29. Heart failure

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Summary

Chronic heart failure has a multifactorial origin and reduced physical performance capacity is common in those who suffer from it. Changes in the peripheral blood circulation are likely just as important as left ventricular function in the reduction of physical performance capacity. Research from the past decade has shown that exercise clearly improves function in patients with heart failure, which is why exercise should be offered to these patients today as a part of treatment.

The table below gives a description of training methods with the levels of intensity, frequency and duration that have been scientifically studied. In day-to-day clinical work, we strive for a combination of aerobic central circulatory training and peripheral muscle training, 2–3 times per week, together with home training.
Suitable activities are aerobic exercise, for example, walking, exercising on land or in water, and cycling combined with peripheral muscle training with, for example, resistance bands (Thera-Band®) or weights and exercise pulleys. The latter form of training is especially suited in cases of pronounced impairment of physical performance capacity. During the exercise session, light to moderate exertion and light to moderate breathlessness are suitable levels of load.

Patients with stable heart failure with predominant systolic dysfunction, New York Heart Association (NYHA) class (I)-II-III, are best suited for exercise. Before training begins, there should be contact with a knowledgeable physiotherapist so that the patient can undergo aerobic and muscular physical function testing. Based on the outcomes of this, a suitable training programme is then drawn up.

**Definition**

The syndrome of heart failure has been defined in a number of ways, but no one definition is complete. A common definition is: Heart failure is a condition where the heart’s pumping capacity is unable to meet the metabolic needs of the peripheral tissues (1).
Cause

In the Western world, ischaemic heart disease and hypertension (elevated blood pressure) are predominant causes of heart failure (2). Other etiological factors include diabetes, valvular disease, endocrine disorders, toxic effect, arrhythmia and systemic disease. There is also an idiopathic type, that is, where the cause is not known (1).

Prevalence/Incidence

The prevalence of heart failure varies between 0.3–2 per cent according to different sources and rises with increasing age, to approximately 10 per cent at 80 years of age (3, 4). The incidence of heart failure is also age-dependent and varies between 1–5 new cases per 1000 individuals per year (2). A Swedish registry study indicates that the incidence of heart failure is decreasing, in part due to improved medical care (5).

Pathophysiology

Loss of myocardial cells, changes in heart muscle tissue and contractility reduce the resting stroke volume. This is compensated for by activation of the neuroendocrine system through increased sympathetic system activity and activation of the renin-angiotensin system. This leads to a pathological remodeling in both the heart and the peripheral vascular system, progressive weakening of arterial and cardiopulmonary baroreflexes and a down-regulation of beta receptors in the heart. The parasympathetic heart activity is also reduced (1). The cause of the reduced exercise capacity in heart failure is complex. Haemodynamic variables at rest correlate poorly to maximal exercise capacity (6), while the correlation during exercise is better (7). It has been proposed that diastolic dysfunction may be more important for exercise capacity than systolic dysfunction (8). However, it is likely factors in the skeletal muscle that have the most significance for the reduction in exercise capacity (9). Peripheral blood flow is decreased due to reduced cardiac output and increased peripheral vascular resistance, which is mediated via changes in endothelial function and directly via vasoactive agents (10–12). Insulin resistance is reduced (13) in chronic heart failure and peripheral skeletal muscle mass is reduced (14, 15). Skeletal muscle strength and endurance are also reduced (16, 17). A change in muscle fibre composition and reduced oxidative capacity in skeletal muscle have also been reported. It moreover appears that inflammatory cytokines play an important role in loss of skeletal muscle mass and fatigue in chronic heart failure (18, 19). Skeletal muscle receptors that are sensitive to exercise, that is, ergoreceptors, are overactive. One of the tasks of these receptors is to control vascular constriction, which can be affected by exercise (20, 21). Whether these changes are contingent on physical inactivity or are a consequence of the heart failure syndrome itself is a topic of discussion (9, 22).
Symptoms and diagnosis
The diagnostic criteria recommended by the Task Force on Heart Failure of the European Society of Cardiology (23) are as follows:

1. Symptoms consistent with heart failure at rest or during exercise.
2. Objective evidence of cardiac dysfunction at rest.
3. Improvement in symptoms in response to treatment directed towards heart failure.

Points 1 and 2 should be met, while point 3 substantiates the diagnosis in cases where there is doubt. Fatigue, shortness of breath and reduced physical performance capacity are cardinal symptoms of heart failure. Functional capacity is often classified with NYHA criteria (24). Typical signs of heart failure are crackling sounds from the lungs, tachycardia, ventricular gallop, peripheral oedema, enlarged liver and stasis of the neck veins.

Heart failure can be caused by systolic and/or diastolic dysfunction. In systolic dysfunction, the ventricular pumping capacity is impaired. Systolic function is usually measured as an ejection fraction (i.e. the stroke volume in relation to the total diastolic volume). Diastolic dysfunction is characterised by typical symptoms or signs of heart failure despite a normal ejection fraction, with concurrent signs of decrease in myocardial compliance. The diagnosis for systolic and/or diastolic dysfunction is made with the aid of a patient history, clinical examination, echocardiogram, x-ray and laboratory tests. Stress tests have limited diagnostic value in heart failure, but a normal stress test in an untreated patient rules out the diagnosis (25).

Prognosis
Despite the fact that the prognosis has improved in recent years (5), it is still poor, with a 5-year survival rate of approximately 50 per cent. The more symptoms a patient has, the worse the prognosis (2).

Treatment principles
The drugs most commonly used in the treatment of heart failure are diuretics, ACE inhibitors and beta blockers. Diuretics are used primarily to relieve symptoms. In addition to relieving symptoms, ACE inhibitors also have a prognostic effect. Beta blockers have a prognostic and symptomatic effect in moderate and severe heart failure. In patients with severe heart failure, spironolactone has a favourable prognostic effect. Angiotensin receptor blockers have been shown to reduce mortality in moderate and severe heart failure.
Effects of physical activity

Acute effects of aerobic exercise

The maximal oxygen uptake (VO₂ max) during arm exercise, cycling and walking in people with heart failure can be 30–40 per cent lower than in healthy individuals (26, 27). Maximal cardiac output is also decreased due to a decrease in maximal stroke volume and insufficient capacity to increase heart rate normally (28, 29). Systolic blood pressure does not increase normally (30), while the sympathetic activity increases more during exercise in patients with heart failure than in healthy individuals (31).

The respiratory minute volume (VE) is increased in patients with heart failure at every given load compared with healthy individuals. At maximal exertion, however, VE is approximately 50 per cent of predicted value, suggesting that this does not limit the exercise capacity (32). The ratio between minute ventilation and carbon dioxide production (VE/VCO₂) increases, and it is most often shortness of breath that is the limiting factor for physical activity in these patients.

The peripheral resistance during exercise decreases in patients with heart failure but is still higher than in healthy individuals. Normally, the blood flow in the skeletal muscle increases during aerobic exercise. In heart failure, the blood flow to the working muscles is reduced when large muscle mass is involved, which is the case with aerobic exercise (11). This results in anaerobic metabolism early on in the physical activity (33). Normal blood flow can be maintained, however, if the exercise is performed using a sufficiently small muscle mass (34).

Acute effects of muscle exercise

Isometric muscle exercise (static exercise)

In healthy individuals, the blood flow in the skeletal muscle in an isometric contraction is inhibited due to increased intramuscular pressure, which compresses the blood vessels (35). During isometric exercise, heart rate and systolic and diastolic blood pressure increase more in patients with heart failure than in healthy individuals (36). The increased afterload (the pressure that the ventricle is subjected to during the ejection phase, i.e. the expulsion resistance) that isometric exercise leads to could be harmful to patients with heart failure. However, in two Swedish studies patients with heart failure performed isometric exercise without any complications (16, 37).

Isotonic muscle exercise (dynamic exercise)

Exercise in the form of leg press at 60–80 per cent of maximum exertion does not yield a larger effect on haemodynamic variables than cycling. Neither does it yield a negative effect on left ventricular function (38, 39).
Acute effects of hydrotherapy

When the body is immersed in water, significant physiological changes occur. The physiological changes are due in part to the temperature and depth of the water. The pressure the body is subjected to is 0.74 mm Hg/1 cm H$_2$O. Immersion in thermoneutral water, approximately 35¡ C, to the upper edge of the sternum (suprasternal fossa) results in an increase in venous return that gives an increased blood volume in the heart. The increased preload results in a haemodynamic improvement with increased stroke volume and ejection fraction, while the heart rate remains unchanged or is somewhat lower. The result is an increase in cardiac output, while the mean arterial pressure is unchanged and the total peripheral resistance decreases due to vasodilatation in the peripheral tissues and abdominal organs (40).

The increased volume in the heart also stimulates cardiopulmonary receptors, which in turn signal the kidneys (via the brain) to reduce sympathetic stimulation and increase urine secretion (diuresis) and reduce the neurohormones renin, aldosteron and angiotensin II (41). This means that warm baths have a similar effect as that sought in modern drug treatment of heart failure. Even an increase in cardiac output, stroke volume and ejection fraction together with a decrease in peripheral resistance has been shown after warm baths. There is at present scientific debate regarding the benefits of hydrotherapy in heart failure.

A group of German researchers (42) suggests that certain patients with severe mitral insufficiency (poorly functioning valve between the left atrium and ventricle) should not exercise in water, as they have measured a market increase in preload in these patients. Studies conducted in Sweden have not been able to confirm this, so further research in this area is needed.

Long-term effects

Aerobic exercise, muscle exercise and hydrotherapy

The long-term effects of exercise in patients with heart failure (Table 2) are in many respects in line with those measured in healthy individuals and patients with heart disease without heart failure (43, 44). The exercise methods studied include central circulatory training, cycling in particular but also exercise classes, walking and peripheral muscle training. Hydrotherapy, tai chi and respiratory muscle training have also been studied.
Table 2. Long-term effects of aerobic central circulatory training and peripheral muscle endurance training in patients with chronic heart failure.

<table>
<thead>
<tr>
<th>Effect variable</th>
<th>Result</th>
<th>Effect variable</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2 max</td>
<td>↑</td>
<td>Sympathetic activity</td>
<td>↓</td>
</tr>
<tr>
<td>Workload</td>
<td>↑</td>
<td>Muscle blood flow</td>
<td>↑</td>
</tr>
<tr>
<td>watt</td>
<td>↑</td>
<td>Oxidative capacity</td>
<td>↑</td>
</tr>
<tr>
<td>workload</td>
<td>↑</td>
<td>Capillarisation</td>
<td>↑</td>
</tr>
<tr>
<td>exercise time</td>
<td>↑</td>
<td>Muscle fibre area</td>
<td>↑</td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
<td>Muscle strength and endurance</td>
<td>↑</td>
</tr>
<tr>
<td>resting</td>
<td>↓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>maximal</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke volume</td>
<td></td>
<td>Endothelium-independent vascular function</td>
<td>↑</td>
</tr>
<tr>
<td>resting</td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>exercise</td>
<td>↑</td>
<td>endothelium-dependent vascular function</td>
<td>↑</td>
</tr>
<tr>
<td>Cardiac output</td>
<td></td>
<td>Dyspnoea</td>
<td>↓</td>
</tr>
<tr>
<td>exercise</td>
<td>↑</td>
<td>Fatigue</td>
<td>↓</td>
</tr>
<tr>
<td>Ejection fraction*</td>
<td>↔</td>
<td>NYHA class</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>↔</td>
<td>6-minute walk test</td>
<td>↑</td>
</tr>
<tr>
<td>resting</td>
<td>↔</td>
<td></td>
<td></td>
</tr>
<tr>
<td>exercise</td>
<td>↓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory minute volume</td>
<td>↓</td>
<td>Quality of life</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↔</td>
</tr>
<tr>
<td>Inflammatory cytokines</td>
<td>↓</td>
<td>Mortality</td>
<td>↓</td>
</tr>
</tbody>
</table>

↑ = Increase, ↔ = No change, ↓ = Decrease.

* Ejection fraction = Ratio of stroke volume to total end-diastolic volume.
**Indications**

Patients with stable heart failure with predominant systolic dysfunction in NYHA class (I)-II-III. Experience from exercise training of patients in NYHA Class IV and patients with diastolic dysfunction is limited (43, 44).

**Primary prevention**

For patients with asymptomatic left ventricular dysfunction or myocardial infarction, training increases exercise capacity and cardiac output (45, 46). Whether training in asymptomatic patients with reduced left ventricular function is able to prevent the development of heart failure is not known.

**Secondary prevention**

Exercise training improves several of the pathophysiological findings found in heart failure disease. Two meta-analyses have shown that longer periods of exercise can have an impact on mortality and morbidity (44, 47). Exercise also has positive effects on underlying factors such as diabetes and hypertension (48, 49).

**Prescription**

**Different types of exercise**

Central circulatory training/aerobic exercise, peripheral/local muscle training on land and in water, and respiratory muscle training, are the most evaluated techniques (see Table 3).

*Central circulatory training*, which has been studied the most, involves the engagement of large muscle groups at the same time, for example, cycling, walking and/or exercise classes (44).
Table 3. Description of training methods investigated in different scientific studies in patients with chronic heart failure.

<table>
<thead>
<tr>
<th>Training method</th>
<th>Intensity</th>
<th>RPE*</th>
<th>Frequency</th>
<th>Duration (min./session)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aerobic central circulatory training</td>
<td>(40) 60–80% of VO$_2$ max**</td>
<td>11–15 central</td>
<td>1 time/week to several times/day</td>
<td>10–60</td>
</tr>
<tr>
<td>Peripheral muscle training</td>
<td>35–70% of 1 RM***</td>
<td>13–15 local</td>
<td>2 times/week to 1 time/day</td>
<td>15–60</td>
</tr>
<tr>
<td>Combination of aerobic central circulatory training and peripheral muscle training</td>
<td>60–80% of VO$_2$ max** 60–80% of 1 RM</td>
<td>13–15 central and local</td>
<td>3 times/week</td>
<td>45–60</td>
</tr>
<tr>
<td>Hydrotherapy</td>
<td>40–80% of HRR****</td>
<td>11–15</td>
<td>3 times/week</td>
<td>45</td>
</tr>
<tr>
<td>Respiratory muscle training</td>
<td>30% of max. inspiratory pressure</td>
<td>11–15</td>
<td>3 times/week to daily</td>
<td>30–60</td>
</tr>
</tbody>
</table>

* RPE = Rating of Perceived Exertion (Borg scale 6–20).
** VO$_2$ max = Maximal Oxygen Uptake.
*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.
**** HRR = Heart Rate Reserve

In *peripheral muscle training*, the skeletal muscle is exercised locally at a relatively high load. Techniques for peripheral/local muscle training include endurance training with different types of weights and bands, for example, Thera-Band® (HCM, Hygenic Corporation, Malaysia) (50).

*Respiratory training* comprises both inspiratory and expiratory resistance training (51, 52).

In the studies of exercise training conducted until now, there is large variation when it comes to intensity, duration, frequency and exercise model. Empirically, patients with more severe heart failure (NYHA III) are considered to need a longer rehabilitation period than patients with ischaemic heart disease without heart failure, for example, in order to develop the habit of incorporating exercise into daily life. Individually adapted exercise programs where consideration is given to functional capacity and recovery time are very important for this patient group. Considering the disease’s pathology, both central circulatory training and peripheral muscle training are probably important. Peripheral muscle training performed in sequences is probably of value to patients with more advanced heart failure, in order to regain lost muscle mass. This is an important step before commencing an exercise programme that engages the central circulation (53). It is essential for the patient’s functional capacity that not only the leg muscles but also the arm and trunk muscles are exercised. Patients with chronic heart failure should always be referred to a physiotherapist for an individually adapted training programme. An intensity corresponding to between 60–80 per cent of VO$_2$ max, that is, 11–15 on the Borg RPE scale, is effective (54, 55). Two to three sessions per week are suitable for out-patient exercise training, while training at home and in hospital can be conducted more frequently.
Functional mechanisms

Oxygen uptake (VO₂)

Oxygen uptake improves by an average of about 15 per cent (2–34%), which is explained by the impact on several interacting factors such as improved heart muscle function, reduced vasoconstriction in the arterioles of the active skeletal muscle, along with an increased oxygen extraction and metabolic function in the active skeletal muscles. The measuring methods and measures of effect in the different studies vary, however, which can make it difficult to compare results between the studies. The effect of exercise training reported in the studies enable the patients to increase their activities at home, during leisure time and at work if applicable (43, 44).

Cardiac function

Cardiac function may be improved by exercise training due to better stroke volume and reduced chronotropic response during acute exercise (44).

Peripheral blood circulation and skeletal muscle function

Exercise normalises the peripheral blood circulation, probably mediated in part by improved endothelial function (56). Increased muscle strength gained through exercise is a result of both increased blood circulation to the muscles, increased muscle volume, and improved metabolic function in the muscle cells themselves. The oxidative capacity improves due to an increase in the amount of oxidative enzymes, mitochondrial volume and the number of type I fibres. Occlusion of intramuscular vessels begins already at 15 per cent of maximal voluntary contraction. The improved muscle function means that the patient is able to perform his or her daily activities at a lower percentage of maximal voluntary contraction, which theoretically results in reduced load on the heart (50).

Respiratory function

Respiratory minute volume (VE) and the ratio between minute ventilation and carbon dioxide production (VE/VCO₂) decreases, blood flow to the respiratory muscles increases, and the metabolic situation improves after exercise training (57). These could possibly be some of the reasons for the reduced breathlessness (dyspnoea) the patient experiences after exercise.

Neuroendocrine and autonomic function, and inflammatory cytokines

Exercise reduces the neuroendocrine activation and the number of inflammatory cytokines (58). The autonomic function also improves (59).
Mortality and hospitalisation

Two meta-analyses suggest that exercise can reduce mortality and hospitalisations in chronic heart failure. As in every case, the results from a meta-analysis must be interpreted with caution (60, 61).

Quality of life and symptoms

Quality of life and symptoms improve through training (44, 62). The underlying cause of this phenomenon may be of both a physical and psychological nature.

Functional tests

A functional test should always be performed to determine the appropriate individual exercise level before an exercise period begins. The same test should in addition always be conducted at the end of an exercise period to evaluate the effect of the exercise programme and for continued prescription.

Cycle/Treadmill test

A standardised maximal or submaximal cycle/treadmill test, preferably with analysis of maximal oxygen uptake, forms the basis for an appropriate design of an exercise programme (55, 63). It is also a way of ascertaining whether the patient tolerates increased physical exertion.

6-minute walk test

A standardised 6-minute walk test (64, 65) is often used to assess exercise capacity in relation to activities of daily life (ADL). The patient is encouraged to walk as far as possible for six minutes on a measured stretch of hallway. Measurement variables are the distance walked, perceived exertion measured on the Borg scale (66), and heart rate.

Muscle function

Strength and endurance, both isometric and isotonic, can be measured with an isokinetic instrument such as KINCOM (KINetic COMmunicator) (37). Clinical endurance tests can also be performed, for example, through one-legged toe raises or shoulder flexion test with weights (67).

Rating of symptoms, quality of life and physical activity

General health-related quality of life can be measured with the SF-36 questionnaire (68), while disease-specific quality of life is often measured with the Minnesota Living with
Heart Failure Questionnaire (69). An example of a survey that measures the level of physical activity is Frändin and Grimby’s activity scale (76).

Symptom severity can be assessed with the visual analogue scale (VAS) (70) or the Borg scale (66). Level of function may be evaluated using, for example, NYHA classification criteria (24).

**Interactions with drugs**

**Beta blockers**
Maximal heart rate is lower in patients with heart failure treated with beta blockers than in patients not receiving beta blockers (71). Exercise capacity increases and the ejection fraction improves both at rest and during exertion after chronic treatment with beta blockers. No effect on the skeletal muscle has been found (72).

**ACE inhibitors**
ACE inhibitors have a moderate effect on exercise capacity. Cardiac output increases and the left ventricular filling pressure falls during exertion (73). The effect of ACE inhibitor therapy on skeletal muscle is not unambiguous. Increased muscle fibre area and changes in myosin composition in skeletal muscle fibres have been described earlier (71, 72).

**Digitalis**
Digitalis increases contractility in the myocardium and thereby the stroke volume, while heart rate decreases. Exercise capacity increases under digitalis therapy (74, 75).

**Diuretics**
An open study of severely symptomatic patients showed increased oxygen uptake during exertion after eight days of treatment with diuretics (76). Diuretics can have potentially negative effects through an increased risk of dehydration and electrolyte disturbances in warm weather.

**Contraindications**

Uncompensated heart failure, obstructive hypertrophic cardiomyopathy, significant valve disease (above all aortic stenosis), active myocarditis, a drop in blood pressure, serious arrhythmia or severe ischaemia during exertion. Other serious diseases such as ongoing infection, uncontrolled diabetes, uncontrolled hypertension or recent pulmonary embolism (54) are also contraindications.
Risks

No serious incidents have occurred during or after training of patients with heart failure (44, 62). However, the heart failure patients who have participated in exercise studies to date have been selected. The mortality is in general high in this patient group, which is why it is important that exercise initially should be implemented in a medical setting in case a serious complication should occur.
References


