

# Comprehensive rehabilitation in chronic heart failure

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## Abstract

Chronic heart failure (CHF) is a complex disease process connected with cardiovascular system as well as other organs and skeletal muscles. In connection with the above, cardiac rehabilitation, consisting of exercise training and diet supplementation, aims at recovery of physical, social and psychic function and removing risk factors influencing the occurrence of circulatory system diseases. Evidence has shown that exercise training in CHF patients, both aerobic and resistance, can increase peak oxygen consumption and exercise capacity, improve NYHA (New York Heart Association) functional class, reduce mortality and improve the quality of life. Evidence suggests that most improvement is due to the effects of training on the peripheral circulation and skeletal muscle, rather than on the heart itself. Exercise training can improve skeletal muscle metabolism, increase blood flow within the active skeletal muscles, increase capillary density, promote the synthesis and release of nitric oxide, improve angiogenesis, and decrease oxidative stress. Physical effort reduces sympathetic arousal and increases parasympathetic arousal, thus reducing cardiac dysrhythmia and ischemia. Mitochondria start working harder, as the demand for energy is higher and electron flow provides energy in the form of ATP. Studies have consistently demonstrated that exercise training is safe and has no deleterious effect on central haemodynamics, left ventricular remodeling, systolic or diastolic function, or myocardial metabolism. Taking several supplements that have documented roles in medical therapy, including vitamins B, C and E, coenzyme Q10, alpha-lipoic acid, chromium, omega-3 polyunsaturated fatty acids, L-carnitine, and quercetin, has beneficiary effect on many diseases, including CHF. In our experience, 19 patients with CHF who undertook resistance (weight) training and food supplementation, returned to their normal activities after 4 months, without any complaints.

## Key words

chronic heart failure, exercise training, physical effort, weight training, supplementation, microelements, vitamins

## INTRODUCTION

Cardiovascular diseases represent a major health problem for the inhabitants of Eastern and Central European countries, including Poland [1, 2, 3, 4, 5, 6, 7]. Among them, chronic heart failure (CHF) is a leading cause of morbidity [8]. It is becoming more prevalent worldwide, mainly due to the ageing of the population and improved survival after acute cardiac events [9, 10]. The estimated prevalence of CHF in people aged 45 years or more ranges between 3 – 5% worldwide, although the true prevalence of CHF may be higher due to under-diagnosis of mild to moderate CHF [11].

CHF may be defined as the inability of the heart to meet the demands of the tissues, which results in symptoms of fatigue or dyspnea on exertion, progressing to dyspnea at rest [12]. This is a complex disease process connected with the cardiovascular system, as well as other organs and skeletal muscles.

In conjunction with the above, cardiac rehabilitation consisting of exercise training and diet supplementation aims at recovery of the physical, social and psychic function, and removing risk factors influencing the occurrence of circulatory system diseases.

## REHABILITATION THROUGH EXERCISE TRAINING

It was once believed that the basic element of treatment is the prone position, which improved diuresis, but reduced physical function and resulted in negative changes in the organism [13, 14, 15]. The concept of exercise training (ET) in patients with CHF developed in the late 1980s [16]. Coats et al. [17], in the first randomised study of training patients with stable CHF, showed that 8 weeks of exercise training led to an increase in exercise capacity and to an improvement of the abnormal sympathovagal balance. Today, supervised exercise programmes are recommended for all CHF patients as part of non-pharmacological management [8, 9, 10, 18, 19, 20, 21, 22, 23, 24, 25, 26]. According to Dafoe & Huston [27], exercise remains a cornerstone of cardiac rehabilitation programmes. Exercise training for patients even above the age of 65 years improves physical capacity by enhancing peak oxygen consumption ( $VO_2$  max) and, at the same time, extends the capacity period by 6–7 years, while resistance (weight) training facilitates the growth of strength and muscle mass, similarly in the case of young people, and should be continued to the end of life [8, 12, 16, 23, 24, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37].

Evidence suggests that most improvement is due to the effects of training on peripheral circulation and skeletal muscle rather than on the heart itself. Exercise training can:

- partially reverse activation of the neuro-hormonal system and reduce levels of pro-inflammatory cytokines;
- improve the ratio of type 1 and type 2 muscle fibres which reduces skeletal muscle fatigability;

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- improve skeletal muscle metabolism;
- increase blood flow within the active skeletal muscles;
- increase capillary density;
- promote the synthesis and release of nitric oxide;
- improve angiogenesis;
- decrease oxidative stress;
- improve vasodilatory capacity and endothelial function;
- reduce systemic vascular resistance;
- reduce skeletal myocyte apoptosis;
- increase IGF-1 expression in skeletal muscles;
- attenuate and/or reverse skeletal muscle atrophy;
- increase mitochondrial volume density;
- increase citrate synthase activity;
- improve oxidative and metabolic muscle capacity;
- reduce dependence on anaerobic metabolism [8, 16, 23, 32, 33, 38].

In a multi-centred study, the European heart failure training group [21, 32] reported that resting concentrations of noradrenaline and adrenaline decreased after training, indicating amelioration of the adrenergic overactivation commonly found in heart failure patients.

Animal studies demonstrated that exercise training attenuates the extent of apoptosis and alters the expression of contractile and heat shock proteins. Systematic training may have the potential to reduce the expression of the atrophy-related ubiquitin-proteasome pathway. Physical training seems to improve muscle protein synthesis through activation of the P13K/Akt molecular signalling pathway as a result of an increase of IGF-1 expression in skeletal and cardiac muscles. In addition, exercise training may attenuate protein degradation by inhibiting fork-head transcription factors (FOXO), which are related to proteolysis-mediated muscle atrophy. Exercise training activates several favourable signalling pathways that lead to increased peroxisome proliferator-activated receptor gamma co-activator (PGC-1 $\alpha$ ) expression in skeletal and cardiac muscles, a transcriptional factor which, in turn, regulates mitochondrial biogenesis and fatty acid oxidation [16].

Moreover, exercise training also has positive impact on the autonomic nervous system, circulatory system, endothelium function, and activation of muscle ergoreceptors for those suffering from heart failure. Exercise has been shown to reduce traditional risk factors, such as hypertension and hyperlipidemia, attenuate cardiovascular responses to mental stress, and reduce myocardial ischemia [39]. Physical effort produces changes in the left chamber of the heart and central haemodynamic parameters, which translates into better effort tolerance, reduction of tiredness and dyspnea; it also improves the capacity of the circulatory system [12, 40, 41, 42]. Studies have consistently demonstrated that training is safe and has no deleterious effect on central haemodynamics, left ventricular remodeling, systolic or diastolic function, or myocardial metabolism [20, 25].

The net functional result of such improvements is an increase in exercise duration and physical work capacity, as well as a reduction in mortality, morbidity and hospital admissions. Evidence suggests that exercise training in CHF patients can increase exercise capacity by 15%, improve NYHA (New York Heart Association) functional class, reduce mortality by 35%, and improve the quality of life [8].

According to Dubach [20], training increases exercise tolerance by an average of 20% in chronic heart failure

regardless of etiology. Belardinelli et al. [31] found that exercise training was associated with growth of peak VO<sub>2</sub> by 18%, improved quality of life, and significantly lower mortality ( $P=0.01$ ), and fewer hospital re-admissions for CHF ( $P=0.02$ ). These results, however, were not confirmed by an EXERT study, except for growth of peak VO<sub>2</sub> [42]. The systematic review carried out by Davies et al. [43, 44] showed that in systolic CHF patients, exercise-based intervention reduces the level of hospitalizations due to heart failure and improves the health-related quality of life, but does not decrease all-cause mortality. By contrast, a comprehensive metaanalysis by Jolliffe et al. [45] reported a 27% reduction in all-cause mortality and a 31% reduction in cardiac mortality. Dalal et al. [46] report that two systematic reviews that included 48 randomised controlled trials showed a 20% reduction in all-cause mortality and a 27% reduction in cardiac mortality at 2 – 5 years.

As patients develop greater exercise tolerance, they experience less fatigue and dyspnea and become more comfortable performing activities of daily living. This, in turn, leads to increased independence, less chronic illness behaviour and depression, and an improvement in the general sense of wellbeing [8, 21].

Most of studies on exercise training have been carried out with the use of aerobic training. Until the late 1990s, resistance training (such as weight lifting) was contraindicated for CHF patients, and the existing guidelines still view it with great caution. However, there is a growing body of evidence that resistance training is indeed safe for most CHF patients, and may even be more effective than aerobic training alone in reversing peripheral metabolic limitations and skeletal muscle myopathy, and in increasing overall functional capacity [16]. Numerous studies have shown that resistance training increases maximal strength and muscular endurance [47]. Węgrzynowska et al. [47], report that after 12 weeks of resistance training, all 10 CHF patients were able to change their classification from III to II NYHA class. According to Dafoe & Huston [27], there appears to be less myocardial ischemia during resistance training than during a standard exercise test.

Patients with chronic, low-risk CHF, in NYHA classes I-III, with moderate to good left ventricular function, without angina or ischaemic ECG disturbances and dysrhythmias, and without uncontrolled hypertension or moderate to severe aortic stenosis, may follow a moderate resistance training programme. Depending on the NYHA class, the strengthening of 4–10 muscle groups of the upper body, lower body and the trunk is suggested. The resistance training session may last 10–30 min and be repeated on 2–3 days per week. The primary goal of exercise prescription is to develop an individualised exercise programme combining aerobic with resistance training in order to enhance cardiorespiratory and muscular fitness, increase total physical capacity, and improve the quality of life of CHF patients [16].

According to the experience of one of the authors of the presented study (A. G.), weight training is necessary with proper individualised management, and extends the exercise endurance and capacity measured with the VO<sub>2</sub> max. Improving motor fitness, strength and strength endurance produces natural chemical compounds in the human organism which lower blood pressure, provides good wellbeing by producing endorphins, reduces pulse and dyspnea during physical effort, and improves lung ventilation

and capacity. Furthermore, it raises the anaerobic metabolism threshold and inhibits catabolic processes concerning heart failure.

Physical effort reduces sympathetic arousal and increases parasympathetic arousal, thus reducing cardiac dysrhythmia and ischemia. Mitochondria start working harder as the demand for energy is higher, and electron flow provides energy in the form of ATP. Enhanced physical activity, in order to retain the growth of glucose burning, produces more enzymes, and in a shortage of glucose it uses fat reserves. A properly adapted organism develops a suitable level of energy production. Enhancing physical activity is accompanied by a concurrent growth of destructive free radicals and NO, which is an excellent antioxidant. Through its activity in the cells of blood vessel endothelium, nitric oxide regulates the tension of the blood vessel, has a spasmolytic effect on blood vessels, and among other neuro-hormonal factors it improves the blood flow, preventing thromboembolic changes [33, 38, 48, 49, 50].

Physical training, when very intense, weakens the immune system of elderly people suffering from cardiovascular diseases; therefore, it must be individually adjusted. People exercising regularly are better adjusted and their energy production is more effective, which is a result of producing a number of basic antioxidant enzymes and general antioxidant stability [51, 52, 53, 54].

Physical exercise reduces the incidence and mortality from heart diseases. It is the greatest available opportunity to slow down damage resulting from the process of ageing. Those who performed work requiring high physical activity were less exposed to sudden death in general, or as a result of cardiovascular diseases, than those whose work required less physical activity [55, 56].

More benefits regarding the prevention of cardiovascular diseases are derived from physical exercise performed in a state of rest and relaxation, than from professional work, which is connected with the psychic attitude towards the purpose of physical activity. It is believed that exercise and the related wellbeing constitute important elements for disease prevention [57, 58].

Unfortunately, in spite of its advantages, exercise training is not common among CHF patients. According to Dalal et al. [10], about 900,000 people are living in the UK with heart failure, but only a small minority participate in cardiac rehabilitation. In another study, Dalal et al. [46] report that home- and centre- based forms of cardiac rehabilitation seem to be equally effective in improving the clinical and health-related quality of life outcomes in CHF patients. Schmidt et al. [59] report that tele-monitoring (mostly by telephone) has the potential to improve CHF patient care in many ways and reduces mortality by ca. 20%. According to Piotrowicz [60], home tele-rehabilitation secured by tele-electrocardiographic-monitoring and tele-heart-rate-monitoring seems to be the optimal form of physical activity for heart failure patients.

## SUPPLEMENTATION WITH NUTRIENTS

Heart failure treatment based only on pharmaceutical (conventional) practices further deteriorates the health problem, as in many cases heart failure is caused by a chronic shortage of vitamins and other nutrients in cardiac muscle cells, while diuretic drugs are oriented towards symptoms, not causes. This shortage leads to the weakening of heart

functioning as a pump, which results in deterioration of organ blood supply, low arterial pressure and no coverage of current metabolic needs of the organism. Kidney functions, which depend on optimal blood pressure, are impaired, water in the tissues stops and, if diuretics are supplied, water soluble vitamins such as C and B, minerals and microelements are removed from the organism.

According to von Haehling et al. [61], dietary deficiencies in micronutrients and macronutrients contribute to the progression of chronic heart failure from stable disease to devastating cardiac cachexia. The evidence suggests that multiple micronutrient supplementation is potentially beneficial for cachectic patients, and should contain antioxidant supplements and B-group vitamins. Heart failure treatment without refuelling cell nutrients is a non-comprehensive treatment [62].

With a long-term shortage of vitamins the recovery process in vascular walls is excessive. The basic elements for the recession of atherosclerosis are lysine and proline, which comprise collagen, and vitamin C. They are decisive factors in optimal regeneration of the connective tissue of vascular walls. With optimal volumes of nutrients, the cells of the smooth muscles of the vascular wall produce a sufficient amount of activity collagen, guaranteeing wall stability. Vitamins C and E prevent excessive growth of muscle cells [62].

According to Janson [63], taking several supplements that have documented roles in medical therapy, including vitamins C and E, coenzyme Q10, alpha-lipoic acid, chromium, L-carnitine, and quercetin, has beneficiary effect on many diseases, including congestive heart failure.

Aberrations in minerals and micronutrient homeostasis that includes Ca<sup>2+</sup>, Mg<sup>2+</sup>, vitamin D, zinc and selenium deficit, appear to be an integral component of pathophysiologic expressions of congestive heart failure and predispose patients to secondary hyperparathyroidism which accounts for bone resorption and contributes to a fall in bone strength that can lead to nontraumatic fractures. Thus, patients with CHF need daily nutrient supplement in addition to their habitual diet [64].

The deposits in blood vessels are mainly caused by lipoprotein which sticks to collagen molecules in arterial walls. The therapeutic goal of preventing the forming of fat deposits in vascular walls is neutralisation of Lp(a) molecule viscosity, and preventing its attaching to the inner structures of vascular walls. Amino-acids – lysine and proline – form a protective layer around Lp(a) molecules, which prevents the depositing of fat molecules in the vascular wall. They release Lp(a) attached to the vascular wall, which are transported to the liver, where they undergo natural metabolic conversion. This process improves circulation [65, 66, 67, 68].

An additional mechanism influencing the development of atherosclerosis, brain stroke or heart attack, is biological oxidation. Free radicals destroy the tissue of vascular walls, contributing to the depositing of atherosclerotic plaques. Vitamins C and E, beta-carotene, among others, are the strongest antioxidants which protect the cardiovascular system against damage [65, 66, 67, 69, 70, 71, 72].

Modern cell medicine presents a new view concerning the cause of secondary risk factors for the blood system and defines methods of heart disease prevention. Cholesterol, triglycerides, low density lipoproteins (LDL), Lp(a) are repair factors and their level increases in a response to structural

weakening of vascular walls. Chronic shortage of vitamins and other nutrients leads to overproduction of repair molecules and deposits of atherosclerotic plaques. The most significant way of reducing the level of cholesterol and other secondary risk factors in the blood system is stabilisation of vascular walls and, in consequence, reduction of the metabolic demand for increased production of those factors in the liver [73, 74, 75, 76].

The best natural sources for reducing risk factors in the blood are, e.g. vitamin C, B3 (nicotinic acid), B5 (pantothenate), vitamin E, carnitine, coenzyme Q10, vitamin B1, which provide better stability of the vessels' connective tissue and reduction of demand for repair molecules.

Coenzyme Q10 (ubiquinone, or coQ10) is the most important element in the respiratory pathway, particularly in the heart muscle. It participates in ATP synthesis and is responsible for cell metabolism. It additionally acts as an antioxidant. Up to the age of 30, the human organism produces this coenzyme, after which the production decreases reaching only 50% at the age of 70. Coenzyme Q10 improves heart capacity and corrects energy flow in mitochondria, the majority of which can be found in heart muscles. CoQ10 is essential for the heart muscle, and it helps lower blood pressure, improve congestive heart failure, and protect the brain in degenerative conditions such as Parkinson's and Alzheimer's diseases. It also reduces the chance of blood platelets sticking together and obstructing the vessels, thus reducing the chance of heart attacks. It improves immunity, physical capacity, reduces the process of ageing, strengthens the immunity system; it is also used in the case of diabetes and obesity [63, 66, 70, 71, 77].

Scientific and clinical research has confirmed the special importance of carnitine, coenzyme Q10 and other nutrients vital for the improvement of heart muscle work and increasing its contraction. Carnitine optimises fatty acid metabolism and reduces triglyceride level. L-carnitine is essential for the transport of free fatty acids across the mitochondrial membrane, where they are metabolized to create energy. Low L-carnitine levels reduce the functional capacity of the myocardium, leading to increases in angina and congestive heart failure [63, 78, 79, 80, 81, 82, 83].

In the event of heart failure, vitamin C should be administered; it provides energy for cell metabolism and supports the activity of group B vitamins, which are carriers of cell metabolism bioenergy, concerning in particular heart muscle cells, improving heart beat. This is further enhanced by the production of prostacyclin in arterial walls, tissue hormone inhibiting blood platelets sticking together and expanding blood vessels, especially the coronary vessels, and ensuring the protection and natural healing of vascular walls and normalising increased production of cholesterol and other risk factors in the liver, and their level in blood.

It is necessary to administer vitamin E, which is an antioxidant and provides protection for cell membranes, lipids and cells against self-oxidation, as well as taurine amino-acid, whose shortage in heart muscle cells is often a cause of heart failure, and carnitine, which supports the administration of bioenergy to mitochondria in cells and coenzyme Q10, the most important element of each cell's respiratory pathway. The production and transportation of electric impulse for regular heart beat is the responsibility of the heart muscle cells, which must be properly supplied with relevant nutrients [62, 65, 69, 70, 71, 78, 79, 84].

Recent epidemiological and experimental studies suggest that omega-3 polyunsaturated fatty acids (w-3PUFA) of marine origin may prevent the development and progression of CHF at relatively low intakes, and can be achieved solely through high consumption of oily fish. w-3PUFA supplementation can favorably affect inflammation, plasma lipid profile, blood pressure, and cardiac mitochondrial function, all of which could prevent the development and progression of heart failure. Clinical studies suggest that a high dietary intake of the w-3PUFA found in fish oils (eicosapentaenoic and docosahexaenoic acids) may lower the incidence of CHF, and that supplementation with pharmacological doses prolongs the event-free survival in patients with established CHF [85].

## PROPERTIES OF VITAMINS AND METHODS OF THEIR ADMINISTERING

**Vitamin C.** Synthesised from glucose in the livers of mammals, except humans, i.e. humans must receive it from an outside source. Vitamin C has the following properties:

- reduces the level of free radicals and slows down the LDL oxidation process;
- enhances immunity and protects against infections;
- regenerates the oxidation properties of vitamin E;
- accelerates wound healing;
- participates in collagen production;
- plays an important role in the growth of blood vessel cells;
- participates in the synthesis of adrenal cortex hormones;
- facilitates iron absorption;
- together with folic acid and vitamin B 12, triggers erythrocyte maturation;
- builds blood vessels;
- inhibits the creation of nitrosamines, which contribute to neoplasm development;
- weakens allergic reactions;
- prevents vein clots;
- prevents scurvy;
- without vitamin C, there is no benefit from the properties of other vitamins, e.g. folic acid and iron;
- vitamin C inhibits the sticking together of blood platelets (prostacyclin) and expands coronary vessels;
- provides energy for cell metabolism and supplies the transport molecules of group B vitamins with necessary bioenergy [62, 74, 86, 87, 88].

**Vitamin E.** Appears in 90% of cases in the form of alpha-tocopherol, and breaks chain reactions that generate free radicals. It is a modulator of the whole system of enzymes, which are related to cell membrane and slows down atherosclerotic changes. The functions of vitamins C and E are interrelated and reduce the risk of atherosclerotic changes in vessels. Vitamin E collects in the liver, heart, muscles, adipose tissue, uterus, testicles, blood, endocrine glands and glands of the bile duct. About 70% of the daily dose is excreted in faeces. This vitamin expands blood vessels and inhibits blood clotting by dissolving the clots. It is very active in cooperation with selenium. Vitamin E:

- inhibits the process of cell ageing;
- with vitamin A protects lungs against air pollution;
- accelerates burn healing;
- protects against miscarriage;
- relieves muscle contraction;

- prevents cardiac ischemia;
- softens scars;
- prevents tiredness;
- the dose of vitamin E should be raised during menopause;
- the dose should also be raised for nursing mothers and those who receive contraceptions, as well as persons who drink chlorinated water;
- the dose of vitamin E should be increased in the case of high pressure;
- inorganic Fe may compromise the activity of vitamin E, unlike organic Fe;
- plays a very important role in the conversion of hydrocarbons, fats and proteins as a medication during convalescence after different kinds of diseases;
- protects polyunsaturated fatty acids and vitamin A against free radicals, preventing atherosclerosis;
- in order to prevent further damage to heart muscle, vitamin E should be administered after heart attack;
- inhibits smooth muscle cell proliferation, platelet adhesion and aggregation, and monocyte endothelial adhesion [89];
- reduces blood pressure and has diuretic, although mild, properties;
- has a positive impact on skin and hair;
- has a positive impact on the nervous system;
- has a positive effect during infections;
- enhances immunity;
- has a supportive effect during lung treatment;
- relieves trembling hands and muscles in Parkinson's disease.

Several studies have reported that vitamin E is associated with decreased chronic disease risk. The Women's Health Study, a 10-year prevention trial in normal, healthy women, found that 600 IU vitamin E decreased overall cardiovascular mortality by 24%, and in women over 65 years of age decreased it by 49% [90].

Lack of vitamin E and a significant shortage may lead to damaging nerves in the spinal cord and damage to the retina.

Drugs reducing cholesterol level inhibit the absorption of vitamin E and other vitamins soluble in fats.

**Vitamins B.** Vitamin B1 (thiamine) provides optimisation of cell metabolism and the supply of bioenergy, and has a positive effect on the muscular system, heart function and nervous system.

Vitamin B2 (riboflavin) provides optimisation of cell metabolism and supply of bioenergy:

- together with vitamin A it retains the proper function of mucous membranes of the urinary system, alimentary tract, blood vessel epithelium and respiratory tracts;
- helps assimilate Fe;

Vitamin B3 (nicotinic acid) reduces excessive production of cholesterol and lipoproteins in the liver.

Vitamin B6 (pantothenic acid) is a structural component of the major metabolic cell molecule (coenzyme A), optimising degradation and synthesis of fats in metabolism. Vitamin B6 and folic acid accelerate the decrease of risk factor of homocysteine in cell metabolism. Vitamin B6 is necessary in the production of red blood cell antibodies and participates in energy acquisition processes [68, 83, 91, 92].

By using cell nutrients, the physical condition is improved, lifespan extended, heart pumping improved, increased heart chambers are normalised and shallow breathing is decreased.

## EXAMPLE OF GROUP REHABILITATION ILLUSTRATING BENEFITS OF RESISTANCE TRAINING ASSOCIATED WITH DIET SUPPLEMENTATION

Clinical research was conducted by one of the authors of the presented study (A. G.) on 19 working patients aged 40–65 years (10 women, 9 men) with advanced heart failure and atrial fibrillation, with coronary pains and ischemia.

The patients were advised to take exercise typical for such diseases, i.e. weight and flexibility training and dietary supplements. Weight training was performed in a properly equipped university gym at the Academic Sports Centre, taking into account all muscles groups of shoulder, hip, lower and upper limbs. The training took place 4 times a week, with an obligation to attend at least 3 sessions per week.

The patients undertook 20–30 different weight exercises, depending on their progress, under the supervision of an instructor. The training was individualised, adjusted to needs and capacities with gradable difficulty, and repeated in 3–5 series. Furthermore, each patient had to perform specific exercises every day at home for 15–30 minutes, and had individual goals defined for them, based on the course of treatment, lifestyle, health condition and age. One-time physical effort, especially performed by a person with a sedentary lifestyle, increases the likelihood of clotting. Appropriate training eliminates such a reaction.

Within the controlled process of rehabilitation the authors strive to achieve goals and then retain physical function at the highest possible level, reducing stress connected with disease and treatment. In the process of rehabilitation, muscle mass should be increased and the volume of adipose tissue reduced, which is achieved by combining physical exercise with proper diet.

The 19 patients were given dietary supplements in the form of Aloe (Barbadensis Miller, dosage 2 × 50 ml before meals in morning and evening), containing amino-acids, such as lysine, proline, carnitine and other (2 × 50 ml daily), large doses of vitamin C (1,000 mg) in tablets produced from briar rose, sea-buckthorn and blackcurrant with bioflavonoids and vitamin E, carotene, group B vitamin, coenzyme Q10, and microelements, such as Ca, Mg, K, Na, Mo, Cu, Cr, and other vitamins and microelements contained in natural ecological products of vegetable and fruit diet.

An improvement in heart muscle function parameters (increasing ejection fraction) was recorded for all patients; pulse rate was balanced, muscle blood supply (including heart muscle) improved, strength and strength endurance enhanced, as well as general endurance. After 4 months of training, the patients did not experience any disorders, even at longer physical effort and returned to their everyday activities. The training was continued after the study period.

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