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Research paper

Review of the evidence on the use of electrical muscle stimulation to treat sarcopenia

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ABSTRACT

Electrical muscle stimulation (EMS) has been shown to increase muscle strength and mass, suggesting that it can be used for the treatment of sarcopenia. We herein present a detailed review of the physiological changes induced by EMS. Only human experimental and clinical studies were selected as reference articles for this review. Low-frequency stimulation appears to have greater effects on strengthening, while high frequency favors an increase in muscle mass. EMS also promotes compositional changes of the muscle fibers and improvements in the oxidative enzymatic activity and glucose uptake. Considering that structural changes towards type II muscle fibers and their motor units have an important role in the loss of muscle strength and that the response to muscular training is based on the capacity to increase muscle activation, an intervention protocol using EMS should focus on stimulating type II muscle fibers, rather than on inducing muscle hypertrophy.

1. Introduction

More than 20 years ago, the term sarcopenia was proposed to describe the "poverty of flesh" that occurs in skeletal muscle due to aging, describing a phenomenon that had been frequently observed but minimally investigated in the aged population [1]. Advances in research have led to the recognition of sarcopenia as a clinical entity. Although sarcopenia was originally defined as a low muscle mass (less than the mean of the younger population–2SD), it is now defined as an impairment of physical function, and its defining criteria is a loss of muscle mass and a decrease in muscle strength and/or gait speed related to the process of aging. These changes in the definition of sarcopenia have since been translated into several diagnostic algorithms based on the gait speed, handgrip strength or knee extension force; along with the lean muscle mass assessed by dual-energy X-ray absorptiometry (DXA), bioelectrical impedance analysis (BIA), or other modalities [2]. With better-delineated parameters to measure, therapeutic interventions for sarcopenia targeting muscle strength and muscle mass have been the objective of many studies. Among the various interventions focusing on muscle strengthening, the use of electrical muscle stimulation (EMS) to promote the enhancement of the physical performance of young subjects based on variables such as strength, speed, power, and sprint time [3] has drawn attention to the potential applicability of this approach to improve and prevent sarcopenia in the older population growing worldwide.

The purpose of this article is to review the neurophysiological properties of muscle strengthening in older adults, to appraise various aspects of EMS interventions for a better understanding of their effects on skeletal muscle and to discuss the benefits of EMS as an alternative/complementary treatment option for sarcopenia.

2. Strength training in the older population

Muscular weakness resulting from sarcopenia can be simplified as the product of two major factors: a decrease in muscular strength and a loss of muscle mass. Therefore, the objective of any treatment should be to increase both the strength and muscular volume/cross-sectional area (CSA). In pioneering research regarding the effects of muscle training assessed by electromyography (EMG), Moritani and deVries described essential differences in how an increase in strength was achieved in older subjects [4]. By devising an EMG-based method to quantify the neuromuscular adaptations during the acquisition of muscle strength, they made it possible to infer two separate factors explaining the variation from the EMG electrical signs data plotted against the muscle-generated

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force/strength. The first factor is the "neural factor," corresponding to the increase in the voluntary activation of electrical muscle activity without changes in the force levels exerted by the muscle fibers or the number of motor units (MUs) recruited. The second factor is the morphological transformation (muscle hypertrophy), visualized in EMG by an increase in the force generation per fiber/MU without any changes in the measured electrical muscle activity.

Next, they successfully demonstrated two different EMG patterns of strength gain when they compared old and young subjects. After an eight-week period (three days/week) of regular resistance training in the dominant arm with dumbbells, the majority of the strength gains in older subjects were ascribed to an increase in the neural factor activity, with no significant muscular hypertrophy (based on both EMG quantification and arm CSA measures). In fact, an analysis of the absolute EMG values in microvolts showed that older men had significantly lower maximum EMG standards (muscle activation level) [4], indicating that they had a lower capacity for strength generation compared to younger men.

In agreement with these findings, studies on the muscle force generation of isometric maximal voluntary contraction (MVC) in older adults showed 11% lower force generation in these patients than in younger adults in their central activation ratio (defined by the equation CAR = MVC force/MVC force + superimposed electrical burst force). Hence, given that the rate of strength loss is higher than that of muscle mass loss during natural aging, it is reasonable to conclude that the neural factors are reduced in the older population [5]. Taken together, these experimental data indicate that older individuals can increase their strength with physical exercises, but their starting point is at lower levels than that of the younger population and is conditioned to a neural factor dependent-response mechanism.

3. Changes in the muscle structure associated with aging

The decrease in strength in older people occurs more rapidly and is more prominent than muscle atrophy, a phenomenon also referred as dynapenia (poverty of strength) [6]. Understanding the underlying anatomical and histological adaptations of the skeletal muscle related to this finding can clarify the inconsistencies in the previous assumption that there is a simple association between muscle weakness and a low muscle mass. In fact, besides the generalized wasting of muscle fibers and motor neurons, which results in a loss of muscle quantity, researchers have described numerous structural modifications in the muscle that occur during aging, such as a shortening of the muscle fascicles, a selective reduction of the diameter of type II muscle fibers, a decrease in the number of satellite cells per muscle fiber and changes in the expression levels of myosin heavy chain (MHC), in order to explain the reduction of muscle strength [7].

The idea that dynapenia is caused by a decline in physical performance due to a muscle strength/power reduction rather than a muscle mass loss is supported by reported changes in the physiology of the central and peripheral nervous systems (primary motor cortex atrophy, cortex hypoexcitability, reduced voluntary MUs recruitment, type II muscle fiber denervation and type I muscle fiber collateral reinnervation) and the intrinsic force generation capacity of the muscular skeletal tissue (perturbation in the excitation-contraction coupling, intramuscular adipose tissue infiltration) [6].

4. Physiology of electrical muscular stimulation

Muscle contraction is the response of resting muscular tissue to a neurophysiological stimulus. The activation of muscle fibers to produce tension is triggered by a biological self-generated electrical stimulus called an action potential, which is carried throughout the nervous system. More specifically, the action potential is carried by the peripheral motor nerves (specifically the motor neurons), whose axonal ramifications innervate all skeletal muscle fibers, establishing the MUs [8]. However, these same MUs can be triggered by artificial/external electrical currents, referred to in this review as EMS, which can be delivered through the skin by surface electrodes, provoking involuntary muscle contractions.

Upon voluntary muscle contraction, a size order recruitment of MUs is executed, starting from the lowest force/smallest MUs to the highest force/largest MUs, promoting a gradual increase in strength generation and a smooth control over the body's movements. On the other hand, involuntary muscle contraction elicited by EMS will preferentially trigger the large MUs, with a synchronized activation of a large number of muscle fibers (resulting in a series of successive twitches), causing a sudden and stronger contraction but also more quickly leading to muscle fatigue [8].

The muscle maximum voluntary contractions (MVCs) analyzed by EMG have revealed that to maintain a constant force output through time, a progressive increase in myoelectrical signals occurs as a compensatory mechanism under physiological conditions because of the fatigue of the initially recruited MUs. Moreover, the slope coefficients of the fatigue curves (sum of the EMG signals vs time) were nine times greater in the biceps brachii than in the soleus muscle at 40% of the MVCs [9]. Indeed, the biceps brachii muscle presented higher fatigability at several different percentages of the MVCs, which can be explained by its higher percentage of type II (Fast Twitch) muscle fibers than that of the soleus muscle.

With regard to EMS, fatigue is related to the frequency of the delivered electrical stimulus, with a higher frequency leading to stronger muscle contraction, but the fatigue can be accelerated by the application of a higher frequency. When testing EMS for the triceps surae muscles at 20 Hz, 50 Hz and 80 Hz, only the lower frequency (20 Hz) could maintain the muscle contraction force constant without changing the amplitude of the EMG signals (evoked muscle action potentials). At 50 Hz and 80 Hz, the force output was matched to the MVCs values; however, a rapid decrease of force within 60 s of stimulation was observed, along with an extremely reduced amplitude of EMG signals [10].

EMS also induces a preferential recruitment of type II fibers (fast MUs) because the axons are of larger size and relatively lower electrical resistance than the smaller axons of type I fibers (slow MUs). This "reverse size principle" of MUs recruitment has been demonstrated in experiments comparing the fatigability of the knee extensor muscles with equal force (10% MVC) voluntary and EMS-elicited contractions; a higher fatigue was observed for type II MUs only in the EMS intervention group [11].

5. Structural changes of the skeletal muscle induced by EMS

There are numerous commercially available EMS devices that have shown therapeutic effects, such as motor nerve regeneration, denervated muscle atrophy prevention and muscle strengthening. A systematic review on the metabolic and structural adaptations promoted by electrical stimulation on the lower limb skeletal muscles reported differential effects according to the stimulation frequency used for EMS [12]. Higher frequency currents (greater than 50 Hz) were associated with an increase in muscle size, while lower frequency currents (lower than 20 Hz) induced an increase in oxidative enzymatic activity. However, conflicting results on the muscle size and muscle fiber composition have been reported. The period of training also varied from 4 to 12 weeks, and the duration of each EMS session ranged from a minimum of 10 min to eight hours/day, but it was one to two hours per day on average [12].