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Research Article

Neuromuscular Electrical Stimulation in Muscular Adaptations in Exercise: A Narrative Review

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Abstract

Nowadays, sports science uses scientific methods and medical devices to assist people with any improvements in sports. Muscle adaptations have significantly benefited as a result of the use of these advanced devices. It has been shown that neuromuscular electrical stimulation (NMES) devices effectively improve muscle function. The use of NMES devices in exercise physiology shows that neuromuscular adaptation is a current research area in both athletes and non-athletes. This narrative review aims to address neuromuscular adaptations and describe neuromuscular changes based on research using NMES. Many researchers and sports trainers will benefit from the results of this article by better understanding neuromuscular adaptations. NMES training has been shown to be an effective way to improve muscle growth, maximum voluntary strength, neuronal drive, oxidative metabolism, and antioxidant defense systems. In addition, NMES is capable of regulating the homeostasis of muscle proteins and increasing oxidative enzyme activity. In animal models, it has also been shown to increase axonal outgrowth, fiber reinnervation, and motor axon regeneration. Various NMES methods may decrease age-related muscle atrophy and functional deterioration. The use of NMES, which is one of the most successful strategies for increasing athletic performance through neuromuscular adaptations, is one of the most promising areas of research.

Keywords:
Neuromuscular Electrical Stimulation, Muscle Adaptations, Exercise, Performance, NMES

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1. Introduction

The use of an electrical current to stimulate muscle contractions has been around since the 18th century when it was first used in neuromuscular tissue (1, 2). Electrical impulses can induce muscle contraction through electrical muscle stimulation. Alternatively, it is referred to as neuromuscular electrical stimulation (NMES) or electromyostimulation. In recent years, NMES has increasingly received more attention for several reasons. Both athletes and healthy individuals can use it for strength training (3-5). Rehabilitation and prevention can be achieved through their use by people who are partially or unable to move. This test can evaluate neurological and/or muscular functions and assist athletes in recovering after exercise (6).

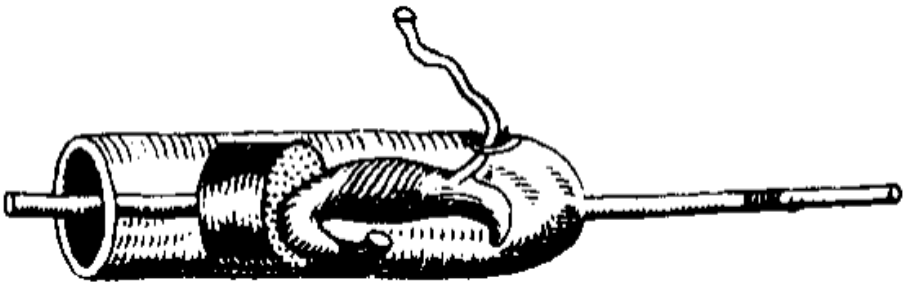

Figure 1: Swammerdam's illustration of a nerve-muscle preparation

About 350 years ago, it was seen that electrical current could be used to cause muscles to contract. Jan Swammerdam (1637–1680) demonstrated in the 1670s that a frog nerve-muscle preparation could be stimulated externally "irritated" via the nerve using scissors. However, he could not describe the specific process leading to muscle activation at that time (Figure 1) (7).

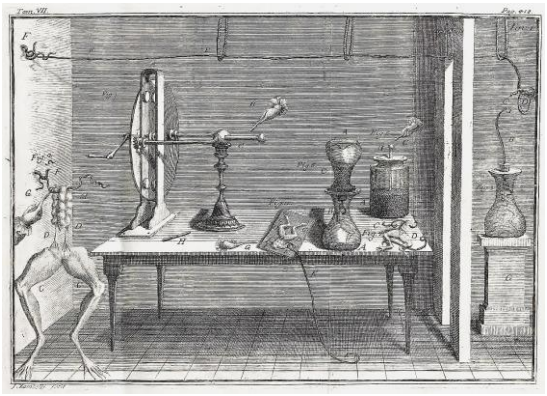

Figure 2: The illustration of Galvani's experiments

After a 3-month therapy session, Jean Jallabert (1712–1768) was able to persistently activate a patient's paralyzed right upper limb using electrical stimulation from a Leyden jar (in other words, a battery) (8). While conducting experiments with static electricity in 1791, Luigi Galvani (1737–1798) dissected a frog on a bench (figure 2) (8, 9). By chance, his assistant touched an exposed sciatic nerve with a metal scalpel that had picked up a charge, and they immediately saw sparks and a strong muscle contraction of the frog's leg as a result. Although Alessandro Volta first misunderstood and hotly contested the physiological principles underpinning muscle activation, Galvani's fundamental discovery was that electrical current might elicit muscular contraction.

Modern electrical generators were made possible by Michael Faraday's (1791–1867) discovery of electromagnetic induction, which was of utmost significance (10). Guillaume Duchenne de Boulogne (1806–1875), regarded as the pioneer of electrotherapy (11), was among the first to employ faradic currents to activate facial muscles using wet surface electrodes (figure 3). He clearly defined the connection between facial muscle contraction and the emotion conveyed by combining photography and electrical stimulation (12).

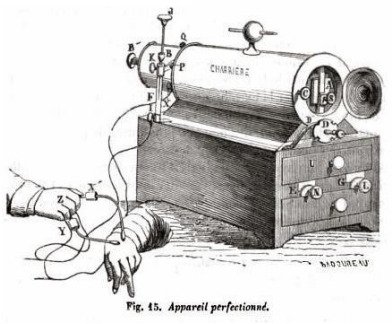

Figure 3: The Woodcut illustration of Duchenne's volta-electric device

Electrical stimulation became an effective therapy for countering muscular atrophy caused by denervation due to the high number of injuries sustained during wars in the first half of the twentieth century. The Russian scientist Yakov Kots hypothesized in 1971 that electrical stimulation of muscles might be more effective than voluntary contractions in boosting maximum strength (8). The groundbreaking Kots findings established the use of electrical stimulation as a technique of muscular performance enhancement, keeping with the well-known Olympic slogan "Faster, Higher, Stronger." This artificial training approach has been seen initially more as a technical gadget than a suitable tool for developing muscular strength, which is not surprising given the poorly managed use. Electrical stimulation has recently been shown to be an effective and legal supplement to voluntary resistance training programs for increasing muscular strength.

A scientific framework for improving the performance of athletes will be developed based on previous research on muscle adaptation. Afterward, according to neuromuscular stimulation, an explanation of possible changes in the mechanism of improving muscle contraction will be provided. For many coaches and trainers of any sports team, this article can help them understand how muscles adapt scientifically. NMES involves intermittent high-frequency trains of electrical stimulation [40–50 Hz] delivered via surface electrodes placed over the motor point to induce (vigorous) contractions of the skeletal muscles as a result of the activation of intramuscular nerve branches. By presenting an overview of the existing evidence about the alterations in muscle performance caused by many bouts of NMES in both healthy individuals and athletes, the advantages and limits of NMES training will be discussed in more detail.

The aim of this narrative review was to provide a general physiological overview of the NMES in muscle adaptations and performance improvement in athletes. Even though much progress has been made in this field, more research is still needed because medical knowledge is constantly changing.

Muscular Adaptations in Exercise

Human Muscle

Muscle fibers are equipped with many metabolic processes to facilitate this adaptation (2). These systems need sensing mechanisms (so the muscle fiber knows it has been worked out), amplification mechanisms (through different and often redundant metabolic pathways that lead to things like the phosphorylation of other proteins), and effector mechanisms (a change in net protein synthesis as a result). The nuclei present throughout the muscle fiber length are involved in the effector processes, which include alterations in transcription of some of the 30,000 genes that make up the genome and posttranscriptional mechanisms leading to changed protein synthesis.

The adductor pollicis muscle is an exciting model for investigating adaptations of muscle contractile properties following exercise. In a study for a 3-month period, one group of subjects performed maximal isometric contractions (10 contractions of 5 s duration) against a load of 30–40% of the maximum (13). This technique was used by Duchateau and Hainaut (1984) to compare the effects of two types of training on human volunteers (14, 15). The maximal muscle force increased significantly (20%) after maximal isometric contractions than after dynamic contractions.

The duration of the training program is also an essential factor in inducing changes in the muscle's force-generating capacity (16). After five weeks of isometric maximal voluntary contractions (MVC) of elbow flexors, McDonagh and colleagues observed no significant changes in the tetanic force of the biceps brachii (BB) (figure 3) (14).

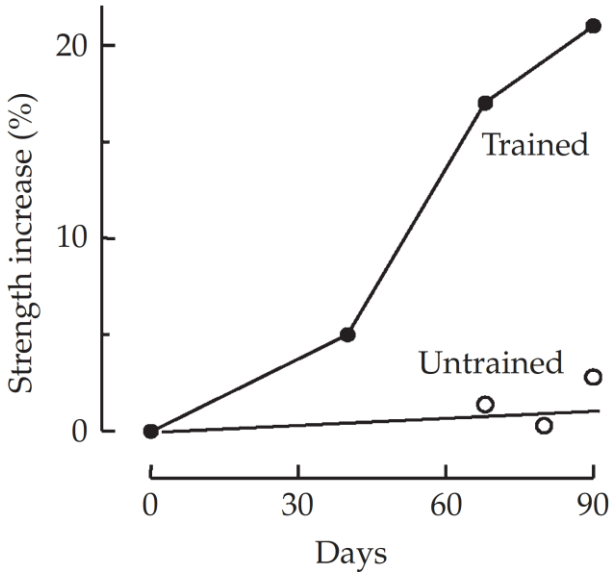

Figure 4: Changes in the adductor pollicis muscle's maximum tetanic force (100 Hz) over the course of a three-month strength-training regimen. The load ranged between 60% and 70% of full capacity. During the first 40 days of training, the trained muscle's tetanic force only went up by 5%. After that, it went up much more, reaching 21% at the end of the training program. The muscle of the contralateral hand remained unchanged.

Motor Unit

There was one research that measured the tetanic force of the motor unit earlier in a human before/after training (17). This technique employs percutaneous electrical stimulation to stimulate single motor axons at many places along the median nerve and to monitor their contractile properties in the thenar muscles. Because the form and magnitude of motor unit action potentials are highly consistent when electrodes are changed in the same position between sessions, longitudinal monitoring of the same motor units is feasible. Using this experimental method, Chan et al. (1999) found that motor unit adaptation to a program of high-frequency electrical stimulation varied in terms of their physiological properties (17). While the twitch and peak tetanic forces of the slower and fatigue-resistant unit grew, the force of the quicker and fatigue-prone unit unexpectedly decreased. The number of motor units per subject is relatively low, which is a significant limitation of the approach for analyzing the effects of a training intervention. For these reasons, other researchers decided to utilize a different technique known as spike-triggered averaging (18). Due to its unique architecture, this is the sole approach that can be utilized to evaluate the contractile properties of a deliberately triggered individual motor unit. In brief, action potentials from a single motor unit discharging at low frequency are employed as "spike triggers" when detected by an intramuscular needle electrode. Since the force produced by this motor unit is time-locked to the action potential (spike), it may be retrieved by averaging the force signal. When comparing the effects of isometric and dynamic training (as mentioned before), unique motor unit changes were detected (19).

Following isometric training, all motor units demonstrated a nearly proportionate increase in peak force without changing the twitch time course. As anticipated, motor units exhibited a minor increase in force after dynamic training, but the time-to-peak for the whole population of motor units was decreased. There was no indication of a change in the "size principle" after either dynamic or isometric training since a linear relationship between motor unit force and recruitment threshold was repeatedly seen. Similar modifications were seen in the tibialis anterior after dynamic exercise (20). These results demonstrate, once again, that muscle changes its contractile characteristics to the type of exercise.

Relation Between Muscle Size and its Strength

The number of parallel muscle fibers and sarcomeres in each fiber, as well as the angle between the fibers and the muscle's longitudinal axis (angle of pennation), are the main factors determining how much force a muscle can produce (2). Consequently, it is possible to measure the strength of a muscle anatomically by measuring its cross-sectional area (CSA)(21, 22). For technical reasons, measuring the anatomical CSA is more accessible, which involves taking measurements perpendicular to the muscle's long axis. This measurement should be done perpendicular to the direction of the muscle fibers. This measurement is often carried out using imaging methods (23).

Even though there is a chance of bias when measuring anatomical CSA, a strong correlation was found between the peak force of the calf muscles during electrically induced contraction and the CSA of a muscle group (23). On the other hand, there is more fluctuation in the connection when force is assessed during an MVC. For instance, the variance in CSA accounts for around half of the difference in strength across patients in specific research (22). This indicates that the capacity of a muscle to generate force is determined by parameters other than its size.

Specific Fiber Tension

Specific tension refers to the maximum force a muscle or muscle fiber exerts per unit CSA ($N.cm^{-2}$). This metric indicates the intrinsic capacity of the muscle or muscle fibers to generate force (2). At the whole muscle level, it has been discovered that depending on the muscle group under investigation, the tension in trained women is either higher or comparable to that in untrained women. In addition, it was found that there was no difference in strength per unit CSA between professional bodybuilders with severe hypertrophy and physical education student (24, 25). However, since the amount of connective tissue differs across individuals, measuring specific stress from the entire muscle may be misleading. The use of anatomical rather than physiological cross-sectional area as an indication of muscle size and the volitional drive's submaximal muscle group activation may contribute to this heterogeneity. Recording particular stress at the muscle-fiber level helps bypass these confounding variables. It has been shown that under these circumstances, type II but not type I vastus lateralis (VL) fiber-specific tension is higher in young, active adults than in older, sedentary adults, and type II but not type I fiber-specific tension is higher in bodybuilders than in sedentary individuals (26).

Additionally, it was shown that particular stress rose after a strength-training regimen. At least two processes may account for variations in specific tension at the muscle fiber level (27, 28). The number of myofilaments in each muscle fiber and how well force is transferred from the sarcomere to the skeleton are examples of these mechanisms.

Alteration in Total Muscle Mass and Construction

The maximum force of a muscle is highly correlated with its CSA, an increase in the latter metric results in an increase in maximal force (figure 4). There are two potential methods for increasing muscle mass: a development in the CSA of individual fibers of muscles (hypertrophy) and an increasing number of muscle fibers (hyperplasia). Most experimental data indicates that hypertrophy is the primary mechanism for increased muscle force (29). However, hyperplasia may occur in humans under certain situations (30).

Muscle Mass

It is now widely recognized that the size of the CSA increase with training relies on some variables, including the individual's starting strength, the load level, the length of the training program, and the training technique (23). For instance, six weeks of isometric training (80% MVC) in novices raised the CSA of the elbow flexor muscles by around 5%, but eight weeks of identical training increased the CSA of the quadriceps femoris by 15% (31). In contrast, 24 weeks of intense strength training by highly competitive bodybuilders had no significant effect on the CSA of BB muscle fibers (32).

However, the reasons are unclear 60–80% of 1-RM with 6–12 repetitions and 6–10 sets, as utilized by bodybuilders, seem to be more effective for muscular growth than larger weights (>80% of 1-RM) with fewer repetitions (33). Moreover, eccentric contractions have been shown to have a more significant effect on muscle hypertrophy, as indicated by the tremendous increase in CSA following a training program for knee extensor muscles that included both concentric and eccentric muscle actions compared to a training program with only concentric muscle actions. Strength training has also reportedly been linked to variations in the hypertrophy of a muscle group's various parts. For instance, following six months of strength training with a load of 80% maximum, the quadriceps CSA rose by roughly 19% in the distal and proximal areas but only by 13% in the central region (22).

Muscle Construction

Muscle architecture may alter with strength training in addition to CSA. Aagaard et al. (2001) evaluated muscle CSA and volume with MRI and VL pennation angle with ultrasound (34). After 14 weeks of training, knee extensor MVC force (16%), quadriceps volume (10%), and VL muscle fascicle pennation angle (36%) increased. The more significant increase in MVC force compared with muscle volume was ascribed to the pennation angle, which increases muscle force per unit volume. As mentioned, these findings affect muscle tension estimation. The physiological cross-section is more accurate than the anatomical cross-section for examining peripheral strength training responses. In specific muscles (e.g., calf muscles), excessive hypertrophy may elevate the pennation angle to a detrimental level for power or speed output.

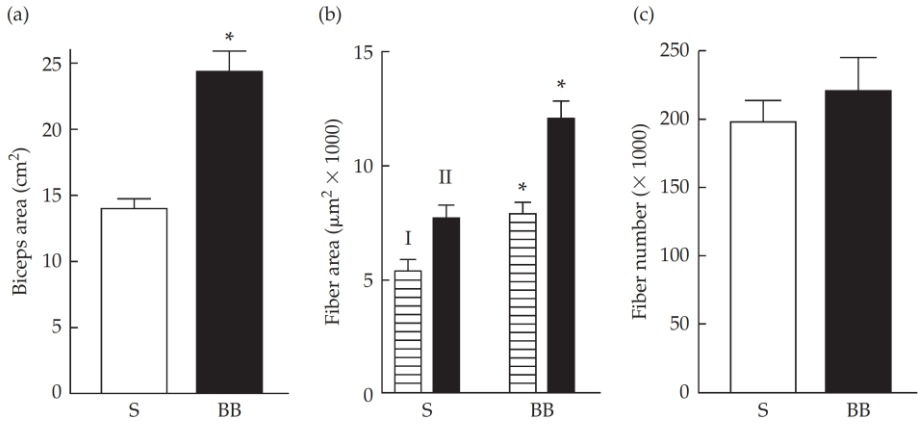

Figure 5: Compared muscle mass, muscle fiber size, and number of fibers in the biceps brachii of sedentary (S) and physically active (A) participants (BB). The mean CSA for the whole muscle, as determined by computed tomographic scans of the upper arm (a), and for the two major kinds of muscle fibers (type I and type II), as determined by needle muscle biopsies (b), differs between S and BB. In contrast, there is no significant difference in the projected mean fiber number derived by dividing the CSA of the whole muscle by the CSA of the average fiber (c).

P0.01, differences between the two groups.

Fiber Adaptations in Response to Exercise

The particular adaptations that result from the various strength-training techniques show that muscles are subject to general adaptations of their various muscle fiber types and changes in the makeup of their fiber types (2). Nevertheless, despite the variation in muscle biopsies, most research found that strength training did not change the percentage of type I and II fibers in muscles, even when MVC force is increased by 45% (32). Nevertheless, after a strength-training program, it is common to see an increase in type IIa fibers and a decrease in type IIb (IIx) fibers in VL (35).

There are more differences in the amount of hypertrophy amongst muscle fiber types. For instance, a 16-week isometric exercise raised the CSA of type I and type II fibers by 20% and 27%, respectively, in the SOL, but the lateral GAST showed a 50% increase in type II fibers but no change in type I fibers. Similar to this, after 14 weeks of exercising the knee extensors, the CSA of type II rose by 18%, while the type I fibers in the VL remained unchanged. In contrast to concentric contractions, eccentric contraction regimens seemed to encourage the hypertrophy of type II fibers (23).

Numerous studies show that the majority of the hypertrophy brought on by intense strength training occurred in type II fibers, even though the quantity of hypertrophy between type I and type II fibers did not consistently vary significantly (23). In fact, after six months of training, the area of type II fibers had risen by 29% and 13%, respectively, under severe load (70-100% of 1-RM) and dynamic contractions (10-60% of 1-RM done at full speed) (33).

Approximately a 4% increase in the CSA of type I and type II fibers was reported (36). Unexpectedly, it has recently been shown that stretch-shortening cycle training may cause a significant increase in muscle fiber CSA of the VL (22–30%). The pretraining level is a determining factor in the relative adaptability between the two primary fiber types, since trained athletes do not demonstrate such a large impact as beginners (32).

The Mechanism of Hypertrophy

Muscle hypertrophy involves changing net protein synthesis and increasing myonuclei to control contractile protein volume. Chronic muscular stretch overload increases protein synthesis over breakdown, resulting in a net contractile protein increase and muscle growth. "Satellite cells" become new myonuclei. Under the basal lamina are quiescent mononucleated satellite cells (23, 30). In response to increased activity, they proliferate, and some merge with a muscle fiber, adding additional nuclei to the current fibers, while others may form quiescent satellite cells (30). New myonuclei create mRNA and proteins like old ones. Protein synthesis causes fiber hypertrophy. In type I and II VL fibers, their quantity looks comparable. Few know the specific signal that causes satellite cells to contribute nuclei to muscle fibers. A single high-intensity exercise session may activate satellite cells, but it is not enough for final differentiation. Satellite cells must grow significantly to become myonuclei. When training increases fiber area by 17%, the number of myonuclei does not change, suggesting that the rise is due to protein synthesis, but more considerable hypertrophy (>30%) is accompanied by an increase in myonuclei (30). The quantity of myonuclei added following weight training correlates strongly with fiber hypertrophy.

Hypertrophy stimuli cause a net protein increase owing to a shift in protein synthesis and breakdown, followed by satellite cell activation and proliferation (23). The fusing of satellite cells to muscle fibers occurs later in the hypertrophy phase when protein synthesis alone cannot produce additional hypertrophy.

Intracellular signaling mechanisms enhance protein production and breakdown (23). The protein kinase B (Akt) triggers the mTOR transduction pathway. The growth factor-1 (IGF-1) isoforms generated by skeletal muscle in response to exercise stress stimulate Akt activation. This IGF-1 isoform, mechano growth factor, boosts protein synthesis. After interacting with a transmembrane receptor, it activates Akt, which promotes protein synthesis via mTOR pathways and inhibits protein breakdown by phosphorylating a transcription factor and reducing protein production. Other exercise cues than mechano growth factor may activate the Akt pathway, and many of the underlying processes of muscle hypertrophy are unclear. Myostatin inhibits muscle development, while IGF-1 stimulates it. Strength training reduces myostatin mRNA, whereas endurance exercise reduces it without muscle fiber growth. Although mRNA isoform expression was increased following a single strength training session, it takes 3–6 weeks of training before protein synthesis produces noticeable improvements in muscle growth (37) and primary muscular strength (23).

Hyperplasia

Despite the fact that the majority of experimental data points to hypertrophy as the primary mechanism behind the rise in muscle mass, hyperplasia may also be involved in the rise in muscle size (2).

However, the degree to which fiber hyperplasia may develop in the muscles of persons who engage in strength training remains debatable. Indirect research implies that athletes have more muscle fibers than untrained persons (33). This contradicts the findings of Sale et al. (1987), who compared the size of the BB of untrained participants to elite and intermediate bodybuilders (38). The number of muscle fibers was determined based on the ratio of total muscle area computed by CT scanning to the average fiber area from needle biopsies. According to the results, there were no significant differences in the average number of fibers across groups. McCall et al. (1996) found no change in BB fiber counts in young males after 12 weeks of rigorous strength training (39). Kadi et al. (2000) postulated that satellite cells might combine to produce new muscle fibers or repair broken muscle fibers (30). These scientists found that, unlike untrained persons, top power lifters had tiny diameter fibers that exhibited embryonic and neonatal myosin heavy chain compositions. Considering these essential breakthroughs, hyperplasia seems moderate in humans. Therefore fiber hypertrophy remains the predominant mechanism of mass muscle growth after strength exercise.

Force Transmission Adaptations to Exercise

Adaptation of muscle contractile characteristics following exercise may also affect MTC SEC. An increase in the SEC's stiffness may be required for more efficient force transmission. This shift in elastic properties improves performance by promoting the release of potential energy during stretch-shortening cycle workouts by decreasing the duration between the stretching and shortening phases. In DJs, plyometric training enhanced muscular pre-activity, supporting this idea.

This modification may have increased muscle-tendon stiffness at the moment of the impact, resulting in increased muscle-tendon stress. In addition to increases in muscle activation, Pousson et al. (1990) found increased stiffness of the SEC following six weeks of eccentric contractions of the elbow flexor muscles, as measured by a quick-release method (40). This shift occurred independent of elbow flexor force but was larger at low force. Cornu et al. (1997) found that plyometric training decreased the slope of the stiffness–force connection in plantar flexor muscles (41). The authors proposed that training can cause opposite changes in the passive structures of the SEC—tendon—and the active structures—muscles around the joints—so that the change in stiffness of the entire musculo-articular system will depend on their respective adaptations. This would explain the apparent discrepancy between the two studies.

Tendon–Aponeurosis Features

As a result of the quick-release technique's inability to discern between changes in either the contractile or tendon structures, ultrasonography is now often utilized to measure and quantify changes in tendon-aponeurosis complex stiffness (23). It has been documented that young and old persons had an increase in tendon stiffness after a strength training program with large loads (42). Similar results have been seen in young adults following training with eccentric movements (43). It indicates that the contraction mode is connected to the degree of tendon adaptation to training. In fact, data from the same lab show that isometric training tends to have a more significant effect than heavy-load training that moves.

In contrast, training with plyometric movements and ballistic isometric contractions did not affect tendon stiffness. Since plyometric exercise increases joint stiffness, the most significant changes may be in the contractile structures. It was further hypothesized that training-induced alterations in the internal structure of the tendon since none of these studies found that the training regimen altered tendon size. Contrary to this finding, long-distance runners had a higher CSA (22%) of the AT than nonrunners (44). This apparent difference may be described by endurance vs. strength training or by the delayed hypertrophic response that arises after a more extended exercise.

Neuromuscular Electrical Stimulation

To induce powerful muscular contractions, NMES involves transmitting preprogrammed trains of stimuli to muscles using surface electrodes placed on the skin. There is currently strong proof that NMES is an appropriate and legal addition to voluntary resistance training routines for increasing muscular strength and hypertrophy in healthy individuals (2).

In medicine, NMES is used for rehabilitation via medical devices in a laboratory (6). For example, it is utilized in physical therapy to prevent muscle atrophy due to inactivity or neuromuscular imbalance, which can occur for various reasons, for instance, after musculoskeletal injuries such as damage to bones, joints, muscles, ligaments, and tendons (21). This is not the same as transcutaneous electrical nerve stimulation, a pain treatment that uses an electric current. In the case of such stimulation, the current is often subthreshold. Therefore, muscle contraction is not detected. Progressive disorders such as cancer or COPD employ NMES to improve muscular weakness in individuals unable or unable to exercise whole-body.

NMES may enhance quadriceps strength, but further study is required since the data is weak (3). The same research suggests that NMES may result in more muscle hypertrophy (45). Insufficient data suggests that adding NMES to an existing fitness program may help ill individuals spend fewer days confined to their beds. During NMES training, complementary muscle groups are frequently targeted alternatingly for specific training goals, such as enhancing the ability to reach an object.

Resistance training has been demonstrated to be an effective treatment for neuromuscular deterioration (45). NMES has been used as a substitute to attenuate or treat muscular mass and strength reductions in aged individuals when resistance exercise intervention is infeasible due to circumstances such as injury and/or prolonged bed rest (46). Electrical stimulator devices cause the motor neuron axons and their branches, or the muscle fiber, to depolarize, which causes the muscle to contract (46). This can be done in several methods, including by stimulating a motor neuron directly or the superficial muscular bellies with self-adhesive surface electrodes. NMES, comprising stimulation-rest cycles, is administered over weeks or months to develop muscular tetany and muscle contraction (47). According to the Henneman size principle, the recruitment of MUs during voluntary contractions follows a pattern from slow twitch muscle fibers to fast twitch muscle fibers and from small MUs to larger ones (48).

On the other hand, the recruitment pattern by NMES is temporally synchronized, spatially fixed, and not selective, as shown by the early recruitment of a large number of fast twitch muscle fibers that get tired quickly (45).

In contrast to motor nerve stimulation, which engages all muscle fibers inside a MU, direct muscle stimulation stimulates all fibers close to the stimulating electrodes, which may or may not include whole MUs (45). Muscle or nerve excitation mainly relies on the proximity of stimulating electrodes, whereas axon depolarization is based on membrane resistance.

The primary effects of neuromuscular abnormalities are tissue atrophy and the inability to generate force effectively. The muscular structure is evaluated based on CSA, muscle fiber length, and pennation angle (49). It is possible that NMES positively increases total muscle size in humans, despite the contradictory evidence resulting from the diverse populations tested, the various aspects of NMES protocols, and the addition of resistance training and/or nutrition (50). In healthy elderly, eight weeks of high-frequency NMES (75Hz) administered to both the vastus lateralis and vastus medialis muscles increased CSA significantly (51). The combination of NMES and voluntary exercise resulted in an even higher rise in knee extensor CSA. Following nine weeks of NMES, histochemical and morphological studies showed that the diameter and proportion of fast-type muscle fibers increased while slow-type fiber diameter decreased (49). In addition, a significant rise in the number of satellite cells in the fast-type muscles was detected in the elderly following NMES, showing that NMES can stimulate muscle regeneration and hypertrophy (50). Evidence suggests that capillary growth and muscle fiber growth happen at the same rate in the skeletal muscles of humans (9, 15, 21, 45). This reveals that there is a positive relationship between capillarization and muscle fiber hypertrophy.

Although these findings on capillary supply adaptations in healthy elderly are limited, it has been observed that high-frequency NMES improved muscle capillarization and preceded the conversion of muscle fiber phenotype, illustrating the significance of angiogenesis and muscle fiber capillarization, specifically in older muscle.

Individual fiber and overall muscle atrophy are caused directly by an imbalance between muscle protein production and muscle protein breakdown. Muscle atrophy can be reduced or prevented by a protein diet plan and exercise. Regardless of dietary consumption, even moderate-intensity physical activity can preserve skeletal muscle mass, underscoring the potential of NMES as an interventional treatment (2). Five days of bed rest with NMES and protein supplementation in healthy older adults did not reduce muscle mass (52). Similarly, Dirks et al. (2016) examined the efficacy of NMES in conjunction with pre-sleep protein consumption on muscle protein synthesis (MPS) in older persons (53). Before 20g protein feeding, a 70-min single bout of NMES was performed unilaterally on the lower limb, and muscle biopsies after 4 hours revealed no change in myofibrillar MPS between the stimulated and control legs (53). However, the similar NMES procedure with 40g of protein rather than 20g increased muscle protein synthesis 8 hours after feeding, indicating that metabolic responses to NMES are sensitive to dietary intervention and time-dependent. Additionally, a single NMES session in elder type 2 diabetics, who are more sensitive to muscle atrophy and functional deterioration, increased MPS by 27% (54). In patients with knee osteoarthritis, four weeks of daily NMES performed at home increased muscle fiber size, which was associated with a rise in MPS.

Depending on the knowledge currently available on NMES and MPS, NMES can improve MPS whether used alone or as an addition to dietary (protein-based) therapies (45). As a result, it can help lessen the anabolic resistance frequently seen in aging muscles. Even though the pathophysiology of muscle aging and inactivity differ, they are frequently closely related. The reduction in their functional ability increases the elderly's tendency for falls and fractures, frequently resulting in immobilization (45). The use of NMES to facilitate the rehabilitation of older females following hip fracture surgery resulted in a quicker return to indoor mobility. In addition, a six-week follow-up evaluation revealed that NMES induced a longer-lasting effect on functional recovery, as evidenced by effectiveness in walking speed, postural stability, and muscle strength (3, 9, 21, 45).

Conclusion

NMES has been used in sports science to manage edema, maintain strength and muscle mass after prolonged immobility, and strengthen muscles. These effects have been achieved using a variety of stimulators, such as twin-spiked monophasic pulsed current, biphasic pulsed current, burst-modulated alternating current, or "Russian stimulator" stimulators.

Several studies have shown enhanced isometric muscular strength in NMES-stimulated and exercise-trained healthy, young individuals compared to unexercised controls, with no significant differences between the NMES and voluntary exercise groups. When NMES and voluntary activity are combined after training compared to either NMES or voluntary exercise alone, there does not appear to be a noticeable difference in muscle strength. NMES also improves functional performance in several strength activities.

Two mechanisms have been proposed to explain the NMES training effects. The first mechanism claims that the improvement of muscular strength by NMES happens similarly to increased muscle strength by voluntary exercise. This mechanism requires NMES strengthening programs to follow standard strengthening methods with few repetitions, high external loads, and high muscle contraction. The second mechanism argues that the muscular strengthening observed after NMES training is due to a reversal of the voluntary recruitment order and a selective increase in type II muscle fibers. Since type II fibers have a greater force than type I fibers, targeted augmentation of type II muscular fibers will boost the muscle's total strength. NMES has been widely explored to prevent muscle atrophy after knee ligament repair surgery or injury. NMES appears to protect against thigh muscle weakening, hypertrophy, and loss of oxidative capacity following knee immobilization.

In specific trials, NMES was found to be more effective at avoiding the atrophic alterations caused by knee immobilization than no exercise, isometric exercise of the quadriceps femoris muscle group, isometric co-contraction of the hamstrings and quadriceps muscle groups, and combination NMES-isometric exercise. Additionally, it has been found that NMES is supplied to the thigh muscles when the knee is immobilized during functional activities. NMES appears to strengthen muscles within muscle groups or sections of muscles selectively. Evidence has been shown for the selective strengthening of the abdominal muscles, back muscles, triceps brachii, and vastus medialis obliquus.

It is unknown whether this selective strengthening results from local changes in the stimulated muscle or muscular region or a change in the relative amplitude of recruitment of the different muscles within a muscle group or the different muscle sections. NMES has been recommended as a potential supplementary therapy for edema. Numerous studies have shown that monophasic pulsed stimulation pumps muscles to reduce acute edema. The last point is that it has been demonstrated that monophasic pulsed stimulation only affects acute edema when it is applied at amplitude values lower than those necessary to cause muscle contraction.

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