Effect of EMG-triggered Electrical Stimulation in Patients with Chronic Hemiplegia

Young-Hee Lee*, Yang Tark Lee, Kyung Hee Park, Sung Hoon Kim, Sang Min Jang, Tae Ho Kim, Myoung Yae Lee

Introduction

Following a cerebrovascular accident (CVA) and traumatic brain injury (TBI), any spontaneous recovery of the upper limb motor function is generally limited to the first six months. During this period, the motor recovery has been reported to be enhanced, beyond that attained by conventional therapy, rehabilitation techniques, including neurofacilitatory physical therapy, EMG bio-feedback and positional feedback, with electrical stimulation. However, there is a consensus that the current rehabilitation techniques are less effective in improving upper limb motor function in a chronic brain lesion (>6 months). As the months after a stroke accumulate into years, individuals typically accept the chronic motor problems, and attempt to compensate for their losses. Wolf et al[1] argued that individuals with upper-extremity motor problems display behaviors that indicate learned nonuse. An affected arm is not used for any voluntary movements, whereas an unaffected arm will attempt to execute all of the motor actions required for daily living. So, control of upper extremity functions, such as wrist and finger extensors, are a challenging aspect in upper extremity recovery. In this study, attempts were made to identify the effect and mechanism of EMG-triggered electrical neuromuscular stimulation for the recovery of hemiplegic arm function.

Methods

Eight subjects, 6 ≥ 1 year after stroke, and 2 with TBI, with chronic upper-extremity impairments, were recruited. All the subjects were male, with a mean age of 40 years (SD=5.8) and an average time after brain lesion of 26 months (SD=19). Six of the subjects had left hemisphere brain lesions, whereas the other two had right hemisphere brain lesions.

Both before and after treatment, six clinical tests were administered to evaluate the effects of the treatment. The Box and Block timed manipulation test, Fugl-Mayer (FM) score and functional independence measure (FIM), were used to evaluate the functional recovery in hand and wrist/finger movements after the brain lesion. A modified Ashworth scale (MAS) was used to evaluate the spasticity. The motor free visual perception test (MVPT) and Loewenstein occupational therapy cognitive assessment (LOTCA) were used to evaluate the perceptual and cognitive function. A quantitative EMG, from the extensor digitorum communis (EDC) muscle, and an excursion of the 2nd metacarpophalangeal joint, using the MP150 ® system (BIOPAC systems Inc.), were administered to evaluate the kinesiologic function.

Using an Automove AM 800 ® (danmeter, Odense), the EMG-triggered electrical stimulation was applied to the EDC muscle of the chronic hemiplegics showing no functional changes for more than 3 months. The stimulation was started when the amplitude of the processed EMG signal, from the same muscle, exceeded a preset threshold. One 60 minute treatment session was performed 5 days per week, for four consecutive weeks. The effects were clinically evaluated. The quantitative EMG, from the EDC muscle during volition, was also evaluated before and after treatment.

In two subjects, one showing, and the other not showing, voluntary finger movement prior to treatment, functional MRI and magnetic evoked potential (MEP) brain mapping were used to evaluate the brain plasticity.
Results

The subjects treated with EMG-triggered electrical stimulation showed significant gain in the amplitude of the quantitative EMG and excursion sums, during maximal exertion, comparing to those prior to treatment (p<0.05)(Figure 1). There was also a decrease of spasticity after treatment, but the functional, perceptual and cognitive measurements were not changed significantly (p>0.05). The MEP mapping and functional MRI showed increases in the motor output area sizes and activated motor cortices in both hemispheres, after four weeks of EMG-triggered electrical stimulation, in a subject that showed voluntary finger movement prior to treatment (Figure 2).

Figure 1. Quantitative EMG from EDC (RMS) and excursion sums of the 2nd MCP joint during voluntary hand movement.
* p<0.05

Brain mapping

Figure 2. MEP Mapping showed an increase in the motor output area sizes in both hemispheres after 4 weeks of EMG triggered electrical stimulation
Discussion

Given that wrist and finger extension control is one of the most difficult motions to regain after a brain lesion, and a key precursor for prehensile activity, the loss of this capability is a primary disabler of hand function. Frequently, the prehensile motions, and wrist/finger extension movements, serve as markers for therapeutic intervention. In a related study on EMG-triggered neuromuscular electrical stimulation, Chae et al.[2] provided acute stroke patients with 15 days (1 hour per day) of treatment. The wrist and finger extension neuromuscular stimulation group demonstrated greater gains in their FM scores, following treatment, than the control group. The motor recovery found in their acute stroke population was dramatic compared with our study group of stroke survivors, with well-defined unilateral motor dysfunctions that had accumulated over many years. The stage of motor recovery is an important distinction between these studies: Chae et al.[2] used acute stroke patients (<1 year after stroke) in their study, whereas the present study tested patients with chronic brain lesions (>1 year after brain lesion). Admittedly, in the present study, there were significant gains in the amplitude of quantitative EMG and the excursion sums during maximal exertion comparing to those prior to treatment. There was also a significant decrease of spasticity after treatment. However, the functional, perceptual and cognitive measurements were not significantly changed.

Nevertheless, a theoretical question about the mechanism still remains: What mechanism does the EMG-triggered neuromuscular stimulation activate that could explain the improved spasticity and kinesiologic values? However, the restricted treatment times involved, and the short training programs, suggest that a muscle training explanation has limitations. As reviewed by Sale[3], no significant increases in muscle hypertrophy, within a 2-week time frame have been shown to occur.

Another viable explanation involves the sensorimotor integration theory. Xerri et al.[4] reported that monkeys generated new cortical representations after microlesions had destroyed specific regions of the somatosensory cortex. The re-emergence of fingertip representations, once the monkeys reacquired previously learned finger manipulation movements, was interpreted as substantial evidence supporting the integration of the sensorimotor signals. This interpretation is consistent with brain plasticity studies in humans during motor skill learning. Researchers have argued that cortical plasticity, following a stroke, goes through similar alterations to those that have been observed during motor skill learning in normal uninjured brain. In our study, there were increases in the motor output area sizes and activated motor cortices in both hemispheres, after four weeks of EMG-triggered electrical stimulation, in a subject that had shown voluntary finger movement in the MEP mapping and functional MRI prior to treatment.

In summary, the use of EMG-triggered neuromuscular electrical stimulation, to the EDC muscle, in individuals with chronic brain lesions, due to a stroke or TBI, resulted in significant improvements in the spasticity and kinesiologic values. There was also functional radiological and physiologic evidence of central nervous system reorganization in a case that showed a marked improvement in finger motion, which might explain the mechanism of effect as being one of brain plasticity. These findings suggest that neuromuscular-triggered electrical stimulation is a beneficial adjunct to the rehabilitation of hand function following a chronic brain lesion.

References