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Comparison of different intensity modes of neuromuscular electrical stimulation in the rehabilitation of elderly patients with decompensated chronic heart failure

Aim	To compare effects of neuromuscular electrostimulation (NMES) with various intensity of induced muscle contractions on its tolerance and effect on physical work ability in elderly patients admitted for chronic heart failure (CHF).
Material and methods	The study included 22 patients older than 60 years admitted for decompensated CHF. NMES was performed from the 2nd or 3d day of stay in the hospital to the discharge from the hospital. Patients choose the stimulation regimen themselves based on the result of the first session: the high intensity to achieve maximum tolerable muscle contractions (group 1) or the lower intensity to achieve visible/palpable muscle contractions (group 2). Prior to the onset and after the completion of the training, the 6-min walk test (6MWT) was performed and the general condition of the patient was assessed with a visual analogue scale (VAS).
Results	More patients, mostly women, chose the less intensive NMES (14 vs. 8). The groups did not differ in age, comorbidity, and functional condition. Both groups achieved considerable increases in the 6MWT distance (7.3 [5.6; 176] and 9.8 [7.0; 9.9] %, respectively, p>0.05) and VAS scores without a significant difference between the groups. Among the patients who were compliant with continuing NMES after the discharge from the hospital, 69% were patients of the group of the less intensive stimulation.
Conclusion	The less intensive NMES (with achieving visible muscle contractions) was characterized by better tolerance and better compliance in elderly patients with decompensated CHF compared to the more intensive NMES (with achieving maximum contractions), but the less intensive NMES was not inferior to the more intensive NMES in effectiveness.

Keywords	Neuromuscular electrostimulation; chronic heart failure; cardiac rehabilitation
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Introduction

Chronic heart failure (CHF) has both a poor prognosis and high prevalence. The increasing number of patients with CHF is primarily due to the aging population, with its prevalence reaching 10% or more among people older than 70 years [1].

CHF is accompanied by reduced tolerance to exercise caused not only by a decrease in cardiac output but also by structural and functional changes in skeletal muscles [2–5]. Graduated exercise, which helps to improve physical performance, quality of life, and even prognosis, is the main recommended rehabilitation method [4]. However, exercises have some limitations, including lack of conditions, extremely low-stress tolerance, and unwillingness, particularly in more severe, older patients and those out of training. In addition, exercise is contraindicated in acute heart failure and decompensated CHF. Thus, neuromuscular electrical stimulation (NMES) is being studied as an alternative, additional, or initial method of cardiac rehabilitation, which requires no efforts from patients, bears no significant hemodynamic load, but is relatively similar to physical exercises in terms of effects on muscle strength and exercise tolerance according to several authors. However, there is no universal approach to NMES. The applied techniques vary in duration, muscle involvement, characteristics of electrodes and stimulation pulses, as well as muscle contraction intensity [4, 6, 7].

Aim

Compare NMES of different intensities of stimulated muscle contractions in terms of tolerance its and effects

on physical performance in elderly patients hospitalized with CHF.

Material and methods

The study included 22 (11 male and 11 female) patients hospitalized in the University Clinical Hospital No. 1 of I. M. Sechenov First Moscow State University. Inclusion criteria were decompensated CHF and age over 60 years. Exclusion criteria were acute infectious or inflammatory diseases, documented venous thrombosis, clinically significant obliterating atherosclerosis of lower limb vessels and severe leg swelling that prevented effective myostimulation. The study met the ethical principles of the Declaration of Helsinki of the World Medical Association. All patients signed the informed consent to participate in the study.

Starting from days 2–3 in the hospital and until discharge, NMES was conducted in addition to the best possible drug therapies using a STIMULUS-01 LF device developed in the Russian State Scientific Center in the Institute of Biomedical Problems of the Russian Academy of Medical Sciences. This device generated symmetrical bipolar rectangular electrical pulses (1±0.05, 25±1 Hz, cyclic mode; 1±0.1 s stimulation, 2±0.1 s pause). The muscles of the front and back of both thighs and lower legs were stimulated simultaneously. The NMES technique has been described in detail in a previous article [8].

Prior to the beginning of NMES, the acting mechanisms, objectives of the examination, usefulness of achieving significant muscle contractions, as well as the gradual increase of the stimulation amplitude to achieve the effect, were explained to patients, who were instructed to inform the physician about any discomfort and possible alterations of health. If patients had no more questions, they signed the informed consent. They also were told that they might discontinue the training at any time.

In the first training session, the amplitude of NMES was selected individually, taking into account strength of muscle response and pain threshold. On patient request, the amplitude increase was discontinued. After additionally motivating the patients and eliminating the interfering factors, an attempt was made to gradually increase the amplitude until the maximum tolerated, painless, effective muscle contraction, which was palpable and visible. If necessary, the intensity was corrected in general or by individual muscle groups. In the following training sessions, the selected amplitude of contraction was used as the basis. Once the patient had adapted, amplitude could be further increased to maintain the highest possible level of muscle contraction. Additional increases in amplitude were possible depending on the patient's capabilities; alternatively, it could be reduced to previous values at the first request. After the first session, the patient was asked to choose whether the amplitude of contractions would be increased in the following sessions according to the described pattern to the maximum tolerated level (Group 1) or would remain at the level of palpable and visible contractions without a further increase (Group 2).

The first training session consisted of a trial and lasted 30 minutes. Blood pressure (BP) and heart rate (HR) were measured prior to commencing stimulation and 10 to 15 minutes following the beginning of the session. At this point, the patient's condition was monitored and the intensity of contractions corrected if any discomfort was reported. The training sessions took place 5 times a week with gradually increasing duration to 90 minutes (n=2). The other group of patients refused to participated in increased session durations of more than 1 hour.

Following the end of a training session, the tolerance and efficacy were evaluated by changes in 6-minute walk distance (6MWD) and subjective assessment of the patient's wellbeing using a visual analog scale (VAS) from 0 (best) to 10 (worst).

The statistical analysis of the obtained data was performed using the SPSS Statistics version 23.0 software suite. The data are represented as mean and standard error (M±m) and median. The Mann–Whitney U-test was used to analyze differences in the independent samples. Changes of the variables were estimated using the Wilcox rank test. The differences were statistically significant at $p<0.05$.

Results

Eight patients chose a more intensive training routine (Group 1), while fourteen chose a less intensive training routine (Group 2). The main characteristics of the two groups are presented in Table 1. Group 2 consisted mainly of women and individuals with a higher body mass index; however, patients of the two groups did not differ significantly by these and other characteristics. Three patients in Group 1 and five patients in Group 2 refused to perform 6MWD due to inability to walk a distance because of severe symptoms of CHF, concomitant musculoskeletal and nervous system pathologies, asthma and deconditioning.

Most patients had 6 to 9 training sessions; the groups did not differ by this indicator and the mean total duration of NMES. Although the achieved stimulation amplitude of all muscle groups was predictably higher in Group 1, the differences were statistically insignificant (Table 2).

One patient in Group 1 refused to continue NMES after the first session due to muscle pain the day after the session. It should be noted that this patient had increased the stimulation intensity to the highest levels during the procedure; this was most likely due to a significant decrease

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Table 1. Clinical characteristics of CHF patients

Parameter	Maximum tolerable NMES (n=8)	Visible contraction of skeletal muscles in NMES (n=14)	p
Sex			
Male	6 (75%)	5 (35.7%)	0.164
Female	2 (25%)	9 (64.3%)	
Age, years	77.2 [67; 86]	77.8 [68.5; 78.5]	0.289
Weight, kg	80.0 [63.5; 102.5]	83.0 [76.5; 92.5]	0.234
BMI, kg/m^2	28.0 [26.8; 31.7]	33.95 [22.5; 36.1]	0.172
AH	7 (87.5%)	12 (85.7%)	0.12
CAD	5 (62.5%)	10 (71.43%)	0.158
AF	3 (37.5%)	4 (28.57%)	0.13
NYHA FC III	6 (75%)	11 (78.57%)	0.1
NYHA FC IV	2 (25%)	2 (14.28%)	0.15
LVEF, %	32.3±3.5	30.8±6.1	0.1
Loop diuretics	8 (100%)	14 (100%)	0.16
ACE inhibitors/ ARBs	7 (87.5%)	13 (92.86%)	0.11
Beta-blockers	7 (87.5%)	13 (92.86%)	0.13
MRA	7 (87.5%)	11 (78.57%)	0.2
Statins	7 (87.5%)	11 (78.57%)	0.159
Systolic BP, median, mmHg	128.6 [114.4; 135.7]	140.0 [137.2; 141.7]	0.13
Diastolic BP, median, mmHg	81.8 [70.1; 86.8]	87.8 [86.8; 87.9]	0.18
HR, median, bpm	69.5 [65.5; 73.4]	65.1 [64.3; 65.1]	0.11
No 6MWD	3 (37.5%)	5 (35.7%)	0.27
6MWD before training, m	345 [305; 435]	410 [380; 440]	0.1
General welbeing before training, score	5.0 [4; 5.5]	7.0 [6.5; 7.0]	0.1

The data are presented in tables as the median and interquartile range and the absolute number (5) or the mean ± standard error.
CHF – chronic heart failure; NMES – neuromuscular electrical stimulation; BMI – body mass index; AH – hypertension; CAD – coronary artery disease; AF – atrial fibrillation; FC – functional class; NYHA – New York Heart Association; LVEF – left ventricular ejection fraction; ACE – angiotensin-converting enzyme; ARB – angiotensin II receptor blocker; MRA – mineralocorticoid receptor antagonist; BP – blood pressure; HR – heart rate; 6MWD – 6-minute walk distance; VAS – visual analog scale.

in pain sensitivity due to neuropathy. Two patients aged 68 and 85 years from Group 2 also refused to continue due to muscle discomfort, even with minimal stimulation amplitude.

16 (72.7%) patients agreed to continue the stimulation training after the discharge. Two more patients in Group 1 and one patient in Group 2 did not continue NMES. Thus, the compliance with NMES was 62.5 and 78.5% (p>0.05) in the higher-intensity and lower-intensity groups, respectively.

The BP and HR response to NMES did not differ in the two groups (Figure 1).

Patients in both groups significantly improved their functional performance and experienced better general wellbeing with longer 6MWD. Patients of the two groups did not differ significantly in the increase in distance traveled and the VAS score (Table 3). All bed-bound patients who did not perform 6MWD at admission were mobile, at least within the ward.

Discussion

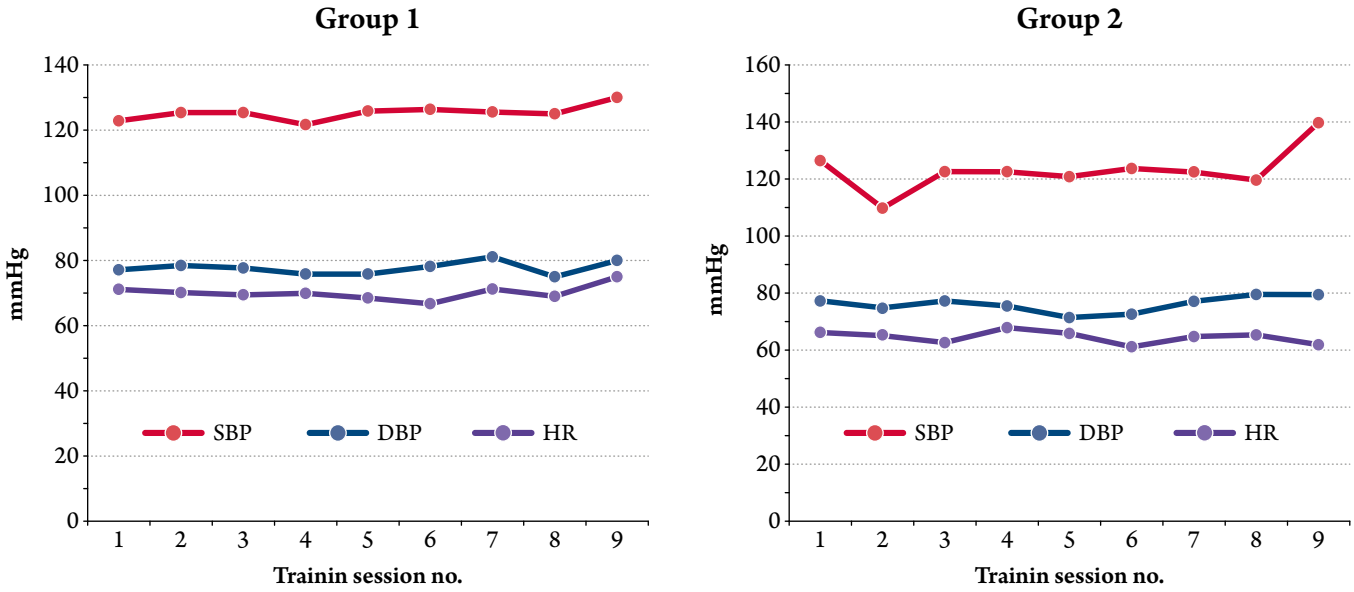
The decreased physical performance in patients with CHF is associated not only with cardiovascular reserves but also

Table 2. Characteristics of NMES training courses

Parameter	Maximum tolerable NMES (n=8)	Visible contraction of skeletal muscles in NMES (n=14)	p
Number of NMES training sessions	7.0 [6.5; 7.5]	9.0 [8.0; 9.5]	0.1
Total duration of NMES, min	480 [413; 560]	540 [525; 560]	0.13
Refused NMES	1 (12.5%)	2 (14.3%)	0.1
Would refuse if NMES could be continued	2 (24.5%)	1 (7.7%)	0.1
Compliance with NMES	5 (62.5%)	11 (78.6%)	0.14
Mean stimulation amplitudes by muscle groups, V			
front left thigh	20.0 [17.4; 22.9]	22.7 [21.1; 23.4]	0.1
back left thigh	20.0 [15.6; 22.6]	19.6 [18.5; 21.1]	0.167
front left leg	23.8 [16.5; 24.9]	18.9 [21.0; 24.9]	0.1
back left leg	23.0 [15.0; 23.7]	19.8 [19.8; 21.3]	0.23
front right thigh	20.0 [15.2; 22.3]	19.7 [19.7; 21.1]	0.14
back right thigh	21.0 [15.0; 22.4]	22.0 [20.6; 22.2]	0.178
front right leg	23.0 [16.5; 23.7]	19.9 [19.3; 21.6]	0.13
back right leg	22.2 [16.2; 23.6]	19.7 [18.4; 21.6]	0.195

NMES – neuromuscular electrical stimulation.

Figure 1. Assessment of the cardiovascular system response after each training session in the group of maximal tolerable neuromuscular electrical stimulation and the group of visible skeletal muscle contractions



SBP – systolic blood pressure; DBP – diastolic blood pressure; HR – heart rate.
The mean values are given for all variables. The numbers of the training session are presented on the horizontal axis, and the blood pressure measurements are shown on the vertical axis. The differences betwee n successive measurements and between the groups are statistically insignificant.

Table 3. Changes in functional performance at end of treatment

Parameter	6MWD before the cessation of training, m	Δ6MWD before the cessation of training, % of the baseline	VAS before the cessation of training, m	ΔVAS before the cessation of training, % of the baseline
Maximum tolerable NMES (n=8)	380.0 [340.0; 452.5]	+ 7.3 [5.6; 17.6]	7 [5.5; 8.5]	50.0 [40.0; 7.5]
Visible contraction of skeletal muscles in NMES (n=14)	450 [417.5; 470.0]	+ 9.8 [7; 9.9]	8.0 [8.0; 8.5]	28.6 [21.45; 30.97]
p	0.13	0.125	0.1	0.15

NMES – neuromuscular electrical stimulation; 6MWD – 6-minute walk distance; VAS – visual analog scale.

with the functional capabilities of skeletal muscles. Disturbed perfusion, the effects of neurohumoral factors and inflammatory cytokines in the skeletal muscles contribute to the development of myopathy characterized by the transformation of myosin fibers, enlargement of apoptosis nuclei, deformation and swelling of myofibrils, decreased enzyme activity, a shift of pH, reduction of effective mitochondria, capillaries and reduced total muscle mass. As a result, muscle strength and tolerance decrease, while deconditioning progresses over time from an inability to perform usual exercises to limitations in self-care. Muscle dysfunction contributes to higher sympathetic tone and thus worsens the course of CHF [9]. Most patients with CHF are old/senile, who experience age-associated sarcopenia, a degenerative loss of muscle, deterioration of the muscle fiber quality and reduced strength of muscle contraction, which contributes significantly to the development of myopathy. For decompensated CHF, hospitalized patients are under the most unfavorable conditions, rapidly losing muscle mass due to lack of training. The result is a dramatic decrease in functional performance despite successful treatment of the underlying disease [10, 11]. Thus, in addition to standard drug therapy, it is necessary to include patients with CHF in cardiac rehabilitation programs. Patients with a combination of age-associated sarcopenia and cardiac pathology are usually deconditioned and unable to perform even minimal exercises indicated as a part of secondary prevention. For such patients, NMES is a valid option.

This method was first used in sports medicine, neurology and traumatology, where high-frequency NMES proved highly effective [12]. In CHF, given better tolerance and higher

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ДОСТИЖЕНИЕ ЦЕЛЕВОГО УРОВНЯ ТРИГЛИЦЕРИДОВ СНИЖАЕТ СС РИСКИ НА 31%[1]

ФЕНОФИБРАТ (ТРАЙКОР®)[1], %

ОХ	ЛНП	ТГ	ЛВП
↓25	↓35	↓50	↑30

ТРАЙКОР® НАИБОЛЕЕ ЭФФЕКТИВНО СНИЖАЕТ ТГ[1, 2]

Трайкор®, 145 мг. Международное непатентованное наименование: фенофибрат. **Регистрационный номер:** ЛСР-002450/08. **Лекарственная форма:** таблетки, покрытые пленочной оболочкой, 145 мг. **Фармакодинамика:** В ходе клинических исследований было отмечено, что применение фенофибрата снижает концентрацию общего холестерина на 20-25 % и триглицеридов на 40-55 % и повышает концентрацию ЛПВП-холестерина на 10-30 %. Учитывая влияние фенофибрата на концентрацию ЛПНП-холестерина и триглицеридов, применение препарата эффективно у пациентов с гиперхолестеринемией, как сопровождающейся, так и не сопровождающейся гипертриглицеридемией, включая вторичную гиперлипопротеинемию, например, при сахарном диабете 2-го типа. Терапия фенофибратом также привела к уменьшению потребности в лазерном лечении диабетической ретинопатии (3,6 % по сравнению с 5,2 %, P=0,0003) в исследовании FIELD. **Показания к применению:** гиперхолестеринемия и гипертриглицеридемия изолированная или смешанная (дислипидемия тип **II**a, **II**b, **III**, **IV**, V по классификации Фредриксона) у пациентов, для которых диета или другие немедикаментозные лечебные мероприятия (например, снижение массы тела или увеличение физической активности) оказались неэффективными, особенно при наличии связанных с дислипидемией факторов риска, таких как артериальная гипертензия и курение. Для лечения вторичной гиперлипопротеинемии препарат применяется в тех случаях, когда гиперлипопротеинемия сохраняется, несмотря на эффективное лечение основного заболевания (например, дислипидемия при сахарном диабете). **Противопоказания:** повышенная чувствительность к фенофибрату или другим компонентам лекарственного средства; тяжелые нарушения функции печени - класс С по шкале Чайлд-Пью (включая билиарный цирроз и персистирующее нарушение функции печени неясной этиологии); тяжелое и умеренное нарушение функции почек (клиренс креатинина ниже 60 мл/мин для данной дозировки препарата); возраст до 18 лет (эффективность и безопасность не установлены); наличие в анамнезе фотосенсибилизации или фототоксичности при лечении фибратами или кетопрофеном; заболевания желчного пузыря в анамнезе; период грудного вскармливания; врожденная галактоземия, недостаточность лактазы, нарушение всасывания глюкозы и галактозы (препарат содержит лактозу); врожденная фруктоземия, недостаточность сахаразы-изомальтазы (препарат содержит сахарозу); пациенты с аллергией к арахису, арахисовому маслу, соевому лецитину или родственным продуктам в анамнезе (в связи с риском развития реакции повышенной чувствительности); хронический или острый панкреатит, за исключением случаев острого панкреатита, обусловленного выраженной гипертриглицеридемией. С осторожностью*: у пациентов с факторами, предрасполагающими к развитию миопатии и/или рабдомиолиза, включая возраст старше 70 лет, отягощенный анамнез по наследственным мышечным заболеваниям, гипотиреоз и злоупотребление алкоголем; применение при беременности; при одновременном приеме пероральных антикоагулянтов, ингибиторов ГМГ-КоА-редуктазы. **Применение при беременности и в период грудного вскармливания***. Фертильность. Клинические данные по влиянию препарата на фертильность у мужчин или женщин отсутствуют. Беременность. Потенциальный риск для человека не известен. Поэтому применять препарат во время беременности можно только после тщательной оценки соотношения ожидаемой пользы к возможному риску. Период грудного вскармливания. Не следует применять препарат во время грудного вскармливания. При необходимости применения препарата в период лактации, грудное вскармливание необходимо прекратить. Способ применения и дозы*: необходимо продолжать соблюдать гипохолестеринемическую диету, которой пациент придерживался до начала лечения препаратом Трайкор® 145 мг. Трайкор® 145 мг можно принимать в любое время дня, независимо от времени приема пищи. Взрослые. По одной таблетке препарата Трайкор® 145 мг один раз в сутки. Пожилые пациенты без нарушения функции почек. Рекомендуется принимать стандартную дозу для взрослых (1 таблетка в сутки). При отсутствии терапевтического эффекта после нескольких месяцев терапии (как правило, после 3-х месяцев) следует рассмотреть целесообразность назначения сопутствующей или альтернативной терапии. Пациенты с нарушениями функции печени. В связи с недостаточным количеством накопленных данных по применению препарата Трайкор® у пациентов с нарушениями функции печени, не представляется возможным дать рекомендации по применению препарата у данной категории больных. Пациенты с нарушениями функции почек. Пациентам с легкой хронической почечной недостаточностью (клиренс креатинина выше 60 мл/мин) коррекция дозы не требуется. Побочное действие: признаки и симптомы расстройства желудочно-кишечного тракта (боль в животе, тошнота, рвота, диарея, метеоризм); повышение активности сывороточных трансаминаз; повышение уровня гомоцистеина в крови. Перечень всех побочных действий представлен в инструкции по медицинскому применению. Передозировка*: специфический антидот неизвестен. При подозрении на передозировку следует назначить симптоматическое и, при необходимости, поддерживающее лечение. Гемодиализ неэффективен. Взаимодействие с другими лекарственными средствами*: фенофибрат усиливает эффект пероральных антикоагулянтов и может повысить риск кровотечений, что связано с вытеснением антикоагулянта из мест связывания с белками плазмы крови. Описано несколько тяжелых случаев обратимого нарушения почечной функции во время одновременного лечения фенофибратом и циклоспорином. При приеме фенофибрата одновременно с ингибиторами ГМГ-КоА-редуктазы или другими фибратами повышается риск серьезного токсического воздействия на мышечные волокна. Такую комбинированную терапию следует проводить с осторожностью и тщательно контролировать состояние пациентов на предмет наличия признаков токсического влияния на мышечную ткань. При одновременном применении фенофибрата и глитазонов сообщалось о нескольких случаях обратимого парадоксального снижения концентрации холестерина ЛПВП. Поэтому при проведении одновременной терапии рекомендуется контроль концентрации холестерина ЛПВП, и в случае выраженного снижения концентрации холестерина ЛПВП препараты отменить. Пациенты, применяющие фенофибрат совместно с лекарственными препаратами, метаболизируемыми изоферментами CYP2C19, CYP2A6 и особенно CYP2C9 с узким терапевтическим индексом, должны находиться под тщательным наблюдением и, при необходимости рекомендуется корректировать дозы этих препаратов. **Особые указания*:** Влияние на сердечно-сосудистую заболеваемость и смертность: Клиническое рандомизированное плацебо-контролируемое исследование ACCORD было проведено с участием 5518 пациентов с сахарным диабетом 2 типа, получавших фенофибрат в дополнение к терапии симвастатином. Анализ подгруппы пациентов с дислипидемией (уровень триглицеридов (ТГ) ≥ 2.3 ммоль/л и уровень холестерина липопротеидов высокой плотности (ЛПВП) ≤ 0,88 ммоль/л), продемонстрировал статистически значимое снижение относительного риска возникновения серьезных сердечно-сосудистых событий на 31 % в группе комбинации фенофибрата с симвастатином по сравнению с группой монотерапии симвастатином. Функция печени: рекомендуется контролировать активность трансаминаз (АЛТ, АСТ) каждые 3 месяца в течение первых 12 месяцев и периодически в течение дальнейшего лечения. Пациенты, у которых на фоне лечения повысилась активность «печеночных» трансаминаз, требуют внимания, и в случае повышения активности АЛТ и АСТ более чем в 3 раза по сравнению с верхней границей нормы прием препарата прекращают. При появлении симптомов гепатита (желтуха, кожный зуд) следует провести лабораторные исследования и, в случае подтверждения диагноза гепатит, отменить препарат Трайкор®. Панкреатит: были описаны случаи развития панкреатита в период лечения препаратом Трайкор®. Мышцы: при приеме препарата Трайкор® и других лекарственных средств, снижающих концентрации липидов, описаны случаи токсического влияния на мышечную ткань, с или без почечной недостаточности, включая очень редкие случаи рабдомиолиза. Частота такого нарушения повышается в случае гипоальбуминемии и почечной недостаточности в анамнезе. Токсическое влияние на мышечную ткань может быть заподозрено на основании жалоб пациента на слабость, диффузную миалгию, миозит, мышечные спазмы и судороги и/или выраженного повышения активности креатининфосфокиназы (КФК) (более чем в 5 раз по сравнению с верхней границей нормы). В этих случаях лечение препаратом Трайкор® 145 мг необходимо прекратить. Почечная функция: в случае повышения концентрации креатинина более чем на 50 % выше верхней границы нормы лечение следует приостановить. Рекомендуется определять концентрацию креатинина в первые 3 месяца и периодически в течение дальнейшего лечения. **Влияние на способность управлять транспортными средствами, механизмами.** Трайкор® 145 мг не влияет или влияет в минимальной степени на способность к вождению транспортного средства и управлению механизмами (риск развития головокружения). **Условия отпуска*:** отпускают по рецепту.*Полная информация представлена в инструкции по применению. СИП от 08.10.2020 г. на основании ИМП от 24.09.2020 г.

ОХ – общий холестерин. ЛНП – липопротеиды низкой плотности. ЛВП – липопротеиды высокой плотности. ТГ – триглицериды. СС – сердечно-сосудистый.

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compliance, it is mainly low-frequency NMES that has been studied. The efficacy of NMES was investigated in several uncontrolled studies and randomized controlled trials that compared the stimulation with lack of training or stimulation simulation and conventional physical training [13]. These studies show NMES to be comparable with conventional physical exercise in patients with CHF. Although it is inferior in terms of the effects on peak oxygen consumption, it is not inferior in terms of effects on muscle strength, 6MWD and quality of life [13–16].

The NMES techniques used by various authors are very diverse, differing both in terms of the technical characteristics of stimuli and stimulation intensity. It is suggested that more intensive stimulation may be more effective but is less tolerable to patients than moderate-intensity NMES. Tolerability issues are particularly relevant in older, fragile and less motivated patients.

We have not found any studies in the literature that compare two effective NMES techniques of different stimulation intensity. Therefore, we compared the tolerability and efficacy of short in-hospital courses of higher intensity NMES with the maximum tolerable muscle contraction achieved, as well as comparing lower intensity NMES with visible and palpable muscle contraction in elderly patients hospitalized with decompensated CHF. The functional performance of patients significantly improved in both groups in terms of subjective assessment of their wellbeing and increased 6MWD without a significant difference between the groups being identified. Here, it should be noted that the amplitude of stimulation pulses in the higher intensity stimulation group insignificantly exceeded those in the lower intensity stimulation group.

The satisfactory toleration of both stimulation modes, which did not cause significant responses of BP and HR, was consistent with the findings obtained by other researchers, such as Kondo et al. [16].

However, patients who had an opportunity to choose the intensity of NMES, mostly selected a lower-intensity regimen. As a result, the compliance with higher intensity and lower intensity regimens was 62.5 and 78.6% ($p>0.05$). Among patients who were willing to continue NMES training following discharge, 69% chose a lower-intensity regimen.

Conclusion

In older patients with decompensated chronic heart failure, neuromuscular electrical stimulation of lower intensity (until visible muscle contraction) may be better tolerated than neuromuscular electrical stimulation of higher intensity (maximum tolerated muscle contraction) and is not less effective. Applying neuromuscular electrical stimulation of lower intensity may be a way to increase compliance with the rehabilitation programs in patients with chronic heart failure.

Limitations

Given the objective of the study, which assumed a comparison of two effective stimulation modes, there was no control group or placebo. The efficacy of NMES in patients with CHF had been shown previously in placebo-controlled trials. The small number of patients and short treatment duration may have affected the significance of differences between the patient groups in this pilot. Further studies are planned to include more patients and a placebo group.

No conflict of interest is reported.

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