Chronic Motor Dysfunction After Stroke
Recovering Wrist and Finger Extension by Electromyography-Triggered Neuromuscular Stimulation

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Background and Purpose—After stroke, many individuals have chronic unilateral motor dysfunction in the upper extremity that severely limits their functional movement control. The purpose of this study was to determine the effect of electromyography-triggered neuromuscular electrical stimulation on the wrist and finger extension muscles in individuals who had a stroke ≥1 year earlier.

Methods—Eleven individuals volunteered to participate and were randomly assigned to either the electromyography-triggered neuromuscular stimulation experimental group (7 subjects) or the control group (4 subjects). After completing a pretest involving 5 motor capability tests, the poststroke subjects completed 12 treatment sessions (30 minutes each) according to group assignments. Once the control subjects completed 12 sessions attempting wrist and finger extension without any external assistance and were posttested, they were then given 12 sessions of the rehabilitation treatment.

Results—The Box and Block test and the force-generation task (sustained muscular contraction) revealed significant findings (P<0.05). The experimental group moved significantly more blocks and displayed a higher isometric force impulse after the rehabilitation treatment.

Conclusions—Two lines of evidence clearly support the use of the electromyography-triggered neuromuscular electrical stimulation treatment to rehabilitate wrist and finger extension movements of hemiparetic individuals ≥1 year after stroke. The treatment program decreased motor dysfunction and improved the motor capabilities in this group of poststroke individuals. (Stroke. 2000;31:1360-1364.)

Key Words: motor activity ■ rehabilitation ■ stroke assessment ■ stroke management

Voluntary movement control is typically impaired after a stroke. Movement control of the body on the contralateral side of the brain lesion proceeds through stages of recovery in which the sensory and motor function are often reestablished abnormally.1,2 In the upper extremity, after a period of flaccidity, a common course of recovery includes the development of an uncontrolled flexion synergy. This pathological synergy is observed in the hemiparetic limb during efforts to use the arm for functional tasks. Individuals with this uncontrolled flexion synergy have great difficulty isolating joint movements out of synergy.2 Indeed, control of wrist and finger extensors is a challenging aspect of upper-extremity recovery.

Residual dysfunction in the hemiparetic limb is frequently observed for extended periods, plateauing in ≈12 months. Moreover, 60% of the poststroke individuals continue with residual motor dysfunction as a long-term disability after the first year.1 These chronic motor problems lead to difficulty in the execution of functional movements (eg, picking up a glass of water or buttoning a shirt) in poststroke individuals. The severity of these motor impairments and their negative impact on function are reasons that Trombly3 encouraged researchers to investigate movement dynamics after stroke.

As the months after stroke accumulate into years, individuals typically accept the chronic motor problems and attempt to compensate for the losses. Wolf et al4 argued that individuals with upper-extremity motor problems display behaviors that indicate learned nonuse. The affected arm is not used for any voluntary movements, whereas the unaffected arm attempts to execute all of the motor actions required for daily living. Consequently, chronic motor problems that are observed from the first year after stroke could lead to learned nonuse as individuals stop trying to voluntarily move their affected upper extremity.

Neurological Mechanisms
The specific neurological mechanisms that mediate the neuromuscular recovery process after a stroke are not completely

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understood. One specific mechanism, spontaneous neural reorganization, has generated considerable debate and research. Stein stated that compensation and substitution of motor function for the unilateral motor dysfunctions may come about by intensive experience-induced reorganization of neuronal activity. Evidence suggests that some motor recovery occurs because the auxiliary cortex areas may take over functions. Two lines of research strongly support this statement: (1) Taub and Wolf reported convincing behavioral evidence from the forced-use paradigm, and (2) Jenkins and Merzenich argued that cortical activity reorganizes with training and experience.

Theoretically, the basis for the electromyography (EMG)-triggered neuromuscular electrical stimulation assistance is that alternative motor pathways can be recruited and activated to assist the stroke-damaged efferent pathways. This theoretical explanation is based on sensorimotor integration theory, which states that sensory input from movement of the affected limb directly influences subsequent motor output. As poststroke individuals voluntarily attempt to extend their affected wrist and fingers, the EMG-monitored neuromuscular stimulation assists the movement, and full extension is experienced. Thus, this study was conducted to evaluate a rehabilitation procedure for stroke individuals who have chronic motor dysfunction. The purpose was to determine the effect of EMG-triggered neuromuscular electrical stimulation on voluntary motor control of the wrist and finger extension muscles.

Subjects and Methods

Subjects
Eleven subjects ≥1 year after stroke with chronic upper-extremity impairments were recruited. The 6 women and 5 men had a mean age of 61.64 years (SD=9.57) and an average time after stroke of 3.49 years (SD=2.56), and 10 subjects had a right hemisphere stroke. Exclusion/inclusion criteria included an upper-limit cutoff point of 75% motor recovery and a lower limit requirement that subjects be capable of voluntarily extending the wrist 20° against gravity from a 90° flexion position. All participants were free of other neurological deficits and were not currently enrolled in rehabilitation therapy.

Pretest-Posttest Instruments and Procedure
Before testing, each participant read and signed an informed consent. Participants were randomly assigned to either an experimental group (n=7) or a control group (n=4). Motor functions were evaluated with 5 dependent measures.

Clinical Measures
Three clinical tests were administered. The Box and Block timed manipulation test is a manual dexterity test with norms established for age groups. The test involved grasping a 2.54-cm-square wooden block with one hand, transporting it to the other side of the box, releasing it, and repeating the procedure for 60 seconds. The goal was to move as many blocks as possible. Both the Motor Assessment Scale and the Fugl-Meyer test were used to evaluate the functional recovery of the hand and wrist/finger movements after stroke. These tests were administered by a physical therapist.

Laboratory Measures
The 2 force-generation tasks (reaction time [RT] and sustained muscle contraction) were consistent with Cramer et al., who advocated the use of computerized measures to observe motor recovery. The same instrumentation was used for both tasks. A 25-lb load cell measured the amount of force generated during the isometric wrist extension movements. Force signals were recorded online by a BioPac hardware data acquisition system and AcqKnowledge Software on a Macintosh computer.

EMG activity was recorded with surface electrodes (silver–silver chloride electrodes, 1 cm in diameter and 2 cm apart with an epoxy-mounted preamplifier). A standard procedure was followed in attaching the electrodes to the dominant muscle area for the extensor communis digitorum and extensor carpi ulnaris muscles of the affected limb. For the RT task, the sampling rate for the EMG and force signals was 800 Hz for 6 seconds. Subjects were instructed to respond as quickly as possible to the onset of an auditory stimulus by initiating the wrist and finger extensor muscles for an isometric contraction against the platform of the load cell. To prevent stimulus onset anticipation, 4 variable foreperiods were randomly presented. A digital LCD clock was used to monitor foreperiod intervals and the 20 seconds between trials.

For the sustained-contraction task, subjects were instructed to gradually increase their wrist/finger extension force to a maximal isometric contraction and hold that level for 5 seconds. This input was collected for 8 seconds, with 20 seconds of rest between trials. The 2 force-generation tasks were tested in random order, with 5 minutes of rest between tasks.

Rehabilitation Training Instrument and Procedure
Before each treatment session, a trainer performed general passive range-of-motion activity with the hemiparetic arm, followed by short periods of gentle stretching to the wrist and finger flexors. The affected wrist was placed in ~10° of flexion to begin training. The electrical activity of the extensor communis digitorum and extensor carpi ulnaris muscles was monitored with surface electrodes (diameter 50 mm). Given the size of the 3 surface electrodes, exact placement was achieved by electrically stimulating a synergistic group of muscles on the back of the forearm until pure wrist and finger extension was observed. Subjects were instructed to initiate wrist/finger extension so that a target threshold level of EMG activity was voluntarily achieved, which triggered the neuromuscular electrical stimulation to assist the muscles to reach a full range of motion. Three practice trials were completed to familiarize participants with the isotonic muscle activation pattern and establish a target threshold. Successful trials were achieved when participants produced voluntary muscle activity of a sufficient level to trigger the stimulation unit (1 second ramp up, 5 seconds of biphasic stimulation at 50 Hz, and 1 second ramp down). The electrical stimulation ranged from 14 to 29 mA.

The target threshold stimulation levels were automatically adjusted on each successive trial according to the voluntary activity produced. If the threshold level was easily met on the previous trial, then the unit was programmed to automatically move the target level slightly higher. If the threshold level was not met, then the Automove unit (AM 800) automatically adjusted the level of threshold closer to the amount of activity that the individual could produce. A 25-second rest period followed each successful trial.

Two treatment sessions of 30 successful movement trials (~60 minutes) were performed 3 days per week for 2 consecutive weeks. Subjects completed 12 treatment sessions for a total of 360 neuromuscular electrical stimulation trials.

The control group followed the same procedure as the experimental group except that they did not receive the neuromuscular electrical stimulation. The hemiparetic limb was moved through a range of motion and stretched, then subjects tried to voluntarily lift their wrist for 2 sessions of 30 trials. Once the control subjects completed the initial 12 sessions and were posttested, they performed 360 full wrist/finger extension trials supplemented with EMG-triggered neuromuscular electrical stimulation.

Design and Analyses
This experiment was a randomized clinical study conducted in a field setting. To avoid the problem of withholding the treatment from the control group, a modified crossover design was used that permitted
Results
All of the findings below include the pooled experimental treatment data. For the control participants of the crossover design, the first posttest was compared with the second posttest (after the neuromuscular treatment program). The motor recovery analyses begin with the results of the 3 laboratory measures. The premotor and total RT analyses did not identify any significant differences in the main effects or interactions. The motor RT analysis indicated a trend for the trial block interaction, $F(1, 44) = 2.27, P < 0.08$. The experimental group showed faster motor RTs at the fourth trial block than the control group.

Sustained Muscle Contraction Task
The force impulse values for the sustained contraction task were analyzed in a mixed-design group×test session×trial block×area (2×2×4×3) ANOVA with repeated measures on the last 3 factors. The analysis revealed a significant test session×group interaction, $F(1, 10) = 6.09, P < 0.04$. Figure 2 shows the different patterns of impulse change for the experimental and control groups across the pretest and posttest. This effect accounted for 7% of the total variance. Figure 3 illustrates the raw force and EMG patterns for a treatment group subject. Note the distinct differences between the test sessions for the force values and the plateau pattern from the pretest (top) to the posttest (bottom).

Discussion
Given that wrist and finger extension control is one of the most difficult motions to regain after a stroke and is a key precursor for prehensile activity, loss of this capability is a primary disabler for hand function. Frequently, prehensile motion and wrist/finger extension movements serve as mark-
by Chae et al, and the present study tested chronic stroke patients (>1 year after stroke). Granted, in the present study, the improvements found in the force-generation sustained muscle contraction task were not strong enough to be significant in the Motor Assessment Scale or Fugl-Meyer test. However, the ordinal scales of these 2 motor recovery tests may not be sensitive enough to detect subtle improvement changes in isolated extension movements. There was a distinct positive trend in the motor recovery functional tests with only 6 hours of EMG-triggered neuromuscular electrical stimulation.

Nevertheless, a theoretical question about the mechanism still abounds: What mechanism does the EMG-triggered neuromuscular stimulation activate that could explain the improved Box and Block scores as well as the increased integrated force impulse values? Of course, a change in the paretic muscle is one part of a possible explanation. However, the restricted treatment time involved and the short training program suggest that a muscle training explanation has limitations. As reviewed by Sale, a significant increase in muscle hypertrophy has not been shown to occur in a 2-week time frame.

Another viable explanation involves sensorimotor integration theory. The voluntary initiation of the electrical activity in the wrist extensor muscles served as a stimulus for the onset of the electrical stimulation. The sensorimotor aspects of this combined movement are closely intertwined. That is, the slight increases in the electrical activity of the muscles that have been dormant since stroke onset trigger the external, supplemental neuromuscular stimulation, and the wrist/fingers move through extension. These movements produce proprioceptive feedback, an afferent signal that returns to the somatosensory cortex, completing the sensorimotor cycle. The voluntary efferent output as well as the afferent input may assist in organizing the distorted signals arising from the damaged brain area. Indeed, Ghez and colleagues have argued that proprioceptive feedback acts in a critical role in motor planning by updating an internal model of the state and properties of the limb.

Further confirmation of the sensorimotor integration theory comes from animal studies. Xerri et al reported that monkeys generated new cortical representations after microlesions destroyed specific regions of the somatosensory cortex. The reemergence of fingertip representations once the monkeys reacquired previously learned finger manipulation movements was interpreted as substantial evidence supporting the integration of sensorimotor signals. This interpretation is consistent with brain plasticity studies in humans during motor skill learning. Researchers have argued that cortical plasticity after a stroke goes through similar alterations that have been observed during motor skill learning in normal uninjured brains.

Moreover, multiple representation and redundancy in the system may underlie motor recovery after a stroke. This
explanation takes advantage of the functional equivalence that underlies movements. Functional equivalence refers to the capability of the motor system in achieving a movement goal through multiple routes. Interactions in the sensorimotor system may achieve a retroactivation of motor commands based on proprioceptive feedback activity from convergent regions. Thus, the basis for sensorimotor integration theory as a mediating mechanism in motor recovery for poststroke individuals is appealing. Moreover, Wagenaar and Van Emmerik suggested relearning motor actions on a perception and action basis as a promising approach to characterizing movement disorders.

In summary, use of EMG-triggered neuromuscular electrical stimulation to the wrist and finger extensors of individuals with chronic hemiparesis due to stroke resulted in significant improvements for grasping small objects and for sustaining extensor contractions. These findings suggest that neuromuscular-triggered electrical stimulation is a beneficial adjunct to the rehabilitation of hand function after chronic stroke.

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References
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