health, HHS.

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