Neuromuscular electrical stimulation (NMES) is used in physical rehabilitation in three ways: diagnostically, functionally, and therapeutically. Diagnostically, electrical stimulation is applied to nerves and muscles to measure various responses and assist in determining their neurophysiological state. Functionally, electrical stimulation is applied to the nerve or motor point of the muscle to mimic natural muscle contraction, producing movement in persons with central nervous system lesions (Vodovnik et al., 1988). Therapeutically, electrical stimulation has been used to reduce involuntary or spastic contraction in spinal muscles (Bajd, Gregoric, Vodovnik, & Benko, 1985; Franek, Turczynski, & Opara, 1988; Vodovnik, Bowman, & Hufford, 1984) and in muscles affected by spinal cord injury or stroke (Baker, Yeh, Wilson, & Waters, 1979; Douglas, Walsh, Wright, Creasey, & Edmond, 1991; Levine, Knott, & Kabat, 1952; Stefanovska, Gros, Vodovnik, Rebersek, & Acimovic-Janexic, 1988); to increase voluntary movement in paretic muscles after a stroke, spinal cord injury, or head injury (Baker, Parker, & Sanderson, 1983; Bowman, Baker, & Waters, 1979; Cozean, Pease, & Hubbell, 1988; Liberson, Hoinquest, Scott, & Dow, 1961; Packman-Braun, 1988; Robinson, Kett, & Bolam, 1988a, 1988b; Teng, McNeal, Kralj, & Waters, 1976); and to increase strength in muscles without dysfunction (Kramer & Mendryk, 1982; Laughman, Youdas, Garrett, & Chao, 1983).

Spasticity, or involuntary contraction of muscles, can limit a person’s ability to perform functional tasks. I have observed that therapists use techniques such as passive stretch (usually done through splinting) to restore muscle tone, and more recently, occupational therapists have used electrical stimulation to reduce tone. The most common application of electrical stimulation is to the antagonist, or nonspastic, muscle. Stimulation of the antagonist muscle makes use of the afferent nerve pathways, which, polysynaptically, inhibit the agonist or spastic muscle (Alon & DeDominico, 1987; Levine et al., 1952). However, the least common application of NMES is to nonantagonist, or spastic, muscles, and few data have appeared in the literature regarding the effects of this application. Is the use of NMES on spastic muscles to reduce tone a viable option to consider? This article compares the merits of maintained passive stretch and NMES applied during a single 10-min treatment session in decreasing resting tone in spastic wrist flexor muscles of 21 persons with stroke.

NMES is a “physical agent modality” (American Occupational Therapy Association [AOTA], 1991a). The AOTA’s official statement on the use of physical agent modalities states that they may be used “as an adjunct to or in preparation for purposeful activity to enhance occupational performance” (AOTA, 1991b, p. 1075).
Method

Subjects

Twenty-one subjects (14 men and 7 women) with chronic wrist flexor spasticity as a result of cerebrovascular accident were recruited for the pilot study through occupational therapists working in four adult rehabilitation programs in the Milwaukee area. The only requirement was the ability to understand the evaluation and treatment to be given. Their ages ranged from 59 years to 72 years, with a mean age of 67 years. Subjects were randomly assigned to two treatment groups: passive stretch (n = 10) or NMES (n = 11).

Instrument

To measure tone of the wrist flexor muscle group, a torque meter developed by McPherson (1986) was used. This device assesses the static muscle tone as "the passive force exerted by the muscle to return to its resting length from a specified extended position" (p. 42).

Procedure

Before treatment, the subjects' affected wrists were placed in the torque meter and passively extended until the meter read 15 cm-kg (see Figure 1). This was the amount of passive resistance exerted by the wrist flexors and was used to standardize maintained passive stretch for all the subjects. While this angle of wrist extension was maintained, the subjects received 10 min of either continued passive stretch or NMES treatment of the wrist flexor muscle group. For both groups, wrist extension was maintained to enhance stretch of the wrist flexor tendons and thus increased the responsiveness of the neurotendinous spindle. Resistance of wrist flexors to passive movement was measured with the torque meter after treatment at the same angle of wrist extension measured before treatment.

For the NMES group, an electrode of moderate size (1.5 in. to 2.0 in. in diameter) was placed intermediately on the proximal volar surface of the forearm, and the indifferent electrode was placed on the distal volar surface of the forearm over the flexor tendons (see Figure 2). The parameters for the electrical stimulation were synchronous mode, 45 Hz pulse rate, 250 μs pulse width, ramp up/down time 3/0 sec, on/off time 10 sec, and amplitude 15 mA to 20 mA. Stimulation with these parameters over a 10-min period resulted in an involuntary contract–relax motion of the spastic wrist flexor muscle group.

Results

Decrease in resistance of the wrist flexor muscle group to passive stretch for each subject is shown in Table 1. The mean decrease in resistance is 4.6 cm-kg for the group receiving only passive stretch and 9.6 cm-kg for the group receiving NMES.

An independent t test indicated that the mean decrease in resistance for the subjects receiving NMES was significantly greater than that for the subjects receiving only passive stretch.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Group A (10-Min Passive Stretch)</th>
<th>Group B (10-Min NMES)</th>
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</table>

Note. NMES = neuromuscular electrical stimulation

* M = 4.6 cm-kg, SD = 1.955

* M = 9.6 cm-kg, SD = 1.968
only maintained passive stretch ($t = -8.0896, p < .001$).

Discussion

This study demonstrates the decreased resistance to passive stretch of the spastic wrist flexor muscle group after either passive stretch or NMES treatment. However, the effect is significantly greater after NMES than after passive stretch.

According to Alon and DeDominico (1987), stimulation of the spastic muscle may inhibit its excitation due to muscle fatigue or autogenic inhibition through increased response of the neurotendinous spindle (golgi tendon organ). Autogenic inhibition describes the disynaptic inhibitory effect of the afferent fibers from the neurotendinous spindle stimulated by increased tension or stretch to the tendon of the electrically stimulated muscle (Alon & DeDominico, 1987). Robinson and colleagues (1988a, 1988b) stated that the decrease in spasticity noted in their subjects after 20 min of electrical stimulation might be explained by muscle fatigue. Research has demonstrated the alternate ways that the golgi tendon organ may react in relation to supraspinal influences, but in some situations (i.e., excessive muscle tension and muscle fatigue), autogenic inhibition occurs on a reflex level (Moore, 1984).

This study demonstrated that NMES is effective in reducing abnormally increased tone in wrist flexors. Future studies might address the use of this technique on other persons with increased tone involving different muscle groups. Furthermore, longer term effects of passive stretch and NMES need to be examined.

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References


