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The use of neuromuscular electrical stimulation of the lower limbs skeletal muscles in cardiac rehabilitation of patients with chronic heart failure

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Abstract

Research conducted in recent years suggests that neuromuscular electrical stimulation of the lower limb muscles (NMES) may be an alternative to physical training in patients with chronic heart failure (CHF). Through stimulating the work of the muscles, NMES raises the exercise tolerance, muscle mass and endurance in patients with CHF. A beneficial effect of NMES on muscle blood flow, aerobic enzymes activity, vascular endothelial function, decrease in pro-inflammatory cytokines and improvement of quality of life has been indicated. It must be emphasized that NMES procedures are comfortable for patients with CHF due to lower exercise load and therefore less intense symptoms of dyspnea. Moreover, these procedures may be conducted at home (after prior training). The possible side effects include temporary muscle soreness and skin impairment resulting from improper placement of electrodes (patients in ambulatory conditions).

Keywords: chronic heart failure; neuromuscular electrical stimulation

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INTRODUCTION

Chronic heart failure has become one of the most serious health issues of people worldwide. The main clinical symptom of CHF is the progressive decrease in physical capacity related to dyspnea and weariness during exercise. In the initial phase of the condition these symptoms occur during physical exercise. However, as the condition progresses, the symptoms begin to occur during daily activities, leading to the limitation of the patient's independence (Mann, 2004). Therefore, CHF is not only a medical but also a social issue, as it leads to a significant decrease in quality of life, disablement and social isolation. Furthermore, CHF contributes to an increased number of hospitalizations which results in the increase of funding necessary for the treatment of the condition.

In the last 20 years, a significant development was noted in the treatment of CHF through introducing converting enzyme inhibitors, aldosterone receptor antagonists, beta-adrenolitics, cardiac resynchronization therapy (CRT) and cardiovascular procedures to the treatment. Nevertheless, despite the use of these modern methods of treatment, the annual mortality due to CHF reaches 10% (Kucio et al., 2011). Therefore, a search for new, more efficient methods of treatment and early diagnosis is one of the main challenges of the XXI century. It seems that the overcoming of this condition cannot be reached through the use of one method alone, whether it be a drug, a gene, a stem cell or a heart transplant. The treatment and prevention of CHF needs to be complex. As the recent studies suggest, an important aspect of the treatment, except pharmacological and invasive methods, is a lifestyle change, embracing the increase in physical activity and properly conducted physical training.

THE IMPORTANCE OF PHYSICAL TRAINING IN NON-PHARMACOLOGICAL TREATMENT OF CHF

Research conducted in the last 25 years indicated that application of physical training in patients with CHF is safe and provides numerous benefits (Table 1). Among these benefits are the improvement of physical capacity and increased oxygen consumption at the peak moment of physical exercise (Pina et al., 2003; Da Silva et al., 2002). Physical training also improves ventilation efficiency, which means that under certain workload a decrease in minute ventilation, delay in the occurrence of anaerobic threshold ventilation and a smaller slope of the line between minute ventilation and production of CO_2 (VE/CO_2) are observed. It seems that the decrease of ventilation after training may depend upon several factors: lower blood lactate levels during exercise, illustrating the increased blood supply to the exercised muscle, increased oxygen uptake by the muscles or better muscular metabolism (Pina et al., 2003). This indicator may depend upon the decrease of musculoskeletal ergoceptor reactions and the increase of baroreceptor reactions (Ponikowski et al., 2001). Due to physical training also increased diffusion capacity of the lungs and capillary plexus extension were observed (Guazzi et al., 2005). Also, physical exercise has a beneficial effect on the myocardium, as an improvement in the left ventricle's diastolic function, both in idiopathic dilated cardiomyopathy and coronary cardiomyopathy was observed. The increase in the left ventricular wall tension during diastole was lower after training, and the increase of $VO2_{peak}$ correlated significantly with the increase of the inflow velocity in the early diastole phase (E-wave) and the decrease in the late auricular wave flow velocity (A -wave) (Belardinelli et al., 1996). Hambrecht (Hambrecht et al., 2000), by contrast, demonstrated the improvement of indicators (ejection fraction) of resting heart rate and cardiac output during physical activity. The improvement was related to the reduction of the end-diastolic volume of the left diastole and peripheral vascular resistance. Also, it was indicated that physical activity counteracts the remodeling of the left diastole in patients with CHF and it may be related to the beneficial effects of physical exercise on neuro-hormonal disorders (Haykowsky et al., 2007).

Table 1. Beneficial impact of physical exercise in heart failure

1. **Central factors: heart and circulation**
- Improved systolic and diastolic function
- ↑ coronary blood flow

1. **Lung function**
- ↑ diffusing capacity of the lungs

2. **Peripheral factors**
- ↑ VO_{2peak} and ↓ VE/VO_{2slope}
- ↑ blood circulation in lower limbs
- ↑ capillary density in muscles
- ↑ endothelial NO release
- ↑ slow-twitch muscle fibers (1^{st} type) and ↓ fast-twitch muscle fibers (2^{nd} type)
- ↑ mitochondrial density in skeletal muscle cells
- ↓ ergoreceptor response activation
- ↓ oxidative stress
- ↓ pro-inflammatory cytokines concentration
- ↓ adrenergic system activity and ↑ cholinergic system activity
- ↓ RAA system activity
- ↓ vasopressin, endothelin and natriuretic peptides concentration

3. **Improved quality of life**
4. **Prolongation of survival and – hospitalization frequency**

Physical training reduces the resting blood concentration of neuro-hormones related to the progression of heart failure such as: angiotensin II, aldosterone, vasopressin, brain natriuretic peptide

(Haykowsky et al., 2007; Passimo et al., 2006). Moreover, physical activity increases cholinergic system activity while reducing adrenergic system activity (Fraga et al., 2007). These beneficial changes may slow the progression of heart failure and vulnerability to ventricular arrhythmia, which often lead to sudden cardiac death. Also, due to physical training, a reduction of oxidative stress and pro-inflammatory cytokines in blood is observed, which in turn leads to reduced inflammation and catabolism of musculoskeletal muscles (Belardinelli, 2007). The effects of the aforementioned activities result in: increased blood flow in muscles, increased capillary vessels density, increased endothelial NO release. Furthermore, increase in the number of slow-twitch muscle fibers (type I) and reduction in the number of fast-twitch muscle fibers (type IIx), as well as increased mitochondrial density and oxidative enzymes activity are observed due to physical training (Ventura-Clapier et al., 2007; Hambrecht et al., 1995). These changes are responsible for the reduction of blood lactates, anaerobic metabolism, phosphocreatine loss and increased ATP resynthesis. An improvement of the impaired oxidative transformation ability in the musculoskeletal muscles of people with CHF is observed (Mann, 2004). Additionally, due to aerobic metabolism improvement and simultaneous reduction of myocyte acidifying ability, physical training can contribute to the reduction of excessive muscular ergoreceptor activity, thereby leading to reduced ventilation frequency during physical activity and adrenergic system stimulation (Ponikowski et al., 2001). By contrast, reduced respiratory drive, sympathetic and vasoconstriction reaction occurring due to physical activity may be the link between musculoskeletal disorders and reduction in physical activity tolerance, dyspnea and lower quality of life in patients with CHF.

Physical training improves not only physical capacity but also the quality of life related to health and well-being (Flynn et al., 2009). Patients are in better physical and mental shape and are able to perform daily activities with less help from other persons, which in turn makes them feel more independent and find the condition less perceptible. It needs to be accentuated that the benefits of a short-term treatment protocol should lead to a long-term lifestyle change.

The research conducted was minor, short-term and evaluated physiological endpoints. For the first time an attempt was made to answer the question whether physical activity reduces mortality and morbidity in patients with CHF through ExTraMATCH meta-analysis (*Exercise Training Meta-Analysis of Trials In Chronic Heart Failure*) (Piepoli et al., 2004). The meta-analysis was based on 9 studies involving 801 patients. A 32% relative risk reduction was indicated for mortality and 23% for combined parameter: the number of deaths or hospitalizations while comparing physical training to pharmacological treatment. In HF-ACTION, a randomized, multicenter (conducted in the United States, Canada and France) study published in 2009, effects of physical training in 2331 relatively young (the mean age was 59 years), clinically stable patients with low or moderate intensity symptoms (63% of patients in activity class II and 35% of patients in activity class III according to NYHA classification) and EF $\leq$35% were evaluated (O'Connor et al., 2009). The evaluated intervention involved 36 training sessions during the first 3 months and individual home training afterwards. The median observation time amounted to 30 months. In the corrected analysis physical training resulted in a 11% decrease in occurrence frequency of the main endpoint, including deaths from any cause and hospitalization from any cause. Moreover, a relative decrease of 15% in secondary endpoint risk, including deaths from cardio-vascular causes and hospitalization from CHF was indicated. Mortality reduction was not indicated and no reservations to the safety of this therapy were brought up. The patients' adherence to the recommendations regarding physical exercise significantly decreased after the supervised trainings phase. The available scientific evidence indicates that physical training is beneficial to patients with CHF, although typical elderly patients were not involved in most studies, and optimal characterization of recommended activity remains unknown. This data has given a basis for the European Cardiology Society to include physical training as one of the non-pharmacological forms of CHF treatment in the 2012 ECS guidelines (guideline class I, evidence level A) **(**McMurray et al., 2012)

APPLICATION OF NEUROMUSCULAR ELECTRICAL STIMULATION IN CHF TREATMENT

The collected epidemiological data indicate that CHF patients suffer from locomotory disorders impeding their physical activity performance. Moreover, CHF patients cannot undergo physical training due to significantly advanced or exacerbated heart failure (NYHA IV). CHF patients also tend to abandon physical training due to the discomfort related to dyspnea (Kucio et al., 2011).

Obviously, neuromuscular electrical stimulation (NMES) consists in the application of properly characterized electricity to the skeletal muscles in order to cause certain reactions, such as:

- Increasing normal muscle strength
- Strengthening of a muscle after surgery, preventing atrophy or restoring strength after extensive atrophy
- Reeducation of motor control of the muscle
- Gaining or increasing range of motion in the joint
- Increasing muscle endurance
- Change in muscle structure and function

Since the implementation of NMES to the clinical practice researchers have been searching for ways to effectively utilize it. Modern technology enables better choice and control of stimulation parameters. The devices tend to progressively decrease in size, become less costly and more mobile. Therefore increasingly more research into the use of NMES for treating other conditions is conducted.

Hitherto, the neuromuscular electrical stimulation method (NMES) was used in the treatment of patients after stroke (Geraldine et al., 2005; Morone et al., 2012) or after orthopedic surgeries (Stevens-Lapsley et al., 2012). In few studies an attempt was made to use NMES in pulmonological rehabilitation of patients with chronic obstructive pulmonary disease (Neder et al., 2002; Bourjeily-Haber et al., 2002; Dal Corso et al., 2007; Zanotti et al., 2003; Vivodtzev et al., 2006)

Research conducted in recent years (Table 2) suggests that neuromuscular electrical stimulation of the lower limb muscles may be an alternative to physical training in patients with chronic heart failure (Banerjee, 2010)

Studies conducted in recent years indicated that the use of NMES in treatment of patients with CHF results in improvement of physical capacity indicators such as: increase in peak oxygen uptake, anaerobic threshold, prolonged stress test time as well as prolonged distance in the 6MWT test (Karavidas et al., 2006; Maillefert et al., 1998; Vaquero et al., 1998; Harris at al., 2003; Nuhr et al., 2004; Dobsak et al., 2006, Dobsak et al., 2006; Deley et al., 2008; Karavidas et al., 2008; Karavidas et al., 2010; Banerjee et al., 2009; Soska et al., 2012). After a series of NMES treatments in patients with CHF increased fatigue resistance is observed (Harris et al., 2003). The strength of the stimulated muscle increases by 11-20% in comparison to the initial value. The aforementioned increase in muscle strength affects both the muscle tension in isokinetic (Dobsak et al., 2006[a]; Quittan et al., 1999; Quittan et al., 2001) as well as isometric conditions (Quittan et al., 1999). It seems that these changes occur due to the increase of aerobic enzyme activity in the skeletal muscles, which leads to increased oxidative ability (Karavidas et al., 2006; Harris et al., 2003; Nuhr et al., 2004; Dobsak et al., 2006[a]; Karavidas et al., 2008). Furthermore, it was observed in the biopsies that under the action of NMES type I myosin content increases, which proves the significant retuning of the muscles towards aerobic metabolism and muscular endurance (Nuhr et al., 2004). Under the action of NMES also increased blood flow in the muscle occurs (Dobsak et al., 2006[a]). This phenomenon may be related to the improvement of anti-inflammatory L10 cytokine to pro-inflammatory TNF cytokine ratio resulting from NMES treatment, which can have a beneficial anti-inflammatory effect. Additionally, it was observed that NMES treatment significantly reduces the number of dissolved forms of adhesion molecules such as intercellular adhesion molecule (sICAM-1) and vascular cell adhesion molecule (sVCAM-1), which occur as the "final products" of interaction between active monocytes and endothelial cells. According to the researchers, NMES is a type of workout which improves endothelial function and increases physical capacity in patients with CHF, therefore breaking the vicious cycle between improper peripheral inflammatory responses, nitric oxide metabolism anomalies and vascular-muscular dysfunction (Karavidas et al., 2006).

It is worth accentuating that in the electrically stimulated muscles no increase in lactic acid concentration or lactic dehydrogenase is observed (Harris et al., 2003). Although, as observed by Dobsak et al. (Dobsak et al., 2006[b]), a minor and statistically insignificant increase in blood creatine kinase and lactic dehydrogenase occurred in patients with severe heart failure (NYHA class IV). These changes, however, are perceived as a reflection of increased straining of untrained muscles in the initial phase of NMES treatment, similar to the reactions occurring in healthy people during traditional exercise.

In the conducted research big muscle groups of both lower limbs were simulated. In most cases NMES of the quadriceps and gastrocnemius muscles was conducted at the same time (Karavidas et al., 2006; Dobsak et al., 2006[a]; Karavidas et al., 2008; Soska et al., 2012; Quittan et al., 2001). Less often, simultaneous stimulation of the quadriceps and sciatic muscles (Nuhr et al., 2004; Quittan et al., 2001) or the quadriceps muscle alone was conducted (Vaquero et al., 1998; Quittan et al., 1999).

In all of the research conducted a current frequency of less than 50Hz was used, which is legitimate with regards to the rules of conducting endurance training, that is a form of activity aimed at training slow-twitched, aerobic muscle fibers of patients with CHF. Current frequencies of 4Hz (Banerjee et al., 2009), 10Hz (Maillefert et al., 1998; Dobsak et al., 2006[a]; Deley et al., 2008; Soska et al., 2012), 15Hz (Nuhr et al., 2004), 25Hz (Karavidas et al., 2006; Harris et al., 2003; Karavidas et al., 2008; Karavidas et al., 2010) and 50Hz (Quittan et al., 1999; Quittan et al., 2001) were used.

It is usually suggested to perform numerous repetitions of transient contractions during endurance training. Each contraction should be followed by a short break, lasting at least the same amount of time as the contraction or 2 to 3 times longer. The contraction time in patients with CHF was usually short and amounted to 2 seconds (Nuhr et al., 2004; Quittan et al., 1999; Quittan et al., 2001) or 5 to 6 seconds (Karavidas et al., 2006; Harris et al., 2003; Karavidas et al., 2008). Contractions of 10 seconds (Vaquero et al., 1998) or even 20 seconds (Dobsak et al., 2006[a]; Dobsak et al., 2006[b]) were rather seldom. The time of

the break between the contractions was usually equal to the time of contraction or 2 to 3 times longer at most (Nuhr et al., 2004; Quittan et al., 1999; Quittan et al., 2001). The procedures were conducted with varying frequency. They were performed 5 times a week (Karavidas et al., 2006; Deley et al., 2008; Karavidas et al., 2008; Karavidas et al., 2010; Banerjee et al., 2009; Quittan et al., 1999, Quittan et al., 2001), sometimes NMES was performed even 7 times a week (Nuhr et al., 2004; Dobsak et al., 2006[a]; Dobsak et al., 2006[b]; Soska et al., 2012). In one case the procedures were performed 2 times a day (Soska et al., 2012). Only in one case the training was less intensive – it was administered only 3 times a week (Vaquero et al., 1998).

Table 2. Research into the use of NMES in cardiological rehabilitation of patients with CHF.

Authors	Number |of subjects	Severity of CHF	Duration (in weeks)	Duration and frequency of sessions	NMES frequency (Hz)	Control group
Maillefert et al., 1998	14	NYHA II-IV	5	1 h; 5x/week	10	None
Vaquero et al., 1998	14	HTR	8	30 min; 3x/week	30 and 50	7 pharmacologically treated patients
Quittan et al., 1999	7	HTX	8	30 min for 2 weeks, then 60 min; 5x/week	50	None
Quittan et al., 2001	42	NYHA II-IV	8	30 min for 2 weeks, then 60 min; 5x/week	50	16 pharmacologically treated patients
Harris et al., 2003	46	NYHA II-III	6	30 min; 5/xweek	25	25 AT patients (70% HR_{max})
Nuhr et al., 2004	20	NYHA II-IV	10	4 h; 7x/week	15	10 patients - „sham" electrical stimulation
Dobsak et al., 2006[a]	15	NYHA III-IV, before HTX	6	1 h; 7x/week	10	None
Dobsak et al., 2006[b]	30	NYHA II-III	8	1 h; 7x/week	10	15 AT patients (40 min, 3x/week)
Deley et al., 2008	44	NYHA II-III	5	1 h; 5x/week	10	12 AT patients (1 h, 5x/week)
Karavidas et al., 2006	24	NYHA II-III	6	30 min; 5x/week	25	8 patients - „sham" electrical stimulation
Karavidas et al., 2008	30	NYHA II-III	6	30 min; 5x/week	25	10 patients „sham" electrical stimulation
Banerjee et al., 2009	10	NYHA II-III	8	1 h; 5x/week	4	Alternatively conducted NMES
Karavidas et al., 2010	31	18 patients NYHA II, 13	6	30 min; 5x/week	25	None
Soska et al., 2012	71	NYHA III-IV NYHA II-III	12	2x60 min; 7x/week	10	26 AT patients, 22 AT + NMES patients

HRT – patients after heart transplant; HTX – patients before heart transplant; HR – heart rate; AT – aerobic training

In two of the conducted meta-analyses, in which electrical stimulation of the lower limbs was compared to physical training with the use of cycloergometer or "supposed" electrical stimulation for certain physical capacity indicators in patients with CHF, it was demonstrated that under the action of NMES of the lower limbs in comparison to physical training a lesser increase in peak oxygen uptake (VO_{2peak}), distance traveled in the 6MWT Test and contraction strength of the thigh muscles is observed. By contrast, in comparison to the "supposed" electrical stimulation, NMES leads to a significant increase in value of these indicators of physical capacity. The results of both of the meta-analyses indicate, that NMES improves physical capacity in CHF patients, however not as significantly as classical physical training (Subruzzi et al., 2010; Smart et al., 2012).

The research conducted also suggest that a combination of classical cardiac rehabilitation (based on physical training) and neuromuscular electrical stimulation (NMES) of the skeletal muscles of the lower limbs does not provide substantial benefits to the improvement of physical capacity in patients with CHF in comparison with traditional rehabilitation (Soska et al., 2012). It may indicate that physical training is the most important element of cardiac rehabilitation. The results also suggest that NMES may be used as an alternative rehabilitation method in patients with severe heart failure (NYHA class IV) or in patients with CHF,

who cannot undergo physical training due to locomotory or central nervous system disorders.

To sum up the positive effects of NMES in patients with CHF, it can be assumed that NMES results in physical capacity changes similar to those occurring due to physical training. Although it needs to be accentuated, that working the muscles with the NMES method does not require a supervision as strict as rehabilitation through physical training and may even be performed at home. Patients tolerate the NMES therapy well and the devices needed to perform the stimulation are easily available and relatively inexpensive. During NMES procedures no health deterioration, skin impairment or pain were observed. Also, no negative side effects such as blood pressure or heart rate disorders were noted. NMES does not result in overstraining or damage of the muscle fibers.

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