Low-Frequency Electrical Stimulation Increases Muscle Strength and Improves Blood Supply in Patients With Chronic Heart Failure

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**Background** This study was designed to evaluate the effects of low-frequency electrical stimulation (LFES) on muscle strength and blood flow in patients with advanced chronic heart failure (CHF).

**Methods and Results** Patients with CHF (n=15; age 56.5±5.2 years; New York Heart Association III–IV; ejection fraction 18.7±3.3%) were examined before and after 6 weeks of LFES (10 Hz) of the quadriceps and calf muscles of both legs (1 h/day, 7 days/week). Dynamometry was performed weekly to determine maximal muscle strength (Fmax; N) and isokinetic peak torque (PTmax, Nm); blood flow velocity (BFV) was measured at baseline and after 6 weeks of LFES using pulsed-wave Doppler velocimetry of the right femoral artery. Six weeks of LFES significantly increased Fmax (from 224.5±96.8 N to 340.0±99.4 N; p<0.001), and also PTmax (from 94.5±41.5 Nm to 135.3±28.8 Nm; p<0.01). BFV in the femoral artery increased after 6 weeks from 35.7±15.4 cm/s to 48.2±18.1 cm/s (p<0.05); BFV values at rest before and after 6 weeks of LFES did not differ significantly.

**Conclusions** LFES may improve muscle strength and blood supply, and could be recommended for the treatment of patients with severe CHF. (Circ J 2006; 70: 75–82)

**Key Words:** Blood flow; Heart failure; Muscle; Stimulation

Chronic congestive heart failure (CHF) is a complex metabolic syndrome resulting from global hypoperfusion and neurohumoral activation. Sympathoadrenergic hyperactivity and stimulation of the renin–angiotensin–aldosterone cascade promote endothelial dysfunction in the macro- and microcirculation, and thus influence the distribution of the terminal blood flow. The increased total peripheral resistance, reduction of blood supply and impaired peripheral vascular dilatation in response to vasodilator stimuli result in atrophy of skeletal muscle and decreased oxidative activity. Physical training could reverse the pathologic changes in patients with CHF and there have been many reports during the past decade that clearly demonstrate the benefits of exercise on functional capacity, ventilation, metabolic status, autonomic control of heart rate (HR) variability and other parameters including skeletal muscle performance and impaired endothelial function. However, most of the actual training protocols are based on systemic exercise requiring increased cardiac output, which cannot be achieved by all patients, and in general are only suitable for patients with a moderately advanced grade of CHF; less attention has been paid to the development of safe and efficient training programs for patients with severe grades of the disease.

**Table 1 Patients’ Characteristics**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>M/F</td>
<td>14/1</td>
</tr>
<tr>
<td>Age (years)</td>
<td>56.5±5.2</td>
</tr>
<tr>
<td>CHF etiology (ischemic/non-ischemic)</td>
<td>10/5</td>
</tr>
<tr>
<td>Duration of severe CHF (months)</td>
<td>2±2.3</td>
</tr>
<tr>
<td>NYHA class (III/IV)</td>
<td>4/11</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>18.7±3.3</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.2±0.8</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>4.3±1.23</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>8.1±6.22</td>
</tr>
<tr>
<td>BMI</td>
<td>24.1±4.7</td>
</tr>
</tbody>
</table>

**Medication**

- ACEI
- Diuretics
- B-blockers
- Nitrates
- Digitalis

**CHF,** chronic heart failure; **NYHA,** New York Heart Association; **LVEF,** left ventricular ejection fraction; **CK,** creatine kinase; **LDH,** lactate dehydrogenase; **CRP,** C-reactive protein; **BMI,** body mass index; **ACEI,** angiotensin converting-enzyme inhibitor.
Recently, some studies have described new, alternative training methods that are of interest, such as graded muscle building\(^8,9\) or low-level exercise training\(^10\). The pioneer studies performed by Maillefert et al. and Vaquero et al., both in 1998, also suggested the possible benefits of low-frequency electrical stimulation (LFES) in patients with CHF\(^11\).\(^12\). The aim of our study was to evaluate the effects of LFES on muscle strength and blood supply in patients with severe grades of CHF.

**Methods**

**Patients**

We evaluated 15 patients with advanced grades of CHF (mean age 51.5±7.2 years; New York Heart Association (NYHA) class III–IV, left ventricular ejection fraction (LVEF) 18.7±3.3\%) who had been admitted for heart graft (Table 1).

**Inclusion Criteria**

Patients had to be symptomatically stable, NYHA class III–IV, LVEF determined by echocardiography, post coronary angiography and on optimized pharmacological treatment that had remained unchanged for 2 months before and throughout the study.

**Exclusion Criteria**

Patients with unstable angina pectoris, progressive ventricular dysrhythmia, intermittent claudication, implanted cardiac pacemakers, diabetes mellitus, and chronic bronchopulmonary disease were not include in the study.

All patients gave informed consent to participate in the study, which was approved by the local Ethics Committee and conforms to the principles outlined in the Declaration of Helsinki and to the GCP guidelines of the European Community.

**Protocol of LFES**

The muscles to be stimulated were the quadriceps and calf muscles of both legs. Self-adhesive surface electrodes 80×130 mm (PALS\(^9\) Platinum, Axelgaard Manufacturing Co, Lystrup, Denmark) were positioned on the thighs approximately 5 cm below the inguinal fold and 3 cm above the upper patella border; for the calf muscles the electrodes were positioned approximately 2 cm under the knee joint and just over the proximal end of the Achilles tendon (Fig 1).

Electrical stimulation was performed 60 min/day, 7 days/week for 6 consecutive weeks, using a dual-channel battery-powered stimulator Elpha-II 2000 (DANMETER\(^\text{®} A/S, Odense, Denmark). The stimulator delivered a biphasic current of 10Hz frequency. The current characteristics were set up as follows: “on” mode stimulus (20 s stimulation, 20 s rest), pulse width 200 ms, rise and fall time 1 s, and maximal stimulation amplitude 60 mA. The stimulation was performed while the patient was supine, at the same time of day (10.00–11.00 h) and under the supervision of medical staff. The first session of stimulation was started at lower amplitudes (≈30 mA) and gradually increased by 10–15 mA/day over the following 2–4 days until the final value of 60 mA was achieved, which was well tolerated by the patients.

**Muscle Strength Measurement**

To determine the maximal muscle strength, isometric dynamometry testing of the quadriceps muscles was performed once weekly using the PC 2 SDT dynamometric system (EXAMO\(^\text{®} Recens, Brno, Czech Republic) with a microprocessor. All measurements were performed while the subject was seated on the device; the chest was fixed by 2 straps, the pelvis and knees flexed at an angle of 90 degrees. The ankle of the tested leg was attached to the strength transducer (D.O.S-SBEAM 1000N, EXAMO\(^\text{®} Recens, Brno, Czech Republic) by a Velcro strip and the patient then carried out 3 consecutive maximal voluntary extensions (contraction time 3 s – resting time 7 s); the highest value was considered as the maximal strength (F\(_\text{max}\); N). The isokinetic muscle strength of the knee extensors was assessed by the same dynamometric system, recording the isokinetic strength as torque. Patients performed 3 consecutive knee extension movements with maximal effort and with an angular speed of 90 degrees/s with both legs; the highest value obtained was regarded as the peak torque (PT\(_\text{max}\); Nm).

**Bood Flow Velocimetry Measurement**

To evaluate the changes in peripheral perfusion, a standard pulsed-wave Doppler assessment of the right femoral artery was performed before and after the end of the 5 weeks of stimulation using an 8-MHz Doppler probe and echograph (Sonos 2000, Hewlett Packard, Andover, OH, USA). The measurements were performed before each session after 15 min rest while supine, and at 15, 30, 45 and 60 min of stimulation (during the “off” 20 s period). The mean value of the blood flow was calculated from 5 single measurements of the peak flow velocity (cm/s).

**Standard Hemodynamic Parameters Registration**

The values of systolic (SBP) and diastolic (DBP) blood pressure were monitored using a manual pressure manome-
Quality of Life (QOL)

The major problem in assessing the QOL of the patients included in this study was that most of them were NYHA IV. Moreover, all patients were hospitalized during the course of the study and were substantially limited in many of everyday life activities. Therefore, using a standard procedure (eg, detailed questionnaire such as the Minnesota QOL) was unsuitable because many activities could not be performed by the patients. Thus, a modified questionnaire for subjective assessment of habitual activities of daily life (ADL) and NYHA classification was used in this study as an indirect index of QOL. For evaluation of the questionnaire, the Borg scale for assessment of fatigue (ratio of perceived exertion (RPE)), ranging from very light to very strenuous (6–20 points) and dyspnea, ranging from none to maximal (0–10 points) was used. The assessment was done for several habitual ADL related to leg muscles strength (eg, dressing, walking, showering and climbing stairs up to the 1st floor without stopping). In order to evaluate the possible influence of the stimulation on homeostasis, several selected biochemical parameters (cholesterol, glycemia, C-reactive protein (CRP), creatine kinase (CK) and lactate dehydrogenase (LDH)) were monitored during the study (Table 1).

Statistics

All data are presented as mean±SD. Statistical analysis was performed using the McNemar test of symmetry and Wilcoxon paired test. A p-value <0.05 was considered significant.

Results

Muscle Strength Evaluation

The measurements of maximal muscle strength (Fmax) assessed by isometric dynamometry already showed a significant increase after 4 and 5 weeks of stimulation (initial value 224.5±96.8 N vs 315.0±91.7 N in the 4th week; and vs 314.2±82.0 N in the 5th week; p<0.01 for both). After 6 weeks of stimulation the final Fmax achieved was 340.0±99.4 N (p<0.001 vs initial value), an improvement of 51% (Fig 2).

Isokinetic Peak Torque (PTmax) Evaluation

The PTmax showed a significant increase after 5 (130.0±30.6 Nm; p<0.05) and 6 weeks (135.3±28.8 Nm; p<0.01) of LFES compared with the initial PTmax value (94.5±41.5 Nm). After 6 weeks of stimulation PTmax was improved by 43% (Fig 3).

Blood Flow Velocimetry Measurement

The Doppler blood flow velocimetry measurements showed significant increases after 6 weeks of LFES compared with the initial value (35.6±3.9 cm/s before LFES vs 48.2±4.1 cm/s after LFES; p<0.05 (Fig 4). In all the subjects the maximal rise in blood flow velocity was achieved after 15 min of stimulation; there were no significant differences between the values recorded after 15, 30, 45 and...
60 min of stimulation (Fig 5). Blood flow values at rest before and after 6 weeks of stimulation (11.9±5.1 cm/s before vs 13.0±4.1 cm/s after LFES; NS), and also the values recorded 15 min after the end of each LFES application did not differ statistically (initial value 13.6±3.3 cm/s vs 14.9±3.1 cm/s after 6 weeks of LFES; NS). Initial value of the inner diameter of femoral artery was 2.32 cm (±0.19) at the beginning of the study and 2.56 cm (±0.25) after 6 weeks of LFES (NS) (Figs 4,5).

QOL and Biochemical Parameters

After 6 weeks of LFES, all patients had less dyspnea and a marked decrease in subjective feelings of fatigue during everyday activities, although this change was not statistically significant. Similar results were observed for NYHA classification: 4 patients moved from NYHA IV to NYHA III after 6 weeks of stimulation and the remainder were unchanged but not (eg, from NYHA III to NYHA IV). In order to evaluate the possible risk of damage to muscle fiber by stimulation, the activity of both CK and LDH was monitored at the beginning, after 1 week and after 6 weeks of stimulation and only insignificant increases after 1 week of stimulation was observed and after 6 weeks of stimulation, the serum levels of both enzymes returned to baseline. Insignificant differences were found when the initial and final values (after 1 and 6 weeks of stimulation) for the serum levels of CRP, glucose and cholesterol were compared. The results of the evaluation of RPE, NYHA, body mass index and selected biochemical parameters are summarized in Table 2.

Hemodynamic Parameters

LFES did not cause any significant changes of SBP and DBP, nor were there significant changes in the recorded values of HR (Table 3).

Discussion

Numerous studies have focused on the effects of LFES of mammalian skeletal muscles. During physical activity the motor units are activated hierarchically, but the muscle activity induced by electrical stimulation is not natural because it bypasses the physiological recruitment and activates all motor units simultaneously. In other words, electrical stimulation can attain a more intensive work level than any type of exercise. Chronic LFES-induced changes in neural regulation and neural motor unit recruitment

Table 2 Values of Selected Biochemical Parameters, NYHA Classification and RPE (Borg) at Initial and Final Examination

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>After 1 week of LFES</th>
<th>After 6 weeks of LFES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.27±0.8</td>
<td>5.38±0.51</td>
<td>5.48±0.44</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>4.39±1.23</td>
<td>4.78±0.97</td>
<td>4.12±1.24</td>
</tr>
<tr>
<td>CK ( kata/L)</td>
<td>1.15±0.44</td>
<td>1.42±0.52</td>
<td>1.29±0.79</td>
</tr>
<tr>
<td>LDH ( kata/L)</td>
<td>5.73±1.19</td>
<td>6.07±2.12</td>
<td>5.76±1.62</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>8.11±6.22</td>
<td>8.05±6.62</td>
<td>7.69±5.82</td>
</tr>
<tr>
<td>NYHA class (III/IV)</td>
<td>4/11</td>
<td>–</td>
<td>8/7</td>
</tr>
<tr>
<td>BMI</td>
<td>24.1±4.7</td>
<td>–</td>
<td>24.7±3.2</td>
</tr>
<tr>
<td>RPE (borg scale)</td>
<td>4.8±3.3</td>
<td>–</td>
<td>3.6±3.2</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>12.3±3.6</td>
<td>–</td>
<td>11.0±3.8</td>
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</tbody>
</table>

RPE, ratio of perceived exertion; LFES, low-frequency electrical stimulation. See Table 1 for other abbreviations.

Table 3 Mean (±SD) Values of Hemodynamic Parameters Obtained at Baseline and After 6 Weeks of LFES (at Rest, After 30 and 60 min of Stimulation)

<table>
<thead>
<tr>
<th></th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>HR (beats/min)</th>
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</thead>
<tbody>
<tr>
<td>At baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest</td>
<td>104.6±11.4</td>
<td>68.8±11.2</td>
<td>74.3±7.3</td>
</tr>
<tr>
<td>30 min</td>
<td>107.5±13.9</td>
<td>71.3±10.0</td>
<td>75.8±7.4</td>
</tr>
<tr>
<td>60 min</td>
<td>105.8±19.9</td>
<td>68.8±10.8</td>
<td>72.3±9.6</td>
</tr>
<tr>
<td>After 6 weeks of LFES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest</td>
<td>107.1±11.1</td>
<td>71.7±10.7</td>
<td>75.5±5.0</td>
</tr>
<tr>
<td>30 min</td>
<td>105.8±8.4</td>
<td>72.1±9.9</td>
<td>71.2±3.8</td>
</tr>
<tr>
<td>60 min</td>
<td>106.8±9.1</td>
<td>69.6±7.5</td>
<td>69.5±5.7</td>
</tr>
</tbody>
</table>

LFES, low-frequency electrical stimulation; SBP, values of systolic; DBP, values of diastolic; HR, heart rate.
Skeletal Muscle Stimulation in CHF

increases the number of fatigue resistant slow (oxidative) fibers, and the concomitant enhancement of neoangiogenesis and capillary density undoubtedly contributes to the increased exercise capacity and nutritive flow to the skeletal muscle mass.\textsuperscript{3,14} The intense structural and metabolic changes induced by LFES in mammalian skeletal muscle fibers are both qualitative and quantitative. The physiognomy of mammalian skeletal muscles is based upon the composition of fiber types and their aptitude to adjust their molecular and metabolic patterns to different stimuli. Different fiber types result from the presence of many regulatory and contractile protein isoforms and their type-specific organization. Based on many previously published reports, muscle fibers should be considered as dynamic entities eligible for phenotype transitions by changes in the isoform composition.\textsuperscript{15} Among many factors influencing these changes, the most important are neuromuscular activity and mechanic loading. From a general point of view, long-term permanently increased neuromuscular activity and/or mechanic load promotes the “fast-to-slow” fiber type transition and increases the number of fatigue resistant slow-type fibers; the inhibition or reduction of neuromuscular activity and/or mechanic load has an inverse effect.\textsuperscript{16} Electrical stimulation has made a fundamental contribution to the understanding of motor neuron activity on muscle fiber phenotypes. Experimental but also clinical investigations using chronic LFES have shown progressive structural, metabolic and biochemical changes in fast glycolytic muscle fibers. These effects are mediated by translational and transcriptional mechanisms, and lead to complete fast-to-slow myosin chain transformation and marked increase of oxidative enzymatic activity and decrease of glycolytic activity.\textsuperscript{17} Moreover, an increased volume density of mitochondria after LFES has been observed.\textsuperscript{18} The muscular structural changes are accompanied by increased capillary-to-fiber ratio, total capillary surface area and arteriolar density. Thus, the enlarged capacity of the microvascular bed increases the blood flow in the stimulated muscles.\textsuperscript{19–22}

There are many reports of the successful clinical application of LFES in neurological diseases,\textsuperscript{23,24} and for prevention of muscle atrophy after surgery.\textsuperscript{25,26} Other important clinical applications of LFES are in cardiomyoplasty\textsuperscript{27} and incontinence management.\textsuperscript{28} As an alternative to classical exercise training, electrical stimulation initiating contraction of large muscle groups could improve the status also in patients with CHF by increasing muscle strength and improving QOL. It is likely that beneficial changes in muscle performance and exercise capacity in CHF patients after LFES would be similar to those observed following exercise training; however, experience with LFES application in such patients is limited and the aim of the present study was to evaluate if LFES could improve the clinical status of patients with advanced CHF who are unable to undertake other types of physical training. Because of the severity of the disease (11 of the 15 patients were classified as NYHA class IV), the number of convenient and safe clinical tests to assess functional changes before and after 6 weeks of stimulation was limited and dynamometry of the lower limbs was finally selected as a reliable tool for regular follow-up of the gradual changes in muscle strength during the study period. Muscle strength, assessed by isometric and isokinetic dynamometry, increased significantly in all subjects, which concords completely with similar clinical reports, highlighting the positive effects of chronic LFES against skeletal muscle atrophy and in enhancing muscle power in these patients.\textsuperscript{29–31} In the present study, the increase in maximal muscle strength was achieved with a frequency of 10 Hz, although Quittan et al\textsuperscript{30,31} reported an increase in muscle strength using a frequency of 50 Hz. The stimulation protocol in our study was rather intense (60 min/day), and was targeted to relatively large muscle masses in both legs (both the quadriceps and calf muscles), which could explain the significant increase in muscle strength (even with lower frequency) although this issue remains unresolved because the optimal frequency for the stimulation protocols has not been determined yet. Endothelium-dependent vasodilatation of the peripheral vasculature is impaired in patients with CHF,\textsuperscript{32} as a consequence of defective nitric oxide synthase (eNOS) activity.\textsuperscript{33} Evidence is accumulating that regular exercise has beneficial effects on vascular reactivity, and that these salutary changes result from exercise-induced increases in blood flow.\textsuperscript{24,35} Long-term changes in blood flow exert their effects on endothelium-dependent vasodilatation by modulating the expression of eNOS. The changes in endothelial reactivity and eNOS expression seen with sustained increases in blood flow are similar to changes in endothelial behavior in vessels exposed for long periods to intermittent increases in flow (as with exercise). Flow-mediated vasodilatation is largely caused by the release of endothelium-derived relaxing factor,\textsuperscript{36,37} but chronic increase in blood flow may also stimulate the production of prostacyclin. This may be particularly important in the circulation (such as the skeletal muscle microvasculature) where this prostananoid plays a larger role in flow-mediated vasodilatation.\textsuperscript{38–40} Excepting NO and prostacyclin production, increased flow-induced shear stress influences directly the expression of many other bioactive substances, such as vascular endothelial growth factor, endothelium-derived hyperpolarizing factor, endothelial progenitor cells, adhesion molecules and chemokines, all of which may participate in the beneficial effects of exercise-induced vascular remodeling and reactivity.\textsuperscript{41,42} Responding to chronic changes of blood flow the vessel wall is capable of significant adjustment and remodeling. Permanent decrease in the arterial flow diminishes the vessel’s caliber in response to structural modifications of the arterial wall, and this vascular remodeling is prevented by removal of the endothelium. In contrast, increased arterial blood flow (and shear stress) causes structural changes that increase the vessel’s diameter,\textsuperscript{43} which suggests that the endothelium may be the transducer of the hemodynamic signal triggering vascular remodeling in response to changes in blood flow. However, the endothelial mechanisms mediating these structural effects remain undefined. In a randomized study Maiorana et al showed that combined aerobic and resistance exercise in healthy men did not significantly affect endothelium-dependent or -independent function\textsuperscript{45} which is in contrast to similar studies done in patients with CHF in whom the NO-related endothelial function was strongly affected.\textsuperscript{46–48} It seems that the essential pathophysiological mechanism proposed to explain lower NO activity in such patients is loss of endothelial synthesis of NO because of abnormal enzymatic activity; these conditions may all be associated with increased oxygen free radicals that alter eNOS activity and with which NO reacts.\textsuperscript{49} Repeated exercise and shear stress stimulation of NO bioactivity redresses this radical imbalance, hence leading to greater potential for bioavailability of vasoactive substances.\textsuperscript{50} Thus, the pathological states associated with impaired endothelial function and
depressed NO production, as in chronically depressed heart pumping may respond, more rapidly to training, but return rapidly to baseline after cessation of training.51,52 Therefore, it is probable that depressed endothelial function is more susceptible to improvement by moderate exercise training of localized muscle groups or whole body exercise, than the well preserved endothelium in healthy subjects. However, the involved mechanisms remain unclear and probably depend upon a specific, as yet undefined etiology.53 In summary, it is likely that short-term training increases NO bioavailability, which regulates the shear stress associated with exercise. If the exercise is maintained for a long time, NO-dependent structural changes follow, leading to arterial remodeling and structural normalization of shear. It is suggested that the improved NO-mediated vascular function following long-term exercise training may improve cardiac function in CHF subjects, possibly by improving vascular compliance and decreasing afterload. In humans, more evidence is required regarding the time course of changes in resistance, conduit and microvessels, and concerning the functional mechanisms aside from those that are NO dependent. The contractions initiated by local electrical stimulation of the strength muscle may cause similar (or identical) vascular reactions as seen during physical exercise, namely the exercise-induced reactive hyperemia in working muscles. Thus, the previously mentioned beneficial effects of LFES on vascular function are most probably related to the effect of increased pulsatile flow on the vessel’s endothelial layer.54,55 It is likely that the LFES-induced changes in blood flow by long-term electrical stimulation are related to modification of endothelial function, and thus may be mostly NO-dependent, but as mentioned earlier, additional mechanisms cannot be excluded, especially the possible growth enhancement of new vessel collaterals, as occurs after physical exercise training.56 The contribution of LFES on vascular remodelling observed in this study may be seen in the insignificant increase of the inner diameter of the right femoral artery at the end of the 6th week of stimulation. The significant increase in blood velocity in the femoral artery during stimulation may reflect the importance of the global vascular benefit for the peripheral muscle mass after 6 weeks of LFES. This finding may also be considered as a sign of improved adaptation of the local muscle vasculature to the exercise workload. During the stimulation we did not observe any life-threatening side effects of LFES on BP or HR and there were neither signs of muscle damage nor complaints of discomfort or pain caused by the stimulation. Slight, insignificant increases in the CK and LDH levels after 1 week of stimulation can be explained as a reflection of increased muscle load during the introductory phase of stimulation, which resembles increased muscle activity in healthy people, such as during exercise. This result also suggests that stimulation does not cause any adverse overload or damage of muscle fibers. Although neither RPE evaluation nor NYHA classification reached statistical significance, the results suggest a tendency of improvement of particular parameters. Thus, we can speculate about the total positive impact of LFES not only on functional capacity (fitness), but also on improved QOL during prolonged hospitalization. However, even if the presented results are very encouraging, we are aware that our observations are based on only a limited number of measurements. So far, only 1 preliminary study has reported an improvement of the blood supply in patients with CHF after 5 weeks of LFES.57 Therefore, any definite conclusion on this matter would be premature, and further investigations on the vascular reactivity to LFES, including a larger number of patients, are required. The present study is 1 of a few clinical reports that have focused on the therapeutic potential of LFES in CHF. We wanted to examine the utility of this method as an auxiliary tool for counterbalancing the decreased physical ability and improving the blood supply in patients with CHF waiting for heart transplantation. Recently, results from the first randomized trial comparing home-based LFES and classical exercise training have demonstrated that both methods could significantly influence functional capacity, muscle strength and QOL of patients with CHF.58 Similar results were shown by Nuhr et al in a group of patients with advanced CHF, and an increase of slow myosin heavy chain isoforms at the expense of the fast ones and increased intensity of oxidative enzymatic activity were also found after LFES.59 The safety of LFES to strengthen muscles in CHF patients with implanted cardiac pacemakers was highlighted by several Austrian authors.60-62 The most important conclusion arising from these reports is that LFES should be considered as an alternative to classical exercise training, one that can be easily performed at home. Although the effectiveness of conventional exercise protocols in cardiovascular rehabilitation is beyond doubt, the safety and easy application of LFES could be of great benefit in the rehabilitation of patients with CHF, especially those with a severe grade of the disease. Future investigations should focus on a much wider application of LFES in cardiovascular rehabilitation, perhaps also in combination with some types of classical exercise training.

Conclusion

LFES can be considered as a safe and well-tolerated method of physical training, without any life-threatening side effects. LFES of the lower limbs may improve muscle strength and probably also blood supply in long-term stimulated muscles. A rehabilitation training program based on this method could be an alternative to conventional exercise training for improving the QOL of patients with CHF.

Acknowledgments

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