Techniques to Improve Function of the Arm and Hand in Chronic Hemiplegia

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ABSTRACT. Kraft GH, Fitts SS, Hammond MC. Techniques to improve function of the arm and hand in chronic hemiplegia. Arch Phys Med Rehabil 1992;73:220-7.

• We evaluated functional improvement in the upper limb of chronic (more than six months' duration) stroke patients who received one of two electrical stimulation treatments, conventional treatment, or no treatment. Twenty-two right-handed patients were assigned to one of four groups studied for 12 months posttreatment. Subjects received (1) EMG-initiated electrical stimulation of wrist extensors (EMG-stim), (2) low-intensity electrical stimulation of wrist extensors combined with voluntary contractions (B/B), (3) proprioceptive neuromuscular facilitation (PNF) exercises, or (4) no treatment. Subjects were treated for three months. Before treatment, upon completion of treatment, and three and nine months after treatment, subjects were evaluated by the Fugl-Meyer (FM) poststroke motor recovery test and by grip strength. Subjects also attempted three Jebsen-Taylor hand function tests and a finger tapping test at the same evaluation sessions, but many were unable to complete these tests. During the course of treatment, FM scores of subjects receiving PNF improved 18%, B/B improved 25%, and EMG-stim improved 42%. The aggregate FM improvement of the treated groups was significant from pretreatment to posttreatment, and the improvement was maintained at three-month and nine-month followups (all $p < .005$). The treated subjects' improvement in grip strength was also maintained at both followups ($p < .10$). In contrast, the control group showed no significant change in FM scores or grip strength. The four treated subjects who were able to perform the hand function tests and finger tapping at all four evaluations also improved on these tests. We conclude that chronic stroke patients can achieve and maintain functional improvements, especially by combining electrical stimulation techniques with voluntary effort.

KEY WORDS: Arm function; Cerebrovascular disorder; Hemiplegia; Rehabilitation

Following a cerebrovascular accident (CVA), any spontaneous recovery of upper limb motor function that occurs is generally limited to the first six months.[1-3] During this period, motor recovery has been reported to be enhanced, beyond that attained by conventional therapy, by rehabilitation techniques including neurofacilitatory physical therapy,[4] EMG biofeedback,[5,6] and positional feedback with electrical stimulation.[7]

However, there is a consensus that current rehabilitation techniques are less effective in improving upper limb motor function in chronic CVA (>6 months).[8] It is unclear whether neuromuscular facilitation is efficacious. EMG biofeedback alone appears to be less effective than in an acute CVA,[9] but may be a useful adjuvant in combination with physical therapy.[10] New technical therapies such as multichannel EMG,[11] teaching patients to copy normal muscle function with the paretic limb,[12,13] presequenced multichannel electrical stimulation of paretic muscles[14,15] and EMG-initiated electrical stimulation[16] may be of therapeutic value, but up to now, no controlled studies of these techniques have been published.

Many of the treatment techniques used to enhance upper limb function in chronic stroke patients involve either biofeedback or electrical stimulation. Feedback cues from electrically stimulated movement may be even more effective than conventional EMG biofeedback training with audiovisual cues because more appropriate afferent pathways (from joint and muscle receptors) are activated. The first report of a device that electrically stimulates a target muscle when triggered by a voluntary EMG signal from that muscle, ie, EMG-stim, reported improvement in wrist range of motion (ROM) during use and recommended using the device for feedback training.[17] The 1987 study by Fields[16] reported improvement in wrist ROM and extensor muscle EMG activity in chronic stroke patients receiving EMG-stim as part of an intensive physical therapy program. In that uncontrolled study it was not clear, however, whether improvement was due to EMG-stim or to the comprehensive physical therapy program of which it was a part.[18]

Low-intensity electrical stimulation of wrist extensor muscles has been shown to reduce flexor spasticity.[19] In addition, stimulation approaching, but not exceeding, the gross motor threshold may also facilitate voluntary recruitment. In an attempt to combine these benefits, we considered the use of noncontingent, low-intensity electrical stimulation of upper limb extensor muscles combined with voluntary effort. We have theorized that this might improve muscle balance by reducing spasticity and enhancing (biasing) the excitation of extensor muscles relative to flex-

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This research was supported by grant no. G008435053 from the National Institute on Disability and Rehabilitation Research and the University of Washington's Department of Rehabilitation Medicine Neurologic Rehabilitation Research Fund.

Submitted for publication January 24, 1991. Accepted in revised form May 15, 1991.

The authors have chosen not to select a disclosure statement.

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0003-9993/92/7303-0002$3.00/0

ors. We believe that an appropriate term for this would be bias/balance (B/B) treatment; this modality is included in the present study.

The purpose of our research was to investigate whether rehabilitation techniques would improve the function of the paretic upper limb in chronic stroke patients when compared to an untreated control group. In addition, we also compared the relative effectiveness of several treatment techniques—EMG-stim, B/B, and proprioceptive neuromuscular facilitation (PFN).

METHODS

Subjects

Twenty-two subjects with chronic upper limb paresis due to CVA entered into either a therapeutic or control program. Subjects were matched in each group by age and time poststroke. Eighteen subjects completed the study, so final grouping was not as balanced; four subjects were treated but excluded from data analysis because they did not complete at least one follow-up evaluation. Three therapeutic programs were evaluated: six subjects received EMG-stim, four received B/B, and three received PNF treatments. Subjects were treated for a 3-month period, and they had two follow-up evaluations during the subsequent nine months. Five subjects were enrolled in the control group and received the same evaluations, but had no therapy.

The table summarizes the characteristics of the subjects represented in the results. All subjects were right-handed chronic stroke patients who were at least one year post-CVA, except one treated subject who was 6 months post-CVA at the beginning of the study. The average time post-CVA was 25.9 months, and it ranged up to 72 months. All subjects were clinically stable (ie, recovery had plateaued), had postural stability, and consented to participate according to procedures approved by the our university's Human Subjects Review Committee. All types of CVA were included since etiology is not considered as important in chronic stroke prognosis as it is in acute prognosis. Patients were instructed to continue their prescribed medications and existing home programs. The seven women and 11 men included 11 with right CVA (left hemiparesis) and seven with left CVA (right hemiparesis). Ages ranged from 46 to 78 years ($\bar{x} = 62.8 \pm 8.9$) at the beginning of the study.

Characteristics of Study Subjects

Treatment Groups	Months Post-CVA	Age	Gender	Side of Paresis	Initial Fugl-Meyer Score
EMG-initiated	6	60	M	L	33
electrical	15	68	M	L	5
stimulation	19	64	M	R	13
($n = 6$)	20	58	M	L	19
	24	57	M	L	42
	72	50	M	R	30
Means	26.0 ± 23.4	59.5 ± 6.2	6M	4L, 2R	23.7 ± 13.8
Bias/Balance	18	77	F	R	51
($n = 4$)	22	66	F	L	19
	49	67	F	L	38
	58	49	M	R	25
Means	36.8 ± 19.8	64.8 ± 11.6	1M, 3F	2L, 2R	33.3 ± 14.2
Proprioceptive	12	68	F	R	17
neuromuscular	14	70	M	L	44
facilitation	17	63	F	R	41
($n = 3$)					
Means	14.3 ± 2.5	67.0 ± 3.6	1M, 2F	1L, 2R	34.0 ± 14.8
Means for all treatment groups ($n = 13$)	26.6 ± 19.9	62.8 ± 7.9	8M, 5F	7L, 6R	29.0 ± 13.9
Control	18	65	M	L	8
($n = 5$)	22	46	M	L	10
	22	57	M	L	18
	25	78	F	L	14
	34	70	F	R	17
Means	24.2 ± 6.0	63.2 ± 12.3	3M, 2F	4L, 1R	13.4 ± 4.3

Treatments

Subjects receiving EMG-stim were treated by an occupational therapist in a hospital outpatient clinic three times a week for a total of 36 one-hour sessions. Transcutaneus electrical stimulation, triggered by a low level of voluntary EMG activity in the target muscle, was delivered to forearm wrist extensor muscles to produce joint movements. Stimulus intensity was set to produce maximum wrist extension, but not maximum force. The level of voluntary surface EMG activity required to trigger stimulation began as low as 5μV, and it was increased gradually by the therapist as voluntary recruitment improved. In addition to wrist extensors, EMG-stim was used over paretic finger or elbow extensors, forearm pronators or supinators, or shoulder elevators or abductors according to the subjects' abilities as determined by the therapist. This EMG-stim was provided by an Automove[a] stimulator in 0.2msec biphasic square-wave pulses at rates of 30 to 90Hz and a constant current of 20 to 60μV for 10 seconds. Figure 1 illustrates electrode placements over wrist and finger extensors, and shows an example of wrist extension.

Subjects receiving B/B stimulation were trained by a physical therapist to administer their own therapy using a Respond II[b] electrical stimulator, according to our protocol, for 30 minutes five times a week for 3 months. Low-intensity stimulation was applied to wrist extensor muscles in 0.3msec square-wave pulses at rates of 30 to 90Hz. Stimulation was applied at an intensity that increased the subject's voluntary range of wrist extension without producing any visible movement at rest; the hand fell into a gravity-assisted flexed position when the subject relaxed. The B/B subjects were instructed to perform three sets (30 contractions or to fatigue) of voluntary wrist extension exercises while stimulation was applied each session. To assure compliance, subjects recorded each home session on a calendar that was reviewed with the therapist in biweekly meetings.

Subjects receiving PNF were treated by a senior member of our university's physical therapy curriculum faculty for approximately one hour three times a week, for a total of 36 sessions over a three-month period. According to methods described by Voss and colleagues,[20] PNF treatments en-

Fig 1—EMG-stim device in place on patient's paretic arm. The three small electrodes are the EMG-sensing electrodes by which voluntary activity triggers FES of the extensor muscles by the two large electrodes. The figure at left shows limited voluntary wrist extension. The figure at right shows full extension during EMG-stim.

compassed the whole upper limb, including wrist extension.

Evaluations

Subjects were objectively evaluated by an experienced physical therapist upon entering the study (pretreatment), within one week after completing therapy (posttreatment), three months after completing therapy (3-month follow-up), and nine months after completing therapy (9-month follow-up). One EMG-stim subject had surgical complications and was not testable at the 9-month follow-up.

Measures for evaluating improvement included four commonly used quantitative tests that were not likely to be influenced by the practice effect inherent in repeated evaluations. These tests were selected to encompass the wide range of abilities among stroke survivors, and included measures of both gross and fine movements.

Our major evaluation tool was the Fugl-Meyer (FM) poststroke motor recovery test.[21] The FM test evaluates reflexes, the quality of movement (synergy-bound and synergy-free), and strength in the arm and hand. Each subject received a total score for the upper limb portion ranging from 0 to 60, based on a rating of 0, 1, or 2 for each of 30 items. The advantage of the FM is that it can be used to quantify improvement in patients with severe hemiplegia as well as those with milder deficits. Its reliability and validity were established by Duncan and associates[22] and Berglund and Fugl-Meyer.[23]

Grip strength was measured in pounds with a Jamar hand dynamometer according to the method used by Mathiowetz and colleagues[24] to establish reliability and validity. All subjects had a measurable grip which, unlike the FM, was not limited by a maximum score.

Although the FM is a comprehensive test of motor control and had the advantage of allowing all subjects to be scored on the same scale, it is less sensitive for subtle deficits than functional performance tests. Therefore, to further quantify functional change in abilities of subjects with less sever deficits, we also used subtests of the Jebsen-Taylor (JT) hand function test.[25] This is a widely used, standardized test of hand function, scored by total seconds required to complete ten subtests. Reliability was established by the original authors. We used the three subtests that were shown by Jebsen and associates to be most sensitive to post-stroke recovery: ie, card turning, simulated feeding, and small objects.[25] All subjects attempted these three subtests, but many were not able to obtain scores.

In the less severely affected patients, we were also able to measure rapid movement with the finger tapping test, according to the method of Reitan.[26] Finger tapping is a commonly used, reliable neuropsychologic test of fine motor control in the distal upper extremity. We recorded the number of taps in ten seconds by the index finger on a telegraph key.

Statistical Analysis

The significance of changes within each group over the four evaluations and the differences between groups were tested by three separate ANOVAs for grip strength (lbs), total seconds for hand function tests, and number of taps in ten seconds. However, nonparametric tests were used to compare FM scores that did not meet the ANOVA requirement of interval level measurement. Wilcoxon T tests were used to assess trends within groups, and Mann-Whitney U tests were used to assess differences between groups.

RESULTS

Fugl-Meyer (FM) test

The 13 treated subjects improved their scores on the FM and maintained the improvement for the entire 9-month follow-up period (fig 2). Average FM scores of the 13 treated subjects increased significantly from pretreatment to posttreatment (29.0 ± 13.9 vs 37.5 ± 13.2; $T_{13} = 0$, p

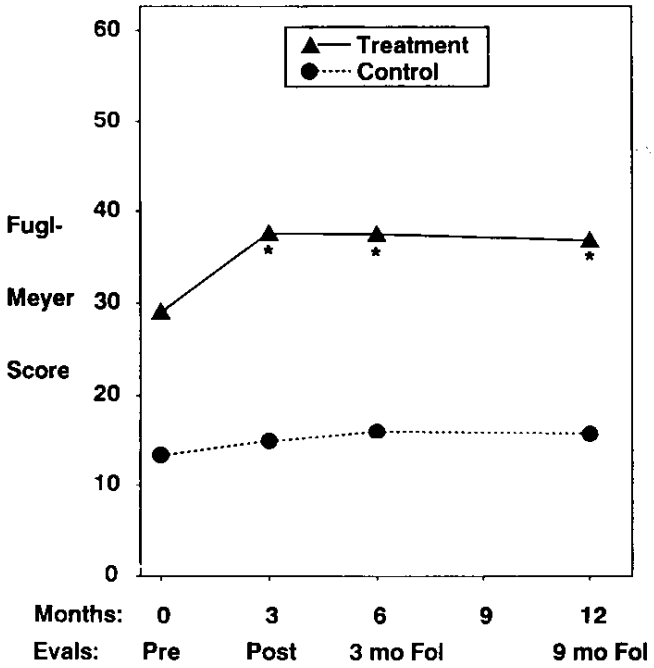

Fig 2—The FM scores of 13 treated subjects improved from pretreatment to posttreatment and remained higher at 3- and 9-month follow-ups (asterisks indicate all $p < .005$). Scores of five untreated control subjects did not improve significantly.

$< .005$), and remained significantly higher than pretreatment at 3-month (37.4 ± 12.7; $T_{13} = 0$, $p < .005$) and 9-month (36.7 ± 12.1; $T_{12} = 3$, $p < .005$) follow-up evaluations. Comparison of the treated and control groups showed that treated subjects improved significantly more than controls from pretreatment to posttreatment ($+8.5 \pm 2.9$ vs $+1.6 \pm 3.2$; $U_{13,5} = 2$, $p < .001$), from pretreatment to 3-month follow-up ($+8.4 \pm 3.5$ vs $+2.6 \pm 3.1$; $U_{13,5} = 8$, $p < .01$), and from pretreatment to 9-month follow-up ($+7.8 \pm 5.6$ vs $+2.4 \pm 2.1$; $U_{12,5} = 11$, $p < .05$). Average FM scores of the five untreated control subjects did not increase significantly over the same intervals (13.4 ± 4.3, 15.0 ± 7.2, 16.0 ± 7.1, and 15.8 ± 6.0 for pretreatment, posttreatment, and 3- and 9-month follow-ups, respectively).

Comparing the treatment groups, the EMG-stim group had the greatest improvement in FM scores from pretreatment to posttreatment (from 23.7 ± 13.8 to 33.7 ± 12.9; $T_6 = 0$, $p < .025$). Comparisons of pretreatment to posttreatment gains in the three therapeutic programs (fig 3) show that the EMG-stim group improved significantly more than the PNF group ($+10.0 \pm 3.3$ vs $+6.0 \pm 0$ $U_{6,3} = 0$, $p < .05$) but not significantly more than the B/B group ($+8.2 \pm 2.1$).

The FM scores of EMG-stim subjects remained higher than pretreatment at the 3-month follow-up (23.7 ± 13.8 vs 33.7 ± 12.9, $T_6 = 0$, $p < .025$) and at the 9-month follow-up (32.0 ± 12.7, $T_5 = 0$). The FM scores of B/B subjects rose from pretreatment to posttreatment (33.3 ± 14.2 vs 41.5 ± 14.5), then fell slightly at three- and nine-month follow-ups (39.8 ± 13.3 and 37.8 ± 10.2). The FM scores of PNF subjects increased at successive evaluations from 34.0 ± 14.8 at pretreatment to 40.0 ± 14.8 at posttreatment, 42.0 ± 17.6 at 3-month follow-up, and 43.0 ± 14.4 at 9-month follow-up. The changes in FM scores were not statistically significant in the smaller treatment subgroups.

We examined the possibility that initial differences in the severity of paresis among the four groups might account for differences in improvement. First, we compared the treated subjects whose pretreatment FM scores were below the median of 30 with subjects who scored above the median. The more severely affected group showed slightly greater improvement (+8.8) from pretreatment to posttreatment than the less severely affected group (+7.3). Then we compared the less severely affected with the more severely affected EMG-stim subjects. There was no appreciable difference in pretreatment to posttreatment FM gains (+10.3 vs +9.7) by EMG-stim subjects with the three lowest (mean = 12.3) vs the three highest (mean = 35.0) initial FM scores. Thus, it does not appear that the treated groups improved solely because they were at a higher level of function at the start. The lack of change in the control group demonstrates that subjects did not improve their performance scores as a result of repeated testing.

Grip Strength

Grip strength improved among the 13 treated subjects from pretreatment to posttreatment (13.0 ± 8.2 vs 14.4 ± 9.5 lbs), but the difference was only marginally significant ($t_{12} = 1.4$, $p < .10$) (fig 4). The treated group's grip

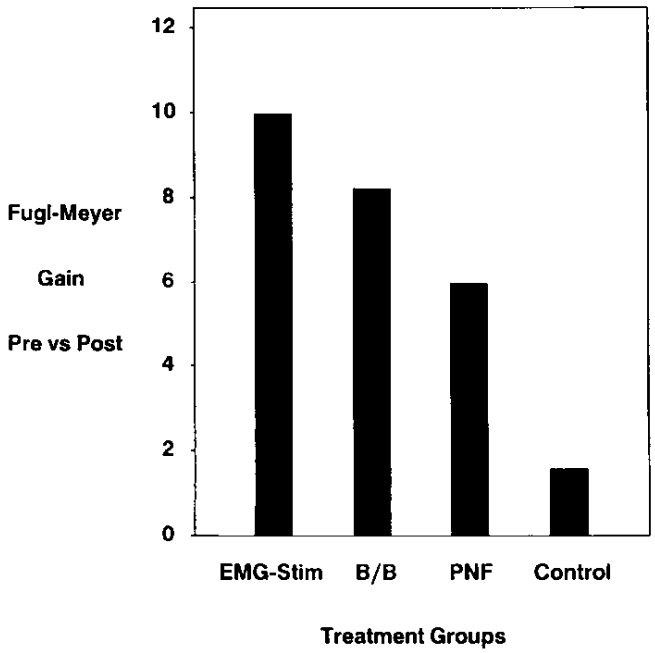

Fig 3—Change in FM scores from pretreatment to posttreatment (36 weeks) in each treatment group. It should be noted that groups did not have identical pretreatment FM scores. Pretreatment FM means were: EMG-stim = 23.7, B/B = 33.3, PNF = 34.0, and control = 13.4. Thus, the EMG-stim group improved 42%, the B/B group improved 25%, and the PNF group improved 18%.

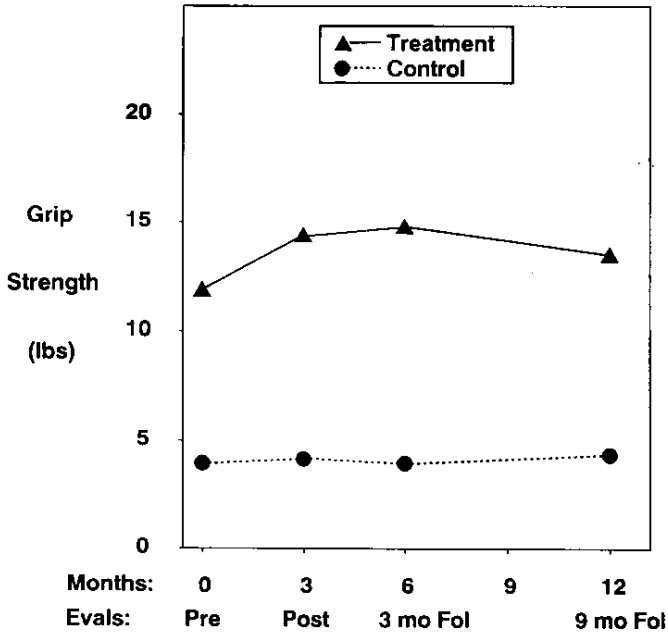

Fig 4—Grip strength of the 13 treated subjects increased, but the improvement was only marginally significant ($p < .10$). Grip strength of the five control subjects did not change.

continued to be slightly stronger at the 3-month follow-up than before treatment (14.8 ± 10.1lbs; $t_{12} = 1.58$, $p < .10$). The 12 treated subjects remaining at the 9-month follow-up had stronger grip (13.5 ± 8.9 vs 11.9 ± 7.5lbs; $t_{11} = 1.64$, $p < .10$) than before treatment. Grip strength in the five untreated control subjects did not change significantly over the same evaluation intervals (4.0 ± 5.8, 4.2 ± 6.3, 4.0 ± 5.8, and 4.4 ± 6.7lbs for pretreatment, posttreatment, and 3- and 9-month follow-ups, respectively).

Comparing grip strength in the three treatment groups, the EMG-stim subjects changed most from pretreatment to posttreatment (+2.3 ± 4.6lbs), but their improvement was not significantly greater than the improvement in the B/B group (+0.8 ± 3.0lbs) or the PNF group (+0.3 ± 2.1lbs). The improvement in grip strength by the EMG-stim group from pretreatment to posttreatment was marginally significant (12.8 ± 11.1 vs 15.2 ± 12.3lbs; $t_5 = 1.23$, $p < .10$), and was maintained at the 3-month follow-up (16.2 ± 12.6 lbs; $t_5 = 1.83$, $p < .10$). The five EMG-stim subjects remaining in the study at the 9-month follow-up evaluation had stronger grip than before treatment (12.8 ± 11.1 vs 10.2 ± 10.1lbs; $t_4 = 1.56$, $p < .10$).

Hand Function and Finger Tapping Tests

Only four treated subjects (1 EMG-stim, 2 B/B, and 1 PNF) had sufficient dexterity to perform all three JT subtests and the finger tapping test at all four evaluations. The total time required to perform the three JT subtests decreased from an average of 154.0 ± 82.8sec pretreatment to 100.0 ± 60.7sec posttreatment ($t_3 = 2.19$, $p < .10$). Performance at both follow-ups was faster than before treatment (125.2 ± 86.7 and 126.0 ± 85.1sec at 3- and 9-month follow-ups, respectively; $t_3 = 1.17$ and 2.15, both $p < .10$). The number of taps in 10 seconds increased from 19.5 ± 9.4 pretreatment to 25.0 ± 7.7 posttreatment ($t_3 = 2.09$, $p < .10$). Performance at both follow-ups was not significantly faster than before treatment (21.0 ± 8.7 and 21.2 ± 6.8 for 3- and 9-month follow-ups, respectively).

DISCUSSION

The results demonstrate that specific types of therapy can improve paretic upper limb movement, strength, and function in chronic stroke patients with moderate to severe deficits. Furthermore, our subjects not only improved with treatment, but they also maintained most gains for nine months. Significant, measurable improvements with treatment are difficult to demonstrate in chronic hemiparetic patients,[3,27] and discrepancies in the literature on effectiveness of treatment for chronic hemiparesis may be due to the wide variability of stroke patients, treatments used, and methods for evaluating improvement.

Facilitation techniques are widely used, although there have been only a few controlled studies of their efficacy. All of these previous studies have been in acute CVA, and results have been contradictory. For example, Logigian and colleagues[28] compared conventional therapy to Bobath's[29] facilitation method in patients less than seven weeks poststroke. The Barthel Index and manual muscle testing showed no differences between the improvement recorded in these two groups. How much of the improvement was due to the spontaneous neurologic improvement after stroke and how much was due to the therapies was unclear. In contrast, Basmajian and associates[4] compared the Bobath method to a program that included EMG biofeedback (a regimen that had been demonstrated to be more effective than conventional therapy[5]) for patients one to 12 months (mean = 16 weeks) poststroke. Significant improvements were achieved and maintained by both groups, and no difference was found between the two therapy methods. In another acute study, Lord and Hall[30] found a facilitation program to be comparable in outcome to a traditional therapy program, with the exception that self-feeding was better in the facilitation group. A recent study of acute patients showed no difference between the results obtained with either Bobath or Brunnstrom neurofacilitatory therapy.[31] Our study evaluated PNF in chronic stroke patients 12 to 17 months post-CVA and compared the results with an untreated control group. Our results suggest that PNF has only limited efficacy in chronic stroke rehabilitation (fig 3).

Previous studies of EMG biofeedback with audiovisual cues demonstrated that this form of treatment was more effective than conventional therapy for patients who were less than three months poststroke or for those who were not too severely affected if treated more than three months poststroke.[5] Ince and colleagues[6] found greater improvement in patients less than nine months poststroke with EMG-biofeedback training than with traditional therapy; they recommended that similar training should be included in all stroke rehabilitation programs. For patients more than six months poststroke, Inglis and associates[10] demon-

strated greater increases in strength and ROM with EMG biofeedback than with conventional therapy; the same patients who had conventional therapy improved further when they subsequently received biofeedback training. Biofeedback applied to agonist and antagonist muscle groups reduced inappropriate coactivation in subjects ten to 62 months post-CVA.[11]

Electrical stimulation of muscles has been used for functional retraining since the 1960s, and it has recently become very popular because of the development of compact, portable devices for stimulation.[32] Merletti and colleagues[33] found the effectiveness of functional electric stimulation (FES)—which produces a muscle contraction and moves a joint—to be less dependent than other types of therapy on age, degree of deficit, side of lesion, or time poststroke. In addition to FES, which sometimes can increase spasticity by spread to flexor muscles, low-intensity electrical stimulation (below motor threshold) also appears to have therapeutic value by reducing spasticity.[19,34] Alfieri[19] found low-intensity electrical stimulation of wrist and finger extensors to produce a decrease in spasticity of antagonist flexor muscles that persisted beyond the period of treatment.

We postulated that low-intensity electrical stimulation of weak upper limb extensor muscles may bias them to assist a weak voluntary contraction, resulting in better balance across a joint. In this study, our B/B treatment combined low-intensity stimulation with voluntary exercise. Our results (fig 3) demonstrate that B/B is effective in chronic stroke patients and only slightly less effective (but not statistically) than EMG-stim.

The EMG-stim treatment that we used in this study combined FES, biofeedback, and exercise. The patient's muscle-specific voluntary effort triggered electrical stimulation that not only moved the target joint through its maximum range, but also produced accompanying sensory (visual, cutaneous, and proprioceptive) feedback—all contingent upon the patient achieving a criterion level of EMG activity in the target muscle. In the only other report of therapy involving EMG-stim treatment, Fields[16] showed that extensive physical therapy, including EMG-stim, increased surface EMG activity in upper limb extensors and lower limb dorsiflexors in 69 patients with chronic hemiparesis. However, evaluation measures were limited, no followup was done, and no control group was evaluated.[19] Our study demonstrated EMG-stim to be the most effective of our three treatment protocols (fig 3), with a statistically significant ($p < .05$) advantage over PNF, but only a slight advantage over B/B.

What accounted for the improvement seen in our treatment groups receiving electrical stimulation? Albert and Andre[35] have pointed out that FES of the type used in EMG-stim produces the following effects: (1) muscle contraction due to direct stimulation of the motor neuron; (2) reduction of spasticity due to afferent stimulation; and (3) an information effect from the joint and muscle afferents, cutaneous sensation, and visual perception of the movements produced. An additional benefit is the preferential contraction of type II muscle fibers. In contrast to voluntary recruitment, which activates type I motor units first, FES selectively activates type II motor units due to their lower axonal input resistance.[36] We hypothesize that this "reverse recruitment" phenomenon may be especially beneficial in hemiplegic atrophy.

Muscle atrophy occurs in chronic stroke patients, with selective atrophy of type II muscle fibers the predominant finding.[37-39] Bohannon and Andrews[40] suggested that muscles with more type II fibers may be weaker poststroke as a result of a loss of the efferent drive; it may not be adequate to meet their threshold for activation, which is higher than that of type I fibers. We electromyographically evaluated some of the subjects in this study and found smaller motor unit action potential (MUAP) amplitudes during maximal effort in paretic arms than in nonparetic or control arms, probably indicating less type II activity as well as fewer superimposed MUAPs.[41]

Low-intensity electrical stimulation, as used in the B/B group, would have effects similar to FES, with the exception of an information effect. Although gross muscle contraction is not produced by B/B simulation, the voluntary muscle contraction might be more functional since the flexor/extensor activity of the extremity is better balanced. A technical advantage of the B/B techique is the relative simplicity of the stimulator when compared with that required for EMG-stim.

Studies of other techniques employing active patient involvement during FES have demonstrated improvements that are consistent with our results. Electrical stimulation triggered mechanically by voluntary wrist extension in patients within four months of stroke was reported to produce a 280% increase in isometric wrist extension torque and a 200% increase in wrist ROM, compared to no increase in torque and only a 50% increase in ROM by conventional physical therapy.[7] Active involvement appears to be a common factor required for motor improvement after CNS lesions.

There are some experimental data supporting the importance of active involvement to produce cortical remodeling. In primates, there is evidence that cortical representation of skin surfaces is remodeled by use throughout life. Such remapping of cutaneous receptive fields is enhanced by repeated tasks that produce cutaneous stimulation of a limited sector of skin on the distal phalanges.[42] Jenkins and Merzenich[43] speculate that a correspondingly greater capacity for reorganization after cortical lesions would be expected in representations higher in the cortical hierarchy, such as "associational" cortical areas. Other animal studies also support the concept of the importance of brain plasticity in recovery from stroke.[44]

The ultimate effect of any central nervous system efferent lesion is to alter the behavior of the motor unit (MU). We previously reported in greater detail that MUs in muscles of a paretic arm respond more slowly after stroke, and MUs in wrist extensor muscles may not be able to maintain their firing.[45] There is also more cocontraction,[46] a slower MU firing rate,[47] a greater number of gaps in the MU firing patterns,[48] and muscle imbalance, with MU activity reduced more in extensor muscles than flexor muscles.[49] After the treatments reported in this paper, gaps in the firing pattern are reduced[50] and cocontraction is altered.[51] Other possible explanations for the improvement seen in

the treated groups include reversal of habitual disuse of the paretic arm,[52] increased peripheral circulation, improved muscle metabolism, and mechanical changes in noncontractile tissue.

New approaches to treating upper limb paresis continue to be reported. Wissel and colleagues[12] and Wolf and associates[13] used a biofeedback approach to train patients to reproduce with the paretic arm EMG patterns recorded from the nonparetic arm during functional tasks. Lagasse and colleagues[14,15] used multichannel FES preprogrammed by muscle activity sequences from a patient's normal limb to stimulate muscle sequences in the paretic limb. Both of these studies reported successful results in chronic stroke patients, although evaluations were limited, there were no controls, and no follow-up was reported. Kiwerski[53] suggested that hand function can be improved with phenol blocks of the median and ulnar nerves in combination with an implanted electrical stimulator of the radial nerve. Spasticity may also be reduced for a prolonged period with a relatively short period of alternately stimulting flexors and extensors.[54]

There are many difficulties inherent in rehabilitating upper limb function after stroke. Rehabilitation techniques have been more successful for the lower limb than the upper limb. This is because, in large part, the demands placed on the lower limb are less complex. To be functional, the lower limbs must only hold the body upright and propel it in the desired direction. The upper limb, however, includes the hand—man's most sophisticated neuromuscular apparatus. The arm and hand must feel, hold, and manipulate. These are more difficult tasks than those required of the lower limb.

In addition, there are physiologic limits to recovery, especially in the upper extremity. As Waxman[55] pointed out, the specificity of the pyramidal system imposes certain limitations on the degree of recovery that can be achieved after destruction by stroke. Porter[56] stressed that destruction of the corticospinal tract produces permanent deficits in fine motor control that prevent recovery of isolated distal movements, especially in the upper limb.

There are also problems inherent in stroke rehabilitation research. Well-designed studies are complex; they must take into account the location, extent, and age of the lesion, the patients' previous and subsequent medical problems, as well as many psychosocial influences.[8] Stroke patients are very heterogeneous. This heterogeneity is illustrated in our study by several subjects who did improve with treatment, but whose complications from chronic disease compromised the maintenance of those gains. The size and complexity of well-designed studies, the labor-intensive characteristics of rehabilitation treatments, and the long period of time required for treatment and follow-up contribute to making stroke rehabilitation research difficult and expensive.[44]

CONCLUSION

Despite the difficulties of research on stroke rehabilitation, we suggest that chronic stroke patients can achieve and maintain improvements in the upper extremity, especially with treatments combining electrical stimulation techniques with voluntary effort.

Acknowledgments: The authors gratefully acknowledge the contributions of three therapists—Laura M. Robinson, MPT, Martha J. Trotter, BS PT, and Nancy Skaale, OTR/L—as well as the editorial assistance of Carolyne Dollar. Equipment for electrical stimulation was provided by Medtronics, Inc., and Biometer, International.

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