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Using Percutaneous Electrical Stimulation
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Two Theories of Muscle Strength Augmentation Using Percutaneous Electrical Stimulation

Electrical stimulation of muscle is a commonly used, well-substantiated strategy that physical therapists use to augment strength in patients with muscle weakness. Two distinctly different theories of strength augmentation using percutaneous muscle stimulation are presented. The first theory proposes that augmentation of muscle strength with electrically elicited muscle contractions occurs in a similar manner to augmentation of muscle strength with voluntary exercise. Electrically elicited muscle contractions of relatively high intensity with low numbers of repetitions strengthen muscle proportionally to the external load on the muscle in a manner that is equivalent to voluntary contraction. The second theory proposes that augmentation of muscle strength using percutaneous stimulation is fundamentally different from augmentation of strength with voluntary exercise. This theory uses the physiological differences between electrically elicited and voluntary contractions, such as the reversal of motor unit recruitment order, as a basis for argument. Both theories are partially substantiated using published literature. Strategies for testing both theories are also presented. [Delitto A, Snyder-Mackler L: Two theories of muscle strength augmentation using percutaneous electrical stimulation. Phys Ther 70:158-164, 1990]

Key Words: Electrotherapy, electrical stimulation; Exercise, strengthening; Muscle performance, general.

Neuromuscular electrical stimulation (NMES) is a well-substantiated clinical strategy to augment muscle performance. A recent review of the literature by Delitto and Robinson cites 25 references that deal specifically with experiments using NMES for muscle strengthening.1 The considerable number of studies reviewed incorporate a variety of designs and subject populations. In these studies, the operational definitions of muscle strength were consistent with that of Knutgen and Kraemer, who define muscle strength as the maximal force or torque that a muscle or muscle group can generate at a specified velocity.2 Methodologies range from randomized clinical trials to single-subject experiments, and subjects represent patient as well as healthy populations. Patient populations include individuals with major knee ligament injuries, patellofemoral disorders, and generalized knee injuries.3-9

Carry-over of muscle strength gains from NMES to functional performance improvements has some support in the literature. Wolf et al reported improvements in vertical-jump and 25-yard-dash performance of subjects involved in NMES and NMES-combined-with-voluntary-exercise regimens.10 Delitto et al reported substantial gains (up to a 20-kg increase) in squat, clean and jerk, and snatch weight-lifting performance after supplementing an elite weight lifter's weight training regimen with four weeks of high-intensity NMES.11 Experiments on the role of NMES in the augmentation of muscle strength converge to convincingly support its therapeutic efficacy. Theories that explain the improvements in subject and patient performance as a result of NMES regimens, however, are lacking.

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The purpose of this article is to present two theories regarding the mechanisms by which NMES may be operating to produce muscle strength augmentation. The first theory proposes that NMES augments muscle strength—via a mechanism similar to that involved in voluntary exercise—by presenting the muscle with an increased functional load, as measured at the tendon. Alternatively, NMES augments muscle strength because it targets and trains the type II muscle fiber more effectively than volitional exercise. We will use a literature review characterized by different interpretations of these mechanisms to present the merits of both proposed theories.

**Literature Supporting Increased Functional Load**

The first theory is that the mechanism for muscle strengthening using an electrically induced muscle contraction is the same as that of a voluntary muscle contraction and is only dependent on the load at the tendon, measured as external force or torque. That is, for the muscle to respond by increasing its contractile force capability, it must be stressed to a greater degree than it is used to being stressed. Usually forces of near-maximal voluntary contractile efforts are used for short durations (ie, high-resistance, low-repetition exercises). Voluntary muscle strengthening is purported to occur based on the principle of increased functional load, which states that in order to improve a function for which an individual is training (eg, muscle strengthening), it is necessary to expose the organism to a stress that is greater than the stress that is normally encountered during everyday life. Training regimens for muscle strengthening include low repetitions of high force. Force is usually expressed as a percentage of maximal voluntary contraction (MVC), which is some measure of force at the tendon or an analogous torque measure.

Many investigators have used NMES with functional overload in mind. We base this statement on the following observations: 1) NMES generators that produce bursted AC at a carrier frequency of 2,500 Hz, as first reported by Kots, were purported to allow for high training intensities with NMES that were more tolerable than with previous current forms; 2) Currier and Mann, McMiken et al, Laughman et al, and Selkowitz all used electrically elicited torque, expressed as a percentage of torque produced during an MVC, when describing their independent variable, analogous to the voluntary exercise literature in which training intensities are usually expressed as percentages of MVC; and 3) Currier (Dean P Currier, PhD; personal communication; February 1987) admits that his rationale for choosing to train his voluntary exercise group at 60% of MVC was based on the overload literature. In the following discussion, we will elaborate on these three observations.

The advent of new stimulus characteristics (bursted AC, or 2,500-Hz carrier waves interrupted at 50 bursts per second [bps]) and reports of their therapeutic efficacy by Kots caused renewed interest in electrical stimulation of muscle and its relationship to voluntary exercise. Kots reported being able to electrically elicit muscle contractions 10% to 30% greater than MVC. He also reported muscle strength gains of 30% to 40% after training subjects with this type of current.

Other investigations using similar current generators followed, with the stated intention of refuting or confirming the reports of Kots. In these investigations, as in those of Kots, increased functional load is strongly implied as the underlying mechanism of muscle strength augmentation by NMES. Currier and Mann assessed whether using 2,500-Hz current at 50 bps provided a greater overload stimulus to the muscle than previously used current forms. The following four groups were compared: 1) a group receiving both voluntary exercise and electrical stimulation simultaneously, 2) a group receiving electrical stimulation only, 3) a group receiving voluntary exercise only, and 4) a control group. No significant difference in isometric muscle strength values were noted among the experimental groups, although all experimental group muscle strength values were significantly higher than those of the control group. The conclusions of this study imply that exercise and NMES were equivalent training stimuli to the muscle. The addition of NMES given simultaneously with voluntary exercise did not increase the training response over electrical stimulation or exercise alone.

In their study, Currier and Mann documented the intensity of an electrically elicited muscle contraction, an important methodological technique that served to improve comparability between their study and subsequent studies. They expressed electrically elicited knee extension torque as a percentage of the torque produced during an MVC of the quadriceps femoris muscle. For the first time in the NMES literature, a standard was expressed that was analogous to the standard used in the voluntary exercise and muscle strengthening literature. In the group receiving electrical stimulation only, the training intensity (or dosage) of the electrically elicited quadriceps femoris muscle contraction was prospectively determined and was to be at least 60% of the torque the subject was able to elicit with an isometric MVC. This regimen parallels voluntary exercise protocols predicated on functional overload, which prescribe various training intensities (50%–100% of MVC) with relatively low repetition numbers (<10).

Prior to the work of Currier and Mann, virtually all of the studies involving NMES defined the treatment of NMES as adjusting the current intensity “to tolerance” without mention of contractile force or torque levels. This methodological flaw made comparison between studies impossible. Currier and Mann's technique of choosing a predetermined level of contraction to achieve with NMES served to better define the independent variable in their study as com-
pared with previous studies using NMES. In addition, other investigators reported training intensities with NMES protocols as a percentage of MVC using a technique similar to that of Currier and Mann, but they did not hold the percentage constant. The NMES in these studies was adjusted to subject "tolerance," but the intensity of the NMES-elicted muscle contractions was documented as a percentage of MVC. Together, these studies describe specific NMES dosages as a percentage of MVC, allowing for better comparison within each study as well as between studies.

Several researchers trained subjects using electrically elicited muscle contractions ranging from 33% to 91% of MVC. Studies testing healthy adults without muscle weakness found no significant difference in strength gains between voluntary exercise and electrical stimulation groups; both groups demonstrated significant gains when compared with control groups. In addition, Selkowitz reported a significant, positive relationship between muscle strength gains and NMES training intensity (expressed as a percentage of MVC).

Currier and Mann's finding of no added benefit to simultaneous NMES and voluntary exercise partially replicated an earlier work of Currier et al., except for the use of a different current form. Currier and colleagues rationale for adding NMES to a voluntary muscle contraction supports our claim that functional overload was implied in their studies. Currier et al. attempted to amplify the intensity of maximal voluntary muscle contractions by having subjects contract at maximal effort while simultaneously applying electrical stimulation to the quadriceps femoris muscles. Their hypothesis was that by using maximal effort voluntary contractions with superimposed NMES, the contraction intensity would be greater than with voluntary contractions alone. Greater contraction intensity translates to greater functional load and consequently to a greater strength training stimulus. The results of Currier and colleagues' studies did not support their hypothesis; there was no greater strength augmentation using simultaneous electrical stimulation and voluntary exercise as compared with voluntary exercise alone.

These findings parallel the voluntary exercise literature based on increased functional load. The major finding of all of the previously mentioned studies is that in 18- to 30-year-old, healthy people (mostly male college students), similarly applied programs of isometric exercise and electrical stimulation result in similar and predictable muscle strength gains. Coupled with the overwhelming conclusion from most authors that electrical stimulation offers equivalent muscle strengthening effects to voluntary exercise, NMES has been designated as merely an alternative form of exercise, exerting its strengthening effect in the same manner as explained in the well-described overload theory.

Problems with Increased Functional Load as an Explanation for Muscle Strengthening After Neuromuscular Electrical Stimulation

All previously mentioned studies incorporated comparison groups that used voluntary exercise protocols. Voluntary exercise and NMES groups trained in parallel with respect to using equal number of repetitions, duration of contractions, visits per week, and so on. One component of the treatment regimen was that not parallel between voluntary exercise and electrical stimulation groups, however, was training intensity. In some studies mentioned earlier, electrical stimulation groups used training intensities ranging from 33% to 91% of MVC. In all voluntary exercise groups, however, training intensity was set at 78% to 119% of MVC.

In the previously cited training studies, the authors concluded that NMES and voluntary exercise regimens provided equivalent muscle strengthening effects, even though a disparity between training intensities of voluntary exercise and electrical stimulation occurred. Laughman et al found it "paradoxical" that after six weeks of training, the electrical stimulation group, which trained at an average of 33% of MVC, tended to have larger muscle strength gains than the voluntary exercise group, which trained at an average of 78% of MVC. A recent study by Soo et al found an increase in isometric quadriceps femoris muscle strength with NMES training biweekly at only 50% of MVC. Strength gains as a consequence of voluntary exercise training regimens using training intensities of 50% of MVC cannot be found in the literature. Lai et al found significant increases in isometric and isokinetic muscle performance after 15 sessions of NMES, even in one group that trained at only 25% of MVC. Strength gains obtained from training at lower intensities using NMES as compared with volitional exercise would argue against increased functional load as the common underlying mechanism and strongly suggest the need for an alternative explanation for strength gains realized as a result of training with NMES.

A closer look at the literature shows substantial evidence that in populations other than the healthy male college students used in the previously cited studies, NMES and voluntary exercise do not result in equivalent outcomes. Most studies found significantly greater increases in muscle strength in subjects training with NMES as compared with voluntary exercise. Following an experiment involving a highly trained athlete, Delitto et al reported substantial gains in weight-lifting performance using electrical stimulation as an adjunct to the subject's ongoing weight training regimen. The single-case experimental design showed clear gains in performance as well as histochemical evidence of changes in the quadriceps
femoris muscle specific to the NMES regimen.

There is an abundance of evidence that electrical stimulation may produce greater muscle strengthening effects compared with similarly applied voluntary exercise in patients with muscle weakness. In a sample of patients referred for quadriceps femoris muscle strengthening, Godfrey et al showed that NMES improved isokinetic peak torque to a significantly greater degree than a voluntary exercise regimen. In a sample of patients who underwent major knee ligament surgery, Eriksson and Hagmark reported less observable atrophy and better muscle function (using an ordered rating scale) in the NMES group as compared with a voluntary exercise group. In patients with anterior cruciate ligament reconstructions, Delitto et al demonstrated electrical stimulation to be superior in isometric strength augmentation of quadriceps femoris and hamstring musculature as compared with a parallel program of voluntary exercise. Wigerstad-Lossing et al demonstrated that NMES in addition to a patient’s volitional exercise program resulted in diminished loss of quadriceps femoris muscle strength, increased size of type II muscle fibers, and less atrophy (as measured by cross-sectional area) than exercise alone.

Thus, a disparity exists in the NMES literature when comparing results obtained with healthy subjects with those obtained with patients. In healthy subjects, NMES has equivalent muscle strengthening effects as compared with voluntary exercise, but nonequivalent effects with patient populations. This disparity may be attributable to nothing more than methodological differences. For example, the findings based on patient populations are difficult to compare because training intensities of both voluntary exercise and NMES groups are not mentioned. Also, NMES groups could be exercising at a higher percentage of MVC than voluntary groups, in which case the results can be explained by functional overload. We believe, however, that this explanation is unlikely. The apprehension inherent with NMES coupled with the fact that any subject (patient or healthy person) will most likely be able to elicit greater muscle forces voluntarily as opposed to electrically argue against greater overload being obtained with NMES as opposed to voluntary exercise. Alternatively, we suggest that strength gains from NMES may result from something other than simply increasing the functional load on the muscle. We will explore this possibility in our discussion of the second theory.

### An Alternative View: Nonequivalent Strengthening Mechanisms Attributable to Physiological Differences Between Electrically Elicited and Voluntary Contractions

The shortcomings of overload as the sole basis for the muscle strengthening effects of electrical stimulation cause us to propose an alternative theoretical explanation. This theory is based on the well-established differences between voluntary and electrically elicited muscle contractions. Major differences between voluntary and electrically elicited muscle contractions are summarized in the Table. These differences combined demonstrate that electrically elicited contractions are different from volitional contractions at all levels of contraction.

In volitional isometric contractions, smaller motoneurons, which innervate type I (slow-twitch) muscle fibers, are activated before larger motoneurons, which innervate type II (fast-twitch) muscle fibers. Because smaller motoneurons have relatively few dendritic branches, branch-point failure is much lower than in the extensively arborized large motoneurons and larger excitatory postsynaptic potentials are produced. The firing thresholds of smaller-diameter motoneurons, therefore, are lower than larger-diameter motoneurons. Electrical stimulation activates the nerve fiber at or near the motor end-plate. Externally applied current (through the tissues) takes the path of least resistance and recruits more lower-resistance (larger-diameter) fibers than higher-resistance (smaller-diameter) fibers. Although this pattern of recruitment varies to some degree with the geometry of the nerve and electrode placements, it generally means that electrical activation is the opposite of Henneman’s size principle, which states the recruitment order within a motoneuron pool progresses from the smallest to the largest motoneuron. All of the motor units whose nerve fibers are originally activated by an electrical stimulus will be active throughout a contraction. They will be activated at the stimulation frequency and will contract until they fatigue.

Garnett and Stephens further point out that the afferent input from cutaneous stimulation results in inhibitory input to type I alpha motoneurons and in excitatory input to type II alpha motoneurons at the level of the spinal cord. The barrage of afferent input accompanying percutaneous NMES may also serve to selectively

<table>
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<tr>
<th>Voluntary</th>
<th>Electrically Stimulated</th>
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<td>Recruitment via size principle (type I precedes type II)</td>
<td>Type II preferential recruitment</td>
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<td>Asynchronous depolarization</td>
<td>Synchronous depolarization</td>
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<tr>
<td>Intermittent, lower firing rates</td>
<td>Constant, higher firing rates*</td>
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*Dependent on frequency set on current generator.
activate fast-twitch motor units at the spinal cord level.

These differences are further substantiated by experimental evidence suggesting that electrical stimulation preferentially affects the type II muscle fibers. Sinacore and colleagues (David R Sinacore, Anthony Delitto, Douglas S King, Steven J Rose; unpublished research; June 1987) performed a single session of 50 repeated electrically elicited contractions (five seconds on, two seconds rest) of the quadriceps femoris muscle. They found histochemical evidence of selective glycogen depletion of the type II fibers as compared with type I fibers. Kabić and colleagues studied the effect of a 19-day NMES training regimen on the morphology of the gastrocnemius muscle.29 Muscle fiber size was increased, as was nuclear volume. An increase in the mitochondrial fraction, the size of single myonuclei, and a decrease in their heterochromatin fraction were significantly more pronounced in the type II fibers than in type I fibers. The authors concluded that NMES predominantly affects the type II fibers. Wigerstad-Lossing et al found an increase in type II fiber area and no change in oxidative or glycolytic enzyme activity when comparing their experimental group with a control group that exercised volitionally.30 Duchateau and Hainaut, in their study of submaximal electrical stimulation, found that volitional exercise increased the muscle's resistance to fatigue, whereas NMES did not.31 These findings suggest that submaximal electrical stimulation does not train the fatigue-resistant type I fibers to any significant degree, but rather preferentially trains the type II fibers that may not be fully activated during submaximal volitional contraction.

In large muscles (deltoid and larger), recruitment of motor units continues to at least 90% of MVC and perhaps to 100% of MVC.30,31 Recruitment appears to be the predominant mechanism by which force is increased in these muscles. Conversely, in small muscles, such as those in the hand, recruitment appears to be complete by approximately 50% of MVC.30,32 Further increases in force are the result of rate coding (an increase in the firing rate from frequencies in the 10-pulses-per-second [pps] range to those in the 50-pps range). Firing rates of up to 60 pps have been reported.33,34

Firing rates tend to be lower (or change less) in the higher-threshold fibers. This tendency means that the type II fibers do not usually fire as frequently as type I fibers, even at high force levels. The larger muscles have a relatively constant peak firing rate of approximately 25 pps across motor unit types; rate coding does not appear to play a significant role in their increases in force generation.33,34

Let us examine what happens as a subject begins and then sustains a high-force, voluntary quadriceps femoris muscle contraction of 10 seconds' duration. First, type I fibers are activated at lower firing rates. Then, type II fibers are activated and type I fibers increase their firing rates. Type II fibers fire at their activation rates for the duration of the contraction.35,36 This activation rate has been reported to be as low as 7 to 12 pps. Recruitment persists throughout the increase in force. Both Kanosue et al31 and De Luca et al35 concluded these findings suggest that the type II fibers are most likely not producing the maximal force that they are capable of producing, even at these high contraction intensities. De Luca suggests that this relatively low firing rate in type II fibers indicates an untapped potential for muscle to generate force.35

Electrically elicited contractions cause fused contractions of all activated fibers, usually at frequencies of 35 to 50 pps, frequencies far above both critical fusion frequency and the normal firing rate for these fibers. If large-diameter fibers are activated first, then higher-force-generating capabilities are theoretically possible using NMES as compared with volitional contraction given the "untapped potential" suggested by De Luca.35

This potential force-generating capability of the unfused type II fibers helps to explain the high torque levels found by both Delitto et al31 and Selkowitz38 in their studies of healthy subjects. It also can be helpful in explaining why NMES is so much more effective for strengthening the muscles in patients with muscle weakness, given the evidence that many patient conditions involving muscle weakness are attributed to significant type II muscle atrophy.50

Physiological differences between the two types of contractions can be argued to be an advantage of electrically elicited muscle activation for augmentation of muscle strength. If reversal of activation does indeed occur, then NMES may be a more efficient means than voluntary exercise of training the largest fast-twitch motor units in muscle. That is, given equal contraction levels (%MVC), there will be more large fast-twitch motor units activated with an electrically elicited muscle contraction than with a voluntary contraction. Greater activation of type II fibers supports the use of electrical stimulation as opposed to voluntary exercise in diminishing the deleterious effects of disease and immobilization in patients.

Near-maximal volitional effort is needed to elicit contraction in the largest, fastest motor units. This level of effort is difficult, if not impossible, for some patients (eg, postsurgically). The finding of superior muscle strength gains with NMES as compared with voluntary exercise in patients with muscle weakness can be explained by this theory, particularly when considering the preponderance of patient populations that display predominant or significant type II muscle fiber involvement.56

**Testing the Two Theories**

One experimental design that may provide supporting evidence for either theory would be to train subjects with NMES and voluntary exercise with parallel regimens, including similar training intensities. Both NMES and voluntary exercise groups would train at, for example, 30%, 50%, and
70% of MVC for 10 contractions, three times per week for four weeks. A pretest-posttest design would be used. The Figure illustrates two contrasting results, each of which supports one theory. If functional overload is the primary explanation for increases in muscle strength, parallel increases from pretest to posttest strength tests would be expected at all training intensities for both groups (Fig. 1A). In contrast, a significant interaction between the groups should be easily identified if the second theory best explains the muscle strengthening effects of NMES (Fig. 1B), with the greatest differences being at lower levels of training intensity.

Although the Figure depicts training intensity and muscle performance gains as linear functions, other functions would likely replace the linear function. Regardless of the shape of the function curve, with the first theory there will be a parallel result with the comparably trained electrical stimulation and voluntary exercise groups. In the second theory, the result will not be parallel; rather, a significant interaction will be expected to occur.

Another technique to investigate these proposed NMES theories is to electrophysiologically examine, via muscle decomposition or muscle conduction velocity studies, firing patterns of individual motor units during electrical stimulation. Using these techniques on healthy as well as atrophied human muscle may help answer questions concerning selective recruitment with NMES of type II fibers. More invasive procedures may be required for other investigative designs to further differentiate these theories. Replication of the preliminary work of Sinacore and colleagues (unpublished research) would serve as an excellent example of such an investigation.

**Conclusion**

Establishing the underlying mechanisms of therapeutic interventions is a major goal in any science. The theories proposed in this article are by no means exhaustive. Our purpose was not to give an exhaustive account explaining the effect of electrical stimulation on the augmentation of muscle strength. Instead, we hope that this dialogue will provoke further discussion that will either clarify the proposed theories or provide solid, logical alternative explanations for electrical stimulation as a means of augmenting muscle strength. Furthermore, we hope that future research concerning NMES will be enhanced by providing investigators with sound theoretical foundations on which to base experimental designs. Finally, we hope a sound theory explaining NMES and its effect on strengthening muscle will enable clinicians to use this modality most effectively with their patients.

**Acknowledgments**

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**Figure.** Muscle performance score (eg, strength score) and training intensity in percentage of maximal voluntary contraction (%MVC). Theory 1, which emphasizes increased functional load where parallel increases in muscle performance between voluntary exercise and neuromuscular stimulation occur across training intensities, would be supported by illustration A. Theory 2, which emphasizes the physiological differences between electrically and voluntarily elicited contractions, would be supported by illustration B. As compared with voluntary exercise, electrical stimulation provides a proportionally greater strengthening stimulus to muscle across training intensities, with the greatest effect at lower intensities.

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