# **Effects of Exercises in Controlling Coronary Artery Disease:**

# **Authors**

1. Bilal Mudasir

PhD Scholar

OPJS University Churu

2. Kawsar ul Islam

Sabarmati University

Ahmadabad Gujarat

**Abstract:**

Coronary artery disease, or angina pectoris or myocardial infarction, is one of our most common diseases. Coronary artery disease is associated with pathological changes, arteriosclerosis, the presence of one or more walls of the coronary artery. Physical inactivity is a strong risk factor for coronary heart disease, but age, gender, and male origin, as well as smoking, high blood pressure, blood lipid disorders, diabetes, and being overweight increase the risk of developing this disease. Prescribing regular exercise for at least 30 minutes a day is an excellent primary prevention against coronary heart disease, and regular exercise is a powerful treatment, aerobic exercise 3-5 times a week and resistance 2-3 times a week against well-established coronary artery disease.

It is generally possible to recommend increased physical activity as the primary preventative goal, but plan the best exercise because secondary prevention requires testing the patient’s aerobic fitness and muscle function.

Assessment begins with a stress / fitness test that includes ECG monitoring, muscle testing, and an assessment of current physical activity. Based on this test and the general condition of the patient, a risk assessment is performed, after which an appropriate exercise and exercise program is developed for the patient.

Early rehabilitation supervised by specialized physiotherapists and access to first aid kits is necessary. Most patients go through cardiac rehabilitation for 3 to 6 months, and exercise can often continue outside of hospital administration once the condition stabilizes.

Key words : Coronary vein,Heart,Physical activity and treatment

***Introduction***

Coronary heart disease is one of the most common diseases, with a population of less than 200,000 in Sweden. Two-thirds of patients are men. Women develop this disease somewhat more slowly than men. It is estimated that 10,000 new cases of angina pectoris are estimated annually (1). The number of deaths and the progression of ischemic heart disease has declined since 2004, and preliminary data also suggest that the number will continue to decline in 2005. According to the Swedish National Board of Health and Welfare, 17,971 people died of ischemic heart disease in 2004. years, which is approximately 9,800 heart attacks. (2). In recent years, treatments and interventions (bypass surgery and percutaneous coronary artery therapy [PCI]) have yielded better results, resulting in more patients experiencing acute disease progression, resulting in a steady increase in the number of patients. rehabilitation. .

The reason/Cause

Coronary artery disease is associated with pathological changes in the walls of one or more coronary arteries, called advancing arterial hardness or hardening of the arteries and is the most common cause of coronary arteries, i.e. acute myocardial infarction or unstable angina pectoris (3). ).

**Risk factors**

Age, male gender, and heredity and risk factors for cardiovascular disease, such as physical activity, smoking, high blood pressure, blood lipid disorders, overweight / obesity, and diabetes, increase the risk of developing coronary heart disease (3).

**Pathological mechanisms, symptoms and diagnosis**

Arterial stiffness (atherosclerosis) is a major cause of acute coronary artery disease. Atherosclerosis primarily attacks the inner lining of the artery wall, the intimate part, which is made up of endothelial cells. First, endothelial cells accumulate in the blood (lipids), where inflammatory cells, macrophages, ingest fat. Macrophages take in lipids until they rupture and form what are called "foam cells". Then a mass of fibers forms around the foam fibers, forming a plaque. This atherosclerotic plaque does not attack all blood vessels, but appears as spots. The area around the main branch is very sensitive (4, 5).

Rumbling

The most common symptom of acute heart disease is pain in the center of the chest and includes unstable angina pectoris and acute myocardial infarction. However, early women often have insignificant symptoms such as shortness of breath, nausea or other types of pain. Angina pectoris (narrowing of the arteries) is described as permanent if the symptoms last for at least a few weeks without obvious signs of worsening. Stress angina is angina caused by physical or mental stress that quickly disappears after cessation of performance (1), and angina pectoris (seizures) is believed to occur with narrowing (narrowing) of the arteries that lasts as long as the heart muscle is open. lack of oxygen causing symptoms. Various angina pectoris may occur during rest, and a mixed type is not uncommon (6). The course of the patient with stable angina pectoris was improved by the introduction of effective treatment options such as antichemical, antithrombotic, antihypertensive and lipid-lowering drugs, as well as coronary artery surgery and catheter intervention, percutaneous coronary intervention (PCI).

The prognosis for a patient with stable angina pectoris is now relatively good. However, it is important to pay attention to the possible instability of angina pectoris, which is characterized by rapid exacerbation, which in most cases requires urgent hospitalization (5). In most people, the onset of the acute portion of the coronary arteries is caused by rupture or rupture of the atherosclerotic plaque on the coronary arteries (4). Additional treatment by platelet activation and plasma clotting results in the formation of blood clots (thrombosis) that completely or partially block the artery. Ischemia occurs when the arteries that supply blood are partially or completely blocked, leaving little oxygen and nutrients available and removing waste products. When arteries become blocked, heart cell death gradually changes, depending on the extent and duration of ischemia (7). Structural changes in the heart after myocardial infarction are not limited to the infarct area, but extend to the "healthy myocardium", which must compensate for the loss of function of the injured area by myocardial hypertrophy, capillary growth, and collagen. healthy area. This leads to a harder heart and a poorer energy balance (8). These side effects can be reduced by treatment with beta-blockers and ACE inhibitors (5).

Assessment of diagnosis and prognosis should begin as soon as possible at the same time as starting treatment. The patient’s medical history, ECG, and biochemical markers of myocardial infarction suggest that most patients can be diagnosed and develop within the first few hours. Additional examinations, such as cardiac ultrasound, coronary radiology, and / or ECG monitoring, are sometimes required. If acute coronary artery disease is suspected, the patient should be admitted to hospital as soon as possible and treated with, for example, PCI (9).

**Principles of care**

Treatment of acute heart disease, which should be started as soon as possible after the onset of symptoms, including resuscitation and / or anti-aromatic drugs. Targeting is usually performed with PCI and / or together with stenting to prevent re-sealing (re-closure) or pharmacological treatment (thrombolysis) or a combination of both (facilitated PCI). . PCI is also used to treat unstable angina pectoris and in this case, after X-rays of the coronary arteries, to check the actual changes in the coronary arteries. In some cases, coronary artery graft (CABG) is required.

In other cases, pharmacological anti-ischemic therapy is available to stabilize the condition, followed by pharmaceutical drugs such as acetosalicylic acid (ASA), beta blockers, nitrates, and calcium channel blockers. In some cases, mainly due to reduced pumping capacity, such as heart failure, ACE inhibitors are used (5).

# Effects of physical activity

The positive effects of physical activity when you already had coronary heart disease were noted in the late 18th century (10, 11). Unfortunately, these findings are lost in memory, and it was not until the mid-1960s that fitness was used as a therapy. for coronary artery disease (12, 13). This was followed by the first fitness-based cardiac rehabilitation program and the first Swedish recommendations issued in 1980 (14).

**Immediate effect**

**Acute physiological effects of exercise in ischemic heart disease**

**Heart rate, stroke Volume and cardiac output**

The immediate response of the cardiovascular system to exercise is an increase in the number of heartbeats due to reduced activity of the pathological nervous system (slowing of the vagal). This is followed by increased activity of the sympathetic nervous system in the blood vessels of the heart and body. Shortly after a heart attack or heart surgery, a relatively fast pulse is often observed during submaximal exercise or rehabilitation after exercise. Abnormally low heart rate during submaximal exercise can be caused by drugs that block beta or increase the number of beats during exercise. The use of beta-blockers, which reduce heart rate, limits the interpretation of heart rate responses during exercise.

At the beginning of the exercise, the volume of the heart increases with increased stroke due to a better ratio of length and tension in the heart muscle. This is called the Frank Starling mechanism and involves increasing strength as muscle fibers grow. The expansion of the heart muscle fibers is caused by an increase in venous refractive flow. The increase in minute volume occurs primarily with an increase in heart rate, which means that the pulse decreases after the application of beta-blockers.

Arrhythmia

The occurrence of various arrhythmias (arrhythmias) in ischemic heart disease is not uncommon. If the arrhythmia is at rest and disappears during exercise, it is usually benign. However, if the arrhythmia increases during exercise, it is advisable to stop exercising and discuss further studies (15).

Blood pressure

Systolic blood pressure rises due to increased heart rate as a result of increased dynamic exercise. Diastolic blood pressure usually remains unchanged or slightly elevated. At the clinical level, it is important to note that diastolic pressure can be a zero sound during exercise, which can lead to false readings.

Exercise can lead to a drop in blood pressure or insufficient blood pressure. During continuous exercise, poor blood pressure rises or falls due to obstructive aortic drainage, severe left ventricular dysfunction, angina pectoris, or beta blockers. However, in some people with heart disease, blood pressure may increase during the recovery phase (above the maximum value measured in the extract).

If exercise is stopped abruptly, some people may experience a significant drop in systolic blood pressure. This decrease in blood pressure is caused by the accumulation of venous blood and a slowdown in the increase in peripheral resistance, which corresponds to a decrease in cardiac output.

Oxygen intake in the heart

Oxygen intake during training can be calculated by the so-called Double Product (product pressure level = RPP) is defined as systolic blood pressure multiplied by the heart rate divided by 100. During oxygen intake, the heart with blood, which mainly occurs in the diastolic phase. During exercise, myocardial circulation can increase the value of relaxation by up to five times. Patients with heart disease are usually unable to maintain proper blood circulation in the part of the heart affected by ischemia, so the metabolic needs of the heart cannot be met during exercise, resulting in a lack of oxygen in the heart. . pectoris.

**Skeletal muscle circulation and peripheral resistance**

Blood circulation in skeletal muscles can increase threefold during exercise, and total peripheral resistance decreases due to increased vasodilation in skeletal muscle function during exercise. In treatment with beta-blockers, blood circulation is slightly improved in the muscles, resulting in greater peripheral muscle fatigue in patients receiving beta-blockers (15).

**EFFECTS OF TERMINOLOGICAL EXPLANATION ON ISCHEMIC ISSUES**

Similar specific changes lead to regular exercise in people with coronary heart disease, such as skeletal capacity and cardiovascular muscle, as well as in healthy people. The effect usually depends on the type of exercise. In the case of more aerobically focused training, the ability to absorb oxygen (VO2) is especially improved, and strength training leads to improved muscle function in specially trained muscles. The resulting training effect allows a person to exercise in greater fitness and / or lower heart rate at all submaximal levels. Studies have shown that moderate and intense aerobic exercise in heart patients and healthy individuals for 8–12 weeks at 45-minute intervals 3–5 times a day leads to a significant increase in maximal and submaximal activity (16).

**Decreased resting heart rate (relaxing bradycardia)**

A lower resting heart rate is perhaps the most obvious effect on regular exercise. The basic mechanisms include balancing autonomous change and increasing the size of religion. This effect has been seen in healthy individuals and individuals with heart disease, with or without beta-blockers (15).

**Low blood pressure**

Blood pressure and blood pressure at a certain level of fitness are lower among highly educated people. Blood pressure in the heartbeat is sometimes peripheral resistance. When peripheral resistance decreases during exercise, this results in decreased left ventricular ejection resistance and increased secretion (i.e., the percentage of blood pumped from the heart during each heartbeat or the heart’s ability to pump) to blood volume and stroke. At all stages of submaximal administration, lower systolic pressure results in a corresponding reduction in side effects, which reduces the risk of cardiac ischemia.

During vigorous exercise, high blood pressure rises in functional muscle groups, which can lead to decreased blood flow to the muscles or even to blockage (blockade), followed by exertion (i.e. contraction or tension of the muscles in the ventricles). walls increase during systole). This results in a limitation of the magnitude of the stroke and the proportion of extinction (15). Intramuscular vascular occlusion begins when a muscle contracts by 15% of maximal voluntary contraction (MVC) and when the muscle is resolved by about 70% of MVC. In patients with impaired heart disease and muscle strength, regular resistance training can improve heart function.

The ethal muscle undergoes vasoconstriction (narrowing) that does not occur in most MVCs (17).

**Increased peripheral vein tone**

Exercise is caused by an increase in veins (tension), which increases the mean blood volume and thus increases the filling pressure of the heart (ventricular overload). This increases cardiac output and reduces the risk of significant post-exercise hypotension (15).

**Increased stroke and heart muscle contraction**

Exercise causes certain contractions of the heart muscle (myocardium). This helps increase the size of the stroke and benefits from oxygen intake. An increased stroke results in approximately the same increase in cardiac functional activity, and an individual can then use that particular physical activity at the lowest level of the highest VO2 level. A lower heart rate reduces the double product, which reduces the oxygen needed by the heart muscle and reduces the risk of oxygen deficiency (angina pectoris). Aerobic exercise can improve blood circulation in the coronary arteries by increasing vascular elasticity and increasing vasodilation of endothelium-dependent arteries. Aerobic exercise is also caused by the regeneration of blood vessels, which increases the surface area of ​​the reservoir and blood vessels, as well as the density of the capillaries of the heart. The above effects help to increase the ischemic threshold with higher rates of angina distribution (15).

**Endothelial function and blood clotting system**

Studies in patients with myocardial infarction show that exercise has a positive effect on fibrinolytic enzymes. Exercise is also important to reduce the ability of platelets to blind. Together with an increase in plasma volume and a decrease in blood viscosity, these changes reduce the risk of blood clots forming in the coronary arteries. Vascular endothelium plays an important role in the regulation of arterial vascular tone, blood pressure and local platelet aggregation, ie. The ability of platelets to adhere by releasing endothelium-dependent release factors. One such factor is nitric oxide (NO), which releases the increased pressure (shear stress) that is applied to the walls of endothelial cells by increasing blood flow. Endothelium-dependent vasodilation is impaired in patients with ischemic heart disease. There is strong evidence that exercise improves endothelial function in healthy individuals and individuals with heart disease by increasing endothelium-dependent vasodilation capacity, especially in the context of increased NO release (18-22). There is also convincing evidence of positive effects of exercise on the blood coagulation system (fibrinolytics) (23, 24).

**Chronic inflammation**

Inflammation has been shown to be closely associated with the development of arteriosclerosis. Research has shown that aerobic exercise reduces the level of C-reactive protein (CRP), which means that regular exercise has an anti-inflammatory effect. There are currently no studies in patients with ischemic heart disease (25-27).

**Autonomic function**

Aerobic exercise can increase the threshold of ventricular tachycardia (extremely fast heart rate that stimulates the ventricles). This effect reduces the risk of sudden death by reducing the activity of the sympathetic nervous system and increasing paracophysical activity. Exercise VO2 max has also been shown to increase atrial fibrillation and ventricular arrhythmias (28-30).

Exercise also has a positive effect on many factors that are important for the development of cardiovascular disease. Examples are blood lipids, sensitivity to cholesterol and insulin. Other lifestyle changes are also important, and by including regular exercise in a new lifestyle, other lifestyle factors such as diet and smoking will have a positive effect. This in turn reduces the risk of cardiovascular morbidity and mortality.

**Exercise and its impact on death**

Cardiac rehabilitation exercise reduced overall mortality (20%), and especially heart disease-related deaths (cardiac death) (26%) compared to conventional treatment (31). Specific mechanisms that can contribute to the reduction of sport-related mortality have not been fully established and are likely to address a number of factors (17). Table 2 describes possible biological mechanisms to reduce mortality.

**Cardiovascular effect**

**Lower heart rate at rest and during exercise. Lower blood pressure at rest and during exercise.**

**Lower oxygen demand in the heart at submaximal levels of exercise training. Increase in plasma volume.**

**Increased myocardial contractility. Increased peripheral venous tone**.

Positive changes in fibrinolytic (blood coagulation) system. Increased endothelium-dependent vasodilatation.

Increased gene expression for production of an enzyme (NO synthase) that helps to produce nitric oxide (NO). Increased parasympathetic activity.

Increase in coronary blood flow, coronary collateral vessels and myocardial capillary density.

**Metabolic effect**

Reduced obesity.

Increased glucose tolerance. Improved blood lipid profile.

**Lifestyle effect**

Reduced likelihood of smoking.

Possible reduction of stress physiological responses. Possible short-term reduction of appetite.

Indication

All healthy people were physically passive and patients with long-term coronary artery disease.

PRIMARY PREVENTION

Numerous scientific studies over the last decade have shown that regular exercise improves the health of all age groups (32). Increased physical activity reduces the risk of death from heart disease (33). Physical inactivity is today considered a major risk factor for the development of coronary heart disease (34) and is an equally strong risk factor such as smoking, elevated blood fat, and high blood pressure (35). There is a dose-response relationship between levels of physical activity, cardiovascular disease and mortality, which means that any increase in activity levels means improvement! "A little is better than nothing, more than a little is better" (36).

Epidemiological studies investigating the effect of physical activity on the development and mortality of cardiovascular disease show that if the total amount of energy used for physical activity exceeds 4200 kJ per week ( 1000 kcal / week), such as walking longer than three hours per week per week plus activity / more intense exercise, the risk of developing coronary heart disease is reduced by 20% in men (37) and 30-40% in women (38). It is not uncommon to divide physical activity into shorter periods (39); the most important thing is that a person burns energy with physical activity.

Secondry prevention

Secondary prevention of coronary artery disease after an obvious cardiac event, such as myocardial infarction, means coronary intervention (coronary artery surgery or PCI) or permanent angina pectoris that can no longer be medically corrected (called refractory angina). it must be accepted to prevent death, recurrence, and progression of the underlying disease in the short and long term (16). Long-term heart disease requires regular, conditional exercise to reduce mortality. This means that training must be adapted to your current physical abilities. Because a person’s clinical picture and performance can vary from right to right, especially in the acute phase, these patients need special care. Before you start training, you should do a stress / fitness test with ECG monitoring and muscle function testing. Based on test results and medical history (history) to identify individual risk factors (physical inactivity, smoking, high blood fat, high blood pressure, overweight, diabetes), risk profile of physical fitness and possible symptoms during exercise.

Regular exercise is a powerful measure in cardiac rehabilitation that reduces the mortality rate by 26 percent. Training includes aerobic exercise 3-5 times a week and resistance training 2 times a week (see Table 3).

**Functional test**

Prior to each exercise, the physiotherapist must undergo some form of fitness test, in which general aerobic fitness and functional abilities are assessed before selecting fitness levels. Monitoring of a stress test / ECG fitness test is mandatory and should be done with current medications. Assessment of muscle function includes testing 10 RM (maximum repetition) before adjusting to the endurance training program.

The level of exercise was assessed using a questionnaire and a pedometer. This experiment can be repeated after the end of the training period to evaluate the results achieved by other training programs and physicians in fitness (40).

**Prescription**

**Type of activity**

The main goal of cardiovascular exercise is to increase aerobic capacity by replenishing the central bloodstream. Large muscle groups must be used for central circulation. Training can be done occasionally or remotely. A study in Norway showed that interval training has a higher VO2 value compared to distance learning (41). However, there are more and more interval studies and distance learning before we can conclude that one type of exercise is better than another for patients with coronary artery disease (42).

Each exercise should always begin with a warm-up phase and end with an activity performed over the same duration of the cooling phase. Interval training makes alternating periods more difficult and easier, while distance training maintains the same level of intensity during work (43). If your chest pain is severe, warm up a little longer than usual.

All exercises should begin 6–10 minutes of consecutive warm-ups with an intensity of up to 40–60% VO2 and 10–12 attempts on the Borg RPE scale (15, 44). The proposed interval training consists of three weight training exercises

4-5 minutes at an intensity of up to 60-80 percent VO2 max and try "a little hard" to "hard", which corresponds to a value of 13-15 on the RPE scale. During the loading interval, follow 4-5-minute workouts, light workouts with a maximum performance intensity of up to 40-60% and 10-12 trials on the RPE scale.

Distance learning means exercising at the same level for about 20-40 minutes. Weights can then be placed on the Borg scale 13-14. Each exercise should be done by cooling and stretching consecutively for at least 6-10 minutes.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Training method** | **Intensity** | **RPE\*\*\*** | **Frequency (times/week)** | **Duration** |
| Aerobic central circulatory training, distance or interval | 50–80% of  VO2 max\* | 12–15 central | 3–5 | 40–60 min./ session |
| Resistance training | 1–3 sets of  10–15 RM\*\*  (65–75% of 1 RM) | 13–16 local | 2–3 | 8–10 exercises |

\* VO2 max = Maximal oxygen uptake.

\*\* RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement only one time.

\*\*\* RPE = Rate of Perceived Exertion (Borg scale 6–20).

Before choosing an activity, there should always be a history of the patient’s physical activity, taking into account aerobic fitness, interests, and current needs. Aerobic exercise can take the form of brisk walking, running, cycling, swimming, fitness or water, skiing, skating, dancing or ball, depending on individual interests. 5 times a day. Sunday. This should be supplemented by daily training of at least 30 minutes, which is neither absolutely necessary at once, but which can include anything from normal movement to walking and climbing stairs (36, 39). The goal is to achieve a daily energy consumption of 660 KJ (² 150 kcal). When the cost of exercise energy is added to daily exercise, energy expenditure exceeds what is considered sufficient to achieve health effects (39).

In recent days, studies have shown that exercise is a safe and effective means of combat, which is sometimes contraindicated in cardiovascular disease (45, 46). Patients should do 1 to 3 sets of 8-10 resistance exercises, 10-15 RM, 2-3 times a week (15). If the patient has very little physical ability, in some cases, peripheral muscle exercise may be the type of exercise needed before other exercises to allow for other types of activity. A more detailed description of this type of exercise can be found in the section on heart failure.

WOMEN, the elderly and some groups of migrants, and cardiac rehabilitation

Studies show that women, the elderly, and some immigrant groups do not use adequate rehabilitation for cardiovascular disease (47), although these patients benefit greatly from cardiac rehabilitation (48, 49). Therefore, it is very important to provide and encourage this group of patients to participate in cardiac rehabilitation exercises.

**Interactions with drug therapy**

**Beta blockers**

Beta-adrenergic receptor blockers have a well-documented effect on coronary heart disease. They reduce the consumption of oxygen in the myocardium primarily by lowering the heart rate, but also by lowering blood pressure and reducing myocardial contractility. This effect is observed at rest and during exercise. The effect was similar to all drugs in this group and was dose-dependent (50). During exercise, local fatigue can occur, especially in the leg muscles due to reduced blood flow and consequent lack of oxygen in the working muscles (51). Regardless of the metabolic and circulatory changes observed in beta-blockers, the ability to absorb oxygen after exercise is improved in people receiving coronary heart disease and concomitant beta-blocker therapy, as well as in people without beta-blockers (52). The effects of exercise are independent of age and are similar to those achieved in healthy individuals (53).

Calcium channel blockers

Some calcium channel blockers ( verapamil, diltiazem) are negative on the chromatograph, which means that they reduce the resting heart rate and reduce the maximum heart rate. This usually limits the maximum amount of VO2, although the drug itself does not pose any particular risk to behavior.

**Diuretic**

Diuretics do not significantly affect heart rate and heart rate, but they do cause decreased plasma volume, peripheral resistance, and blood pressure. Diuretics can also cause hypokalemia, leading to muscle weakness and external ventricular pulse.

In hot weather, diuretics may have adverse effects due to an increased risk of dehydration and electrolyte disturbances (15).

**ACE inhibitors**

ACE inhibitors have a secondary inhibitory effect after myocardial infarction, especially in individuals with concomitant heart failure (50). From a hemodynamic point of view, this drug has the same effect during rest and exercise and lowers blood pressure by reducing peripheral resistance. There are no drugs that adversely affect the hemodynamic response during exercise.

**Nitrate**

The oldest drug still used in coronary heart disease is nitroglycerin. Nitrates are a short-term form that prevents individual attacks, and a long-term preventive form. Neither affects physical fitness and can sometimes be taken before training for preventative purposes (50).

**Contraindicated**

Absolute contraindications for physical activity and exercise include unstable angina pectoris and / or new, very debilitating symptoms. These people should be admitted to the hospital with medical and / or invasive care. Severe cardiac arrhythmias (e.g., ventricular tachycardia, complete atrioventricular block) cause obstruction, as well as poorly controlled hypertension and persistent infection that affects the general condition of the patient.

Relative contraindications. Tolerance to arrhythmias is usually reduced if the patient is hypoglycemic (low blood sugar) and / or dehydrated. It is therefore important to monitor these factors in all types of education, especially among those with heart disease.

**Risk**

The relative safety of controlled exercise in cardiac rehabilitation is well documented. The incidence of cardiovascular events during controlled exercise was low and ranged from 1/50 000 to 1/120 000 hours of exercise in non-fatal cardiac events and 1 death / 750 000 hours of exercise. Cardiac rehabilitation has always been associated with risk stratification in patients at increased risk of exercise-related cardiovascular events (15).

However, it is important to note that about half of all cardiac complications occur during the first month after an acute coronary event. After one year of follow-up, high-risk patients doubled their risk of heart attack compared to low-risk patients. Therefore, basic rehabilitation is under the supervision and guidance of a physical therapy specialist with access to a first aid kit. Pre-workout stress test ECG monitoring is an important tool for determining exercise levels as well as possible exercise-related symptoms that could negatively affect exercise ability (54).

# References

1. Libby P, Bonow RO, Zipes, DP, Mann DL. Braunwald’s Heart Disease. London: Saunders; 2007.

1. Davies MJ. The composition of coronary-artery plaques. N Engl J Med 1997;336: 1312-4.
2. Wallentin L. Akut kranskärlssjukdom. [Acute Coronary Artery Disease] Stockholm: Liber; 2005.
3. Lanza GA. Cardiac syndrome X. A critical overview and future perspectives. Heart 2007;93:159-66.
4. Roque M, Badimon L, Badimon JJ. Pathophysiology of unstable angina. Thromb Res 1999;95:V5-14.
5. Willems IE, Arends JW, Daemen MJ. Tenascin and fibronectin expression in healing human myocardial scars. J Pathol 1996;179:321-5.
6. Libby P, Bonow R, Braunwald E, Zipes D. Pathophysiology of heart failure. Amsterdam: Elsevier; 2004.
7. Heberden W. Pectoris dolor. In: Payne T, Ed. Commentaries on the history and cure of diseases. London; 1807.
8. Parry C. An inquiry into the symptoms and causes of syncope angionosa, commonly called angina pectoris. Illustrated by dissections. London: R Cutwell for Cadell and Davies; 1799.
9. Sanne H, Selander S. Mobilization and rehabilitation in cases of myocardial infarction. Läkartidningen 1967; 64:1539-45.
10. Hellerstein HK. Exercise therapy in coronary disease. Bull N Y Acad Med 1968;44: 1028-47.
11. Ekelund C, Ekelund L-G, Kinnman A, Rydén L, Sanne H, man-Rydberg A. Åter- anpassning efter hjärtinfarkt [Readjustment after Myocardial Infarction]. Stockholm: SPRI; 1980.

Fletcher GF, Balady GJ, Amsterdam EA, Chaitman B, Eckel R, Fleg J, et al. Exercise standards for testing and training. A statement for healthcare professionals from the American Heart Association. Circulation 2001;104:1694-740.

1. Balady GJ, Williams MA, Ades PA, Bittner V, Comoss P, Foody JM, et al. Core components of cardiac rehabilitation/secondary prevention programs. 2007 Update. A scientific statement from the American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee, the Council on Clinical Cardiology; the Councils on Cardiovascular Nursing, Epidemiology and Prevention, and Nutrition, Physical Activity, and Metabolism; and the American Association of Cardiovascular and Pulmonary Rehabilitation. Circulation 2007; 115:2675-82.
2. Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. Circulation 1999; 99: 963-72.
3. Edwards DG, Schofield RS, Lennon SL, Pierce GL, Nichols WW, Braith RW. Effect of exercise training on endothelial function in men with coronary artery disease. Am J Cardiol 2004;93:617-20.
4. Farsidfar F, Kasikcioglu E, Oflaz H, Kasikcioglu D, Meric M, Umman S. Effects of different intensities of acute exercise on flow-mediated dilatation in patients with cor- onary heart disease. Int J Cardiol 2007 Mar 16. (Epub ahead of print).
5. Gielen S, Adams V, Niebauer J, Schuler G, Hambrecht R. Aging and heart failure. Similar syndromes of exercise intolerance? Implications for exercise-based interven- tions. Heart Fail Monit 2005; 4:130-6.
6. Linke A, Erbs S, Hambrecht R. Exercise and the coronary circulation-alterations and adaptations in coronary artery disease. Prog Cardiovasc Dis 2006;48:270-84.
7. McAllister RM, Laughlin MH. Vascular nitric oxide. Effects of physical activity, importance for health. Essays Biochem 2006;42:119-31.
8. deJong AT, Womack CJ, Perrine JA, Franklin BA. Hemostatic responses to resistance training in patients with coronary artery disease. J Cardiopulm Rehabil 2006; 26:80-3.
9. Paramo JA, Olavide I, Barba J, Montes R, Panizo C, Munoz MC, et al. Long-term cardiac rehabilitation program favorably influences fibrinolysis and lipid concentra- tions in acute myocardial infarction. Haematologica 1998;83:519-24.
10. Caulin-Glaser T, Falko J, Hindman L, La Londe M, Snow R. Cardiac rehabilitation is associated with an improvement in C-reactive protein levels in both men and women with cardiovascular disease. J Cardiopulm Rehabil 2005; 25:332-6, Quiz 337-8.
11. Gielen S, Walther C, Schuler G, Hambrecht R. Anti-inflammatory effects of physical exercise. A new mechanism to explain the benefits of cardiac rehabilitation? J Cardiopulm Rehabil 2005; 25:339-42.